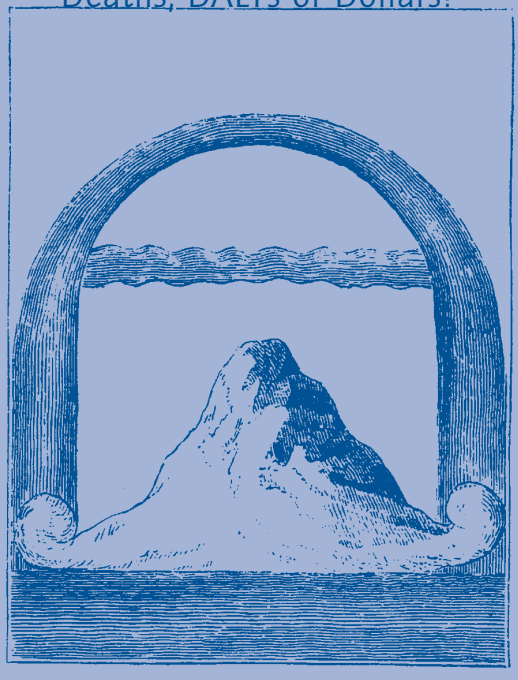
Assessing and evaluating the health impact of environmental exposures

"Deaths, DALYs or Dollars?"



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"Deaths, DALYs or Dollars?"

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Het schatten en beoordelen van de gezondheidseffecten van milieuverontreinigingen:
doden, DALYs of duiten? (met een samenvatting in het Nederlands)

Proefschrift ter verkrijging van de graad van doctor
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Content

Preface

About twenty years ago (less a month), June 1984, I began my first serious job at the Health Council of the Netherlands in the Hague. For some mysterious reason I was picked out and enabled to leave behind the impressive army of unemployed academics of those days. As a junior scientific secretary to this renowned institution I was appointed to the task of supporting a committee of distinguished scientist in reviewing a series of criteria documents on environmental pollutants, produced by the National Institute of Public Health and the Environment (RIVM) in co-operation with several other research institutes.

Influential reports on the state of the environment such as 'Limits to Growth', 'Silent Spring' and -last but not least- the Dutch 'Concern for Tomorrow'-report by RIVM had evoked a great sense of environmental urgency and the Dutch Government had started an ambitious program to regulate the most dangerous industrial chemicals. These were often animal carcinogens produced in high quantities as *mutagenicity* and *carcinogenicity* tests were simply the best toxicology had to offer at that time ('looking for lost car keys under a streetlight'). Attributable disease burden was an unknown phenomenon yet. The committees simply had to come up with safe or at least tolerable exposure levels, applying straight forward, inherently conservative extrapolation models to the results of animal assays, even if it concerned tumours in organs humans didn't have. Zero-risk was still generally considered to be a feasible target, and we were explicitly asked to always 'err on the safe side'.

As I grew slightly more senior at the Council my scope broadened and included more down to earth topics, such as the hygienic conditions in therapeutic spas, whirl and swimming-pools (tropical theme parks), passive smoking, smog episodes of particulate and photochemical air pollution, risk assessment and risk management. Gradually my peers in health risk assessment and I developed a better feeling for analysing environmental dangers at the appropriate level: public health (including the perils of safer than safe). What disease can we attribute to environmental exposures? What health is gained by health protection? What health is encompassed by 'one in a million per year'?

At the end of 1991 I moved to the RIVM to join the 'wondrous world' of models and scenario tools developed in the framework of National, European and Global Environmental Outlooks (in succession to the 'Concern'-report). In this domain the question of quantifying environmental disease burden was even more pertinent, especially since at the same time the World Bank/WHO Global Burden of Disease Project started to pay off, offering whole new opportunities for comparative (environmental) health impact assessment. In the first Environmental Balance ('de Milieubalans'), -the annual state of the environment report- we were able to put the sense of urgency with respect to environmental health loss in its proper context. Since 1850 public health status has improved enormously, above all as a result of public hygiene, gaining control over environmental factors: hence focus has shifted from control to maintenance and improvement, from quantity to quality of life, including well-being. Or in economic terms: from the perspective of public health, the marginal returns of investments in environmental quality have decreased substantially. In the fifth Environmental Outlook we elaborated on these insights by presenting a framework for urban environmental quality assessment, incorporating social-economic, spatial characteristics and 'well-being' as well.

Of course the final logical step in this permanent education process is not only looking at health gains of (environmental) interventions in a comparative way, but also at the societal costs of environmental policies, analysis of cost-effectiveness and cost-benefits, with an open eye for distribution aspects (equity). 'Money is always tight, so when and how can we try to get the best deal in risk management?' In the past

years, in close co-operation with many others I had the pleasure of putting together these old en new ideas in a report tot the State Secretary of the Environment on 'dealing with environmental risks'. The report appears to be just in time to fuel a necessary and fruitful discussion on the fundamentals of health protection.

Both at the Health Council and RIVM (in different positions) I had the opportunity to work on health impact assessment methodology with excellent national and international experts (a/o EU, WHO, OECD) in numerous interesting projects, fairly independent and with an open mind. In this book I have brought together some examples of this work.

1 Friend and foe: health and the environment from an historical-epidemiological perspective[i]

Augustinus E. M. de Hollander, Pieter Bol

Abstract

Until Modern Times (20th century) health status in the Western world was primarily determined by environmental factors, ranging from physical phenomenon, such as severe climatic conditions, floods, fires, earthquakes to biological factors including famine, macro and micro-predators (pathogens). We briefly describe the interaction between man and the environment, in particular the microscopic world during the last ten millennia. In those 10,000 years our world has seen an evolution from a patch work of hundreds of thousands of small communities towards a 'global village'.

Since the public health transition during the 19th century unfavourable social and life-style factors have gradually become the most significant causes of avoidable health loss. In the future a fourth group of endogenous factors may gain public health significance: individual genetics. This chapter identifies the principal determinants of health, and reviews the changing nature and importance of environmental and societal forces for health status. It is concluded that the current situation calls for new definitions of health and well being, new indicators of environmental impact, as well as for a new demarcation of the field of environmental quality. Environmental health is preferably part of an integrated, multi-disciplinary, multi-sectoral approach of spatial, social, economic and urban development policies.

[i] Based on *Hollander AEM de, Staatsen BAM. Health, environment and quality of life: an epidemiological perspective on urban development. Landscape Urban Planning 2003; 65: 53-62.; Bol P, Hollander AEM de. The 'decomparting' of the World. In: RIVM. Bouwstenen voor het NMP4. Aanvullingen op de Nationale Milieuverkenning 5. Bilthoven: RIVM, 2001; Hollander AEM de, Bol P, Niessen LW. Volksgezondheid en omgevingsfactoren: van beheersing naar beheer. In: Rijksinstituut voor Volksgezondheid en milieu. (Achtergronden bij de) Milieubalans 1995. Alphen aan den Rijn, Samson H.D. Tjeenk Willink bv, 1995.*

1.1 Introduction

A host of environmental risks pops up in headlines almost every day. BSE-prions causing Creutzfeldt-Jakob disease among meat eaters all over Europe - the reverse of a cynical bio-industry turning herbivores into carnivores-, genetically modified 'Frankenstein' foods threatening the integrity of the fine web of ecology, as well as our health, trillions of ultra fine particulates from exhaust pipes stuffing our lungs and killing more people than traffic accidents, climate change and the ozone hole.

Are these threats all symbols of modern post-industrial society wasting the health of its citizens or are they merely a sign of distorted risk perception by the public? If one bothers to analyse available public health statistics a slightly more refined picture emerges. Never in our Western-European history we have been as healthy as we are now. Children who were born in the beginning of this new millennium may count on almost eighty years of life expectancy[1]. In this paper we will analyse the past and current public health status, and the factors that determine it, in order to identify important aspects of the environmental health agenda for the next decade and to set the stage for this book on the assessment and evaluation of health impacts of environmental conditions.

1.2 Determinants of health

We will evaluate the development and significance of 'environmental health'[ii] using an elaborated version of the conceptual model that was proposed by the Canadian Minister of Health Marc Lalonde in 1974[2,3], (see *Figure 1-1*). According to this model health status can be regarded as a function of a variety of endogenous and exogenous factors. Basically four groups of determinants can be distinguished: human biology or *endogenous individual* attributes, either part of our *genetic* make-up or *acquired* during life, the *environment* (including *physical* and *social* aspects), general *behaviour* (or *life-style*), and *health care* (including prevention and health promotion).

The most appealing example of a *genetic* factor affecting a person's state of health is, of course, gender. Regardless of the prevailing health system, all over the world women live several years longer than men do, and as far as we know they always did[4,5,6]. Most endogenous, individual characteristics develop through interactions between genes and environmental factors and thus have both a genetic and an *acquired* component, for example length, blood pressure, blood lipoprotein composition (familial risk factors), and personal (psychological) attributes[2,7,8,9,10]. The interactions between these exogenous and endogenous determinants explain why the response to environmental exposures may vary substantially from one individual to the other[7,11,12,13].

The *physical* and *social* environments, as well as *life-style* are regarded as *exogenous* determinants. The physical environment includes factors such as radiation, noise and heat (*physical* factors), hazardous substances in the outdoor and indoor environment, including our working environment, the air we breath and the food we eat (*chemical* factors); and - needless to say - a myriad of bacteria, viruses and other micro- and macro-organisms (predators), which may have both positive and negative effects on health status (*biological* factors).

Micro-organisms have been a predominant cause of human diseases throughout human history, and hence play an important role in this chapter. But that is not the whole story. At the same time the microbial world has been essential for the evolution of the more complex life forms on the planet. *Mankind*

[ii] A linguistically inappropriate term tot describe health in relation to environmental conditions or exposures

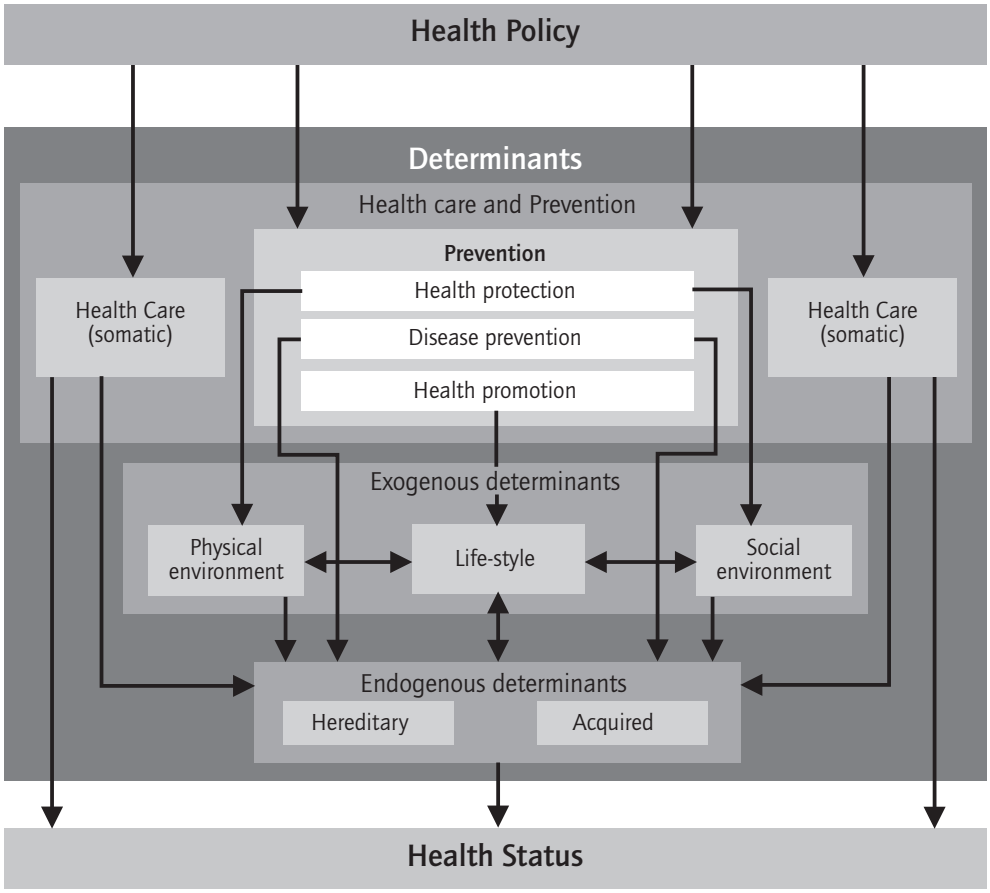

Figure 1-1. Conceptual model: determinants of public health status[2]. Three clusters of factors are discerned: endogenous or personal factors, including genetic make-up and factors (susceptibilities) acquired in the course of life; the factors in this cluster are influenced by exogenous determinants, social and physical environment and life-style, that form the second cluster; the third cluster comprises the health care and prevention system that influences the state of health, with or without interacting with exogenous and/or personal determinants.

may think of itself as the summit of evolution, as it dominates this world together with its domestic plants and animals. But man, just like other complex multicellular organisms, lives at the mercy of the myriads of microscopic creatures of which they are partly composed. Manipulations of the balance between man and micro world, with herbicides, insecticides, antibiotics, detergents, vaccinations and the like, can offer advantages, but their effects in the long run are difficult to predict. Thus, micro-organisms are neither 'good' or 'bad'; they just do exist, for a much longer time than creatures like us, and they have created the basic conditions that made our life on this planet possible.

In our present world, urbanised now for more than 50%, of course housing and spatial characteristics at the neighbourhood level, such as building density and variation, traffic related 'soundscapes', infrastructure barriers public transport accessibility, or the nearness of open and green space for recreational purposes, are part of the physical environment as well. *Life-style* factors include diet, smoking,

drug abuse, sexual behaviour, physical (in)activity and such. The social environment includes socio-economic status, the pattern of social networks, and cultural factors[3].

The concept of health as a dynamic equilibrium emphasises the interaction between determinants, resulting in a particular state of health. While exogenous determinants act on endogenous ones, there are also many interactions within the group of exogenous determinants. Life-style for instance, is to a considerable extent determined by social environment (e.g. family situation). At the level of neighbourhoods social quality, such as cohesion or social safety, may depend largely on the quality of the physical environment[14]. Aspects of lifestyle or behaviour, such as sunbathing, smoking, (un)safe sex, and personal hygiene may, in turn, largely determine exposure to factors from the physical environment such as UV radiation, carcinogenic substances, or pathogenic organisms. *Figure 1-1* also outlines the role of health care and prevention acting on both exogenous and endogenous determinants in various ways.

1.3 From a patchwork of isolated communities to the 'global village'

Before looking forward, we briefly describe the interaction between man and the environment, particularly the microscopic world during the last ten millennia. In those 10,000 years our world has seen an evolution from a patchwork of ten thousands of small communities towards a 'global village' where everything is in contact with everything. Neither distance nor time is an effective barrier anymore against massive and swift distribution of organisms from their original reservoirs.

The approximately 10 million people that inhabited the planet after the last Ice Age, lived in scattered bands as hunters and gatherers, subjected to the whims of the elements[15]. Their food supply was unsure; they were exposed to the weather of all seasons, to predators, and natural disasters like floods, fires and droughts frequently took their toll. In those communities the median age at death (the age at which half the individuals from the same birth year have passed away) was well under 5 years. In this epoch, as for the greater part of our history, infectious disease was the predominant cause of morbidity and often fatal, but the causal organisms originated mainly from the individual's own flora and contagion by tribe members or the direct environment. Early in life mothers and others handed down pathogenic micro-organisms to the infants, sometimes even before birth. For instance bacterial meningitis, until recently killing 1 out of 10 nomadic children before the age of five, is caused by bugs from the direct family[iii,16].

In the course of the last ten millennia man struggled for independence from the uncertainties of life. Gradual domestication of successively dogs, bovines, camels, sheep and goats meant a big leap towards a more guaranteed food supply and selective breeding improved the herds. But the triumph came with the nomads who started to manipulate the plants and fruits that they previously had gathered from the wild; soon larger crops could be yielded. Being a herdsman means moving around with animals, but taking care of plants (e.g. wild cereal cultivation) implies a sedentary life. The first significant transition that mankind experienced was in the era of the Neolithic (Agrarian) Revolution[iv]. This 'revolution' was not a momentous

[iii] Infections from the patient's own micro-flora are still an important cause of morbidity and mortality. Fatal pneumonia (poor man's friend) is the sixth cause of death in the USA and is mostly caused by the patient's own pneumococci. All urinary tract infections and virtually all cases of otitis have an endogeneous cause.

[iv] Transition is a term originating from economics. It describes the development of small scale local activities towards larger scale systems and increased complexity. Some authors claim the cultivation of fire by humans is the first transition[18]. At the moment also demography and medicine use the term, speaking of demographic and health transitions. The demographic transition moves from a situation with high birth and death rates (a high 'turn over') to a condition with low birth and death rates (a low 'turn over') and hence is coinciding with an increase in life expectancy. The health transition moves from morbidity and mortality originating predominantly from infectious diseases and nutritional problems to a situation where chronic morbidity and death by mainly cardiovascular diseases and cancer prevail.

event, but rather a process of intensification of the relationship between *human communities* and their *environment* and, of course, among each other[17]. In some areas such as Turkey's South coast this epoch started as long ago as 8,000 BC, but in the America's presumably not before 1000 BC[15,16].

In the villages and towns that were the result of the Agrarian Revolution we would observe labour division and specialisation, and successively social stratification. Food reserves were created in the surrounding countryside and this surplus implied opportunities for people outside the food production to dedicate themselves to, for instance, tool making or writing. Agrarian populations were more productive than foragers, at least per unit of land (definitely not per unit of labour), they were also much more vulnerable. Therefore two types of specialisation probably represent the oldest professions in the world: *priest* and *warrior*. Priests were needed to invoke the higher powers for good crops, to preserve knowledge and to strengthen the self-restraint and the discipline required for a hard farming life (saving sowing-seed in hard times). Warriors of course were needed for protection as dangers from the extra-human world (droughts, floods, (micro)predators) were more or less exchanged for inter-human danger (hostile neighbours, invading armies). Often these processes led to fatal interaction of military protection and economic exploitation[18,19].

Nonetheless, the settlements, especially the larger towns and first cities, experienced also the disadvantages of crowding: high densities of the human species in a restricted area, often combined with relatively high densities of herded or domesticated animals, and proliferation of vermin, parasites and other disease vectors, favouring the closing of contamination cycles and thus creating ample opportunity for old and new pathogens. Furthermore, although their crops were high in net calorie value, the diets of the early agrarians were much more monotonous than those of the hunter-gatherers, lacking important 'secondary' food components that are essential to human health, such as certain proteins, minerals and vitamins[20]. Only in the past century the rich diversity of the hunter-gatherers diets has been regained in the Western world.

Despite these micro-ecological problems the Neolithic towns and cities were a success. Median age of death there rose sometimes even over 20 years. The growing complexity of Sumerian towns in the second millennium BC is demonstrated very well by the detailed hygienic regulations regarding water supply and garbage disposal that can be read from cuneiform script on clay tablets, also containing well defined punishments for ancient city dwellers producing too much environmental noise. Archaeologists have demonstrated that these texts resulted in a well-organised urban life. Evidently the advantages more complex sedentary societies were outclassing the disadvantages like the threat of contagious diseases cause of crowding. Of course this holds true as long as a town and its environment were still an islet without much interaction with the outer world. In that case the old threat – uncertainty about the supply of essential goods – had been successfully countered.

1.4 An unhealthy network

The Bronze and Iron Age brought the next health transition. For now germs could arrive from far away. The development of more complex technologies implied the emergence of long trade routes along which tin, copper, lead, iron (ore) and the like were transported. Hand in hand with this development the exchange of production surpluses like grain, salt, furs, silk or pottery arose. Micro-organisms, of course, took their chance and hiked with caravans and ships. One bug could not always cover large distances as the recovery of a sick man or animal would most of the times mean the end of the germ's journey. Consequently, the spread often had the character of a relay race ('estafette'); but in tens or hundreds of years germs could travel far, as far as man. Not surprisingly, it was the time that the great plagues were beginning to spread over large

areas, particularly around he Indian Ocean – in those days the centre of world traffic[21]. Smallpox, cholera, plague and typhus could kill vast masses of people in this opened-up world. In fact, the term 'pandemia' (world wide epidemic) could already be used for these sweeping attacks on mankind in that era.

A truly dangerous era arrives when at the one hand cities become as large as to harbour hundreds of thousands or even more than one million inhabitants and at the other traffic intensifies, goes faster and reaches for the outposts of the earth. This makes the vast population vulnerable to contagious diseases for in the large centres there is a to-ing and fro-ing of pathogenic micro-organisms. Rome is a good example of this turning point in urban history, being a spider in a vast web of land and sea transport ("All roads lead to Rome"). For instance, there was an extended and lively grain trade with Egypt. The unhealthy aspects of the capital amazed contemporary writers like Juvenal, describing elaborately the unhygienic streets, stench and an enormous risk for acquiring diseases. In Rome one lived much shorter than in the rest of Italy. Median age of death was about 5 years.

It took 1500 years to double the world population, from the year 0 until the beginning of the European Renaissance. The 250 million of the time of Caesar only became twofold after many ups and downs in several parts of the world. War, chaos and famine, in their check on population growth, were even surpassed by the great plagues. "From hunger, plague and war, save us, O Lord", medieval man prayed, and he knew that, once, one of the three was around, the other two couldn't be too far away[22]. The epidemic of pestilence of 1346-50, well known as the Black Death, led one third of the European population into the grave (25 million). The disease struck and killed people with terrible swiftness, or as the Italian author Boccaccio phrased it: 'its victims often ate lunch with their friends and had dinner with their ancestors in paradise'. The causal bacterium had been supplied via one of the Silk Routes. Not surprisingly, the first rational effort to control epidemics, the 'quarantaine' (quarantine), originates from these medieval times.

The New Time, from 1500 on, announces the definitive opening up of the whole world into one large global system. The study of the world maps produced from 1450 to 1650 reveals an astonishing and swift filling up of the white spots called 'Terra incognita'. This cartographic aspect is the pendant of a real-life process: an exchange on a never preceded -global- scale. Not only of goods, but also of plants, animals, man himself, and last but not least, their microbial companions; in two directions. Germs from newly opened up areas reached the 'World system', like the cause of syphilis, that was presumably brought by Columbus to the Old World as early as 1493, and soon found its way all over the world - even to Japan - during the 16th century. But the microscopic business presents ('friendship tokens') that the Old World offered the New, dwarfed the horror of syphilis by all means. Smallpox alone was literally decimating the population of Mexico and Peru in the first half of the 16th century. Smallpox, measles, tuberculosis, yellow fever, and many other contagious diseases of the Old World had been unknown in remote areas with populations that were immunologically virginal, like the Americas [v].

The reports of the impact of the incorporation of 'rest areas' into the 'World System' provide depressing reading. One single missionary could be the cause of extermination of the target of his religious zeal. Often he not only forwarded the Word but, for instance, the measles virus as well. This meant a disaster for, among others, Greenland, North Canada and Tierra del Fuego. As late as in the 19th century it could happen that on the Fiji Isles more than 90% of the population succumbed after the introduction of measles.

[v] Earlier export of smallpox to the New World (in the period 1492-1520) did not happen since the crossing took too long for the poxvirus. Even if an explosion of smallpox occurred on a ship, the crew had either died or recovered before landing, and the survivors did not harbor the virus any more.

1.5 A 17th century Health Status and Forecast Report

We are indebted to the 17th century draper John Graunt for thorough statistical insight in the public health of London, during the grim dictatorship of Oliver Cromwell. Fascinated by the weekly appearing London's Bills of Mortality he became one of the world's first demographers by collecting numbers and causes of death to construct the first life tables (see figure 2) . His report reads like a 17th century Public Health Status Report (such as our National Health Status and Forecast Reports: VTV). Half of the Londoners of his times died before their fifteenth birthday, 30% before the age of 5. Only slightly more than 10% lived beyond their fortieth year. Between 10 and 20% of the new-borns died before their first birthday, presumably of infectious disease such as diarrhoea, measles and whooping cough. In two of the 22 years Graunt investigated, plague was the largest cause of death, but most of the time it was outnumbered by 'consumption and cough', the 'pale death' or tuberculosis. On average almost 70% of mortality was due to infectious diseases, including leprosy (6%). Diseases of civilisation, such as cardiovascular disease or cancer were very rare, except perhaps for stroke. However, still 7% of the Londoners survived into old age (> 55 year). Of course these figures are not very accurate. In those days deaths were reported by so-called searchers who only knew a limited number of causes of death. Autopsy or coroner's reports were rare[27].

1.6 Splendid isolation

Remnants of ancient times support the notion that the large cities of post-medieval Europe were a dangerous place to live, at least until the middle of the 19th century. Archaeologists have constructed life tables from skeletons found at burial sites in the Çatal Hüyük in Anatolia, were the earliest sedentary,

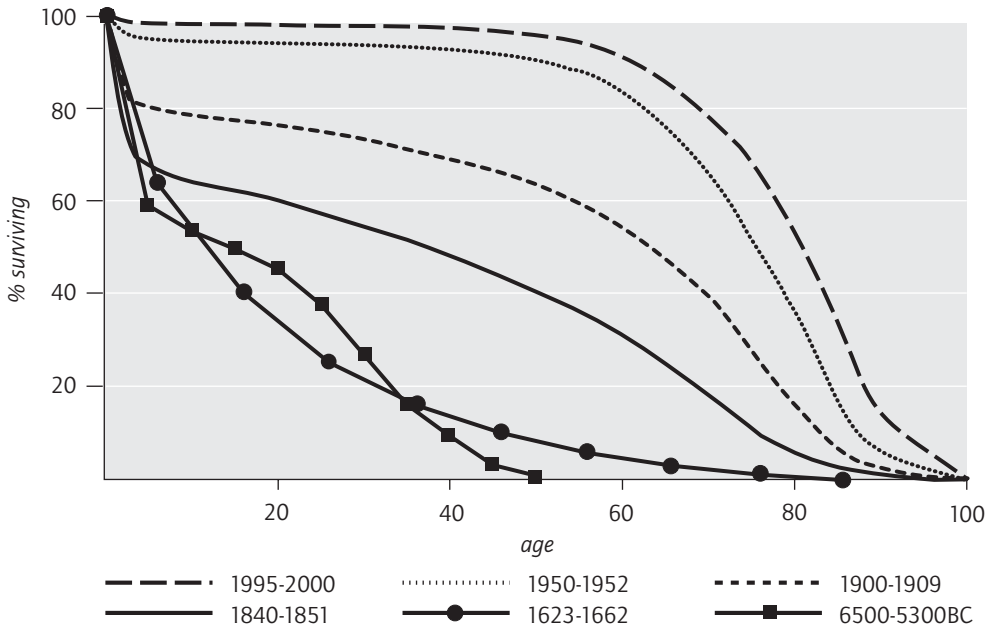

Figure 1-2. Survival curves from the people of Çatal Hüyük in Anatolia (determined from burials between 6500 and 5300 BC), John Graunt's London (1620-1660), and The Netherlands between 1840 and 2000 (adapted from Wills, 1996[27], Ruwaard & Kramers, 1998[2]).

agricultural communities have been found (see *Figure 1-2*). Infant mortality was extremely high, and virtually none of this people made into old age, probably due to severe environmental rigours (or the casting out of the unproductive members). However apart from those most susceptible members of the tribe, the middle years of the people from Çatal Hüyük living 8000 years earlier were considerably less perilous than for the people in Restoration London[24]. Again, the reason for this is clear: the Neolithic 'town and adjacent areas' formed a virtually *closed off* economic unit, whereas the large towns and cities of 200-500 years ago were centres of world traffic *without* adequate public health policy. Such a community, without comprehension of causes and spread of diseases, provides a golden chance to the many germs introduced there. The introduction of cholera in Europe in the thirties of the 19th century illustrates very well the impact of world trade traffic on infectious disease prevalence. Only when the sailing ships became fast enough, especially the tea clippers, the *Vibrio cholerae* could get in our parts of the world with the ballast water, taken on from the brackish estuaries of the great Indian rivers and discharged into the identical environment of the Thames, the Elbe, and the New Waterway[25].

From a demographer's perspective up until the 20th century cities were 'black holes' not capable of sustaining their own populations, due to extremely high mortality rates. Cities not only absorbed the agricultural production of the surrounding rural areas, but their excess births as well. Obviously at the same time cities were the centres of religion, cultural, political and technological development[28,16].

1.7 An end to the Englishman's right to be filthy[vi]

As the cities were connected by trade routes, plagues could spread rapidly across the continents. Plagues swept the 'virgin' population of cities time and time again, especially the young who did not yet developed immunity: smallpox, measles, plague and finally in the 19th century cholera. The variolation of the 18th century and the cowpox vaccination of the 19th century were far more popular in the countryside than in cities (despite the 'conservative' image of the countryside), for in the more isolated areas the victims were relatively often adults. In cities like London, the regular supply of the virus was so efficient that mainly infants and toddlers were affected and often died. That's why in a census not those children were comprised who had not yet acquired smallpox[26].

To really appreciate the public health blessings of the industrial revolution and the economic development it yielded, one should throw a glance at the pre-industrial cities of the 18th century. Large cities, such as London, Paris, Amsterdam had already reached high population numbers and densities, that were able to 'sustain' infectious diseases, such as typhus and tuberculosis[16,27,28,29]. At the same time public hygiene was underdeveloped; there were no facilities for safe drinking water, nor closed sewerage systems. Families lived packed together in houses, sometimes even several families sharing one room. Hygienic conditions offered ample opportunities for infection cycles to be sustained, humans, (domesticated) animals and vermin sharing pathogenic micro flora and vector organisms. Human and animal excrements and waste were dumped in the same canals where people obtained their drinking water. The only defence against infectious disease consisted of people's immune system, which was often weakened by malnutrition and intoxication[30,31,32]. Before the second agrarian revolution in the 19th century the common men's diet was staple-based, monotonous, and thus prone to nutritional deficits, malnutrition and recurring famines. Violence, fires and accidents presented another substantial risk of premature mortality, morbidity and malformation.

[vi] Paraphrasing a 19th century Member of the British Parliament[37]

Compared to our present day societies – striving for zero health risk -, official health protection facilities or measures were scarce in the 18th and 19th century: *fire brigades* were already known in the Roman empire (bands of slaves) and in many cities since medieval times, *fire prevention legislation* for instance regulating the distance between buildings and building materials - no straw and wood - (e.g. implemented after great fires, such as in London in 1666), *lightning conductors* were applied on palaces and official buildings, *light houses* guided ships at sea, societies for *saving drowning*, *shipwrecked sailors* were organised, there was some *road maintenance*, and already soon after the introduction of steam engines the steam boiler inspection ('het stoomwezen, 1855') was installed. Since 1250 in Holland special governmental bodies, de 'waterschappen' were entrusted with water management, including the control over the *dikes* that protected against floods[33]. Apart from the variolation or cowpox vaccination we already mentioned, the *food and ware inspection services* (although predominantly installed to inspect the sincerity rather than the microbiological or chemical quality of trade), *quarantine* for ships with disease aboard and rodent control in the harbour quarters of town were the most significant medical prevention measures of those times.

By the mid-nineteenth century the needs of public hygiene finally burst through. Beginning in Great Britain the tempestuous economic growth of the industrial revolution brought about a tremendous population drift from the countryside to the larger industrial cities. In 1800 London was inhabited by around a million souls, by 1880 that number had swelled to 4.5 million. This caused enormous housing problems, crowding, poverty, the breaking down of traditional ways of living, and pollution of water, soil and air. In short there was urban pauperisation that probably even Dickens could not describe adequately[34]. Industrial cities like Manchester and Liverpool burst from overpopulation[35].

In particular the efforts of the so-called Hygienists (or Sanitarians), an alliance of physicians and civil engineers, made the difference. A series of reports described the abominable hygiene, the moral degeneration and the health consequences for the paupers living in the slums. Not just an act of humanity, one of the key motives of Sir Edwin Chadwick's proposals regarding drinking water services, drainage systems, and the removal of refuse, was increased labour productivity. The median life expectancy among the working class was 15 years. Chadwick calculated that the improvement of housing, drinking water and sewerage services might add another 13 years to the productive life of the average worker (e.g. Chadwick, 1842)[36]. It is almost ironic that Chadwick's health-engineers were proponents of the miasma theory: 'miasma' (Greek for pollution), a noxious form of bad air in filthy environments was the cause of disease. Although John Snow at the end of the 1848-49 cholera-epidemic already suggested in an essay that the disease was caused by material from the excreta of patients contaminating drinking water, the miasma theory dominated public hygiene policy making. Drastic measures were thus proposed in the British Public Health Acts ignoring the germ theory, which is nowadays considered to be the best evidence[37].

Most historians agree that three great cholera pandemics finally defeated the prevailing 'laissez faire' attitude among the ruling class, most of which already had escaped the unhygienic hell into the suburbs. With a centrally controlled network of local boards of health, established under the 1848 Public Health Act, Chadwick and his fellow hygienists eventually addressed the composite problem of economic deprivation, urban squalor, and severe social-economic health-inequalities. The Sanitation Act of 1866 gave sanitary powers to local municipalities and -employing new sanitary technology- drinking water and sewage systems were modernised at a fast pace, and turned from private into public services. Urban development programs improved housing quality, although not always primarily aimed at the lower classes of ('deserving') poor[e.g.38]. The rest of Europe followed, some nations, such as France almost immediately, others only after several decades (e.g. the Netherlands[39]).

1.8 Very healthy people: a triumph over the micro world

The development of survival curves for the Netherlands since the middle of the 19th century show that the Dutch have become a very healthy people, just like the rest of the Western Europeans (*Figure 1-2*). Thanks to public hygiene and the improved quality of houses the scourge of infectious disease has been brought under control. Furthermore vaccination programmes, wide application of antibiotics, health education programmes, the development of effective medical technologies (e.g. insulin for diabetes) and, of course, a general improvement in standards of living have all contributed much to the current improved public health status[40,41]. Especially the 'bourgeois' civilisation offensive at the end of the 19th and the beginning of the 20th century, aimed at young (future) mothers, proved effective, teaching them hygienic housekeeping, and the timely recognition of infant disease symptoms. A comprehensive system of health protection regarding food, drinking water, and consumer safety, labour and environmental conditions has enhanced health, although the improvement is often difficult to quantify (see figure 1-1). In 1850 half of the birth cohort had already deceased at the age of 37 (men 36, female 38). Nowadays at birth we may expect a life span of almost 80 years, of which more than 60 years will be spent in reasonably good health (without overt physical limitations). Of course this a typical picture for modern post-industrialised countries. In many mega-cities in the third world public health transition is still in progress, or in some cases has stagnated[31].

These enormous differences in health status and disease patterns before and after the lengthy struggle that accomplished the health transition are reflected in the public health status of developed Western countries and the developing world. Acute infectious diseases cause most of the disease burden in low-income countries; while in the high-income countries chronic diseases at older ages (cardiopulmonary diseases and cancer) dominate the picture (*Figure 1-3*, and *figure 7-1* in chapter 7)[vii,42,43]. Furthermore the health loss due to exposures from the physical environment is much higher in low-income countries compared with Western countries. This is also reflected in a totally different disease pattern. In low-income, more traditional countries, the environmental problems chiefly concern the lack of access to clean water, and inappropriate housing and are primarily related to infectious diseases, indoor air pollution and malnutrition.

Of course in high-income countries the traditional environmental risks may be in part replaced by risk associated with industrialization, such as large-scale air (and noise) pollution associated with transport and energy production and reduced liveability in urban areas. However, the Danish economist and 'sceptical environmentalist', Lomborg, appears to have a point when he states that environmental pollution is merely a problem of the past, the growing pains of rapid industrialization that have been solved in most post-industrial societies. For instance air pollution levels in London peaked around 1900, but nowadays they are as low as in the 16th century[44]. The fraction of the burden of disease attributed to the environment was recently estimated at 1.5-4.5% for the OECD region and 25-30% for non-OECD (low income) countries[45,46].

So, in Western countries death by infectious diseases has been drastically reduced. In 1850 still 30-40% of the Dutch population died from these illnesses, tuberculosis being the major cause (10-15% of all deaths). Now less than 5% of mortality is due to infectious diseases, a major cause being pneumonia at old age ('poor man's friend'). On a global scale we see a drastic reduction of infectious diseases mortality

[vii] Nevertheless, in the developing world as well the cardiovascular disease burden is already massive. Figure 3 is based on data of 1990; the enormous infectious disease burden among young children in the developing world has already decreased drastically.

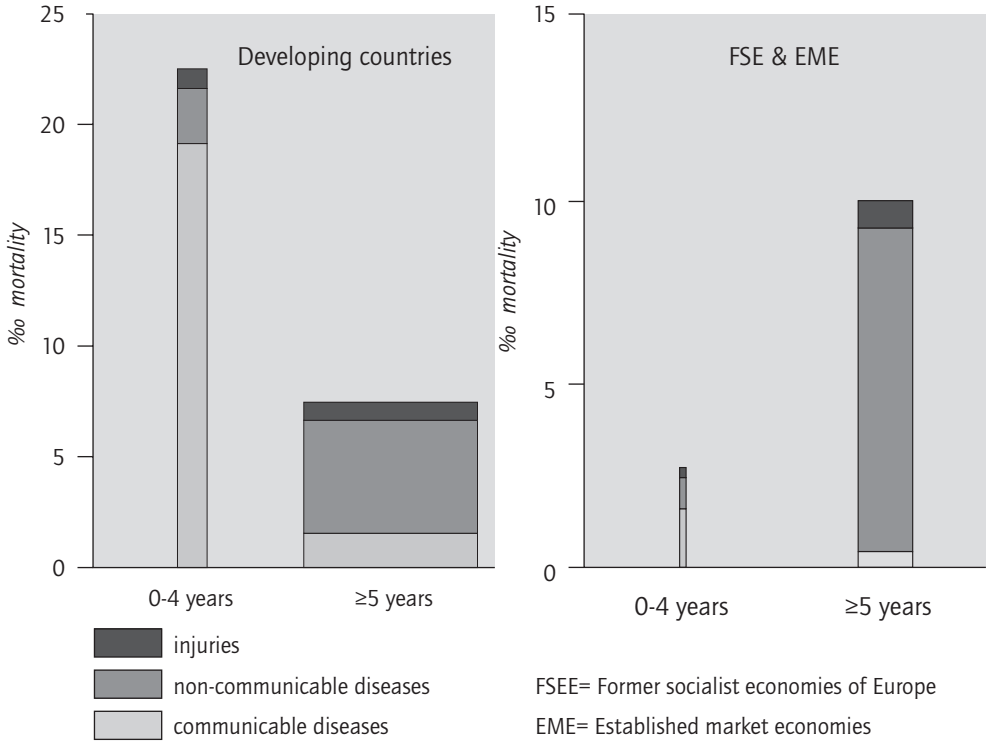

Figure 1-3. Distribution of the 50 million registered global deaths by three causes in 1990. The four bars represent the division by two age classes (0-4 years and >5 years) and two spaces (developing countries and established market economies and former socialist economies). The vertical axis gives mortality in per mill, the horizontal axis corresponds with the size of the population involve. Hence the surfaces of the four bars are proportional to their share in the total global mortality (World Bank, 1993, RIVM, Bol en Niessen, 1997).

as well, despite the stubborn resistance of infectious diseases like malaria and tuberculosis, and the ever-expanding AIDS epidemic. This is the major reason why the global life expectancy is momentarily already over 60 years. In China this is even 70 years, and India is heading for that number too.

It should be noticed that this global distribution of good health has also a dark side: the life support systems of the planet could be endangered by it. Poverty can and does lead to severe environmental damage (slash-and-burn agriculture, overgrazing, overfishing), but the healthy and prosperous can be excessively damaging as well[47]. Within decades adults will not comprise half of mankind, but three quarters[48]. Children *can* be very demanding, adults nearly always *are*. Since we have enlarged our economical systems into all corners and cracks of the planet's life support systems, the automatic fulfilment of all the wishes of the present 6 billion people, and the presumed 8 billion in 2030, should be discussed. The scale and magnitude of man-induced environmental changes, including the global atmosphere and climate, stocks of biodiversity, freshwater supplies, and food producing ecosystems is unprecedented. In other words: recent gains in worldwide public health have depended largely on the depletion of environmental capital[13,48,49].

1.9 Life style and the social environment

In post-industrial society *life-style* is responsible for the largest avoidable health loss. A comprehensive analysis of the Dutch situation shows that a substantial fraction of annual mortality, disease burden and care demand is associated with life style risk factors, such as smoking (15% of mortality), dietary habits, such as high unsaturated fat and deficient fruits and vegetables intake (10 %), sedentary life-styles and lack of physical activity (6%). The same applies to related endogenous factors: obesity (6%) and hypertension (6%) are a huge source of avoidable disease burden. Especially among youth and lower socio-economic classes trends in unhealthy behaviour are alarming[50].

At the ecological level of large populations, equally alarming is the doubling of severe obesity prevalence in the developed world during the pas two decades, especially in the light of the diabetes type 2 epidemic that is building up. These trends may be the result of radical changes of human ecology, such as the sedentary life-styles in our modern highly mechanised, ICT-driven societies and our diets rich in saturated fat, simple sugars and salt, and low in fibre.

A comprehensive body of evidence clearly indicates the key role of the *social environment* in the health status of populations as measured by mortality rate, life expectancy, perceived health, the prevalence of chronic disease, and physical limitations. Even in a relatively egalitarian society such as the Dutch, men from the highest socio-economic groups live around 5 years longer than men from the lowest group, for women this difference is 2.6 years. In terms of healthy life expectancy, e.g. life expectancy without chronic disease, the difference is as high as 10 years. In terms of self-perceived health the difference is even greater, less educated men and women have 15.8 and 14.0 fewer 'healthy' life-years, respectively[50]. Furthermore, geographical differences in health status are highest at the level of residential neighbourhoods, in particular in large cities, again implicating an important role of the social environment.

The social-demographically determined health differences are to some extent explained by an unequal distribution of unfavourable life-styles. Impaired health is often due to combinations of risk behaviour, such as smoking, alcohol abuse, high intake of (saturated) fat, little fruit and vegetables, and lack of physical activity. The interaction between physical and social environment, and life-style has been known longer than we often think. Already in 1842 Chadwick's Poor Law Commissioner's report observes 'that the population so exposed is less susceptible of moral influences, and the effects of education are more transient than with a health population. That these adverse circumstances tend to produce an adult population short-lived, improvident, reckless, and intemperate, and with habitual avidity for sensual gratification....'

The socio-economic gradient is almost universal, and whether one compares countries, neighbourhoods or individuals, the same picture arises. It appears that the absolute income level is much less important than the relative inequity. Recently in the United States Diez Rouz et al. observed a socio-economic gradient in the prevalence of cardiovascular disease in 'whites' as well as in 'blacks'. However at the same income level the cardiovascular risk of the whites was much higher than among blacks, as the 'whites' were at the bottom end of the income distribution, while the 'blacks' were more or less at the top[51]. The famous 'Whitehall' study showed a similar picture[52]. These differences are probably caused by a complex of social-psychological factors, such as social support (networks), employment (and self-esteem), number of life-events (e.g. divorce, loss of friends or relatives), opportunities to escape from a stressful daily existence, the ability of controlling one's own life, job insecurity, or possibilities for consumption, especially compared to others[53,54,55,56,57,58]. In this context unhealthy behaviour may be seen as some form of stress handling. Or in the words of Michael Marmot: 'the mind is a crucial gateway through which social influences affect physiology to cause disease. The mind may work through effects on health-related behaviour, such as

smoking, eating, drinking, physical activity, or risk taking, or it may act through effects on neuro-endocrine or immune mechanisms'[59]. Furthermore, the accessibility and quality of certain care facilities appear to be less favourable among individuals of the lowest social groups[60]. There may also be significant differences in the quality of the occupational (blue-collar labour) and environmental conditions (including noise and air pollution, traffic density and safety, crime rates)[61,62]. Unequal distribution of exogenous determinants across SES-groups is reflected in the distribution of endogenous (or intermediate) factors, such as hypertension, unfavourable blood lipoprotein composition, and obesity[2,50].

1.10 Overlapping and interacting environments: an accumulation of trouble?

1.10.1 Unhealthy neighbourhood

This brings us back from the social to the physical, or rather the neighbourhood environment. It is obvious that there is an important interaction between the physical and social environments, and life-style, as is suggested by socio-economic health differences on the level of neighbourhoods, where often an accumulation of unfavourable social, spatial, environmental factors can be demonstrated[64,65,66]. Recently Bouwman et al. clearly demonstrated an unequal distribution of unfavourable environmental conditions over socio-economic groups on the level of 6-digit postal code ('street corner level', *Figure 1-4*). The analysis involved almost 2.5 million Dutch households[63].

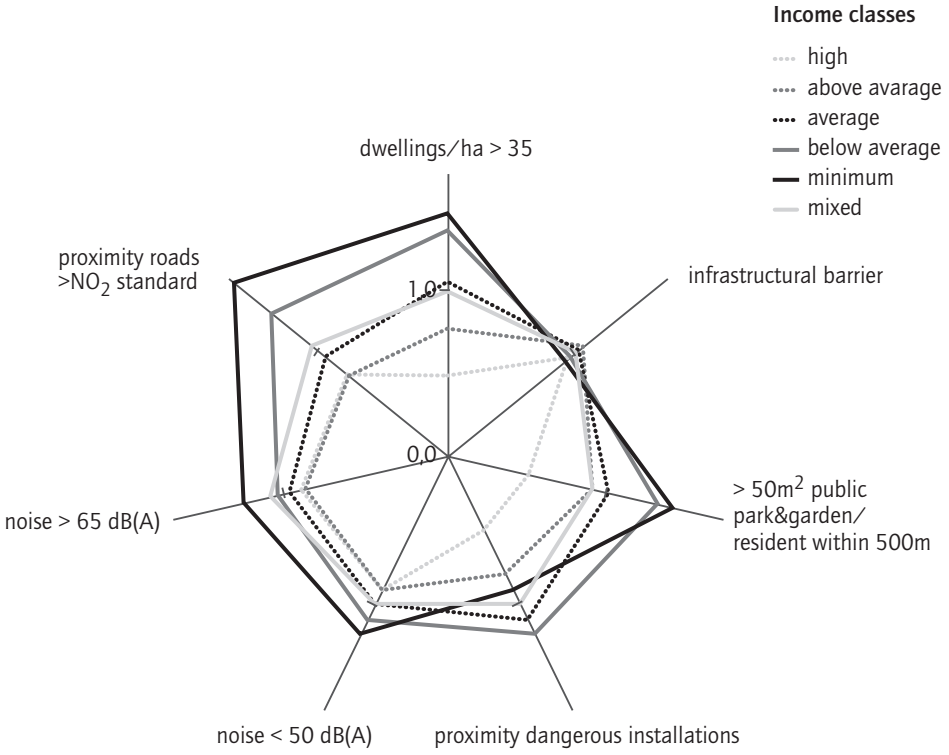

Figure 1-4. Socio-economic distribution of unfavourable environmental factors on the level of neighbourhoods (6-digit postal code); index 1 is the population average (Bouwman et al., 2001[63]).

Aside from causal mechanisms, geographical health differences may at least in part be due to *selection*, as people in poor health are excluded from education and employment. But more significant is selection due to the fact that over recent decades people from higher-income groups have moved out of the older neighbourhoods of cities, and the vicinity of industrial zones to settle down in suburbia and dormitory towns where they found better conditions with respect to housing, working and the quality of the local environment. At the same time the influx of immigrants of lower socio-economic status, was at its peak. Especially these people found housing in the neighbourhoods abandoned by the more prosperous. The more socially disadvantaged groups were left behind, which increased the geographic accumulation of unfavourable social-economic conditions[67]. Living in disadvantaged neighbourhoods may be bad for health because of lack of access to amenities, (healthful foods), to opportunities for physical activity, and to medical and other services. In addition, insecurity, fear of crime, suffering from the effects of a low position in the socio-economic hierarchy, and lack of social support are all features of disadvantaged communities that might increase inequalities in health[59].

1.10.2 International differences

The same mechanisms can be observed on the more or less 'ecological' level of nations, where a certain level of equity, social security and stability seem to play an important role. The plunge of (male) life expectancy with about six years to less than sixty years in the Soviet Union is a good example of the close association between social economics (equity) and health on the 'ecological' level of nations. The regime in Russia has changed from repressive, authoritarian and communist government forcing equity upon the population (social welfare, 'full employment', and a secure old age) to a 'savage' free market economy giving a small minority ample opportunity to profit, leaving a considerable majority of the Russians living below or around the poverty line[13]. Poverty per sé is not at the root of the associations between ill health and low socio-economic status of both individuals and neighbourhoods. Some poor countries appear to achieve good health at low cost. Life expectancy in China, Sri Lanka, and Kerala (India) exceeds 70 years, despite the fact that their gross national product per capita is less than $1,000. Even in the Harlem ghetto a median family income in the nineties was still around $25,000. Poor people in the United States are rich by world standards, but they have worse health than the average in some poor countries[59].

Japan is the current leader in the life-expectancy league, with almost 78 years for men, more than 81 years for women. Between 1955 and 1986 life expectancy in Japan increased enormously 12 and 13 years respectively for men and women, against around 4.5 years in the UK. Since 1977 Japan is outdoing the entire Western world. Individual life-style factors do not appear to be that important, e.g. smoking rates are not particularly low among Japanese men. Neither are genetic factors, as Japanese immigrants in Hawaii and Brazil gradually take over the local morbidity and mortality patterns. Most authors think the 'egalitarian' and stable society the Japanese build after World War II might be an important cause of good public health figures. There is much equity in income and education. There is much mutual respect and loyalty between workers and companies (during recent economic crises executives and managers took cuts in pay rather than lay of employees), and there is social and economic care from cradle to grave, in which the (extended) family plays a dominant role. The significant role of these factors would explain the high ranking of other egalitarian societies such as Canada and Sweden, and the low rank of the United States for that matter[68].

Respect for and a significant position in the social and family hierarchy of people of high age may be another crucial factor as well. He or she who reached 65 must 'fancy' becoming 75; to become 85 one must have a strong motivation, something to get up for in the morning. High mortality rates among the

elderly in France during the heat wave of August 2003 may be an indication of lack of social structure (at least during holidays)[69,70,71].

1.11 Discussion: an environmental health agenda

1.11.1 Changing foes and friends

Figure 1-5 brings together the several aspects that determine health, disease and death in a diagram displaying a notion of the Dutch situation from 1800 until 2000. In these two centuries life expectancy has been expanding from less than 35 years to more than 75 years. Nevertheless the total overall disease burden (in absolute DALYs) has been more than halved in that period, mainly due to a drastic reduction in environmental pressure. Many of the impacts of environmental factors have been annihilated or reduced to be 1/10 to 1/20 of their original impact[viii]. This shows how massive and effective the communal efforts to ward off health risks have been. Collective (cultural) life style is reckoned to be part of the environment.

In contrast, individual life style is now having a far larger impact than 200 years ago: then, for most people, there was little to choose. Also many effects of life style are only expressed at riper age, so nowadays a much larger percentage of the population is suffering from them.

The influence of genetic factors has been steadily growing in the last 200 years, despite the fact that the genetic make up has not changed much. Many diseases that are (partly) genetically determined only express themselves at advanced age, like diabetes type 2 or the impacts of hypercholesterolaemia (both closely linked to family history).

Another contrast with 1800 is the present compression of disease within the years over 60, about 15 years for men and 20 for women. So, the diminished disease toll is suffered mainly by the elderly, whose last years resemble the condition many children en younger adults were experiencing around 1800.

We conclude that until the 20th century the *physical* environment was the largest source of avoidable disease burden, with attributable fractions in the order of 70 to 80 percent. Thanks to the public health revolution nowadays these factors are only responsible for minor health loss: probably less than 5%[72], while *life-style* is responsible for the bulk of the current avoidable disease burden (25-30% of total disease burden)[73]. The next substantial improvement of public health (transition) will probably be found in knowledge of and control over the *endogenous determinants*, genetic and acquired, at the individual level. In the near future this will primarily concern early detection of disease susceptibility, enabling medical doctors to give life-style advice or medication tailored to the genetic characteristics of the individual patient. Life-style drugs, such as 'statines', may be able to compensate the effects of poor life-style. Implementation of effective gene therapy technology will most likely take another couple of decades, with a restricted impact since the large disease entities have no single gene defect as a cause[74,75,76,77].

1.11.2 From quantity to quality of life

Public health focus has gradually shifted from life expectancy to health expectancy. Or in other words: postponing as long as possible or mitigating the physical, mental or social limitations brought about by

[viii] It is difficult to obtain reliable morbidity data for the 19th century, but mortality shows the same trends. For example, contagious diseases caused 30-40% of the mortality in 1800, but only 1-2% in 2000. Tuberculosis killed one in seven citizens in the 18 hundreds, now only one on 10,000 becomes ill with tuberculosis per year, and only a small fraction of these patients die. Unintentional drowning took the lives of more than 20 times as many people per 1 million in 1900, compared with 2000.

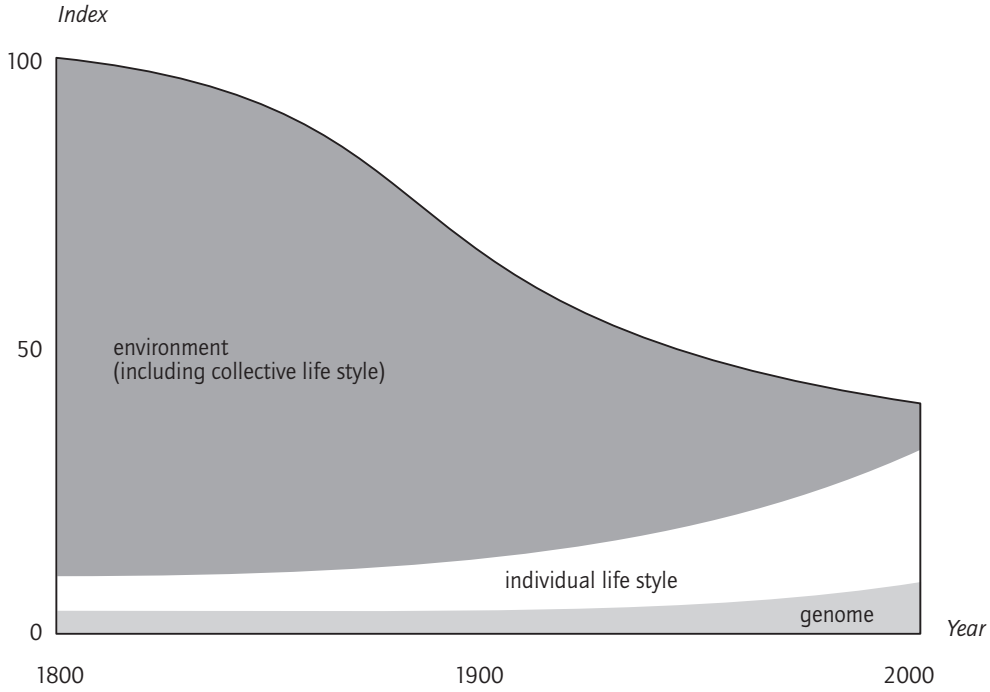

Figure 1-5. Diagram of life time disease burden (in DALYs) in The Netherlands during the last 200 years, distributed by three main categories of causes: environment, individual life style and genome. The index is 100 for 1800.

the chronic diseases of older age ('not adding years to life, but life to the years')[78,79,50]. In the framework of environmental health impact assessment a similar situation has arisen. Health impact no longer predominantly involves clear mortality risks or loss of life expectancy, but rather comprises aspects of the quality of life in a broad sense, such as:

aggravation of pre-existing disease symptoms, e.g. asthma, chronic bronchitis, cardiovascular disease or psychological disorders

severe annoyance, sleep disturbance, as well as a reduced ability to concentrate, communicate or perform normal daily tasks

feelings of insecurity or alienation, unfavourable health perception and stress in relation to poor quality of the local environment and perceived danger of large fatal accidents.

This shift to attention for the quality of life both in policy-making and public opinion is in agreement with Maslow's theory, which postulates that human needs are organised in a hierarchic fashion[14]. When primary needs such as food, shelter and security needs are fulfilled, social and ego needs become more salient. The same can be observed with regard to environmental quality. Since most physical and chemical dangers have been brought under control, environmental quality may now be looked upon as the extent to which the environment fulfils the social needs of communities and individuals[14].

1.11.3 Impact assessment: from 'health' to 'well-being'?

In the Western world the degradation of the environment associated with industrialisation has been brought under control for the greater part. Environmental problems predominantly persist on the highest

and the lowest scale: 'global warming, ozone depletion versus air and noise pollution, industrial safety and issues related to the liveability of urban areas[80]. A clustering of social and health problems at a local level can be observed, mainly in deprived neighbourhoods with an accumulation of unfavourable environmental, spatial and social quality. There is a call for (further) development of healthy environments with a high living quality.

The question is where we should draw the line. It is not possible to create an environment that fulfils all community needs. For this reason we suggest a split approach. We should define minimal standards for a healthy and safe environment. In addition, we should try to derive definitions for a high living quality, at the same time realising that this should be considered as a maximum quality to be aspired. The challenge for researchers is how to improve the current models. How can we combine physical, social and spatial aspects to predict and evaluate the (perceived) quality of the environment? What are the characteristics of vital, attractive, stimulating living-environments? What is the effect of spatial planning on liveability? Can we translate this into conditions and criteria for the design of new urban areas? And how can we monitor whether these environments fulfil these conditions?

We propose a tiered approach, combining the following components:

An analysis of ' high-risk' areas, combining data on spatial, environmental and social qualities on a small-area scale by using GIS, resulting in maps of cumulative exposures at a small area level.

A study of the geographical distribution of the cumulative health impact of environmental quality using aggregated measures, such as DALYs (see figure 2-5, chapter 2). In addition, aggregate indicators can be applied to explore the health score of different options in the planning of extensive infrastructure projects involving a range of exposures.

Evaluation of the perception of local communities looking at psychosocial responses such as residential satisfaction, annoyance and risk perception.

A comparison of areas that differ in health status or residential satisfaction. What are the main determinants and driving forces behind these differences?

The challenge is how to combine and weigh these different aspects in order to enhance the development of healthy environments with a high living quality. The success of this approach, however, will depend on the collaboration between planners, health and environmental experts, policy-makers and the community. In this, we can learn from the successes of the hygienists in the 19th century.[81]

1.11.4 The micro world revisited

Will (re-)emerging infectious disease become the health issue of the 21st century? In the next few decades micro-organisms and infectious diseases will ask much attention from health authorities, scientists and politicians. Aids, tuberculosis and malaria are examples of expanding problems. Nevertheless, morbidity and mortality by infectious diseases is in a steep descent. This is mirrored by a fast increase of life expectancy in most developing countries. The 'opening up' of the world that has been virtually completed by now, will continue to cause acute dangers in the form of diseases popping up in unsuspected places. Also new diseases like SARS and 'chicken flu' will emerge. But the experiences of the last decades show that we can react adequately. Extensive cattle breeding forms a potential hazard for infections among animals but also from animals to man; our ways of growing animals and plants should be reconsidered. The chance that biomedical laboratories will haphazardly introduce dangerous new germs seems small, at the moment. A much larger threat is put by eco- en bioterrorism. Scientifically there is no threshold any more for fundamentalist groups to develop biological and nuclear weapons. It is an important task for politicians in the coming decades to keep dream and act separated in this respect[26].

1.11.5 This book

Based on this historical analysis we will take a closer look at relevant aspects of the impact on public health of the environmental conditions of today. In the next chapter we outline a framework for health impact assessment, especially geared to large infrastructure projects, such as the expansion of the national airport 'Schiphol'. This is followed by a discussion in chapter 3 on dealing with environmental health risks in a 'sensible' way, an effort to 'encapsulate' comprehensive, sometimes even emotional discussions in support of the current policy program to 'rationalise' risk management procedures. The last four chapters describe a number of exercises in which we investigate the application of aggregate health impact measures for comparative health impact assessment, comprising Deaths (numbers), DALYs (disability adjusted life-years) and Dollars (monetarisation of health loss).

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2 A framework for assessing the significance of health impacts of environmental exposures[i]

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Abstract

In this chapter we present a framework for the assessment of the public health impacts associated with environmental exposures, for instance in connection with the operation of large airports or road traffic infrastructure. First we discuss concepts of health, in particular with respect to where we draw the line as to what constitutes a 'health' effect. Based on a conceptual model we outline the interacting determinants of health, and the relevance of the physical environment. In the fourth section we classify the manifold and multiform health responses that are attributable to environmental exposures, while presenting a conceptual chain of events leading from exposure to manifest adverse health effects. These concepts and definitions may be useful in the evaluation of the public health significance of health responses that can be attributed to environmental exposures.

[i] Based on contributions to the report of the *Health Council of the Netherlands: Committee on the health impact of large airports. Public Health Impact of Large Airports. The Hague: HCN, 1999;1999/14, Oers JAM van (Eds.). Health on course? National Public Health Status and Forecast Report 2002. Houten: Bohm Stafleu Van Loghum, 2003 and Kamp I van, Leidelmeijer K, Marsman G, de Hollander AEM. Urban environmental quality and human well-being. Towards a conceptual framework and demarcation of concepts: a literature study. Landscape Urban Planning 2003; 65: 5-18.*

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2.1 Introduction

The physical planning of large infrastructure projects with sizeable environmental impacts, such as the construction of a new railway connection, the rearrangement of a road network, or the extension of an airport, increasingly provokes public and political controversy. Several arguments and positions shape the debate, such as discontent with the way society is developing in general, obvious unequal distribution of risks and benefits (inequity), and concern about unfavourable impacts on public health associated with the new environmental conditions (e.g. with respect to pollution, noise, safety)[1,2,3]. The latter often dominates the public and political debate.

To facilitate the decision process with respect to strategic societal developments with substantial environmental impacts, such as the future of the Dutch aviation infrastructure often renowned, independent institutions, such as the National Institute of Public Health and the Environment (RIVM) or the Health Council of the Netherlands are requested to evaluate the state of science with respect to associated public health impacts. Several assumptions favour such a role of scientific counsel in the area of public health, e.g.[4]:

health is one of the most important human assets and is therefore nearly inviolable[5]

health can be clearly defined

health science is objective, more or less free of social values[3,6].

All of these assumptions need to be put in perspective. Health is not *an isolated* entity. As we have seen in chapter 1 the concept is very much tied up with the quality of the physical and social-cultural environment (e.g. educational status, wealth). Philosophers throughout history have emphasised the *comprehensiveness* and *complexity* of the concept of health[7,8,9,iii]. Nevertheless several attempts to make the concept of health operational have been proposed in the literature, all requiring *value-laden choices* to be made. Furthermore, there is much *uncertainty* with respect to the (quantitative) relation between environmental quality and (aspects of) health, leaving ample room for interpretation. Describing this relation, either qualitatively or quantitatively, requires many assumptions to be made, which once more may be influenced by personal or *social values*[6].

In this chapter we present a framework to be used in the assessment of the public health impacts associated with environmental exposures, especially as a result of physical planning, for instance with regard to the operation of (large) airports or traffic infrastructure. First we discuss *concepts of health*, in particular with respect to where we draw the line as to what constitutes a health effect. Based on a conceptual model we outline the interacting determinants of health, and the *relevance of the physical environment*[10,11]. In the fourth section we classify the manifold and multiform health responses that are attributable to environmental exposures, while presenting a *'conceptual' chain* of events leading from exposure to manifest adverse health effects. These concepts and definitions may be used in the evaluation of the public health significance of health responses that are attributed to environmental exposures and discuss the implication of various choices.

2.2 Health

What is health? That is the key question in any attempt to evaluate health impacts associated with environmental exposures? The concept of health may differ from era to era, from region to region, since it

[iii] E.g. 'Non est vivere, sed valere est' (life is not just being alive, but being well, Martial, AD 40-103).

reflects changes or differences in social and cultural beliefs, in medical technology, and socio-economic conditions[10].

2.2.1 Health as successful adaptation

Several authors conceptualise health as an *optimum dynamic equilibrium* between individual capabilities and exogenous circumstances, enabling individuals to deal with external disturbances and pressures[7,10,12]. In such an approach health is looked upon as an individual's ability to cope with the demands of everyday life[13,14,15,16]. Successful adaptation to environmental circumstances in the broadest sense implies living an independent and productive life, maintaining optimal economic conditions and social interactions in all stages of life, and thus may very well include well-adjusted people with physical handicaps[17]. Or as Florence Nightingale already put it in 1893: "health is not only to be well, but to use well every power we have". In the context of environmental health impact assessment health problems may arise among those who lack the mental and physical resources to adapt to certain exposures, such as noise, air pollution, lack of open space, traffic density or the threat of a large accident[18,19].

2.2.2 Health equals happiness?

Right after the Second World War in 1946 the founding charter of the World Health Organisation stated that health is 'a state of complete physical, mental and social well-being and not merely the absence of disease and infirmity'[20]. An important merit of this definition is the explicit appreciation of the subjective experience of health, the inclusion of psychological and social dimensions. A disadvantage is its shortage of operational value or as Richard Doll put it: 'this definition is a fine and inspiring concept and its pursuit guarantees health professionals unlimited opportunities for work in the future, but it is not of much practical use'[21]. More recently Saracci argued that 'a state of complete physical, mental and social well-being corresponds much closer to *happiness* than to health'. The consequence of not clearly distinguishing 'health' from 'happiness' is that any disturbance to well being, however minimal, can be regarded as a health problem. As the quest for happiness is essentially boundless, the quest for health also becomes boundless as well. He proposed to view health as 'a condition of well being free of disease and infirmity and a basic and universal human right', but at the same time to link the concept to appropriate indicators of mortality, morbidity and (health-related) quality of life[22].

2.2.3 Healthy until proven otherwise?

In its report on Health Care the Dutch 'Scientific Council for Governmental Policy' (WRR) advocated a more or less similar position. Well-being and coping with everyday life requires much more than good health alone. It is not very likely that the contribution of public health policy would be the most efficient in such a broad field. The Council proposes to limit the definition of health even more: 'the absence of disease and other health problems of a physical or psychological nature'[23]. Of course, these views bear first and foremost on controlling the costs of health care and cure in ageing populations. However, in the field of environmental health protection one is confronted with similar needs for effective and efficient allocation of resources (including opportunity costs)[2,24].

2.2.4 Everything is a market and health is no exception

Taking this one step further we approach the adage of an old bureaucrat: 'there are no *policy* issues, only *resource* issues'. There is always a trade-off between the collective benefit and the collective 'pain' associated with (health) policy measures. What are the expected public health returns (risk reduction) and

what are the costs, including opportunity costs (such as money that cannot be spent on other issues cherished by society)? At the present juncture this motto is often interpreted as: 'are we not wasting the tax payer's money?' or 'are these policy measures really suitable (grasping the notion of 'efficiency' as well)?'[25,26]. A shift has been made towards policies based on 'utility', putting the money where public health profits most. This is not without significance as environmental quality standards traditionally stand for *equity*, the right to protection from adverse effects for everybody, regardless of age, health, and susceptibility (right-based decision rules, see chapter 3)[27].

To assess the loss of *economic utility* associated with the health end-points involved, one would require an economic valuation of the full quality of life impact to the affected individual. This would include expenses such as medical costs and lost income (often referred to as cost of illness, COI), and less tangible effects on well being such as pain, discomfort and restriction of everyday activities. The definition of health is even more crucial here. We will discuss a more utilitarian approach towards health impact assessment and evaluation, including equity aspects in more detail in chapters 3 and 5 on 'health risk management' and 'economic valuation of environmental health end-points'.

2.2.5 Health: just an ICD-code away

These points of view bring us back to a more or less conservative approach in which people are considered healthy until they are diagnosed not to be so (preferably by a medical doctor). The health status of specific (exposed) populations can only be determined by means of a range of comparative population measures of mortality, morbidity, and impairment. One might rely on routinely available health statistics, such as mortality rates or the use of health services, or one might conduct surveys to reveal specific exposure-attributable response variables, such as lung function measurements or specific reported symptoms. But once again one is confronted with the difficulty of defining 'disease' at the margins: can a clear distinction be made between clinical depression and anxiety on the one hand and anger, annoyance, irritation, or loss of morale on the other[18]?

2.2.6 Measuring health

Conceptualisations of health can also be found, implicitly as well as explicitly, in the many recent papers on health status measurements[13,16]. Initially clinicians and clinical psychologists developed these health status measurement instruments to assess and compare quality of life after different options for medical intervention (e.g. quality adjusted life years: QALY's). In recent decades these instruments were adapted to measure 'disease burden' on the level of populations, primarily to support the planning of public health programs (e.g. to assess the efficiency of different options)[13,16].

The most straightforward health status measurement approach is probably the International Classification of Functioning, Disability and Health (ICF, formerly known as Impairments, Disabilities and Handicaps: ICIDH). In the old concept impairment were defined as *any loss or abnormality of psychological, physiological or anatomical structure or function (organ level)*. Impairment may lead to disability defined as *any restriction or lack of ability to perform an activity in the manner or within the range considered normal* for human beings (individual level). Disability may lead to handicap, defined as *any disadvantage that limits or prevents an individual's fulfilment of a role that is normal*, given age, gender, environmental conditions and social-cultural context[28]. It is important to note that the degree to which a disability becomes a disadvantage depends also on the societal response, e.g. the 'conviction' one is unable to work properly or social isolation amongst mentally retarded persons. The new concept of ICF has moved away from 'a consequence of disease' classification to a more positive focus on 'consequences

of health' classification. It distinguishes three components: the *body* (physiology, psychology), *activities and participation* and the *context* (personal and environmental factors)[29].

The slightly divergent framework of health-related quality of life (HRQoL) encompasses a broad range of health metrics. On the highest level one can distinguish broad concepts such as *opportunity, health perception,* and *functional status*[30]. 'Opportunity' comprises issues such as cultural and socio-economic disadvantages, or loss of resilience. Health perception relates to expectations about health (and health care) and satisfaction, and of course often reflects the cultural images of health[7]. Functional status includes physical, psychological and social functioning, for instance the (dis)ability to perform 'activities of everyday life' in four domains: procreation, occupation, education and recreation, as applied by Murray and Lopez in their first Global Burden of Disease report[5].

A common feature of all HRQoL measurement instruments is their multidimensional nature. Pain and anxiety can only be perceived by the individual, while for instance cognitive or affective disorder can only be experienced by an observer. Blindness or limpness may be experienced by both. Of course *self-reports* and *observations* may diverge substantially, as self-reports will be influenced by socio-economic status, level of health care, base line health status, health culture etc[13,31]. EuroQOL, one of several well-studied examples of an instrument to measure HRQoL[32], rates health states employing a 3-point scale for 5 health attributes: mobility, self-care, daily activities, pain/discomfort, and anxiety/depression. Sometimes another health attribute is added: cognitive function[33].

A third, merely utilitarian perspective on health measurement defines loss of health as loss of the individual's utility. In most cases this boils down to measuring preferences with respect to time spent in a certain health state, as compared to complete death (0) or perfect health (1)[34]. Several techniques are available to measure these preferences, such as time trade-off, person trade-off, standard gambling or rating scales[35,36]. Again one has to solve an important dilemma with respect to the perspective one wants to take: who's preference should be measured: the general public, health care providers, individuals in certain health states, or their family and friends[13,37].

2.2.7 Health in the environmental context

As we have seen in Chapter 1 nearly two centuries of successful public health policy and preventive medicine have stretched life expectancy in Western societies substantially, some would say almost to the limit[12]. Subsequently, the marginal yields of environmental policies have decreased as well, at least compared to for instance health policies aimed at life-style or the social environment (in some presentations we provocatively used the phrase 'polishing of the pushpins'[iv] for some environmental policies). Some scientists even claim our thoroughly cleaned environment is depriving the developing immune system of stimuli, causing unbalance and thus giving rise to new epidemics of allergy and autoimmune diseases, especially in the well-developed countries[38,39].

Consequently public health focus has gradually shifted from life expectancy to health expectancy. Or in other words: postponing as long as possible or mitigating the physical, mental or social limitations brought about by the chronic diseases of older age[10,40,41]. In the framework of environmental health impact assessment a similar situation has developed. Infectious diseases, the deleterious occupational and environmental conditions of the past have largely been brought under control in the epidemiological

[iv] Famous expression of ex-minister Hans Gruyters of housing when he was interrogated during the parliamentary investigation of the governmental house building (subsidy) policies of the sixties and seventies, meaning being especially busy with futile matters.

transition, at least in most Western countries[42]. As a result health impacts no longer predominantly involve clear mortality risks or loss of life expectancy, but rather aspects of the quality of life in a broad sense. Important examples of these aspects in the framework of this report are:

aggravation of pre-existing disease symptoms, e.g. asthma, chronic bronchitis, cardiovascular or psychological disorders

severe annoyance, sleep disturbance, as well as a reduced ability to concentrate, communicate or perform normal daily tasks

feelings of insecurity or alienation, unfavourable health perception and stress in relation to poor quality of the local environment and perceived danger of large fatal accidents[73].

Not all of these responses clearly fall within the strictest definitions of health discussed in the former sections.

Including indicators of social response (social well-being), such as the latter category, in the definition of health impact one has to take account of a number of dilemmas.

'Health' has become one of the most treasured goods in Western societies, almost synonymous to 'good'[7]. Some authors have pointed towards the tendency to put social problems into a health context ('medicalisation') bringing about the obvious advantage that in general one doesn't negotiate public health or only at a very high price[13,43,44].

There are several indications that people with higher education and income are inclined to accredit lower scores to subjective topics such as environmental quality or their own health, and display more readiness to complain than people with a lower socio-economic status[9,45]. Of course this necessitates cautiousness with respect to the application of such indicators in decision-making[46,v].

Finally, 'medicalisation' of everyday life problems by itself might just as well erode people's basic trust in their own health, which in turn may affect their well-being.

Following the reasoning implicated by the different concepts of health discussed above we propose that 'health' impact assessment of environmental exposures should focus primarily on outcomes that are significant to mortality, morbidity and health-related quality of life[vi]. Obviously this includes intermediate risk indicators, such as lung function deficits, hypertension, cardiac arrhythmia, aggravation of asthma, or sleeping problems, as well as 'derivative' indicators such as use of medical services or self-medication.

However, while analysing the health impacts of environmental exposures we think evidence of *indirect* health outcomes of environmental exposures should be included explicitly, for instance through prolonged stress or worry as a result of exposure to high levels of noise in a deprived environment. Several authors have suggested that 'physical' health responses may be mediated through social responses, such as 'stress'[vii] or severe annoyance. Indirect behavioural response may comprise social isolation, aggression as well as excessive use of alcohol, tobacco, drugs or food[18,47]. Stress-related physiological responses may

[v] For more or less the same kind of reasons Christopher Murray explicitly excluded self-perceived or self-rated health as an indicator of morbidity in his Global Burden of Disease project. Subjective health is pretty much determined by cultural and socio-economic background. Therefore this type of health indicators may be inadequate to support decision-making with respect to allocation of resources, at least from the perspective of equity in health. He referred to an Australian survey in which aboriginal people in general experienced a better health than Caucasian Australians, while objective health indicators, such as mortality rate, definitely revealed the opposite. Based on perceived health indicators alone one would reallocate health services resources from Aboriginal to white Australian communities.

[vi] 'Preferably ICD or DSM-coded', as it were.

[vii] Defined here as an unbalance between the individual and his or her environment that may occur when one is faced with demands, which seem unable to meet, or which can only be met through extreme effort.

include hypertension, unfavourable blood lipoprotein composition, and cardiac arrhythmia[48]. It has to be noticed that the evidence for such stress-mediated physiological responses is still rather inconclusive. In particular, measurement and adjustment for important confounding variables are far from adequate in most studies[47,49,50,51].

There are several indications that social responses to environmental interventions, such as the expansion of an airport may lead to an increase of medical consumption, such as medication use, GP-visits or hospital admission[52,53,54]. Furthermore, several authors have pointed out the important role of socio-economic inequalities and social position within societies with regard to public health status. Independent of the absolute level of income, material insecurity, social exclusion, lack of self-esteem, loss of social cohesion may lead to a higher prevalence of health problems among the more deprived[55,56,57,58].

In conclusion, for the sake of clarity we propose to distinguish (at least) two types of end-points in the impact assessment of environmental exposures: *psychosocial* effects, such as disturbance of performance, risk perception and *clinical* end-points that can be linked to diseases in a strict sense (e.g. by an ICD-code). This doesn't imply that the one is worse than the other.

2.3 Determinants of health

2.3.1 Complex relations and interactions: a model

To evaluate the manner in which environmental exposure may influence public health we consider a conceptual model that was developed in the framework of the Public Health Status and Forecasts (PHSF) report issued by the RIVM (see *Figure 2-1*)[viii]. The model illustrates that health status can be contemplated as a function of many (interacting) determinants (a complex of causality), including the quality of the physical environment[ix]. Endogenous as well as exogenous determinants are involved, which may explain why the response to environmental exposures may vary substantially from one individual to the other[21,52,59].

Endogenous determinants may be *genetic* or *acquired* in the course of life. *Genetic* predisposition may involve clear abnormalities such as haemophilia or colour blindness. However often a particular feature in a population may show a more complex genetically determined distribution, reflecting differences in susceptibility to pathogenic factors. Examples are variations in the ability to detoxify harmful substances, susceptibility to carcinogenic substances, or skin pigmentation in connection with damage caused by UV radiation (genetic polymorphism). Probably most endogenous determinants develop through interactions between genes and environmental factors and thus have both a genetic and an acquired component[x], for example length, blood pressure, blood lipoprotein composition (familial risk factors), DNA-repair, and personal (psychological) attributes[60,61,62,63,64].

Acquired attributes are built up in the course of life, for instance immunity acquired through vaccination or prior infection, reduced lung function as a result of an earlier respiratory infection, many years of smoking or adverse occupational exposures. In the context of airports earlier experiences with

[viii] The model elaborates on previously published models, in particular the model proposed by the Canadian Minister of Health Lalonde in 1974.

[ix] Furthermore it shows health status may provide directions to health policy, which will in turn influence health status via determinants. This whole dynamic process is influenced by autonomous developments in demographic, socio-cultural, economic and technological areas.

[x] 'Gene-environment interaction' is a (new) hype in health science nowadays.

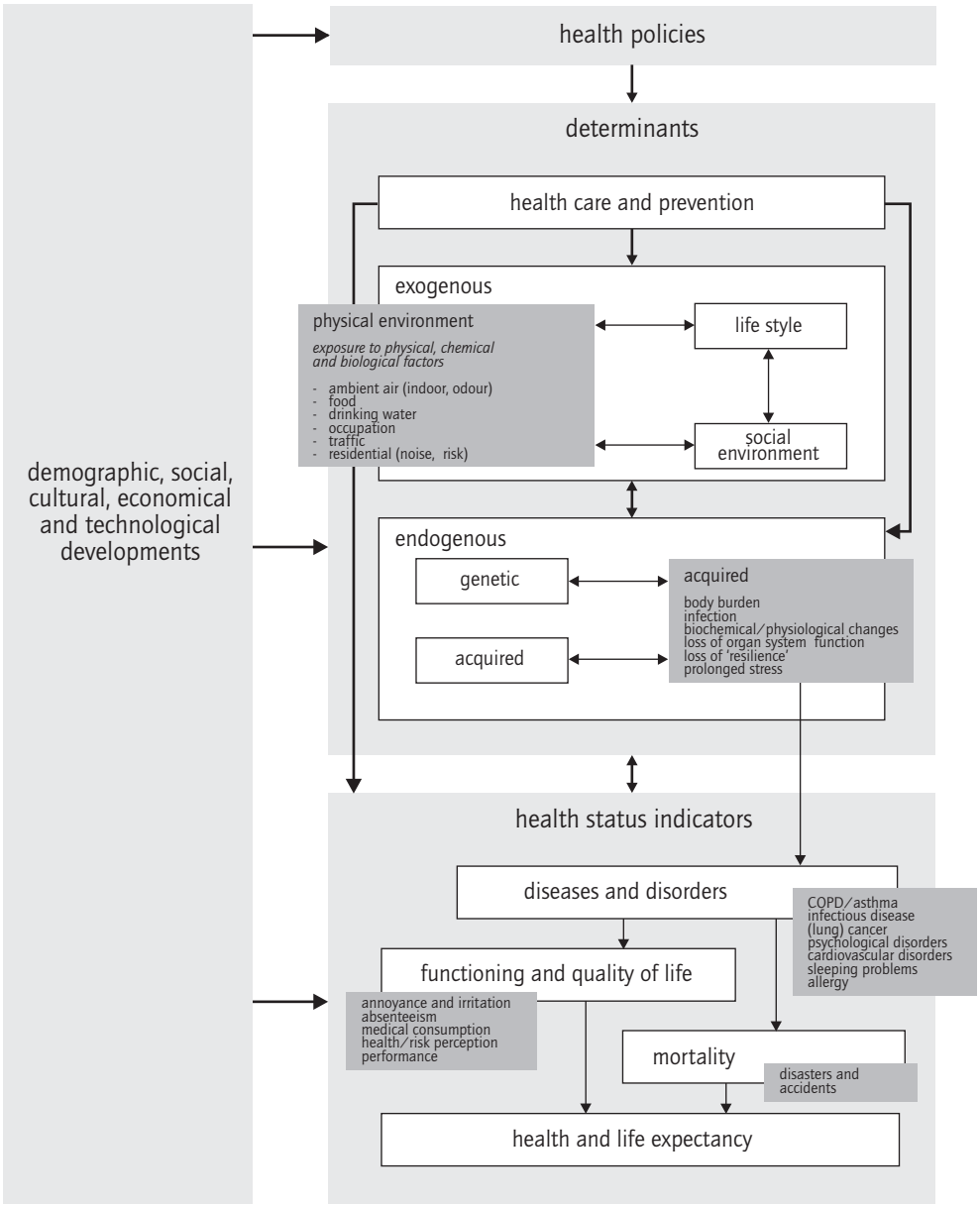

Figure 2-1. The physical environment as a determinant of health status, elaborated in the conceptual model underlying the Public Health Status and Forecasts Reports[10].

perilous environmental accidents may increase one's susceptibility to anxiety or annoyance. Of course *age* (and the ageing processes associated with it) is an important endogenous 'acquired' determinant of health. Inevitably our chances of suffering from chronic diseases and associated functional limitations increase, as we grow older. Some theories assume we are accumulating adverse physiological events during our life, such as DNA-deficiencies or tissue damage, others claim our physical and mental decline is

programmed, for instance as a result of some evolutionary process[65]. A person's 'resilience' with regard to the effect of exogenous determinants alters with age. Many health problems occur later on in life, often in connection with the effect of exogenous determinants earlier in life.

With respect to exogenous determinants a distinction is made between the physical environment, life-style factors and the social environment. The *physical environment* includes radiation, noise and heat (physical factors); oxygen supply, nutrients, hazardous substances in the outdoor and indoor environment, including the working environment (e.g. chemicals); bacteria, viruses and other (micro)organisms which may have both positive as well as negative effects on health status (biological factors). *Life-style* factors include diet, smoking, drug abuse, sexual behaviour, physical (in)activity and such. The *social environment* includes the pattern of social networks and socio-economic status.

Exogenous determinants act on endogenous ones. Behind this lies the concept of health as a dynamic equilibrium (interaction), in which a particular state of health is the result of the interaction between exogenous and endogenous factors. There are many interactions within the group of exogenous determinants. Life-style, for instance, is to a considerable extent determined by social environment (e.g. family situation). Aspects of lifestyle or behaviour, such as sunbathing, smoking and personal hygiene may largely determine exposure to factors from the physical environment such as UV radiation, carcinogenic substances, or pathogenic organisms.

2.3.2 'Noisy' interactions

In the context of environmental health impact assessment the complexity outlined here is well demonstrated by diverging responses to exposure to aircraft noise. In addition to the level of noise per se the extent of stress and annoyance will be influenced by individual characteristics. Some people manifest a proactive, instrumental coping strategy towards the sources of stress (aimed at solving the problem, or seeking social support), other react in more defensive way by trivialising or denying the source. The latter, more 'palliative' coping strategy -seeking distraction in other matters- may in the end worsen one's health risk profile in an indirect manner as comfort is sought in 'bad habits' like smoking and drinking[18]. Regardless to what extent these are genetically determined or acquired, both coping strategies may lead to *social* or *health* impacts of different severity (e.g. the extent of annoyance) .

Situational and *attitudinal* factors, such as perceived personal or societal benefits of noise generating activity, the perception of risk (an aircraft crash, cancer, immune disorders), and the perceived control over the source play an important role as well with respect to the degree of annoyance reported. Interference with activities requiring concentration is another factor increasing reported noise annoyance and anxiety. Furthermore, a distinct subgroup of people who report themselves to be 'noise sensitive' are indeed more likely to indicate intense annoyance reactions[18]. However the associations found in a sizeable series of social studies between noise annoyance on the one hand and age, gender, social-economic variables (including residence ownership), time-activity pattern and background noise levels are not at all unequivocal[67].

In a large residential survey around London's Heathrow Airport a significant association was found between the level of *noise annoyance* and reported *symptoms*, such as waking, depression, irritability, minor accidents, chronic tinnitus (buzzing in one's ear) and health service use, *irrespective* of the actual level of noise exposure[68,69]. Further analysis showed that psychiatric patients were disproportionately annoyed, indicating that the aircraft noise may affect these people more severely than the general community[70]. In several other studies similar associations were seen between reported symptoms on the one hand and noise annoyance or related personal attitudes on the other, independent of actual noise

exposure[18]. The sensorial perception of air pollution through smell, such as the scent of kerosene, may enhance public concern about health risks substantially. Studies on drinking-water pollution incidents show that detection by smell increases the number of reported symptoms and the use of health services significantly, regardless of the harmfulness of the chemicals[71].

The harmfulness of possible 'stress'-mediated haemodynamic or biochemical responses will depend largely on the individual's endogenous constitution. It is plausible that on the level of population small shifts in blood pressure, serum cholesterol composition, and pulse rate or rhythm that are harmless to most of us constitute a serious risk factor among (older) people with hypertension, or severe cardiovascular disorders (see chapter 6 for a more elaborate review)[47].

2.3.3 Health differences

A comprehensive body of evidence clearly indicates the key role of the social environment in the health status of populations as measured by mortality rate, life expectancy, perceived health, the prevalence of chronic disease and limitations[58,72,73]. Even in a relatively egalitarian society such as the Dutch, individuals from the highest socio-economic groups live around 3.5 years longer than individuals from the lowest group. In terms of healthy life expectancy differences as high as sixteen years are found[11]. In general socio-economic status is measured using education level, income as well as professional status. Other important attributes of the social environment are employment status (jobless, incapacitated for work), marital status and household composition, and ethnicity. Furthermore, geographical differences in health status are highest on the level of residential neighbourhoods, in particular in large cities, again implicating an important role for the social environment[72].

As we have seen in chapter 1, these social-demographically determined health differences are to some extent explained by an unequal distribution of unfavourable life-styles, lack of social support, and (job) insecurity, and maybe occupational (blue-collar labour) and environmental conditions (including noise and air pollution, traffic density and safety, crime rates). Most analyses show education level and material standards are more important than psychosocial factors[72]. Aside from these causal mechanisms, social-demographic health differences may in part be due to selection, in particular with respect to geographically determined health differences. In the last decades people from higher-income groups have moved out of the older neighbourhoods of cities, the vicinity of industrial zones to settle down in suburbia and dormitory towns. The more socially disadvantaged groups were and are left behind increasing the geographic accumulation of unfavourable social-economic conditions (see Figure 2-2)[73,74].

There are indications that socio-economic status has a direct influence on health status as well. Intermediate factors might not explain the full extent of social-economic health differences. Furthermore some evidence for a direct influence can be derived firstly from the fact that mortality rates are more closely linked to relative income within countries than to differences in absolute income between them. Secondly, national mortality rates tend to be lowest in countries that have smaller income inequalities and thus have lower levels of relative deprivation[xi,75]. Thirdly, most of the long-term rise in life expectancy seems unrelated to long-term economic growth, implicating some threshold beyond which further growth of the GNP no longer induces extension of life expectancy (reaches a plateau)[55].

[xi] This mechanism is consistent with developments of public health indicators in the UK during the Thatcher administration and in Russia and Eastern Europe after the Wall tumbled down.

Figure 2-2. Not just the 'big 4'. Neighbourhoods (4 digit postal codes) showing accumulation of unfavourable scores on a number of indicators of the social and physical environment: high density/low quality of dwellings, low availability/accessibility of open space (public gardens, shrubbery, parks etc.), low income, high fraction rented houses, high level of noise exposure[76].

Concern over the overt health inequalities, even in our egalitarian Dutch society, as well as clear disproportional distribution of environmental stress in the light of accumulation of unfavourable physical, spatial and social factors in many deprived neighbourhoods has favoured the equity principle in the field of environmental health (equity and ethics). This implies that distributional effects are an important aspect of health impact assessment as well[77].

2.3.4 Hidden impacts of environmental exposure?

Among the strong and consistent health impacts of social-economically-determined factors discussed here one will often search in vain for an independent effect of environmental exposures in available health statistics, such as mortality and morbidity rates or medical consumption[21]. The strong association between socio-economic and environmental conditions, the expected small increases of health risks attributable to environmental exposures combined with random variation produce a 'signal-to-noise'-ratio that goes way beyond the resolution of available epidemiological methods[59].

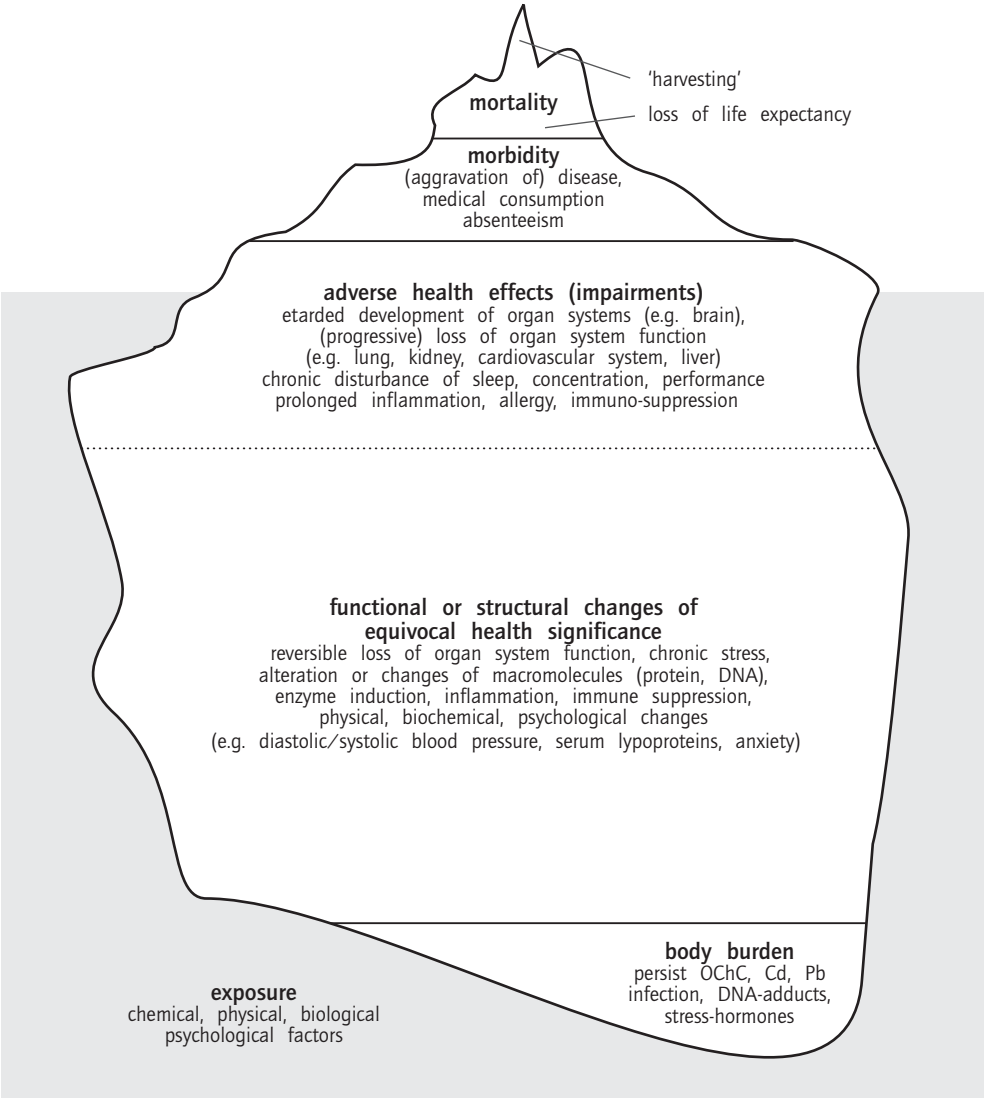

Figure 2-3. Diagram representing the public health relevance of end-points. Only the severe responses end up at the doctor's practice, in hospitals and thus in official health statistics (above the surface). Moreover, apart from few rare exceptions, these responses are not specific for environmental exposures, but dominated by socio-demographic, life-style and genetic factors. Responses beneath the surface are only seen in special surveys.

Only when health outcomes are more or less specific for a certain environmental exposure, one might use health statistics to reveal a quantitative association (see *Figure 2-3*). However, examples of such relations are rare and mostly derived from occupational exposures to high levels (asbestos and mesothelioma, vinyl chloride and angiosarcoma, benzene and leukaemia).

Therefore to detect environmental exposure specific health impacts one needs to investigate more specific end-points, such as body burden, long function parameters, and specific respiratory or

psychological symptoms. However, there is a strong geographic association between socio-economic status and poor residential environmental quality in the vicinity of important sources, such as airports, freeways, and industrial areas (see figure 1.4, chapter 1)[18,54,73,74,78]. One has to acknowledge the fact that social studies of a cross-sectional, sometimes ecological nature often simply lack the potential for an unbiased unravelling of all the possible relations and interactions dealt with in this chapter. Most study designs offer only limited possibilities to deal with socio-economic confounding in a satisfactory manner[18,47,52,79].

In short, the severe, clearly significant responses end up at a doctor's practice, in hospitals and thus in official health statistics (above the surface). However, apart from a few rare exceptions, these responses are not specific for environmental exposures, but often dominated by socio-demographic, life-style and genetic factors. On the other end we can observe exposure specific responses in special surveys, but it is often difficult to really assess their direct public health significance (*Figure 2-3*).

2.4 Indicators of environmental health impact

2.4.1 A huge variety of potentially significant responses associated with environmental exposures

The impact of hazardous environmental exposures on human health can take numerous shapes of various severity and clinical significance. Among the many responses that have been attributed to environmental exposures, are disturbed cognitive development in children, several types of cancer, reduced fertility, immune-suppression, severe noise annoyance and sleep disturbance[80,81,82]. During air pollution episodes well-studied human responses range from slight reversible lung function deficits in virtually everyone exposed, to aggravation of symptoms among asthmatics, and from hospital admission of patients with cardiopulmonary disease to the premature death of some of the very weak[83,84,85,86] (see *Figure 2-3*[87,88]).

Some effects occur soon after the onset of exposure; others emerge after long-term cumulative exposure, including a latency period. As discussed in the previous chapter the public health significance of any biochemical, physiological or psychological response to an environmental insult depends on many endogenous and exogenous factors. Whether or not an environmentally induced change affects individual health may be a function of its reversibility, individual possibilities of compensation or level of resilience. In their 2000 report the American Thorax Society state that the decision on 'where to draw the line' to categorize a response as an adverse health effect or an action level between pathophysiology or physiologic change is probably best left to the individual or the community'[88]. As often many pieces of the puzzle are missing, quantitative information on all aspects of the chain of events from exposure to clinical health outcome might be of relevance in characterising health risk.

2.4.2 Markers of exposure

In the Netherlands the most relevant types of emissions are often related to physical planning and infrastructure, such as *air pollution* due to road traffic (e.g. particulate matter, ozone, PAH and benzene), and *noise* and *vibrations* due to air, train and road traffic. Taxiing and test running of aeroplanes, as well as emergency discharges produce substantial *odour pollution* ('kerosene'). Apart from these chemical and physical factors there is 'exposure' to the *risk of accidents (third-party risk)*, e.g. aircraft crashes or traffic accidents involving dangerous substances, and a possible negative effect of the infrastructure on the quality of residential or local environment. Box 1 presents a number of indicators of exposures, specifically viewed from a public health point.

Box 1. *Examples of exposure indicators*

number of people exposed
intensity, frequency and timing of exposure
personal behaviour (at home, in the workplace, or outdoors)

Important context attributes are:
degree of personal or social control over sources of adverse environmental exposures
background level of adverse exposure
geographical and/or social distribution of exposure
perceived personal and/or collective benefit of causal activities
geographical and/or social accumulation of exposure to (exogenous) risk factors.

In general the population exposure distributions around airports or busy traffic roads are quantified combining modelled 'exposure' densities with population densities employing geographic information systems[89,90]. It has to be noted that personal exposure may depend on many variables especially with respect to life-style and daily activities[53,91,92].

Road traffic is by far the most important source of air pollution components, such as fine particulates, black smoke, PAH and volatile organic compounds. Concentrations of NO_2, CO and black smoke show clear gradients with the distance to busy roads, while particulate matter concentrations are only slightly elevated near the source. Ozone is formed exclusively in the atmosphere by secondary photochemical reactions within air pollution mixtures containing volatile hydrocarbons and nitrogen oxides. Although traffic is an important source of these precursors the highest levels of ozone air pollution are not found in the vicinity of busy roads, due to longer reaction times and the scavenging effect of other traffic emissions.

The contribution of aircraft per se is relatively small compared to the (large-scale) air pollution background and the local contribution of road traffic emissions. As all sorts of (new) economic activity are stimulated the 'siting' or extension of an airport will cause a substantial increase of road traffic. Naturally increased traffic emission should be evaluated as well in the framework of an environmental health impact assessment[53,93].

While evaluating environmental exposures due to infrastructure and spatial planning, such as the operation of airports, it is essential to include possible geographical and/or social accumulation of health risk factors. On the one hand socio-economically-determined risk factor accumulation may explain variation in health outcome prevalence, on the other it may indicate differences in susceptibility to environmental exposures[94]

2.4.3 Body burden indicators

Exposure to environmental pollution may lead to a certain *body burden*. Exposure to chemicals may be indicated by metabolite concentrations in blood, urine, breast milk or bone. These may give an impression of integrated exposure via various routes over a certain period of time. However, knowledge of the toxicokinetic properties for a meaningful interpretation of (most) biomarker results is often sparse. Likewise, indicators body burden often cannot be extrapolated quantitatively into health risk due to lack of knowledge[94].

In the framework of environmental health impact assessment chemical body burden indicators are probably of small relevance. It may only involve components of air pollution, such as PAH and related compounds or concentration of stress hormones in urine as a result of noise exposure.

Somewhat speculatively one might distinguish some sort of social-psychological 'body burden' if individuals become 'sensitised' by earlier confrontations with aircraft accidents, or some other type of

environmental incident (neighbouring waste sites or polluted soils), and develop a certain susceptibility for anxiety, stress or perceived risk[53].

2.4.4 Effect markers: exposure attributable changes of equivocal significance to health (early health effect indicators)

Environmental exposures may induce *biochemical, physiological or (socio)psychological changes* that more or less fall within the normal range of biological variation. Whether these changes are of any significance to health depends above all on the degree to which the function of organ systems (or social-psychological functioning) is affected, the reversibility and duration of the changes and the possibilities for recovery or compensation, and on the possible loss of resilience[87,88].

Acute, transient elevations of blood pressure and pulse rate, palpitations, increased serum levels of stress hormones due to noise or air pollution exposure might all fall largely within normal homeostasis. On the one hand it is still not clear whether there is any relation to sustained hypertension or prolonged stress-mediated hypercholesterolemia. Sustained haemodynamic effects would surely be significant to public health. Given a certain population distribution of for instance serum cholesterol level or systolic blood pressure, even a small shift due to environmental exposure may yield a substantial increase in the prevalence and mortality of cardiovascular disease (see chapter 6)[18,52,57,95]. On the other hand one has to appreciate the fact that the very same physiological responses, such as transient elevations of stress-hormones, haemodynamic and biochemical variables, are considered beneficial when induced in physical exercise or sports[47].

Psychosocial responses like annoyance, anxiety or perceived health risk may or may not fall within the background 'noise' of normal daily hassles. Of course at some point a clear distinction from clinically defined anxiety or depression can hardly be made. Unfortunately good longitudinal data are lacking with respect to onset and development of psychological disorders in this respect. A similar argument can be made for sleep disorders. Sleeping problems and their influence on mood and performance the next day are part of every normal life. However, at some point sleeping problems may become clinically significant as normal physical, mental and social functioning is hampered.

An important question in the context of health impacts of large airport is how (severe) annoyance should be evaluated from a public health point of view. Although no ICF- or DSM-codes have been defined for annoyance, it is clear severe annoyance affects several aspects of health, as described in chapter 2. Another committee of the Health Council defined 'annoyance' *'as feelings of aversion, anger, discontent, dissatisfaction or hurt that arise when exposure to noise (odour, or vibration) affects a persons thoughts, emotions or activities'*. The degree of annoyance associated with exposure to community noise, odour or air pollution can be quantified as the proportion of the population severely annoyed as determined by standardised questionnaires. However, there is some doubt whether these questionnaires measure annoyance according to the definition presented here (see chapter 6).

From a public health perspective some of the acute responses listed here may still be considered undesirable. At the level of populations these 'subtle' effects may cause some increase in acute morbidity and mortality (see *Figure 2-3*). For instance several studies have shown some association between average noise levels and the incidence rates for myocardial infarctions[52,49]. It is plausible that these infarctions have been initiated by 'subtle' haemodynamic changes due to noise exposure, comparable to precipitation of morbidity or mortality ('harvesting') as described with regard to air pollution episodes[85]. A similar mechanism probably applies to the association of admissions to psychiatric hospitals with traffic noise levels found in some studies[96]. It is not very likely people are driven to madness merely by noise exposure, but it might induce episodes of symptom aggravation.

Exposure to elevated levels of air pollution, especially particulates is clearly associated not only with respiratory, but also with more systemic cardiovascular endpoints, acute as well as chronic (see chapter 5)[86]. The pathogenetic mechanisms by which air pollution affects non-respiratory organs is still unclear, although an effect has been seen on indicators of autonomic heart function, such as heart rate, heart rate variability, electrocardiogram, and ventricular fibrillation. This may be mediated either by anaphylactic-like reactions decreasing heart rate and blood pressure, by oxidative stress (e.g. low density lipoprotein oxidation and successive destabilisation of atheromatous plaques), inflammation, neural or humoral mechanisms[97,98].

Box 2. *Examples of 'early effect' indicators*

Air pollution
- damage of cellular DNA or protein as a result of exposure to genotoxic substances
- small reversible deficits of pulmonary function (flow/volume variables) in relation to air pollution (particulates, ozone)
- inflammation cells and mediators in nasal (or bronchoalveolar) lavage in relation to air pollution exposures
- airway hyper-reactivity in challenge tests, in relation to air pollution exposure (ozone)
- impaired clearance

Non-respiratory, cardiovascular endpoints
- heart rate, heart rate variability
- cardiac arrhythmia (e.g. discharges of implanted automatic cardiac defibrillators)
- blood pressure fluctuation (hypo- and hypertension)
- hypoxemia (blood oxygen saturation)
- myocardial injury biomarkers (e.g. troponin)
- anaphylactic response indicators
- oxidative stress, low density lipoprotein oxidation
- destabilisation of atheromatous plaques

Noise, odour
- alteration of sleeping patterns in relation to noise
- neuro-behavioural functioning (performance) in relation to noise, odour, (solvent or pesticide exposures)
- acute elevations of stress hormone concentration in blood
- changes in leukocyte and lymphocyte concentration and composition in blood
- acute alterations of blood concentrations of lipoproteins (cholesterol), platelets, fibrinogen associated with noise exposure
- acute elevations of diastolic and (acute) decreases in systolic blood pressure in relation to noise
- cardiac arrhythmia due to (occupational) noise exposure (including ECG-abnormalities).

2.4.5 Indicators of morbidity

Functional or structural changes attributable to environmental exposures may (temporally) affect the normal function of organ systems or disturb mental or social functioning, in the end initiating disease or aggravating symptoms of pre-existing disease (frequency, intensity and duration). Whether disease indeed occurs depends largely on the individual vulnerability (genetic susceptibility, lifestyle, disease history, gender or age).

Given an unfavourable genetic make up environmental carcinogens may indeed initiate carcinogenesis in some unfortunate individuals (by timely switching on oncogenes or switching off suppresser genes) as a result of a series of chance events (specific DNA-interactions). Only when specific (not necessarily environmental) conditions for tumour promotion and progression are met in all stages the tumour might become clinically manifest. In accordance to conservative risk assessments around 10% of the lung cancer cases can be attributed to indoor exposures to radon or tobacco smoke. Still it is impossible to attribute individual cases to specific exposures. Occupational studies have linked prolonged exposure to high levels of benzene to the incidence of acute non-lymphatic leukaemia[63]. Whether environmental exposures to substantially lower levels would produce any cases among the general population is subject of an ongoing debate .

Another example of disease prevalence clearly initiated by environmental exposure is asthma among young children, predominantly those who are constitutional atopic. There is an established association with exposure to indoor dampness, and associated bio-allergens (house mite excrements, compounds of bacteria or fungi) .

At the population level one would expect morbidity to be reflected in *absenteeism*, use of *health services*, and *medication* (including self-medication). In a survey of populations in the vicinity of Amsterdam Airport Schiphol indications were found of increased use of medication for hypertension, cardiovascular disorders, allergy and asthma, tranquillisers and sedatives[53].

(Self) perceived health is another 'aggregate' measure to evaluate a population's health status. In general perceived or self-rated health measures are not very specific, as many determinants are involved, among which social-demographic factors (age, gender, ethnicity), prevalence of chronic disease and functional limitations, as well as social-psychological well being.

Box 3. *Examples of indicators to describe environment related morbidity*

disturbed intellectual development in children as a result of chronic lead poisoning
accelerated decrease in lung function, resulting in earlier onset of chronic obstructive pulmonary disease
aggravation of respiratory or cardiovascular symptoms (resulting in absenteeism, medical consumption, see next)
absenteeism from work or school
medical consumption, such as GP- and emergency room visits, and hospital admission rates
medication use, in particular with respect to disorders which can be related to a specific exposure (e.g. medication for cardiovascular disease, sleeping problems, sedatives and tranquillisers in relation to noise, or inhalers in relation to air pollution)
exposure specific morbidity (e.g. prevalence of cardiovascular disease, sleep disturbance, asthma, lung cancer etc.)
self-rated health

2.4.6 Indicators of Mortality

Mortality is the ultimate irreversible outcome of ageing or pathological processes. Age-specific overall mortality is an important and frequently used indicator to describe the public health status of the population. Since everybody dies at some point, the age at which death occurs or the years of life lost (reduction of life expectancy) are important attributes of mortality. 'Precipitated' mortality during particulate air pollution episodes involving predominantly the old and frail may in some cases only cost several days or months of unhealthy life[101,102], while the impact associated with fatal accidents involving individuals with a 'random' age distribution may amount to a loss of many healthy years[103]. It might be interested to look at mortality or life-expectancy at older ages as well, for instance at 65 or even 80 years. These age groups appear to be more sensitive to environmental exposures, such as air pollution (indoor as well as outdoor), and heat waves (e.g. associated with global warming), especially when the (health) care system fails (see chapter 1). The National Environmental Outlook predicts an increase of disease burden due to air pollution in 2030, although future levels will decrease or stabilise, based on the fact that the susceptible population is growing rapidly[73].

It is obvious that the general public does not perceive risk of large accidents, such as an aircraft crash in a residential area, simply in terms of annual death toll or even loss of life expectancy. For instance a calculated average third party mortality risk of around 1 death annually is fairly insignificant compared to the total of around 160,000 annual deaths in the Netherlands or the 20,000 deaths attributable to smoking. Not the public health burden per se but the involvement of tens or hundreds of victims at the same time and the social disruption that results from large accidents predominantly legitimates the high position of third party risk on the societal agenda (see chapter 3)[6,2].

To describe mortality in the population, age-, sex- and cause-specific figures are the indicators of choice, see box 4.

Box 4. *Examples of indicators of environment related mortality*

annual (age-specific) respiratory and cardiovascular mortality associated with particulate air pollution;
cancer mortality in relation to exposure to carcinogens (chemicals or radiation)
mortality risk at 65, or 80
average loss of life expectancy (life table analysis).

2.4.7 Aggregate measures of health loss

In the past decade the focus of health impact assessment has gradually shifted towards a more comparative approach, enabling policy makers to consider costs and benefits of preventive public health policies, given limited resources (chapter 2)[16,104]. Of course, for comparative quantification of health impact some sort of public health currency is required. In recent years several indicators have been constructed to integrate health loss on the level of populations by combining years of life lost and years lived with disability that are standardised by means of severity weights. An important example of the latter is the Global Burden of Disease project led by Murray and Lopez. To assess the global disease burden, and consequently the health policy priorities in different regions in the world, they applied disability-adjusted life years (DALYs)[9].

To assess the public health burden of environmental exposures one must consider at least three important dimensions of public health, viz. loss of life expectancy, loss of quality of life, and number of people affected (social magnitude). The diagram in *Figure 2-4* sketches the basic idea behind this and comparable approaches. Time is the unit of measurement. Public health loss is defined as time spent with reduced quality of life, aggregated over the population involved, and combining years of life lost and years lived with disability that are standardised by means of severity weights[xii,105,106]. In this diagram 'health loss' due to residential noise annoyance is suggestively added.

To assess the environmental exposure-attributable loss of DALYs, information on population exposure distribution, exposure-response relationships, and incidence and prevalence rates is combined to estimate annual numbers of people affected and the duration of the condition, including premature death[107].

From the policy maker's point of view there are at least three good reasons for this type of aggregation: comparative risk evaluation (e.g. setting priorities), evaluation of the efficiency of environmental policies in terms of health gain, and characterising health risks associated with the geographical accumulation of multiple environmental exposures.

An aggregate indicator of public health impact can also be applied to map spatial accumulation of environmental stress from the viewpoint of public health. In a pilot project we used DALYs to quantify (map) the accumulated health impact of noise (local streets, regional roads, motorway, aircraft, railroad), damp houses, road traffic air pollution, and industrial safety at the level of 6-digit postal code areas (neighbourhood, see *Figure 2-5*).

2.4.8 Social response indicators

In addition to the more or less objective measures of health status mentioned above, many social-science

[xii] To arrive at weights for this severity standardisation micro-level HRQL-measurements (e.g. health adjusted life expectancy, HALE) as well as preference-measurements (disability adjusted life-years: DALY's) can be applied.

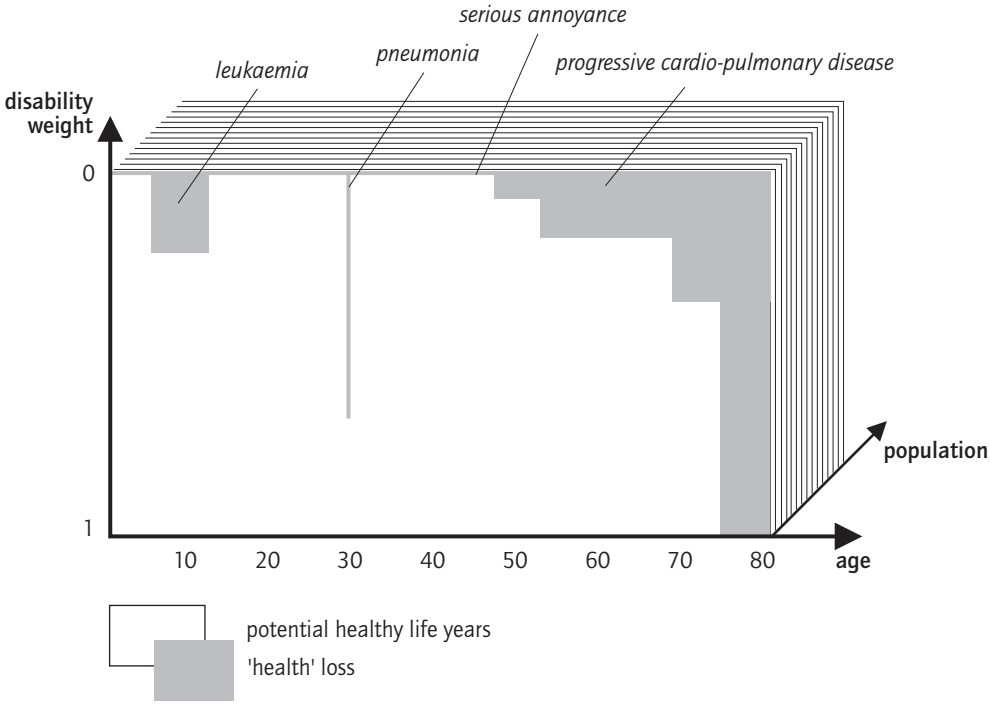

Figure 2-4. Diagram of the concept of disability adjusted life years.

studies have investigated social responses to environmental exposures of various kinds, either through 'face-tot-face' or telephonic interviews or by means of questionnaires.

Examples of social responses that in principle can be measured by standardised interview or questionnaires are shown in box 5.

Box 5. *Examples of indicators of social psychological response.*

annoyance
perception of risk (accidental death or injury, chronic disease)
complaints (about odour, noise, indoor air quality) registered by local health and environment authorities
residence or neighbourhood appreciation
willingness to move
social isolation
alienation
real estate prices.

2.4.9 Indicators of susceptibility

Based on our discussion of determinants of health and on several reviews, particularly on the health impact of noise and air pollution we can define some susceptible groups (indications of susceptibility)[18,52,79,85,101,108]. Box 6 presents some examples of susceptibility indicators, especially with respect to infrastructure-associated exposures.

Box 6. *Examples of indicators of susceptibility for environmental exposures*

Noise (annoyance, stress)
- self-reported noise sensitive individuals (a bit tautological)
- individuals who show much concern about the health impacts of noise or other aspects of the noise generating activity (e.g. air pollution, ill vibrations, aircraft accidents)
- individuals who feel they lack control over the situation (shortage of possibilities for instrumental coping, such as closing windows or organising consultation with airport management)

Noise (stress-mediated responses, risk perception)
- familial cardiovascular risk factors
- highly annoyed individuals
- patients with severe cardiovascular disease

Noise (sleep)
- individuals with physical or mental disorders
- individuals with sleeping disorders
- shift workers
- elderly
- individuals with clinical anxiety disorders or depression

Noise (communication and performance)
- individuals with impaired hearing
- children with learning difficulties

Air pollution (aggravation of symptoms necessitating medical help)
- individuals with respiratory disease (asthma, COPD)
- (older) patients with severe cardiopulmonary disorders
- children with constitutional atopy.

2.4.10 Economic evaluation indicators

While DALYs measure health loss in units of time, applying the willingness to pay (WTP) or willingness to accept (WTA) approach is an effort to measure health loss in terms of money. Embedded in welfare economics it should be seen as the rate of substitution between health and wealth. Health is regarded as an economic good. An individual's preference for one health condition over a certain period of time can be represented by a change of income or wealth, or, in other words decreased possibilities to purchase other valued goods. In principle there are two ways to produce these estimates:

investigating how health risks that are related to certain risky occupations are allowed for in the differences in salary, or what extra amount people are prepared to pay for safer or healthier products (for example, cars with airbags, or houses in quieter surroundings);

using questionnaires to find out what people are prepared to pay for one extra life-year or one year free of disease or disabilities (contingency valuation).

Significant methodological objections are attached to both methods in connection with the transferability of implicit (behaviour) or explicit (survey) preferences of people from one circumstance to another, including a series of shortcomings that are characteristic of all questionnaire-based surveys[109,110,111]. In terms of the value of a statistical life, the outcomes of the above-mentioned methods are nevertheless reasonably consistent. On average Americans, Canadians or Europeans are willing to pay in the order of two million euros for a statistical live (1.5 to 7 million Euro), which is approximately 70,000 to 80,000 Euro a year at a discount rate of 3%[112,113].

Disability free life expectancy (DFLE) is simply a special case of HALE as a threshold between healthy (1) and disabled or dead is applied (0).

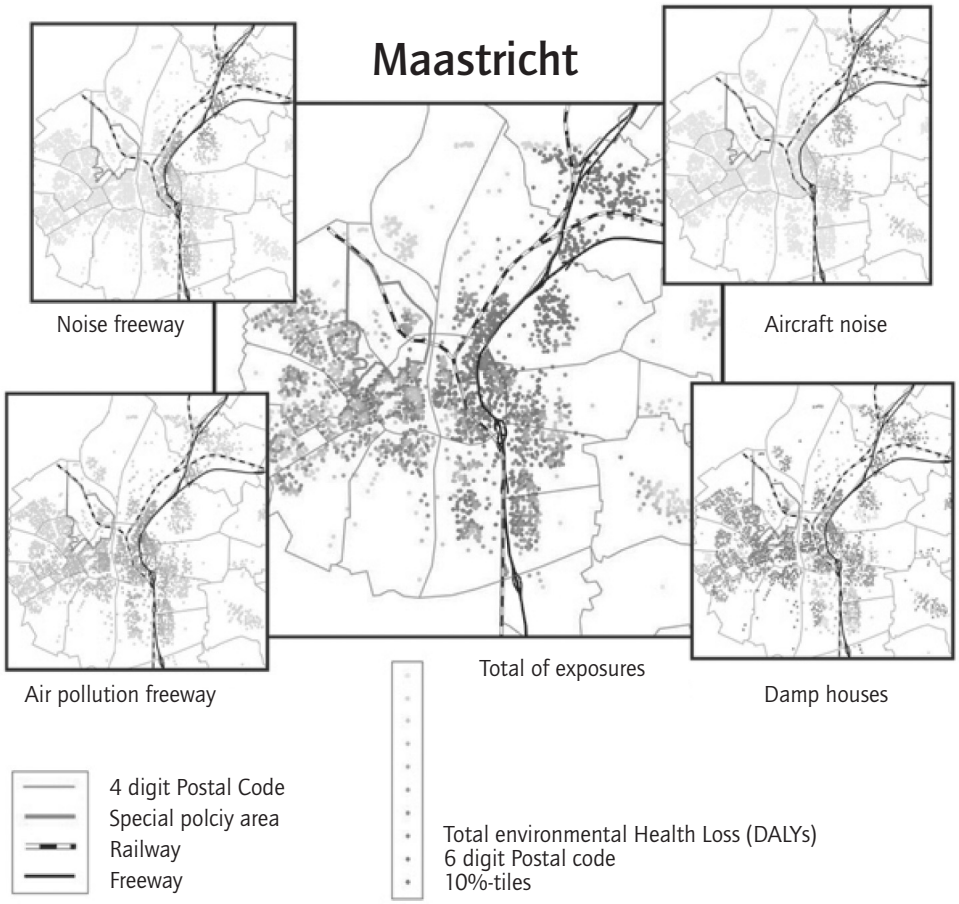

Figure 2-5. Application of DALYs to map local accumulation of environmental exposures at the level of 6 digit postal code: the example of Maastricht. Exposures include noise from freeways, connecting road, inner city road traffic, and aircraft, air pollution from connecting roads and freeways, and dampness in houses. DALY calculations were made presuming a standard Dutch population[114].

2.4.11 Conclusions

Figure 2-3 provides a schematic representation of the distribution of health effects in the population due to pollutant exposure. It clarifies the significance of apparently harmless effects such as a slight reduction in pulmonary function. Environmental exposures may cause transient functional changes in a relatively large proportion of the population exposed. In a smaller susceptible proportion of the population these changes may lead to physical or mental symptoms and in the end promote the initiation of disease or the aggravation of its symptoms. At the top of the iceberg environmental exposure may precipitate mortality among the weakest. However, in some cases the loss may amount to many years of healthy life, for instance in case of fatal accidents or environmental exposure as sufficient cause for disease. For most of the known pollutants the information with regard to the causal chain of events as described in *Figure 2-3* is far from complete, with information on major parts of the chain missing (e.g. type and extent of adverse health effects).

2.5 Qualitative assessment of the available exposure response relations

To use the framework discussed here for the assessment of possible health impacts of the operation of a large airport, we add three sets qualitative criteria to evaluate available exposure response data, dealing with the strength of evidence, the severity of the response and the distribution of effects. Box 7 briefly describes the qualitative assessment we have produced in the context of the Netherlands Health Council advice on the Health Impacts of Large Airports[19].

2.5.1 Evidence

To assess the evidence from (human) studies we propose to use the classification of International Agency for Research on Cancer (IARC) into the following categories:

Sufficient evidence: a causal relationship has been established between exposure to the agent, mixture or exposure circumstance and the health effect. That is, a positive relationship has been observed between the exposure and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence.

Limited evidence: A positive association has been observed between exposure to the agent, mixture or exposure circumstance and the health endpoint for which a causal interpretation is considered to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

Inadequate evidence: The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and the endpoint.

Evidence suggesting lack of causality: Several adequate studies covering the full range of levels of exposure that human beings are known to encounter, are mutually consistent in not showing a positive association between exposure to the agent, mixture or exposure circumstance and any studied endpoint at any observed level of exposure. Of course, the possibility of a very small risk at the levels of exposure studied can never be excluded.

2.5.2 Severity of health responses

We propose to classify health endpoint as light, moderate and severe (a 'triage'). A *light* response falls within normal biological variation, does not affect normal functioning or if it does, only temporarily. A severe response represents a serious handicap in everyday functioning, and are in general clinically significant (demand medical care). Moderate responses are somewhere in between, e.g. because they may be adverse to very susceptible individuals.

2.5.3 Distribution of health responses

Of course the number of affected people within the exposed population is relevant as well. This is often a function of the intensity (distribution) of the exposure and the susceptibility of the exposed. We propose three categories: highly exposed and/or susceptible individuals, specific sub-groups, such as patients, elder people with frail health, people with certain professions, inhabitants of deprived and heavily exposed areas, and a substantial part the total population.

2.6 Finally

The persistent health inequalities are a major focus of public health research and policy in Western societies. There is a consistent association between socio-economic status and environmental quality, both

socially and geographically. Although it is unlikely that health inequalities can be attributed directly to environmental conditions around large infrastructure, it is plausible that the social impact of large environmental interventions contributes to the complex social-psychological processes affecting social cohesion and thereby community health[57].

And of course, we must acknowledge that little or weak evidence does not necessarily constitutes evidence for little, weak or no health impact[18].

Box 7. *The example of the evaluation of the health impact of the extension of a large airport*

In tables 1, 2 and 3 the main conclusions concerning relevant health impacts of the operation of large airports are summarised. Statements with respect to number of people affected, clinical significance and evidence are based on several reviews we have consulted (table 1).

Noise, smell, risk

It is well established that as levels of noise exposure increase a higher percentage of any representative population will report to be severely annoyed. No doubt this presents an important social problem. Whether this is a health problem as well is less obvious. It is a fact that several studies have found an association between serious annoyance and highly correlated social responses, such as perceived stress, anxiety, and risk perception on the one hand and reported (psychological) symptoms, cognitive complaints, (self-)medication and use of health services on the other. The causality of this association however is far from obvious. As discussed in chapter 3 it would potentially be confounded by many interacting factors, which may explain the contradictory results seen over all studies. Furthermore, the attitude towards the source of noise, and sensitivity to noise accounts for more variation than the level of noise exposure by itself[47,108]. People who report themselves seriously annoyed might just tend to report symptoms, use self-medication or even visit a GP, irrespectively of actual noise levels.

The case for stress-related responses, such as elevated diastolic blood pressure, stress hormones, causing prolonged hypertension or hypercholesterolemia, attributing to the prevalence of cardiovascular disease at the level of populations is far from strong. However it is conceivable these types of acute heamodynamic responses may contribute in one way or another to the onset of acute cardiac infarctions (as many other stimuli may).

There is not much doubt that nocturnal noise may reduce the quality of sleep, change sleep patterns (including awakenings, EEG's) and may lead to chronic loss of sleep in the end. Poor quality of sleep affects daily mood, and performance, among other things. There is much uncertainty on the ability of residents to habituate to noise and the long term consequences of any particular degree of noise induced sleep disturbance. Excessive sleep disturbance will eventually compromise social-psychological well being, but until now the amount of sleep loss required to adversely affect health cannot be defined properly.

In a recent study in the Netherlands indications were found that the use of psychotropic and cardiovascular medication was associated with the level of noise exposure. The results are consistent with those of other cross-sectional studies (and one cohort-study). The impact on medication use is consistent with observed social responses, such as increased levels of stress, concern about health and mortality risks, and noise and smell annoyance.

There is no convincing evidence for a direct effect of exposure to noise, smell, vibrations or risk on health outcomes such as congenital abnormalities, birth weight, or disorders related to the immune system (infectious or auto-immune disease). So far plausible mechanisms of action for these disorders are lacking.

The indications that noise would contribute to the prevalence of cardiovascular and psychiatric diseases are inconclusive. However, with respect to these disorders the evidence is not strong enough to reject the hypothesis noise is in some way involved in the multi-causal process leading to disease.

Table 1. Overview of reported health related responses to community noise exposure.

Response	number affected[1]	clinical significance[2]	evidence[3]
Social responses			
annoyance	***	*	***
performance	**	*	**
symptoms (anxiety, depression)	**	*	**
perceived health	**	*	**
Sleep			
sleep pattern	**	*	**
disturbance, awakenings, loss	**	*	**
reported quality	**	*	***
mood, performance	*	**	*
Stress related responses			
blood pressure	**	*	**
pulse rate, arrhythmia	**	**	**
hypertension	**	**	**
serum lipoprotein composition	**	**	*
cardiovascular disorders	*	***	*
ischaemic heart events	*	***	**
psychiatric disease	*	***	*
immune system	**	*	*
stress-hormones	**	*	*
birth weight	*	**	*
congenital abnormalities	*	***	-

1. * = highly exposed and/or susceptible individuals, ** = specific sub-groups, *** = substantial part of the total population
2. * = clearly within normal biological variation, ** = possibly adverse to susceptible individuals, *** = clinically relevant (ICD-coded)
3. * = inadequate, inconclusive evidence, ** = limited evidence, *** sufficient evidence, - lack of evidence

Air pollution

Since the beginning of the nineties a surge of epidemiological studies has been published showing associations between elevated levels of air pollution and various health end-points, in particular daily mortality and respiratory health care events. Although the validity and causality of these associations is subject of an ongoing debate in the epidemiological arena, no one really doubts air pollution still affects public health, even in Western societies were a relatively high level of control is established. Controversy involves primarily the nature, magnitude and public health significance of the indications for health effects. Furthermore, there is much discussion on which the compounds would be indicative of the toxicity of air pollutant mixtures[115,116,117,118,119,120,121,122].

The short-term effects presented in table 2 are confirmed in many time series analyses carried out all over the world. The long-term effects are only seen in a limited number of cohort studies. From a public health perspective these responses are potentially much more significant than short-term aggravations as they imply a role of air pollution in the onset and development of disease and potentially substantial loss of healthy life expectancy[107]. However, exactly this structural impact on survival needs to be confirmed in other well-designed studies. Results of series of studies in which specific indicators for traffic-related air pollution were linked to respiratory health measurements indicate a substantial contribution of (heavy) traffic to prevalence of respiratory symptoms and disease.

Evidence for carcinogenicity of specific compounds such as benzene and PAH is predominantly derived from occupational studies and animal assays at relatively very high levels of exposure. It is not clear whether these effects are relevant (or significant) at much lower environmental exposure levels.

As the operation of an airport inevitably increases the local and regional levels of air pollution indicators, such as fine particulates, ozone, black smoke, sulphur dioxide and nitrogen oxides, some effect on respiratory and cardiovascular health might be expected.

Insulation of homes may lead to an increase of indoor levels of dampness-related allergens, radon and environmental tobacco smoke. Adaptation of time-activity patterns to outdoor noise may increase indoor exposure as well.

Table 2. Overview of reported health related responses to particulate and photochemical (ozone) air pollution

Response	number affected[1]	clinical signif[2]	evidence[3]
Long-term exposure (cohort studies)[4]			
overall mortality	**	***	**
cardiopulmonary mortality	**	***	**
lung cancer mortality	**	***	**
chronic resp. symptoms in children	**	***	**
chronic bronchitis in adults	**	***	**
odour annoyance	**	*	***
Short term exposure (time series)			
mortality (respiratory, coronary heart disease, pneumonia)	*	***	***
exacerbation of respiratory and cardiovascular disorders resulting in hospital admission	**	***	***
exacerbation of asthma (attacks, use of bronchodilators)	**	***	**
aggravation of respiratory symptoms (upper and lower tract)	**	**	**
affected lung function	***	**	***

1. * = highly exposed and/or susceptible individuals, ** = specific sub-groups, *** = substantial part of the total population
2. * = clearly within normal biological variation, ** = possibly adverse to susceptible individuals, *** = clinically relevant (ICD-coded)
3. * = inadequate, inconclusive evidence, ** = limited evidence, *** sufficient evidence
4. only investigated with respect to particulate air pollution

Table 3. Reported health related responses to specific air pollutants

Response	number affected[1]	clinical signif[2]	evidence[3]
Carcinogenic air pollutants			
lung cancer (e.g. PAH-derivatives, radon[4], environ. tobacco smoke[4])	*	***	**
leukaemia (e.g. benzene)	*	***	*
Dampness related allergens4			
lower respiratory symptoms	**	***	***
asthma	**	***	***

1. *= highly exposure and/or susceptible individuals, ** = specific sub-groups, *** = substantial part of the total population
2. *= clearly within normal biological variation, ** = possibly adverse to susceptible individuals, *** = clinically relevant (ICD-coded)
3. *= inadequate, inconclusive evidence, ** = limited evidence, *** sufficient evidence
4. increased indoor exposure as a result of measures to avoid noise exposure (home insulation, indoor activity)

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3 Dealing 'sensibly' with environmental risk[i]

Augustinus E.M. de Hollander, Aldert H. Hanemaaijer

Abstract

In this chapter we take a closer look at an important instrument of collective health protection, risk assessment and management. We establish that in principle any decent government should apply rights-based decision rules in environmental risk policy, guaranteeing every citizen an equal right to a certain level of protection. However, we have also seen that purely right-based policies may go beyond the bounds of efficiency or affordability. Vice versa, if maximisation of efficiency is the main target, there are inevitably distribution consequences: individuals or specific populations being treated unfair or even harmed in the name of efficient use of scarce resources: the traditional trade-off between 'equity' and 'efficiency' of welfare economics.

We discuss a number of 'sobering', complicating features of the concept of health risk, at least from the perspective of the average environmental engineer. The concept of risk consists not exclusively of 'objectively' measurable qualities of systems; but risk is also a social construct shared by our equals, in which qualitative, social-psychological attributes may be decisive in the acceptance of the dangers around us.

To deal with risk as a 'social construct', as well as with equity versus efficiency trade-offs, we tentatively propose a typology of risk problems with matching procedures of increasing comprehensiveness, comprising 1) 'business as usual', traditional, quantitative analysis and management, 2) appropriate and proportional use of 'scarce resources', 3) a way out when 'calculations are simply not the issue', or 4) wisdom, when 'ignorance' is recognised in time. Procedures involve decision rules, goals, solutions, strategies (discourses) and instruments.

[i] Chapter based on contributions as scientific secretary to reports of the Health Council of the Netherlands (1995, 1996, in co-operation with dr. W.F. Passchier) and a memorandum written at the request of the State Secretary of the Environment, published in Dutch by the Office of Environmental Policy Assessment (de Hollander & Hanemaaijer, 2003). To be submitted as Hollander AEM de, Brunekreef B, Woudenberg F, Hanemaaijer A. Dealing 'sensibly' with risk.

3.1 Introduction

3.1.1 The State Secretary's question

How should we deal with the deadly Legionella-bacteria, uninvited guest in every corner of our treasured drinking water systems? Can we really permit young children to visit schools in the sphere of extreme low frequency electromagnetic fields generated by high power lines? Should the expansion of our national pride, Schiphol Airport, be bridled, simply because safety standards with respect to aeroplane crashes can't be met completely, partly due to residential areas edging themselves towards every last acre of open (flying) space? Don't we like flying cheap; doesn't a large airport generate jobs and wealth?

Recently the State Secretary for the Environment, Mr Van Geel pleaded in several media for a more rational appraisal of the urgency of environmental health risks. A renewed attitude of sensibility would help us to spend available resources for risk reduction in a more proportionate manner, free from irrational fears or political correctness[1], and conscious of the simple truth that risks are a fact of life. Why do we bother so much about an extremely small additional chance of childhood leukaemia, if existent at all, while at the same time ostensibly we hardly worry about the many victims of daily traffic, or even worse, we carelessly light our next cigarette (see table 3-1). Do we have to spend billions of Euros to expel the Legionella-bacteria from drinking water systems throughout the Netherlands, while we know by doing this we will only prevent a limited fraction of all cases of legionnaire's disease? After all, as far as we know, more than half of the infections are caught abroad, e.g. on holidays around the Mediterranean[2]. In sum, is there reason to believe we are spending our limited resources for health risk management inefficiently, on the waves of emotions, however real (genuine) these emotions and concerns may be[ii]? This debate is going on for several decades now[3,4].

The State Secretary requested the National Institute of Public Health for a contribution to the debate on dealing 'sensibly' with risks[iii]. Assigned to deal with this request we defined two sides of the problem, the *why*-question and the *how*-question. Why do we (society and politics) respond so 'irrationally' and 'inconsistently' to emerging environmental risks? Or, don't we? How should we deal with 'real' risks, different perceptions and (ir)rationalities in a 'nuchter' manner?. With this request the State Secretary touches upon a sore spot in the field of risk assessment. Often, there is an 'unofficial' or at least an implicit policy makers urge behind these requests: the need for well-founded scientific algorithms to make a distinction between 'real' risks and 'perceived' risk, between scientific truth and just another issue (delusions) of the day or, in other words. "Please provide us with sobering numbers that tell us how to respond to (emerging) risk in a cost-effective way without running the risk of getting our political head chopped off!".

From the viewpoint of the average policy maker one risk measure to determine the seriousness of risks, preferably accepted by all parties would be a powerful tool to develop proportionate risk reducing strategies. However, the past 30 years have taught us such simple algorithm is probably best regarded as

[ii] In the last decade in the Netherlands we have seen a Boeing 747 digging itself into a block of flats, an exploding firework storage blowing away a complete neighbourhood, a whirlpool like machine spraying a fine mist of droplets of Legionella spec. among a large susceptible population of elderly people visiting a flora exhibition and so on. In all those cases the risks were supposed to be managed in a rational way.

[iii] He actually used the Dutch word 'nuchter' for which a series of translation can be found in dictionaries: 'rational', 'practical', 'sensible', commonsensical', 'down-to-earth', 'matter-of-fact', 'reasonable', 'non-emotional', 'sober', or 'sober-minded', probably the heart of the matter.

the unattainable Holy Grail, even if one restricts oneself to human health risks, as we do here. At the same time, that doesn't mean it wouldn't be fruitful to go on a quest.

3.1.2 Methods

Based on a comprehensive review of the literature we will analyse the *'why'-question*, discussing different approaches of the concept of risk, the aspects of risks that determine people's perception and acceptance, and how experts have tried to measure risks. We will outline aspects of complexity and uncertainty that are involved in scientific analysis of risks and how to deal with them. Finally, we investigate the perspective of risk as a social construct, not just as a set of quantifiable qualities of physical, spatial or social processes.

Based on this analyses we will deal with the *'how'-question* by constructing a typology of risk problems based on quantitative as well as qualitative aspects, such as probability and magnitude of the consequences, degree of uncertainty and complexity, and social stakes (societal benefit, appropriateness). Successively this typology will provide guidance on the manner in which risks are analysed (e.g. the choice of risk attributes, measures and criteria), the decision procedures and rules, and on the involvement of social groups and stakeholders.

The development of this framework has been guided by comprehensive consultation and discussions in working groups, workshops with participants from relevant disciplines within and in some cases outside the National Institute of Public Health and the Environment (RIVM).

3.2 Risk: a dragon with many heads

3.2.1 Risk: different definitions

If you look in Webster's Dictionary, 'risk' is defined as 'chance of injury, damage or loss'; in this definition danger is interpreted as 'evil chance': 'the possibility that something will go wrong'. Other authors claim that both 'evil' and 'good' chance come together in the word 'risk'. It is thought that the word 'risk' is derived from the Italian word 'rischiare' which in turn can be traced back to the Greek word 'rhiza', meaning root. On Crete the rocks jut out of the coast, often submerged like roots underneath sea level[5]. Sailing around these dangerous rocks of course requires helmsmanship, but the benevolence of the Gods as well. The possibility of damage or loss is directly linked to the expectancy of reaching one's goal as a reward of skilful effort: success and failure are two sides of the same coin[6,7]. Or, viewed from an even wider global historical perspective: exactly the ability to adopt new technologies, accepting the associated unknown risks is what has brought civilisations success and cultural dominance[8]. In the provocative words of hippy-writer Tom Robbins: "humanity has advanced, when it has advanced, not because it has been sober, responsible, and cautious, but because it has been playful, rebellious, and immature".

Within the domain of environmental risk management several committees of distinguished scientists sought to propose an appropriate, if not final definition of the risk concept, almost always from the viewpoint of 'evil chance'. In a report to the government the Health Council of the Netherlands proposed: 'the possibility, with a certain degree of probability, of damage to health, the environment and goods, in combination with the nature and the magnitude of the damage'[9]. In their definitions of risk other influential institutions such as the European Environmental Agency and the Environmental Protection Agency in the US also distinguish both the aspect of *probability* and *nature* and *severity* of undesired consequences[10].

Table 3-1. Crude estimates of annual mortality and loss of health adjusted life-years attributable to a number of risk factors in the Netherlands[26,11].

Risk factor	Annual mortality	DALYs/year
smoking cigarettes	20,000	440,000
obesity	8,000	170,000
physical inactivity	8,000	135,000
unfavourable diet (saturated fat)	7,000	137,000
particulate air pollution (long term)	2,500	32,000
alcohol	2,200 *	195,000
accidents at home	2,200	52,500
traffic accidents	1,200	85,000 **
air pollution (pm)***	1,300	1,800
indoor radon	800	7,900
environmental tobacco smoke	530	6,300
Legionella drinking water systems	80	560 ****
benzene (ambient air)	3	140
industrial accidents	1	40 ****
lightening	1,5	40

* traffic accidents excluded
** only permanent injury
*** based on time series analyses of daily mortality and air pollution
**** loss of life years only

3.2.2 Risk: causal chains in a traditional approach

Based on this type of probabilistic definitions traditional risk assessment approaches deal with risks as objective, measurable, countable, or even weighable phenomenon that can be unequivocally quantified by analysing physical, chemical, biological and sociological processes. Damage or loss is the result of processes that can be described by causal chains (see Figure 3-1)[12,13,14]. Moreover, analysis of these chains may identify the options for risk reducing measures, such as reducing emissions of toxic substances, changing human behaviour through public information campaigns or tax measures.

In formal system analysis often the DPSIR-cycle is applied, describing Driving forces, Pressure, State, Impact and societal Response. The DPSIR framework is often used to organise indicators, especially in the context of (international) environmental problems and sustainable development. As a result of driving forces such as industrial production, spatial planning or consumerism the human system puts pressure on the environmental system (e.g. through toxic emissions or noise), affecting the quality or state of the environment. This in return may affect human well-being or health. Responding to overt health damage or economic losses due to a poor state of the environment, the human system may decide to put more effort into cleaner technology or the spatial separation of transport and residential functions[15,16].

Furthermore, the concept of risk is characterised by uncertainty regarding how and when damage or loss will occur as a result of human activity, as well as the nature and extent of the consequences. One stage in the cause-effect sequence shown in figure 3-1 does not necessarily have to result in the next, or at least not to the same extent. This all depends on the processes within the sequence and on external factors, such as natural processes. Human activity will always yield small- or large-scale changes in substance and energy flows but these changes are not necessarily a threat to the health of humans or the environment. In this concept risk can be regarded as an attribute of processes in society, the possibility that things get out of control. Of course *Figure 3-1* is a simplification. In practice there will always be a web of causal sequences, including all kinds of feed back loops, counteracting or sometimes boosting changes[14].

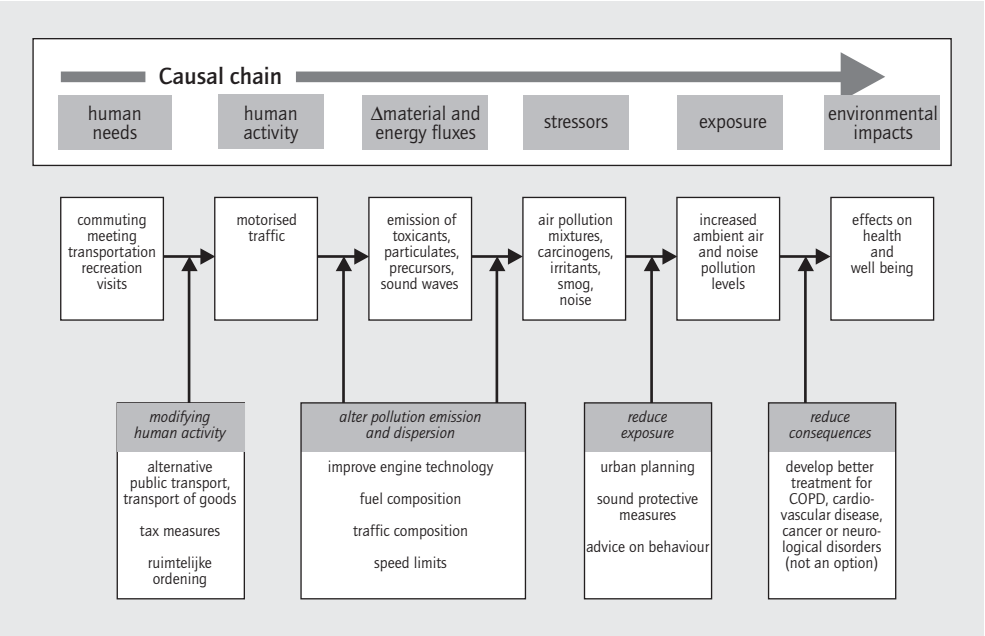

Figure 3-1. Relevance of causal chains for risk analysis and management; the example of traffic emissions

3.2.3 Risk: just a number, just another process attribute

Traditional risk management requires measures and numbers to express the 'magnitude' of risks. Expressed as a number risks can be handled, at least to a certain extent, risks can mutually be compared, and thus fair exposure standards for a range of risks can be established[17,12]. Often probability has been used to quantify risk, e.g. the probability that an undesired event, such as an increase in the number of (statistical) deaths, will take place in one year. In risk management of chemical carcinogens and radiation risk is often quantified as the number of additional cancer deaths among a large group of persons (often a million) exposed for one year. The magnitude of the consequence can also be part of risk quantification. In Dutch regulations with respect to large industrial accidents the accepted probability of an accident is a non-proportional function of the number of people simultaneously affected, to account for the social disruption that is associated with a sudden large number of deaths and/or wounded (high fatality counts in contrast to equally large, but the gradually and more diffuse incidence of for instance road casualties)[17].

A committee of the Health Council of the Netherlands stresses the fact that a variety of attributes is linked to the concept of risk. Like probability and number of immediate victims, some can be expressed in measures and numbers: attributable healthy life-expectancy or fertility reduction. Others are more qualitative in nature, such as the degree to which people (perceive they) are voluntarily exposed or perceive they can avoid or control the risk[12]. We will discuss these qualitative risk attributes later on.

3.2.4 Risk: a matter of delineation

Conscientious definition of the boundaries of a risk problem is an important part of risk assessment, often decisive for the outcome of quantitative analysis. What is part of the risk problem, and what is not? For

instance, while planning the site of an oil refinery, do we only bother about the emission of hazardous substances or do we include the probability of large accidents as well, within the premises and around the plant? And what about the possibility of accidents during transport of raw materials or end products? The establishment of an oil refinery will affect the local environmental quality, but should we also concern ourselves with the contribution of fossil fuel use to the greenhouse effect? Making an assessment of possible damage or loss, choices will have to be made on what we include in our scope: human health, ecological integrity, material goods, the local society, our nation, or even the world population? Or, one step further, do we always have to make our assessment from the perspective of global sustainability?

Analysis of past controversies shows that discrepancies in the assessment of risks and disagreement on management strategies are often caused by differences in the delineation of the risk generating system, the problem formulation that is implicitly or explicitly applied by experts as well as lay-people. Furthermore, this delineation of the system often depends on the societal perspective of the institution performing the risk assessment. Using the old case of the herbicide 'Alachlor', recently Hatfield and Hipel showed that to solve the controversy three different, influential institutions, primarily guided by normative, political values, defined different 'risk systems'. The decision whether this agricultural pesticide should be approved for the market, can be confined to possible unfavourable effects of the chemical on human health and ecological integrity of the candidate. However, one could also consider the unfavourable effects of competing, already permitted pesticides by way of comparison. One could even consider the economic advantages, such as the destruction of an undesired monopoly, or the advantages for public health, such as the availability of low-priced, healthy fruit and vegetables for the less thriving. Of course these 'extra-scientific' factors, or in other words value based decisions, are closely related to societal positions of the institutions (or the stakeholders they represent)[18,19,20,21]. In sum, an important step in environmental risk assessment is the definition of the system boundaries. This may involve the following questions:

which activities
what kind of damage and loss
what kind of benefits or gains (if any)
which causal chains
which geographical scale
what time scale (horizon)?

The report of the Health Council of the Netherlands on the health effects of large airports is a good example of explicit delineation of the 'airport system'. This system comprises a large area of many square kilometres surrounding the airport, including the economic activity and infrastructure to operate the airport, including the new housing development that is claiming the once empty Haarlemmermeer polder at the cost of extension potential[22]. Of course, this delineation is not a purely scientific matter as it involves social values. We will come back to this later.

3.2.5 Risk: uncertainty and complexity

In a pure, traditional scheme of risk management scientists analyse the probabilities of damage or loss in a quantitative, 'value free' and thus 'indisputable' manner. Successively, policy makers, after consultation with relevant stakeholders decide to what level risks are tolerable from a societal point of view, and where and at what pace risks should be reduced[23]. In more developed schemes there is also consultation between risk assessors and managers during the process for instance on problem formulation and risk characterisation[12]. However it has become obvious that these models fail to deal properly with the limitations of our scientific knowledge base. Every risk analysis, even when performed by a group of

excellent Nobel-prize winners, is limited by some degree of uncertainty. In some cases we are only dealing with lack of *exactness*. Only a limited number of measurements is available, so we don't know the precise value and variance of one of the parameters in our risk equation. The detail of the maps we apply is just a little bit too coarse, or we have to add up apples and oranges (and get a result for fruit). In other cases our risk analyses may lack *validity*. We don't really understand which are the relevant causal chains and how they function. We did model the scant data, but we can't be sure about the causality of the associations, and thus we can't be too sure about the truthfulness of our calculations. Very complicated statistical analysis tell us there is a positive association between daily mortality or cardiovascular hospital admission on the one hand and meteorology dependent air quality on the other, but we can't yet be sure that further reduction of traffic emission will really improve public health. Sometimes we are close to *ignorant*. We don't have a clue about how the system functions, where it starts or where it ends, or we can only identify and describe a number of specific elements in a much more extended system. We are in the dark whether it concerns the probability, the magnitude or the timing of exposure, whether it concerns the nature, the seriousness or the scale of the consequences. We don't even know what we don't know.

Funtowicz and Ravetz propose a very similar division into *technological, methodological* and *epistemological* uncertainty[24]. Other divisions are also applied, such as 'lack of knowledge' (inexactness, lack of observations, practically immeasurable) versus 'structural' or 'systematic' uncertainty (conflicting evidence, reducible ignorance, indeterminacy, irreducible ignorance) or 'criterion' versus 'construct' validity often applied in epidemiology[25,26]. It is important to note here, that with respect to uncertainty we tend to make careful, conservative assumptions, 'better safe than sorry' (or at least think we do). In sum, our values lie hidden in our policy analyses: 'you can't take politics out of analysis'[27]. We will come back to that issue later.

Complexity, another concept often applied in risk assessment, is closely associated with uncertainty, but far from synonymous to it. It refers to the extensiveness of the web of causal chains, discussed in a previous section, the extent of the total of relations that make up the risk system, and thus to the difficulty one may have to identify the relevant cause-effect sequences, feed back loops and interactions.

Climate change is the obvious example of an extremely complex (risk) system: a large scale (global), long term (centuries) phenomenon, multi-disciplinary by nature, concerning all economic sectors, taking place on different, interacting geographic scales, comprising a vast web of causal chains. On the other end of the spectrum of complexity are the simple exposure-response phenomena. For many chemicals there is sufficient toxicological data to model potential human health impacts in a reasonably reliable manner, one by one[iv]. However, in real life people are never exposed to one chemical after another, but to mixtures of many individual substances. Should we desire to make a similar toxicological assessment of the health effects of combinations of, let say, ten chemicals or more, then the necessary animal experiments data will soon reach astronomic numbers. So, relatively simple problems can be highly uncertain, due to lack of data (practically immeasurable). Calculations of the probability of large industrial accidents causing fatalities assume safety legislation is being implemented, enforced and inspected. However, evaluation studies of the plane crash of 1992 in the Bijlmer and the exploding fireworks storage facility in Enschede in 2000 have shown this assumption is often false[28].

One of the ways in which scientists deal with complexity is mathematical modelling, reducing reality into a system of essentially stocks and flows, states and transitions or actors, compartments and relations.

[iv] For the sake of simplicity here we disregard the many scientific controversies on extrapolation from animal assay to humans (see chapter 4).

From the moment Meadows and co-workers identified the 'limits to our growth' in the seventies, using a relatively simple world model, application of computer models and corresponding scenario techniques in environmental sciences has grown exponentially[29]. The old and trusted scientific method, requiring hypothesis formulation, testing and falsification under the controlled circumstances of the lab[30], is traded for the 'wondrous world' of mathematical models. Of course model output can be validated partly by comparison with historical data sets, but the forecasted behaviour of the world system cannot be tested in a classical experimental way. The complexity of large scale, irreversible issues, such as the greenhouse effect or globalisation, the necessary integration of time and geographic scales is far beyond the limited domain of traditional, empirical accumulation of knowledge. Of course, we wouldn't want to dissuade anybody from using models, on the contrary. However, it is a lot harder to present model results as science 'free of values', or beyond doubt, as we would from a positivistic perspective. After all, complexity has to be reduced one way or another, gaps in knowledge have to be bridged with assumptions, and huge uncertainties have to be accounted for. A myriad of decisions, some big, many small, will -often implicitly- be guarded by and founded on social values and ideology[31].

There are methods to deal with at least part of the uncertainties associated with risk assessment, if necessary making hidden values explicit in the process (see Figure 3-2). Uncertainties on the level of statistics (variance, measurement error) can be described by probabilistic techniques, such as Monte Carlo or sensitivity analysis. Model results are presented as probability distributions or ranges instead of single

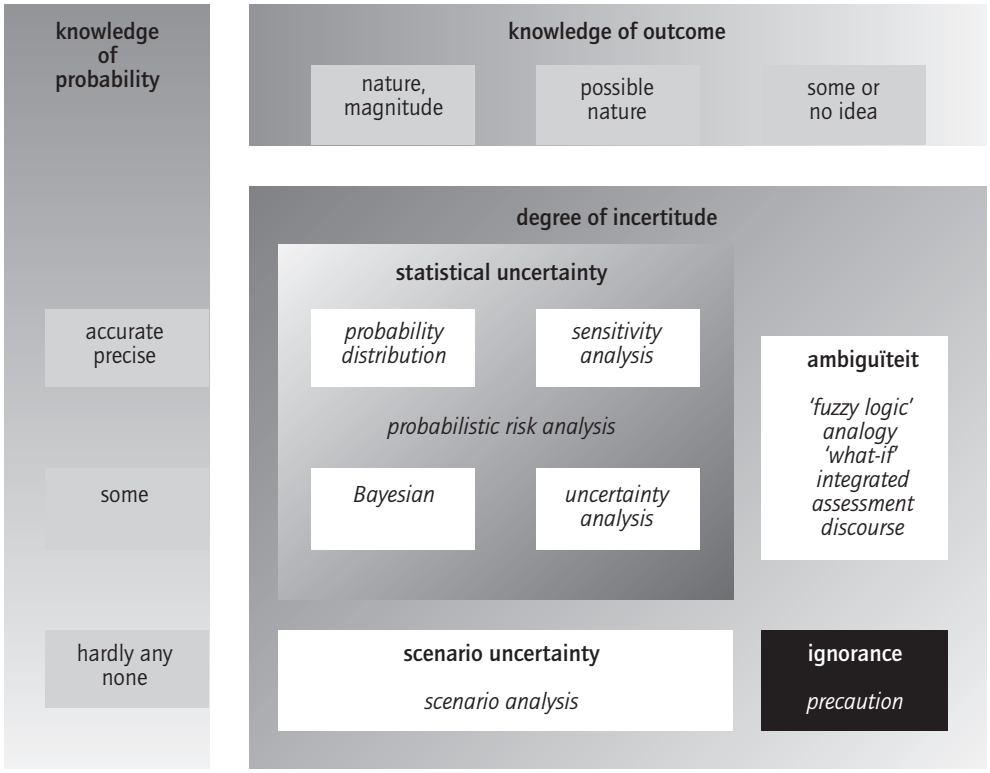

Figure 3-2. Dealing with different types of uncertainty (after 19).

values. When we are dealing with uncertainties on the level of models or constructs, we could apply uncertainty or scenario-analysis. We make our calculations under different assumptions or scenarios to get some sort of hunch of the range of probabilities, nature and magnitude of risk consequences. In case of structural uncertainty (indeterminacy, ambiguity or ignorance), assessment can only be based on analogies, formal logical reasoning, 'what-if' scenario-analysis, semi-quantitative frameworks to deal with vague problem definitions and inexactness ('fuzzy logic'), and of course reflective discussions with stakeholders (participatory sensitivity analysis, interactive uncertainty structuring). In case of ignorance, for scientists modesty would be an appropriate attitude. Here we are in the domain where risk problems cannot be properly described qualitatively, let alone quantitatively and precaution might be a good strategy.

3.2.6 Risk as a social construct: a broader approach

Quality or construct

In the traditional approach risk is conceived and measured as an objective and unequivocal phenomenon. According to this approach the fact that our knowledge is often limited and uncertainties may be huge does not rule out the existence of 'real' risk as quality of human and natural systems that can be objectively determined. More social-psychological or philosophically oriented scientists dismiss this notion of risk. They argue that risks do not lie on the street, around airports or industrial areas waiting for research institutions to get measured and assessed[31]. In this perspective the concept risk is rather a social construct than some sort of natural or physical quantity: a formula collectively put together in the long course of time that enables us to deal with everyday hazards and incertitude. During the larger part of human history we were obliged to arm ourselves against a multitude of threats. The acquirement of a well-developed risk feeling has turned out to be an important survival advantage[32,33]. Apart from knowledge of probabilities, nature and magnitude of undesired consequences these mental constructs comprise a variety of other, often social-psychological aspects: a rich mishmash of daily experience, values, convictions, and political preferences, negotiated and shared with like-minded and congenial groups. This more or less Darwinist view on human risk behaviour is supported by recent discussions on the validity of the rational behaviour model in economics. Do we really act on maximisation of our expected utility? Do we really calculate all possible consequences of all our possible choices, taking a range of uncertainties into account, before we decide? There is much reason to doubt this model is operating in everyday practice[34]. In recent publications persuasive evidence is presented that we confront emerging danger with two different systems: feelings and analysis. The first system is fast, associative, affective, automatic, emotional and is withdrawn from conscious control (similar to a reflex as a short-cut directly through our spinal chord); the second system works with rules for reasoning, algorithms, and formal logic, but is much slower, requires effort, learning ability, and consciousness[32,35,36]. Neurological research indicates both systems, emotional/affective and analytic, including their advantages and weaknesses, their biases and limitations, are necessary in making effective and rational decisions. Thus, the individual assessment of risk is the result of both affective (associative) and analytic processes. Furthermore, if they point in different directions, in most cases the 'feeling' will be decisive in our judgement[37,32,31,38]. Obviously, precisely our affective system is closely connected to typically qualitative risk attributes. Entirely consistent with this line of thought our judgement and perception of risk appears to be domain specific. Our assessment of qualitative attributes, such as voluntariness, perceived control, familiarity and equity differ whether it concerns health care, food or consumer safety, hazardous industry, financial matters, traffic or social safety issues.

So, risk is at least in part a social construct. Social scientists never get tired of pointing out that experts apply all kinds of qualitative and quantitative assumptions in modelling their 'objective' risk problems. And, of course, these assumption are as founded on values and subjective judgements as the 'models' lay-people use assessing the dangers of daily life[31]. However, we have to emphasise the fact the scientific context stands for a certain discipline of argument and is coded by good scientific practice[19].

The relevance of the debate between 'objectivists' and 'constructivists' -slightly accentuated here for clarity's sake- is well illustrated by the seemingly simple choice of risk measures. Even when mortality (rate) is concerned, surely one of the most unequivocal health endpoints we know, we will have to make choices with marked normative consequences. The health loss attributed to certain risk-bearing activities or situations, can be measured by annual mortality, annual loss of life expectancy, or loss of healthy life expectancy in an exposed group. Annual mortality is the most clear-cut measure and guarantees that everybody, young or old, rich or poor, healthy or sick, is regarded as equal and consequently is protected equally. However, at the same time that choice implies that 'acceleration' of death among the very ill with just a small number of days or weeks as it might result from an episode of smog, is of the same value as the accidental death of a father of a number of young children. If, on the other hand, we apply the number of life-years lost as a measure of risk, in principle we should profit from passing on the risk to the elderly, as they have a shorter life-expectancy to loose. From the utilitarian perspective this might be defensible, but surely not from the perspective of social justice. Expressing the health loss in terms of health adjusted life years has the advantage that non-lethal damage to health or well-being can be included in our measure, but it would imply at the same time that people with a chronic illness count for less, as their years are spent in reduced health. In sum, even ostensibly neutral measures are 'value-laden', whether we like it or not; the choice of risk measure can only be derived from normative choices we make as a starting point for risk management.

To highlight this somewhat more we may look at complicated, technological developments, such as the application of genetically modified organisms (GMO) in agriculture. Hazards and advantages regard human health (immune toxicity versus nutritional value), but concern ecological integrity (bio-diversity, 'escaping' genes versus reduced or more specific pesticide use), economy (advantages to producers versus consumers), agriculture (food security, sustainability), as well as ethics (animal well-being, playing God). In addition, several important risk attributes come up, such as indeterminacy, lack of control (in time and space), irreversibility, persistence, inequity, and the potential to cause societal commotion. Its is obvious that al these incomparable aspects will never be captured in one or even a few quantitative risk measures (incommensurability)[v,39]. It is not very likely that the question how society is going to deal with risks associated with agricultural GMO applications is going to be solved by a standard risk approach, applying only metrics and numbers.

Qualitative risk attributes

A large amount of psychosocial research, going back to the sixties, shows that when assessing risk generating activities, people tend to involve many aspects besides probabilities and magnitude of possible damage. Moreover, the latter quantitative aspects often appear to play only a minor role in public risk acceptance. In this the discrepancy in the perception of health risks between experts and lay-people may often originates. One assesses the same risk problems using a completely different set of risk attributes[31,40,41].

[v] Kenneth J Arrow won a Nobel-prize by formally proving that in a multiform society multidimensional preferences cannot be aggregated (and ordered) in a definite analytical way.

Most overviews will contain the following row of aspects.

spatial and time scale, potential magnitude of damage
catastrophic potential
lack of voluntariness ('free choice')
inequity ('who benefits, who runs the risk?')
(perceived) controllability of incidence or consequences (degree of personal influence)
lack of trust in authorities and others carrying responsibility, such as industry (often lack of openness)
unfamiliar, new technology (e.g. as opposed to 'natural threats')
hidden, delayed, and irreversible damage (e.g. cancer many years after exposure, or future generations)
indistinctness over societal benefits of risk generating activity
possibility of identification with victims
harmful intentions of persons of institutions producing risk (crime, terrorism, capitalist, sabotage).

After accidents have taken place, nowadays blameworthiness (e.g. negligence) appears to be an important aspect.

Psychometric research demonstrates that every risk problem has its own unique pattern of scores over the risk attributes[42]. Apart from that, these attributes are often correlated over a range of risk problems. Voluntariness, trust and controllability are associated, just as catastrophic potential, delayed hazards for future generations, and irreversibility. In their early publications Slovic and co-workers used the concept of (perception) factor 'space', a co-ordinate system, based on two higher-order factors: an axis 'dread' (fatal, global scale, catastrophic, irreversible, uncontrollable etc.) and an axis 'unknown' (unknown to science and exposed, not observable, delayed effects, new, etc.)[43]. Other authors propose two other categories of aspects that determine risk assessment and acceptance: 'deontological' and 'consequentialist' evaluation. On the one hand, aspects focused on causation: who is to blame, what ethical principles have been violated? (the past); on the other hand aspects that cover the consequences, what is the nature, what is the magnitude (the future)?[44].

Social amplification?

The risk attributes discussed here are also relevant to a phenomenon that is referred to as 'social amplification'[45]. Relatively innocent incidents provoke an enormous societal turmoil which, at least in terms of damage to public health, ecological integrity or material goods appears to be excessive. Although good scientific evidence is scarce, one mechanism forces itself upon us. Outwardly insignificant incidents are seen as the forerunners of huge, uncontrollable, and irreversible disaster, as an omen of doom[46]. Small incidents with fairly new, unfamiliar technologies, such as genetic modification, bio-industry provoke much more distress than a train calamity with many victims. In some cases the economic losses caused by these phenomena of societal distress and distrust are immense, in the form of reduced trade volumes or/and compensation. Recent examples are not hard to find: SARS (severe acute respiratory syndrome), anthrax spores in the mail, or Creuzfeldt-Jacob disease associated with BSE. Furthermore, this 'signal'-concept offers a good explanation for the success of present-day terrorism, being a poorly understood, unfamiliar, sinister form of danger that can easily be comprehended as an omen for an irrational, and ruthless, global outbreak of an uncontrollable series of catastrophes with exponentially increasing body counts. Relative small investments ('signals') cause enormous societal response[47,48].

Mass media

Of course the role of the (mass) media is important, if not crucial here. Although poorly understood, the

'asymmetry' of the coverage may contribute significantly to some sort acoustical feedback: 'bad news is good news, good news is no news'. It is apparent that the mass media cover risks selectively. Often risks that are rare or dramatic -that have 'story value'- receive disproportionate coverage, while more commonplace risks are downplayed[49].

Recently American researchers introduced the concept of stigma within the framework of social amplification. Amplified by the pervasive power of the media, public concern about health and ecological risks can develop into a new and very significant social phenomenon: 'technological stigma.' Stigmas are put on places, such as transport routes for nuclear waste, products, such as GMO ('Frankenstein') food or blood products, and new technological developments to tell us they should be avoided not just because it is dangerous, but because it destroys a positive condition. They 'signal' that what used to be something good or true is now blemished or tainted. "Stigmas represent a powerful force in our modern industrial society because science, technology, and communications media often interact with the idiosyncrasies of human cognition, perception, and emotion to produce extreme disruption in the lives of industries, products, communities, and people. In many instances the social and economic response is exaggerated, even unwarranted, leading to impacts far more serious than the initial threat"[50]. The resulting aversions can produce enormous economic impacts and social consequences, such as those experienced in recent years with British beef and cloning[51]. However, empirical evidence on the role of the media is often inconsistent. Media, including the Internet, do not only have the power to inflate concern over minor hazards, they may dull concern over serious ones as well[52,53].

3.2.7 Sobering answers to the why-question

The answer to the 'why-question posed in the introduction can be quite simple. We do *not* respond particularly irrationally or inconsistently to (emerging) environmental risks. We only tend to involve many other, often qualitative aspects of risk in our evaluation, such as freedom of choice to be exposed to a danger or not, the feeling we can personally control hazards, or at least the possible consequences, or our perception of societal utility justifying health risks being imposed on third-party bystanders. That may be one of the reasons we do not rank health risks in accordance with hard statistics such as 'annual attributable fatalities'. This juggling around with quantitative and qualitative aspects fits very well with indications from psycho-neurological research that two, sometimes antagonistic systems are operating when we make decisions, an *emotional*, *affective* and a *rational* system (see previous section). Apart from the fact sometimes people simply lack sufficient knowledge or are guided by misperceptions.

Of course, all this doesn't mean we are not prone to bias or a certain degree of subtle deception, even manipulation. Analysis of mass media coverage suggest the overall picture of health risks we get might be somewhat distorted, far from balanced. But then again, that is a fact of life we cope with in many instances or public domains. Or in the words of Baruch Fischhoff: "Attempts to change or adjust perceptions, evaluations or behaviours that technological 'stigma' may produce, presume a moral superiority on the part of those applying strategies to cope with them. This may not be a comfortable or appropriate role for scientists"[54,55].

Before we go on with the 'how-question' of dealing sensibly with risks, first a number of sobering interim conclusions.

The concept of risk consists not exclusively of objectively measurable qualities of systems

Risk is also a social construct; in which qualitative, social-psychological attributes may be decisive in our acceptance of health risks

There is no universal, unequivocal measure to quantify health risk; picking a measure always implicates a choice of normative points of departure for policy making

The result of (quantitative) risk assessment depends largely on the definition of the system boundaries

Therefore different risks can not be compared, just like that

Risk assessments are always uncertain to some extent; incertitude may range from statistical inexactness, construct or model uncertainty and indeterminacy, all the way to ignorance.

However, we still think risks can be adequately characterised, at least to a certain extent, applying a (limited) set of quantitative and qualitative attributes, such as probability, nature and magnitude of consequences, spatial and temporal scale, persistence, irreversibility, inequity, delayed consequences, and potential to provoke societal turmoil. The composition and extensiveness of the set will depend largely on the characteristics of the risk problem.

3.3 A proposition for sensible procedures

3.3.1 Where do we go from here?

3.3.1.1 Both objective 'quality' and 'social construct'

It is not very likely the debate between 'objectivists' and 'constructivists' about the essence of risk will ever be settled in a satisfactory manner. Here, maybe slightly spineless, we choose not the make a choice between both fundamental viewpoints.

If policy makers or authorities merely act on quantifiable measures, involved citizens might feel not understood, not taken seriously and irritated. They will not recognise themselves in a narrow technological definition of the risk problem, and thus will not commit themselves to the diagnosis, the assessment, the decision rule, nor to the proposed policies[56]. On the other hand, the prospect of complete replacement of positive, universal knowledge by democratisation of risk analysis and management isn't particularly attractive as well. "May I have your votes for the earth being flat". Nature and magnitude of health loss, and effectiveness and efficiency of risk reducing measures should always be part of dealing with risks in governmental policy, even if it concerns risks that for one reason or another do not appeal very much to the general public. The introduction of public health policies, involving sizeable programmes dealing with safe drinking water, sewage systems, and proper housing at the end of the 19th century[vi], extended life-expectancy with decades. It was initiated by the work of clever physicians and civil engineers with a scientific, public health orientation, not primarily by democratic views of general public[57]. We deliberately painted the picture in black and white. In practice it is rather a matter of a different emphasis put on quantitative or qualitative aspects of risk by various parties[vii,58].

3.3.1.2 A new role for science

After the sobering conclusions of the previous sections do we have to write of a decisive role of science (including social and gamma sciences) in dealing with risks? Of course not. Science can produce valid quantitative measures of probability and magnitude of undesired consequences. Furthermore in a slightly

[vi] Sometimes referred to as the Public Health Transition or Revolution

[vii] Of course there is no such thing as universal, objective, certain knowledge, but at the same time with stunning ease our 'post-normal philosopher' catches a plane, opens his laptop and starts working while flying at 10,000 feet, and after touch down checks his e-mail, just an hour before presenting a paper claiming that all knowledge is relative.

broader interpretation science stands for 'systematic analysis', a 'sceptical' attitude, 'peer review', 'independence', 'transparency', 'being answerable' and a 'capacity to learn' while doing the job. Not being right all the time in a value free manner, but precisely these, often more procedural attributes of science are of great value in the assessment and management of health risks.

Only science by itself is not enough. As the uncertainty become more substantial, as well as the social stakes and turmoil, the necessity of interaction with society increases as well. An open, critical, and pluralistic discourse with society is inevitable when it comes to the delineation, definition and modelling of the risk problem, the interpretation of uncertainties and dealing with them, and the choice of relevant risk attributes, criteria and decision rules[9,12].

3.3.1.3 Structure of our propositions

The remainder of this chapter will deal with procedures to be followed in 'sensible' risk management. We will first discuss the decision rules that are explicitly or sometimes implicitly used in dealing with risks, and the values behind them. Successively we will discuss their practical, and sometimes philosophical boundaries. Then, finally we will combine the information on risk attributes, and decision rules and propose a typology of risk based on several relevant aspects. Finally for 4 types of risks problems we propose 4 different procedures taking into account different challenges, different goals, different strategies and participation and instruments and decision rules to be applied.

3.3.2 Decision rules

If we want to develop procedures to deal with risk sensibly we will have to consider the rules or principle we have been using in the past to decide about risks. After all, even if we agree on how the risk is characterised, there are still many ways to come to a decision. Morgan identified three important rules in the domain of risk management: right-based, utility-based, technology-based (see below)[59]. Apart from these, the precautionary principle is increasingly prominent in environmental protection[60]. In practise often a mix of these principles is applied[viii].

In a right-based approach every citizen merits protection against risks above a certain level (for the benefit of one group risks cannot be imposed on others, at least to a certain extent). In a utilitarian approach one seeks to maximise utility for society as a whole; or in other words it is about the largest yield in terms of risk reduction (or public health) at the lowest costs (efficiency). A technology-based approach involves neither rights nor costs, but requires the best available technology, or demands a risk level 'as low as reasonably achievable' (ALARA[ix]). Therefore risk reduction is a moving target that parallels technological development. The precautionary principle is not exclusively based on quantification of risk. Often, here more qualitative risk attributes such as complexity, uncertainty and ignorance, a long time horizon, irreversibility (physical as well as socio-economical), uncontrollability, stock externalities, and future scientific progress have to be taken in account, as it deals with short-term strategies in the face of long-term uncertainties[61].

[viii] In the American literature two other principles can be found: 'innocent until proven guilty': we will not act until we are beyond reasonable doubt (in fact the counterpart of the precautionary principle), and 'non-interference': in the end citizens of non governmental organisations themselves will act, in court if necessary (liability rules).

[ix] This is a simplification: in many cases, including the Dutch and British regulations application of ALARA is guided by several social and economical conditions, including costs and benefit considerations, and therefore more than just a technological decision rule.

3.3.2.1 Right-based rules

The Dutch environmental risk policy of disregarding, accepting or prohibiting risk generating activities according to quantified risk criteria is a typical example of right-based decision rules for management. Basically Dutch policy distinguishes a level below which risks can be *neglected*[x] and a *maximal* level above which risks are *no longer tolerable*. In short this means that an activity (or source) is not allowed if one year of exposure increase anybody's probability of premature death with more than one in a million. If this additional probability is below one in a hundred million, it is disregarded[xi]. In between those *neglected* and *maximal tolerated* risk levels risk reducing measures are indicated[17]. Generally speaking in drinking water and food safety regulations similar decision rules are being applied. Residues, additives or genetic modifications may not significantly increase the health risk of consumers, irrespective of economic, social or even public health benefits. The right to protection of every citizen, regardless of age, income, gender, social status and so on is essential. Risks may not be passed on to certain societal groups however great the benefits may be. It is important to note that there is no scientific basis on which we can decide whether a certain risk level is tolerable or can even be neglected. Of course, a mortality risk of one in a million is low, virtually nil, compared to other daily hazards. The average annual mortality risk of a randomly chosen individual in the Netherlands is in the order of 0,01. Indeed, our life-expectancy is around 80 years[xii].

3.3.2.2 Utility-based rules

In a utilitarian tradition one is guided primarily by the trade-off between collective benefit and collective pain associated with policy measures[xiii,62]. What are the expected public health returns (risk reduction) and what are the costs, including opportunity costs? At the present juncture this motto is often interpreted as: 'are we not wasting the tax payer's money?' or 'are these policy measures really suitable (grasping the notion of efficiency as well)?'[63,64]. Especially in health care since the eighties a key-role is imputed on 'efficiency' in the battle against the health care cost getting rapidly out of hand. Economic evaluation research, by now a booming discipline within the public health sciences, is an important instrument supporting discussions on efficiency and suitability in health care. Roughly speaking, economic evaluation aims at proving insight in the relation between costs and health yields in health care and prevention[65]. As we are focussed on risk from a public health perspective in this chapter, we will zoom in a little on this field.

In Box 1 a number of cost-effectiveness assessments are collected of interventions in various domains of public health. Most striking is the enormous variation in cost per quality-adjusted life-year (QALY) saved. Disease prevention is often relatively cheap, just as health promotion and traffic safety. Cure interventions range all the way from cost saving interventions (medical costs that are prevented may be subtracted) to very costly life-years, saved with impressive (and impressively expensive) technology. In addition, several environmental interventions are expensive, at least per life-year saved. A paper that was published in the

[x] In most cases the negligible risk level never made it into practical Dutch risk regulation.

[xi] Of course there are many practical and theoretical problems associated with this approach, e.g. what is premature death? We will all die at sometime!

[xii] Even in the most favourable age group of 10-14 years the annual mortality risk is somewhere between 1 and 6 in ten thousand.

[xiii] Utilitarism was already defined around 1800 by the philosophers Jeremy Bentham and John Stuart Mill: activities are good when they yield more happiness than unhappiness, irrespective of the actor's intentions and motives; much later the utilitarism was shaped in the mathematical-economic form of the 'expected utility theory' (von Neumann and Morgenstern, 1944).

nineties suggested that the cost per life-year saved of a large number of environmental regulations was excessive, such as the reduction of benzene emission in the tire industries costing more than 20 billion dollar per life-year saved[66].

Box 1.

Table 3-2 shows a number of estimates of cost-effectiveness of interventions in different domains of public health. Use was made of a series of databases that have been made available on the Internet by various international organisations (HCRA; US Dept. HHS CDC; NHS EED; Office of Health Econommics HEED; SWOV). In addition, various, sometimes raw, calculations for the Dutch situation have been include. When making such comparisons account must be taken of considerable uncertainties in the calculation of both the costs and the benefits. Significant differences in the calculation method can be related to:

- the measurement of the benefits (clinical measures, life-years saved, possibly adjusted for health)
- the costs that are included (medical care: personnel, equipment, transport, medicines and devices, or also social: productivity, cost of care)
- the situation used as a reference (no intervention or conservative medical treatment)
- time horizon and discount rate.

Furthermore, the efficacy of (medical) technologies can improve rapidly. The statistics must therefore not be taken too literally. They will only provide an indication of the order of magnitude, purely by way of illustration.

Table 3-2. Overview of cost-effectiveness calculations for a series of interventions in different population health status domains (more concise).

Costs: euro/QALY[a]	Intervention
< 0 (cost-saving)	National vaccination programme (DP)[b] PKU test, neonatal heel prick (DP) Screening of pregnant women for syphilis (DP) Influenza vaccination for chronically ill elderly people (DP) Smoke detector in the home (HPt) Help with addiction to smoking (HP) Removal of lead from petrol and paint, stripping lead-based paint coats (HPt)
0-1,000	Mandatory safety belt (HPt) Disease coping training for asthma (MC) Screening and treatment of chlamydia (DP) Practical test for moped and autocycle (low-speed moped) riders (HPt)
1,000-10,000	Chlorination of drinking water (HPt) Specific vaccinations, e.g. meningococcus C (DP) Treatment of mild to moderate hypertension with beta blockers and anti-diuretics (DP) HIV screening of visitors to sexually transmitted disease (STD) clinics (DP) Influenza vaccination for all elderly people (DP) Cholesterol test and dietary advice (DP) Bypass operation (MC) Stroke units (MC) Viagra (MC) Mammography population survey (DP)
10,000-100,000	Heart transplant (MC) Controlling Legionella in (health) care facilities (HPt) Pneumococcal vaccination for the elderly (DP) Kidney replacing treatments (dialysis) (MC) Smear and treatment for cervical cancer (DP) Periodic automobile test (HPt) Treatment for mild to moderate hypertension with ACE inhibitors, etc. (DP) Airbags (HPt) Ban on asbestos in brake blocks (HPt) Helicopter trauma team (MC) Lung transplant (MC)

Costs: euro/QALY[a]	Intervention
100,000-1,000,000	Reduction of radon in existing dwellings (HPt) Neurosurgery for malignant brain tumours (MC) EPO for anaemia in renal dialysis patients (MC) General measures for controlling Legionella in water distribution systems (HPt)
>1,000,000	Measures for reducing industrial benzene emission in the USA (HPt) Measure to reduce dioxin emissions from waste incinerators (HPt) General measures to reduce exposure to ELF associated with electric power lines (HPt) Earthquake-proof dwellings in parts of the USA. (HPt)

a. QALY: according to quality-adjusted life-year.
b. DP: disease prevention, HPt: health protection, HP: health promotion, MC: medical care.

It is important to note that environmental policies often have ancillary benefits in other domains as well, such as sustainable development, technological innovations, or ecological integrity. These benefits are not always taken into account in formal cost-effectiveness analysis[67]. The more qualitative aspects of health risk, as discussed in the previous paragraphs are also hard to implement in formal techniques of cost-benefit analysis. As we have learned, in the case of social amplification this shortcoming can have serious implications[33,47].

3.3.2.3 Technology based rules

Best available technology and as low as reasonably achievable (ALARA) are an important point of departure of Dutch environmental policy. Even when risk-generating activities are below the maximum tolerated risk level one is more or less expected to apply the ALARA-principle. In the Dutch situation the principle has been more or less extended in an utilitarian way to finding a balance between the benefits of risk reduction and the societal costs in an iterative process. The ALARA principle is not without controversy. Provocatively the acronym is sometimes translated into 'as large as regulators allow'. In everyday practise there is often a lack of hard criteria to evaluate the reasonableness or best availability of technology. The best guarantee clean and safe technology will be applied is often stringent regulations. In a limited number of years the automobile industry has accomplished impressive emission reductions, under the strain of societal pressure and strict regulations. The reverse situation applies to the energy sector in the United States: more or less outdated, inefficient technology, at least compared to Western Europe, producing high emissions associated with tolerant regulations[68].

3.3.2.4 Precaution

In this overview of decision rules, the precautionary principle (PP) cannot be overlooked. Since the seventies the principle has slowly gained importance and today it very nearly dominates the international discussions on the environment and sustainability. In the Rio Declaration the precautionary principle (15) is formulated as:

"In order to protect the environment, the precautionary approach shall be widely applied by the 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" (Rio Declaration).*

The basic premise of the precautionary principle is that governments should not wait for conclusive evidence before considering measures to protect citizens, consumers or the environment. Thus, contrary to right-based and utility-based rules the PP is not based on any sort of quantification of risk. Despite the seemingly clear definition, its interpretation has been the source of endless controversy, especially between the United States and the European Committee, whether it concerns growth hormones in beef, the application of genetically modified organisms in foodstuffs or the existence or health effects of global warming. A nasty, critical editorial in the Wall Street Journal referred to the precautionary principle as 'an environmentalist neologism, invoked to trump scientific evidence and move directly to banning things they simply don't like: biotech, wireless technology, hydrocarbon emissions'. In spite of this all, the US is already acquainted with regulation based on precaution for a much longer period than the EU, since the Delaney Clause (1957, revoked in 1996) prohibited detectable levels of animal assay carcinogens in food. The United States were also much sooner in banning substances that are involved in the depletion of the ozone layer, regulations to control 'mad-cow' disease, just as prohibiting the application of DES as a growth hormone in cattle[69]. Nevertheless, contrary to the US administration, since the year 2000 the European Commission has officially accepted the precautionary principle as its point of departure for policy making[70]. A recent analysis shows that neither the European Union, nor the United States can claim to practise precaution more than the other. Furthermore, the fact that both economic superpowers from one risk to the next alternately apply a different degree of precaution cannot be traced back to fundamental transatlantic dissimilarities in politics and policy-making[71].

In its most extreme interpretation the precautionary principle requires absolute proof of safety before innovations can be adopted. On the one hand some authors doubt whether the precautionary principle can ever be a robust decision rule: When can something be marked as a threat, and what damage is serious or irreversible; how much scientific certainty is sufficient, and what is an appropriate or proportional intervention? Nonetheless, it seems precaution corresponds closely with our intuitively, maybe even evolutionary attitude towards risks as we are usually rather safe than sorry[32,72].

Key question is how to manage risk under conditions of imperfect knowledge. Intuitively one is inclined to postpone costly preventive measures until more and better science concerning health risks is available. Experience with climate change policies emphasizes the need to develop a *sequential* approach to decision-making. The choice between moderate or aggressive emission reductions is determined today by foreseen future improvements of scientific understanding of climate change and our ability or flexibility to switch to other energy sources[69].

An example of a very practical way in which the precautionary principle can be violated, was recently discussed by the European Environmental Agency. Analysing the methodological features of experimental and observational health risk research it was concluded that in trying to avoid false positives ('Whoops! It really isn't dangerous after all...'), 'sound science' appeared to be seriously biased towards producing false negatives ('Whoops! It really is dangerous after all....')[69].

3.3.2.5 At the boundaries of the decision rules

It is important to stress that decision rules, and the social objectives behind them, will at some point be conflicting. Most governments in the Western world are especially concerned with distribution issues, equity: everybody's right to a certain level of protection or health care. However, purely right-based policies may go beyond the bounds of affordability or efficiency. Vice versa, if maximisation of efficiency is the main target, that is, aiming for great benefit-cost scores, there are inevitably distribution consequences: individuals or specific populations being treated unfair of even harmed in the name of efficiency[73]. Here

we are confronted with the traditional trade-off between 'equity' and 'efficiency' within the realm of welfare economics[74]. This trade-off can be seen in many domains of public health and health care, for instance when priority is being given to younger recipients with better survival benefits given the short supply of donor organs at the cost of the total patient population. Another example is the choice of screening program coverage: do we aim efficiently at a limited high-risk group, or do choose for a fair inclusion of the total population[75]. Do we give all states or large municipalities a fair share of the federal HIV-prevention pie, proportional to reported AIDS-cases, do we distribute in accordance with cost-effectiveness considerations, or is there some middle ground to promote both objectives[76]. Environmental policies, such as clean air regulations are not primarily aimed at balancing marginal air pollution reductions and associated health gains with marginal social losses (e.g. as a result of opportunity costs). They are aimed at protecting the most susceptible individual, regardless of the costs[73]. But of course the final question is: can we afford this in the end?

In the Netherlands this trade-off between efficiency, equity, and perhaps even precaution is nicely illustrated by the recent political anxiety with respect to Legionella in drinking water systems. In 1999 more than 300 reported cases of this pneumonia-like disease, of which 29 died, were attributed to the improper operation of a number whirlpools and (probably) water atomising machines on a Flora exhibition in Bovenkarspel. Pressed by media and to a lesser extent the parliament, the Dutch government soon developed rather drastic measures to minimise the risk of Legionella infection in the general populations. Referring to the Precautionary Principle the Minister claimed that drinking water infections should not occur in a decent, civilised country. These 'temporary' measures forced local authorities, responsible for sport facilities, nursing and care institutions as well as private owners of sport, fitness and other public places to perform a risk analyses whenever there were hot water installations present. If necessary, technical installations had to be adjusted and every facility was obliged to make a water management plan. As it concerned hundreds of thousands individual facilities and institutions the economic costs of this policy were substantial; estimations ranged from less than one billion to several tens of billions of Euro. For several reasons the efficacy of these general measures was thought be rather limited. The number of reported and confirmed cases of Legionnaire's disease in the Netherlands is relatively modest, around 200 per year, although the true number can be several times higher. At least half of the infections are probably caught abroad (e.g. holidays around the Mediterranean). Furthermore, residential water systems could be a significant cause as well. Crude calculations indicated that, making conservative assumptions, the investments to save one health-adjusted life-year could easily exceed one million Euros. The Dutch Association of Municipalities claimed that the new Legionella control measures would cost 3 Euro per inhabitant, 1 Euro more than their total expenses on infectious disease control of 2 Euro per inhabitant[77].

After a fierce debate, involving several stakeholder parties, it was decided to focus policies on high-risk situations, such as nursing and care institutions with a susceptible population, yielding much better cost-effectiveness scores. Of course implicitly, this means abandoning a pure rights-based approach. A committee of the Health Council of the Netherlands stressed the importance of early diagnosis and treatment, and the necessity of proper focus of other high-risk systems, such as large-scale events where water atomisers are being used[2]. After all, that is where the 'Legionella-case' started in the first place. In *Box 2* a number of other environmental cases are presented.

Box 2. *Cost-effectiveness of risk reducing measures*

For a number of cases here we briefly and crudely estimate the expected costs of risk reducing measures. The estimates are tentative, as they are based on contemporary knowledge and technological state of the art. Cost-effectiveness of technologies often improves with time as a result of 'learning' effects and economy of scale.

Electric power lines
In the Netherlands approximately 11,500 children (in 23,000 houses) are exposed to magnetic fields from power lines that may increase their leukaemia risk. Assuming there is a causal relation, each year 0.2 to 1 extra case of leukaemia can be attributed to this exposure (within this population this is well above the annual accepted risk level of 10^{-6}). Measures to reduce exposure to extremely low frequency electromagnetic fields (ELF) include: 1) optimising the 'clock number' by changing the position of the wires on the masts, 2) replacing three wires on each side of the masts by four (phase splitting), 3) relocate the power lines to a more favourable position, and 4) bury the power lines into the ground. Costs and effects of these measures in the Netherlands are:

measure	remaining houses	total costs (Euro)	costs/house (Euro)
1) clock numbers	15,000	140 million	18,000
2) phase splitting	15,000	450 million	55,000
3) relocation	2,500	2,600 million	128,000
4) underground	1,000	14,000 million	655,000

There are many technological problems connected to burying all power lines in the Netherlands. Furthermore approximately it will cost four times as much as simple relocating the 23,000 houses involved. Implementing the presented possibilities on a local scale may be significantly less expensive, but will never be an example of cost-effective public health polic , .

Indoor radon
Exposure to indoor radon increases the lung cancer risk of inhabitants. As a results of measures to reduce energy losses in the last decades residents have become more airtight, normal ventilation rates have dropped significantly. In dwellings built in 1970 radon concentrations were around 18 Bq/m^3, in dwellings built after 1980 the concentrations are around 30 Bq/m^3. Calculations based on epidemiological models point at around 800 (100-1200) cases each year that might be attributable to radon exposure (roughly 2.2 fatalities per million exposed per year per Bq/m^3). It is important to note that from an international point of view Dutch indoor concentrations are relatively favourable (much lower than for instance in Finland).
Measures to reduce radon concentrations in existing houses are relatively drastic and expensive: ventilation facilities in the crawl space beneath the houses and sealing of the ground floor. Annual costs of that type of measures in existing stock of residents were estimated to be in the order of 680 million Euro (for only 5% of the total stock). Introduction of standards for radiation performance (SPN) in newly built houses would be much less expensive. Adjustment of the ventilation rate and evaluation of construction materials would probably not exceed 620 Euro per new house. Additionally the heating costs would increase with a maximum of 40 Euro per year. This type of measures would cost in the order of 50,000-150,000 Euro/QALY saved. The SPN is not yet implemented in building legislation due to changes of government in the Netherlands. The introduction of the SPN might have complications for free trade within the EU.

LPG stations in residential areas
Due to the proximity of LPG stations, associated with a small but significant chance of explosion, at this moment more than 15,000 people are exposed to a mortality risk exceeding one in a million annually. Within urban areas it primarily concerns houses within 15 and 80 meter around the LPG-stations, as surface of approximately 0.5 hectare. The relocation of LPG-stations will cost around 0.25 million Euro which is in almost all cases much less expensive than pulling down existing houses. The cost of an avoided fatality by means of relocating a LPG-station is in the order of 3 million (±100,000 Euro/QALY).
Including opportunity costs as well a completely different picture emerges. Removing LPG-stations from the built environment means grounds within risk zones is becoming available for building dwellings or offices. Reorganising the existing LPG-stations would yield around 2000 hectare of ground to be developed. In the event building projects are developed here, this would mean a gain of around 25 billion Euro.

Air pollution
Approximately 3000 people die annually as a result exposure to air pollution (fine particulate matter and ozone). Road traffic is the most important source of air pollution in the Netherlands. In recent decades road traffic emissions have decreased substantially, in spite of large volume growth. In the next decade further reductions are expected of new measures. In the period 1985-1995 annual costs were around 500 million Euro yielding a emission reduction of around 4% per year and proportionally 120 to 210 less fatalities: that is 2 to 4 million Euro per life saved. For the period 1995-2010 an amount of 5-10 million Euro per life saved can be calculated. These cost per life saved are 30 to 75 times higher than the costs of traffic safety measures per life saved in the period 1980-2000. It is important to note that traffic

emission reductions have benefits outside the public health domain as well. Reduction of acid and nutrient (nitrogen) deposition has gradually improved the ecological quality of many natural systems in the Netherlands.

Firework storage
The storage of heavy firework within 400 meter distance of the built environment has been prohibited by law for many years. If operation and inspection of permits had been in order the probability of a disaster, such as the one that blew away a complete residential neighbourhood in Enschede, would have been nil. As a result of incomplete enforcement in practise safety risk are much higher than prescribed. This applies not only to firework storage, but to the safety of most large industrial installations. Therefore theoretically resources made available for relocation of storage facilities do not yield extra safety; in practise of course the do.

Looking at *Box 1* and, to a lesser extent, to *Box 2*, should we conclude that in several domains governmental policy is inappropriate and that it is time for a complete change? The US researchers of the 'Harvard Centre for Risk Analysis' are convinced the time is right. They claimed that more efficient investment of prevention money could save more than 200,000 life-years each year[66]. They proposed to start with the most cost-effective intervention, then the next cost-effective and repeat that procedure until the resources as spent. This new voice sounded very well in the choir of members of Congress, corporate groups, and economists who turned cost-effectiveness analysis into a weapon against the regulation idiocy that, in their view, held the United States hostage. They mounted a vigorous and successful campaign to make benefit cost analysis the sole basis for risk management decisions. As a cost-benefit test now often is more or less compulsory in new legislation, at this moment in the United States a fierce debate is going on regarding the validity of this type of calculations[xiv,80,81,82].

As already discussed health and safety policy based on this limited definition of appropriateness may conflict with other important equity aspects, that are treasured values in most Western countries:

good quality and equal accessibility of health care
solidarity with respect to high cure and care costs (people don't choose their 'expensive' disease themselves)
the availability of proper nursing and care facilities, when cure is no longer an option, and
the right to a high level of health protection[64,83].

Another, more pragmatic reason to critically observe simplistic cost-effectiveness consideration in environmental regulation is the phenomenon of 'social amplification', as discussed in the previous section. Small as the quantifiable health losses may be, the economic damages can be immense when consumers all at once start casting doubts on the integrity of industrial products, for instance in the case of BSE and the bio-industry or food stuffs that involve genetically modified organisms[50,55,69,47].

In short, dealing with risks in several domains often is a combination of different decision rules. In some cases these rules are conflicting, e.g. 'efficiency versus equity' or 'precaution versus efficiency'[73,84]. To deal with this politics and policy makers should make explicit, transparent, choices for a balance between efficiency, equity and precaution.

[xiv] This debate is partly provoked by appointment of the Harvard professor John D. Graham, head of the Risk Analysis Unit by the Bush junior administration to become head of the Office of Management and Budget of the White House.

3.3.3 A proposal for risk management procedures

3.3.3.1 Prelude

How are we going to use all the information we have brought together here on the nature of risk problems, the way risks are perceived by social groups, politicians and policy makers and the often implicit rules we apply to deal with them?

The complexity of our society is enormous, and as a result of rapid technological and social changes we have lost sight of what is going on. They have made life less predictable and a truckload of often-contradictory information increases our uncertainty. Individualisation has corroded social control, solidarity and cohesion. As modern citizens we refuse to accept health risk as dumb fate or Divine destiny as our ancestors use to. Instead we demand responsibility from the entrepreneur who benefits from risk generating activities, and of course the government[85,86,87]. When in 1992 a Boeing 747 crashed into an apartment building in a deprived area of Amsterdam, emphasis was still on the unspeakable grief, desperation and possible long-term health effects. Following recent disasters, such as an exploding firework storage that destroyed a complete neighbourhood and a fire in a bar ruining the lives of many young people, politics and the media were predominantly concerned with who is to blame.

On the idea that society can be constructed according to well-designed plans we have started to believe that the government can guarantee our lives to be without risk, without bad luck. As society we are prepared to make great offerings for further reduction of often-futile risks. To reduce the chances of flooding we spent billions of Euros and sacrificed large parts of our much cherished river landscape. Rather unscrupulously tens of thousands of cattle, pigs and poultry are systematically being cleared away simply to restore our faith in the safety of our food (and economic interests) without a clear picture of the real human health risks of BSE, foot-and-mouth disease, hog cholera and so on.

For a number of reasons this perceived control over man-made risks is fictitious. The risk phenomenon is essentially incompatible with total control as unfortunate concurrence of circumstances is by definition ineradicable, especially in a world of increasing complexity. The number of man-made risk generating activities and situations only increases. No stream of regulations cooked up in the Hague where our government resides (or anywhere else) will ever create a zero-risk situation for Dutch citizens. There is simply not enough money, human resource and capacity for inspection of adequate execution of rules and regulation. Another more or less paradoxical reason is the act that citizens tend not to be too worried about what the government wants from them. Although much concerned with safety and health risk issues, citizens tend to dismiss any restrictions of their freedom of action or undertaking[88].

Based on the insights that risks come in many sizes and colours, and that a number of explicit or implicit decision rules guide our dealings with risk, we will define a number of strategies based on quantitative and qualitative aspects of risks. We will start with discussing different possibilities ways for deliberation processes, and the degree to which shareholders should be involved.

3.3.3.2 Types of discourse

Deliberation is an essential element of problem definition, uncertainty management, and balancing decision rules during the process of risk management[20]. Some science-philosophical authors distinguish different levels of discourse. Dealing with relative simple, operational and uncontroversial risk problems, the discourse is often internal, within the agency, the company's risk management unit or policy department. Standard risk assessment tools and management criteria are undisputed and lead to solutions that are accepted by all parties[72,89,90]. In a *cognitive* discourse experts, preferably from involved stakeholder parties argue about an appropriate delineation, definition and explanation of the risk phenomenon, adequate and consistent

criteria for risk assessment. The more complex and uncertain the risk problem, the more multidisciplinary and deliberative the process to arrive at consensus or clarification of dissenting views. If there is uncertainty on the level of constructs or models (paradigms), scientific input is only one aspect of the evaluation procedure. A *reflective* discourse involves clarification of knowledge, but also discussion on normative aspects, such as the right balance between precaution and a harmless until proven dangerous philosophy, between efficiency and equity, or between a risk averse and a risk keen attitude towards technological innovation. Besides scientist also expert representatives of involved stakeholder groups ought to take part in these evaluative discourses, as scientific knowledge as well as societal values are at stake. A participative discourse is the most comprehensive form of deliberation. Here ambiguity and ignorance are substantial and thus the framing of the risk problem, measurement issues and formulation of possible solutions depend very much on values, attitudes and political preferences. Fundamental views on equity, environmental justice, the blessings of technological development and social change are often decisive in these deliberations. Thus this discourse involves all democratic parties as it is aimed at societal consensus on how to define risk problems, in spite of ambiguity, ignorance and often social distrust[72].

3.3.3.3 Four strategies for risk management

In four risk management strategies we will try to combine the insights in the multi-attributive nature of risk, different degrees of incertitude, the limitations of risk analysis, combinations of different decision rules and the types of discourse that are applicable. Tentatively we propose four risk types, everyone with a corresponding risk management strategy (Table 3-3).

Operational decisions with respect to simple risk problems

If there is no discussion on problems of uncertainty, unresolved complexity or efficiency, traditional quantitative methods of risk assessment and management will generally suffice. Calculation of health risk in terms of probability and effect (mortality) can support decisions on risk acceptance, applying standard evaluation frameworks, such as an accepted and a negligible individual mortality risk of 10^{-6} and 10^{-8} respectively according to the Dutch government's 'Premises for risk management'[17].

Table 3-3. Risk management strategies for different types of risk problems with respect to challenge, goal, function, instrument and decision rule[72,90].

Problem	Instrument	Strategy (how?)	Solution (what?)	Goal	Rule
simple, minor complexity	quantitative risk analysis risk comparison	routine, cognitive discourse, involving agency, external experts discussion on the usual interpretation of available data and uncertainty	agreement on causality and effective measures application of general risk criterion (e.g. 10^{-6})	health protection protection of most susceptible individual	rights-based
appropriateness	analysis of cost-effectiveness and cost-benefit analysis of distribution effects historical analogies	cognitive and reflective discourse, involving agency, external experts, stakeholder experts evaluating trade-off between maximising health gains, equity and (opportunity) costs deliberating compensation	well-balanced measures given socio-economic and psychological context	efficient as well as fair health protection	rights and utility-based

Problem	Instrument	Strategy (how?)	Solution (what?)	Goal	Rule
incommen surability controversy	multi-attributive utility analysis sensitivity, uncertainty and scenario-analysis negotiated rule making historical analogies	cognitive and reflective discourse, involving agency, external experts, stakeholder experts social support for problem definition and delineation, relevant risk attributes, risk criteria and decision rules	shared concept of problem and solutions dispel of mistrust, misunderstanding about mutual positions freedom of choice	trust consensus empowerment	utility and technology
ambiguity, ignorance	integrated assessments/system analysis analogy, what-if', fuzzy logic analyse public involvement: citizen panels, scenario workshops, focus groups etc. value-analysis	participative discourse, involving agency, external experts, stakeholder experts, democratic parties seeking consensus in society avoid postponement of management technology control: diversity, containment, flexibility, substitution development, transition management close interaction between scientific development and policy formulating (sequential, flexible)	'democratic' resolving of conflicts of values, preferences, concern, visions	maintaining resilience, flexibility, freedom of choice 'no-regret' broad social acceptance sequential decision making	technology and precaution

The discourse is internal or cognitive among experts from the agency, advising institutions and, if necessary, from involved parties (e.g. industry). Crude costs-effectiveness analysis can help decide whether the Euro for risk reduction is well spent. Main objective is *health protection of the most susceptible individual* from the perspective of everyone's right to a high level of protection. Measures should be aimed at the reduction of the additional probability of health loss to a generally accepted level in a more or less standard operational manner (the additional annual mortality risk of one in a million is a very good deal in terms of public heath). Good examples are the setting of exposure standards for toxic compounds in ambient air, at the workplace or for food additives or residues.

Appropriate decisions in risk management

However, 'free lunches' are scarce. A rights-based approach can be excessively expensive (total or per life-year saved) or conflicting with other interests highly valued in society (including opportunity costs), e.g. safety around large airports, Legionella in drinking water systems, pathogen-free slaughterhouses or natural toxin-free foods. Although calculated health risks (or losses) are a good indicator of the risk problem, shared by all parties, the proportionality of investments needed to save lives, life years or health adjusted life-years is disputed.

Typically in these cases a balance has to be found between every citizen's right to a high level of health protection and precaution on the one hand, and efficient use of collective resources for public health on the other. As shown in *Box 2* the cost-effectiveness of risk reduction can be significantly

improved by more specific measures aimed at high risk groups (Legionella in drinking water systems), or by focussing on new situations, instead of already existing ones (indoor radon, noise or air pollution abatement).

Finding the right balance between efficiency and equity of course is not a matter of science. Based on good scientific analysis of risk reduction, cost-effectiveness, and distribution effects, a reflective discourse involving representatives of social parties involved should lead to a broadly accepted compromise, which may include compensation for citizens who get the worst of it. An important consequence may be that not all citizens are equally protected, but at the same time collective resources are not wasted on inefficient risk reduction.

To facilitate discussion on alternative levels of acceptable risk among stakeholders, analysis of historical analogies may be helpful. Collective mortality risk acceptance that has taken shape in society over a number of years, as well as the resources spend on risk reduction may be a function of important qualitative risk attributes such as voluntariness of exposure, perceived controllability or familiarity of technology. Historical analysis may teach us something about the relation between qualitative risk attributes profiles and 'solidified' risk acceptance (and willingness to pay)[91].

In medical technology assessment implicitly and sometimes explicitly a limit of around 45,000 Euro per QALY is applied for new interventions that can reasonably be admitted as cost-effective (see also Box 1) . Of course these types of cost-effectiveness limits will differ from one domain of public health care to another. Nevertheless they can help in making considerations more explicit. Of course further empirical research is required to investigate the applicability of this idea.

Controversial, tactic decisions in risk management

Traditional, quantitative risk management will also fail when probability and health effects (especially mortality) are not the issue in the social commotion; when calculations of mortality or morbidity risk simply do not represent the risk as perceived by the public involved. Good examples of such risk problems are the antenna masts for cell phones (GSM), electricity power lines and possible child leukaemia risk, BSE and endocrine disrupters. The acceptability of nuclear power facilities is often discussed in terms of very small probabilities of a serious accident, while the public is primarily concerned by the magnitude of the consequences. The same goes more or less for aircraft accidents, especially after the 'Bijlmer' catastrophe in Holland: experts may claim that thanks to improvement of aviation technology the probability of another accident is decreasing. However, people are primarily worried about the next airplane hitting a crowded residential area. 'Mad cow' disease among European cattle did not cause great turmoil among the public solely as a result of a high number of fatalities. Qualitative attributes have been much more important, such as the terrible fate of young Creutzfeldt-Jakob disease patients, the stigma of BSE as an omen of great disaster brought down upon us by a rationalised bio-industry that turned herbivore cattle into 'carnivores'.

In these cases, risk management procedure should aim at achieving consensus among involved parties on problem definition and delineation, on risk attributes that should be taken in consideration and on efficacy, efficiency, and distribution effects of measures. The main purpose of the procedures is beyond simple health risk reduction, it is about appropriate deployment of resources, without loosing sight of social justice, and in some cases it is merely about gaining trust, decreasing commotion among involved citizens or about well-argued precaution.

Strategic decisions in ambiguity or ignorance

To the extent that ambiguity increases, just as complexity and the possible magnitude of (health)

consequences the management approach will go beyond traditional quantitative risk management procedures. Problem structuring, approaches of risk quantification, dealing with uncertainty and ignorance, application of precaution, and the design decision procedures will have to be developed in close interaction with society. Science is no longer advisor or even 'great arithmetician', but rather 'facilitator'. This implies that politicians will have to decide in explicit uncertainty. The main objective is to arrive at a broadly accepted definition of the risk problem, an approach to 'measure' and monitor the nature and magnitude of the risk, and a framework for making decisions in pace with developments in scientific knowledge. The Intergovernmental Panel for Climate Change (IPCC), comprising not only scientific analysis, but political and socio-economic evaluation as well, is a good example of such a 'post-normal' approach[93].

3.4 Discussion

Obviously the framework outlined in table 3-3 is rather theoretical and has to prove itself in everyday practice. Several exercises with experts from different backgrounds and with stakeholder representative indicate that the application of this type of framework may at least help to structure the available information, to identify the crucial aspects of the risk problem, to focus on the criteria that matter, and in the end to come up with definitions of the risk problem and possible solutions that are shared by different stakeholders[94,95]. But of course these exercises do not reflect the 'stubborn' practice of everyday environmental risk regulation and maintenance. Ultimately the authorised government will have to decide on which measures to take.

The State Secretary for the Environment has recently launched new proposals for 'dealing with risk in a sensible manner' in the Dutch parliament, especially with regards to a number of difficult dossiers, including indoor radon, power lines and base stations for mobile phones. The memorandum is consistent with some of the ideas discussed here. The State Secretary proposes explicit evaluation of costs and benefits, participation of citizens, explicit responsibilities for business, industry and citizens, and transparency in decision-making[96].

An important aspect of the proposed procedure may be a good a priori appraisal of the risk typology and corresponding strategy. If one starts too high on the 'risk ladder' much time, energy and resources may be wasted. If one starts too low, one might provoke unnecessary escalation, social disruption and loss of esteem for the government. Case studies will provide better knowledge of how to deal with risk typologies, and risk strategies in practise, the equivocally and acceptance by involved parties, to finally arrive at clear rules of argumentation, criteria and analogies.

Several of the approaches presented in table 3-3 require community participation, as is also advocated in the Gothenburg consensus paper[97]. Community participation may often have a positive impact of the success of project development and implementation, it may change the attitude towards causes of ill health, promote a sense of responsibility and confidence among involved groups or individuals. The involvement in the policy process may decrease the alienation among excluded groups and improve relationships with professional policy makers. However, participation is time-consuming, communities of busy people may question the value of invested time and effort. There may be problems with the legitimacy of those representing the views of often-heterogeneous communities. Some stakeholders will mobilise professional, well-informed participants much easier than others. Furthermore, the difficulty of organising participation will often conflict with the speed required for timely health impact assessment and management. So, community participation may be very appealing, but practically difficult[98,99].

Obviously, more emphasis on the societal costs of health risk reducing measures may improve policy

making. However, cost-benefits should be analysed from a comprehensive perspective (e.g. large enough time-horizon, broad spectrum of possible costs and benefits), encompassing not only the present costs of implementation, but also possible future, ancillary benefits of technological innovation, stimulated by measures to protect the environment. Ex post analyses indicate that ex ante cost assessments tend to overestimate the actual costs, often as a result of unanticipated gains of innovation (e.g. emission reduction of CFC and SO_2, asbestos ban, and occupational lead exposure reductions in the United States)[100,101].

Often prevention at new situations will prove much less expensive than solving problems at existing situations. This applies to building underneath power lines, radiation standards for construction materials, as well as to building within safety risk zones or explicitly evaluate health aspects in urban planning. Better enforcement of existing guidelines may in many instances be a very cost-effective solution to avoid expensive retrofit measures in existing situations.

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4 An aggregate public health indicator to represent the impact of multiple environmental exposures[i]

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In this paper we present a framework to aggregate divergent health impacts associated with different types of environmental exposures, such as air pollution, residential noise and large technological risks. From the policy maker's point of view there are at least three good reasons for this type of aggregation: comparative risk evaluation (e.g. setting priorities), evaluation of the efficiency of environmental policies in terms of health gain, and characterizing health risk associated with geographical accumulation of multiple environmental exposures. The proposed impact measure integrates three important dimensions of public health, viz. *life expectancy, quality of life,* and *number of people* affected. Time is the unit of measurement. 'Healthy life years' are either lost by premature death, or by loss of quality of life, measured as discounted life-years within a population.

Using data from the fourth Dutch National Environmental Outlook we estimated that the long term effects of particulate air pollution account for almost 60% of the total environment related health loss in the Netherlands we modeled here. Environmental noise accounts for 24%, indoor air pollution (environmental tobacco smoke, radon, and dampness), as well as lead in drinking water for around 6%, and food poisoning (or infection) for over 3%. The contribution of this set of environmental exposures to the total annual burden of disease in the Netherlands is less than 5%.

[i] Based on: Hollander AEM de, Melse JM, Lebret E, Kramers PGN. An aggregate public health indicator to represent the impact of multiple environmental exposures. Epidemiol 1999; 10: 606-17. Prüss A, Corvalán C, Pastides H, Hollander AEM de. Methodological considerations in estimating burden of diseases from environmental risk factors at national and global levels. Int J Occup Environ Health 2001; 7: 58-67. Havelaar AH, Hollander AEM de. Teunis PFM, Kranen HJ van, Versteegh FM, Koten JEM van, Slob W. Balancing the risks and benefits of drinking water disinfection: Disability Adjusted Life-Years on the Scale. Environ Health Perspect 2000; 108: 315-21.

4.1 Introduction

As we have seen in chapter 3, the impact of hazardous environmental exposures on human health can take numerous shapes of various severity and clinical significance. Among the many responses that have been attributed to environmental exposures, are disturbed cognitive development in children, several types of cancer, reduced fertility, immuno-suppression, severe noise annoyance and associated sleep disturbance[1,2,3]. During air pollution episodes well-studied human responses range from slight reversible lung function deficits in virtually everyone exposed, to aggravation of symptoms among asthmatics, and from hospital admission of patients with cardiopulmonary disease to the premature death of some of the very weak[4,5,6] (see *figure 1*[7,8]).

Most risk measures that are commonly used in quantitative risk assessment and risk management fail to address this diversity as they are primarily geared to *probability*, rather than to the *nature* and *magnitude* of adverse health consequences[9]. Probabilistic risk measures, such as the annual mortality risk associated with a certain exposures, appear unambiguous and easy to comprehend. Therefore, they are often applied as the most suitable criterion in the risk management process[10]. However, in some instances these measures may be inadequate for decision making, as they do not pertain to the full range of relevant health dimensions associated with a certain environmental health problem. In these cases incorporating various relevant health attributes in quantitative risk assessment may improve the decision making process[11,12,13].

In this paper we present an aggregate health impact indicator to deal with the diverging environmental health impacts of various types of environmental exposures. We developed this indicator in

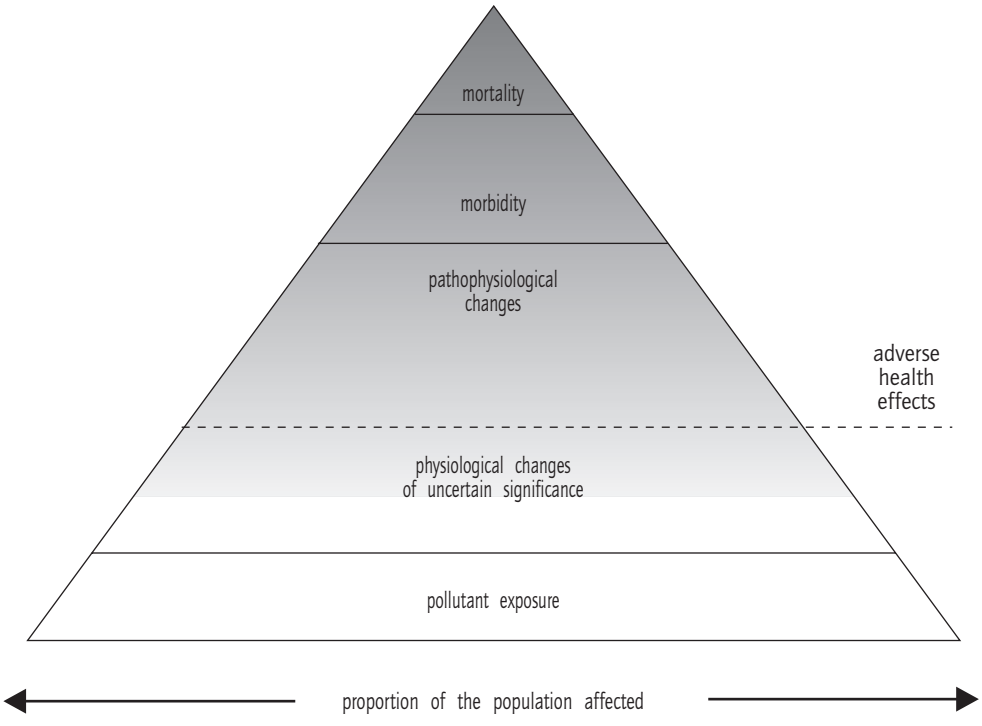

Figure 4-1. Schematic representation of the distribution of air pollution responses in the population[6].

the framework of the Fourth Dutch National Environmental Outlook, which was published in 1997. These outlooks are produced every four years by the National Institute of Public Health and the Environment (RIVM) to assess the current and future state of the environment. Several indicators are applied to describe demographic and economic developments, sustainability, pollutant emissions ('pressure'), environmental quality ('state'), as well as ecological and public health loss due to environmental deterioration ('response'). The efficiency of environmental policies is explored by means of scenario study, in which obviously the impact on public health is one of the key issues[14]. The last National Environmental Outlook (2000-2030) was published in 2000[15].

From the point of view of policy makers, for which these outlooks are produced in the first place, an aggregate health impact indicator may serve as some sort of 'public health currency unit' to

enable *comparative* evaluation of environmental health risks of a multitude of pollutants and, consequently, the setting of priorities ('how bad is this exposure?')

evaluate the *efficiency* of different policy options ('how much health do we gain by implementing this policy compared to other options?')

assess the health significance of geographical *accumulation* of multiple environmental risk factors ('how do we evaluate the multiple environmental stress in this neighborhood from a public health point of view?')

improve risk communication.

4.1.1 Risk comparison

In traditional quantitative risk analysis, health risks are measured and, often implicitly, compared in terms of mortality risk. Risk managers all over the world use the annual mortality risk criterion of 10^{-6} as a limit of acceptability[16,17,18]. However, gradually it has become clear that the one 'annual ten to the minus six' risk may differ substantially from the other in several important aspects[19]:

In terms of loss of *life expectancy*. 'Precipitated' mortality during particulate air pollution episodes involving the old and weak may in many cases cost several months of unhealthy life at the most[20,21,22,23,24], while the impact associated with fatal accidents involving individuals with a 'random' age distribution may amount to a loss of many healthy years[25].

In terms of *non-lethal health outcomes*. In Western society public health focus has gradually changed from life expectancy to health expectancy, i.e. postponing as long as possible or mitigating the functional limitations that come with chronic disease of older age and that affect the ability to cope with the demands of daily life[26,27,28]. More or less the same goes for the health impact of environmental exposures. In many cases these do not involve mortality, often not even morbidity, but rather aspects of the quality of life, such as severe annoyance, sleep disturbance, aggravation of pre-existing disease symptoms or risk perception[29]. This implicates that mortality risk might often not be the most appropriate indicator of environmental risk.

4.1.2 Policy efficiency

Rational policy making involves balancing the costs and benefits of different environmental policy options. This refers not only to the best buy in risk reduction technology, but may also concern risks which are generated as a by-product of measures to mitigate the original health risk. An elegant example of this kind of dilemmas in risk management is the case of drinking water chlorination. At this moment chlorine is probably the most efficient disinfectant available in drinking water production and distribution. On the other hand there is some indication that chlorination or ozonisation of drinking water might increase the

consumer's risk of cancer. Some of the by-products appear to be mutagenic; moreover weak but fairly consistent indications for some carcinogenic potency have emerged from epidemiological studies. Consequently good risk management requires a comparison of the short-term health gain, avoiding water borne infectious disease, against possible health loss in the long run due to an increase of cancer incidence[30,31,32,33,34,35]. Of course the risk management would have to consider the validity of both risk assessments as well, but this is not the issue here.

4.1.3 Geographical accumulation of environmental stress

Recently the geographical accumulation of poor environmental conditions in 'deprived' urban areas was identified as one of the major environmental problems in the Netherlands[36]. Spatial clustering of societal functions, such as housing, working, transportation and recreation, combined with unfavourable developments in urbanisation (e.g. socio-economic segregation) have lead to the accumulation of health risk factors in certain neighbourhoods. Among these are pollution of air and soils, noise and odour pollution, traffic congestion, and bad housing[14,15]. An aggregate measure may facilitate explicit evaluation and comparison of environmental conditions across different geographical locations.

4.1.4 Risk communication

Many public controversies concerning the assessment and management of environmental health risks in the past have shown that the expert and public perception often differ considerably. The discrepancy between expert and lay judgements may be largely due to differences in conceptualisation and definition of risk problems[10,37]. In the words of Fischhoff, addressing risk assessors and managers: it is not just a question of "getting the numbers right", "telling them the numbers" or even "explaining what the numbers mean"[38]. Risk communication should be an interactive, two-way process, taking account of existing audience knowledge, interests and behaviour. Putting environmental health impact assessment into a public health perspective, relying on accepted basic concepts, such as loss of life and health expectancy, might certainly improve the debate.

In the next section we will present the framework for quantitative aggregation of different aspects of health impact of environmental exposures. The feasibility of the approach is demonstrated, calculating the health loss associated with a number of important environmental exposures in the Netherlands. These calculations are based on health impact estimates for the Dutch population, many of which were produced in the framework of the fourth National Environmental Outlook[14]. Data, methods and assumptions used in these calculations will be discussed in more detail in a separate publication[39].

4.2 Concepts and methods

4.2.1 Measuring health using time as a metric

To estimate the health loss associated with several environmental exposures, we used an approach largely based on the 'burden of disease' measure that was developed by Murray and Lopez. To assess the global disease burden and, consequently, the health policy priorities in different regions in the world they applied disability adjusted life years (DALY's). This health impact measure combines years of life lost and years lived with disability that are standardised by means of severity weights[26,40]. Our adaptation of the DALY-concept was inspired by the notion that the multiform health loss due to environmental exposure is fairly well characterised by three dominant aspects of public health, viz.

quantity of life (life expectancy)
quality of life, and
social magnitude (or number of people affected).

Thus, environmental health loss is defined as time spent with reduced quality of life, aggregated over the population involved (figure 4-2). Based on this concept health loss attributable to environmental exposures can be assessed by:

defining responses which are associated with environmental exposure
calculating the number of people affected (N),
estimating the average duration of the response, including loss of life expectancy as a consequence of premature mortality (D), and
attributing discount weights to the unfavorable health conditions (S).
calculating the annual number of DALY's lost, using the equation:

$$DALY_{exp} = N*D*S$$

4.2.2 Aspects of health: weighing environmental burden of disease

To attribute weight to environmental health impacts we took much advantage of both the Global Burden of Disease project[41], Dutch Burden of Disease Project and the European Disability Weight Project[42,43].

Key question in any attempt to quantify health loss using one common denominator is 'what is health'? The concept of health may differ from era to era, from region to region, since it reflects changes or differences in social and cultural beliefs, in medical technology, and economic conditions. Already in 1946 the founding charter of the World Health Organisation stated that health is not 'merely the absence of disease and infirmity'. An individual's capability to function well physically, mentally, as well as socially is the central issue in most papers on health status measurements[44,45]. Or, to put it in another way: the ability to cope with the demands of everyday life[46].

Initially Murray and co-workers applied disability weight definitions which were primarily based on functionality, the (dis)ability to perform 'activities of everyday life' in four domains: procreation, occupation, education and recreation[26]. The approach was received with a fair amount of criticism, some involving the procedures of attributing weights, other the fact that the definitions did not fully comprise important dimensions of health such as pain, distress, discomfort, anxiety and depression. Aggregated scores would not adequately reflect preferences of various 'stakeholders'. To meet these objections in their revision of the DALY-approach Murray et al. applied the concept of 'indicator conditions'. Using formal instruments to measure health preferences, 22 indicator conditions were given weights in series of consensus meetings involving physicians and public health scientists from different regions. These states reflected several distinct attributes of non-fatal health outcomes, such as large physical manifestations or limitations, psychological ands social limitations, pain, as well as disturbed sexual and reproductive functions. These indicator conditions were used subsequently to attribute disability weights to most other states (see *table 1*).

Table 4-1. Revised disability classes; indicator conditions and severity weights for the Global Burden of Disease Project[47].

class	indicator conditions	weight
1	vitiligo on face, weight-for-height less than 2 SDs	0.00-0.02
2	watery diarrhoea, severe sore throat, severe anemia	0.02-0.12
3	radius fracture in a stiff case, infertility, erectile dysfunction, rheumatoid arthritis, angina	0.12-0.24
4	below-the-knee-amputation, deafness	0.24-0.36
5	rectovaginal fistula, mild mental retardation, Down syndrome	0.36-0.50
6	major depression, blindness, paraplegia	0.50-0.70
7	active psychosis, dementia, severe migraine, quadriplegia	0.70-1.00

In order to estimate the burden of disease for the Dutch population, Stouthard et al selected 55 diagnoses of greatest public health significance in terms of number of patients, and years of (healthy) life lost. These diagnoses were divided in more or less homogeneous 176 health states of various severity (and/or progression)[42].

According to the protocol designed by Murray et al.[47] physicians with ample clinical experience were invited to perform the weighing procedures, which consisted of two steps. At first they evaluated a selection of 16 representative indicator states, using two varieties of a person-trade-off approach. A visual analogue scale (VAS) was added as another instrument of valuation, mainly for the purpose of validation. Furthermore, a standardised classification of the indicator states according to EuroQuol (6D) was provided to assist the panel members[48]. This classification instrument involves a three-point scale for six health dimensions, viz. mobility, self-care, daily activities, pain/discomfort, anxiety/depression and cognitive functions. Using the indicator states for 'calibration' the remaining health states were valued by means of interpolation (ranking health states similar to one or in between two consecutive indicator conditions).

Not all health states associated with environmental exposures were valued in both exercises described above. For these states, among which were 'serious annoyance' and 'sleep disturbance', we drafted definitions based on environmental epidemiological reports and expert judgements. Subsequently these state definitions were interpolated by a panel of environment oriented physicians, employing the scale of 'calibration' states, which was drawn up by Stouthard et al.[42] (see figure 5-4, chapter 5).

4.3 Health impact assessment

4.3.1 Environmental outlook

Most health impact assessments produced within the framework of the National Environmental Outlook are based on a three steps procedure[49,50,51,52,53,54,55]:

assessing population exposure distribution

defining health outcomes and quantifying the association between exposure and response

risk characterisation: estimating the number of people affected, duration and severity of the condition.

4.3.2 Exposure assessment

Depending on the nature and availability of data population exposure was assessed by:

combining time-activity patterns for sub-populations with concentration distributions in micro and macro environments[56,57](carcinogenic air pollutants, PAH, radon)

combining data on population density with environmental quality by means of Geographic Information Systems (noise, fine particulate matter and ozone air pollution, large technological accidents)[55]

estimating (often dichotomous) distribution of exposures based on monitoring programs (environmental tobacco smoke, home dampness, lead in drinking water).

4.3.3 Exposure-response modeling

Quantitative exposure-response data were derived from either occupational or environmental epidemiological studies. In some cases data from animal assays were considered as additional evidence. For every environmental exposure we selected a set of response variables, considered plausible as well as significant to public health and for which enough data were available. For each response variable a quantitative association with exposure was modeled based on (meta-)analysis of available studies[58]. The nature of the mathematical models depended largely on the definition of exposure (dichotomous, exposure categories or continuos exposure).

4.3.4 Risk characterisation

We estimated the number of disability adjusted life years that is lost per year of exposure, using the following equation:

$$DALY_{e.e.} = \sum_{i=1}^{n} \sum_{k} I_k * f_k(RR_i, C_i) * S_k * D_k$$

in which

$DALY_{e.e.}$ = health loss related to n environmental exposures, measured as disability or quality adjusted life-years per year of exposure,

$f_k(RR_i, C_i)$ = a set of functions (including exposure C_i and associated relative risk measures RR_i) representing the population attributable fraction (PAF) of condition k,

I_k = annual incidence of response k, S_k = severity factor discounting time spent with the condition (see previous paragraph, D_k = duration of the condition; in case of premature mortality: loss of life expectancy.

To arrive at the number of people affected we calculated population attributive fractions (PAFs) by combining population exposure distributions with quantitative exposure-response information, applying the following equation (or one its derivatives):

$$PAF = \frac{\sum_{i>0} (RR_i - 1) * p_i}{\sum_{i \geq 0} RR_i * p_i}$$

in which RR_i = relative risk in exposure class i and p_i = exposure probability in class i.

Subsequently we estimated the number of people affected by a certain response by multiplying the PAF with annual incidence figures, obtained from Dutch health statistics, primary care registrations, or specific surveys. In some instances the number of people involved could be derived directly from (routine) registrations, such as domestic and traffic accidents, and food-borne acute gastroenteritis[36].

Depending on the nature of the pollution-related condition, duration was determined from case definitions used in the epidemiological studies involved, e.g. respiratory symptoms, hospital admissions, and severe noise annoyance. In case of well-defined diseases, duration was calculated from Dutch prevalence and incidence statistics, implicitly assuming similarity among average cases and cases attributable to environmental exposures. The loss of life expectancy due to premature mortality was calculated with data from vital statistics (using standard life-table techniques)[21,39].

For each exposure-related disease a severity factor was composed as a prevalence-weighted average of compound disease states[59], assuming that the environmental exposure had no effect on disease prognoses. In some cases weights referred to transition from one severity state to another, for instance from mild to severe asthma ('aggravation of asthma')[4].

4.3.5 Uncertainty analysis

We analyzed the uncertainty in the calculations of environmental DALY's by means of Monte Carlo techniques[60,61,62]. In Monte Carlo simulation model input parameters are treated as random variables. These are combined by means of computerized random sampling to estimate distributions for one or more output variables. For each of the input parameters, such as population exposure, exposure-response function estimates, average duration of the response and discount factors, a probability distributions function was estimated, representing parameter uncertainty. These distribution functions were based on available measurement statistics for each of the parameters. In few cases, where data were lacking, we relied on expert judgment (see 39 for detailed descriptions of input probability distributions). Subsequently, an output distribution for the different environmental DALY losses was estimated by iterative (Latin hypercube) sampling from each of the defined parameter distributions, followed by recalculation. This repetitive recalculation process is run until mean, standard deviation and percentile values of the output probability distribution are stable (change less than a predetermined threshold percentage of 1% when a set of new 'realizations' is added)[39]. The distribution of the output variable represents the uncertainty in the point estimate of the annual loss of DALY's attributable to the different environmental exposures. Here we will present the 5- and 95-percentiles of this distribution as a measure of uncertainty.

4.4 Results

For 18 environmental 'exposures' we considered the data to be of sufficient quality to calculate the annual, attributable number of DALYs lost. We included traffic and domestic accidence, although one might argue these are not typically environmental health risks. On the other hand, as familiar, established high risks they do add some public health perspective. The results of our calculations are presented in *table 2* and *Figure 4-2*. In *Figure 4-2* the point estimates as well as the 5 and 95-percentiles of the probability distribution for the number of exposure attributable DALY's as derived from the Monte Carlo analysis are shown as a measure of uncertainty. In most cases this uncertainty range is substantial, but less than one order of magnitude, which is considered moderate in the framework of health risk analysis[61].

In *Figure 4-3* we present a series of short-term responses associated with exposure to particulate matter, ranging from respiratory symptoms to premature death. As is clearly shown, relatively mild responses, such as respiratory illness or aggravation of symptoms may have high scores, because of the large number of annual events.

Table 4-2. Summary of disability adjusted life-years lost to selected environmental exposures in the Netherlands.

Environmental factor	Health outcome	#[a]	S[b]	D[c]	DALYs	5-/95%-tile
particulate	*mortality*					
air pollution	- total	15594	1.0	10.9[d]	169000	72800-276600
long-term exposures	- cardiopulmonary mortality	8041	1.0	8.2[d]	65750	40500-93200
	- lung cancer	439	1.0	13.0[d]	5400	-9740-20650
	morbidity					
	- chron. resp. sym. childr.	10138	0.17	1.0[f]	1710	300-3600
	- chronic bronch. adults	4085	0.31[e]	1.0[f]	1920	170-4450

Environmental factor	Health outcome		#[a]	S[b]	D[c]	DALYs	5-/95%-tile
particulate	*mortality*						
air pollution	- respiratory		218	0.7[g]	0.25[h]	37	2-114
short-term exposures	- coronary heart disease		253	0.7[g]	0.25[h]	42	2-183
	- pneumonia		191	0.7[g]	0.25[h]	33	3-94
	- other		452	0.7[g]	0.25[h]	92	0-351
	hospital admission						
	- respiratory		3520	0.64	0.038[i]	86	25-195
	- cardiovascular		6060	0.71	0.038[i]	164	48-380
	emergency room visits						
	- respiratory		32500	0.51	0.033[i]	584	0-1756
	aggravation of asthma						
	- asthmatic attacks		212000[j]	0.22[k]	0.005[l]	253	-76-751
	- use of bronchodilators		530000[j]	0.22[k]	0.005[l]	630	133-1290
	aggravation of respiratory symptoms						
	- upper respiratory tract		237500[j]	0.05	0.02	215	8- 555
	- lower respiratory tract		94300[j]	0.21	0.04	760	57-1881
	affected lung function						
	- decreased FEV1 >10%		548000[j]	0.000	0.003	0	
ozone air pollution	*mortality*						
	- respiratory		198	0.7[g]	0.25[h]	33	0-121
	- cardiovascular		1946	0.7[g]	0.25[h]	340	22-1029
	- pneumonia		751	0.7[g]	0.25[h]	131	9- 410
	- other		945	0.7[g]	0.25[h]	250	0-1101
	hospital admission						
	- respiratory disease		4490	0.64	0.038[i]	103	9-281
	emergency room visits						
	- respiratory disease		30840	0.51	0.033[i]	550	44-1520
	decreased FEV1 >10%		2753000[j]	0.000	0.003	0	
PAH (BaP)	*lung cancer*	-morbidity	17	0.43[e]	2.9[m]	16	1- 32
		-mortality	17	1.00	13.5[d]	224	20-460
benzene	*leukemia*	-morbidity	5.3	0.83[e]	2.7	12	2-29
		-mortality	5.3	1.00	21.2[d]	113	18-278
ethylene oxide	*leukemia*	-morbidity	0.1	0.83[e]	2.7	0,2	0- 0,4
		-mortality	0.1	1.00	15.1[d]	2	0,2-2,9
vinyl chloride	*hepato-angiosarcoom*						
.		-morbidity	0.8	0.53	4.4[m]	2	0.2- 3.6
		-mortality	0.8	1.00	13.3[d]	10	1.0-19.0
1,2-dichloroethane	*cancer*	-morbidity	0.1	0.53	4.4[m]	0.1	0.0-0.2
		-mortality	0.1	1.00	13.3[d]	0.7	0.1-1.3
acrylonitril	*lung cancer*	-morbidity	0.1	0.43[e]	2.9[m]	0.0	0.0-0.1
		-mortality	0.1	1.00	13.5[d]	0.7	0.7-1.3
radon (indoor)	*lung cancer*	-morbidity	122	0.43e	2.9[m]	111	60-170
		-mortality	122	1.00	13.5[d]	1645	970-2320
damp houses	*lower respiratory dis.*						
	- children		16920[j]	0.21	0.04	135	21-292
	- adults		55630[j]	0.21	0.04	440	70-970
	asthma						
	- children		3400	0.08[e]	1.0f	270	147-425
	- adults		10534	0.08[e]	1.0f	840	450-1320

Environmental factor	Health outcome		#[a]	S[b]	D[c]	DALYs	5-/95%-tile
ETS	*lung cancer*	-morbidity	14	0.43[e]	2.9[m]	17	11-25
	(female)	-mortality	14	1.00	13.5[d]	188	132-242
	lung cancer	-morbidity	20	0.43[e]	2.9[m]	24	5-46
	(male)	-mortality	20	1.00	13.5[d]	263	58-478
	IHD -	-morbidity	1822	0.29[e]	1.0[m]	527	266-856
	(females)	-mortality	256	1.0	1.0[n]	254	40-623
	IHD	-morbidity	1801	0.29[e]	1.0[m]	521	256-840
	(males)	-mortality	234	1.0	1.0[n]	233	37-575
	aggravation of asthma		661198	0.22[k]	0.005	784	181-1461
	lower respir. Symptoms		28990[j]	0.21	0.04	233	40- 462
	otitis media acuta		136	0.31	0.06	3	0-6
	sudden infant death		16	1.0	70	1125	1027-1223
lead (drinking-water pipes)	*neuro-cognitive development (1-3 IQ points)*		1764	0.06	70	7950	982-18722
noise	*psycho-social effects*						
	- severe annoyance		1767000	0.01	1.00	17700	5210-32070
	- sleep disturbance		1030000	0.01	1.00	10990	2149-21240
	hospital admissions (IHD)		3830	0.35	0.038[i]	50	7-140
	mortality (IHD)		40	0.7g	0.25[h]	10	0.5-26
food-borne	*acute gastroenteritis*						
	- symptoms		1093000	0.09	0.037	3680	234-11710
	- mortality		48	1.00	11.7[d]	562	530- 590
large industrial accidents	*mortality*		0.5	1.00	41.9[o]	20	6-35
UV-A/UV-B	*melanoma*	-morbidity	24	0.10[e]	6.9[m]	17	3-38
O_3-layer degradation		-mortality	7	1.00	23.0[d]	159	66-266
	Basal		2150	0.053	0.21	24	4-53
	Squamous		340	0.027[e]	1.5[m]	14	2-30
	other mortality		13	1.0	20.2[d]	317	143-511
traffic accidents	incidence[m]		200900[p]				
	- hospital admissions		42000	0.35	0.038	548	79-1514
	- disability > 1 year		6193	0.43[e]	9.50	26520	11830-41950
	- mortality		1322	1.00	35.90[j]	47500	41000-54000
domestic accidents	incidence[m]		1630300[p]				
	- hospital admissions		130000	0.35	0.038	1700	240-4740
	- disability > 1 year		9119	0.17[e]	9.50	19660	3100-39000
	- mortality		2017	1.00	41.9[o]	84710	70030-99390

a. number of people affected annually.
b. severity weight, 0=perfect health, 1=death; prevalence weighted average in case several health states are involved.
c. duration of the health state (years).
d. based on standard life-table analysis.
e. prevalence weighted average of different disease states.
f. attributable prevalence (instead of cases), implying a duration of 1 year.
g. assuming a disability weight of cases between 0 (healthy) and 0.6 (severe cardiopulmonary disease).
h. assuming a 'harvesting' effect among patient with severe cardiopulmonary disease; minimum 1 day, most likely: two months, mean: 3 months, maximum: 5 years.
i. weighted average of duration of exacerbations requiring ER visit or hospital admission.
j. events instead of cases.
k. disability weights from transition of one COPD/asthma health state to the next (mild -> moderate, moderate -> severe).
l. average length of episode estimated to be 2 days.
m. calculated from incidence and prevalence data assuming steady state.
n. assuming a 'harvesting' effect as well as long-term attributive risk; minimum: 1 day, most likely: 0.5 year, mean: 1 year; maximum 11 years.
o. assuming a random age distribution among victims
p. including accidents without significant injury

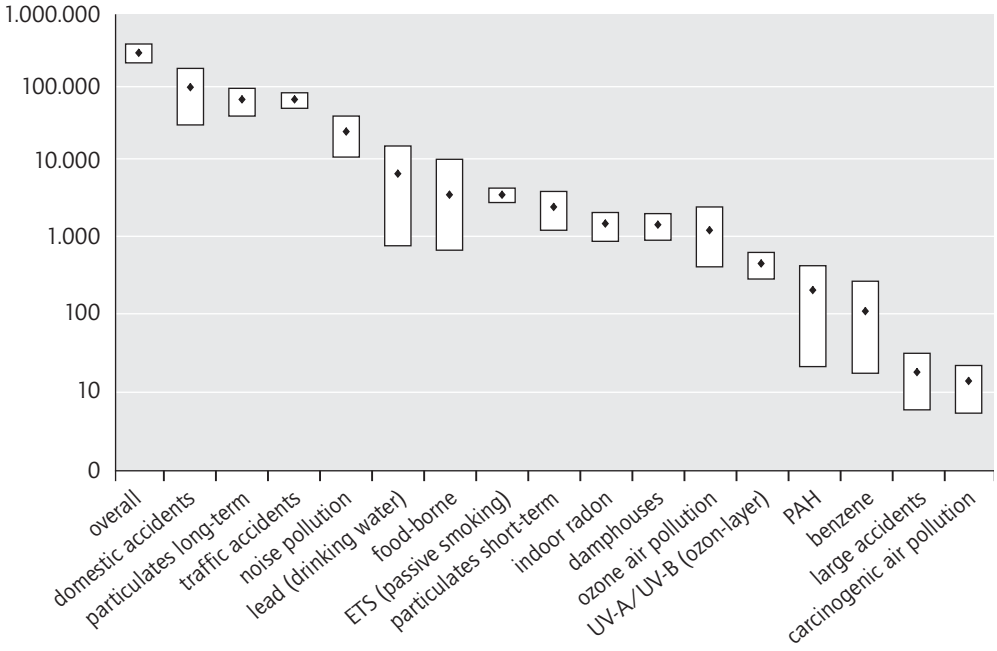

Figure 4-2. Annual health loss in disability-adjusted life-years' for selected environmental exposures in the Netherlands; point estimates and 5 and 95-percentiles of probability interval.

As expected the health loss attributable to environmental exposures is relatively small in the Netherlands. Recently the total annual burden of disease was estimated to be in the order of 2.6 million DALY's[36]. According to the calculations presented here, less than 5 percent of this disease burden would be attributable to environmental exposures, excluding accidents (12% when accidents are included).

The dominance of traffic and domestic accidents is obvious from these calculations. Most striking is the annual health loss associated with the long-term effects of particulate air pollution, which amounts to almost 60% of the total pollution-related disease burden, accidents excluded. Furthermore, the significance of indoor exposures (ETS, radon, and damp related allergens) is intriguing, especially given the little attention it is receiving from policy makers (see Figure 4-2). The large public health relevance of noise (around 25%) is in accordance with recent discussions on environmental quality, for instance in relation to large infrastructure projects, such as airports, highways and railway lines. Furthermore, both food poisoning (and infection) (3,3%) and lead in drinking water (6%) appear to cause substantial health loss.

4.5 Discussion

On deciding to attribute DALY's to environmental exposures, one has to consider several potential flaws. Most of these are not specific to the approach we have proposed here, but concern health impact assessment in general. Recurring, almost inevitable shortcomings involve the imprecision of population exposure assessments, the unknown, and probably 'unknowable' shape of the exposure-response curves at low, environmental exposure levels, and the translation of exposure-response information from one species to another, as well as from one population to another. Another important issue in this domain is the internal and external validity of epidemiological results. In view of the way in which our aggregated results

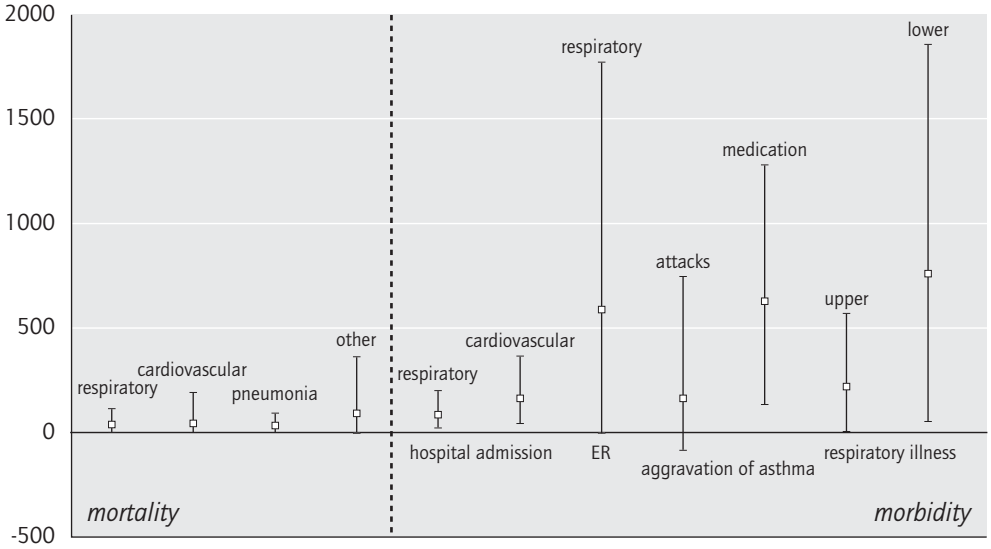

Figure 4-3. Annual health loss in 'disability-adjusted life-years' due to several short-term endpoints associated with exposure to particulates in the Netherlands; point-estimates and 5 and 95-percentiles of probability interval.

are dominated by the long-term public health effects of particulate air pollution, we have to stress the fact that these estimates are based on the results of only two American cohort studies[63,64]. Given the inherent shortcomings of this type of retrospective epidemiological studies, the rather dramatic adverse health impacts shown still need to be confirmed in other well-designed studies. Even though several criteria for causality are met, still substantial uncertainties remain unsolved with respect to specific causative agents and mechanism of actions[6,20,65,66].

Problems associated with health impact assessment have been discussed in numerous publications. Since we have restricted ourselves to generally accepted health impact assessment methodology, in the next part of this section we will only address issues that are typically relevant to the aspect of health impact aggregation.

4.5.1 Are we able to provide a complete picture?

The most fundamental problem we encountered is lack of complete and quantitative insights in how environmental exposures are involved in the onset and development of human disease. We have only incorporated exposure-response associations, which have been studied comprehensively in epidemiological research and for which clearly defined health outcomes have been established. This leaves many potentially adverse factors 'uncovered'.

4.5.2 Inadequate response definitions

Many chemicals have exhibited adverse effects in experimental animals, ranging from slight changes in biochemical activity to pronounced pathological changes and organ dysfunction, sometimes leading to premature death. These toxicological findings represent a powerful tool in health protection[67]. However, for several reasons they are rather inadequate for human health impact assessment. To name a few, high exposure levels used in bioassays to avoid false negative results yield responses which are oft not relevant

for normal environmental exposure conditions in most cases, e.g. rodent bioassays for carcinogens[68]. Furthermore, species differences in biochemical or physiological response to chemical exposures can be rather substantial; differences in susceptibility between species may amount to many orders of magnitude. Probably the most important shortcoming consists of the inability to formulate clear response definitions within a public health context solely based on toxicological information. Many, if not most of standard toxicological response variables are not specific for disease genesis in humans, and therefore cannot be properly translated to real life conditions. In short, it cannot be excluded that exposure to a myriad of chemicals is associated with certain health risks. However, in most cases it is impossible to determine the nature and magnitude of the consequences to public health, based on the available evidence.

4.5.3 Attributing weights to environmental health impacts

Attributing weights to environment related conditions is not a positive, purely scientific exercise, as it involves social and individual values and preferences. This does not always agree very well with the scientific traditions in several disciplines. At the same time, normative evaluation of health-endpoints, no matter how, is virtually inevitable in health risk assessment. In most common health risk measures health preferences remain implicit. One has to bear in mind that even the annual mortality risk is value-laden, as non-fatal health outcomes as well as age at death (loss of life expectancy) are implicitly ignored. The same applies to health-based exposure guidelines, for which value judgements have to be made regarding the health significance of toxicological or epidemiological response variables. Furthermore, it is important to notice that health preference measurements tend to be rather stable and reproducible, even across countries, provided they are performed cautiously[44,47,69].

Uncertainty analysis shows that altering weights within the variance seen in most weighing exercises does not substantially affect the overall picture. Compared with the huge uncertainties that are often connected with health impact estimates, the effect of the possible variance in attributed weights appears rather small. There is an important exception, however. It has to be noted that the lower the disability weights attributed to health states get, the more sensitive they are to variation. It is much easier to double the small weight given to severe noise annoyance than to double that of the terminal state of lung cancer. This is reflected in the results of the uncertainty analysis with respect to 'noise', 'lead' and 'food-poisoning'. The variation of the output probability distribution is largely due to variation in the severity weights given by panel members. Since the less severe responses tend to affect the highest number of people, there is some room for 'manipulation' of results. This implies that severity weights for 'small' and sometimes even controversial responses, such as serious noise annoyance or sleep disturbance must be established with great care.

Another question that needs addressing in this context is where we draw the line with what goes into the measure? Where do we stop regarding responses as relevant to our health state? Do we regard 'severe annoyance' or 'inability to concentrate or communicate' due to residential noise as health conditions or do they merely reflect to the broader concept of well being? In the Dutch tradition of occupational and environmental hygiene annoyance is regarded as a significant health effect[70], but this may differ from traditions in other parts of the world. Including more subjective indicators of well being, such as risk perception or self-perceived health into the aggregate may seem obvious, but could on the other hand reduce its applicability as an instrument to support decision making. There are some indications that people with higher education and income would be inclined to allot lower scores to subjective topics as environmental quality or their own health than people with a lower socio-economic status[47].

4.5.4 Co-morbidity

We were not always able to take proper account of co-morbidity. Responses to air pollution such as lung function deficits, respiratory symptoms, and hospital admissions may concern the same people. Another example of possible co-morbidity would be severe annoyance and sleep disturbance in relation to exposure to residential noise. This known co-morbidity may lead to some overestimation of the environment-related burden, but it would be well within the overall range of uncertainty. At the same time we might have to deal with some underestimation as a result of missing important health outcomes, simply because they were never measured in epidemiological research. Of course there is no way of quantifying this possible underestimation.

Apart from co-morbidity there might be some overlap in exposure indicators and thus in attributed health loss. For instance lung cancer cases attributed to particulate matter and PAH may be the same, as most of the PAH is inhaled as 'coated' particles.

4.5.5 Consistency with policy and scientific priorities

Some of the results of this provisional exercise correspond rather poorly with the spearheads of environmental policy efforts in the Netherlands, as well as in many countries of the world. In spite of the application of rather conservative estimation methods, carcinogenic pollutants in ambient air, for instance, don't seem to contribute much to the disease burden. Still a lot of policy effort is put in monitoring, evaluating and managing outdoor exposure levels of pollutants, such as policyclic aromatic hydrocarbons and benzene. The quality of the indoor environment, on the other hand, scores high in our exercise, but is a rather neglected issue in Dutch and European environmental policy[14].

Nonetheless, we would not dare to suggest an immediate change of environmental policy priorities based on these calculations. Discrepancy may be partly explained by dimensions of health risk perception, which are not captured in our approach, such as 'dread', voluntariness of exposure, the perceived controllability or familiarity of risk generating processes (e.g. traffic), the social distribution of risk and benefit[12,13]. In the Netherlands the actual number of victims of large technological accidents (industry, airplanes) is very small. Nevertheless, several surveys have shown people are worried about them, however small the odds calculated by experts[12]. Of course these perceptions by themselves can seriously affect well being and quality of life.

4.5.6 Uncertainty

The degree of certainty varies a lot from one health impact estimate to another. This may pertain to available data for modelling (criterion validity) as well as to the impact assessment models themselves (construct validity). With respect to criterion validity we performed Monte Carlo analysis, which provided us with probability distributions for the outcome variables. As expected, one should be very careful prioritising environmental health issues solely based on point estimates, given the overlapping uncertainty ranges. On the other hand one can quite clearly discern groups of high-risk exposures (accidents, long-term exposure to particulates) from groups of moderate (lead, food, ETS, radon) and low risk exposures (carcinogenic air pollutants). Continuous efforts along the lines we sketched here should involve dealing with construct or model uncertainty as well, as uncertainty is another important attribute that should be of consequence in decision making[61,71,72,73].

4.5.7 In conclusion

We conclude from our exercise that in spite of methodological and ethical problems, our approach offers a

promising framework for *explicit evaluation* and *comparison* of health loss associated with different environmental exposures, involving a wide variety of non-fatal health outcomes. It enables the incorporation of the public health interest in decision-making with respect to environmental quality and spatial planning. Especially in the planning of extensive infrastructure projects involving a range of diverging, often accumulating exposures the proposed health impact aggregate may be of use. For instance in scenario studies the aggregate can be applied to explore the 'health' score of different options.

As far as risk communication is concerned we are somewhat less sure. After disaggregation of the measure, environmental risk attributes such as loss of life expectancy, quality of life, and social magnitude, measured using time as the single denominator might appeal more to the public than merely the annual risk of dying. On the other hand we have not yet tested the 'face-validity' of this approach among the public. Only when results of comparative health impact assessment are in accordance with the intuitions of the public, one might hope to improve risk communication.

4.6 References

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5 Valuing the health impact of air pollution: Deaths, DALYs or Dollars?[i]

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In this chapter we investigate the feasibility of different indicators to quantify the health impact of air pollution: 1) attributable mortality *risk*, numbers of fatalities, 2) attributable *burden of disease* (disability-adjusted life-years: DALYs), and 3) monetary evaluation of health loss (*'willingness to pay'*). We focus on the health loss associated with the major air pollution phenomena in the Netherlands (indoor as well as outdoor): particulates (long and short term), ozone, ETS, indoor radon, and dampness. Our calculations are based on health impact estimates for the Dutch population, which were produced in the framework of the fifth National Environmental Outlook.

[i] Based on Hollander AEM de, Melse JM. Valuing the health impacts of air pollution: deaths, DALYs or dollars. In: Ayers J, Maynard B (eds) Air pollution and Health. Londen: Imperial College Press (in press, 2004).

5.1 Fifty years on

Fifty years after the infamous London smog of 1952 killed 4,000 to 12,000 inhabitants air pollution might still be a significant health risk factor[1,2]. Since those days air pollution in Western Europe has changed greatly, with regard to both composition and concentrations. In the 19th century, despite some legislation to prevent smoke nuisance from industrial emissions, levels of fossil fuel-related pollutants, such as smoke and sulphur dioxide, were at least tenfold higher than nowadays. Until the 1960s, in large cities such as London, Paris or Amsterdam, the bulk of air pollution came from domestic sources. Since then the widespread use of coal, especially for domestic fires has disappeared almost completely in highly developed countries. High sulphur coal of varying quality was replaced by natural gas of which abundant stocks were discovered in the sixties, e.g. in the North Sea and north-eastern parts of the Netherlands. However, in the same period the volume of road traffic grew exponentially and in spite of impressive, concurrent development of clean engine technology, road traffic is now the most important source of air pollution, accounting for more than one third of fine particulate matter (PM_{10}) emissions (for the smaller fractions, $PM_{2,5}$, more than half), and more than half of nitrogen dioxide emissions[3,4].

It is generally accepted modern day ambient air pollution still affects public health, although the precise causal fraction of the air pollutant mix remains a subject of fierce debate[5,6]. As we spend the greater part of our days inside houses and buildings, indoor air pollution is inevitably of great relevance to public health as well, in particular exposure to radon, second hand tobacco smoke and dampness related allergens all of which contribute significantly to disease burden[7]. In most Western European countries environmental policy is not very much concerned with indoor air quality, at least not in the light of recent discussions on (proposed) legislation with respect to ambient air pollution. Nevertheless in this chapter health impacts of indoor air pollution will be considered as a reference to put the ambient air pollution health risk debate in a wider perspective.

5.2 Problems for policy makers

The rapid advancements in statistical methodology have not really made life easier for policy makers involved in air pollution control. In the 'good old days' risk managers would judge the air quality by compliance with health-based standards. As long as concentrations of air pollutants were below these standards the air was 'safe' to breathe. Whenever concentrations started to exceed standards regularly public health was at stake and risk-reducing measures had to be considered. These standards were - and still are - often primarily based on guidelines derived by expert committees after careful consideration of available toxicological and epidemiological evidence. These 'compound by compound' evaluations are completed with proposals for safe ambient air pollution concentrations using simple, quantitative models. In these models science, societal preferences and policy are elegantly mixed, as establishing air quality guidelines requires several normative choices to be made[8,9]. For instance, one has to define critical toxicological end-points and decide on the extent of the safety margins, given the quantity and quality of the exposure-response data.

Current epidemiological insights do not comply very well with this type of quality standard-based risk management as clear evidence of a threshold for the health end-points considered appears to be lacking, at least at realistic levels of ambient air pollutant concentrations[6]. Furthermore, the observed end-points include a large variety of health effects, ranging from mild, reversible lung function deficits, slightly restricted potential for physical activity to hospital admission and mortality among the susceptible (figure 4-1). Obviously the nature and severity of the health effects depend largely on the individual health status. In this situation drawing the line between trivial and health threatening responses is difficult (see chapter 2).

In the absence of clearly defined safe or virtually safe levels there is no easy way out of discussions on the acceptability of health responses associated with population exposure to air pollution[10,11]. In the last few decades much effort has been put into controlling air pollution associated with key activities, such as transport, energy production, industry, and waste treatment. Current levels are largely determined by best available technology and further emission reductions to meet more stringent standards require ever-bigger resources. Beside constraints on the opportunities for economic activity that might produce more wealth and well being, these resources cannot be used for other things we value, such as health care or the quality of the urban environment. Furthermore, several analyses have cast some doubt on the efficiency of standard-based health risk management, as very expensive measures often appear to yield fairly minor public health gains (and vice versa)[12]. Some authors even suggest that very expensive life-saving regulations might be counterproductive, as a drop in the aggregate national income of around 8 million Euro will theoretically induce one extra fatality[13]. Table 3.3 in chapter 3 shows a remarkable variation in costs per healthy life-year that is saved for a broad range of life saving interventions, of which environmental measures are often found at the more expensive end of the distribution[14].

5.3 Putting money where public health profits most

As pure standard-based decision rules no longer seem to apply fully, a shift is being made towards rules based on 'utility', putting the money where public health profits most[15]. This is not without significance as environmental quality standards traditionally stand for *equity*, the right to protection from adverse effects for everybody, regardless of age, health, and susceptibility (right-based decision rules). On the other hand, from an utilitarian perspective maximisation of utility (efficiency) may require a more skewed distribution of health risk over societal groups, simply because it would be more cost-effective. Or, in other words: guaranteeing the last citizen in town the general level of health protection can be extremely expensive[16]. Here, according to neo-classical economic theory, the trade-off between efficiency and equity becomes an issue.[17]

From a utilitarian perspective, when considering air pollution, certain questions have to be addressed:

- How bad is this environmental exposure, e.g. compared to other environmental exposures or other health risks in general?
- How does public health benefit from policy measures to reduce public exposure?
- What policy measures are most efficient or what is the optimal deployment of available resources in terms of health gains?

To describe and compare the health impact of various environmental exposures and, eventually, to perform cost effectiveness analysis of options for environmental policies obviously some sort of 'denominator' is required. Generally speaking three ways of characterising potential health benefits are being used: health risk reduction (numbers), (health adjusted) life-years (e.g. QALYs or DALYs) or money ('monetarised' health endpoints) .

5.3.1 Numbers

To characterise the health impact of air pollution or any other risk factor, one can simply calculate the number of cases of health damage associated with a certain exposure distribution, such as deaths, hospital admissions or number of asthma attacks. In traditional quantitative risk analysis, health risks are measured and, often implicitly, compared in terms of annual mortality risk: numbers per year. In several Western countries environmental regulation with respect to industrial safety (within or around the facility), radiation protection or chemical pollutants is based on a small 'accepted' annual mortality risk for each

exposed individual. Often a individual risk criterion in the order of 10^{-5} or 10^{-6} is being used as a threshold of acceptability[19,20,21]. Such an approach at least guarantees everybody is treated in the same way, young and old, rich and poor, executive and unemployed; it prevents inequity as a result of 'unloading' health risk on smaller groups of individuals, often the low-cost solution[16].

5.3.2 Health adjusted life-years (e.g. DALYs)

However, it has become clear that one 'annual ten to the minus six' risk may differ substantially from another in several important aspects[22], such as loss of *life expectancy* and *non-lethal health outcomes*. For instance mortality during particulate air pollution episodes may at least in partly involve 'precipitation' of death among the old and weak, thus costing several months of unhealthy life at the most[23,24,25], while the impact associated with fatal accidents involving individuals with a 'random' age distribution may amount to a loss of many healthy years[26]. Interestingly, recent analysis of fatal traffic accidents in the Netherlands show that, apart from a well-known peak in traffic deaths around age 20, the age distribution is very similar to that of total mortality (primarily because old people are more frail and have lower survival probabilities than younger people)[27]. In addition, public health focus has gradually changed from life expectancy to health expectancy, i.e. postponing as long as possible or mitigating the functional limitations that come with chronic disease of older age and that affect the ability to cope with the demands of daily life[28,29]. More or less the same goes for the health impact of air pollution. In many cases these do not involve mortality, but rather aspects of the quality of life, such as

- aggravation of pre-existing disease symptoms, e.g. asthma, chronic bronchitis, cardiovascular or psychological disorders
- severe annoyance, sleep disturbance, as well as a reduced ability to concentrate, communicate or perform normal daily tasks
- feelings of insecurity or alienation, unfavourable health perception and stress in relation to poor quality of the local environment and perceived danger of large fatal accidents[30].

This implies that mortality risk might not always be the most appropriate indicator of environmental risk.

In an utilitarian approach to maximising cost-effectiveness for society, one would want to employ some sort of public health currency unit representing the full attributable health loss. Over the last few years much effort has been put into the development of metrics in which any type of morbidity or mortality is transformed into an equivalent number of life years (quantity plus quality of life). This type of health aggregating metric allows formal analysis of cost-effectiveness of environmental policy measures, which by now has more or less become common practice in medical technology assessment and public health research[31,32,18].

5.3.3 Monetary value

Alongside the cost-effectiveness analysis, cost-benefit analysis is a form of evaluation in which the (health) benefits are also expressed in monetary terms. Efficiency calculations are made easier by putting cost and benefits under one heading, namely money. Furthermore, it is easier to include non-health aspects on the benefit side (equity, productivity, well-being) . In principle, investments in the health domain can be compared with investments outside, for instance transport safety, education, or ecological quality. Obviously this form of analysis requires the difficult task of expressing loss of life, life-years, or burden of disease in money. In such a 'hardcore' economic approach one seeks to attach a price tag to the incidence of different health end-points, e.g. by investigating willingness of people to pay (WTP) to prevent defined health endpoints, or the amount of money for which people are willing to accept (WTA) a certain level of

health risk. In some studies primarily the costs of productivity loss and health care use are estimated[33,34,35,36,37]. Of course, the latter approach would not include the price of individual suffering.

In this chapter we investigate the feasibility of paradigms of risk management (mortality risk, numbers), attributable burden of disease (disability-adjusted life-years: DALYs) and monetary economic evaluation to support public health policy with respect to air pollution. We will focus on the health loss associated with the major air pollution phenomena in the Netherlands (indoor as well as outdoor). Our calculations are based on health impact estimates for the Dutch population, which were produced in the framework of the fifth National Environmental Outlook[38].

5.4 Healthy time as a metric

Accepting the fact that annual mortality or even loss of life expectancy do not fully represent the environmental health loss, we have applied an approach largely based on the 'burden of disease' measure developed by Murray and Lopez. To assess the global disease burden, and consequently the health policy priorities in different regions in the world, they used disability adjusted life years (DALYs). This health impact measure combines years of life lost and years lived with disability that are standardised by means of severity weights[28,39]. Our adaptation of the DALY-concept was inspired by the notion that the multiform health loss due to environmental exposure is fairly well characterised by three dominant aspects of public health, viz.

quantity of life (life expectancy)

quality of life, and

social magnitude (or number of people affected).

The diagram in figure 2-4 (chapter 2) sketches the basic idea behind our approach. At birth each of us may expect around a potential eighty years of healthy life. However, due to our genetic programming, our often-unfavourable life-styles, poverty, occupational or environmental conditions or just bad luck, most of us will encounter disease that will reduce the quality of part of our life-years. These diseases may manifest themselves in episodes, as chronic disease or even progressive disability until death. Some of us will die abruptly, for instance caused by an accident or an infectious disease. Thus, public health loss is defined as time spent with reduced quality of life, aggregated over the population involved. Based on this concept health loss attributable to air pollution can be assessed by:

defining responses that are associated with air pollution exposure

calculating the number of people affected (N),

estimating the average duration of the response (including loss of life expectancy as a consequence of premature mortality, D), and

attributing disability weights to each unfavourable health condition (Box 1)

Finally we estimate the number of disability adjusted life years that is lost per year of exposure, using the following equation:

$$DALY_{e.e.} = \sum_{i=1}^{n} \sum_{k} I_k * f_k(RR_i, C_i) * S_k * D_k$$

in which

$DALY_{e.e.}$ = health loss related to n environmental exposures, measured as disability or quality adjusted life-years per year of exposure,

$f_k(RR_i, C_i)$ = a set of functions (including exposure C_i and associated relative risk measures RR_i) representing the population attributable fraction (PAF) of condition k,

I_k = annual incidence of response variable (baseline risk) k, S_k = severity factor discounting time spent with the condition (see previous paragraph), D_k = duration of the condition; in case of premature mortality: loss of life expectancy.

To estimate the number of people affected we calculated PAFs by combining population exposure distributions with quantitative exposure-response information, applying the following equation (or one its derivatives):

$$PAF = \frac{\sum_{i>0} (RR_i - 1) * p_i}{\sum_{i \geq 0} RR_i * p_i}$$

in which RR_i = relative risk in exposure class i and p_i = exposure probability in class i.

Subsequently we estimated the number of people affected by a certain response by multiplying the PAF with annual incidence figures, obtained from Dutch health statistics, primary care registrations, or specific surveys.

5.5 Trading health for wealth or wealth for health

While DALYs measure health loss in units of time, applying the WTP (or WTA) approach is an effort to measure health loss in terms of money. Embedded in welfare economics it should be seen as the rate of substitution between health and wealth. Health is regarded as an economic good. An individual's preference for one health condition over a certain period of time can be represented by a change of income or wealth, or, in other words decreased possibilities to purchase other valued goods[17]. There are two broad ways to produce these estimates:

revealed preferences: investigating how health risks that are related to certain risky occupations are allowed for in the differences in salary, or what extra amount people are prepared to pay for safer or healthier products (for example, cars with airbags, or houses in quieter surroundings);

stated preferences: using questionnaires to find out what people are prepared to pay for one extra life-year or one year free of disease or disabilities (contingency valuation).

Significant methodological objections are attached to both methods in connection with the transferability of implicit (behaviour) or explicit (survey) preferences of people from one circumstance to another, including a series of shortcomings that are characteristic of all questionnaire-based surveys[40,41,42]. In terms of the value of a statistical life, the outcomes of the above-mentioned methods are nevertheless reasonably consistent. On average Americans, Canadians or Europeans are willing to pay in the order of two million euros for a statistical life (1.5 to 7 million Euro), which is approximately 70,000 to 80,000 Euro a year at a discount rate of 3% (which for most of us is considerably more than our earning capacity, another commonly used proxy)[43,44].

5.6 Impact assessment

5.6.1 Health responses to exposures

Table 5-1, table 5-2, and table 5-3 present an overview of the population exposure-response functions for each air pollution type observed in a range of environmental and occupational epidemiological analyses. The associations for fine particulate matter (table 5-1) and ozone (table 5-2) are presented as increases in end-point incidence per 10 and 150 $\mu g/m^3$ increase of the pollutant concentration, respectively, either based on results of published Dutch studies or simple random effects meta-analysis[45,46].

In all tables an estimate is given of either incidence (including mortality) or prevalence of the health end-points in the Netherlands. These were based on available Dutch health statistics, summarised in the framework of the National Public Health Status and Forecast Report[29,47].

Table 5-1. Exposure response functions for health effects associated with particulate air pollution.

Health response *(particulate matter, PM_{10})*	**study design**	**Effect estimate (% per 10 μg/m³)**	**Inc/Prev (p_0)**
Long term mortality	cohort[48,49,109]		
overall annual mortality		2.4 (0.1-6.1)[a]	0.0078
cardiopulmonary		4.3 (0.5-11.0)[a]	0.0032
lung cancer		2.0 (-4.5-8.1)	0.00054
chronic respiratory disease			
chron respir symp. children	cohort[50,51]	8.7 (0.7-16.6)	0.057
chronic bronchitis adults	cohort[52,53,54,55,56]	2.5 (1.2-3.8)	0.018
daily mortality	time series[57]		
total		0.36 (0.25-0.47)	0.000024
respiratory disease (COPD)		1.11 (0.64-1.61)	0.00000011
cardiovascular		0.25 (-0.09-0.42)	0.0000071
pneumonia		1.21 (0.65-1.80)	0.00000065
hospital admission	time series[58]		
respiratory		0.8 (0.5-1.1)	0.0000195
cardiovascular		0.7 (0.5-0.9)	0.0000384
emergency room visits	time series[77]		
respiratory		1.5 (0.5-2.6)	0.022
aggravation of asthma	panel studies[59,60,61,62]		
attacks		4.4 (-2.0-10.5)	0.00023
use of bronchodilators		7,0 (0.64-12.9)	0.00059
respiratory symptoms	panel studies[77]		
upper respiratory tract		2.0 (-0.13-4.1)	0.19
lower respiratory tract		3.8 (0.3-7.1)	0.038

a. calculated for the fraction highly exposed living within 50, respectively 100 meters distance from a major inner-city road or a freeway, roughly comparable with a concentration difference of 10 mg/m³ for black smoke, 30 mg/m³.for NO_2 and 40 mg/m³ for PM_{10}[3 109]

Table 5-2. Exposure response functions for health effects associated with ozone air pollution.

Health response *photochemical (ozone)*	**Study design**	**Effect estimate (% per 150 μg/m³, 8h average)**	**Inc/Prev (p_0)**
daily mortality	time series[57]		
total		4.1 (2.0-5.9)	0.000024
respiratory disease		12.7 (4.8-21.1)	0.00000011
cardiovascular		3.2 (0.3- 6.1)	0.0000071
pneumonia		18.8 (9.0-29.9)	0.00000065
hospital admission	time series[58]		
respiratory		6.5 (3.9- 9.1)	0.0000195
emergency room visits	time series[45]		
respiratory		9.1 (2.9-13.8)	0.035

Population-weighted exposure distributions for ozone and particles were derived through linear interpolation of data from stationary air pollution monitors of the national network. As urban concentrations of particles tend to be higher adjustments were made based on the virtual diameter of the urban agglomerations (> 40.000 inhabitants), employing an empirically determined factor. Subsequently, the spatial distribution of air pollutant concentrations was combined with data on population density by means of Geographic Information Systems (table 5-4)[63,64].

Table 5-3. Exposure response functions for health effects associated with selected air pollutants.

Air pollutant	Health end-point	Effect estimate	P_o
damp houses[65]	asthma symptoms children	RR_{expo}: 1.41 (1.23-1.71)	0.135
	asthma prevalence adults	RR_{expo}: 1.56 (1.25-1.95)	0.012
ETS[66]	Lung cancer ns[a] men	RR_{expo}: 1.34 (1,00-1.84)	0.000016
	Lung cancer ns women	RR_{expo}: 1.24 (1.13-1.36)	0.00000074
	IHD[b] non-smoking men	RR_{expo}: 1.23 (1.03-1.47)	0.0006
	IHD non-smoking women	RR_{expo}: 1.19 (0.97-1.45)	0.0005
	aggravation of asthma	RR_{expo}: 1.63 (1.30-1.96)	0.004
	lower respir. symptoms	RR_{expo}: 1.46 (1.33-1.60)	0.037
	otitis media	RR_{expo}: 1.29 (1.05-1.35)	0.00012
	sudden infant death	RR_{expo}: 1.94 (1.55-2.43)	0.0000037
Radon[67,119]	lung cancer	Add. Risk 0.0004 (0.0002)/WML[c]	

[a] non-smoking
[b] ischaemic heart diseaese
[c] working month level (exposure measure)

We recognise the fact that this type of exposure characterisation only poorly reflects personal exposure, which is a function of time-activity patterns and micro-environmental concentrations. On the other hand epidemiological studies incorporate the same kind of relatively poorly defined ambient air pollution data. Furthermore, at least with respect to particles, a fairly consistent correlation has been shown between ambient and personal exposure indicators[68].

The prevalence of exposure to spousal environmental tobacco smoke was based on cross-sectional monitoring data[69], assuming smokers had a risk of living with a smoker three times as high as a non-smoker. Prevalence of damp homes was based on an investigation by the Dutch ministry of housing[30]. Based on an extensive monitoring program in more than 1,500 Dutch dwelling, (future) exposure to indoor radon of the Dutch population was modelled based on characteristics of the total stock of dwellings, such as air-tightness, building materials and ventilation behaviour[30,70].

5.6.2 Long-term versus short term mortality

For the sake of clarity (and simplicity) we make a distinction between exogenous risk factors that are involved in the onset and progression of (chronic) disease and risk factors that primarily 'accelerate' death among the weak. The first type of interaction can be shown in cohort studies in which populations are followed up during a sufficiently long period to see whether certain exposures affect the incidence or mortality of disease, allowing for gender, age, smoking, occupational status and diet. Examples of these are studies among workers exposed to hazardous substances in an occupational setting (radon, benzene, polycyclic aromatic hydrocarbons) and a number of American cohort studies retrospectively relating survival among citizens to air pollution levels[71,72,73,74]. Results of both the American Cancer Society Cohort

(ACS) and the Harvard Six Cities studies stood up to extensive scrutiny by the Health Effects Institute[75]. An extension of the follow-up of the ACS study yielded results that were consistent with earlier reports[76]. Therefore it is reasonable to assume that long term exposure to particulate air pollution indeed affects survival, especially by increasing the risk of cardiopulmonary disease and lung cancer. 'Harvesting', the bringing forward of death among the susceptible, for instance through aggravation of disease during unfavourable air pollution conditions, would go unnoticed in this type of study, given the extended 'time-window' of many years. This effect is especially seen in time series analysis, observing day-to-day variations (see figure 4-1)[24,77]. Of course just the accumulation of these day-to day health insults underlying changes in mortality and hospital admission may very well be a causal factor of chronic morbidity and survival loss[78].

Table 5-4. Exposure distributions for air pollution types.

Air pollutant	Metric	Mean (stDev)
PM10[63]	ann.av. 24-h (μg/m^3) ann.av. 8-hour (μg/m^3)	34.4 (3.6)
Ozone (8-hours μg/m^3)[63]		45.6 (6.8)
Environmental Tobacco Smoke	Prevalence	
non-smoking men 1970	smoking partner	0.26
non-smoking women 1970	smoking partner	0.60
non-smoking men 1990	smoking partner	0.22
non-smoking women 1990	smoking partner	0.30
children 1995	smoking parent	0.40
Damp homes[79]	prevalence	0.175 (0.07)
Radon[80]	average indoor concentration (Bq m^{-3})	23-24 Bq m^{-3}

To estimate loss of life-years we applied the concept of attributable risk, assuming that cause specific mortality due to environmental exposure is similar to any 'other' cause specific mortality, with respect to onset and time of dying. For each end-point we estimated the years of life lost by means of life-table analysis (the sum of number of cases per age-group times remaining life expectancy divided by total number of cases), similar to the way in which several authors have calculated the impact on *national* average life-expectancy[24,81,82]. We also investigated the sensitivity of the result to changing relative risks with age or implementing a certain length follow-up (table 5-5).

Several sophisticated epidemiological analyses have been undertaken to estimate the loss of life-years due to the short term mortality that is revealed by the analysis of time series of daily mortality and air pollution data. Most analyses suggest the average loss of lifetime would range from a few days to several months. In some cases the loss may extend to one year or more, for instance due to pneumonia or a heart attack[83,84,85,86]. Furthermore, several analyses have shown most of the air pollution associated deaths occur outside the hospital, implying that these effects are not limited to the terminally ill[87]. Here we will *arbitrarily* apply a rather non-informative subjective distribution ranging from one week to one years, with an average of three months (table 5-5).

Table 5-5. Estimated duration of effects.

Health response (particulate matter, PM_{10})	distribution applied in Monte Carlo analysis	parameters (years)
Long term mortality	**(Dutch life table)**	
overall annual mortality	no	10.9
cardiopulmonary	no	8.2
lung cancer	no	13.0
ischemic heart disease	**uniform**[a]	
	minimum	1
	maximum	11
sudden infant death	no	70
daily mortality	**subjective Beta**[b]	
	minimum	0.02
	most likely	0.25
	mean	0.5
	maximum	10
hospital admission	**subjective Beta**[c]	
respiratory	minimum	0.011
cardio-vascular	most likely	0.019
	mean	0.038
	maximum	0.167
emergency room visits	**normal**	
respiratory	mean	0.033
	stand. dev.	0.021
aggravation of asthma	**uniform**	
attacks	min	0.0027
use of bronchodilators	max	0.0055
respiratory symptoms	**point estimate**	
upper respiratory tract		0.019
lower respiratory tract		0.038
lung cancer morbidity	**point estimate**[d]	2.9

[a] A uniform distribution: minimum loss of life expectancy 1 year, maximum 11.3, being the loss based on life-table calculation (passive smoking as dominant cause of morbidity and death).

[b] A subjective BETA distribution allowing to incorporate the following quantitative assumptions: minimum loss of life expectancy 1 week, most likely 6 months, maximum 10 years, being the loss based on life-table calculations (e.g. air pollution impedes recovery from pneumonia).

[c] A subjective PERT distribution allowing to incorporate the following expert assumptions: minimum duration of disease aggravation episode 4 days, most likely two weeks, maximum two months.

[d] Based on Dutch data on incidence and prevalence.

As in the case of long-term mortality here we apply the concept of population attributable risk. The fraction attributable to air pollution exposure is assumed to be similar to the total morbidity load. To assess the time spend with a certain morbidity we use year prevalence data (asthma, ischaemic heart disease). In a stable situation by definition prevalence equals the number of new cases times the average duration of the condition[88].

Estimates for the duration of health care events were either derived from the literature, the Dutch health care registration (NIVEL) or from expert consultation[89,90,91,92,93]. An overview of duration estimates is presented in table 5-5.

Box 1. Severity weights for disease states

In the framework of the National Public health Status and Forecast Reports an estimate was produced of the burden of disease within the Dutch population[29]. To define 'Dutch' severity weights Stouthard et al selected 55 diagnoses of greatest public health significance in terms of number of patients, and years of (healthy) life lost. These diagnoses were divided in 176 health states of various severity (or disease stage)[94].
According to the protocol designed by Murray et al[95], physicians with ample clinical experience were invited to perform the weighting procedures, which consisted of two steps. At first they evaluated a selection of 16 representative indicator states, using two varieties of a person-trade-off approach. This first step of the valuation process was performed during workshops, as deliberation is an explicit part of the protocol. A visual analogue scale (VAS) was added as another instrument of valuation, mainly for the purpose of validation. Furthermore, a standardised classification of the indicator states according to EuroQol-5D+ was provided to assist panel members[96]. This classification instrument involves a three-point scale for six health dimensions, viz. mobility, self-care, daily activities, pain/discomfort, anxiety/depression and cognitive functions. Using the indicator states for 'calibration' the remaining health states were valued individually by means of interpolation (ranking health states similar to one or in between two consecutive indicator conditions, figure 5-4).
For air pollution exposure-related chronic disease morbidity for which different health states have been defined, such as asthma or ischaemic heart disease, a severity factor was composed as a prevalence-weighted average, assuming that the environmental exposure had no effect on disease prognoses[88] (e.g. mild, moderate and severe asthma). In some cases weights referred to transition from one severity state to another, for instance from mild to severe asthma ('aggravation of asthma').

5.6.3 The health market

To assess the loss of economic utility associated with the health end-points involved, one would require an economic valuation of the full quality of life impact to the affected individual. This would include expenses such as medical costs and lost income (often referred to as cost of illness, COI), and less tangible effects on well being such as pain, discomfort and restriction of everyday activities. One way of assessing the economic utility loss is to ascertain the individual maximum WTP for the reduced incidence of illness and adverse symptoms. To compile a set of WTP-values for the health impacts quantified here we drew upon a number of studies that reviewed the literature on WTP for avoiding changes in risk of death, chronic disease as well as milder morbidity effects[33,34,35,37,97,98]. These values were primarily derived from studies in which preferences of both healthy and infirm individuals were revealed through questionnaires, the so-called contingent valuation studies. In some cases, in the absence of reliable data, COI-estimates were used as a proxy, adjusted upwards by a factor of 2. Table 5-6 lists the WTP values derived from these reviews.

Table 5-6. Monetary values for health endpoints based on 'willingness to pay' (WTP).

Health response	distribution	Euros
Long term mortality overall annual mortality cardiopulmonary lung cancer ischemic heart disease sudden infant death	**discrete** lower (0.33) middle (0.5) upper (0.17)	 1,950,000 4,360,000 6,760,000
chronic bronchitis	**discrete** lower (0.33) middle (0.33) upper (0.33)	 150,000 [a] 220,000 390,000
asthma	**discrete** lower (0.33) middle (0.33) upper (0.33)	 65,000 [b] 105,000 130,000

Health response	distribution	Euros
chronic respiratory symptoms (1 year)	**discrete**	
	lower (0.33)	165 [c]
	middle (0.33)	330
	upper (0.33)	495
daily mortality	**subjective PERT**	
	minimum	1,200 [d]
	most likely	38,000
	mean	109,000
	maximum	4,360,000
hospital admission	**discrete**	
respiratory	lower (0.33)	1,100 [e]
	middle (0.33)	7,000
	upper (0.33)	14,000
cardiovascular	lower (0.33)	1,100 [e]
	middle (0.33)	7,000
	upper (0.33)	15,000
emergency room visits	**discrete**	
respiratory	lower (0.33)	260 [f]
	middle (0.33)	520
	upper (0.33)	780
aggravation of asthma	**discrete**	
attacks	lower (0.33)	35 [g]
use of bronchodilators	middle (0.33)	70
	upper (0.33)	140
respiratory symptoms	**discrete**	
upper respiratory tract	lower (0.33)	6 [h]
	middle (0.33)	12
	upper (0.33)	18
lower respiratory tract	**discrete**	
	lower (0.33)	6 [i]
	middle (0.33)	38
	upper (0.33)	330

a based on contingent valuation study in US[34,99]
b based on contingent valuation study in US[34,37]
c based on adjusted cost of illness (COI) for cases of acute bronchitis in US[34]
d see text[25]
e lower estimate based on empirical relationship between WTP and quality of life in accordance with ref. 25; central estimate based on contingent valuation study in US[37]; upper estimate adjusted COI in the US[34]
f based on WTP-study in US[34,37]
g lower estimate WTP from adjusted COI[34]; central estimate WTP non-asthmatic respondents, upper estimate WTP asthmatic respondents in Norwegian CVM-study
h based on CVM-estimates in US[34] and a Norwegian study[100]
i lower estimate based on CVM-study in US[34]; central estimate based on Norwegian study[100]; upper estimate WTP for acute bronchitis in US[34].

From a review of these studies we suggest a range for the value of a (statistical) death of 2-7 million Euro, with a central estimate of around 4,500,000 Euro, independent of age or disability. The contingency valuation method produces the highest estimates, while the consumer market studies yield the lowest values[37].

The economic valuation of daily mortality presents a problem as most reviews suggest that recorded deaths may often involve old people suffering from severe chronic disease. As compared to fatalities among young or middle aged, healthy individuals, a number of adjustments appear to be justified. These

would concern the loss of life-years, the average age distribution, and the quality of life of the cases involved. Much in accordance with the report of the Ad-Hoc Group on the Economic Appraisal of the Health Effects of Air Pollution in the UK we propose a lower limit of 1,200 Euro for day-tot-day mortality. Here a maximum reduction factor is applied adjusting for loss of life-years, age and disability (0.083: assuming a minimum individual loss of life expectancy of one week instead of 12 years; 0.75 to adjust for a lower valuation of a prevented statistical fatality at older ages[37,101], and 0,2 as the lower bound of the disability weight for the very ill)[25]. The upper bound estimate of the value of a prevented statistical fatality would equal the unadjusted (central) estimate, given the unlikely possibility daily mortality would involve a healthy subject with an average life expectancy. For the 'most likely' monetary estimates the loss of life-time per case is assumed to be 6 months respectively; a factor of 0.6 is applied to adjust for age and disability[44,83].

As immediate benefits in general are more valuable to people than benefits some time in the future, many valuation systems used in cost benefit analysis apply discount rates, not just for costs but for health benefits as well (e.g. 3% annually). As we did not make a formal cost-effectiveness analysis, here we have not applied discount rates. In our exercise discount rates would apply to chronic effects, such as premature mortality or an extra case of chronic bronchitis. A discount rate of 3% over 10 years would reduce the valuation in DALYs or Euros by around 13%. This would fall well within the range of uncertainty we deal with in this analysis.

5.6.4 Uncertainty

We analysed the uncertainty in the calculations of air pollution related DALYs and Euros by means of Monte Carlo techniques[102,103,104]. In a Monte Carlo simulation model input parameters are treated as random variables. For each of the input parameters, such as population exposure, exposure-response function estimates, average duration of the response and discount factors, a probability distributions function was estimated, representing parameter uncertainty. Subsequently, an output distribution for the different health impact measures was estimated by iterative (Latin hypercube) sampling from each of the defined parameter distributions, followed by recalculation. Here we will present the 5- and 95-percentiles of this distribution as a measure of uncertainty.

5.7 Deaths, DALYs and dollars (Euros)

Point estimates as well as the 5 and 95-percentiles of the probability distribution for impact metrics are shown as a measure of uncertainty (table 5-7, figure 5-1). In most cases this uncertainty range is substantial, but less than one order of magnitude[102,103,104]. Strikingly, the rank order of the exposures with respect to attributable burden differs from one impact indicator to another, with the exception of long term exposure to particulate matter which is always on top of the list, outpacing the other factors.

In our calculations the health loss related to air pollution is substantially dominated by the long-term effects of particulate air pollution, which amounts to 40% of deaths, 65% of total DALY-loss and 75% of the total monetary loss related to air pollution (Table 5-7). The estimated 'monetarised' loss is huge, almost 16 billion Euro (5-95%-tiles: 7-25 billion), which is between one quarter and one third of the total annual budget for Health Care in the Netherlands, but it must be stressed that this is only an indicative figure as the WTP-amount of money is not 'real money'.

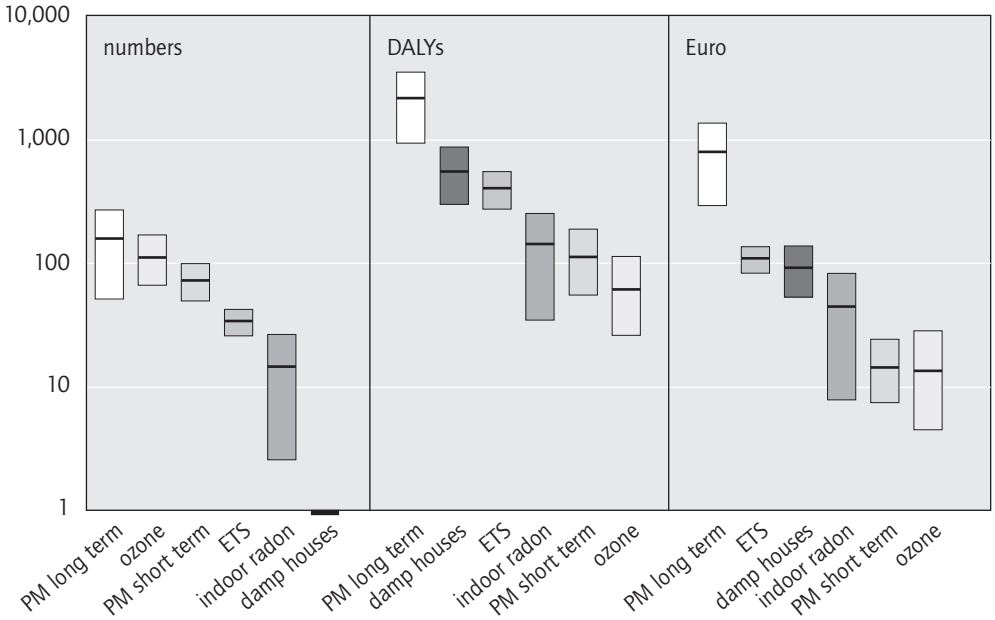

Figure 5-1. Annual health loss for a number of air pollution phenomenon expressed in terms of fatality number, DALYs and million Euro per million inhabitants (average and 5-95%-tiles of uncertainty distribution).

Table 5-7. Summary of health impact estimates for a number of air pollution phenomenon, expressed in fatalities, DALYs and Euros.

Environmental factor	Health outcome	#/million[a]	5-/95% -tile	DALYs/ million	5-/95% -tile	Euros (x106)/ million	5-/95% -tile
particulate	*mortality*						
air pollution	- cardiopulmonary	135	60-220	780	256-1480	585	230-1060
long-term	- lung cancer	30	-60-120	230	-450-975	115	-225-450
	morbidity						
	- chronic resp. sympt.	3,300	825-6,200	135	28-275	1.1	0.3-2.2
	- chronic bronchitis	400	90-710	1050	225-2030	100	20-190
total long term		*160[b]*	*52-275*	*2,200*	*950-3,560*	*800*	*295-1370*
particulate	*mortality*						
air pollution	- total	73	50-100	25	5-64	8	1-21
short-term	- respiratory	10	6-14	3.5	0.6-9.0	1.1	0.1-3.0
	- coronary heart dis	17	7-27	6	1-15	2.0	0.2-5.1
	- pneumonia	7	4-10	2.5	0.5-6.0	0.7	0.1-2.0
	Hospital admission						
	- respiratory	85	440-144	1.9	0.7-3.7	0.6	0.2-1.3
	- cardiovascular	86	42-150	2.1	0.8-4.1	0.7	0.2-1.4
	emergency room visits						
	- respiratory	1530	550-2900	24	4-61	0.8	0.3-1.6
	aggravation of asthma						
	- asthmatic attacks	4,7000	6,500-105,000	26	2-73	4	0.5-9.5
	- use of bronchodil.	51,000	6,800-116,000	28	2-82	5	0.5-10
	aggravation of resp. symptoms						
	- upper resp. tract	9,860	1250-19,100	8.2	0-24	0.1	0-0.2
	- lower resp. tract	3,930	870-7200	25	2-72	0.5	0.1-1.3
total short term		*73[b]*	*50-100*	*113*	*55-190*	*15*	*7.5-25*

Environmental factor	Health outcome	#/million[a]	5-/95% -tile	DALYs/ million	5-/95% -tile	Euros (x106)/ million	5-/95% -tile
ozone	*mortality*						
	- *total*	112	76-170	39	6-102	12	1.5-35
	- respiratory	16	8-28	6	1-15	1.8	0.2-5.0
	- cardiovascular	34	7-90	12	1-40	3.7	0.2-13.0
	- pneumonia	9	5-13	3.1	0.5-8.0	1.0	0.1-2.6
	hospital admission						
	- respiratory disease	72	46-98	2.0	1.0-3.5	0.5	0.2-1.1
	emergency room visits						
	- respiratory disease	1,290	520-2,300	21	3.4-62	0.8	0.3-1.6
total ozone		*112[b]*	*67-172*	*61*	*26-114*	*13.5*	*4.6-29*
damp houses	*Asthma*						
	- children	1665,	898-2622	133	58-235	41	21-68
	- adults	1814	680 –3430	145	47-302	45	16-87
	lover resp. disease						
	- children	661	300-1165	27	5.6-61	0.5	0.2-1.1
	- adults	6092	3140-9730	250	53-520	5	1.9-9.2
total dampness		*0*	*0*	*554*	*300-880*	*92*	*53-142*
ETS	*lung cancer* -morbid.	1,9	1,3-2,4	0.8	0.5-1.2		
	(female) -mortality	0.9	0.6-1.2	12.2	8.5-15.7	2.8	1.9-3.7
	lung cancer -morbid.	2.7	0.7-4.7	1.2	5- 45		
	(male) -mortality	1.3	0.3-2.3	17	4.5-30.5	3.9	1.0-7.1
	IHD						
	- morbidity females	118	75-162	34	17-55		
	- mortality females	16.5	10.4-23	90	22-175	50	32-71
	- morbidity males	116	73-162	34	17-54		
	- mortality males	15	9.5-21	83	21-161	47	29-66
	aggravation of asthma	91780	42720-1538200	51	9-118	3.4	1.3-6.3
	lower respir. sympt.	1900	1600-2150	12	1.5-28	0.2	0.0-0.5
	otitis media acuta	2700	1250-4100	30	1.1-76	0.3	0.1-0.8
	sudden infant death	0,6	0,1-1,1	43	5-76	1.9	0.2-3.4
total ETS		*35[b]*	*26-43*	*410*	*280-553*	*110*	*84-139*
radon	*lung cancer morbid*	*15*	*3-27*	*13*	*2.2-25*	*46*	*8-85*
	lung cancer mort	*15*	*3-27*	*132*	*23-241*		
total radon		*15*	*3-27*	*145*	*35-256*	*46*	*8-85*
overall		*395[b]*	*273-525*	*3,485*	*2,175-4,905*	*1,075*	*560-1,650*

[a] number of people affected
[b] number of deaths only

Over the past decade annual investments in traffic emission reduction in the Netherlands added up to somewhere between 250 and 350 million Euro, yielding an emission reduction of around 4% per year[105,106]. At the same time traffic volume has grown with over 3 % per year. If we assume there is a linear relation between emission and actual population exposure, the benefits of these investments in terms of monetarised health gain are in the same order of magnitude, even somewhat higher (7-8% of an annual disease burden of 13 billion Euro: 550 million Euro). Based on the same assumption the cost of air pollution control measures per DALY saved is around 50,000 Euro. In recent discussions among health economists an investment of 40,000 to 50,000 Euro for each equivalent year of perfect health gained by an intervention is regarded as 'acceptable value for money'[107]. The World Bank recently proposed 3 times the Gross Domestic Product per capita; for the Netherlands that would be around 75,000 Euro/DALY

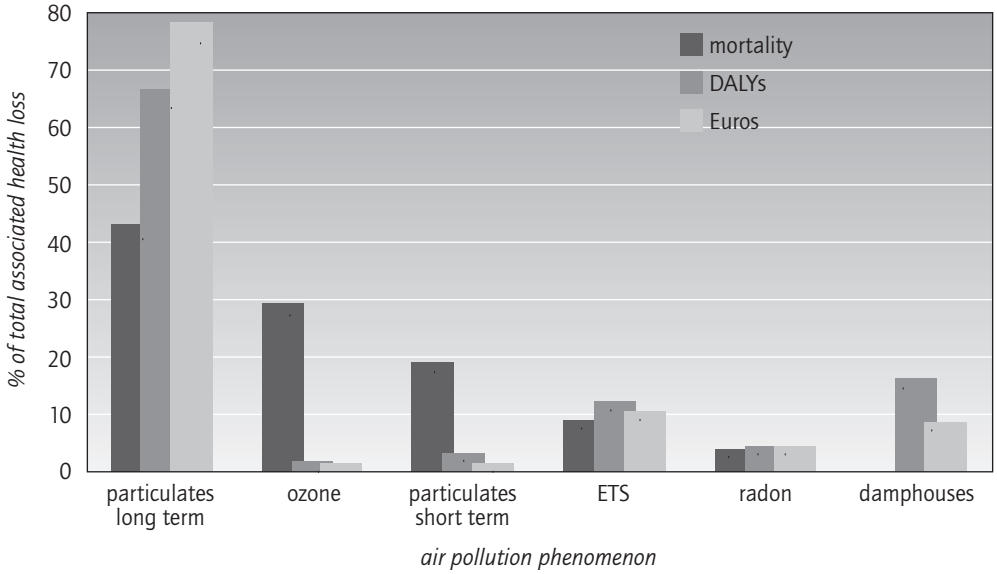

Figure 5-2. Overview of health impact of air pollution type as percentages of total air health loss expressed in terms of fatality numbers, DALYs and Euro.

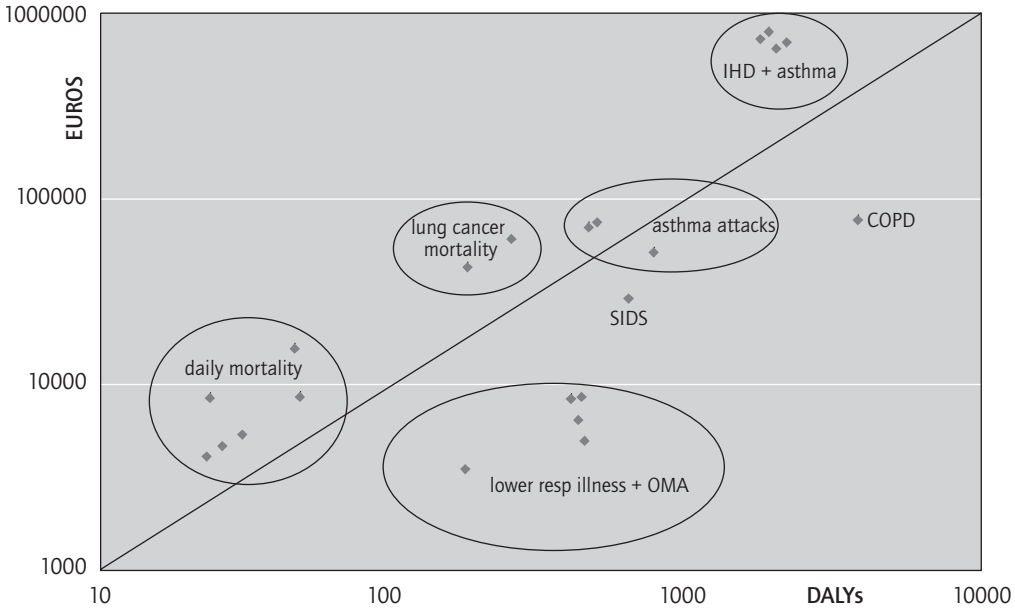

Figure 5-3. Response specific DALYs plotted against response specific euro.

saved. We emphasise that besides health gains the investments in emission reduction have yielded several other benefits, such as a substantial decrease in ecological impacts, improved and more energy-efficient engine technology. In sum, according to these calculations it was money well spent.

The total air pollution attributable disease burden is estimated to be in the order of 50,000 (5-95%-tiles: 25,000-75,000) DALYs annually. As expected the health loss attributable to environmental exposures is small but substantial in the Netherlands. Recently the total annual burden of disease was estimated to be in the order of 3 million DALYs[47]. According to the calculations presented here, the air pollution impact described here would contribute slightly less than 2 percent to this total disease burden. In the Netherlands life-style factors, such as smoking, physical inactivity cause a burden of disease of 14.7, and 4.5% of total disease burden (DALYs) respectively[14]. High cholesterol is an example of a single factor for which the attributable disease burden is in the same order of magnitude, 2.5%.

The metric used to express air pollution associated health loss, mortality risk, DALYs or Euros has an obvious effect on rank order of the different types of air pollution effect (figure 5-2). The mortality counts associated with air pollution episodes are not reflected in the amounts of lost DALYs and Euro, as a part of it is considered to be due to 'harvesting' of those with only a limited time to live. No mortality is calculated for damp houses, but chronic bronchitis associated with damp housing weighs heavily in terms of DALYs and Euro.

In figure 5-3 for each air pollution exposure the disease burden in DALYs is plotted against monetarised health loss on a log-scale. On average the DALY is worth around 300,000 Euro, but the plot is clearly very scattered.

5.8 Discussion

5.8.1 Health impact assessment

Several potential flaws are associated with this type of impact assessment. Most of these are not specific to the approaches and metrics we have proposed here, but concern health impact assessment in general. Recurring, almost inevitable shortcomings involve the imprecision of population exposure assessments, the unknown, and probably 'unknowable' shape of the exposure-response curves at low, environmental exposure levels, and the translation of exposure-response information from one species to another, as well as from one population to another.

Another important issue is the internal and external validity of epidemiological results. In view of the way in which our aggregated results are dominated by the long-term public health effects of particulate air pollution, we have to stress the fact that these estimates are based on the results of only two American cohort studies and a small Dutch pilot study[71,72,108,109]. Given the inherent shortcomings of this type of retrospective epidemiological study, especially with respect to the validity of the exposure indicators, the relatively large health impact calculated here still needs to be confirmed in other well-designed studies, such as the Dutch study in which exposure assessment is at least performed on an individual level. Even though most of Hill's criteria for causality are met, substantial uncertainties remain unsolved with respect to specific causative agents and mechanism of actions[18,19,110,111].

Most of the quantitative health impact assessments presented here are fairly sensitive to the choice of reference level of exposure. Suspended particles, ozone and other oxidants in outdoor air, radon or allergens in indoor air are a fact of life, even without any human activity. Here we have more or less applied the concept of feasible minimum exposure distribution. If we optimise the way we build our houses as well as our ventilation behaviour radon levels should not exceed 16 Bq/m^3 and damp houses would be

very rare[112]. In principle a world without smokers is possible, so exposure may be zero. For particulates (PM_{10}) and ozone we tentatively applied annual concentrations of 10 and 15 mg/m^3 as baseline exposure levels.

The most fundamental problem we encountered was lack of complete and quantitative insights into how air pollutant exposures are involved in the onset and development of human disease. We have only incorporated exposure-response associations which have been studied comprehensively in epidemiological research and for which clearly defined health outcomes have been established. 'Health events' that are recorded in epidemiological surveys or health care registration may only be the tip of the iceberg, with many minor impairments remaining beneath the surface undetected (see figure 4-2)[30]. In particular, time-series analyses provide us with the means to detect relatively small elevations of mortality and hospital admission during episodes. It is almost inconceivable these relatively severe health events would occur without a greater part of the population suffering from transient lung function deficits, or asthma or angina attacks, resulting in an increase use of medication, visits to the general practitioner or even an emergency room among the more susceptible. However, obvious means of investigation, such as panel studies appear to lack sufficient power to detect an increased incidence of such endpoints[6,113].

5.8.2 Environmental health impacts on a scale

Attributing impact weights to air pollution related conditions involves societal and individual values and preferences which does not always agree well with the scientific traditions in several disciplines. At the same time, normative evaluation of health-endpoints is virtually inevitable in health risk assessment (chapter 2). Even using annual mortality risk as an impact measure is value-laden, as non-fatal health outcomes as well as age at death (loss of life expectancy) are implicitly ignored. The same applies for health-based exposure guidelines, for which value judgements have to be made regarding the health significance of toxicological or epidemiological response variables.

Whose values matter and how are these best assessed? A large number of studies have revealed that the health related quality weights attributed to certain health states may differ between patients and non-patients[14]. Health status weights for DALYs are mostly derived by calling in health professionals, who are able to assess the various dimensions of disease. WTP-values are determined with the help of large-scale surveys. Both have their own systematic biases, but policy decisions about resource allocation should adopt a societal perspective and thus may involve these types of generic preference classifications. Furthermore, it is important to notice that health preference measurements tend to be rather stable and reproducible, even across countries[38,41,115].

In health economics there are five main ways to elucidate preferences with respect to health status quality: standard gamble (choosing between probabilities of possible outcome), time trade-off (exchanging time with different health states), person trade-off (exchanging persons with different health states), contingent valuation and revealed preference (wage-risk, hedonic pricing etc.). Results produced by different methods may differ substantially indicating that many socio-psychological processes are not accounted for. An appealing example is that most people find it impossible to exchange relatively minor morbidity outcomes among many with death among few (e.g. reluctance to give up life). Several authors have claimed that real life preferences with respect to health and life expectancy are much too complicated to be covered by the simple concept of DALY. They argue that the plain observation that preferences are not stable over a life-time and will depend largely on whether health states will occur near or far into the future makes application of DALYs questionable. Others favour a more practical approach: there are simply no better alternatives[116].

Uncertainty analysis shows that altering weights within the variance seen in most weighting exercises does not substantially affect the overall picture. Compared with the huge uncertainties that are often connected with health impact estimates, the effect of the possible variance in attributed weights appears rather small. There is an important exception, however. The lower the disability weights attributed to health states, the more sensitive they are to variation. It is much easier to double the small weight given to severe noise annoyance than to double that of the terminal state of lung cancer. This is reflected in the results of the uncertainty analysis with respect to 'respiratory illness'. The variation of the output probability distribution is largely due to variation in the severity weights given by panel members. Since the less severe responses tend to affect the highest number of people, there is some room for 'manipulation' of results, e.g. increasing the public health significance of 'one's favourite health risk'.

5.8.3 Worth the money?

The calculations with respect to the monetary valuation of air pollution health impacts indicate an enormous loss in economic terms. However, the loss is primarily due to the long-term effects on survival associated with particle exposure. Regarding this association there is still substantial uncertainty on biological mechanisms and causal agents. This dilemma of uncertain health impacts yielding a huge health loss stresses the necessity to complete the economic appraisal with accepted methods for economic valuation of uncertainty[37,25].

As already mentioned, transfer of WTP-values from other studies can be tricky, since valuations may be largely determined by contextual variables, comprising risk perception or the respondent's perspective (individual versus collective, altruistic versus self-centred). An economic appraisal of air pollution health effects may profit from WTP-estimates based on European contingent valuation studies that directly concern air pollution health effects in the right context.

Air pollution attributable health loss expressed in DALYs is not completely proportional to the health loss in terms of Euros (Figure 7), although there is some convergence. Theoretically this might be explained by the fact that both measures are based on different principles. Monetary valuation may involve all possible dimensions of reduced health, trading off health and other valued services money can buy, while DALYs are an aggregate of only three formal dimensions, number of people involved, duration and severity. It is unlikely that the same dimensions are valued for all diseases. For instance, the mortality-aspect of DALYs (YLL) depends on life expectancy alone, while the non-linear relation between WTP-values and age at death suggests that individuals value other dimensions too. Health responses to exposures that are dominated by mortality, such as the impacts of ozone and particles (long-term) produce relatively higher health loss estimates for Euros than for DALYs and vice versa (short-term responses and morbidity due to particles). Of course one has to bear in mind that many shortcomings are connected to an economic appraisal of the prevention of statistical fatalities as well as morbidity risks. For instance the wage-risk method relies on the assumption of enough labour mobility for workers to really have a free choice, which is doubtful in most cases[37]. With respect to the contingent valuation method to unveil general WTP figures a number of objections have been raised. In the first place it is a highly hypothetical number, i.e. it is not real money simply what people claim they would spend in this 'virtual' market place. Respondents may also show 'strategic' behaviour. People with 'green' preferences may intentionally exaggerate their willingness to pay; others may provide interviewers with politically correct answers. Respondents may feel it is unethical to put a price tag on someone else's death or illness. Furthermore, given the often-complex nature of the questionnaires contingent valuation methods are vulnerable to many potential biases ('starting point bias', 'interviewer bias', and 'embedding bias'). Some health end-points may be rather

hypothetical to most of the respondents, but at the same time familiar to others, e.g. asthmatic attacks. Familiarity with end-points such as asthmatic attacks, and chronic bronchitis appears to increase WTP[44,117].

Thus, there is reason for concern about the stability of the quantitative preferences of respondents into which we have only limited insight. For instance, many risk perception studies underline the importance of context variables, such as personal interest in risk source, perceived personal and institutional control over risk generating activities, degree of voluntariness of exposure, inequity with respect to distribution of risks and benefits[118]. Other socio-economic and demographic variables may also be important, such as base-line risk, or personal income and education. This type of uncertainty may not fully justify the transfer of WTP-measurements to health impact assessment of air pollution. In particular, most of the WTP-studies involving morbidity risks have been done in the US and there are systematic differences between the US and Europe in the cultural and socio-economic dimensions discussed here. Therefore the results of WTP-calculations should be regarded as crude estimates, allowing comparisons between different risks rather than being real money.

In the framework of this study we have not fully considered the feasibility or costs of measures to reduce air pollutant exposure. It is clear that current ambient air concentration of particles and ozone can only be reduced at high economic and societal costs, while a certain level must be considered as a fact of life. For environmental tobacco smoke and dampness in homes measures to reduce population exposure may be less expensive, for a comparable benefit although data on these indoor factors in this context are lacking. Measures to reduce radon levels in newly constructed houses, such as performance standards for building materials and facilities to increase the ventilation rate (including energy costs) would cost somewhere between 50,000 and 150,000 Euro/DALY (see chapter 2)[119].

5.8.4 Equity and efficiency

It is important to note here that the choice of an indicator to represent environmental health loss is not just an academic question. It is not just coincidence or lack of methodology that lies in the choice of mortality risk as the classical health loss indicator. Managing risk based on mortality guarantees everybody is treated equally, while simple application of health adjusted life-years as a measure of health impact is not without serious distribution or ethical consequences. In principle, society would benefit from passing health risks to the elderly since they have less life-years and health to lose. Furthermore, the use of DALYs implies that people with a disease count less than healthy people. In this respect some authors warn against double jeopardy. People with poor health suffer a disadvantage twice: first they are disabled, and secondly the saving of one year of their lives counts less than that of an healthy person. One can strive to maximise utility, but one can also strive to concentrate efforts on people with the worst health, or the greatest improvement potential[120].

WTP-values are also dependent on income. Application of WTP may thus violate equity principles, especially when locally derived WTP values are applied globally, typically giving less weight to third world health problems[18].

5.8.5 Conclusion

In spite of methodological and ethical problems, presenting health impact in terms of DALYs and/or money offers a useful framework for *explicit evaluation* and *comparison* of health loss associated with different environmental exposures, involving a wide variety of non-fatal health outcomes. It enables incorporation of the public health interest in decision making with respect to environmental quality and spatial planning (e.g. extensive infrastructure projects involving a range of diverging, often accumulating

exposures). For instance, in scenario studies aggregates can be applied to explore the 'health' score of different options, evaluated from the perspective of different policy philosophies.

The calculation of DALYs associated with environmental exposures provides a comparative picture from the viewpoint of public health. However, we would not suggest an immediate change of environmental policy priorities based on this type of calculation. Policy priorities may be partly explained by dimensions of health risk perception, which are not captured in our approach, such as 'dread', voluntariness of exposure, the perceived controllability or familiarity of risk generating processes (e.g. traffic). In particular, the social distribution of risk and benefit[118] is not covered in our approach, as by definition a slight reduction of health amongst many may be equal to severely affected health amongst few. From the viewpoint of policy makers the principle of equity may prevail.

Application of monetary valuation of health-endpoints in principle allows formal cost-benefit analysis of policy options to improve the air pollution situation from different perspectives. As further air pollution control becomes more and more expensive and will increasingly affect individual behaviour rather than institutions (e.g. the energy or industrial sector), a crude indication of what we are willing to pay to avoid health impacts attributable to air pollution may improve the policy making process. However, this would necessitate more adequate WTP-estimates, preferably from European studies explicitly dealing with air pollution situations.

Application of aggregate health metrics, such as DALYs and WTP can be of help in environmental health policy as long as they are not considered as the ultimate 'health coin', and as long as other criteria, such as equity and solidarity are incorporated in decision making as well.

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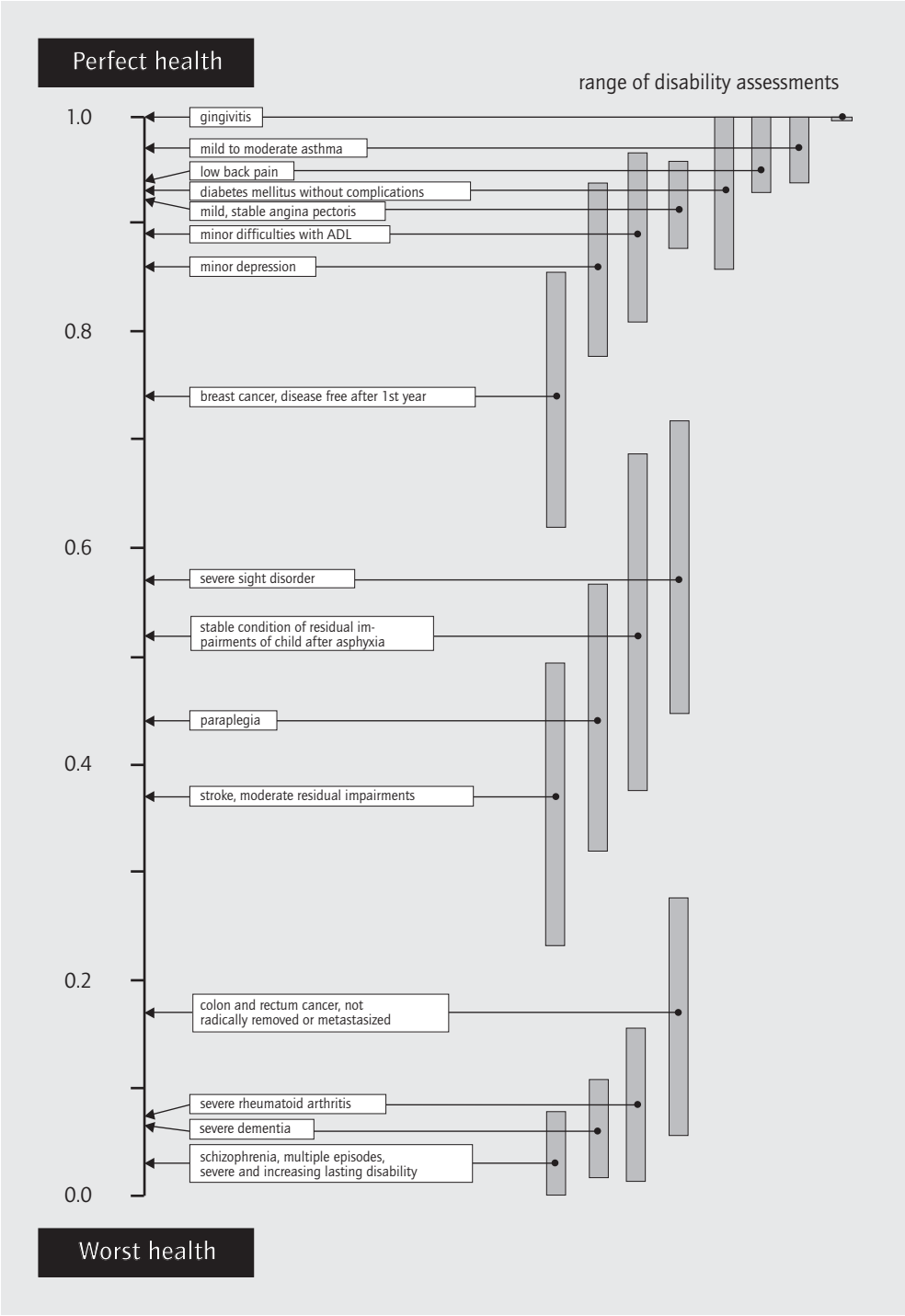

Figure 5-4. Scale of 'calibration' health states, employed to attribute weights to health states associated with environmental exposures.

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6 Community noise burden of disease: an impossible choice of endpoints?[i]

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Abstract

In this chapter we discuss a number of approaches to assess the impact of exposure to community noise on public health. Briefly noise characteristics and how they are accounted for in noise metrics are discussed. Successively we assess the current evidence on a range of health endpoints published in the open literature. We apply a conceptual model to make a synthesis of current evidence, linking together the information on individual responses proposing a mechanistic explanation of health effects shown to be associated with noise exposure. Finally, we present a number of different options for crude estimation of burden of disease attributable to noise exposure. This yields an interesting dilemma: the association between noise and 'annoyance' has a high validity, but the public health significance (content validity) is controversial. The other way round, the clinical significance of cardiovascular disease is obvious, but a causal relation with noise exposure is not yet fully supported by available epidemiological data or scientific consensus.

[i] *Written as a discussion paper to support the development of a guide for Environmental Burden of Disease estimation to be published by the World Health Organisation (WHO) in Geneva and for the implementation of the UNECE –WHO Transport Health and Environment Pan European Program (PEP) and the Ministerial Conference on Environment and Health in Budapest in 2004, by* Augustinus E.M de Hollander, Elise EMM van Kempen, Brigit AM Staatsen.

6.1 An inescapable nuisance

6.1.1 Community noise

Noise is a ubiquitous, persistent and largely inescapable environmental pollutant in most of the (post-)industrial and motorised world (see table 6-1). Transport, comprising road and rail traffic, aviation and shipping, is the most important source of community noise. In specific local situations, stationary sources, such as industry, building sites, sport and leisure facilities may also be a significant source of community noise. In most surveys noise produced in and around the house is reported as a significant cause of annoyance as well, especially in urban environments. This paper will address primarily the first category, as it is typically part of the policy domain of environmental protection.

Table 6-1. The changing 'soundscape'. Based on a comprehensive analysis of historical documents, literature, paintings and poems Schafer estimated the relative contribution of natural, human and machine sound to total noise exposure in different era of human civilisation[1].

Epoch	natural sound (%)	human sound (%)	machine sound (%)
early civilisation	69	26	5
medieval times	34	52	14
industrial age	9	25	66
present	6	26	68

In the Western world engine sound emission levels per vehicle have decreased substantially due to rapid improvements of technology. However, as a result of the application of broader tires the net sound production of private cars has not decreased. New generations of aircraft have become substantially more silent[2]. Furthermore modern urban development often includes spatial separation of transport and residential functions. However, average exposure to noise is still increasing in most countries as a result of tremendous growth in volumes, especially in the developing world. According to the OECD growth will increase even more rapid in the next 20 years. In its Environmental Outlook projections predict an increase in motor vehicle kilometres of 40% in the OECD regions and of 86% in the developing rest of the world. The largest increase is expected in aviation, where the number of passenger kilometres will certainly triple, with the highest growth rates in Asia[3]. Furthermore, the '24 hours economy' will not only increase the level and area of noise exposure but will also affect the brims of the night[4].

In European OECD countries around 30% of the population is estimated to be exposed to levels of road traffic noise above 55 dB(A), and around 13% above 65 dB(A) (L_{dn}-values)[5]. Other estimates yield even higher fractions of the population exposed to levels exceeding health-based guidelines[6]. Some 10% of the EU-population is estimated to be seriously disturbed by aircraft noise. Estimates from other regions in the World are scant, but occasional reports indicate the problem of residential noise pollution is not confined to the developed world[7]. Anecdotal surveys report high levels cities in developing countries with scant regulations and little formal urban planning, such as Karachi city, Bombay and Calcutta, where noise levels along busy roads ranged between 80-85 dB(A) (Leq, 8h), respectively 80-92 dB(A) (Leq, 24h), with peaks up to 140 dB(A) during rush hours[8,9].

6.1.2 Somewhere between well-being and health

Long-term exposure to noise has been associated with a wide range of effects on human health and well-being. Even though they are not independent, following the argumentation of chapter 3 one might make a

distinction between *social psychological* responses, such as annoyance and disturbance of daily activities on the one hand, and *clinical* effects on the other, such as hearing loss, hypertension and aggravation of cardiovascular symptoms[10].

Noise is a major problem in modern societies. Not many people will argue with that. It is well established that as levels of noise exposure increase a higher percentage of any representative population will report to be severely annoyed. Noise intrudes into our personal privacy, disturbs our daily activities, causing annoyance, stress and affecting the quality of our life. No doubt this presents an important social problem. Whether this is a health problem as well is less obvious. Nonetheless, it is a fact that several studies have demonstrated an association between serious annoyance, and highly correlated social responses, such as perceived stress, anxiety, and risk perception on the one hand and reported (psychological) symptoms, cognitive complaints, (self-)medication and use of health services on the other. The causality of this association however is far from obvious[11].

6.1.3 Reader

In this chapter we present and discuss some proposals for simple, spreadsheet assessment of the health impact of exposure to noise at the population level. As promoted by the World Health Organisation comparative assessment of health impacts may facilitate policy makers to properly involve public health interest in infrastructure and urban planning[12,13].

We will begin with a short explanation of noise characteristics and how they are accounted for in noise metrics. This is followed by an assessment of the current evidence on a range of health endpoints published in the open literature. We will discuss a conceptual model to make a synthesis of current evidence, linking together the information on individual responses proposing a mechanistic explanation of health effects shown to be associated with noise exposure. Finally we present a number of simple methods for a crude estimation of burden of disease attributable to noise exposure, using the Dutch situation as an example.

To arrive at a quantitative assessment of disease burden associated with population exposure to community noise we will follow an exposure-based approach consisting of the following steps[14]:

review of causality, internal and external validity of health outcomes associated with noise exposures, followed by a *quantification of exposure response relations*

assessment of a *population exposure distribution*, using either national noise monitoring data and exposure models if available, or a robust model using data on degree of urbanisation, traffic or vehicle density, vehicle composition and emission data, aircraft noise distribution patterns etc.

estimation of *noise exposure attributable disease burden*, incorporating location specific base-line incidence or prevalence risks for health outcomes*15*.

6.2 Characterisation of noise exposure

6.2.1 Characteristics

Sound is a physical phenomenon of alternating compression and expansion of air that propagates from a source in all directions. These can be described as small pressures around the atmospheric pressure. The frequency of these alternations determines the pitch of a sound. A high pitch tone (e.g. 4,000 Hertz) has a squeaking sound; a low pitch tone (e.g. 200 Hertz) has a humming sound. Noise is adequately defined as "unwanted sound" (in physics there are formal definitions as well[11]).

To assess the impacts on human health and well being various biophysical metrics of noise are relevant. In general these metrics are based on physical quantities to which "corrections" are applied that

take into account certain human noise sensitivity. These corrections depend on the frequency, noise characteristics (impulse, intermittent or continuous noise levels), and the source of noise. within the framework of this chapter the following metrics are important.

Sound pressure level. The sound pressure level (L) is a measure of the air vibrations that make up sound. Because the human ear can detect a wide range of sound pressure levels (from 20 micro-Pascal up to 200 Pascal), sound levels are expressed on a logarithmic scale with units of decibels (dB). An increase of 10 dB is experienced by the human ear as a doubling of the loudness of the sound

Sound level. The human ear is not equally sensitive to sounds at different frequencies. To reflect the spectral sensitivity of the human ear, spectral sensitivity factors are used to weigh the sound pressure level at different frequencies (A-filter). These, so called A-weighted sound pressure levels are expressed in dB(A).

Equivalent sound levels. When sound levels fluctuate in time, as is usual the case with community noise, the equivalent sound level is determined over a specific period of time. For this purpose the A-weighted sound level is averaged over a period of time (T), using a prescribed procedure (symbol $L_{Aeq,T}$). A common exposure period T in community studies/regulation is from 7 to 23 hours ($L_{Aeq,7\text{-}23hr}$).

Day-night level (L_{dn}). This level is used in environmental impact assessment as it correlates much better with community annoyance than the equivalent sound level. L_{dn} is the equivalent sound level over 24 hours, increasing the sound levels during the night (23-07 hours) by 10 dB(A). Also a "day-evening-night level" (L_{den}) is constructed in a similar way, increasing the sound levels in the evening (19-23 hours) by 5 dB(A) and those during the night (23-07 hours) by 10 dB(A).

Sound exposure level of a noise event. The sound exposure level (SEL) of a noise event, such as the noisy passage of an aircraft is the equivalent sound level during the event normalised to a period of one second.

Usually, the values of these metrics are assessed outdoors. In some sleep disturbance studies indoor noise levels have been measured.

In European countries, A-equivalent indices (L_{Aeq}-type) are more common than statistical indices (L_{10} -, L_{50}-type). Unfortunately, noise indices differ per country and within a country even per transport mode. Especially the indices used to describe noise exposure by aircrafts vary considerably between the different countries[16,17].

6.2.2 Calculation methods

Noise exposure (distribution) is usually assessed by calculations according to national noise calculation methods. In the Netherlands, for road traffic 'RMV2002' is the method, prescribed by law. Like all national European noise calculation methods, RMV2002 first calculates the noise emission by the source, taking into account the characteristics of the source (type of car, speed, type of pavement, height of the source etc.). The next step is the calculation of noise loads at the receiver.

To do so, characteristics of the noise propagation path have to be taken into account (e.g. distance, type of ground, presence and type of buildings or other objects etc.). Furthermore, air absorption, determined by the air temperature and humidity, plays a role. Corrections can be made for the meteorological conditions (temperature, wind). To estimate the number of people exposed, noise propagation models are combined with demographic data e.g. using a geographic information system[18].

6.3 Reported effects on human health and well being

In this section a brief review is presented of all health and well-being impacts that have been reported to

be associated with the exposure of populations to noise. We will distinguish two types of end-points: *social-psychological* effects and *clinical* end-points that can be linked to diseases in a strict sense (e.g. by an ICD-code, see chapter 2). This review is based on a fair number of recent reviews by national and international scientific advisory committees or established scientists[19,20,21,22,23,24,11].

6.3.1 Social-psychological endpoints

6.3.1.1 Annoyance

Annoyance is probably the best-studied response to noise exposure, while at the same time it is probably the least well defined. Several definitions have been proposed: "a feeling of displeasure associated with any agent or condition, known or believed by an individual or group to adversely affect them"[22] or "a feeling of resentment, displeasure, discomfort, dissatisfaction or offence which occurs when noise interferes with someone's thoughts, feelings or daily activities"[25]. The degree of annoyance provoked by noise exposure depends on several characteristics, such as sound level, spectral characteristics and variations with time of the day or season; evening noise is more annoying. However, annoyance also depends on non-acoustical factors, cognitive factors. Noise exposure explains only 25-30% of observed variance in annoyance reports. These may be endogenous, such as age, psychological status, individual noise sensitivity, fear with respect to the source, perceived control over the situation, perceived economic or societal advantages of noise generating activity, or exogenous, such as poor housing, or other environmental exposures[26,27,28].

Noise annoyance is always assessed on the level of populations using questionnaires. Based on the results of that type of investigation exposure response relations have been derived for the three main types of transportation noise: road, rail and air traffic (see box 1). These relations pertain to populations exposed to noise at specific levels for more than a year. The effect is given as the percentage of the people highly annoyed by a specific community noise, involving those individuals in the studied population who report a degree of annoyance in the worst quarter range of answer categories[23,29]. These results are fairly consistent on the level of populations; of course they have very little predictive value on the individual level.

People start reporting severe annoyance at L_{dn} values of around 42 dB(A), annoyance at 37 dB(A), measured at the front of dwellings. At increasing levels annoyance increases most with aircraft noise, followed by highway traffic noise, normal road traffic and railway noise. Studies have all been done in Western-European, Australia, Japan or the United States[7]. No studies from other regions are available.

6.3.1.2 Psychosocial well-being and psychiatric disorders

Several cross-sectional studies indeed revealed a higher prevalence of symptoms such as "headaches", "being tense and edgy", "social orientation", "acute symptoms of depression irritability", "burns, cuts and minor accidents" in high noise areas compared to low noise areas. A number of studies applying psychometric questionnaires to assess psychological morbidity yielded inconsistent results, including specific research of school children[21,11,22,30]. Early studies have found associations between the level of aircraft noise and admissions to psychiatric hospitals in London and Los Angeles. Other studies, and reanalyses of old studies failed to confirm these findings. Noise exposure does not appear to be a decisive factor in hospital admission, among many other psychosocial variables[11,20,21].

Due to inconsistent results and inherent methodological shortcomings no definite conclusion with respect to the causal role of community noise in psychological morbidity can be drawn.

6.3.1.3 Performance

Laboratory studies reveal that noise exposure may have a significant effect on performance. While performing tasks, noise may contribute to arousal, alter (mental) task strategy, distract attention to task aspects. Noise may also affect social performance, mask speech, impair communication, and distract attention from relevant social cues. Results of several studies indicate noise may affect verbal memory tasks, and influence the process of selectivity in memory and attention.

Epidemiological studies demonstrate that equivalent sound levels during school time over 65-70 dB(A) may impair the performance of schoolchildren in cognitive tasks. Children are vulnerable to noise effects because noise may interfere with learning during a critical developmental period, and children have less capacity than adults to anticipate, understand or cope with stressors. Tasks that involve central processing and language comprehension, such as reading, attention, problem solving and memory appear most influenced by noise exposure. Prolonged exposure to noise is found to be associated with deficits in sustained attention, visual attention and concentration, with poorer auditory discrimination and speech perception, memory impairment and poor reading ability and school performance[30,31,32]. Whether community noise is actually affecting the mental health (hyperactivity, depression, anxiety) of school children is still subject of debate, partly due to disagreement on definitions of mental health outcomes[33,33].

Human society depends strongly on speech, which is subject to masking by noise. Community noise, especially varying and intermittent noise, can interfere with many activities involving speech. The extent to which any particular degree of speech interference can be overcome or contribute to stress in various situations is not well understood[22].

6.3.1.4 Sleep disturbance

Night-time noise may affect sleep in various ways, resulting in degradation of sleep quality (primary effects), disturbance of functioning, performance or mood the next day (secondary effects). Noise of sufficient intensity may affect the sleep *pattern*, increasing the number of people reporting difficulties falling asleep. There may also be a chronic effect, such as medication use, of prolonged stress. Sleep disturbance as a result of night-time noise is measured by various indicators, such as sleep-pattern, (self-perceived) sleep quality, attention tests (performance) or mood-questionnaires the next day. Motility as measured by wrist-actimetry is an indication for the number of awakenings during the night.

Noise increases the changes between sleep *stages* (awake, 1, 2, 3, 4, REM) and the number of awakenings during the night, respectively starting from SEL levels of about 35 and 60 dB(A). Reported sleep quality is likely to be affected at nightly noise levels above 40 dB(A). In most studies an effect of night-time noise on performance and mood the next day is only seen at levels above 60 dB(A). In large field studies often motility is measured as an important indicator of noise induced arousal and sleep disturbance. Age, sex, season, medical condition and medication are important factors of influence with regard to the level of sleep disturbance[34].

Night-time noise exposure may increase heart rate during the night; habituation to this effect does not seem to occur. The observation thresholds is a SEL value of 40 dB(A). There are indications noise-induced increased sleep stage changes and awakenings are associated with elevated hormone levels (ephedrine)[25].

6.3.1.5 Absenteeism

Several epidemiological studies suggest the level of noise during working hours has an influence on the rate of absenteeism, even among office workers ($L_{Aeq,8h}$ > 75-90 dB(A)). However, most studies are flawed

in several aspects and no firm conclusions can be drawn. There are no studies on community noise and absenteeism[23].

6.3.2 Clinical effects

6.3.2.1 Cardiovascular disease

Recently in a meta-analysis we reviewed studies on the association between exposure to noise and cardiovascular risks both in occupational and environmental settings. Responses included increased blood pressure, hypertension, use of anti-hypertension drugs, consultation of GP or specialist, use of cardiovascular medicines, Angina pectoris, myocardial infarction and prevalence of ischaemic heart disease[10]. The analysis revealed some inconsistencies among sometimes contradictory results of individual studies, and summary relative risks were statistical significant only in a limited number of end-points.

As most of the studies were of the cross sectional type, several methodological flaws could be identified, such as poor (retrospective) exposure assessment. In most studies there were limited possibilities of controlling confounding variables and selection bias (self-selection: healthy worker, healthy neighbour effect), both extremely important with respect to cardiovascular disease.

Results of a series of recent cross-sectional studies are consistent with a slight increase in the prevalence of hypertension due to noise exposure[35,36,37,38,39]. But methodological limitations, especially with respect exposure assessment, and lack of statistical significance still do not allow firm conclusion. In most reviews the statistical evidence for a causal relation between noise exposure and cardiovascular health risk is considered to be on the verge of conclusive. However, in most of them a small effect on cardiovascular risk is deemed highly plausible, especially while the overall results on the full range of endpoints from slight elevation of blood pressure to Angina pectoris are consistent with known cardiovascular disease progression and supported by laboratory studies on blood dynamics. Nevertheless, well-designed cohort studies will be needed to confirm these indications[21,22,23,20,10,40,41,42].

6.3.2.2 Noise induced hearing loss

Hearing impairment is typically defined as an increase in the threshold of hearing, as assessed by audiometry. Even small values of hearing damage may affect understanding of speech in conditions of every day life. Since speech is the most common means of communication among people, hearing loss should be considered as a severe social handicap. Prolonged occupational noise exposure below a $L_{Aeq,8h}$ of 75 dB(A) is not likely to induce a permanent shift in threshold of hearing. Scarce epidemiological studies of exposure to community noise fail to demonstrate hearing damage below a $L_{Aeq,24h}$ of 70 dB(A).

Current evidence suggests the risks at typical environmental levels of exposure are low. Exposure of children and young adults to loud music during leisure activities (concerts, headphones) may give rise to concern, especially when peak sound pressure level exceed 120-140 dB(A)[23].

6.3.2.3 Other clinical health effects

There is no convincing evidence for a direct effect of exposure to noise on health outcomes such as congenital abnormalities, birth weight, or disorders related to the immune system (infectious or auto-immune disease). So far plausible mechanisms of action for these disorders are lacking[21].

6.4 'Noisy' epidemiological data

6.4.1 Noise and signals

Among the strong and consistent health impacts of (potentially confounding) social-economically-determined factors, such as life-style or occupational health risks one will often search in vain for an independent effect of environmental exposures in available health statistics, such as mortality and morbidity rates or medical consumption (see chapter 3)[43,44]. Furthermore, the attitude towards the source of noise, and sensitivity to noise may account for more variation than the level of noise exposure by itself[21,26]. People who report themselves seriously annoyed might just tend to report symptoms, use self-medication or even visit a GP, irrespectively of actual noise levels. The strong association between socio-economic and environmental conditions, the expected small increases of health risks attributable to environmental exposures combined with random variation produce a 'signal-to-noise'-ratio that goes beyond the resolution of available epidemiological methods (see chapter 2). This is reflected in the fairly inconclusive nature of the results from epidemiological research on noise and cardiovascular disease.

6.4.2 Health significance

The impact of hazardous environmental exposures on human health can take numerous shapes of various severity and clinical significance. Some effects occur soon after the onset of exposure; others emerge after long-term cumulative exposure, including a latency period. Environmental exposures may induce *biochemical, physiological or (socio)psychological changes* that more or less fall within the normal range of biological variation. Whether these changes are of any significance to health depends above all on the degree to which the *function* of organ systems (or social-psychological functioning) is affected, the *reversibility* and *duration* of the changes and the possibilities for *recovery* or *compensation*, and on the possible loss of *resilience*. Acute, transient elevations of blood pressure and pulse rate, palpitations, increased serum levels of stress hormones due to noise exposure might all fall largely within normal homeostasis. On the other hand sustained haemodynamic effects, such as hypertension or prolonged stress-mediated hypercholesterolemia would surely be significant to public health. Given a certain population distribution of for instance serum cholesterol level or systolic blood pressure, even a small shift due to environmental exposure may yield a substantial increase in the prevalence and mortality of cardiovascular disease[23,40,45,12].

6.4.3 Harvesting?

At the level of populations these 'subtle' effects may cause some increase in acute morbidity and mortality. For instance several studies have shown some association between average noise levels and the incidence rates for myocardial infarctions[10].

It is plausible that these infarctions have been initiated by 'subtle' haemodynamic changes due to noise exposure, comparable to precipitation of morbidity or mortality ('harvesting') as described with regard to air pollution episodes[46,47]. A similar mechanism could probably apply to the association of admissions to psychiatric hospitals with traffic noise levels found in some studies[48]. It is not very likely people are suffering from psychological disorders merely by noise exposure, but it might induce episodes of symptom aggravation (see figure 2-3). On the other hand one has to appreciate the fact that the very same physiological responses, such as transient elevations of stress-hormones, haemodynamic and biochemical variables, are considered beneficial when induced in physical exercise or sports[20].

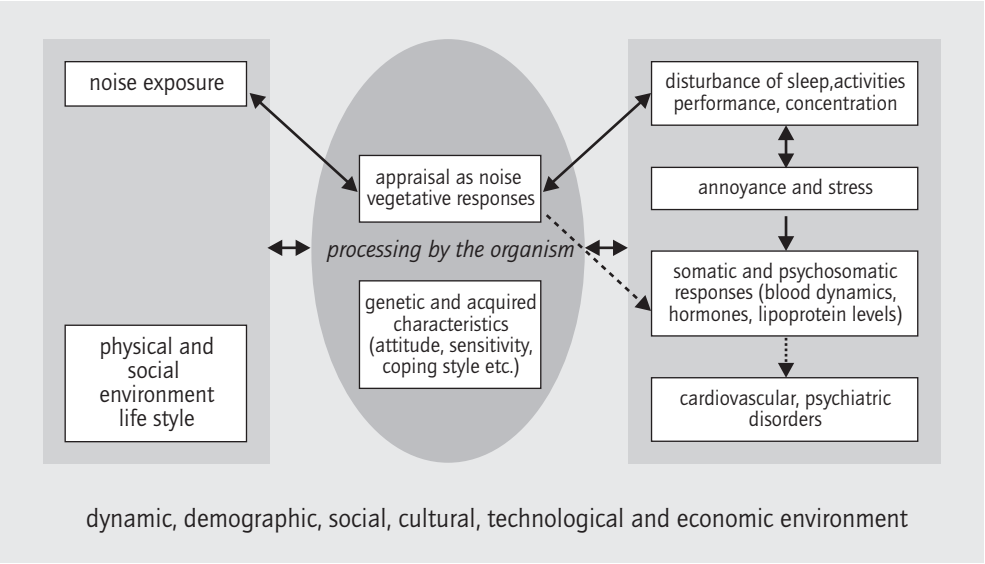

Figure 6-1. Conceptual model representing the relation between noise exposure, health and quality of life[21,67].

6.4.4 Well being and health

Social responses like annoyance, anxiety or perceived health risk may or may not fall within the background 'noise' of normal daily hassles. Of course at some point a clear distinction from clinically defined anxiety or depression can hardly be made. Unfortunately good longitudinal data are lacking with respect to onset and development of psychological disorders in this respect. A similar argument can be made for sleep disorders. Sleeping problems and their influence on mood and performance the next day are part of every normal life. However, at some point sleeping problems may become clinically significant as normal physical, mental and social functioning is hampered.

6.4.5 Conceptual model

An appealing conceptual model depicting the possible mechanisms involved in noise-induce disease genesis was proposed by the Health Council of the Netherlands (see figure 6-1). Noise may directly or indirectly influence vegetative, hormonal, cognitive and emotional regulation mechanisms of the organism. Indirect influences may be related to disturbed activities such as communication, recreation or sleep. The negative appraisal of noise may lead to short-term impairment of the organism, for instance through the production of stress hormones or by annoyance and resignation. Continuing noise exposure would result in chronic dysregulation of the organism. This might even include unfavourable life-style aspects, such as taking up smoking or drinking, or other risk seeking activities. On the level of populations this all may lead to an increased prevalence of chronic disease.

The model suggests that exposure to noise in itself is a health risk factor. However, various other factors may modify the way in which the "noise signal" is processed by the organism and will increase the impacts on health and well-being. Examples of such factors are the familiarity, individual or community attitude towards the noise source, and, of course, individual noise sensitivity[21].

6.5 Approaches to assess disease attributable to noise

6.5.1 Introduction

As we have seen in chapters 2, 3 and 4 disability-adjusted life-years (DALY) may be an appropriate aggregate indicator to represent the multiform health loss due to environmental exposure to noise and thus enable comparative risk assessment. At least three important dimensions of public health are covered in the health adjusted life-years, such as DALYs, viz. loss of life expectancy, loss of quality of life, and number of people affected (social magnitude)[49]. Time is the unit of measurement. Public health loss is defined as time spent with reduced quality of life, aggregated over the population involved, and combining years of life lost and years lived with disability that are standardised by means of severity weights (figure 2-4, in this diagram 'health loss' due to severe noise annoyance is suggestively added)[ii,50]..

To assess loss of DALYs attributable to environmental exposures, information on population exposure distribution, exposure-response relationships, and incidence and prevalence rates is combined to estimate annual numbers of people affected and the duration of the condition, including premature death (see chapter 4).

From the policy maker's point of view there are several good reasons for this type of aggregation: prioritising and planning preventive actions in health and environment management, assessing performance of environmental protection system, assessing policy efficacy and efficiency, and identifying high risk populations, for instance in situations of geographical accumulation of multiple environmental exposures (see chapter 2)[15].

6.5.2 Exposure assessment

In few countries highly sophisticated population exposure models have been developed for policy analysis, in which detailed source information at street, grid and city level is combined with population and built environment data in Geographic Information Systems. However in most case such models will not be available. In such cases a more crude approach might be used.

An approach for population exposure to road-traffic noise might be based on strong relations between the size of the city and the population exposure distribution. Roovers et al. proposed a simple modelling approach for the European scale that might be used as an example. Residential areas were classified into five categories of noisiness, based on both city size and regional characteristics, ranging from rural to extremely noisy. Regional characteristics involved factors such as latitude (southern cities tend to be noisier), traffic technology (engines, tires) and densities, urban traffic infrastructure, and meteorological factors (ventilation behaviour) etc[51].

Through analysis of existing data for a series of European cities a crude population exposure distribution for these categories was determined. Successively, these distributions were used to assess the traffic noise exposure for the European population (see table 6-2).

[ii] To arrive at weights for this severity standardisation micro-level HRQL-measurements (e.g. health adjusted life expectancy, HALE) as well as preference-measurements (disability adjusted life-years: DALY's) can be applied. Disability free life expectancy (DFLE) is simply a special case of HALE as a threshold between healthy (1) and disabled or dead is applied (0).

Table 6-2. Traffic noise exposure distribution of EU inhabitant[51].

	noise level inhabitants	<55dB(A) L_{DN} %	mln	55-65 dB(A) L_{DN} %	mln	65-75 dB(A) L_{DN} %	mln	>75 dB(A) L_{DN} %	mln
EU	371.602.000	68	251,3	19	71,2	11	44,4	2	7,7

Data on exposure to aircraft noise are even less available in general. Even in the EU data on population exposure are scant and unreliable. A first approach would be a crude estimate of exposure based on the size of the airport.

For the purpose of this exercise we use population exposure distributions for cumulative noise data modelled in the framework of the Dutch Environmental Balance[2], expressed in different measures, as shown in table 6-3.

Table 6-3. Examples of cumulative noise distributions for the Netherlands.

Exposure dB(A)	L_{24h} % of population	L_{den} % of pop.	L_{night} % of pop.	$L_{Aeq(7\text{-}19)}$ % of population used for cardiovascular end-points assessment	
<40	4	5	43		
41-45	6	7	31		
46-50	14	17	19	51-54	25
51-55	27	29	5	55	5
56-60	30	27	1	56-60	15
61-65	15	11	0	61-65	4
66-70	4	3	0	66-70	1
71-75	1	0	0	71-72	0

Source: EMPARA; Environmental Balance 2004, Environmental Assessment Agency, RIVM.

6.6 Different approaches for disease burden estimation

Given the current quantitative insights in health and well being responses to noise exposure, the strength of the evidence and starting points with respect to definitions of health and health loss, there are basically four manners in which quantitative assessments of noise related disease burden could be achieved. The impact assessment can be based on:

- severe annoyance prevalence,
- sleep disturbances (both as proxies for decreased quality of life),
- noise related population attributive risk for cardiovascular disease
- a noise induced shift of systolic blood pressure distribution or increased hypertension prevalence as risk factors for cardiovascular disease.

The latter two should be considered scenario-wise: what if the indications for an association between noise exposure and cardiovascular disease are true?

6.6.1 Uncertainty assessment

To analyse the uncertainty in the calculations of environmental disease burden we have often applied Monte Carlo techniques (see chapter 4). However in the case of noise health impact assessment uncertainty may just as well be tested by simple well-reasoned worst/best (or what-if) case calculations, using lower, central and high estimates for the model parameters[52].

6.6.2 Annoyance-based impact assessment

For noise annoyance ample quantitative exposure response relationships are fairly well established. Based on a pooled analysis of original datasets from noise-annoyance surveys carried out in Europe, Australia, Japan and North-America, exposure response relationships have been derived for road, rail and air traffic noise[7]. These curves have been derived from probably the most elaborate database currently available and can be used to predict the number of annoyed people in an exposed population (in general three degrees of annoyance are defined: 'little', 'moderate' and 'severely' or 'highly annoyed'). They are recommended for use in the EU Directive on the Assessment and Management of Environmental Noise[53]. It has to be noted that there is still debate on the procedures used for meta-analysis. Furthermore the differences between the three noise sources may be due to methodological differences in the original studies (e.g. poor exposure assessment, differences in adequacy noise insulation[54,55].

The fraction of severely annoyed people for sound levels in L_{dn} en L_{den} can be estimated using the analyses of Miedema and Oudshoorn (see Box 1.).

Box 1. Exposure response relations for severe noise annoyance (HA) for L_{DN} en L_{DEN}

Aircraft: $\%HA = -1.395*10^{-4} (DNL-42)^3 + 4.081*10^{-2} (DNL-42)^2 + 0.342 (DNL-42)$;
Road traffic: $\%HA = 9.994*10^{-4} (DNL-42)^3 - 1.523*10^{-2} (DNL-42)^2 + 0.538 (DNL-42)$;
Railways: $\%HA = 7.158*10^{-4} (DNL-42)^3 - 7.774*10^{-3} (DNL-42)^2 + 0.163 (DNL-42)$.

Aircraft: $\%HA = -9.199*10^{-5} (DENL-42)^3 + 3.932*10^{-2} (DENL-42)^2 + 0.2939 (DENL-42)$;
Road traffic: $\%HA = 9.868*10^{-4} (DENL-42)^3 - 1.436*10^{-2} (DENL-42)^2 + 0.5118 (DENL-42)$;
Railways $\%HA = 7.239*10^{-4} (DENL-42)^3 - 7.851*10^{-3} (DENL-42)^2 + 0.1695 (DENL-42)$.

If the number of severely annoyed people is estimated, the next step would be to assess the disease burden. Within the concept of health adjusted life-years this can be done by simply multiplying the number of people reporting to be severely annoyed with a severity weight factor discounting for the disease state "severely annoyed", thus yielding an estimate of the annual 'health' loss (duration 1 year). In the framework of an environmental burden of disease assessment a panel of health professionals defined a severity weight for severe noise annoyance in comparison with other diseases or limitations in functioning (see Box 2)[56,52]. However, it is noted here that not everyone would agree that severe annoyance should be regarded as health loss in the strict sense, but at the same time it could lead to various symptoms or a limitation in functioning regarding daily tasks or activities.

Box 2. A severity weight factors for noise exposure

'Severe annoyance'
In an environmental disease burden study in the Netherlands "severely annoyed" was selected as a health significant endpoint and evaluated following the protocol of the Dutch Burden of Disease Study[37]. A definition of severe annoyance was included in a severity weigh exercise to arrive at discount factors. The following definition was evaluated by a group of environmental physicians, epidemiologists and public health professionals n=35). "*Regularly (and partly inescapably) persons are disturbed during daily activities, such as having conversation, listening to the radio, watching television, reading a book or a magazine. Furthermore persons may have feelings of resentment, displeasure, discomfort, dissatisfaction or offence, occurring when noise interferes with their thoughts, feelings or daily activities*". This definition was presented together with a Euroqol 5D+ description to assist in structuring the evaluation (see chapter 2 and 4).

No problems walking about (mobility)
No problem with washing or dressing oneself
No problems with usual activities (work, study, housework, family of leisure activities)
No pain or discomfort
Not (95%) or mildly (5%) anxious or depressed
No (95%) to some (5%) cognitive impairment (e.g. problems with memory, concentration, disorganisation, IQ-level).

The respondents evaluated the health states by means of interpolation using indicator conditions ranked on a disability scale (see figure 5.4). The severity factor derived in this exercise was used to discount for the health state "sever annoyance". This procedure yielded a severity weight factor of 0.12 (standard deviation: 0.16, median 0.07).

Severe sleep disturbance
The following definition was evaluated by a group of environmental physicians, epidemiologists and public health professionals (35). *"Regularly persons are disturbed in their sleep, manifest as an increase of awakenings and/or shifts in sleep stages during the night. This affects the perceived quality of sleep and their mood the next day. During the day they might experience sleepiness, slight fatigue, and concentration difficulties".* This definition was presented together with the following Euroqol 5D+ description to help the respondents structure their evaluation.

No problems walking about (mobility)
No problem with washing or dressing oneself
No problems with usual activities (work, study, housework, family of leisure activities)
No pain or discomfort
Not anxious or depressed
No (90%) to some (10%) cognitive impairment (e.g. problems with memory, concentration, disorganisation, IQ-level).

The respondents evaluated the health states by means of interpolation using indicator conditions ranked on a disability scale (see figure 5.4). The severity factor derived in this exercise was used to discount for the health state "highly disturbed". This procedure yielded a severity factor of 0,1 (standard deviation: 0.1, median: 0.08)[56].

Calculations of the burden of noise annoyance are fairly simple applying the above mentioned exposure response relations on a given population noise exposure distribution (see table 6-4)[58]. Based on the exposure-response curves from box 1 it can be calculated that the current noise exposure distribution in the Netherlands produces around 64,000 severely annoyed persons per million (around 6.4% of the population). Applying a severity weight of 0.07 (median) this would mean an annual disease burden of around 4,500 disability (severe annoyance) adjusted life years.

In this example we used the exposure response curve for the percentage of *highly* annoyed persons due to road traffic[iii,iv]. Doing the calculations with the range of reported severity weights would be another way to investigate the uncertainty of this type of calculations, clearly showing a high sensitivity of the calculations to attributed severity weights (ranging from 0.00 to 0.33 among 34 professional respondents). In a preliminary Swiss study among 41 physicians disability weights of 0.033 and 0.055 were assigned to communication and sleep disturbance respectively[59]; in an earlier disease burden study we applied 0.005 (0.00-0.02), being the least sever category of the first GBD-study (see chapter 4)[60,2].

[iii] In case no specific noise exposure distributions are available one could consider to use curves for railroad noise (lowest annoyance potency) and aircraft noise (more potent) to calculate a range between lower and higher estimates of the fraction severely annoyed: here 2.5-10% of the population.

[iv] These estimation are in reasonable accordance with the results of a recent survey (3% for private cars – 10% for truck traffic); however 19% of the respondents reported themselves to be severely annoyed by the noise of mopeds[58].

Table 6-4. Example of spreadsheet calculation of number of severely annoyed people per million, based on exposure distribution in the Netherlands (cut-off 'severe': 28% most annoyed, Miedema et al., 2002)

exposure category dB(A)	average L_{DEN} dB(A)	exposure distribution: % of population	% severely annoyed	number/ 1000.000
<40	40	5	0,0	0
41-45	43	7	0,5	364
45-50	48	17	2,7	4,535
51-55	53	28	5,4	14,983
56-60	58	26	21,3	22,922
61-65	63	11	13,8	15,098
66-70	68	2	21,3	5,279
>71	73	0,3	31,8	1,011
total		**100**		**64,193**

6.6.3 Sleep disturbance based impact assessment

In a similar way the fraction of people experiencing severe sleep disturbance can be assessed. However, there is much less consistency in and consensus on appropriate exposure response functions. Based on an analysis of original data from 15 datasets (12 field studies, 12000 observations), relationships have been proposed (box 2) that give the percentage highly sleep disturbed *(%HSD)*, sleep disturbed *(%SD)*, and (at least) a little sleep disturbed *(%LSD)* by road traffic and railway noise as a function of the outdoor L_{night} at the most exposed façade. Sleep disturbance questions vary a lot between surveys, in wording and in the number of response categories.

Box 3. Exposure response relations for sleep disturbance for L_{night}

Road traffic:	%HSD = 20.8 − 1.05Lnight + 0.01486Lnight2
	%SD = 13.8 − 0.85Lnight + 0.01670Lnight2
	%LSD = -8.4 + 0.16Lnight + 0.01081Lnight2,
Railways:	%HSD = 11.3 − 0.55Lnight + 0.00759Lnight2
	%SD = 12.5 − 0.66Lnight + 0.01121Lnight2
	%LSD = 4.7 − 0.31Lnight + 0.01125Lnight2,

In order to obtain comparable disturbance measures the sets in the selected studies were translated into a scale from 0 to 100. Cut-off points to assess the percentage of highly sleep-disturbed persons were used analogue to the definitions of the percentage (highly) annoyed persons. No relationships for aircraft noise were proposed because of the large variance in results.

Box 3 presents the curves Miedema et al. proposed for the percentage people reporting to be highly sleep disturbed *(%HSD)*, sleep disturbed *(%SD)*, and (at least) a little sleep disturbed *(%LSD)* as a function of the outdoor L_{night} at the most exposed façade for the source concerned[34].

The exposure-response relationships presented in box 3 can be used to calculate the prevalence of highly disturbed in a given population. To estimate the burden of disease from sleep disturbance, a severity weight has to be defined (see box 2).

Calculation of sleep disturbance burden is similar to the calculation of annoyance burden, applying the exposure response relations on a given population noise exposure distribution (L_{night}-distribution). Using the exposure response curve for the percentage of *highly* sleep disturbed persons due to road traffic (cut-off: ≥35 dB(A)) a fraction of 0.7-1.4% can be calculated to be highly sleep disturbed as a result of cumulative community noise exposure. Using a severity factor of 0.8 (median) this number yields an

annual burden of 550 - 1,100 disability ('severe sleep disturbance') adjusted life-years'/million. Again, these calculations are a highly sensitivity to attributed severity weights (ranging from 0,0 to 0.40 among 35 professional respondents).

6.7 Impact assessment based on cardiovascular endpoints

6.7.1 Epidemiological data

Meta-analysis of the epidemiological research on the effects of community noise on cardiovascular health yields few estimates for the calculation of noise attributable fractions of cardiovascular disease (see table 6-5). They are defined as 'day-time' levels $L_{Aeq,\ 7\text{-}19h}$, $L_{Aeq,\ 6\text{-}22h}$, $L_{Aeq,8h}$ in dB(A) for aircraft, road traffic and occupational exposure, respectively. For aircraft noise the analysis produced a statistically significant RR-estimate for hypertension (defined as systolic BP > 160 mmHg, diastolic BP > 95 mmHg), and a borderline significant estimate for the use of cardiovascular drugs. For road traffic the results of three studies are contradictory (see table 6-5). The RR-estimate for IHD prevalence is clearly statistical significant (but based on two cross-sectional studies) and the RR-estimate for past myocardial infarction is borderline significant. The results of several recent studies are consistent with a small effect on hypertension (see above)[35,36,37,38,39]. We emphasise that the following health impact assessments should be regarded as a scenario-like exercise: *what* is the public health significance of current community noise exposures *if* the cardiovascular connection were to be true?

Table 6-5. Summary estimates for the association between noise exposure, hypertension, and ischemic heart diseases, adjusted for sex and age,. expressed as $RR_{5\ dB(A)}$ (increase per 5 dB(A))[10].

Noise exposure[a]	Outcome	$RR_{5\ dB(A)}$	95% CI[d]	number of estimates[10]	Measurement range (dB(A))
Occupation	Systolic blood pressure mmHg/5dB(A)	0.51	0.01 -1.00	14	55 – 116
Occupation	Hypertension[b]	1.14	1.01 – 1.29 *	9	55 – 116
Road traffic	Hypertension	0.95	0.84 – 1.08	2	<55 – 80
	Use of antihypertensives	0.96	0.76 – 1.22	2	>50 – 73
	Consultation of GP/specialist	0.91	0.73 – 1.12	1	55 – 70
	Angina Pectoris	0.99	0.84 – 1.16	2	51 – 70
	Myocardial Infarction[c]	1.03	0.99 – 1.09	3	51 – 80
	IHD-total[c]	1.09	1.05 – 1.13 *	2	51 – 70
Air traffic	Hypertension	1.26	1.14 – 1.39 *	1	55 – 72
	Use of anti-hypertensives	0.99	0.87 – 1.14	1	55 – 72
	Consultation of GP/specialist	1.10	0.95 – 1.27	2	55 – 77
	Use of cardiovascular drugs	1.05	0.99 – 1.11	2	38 – 77
	Angina Pectoris	1.03	0.90 – 1.18	1	55 – 72

[a] The noise exposure measures differed between the noise exposure sources: occupational noise exposure expressed in $L_{Aeq,\ 8h}$, in dB(A), road traffic noise exposure expressed in $L_{Aeq,\ 6\text{-}22h}$, in dB(A) and air traffic noise exposure expressed in $L_{Aeq,\ 7\text{-}19h}$, in dB(A).
[b] Adjusted for age, sex and worktype.
[c] Ischaemic heart disease (ICD-9:410-414), only prevalence estimates.
[d] CI = Confidence Interval * Significant, p<0.05.

6.7.2 Calculation of cardiovascular disease attributable to noise

For estimating the burden of cardiovascular disease due to noise we make no qualitative distinction between aircraft and road traffic noise, although annoyance studies show they may be processed in different ways. We simply use the combined noise levels from the Dutch national noise calculation system (EMPARA) in $L_{Aeq,\ 7\text{-}19\ dB(A)}$ as exposure measure (which comprises mainly road traffic) and is in fair accordance with the other two definitions.

To estimate the number of people affected we calculated population attributable risks (PAR's) by combining population exposure distributions (see table 6-3) with quantitative exposure-response information, applying the following equations:

$$RR_i = e^{\frac{(L_i - L_{cut-off})}{5} * \beta}$$

$$PAR = \sum \frac{p_i * (RR_i - 1)}{p_i * (RR_i - 1) + 1}$$

in which RR_i = relative risk in exposure class i and p_i = exposure probability in class i, L_i = exposure level in class i, expressed in dB(A), $L_{cut\text{-}off}$ = cut-off (e.g. 54 dB(A)), and β is the risk function estimate from the meta-analysis (see table 6-5).

Subsequently we estimated the noise attributable prevalence of cardiovascular endpoints by multiplying the PAR with annual prevalence data, obtained from Dutch health statistics[61].

Table 6-6. Estimation of community noise attributable cardiovascular endpoints for the Dutch situation, per 1000,000 inhabitants per year; exposures ≥ 55 dB(A), exposure distribution in $L_{Aeq\ (7\text{-}19\ h)}$.

Cardiovascular endpoint (P_0)	RR-estimate (95%-CI) from meta-anlysis[10]	noise attributable number / 1000,000	results using lower and upper RR-estimate	
Hypertension prevalence (0.196)	air traffic: 1.26 (1.14-1.39)	11,500	6,000	18,000
Hypertension prevalence (0.196)	occupation: 1.14 (1.01-1.29)	6,200	440	13,000
Ischaemic Heart Disease prevalence (0.035)	road traffic: 1.09 (1.05-1.13)	700	390	1,020
Past myocardial infarct prevalence (0.017)	road traffic: 1.03 (0.99-1.09)	110	0	340

In table 6-6 results are presented of simple calculations of the noise exposure attributable numbers of attributable prevalent cases of hypertension, ischaemic heart disease and past myocardial infarction per 1000,000 inhabitants, assuming that the exposed population is similar to the total population. Attributable fractions were calculated based on the $L_{Aeq\ (7\text{-}19h)}$-exposure distribution for the Netherlands and RR-estimates from air traffic, road traffic and occupational studies, respectively (cut-off at ≥55 dB(A), including around 25% of the Dutch population, see table 6-3).

These calculations are highly sensitive to the choice of cut-off point. Applying a cut-off point of 51 dB(A) instead of 55 dB(A) increases the number of attributable prevalent IHD or past myocardial infarction cases with a factor of 2.6, especially due to the substantial increase of the fraction of the population exposed to 'effective' levels (see table 6-3, 25% in class 51-54 dB(A)).

6.7.3 Impact assessment of cardiovascular disease burden

6.7.3.1 Ischaemic heart disease burden

The most straightforward approach to estimate disease burden attributable to community noise is calculating the disability adjusted years (YLD) associated with noise attributable *ischaemic heart disease* prevalence (see table 6-6), over one year, using the aggregate severity weight from the Dutch Burden of Disease study (0.288)[62]. This yields an annual number of 200 DALYs (110-295) per million. If we assume these cases will eventually die, having the same loss of life expectancy as general cases, we can also approach noise associated disease burden with the attributable fraction of coronary heart disease burden per million in the Netherlands (around 22,000 DALYs/year): 445 DALYs (245-640)/year/million.

6.7.3.2 Mortality due to noise attributive hypertension

As discussed in section 6.3.2.1 it is assumed that noise exposure has an effect along the overall process of cardiovascular disease genesis. Positive associations are found for a number of manifestations of cardiovascular disease often in individual studies: elevated blood pressure, hypertension, cardiovascular medication use (including hypertensives), consultation of the general practitioner or cardiologist, prevalence of Angina pectoris and/or ischeamic heart disease[10]. Following the reasoning behind the conceptual model (figure 6-1) we assume that the risk elevations associated with noise exposure for these different endpoints are a fair indication of a small contribution to total disease prevalence.

From this perspective, besides calculating noise attributable IHD, one could also approach diseased burden by calculating the noise population attributable fraction of annual hypertension population attributable mortality.

$$PAR_{mortality/noise} = PAR_{mortality/hypertension} * PAR_{hypertension/noise}$$

In this exercise we applied a RR-estimate of 1,4 for total mortality risk in a population with hypertension relative to a population without[63]. Given the current noise exposure distribution for the Netherlands an attributable fraction of total mortality can be estimated: 0.0059, representing 38 cases per million inhabitants. Assuming a loss of life expectancy of around ten years associated with general cardiovascular cases this yields an annual loss of 380 (200-580) DALYs (YLLs) per million. Again these calculation are sensitive to the choice of cut-off point.

6.7.3.3 Noise attributable increase in systolic blood pressure

Another way of crude estimation is applying the adjusted estimate for noise attributable increase of usual systolic blood pressure derived from a slightly more solid epidemiological database: a meta-analysis of 14 occupational studies, yielding an estimate for a noise induced increase of (usual) systolic blood pressure: 0.01-1.0 mmHg for every 5dB(A) increase in noise exposure (see table 6-5). Of course in occupational studies risk estimates are based on 8-hour measures ($L_{Aeq, 8h}$–exposure measure). However this distribution is fairly similar to the $L_{Aeq\ (7\text{-}19\ h)}$-distribution we have used so far. This exposure-response function allows the calculation of a population attributive fraction for total annual disease burden based on a noise attributable shift in (usual) systolic blood pressure.

Within each relevant exposure category (≥55 dB(A)) this shift results in an elevated relative risks for ischaemic heart disease and stroke. Age and gender specific RR-functions for ischaemic heart disease and stroke were taken from the Asia Pacific Cohort Study (a meta-analysis comprising data of over 425,000 individuals and over 3 million observation years)[64,65,66]. Similar to the global burden of disease study that

was done for lead exposure, we applied a relative risk of 1.057 per mmHg increase in systolic blood pressure (SBP) for ischaemic heart disease and 1.066/mmHg for stroke (adjusted Dutch population characteristics). Using 0.51 (0.1-1.0) mmHg as a crude estimate of population SBP-shift per 5 dB(A) noise attributable disease burden in the Dutch population for ischaemic heart disease is 140 (3-270) and for stroke[v] 88 (2-169) DALYs. However, no clear indications for an association between noise exposure and stroke have been reported so far.

Table 6-7. Summary of results of crude estimations of annual noise attributable disease burden: DALYs/1000,000 inhabitants

Type of estimate	central (DALYs/million)	lower (DALYs/million)	upper (DALYs/million)
'annoyance adjusted life years'		640 *	4,500
'sleep disturbance adjusted life-years'		60 *	1,100
IHD based on prevalence years (YLD)	215	125	305
IHD based (YLD+YLL)	390	225	550
Noise attributable hypertension mortality	380	200	580
IHD and CVA based on BP-shift (DALY)	230	5	450

* applying 0.01 as disability weight factor, being the least severe category from the first Global Burden of Disease Study

The results of the proposed exercises of tentative disease burden assessment are presented in table 6-7. Noise attributable loss of 'disability' adjusted life-years is potentially largest for the social psychological endpoints 'severe annoyance' and 'severe sleep disturbance'. Estimates for noise attributable cardiovascular disease are all in the same order of magnitude (200-600 DALYs/year/million), somewhere between 0.1 and 0.2% of total disease burden.

6.8 Discussion

6.8.1 In case of a causal association: a small, but significant disease burden

Assuming the indications for an association between community noise exposure and cardiovascular disease are true, a small but significant disease burden can be attributed to current exposure levels in the Netherlands (no more than 2% of total ischaemic heart disease burden). Given the definition of the exposure distribution, obviously this assessment does not include a possible effect of night-time noise. There are some indications that night-time noise is more effective with respect to cardiovascular endpoints, such as hypertension[37]. At the same time these explorative calculations show that the social-psychological impacts of community noise exposure may constitute a substantially greater burden to health and well-being.

These explorative health impact assessment exercises are to some extent acceptable if we take into account that the epidemiological database on a range of observed end-points is fairly consistent with known cardiovascular disease progression. Small transient, stress related haemodynamic responses to noise exposure may in the end result in a slight increase of cardiovascular risk at level of populations[10,41]. The proposed approaches at least provide some sense of the order of magnitude of possible noise attributable health loss, in case the associations are true: clearly a 'what if scenario approach. This may help giving community noise exposure its proper place on the environmental agenda. Furthermore, it cannot be ruled out that the next well-performed cohort study may reveal a greater effect of community noise on cardiovascular disease incidence.

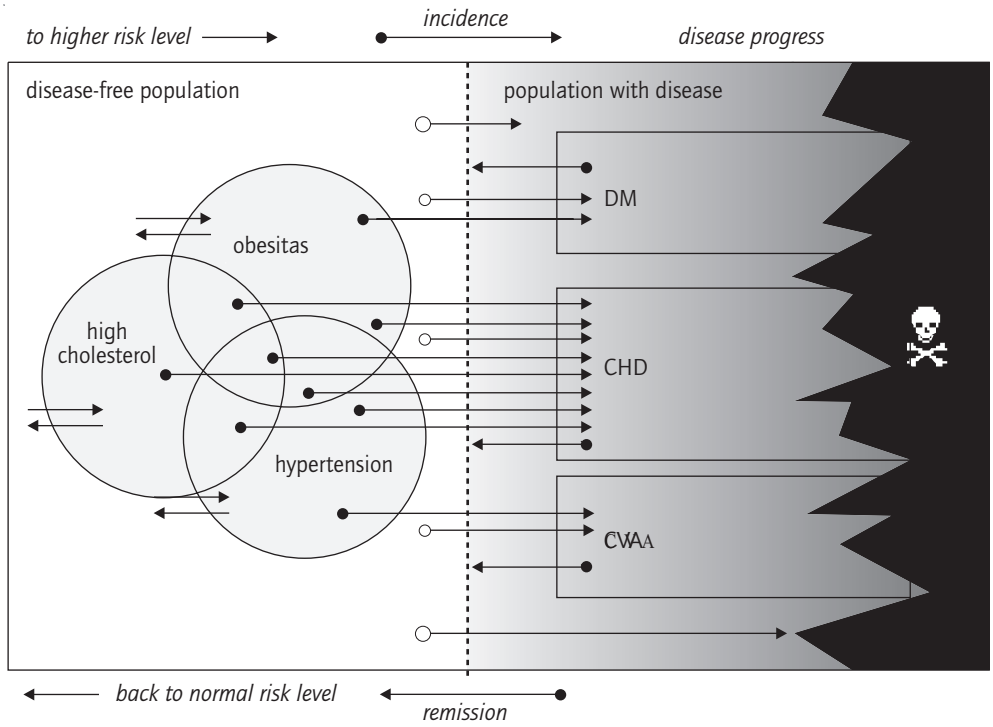

Figure 6-2. Diagram representing disease burden calculations with respect to noise induced blood pressure shift. A shift in blood pressure on a population level would result in an increased number of persons with hypertension, increasing the incidence and thus prevalence of patients with coronary heart (CHD) disease or stroke (CVA), increasing disease specific mortality (and loss of life years).

6.8.2 Different sources of noise

Due to lack of good data in the examples of health impact calculations we could not properly take differences between noise from different environmental sources into account. Meta-analysis of studies of noise related annoyance suggests there is a difference in the way different sources of noise are 'processed' by exposed individuals. At the same level of exposure aircraft noise appears to provoke more annoyance than road traffic noise; railway noise causes the least effect. And of course their phenomenology can be quite different. On the other hand, there are some indications that differences in exposure response relations for different transport sources may be due to differences in methodology of the original studies, such as exposure assessment (e.g. taking noise insulation of dwellings into account)[54,55].

6.8.3 The severity of annoyance

From comfortable hindsight one might question the appropriateness of the definition of 'severe annoyance' (box 2) applied in the weight-attributing exercise. This definition that we took from Health Council reports on noise[25,67], may be slightly overstating the condition people report in the daily practice of large-scale noise-surveys: annoyed by noise on a scale from zero to ten. Of course a fraction of the population, in particular typically noise-sensitive people, will experience to be *'regularly (and partly inescapably) disturbed during daily activities, such as having conversation, listening to the radio, watching television, reading a book or a magazine* and thus *have feelings of resentment, displeasure, discomfort, dissatisfaction or offence, occurring when noise interferes with their thoughts, feelings or daily activities'*. However it is hard

to conceive people being disturbed in conversation, listening to the radio due to exposures as low as of 42 dB(A) at the facade of their homes, resulting in substantially lower levels inside. Other, non-acoustical factors may play a significant role as well in the reported annoyance.

Moreover we assumed people to be in a state of 'severe annoyance' throughout the whole year. It is likely that the reported severe annoyance may concern much more restricted periods, such as 'sunny' weekends in which people tend to pass time outside the house or go out for a walk and are thus much more bothered by community noise.

It is reasonable to assume that the application of the severity weigh factors from the official weighing exercises tends to produce relatively high noise attributable 'disease burden' associated with annoyance. The same applies to a lesser extend for sleep disturbance. Therefore, it might be a good suggestion to implement some form of severity weight exercise into noise annoyance surveys to produce more realistic estimates for disability weight factors.

6.8.4 Uncertainties

In these types of integrated assessments many substantial uncertainties are accumulated. This may pertain to available data for modelling (criterion validity) as well as to the impact assessment models themselves (construct validity). With respect to criterion validity 'what-if'- or Monte Carlo analysis is a simple and adequate way of providing probability distributions for the outcome variables. However, in this case many of the uncertainties may concern the construct validity, the validity of the exposure-response relations themselves. This means much effort will have to be put into conveying the right message to policy makers and the public. As is often the case, the association between noise and 'annoyance' has a high criterion and construct validity, but the public health significance (content validity) is more controversial. The other way round, the clinical significance of cardiovascular disease is obvious, but a causal relation with noise exposure is not yet fully supported by data or scientific consensus.

6.8.5 An impossible choice of endpoints?

So, which of the proposed endpoints should be applied in comparative risk assessment to support environmental and spatial policy? *Numbers* per million for a conceptually consistent range of social-psychological and cardiovascular endpoints (table 6-5 and 6-6) clearly indicate that public health may be at stake, but is less appropriate in a framework of comparative risk assessment. The calculations of attributable burden of cardiovascular disease *(DALYs)* indicate a small, but clinically significant health loss of less than 2% of total ischaemic heart disease burden and less than 0.1% of total national disease burden[68]. At the same time these values are the result of a 'what if the epidemiological data are valid' scenario based on the best available data. Besides that, we have calculated a burden expressed in 'severe annoyance' adjusted life-years, which –when allowed in the same league as pure clinical health endpoints-, may amount to 2% of total disease burden, especially depending on attributed severity weights.

In chapter 3 we argued in favour of stakeholder involvement in risk management procedures when risk analysis is highly uncertain and fundamentally normative choices have to be made. The community noise problem may just be a good example for such an approach. There is still much uncertainty, but awaiting better science, we could facilitate the decision process by clearly framing the problem and calculating the results of different options. The various exercises proposed here, could have a meaningful place in such a procedure.

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7 Environment and Health in the OECD region: "losing health is losing money"[i]

Augustinus E.M. de Hollander, Johan M. Melse

This chapter describes a study performed within the framework of the Environmental Outlook Environmental Outlook of the OECD, in which we aim to provide some quantitative information on the public health impact of environmental exposures. We use Global Burden of Disease estimates provided by WHO to enable comparison between various (environmental) health risks, consequently the setting of priorities, and evaluation of the efficiency of different policy options. In order to facilitate comparison to other assets valued by society, we also provided some monetary estimates of the health loss involved.

[i] Based on *JM Melse and AEM de Hollander. Environmental health within the OECD-region: lost health lost money. Background document to the OECD Environmental Outlook. RIVM-report 402101001. Contribution to Chapter 21. Human Health and the Environment. OECD Environmental Outlook. Paris: Organisation for Economic Co-operation and Development, 2001. Melse JM, Hollander AEM de, Hilderink H, Martens P. Human health and the environment. In: Netherlands Human Dimension Program Committee. Kaleidoscopic view on scientific global change research in the Netherlands. Amsterdam: Royal Society of Arts and Sciences, 2001.*

7.1 Introduction

7.1.1 Health and economics

Traditionally public health concerns have been an important motive for the political priority given to environmental issues. Nowadays, the threat of global warming, decreasing biodiversity and the urgency for sustainable growth are other good reasons for political engagement, even though the immediate effects of these developments on health are often less visible. In the recent OECD Environmental Outlook[1] ample attention is given to the human health dimension of environmental issues. This chapter briefly describes the public health assessments and subsequent monetarisation we produced in the framework of that report[2].

The relation between environmental conditions and human health has been established as early as the seventeenth century. In 1690 Sir William Petty demonstrated a considerable influence of sanitary conditions on human mortality in his investigations on 'Political Arithmeticks'[3]. The removal of the Broad Street pump handle in 1854 by John Snow to stop an outbreak of cholera -a story of nearly mythical proportions amongst epidemiologists[4]-, marks the beginning of current public health practices in which environmental factors are regarded as major determinants for the health status of a population (see chapter 1).

Within an economic perspective, health can be viewed as a return from investments in environmental and human capital, but also as capital in itself, returning happiness and healthy time to be used for production, relationships or recreation[5]. Health as capital goes very well with recent thinking on sustainable development, especially defined in the original manner as 'leaving future generations at least as many opportunities and choices as we have had ourselves'[6]. Developing social and human capital, rather than primarily focusing on the accumulation of material resources, will contribute to sustainability of modern societies. Expenditures on environmental interventions or health care are therefore not only costs, but also investments with valuable and worthwhile returns, both in health and money. Moreover, the health care sector has become an important economic sector (within OECD 4-14% of GDP in 1997, compared to 2-5% in 1960), providing income and meaningful work to many (up to 10% of total employment)[7,8].

7.1.2 Transitions in environmental risks and health

As we have seen in chapter 1, patterns of environmental risks and their effect upon public health show considerable changes over time and place, and can be considered as the outcome of historical economic developments. Different stages in the socio-economic transition of societies from traditional to contemporary also show differences in the nature and size of environmental challenges and corresponding health effects. Table 7-10 sketches the economic transition from agricultural societies to the contemporary globalising economy, in connection with the changes in both environmental issues and associated health impacts. It also identifies the sectors producing the environmental health threats, and potential intervention targets at the same time.

In agricultural, pre-industrial societies, environmental problems predominantly involve access to clean water, public sanitation, appropriate housing, poverty and protection against natural disasters. Public health issues are predominantly related to infectious diseases, maternal and perinatal conditions and nutritional deficiencies. Although economic progress has provided the means to reduce these risks to acceptable minimum levels in much of the OECD region, it also evokes new risks, mostly linked to modern large-scale rationalised production. Industrial and agricultural emissions of chemicals in water, air and food

have been associated with respiratory diseases and various types of cancer, while (the possibilities of) large industrial accidents may cause societal disruption. Within the established market economies of late-capitalist, 'post-modern' societies, emphasis is now shifting from industrial production to escalating levels of consumption of goods and services from all over the world. Knowledge and tourism crossing all geographical and cultural divisions[9] are increasingly important economic factors[10,11]. Although the environmental risks of much of modern industry are more or less controlled, greatly increased transportation and energy use produce large-scale transboundary air pollution (particulate matter, ozone). This results in increasing noise pollution, reducing liveability in many urban areas. Furthermore there are indications that the 'global village' may enhance the risks of pandemics of new infectious diseases (SARS, 'chicken flu'). Of course substantial environmental disease burden is also manifest in regions outside of the OECD, where much of the production of OECD-destined goods takes place.

For the (nearby) future, the rapid growth of the world population, the ageing of western populations and their effect upon the use of resources and energy, and the globalisation of Western high-consumption economies is expected to increasingly disrupt the biosphere's life supporting systems, and to reshape human health risks. Adverse changes of distribution patterns of new and known infectious diseases may be one of the symptoms[12]. Within this context the most recent Environmental Outlook of the Organisation for Economic Co-operation and Development identified environmental drivers and pressures and the resulting environmental impacts using an economy-based vision of developments to 2020, concluding that the most critical environmental concerns facing OECD countries are the unsustainable use of renewable natural resources, the degradation of ecosystems and the disruption of the environmental systems that support human life[1].

7.1.3 Loss of health is loss of money

In this chapter we briefly describe a study we performed within the framework of the OECD Environmental Outlook. It aims at providing quantitative information on the public health impact of environmental exposures. Application of Global Burden of Disease Estimates in terms of DALYs as an aggregate health impact indicator enables comparison between various (environmental) health risks and consequently the setting of priorities ('how bad is this exposure?'), and evaluation of the efficiency of different policy options ('how much health do we gain by implementing this policy compared to other options?'). In order to facilitate comparison with other valued societal assets, the study also provides some crude monetary estimates of the health loss involved. An outcome, or rather a disease-based approach is applied. Using the WHO database on worldwide disease burden as a starting point plausible attributable fraction are established for relevant environmental exposures[13].

7.2 Concepts and methods

7.2.1 What is environmental health?

In chapters 1 and 2 we extensively discussed the complex concept of health. As with health, the definition of the 'environment' or 'environmental' can be approached from different perspectives (ecological, medical, social, physicochemical or economical). Based on a classical nature-nurture dichotomy, all factors that are not genetic can be regarded as environmental[14], or as Einstein put it: 'the environment is everything that isn't me'. However on larger time-scales, taking evolutionary mechanisms into account, even current genetic conditions can be seen as the result of past gene-environment interactions. This would imply all diseases are environmental.

Contemporary risk management emphasises another distinction, i.e. the extent to which exposure is voluntarily and subject to personal choice and action. Behavioural and life-style factors are consequently not viewed as environmental, although a definite influence of the 'social' environment on life-style and health may cast doubt on the 'voluntariness' of such factors (see chapters 1 & 2).

The definition of *environmental health* of the World Health Organisation echoes the broad scope of its public health tradition: 'environmental health comprises those aspects of human health, including quality of life, that are determined by physical, chemical, biological, social, and psychosocial factors in the environment. It also refers to the theory and practice of assessing, correcting, controlling, and preventing those factors in the environment that can potentially affect adversely the health of present and future generations.'[15]. In practice, these factors have been classified into 'targets': air pollution, water pollution (both drinking and wastewater), hazardous waste, human ecology and settlements (incl. indoor air), food safety, monitoring, occupational health and safety[16].

As outlined in chapter 2 we will focus environmental health assessment primarily on outcomes that are significant to mortality, morbidity and health-related quality of life. Obviously this includes all clear intermediate risk indicators, such as lung function deficits, aggravation of asthma, or sleeping problems, as well as 'derivative' indicators like the use of medical services or self-medication. For the purpose of this exercise 'environmental' refers to physical, chemical and biological exposures that are the result of human activity, excluding occupational health and safety, falls, drowning and war (being of disputable 'environmentalness'). We also exclude important life-style determinants of health such as smoking behaviour and dietary patterns. We did however take a small environmental fraction of traffic accidents into account[14].

7.2.2 How much ill health can be attributed to the environment: 'attributable risk'

In general, the extent to which a certain phenomenon causes diseases can be characterised by the 'attributable risk', indicating the percentage of a particular disease category that would be eliminated the phenomenon's influence would be reduced to its lowest value[14,17]. Disease genesis is a multi-causal process, involving multiple risk factors of which the environment is but one. As discussed in chapter 2 the role of (environmental) risk factors can often be envisaged as an iceberg accumulating ice at the bottom, forcing an upward movement of pre-existing physiological or psychological aberrations just within or across the borders of biological variations towards more severe symptoms and manifestations of 'disease'. Mortality is at the tip (see figure 2-3). Only the more severe responses end up in a consulting room, in hospitals and thus in official health statistics (above the surface), while responses beneath the surface are only seen in special surveys. Moreover, apart from rare exceptions, responses are not specific for environmental exposures but often dominated by life-style, genetic and socio-demographic factors. From this perspective it is understandable that sum of attributable risk percentages for a number of risk factors may well add up to more than 100%[14]. For example, attributing large fractions of acute respiratory infections to air pollution and poor housing is not incompatible with attributing large fractions to malnutrition or lack of immuno-competence as well. Risk factors often interact: reducing one risk factor will influence the attributable risk of others[18].

Whether a risk factor is defined as being environmental also depends on the interdependent choices of time period and baseline. As discussed above, when a sufficiently long period is taken, all diseases can be regarded as environmental, while taking a shorter time horizon would exclude long term environmental health threats and effects, such as climate change effects and skin cancer due to ozone layer reduction. Because humans have never lived without (environmental) risks, it can be argued that a certain level of risk

and health effects is unavoidable, tolerable or even acceptable. A baseline of a zero exposure level might therefore be suitable for synthetic chemicals, but not for airborne particles, or ionising radiation. A baseline can also be seen as a 'counterfactual' or alternative scenario, to which the current level of risk factor is compared. Murray and Lopez (1999) distinguished three counterfactual scenarios[19]: the *theoretical minimum*, representing a scenario where the exposure distribution is associated with the least population risk (e.g. no tobacco use), and the *feasible minimum* risk, corresponding to the scenario with the minimal population risk that has been achieved elsewhere in the world with current technologies and in optimal conditions. A *plausible minimum* risk scenario is somewhere between the two former ones. Given the presence of tobacco or alcohol addiction, a non-smoking or non-drinking society is hard to imagine, just like a society with full employment or without some obesity. On the other hand one could come up with well-argued scenarios that are more ambitious than the feasible minimum.

7.2.3 An aggregate health impact indicator

In past decades several integrative indicators have been constructed to aggregate health losses on the level of populations[20]. Within the World Health Organization - World Bank Global Burden of Disease project the disability-adjusted life-year (DALY) was developed to assess the global disease burden and consequently the health policy priorities in different regions in the world[21]. This health impact measure combines years of life lost and years lived with disability that are standardised by means of severity weights; it thus measures health using time as the metric. The basic calculations are as follows:

the annual burden of disease is equal to mortality plus morbidity burdens, measured as

DALY = Years of Life Lost (YLL) + Years Lived with Disability (YLD), in which:

YLL = annual Number of deceased (mortality) * remaining age group Life Expectancy, and

YLD = annual Number of diseased (new cases) * Severity weight * Duration of disease.

To estimate the number of people affected, two basic approaches may be used: the exposure-based and the outcome-based approach[13]. The first assesses the exposure in the population and combines exposure distribution with dose-response relations and incidence data, resulting in the number of people suffering an adverse health outcome. The second collects data on relevant disease burden on the population level and then determines the fraction can be attributed to certain environmental risk factors. Obviously the two methods require different sets of data. The duration of disease and the 'duration' of mortality can often be derived from clinical data, disease-specific mortality figures and life tables.

Of course, there are many questions about concepts and methods underlying aggregate indicators such as the DALY. In particular procedures and values for the weights for severity, age weighting and discounting for future health loss have evoked fierce discussion. The use of composite health outcome measures implies several normative choices, such as the reference life table to be used, the valuation procedures, etc. These choices have been extensively discussed in the literature[22,23,24,25,26]. However, key advantages of the DALY are its aggregate nature, combining quantity and quality of life, as well as its transparency and explicit appreciation of many of its assumptions, allowing for open discussion and the testing of different preferences. Well-established public health indicators such as mortality and morbidity also rest upon a number of rather implicit assumptions (e.g. implicitly valuing death at younger and older age equally), which most of the time go unnoticed (see chapter 2).

7.2.4 Monetary valuation of environmental health impact

Using a burden of disease approach allows the quantification of a wide range of health outcomes into one comparative measure, whatever their cause or nature is. Among economists money is the usual metric to

express and compare both material and immaterial goods. From the economist's perspective, health yields of public health interventions should be compared on a monetary basis, thus enabling prioritisation of interventions in different policy domains based on expected benefit-cost ratios. Assuming a person's welfare depends on her or his satisfaction of individual preferences, welfare change can be measured by observing how much a person is willing to pay for it, or, in other words, willing to give up in terms of other consumption opportunities. This approach can also be applied to non-market goods like environmental quality and health, although obviously often much more complicated than with respect to market goods and services. Two central assumptions of CBA are often criticised: individual well-being can be measured in terms of preference satisfaction and collective social well-being is simply the aggregation of individual well-being. The first objection is pertinent not only to CBA but to all possible forms of health impact assessment, the second is very true, and thus requires explicit incorporation of social values in the policy process, such equity, ecological integrity, and sufficient precaution[27].

Since no proper markets exist for environmental benefits such as clean air or improved health, their monetary values cannot be readily observed. Thus monetary values of health responses have to be derived by other means[28]. Two of the most widely applied approaches are:

- measuring people's *willingness-to-pay* (WTP) for an environmental benefit (the welfare cost to an individual) or conversely their willingness to accept compensation for environmental degradation
- estimating the (avoided) *cost-of-illness* (CoI) related to environmental causes or interventions (costs for the society, e.g. health care costs, loss of productivity or income).

WTP-measuring does not take into account costs borne collectively, while the CoI-approach excludes intangible costs such as the impact of disability and decreased quality of life borne by the individual. As a result CoI-values are often much lower than WTP-values. Willingness to pay values appear reasonably stable in Western societies; CoI-values may vary substantially, depending on the economic situation, the GDP-share invested in the health sector and the structure and size of the health system in a particular region.

A number of studies have collected data on people's willingness to pay for a reduction in mortality risks. It is important to note people value a reduction in mortality risk not loss of their life. Values can be obtained from investigations on how health risks related to certain risky occupations are allowed for in the differences in salary, or from the extra amount people are prepared to pay for safer or healthier products (for example, cars with airbags, or houses in quieter surroundings)[29]. This integration of economics with health science requires matching as closely as possible the starting point of the valuation analysis to the health end point: health responses, such as symptom days or an increase in mortality risk) or a health consequences, such as a hospitalisation or bed-disability day. Table 7-11 presents an overview of unit values for air pollution health outcomes as used in several major studies or models taken from Davis et al.[29].

As our primary focus is on the assessment of the environmental burden of disease, here we will not deal with all the conceptual and methodological issues and uncertainties involved in the monetary valuation of health impacts. We think it useful to provide at least a first indication of the environmental health costs in monetary terms and therefore of potential benefits of possible interventions. Because the environmental burden of disease has been expressed in DALYs, a monetary value per unit burden of disease would be required. Unfortunately, no undisputed WTP value for such an aggregate measure of health loss has yet been derived. Since a disease burden of one DALY is by definition equal to the full loss of one year of healthy life, we tentatively estimate the WTP value for one DALY by dividing the WTP value for mortality by the average number of years lost with each death (YLL). The relation between DALYs and WTP methods remains to be studied and uncertainties, such as differences in dimensions measured cannot

be ruled out. The same applies to the cost of illness approach. No CoI values per unit burden of disease have been published yet. We assume that CoI-values based on health care costs are to some extent correlated with the morbidity part of the burden of disease: Years Lived with Disability (YLD), much less with the mortality part: Years of Life Lost (YLL).

7.2.5 Uncertainty

We analysed the uncertainty in the calculations by means of Monte Carlo techniques[30,31,32]. In a Monte Carlo simulation model input parameters are treated as random variables. For each of the input parameters a probability distributions function was estimated, representing parameter uncertainty (see Table 7-1). Subsequently, an output distribution for the different health impact measures was estimated by iterative (Latin hypercube, 5000 samples) sampling from each of the defined parameter distributions, followed by recalculation. Calculations were performed in the @RISK add-in for Monte Carlo uncertainty analysis. Here we will present the 5- and 95-percentiles of this distribution as a measure of uncertainty.

7.3 Environmental health impact in the OECD

7.3.1 Estimating the environmental burden of disease

In this section we will present WHO-estimates of the total burden of disease aggregated for the OECD and non-OECD countries. Successively an assessment is made of the environmental exposure attributable burden of disease. Finally the monetary costs of health lost to environmental exposures are estimated using different methods of monetarising health loss.

7.3.1.1 Disease Burden

To estimate the health impact of environmental factors, 16 disease categories are selected that are assumed to be related to environmental exposures: tuberculosis, HIV/AIDS, diarrhoea, childhood diseases, malaria, acute respiratory infections, maternal conditions, perinatal conditions, malnutrition, cancer, depression, ischaemic heart disease, cerebrovascular disease, chronic respiratory diseases, congenital abnormalities and road traffic accidents (each causing more than 2% of burden of disease in developing countries). Disease specific and total burden of disease estimates in DALYs for the OECD and non-OECD regions are derived from the 1999 World Health Report (WHR)[33] presenting estimates for the world and 11 geographical and income regions, divided into three major disease categories with numerous disease subcategories (classification slightly different from the International Classification of Diseases (ICD-9). DALYs-estimates are only available with age-weighted and future-discounts (no uncertainty intervals). Figures were calculated using the World Bank income classification[34], and assuming that the countries or region's share in the burden of disease of its corresponding WHR region is equal to its share in that region's population.

7.3.1.2 Attributable fraction

For each category of diseases we made an explicit assessment of the environmental attributable fraction. As briefly discussed in par. 7.2.2, attributable environmental risk refers to the fraction of disease that is eliminated if environmentally exposure was reduced to the lowest feasible level. Of course this cannot always be defined very sharply as it may depend on available technology or degree of development[19]. Its limits are somewhere between plausible or conceivable and feasible defined as the most favourable level observed. We will avoid in depth discussions on whether attributable fraction refers to etiological, excess or

rate fractions, future disease burden or past exposures. Our purpose is simply to come up with a rough estimate of the fraction of disease that could be avoided by feasible and conceivable reductions of environmental exposures[35]. We accept the fact that attributable fractions added up may account for an attributive percentage well above 100%, appreciating the multi-causal nature of disease aetiology, and the interaction (or interdependency) between different causes. We calculate the attributable fraction, presuming no other preventive measures have been taken).

Using GBD-estimates as a starting point we implicitly assume that cases attributed to environmental exposure are similar to general cases with respect to duration, survival and disease burden. In some cases, in particular exposure to air pollution and noise, epidemiological risk estimates are only available for indicators of disease, such as asthma exacerbation (dairies, bronchodilators use), GP-visits, hospital admission, daily mortality or medication use (see chapter 6). In these cases attributable fractions over a range of plausible (intermediate) disease manifestations were presumed to be representative for total disease genesis and progression (as a 'worst-case assumption').

Whenever possible we used the epidemiological concept of attributable risk as a function of relative risk and exposure. Relative risks were taken from recently published, adequate quantitative reviews. Exposure levels were derived from international reports, if relevant a distinction was made between levels in the less and the more developed world. Where good epidemiological data were non-existent we relied on published estimates, often based on geographical difference in disease prevalence[14].

Health impact assessment, especially regarding regional disease burden involves large uncertainties, quantitatively as well as qualitatively. The causality of several exposure response relations is not beyond suspicion, due to the many sources of (residual) confounding and bias in standard epidemiological research. Often information on exposure distribution is scarce and incomplete. Other uncertainties comprise the *transferability* of risk ratios from one population to another (differences in the susceptibility of the populations, base-line risk, in some cases extrapolation of animal assay results to humans), the concentration and composition of pollutant *mixtures* (e.g. particle composition and size), and *differences* in local behaviour, time activity patterns).

Thus we used the range of published or meta-analysed relative risk estimates as a starting point (upper and lower estimates), and combined these with information on exposure distributions to approach the attributable disease fraction. To reflect the large uncertainties identified here, we define AR-estimates in fairly broad classes with a lower and an upper and best estimate if possible. To be used in the calculations of disease burden and economic losses with the help of MonteCarlo techniques, successively we defined subjective uncertainty distributions, using Pert-distributions, defined by an upper and lower limit and a best estimate, or non-informative uniform distribution, defining an equal probability within an interval. In the next paragraphs we briefly describe the current state of science with respect to the selected environment related diseases and establish with robust estimates of attributable fractions. The applied distributions are summarised in Table 7-1.

7.3.1.3 Disease specific estimates of environment attributable risk

Environmental burden of disease

The diseases we included cause 53% of the total burden of disease within the OECD, 64% in the non-OECD countries and 63% world-wide, as calculated from the World Health Report[data37]. Results from the literature evaluation and the subsequently chosen distributions for the environmental fraction of disease burdens are presented below.

Pulmonary tuberculosis

Worldwide tuberculosis represents an enormous burden of diseases. It causes around 2 million premature deaths each year, primarily in the developing world and it is estimated that almost 2 billion people have latent infections[36,37]. Apart from crowding and chilling associated with poor housing, indoor air pollution from solid fuel (biomass, such as wood, crop residues and dung) appears to be an important risk factor, of course in close connection with socio-economic factors, such as income, education and nutritional status. Recent studies in India, China, and Mexico City of indoor air pollution report elevated tuberculosis risks ranging from 1.1 to 2,5 times the background[38,39]. Interference with both cellular and humoral immunity, thus enhancing the virulence of airway pathogens, is suggested as a mechanism of action[40,41,42,43]. Based on crude estimates of solid fuel use attributable risks in OECD and non-OECD countries may amount to 5% (1-10) and 25% (5-45) respectively. Global burden of disease estimates from WHO do not include tuberculosis in HIV sero-positive individuals, a rapidly growing fraction, especially in Africa.

HIV/AIDS

Sexually transmittable diseases, such as HIV have no obvious relation with environmental factors. One might argue that land use developments, environmental disruptions and 'global village' travelling behaviour provide ample opportunities for microbes and viruses to switch from animal to human hosts (<1%)[44,45].

Diarrhoea

Dhiaroeal diseases primarily affect young children and are closely related to environmental factors, such as poor sanitation, public hygiene and access to clean water and safe food. Estimates of the fraction associated with environmental conditions are as high as 80-90%, based on variation between different regions in disease burden[14,46].

Child cluster

Most childhood infectious diseases, such as measles, neonatal tetanus, poliomyelitis, diphtheria and pertusis are preventable by vaccination. National immunisation programs will reduce the disease burden, which is still huge in the developing world (around 15% among children). However, transmission of these diseases, in particular measles, tetanus and pertusis, is largely associated with poor living conditions, crowding, safe drinking water and food. According to WHO improvement of housing and environmental conditions could contribute 5-10% to the reduction of disease burden associated with child cluster diseases in the developing world[46,47].

Table 7-1 Point estimates and probability distributions for Monte Carlo analysis.

Parameter	Mean	Probability distribution
Selected disease categories	*non-OECD;OECD*	
Tuberculosis	0.25; 0.05	Pert(0.05, 0.25, 0.45); Pert(0, 0.05, 0.10)
HIV/AIDS	0.002	Pert(0, 0.001, 0.01)
Diarrhea	0.85	Uniform(0.8, 0.9)
Childhood diseases	0.059; 0.002	Pert(0.05, 0.051, 0.1); Pert(0, 0.001, 0.01)
Malaria	0.75; n.a.	Uniform(0.7, 0.8); not applicable
Acute respiratory infections	0.30; 0.06	Pert(0.15, 0.30, 0.45); Pert(0.03, 0.05, 0.12)
Maternal conditions	0.03; 0.02	Pert(0.01, 0.03, 0.05); Pert(0, 0.02, 0.05)
Perinatal conditions	0.03; 0.02	Pert(0.01, 0.03, 0.05); Pert(0, 0.02, 0.05)
Malnutrition	0.07; 0.002	Pert(0.05, 0.07, 0.1); Pert(0, 0.001, 0.01)
Cancer	0.03	Pert(0.01, 0.03, 0.05)
Depression	0.02	Pert(0.01, 0.011, 0.05)
Ischaemic heart disease	0.1; 0.08	Pert(0.05, 0.1, 0.2); Pert(0.05, 0.08, 0.15)
Cerebrovascular disease	0.03	Pert(0.01, 0.025, 0.05)
Chronic respiratory diseases	0.34; 0.026	Pert(0.2, 0.35,0.45); Pert(0, 0.02, 0.06)
Congenital abnormalities	0.012; 0.007	Pert(0, 0.015, 0.03); Pert(0, 0.005, 0.01)
Road traffic accidents	0.075; 0.03	Uniform(0.05, 0.1); Uniform(0.01, 0.05)
Willingness to pay values		
WTP of mortality (million 1990US$)	4.76	Weibull(1.5, 5.3)
WTP of risk reductions 1/1000 and 5/1000 in 10 year (1999Can$) (sampling chance 50% each)	376; 598	Weibull(0.88, 560); Weibull(0.78, 326)
Lost life expectancy (1996) 7		
at death at population mean age (38.2)	44.0	General(4, 72, $LE_{age\ class\ i}$, Pi(LE))
at death < 65 yr (mean age 35.9)	45.2	General(17.3, 72, $LE_{age\ class\ i}$, Pi(LE))
at death > 65 yr (mean age 75.1)	11.7	General(4, 17.3, $LE_{age\ class\ i}$, Pi(LE))

Notes: Presented means were defined prior to simulation. The General distribution was defined with minimum and maximum values, age class life expectancy and age class size as the chance for drawing a particular life expectancy; a Pert distribution is defined with minimum, most likely and maximum values; a Uniform distribution with minimum and maximum values.

Malaria

Malaria contributes significantly to global burden of disease, of course especially in the developing world (Africa). The disease burden associated with malaria and a range of other vector-borne diseases is still increasing and might be unfavourably influenced by greenhouse-like climate changes. As nearly all malaria is related to environmental conditions, including land and water management, we apply an attributable fraction estimate of 75% (70-80)[46,50].

Acute respiratory infections

Acute respiratory infections include infections of upper and lower respiratory tract, such as pneumonia. On a global scale these diseases represent one of the largest proportions of total disease burden, in particular due to a high fatality among very young children[41,51].

In the developing world indoor pollution levels may be extremely high due to burning of bio-mass for cooking and heating, such as wood, dung, and coal. In many parts of the developing world prevalence of exposure is high, ranging from 25% in South-America to over 75% in India. Reported relative risks range from 1.7-2.7. Therefore Smith and co-workers estimated indoor air pollution attributable fractions up to 50% of acute respiratory infections in regions of Asia and Africa[41,52,53,54].

Epidemiological studies on respiratory health of children show elevated outdoor air pollution levels may increase the prevalence of upper and lower respiratory symptoms fractions ranging from 3 to 10% per

increase of 10 μg/m^3 PM_{10}[55,56,57,58,59]. An impressive series of time series analyses performed world-wide, show an increase in daily pneumonia mortality and morbidity (emergency hospital admission) associated with particulate air pollution ranging from 1 to 3% per 10 μg/m^3 PM10[60,61].

Analogously, epidemiological studies, primarily concerning young children have revealed an effect of indoor air pollution on respiratory health. Indoor air pollutants such as environmental tobacco smoke, NO_2 (from unvented heat appliances, solid fuels), dampness may increase prevalence and incidence of lower respiratory infections with 7-20%[62,63,64,65,66,67].

Based on these figures we propose an environment attributable fraction of 35% (15-45) in the developing world and 5% (3-12) in the OECD.

Perinatal and maternal conditions, congenital abnormalities

Perinatal conditions linked with environmental factors may include low birth weight, associated with (indoor) air and noise pollution (<5%)[68], sudden infant death syndrome due to ETS (10-25%)[69]. Poor housing conditions may affect the health of the mother and the new-born child. Furthermore, there are indications that intra-uterine and breast milk exposure to persistent organic pollutants, such as dioxin and PCB's, may affect neurological development of young children (e.g. mediated through thyroid metabolism). As low birth weight is an important factor in perinatal mortality we estimate the environmental fraction to be between 1 and 5%.

Maternal conditions are linked to environmental conditions through poor household conditions (crowding, chill, indoor air pollution, poor ventilation, poor sanitation; AR: 1-5%, respectively 0-5% in OECD). Congenital anomalies are another environmental health concern, as a large number of chemicals have been shown to induce teratogenic responses in experimental animals. A series of epidemiological studies of occupational and environmental exposures to pesticides, certain persistent organic pollutants, endocrine disrupters and/or chemical waste sites indicate some elevated risk for congenital malformations, such as neural tube and orofacial clefts and male genital defects[70,71]. However, equivocal results or often merely circumstantial evidence suggest only a minor influence of environmental exposure on incidence of congenital defects ($AR_{non\text{-}oecd/oecd}$: <3%, <1%).

Malnutrition

The disease burden estimate for malnutrition includes only disease associated with nutritional deficiencies, such as minerals (iron, iodine), proteins, and vitamins. The influence of malnutrition on incidence and prognosis of other diseases is not encompassed. Here it is assumed that environmental conditions, such as land degradation and soil pollution, have only a minor impact on availability and quality of foods ($AR_{non\text{-}oecd/oecd}$: 5-10%, <1%)[46].

Cancer

Lung cancer is by far the most important individual neoplasm in terms of disease burden, ranging from more than 10% of total cancer burden in low and middle-income countries to around 20% in the high-income countries)[33]. Most lung cancer is caused by tobacco smoking (AR: 80-90%)[72,73], there are several established relations with environmental factors, such as indoor radon (AR: 3-10%)[73,74], environmental tobacco smoke (1-5%)[75], indoor and outdoor air pollution (AR: outdoor particles, PAH, 4-10%)[76,77,78,79,80].

Especially in developing countries stomach cancer is the next most important neoplasm. There are weak toxicological indications that high nitrate concentrations in drinking water may affect gastrointestinal cancer risk as the may be converted into nitrosamines, a family of highly carcinogenic

compounds. However, recently Heliobacter pyloris infection was identified as a major important risk factor, as it induces chronic superficial gastritis, which untreated will persist for decades, leading to ulcers and ultimately to stomach cancer. In developing countries H. pyloris infection is quite common. Infection is most probably facilitated by poor sanitation and crowding. As living conditions improved in the developed world, the rate of H. pylori infection has decreased substantially. In the same era stomach cancer incidence decreased and is now far down on the list. (AR: 20-50%)[81,82].

There are weak indications for associations between environmental exposure to radiation (including electromagnetic fields), and/or some widely used chemicals such as benzene and the incidence of leukaemia, and related neoplasms of the blood and lymph system[83,84,85]. However, epidemiological studies lack consistency. Furthermore, however tragic the individual cases, these neoplasm are very rare and from the public health perspective constitute only a very minor disease burden.

In the developed world breast cancer has gradually become one of the most important cancer in terms of health loss. Among the established risk factors are early menarche, late menopause, no pregnancy or late childbearing, prolonged hormone replacement therapy, higher socio-economic status and obesity. Most of these appear to be associated with hormone status, and long-term internal exposure to estrogens and other (sex) hormones. Several authors have been pointing at persistent organic compounds (organochlorines), such as dioxins, PCB's and certain pesticides, ubiquitously present in the environment to be involved in breast cancer genesis. These chemicals may interfere with hormone metabolism, for instance by mimicking estrogenic bioactivity. However, empirical evidence is scant and inconsistent. Furthermore the estrogenic potency of these POP's appears to be much weaker than endogenous sex hormones, even weaker than constituents in normal vegetables[86,87,88]. The attributable risk would most probably not exceed 3%.

The relation between exposure to UV radiation (sun, appliances) and skin cancer is probably causal. Of course exposure depends very much on life-style and contemporary fashion. Nevertheless a small proportion of skin cancer incidence, including the very fatal melanoma's, can be attributed to ozone layer depletion increasing the amount of UV reaching our bare skin: 1-5%. It has to be noted that the most prevalent types of skin cancer (basal and squamous cell carcinomas) cause very little fatality and only relatively mild disease burden[89,90,91].

Adding up the partial attributable fractions an estimate between 1 and 5% is plausible for the contribution of the environmental to total neoplasm burden.

Depression

Mental disorders, ranging from mild anxiety and depressive states to severe schizophrenia, are a growing public health concern. At this moment they account for more than 10% of total burden of disease, and projections suggest depression will become the second leading cause of disease burden in the next decades, right after ischaemic heart disease[14,92]. Although some chemical pollutants are known to interact with the psycho-neurological system (lead, mercury, carbon monoxide, toluene, organophosphorous pesticides), it is assumed here mental condition is primarily affected by the quality of the (local) environment, including noise and odour pollution, poor industrial safety, lack of social coherence and spatial quality (crime, population density, urban stress). Several spatial analyses on the level of neighbourhoods in large urban areas demonstrate a consistent association between poor spatial and environmental quality on the one hand and unfavourable perceived health and a negative perception of liveability in neighbourhoods (see chapter 1). However empirical studies on the association between high-level noise exposures around airports and hospital admission for psychological disorders are scant and inconclusive. Here we assume the attribution of environmental quality is modest (AR: 1-5%)[93].

Ischaemic heart disease

There is ample empirical evidence for an association between outdoor air pollution (e.g. particulates, ozone and indoor ETS) and ischaemic heart disease. Global data on air pollution are scant. Based on the fairly deficient database WHO Air Management Information System (AMIS) it was estimated that 65% of the non-OECD population was exposed to average annual PM_{10} levels of less than 10 $\mu g/m^3$, 5% to levels between 10-50 $\mu g/m^3$, 5% between 50 and 100, and 25% to levels >100 $\mu g/m^3$, especially comprising the population of the (mega-)cities of Asia where levels sometimes exceed 200 $\mu g/m^3$ (e.g. New Delhi). For the OECD these estimates are 25%, 54%, 20% and 1% respectively[94].

Studies statistically analysing the association between day-to-day variations in air pollution levels and disease specific mortality and morbidity (hospital admission) show a consistent increase in disease manifestations ranging from 1 to 5% at higher pollution levels (roughly 1% (0.5-1.5) per 10 ug/m^3 PM_{10} increase). These air pollution attributable elevations in recorded morbidity and mortality may at least be interpreted as aggravation of pre-existing disease symptoms leading to (increased) medical consumption and, in some cases, death due to cardiac arrest. In other words, we may interpret these acute responses to air pollution as an indicator of increased, aggravated morbidity burden. However, recent analyses cast doubt on the initial assumption that day-to-day variations in mortality and morbidity events were merely the result of 'harvesting'. Schwartz and co-workers revealed most of the cardiovascular mortality occurred out of hospital. Application of complex statistical techniques ('distributed lag-times') revealed the extent of lifetime lost would in general exceed three months. Furthermore, in several studies an association was found between high levels of $PM_{2.5}$ and reduced heart rate variability (a known risk factor for arrhythmia and sudden death), thus providing a biological mechanism linking cardiovascular mortality with air pollution[95,96,97]. Other study results suggest another, more systemic effect of particulate air pollution on immune status (inflammation) and blood dynamics (plasma coagulation: blood viscosity, fibrinogen and C-reactive protein)[98,99,100,101].

Results of three cohort studies in the US indicate that long-term exposure to outdoor air pollution (sulphates, particulates, ozone) increases (cardiopulmonary) mortality and thus affects survival. Preliminary results of two European cohort studies in France and the Netherlands appear to confirm these results. Comparing most with least polluted cities, the US-studies indicate that the air pollution attributable fraction might range up to 5% for an increase of 10 $\mu g/m^3$ PM_{10}[102].

Indoor air pollution studies have shown that spousal exposure to environmental tobacco smoke leads to a similar increase of IHD risk. Ecological studies in less developed parts of India and Nepal have recorded very high IHD mortality rates, apparently due to high levels of indoor pollution associated with (unvented) burning of solid fuels[41]. Until new epidemiological insights emerge 15% is a reasonable upper estimate of attributable risk.

A number of studies suggest an association between prolonged exposure to high levels of residential noise (traffic and aircrafts) and several manifestations of ischaemic heart disease (hypertension, hypertensive-use, GP-visits, Angina pectoris, Ischaemic heart disease diagnosis, cardiovascular accidents). If causal, attributable fractions would be somewhere between 1-5%[103,104].

Considering these risk estimates we propose the following attributable fractions for cardiovascular disease; $AR_{non\text{-}oecd,oecd}$: 5-15%, 5-10%.

Cerebrovascular disease

Reports of time series analysis indicate a link between air pollution and cerebrovascular morbidity and mortality. Based on analogy with risk actors such as smoking, and diet we assume the mechanism to be

roughly similar to time-series analysis with respect to ischaemic heart disease. However, recent studies indicate associations between cerebrovascular risk and particulate air pollution are less strong (AR: 1-5%)[105].

Chronic respiratory cluster (COPD & asthma)

Chronic bronchitis and asthma cause the bulk of disease burden in this category. Asthma prevalence is highest among children and young adults and its incidence appears to be affected by indoor air quality (e.g. dampness, house dust mite and fungus allergen, AR: 20%).

Studies in the developing world suggest solid fuel use represent an important cause of chronic respiratory disease, with relative risks ranging from 1.5 to 3.5. Around 66% of the households in the developing world use solid fuels, yielding attributable risk up to 50%, especially among older women of lower socio-economic status. Furthermore, acute respiratory infections in earlier life, which are also associated with air pollution, may affect the risk of chronic bronchitis.

An association between outdoor (particulates) and indoor (e.g. ETS) air pollution and asthma morbidity is evident (symptoms, medicine use, GP and/or emergency room visits, hospital admission). Although it is unclear whether this concerns only aggravation of existing disease or initiation as well, most authors assume air pollution exposure affects susceptibility throughout life[106,107].

Chronic bronchitis is most prevalent among older people (often an asthma diagnosis eventually leads to chronic bronchitis at older ages). Cigarette smoking causes most chronic bronchitis, occupational exposures may also play an important role. However, among non-smokers chronic respiratory symptoms are clearly associated with high level of indoor and outdoor air pollution. The American cohort studies suggest a structural effect of long-term exposures to air pollution on respiratory disease and survival (5-15%).

Based on the available figures we propose significant environmental fractions; $AR_{non\text{-}oecd}$: 35% (20-45); $AR_{non\text{-}oecd}$: 3% (1-10).

Other important causes of disease burden

There is obviously some association between traffic accidents and environmental conditions, e.g. structure and density of urban areas (layout and hierarchy of road systems and residential areas), but good estimates are lacking ($AR_{non\text{-}oecd}$: 5-10%; AR_{oecd}: 1-5%)[14]. Homicide/violence and suicide/self-inflicted harm may to some extend be connected to deprivation of urban neighbourhoods and degraded, unsafe environments. However, this relationship is very much confounded by associated socio-economic factors. Dementia is a rapidly growing public health concern in Western societies with typically ageing populations. Apart from some anecdotal reports linking dementia to environmental chemical exposures, there is no real evidence for an environmental link.

Table 7-2 first presents burden of disease estimates for various geographical divisions. Contrasting more and less developed regions, both within the OECD and between OECD and non-OECD, clearly shows that the burden of disease is considerably higher in the less developed countries, with non-OECD regions bearing nearly twice the burden of disease per capita compared to OECD countries. The burden of disease in less developed countries can be attributed to a larger extent to communicable disease, while in more prosperous regions health is lost primarily through the non-communicable, the latter causing approximately the same burden of disease for all regions (in absolute terms per capita). To some extent this can be regarded as demonstrating the so-called epidemiological transition, although spatial differences should not be confused with temporal changes.

Table 7-2. Burden of disease: total, by major disease category and environmental.

Region	OECD			non-OECD	World
	high income	lower income	total		
Population in millions	884	224	1 108	4 797	5 905
(%)	(80%)	(20%)	(19%)	(81%)	(100%)
Burden of disease					
(in DALY/1000 capita)	120	190	134	258	235
Communicable diseases	*8 (7%)*	*41 (22%)*	*15 (11%)*	*115 (44%)*	*96 (41%)*
Non-communicable dis.	*97 (81%)*	*115 (60%)*	*101 (75%)*	*101 (39%)*	*101 (43%)*
Injuries	*14 (12%)*	*34 (18%)*	*18 (14%)*	*42 (16%)*	*38 (16%)*
Environmental fraction	2.1%	4.3%	2.7%	12.5%	11.5%
(5^{th}-95^{th} percentiles)	(1.7-2.5)	(3.9-4.6)	(2.4-3.1)	(11.7-13.3)	(10.8-12.2)

Notes: Classification of diseases and burden of disease from WHR[33]. OECD high income countries: Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Italy, Japan, Korea, Luxembourg, Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, Switzerland, United Kingdom, United States: lower income countries: Czech Republic, Hungary, Mexico, Poland, Turkey (using World Bank income division[34]).

Table 7-2 also presents the estimated environmental fraction of the total burden of disease, as calculated from simulating the distributions for the 16 selected diseases given above. It shows that the environmental burden of disease is again much higher in poorer countries, not only because of a larger absolute total burden of disease but also in relative terms, with mean estimates ranging between 2% of the burden of disease for the highest income region to 12% for non-OECD countries. Environmental burden of disease is higher in lower income countries mainly due to diarrhoea, acute respiratory infections and malaria, diseases highly related to detrimental hygienic and housing conditions and long since recognised as major targets of public health efforts.

Figure 7-1 presents both total and environmental burden of disease as percentages of total disease burden. On the lower axis, it first shows that the selected diseases clearly differ in their impact upon the population's health when comparing lower with higher income regions. In the former, acute respiratory infections and perinatal conditions each amount more than 6% of the total burden of disease, while in developed countries cancer (14%), ischaemic heart disease and depression (each 8%) are the most important. Second, the environmental burden of disease in developing countries is larger and clearly concentrates in typical infectious diseases such as diarrhoea, malaria and acute and chronic respiratory disorders. The upper axis finally adds an age perspective showing that in less developed regions the very young bear about half of the total burden of disease, compared to less than 10% in high income regions (based on data for the Established Market Economies vs. non-EME[21])

7.3.2 Estimating environmental health costs

To produce some sense of the magnitude of environmental health burden expressed in monetary values, we applied 4 approaches:

- the environment attributable fraction of total health expenditures, proportional to the fraction of total burden of disease,
- the environmental fraction of total YLL (the mortality part of DALYs), monetarised as annual (regional specific) GDP/capita
- the environmental fraction of YLL, monetarised by using WTP-values

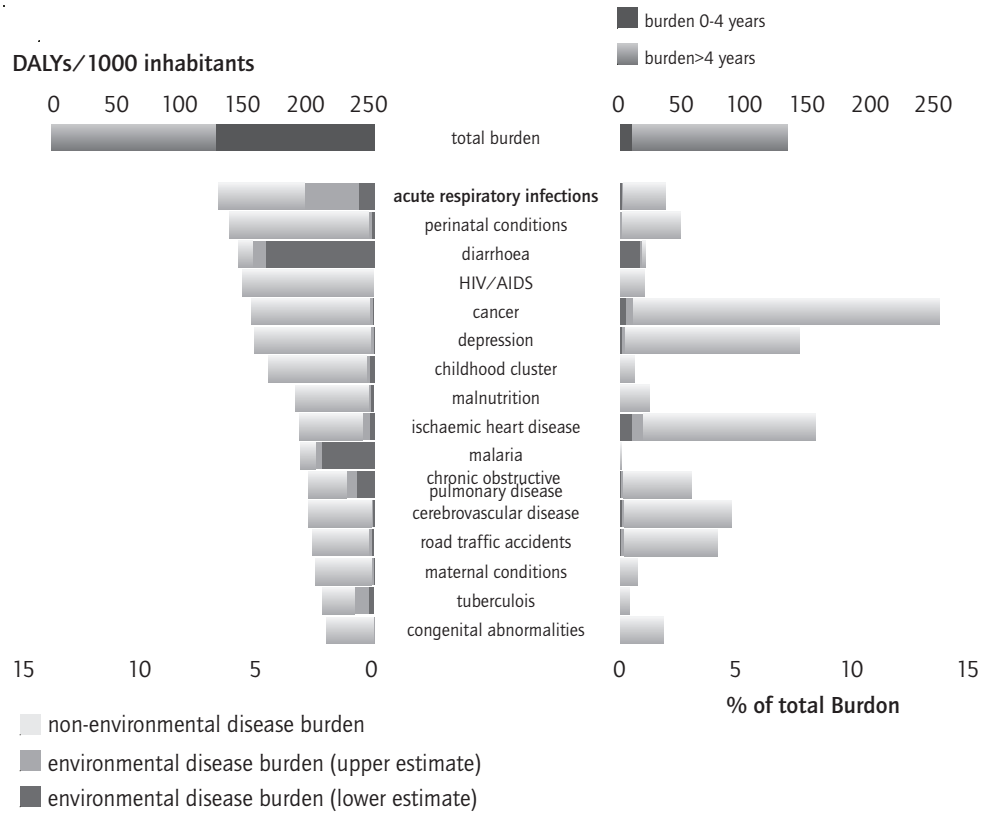

Figure 7-1. Patterns of total and environmental Burden of Disease in the developing and the developed world (non-OECD versus OECD).

the environmental fraction of YLD (the morbidity part of DALYs), monetarised by means of disease specific costs of illness

Of course the fourth approach of morbidity costs can be combined with either the second or the third approach of mortality costs to approach total monetarised health loss.

7.3.2.1 The environmental fraction of total health expenditure

Environmental health costs -and therefore the potential benefits of environmental interventions- can be estimated by applying the environment attributable fraction of total burden of disease to the total expenditure on health for the OECD regions7. Total expenditure on health includes all medical costs: personal health care services, medical goods dispensed to out-patients, services of prevention and public health, health programme administration and health insurance, investments into medical facilities, education and training of health personnel and costs of health research and development. In this approach interventions reducing the environmental burden of disease are assumed to lower health expenditures with a corresponding proportion, to be interpreted as an estimate of the potential monetary value of the health benefits of environmental interventions.

7.3.2.2 The environmental fraction of monetarised mortality and morbidity

Total health expenditures probably poorly represent mortality, while at the same time mortality comprises the major proportion of total burden of disease. Furthermore, using similar monetary DALY values for all disease categories may also obscure substantial costs differences. We therefore divided both total and environmental burdens of disease into their mortality (YLL) and morbidity (YLD) portion and valued these separately[21].

The monetary value of a YLL can be approached by the contribution to the Gross Domestic Product during one year of life, simply by dividing the annual GDP within a region by its population (GDP/capita). As an alternative, incorporating indirect (social) costs as well, willingness-to-pay (WTP) values can be applied. A number of studies have measured how much people are willing to pay for an environmental benefit or to avoid an adverse outcome, providing values for a variety of environment related health outcomes. The 1999 US-EPA[108] report on the Benefits and Costs of the Clean Air Act summarised 5 contingency valuation and 21 labour market studies, with mean WTP values for mortality ranging between 0.6 and 13.5 million 1990-US$. A well designed contingency valuation study by Krupnick et al[109] recently produced numbers between 1.2 and 3.8 million 1999-Can$. Regarding the question whether or not respondents implicitly discount the extent of futurity of a risk (reduction), as also observed by Krupnick et al., a UK government study[110] suggested a 1% discount rate per year similar to the Treasury's Green Book (6% is usually applied within the UK public sector). With respect to age effects, an earlier EPA report[111] proposed two different mortality WTP values for deaths at age below and over 65 years of age, respectively 9 million (high estimate) and 1.9 million 1994-US$ (low estimate), while the UK report suggested declining values for mortality WTP for people over 70. Krupnick et al also reported lower WTP's amongst respondents over 65's (about 15% lower), although not significant possibly due to the small study group size.

Here we apply the data from the 1999 EPA study[108] and Krupnick et al[109]. to define Weibull distributions for willingness to pay values (in accordance with these studies), taking into account Purchasing Power Parity (PPP) factors and inflation rates. We have also studied the effect of an implicit 3% future discount, similar to the value used in the WHR[33] for discounting future health loss and neatly between the two values mentioned in the UK study[110]. The influence of age is taken into account applying the UK age factors (80% of WTP for mortality if death occurs at age 70-74y, 65% at 75-79y, 50% at 80-84y, and 35% at death above 85y). We sampled from these distributions in accordance with the OECD high-income population structure (with 87% below 65 years)[7]. Dividing the WTP value by the remaining life expectancy of the appropriate age group within each simulation, finally produces monetary values for one YLL. Age group life expectancies were drawn from a general distribution based on the OECD high income life expectancy data and population structure[7] (Table 7-1).

Table 7-1 presents defined means and distributions. Both the GDP and the WTP monetarisation is performed for the high income OECD region only because of morbidity data availability (see below).

7.3.2.3 Monetarising morbidity based on Cost of Illness

DALYs related to *morbidity*, YLD, are monetarised by using direct costs of illness, defined as the costs of health system activities to prevent, diagnose and treat health problems[7], specific for 17 major disease categories as used in the ICD-9. Recent data (in local currencies) were available for 7 out of 29 OECD-countries for various years between 1990 and 1996, all belonging to the OECD high income region: Australia (most recent data: 1993), Canada (1993), (West-) Germany (1990), Japan (1993), Netherlands (1994), Spain (1993) and Sweden (1991)[7]. Data are made comparable by expressing the costs as percentages of the national GDPs, applying these to their 1997 GDPs, and subsequently calculating

percentages for the combined 1997 GDP of the 7 countries, being a proxy for the whole OECD high income region distribution of costs of illness over the 17 ICD disease categories. A monetary value per morbidity DALY for each disease category is finally derived through dividing the ICD category specific cost estimate by the number of DALYs related to morbidity caused by that category.

7.3.2.4 Calculations

So, the estimated environmental burden of disease is monetarised in three different ways. Table 7-3 presents the environmental health costs for the OECD region as an environmental fraction of total health expenditures. Means range from only 16 US$ per capita per year in the lower income countries (combining higher environmental fractions with lower GDP and total health expenditures percentage) to more than 50 US$ in high income and total OECD regions.

Table 7-3. Environmental health costs based on total expenditure on health.

OECD		high income	lower income	total
GDP (PPP)	- in billion US$	20 811	1 656	22 467
	- $ per capita	23 700	7 700	20 500
Total expenditure on health (% GDP)		10%	4.9%	9.9%
Environmental fraction of total BoD (5^{th}-95^{th})		2.1% (1.7-2.5)	4.3% (3.9-4.6)	2.7% (2.4-3.1)
Environm. health costs	- billion US$ (5^{th}-95^{th})	45 (37-53)	3.5 (3.2-3.7)	61* (53-68)
	- $ per capita (5^{th}-95^{th})	51 (43-60)	16 (15-17)	55* (48-62)

Notes: Economic data for 1997[7].GDP=Gross Dometic Product, PPP=Purchasing Power Parity, BoD=Burden of Disease. * This health cost figure for the total OECD region is not the sum of the high and lower income region. It is calculated by using environmental percentages, GDP's and total health expenditures and population for the total OECD region.

For the OECD high income region only, the cost of environmental disease burden expressed in YLL is monetarised using 1997 GDP(PPP) per capita: 23,700 US$/YLL. Multiplying this value with the mortality fraction (63%, equal to 100% minus the morbidity fraction, see Table 7-4) of the previously calculated environmental burden of disease, results in total environmental mortality costs of 36 billion US$ (5^{th}-95^{th} percentiles 29-44) or 41 US$ per capita (33-50) per year. These estimates are lower than those approached by the environmental fraction of total health expenditures.

Estimates based on willingness-to-pay values for avoiding mortality are presented in Table 7-5. The WTP values for mortality range from 2-10.6 million US$/years. Dividing these by the remaining age group life expectancy produces a monetary value between 80 and 260 thousand US$ per YLL (remaining age group life expectancies for total OECD high income population: 44 years (5^{th}-95^{th} percentiles 20-69); for death below age 65: 45 years (26-69), for death above age 65: 12 years (6-17)). Combing this value with the mortality fraction of the environmental burden of disease finally results in environmental mortality costs: 110 to 400 billion US$ or 150-480 US$ per capita per year, considerably higher than the previous values. Assuming a future discount rate of 3% being implicit in the collected WTP values produced nearly doubled mean WTP values for mortality and environmental mortality costs, while the use of age factors as proposed in the UK study[110] one the other hand made hardly any difference.

Table 7-4. Environmental morbidity costs based on direct costs of illness.

Selected environmental disease category	Environmental burden of disease (1000 DALYs)		YLD per DALY (%)	Direct CoI per YLD (1000 US$)	Environmental morbidity costs (million US$)	
	mean	(5th-95th)			mean	(5th-95th)
Tuberculosis	6.3	(1.9-11)	9.3	26	15	(4.5-27)
HIV/AIDS	2.3	(0.3-5.5)	25	26	15	(1.9-35)
Diarrhea	240	(230-260)	90	26	5 700	(5 400-6 000)
Childhood diseases	0.8	(0.1-1.8)	76	26	16	(2.0-3.6)
Malaria	0			26	-	-
Acute respiratory infections	81	(50-120)	11	33	310	(190-460)
Maternal conditions	6.6	(1.7-12)	93	113	690	(180-1 300)
Perinatal conditions	36	(9-67)	13	6.5	120	(31-230)
Malnutrition	2.1	(0.3-4.8)	86	20	35	(4.4-80)
Cancer	510	(300-720)	13	46	2 900	(1 700-4 200)
Depression	150	(91-250)	100	5.1	760	(460-1 300)
Ischaemic heart disease	810	(550-1 100)	9.3	74	5 100	(3 500-7 000)
Cerebrovascular disease	140	(77-200)	31	74	3 100	(1 800-4 700)
Chronic respiratory diseases	93	(28-170)	70	33	2 200	(660-3 900)
Congenital abnormalities	12	(6.4-17)	45	6.5	37	(19-51)
Road traffic accidents	130	(54-210)	31	22	910	(360-1 500)
Sum of all selected diseases	2 200	(1 800-2 600)	37	32	22 000	(19 000-25 000)

Notes: Calculations for OECD high income region. YLD=Year Lived with Disability, equals one DALY due to morbidity. CoI=Costs of Illness.

Table 7-5. Environmental mortality costs based on willingness to pay values.

	WTP for mortality (million US$)		Monetary value of one YLL (million US$)		Environmental mortality costs (billion US$)	
	mean	(5th-95th)	mean	(5th-95th)	mean	(5th-95th)
EPA data[108]	5.8	(0.8-13.7)	0.16	(0.02-0.4)	240	(29-623)
+ implicit 3% future discount	10.6	(1.5-25.5)	0.26	(0.04-0.6)	403	(54-990)
+ age factors	5.8	(0.8-13.6)	0.15	(0.02-0.4)	231	(29-605)
+ discount and age factors	10.6	(1.5-25.5)	0.26	(0.04-0.6)	393	(54-970)
Krupnick data[109]	2.0	(0.04-7.9)	0.08	(0-0.29)	120	(2-447)
+ implicit 3% future discount	3.7	(0.07-14.6)	0.13	(0-0.50)	201	(4-768)
+ age factors	2.0	(0.04-7.9)	0.07	(0-0.28)	114	(2-435)
+ discount and age factors	3.7	(0.07-14.3)	0.13	(0-0.49)	195	(4-753)

Notes: Calculations for OECD high income region. YLL=Year of Life lost, equal to one DALY due to mortality.

Monetary values for *morbidity* DALYs (or YLDs, 37% of total, see Table 7-4) are presented in Table 7-7 and Table 7-4. Monetary values for YLL range widely between 5 and 130 thousand US$, depending on disease category; the average is 23,000 US$.

Table 7-6 Direct costs of illness per morbidity DALY (YLD) by ICD category.

ICD category	Burden of disease (1000s)	YLD per DALY (%)	Direct costs of illness (% of total)	Costs per YLD (1000 US$)
Infectious and parasitic diseases	2 800	46	2.7	26
Neoplasms	16 900	12	7.8	46
Endocrine, nutritional and metabolic diseases	5 200	70	5.2	20
Diseases of the blood and blood forming organs	-	-	0.6	-
Mental disorders	22 400	96	8.9	5
Diseases of nervous system and sense organs	2 200	58	6.5	63
Diseases of circulatory system	19 100	17	19.0	74
Diseases of respiratory system	6 200	52	8.8	33
Diseases of digestive system	5 200	56	8.8	37
Diseases of genitourinary system	1 200	46	5.7	128
Complications of pregnancy, childbirth and puerp.	360	93	3.1	113
Diseases of the skin and subcutaneous tissue	130	-	2.3	-
Diseases of the musculoskeletal system and connective tissue	4 400	95	8.1	23
Congenital anomalies	1 900	45	0.4	7
Certain conditions originating in the perinatal per.	1 900	13	0.5	26
Symptoms, signs and ill-defined conditions	-	-	4.3	-
External causes of injury and poisoning	12 400	33	7.2	22
Sum of all ICD categories	106 000	50	100	23

Notes: Calculations for OECD high income region. YLD=Year Lived with Disability, equals one DALY due to morbidity. Since the classification of diseases as used in burden of disease calculations (21, p.120-2) sometimes differs from the ICD, burden of disease estimates for each ICD-category had to be calculated by slightly adjusting WHR estimates. The burden of Diseases of the blood is included in Endocrine, nutritional and metabolic diseases. Diseases of nervous system and sense organs includes the burden of other neuropsychiatric disorders. For Diseases of the skin no YLD were provided. The burden of Symptoms, signs and ill-defined conditions is added to GBD and WHR Group I (for people < 5 yr) or to Group II (>5yr). Economic data (PPP, 1997)[7] for the 7 countries: Direct Costs of Illness 424 billion US$, total health expenditure 564 billion US$, GDP 7 205 billion US$.

Environmental morbidity costs were finally estimated through combining the morbidity fraction of the 'environmental' disease burden of each selected disease with the monetary morbidity DALY value for the appropriate ICD category, as shown in the subsequent rows of Table 7-4. Estimated total environmental morbidity costs were 22 billion US$ (5^{th}-95^{th} percentiles 19-25) per year or 25 US$ per capita (21-29) per year, about 1.4 % (1.2-1.6) of total direct costs of illness in the OECD high income region (as estimated from costs of illness for the 7 countries and total health expenditures for the 7 countries and the whole high income region).

Table 7-8 finally summarises results of the three estimates for the OECD high income region: first as approached with total health expenditures resulting in 51 US$/cap (43-60) per year, second and third by summing GPD or WTP based environmental mortality costs with morbidity costs based on direct costs of illness, respectively 66 US$/cap (55-78) per year and a range of means from 150 up to 480 US$ per capita per year. To give an indication of potential benefits of environmental policies the monetarised yields of a 5% reduction of environmental burden of disease are also presented.

Table 7-7 Summary of total environmental health costs as estimated by three methods.

Estimation of environmental health costs based on:	Total environmental health costs		Benefits of a 5% reduction in environmental disease burden	
	billion US$	US$ / capita	billion US$	US$ / capita
Total Expenditure on Health (with 5th-95th percentiles)	45 (37-53)	51 (43-60)	2.3 (1.9-2.7)	2.6 (2.1-3.0)
GDP/capita *plus* Direct Costs of Illness (with 5th-95th perc.)	58 (48-69)	66 (55-78)	2.9 (2.4-3.5)	3.3 (2.7-3.9)
Willingness To Pay *plus* Direct Costs of Illness (lowest and highest mean of different distributions)	136 – 425	154 - 481	6.8 – 21	7.7 - 24

Note: Calculations for OECD high income region. GDP=Gross Domestic Product.

7.4 Discussion

This exploratory study suggests that the environmental burden of disease (EBD) might be up to 12% of the total global burden. As expected EBD is substantially larger in the lower income regions; within the OECD region it ranges between 1.7 and 4.6%. Estimates of environmental health costs in money indicate that potential benefits of intervention policies aiming at reduction of environmental pollution can be quite substantial, not only in terms health but also terms of money. Covering such a wide area of diseases, geographical regions and important dimensions of human life such as health and economics obviously implies a number of potential flaws and uncertainties. In this section we will deal only with those that are specific to the way in which we approached environmental burden of disease and subsequent monetarisation.

7.4.1 Environmental Burden of Disease in DALYs

The GBD-method to characterise and quantify environmental health loss has by now become an important tool for world-wide assessment of disease burden and possible health yields of health policies, e.g. in the World Health Reports[8]. As with any other new approach, it has also been extensively criticised, e.g. with respect to the way in which severity was weighted (how and whom), the use of age weights, the derivation of morbidity from mortality data, etc. to which its original inventors Murray and Lopez and other authors have on many occasions replied[112,113,114,115,116,117]. It is stressed here that aggregate public health indicators as the DALYs should only be applied on a population level as a tool for public health impact quantification and an aid for policy making and evaluation. The DALY-paradigm takes the policymaker's perspective, having to choose between expensive interventions, given limited resources to protect or promote public health.

A number of other possible flaws of quantification of environment attributable fractions are already discussed in numerous publications. They concerns health impact assessment in general, such as the imprecision of population exposure assessments, the unknown and probably 'unknowable' shape of the exposure-response curves at low, environmental exposure levels, and the translation of exposure-response information from one species to another as well as from one population to another. Obviously the manner in which we established attributable fraction in this study was little sophisticated, involving many subjective choices. However, by applying relatively wide intervals at least a first order sense of magnitude can be given. Further work on this type of assessment must involve more precise exposure distribution assessment, as has been undertaken in the framework of the World Heath Report 2002[8].

In this study the environmental burden of disease was estimated to be somewhere between 2-5% in the OECD and 7-12% in the rest of the world. As reported in chapter 4 in 1999 we estimated the

environmental burden of disease for the Netherlands at less than 5 percent of the total burden of disease. That result is strikingly similar to those of the present study, considering the fact that the both studies apply different methodologies. The 1999 study employed an exposure-based approach with exposure specific health outcomes while the current study applied an outcome-based approach combined with crude assessments of environmental attributable fractions. In 1999 Smith et al. attributed a substantial higher fraction of world-wide disease burden to environmental exposure (25-33%). This is partly due to a slightly more 'generous', worst case attribution of disease specific burden to environmental exposures (5% was the default minimum environmental component of any disease category); in particular their estimates of attributable fractions for acute respiratory infections, cancer, and road traffic accidents important categories are considerably higher. Furthermore they included important categories, we excluded, such as falls, war, violence and drowning[14]. In the 2002 World Health Report 'Reducing risks, promoting healthy life' disease burden estimates were presented for a limited number of environmental exposures. For unsafe water, sanitation and hygiene estimates of disease fractions are similar to the ones presented here; estimates for outdoor air pollution attributable fractions are substantially lower for cardiorespiratory disease (2%, only pertaining to mortality), but similar for (lung) cancer. WHO-estimates of attributable fractions of respiratory infections due to indoor air pollution in the developing world are slightly higher than ours (37.5%), but within uncertainty limits, and fairly similar for COPD and cancer[8].

7.4.2 Monetarisation of environmental burden of disease

Comparative assessments of public health loss due to environmental exposures, enabling evaluation of interventions can be essential to policy makers. Cost-benefit assessment through monetarising health yields may facilitate setting priorities in a broader field than public health alone. In addition, expressing health losses and gains in money may be more appealing to decision makers. However, it is important to take into account some considerable potential flaws of our crude approximations of environmental health costs.

Approaches based on an environmental fraction of the total health expenditure may introduce considerable over- or underestimation, since both environment attributable burden and health expenditures and may differ substantially from one disease category to another. Furthermore, expenditures on health hardly include the social and economic costs of mortality. In a burden of disease approach loss of life-years constitute a substantial fraction. Therefore monetarisation based on health expenditures can be considered as a lower limit.

Lower income countries combine a substantial higher burden of disease with much lower health expenditures and GDP. As a results environmental health costs in developing countries are much lower than in high-income countries, both per capita and in absolute terms. Likewise, a reduction of (environmental) burden of disease would yield much smaller benefits. If looked at in a static way, this would lead the slightly perverse suggestion that investing in reduction policies in these countries would be less worthwhile than in high-income regions. It would be better to interpret this lower yield of environmental health policies as an illustration of an undesirable health gap. Monetarising health gains of environmental policies in lower income countries therefore preferably include otherwise necessary investments in the health care system as well.

Expenditures on health obviously primarily concern morbidity. Therefore we came up with separate approaches for mortality and morbidity cost estimations. The availability and reliability of ICD category specific health expenditure is poor: for only 8 out of 24 high income countries complete data for some year are available (one was excluded due to data inconsistency). Other shortcomings are: large differences

between countries with respect to the health care system that are difficult to take into acount, the definition of health care sectors and methods of apportioning to these sectors.

The monetarisation of mortality based on loss of productivity has the advantage of the availability of generally undisputed economic data, such as actual national product or income data. However, such an approach does not include intangible, emotional and societal costs of disease and death. Numerous studies have investigated people's willing to pay for an environmental benefit, based on the idea that the preferences of individuals extend from material goods to immaterial goods, such as health and environmental quality. Although no markets exists for environmental benefits, it appears possible to deduce how individuals trade off such (im)material goods against each other, taking into account all the aspects they value[118]. This approach has by now widely gained credibility (e.g. UK government, US-EPA, IPCC). However, it is still questionable to which extent individual and societal values, such as care or self-fulfilment, socio-economic or generational equity can be sufficiently incorporated into individual willingness to pay based on utilitarian assumptions. Furthermore, societal value is not by definition the same as the aggregated individual values. Within a public health impact approach and from the perspective of the policymaker allocating public resources, such values and their discussion in politics and other societal arena's are often key inputs in decision making.

Adverse environmental health outcomes show a huge variety ranging from acute respiratory symptoms and restricted activity days to asthma hospital admissions, cancer and death. The burden of disease approach as well as monetarisation weigh the impact of these different outcomes: DALYs incorporate duration and severity of adverse health responses, while WTP trades off health and other valued services money can buy. It is unlikely however that the same dimensions are valued for all diseases. For instance, the mortality-aspect of DALYs (YLL) depends on life expectancy alone, while the non-linear relation between WTP-values and age at death[110,111] suggests that individuals value other dimensions too. In addition, the use of costs of illness to monetarise environmental morbidity burden implies that disease severity and duration are related to the actual care costs of a disease. This assumption is not totally implausible, but not (well) documented either. Other differences between the DALY approach and WTP or health costs monetarisation involve the reference year and the use of discount rates and age-weights. While DALYs and WTP both deal with future health loss -DALYs ascribe disease burden to annual cases (or mortality)- actual health expenditures concern current health loss caused by past incidence. The effect of this difference in time frame will be small as long as disease patterns show only minor change, especially as losses in the far future have less significance due to the discount rate. However, for some diseases with rapidly changing incidences or treatment costs this effect may be considerable. Age weights and a 3% future discount are explicitly used in the DALY methodology, but whether WTP valuations -implicitly- do so is unclear but of great importance as shown.

Recently, some other estimates of environmental health costs were published as well. Seethaler et al[119] estimated the environmental health costs within Austria, France and Switzerland using a WTP approach at 765 US$ per capita due to air pollution, while Davis et al. report that an annual reduction of unit (1 $\mu g/m^3$) in $PM_{2.5}$ can generate benefits no less than about 250 US$ per capita[120]. A recent study by the United Nations assessed transboundary air pollution costs and benefits within the European Union of emission reduction from current reduction plans and legislation to the maximum technically feasible reduction using a WTP approach, including health benefits as well as benefits to crops and materials[121]. Benefits were estimated at 125-540 US$ per capita (additional calculation by the authors), exceeding costs already at the lowest estimate of mortality due to acute particulate matter exposure (excluding long term mortality that would dominate air pollution health impact)[80]. Although these studies concern air

pollution only and differ in covered area, selection of diseases and methodological aspects (e.g. not applying a EBD-approach), they all provide estimates in the same order of magnitude as our WTP based environmental health costs. Thus we regard estimation of the total environmental health costs, based upon a monetarisation of the environmental fractions of the burden of disease, as a provisional but promising approach. Despite the uncertainties and potential flaws discussed in earlier sections, the estimated WTP based environmental health costs are apparently a reasonable upper estimate. Costs based on total health expenditure may be regarded as a reasonable lower estimate.

Table 7-8. Schematic and tentative presentation of the relative contribution of issues and sectors to the Environmental Burden of Disease in higher income OECD.

Environmental issue	**Contribution to environmental BoD**	**Contribution to each issue by sectors**				
		Transport	Energy use	Industry	Agriculture	Housing
Air pollution	®®®®	++	+			
Noise	®®®	+++		+		
Indoor environment	®®			+		++
Food- and waterborne	®®			+	+	
Road traffic accidents	®®	+				
Chemicals	®			+	+	
UV-radiation	®			+		
Climate change	as yet unknown, expected very high	++	++	+	++	+

Notes: The number of 'plusses' is only meaningful within each row.

Table 7-8 tentatively sketches the most important environmental exposures and corresponding sectors. Linking environmental issues with health losses to sectors may provide a better understanding of the relation between economic developments and public health as far as the environment is the intermediary variable. As mentioned earlier, the relations between health and economy are numerous and complex. Moreover, sectors often overlap to some extent and not all of the environmental health losses can be unequivocally assigned to a certain sector. Important environmental causes of lost health in developed countries are air pollution and noise problems, to which mainly the transport and the energy production sectors contribute.

Table 7-10. Tentative prioritization of diseases, issues and sectors from an Environmental Burden of Disease perspective.

	OECD	
	high income	lower income
Diseases	cardiopulmonary diseases cancer depression	communicable diseases cardiopulmonary diseases cancer
Issues	air pollution chemicals noise/liveability	sanitation/food/housing air pollution chemicals
Sectors	transport industry/agriculture housing	public hygiene/housing transport/energy industry/agriculture

Table 7-9 presents a prioritisation of diseases, issues and sectors in lower and higher income OECD-countries, based on the previously estimated amounts of environmental burden of disease. Environmental issues are both global (transboundary air pollution) and local (noise and urban liveability), pointing to the transportation sector as the first target for intervention in the OECD. Communicable diseases are still relevant in the OECD's lower income countries, pressing for further improvement of social and living conditions

Table 7-10. Transitions from traditional to (post-)modern economies.

Economic transition	**Agriculture and early industry** →	**Industry and services (focus on production)** →	**ICT and economic globalization (focus on consumption)** →	***The Future:* Sustainable Development *or* Endangered Global Life Support Systems?**
Environmental issues	Clean water scarcity Food quantity and quality Indoor air Disease vectors	Chemical emission and exposure in water, food and air (pesticides, heavy metals, POPs, VOCs, etc) UV-radiation Food (microb.)	Transboundary air pollution (particulate matter, ozone, etc.) Local urban air pollution Urban noise Urban liveability Food (microb.) Decreasing exposure to chemicals	Global warming (disrupted carbon and nitrogen-cycles) Natural disasters Increased disease vector spread Overpopulation Land degradation Water scarcity
Health effects	Communicable diseases Maternal and perinatal conditions Nutritional deficiencies	Various cancers Respiratory diseases Neurological disorders Large accidents Gastrointestinal disorders Allergies Reproductive disorders	Respiratory diseases (acute and (aggrevation of) of chronic) Gastrointestinal disorders Mortality harvesting effects (cardiopulmonary disorders) Psychosocial effects Neurocognitive dysfunction Reduced quality of life	'Return' of known and new communicable diseases (*eg*. malaria, aids, dengue) Heat/coldstress Natural disasters
Sectors (pollution sources and/or intervention opportunities)	Public hygiene and sanitation Housing Land and water management	Industrial processes Agriculture (fertilizers and pesticides) Waste disposal Use of ozone depleting substances (cfc's)	Transportation (mainly road and air) Energy generation and use Tourism	World ecological system Possible intervention areas: - technology (greening) - consumption (altered patterns) - population (size and composition)

Table 7-11. Comparison of unit values used in several major studies or models (in $1990; reproduced from Davis et al[29]).

	US - EPA[a]			**US - TAF[b]**		
	Low	Central	High	Low	Central	High
Mortality	1560000	4800000	8040000	1584000	3100000	6148000
Chronic Bronchitis	-	260000	-	59400	260000	523100
Cardiac Hosp. Admissions	-	9500	-	-	9300	-
Resp. Hosp. Admissions	-	6900	-	-	6647	-
ER Visits	144	194	269	-	188	-
Work Loss Days	-	83	-	-	-	-
Acute Bronchitis	13	45	77	-	-	-
Restricted Activity Days	16	38	61	-	54	-
Resp. Symptoms	5	15	33	-	12	-
Shortness of Breath	0	5.3	10.60	-	-	-
Asthma	12	32	54	-	33	-
Child Bronchitis	-	-	-	-	45	-
	Canada - AQVM[c]			**Europe – ExternE[d]**		
	Low	Central	High	Central		
Mortality	1680000	2870000	5740000	3031000		
Chronic Bronchitis	122500	186200	325500	102700		
Cardiac Hosp. Admissions	2940	5880	8820	7696		
Resp. Hosp. Admissions	2310	4620	6860	7696		
ER Visits	203	399	602	218		
Work Loss Days	-	-	-	-		
Acute Bronchitis	-	-	-	-		
Restricted Activity Days	26	51	77	73		
Resp. Symptoms	5	11	15	7		
Shortness of Breath	-	-	-	7		
Asthma	12	32	53	36		
Child Bronchitis	105	217	322	-		

Notes:

a. Low and high estimates are estimated to be 1 standard deviation below and above the -mean of the Weibull distribution for mortality. For other health outcomes they are the minimums and maximums of a judgmental uniform distribution (US-Environmental Protection Agency. The benefits and costs of the clean air act1990 to 2010. USEPA Report nr. EPA-410-R-99-001).

b. Low and high estimates are the 5% and 95% tails of the distribution (Tracking and Analysis Framework, developed by a consortium of U.S. institutions, including RFF).

c. Low, central, and high estimates are given respective probabilities of 33%, 34%, and 33% (Air Quality Valuation Model Documentation, Stratus Consulting for Health Canada).

d. Uncertainty bounds are set by dividing (low) and multiplying (high) the mean by the geometric standard deviation (2) (ExternE. Externalities of energy, vol.7. EU,1999).

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8 Summary and final remarks

So, what have learned from the heterogeneous mix of historical, philosophical and more practical analyses in the seven chapters of this book dealing with the relation between environment and health?

8.1 Relevance of environmental factors

In the first chapter we have seen that it took humanity quite some time to struggle itself out of the grasp of a capricious, often hostile environment, comprising famine, floods, fires, drought, earthquakes, hard climatologic conditions, predators (including man), and -in particular- the pathogenic micro-world. More than ten millennia after the Neolithic 'transition' we finally brought the environmental conditions under control.

Never in our Western-European history we have been as healthy as we are now. If we take the triangle of basic health determinants as a starting point, until the 20th century the *(physical)* environment was the source of 70-80 percent of disease burden. Thanks to the public health revolution beginning halfway through the 19th century, nowadays, environmental factors probably contribute less than 5%, while *life-style* is responsible for the bulk of the current avoidable disease burden (around 25% of total). Of course life-style can only be observed in connection with the social environment, which is still an important determinant of health status as shown by persistent socio-economic health differences.

Obviously the public health transition has changed the definition of health drastically. Public health focus has gradually shifted from life expectancy to health expectancy: postponing as long as possible or mitigating the physical, mental or social limitations brought about by the chronic diseases of older age. In the framework of environmental health impact assessment a similar situation has arisen. The impact of environmental exposures no longer predominantly involves clear mortality risks or loss of life expectancy, but comprises aspects of the quality of life in a broad sense as well: aggravation of pre-existing disease symptoms, social-psychological endpoints, such as severe annoyance, sleep disturbance, and unfavourable health perceptions and stress in relation to a poor quality of the local environment.

This shift to attention for the quality of life both in policy-making and public opinion is in agreement with Maslow's theory, which postulates that human needs are organised in a hierarchic fashion. When primary needs such as food, shelter and security needs are fulfilled, social and ego needs become more salient. The same can be observed with regard to environmental quality. As most direct physical and chemical dangers have been brought under control, environmental quality may now be looked upon as the extent to which the conditions fulfils the social needs of communities and individuals. Environmental problems predominantly persist on the highest and the lowest scale: 'global warming, transboundary air pollution versus air and noise pollution, the danger of large industrial accidents and issues related to the liveability of urban areas. A clustering of social and health problems at a local level can be observed, mainly in deprived neighbourhoods where unfavourable environmental, spatial and social factors accumulate. This requires (further) development of healthy environments with a high living quality, as a result of collaboration between planners, health and environmental experts, policy-makers and the community. In this, we can learn from the successes of the hygienists in the 19th century.

8.2 Dealing with risk in four sensible manners

In chapter 3 we looked closely at an important instrument of collective health protection, risk assessment and management. We established that in principle any decent government should apply rights-based decision rules in environmental risk policy, guaranteeing every citizen an equal right to a certain level of protection or health care. However, we have also seen that purely right-based policies may go beyond the bounds of efficiency or affordability. Vice versa, if maximisation of efficiency is the main target, there are inevitably distribution consequences: individuals or specific populations being treated unfair or even harmed in the name of efficient use of scarce resources: the traditional trade-off between 'equity' and 'efficiency' of welfare economics. Environmental policies, such as clean air regulations are not primarily aimed at balancing marginal air pollution reductions and associated health gains with marginal social losses (e.g. as a result of opportunity costs). They are aimed at protecting the most susceptible individual, regardless of the costs.

We discussed another number of 'sobering', complicating features of the concept of health risk, at least from the perspective of the average environmental engineer:

The concept of risk consists not exclusively of objectively measurable qualities of systems; risk is also a social construct shared by our equals, in which qualitative, social-psychological attributes may be decisive in the acceptence of the dangers around us.

There is no universal, unequivocal measure to quantify health risk; selecting a measure always implicates a choice of normative points of departure for policy making.

The result of (quantitative) risk assessment depends largely on the definition of the system boundaries Therefore different risks cannot be compared, just like that.

Risk assessments are always uncertain to some extent; incertitude may range from statistical inexactness, construct or model uncertainty and indeterminacy, all the way to ignorance.

However, to some extent risk can be adequately characterised, applying a limited set of quantitative and qualitative attributes, such as probability, nature and magnitude of consequences, spatial and temporal scale, persistence, irreversibility, inequity, delayed consequences, and potential to provoke societal turmoil. Of course, the composition and comprehensiveness of the most set of attributes will differ from one risk problem to another.

To deal with risk as a 'social construct', as well as with equity versus efficiency trade-offs, we tentatively propose a typology of risk problems with matching procedures of increasing comprehensiveness, comprising 1) 'business as usual', traditional, quantitative analysis and management, 2) appropriate and proportional use of 'scarce resources', 3) a way out when 'calculations are simply not the issue', or 4) wisdom, when 'ignorance' is recognised in time. Procedures involve decision rules, goals, solutions, strategies (discourse) and instruments.

If there are no problems with respect to uncertainty, unresolved complexity or efficiency, traditional quantitative methods of risk assessment and management will generally suffice. Calculation of health risk in terms of probability and effect (mortality) can support decisions on risk acceptance, applying standard evaluation frameworks (e.g. a individual mortality risk of 10^{-6} as limit of tolerable risk). The discourse is internal and merely cognitive, among experts from the agency, advising institutions and, if necessary, from involved parties (e.g. industry). Crude costs-effectiveness analysis can help decide whether the Euro for risk reduction is well spent. Main objective is *health protection of the most susceptible individual* from the perspective of everyone's right to a high level of protection. Measures should be aimed at the reduction of the additional probability of health loss to a generally accepted level in a more or less standard operational manner (the additional annual mortality risk of one in a million is a very good deal in terms of

public heath). Good examples are the setting of exposure standards for toxic compounds in ambient air, at the workplace or for food additives or residues.

A rights-based approach can be excessively expensive (in total or per life-year saved) or be in conflict with other interests highly valued in society (including opportunity costs), e.g. safety around large airports. Calculated health risks (or losses) are a good indicator of the risk problem, agreed upon by all parties. Nevertheless, the proportionality of investments needed to save lives, life years or health adjusted life-years is disputed.

Typically in these cases a balance has to be found between every citizen's right to a high level of health protection or precaution on the one hand, and efficient use of collective resources for public health on the other. The cost-effectiveness of risk reduction can be significantly improved by more specific measures aimed at high risk groups (Legionella in drinking water systems), or by focussing on new situations, instead of already existing ones (indoor radon, noise or air pollution abatement).

Finding the right balance between efficiency and equity of course is not a matter of science. Based on good scientific analysis of risk reduction, cost-effectiveness, and distribution effects, a reflective discourse involving representatives of social parties involved should lead to a broadly accepted compromise, which may include compensation for citizens who get the worst of it. An important consequence may be that not all citizens are equally protected, but at the same time collective resources are not wasted on inefficient risk reduction.

Traditional, quantitative risk management will also fail when probability and health effects (especially mortality) are not the issue in the social commotion; when calculations of mortality or morbidity risk simply do not represent the risk as perceived by the public involved. Good examples of such risk problems are the base stations for cell phones (GSM), electricity power lines and possible child leukaemia risk, BSE and endocrine disrupters. For instance, the acceptability of nuclear power facilities is often discussed in terms of very small probabilities of a serious accident, while the public is primarily concerned by the magnitude of the consequences. In these cases, risk management procedure should aim at achieving consensus among involved parties on problem definition and delineation, on risk attributes that should be taken in consideration and on efficacy, efficiency, and distribution effects of measures. The main purpose of the procedures is beyond simple health risk reduction, it is about appropriate deployment of resources, without loosing sight of social justice, and in some cases it is merely about gaining trust, empowerment of consumers (e.g. labelling), or about well-argued precaution.

To the extent that ambiguity increases, just as complexity and the possible magnitude of (health) consequences the management approach will go beyond traditional quantitative risk management procedures. Problem structuring, approaches of risk quantification, dealing with uncertainty and ignorance, application of precaution, and the design decision procedures will have to be developed in close interaction with society. Science is no longer advisor or even 'great-arithmetician', but rather 'facilitator'. This implies that politicians will have to decide in explicit uncertainty. The main objective is to arrive at a broadly accepted definition of the risk problem, an approach to 'measure' and monitor the nature and magnitude of the risk, and a framework for making decisions in pace with developments in scientific knowledge. The Intergovernmental Panel for Climate Change (IPCC), comprising not only scientific analysis, but political and socio-economic evaluation as well, is a good example of such a 'post-normal' approach.

8.3 Deaths or DALYs?

In the 'good old days' risk managers could judge the environmental quality by compliance with health-based standards, e.g. based acceptable mortality risk. As long as concentrations of pollutants were below

these standards the air was 'safe' to breathe, water appropriate to drink, soil suitable to build on. Whenever concentrations started to exceed standards regularly public health was at stake and risk-reducing measures had to be considered. Current epidemiological insights do not comply very well with this type of quality standard-based risk management as clear evidence of a threshold for the health end-points considered often appears to be lacking, at least at realistic exposure. Furthermore, the observed end-points include a large variety of adverse effects on health and well-being.

To analyse environmental health impact in a more comparative manner, aggregating the divergent health effects associated with different types of environmental exposures, we explored the application of DALYs as some sort of 'public health currency'. The DALY, developed in the framework of the World Bank/WHO Global Burden of Disease Project, is a widely used member of a family of aggregate indicator of disease burden (health loss). These indicators attempt to integrate three important dimensions of public health, viz. *life expectancy, quality of life*, and *number of people* affected. Time is the unit of measurement: 'healthy life years' are either lost by premature death, or by loss of quality of life, measured as discounted life-years within a population.

From the policy maker's point of view there are at least three clear advantages of this type of aggregation. It enables comparative risk evaluation from the perspective of public health (e.g. setting priorities), evaluation of the efficiency of environmental policies in terms of health gain, and the characterising health risk associated with geographical accumulation of multiple environmental exposures.

Despite many methodological and even ethical problems the health-adjusted life-years paradigm has proven to be a promising framework for explicit evaluation of disease burden due to environmental exposures. Especially, since more accepted risk impact measures such as annual mortality risk or non-compliance with environmental quality guidelines suffer similar implicit shortcomings (such as hidden non-scientific values). Rejecting integrate public health measures because of ethical shortcomings, in practice implies rejecting comparative health impact assessment in general, which of course is a legitimate position as well.

Using data from the Dutch National Environmental Outlook we estimated that the *long-term effects of particulate air pollution* appear to account for the greater part of the total environment related health loss in the Netherlands. Furthermore, the indoor environment appeared to be an important source of disease burden (radon, dampness, environmental tobacco smoke). Our exercises also revealed that the disease burden of environmental noise might be substantial, be it depending largely on whether the definition of health includes psychosocial responses like annoyance or sleep disturbance. Some of the typical priority pollutants, such carcinogenic air pollutants appear to be of minor relevance in the present day situation.

8.4 Health as an economic value

One of the most appealing advantages of the application of aggregate indicators like the DALY in an environmental context is the opportunity they offer for cost-effectiveness analysis (CEA). The next logical step in the development of the paradigm of comparative risk assessment is to link the health gains of environmental health policies with the necessary investments in a comparative way. 'Money is always tight, so when and how can we try to get the best deal in risk management?' Furthermore explicit and comparative evaluation of health gains in connection with policy costs enhances the accountability of governments.

We have seen that the costs per DALY saved, may vary considerably in different domains of public health policy. Especially environmental and food safety policies can be excessively expensive, partly brought about by the right-based oriented traditions of this policy domain. We have also seen that crude

calculations of health yields indicate that the air pollution abatement policies of the last decades are reasonably cost-effective, not even taking into account the obvious valuable technological developments they have brought about.

The applicability of comparative risk assessment within a policy context can be improved even further by valuing health yields in monetary terms as well, allowing comparison with policy domains outside the public health field, such as ecology, social welfare or spatial planning ('accessibility'). This is probably the final step: cost-benefit analysis (CBA), albeit many economists would consider CEA simply to be a subset of cost-benefit analysis.

CBA not only measures the costs of policies in monetary values, but also the yields, applying monetary measures as a metric of the aggregate change of individual well being resulting from a policy decision. Assuming a person's welfare depends on her or his satisfaction of individual preferences, welfare change can be measured by observing how much a person is willing to pay for it, or, in other words, willing to give up in terms of other consumption opportunities. This approach can also be applied to non-market goods like environmental and health, although obviously often much more complicated than with respect to market goods and services. Two central assumptions of CBA are often debated: individual well-being can be measured in terms of preference satisfaction and collective social well-being is simply the aggregation of individual well-being (usually just simple summation). The objections to the first assumption are pertinent not only to CBA but to all possible forms of health impact assessment, objections to the second are very true, and thus requires explicit incorporation of social values in the policy process, such equity, ecological integrity, and sufficient precaution.

Our provisional calculations of health capital lost to environmental exposures in chapters 5 and 7 indicate ample opportunities for cost efficient investments in environmental quality from the perspective of public health. However, especially with respect to valuing health losses -and thus potential gains-, still much work has to be done. More sophisticated contingent valuation studies, especially tailored to local conditions will improve the credibility of the monetary estimates of environmental pollutant attributable health loss. In our OECD study we applied GDP per capita, WTP-estimates, and disease group specific costs of illness to roughly approach the monetary value of environment related mortality and morbidity, respectively. Furthermore, it is clear the credibility as well as the transparency of this type of analyses should be improved. Most important aspects of future developments are probably: systematic peer review and involvement of stakeholders in the definition of the framework for CEA and CBA.

Finally, deaths, DALYs or dollars may all be fruitfully applied as health endpoints in comparative health impact assessment, as long as their philosophical meaning has been taken properly into account and match the health protection goals of the risk management, policymakers and, of course, society.

Samenvatting

Milieu en gezondheid

Tot in de 20ste eeuw werd de volksgezondheid in Nederland (en in de rest van de Westerse wereld) voornamelijk bepaald door omgevingsfactoren, zoals strenge klimatologische omstandigheden, hongersnood, overstromingen, aardbevingen, roofdieren en, bovenal, ziekteverwekkende micro-organismen. De historisch-epidemiologische analyse van hoofdstuk 1 beschrijft de interactie tussen mens en zijn omgeving, in het bijzonder de micro-organismen, gedurende de laatste tien millennia. In die lange periode evolueerde de wereld van een lappendeken van honderdduizenden kleine lokale geïsoleerde gemeenschappen tot een 'Global village' waarin alles aan elkaar is geschakeld. Afstand en tijd vormen geen barrière meer voor snelle distributie van (micro)organismen vanuit hun oorspronkelijke reservoir.

Inmiddels zijn we gezonder dan ooit. Dankzij de volksgezondheidstransitie die in Nederland halverwege de 19e eeuw begon met onder meer de aanleg van betrouwbare drinkwatervoorzieningen, riolering, verbeterde huisvesting en toenemende scholingsgraad, is de invloed van fysieke omgevingsfactoren op ziekte en sterfte teruggebracht van 70-80% tot waarschijnlijk minder dan 5%. In die tijd steeg de levensverwachting van 36 jaar tot 78 jaar. Steeds meer is leefstijl de belangrijkste bron van vermijdbaar gezondheidsverlies geworden, verantwoordelijk voor ongeveer een kwart van de ziektelast. Uiteraard hangt leefstijl nauw samen met de sociale omgeving, nog steeds een belangrijke bepalende factor voor de gezondheid, ook al gezien de hardnekkige sociaal-economische gezondheidsverschillen.

Die transitie gedurende anderhalve eeuw heeft ook effect gehad op de definitie van gezondheid. De focus van het volksgezondheidsbeleid is langzamerhand verschoven van levensverwachting naar gezondheidsverwachting: het zo lang mogelijk uitstellen of verminderen van de fysieke, geestelijke en sociale beperkingen die de chronische aandoeningen van de oude dag met zich meebrengen. In het domein van milieu en gezondheid is een soortgelijke verandering waarneembaar. Bij milieuverontreiniging gaat het niet langer om voornamelijk sterfterisico's of verlies aan levensverwachting, maar steeds meer om de aantasting van de kwaliteit van leven. Voorbeelden daarvan zijn het verergeren, vaker of heftiger voorkomen van symptomen van reeds bestaande ziekte, sociaal-psychologische eindpunten, zoals hinder of verstoring van dagelijkse activiteiten of slaap, of een als slecht ervaren gezondheid en stress in relatie tot een ongunstige kwaliteit van de leefomgeving. Terwijl de meeste directe fysieke en chemische bedreigingen onder controle zijn gebracht, kijkt men nu naar de mate waarin de leefomgeving aan de behoeften van gemeenschap en individu voldoet.

Milieuproblemen lijken het meest hardnekkig op zowel het hoogste als juist het laagste schaalniveau: klimaatverandering, grensoverschrijdende luchtverontreiniging versus lokale luchtverontreiniging, geluidshinder, gevaar van grote (industriële) ongevallen en de leefbaarheid van wijken. Op buurtniveau gaan sociale en gezondheidsproblemen vaak samen met een opeenstapeling van ongunstige kwaliteit van ruimte, woningen en milieuomstandigheden. Dit alles vraagt om een nieuwe definitie van gezondheid en welzijn in relatie tot omgevingskwaliteit, verdere ontwikkeling van gezonde, leefbare omgevingen door multidisciplinaire en multisectorale samenwerking van stadsplanners, experts op het gebied van milieu en gezondheid, beleidsmakers en vertegenwoordigers van de gemeenschap. Wat dat betreft kunnen we leren van de Hygiënisten uit de 19e eeuw.

In hoofdstuk 2 wordt een raamwerk gepresenteerd voor de beoordeling van de gezondheidskundige betekenis van de effecten van blootstelling aan milieuverontreiniging, met name in relatie tot grote infrastructurele ingrepen in de omgeving, zoals de uitbreiding van een luchthaven. Verschillende definities van gezondheid komen aan bod, in het bijzonder met betrekking tot waar de grens tussen welbevinden en

gezondheid zou kunnen worden gelegd. Op basis van een conceptueel model wordt een uitgebreide en veelvormige reeks van indicatoren besproken uit de keten van blootstelling, lichaamsbelasting, intermediaire effecten -al dan niet van gezondheidskundige betekenis- tot aan wezenlijke aantasting van de gezondheid.

Op vier nuchtere manieren omgaan met risico's

In hoofdstuk 3 kijken we zorgvuldig naar een belangrijk instrument van collectieve gezondheidsbescherming: kwantitatieve risicoanalyse en risicomanagement. We stellen vast dat een 'fatsoenlijke' overheid in beginsel uitgaat van op rechten gebaseerde beslisregels, die een gelijk recht op een zekere mate van gezondheidsbescherming en zorg voor iedere burger garanderen. We laten echter ook zien dat een zuiver op rechten gebaseerd beleid in bepaalde gevallen tegen grenzen van doelmatigheid of zelfs betaalbaarheid oploopt. Vice versa, heeft het maximaliseren van de efficiëntie van risicobeleid onontkoombaar een invloed op de 'eerlijke' verdeling van bescherming tegen gezondheidsgevaren: kortom de klassieke tegenstelling tussen efficiëntie en billijkheid (rechtvaardigheid). Traditioneel is het Nederlandse milieubeleid niet in eerste instantie gericht op het evenwichtig afwegen van – enerzijds - marginale reducties in milieuverontreiniging en daarmee verbonden gezondheidswinsten tegen anderzijds de maatschappelijke kosten daarvan. In beginsel gaat het om de bescherming van de gezondheid van het meest gevoelige individu, in eerste instantie ongeacht de kosten.

We bespreken een aantal ontnuchterende, complicerende eigenschappen van het begrip gezondheidsrisico.

Het begrip risico bestaat niet uitsluitend uit objectief meetbare eigenschappen van systemen.

Risico is ook een sociaal construct, waarbij kwalitatieve, sociaal-psychologische aspecten de doorslag kunnen geven bij onze beoordeling ervan.

Er bestaat dan ook geen universele maat voor het kwantificeren van risico's; de keuze voor een maat impliceert altijd een keuze voor normatieve uitgangspunten, context en waarden.

Risicoschattingen zijn altijd in bepaalde mate onzeker; deze onzekerheid loopt evenwel sterk uiteen van gebrek aan exactheid via onbetrouwbaarheid en onbepaaldheid tot bijna absolute onwetendheid.

Het resultaat van kwantitatieve risicoanalyse hangt sterk af van de definitie van de systeemgrenzen

Risico's kunnen daarom niet zo maar vergeleken worden.

Risico's zijn echter wel aan de hand van een beperkte set criteria te karakteriseren; vaak worden de volgende criteria genoemd: 'waarschijnlijkheid', 'ernst en omvang van nadelige gevolgen', 'onzekerheid', 'alomtegenwoordigheid (schaal en ruimte en tijd)', 'persistentie', 'onomkeerbaarheid', 'latentietijd', '(on)billijkheid', 'vermogen om maatschappelijke onrust op te wekken'. Uiteraard zal de aard en de omvang van de set van risicoattributen van probleem tot probleem verschillen.

Om tegemoet te komen aan deze 'ontnuchterende' eigenschappen van het begrip risico, stellen we een typologie van risicoproblemen voor met bijbehorende procedures, veelal met een toenemende reikwijdte: 1) traditionele kwantitatieve risicoanalyse en –management, 2) doelmatig en proportioneel gebruik van schaarse middelen, 3) een benadering als risicorekensommen de kern van de zaak niet raken, en 4) een benadering bij grote onzekerheid en complexiteit. De procedures hebben betrekking op de beslisregels, de doelen van het risicobeleid, de typen van oplossingen, de betrokkenheid van belanghebbenden en de (wetenschappelijke) instrumenten.

Bij risicoproblemen met een geringe complexiteit en waar weinig onzekerheid in het geding is (vooral op statistisch niveau), voldoen de klassieke methoden van risicoanalyse en -beheersing (management). Risico's kunnen op gebruikelijke wijze worden gekwantificeerd in termen van kans maal effect en zoveel

mogelijk beoordeeld aan de hand van een standaard beoordelingskader, denk aan de nota 'Omgaan met Risico's'.

Een wetenschappelijke discussie blijft beperkt tot de deskundigen van betrokken adviserende, regelgevende instanties en ander betrokken partijen (bijvoorbeeld industrie). Op basis van ruwe analyse van kosteneffectiviteit kan worden vastgesteld of de risico-euro goed besteed is.

Het doel van de benadering is vooral de bescherming van de gezondheid van het meest gevoelige individu, vanuit het perspectief van eenieders recht op een hoog niveau van bescherming. Maatregelen zijn gericht op het terugdringen van de toegevoegde kans op ongezondheid (sterfte) tot een algemeen aanvaard niveau, bij voorkeur tegen redelijke kosten. Een belangrijk middel is de klassieke kwantitatieve risicoanalyse. Goede voorbeelden is de regelgeving voor chemische stoffen in de buitenlucht, op de werkplek of voor residuen of additieven in voedingsmiddelen.

In sommige gevallen zal de standaard benadering tegen grenzen aanlopen: de kosten zijn zeer hoog (totaal of per gewonnen levensjaar) of andere, hooggewaardeerde maatschappelijke belangen worden geschaad (denk aan legionellabacteriën in drinkwatersystemen, Schiphol, of het al dan niet sluiten van een kerncentrale). Dan zal, bij voorkeur in samenspraak met betrokken partijen (producenten, consumenten, overheid, bewonersgroepen), gezocht moeten worden naar optimalisering van het risicomanagement, bijvoorbeeld door de risicobenadering te richten op risicogroepen of welomschreven riskante omstandigheden, opdat een beter 'rendement' gehaald kan worden. Ook differentieren tussen *bestaande* en *nieuwe* situaties verhoogt vaak de doelmatigheid van risicoreductie. De discussie is hier uiteraard niet meer louter inhoudelijk, maar gaat ook over maatschappelijke keuzen, bijvoorbeeld tussen doelmatigheid en billijkheid. Het gelijke recht op bescherming wordt immers deels losgelaten. Gebaseerd op een goede analyse van risicoreductie, kosteneffectiviteit en verdelingseffecten zal met betrokken belangengroeperingen tot een breed gedragen compromis gekomen moeten worden.

In sommige gevallen raken de kwantitatieve risicoschattingen, uitgedrukt in extra sterfte of ziektekansen, domweg de kern van de zaak niet. Mensen voelen zich veel meer aangesproken door andere aspecten van het risicoprobleem, zoals de mogelijkheid dat alles uiteindelijk uit de hand loopt en er een rampzalige, onbeheersbare, onherstelbare situatie ontstaat bij een onbekende en dus onbeminde, nieuwe technologie als genetische modificatie of nanotechnologie. In die gevallen kan overleg met betrokken maatschappelijke groeperingen over de definitie van het risicoprobleem (waar gaat het eigenlijk om?), de te volgen procedure (b.v. toetsingscriteria), doelmatigheid en evenwichtigheid van maatregelen de kwaliteit van besluitvorming helpen en wellicht dure maatregelen overbodig maken. Hierbij kan men denken aan etiquettering, waarmee de burger zelf zijn of haar keuzes kan maken. Het doel is dan niet louter het terugbrengen van gezondheidsrisico's, maar evenzeer de doelmatige inzet van collectieve middelen, het winnen van vertrouwen, het versterken van de invloed van de consument, en soms ook welberedeneerde voorzorg.

Naarmate de onzekerheden toenemen, evenals de ernst en omvang, verschuift de nadruk naar een benadering voorbij de klassieke kwantitatieve methoden en in aanvulling daarop: een "post-normale" benadering, waarbij afbakening en structurering van het probleem, het kwantitatief in kaart brengen, het omgaan met onzekerheid en onwetendheid, het toepassen van principes als voorzorg, afwegingsinstrumentarium, en het ontwerp van de beslisprocedures in interactie met de samenleving moet worden vormgegeven. De rol van de wetenschap is dan veranderd van louter adviseur of rekenmeester naar facilitator. Het wetenschappelijke zit dan vooral in de hiervoor genoemde attributen als *"transparantie"*, *"systematische analyse"*, een *"sceptische houding"*, *"peer review"*, *"onafhankelijkheid"*, het *"rekenschap* kunnen *geven"*, en al doende *"leren"*. Dit impliceert tevens dat de politiek moet beslissen in expliciete

onzekerheid. Het doel is vooral te komen tot een breed gedragen definitie van het risicoprobleem, een wijze waarop het probleem 'gemeten' of de vinger aan de pols gehouden kan worden, en uiteindelijk een beslissingkader. Een sprekend voorbeeld hiervan is het werk van het Intergouvernementele Panel voor Klimaatverandering, waarbij naast wetenschappelijke analyse een duidelijke rol weggelegd is voor meer politiek-maatschappelijke evaluatie.

Een maat voor aan milieu toe te schrijven gezondheidsverlies

Traditioneel beoordelen beleidsmakers de milieukwaliteit aan de hand van gezondheidskundige advieswaarden, vaak gebaseerd op een zeer klein additioneel sterfterisico per jaar. Zo lang de concentraties van verontreinigende stoffen onder deze waarden bleven, kan de lucht veilig ingeademd worden, het water gedronken, of de grond bebouwd. Als concentraties boven de gezondheidskundige normwaarden dreigen uit te komen, komt de volksgezondheid in het geding en is het dus tijd om risicoreducerende maatregelen te overwegen. Het huidige epidemiologische inzicht verhoudt zich vaak slecht met dergelijke op normstelling gebaseerde regelgeving. Hetzij omdat een duidelijke indicatie voor een veilige drempel voor gezondheidseffecten ontbreekt bij realistische niveaus van blootstelling, hetzij omdat een scala aan (niet-letale) effecten op gezondheid of welzijn kan worden aangetoond, van mild en tijdelijk, tot ernstig en blijvend.

Om de invloed van milieuverontreiniging op de gezondheid vergelijkenderwijs te analyseren, hebben we de toepassing onderzocht van 'disability-adjusted life years' (DALYs), ofwel gezondheidsgewogen levensjaren, als een soort gezondheidspasmunt om allerlei typen van gezondheidseffecten van blootstelling aan milieufactoren onder één noemer te brengen. De DALY integreert drie belangrijke dimensies van volksgezondheid, namelijk verlies van *levensverwachting* (kwantiteit van leven), verlies van *kwaliteit* van leven en het *aantal* individuen dat gezondheidsschade ondervindt. Tijd is de meeteenheid: gezonde jaren worden verloren door vroegtijdige sterfte of door verlies van kwaliteit van leven, gemeten als voor de ernst van aandoeningen gedisconteerde levensjaren in een populatie. Voor de beleidsmaker zijn er zeker drie voordelen verbonden aan toepassing van dit soort geaggregeerde gezondheidsmaten. Het maakt vergelijkende risicoanalyse mogelijk vanuit het perspectief van de volksgezondheid (welke factoren veroorzaken de grootste ziektelast), alsook het beoordelen van de doelmatigheid van milieubeleid in termen van gezondheid (DALYs/Euro). Ook kunnen geaggregeerde maten van dienst zijn bij het in kaart brengen van ruimtelijke stapeling van verschillende gezondheidsschadelijke milieufactoren.

Gebruik makend van de gegevens van de Nationale Milieuverkenning 1997, hebben we in hoofdstuk 4 berekend dat de lange termijn effecten van deeltjesvormige luchtverontreiniging ('fijn stof') veruit de belangrijkste bron van aan milieu gerelateerde ziektelast is. Daarnaast blijkt het binnenmilieu in de woning een belangrijke bron van ziektelast (radon, vochtigheid, tweedehands tabaksrook). De oefening liet tevens zien dat omgevingsgeluid een belangrijke factor is, overigens sterk afhankelijk van de mate waarin men meer sociaal-psychologische eindpunten, zoals ernstige hinder of slaapverstoring tot het domein van gezondheid rekent. Enkele typische prioritaire milieuverontreinigende componenten, zoals kankerverwekkende stoffen in de buitenlucht blijken op dit moment nog maar weinig relevant voor de gezondheid.

In hoofdstuk 6 hebben we geprobeerd het gezondheidsverlies dat kan worden toegeschreven aan blootstelling aan omgevingsgeluid in kaart te brengen. Dat kan enerzijds door een schatting te maken van het aantal mensen dat (ernstig) gehinderd is of verstoring van de slaap ondervindt. Anderzijds kan men proberen de zwakke aanwijzingen die er zijn voor een mogelijke invloed van langdurige geluidbelasting op het optreden van hart- en vaataandoeningen te vertalen naar een jaarlijkse ziektelast (in DALYs). Hier stuitten we op een belangrijk dilemma. De aantallen mensen die ernstige hinder of slaapverstoring

ondervinden kan weliswaar met een redelijke mate van zekerheid geschat of gemeten. Echter, de gezondheidskundige betekenis hiervan is niet onomstreden: mag men ernstige hinder of verstoring van de slaap uitdrukken in voor verminderde gezondheid gewogen jaren (DALYs)? De gezondheidkundige betekenis van een toename van cardiovasculaire aandoeningen daarentegen is onmiskenbaar; echter de epidemiologische aanwijzingen voor een relatie tussen chronische blootstelling aan geluid en het optreden van hart- en vaataandoeningen zijn (nog) erg zwak en soms tegenstrijdig. Mocht geluidbelasting daadwerkelijk van invloed zijn op het optreden van hart- en vaataandoeningen, dan berekenen we een klein, maar significant aan geluidblootstelling toe te schrijven, aandeel van minder dan 2%.

Ondanks vele methodologische en soms zelfs ethische problemen lijken gezondheidsgewogen levensjaren (zoals DALYs) een veelbelovend kader voor een expliciete beoordeling van het gezondheidsverlies dat aan milieuverontreiniging kan worden toegeschreven. Zeker als men bedenkt dat bestaande, meer geaccepteerde manieren om gezondheidsrisico's te meten, zoals additionele sterftekans per jaar van blootstelling of overschrijding van gezondheidskundige advieswaarden vaak dezelfde impliciete tekortkomingen hebben. Met name geldt dat de in de rekensommen verborgen niet-wetenschappelijke waarden (subjectieve oordelen). Het afwijzen van dit soort geïntegreerde volksgezondheidsmaten vanwege in de rekensommen 'verborgen waarden' impliceert in de praktijk dan ook het afwijzen ven elke soort van vergelijkende risicoanalyse.

In Hoofdstuk 5 wordt een onderzoek beschreven naar de bruikbaarheid van drie benaderingen om het gezondheidsverlies verbonden met blootstelling aan luchtverontreiniging kwantitatief in kaart te brengen: 1) attributieve *sterfte* (jaarlijkse toewijsbare mortaliteit), 2) attributieve *ziektelast* (uitgedrukt in jaarlijks verlies van gezondheidsgewogen jaren: DALYs) en 3) schattingen van de geldwaarde van de attributieve gezondheidschade ('willingness to pay'). We hebben de gezondheidseffecten van de belangrijkste verschijnselen in Nederland geanalyseerd: 'fijn stof' en ozon in de buitenlucht, radon, omgevingstabaksrook en vocht binnenshuis. De lange termijn effecten van 'fijn stof' scoren op alle gezondheidsindicatoren het hoogst; de rangorde voor de overige vormen van luchtverontreiniging wisselt per benadering: jaarlijkse sterfte, ziektelast of in geld uitgedrukt gezondheidsverlies (Euro).

Hoofdstuk 7 beschrijft een studie waarin voor de OECD-landen robuuste schattingen zijn gemaakt van het aan milieufactoren toe te schrijven aandeel van de ziektelast voor de belangrijkste ziektecategorieën (op basis van de 'Global Burden of Disease'-schattingen van de Wereldgezondheidsorganisatie). Naast schattingen in termen van 'gezondheidsgewogen levensjaren' (DALYs), hebben we aan milieufactoren gerelateerd gezondheidsverlies geprobeerd uit te drukken in monetaire indicatoren: Bruto Nationaal Product per capita, schattingen van 'bereidheid tot betalen' om sterfterisico's te vermijden voor mortaliteit, respectievelijk schattingen van kosten van ziekte ('Costs of Illness') voor morbiditeit. Schattingen van op deze wijze in geld uitgedrukt gezondheidsverlies ten gevolge van milieuverontreiniging in de rijke OECD-landen lopen uiteen van 150 tot 500 US$ per inwoner per jaar.

Gezondheid als economische waarde

Een van de meest aansprekende voordelen van de toepassing van geïntegreerde gezondheidsmaten als de DALYs is de mogelijkheid die ze bieden om vergelijkenderwijs de kosteneffectiviteit van interventies te analyseren. De volgende logische stap in de vergelijkende analyse van gezondheidseffecten van milieu is uiteraard het op een vergelijkbare manier in verband brengen van gezondheidswinsten van milieuinterventies met de noodzakelijke investeringen. Middelen zijn vaak schaars, dus wat is de beste deal in risicomanagement? Bovendien kan de overheid met het expliciet en vergelijkbaar beoordelen van gezondheidsopbrengsten en beleidskosten beter rekenschap afleggen van haar beleid.

In hoofdstuk 3 werd aangegeven dat de kosten per gewonnen DALY zeer sterk uiteen kunnen lopen in de verschillende domeinen van de volksgezondheid. Vooral milieu- en voedingsbeleid is soms kostbaar per gewonnen levensjaar, mede als gevolg van een op rechten gebaseerde traditie van risicobeleid. In hoofdstuk 5 hebben we laten zien dat ruwe berekeningen van de gezondheidsopbrengsten uitwijzen dat het beleid voor de bestrijding van luchtverontreiniging van de afgelopen decennia redelijk kosteneffectief is geweest, zelfs zonder de waarde van technologische innovaties die het heeft gestimuleerd mee te nemen.

Naast de analyse van *kosteneffectiviteit*, waarbij opbrengsten in gezondheidseindpunten als DALYs worden gemeten, is *kosten-batenanalyse* een vorm van evaluatieonderzoek waarbij naast de kosten ook de opbrengsten van gezondheidszorg in geld worden uitgedrukt. Door kosten en opbrengsten onder één noemer te brengen, namelijk geld, zijn efficiëntiesommen gemakkelijker te maken. Aan de opbrengstenkant kunnen op deze manier ook aspecten die niet op de gezondheid betrekking hebben, worden meegenomen. In beginsel kunnen daarmee investeringen in het gezondheidsdomein vergeleken worden met investeringen daarbuiten, bijvoorbeeld verkeersveiligheid, sociale zekerheid of ecologie. Bij deze vorm van analyse ontkomen we er uiteraard niet aan om gezondheidsopbrengsten, zoals statistische levensjaren of kwaliteit van leven, in geld uit te drukken.

De berekeningen van verlies aan gezondheidskapitaal door milieuverontreiniging in hoofdstuk 5 en 7 geven aan dat er goede mogelijkheden zijn voor efficiënte investeringen in milieukwaliteit vanuit volksgezondheidsperspectief. Echter, vooral met betrekking tot het geldelijk waarderen van gezondheidsverliezen, ontbreekt nog veel inzicht. In de in hoofdstuk 7 beschreven studie voor de OECD hebben we bruto nationaal product per capita, 'willingness to pay'-schattingen uit de literatuur en kosten van ziekten per brede ziektecategorie gebruikt als een grove, eerste benadering van de monetaire waarde van aan milieuverontreiniging gerelateerde sterfte en ziekte. Het is duidelijk dat meer verfijnde, op de lokale condities afgestemde, methodologische degelijke studies nodig zijn om de geloofwaardigheid van dit soort kosten-batenanalyses te vergroten. Tevens dient de betrouwbaarheid en transparantie te worden vergroot door systematische peer review, alsmede de betrokkenheid van belanghebbende bij de definitie van het kader waarbinnen analyses kosteneffectiviteit of kostenbaten van milieubeleid worden uitgevoerd.

Tenslotte: aantallen doden, DALYs of Euro's?

Het kiezen van de gezondheidsmaat luistert nauw. Zelfs bij het meten van sterfte, zonder twijfel het meest eenduidige gezondheidseffect dat we kennen, moeten keuzen gemaakt worden, met uitgesproken normatieve consequenties. De aan een bepaalde riskante activiteit of situatie verbonden gezondheidsgevaren kan men onder andere meten als sterfte per jaar, als jaarlijks verlies aan levensverwachting of voor gezondheid gewogen levensverwachting. Sterfte per jaar is de meest simpele maat en garandeert in ieder geval dat eenieder, jong of oud, rijk of arm, gezond of ziek, gelijk is en gelijke bescherming krijgt. Die keuze betekent echter wel dat het met enkele dagen vervroegde overlijden van een ernstig zieke, oudere patiënt tijdens een episode van smog gelijkwaardig is aan het overlijden van een jonge vader met kinderen door een ongeval. Kijkt men daarentegen naar verloren levensverwachting als maat voor risico, zou men er in beginsel baat bij hebben het risico vooral af te wentelen op de ouderen, aangezien zij de minste levensverwachting te verliezen hebben: uit utilitair oogpunt wellicht te verdedigen, maar niet uit oogpunt van sociale rechtvaardigheid. Het uitdrukken van de schade in 'gezondheidgewogen' levensjaren heeft het voordeel dat ook niet-dodelijke schade kan worden meegenomen, maar betekent tegelijkertijd impliciet dat zieke mensen minder tellen dan gezonde. Drukt men de gezondheidsschade uit in geld, dan zou iemand met een hoger inkomen in beginsel meer meetellen dan iemand met een laag

inkomen. Iemand met een hoger inkomen heeft immers meer te besteden om in zijn of haar individuele behoeften te voorzien. Kortom, zelfs zeer neutraal ogende risicomaten zijn 'geladen' met waardeoordelen, of we dat nu willen of niet. Er is geen universeel geldende risicomaat; de keuze voor een risicomaat is een afgeleide van de normatieve uitgangspunten die we bij het beheersen van een risico wensen te hanteren.

De effecten van milieuverontreiniging op de gezondheid kunnen in aantallen slachtoffers, DALYs of Euro's worden geteld, als men zich maar goed rekenschap heeft gegeven van de normatieve implicaties: Wat zijn precies de beschermingsdoelen van het beleid, de politiek of het publiek?

Nawoord

Ik ben dank verschuldigd aan alle mensen met wie ik bij of via de Gezondheidsraad en het RIVM heb gewerkt aan onderwerpen op het gebied van gezondheid en milieu. In het bijzonder bedank ik mijn medeauteurs Pieter Bol, universitair hoofddocent Algemene hygiëne en epidemiologie aan de Technische Universiteit Delft, Aldert Hanemaaijer en Johan Melse van het Milieu- en Natuurplanbureau, Erik Lebret, Elise van Kempen, en Brigit Staatsen van het Centrum voor Milieu-Gezondheid Onderzoek en Pieter Kramers van het Centrum voor Volksgezondheid Toekomst Verkenningen. Belangrijk voor dit proefschrift waren ook Wim Passchier, mijn 'mentor' en medesecretaris van de Risicocommissie van de Raad en Fred Langeweg die ons figuurlijk het mes op de keel zette om nu eens eindelijk met getallen te komen over het aandeel milieu in de ziektelast. En natuurlijk ben ik mijn promotor Bert Brunekreef dankbaar dat hij mij die hoogstnoodzakelijke laatste zet heeft gegeven. Meer namen noem ik niet, opdat ik niemand vergeet.

Curriculum vitae

Ik ben geboren op maandag 15 juli 1957 te Uithoorn. Na mijn diploma Atheneum B gehaald te hebben aan het Alkwin College in Uithoorn, begon ik in 1975 een studie biologie aan de Universiteit van Amsterdam. In juni 1983 studeerde ik af met een breed pakket, variërend van Bijzondere plantkunde, ecologie en vegetatiekunde, Voedingsleer en toxicologie tot aan Wetenschapsfilosofie. Mijn wetenschapsfilosofische interesse hield vooral verband met de sterke behoefte aan meer maatschappelijke relevant onderzoek die we destijds als student uitdroegen.

Na een jaar van reizen en vrijwilligerswerk (o.a. bij het Voedingsinstituut TNO), begon ik juni 1984 als junior wetenschappelijk secretaris bij de Gezondheidsraad in Den Haag. In dienst van verschillende commissies schreef ik een flink aantal adviezen over kankerverwekkende en andere gevaarlijke stoffen in het milieu, luchtverontreiniging buiten en binnenshuis ('passief roken'), de kwaliteit van de lokale leefomgeving, hygiëne in zwem- en therapiebaden, en over het schatten en 'managen' van milieurisico's (van toxische stoffen tot ontplofbare installaties).

Inmiddels senior-secretaris van de Beraadsgroep Gezondheid en Omgeving, verruilde ik eind 1999 toch de Haagse Gezondheidsraad voor het Rijksinstituut voor Volksgezondheid en Milieuhygiëne (RIVM). In eerste instantie kwam ik terecht bij de afdeling Milieu-epidemiologie van het Centrum voor Chronische Ziekten en Milieu-epidemiologie. Dat bevond zich toen nog in het centrum van Utrecht, hetgeen de overstap extra aantrekkelijk maakte. Als senioronderzoeker 'milieu en gezondheid' was ik was betrokken bij een reeks Milieuverkenningen en Milieubalansen. Tevens trok ik verschillende projecten voor het schatten van gezondheidrisico's. Na een korte periode als projectleider van het 'modellenbureau' Chronische ziekten van het Centrum voor Chronische Ziekten Epidemiologie vertrok ik naar het Milieu en Natuurplanbureau waar ik bijna drie jaar werkte als programmacoördinator Lokale leefomgeving en leefbaarheid.

Sinds juni 2002 werk ik bij het centrum voor Volksgezondheid Toekomst Verkenning, eerst als lid van het redactieteam voor het Samenvattend rapport Volksgezondheid Toekomst Verkenning 2002, inmiddels als projectleider voor het Samenvattend rapport VTV2006.