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

Augustinus E.M. de Hollander, Johan M. Melse, Erik Lebret, Pieter G.N. Kramers

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

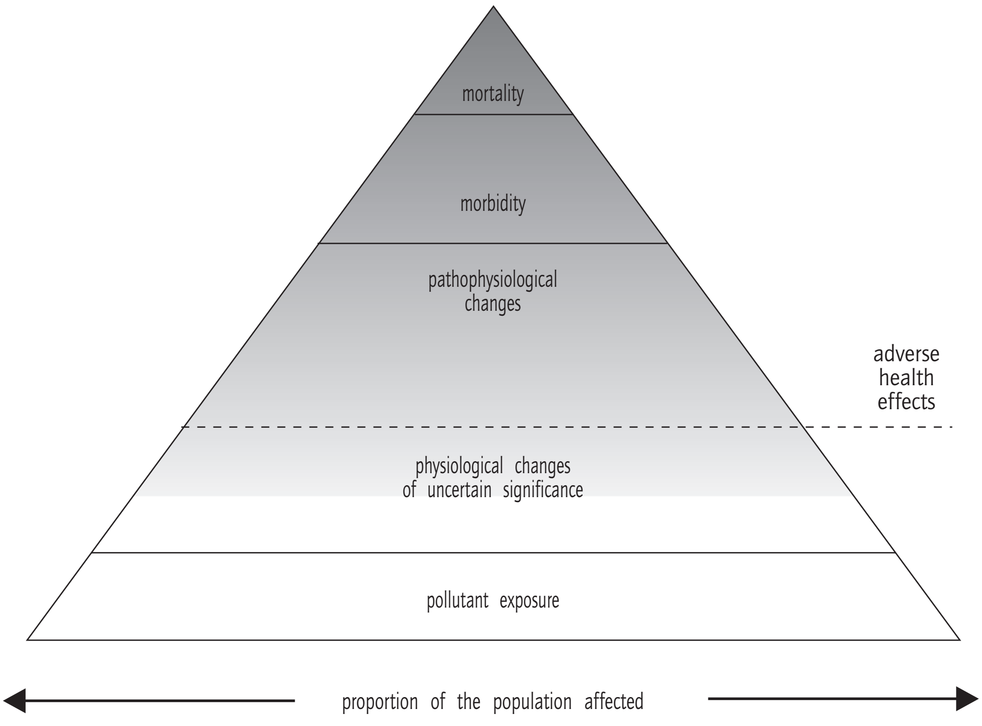

Figure 4-1. Schematic representation of the distribution of air pollution responses in the population[6].

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	*neuro-cognitive development*					
(drinking-water pipes)	*(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

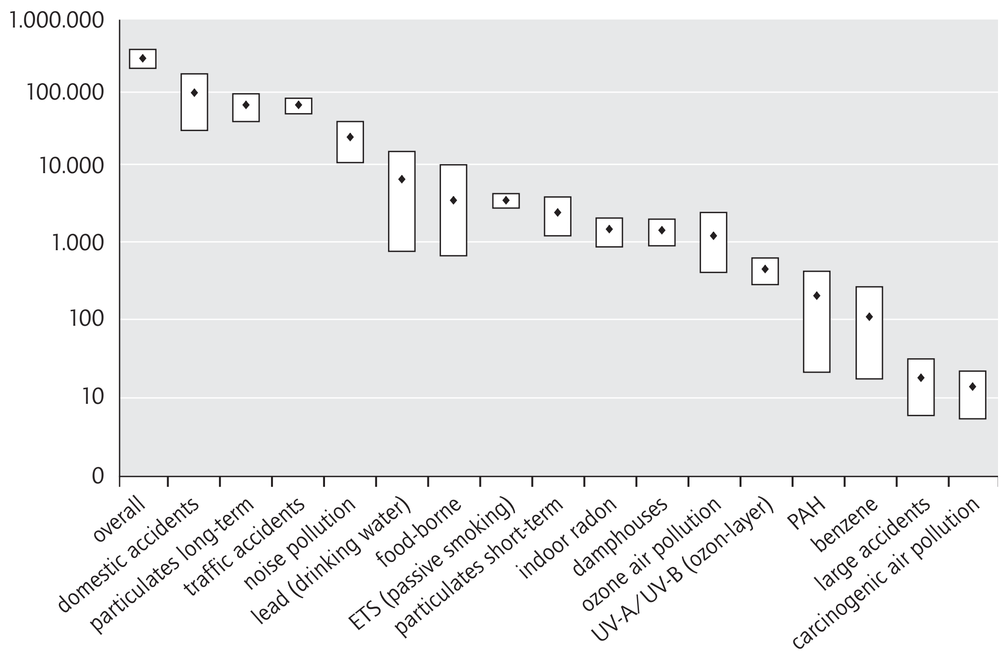

Figure 4-2. Annual health loss in disability-adjusted life-years' for selected environmental exposures in the Netherlands; point estimates and 5 and 95-percentiles of probability interval.

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

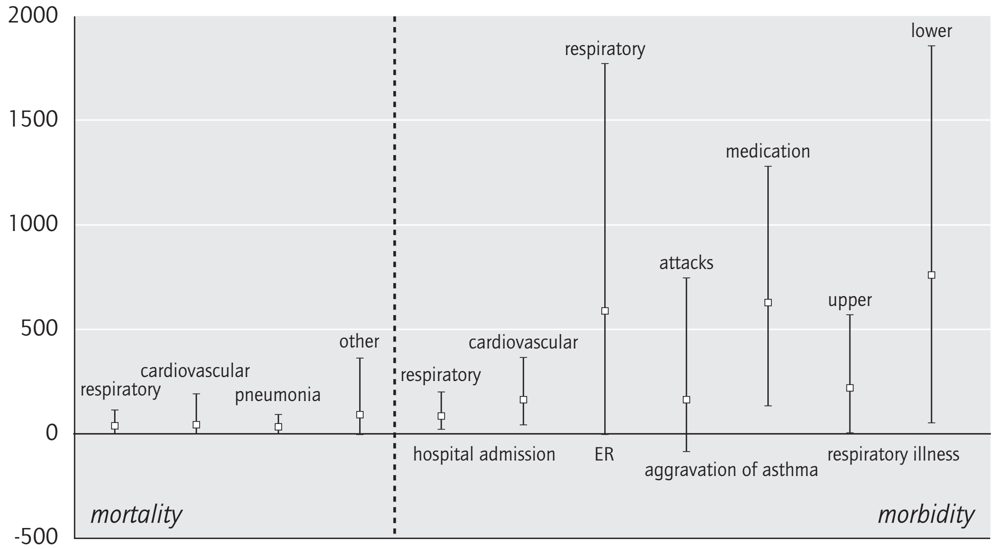

Figure 4-3. Annual health loss in 'disability-adjusted life-years' due to several short-term endpoints associated with exposure to particulates in the Netherlands; point-estimates and 5 and 95-percentiles of probability interval.

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

1 Shore RE. Epidemiologic data in risk assessment - imperfect but valuable. Am J Public Health 1995; 85: 474-5.

2 Hertz-Picciotto I. Epidemiology and quantitative risk assessment: a bridge from science to policy. Am J Public Health 1995; 484-91.

3 World Health Organization. Concern for Europe's Tomorrow: health and the environment in the WHO European Region. Copenhagen: WHO European Center for Environment and Health. Stuttgart: Wiss Verl-Ges, 1994.

4 Brunekreef B, Dockery DW, Krzyzanowski M. Epidemiological studies on the short-term effects of low levels of major air pollution components. Environ Health Perspect 1995; 103(S): 3-13.

5 Brunekreef B, Holgate ST. Air pollution and health. Review. Lancet 2002; 360: 1233-42.

6 Lipfert FW. Air pollution and human health: perspectives for the '90s and beyond. Risk Analysis 1997; 17: 137-46.

7 American Thorax Society. Guidelines as to what constitutes an adverse respiratory health effect, with special reference to epidemiological studies on air pollution. Am Rev Respir Dis 1985; 131: 666-8.

8 American Thorax Society. What constitutes an adverse health effect of air pollution? Am J Respir Crit Care Med 2000; 161: 665-73.

9 Covello VT, Merkhofer MW. Risk assessment methods. Approaches for assessing health and environmental risks. New York: Plenum Press, 1993.

10 Morgan MG. Risk analysis and management. Scientific American 1993, July: 24-30.

11 Sexton K. Science and policy in regulatory decision making: getting the facts right about hazardous air pollutants. Environ Health Perspect 1995; 102(suppl6): 213-22.

12 Vlek CAJ. Understanding, accepting and controlling risks: a multistage framework for risk communication. Eur Rev Appl Psychol 1995; 45: 49-54.

13 Presidential/Congressional Commission on Risk Assessment and Risk Management. Risk Management and Regulatory Decision-Making. Chapter 3; Final Report Volume 2. Washington DC, 1997: 39-49.

14 National Institute of Public Health and the Environment. Nationale Milieuverkenning 1997-2020 (National Environmental Outlook 1997-2020, backgrounds). Alphen a/d Rijn: Samsom HD Tjeenk Willink bv, 1997.

15 National Institute of Public Health and the Environment. The National Environmental Forecast Report 5, 2000-2030. Alphen a/d/ Rijn: Samson H.D. Tjeenk Willink bv, 2000 [in Dutch].

16 Ministry of Housing, Physical Planning and Environmental Protection. Premises for Risk Management (annex to the Netherlands National Environmental Policy Plan 1990-1994). The Hague: Ministry of VROM, 1989.

17 World Health Organization. Air quality guidelines for Europe. Copenhagen: WHO Regional Office for Europe; Regional Publications, European Series no 23, 1987.

18 National Research Council. Risk assessment in the federal government: Managing the process. Washington DC: National Academy Press, 1983.

19 Health Council of the Netherlands: Committee on Risk Measures and Risk Assessment. Risk is more than just a number. The Hague: Health Council of the Netherlands, 1996; publication no. 1996/03E.

20 Vedal S. Ambient particles and health: lines that divide. J Air Waste Manag Assoc 1997 47: 551-81.

21 Brunekreef B. Air pollution and life expectancy: is there a relation? Occup Environ Med 1997; 54: 781-4.

22 Zanobetti A, Schwartz J, Samoli E, et al.. The temporal pattern of mortality responses to air pollution: a multi-city assessment of mortality displacement. Epidemiol 2002; 13: 87-93.

23 Zeger SL, Dominici F, Samet J. Harvesting-resistant estimates of air pollution effects of mortality. Epidemiol 1999; 10: 171-75.

24 Kunzli N, Medina S, Kaiser R, Quenel P, Horak FJr, Studnicka M. Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? Am J Epidemiol 2001; 153(11): 1050-5.

25 Ten Berge WF, Stallen PJM. How to compare risk assessments for accidental and chronic exposure. Risk Analysis 1995; 15: 111-3.

26 World Bank. World Development Report 1993: Investing in Health – world development indicators. New York: Oxford University Press, 1993.

27 Ruwaard D, Kramers PGN, van den Berg Jeths A, Achterberg PW. Public Health Status and Forecasts, the health status of the Dutch population over the period 1950-2010. The Hague: SDU Uitgeverij Plantijnstraat, 1994.

28 Olshanski SJ, Rudberg MA, Carnes BA, Cassel CK, Brody JA. Trading of longer life for worsening health. J Aging Health 1991; 3: 194-216.

29 National Institute of Public health and the Environment. Milieubalans 1995, het Nederlandse milieu verklaard. (the environment, the Balance for 1995). Alphen a/d Rijn: Samsom HD Tjeenk Willink bv, 1995.

[30] 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.

[31] Salazar-Lindo E, Alegre M, Carrión P, Razetto N. The Peruvian cholera epidemic and the role of chlorination in its control and prevention. First International Conference on the Safety of Water Disinfection: balancing Chemical and Microbial Risks. Washington DC; August 31-September 3, 1992.

[32] Neutra RR, Ostro B. An evaluation of the role of epidemiology in assessing current and future disinfection technologies for drinking water. Sci Total Environ 1993; 127: 91-138.

[33] Morris RD. Drinking water and cancer. Environ Health Perspect 1995; 103 (Suppl. 8): 225-31.

[34] Cantor KP, Lynch CF, Hildesheim ME, Dosemeci M, Lubin J, Alavanja M, Craun G. Drinking water source and chlorination byproducts. I. Risk of bladder cancer. Epidemiology 1998; 9(1): 21-8.

[35] Hildesheim ME, Cantor KP, Lynch CF, Dosemeci M, Lubin J, Alavanja M, Craun G. Drinking water source and chlorination byproducts. II. Risk of colon and rectal cancers. Epidemiology 1998; 9(1): 29-35.

[36] Ruwaard D, Kramers PGN (eds). Public Health Status and Forecasts 1997. Health, prevention and health care in the Netherlands until 2015. Bilthoven/Maarssen, the Netherlands: National Institute of Public Health and the Environment, Elsevier/de Tijdstroom, 1998.

[37] Fischhoff B, Bostrom A, Quadrel MJ. Risk perception and communication. Ann Rev Public Health 1993; 14: 183-203.

[38] Fischhoff-B. Risk perception and communication unplugged: twenty years of process. Risk Anal 1995; 5: 137-45.

[39] AEM de Hollander, JM Melse, EEMM van Kempen, A Dusseldorp, E Lebret. Calculating the annual number of disability adjusted life-years (DALYs) lost to environmental exposures; the example of the Netherlands. Bilthoven: RIVM, 1999.

[40] Murray CJL, Lopez AD (eds). The global burden of disease; a comprehensive assessment of mortality and disability from disease, injury, and risk factors in 1990 and projected to 2020. Global burden of disease and injury series, volume I. Harvard University Press, 1996.

[41] Murray CJ, Lopez AD. Regional patterns of disability-free life expectancy and disability-adjusted life expectancy: global Burden of Disease Study. Lancet. 1997; 349(9062): 1347-52.

[42] Stouthard MEA, Essink-Bot ML, Bonsel GJ, Barendregt JJ, Kramers PGN, van de Water HPA, Gunning-Schepers LJ, van der Maas. Disability weights for diseases in the Netherlands. Rotterdam: Erasmus University Rotterdam, Dept Public Health, 1997.

[43] Essink-Bot M-L, Pereira J, Paxcker C, Schwarzinger M, Burström K, the European Disability Weights Group. Cross National comparability of burden of disease estimates: the European Disability Weights Project. Bull World Health Organisation 2002; 80: 644-52.

[44] Goerdt A, Koplan JP, Robine JM, Thuriaux MC, van Ginneken JK. Non-fatal Health Outcomes: concepts, instruments and indicators. In: Murray CJL, Lopez AD (eds). The global burden of disease; a comprehensive assessment of mortality and disability from disease, injury, and risk factors in 1990 and projected to 2020. Global burden of disease and injury series, volume I. Harvard University Press, 1996.

[45] Froberg DG, Kane RL. Methodology for measuring health-state preferences I-IV. J Clin Epidemiol 1989;42: 345-54, 459-71, 585-92, 675-85.

[46] Committee on Medical Cure and Care. Report on choices in medical cure and care. The Hague: Ministry of Welfare, Health and Culture, 1991.

[47] Murray CJL. Rethinking DALYs. In: Murray CJL, Lopez AD (eds). The global burden of disease; a comprehensive assessment of mortality and disability from disease, injury, and risk factors in 1990 and projected to 2020. Global burden of disease and injury series, volume I. Harvard University Press, 1996.

[48] Essink-Bot ML. Health status as a measure of outcome of disease treatment. Erasmus University Rotterdam (Thesis), 1995.

[49] Twickel-group. Quantitative assessment of health effects associated with exposure to particulate air pollution in the Netherlands. Bilthoven: National Institute of Public Health and the Environment, RIVM-report 623710002, 1995.

[50] Passchier-Vermeer W. Noise and Health. The Hague: Health Council of the Netherlands; publication no A93/02E, 1993.

[51] Krzyzanowski M. Methods for assessing the extent of exposure and effects of air pollution. Occup Environ Med; 1997: 145-51.

[52] Melse JM, de Hollander AEM. Health impact assessment of environmental benzene exposure, assumptions and science policy. Am J Epidemiol 1996; 7: S48.

[53] Slaper H, Velders GJM, Daniel JS, Gruijl FR de, Leun JC van der. Estimations of ozone depletion and skin cancer incidence to examine the Vienna Convention achievements. Nature 1996; 384: 256-8.

[54] Leenhouts HP, Stoop P, van Tuinen S. Non-nuclear industries in the Netherlands and radiological risks. Bilthoven: National Institute of Public health and the Environment, report 610053003, 1994.

[55] Ale BJM, Janssen MPM, Pruppers MJM (eds). Book of papers RISK 97: International conference on Mapping Environmental Risks and Risk Comparison, Amsterdam, October 21-24, 1997. Bilthoven: National Institute of Public Health and the Environment, 1997.

[56] Ryan PB. An overview of human exposure modeling. J Expo Environ Epidemiol 1992; 1: 453-73.

[57] Eerens HC, Sliggers CJ, van den Hout KD. The CAR model: the Dutch method to determine city street air quality. Atmos Environ 1993; 27b: 389-99.

[58] Hollander AEM de, EA Preller, S Heisterkamp. Hospital admission due to summer smog: a Bayesian approach to meta-regression analysis. Annual Scientific Report, Bilthoven: National Institute of Public health and the Environment, 1997.

[59] Melse JM, Kramers PGN. A national burden of disease calculation: Dutch DALY's. Bilthoven: RIVM, report no. 431501028, 1998.

[60] Burmaster DE, Anderson PD. Principles of good practice for the use of Monte Carlo techniques in human health and ecological risk assessment. Risk Analysis 1994; 14: 477-81.

[61] Hoffman FO, Hammonds JS. Propagation of uncertainty in risk assessments: the need to distinguish between uncertainty due to lack of knowledge and uncertainty due to variability. Risk Analysis 1994; 14: 707-12.

[62] Thompson KM, Burmaster DE, Crouch EA. Monte Carlo techniques for quantitative uncertainty analysis in public health risk assessments. Risk Analysis 1992; 12: 53-63.

[63] Dockery DW, Pope III CA, XU X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE. An association between air pollution and mortality in six US cities. N Engl J Med 1993; 340: 1010-4.

[64] Pope III CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Health CW. Particulate air pollution as a predictor of mortality in a prospective study of US adults. Am J Respir Crit Care Med 1995; 151: 669-74.

[65] CMEAP. Non-biological particles and Health. London: HSMO Committee on the Medical Effects of Air Pollution, 1995.

[66] USEPA. Air quality guidelines for particulate matter. Washington DC: US Environmental Protection Agency, 1996.

[67] World health Organization: International Program on Chemical Safety. Assessing human health risks of chemicals: derivation of guidance values for health-based exposure limits. Geneva: WHO, Environmental Health Criteria 170, 1994.

[68] Ames BN, Gold LS. The causes and prevention of cancer: gaining perspective. Environ Health Perspect 1997; 105(S): 865-73.

[69] Nord E. Methods for quality adjustment of life years. Soc Sci Med 1992; 34: 559-69.

[70] Zielhuis RL, Wibowo AAE. Standard setting in occupational health: philosophical issues. Am J Ind Med 1989; 16: 569-598.

[71] Johnson BB, Slovic P. Presenting uncertainty in health risk assessment: initial studies of its effects on risk perception and trust. Risk-Analysis 1995; 15: 485-94.

[72] Rhomberg L. Beyond screening: problems and prospects for risk characterization of endocrine disrupters. Regul Toxicol Pharmacol 1997 ; 26: 74-9.

[73] Advisory Committee on Dangerous Pathogens (ACDP). Seminar on microbiological risk assessment, 28 January 1997. J Appl Microbiol 1997 ; 83: 659-64.