An Aggregate Public Health Indicator to Represent the Impact of Multiple Environmental Exposures

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

We present a framework to aggregate divergent health impacts associated with different types of environmental exposures, such as air pollution, residential noise, and large technologic risks. From the policy maker's point of view, there are at least three good reasons for this type of aggregation: comparative risk evaluation (for example, 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: *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. Severity weights (0 for perfect health, 1 for death) are assigned to discount the time spent with conditions associated with environmental exposures. We combined information on population exposure distribution, exposure response relations, incidence, and prevalence rates to estimate annual numbers of people affected and the duration of the condition, including premature death. 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 as 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 more than 3%. The contribution of this set of environmental exposures to the total annual burden of disease in the Netherlands is less than 5%. (Epidemiology 1999;10:606–617)

Keywords: health impact assessment, environmental exposure, disability-adjusted life year (DALY).

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, immunosuppression, severe noise annoyance, and associated sleep disturbance.[1–3] During air pollution episodes, well studied human responses range from slight, reversible lung function deficits in virtually everyone exposed, to aggravation of symptoms among those with asthma, and from hospital admission of patients with cardiopulmonary disease to the premature death of some of the very weak[4–6] (see Figure 1[7]).

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.[8] Probabilistic risk measures, such as the annual mortality risk associated with certain exposures, appear unambiguous and easy to comprehend. Therefore, these measures are often applied as the most suitable criterion in the risk-management process.[9] 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.[10–13]

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 the framework of the Fourth Dutch National Environmental Outlook, which was recently published. These outlooks are produced every 4 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 ecologic 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]

From the point of view of policy makers, for which these outlooks are produced in the first place, an aggre-

From the Department of Chronic Diseases and Environmental Epidemiology, National Institute of Public Health and the Environment, P.O. Box 1, 3720 BA Bilthoven, the Netherlands.

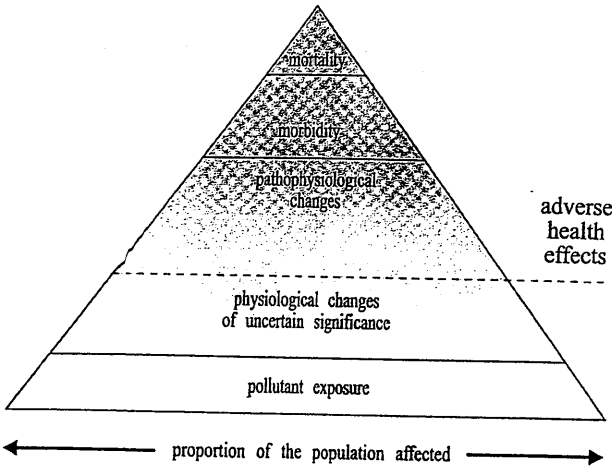

FIGURE 1. Schematic representation of the distribution of air pollution responses in the population.[6]

gate 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 with 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?"); and
- Improve risk communication.

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.[15–17] However, gradually it has become clear that the one "annual ten to the minus six" risk may differ substantially from the other in the following important aspects:[18]

- *Life expectancy*. "Precipitated" mortality during particulate air pollution episodes involving predominantly the old and weak may cost several months of unhealthy life at the most,[19,20] whereas the impact associated with fatal accidents involving individuals with a "random" age distribution may amount to a loss of many healthy years.[21]
- *Nonlethal health outcomes*. In Western society, public health focus has gradually changed from life expectancy to health expectancy, *ie*, 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.[22–24] 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 preexisting disease symptoms.[25] This implies that mortality risk might often not be the most appropriate indicator of environmental risk.

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 that are generated as a byproduct of measures to mitigate the original health risk. An elegant example of this kind of dilemma 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 of drinking water might increase the consumer's risk of cancer. Some of the byproducts appear to be mutagenic; moreover, weak but fairly consistent indications for some carcinogenic potency have emerged from epidemiologic 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.[26–30] Of course, the risk-management process would have to consider the validity of both risk assessments as well, but this is not the issue here.

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.[31] Spatial clustering of societal functions, such as housing, work, transportation and recreation, combined with unfavorable developments in urbanization (for example, socioeconomic segregation) have led to the accumulation of health risk factors in certain neighborhoods. Among these are pollution of air and soils, noise and odor pollution, traffic congestion, and bad housing.[14] An aggregate measure may facilitate explicit evaluation and comparison of environmental conditions across different geographical locations.

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 judgments may be largely due to differences in conceptualization and definition of risk problems.[9,32] In the words of Fischhoff, in an article 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."[33] Risk com-

munication should be an interactive, two-way process, taking account of existing audience knowledge, interests, and behavior. 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.[34]

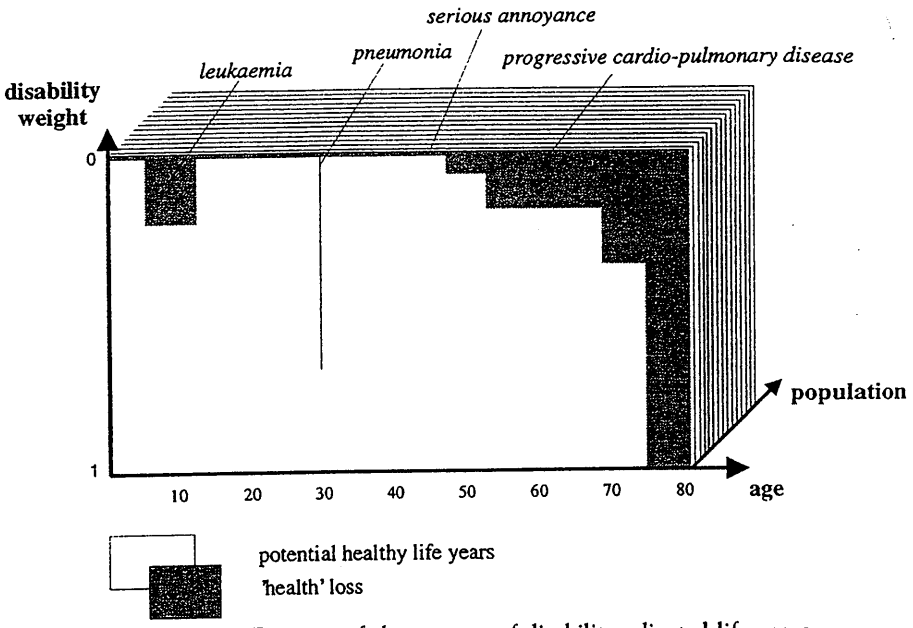

FIGURE 2. Diagram of the concept of disability-adjusted life years.

Concepts and Methods

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.[35] 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 standardized by means of severity weights.[22,35] Our adaptation of the DALY concept was inspired by the notion that the multiform health loss due to environmental exposure is fairly well characterized by three dominant aspects of public health: *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 2). On the basis of this concept, health loss attributable to environmental exposures can be assessed by the following:

- Defining responses that 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);
- Attributing discount weights to the unfavorable health conditions (S); and
- Calculating the annual number of DALYs lost, using the equation: $DALY_{exp} = N \times D \times S$.

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[36] and the Dutch Burden of Disease project. The latter project started in 1996 and was recently reported.[31,37]

A 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 and from region to region, because it reflects changes or differences in social and cultural beliefs, in medical technology, and in economic conditions. Already, in 1946, the founding charter of the World Health Organization stated that health is not "merely the absence of disease and infirmity." An individual's capability to function well physically and mentally, as well as socially, is the central issue in most papers on health-status measurements.[38,39] The Committee on Medical Cure and Care referred to health as the ability to cope with the demands of everyday life.[40]

Initially, Murray and coworkers applied disability weight definitions that were primarily based on functionality, the lack of ability to perform "activities of everyday life" in four domains: procreation, occupation, education, and recreation.[22] The approach was received with a fair amount of criticism, some focused on the

TABLE 1. Revised Disability Classes: Indicator Conditions and Severity Weights for the Global Burden of Disease Project[41]

Class	Indicator Conditions	Severity Weight
1	Vitiligo on face, weight-for-height less than 2 SDs	0.00–0.02
2	Watery diarrhea, 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

procedures of attributing weights and others on 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 and Lopez[36] applied the concept of "indicator conditions." Using formal instruments to measure health preferences, 22 indicator conditions were given weights in a series of consensus meetings involving physicians and public health scientists from different regions. These states reflected several distinct attributes of nonfatal health outcomes, such as large physical manifestations or limitations, psychological and social limitations, pain, and disturbed sexual and reproductive functions. These indicator conditions were used subsequently to attribute disability weights to most other states (see Table 1).

To estimate the burden of disease for the Dutch population, Stouthard *et al*[37] selected 52 diagnoses of greatest public health significance in terms of number of patients and years of (healthy) life lost. These diagnoses were divided in 175 more or less homogeneous health states of various levels of severity (and/or progression).[37]

According to the protocol designed by Murray *et al*,[41] 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-tradeoff approach. A visual analogue scale was added as another instrument of valuation, mainly for the purpose of validation. Furthermore, a standardized classification of the indicator states according to EuroQol (5D+) was provided to assist the panel members.[42] This instrument to measure health-related quality of life involves a three-point scale for six health dimensions, which are 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 on the basis of environmental epidemiologic reports and expert judgments. Subsequently, these state definitions were interpolated by a panel of environment-oriented physicians using the scale of calibration states, which was drawn up by Stouthard *et al*[37] (de Hollander AEM, van Kempen EEMM, Melse JM. Disability weights for health responses associated with major environmental exposures, 1999).

Health Impact Assessment

Environmental Outlook

Most health impact assessments produced within the framework of the National Environmental Outlook are based on a three-step procedure[43–49]:

- Assessing population exposure distribution;
- Defining health outcomes and quantifying the association between exposure and response; and
- Risk characterization, *ie*, estimating the number of people affected, duration, and severity of the condition.

Exposure Assessment

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

- Combining time-activity patterns for subpopulations with concentration distributions in micro and macro environments[50,51] [carcinogenic air pollutants, polycyclic aromatic hydrocarbons (PAH), radon];
- Combining data on population density with environmental quality by means of geographic information systems (noise, fine particulate matter and ozone air pollution, and large technologic accidents)[49]; and
- Estimating (often dichotomous) distribution of exposures on the basis of monitoring programs (environmental tobacco smoke, home dampness, and lead in drinking water).

Exposure-Response Modeling

Quantitative exposure-response data were derived from either occupational or environmental epidemiologic 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.[52] The nature of the mathematical models depended largely on the definition of exposure (dichotomous exposure categories or continuous exposure).

TABLE 2. Summary of Disability-Adjusted Life Years (DALYs) Lost to Selected Environmental Exposures in the Netherlands

Environmental Factor	Health Outcome	No. Affected Annually	S*	D†	DALYs	5th–95th Percentile
Particulate air pollution, long-term exposures	Mortality					
	Total	15,594	1.0	10.9‡	169,000	72,800–276,600
	Cardiopulmonary mortality	8,041	1.0	8.2‡	65,750	40,500–93,200
	Lung cancer	439	1.0	13.0‡	5,400	−9,740–20,650
	Morbidity					
	Chronic respiratory symptoms, children	10,138	0.17	1.0§	1,710	300–3,600
	Chronic bronchitis, adults	4,085	0.31‖	1.0§	1,920	170–4,450
Particulate air pollution, short-term exposures	Mortality					
	Respiratory	218	0.7¶	0.25#	37	2–114
	Coronary heart disease	253	0.7¶	0.25#	42	2–183
	Pneumonia	191	0.7¶	0.25#	33	3–94
	Other	452	0.7¶	0.25#	92	0–351
	Hospital admission					
	Respiratory	3,520	0.64	0.038**	86	25–195
	Cardiovascular	6,060	0.71	0.038**	164	48–380
	Emergency room visits					
	Respiratory	32,500	0.51	0.033**	584	0–1,756
	Aggravation of asthma					
	Asthmatic attacks	212,000††	0.22‡‡	0.005§§	253	−76–751
	Use of bronchodilators	530,000††	0.22‡‡	0.005§§	630	133–1,290
	Aggravation of respiratory symptoms					
	Upper respiratory tract	237,500††	0.05	0.02	215	8–555
	Lower respiratory tract	94,300††	0.21	0.04	760	57–1,881
Ozone air pollution	Mortality					
	Respiratory	198	0.7¶	0.25#	33	0–121
	Cardiovascular	1,946	0.7¶	0.25#	340	22–1,029
	Pneumonia	751	0.7¶	0.25#	131	9–410
	Other	945	0.7¶	0.25#	250	0–1,101
	Hospital admission					
	Respiratory disease	4,490	0.64	0.038**	103	9–281
	Emergency room visits					
	Respiratory disease	30,840	0.51	0.033**	550	44–1,520
PAH (BaP)	Lung cancer morbidity	17	0.43‖	2.9‖‖	16	1–32
	Lung cancer mortality	17	1.00	13.5‡	224	20–460
Benzene	Leukemia morbidity	5.3	0.83‖	2.7	12	2–29
	Leukemia mortality	5.3	1.00	21.2‡	113	18–278
Ethylene oxide	Leukemia morbidity	0.1	0.83‖	2.7	0,2	0–0,4
	Leukemia mortality	0.1	1.00	15.1‡	2	0,2–2,9
Vinyl chloride	Hepatoangiosarcoma morbidity	0.8	0.53	4.4‖‖	2	0.2–3.6
	Hepatoangiosarcoma mortality	0.8	1.00	13.3‡	10	1.0–19.0
1,2-Dichloroethane	Cancer morbidity	0.1	0.53	4.4‖‖	0.1	0.0–0.2
	Cancer mortality	0.1	1.00	13.3‡	0.7	0.1–1.3
Acrylonitrile	Lung cancer morbidity	0.1	0.43‖	2.9‖‖	0.0	0.0–0.1
	Lung cancer mortality	0.1	1.00	13.5‡	0.7	0.7–1.3
Radon (indoor)	Lung cancer morbidity	122	0.43‖	2.9‖‖	111	60–170
	Lung cancer mortality	122	1.00	13.5‡	1645	970–2,320
Damp houses	Lower respiratory disease					
	Children	16,920††	0.21	0.04	135	21–292
	Adults	55,630††	0.21	0.04	440	70–970
	Asthma					
	Children	3,400	0.08‖	1.0§	270	147–425
	Adults	10,534	0.08‖	1.0§	840	450–1,320
ETS	Lung cancer (female) morbidity	14	0.43‖	2.9‖‖	17	11–25
	Lung cancer (female) mortality	14	1.00	13.5‡	188	132–242
	Lung cancer (male) morbidity	20	0.43‖	2.9‖‖	24	5–46
	Lung cancer (male) mortality	20	1.00	13.5‡	263	58–478
	IHD (female) morbidity	1,822	0.29‖	1.0‖‖	527	266–856
	IHD (female) mortality	256	1.0	1.0¶¶	254	40–623
	IHD (male) morbidity	1,801	0.29‖	1.0‖‖	521	256–840
	IHD (male) mortality	234	1.0	1.0¶¶	233	37–575
	Aggravation of asthma	661,198	0.22‡‡	0.005	784	181–1461
	Lower respiratory symptoms	28,990††	0.21	0.04	233	40–462
	Otitis media, acute	136	0.31	0.06	3	0–6
	Sudden infant death	16	1.0	70	1125	1,027–1,223
Lead (drinking-water pipes)	Neurocognitive development (1–3 IQ points)	1,764	0.06	70	7950	982–18,722
Noise	Psychosocial effects					
	Severe annoyance	1,767,000	0.01	1.00	17,700	5,210–32,070
	Sleep disturbance	1,030,000	0.01	1.00	10,990	2,149–21,240
	Hospital admissions (IHD)	3,830	0.35	0.038**	50	7–140
	Mortality (IHD)	40	0.7¶	0.25#	10	0.5–26

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

Risk Characterization

We estimated the number of DALYs lost per year of exposure, using the following equation:

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

in which $DALY_{e.e.}$ = health loss related to n environmental exposures, measured as disability-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), and D_k = duration of the condition (for premature mortality D_K = loss of life expectancy).

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

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

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 foodborne acute gastroenteritis.[31]

Depending on the nature of the pollution-related condition, duration was determined from case definitions used in the epidemiologic studies involved, for example, respiratory symptoms, hospital admissions, and severe noise annoyance. In the case of well defined disease, duration was calculated from Dutch prevalence and incidence statistics, which implicitly assumed 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).[20,34]

For each exposure-related disease, a severity factor was composed as a prevalence-weighted average of compound disease states,[33] 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]

Uncertainty Analysis

We analyzed the uncertainty in the calculations of environmental DALYs by means of Monte Carlo techniques.[34–36] 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, or average duration of the response and discount factors, a probability-distribution function was estimated, representing parameter uncertainty. These distribution functions were based on available measurement statistics for each of the parameters. In a few cases, in which data were lacking, we relied on expert judgment (see Ref [34] 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).[34] The distribution of the output variable represents the uncertainty in the point estimate of the annual loss of DALYs attributable to the different environmental exposures. Here, we will present the 5th and 95th percentiles of this distribution as a measure of uncertainty.

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 accidents, 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 3. In Figure 3 the point estimates, as well as the 5th and 95th percentiles of the probability distribution for the number of exposure-attributable DALYs 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.[35]

In Figure 4, 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.

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 on the order of 2.5 million DALYs.[23] According to the calculations presented here, less than 5% of this disease burden would be attributable to environmental exposures, excluding accidents (12% when accidents are included).

TABLE 2. Continues

Environmental Factor	Health Outcome	No. Affected Annually	S*	D†	DALYs	5th–95th Percentile
Foodborne	Acute gastroenteritis					
	Symptoms	1,093,000	0.09	0.037	3,680	234–11,710
	Mortality	48	1.00	11.7‡	562	530–590
Large industrial accidents	Mortality	0.5	1.00	41.9***	20	6–35
UV-A/UV-B exposure, ozone-layer degradation	Melanoma morbidity	24	0.10‖	6.9‖‖	17	3–38
	Melanoma mortality	7	1.00	23.0‡	159	66–266
	Basal	2,150	0.053	0.21	24	4–53
	Squamous	340	0.027‖	1.5‖‖	14	2–30
	Other mortality	13	1.0	20.2‡	317	143–511
Traffic accidents	Incidence	200,900##				
	Hospital admissions	42,000	0.35	0.038	548	79–1,514
	Disability >1 year	6,193	0.43‖	9.50	26,520	11,830–41,950
	Mortality	1,322	1.00	35.90§	47,500	41,000–54,000
Domestic accidents	Incidence	1,630,300##				
	Hospital admissions	130,000	0.35	0.038	1,700	240–4,740
	Disability >1 year	9,119	0.17‖	9.50	19,660	3,100–39,000
	Mortality	2,017	1.00	41.9***	84,710	70,030–99,390

IHD = ischemic heart disease; ETS = environmental tobacco smoke; PAH = polycyclic aromatic hydrocarbons; BaP = benzo(a)pyrene; UV-A and UV-B = ultraviolet light.
* Severity weight: 0 = perfect health, 1 = death; prevalence weighted average in case several health states are involved.
† Duration of the health state (years).
‡ Based on standard life-table analysis.
§ Attributable prevalence (instead of cases), implying a duration of 1 year.
‖ Prevalence weighted average of different disease states.
¶ Assuming a disability weight of cases between 0 (healthy) and 0.6 (severe cardiopulmonary disease).
# Assuming a "harvesting" effect among patient with severe cardiopulmonary disease; minimum 1 day; most likely, 2 months; mean, 3 months; maximum, 5 years.
** Weighted average of duration of exacerbations requiring emergency room visit or hospital admission.
†† Events instead of cases.
‡‡ Disability weights from transition of one chronic obstructive pulmonary disease/asthma health state to the next (mild to moderate, moderate to severe).
§§ Average length of episode estimated to be 2 days.
‖‖ Calculated from incidence and prevalence data assuming steady state.
¶¶ 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.
Including accidents without significant injury.
*** Assuming a random age distribution among victims.

exposures (environmental tobacco smoke, radon, and dampness-related allergens) is intriguing, especially given the little attention it is receiving from policy makers (see Figure 5). 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.

Discussion

In deciding to attribute DALYs to environmental exposures, one has to consider several potential flaws. Most of these are not specific to the approach we have proposed here, but they 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 epidemiologic 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.[57,58] Given the inherent shortcomings of this type of epidemiologic study, 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, substantial uncertainties remain unsolved with respect to specific causative agents and mechanisms of action.[6,19,59,60]

Problems associated with health impact assessment have been discussed in numerous publications. Because 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.

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 epidemiologic research and for which clearly defined health outcomes have been established. This leaves many potentially adverse factors "uncovered."

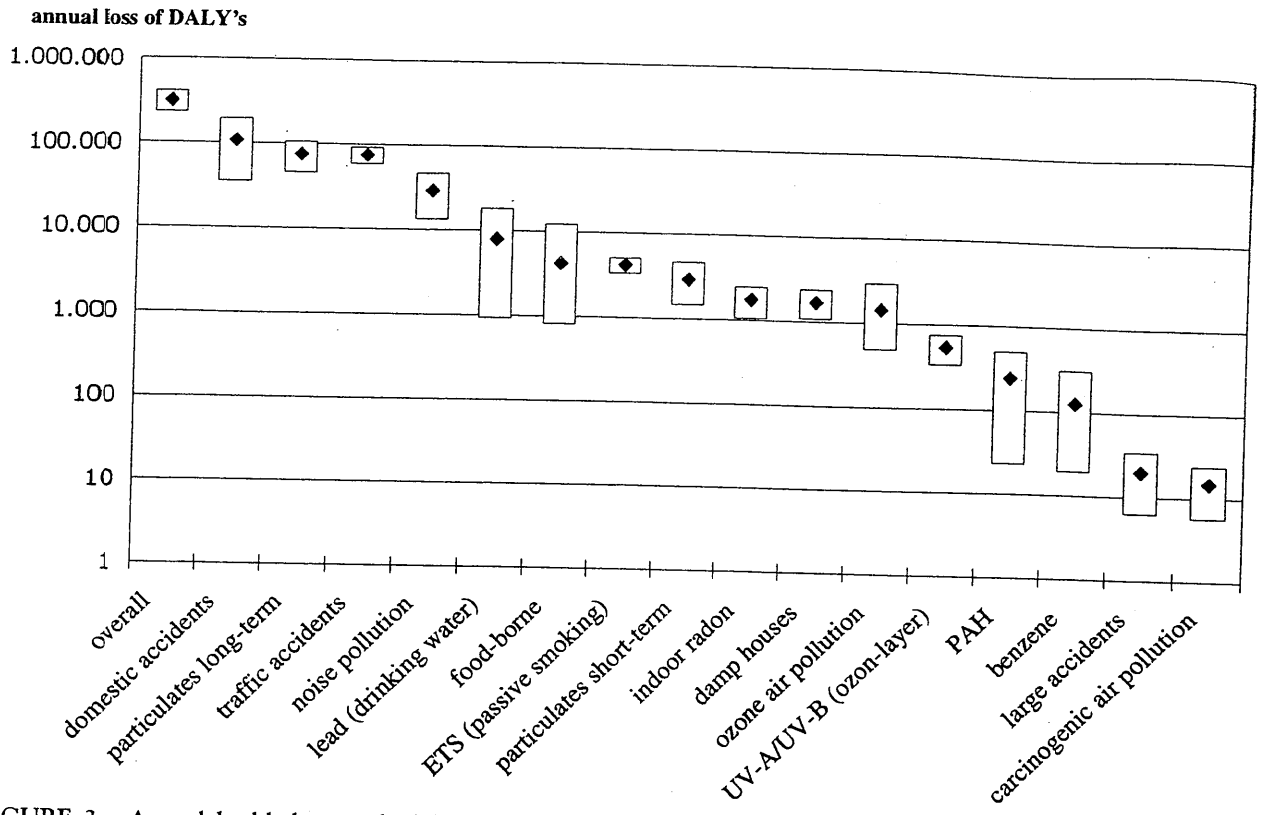

FIGURE 3. Annual health loss in disability-adjusted life years (DALYs) for selected environmental exposures in the Netherlands; shown are point estimates and 5th and 95th percentiles of probability intervals. ETS = environmental tobacco smoke; UV-A and UV-B = ultraviolet light; PAH = polycyclic aromatic hydrocarbons.

Inadequate Response Definitions

Many chemicals have exhibited adverse effects in experimental animals, ranging from slight changes in biochemical activity to pronounced pathologic changes and organ dysfunction, sometimes leading to premature death. These toxicologic findings represent a powerful tool in health protection.[61] However, for several reasons they are rather inadequate for human health impact assessment. First, high-exposure levels used in bioassays to avoid false-negative results yield responses that are often not relevant for normal environmental exposure conditions in most cases, for example, rodent bioassays for carcinogens.[62] Furthermore, species differences in biochemical or physiologic 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 on the basis of toxicologic information. Many, if not most standard toxicologic 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 on the basis of the available evidence.

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 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 nonfatal 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 judgments have to be made regarding the health significance of toxicologic or epidemiologic 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.[38,41,63]

Uncertainty analysis shows that altering weights within the variance seen in most weighing exercises does

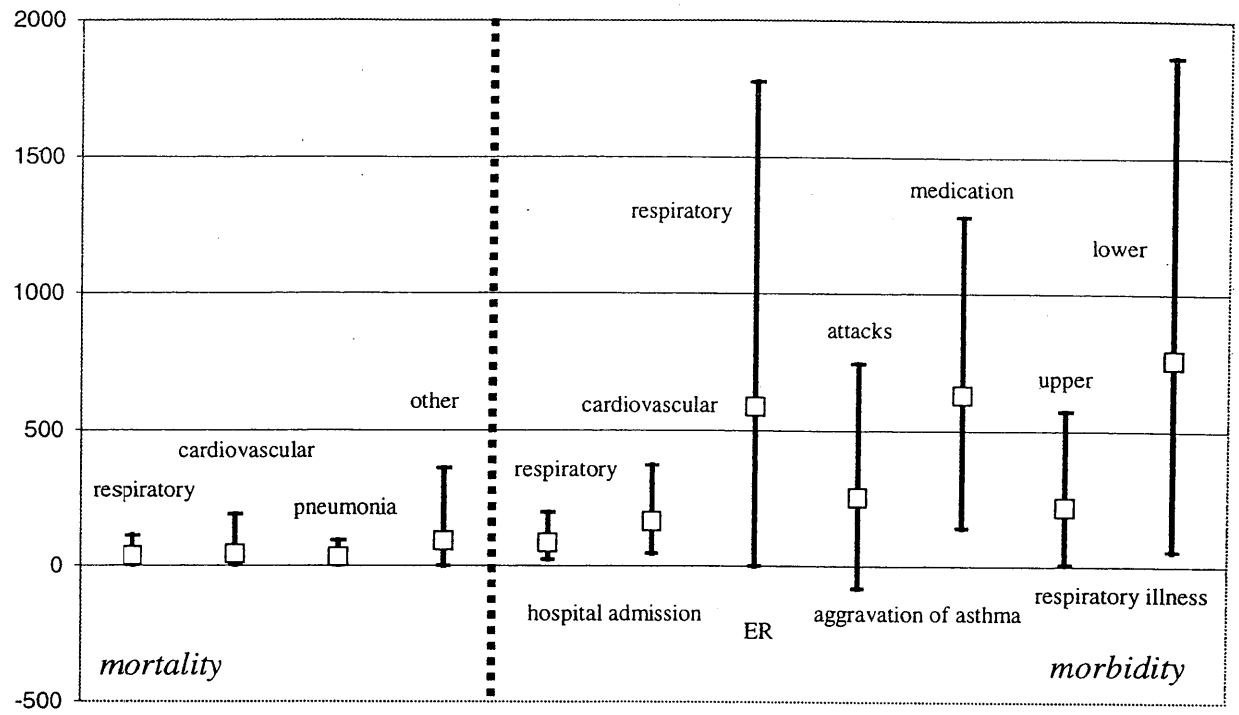

FIGURE 4. Annual health loss in disability-adjusted life years caused by several short-term endpoints associated with exposure to particulates in the Netherlands; shown are point estimates and 5th and 95th percentiles of probability intervals. ER = emergency room visit.

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. As the less severe responses tend to affect the highest number of people, variation in severity weights has a large impact on the estimates of disease burden. 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.

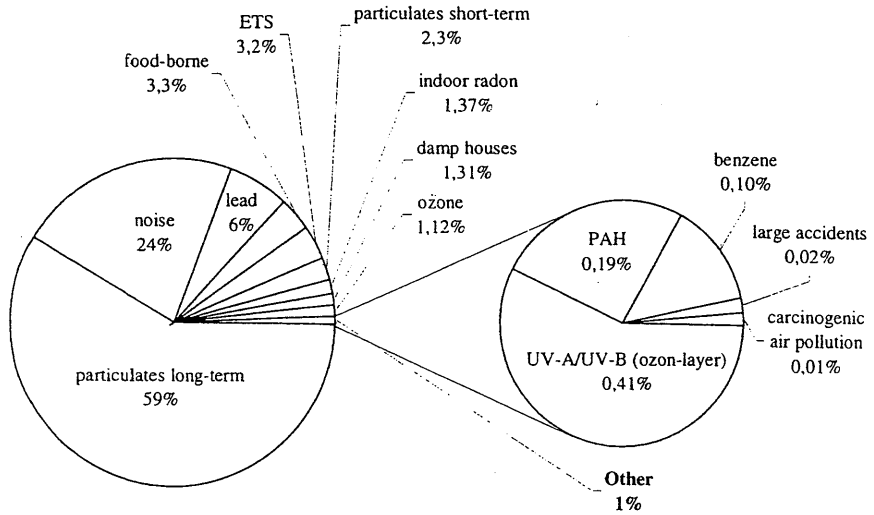

FIGURE 5. Relative health loss associated with selected environmental exposures in the Netherlands (traffic and domestic accidents excluded). ETS = environmental tobacco smoke; PAH = polycyclic aromatic hydrocarbons; UV-A and UV-B = ultraviolet light.

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,[54] 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, in the aggregate may seem obvious, but could 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 such as environmental quality or their own health than people with a lower socioeconomic status.[11,41]

Comorbidity

We were not always able to take proper account of comorbidity. Responses to air pollution such as lung function deficits, respiratory symptoms, and hospital admissions may concern the same people. Another example of possible comorbidity would be severe annoyance and sleep disturbance in relation to exposure to residential noise. This known comorbidity 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 epidemiologic research. Of course, there is no way of quantifying this possible underestimation.

Apart from comorbidity, 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.

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. Despite the application of rather conservative estimation methods, carcinogenic pollutants in ambient air, for instance, do not seem to contribute much to the disease burden. Still, much policy effort is put in monitoring, evaluating, and managing outdoor exposure levels of pollutants, such as PAH 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.[3,14]

Nonetheless, we would not dare to suggest an immediate change of environmental policy priorities on the basis of these calculations. Discrepancies may be partly explained by dimensions of health risk perception, which are not captured in our approach, such as "dread," whether exposure is voluntary, perceived controllability or familiarity of risk-generating processes (for example, traffic), or social distribution of risk and benefit.[11,12,13] In the Netherlands the actual number of victims of large technologic accidents (industry accidents or airplane crashes) is very small. Nevertheless, several surveys have shown that people are worried about them, however small the odds as calculated by experts.[11,12] Of course, these perceptions by themselves can seriously affect well being and quality of life.

Uncertainty

The degree of certainty varies from one health-impact estimate to another. This may pertain to available data for modeling (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 prioritizing environmental health issues solely on the basis of point estimates, given the overlapping uncertainty ranges. On the other hand, one can quite clearly discern groups of high-risk exposures (accidents or long-term exposure to particulates) from groups of moderate-risk (lead, food, environmental tobacco smoke, or radon) and low-risk exposures (carcinogenic air pollutants). Continuous efforts along the lines we have 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.[55,65–67]

Conclusion

We conclude from our exercise that despite methodologic 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 nonfatal 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.

Regarding risk communication, 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. We have not yet tested the face validity of this approach with the public, however.

References

1. Shore RE. Epidemiologic data in risk assessment: imperfect but valuable. Am J Public Health 1995;85:474–475.
2. Hertz-Picciotto I. Epidemiology and quantitative risk assessment: a bridge from science to policy. Am J Public Health 1995;85:484–491.
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 suppl:3–13.
5.

5. Koren HS, Utell MJ. Asthma and the environment (Meeting Report). Environ Health Perspect 1997;105:534–537.
6. Lipfert FW. Air pollution and human health: perspectives for the '90s and beyond. Risk Anal 1997;17:137–146.
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;131:666–668.
8. Covello VT, Merkhofer MW. Risk assessment methods: approaches for assessing health and environmental risks. New York: Plenum Press, 1993.
9. Morgan MG. Risk analysis and management. Sci Am 1993;269:24–30.
10. Sexton K. Science and policy in regulatory decision making: getting the facts right about hazardous air pollutants. Environ Health Perspect 1995; 102(suppl 6):213–222.
11. Health Council of the Netherlands, Committee on Risk Measures and Risk Assessment. Not All Risks Are Equal: A Commentary on Premises for Environmental Risk Assessment. Pub. No. 1995/06E. The Hague: Health Council of the Netherlands, 1995.
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. ch. 3. Final report, vol. 2. Washington DC: U.S. Government Printing Office, 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. Ministry of Housing, Physical Planning and Environmental Protection. Premises for Risk Management (Addendum to the Netherlands National Environmental Policy Plan 1990–1994). The Hague: Ministry of Housing, Spatial Planning, and the Environment, 1989.
16. World Health Organization (WHO). Air Quality Guidelines for Europe. Regional Publications, European Series No. 23. Copenhagen: WHO Regional Office for Europe, 1987.
17. National Research Council. Risk Assessment in the Federal Government: Managing the Process. Washington DC: National Academy Press, 1983.
18. Health Council of the Netherlands: Committee on Risk Measures and Risk Assessment. Risk Is More Than Just a Number. Pub. No. 1996/03E. The Hague: Health Council of the Netherlands, 1996.
19. Vedal S. Ambient particles and health: lines that divide. J Air Waste Manage Assoc 1997;47:551–581.
20. Brunekreef B. Air pollution and life expectancy: is there a relation? Occup Environ Med 1997;54:781–784.
21. Ten Berge WF, Stallen PJM. How to compare risk assessments for accidental and chronic exposure. Risk Anal 1995;15:111–113.
22. World Bank. World Development Report 1993: Investing in Health: World Development Indicators. New York: Oxford University Press, 1993.
23. 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.
24. Olshanski SJ, Rudberg MA, Carnes BA, Cassel CK, Brody JA. Trading of longer life for worsening health. J Aging Health 1991;3:194–216.
25. 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.
26. 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.
27. 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.
28. Morris RD. Drinking water and cancer. Environ Health Perspect 1995;103 (suppl 8):225–231.
29. 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;9:21–28.
30. 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;9:29–35.
31. 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.
32. Fischhoff B, Bostrom A, Quadrel MJ. Risk perception and communication. Annu Rev Public Health 1993;14:183–203.
33. Fischhoff B. Risk perception and communication unplugged: twenty years of process. Risk Anal 1995;5:137–145.
34. 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, the Netherlands: RIVM (National Institute of Public Health and the Environment) (in press), 1999.
35. 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. vol. I. Cambridge, MA: Harvard School of Public Health, 1996.
36. 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):1:347–532.
37. 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: Department of Public Health, Erasmus University Rotterdam, 1997.
38. 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. vol. I. Cambridge, MA: Harvard School of Public Health, 1996.
39. Froberg DG, Kane RL. Methodology for measuring health-state preferences. I–IV. J Clin Epidemiol 1989;42:345–354, 459–471, 585–592, 675–685.
40. Committee on Medical Cure and Care. Report on choices in medical cure and care. The Hague: Ministry of Welfare, Health and Culture, 1991.
41. 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. vol. I. Cambridge, MA: Harvard School of Public Health, 1996.
42. Essink-Bot ML. Health status as a measure of outcome of disease treatment. Erasmus University Rotterdam (Thesis), 1995.
43. Twickel Group. Quantitative assessment of health effects associated with exposure to particulate air pollution in the Netherlands. RIVM report 623710002. Bilthoven, the Netherlands: National Institute of Public Health and the Environment, 1995.
44. Passchier-Vermeer W. Noise and Health. Pub. No. A93/02E. The Hague: Health Council of the Netherlands, 1993.
45. Krzyzanowski M. Methods for assessing the extent of exposure and effects of air pollution. Occup Environ Med 1997;54:145–151.
46. Melse JM, de Hollander AEM. Health impact assessment of environmental benzene exposure, assumptions and science policy. Am J Epidemiol 1996;7: S48.
47. Slaper H, Velders GJM, Daniel JS, de Gruijl FR, van der Leun JC. Estimations of ozone depletion and skin cancer incidence to examine the Vienna Convention achievements. Nature 1996;384:256–258.
48. Leenhouts HP, Stoop P, van Tuinen S. Non-nuclear industries in the Netherlands and radiological risks. Report 610053003. Bilthoven, the Netherlands: National Institute of Public Health and the Environment, 1994.
49. Ale BJM, Janssen MPM, Pruppers MJM, eds. RISK 97: International Conference on Mapping Environmental Risks and Risk Comparison, Amsterdam, October 21–24 (Book of papers). Bilthoven, the Netherlands: National Institute of Public Health and the Environment, 1997.
50. Ryan PB. An overview of human exposure modeling. J Expo Environ Epidemiol 1992;1:453–473.
51. Eerens HC, Sliggers CJ, van den Hout KD. The CAR model: the Dutch method to determine city street air quality. Atmos Environ 1993;27b:389–399.
52. Hollander AEM de, EA Preller, S Heisterkamp. Hospital admission due to summer smog: a Bayesian approach to meta-regression analysis. Annual Scientific Report, Bilthoven, the Netherlands: National Institute of Public Health and the Environment, 1997.
53. Melse JM, Kramers PGN. A national burden of disease calculation: Dutch DALYs. Report No. 431501028. Bilthoven, the Netherlands: RIVM (National Institute of Public Health and the Environment), 1998.
54. Burmaster DE, Anderson PD. Principles of good practice for the use of Monte Carlo techniques in human health and ecological risk assessment. Risk Anal 1994;14:477–481.
55. 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 Anal 1994;14:707–712.
56. Thompson KM, Burmaster DE, Crouch EA. Monte Carlo techniques for quantitative uncertainty analysis in public health risk assessments. Risk Anal 1992;12:53–63.
57. Dockery DW, Pope CA III, XU X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE. An association between air pollution and mortality in six U.S. cities. N Engl J Med 1993;340:1010–1014.
58. Pope CA III, 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 U.S. adults. Am J Respir Crit Care Med 1995;151:669–674.
59. Committee on the Medical Effects of Air Pollutants. Non-Biological Particles and Health. London: Her Majesty's Stationary Office Committee on the Medical Effects of Air Pollution, 1995.

60. U.S. EPA. Air Quality Guidelines for Particulate Matter. Washington DC: US Environmental Protection Agency, 1996.
61. World Health Organization (WHO): International Program on Chemical Safety. Assessing human health risks of chemicals: derivation of guidance values for health-based exposure limits. Environmental Health Criteria 170. Geneva: WHO, 1994.
62. Ames BN, Gold LS. The causes and prevention of cancer: gaining perspective. Environ Health Perspect 1997;105 suppl:8:65–73.
63. Nord E. Methods for quality adjustment of life years. Soc Sci Med 1992;34: 559–569.
64. Zielhuis RL, Wibowo AAE. Standard setting in occupational health: philosophical issues. Am J Ind Med 1989;16:569–598.
65. Johnson BB, Slovic P. Presenting uncertainty in health risk assessment: initial studies of its effects on risk perception and trust. Risk Anal 1995;15: 485–494.
66. Rhomberg L. Beyond screening: problems and prospects for risk characterization of endocrine disrupters. Regul Toxicol Pharmacol 1997;26:74–79.
67. Advisory Committee on Dangerous Pathogens. Seminar on Microbiological Risk Assessment, 28 January, 1997. J Appl Microbiol 1997;83:659–664.