Annex 6.1:

12th Annual Meeting of the International Society for Environmental Epidemiology
(ISEE 2000)

Session on Environmental Burden of Disease

Programme – 22 August 2000

Chair: Carlos Corvalán, World Health Organization

Introduction and background to environmental burden of disease assessment, Carlos Corvalán, Protection of the Human Environment, World Health Organization, Geneva, Switzerland

Methodological approaches to environmental burden of disease assessment, Annette Pruess, Protection of Human Environment, World Health Organization, Geneva, Switzerland

Assessing environmental disease burden: examples from the Netherlands, Hollander AEM de, Kempen EEA, Staatsen BA, Center for Chronic Disease and Environmental Epidemiology, National Institute of Public Health and the Environment, Bilthoven, Netherlands

Global burden of disease from exposure to indoor air pollution, Sumi Mehta, Kirk Smith, School of Public Health, University of California, Berkeley, USA

Approach for burden of disease estimation for exposure to lead, Lorna Fewtrell, Centre for Research into Environment and Health, Crewe, UK

Assessing the global burden of disease attributable to climate, Tony McMichael, Department of Epidemiology and Population Health, London School of Hygiene and Tropical Medicine, London, UK

Discussion
Information about the impact of environmental risk factors on human health, at different levels (village, city, province or country), is necessary in order to support management and the decision-making process for environmental health protection. Decision-makers need this information in order to develop preventive strategies, to compare the potential effects of different decisions and choices and to assess the impacts of their decisions. The development of a scientifically sound methodology and estimates of the environmental burden of disease is, however, a major challenge. WHO has been developing activities supporting such initiatives for several years (slide 1). Particular efforts are currently under way to develop methodologies for country and regional level assessments. In parallel, disease burden for selected risk factors is being estimated at global level (slide 2).

To introduce the presentations in this special session on environmental burden of disease, we need to briefly address the basic concepts of burden of disease (slide 3) and summary measures of population health used to assess it (slide 4); the term Disability Adjusted Life Years (slide 5), and the main results of the burden of disease study by Murray & Lopez (slide 6).
The rationale for generating environmental burden of disease estimates at national and international level are summarized in slides 7 and 8. The presentations to follow in this symposium will address the methodological framework in environmental burden of disease, examples of current studies and applications in specific settings.

**Disability-Adjusted Life Years**

\[ \text{DALY} = \text{YLL} + \text{YLD} \]

- years of life lost because of premature death (YLLs)
- years of life lived with disability (YLDs)

**Burden = Mortality + Disability**

- one DALY = one last year of healthy life
- Death of a male at 50 = 30 DALYs
- Schizophrenia at 20 = 30 DALYs

**Aims of GBD project: national/regional level**

- To provide a tool for quantifying BoD from major environmental risk factors
- Provides information on burden of disease and preventable part
- Together with cost-effectiveness of interventions and social and ethical framework provides rational basis for priority setting in research, implementation and policy development
- Monitor progress
- Points to vulnerable population subgroups
- Compares environmental health to other areas

**Aims of GBD: international level**

- Provide a worldwide picture of disease burden due to environmental risk factors
- Provides information for major policy directions / international efforts
- Highlights main problems at global level
- Provides information to donors
- Points to countries in greatest needs for support on selected issues

**Top ten: 1999 and trends to 2020**

<table>
<thead>
<tr>
<th>Rank</th>
<th>Cause</th>
<th>DALY %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Lower respiratory infection</td>
<td>6.7</td>
</tr>
<tr>
<td>2</td>
<td>HIV/AIDS</td>
<td>6.2</td>
</tr>
<tr>
<td>3</td>
<td>Diarrhoeal diseases</td>
<td>5.0</td>
</tr>
<tr>
<td>4</td>
<td>Unipolar major depression</td>
<td>4.1</td>
</tr>
<tr>
<td>5</td>
<td>Ischaemic heart disease</td>
<td>4.1</td>
</tr>
<tr>
<td>6</td>
<td>Cerebrovascular disease</td>
<td>3.5</td>
</tr>
<tr>
<td>7</td>
<td>Malaria</td>
<td>3.1</td>
</tr>
<tr>
<td>8</td>
<td>Road traffic accidents</td>
<td>2.8</td>
</tr>
<tr>
<td>9</td>
<td>Chronic obstruct. pulmonary dis.</td>
<td>2.7</td>
</tr>
<tr>
<td>10</td>
<td>Congenital abnormalities</td>
<td>2.5</td>
</tr>
</tbody>
</table>
Annex 6.3: Environmental burden of disease—Methodological approaches

Annette Prüss
Protection of the Human Environment, World Health Organization

Countries are increasingly interested in looking at causative life-style, social or physical factors and wish to quantify the disease burden they cause. Environmental health factors are at the origin of a large part of the disease burden world wide. WHO is intensifying its effort to provide support in the assessment of environmental burden of diseases. The main emphasis is on national or regional assessments, as decision-making is usually taking place at that level and typically relies on national and regional assessments (besides issues with of global impacts, such as climate change).

Planned activities in the assessment of environmental burden of disease are summarized in Slide 1. Slide 2 shows the additional type of information which the burden of disease assessment can feed into the policy debate.

For example, a study performed in the Netherlands and the USA on the positive and negative consequences of adding disinfection products to drinking water has compared potential health outcomes in terms of disease burden. Potential burden of microbiological disease due to lower disinfection levels were compared to the potential disease burden from cancers suspected to be associated with disinfection by-products.
For comparability of results between disease outcomes and risk factors, some common features or methodologies are needed when estimating environmental disease burden, which is yet to be developed (Slide 3).

The health and environment cause-effect framework (Slide 4), links measurable indicators to environmentally caused diseases and relates distal and proximal causes in a global perspective. It could be expanded to include the analytical aspects and consideration of interactions between causal parameters, which is necessary for quantification of the disease burden, in particular when interactions between risk factors and disease outcomes are complex. Its application to transport policy is outlined in Slide 5. A more analytical version of such frameworks is needed to support the estimation of environmental disease burden assessments.
Main issues which will need to be addressed to support initiatives in environmental burden of disease are described in Slide 6. Working definitions will need to be established, and alternative (or counterfactual) scenarios will need to be defined (Slides 7 and 8).

The limited data availability in environmental health, and the weakness of the evidence in some areas results in important limitations in many applications in this area, and should be noted (Slide 8). A recapitulation of activities planned in the framework of this project are outlined in Slide 9.

Other issues outlined during this presentation are described in the Background document, in Annex 1 of this document.
Annex 6.4: Assessing environmental disease burden
the example of noise in the Netherlands

Augustinus EM de Hollander, Elise EMM van Kempen, Rudolf T Hoogenveen
National Institute of Public Health and the Environment (RIVM)

The RIVM produces National Environmental Outlooks (NEO) every 3 or 4 years to support environmental policy making by the government. The first one was produced in 1987, and now we are about to publish number 5.

Basically we try to assess the current as well as the future state of the environment using different scenarios for the future. In the fifth NEO we try to look 30 years ahead. Indicators are used from one end of the causal chain, driving forces such as demography, economy, public, health, pressure, state and impact: for instance ecological and human capital.

From the public health perspective it is necessary to assess the health loss to environmental exposures, as there are indications that the perception of environmental health risks may be somewhat distorted in our society.

To do so one has to apply a public health currency unit that encompasses the very diverse responses that may be associated with environmental pollution. That may range from slight aggravation of respiratory disease all the way up to the loss of many potentially healthy life years due to premature mortality. Ergo: this measure had to comprise important aspects of health such as quantity of life, quality of life and number of people involved. Inspired by the Global burden of disease project we applied a concept very close to the disability adjusted life years DALYs (Slide 1).

Slide 2 represents a simplified diagram of the basic idea behind DALYs. At birth we all have eighty years of potentially healthy life ahead. Unfortunately most of us will suffer from diseases, due to our genetic program, our unhealthy life styles, dietary, occupational, environmental factors or just bad luck. The aim here is to estimate the loss of DALYs that can be attributed to environmental exposures.
The reasons for applying an aggregate health impact indicator include the following (Slide 3):

- To compare the significance of exposures with other environmental exposures or life style factors. Most common risk measures are non informative (probabilistic, death, non-fatal health outcomes): PM versus noise.

- To evaluate the most effective policy options in terms of health gain (classical example chlorination drinking water, acute infectious disease compared to cancer). Should we concentrate on carcinogenic air pollution or would the abatement of noise exposure provide better returns. Or is particulate matter the only thing that really matters?

- In the NL there is significant spatial accumulation of environmental stress, especially in urban areas. Environmental DALYs may help us to compare one situation to another.

In public health terms, one should remember that there is more to good risk communication than finding the right impact measure.

Before going any further, one might have a look at the health definition according to the WHO-charter; this definition is quite close to the definition of happiness; others would prefer to only consider responses that can be clearly defined by medical doctors (Slide 4).

**Why an aggregate risk indicator?**

- comparative evaluation of environmental health impact (‘how bad is it?’)
- evaluation of environmental policy efficiency (‘best buy in reduction of health loss’)
- assessment of accumulation environmental exposures (urban environments)
- communicating health risk (?)

**Key Question: define health?**

- ‘a state of complete physical, mental and social well-being, and not merely the absence of disease or infirmity’ (WHO charter, 1946)
- ‘the ability to cope with the demands of daily life’ (the Dunning Committee on Medical Cure and Care, 1991)
- the absence of disease and other physical or psychological complaints (NSCGP, 1999)

In our very densely populated country environmental noise really is a major problem. Cities are built in a very compact way; there is a lot of traffic congestion; and last but not least we want to operate a relatively large airport in the most densely populated area of the Netherlands: Schiphol. 27% of the Dutch population reports themselves to be severely annoyed by traffic noise, for air traffic noise this percentage is 17.

An interesting feature of the health effects of noise is that one might distinguish social and clinical responses, depending on the definition of health one is using. (uncertainty of health responses: annoyance no problem, cardiovascular disease inconclusive, borderline significant), Slide 5.

The Netherlands have the advantage to have relatively good data on noise emissions and exposure. These are processed by quite sophisticated models to assess the effectiveness of policy measures, using geographic information systems. These encompass mobile source characteristics (cars, planes road surface characteristics, large noise shields), spatial characteristics (such as how residential areas and traffic roads are organized), Slide 6.
Slide 7 shows a crude output of this national model for road traffic and air traffic in 1994 and projections for 2030. Traffic noise exposure roughly stabilizes, while exposure to airplane noise significantly increases given the expansion Schiphol airport.

A conceptual model describing the impact of noise is represented in Slide 8. Response are determined by noise levels and characteristics of course, but may be modified by social and endogenous factors such as attitude, coping style etc. Noise induces disturbance of sleep and daily activities, annoyance, stress which may lead to various intermediate responses, such as hypertension, increased stress hormone levels, shifts in cholesterol composition etc. In turn these may affect the risk of cardiovascular disease. This model is still controversial; there is mechanistic evidence from clinical studies, and there are epidemiological indications for an association between noise exposure and cardiovascular endpoints, be it still inconclusive and controversial.

To assess what would be the public health significance of noise exposure for cardiovascular disease, if the association was causal, we used the results of a comprehensive meta-analyses of all published studies to assess the noise attributable cardiovascular disease burden. Relative risk estimates were combined with exposure distributions and Dutch prevalence and incidence data on cardiovascular disease. These are preliminary estimates, keeping in mind that some of the estimates were far from statistical significant.
Slide 9 shows the results for road traffic exposure, which display a pyramid shape: Many people suffering from mild effects such as annoyance or sleep disturbance, relatively few people having serious cardiovascular symptoms. We are still in the process of refining the calculations especially with respect to uncertainty analyses (Monte Carlo).

- severe annoyance: 1,500,000-2,000,000
- sleep disturbance: 400,000-1,000,000
- GP consult: 15,000-40,000
- hypertension: 9,000-25,000
- anti-hypertensives: 1,500-13,000
- Angina pectoris: 0-1,100
- death: 0-21
Cardiovascular health end-points associated from air traffic noise show a similar pattern (Slide 10). Some of these end-points have a lower limit of zero, reflecting non-significant meta-analysis results.

- Annoyance: 300,000-600,000
- Sleep disturbance: 100,000-160,000
- Hypertension: 0-68,000
- Anti-hypertensives: 0-25,500
- Ischaemic heart disease: 1,400-3,000
- Angina Pectoris: 0-3,700
- Myocard infarction: 150,500
- Death: 0-82

Sleep disturbance is measured sleep logs and diaries, actimeters (watch-like instruments recording nocturnal movements: subjective sleep quality measurements, and number of awakenings during sleep period time. A good test model is urgently needed.

To estimate the actual health loss associated with noise exposure in terms of disability adjusted life years, we used a chronic disease model developed at our institute (Slide 11). Basically this model can be regarded as a sophisticated life-table. Applying a demographic module and trends in (common) risk factor prevalence it simulates annual changes in disease-specific morbidity as a result of incidence, recovery, disease progression or death. By using noise attributable changes in hypertension prevalence as input we were able to calculate attributable morbidity and excess mortality rates (incidence, initial prevalence and mortality were derived from Dutch health data collected in the framework of our Public Health Status and Forecast Report. By combining years of life lost and years spent with disease we were able to calculate the loss of DALYs due to noise exposure).
Slide 12 provides some provisional results compared to disease burden estimates for a number of other environmental exposures for a 2030 scenario.

Slide 13 represents disease burden in the hypothesis that social responses such as annoyance and sleep disturbance are considered as a genuine health effect (cumulative not source specific). In fact annoyance and sleep disturbance was included in our formal exercises to attribute severity weights to health states by panels of physicians. Very few
of the panel members objected to giving weight to these states for not being a health end-point. Although the weights were very low in general, due to the large number of cases the resulting health burden was very substantial. It is disputable whether these end points can be evaluated in the same league.

A number of critical points to conclude:

The epidemiological evidence with respect to noise and cardiovascular disease is relatively poor and inconclusive, especially the exposure assessment is often very poor, furthermore most studies are of a cross-sectional design. Substantial confounding due to social-economic status is suspected, which makes it difficult to detect the small attributable risk due to noise.

The discussion on what to consider as health effect is interesting. Healthy life expectancy in postmodern society has remarkably increased and quality of life issues are increasingly dominating the discussion.

The application of severity weights, although formally derived in a relatively sophisticated way, introduces a subjective aspect into the model, which is sometimes disputed. These severity weights only seem to be critical with respect to mild response with a substantial prevalence.

In these types of integrated assessments many substantial uncertainties are accumulating. Despite available methods to describe and quantify uncertainty, it will be difficult to convey the right message to policy makers and the public. Uncertainties may even regard the constructs we use.
Annex 6.5: Estimating the global burden of disease from indoor air pollution

Kirk R. Smith and Sumi Mehta

*University of California at Berkeley*

Human exposure to air pollution is dominated by the indoor environment. Here, we address indoor exposures from indoor sources. A significant amount of indoor air pollution comes from outdoor sources, and vice-versa, depending on the exposure scenario. However, here we do not address indoor exposures resulting from outdoor sources, nor do we address how indoor sources can affect outdoor pollution levels. Sources of indoor air pollution in the household environment are described in Slide 1 below:

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Particles</td>
<td>Solid fuel combustion, smoking, cleaning</td>
</tr>
<tr>
<td>Combustion Byproducts</td>
<td>Fuel combustion, smoking</td>
</tr>
<tr>
<td>Volatile Organic Compounds (VOCs)</td>
<td>Furnishings, household products, smoking, solid fuel combustion</td>
</tr>
<tr>
<td>Biological Pollutants</td>
<td>Furnishings, ventilation, moist areas in home</td>
</tr>
<tr>
<td>Pesticides</td>
<td>Household products, outdoor dust</td>
</tr>
<tr>
<td>Radon</td>
<td>Ground beneath structure, ventilation</td>
</tr>
</tbody>
</table>

*Slide 1*

We focus on the household environment, as the largest fraction of time spent indoors occurs at home. Other key indoor environments include schools, vehicles, and the workplace. However, there is a lack of exposure-response studies in schools and vehicles, and workplaces exposures are diverse and better dealt with separately.

This project focuses on three major indoor air pollution exposures, as detailed below (Slide 2):
3 Major IAP Exposures

1. Largest traditional source of exposure:
   **Cooking and heating with solid fuels** (wood, coal, dung, charcoal, agricultural residues)

2. Largest modern source of exposure:
   **Environmental tobacco smoke (ETS)**

3. Potentially large source of exposure:
   **Radon**

*Slide 2*

Four major approaches have been used to estimate the GBD from IAP (Slide 3). Each approach uses different types of data and methodology. It should be noted that the exposure-based approach, which involves a disease-by-disease summation of associated health effects, is the only method likely to result in an underestimate of GBD.

<table>
<thead>
<tr>
<th>Approach</th>
<th>Method</th>
<th>Likely Bias</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pollutant-based</td>
<td>Exposure-response extrapolation</td>
<td>Overestimate</td>
</tr>
<tr>
<td>Child Survival</td>
<td>Survival analysis</td>
<td>Overestimate</td>
</tr>
<tr>
<td>Cross-National</td>
<td>Regression</td>
<td>Overestimate</td>
</tr>
<tr>
<td>Exposure-based</td>
<td>Disease by disease summation</td>
<td>Underestimate</td>
</tr>
</tbody>
</table>

*Slide 3*

The Slide below (Slide 4) demonstrates how estimates of annual total mortality from indoor air pollution from household biomass use in India differ depending on the approach used.
This project uses the exposure-based approach to quantify the global burden of disease (GBD) from household sources of indoor air pollution. A description of the methodology used in this approach is provided in Slide 5 below.

**The Exposure Based Approach**

- Estimated prevalence of exposure
- Relative risk estimates from epidemiological studies
- Morbidity and mortality estimates from the Global Burden of Disease Study (WHO/Harvard 1996)
- Population Attributable Risk (PAR)

\[
\text{PAR} = \frac{P_e(RR-1)}{1 + P_e(RR-1)}
\]

As with all approaches, the exposure based approach has strengths and limitations (Slide 6).
An application of the attributable risk calculation is demonstrated for acute respiratory infections (ARI) associated with solid fuel use in Slide 7 below. India and the Latin American / Caribbean region have very different patterns of solid fuel use, resulting in very different percentages of population attributable risk (PAR) even when the same relative risk estimate is used. When these PAR are used in conjunction with the different incidences of ARI in the two regions, very different patterns of disease burden (here, mortality from ARI) emerge.

**Example: ARI from Solid Fuel Use**

**India:**
- 81% solid fuel use
- This translates into 53% PAR
- \( \approx 400,000 \) deaths from ARI attributable to IAP

**Latin American Countries:**
- 25% solid fuel use
- This translates into 27% PAR
- \( \approx 30,000 \) deaths from ARI attributable to IAP

**Indoor Air Pollution from Solid Fuel Use**

Slide 8 details the health outcomes are addressed in the solid fuel use section, and their resulting burden of disease. For health outcomes with strong epidemiological evidence, the geometric mean of the low and high relative risk estimates were used. For health outcomes with moderate or limited evidence, the low relative risk estimate was utilized. It should be noted that ‘moderate’ and ‘limited’ do not refer to inconclusive findings. Rather, they suggest that additional, carefully conducted studies are needed to strengthen the evidence base.
Methodology for assessment of environmental burden of disease

GBD from Solid Fuel Use: Health Outcomes Addressed

<table>
<thead>
<tr>
<th>Illness</th>
<th>Population</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Respiratory Illness (ARI)</td>
<td>Children &lt;5</td>
<td>Strong</td>
</tr>
<tr>
<td>Chronic Obstructive Pulmonary Disease (COPD)</td>
<td>Women ≥15</td>
<td>Strong</td>
</tr>
<tr>
<td>Lung Cancer (coal only)</td>
<td>Women ≥15</td>
<td>Strong</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Women ≥15</td>
<td>Moderate</td>
</tr>
<tr>
<td>Blindness (Cataracts)</td>
<td>Women ≥15</td>
<td>Moderate</td>
</tr>
<tr>
<td>Asthma</td>
<td>Women ≥15</td>
<td>Moderate</td>
</tr>
<tr>
<td>Ischaemic Heart Disease</td>
<td>Women ≥15</td>
<td>Limited</td>
</tr>
</tbody>
</table>

* Insufficient evidence to address other potential impacts, including low birth weight and other adverse pregnancy outcomes

The following four slides (Slides 9 – 12) provide a brief description of our findings. Solid fuel use is associated with nearly 2 million deaths in 1990. Over 1.2 million of these deaths are attributable to ARI in children under five years of age, with India and Sub-Saharan Africa bear the largest burden of these deaths. Solid fuel use accounts for around 4.9% of deaths and 4.4% of DALYs in developing countries. When compared to other major risk factors in developing countries quantified in the original burden of disease study, this ranks below malnutrition (14.9% of deaths, 18% of DALYs) and water/sanitation (6.7% deaths, 7.6% DALYs), but much higher than outdoor air pollution (0.7% deaths and 0.4% DALYs). It should be noted, however, that all of these other risk factors are currently being re-evaluated, so their ranking is likely to change.

GBD from Solid Fuel Use, 1990

<table>
<thead>
<tr>
<th>Illness</th>
<th>Deaths</th>
<th>DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Respiratory Illness (ARI)</td>
<td>1,230,000</td>
<td>43,284,000</td>
</tr>
<tr>
<td>Asthma</td>
<td>9,000</td>
<td>511,000</td>
</tr>
<tr>
<td>Blindness COPD</td>
<td>417,000</td>
<td>5,243,000</td>
</tr>
<tr>
<td>Ischaemic Heart Disease</td>
<td>98,000</td>
<td>785,000</td>
</tr>
<tr>
<td>Lung Cancer</td>
<td>31,000</td>
<td>296,000</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>185,000</td>
<td>3,588,000</td>
</tr>
<tr>
<td>TOTAL</td>
<td>1,970,400</td>
<td>54,339,000</td>
</tr>
</tbody>
</table>

GBD from Solid Fuel Use in 1990

<table>
<thead>
<tr>
<th>Region</th>
<th>Deaths</th>
<th>DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle Eastern Crescent</td>
<td>409,000</td>
<td>110,000</td>
</tr>
<tr>
<td>Latin America and Caribbean</td>
<td>253,000</td>
<td>60,000</td>
</tr>
<tr>
<td>Sub-Saharan Africa</td>
<td>130,000</td>
<td>20,000</td>
</tr>
<tr>
<td>China</td>
<td>90,000</td>
<td>5,000</td>
</tr>
<tr>
<td>Other Asia and Islands</td>
<td>143,000</td>
<td>35,000</td>
</tr>
<tr>
<td>Middle Eastern Crescent</td>
<td>1,230,000</td>
<td>390,000</td>
</tr>
</tbody>
</table>

Nearly Two Million Deaths from Solid Fuel Use in 1990

How does solid fuel use compare with other major risk factors in developing countries?

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Percent of Total LDC Deaths</th>
<th>Percent of Total LDC DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malnutrition</td>
<td>14.9%</td>
<td>18%</td>
</tr>
<tr>
<td>Water/ Hygiene/ Sanitation</td>
<td>6.7%</td>
<td>7.6%</td>
</tr>
<tr>
<td>Solid Fuel Use</td>
<td>4.9%</td>
<td>4.4%</td>
</tr>
<tr>
<td>Unsafe Sex/ Unwanted Pregnancies</td>
<td>2.5%</td>
<td>3.7%</td>
</tr>
<tr>
<td>Alcohol</td>
<td>1.6%</td>
<td>2.7%</td>
</tr>
<tr>
<td>Occupation</td>
<td>2.5%</td>
<td>2.5%</td>
</tr>
<tr>
<td>Traffic Accidents</td>
<td>1.8%</td>
<td>2.2%</td>
</tr>
<tr>
<td>Tobacco</td>
<td>1.7%</td>
<td>1.4%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3.8%</td>
<td>0.9%</td>
</tr>
<tr>
<td>HIV/AIDS</td>
<td>0.2%</td>
<td>0.6%</td>
</tr>
<tr>
<td>Outdoor Air Pollution</td>
<td>0.7%</td>
<td>0.4%</td>
</tr>
</tbody>
</table>

*Estimates for other risk factors from The Global Burden of Disease, WHO/Harvard 1996
Indoor Air Pollution from Environmental Tobacco Smoke (ETS)

The biggest challenge in quantifying the burden of disease from ETS comes with determining how to estimate ETS exposure from information on smoking prevalence. Slide 13 below details the assumptions used in determining exposure to ETS for each region, and includes information on each of the three components (smoking prevalence, ventilation, and number of people exposed per smoker) that affect the exposure estimate.

**How do we estimate ETS exposure from smoking prevalence?**

*Exposure:*

\[
\text{Smoking Prevalence} \times \text{Ventilation Coefficient} \times \text{People exposed per smoker}
\]

**Smoking prevalence:**

- estimates from World Bank, 1999

**Ventilation coefficient:**

1 - (proportion of households using solid fuels)

- 97% of a cigarette smoked in EME results in ETS exposure, compared to 22% in India

**Number of people exposed per smoker:**

- Adults: 0.25 – 0.50
- Children: 0.25 – 1.0

The ventilation co-efficient was used to estimate the effective ETS exposure resulting from smoking indoors. A cigarette smoked in a tightly sealed house in EME would result in much greater indoor exposure to ETS than a cigarette smoked in a hut with a thatched roof and open doorway. In general, there seems to be a trend in household ventilation that is inverse to the energy ladder, so that shifts up the rungs of the energy ladder are associated with decreased ‘openness’ of homes (i.e. less open doors and windows). In the absence of regional differences in household ventilation, the ventilation coefficient was estimated to be 1-(proportion of households using solid fuels) in each region. For example, 97% of a cigarette smoked in EME could result in ETS exposure, compared to 22% in India.

Slide 14 lists the health outcomes addressed by this project, and the relevant populations to which the relative risk estimates were applied.

**GBD from ETS:**

<table>
<thead>
<tr>
<th>Health Outcomes Addressed</th>
<th>Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower Respiratory Infections (LRI)</td>
<td>&lt;5 years</td>
</tr>
<tr>
<td>Otitis Media</td>
<td>&lt;5 years</td>
</tr>
<tr>
<td>Asthma</td>
<td>&lt;15 years</td>
</tr>
<tr>
<td>Lung Cancer</td>
<td>&gt;15 years</td>
</tr>
</tbody>
</table>
Slides 15 and 16 present our working estimates of the burden of disease from ETS exposure. While ETS is generally regarded as a developed country exposure, these findings suggest that the Middle Eastern Crescent, China, and Sub-Saharan Africa bear a large proportion of the burden of disease from ETS. In addition, most of the deaths attributable to ETS exposure are occurring in young children from lower respiratory infections. While reliable information on smoking prevalence trends are not currently available for many regions of the world, the certain increase in smoking prevalence that is taking place in developing countries is likely to result in an even greater disease burden in the future.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Deaths</th>
<th>Episodes</th>
<th>DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td>LRI</td>
<td>61,000</td>
<td>6,146,000</td>
<td>2,141,000</td>
</tr>
<tr>
<td>Otitis Media</td>
<td>400</td>
<td>3,510,000</td>
<td>16,000</td>
</tr>
<tr>
<td>Asthma</td>
<td>140</td>
<td>201,000</td>
<td>69,000</td>
</tr>
<tr>
<td>Lung Cancer</td>
<td>5,000</td>
<td>6,000</td>
<td>48,000</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td>67,000</td>
<td>9,863,000</td>
<td>2,274,000</td>
</tr>
</tbody>
</table>

Indoor Air Pollution from Radon

Estimates of mortality from lung cancer associated with radon exposure are only reliable for the U.S., where there is information available on residential radon exposures. Attributable risks from the NAS Beir VI report were applied to 1990 lung cancer mortality for the U.S from the National Center for Health Statistics to estimate the deaths and YLL from lung cancer in the U.S.

As these attributable risks are based on U.S. levels of residential radon exposure and smoking prevalence (due to the strong interaction between radon exposure and smoking), they are not directly generalizable to other regions of the world. However, as Slide 17 suggests, these results suggest a potentially large global burden of disease.
Lung Cancer from Radon in The United States, 1990

<table>
<thead>
<tr>
<th></th>
<th>DEATHS</th>
<th>YLL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>11,000</td>
<td>78,000</td>
</tr>
<tr>
<td>Female</td>
<td>6,500</td>
<td>47,000</td>
</tr>
<tr>
<td>TOTAL</td>
<td>17,500</td>
<td>125,000</td>
</tr>
</tbody>
</table>

Discussion

Slide 18 lists some of the limitations of attributable risk, and then continues by addressing the problem of determining avoidable burdens. There is a fundamental difference between attributable and avoidable risk, which is demonstrated here by comparing ARI, an acute health outcome influenced by recent exposures, with chronic diseases such as COPD, which are influenced by an accumulation of exposures over time. As this Slide 19 suggests, an intervention put in place today could vastly affect the incidence of ARI in the future. However, the incidence of chronic diseases will decline over time, as illness in the future can still be attributed to accumulated past exposures.

Discussion

- Limitations of attributable risk
  - How can we deal with interaction between different risk factors and health outcomes?
  - How can we calculate attributable risk when we have multiple risk factors for the same health outcome?
- Determining avoidable burdens
  - Lack of trend information for fuel use, smoking
  - What should be the counterfactual level of exposure?
Finally, it is important to underscore the fact that attributable risk only looks at one class of exposure and outcome at a time. In reality, there are a complex set of interactions between multiple risk factors and health outcomes, which cannot be addressed by this framework. To demonstrate this, Slide 20 provides a schematic representation of a ‘causal web’ of household environmental exposures and children’s health. Indoor air pollution is clearly an important risk factor in and of itself. However, when located within the context of the household environment, the complexities involved with characterizing the health effects of interrelated risk factors becomes apparent.
Annex 6.6: Estimating the global burden of disease from environmental exposure to lead

Lorna Fewtrell
Centre for Research into Environment and Health (CREH)

Lead is a normal constituent of the earth’s crust. It is also abundant, easy to mine and has a number of uses. Unfortunately for man it is also highly toxic and doesn’t degrade in the environment. Lead has been implicated in a number of health effects, ranging from severe encephalopathy and death to subtle effects on IQ. For the purposes of the initial estimate of the global burden of disease relating to lead a small number of effects have been selected. In children these include:

- IQ loss
- Colic
- Anaemia
- Nephropathy
- Encephalopathy
- Death

Because of its range of uses, people are exposed to lead through air, water and food. Exposure leads to a measurable burden of lead within the body, which is most often assessed as blood lead level.

CREH has been fortunate to obtain a draft copy of the ‘Lead Information’ database that is currently under development by CDC. This is serving as the primary source of information on lead exposure. The database contains over 700 references reporting human lead levels, over which, over 85% of the studies report blood lead level.

The approach for determining exposure involves examining the blood lead levels in the database on a regional basis (driven by the 14 regions defined by the World Health Organization). Results from individual countries within any one Region are examined statistically before being pooled. Data from children are being analysed separately from adults, and where there are sufficient data it is hoped to examine children under the age of five as an additional group. The mean data (derived from the individual studies reported in the database) appear to be log normally distributed, therefore the mean and standard deviation of the pooled studies can be used to determine a probability density function. Health effects can then be superimposed onto the exposure distribution (pdf) to determine the number of people affected and to what extent.

Slide 1 shows the pdf derived from data from Canadian children (from studies conducted between 1984 – 1992), with the bands representing health effects, determined from cut off points. This results in an estimate of almost 300 children/1000 affected by IQ reduction. With IQ reduction being the only health effect seen.
Canadian exposure and health effects

Slide 1

Amr B exposure and health effects

Slide 2
The situation (from studies conducted between 1980 – 1996) is rather different in the Amr B region where there is a far greater burden of disease in children due to lead (Slide 2). The complete spectrum of health effects can be seen, including an estimate of three deaths/1000 population.

The last stage, in terms of the global burden of disease is to convert these figures to Disability Adjusted Life Years (DALYs) using a severity weighting.

The use of the blood lead level data, to derive probability density functions, is not without its problems.

- Many studies in the database concentrate on high-risk groups, such as occupationally exposed adults or children living close to a lead smelter. Groups can be split into ‘controls’ and ‘exposed’ but then there is the additional problem of ascribing population figures to each group.

- Blood lead level is not the ideal marker. It can be fraught with contamination problems, especially if capillary samples are taken, and it indexes recent, rather than long-term, exposure.

- Many of the studies do not report their quality control measures, so it is not possible to determine if, for example, lead free sampling kit and reagents have been used.

- The database covers studies ranging over a number of years. Many countries have implemented lead reduction programmes, which have had a significant effect on lead exposure; the dates, measures taken and the effectiveness of these programs vary from country to country.

Overall, the use of probability density functions provides a simple and transparent way of describing lead exposure. Their use allows easy visual comparison between areas. With further refinements, to account for some of the problems outlined above, they represent a useful way forward in terms of describing exposure to environmental contaminants.
Annex 6.7: Comparative risk assessment of the health effects of climate change

Tony McMichael, Diarmid Campbell-Lendrum, Sari Kovats
London School of Hygiene and Tropical Medicine

Many aspects of human health are highly sensitive to temporal and geographic variations in climate. It is clear that the global climate has changed significantly over the last century, characterised principally by an increase in average temperatures. There is accumulating evidence both that this change is largely due to anthropogenic emission of greenhouse gases (GHGs) (IPCC 1996), and that the resulting climate change is likely to have significant, mainly adverse, affects on human health (McMichael et al. 1996, Patz et al. 2000). Climate change caused by GHG emissions can be considered an environmental risk factor for health, and a risk factor that may be altered by human intervention. WHO has therefore requested an assessment of the human health benefits of amelioration of climate change through reduction in GHG emissions, using the comparative risk assessment (CRA) framework.

PROPOSED METHODS:

Due to the long-term nature of the relationship between human actions, GHG emissions and climate, actions taken to reduce climate change now are likely to result in avoidance of future, rather than present, health burdens. Estimation of climate change effects on health is therefore a predictive exercise, comparing the expected health consequences of the future climate scenarios that are predicted to result from different, more or less feasible, changes to GHG emissions trajectories (Slide 1). Following CRA terminology, we propose to use the following definitions for a comparison of the possible future health effects of climate change.

Risk factor:
Future changes in global climate attributable to increasing atmospheric concentrations of greenhouse gases (GHGs).

Units of “exposure”:
Discrete climate scenarios derived from alternative future trajectories of GHG emissions, as defined by the Intergovernmental Panel on Climate Change (IPCC) in 1995.

Reference scenario:
Business as usual (BAU), i.e. unmitigated current emissions trends (reference scenario)

Alternative or counterfactual scenarios for comparison:

1) Stabilization at 750 ppm CO$_2$-equivalent (can be considered the feasible minimum)

2) Stabilization at 550 ppm CO$_2$-equivalent (plausible minimum)

3) 1961-1990 levels of GHGs and associated climate, (the World Meteorological Office definition of baseline climate, which can be considered the theoretical minimum).

Time slices for estimation:
Averages from 30 year time-windows, centred on the 2020s and 2050s.
Estimation of the attributable and avoidable burdens of disease (Slide 2) may be generated by integrated assessment modelling, summarised in Slide 3. This consists of linking predictive models describing the chain from GHG emissions to climate, to impacts on health-related outcomes, to health outcomes recognised under the GBD system (i.e. which have either ICD or GBD codes).

This preliminary assessment will be based on existing models for specific health impacts, rather than new analyses. Although such modelling is in its infancy and remains subject to multiple uncertainties, some form of quantitative predictive model is available for a range of health impacts.
### Methodology for Assessment of Environmental Burden of Disease

#### a) Outcomes which can be estimated directly or (often very) indirectly from existing models.

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
<th>GBD code</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct impacts of heat and cold:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence of deaths due to</td>
<td>Cardiovascular diseases</td>
<td>(G089)</td>
</tr>
<tr>
<td></td>
<td>Respiratory diseases</td>
<td>(G094)</td>
</tr>
<tr>
<td>Incidence of non-specific hospital admissions</td>
<td></td>
<td>(G136)</td>
</tr>
<tr>
<td>Food and water-borne disease:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence of episodes of</td>
<td>Diarrhoea</td>
<td>(G009)</td>
</tr>
<tr>
<td>Vector-borne disease:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence of cases of</td>
<td>Malaria</td>
<td>(G018)</td>
</tr>
<tr>
<td></td>
<td>Dengue</td>
<td>(G027)</td>
</tr>
<tr>
<td></td>
<td>Schistosomiasis</td>
<td>(G022)</td>
</tr>
<tr>
<td></td>
<td>Trypanosomiasis</td>
<td>(G020)</td>
</tr>
<tr>
<td></td>
<td>Onchocerciasis</td>
<td>(G025)</td>
</tr>
<tr>
<td></td>
<td>Leishmaniasis</td>
<td>(G023)</td>
</tr>
<tr>
<td></td>
<td>Chagas disease</td>
<td>(G021)</td>
</tr>
<tr>
<td></td>
<td>Lymphatic filariasis</td>
<td>(G024)</td>
</tr>
<tr>
<td>Natural disasters:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence of deaths due to</td>
<td>Drowning</td>
<td>(G129)</td>
</tr>
<tr>
<td></td>
<td>other unintentional injuries</td>
<td>(G131)</td>
</tr>
<tr>
<td>Risk of malnutrition</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalence of deficiencies in recommended calorie intake</td>
<td></td>
<td>(G048)</td>
</tr>
<tr>
<td>Lack of water</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incidence of death/diseases</td>
<td>attributable to water shortages</td>
<td>(G136)</td>
</tr>
</tbody>
</table>

#### b) Health impacts for which no quantitative models exist, which may therefore have to be assessed qualitatively.

- Health impacts of population displacement due to natural disasters, crop failure, water shortages
- Possible outcomes include all health impacts of refugee status, increased risk of conflicts.
- Health effects of reduction in biodiversity and ecological stability
- Increased risk of outbreaks of new or previously rare infectious diseases.

### Example of Quantitative Impact Assessment Modelling:

Substantial research effort has been directed towards estimating the potential effect of future climate change on malaria transmission. Martens et al. (1999) have integrated published estimates of the effects of temperature on the main components of vectorial capacity (slide 4), in order to estimate the potential effect of future climate change on the geographic distribution of malaria (slide 5), and hence the potential change in the future population at risk of the disease.

Such models are flexible, and may be applied to alternative scenarios describing future climate and other consequences of population growth and development. For example, new alternative scenarios defined by IPCC (SRES scenarios) include not only future changes in climate, but associated changes in population and development (slide 6). When these are applied to the malaria model, they result in slightly different estimates of the number of people at risk of malaria, although substantial increases are still predicted under each scenario (slide 7). Changes in the proportion of people at risk are less dramatic, but still significant (slide 8).
SLIDE 4

ALTERNATIVE METHODS FOR MODELLING CLIMATE EFFECTS:

ALTERNATIVE METHODS FOR MODELLING CLIMATE EFFECTS:
Biological process modelling
Uses accepted theory to integrate published effects of climate on components of transmission cycle (e.g. Martens, 1999).

Baseline climate

Greenhouse gas emissions

Baseline climate

SLIDE 5

2020s Changes in Falciparum Malaria Transmission Potential, vs. baseline scenario (HadCM2, with vector limits)
(Martens, et al., 1999)

2050s

2080s

SLIDE 6

A1. World Markets
Very high economic growth
2100 population: 7 billion
Medium mitigation, high adaptation
Temp (2050s) +1.6°C
Rainfall: +11% winter, -7% summer

B1. Global Sustainability
High economic growth
2100 population: 7 billion
High mitigation, low adaptation
Temp (2050s) +0.8°C
Rainfall: +7% winter, -1% summer

A2. Provincial Enterprise
Moderate economic growth
2100 population: 15 billion
No mitigation, low adaptation
Temp (2050s) +2.2°C
Rainfall: +14% winter, -10% summer

B2. Local Stewardship
Low economic growth
2100 population: 10 billion
Variable mitigation and adaptation
Temp (2050s) +1.6°C
Rainfall: +11% winter, -7% summer

SLIDE 7

GLOBAL TOTAL POPULATIONS AT RISK UNDER 3 CLIMATE SCENARIOS, BY CATEGORY OF MALARIA OCCURRENCE

SLIDE 8

Climate-attributable excess persons at risk of malaria (millions)

Population (millions)

2050s

2080s
UNCERTAINTIES AND KNOWLEDGE GAPS:

There are considerable uncertainties in predicting the effects of future climate change on health. The most important of these relate to:

- Future emissions of greenhouse gases (based on population and economic growth etc.).
- Effects of simplifying assumptions and choice of initial conditions and parameter values within global climate models.
- Natural variability of climate.
- Effects of simplifying assumptions and choice of initial conditions and parameter values within health impact models.
- Levels and effects of non-climate determinants of health in the future - particularly socio-economic aspects that determine "vulnerability".
- Limited opportunity for directly assessing the accuracy of predicted health outcomes.

Some of these will be addressed as more baseline data is collected, as the field of impact assessment modelling expands and improves, and as alternative approaches are compared. For example, Rogers and Randolph (in press) describes direct statistical correlations between climate variables and the current distribution of malaria (rather than attempting to model individual components of the transmission cycle) and links these derived relationships to climate prediction models (Slide 9).

SLIDE 9

Although the resulting predictions of changes in the number of people at risk are much lower than previous estimates, it is not yet clear how much the discrepancies are due to the different modelling approaches employed, or differences in definition of the outcome predicted: populations living in areas climatically suitable for malaria vs. living in areas where malaria is actually predicted to occur. It should be noted that due neither model directly estimates the most important outcome for this exercise; proportional changes in numbers of malaria cases.

CONCLUSIONS:

The primary objective of the CRA exercise is to generate the best estimates that can currently be made of the net health effects of future climate change. Perhaps more importantly, the CRA
should also stimulate testing and improvement of existing models, generation of new models for health impacts which have not yet been investigated, and help to focus future modelling efforts on the questions of greatest relevance to policy.

REFERENCES:


