Annu. Rev. Environ. Resour. 2005. 30:291–333
doi: 10.1146/annurev.energy.30.050504.144424

HOW ENVIRONMENTAL HEALTH RISKS CHANGE WITH DEVELOPMENT: The Epidemiologic and Environmental Risk Transitions Revisited

Kirk R. Smith[1] and Majid Ezzati[2]
[1]*School of Public Health, University of California, Berkeley, California 94720-7360; email: krksmith@berkeley.edu*
[2]*School of Public Health, Harvard University, Boston, Massachusetts 02215; email: mezzati@hsph.harvard.edu*

Key Words burden of disease, household risks, community risks, global risks, mortality transition, global health, environmental Kuznets curve

■ **Abstract** Transition frameworks are used to envision the important changes that occur during economic development from poor to middle-income or rich countries. We explain the derivation of and use data from the Global Burden of Disease (GBD) and Comparative Risk Assessment (CRA) projects of the World Health Organization (WHO) to explore the classic epidemiologic transition framework, which describes the changes in causes of illness and death during economic development. We provide the first full empirical test of the environmental risk transition framework, which describes the shift in environmental risks during development from household, community, and global risk factors. We find that the simplistic conclusions commonly drawn about the epidemiologic transition, in particular the increase in chronic diseases with development, are not supported by current data; in contrast, the conceptual framework of the environmental risk transition is broadly supported in a cross-sectional analysis. We also describe important kinds of environmental health risks and diseases that are not well estimated using current methods.

CONTENTS

INTRODUCTION: TRANSITION FRAMEWORKS IN DEVELOPMENT 292
The Demographic Transition 292
The Epidemiologic Transition 293
The Risk Transition 294
THE GLOBAL BURDEN OF DISEASE PROJECT 297
Measuring Nonfatal Health Outcomes in the Global Burden of Disease 298
HEALTH OUTCOMES AND HEALTH RISK FACTORS 299
THE COMPARATIVE RISK ASSESSMENT PROJECT 300
Methods for Estimating the Health Effects of Risk Factors 301
Population Attributable Fraction for Individual Risk Factors 301
Estimated Mortality and Burden of Disease Attributable to Selected Risks 306

EPIDEMIOLOGIC TRANSITION REVISITED 309
Mortality Transition Revisited 309
Burden of Disease Transition 309
QUANTIFYING THE ENVIRONMENTAL RISK TRANSITION 314
Measuring Development 315
Risk Metric 319
Household Environmental Risks 319
Community Environmental Risks 320
Global Environmental Risks 321
ENVIRONMENTAL RISK TRANSITION REVISITED 321
Risk Overlap 323
The Environmental Kuznets Curve 323
LIMITATIONS OF EPIDEMIOLOGIC RISK ASSESSMENT 324
Environment as a Super Distal Risk Factor 325
High-Consequence, Low-Probability Hazards 325
CONCLUSION 327

INTRODUCTION: TRANSITION FRAMEWORKS IN DEVELOPMENT

Attempting to understand the process and impacts of socioeconomic development, both positive and negative, has occupied much scholarly and policy effort. In spite of this attention, it is still difficult to make generalizations that both fit a significant number of observed situations in different countries and can be used to mold successful policy. Although most attention is paid to development measured in economic terms, there are also development indices that include social, health, and environmental parameters. Perhaps driven by the common distinction made between poor and rich, developed and less-developed, or similar terms, and the persistent use of such categories in international negotiations and foreign aid decisions, the most enduring concept used in development discussions are various transition frameworks.

The Demographic Transition

The earliest to be employed and most fundamental of these frameworks is the "demographic transition," which was first described in the 1940s (1, 2). In its simplest formulation, the demographic transition lays out the shift in societies during development from a situation of high fertility (birth rates) and high mortality (death rates), when population sizes were fairly stable over long periods, although perhaps varying dramatically because of epidemics, famines, and other reasons. Studies of the historical patterns show that mortality has tended to drop before fertility, leading to a period of rapid growth in population size while the two rates are unequal. Eventually, however, as countries continue to develop, fertility eventually drops to roughly match mortality again, but at low levels instead of the high levels before development, leading again to more or less stable populations.

Volumes have been written to describe and explain the demographic transition in different locations at different points in history. Acrimonious debate has also sometimes erupted, principally focused on whether observed historical patterns in one set of countries (e.g., the currently developed countries in Europe) can be used as predictors of what will happen in the currently developing countries in Asia, Africa, and elsewhere. Perhaps the most crucial element of that debate is whether fertility declines will inevitably follow mortality declines. It is pointed out that some countries, Costa Rica for example, have had large mortality declines without consequent fertility declines until special efforts were made to encourage them (3). Critics also argue that even if the birth rate declines, it may do so too slowly, leaving too long a time with large differentials between mortality and fertility and thus large and rapid growth in population sizes. Leaving aside the question of whether the demographic transition framework can be used as prediction, it is clearly a useful framework for management, i.e., a helpful way of laying out what is happening in a population.

The Epidemiologic Transition

Around 1970, an additional framework, the "epidemiologic transition," was proposed to describe the changes in health characteristics of developing societies preceding and during the demographic transition (4). It noted that there tended to be a shift in the causes of ill health during development from infectious, communicable, or acute diseases, such as measles, malaria, and diarrhea, to noninfectious, noncommunicable, or chronic diseases, such as cancer, heart disease, and stroke. The evidence originally related solely to causes of death, for which, unlike disease incidence and prevalence, records are available in most parts of the world. Thus, a better term might be the "mortality transition."

As with the demographic transition, longitudinal evidence for the mortality transition was available for only a few European countries that had reliable population statistics over long periods. The simple division of causes of death to infectious and noninfectious diseases was soon seen not to be sufficient, however. Thus, these are now commonly divided into three more complex categories:

I. Traditional, infectious, nutritional, perinatal, and maternal causes;

II. Modern, cancer, heart, neuro-psychiatric, chronic lung, diabetes, and congenital causes;

III. Nontransitional injuries, both unintentional (accidents) and intentional (violence).

Although there is always a risk in extrapolating cross-sectional data to what might happen in any one place over time, evidence from current conditions around the world does tend to support the classic epidemiologic/mortality framework in which clear trends for the proportion of deaths in categories I (down) and II (up) and no obvious trend for the proportion of deaths in category III (flat) in regions ordered across development categories by current per capita income or other such

measures. The demographic and mortality transitions are closely linked in that mortality from category I diseases is highest in young children and for category II diseases highest in older adults. As the early public health measures in developing countries involve vaccination, nutrition, and sanitation, which mainly address child mortality, the proportion of category I deaths goes down. Since everyone dies of something, therefore, category II deaths rise, with category III (nontransitional) injuries, showing no clear trends as a percentage of the total. This explains a substantial overall drop in mortality rates and extension of life expectancy, the first stage of the demographic transition. See Reference 5 for a modern critique of the epidemiologic transition framework.

The Risk Transition

Proposed around 1990 was yet a third related framework, the "risk transition" (6, 7), which identified a shift in the character of environmental risks during the development period[1] and was later applied in studies of urbanization (10) and of China (11). The idea was refined to focus on the logic that before there could be a shift in the causes of death and disease, there needed to be a shift in the risk factors that lead to disease. In some cases, the change in diseases might come quickly upon changes in the risk factor (e.g., reduction in diarrheal diseases from better sanitation/water); in other cases, the shift in risk factor may come years or decades before the change in disease, for example, cancer from smoking. In general, risk factors and disease were more closely linked in time for category I compared to category II diseases, although not in every case. From a public health perspective, of course, society prefers to prevent rather than respond to diseases, and thus an understanding of the risk transition is vital for designing timely intervention strategies. The linkages among the three transition frameworks are shown in Figure 1.

In the case of the "environmental risk transition," the environmental factors leading to ill health were originally categorized as traditional and modern, in parallel to those for the mortality transition (7). Subsequently, as shown in Figure 2, there was a shift to a more explicit spatial framework and addition of a third category, resulting in a three-phase transition of environmental risks at the household, community, and global scales (12–14). [The three phases have also been termed the brown, gray, and green agendas (15).] This categorization was based on the premise that the major environmental causes of category I diseases were due to problems at the household level (e.g., poor household fuel, water, sanitation, ventilation, food quality). As these were addressed during development, there was an increase in the relative importance of the major environmental causes of category

[1]Some identification of the importance of risk, as opposed to disease, is partly embedded in the key epidemiologic transition papers (4, 8, 9), using terms like diseases of pestilence and affluence.

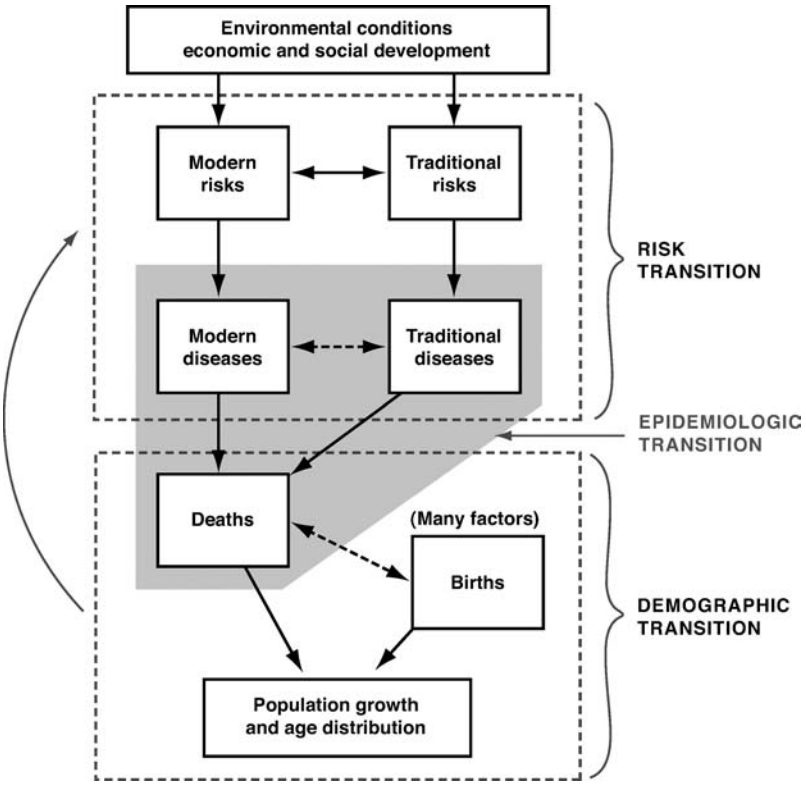

Figure 1 Relationship between the transition frameworks discussed in the text. Risk factors must change before patterns of disease and death change, but changes in death rates differentially affect age distribution, which in turn has an impact back on risk factors and consequent disease and death patterns.

II diseases, which operated at the community level (i.e., urban air quality, occupational hazards, toxic chemicals, motorization). As these were addressed in richer societies, however, a further transition occurred to increase the importance of environmental hazards at the global level (e.g., climate change) among environmental health risk factors.

Thus, to put it starkly, the environmental risk transition framework would indicate a tendency for societies to sweep environmental health problems out of the house and into the community during the first stages of development and then out from the community to the general global environment during later stages. Not addressed directly by this conceptual framework, however, was the scale of health risks, either in absolute or relative terms. How important were environmental risk factors within the overall health picture in terms of actual death and disease and in

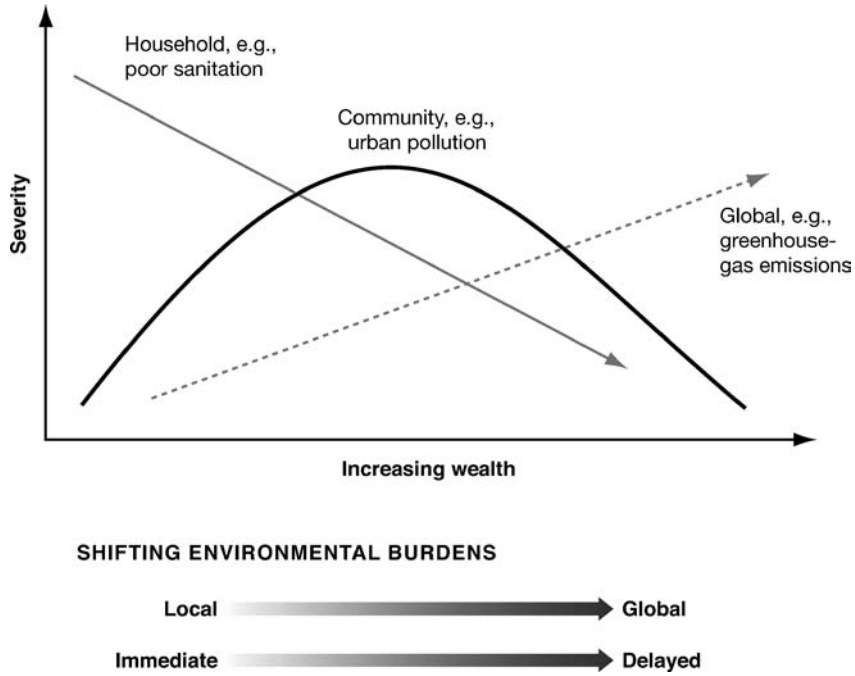

Figure 2 Environmental risk transition framework in which household risks fall with development, community risks rise and then fall, and global risks rise throughout the development process.

proportion to the total, at each level of development and in each of the three stages (household, community, and global)? The obstacle to this analysis has been the absence of a complete coherent global database of environmental risk factors and related diseases that would provide information in a consistent manner to allow such analysis. Indeed, even the task of putting reliable numbers on the vertical scale of presentations of the classic epidemiologic (mortality) transition was also not possible until relatively recently because of the lack of a reliable global mortality database.

In this review, we use relatively new data from the Global Burden of Disease (GBD) and Comparative Risk Assessment (CRA) projects of the World Health Organization (WHO) to provide more empirical detail related to the epidemiologic transition and to provide the first empirical test of the environmental risk transition framework. We find that many of the conclusions drawn in the past about the epidemiologic transition are not supported by current data, but in contrast, the conceptual framework of the environmental risk transition is well supported.

THE GLOBAL BURDEN OF DISEASE PROJECT

As noted, until relatively recently, the international health community lacked a coherent, consistent, and comprehensive dataset describing the extent and distribution of ill health. Simple questions such as how many deaths were from what disease, at what age, in which part of the world could not be answered reliably. The absence of data and the inconsistencies in figures reported by different agencies and experts were likely an outcome of the natural tendency of interest groups to cite the higher end of uncertainty ranges, differences in treating evidence among groups, as well as true conceptual difficulties, especially with assigning deaths from multiple causes to one disease category (e.g., should tuberculosis deaths in an Human Immunodeficiency Virus (HIV)-positive individuals be accounted as HIV or tuberculosis?).

This is quite primitive compared to other major arenas in society. We expect that trade statistics will match imports and exports by region and commodity. So too, for example, with energy and food production and consumption, the net of births minus deaths for population growth, and financial flows. Until the mid-1990s, however, health policy professionals had to live with health databases that did not balance. Without such data, trying to determine the disease attributable to any one risk factor was lost in a sea of inconsistent and competing claims. Imagine, for example, trying to determine what fraction of energy is produced by coal if one had neither a reliable number for total energy use nor an agreed upon way to distinguish coal from oil.

Initiated in the early 1990s, the first version of such a database was originally published by the World Bank (16), and later versions were incorporated into the statistical apparatus of the WHO. To do so, it was necessary to bring together clinicians, disease experts, demographers, epidemiologists, statisticians, and others with relevant angles of view in a number of different fora around the world and impose a consensual discipline never before attempted. No longer could one disease interest group claim large numbers of deaths unchallenged because it would have to take into account the often directly conflicting estimates of other disease interest groups, which also had evidence among the same populations. Since all the groups were engaged together and obliged, in sum, not to exceed the known number of deaths, coherence and consistency were enforced.

The first complete publication of the GBD database occurred in 1996 using 1990 data (17). The WHO now publishes yearly updates of the GBD, which comes out in summary form in its annual World Health Reports with detailed versions available on the web (see http://www3.who.int/whosis/menu.cfm?path=whosis,burden,burden_estimates&language=e). These delineate more than 150 causes of death and illness (including injury) by age and sex, organized according to categories I through III, as described above. Analysis is conducted for 14 epidemiologic subregions on the basis of a combination of official WHO regions and child and adult mortality levels (18).

Measuring Nonfatal Health Outcomes in the Global Burden of Disease

Many diseases (e.g., neuropsychiatric conditions and hearing loss) and injuries may cause considerable ill health but no or few direct deaths. Therefore, separate measures of survival and of health status among survivors, although useful inputs into the health policy debate, need to be combined in some fashion to provide a single, holistic measure of overall population health. A full discussion of summary measures of population health (SMPH), their typologies, and their assumptions and performances is provided by Mathers et al. (19). In brief, summary measures of population health can be divided into two classes: health expectancies and health gaps. The bold curve in Figure 3 is an example of a survivorship curve for a hypothetical population. The survivorship curve indicates, for each age along the horizontal axis, the proportion of an initial birth cohort that remains alive at that age. The area under the survivorship function is divided into two components, *A*, which is time lived in full health, and *B*, which is time lived at each age in a health state less than full health (e.g., with one or multiple diseases and injuries).

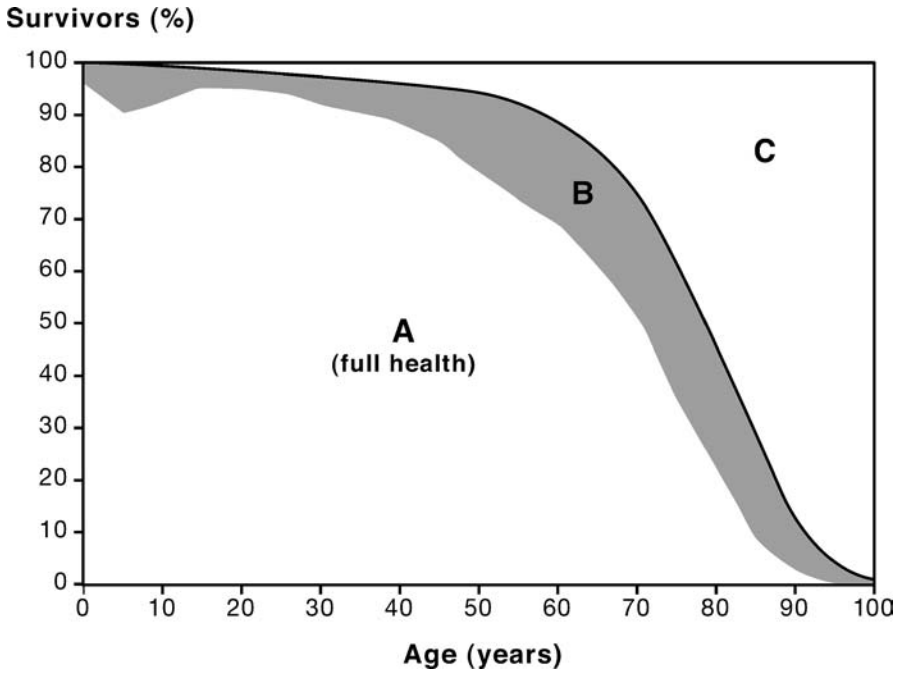

Figure 3 Hypothetical survival curve. The upper line shows the proportion of population alive at each age and the lower line shows the proportion in full health. Therefore, area *A* is time lived in full health, *B* is time lived in a health state less than full health, and *C* time lost to mortality relative to some normative life expectancy.

The familiar measure of life expectancy at birth is simply equal to $A + B$ (the total area under the survivorship curve). A "health expectancy" SMPH is generally of the form $A + f(B)$, where f is a function that weights time spent in B by the severity of the health states that it represents (further, B can be divided into B_1, B_2, etc., which indicate different severities of health loss). In contrast to health expectancies, "health gaps" quantify the difference between the actual health of a population and some stated norm or goal for population health. The health goal implied by Figure 3 is for everyone in the entire population to live in ideal health until the age indicated by the vertical line enclosing area C at the right (100 years in this hypothetical example). By selecting a normative goal for population health, the gap between this normative goal and current survival, area C, quantifies premature mortality. A health gap is generally of the form $C + g(B)$, where g is a function that weights time spent in B by the severity of the health states that B represents.

Contributions of diseases and risk factors in this review are examined in a health gap framework, referred to as the "burden of disease" (17). Burden of disease, measured in disability-adjusted life year (DALY), is the sum of years of life lost (YLL) to premature mortality and years of life lived with disability (YLD). Definitions, assumptions, and methods for calculating DALYs (YLDs + YLLs) are described by Murray (20). One DALY is approximately equivalent to the loss of one year of life in perfect health.

HEALTH OUTCOMES AND HEALTH RISK FACTORS

A complete global database of death, disease, and injury by age, sex, and region allows a broad set of additional analyses, such as systematic examinations of more distal risk factors for ill health as compared to the proximate causes such as particular diseases. Indoor air pollution (IAP) is a more distal cause of death than lung cancer, for example, and poor building ventilation or poverty would be even more distal. Even though only lung cancer would be listed on a death certificate and in the GBD database itself, some fraction is attributable to IAP, and with appropriate data and models, the total burden of lung cancer and other diseases due to IAP can be estimated. Furthermore, some cases of disease are caused by multiple risk factors because the effects of more distal risk factors (e.g., poverty) are mediated through more intermediate ones (e.g., IAP) and because risks act in combination with one another (e.g., child mortality as a result of IAP is particularly high among malnourished children without access to health services). Multicausality also means that a range of interventions can be used for disease prevention, with the specific choice determined by factors such as cost, available technology, infrastructure, and cultural preferences.

Investigators from natural, physical, social, and health sciences increasingly address similar questions on the relationship between different types of causal risk factors and human health. One suggested framework identifies the following four

broad categories in the causal chain leading to loss of functional health status or death: (*a*) health determinants, themselves divided into broad distal groups, such as socioeconomic factors, and more proximal environmental and individual risk factors; (*b*) diseases and injuries; (*c*) functional health status; and (*d*) death (21). Other approaches, considering more complex interactions of causes of health, have also been proposed, such as those that address the social, behavioral, nutritional, and medical determinants of child health (22); the policy and social drivers of risk (23); or the technological and behavioral determinants of exposure to environmental risks (24). Similarly, Murray and colleagues (25, 26) provide a basic framework for examining environmental, behavioral, and physiological health risk factors in multiple layers of causality and with interactions.

Empirically, a substantial body of work has focused on the quantification of causes of mortality and more recently burden of disease (27, 28). Analysis of morbidity and mortality due to risk factors, however, has frequently been conducted in the context of methodological traditions of individual risk factors and in a limited number of populations (29–36). As a result, most such estimates have been made relative to an arbitrary, constant level of population exposure without standardization of the baseline exposure across risk factors. For example, the implicit baseline for much of the estimates of occupational injuries has been no work. Further, the criteria for evaluating scientific evidence on prevalence, causality, and hazard size have varied greatly across risk factors, resulting in lack of comparability of estimated population health impacts. Finally, the outcome has been morbidity or mortality due to specific disease(s), making comparison among different risk factors difficult.

THE COMPARATIVE RISK ASSESSMENT PROJECT

Using the GBD as a starting point, attributable burdens of disease for 26 important risk factors were calculated by over 100 investigators worldwide in the largest comparative risk assessment (CRA)[2] ever attempted (38, 39). The criteria for selection of risk factors in this WHO-managed CRA included (*a*) likely to be among the global or regional leading causes of disease burden; (*b*) not too specific (e.g., every one of the thousands of occupational chemicals) or too broad (e.g., environment or occupation considered as a single risk); (*c*) high likelihood of causality based on the collectivity of scientific knowledge; (*d*) reasonably complete data on exposure and risk levels or methods for extrapolation when needed; (*e*) potentially modifiable using known techniques. The risk factors were broadly divided into childhood and maternal undernutrition, other diet-related and physical inactivity

[2]Somewhat different definitions of CRA have been used (e.g., Reference 37), but in the WHO exercise, the focus was on commonality in methods, accounting frameworks, data consistency, validity of source databases, criteria for choosing evidence, metrics of risk and health outcome, and platforms for statistical analysis.

risks, addictive substances, sexual and reproductive health, environmental and occupational risks, and other selected risk factors that did not belong to these specific groups. Detailed descriptions of the environmental and occupational risk factors are presented in Table 1.

Under the principle of consensual discipline, the WHO CRA groups met together twice to develop standardized methodology and to agree about the quality and quantity of evidence that would be expected from each risk factor. In addition to the meetings, the CRA core and editorial team at WHO maintained close contact with all groups and produced standard databases (e.g., the GBD 2000 database of disease and injury outcomes, population projections) and data-processing tools needed by all groups. All chapters were anonymously peer reviewed, multiple times if appropriate. The differences in data availability were substantial in many cases. There were, for example, large blood pressure and childhood nutrition surveys in many parts of the world for use in developing exposure distributions. Risks such as blood pressure and cholesterol benefited from dozens of large double-blind placebo-controlled randomized interventions (the "gold standard" of epidemiology) to determine exposure-response relationships and causality. The climate change group, by contrast, had little evidence of either type and had to rely on climate forecast and health impact models.

Methods for Estimating the Health Effects of Risk Factors

Mathers et al. (21) describe two traditions for causal attribution of health outcomes or states: categorical attribution and counterfactual analysis. In categorical attribution, an event such as death is attributed to a single cause (such as a disease or risk factor) or group of causes according to a defined set of rules. An example of categorical attribution is provided by the International Classification of Disease (ICD) system for attribution of causes of death (40). In counterfactual analysis, the contribution of one or a group of risk factors to disease or mortality is estimated by comparing the current (or expected future) disease burden with the levels that would be expected under some alternative hypothetical scenario, referred to as the counterfactual (see Reference 41 for a discussion of conceptual and methodological issues in use of a counterfactual). In theory, causal attribution of the burden of disease to risk factors can be done using both categorical and counterfactual approaches. For example, categorical attribution has been used in attribution of diseases and injuries to occupational risk factors in occupational health registries (34) and attribution of motor vehicle accidents to alcohol consumption. Categorical attribution to risk factors, however, overlooks the multiple causes of many diseases (42).

Population Attributable Fraction for Individual Risk Factors

The contribution of a risk factor to disease or mortality relative to some alternative exposure scenario [i.e., "population attributable fraction" (PAF) defined as the

TABLE 1 Comparative Risk Assessment environmental and occupational risk factors, exposure variables, theoretical minima, and disease outcomes[a]

Risk factor	Exposure variable	Theoretical-minimum-risk exposure distribution	Disease outcomes	Attributable mortality (thousands of deaths)[a]	Attributable disease burden (% total global burden of disease)[a]
Environmental risks					
Unsafe water, sanitation, and hygiene	Six scenarios, ranging from regulated water and sanitation with hygiene through to no improved water supply and no improved sanitation	Absence of transmission of diarrheal disease through water, sanitation, and hygiene	Diarrheal diseases	1727	3.7
Urban air pollution	Estimated annual average particulate matter concentration for particles with aerodynamic diameters less than 2.5 or 10 microns ($PM_{2.5}$ or PM_{10})	7.5 $\mu g/m^3$ for $PM_{2.5}$ 15 $\mu g/m^3$ for PM_{10}	Mortality from combined respiratory and selected cardiovascular diseases in adults >30 years of age, lung cancer, acute respiratory infection mortality in children <5 years of age, cardiovascular and respiratory morbidity[b]	798	0.9

Indoor air pollution from household use of solid fuels	Household use of solid fuels and ventilation	No household solid fuel use	Acute lower respiratory infections in children <5 years of age, chronic obstructive pulmonary disease, lung cancer (coal); low birth weight,[b] cataracts,[b] tuberculosis,[b] asthma,[b] lung cancer from biomass[b]	1619	2.6
Lead exposure	Current blood lead levels	0.016 μg/dl blood lead levels[d]	Cardiovascular diseases, mild mental retardation, anemia,[b] gastrointestinal effects,[b] nervous and reproductive system effects,[b] social consequences of IQ loss[b]	233	0.4
Global climate change	Climate scenarios based on various carbon emissions and concentrations	1961–1990 concentrations	Diarrhea, flood injury, malaria, malnutrition and associated disease outcomes, dengue fever,[b] cardiovascular mortality,[b] effects arising from population movement[b]	152	0.4
Occupational risks					
Injuries	Current proportions of workers exposed to injury risk factors	Exposure corresponding to lowest rate of work-related fatalities observed: one per million per year for 16- to 17-year-olds employed as service workers in the United States	Unintentional injuries, intentional injuries[b]	308	0.7

(*Continued*)

TABLE 1 (*Continued*)

Risk factor	Exposure variable	Theoretical-minimum-risk exposure distribution	Disease outcomes	Attributable mortality (thousands of deaths)[a]	Attributable disease burden (% total global burden of disease)[a]
Carcinogens	Proportions of workers exposed to background, low, and high levels of workplace carcinogens	No work-related exposure above background to chemical or physical agents that cause cancer	Leukemia, lung cancer, mesothelioma,[b] cancers of multiple other sites[b]	118	0.1
Airborne particulates	Proportions of workers with background low and high levels of exposure	No work-related exposure above background	Chronic obstructive pulmonary disease, asthma, pneumoconiosis,[b] silicosis,[b] and asbestosis[b]	356	0.4
Ergonomic stressors	High, moderate, and low exposure based on occupational categories	Physical workload at the level of managers and professionals (low)	Lower back pain	0	0.1
Noise	High and moderate exposure categories (>95 dBA and 85–90 dBA)	Less than 85 dBA on average over eight working hours	Hearing loss	0	0.3

Selected important outcome-based burdens (diseases/injuries)[c]					
Childhood diseases	Environmental determinants not important compared to lack of vaccination	It is assumed that 100% could be eliminated by vaccination	Pertussis, poliomyelitis, diphtheria, measles, tetanus	13	3.5
Lack of malaria control	Environmental determinants are important	Rate of SEAR-B (Southeast Asia) taken as counterfactual (this is about 93% of the global total for malaria illness/death)		1004	2.5
Lack of road traffic accident control	Environmental determinants are important	Rate of WPR-A (Japan/Australia/NZ) taken as counterfactual (this is about 50% of the global total of injury/death from road traffic accidents)		630	1.3

[a]See (38, 39) for other risk factors and for data sources. Table 1 shows the estimated mortality and disease burden for each risk factor considered individually, relative to its own theoretical-minimum-risk exposure distribution. These risks act, in part, though other risks and act jointly with other risks. Consequently, the burden due to groups of risk factors will usually be less than the sum of individual risks, i.e., they should not be simply summed.

[b]Outcomes are those that are likely to be causal but not quantified owing to a lack of sufficient evidence on prevalence and/or hazard size.

[c]For outcome-based diseases/injuries, a proportion of deaths and disease burden could be avoided through implementation of known and new interventions that affect their multiple causes. The figures presented here are only approximate estimates, however, as they have not been applied for particular interventions by age, sex, and region, as was done in the comparative risk assessment exercise to determine the burden from risk factors in the first part of the table.

[d]Theoretical minimum for lead is the blood lead levels expected at background exposure levels. Health effects were quantified for blood lead levels above 5 μg/dl by epidemiologic studies.

proportional reduction in population disease or mortality that would occur if exposure to the risk factor were reduced to an alternative exposure scenario (43–45)] is given by the generalized "potential impact fraction" (*PIF*) in Equation 1. For each risk factor-disease pair, the population attributable fraction is then multiplied by total deaths or burden of disease to estimate the mortality or burden of disease attributable to that risk factor.

$$PIF = \frac{\int_{x=0}^{m} RR(x)P(x)\,dx \; - \; \int_{x=0}^{m} RR(x)P'(x)\,dx}{\int_{x=0}^{m} RR(x)P(x)dx}, \qquad 1.$$

where $RR(x)$ is the relative risk at exposure level x, $P(x)$ is the population distribution of exposure, $P'(x)$ is the counterfactual distribution of exposure, and m is the maximum exposure level.

The estimates of burden of disease and injuries due to risk factors in the CRA project are based on the counterfactual exposure distribution referred to as the "theoretical-minimum-risk exposure distribution," which would result in the lowest population risk, irrespective of whether currently attainable in practice (25, 26). Using the theoretical-minimum-risk exposure distribution as the counterfactual has the advantage of providing a vision of potential gains in population health by risk reduction from all levels of suboptimal exposure in a consistent way across risk factors. The theoretical-minimum-risk exposure distribution was zero for risk factors for which zero exposure could be defined and reflected minimum risk [e.g., no smoking (46) or no solid fuel use (47)]. For some risk factors, zero exposure was an inappropriate choice because there may be physical lower limits to exposure reduction [e.g., ambient particulate matter concentration (48) or occupational noise]. For these risk factors, the lowest levels observed in specific populations and epidemiologic studies were used to choose the theoretical-minimum-risk exposure distribution. The theoretical-minimum-risk exposure distribution for the environmental and occupational risk factors are reported in Table 1.

Estimated Mortality and Burden of Disease Attributable to Selected Risks

One major value of the CRA project is to enable policy makers to estimate the size and, with further analysis, cost-effectiveness (cost per unit health benefit) of potential interventions across a range of risks and diseases. The list of 26 risk factors in the WHO CRA, however, does not contain all of the major classes of risks and interventions of interest to policy makers. In some cases, this was due to lack of sufficient published worldwide exposure evidence for a full-scale risk assessment, as was the case for environmental tobacco smoke or food safety, for example. In other cases, however, it was because the risk factor produced unique impacts and thus required no sophisticated risk assessment. All measles cases, for example, can be attributed to lack of measles vaccination. Other disease outcomes,

whose risk factors were not sufficiently addressed in the CRA project, were those with diffuse determinants among environmental and behavioral factors (e.g., some cancers, perinatal conditions, and neuropsychiatric diseases) or those with more complex, multifactor etiology and often with heterogeneous determinants in different populations, and these are therefore difficult to quantify without data at a very small scale (e.g., tuberculosis and injuries).

Although not needing a sophisticated risk assessment, efforts to eliminate the so-called child cluster diseases (measles, tetanus, pertussis, diphtheria, polio), those killing mainly children and addressed by readily available and highly effective vaccines, are nevertheless clearly on the menu of important interventions to be considered by policy makers. Thus, here we added a few more important policy-relevant outcomes (diseases/injuries), those that cause at least 1% of the GBD or one million deaths annually, and their risk factors to the list of those addressed by the CRA project. Judgments, similar to those used in choosing the theoretical-minimum-risk distributions, must be applied to counterfactual levels for this additional set of health risks.

- Child-cluster diseases can be essentially eliminated, as seen in developed countries, through vaccination.
- Malaria, as demonstrated by historical experiences in Europe and the United States, can be essentially eliminated through household and community controls. We used the rate of malaria in WHO region SEA-B (Southeast Asia) as an achievable counterfactual level for tropical regions that currently experience malaria burden (49, 50).
- Road traffic accidents may not be entirely eliminated as long as motor vehicles remain important. In the absence of a systematic CRA, therefore, we arbitrarily set the feasible counterfactual level to be that of the lowest per capita burden in the world with high levels of vehicle use, the WHO region WPR-A (Japan/Australia/NZ), which is about half the global rate (51)[3].

Figures 4*a*,*b* show the contribution of the resulting leading global risk factors to all causes of mortality and to the burden of disease. The different ordering of risk factors in their contributions to mortality and disease burden reflect the age profile of mortality (e.g., deaths in younger ages make larger contributions to disease burden) and the nonfatal effects (e.g., risks with nonfatal health outcomes such neuropsychological diseases and injuries make more contributions to disease burden than to mortality). Distributions of deaths and disease burden for the environmental risks by age and by gender are also shown in Table 2.

[3]In addition, we use the regional distribution of road traffic accident deaths in 2000 from a detailed recent study (51), adjusted to the totals in the 2000 GBD.

TABLE 2 Distribution of risk-factor-attributable mortality and burden of disease by age and sex[a]

	Mortality						Disease burden					
	0–4	**5–14**	**15–59**	**≥60**	**Males**	**Females**	**0–4**	**5–14**	**15–59**	**≥60**	**Males**	**Females**
Environmental risks												
Unsafe water, sanitation, and hygiene	68	5	13	14	52	48	77	8	13	3	51	49
Urban air pollution	3	0	16	81	51	49	12	0	40	49	56	44
Indoor air pollution from household use of solid fuels	56	0	5	38	41	59	83	0	8	9	49	51
Lead exposure	0	0	41	57	66	34	75	0	16	8	55	45
Global climate change	86	3	6	5	49	51	88	5	6	1	49	51
Occupational risks												
Injuries	0	0	85	14	94	6	0	0	95	5	93	7
Carcinogens	0	0	28	72	85	15	0	0	51	49	83	17
Airborne particulates	0	0	17	83	74	26	0	0	65	35	77	23
Ergonomic stressors	0	0	0	0	0	0	0	0	95	5	59	41
Noise	0	0	0	0	0	0	0	0	89	11	67	33
Selected diseases/ injuries with environmental determinants												
Childhood diseases	79	15	5	1	50	50					50	50
Malaria	85	7	6	1	47	53	86	8	6	0	47	53
Road traffic accidents	4	8	71	17	71	29	7	16	74	14	70	30

[a]All numbers show the percentage of total risk-factor-attributable deaths or DALYs in the specific subgroup (39, 51a).

EPIDEMIOLOGIC TRANSITION REVISITED

With the GBD database, it is possible for the first time to evaluate the health transition framework in more systematic ways, both in terms of mortality and more complete approaches, such as ill health and risk factors.

Mortality Transition Revisited

Shown in Figure 5 are representations of the mortality transition, using only the simple distinctions among the four income categories used by the World Bank.

- Figure 5*a* shows the classic mortality transition in terms of proportion of total deaths in 2002. The figure illustrates the decline of category I and the rise of category II diseases with increasing income, and the lack of trend for category III. Because the total at each income class is always 100%, however, it does not indicate whether any progress has been made in overall mortality rates.
- Figure 5*b* shows the actual numbers of deaths in each category at each income level, illustrating the large number of deaths from infectious diseases still existing in the poorest parts of the world. The numbers for the different income levels, however, are not comparable because they have different populations.
- Figure 5*c* presents the results in terms of deaths per 1000 population, thereby correcting for population size. Here the size of the infectious risk in poor countries persists, but an apparent increase in chronic risks in rich countries emerges. Even more striking, perhaps, total mortality, which falls dramatically from poor to middle-income countries, actually seems to rise in the shift from middle-income to rich countries.
- Corrected also for age distribution by standardizing according to the world age distribution in 2002, Figure 5*d* shows that this apparent rise in category II deaths is just an artifact of the different age distributions within the regions. Rich countries do not have higher true death rates than poor countries in any disease category, but rather larger proportions of old people, who have higher death rates in every society. This figure represents the true mortality risks as experienced by individuals within each income group as they go through their life course. As shown, the mortality risks for all three categories of disease decline with development, by factors of 18, 1.8, and 2.5, for categories I, II, and III, respectively, when comparing the richest and poorest country groups.

Burden of Disease Transition

As the GBD database also coherently incorporates time lost to premature death and to nonfatal health outcomes (diseases and injuries) weighted by a disability

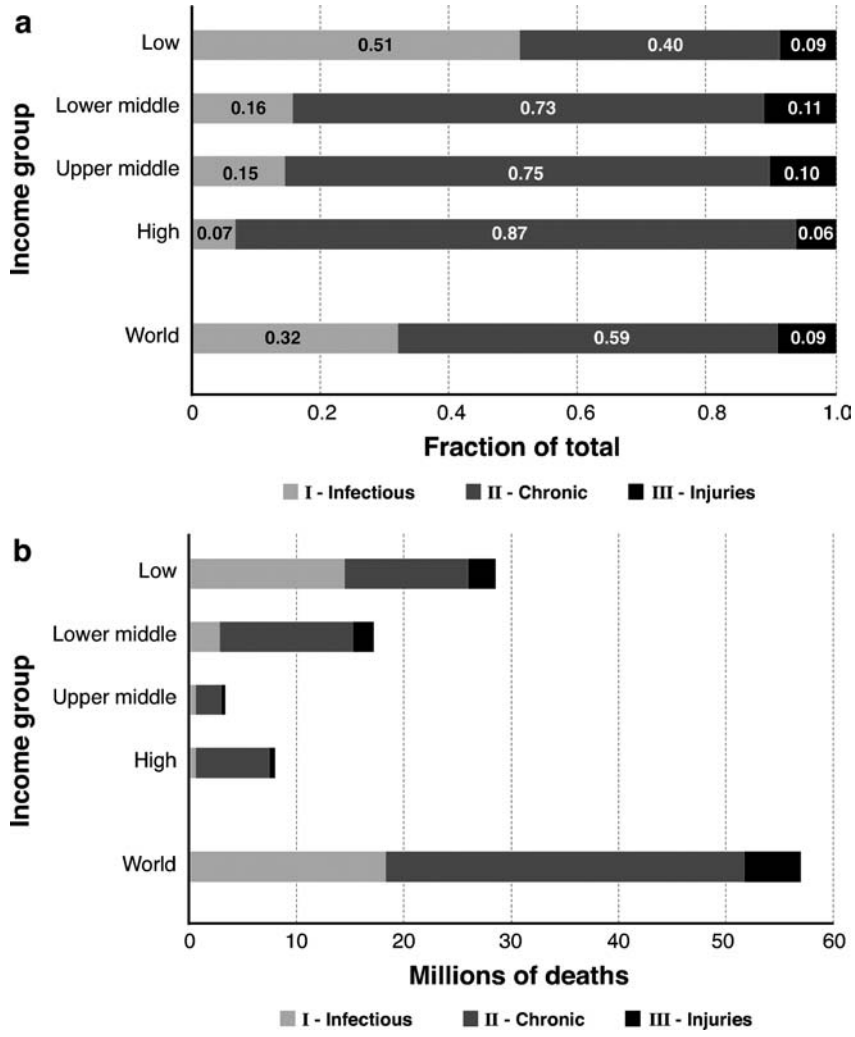

Figure 5 Original form of the epidemiologic transition or the mortality transition because it addresses only dealths in each category (I, infectious; II, chronic; III, injuries) and in each income group of countries as (*a*) proportion of total deaths, (*b*) the absolute number of deaths, (*c*) deaths per 1000 capita, and (*d*) deaths per capita adjusted to world age distribution. The income categories are low income >$735 per capita, lower middle income = $735–$2935, upper middle income = $2935–$9076, and high income >$9076, using 2002 dollars per capita purchasing power ($PPP) (52). Health data from the WHO GBD database for 2002 (52a).

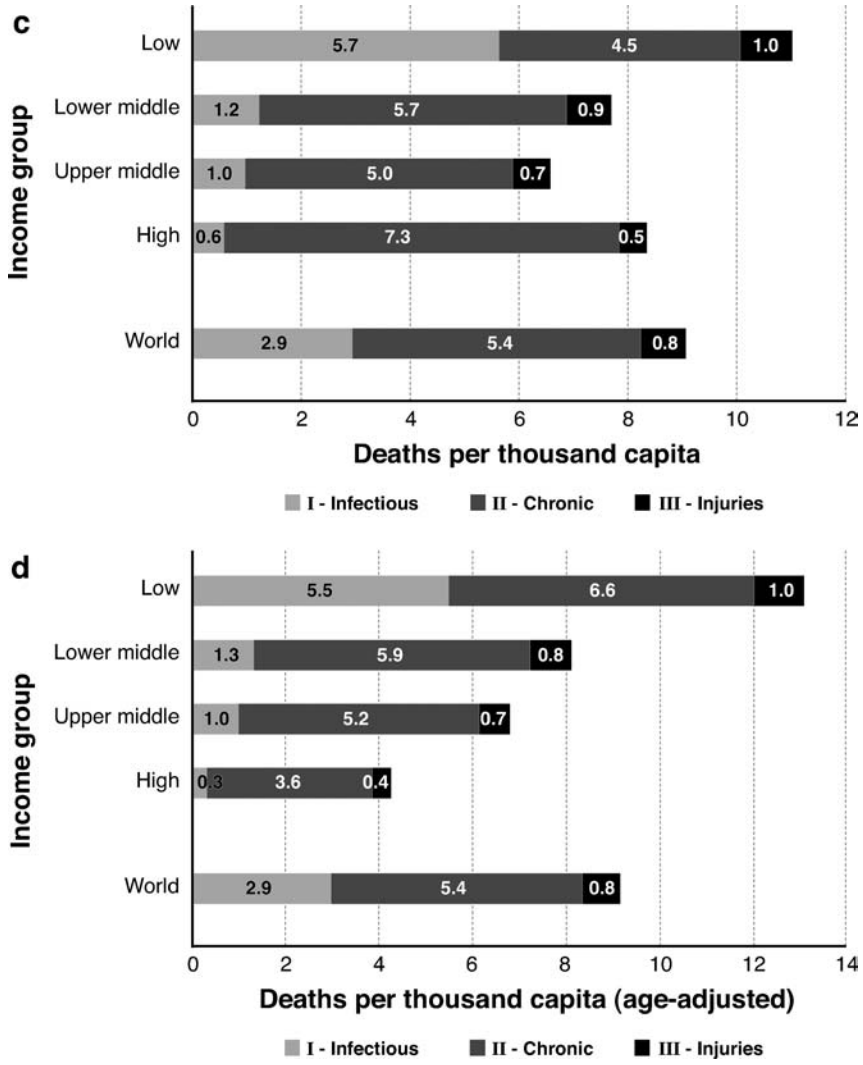

Figure 5 (*Continued*)

weight according to the severity of each outcome, it is possible for the first time to describe what might be called the true epidemiologic transition, using morbidity as well as mortality data combined in the DALY metric used in the GBD databases.

- Figure 6*a* shows the epidemiologic transition using proportion of ill health in each disease category in 2002 as measured in DALYs. Compared to the mortality transition in Figure 5*a*, it shows an even stronger shift from

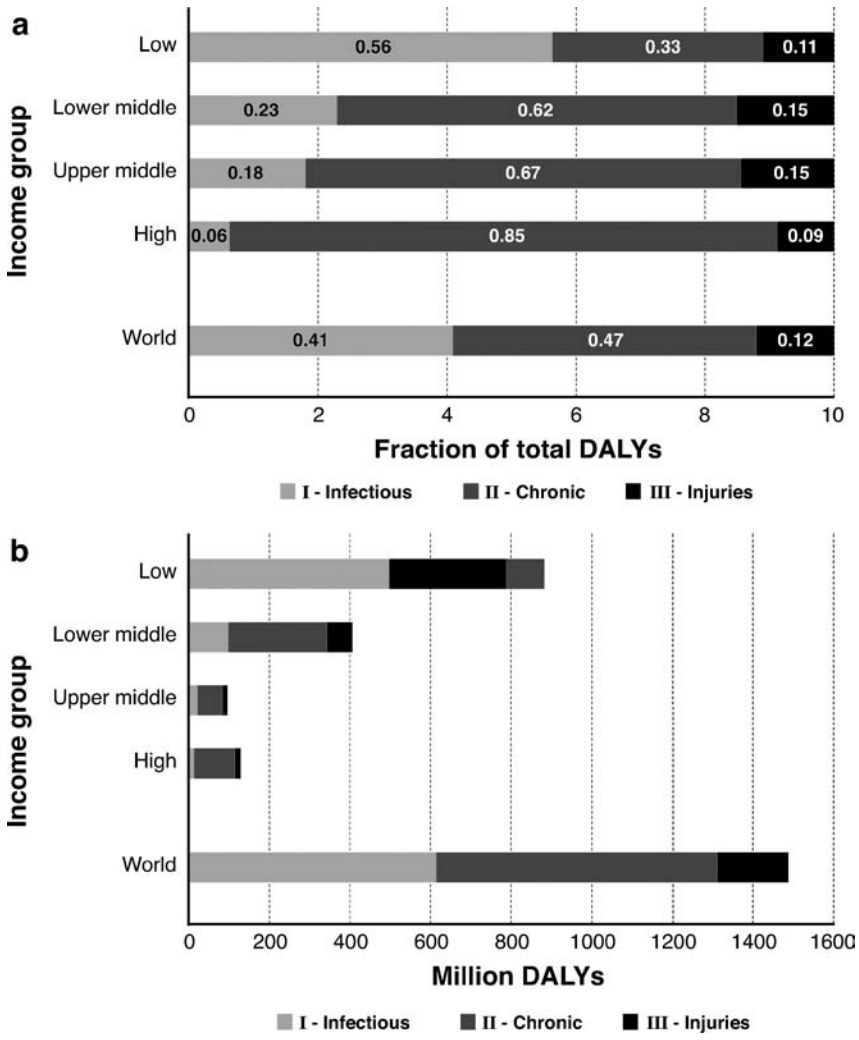

Figure 6 (*a*) True epidemiologic transition using DALYs, a measure of lost time that includes allowance for both premature death and illness and injury as fraction of total lost DALYs by disease category (category I is infectious, category II is chronic, and category III is injuries) and income group. (*b*) Epidemiologic transition in absolute DALYs by income group. (*c*) Epidemiologic transition per capita, shown in DALYs per thousand individuals. (*d*) Epidemiologic transition adjusted to world age distribution, shown in DALYs per 1000 captita. Health data from the WHO GBD database for 2002 (52a).

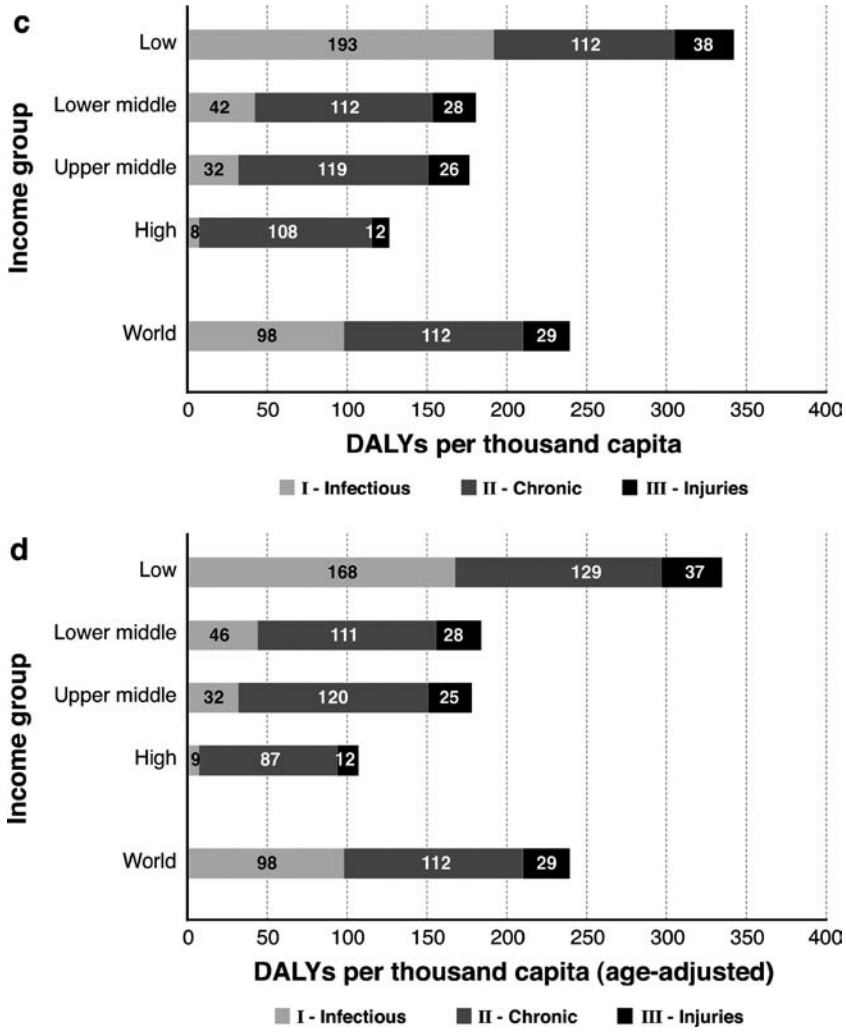

Figure 6 (*Continued*)

infectious to chronic diseases because many of the infectious disease deaths occur in younger ages and hence account for a larger loss of health life years.

- Figure 6*b*, using total DALYs, shows again how important infectious diseases in the poorest countries are in the world picture.
- Figure 6*c*, corrected for population size, shows the same features as Figure 5*c*, its parallel in mortality, and much different from Figure 7*a*, the

classic mortality transition presented in the literature or its epidemiologic equivalent, Figure 6*a*.

- Figure 6*d*, corrected for age distribution by adjusting all regions to the world distribution of population in 2002, shows a pattern similar to Figure 6*c* partly because any analysis using the DALY metric incorporates some age adjustment owing to its use of lost life years for mortality. This figure, however, is probably the best single representation of the true risks of disease, injury, and illness across income groups from the standpoint of individuals. The figure illustrates that even diseases traditionally associated with westernization or modernization have larger age-specific risks in developing countries. In other words, those people, who survive childhood infectious diseases in the developing world, are still at a disadvantage for chronic diseases compared to people in rich countries.

In summary, Figure 6*a*,*b*,*c*,*d* shows

1. The huge burden of infectious diseases that persists in the poorest countries.
2. Most of the improvement in overall health status during development occurs through reduction of the category I diseases (e.g., about three quarters of the total lowering of burden per capita comparing low- to high-income countries).
3. The so-called nontransitional injuries actually do seem to decrease with income (by a factor of three between low- and high-income countries).
4. Most different from many previous interpretations of the impacts of development on health, it shows that chronic diseases (category II) also generally decline with development (down by one third from low- to high-income countries), although they remain approximately constant across the two middle-income categories, in contrast to their pattern of mortality in Figure 5*d* (53).
5. The apparent increase in chronic disease (category II) during development, found in classic descriptions of the mortality transition, is actually because they become relatively more important as category I and III diseases decline and the age distribution becomes older, not by absolute increases in risk to individuals.

QUANTIFYING THE ENVIRONMENTAL RISK TRANSITION

With the coherent and consistent databases provided by the WHO GBD and the CRA projects, it is now also possible to put flesh on the conceptual skeleton of the risk transition hypothesis by examining the trends of environmental health

impacts with development. For many risk factor-disease combinations, there are relatively short delays between imposition of the exposures to environmental hazards and occurrence of the disease. Exposure to fecal-contaminated water, malarial mosquitoes, and poor traffic safety, for example, precede disease and injury by relatively short periods. Thus, the risk and the burden are closely linked. For other risk factor-disease combinations, e.g., lung cancer from air pollution and malnutrition from climate change, the latency periods would be much longer. Further, additional health determinants, such as nutrition and access to health care, affect the same health outcomes as those influenced by environmental risks. For example, in China, the decline in child mortality from lower respiratory infections, diseases affected by IAP from solid fuel use, has likely been a result of changes in energy use (types of fuels and stoves) and improved access to medical interventions. As a result, the decline in disease outcome has occurred more rapidly than risk factor exposure. The current analysis using the disease burden associated with environmental health risks does not address the issue of lags and possible divergence between exposure change and health impacts, which will require additional data.

Before discussing the trends of environmental health risk with development, however, we need to choose both the metric of development to use and the metric of risk.

Measuring Development

Much has been written about the advantages and disadvantages of different indicators or combinations of indicators (indices) of development (54, 55). Two well-established indices, updated annually in global databases, are

- Income per capita (in U.S. dollars, open ended), but adjusted for local purchasing power in 2000 (56). Here, dollars of gross domestic product/capita, adjusted for purchasing power, is termed $PPP/capita.
- Human Development Index (HDI) (dimensionless, from 1 to 100), developed originally by the UN Development Programme (57). HDI combines purchasing power income per capita, life expectancy, and education level in a nonlinear fashion using 2000 data.

Because the same measure of income is included in both, the two are related ($r^2 = 0.76$ in 2000) and thus might be thought to be duplicative. Unlike the simple income metric, however, the HDI weights income on a log scale, and thus an extra $100 per capita produces a bigger change in HDI in a poor society than in a rich one. The HDI also includes factors (health and education) that, although often correlated with income, do not always move together because nations at the same incomes can put more or less resources into development of human capital. Therefore, the two indices result in a different relative ranking of individual nations or regions. The values used for $PPP/capita and HDI for each of the 14 WHO subregions used in this review are shown in Table 3.

TABLE 3 Global Burden of Disease 2000 subregions and the development indicators used in this analysis[a]

WHO region	Mortality stratum[a]	Countries	2000 population (thousands)	$GDP/capita (2000) purchasing power parity[b]	2000 HDI[c]	Natural debt[d]
African region (AFR)	D	Algeria, Angola, Benin, Burkina Faso, Cameroon, Cape Verde, Chad, Comoros, Equatorial Guinea, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Madagascar, Mali, Mauritania, Mauritius, Niger, Nigeria, Sao Tome and Principe, Senegal, Seychelles, Sierra Leone, Togo	294,078	1,670	0.47	3.7
	E	Botswana, Burundi, Central African Republic, Congo, Côte d'Ivoire, Democratic Republic of the Congo, Eritrea, Ethiopia, Kenya, Lesotho, Malawi, Mozambique, Namibia, Rwanda, South Africa, Swaziland, Uganda, United Republic of Tanzania, Zambia, Zimbabwe	345,515	2,050	0.45	7.6
Region of the Americas (AMR)	A	Canada, Cuba, United States of America	325,183	32,500	0.94	119
	B	Antigua and Barbuda, Argentina, Bahamas, Barbados, Belize, Brazil, Chile, Colombia, Costa Rica, Dominica, Dominican Republic, El Salvador, Grenada, Guyana,	430,932	7,790	0.78	11

		Honduras, Jamaica, Mexico, Panama, Paraguay, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Suriname, Trinidad and Tobago, Uruguay, Venezuela				
	D	Bolivia, Ecuador, Guatemala, Haiti, Nicaragua, Peru	71,230	2,820	0.64	5.6
Eastern Mediterranean region (EMR)	B	Bahrain, Cyprus, Islamic Republic of Iran, Jordan, Kuwait, Lebanon, Libyan Arab Jamahiriya, Oman, Qatar, Saudi Arabia, Syrian Arab Republic, Tunisia, United Arab Emirates	139,059	7,040	0.73	18.2
	D	Afghanistan, Djibouti, Egypt, Iraq, Morocco, Pakistan, Somalia, Sudan, Yemen	342,576	2,420	0.54	3.5
European region (EUR)	A	Andorra, Austria, Belgium, Croatia, Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland, United Kingdom	411,889	23,300	0.92	57.8
	B	Albania, Armenia, Azerbaijan, Bosnia and Herzegovina, Bulgaria, Georgia, Kyrgyzstan, Poland, Romania, Slovakia, Tajikistan, the former Yugoslav Republic of Macedonia, Turkey, Turkmenistan, Uzbekistan, Yugoslavia	218,458	6,060	0.76	44

(*Continued*)

TABLE 3 (*Continued*)

WHO region	Mortality stratum[a]	Countries	2000 population (thousands)	$GDP/capita (2000) purchasing power parity[b]	2000 HDI[c]	Natural debt[d]
	C	Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, Russian Federation, Ukraine	243,184	7,280	0.77	73
Southeast Asia region (SEAR)	B	Indonesia, Sri Lanka, Thailand	293,819	3,790	0.70	3
	D	Bangladesh, Bhutan, Democratic People's Republic of Korea, India, Maldives, Myanmar, Nepal	1,241,806	2,200	0.56	2.8
Western Pacific region (WPR)	A	Australia, Brunei Darussalam, Japan, New Zealand, Singapore	154,354	26,300	0.93	45
	B	Cambodia, China, Cook Islands, Fiji, Kiribati, Lao People's Democratic Republic, Malaysia, Marshall Islands, Federated States of Micronesia, Mongolia, Nauru, Niue, Palau, Papua New Guinea, Philippines, Republic of Korea, Samoa, Solomon Islands, Tonga, Tuvalu, Vanuatu, Viet Nam	1,532,933	4,320	0.72	7.3
World totals			6,045,016	7,340	0.69	20

[a]Abbreviation and symbols: A, very low child mortality and very low adult mortality; B, low child mortality and low adult mortality; C, low child mortality and high adult mortality; D, high child mortality and high adult mortality; E, high child mortality and very high adult mortality; HDI, Human Development Index. High-mortality developing regions include AFR-D, AFR-E, AMR-D, EMR-D, and SEAR-D; lower-mortality developing regions inclue AMR-B, EMR-B, SEAR-B, and WPR-B; developed regions include AMR-A, EUR-A, EUR-B, EUR-C, and WPR-A (18, 82).

[b]GDP per capita adjusted for purchasing power parity (56).

[c]The values shown here are the simple population-weighted means of the country HDI values in Reference 57. As the HDI does not scale linearly with its indicators (purchasing power income per capita, life expectancy, and education level), it would be more accurate to take the population-weighted means of the indicators for all the countries and then recreate a new HDI for the subregions and globe. When this is done, for example, the global value is 0.72, somewhat above the 0.69 shown here (57).

[d]Measured as tons of carbon per capita depleted carbon as carbon dioxide remaining in the atmosphere from national emissions 1950–1991 (67).

Risk Metric

From the standpoint of national policy, the relative importance of risk factors as fractions of the total impact is critical in deciding the distribution of available (and always scarce) resources for interventions. For international comparisons of risks, however, a per capita measure of the impact of a risk factor is more relevant to understanding how it affects different populations. Thus, we focus on two risk metrics: (*a*) percent of total burden, measured in DALYs, and (*b*) burden per capita, measured as DALYs per 1000 capita.

Household Environmental Risks

Of the environmental risk factors and diseases in Table 1, three stand out as principally operating at the household level and mainly affecting young children (diarrhea, acute lower respiratory infections, and malaria).

- Poor water, sanitation, and hygiene.[4] These three factors are grouped together because studies have shown that improving one alone does not necessarily produce a reliable reduction in ill health, and there are complex interrelationships among quantity and quality of water, sanitation, waste disposal technology and hygiene behavior (58, 59). The only disease included in the CRA analysis for this risk factor is diarrhea, which is caused by pathogens in human waste finding their way back into households through contaminated water and food or poor hygiene practices. Although both community (sewage) and household (latrine) technologies and behaviors play roles in reducing impacts, the hazards are created by household activities, and household-level interventions hold the key to the major improvements.
- IAP from solid fuel use. About half the households in the world use solid fuels (biomass or coal) for cooking and heating in simple stoves that emit health-damaging air pollutants into living areas. The CRA project identified good evidence that such pollution contributes significantly to three major diseases worldwide: acute lower respiratory infections, chronic obstructive pulmonary disease, and lung cancer (47). Solutions (cleaner fuels, better stoves, ventilation, and hygiene) lie principally at the household level, although obviously community efforts, such as fuel and stove subsidies and energy infrastructure, play roles.
- Although community efforts, such as habitat management, are important for reducing malaria (60), household-level interventions (screening, pesticides, and bed nets) offer the significant opportunities for control today and help explain the reduction of malaria in areas where it has been eliminated in the

[4]Here, hygiene refers to individual/family behavior, such as water storage, waste disposal, and hand washing, which effect fecal-oral transmission.

past. The level of malaria in Southeast Asia (SEAR-B) is taken as the level other regions could reach. Note that more than 90% of the global burden falls on sub-Saharan Africa (AFR-D,E) (61, 62).

Figures 7*a*,*b* show that in both relative and absolute terms these household-level environmental risks decline markedly with development, measured by $PPP/capita. There are more than two orders of magnitude fall in burden per capita between poor and rich countries. Although changing the regional relationships to some degree, Figures 7*c*,*d* show that use of HDI as the development metric does not illuminate a largely different pattern, and thus to save space, we focus on $PPP per capita as the development indicator for the rest of this analysis.

Note the anomalous dip in the trends shown in Figures 7*a*,*b* for the region at about $7300 PPP/capita (a pattern that is repeated in most of the other environmental risk transition figures). This is for WHO region EUR-C (Table 3), which includes most of the former Soviet Union plus a few eastern European countries. It seems that its household environmental health risks are lower than what might be indicated by its current income. This partly reflects the sustained low levels of child mortality, a major outcome of household environmental risks, in the countries in this region despite the stagnant or declining income in the early 1990s. Note also the anomalous rise in the trends around $2400 PPP/capita, which is in the eastern Mediterranean Region D (EMR-D). Apparently whether measured by HDI or $PPP/capita, the poorer countries of North Africa and the eastern Mediterranean plus Pakistan have greater household environmental risks than might be expected by their development status.

Community Environmental Risks

Of the quantified environmental risks and diseases, several stand out as community-based exposures.

- Urban outdoor air pollution: Although some portion is due to household activities, the principal sources and solutions mainly lie at the community level. This risk assessment only examined cities above 100,000 in population and thus is an underestimate of the global burden from all urban outdoor air pollution (48). (Rural areas can also have significant outdoor air pollution levels.)
- Lead pollution: Although there are household sources (e.g., paint), the chief sources of lead pollution worldwide have been leaded gasoline and industrial sources (63).
- Occupational risks: Separately quantified were risks of airborne particles, carcinogens, injuries, noise, and poor ergonomics, which are combined here (64).
- Road traffic accidents: These include the burden on pedestrians as well as vehicle occupants.

Figures 8*a*,*b* show that the pattern of these risks with development is somewhat more complex than those of household risks, with some rising, some falling, and some first rising and then falling. In general, the health impacts of road traffic accidents and urban air pollution rise with development, then fall, although the pattern is less dramatic with air pollution. In contrast, lead and occupational burdens vary quite a bit, presumably owing to differential timing of control measures for lead and different mixtures of industries and occupational safety regulations for occupational hazards.

Note again the anomalously good performance for EUR-C and the mixed performance for EMR-D, compared to their income levels. Note also that, compared to the others, health impacts of urban ambient air pollution falls earlier in the development process, illustrating the increasing concentration of the health hazards of this exposure in poor societies.

Global Environmental Risks

The only global environmental risk factor examined in the WHO CRA was climate change due to human release of greenhouse pollutants into the global atmosphere (65). Other global impacts, such as stratospheric ozone depletion and land-use changes, were not addressed. As shown in Table 1, climate change today has had a relatively small impact on human health worldwide. Unlike the other environmental risk factors examined, however, exposure will likely increase in future years, with associated health impacts, depending on public efforts to control diseases, such as malaria, that are magnified by climate change (50). Figures 9*a*,*b* show that the risks from climate change dramatically decline with development, reflecting the greater vulnerability of poor populations to the disease categories affected: diarrhea, malaria, natural disasters, and malnutrition.

This trend may seem to be counter to the environmental risk transition framework shown in Figure 2, wherein global risks are shown to increase with development. The discrepancy is explained by the different perspectives involved, however. Figure 9 indicates where the risk is experienced but not where it originates. Because the source of climate change risk [greenhouse-gas (GHG) emissions] increases with development, however, the size of the risk imposed does rise with development, as shown below.

ENVIRONMENTAL RISK TRANSITION REVISITED

Figures 10*a*,*b* combine the separate risks in the three spatial groups—household, community, and global. Instead of using the risk experienced for climate change (Figure 9), the total damage (Table 1) is redistributed according to the "natural debt" of each of the subregions, which is a measure of how much global warming has been contributed by different countries (66). Measured in tons of carbon remaining in the atmosphere per capita, it is determined from total fossil fuel carbon emissions since

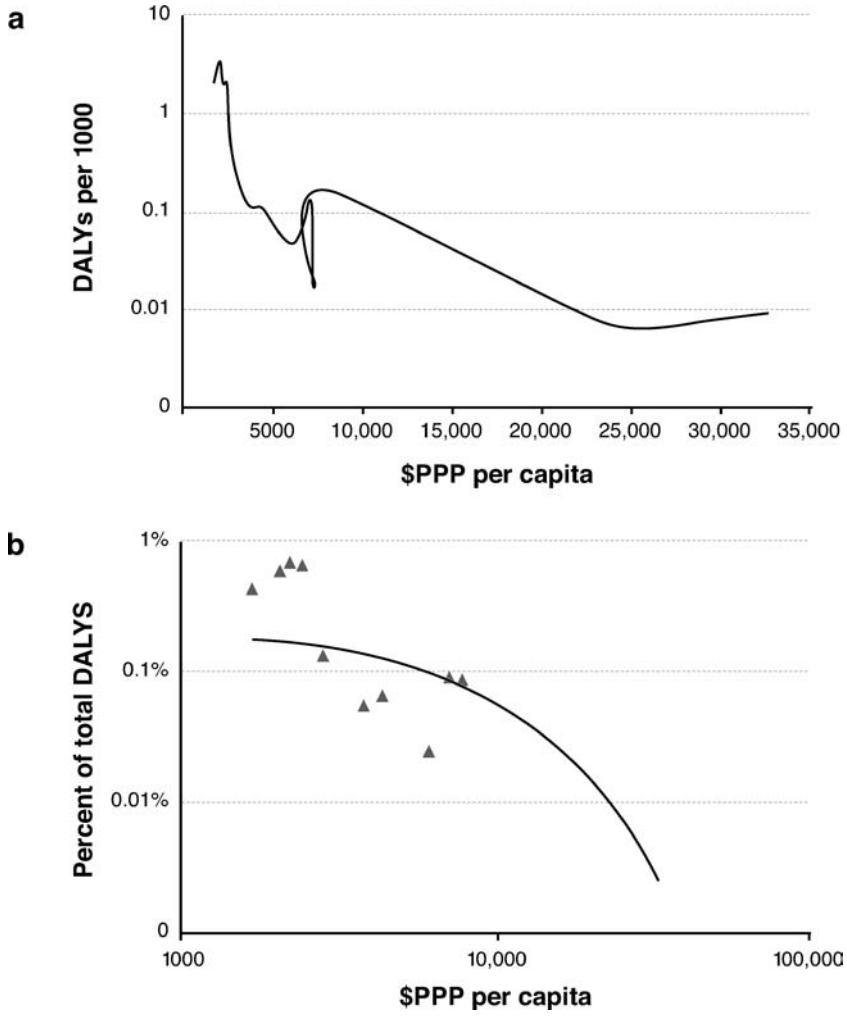

Figure 9 Trend of experienced global environmental risk transition in panel (*a*) as DALYs per 1000 capita and in panel (*b*) as percent of total DALYs with development measured as per capita purchasing power ($PPP). Panel (*b*) also shows a trend line. Sources: WHO Comparative Risk Project (39) and UN Development Programme Human Development Report (57).

1950, depleted by natural absorption processes (67). Use of other GHG emission measures, such as cumulative emissions extending to earlier periods, undepleted cumulative emissions, or even current emissions of total GHGs, are unlikely to significantly change the pattern. The estimated natural debt of each region in tons carbon per capita is shown in Table 3.

The patterns of Figure 10 do roughly support the conceptual framework shown in Figure 2, i.e., traditional or household environmental risks do tend to decline with development, no matter which metric is used. Modern or community risks tend first to rise and then to fall as development proceeds, although the pattern is less consistent and local exceptions exist. This complexity of community risks likely reflects the diverse role of technological and policy options that have been differentially adopted for the control of these risks by societies at similar levels of development. Imposition of global or postmodern risks tends to rise with development, with no diminution at the highest levels yet in evidence.

Risk Overlap

Within the original environmental risk transition framework was the concept of "risk overlap," describing communities experiencing significant amounts of both traditional and modern (household and community) risks at the same time (68, 69). Most obvious among these groups are populations living in urban slums of poor countries, which tend to retain poor household environments while living in the most contaminated parts of cities in terms of air pollution, traffic, and solid waste. A number of interactions were associated with such risk overlap situations including

- Risk genesis: New types of risk are created, such as toxic wastes in garbage dumps.
- Risk transfer: Attempts to control one type can make other types worse, such as pesticides used for malaria control.
- Risk synergism: Risk of one type changes sensitivity to other risks in absolute or relative terms, such as increased diarrhea or pneumonia mortality among undernourished children who also have limited access to transportation to health services. This synergy and vulnerability further magnify and concentrate the burden of disease and ill health caused by environmental exposures.

The distinction between imposing and experiencing global environmental health risks exhibits another even more prevalent risk overlap situation. Measured as the ratio of risk imposed to the risk experienced, it ranges by more than a factor 7000 from the ratio of 0.08 in poor Africa to 560 in North America. Poor populations already burdened by high household and community risks are thus also bearing the major portion of the health risks imposed by global climate change in what might be termed a triple risk overlap.

The Environmental Kuznets Curve

A large literature and debate has revolved around the question of whether environmental impacts tend to first rise and then fall with economic development (inverted U shape), as described for income inequity by Nobel economist Simon Kuznets in

the 1950s (70). Not only the shape but its implication was disputed, i.e., whether countries could grow their way out of environmental problems (71–75). The environmental risk transition framework provides one explanation of how different observers have sometimes come to quite different conclusions about this hypothesis when examining the evidence, at least in terms of human health impacts.

Such examinations of the environmental Kuznets curve hypothesis in their simplest forms have generally not

- Made careful distinction between risks to environmental health and those to environmental quality and/or ecosystems, partly because of limited access to comprehensive and coherent databases such as those of the GBD and CRA projects;
- Carefully defined and used consistent metrics for risk and development in their analyses;
- Conducted careful examination of environmental risks across the spatial spectrum, for example, often leaving out household and occupational risks.

Figure 10, nevertheless, would seem to support a modified environmental Kuznets curve hypothesis in the following three-part form:

1. Community-level environmental health impacts tend to be highest in mid-income countries and to be less in the poorest and richest countries in both absolute and relative terms, although there are exceptions depending on local circumstances and the particular risk factor.
2. Household-level environmental health risks, which represent a much larger burden, tend to show declines at every level of increased development.
3. Although being currently relatively small, the impact of global risks falls with development, but the imposition of these risks steadily rises with development.

The shift in spatial scales seen in the environmental risk transition framework is accompanied, imperfectly, by a shift to longer temporal scales, i.e., the lag between risk and disease is, on average, shorter for household than for community risks and shorter in community hazards than global ones. There is evidence that rising income affects societal discounting in time and space (low income being accompanied by high effective discount rates), which might help explain why the tendency to "pass the buck" moderates in its temporal and spatial scales in the way shown by the environmental risk transition framework (76).

LIMITATIONS OF EPIDEMIOLOGIC RISK ASSESSMENT

Environmental health risks include important categories that are not well addressed by the type of risk assessment conducted here (77).

Environment as a Super Distal Risk Factor

Defining exactly what constitutes an "environmental" risk is not straightforward because environment, by definition, affects health in a number of positive and negative ways. Early estimates of the health burden due to environmental factors used the term in its most general form, i.e., anything that was not genetic was environmental (nature and nurture) (77a). A more recent estimate (32) attempted to define environment more exactly and to use the first burden of disease database (17) to take advantage of its complete and coherent presentation of global disease burdens. Done prior to the more formal and detailed CRA described above, which started from the major intervention categories (e.g., air pollution, lead), the overall burden of environmental health risks was determined instead by estimating the environmental portion disease by disease.

Environment, however defined, is one of a handful of what one might call "super distal" risk factors (or more appropriately health determinants). Other such factors include nutrition, poverty, and social inequality, which all have broad positive and negative effects on many diseases. A super distal risk factor affects essentially every disease, even if the pathways are not always well understood. For example, although there may be no environmental factor clearly increasing the chance of becoming infected with HIV, someone with acquired immuno-deficiency syndrome (AIDS) who goes home to a dirty slum environment will have a poorer outcome (more severe disease and earlier death) than someone in a clean environment. It is just so with gunshot wounds, diabetes, schizophrenia, and essentially every other kind of nonenvironmental disease as well. In a burden of disease framework, disease outcomes count as well as contracting the disease. With this argument, the environmental burden of disease assessment mentioned above (32), assigned at least 5% of every disease to environment, with larger proportions to diseases, such as diarrhea, with attributable proportions to more direct environmental factors. In total, it was estimated that environmental risks might be responsible for 25% to 40% of the GBDs, with larger proportions in poor countries and lower proportions in rich countries (32). Undoubtedly, however, a uniform 5% is not realistic and some nonenvironmental diseases are more influenced by indirect pathways from environmental factors than others. To determine these relationships in a systematic quantitative fashion would require resolving a full causal web linking various environmental and other risk factors to their related diseases and well-defined counterfactual scenarios of exposure and disease.

High-Consequence, Low-Probability Hazards

The CRA project did not attempt to determine the expected health damage from high-consequence, low-probability events. These include natural disasters (e.g., earthquakes, volcanic eruptions, tsunamis, and asteroid impacts), high-impact technological failures (e.g., intentional and accidental nuclear explosions), and new and old (emerging and reemerging) infectious diseases caused by evolving

human activities, such as those associated with trade, tourism, terrorism, blood supplies, and human interaction with natural environments (78). Although not susceptible to the type of epidemiologic analysis done in the CRA project, as shown by the 2004 Tsunami in the Indian Ocean and the 2003 SARS (Severe Acute Respiratory Syndrome) outbreak, such events can exact a considerable toll of ill health and also generate large emotional, technical, economic, and political responses both locally and globally.

The kind of environmental risk assessment described here, although subject to uncertainty because of our imperfect knowledge of human exposure levels and exposure-response relationships, deals with common population-level exposures and not those for which occurrence at any given time or place is subject to great uncertainty. We may not know exactly how many people are exposed to how much human waste from lack of sanitation at what precise individual risk, but we are sure that such exposure is a daily occurrence for millions and that important effects exist daily. Whether and when another tsunami or SARS epidemic will occur is extremely hard to estimate and thus how much health burden might occur is even more so. Indeed, entirely different methods would be needed in the realm of the kind of probabilistic risk assessments (PRAs) done for nuclear power plants and space missions (78a). Even when done, the results of PRA and CRA are hard to compare fairly, as they are based on different assumptions and subject to different types of uncertainty.

Human perception being what it is (79, 80), of course, SARS or a major disaster like a tsunami evokes larger responses in the press and in policy circles than ancient day-in-day-out risks like diarrhea from poor sanitation. From a public health perspective, there may seem to be apparent tension between responding to unknown, uncertain risks and large risks of day-to-day life, especially those of the poor. Using deaths as the indicator, SARS killed in six months about 800 people, roughly the number of children killed in a few hours in India alone by acute lower respiratory infections, which because they can kill as, or even more, rapidly have been dubbed "old SARS" (81). Every month, about as many children die from the three big environmental diseases alone—acute lower respiratory infections, diarrhea, and malaria—in the regions most affected by the 2004 Tsunami as the total mortality from the tsunami (82). How does one compare, for illustration, the risk of poor sanitation in a region estimated at 600–1400 thousands deaths per year with a 20% chance of an earthquake killing 600–1400 thousand in the next 50 years, which would be one of history's greatest natural disasters? Is a straightforward expected value calculation appropriate in which the earthquake risk is treated as equivalent to 4 thousand deaths a year? The amount of resources that could be justified for earthquake interventions would pale to nothing, but this is clearly not the way public or media perceptions always operate.

The magnitude of the direct disease burden, of course, is not the only characteristic that makes an environmental risk worthy of concern. The economic damage that went along with the 2004 Tsunami and consequent difficulties of reestablishing livelihoods is an important factor, possibly with long-term health

consequences. For the SARS epidemic, the uncertainty about where it would stop or stabilize justified a large response. In a similar although not quite as dramatic fashion, the HIV epidemic is of special concern because it is still rising and no stable point is in sight. Among the environmental health risks examined here, it is those from climate change, small as they are now, that ought to worry us from this perspective because we do not know where they are going and our assessment techniques are poor. Although the importance of risk perception and risk management is outside the scope of this review, it has been argued that strong public health and environmental health institutions that reduce vulnerability and exposure to day-to-day risks are arguably also the best defense against unknown risks (82, 83).

CONCLUSION

It is important to emphasize that cross-sectional comparisons are not always good predictors of what will happen in any one place on a longitudinal basis. Given the great efforts it took to create the first truly comprehensive and coherent databases of death, disease, and injury by age and sex in 14 regions covering the world and the attributed burden for major risk factors, environmental and otherwise, it will be a while before we obtain true time-series data on a such a comprehensive and coherent basis. In the meantime, we need to glean what insight we can from these cross-sectional comparisons. In addition, what is reported in this review from the CRA project was the outcome of the exposure to environmental hazards, not the exposure itself. Although the two are closely linked for some risk-disease relationships, there are other important factors that affect how exposures translate into illness and mortality, most importantly nutrition and access to medical care. Thus, strictly speaking, what we have explored is not the transition of the risks, but their expression.

Finally, the kind of quantification in the CRA project is based on techniques using epidemiologic measures of exposure-response and nonstochastic measures of exposure. It cannot be applied to the high-consequence low-probability environmental risks, such as natural disasters, or new/emerging disease risks with which humanity has had no previous experience, such as SARS.

Despite these limitations of the basic risk framework, the investigation of the epidemiologic transition using the GBD database (Figures 5 and 6) reveals several important characteristics of disease patterns across development categories.

- No matter what metric is used, category I (infectious disease) in poor countries still dominates the world burden of ill health. Much of this burden falls on young children.
- There is no apparent substitution of category II (chronic) disease for category I during development, as is often stated. Using risk per capita burden, corrected for age and including both mortality and morbidity,

Figure 6*d* shows that both categories decline as income increases, with the possible exception of nearly static levels in the middle-income regions for category II.

- Category III risks (injuries) decline as well and are not nontransitional as sometime claimed.

Although a full test of the environmental risk transition hypothesis using pure measures of environmental stressors, freed from interactions with other factors such as nutrition and access to medical care, is still beyond existing databases, the database from the WHO CRA project allows a cross-sectional analysis of the environmental health transition, i.e., how the health impacts of environmental risk factors vary across development categories. Some major points emerge from this analysis (Figures 7–10):

- A good deal of the burden of infectious disease borne in poor countries is due to household environmental risks, as much as one third of the burden of all category I disease or one fifth of all disease in poor countries.
- Household environmental risks, however, decline markedly and nearly uniformly with development.
- Although at 4.3% of the GBD, community risks are about half those from household risks globally (9%), they are spread differently across the development spectrum with the highest rates in middle-income countries, measured as either percent of total burden or burden per capita, fitting the environmental Kuznets relationship.
- Impacts from lead and occupational risks show less consistent trends than those from road traffic accidents and urban air pollution, possibly because of the clear and strong role of technological and regulatory interventions.
- Although at 0.4% of the GBD, global risk from climate change is currently much smaller; its expression trends much like household environmental risks, i.e., sharply declining with development.
- The imposition of global risks, determined by natural debt, however, trends steeply upward, with rich countries imposing much more risk because of historically longer and higher emissions of greenhouse gases.
- The trends of environmental risk at different spatial scales (household, community, global) across development levels do generally support the environmental risk transition hypothesis.
- For their income levels and for some risks, the EUR-C region seems to have lower levels than might be expected, and EMR-D has somewhat greater levels.

Imposed and experienced climate change risks are unequally distributed in the world. As a result, the ratio of the two varies by nearly four orders of magnitude (7000×) across development regions. Although by any standard, the total health

impact from climate change is not large at present, the potential for growth is high under current emission trends. The differences in risk perception among regions, owing to widely different local ratios of imposed to experienced risks, however, are likely to continue to complicate efforts to reach international consensus on strong control measures.

ACKNOWLEDGMENT

We appreciate the assistance given by Jamesine Rogers in collecting literature for this review.

LITERATURE CITED

1. Demeny P. 1968. Early fertility decline in Austria-Hungary: a lesson in demographic transition. *Daedalus* 97:502–22
2. Demeny P. 1986. Population and the invisible hand. *Demography* 23:473–87
3. Vallin J. 2002. The end of the demographic transition: Relief or concern? *Popul. Dev. Rev.* 28:105–20
4. Omran AR. 1971. The epidemiologic transition: a theory of the epidemiology of population change. *Milbank Mem. Fund Q.* 49:509–38
5. Salomon JA, Murray CJ. 2002. The epidemiologic transition revisited: compositional models for causes of death by age and sex. *Popul. Dev. Rev.* 28:205–28
6. Smith KR. 1989. The risk transition and developing countries. In *Understanding Global Environmental Change: The Contributions of Risk Analysis and Management*, ed. JX Kasperson, RE Kasperson, pp. 148–72. Worcester, MA: Tokyo/New York: Univ. Press
7. Smith KR. 1990. The risk transition. *Int. Environ. Aff.* 2:227–51
8. Olshansky SJ, Ault AB. 1986. The fourth stage of the epidemiologic transition: the age of delayed degenerative diseases. *Milbank Mem. Fund Q.* 64:355–91
9. McKeown T. 1988. *The Origins of Human Disease*. Oxford: Blackwell
10. McGranahan G, Songsore J. 1994. Wealth, health, and the urban household. *Environment* 36(6):4–11
11. Suttmeier RP. 1994. Risk in China: comparative and historical perspectives on its social construction and management. *Technol. Forecast. Soc. Change* 46:103–23
12. Holdren JP, Smith KR. 2000. Energy, the environment, and health. In *World Energy Assessment*, ed. J Goldemberg, JW Baker, S Ba-N'Daw, H Khatib, A Popescu, et al., pp. 61–110. New York: UN Dev. Programme, UN Dep. Econ. Soc. Aff., World Energy Counc.
13. McGranahan G, Jacobi P, Songsore J, Surjadi C, Kjellen M. 2000. *Citizens at Risk: From Urban Sanitation to Sustainable Cities*. London: Earthscan
14. Smith KR, Akbar S. 2003. Health-damaging air pollution: a matter of scale. In *Health and Air Pollution in Rapidly Developing Countries*, ed. G McGranahan, F Murray, pp. 1–20. London: Earthscan
15. Marcotullio PJ, Lee YSF. 2003. Urban environmental transitions and urban transportation systems—a comparison of the North American and Asian experiences. *Int. Dev. Plan. Rev.* 25:325–54
16. World Bank. 1993. *World Development*

Report 1993: Investing in Health. New York: Oxford Univ. Press
17. Murray CJL, Lopez AD, eds. 1996. *The Global Burden of Disease*. Cambridge, MA: Harvard Sch. Public Health/Geneva: WHO/World Bank
18. Mathers CD, Stein C, Ma Fat D, Rao C, Inoue M, et al. 2002. *The Global Burden of Disease 2000: Version 2, Methods and Results (GPE Discuss. Pap. 50)*. Geneva: Glob. Program. Evid. Health Policy/WHO. http://www.who.int/evidence
19. Mathers CD, Salomon JA, Murray CJL, Lopez AD. 2002. Alternative summary measures of average population health. See Ref. 84, pp. 319–34
20. Murray CJL. 1996. Rethinking DALYs. See Ref. 17, pp.1–89
21. Mathers CD, Ezzati M, Lopez AD, Murray CJL, Rodgers A. 2002. Causal decomposition of summary measures of population health. See Ref. 84, pp. 269–86
22. Mosley WH, Chen LC. 1984. An analytical framework for the study of child survival in developing countries. *Child Survival, Popul. Dev. Rev.* 10(Suppl.):25–45
23. Corvalan CF, Kjellstrom T, Smith KR. 1999. Health, environment, and sustainable development: identifying links and interactions to promote action. *Epidemiology* 10:656–60
24. Ezzati M, Utzinger J, Cairncross S, Cohen A, Singer BH. 2005. Environmental risks in the developing world: exposure indicators for evaluating interventions, programs and policies. *J. Epidemiol. Community Health* 59:15–22
25. Murray CJL, Lopez AD. 1999. On the comparable quantification of health risks: lessons from the global burden of disease. *Epidemiology* 10:594–605
26. Murray CJL, Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S. 2003 . Comparative quantification of health risks: conceptual framework and methodological issues. *Popul. Health Metrics* 1(1):1
27. Preston SH. 1976. *Mortality Patterns in National Populations: With Special Reference to Recorded Causes of Death*. New York: Academic
28. Murray CJ, Lopez AD. 1997. Mortality by cause for eight regions of the world: Global Burden of Disease Study. *Lancet* 349:1269–76
29. Peto R, Lopez AD, Boreham J, Thun M, Heath C Jr. 1992. Mortality from tobacco in developed countries. *Lancet* 339:1268–78
30. McGinnis JM, Foege WH. 1993. Actual causes of death in the United States. *JAMA* 270:2207–12
31. Single E, Robson L, Rehm J, Xie X. 1999. Morbidity and mortality attributable to alcohol, tobacco, and illicit drug use in Canada. *Am. J. Public Health* 89:385–90
32. Smith KR, Corvalan CF, Kjellstrom T. 1999. How much global ill health is attributable to environmental factors? *Epidemiology* 10:573–84
33. Smith KR. 2000. The national burden of disease from indoor air pollution in India. *Proc. Natl. Acad. Sci. USA* 97: 13286–93
34. Leigh J, Macaskill P, Kuosma E, Mandryk J. 1999. Global burden of disease and injury due to occupational factors. *Epidemiology* 10:626–31
35. Kunzli N, Kaiser R, Medina S, Studnicka M, Chanel O, et al. 2000. Public health impact of outdoor and traffic-related air pollution: a European assessment. *Lancet* 356:795–801
36. Willett WC. 2002. Balancing life-style and genomics research for disease prevention. *Science* 296:695–98
37. Morgenstern RD, Shih J, Sessions SL. 2000. Comparative risk assessment: an international comparison of methodologies and results. *J. Hazard. Mater.* 78:19–39
38. Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ, Group CRA. 2002. Selected major risk factors and global

and regional burden of disease. *Lancet* 360:1347–60

39. Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. 2004. *Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors*. Geneva: WHO
40. World Health Organ. 1992. *International Statistical Classification of Disease and Related Health Problems*. Geneva: WHO. 10th ed.
41. Maldonado G, Greenland S. 2002. Estimating causal effects. *Int. J. Epidemiol.* 31:422–29
42. Rothman KJ. 1976. Causes. *Am. J. Epidemiol.* 104:587–92
43. Miettinen OS. 1974. Proportion of disease caused or prevented by a given exposure, trait, or intervention. *Am. J. Epidemiol.* 99:325–32
44. Eide GE, Heuch I. 2001. Attributable fractions: fundamental concepts and their visualization. *Stat. Methods Med. Res.* 10:159–93
45. Walter SD. 1980. Prevention of multifactorial disease. *Am. J. Epidemiol.* 112:409–16
46. Ezzati M, Lopez AD. 2004. Smoking and oral tobacco use. See Ref. 39, pp. 883–958
47. Smith KR, Mehta S, Maeusezahl-Feuz M. 2004. Indoor air pollution from household solid fuel use. See Ref. 39, pp. 1435–93
48. Cohen A, Anderson R, Ostro B, Pandey KD, Krzyzanowski M, et al. 2004. Urban ambient air pollution. See Ref. 39, pp. 1353–433
49. Wishnow RM, Steinfeld JL. 1976. The conquest of the major infectious diseases in the United States: a bicentennial retrospect. *Annu. Rev. Microbiol.* 30:427–50
50. Reiter P. 2001. Climate change and mosquito-borne diseases. *Environ. Health Perspect.* 109:141–61
51. Kopits E, Cropper M. 2005. Traffic fatalities and economic growth. *Accid. Anal. Prev.* 37:169–78

51a. Rodgers A, Ezzati M, Vander Hoorn S, Lopez AD, Lin R-B, et al. 2004. Distribution of major health risks: findings from the Global Burden of Disease Study. *PLoS Med.* 1(1):44–55

52. World Bank. 2004. *World Development Report 2004: Making Services Work For Poor People*. New York: Oxford Univ. Press

52a. World Health Organ. 2002. *Global burden of disease database*. http://www3.who.int/whosis/menu.cfm?path=evidence,burden,burden_estimates,burden_estimates_2002N,burden_estimates_2002N_2002Rev,burden_estimates_2002N_2002Rev_Income&language=english

53. Ezzati M, Vander Hoorn S, Lawes CMM, Leach R, James WTP, et al. 2005. Rethinking the "diseases of affluence" paradigm: global patterns of nutritional risks in relation to economic development. *PLoS Med.* 2:e133
54. Anand S, Sen A. 1994. *Human Development Index: Methodology and Measurement (Occasional Pap. 12).* New York: UN Dev. Programme, Hum. Dev. Rep. Off. http://hdr.undp.org/publications/papers.cfm
55. Green D, Smith KR. 2002. Implications of graduation: Why developing countries will never produce more greenhouse gases than developed countries. *J. Energy Dev.* 28:15–40
56. World Bank. 2002. *World Development Indicators*. Washington, DC: World Bank
57. UN Dev. Programme. 2002. *Human Development Report*. New York: UN Dev. Programme
58. Prüss-Üstün A, Kay D, Fewtrell LJB. 2004. Unsafe water, sanitation, and hygiene. See Ref. 39, pp. 1321–54
59. Curtis V, Cairncross S, Yonli R. 2000. Domestic hygiene and diarrhea—pinpointing the problem. *Trop. Med. Int. Health* 5:22–32
60. Carter R, Mendis KN, Roberts D.

2000. Spatial targeting of interventions against malaria. *Bull. WHO* 78:1401–11

61. Humphreys M. 2001. *Malaria: Poverty, Race, and Public Health in the United States*. Baltimore, MD: Johns Hopkins Univ. Press
62. Lindsay SW, Jawara M, Paine K, Pinder M, Walraven GE, Emerson PM. 2003. Changes in house design reduce exposure to malaria mosquitoes. *Trop. Med. Int. Health* 8:512–17
63. Prüss-Üstün A, Fewtrell L, Landrigan PJ, Ayuso-Mateos JL. 2004. Lead exposure. See Ref. 39, pp. 1495–542
64. Concha-Barrientos M, Nelson DI, Driscoll T, Steenland NK, Punnett L, et al. 2004. Selected occupational risk factors. See Ref. 39, pp. 1651–802
65. McMichael A, Campbell-Lendrum D, Kovats S, Edwards S, Wilkinson P, et al. 2004. Global climate change. See Ref. 39, pp. 1543–650
66. Smith KR. 1991. Allocating responsibility for global warming: the natural debt index. *Ambio* 20:95–96
67. Smith KR. 1996. The natural debt: North and South. In *Climate Change: Developing Southern Hemisphere Perspectives*, ed. T Giambelluca, A Henderson-Sellers, pp. 423–48. London: Wiley
68. Smith KR. 1997. Development, health, and the environmental risk transition. In *International Perspectives in Environment, Development, and Health*, ed. G Shahi, B Levy, A Binger, T Kjellstrom, R Lawrence, pp. 51–62. New York: Springer
69. Smith KR. 1994. *Environmental hazards during economic development: the risk transition and overlap*. Presented at Assess. Manag. Health Risks Drink. Water Contam.: Approaches Appl., Rome
70. Barbier EB. 1997. Introduction: environmental Kuznets curves. *Environ. Dev. Econ.* (Spec. issue) 2:369–81
71. Antle JM, Heidebrink G. 1995. Environment and development: theory and international evidence. *Econ. Dev. Cult. Change* 43:603–25
72. Ezzati M, Singer BH, Kammen DM. 2001. Towards an integrated framework for development and environment: the dynamics of environmental Kuznets curves. *World Dev.* 29:1421–34
73. Arrow K, Bolin B, Costanza R, Dasgupta P, Folke C, Holling CS. 1995. Economic growth, carrying capacity, and the environment. *Science* 168:520–21
74. Ekins P. 1997. The Kuznets curve for the environment and economic growth: examining the evidence. *Environ. Plan. A* 29:805–30
75. Winslow MA. 2002. *Environmental Quality, Economic Growth, and Democracy: A Theoretical and Empirical Examination of the Linkages*. Berkeley: Univ. Calif.
76. Ansuategi A, Perrings C. 2000. Transboundary externalities in the environmental transition hypothesis. *Environ. Resource Econ.* 17:353–73
77. McMichael AJ, Kjellstrom T, Smith KR. 2001. Environmental health. In *International Public Health*, ed. MH Merson, RE Black, AJ Mills, pp. 379–438. Gaithersburg, MD: Aspen

77a. Maugh TH. 1979. Cancer and environment: Higginson speaks out. *Science* 205(4413):1363–66

78. Daily GC, Ehrlich PR. 1996. Development, global change, and the epidemiological environment. *Environ. Dev. Econ.* 1:311–46

78a. Bedford T, Cooke R. 2001. *Probabistic Risk Analysis: Foundations and Methods*. New York: Cambridge Univ. Press

79. Slovic P. 1987. Perception of risk. *Science* 236:280–85
80. Slovic P, Finucane ML, Peters E, MacGregor DG. 2004. Risk as analysis and risk as feelings: some thoughts about affect, reason, risk, and rationality. *Risk. Anal.* 24:311–22
81. Smith KR. 2003. Indoor air pollution and

acute respiratory infections. *Indian Pediatr.* 40:815–19

82. World Health Organ. 2004. *World Health Report 2004: Changing History*. Geneva: WHO
83. Baker EL Jr, Koplan JP. 2002. Strengthening the nation's public health infrastructure: historic challenge, unprecedented opportunity. *Health Aff.* 21:15–27
84. Murray CJL, Salomon JA, Mathers CD, Lopez AD, eds. 2002. *Summary Measures of Population Health: Concepts, Ethics, Measurement and Applications*. Geneva: WHO

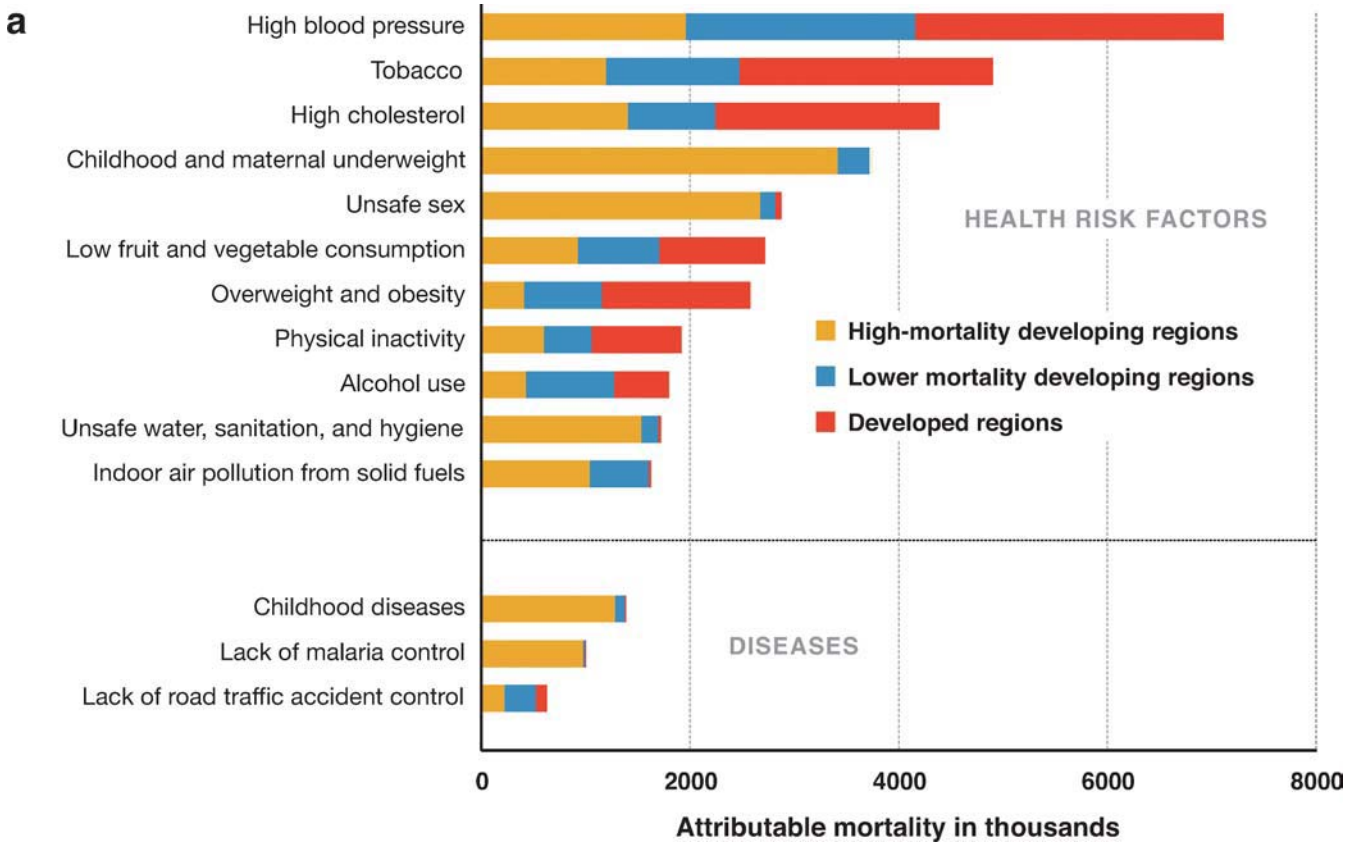

Figure 4 See legend on page C-3

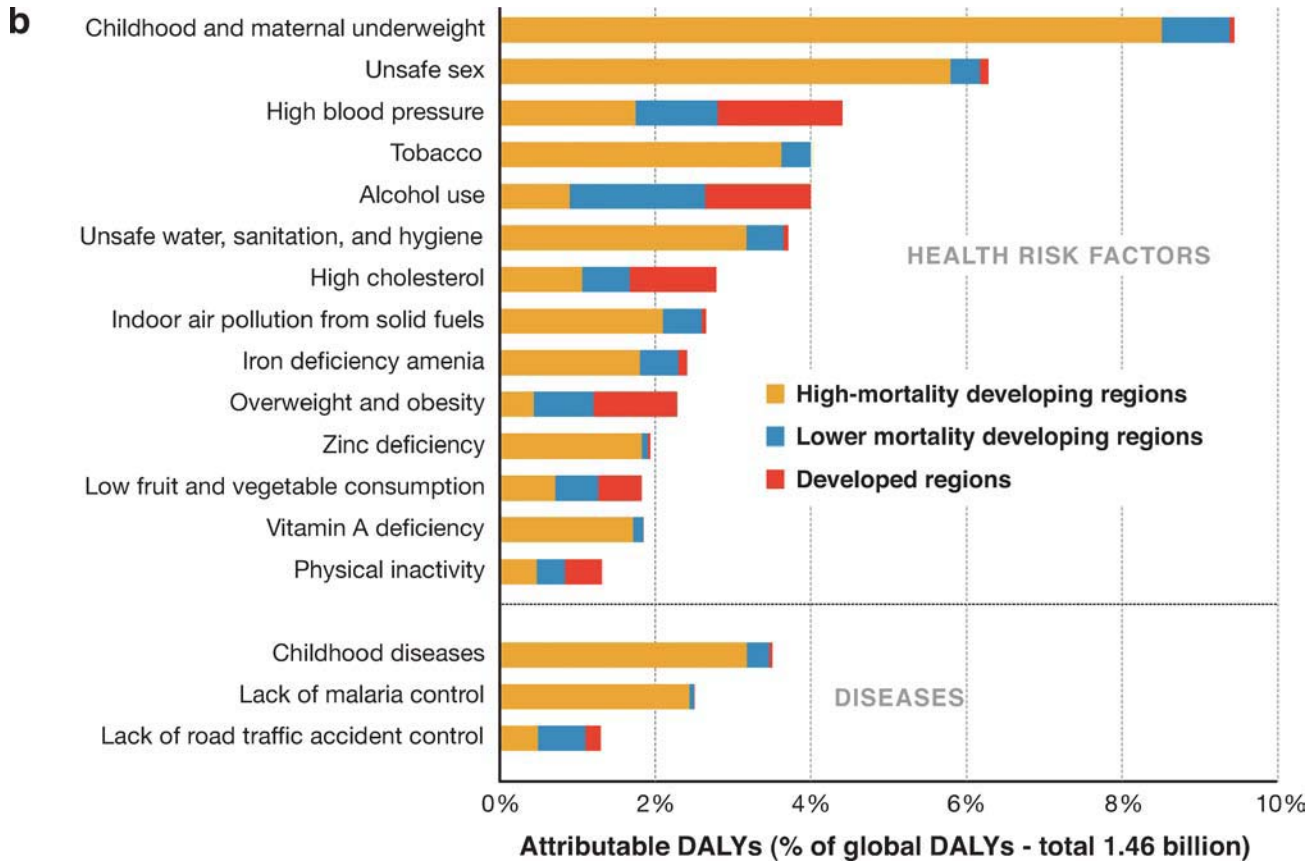

Figure 4 (*Continued*) See legend on next page

Figure 4 (*a*) Mortality and (*b*) burden of disease attributable to leading global risk factors and to major diseases, limited to those with at least 1 million deaths or 1% of the total GBD (see Table 1 for other risk factors). The top group in each figure shows the estimated mortality and disease burden for each risk factor considered individually. These risks act in part through other risks and act jointly with other risks. Consequently, the burden due to groups of risk factors will usually be less than the sum of individual risks. The bottom group in each figure is calculated directly from the burden of each disease (including injuries) based on the assumption that a portion of deaths and burden could be avoided through implementation of feasible interventions that affect their multiple causes (see the section titled Estimated Mortality and Burden of Disease Attributable to Selected Risks). Burden of disease, measured in disability-adjusted life year (DALY), is the sum of years of life lost (YLL) to premature mortality and years of life lived with disability (YLD).

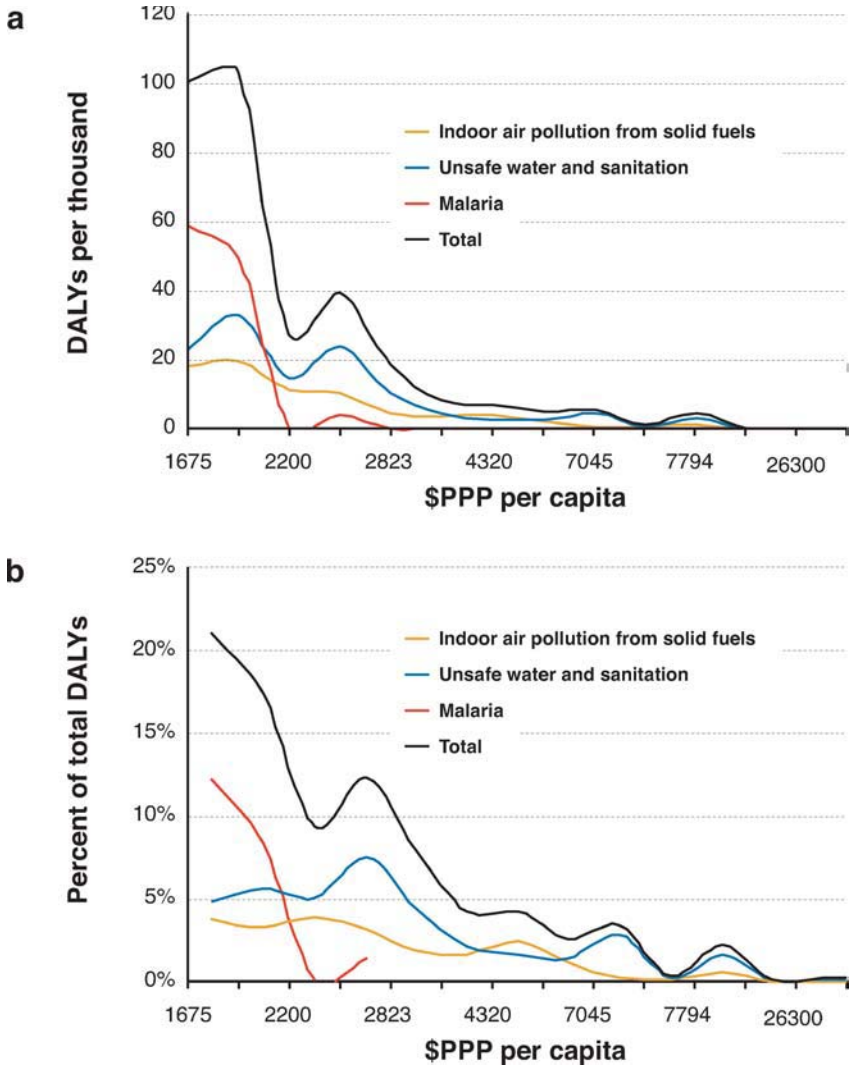

Figure 7 (*a*) Trend of household environmental risks as DALYs per 1000 capita by income measured in dollars of gross domestic product per capita purchasing power ($PPP/capita). (*b*) Percent of total DALYs with development measured in $PPP/capita. (*c*) DALYs per 1000 capita by Human Development Index (HDI). (*d*) Percent of total DALYs with development by HDI. Sources: WHO Comparative Risk Project (39) and UN Development Programme Human Development Report (57).

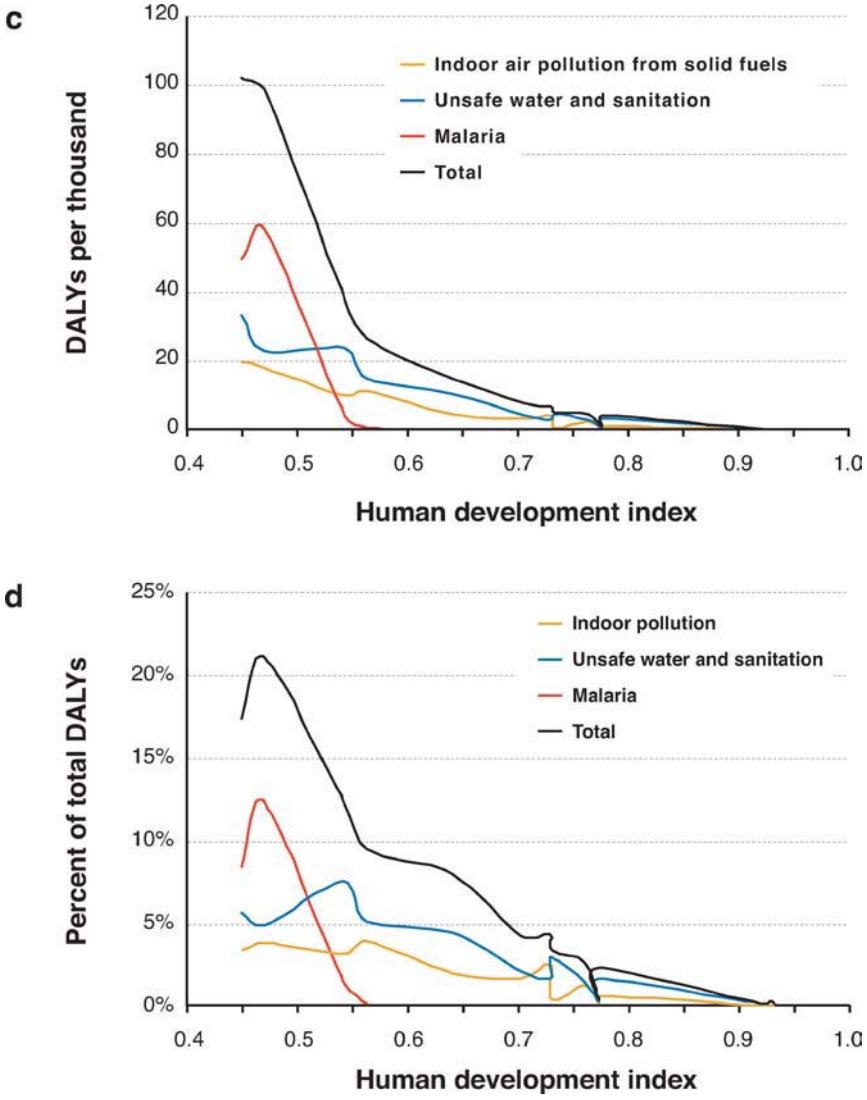

Figure 7 (*Continued*)

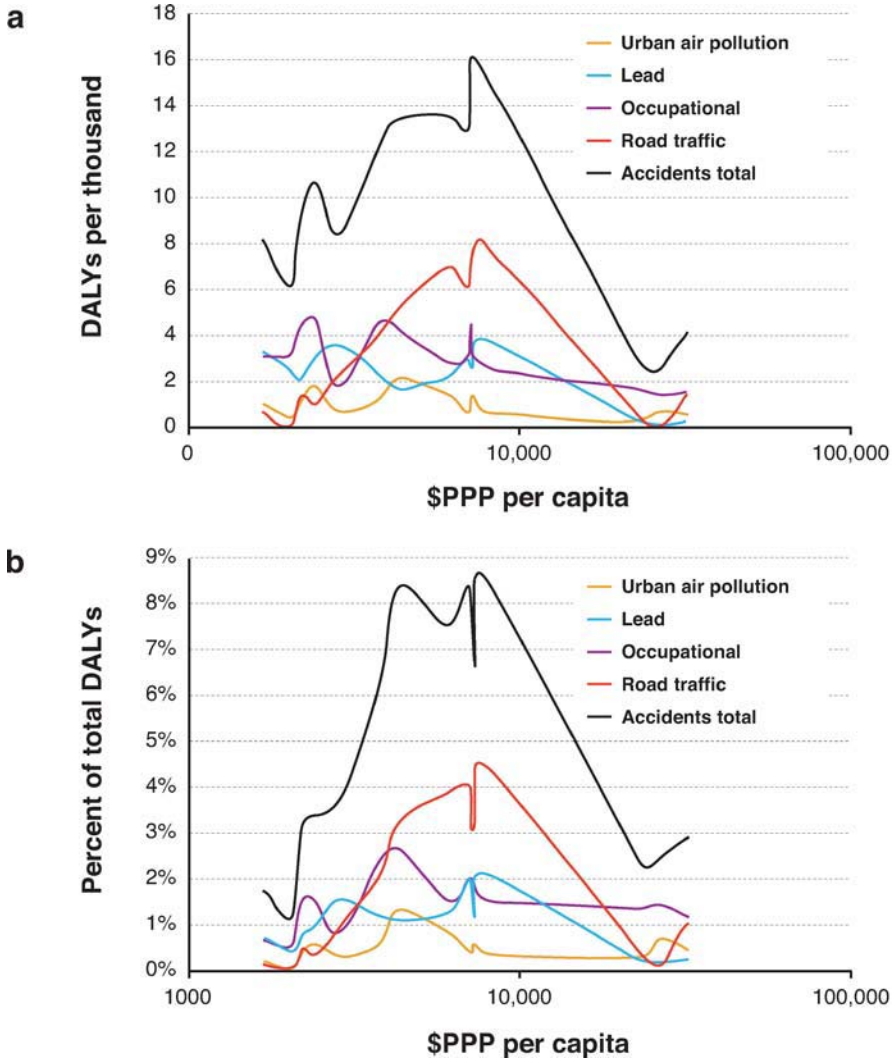

Figure 8 Trend of community environmental risks shown in panel (*a*) as DALYs per 1000 capita and in panel (*b*) as percent of total DALYs and with development measured as per capita purchasing power ($PPP). Sources: WHO Comparative Risk Project (39) and UN Development Program Human Development Report (57).

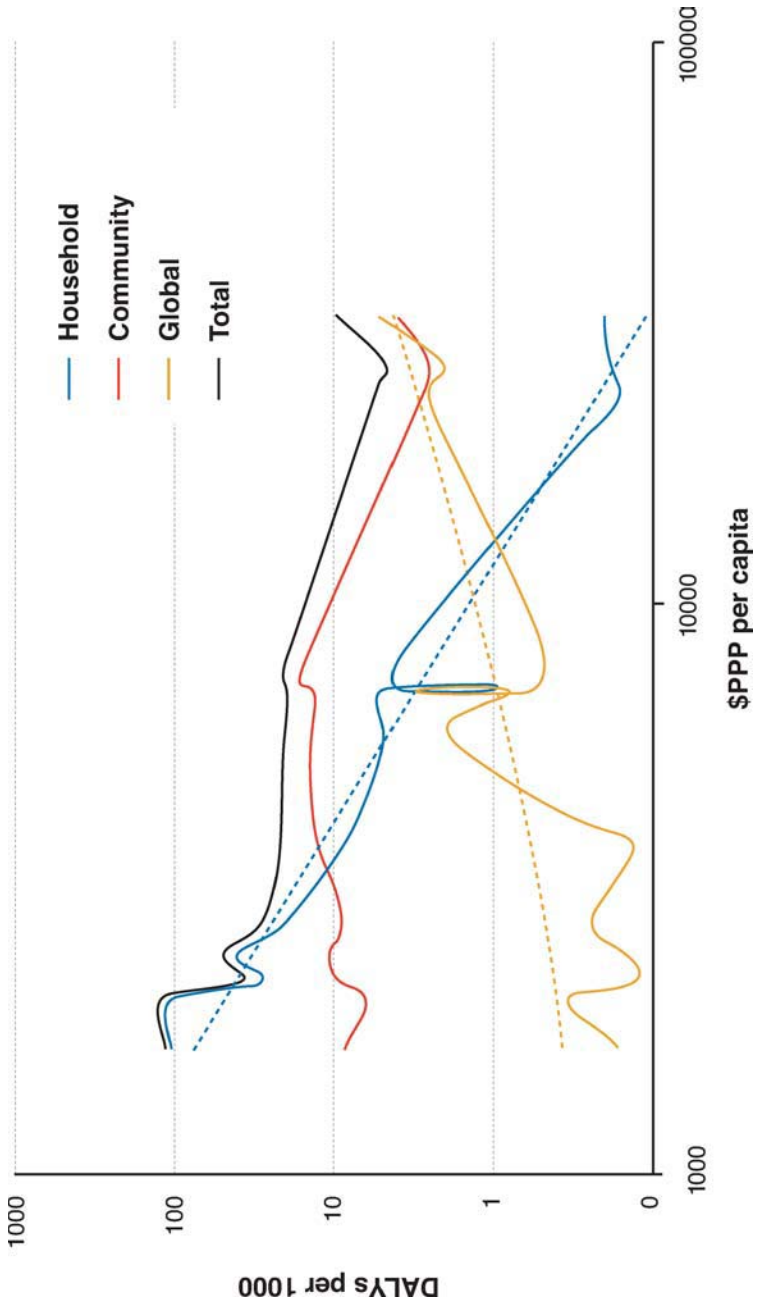

Figure 10 See legend on next page

Figure 10 Full environmental risk transition revisited, using data from Figures 7, 8, and 9, showing trends for household, community, imposed global risks, and total environmental risks. Imposed global risks in each income group are allocated according to each group's mean natural debt. For clarity, trend lines are shown for household and global risks.

Annual Review of Environment and Resources
Volume 30, 2005

CONTENTS

I. EARTH'S LIFE SUPPORT SYSTEMS

Regional Atmospheric Pollution and Transboundary Air Quality Management, *Michelle S. Bergin, J. Jason West, Terry J. Keating, and Armistead G. Russell* 1

Wetland Resources: Status, Trends, Ecosystem Services, and Restorability, *Joy B. Zedler and Suzanne Kercher* 39

Feedback in the Plant-Soil System, *Joan G. Ehrenfeld, Beth Ravit, and Kenneth Elgersma* 75

II. HUMAN USE OF ENVIRONMENT AND RESOURCES

Productive Uses of Energy for Rural Development, *R. Anil Cabraal, Douglas F. Barnes, and Sachin G. Agarwal* 117

Private-Sector Participation in the Water and Sanitation Sector, *Jennifer Davis* 145

Aquaculture and Ocean Resources: Raising Tigers of the Sea, *Rosamond Naylor and Marshall Burke* 185

The Role of Protected Areas in Conserving Biodiversity and Sustaining Local Livelihoods, *Lisa Naughton-Treves, Margaret Buck Holland, and Katrina Brandon* 219

III. MANAGEMENT AND HUMAN DIMENSIONS

Economics of Pollution Trading for SO_2 and NO_x, *Dallas Burtraw, David A. Evans, Alan Krupnick, Karen Palmer, and Russell Toth* 253

How Environmental Health Risks Change with Development: The Epidemiologic and Environmental Risk Transitions Revisited, *Kirk R. Smith and Majid Ezzati* 291

Environmental Values, *Thomas Dietz, Amy Fitzgerald, and Rachael Shwom* 335

Righteous Oil? Human Rights, the Oil Complex, and Corporate Social Responsibility, *Michael J. Watts* 373

Archaeology and Global Change: The Holocene Record, *Patrick V. Kirch* 409

IV. EMERGING INTEGRATIVE THEMES

Adaptive Governance of Social-Ecological Systems,
Carl Folke, Thomas Hahn, Per Olsson, and Jon Norberg 441

INDEXES

Subject Index 475
Cumulative Index of Contributing Authors, Volumes 21–30 499
Cumulative Index of Chapter Titles, Volumes 21–30 503

ERRATA

An online log of corrections to *Annual Review of Environment and and Resources* chapters may be found at http://environ.annualreviews.org