People have recognized the relationship between the state of the environment and human health for many years. Dr. John Snow, considered the father of modern epidemiology, is best remembered for linking the 1854 London cholera outbreak to the local drinking water supply (McMichael 2001). The smog episodes in Donora, Pennsylvania, in late October 1948 and in London in December 1952 (Davis 2002), as well as Rachel Carson’s work on the effects of agricultural chemicals (Carson 1962) were key events in initiating the modern environmental movement. In recent years, concern about the implications of the environment for health has expanded to include such emerging threats as climate change, stratospheric ozone depletion, antibiotic resistance, and the potential effect of hormonally active agents in the environment (Daily and Ehrlich 1996; Diamanti-Kandarakis et al. 2009; National Research Council 1999; WHO 2008b).1

Smith and Ezzati (2005), in their concept of an environmental risk transition, captured the progression from more local and traditional to more global and modern environmental threats to human health as societies develop. In this chapter, we explore the changing relationships between selected aspects of environment and human health and how these may play out in the context of environmental change. In doing so, we must cope with significant uncertainty, not only in identifying the specific roles that the environment plays as a determinant of human health, but also in incorporating environmental change into our projections of health outcomes. As Corvalán and Campbell-Lendrum noted, while we often know enough about environmental influences to make either quantitative or qualitative projections of eventual health outcomes … even for relatively well studied exposures, it is possible to make only approximate and incomplete projections, because we will always lack quantitative...
information on some of the multiple inter-linkages between environmental drivers and health. (2005: 23–24)

This problem is compounded by the fact that in many cases we also lack quantitative information on the environmental drivers themselves and on the numerous other factors that mediate the effect of the environment on human health. Still, there is much we can and need to say about the role of the environment in human health.

**Environmental Risk Factors and the Environmental Risk Transition**

In Chapter 2, we placed human health into a more general framework of human-environment relationships (see again Figure 2.4). Reflecting the environment’s ubiquitous nature, Smith and Ezzati (2005: 325) referred to the environment as a super-distal risk factor in that it “affects essentially every disease, even if the pathways are not always well understood.” In a similar vein, Huynen (2008) identifies the many roles that the environment can play as a driver of human health outcomes at the contextual, distal, and proximate levels. The effects include direct impacts from exposure to physical (e.g., temperature and radiation), biotic (e.g., disease pathogens), and chemical (e.g., pollution) factors, as well as indirect impacts related to effects of the environment and environmental change on other drivers of human health. An example of the latter would be the potential impact of climate change on food production and, consequently, its effects on childhood underweight and associated diseases.

As we noted in Chapter 2, given the role that environment plays in the evolutionary mechanisms of mutation and natural selection, a complete consideration of the environment would ideally include not only the environment to which individuals are exposed but also the environment to which their ancestors were exposed (Smith, Corvalán, and Kjellström 1999). Such a broad exploration of the role of the environment would take us well beyond the scope of the present analysis. It would also bring us up against the boundaries of society’s present understanding of the linkages between the environment and human health, especially in terms of our ability to quantify these linkages. This is frustrating, particularly as it limits our capacity to address many of the most significant and growing concerns about the environmental drivers of human health in the future. These include the effects of the growing chemical body burden associated with modern economies (Thornton, McCally, and Houlihan 2002); the growth of antibiotic resistance (Martínez 2009); and the (re-)emergence of old and new infectious diseases (Jones et al. 2008).

Most of the analysis presented in this chapter draws from and builds on the work of the World Health Organization’s (WHO) Comparative Risk Assessment (CRA) project. Recall from Chapter 2 that the CRA limited its focus to “risk factors for which there was good potential for satisfactory quantification of population exposure distributions and health effects using the existing scientific evidence and available data, and for which intervention strategies are available or might be envisioned to modify their impact on disease burden” (Ezzati et al. 2004b: xx). As such, the CRA project has included the following specific environmental risk factors: unsafe water, sanitation, and hygiene; urban air pollution; indoor smoke from solid fuels; lead exposure; and climate change (Ezzati et al. 2004a). Even these analyses have been somewhat limited in scope; for example, the quantitative estimates for urban air pollution have extended only to the effects from particulate matter in urban areas with populations over 100,000 and national capitals on three health outcomes: acute respiratory infections in children under five, and lung cancer and selected cardiovascular diseases for adults over 30 (Cohen et al. 2004).

As part of its work on the environmental burden of disease, WHO has expanded this assessment to include solar ultraviolet radiation and mercury among other risk factors, as well as paying attention to the relationships between the environment and malnutrition and between the environment and poverty. The assessments also often include occupational risk factors as part of their consideration of environmental risk factors, which we have chosen not to do. Furthermore, they consider a much wider range of health outcomes (Prüss-Üstün and Corvalán 2006). All of this is important to keep in mind when interpreting WHO’s quantitative estimates of the environmental burden of disease, to which we turn in the next section.
In Chapter 5, we introduced the concept of a transition in health risks over time (see Figure 5.1) in which the work of Smith (1990) was seminal. While Smith paid special attention to environmental issues from the start, he soon developed the specific concept of an *environmental risk transition* (Smith 1997). As a complement to the concepts of demographic and epidemiologic transitions, this concept can help us structure our thinking about the evolution of environmental health risks over time. The basic premise is that as societies develop, environmental risks have a tendency to move from the household (e.g., poor water, sanitation, and hygiene and indoor air pollution), to the community (e.g., outdoor air pollution), and then to the globe (e.g., climate change and stratospheric ozone depletion), in what Smith (2001) has referred to as a “sequential housekeeping effort” (see Figure 6.1). In the case of the move toward global environmental risks, it is important to note that these are defined not only by where the impacts occur but also by where the risks originate. For example, the emission of greenhouse gases from the burning of fossil fuels, the primary driver of global climate change, shows a clear increase with income even as the potential effects are expected to fall primarily on persons in poorer countries.

A number of important aspects of the environmental risk transition may not be evident from this simple description, and are not captured in Figure 6.1. First, both the absolute level of health risk from environmental factors and the share of the total burden of disease attributable to the environment are expected to fall as societies proceed along the risk transition. Second, as societies proceed through the risk transition, in addition to moving from more local to more global scales, there is a tendency for an increasing time delay between what causes the risk and the emergence of the risk, as well as between the emergence of the risk and its subsequent health effects. Chapters 3 and 5 discussed this issue of *latency period* with respect to smoking, but it is also significant with respect to environmental pollutants such as airborne lead and the effects of ozone depletion and climate change. Third, the health impacts realized along the risk transition have a tendency to increasingly reflect multiple stresses, making it harder to draw a clear association between the cause and the effect. In addition, there is an increasing potential for low-probability, high-consequence events, as is currently being discussed in relation to climate change. Finally, recalling the double burden of disease discussed in previous chapters, during the environmental risk transition there will be periods of overlap, where groups continue to be affected by traditional risks even as exposure to modern risks is increasing. A clear example of this occurs in the slums of rapidly growing urban areas, where there is frequently a low level of access to improved sources of drinking water and sanitation combined with urban air pollution.

The premise of the “environmental risk transition” is that as societies develop, environmental risks move from the household to the community and then to the globe.

**Figure 6.1 The environmental risk transition**

![Figure 6.1 The environmental risk transition](image)


**The Environment and Human Health: The Empirical Evidence**

WHO’s work on the environmental burden of disease provides some of the only, and certainly the most comprehensive and
consistent, quantitative information on the importance of the environment to human health.\(^4\) In *Preventing Disease Through Healthy Environments: Towards an Estimate of the Environmental Burden of Disease*, Prüss-Üstün and Corvalán (2006) presented, for the year 2002, the first comprehensive estimates of the impact of the environment on 85 disease and injury categories. They derived these results using a combination of methods developed in the CRA project, other estimates from the literature, and a survey of 100 experts. More recently, in *Global Health Risks: Mortality and Burden of Disease Attributable to Selected Major Risks* (WHO 2009a) and on an associated website,\(^5\) they provide estimates for the year 2004; the 2004 estimates are based on the methods developed in the CRA project only.

Table 6.1 summarizes the estimated attributable deaths and disability-adjusted life years (DALYs), as well as the share of specific diseases attributable to each listed risk factor (PAF), at the global level for the year 2004. The authors argued that these should be considered as conservative estimates of the total burden of disease from the environment for the following reasons. First, they included only the major environmental risk factors. Second, the disease burden attributable to environmental factors is not always quantifiable, even where health impacts are readily apparent and fairly well understood. Third, as they themselves noted, their definition of the environment is not comprehensive, because it only includes those aspects of the natural environment that are

The burden of disease from environmental risk factors most affects children and the elderly in poorer regions.

<table>
<thead>
<tr>
<th>Risk factor and major associated diseases</th>
<th>Deaths</th>
<th>DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total (thousands)</td>
<td>PAF</td>
</tr>
<tr>
<td>Unsafe water, sanitation, and hygiene</td>
<td>1,908</td>
<td>88.2%</td>
</tr>
<tr>
<td>Diarrheal diseases</td>
<td>1,908</td>
<td>88.2%</td>
</tr>
<tr>
<td>Indoor smoke from solid fuels</td>
<td>1,965</td>
<td>35.0%</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>1,058</td>
<td>35.0%</td>
</tr>
<tr>
<td>Lower respiratory infections</td>
<td>872</td>
<td>20.9%</td>
</tr>
<tr>
<td>Urban outdoor air pollution</td>
<td>1,152</td>
<td>2.8%</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>341</td>
<td>4.7%</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>298</td>
<td>5.2%</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>168</td>
<td>5.5%</td>
</tr>
<tr>
<td>Lower respiratory infections</td>
<td>118</td>
<td>2.8%</td>
</tr>
<tr>
<td>Trachea, bronchus, lung cancers</td>
<td>108</td>
<td>8.2%</td>
</tr>
<tr>
<td>Lead exposure</td>
<td>143</td>
<td>8,977</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>64</td>
<td>0.9%</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>53</td>
<td>0.9%</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>19</td>
<td>1.9%</td>
</tr>
<tr>
<td>Mental retardation, lead-caused</td>
<td>0</td>
<td>0.0%</td>
</tr>
<tr>
<td>Global climate change</td>
<td>141</td>
<td>5,404</td>
</tr>
<tr>
<td>Diarrheal diseases</td>
<td>65</td>
<td>3.0%</td>
</tr>
<tr>
<td>Malaria</td>
<td>27</td>
<td>3.0%</td>
</tr>
<tr>
<td>Lower respiratory infections</td>
<td>17</td>
<td>0.4%</td>
</tr>
<tr>
<td>Total</td>
<td>5,309</td>
<td>128,378</td>
</tr>
</tbody>
</table>

Notes: Totals for risk factors in each grouping include diseases in addition to those listed, so may exceed the sum of the risk factors for the listed diseases; PAF is the “population attributable fraction,” or the share of the burden of each disease attributable to the risk factor with which it is listed.

Unsafe water, sanitation, and hygiene (WSH) and indoor air pollution constitute the most significant environmental risk factors, each accounting for nearly 2 million annual deaths and more than 40 million DALYs. Prüss-Üstün and Corvalán estimated further that urban outdoor air pollution accounts for more than a million deaths, but approximately the same number of DALYs as lead exposure. Although lead exposure and global climate change currently represent much smaller risks in terms of mortality, there is much greater concern about the potential of the latter to become increasingly important in future years, both directly and through its influence on other risk factors.

In interpreting the results in Table 6.1, it is important to note that the values for total deaths and DALYs conflate the extent to which the environment contributes to a specific disease and the total burden represented by that disease. For some diseases, such as intestinal nematode infections, trachoma, schistosomiasis, dengue, and Japanese encephalitis, more than 95 percent of the disease burden can be attributed to environmental risk factors, but the total incidence of these diseases is so small that they contribute only a small amount to the total burden of disease attributable to environmental risk factors. Alternatively, even though only 14 percent of the total incidence of cardiovascular diseases is attributable to environmental factors, they rank first among all diseases in terms of total global deaths attributable to the environment because cardiovascular diseases are much more common (Prüss-Üstün and Corvalán 2006).

There are significant differences in the distribution of the burden of disease from environmental risk factors across regions and age groups. The risks most affect children and the elderly in poorer regions. This differs to some degree based on the specific risk factor and disease, as we will see later in this chapter. Returning to our earlier discussion of the environmental risk transition, Figures 6.2 and 6.3, using cross-sectional data for the year 2004, lend support to two of its main hypotheses. Figure 6.2 shows that not only do total DALYs decline with increasing income but...
so does the share of total DALYs attributable to environmental risk factors. A similar graph using deaths instead of DALYs would show the same pattern. Figure 6.3 illustrates a pattern similar to that shown in Figure 6.1—namely, a clear downward trend with income for unsafe water, sanitation, and hygiene and indoor air pollution, which are characterized as household environmental risks. For outdoor air pollution, a community environmental risk, there is some suggestion of an increase as incomes move from a very low to a medium level, followed by a decline at higher income levels. The significant amount of deviation from these general patterns, however, points to the importance of factors other than average income as determinants of environmental risk (see again Box 5.1).

Environment and Human Health: Risk Factors Now and in the Future

Identifying, much less quantifying, the current and future effects of the environment on human health can be a daunting task. Our general approach to quantification is to start with WHO’s work on the current environmental burden of disease and to extend this dynamically so as to forecast how selected environmental risk factors might affect the future burden of disease.

Following the categorization laid out by Smith (1990), we start with household risk factors (unsafe water, sanitation, and hygiene and indoor air pollution), then move to a community risk factor (outdoor air pollution), and then explore a global risk factor (climate change). We have not included lead exposure, due to both its relatively small and decreasing role compared to the other risk factors and the difficulty in adapting the methods used by WHO to estimate the burden of disease associated with this risk factor. This is not to minimize its importance. More generally, we recognize that we are addressing an incomplete set of environmental risk factors and diseases (we focus mostly on traditional risks affecting developing countries). Certainly, there is a much larger set of risk factors and pathways through which the environment has and will play a role in determining human health.

Water, sanitation, and hygiene

Water is fundamental to human health. We use it to clean ourselves, our food, our clothes, and our general surroundings; we ingest it directly; we are largely made of it. At the same time, it provides a breeding ground and source of transmission for a number of disease vectors. Thus, it is not surprising that access to clean water, or more commonly the lack of it, has been a focus of attention in the discussion of health and the environment. The Millennium Development Goals reflect this—target 3 of MDG 7 is to halve, between 1990 and 2015, the proportion of the population without sustainable access to safe drinking water and basic sanitation (UN 2009). Prüss-Ustün et al. (2004: 1322), in defining unsafe water, sanitation, and hygiene (WSH) as a human health risk, included “the ingestion of unsafe water, lack of water linked to inadequate hygiene, poor personal and domestic hygiene and agricultural practices, contact with unsafe water, and inadequate development and management of water resources or water systems.” Thus, there are many transmission pathways by which unsafe water, sanitation, and hygiene present a risk to health. The diseases most associated with unsafe water, sanitation, and hygiene fall into two general categories: (1) those primarily affected by water supply, sanitation, and hygiene—diarrheal diseases, intestinal nematode infections, diseases related to malnutrition, schistosomiasis, trachoma, and lymphatic filariasis; and (2) those primarily affected by poor water resources management—malaria, onchocerciasis, dengue, and Japanese encephalitis. Unsafe water, sanitation, and hygiene has a further, indirect, health impact through its impact on the level of childhood underweight and associated diseases. As noted in the previous chapter, even when children have access to adequate amounts of food, if they suffer from diseases such as diarrhea they do not retain the full calories they consume.

WHO estimated that globally nearly 2 million deaths and over 64 million DALYs related to diarrheal diseases were attributable to unsafe water, sanitation, and hygiene in 2004 (see again Table 6.1). These represented approximately 88 percent of the total burden of disease from diarrheal diseases, and 3.2 percent of all deaths and 4.2 percent of all DALYs. Including all diseases, Fewtrell et al. (2007) estimated that 6.3 percent of all deaths and 9.1 percent of all DALYs were attributable to unsafe water, sanitation, and hygiene in 2002.
Prüss-Üstün et al. (2004) and Fewtrell et al. (2007) described the methods used to estimate these figures. It is only for diarrheal diseases that they spelled out a detailed methodology for estimating the share of the diseases attributable to unsafe water, sanitation, and hygiene. They assumed that all incidences of intestinal nematode infections, schistosomiasis, trachoma, and some vector-borne diseases (e.g., Japanese encephalitis and dengue in certain regions) result from unsafe WSH. They did not include other vector-borne diseases—malaria, lymphatic filariasis, onchocerciasis, and dengue in certain regions—but for future estimates they recommended expert judgment based on local circumstances. This is also the case for malnutrition.

We adapt their methodology for diarrheal diseases in order to get a sense of how changes in the access to improved drinking water and sanitation may influence future health outcomes. Since our methodology and analysis are limited to diarrheal diseases, the results presented here are necessarily underestimates of the health impacts of inadequate WSH.

Drivers and forecasts of access to improved drinking water and sanitation
The development of infrastructure providing access to improved drinking water and sanitation has been a fundamental component of development in modern times, particularly in urban areas. Yet only since 2000, with the Global Water Supply and Sanitation Assessment (WHO and UNICEF 2000) and the WHO/UNICEF Joint Monitoring Programme (WHO and UNICEF 2008), have there been comprehensive data on levels of access in most countries.

Rather than forecasting actual levels of access to improved drinking water and sanitation, most studies identify a target level, as has been done for the MDGs (see, for example, OECD 2006). In one of the only examples providing future projections, WHO (Prüss-Üstün et al. 2004) assumed that the number of people who acquired coverage between 2000 and 2030 would follow the trend of 1990 to 2000, except for countries in Central and Eastern Europe, which actually saw declines during that decade. For these countries, they assumed no change in coverage. In total, they projected the share of the global population without access to improved water would decrease from 23 percent in 2000 to 7 percent in 2030, and those without access to improved sanitation would fall from 51 percent in 2000 to 17 percent in 2030.

Lacking an existing formulation, we estimated separate cross-sectional relationships between access to improved drinking water and sanitation...
and other socio-economic indicators using recent historical data. The key explanatory variables we identified were income per capita, income distribution, education, government expenditures on health, and the rural share of population.

Figure 6.4 presents the results for our base case scenario. At the global level, we forecast less of an improvement than the earlier WHO estimates for 2030, with the percentage of population without access to improved water and sanitation being 10 percent and 27 percent, respectively. Figure 6.4 also highlights significant differences across regions at the present time, with sub-Saharan Africa lagging furthest behind, particularly in terms of access to improved sources of water. These differences persist even as all regions improve their access over the scenario period. Finally, the access to improved sanitation tends to lag behind access to improved drinking water.

**Improved drinking water and sanitation: Health effects under alternative scenarios**

Of the more than 2.1 million deaths from diarrheal diseases in 2005, 1.7 million occurred among children under five. Given our base case forecast of progress in access to improved drinking water and sanitation, the burden of disease from diarrheal diseases is likely to fall significantly in the future. The number of children under five dying from diarrheal diseases is projected to fall to just over half a million by 2030 and around 130,000 in 2060 in our base case scenario. Sub-Saharan Africa remains the most affected region in terms of both absolute numbers and mortality rates, but even here dramatic improvement is seen, with the probability of a child dying from diarrheal diseases falling from 25 per 1,000 live births in 2005 to only 2.4 per 1,000 live births in 2060.

Still, there is room for action to enhance these improvements by directly addressing the issue of access to improved water and sanitation. In addition to our base case, we considered two alternative scenarios: a fast improvement scenario and a slow improvement scenario. In these cases, the percentages of households without access are gradually adjusted such that they are, respectively, one standard error below or above the base case projections by the year 2030 and remain one standard error below or above the base case forecasts for the remainder of the period. The standard errors for water and sanitation were taken from estimated cross-sectional relationships, following the logic laid out in Box 5.1. Figure 6.5 presents the differences between these two scenarios in terms of childhood mortality from diarrheal diseases. The effect is most dramatic in the earlier years, when diarrheal diseases are more prevalent in general, with a peak difference of more than 330,000 deaths in the mid-2020s. After this time, the differences between the scenarios decrease as the general decline in diarrheal deaths in both scenarios overtakes the effects of differences in access to improved water and sanitation. The largest differences occur in sub-Saharan African and South Asia, reflecting their dominant share in diarrheal deaths.

**Indoor air pollution**

People spend much of their time indoors. While shelters provide some protection against air pollution from the outside, there are also numerous sources of air pollution inside buildings. Examples include inter alia the structures themselves, furnishings, cleaning products, fuels used for heating and cooking, certain behaviors such as smoking, and sources from the underlying soil such as radon.
Using solid fuel use in households as a proxy, WHO estimated that in 2004 indoor air pollution caused almost 10 percent of all deaths in some countries.

Over 80 percent of the population in sub-Saharan Africa use solid fuels as a primary household energy source; in our base case 30 percent still do so in 2060.

“Fast” rather than “slow” improvement in indoor air pollution might avert as many as 225,000 child deaths annually from respiratory infections alone in the early 2020s.

These result in exposures to numerous air pollutants that can exceed those from outdoor air pollution by orders of magnitude (Desai, Mehta, and Smith 2004; Smith, Mehta, and Maeusezahl-Feuz 2004; Zhang, Bi, and Hiller 2007). The health effects of many of these pollutants, particularly of newer chemicals, are not currently well understood, but they have been significant enough to introduce a new term in the health lexicon—“sick building syndrome” (Zhang and Smith 2003).

WHO (2009a) provided estimates of the burden of disease from indoor air pollution for three disease outcomes—acute respiratory infections (ARI) for children under five, chronic obstructive pulmonary disease (COPD) for adults over 30, and trachea, bronchus, and lung cancers for adults over 30.9 Lacking detailed data on actual exposure to indoor air pollution, WHO researchers used estimates of solid fuel use in the household, adjusted for ventilation, as a proxy. Notably, they did not include exposure from tobacco smoke or the use of synthetic chemicals (Zhang and Smith 2003).

While recognizing these limitations, WHO (2009a) estimated that in 2004 indoor air pollution was responsible for a minimum of 1.9 million deaths and 40.4 million DALYs (see again Table 6.1). In some countries, indoor air pollution accounted for nearly 10 percent of all deaths and DALYs.10 COPD and ARI made up 54 and 44 percent of these deaths, respectively; because it affects children, however, ARI dominated DALYs—75 percent versus 24 percent for COPD. Cancers presented a much smaller burden of disease. ARI is fairly evenly split among boys and girls (52 percent versus 48 percent of both deaths and DALYs in 2004), but COPD and cancers tend to affect women more than men (60 percent of deaths and 56 percent of DALYs in 2004). Separately, Rehfuess, Mehta, and Prüss-Üstün (2006) estimated that indoor air pollution is responsible for 40 percent of global deaths from COPD among women, independent of the effects of smoking; for men, it is responsible for around 10 percent (women have greater exposure because they do most of the cooking). Finally, Figure 6.3 illustrated that the burden of disease attributable to indoor air pollution generally falls with income levels, in line with a decline in solid fuel use in the home.

We adapted the methodology described in Desai, Mehta, and Smith (2004) and Smith, Mehta, and Maeusezahl-Feuz (2004) to estimate the effects of solid fuel use on ARI for children under five and COPD for adults over 30. We have not included cancers as their contribution to the burden of disease from this risk factor is quite small compared to those of ARI and COPD. Given our disease categories, we use respiratory infections and respiratory diseases as proxies for ARI and COPD, respectively.11

Drivers and forecasts of solid fuel use for heating and cooking

Historical values on solid fuel use for heating and cooking are available from WHO12 and as part of the data for the Millennium Development Goal Indicators data set,13 and Smith, Mehta, and Maeseuzel-Feuz (2004) and Rehfuess, Mehta, and Prüss-Üstün (2006) provided estimates of ventilation. In general, there is a relative paucity of these data, at least at a globally consistent level. And we were unable to find any projections of solid fuel use for heating and cooking. Smith, Mehta, and Maeseuzel-Feuz (2004) and Rehfuess, Mehta, and Prüss-Üstün (2006) described formulations they have used to estimate solid fuel use for countries for which they had no data. We were unable to adopt these directly, because either they did not provide sufficient detail or they included explanatory factors that are not currently in IFs.

Lacking an existing formulation, we estimated a cross-sectional relationship between the use of solid fuels for heating and cooking and other socio-economic indicators using recent historical data. The key explanatory variables we identified were income per capita, income distribution, and education.14

Figure 6.6 presents current and future estimates of solid fuel use for our base case scenario. We did not assume any changes in the ventilation coefficients over time in this scenario. The data show significant differences across regions. As with unsafe water and sanitation, the greatest exposure is in sub-Saharan Africa, where more than 80 percent of the population currently uses solid fuels as a primary household energy source. South Asia and East Asia and Pacific also exhibit high levels of solid fuel use, but it should be noted that in China, which dominates the East Asia and Pacific
region, there is extensive use of cleaner stoves and improved ventilation, which results in less exposure to indoor air pollution (Smith, Mehta, and Maeusezahl-Feuz 2004). The levels fall over time in the base case, but we forecast that even in 2060 more than 30 percent of the population in sub-Saharan Africa and more than 10 percent in South Asia will still use solid fuel.

**Indoor air pollution: Health effects under alternative scenarios**

As we did with unsafe water, sanitation, and hygiene, we explore three scenarios in order to understand the effect of indoor air pollution on future health. These include the base case scenario, a fast improvement scenario, and a slow improvement scenario. In these latter two cases, the percentage of households using solid fuels are gradually adjusted such that they are, respectively, one standard error below or above the base case projections by the year 2030 and then remain one standard error below or above the evolving base case projections for the remainder of the period. The standard errors for solid fuel use were taken from estimated cross-sectional relationships, following the logic laid out in Box 5.1. Please note that these differences can be interpreted as a combination of differences in reductions in solid fuel use and improvements in ventilation through such actions as the increased use of improved cook stoves.

The story for respiratory infections among children under five is very similar to that for diarrheal diseases discussed in the previous section. In the base case, total global deaths fall from just under 1.8 million in 2005 to around 590,000 in 2030 and 158,000 in 2060. Most of these deaths occur in sub-Saharan Africa and South Asia, with these two regions accounting for over 85 percent of the deaths in 2005 and over 90 percent in 2060. A comparison of the slow and fast improvement scenarios (Figure 6.7) shows a peak difference on the order of 225,000 deaths in the early 2020s. As with diarrheal diseases, the size of this difference in terms of number of deaths decreases in later years as the overall level of mortality from respiratory infections among children declines. Even so, there are still over 50,000 more deaths in the slow improvement scenario in 2060.

The story for respiratory diseases among adults over 30 is somewhat more complicated. This is in large part because the size of the adult, and particularly the elderly, population is projected to grow significantly between now and mid-century. In our base case scenario, global mortality from respiratory diseases among adults over 30 rises...
from around 3.4 million in 2005 to almost 7 million in 2030 and over 14 million in 2060. In addition, the population attributable fraction (PAF) of indoor air pollution on respiratory diseases among adults over 30 is somewhat larger than on respiratory infections of children. Thus, we have a larger PAF operating on a much larger base over time. Figure 6.8, which shows the difference in mortality between the slow and fast improvement scenarios, reflects this. The difference peaks around 2030, when it exceeds 915,000 deaths, after which time it declines only slowly, still maintaining a level over 875,000 in 2060. Also, while a few regions still dominate the results, the effects are spread more evenly across regions, reflecting the broader distribution of respiratory diseases among adults in general.

**Urban outdoor air pollution**

At the start of this chapter, we noted the smog episodes in Donora, Pennsylvania, and in London as key events that stimulated the modern environmental movement. Although many countries have worked to improve their air quality over the past several decades, air pollution continues to be a significant health threat, particularly in rapidly growing urban areas in developing countries. Furthermore, recent studies point to significant adverse health effects even at the relatively low concentrations of urban areas in developed countries (Cohen et al. 2004; Krewski et al. 2009; Pope and Dockery 2006).

WHO (2009a) estimated that urban outdoor air pollution was responsible for at least 1.1 million deaths and 8.7 million DALYs in 2004 (see again Table 6.1). These estimates only considered urban areas with populations over 100,000 and national capitals. More notably, they only considered the effect of particulate matter, even though other air pollutants, specifically ground-level ozone, are also known to have significant health effects (Cohen et al. 2004). Unlike some of the other risk factors considered in this chapter, the poorest countries do not dominate the disease burden from outdoor air pollution. Instead, it is those countries that are in the middle stages of development (see again Figure 6.3). This is in line with Smith and Ezzati’s (2005) description of outdoor air pollution as a “community” risk.

For our analysis, we followed the general procedure laid out by Cohen et al. (2004) and by Ostro (2004) for WHO. This narrowed our focus to particulate matter, and we look at those particulates with a diameter of 2.5 micrometers or less (PM$_{2.5}$). Furthermore, we concentrate on the effects on cardiopulmonary diseases for persons over 30. As a proxy for cardiopulmonary diseases, we use the sum of respiratory infections and respiratory diseases plus one half of cardiovascular diseases.

**Drivers and forecasts of urban air quality**

Local economic activity, particularly through pollution from industry, electricity generation, and transportation, is the primary driver of urban air quality. At the same time, local topography and meteorologic patterns strongly influence it, as is epitomized by the Los Angeles basin, where the mountains that border the region on the east act to impede the movement of air masses. Most countries have implemented policies to try to improve urban air quality due to its known health effects. Many large urban areas monitor air quality, including particulate matter concentrations.

In recent years, the Global Model of Ambient Particulates (GMAPS) (Pandey et al. 2006) and the Global Urban Air quality Model (GUAM)
(Bakkes and Bosch 2008) have been developed to fill data gaps in PM$_{10}$ levels in world cities using demographic, geographic, meteorologic, and emissions data. We cannot use such models for forecasting within IFs, however, because of their data requirements and the local scale at which they operate. Fortunately, the World Bank has used the more detailed data to develop national estimates of population-weighted annual average PM$_{10}$ concentrations in residential areas of urban centers, which they provide as part of their World Development Indicators. Using these data, we were able to estimate a relationship between the PM$_{10}$ values and key socio-economic variables, including income per capita, income distribution, education, government health expenditures, and a time trend reflecting general technological progress. We then used the regional factors provided by Cohen et al. (2004) and by Ostro (2004) to convert PM$_{10}$ concentrations to PM$_{2.5}$ concentrations in different regions.

Figure 6.9 presents current estimates and future forecasts of population-weighted annual average PM$_{2.5}$ concentrations from our base case scenario. Unlike access to improved water and sanitation and the use of solid fuels, the greatest current risk exposure is not in sub-Saharan Africa, but rather in South Asia, Middle East and North Africa, and East Asia and Pacific. By the end of our time horizon, however, the levels in these four regions are similar, as the latter see more rapid declines. Nonetheless, they remain significantly above the levels in Europe and Central Asia, Latin America and the Caribbean, and the high-income countries.

Urban outdoor air pollution: Health effects under alternative scenarios

Again, we explore three scenarios in order to understand the effect of outdoor air pollution on future health. These include our base case scenario, a fast improvement scenario, and a slow improvement scenario. In the latter cases, the concentrations of PM$_{2.5}$ are gradually adjusted such that they are, respectively, one standard error below or above the base case projections (see Figure 6.9) by the year 2030 and remain one standard error below or above the base case projections for the remainder of the period. The standard errors were taken from estimated cross-sectional relationships listed in the previous section, following the logic laid out in Box 5.1.20

As noted in our exploration of indoor air pollution, the size of the adult (particularly the elderly) population is projected to grow significantly between now and mid-century. Furthermore, as we have defined cardiopulmonary diseases to include respiratory infections, cardiovascular diseases, and chronic respiratory diseases, the number of persons affected is quite large. In our base case, the global mortality among adults over 30 from cardiopulmonary diseases rises from over 12 million in 2005 to over 20 million in 2030 and over 33 million in 2060. Only a small share of these are attributable to outdoor air pollution (see again Table 6.1), but given this large base, the impacts of different trajectories can be significant. Figure 6.10 shows the difference between slow and fast improvements in urban air quality, rising to a peak of over 2.6 million deaths around 2030 and still hovering close to 2 million deaths in 2060.

The Organisation for Economic Co-operation and Development has also projected changes in the health impacts of urban air pollution between the years 2000 and 2030 (OECD 2008). The OECD projections were based on methods described in Cohen et al. (2004) and Ostro (2004) to include the effects not only...
of particulate matter but also ground-level ozone (Bakkes and Bosch 2008). As with WHO’s estimates of the current burden of disease, these projections were only for urban areas with populations greater than 100,000 as of the year 2000. Table 6.2 presents the results from OECD’s baseline scenario, which assumed continuation of current trends in such factors as pollutant emissions. If we assume that the PAF for cardiopulmonary diseases remains around 5 percent (see Table 6.1), then just over 1 million of the 20 million deaths in 2030 due to cardiopulmonary diseases in the IFs base case can be attributed to outdoor air pollution in that year. This is significantly lower than the OECD’s estimate of 2.8 million deaths, reflecting a more rapid reduction in urban air pollution in the IFs base case.

We note other items of interest in the OECD’s analysis. With respect to exposures to particulate matter, acute respiratory infections for children under five are projected to decline, but lung cancers and cardiopulmonary diseases in adults are projected to increase significantly. Continued increases in particulate emissions combined with continued population growth and urbanization in developing countries primarily drive these changes. The results for ground-level ozone are, if anything, more striking, with projected mortality increasing sixfold and the total burden of disease tenfold. These projected increases are more evenly spread across the globe as less improvement is forecast in ground-level ozone concentrations in developed countries than for particulate matter concentrations.

### Climate change

Climate change epitomizes the third phase of the environmental risk transition presented by Smith and Ezzati (2005). Both climate change and its health impacts are extremely complex phenomena: the drivers of a changing climate have accelerated as countries develop; climate change operates on a global scale; there is a

#### Table 6.2 Global projections of health impacts of urban air pollution in the baseline scenario of the OECD environmental outlook to 2030

<table>
<thead>
<tr>
<th></th>
<th>Deaths 2000</th>
<th>Deaths 2030</th>
<th>DALYs 2000</th>
<th>DALYs 2030</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Particulate matter</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute respiratory infections, children under five</td>
<td>33,879</td>
<td>24,548</td>
<td>1,202,201</td>
<td>860,839</td>
</tr>
<tr>
<td>Lung cancer, adults over 30</td>
<td>70,432</td>
<td>312,593</td>
<td>656,814</td>
<td>2,672,792</td>
</tr>
<tr>
<td>Cardiopulmonary disease, adults over 30</td>
<td>853,963</td>
<td>2,779,769</td>
<td>7,714,354</td>
<td>21,829,460</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>958,273</td>
<td>3,116,910</td>
<td>9,573,369</td>
<td>25,363,090</td>
</tr>
<tr>
<td><strong>Ground-level ozone</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All diseases, adults over 30</td>
<td>40,292</td>
<td>252,313</td>
<td>339,093</td>
<td>3,349,122</td>
</tr>
</tbody>
</table>

*Note: Projections are only for urban areas with populations greater than 100,000 in 2000. Source: Compiled from data in OECD (2008).*

---

Our base case forecast is for decline in particulate matter in all regions; even so outdoor air pollution contributes significantly to adult cardiopulmonary deaths.
lengthy time delay between the drivers of the risk and the subsequent health effects; the health impacts are varied and reflect complex pathways; there is a significant potential for low-probability, high-consequence events.

The potential health impacts of climate change

Although the potential impacts of climate change on human health received attention almost from the start of the debate on climate change (Weihe and University Hospital 1979), the first comprehensive review of them did not appear until 1996 (McMichael et al. 1996). Since then they have received increasing attention as both the evidence of a changing climate and the understanding of the links between climate change and human health have become more established. Climate change has been characterized as a “silent crisis” that is already affecting global health today (Global Humanitarian Forum 2009), as the “biggest global health threat of the 21st century” (Costello et al. 2009), and, due to the disparity between those who have contributed most to the risk and those who will suffer the most from its effects, as a “growing ethical crisis” (Patz et al. 2007).

We and other analysts face many challenges in our efforts to identify and quantify the present health effects of a changing climate, let alone to understand and forecast future effects. The challenges to identifying and quantifying present effects include an incomplete understanding of many disease mechanisms and a lack of reliable data, particularly from developing countries. Together, the challenges pose problems for conceptualizing models, applying them to multiple regions, validating the results, and addressing uncertainty (Ebi 2008; Ebi and Kovats 2007; Martens, Rotmans, and Rothman 2002; Tamerius et al. 2007). In any attempt to forecast future impacts, to these challenges we can add the uncertainties surrounding our ability to project changes in the climate at the appropriate spatial and temporal scales, as well as the demographic, social, technological, economic, and other environmental responses that together will determine the ultimate health effects of a changing climate.

Figure 6.11, modified from McMichael et al. (2004: 1549), illustrates the range and complexity of the pathways by which climate change may affect human health, as well as the significance of moderating influences and adaptation measures. We show health outcomes in a final box in the figure, organized by the three major disease cause-groups used by both

**Figure 6.11 Climate change and health: Impacts and pathways**

- **Global climate change**
  - Impact on stratospheric ozone
    - UV radiation
  - Regional weather changes
    - Heatwaves
    - Extreme weather
    - Temperature
    - Precipitation

- **Moderating influences and adaptation measures**

- **Intermediate effects**
  - Air quality
  - Water availability
  - Distribution of disease vectors
  - Agricultural production
  - Pollen production
  - Social and economic disruption

- **Health impacts**
  - Communicable diseases
    - Malaria
    - Dengue fever
    - Other vector-borne diseases
    - Protein-energy malnutrition
    - Diarrheal diseases
  - Noncommunicable diseases
    - Cardiovascular diseases
    - Respiratory diseases
  - Injuries
    - Drownings and other unintentional injuries
    - Intentional injuries related to conflict

Source: Authors; modified from McMichael et al. (2004: 1549).
Both climate change and its health impacts are extremely complex phenomena, presenting forecasters with many challenges.

Currently most of the health effects of climate change are related to agricultural yields and subsequent impacts on food availability and undernutrition. 

WHO and IFs (as well as others). Alternatively, Ebi (2008: 1) grouped impacts into three categories based on their relationship to the changing climate: impacts relatively directly related to climate variability; impacts resulting from environmental changes that occur in response to climate variability and change; and impacts resulting from consequences of climate-induced economic dislocation and environmental decline (e.g., undernutrition due to prolonged drought).

Researchers have focused primarily on heat and cold stress and weather-related disasters in their exploration of the direct health effects of a changing climate. Kovats and Hajat (2008) and Gosling et al. (2009) reviewed much of the recent literature on heat and cold stress, human health, and climate change; much of this work received additional attention following recent heat waves, most notably that in 2003 in Europe. Among the issues the reviews address are the precise nature of the relationships between heat and cold stress and human health, the expected net effect of heat and cold stress in a future climate, the interaction between heat stress and air pollution, human adaptability, and mortality displacement (the idea that persons who die from heat and cold stress would have died shortly afterward irrespective of the extreme temperatures). The population group of most concern is the elderly and, to a somewhat lesser extent, adults with chronic diseases and children. In addition, studies focus on urban areas, both because of the heat island effect whereby cities tend to be warmer than their surroundings and because of the trend toward continued urbanization in much of the world. The health outcomes occur mostly in the form of cardiovascular and respiratory diseases. In general, both reviews reached the following conclusions: increasing mortality from additional heat stress is likely to far exceed decreasing mortality from reduced cold stress at a global level; there is very little evidence of mortality displacement in extreme heat waves; and although there is some evidence of synergistic effects between temperature and air pollution, the effect of temperature alone remains significant even when the effects of air pollution are controlled. Finally, both reviews pointed to the role that public and private health measures, including providing access to air-conditioned and heated facilities, can play in mediating the effects of warmer and colder temperatures.

Some weather-related disasters—for example, coastal and inland floods, landslides, and windstorms—are clearly related to climate. In recent years, these have been increasing at a rate that significantly exceeds the changing incidence of other natural disasters (Global Humanitarian Forum 2009). Campbell-Lendrum and Woodruff (2007) pointed to the potential for increasingly variable weather, including more intense storms and sea level rise, to increase future mortality. The Global Humanitarian Forum (2009) estimated that presently 40 percent of the impact of weather-related disasters can be attributed to climate change, and that this will rise to 50 percent by 2030.

Vector-borne and water-borne diseases stand out in terms of impacts resulting from environmental changes that occur in response to climate variability and change. Insect, rodent, and other intermediate hosts (generally referred to as vectors) carry malaria and many other diseases. Tamerius et al. (2007) noted that climate is a key determinant of the spatial and temporal variations of both the vectors and the underlying disease-causing agents (viruses, bacteria, protozoa, and helminths), as well as their behavior and potency (e.g., biting rates and incubation periods). At the same time, climate is only one factor in the complex relationship between these organisms and the natural environment. Human actions, ranging from the use of bed nets to deliberate modifications of the landscape (in order to reduce vector populations) to traditional and modern medications, further mediate the ultimate effects on human health. Most researchers agree that climate change will have significant effects on vector-borne diseases, at least in terms of their spatial and temporal distribution; however, debate remains around the net effect of climate change on mortality and morbidity from these diseases.

Climate factors also strongly influence the life cycles of, and human exposure to, many pathogens transmitted through contaminated food and water (Campbell-Lendrum and Woodruff 2007; Lipp, Huq, and Colwell 2002; Lloyd, Kovats, and Armstrong...
The resulting diseases include cryptosporidiosis and cholera, as well as diarrheal diseases more generally. Although a range of climate variables, including atmospheric temperature, sea surface temperature, sea surface height, precipitation, and floods are implicated in the occurrence of the diseases associated with these pathogens, most studies to date have focused on the impacts of temperature.

Among the impacts resulting from consequences of climate-induced economic dislocation and environmental decline, food production and resulting levels of malnutrition have received significant attention from researchers and the general public. Climate change is expected to have adverse impacts on food security (Schmidhuber and Tubiello 2007). The Intergovernmental Panel on Climate Change has stated that, whereas moderate warming may increase crop and pasture yields in mid- and high-latitude regions, even slight warming will lead to decreases at lower latitudes, particularly in drier regions (Easterling et al. 2007). The increasing level of atmospheric carbon dioxide, which drives much of the climate change, may ameliorate these declines somewhat, but the most recent evidence indicates that its ameliorating effect on crops is likely to be much less than previously estimated (Leakey et al. 2009; Long et al. 2006). Farmers will certainly try to adapt to the changing conditions, but adaptation is likely to be limited by the availability of key resources, such as arable land and water, which will also face stresses from a changing climate (Cline 2007; Easterling et al. 2007). Factors other than climate obviously affect global food production, and in turn, factors beyond total food availability, including food prices, incomes, and other health conditions, affect levels of undernutrition. Still, the net effect of climate change on global food production, even if it is only slower growth rather than absolute decline, will make it more difficult for individual countries and the world as a whole to address the issue of undernutrition.

In Table 6.1, we noted that WHO has estimated that approximately 141,000 deaths and 5.4 million DALYs were attributable to climate change in the year 2004. While the results represented only a small share of the total burden of disease, it is important to note that they reflected the effects of climate change over a relatively short period, as the baseline climate year used in the analysis was 1990 (McMichael et al. 2004). In addition, WHO considered only a subset of the possible health effects of climate change (Kovats, Campbell-Lendrum, and Matthies 2005; Patz et al. 2005; Zhang, Bi, and Hiller 2007).

Table 6.3 breaks these estimates down by disease, region, and age. The message that these numbers convey is that, at the present time, children in poor countries bear almost the entire health burden from global climate change. Furthermore, the dominant impacted diseases are all affected by undernutrition, indicating that the majority of the current effect of climate change is due to its impact on agricultural yields and the subsequent availability of food.

Table 6.3 Burden of disease (deaths and DALYs) due to global climate change by disease category, region, and age (2004)

<table>
<thead>
<tr>
<th>By Disease and Injury Category</th>
<th>Deaths</th>
<th>DALYs</th>
<th>By Age</th>
<th>Deaths</th>
<th>DALYs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diarrheal diseases</td>
<td>45.9%</td>
<td>40.2%</td>
<td>0-4</td>
<td>85.2%</td>
<td>87.3%</td>
</tr>
<tr>
<td>Malaria</td>
<td>19.1%</td>
<td>19.3%</td>
<td>5-14</td>
<td>4.4%</td>
<td>5.6%</td>
</tr>
<tr>
<td>Lower respiratory infections</td>
<td>11.8%</td>
<td>11.0%</td>
<td>15-29</td>
<td>1.8%</td>
<td>3.0%</td>
</tr>
<tr>
<td>Measles</td>
<td>4.4%</td>
<td>4.0%</td>
<td>30-44</td>
<td>2.0%</td>
<td>2.1%</td>
</tr>
<tr>
<td>Pertussis</td>
<td>3.9%</td>
<td>3.9%</td>
<td>45-59</td>
<td>2.0%</td>
<td>1.2%</td>
</tr>
<tr>
<td>Protein-energy malnutrition</td>
<td>3.5%</td>
<td>8.8%</td>
<td>60-69</td>
<td>1.5%</td>
<td>0.5%</td>
</tr>
<tr>
<td>30 others</td>
<td>11.3%</td>
<td>12.8%</td>
<td>70-79</td>
<td>1.5%</td>
<td>0.2%</td>
</tr>
<tr>
<td>80+</td>
<td>1.6%</td>
<td>0.1%</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>By Region</th>
<th>Deaths</th>
<th>DALYs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>East Asia and Pacific</td>
<td>4.9%</td>
<td>5.4%</td>
<td></td>
</tr>
<tr>
<td>Europe and Central Asia</td>
<td>0.5%</td>
<td>0.5%</td>
<td></td>
</tr>
<tr>
<td>Latin America and Caribbean</td>
<td>1.2%</td>
<td>1.5%</td>
<td></td>
</tr>
<tr>
<td>Middle East and North Africa</td>
<td>3.0%</td>
<td>3.2%</td>
<td></td>
</tr>
<tr>
<td>South Asia</td>
<td>46.4%</td>
<td>48.0%</td>
<td></td>
</tr>
<tr>
<td>Sub-Saharan Africa</td>
<td>43.8%</td>
<td>41.2%</td>
<td></td>
</tr>
<tr>
<td>High-income countries</td>
<td>0.1%</td>
<td>0.2%</td>
<td></td>
</tr>
</tbody>
</table>

Source: Computed from WHO data on burden of disease by risk factor, disease, or injury cause by age and sex at the regional level (see http://www.who.int/healthinfo/global_burden_disease/risk_factors/en/index.html).
represent biological and social processes, and statistical approaches, which use statistical relationships between climatic and social variables and specific impacts. In either case, they also require projections of future climate and social variables. To date, most studies have focused on a single impact. Even those that have looked at multiple impacts have generally not done so in an integrated fashion beyond assuming a common climate scenario (McMichael and Campbell-Lendrum et al. 2003; Global Humanitarian Forum 2009). Ebi (2008) laid out an agenda for the development of models to quantitatively estimate the potential health effects of climate change in an integrated fashion, noting the challenges faced, the limited progress to date, and the opportunities for advancement in this area. The TARGETS (Rotmans and de Vries 1997) and MIASMA (Martens 1998) models were early attempts to do so. Hilderink and Lucas (2008) and Pitcher, Ebi, and Brenkert (2008) present more recent efforts in this area.

For this volume, we have limited our quantitative analysis to the potential effects of a changing climate on crop production, with ensuing impacts on food availability and undernutrition. As with the more general analysis of undernutrition described in Chapter 4, the health outcomes of interest are mortality and morbidity from all communicable diseases for children under five. Thus, we examine only one of the potential effects of climate change, but it is the one that many estimate to be having the greatest effect currently and also expect to have the largest effect in the future (Global Humanitarian Forum 2009).

Using the IFs model, we analyze these impacts in the context of a fully integrated social, economic, and environmental structure. Our analysis begins with our base case forecast of the atmospheric concentration of carbon dioxide, driven by land use changes and emissions of carbon dioxide from fossil fuel use. We compute global temperature change from the atmospheric carbon concentration. From global temperature change we derive national-level changes in temperature and precipitation, using data compiled for the MAGICC/SCENGEN climate model (Wigley 2008). Building on detailed work by Cline (2007) and Rosenzweig and Iglesias (2006), we estimate the impact of national-level changes in temperature and precipitation and changes in the atmospheric concentration of carbon dioxide on crop yields. And we estimate levels of childhood undernutrition in a relationship with calorie availability that is responsive to crop production.28

In order to understand the effect of a changing climate, we consider three scenarios: (1) a “no climate change impact” scenario; (2) the base case, which includes the full effects of changing climate on crop production (the effects of change in temperature and precipitation as well as carbon fertilization); and (3) a “no CO2 fertilization” case in which we shut off only the fertilization effect. We include the latter case because significant debate continues over the level of amelioration CO2 provides for the otherwise negative effect of climate change on crop production. And in fact, most studies looking at the effect of climate change on agriculture present results with and without a CO2 fertilization effect.

The changes in climate are basically identical in all three scenarios as the feedbacks from differences in crop production back to the drivers of climate change are fairly minimal. The atmospheric concentration of CO2 increases from approximately 380 parts per million (ppm) to 450 ppm in 2030 and 550 ppm in 2060. This results in increases in global average surface temperature of 0.75°C and 1.6°C from 2005 to 2030 and 2060, respectively, forecasts that are within the range of most projections (IPCC 2007). At regional levels, by 2060 the temperature increases range from 1.9°C to 2.6°C29 and changes in annual precipitation range from a decrease of 17.9 percent to an increase of 12.9 percent.

Before we look at the global impact of climate change, it is useful to see how the dynamics of the IFs system modify the initial effect. Figure 6.12 traces out how this occurs, using Nigeria in 2060 as an example. Starting at the left side of this figure, the bars labeled “Climate yield effect” show that the changing climate implies that yields per hectare would be more than 9 percent lower than would otherwise be the case; the decline would actually be closer to 15 percent if it were not for the modeled direct fertilization effect of CO2. Because of other adjustments
in the model (e.g., in capital investments in agriculture), the actual difference in “Yield per hectare” between the scenarios is slightly less. Total production falls less than the yield per hectare, implying an expansion in the area devoted to crops, driven in part by the rise in crop prices. There is more than a 25 percent increase in crop prices in the scenario where no CO₂ fertilization effect is included vis-à-vis an approximately 5 percent increase where it is. Declines in available calories per capita in both cases result in an increased share of undernourished children (approximately 2 and nearly 8 percent in the two scenarios). Finally, there is a more than 1 percent increase in mortality from communicable diseases other than HIV/AIDS for children under five in the scenario with CO₂ fertilization and an increase of over 3 percent when there is no CO₂ fertilization effect. In either case, this is less than might have been expected from the direct effect of climate change on crop yield.

In examining individual countries, we find also that there can be dramatic spikes in mortality in specific years. These occur in countries with very low food reserves, where the changing climate can lead to food shortages when they would not occur otherwise. While this result makes sense, and highlights one of the benefits of using an integrated model, we do not wish to claim that we are able to predict such specific events. Therefore, in Figure 6.13 we present a 10-year moving average of the projected effect of climate change on mortality from communicable diseases other than HIV/AIDS among children under five over time. This allows us to see the more general pattern of the effect. The measures are the forecast differences between the “no climate change” scenario and the scenario without carbon fertilization.

The reader, as were we, at first may be struck by the fairly small size of the effect, peaking at just over 70,000 additional deaths of children under five years of age around 2050. Upon reflection, though, this should not be surprising. Given the significant decline in undernutrition and the total number of children dying from communicable diseases other than HIV/AIDS in our base case scenario, even significant percentage changes due to climate result in fairly small changes in

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**Figure 6.12 Climate change impacts via effects on crop yields in Nigeria: Base case and “no CO₂ fertilization” scenario compared to “no climate change impact” scenario (percentage differences in 2060)**

Note: The “no climate change impact” scenario (not shown) is the reference case for this figure; the IFs base case has full climate change impact (from temperature, precipitation, and CO₂ fertilization effects); the more negative “no CO₂ fertilization” scenario has the often-negative impacts of climate change (from temperature and precipitation change) but not the generally positive impact of CO₂ fertilization.

Source: IFs Version 6.32.

**Figure 6.13 Difference in forecasts of deaths (thousands) of children under five from communicable diseases other than HIV/AIDS due to climate change impacts on crop yields**

Source: IFs Version 6.32.

Notes: Figure shows a 10-year moving average of deaths. See text for explanation.
absolute numbers. We explore this further in Box 6.1. Over time, we see a steady rise until around 2030, followed by a small decline and then a further increase until around 2050, when the number of deaths begins to decline to just over 40,000 in 2060. Recalling that the total number of children dying from communicable diseases other than HIV/AIDS is much smaller by this later period (nearly 6 million in 2020 versus fewer than 1.5 million in 2060 in the base case), the additional deaths related to climate change in 2060 represent a significantly larger share. Finally, it is not surprising that the largest effects occur in sub-Saharan Africa and South Asia, which have the largest absolute numbers of deaths related to these diseases.

**Comparing and Combining Analyses of Proximate Risk Factors**

This chapter and the previous one have considered the health impacts of eight different risk factors and have explored the implications of alternative scenarios with respect to each one. However, the discussion of individual risk factors leaves two related questions unanswered.

First, how do interventions across risk factors compare in their possible effects on future health? With respect to each factor other than climate change, we structured somewhat more favorable and less favorable scenarios around the base case, scenarios that take into account the variation in risk factor levels that countries have recently demonstrated relative to a cross-sectional relationship with GDP per capita. In each instance, we have phased in changes to risk factors over aggressive (but hopefully reasonable) periods of 25 years. For climate change, the more optimistic scenario assumed no impact on crop yields from a changing climate, and the more pessimistic scenario included the effects of a changing climate but excluded the potentially positive direct effects from carbon dioxide fertilization. This general uniformity of approach allows us to consider comparative implications for morbidity and mortality. However, at this point we caution against using the analysis across proximate risks as a strong basis for policy analysis, particularly since we have not considered the costs of interventions necessary to shift paths.

The second question addresses the possible implications of combining risk factor interventions. Methodologically, such combination raises many questions, as we identify below. Nonetheless, most societies target multiple proximate risks in order to improve health, making the question an important one. Moreover, combined risk analysis will set the stage for a broader scenario analysis to which Chapter 7 and especially Chapter 8 will return.

**Comparative risk analysis**

Table 6.4 presents the total difference between the optimistic and pessimistic (or favorable and unfavorable) scenarios in our forecasts of global deaths, years of life lost (YLLs), and disability-adjusted life years for the period 2005–2060 for each risk factor discussed in this and the previous chapter. These values show that actions directed at the individual proximate risk factors have the potential to prevent tens of millions of global deaths and several billion
YLLs and DALYs. Because of the different age groups that specific risks most affect, and the different disability weights for specific diseases, the relative impact of some risk factors changes somewhat across the different measures.

Again, these results, while indicative, should not necessarily be seen as a basis on which to set priorities. For example, although we attempted to tie the more optimistic scenarios to what appear to be possible changes in the proximate drivers (based on cross-sectional analysis), our efforts were somewhat crude. And we used a blunt instrument by applying uniform changes to all countries. Further, we made no attempt to consider the costs of such changes in the course of proximate drivers. Finally, to the extent that investments to reduce exposure to these proximate risks may affect other determinants of health, such as growth in average income or education levels, there will be secondary effects (potentially both positive and negative) for society to consider. Despite all these caveats, this analysis does give us some general sense of the potential that attention to different risk factors offers.

**Combined risk analysis: WHO and World Bank approach**

Recent major reports from the World Bank’s Disease Control Priorities Project (Lopez et al. 2006a) and WHO (Ezzati et al. 2004a; WHO 2009a) have gone beyond the analysis and comparison of individual risks to consider also the implications of simultaneously reducing multiple risk factors to theoretical minimum levels. Their theoretical minimums assumed zero exposure when that was theoretically possible (for example, with respect to smoking); when zero exposure was not theoretically possible (e.g., body mass index), the lowest levels observed in some population were used as the theoretical minimums.

Because many diseases involve more than one risk factor, and the reduction or elimination of any one factor can prevent the disease in significant numbers of (but seldom all) people, the combined effect of hypothetically eliminating many risk factors simultaneously is generally less than the calculated sum of the effects of eliminating individual risks. For example, based on an analysis that assumed immediate reduction of risks to theoretical minimum levels, WHO estimated that

- of all infectious and parasitic child deaths . . . , 35% can be attributed to underweight; 26% to unsafe water, hygiene and sanitation; and 15% to smoke from indoor use of solid fuels. The joint effect of all three of these risk factors is, however, 46%.
- Similarly, 45% of cardiovascular deaths among those older than 30 years can be attributed to raised blood pressure, 16% to raised cholesterol and 13% to raised blood glucose, yet the estimated combined effect of these three risks is about 48% of cardiovascular diseases.

In WHO’s most recent report on health risks, researchers attributed 25 percent of deaths globally to the five leading risk factors (in order of impact: high blood pressure; tobacco use; high blood glucose; physical inactivity; and overweight and obesity), 33 percent to the ten leading factors (adding, again in order of impact: high cholesterol; unsafe sex; alcohol use; childhood underweight; and indoor smoke from solid fuels), and 44 percent to all 24 proximate risk factors in its analysis set (WHO 2009a: 30, Table 7). Looking across all regions of the world and all risk factors in the CRA set, they concluded

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Cumulative deaths (millions)</th>
<th>Cumulative YLLs (millions)</th>
<th>Cumulative DALYs (millions)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undernutrition</td>
<td>70</td>
<td>2,254</td>
<td>2,855</td>
</tr>
<tr>
<td>Obesity</td>
<td>68</td>
<td>1,054</td>
<td>1,064</td>
</tr>
<tr>
<td>Smoking</td>
<td>31</td>
<td>258</td>
<td>272</td>
</tr>
<tr>
<td>Road traffic accidents</td>
<td>107</td>
<td>2,712</td>
<td>3,248</td>
</tr>
<tr>
<td>Unsafe WSH</td>
<td>53</td>
<td>1,696</td>
<td>2,103</td>
</tr>
<tr>
<td>Indoor air pollution</td>
<td>22</td>
<td>471</td>
<td>649</td>
</tr>
<tr>
<td>Outdoor air pollution</td>
<td>87</td>
<td>1,033</td>
<td>1,167</td>
</tr>
<tr>
<td>Climate change</td>
<td>1</td>
<td>67</td>
<td>123</td>
</tr>
</tbody>
</table>

*Source: IFs Version 6.32*
Had these 24 risks not existed, life expectancy would have been on average almost a decade longer in 2004 for the entire global population . . . . Low and middle income countries have much more to gain than the richest countries: for example, life expectancy would have grown by nearly 13 years in the African Region, but by less than 6 years in the high-income countries. The five leading risks alone shortened life expectancy by about 9 years in Africa in 2004. (WHO 2009a: 29 and 31)

Combined risk analysis: The IFs approach
We similarly conducted an analysis of the combined impacts of simultaneous rapid reduction of multiple risk factors in IFs in comparison with the IFs base case. To do this, we created a model intervention in which we reduced the eight risk factors currently covered in IFs to their theoretical minimum levels between 2005 and 2010.10 It is important to emphasize that such analysis goes well beyond the favorable risk factor interventions in Chapters 5 and 6. For instance, it requires the complete elimination of undernutrition, smoking, unsafe water and sanitation, and the indoor use of solid fuels in just five years. Neither WHO nor we assume that such elimination of risk factors is possible; the purpose of such analysis is to explore the contemporary health impact of individual and/or multiple risk factors.

We should quickly caution the reader that in addition to its focus on a limited set of risk factors, our combined analysis of multiple risks has very significant weaknesses, and two in particular. First, our formulations do not account for all of the overlapping or interacting impacts of risk reductions (we do account for some of the largest ones, such as that between malnutrition and water and sanitation); not doing so leads to summing of some of the impacts and biases our joint risk analysis toward overestimation of the impacts of combined reductions in risks. Second, and less important, for simplicity and sharpness of analysis, our formulations link risk factors only to age categories of populations that are most at risk, such as linking undernutrition only to children under five years of age and cardiovascular diseases only to adults over 30 years of age; this slightly biases both individual and joint risk analysis toward underestimation of the impacts of reduction of risks. The two problems, albeit opposite in direction, obviously do not cancel each other out.

 Nonetheless, Table 6.5 shows the joint risk analysis of the eight proximate risks currently represented in IFs and suggests some interesting insights. Overall, the near-immediate reduction in deaths, a total of 19 percent in 2010, is less than the 25 percent that WHO attributes to the five leading global factors alone. The most important reason is that we include only two of the top five risk factors in our analysis (tobacco use and overweight and obesity), albeit in conjunction with our other risk factors. Second, as mentioned above, our formulations tie risk reduction only to the age categories at greatest risk (see again Table 2.1). Also highly important is the very long delay within IFs (and the real world) in the impact of smoking on cardiovascular diseases. That is, even if the world were able immediately to reduce the risk factor to zero, reductions in deaths would appear only very slowly. By 2020, the effects of having eliminated all smoking by 2010 are very apparent in our modeled reduction in noncommunicable diseases. In contrast, the WHO analysis introduced the assumption that the entire population immediately consisted of lifelong nonsmokers.

Another striking result apparent in Table 6.5 is that the impact of the movement of risks to theoretical minimums tends to erode over time, especially with respect to deaths from communicable diseases. In our base case,

| Table 6.5 Global reduction in deaths between 2010 and 2060 by disease group with near-immediate shift to theoretical minimum risk levels |
|--------------------------------------------------|---------|---------|---------|---------|---------|---------|
| 2010 | 2040 | 2060 | Cumulative reduction |
| Percent | Percent | Percent | Percent | Million |
| Communicable diseases | 35.6 | 19.5 | 7.9 | 23.4 | 131.6 |
| Noncommunicable diseases | 11.2 | 13.0 | 3.1 | 7.6 | 242.0 |
| Injuries | 24.8 | 28.0 | 20.6 | 25.1 | 103.5 |
| Total | 19.1 | 15.2 | 5.1 | 11.4 | 477.1 |

Note: Percentages are relative to the base case.
Source: IFs Version 6.32 minimum risk scenario
communicable diseases already decrease rapidly over time, so the incremental leverage of near-immediate reductions of their proximate drivers to theoretical minimums relative to the base case erodes over time.

The very sizable impact of risk reduction in injuries is still another interesting result of the analysis. In IFs this stems largely from hypothetical movement to zero traffic deaths in the near-immediate reduction scenario. The modeled cumulative death reduction in this category is significant, especially relative to injuries as a cause of death in 2005, and suggests a considerable and growing potential for leverage from interventions to enhance road traffic safety. One implication of the analysis is that a world that planned ahead to reduce deaths would begin now to put additional attention not only on smoking (with its lags in health payoffs) but also on road traffic accidents with their growing rather than shrinking base. To date the CRA framework (see again Table 2.1) has not included road traffic accidents in its analyses; given the potential leverage of interventions it almost certainly should.

Another important factor is that in our analysis, which incorporates forward linkages (as we will discuss further in Chapter 7), reduced deaths in the near term become additional or delayed deaths in the longer term, resulting not only in an aging population that will die of other causes but also in a population that is considerably larger overall (more than 400 million larger in 2060).

To put combined risk analysis into the broader context of this volume, we emphasize dynamic analysis throughout. Across Chapters 5 and 6 we have stressed that proximate drivers are not likely to move rapidly away from the trajectories of the base case forecast, either in negative directions from neglect and bad policy or in positive directions with concerted positive interventions and behavioral changes. Nor are societies likely to succeed in moving risk factors to their theoretical minimums; we chose instead to use one standard deviation as a more likely representation of aggressive action.

The overall orientation of our analysis, therefore, is not static consideration of counterfactuals but rather dynamic analysis related to possible interventions or alternative futures. If we turn from the near-immediate movement of proximate drivers to theoretical minimum levels (as represented in the discussion of CRA results and the IFs results of Table 6.5) to a combination of aggressive but phased-in human action on these eight major drivers as discussed throughout Chapters 5 and 6, we might avert approximately 203 million deaths over the horizon through 2060 and avoid about 4.2 billion discounted years of life loss. This is somewhat less than half of the total with more static analysis and is, we would argue, a more policy-relevant figure.

Conclusion
This chapter has illustrated that analyses cannot ignore the environment in efforts to forecast and, more importantly, to influence future levels of population health. The reduction in infectious and communicable diseases, such as diarrheal and respiratory infections seen in the early stages of the epidemiologic transition, is very much a function of efforts to reduce traditional environmental risk factors such as the lack of access to clean water and sanitation and indoor air pollution. The extent to which currently developing countries will complete this phase of the transition will depend largely on how they are able to address these risk factors. At the same time, continued development has introduced other environmental risk factors, including community risk factors, such as outdoor air pollution and the introduction of man-made chemicals into the work place and wider environment. Further, global risk factors, such as global climate change and stratospheric ozone depletion, are now beginning to have a clear and growing impact on health. Thus, we need to see environmental policy as a key element of health policy.

Even as concern is great and growing in many quarters, tremendous uncertainty remains around the myriad potential health effects of environmental risk factors, particularly in the case of the more modern risk factors. This makes estimating and projecting the quantitative level of these effects both a necessary and a challenging task. The challenge is perhaps most starkly reflected in the limited set of analyses presented in this chapter. We excluded many of the most significant and
growing concerns about environmental drivers of human health in the future, particularly in developed countries.

Regardless of uncertainties and the absence of much-needed analysis, the evidence strongly indicates that the environment has played, and will continue to play, a significant role in human health. Moreover, the exposure to environmental risks differs substantially across countries at similar levels of development as defined by traditional measures such as average income. Because health outcomes reflect these differences, there appears to be very considerable potential for improved health from policies that reduce environmental risks.

This chapter also returned to the broader analysis of proximate drivers, comparing the health implications of the environmental risks analyzed in this chapter with those of the risks that Chapter 5 explored, and considering the implications of combined risk reduction. Although comparative risk analysis suffers from the great differences in various risk factors’ character and susceptibility to reduction, we have attempted to create as much comparability as we could. We find that all of the eight risks are important, but that there are clear variations in the impact of alternative assumptions. For instance, the morbidity and mortality associated with climate change over our horizon is perhaps less great than many would expect (which is not to downplay its growing importance over time). And we find that risks and opportunities around traffic accident deaths are probably more significant than many would anticipate. In fact, across our horizon they exceed those associated with undernutrition, an appropriately primary focus of much current policy. The reality, however, is that income growth is likely to reduce the base impact of undernutrition significantly by 2050, while that same growth will raise the impact of road traffic accidents, especially in lower- and middle-income countries.

Finally, we considered the proximate risks in combination. In spite of the fact that multiple risks are often complicit in any death or disability, and that reduction of multiple risks will therefore save fewer lives than the sum of reductions across them individually, we found that the combined impact of multiple risk reduction is truly huge. At the same time, however, we returned to the importance of undertaking dynamic analysis of health futures for such combined analysis. Such analysis should also ideally take into account the forward linkages of health interventions and the broader contexts of change and uncertainty in which they are made. Those are the topics of Chapters 7 and 8.

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1 The range of international efforts reflects significant increase in international attention to questions of health and the environment in recent years. The Millennium Ecosystem Assessment and Fourth Assessment Report of the Intergovernmental Panel on Climate Change (Confalonieri et al. 2007; Corvalán et al. 2005; MA and WHO 2005) paid particular attention to the potential health effects of environmental change. More recently, researchers and policymakers have launched a number of international efforts that focus on the relationships between the environment and human health and the means by which to address problems in this area. Among these are the Earth System Science Partnership’s joint project on Global Environmental Change and Human Health (Confalonieri and McMahan 2007), the International Council for Science’s Planning Group on Health and Wellbeing in the Urban Environment (ICSU 2007), and the Health and Environment Linkages Initiative of the World Health Organization and the United Nations Environment Programme (WHO and UNEP 2008).

Because of children’s particular vulnerability, there are also specific efforts focusing on children’s health and the environment (CEC 2006; Gordon, Mackay, and Rehfuss 2004). Finally, in an extension of its work on the Global Burden of Disease and building on earlier efforts and the Comparative Risk Assessment, WHO has established a program on quantifying the environmental burden of disease (Prüss-Ustün and Corvalán 2006; Smith, Corvalán, and Kjellström 1999).
2 Huynen (2008: 84) defines the contextual level as consisting of “the macro-level conditions that form the context in which the distal and proximal factors operate and develop.” With respect to the environment, she refers to general ecological settings, including climate.

3 Reports in this series can be found at http://www.who.int/quantifying_ehimpacts/national/en/index.html.

4 For further information on this work, see http://www.who.int/quantifying_ehimpacts/en/.

5 The additional data on their website include estimates at the regional level by risk factor, disease, or injury cause, and age and sex (see http://www.who.int/healthinfo/global_burden_disease/risk_factors/en/index.html), and country profiles of the environmental burden of disease (see http://www.who.int/quantifying_ehimpacts/national/countryprofile/intro/en/index.html). Most of the historical data presented in this chapter are taken directly from, or derived from, these data.

6 Health outcomes and risk factors included are: all diarrheal diseases from lack of access to an improved drinking water source and improved sanitation facilities; acute respiratory infections (children under age five); chronic obstructive pulmonary disease (adults over 30 years); lung cancer (adults over 30 years) from the use of solid fuels in the house; and respiratory infections and diseases, lung cancer, and selected cardiovascular diseases from the exposure to fine suspended particles of less than 10 microns in diameter in cities with more than 100,000 inhabitants and in national capitals.

7 Details of this analysis are provided in the technical documentation of the IFs health model (Hughes et al. 2010) available at www.ifsd.u.edu.

8 Details of this analysis are provided in the IFs health forecasting technical document available at www.ifsd.u.edu.

9 Indoor air pollution was also considered to be a potentially significant risk factor for other health impacts—specifically, asthma, cataracts, perinatal effects, and tuberculosis—for which a lack of sufficient data precluded estimates (Desai, Mehta, and Smith 2004; Smith, Mehta, and Maezusahli-Feuze 2004).

10 From data publishedly available from WHO. The specific spreadsheets used were “Estimated deaths and DALYS attributable to selected environmental risk factors” (http://www.who.int/entity/quantifying_ehimpacts/countryprofiles2004.xls) and “Estimated deaths and DALYS by cause and WHO member state”(http://www.who.int/entity/healthinfo/global_burden_disease/gbdeathdalycountries2004.xls).

11 In 2004, ARI and COPD accounted for 98 percent and 75 percent of global deaths from respiratory infections and respiratory diseases, respectively (calculated from disease and injury regional estimates, available at http://www.who.int/healthinfo/global_burden_disease/estimates_regional/en/index.html).


14 Details of this analysis are provided in the IFs health forecasting technical document available at www.ifsd.u.edu.

15 Details of this analysis are provided in the IFs health forecasting technical document available at www.ifsd.u.edu.

16 Various other studies have provided concentration-response functions for ozone and particulate matter and a range of health outcomes (Bakkes and Bosch 2008; Cohen et al. 2004; Jerrett et al. 2009; Ostro 2004; Pope and Dockery 2006). In addition, the U.S. Environmental Protection Agency has developed an Environmental Benefits Mapping and Analysis Program (http://www.epa.gov/air/bemmap/index.html), which uses many of these relationships.


18 The latter has also been used to estimate future levels of air quality as part of the OECD’s Environmental Outlook to 2030 (OECD 2008).

19 Details of this analysis are provided in the IFs health forecasting technical document available at www.ifsd.u.edu.

20 Details of this analysis are provided in the IFs health forecasting technical document available at www.ifsd.u.edu.

21 Recent summary reviews of climate change and its current and potential effects on human health include: Comrie 2007; Confalonieri et al. 2007; Costello et al. 2009; Haines et al. 2006; Haines and Patz 2004; McMichael, Woodruff, and Hales 2006; Patz et al. 2005; McMichael, Woodruff, and Hales 2006; Patz et al. 2007; and Zhang, Bi, and Hiller 2007. WHO’s Comparative Risk Assessment (McMichael et al. 2004) and Environmental Burden of Disease work (Campbell-Lendrum and Woodruff 2007) identified climate change as a key risk factor. Climate change was a primary focus of the 2008 Annual Review of Public Health, and WHO devoted the 2008 World Health Day to climate change (WHO 2008b). It is a key focus of the Global Humanitarian Forum (2009). Numerous other studies have considered specific health effects, and Confalonieri et al. (2007) reviewed recent studies that provided quantitative projections of climate change impacts on specific health outcomes. These focused on malaria, dengue fever, and other infectious diseases; heat- and cold-related mortality; and health effects related to urban air quality, particularly tropospheric ozone.

22 Among the other diseases are bubonic plague; chikungunya; dengue fever; hantavirus; Lassa fever; leishmaniasis; Lyme disease; lymphatic filariasis; lyssavirus; onchocerciasis (river blindness); schistosomiasis; tick-borne encephalitis; and West Nile virus.

23 See, for example, the recent forum in Ecology (volume 90, issue 4, 2009) stimulated by Lafferty (2009).

24 For example, Calicivirus, Campylobacter, Cryptosporidium, Clostridium, Giardia, Norovirus, Salmonella, Shigella, and Vibrio cholerae (Campbell-Lendrum and Woodruff 2007; Tamerius et al. 2007).

25 Even the discussions on climate-induced conflict and associated mortality—for example, in Dyer (2008) and Campbell et al. (2007)—tend to point back to changes in food production as the key driving force behind the conflict.

26 Other ways in which climate change may influence food production include its effect on pests and pathogens, ultraviolet radiation, ground-level ozone, weather extremes, and water quality, which will impact freshwater and marine fisheries (Brander 2007; Easterling et al. 2007; Gregory et al. 2009; Tubiello et al. 2007).

27 This is an update of the analysis of WHO’s Global Burden of Disease and Comparative Risk Assessment projects (McMichael and Campbell-Lendrum et al. 2003). We were able to trace almost every global estimate, in all studies that we found, back to this work. The Global Humanitarian Forum (2009) was the one exception, and even there most of the estimation methods used were the same. Epstein and Mills (2005) provide an interesting integrative analysis covering a number of diseases, but their projections are primarily qualitative. Confalonieri et al. (2007) list 16 national/regional health impact assessments of climate change published between the third and fourth IPCC assessments, not including a more recent study by the United States (Gamble et al. 2008). Of these, McMichael and Woodruff et al’s (2003) study for Australasia has had, perhaps, the most significant impact. For additional results and results presented using different regional or gender breakdowns from the original WHO study, see Ezzati et al. 2002; Patz et al. 2005; and WHO 2002.

28 Climate change may also affect access to improved water and sanitation, which is also one of the explanatory variables for childhood undernutrition. IFs does not currently try to capture this effect.

29 The reason that the forecasted global temperature change falls below the forecasted range for the regions is that the former also includes temperature changes over the oceans, which, in general, warm more slowly than land surfaces.

30 The assumption of reductions over a five-year period was necessitated by the dynamic nature of our model. Our five-year hypothetical phase-in period is in distinction from the WHO 2009 study (WHO 2009a) in which reductions to theoretical minimums all occurred simultaneously and as if at a single point in time.