Chapter 20

Global climate change

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Summary

Accumulating evidence suggests that the global climate (i.e. conditions measured over 30 years or longer) is now changing as a result of human activities—most importantly, those which cause the release of greenhouse gases from fossil fuels. The most recent report (2001) from the United Nations' Intergovernmental Panel on Climate Change (IPCC) estimates that the global average land and sea surface temperature has increased by 0.6±0.2 °C since the mid-19th century, with most change occurring since 1976. Patterns of precipitation have also changed: arid and semi-arid regions are becoming drier, while other areas, especially mid-to-high latitudes, are becoming wetter. Where precipitation has increased, there has been a disproportionate increase in the frequency of the heaviest precipitation events. Based on a range of alternative development scenarios and model parameterizations, the IPCC concluded that if no specific actions were taken to reduce greenhouse gas emissions, global temperatures would be likely to rise between 1.4 and 5.8 °C from 1990 to 2100. Predictions for precipitation and wind speed were less consistent, but also suggested significant changes.

Risks to human health from climate change would arise through a variety of mechanisms. In this chapter, we have used existing or new models that describe observed relationships between climate variations, either over short time periods or between locations, and a series of health outcomes. These climate–health relationships were linked to alternative projections of climate change, related to unmitigated future emissions of greenhouse gases, and two alternative scenarios for greenhouse gas emissions. Average climate conditions during the period 1961–1990 were used as a baseline, as anthropogenic effects on climate are considered more significant after this period. The resulting models give estimates of the likely future effects of climate change on exposures to thermal extremes and weather disasters (deaths and injuries associated with

floods), the distribution and incidence of malaria, the incidence of diarrhoea, and malnutrition (via effects on yields of agricultural crops). As there is considerable debate over the extent to which such short-term relationships will hold true under the longer-term processes of climate change, we made adjustments for possible changes in vulnerability, either through biological or socioeconomic adaptation. Estimates of future effects were interpolated back to give an approximate measure of the effects of the climate change that have occurred since 1990 on the burden of disease in 2000.

The effects considered here represent only a subset of the ways in which climate change may affect health. Other potential consequences include influences of changing temperature and precipitation on other infectious diseases (including the possible emergence of new pathogens), the distribution and abundance of agricultural pests and pathogens, destruction of public health infrastructure, and the production of photochemical air pollutants, spores and pollens. Rising sea levels may cause salination of coastal lands and freshwater supplies, resulting in population displacements. Changes in the availability and distribution of natural resources, especially water, may increase risk of drought, famine and conflict.

Our analyses suggested that climate change will bring some health benefits, such as lower cold-related mortality and greater crop yields in temperate zones, but these will be greatly outweighed by increased rates of other diseases, particularly infectious diseases and malnutrition in developing regions. We estimated a small proportional decrease in cardiovascular and respiratory disease mortality attributable to climate extremes in tropical regions, and a slightly larger benefit in temperate regions, caused by warmer winter temperatures. As there is evidence that some temperature-attributable mortality represents small displacements of deaths that would occur soon in any case, no assessment was made of the associated increase or decrease in disease burden. Climate change was estimated to increase the relative risk of diarrhoea in regions made up mainly of developing countries to approximately 1.01–1.02 in 2000, and 1.08–1.09 in 2030. Richer countries (gross domestic product [GDP] >US$ 6000/year), either now or in the future, were assumed to suffer little or no additional risk of diarrhoea. This modest change in relative risk relates to a major cause of ill-health, so that the estimated associated disease burden in 2000 is relatively large (47 000 deaths and 1.5 million disability-adjusted life years [DALYs]). Effects on malnutrition varied markedly even across developing subregions,[1] from large increases in SEAR-D (RR=1.05 in 2000, and 1.17 in 2030) to no change or an eventual small decrease in WPR-B. Again, these are small relative changes to a large disease burden, giving an estimated 77 000 deaths and 2.8 million DALYs in 2000. We calculated much larger proportional changes in the numbers of people killed in coastal floods (RR in EUR-B of up to 1.8 in 2000, and 6.3 in 2030), and inland floods (RR in AMR-

A of up to 3.0 in 2000, and 8.0 in 2030). Although the proportional change is much larger than for other health outcomes, the baseline disease burden is much lower. The aggregate health effect in 2000 is therefore comparatively small (2000 deaths and 193 000 DALYs). We estimated relatively large changes in the relative risk of falciparum malaria in countries at the edge of the current distribution. However, most of the estimated attributable disease burden (27 000 deaths and 1 million DALYs) is associated with small proportional changes in regions that are already highly endemic, principally in Africa.

Overall, the effects of global climate change are predicted to be heavily concentrated in poorer populations at low latitudes, where the most important climate-sensitive health outcomes (malnutrition, diarrhoea and malaria) are already common, and where vulnerability to climate effects is greatest. These diseases mainly affect younger age groups, so that the total burden of disease due to climate change appears to be borne mainly by children in developing countries.

Considerable uncertainties surround these estimates. These stem partly from the complexity of climate models, partly from gaps in reliable data on which to base climate–health relationships, and, most importantly, from uncertainties around the degree to which current climate–health relationships will be modified by biological and socioeconomic adaptation in the future. These uncertainties could be reduced in subsequent studies by (i) applying projections from several climate models; (ii) relating climate and disease data from a wider range of climatic and socioeconomic environments; (iii) more careful validation against patterns in the present or recent past; and (iv) more detailed longitudinal studies of the interaction of climatic and non-climatic influences on health.

1. Introduction

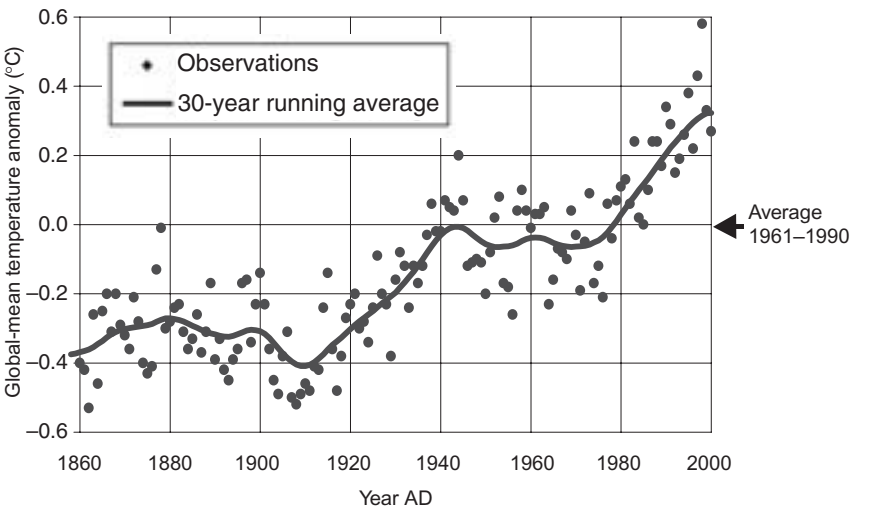
1.1 Evidence for climate change in the recent past and predictions for the future

Humans are accustomed to climatic conditions that vary on daily, seasonal and inter-annual time-scales. Accumulating evidence suggests that in addition to this natural climate variability, average climatic conditions measured over extended time periods (conventionally 30 years or longer) are also changing, over and above the natural variation observed on decadal or century time-scales. The causes of this climate change are increasingly well understood. Climatologists have compared climate model simulations of the effects of greenhouse gas (GHG) emissions against observed climate variations in the past, and evaluated possible natural influences such as solar and volcanic activity. They concluded that "... there is new and stronger evidence that most of the warming observed over the last 50 years is likely to be attributable to human activities" (IPCC 2001b).

The Third Assessment Report of the IPCC (IPCC 2001b) estimates that globally the average land and sea surface temperature has increased by 0.6±0.2 °C since the mid-19th century, with much of the change occurring since 1976 (Figure 20.1). Warming has been observed in all continents, with the greatest temperature changes occurring at middle and high latitudes in the Northern Hemisphere. Patterns of precipitation have also changed: arid and semi-arid regions are apparently becoming drier, while other areas, especially mid-to-high latitudes, are becoming wetter. Where precipitation has increased, there has also been a disproportionate increase in the frequency of the heaviest precipitation events (Karl and Knight 1998; Mason et al. 1999). The small amount of climatic change that has occurred so far has already had demonstrable effects on a wide variety of natural ecosystems (Walther et al. 2002).

Climate model simulations have been used to estimate the effects of past, present and likely future GHG emissions on climate changes. These models are primarily based on data on the heat-retaining properties of gases released into the atmosphere from natural and anthropogenic (man-made) sources, as well as the measured climatic effects of other natural phenomena, as described above. The models used by the IPCC have been validated by "back-casting"—that is, testing their ability to explain climate variations that already occurred in the past. In general, the models are able to give good approximations of past patterns only

Figure 20.1 Observed global average land and sea surface temperatures from 1860 to 2000



Source: Climatic Research Unit, Norwich, England.

when anthropogenic emissions of non-GHG air pollutants (particulates, dust, oxides of sulfur, etc.) are included along with natural phenomena (IPCC 2001b). This emphasizes that (i) the models represent a good approximation of the climate system; (ii) natural variations are important contributors to climatic variations, but cannot adequately explain past trends on their own; and (iii) anthropogenic GHG emissions are an important contributor to climate patterns, and are likely to remain so in the future.

Considering a range of alternative economic development scenarios and model parameterizations, the IPCC concluded that if no specific actions were taken to reduce GHG emissions, global temperatures would rise between 1.4 and 5.8 °C from 1990 to 2100. The projections for precipitation and wind speed are less consistent in terms of magnitude and geographical distribution, but also suggest significant changes in both mean conditions and in the frequency and intensity of extreme events (Table 20.1).

1.2 Estimating the effects of climate change on health

Human health is sensitive to temporal and geographical variations in weather (short-term fluctuations in meteorological conditions) and climate (longer-term averages of weather conditions). Weather has not historically been considered as subject to alteration by human actions, although its effects may be lessened by adaptation measures (e.g. Kovats et al. 2000b). While adaptation is also a very important determinant of the health consequences of climate change, the effect of anthropogenic GHG emissions on climate means that climate change can in principle be considered a risk factor that could potentially be altered by human intervention, with associated effects on the burden of disease.

The effects of GHG emissions on human health differ somewhat from the effects of other risk factors in that they are mediated by a diversity of causal pathways (e.g. Figure 20.2; McMichael et al. 1996; Patz et al. 2000; Reiter 2000) and eventual outcomes, typically long delays between cause and effect, and great difficulties in eliminating or substantially reducing the risk factors. An additional challenge is that climate change occurs against a background of substantial natural climate variability, and its health effects are confounded by simultaneous changes in many other influences on population health (Kovats et al. 2001; Reiter 2001; Woodward et al. 1998). Empirical observation of the health consequences of long-term climate change, followed by formulation, testing and then modification of hypotheses would therefore require long time-series (probably several decades) of careful monitoring. While this process may accord with the canons of empirical science, it would not provide the timely information needed to inform current policy decisions on GHG emission abatement, so as to offset possible health consequences in the future. Nor would it allow early implementation of policies for adaptation to climate changes, which are inevitable owing

Table 20.1 Estimates of confidence in observed and projected changes in extreme weather and climate events

Changes in phenomenon	*Confidence in observed changes (latter half of 1900s)*	*Confidence in projected changes (during the 21st century)*
Higher maximum temperatures and more hot days over nearly all land areas	Likely[a]	Very likely[a]
Higher minimum temperatures, fewer cold days and frost days over nearly all land areas	Very likely[a]	Very likely[a]
Reduced diurnal temperature range over most land areas	Very likely[a]	Very likely[a]
Increase of heat index[b] over land areas	Likely,[a] over many areas	Very likely,[a] over most areas
More intense precipitation events[a]	Likely,[a] over many northern hemisphere mid- to high-latitude land areas	Very likely,[a] over many areas
Increased summer continental drying and associated risk of drought	Likely,[a] in a few areas	Likely,[a] over most mid-latitude continental interiors. (Lack of consistent projections in other areas)
Increase in tropical cyclone peak wind intensities[c]	Not observed in the few analyses available	Likely,[a] over some areas
Increase in tropical cyclone mean and peak precipitation intensities[d]	Insufficient data for assessment	Likely,[a] over some areas

[a] Judgement estimates for confidence: *virtually certain* (greater than 99% chance that the result is true); *very likely* (90–99% chance); *likely* (66–90% chance); *medium likelihood* (33–66% chance); *unlikely* (10–33% chance); *very unlikely* (1–10% chance); *exceptionally unlikely* (less than 1% chance).

[b] Past and future changes in tropical cyclone location and frequency are uncertain.

[c] For other areas, there are either insufficient data or conflicting analyses.

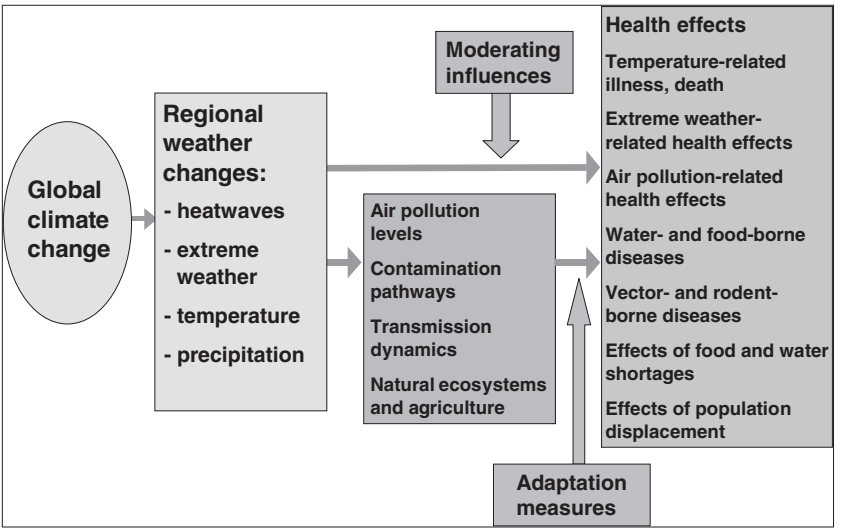
[d] Based on warm season temperature and humidity.

Source: Adapted from IPCC (2001b).

to both natural variations and past GHG emissions. Therefore, the best estimation of the future health effects of climate change will necessarily come from modelling based on current understanding of the effects of climate (not weather) variation on health from observations made in the present and recent past, acknowledging the influence of a large range of mediating factors.

Since the early 1990s, IPCC Working Group II has collated some of the accumulating predictions of climate effects on health (IPCC 2001a). The health effects of climate variability and change have also been reviewed by a national committee in the United States of America

Figure 20.2 Pathways through which climate change may affect health



Source: Adapted from Patz et al. (2000).

(National Research Council 2001a) and, more recently, in a book by the World Health Organization (WHO) (McMichael et al. 2003). As yet, however, there has been no concerted attempt to integrate these various research findings into a single standardized estimate of the likely net health effects of climate change, nor to estimate the possible health gains associated with different mitigation and amelioration strategies. In addition to the uncertainties of future climate projections, there are several obstacles to achieving this aim.

- Not all of the probable health outcomes have been modelled, often due to lack of parameterization data and the complexity of causal pathways. Modelling efforts so far have tended to concentrate on those causal relationships that can more easily be modelled, rather than those with the potentially greatest effects (e.g. extreme temperatures on cardiovascular disease mortality, rather than sea-level rise on the health of displaced populations).
- Little emphasis has been given to the validation of models relating climate change to health. Validation would provide a basis for making uncertainty estimates around projections, and would afford an objective criterion for choosing between different models or modelling approaches.

- Adaptations to climate change (i.e. autonomous or planned responses that reduce the vulnerability of populations to the consequences of climate change) are often not addressed.
- Interactions between the effects of climate change and other changes to human populations (e.g. investment in health infrastructure, level and equity of distribution of wealth) are seldom explicitly estimated.
- The various disease-specific models invariably generate outputs in different units, which may be only indirectly related to disease burden (e.g. populations at risk of disease transmission, rather than disease incidence). This hampers estimation of the aggregated health impacts of different scenarios.
- Little effort has previously been directed to describing and understanding the geographical variations in likely impacts.

The first four obstacles are likely to be at least partially addressed in the future, as disease-specific models become more sophisticated and, perhaps more importantly, through the accumulation of greater quantities of reliable data for model parameterization and testing. In this chapter, we have estimated the relative risk of a series of health outcomes under a range of scenarios of climate change, variously mitigated by reducing GHG emissions. In all cases, care was taken to describe explicitly the scientific basis for our estimation, the assumptions that were built into the quantitative models, and to give realistic uncertainty estimates around projections. Later sections describe ways in which specific disease models may be improved.

2. Risk factor definition and measurement

2.1 Definitions of risk factor and exposure scenarios

The risk factor was defined as current and future changes in global climate attributable to increasing atmospheric concentrations of greenhouse gases (GHGs).

Composite climate scenarios are adopted instead of the (more preferable) continuous measurements of individual climate variables because (i) climate is a multivariate phenomenon, including temperature, precipitation, wind speed, etc., and therefore cannot be measured on a single scale; (ii) climate changes will vary significantly with geography and time: these are not fully captured in global averages of climate variables; and (iii) all aspects of climate are likely to be altered by GHG levels in the atmosphere.

The exposure categories considered here are global climate scenarios resulting at specified points in time over the coming half-century from:

1. unmitigated emissions trends, that is, approximately following the IPCC IS92a scenario;

2. emissions reduction, resulting in stabilization at 750 ppm CO_2 equivalent by the year 2210 (s750);
3. more severe emissions reduction, resulting in stabilization at 550 ppm CO_2 equivalent by the year 2170 (s550);
4. average climate conditions for 1961–1990, the World Meteorological Organization (WMO) climate normal (baseline).

Although future GHG emissions are inherently uncertain, the unmitigated emissions scenario adopted here was, until recently, the IPCC mid-range projection, and was very widely used in climate impact modelling. The stabilization categories used here represent plausible, though economically and technically challenging, projections that are dependent on there being major efforts to curtail emissions. Estimated changes in CO_2 concentrations, and associated changes in global temperature and sea level, are shown in Table 20.2 and Figure 20.3. Although alternative emissions scenarios for climate stabilization are available, they have not been applied to a wide range of impact models.

We do not attempt to estimate all health outcomes of specific policy/development pathways through which these, or other, GHG levels could be achieved: for example, compliance with the Kyoto Protocol of the United Nations Framework Convention on Climate Change (UNFCCC), or of the world following one or other of the IPCC Special Report on Emissions Scenarios (SRES)—both of which also include descriptions of alternative future global socioeconomic development scenarios. The costs or additional benefits of specific interventions to

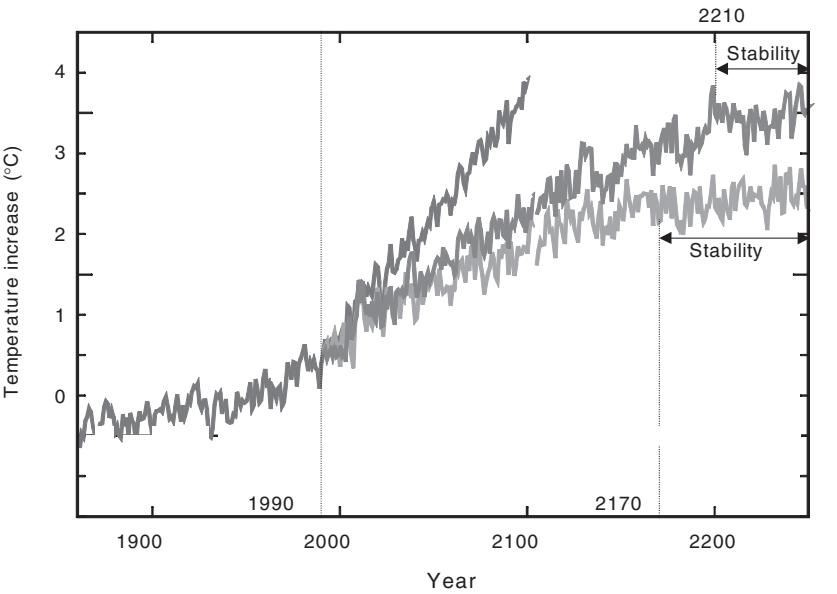
Table 20.2 Successive measured and modelled CO_2 concentrations, global mean temperature and sea-level rise associated with alternative emissions scenarios

	1961–90	*1990s*	*2020s*	*2050s*
Carbon dioxide concentration (ppm) by volume				
HadCM2 unmitigated emissions	334	354	441	565
S750	334	354	424	501
S550	334	354	410	458
Temperature (°C change)				
HadCM2 unmitigated emissions	0	0.3	1.2	2.1
S750	0	0.3	0.9	1.4
S550	0	0.3	0.8	1.1
Sea-level (cm change)				
HadCM2 unmitigated emissions	0	—	12	25
S750	0	—	11	20
S550	0	—	10	18

— No data.

Source: McMichael et al. (2000a).

Figure 20.3 The global average temperature rise predicted from the unmitigated emissions scenario (upper trace), and emission scenario which stabilizes CO_2 concentrations at 750 ppm (middle trace) and at 550 ppm (lower trace)



Note: All values are relative to mean values for the period 1961–1990, and may therefore be either positive or negative.

Source: Hadley Centre (1999).

achieve this reduction are artificially separated from the resulting health benefits. For such a distal risk factor, this separation of intervention from exposure and resulting health consequences may potentially introduce inconsistencies: for example, the economic changes necessary to achieve GHG stabilization are more consistent with some projections of levels and distribution of population and GDP than others. These socioeconomic factors are themselves likely to effect disease rates, potentially in interaction with climate. Integrated assessment of all effects of interventions would be conceptually more consistent, but this has not been attempted here, since it would introduce an additional layer of uncertainty and assumptions into the models and has previously only been explored for a few health outcomes (Tol and Dowlatabadi 2001).

The choice of baseline or "theoretical minimum" exposure follows the WMO and IPCC practice of using the observed global climate normal (i.e. averages) for 1961–1990 (New et al. 1999) as a reference point. Alternative baselines, such as pre-industrial climate, are not used,

because of the absence of a published consensus among climatologists on definitions of an appropriate time period and the relative roles of anthropogenic and natural influences before 1961–1990. This choice of a 1961–1990 baseline will therefore generate relatively conservative estimates of change in exposure and associated disease outcomes, as it does not address any human-induced climate change that occurred before this period. Indeed, as explained in section 2.6 below, the further choice of 1990 as the actual baseline year for linear-regression based estimates at current and future years heightens the conservative nature of these estimates.

The approach here treats climate change as a slowly evolving and continuous exposure, with the majority of disease models linked to those changes for which climate models make the most consistent predictions: gradual changes in temperature and, to a lesser extent, precipitation. This is again a limited approach. As shown in Table 20.1, it is very likely that climate change will also increase the frequency of extreme conditions, with likely effects on health. However, quantitative estimates of increased frequency have only very recently become available for some measures of extremes (e.g. wet winters; Palmer and Ralsanen 2002), and are not yet available for different GHG scenarios. The consequences of such changes are modelled here only in the context of inland flooding, but they could potentially be applied to other health end-points in the future.

Finally, there is some concern that disruption of the climate system may pass critical thresholds, resulting in abrupt rather than gradual changes (Broecker 1997; National Research Council 2001a) and associated rapid impacts on health. There is no consensus on the probability of such events, and they have therefore not been included in any published health outcome assessment studies. However, they should be borne in mind as a "worst-case" scenario.

2.2 Methods for estimating risk factor levels

Projections of the extent and geographical distribution of climate change were generated by applying the various emissions scenarios described above to the HadCM2 global climate model (GCM) of the Hadley Centre in the United Kingdom of Great Britain and Northern Ireland (Tett et al. 1997). This is one of several alternative GCMs used by the IPCC; it generates projections of changes in temperature and other climate properties which have been verified by back-casting (Johns et al. 2001), and which lie approximately in the middle of the range generated by alternative models.

The HadCM2 model generates estimates of the principal characteristics of climate, including temperature, precipitation and absolute humidity, for each cell of a global grid at resolution 3.75° longitude by 2.5° latitude. As for most climate change models, HadCM2 generates daily projections representing both long-term trends and the degree of natural

climate variability, but not necessarily its specific temporal pattern (i.e. the models do not accurately predict the climate of specific days or months). In order to account for such natural variability, the outputs that are most commonly used for modelling consequences of climate change are monthly means for average 30-year periods centred on the 2020s, 2050s, 2080s, etc. The baseline climate (1961–1990) describes the same properties for the land surface of the world at $0.5° \times 0.5°$ resolution.

The climate model projections describe forecast changes in global climate conditions. Therefore, we did not attempt to estimate the "exposure prevalence": the entire world population was assumed to be exposed to one or other global climate scenario (i.e. exposure prevalence = 100%). However, it should be noted that the climate scenarios incorporate geographical variation in both current climate (e.g. the lower temperatures in higher latitudes) and projected climate change (e.g. more rapid and intense warming is predicted in high northern latitudes than elsewhere). Different populations will therefore experience different climate conditions under any one climate-change scenario.

2.3 Uncertainties in risk factor levels

Two major sources of uncertainty surround the forecasting of future climate scenarios: (i) uncertainty in changes in factors such as population, economic growth, energy policies and practices on GHG emissions; and (ii) uncertainties over the accuracy of any climate model in predicting the effects of specified emissions scenarios on future climate in specific locations, against the background of substantial natural climate variability over time, and in space (i.e. downscaling). Climatologists have only very recently provided probabilistic measurements of uncertainty incorporating one or both of these sources (Knutti et al. 2002; Stott and Kettleborough 2002), and there is still debate over the reliability and utility of such measures (Schneider 2002). They have not previously been applied in impact studies (Katz 2002), and were therefore not used in this assessment. More importantly, however, there remains considerable uncertainty over the accuracy with which any single model can predict future climate. This is usually addressed by using outputs from a range of models, from independent groups. This was not possible here, as the particular GHG stabilization scenarios have only been applied, and fed through to estimates of likely consequences, for the HadCM2 model.

For this analysis, we did not address the first and last sources of uncertainty explicitly, and assumed that it is incorporated in the various alternative exposure scenarios, reflecting different trajectories of GHG emissions. The second source of uncertainty was partially addressed by using 30-year averages of climate conditions, which helps to "smooth out" the effects of natural climate variability. Further, the single model used was run with slight variations in initial conditions, allowing the calculation of an "ensemble mean". Four runs were used to generate an ensemble mean for the unmitigated emissions scenario. However, only

single climate runs were available for the stabilization scenarios. Therefore, the climate scenarios associated with those emissions scenarios are more uncertain.

Although it was not feasible to generate formal uncertainty ranges and feed these through the disease models, the approximate degree of uncertainty is illustrated in Appendix A. Here, the stabilization scenarios were run on a suite of 14 simple climate models, making different plausible assumptions about climate sensitivity to GHG emissions using the simplified COSMIC climate model described by Schlesinger and Williams (1997). This illustrates the degree of variation between the projections for future temperature and precipitation patterns. (Note that projections for precipitation vary more between models than does temperature—therefore models that rely on precipitation estimates from single scenarios have an additional component of uncertainty.)

3. Risk factor–disease relationship

3.1 Outcomes included

The health outcomes addressed here were selected on the basis of observed sensitivity to temporal and geographical climate variation, importance in terms of mortality and/or burden of disease (Longstreth 1999; McMichael et al. 1996; Patz et al. 2000) and availability of quantitative global models (or feasibility of constructing informative models in the time available) (Table 20.3). More detail on evidence for causality and quantitative estimation methods for each health outcome is given below.

Climate change is by and large a relatively distal risk factor for ill-health, often acting through complex causal pathways which result in heterogeneous effects across populations. There is, therefore, a series of additional likely outcomes that have not yet been formally modelled. They include the potential health consequences of climate change on:

Table 20.3 Health outcomes considered in this analysis

Outcome class	*Incidence/ prevalence*	*Outcome*
Direct effects of heat and cold	Incidence	Cardiovascular disease deaths
Foodborne and waterborne diseases	Incidence	Diarrhoea episodes
Vector-borne diseases	Incidence	Malaria cases
Natural disasters[a]	Incidence	Deaths due to unintentional injuries
	Incidence	Other unintentional injuries (non-fatal)
Risk of malnutrition	Prevalence	Non-availability of recommended daily calorie intake

[a] All natural disaster outcomes are separately attributed to coastal floods and inland floods/landslides.

- changes in pollution and aeroallergen levels;
- the rate of recovery of the ozone hole, affecting exposure to ultraviolet radiation (Shindell et al. 1998);
- changes in the distribution and transmission of other infectious diseases, particularly other vector-borne diseases, geohelminths and rodent-borne diseases, and possible emergence of new pathogens;
- the distribution and abundance of plant and livestock pests and diseases, affecting agricultural production (Baker et al. 2000; Rosenzweig et al. 2001);
- the probability of crop failure through prolonged dry weather and famine, depending on location and crisis management;
- population displacement due to natural disasters, crop failure, water shortages; and
- destruction of health infrastructure in natural disasters.

3.2 Methods for estimating risk factor–disease relationships

Various methods have been developed for the quantitative estimation of health outcomes of climatic change (reviewed by Martens and McMichael 2002; McMichael and Kovats 2000). It is not yet feasible to base future projections on observed long-term climate trends, for three reasons: (i) the lack of standardized long-term monitoring of climate-sensitive diseases in many regions; (ii) methodological difficulties in measuring and controlling for non-climatic influences on long-term health trends; and (iii) the small (but significant) climate changes that have occurred so far are an inadequate proxy for the larger changes that are forecast for coming decades (Campbell-Lendrum et al. 2002).

Instead, estimates are based on observations of the effects of shorter-term climate variation in the recent past (e.g. the effects of daily or inter-annual climate variability on specific health outcomes) or the present (e.g. climate as a determinant of current disease distribution), or on specific processes that may influence health states (e.g. parasite and vector population dynamics in the laboratory, determining the transmission of infectious diseases). These quantitative relationships were then applied to future climate scenarios (Figure 20.3). Such an approach makes the important assumption that such associations will be maintained in the future, despite changes in mediating factors such as socioeconomic variables, infrastructure and technology. This introduces significant uncertainty, and possibly bias, in the estimates.

The extent and type of modelling applied to different health effects vary considerably. Consequently, several outcomes can only be estimated by crude adaptation of the outputs of available models. For example, some of the predictive models generate health-relevant outputs that do

not correspond directly to categories of disease states used in the Global Burden of Disease (GBD) study. These include the incidence of deaths and injuries due to, specifically, floods (rather than injuries due to all causes), or populations at risk of hunger or malaria infection (rather than prevalence of malnutrition, or incidence of clinical malaria). Currently, there are spatial resolution differences between models, which is not ideal. These relate to how the models account for geographical variation in: (i) baseline climate (potentially differentiated to 0.5° globally, or higher resolutions for some regions); (ii) climate change (usually at the level of the GCM projections: 3.75° longitude by 2.5° latitude); and (iii) aggregation of the final results (occasionally according to regions other than subregions, depending on the purpose of the original model). Levels of spatial resolution for each disease model are described in section 3.6 and onwards. All of the above considerations are represented in the descriptions of strength of evidence and quantitative estimates of uncertainty for specific health outcomes.

Assumptions

Simplifying assumptions have been made to facilitate clear definition of scenarios and associated consequences.

Different mechanisms for reducing GHG emissions

The alternative GHG emissions scenarios outlined above could be achieved through an almost infinite variety of changes to economic and social development and energy use policies. As outlined in section 2.1, we did not attempt to estimate the secondary effects of GHG mitigation policies on health. These effects are potentially large. They include relatively direct mechanisms which may be *negative*, such as the potential negative effects of GHG emissions policies on economic development, personal wealth and vulnerability to disease (Tol and Dowlatabadi 2001), or *positive*, such as reduction in the levels of ozone and other outdoor (Kunzli et al. 2000) and indoor air pollutants (Wang and Smith 1999). They may also act through more complex routes, for example, avoiding production of aeroallergens in CO_2-enriched environments (Wayne et al. 2002; Ziska and Caulfield 2000).

Population growth

The models described below estimated the relative per capita incidence of specific health outcomes under the different climate scenarios. The size and distribution of current and future populations therefore affect the relative risk estimates either (i) where the climate hazard is not evenly distributed geographically throughout the region, or (ii) where population is an integral part of the model—for example, in the risk-of-hunger model, where population size has an influence on food availability per capita. In adjusting the relative risks for these population effects, the distribution of future populations was estimated by applying the World

Bank mid-range estimate of population growth either at the national level (for malnutrition), or to a 1°×1° resolution grid map of population distribution (Bos et al. 1994) for all other outcomes.

Modifying factors: adaptation and vulnerability

Factors such as physiological adaptation, technological and institutional innovation and individual and community wealth will influence not only the exposure of individuals and populations to climate hazards, but also the associated hazards (e.g. IPCC 2001a; Reiter 2000; Woodward et al. 1998). For some assessments, simpler modifying factors are integrated into the models for both present and future effects. For example, estimates of changes in the number of people at risk of hunger incorporate continental projections of economic growth, affecting capacity to buy food (Parry et al. 1999). Other models incorporate the effects of existing modifiers when defining current climate–disease relationships, such as estimates of the global distribution of malaria based on current climate associations (Rogers and Randolph 2000). Such models implicitly capture the *current* modifying effects of socioeconomic and other influences on climate effects, but do not attempt to model *future* changes in these modifiers. Finally, some models make no estimate of such modifying influences in either the present or future; for example, models that estimate future changes in the geographical range which is climatically suitable for malaria transmission, and associated populations at risk (Martens et al. 1999). To generate consistent estimates in this analysis:

- we attempted to account for current geographical variation in vulnerability to climate, where not already incorporated into the predictive models.
- we attempted to account for future changes in disease rates due to other factors (e.g. decreasing rates of infectious disease due to technological advances/improving socioeconomic status), and for changes in population size and age structure (e.g. potentially greater proportion of older people at higher risk of mortality related to cardiovascular disease in response to thermal extremes). This was addressed by calculating only relative risks under alternative climate change scenarios, which should be applied to GBD projections of disease rates and population size and age structure. The GBD projections take into account the effects of changing GDP, "human capital" (as measured by average years of female education), and time (to account for trends such as technological development) (Murray and Lopez 1996) on the overall "envelope" of cause-specific mortality and morbidity for diseases affected by climate change.
- all quantitative estimates of the health effects of climate change were based on observed effects of climate variations either over short time

periods, or between locations. They therefore made the important assumption that these relationships are also relevant to long-term climate change. To avoid unrealistic extrapolation of short-term relationships, we included consideration of mechanisms by which climate-health relationships may alter over time (i.e. adaptation). We considered whether each disease in turn was likely to be significantly affected by biological adaptation (i.e. either behavioural, immunological or physiological) and/or by generally improving socioeconomic conditions (i.e. increasing GDP) (see Table 20.4). In each case we defined appropriate adjustments to the relative risk estimates, in line with published studies. The different factors for each health outcome were then applied to the same projections of future GDP (WHO/EIP/GPE, unpublished data, 2001) and changing climate (from our models), to adjust the relative risks over the time course of the assessment. There is, however, substantial uncertainty over the most likely degree of adaptation under different conditions. This was reflected in

Table 20.4 Assumptions on adaptation and vulnerability

	Biological[a] *adaptation affecting RRs*	*Socioeconomic adaptation affecting RRs*
Direct physiological effects of heat and cold	Yes. Temperature associated with lowest mortality was assumed to change directly with temperature increases driven by climate change	None
Diarrhoea	None	Assumed RR = 1 if GDP per capita rises above US$ 6000/year
Malnutrition	None	Food-trade model assumed future increases in crop yields from technological advances, increased liberalization of trade, and increased GDP[b]
Disasters: coastal floods	None	Model assumed the RR of deaths in floods decreases with GDP, following Yohe and Tol (2002)
Disasters: inland floods and landslides	None	Model assumed the RR of deaths in floods decreases with GDP, following Yohe and Tol (2002)
Vector-borne diseases: malaria	None	None (for RR)

[a] Physiological, immunological and behavioural.

[b] GDP scenarios are developed from EMF14 (Energy Modeling Forum 1995).

the uncertainty estimates for the relative risks for each disease. Quoted uncertainty estimates therefore describe uncertainties around climate change predictions, about current exposure–response relationships, and around the degree to which these are likely to be maintained in the future. Since, to date, these have not been formally modelled, they were generated here by qualitative assessment in collaboration with the original modelling group. The uncertainty estimates should therefore be interpreted with caution.

- we ignored more complex aspects of future vulnerability. Whereas projected trends for average income (which is included in estimating baseline rates and, where possible, relative risks) are broadly positive, other factors may have opposite effects. These include income distribution, maintenance of disease surveillance, control and eradication programmes, technological change and secondary or threshold effects, such as the protective effect of forests in reducing the frequency and intensity of flooding (e.g. Fitzpatrick and Knox 2000).
- we made no attempt to estimate the effects of actions taken specifically to adapt to the effects of climate change (e.g. the upgrading of flood defences specifically to cope with sea-level rise attributable to climate change). Therefore, our estimates represent a "business-as-usual" scenario of the health effects associated with global climate change.

3.3 Estimation for different time points

As stated above, climate model outputs are usually presented as averages over 30-year periods, for example, centred on 2025 and 2055. In order to generate estimates for any specific year, we defined 1990 (i.e. the last year of the baseline climate period) as year 0. Central, lower and upper estimates of relative risks were calculated for the 2020s (i.e. centred on 2025) and the 2050s, using models described elsewhere. Quoted estimates for the years 2001, 2005, 2010 and 2020 were estimated by linear regression against time between 1990 and 2025. Estimates for 2030 were estimated by linear regression between 2025 and 2055. Using this method, our choice of 1990 as the baseline year rather than the middle of the 1961–1990 period led to conservative estimates of health consequences, particularly in the near future.

3.4 Risk reversibility

In the context of this assessment, risk reversibility describes the proportion of the health consequences that would be avoided if the population were shifted to a different exposure scenario. Given the definition of exposure scenarios, complete avoidance of climate change (i.e. populations exposed to baseline climate conditions rather than unmitigated climate change) would avoid all of the health consequences that we have described. Risk reversibility would therefore be 100% in this case.

3.5 Criteria for identifying relevant studies and for estimating strength of evidence

In identifying relevant studies, we have considered all publications reviewed in the IPCC Third Assessment Report (IPCC 2001a), as well as those found in more recent literature searches (Appendix B). However, as the field is relatively new and expanding rapidly, we have also used some studies that are either in-press or submitted for publication (material available on request). As these may not be easily accessible to readers of this report, the major underlying assumptions are described.

There are still relatively few models that link climate change models to quantitative global estimates of health or health-relevant outcomes (e.g. numbers of people flooded or at risk of hunger). Where global models did not exist, we have extrapolated from models that make local or regional projections. Methods for extrapolating to the subregions are described separately for each disease. Where there was more than one published model for a particular outcome, decisions on model selection were based as much as possible on validation against historical/geographical patterns. We excluded models: (i) that have been shown to be significantly less accurate than equivalent models in predicting historical or geographical trends; or (ii) that have been superseded by later models by the same group; or (iii) that are based on unrealistic biological assumptions; or (iv) that cannot be plausibly extrapolated to a wider area. Given the very limited means for model validation, these results involved choices made in this work among models and resulted in very large uncertainty.

As climate change impact assessment is, at this stage, predominantly a model-based exercise, the assessment of strength of evidence for causality is necessarily indirect. It is based on two considerations: (i) the strength of evidence for the current role of climate variability in affecting the health outcome, and (ii) the likelihood that this relationship between climate and health will be maintained through the process of long-term climate change.

Several of the uncertainties involved in this exercise, particularly those relating to climate modelling and those around the quantitative relationships between climate and health, should decrease through improvements in modelling, and more importantly, through better empirical research. However, estimation of climate change effects is currently a predictive exercise, based on some form of indirect modelling (e.g. analysis of temporal or geographical variation in climate), rather than direct experience (i.e. of a change process that has previously occurred). It is therefore possible that some of the health outcomes listed above may not respond in the predicted manner. On the other hand, we do not yet have direct experience of the full range of health outcomes that may be associated with exposure.

The analyses described here were specifically to estimate the future disease burden, which may be avoided by climate change mitigation policies. However, it must be emphasized that these burdens may also be reduced by adaptation interventions to reduce the vulnerability of populations. Given the "exposure commitment" (unavoidable climate change due to past GHG emissions), and the large gap between even plausible mitigation scenarios and the baseline climate, adaptation strategies are essential to the goal of reducing adverse health effects. These include early warning systems and defences for protection from natural disasters, and improved health infrastructure (e.g. water and sanitation, control programmes for vector-borne diseases) to reduce the baseline incidence of climate-sensitive diseases.

3.6 Direct effects of heat and cold on mortality

An association between weather and daily mortality has been shown consistently in many studies in diverse populations. The effect of extreme temperature events (heat-waves and cold spells) on mortality has also been well described in developed countries. However, temperature-attributable mortality has also been found at "moderate" temperatures (Curriero et al. 2000; Kunst et al. 1993).

Causality is supported by physiological studies of the effects of very high or very low temperatures in healthy volunteers. High temperatures cause some well described clinical syndromes such as heat-stroke (see review by Kilbourne 1989). Very few deaths are reported as directly attributed to "heat" (International Classification of Diseases, ninth revision [ICD-9 code 992.0]) in most countries. Exposure to *high* temperatures increases blood viscosity and it is therefore plausible that heat stress may trigger a vascular event such as heart attack or stroke (Keatinge et al. 1986a). Studies have also shown that elderly people have impaired temperature regulation (Drinkwater and Horvath 1979; Kilbourne 1992; Mackenbach et al. 1997; Vassallo et al. 1995). Clinical and laboratory studies indicate that exposure to *low* temperatures causes changes in haemostasis, blood viscosity, lipids, vasoconstriction and the sympathetic nervous system (e.g. Keatinge et al. 1986b, 1989; Khaw 1995; Woodhouse et al. 1993, 1994). The strongest physiological evidence is therefore for cardiovascular disease.

Population-based studies also provide evidence that environmental temperature affects mortality due to both cardiovascular and respiratory disease. The best epidemiological evidence is provided by time-series studies of daily mortality. These methods are considered sufficiently rigorous to assess short-term associations (days, weeks) between environmental exposures and mortality, if adjustment is made for longer-term patterns in the data series, particularly the seasonal cycle and any long-term trends, such as gradually decreasing mortality rates over decades (Schwartz et al. 1996).

The effect of a "hot" day is apparent for only a few days in the mortality series; in contrast, a "cold" day has an effect that lasts up to two weeks. Further, in many temperate countries, mortality rates in winter are 10–25% higher than death rates in summer. The causes of this winter excess are not well understood (Curwen and Devis 1988). It is therefore plausible that different mechanisms are involved and that cold-related mortality in temperate countries is related in some part to the occurrence of seasonal respiratory infections.

Although the physiological evidence for causality of an effect of temperature on mortality is greatest for cardiovascular, followed by respiratory, diseases, temperature has been shown to affect all-cause mortality in areas where cardiovascular disease rates are relatively low and infectious disease mortality is relatively high. Many studies report the seasonal patterns of infectious disease in developing countries, but the role of temperature is not well described. It is likely that seasonal rains influence the seasonal transmission of many infectious diseases. High temperatures encourage the growth of pathogens and are associated with an increased risk of diarrhoeal disease in poorer populations (see section 3.7).

Studies have also described mortality and morbidity during extreme temperature events (heat-waves). However, these events are, by definition, rare. It is therefore difficult to compare heat-waves in different populations and for different intensities. The studies that have been used to describe the effects of heat-waves (episode analyses) also use different methods for estimating the "expected" mortality, which makes comparison difficult (Whitman et al. 1997). An assessment of the health consequences of climate change on thermal stress requires estimation of past and future probabilities of extreme temperature events. The current methods of assessment use 30-year averages of monthly data, and few scenarios consider change in the frequency or magnitude of extreme events. This is because: (i) suitable methods have not been developed, and (ii) climate model output at the appropriate spatial and temporal resolution is not readily available (Goodess et al. 2001).

There is little published evidence of an association between weather conditions and measures of morbidity such as hospital admissions or primary care consultations (Barer et al. 1984; Ebi et al. 2001; Fleming et al. 1991; McGregor et al. 1999; Rothwell et al. 1996; Schwartz et al. 2001). A study of general practitioner consultations among the elderly in Greater London found that temperature affected the rate of consultation for respiratory diseases but not that for cardiovascular diseases (Hajat et al. 2001). However, it is not clear how these end-points relate to quantitative measures of health burden.

Estimating the temperature–mortality relationship

A review of the literature was undertaken to identify studies that report relationships between daily temperature and mortality (see Appendix B).

The following criteria were used to select studies for deriving the modelled estimates.

- A study that uses daily time-series methods to analyse the relationship between daily mean temperature and mortality.
- A study that reports a coefficient from linear regression which estimates the percentage changes in mortality per degree centigrade changes in temperature above and below a reported threshold temperature.

Several studies have estimated future temperature-related mortality for a range of climate scenarios (e.g. Guest et al. 1999; Kalkstein and Greene 1997; Langford and Bentham 1995; Martens 1998a). These methods were not considered appropriate for this project, as described in Appendix B.

The best characterized temperature–mortality relationships are those for total mortality in temperate countries. Fewer studies have also looked at the particular causes of death for which physiological evidence is strongest: cardiovascular disease, and to a lesser extent respiratory disease. In this study, we used the specific relationships for cardiovascular disease where these were available (temperate and cold-climate zones), and the general relationships for all-cause mortality for climatic regions where such disease-specific relationships could not be found in the literature review (all tropical populations) (Table 20.5).

As outlined in section 2.2, it was assumed that everybody is exposed to the ambient temperatures prevailing under the different climate scenarios. However, populations differ in their responses to temperature variability, which is partly explained by location or climate.

The global population distribution was divided into five climate zones (Table 20.6), according to definitions developed for urban areas by the

Table 20.5 Temperature-related mortality: summary of exposure–response relationships, derived from the literature[a]

		Medical all-cause mortality[b]		*Cardiovascular mortality*	
Climate zone	*Threshold (T_{cutoff})*	*Heat*	*Cold*	*Heat*	*Cold*
Hot and dry	23	3.0	1.4	—	—
Warm humid	29	5.5	5.7	—	—
Temperate	16	NA	NA	2.6	2.9
Cold	16	NA	NA	1.1	0.5

— No data.

NA Not applicable.

[a] Change in mortality per 1 °C change in mean daily temperature (%).

[b] Excludes external causes (deaths by injury and poisoning).

Table 20.6 Climate zones

Zone	*Climate definition*	*% of world population in zone (1990s)*	*City from which representative daily temperature distribution was derived*	*Mean temperature (°C) (5th–95th percentile)*
Hot/dry	Temperature of warmest month >30 °C	17	Delhi	25.0 (13.5–35.2)
Warm/humid	Temperature of the coldest month >18 °C, warmest month <30 °C	21	Chiang Mai	26.3 (21.6–29.5)
Temperate	Average temperature of the coldest month <18 °C and >–3 °C, and average temperature of warmest month >10 °C	44	Amsterdam	9.6 (2.0–17.8)
Cold	Average temperature of warmest month >10 °C and that of coldest month <–3 °C	14	Oslo	5 (–6.3–16.5)
Polar	Average temperature of the warmest month <10 °C	0.2	NA	NA

NA Not applicable.

Australian Bureau of Meteorology (BOM 2001). The population in the polar zone is small (0.2% of world population) and was excluded.

It was necessary to estimate daily temperature distributions in order to calculate the number of attributable deaths. Daily temperature distributions clearly vary a great deal, even between localities within the same country. However, it was not feasible within this assessment to obtain sufficient meteorological data to estimate daily temperature distributions throughout the world at a fine spatial resolution. Therefore, a single distribution was chosen to represent each climate zone. New daily temperature distributions were then estimated for each climate scenario, by shifting the currently observed temperature distributions by the projected change in mean temperatures for each month, and of the variability of daily temperatures as well as changes in the mean.

Estimating temperature-attributable mortality

An exposure–response relationship and threshold temperature (T_{cutoff}) was applied within each climate zone (Table 20.7). The average temperature difference above (hot days) and below (cold days) this temperature was calculated for baseline climate and each of the climate scenarios.

The short-term relationships between daily temperature and mortality (Table 20.5) were used to estimate the annual attributable fraction of deaths due to hot days and cold days for each of the climate

Table 20.7 Threshold T_{cutoff} for each scenario in each climate zone (original $T_{cutoff} + Dt_{summer}$ rounded to integers)

Climate zone	*Baseline*	*BaU 2020s*	*BaU 2050s*	*S550 2020s*	*S550 2050s*	*S750 2020s*	*S750 2050s*
Cold	16	17	18	17	17	17	17
Temperate	16	17	19	17	17	17	18
Warm/humid	29	29	31	29	29	29	30
Hot/dry	23	25	26	24	25	24	25

BaU Business-as-usual scenario.

scenarios (i.e. annual temperature distributions based on averages over 30 years). Deaths attributable to climate change were calculated as the change in proportion of temperature-attributable deaths (i.e. heat-attributable deaths plus cold-attributable deaths) for each climate scenario compared to the baseline climate. The $1° \times 1°$ resolution grid map of population distribution (Bos et al. 1994) was then overlaid on the maps of climate zones in a geographical information system (GIS), to estimate the proportion of the population in each subregion who live in each climate zone. The proportional changes in temperature-attributable deaths were therefore calculated by taking the average of the changes in each climate zone represented in the subregion, weighted by the proportion of the subregion's population living within that climate zone.

Adaptation

Acclimatization includes autonomous adaptation in the individual (physiological adaptation, changes in behaviour) and autonomous and planned population-level adaptations (public health interventions and changes in built environment). Acclimatization to warmer climate regimes is likely to occur in individuals and populations, given the rate of change in *mean* climate conditions currently projected by climate models. However, it is uncertain whether populations are able to adapt to non-linear increases in the frequency or intensity of daily temperature extremes (heat-waves). Even small increases in average temperature can result in large shifts in the frequency of extremes (IPCC 2001b; Katz and Brown 1992).

Few studies have attempted to incorporate acclimatization into future projections of temperature-related mortality (Kalkstein and Greene 1997), but all studies report that acclimatization would reduce potential increases in heat-related mortality. Our estimates incorporated an assumption regarding acclimatization of the populations to the changing climate that describes this reduced effect. We assumed that the threshold temperature (T_{cutoff}) is increased as populations adapt to a new

climate regime, reflecting physiological and behavioural acclimatization that can take place over the time-scale of decades. Changes in T_{cutoff} are region and scenario specific, as they reflect the rate of warming experienced. Therefore, they were assumed to be proportional to the projected change in average summer temperature (Dt_{summer}, equal to the mean of the three hottest months) from the climate scenario. The temperature–mortality relationships were assumed not to change over time; that is, populations biologically adapt to their new average temperatures, but remain equally vulnerable to departures from these conditions. We made no explicit adjustment for an effect of socioeconomic development and technological change on temperature-related mortality. The resulting relative risks are given in Table 20.8.

Short-term mortality displacement

Evidence suggests that the increase in mortality caused by high temperatures is partially offset by decreased deaths in a subsequent "rebound" period (Braga et al. 2001). This indicates that some of the observed increase in heat-related mortality may be displacement of deaths among those with pre-existing illness, which would have occurred soon in any case. However, this effect has not been quantified for temperature exposures and was not included in the model. The estimates are therefore used to calculate only attributable deaths, but not DALYs, as the estimate of attributable years of life lost was highly uncertain.

In subregions with predominantly temperate and cold climates, reductions in cold-related mortality are likely to be greater than increases in heat-related mortality. Therefore, all climate scenarios show a net benefit on mortality in these subregions, consistent with the IPCC conclusions described above. The effect of the adaptation assumption is to reduce relative risks and therefore net mortality.

Uncertainty estimates

The principal uncertainty in these estimates, and for all other health effects of climate change, relates to the extrapolation of a short-term climate-health relationship to the long-term effects of climate change. The degree to which this is a reasonable extrapolation relates to the degree to which populations will adapt to changing temperatures, both in terms of reducing the additional mortality attributable to heat, and the possible benefits of avoiding cold deaths. This outcome is unusual, in that the predicted health effects of climate change are negative in some regions, but positive in others. We therefore use slightly different terminology to describe the range of uncertainty around the estimates. The mid-range estimate was given by applying the model described above (i.e. making an adjustment for biological adaptation). The "high-impact" estimate assumes that there was no physiological or behavioural adaptation, and therefore no change in the dose–response relationship over time. This maximizes both positive and negative effects. The low-impact

Table 20.8 Central, low and high estimates of the relative risk of cardiovascular disease (all ages) for alternative climate scenarios relative to baseline climate[a]

		2000			*2001*			*2005*			*2010*			*2020*			*2030*		
Subregion	*Climate*[b]	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
AFR-D	2	1.001	1.000	1.002	1.001	1.000	1.002	1.002	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.006	1.004	1.000	1.008
	3	1.001	1.000	1.003	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005	1.004	1.000	1.008	1.005	1.000	1.009
	4	1.002	1.000	1.004	1.002	1.000	1.004	1.003	1.000	1.005	1.004	1.000	1.007	1.005	1.000	1.011	1.007	1.000	1.013
AFR-E	2	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.002	1.002	1.000	1.003	1.002	1.000	1.005	1.003	1.000	1.006
	3	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.006	1.003	1.000	1.007
	4	1.001	1.000	1.003	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005	1.004	1.000	1.008	1.005	1.000	1.010
AMR-A	2	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	1.000	1.000	0.999
	3	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	1.000	1.000	0.999
	4	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	1.000	1.000	0.999
AMR-B	2	1.001	1.000	1.001	1.001	1.000	1.001	1.001	1.000	1.002	1.001	1.000	1.002	1.002	1.000	1.003	1.002	1.000	1.004
	3	1.001	1.000	1.001	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005
	4	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.006	1.004	1.000	1.007
AMR-D	2	1.001	1.000	1.001	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005
	3	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005	1.003	1.000	1.007
	4	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.002	1.000	1.005	1.004	1.000	1.007	1.005	1.000	1.009
EMR-B	2	1.000	1.000	1.001	1.001	1.000	1.001	1.001	1.000	1.001	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004
	3	1.001	1.000	1.001	1.001	1.000	1.001	1.001	1.000	1.002	1.001	1.000	1.002	1.002	1.000	1.004	1.002	1.000	1.004
	4	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005	1.003	1.000	1.007
EMR-D	2	1.000	1.000	1.001	1.001	1.000	1.001	1.001	1.000	1.001	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004
	3	1.001	1.000	1.001	1.001	1.000	1.001	1.001	1.000	1.002	1.001	1.000	1.002	1.002	1.000	1.004	1.002	1.000	1.005
	4	1.001	1.000	1.002	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005	1.003	1.000	1.007

EUR-A	2	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999
	3	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.999
	4	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.999
EUR-B	2	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.999
	3	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.999
	4	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.998
EUR-C	2	1.000	1.000	0.999	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.998	0.999	1.000	0.998
	3	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.998	0.999	1.000	0.998
	4	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.998	0.999	1.000	0.997	0.998	1.000	0.997
SEAR-B	2	1.001	1.000	1.002	1.001	1.000	1.003	1.002	1.000	1.004	1.002	1.000	1.005	1.004	1.000	1.007	1.004	1.000	1.009
	3	1.001	1.000	1.003	1.002	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.006	1.004	1.000	1.009	1.005	1.000	1.011
	4	1.002	1.000	1.004	1.002	1.000	1.004	1.003	1.000	1.006	1.004	1.000	1.008	1.006	1.000	1.011	1.007	1.000	1.014
SEAR-D	2	1.001	1.000	1.002	1.001	1.000	1.002	1.002	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.006	1.004	1.000	1.008
	3	1.001	1.000	1.003	1.001	1.000	1.003	1.002	1.000	1.004	1.003	1.000	1.005	1.004	1.000	1.008	1.005	1.000	1.009
	4	1.002	1.000	1.004	1.002	1.000	1.004	1.003	1.000	1.005	1.004	1.000	1.007	1.005	1.000	1.011	1.007	1.000	1.013
WPR-A	2	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999
	3	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.999
	4	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	0.999	1.000	1.000	0.999	0.999	1.000	0.999	0.999	1.000	0.999
WPR-B	2	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000
	3	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000
	4	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000	1.000

[a] Note that for SEAR-B, there are no suitable studies of the relationship between temperature and cardiovascular disease-specific mortality; they are therefore based on relationships with all-cause mortality.

[b] 2 = s550, 3 = s750, 4 = unmitigated emissions.

estimate assumes complete adaptation to a changing climate, and therefore no change in the relative risk as the climate changes.

There is therefore a need for further time-series studies, applying a standard approach to populations living in as wide a range of climates as possible. Previous analyses have focused mainly on temperate zones, where the winter effects are greatest, potentially causing over-estimation of the reduced burden attributable to climate change (e.g. Martens 1998a). More analyses of temperature–mortality relationships are therefore required in tropical developing countries.

Such studies should attempt to estimate formally the degree to which adaptation may decrease mortality, and to which observed associations between climate and mortality reflect displaced rather than additional deaths. Finally, there is a need for greater investigation of the health burden of morbidity associated with temperature extremes, including, for example, inability to work in extreme temperatures.

3.7 Diarrhoeal disease

Diarrhoeal diseases are highly sensitive to climate, showing seasonal variations in numerous sites (Drasar et al. 1978). This observation is supported by regression analyses of the effects of seasonal and longer-term variation in a limited number of sites (Checkley et al. 2000; Singh et al. 2001). The results of a literature search on the associations between diarrhoeal disease and climate are shown in Appendix B, Table B.2. The climate-sensitivity of diarrhoeal disease is consistent with observations of the direct effects of climate variables on the causative agents. Temperature and relative humidity have a direct influence on the rate of replication of bacterial and protozoan pathogens, and on the survival of enteroviruses in the environment (Blaser et al. 1995). Rainfall may affect the frequency and level of contamination of drinking water (Curriero et al. 2001).

Quantitative relationships can be defined between climate variations and incidence, which can in turn be directly linked to the outputs of global climate change models. There are, however, challenges and uncertainties in estimating the magnitude of effects.

- The sites from which these relationships were defined cover only a small part of the spectrum of global climate variation. Different relationships may apply at higher or lower temperatures.
- The relative importance of different pathogens and modes of transmission (e.g. via water, food, insects or human–human contact) varies between locations, and is heavily influenced by level of sanitation (Black and Lanata 1995). As the pathogens are known to vary in their response to climate (e.g. Cook et al. 1990; Chaudhury et al. 1996), this will cause uncertainty in extrapolating temperature relationships from local studies to other regions with different levels of development.

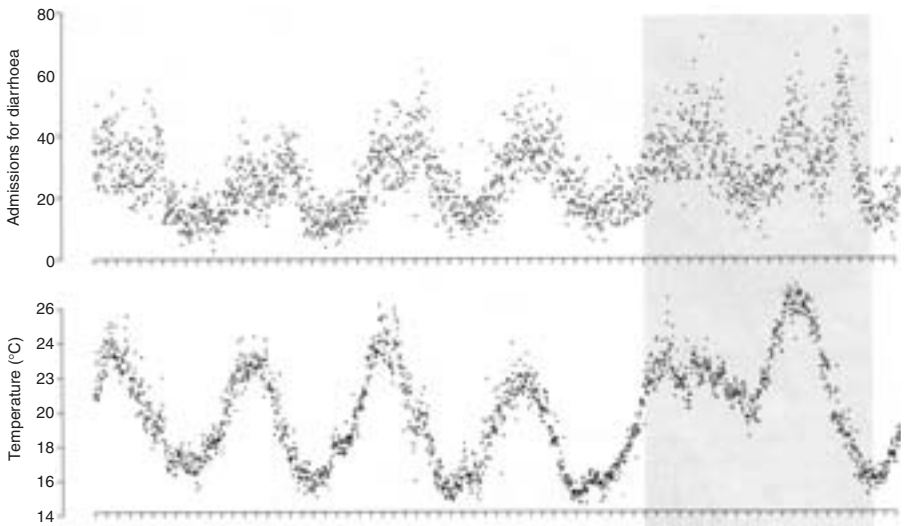
- Pathogens vary in the severity of clinical symptoms, and the likelihood that they will be reported to health services (e.g. Wheeler et al. 1999). Therefore, climate–disease relationships derived only from passive reporting may differ from those based on other methods of surveillance.
- While several studies describe climate effects on particular diarrhoea pathogens (e.g. Eberhard et al. 1999; Konno et al. 1983; Purohit et al. 1998), these cannot be used directly to estimate effects on diarrhoeal disease without information on: (i) their relative contribution to overall disease incidence, and (ii) equivalent data on climate sensitivity and relative prevalence for all other diarrhoea pathogens.
- Despite convincing evidence on the effect of extreme rainfall on waterborne outbreaks of diarrhoea, even in highly developed countries (Curriero et al. 2001), this cannot easily be generalized to the total burden of diarrhoeal disease without information on the relative contribution of such outbreaks to overall diarrhoea incidence.
- Rainfall effects on overall diarrhoea (where observed) are non-linear, and cannot easily be extrapolated to other regions.

In order to minimize these uncertainties, we restricted our estimates to the effect of increasing temperatures on the incidence of all-cause diarrhoea reported to health services (i.e. without attempting to make separate estimates for different pathogens, transmission routes, severities, or for the more complex associations with rainfall). There are important residual uncertainties related to the extrapolation of temperature relationships from specific study sites to others with different temperature regimes and levels of development, and climate effects on reported diarrhoea compared to the true burden of disease. Projections of increasing frequency of extreme wet seasons are very large (e.g. two to five times increase in the regions analysed; Palmer and Ralsanen 2002), and extreme precipitation is associated with increased diarrhoea in both developed (Curriero et al. 2001) and developing (Singh et al. 2001) countries.

Graded maps of 3.75° longitude by 2.5° latitude resolution showing the change in temperature under the alternative scenarios were overlaid with 0.5°×0.5° resolution maps of predicted population distributions for the 2020s and 2050s in a GIS. The GIS was used to calculate the average change in exposure (temperature) for each population grid-cell.

Although seasonality of diarrhoeal disease is well recognized, the quantitative relationship between climate and overall diarrhoea incidence has only been explicitly measured in two studies. Both studies described relationships with all-cause diarrhoea, that is, specific pathogens were not differentiated. Checkley et al. (2000) used time-series analysis to correlate measurements of temperature and relative humidity against daily hospital admissions at a single paediatric diarrhoeal

Figure 20.4 Hospitalizations for diarrhoea (upper line) correspond closely with temperature (lower line) at a clinic in Lima, Peru



Note: Shaded region corresponds to the 1997–1998 El Niño event.

Source: Checkley et al. (2000).

disease clinic in Lima, Peru (Figure 20.4) for just under 6 years. A total of 57 331 admissions were recorded during the study. The analysis showed a 4% (95% CI 2–5%) increase in admissions for each degree centigrade increase in temperature during the hotter months, and a 12% (95% CI 10–14%) per degree centigrade increase in the cooler months, averaging at an 8% (95% CI 7–9%) per degree centigrade increase over the course of the study. During the 1997–1998 El Niño period there was an additional increase in admissions above that expected on the basis of pre-El Niño temperature relationships, but no association with relative humidity independent of temperature. No rainfall during that period.

In the Checkley et al. study, exposure (climate) data were recorded at local meteorological stations, and can be considered to have negligible measurement error at the population level. The analysis independently controlled for seasonal variations and long-term trends, imparting high confidence to the observed effect of temperature on the outcome recorded. The positive correlation with temperature is also biologically plausible, as a high proportion of diarrhoea cases in many tropical developing countries are caused by bacteria, entamoeba and protozoa (Black

and Lanata 1995), which are favoured by high temperatures. The principle limitations of the study by Checkley et al. are that the outcome recorded may not be representative of climate effects on: (i) less severe disease (i.e. not requiring hospitalization) or more severe disease (diarrhoeal deaths), or (ii) disease in adults rather than children.

Singh et al. (2001) used similar time-series analyses to correlate monthly reported incidence of diarrhoea throughout Fiji against variations in temperature and rainfall, after allowing for the effects of seasonal variation and long-term trend. The study covered the period between 1978 and 1998, with an average of some 1000 reported cases for each of the 228 study months. Reported incidence increased by approximately 3% (95% CI 1.2–5.0%) for each degree centigrade increase in temperature, by 2% (95% CI 1.5–2.3%) per unit increase in rainfall above average rainfall conditions (5×10^{-5} kg/m^2 per min), and by 8% per unit decrease below average conditions. The pattern is supported by a positive geographical correlation between temperature and incidence in 18 Pacific Island countries (Singh et al. 2001). Climate measurements were from a 2.5°×2.5° cell of a global gridded data set corresponding to Fiji. The use of monthly averages of climate data from this large geographical area may not have reflected the full range of climate exposures of the population; this would have introduced random error and decreased sensitivity. Low rainfall may force use of contaminated water, while high rainfall may contaminate water through flooding. The major limitations of this study are lack of a clear clinical or laboratory definition for diarrhoea, and lack of information on the age distribution of cases.

We are not aware of any similar studies of climate effects on all-cause diarrhoea in developed regions, although studies have been carried out on some subsets of total incidence. Bentham (1997) showed that the incidence of food poisoning, usually caused by bacteria, increased by approximately 9% per degree centigrade in England and Wales. Konno et al. (1983) demonstrated a non-linear inverse relationship between rotavirus infection and temperature in Japan. The relative importance of pathogens that thrive at lower temperatures appears to be greater in populations with higher standards of living, who have access to clean water and sanitation and for whom there is no clear and consistent evidence for peaks in all-cause diarrhoea in warmer months. This is in contrast to the situation of less well-off populations, where diarrhoea is usually more common in warmer, wetter months, as well demonstrated by clear summer peaks of diarrhoea in black, but not white, infants in Johannesburg during the 1970s (Robins-Browne 1984).

For this assessment we defined developing countries as those with per capita incomes lower than the richer of the two study countries (Fiji) in the year 2000—approximately US$ 6000/year in 1990 US dollars. For such countries, we applied a dose–response relationship of 5% increase in diarrhoea incidence per degree centigrade increase in temperature, to

both sexes and all age groups. This is consistent with the relationships derived from the two studies described above. We chose 5% rather than the arithmetic mean of the constants from the two studies (5.5%) for two reasons: (i) to avoid giving a false impression of precision based on only two estimates, each with their own confidence intervals, and (ii) in order to be conservative.

Although the confidence intervals around the estimates from the individual studies are relatively small, these clearly cover only a small range of climatic and socioeconomic environments. As described above, Checkley et al. (2000) showed that even in a single socioeconomic setting the temperature dependence of diarrhoeal disease may vary across the temperature range or, less plausibly, with non-climatic seasonal variations. This introduces uncertainty when extrapolating such a single relationship. There is also potential bias: if the temperature-responsiveness is indeed greater at low temperatures, extrapolation of an average value will tend to underestimate effects in areas that are on average colder, and overestimate in hotter regions. However, the average annual temperatures experienced by the residents of the areas to which we extrapolated this relationship was 20.3 °C, as calculated by averaging the temperatures in each 1 °×1 °grid-cell weighted by the population. This is in the mid-range of the temperatures experienced throughout the year in Lima (16–26 °C), and cooler than those in the capital of Fiji (23–27 °C) (World Climate 2002). Our extrapolation tended towards being conservative, but with significant uncertainty. Therefore, we place a wide uncertainty range (0–10%) on this value.

As there are no such studies published for developed regions, we made the assumption that overall diarrhoea incidence in richer countries is insensitive to climate change, that is, 0% (–5 to +5%) change per degree centigrade temperature change. Relative risks for each country under each scenario were calculated by multiplying the projected increase in temperature by the relevant exposure–response value. The quoted estimate for each subregion is the population-weighted average of the relative risks for each country in the subregion (Table 20.9). In addition to changes in baseline diarrhoeal disease over time, we assumed that the climate sensitivity of diarrhoea in developing countries will decrease as they become better off. For projections of relative risks for years after 2000, we used projections of future changes in GDP (WHO/EIP/GPE, unpublished data, 2000) to apply the relationship used above—that is, overall diarrhoea incidence does not respond to temperature in any country that attains a per capita GDP of at least US$ 6000/year. Relative risks for each time point were then calculated as above.

The quantitative estimates in this analysis were highly sensitive to the exposure–response relationship, around which there is substantial uncertainty due to the very small number of analysed time-series. The estimates could be rapidly improved by analysis of climate exposure–response relationships from sites from a wider climatic and

Table 20.9 Central, low and high estimates of the relative risk of diarrhoea for alternative climate scenarios relative to baseline climate

		2000			2001			2005			2010			2020			2030		
Subregion	*Climate*[a]	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
AFR-D	2	1.01	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.04	1.00	1.08	1.05	0.99	1.10
	3	1.02	1.00	1.04	1.02	1.00	1.04	1.03	1.00	1.05	1.04	1.00	1.07	1.05	1.00	1.11	1.06	0.99	1.13
	4	1.02	1.00	1.05	1.02	1.00	1.05	1.03	1.00	1.07	1.05	1.00	1.09	1.07	1.00	1.14	1.08	0.99	1.16
AFR-E	2	1.01	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.04	0.99	1.08	1.05	0.99	1.11
	3	1.02	1.00	1.04	1.02	1.00	1.04	1.02	1.00	1.05	1.03	1.00	1.07	1.05	0.99	1.10	1.06	0.99	1.13
	4	1.02	1.00	1.05	1.03	1.00	1.05	1.03	1.00	1.07	1.04	0.99	1.09	1.06	0.99	1.13	1.08	0.99	1.16
AMR-A	2	1.00	0.99	1.02	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.05	1.00	0.95	1.06
	3	1.00	0.99	1.02	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.05	1.00	0.94	1.06
	4	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.04	1.00	0.94	1.06	1.00	0.93	1.08
AMR-B	2	1.00	0.99	1.02	1.00	0.99	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.04	1.00	0.95	1.05
	3	1.00	0.99	1.02	1.00	0.99	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.05	1.00	0.94	1.06
	4	1.00	0.98	1.02	1.00	0.98	1.03	1.00	0.97	1.03	1.00	0.96	1.04	1.00	0.94	1.06	1.00	0.92	1.08
AMR-D	2	1.01	1.00	1.03	1.01	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.05	1.02	0.98	1.06	1.02	0.96	1.07
	3	1.02	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.05	1.03	1.00	1.06	1.02	0.97	1.07	1.02	0.96	1.08
	4	1.02	1.00	1.04	1.02	1.00	1.05	1.03	1.00	1.06	1.04	1.00	1.08	1.03	0.97	1.09	1.02	0.95	1.10
EMR-B	2	1.01	1.00	1.03	1.01	1.00	1.03	1.02	0.99	1.04	1.02	0.99	1.06	1.00	0.95	1.05	1.00	0.94	1.06
	3	1.01	1.00	1.03	1.01	1.00	1.03	1.02	0.99	1.04	1.02	0.99	1.05	1.00	0.95	1.05	1.00	0.94	1.06
	4	1.02	0.99	1.04	1.02	0.99	1.05	1.03	0.99	1.06	1.04	0.99	1.08	1.00	0.93	1.08	1.00	0.91	1.09
EMR-D	2	1.02	1.00	1.03	1.02	1.00	1.04	1.02	1.00	1.05	1.03	1.00	1.06	1.05	1.00	1.10	1.06	1.00	1.12
	3	1.02	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.05	1.03	1.00	1.07	1.05	1.00	1.10	1.06	1.00	1.13
	4	1.03	1.00	1.05	1.03	1.00	1.06	1.04	1.00	1.08	1.05	1.00	1.10	1.08	1.00	1.15	1.09	1.00	1.19

continued

Table 20.9 Central, low and high estimates of the relative risk of diarrhoea for alternative climate scenarios relative to baseline climate (*continued*)

		2000			2001			2005			2010			2020			2030		
Subregion	*Climate*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
EUR-A	2	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.95	1.05	1.00	0.94	1.06
	3	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.97	1.03	1.00	0.95	1.05	1.00	0.94	1.06
	4	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.04	1.00	0.94	1.06	1.00	0.92	1.08
EUR-B	2	1.01	0.99	1.03	1.01	0.99	1.03	1.01	0.99	1.04	1.02	0.98	1.05	1.02	0.97	1.07	1.01	0.94	1.07
	3	1.01	0.99	1.03	1.01	0.99	1.03	1.01	0.99	1.04	1.02	0.98	1.05	1.02	0.97	1.07	1.01	0.94	1.08
	4	1.01	0.99	1.03	1.01	0.99	1.04	1.02	0.98	1.05	1.02	0.98	1.06	1.02	0.96	1.09	1.01	0.93	1.09
EUR-C	2	1.02	1.00	1.03	1.02	1.00	1.04	1.01	0.98	1.03	1.01	0.98	1.04	1.01	0.96	1.06	1.00	0.94	1.07
	3	1.02	1.00	1.03	1.02	1.00	1.04	1.01	0.98	1.03	1.01	0.98	1.04	1.01	0.96	1.06	1.00	0.94	1.07
	4	1.02	1.00	1.04	1.02	1.00	1.05	1.01	0.98	1.04	1.01	0.97	1.06	1.01	0.95	1.08	1.00	0.92	1.08
SEAR-B	2	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.00	1.03	1.00	0.98	1.03	1.00	0.97	1.04	1.00	0.95	1.05
	3	1.01	1.00	1.03	1.01	1.00	1.03	1.02	0.99	1.04	1.00	0.97	1.04	1.00	0.95	1.05	1.00	0.94	1.06
	4	1.02	1.00	1.04	1.02	1.00	1.04	1.02	0.99	1.05	1.00	0.96	1.04	1.00	0.94	1.07	1.00	0.92	1.08
SEAR-D	2	1.02	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.05	1.03	1.00	1.07	1.05	1.00	1.10	1.06	1.00	1.13
	3	1.02	1.00	1.04	1.02	1.00	1.04	1.03	1.00	1.06	1.04	1.00	1.08	1.06	1.00	1.12	1.07	1.00	1.15
	4	1.03	1.00	1.05	1.03	1.00	1.06	1.04	1.00	1.08	1.05	1.00	1.10	1.07	1.00	1.15	1.09	1.00	1.19
WPR-A	2	1.00	0.99	1.01	1.00	0.99	1.01	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.04	1.00	0.95	1.05
	3	1.00	0.99	1.01	1.00	0.99	1.01	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.96	1.04	1.00	0.95	1.05
	4	1.00	0.98	1.02	1.00	0.98	1.02	1.00	0.97	1.03	1.00	0.96	1.04	1.00	0.94	1.06	1.00	0.93	1.07
WPR-B	2	1.01	1.00	1.03	1.01	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.00	0.96	1.05	1.00	0.95	1.06
	3	1.01	1.00	1.03	1.01	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.00	0.96	1.05	1.00	0.95	1.06
	4	1.02	1.00	1.05	1.03	1.00	1.05	1.03	1.00	1.07	1.05	1.00	1.09	1.00	0.93	1.08	1.01	0.92	1.09

[a] 2 = s550, 3 = s750, 4 = unmitigated emissions.

socioeconomic spectrum. Future studies should also explicitly measure the degree to which economic development and improved levels of sanitation influence vulnerability to the effects of climate variation on diarrhoeal disease.

3.8 Malnutrition

Multiple biological and social factors affect the incidence of malnutrition, but one of the fundamental determinants is the availability of staple foods. Climate change may affect this through the balance of the (broadly negative) effects of changes in temperature and precipitation, and (broadly positive) effects of higher CO_2 levels on yields of major food crops (e.g. IPCC 1996; Rosenzweig and Parry 1994; see also Appendix B, Table B.4). These effects are likely to vary markedly with geography: productivity is projected to increase in higher-latitude producers such as Canada and the United States, but to decrease closer to the equator. The global food trade system may be able to absorb these effects at the global level. However, climate change can be expected to have significant effects on food poverty in some regions, owing to variation in both productivity and in economic capacity to cope (Parry et al. 1999).

Crop models have been validated at 124 sites in 18 countries over a wide range of environments (e.g. Otter-Nacke et al. 1986). However, estimation of changes in food availability (and by inference malnutrition) requires additional analyses of the geographical variation in effects on food production, and of food trade patterns. Only one modelling group (Parry et al. 1999) has integrated a basic physiological model of climate change effects on region-specific crop production with food trade models, in order to make projections of the numbers of people actually at risk of hunger. All results presented here were based on the model described in Parry et al. (1999) and other work by the same group.

As for other potential effects of climate change, there is considerable uncertainty over the degree to which current relationships, such as those between climate and crops, and food trade systems will remain constant over time. The most important uncertainties probably relate to the ability of the world food trade system to adapt to changes in production (Dyson 1999; Waterlow et al. 1998). Although these are the most complete models currently available, they do not describe the likely effect of climate change on more complex pathways, such as animal husbandry, or the relative importance of fruit and vegetable production. These in turn may affect micronutrient (e.g. vitamin A, iodine, iron and zinc) deficiency.

Global distribution of temperature, rainfall and CO_2 were mapped for each of the alternative scenarios, as described above. Climate dose–response relationships have been defined for yields of major grain cereals and soybean, which account for 85% of world cereal exports.

Effects of temperature and precipitation, and the beneficial effects of higher CO_2 levels, have been defined using the IBSNAT-ICASA dynamic crop growth models (IBSNAT 1989). The exposure distributions described above were applied to these crop growth models, and the derived yield functions were extrapolated to other crops and regions on the basis of agro-climatic similarity.

These crop yield estimates are used as inputs for the Basic Linked System world food trade model. This consists of a linked series of 40 national and regional food models, representing food production, the effects of market forces and government policies on prices and trade, and trends in agricultural, economic and technological conditions over time (see Fischer et al. 1988 for a full description).

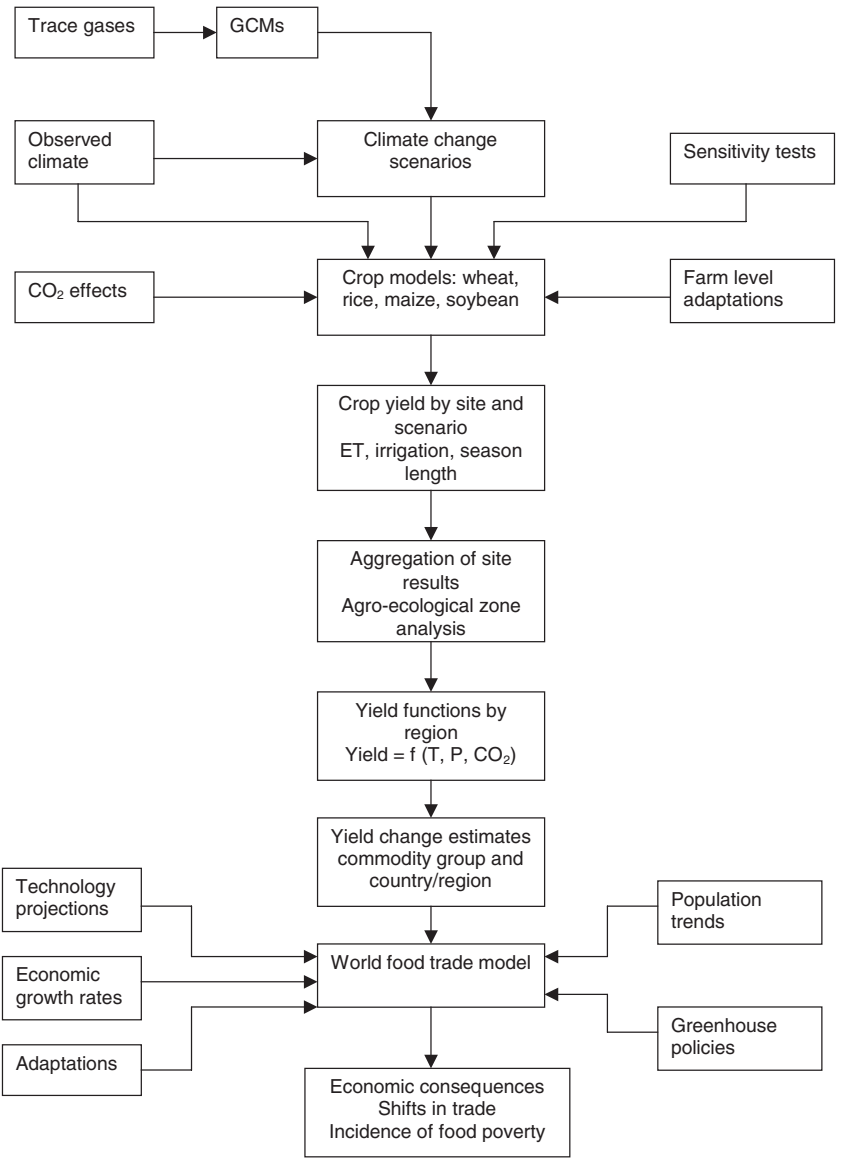
The model is represented schematically in Figure 20.5. Principal characteristics of this model are:

- no major changes in the political or economic context of world food trade or in food production technology;
- population growth to occur following the World Bank mid-range estimate (World Bank 1994) i.e. 10.7 billion by the 2080s;
- GDP to accumulate as projected by EMF14 (Energy Modeling Forum 1995);
- a 50% trade liberalization in agriculture is introduced gradually by 2020.

The model results in an estimation of national food availability. This is used to generate an estimate of per capita food availability in each country, assuming that this food is distributed among the population following a skewed (beta) distribution. The final model output is the number or proportion of the population in each subregion who do not have access to sufficient food to maintain a basal metabolic rate of 1.4, which is the Food and Agriculture Organization of the United Nations' (FAO) definition of undernourishment (FAO 1987).

The model generates outputs for continents made up principally of vulnerable developing countries (thus excluding China and the former Soviet Union, countries in North America, and in western and eastern Europe). As these model continents do not map directly on to the sub-regions, we generated estimates for each subregion by calculating the proportion of the population that lives within each continent in the food availability model. Where more than 90% of the subregion population live within a single model continent, we quoted the model estimate for that continent. Otherwise an average was calculated, weighted by the distribution of the subregion population among the various continents. While the subregions mapped reasonably well on to the climate change/food availability model continents, the aggregation meant that some of the geographical variation in vulnerability was lost. Most notably, despite severe problems in some countries, EUR-C was assumed

Figure 20.5 Key elements of the crop yields and world food trade study



ET Evapotranspiration.

Source: Rosenzweig et al. (1993).

not to suffer from malnutrition as it lies within the "developed" European continent of the food availability model.

Preliminary analysis correlating estimates for the 1990s at the level of the model regions (data not shown) indicated that the model output was positively related to more direct measures of malnutrition, such as the incidence of underweight, and stunting and wasting in children aged <5 years, as measured by the WHO Global Database on Child Growth and Malnutrition (WHO 2002). The aggregation of the food availability model means that this correlation was based on only a small number of independent data points. We therefore did not attempt to make any quantitative estimate of these relationships. Instead, the relative risk of the incidence of energy shortfall (Table 20.10) was interpreted as being directly proportional to the relative risk of suffering from the risk factor "underweight". The relative risk estimates were therefore applied to all diseases affected by the underweight risk factor (see chapter 2). These include diarrhoea and malaria. We therefore assumed that these diseases are affected in two distinct ways by climate change—through meteorological effects on the pathogens and vectors, and through increased susceptibility of the human population due to undernutrition.

In common with most climate change impact assessments, the published studies do not quote uncertainties around the various relationships in the model, either separately or aggregated. Small variations in the initial conditions of a single climate model (the HadCM2 ensemble) generated only slight variations in projections of crop production and incidence of food shortfall. Using a different climate model (HadCM3), however, generated markedly different projections (Parry et al. 1999). These comparisons relate to only part of the possible error, as they do not address uncertainties in the trade and social components of the model.

From the published descriptions of the model, there is no reason to assume that the estimates generated were systematically biased either upward or downward. In the absence of formal sensitivity analyses of the complete model, however, uncertainty estimates are arbitrary. We presented the relative risks generated above as mid-range estimates, with the upper and lower range covering a complete adaptation to any changes in agricultural output (i.e. no change in risk), to a doubling of the estimate of the relative risk calculated above. However, this uncertainty range should be treated with caution. Priorities for the future are investigations of:

- variation in output when using a wider range of food production models as inputs to the food trade/availability models;
- sensitivity of estimates to the various climate scenarios;
- sensitivity analyses to estimate uncertainty around the exposure–response relationships;

Table 20.10 Central, low and high estimates of the relative risk of malnutrition for alternative climate scenarios relative to baseline climate

		2000			2001			2005			2010			2020			2030		
Subregion	*Climate*[a]	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
AFR-D	2	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.03	1.00	1.06
	3	1.01	1.00	1.03	1.01	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.05	1.04	1.00	1.08	1.04	1.00	1.09
	4	1.01	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.04	1.02	1.00	1.04
AFR-E	2	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.03	1.00	1.06
	3	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.04	1.02	1.00	1.05	1.04	1.00	1.07	1.04	1.00	1.08
	4	1.01	1.00	1.01	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.04	1.02	1.00	1.05
AMR-A	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
AMR-B	2	1.02	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.05	1.03	1.00	1.06	1.05	1.00	1.09	1.05	1.00	1.10
	3	1.03	1.00	1.07	1.04	1.00	1.07	1.05	1.00	1.10	1.07	1.00	1.13	1.10	1.00	1.20	1.11	1.00	1.22
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.01	1.00	1.00	1.00
AMR-D	2	1.02	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.05	1.03	1.00	1.06	1.05	1.00	1.09	1.05	1.00	1.10
	3	1.03	1.00	1.07	1.04	1.00	1.07	1.05	1.00	1.10	1.07	1.00	1.13	1.10	1.00	1.20	1.11	1.00	1.22
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.01	1.00	1.00	1.00
EMR-B	2	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.03	1.00	1.06
	3	1.02	1.00	1.04	1.02	1.00	1.04	1.03	1.00	1.06	1.04	1.00	1.08	1.06	1.00	1.12	1.06	1.00	1.13
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.01	1.00	1.00	1.00
EMR-D	2	1.02	1.00	1.04	1.02	1.00	1.05	1.03	1.00	1.07	1.04	1.00	1.09	1.07	1.00	1.13	1.07	1.00	1.15
	3	1.03	1.00	1.07	1.04	1.00	1.07	1.05	1.00	1.10	1.07	1.00	1.13	1.10	1.00	1.20	1.11	1.00	1.22
	4	1.02	1.00	1.05	1.03	1.00	1.05	1.04	1.00	1.07	1.05	1.00	1.10	1.07	1.00	1.15	1.08	1.00	1.16

continued

Table 20.10 Central, low and high estimates of the relative risk of malnutrition for alternative climate scenarios relative to baseline climate (*continued*)

		2000			*2001*			*2005*			*2010*			*2020*			*2030*		
Subregion	*Climate*[a]	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
EUR-A	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
EUR-B	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
EUR-C	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
SEAR-B	2	1.02	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.05	1.03	1.00	1.06	1.05	1.00	1.09	1.05	1.00	1.10
	3	1.03	1.00	1.06	1.03	1.00	1.07	1.04	1.00	1.09	1.06	1.00	1.12	1.09	1.00	1.18	1.10	1.00	1.19
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.01	1.00	1.00	1.01	1.00	1.00	1.01
SEAR-D	2	1.04	1.00	1.07	1.04	1.00	1.08	1.06	1.00	1.11	1.07	1.00	1.15	1.11	1.00	1.22	1.12	1.00	1.25
	3	1.05	1.00	1.10	1.06	1.00	1.12	1.08	1.00	1.16	1.10	1.00	1.21	1.16	1.00	1.31	1.17	1.00	1.35
	4	1.05	1.00	1.10	1.06	1.00	1.11	1.08	1.00	1.15	1.10	1.00	1.20	1.15	1.00	1.31	1.17	1.00	1.33
WPR-A	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
WPR-B	2	1.00	1.00	1.01	1.00	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.02	1.01	1.00	1.02
	3	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.03	1.03	1.00	1.05	1.03	1.00	1.05
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	0.99	1.00	1.00	0.99	1.00	1.00

[a] 2 = s550, 3 = s750, 4 = unmitigated emissions.

- back-casting of climate/hunger relationships for verification of model accuracy;
- a finer geographical (e.g. country level) breakdown of the outputs of malnutrition models;
- correlations between model outputs and health outcomes at a high spatial resolution; and
- investigation of the synergistic effects of water availability and poverty on malnutrition.

3.9 Disasters caused by extreme weather events: coastal floods, inland floods and landslides

Natural disasters are ultimately a function of both the average and degree of variability of weather conditions, which are further modulated by multiple aspects of population vulnerability such as topography, housing quality and early warning systems (Alexander 1993; McMichael et al. 1996). They are therefore likely to be directly affected by the observed and predicted trend towards increasingly variable weather.

In addition to the axiomatic link between extreme weather events and weather-related deaths and injuries, there is strong statistical evidence that long-term weather cycles (e.g. the ENSO quasi-periodic cycle) correlate with incidence of deaths and injuries attributable to natural disasters (Bouma et al. 1997a; Kovats et al. 1999). The evidence for increased frequency of different categories of extreme events in the past, and the likelihood of changes in the future (Table 20.1), has been strengthened by recent demonstrations of increases in the frequency of large floods during the 20th century (Milly et al. 2002). This evidence is reinforced by projections of several-fold increases in the frequency of what are currently considered extreme wet seasons, for various regions over the world, using a range of climate models (Palmer and Ralsanen 2002).

Based on this, we presented estimates of consequences of increasing frequency of coastal flooding caused by sea-level rise, and inland flooding and landslides caused by increased frequency of extreme precipitation events, which are described by IPCC as very likely (90–99% probability) to increase in many areas under climate change. We did not attempt to estimate the effects of changing frequency and intensity of wind storms, owing to inconsistencies between models (IPCC 2001b) and a lack of quantitative projections of changes in exposure under different climate change scenarios. Our estimates excluded the direct effect of thermal extremes (e.g. heatstroke, increased risk of cardiovascular disease), which are dealt with elsewhere. We also excluded any potential longer-term health consequences arising through mechanisms such as population displacement, economic damage to public health infrastructure, increased risk of infectious disease epidemics and mental illness (Jovel 1989; Menne et al. 1999; WHO 1992).

Climate change is likely to have different effects on the frequency of coastal versus inland floods. Changes in the frequency of coastal floods were defined using published models (Hoozemans and Hulsburgen 1995; Nicholls et al. 1999) that estimate change in sea level for each climate scenario. These changes were applied to topographical and population distribution maps to estimate the change in incidence of exposure to flooding by subregion. The predictions did not account for changes in frequency of storm surges. The global model used here has been shown to be relatively accurate in validations against more detailed assessments at the national level (summarized in Nicholls et al. 1999).

Inland floods and landslides are not affected by sea-level rise, but will be influenced by any increase in the frequency of intense precipitation. Despite the clear causal link, this relationship is poorly researched (Pielke 1999), and has not previously been modelled as a health exposure. There are no published analyses of global relationships between intensity of precipitation, the likelihood of a declared disaster, or the magnitude of health consequences. Clearly, at the local level the frequency of health effects will be determined by the temporal distribution of rainfall (i.e. not only by the average amount of rain over an extended period, but by the peak amount falling in a week, day or hour), and modulated by topography and social aspects of vulnerability (Kundzewicz and Kaczmarek 2000). However, in the absence of detailed data on these variables and their effects, we made the *a priori* assumption that flood frequency is proportional to the frequency with which monthly rainfall exceeds the 1 in 10 year limit (i.e. upper 99.2% CI) of the baseline climate. We also assumed that determinants of vulnerability are distributed evenly throughout the population of a subregion—so that the change in relative risk of health consequences is proportional to the per capita change in risk of experiencing such an extreme event. For each $1° \times 1°$ population grid-cell, we estimated the 99.2% upper CI for the "baseline climate" using means and standard deviations derived from the 1961–1990 averages for each month of the year. Using equivalent data for future scenarios and time points, we estimated the change in frequency with which such a "1 in 10 year event" occurs.

The difference (in standard deviates of the new distribution) between the new mean and the previously defined "1 in 10 year limit" is given by:

$$((X_1 + 2.41 \times u_1) - X_2)/u_2$$

where X_1, u_1=mean and intra-annual deviation from 1961–1990 and X_2, u_2=mean and intra-annual deviation under new scenario.

The probability that this difference will be exceeded in any one month under the new distribution was taken from probability tables. This was divided by the frequency of occurrence under the baseline scenario (=0.008) to give the relative frequency of exceeding the 1 in 10 year limit

for each future scenario. Results were weighted by the population in each cell, and averaged across the countries in each subregion. The final measure of exposure was therefore the relative frequency with which each person in a subregion experiences 1 in 10 year rainfall events.

The process of estimating the disease burden of this change in frequency differs from that for other climate-sensitive health outcomes, as flood effects do not have a specific GBD code. Relative risks should therefore be applied to estimates of health consequences such as deaths and injuries *attributable to these climate events* under baseline climate (i.e. rather than change in total incidence of this outcome).

The EM-DAT database records numbers of deaths and injuries attributed to each natural disaster reported by the media or aid agencies in the last 100 years. Disasters are defined as events that resulted in at least one of the following conditions: (i) >10 people killed, (ii) >200 injured, (iii) a call for international assistance. Although this is the best comprehensive data source available on the current health consequences of natural disasters, all such sources may be subject to underreporting (Noji 1997). Estimates of attributable burden of disease derived from these figures are therefore conservative.

Individual events in the database were classified as inland or coastal floods on the basis of geography, or descriptions of events in the database. Total numbers of deaths for each class of event were summed for subregions. Although the EM-DAT database also records the number of injuries in flooding events, these are not included in this assessment, as they are considered particularly unreliable for floods (Guha-Sapir, personal communication, 2002). The effects of events that could not be identified as inland or coastal were assigned in proportion to the distribution of consequences of classified events in each subregion. The annual incidence of flood death under baseline climate conditions was estimated by dividing the annual average over the last 20 years by the subregional population in 1990.

These baseline incidence rates alter over time, depending on the balance between factors that decrease vulnerability (particularly improving flood defences as populations become richer), and those which increase vulnerability (particularly increasing population density in coastal zones and other flood-prone areas). Baseline estimates for future years were therefore adjusted as far as possible for these effects. For coastal flooding, the effects of projected changes in population distribution in relation to coastline and improving coastal defences in line with GDP were incorporated in the model of Nicholls et al. (1999). The baseline estimates of the incidence of deaths and injuries in years after 1990 were therefore scaled by the ratio of the model projections for numbers of people flooded in each year compared to that in 1990 (Table 20.11).

Such vulnerability effects have not been explicitly modelled for inland flooding. However, Yohe and Tol (2002) have carried out a cross-

Table 20.11 Annual incidence of deaths per 10 000 000 population caused by coastal floods, in the absence of climate change

Subregion	*1980–1999*	*2000*	*2001*	*2005*	*2010*	*2020*	*2030*
AFR-D	0	0	0	0	0	0	0
AFR-E	0	0	0	0	0	0	0
AMR-A	0	0	0	0	0	0	0
AMR-B	2.00	1.59	1.56	1.43	1.29	1.08	0.96
AMR-D	0.40	0.41	0.41	0.41	0.41	0.41	0.34
EMR-B	0	0	0	0	0	0	0
EMR-D	0	0	0	0	0	0	0
EUR-A	0	0	0	0	0	0	0
EUR-B	0	0	0	0	0	0	0
EUR-C	0.10	0.11	0.11	0.12	0.12	0.14	0.15
SEAR-B	0.10	0.11	0.11	0.11	0.12	0.12	0.11
SEAR-D	1.20	1.39	1.40	1.47	1.54	1.69	1.78
WPR-A	0.10	0.10	0.10	0.10	0.10	0.11	0.11
WPR-B	0.90	0.98	0.99	1.03	1.07	1.15	1.22

Source: EM-DAT (2002) for the period1980–1999. Estimates are based on changing GDP for other time points (see text).

sectional analysis of the effect of per capita income on the incidence of death due to all natural disasters (as reported in the EM-DAT database) for the period 1990–2000. They conclude that increasing wealth has a protective effect, best described by:

$$\text{Ln (proportion of population killed/decade)} = 4.7271 - 0.3858 \text{ (Ln GDP per capita)}$$

The effect of income is marginally non-significant at the 5% level ($P<0.07$), generic to all natural disasters and does not take account of the magnitude of the physical hazard. However, this is likely to introduce noise rather than bias in the relationship, and the relationship represents the only published basis for projection of the protective effects of economic development. The relationship is therefore applied to future projections of GDP (WHO/EIP/GPE, unpublished data, 2000). Our projected baseline incidence of deaths for years after 1990 were scaled by the ratio of the projections of deaths due to all natural disasters in that year divided by the estimate for 1990 (Table 20.12).

Some evidence from studies of a small number of earthquakes (Beinin 1981) and famines (Rivers 1982) suggests that women and young children are more vulnerable than men to the acute effects of natural disasters. However, there are insufficient data to derive subregional estimates

Table 20.12 Annual incidence of deaths per 10 000 000 population caused by inland floods and landslides, in the absence of climate change

Subregion	*1980–1999*	*2000*	*2001*	*2005*	*2010*	*2020*	*2030*
AFR-D	2.7	2.7	2.7	2.7	2.6	2.4	2.2
AFR-E	6.5	6.6	6.6	6.5	6.4	6.0	5.4
AMR-A	2.2	2.1	2.1	2.0	1.9	1.8	1.6
AMR-B	52.2	48.4	48.1	46.6	44.8	41.0	36.9
AMR-D	52.1	49.0	48.7	47.2	45.4	41.6	37.4
EMR-B	14.9	13.8	13.7	13.4	13.1	12.1	11.0
EMR-D	32.2	30.9	30.6	29.5	28.2	25.6	23.0
EUR-A	1.3	1.2	1.2	1.1	1.1	0.9	0.8
EUR-B	8.9	9.2	9.1	8.7	8.2	7.4	6.6
EUR-C	1.2	1.4	1.4	1.3	1.2	1.1	1.0
SEAR-B	9.9	8.2	8.1	7.5	6.8	5.8	5.1
SEAR-D	20.3	17.7	17.5	16.5	15.4	13.5	12.0
WPR-A	3.7	3.3	3.3	3.0	2.7	2.3	2.0
WPR-B	13.8	10.6	10.4	9.6	8.7	7.4	6.5

Source: EM-DAT (2002) for the period 1980–1999. Estimates are based on changing GDP for other time points (see text).

of the relative vulnerability of different age groups and sexes to the consequences of flooding: we therefore made the assumption, that all age groups are equally at risk.

The models presented here for coastal flooding assumed that protection evolves over time in proportion to projected increases in GDP. The mid-range estimates presented therefore incorporated an effect of increasing wealth, not only in the baseline estimates, but assumed the same proportional change in the relative risks (i.e. as described by Yohe and Tol 2002). This accounted for the effect of increasing wealth not only in reducing the likely health consequences of "baseline" (i.e. climate change independent floods), but also providing better adaptive capacity for increases driven by climate change. The mid-range estimates did not include any further adjustments for biological/behavioural adaptation to increased flood risk (Table 20.13).

As for other health outcomes of climate change, the only published sensitivity analyses relate to an unmitigated emissions scenario applied to the HadCM2 model with four slightly varying sets of initial conditions, and a comparison with the same emissions scenario run on the HadCM3 model. These resulted in almost no difference in model outputs over the time-scale considered in this assessment. Uncertainties in the model relate to the degree and manner to which individuals respond to

Table 20.13 Central, low and high estimates of the relative risk of death in coastal floods for alternative climate scenarios relative to baseline climate

		2000			*2001*			*2005*			*2010*			*2020*			*2030*		
Subregion	*Climate*[a]	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
AFR-D	2	1.07	1.04	1.14	1.07	1.04	1.15	1.09	1.05	1.18	1.11	1.05	1.21	1.13	1.06	1.26	1.44	1.22	1.89
	3	1.07	1.04	1.15	1.08	1.04	1.16	1.10	1.05	1.19	1.11	1.06	1.23	1.14	1.07	1.27	1.48	1.24	1.96
	4	1.10	1.05	1.20	1.11	1.05	1.21	1.13	1.06	1.25	1.15	1.07	1.30	1.18	1.09	1.36	1.64	1.32	2.29
AFR-E	2	1.06	1.03	1.12	1.06	1.03	1.13	1.07	1.04	1.15	1.08	1.04	1.17	1.09	1.05	1.19	1.12	1.06	1.25
	3	1.06	1.03	1.13	1.07	1.03	1.14	1.08	1.04	1.16	1.09	1.04	1.18	1.10	1.05	1.20	1.13	1.07	1.27
	4	1.09	1.04	1.17	1.09	1.04	1.18	1.10	1.05	1.21	1.12	1.06	1.23	1.13	1.07	1.27	1.18	1.09	1.35
AMR-A	2	1.03	1.02	1.06	1.04	1.02	1.07	1.06	1.03	1.11	1.08	1.04	1.16	1.12	1.06	1.25	1.13	1.06	1.25
	3	1.03	1.02	1.07	1.04	1.02	1.08	1.06	1.03	1.12	1.09	1.04	1.17	1.13	1.07	1.27	1.14	1.07	1.27
	4	1.05	1.03	1.10	1.06	1.03	1.12	1.09	1.04	1.17	1.12	1.06	1.24	1.18	1.09	1.37	1.19	1.09	1.38
AMR-B	2	1.24	1.12	1.48	1.27	1.13	1.54	1.38	1.19	1.75	1.52	1.26	2.04	1.84	1.42	2.69	1.90	1.45	2.81
	3	1.26	1.13	1.51	1.29	1.14	1.57	1.40	1.20	1.80	1.55	1.28	2.11	1.90	1.45	2.80	1.96	1.48	2.93
	4	1.34	1.17	1.68	1.38	1.19	1.75	1.53	1.26	2.05	1.73	1.36	2.46	2.18	1.59	3.37	2.27	1.64	3.54
AMR-D	2	2.07	1.53	3.14	2.15	1.58	3.30	2.45	1.73	3.91	2.77	1.89	4.55	3.27	2.14	5.55	3.58	2.29	6.17
	3	2.14	1.57	3.28	2.23	1.61	3.45	2.55	1.77	4.10	2.89	1.94	4.78	3.42	2.21	5.84	3.76	2.38	6.52
	4	2.50	1.75	4.00	2.62	1.81	4.23	3.04	2.02	5.08	3.49	2.24	5.97	4.19	2.59	7.38	4.64	2.82	8.28
EMR-B	2	1.14	1.07	1.29	1.16	1.08	1.32	1.22	1.11	1.45	1.31	1.16	1.63	1.52	1.26	2.03	1.53	1.27	2.06
	3	1.15	1.08	1.31	1.17	1.08	1.34	1.24	1.12	1.48	1.33	1.17	1.67	1.55	1.28	2.10	1.57	1.28	2.13
	4	1.20	1.10	1.40	1.22	1.11	1.45	1.31	1.16	1.63	1.44	1.22	1.88	1.72	1.36	2.45	1.75	1.37	2.50
EMR-D	2	1.53	1.27	2.07	1.57	1.28	2.14	1.68	1.34	2.36	1.79	1.39	2.58	1.94	1.47	2.88	3.01	2.01	5.02
	3	1.57	1.28	2.14	1.61	1.30	2.21	1.73	1.36	2.45	1.84	1.42	2.68	2.00	1.50	3.00	3.18	2.09	5.36
	4	1.75	1.38	2.50	1.80	1.40	2.59	1.96	1.48	2.91	2.11	1.55	3.21	2.32	1.66	3.63	3.91	2.46	6.82

EUR-A	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.09	1.04	1.18
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.10	1.05	1.20
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.14	1.07	1.29
EUR-B	2	1.57	1.28	2.13	1.64	1.32	2.27	1.95	1.48	2.91	2.45	1.73	3.91	4.05	2.52	7.09	4.78	2.89	8.55
	3	1.60	1.30	2.21	1.68	1.34	2.36	2.02	1.51	3.03	2.55	1.77	4.10	4.24	2.62	7.49	5.02	3.01	9.05
	4	1.79	1.40	2.59	1.89	1.45	2.79	2.34	1.67	3.68	3.04	2.02	5.08	5.27	3.14	9.54	6.31	3.65	11.61
EUR-C	2	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.01	1.02	1.02	1.01	1.03	1.02	1.01	1.04	1.03	1.01	1.06
	3	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.01	1.03	1.02	1.01	1.03	1.02	1.01	1.05	1.03	1.02	1.06
	4	1.01	1.01	1.02	1.01	1.01	1.03	1.02	1.01	1.03	1.02	1.01	1.04	1.03	1.02	1.06	1.04	1.02	1.08
SEAR-B	2	1.11	1.06	1.22	1.12	1.06	1.24	1.15	1.08	1.31	1.19	1.09	1.38	1.24	1.12	1.49	1.28	1.14	1.56
	3	1.12	1.06	1.24	1.13	1.06	1.26	1.16	1.08	1.33	1.20	1.10	1.40	1.26	1.13	1.52	1.30	1.15	1.59
	4	1.16	1.08	1.32	1.17	1.09	1.34	1.22	1.11	1.43	1.26	1.13	1.53	1.34	1.17	1.68	1.39	1.20	1.78
SEAR-D	2	1.01	1.00	1.02	1.01	1.01	1.02	1.01	1.01	1.03	1.02	1.01	1.03	1.02	1.01	1.04	1.03	1.01	1.05
	3	1.01	1.01	1.02	1.01	1.01	1.02	1.01	1.01	1.03	1.02	1.01	1.03	1.02	1.01	1.04	1.03	1.01	1.05
	4	1.01	1.01	1.03	1.01	1.01	1.03	1.02	1.01	1.04	1.02	1.01	1.04	1.03	1.01	1.05	1.04	1.02	1.07
WPR-A	2	1.01	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.02	1.01	1.01	1.03	1.02	1.01	1.04	1.03	1.01	1.06
	3	1.01	1.00	1.01	1.01	1.00	1.01	1.01	1.01	1.02	1.02	1.01	1.03	1.02	1.01	1.05	1.03	1.02	1.06
	4	1.01	1.00	1.02	1.01	1.01	1.02	1.02	1.01	1.03	1.02	1.01	1.04	1.03	1.02	1.06	1.04	1.02	1.09
WPR-B	2	1.01	1.01	1.02	1.01	1.01	1.03	1.02	1.01	1.04	1.02	1.01	1.05	1.03	1.02	1.06	1.04	1.02	1.07
	3	1.01	1.01	1.03	1.01	1.01	1.03	1.02	1.01	1.04	1.02	1.01	1.05	1.03	1.02	1.07	1.04	1.02	1.08
	4	1.02	1.01	1.04	1.02	1.01	1.04	1.03	1.01	1.05	1.03	1.02	1.07	1.04	1.02	1.09	1.05	1.03	1.10

[a] 2 = s550, 3 = s750, 4 = unmitigated emissions.

the increased risk (Hoozemans et al. 1993). The lower estimates therefore assumed that 90% of the risk could be avoided either by highly efficient coastal defences or individual adaptations. The higher estimates assumed no adaptation either with increasing GDP or individual level measures.

The model for inland flooding was subject to the same uncertainty over adaptive responses. Both the baseline and relative risks were assumed to change with GDP, as described for coastal flooding. As outlined above, however, the uncertainty is also greater for a hazard driven by the magnitude and temporal variation of precipitation (which varies considerably between climate models), rather than the more predictable process of temperature-driven sea level rise. This consideration is particularly important as only one climate model was used in this assessment. Although the relative risks estimated by our method (e.g. 5.53 for our estimates for EUR-A by the year 2030 under an unmitigated emissions scenario) were broadly comparable to estimates of changes in frequency of extreme wet seasons generated using multi-climate model analyses (fivefold for northern Europe for the period 2060–2080; Palmer and Ralsanen 2002), more formal analyses would be necessary to give more accurate estimates. We therefore gave a larger uncertainty range around these predictions than for coastal flooding, by assuming a 50% greater exposure and no adaptation with GDP for the high estimate, and no increase in risk under any scenario for the lower estimate (Table 20.14). As for the other outcomes, this uncertainty range should be interpreted with caution.

The potential health consequences of changing frequency and intensity of extreme weather events are surprisingly poorly researched. Substantial improvements in assessment could be made through better estimates of the current health impacts of natural disasters, which suffer from poor baseline data and probably severe underreporting, particularly in developing countries (Noji 1997). Analyses could also be greatly improved by geo-referencing and more detailed descriptions of disasters to allow differentiation of inland and coastal events, detailed analysis of the relationships between intensity of precipitation and health effects, and projections of future precipitation at higher temporal and spatial resolution, using output from a range of climate models. In order to generate better uncertainty estimates, formal sensitivity analyses of the contributions of each model parameter to the final uncertainty estimates are required.

Finally, it should be stressed that the estimates given here represent the immediate acute consequences of natural disasters, which are likely to be only one component of the total attributable disease burden. Other outcomes of these natural disasters need to be considered, such as the probability of outbreaks of water, vector- and rodent-borne diseases, the effects of sequential disasters on both public health defences and stability of natural ecosystems limiting disease outbreaks (Epstein 1999), and

Table 20.14 Central, low and high estimates of the relative risk of death in inland floods/landslides for alternative climate scenarios relative to baseline climate

		2000			*2001*			*2005*			*2010*			*2020*			*2030*		
Subregion	*Climate*[a]	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
AFR-D	2	1.40	1.00	1.60	1.44	1.00	1.66	1.60	1.00	1.89	1.77	1.00	2.19	2.09	1.00	2.79	2.30	1.00	3.13
	3	1.30	1.00	1.45	1.32	1.00	1.50	1.44	1.00	1.68	1.58	1.00	1.89	1.83	1.00	2.34	1.99	1.00	2.64
	4	1.18	1.00	1.27	1.20	1.00	1.30	1.26	1.00	1.41	1.35	1.00	1.54	1.50	1.00	1.81	1.66	1.00	2.08
AFR-E	2	1.37	1.00	1.54	1.40	1.00	1.60	1.53	1.00	1.83	1.71	1.00	2.10	2.03	1.00	2.64	2.30	1.00	3.18
	3	1.26	1.00	1.41	1.30	1.00	1.45	1.39	1.00	1.60	1.53	1.00	1.81	1.75	1.00	2.22	1.99	1.00	2.65
	4	1.22	1.00	1.33	1.24	1.00	1.36	1.33	1.00	1.50	1.43	1.00	1.66	1.62	1.00	1.99	1.86	1.00	2.44
AMR-A	2	4.19	1.00	6.00	4.66	1.00	6.51	5.76	1.00	8.50	7.33	1.00	11.0	10.5	1.00	16.00	11.5	1.00	18.69
	3	3.62	1.00	5.10	4.00	1.00	5.50	4.90	1.00	7.15	6.19	1.00	9.19	8.77	1.00	13.29	9.66	1.00	15.61
	4	3.02	1.00	4.18	3.34	1.00	4.50	4.03	1.00	5.77	5.03	1.00	7.36	7.03	1.00	10.54	7.99	1.00	12.79
AMR-B	2	1.43	1.00	1.69	1.50	1.00	1.75	1.66	1.00	2.04	1.88	1.00	2.37	2.26	1.00	3.06	2.60	1.00	3.67
	3	1.59	1.00	1.98	1.72	1.00	2.07	1.93	1.00	2.46	2.25	1.00	2.94	2.78	1.00	3.91	3.18	1.00	4.65
	4	1.56	1.00	1.89	1.66	1.00	1.98	1.87	1.00	2.34	2.13	1.00	2.79	2.63	1.00	3.67	3.03	1.00	4.39
AMR-D	2	1.53	1.00	1.83	1.60	1.00	1.92	1.79	1.00	2.25	2.06	1.00	2.65	2.52	1.00	3.48	2.92	1.00	4.20
	3	1.32	1.00	1.50	1.36	1.00	1.56	1.48	1.00	1.75	1.63	1.00	2.01	1.92	1.00	2.50	2.26	1.00	3.10
	4	1.36	1.00	1.57	1.42	1.00	1.62	1.54	1.00	1.84	1.73	1.00	2.13	2.03	1.00	2.70	2.40	1.00	3.33
EMR-B	2	1.61	1.00	1.99	1.71	1.00	2.10	1.98	1.00	2.49	2.29	1.00	2.98	2.83	1.00	3.97	3.20	1.00	4.63
	3	1.85	1.00	2.40	2.01	1.00	2.53	2.37	1.00	3.09	2.82	1.00	3.79	3.57	1.00	5.19	4.04	1.00	6.03
	4	1.89	1.00	2.44	2.05	1.00	2.58	2.41	1.00	3.15	2.88	1.00	3.87	3.64	1.00	5.31	4.04	1.00	6.01
EMR-D	2	2.32	1.00	3.09	2.51	1.00	3.28	3.01	1.00	4.12	3.66	1.00	5.16	4.78	1.00	7.24	5.29	1.00	8.17
	3	2.07	1.00	2.68	2.23	1.00	2.85	2.62	1.00	3.52	3.14	1.00	4.36	4.05	1.00	6.04	4.56	1.00	6.94
	4	2.13	1.00	2.77	2.29	1.00	2.94	2.70	1.00	3.64	3.26	1.00	4.53	4.20	1.00	6.30	4.68	1.00	7.15

continued

Table 20.14 Central, low and high estimates of the relative risk of death in inland floods/landslides for alternative climate scenarios relative to baseline climate (*continued*)

		2000			2001			2005			2010			2020			2030		
Subregion	*Climate*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
EUR-A	2	2.13	1.00	2.83	2.34	1.00	3.01	2.67	1.00	3.75	3.44	1.00	4.66	3.99	1.00	6.48	5.30	1.00	8.28
	3	2.13	1.00	2.83	2.34	1.00	3.03	2.69	1.00	3.76	3.44	1.00	4.68	4.01	1.00	6.51	5.27	1.00	8.20
	4	2.22	1.00	2.98	2.44	1.00	3.18	2.82	1.00	3.97	3.64	1.00	4.95	4.24	1.00	6.93	5.53	1.00	8.65
EUR-B	2	1.31	1.00	1.44	1.32	1.00	1.48	1.42	1.00	1.66	1.55	1.00	1.89	1.79	1.00	2.32	2.32	1.00	3.22
	3	1.64	1.00	1.92	1.67	1.00	2.01	1.88	1.00	2.38	2.15	1.00	2.83	2.66	1.00	3.75	3.16	1.00	4.65
	4	1.35	1.00	1.53	1.38	1.00	1.57	1.50	1.00	1.78	1.66	1.00	2.04	1.94	1.00	2.56	2.46	1.00	3.45
EUR-C	2	1.33	1.00	1.42	1.30	1.00	1.45	1.39	1.00	1.62	1.52	1.00	1.83	1.75	1.00	2.25	2.45	1.00	3.42
	3	2.05	1.00	2.37	2.00	1.00	2.50	2.26	1.00	3.04	2.68	1.00	3.72	3.49	1.00	5.08	4.31	1.00	6.46
	4	1.70	1.00	1.90	1.66	1.00	1.99	1.84	1.00	2.37	2.11	1.00	2.82	2.67	1.00	3.72	3.45	1.00	5.04
SEAR-B	2	1.33	1.00	1.59	1.43	1.00	1.65	1.56	1.00	1.89	1.71	1.00	2.19	2.01	1.00	2.77	2.51	1.00	3.60
	3	1.61	1.00	2.11	1.81	1.00	2.23	2.04	1.00	2.67	2.34	1.00	3.22	2.89	1.00	4.33	3.57	1.00	5.37
	4	1.30	1.00	1.54	1.40	1.00	1.60	1.50	1.00	1.81	1.65	1.00	2.08	1.92	1.00	2.62	2.39	1.00	3.37
SEAR-D	2	1.21	1.00	1.36	1.26	1.00	1.41	1.34	1.00	1.56	1.45	1.00	1.74	1.65	1.00	2.10	1.73	1.00	2.22
	3	1.10	1.00	1.20	1.14	1.00	1.21	1.19	1.00	1.29	1.24	1.00	1.38	1.33	1.00	1.57	1.39	1.00	1.68
	4	1.05	1.00	1.11	1.08	1.00	1.11	1.09	1.00	1.15	1.13	1.00	1.20	1.18	1.00	1.30	1.21	1.00	1.36
WPR-A	2	1.46	1.00	1.80	1.58	1.00	1.87	1.73	1.00	2.19	1.95	1.00	2.59	2.35	1.00	3.37	2.91	1.00	4.29
	3	1.20	1.00	1.33	1.24	1.00	1.38	1.31	1.00	1.50	1.40	1.00	1.68	1.56	1.00	2.01	2.04	1.00	2.80
	4	1.30	1.00	1.50	1.36	1.00	1.54	1.45	1.00	1.75	1.59	1.00	1.99	1.85	1.00	2.49	2.32	1.00	3.28
WPR-B	2	1.17	1.00	1.35	1.26	1.00	1.38	1.31	1.00	1.51	1.42	1.00	1.69	1.58	1.00	2.04	1.88	1.00	2.50
	3	1.22	1.00	1.41	1.29	1.00	1.45	1.39	1.00	1.62	1.49	1.00	1.83	1.70	1.00	2.23	2.00	1.00	2.70
	4	1.40	1.00	1.60	1.44	1.00	1.66	1.60	1.00	1.89	1.77	1.00	2.19	2.09	1.00	2.79	2.30	1.00	3.13

[a] 2 = s550, 3 = s750, 4 = unmitigated emissions.

longer-term effects such as post-traumatic stress after floods (e.g. Phifer 1990) and of population displacement through coastal flooding. More importantly, it is necessary to expand the range of natural disasters beyond those considered in this chapter. There is an obvious causal chain between apparently increasing variability in precipitation frequency of droughts and their associated health consequences, particularly food shortages and possible famine (UNDMTP 1990; WHO/PTC 1995). While these impacts may be expected to increase under climate change, no models have yet been developed to link climate scenarios, frequency of drought, and associated health effects, so that quantitative estimation of climate change effects is not currently possible. Given the global importance of drought-related disease (WHO/EHA 1998) and the effectiveness of early preparation and rapid response for avoiding health impacts (Gupta 2000; WHO/EHA 2002), this is clearly a priority area for research.

3.10 Vector-borne diseases

Viruses, bacteria, protozoa and helminths transmitted by biting insects and other intermediate hosts are among the most important causes of ill-health in tropical regions (WHO 2000b). The climate sensitivity of such diseases has long been recognized (e.g. Celli 1933), and knowledge of the relationships has been used to help predict epidemics of vector-borne diseases since at least the early years of the last century (e.g. Christophers 1911; MacDonald 1957). More recently, there have been many quantitative studies of the effects of climate variables on the population biology of vectors and pathogens in the laboratory (e.g. reviews by Martens 1998b; Massad and Forattini 1998), and on spatial and temporal variations in vector abundance and disease incidence in the field (review by Kovats et al. 2000a). The results of a literature review are shown in Appendix B, Tables B.5 and B.6.

This climate sensitivity has prompted several studies correlating long-term changes in vector distribution (Lindgren et al. 2000) or disease incidence (e.g. Bouma et al. 1996; Loevinsohn 1994) with local climate trends, which apparently reflect global climate change. However, many of the inferences about resulting health consequences have been called into question, due to concurrent local changes in crucial non-climatic factors, such as human behaviour, disease reporting or control programmes (Hay et al. 2002; Mouchet 1998; Randolph 2001; Reiter 2001).

As for other health effects, it is unlikely that simply correlating long-term trends in disease against trends in climate will soon (or perhaps ever) give unequivocal evidence of the effects of gradual climate change. As climate varies naturally between years, long time-series (i.e. two to three decades) will be needed for statistical tests of association in long-term trends. It is almost inevitable that non-climatic determinants of risk will also change over such long periods, either obscuring or offering

alternative explanations for any effects of climate change. In addition, reliable multi-decadal time-series for vector-borne diseases in developing countries are rare, and it is problematic to interpret analyses of a few data sets as evidence of a general global pattern. Recent reviews therefore suggest that a general effect of climate change on vector-borne disease, while suspected and possible, is highly dependent on other non-climatic factors that act at a small scale (IPCC 2001a; Kovats et al. 2001, Reiter 2001, Reiter et al. 2003).

Despite the practical problems in making direct correlations with recent trends, the extreme climate sensitivity of vector-borne diseases means that it is almost inevitable that they will respond in some way to climate change, in some settings and to some degree. The most reliable basis for estimating such changes should come from information on the relationships between variations in climate and disease in either the past or present. Several studies have used such data to model the effect of predicted climate change on either the distribution of vector-borne diseases, and/or measures of risk within existing or predicted newly endemic areas. The data, techniques and assumptions used in the various analyses are reviewed in detail in later sections.

Whatever the quality of the data and modelling techniques used for predictions, they will remain contingent upon other determinants of disease. Socioeconomic conditions, control programmes, human immunity and the specific combinations of climate variables required by particular vector species or transmission cycles also affect disease incidence, and may be more important than global climate trends, particularly at small spatial scales (Mouchet and Manguin 1999; Randolph et al. 2000; Reiter 2001; Rogers and Randolph 2000; Sutherst 1998).

The IPCC has reviewed the observed and predicted effects of climate variability and change in the context of the other factors listed above (IPCC 2001a). On balance, the IPCC concludes that climate change is likely to expand the geographical distribution of several vector-borne diseases, including malaria, dengue and leishmaniasis to higher altitudes (high confidence) and higher latitudes with limited public health defences (medium/low confidence), and to extend the transmission seasons in some locations (medium/high confidence). For some vector-borne diseases in some locations, climate change may decrease transmission by reductions in rainfall or temperatures too high for transmission (medium/low confidence).

The associations between climate and infectious diseases have also been reviewed by the United States National Research Council (National Research Council 2001b). The review highlights the climate sensitivity of vector-borne diseases, and describes some studies modelling the potential consequences of future climate change on vector-borne diseases. The report re-emphasizes the need for caution when making future projections, and stresses the importance of public health provision in mitigating increases in incidence driven by climate change. No judgement is

made on the likely effects of future climate change on specific diseases, owing to the limited evidence.

Aside from analyses of the potential effect of gradual changes in average climate conditions, predicted increases in climate variability, including possible increases in the frequency and intensity of El Niño events (IPCC 2001b), may also affect vector-borne diseases. There is evidence that the El Niño cycle causes inter-annual variations in disease incidence in several areas (e.g. Bouma et al. 1997b). However, even in these sites, other determinants such as seasonal variations and control programmes exert a larger influence, and epidemic cycles also occur in areas where El Niño has little or no effect on climate, apparently driven by gradual post-epidemic waning of herd immunity (Hay et al. 2000). As the effect of climate variability differs so greatly between sites, global projections of the associations described so far are unlikely to be informative.

Modelling of specific diseases

Most modelling of the effects of climate change has focused on malaria, and to a lesser extent dengue. These are therefore the only vector-borne diseases considered here. Some preliminary modelling work has been carried out on schistosomiasis (Martens et al. 1997), but this is based on a relatively small data set and has not been validated against current distributions. Randolph and Rogers (2000) have also modelled the potential effects of climate change on tick-borne encephalitis (TBE) in Europe, demonstrating that increased temperatures are likely to reduce the endemic range. Although TBE is a relatively small public health problem in global terms, and was not covered in this assessment, this study does demonstrate that climate change may potentially decrease, rather than increase, the transmission of some diseases. Effects on other major vector-borne diseases have been investigated either qualitatively (e.g. Carcavallo and Curto de Casas 1996 for American trypanosomiasis) or in terms of distribution of vectors rather than human disease (e.g. Rogers and Packer 1993 for African trypanosomiasis).

Falciparum malaria

Falciparum malaria is unusual in that several research groups have independently modelled the relationships between climate and disease distribution (Appendix B, Table B.5). The models used can be broadly classified as structural/biological, based on aggregating the effect of climate on the individual components of the disease transmission cycle, or "statistical", derived from direct correlations between geographic or temporal variations in climate, and associated variations in disease incidence or distribution, either in the present or recent past.

Published biological models for the global distribution of falciparum malaria use laboratory data to define the relationship between temperature and the extrinsic incubation period of the parasite, and therefore

the probability of completing development during the lifetime of the mosquito and completing the transmission cycle (Martin and Lefebvre 1995). Later models incorporate temperature effects on the survival probability and biting frequency of mosquitoes (Jetten et al. 1996; Martens et al. 1995a, 1995b, 1999). In these later models, the various temperature-dependent relationships are aggregated into the entomological version of the equation for R_o, the number of cases arising from each new case in a completely susceptible population (Anderson and May 1991; Dye 1992; Garrett-Jones 1964). Because of the lack of data on several key parameters, these are set as biologically plausible constants, allowing the calculation of the critical vector density required for sustainable disease transmission (i.e. $R_o > 1$). This threshold is lower under more suitable (generally warmer) climate conditions. The inverse of the critical density threshold, the "transmission potential", is used as a relative measure of transmission intensity under different climatic conditions. The models also assume a threshold level of transmission potential required to sustain transmission, which allows the identification of areas that are climatically suitable for transmission under both observed and projected climate scenarios.

These studies highlight the extreme climate sensitivity of several stages of the malaria transmission cycle. By aggregating these effects into a single measure related to R_o, they demonstrate that even small temperature increases could potentially cause large relative increases in risk, particularly at the edges of the distribution where temperature may be a limiting factor. They also suggest that those areas climatically suitable for *Plasmodium falciparum* transmission could expand substantially.

While valid for their original purpose as sensitivity analyses for relative changes in risk, these models are not ideal for defining the most probable changes in either geographical distribution or disease burden within endemic areas. Both outputs require the calculation of absolute rather than relative values of R_o, so as to identify areas where $R_o > 1$, allowing disease transmission to persist. In these incomplete biological models, such calculations are partly dependent on parameter values that are arbitrarily defined in the absence of empirical data (Rogers and Randolph 2000). As the models are based on temperature relationships derived from the laboratory, they also rely on the assumption that meteorological station data accurately represent the climatic conditions that mosquitoes and parasites experience in the field—which disregards the possibility that vectors might exploit microhabitats that are very different from those in meteorological stations. Since the outputs from these models have not been validated against current disease distributions, they were not used in this assessment.

In the absence of data to generate complete biological models of all stages in the transmission cycle, an alternative approach is to use statistical relationships to define only the distributional limits of disease. Although this approach does not allow disaggregation of the specific

mechanisms driving the climate-sensitivity of vector-borne diseases, it is generally considered more objective than the use of incomplete biological models, in that model outputs are not dependent on arbitrarily defined parameter values.

The international MARA (Mapping malaria risk in Africa) collaboration generated a model that used a combination of biological and statistical approaches to define the limits of climate suitability for falciparum malaria in Africa (Craig et al. 1999). Laboratory data on the rate of development of falciparum parasites (Detinova 1962) and laboratory and localized field observations of temperature effects on mosquito survival (Haddow 1943; Jepson et al. 1947; Le Sueur 1991; Maharaj 1995) were used to define upper and lower thresholds for mean monthly temperatures, and winter minima, which would allow both mosquito survival and the completion of the parasite extrinsic incubation period during the lifetime of mosquitoes, thereby permitting transmission. Rainfall thresholds were defined by comparing regions with and without stable malaria transmission, which have similar temperature conditions but different precipitation profiles. In order to take account of uncertainty about the precise values of the upper and lower bounds of temperature and rainfall necessary for transmission, climatic conditions near to the thresholds were not defined as either entirely suitable or unsuitable. Instead, they were assigned a probability of suitability between 0 and 1, defined by a "fuzzy membership curve", which is assumed to follow a pre-specified sigmoidal shape between the plausible values for the upper and lower thresholds for each climate variable (e.g. a decreasing probability of suitability between mean temperatures of 32 and 40 °C). These relationships were applied to high-resolution interpolated maps of climate throughout Africa (Hutchinson et al. 1996) to define areas that meet all suitability conditions (i.e. both temperature and rainfall) throughout the continent. For validation, model outputs were visually compared with independent high-resolution maps of the edges of the distribution, based either on field surveys or expert opinion. The model showed a good fit to the observed distributions in both southern Africa and East Africa.

The main advantages of this approach are that the model:

- describes only the cut-offs for any level of transmission, rather than quantitative estimates of transmission risk: it therefore does not rely on arbitrarily defined parameter values to complete the R_o equation;
- represents uncertainty around the edges of the distribution;
- allows description of seasonal patterns of transmission, based on the suitability of individual months; and
- most importantly, has been compared with current and historical distribution maps which are apparently independent of the model building process.

The main caveats are:

- the reliance on laboratory data and a small number of field studies to define climate cut-offs;
- apparent subjectivity in at least one parameter estimate (the proportion of mosquitoes that need to survive the sporogonic cycle in order to maintain transmission, which defines the precise value of the lower temperature cut-off);
- the need to make an assumption about the shape of the "fuzzy membership curve"; and
- lack of systematic empirical validation (validation by visual comparison, rather than calculating diagnostic statistics).

While each of the assumptions can legitimately be questioned, the visual validation suggests that the data and assumptions used are at least reasonably accurate. Again, there are further caveats in using the model to try to describe the true global distribution of falciparum malaria (rather than just climatically suitable areas), either now or in the future. To use such models, it is necessary to make the assumptions that distributions vary directly with climate, without any interactive effect of control programmes, or socioeconomic conditions. The comparisons of model outputs with current data suggest that this is a reasonable assumption for most of sub-Saharan Africa, although control programmes have altered distributions in South Africa. The assumption is much less secure for other endemic regions, which are invariably richer.

The relationship between climate variables and the global distribution of malaria can also be defined in statistical terms. Rogers and Randolph (2000) converted WHO maps (WHO 1997) of the limits of reported malaria distribution in the 1990s into $0.5° \times 0.5°$ resolution grids, coding each cell as either endemic or non-endemic. These grid maps were overlaid on $0.5°$ grid maps describing climate surfaces for the period 1961–1990. A statistical model was generated by randomly selecting a subsample of 50% of the observations of disease presence or absence. First, grid-cells were assigned by applying *k*-means clustering to six groups based on climatic similarity, thereby allowing for potentially different climate–disease relationships in different ecological zones. Stepwise discriminant analysis was applied to find the combination of temperature, rainfall and humidity parameters that gave the greatest statistical differentiation between positive vs negative grid cells within each cluster. The model was then applied to the remaining 50% of the observations to assess model accuracy. The fit of the predicted distributions to the observed WHO malaria maps was found to be significantly better than for previous falciparum malaria models: 77.71% of grid cells correctly predicted for this model vs 75.79% for Martin and Lefebvre (1995) and 67.26% for Martens et al. (1999).

The principal advantage of this approach is that it is entirely data driven. The relationships between global climate and malaria distributions are defined using transparent statistical techniques, applied to both disease and climate data from throughout the globe. They therefore do not require either unsupported assumptions of parameter values in order to complete a biological model, or global extrapolation of data from a limited number of observations. In addition, by mapping the observed distribution rather than climatically suitable areas, such analyses do not require the assumption that distributions are defined only by climate.

Despite these advantages, the quality of the data available at the global scale places several limitations on global statistical models. WHO maps of observed distributions are based on a combination of field observations and expert opinion to draw "inclusive" boundaries of the extremes of distributions. In many cases the maps define large areas as endemic (e.g. all of sub-Saharan Africa north of South Africa, Namibia and Botswana), although significant areas are actually disease free, often apparently due to unsuitable climate. WHO maps (and therefore the statistical relationships) also do not differentiate between malaria caused by *P. falciparum* and *Plasmodium vivax*, which have quite different sensitivities to temperature (Detinova 1962; MacDonald 1957). It is unclear what effect these two simplifications may have on the climate sensitivity of the model.

For this assessment, projected changes in temperature and rainfall under each of the alternative climate scenarios relative to the baseline (1961–1990) climate were mapped at the resolution of the HadCM2 climate model (3.75° longitude by 2.5° latitude). Maps of future climate were then generated by adding these values to maps of baseline climate for the 1961–1990 climate at 0.5° resolution (0.05° resolution for the MARA malaria model in Africa; Hutchinson et al. 1996).

An adapted version of the MARA climate model described in detail above (Tanser et al. 2003) was used to generate the mid-range estimates for this assessment. This decision was based on the independent (though continental rather than global) verification, plus the fact that the model was developed and tested using data from throughout Africa (where the overwhelming majority of the burden of malaria currently occurs, and where accuracy is therefore most important), and the potential for developing projections of increased force of infection and disease incidence in the near future.

The model was applied to the global climate maps for baseline climate, and for the unmitigated emissions scenario for the 2020s and 2050s. Relative risk estimates presented here were the ratios of the projected population at risk (i.e. living in areas climatically suitable for >1 month falciparum malaria transmission per year) in each subregion under climate change, relative to the population at risk under the 1961–1990 climate. As an approximation, estimates for the s750 and s550

scenarios were derived proportionately by multiplying the relative risks under unmitigated climate change by the ratio of global temperature change under each scenario/change under unmitigated emissions (Table 20.15). This model gave considerably larger estimates of changes in population at risk than the statistical model of Rogers and Randolph (2000), which predicted approximately no overall change under an unmitigated emissions scenario by the 2050s. In the absence of further comparisons and formal uncertainty assessments, our lower range estimate therefore included the possibility of no change in risk in any subregion. The upper range estimate is a doubling of the mid-range estimate from the MARA model. We emphasize that given the difficulties in validating any specific model and its suitability for extrapolation to other subregions, the choice of MARA climate model over the other possible models was somewhat arbitrary.

In addition, the calculation of disease burdens requires estimates of change in incidence within each subregion, rather than population at risk. In the absence of models for changes in malaria incidence within endemic regions, we therefore made the assumption that relative changes in incidence will vary in direct relation to predicted changes in population at risk—that is, a doubling of the population at risk within a region will lead to a doubling of the clinical disease incidence.

These measures are related in broad terms: countries or regions with higher populations at risk tend to have higher incidence and disease burdens. However, this relationship is a crude generalization, as it assumes that these relationships will remain constant as the population at risk expands or contracts. This may lead to underestimation of effects, if there is an increase in transmission within already at-risk populations, driving up infection incidence. Alternatively, this relationship may overestimate risk, depending on the extent to which increasing vectorial capacity promotes herd immunity (Rogers et al. 2002), and causes first infections to occur earlier in life, when patients suffer less severe clinical symptoms for some diseases, potentially conferring immunity on the more clinically vulnerable older age-groups (Coleman et al. 2001; Snow and Marsh 1995). In addition, socioeconomic conditions and control programmes clearly influence vector-borne diseases. Future changes in these factors are likely to affect (and hopefully reduce) transmission, as they have done in some regions in the past (e.g. Jetten and Takken 1994; Reiter 2001). The role of adaptation was discussed in section 2.5.

By applying relative risks to baseline incidences of zero in some subregions, our assessment did not allow for the spread of disease from endemic subregions to non-endemic subregions. This is a reasonable, but conservative assumption: non-endemic subregions have better developed health systems and a less amenable socioeconomic environment, in addition to usually being cooler. These factors may protect against re-establishment of vector-borne disease transmission (IPCC 2001b; Kuhn et al. 2003), providing they are maintained.

Table 20.15 Central, low and high estimates of the relative risk of falciparum malaria for alternative climate scenarios relative to baseline climate

		2000			2001			2005			2010			2020			2030		
Subregion	*Climate*[a]	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
AFR-D	2	1.00	1.00	1.01	1.00	1.00	1.01	1.00	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.02	1.01	1.00	1.02
	3	1.00	1.00	1.01	1.00	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.02	1.01	1.00	1.02	1.01	1.00	1.03
	4	1.01	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.04	1.02	1.00	1.05
AFR-E	2	1.02	1.00	1.04	1.02	1.00	1.04	1.03	1.00	1.06	1.04	1.00	1.08	1.06	1.00	1.12	1.07	1.00	1.15
	3	1.02	1.00	1.05	1.03	1.00	1.05	1.04	1.00	1.07	1.05	1.00	1.10	1.07	1.00	1.15	1.09	1.00	1.18
	4	1.04	1.00	1.08	1.04	1.00	1.09	1.06	1.00	1.12	1.08	1.00	1.16	1.12	1.00	1.23	1.14	1.00	1.28
AMR-A	2	1.08	1.00	1.15	1.08	1.00	1.17	1.11	1.00	1.23	1.15	1.00	1.30	1.23	1.00	1.46	1.27	1.00	1.53
	3	1.09	1.00	1.19	1.10	1.00	1.20	1.14	1.00	1.28	1.19	1.00	1.37	1.28	1.00	1.56	1.33	1.00	1.65
	4	1.15	1.00	1.29	1.16	1.00	1.32	1.22	1.00	1.44	1.29	1.00	1.59	1.44	1.00	1.88	1.51	1.00	2.03
AMR-B	2	1.02	1.00	1.04	1.02	1.00	1.05	1.03	1.00	1.07	1.04	1.00	1.09	1.07	1.00	1.13	1.08	1.00	1.16
	3	1.03	1.00	1.05	1.03	1.00	1.06	1.04	1.00	1.08	1.05	1.00	1.11	1.08	1.00	1.16	1.10	1.00	1.19
	4	1.04	1.00	1.09	1.05	1.00	1.09	1.06	1.00	1.13	1.09	1.00	1.17	1.13	1.00	1.26	1.15	1.00	1.30
AMR-D	2	1.01	1.00	1.02	1.01	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.05	1.03	1.00	1.07	1.04	1.00	1.09
	3	1.01	1.00	1.03	1.02	1.00	1.03	1.02	1.00	1.04	1.03	1.00	1.06	1.04	1.00	1.08	1.05	1.00	1.10
	4	1.02	1.00	1.04	1.02	1.00	1.05	1.03	1.00	1.07	1.04	1.00	1.09	1.07	1.00	1.13	1.08	1.00	1.17
EMR-B	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
EMR-D	2	1.03	1.00	1.07	1.04	1.00	1.07	1.05	1.00	1.10	1.07	1.00	1.13	1.10	1.00	1.20	1.15	1.00	1.30
	3	1.04	1.00	1.08	1.04	1.00	1.09	1.06	1.00	1.12	1.08	1.00	1.16	1.12	1.00	1.24	1.19	1.00	1.37
	4	1.06	1.00	1.13	1.07	1.00	1.14	1.10	1.00	1.19	1.13	1.00	1.26	1.19	1.00	1.38	1.29	1.00	1.59

continued

Table 20.15 Central, low and high estimates of the relative risk of falciparum malaria for alternative climate scenarios relative to baseline climate (*continued*)

		2000			2001			2005			2010			2020			2030		
Subregion	*Climate*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*	*Mid*	*Low*	*High*
EUR-A	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
EUR-B	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
EUR-C	2	1.07	1.00	1.13	1.07	1.00	1.15	1.10	1.00	1.20	1.13	1.00	1.27	1.20	1.00	1.40	1.25	1.00	1.50
	3	1.08	1.00	1.16	1.09	1.00	1.18	1.12	1.00	1.25	1.16	1.00	1.33	1.25	1.00	1.49	1.31	1.00	1.61
	4	1.13	1.00	1.26	1.14	1.00	1.29	1.19	1.00	1.39	1.26	1.00	1.52	1.39	1.00	1.78	1.48	1.00	1.97
SEAR-B	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
SEAR-D	2	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.01	1.00	1.00	1.01
	3	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.01	1.01	1.00	1.01
	4	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.01	1.00	1.00	1.01	1.01	1.00	1.01	1.01	1.00	1.02
WPR-A	2	1.07	1.00	1.14	1.08	1.00	1.15	1.11	1.00	1.21	1.14	1.00	1.28	1.21	1.00	1.42	1.25	1.00	1.49
	3	1.09	1.00	1.17	1.09	1.00	1.19	1.13	1.00	1.26	1.17	1.00	1.34	1.26	1.00	1.52	1.30	1.00	1.60
	4	1.14	1.00	1.27	1.15	1.00	1.30	1.20	1.00	1.41	1.27	1.00	1.54	1.41	1.00	1.81	1.48	1.00	1.95
WPR-B	2	1.06	1.00	1.12	1.07	1.00	1.14	1.09	1.00	1.18	1.12	1.00	1.25	1.18	1.00	1.37	1.22	1.00	1.43
	3	1.08	1.00	1.15	1.08	1.00	1.17	1.11	1.00	1.23	1.15	1.00	1.30	1.23	1.00	1.45	1.26	1.00	1.53
	4	1.12	1.00	1.24	1.13	1.00	1.26	1.18	1.00	1.36	1.24	1.00	1.48	1.36	1.00	1.71	1.42	1.00	1.83

[a] 2 = s550, 3 = s750, 4 = unmitigated emissions.

Dengue

Several studies have explored the relationship between climate and the distribution of intensity of dengue transmission (see Appendix B, Table B.6). Most are derived from a series of biological models that relate climate variables to determinants of the population biology of *Aedes* vectors (Focks et al. 1993a, 1993b) and dengue transmission (Focks et al. 1995). Adaptations of these models to map climate relationships at the global scale (Jetten and Focks 1997; Martens et al. 1997; Patz et al. 1998) have used field and laboratory data to define the relationships between temperature and the length of the gonotrophic cycle (and the associated feeding frequency), larval weight (and therefore the need to take multiple feeds within a single cycle), and the extrinsic incubation period of dengue virus within the vector. Mosquito survival, human biting habit and the duration of human infectiousness are set as temperature-independent constants, with parameter values defined using field data from a number of sites.

As for the biological models of malaria, these relationships are aggregated into a simplified version of the R_o equation, excluding measures of vector abundance. This equation is again used to define the critical density threshold (the number of mosquitoes which would be required to maintain $R_o>1$), and its inverse, "transmission potential" or "transmission intensity". The model has been applied to local climate data in a series of sites, and the rank order of monthly predicted values of transmission potential showed good correspondence with the observed seasonal distribution of dengue cases (e.g. Pearson's $R=0.837$ in San Juan, Puerto Rico).

These models have similar characteristics to the biological models for malaria. By aggregating temperature effects from various stages of the transmission cycle, they demonstrate that overall dengue risk is likely to be extremely sensitive to even small changes in temperature. Sensitivity analyses show that a 2 °C increase in global temperature would lead to large (commonly 1–5 times) increases in transmission intensity in many regions of the world. However, these models are also subject to the same caveats. As there are no data available on mosquito abundance throughout the globe, or the relationship between vector abundance and climate variables, the transmission potential remains a relative rather than absolute measure of R_o. It is consequently unsuitable for defining the conditions under which transmission can persist, and therefore mapping the distributional limits of dengue either now or in the future. In addition, although the validation exercise demonstrates that transmission potential is correlated to the incidence of infection and clinical disease, in that months with higher transmission potential have more cases, the quantitative relationships have not been explicitly defined. It is therefore difficult to interpret exactly what effect a doubling of transmission potential (for example) would have on the burden of disease caused by dengue.

Methods similar to those used by Rogers and Randolph (2000) for malaria have recently been applied to define the relationship between multiple climate parameters and the reported geographic distribution of dengue. Hales et al. (2002) used WHO data (WHO 2000a) to map reports of dengue transmission during the period 1975–1996 at the level of the country, or smaller administrative area when this was specified in the report. These were converted into grid maps at 0.5° resolution. Logistic regression analysis was then used to correlate presence or absence of reported transmission against the average values of various climate parameters (monthly average rainfall, vapour pressure, and maximum, minimum and mean temperature) for the corresponding grid-cells throughout 1961–1990. Vapour pressure (approximately equivalent to absolute humidity, and reflecting both temperature and precipitation) gave the best discrimination between areas with and without reported dengue transmission, with no other climate variables adding significant explanatory power. The accuracy of the model was assessed by cross-validation, repeatedly using 95% of the data to generate predictions for the remaining 5%. The model gave correct predictions of observed presence or absence for an average of 89–92% of grid-cells, with the precise accuracy depending on the radius over which vapour pressure was considered. The limitations of this model are similar to the statistical model for malaria. To be consistent with the choice and exclusion of models for malaria, the model of Hales et al. (2002), which is similar to the excluded model of Rogers and Randolph (2000), was not used in the estimates of disease burden.

Future research

A comprehensive analysis would require the generation of predictive models for other important vector-borne diseases. There is also a need to investigate the effect of the predicted increases in climate variability (e.g. more frequent or more extreme El Niño events), especially on the frequency and intensity of epidemics.

Most importantly, more reliable estimation would require models that cover all stages of the causal chain from climate change to clinical outcome. These should explicitly address the role of socioeconomics and control in determining absolute, rather than relative, measures of R_o. This would allow better definition both of the geographical limits of transmission, and of variations in the incidence within existing or potentially newly endemic regions. They should also address the role of host-immunity in protecting individuals and populations from clinical disease, as exposure to infection changes. Such models would represent a considerable advance over the assumption made here, that changes in the proportion of the populations at risk within a subregion will be reflected in proportional changes in the burden of disease.

For the purpose of health assessments, the nature of the model (i.e. whether each of the biological processes are modelled separately and

then aggregated, or whether climate is statistically related directly to empirical measures of disease burden) is probably less important than model accuracy. There is a clear need for greater model validation, by comparison of predictions with geographical and temporal patterns of infection and disease in the present and recent past. The most urgent requirement for all of these objectives is the availability of better quality surveillance data on infection and disease, from a wide variety of geographical and socioeconomic settings.

Finally, the potential effects of future climate change on vector-borne diseases (or other diseases) should not divert attention or resources from current control efforts. On the contrary, they provide the additional argument that disease control now should also reduce vulnerability to climate change in the future.

4. Results

Summary measures of the effects of climate change on health are presented in this chapter only for the estimated current effects, using the relative risks obtained by extrapolation of the future predictions, as described in section 2.6. The estimates presented here did not attribute DALYs to cardiovascular deaths due to thermal extremes, and excluded any increase due to dengue (see sections 3.6 and 3.10). They should be interpreted with caution as, in contrast to most other risk factors, they relied on modelled rather than directly observed outcomes. They did, however, indicate the estimated distribution of impacts both among geographical regions and among the various causes of disease (Tables 20.16 and 20.17). The models may also be useful for the secondary purpose of indicating the magnitude of health impacts that might already be caused by climate change, but which may not be detected by direct observation using current surveillance systems.

The various causes considered here differed markedly in their contribution to the estimates of the overall burden of disease. In our analysis, climate-change effects on malnutrition, diarrhoea and vector-borne diseases appeared considerably more important than effects on flooding, or on deaths attributable to thermal extremes. It should be noted that, with the exception of malaria, these outcomes are relatively poorly studied in comparison with the direct effects of thermal stress.

The health consequences of climate change are distributed very unevenly among regions. Estimated DALY burdens *per capita* are several hundred times greater in the poorer regions of Africa, parts of the Eastern Mediterranean region and South-East Asia than in western Europe, North America and the more developed regions of the Western Pacific. This is largely a reflection of the much higher baseline incidence of the most important climate-sensitive diseases (malaria, diarrhoea and malnutrition) in these poorer regions, but also of greater vulnerability to climate change effects. Because these major climate-sensitive diseases

Table 20.16 Estimated mortality (000s) attributable to climate change in the year 2000, by cause and subregion

Subregion	*Malnutrition*	*Diarrhoea*	*Malaria*	*Floods*	*CVD*	*All causes*	*Total deaths/million population*
AFR-D	8	5	5	0	1	19	66.83
AFR-E	9	8	18	0	1	36	109.40
AMR-A	0	0	0	0	0	0	0.15
AMR-B	0	0	0	1	1	2	3.74
AMR-D	0	1	0	0	0	1	10.28
EMR-B	0	0	0	0	0	1	5.65
EMR-D	9	8	3	1	1	21	61.30
EUR-A	0	0	0	0	0	0	0.07
EUR-B	0	0	0	0	0	0	1.04
EUR-C	0	0	0	0	0	0	0.29
SEAR-B	0	1	0	0	1	2	7.91
SEAR-D	52	22	0	0	7	80	65.79
WPR-A	0	0	0	0	0	0	0.09
WPR-B	0	2	1	0	0	3	2.16
World	77	47	27	2	12	166	27.82

CVD Cardiovascular disease. As described in section 3.6, the estimated cardiovascular deaths represent temperature-related mortality displacement. Therefore no disease burden is estimated for deaths from this cause in Table 20.17.

mainly affect younger age groups, the health burden associated with climate change appears to be borne mainly by children rather than adults.

5. Discussion

The collective scientific evidence indicates that anthropogenic climate change has already begun and will continue, with potential consequences for human health. Global warming over the past quarter-century was of the order of half a degree centigrade. Such a gradual change is partly obscured by natural climate variability and affects health through complex causal pathways. These characteristics, coupled with considerably larger effects of other factors in the most vulnerable populations, mean that it is inherently difficult to measure directly net health losses or gains attributable to the climate change that have occurred until now.

However, climate change differs from most other health determinants in that considerable effort has been devoted to generating and evaluating formal models to forecast future climate, in response to likely trajectories of atmospheric gas composition. These models are in general agreement that over the next 50–100 years global warming will be approximately five times greater than has been experienced in the last

Table 20.17 Estimated disease burden (000s of DALYs) attributable to climate change in the year 2000, by cause and subregion

Subregion	*Malnutrition*	*Diarrhoea*	*Malaria*	*Floods*	*All causes*	*Total DALYs/million population*
AFR-D	293	154	178	1	626	2185.78
AFR-E	323	260	682	3	1267	3839.58
AMR-A	0	0	0	4	4	11.85
AMR-B	0	0	3	67	71	166.62
AMR-D	0	17	0	5	23	324.15
EMR-B	0	14	0	6	20	147.57
EMR-D	313	277	112	46	748	2145.91
EUR-A	0	0	0	3	3	6.66
EUR-B	0	6	0	4	10	48.13
EUR-C	0	3	0	1	4	14.93
SEAR-B	0	28	0	6	34	117.19
SEAR-D	1918	612	0	8	2538	2080.84
WPR-A	0	0	0	1	1	8.69
WPR-B	0	89	43	37	169	111.36
World	2846	1459	1018	193	5517	925.35

25 years, with associated changes in other potentially hazardous climate characteristics, such as the frequency of extreme precipitation events.

Such modelling is at a relatively early stage. Few modelling studies have estimated health effects at the global scale, and not all of these directly estimate incidence or prevalence of GBD outcomes. However, they provide the best current basis for making indicative forecasts in order to inform policy decisions. These models nevertheless make only crude adjustments for the effects of other variables (such as decreasing poverty), which may both determine the vulnerability of populations to potential health effects of climate change, and exert much larger independent effects on health. Taking each disease in turn:

1. We estimated a small proportional decrease in cardiovascular and respiratory disease mortality attributable to climate extremes in tropical regions and a slightly larger benefit in temperate regions, caused by warmer winter temperatures. Although these proportional changes are modest, they apply to significant causes of death. Uncertainties around these estimates are largely due to lack of knowledge of the degree to which populations physiologically and behaviourally adapt to increasing temperatures.

2. The relative risk for diarrhoea in 2030 in developing regions was estimated to be between 1 and 1.1 under unmitigated emissions compared with baseline climate. Richer countries (GDP >US$ 6000/year),

either now or in the future, were assumed to suffer little or no additional risk of diarrhoea. Again, these small changes in relative risk relate to a major cause of ill-health. Uncertainties were mainly due to poor characterization of variations in the relationship between climate and diarrhoea in more or less developed regions, which have different balances between pathogens preferring higher or lower temperatures.

3. Estimated effects on malnutrition varied markedly across subregions. By 2030, the relative risks for unmitigated emissions relative to no climate change varied from a large increase (RR=1–1.33) in SEAR-D to a small decrease (RR=1–0.99) in WPR-B. Developed countries were assumed to be immune to climate change effects on malnutrition. There was no consistent pattern of reduction in relative risk with intermediate levels of climate change stabilization. Apparent inconsistency in the estimates may be due to the high sensitivity of the models to regional variations in precipitation, for which future projections are much more uncertain than for temperature. Although these estimates are somewhat unstable, they are relatively large, and again relate to a major disease burden.

4. We estimated much larger proportional changes in the numbers of people killed in coastal floods (RR of up to 6.3 in EUR-B for unmitigated emissions compared to baseline conditions in 2030), but applied to a very low burden of disease. Consequences of inland floods were predicted to increase by a similar order of magnitude (RR= 1–18.5 in AMR-A), and are generally more common. In contrast to most other outcomes, the increase in relative risk tended to be at least as high in developed as developing subregions. However, these apply to baseline rates that *are* much higher in developing than developed countries. Both estimates are subject to uncertainty around the likely effectiveness of adaptation measures. Inland floods are subject to additional large uncertainties around the quantitative relationships between changes in the intra-annual variation in precipitation (on which our model is based), the magnitude and geographical distribution of extreme precipitation events, and in turn the frequency of flooding and its health consequences. The suggestion of a trend towards decreasing incidence with increasing GHG emissions in some regions is probably due to the uncertainties in predicting precipitation trends. As projections for precipitation are less secure than for temperature, mid-range estimates and uncertainties around the effects of inland floods could be much better described using multiple climate models, rather than the single model used in this assessment.

5. We estimated relatively large changes in the relative risk of falciparum malaria in countries at the edge of the current distribution, for example, increases in relative risk of falciparum malaria of between

1 and 1.83 in WPR-B by 2030. Relative changes were much smaller in areas that are already highly endemic for these diseases. The principal uncertainties specific to these estimates related to the reliability of extrapolations made between subregions, the relationship between changes in the population at risk of these diseases, and incidence (and therefore disease burden), and over the degree to which changes in the non-climatic influences on vector-borne diseases could affect not only the baseline rates of disease, but also interact with climate change to affect the relative risks.

The estimates for the year 2000 (Tables 20.16 and 20.17) suggest that, according to models summarizing our current knowledge of the relationships between climate and health, past climate change may already be causing some health consequences.

These relative risk estimates are much greater for projections into the future, as climate change continues. Both current and future estimates show extreme variations in the estimated effects among geographical regions. Negative consequences are overwhelmingly concentrated in the developing regions of the world (particularly in Africa and the poorer regions of the Eastern Mediterranean and South-East Asia). This is partly a function of variation in baseline climate (hot regions suffer more from increases in temperature), but more importantly due to population vulnerability (e.g. developed countries are assumed to be completely immune from some diseases, such as malnutrition).

Global models have not yet addressed all of the likely effects of climate change on health. The potential omissions are many infectious diseases, the health consequences of drought and famine (beyond those included in current estimates of malnutrition), population displacement, destruction of health infrastructure in natural disasters, increased pollution and aeroallergen levels, effects of plant pests and diseases on agriculture, and risk of conflict over declining natural resources. It is likely that these health consequences will be larger than those estimated in this chapter.

Although incomplete and encompassing a wide uncertainty range, the results of these analyses suggested that the attributable burden of climate change is likely to be significant, even under the relatively short (in climatological terms) time-scale considered for the comparative risk assessment (CRA). The effect of plausible reductions in climate change was estimated to be relatively small over this assessment period. However, the health gains would clearly be much greater over longer time periods. Given the long time-lag and apparent irreversibility of climate change, early mitigation should therefore result in long-term health benefits. Our results therefore indicate the urgent need for: (i) consideration of optimal policies to reduce climate change; (ii) strengthening of current actions to control climate-sensitive diseases, both as ends in themselves, and as adaptation to future climate change; and (iii) con-

tinued research to revise and narrow the uncertainty range around the estimates of disease burdens.

This assessment has highlighted the most important gaps in data and understanding that should be addressed for the next global burden of disease assessment. Marked improvements in our assessment of this risk factor would come from:

- the use of multiple climate models;
- climate–health relationships derived from a greater range of climatic and socioeconomic environments;
- more explicit and routine validation of the accuracy of disease models in the present or recent past;
- formal analyses to aggregate uncertainty arising from multiple causes (i.e. GHG emissions scenarios, climate models, climate–health relationships, and effect modifiers);
- efforts to formally model climate change effects through to disease burden, rather than intermediate indicators such as population at risk;
- a greater emphasis on investigating the consequences of increased climate variability, rather than gradual changes in mean conditions; and
- the development of analytical tools to assess outcomes acting through more complex causal mechanisms.

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Note

1 See preface for an explanation of this term.

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Appendix A: Uncertainty around climate predictions

Results presented here were generated using COSMIC (Country Specific Model for Intertemporal Climate) (Schlesinger and Williams 1997). This contains simplified versions of the 14 different climate models used by the IPCC, and gives output at the country level. Each model was run specifying three different plausible sensitivities (1.5, 2.5 and 4.5 °C) to the effects of a doubling of atmospheric CO_2, generating 42 alternative future climate scenarios for each GHG emissions scenario. Estimates for changes in annual average temperature and precipitation were generated for each subregion by taking population weighted averages of the country level estimates. Means, ranges and 95% confidence intervals assume independence, and equal probability for each future model and climate sensitivity. All values in figures and tables are for changes relative to 1990.

Figure A.1 Mean and 95% CI of COSMIC model outputs for temperature change in the 2020s (relative to 1990) for each subregion under each GHG emissions scenario

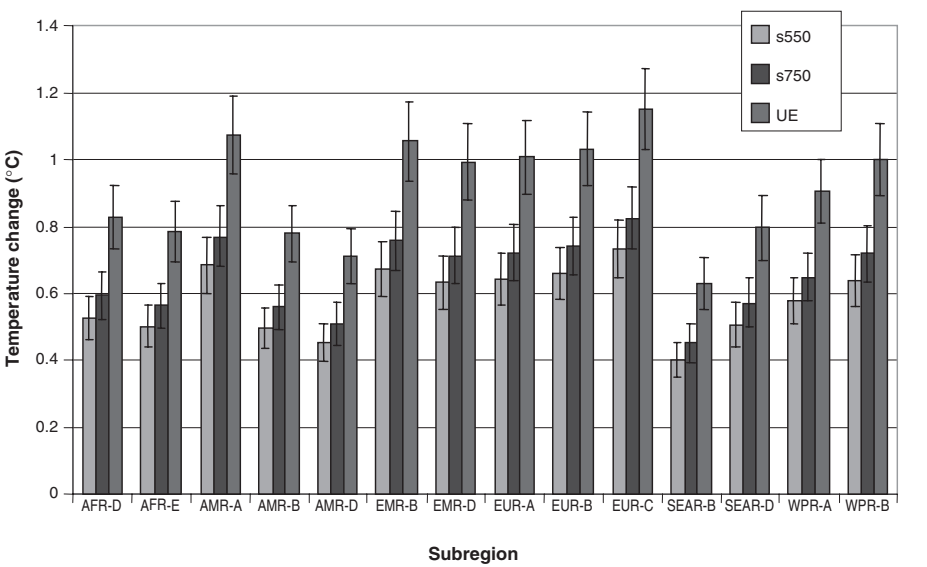

Table A.1 Mean and range of COSMIC model outputs, for temperature change in the 2020s, relative to 1990[a]

(a) S550

Subregion	*Mean dT (°C) 95% CI*	*Min–max range*	*COSMIC version HadCM2 (2.5 °C sensitivity)*
AFR-D	0.53 (0.46–0.59)	0.24–0.97	0.53
AFR-E	0.50 (0.44–0.56)	0.23–0.96	0.50
AMR-A	0.68 (0.60–0.77)	0.32–1.21	0.70
AMR-B	0.50 (0.44–0.56)	0.25–0.87	0.47
AMR-D	0.45 (0.39–0.51)	0.21–0.86	0.42
EMR-B	0.67 (0.59–0.76)	0.28–1.25	0.67
EMR-D	0.63 (0.55–0.71)	0.28–1.19	0.62
EUR-A	0.64 (0.56–0.72)	0.31–1.12	0.64
EUR-B	0.66 (0.58–0.74)	0.28–1.18	0.73
EUR-C	0.73 (0.65–0.82)	0.37–1.23	0.72
SEAR-B	0.40 (0.35–0.45)	0.17–0.77	0.35
SEAR-D	0.51 (0.44–0.57)	0.22–0.98	0.50
WPR-A	0.58 (0.51–0.64)	0.27–0.97	0.54
WPR-B	0.64 (0.56–0.71)	0.31–1.25	0.61

(b) S750

Subregion	*Mean dT (°C) 95% CI*	*Min–max range*	*COSMIC version HadCM2 (2.5 °C sensitivity)*
AFR-D	0.59 (0.52–0.66)	0.28–1.08	0.61
AFR-E	0.56 (0.49–0.63)	0.27–1.06	0.57
AMR-A	0.77 (0.68–0.86)	0.37–1.35	0.79
AMR-B	0.56 (0.49–0.62)	0.29–0.97	0.53
AMR-D	0.51 (0.45–0.57)	0.24–0.95	0.48
EMR-B	0.76 (0.67–0.85)	0.33–1.39	0.76
EMR-D	0.71 (0.63–0.80)	0.33–1.32	0.71
EUR-A	0.72 (0.64–0.81)	0.36–1.25	0.72
EUR-B	0.74 (0.65–0.83)	0.32–1.31	0.83
EUR-C	0.82 (0.73–0.92)	0.43–1.37	0.81
SEAR-B	0.45 (0.39–0.51)	0.20–0.86	0.40
SEAR-D	0.57 (0.50–0.64)	0.25–1.09	0.57
WPR-A	0.65 (0.58–0.72)	0.31–1.08	0.61
WPR-B	0.72 (0.63–0.80)	0.36–1.39	0.69

Table A.1 Mean and range of COSMIC model outputs, for temperature change in the 2020s, relative to 1990[a] (*continued*)

(c) Unmitigated emissions

Subregion	*Mean dT (°C) 95% CI*	*Min–max range*	*COSMIC version HadCM2 (2.5 °C sensitivity)*
AFR-D	0.83 (0.73–0.92)	0.41–1.47	0.85
AFR-E	0.78 (0.70–0.87)	0.39–1.45	0.80
AMR-A	1.07 (0.96–1.19)	0.54–1.83	1.11
AMR-B	0.78 (0.69–0.86)	0.42–1.32	0.75
AMR-D	0.71 (0.63–0.79)	0.35–1.29	0.67
EMR-B	1.06 (0.94–1.17)	0.48–1.88	1.08
EMR-D	0.99 (0.88–1.11)	0.48–1.79	1.00
EUR-A	1.01 (0.90–1.12)	0.52–1.69	1.02
EUR-B	1.03 (0.92–1.15)	0.46–1.78	1.17
EUR-C	1.15 (1.03–1.27)	0.63–1.86	1.15
SEAR-B	0.63 (0.55–0.70)	0.29–1.17	0.56
SEAR-D	0.80 (0.70–0.89)	0.36–1.49	0.80
WPR-A	0.91 (0.81–1.00)	0.45–1.46	0.86
WPR-B	1.00 (0.89–1.11)	0.53–1.89	0.97

[a] Results of the simplified version of the HadCM2 model at medium (2.5 °C) sensitivity are shown for comparison.

Figure A.2 Mean and 95% CI of COSMIC model outputs for precipitation change in the 2020s (relative to 1990), for each subregion under each GHG emissions scenario

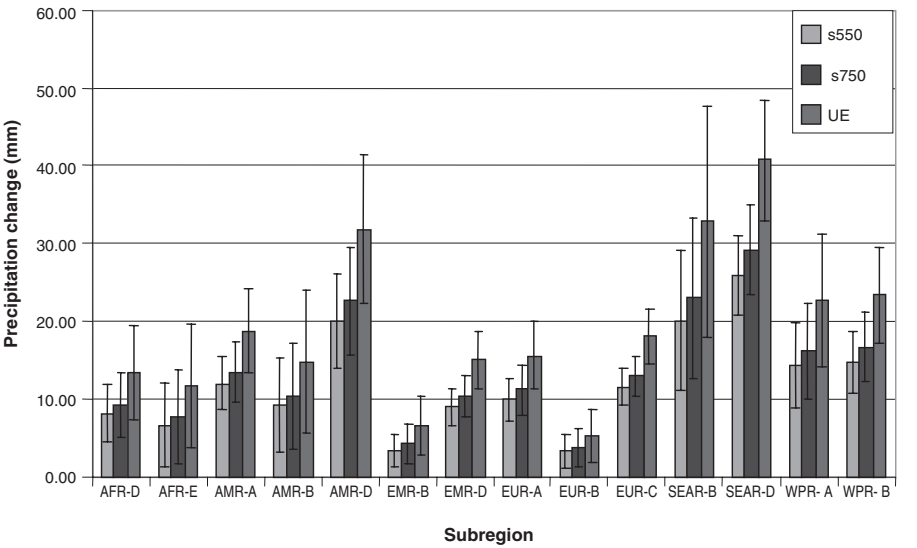

Table A.2 Mean and range of COSMIC model outputs, for change in annual precipitation in the 2020s, relative to 1990[a]

(a) S550

Subregion	*Mean dPrecipn (mm) 95% CI*	*Min–max range*	*COSMIC version HadCM2 (2.5 °C sensitivity)*
AFR-D	8.18 (4.36–11.99)	–15.8–43.88	4.92
AFR-E	6.71 (1.29–12.14)	–31.83–57.55	14.88
AMR-A	12.01 (8.53–15.48)	–10.38–54.00	13.56
AMR-B	9.17 (3.12–15.22)	–41.47–60.79	5.28
AMR-D	20.03 (13.87–26.18)	–31.17–71.40	33.48
EMR-B	3.45 (1.38–5.52)	–7.10–25.61	2.52
EMR-D	9.05 (6.71–11.38)	–1.83–32.97	5.76
EUR-A	9.94 (7.10–12.77)	–9.58–43.69	14.88
EUR-B	3.33 (1.18–5.47)	–9.55–15.18	7.56
EUR-C	11.50 (9.14–13.86)	0.72–37.68	12.48
SEAR-B	20.11 (11.05–29.16)	–35.40–135.16	7.20
SEAR-D	25.87 (20.74–31.01)	4.40–71.19	17.64
WPR-A	14.39 (8.93–19.84)	–33.56–68.61	1.32
WPR-B	14.82 (10.82–18.82)	–7.36–55.37	13.44

(b) S750

Subregion	*Mean dPrecipn (mm) 95% CI*	*Min–max range*	*COSMIC version HadCM2 (2.5 °C sensitivity)*
AFR-D	9.25 (4.97–13.52)	–17.66–48.79	5.60
AFR-E	7.67 (1.66–13.68)	–31.02–63.98	16.83
AMR-A	13.52 (9.63–17.40)	–11.79–60.07	15.43
AMR-B	10.32 (3.54–17.10)	–46.11–67.59	5.97
AMR-D	22.61 (15.73–29.49)	–34.64–79.40	37.88
EMR-B	4.24 (1.62–6.86)	–7.89–37.10	3.03
EMR-D	10.33 (7.71–12.94)	–2.05–36.66	6.47
EUR-A	11.19 (8.02–14.35)	–10.85–48.58	16.87
EUR-B	3.74 (1.34–6.15)	–10.82–16.88	8.60
EUR-C	12.95 (10.34–15.57)	0.83–41.90	14.18
SEAR-B	23.00 (12.69–33.32)	–39.37–150.27	8.23
SEAR-D	29.15 (23.45–34.85)	5.07–79.18	19.94
WPR-A	16.22 (10.11–22.33)	–37.30–76.29	1.49
WPR-B	16.70 (12.24–21.17)	–8.19–62.12	15.22

Table A.2 Mean and range of COSMIC model outputs, for change in annual precipitation in the 2020s, relative to 1990[a] (*continued*)

(c) Unmitigated emissions

Subregion	*Mean dPrecipn (mm) 95% CI*	*Min–max range*	*COSMIC version HadCM2 (2.5 °C sensitivity)*
AFR-D	13.39 (7.44–19.35)	–23.85–66.26	7.96
AFR-E	11.67 (3.69–19.64)	–37.40–86.89	23.74
AMR-A	18.81 (13.49–24.13)	–16.60–81.56	21.78
AMR-B	14.76 (5.64–23.88)	–52.87–92.27	8.42
AMR-D	31.84 (22.38–41.31)	–47.05–107.81	53.44
EMR-B	6.54 (2.75–10.34)	–10.49–54.18	4.69
EMR-D	15.10 (11.37–18.82)	–2.55–49.78	9.14
EUR-A	15.58 (11.24–19.92)	–15.30–65.98	23.79
EUR-B	5.22 (1.90–8.53)	–15.27–22.92	12.13
EUR-C	18.04 (14.50–21.58)	1.22–56.88	19.99
SEAR-B	32.75 (17.95–47.54)	–53.44–220.93	11.62
SEAR-D	40.72 (33.00–48.44)	7.38–107.89	28.12
WPR-A	22.67 (14.23–31.11)	–50.64–104.49	2.09
WPR-B	23.33 (17.18–29.47)	–11.13–86.26	21.45

[a] Results of the simplified version of the HadCM2 model at medium (2.5 °C) sensitivity are shown for comparison.

Appendix B: Literature review

For all impacts, we attempted to find all models that directly related climate change to the selected health effects, either at the global or large-regional level. We describe all such models, and outline reasons for using or not using them in the assessment. Note that the comments given relate to their relative suitability for generating the estimates required for this assessment, rather than a general judgement on their merits. For the effects of thermal extremes, we also include a summary of the study populations that were used to derive the heat and cold estimates used in this assessment.

Where existing global or large scale regional models are not considered appropriate (thermal extremes), or do not exist at all (diarrhoea and floods), the literature search describes the procedure for identifying relevant studies from which to derive new quantitative climate–health relationships and generate models.

THERMAL EXTREMES

Information sources:

- all relevant papers cited in IPCC (2001a)
- Medline search for all references (1966–2002) containing the terms "temperature"; "mortality"; "cardiovascular disease" (or CVD); "respiratory"; "weather"
- references cited in these papers.

Table B.1 Results of literature search for effects of climate change on deaths due to thermal extremes[a]

Reference	*Method*	*Inputs*	*Region*	*Key findings*	*Suitability for generating estimates for this assessment*
Langford and Bentham (1995)	Regression model: monthly mortality and temperature series (flu included)	Seasonal average dT from United Kingdom scenarios (Warrick and Barrow 1991)	England and Wales	Winter deaths avoided: 2010: all cause 3301, IHD 1308, CVD 429 2030: all cause 6353, IHD 2550, CVD 836 2050: all cause 8922, IHD 3631, CVD 1187	No population growth, ageing. No seasonal adjustment. Local rather than global
Martens (1998a)	MIASMA v1.0—empirical-statistical model, meta-analysis. A single temperature-mortality relationship was applied to all cities	ECHAM1-A, UKTR, GFDL89 (IPCC scenarios)	Cause+age specific. Global—20 cities	Changes in mortality rates for CVD (<65), CVD (>65), respiratory, and total mortality Net reductions in mortality in all cities except respiratory mortality in a few cities	Lack of control for seasonal variations, perhaps over-estimating heat effects
McMichael et al. (2000b)	MIASMA v.1.0—as for Martens (1998a)	HadCM2, ensemble mean+HadCM3	Global—20 cities	Net reductions in mortality in all cities except Athens, due to decreases in winter mortality	As for Martens (1998a)

continued

Table B.1 Results of literature search for effects of climate change on deaths due to thermal extremes[a] (*continued*)

Reference	*Method*	*Inputs*	*Region*	*Key findings*	*Suitability for generating estimates for this assessment*
Kalkstein and Smoyer (1993)	Model derived from observed relationship between synoptic air masses and mortality	GCM scenarios, no downscaling	Global—27 cities	Significant increases in heat-related mortality under various climate change scenarios, with or without acclimatization, projected for all cities	Methods used rely on synoptic classification of local air masses; therefore cannot be directly related to changes in temperature described by climate scenarios
Kalkstein and Greene (1997)	Model derived from observed relationship between synoptic air masses and mortality	GCM scenarios, no downscaling	44 cities in the USA	Increases in heat-related mortality are much larger overall than decreases in cold-related mortality	Methods used rely on synoptic classification of local air masses; therefore cannot be directly related to changes in temperature described by climate scenarios
Duncan et al. (1997)	Model derived from observed relationship between synoptic air masses and mortality	Climate scenarios for Canada	10 cities in Canada	240–1140 additional heat-related deaths/year in Montreal by 2050; 230–1220 additional deaths in Toronto, assuming no acclimatization. No climate/mortality relationship in some cities	Country specific
Guest et al. (1999)	Regression model: daily mortality and synoptic indices	CSIRO Mark 2 model, CO_2 doubling; high and low scenarios estimated	5 cities in Australia	Net decrease in heat-related mortality, particularly in the age group ≥65 years; range of 47–62 fewer deaths/year in all 5 cities. Significant increase in summer deaths in Sydney (76–239)	Country specific
Dessai (2003)	Empirical statistical model, observed-expected	2 regional climate models—PROMES and HadRM2	Lisbon, Portugal	Heat-related death rates increase by 57–113% by 2020s, by 97–255% by 2050s. Acclimatization assumptions, reduce estimates	Country specific

Key: CVD, cardiovascular disease; IHD, ischaemic hearth disease; GCM, global climate model.

[a] A total of 76 other publications were not used, either because they were analyses of specific episodes (35 studies), or because they did not match the criteria outlined above (41 studies).

Estimates of temperature–mortality relationship

Table B.2 below lists all studies (n=4) that meet the following criteria for inclusion in the meta-analysis. The study:

- uses daily time-series methods to analyse the relationship between daily mean temperature and mortality;
- Reports a coefficient from log linear regression that estimates the percentage changes in mortality per degree centigrade change in temperature, above a reported threshold temperature;
- has controls for the following confounders: season, air pollution and influenza;
- is published in English language only; and
- reports confidence intervals around the coefficient and is within the range of other reported estimates.

The studies are classified according to climate zone. The Netherlands population is used to approximate a population in the "cold" zone as there are no other appropriate time-series studies.

Note that some studies are included that are not yet published. However,

- they are the only studies available that provide estimates for developing country populations; and
- the results will be submitted to peer reviewed journals and the methods are at least as rigorous as those applied in previous published studies.

All estimates have been rounded to one decimal place. Where more than one estimate was available, the average estimate was calculated.

Table B.2 Results of literature search for temperature–mortality relationship

Population/ climate zone	*Age*	*Cause of death*	*% change[a] per 1 °C decrease*	*Cut-point (°C)*	*Lag*	*% change[a] per 1 °C increase*	*Cut-point (°C)*	*Lag*	*Reference*
			COLD			HEAT			
Netherlands	All	All cause	0.41	16.5	7–15	1.23	16.5	1–2	Kunst et al. (1993)
Netherlands	All	CVD	0.46	16.5	7–15	1.13	16.5	1–2	Kunst et al. (1993)
Netherlands	All	Respiratory	1.43	16.5	7–15	3.11	16.5	1–2	Kunst et al. (1993)
COLD		CVD	0.5			1.1			
Bulgaria									
Sofia	All	All	2.69 (0.88–4.54)	–2	0–13	1.93 (1.41–2.45)	17	0–2	ISOTHURM (forthcoming)
Chile									
Santiago	All	All	5.21 (3.55–6.89)	11	0–13	0.92 (0.44–1.31)	16	0–2	ISOTHURM (forthcoming)
Slovenia									
Ljubljana	All	All	0.77 (–0.16–1.70)	7	0–13	2.25 (1.09–3.42)	17	0–2	ISOTHURM (forthcoming)
South Africa									
Cape Town	All	All	3.32 (2.89–3.75)	19	0–13	1.02 (–0.32–2.38)	21	0–2	ISOTHURM (forthcoming)
Spain									
Madrid	All	All	1.7	20	7	0.97	20	0–1	Alberdi (1998)
Valencia	All	All	3.2 (1.8–4.6)	15	7–14	3.6 (1.2–6.0)	24	1–2	Ballester et al. (1997)
Valencia	>70	All	3.7 (2.1–5.4)	15	7–14	5.0 (2.1–8.0)	24	1–2	Ballester et al. (1997)
Valencia	All	CVD	4.3 (2.1–6.4)	15	7–14	2.3 (–1.5–4.5)	24	1–2	Ballester et al. (1997)
Valencia	All	CVD	1.5 (–0.3–3.3)	15	3–6	2.9 (–0.4–7.4)	24	3–6	Ballester et al. (1997)
Valencia	All	Respiratory	1.7 (–0.4–6.0)	15	7–14	5.7 (–2.9–8.2)	24	1–2	Ballester et al. (1997)

TEMPERATE		CVD	2.9			2.6			
Brazil									
São Paulo	All	All cause	3.92 (3.43–4.40)	19	0–13	2.28 (2.11–3.66)	23	0–2	ISOTHURM (forthcoming)
São Paulo	≥65	All cause	5.5	20	0–21	2.5 (2.1–2.8)	20	0–1	Gouveia et al. (2003)
El Salvador									
San Salvador	All	All cause	−13.5 (−32.35–10.9)	23	0–13	1.59 (0.86–2.32)	23	0–2	ISOTHURM (forthcoming)
Mexico									
Mexico City	All	All cause	8.60 (7.86–9.34)	15	0–13	0.6 (0.21–1.00)	18	0–2	ISOTHURM (forthcoming)
Mexico									
Monterrey	All	All cause	5.54 (4.52–6.58)	17	0–13	19.85 (14.69–25.25)	31	0–2	ISOTHURM (forthcoming)
Thailand									
Bangkok	All	All cause	4.13 (1.71–6.61)	28	0–13	7.66 (5.87–9.47)	30	0–2	ISOTHURM (forthcoming)
WARM HUMID	All	All cause	5.5			5.7			
India									
Delhi	All	All cause	1.36 (0.56–2.16)	19	0–13	3.03 (2.48–3.58)	28	0–2	ISOTHURM (forthcoming)
HOT DRY	All	All cause	1.4			3.0			

CVD Cardiovascular disease.

[a] % change = (RR − 1) × 100.

DIARRHOEA

Information sources:

- all relevant papers cited in IPCC (2001a)
- Medline search for all references (1966–2002) containing the terms "Diarrhoea (or diarrhea) and climate (or climatic) change"; "diarrhoea (or diarrhea) and climate"; "diarrhoea (or diarrhea) and weather"
- references cited in these papers.

Table B.3 Results of literature search for effects of climate change on diarrhoea[a]

Global models relating climate/climate change to diarrhoea	*Local studies quantitatively relating all-cause diarrhoea to temperature*	*Local studies showing seasonal patterns in all- or multiple-cause diarrhoea (non-quantitative)*	*Local studies showing pathogen-specific associations with climate*
None	Checkley et al. (2000); Singh et al. (2001)	Anjaneyulu et al. (1975); Becker (1981); Brewster and Greenwood (1993); Dean and Jones (1972); Hoge et al. (1996); Jin et al. (1996); Ling and Cheng (1993); Merlin et al. (1986); Parashar et al. (1999); Pinfold et al. (1991); Robins-Browne (1984); Rousham and Mascietaylor (1995); Saidi et al. (1997); Sawchuk (1993); Shaikh et al. (1990); Sutra et al. (1990); Tsukamoto et al. (1978); Van den Broeck et al. (1993); Williams et al. (1986); Yang et al. (1990)	Showing association: Adegbola et al. (1994); Adkins et al. (1987); Aggarwal et al. (1983); Ansari et al. (1991); Armah et al. (1994); Beards and Graham (1995); Bockemuhl (1976); Callejas et al. (1999); Chakravarti et al. (1992); Chan et al. (1998); Chaudhury et al. (1996); Cook et al. (1990); Cunliffe et al. (1998); da Rosa e Silva et al. (2001); Douglas and Kurien (1997); Eberhard et al. (1999); Fujita (1990); Henry and Bartholomew (1990); Hirschl et al. (1987); Ijaz et al. (1985); Konno et al. (1983); Laursen et al. (1994); LeBaron et al. (1990); Malakooti et al. (1997); Muhuri (1996); Musa et al. (1999); Nchito et al. (1998); Parashar et al. (1998a, 1998b); Pazzaglia et al. (1993); Purohit et al. (1998); Qiao et al. (1999); Ram et al. (1990a, 1990b); Reyes et al. (1996); Rytlewska et al. (2000); Sallon et al. (1991); Sethi et al. (1984); Shahid et al. (1987); Shkarin et al. (1983); Sorvillo et al. (1998); Stewien et al. (1991); Stintzing et al. (1981); Tswana et al. (1990); Utsalo et al. (1991); Vlasov et al. (1983); Wilcox et al. (1992); Wuhib et al. (1994) Failing to find association: Bishop et al. (2001); Conteas et al. (1998); Varoli et al. (1989)

[a] A total of 597 publications were rejected on the basis of lack of information in abstract, or irrelevance to quantitative assessment (mainly reviews, clinical and laboratory studies).

MALNUTRITION

Information sources:

- all relevant papers cited in IPCC (2001a)
- Medline search for all references (1966–2002) containing the terms "Climate (or climatic) change and malnutrition (or food)"
- references cited in these papers.

Table B.4 Results of literature search for effects of climate change on malnutrition[a]

Study	*Model type*	*Model output*	*Area covered*	*Conclusions*	*Suitability for generating estimates for this assessment*
Fischer et al. (1994)	Coupled crop and food trade model	Socioeconomic impacts, divided by region	Global	Significant impacts under climate change, concentrated in specific regions	More recent models available
Rosenzweig and Parry (1994)	Coupled crop and food trade model	Prevalence of insufficient energy intake, divided by region	Global	Significant impacts under climate change, concentrated in developing countries	More recent models available
Parry et al. (1999)	Coupled crop and food trade model	Prevalence of insufficient energy intake, divided by region	Global	Significant impacts under climate change, concentrated in developing countries	Not run on stabilization scenarios
Parry et al. (2001)	Coupled crop and food trade model	Global prevalence of insufficient energy intake	Global	Significant increase in food shortage, concentrated in developing countries	Not run on stabilization scenarios
Arnell et al. (2002)	Coupled crop and food trade model	Global prevalence of insufficient energy intake	Global	Significant impacts under climate change, CO_2 stabilization would generally reduce prevalence	Regional breakdowns of these results used as a proxy measure of prevalence of malnutrition

[a] A total of 281 other publications were rejected as they are either specific to particular crops or regions (10 studies), or irrelevant to quantitative assessment (mainly reviews, impacts on other ecological systems).

COASTAL AND INLAND FLOODING

Information sources:

- all relevant papers cited in IPCC (2001a)
- ISI-Web of Science search for "Floods (or flooding) and climate (or climatic) change"; "Floods (or flooding) and health (or death or injury or mortality)"; "Extreme precipitation and health (or death or injury or mortality)"
- Medline search for "Floods (or flooding) and climate (or climatic) change"
- References cited in these papers
- Inspection of descriptions of all flood events listed in EM-DAT enhanced database 1980–1999 (EM-DAT 2002).

Table B.5 Results of literature search for effects of climate change on deaths and injuries due to floods and landslides[a]

Large area studies quantitatively relating precipitation or sea-level rise to deaths/injuries	*Global or large regional models predicting climate change effects on frequency of extreme precipitation events or inland flooding*	*Global models predicting climate change effects on frequency of coastal flooding*	*Causes of flood events which caused deaths and/or injuries reported in the EM-DAT enhanced database (1980–1999)*	*Causes of landslides which caused deaths and/or injuries reported in the EM-DAT enhanced database (1980–1999)*
None	Booij (2002), western Europe	Baarse (1995)	Associated with precipitation	63 associated with precipitation
	Fowler and Hennessy (1995), specific locations worldwide	Hoozemans and Hulsburgen (1995)	2 with high tides and storm surges	10 associated with other causes (e.g. landslides at mines, cliffs collapsing)
	Jones and Reid (2001) United Kingdom	Nicholls and Mimura (1998)	19 other causes (e.g. cyclones, ice melt, dams bursting)	115 without specific information on cause
	Milly et al. (2002) specific locations worldwide	Nicholls et al. (1999)	559 without specific information on cause	
	Palmer and Ralsanen (2002) northern Europe, South-East Asia			

[a] A total of 342 other publications were rejected as they are either specific to particular regions, or irrelevant to quantitative assessment (mainly reviews, descriptions of specific events).

MALARIA

Information sources:

- all relevant papers cited in IPCC (2001a)
- Medline search for all references (1966–2002) containing the terms "Malaria and climate (or climatic) change"
- references cited in these papers.

Table B.6 Results of literature search for effects of climate change on malaria

Study	*Model type*	*Model output*	*Area covered*	*Conclusions*	*Suitability for generating estimates for this assessment*
Matsuoka and Kai (1994)	Biological model based on ecoclimatic index (EI) described in Sutherst et al. (1996)	Maps of changes in endemicity and the distribution of malarious areas	Global, China	Change in distribution patterns under CC	Lack of detail in methods, and independent verification
Martin and Lefebvre (1995)	Biological model based on EIP	Maps of TP. Areas at risk of epidemics and endemic malaria	Global	Expansion of distribution, increasing TP under CC	Consideration only of effects on parasite, rather than vector
Martens et al. (1995b)	Biological model—MIASMA v1.0	Changes in malaria risk relative to 1990. Maps of TP and change in TP	Global	Expansion of distribution, increasing TP under CC	Prediction of climate suitability for transmission, rather than actual transmission. Uncertainty over cut-off values to define endemic/non-endemic areas. Vector distributions not considered
Martens et al. (1995a)	Biological model—MIASMA v1.0	Changes in malaria risk relative to 1990. Maps of TP and change in TP	Global	Expansion of distribution, increasing TP under CC	Prediction of climate suitability for transmission, rather than actual transmission. Uncertainty over cut-off values to define endemic/non-endemic areas. Vector distributions not considered

continued

Table B.6 Results of literature search for effects of climate change on malaria (*continued*)

Study	*Model type*	*Model output*	*Area covered*	*Conclusions*	*Suitability for generating estimates for this assessment*
Bryan et al. (1996)	CLIMEX model	Vector distributions only	Northern Australia	Change in distribution under CC	Single vector, local rather than global predictions
Jetten et al. (1996)	Biological model—MIASMA v1.0	Maps of TP and change in TP	Global	Expansion of distribution, increasing TP under CC	Prediction of climate suitability for transmission, rather than actual transmission. Uncertainty over cut-off values to define endemic/non-endemic areas. Vector distributions not considered
Martens (1997)	MIASMA v1.0	Maps of TP and change in TP, *P. vivax* and *P. falciparum*	Global	Expansion of distribution, increasing TP under CC	Prediction of climate suitability for transmission, rather than actual transmission. Uncertainty over cut-off values to define endemic/non-endemic areas. Vector distributions not considered
Lindsay and Martens (1998)	MIASMA v1.0—biological model based on vectorial capacity	Maps—epidemic potential	African Highlands	Increase in latitude under CC	Regional projections only
Rogers (1996)	Empirical statistical mapping	Maps of distribution of *Anopheles gambiae*	Southern Africa	Change in distribution under CC	Regional projections only
Martens et al. (1999)	Biological model, overlaying a population grid. MIASMA v2.0	Maps of TP, maps of changes in seasonal transmission, additional population at risk	Global	Expansion of distribution, increasing TP under CC	Prediction of climate suitability for transmission, rather than actual transmission

Arnell et al. (2002)	Biological model, overlaying a population grid. MIASMA v2.0	Maps of changes in *P. falciparum* TP, population at risk, seasons suitable for transmission relative to 1961–1990	Global	Expansion of distribution, increasing TP under CC	Prediction of climate suitability for transmission, rather than actual transmission
Rogers and Randolph (2000)	Empirical-statistical model	Changes in populations at risk: *P. falciparum*	Global	Approximately no change in distribution under climate change	Additional population at risk ±25 million. Validation of model derived from a subset of the data against the remaining observations. (Used to inform uncertainty range around the projections)
Tanser et al. (2003)	MARA biological/ statistical model (Craig et al. 1999)	Changes in populations at risk—months suitable for transmission	Africa	Expansion in population at risk under CC	Biological model based on localized field studies, used to predict population at risk and numbers of months at risk throughout Africa, but not elsewhere. Potential for developing into predictions of incidence. Validated against detailed independent data set. (Used to determine mid-range estimates of population at risk, as a relative measure of change in incidence)

Key: CC, climate change; EIP, extrinsic incubation period; TP, transmission potential (also called EP, epidemic potential).

[a] A further 41 publications were rejected due to lack of relevance for making global projections (mainly reviews, local studies).

DENGUE

Information sources:

- all relevant papers cited in IPCC (2001a)
- Medline search for all references (1966–2002) containing the terms "Dengue and climate (or climatic) change"
- Secondary references cited in these papers.

Table B.7 Results of literature search for projected effects of climate change on dengue[a]

Study	*Model type*	*Model output*	*Area covered*	*Conclusions*	*Suitability for generating estimates for this assessment*
Martens et al. (1997)	Biological model summarizing climate effects on vector and parasite population dynamics MIASMA v1.0, coupled with DENSIM model of Focks et al. (1995)	Maps of change in epidemic potential under climate change	Global	Large increase in transmission potential with small temperature increase	Relationship between TP and disease incidence not characterized. Uncertainty over definitions for threshold TP to define limits of distribution

Patz et al. (1998)	Biological model summarizing climate effects on vector- and parasite population dynamics	Maps of change in epidemic potential under climate change	City specific (Athens, Bangkok, Mexico City, Philadelphia, Puerto Rico, San Juan)	Large increase in transmission potential with small temperature increase	Model is most accurate for non-endemic areas bordering endemic areas and may underestimate changes in transmission in temperate zones. Relationship between TP and disease incidence not characterized
Jetten and Focks (1997)	Biological model summarizing climate effects on vector- and parasite population dynamics	Maps of changes in Critical Density Threshold	Global	Large increase in areas suitable for transmission and length of transmission season under climate change	Relationship between TP and disease incidence not characterized. Uncertainty over setting of cut-offs for transmission
Hales et al. (2002)	Empirical statistical model	Maps of population at risk for dengue transmission	Global	Absolute humidity accurately explains current distribution of dengue. Large increases in population at risk predicted under climate change	Validation of model derived from a subset of the data against the remaining observations. (Used to determine population at risk, as a relative measure of change in incidence)

[a] A further 12 publications were rejected due to lack of relevance for making global projections (mainly reviews, local studies).

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