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MODELLING THE EFFECT OF GLOBAL WARMING ON THE PREVALENCE OF SCHISTOSOMIASIS

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SUMMARY

One of the potential health consequences of global warming is a (re-)introduction of vector-borne diseases into certain regions which may, or may not have been, previously endemic. In most tropical countries, such diseases are a major cause of illness and death. One of the most important among them is schistosomiasis, which is associated with one of three species of parasite each of which is transmitted by a specific snail (intermediate host). Temperature and precipitation changes would directly affect the behaviour and geographical distribution of the vectors and the development of the parasites. Climate change could also have an indirect effect by influencing environmental factors such as vegetation and the availability of breeding sites.

The *direct* effects of a change in temperature and precipitation on the transmission potential of the snail population and the consequent effects on human health is evaluated by assessing the change in potential schistosomiasis risk areas and the change in world schistosomiasis prevalence. General Circulation Model based scenarios of anthropogenic global climate change are deployed in the assessment of potential changes in areas vulnerable to the transmission of schistosomiasis. The study shows that the transmission potential of this vector-borne disease is, although to a lesser extent than malaria, very sensitive to climate changes on the periphery of the present endemic areas and at higher altitudes within these areas. The health impact will be most pronounced among populations living in the economically less developed temperate regions in which endemicity has previously been low or absent. In more developed areas it is expected that no major problems will occur since effective control measures are economically feasible as transmission potential would increase.

SAMENVATTING

Een van de mogelijke gezondheidseffecten van een door de mens geïnduceerde klimaatverandering is een (her)introductie van vector-gebonden infectieziekten in bepaalde gebieden, voorheen al dan niet endemisch. In veel tropische landen zijn vector-gebonden infectieziekten een belangrijke oorzaak van ziekte en sterfte. Een van de meest belangrijke van deze ziekten is schistosomiasis. Drie verschillende soorten parasieten zijn verantwoordelijk voor het ontstaan van schistosomiasis en deze parasieten worden verspreid middels een specifieke soort slakken (vector). Veranderingen in temperatuur en neerslag kunnen direct het gedrag en geografische spreiding van de vector beïnvloeden alsmede de ontwikkeling van de parasiet. De verspreiding van de ziekte kan indirect door een klimaatverandering beïnvloed worden als milieu-factoren, zoals vegetatie-patronen en de beschikbaarheid van broedplaatsen, veranderen.

De *directe* effecten van een verandering van temperatuur en neerslag-patronen op het transmissiepotentieel van de slakken populatie en de hieruit voortvloeiende effecten op de menselijke gezondheid zijn geëvalueerd door de verandering in potentiële schistosomiasis gebieden en de verandering in schistosomiasis prevalentie te schatten. Scenario's voor een klimaatverandering, gebaseerd op 'General Circulation Models', zijn gebruikt in de schatting van mogelijke veranderingen in gebieden gevoelig voor de verspreiding van schistosomiasis. Deze studie toont aan dat de transmissie intensiteit van deze vector-gebonden ziekte gevoelig is voor een klimaatverandering (echter niet zo gevoelig als malaria), met name aan de grenzen van de huidige endemische gebieden en in de hoger gelegen gebieden in deze endemische gebieden. Het effect op de menselijke gezondheid zal het meest merkbaar worden in populaties die in minder ontwikkelde economische gebieden wonen, waar voorheen de transmissie laag of afwezig was. In de meer ontwikkelde gebieden zullen naar alle waarschijnlijkheid afdoende maatregelen genomen kunnen worden indien het transmissie potentieel stijgt.

1. INTRODUCTION

A major challenge facing public health research is the assessment of potential future impacts associated with global environmental changes. Anticipating and quantifying the impact of such environmental changes on human health requires an approach which differs from the conventional epidemiological methods, since scenario-based projections for the future are involved rather than the estimation of effects from empirical data (McMichael, 1993; McMichael & Martens, 1995).

One of the major concerns is the projected enhancement of the natural greenhouse effect which would cause an increase in temperature and a change in precipitation patterns in the coming century at a rate which might prove to be much more rapid than the average rate of natural change. A potential health effect of human-induced climate change may well include a change in the distribution and incidence of vector-borne diseases which are major causes of morbidity and mortality in many tropical countries (WHO, 1990).

Schistosomiasis is one of the world's most important vector-borne diseases with a worldwide prevalence of about 200-250 million within a population of 600 million at risk in 1990. Its incidence is determined by a variety of factors, in particular the abundance of the snail which acts as intermediate host to schistosomes, human behaviour and the presence of the schistosome parasites. Anthropogenic climate change may directly affect the behaviour and geographical distribution of the schistosomiasis snails and the life cycle of the parasite, and thus change the incidence of the disease. Indirectly, climate change could also have an effect by influencing environmental factors such as vegetation and the availability of breeding sites.

In order to estimate the impact of global climate change on the incidence of schistosomiasis as a whole, a thorough knowledge of both direct and indirect consequences of climate changes and their effects on vector-borne disease transmission is required. However, estimates of climate change effects, other than temperature changes, are at best provisional. Furthermore, the possible consequences of simultaneous changes in direct and indirect climate change effects on vector-borne diseases are poorly understood (Bradley, 1993). This study assesses the *direct* effects of projected levels of global climate change on the transmission dynamics pertaining to this vector-borne disease. (The approach adopted in this study thus resembles that employed in studying climate change impacts on malaria risk (Martens, *et al.*, 1994, 1995a)). Although the model only generates broad estimates of future trends and does not include all relevant factors which would influence the (future) distribution of this vector-borne disease (e.g. irrigation practices, the use of molluscoicides, etc.), it nevertheless addresses the question: " If other things remained constant in the world, what would be the impact of climate change *per se* on the distribution of schistosomiasis? "

The model is designed for integration in TARGETS (**T**ool to **A**ssess **R**egional and **G**lobal **E**nvironmental and health **T**argets for **S**ustainability (Rotmans *et al.*, 1994)), which is an integrated, comprehensive modelling framework for environment and public health, developed within the research programme 'Global Dynamics and Sustainable Development'. The TARGETS-framework consists of pressure subsystems (social and economic dynamic systems), environmental subsystems (biosphere dynamics systems) and impact subsystems (ecological, population and health and socio-economic impact dynamics) (see *Figure 1.1*). The programme is designed to yield a more lucid exposition and operationalization of the concepts of global change and sustainable development as a guide in framing global policies.

The structure of this report is as follows: chapter 2 deals with the geographical distribution of schistosomiasis and outlines the main processes in its transmission. In chapter 3, a systems approach is applied to model the impact of a given instance of climate change on schistosomiasis epidemiology. The relations between climate factors and the dynamics of schistosomiasis are quantified in chapter 4 and a number of results are presented in chapter 5. The major findings resulting from the study are discussed in chapter 6.

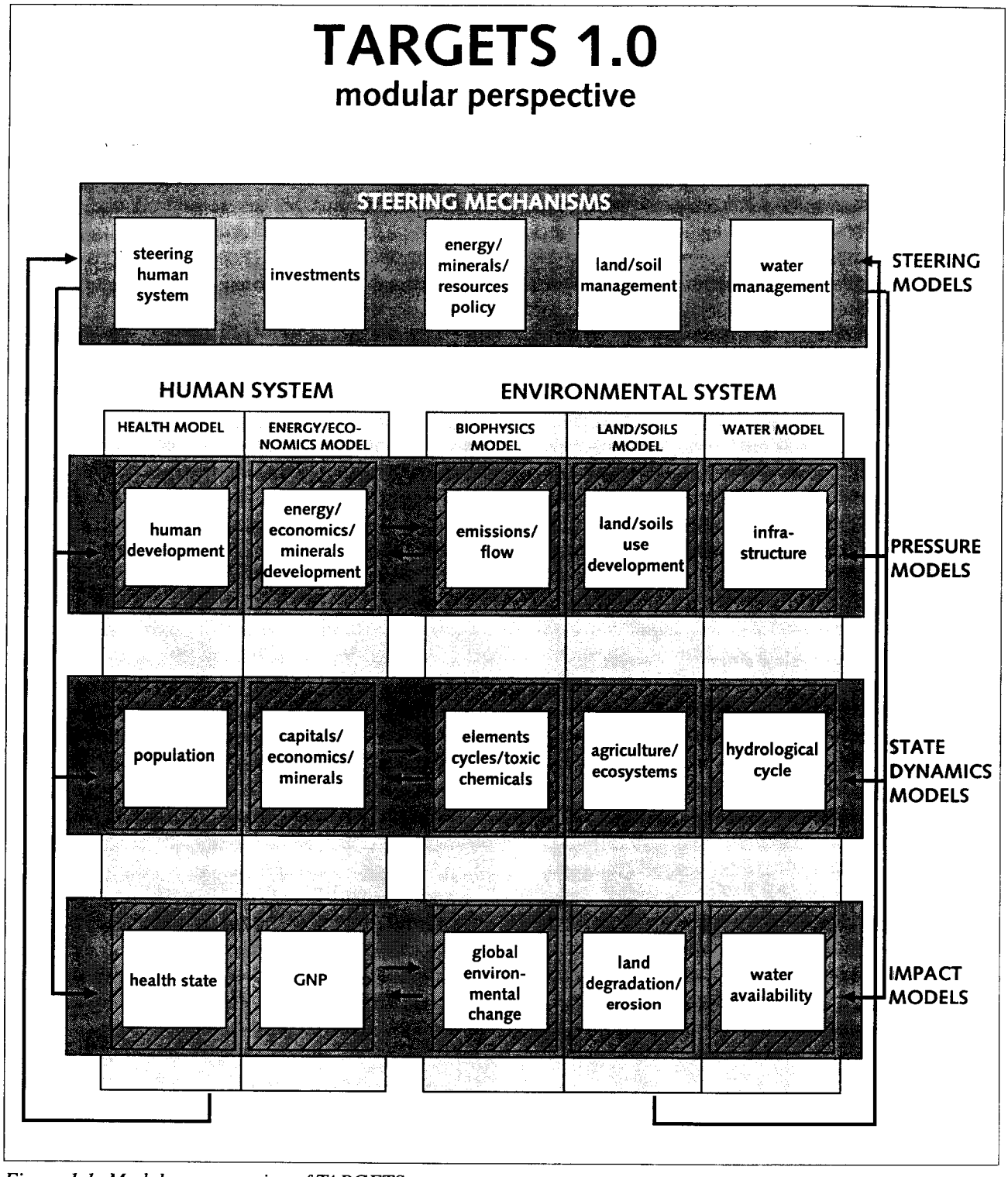


Figure 1.1: Modular perspective of TARGETS

2. SCHISTOSOMIASIS

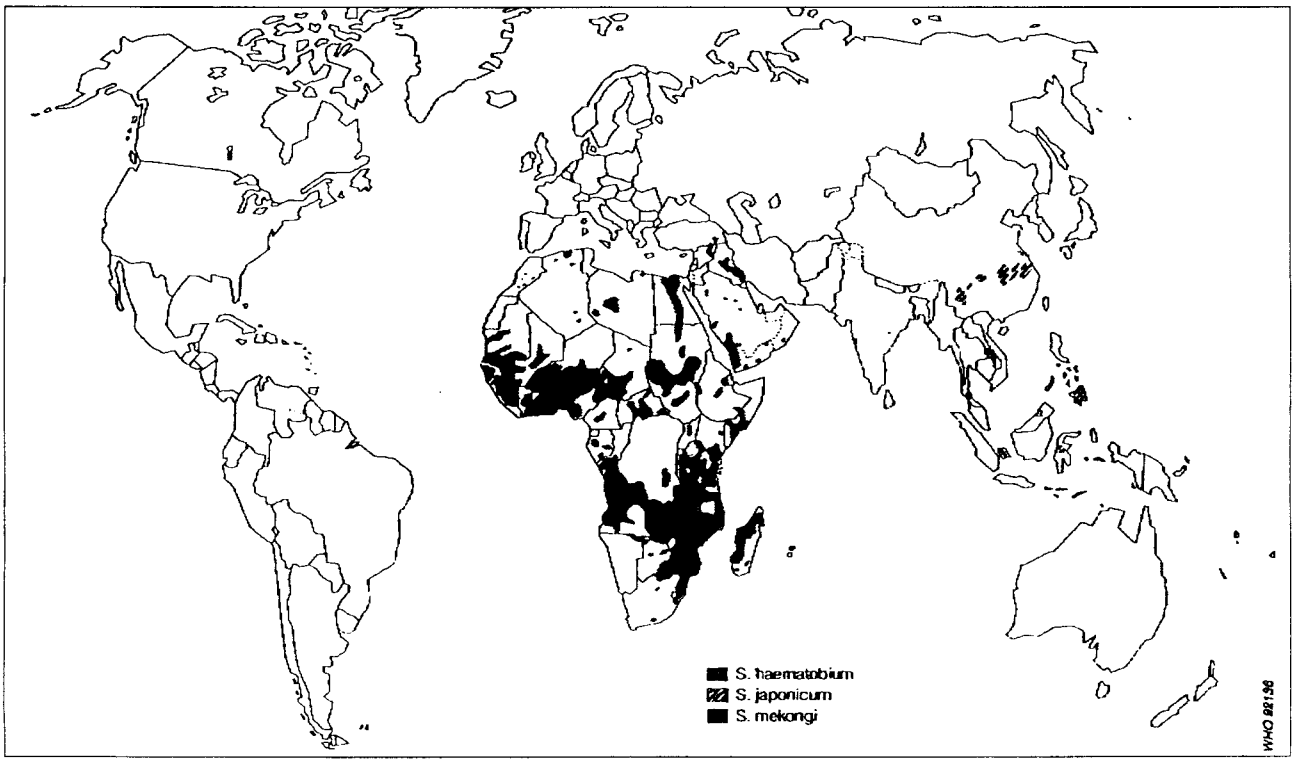
2.1 Schistosomiasis as a global problem

Schistosomiasis, or bilharzia as it was formerly called, is a debilitating infection affecting the well-being of about 200 million people while a total of 600 million are at risk. The WHO (1993) reports that throughout the world, schistosomiasis is endemic in 74 countries, mainly in Africa, Eastern Mediterranean, South America, the Caribbean, south-east Asian and Western Pacific countries (see *Figures 2.1* and *2.2*). Within such endemic areas, the distribution is not uniform. This is due not only to the presence or absence of suitable snails, but also to variations in water supply, water usage and population densities. Moreover, the provision of health services can ameliorate the degree of endemicity. As the schistosome eggs reach the water through faeces and urine, schistosomiasis transmission is facilitated by the inadequate waste disposal and water supplies in many third-world areas. Therefore, inadequate sanitation and water supply are major contributors to schistosomiasis prevalence. It is estimated that 27 % of all schistosomiasis prevalence is caused by inadequate sanitation and water supply and that a 73 % reduction in morbidity due to schistosomiasis could be achieved by improving water quality and the availability of sanitation services (Gleick, 1993). There are several examples of increased transmission of schistosomiasis as a result of irrigation and the development of man-made water resources, although this relation is not always clearly present (Grosse, 1993). The construction of water reservoirs for agriculture may accelerate the transmission of schistosomiasis in endemic areas, although the impact of such impoundments is difficult to gauge.

2.2 The epidemiology of schistosomiasis

Three different species of schistosome account for most human schistosomiasis: *S. mansoni*, *S. japonicum* and *S. haematobium*. The different species of schistosome that infect humans have similar life cycles. The eggs hatch in water as free-swimming

Figure 2.1: Global distribution of schistosomiasis due to Schistosoma haematobium, S. japonicum and S. mekongi (Reprinted with permission from WHO (1993)).



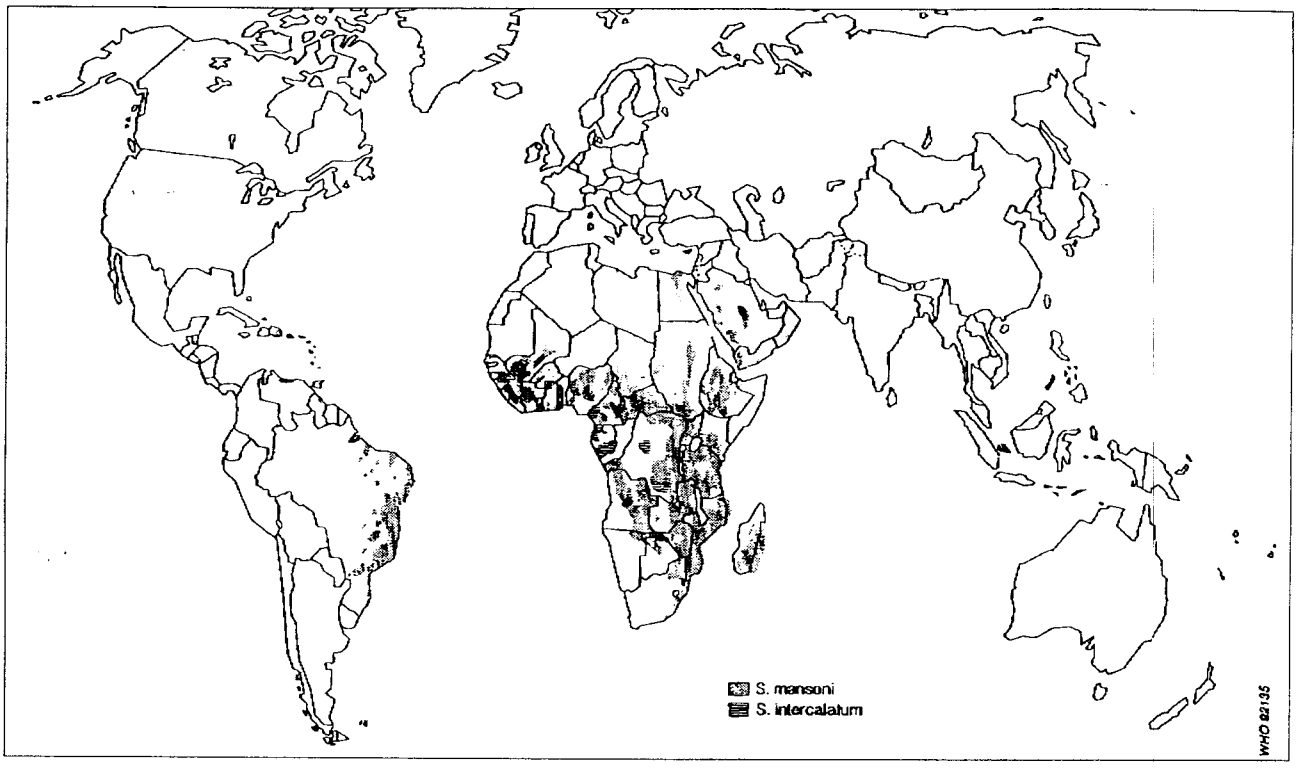


Figure 2.2: Global distribution of schistosomiasis due to Schistosoma mansoni and S. intercalatum (Reprinted with permission from WHO (1993)).

larvae called miracidia. A miracidium must penetrate an appropriate snail with a certain time span (approximately thirty-two hours). Each species of schistosomes can infect only a single species of snail. Therefore, the possible transmission of each form of schistosomiasis depends on the presence of a suitable host. The snail genera responsible for schistosomiasis transmission are *Biomphalaria* (*S. mansoni*), *Bulinus* (*S. haematobium*) and *Oncomelania* (*S. japonicum*) (Jordan & Webbe, 1982).

In the snail, asexual reproduction of the schistosome takes place. A single miracidium produces about 200 to 400 free-swimming larvae, called cercariae, that are shed from the snail and must penetrate the skin of a human host within approximately two days. The penetration of the skin takes about two minutes. After penetration, the schistosome migrates through the host's circulatory system to the liver. On reaching maturity and mating, the schistosome migrates to the veins in the intestines (*S.mansoni* and *S.japonicum*) or to the veins in the bladder (*S. haematobium*). From here the eggs are deposited through faeces or urine into the water (Weil & Kvale, 1985). The majority of the persons infected do not present symptoms of the disease. Symptoms do not result from the adult worm, but rather from the eggs that remain in the host tissue.

The type of health complications appears to vary according to the species and strain of parasite and the characteristics of the human population. Schistosomiasis usually causes blood and nutrient loss in either the stool or the urine, which may lead to anaemia and retarded physical growth. More serious complications include bladder or ureter calcification in urinary schistosomiasis and an enlarged liver and spleen in intestinal schistosomiasis. An association between schistosomiasis and bladder and colorectal cancer has also been found (WHO, 1993).

3. CLIMATE CHANGE AND SCHISTOSOMIASIS: A SYSTEMS APPROACH

3.1 Introduction

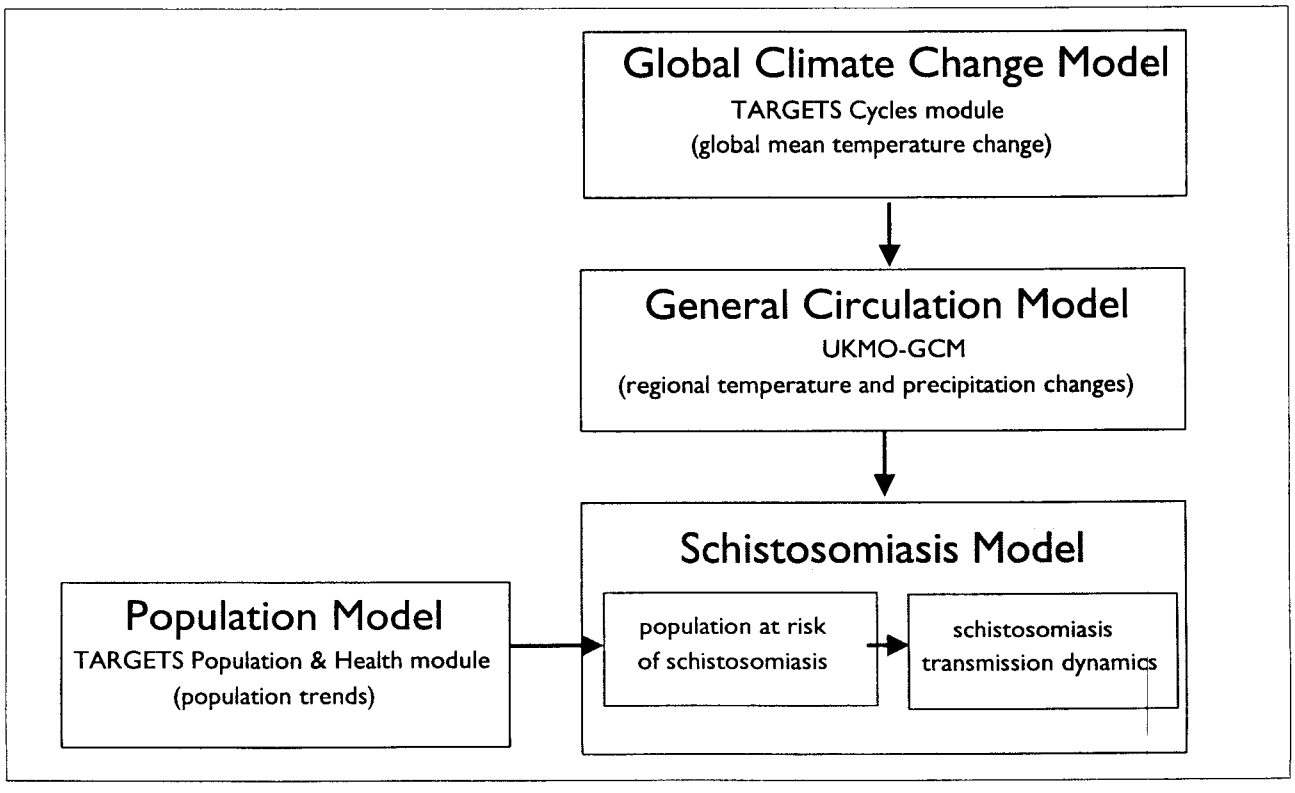
This study assesses the effects of a projected change in temperature and precipitation on the schistosome snail and the development of the parasites inside this vector. If the impact of a human-induced climate change on schistosomiasis transmission is to be understood, the entire cause-effect chain must be described and analyzed comprehensively. The systems approach seems to be an appropriate method to capture the complexity of the interrelationships between the climate factors, demographical and behavioural factors in the human population and snail and schistosome characteristics. The systems analysis not only studies the components of the various (sub)systems, but also the interactions and processes between them, rather than focusing on each subsystem in isolation (Rotmans *et al.*, 1994). Given the complexity of the systems under consideration, and the relative ignorance about the basic processes and interactions that determine their dynamics, the systems approach can help to foster understanding of the causal relationships between a human-induced climate change and demographical changes and schistosomiasis prevalence.

3.2 Model structure and dynamics: position within TARGETS

The structure of the study to assess the impact of anthropogenic climate change on the distribution and incidence of schistosomiasis is illustrated in *Figure 3.1.*

The schistosomiasis model is an integral part of the TARGETS modelling framework, (Rotmans *et al.*, 1994), and combines inputs from various modules within TARGETS (see also Figure 1.1). In our simulation, the main climate factors which have a bearing on the transmission of schistosomiasis are temperature and precipitation, i.e. factors derived from the global Cycles model (Elzen den, *et al.*, 1995). Estimates of climate change effects, other than temperature changes, are at best provisional and data for other environmental factors which influence the distribution of schistosomiasis are limited.

Figure 3.1: Key elements of the model to assess the impact of a climate change on the incidence of schistosomiasis.



Population figures are derived from the Population and Health model (Niessen and Rotmans, 1993; Niessen, *et al.*, 1995). Depending on the prevailing climate, a section of the world population is at risk of contracting schistosomiasis. The schistosomiasis model describes the transmission of the disease in the population at risk, and calculates prevalence and incidence figures (see *Figure 3.2*).

The period adopted for simulation runs on the schistosomiasis systems is 110 years, from 1990 to 2100, and thus differs from the simulation period of 200 years (1900-2100) for the Cycles and Population and Health modules of TARGETS. The various modules will be described in the sections which follow.

3.3 The cycles module

The Cycles module simulates the biogeochemical and physical processes that result in the cycling of carbon, nitrogen, phosphorus, and sulphur between the atmosphere, terrestrial biosphere, lithosphere (soils) and hydrosphere (rivers and oceans). The global elements cycle model triggers, amongst other, the climate assessment model in TARGETS, which compromises an atmospheric chemistry model, a radiation model and a climate model (Elzen den, 1993; Elzen den *et al.*, 1995). Using the method proposed by Santer *et al.* (1990), the simulated global mean temperature changes are converted into time-dependent series of changes in regional seasonal temperature and precipitation by standardizing the output of a General Circulation Model (GCM).

The GCM used in this study is that developed by the UK Meteorological Office (Wilson & Mitchell, 1987), using a grid resolution of 5° latitude by 7.5° longitude, with a climate sensitivity of 5.2. This equilibrium mean global temperature change of 5.2°C that would eventually occur if the CO_2 level were doubled, falls outside the current uncertainty range (1.5-4.5 C) projected by the IPCC (1990, 1992). Although our projected changes in schistosomiasis transmission will inevitably be more pronounced than those generated in experiments using less sensitive GCMs (see Martens et al. (1995b) for an analysis with a transient GCM), the general pattern of these changes will not be influenced significantly. The baseline climatology relies upon precipitation and temperature data for the period 1951-1980.

3.3.1 IPCC scenarios

In this study, climate scenarios published by the International Panel on Climate Change (IPCC) in 1990 are used, namely: the Business-as-Usual (BaU) scenario, and the Accelerated Policies (AP) scenario. The BaU-scenario assumes that few or no steps are taken to limit greenhouse gas emissions. Energy use and the clearing of tropical rainforests continue to increase, and fossil fuels, notably coal, remain the world's primary energy sources. The emissions of the major greenhouse gases, CO_2, CH_4 and N_2O, increase apace, and CO_2 emissions in particular are doubled within forty years. The Montreal protocol for ozone-depleting substances is not tightened up and the participation of the developing countries is assumed to be only 85%. While the emissions of the CFCs decline, the emissions of the alternatives, e.g. HCFC-22 are assumed to increase continuously. In contrast, according to the AP-scenario the development and market penetration of renewable energy sources and nuclear energy is strongly encouraged. This results in a decline in CO_2 emissions related to fossil fuels after the year 2000, such that by 2100 the levels are half of those in 1985. The reinforced Montreal Protocol seeks the phasing out of CFCs by the year 2000. As a result of new agricultural practices, emissions of CH_4 and N_2O start to decline in the middle of next century. Deforestation is assumed to have been halted by the turn of the century and a net increase of forested acreage through large-scale reforestation programmes is envisaged. For a more detailed description of these two scenarios, see the IPCC (1991) publication.

3.4 The population and health module

The Population and Health module simulated changes in fertility, mortality and morbidity under varying socio-economic and environmental conditions (Niessen *et al.*, 1995). The global Population and Health module includes a demographic subsystem, two causal subsystems, i.e. health services and determinants, and two effect subsystems, i.e. diseases and deaths. In this study, future population figures are used, calculated by the Population and Health module, based on UN projections (Zachariah & Vu, 1988). Global population reaches 9.5 billion by 2050 and 10.4 billion by 2100 with most of the growth in the currently developing countries. The human population at risk to schistosomiasis is defined for five age classes (from 0-14, 15-44, 45-64, 65-74 years old, and an age group of 75 years and over).

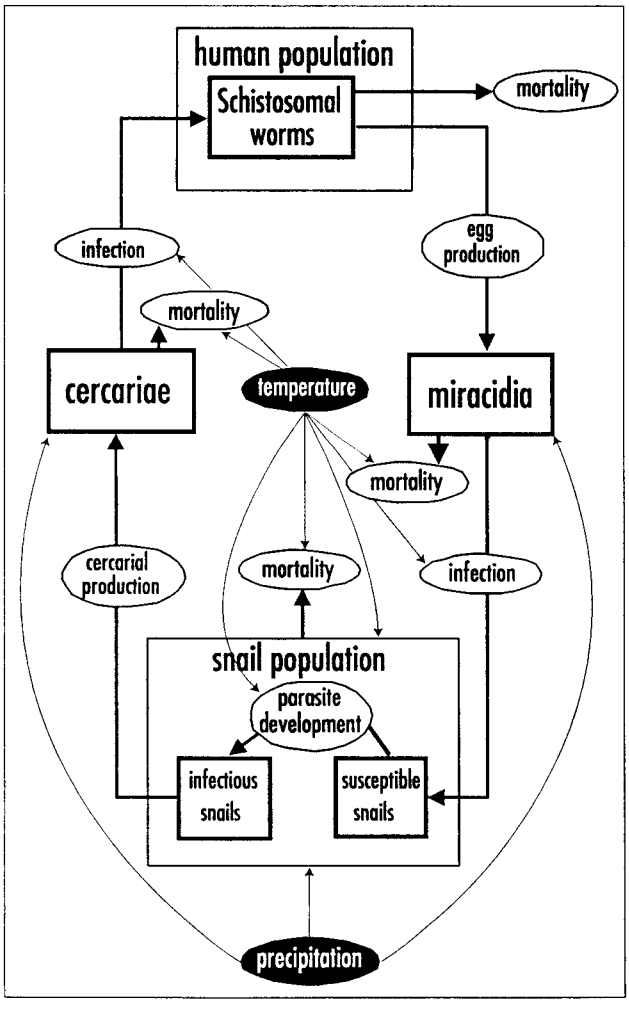
3.5 The schistosomiasis model

Many models concerned with the transmission dynamics of schistosomiasis have been developed,

varying in degree of geographical detail, input requirements etc. (e.g. Rosenfield *et al.*, 1977; De Almeida Machado, 1979; De Vlas *et al.*, 1992). For this study, the general deterministic model of Anderson & May (1991), which summarizes the main themes of the research on mathematical models (see e.g. MacDonald, 1965; May, 1977) and includes variables in the transmission cycles which are highly temperature dependent, appeared to be the most suitable.

The general structure of the schistosomiasis system is depicted in *Figure 3.2*. The incidence of schistosomiasis depends on transmission rates between human and snail and vice versa, such rates being subject to changing climatic conditions. Having been infected, a person runs a standard risk of schistosomiasis disease, since the general level of medication use is, and probably will remain, low in the populations concerned. We used a series of differential equations to describe transmission between the human population at risk and the snail population. In Anderson & May (1991) a full description of the derivation of most of the formulas described below can be found.

Figure 3.2: Systems diagram of the main population and rate processes involved in the life cycle of the schistosome and the influence of temperature and precipitation.



3.5.1 *Prevalence in the human host*

The epidemiological measure of prevalence and average intensity of infection is the summarized statistic of the frequency or probability distribution of parasite numbers per human host. In the case of schistosomiasis the distribution is highly aggregated in form so that a few individuals harbour the majority of the parasite population. The prevalence of infection (the proportion infected), P, is expressed as:

$$P = 1 - (1 + MWB/k)^{-k} \quad (1)$$

where MWB is the mean worm burden (see section 3.5.2) and k the aggregation or clumping parameter.

Parasite aggregation may arise as a consequence of a variety of factors (Anderson & Gordon, 1982). These include heterogeneity in exposure to infection (due to, for example, social, environmental or behavioural factors), differences in susceptibility to infection (due to, for example, nutritional factors), or to variability in parasite survival within the human host (due to, for example, genetic factors and development of immunity). We used average values of 0.3, 0.05, 0.03, 0.03 and 0.03 for parasite aggregation for the five age classes (from 0-14, 15-44, 45-64, 65-74 years old, and an age group of 75 years and over), respectively[1] (Anderson & May, 1991).

3.5.2 *Worm burden*

Relative to the life expectancy of the adult worm, the life spans scales of the miracidia, cercariae and snails are all short. By assuming that densities of miracidia, cercariae and snails are in equilibrium, we are able to derive a single equation for change in mean worm burden (MWB)with respect to time:

$$\frac{dMWB}{dt} = \mu_{worm} * MWB * \left(\frac{\frac{1}{2} * T_{sh} * T_{hs} * MP}{\frac{1}{2} * T_{hs} * MP * MWB + 1} - 1 \right) \quad (2)$$

[1] Epidemiological studies in endemic areas indicate that the prevalence and intensity of infection rise during the first 15 years of life, followed by a decline suggesting a gradual diminution of infection coupled with the development of resistance (WHO, 1993).

where μ_{worm} is the per capita death rate of the mature worm (the average life expectancy of the adult schistosomiasis worm is assumed to lie between 3-5 years (Warren *et al.*, 1993); in our model the life-expectancy is assumed to be 4 years, so μ_{worm} = 0.000685 day^{-1}) and T_{hs} and T_{sh} characterize the transmission from human to snail and snail to human, respectively. The factor 1/2 arises from the assumed 1:1 sex ratio of male to female parasites. *MP* represents the probability that a female worm is fertilized within the human host. This probability of fecundity approaches unity in most endemic areas (Anderson & May, 1985).

3.5.3 *Transmission from human to snail*

The transmission from human to snail can be expressed as:

$$T_{hs} = \frac{k_1 * e^{-\mu_{SLS} * LP} * RI_{snail} * N_{human}}{\mu_{SS} * (RI_{snail} * N_{snail} * + \mu_{mir})} \quad (3)$$

The probability of an egg producing a miracidium which infects a susceptible snail is $k_1 * IR_{snail} / (IR_{snail} * N_{snail} + \mu_{mir})$, thus depending on the per capita snail-infection rate of the miracidia (IR_{snail}), the snail density (N_{snail}) and the death rate of the miracidia (μ_{mir}). N_{human} is the human density, $e^{-\mu SLS * LP}$ is the proportion of latent snails that survive to release cercariae (μ_{SLS} is the death rate of the susceptible and latent snails and *LP* is the latent period of the parasite inside the snail), and μ_{SS} is the death rate of the shedding snails. Furthermore, k_1 is a constant (=0.002) representing factors which are assumed to be independent of temperature changes, and which, in the formula for the transmission from human to snail, include: the rate of egg production by female worms, the rate of cercarial production per snail, and human sanitation habits.

3.5.4 *Transmission from snail to human*

Likewise, the transmission from snail to human is described as:

$$T_{sh} = \frac{k2 * RI_{human} * N_{snail}}{(RI_{human} * N_{human} * + \mu_{cer})} \quad (4)$$

$k_2 * RI_{human} / (RI_{human} * N_{human} + \mu_{cer})$ is the probability of a cercaria infecting a given human host, whereby RI_{human} is the rate at which cercariae establish themselves within the human host (infection rate) and μ_{cer} the cercarial death rate. Like k_1 above, k_2 (=0.001) is a constant representing factors which, in our study, are assumed to be independent of temperature changes, and which, in the transmission from snail to human, include: water contact rates and the average exposure time of the human population to contaminated water, the rate of cercarial shedding per infected snail and the death rate among mature worms

3.5.5 *Epidemic potential*

A unit of measurement which encapsulates many of the important processes in the transmission of infectious diseases is the basic reproduction rate (R_0). In the case of the transmission of macro-parasitical schistosomiasis worms, R_0 is the average number of female offspring produced throughout the lifetime of the female parasite. To put it in simple terms, if R_0 exceeds the value one the disease will proliferate indefinitely; if its value is less than unity the disease will gradually die out.

Vector density is one of the parameters involved in the basic reproduction rate which is strongly related to local environmental conditions. The change in the prevailing schistosomiasis vector numbers over time varies greatly between species, being determined by numerous biological and physical factors, such as the availability of species-specific breeding sites. A reliable estimate of the change in vector abundance over large areas as a result of temperature, precipitation and humidity changes is impossible to arrive at in an aggregated model such as ours. Nevertheless, the basic reproduction rate allows us to calculate the critical density threshold of vector populations necessary to maintain parasite transmission. The critical density for schistosomiasis parasites may be expressed as the human density (N_1) multiplied by the snail density (N_2):

$$N_1 N_2 = k_1 * k_2 * \frac{\mu_{mir} * \mu_{SS} * \mu_{cer} * (\mu_{SLS} + \frac{1}{LP})}{(RI_{snail} * RI_{human} * \frac{1}{LP})} \quad (5)$$

We defined the epidemic potential (*EP*) of schistosomiasis as the reciprocal of the host density threshold (Anderson & May, 1991). This epidemic potential is a key summary parameter which is used as a comparative index in estimating the effect on the *potential* risk of schistosomiasis represented by a change in ambient temperature and precipitation patterns, as simulated using the GCM referred to above. A high epidemic potential indicates that despite a smaller vector population, or alternatively, a less potent vector population, a given degree of endemicity may be maintained in a given area.

4. CLIMATE AND SCHISTOSOMIASIS: QUANTIFYING THE RELATIONS

4.1 Introduction

The snails, i.e. the intermediate hosts, which transmit schistosomiasis, live in a variety of natural habitats in which conditions may vary widely, ano their distribution and population dynamics are probably governed more by abiotic than by biotic factors (Southwood, 1977). Among the most important possible abiotic influences on the transmission cycle of schistosomiasis are temperature and precipitation. While some of the effects of environmental change on vector-borne diseases are highly local in character, others, in particular the effects of rising temperature, are more general (i.e. not place-specific) (Bradley, 1993). (However, in any body of water, snails and schistosome larvae may still be able to select a micro-environment in which the temperature is optimal for survival (Jordan *et al.*, 1980)).

The influence of climatic conditions on the schistosome snail and parasites has been addressed by numerous studies, in most of which attention is restricted to the effect of temperature on a single species of parasite or vector. Although separate detailed data on each species is usually lacking, we assume that the relationships between climate parameters (in particular temperature) and specific parasite and vector characteristics hold for all species. In the case of schistosomiasis, the empirical relations between temperature and transmission dynamics are consistently defined with respect to *water* temperature. As the thermal conditions of shallow waters usually reflect the ambient temperature of the air (Baptista *et al.*, 1989), our simulations use ambient air temperature as an approximation of the water temperature. In *Figure 4.1* the relationship between temperature and transmission parameters, which will be discussed in the next sections, are depicted. *Table 1* summarizes the main temperature dependent parameters used in our model.

4.2 The effects of temperature

4.2.1 Miracidia and cercariae

The probability that miracidia successfully infect snails, and cercariae humans respectively, is determined by the longevity of their respective larval stages and their infection rates (Prah & James, 1977; Anderson *et al.*, 1982).

4.2.1.1 Larval longevity

Miracidia and cercariae are short-lived, non-feeding organisms which rely on relatively large glycogen reserves. The duration of the viability of these larval stages depends on the volume of these storehouses of energy. Extrinsic factors, and especially temperature, which stimulate the use of the glycogen curtail larval viability (Jordan & Webbe, 1982). *S. mansoni, S. japonicum* and *S. haematobium* miracidia respond to different temperatures in a very similar manner, surviving longer at moderate temperatures than at high or low ones. Optimum temperatures for survival hover around 15°C (Prah & James, 1977). It is assumed that the same relationship between temperature and longevity applies in the case of cercariae. As the latter generally live longer, their longevity is 1.5 times the life span of miracidia (see *Figure 4.1a*). The death rate in the larval stages is 1 divided by the longevity.

Table 1: *Some important temperature-dependent parameters in the schistosomiasis model.*

Parameter		Value in model
μ_{mir}	(death rate miracidia)	bell-shaped function of T
μ_{cer}	(death rate cercariae)	bell-shaped function of T
RI_{snail}	(infection rate of miracidia)	linear function of T
RI_{human}	(infection rate of cercariae)	curvilinear function of T
LP	(latent period parasite)	hyperbolic function of T
μ_{SLS}	(death rate non-shedding snails)	linear function of T
μ_{SS}	(death rate shedding snails)	linear function of T

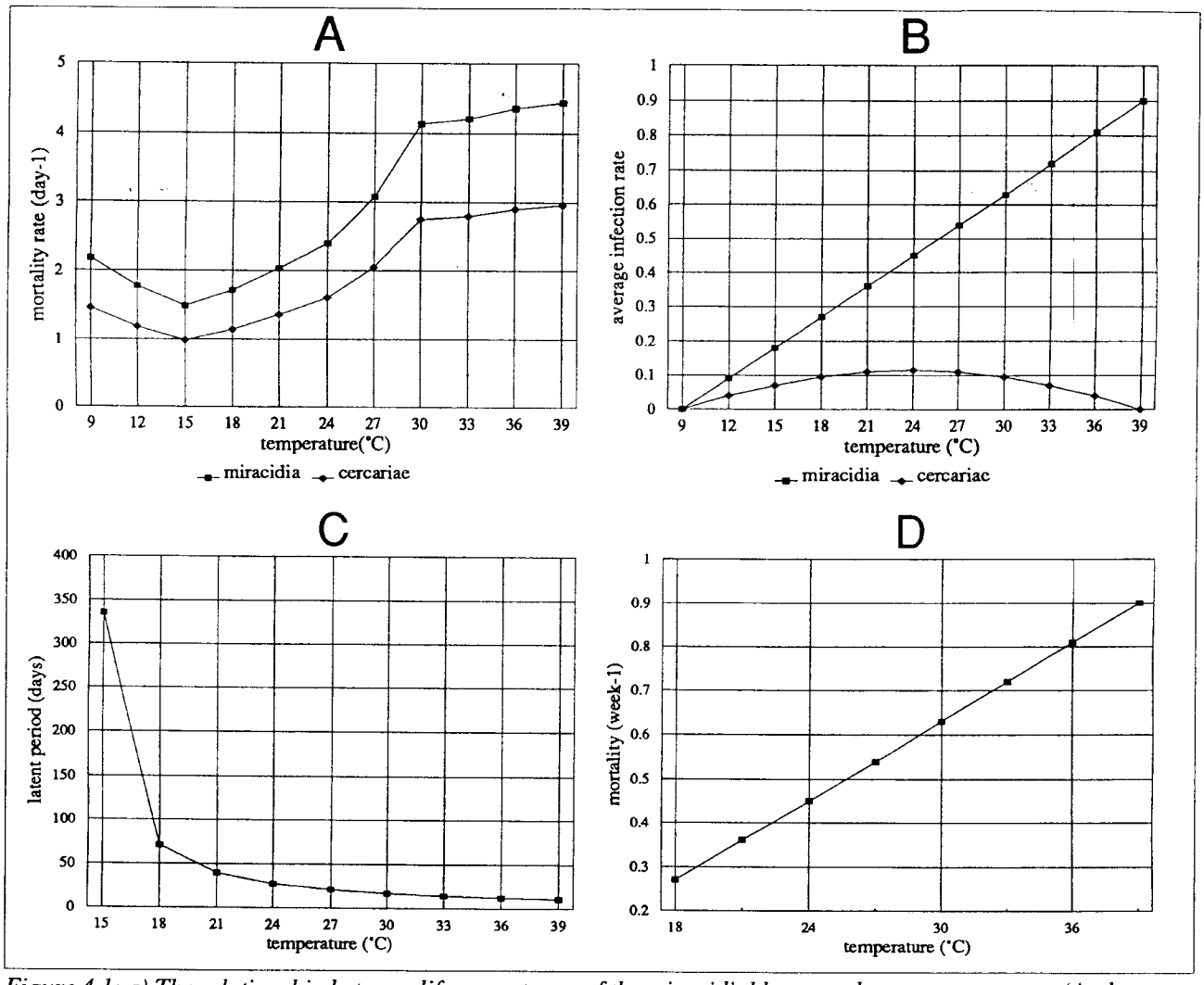


Figure 4.1: a) The relationship between life-expectancy of the miracidial larva and water temperature (Anderson et al., 1982). b) The relationship between the average infection rate (RI) of the miracidia and cercariae and water temperature (Purnell, 1966). c) The relationship between the latent period of infection (LP) and the temperature (-Plüger, 1980). d) The relationship between the per capita adult mortality rate and mean water temperature (Woolhouse and Chandiwana, 1990).

4.2.1.2 Infection rate

The effect of temperature on the infectivity of miracidia and cercariae has been recorded in several studies (e.g. Standen, 1952; Stirewalt, 1954; Purnell, 1966; Ghandour, 1976; Prah & James, 1977; Anderson *et al.*, 1982). The effect of temperature on the ability of cercariae to infect humans is not identical to the effect on miracidia in infecting snails. At low temperatures, both miracidia and cercariae move slowly, so the chance of infection during the larval stages is low. As temperature increases, both snail and miracidia become more active, leading to more favourable conditions for contact between the miracidia and the snail. The infection rates for all three species of miracidia increase as temperature rises, while infection does not take place at all at temperatures below 9°C. The cercariae achieve a maximum infection rate at 24-27°C. A linear function describes the relation between the infection rates achieved by miracidia and temperatures up to the thermal death point of the snails (39°C). In the case of cercariae, the infection rate increases curvilinearly to a peak of 24°C and then decreases symmetrically (see *Figure 4.1b*) (Purnell, 1966).

4.2.2 Snail population

Besides affecting infection rates (as described above), rainfall and temperature are also likely to influence other factors which play a role in determining the prevalence of infection within snail populations. In particular, temperature will influence snail survival rates and the duration of the latent period of

infection within the snail (Anderson & May, 1979).

4.2.2.1 *The latent period of the parasite inside the snail*

The rate of larval development is critically dependent on temperature. When temperatures are rising, larval development rate increases for all three parasite species, independent of the species of snail in which larval development occurs (Anderson & May, 1979). Below approximately 15°C larval development is, in general, completely inhibited, while above 39°C snail and parasite death begin to occur rapidly. The relation between temperature and the latent period of the parasite inside the snail can be expressed as the following temperature sum (Plüger, 1980):

$$LP_{schis} = \frac{DD}{T - T_{\min}} \qquad (6)$$

,where LP is the latent period of the schistosomes inside the snail (days), DD the number of degree days (i.e. the accumulation of temperature units over time) needed for schistosome development inside the snail (= 268 °C day), T the water temperature (-between minimum (14.2°C) and maximum temperature (39°C) for parasite development) and T_{min} the minimum temperature needed for parasite development (= 14.2°C) (*Figure 4.1c*). The intermediate host, i.e. the snail, is able to survive greater fluctuations in temperature than the parasite which is developing inside it. This stage is thus highly sensitive to climatic conditions and can be considered as the 'weakest link' in the transmission chain insofar as temperature is concerned.

4.2.2.2 *Snail mortality*

Temperature affects the complex relationship between snail mortality and infection rate. Infection tends to reduce the life expectancy of the snail by a factor of 3, so the change in infection rate as temperature fluctuates indirectly determines snail mortality. Moreover, temperature as such also directly influences snail mortality, since it increases as temperature rises. Natural mortality rates may vary between 0.087 week^{-1} (*B. truncatus* (Chu *et al.*, 1966)) and 0.607 week^{-1} (*B. glabrata* (Sturrock & Webbe, 1971)). We used a linear relationship between the per capita adult snail mortality rate and mean water temperature, as adopted by Woolhouse and Chandiwana (1990) for *B. globosus* in Zimbabwe:

$$\mu_{SS} = c + m * T \qquad (7)$$

,where μ_{SS} *is* the death rate of the shedding snails, c (=-0.374) and m (=0.0329) are constants, and T is the mean water temperature (*Figure 4.1d*).

4.2.2.3 *Snail density*

Snail populations are subject to wide variations over time and among localities. Consequently, it is very difficult to arrive at an estimate of snail numbers in a specific area. Furthermore, the change in snail numbers over time varies greatly between species and is determined by numerous environmental, bionomic and physical factors. Important among these are, for instance food availability, velocity of flow and ionic state of the water and nature of vegetation. Although snail densities are likely to be influenced by climate changes, the actual change in snail abundance in large areas as a result of temperature and precipitation change is almost impossible to estimate satisfactorily. Snail density is therefore assumed to be constant (see also sections 3.5.5 and 5.4.1).

4.3 Rainfall

Rainfall, or the lack of it, plays a crucial role in schistosomiasis epidemiology. Rainfall determines the duration of desiccation, and the snails are affected by fluctuations in the water level as well as by the rapid increase in the velocity of flows of water after heavy rains (Sturrock, 1973). On the other hand, the introduction of large-scale irrigation schemes has also reduced the significance of local rainfall in vector-borne disease epidemiology to some extent. However, because a shortage of rainfall may be a limiting factor in the ability of the vector to breed, we have assumed that a minimum seasonal average amount of precipitation (1.5 mm per day) is needed for mosquito and snail development. Arid areas are thus excluded from our model simulations of vector-borne disease transmission.

5. RESULTS

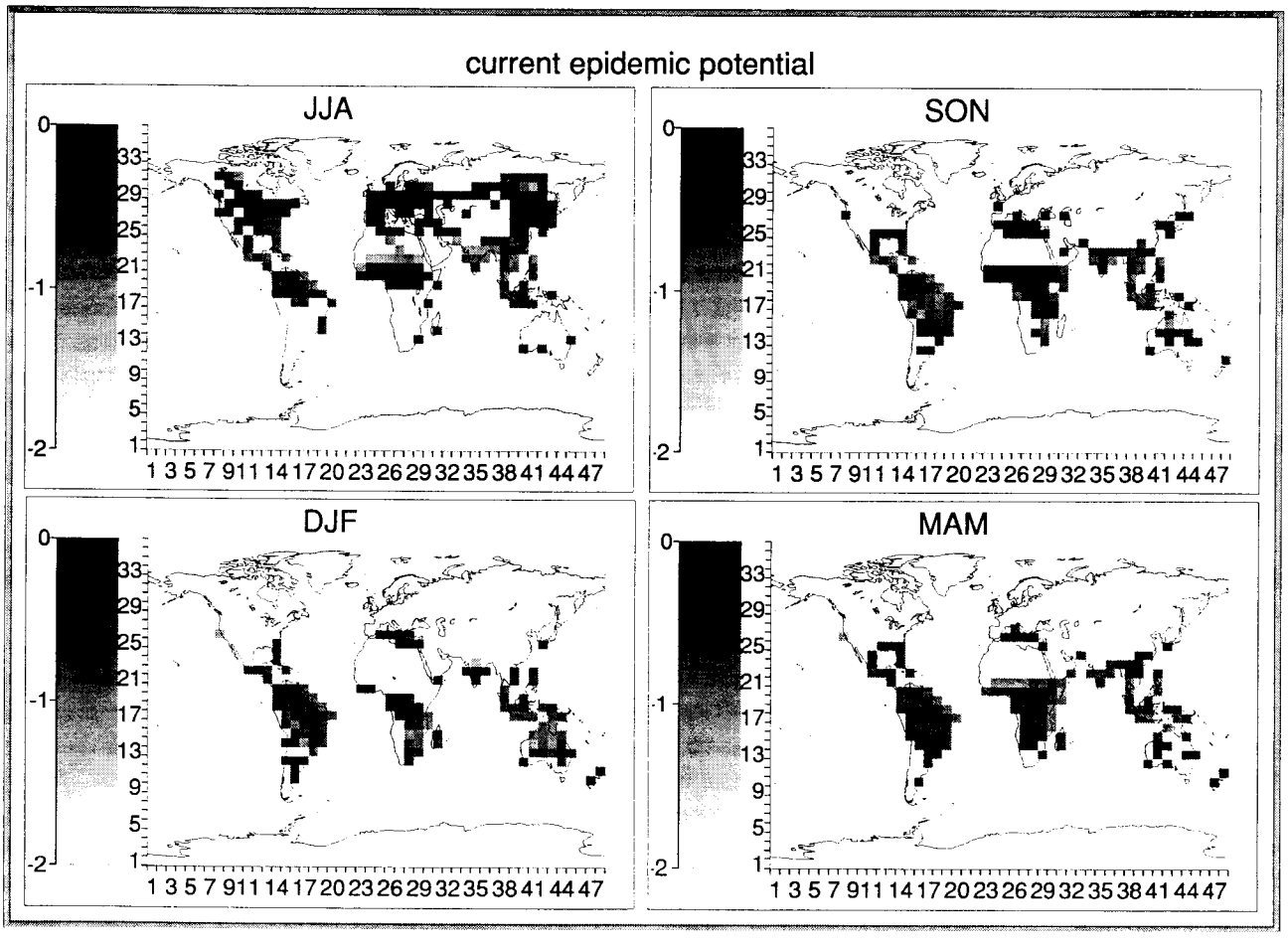
5.1 Introduction

Our efforts to assess the possible effects of climatic changes on schistosomiasis have been informed two complementary approaches. The one involved estimating the possible spatial shift in areas amenable to schistosomiasis transmission, using the epidemic potential as a comparative index, while the other concentrated on possible changes in the world population at risk and prevalence numbers of schistosomiasis. We shall discuss these in turn.

5.2 The shift of potential schistosomiasis areas

The epidemic potential as defined in formula 5 (section 3.5.5) is used as a comparative index to estimate the effect on schistosomiasis transmission potential of a change in average seasonal temperature and precipitation patterns (the seasons being December, January and February (DJF); March, April and May (MAM); June, July and August (JJA); September, October and November (SON)), as estimated using the UKMO-GCM (see section 3.3). Since there are no reliable field estimates of k_1 and k_2 available, the *absolute* values of *EP* should be treated with caution. Nevertheless, the *relative* differences in *EP* over time still hold.

Figure 5.1: Potential schistosomiasis risk areas in 1990, calculated from average temperatures and precipitation for the months March, April and May (MAM), June, July and August (JJA), September, October and November (SON), and December, January and February (DJF). (Expressed as the logarithm of the epidemic potential ($^{10}log(EP)$))



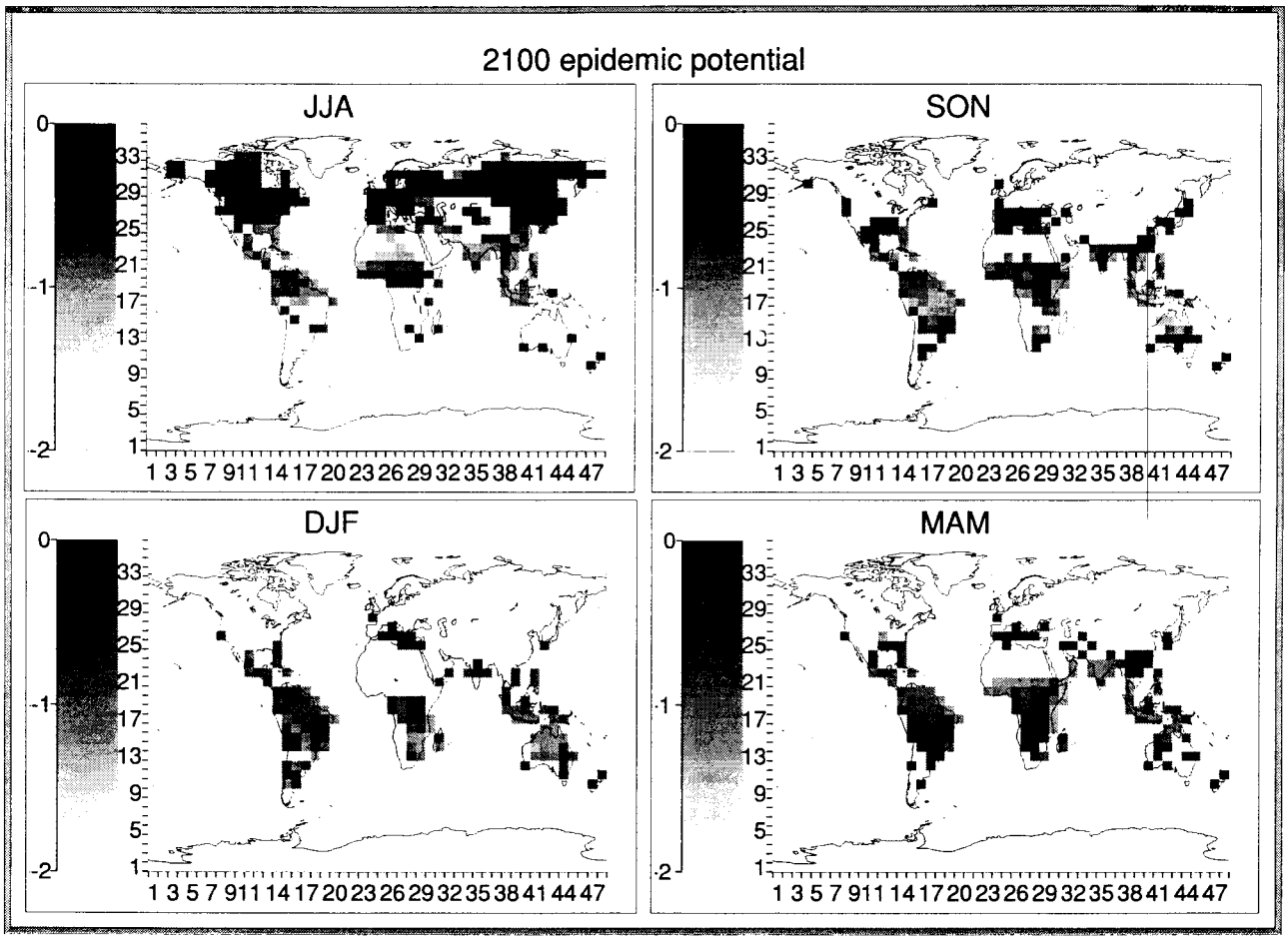


Figure 5.2: Potential schistosomiasis risk areas in 2100 (calculated as in Figure 5.1), with climate changes according to the BaU-scenario.

Figures 5.1, 5.2 and *5.3* depict the global distribution of the potential risk areas and the estimates of absolute limits of the current potential geographical extent of schistosomiasis transmission as arrived at using the UKMO-GCM. Besides the actual current endemic schistosomiasis areas in Africa, South-East Asia and South America, potential schistosomiasis areas simulated include Europe, North America and large parts of Asia. The three Figures show that an expansion of the geographical areas susceptible to schistosomiasis transmission is to be expected as the climate changes. The main changes, relative to 1990, would occur in the areas with temperate climates in which vectors capable of transmission already occur, but development of the parasite has hitherto been inhibited by temperature (*Figure 5.4*). In 1990, in Europe, North-America, and large parts of Asia, transmission would only have been possible during the summer-months (June, July and August). The other seasons are too cold for transmission to take place. However, a global mean temperature increase of approximately 3°C, consistent with the BaU-scenario, would cause large parts of the above-mentioned regions to be susceptible to transmission during two or more successive seasons (in these regions the winter remains still too cold for transmission). At the periphery of several endemic regions in Africa and South-America, seasonal transmission may become perennial. In Algeria, for example, snails tend to shrug off their schistosome infections during the winter, but as soon as global temperature increases, surviving snails may act as intermediate hosts for schistosomiasis throughout the year.

In other parts of the currently endemic areas, an increase in temperature would decrease the transmission potential of the parasite, since temperatures are likely to exceed optimum levels, thus increasing snail, miracidia and cercariae mortality rates. In some areas, perennial transmission may therefore become seasonal (*Figure 5.1* and *5.2*).

Comparing the potential geographical extent of the disease to the actual distribution indicates that the simulation of future risk areas must be interpreted to

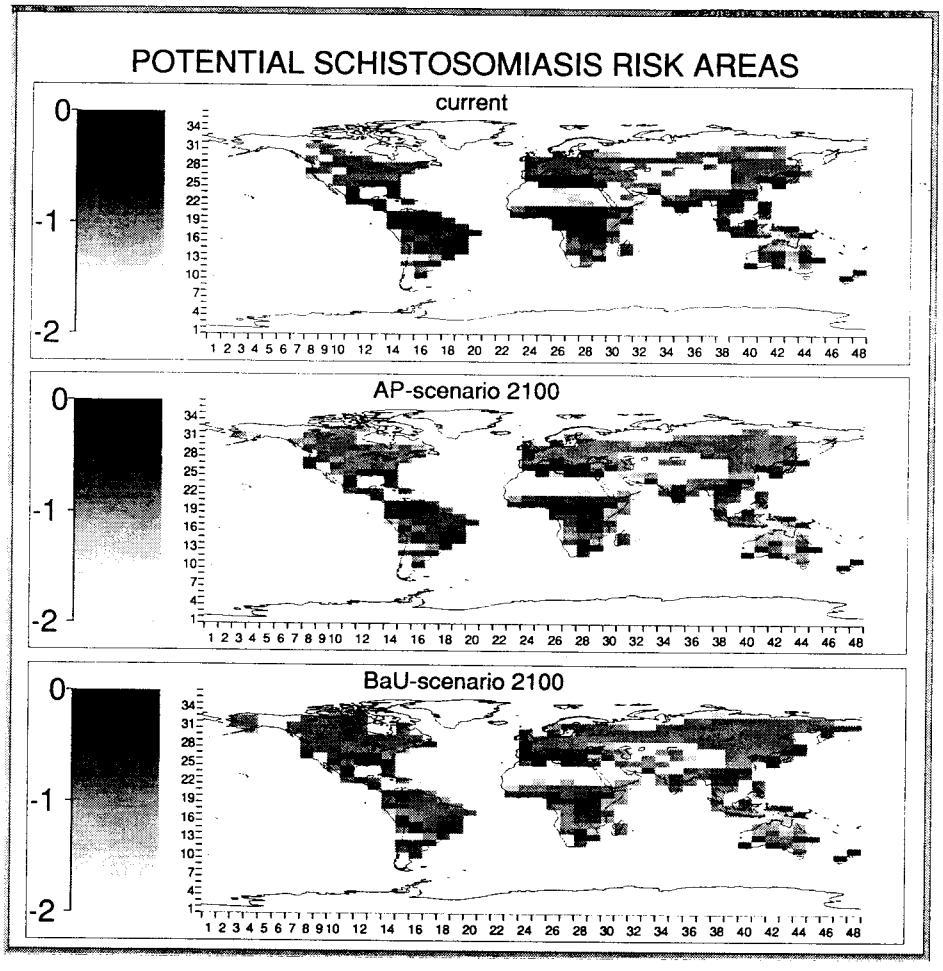


Figure 5.3: Potential schistosomiasis risk areas in 1990 (current) and 2100, with climate changes according to the AP- and BaU-scenario. (Expressed as annual averages of the logarithm of the epidemic potential ($^{10}log(EP)$))

take account of local conditions and developments. In tropical and sub-tropical regions, climatic conditions are already favourable for vector breeding and reproduction, resulting in vector densities which exceed the critical value during large parts of the year. However, in other regions disease transmission is absent, although climatic factors, and in particular temperature, are apparently conducive to transmission. Effective vector control measures, the treatment of infected individuals, and the specific characteristics of the human and/or vector population may explain this anomaly.

5.3 Change in global schistosomiasis prevalence

It would seem a demanding task to model the change in worldwide prevalence of schistosomiasis which is due to climate changes. Not only because dissimilar populations respond differently to any specific changes in transmission rates, but also because prevalence will to a significant extent depend on other, more local, factors. Nevertheless, we have attempted to generate a number of plausible projections of changes in schistosomiasis prevalence resulting from climate changes.

Although it is hardly possible to foresee whether *potential* risk areas are likely to become endemic in the future, for illustrative purposes we assumed that the present endemic areas will be supplemented by the areas in which: a) the simulated annual average epidemic potential increases by a factor greater than two, or b) increases by a factor greater than five, both relative to 1990 (provided that the intermediate hosts invade the previously unfavourable areas and are able to compete successfully for survival). A doubling of the epidemic potential or an increase of the epidemic potential by a factor five means that the vector density needed to establish an endemic situation decreases with a factor two or by a factor five, respectively. To account for regional differences in

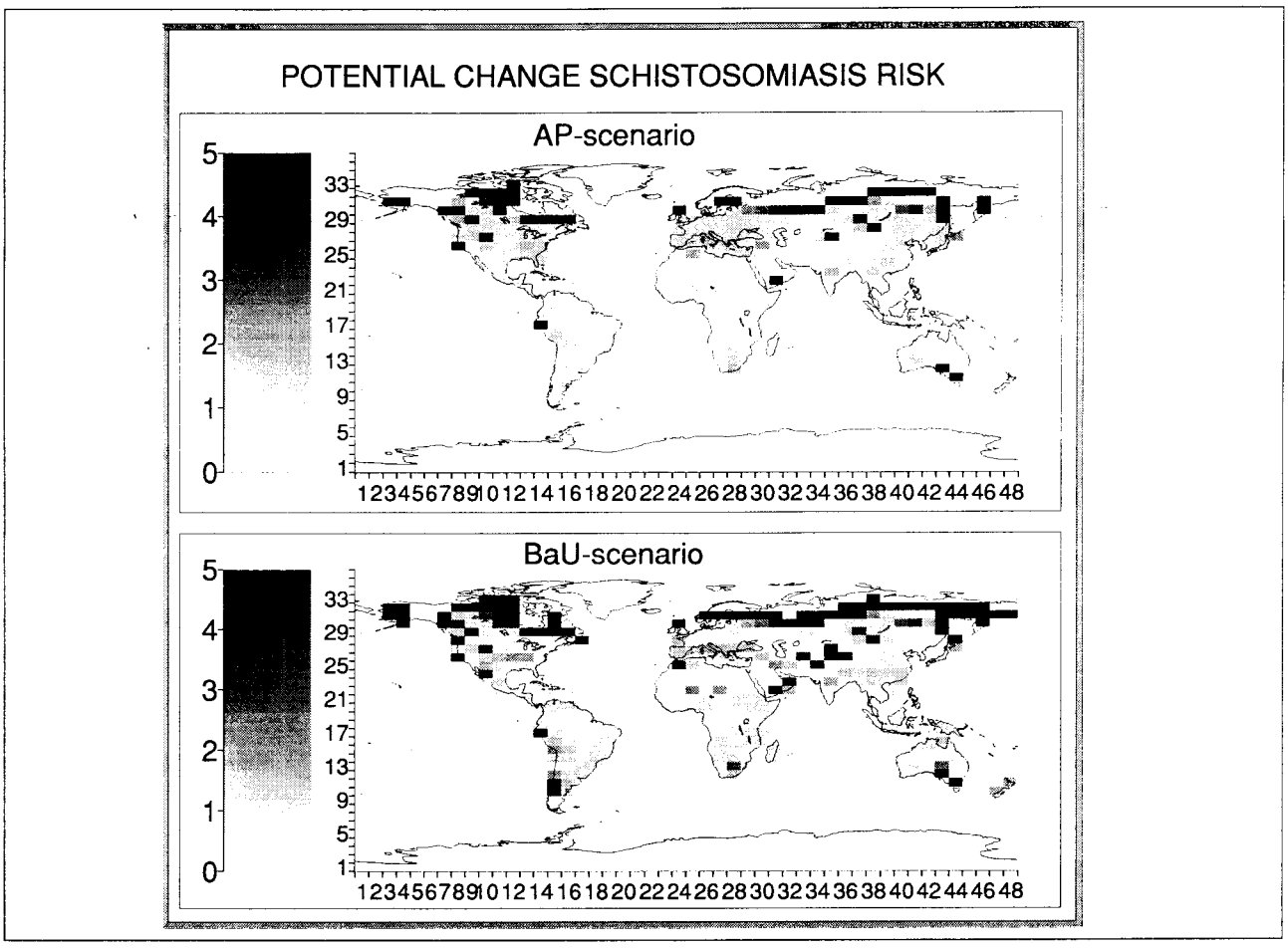


Figure 5.4: Change in potential schistosomiasis risk in 2100 as compared with the year 1990 for the AP- and BaU-scenario.

climate in these areas, we used the climate data derived from the grid cells which roughly correspond to these areas. We restricted ourselves to the changes in epidemic potential in less economically-developed areas, as most developed countries will be in a position to take mitigating measures as transmission potential increases. The population is assumed to be evenly distributed in the areas under consideration, an assumption which enables us to employ changes in regional transmission rates in simulating prevalence changes in the total population at risk.

Figure 5.5 shows the effect of a human-induced climate change on schistosomiasis prevalence and the population at risk of schistosomiasis transmission. In the absence of climate change, an increase in prevalence is to be expected due to population growth. Within the endemic areas, on average, an increase in temperature may decrease the average reproductive potential of the parasite. Hence, an increase in the temperature in endemic schistosomiasis areas would decrease prevalence rates in some of the present endemic areas, although the transmission potential of schistosomiasis will probably remain high enough for endemicity to remain established.

However, if the wider areas become endemic, the effect of a human-induced climate change on world prevalence will be more pronounced. In the year 2100, the world population at risk of schistosomiasis transmission is estimated to increase with 0.1-1.3%, depending on the climate scenario employed. Assuming that population doubles up to a total of ~9.5 billion by the year 2050 and ~10.4 billion by 2100, and that the population is evenly distributed in the areas under consideration, we can estimate the *supplementary* schistosomiasis prevalence and the number of people at risk due to anthropogenic climate change. Around 2050, according to the BaU-scenario, the number of people at risk may increase up to ~40 million people, which amounts to an increment in prevalent cases of some 5 million. As the year 2100 approaches, the BaU-simulation has it that the additional number of people at risk of schistosomiasis transmission may number some 130 million people with an increase in prevalence of 20 million, compared with the increase in prevalence from about

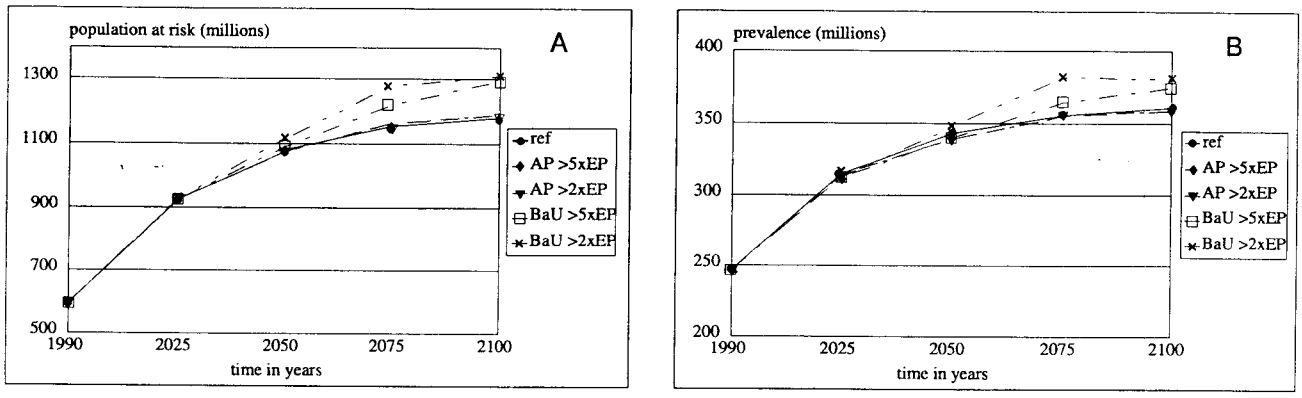


Figure 5.5: Effect of a human-induced climate change on A) the world population at risk of contracting schistosomiasis and B) schistosomiasis prevalence, simulated for the reference scenario (population growth in the absence of climate changes) and AP- and BaU-scenario. (Assuming that present endemic areas will be suplemented by the areas in which the epidemic potential increases more than twice or five times (>5x EP, >2x EP).)

250 million in 1990 to 360 million in 2100 due to population growth alone. These estimated numbers emphasize the potential influence which climate change may invoke on vector-borne transmission in the future. Table 2 summarizes the change in the population at risk of contracting schistosomiasis and the prevalence of the disease as simulated by the model.

5.4 Assumptions, modelling strategies and uncertainties

5.4.1 Aggregation level

Modelling the impact of climate change on the complex schistosome transmission cycle on this high level of aggregation is beset with numerous uncertainties so that several assumptions need to be con-

Table 2: Change in world schistosomiasis population at risk and prevalence due to climate changes, compared with reference scenario (= no climate changes). Estimates are rounded off to one tenth procent [2]

scenario	change in average global temperature(°C)[3]		change in population at risk(%)[4]		change in prevalence (%)[4]	
	2050	2100	2050	2100	2050	2100
AP >5x EP	0.95	1.32	0.0	0.2	0.0	-0.1
AP >2x EP	0.95	1.32	0.0	0.2	0.0	-0.1
BaU >5x EP	1.61	3.12	0.2	1.1	0.0	0.1
BaU >2x EP	1.61	3.12	0.4	1.3	0.1	0.1

[2] Absolute population at risk in the reference scenario is ~1080 million in 2050 and ~1180 million in 2100; absolute prevalence reference scenario is ~340 million in 2050 and ~460 million in 2100.

[3] Compared with 1990.

[4] An increase in the population at risk of, for example 1.3 % means that the population at risk increases from 11.3 % of the world population in 1990 to 12.6 % of the world population in 2100; an increase in prevalence of 0.1 % means that numbers of people infected with the schistosomiasis parasite increase from 4.7% in 1990 to 4.8 % in 2100.

sidered. In this section some of the major assumptions pass the revue and a simple, straightforward sensitivity analysis is performed on some major model parameters.

An important assumption is that all snail and schistosome species have similar characteristics. For instance, susceptibility to infection varies within each species and sometimes within each species. However, in general, the shape of the curve describing the relation between temperature, mortality and infection rates and parasite development is similar. A further simplification is the use of mean temperatures which have been averaged seasonally, although daily temperature may fluctuate by several degrees. Given the uncertainties inherent in projections of climate changes, it does not seem feasible to run the model on data which accommodates daily (minimum and maximum) temperatures.

A number of effects of temperature on snail and human proclivity are not taken into account. For instance, changes in snail longevity and reproduction rates will influence the age structure of the snail population, which will in turn influence the mean mortality and the mean physical size of the snail population, as well as snail numbers, which thus implies changes in transmission rates of the parasites. Furthermore, climate changes may affect human behaviour as well (reduced water contact rates during the cool season), but changes in human behaviour resulting from temperature increases are not considered here. Neither is the indirect effect of climate changes on transmission dynamics (e.g. changes in irrigation practices, desertification) taken into account in the model. Since, in many areas the prevalence of schistosomiasis is closely linked to irrigated agriculture, predicted increase in rice cultivation in new areas may lead to a further increase in this disease (Hunter *et al.*, 1993). The effect of precipitation changes is included by means of a cut-off point for schistosomiasis transmission, implying an oversimplification of the complex relations between temperature, precipitation, humidity and snail densities. Next versions of the model will include a more detailed analysis of the importance of these interactions. Furthermore, we have assumed that the number of people lacking an adequate water supply and sanitation remains constant (Gleick, 1993).

Thus it seems that it would hardly be possible to quantify *all* of the climate change impacts on the transmission cycle of schistosomiasis. The model therefore describes the *direct* effects of a human-induced climate change on major parameters in the transmission cycle of schistosomiasis.

5.4.2 Uncertainties

The estimation of direct climate change impacts on vector-borne disease incidence is beset with uncertainties regarding the geographical characteristics, time-path and magnitude of climate changes, as well as uncertainties associated with schistosomiasis disease dynamics.

GCMs have been shown to simulate current temperature reasonably well, although they do not simulate precipitation accurately. Neither can they reliably project changes in climate variability, such as changes in the frequency of droughts, which also could have a significant effect on vector-borne disease transmission. Nevertheless, GCMs currently provide the most advanced means of projecting potential future climate consequences (IPCC, 1990). The use of two different climate change scenarios captures a range of possible effects and sets limits on the associated uncertainties.

Our estimates of epidemic potential likewise incorporate several uncertainties. Ideally, the epidemic potential should be calculated separately for each vector and parasite species in a given location. Since epidemic potential is most sensitive to changes in intermediate host mortality rates and incubation time of the parasite, the effects of different values of snail longevity and minimum temperature requirements for parasite development vis-à-vis epidemic potential are illustrated in Figure 5.6. A more elaborate sensitivity and uncertainty analysis will be performed in the near future.

In the case of schistosome species, the increase in epidemic potential associated with the increasing infective potency of the miracidia and the shortening of the latent period of the schistosome parasite at higher temperatures are cancelled out by the increase in mortality rates among both miracidia and cercariae, as well as among snails.

The distributions set out in *Figure 5.6* indicate that, in temperate climates, small increases in temperature can result in large increases in epidemic potential, irrespective of the values chosen for the mortality rate or minimum temperatures assumed for parasite development. It should be noted that, although the maximum values for the epidemic potential of schistosomiasis are found in the ranges 16-18°C, the actual transmission intensity also depends on vector abundance. The optimal temperature for a population of schistosome snails to breed and reproduce is found to lie in the range 20-30°C (Jordan & Webbe, 1982). Therefore, if such a temperature range obtains while rainfall and humidity are optimal for vector

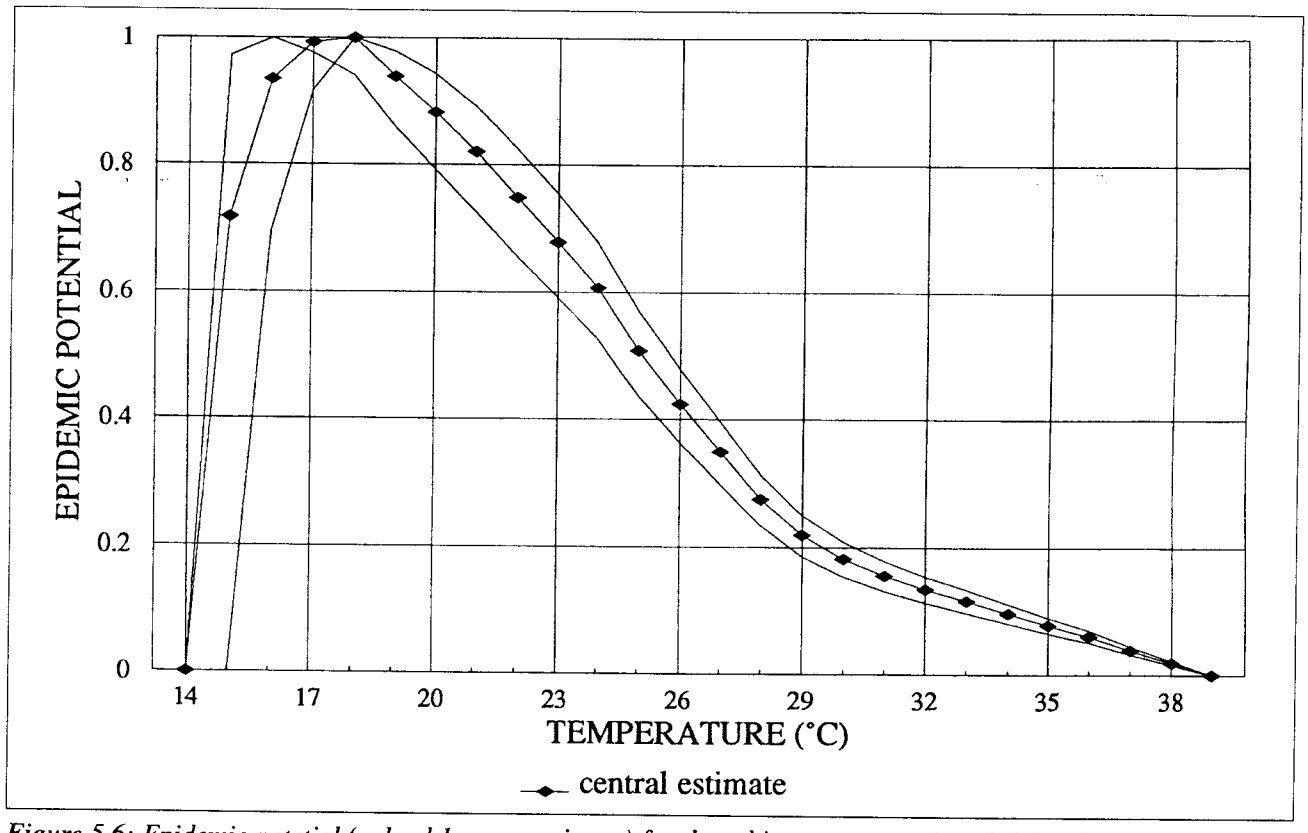


Figure 5.6: Epidemic potetial (valued 1 as a maximum) for the schistosome parasites (left-hand curve $\mu_1(25°C)$ = 0.1/week (Chu et al., 1966) and Tmin = 14°C; central estimate $\mu_1(25°C)$ = 0.44/week (Sturrock and Webbe, 1971) and Tmin = 14.2°C; right hand curve $\mu_1(25°C)$ = 0.6/week and Tmin = 15°C)

breeding, our analysis will probably underestimate the change in transmission potential of schistosomiasis vector populations. On the other hand, if the amount of rainfall (above the threshold limit of 1.5 mm/day) is not optimal for snail breeding and development, our results are likely to be distorted in that the changes in schistosomiasis risks have been overrated.

6. CONCLUSIONS AND DISCUSSION

Our projections of the impact of climate change effects on the extent and distribution of schistosomiasis depend on the climate scenario which is adopted. The analysis with both the AP and BaU climate change scenarios, although crude, suggests that in large parts of the world an increase in transmission potential of the schistosome snail population and an extension of the areas conducive to schistosomiasis transmission may occur. An increase in susceptibility to this disease in higher latitudes and altitudes can therefore be expected. In some parts of the present endemic areas, climatic factors may become less favourable for disease transmission. However, both climate change scenarios were found to imply an increase in the populations at risk of schistosomiasis.

It transpires that both in the case of schistosomiasis (this study) and malaria (Martens *et al.*, 1994, 1995a,b), as climate changes, the most vulnerable populations are those that live in the less economically-developed temperate regions. Comparing both studies shows that malaria is more sensitive to the direct effects of a change in temperature than schistosomiasis. In view of their high potential receptivity the highest risks of the introduction of malaria schistosomiasis transmission reside in the hitherto non-endemic regions which border on endemic areas. The increase of the total word population at risk of schistosomiasis, related to an extension of the present endemic areas, may number some 130 million people in the year 2100. An aspect of particular importance is the increase in epidemic potential at higher altitudes within endemic areas such as the Eastern Highlands of Africa or the mountainous region in Western China, where an increase in temperature of a couple of degrees may raise the epidemic potential sufficiently to transform formerly non-endemic areas into areas subject to seasonal epidemics. Given an unbalanced socio-economic development and that there are insufficient resources to take the adaptive and preventive measures which are required to deal with vector-borne diseases adequately, the potential effects of anthropogenic climate change must be taken seriously. In the current highly-endemic schistosomiasis areas, the prevalence of infection is persistently high, and will be only little affected by climate-induced changes in the factors which have a bearing on transmission.

The risk of renewed transmission of schistosomiasis in countries in which, until recently, loci of transmission were present (e.g. Japan and Portugal) will increase as more successive potential transmission seasons will occur. Given the fact that in these countries, effective control measures are economically feasible, it is not to be expected that human-induced climate changes would lead to a return of a state of endemicity in these areas. Increased vigilance in previously endemic, but not vector-free, areas will, however, be necessary.

When discussing the potential health effects of global climate changes, it is necessary to put them in perspective. First and foremost, changes in health associated with such environmental disturbance will be superimposed upon changes associated with socio-economic development, control programs and population growth. Secondly, in this study, one specific health impact of an anthropogenic induced climate change is being investigated separately although in many instances interactions between the various health effects of a climatic change are possible if not probable (e.g. synergism between infectious diseases and levels of undernutrition) and they may accumulate in vulnerable populations.

Furthermore, the total of global atmospheric changes (i.e. climate change, ozone depletion and air pollution) will jointly impact human health and these effects are likely to be synergetic. Examining the health impact of global environmental problems by dividing them into components may well be a useful initial approach to analyzing the contribution of the various impacts. However, if these problems are to be analyzed comprehensively, and where responses need to be developed, a holistic integrated approach would seem to be the most promising. To this end, analyses accounting for possible synergetic effects of climate change, ozone depletion and air pollution, against a background of changes in health investments and socio-economic developments, are currently being performed.

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Figure captions

Figure 1.1: Modular perspective of TARGETS.

Figure 2.1: Global distribution of schistosomiasis due to Schistosoma haematobium, S. japonicum and S. mekongi (Reprinted with permission from WHO (1993)).

Figure 2.2: Global distribution of schistosomiasis due to Schistosoma mansoni and S. intercalatum (Reprinted with permission from WHO (1993)).

Figure 3.1: Key elements of the model to assess the impact of a climate change on the incidence of schistosomiasis.

Figure 3.2: Systems diagram of the main population and rate processes involved in the life cycle of the schistosome and the influence of temperature and precipitation.

Figure 4.1: a) The relationship between life-expectancy of the miracidial larva and water temperature (Anderson et al., 1982). b) The relationship between the average infection rate (RI) of the miracidia and cercariae and water temperature (Purnell, 1966). c) The relationship between the latent period of infection (LP) and the temperature (Plüger, 1980). d) The relationship between the per capita adult mortality rate and mean water temperature (Woolhouse and Chandiwana, 1990).

Figure 5.1: Potential schistosomiasis risk areas in 1990, calculated from average temperatures and precipitation for the months March, April and May (MAM), June, July and August (JJA), September, October and November (SON), and December, January and February (DJF). (Expressed as the logarithm of the epidemic potential ($^{10}log(EP)$))

Figure 5.2: Potential schistosomiasis risk areas in 2100 (calculated as in Figure 5.1), with climate changes according to the BaU-scenario.

Figure 5.3: Potential schistosomiasis risk areas in 1990 (current) and 2100, with climate changes according to the AP- and BaU-scenario. (Expressed as annual averages of the logarithm of the epidemic potential ($^{10}log(EP)$))

Figure 4.3. The integrated population and health module

Figure 5.4: Change in potential schistosomiasis risk in 2100 as compared with the year 1990 for the AP- and BaU-scenario.

Figure 5.5: Effect of a human-induced climate change on A) the world population at risk of contracting schistosomiasis and B) schistosomiasis prevalence, simulated for the reference scenario (population growth in the absence of climate changes) and AP- and BaU-scenario. (Assuming that present endemic areas will be suplemented by the areas in which the epidemic potential increases more than twice or five times (>5x EP, >2x EP).)

Figure 5.6: Epidemic potetial (valued 1 as a maximum) for the schistosome parasites (left-hand curve $\mu_1(25°C)$ = 0.1/week (Chu et al., 1966) and Tmin = 14°C; central estimate $\mu_1(25°C)$ = 0.44/week (Sturrock and Webbe, 1971) and Tmin = 14.2°C; right hand curve $\mu_1(25°C)$ = 0.6/week and Tmin = 15°C)