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CLIMATE CHANGE AND MALARIA RISK

an integrated modelling approach

W.J.M. Martens
J. Rotmans
L.W. Niessen

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NATIONAL INSTITUTE OF PUBLIC HEALTH AND ENVIRONMENTAL PROTECTION

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SUMMARY

That the enhanced greenhouse effect may prove to influence human health will come as no surprise. One of the potential health consequences is a (re-)introduction of vector-borne diseases to certain regions. In tropical countries, such diseases are a major cause of illness and death. One of the most important vector-borne diseases in the world is malaria, which is associated with one of four species of parasite and transmitted by a mosquito vector. Climatic conditions, and temperature in particular, directly influence mosquito development, feeding-frequency and longevity of the mosquito, as well as the time in which the parasite develops inside the mosquito. Other environmental factors such as vegetation and breeding sites are indirectly influenced by climate conditions.

In order to assess the impact of an anthropogenic climate change on the transmission of malaria, an integrated assessment model has been developed. In this integrated model, the direct effects of a change in temperature and precipitation on the transmission potential of a mosquito population are assessed by means of the vectorial capacity (defined as the number of potentially infected contacts inflicted by the mosquito population per infectious person per day) and the related critical mosquito density (the minimum number of mosquitoes needed to maintain malaria at an endemic level). The most important parameters of the vectorial capacity and critical mosquito density are: the longevity of the mosquito, the frequency of taking blood meals from humans and the duration of the development of the parasite inside the mosquito. In this study, the effect of a human-induced climate change on human health is evaluated by assessing the change in malaria prevalence and disease burden. Furthermore, a sustainable development index, which is an aggregate of an environmental pressure indicator, a health indicator and a socio-economic development indicator is introduced and discussed. Such an index can be used to determine whether future projections are sustainable.

The simulation results indicate a worldwide increase of potential malaria risk and an extension of the areas conducive to malaria transmission. In the endemic malarious areas of the tropics and sub-tropics, malaria prevalence and consequently the number of years of healthy life lost due to malaria may increase, so that the target of zero increase in numbers of years of healthy life lost due to malaria is not achieved. The rate of temperature change is also expected to exceed the recommended target level of 0.1 °C per decade. In non-malarious areas the risk of introduction of malaria associated with imported cases of malaria increases to some extent as a result of the increasing importance of modern transport systems (e.g. air travel) in introducing malaria into receptive areas. However, sound interpretation of the change in malaria risk as simulated must be performed within the framework of local conditions and socio-economic developments.

1. INTRODUCTION

Human activities have reached a level at which their impact on the environment is global. The atmosphere, biosphere and hydrosphere are being perturbed in a manner which is likely to prove irreversible, mainly as the result of a rapidly expanding world population, industries which consume non-renewable resources and non-sustainable agriculture. Environmental problems which exist on a global scale include anthropogenic climate change, the depletion of the ozone layer, land degradation and topsoil erosion, water scarcity and depletion of groundwater, reduction of biodiversity, chemical pollution, and acid rain. Only recently has attention been paid to the possible consequences of such environmental problems for human health (Leaf, 1989; WHO, 1990; Doll, 1992; McMichael, 1993). A major conclusion which is common to these studies is that the impact of environmental disturbance on human health may become an important factor in determining the health status of the world population in the coming century.

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. The effects of climate change on human health may find expression in various ways. An increased incidence of heat waves will result in higher mortality and morbidity among vulnerable groups of people. The incidence of strokes and cardiovascular diseases associated with heat stress would increase. Other important effects on human health would be consequent upon any changes in agricultural viability and desertification as well as on large-scale migration in response to the rise in sea level. Such developments would have serious consequences vis-à-vis the production of food and the maintenance of public health. Climate change may well include the (re-)introduction of vector borne diseases. Temperature and precipitation changes might influence the behaviour and geographical distribution of vectors, and thus change the incidence of vector-borne diseases which are major causes of illness and death in most tropical countries (WHO, 1990).

One of the world's most important vector-borne diseases is malaria, and there are few infectious diseases which have as great an impact on the social and economic development of societies. At present, the distribution of malaria is mainly restricted to the tropics and sub-tropics, although before the Second World War malaria was a common disease in many temperate areas of the world (Bruce-Chwatt & De Zulueta, 1980). Malaria eradication campaigns and socio-economic development caused malaria to disappear from areas in which it had previously been endemic, although mosquito densities still allow transmission in these areas. The eradication of malaria would seem to be an impossible dream, and indeed, during recent years the situation has actually deteriorated in many areas (Clyde, 1987; WHO, 1992A, B and C). The incidence of malaria is determined by various factors: the abundance of *Anopheline* species, the propensity of the mosquitoes to bite human beings, the longevity of the mosquitoes and the rate at which the parasite in the mosquito develops. Anthropogenic climate change may directly affect one or more of these factors. Indirect climate change could have a significant (net) effect on other environmental factors such as vegetation and breeding site availability. Social and economic conditions also play an important role.

The aim of the present study is to analyze the impact of a given instance of climate change on malaria from an integrated perspective, not only in a qualitative sense, but also quantitatively. In other studies models have been used as tools for understanding various aspects of vector population dynamics and disease transmission (see MacDonald, 1957; Haile & Weidhaas, 1977; Molineaux & Gramiccia, 1980; Dietz, 1988). However, none of these attempted the comprehensive modelling of the cause-effect chain which is essential to a quantitative assessment of the impact of a climate change on malaria risk. Therefore, an integrated risk assessment model for malaria has been developed, which combines and integrates results from various research areas, such as climatology, epidemiology and entomology. The model can be used to study the changes in malaria transmission according to various socio-economic and climate scenarios and is applied to obtain a comprehensive quantitative

evaluation of the cause-effect chain.

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 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, health (Niessen & Rotmans, 1993) and socio-economic impact dynamics). 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: in section 2 the potential effects of an anticipated climate change on the occurrence of vector-borne diseases is described in some detail. Section 3 deals with the geographical distribution of malaria and outlines the main processes in its transmission. In section 4, a systems approach is applied to model the impact of a given instance of climate change on malaria epidemiology from an integrated perspective. A proposed index for a sustainable malaria policy is discussed in section 5 and a number of results are presented in section 6. The major findings resulting from the study are discussed in section 7.

2. CLIMATE CHANGE AND VECTOR-BORNE DISEASES

The occurrence of vector-borne diseases ranges from the tropics and subtropics to the temperate climate zones, and until relatively recent times, malaria was endemic in parts of Europe and North America (Bruce-Chwatt & De Zulueta, 1980). With a few exceptions, vector-borne diseases do not occur in the cold climates of the world. In *Table 2.1* some of the world's most important vector-borne diseases are listed.

The extent to which vector-borne disease transmission occurs in a specific area is determined by two factors: first, the presence of (an abundance of) vectors capable of transmitting the disease, and second the presence of the relevant parasite. Any factor influencing these two determinants hence influences disease transmission. In *Figure 2.1* an overview is given of the impact of a climate change on vector-borne disease transmission.

Direct effects of the anticipated changes in: global and regional temperature, precipitation, humidity and wind patterns resulting from anthropogenic climate change are the factors which have an impact on the vectors' reproduction habits

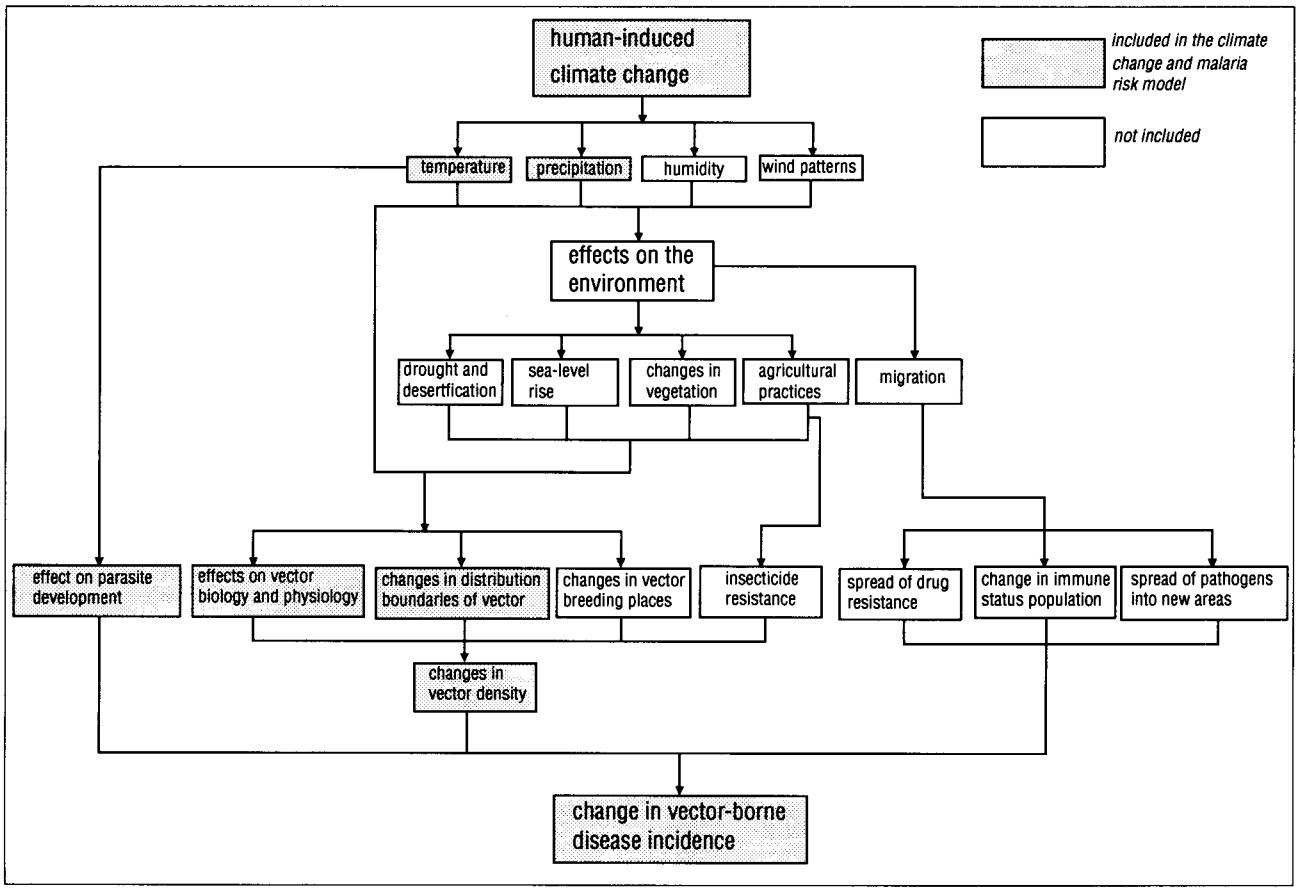
Table 2.1: Global status of the major vector-borne diseases in 1990 (source: WHO, 1990).

disease	vector[1]	populations at risk (millions)	prevalence of infection (millions)
malaria	mosquito	2200	270
schistosomiasis[2] (bilharzia)	snail	600	200
lymphatic filariasis	mosquito	90	90.2
onchocerciasis	blackfly	90	17.8

[1] Living agents transmitting diseases from one organism to another are called vectors. Diseases which involve disease-inducing agents (e.g. viruses, protozoa, flukes) being transmitted by means of a vector, are called vector-borne diseases.

[2] Strictly speaking, snails do not transmit schistosomiasis, and rather than being vectors, they are intermediate hosts.

Figure 2.1: Schematic diagram of the major climate change implications for vector-borne diseases.



and on their longevity, and are thus associated with changes in annual vector density. In general, the rate of development of a parasite accelerates as the temperature rises. An increase in temperature may therefore result in the completion of the life cycle of a parasite in areas in which previous temperatures were too low for the parasite to reach maturity.

Indirect effects of climate change include changes in vegetation and agricultural practices which are mainly caused by temperature changes and trends in rainfall patterns. These changes either promote or delay disease transmission by their association with increased or decreased vector density. Irrigated land (such as paddyfields) provides a suitable breeding ground for a number of vectors. Vector-borne diseases which are highly affected by changes in irrigation practices and in the distribution of irrigated areas include malaria and schistosomiasis (FAO, 1987). In areas in which extensive use is made of pesticides, resistance among vectors for insecticides is reported, with major consequences for disease transmission (Pant, 1988). Another indirect effect of climate change is associated with the rise in sea level and the resulting coastal flooding. The proliferation of brackish water lagunae influences the availability of habitat and either encourages or discourages vector species depending on whether they prefer brackish water. Generally speaking, drought and desertification, including a migration or extension of global desert belts, could be expected to decrease vector-borne disease transmission. After all, vector breeding generally relies upon an aquatic environment and drought conditions severely curtail the vector's longevity. Thus climate change may lead to a change in the location of habitats capable of supporting vectors. The migration of certain vectors to higher latitudes and altitudes can therefore be expected.

The influence which climate change is likely to exert on human populations may also play an important role in the dynamics of disease transmission. For example, the large-scale migration of populations from areas in which vector-borne diseases are endemic into receptive areas, a movement induced by rural impoverishment, and inevitably influenced by the dynamics of climate change, including the effects of sea level rise on low-lying coastal areas, will prove to be significant.

It is thus evident that major changes in the incidence of vector-borne diseases associated with a climate change might be expected, and that the manifestation of these changes is closely related to socio-economic development and the provision of health services (see also section 3.2.2.)

3. MALARIA

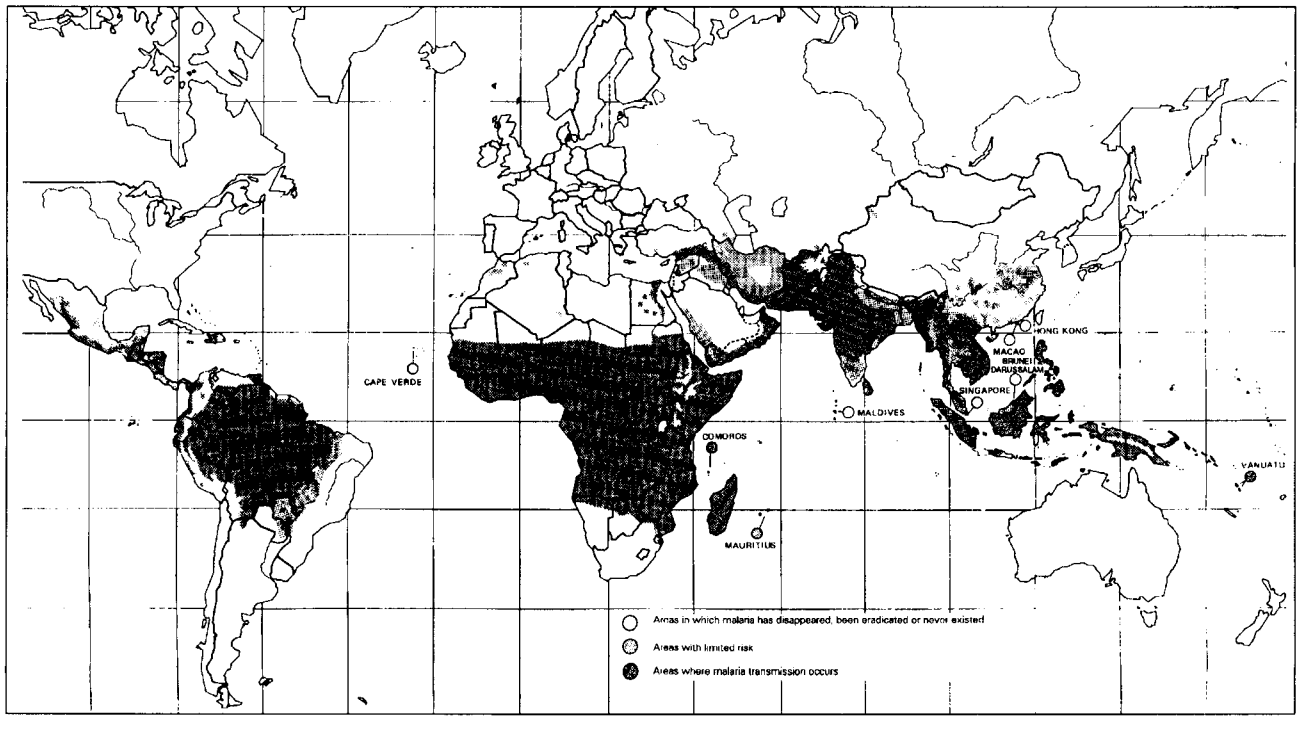
3.1 Geographical distribution

Some 270 million people are believed to be suffering from malaria and the disease is widely distributed in Africa, Asia, and Latin America (see *Figure 3.1*). At present, approximately 110 million clinical cases of malaria occur annually and more than one million people, mostly children, die. Accurate data is unavailable since the accuracy of reporting varies considerably, and in tropical Africa it is especially irregular and fragmentary. Reported cases are believed to represent a mere 2 to 8 percent of the actual cases (Nájera *et al.*, 1992).

The world's population can be classified according to the stability and endemicity of the malarial situation in which they live[1] (WHO, 1992A; Nájera *et al.*, 1992). Given a total world population (in 1990) of about 5,300 million, we may assert that 1,371 million people (27%) live in areas in which malaria never existed or has disappeared without antimalarial interventions. Areas which enjoy cooler climates (e.g. Canada) fall into this category. Some 1,617 million people (32%) live in areas in which endemic malaria was eradicated by the implementation of specific campaigns, and to which the disease has not returned. This region includes parts of Europe, North America and Australia. A population of the size of 1,599 million people (32%) lives in areas in which the endemic malaria has been considerably reduced or even temporarily eliminated, although transmission has reoccurred and the situation is either unstable or deteriorating. Large areas in South and Central America, South-East Asia and the Pacific belong to this category. Areas in which malaria transmission is highly intense are inhabited by some 474 million people (9%), and mainly concerns tropical Africa. Tropical Africa is estimated to account for more than 70% of all malaria cases (see *Figure 3.2*). Of the remaining cases, 75% are concentrated in 9 countries, namely: India, Brazil, Afghanistan, Sri Lanka, Thailand, Indonesia, Vietnam, Cambodia and China. In these

[1] In stable malarious areas, the prevalence of infection is persistently high and only little affected by natural or man-made change in the factors of transmission, whereas in areas of lower endemicity, the prevalence is very sensitive to even relatively small changes. Malaria is described as endemic when there is a measurable incidence both of cases and natural transmission over a succession of years. Stability increases with the endemic level (Molineaux, 1988).

Figure 3.1: Epidemiological assessment of the status of malaria in 1990 (from WHO, 1992C).



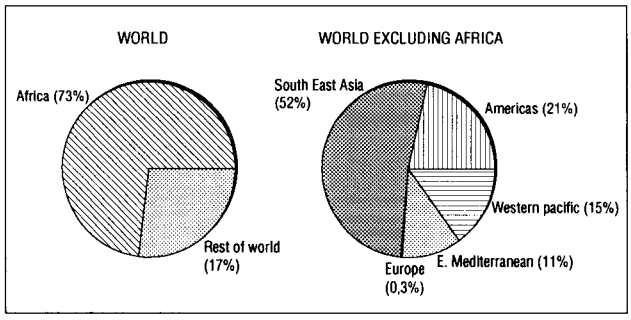


Figure 3.2: World malaria prevalence in 1990; for Africa 1989 data has been used (WHO, 1992A, B and C).

countries, malaria is often confined to specific regions and localities (WHO, 1992 A,B and C)[2].

The ability of the malaria parasite *P.falciparum* to resist antimalarial drugs is a worldwide phenomenon and the rapid evolution of this resistance in Africa, South-East Asia and South America threatens to hamper the provision of adequate treatment. Another important factor inhibiting malaria eradication is the development of resistance to insecticides among some of the most important malarial vectors. The regions in which resistance to insecticides is most pronounced are to be found in Africa, Central America and in West and South-East Asia (Bruce-Chwatt, 1980; Pant, 1988). The geographical distribution of malaria is thus far from uniform; there is a great variety of the transmission intensity of malaria between regions and also within a region the malaria situation may vary considerably.

3.2 Malaria transmission

Malaria is caused by one or more of four species of parasites of the genus *Plasmodium*, and the vector responsible for malaria transmission is the mosquito of the genus *Anopheles*. The life cycle of the malaria parasite involves transmission both from mosquito to man and from man to mosquito, effected by the bite of a female mosquito. The parasite multiplies within the mosquito by means of sexual reproduction, and following an incubation period of several days (depending on the temperature and the species of parasite), malarial parasites can be found in the salivary glands of the insect. When an infected mosquito bites a human host, saliva is also injected and parasites are thus transferred to (hitherto non-infected) people. In the human host asexual multiplication takes place. Having received an infective bite, there is an incubation period in the patient which varies between 10 and 40 days, depending on the species of parasite. Towards the end of the incubation period the infected person may suffer from headaches, pains in the arms and legs, backache, nausea and vomiting. The incubation period culminates in a severe attack which is caused by the destruction of infected blood cells and the release of toxins into the bloodstream. Infections involving *P.falciparum* are often associated with fatal complications (e.g. anaemia and cerebral malaria).

Although malaria can manifest itself throughout the world, the incidence shows marked regional variations, which depend upon four groups of interacting factors, namely: the human host, the malaria parasite, the mosquito as vector and the environment, whereby the latter is understood to subsume physical, biological, and socio-economic elements (Clyde, 1987). These factors will be discussed in the sections which follow.

3.2.1. The human host

Two main factors determine an individuals' propensity to succumb to malarial infection, on the one hand, genetic factors affect the ability of the parasite to penetrate and maintain itself within the erythrocyte, and, on the other, some degree of immunity may have been acquired by virtue of a previous history of infection. Among the genetic factors associated with protection against malarial infection are the sickle-cell trait and the Duffy factor (Molineaux, 1988). Those who have survived an attack of malaria acquire some degree of immunity to the disease. The number of parasites in the blood is lower and the infection may present few, if any, clinical symptoms. Consequently, in areas where malaria is rife, deaths from the disease occur mainly in the category of children aged between 6 months and five years. The high collective levels of acquired immunity of populations in these stable endemic areas reduce the likelihood that malaria epidemics will occur (Clyde, 1987).

3.2.2. The parasite

There are four species of the malaria parasite of the genus *Plasmodium*, namely: *P.falciparum*, the most common species in tropical areas and the most

[2] The figures in this section are based on publications by the World Health Organization (WHO, 1992A,B and C), which collates information on the number of malaria cases recorded by surveillance programmes. Malaria cases are commonly defined as cases requiring treatment which have been being confirmed by means of microscopy. In countries lacking microscopic facilities (i.e. most African countries), cases are generally diagnosed on clinical grounds.

dangerous clinically; *P.vivax*, which has the broadest geographic range including many temperate zones; and *P.ovale* and *P.malariae* which are less prevalent (Clyde, 1987). Virulence varies greatly from species to species. The ranking in decreasing order of virulence is as follows: *P.falciparum, P.vivax, P.malaria* and *P.ovale*. When left untreated *P.falciparum* does not survive in the human body for longer than two to three years, whereas infection by *P.vivax* may last between two and more than 11 years. The duration of infection by *P.ovale* and *P.malariae* may last 1 year and up to 53 years, respectively.

3.2.3. The mosquito

Malaria is transmitted to humans by female mosquitoes of the genus *Anopheles*. Male mosquitoes feed on plant juices and nectar rather than on blood, and therefore do not transmit the disease. The mosquito of the genus *Anopheles* belongs to a very large genus which includes hundreds of species throughout the world, although only 60 of these are actual or potential malarial vectors. Some species prefer to take their blood meals from animals (zoophilic) and thus transmit malaria to humans very rarely or do not live long enough to allow the parasites to multiply and to develop inside them, while in some species the parasites seem to be incapable of development. (Gillies, 1988). Although *Anopheles* mosquitoes occur most frequently in tropical or sub-tropical regions they are found in temperate climates and even in the polar regions during summer. In countries in which malaria has been eradicated, mosquito vectors capable of transmitting malaria nevertheless still exist. As a rule, *Anopheles* are not found at altitudes above 2000-2500 metres (Bruce-Chwatt, 1980).

There are four distinct stages in the life cycle of the mosquito: the egg, larval, pupal and the adult stage. In order to produce eggs a female mosquito must take a blood meal, and the eggs are normally laid after the blood meal has been digested. Development and survival during the larval and adult stages of the *Anophelines* depends on favourable climatic temperature, humidity and rainfall. Temperature governs the growth rate of a mosquito population by determining the time needed for one generation to develop. The optimum temperature for most malaria vectors is found in the range 20-30°C. Relative humidities in excess of 60% are preferred by vectors. Rain may prove beneficial to mosquito breeding if moderate, but if excessive may flush out the mosquito larvae.

3.2.4. The environment

The physical, biological, and socio-economic environment plays an essential part in the epidemiology of malaria, and some of the physical factors have already been mentioned above. Among the biological factors which play a role in malaria transmission are the presence of predators and the presence of domestic animals. Cattle may be employed as sources of blood and hence divert mosquitoes from feeding on people. When considering the connections between malaria and socio-economic variables, two apparently contradictory relations can be identified. Firstly, in the long run, social and economic development is associated with a downward trend in malaria. This is partly due to the effects of socio-economic development on the provision of health services, both curative and preventive. Socio-economic development can also suppress malarial prevalence independently of deliberate control measures (e.g. the enhancement of public hygiene, drainage and housing) as the cases of Europe and North America would suggest (Bruce-Chwatt, 1980). On the other hand, in the short run, development projects may lead to an increased incidence of malaria, in particular when such projects are divorced from social development (e.g. when they involve deforestation, irrigation, colonization of new territory etc.). It is important to bear in mind that there is a direct feedback from the incidence of malaria to the socio-economic development of a country. Some of the major elements of social and economic damage caused by malaria morbidity and mortality are (Wernsdorfer & Wernsdorfer, 1988):

- falls in the productivity of labour;
- loss of agricultural and industrial production;
- pressure on health services (e.g. costs of treatment and hospitalization);
- limitation of land use in areas with a high degree of malaria transmission;
- negative effects on tourism;
- educational absenteeism due to childhood illness.

This feedback may prevent many low-income countries from achieving an efficient level of malaria control and/or eradication programme and such countries could prove incapable of escaping from this vicious circle.

4. CLIMATE CHANGE AND MALARIA RISK: A SYSTEMS APPROACH

4.1 Introduction

If the impact of a human-induced climate change on malaria risk is to be understood, the entire cause-effect chain must be described and analyzed comprehensively. The systems approach seems to be the only approach capable of adequately reflecting the complexity of the interrelationships between the climate system, the mosquito system and the human system (Martens, 1993). 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 changing malaria risks.

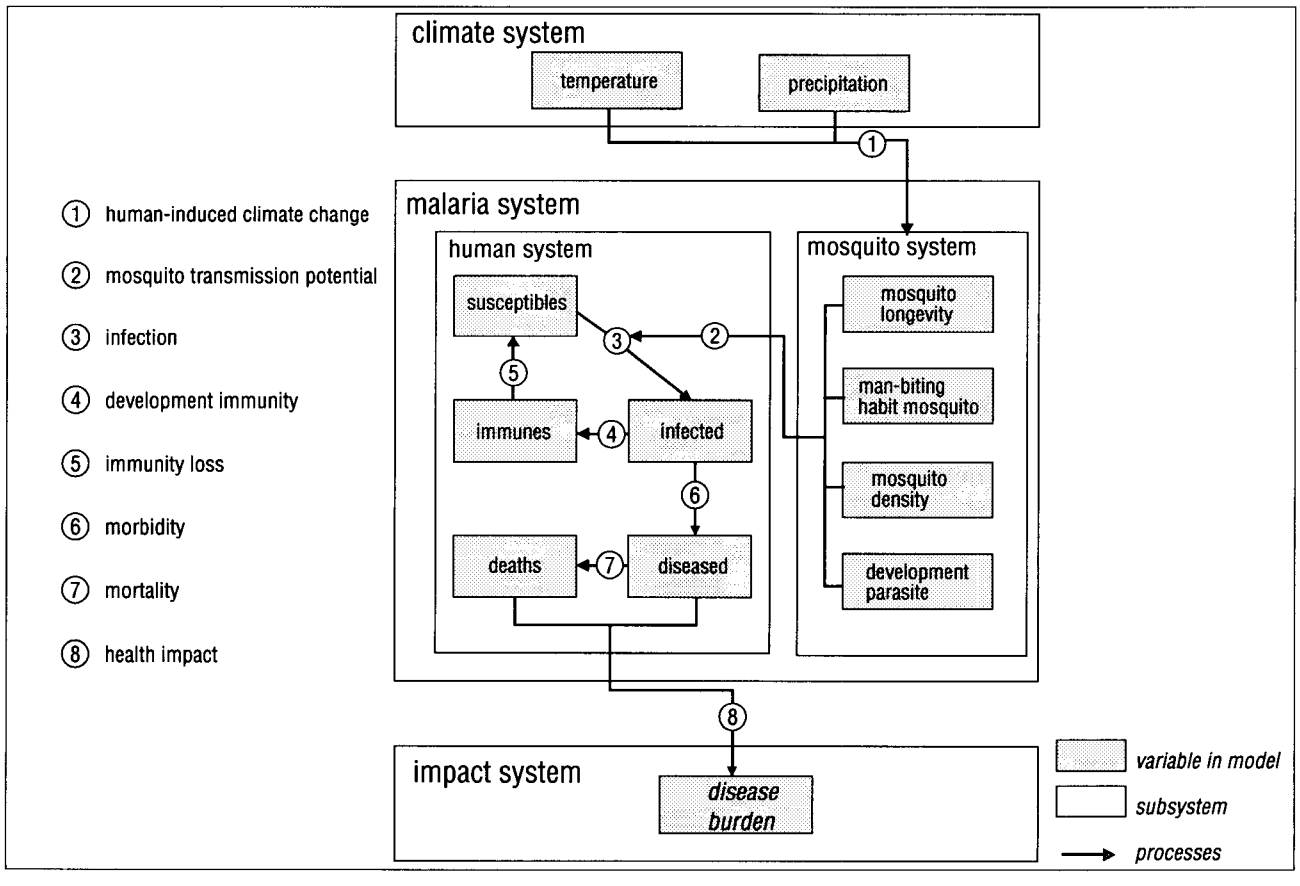
4.2 System structure and dynamics

The conceptual model designed to assess the impact of an anthropogenic climate change on the incidence of malaria is represented by a systems diagram, illustrated in *Figure 4.1.*

The model consists of several linked modules (i.e. systems), namely: the climate system, the malaria system (divided into a human subsystem and a mosquito subsystem) and the impact system. The systems are linked in a straightforward manner, the output of one system serving as an input to the next. The outputs and inputs, denoted by arrows, are state variables (temperature, precipitation, mosquito transmission potential, morbidity and mortality); Arrows, however, may also denote the transmission among the various classes of people, like in the human system. Consequently, the notation of the systems diagram is not homogenous.

The main climate factors which have a bearing on the malarial transmission potential of the mosquito

Figure 4.1: Systems diagram of the model designed to assess the impact of a climate change on the incidence of malaria.



population are temperature and precipitation, i.e. factors derived from the climate system. The interaction between the human system and the mosquito system determines the transition rates among the susceptible, the infected and the immune, respectively. The impact system is at the end of the cause-effect chain and yields rough estimates of the health impact of a climate change on malaria. This health impact is described by the relationships between inputs (morbidity and mortality) and output (disease burden due to malaria).

The period adopted for simulation runs on the malaria and impact systems is 110 years, from 1990 to 2100, and thus differs from the simulation period of 200 years (1900-2100) for the climate system. The various systems will be described in the sections which follow. An overview of the parameters used in the model is given in Appendix 1.

4.3 The climate system

The climate assessment module is a version of the Integrated Model to Assess the Greenhouse Effect (IMAGE version 1.6; Den Elzen, 1993), an improved version of the original model described by Rotmans (1990). IMAGE is a simulation model designed to develop scenarios of greenhouse gas emissions and to calculate and evaluate their effects on global mean temperature and sea-level rise. In essence, IMAGE is designed to study the entire cause-effect relationship with respect to climate change.

If a regional climatology (resolution grid of 10° latitude by 7.5° longitude) is to be derived from a global-mean temperature for a chosen future year, two inputs are required, namely: an observed baseline for mean seasonal temperature and precipitation climatology, and fields which indicate the magnitude of mean seasonal temperature and precipitation change for a given increment of global-mean warming. The baseline for the climatology was calculated from observed station data collected during the period 1951-80. The climate change fields are constructed from experiments with the UK Met. Office (UKMO) General Circulation Model (Mitchell *et al.*, 1990 using a procedure originally developed by Santer (Santer *et al.*, 1990).

4.4 The malarial system

The malaria module consists of two subsystems which interact with each other, i.e. the human and the mosquito subsystems. A description of both systems is given in the sections which follow.

4.4.1. Mosquito population

The dynamics of the mosquito population are much more rapid than human population dynamics, so the mosquito system can be considered in equilibrium with respect to changes in the human population. Following Garret-Jones (1964B), the entire mosquito population is incorporated in a single state variable, the vectorial capacity:

$$VC = \frac{m*b*c*a^2*p^n}{-ln(p)} \qquad (4.1)$$

where VC is the vectorial capacity, defined as the number of potentially infective contacts inflicted by the mosquito population per infectious person per day; where m denotes the density of the mosquito population in relation to man (number of mosquitoes/number of people), b the proportion of infectious bites on humans that produces a patent infection, c the proportion of bites by susceptible mosquitoes inflicted on infectious people that produce a patent infection, a the man-biting habit (number of blood meals taken from humans per mosquitoes per day), p the daily survival probability of the mosquito and n the incubation period of the parasite in the vector (in days).

The formula expresses the capacity of a vector population to transmit malaria, a fact which can be illustrated by the following derivation: an infectious person is bitten by $m*a$ vectors in one day; and among these bites a proportion c is actually infective; a fraction p^n of the infected vectors survives the parasite's incubation period; whereby the mean longevity of the mosquito population is $1/-ln(p)$ days, during which they feed on a persons per day, resulting in $a*b$ infections.

A number of basic assumptions are implied when the term vectorial capacity is used (Molineaux, 1988) namely a) that vectors die at a constant rate, independent of age; b) that longevity is unaffected by infection and c) that the probability of feeding on man is the same for all meals among all members of a given population of vectors. Dye (1986, 1990) pointed out that at best, an estimate of the vectorial capacity will be useful as a 'comparative index changing proportionally with the true vectorial capacity from site to site, from vector to vector, and within and between transmission seasons'. Nevertheless, vectorial capacity provides a means by which to study the effect of climate change on malarial epidemiology.

The parameters of the vectorial capacity, which will be described in the following sections, are

determined by characteristics of the parasite (n), the mosquito (m, a, c and p) and the human population (b) and are influenced by environmental factors (i.e. physical, biological and socio-economic factors). Climate affects the vectorial capacity in several ways. By inserting changing climatic conditions in vectorial capacity, a change in transmission potential can be simulated.

4.4.1.1. The proportion of bites resulting in an infection

There is a wide variation in the ability of different species of vectors to transmit different species of parasites (denoted by parameter c), an ability which is genetically determined. There is usually a degree of co-adaption between vectors and parasites from the same geographical area and this may afford some protection against imported parasites or vectors, although information on the ability of vectors to transmit malaria parasites is scarce (WHO, 1982; Molineaux, 1988). The probability that a human becomes infected when bitten by an infectious mosquito may depend on genetic factors, while immune processes may also affect b. A detailed description of these parameters is given by Nedelman (1984). The probability of a mosquito becoming infected with the parasite (c) and the probability of a human becoming infected when bitten by an infectious mosquito (b) do not depend on climate conditions, and is set to 1 in the simulations.

4.4.1.2. The duration of sporogony

The incubation period (duration of sporogony) in the vector must have elapsed before the infected vector can transmit the parasite. This latent period depends on the critical factors of species of parasite and the ambient temperature. The parasites develop in the vector only within a certain temperature range, whereby the minimum temperature for parasite development lies between 14.5 and 15°C in the case of *P.vivax* and between 16 and 19°C for *P.falciparum*, while the proportion parasites surviving decreases rapidly at temperatures over 32-34°C (Horsfall, 1955; Macdonald, 1957; Detinova, 1963). The relation between the incubation period and temperature can be expressed in the following equation (see *Figure 4.2*) (Macdonald, 1957):

$$n = \frac{DD_s}{T - T_{\min,s}} \qquad (4.2)$$

where n is the incubation period of the parasite inside the vector (in days DD_s the number of 'degree-days' required for the development of the parasite (=105 and 111 °C days for *P.vivax* and *P.falciparum*, respectively (Detinova, 1963)), T the actual average temperature (between $T_{min,s}$ and a maximum temperature of about 40°C; in °C) and $T_{min,s}$ the minimum temperature required for parasite development (14.5 and 16°C for *P.vivax* and *P.falciparum*, respectively).

4.4.1.3. The man-biting habit

The man-biting habit depends on the frequency with which the vector takes a blood meal and the frequency of these blood meals being taken from man.

Figure 4.2: The duration of sporogony of malaria parasites in the mosquito host in relation to environmental temperature, according to equation 4.2.

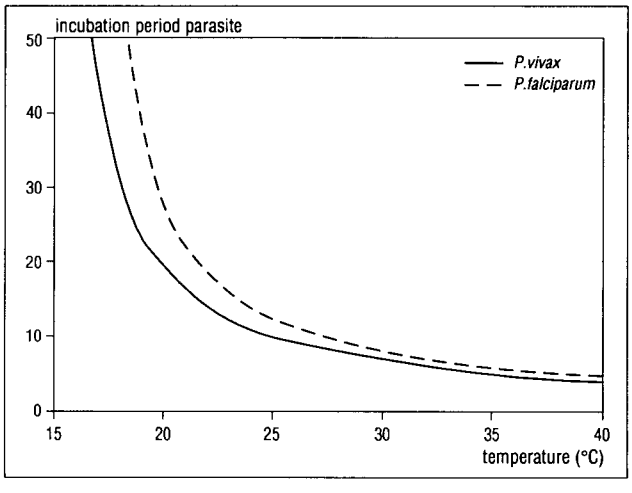
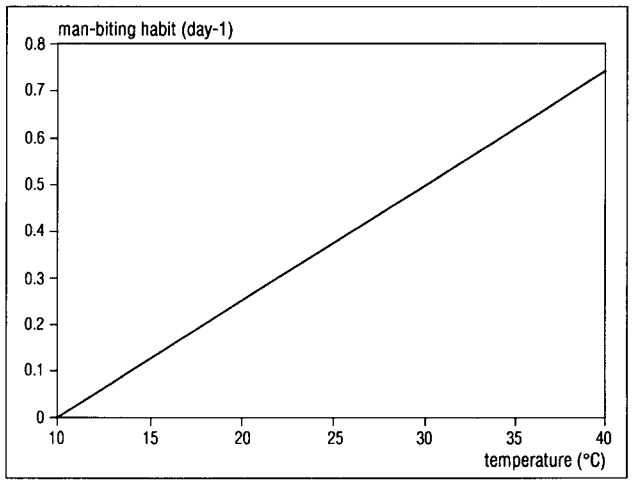


Figure 4.3: The man-biting habit of the mosquito host in relation to environmental temperature (relative humidity 60-90%; HBI = 0.9).



$$a = \frac{HBI}{DUR_{bd}} \quad (4.3)$$

where a is the man-biting habit (day^{-1}) and *HBI* the human blood index i.e. is the estimated proportion of the blood meals taken by a mosquito population which are obtained from man (Garret-Jones, 1964A; Garret-Jones *et al.*, 1980). The human blood index provides an indication as to whether a mosquito species is anthropophilic in its feeding behaviour (a high *HBI* indicates a preference for biting man) or zoophilic (a low *HBI*: species feeds mainly on animals). The *HBI* may vary within one species due to geographical variations and differences in saturation by insecticide in an area. The frequency of feeding depends mainly on the rapidity of digestion of a blood-meal (DUR_{bd} (days)) (Detinova, 1963; Service, 1980), which increases as temperature rises and, at optimal temperature, results one meal being taken every 48 hours (Muirhead-Thompson, 1954). The relation between temperature and the rapidity of blood digestion is given in Detinova (1963) as:

$$DUR_{bd} = \frac{DD_{bd}}{T - T_{\min,bd}} \quad (4.4)$$

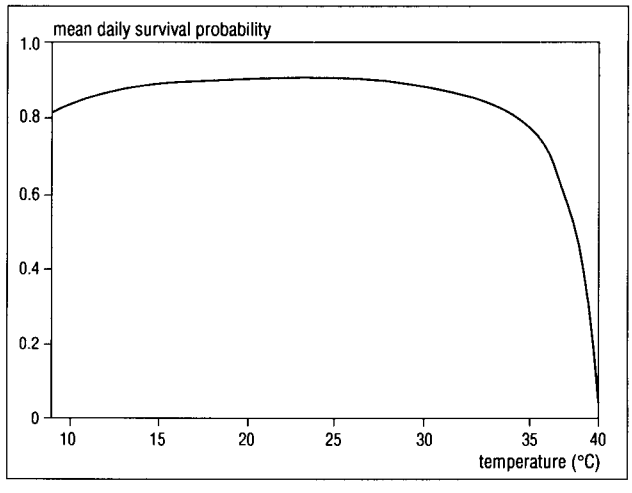
where DD_{bd} is the number of 'degree-days' required for the digestion of a portion of ingested blood, depending on the relative humidity (36.5 °C day), $T_{min,bd}$ is the minimum temperature required for the digestion of the blood meal (9.9 °C) and T is the actual average temperature (in °C; range between $T_{min,bd}$ and the maximum temperature (about 40°C)) (see *Figure 4.3*).

In this study, the above relationship is assumed to be similar for all *Anopheles* species. According to Detinova (1963), the time between two blood meals is equal to the duration of the blood digestion plus two days: one day being needed by the female mosquito to find a host to take a meal on and one day to find a suitable place to oviposit. Assuming that the frequency of feeding is equal to the blood digestion period (thus ignoring the time needed to find a host and oviposition site), Detinova's (1963) account fairly well agrees with observations made by Boyd (1949), Macdonald (1957), Gillies & De Meillon (1968), Garrett-Jones & Shidrawi (1969) and White (1982).

4.4.1.4. The longevity of the vector

The vector's longevity determines its ability to transmit a parasite, since the female mosquito has to live long enough for the parasite to complete its development. There is presumably an optimum temperature and an optimum humidity for each species of mosquito. Between certain limits, longevity decreases with rising temperature and increases with increasing relative humidity (Boyd, 1949; Molineaux, 1988). Longevity is considerably increased under both hibernation conditions (at low temperatures) and aestivation conditions (high temperatures) although the impact of transmission is minimized by low density of active mosquitoes and reduced feeding frequency (Molineaux, 1988). Data reported by Boyd (1949) and Horsfall (1955) on mosquito longevity indicate an optimum temperature of about 20-25°C and an optimum relative humidity of 60-90%, and our assumption about the relation between the longevity of the *Anopheles* mosquito and temperature is based on these data (see also *Figure 4.4*). A mean life expectancy[3] of 5 days (p = 0.82) is assumed at 9 °C (taken as the minimum temperature at which a mosquito becomes active). The maximum mean longevity is assumed to be 10 days (p = 0.9) at temperatures of about 20 °C. At a temperature of 40 °C, the mean longevity is deemed to be one day (p = 0.04), and temperatures above 40 °C are considered to be unfavourable for the mosquito population. In the systems approach, the relative humidity is assumed to remain at a level favourable for mosquito development and is assumed not to change with changing precipitation. A number of survival probability values for various *Anopheles* species are listed in Appendix 2.

Figure 4.4: Assumed relationship between temperature and daily survival probability of the adult mosquito.

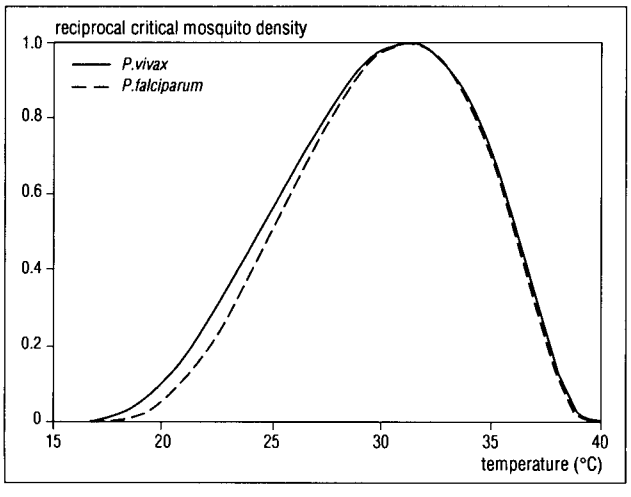


[3] The relation between the daily survival probability (p) and the mean longevity (ML; in days) of a mosquito population is: ML = 1/-ln (p) (MacDonald (1957)).

4.4.1.5. The density of vectors

Mosquito density is subject to wide variations over time and among localities. Consequently, it is very difficult to arrive at an estimate of mosquito numbers in a specific area. Furthermore, the change in time in mosquito numbers varies greatly between species and is determined by numerous environmental, biological and physical factors. Important among these are, for instance the availability and the surface area and depth of water in species-specific breeding places, the presence of predacious fish or other natural enemies, the hydraulics of bodies of water and the type of vegetation present. Similarly the concentration of mineral salts, nitrates, hydrogen-ions and pollutants in the water and the availability of larval food determines whether species can survive (Bates, 1949). Although mosquito densities are likely to be influenced by climate changes (see section 3.2.3), the change in mosquito abundance in large areas as a result of temperature and precipitation change is almost impossible to estimate satisfactorily. The mosquito density is therefore assumed to be constant, in the simulation of the change in vectorial capacity. However, a critical mosquito density per human (m_{crit}) needed to maintain malaria at a positive endemic level, can be used as an index of potential malaria risk (epidemic potential). Potential malaria risk is defined as 1 divided by the critical density. The critical density is based on the critical vectorial capacity (VC_{crit}) (Dietz, 1988), which is derived from the mean period during which a malarial patient is infectious (Molineaux, 1988; the VC_{crit} for *P.vivax* and *P.falciparum* is 0.0009 and 0.0027 day^{-1}, corresponding with mean infectious periods of 3 years and 1 year, respectively).

Figure 4.5: The reciprocal critical mosquito density as a function of the temperature.



$$m_{crit} = \frac{VC_{crit} * (-\ln(p))}{b * c * a^2 * p^n} \qquad (4.5)$$

Expressing the malaria potential by means of the critical mosquito density provides a means by which to assess malaria transmission, in the areas potentially at risk (Jetten & Martens, 1994).

4.4.1.6. Temperature and critical mosquito density

In *Figure 4.5* the influence of increasing temperature on the reciprocal of the critical mosquito density is shown (this inverse critical mosquito density is given the value one at its maximum). The malaria transmission potency of a mosquito population increases as the reciprocal critical density increases. As temperature increases, the inverse of the mosquito density increases until a maximum is reached. At high temperatures, the accelerated development of the parasite and the increased bite frequency can no longer compensate for the decreasing mean life expectancy among the mosquitoes.

4.4.1.7. Precipitation and critical mosquito density

By providing the medium for the aquatic stages of the mosquito life cycle, rainfall obviously plays a crucial role in mosquito reproduction and malaria epidemiology. Rainfall may also increase the relative humidity and hence the longevity of the adult mosquito. The relations between changing temperatures, precipitation and relative humidity, however, are quite complicated and the processes affecting atmospheric humidity suggest only a small change in relative humidity as the atmosphere gets warmer (Mitchell & Ingram, 1992). An attempt to quantify the impact of accumulated rainfall on mosquito density is made in Kruijf, de, *et al.* (1973). The sizes of some mosquito populations are strongly correlated with the pattern of rainfall, whereas other species show no correlation at all. The introduction of large-scale irrigation schemes has however, reduced the significance of rainfall in malaria epidemiology to some extent (Muir, 1988).

In view of the great divergence among localities and mosquito species, a universal relation between rainfall and the dynamics of the mosquito population would seem to be impossible to arrive at. However, because rainfall may be a limiting factor in mosquito breeding, in this study seasons with less precipitation then 15 mm are considered unsuitable

for mosquito breeding.

4.4.2. Human population

The model used to describe the transition between the reservoirs of the human population at risk is based on a microparasite-epidemiological model as described in Aron & May (1982), Bailey (1982), Levin *et al.* (1989) and Anderson & May (1991). The human population subject to a risk of malaria is divided into three categories for each of two different age classes: susceptible persons ($HSUS_{age}$), infected persons ($HINF_{age}$) and immune persons ($HIMM_{age}$) (see *Figure 4.1*). The latent reservoir is omitted, because the duration of a stay in this reservoir is usually very short in comparison to the residence time in the other reservoirs.

The dynamic behaviour of the human system can be described by:

$$\frac{dHSUS_{age}}{dt} = LIM(t) * HIMM_{age}(t) - (RI(t) + \mu_h(t)) * HSUS_{age}(t) \quad (4.6)$$

$$\frac{dHINF_{age}}{dt} = RI(t) * HSUS_{age}(t) - (LIF_{age}(t)\,\mu_h(t) + \beta_{age}) * HINF_{age}(t) \quad (4.7)$$

$$\frac{dHIMM_{age}}{dt} = LIF_{age}(t) * HINF_{age}(t) - (LIM(t) + \mu_h(t)) * HIMM_{age}(t) \quad (4.8)$$

where $RI(t)$, $LIM(t)$ and $LIF_{age}(t)$ are defined as follows:

$$RI(t) = 1 - e^{-VC(t) * HINF_p(t)} \quad (4.9)$$

$$LIM(t) = \frac{RI(t)}{e^{RI(t)/BLIM} - 1} \quad (4.10)$$

$$LIF_{age}(t) = \frac{RI(t)}{e^{RI(t)/BLIF_{age}} - 1} \quad (4.11)$$

The number of susceptible persons may change over time, as their number falls due to 'natural deaths' (i.e deaths not associated with malarial infection) at a rate μ_h, or as they become members of the infected class at a rate RI. Infected individuals either die (naturally at a rate μ_h or from infection at a rate β_{age}) or recover to join the immune class (at a rate LIF_{age}). Immune persons lose their immunity at a rate LIM, and those who have lost their immunity return to the reservoir of susceptible persons. All newborn babies are assumed to be members of the class of susceptibles, and as they grow older they graduate from the younger age class to the older. The chance that a person will receive at least one infective bite during a particular day is calculated by means of a Poisson distribution. This rate at which individuals become infected (RI) depends on the vectorial capacity (VC), which represents the transmission potential of the mosquito population (see section 4.4.1)) and on the proportion of infected people in the human population ($HINF_p$). Rates of recovery from infection depend on the species of parasite and appear to increase with the longevity of people in endemic areas. Assuming that re-exposure does not occur, infection and immunity endure for a fixed period of time. However, if a person is further exposed before this period has elapsed, infection and immunity is sustained. The basic loss rate of infection $BLIF_{age}$ is defined as 1/average duration of infectiousness (average three years infected with *P.vivax* and one year with *P.falciparum* in the case of children under 5 when left untreated (Molineaux, 1988); for the age group above 5 years, $BLIF_{age}$ is assumed to be 5 times higher). The basic loss rate of immunity $BLIM$ is 0.67/year, corresponding with a mean duration of immunity of 1.5 years (Aron & May, 1982)). If infection occurs at a per capita rate RI, the average per capita rate of loss of infection (LIF_{age}) and loss of immunity (LIM) as a function of RI is expressed as described in formulae 4.10 and 4.11. The fatality rate (β_{age}) associated with *P.falciparum* is assumed to be 4% for those below the age of 5 and 1% among their seniors, whereas infection with *P.vivax* is considered not to be fatal at all (WHO, 1980; Greenwood *et al.*, 1987). Thus, given a certain pattern of vectorial capacity, we can use the equations set out above to simulate the transmission dynamics in a given population.

The framework used to describe the malaria transmission dynamics in the human population ignores two important aspects (Anderson & May, 1991). First, in this model no distinction is drawn between infection and infectiousness. An infected person may harbour the parasite in certain stages of its development (in liver and blood) but not harbour parasites at a stage which is infective to the vector.

Neither is the intensity of the infection described in the model employed (since only the presence or absence of infection is modelled). The second shortcoming concerns the nature of acquired immunity to malarial infection. Shaking of the infection does not imply full protective immunity against reinfection. To speak of a single reservoir of immune persons is an oversimplification of the true complexities of acquired immunity to malaria.

4.5 Socio-economic impact of malaria

The estimation of the socio-economic impact of malaria and the expression of this impact in terms of money is highly precarious. Economic losses associated with malarial morbidity and mortality are not easily quantifiable, while demographic consequences and the cost of providing medical care to people are also very difficult to determine. Morbidity and mortality from malaria will generally express itself in accordance with the epidemiological features of the disease. In areas with stable, highly endemic malaria, morbidity and mortality in the economically-active age groups are expected to be relatively lower than in areas of low to moderate endemicity, since in the latter practically all age groups are involved alike (Wernsdorfer & Wernsdorfer, 1988). Social and economic consequences of malaria are in general directly related to its severity as measured by disability and death and these effects can be quantified in terms of the number of years of healthy life that were lost. In this study, the concept of a disability-adjusted life year (DALY), developed and promoted by the World Bank (World Bank, 1993), is used as a measure of the health impact of malaria. A more elaborate study of the socio-economic impact of malaria will be carried out in the near future (Resource Analysis, 1994).

4.5.1. Disease burden

The disease burden (*DB*) is expressed as the numbers of years of healthy life lost (World Bank, 1993). For each death, the number of years of life lost is defined as the difference between the actual age at death and the life expectancy at that age. In the case of disability due to malaria, the incidence per year of the disease (*INC*) is multiplied by the expected duration of the condition of disability (*DC* = 14 days, assuming that a clinical attack lasts for 7 days, occurring twice a year). A refinement is introduced by allocating a severity weight (for malaria a *SW* of 0.4 is taken). The death and disability losses are combined and allowance is made for a discount rate of 3% (so that the future years of healthy life are valued at progressively lower levels (World Bank (1993)) and for age weights (so that years of life lost at different ages are assigned with different relative values). The combination of discounting and age weighting yields the pattern of DALYs (Disability Adjusted Life Years) lost by death and age ($DALYs_{death}$ = 32.5 and 25 years for the age group 0-5 years and above 5 years, respectively).

The burden of disease, as described above, can be expressed for malaria as follows:

$$DB_{mal}(t) = DB_{mal.death}(t) + DB_{mal.dis}(t) \quad (4.12)$$

The disease burden due to death and disability losses ($DB_{mal.death}$ and $DB_{mal.dis}$) are calculated as follows:

$$DB_{mal.death}(t) = \sum_{i=1}^{age} DEATHS(age,t) * DALYS_{death}(age) \quad (4.13)$$

$$DB_{mal.dis}(t) = \sum_{i=1}^{age} INC(age,t) * SW * DC \quad (4.14)$$

If incidence and death rates are absolute numbers, the disease burden is expressed as the total number of disability-adjusted life years lost. The effect of changing population numbers can be allowed for by expressing the disease burden per 1,000 inhabitants. The DALYs per 1,000 population are obtained by expressing the incidence and death rates in numbers/1,000 population.

5. CLIMATE CHANGE AND MALARIA: A SUSTAINABLE DEVELOPMENT INDEX

5.1 Introduction

In general, indicators describe complex phenomena in a quantitative way by simplifying them in such a way that communication is possible with specific target groups. This implicates that indicators should have added value vis-à-vis observations or data sets. In this study an indicator is defined as a characteristic of the status and the dynamic behaviour of the system under concern. From this systems-based definition it follows that an indicator is a one-dimensional systems description, which may consist of a single variable (absolute indicator) or of a set of variables (relative indicator). Practicable key conditions for indicators to be satisfied are formulated in Rotmans *et al.* (1994): quality of data and data collection; sensitivity to human-induced variations in space and time; policy relevance with a scientific base; and recognisability and clarity.

The distinction between indicators and indices is based on a difference in aggregation level. An index is here defined as a multi-dimensional composite made up from a set of indicators. As a result of their composite, and therefore abstract, character the requirements mentioned above do not necessarily hold for an index. A prerequisite for sustainable development indices (SDIs) i.e. sustainability indices is that they represent environmental pressure, and the state of, and impact on environmental conditions. In other words, they should capture as much as possible of the cause-effect chains they represent and relate pressure and effects to criteria for sustainable development (Greef, de & Vries, de, 1991; Rotmans *et al.*, 1994).

SDIs should not only yield modelling information relating to a given moment, but also insights into future intrinsic dynamic behaviour. Therefore the following potential index components have been designed (Rotmans *et al.*, 1994): pressure on the system; rate of change of the system; state of the system; actual effect or potential risk for the system; recovery or buffer capacity of the system; and societal (system's) response. This implies that each SDI basically has the following structure:

SDI = (pressure, rate of change, state, effect/risk, recovery/buffer capacity, response)

For each of the components, the initial state, reference state and target state should be defined. The initial state refers to the starting point of the simulation; the reference state represents the undisturbed state of the system; and the target state is the desirable (sustainable) state to be achieved by the index.

5.2. A SDI for the impact of global climate change on malaria incidence

The process leading to the impact of a human-induced climate change on malaria incidence can be represented by a sequence of indicators representing the cause-effect chain. For malaria the major environmental pressure indicator is the change in global mean surface temperature. The changes in vectorial capacity and the possible subsequent changes in the disease burden due to malaria (DB_{mal}), calculated on the basis of prevalence and incidence figures, represent the next steps in the cause-effect chain. An indicator of socio-economic development can be incorporated by adopting the magnitude of investments in health services. The amount of resources allocated to health services strongly depends on the socio-economic development of a country, and relative affluence may serve as buffer against the adverse health effects of global environmental change. The dynamics and the level of economic development can be considered as an indicator of the means placed at the disposal of the public health sector, which can be expressed by the ratio GNP growth/population growth. The higher this ratio, the more support is likely to be given to public services such as education and health care (Wernsdorfer & Wernsdorfer, 1988).

Because interdependencies exist between the various indicators of the sustainable development index of global climate change on malaria incidence ($SDI_{cc\ \&\ mal}$), this SDI can be aggregated to a three-dimensional vector, resulting in the following:

$SDI_{cc\ \&\ mal}$ = (rate of temp. change., change DB_{mal}, GNP growth/population growth)

In order to render this index useful with respect to

sustainable development, targets and reference values need to be determined. For temperature, a maximum global mean (surface) temperature increase of 2°C above pre-industrial global mean temperature and a global rate of temperature change of 0.1 °C per decade can be viewed as an upper limit beyond which the risks of considerable damages to ecosystems and sensitive coastal areas, and of unexpected sudden changes in the climate system are expected to increase rapidly (AGGG, 1990). Being more sensitive to climate changes, the relative temperature target is adopted in the index. The disease burden per 1,000 inhabitants can be used to compare regions, as well as risk factors, risk groups and gender factors (World Bank (1993); see section 4.5.1). In an ideal world the disease burden associated with malaria would be zero, although this level will probably never be reached. The more realistic target proposed here is therefore a zero change in number of DALYs lost. This implies that any increase in disease burden is considered as an unsustainable development. A low value of the ratio GNP growth/population growth indicates an economic state of affairs which is not conducive to the elimination of malaria. Available data (WHO, 1974) suggest that a ratio > 2.5 would be required to control malaria adequately.

The values of the various indicators of the $SDI_{cc\ \&\ mal}$, described above, can be used to determine whether future projections and developments are, according to the above-mentioned target and reference levels, sustainable or not.

6. RESULTS

6.1 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 continuously, and the CO_2 emissions in particular are doubled within forty years. The Montreal protocol for ozone-depleting substances is not strengthened 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 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 through large-scale reforestation programmes is envisaged. Population growth is the same in the two scenarios and is based on World Bank data (Zachariah & Vu, 1988). For a more detailed description of these two scenarios see IPCC (1991). In Figure 6.1 the estimated global mean temperature increase according to the two IPCC-1990 scenarios is depicted, and in Figure 6.2 the rate of global mean temperature increase, both calculated with the TARGETS model.

6.2 Malaria risk due to climate change

6.2.1 Introduction

On basis of the (changes in) regional temperature and precipitation patterns according to the BaU-scenario, which are derived from the global mean temperature projections in Figure 6.1, the change in geographical malaria potential is simulated in section 6.2.2. The risk of renewed transmission in non-endemic areas is assessed by means of studying the receptivity and vulnerability of the area. The former term reflects the transmission potential by the local mosquito population, and the reciprocal of

Figure 6.1: Global mean temperature increase according to the two IPCC-1990 scenarios over the period 1990-2100. (shaded area indicates where the absolute temperature target of 2°C is exceeded (temperature increase 1900-1990 is assumed to be 0.5°C); see section 5.2).

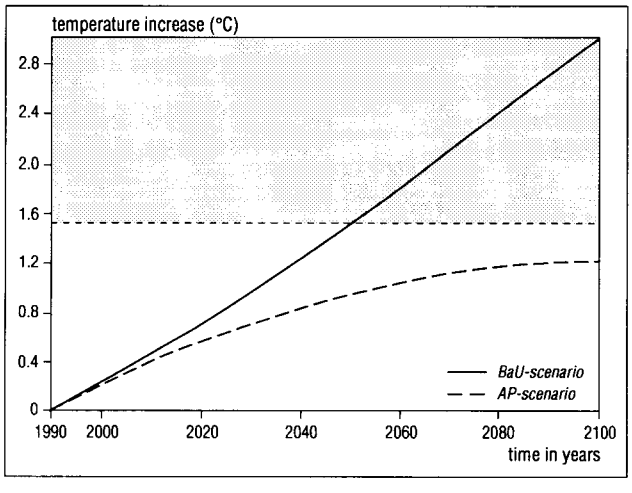
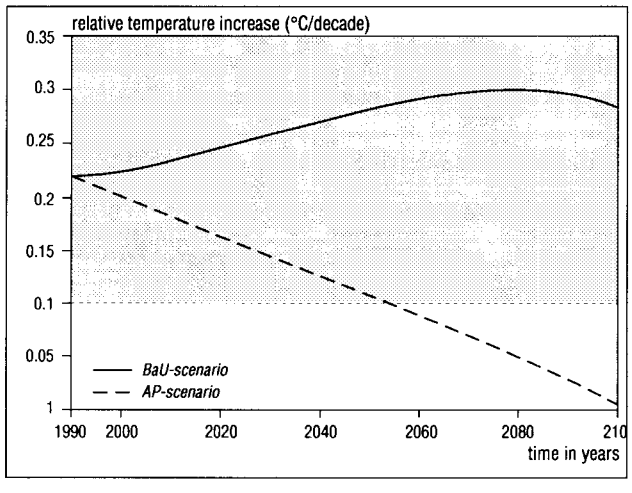


Figure 6.2: Rate of global mean temperature increase according to the two IPCC-1990 scenarios over the period 1990-2100. (shaded area indicates where the relative temperature target of 0.1°C/decade is exceeded; see section 5.2).

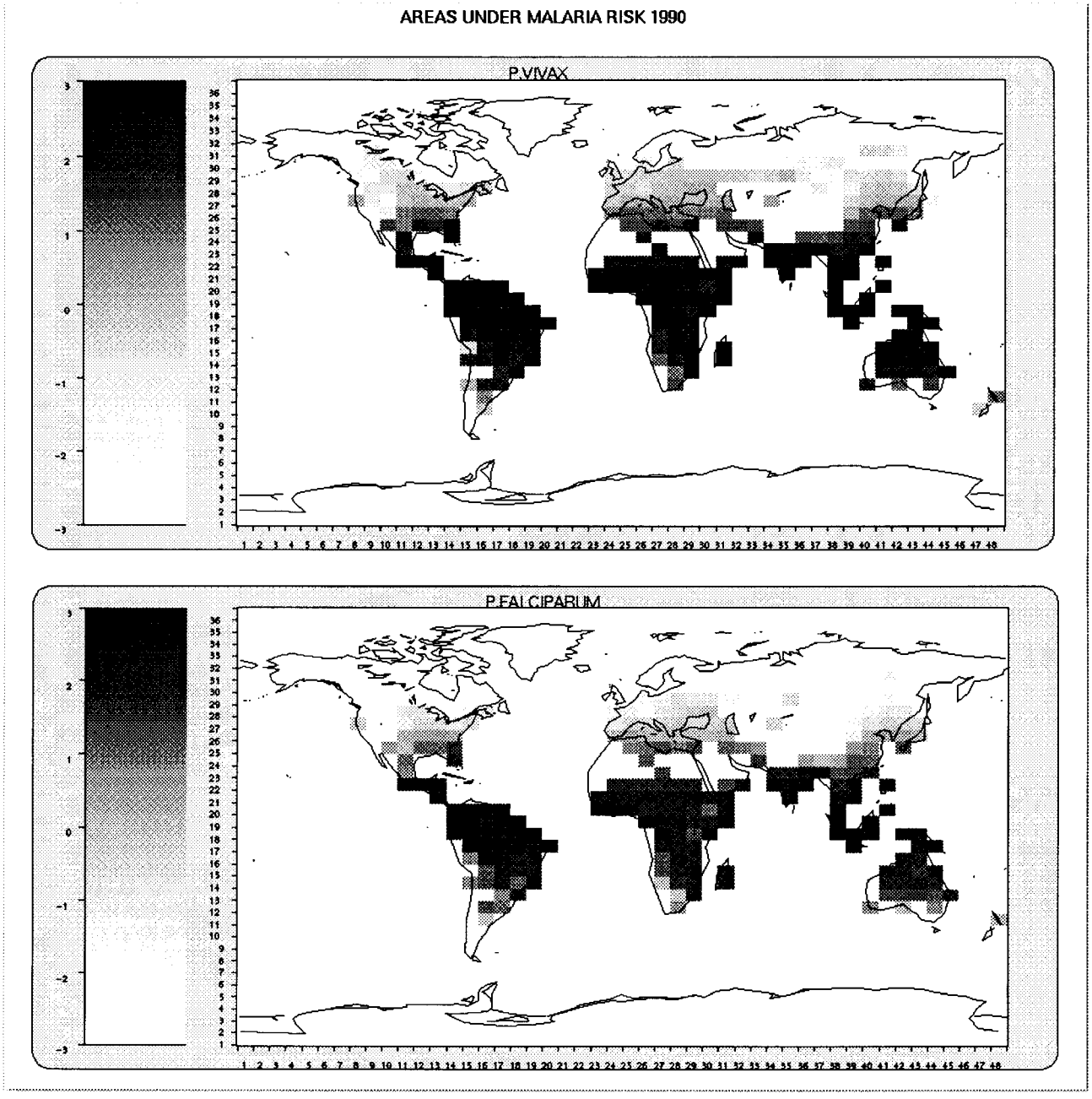


the critical mosquito density is chosen as a measure for this factor. The latter term is related to the numbers of imported cases over a unit of time. As an example, the change in malaria risk is simulated for Greece, formerly the most malarious country in Europe. These results are presented in section 6.2.3. The effect of a human-induced climate change on malaria prevalence and disease burden is evaluated for highly endemic areas which are mainly found in tropical Africa, and areas of lower endemicity found in other parts of Africa, South-America and South-East Asia (section 6.2.4). In tropical Africa, attention is restricted to *P.falciparum* as it remains the predominant species, responsible for almost all malaria mortality. In areas of lower endemicity, changes in the prevalence of both *P.vivax* and *P.falciparum* are simulated.

6.2.2 *Change in geographical pattern of malaria risk*

In the simulation model seasonal temperature and precipitation are incorporated in calculations of the hypothetical mosquito density needed to cause a

Figure 6.3: Potential malaria risk areas in 1990, calculated from seasonal temperature and precipitation as annual averages. (expressed as the logarithm of the reciprocal critical mosquito density)



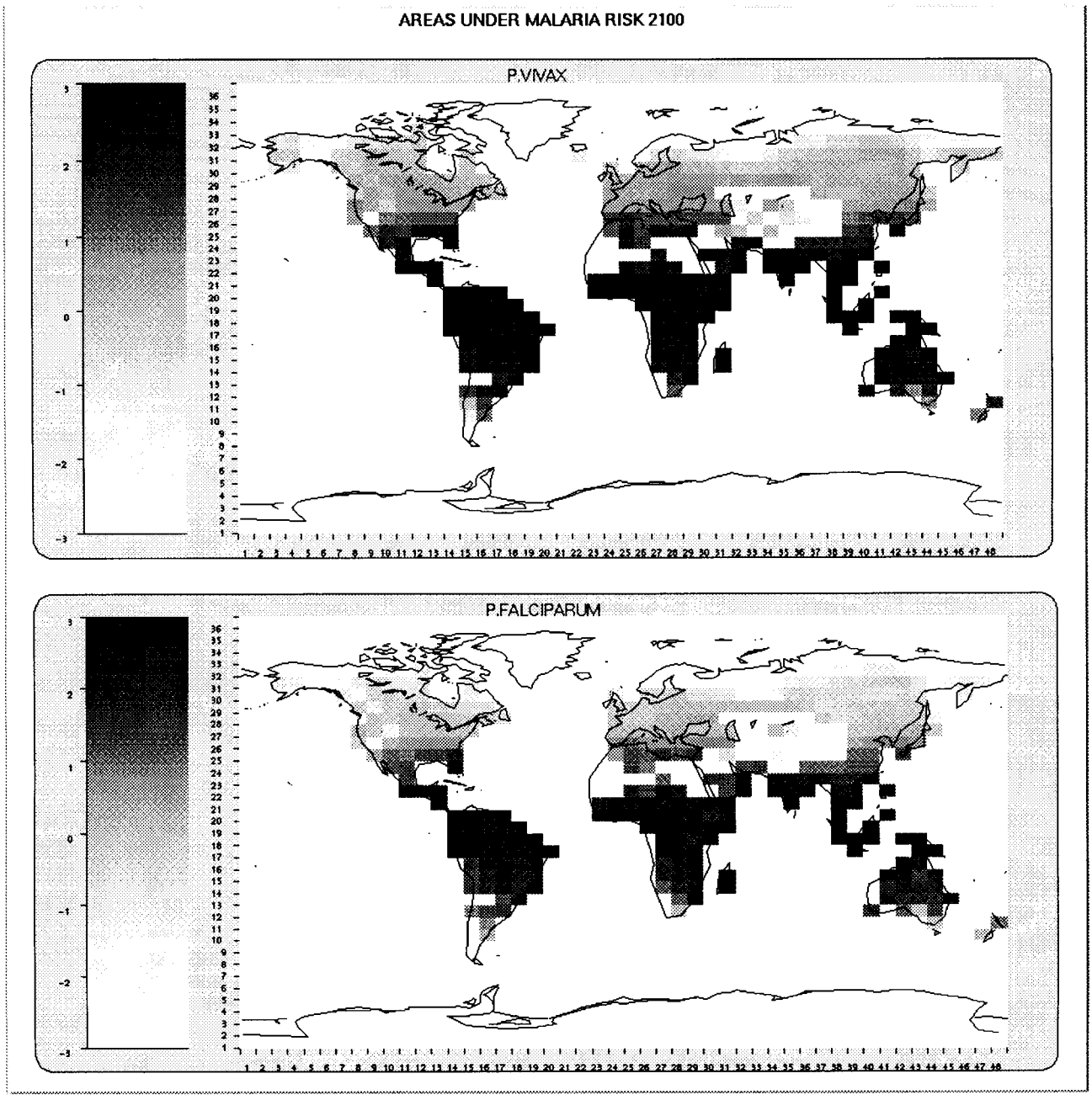


Figure 6.4: Potential malaria risk areas in 2100, calculated from seasonal temperature and precipitation as annual averages. (expressed as the logarithm of the reciprocal critical mosquito density)

malaria epidemic (the critical mosquito density) (see 4.4.1.5). As *Anopheles* species are in general more anthrophophilic in warmer climates than in colder climates, the human blood index (*HBI*) is assumed to vary from 0.9 in the tropical regions to 0.1 in temperate zones.

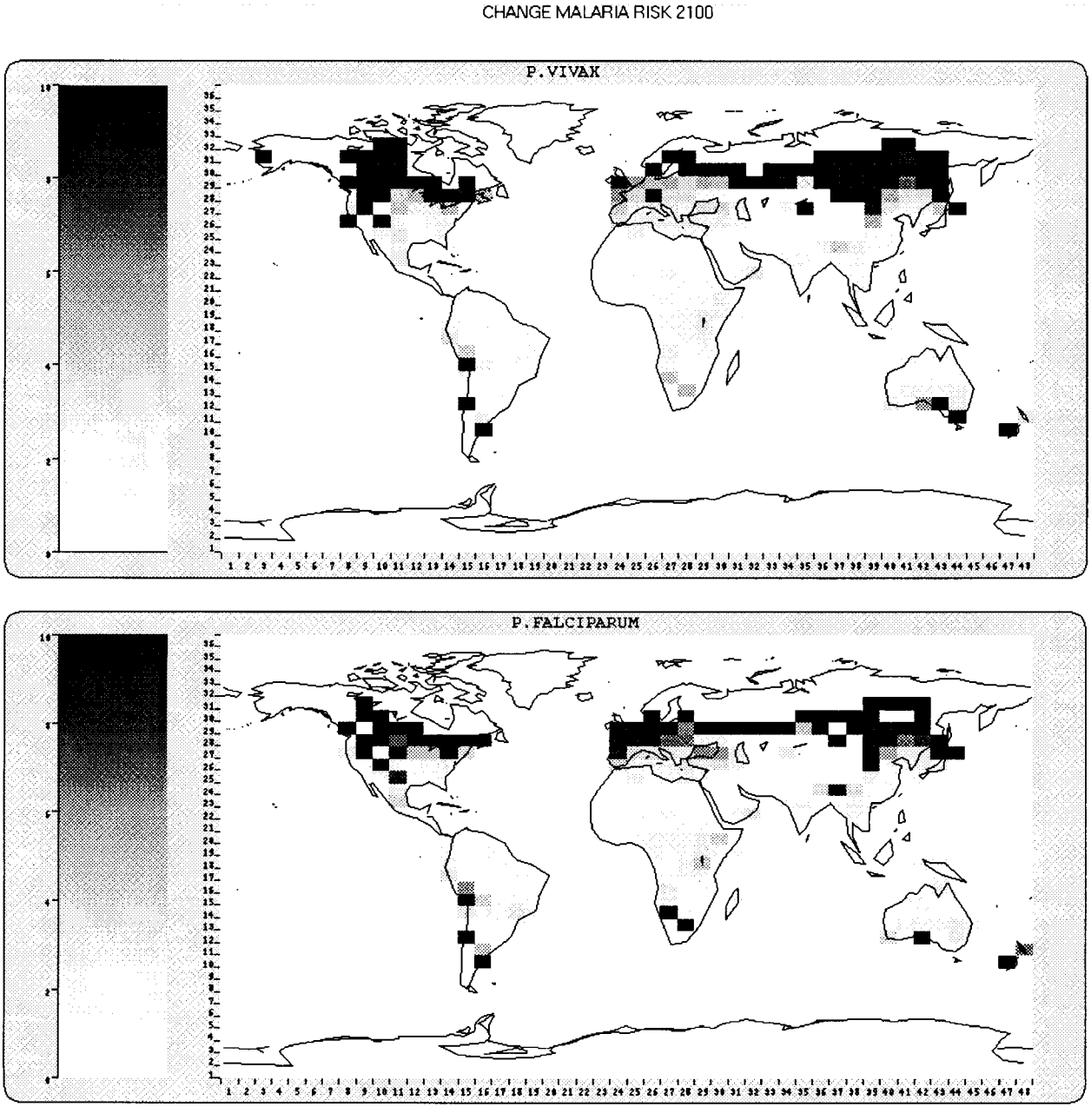
Figure 6.3 depicts the global distribution of the potential malaria risk areas and the estimates of absolute limits of the possible geographical extension of malaria transmission in the year 1990. The areas where less than 10 mosquitoes per human are needed to transmit malaria ($\log(1/m_{crit}) < -1$) roughly correspond to the areas where endemic malaria transmission occurs if mosquitoes are abundant. For *P.vivax* this includes large parts of the United States up to the Canadian border, Southern and Central Europe, Turkey, Southern Russia, China and Japan. *P.falciparum* is more restricted to tropical areas because parasite development needs a minimum of at least 16°C. *Figure 6.4* shows that an expansion of the geographical areas susceptible to malaria transmission and a worldwide increase of potential malaria risk is to be expected as climate changes. The main changes occur in the areas with

temperate climates where mosquitoes already occur but where development of the parasite is limited by temperature (*Figure 6.5*); by the year 2100 in large parts of North-America, Europe and Asia, a mosquito density ten or more times smaller than in 1990 may induce malaria transmission. However, because of their high potential receptivity, the highest risks of the reintroduction of malaria transmission remain in the non-endemic regions bordering on malarious areas. An increase in the potential transmission period might transform a previously non-malarious area into one subject to seasonal epidemics. Furthermore, in areas in which malaria was previously only transmitted during parts of the year, malaria may become perennial.

The computations also partly explain why the situation in tropical Africa and other tropical countries differs so much from that in the more temperate regions: in tropical climates the critical mosquito densities are several times lower than those required in cooler climates to maintain malaria at a positive endemic level. For instance, in order to eradicate malaria in some parts of Africa one would need to effect a reduction in the mean density of mosquitoes per human more than 100 times greater than that necessary in Europe.

Comparing the potential geographical extent of malaria to the actual malaria distribution (*Figure 6.3*

Figure 6.5: Change in potential malaria risk in 2100, as compared wih the year 1990.



and *Figure 3.1*) indicates that interpretation of the risk areas simulated must be carried out in a manner which takes into account local conditions and developments e.g. socio-economic conditions and mosquito densities. In tropical and sub-tropical regions, climatic conditions are already favourable for the breeding and reproduction of the mosquito, resulting in mosquito densities exceeding the critical value during large parts of the year. However, in many world regions a situation characterized as '*Anophelism* without malaria' exists, a term referring to the absence of malaria in the presence of *Anophelines* as well as climate factors, in particular temperature, which are apparently conducive to transmission. Socio-economic conditions, effective vector control measures, the treatment of infected individuals, and the specific characteristics of the human and/or mosquito population (see sections 3.2.1 and 3.2.3) may explain this phenomenon. In Central and South Pacific, no potential vectors are present, establishing a malaria-free zone in this area.

6.2.3 *Risk of (re-)introduction of malaria in non-endemic areas*

In *Figure 6.6*, the change in malaria transmission potential is simulated for the case of Greece. The threat of imported malaria in countries where the disease has been eradicated is closely related to the increase in human mobility. Therefore, the change in malaria risk is simulated for two situations: in the first, the number of imported malaria cases does not change in time (i.e. vulnerability remains constant); and in the second, an annual increase in imported malaria of 5% is assumed, the latter reflecting the probable situation more faithfully than the first (Bruce-Chwatt, 1982). The risk of introduction of the parasite *P.falciparum* in the Mediterranean countries of Europe is negligible, as *Anophelines* in this region are refractory to it (WHO, 1982). Therefore, attention is restricted to the risk of the reintroduction of *P.vivax*.

Figure 6.6: Change in malaria transmission potential in Greece for the AP and BaU scenarios (P.vivax).

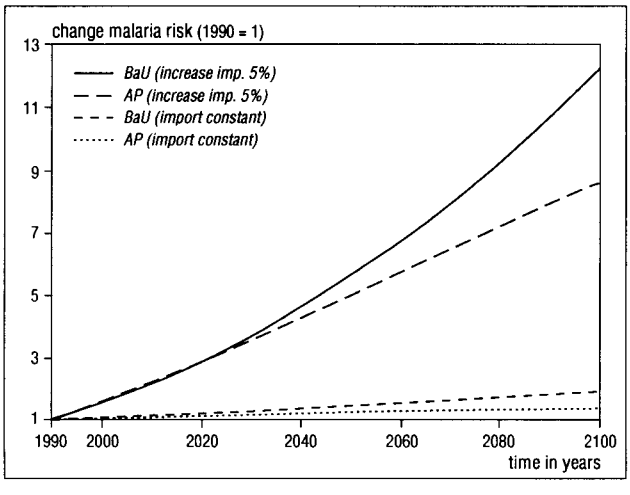


Figure 6.6 shows that the potential risks of renewed transmission increase, whereby the relative importance of the contribution of increasing numbers of imported cases to this risk is clearly shown. Note that the critical mosquito density expresses the potential vulnerability of an area, while risk of reintroduction of malaria also depends on actual mosquito densities present. As increasing temperatures may increase the growth rate of a mosquito population and thus mosquito numbers, the risk of renewed malaria transmission will further increase. Thus the results presented arguably underestimate the change in risk of renewed malaria transmission.

The results seem to justify asking the question of whether a human-induced climate change in combination with imported cases of malaria may give rise to the development of local epidemics in regions which were endemic in the past. The risk of renewed malaria transmission will be of particular concern to the national health authorities in Australia and the USA as well as in parts of Europe. Moreover the reintroduction of *P.falciparum* will also give cause for concern to the Australians and Americans since vectors in these regions are non-refractory to it (Molineaux, 1988).

6.2.4 *Projected increase in risk of malaria in endemic areas*

All results shown deal with excess figures, defined as the extra numbers of cases per year expected as a consequence of a human-induced climate change. As excess prevalence and disease burden are also influenced by the age distribution of the population, values for them are obtained by subtracting the baseline estimates (demographical changes with an unchanging infection rate) from the estimates obtained according to the two scenarios under consideration.

Although actual prevalence and incidence figures are not very reliable in most endemic regions, a good estimate of the infection rate can be obtained from the rate of increase of prevalence with age in young children. In our calculations, the initial vectorial capacities are chosen such that the force of infection is 2.0 per annum for the year 1990 in highly endemic regions and 0.1 in areas of lower

Table 6.1: Vectorial capacities (VC) and equilibrium values in the initial year (1990).

	high endemicity P.falciparum	low endemicity P.vivax	low endemicity P.falciparum
VC (/day)	0.074	0.0043	0.012
equilibrium values			
RI(/year)	2.0	0.1	0.1
prevalence (/1000)			
age -5	452	159	72
age 5+	27	54	17
deaths(/1000)			
age -5	18	0	2.9
age 5+	0.3	0	0.2
disease burden (DALYs/1000 population)	73.3	1.41	5.8

endemicity. Although these values are chosen rather arbitrarily, they lie within the range of the values found in several studies on the pristine force of infection in young children. Choosing other initial values may alter the magnitude of change, but the direction of change remains the same. In the estimates of the excess prevalence and disease burden in endemic areas, the malaria conditions are assumed to be in equilibrium in the year 1990. For the stable, highly-endemic regions of tropical Africa, this assumption seems to be justified. However, for the unstable areas of lower endemicity, this assumption will often be inappropriate. Since the feedback associated with socio-economic development is not yet included in the model, the simulation results can only identify possible trends and must be considered to be tentative. *Table 6.1* tabulates the equilibrium values in the initial year (1990) of the simulation run.

Large tracts of the equatorial regions of Africa, South-America and South-East Asia show no great seasonal fluctuations in temperature. Therefore, for the simulation runs an average annual temperature of 25°C in 1990 is adopted.

Figure 6.7 shows the increase in the average annual vectorial capacity in endemic regions according to the AP and the BaU scenarios. The resulting changes in malaria prevalence are set out in *Figures 6.8-6.10*. The results show that an increase in ambient temperature of several degrees due to a human-induced climate change may lead to an increase in transmission potential of the mosquito population. In highly-endemic areas, where the malaria situation is relatively stable, the result may

Figure 6.7: Simulated change in vectorial capacity according to the two IPCC-scenarios for endemic malaria regions.

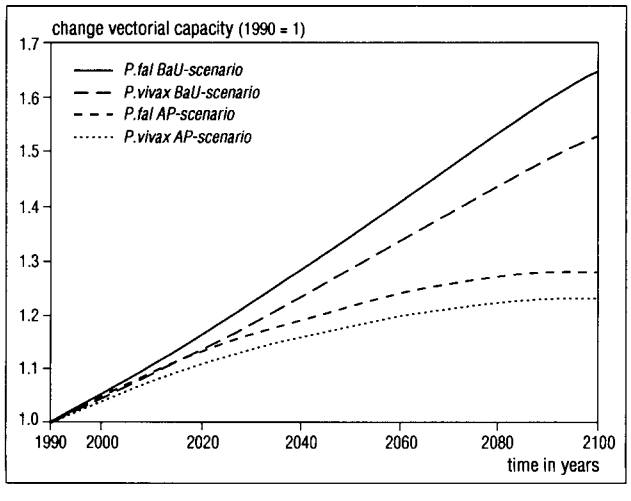
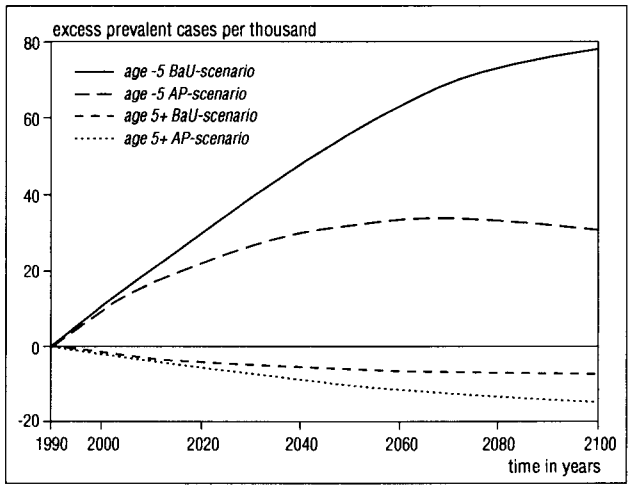


Figure 6.8: Excess prevalence of P.falciparum in highly-endemic areas.



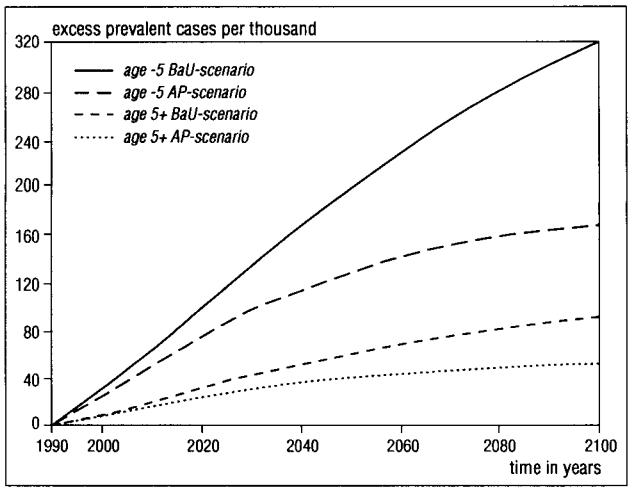


Figure 6.9: Excess prevalence in areas of lower endemicity (P.vivax).

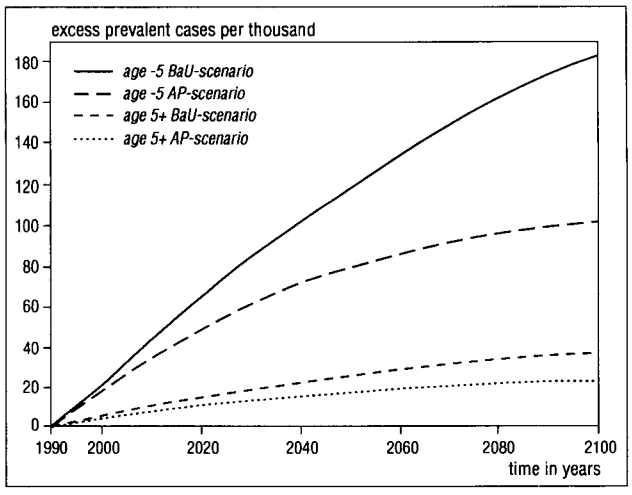


Figure 6.10: Excess prevalence in areas of lower endemicity (P.falciparum).

be a decrease in prevalence in the older age group as collective immunity increases. However, the increase in prevalence in children under five is so pronounced that the disease burden per thousand population will increase. In areas of lower endemicity, a relatively small increase in malaria transmission potential may lead to a considerable increase in the prevalence of people suffering from malaria for both age groups, assuming that no special control measures are taken (e.g. increased application of (new) anti-malarial drugs). Although the increase in the disease burden in the areas of lower endemicity is higher (*Figures 6.11 and 6.12*), the major part of the disease burden due to malaria remains in the highly-endemic countries of tropical Africa. As *P.vivax* is considered not to be fatal, the disease burden due to infection with *P.vivax* is small compared to infection with *P.falciparum*.

6.3 Uncertainty analysis

The results presented in this paper are to a great extent based on two related state variables: the vectorial capacity (*VC*) and the critical mosquito density (m_{crit}), variables which are supposed to be highly sensitive to changes in the mean daily survival probability of the mosquito population (p): *VC* and the reciprocal m_{crit} change to the n^{th} power of the daily survival rate of the vector, where n represents the duration of the development of the parasite. A preliminary, straightforward and simple uncertainty analysis is performed on these variables, whereby the maximum daily survival probability is varied between 0.8-0.95, and the minimum temperature required for parasite development ($T_{S,min}$) between 14.5-15°C for *P.vivax* and 16-19°C for *P.falciparum*. The uncertainty ranges presented

Figure 6.11: Excess DALYs lost in highly-endemic areas.

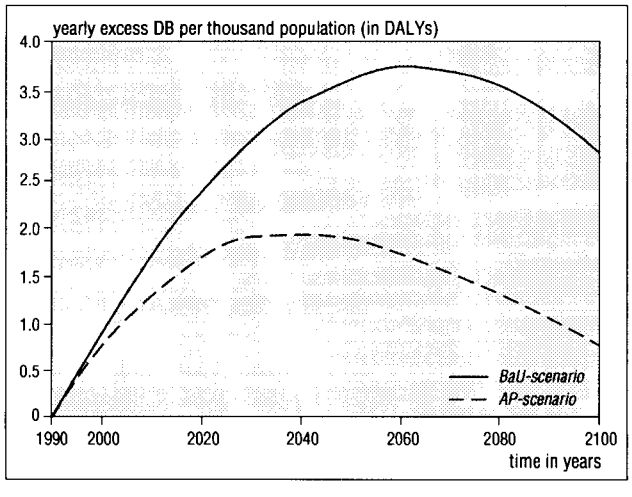
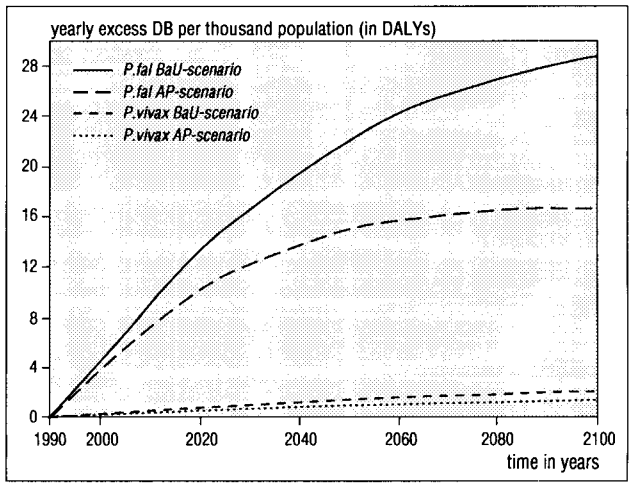


Figure 6.12: Excess DALYs lost in areas of lower endemicity.



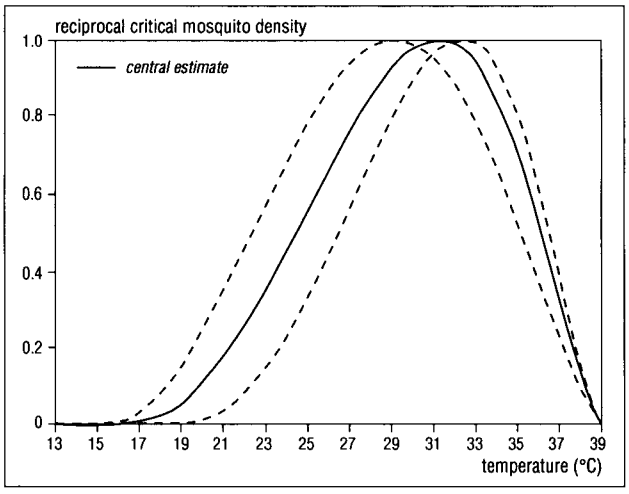


Figure 6.13: Uncertainty range of the relative reciprocal critical mosquito density as a function of the temperature (P.vivax). (left-hand curve: p = 0.8, $T_{s,min}$ = 14.5°C; central estimate : p = 0.9, $T_{s,min}$ = 14.5°C; right-hand curve: p = 0.95, $T_{s,min}$ = 15°C)

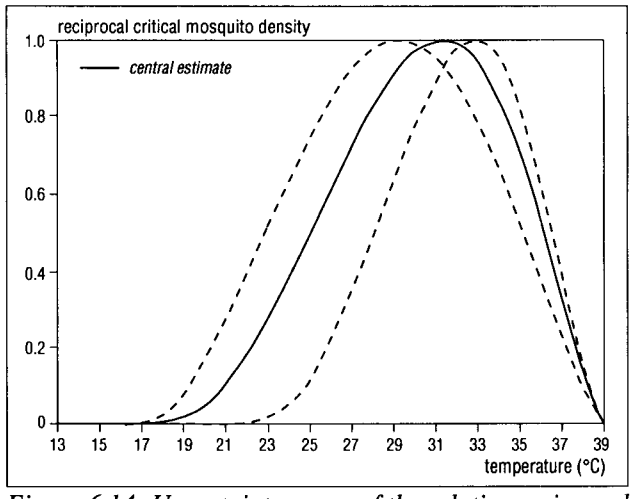


Figure 6.14: Uncertainty range of the relative reciprocal critical mosquito density as a function of the temperature (P.falciparum). (left-hand curve: p = 0.8, $T_{s,min}$ = 16°C; central estimate : p = 0.9, $T_{s,min}$ = 16°C; right-hand curve: p = 0.95, $T_{s,min}$ = 19°C)

hereafter are based on variations of some (crucial) parameter values, and are therefore only indicative and not representative for the full range of uncertainty. In *Figures 6.13* and *6.14* an uncertainty range of the relative reciprocal m_{crit} to changes in p and n is shown. The temperature range conducive to malaria transmission becomes narrower as $T_{s,min}$ increases, which implies that the simulated absolute limits of the possible geographical extension of malaria transmission in the year 1990 will be narrower. In *Figures 6.15* and *6.16* an uncertainty range is calculated for the change in vectorial capacity in the endemic regions. The Figures show that the effect of a human-induced climate change on the transmission potential of a mosquito population will be more pronounced in the case of relatively less potent mosquito populations (given constant mosquito densities). In an integrated assessment model such as that presented in this report, a cumulation of uncertainties throughout the cause-effect chain will occur. Obviously, uncertainties in the climate module will influence the uncertainty range of the vectorial capacity and the critical mosquito density. *Figures 6.17* and *6.18* show the cumulation of uncertainties associated with climate and vectorial capacity. To obtain an uncertainty range for the global-mean temperature the major determinant of uncertainty, the climate sensitivity parameter, is changed (Den Elzen, 1993). Comparing the uncertainty ranges of *Figures 6.15*

Figure 6.15: Uncertainty range of the simulated change in vectorial capacity according to the two IPCC-scenarios for endemic malaria regions (P.vivax). (upper limit: p = 0.8, $T_{s,min}$ = 14.5°C; central estimate : p = 0.9, $T_{s,min}$ = 14.5°C; lower limit: p = 0.95, $T_{s,min}$ = 15°C)

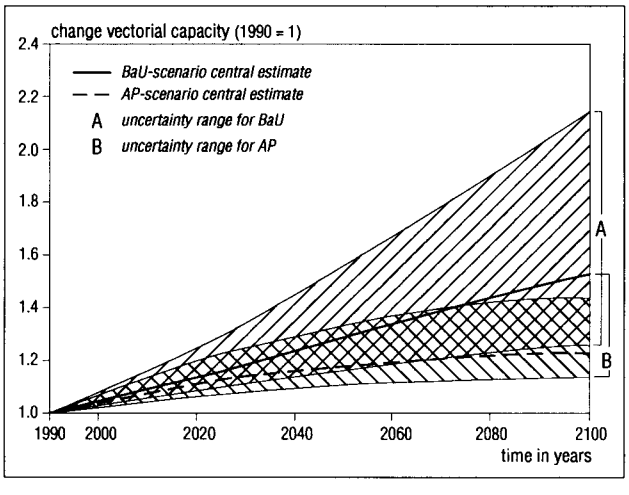
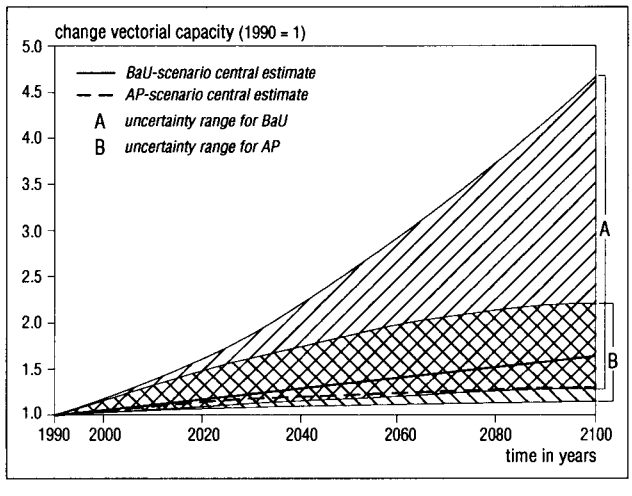


Figure 6.16: Uncertainty range of the simulated change in vectorial capacity according to the two IPCC-scenarios for endemic malaria regions (P.falciparum). (upper limit: p = 0.8, $T_{s,min}$ = 16°C; central estimate : p = 0.9, $T_{s,min}$ = 16°C; lower limit: p = 0.95, $T_{s,min}$ = 19°C)



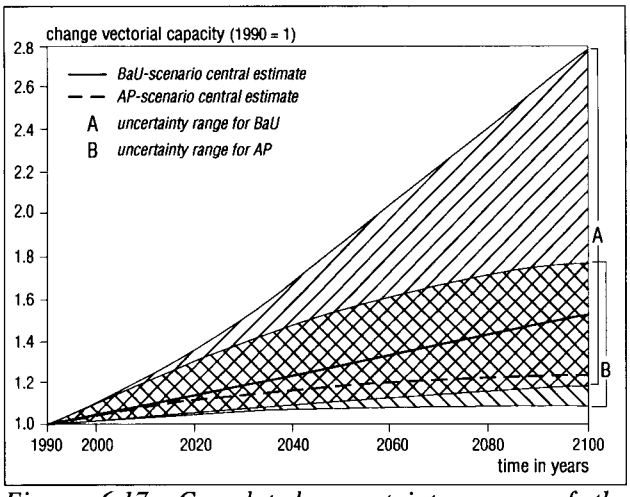


Figure 6.17: Cumulated uncertainty range of the simulated change in vectorial capacity according to the two IPCC-scenarios for endemic malaria regions (P.vivax). (upper limit: p = 0.8, $T_{s,min}$ = 14.5°C; central estimate : p = 0.9, $T_{s,min}$ = 14.5°C; lower limit: p = 0.95, $T_{s,min}$ = 15°C)

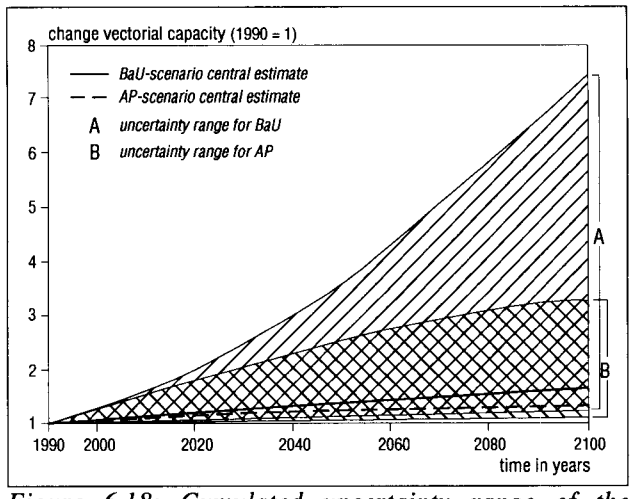


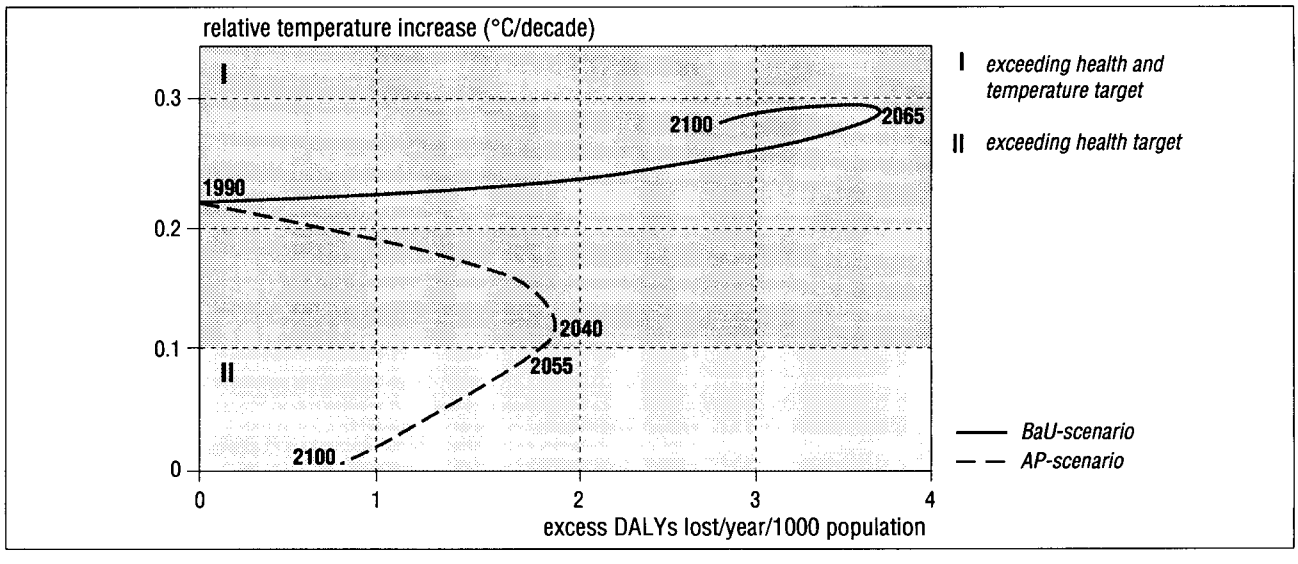
Figure 6.18: Cumulated uncertainty range of the simulated change in vectorial capacity according to the two IPCC-scenarios for endemic malaria regions (P.falciparum). (upper limit: p = 0.8, $T_{s,min}$ = 16°C; central estimate : p = 0.9, $T_{s,min}$ = 16°C; lower limit: p = 0.95, $T_{s,min}$ = 19°C)

and *6.16* with *Figures 6.17* and *6.18* shows that the main uncertainties arise in the calculation of the vectorial capacity. The large uncertainty range in the change of the vectorial capacity associated with *P.falciparum* results from the large range of minimum development temperature required for development of this parasite. The cumulation of uncertainties associated with uncertainties in parameters of the human system is not assessed here. To this end, a more elaborate uncertainty analysis will be performed in the near future.

6.4 Climate change and malaria: a sustainable development ?

In *Figures 6.19* and *6.20* the excess disease burden is expressed as a function of the relative temperature increase. The relative global mean temperature target of 0.1°C per decade is exceeded throughout the simulation period (1990-2100) according to the BaU-scenario, while the AP-scenario foresees the target being exceeded until the middle of the next century. The target of no increase of healthy life years lost due to malaria is not met in any of the calculations. The ratio of GNP growth to population growth in the decade from 1980-1990 was 0.6 for the malarious regions (which are all either low or middle income economies; World Bank, 1993). This value is lower than the target value of 2.5 below which malaria control is assumed to be difficult to establish, and projections show no major improvement in this ratio (World Bank, 1993). This

Figure 6.19: Two IPCC-1990 scenarios related to temperature and health targets for regions of high endemicity.



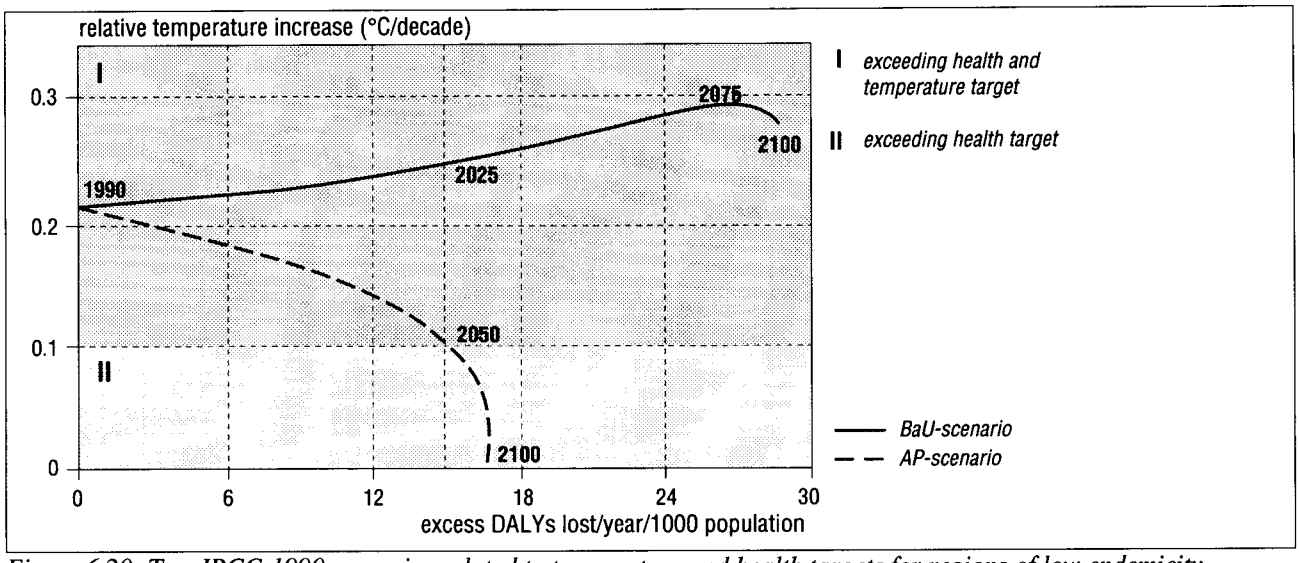


Figure 6.20: Two IPCC-1990 scenarios related to temperature and health targets for regions of low endemicity.

implies that available resources in the endemic areas will probably not be sufficient to enable the adaptive or preventive measures to be taken which would be required to deal with malaria adequately. In these regions, malaria remains a significant threat to human health and a deterioration in the situation is to be expected as a result of a human-induced climate change.

7. CONCLUSIONS AND DISCUSSION

Attempts to arrive at estimates of malaria risks associated with a human-induced climate change have been addressed in various other studies (e.g. Haile, 1989; Leaf, 1989; WHO, 1990; Doll, 1992; McMichael, 1993), although an integrated approach as presented in this report has hitherto not been adopted. The other studies also more or less restrict themselves to be descriptive. The model developed here is intended as a first step towards analyzing the impact of climate change on malaria risk more quantitatively.

An anthropogenic climate change affects the mosquito population directly, i.e. affecting mosquito development, feeding-frequency and longevity of the mosquito, as well as the maturation period of the parasite inside the mosquito. The results yielded by simulation experiments on the integrated model show a projected worldwide increase in transmission potential of the mosquito population and an extension of the areas conducive for malaria transmission as climate changes. A global mean temperature increase of approximately 3°C in the year 2100 (BaU-scenario) increases the epidemic potential of the mosquito population in tropical regions twice and more than 10 times in temperate climates. The risk of introduction of malaria transmission in non-malarious areas, including large parts of Australia, the United States, and Southern and Central Europe, associated with imported cases of malaria is a real one, since the former breeding sites of several *Anopheles* species are still available. Given the fact that in the most developed 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 malarious but not *Anopheles*-free areas will, however, be necessary. A different situation can be expected in currently endemic areas and areas bordering on them, especially in the subtropics. In the highly endemic malarious areas of tropical Africa, the malaria prevalence and consequently the number of years of healthy life lost due to malaria may increase. In the malarious areas of lower endemicity, however, the prevalence of infection is far more sensitive to climate changes. Therefore, a human induced climate change may have profound effects on prevalence and on years of healthy life lost in such areas. Worldwide, the model calculates an increase in malaria cases of several millions in the year 2100. Towards the middle of the next century more than one million people may die annually as a result of the impact of a human-induced climate change on malaria transmission.

The change in malaria risk as simulated must be interpreted within the framework of local conditions and developments in e.g. the prevailing health services, the parasite reservoir and mosquito densities. The extent of an increase in malarial risk will be superimposed upon the change in malaria transmission associated with socio-economic development and the (in)effectiveness of control measures. During all or part of the various simulation runs, there is a failure to meet targets for sustainability, defined in terms of temperature and human health. Given insufficient resources to deal with malaria adequately in the most affected regions, the anticipated risk of climate change tends to unacceptable levels.

Although the model only generates broad estimates of future trends and includes simplifications and assumptions, it nevertheless demonstrates that modelling the impact of a man-induced climate change on vector-borne diseases is feasible and can provide valuable insights into the interdependencies among climate change, vector population dynamics and human disease dynamics. In this study, the direct effects of a changing temperature and precipitation on malaria transmission were considered, no appraisal of the indirect effects of a human-induced climate change on the human and mosquito population could be attempted.

Additional research on the biological, ecological and socio-economic factors important in malaria transmission will be required for a more complete analysis of the impact of a human-induced climate change on this vector-borne disease.

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APPENDIX 1: Parameters used in the model

Mosquito subsystem

symbol	name	definition	value
VC	vectorial capacity	number of potentially infective contacts made by the vector vector population per infectious person per day; $m*b*c*a^2*p^n/-\ln(p)$	T P
VC_{crit}	critical vectorial capacity	minimum vectorial capacity needed to maintain malaria at a positive endemic level	P.vivax: 0.0009/day P.falciparum: 0.0027/day
m	relative vector density	density vectors in relation to man	#
m_{crit}	critical mosquito density	mosquito density needed to maintain malaria at a positive endemic level; $VC_{crit}*(-\ln(p))/b*c*a^2*p^n$	T P
b	infection probability humans	proportion infective bites on humans producing an infection	1
c	infection probability mosquitoes	proportion bites by susceptible mosquitoes on infectious people producing an infection	1
a	man-biting habit	number of blood meals taken on man per mosquito per day; HBI/DUR_{bd}	T
HBI	human blood index	proportion of blood meals of a mosquito population taken on man	varying between 0.1 for temperate and 0.9 for tropical climates
DUR_{bd}	duration blood digestion	days needed for the digestion of a blood meal; $DD_{bd}/T\text{-}T_{min,bd}$	T
DD_{bd}	degree-days needed for digestion of portion blood	number of degree-days needed for the blood digestion	36.5 °C day
$T_{min,bd}$	minimum temperature required for blood digestion	minimum temperature required for the digestion of the blood meal	9.9 °C
p	daily survival probability	probability that the vector survives one day	T
n	duration sporogony	incubation period of the parasite in the vector in days; $DD_S/(T\text{-}T_{min,s})$	T
DD_S	degree-days needed for sporogony	number of degree-days needed for the development of the parasite inside the vector	P.vivax: 105 °C day; P.falciparum: 111 °C day
$T_{min,s}$	minimum temperature required for sporogony	minimum temperature required for parasite development	P.vivax: 14.5 °C; P.falciparum: 16 °C

Human subsystem

symbol	name	definition	input value
HSUS	human susceptible	number of susceptible individuals in the human population	*
HINF	human infectious	number of infectious individuals in the human population	*
HIMM	human immune	number of immune individuals in the human population	*
HINFp	proportion infectious	proportion of infectious individuals in the human population	*
μ_h	death rate	number of deaths per capita	0.015/year
β_{age}	age specific fatality rate	number of deaths due to malaria infection	P.vivax: 0% P.falciparum: age -5: 4% age 5+: 1%
RI	rate of infection	probability that a susceptible person acquires the infection; $1\text{-}e^{-VC*HINFp}$	*
LIF_{age}	age specific actual loss rate of infectiousness	rate of losing infectiousness (recovery rate) depending on the rate of infection; $RI/(e^{RI/BLIFage}\text{-}1)$	*
LIM	loss rate of immunity	rate of losing immunity depending on the rate of infection; $RI/(e^{RI/BLIM}\text{-}1)$	*
$BLIF_{age}$	age specific basic loss rate of infectiousness	rate of losing infectiousness if no reexposure takes place; 1/average duration of infectiousness	P.vivax: age -5: 0.33/year age 5+: 1.65/year P.falciparum: age -5: 1/year age 5+: 5/year
BLIM	basic loss rate of immunity	rate of losing immunity if no reexposure takes place; 1/average duration of immunity	0.67/year

Health impact:

symbol	name	definition	input value
DB_{mal}	disease burden malaria	numbers of years of healthy life lost due to malaria	^
$DB_{mal.death}$	disease burden due to death	numbers of years of healthy life lost due to malaria mortality	^
$DB_{mal.dis}$	disease burden due to disability	numbers of years of healthy life lost due to malaria morbidity	^
DEATHS	malaria deaths	deaths due to malaria infection	^
INC(age)	age specific malaria incidence	numbers of new malaria cases per year	^
$DALYs_{death}$ (age)	age specific disability adjusted life lost by death	years of healthy life lost by death and age, discounted and age-weighted	age -5: 32.5 years age 5+: 25 years
SW	severity weight	severity measure of the disability in comparison with loss of life	0.4
DC	duration disability condition	expected duration of the condition of disability	0.041 year (=15 days)

Notes: T = depending on temperature inputs from the climate system
P = depending on precipitation inputs from the climate system
* = depending on inputs from the mosquito system
^ = depending on inputs of the human system
# = the initial value of the mosquito density is based on the initial vectorial capacity and is assumed to remain constant during the simulations

APPENDIX 2: Values of daily survival probabilities of several Anophelines.

Anopheles species	p	reference
A.albimanus	0.65-0.91[a]	Weidhaas, *et al.* (1974)
A.antroparvus	0.85	Horsfall (1955)
A.coustani	0.89	Garret-Jones & Grab (1964)
A.culcifaries	0.94	Zoysa, de, *et al.* (1988)
A.freeborni	0.97	Horsfall (1955)
A.freeborni	0.72-0.75	McHugh (1989)
A.funestus	0.91	Garrett-Jones & Grab (1964)
A.funestus	0.89	Service (1965)
A.funestus	0.89	Garret-Jones & Shidrawi (1969)
A.funestus	0.82-0.87	Molineaux & Gramiccia (1980)
A.gambiae	0.91-0.93	Davidson (1954)
A.gambiae	0.88	Garret-Jones & Grab (1964)
A.gambiae	0.90	Service (1965)
A.gambiae	0.83-0.88	Garret-Jones & Shidrawi (1969)
A.gambiae	0.76-0.94[b]	Zahar (1974)
A.gambiae	0.82-0.87	Molineaux & Gramiccia (1980)
A.jeyporiensis	0.81	Khan & Talibi (1972)
A.messae	0.81-0.85	Horsfall (1955)
A.minimus	0.90	Khan & Talibi (1972)
A.nili	0.84	Garret-Jones & Grab (1964)
A.pharoensis	0.62[b]	Zahar (1974)
A.philippinensis	0.72	Khan & Talibi (1972)
A.quadrimaculatus	0.97[d]	Horsfall (1955)
A.sacharovi	0.64-0.85[c]	Horsfall (1955)
A.sinensis	0.96[d]	Horsfall (1955)
A.stephensi	0.83-0.87	Zahar (1974)
A.vagus	0.96[d]	Horsfall (1955)
A.vagus	0.80	Khan & Talibi (1972)

Notes:
[a] wet season p between 0.73-0.91
dry season p between 0.65-0.70
[b] under insecticide spraying
[c] low values according to low relative humidity
[d] bred in cages