Journal of Global Environment Engineering, Vol. 1, pp. 43–57, 1995

AN ESTIMATION OF CLIMATIC CHANGE EFFECTS ON MALARIA

Yuzuru Matsuoka[1]
Keiko Kai[2]

Abstract

In this study, we focus on one of the socio-economic impacts of global warming, that of human health, and quantitatively estimate the increased risk of malaria infection. The climate change expected in the next century will cause a surface temperature rise, an increase in precipitation, and a sea level rise. These changes are expected to affect human lives in various ways such as land loss, impacts on agricultural productivity, natural ecosystem, and human health. In this study, we will first describe the estimated magnitude of these impacts and then focus on the health risk by malaria. For malaria, in particular, although the impacts on propagation of *Anopheles* (the disease vector) and so on are considered to greatly increase the likelihood of the disease, these factors have never been evaluated quantitatively. In this study, we estimate the increase in risks to health caused by malaria. First, we estimated the current and post-global warming distribution of *Anopheles* by calculating its eco-climatic matching using annual and daily temperature and soil moisture as basic parameters. These parameters were deduced from a water balance model based on General Circulation Models' results. Then, we quantitatively analyzed the change in malaria endemicity taking into account the relationship between temperature and the duration of sporogony of malaria parasites in the *Anopheles*. We concluded that climate change caused by a doubling of CO_2 will allow the malarial area to increase by 10%~30% (population percentage).

KEYWORDS: *Climate Change, Malaria*

1. What are the impacts of global warming?

By March 1994, more than 50 nations had ratified the Framework Convention on Climate Change which was adopted at the Earth Summit in 1992 and agreed to apply its provision. The objective of this convention is *to achieve stabilization of greenhouse gas concentrations in the atmosphere at a level that would prevent dangerous anthropogenic interference with the climate system.* Researchers around the world were requested to quickly identify the routes of, and to evaluate the degree of, *dangerous interference* which would be caused by climate change (Bolin, 1994). Determining *the allowable range of climate change* is one of the ultimate aims of the research on global warming and can not be completed lustily. The range of impacts is quite diverse so it is not possible to treat them all in the same way. Many problems remain unsolved if we try to estimate them quantitatively. However, there is some research which has tried to comprehensively quantify global warming damage. Nordhaus (1991), Cline (1992), Fankhauser (1992), Titus (1992), and Hohmeyer *et al.* (1992) estimated the accumulated damage caused

1 D. Eng., Associate Professor, Faculty of Engineering, Kyoto University, Kyoto 60601 Japan
2 Senior Researcher, Global Env. Div., National Institute for Environmental Studies, Tsukuba 305 Japan

Table 1. An estimate of the damage caused by global warming (billion US$, km^2 area : lost area, add. death: increased number of death, source: Fankhauser, 1992)

sector	EC	USA	OECD†	CIS	China	ROW‡	World
coastal defense	0.1	0.2	0.2	0.0	0.0	0.6	1.1
dryland loss	0.3	2.1	5.7	1.2	0.0	4.7	14.0
(km^2 area)	1,596	10,695	28,129	23,920	0.0	75,583	139,923
wetland loss	4.9	5.6	6.4	1.2	0.6	12.9	31.6
(km^2 area)	9,887	11,121	12,854	9,788	11,918	197,417	252,985
ecosystems loss	9.8	7.4	8.3	2.3	2.2	10.5	40.5
agriculture	9.7	7.4	6.0	6.2	7.8	2.0	39.1
forestry	0.2	1.0	1.8	0.6	0.0	-0.2	3.4
energy	7.0	6.9	6.6	-0.7	0.7	2.6	23.1
water supply	14.0	13.7	7.1	3.0	1.6	7.3	46.7
life/morbidity	21.9	16.6	18.8	3.9	4.9	15.9	82.0
(add. deaths)	14,625	11,070	12,210	12,870	48,690	129,780	229,545
air pollution	3.5	6.4	2.0	2.1	0.2	1.2	15.4
migration	1.0	0.5	0.5	0.2	0.6	1.5	4.3
Nat. hazards	0.0	0.2	0.9	0.0	0.1	1.8	3.0
(add. deaths)	0	80	272	49	876	7,723	9,000
Total(bn$)	72.4	68.0	64.3	20.0	18.7	60.8	304.2
(% GNP)	1.6	1.4	1.2	0.8	5.3	2.2	1.5

OECD†: OECD excluding EC and USA, ROW‡: Rest of the World

by a doubling of CO_2. Nordhaus, Cline, and Titus estimated the situation for the U.S., while Fankhauser and Hohmeyer *et al.* looked at the global situation. Table 1 shows typical results obtained by Fankhauser(1992). Wide range of impacts, e.g. land loss caused by sea level rise, agriculture, forestry and fisheries, natural ecosystem, water use, human health, air and water pollution, and intensification of natural disasters were considered and the economic damages of these impacts summed up to 1.5% of the GNP, assuming the temperature rise to be 2.5°C (a global average) and the sea level rise to be 50 cm. Nordhaus, Cline and Titus estimated almost the same level of damage (1.0%~2.5% of GNP for the U.S.). On the other hand, Hohmeyer *et al.* calculated a huge damage cost that mounted to US$ 10 trillion (50% of GNP) and estimated the increase in the number of death due to heat stress, tropical diseases, and storms to be 3 millions. Such differences were caused by the methods these researchers chose and extrapolated information of low reliability. Although these incompatibilities are often seen and each of the estimation has been criticized, many researchers agree on the seriousness of the future damage to human health.

2. What are the impacts of global warming on human health?

As for the impact of global warming on human health, a number of studies have been conducted (such as WHO, 1990). These studies assert that there are comparatively direct influences on human health, such as an increase in heat stress, nutrition disorders caused by

Table 2. Infections influenced by global warming (source: mainly based on WHO, 1990)

Disease	Population at risk	Prevalence of infection	Present distribution	Possible change by climate change
Malaria	2530	270	tropics/subtropics	+++
Dengue	NA	NA	tropics/subtropics	++
Schistosomiasis	600	200	tropics/subtropics	++
Lymphatic filariasis	900	90	tropics/subtropics	++
Onchocerciasis	90	18	Africa/Latin America	+
African trypanosomiasis	50	0.025 new cases/y	tropical Africa	+
Yellow fever	NA	NA	Africa/Latin America	+
Japanese encephalitis	NA	NA	East/South East Asia	+
Leishmaniases	350	12	Asia/S.Eurp. /Africa/S.Amer.	?

million people, +++ = highly likely, ++ = very likely, + = likely, ? = not known

changes in food supply, and changes in disease propagation mechanisms. Moreover, there are comparatively indirect influences, such as stress increases by air and water pollution, disasters caused by sea level rise, and the increase in floods. In relation to the increase in the death rate caused by heat stress, Kalkstein (1989) estimated an increase of about 7000 deaths a year (only in the urban areas of the U.S.) by climate changes based on the GCM calculation results by Goddard Institute of Space Sciences (GISS). His study was based on an analysis of city mortality statistics in the U.S. and took into account adaptation to heat stress. Fankhauser extrapolated this result (a mortality rate of 4.5 in 100,000 people) to the whole world and estimated that the economic loss would amount to 31% of all the economic impacts by global warming. As for the impacts on food availability, Rosenzweig *et al.* (1994) conducted an international project, with the research organizations of 18 countries and concluded that the number of people at risk of hunger would be 640 millions in 2060 without global warming. And the global warming would increase this figure by 10%~60%. As for changes in infection mechanisms, various factors such as changes in vector ecosystems or deterioration of drinking water quality caused by hydrological changes (WHO, 1990) have been indicated. Malaria, schistosomiasis, dengue fever, and other diseases shown in Table 2 are estimated to have great social impacts resulting from climate changes because their current impacts are serious and their climate sensitivities are high.

Currently, the human population in malarial areas is estimated to be more than 2 billions, with the number of sufferers at 270 millions a year, and the number of deaths per year to be more than 1 million (WHO, 1992). It is still a disease with a grave effect on human health. Although the number of deaths it causes in the Sub-Sahara region is estimated to be quite high, information is unreliable and the exact situation is not clear. Other areas such as Afghanistan, Brazil, China, India, Mexico, the Philippines, Sri Lanka, Thailand, and Vietnam account for 83% of the sufferers. However, some countries and areas have a growing death rate because of an increased number of refugees, public disorder, and the appearance of chemically tolerant

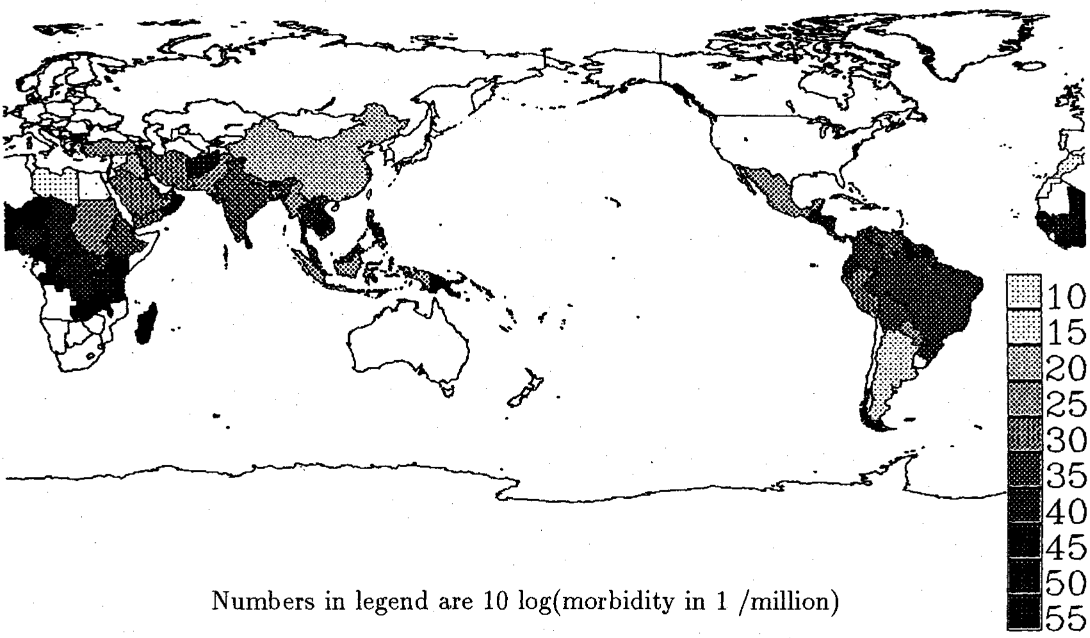


Figure 1. Annual malaria morbidity, source: recent issues of WHO World malaria situation, WHO.

parasites. Also, it has been reported that malaria morbidity in these countries is connected with climate change caused by El Niño (Nicholls, 1993). It is believed that climate change resulting from global warming will worsen the situation. Figure 1 presents the latest malaria death rates.

3. How are climatic factors related to malaria endemicity?

Malaria is caused by malarial parasites when they enter the human body as sporozoite through the bite of the *Anopheles* mosquito. The malarial parasites are Protozoa. Important species causing malaria are *Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae,* and *Plasmodium ovale.* The most dangerous one is *Plasmodium falciparum.* Its life cycle consists of pre-eryhrocytic phases in human liver cells and red blood cells plus sporogony. They are injected by mosquitoes and develop to microgametes and macrogametes in the mosquito's stomach. Microgametes fertilize macrogametes, to produce zygotes.

After fertilization, the zygote bores into the gutwall and forms a small cyst called an oocyst, in which sporozoites are produced. Eventually, the oocyst ruptures and liberates a great number of sporozoites. They invade the mosquito's salivary glands and are transmitted to humans when the mosquito bites again. Thus, propagation of the malaria parasite has two stages, one each inside the bodies of men and the *Anopheles.* It will not be directly affected by climate while inside a man. However, when inside the *Anopheles,* temperature has a great influence on its sporogony. Sporogony is impossible when the temperature is below 16°C and is impeded when it is over 32°C. The number of days needed for sporogony depend on the kind of parasite and on temperature, for which MacDonald (1957) proposed the following formula:

$$n = \frac{M}{T - T_0}, \qquad T \geq T_0 \tag{1}$$

Here, n is the number of days needed for sporogony, T is temperature, M and T_0 are constants determined by the kind of parasite, which, in the case of *Plasmodium falciparum*, are 111°C·d, 16.0°C. The ecology of *Anopheles*, the malaria vector, depends greatly on climate. About 400 species of *Anopheles* have been found so far, 10% of which are important malaria vectors. They belong to Holometabola and have four metamorphoses which are egg, larva, pupa, and imago. The locations for egg laying vary with species; some prefer still water whereas others prefer running water. The area of activity sometimes extends to several kilometers away from the breeding places. Availability of water is an important factor of their propagation. Adults are considered to be unable to survive the dry season. Their ecology changes greatly with temperature. Although they can survive a temperature of 12.7°C if the atmosphere is saturated, their optimum conditions is considered to be a relative humidity of 60% and temperature between 22°C ~ 30°C . Within this temperature range, their growth rate increases as the temperature rise. However, high temperatures shorten their life span, so their numbers and population density declines (Dutta *et al.*, 1978).

How are these relationships between climatic factors and life cycles of malaria parasites and *Anopheles* generally connected with malaria endemicity? To analyze this, we will use the following symbols: m: the number of vectors per person, a: the frequency of bites of a vector per day, p: daily survival probability, r: the recovery and death rates of patients. The relationship between the average life length of vectors e and p is expressed by the following formula.

$$e = \int_0^\infty p^t dt = \frac{1}{-\log_e p} \tag{2}$$

We will conduct an analysis of malaria infection using these symbols and the above formula. An infected person is bitten by $m \cdot a$ mosquitoes a day. The mosquito which is inoculated with gametocytes of malaria parasite needs to survive for more than n days, in order to obtain malaria endemicity. Thus, $m \cdot a \cdot p^n$ mosquitoes per day will have an endemicity from one person. Their average life span is $1/(-\log_e p)$ and so they infect a person a day during that period. Accordingly, one infected person will produce $C = ma^2p^n/(-\log_e p)$ infected people each day. Considering such production and also allowing for recovery and death expressed with r, the differential equation for the number of infections P is as follows,

$$\frac{\mathrm{d}P}{\mathrm{d}t} = (C - r) \cdot P \tag{3}$$

C is an index which Garrett-Jones *et al.* (1969) calls the *vectorial capacity*. In deriving this formula, many assumptions must be introduced. For example, the rate of infection is low and the following bite is of a non-infected person. The impacts of superinfection are not taken into account, the transfer of parasites by biting and their developments are assumed to be effective, the death rate of vectors is fixed independently of age, and the life-span of mosquitoes is not altered by infection. However, we consider C or C/r (= R_0: the basic reproduction rate used by MacDonald) as first approximates which represent the possibility of the occurrence of malaria. Thus, we assume that the impacts of climate change will effect malaria endemicity through the parameters C or R_0.

As mentioned above, m and n differ greatly with temperature, whereas m also depends on moisture. Although a and p have also been reported to vary with temperature and humidity,

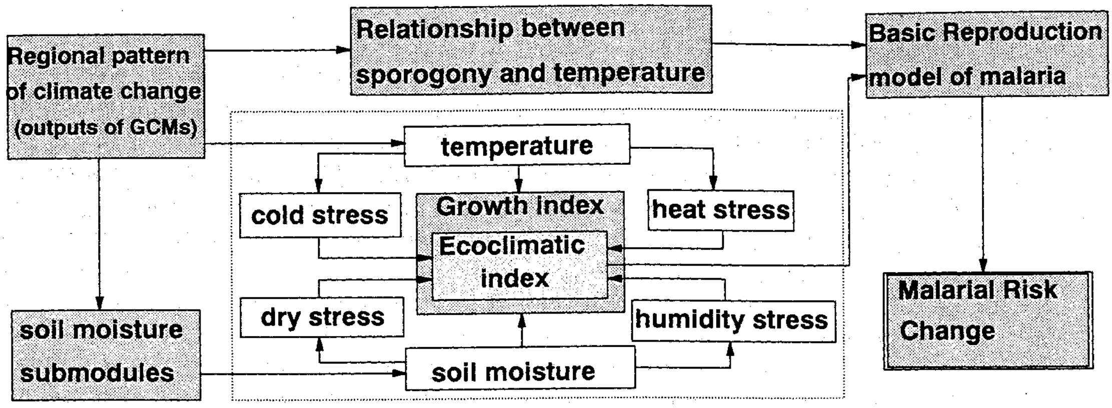


Figure 2. Assessment framework of this study

they have never been quantitatively evaluated. Thus, in this study, we assume that climate change will affect malaria endemicity through changes in m and n.

Figure 2 shows the assessment framework used in this study. The major components of the frame are the relationship between sporogony and temperature and the ecoclimatic index model which shows climatic response of vectors. Complemented with these components, a soil moisture submodule and outputs from equilibrium experiments with GCMs are attached.

The primary climatic variables of this framework are surface temperature and precipitation distributed spatially and temporally in both the current situation and that after climate change. The response of the ecoclimatic index will be given as the change of m, and the response of sporogony to temperature will be given as the change of n. These are combined as the basic reproduction rate and used to estimate the changes in the areas of malaria occurrence.

Modeling the prevalence dynamics of malarious infection has been studied over a long time (Ross, 1977; MacDonald, 1957; Dietz *et al.,* 1984). Research to establish the vector's population dynamics has also been conducted (Haile *et al.,* 1977; Fine *et al.,* 1979). However, these studies are aimed to support epidemic prevention and did not focus on climate change. Although some research on climate change (Haile, 1989; Sutherst, 1993) has also been conducted recently, mechanisms taken into consideration are limited in order to estimate the total figure of the impacts of malaria. In this study, we made an effort to integrate the knowledge and information of former research on the relationships with climate parameters, based on the framework given in Figure 2, and to evaluate the impacts on global human health.

4. Climatic preferences of *Anopheles*

As shown in the previous section, the relative population density of *Anopheles*, as expressed by m, depends greatly on temperature and moisture. To describe the dependence quantitatively, it is necessary to estimate various parameters of their life cycle and to develop a population dynamics model based on them. However, with the knowledge available at this time, it is almost impossible to do so for each of the 40 species of vectors, and this situation will last for some time. Thus, we chose a simpler way than the above. We accepted the ecoclimatic index (EI) proposed by Sutherst *et al.* (1985) in order to estimate the impacts of climate change on m. EI is composed of climatic factors which are concerned with growth and with stress, and describes the favorability of climatic outcomes as their products.

$$EI = GI \cdot \max(1 - CS, 0) \cdot \max(1 - DS, 0) \cdot \max(1 - HS, 0) \cdot \max(1 - WS, 0) \tag{4}$$

Where, GI is the index related to growth, CS is the stress caused by coldness, DS is that for dryness, HS is that for heat, and WS is the stress caused by humidity. GI is estimated by integrating the product of the temperature index TI and the moisture index MI.

$$GI = \frac{\int_{\text{within a year}} TI_t \cdot MI_t \cdot dt}{\text{time length of a year}} \tag{5}$$

TI_t is the product of $I_{Q,t}$ and $I_{H,t}$,

$$TI_t = I_{Q,t} \cdot I_{H,t} \tag{6}$$

I_Q is the normalized accumulated temperature which was calculated with the following formula for inter-diurnal variation of the surface temperature.

$$I_Q = \frac{\int_{\text{daily cycle}} \max(T - T_{V0}, 0) \cdot dt}{(T_{V1} - T_{V0}) \cdot \text{time length of a day}} \tag{7}$$

I_H is the growth inhibition function which is linearly interpolated between the lower temperature T_{V2} where inhibition starts and the higher temperature T_{V3} where the growth stops.

$$I_H = \begin{cases} 1 & T_{\text{daily max}} \leq T_{V2} \\ \dfrac{T_{V3} - T_{\text{daily max}}}{T_{V3} - T_{V2}} & T_{V2} < T_{\text{daily max}} \leq T_{V3} \\ 0 & T_{V3} < T_{\text{daily max}} \end{cases} \tag{8}$$

MI_t is calculated from the soil moisture index SM ((soil moisture + surplus moisture)/field moisture capacity) using the following equation.

$$MI = \begin{cases} 0 & SM \leq SM_0 \\ \dfrac{SM - SM_0}{SM_1 - SM_0} & SM_0 < SM \leq SM_1 \\ 1 & SM_1 < SM \leq SM_2 \\ 1 - \dfrac{SM - SM_2}{SM_3 - SM_2} & SM_2 < SM \leq SM_3 \\ 0 & SM_3 < SM \end{cases} \tag{9}$$

Where SM_0 and SM_3 are the lower and upper boundaries of soil moisture for the mosquito to survive and SM_1 and SM_2 are the preferable boundaries for the insect. The stress indices are integrated values of the stress within a year which occur when climatic factors exceed their thresholds. The calculation was conducted each week as a unit time length. For example, CS is estimated as follows:

$$CS = \frac{\sum_{\text{within a year}} \sum_t \max(0, T_{CS} - T) \cdot H_{CS} \cdot t_{\text{duration}}}{\text{time length of a year}} \tag{10}$$

Here, T_{CS} is the lower critical temperature, whereas H_{CS} is its stress rate and t_{duration} is the time after the stress begins. Stresses of dryness, heat, and moisture are also expressed by a similar equation. Based on this equation, the effects of stress increase rapidly when these conditions continue.

Table 3. Model parameters of *Anopheles*

i	0	1	2	3
SM_i	0.1	0.65	2.0	4.0
T_{Vi}	12.7	22.0	30.0	40.0
SM_{DS}	0.2	H_{DS}	0.001	
SM_{WS}	0.2	H_{WS}	0.002	
T_{CS}	12.7	H_{CS}	0.0001	
T_{HS}	35.0	H_{HS}	0.001	

A lot of research, such as by Dutta *et al.* (1978), on global ecology of *Anopheles* has been conducted. Using this research, we set each parameter that appears in EI as shown in Table 3. By the parameters, the suitable temperature for *Anopheles* is 22 $\sim$ 30°C. They are exposed to severe temperature stress when the temperature is below 12.7°C and above 35°C, and cannot exist when it is over 40°C. The soil moisture parameters were based on the favorable conditions in its current distribution estimated using the soil moisture model which is explained in the next section.

5. Temperature and Soil Moisture Models

For the analysis in the previous section, information on inter-annual and diurnal variation of temperature and soil moisture is necessary. In this study, we used the monthly mean temperatures calculated by Legates *et al.* (1989) for the current inter-annual temperature variation. As for the inter-diurnal variation, we used a diurnal temperature range compiled as databases by NCAR(ds512.0, 1979~1992, 7500 points) and CLIMEX by CSIRO (Maywald *et al.* , 1991), assuming a sine curved variation.

Soil moisture was estimated from a surface water balance model based on temperature, precipitation, and field moisture capacity. Though the GCMs also calculate soil moisture, it is not suitable for impact studies such as what we intended in this paper (Argonne National Laboratory, 1994). This model estimates soil moisture W and evapotranspiration ET from monthly precipitation P, temperature T, and potential evapotranspiration PET. Soil moisture is dependent on the balance of precipitation Pr, snow melt Rs, and PET. It can be increased to the value of the field moisture capacity FC in humid months when the sum of precipitation and snow melt are more than PET. For dry months, when this value is less than PET, we calculated it using a formula that assumes a linear relationship between $\Delta \log SM$ and $\sum[PET - (Pr + Rs)]$ (Vorosmarty *et al.* , 1989):

$$\frac{dSM}{dt} = \begin{cases} (Pr + Rs - PET) & Pr + Rs \geq PET, SM < FC \\ 0 & Pr + Rs \geq PET, SM = FC \\ -a \cdot SM[PET - (Pr + Rs)] & Pr + Rs < PET \end{cases} \tag{11}$$

Where a is determined from the field moisture capacity FC(mm) using the following formula (Pastor *et al.* , 1984).

$$a = \frac{\ln(FC)}{(1.1282 \cdot FC)^{1.2756}} \tag{12}$$

As for the remaining surplus moisture that is in excess of the field moisture capacity, some

Table 4. Used GCM outputs and estimated malaria risks

	Present	GFDL-R30	CCC	GISS	OSU	UKMO	Qflux
equilibrium surface temperature change on doubling CO_2, °C							
global	-	4.04	3.49	4.17	2.85	5.29	3.98
land	-	4.33	4.33	4.45	3.10	6.34	4.38
percentage change in precipitation, %							
global	-	11.4	5.8	14.6	10.8	22.1	8.6
land	-	16.9	5.0	20.8	17.4	24.4	9.8
population of malarious area (millions)							
$R_{\mathrm{model}} > 1.0$	2315	2771	2525	2654	2463	2783	2614
$R_{\mathrm{model}} > 6.5$	1130	1518	1248	1509	1289	1541	1355
land coverage ratio of malarious area							
$R_{\mathrm{model}} > 1.0$	0.335	0.425	0.392	0.412	0.358	0.427	0.406
$R_{\mathrm{model}} > 6.5$	0.160	0.238	0.199	0.236	0.178	0.244	0.212

is removed as surface runoff and the rest is stored as surface impoundment. Precipitation is treated as rainfall when the temperature is above 0°C and as snow when it is below 0°C. When the temperature is above 0°C the amount of snow melt is assumed to be $6 \cdot T + 0.0125 \cdot Pr \cdot T$ (Sugawara, 1972). Potential evapotranspiration was estimated using the Thornthwaite equation. Although the use of this method on a global scale may be disputed, Mintz *et al.* (1993) and Vorosmarty *et al.* (1989) reported that it does not produce values that differ greatly from observed or estimated values by other methods. Field moisture capacity depends on vegetation and soil properties. On this, some research has been conducted and several sets of global data have also been published (Wilson *et al.*, 1985, Webb *et al.*, 1993, Bouwman *et al.*, 1993). In this study, we compared the surface runoff estimated by the model with the above data sets and observed river discharges reported by McMahon (WMO Infoclima #1067), then adopted the most fitted moisture capacity data sets. The field moisture capacity we adopted was the lowest of the three values Webb *et al.* estimated from soil texture, soil-profiles, root-zone thickness. As for soil moisture and runoff, we employed the results a typical year after several years' spin up calculation.

Spatial resolution of 0.5° × 0.5° was used in the water calculation and averaged to 1° × 1° grids when calculating with the mosquito's model.

The climate parameters for global warming were obtained by adding the change profile from GCM calculations to the observed temperature, or multiplying ratio changes with the observed precipitation profile of Legates *et al.* (1989). As for inter-diurnal ranges of temperature, we assumed no change. Table 4 presents the climate sensitivities of the 6 GCMs we used. The names of GCMs' outputs follow the IPCC 1990 report (Houghton *et al.* 1990). Of these, GFDL R-30 was estimated at NOAA Geophysical Fluid Dynamics Laboratory, USA, in 1989 and we used it as the standard case of this study, because it was the high resolution calculation of 3.75° × 2.23° and a number of calculation variables were released. In the rest of this paper we exemplify the results of GFDL R-30 except where otherwise stated.

6. To what extent is the spread of malaria affected?

We stated in section 3 that the change in malaria endemicity could be estimated from the change in the basic reproduction rate R_0. In this paper, we considered only the relative change in R_0 and did not take its absolute value into account. Thus, we first normalized EI in an arbitrary range and assumed that its relative change is proportional to that of m. Also by assuming that the changes in r and a can be neglected, the change in R_0 can be observed from the change in R_{model} in the following equation. We focused on *P. falciparum* which has the

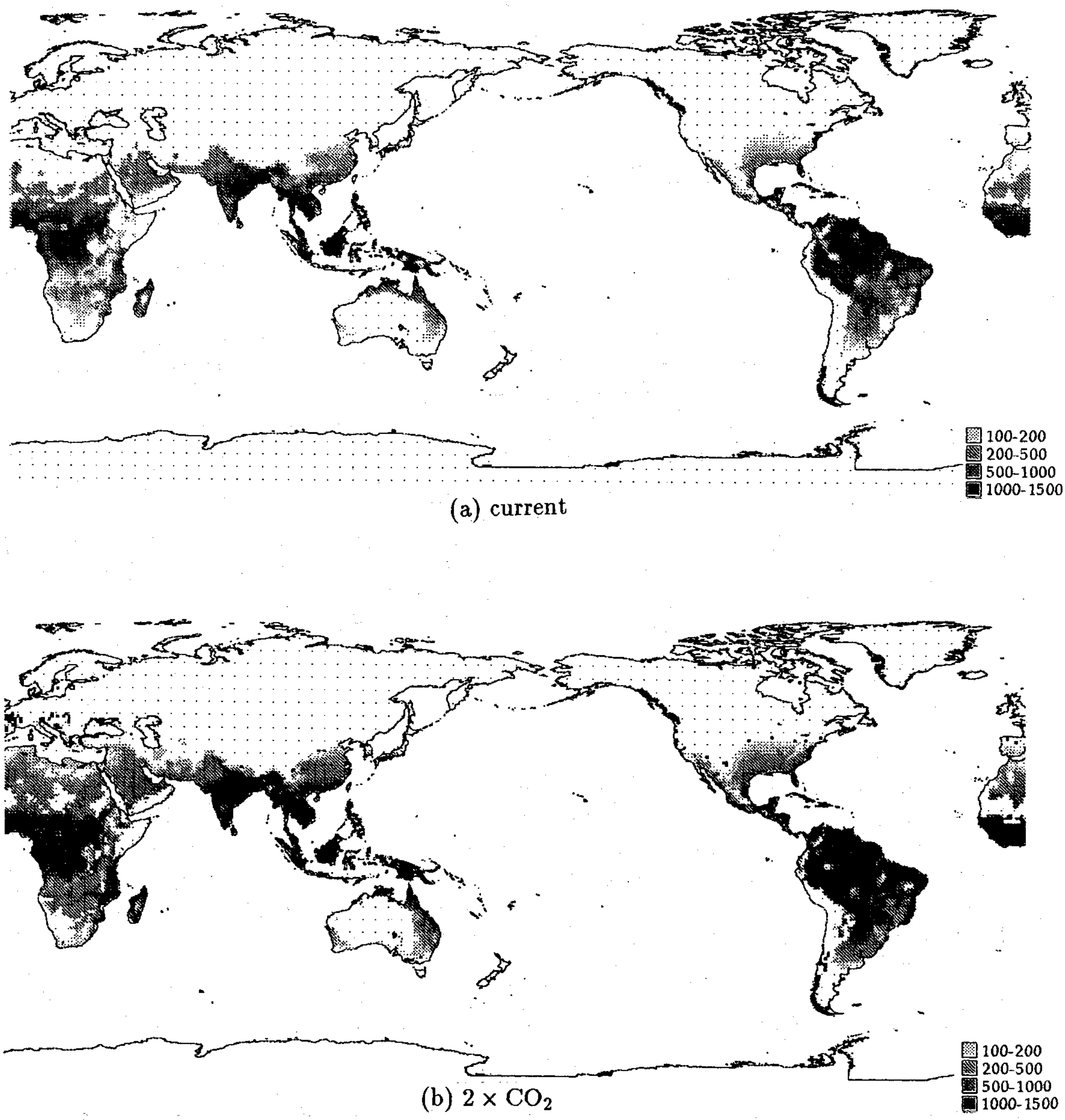
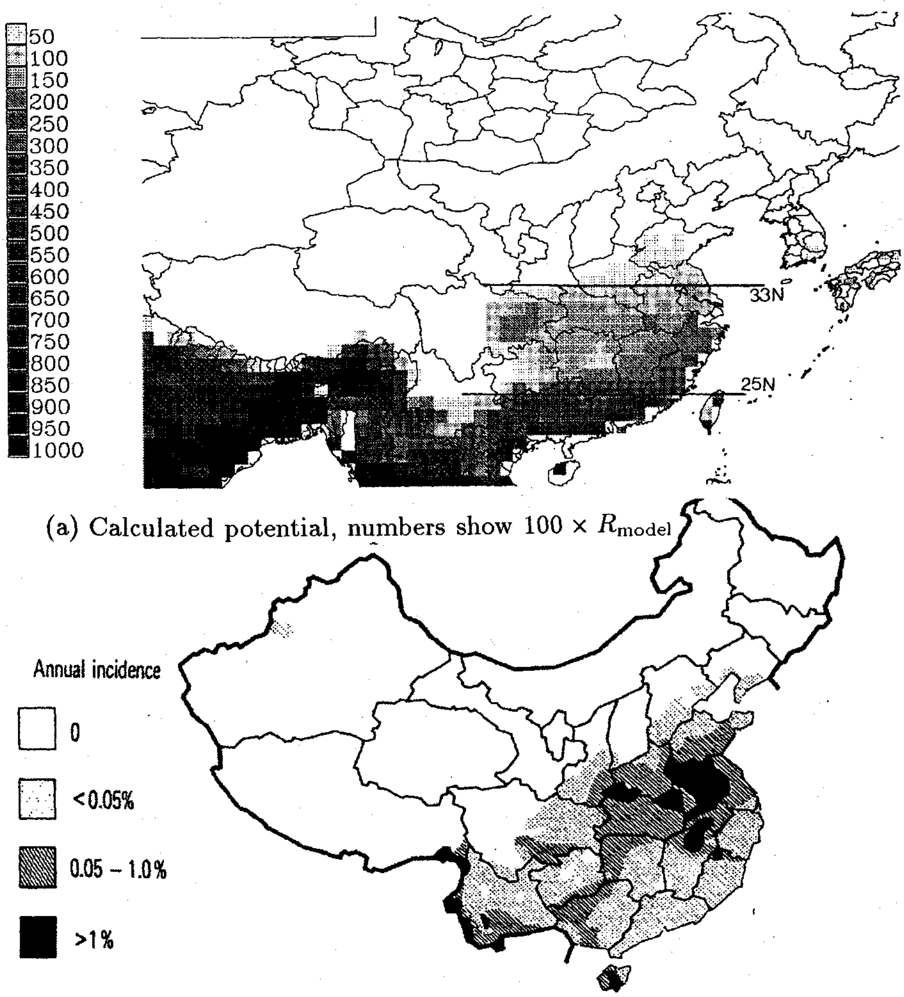


Figure 3. Relative reproduction rate (a) current, (b) $2 \times CO_2$



(a) Calculated potential, numbers show 100 × R_{model}

(b) Annual incidence of malaria, 1979, source: Zuou, 1981

Figure 4. Calculated potential and annual incidence of malaria in China

highest death rate for malarias. To make the calculation simple, we fixed p at 0.8(1/d).

$$R_{\text{model}} = EI \cdot \frac{p^n}{-\log_e p} \tag{13}$$

Figure 3 presents the R_{model} profiles for both the current climate and that when CO_2 is doubled. We can see that the malarial area is not only concentrating in the three tropical areas of Africa, America, and Asia, but also widely dispersed in China, Japan, the United States, Mediterranean nations, and Australia. Values in the figure show 100 × R_{model}.

As for this calculation, what level is the significant for malaria epidemicity? China experienced severe levels of malaria during the 1940's with about 30 million victims a year. During

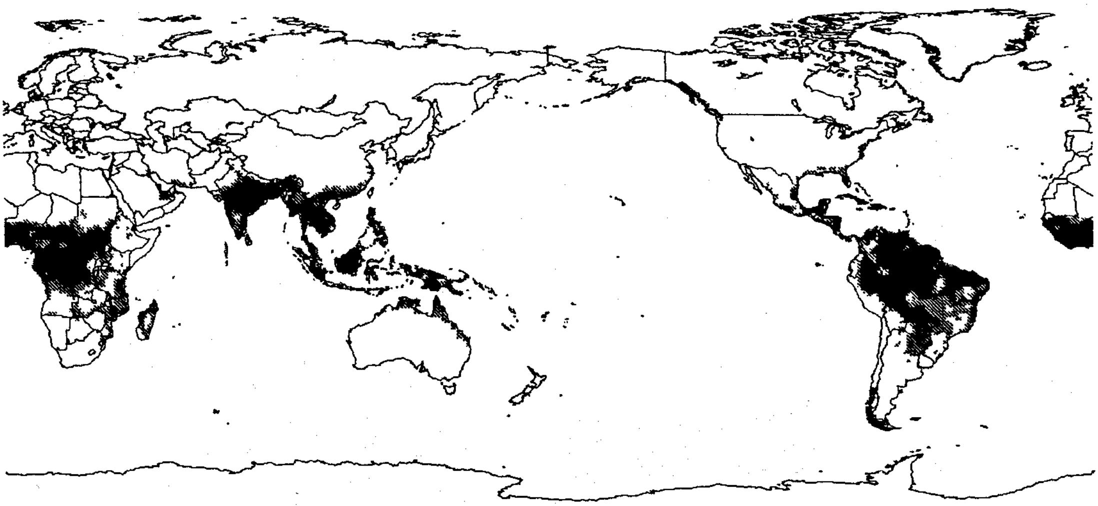


Figure 5. Extend of malaria endemic areas caused by global warming

the period when eradication efforts were not enough, it was hyperendemic in areas south of 25N, and mesoendemic in areas between 25~33N and east of the Szechwan Basin(CACPD, 1989).

Figures 4(a) and 4(b) are the comparison of $100 \times R_{\text{model}}$ and recent observed malaria morbidity (Zuou, 1981) in these areas, and we assume that the risk level of malaria becoming mesoendemic when R_{model} is over 1.0, and hyperendemic when R_{model} is over 6.5.

Figure 5 shows how the areas where R_{model} is over 6.5 will expand because of global warming. Black areas are the current hyperendemic areas and gray areas are those in which malaria will become hyperendemic because of global warming. The fringelands of current malaria occurring areas such as the southern part of China, India, Tanzania, and Brazil are absorbed into malarious areas.

In Table 4, we summarize the populations of malarial areas estimated by this model. The current population in malarial areas (2.3 billions) is estimated to increase to 2.5 ~ 2.8 billions after global warming. In these estimations, we assumed the same population distribution as present.

7. Will global warming greatly affect humanity through malaria?

Although malaria was eradicated in many areas in the 1950's and 60's, it was not possible to go on at the same pace after the 1970's, and now it is almost in stable equilibrium. However, malaria has occurred in some areas because of the migration of refugees due to lack of civil rest. As a result of the failure of eradication efforts, and the appearance of new distribution patterns of *Anopheles* caused by agricultural development and deforestation, the incidence of malaria has increased in some areas. For example, although the number of victims in the Amazon at 1970 was 5100, it expanded to 1 million by 1990 due to deforestation and the influx of immigrants with no immunity. Thus, the present distribution and occurrence of malaria and the eradication efforts are hardly in balance. In this situation, as shown in Table 4, the potential areas of incidence will increase by 10 ~ 30% due to global warming of a doubled CO_2 level. Furthermore, considering that this will happen in fringelands where the inhabitants have low immunity and that the disease will be brought by chemically tolerant parasites and vectors, the health of these people will be seriously affected.

The global burden of disease caused by malaria (shortened life spans due to disease and accidents after socio-economic adjustment) accounted for 2.6% of the total burden of all diseases in 1990 (World Bank, 1993). This ratio is greater than that of AIDS (1.4%) and traffic accidents (2.2%), and the increase caused by global warming accounts for a further 0.5%.

Measures to counter malaria have required a great number of people and expenses. Costs for studies, research and agricultural chemicals are estimated to be 0.2 ~ 0.4US$ per person, while annual medical expenses per person in countries with low incomes are estimated to be US$ 2 ~ 40 (World Bank, 1993). Although malaria infected areas have heavy expenses because of the disease, this is not large in a global context. Thus, we consider that it is possible to lighten or avoid those impacts on human health with appropriate policies and prudent foresighted planning.

References

Argonne National Laboratory (1994): *Guidance for Vulnerability and Adaptation Assessments*, U. S. Country Studies Program, U. S. Country Studies Management Team (PO-63), Washington D. C., USA.

Bolin, B, (1994): Next step for climate-change analysis, *Nature*, Vol. 368, p. 94.

Bouwman, A. F., I. Fung, E. Matthews and J. John (1993): Global analysis of the potential for N_2O production in natural soils, *Global Biogeochemical Cycles*, Vol. 7(3), pp. 557–597.

Bradley, D. J. (1993): Human tropical diseases in a changing environment; Lake, J. V., G. R. Bock and K. Ackrill (eds.): *Environmental Change and Human Health*, Ciba Foundation Symposium 175, Chichester, pp. 146–170.

Cline, W. (1992): *The Economics of Global Warming*, Institute for International Economics, Washington D. C.

China Advisory Committee on Parasitic Diseases (1989): Malaria situation in China, *Chinese J. Parasitol. Parasitic Diseases*, Vol. 7, pp. 241–244.

Dietz, K., L. Molineaux and A. Thomas (1984): A malaria model tested in the African savannah, *Bull. WHO*, Vol. 50, pp. 347–357.

Dutta, H. M. and A. K. Dutt (1978): Malarial ecology: A global perspective, *Soc. Sci. & Med.*, Vol. 12, pp. 69–84.

Fankhauser, S. (1992): *Global Warming Damage Costs: Some Monetary Estimates*, CSERGE GEC Working Paper 92-29, University College London and University of East Anglia.

Fine, P. E. M., M. M. Milbey and W. C. Reeves (1979): A general simulation model for genetic control of mosquito species that fluctuate markedly in population size, *J. Med. Entomol.*, Vol. 16, pp. 189–199.

Garrett-Jones, C. and G. R. Shidrawi (1969): Malaria vectorial capacity of a population of *Anopheles gambiae*, *Bull. WHO*, pp. 531–545.

Haile, D. G. and D. W. Weidhaas (1977): Computer simulation of mosquito populations (*Anopheles albimanus*) for comparing the effectiveness of control techniques, *J. Med. Entomol.*, Vol. 13, pp. 553–567.

Haile, D. G. (1989): *Computer Simulation of the Effects of Changes in Weather Patterns of Vector-Borne Disease Transmission*, Report to United States Environmental Protection Agency, Project No. DW12932662-01-1., Washington D. C.

Hohmeyer, O. and M. Gartner (1992): *The Cost of Climate Change*, Report to the Commission of the European Communities Directorate General XII, July 1992.

Houghton, J. T., G. J. Jenkins and J. J. Ephraums (1990): *Climate Change*, Cambridge University Press, Cambridge, U. K.

Kalkstein, L. S. (1989): *The Impact of CO_2 and Trace Gas-Induced Climate Changes upon Human Mortality*, Report to U. S. EPA, Contract No. CR81430101, Washington D. C.

K. C. S. (1991): *Survey on Vector-Borne Diseases by Global Warming*, Report to Japan Environment Agency, Tokyo.

Legates, D. R. and C. J. Willmott (1989): *Monthly Average Surface Air Temperature and Precipitation*, NCAR, Boulder, CO.

Maywald, G. F. and R. W. Sutherst (1990): *User's Guide to CLIMEX*, CSIRO Division of Entomology Report, No. 35., CSIRO.

MacDonald, G. (1952): The analysis of sporozoite rate, *Tropical Disease Bulletin*, Vol. 49(6), pp. 569–586.

MacDonald, G. (1952): The analysis of equilibrium in malaria, *Tropical Disease Bulletin*, Vol. 49(9), pp. 813–829.

MacDonald, G. (1957): *The Epidemiology and Control of Malaria*, Oxford Univ. Press.

Molineaux, L. H. (1990): The epidemiology of human malaria as an explanation of its distribution, including some implications for its control; Wernsdorfer, W. H. and I. A. McGregor (eds.): *Malaria*, pp. 913–998, Churchill Livingstone.

Najera, J. A., B. H. Liese and J. Hammer (1992): *Malaria: New Patterns and Perspectives*, World Bank Technical Paper No. 183.

Nicholls, N. (1993): El Niño-Southern Oscillation and vector-borne disease, *The Lancet*, Vol. 342, pp. 1284–1285.

Nordhaus, W. D. (1991): To slow or not to slow: The economics of greenhouse effect, *Economic Journal*, Vol. 101(407), pp. 920–937.

Rastor, J. and W. M. Post (1984): Calibrating Thornthwaite and Mather's actual evapotranspiration using an approximating function, *Can. J. For. Res.*, Vol. 14, pp. 466–467.

Rogers, D. J. (1993): Vector-borne diseases, models, and global change, *The Lancet*, Vol. 342, pp. 1282–1284.

Rosenzweig, C. and M. L. Parry (1994): Potential impact of climate change on world food supply, *Nature*, Vol. 367, pp. 133–138.

Ross, R. (1911): *The Prevention of Malaria*, Murray, London.

Sugawara, M. (1972): *Water Runoff*, Kyoritsu Syuppan, Tokyo.

Sutherst, R. W. (1993): Arthropods as disease vectors in a changing environment; Lake, J. V., G. R. Bock and K. Ackrill (eds.): *Environmental Change and Human Health*, Ciba Foundation Symposium 175, Chichester, pp. 124–145.

Sutherst, R. W. and G. F. Maywald (1985): A computerized system for matching climates in ecology, *Agriculture, Ecosystems and Environment*, Vol. 13, pp. 281–299.

Titus, J. (1992): The cost of climate change to the United States, *Global Climate Change: Implications, Challenges and Mitigation Measures*, Pennsylvania Academy of Science, Pennsylvania.

Webb, R. S. and C. E. Rosenzweig (1993): Specifying land surface characteristics in general circulation climate models: soil profile data set and derived water-holding capacities, *Global Biogeochemical Cycles*, Vol. 7(1), pp. 97–108.

White, G. B. (1982): Malaria vector ecology and genetics, *British Medical Bulletin*, Vol. 38(2), pp. 207–212.

White, G. B. (1989): Malaria; Vector Biology and Control Division, World (ed.): *Geographical Distribution of Arthropod-Borne Diseases and Their Principal Vectors*, Health Organization, WHO/VBC/89.967, pp. 7–22.

White, M. R. and I. Hertz-Picciotto (1986): Human health: Analysis of climate related health; White, M. R. (ed.): *Characterization of Information Requirements for Studies of CO_2 Effects: Water Resources, Agriculture, Fisheries, Forests and Human Health*, pp. 172–206, DOE/ER-0236, United States Department of Energy.

WHO (1990): *Potential health effects of climatic change*, Report of a WHO Task Group, WHO/PEP/90/10.

WHO (1992): *Our Planet, Our Health*, Report of the WHO Commission on Health and the Environment, Geneva.

Wilson, M. F. and A. Henderson-Sellers (1985): A global archive of land cover and soils data for use in general circulation climate models, *J. Climatology*, Vol. 5, pp. 119–143.

World Bank (1993): *World Development Report 1993*, Oxford Univ. Press.

Vorosmarty, C. J. *et al.* (1989): Continental scale models of water balance and fluvial transport: An application to South America, *Global Biogeochemical Cycles*, Vol. 3(3), pp. 241–265.

Zuou, Z. J. (1981): The malaria situation in the People's Republic of China, *Bull. WHO*, Vol. 59(6), pp. 931–936.

(Received September 1, 1994; revised version accepted March 2, 1995)