Economic Epidemiology of Malaria [1]

by
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Abstract

Malaria is an extremely malicious disease which is prevalent among the poor in the poorer countries, 80 percent of the disease and its fatality being concentrated in African countries. It attacks an estimated 300 million people every year, killing about 2 millions of them. Fifty percent of the high infant deaths in developing countries is directly due to malaria, and the number of deaths are even higher when account is taken of the other causes of death that are inflicted by malaria. This paper models the equilibrium dynamics of the disease and aggregate income, incorporating the utility maximizing private human behaviors regarding malaria related health care investment. The models in epidemiology literature of malaria do not incorporate individual human behaviors that are guided by economic incentives. Thus these models prescribe different types of public policies to control the disease. The paper contrasts how the economic and biological epidemiology models differ in terms of their predictions of the short-run and long-run behavior of the disease, and its effect on economic growth. The paper also studies the effects of various public policies.

JEL Classifications: I120, O150

Keywords: Malaria, disease dynamics and economic development.

1 Introduction

Malaria is prevalent among the poor in poorer countries, especially in tropical countries, with 80 percent of the disease incidence and fatality being concentrated in African countries. It affects an estimated 300 million people every year, resulting in about 2 million deaths. Fifty percent of the high infant death rate in developing countries is directly due to malaria, and the number of deaths is even higher when indirect causes of death related to malaria are taken into account. Children are more susceptible to malaria and many die from it without proper treatment. Survivors acquire partial immunity, however, at a cost of higher morbidity and other health problems such as severe anemia and spleen diseases. Hence, malaria affects the productivity and income of surviving workers who have had the disease. In addition, being infectious, the disease creates negative external effects on other individuals. As studies by Barlow [1968], Conly [1975], Kaewsonthi [1989] and Newman

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[1968] show, there is some evidence of non trivial private costs due to lost earnings and preventive care, of social costs due to new infections, and overall negative effect on aggregate income. Additional literature can be found in the literature surveys by Hammer[1993] and Gomes[1993]. Recent empirical studies by Gallup and Sachs [1998a, 1998b] and by McCarthy,Wolf and Wu [2000] find substantial growth costs of malaria, especially in the African countries.

In this paper, I model the dynamics of the disease and its effect on aggregate income growth, incorporating utility maximizing private human behavior under a variety of public policy environments. An important part of this exercise is to identify a set of important parameters that describe the behavior of Plasmodium parasites, their vector carriers, and the behavior of victimized human hosts in order to better understand the disease dynamics and analyze the effect of disease control policies. My model differs from models of malaria in epidemiology literature, by incorporating investment in malaria related health care that are guided by economic incentives. I follow the human capital approach to investment in health care. I also contrast the predictions of this model with the predictions of the purely epidemiological models. Two groups of models differ in terms of their predictions of the short-run and long-run behavior of the disease, its effect on economic growth, welfare losses, and implications for public policies - for example, the extent to which public control programs could be privatized through tax-subsidy policies or community level incentives, and the role of public education regarding the effect of malaria on productivity losses.

In section 2, I briefly sketch the biological process of malaria transmission. In section 3, I formulate a utility maximization model of malaria preventive care choices under the assumption that all individuals have complete knowledge about the effects of malaria on productivity, and then study the nature of disease dynamics. In section 4, I study the demand for preventive care and its effect on disease dynamics when some people are ignorant about the productivity effects of malaria. In section 5, I integrate the disease dynamics with an income growth model. Section 6 concludes the paper with policy suggestions.

2 Biology of malaria parasites and health effects on human hosts

Malaria disease results from biological developments of micro-organisms known as protozoal parasites after they are infused in human blood by mosquitoes during their blood meal. The parasite varies by type and in deadliness. Most notable are P. falciparum, P.vivax, P.ovale, and P.malariae – with the first type being the deadliest and most prevalent in Africa,

and the last one being prevalent in most other tropical countries of Asia and South America.

The parasite's life cycle is split between a human host [2] and an Anopheline mosquito vector. Only 60 out of the 380 species of Anopheline mosquito can transmit malaria. Only the female mosquitos need blood meal from humans during hatching eggs, and thus involved in spreading of malaria.

During blood meal, the mosquito must inject anticoagulant saliva for an even flowing of blood meal. During that time, sporozoites from mosquito salivary gland are injected in to human blood. After fighting and camouflaging the immune system of partially immuned humans, the survived sporozoites arrive in the liver within 45 minutes of the mosquito bite, and penetrate hepatocytes, where they remain for 9-16 days. Through asexual reproduction or cloning the plasmodium multiply within the cells. While P.falciparum and P.malariae sporozoites trigger immediate schizogony, P.ovale and P.vivax sporozoites may either trigger immediate schizogony or have a delayed trigger, resulting in dormant hypnozoites. Some strains, such as the North Korean strain, seem to consist of sporozoites with universally delayed triggers, so they all form long lasting hypnozoites. P.vivax may have an incubation period of up to 10 months. Gametocytes produced in the primary attack seem to contain all the genetic information required to create sporozoites of several different activation times. The same seems true for gametocytes produced in relapses where the hypnozoites become activated. The immune system may produce antibodies to the gametocytes at this stage. Upon release, they penetrate red blood cells, and produce either merozoites or micro and macrogametocyte. Merozites, after some time lapse can transform into micro and macrogametocyte. See figure 1.

When a mosquito feeds on the blood, it may intake these gametocytes into its gut, where through random matching with the microgametocytes, the macrogametocytes are fertilized. After fertilization, the resulting ookinete enters the wall of a cell in the midgut of the mosquito after 18 to 24 hours, and there it transforms into an oocyst and then blasts. Many sporozoites are born within the oocyst. After the oocyst is raptured, the new sporozoites, then migrate to the salivary gland, where they lie up to 59 days, mature and turn into 1000 times more infective than when in the oocyst. In the gland the sporozoites wait for injection into another human host. The sporozoites are single cell micro organism of about 12μm long and 1μm across, with a single nucleus. One bite of a mosquito transfers only about 10% of its sporozoite load into the human blood stream. Plasmodium parasites seem capable of adapting to any suitable anopheline mosquito, given sufficient time and contact.

[2] An exception is the parasite, P.malarie, which may affect other higher primates.

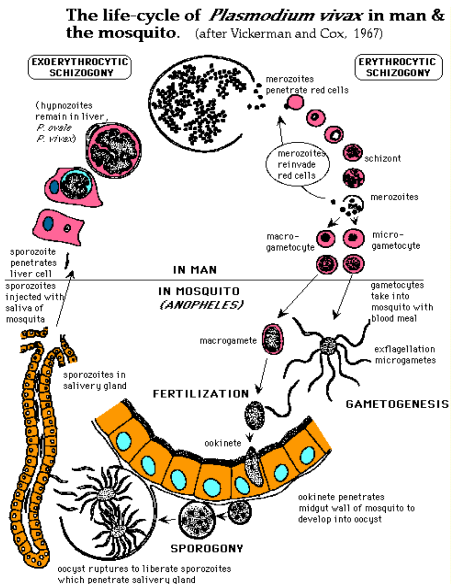

Figure 1: Life cycle of Plasmodium microorganisms

2.1 Symptoms and Health Complications:

As the shizonts mature in the liver, huamn host gets high fever (maybe above 41oC), shivering, pain in the joints, headache, repeated vomiting, convulsions and coma. In severe cases of P. falciparum infection, the patient will have fits, coma and may lead to death. Other health complications include cerebral malaria (unrousable coma), generalized convulsions, normocytic anaemia, renal failure, fluid, electrolyte and acid-base disturbances, pulmonary oedema, circulatory collapse, shock, disseminated intravascular coagulation, hyperpyrexia, hyperparasitaemia, and malarial haemoglobulinurea. These features may occur singly or in combinations. In kids, malaria may cause hypoglycaemia.

Malaria during pregnancy leads to very severe health consequences for both the mother and the child. For instance, it may lead to severe anemia and then to hemorrhage and ultimately to death, or to low birth weight and premature delivery. During pregnancy, a woman acquires reduced immunity against malaria and treatment for acute malaria is more complicated during pregnancy.

3 Basic Economic framework

3.1 Households

The incidence and the health effects of malaria infection depend on the age and nutritional level of an individual, type of health care investments provided by parents when children are very young, and also if an individual was infected by malaria before since infection before may give partial immunity in later stage of life. It is, however, analytically intractable to incorporate these factors into an economic model of malaria. I will first consider a simple model abstracting from many of these features to get analytical insights and then later I will consider extensions incorporating these features for the purpose of policy simulation exercises.

Consider an economy in which people live a certain finite number of periods $\bar{T}$, and they differ only in their schooling level s, which together with their health status determine their earnings in the labor market. Assume continuum of schooling levels and for simplicity assume that schooling level is normalized to be in the unit interval $[0,\, 1]$. Let $f\,(s)$ be the probability density function of the schooling distribution in the population in each period. In each period, he might be infected with malaria from which he may survive with some probability but he loses some of his human capital and work time. I now make a very strong simplifying assumption that even though he loses some human capital during the period he is infected, at the end of the period he reenergizes himself to be identical to any other person of the same schooling level and a sound health status. To keep the demographics of the population at a manageable level, I also assume that whenever a person dies, either naturally, or from malaria, the same number of new borns are added to the population, and to each living person during any period, n new borns are added to the population. Thus in the economy the population growth rate is n per period.[3] Denote the total population at time t by L_t.

Let p be the probability of no-malaria in period t. p depends on the amount of preventive care expenditure, x_t^p, and also on the prevalence rate of the disease in the previous period, i_{t-1} which is the fraction of population infected with malaria in period $t-1$. Denote this dependence by $p\,(x_t^p; i_{t-1})$. The probability of no-malaria, $p\,()$ is increasing in x_t^p and decreasing in i_{t-1}. Furthermore, $p\,()$ depends on climate, extent of population migration, and government policies regarding sewage management and DDT spraying, all these determine the size of the mosquito population. The probability of no-malaria also depends on

[3] I make these assumptions to make the analysis teachnically tractable. These are same as assuming that agents are myopic. They make their dicisions in each period and their health capital depeletes during the period the individual is infected, and at the end of the period he is reenergized.

population genetics of malaria pathogens, and the composition of drug resistant strains in that population which in turn depends on the extent of selection by drug pressure. In this paper I assume that other than the population of malaria parasites of the previous period as represented by the infection rate in the previous period i_{t-1} and the choice of the individual preventative care against malaria x_t^p, all other factors are parameters of the function $p(.)$.

Once infected with malaria, his survival probability q and the time to recover from malaria T depend on the amount of curative care expenditure, x_t^c. I denote an agent by α which is in general a vector with components representing his nutrition level, parental income and schooling level. In this paper, however, I restrict the heterogeneity of agents only to schooling level s, and denote an agent by $\alpha = s$. An individual in each period can be in either of two health states (health state variable is denoted by ω): have malaria, denoted as $\omega = M$ or do not have malaria, denoted as $\omega = N$. His human capital in period t is a combination of his health status ω in that period and his schooling level s, It is denoted by $h_t(s, \omega)$. Consistent with the medical facts on the effects of malaria on health such as severe anemia and other diseases and higher morbidities and so on, I assume that $h_t(s, M) < h_t(s, N)$. For a survey of how health status affect earnings see Strauss and Thomas [1998]. For now I assume that all individuals know perfectly well this relationship. This is, however, a serious assumption and in the next section I explore the implications on demand for preventive care and the disease dynamics when some people are ignorant about this relationship, i.e., some individuals have *incomplete information* about the relationship.

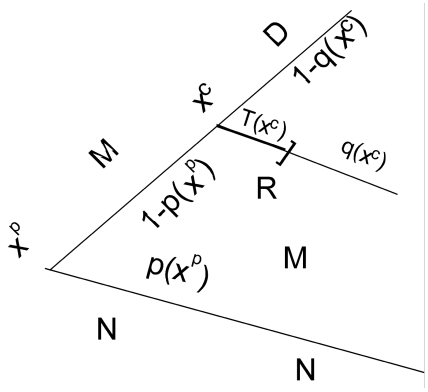

Figure 2: Decision Tree

Assume for simplicity that there is a fixed preventive care malaria package which costs $\bar{x}_t^p$. While preventive care may only reduce the probability of malaria significantly, for simplicity of exposition, I assume that investment in preventive care $\bar{x}_t^p$ can completely

eliminate the chances of malaria, i.e.,

$$p\left(x_t^p, i_{t-1}\right) = \begin{cases} 1 & \text{if } x_t^p \geq \bar{x}_t^p \\ 1 - p\left(0, i_{t-1}\right) & \text{otherwise} \end{cases}, \quad 0 < p\left(0, i_{t-1}\right) \leq 1, \text{ if } i_{t-1} > 0$$

In each period he makes a sequential decisions in two stages as follows: At the beginning of period t, given the prevalence rate of malaria infection in the previous period i_{t-1}, the agent $\alpha = s$ makes his first stage decision whether to invest in preventive care or not. After this decision is made, the uncertainty about malaria infection is resolved. In the second stage, if he is infected with malaria, i.e., $\omega = M$, he decides how much to invest in curative care, x_t^c. The survival probability q, the duration of his infection, T and the likelihood of creating drug resistant malaria pathogens depend on this decision. In this paper, I assume these to be fixed parameters. For notational convenience, view T and x_t^c to be random variables both of which assume their original values in the event of malaria infection and both of which assume value 0 in the event of no malaria.

For notational convenience, assume that for the agent under consideration time t is same as his age. This is a harmless assumption given our previous assumption of reenergization of health stock in each period. At any age t the agent may die from malaria infection with probability π_t. It is easy to see that

$$\pi_t = \left(1 - p\left(x_t^p, i_{t-1}\right)\right) \cdot (1 - q)$$

Let us denote his survival probability up to age t by $\bar{F}_t \equiv \sum_{\tau > t} \pi_\tau$. Let $c_t(\omega)$ be his consumption in period t in the event ω. If he dies from malaria, his utility level is zero. His von Neumann-Morganstern one period utility function is given by

$$u(c) = \frac{c^{1-\rho}}{1-\rho}, \rho \neq 1$$

Given his preventive care plan $\{x_t^p\}$, and the malaria infection rates $\{i_{t-1}\}$, his von Neumann-Morganstern expected utility function is defined by

$$E\left[U \middle| \{x_t^p\}, \{i_{t-1}\}\right] = \sum_{t=0}^{\bar{T}} \bar{F}_t \beta^t E\left[u\left(c_t(\omega)\right) \middle| x_t^p, i_{t-1}\right] \tag{1}$$

where,

$$E\left[u\left(c_t(\omega)\right) \middle| x_t^p, i_{t-1}\right] = \left(1 - p\left(x_t^p; i_{t-1}\right)\right) \cdot q \cdot u\left(c_t(M)\right) + p\left(x_t^p; i_{t-1}\right) \cdot u\left(c_t(N)\right) \tag{2}$$

The agent's decision can be denoted by a binary variable θ, taking value 1 if he decides to invest the amount $\bar{x}_t^p$ in the preventive care package, and taking value 0 otherwise.

He has one unit of labor time to supply to the market. Thus he earns $(1 - T(\omega)) h_t(s, \omega) w_t$, where w_t is the wage rate of a unit of labor in efficiency unit in period t. The budget constraint of the agent $\alpha = s$ in period t is then given by,

$$c_t(s, \omega|\theta) = (1 - T(\omega)) h_t(s, \omega) \cdot w_t - \theta \cdot x_t^p - x_t^c(\omega). \tag{3}$$

3.2 Optimal solution

Given our assumption about the health reenergization at the end of each period, the computation of demand for preventive care in period t is equivalent to solving the following one period utility maximization problem: Given the prevalence rate of malaria i_{t-1},the agent $\alpha = s$ chooses $\theta = 1$ if and only if

$$u(c_t(s, N|\theta = 1)) \geq p(0, i_{t-1}) \cdot u(c_t(s, N|\theta = 0)) + (1 - p(0, i_{t-1})) \cdot q \cdot u(c_t(s, M|\theta = 0)) \tag{4}$$

Given our assumption on utility function, the above choice of preventative care decision reduces to $\theta = 1$ if and only if

$$\left[1 - \frac{\bar{x}_t^p}{h_t(s, N) w_t}\right]^{1-\rho} \geq p(0, i_{t-1}) + (1 - p(0, i_{t-1})) \cdot q \cdot \left[(1 - T) \cdot \frac{h_t(s, M)}{h_t(s, N)}\right]^{1-\rho} \tag{5}$$

Equation (5) says that an agent $\alpha = s$ will invest in preventive care (i.e., $\theta = 1$) if the malaria prevalence rate i_{t-1} is high, or income loss due to sickness either because of time loss, i.e., T, is high, or loss in human capital due to malaria is high, i.e., the ratio, $\frac{h_t(s,M)}{h_t(s,N)}$, is low, and the cost of preventive care x_t^p as a percentage of potential full income without malaria, $h_t(s, N) w_t$, is low. Sometimes I will denote the optimal solution for agent $\alpha = s$ given the malaria prevalence rate i_{t-1} by $\theta(s, i_{t-1})$.

Given the malaria prevalence rate i_{t-1}, in the beginning of period t, **the willingness-to-pay for malaria preventive care** for agent $\alpha = s$ is that x_t^p which equates both sides of (5). Assume that $\frac{h_t(s,M)}{h_t(s,N)} = (1 - \eta)$, $0 < \eta < 1$, for all $s \in [0, 1]$. Although we expect higher variability, i.e., higher value of η for higher schooling levels, but it is convenient to assume it to be constant for technical tractability. Notice that treating (5) as an equality, we can solve for i implicitly as a function of schooling level s, which we denote by $i(s)$. It can be shown easily that $i'(s) < 0$, i.e. $i()$ is a decreasing function. This curve is shown in figure 3, under the label "with complete information". It is clear from equation (5) that given i_{t-1}, there exists a $\sigma^*(i_{t-1})$ such that the right hand side of equation (5) is satisfied for all $s \in [\sigma^*(i_{t-1}), 1]$. This curve is useful since it tells us in any period, given

the prevalence rate of malaria i_{t-1}, the population that will invest in preventive care consist of those whose education level exceeds $\sigma^*(i_{t-1})$. It is now easy to calculate the size of the population susceptible to malaria per unit of total population as

$$\begin{aligned}\left(1-\overline{\theta}\left(i_{t-1}\right)\right) &= 1-\int\theta(s)\,f(s)\,ds=\int_0^{\sigma^*(i_{t-1})}f(s)ds \\ &= \sigma^*(i_{t-1}) \text{ under the assumption that } f(.) \text{ is uniformly distributed.}\end{aligned}$$

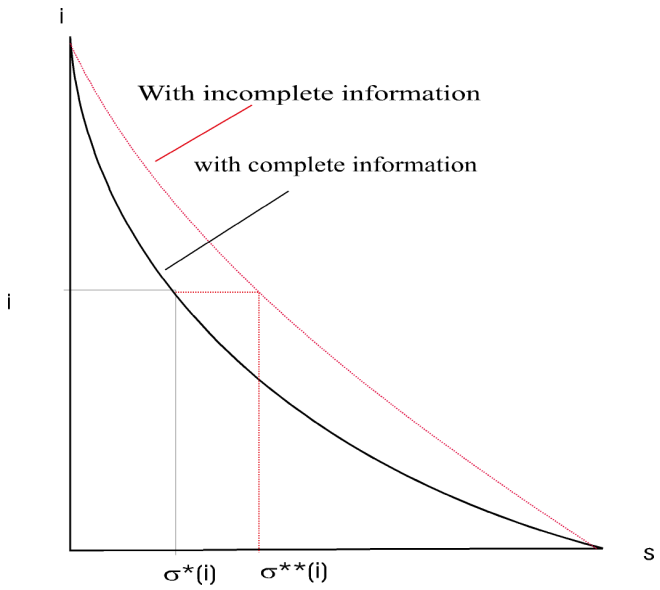

Figure 3: Implicit solution of Equation (5)

For an example, assume Mincer earnings function for a healthy individual of schooling level s, i.e.,

$$w_t(s,N)\equiv h_t(s,N)\,w_t=w_te^{\mu s}$$

where μ is the rate of returns from s years of schooling. Assume also that $x_t^p/w_t=x^p$ for all t. Equation (5) then simplifies to

$$s\geq\frac{\ln\left(x^p/\left(1-\xi\left(i_{t-1}\right)\right)\right)}{\mu}\equiv\sigma^*\left(i_{t-1}\right)$$

where $\xi(i_{t-1})=\left[p(0,i_{t-1})+(1-p(0,i_{t-1}))\left(q\cdot(1-T)^{1-\rho}\cdot(1-\eta)^{1-\rho}\right)\right]^{1/(1-\rho)}$. Note that $\sigma^*()$ is a decreasing function as expected.

3.3 Disease dynamics

The susceptible population in period $t+1$ is given by $\left(1-\overline{\theta}(s,i_t)\right)L_{t+1}$. To formulate the dynamics in discrete time, I assume that at the beginning of a period, female Anopheline

mosquitoes bite humans for blood meals. A susceptible human gets malaria parasites (P. falciparum sporozoites) from an infected mosquito's bites. The **asexual reproduction** part of the life-cycle of P. falciparum microorganisms, beginning with evasion of human immune system to penetrate into liver cells of human host for cloning, to the looped circulation of merozoites and then to the final stage of producing micro and macro gametocytes (i.e., gametes for sexual reproduction) that are ready to be passed to mosquito during blood meal, all these events in the parasite's life cycle occur during the first part of the period. This is the infection period, which lasts for T periods, $0 < T < 1$. During this infection period, the Anopheline female mosquitoes take blood meals. The **sexual reproduction** part of P. falciparum microorganisms, starting from fertilization of gametocytes by random mating of micro and macro gametocytes to form ookinetes, to the last stage of penetration of new vicious sporozoites into the saliva gland of the host mosquito, all these events occur during the rest of period t. Even though I abstract from many realistic features of plasmodium's life-cycle, and its relationship to human and vector hosts, the model retains most important elements of that process to carry out the economic and policy analysis. For details, see the life-cycle of the plasmodium in figure 1.

The expected number of malaria infection, i_{t+1},which is also the malaria prevalence rate in period $t+1$ is then given by

$$i_{t+1} = \sigma^* (i_t) \cdot (1 - p(0, i_t)). \tag{6}$$

It is convenient to express the above in the following form:

$$(1 - p(0, i_t)) = (1 + \rho(i_t)) i_t \tag{7}$$

The shape of the function $\rho(i)$ is presumably as shown in figure 4. This is a reasonable assumption on the shape of $\rho(i)$ when individuals do not use any preventive care, since when infection rate is very low, growth rate of infection is also low. When the disease incidence rate is already very high, say close to 1, there is little room for i to grow and hence the growth rate is very low. At intermediate levels, i can grow at higher rates.

Under the above assumption about the shape of $\rho(i)$, it is clear that there are two locally stable steady-state equilibria and another unstable steady-state equilibria. The locally stable equilibrium i^* with strictly positive rate of malaria infection is said to be **malaria endemic equilibrium,** and the other locally stable equilibrium at zero level of infection rate is said to be a **malaria free equilibrium**. The unstable equilibrium is between these two equilibrium level of disease infection rates and it is labeled in figure 4 as i^c. This unstable equilibrium

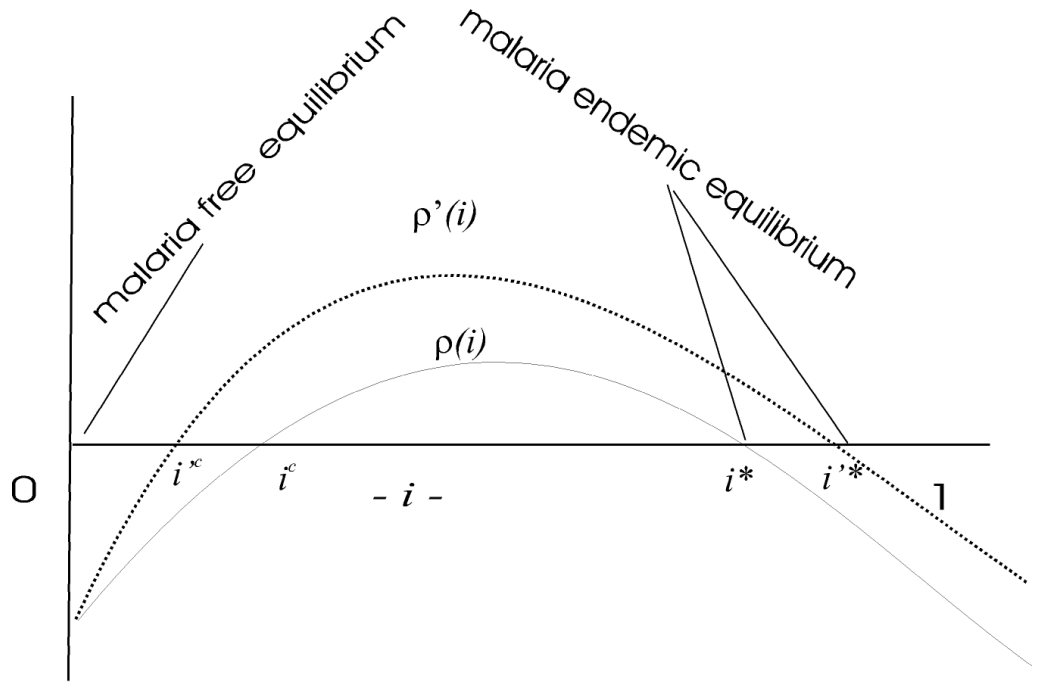

Figure 4: Transmission rate $\rho(i)$

rate determines a critical level of infection rate: If an economy has its infection rate below this critical level, eventually the economy will converge to the disease free equilibrium without any public intervention, and if it is above this critical level but below i^*, the rate of infection will rise over time until it settles down to the rate i^*; if the infection rate is above i^* then the disease rate will decrease over time until it converges to the infection rate i^*.

Notice that the growth curve $\rho(i)$ depends on two important parameters – one is $p(0,i)$, which depends on climates and government policies towards sewage and DDT spraying, and the other one is $\sigma^*(i)$ which is determined by individual incentives to invest in malaria preventive care, which in turn also depends on $p(0,i)$ and the knowledge about the productivity effects of malaria infection.

Notice that a pure epidemiological model would postulate $i_{t+1} = \sigma \cdot (1 - p(0, i_t))$, where σ is the fraction of population susceptible to malaria in period t, and would predict the dynamics of the disease quite differently than an economic epidemiology model in which individual/private economic decisions make σ a decreasing function of i_t.

4 Malaria Education, Demand for Preventive Care and the Disease Dynamics

Malaria causes serious anemia and other health complications which can permanently lower one's productivity and leads to higher morbidity. A very few knows about these effects. In this section I will study how the demand for preventive care is affected due to this incomplete information. What is the economic value of educating people about malaria? What happens to dynamics when a government spends some resources to educate people about these permanent side effects of malaria on productivity loss and morbidity?

Suppose that only a small fraction $n_1$of the population with higher education knows about the permanent effect of malaria on productivity loss and the rest of the population do not know. Under the assumption of uniform distribution $f()$,we have that all individual's of education level $s \geq n_1$knows about the relationship and the rest of the population is ignorant. What happens to the disease dynamics and long-run equilibrium?

Notice that for the ignorant people, $\frac{h_t(s,M)}{h_t(s,N)} = 1$, which is greater than $1 - \eta$ in the right hand side of equation (5). Then it follows that the $i(s)$ curve for the population with incomplete information will to the right of the one with complete information as shown in figure 3 under the label "with incomplete information". This means that given a rate of disease incidence, i_{t-1}, the set of population who invest on malaria preventive care will be lower under incomplete information than with complete information. This is shown in figure 3 by $\sigma^{**}(i)$. It then follows that the growth curve under incomplete information denoted by $\rho'(i)$ is to be shifted upward, leading to higher equilibrium rate of malaria endemic disease rate denoted i'^{*} and lower level of the critical level denoted by i'^{c} . Such ignorance could be self-fulfilling in equilibrium.

If government spends some resources to educating or making people to realize the effect of malaria, there will be a substantial gains from lower disease incidence and also prevalence rate of malaria will be lower in the steady-state. An interesting policy implication of our analysis in this section is that if the malaria incidence rate with the fraction of ignorant people is between the two critical levels, i'^{c} and i^{c}, then such an economy can eradicate malaria purely by public education about the true productivity effects of malaria infection.

5 Malaria and Growth

In this section I will introduce the interaction between disease dynamics and growth in human capital and study how the dynamics of disease constrains the growth path of the

economy. The general case with heterogenous schooling group is analytically intractable for this purpose, so I follow the standard practice of assuming a representative agent model in which population consists of one schooling level s for each country. The countries may, however, vary in terms of schooling level. I further assume that people live for two periods. In period 1 he is young and make all his decisions and in period 2 he is old and lives off his accumulated capital. Let us denote by $c_t^1(\omega)$, $c_{t+1}^2(\omega)$, and $A_t(\omega)$ respectively his consumption in the first and second period of life and his savings in the event ω. If he dies from malaria, his utility level is zero. If he survives, his utility level is given by

$$U\left(c_t^1, c_{t+1}^2\right) = \frac{\left[\left(c_t^1\right)^{\alpha} \cdot \left(c_{t+1}^2\right)^{1-\alpha}\right]^{1-\rho}}{1-\rho}, 0 < \alpha < 1, \rho \neq 1 \tag{8}$$

The expected utility is then given by

$$EU = (1 - p(x_t^p; i_{t-1})) \cdot q \cdot \left[U\left(c_t^1(M), c_{t+1}^2(M)\right)\right] + p(x_t^p; i_{t-1}) \cdot \left[U\left(c_t^1(N), c_{t+1}^2(N)\right)\right] \tag{9}$$

Given his malaria preventive care investment decision θ, let us denote the earnings of agent $\alpha = s$ in period t by

$$y_t(s, \omega|\theta) = (1 - T(\omega)) h_t(s, \omega) \cdot w_t - \theta \cdot x_t^p - x_t^c(\omega). \tag{10}$$

Thus his choice problem reduces to choosing θ to maximize his expected utility in equation. (9) subject to the following budget constraints:

$$\begin{aligned} c_t^1(\omega) &= y_t(s, \omega|\theta) - A_t(\omega) \\ c_t^2(\omega) &= (1 + r_t) A_t(\omega) \end{aligned} \tag{11}$$

Applying the dynamic programming technique to solve the above problem sequentially, it is easy to note that the optimal savings decision at the last decision stage is given by

$$A_t(s; \omega|\theta) = (1 - \alpha) \cdot y_t(s, \omega|\theta) \tag{12}$$

Substituting the above in the expected utility function, our problem for agent $\alpha = s$ reduces to the following standard one period expected utility maximization problem:

$$\max_{\theta \in \{0,1\}} \tilde{u}(y_t(s, \omega|\theta))$$

where,

$$\tilde{u}(c) = K \cdot \frac{c^{1-\rho}}{1-\rho}, \rho \neq 1, K \text{ is a constant.} \tag{13}$$

This is the same problem that we solved earlier in equation (5). The average human capital of the whole labor force is then given by

$$\begin{aligned} \bar{h}_t(i_t; s) &= [(1-T)(1-\eta) i_t + (1-i_t)] h_t(s, N) && (14) \\ &= \xi(i_t) \cdot h_t(s, N) && (15) \end{aligned}$$

where, $\xi(i_t) = 1 - (T + \eta - \eta T) i_t$.

We assume that a worker of schooling level s with good health from no-malaria infection adds to social knowledge stock at the rate of $\varphi(s)$ per period, and a worker of schooling s with poor health from malaria infection contributes at the rate of $(1-\gamma)\varphi(s)$ per period. The total addition to useful public knowledge during period t which depends on the malaria prevalence rate i_t is given by,

$$\gamma(i_t; s) = [1 - (T + \gamma - \gamma T) i_t] \varphi(s). \tag{16}$$

We assume that $h_t(s, N)$ grows over time as follows:

$$h_{t+1}(s, N) = (1 + \gamma(i_t; s)) h_t(s, N), \ t \geq 0 \tag{17}$$

The aggregate labor in efficiency unit, i.e., effective labor, $\tilde{L}_t$ in period t is given by $\tilde{L}_t = \bar{h}_t \cdot L_t$. Assume that capital depreciate in one period. Then, we have

$$K_{t+1} = L_t \sum_{\omega = M,N} A_t(\omega) \tag{18}$$

Denoting the capital labor ratio in efficiency unit in period t by $\tilde{k}_t = K_{t+1}/\tilde{L}_t$, after a few algebraic manipulations, one can show the following difference equation for $\tilde{k}_t$,

$$\begin{aligned} \tilde{k}_{t+1} &= \begin{cases} \frac{(1-\alpha)}{(1+n)(1+\gamma(i_t;s))} (1 - [x^c + \eta + T - \eta T] i_t) \omega\left(\tilde{k}_t\right) & \text{if } i_t < \bar{\imath}(s) \\ \frac{(1-\alpha)}{(1+n)(1+\gamma(i_t;s))} (1 - x^p) \omega\left(\tilde{k}_t\right) & \text{if } i_t \geq \bar{\imath}(s) \end{cases} \\ &\equiv \Psi\left(\tilde{k}_t, i_t\right) \text{ say} && (19) \end{aligned}$$

The system of equations (6) and (19) determines the dynamics of the economy. Notice that if i_t is constant over time, the above is the difference equation of a standard growth model.

5.1 The steady-state economic and disease equilibrium

The system of difference equations, (6) and (19) with schooling level s as fixed parameter, determine the equilibrium economic and disease dynamics in our model. In our special

case, notice that the disease dynamics in equation (6) does not depend on the dynamics of $\widetilde{k}_t$. Thus the process i_t act as an exogenous forcing factor on the dynamics of $\widetilde{k}_t$.

Corresponding to two locally stable steady-state equilibria for malaria incidence rates $i = i^* > 0$, and $i = 0$ in 4, there corresponds two long-run balanced growth rates of per capita income. The long-run growth rate of per capita income y_t in these two types of equilibria are $\gamma(i^*, s)$ at the malaria endemic equilibrium which is smaller than the growth rate at the malaria free equilibrium $\gamma(0, s)$. In the short-run, the growth effect around these two equilibria will be even higher. For instance, around the disease free equilibrium, the disease infection rate will be falling and hence the lost time from infection will be declining, and contribution to social knowledge for productivity growth will be increasing over time. The scenario is exactly opposite around and below the malaria endemic equilibrium rate of infection i^*.

Some of the African countries and Asian countries might be stuck at the malaria endemic bad equilibrium and others in the malaria free good equilibrium. The point to note is that without government and foreign aid, the malaria disease prone tropical countries will be stuck in a malaria endemic equilibrium with low or negative growth in labor productivity and in per capita income as compared to the malaria free non-tropical countries which will settle in a good equilibrium with no malaria and higher growth in productivity and per capita income. Two types of interventions that can move the malaria prone tropical economies from malaria endemic bad equilibrium to malaria free good equilibrium are: 1) external natural disasters such as extreme drug resistance or some natural evolutionary phenomena leading to high growth of disease incidence and malaria prevalence rate $i > \bar{i}$, so that there is a complete behavioral change for everyone to adopt to malaria prevention care; 2) since the government in these countries have limited resources, foreign governments may provide aid to subsidize the cost of malaria preventive care until the disease prevalence rate falls below the level i^c in the diagram so that the momentum of the disease spread move downwards and ultimately converge to malaria free good equilibrium. In neither case, the malaria free good equilibrium will be sustained for ever. For instance, any external factors, such as migration of malaria parasite may ignite the disease spreading process. We name this phenomena as **neo-Malthusian.**

6 Policy Implications and conclusions

It is clear from (6) that prevalence of infection rises at time t, i.e., $i_{t+1}/i_t > 1$ if and only $R_0 = (1 + \rho(i_t))(1 - \bar{\theta}(i_t)) > 1$, following the convention of continuous time infectious disease dynamics literature, I will refer to R_0 as the **reproduction rate of the disease.** Notice that ρ depends on the individual economic decisions about curative care which affect duration of malaria infection, and on the prevalence of Anopheline mosquito population. Notice that by providing medical cares at subsidized rates can reduce the duration of malaria infection T and in turn reduce $\rho(i_t)$, and any public malaria control programs such as spraying of pesticides to control mosquito population will also lower $\rho(i_t)$. Similarly, any policy such as subsidization of insecticide impregnated bed nets can increase $\bar{\theta}(i_t)$. These policies can have a long-run effect in terms of having lower level of malaria prevalence rate i^* at the malaria endemic equilibrium, and thus a higher rate of growth in per capita income and lower infant mortality and morbidity. Such policies will also have short-run gains by lowering the disease burden and having higher rate of growth in income along the transition path.

This leads to the Threshold Theorem of Kermack and McKendrick for public intervention for malaria control: in periods during which reproduction rate of the disease R_0 crosses the threshold level 1, public intervention is needed to increase $\bar{\theta}(.)$ and to reduce ρ and thus lower the value of R_0 below the level 1, otherwise the disease will become epidemic.

Another important policy issue is to examine the effect of public education about true effects of malaria on productivity loss. I have shown that there are economies for which such public education can generate enough private demand for malaria preventive care such that eventually malaria can be eradicated. Even for other economies, public education will definitely boost private demand for preventive care which can postpone drug resistance, and lower the incidence of malaria infection in the steady-state and increase the rate of growth of per capita income both in the short-run and long-run. Whether these benefits of public education regarding malaria exceeds the cost of providing such education is an empirical question need further investigation empirically.

A basic element of this framework is that one can compute the private and social burden of the disease in terms of lost earnings, lost lives, and lost rate of productivity growth. In my future work, I intend to simulate this model calibrating the parameters of the model and introducing individual choices in curative care and taking its effects on drug resistance as observed in many parts of the world.

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