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Malaria: Disease Impacts and Long-Run Income Differences

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Abstract

The World Health Organization (WHO) reports that malaria, a parasitic disease transmitted by mosquitoes, causes over 300 million episodes of ”acute illness” and more than one million deaths annually. Most of the deaths occur in poor countries of the tropics, and especially sub-Saharan Africa. Some researchers have suggested that ecological differences associated with malaria prevalence are perhaps the most important reason why some countries today are rich and others poor. This paper explores the question in an explicit dynamic general equilibrium framework, using a calibrated model that incorporates epidemiological features into a standard general equilibrium framework.

Journal of Economic Literature Classification: I1, O11, E13, E21

Keywords: Malaria, Epidemiology, GDP, Disease prevention, Sub-Saharan Africa.

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1. Introduction

The World Health Organization (WHO) reports that malaria, a parasitic disease transmitted by mosquitoes, causes over 300 million episodes of "acute illness" and more than one million deaths annually.[1] Most of the deaths occur in poor countries of the tropics, and about 90 percent occur in sub-Saharan Africa. Infants and children account for most of the mortality from malaria; the disease is thought to account for one of every five child deaths in the world.[2]

Even where people survive malaria, the disease causes numerous health and cognitive problems. It is associated with maternal anemia during pregnancy, with low birth weight for babies, and it is a major cause of childhood anemia. Severe disease episodes (*i.e.*, "cerebral" malaria) have been shown to cause severe long-term physical and neurological disability. There is no clear evidence on the cognitive impact of malaria on individuals who contract less severe cases of the disease, although there are reasons to suspect non-trivial effects on learning among schoolchildren.[3]

There is no effective vaccine or inoculation to prevent malaria. However, the disease can be treated at relatively low cost (at least in its milder forms) with drugs or even simple measures to reduce the severity of symptoms. Prevention measures are also relatively inexpensive. For example, mosquito nets impregnated with insecticides, available for $5-$10 each (or less), can significantly reduce exposure to mosquitoes and thereby limit malaria morbidity and mortality.[4] But these measures may require careful implementation and recurring maintenance, which may not be feasible for many affected families.

At present, many of the world's poor countries face high rates of malaria endemism. By one estimate, about 40 percent of the world's population lives in areas where malaria is endemic, and these people are on average very poor. According to the United Nations Children's Fund (UNICEF), "Malaria is truly a disease of poverty. It afflicts primarily the poor, who tend to live in malaria-prone areas in dwellings that offer few, if any, barriers against mosquitoes" (UNICEF 2005). Sachs and Malaney (2002) argue that "[a]s a general rule of thumb, where malaria prospers most, human societies have prospered least…. The extent of the correlation suggests that malaria and poverty are intimately related."

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[1] Reported by WHO on the "Roll Back Malaria" program website at: http://mosquito.who.int/cmc_upload/0/000/015/372/RBMInfosheet_1.htm, January 30, 2005.
[2] Reported by the United Nations Children's Fund (UNICEF), http://www.unicef.org/health/index_malaria.html, accessed June 10, 2005.
[3] A useful survey is Holding and Snow (2001).
[4] UNICEF reports that the use of such bednets can reduce child mortality from malaria by 20 percent (http://www.unicef.org/health/index_malaria.html, accessed June 10, 2005).

The causality of this relationship is complicated, however. Does malaria cause poverty? Or does poverty cause malaria? Both channels of causation seem reasonable. It is also possible, as noted by Sachs and Malaney (2002), that the correlation could be spurious, caused perhaps by some other direct connection between climate and geography with growth rates or income levels. Resolving these causality issues has been difficult for researchers trying to assess the economic impact of malaria.

In spite of the difficulties involved, two widely publicized papers have found that malaria appears to slow economic growth in poor countries. Both papers use cross-country regression techniques and attempt to use instruments or controls to address the obvious causality problems. McCarthy, Wolf, and Wu (1999) find that malaria prevalence is negatively related to growth of per capita income. In turn, they find that malaria morbidity is linked to climatic differences across countries. The magnitude of malaria's effect on growth is substantial: they find that Sub-Saharan African countries experience a reduction in income growth of 0.55 percent annually because of malaria. Using a relatively similar methodology, Gallup and Sachs (2000) find that countries with "intensive" malaria experience a reduction in per capita income growth of 1.3% annually. They suggest that, everything else being equal, a country experiencing intensive malaria would have its long-term level of income per capita reduced by one-third, compared with the same country in the absence of malaria.[5]

Based on this analysis, Sachs and other authors have suggested increasing current spending on malaria control by more than an order of magnitude. Global spending on malaria prevention and control is currently around $100-200 million annually.[6] But based in large part on his estimates of the economic impacts of the disease, Sachs (2005b) has estimated that $2-3 billion in annual spending would be needed to control the disease effectively in Africa alone. These larger sums are clearly within the capacity of the international community, but they would represent a substantial fraction of total aid disbursements by rich countries.[7] As a result, the increases would either require significant reallocation of existing aid portfolios or increases in the total quantities of foreign assistance given by rich countries.

To the extent that such increases in expenditure are justified by appealing to the likely impact on income levels and growth rates in malarial countries, it is useful to look further at the evidence for malaria's impact on income levels.

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[5] These numbers passed from academic research into policy; in the Abuja Declaration of 2000 40 African heads of state and governments signed on to a major commitment to fight malaria, citing these numbers as one major justification.

[6] Sachs and Malaney (2002) use the lower estimate.

[7] In 2002, OECD countries gave $58.3 billion in foreign assistance of all kinds.

In the empirical literature, Acemoglu and Johnson (2006) offer a far more skeptical view of the growth effects of disease, based on an instrumental variables approach. They are joined in this skepticism by Weil (2006) and Cutler, Fung, Kremer and Singhal (2007). Some other authors (Bleakley 2007, Lucas 2005), however, find evidence that malaria eradication campaigns resulted – after very long time lags – in quantitatively significant impacts on health, fertility, and income. Some difficulties with the empirical literature include the paucity of reliable data and the inherent difficulty of identification.

To provide a different perspective on the issue, we find it useful to present a formal model of malaria in a dynamic setting. Our paper is somewhat related to work by Chakraborty, Papageorgiou, and Pérez Sebastiàn (2007) that looks at an overlapping generation economy with malaria.[8] But because their analysis is based on a two-period model, quantitative results on the magnitude of disease impacts are hard to interpret.

Our paper brings an explicit dynamic general equilibrium framework to the question of malaria's impacts. We incorporate an epidemiological model of disease (following Gersovitz and Hammer 2004, 2005 or Philipson 2000), with a standard general equilibrium framework. Using a calibrated version of the model, we examine the impact of malaria on steady-state economic outcomes in the absence of prevention and control measures. We also model the impact of costly prevention measures, including measures that are less than fully effective.

2. Background

Malaria is an ancient disease, although its exact origins and evolutionary history are unclear. It was described in China some five thousand years ago. It is thought to have originated in Africa and to have spread subsequently into Asia and the Mediterranean. Greek writers recognized the disease and its symptoms, and one source notes that malaria was responsible for the decline of city-state populations and depopulation of rural areas.[9] The disease appears to have migrated to the New World following the Columbian exchange, and to this day, fewer different strains of malaria are found in the Americas than in Africa and Asia. Recent research suggests that the origins and spread of the disease in the Old World paralleled the spread of sedentary agriculture (Tishkoff et al., 2001).[10]

[8] An earlier version of this paper is circulated as Papageorgiou, Chakraborty, and Pérez Sebastiàn (2005); this covers similar ground using a closely related model, but with an endowment economy that offers little insight into the interaction between income and disease.
[9] See http://www.cdc.gov/malaria/history (cited June 2005).
[10] See also McNeill (1976), pp. 219-221.

Historically, malaria was endemic in most regions of the world. The morbidity and mortality burden of malaria differ from country to country, in part because the prevalence of the disease (and the conditions that give rise to the disease) differ substantially across regions. Hamoudi and Sachs (1999) report that historically, malaria was found as far north as 64° N latitude (farther north than Stockholm or Moscow) and as far south as 32° S.

2.1 Disease biology and ecology

Malaria is a disease caused by a family of macroparasites that infect humans. There are in fact four species of *Plasmodium* parasites that cause malaria in people. These four species have similar life cycles; all are transmitted to humans by a mosquito vector (various species of *Anopheles* mosquitoes) and live a portion of their life cycle in the mosquito host.

A person is infected with malaria when he or she is bitten by an infected mosquito, which passes the *Plasmodium* parasite into the person's bloodstream in a form known as a sporozoite. The parasites lead a complex life cycle inside the human host, living at various stages in liver cells and red blood cells. From time to time, they cycle through stages in which they destroy numerous red blood cells. It is at this stage that the disease generates its most severe symptoms in infected people. Eventually, the parasites become gametocytes which are in turn ingested by mosquitoes that bite the human host. Inside the mosquito, the gametocytes mature, reproduce sexually, and migrate into the mosquito's salivary glands, at which stage the life cycle is repeated.[11] For some species of *Plasmodium,* the parasites may persist in the liver for months or years, resulting in chronic and recurring eruptions of merozoites that correspond to episodes of fever and sickness.

The disease varies with the infecting species of *Plasmodium* and with the individual's prior health and immune status. Typically, it causes fever and chills, along with headaches, vomiting, and diarrhea. It may also cause long-term anemia, liver damage, and neurological damage. The most dangerous species, *P. falciparum*, can cause cerebral malaria, a frequently fatal condition involving the brain and central nervous system. Those who survive cerebral malaria may experience lasting brain damage.

The prevalence of the disease varies across the globe, largely due to differences in the human exposure to *Anopheles* mosquito bites. Some of this variation is geographic and climatic: these mosquitoes are not found in areas of

[11] The life cycle is described and illustrated at: http://www.dpd.cdc.gov/dpdx/HTML/Malaria.htm (viewed June 2005).

intense cold or in deserts (Sachs and Malaney 2002). Human exposures are also reduced in areas where mosquitoes spend winter months as eggs or in dormant stages of their life cycle. Exposures may also be reduced in areas where people spend significant fractions of their time indoors in enclosed or screened buildings, or where people are dressed in ways that will reduce exposure.

The ecological adaptation of different mosquito species is also important. Although many species of *Anopheles* mosquitoes are capable of transmitting the *Plasmodium* organisms, transmission occurs only when a mosquito first bites an infected human and then subsequently bites another (uninfected) human. Some species of mosquitoes, however, prefer not to feed on humans (although they will do so if other food sources are not available). Others are anthropophilic; *i.e.*, they prefer to feed on humans. Anthropophilic mosquitoes are obviously more likely to transmit malaria from individual to individual. Thus, areas where anthropophilic mosquitoes are prevalent are likely to face more acute malaria burdens.

McNeill (1976) notes that the geographic distribution of mosquito species is largely due to chance, from the perspective of humans. The distribution depends on highly local ecological differences (trace minerals in the water, salinity of water, types of habitat, etc.). Thus, pure ecological chance had large effects on the relative prevalence of different mosquito species, across the globe, and hence on the relative prevalence of malaria. McNeill notes that "the mosquito species which is Europe's most efficient vector of malaria… prefers to feed on cattle. If enough alternate sources of blood are available to them, these mosquitoes will eschew potential human hosts and thus interrupt the chain of infection, since cattle do not suffer from malaria" (p. 117).

In fact, one of the apparent reasons for the extensive malaria burden in sub-Saharan Africa is the prevalence of two species of highly anthropophilic species of *Anopheles* mosquitoes: *An. gambiae* and *An. funestus*. These two species together inhabit much of the humid zone of Africa, and only the northern and southern extremities of the continent are free from these strongly anthropophilic species.[12] This clearly plays a significant role in accounting for malaria's impact in the region, although poverty may also play an important part (Sachs and Malaney 2002).

It is true that the types of mosquito prevalent in different regions are, in an ecological sense, related to human impacts on the landscape. It is also true, however, that the distribution of mosquito species across the landscape is largely exogenous in the short run. For the purposes of this paper, we will treat mosquito habitats as an exogenous characteristic of a place.

[12] The distribution of *Anopheles* species around the globe is shown at: http://www.cdc.gov/malaria/biology/mosquito/map.htm.

2.2 Economic impact of malaria

As noted above, malaria causes morbidity and mortality with obvious economic consequences. Sachs and Malaney (2002) survey a number of the impacts of malaria. The direct individual economic impacts of the disease include the value of lives lost, the value of time lost to sickness, and the expenditures on medical care, treatment, and prevention. Direct social costs include government expenditures on malaria control and prevention. The indirect costs may be greater still. These include changes in human settlement and labor patterns induced by the disease (e.g., changes in the locations where people live or farm). Indirect costs also include the consequences of the disease on fertility, demography, and human capital investments; on trade patterns and investment; and potentially on managerial quality and technology adoption. (For example, skilled managers may prefer not to work in malarial regions, resulting in reduced productivity levels.)[13]

Impacts of malaria on fertility and human capital decisions are difficult to document, since all are related to income levels. The same is true of malaria's impacts on trade and investment and many other variables that are correlated with income levels.

3. The Base Model

To consider the impact of malaria on income levels, we need a model environment in which both channels of causation are present: in other words, malaria can affect income and/or productivity, and simultaneously, income and/or productivity can affect the prevalence of the disease. We also need to use a model in which it is possible to consider the behavioral responses that individuals may take to reduce the short-term and dynamic impact of the disease. Finally, we need a model in which it is possible to consider alternative policies for controlling malaria.

We present here the simplest possible model that has all the necessary features for addressing the link between income and disease prevalence. It tracks the dynamics of the spread of malaria, has endogenous production and prices, factors

[13] At an even more remote level, it might be possible to view human biological adaptations to malaria as part of the indirect cost. Thus, sickle cell traits, found in some individuals of African descent, are damaging and costly in their own right. Medical literature strongly suggests that the sickle cell trait confers some resistance to malaria and is thus an adaptive evolutionary response to the disease. Arguably, then, we could count the costs of sickle cell anemia as one of the indirect costs of malaria.

in idiosyncratic shocks – both from malaria and other sources – and takes into account the differential mortality for sick and disease-free people. There are no insurance markets, but households can accumulate assets to self-insure against persistent idiosyncratic shocks. Essentially, this is a Huggett (1996) economy with epidemiological features embedded. The epidemiological aspects of the model are similar to those presented by Philipson (2000), and we borrow from his analysis of "rational epidemics."

3.1 Model environment

The model environment has many individuals, born identical. New individuals are born each period. Some individuals die in each period, with mortality rates dependent on infection rates. Individuals are exposed to the disease in each period; some fall sick. The probability that an individual will become sick is positively related to the fraction of individuals in the population who are already sick, so infection rates are endogenous to the model. Sick individuals face heightened probabilities of death and lower labor productivity. To avoid population explosions or collapses, we model fertility rates as consistent with a constant population level.

Individuals are born healthy. They have zero initial asset holdings, but they accumulate assets through their lives. Assets can be rented to a representative firm in a perfectly competitive market for current-period production. However, there is no credit market, nor is there any insurance market. Therefore, individuals in the economy will use precautionary savings to protect themselves from idiosyncratic shocks. Assets vanish when people die.[14]

Note that some characteristics of this asset make it similar to human capital: people are born with no positive endowment; they cannot hold negative amounts; and their holdings disappear upon death. It is also the only savings technology in a rudimentary economy. In other respects, however, the asset is perhaps more like physical capital: it is measured in the same units as output and consumption, and thus it can be used to smooth consumption or to make "lumpy' purchases. In these respects, the asset is more analogous to physical capital than to human capital.

As in Philipson (2000), individuals may, at any point during their lives, make a lumpy purchase of a preventive good that will confer future protection from malaria. This lifetime prophylaxis requires a one-time expenditure of q units of consumption good. We think of this as the present value of a lifetime expenditure

[14] This assumption effectively serves as a type of depreciation in the economy. We could equally well allow for assets to be redistributed to the new generation. The qualitative results of the model would not change significantly.

stream on bednets, drugs, and other preventive goods. Alternatively, if an effective vaccine were to become available for malaria, we could model this as the cost of the vaccine. Note that this is an indivisible purchase, and we initially model it as being totally and perfectly effective. In other words, once an individual has purchased the prophylaxis, he or she does not subsequently contract malaria, and there is no need for future spending. Subsequently, we will relax the assumption of perfect efficacy. In fact, our quantitative results, reported below, show that that when the preventive goods offer imperfect protection, there are large quantitative impacts on uptake, infection rates, and economic outcomes.

3.2 Preferences and endowments

Preferences for any household *i* are given by the period utility function:

$$u\left(c_{it};s_{it}\right)=\frac{s_{it}\left[\gamma\left(c_{it}-\overline{c}\right)\right]^{1-\rho}}{1-\rho}$$

with lifetime utility given by: $\sum_{t=0}^{\infty}\beta^{t}u\left(c_{it};s_{it}\right)$, where s_{it} reflects a utility cost of being sick, such that $s_{it}\in\left\{\overline{s},1\right\}$, $0\leq\overline{s}\leq 1$. A value of $s_{it}=1$ corresponds to health, and a value of $s_{it}=\overline{s}$ corresponds to sickness. The parameter γ is a scalar that will determine the utility level of subsistence, and it will be calibrated below to give a plausible value for the "value of life" for people in the model economy.

Given their health status, households care only about consumption. They also face a subsistence consumption requirement, $\overline{c}$. This may be important in determining the affordability of disease prevention measures for different households.

Individuals are endowed with one unit of labor time in each period, which they supply inelastically to the labor market. Their effective labor units depend on health status, s_{it}, and π_{it}, which is an indicator of labor efficiency. This efficiency parameter is subject to idiosyncratic shocks and evolves according to a Markov process. Healthy individuals supply one raw unit of labor; if they are sick, however, their raw labor supply is reduced to $\overline{h}$. Effective labor units are determined by the raw labor supply and the idiosyncratic shock, so that:

$$h_{it}=\begin{cases}\pi_{it}\overline{h}, \text{ if } s_{it}=\overline{s}\\ \pi_{it}, \text{ if } s_{it}=1\end{cases}$$

.

Individuals have the capacity to influence their health status through the decision of whether or not to purchase prophylaxis against malaria. We define q to be a basket of consumption goods necessary to achieve permanent disease protection (described in more detail below), and p_t is individual i's decision to purchase q. This choice is a binary choice, such that $p_{it} \in \{0,1\}$.

Given this setup, the individual's period budget constraint is given by:

$$c_{it} + k_{i,t+1} + p_{it}q \leq w_t h_{it} \pi_{it} + r_t k_{it}$$

where $k_{it} > 0$ denotes accumulated assets, r_t is the return to capital, and w_t is the wage.

3.3 Technology

The technology side of our model economy is characterized by an aggregate technology with constant returns to scale. Individual effective labor units aggregate to $L_t = \sum_i h_{it}\pi_{it}$, and individual asset holdings aggregate to the physical capital stock $K_t = \sum_i k_{it}$. These are used to produce output Y_t according to the Cobb-Douglas production function:

$$Y_t = K_t^{\alpha} L_t^{1-\alpha} .$$

Factor prices then correspond to the marginal products of the factors. Thus, we assume that there is a perfect rental market for factors in this economy, with only spot markets available. Firms earn zero profits, and since there are no fixed costs, we can treat the economy as having a single cost-minimizing aggregate firm which rents capital and labor from the population and earns zero profits in equilibrium.

3.4 Population dynamics

In such an environment, population dynamics become important. We need to specify birth and mortality rates, which are differentiated across populations of sick and healthy people. We also need to model the risk of infection. Let d_h and d_s be the death rates of healthy and sick people, respectively. Let their fertility rate be f.

Defining N as the total population, we denote S as the proportion of sick people:

$$S = \frac{\sum_i S_i}{N}, \text{ where } S_i = \begin{cases} 1, \text{if } s_i = \overline{s} \\ 0, \text{otherwise} \end{cases}.$$

Trivially, the proportion of healthy people in the economy can be written as $H = 1 - S$. Let V be the proportion of people who have purchased prophylaxis. This is effectively a stock variable. In each period, there are also people purchasing prophylaxis; this fraction is given by:

$$P = \frac{\sum_i p_i}{N}.$$

This group can in turn be divided into those who purchase when healthy and those who purchase when already sick. The healthy purchasers are given by:

$$P_h = \frac{\sum_{i \in H} p_i}{H},$$

while those purchasing q when already sick are given by:

$$P_s = \frac{\sum_{i \in S} p_i}{S}.$$

Define the indicator variable v_i such that it takes a value of unity for individuals who have ever purchased protection and zero for all others. Then the fraction of individuals who are protected from disease is given by:

$$V = \frac{\sum_i v_i}{N}.$$

Note that these individuals may be sick or healthy at the time when they purchase protection. Thus, we have $V_s = \frac{\sum_i v_i S_i}{N}$ and $V_h = \frac{\sum_i v_i (1 - S_i)}{N}$. In equilibrium, people who are sick will not choose to purchase protection, since it will not cure them of the disease. (We could model this differently, without any substantive change in the results.)

3.5 Laws of motion

Armed with this notation, we can write the laws of motion for different groups in the economy as follows:

For population, the net increment to population comes from deaths of sick and healthy people and from the fertility of sick and healthy people. Note that we do not treat men and women separately, nor do we model fertility rates as age-dependent, so that all individuals in the model economy can bear children. Thus:

$$N' = N - d_s S - d_h H + fN.$$

The proportion S of sick people depends on births, deaths, and infection. Let I be the infection rate for healthy people who have not purchased prophylaxis. Then:

$$S' = \frac{N\left[S - d_s S + IH\left(1-V\right)\left(1-d_h\right)\right]}{N - d_s S - d_h H + fN}$$

The proportion of people who are protected from disease evolves according to the law of motion

$$V' = \frac{N\left[V - d_s V_s - d_h V_h + P_h H\left(1-d_h\right) + P_s S\left(1-d_s\right)\right]}{N - d_s S - d_h H + fN}.$$

We need still to characterize the infection rate I that applies for healthy people who have not purchased prophylaxis. Following Philipson (2000), we assume that the probability of contracting an infection depends on the proportion of people already infected and also on the inherent ecology of the disease. Thus, we make use of a formulation in which the infection rate itself evolves according:

$$i = Z\left(\frac{S}{N}\right)^{\mu}$$

where $\left(\frac{S}{N}\right)$ is the fraction of the population that is currently sick, Z is an index of malaria ecology, and μ is a parameter. This function has important properties. If either the population is fully healthy or the malaria ecology is zero, the next period's infection rate will be zero: this is a steady state. It is also the case that if both the infection rate and the ecology are at 1, this is another steady state. Note that our treatment of infection differs slightly from that of Philipson, whose "hazard rate" for infection combines both the natural rate of infection and the behavioral response. We define i here to be the probability that an <u>unprotected individual</u> will become infected in the next period; i.e., conditional on the

individual not purchasing protection. Philipson's hazard rate, by contrast, gives an unconditional probability.

Finally, defining $C_t = \sum_i c_{it}$, and dropping time subscripts, the law of motion for the aggregate capital stock is given by $K' = K + Y - C - PqN - d_s K_s - d_h K_h$, where K_s and K_h are respectively the aggregate capital held by the sick and the healthy**.** Note that the distribution of capital across individuals is non-degenerate. Indeed, good and bad luck with idiosyncratic shocks and health determine how much an individual accumulates. There is no borrowing or lending, nor is there insurance, so capital acts as a "rainy day fund" for individuals in the economy.

3.6 Equilibrium

We will define an equilibrium in this economy using a recursive approach. An equilibrium will consist of functions of the state variables for the economy and for the individuals:

- Functions for prices and wages;
- Functions for individual consumption, asset holdings, labor supply, and disease protection decisions;
- Distributions of health status and capital across individuals.
- Functions for the aggregate labor and aggregate capital employed in production, and the aggregate output produced;
- Laws of motions for each type's endogenous state

such that individuals of each type maximize utility subject to budget constraints, across states; the representative firm maximizes profits, subject to zero profits; factor markets and goods markets clear; the distributions of health status and capital are invariant; and the individual functions are consistent with the aggregate laws of motion for the economy.

Characterizing and solving for the equilibrium of this economy can be complicated. Note that disease dynamics imply that this economy will display multiple steady states. To see this, observe that with $S = 0$, there will be a steady state regardless of how many people purchase prophylaxis. In general, the existence of an interior steady state ($0 < S < 1$) will depend on the cost of the protective goods, q, relative to the subsistence consumption requirement and the distribution of capital per person in the economy. With higher levels of capital, the economy can jump from one in which prophylaxis is generally unprofitable to

one in which it is universal. Some poor economies, however, will never escape the high-disease trap. By contrast, other economies will start with sufficiently high levels of capital per worker that they will defeat the malaria burden.

The steady states here differ a little from those of standard Solow models. Like other models of this type, the steady state is determined as the point at which asset accumulation (initial asset endowments of the newly born plus savings from those who are alive) exactly offsets the loss of capital that occurs when individuals die. Our models display multiple steady states, because of the disease dynamics involved.

We view the multiplicity of steady states in the model as a substantively useful one for thinking about why some countries have been able to leave behind the problems of malaria, while other countries – even those with similar climate and geography – remain caught in a trap characterized by low productivity and high infection. For example, Singapore has effectively eliminated malaria infection, whereas Congo – a country that is reported to have a comparable malaria ecology – suffers from vastly higher rates of infection. Pakistan and Sri Lanka have roughly comparable malaria ecologies (Sachs et al. 2004), and income per capita in Sri Lanka is almost double the level in Pakistan (Heston et al. 2002), but Sri Lanka has a reported malaria prevalence rate that is 20 times that of Pakistan (Asian Development Bank 2005).

In our model, multiplicity allows for countries at similar income levels and with similar malaria ecologies to have different equilibrium levels of malaria prevalence, prevention, and other variables.

Finally, we note that the model economy – as is typical of models with infectious disease – is characterized by an important externality related to the transmission of disease. An individual contemplating the decision of whether or not to purchase prevention does not take into account the potential impact of her decision on the infection rates faced by others. As a result, private actors are likely to purchase inefficiently low levels of the preventive good. Thus, there may be a role for the government to subsidize the bundle of preventive goods.

4. Calibration

We are interested in a set of quantitative experiments in which we assess the effects on aggregate output of various (exogenous) changes that will affect both malaria prevalence and economic variables of interest. The first such experiment is to ask simply how large an effect malaria can have in an economy where no protective measures are available; in other words, where there is no behavioral response that is effective in reducing the burden of malaria. Arguably, this is a

useful framework for thinking about the impact of the disease in some of the most severely affected environments, where neither spraying nor chemoprophylaxis nor drug treatments are effectively able to reduce the proportion of people suffering from the disease.

Specifically, the experiment we conduct is to calibrate the model to a set of benchmark parameters and then to suppose that the cost of a preventive bundle of goods, represented by q, is prohibitive.

To carry out this and our subsequent experiments, we need to select values for the parameters of the model. A number of the parameters we take from the literature, and others we choose to match observations for a stylized poor malarial country. For all the important parameters of the model, we perform robustness checks, as described below.

The parameters for preferences we take to be standard. The discount factor β we set to 0.95, assuming annual frequency, and we set the risk aversion parameter $\rho = 1$. The disutility of sickness is measured by the parameter $\bar{s}$, which we set equal to 1.0 in the benchmark economy, implying no disutility. We also report robustness checks using a value of 0.9, which is consistent with estimates of "disability weights" such as those reported by Murray and Lopez (1996). The change is not quantitatively important in our model.

Since malaria increases the probability of death in our model, we also need to consider the value that people associate with living as opposed to dying. For this we draw on estimates from the U.S. that estimate the statistical value of a life at approximately $4 million to $9 million (Viscusi and Aldy 2003). Taking $7 million as a reasonable middle number, we compute that this is approximately 11.3 times lifetime consumption in the U.S. As a result, we set the subjective value placed on living at 11.3 times annual consumption in the benchmark economy, which pins down a value of $\gamma = 11.3$. This number is also subjected to some robustness checks, which are reported below.

We use a value of 0.9 for the labor efficiency units of a person infected with malaria. This reflects a number of micro studies in the literature and is broadly consistent with Bleakley's work (2003) looking at malaria in the U.S. South. The subsistence constraint is set to zero in the benchmark economy.

Individuals also face idiosyncratic shocks independent of the risk of contracting malaria. We need to specify both the transition matrix for shocks and the magnitude of the shocks. In the experiments reported here, the magnitude of the shocks is taken to be 0.224 (following Domeij and Heathcote 2004), while the transition matrix is set to:

$$\begin{bmatrix} .900 & .100 \\ .100 & .900 \end{bmatrix}.$$

We use a capital share on the aggregate production technology of 0.36, in keeping with standard practice in the literature.

This leaves fertility rates and death rates for healthy and sick people, plus the crucial parameters relating to the cost of preventive goods and the infection rate.

Death rates are taken to be 0.075 for sick people (i.e., those infected with malaria) and 0.015 for healthy people. It is difficult to know which observations in the data to use for calibrating these parameters, but we believe the results to be quite robust to the death rates. To simplify the analysis, we set the fertility rate such that population will be stable in equilibrium. In other words, we allow the fertility rate to adjust to offset the deaths of sick and healthy people.

The cost of prophylaxis is another critical parameter for the model. Chima *et al*. (2003) provide a good summary of the literature on the costs of prevention and treatment of malaria in Africa. These numbers are hard to interpret, because (a) the figures given are often averages that include people who did not purchase preventive goods; (b) the goods on which people are spending money are not in fact effective in prevention (e.g., mosquito coils); and (c) the expenditure on bednets, screens, and mosquito coils is only partly intended to reduce malaria incidence, while also serving the purpose of reducing the annoyance of mosquito bites. Nevertheless, some reasonable numbers come out: bednets cost between $5 and $10 per person and last perhaps five years under reasonable use. At an interest rate of 0.05, the present value of a lifetime stream of bednet purchases at $5 is about $20-$25 per person, which assuming per capita income of about $500 could be modeled as a one-time fixed cost of 4-5% of annual per capita income. At $10 per bednet, obviously, the number rises to 8-10% of per capita income. (At an interest rate of 0.10, this falls back to 5%.) The estimates of eventual vaccination costs are not much different in NPV terms, with estimates of $20-$60.[15] Thus, it seems that realistic values for this cost might range from 0.05 to 0.10 of annual income.

Finally, we have the parameters Z and μ for the infection rate process. Using the malaria ecology index of Sachs et al. (2004), we re-scale to define the index on the interval [0,1] and then find a value of 0.7 for a "typical" malarial country. In the data, this corresponds roughly to the level prevalent in Cambodia, Mozambique, Guinea-Bissau, or Congo. For that matter, it is also the malaria ecology prevalent in Singapore, a country with essentially no malaria. Thus, the malaria ecology value that we choose is consistent in the real world with both malarial countries and malaria-free countries.

[15] At present, of course, no effective vaccine is available.

The parameter μ gives the elasticity of next period's infection rate with respect to the malaria ecology. We can estimate this by regressing infection rates on malaria ecology. A value of 0.122 was obtained from this regression.

5. Experiments and Results

Using the calibrated model, we conducted a number of experiments that we report below. The first experiment considers an economy in which protection from malaria is not possible. We compare its healthy and unhealthy steady states. The second experiment considers the same question for an economy in which malaria protection is available, though costly. We carry out this experiment for a large range of possible costs. Finally, we repeat the second experiment for a range of possible parameter values, to assess the robustness of our results.

Experiment 1:

The first experiment that we consider is one in which we compare the benchmark economy in two steady states, one of which has everyone healthy and the other of which has essentially all people sick. A simple way to arrive at these steady states is to set the cost of the preventative good at a very high level, so that it is effectively unavailable. The two steady states can be found by initializing the economy with all sick people or all healthy people. Both steady states are feasible, and initial conditions in the model economy will determine which one pertains. An economy that begins poor and sick will tend to say poor and sick, while one that starts with better health or higher initial assets will end up at a better steady state.

The comparison of these two steady states offers an insight into the maximum possible impact of the disease within the model economy. In effect, we are examining the case in which there is no behavioral response to malaria. This provides a kind of upper bound of the disease's impact, within the model.

Table 1 shows the results of this experiment. The impact of the disease in this case is large. The steady state with widespread malaria infection has an income per capita that is 43 percent lower than that in the healthy steady state. Per capita consumption is even lower, with a 49 percent reduction from the healthy steady state. The proximate cause of the reduction is that steady state asset holdings are only 25 percent of the value in the healthy steady state. This reflects the shorter average lifespans of people in the malarial steady state: they do not live as long as those in the healthy steady state, nor do they expect to live as long, and they are poorer while alive. As a result, they save at a lower rate and accumulate assets over a shorter period. Figure 1 shows the distributions of asset holdings for the healthy steady state and the malarial steady state, and the impact of the disease is

evident. It is this effect, rather than the direct impact of the disease on effective labor units, that has the greatest impact.

One way to think about this experiment is to view it as the benefits to the model economy of escaping from its malarial steady state and moving to its healthy steady state. Are there substantial impacts on income, as Sachs seems to suggest? Can we identify important *ex post* differences between the two economies?

The answer here is that there is a large difference in steady-state incomes between the two economies. The difference is not sufficient to explain why malarial countries are poor and non-malarial countries are rich, but it is true that in the model economy, eradication of malaria would lead approximately to a doubling of per capita income. This is a large impact. Note that the macro impacts of the response are far greater than the micro impacts; although individuals lose only 10 percent of their labor productivity to the disease, the lower asset accumulation leads to an amplification mechanism through which the disease impacts are multiplied.

Experiment 2:

In the second experiment, we ask again about the impact of a single economy moving between its "healthy" steady state and its "sick" steady state. In contrast to the first experiment, however, we assume that an effective preventive good is available, though costly. Table 2 reports the results for a cost of prevention approximately equal to 25 percent of steady-state annual income; this would be comparable to a one-time cost of prevention of around $90, in an economy in which annual income is about $1 per day.[16]

What is the quantitative impact of the disease when costly but effective prevention measures are available? Table 2 shows the results of the experiment, comparing outcomes across the low and high steady states for a model economy in which $q = 0.6$. In this experiment, the economy in its low steady state has essentially the entire population protected from malaria, even though the protection is quite costly. People are willing to spend a large fraction of income to avoid getting sick.

The disease is not entirely eradicated, however, and people cannot forego the costs of protection. Indeed, as long as some malaria is present, there are individuals who become infected as newborns before they are able to buy prophylaxis. Clearly this is rare, but it does imply that the steady state of this economy gives slightly lower welfare one in which the disease is actually

[16] This is not far from the expected lifetime costs of a vaccine that has to be readministered at five-year intervals, or from the total expected lifetime cost of insecticide-treated bednets, as calculated by Johnson (2007).

eradicated, where no one needs to bear the cost of the preventive good. Asset holdings, production, and consumption are all slightly lower than in the steady state with no malaria.

This result – that malaria matters only little – is at first sight surprising, given that we have given the disease every chance to have a major impact. The cost of lifetime protection is substantial and must be paid in full up-front (i.e., there is no borrowing to finance prophylaxis); agents are born with no assets; and they must also hold capital for precautionary savings. The risks of infection are low, since others are generally healthy. Yet even so, individuals in this economy are still willing to pay for protection as soon as they can afford it, and they afford it rapidly.

This seems to cast doubt on the potential for the disease to cause large macro effects in reality. Why would people in endemic areas not behave like individuals in the model? Even if the individual costs of the disease are modest, would it not pay for people to purchase bednets or screens or drugs to prevent or treat malaria for themselves or their children? Our model seems to indicate that the disease should have little macro impact where there are effective protective measures available, even if they are somewhat costly.

Experiment 3:

Consider now the effects of varying the cost of protection from malaria. How large does the cost need to be before it is viewed as effectively unaffordable or undesirable (as in Experiment 1)? Figure 2 graphs a discrete approximation of the relationship between protection costs and the steady-state levels of output, consumption, assets, and the proportion of people sick and protected. As the protection cost rises, steady-state output falls in a weakly monotonic fashion.

A crude rule of thumb is that, for values of q less than one year's average income, essentially everyone in the model economy purchases the preventive good and buys protection from the disease. For costs much higher than one year's average income, some people opt not to purchase protection. Typically, these are individuals who have accumulated little capital and have had bad draws of the persistent idiosyncratic shock. For costs greater than twice the steady-state average income, essentially no one buys protection, including the "lucky rich." As a result, the economy faces the full force of the disease.

Thus, for malaria to have a big impact on income per capita, it must be true either that (*a*) there is not a truly effective bundle of preventive goods or actions; or (*b*) people are not aware of the effectiveness of the preventive goods; or (*c*) the cost of the preventive goods or actions is very high – in excess of one year's annual income. The model suggests strongly that a moderate charge for bednets or spraying or drugs, if these prevention and control measures were truly effective,

would not deter people from purchasing these goods, simply for their private benefits.

Panel (d) of Figure 2 shows the proportions of people sick and protected as a function of the cost of q. Clearly the proportion sick rises as q increases, while the proportion buying protection falls (consistent with the law of demand).

Are the results of Experiment 3 driven by specific parameter values? Figures 3 and 4 demonstrate the robustness of the basic results to changes in the impact of the disease on the full range of parameters. It is striking that the qualitative results survive reasonably large changes in the parameter values.

Experiment 4:

Experiments 2 and 3 seem to raise a puzzle. Why is it that in actual malarial economies, relatively few people seem to use the protection measures that are available? In most countries, bednet use is very low, and the private demand for indoor residual spraying is even lower. In the model economy, by contrast, people seem willing to pay for preventive measures even when they are expensive. Why do individuals in malarial countries not take greater advantage of the available preventive goods? One hypothesis might be lack of information; another might be limited availability of the necessary items. But in most malarial countries, the basic preventive goods are widely available, and they are well understood, since government and non-governmental programs have been promoting their use for many years and, in some cases, even giving them away.

In this experiment, we ask whether a possible explanation might arise from limited efficacy. Hitherto, we have assumed that an individual who buys the preventive bundle is fully protected for life. In reality, however, the available protective goods are far less than one hundred percent effective.

Our model predicts that if the bundle of preventive goods is less than fully efficacious, there will be a dramatic reduction in the fraction of people purchasing protection. The intuition behind this result is simple enough; the only thing worse for people in the model than getting sick would be to get sick after buying the preventive good.

Figure 5 shows the rapid drop-off in the fraction of people purchasing protection, for the cases where the protective bundle is less than fully effective. It is striking that even a relatively modest loss of efficacy would have large impacts on the economy. For example, at the benchmark level of prevention cost – of q equal to approximately one-fourth of annual income – a reduction from 100 percent efficacy to 99 percent efficacy would have a very small but measurable impact on the fraction of the population purchasing protection. At a higher prevention cost of q equal to two years' income, however, a reduction from 100 percent efficacy to 99 percent efficacy would induce a decline of ten percentage

points in the fraction of people purchasing prevention. A decline to 95 percent efficacy would reduce by half the number of people purchasing protection.

The effects on steady-state output would also be large. Figure 6 shows how steady-state output would be affected by decreases in the efficacy of the preventive good, holding all else constant. Again, at the benchmark prevention cost of one-fourth of annual income, a decline from perfect efficacy to 99 percent efficacy would reduce steady-state output by about seven percent. With q equal to two years' income, a decline from perfect efficacy to 99 percent efficacy would lead to approximately a ten percent drop in steady-state output. With $q = 2$, a decline to 95 percent efficacy would reduce steady-state output by about one-third.

Although the model is clearly stylized, the analysis of efficacy has potentially important implications for policy. Where malaria prevention and control methods are less than fully effective – and the measures available today most probably have lower rates of effectiveness than the numbers analyzed here – it is to be expected that take-up rates will be very low, given any significant costs. Low take-up rates would be rational in this case, rather than reflecting ignorance or lack of information.

6. Conclusions

These results point to several notable conclusions. First, it is entirely possible for an economy to arrive at a "malaria trap," in which sickness begets poverty and poverty makes disease prevention unaffordable. In the model economy, we can quantify the magnitude of this "malaria trap." It can reduce income per capita by about half. By point of comparison, Gallup and Sachs (2000) note that the 44 countries with intensive malaria burdens in 1995 had per capita income of $1,526, compared with $8,268 for the 106 countries without intensive malaria burden. Our model suggests that the disease alone could account for just under half of this income gap.

Economies in this situation could certainly benefit from being helped across the disease threshold into an alternate steady state in which the disease is essentially eradicated. The problem, of course, is that this intervention may be very expensive, in the model economy as well as in the real world. A related problem is that efficacious preventive measures may not exist. However, in the model economy, where prevention is available, people will be willing to pay considerable amounts for it – assuming efficacy.

Where the costs of the disease are not large, however, a striking result of the model is that the private incentives are powerful enough that people will bear the cost of protection. For costs that are modest – as appears to be true in reality for

bednets and spraying, and as may eventually be true for a vaccine – people in the model economy purchase protection. Costs must be large, relative to average annual income, before they affect the take-up of the preventive good. However, if he preventive good is not fully effective, there will be powerful impacts on take-up and on real outcomes.

Another point worth noting here is that all of our analysis looks at private responses to malaria. But we know that decentralized outcomes in this economy are not optimal, because there are important infection externalities operating. Individuals in our model economy do not weigh in their decisions the potential impact of their actions on others. In particular, they are likely to under-invest in prevention relative to the social planner's optimum. This suggests that there may be a significant role for the public sector to undertake campaigns of prevention and/or treatment.

A few final points deserve reflection. First, utility comparisons across steady states in this model are complicated. Many more people are born and die in the steady states where people are poor and sick, and it is difficult to know what utility weight to assign to births and deaths. A simple comparison of income levels for the living is inadequate.

In the same vein, it is important to note that the ultimate justification for investments in malaria control and treatment is the welfare cost, rather than the reduction in steady-state income per capita. Even if we found that the impacts on steady-state income were small, there are many other reasons why we should care about malaria and the enormous and tragic harm that it does. For hundreds of thousands of families, malaria is killing their infants and children. In many other families, the disease interferes with daily life, including schooling. Whether or not these effects are important for national income, they matter deeply to the individuals and communities that are affected. We do not need to justify malaria control programs on the grounds that they will contribute to GDP or to GDP growth. This must be one ingredient of our thinking, but the moral imperative alone is surely sufficient to justify some efforts for prevention, control and treatment.

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Table 1: Experiment 1 Results (Multiple steady states with prohibitively expensive protection).

q = 1000 z = 0.7	Low	High
Endogenously determined fertility rate	0.069	0.0150
Proportion sick	0.9007	0.0000
Proportion protected from disease	0.0000	0.0000
Average assets	2.9596	12.0797
Average output	1.3913	2.4521
Average consumption	1.1565	2.2668

Table 2: Experiment 2 Results (Multiple steady states with feasible but costly disease protection).

q = 0.6 z = 0.7	Low	High
Endogenously determined fertility rate	0.015	0.0150
Proportion sick	0.0006	0.0000
Proportion protected from disease	0.9770	0.0000
Average assets	12.0631	12.0797
Average output	2.4488	2.4521
Average consumption	2.2553	2.2668

Figure 1. Distributions of individual asset holdings in benchmark economy, steady states with and without malaria.

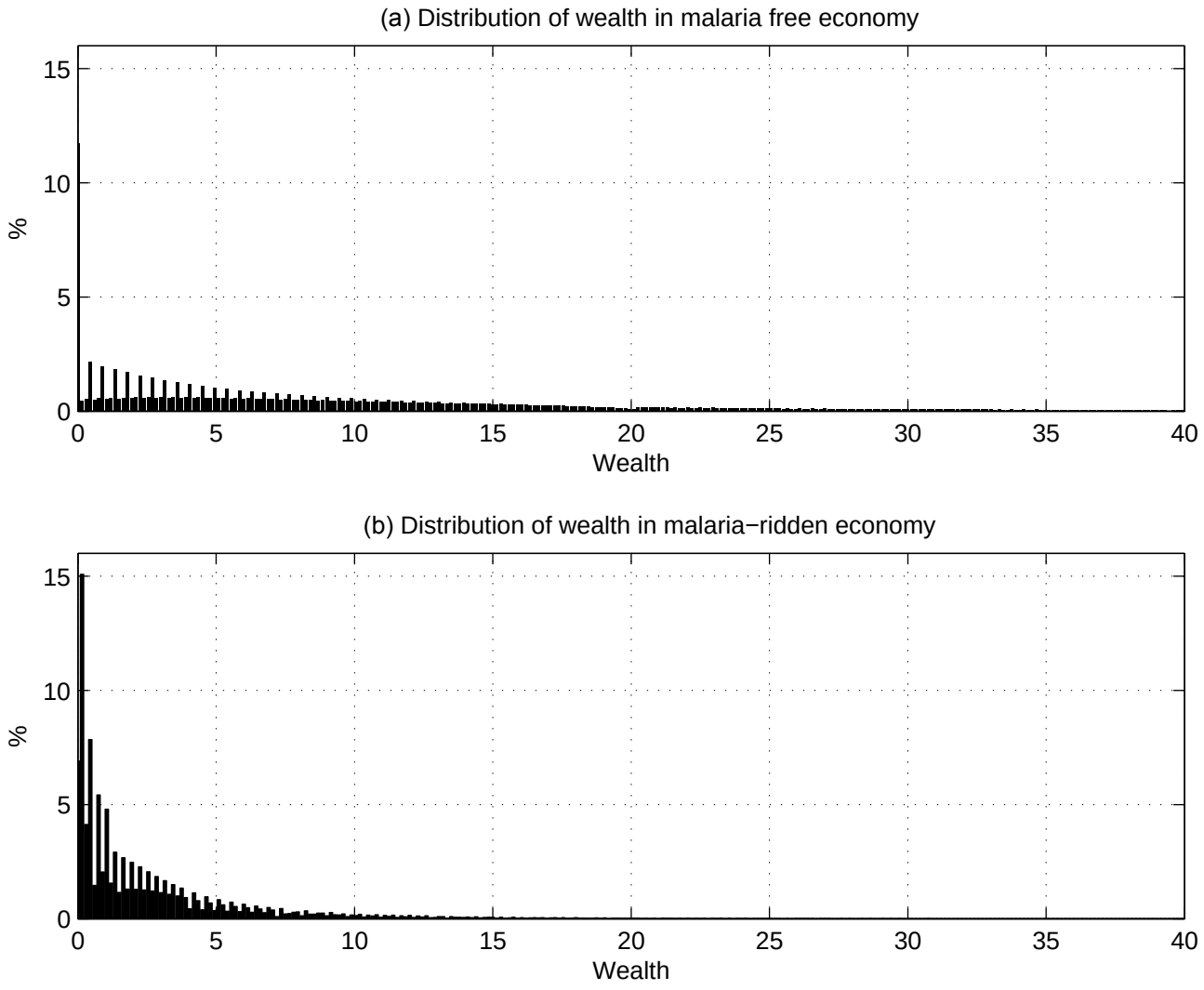
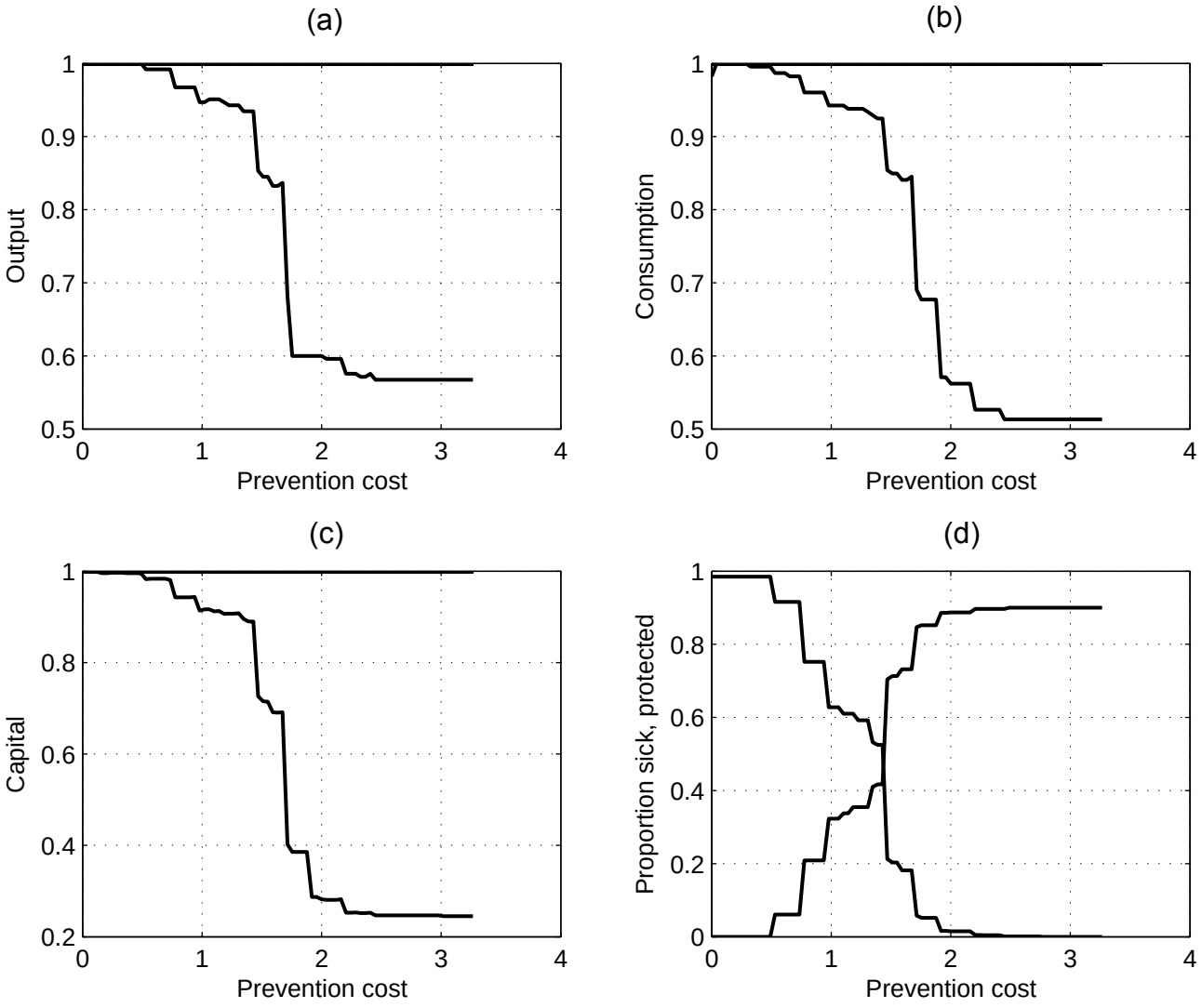
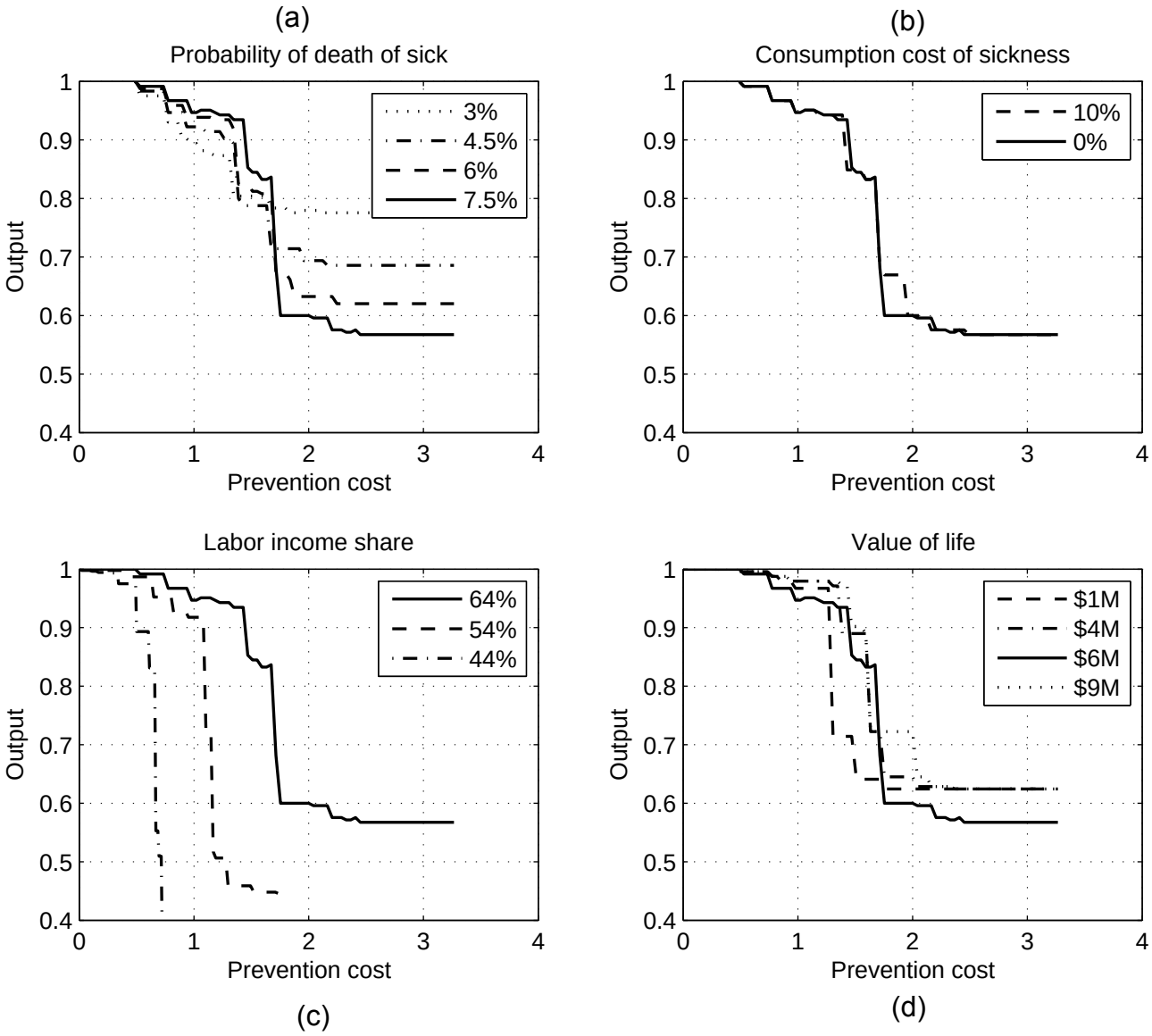


Figure 2: Benchmark economy, showing response of key variables to changes in the cost of protection from disease, in "sick" and "healthy" steady states.



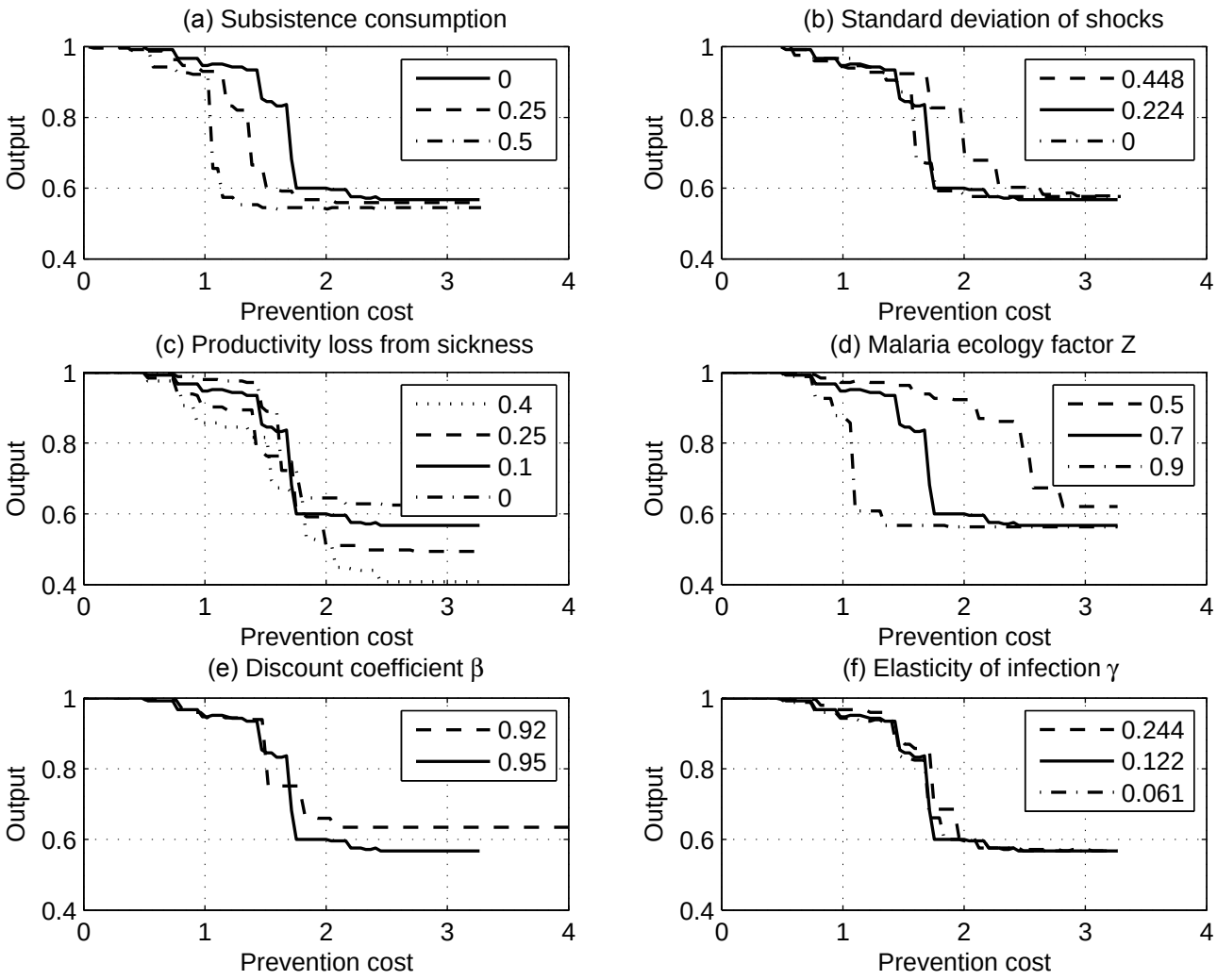
Prevention costs are measured relative to average income in the malaria-free economy.

Figure 3: Robustness checks -- sensitivity of the model to changes in parameter values.



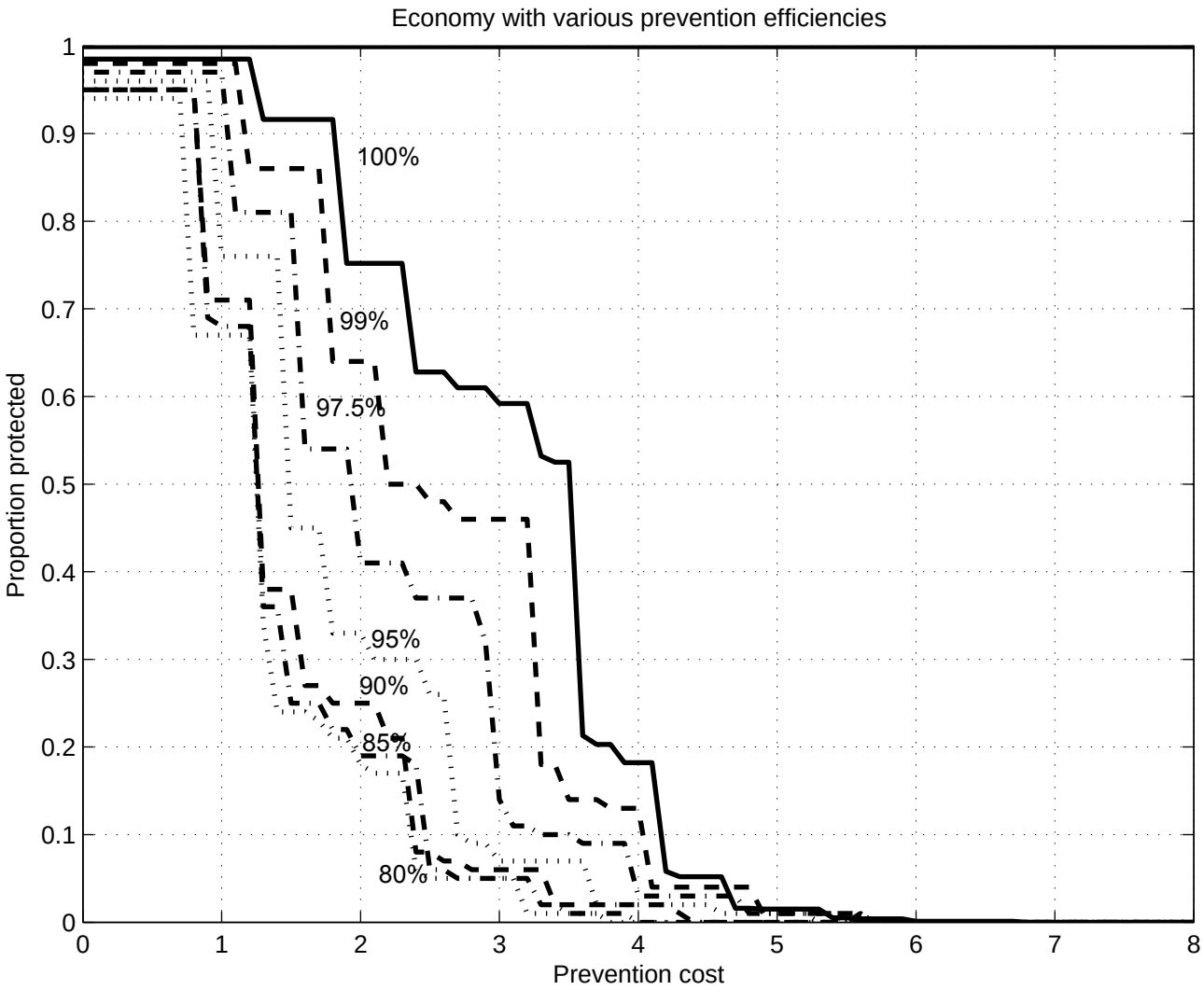
Prevention costs are measured relative to average income in the malaria-free economy.

Figure 3: Robustness checks -- sensitivity of the model to changes in key parameter values.



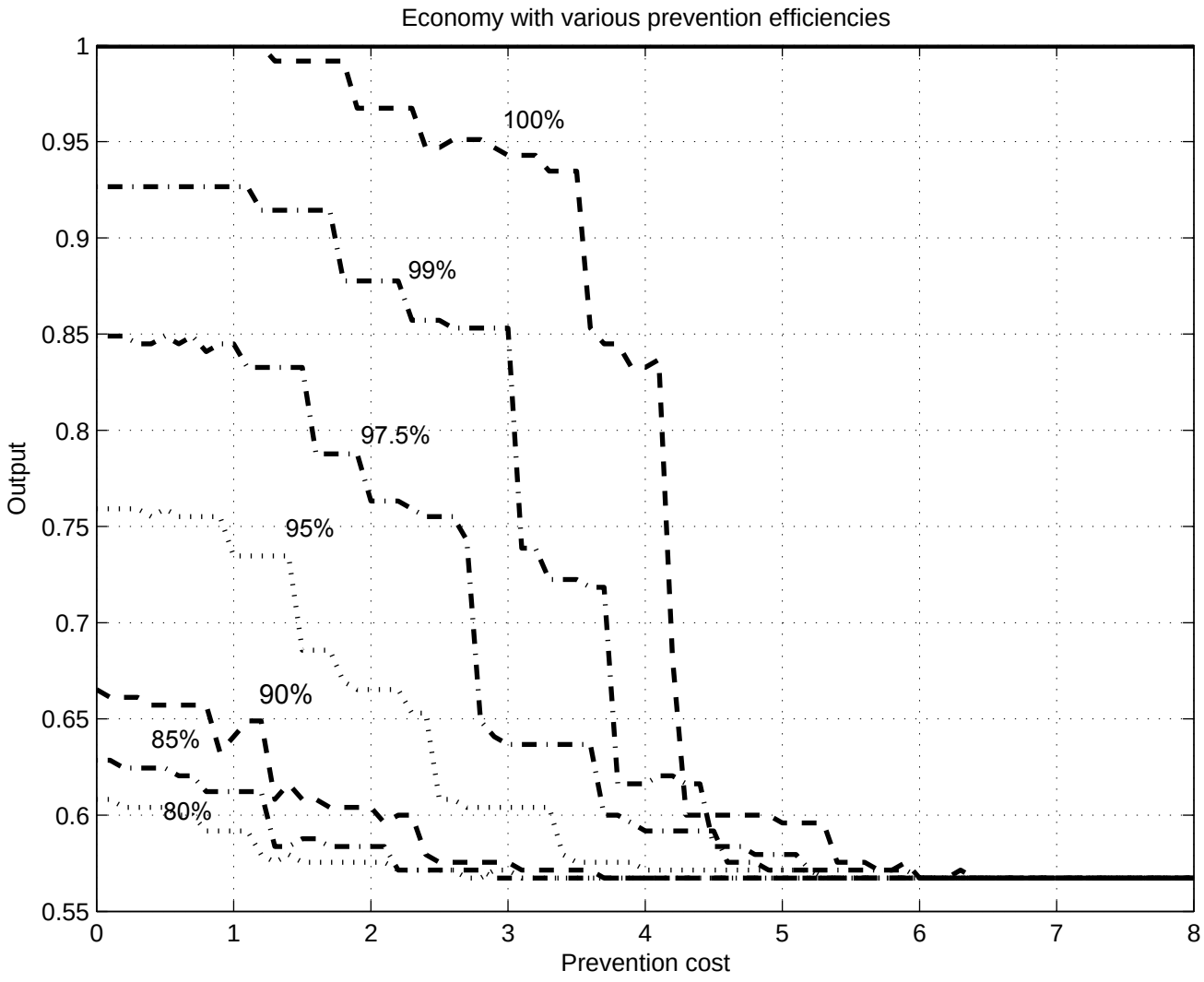
Prevention costs are measured relative to average income in the malaria-free economy.

Figure 5: Proportion of people who have purchased protection in steady-state, for economies differing in the degree of efficacy of the preventive good.



(relative to income in the malaria-free economy)

Figure 6: Output per person relative to benchmark economy,
for economies differing in the degree of efficacy of the preventive good.



(relative to income in the malaria-free economy)