

Global Climate Change and the Resurgence of Tropical Disease: An Economic Approach*

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Abstract

How will global climate change affect the prevalence of tropical diseases? In general, warmer temperatures will expand the areas in which these diseases are endemic. However, if households can take actions to protect themselves from the disease – such as purchasing bednets or insecticidal sprays – then economic factors may greatly mitigate the effects of climate change. These actions are costly, however, and particularly in poor countries, many households face borrowing constraints. The household's objectives and constraints can be combined with a standard model of disease transmission in a calibrated dynamic general equilibrium model. In this framework, it appears that a temperature increase of 3°C will induce modest changes in disease prevalence and output. These effects can be mitigated by improvements in the efficacy of disease prevention.

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1 Introduction

Public health officials often use the term “tropical diseases” to refer collectively to a list of infectious diseases that are found primarily in developing countries. These include malaria, schistosomiasis, dengue, trypanosomiasis, leprosy, cholera, and leishmaniasis, among others. Many of these diseases are spread by insect vectors, and all of them disproportionately affect the world’s poor. Malaria is the most severe of these, with the World Health Organization (WHO 2008a) estimating that the disease causes about 250 million episodes of “acute illness” and perhaps 880,000 deaths annually.¹ But other diseases, less well known and sometimes described as “neglected,” also impose grave burdens on people living in the tropics. The World Health Organization estimates that “neglected tropical diseases” affect over one billion people each year and cause about 570,000 deaths annually (World Health Organization 2009). For example, some 200 million people are currently infected with schistosomiasis, a parasite that is transmitted through poor sanitation.² Perhaps 50 million cases of dengue occur each year, of which 500,000 lead to devastating hemorrhagic fever, with 22,000 resulting deaths.³

What makes these diseases “tropical”? The diseases themselves are perfectly capable of infecting people in all climates, but they are transmitted by vectors that are most common in hot environments: mosquitoes (malaria and dengue); biting flies (trypanosomiasis and Chagas disease); sandflies (leishmaniasis); and freshwater snails (schistosomiasis). As Masters and McMillan (2001) have pointed out, most of these vectors do not fare well in temperate zones that are susceptible to frost. Regular seasonal frost does not eliminate the vectors, but it does appear to interfere with cycles of transmission, making it harder for the diseases to sustain high levels of infection.

If cold weather is responsible for suppressing the vectors that transmit tropical diseases, how will climate change alter the current distribution of these diseases? Will malaria and other tropical diseases sweep through rich countries? At present,

¹Reported by WHO on the “Roll Back Malaria” program website at: <http://malaria.who.int/wmr2008/malaria2008.pdf>.

²Of these, several million display serious health consequences. (Source: <http://www.who.int/schistosomiasis/en/index.html>.)

³Dengue is also perhaps the fastest-growing of the tropical diseases, with a recent dramatic increase in the number of cases observed around the world. See <http://www.who.int/csr/disease/dengue/impact/en/> for further information.

infectious disease is a relatively minor source of the disease burden in today's rich temperate-zone countries. Will this change in future? Detailed studies (for example, Martens et al. 1997) suggest that large geographic areas may become susceptible to epidemic levels of malaria under plausible climate change scenarios, and they note that people in affected areas may not have natural immunity to the disease, so that mortality rates could be quite high.

Summarizing the available literature, the Intergovernmental Panel on Climate Change (IPCC) has specifically investigated the changing burdens of infectious disease that may result from climate change. One IPCC background report notes that:

Climate plays a dominant role in determining the distribution and abundance of insects and tick species – directly, through its effects on vector and parasite development, and indirectly through its effects on host plants and animals and land-use changes.... Therefore, it is anticipated that climate change will have an effect on the geographical range and seasonal activity of vector species and, potentially, disease transmission...

The IPCC studies soberly suggest that the capacity of medical and public health systems in most rich countries are sufficient to prevent tropical diseases from reaching epidemic levels, under the assumed range of warming that is expected to accompany climate change. The IPCC is more cautious in discussing climate change impacts on disease prevalence in poor countries, where the diseases are already widespread. However, some observers argue that there could be large impacts on populations in areas that are currently marginal for malaria and other tropical disease. One scholar argues that "...[A] warming and unstable climate is playing an ever increasing role in driving the global emergence, resurgence, and redistribution of infectious diseases" (Epstein 2004, p. 383). Strong concerns are also expressed in Parry et al. (2001), who summarize some of the background research for the IPCC studies and suggest that a 3^o C increase in temperatures could cause as many as 200 million additional people to live at risk of malaria by 2080.

How will climate change alter the distribution and severity of tropical diseases? Will increasing temperatures lead to the spread of mosquitoes and other insect vectors and thereby expand infectious diseases to the previously "safe" countries of the temperate zone? Or will the biological impacts of climate change be mitigated by

human adaptation and behavioral responses? If both effects are present, how will they be balanced?

The answers fundamentally rely on human behavioral choices – at the individual level as well as at the level of government policies. Although climate change may affect the spatial distribution of vectors and pathogens, human exposure to disease is not a mechanical function of environmental conditions. In many or most tropical diseases, either the vector or the pathogen must spend at least a portion of its lifecycle in humans. This means that humans affect the level of disease prevalence through their choices about prevention and treatment of the diseases.

Given that human choices are critical, it is helpful to analyze the problem using a model in which individuals make conscious and rational choices that affect their disease exposure and infection status. Individual choices may not be socially optimal: there are infection externalities operating in this environment. Nevertheless, a useful starting point is the competitive equilibrium of a dynamic general equilibrium model in which people face a risk of infection from diseases in each period. People in the model economy make choices that affect their exposure to the disease. This corresponds to the real-world choices that individuals and families make concerning purchases of drugs, bednets, or other goods that allow them to prevent or treat the diseases that they face.

The effects of climate change on disease prevalence can be modeled as changes in a parameter that describes the “ecology of disease” in the model. This parameter governs the ease with which the disease can be transmitted from one person to another. Each level of the parameter corresponds to a steady-state level of infection and output within the model economy.

A number of other recent economic studies have attempted to model the impact of climate change on disease and related outcomes. Bosello, Roson, and Tol (2005) use a computable general equilibrium model to assess the health and economic impacts of climate change, based on the GTAP model of global agricultural trade. This research does not specifically target the countries most vulnerable to malaria; it instead offers a global perspective with particular focus on the multiple agricultural sub-sectors that are modeled under GTAP. The model is also static, and it does not explicitly incorporate individuals’ behavioral responses to the presence of disease.

Another related work is an econometric analysis by Tol, Ebi, and Yohe (2007), who

model the health and development impacts of climate change in sub-Saharan Africa. Their approach treats health outcomes as the dependent variable in an analysis in which economic growth and climate are both changing. Their research relies on a directly empirical framework in which regressions are used to construct elasticity estimates for countries' vulnerability to climate change, and then various scenarios are analyzed using these elasticities. Tol and Dowlatabadi (2001) report the results of simulations carried out using an integrated assessment model in which emissions reductions from OECD countries are costly, leading to reductions in income levels in the OECD and — via mechanical spillovers — in the rest of the world. These income losses increase morbidity and mortality in the model, which partly offsets the improvements that would come from reducing emissions and controlling climate change.

2 The Model

A useful model of climate change and its impact on disease must have certain features. For example, it must represent infection through some plausible process. Disease should affect people's choices and perhaps also their resources and abilities. And it is useful to consider actions that people can take that alter their exposure to the disease — either by avoiding infection or by treating (or curing) infections once they occur. In short, to understand the impact of climate change on the prevalence of a human disease, it is essential to take into account how people react and do something about the environment they live in. Thus, a model needs the following components:

1. agents that react to the disease and economic environment;
2. an economic environment that is influenced by the prevalence of the disease;
3. a law of motion for the disease that is at least partially influenced by humans;
4. economic constraints that potentially prevent an eradication of the disease;
5. heterogeneity among agents, as only some carry the disease, and not all face the same economic constraints;

6. general equilibrium effects, as the prevalence of the disease does not only affect the ailing agents, but also the healthy ones through prices and potential exposure to the disease.

These ingredients can be combined in a dynamic stochastic general equilibrium model with heterogeneous agents, following Gollin and Zimmermann (2010). This is a model of perpetual youth, where agents stochastically catch a disease, and die with probabilities that depend on their health status. The likelihood of falling sick depends on the proportion of people already sick, as well as on ecological factors that can change with climate change. Once sick, people remain so for the rest of their lives and suffer productivity losses because of absences from work or diminished abilities.

However, economic agents have access to a technology that can at least partially protect them from getting infected. This technology is costly and needs to be paid for in lump sum fashion before it is used. This might correspond to a vaccine, or alternatively a long-lasting bednet or a set of screens for a house. The fixed cost of this preventive measure poses a problem for individuals in the economy, as agents do not necessarily have the funds available for this purchase, especially at the beginning of their economic life. Indeed, borrowing is not possible in this economy.

Agents earn income in two ways. First, they work and obtain a wage that corresponds to their marginal productivity of labor. The latter is influenced by their health status, an idiosyncratic and persistent productivity shock and the overall marginal productivity of labor. Second, they earn interest on their savings, at the overall marginal productivity of capital. Factor prices are determined from a production function that uses aggregates of efficient labor units and savings across the entire model economy.

There is a unit measure of individuals in this economy, and they maximize their lifetime expected discounted utility subject to a budget constraint and a liquidity constraint:

$$\begin{aligned}
& \max_{\{c_{it}, k_{i,t+1}\}_{t=0}^{\infty}} && \sum_{t=0}^{\infty} \beta^t (1 - d(h_{it})) u(c_{it}), \\
& \text{s.t.} && c_{it} + k_{i,t+1} + p_{it}q \leq w_t h_{it} \pi_{it} + r_t k_{it}, \quad \forall t, \\
& && k_{it} > 0, \quad \forall t,
\end{aligned}$$

where, for agent i in period t , c_{it} is consumption, k_{it} is capital, p_{it} is a binary decision to purchase protection against the disease, h_{it} is the health status and π_{it} is an idiosyncratic productivity shock. $u(\cdot)$ is a utility function with the usual properties, β is the discount rate, $d(\cdot)$ is the probability of death, q is the price of health protection, w_t is the efficiency wage and r_t is the return of capital. Idiosyncratic productivity shocks π_{it} follow a persistent binomial Markov process.

Individuals are born healthy and without assets, replacing those who just died. Within each period, an unprotected individual may catch a disease with probability s_t . Being sick is an absorbing state and entails a loss in productivity, so that $h_{it} = \bar{h} < 1$ instead of $h_{it} = 1$.

At the aggregate level, efficiency units of labor and individual capital are summed and deployed in a standard production function:

$$\begin{aligned} L_t &= \sum_i h_{it} \pi_{it}, \\ K_t &= \sum_i k_{it}, \\ Y_t &= F(K_t, L_t). \end{aligned}$$

Factor markets are competitive in this economy, which implies that factor prices correspond to the respective marginal products:

$$\begin{aligned} r_t &= \frac{\partial F(K_t, L_t)}{\partial K_t}, \\ w_t &= \frac{\partial F(K_t, L_t)}{\partial L_t}. \end{aligned}$$

Similarly, the prevalence of the disease is measured by the proportion of sick people:

$$S_t = \sum_i \mathbb{I}_{h_{it}=\bar{h}}.$$

This translates into the probability of catching the disease s_t by combining S_t with an ecological factor Z and the efficacy of protection e , if previously purchased ($v_{it} = 1$):

$$s_{it} = \begin{cases} ((1 - v_{it}) + v_{it}e) f(Z, S_t) & \text{if } f(Z, S_t) > \bar{s} \\ 0 & \text{if } f(Z, S_t) \leq \bar{s} \end{cases}$$

Below a threshold \bar{s} , a disease is not sustainable and vanishes.

This model has at least one steady-state equilibrium. A recursive steady-state equilibrium is defined in this economy by decision rules for consumption, capital and protection purchase; prices for labor and capital; distributions of agents over health status and capital holdings; and aggregate labor, capital and disease prevalence such that individuals optimize given prices and aggregates; firms maximize profits; factors markets clear efficiently; distributions are ergodic; and individual decisions are consistent with aggregates.

There is always at least one equilibrium, a disease-free one. If the threshold $\bar{s} = 0$, there is another one with some disease prevalence. Indeed, even if everyone buys protection, newborns can still catch a disease in the first period and perpetuate it. If the threshold is higher, such an equilibrium may not exist.

3 Calibration

To obtain quantitative answers to the questions posed above, the functional forms and parameter values for the model economy must be fully specified. The results below are based on a calibration that follows Gollin and Zimmermann (2010).

The production function is the standard Cobb-Douglas formulation:

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

Similarly, the utility function is a standard power function,

$$u(c_{it}) = \frac{(\gamma c_{it})^{1-\rho}}{1-\rho}.$$

In this specification, γ is a multiplier that captures the value of life. Without it, utility may be negative, and agents may prefer death, which has zero utility.

Finally, the probability that a person will become infected with the disease, conditional on being unprotected, is given by a functional relationship between the disease ecology Z and the fraction of individuals in the economy who are sick, S :

$$f(Z, S_t) = Z S_t^\mu,$$

where $0 < Z < 1$ and $0 < S_t < 1$; in addition, μ is strictly positive.

For parameterization of the model, a time period is set to be one year. Correspondingly, the discount rate β is set to 0.95 and $\rho = 1$, implying logarithmic utility. The calibration of γ is based on the observation by Viscusi and Aldy (2003) that the value of life in the US is about \$7 million, or 11.3 times lifetime consumption. In line with this observation, γ is set at 11.3.

The productivity loss due to sickness is set to 10% in the model economy, in keeping with Bleakley's work on malaria (2003). This is a middle-of-the-road estimate from a wide distribution of micro studies in the literature. For some other diseases, infected people may be severely debilitated and may suffer from greater productivity losses. Others, because they cause only sporadic ill effects, may have smaller effects on lifetime income. A 10% reduction in productivity corresponds to $\bar{h} = 0.9$.⁴

Domeij and Heathcote (2004) suggest a calibration of the binary process for the idiosyncratic productivity shocks π_{it} . Shocks are $\pm 22.4\%$, with a 90% chance of repeating the following year. The annual death rate is set to 1.5% for healthy people and 7.5% for sick people. The fertility rate is set to keep population constant, as required by a steady-state.

The model also requires parameters that relate to the efficacy and cost of the protective goods. In the model, the preventive good is purchased one time and then provides continued protection for a lifetime. Given the borrowing constraint, this would appear to impose strong limitations to the availability of protection. Yet Gollin and Zimmermann (2010) show that this cost must rise to 1.5 times the average annual income to prevent everyone in the economy from purchasing prevention as soon as they can possibly afford it. People in the model economy display a strong desire to protect themselves from disease. This desire would be even greater if the private cost of being sick – measured in terms of lost earnings – were greater; that is, for a lower value of \bar{h} .

For the simulations presented here, the cost of the preventive good is set at one quarter of the annual average income in a disease free economy. As discussed pre-

⁴Setting the productivity loss higher does not necessarily increase the impact of the disease in equilibrium; instead, as the disease becomes more costly to individuals, there are greater incentives for people to purchase protection. In equilibrium, this may decrease the fraction of the population infected, leading to a reduction in the aggregate costs of the disease. This effect is considered in the sensitivity analysis of Section 4.1.

viously, considerable variation in this cost will not affect results. Although people do not need to purchase the preventive good again, the protection that it provides may be imperfect. For example, a proposed malaria vaccine is expected to be less than 50% effective, and mosquito nets or screens also provide only limited protection from disease (perhaps 60-70% when used consistently). Combining different forms of prevention may increase the efficacy further, but the protection will still be less than perfect, and the costs of multiple measures will of course be higher. In the analysis that follows, the efficacy of protection is set to different values between 70% and 90%.

The model also requires two parameters that characterize the infection process. First, the malaria-ecology index of Sachs et al. (2004), rescaled to the unit interval, offers a measure of the disease ecology, Z . Second, the elasticity μ can be estimated by regressing observed infection rates on the malaria ecology. This yields a value of $\mu = 0.122$.

Finally, the model has a threshold value of prevalence, \bar{s} , below which the disease will die out; once the fraction of sick people falls below \bar{s} , the number of people infected in each period will be smaller than the number of infected people who die. As a result, the proportion of sick will go to zero, with eventual eradication. Unfortunately, the literature is not clear about whether eradication is feasible for most tropical diseases. For some diseases that have non-human alternate hosts, eradication in humans is not sufficient to lead to eradication of the disease. Even for diseases without alternate hosts, eradication may not be feasible except in theory. In the case of malaria, for example, few organizations are willing to commit to any hard numbers for thresholds. Those that do disagree: World Health Organization (2008b) claims a country may be ready for a concerted eradication effort if the annual infection rate falls below 0.1%. Others claim it needs to be significantly lower than that. Because of this uncertainty, the sensitivity analysis considers a range of different values, starting with a value of zero (no threshold effect).

4 Quantitative Analysis and Results

In the model economy, an increase in temperature will be represented by an increase in the malaria ecology parameter, Z . In the original index developed by Sachs et al. (2004), temperature is one of several variables used to construct the index value.

Because Z is a re-scaled version of Sachs's index, it is not surprising that there is a strong positive relationship between country Z -values and average annual temperatures for 1972-2001, especially for countries with values of $Z > 0.3$, as shown in Figure 1.⁵

To simulate the effect of climate change, the model economy as calibrated above is solved for different values of Z between 0.3 and 1.0 and for different degrees of protection efficacy. In this context, efficacy refers to the ability of the protective bundle to provide protection from the disease. Perfect efficacy of 100% would imply that someone who purchased the protective bundle would never get infected; efficacy of 90% implies that someone purchasing the protective bundle faces a 90% reduction in the probability of infection; and so on. These scenarios consider protection efficacies of 70%, 80% and 90%.⁶ The results are summarized in Figure 6 for output and Figure 3 for the proportion of sick people.

For output, Figure 6 shows that for a given level of protection efficacy, a higher level of Z implies lower steady-state output per person; hence, each of the curves shown in the figure is downward sloping. Higher levels of efficacy also lead to increases in output per person (shifting the curves as illustrated). Climate change here can be viewed as a movement along an iso-efficacy curve.

The figures suggest that the consequences of climate change may be relatively modest in this model economy. Similar patterns are evident in the effects of an increase in temperature on disease prevalence. Figure 3 shows the relationship between infection rates and Z for the same three different levels of efficacy. The differences are not large, in a qualitative sense.

What do these numbers mean for climate change of the magnitudes currently predicted by climate models? Climate change is estimated to increase average world temperature by 1 to 6° C. Taking 3° C as a benchmark, this would effectively give New York City a climate like that of present-day Washington, DC; Rome would see

⁵Data on temperature were taken from the appendix to Jones and Olken (2010), which are available online at: Climate Shocks and Exports with Ben Jones. American Economic Review Papers and Proceedings, forthcoming 2010. Data and programs at: <http://econ-www.mit.edu/files/5131>. The temperatures used here are based on detailed observations for different geographic cells within countries; the country averages are based on population-weighted averages of these temperatures.

⁶At a protection efficacy of 100%, aggregates are unaffected as everybody but a tiny proportion of the population is immune, and changes in the ecological environment have a minimal impact. In any case, 100% is by far not realistic with current technologies.

its average annual temperature rise to that of present-day Algiers. In terms of malaria ecology, such a temperature change would correspond to an increase in Z of 0.192, based on the relationship shown in Figure 1.⁷

Table 1 shows that the impact of climate change through disease is relatively modest within this range. Across the three efficacy scenarios, the change in output corresponds to less than a year in real GDP growth; in other words, the model predicts that the macroeconomic effects of climate-induced disease would be overshadowed by business cycle variations. The change in the number of sick people would also be minimal (it is a percentage of a percentage). Locally the impact could be stronger or reversed, depending on changes in rainfall, which are predicted to vary depending on location.

Why is the impact so small? For one, temperature is only one factor in malaria ecology. Also, diseases like malaria may not have that much of an aggregate impact on rich economies, because people are likely to pay for protection. Only if the protective technologies are ineffective or if people are simply unable to afford them would we expect to find large economic impacts.⁸ It should be noted that these results are qualitatively consistent with other estimates that suggest very small impacts of climate change on output per person through the channel of human health. For example, Bosello et al. (2005) estimate the effects of climate change on GDP via the health channel at -0.101% for a region identified as “Rest of World,” which includes much of Africa. Their study also estimates a worldwide figure of approximately 65,000 additional malaria deaths by 2050 due to climate change, which is about 8% of the current level. The model economy described above gives a smaller estimate for the increase in people sick with malaria, but Bosello et al. do not include allowances for the offsetting effects of income growth through savings. In the analysis of Tol et al. (2007), malaria deaths in Nigeria alone might rise by 15,000 in 2030, but they predict fewer than 2,000 additional deaths by 2100, largely due to the mitigating effects of economic growth.

Would climate change make malaria sustainable where it was not before? Seeing the disease prevalence in Figure (3), it seems unlikely that any of the thresholds for

⁷Specifically, this number is derived from a linear regression of Z on a constant and the average annual temperature variable, measured in degrees C. This yielded a highly significant coefficient on temperature of 0.064. For a shift of 3^o C, this gives an increase in Z of approximately 0.192.

⁸However, that there may be large welfare impacts even if GDP per person is not greatly affected.

eradication would cross any of the curves. Although some areas with high malaria potential currently are free from the disease (Singapore, for example), it is not because they are below the threshold. In other words, rich temperate-zone countries will not avoid the burdens of tropical disease by actually eradicating them; instead, the model suggests that the disease burdens will be very modest because people are able to protect and treat themselves easily. As a result, very few people will ever be infected, and transmission will be very low – though not impossible.

The model suggests that climate change will slightly increase the burden of tropical diseases. However, rather modest improvements in protection efficacy could compensate for the consequences of climate change. For example, an improvement in efficacy from 70% to 80% improves GDP by 6–8% and decreases the number of sick people by about 10%, much larger effects than those mentioned in Table 1.

At least some of the improvement in efficacy can come from better uptake of protection. Indeed, in most countries, bednet use is very low, and the private demand for indoor residual spraying is even lower. For example, the World Malaria Report 2010 reports household survey data for many countries in Africa that show substantial variation in the use of bednets and other forms of prevention. In most countries, fewer than half of young children sleep under bednets, even when households own them (WHO 2010, Annex 6A).

4.1 Sensitivity analysis

How sensitive are the results of this analysis to the particular choices of parameter values? Table 2 shows – for a fixed level of efficacy – how the results of the model would change in response to changes in the parameter values. The benchmark scenario here is the same as that in Table 1, and the columns show the effect of a 3^o C increase in temperature on output and number of sick people. In the benchmark, output falls by less than one percent, and the fraction of sick people in the economy rises by 0.32% to 1.62%.

The sensitivity analysis reports the effects of changes in the parameter ρ , which affects the curvature of the utility function and corresponds to risk aversion. Increasing the risk aversion of individuals in the economy to $\rho = 2.5$ has the effect of reducing the effect of warming on both output and disease prevalence. The intuition for this

change is that risk-averse people will sacrifice current consumption more readily to avoid becoming sick. This means that savings rates rise – leading to a comparatively smaller effect on output – and that rates of sickness fall, compared to the benchmark. The same logic applies to the case with $\rho = 0.5$, in which individuals are slightly less risk averse than under the benchmark log utility; in this scenario, there are very slight increases in the effects of warming on output and disease prevalence, compared to the benchmark case.

What happens if the disease has a smaller effect on the labor supply of the sick? As noted above, this might paradoxically increase the effects of the disease by reducing the incentives for individuals to purchase protection. If the disease has no effect at all on labor supply, then $\bar{h} = 1.0$, in contrast to the benchmark case in which $\bar{h} = 0.9$. In this case, the number of sick people rises slightly relative to the benchmark. The big effect, however, is to reduce the incentives for precautionary savings – and through that channel to reduce the capital stock in the economy. As a result, output falls substantially more under this scenario than under the benchmark. But these changes are still modest; the warming now causes a decline in output on the order of 1.8-3.3%, compared with the benchmark decline of 0.1-1.0%.

What happens if the value of a life, associated in the model with the parameter γ , is higher or lower than in the benchmark calibration – which sets it to 11.3 times the value of lifetime consumption? The results are quite robust to substantial deviations from the benchmark value of $\gamma = 11.3$. At $\gamma = 1.9$, the value of a life is approximately one-fifth of the benchmark; and at a value of $\gamma = 17.0$, a life is about fifty percent more valuable than in the benchmark. Interestingly, neither of these changes has an appreciable effect on the results of the model – essentially because the value of life enters the model in a fairly peripheral way. The parameter matters primarily for the disutility of dying. In the model, people seek to prevent the disease because of the loss of lifetime consumption that ensues – but also because malaria is associated with a higher mortality risk, and people place a high value on the risk of death. However, in the quantitative exercise reported here, the *additional* utility loss associated with a higher value of life is too small to induce any consequential change in the model’s outcomes.

Finally, what would happen if disease protection costs are lower than in the benchmark equilibrium? In the benchmark scenario, the cost is set to $1/4$ of the average

annual income in the economy. For comparison, suppose the cost of protection fell to an amount equivalent to one month's income ($p = y/12$). As can be seen in the last row of Table 2, this change also has a relatively modest effect on the model's variables of interest. Because many people already purchase protection, a reduction in the cost of the preventive good mostly allows them to do so earlier in their lives. This does not have much effect on output or sickness.

Overall, the sensitivity analysis suggests that the model is highly robust to changes in the major parameter values. The results are driven strongly by the underlying structure of the model, rather than particular parameter values. When the (private) economic and utility losses from disease are large, people face strong incentives to protect themselves. Climate change does not alter this basic story; it simply exposes more people to higher risks of morbidity and mortality. These people still face incentives to protect themselves, and to the extent that they live in relatively high productivity economies, they will be able to afford protective measures.

5 Conclusion

Will global climate change increase significantly the prevalence of tropical diseases such as malaria? In a heterogeneous agent dynamic general equilibrium model, where households have the opportunity to react to the prevalence of diseases, an increase in temperature of 3° C would lead to a rather modest increase in disease prevalence and very modest disease-induced losses in GDP per person. Because households will acquire protection as soon as they can afford it, the effects of disease will be mitigated substantially.

In particular, it appears that the cost of protection is relatively unimportant in the model economy: households value life sufficiently to absorb considerable expenses in the name of prophylaxis. A more important factor is the efficacy of protection. With current technology, efficacy between 70% and 90% is attainable, and future developments will certainly drive up these figures. The effects of improvements in disease protection will, in this sense, more than compensate for the effect of global climate change. Improvements in the efficacy of disease prophylaxis can potentially have large impacts on welfare.

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Table 1: Effect of a 3^o C increase in temperature on output and number of sick people (in %)

Protection efficacy	output	sick
90%	-0.40 – -0.96	+0.96 – +4.25
80%	-0.26 – -1.22	+0.60 – +3.14
70%	-0.13 – -1.03	+0.43 – +2.36

Table 2: Sensitivity analysis: effects of changes in parameter values on output levels and disease prevalence, under the assumption of 70% efficacy.

Scenario	Output		Sick	
	Low	High	Low	High
Benchmark	-0.13	-1.03	0.43	2.36
$\rho=0.5$	-0.12	-1.20	0.43	2.48
$\rho=2.5$	-0.03	-0.24	0.15	1.04
$\bar{h}=1$	-1.76	-3.31	0.43	2.48
$\gamma=1.9$	-0.14	-1.04	0.53	2.48
$\gamma=17.0$	-0.14	-1.03	0.43	2.48
$p = \frac{y}{12}$	-0.18	-1.08	0.43	2.48

Figure 1: Temperature and Malaria Ecology, Cross-Section Data

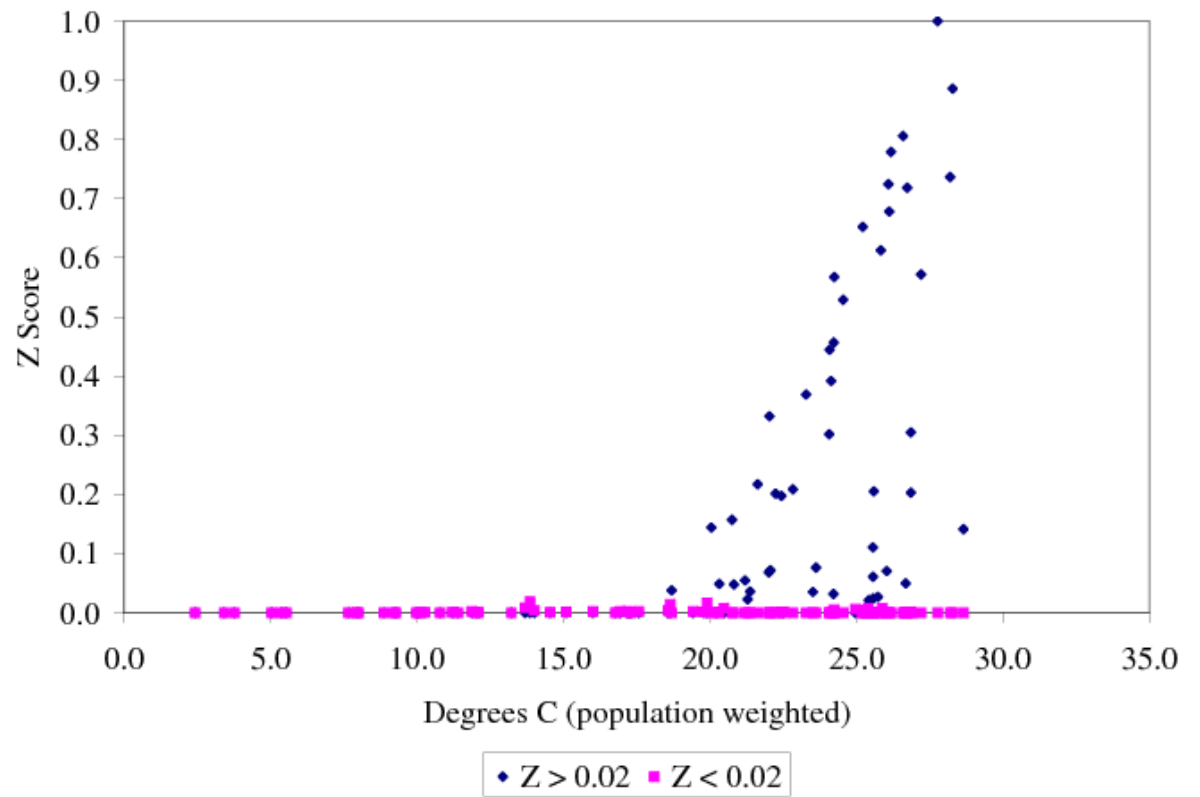


Figure 2: Output levels for different disease ecologies, by efficacy of preventive measures

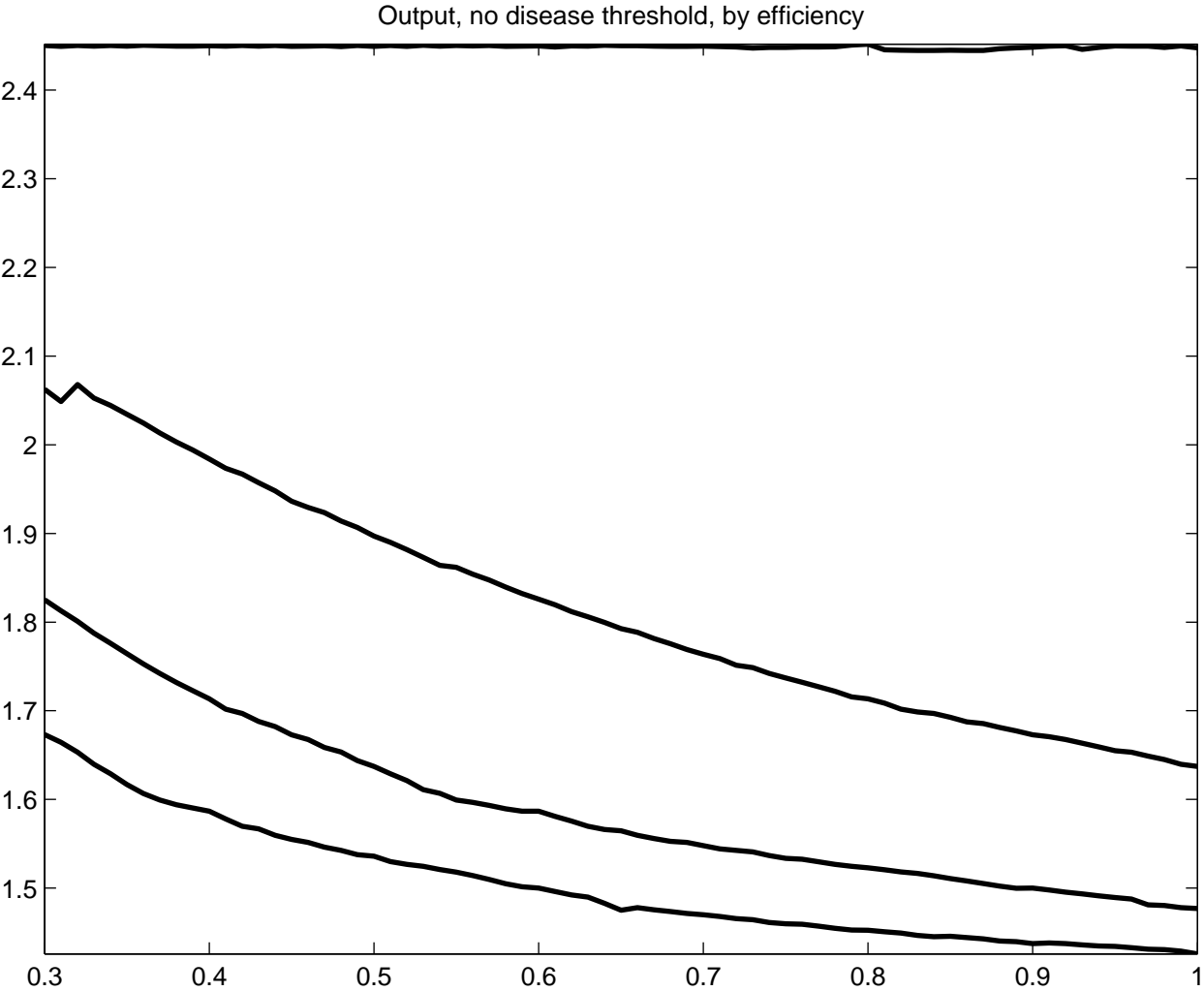


Figure 3: Proportion of the population sick, for different disease ecologies, for varying degrees of efficacy.

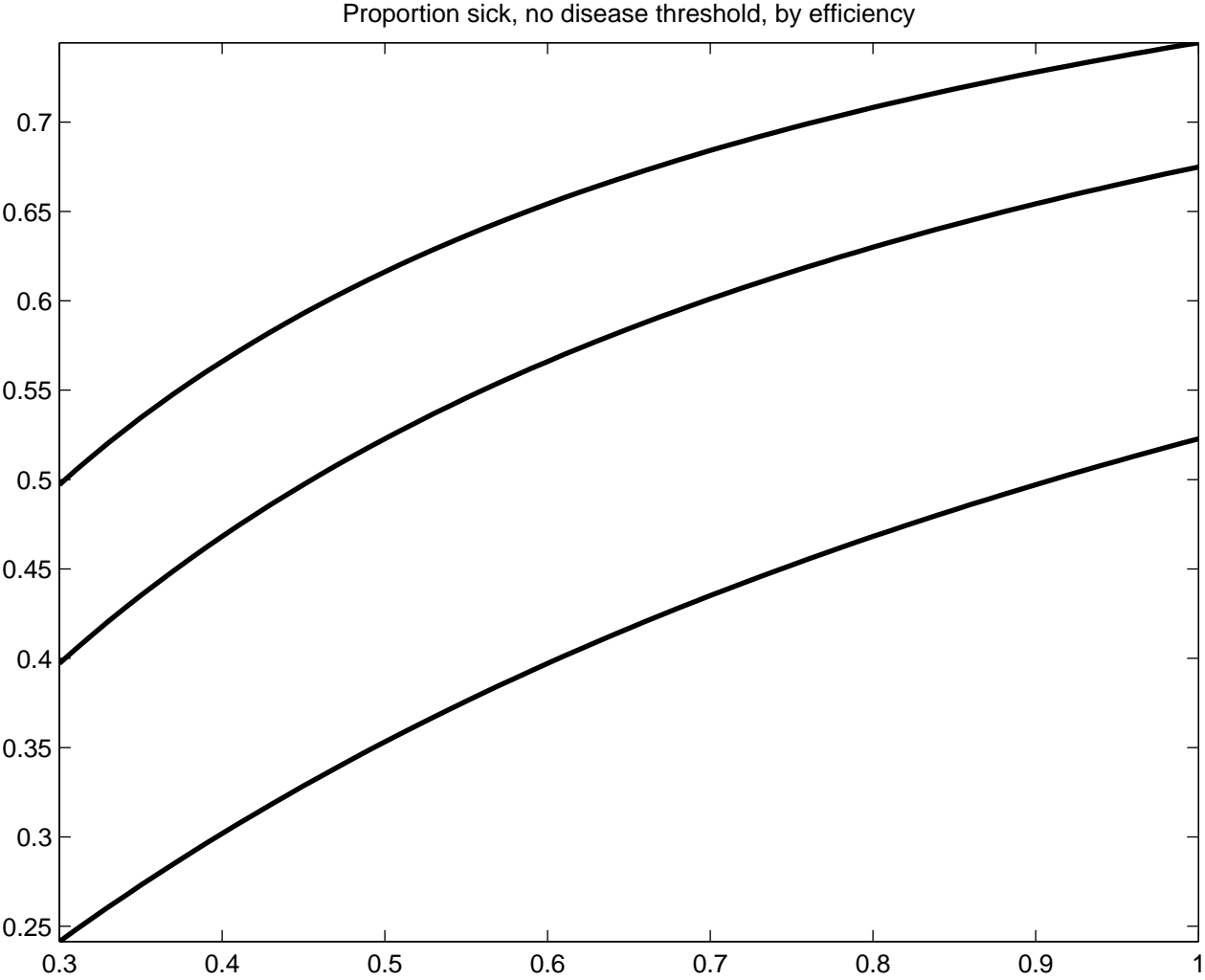


Figure 4: Proportion of population protected, by disease ecology, for different levels of efficacy.

