

# The Economic Epidemiology of Malaria and Economic Growth

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## Abstract

This paper develops an economic epidemiological framework that endogenizes the susceptible population by integrating utility-maximizing individual choices regarding preventive and curative care with aggregate disease dynamics and income growth. Unlike classical biological models, this approach treats the proportion of the population vulnerable to malaria as a behavioral response to prevailing disease prevalence, prevention costs, and the perceived productivity losses of infection. The resulting behavioral margins create a non-linear transmission map characterized by multiple stable equilibria, which can trap economies in a Neo-Malthusian poverty cycle of high endemicity and low productivity growth. Calibrated to sub-Saharan African data, the model highlights a critical “risk compensation” effect, where public vector control is partially offset by a reduction in private preventive investment. Ultimately, the analysis reveals that while single-lever interventions—such as prevention subsidies or awareness education—can reduce equilibrium prevalence, only a coordinated, integrated policy package combining subsidies, education, and aggressive vector control can push an economy across the critical threshold required to achieve a malaria-free equilibrium and unlock higher long-run growth trajectories.

**JEL Classifications:** I18, O4

**Keywords:** Economic Epidemiology, Malaria, Disease Dynamics, Growth Theory, Poverty Trap, Public Health Policy

# 1 Introduction

Epidemiology primarily focuses on the transmission and control of diseases. Individual health-investment behavior is a critical determinant of the speed and scale of disease transmission; such behaviors are typically driven by economic considerations that traditional epidemiological models often overlook. This paper develops an economic epidemiological model that treats health investment as a rational economic decision, employs this framework to analyze how individual investment choices influence aggregate disease and income dynamics, and estimates the broader economic burden of malaria.

Malaria remains one of the most consequential infectious diseases in human history, disproportionately burdening the poorest populations in tropical countries. According to the World Health Organization (World Health Organization (2023)), there were approximately 249 million malaria cases globally in 2022, resulting in an estimated 608,000 deaths, with over 94% of both cases and fatalities concentrated in sub-Saharan Africa. Children under age 5 account for approximately three-quarters of all malaria deaths. Between 2000 and 2015, the scale-up of long-lasting insecticidal nets (LLINs), artemisinin-based combination therapies (ACTs), and indoor residual spraying contributed to a near 50% reduction in malaria mortality (Bhatt et al. 2015). Since then, however, progress has plateaued, and in some areas, these gains have even been reversed. This reversal is attributable to insecticide resistance, disruptions caused by the COVID-19 pandemic, and the accelerating threat of climate change expanding the geographic range of *Anopheles* mosquito vectors (Caminade et al. 2014).

The toll of malaria extends far beyond immediate mortality. Repeated childhood infections induce a cascade of negative externalities, including severe anemia, cognitive impairment, and stunted human-capital accumulation, which depress lifetime earnings for survivors (Bleakley 2010; Aklilu et al. 2025; Snyman et al. 2026). Early empirical work (Barlow 1967; Conly 1972; Kaewsonthi et al. 1988; Newman 1965, 1977) and subsequent syntheses (Hammer 1993; Gomes 1993) documented the substantial private and social costs of the disease. Landmark contributions by Gallup et al. (1998), Gallup and Sachs (1998), and McCarthy et al. (2000) established that malaria exerts a significant drag on aggregate growth, particularly in Africa. More recent causal evidence reinforces this: Bleakley (2010) demonstrates the persistent income effects of early childhood exposure, while Acemoglu and Johnson (2007) link disease environments to long-run developmental divergence. Furthermore, Sachs and Malaney (2002) illustrate how the malaria burden suppresses savings rates and discourages foreign direct investment. Despite this evidence, the precise behavioral and epidemiological mechanisms linking individual health-investment choices to macroeconomic trajectories remain underdeveloped.

This paper bridges this gap by embedding a utility-maximizing model of individual health behavior within a discrete-time overlapping generations (OLG) framework. The central theoretical

departure from the classical epidemiological tradition—exemplified by Kermack and McKendrick (1927) and the Ross-Macdonald framework (Ross 1911; Macdonald 1957)—is the endogenization of the susceptible population. I argue that the fraction of the population susceptible to malaria is not a fixed biological parameter, but a behavioral response determined by the interaction of disease prevalence, the costs of preventive care, and the perceived productivity losses of infection. Drawing on the human capital approach to health investment (Strauss and Thomas 1998), I contrast two paradigms: *biological epidemiology*, where susceptibility is exogenous, and *economic epidemiology*, where it is endogenous. This distinction leads to radically different predictions regarding disease stability and the efficacy of policy interventions.

The analysis reveals the conditions under which malaria-endemic economies fall into a *Neo-Malthusian poverty trap*—a locally stable equilibrium characterized by high disease prevalence and persistently low productivity growth. Furthermore, this trap is reinforced by “risk compensation” (a crowding-out effect), where public vector control can be partially offset by a reduction in private preventive investment, potentially raising the endemic equilibrium. The paper concludes that escaping such a trap requires a coordinated, integrated policy package—combining prevention subsidies, awareness education, and aggressive vector control—rather than reliance on any single lever.

The present work is related to the literature on behavioral responses to disease (Philipson (2000); Francis (1997); Kremer (1996)), but differs by focusing on the periodic nature of malaria and the strategic choice between preventive and curative care. It also complements quantitative transmission models (Griffin et al. 2010) and empirical studies of eradication campaigns (Bleakley 2010) by providing a micro-founded link between individual utility and aggregate growth.

The paper is organized as follows. Section 2 establishes the biological foundations of malaria transmission and the clinical evidence that motivates the model’s key parameters. Section 3 formulates the economic framework, deriving the demand for preventive care and its interaction with disease and growth dynamics. Section 4 presents a calibration to sub-Saharan African data and simulates various policy scenarios. Section 5 concludes with a discussion of policy implications and directions for future research.

## 2 Biology of Malaria Parasites and Health Effects on Human Hosts

To develop a micro-founded economic model of malaria, it is first necessary to identify the biological constraints and clinical manifestations that dictate individual health-investment behavior and aggregate disease dynamics. This section outlines the life cycle of *Plasmodium* parasites and the resulting health complications, providing the empirical justification for the key parameters

used in the subsequent framework.

Malaria is caused by protozoal parasites of the genus *Plasmodium*, which require both a human host and a female *Anopheles* mosquito vector. While several species exist, *P. falciparum* remains the primary driver of mortality in sub-Saharan Africa, while *P. vivax* is the most geographically widespread (White et al. 2014; Cowman et al. 2016).

The transmission cycle is governed by a series of discrete biological stages. First, an infected mosquito inoculates sporozoites into the human bloodstream. These parasites migrate to the liver, where they undergo asexual reproduction (schizogony) over 9 to 16 days. The timing of this process provides a biological basis for the discrete-time formulation of our model, as it creates a natural lag between inoculation and the onset of clinical symptoms. The subsequent rupture of hepatocytes releases merozoites into the bloodstream, which invade erythrocytes and trigger the characteristic bouts of fever and chills.

The asexual replication cycle of *P. falciparum* and *P. vivax* occurs approximately every 48 hours. This periodicity justifies the parameter  $T$ , representing the proportion of time lost to productive labor per malaria episode. Furthermore, the repeated cycle of erythrocyte invasion and rupture causes severe clinical manifestations—including cerebral malaria, severe normocytic anemia, and respiratory distress—which can progress rapidly to fatality, particularly in non-immune adults and children (White et al. 2014; Cowman et al. 2016).

In high-transmission settings, individuals acquire a partial, non-sterilizing immunity known as *premunity*. This immunity attenuates clinical symptoms without fully eliminating the parasite, meaning that while adults may be less likely to suffer severe illness, they remain infectious vectors. For children, however, the burden is higher: repeated infections cause long-term deficits in human capital through school absenteeism and permanent cognitive impairment. These findings provide the empirical basis for the health-capital depreciation parameter  $H$  in the economic model, representing the permanent productivity loss associated with childhood infection.

The current landscape of malaria control involves three primary levers:

**Preventive Care.** Long-lasting insecticidal nets (LLINs) and indoor residual spraying (IRS) reduce the probability of infection by interrupting the vector-human contact. The scale-up of LLINs significantly reduced mortality between 2000 and 2015 (Bhatt et al. 2015). In our model, these interventions shift the probability of infection  $1 - p(i_t)$  and modulate the critical threshold  $\bar{i}$  separating the basins of attraction for the malaria-free and endemic equilibria.

**Curative Care.** Artemisinin-based combination therapies (ACTs) are the WHO-recommended first-line treatment for uncomplicated *P. falciparum*. ACTs rapidly clear parasitemia and reduce the duration of infectivity. In the model, curative care reduces the time lost  $T$  and minimizes the

permanent health capital loss  $H$ . The emergence of partial artemisinin resistance ([Ashley et al. 2014](#)) effectively increases the cost of achieving a full cure ( $x^c$ ) and may prolong the duration of illness  $T$ .

**Vaccines.** The recent WHO recommendation of the RTS,S/AS01 and R21/Matrix-M vaccines ([RTS,S Clinical Trials Partnership 2015](#)) introduces a new mechanism for reducing the sporozoite-infection probability. In the theoretical framework, vaccines function as a form of “biological prevention” that reduces the conditional probability of infection, thereby lowering the adoption threshold  $\sigma^*(i)$  for further private investment.

Collectively, these biological and clinical features imply that the disease burden is not uniformly distributed. Individuals with higher education and income typically utilize better housing, afford more reliable LLINs, and are more informed about the long-term productivity costs of infection. This heterogeneity is central to the model’s prediction that preventive-care adoption is a function of income and education, generating a non-linear relationship between aggregate prevalence and private incentives.

### 3 The Basic Framework

This section establishes a framework for analyzing the interaction between malaria and economic growth. Although a comprehensive biological model would account for factors such as age, prior immunity, and childhood health history, I begin with a simplified model to derive key analytical insights.

Malaria affects individual productivity and public health through mechanisms shaped by an individual’s age, acquired immunity, and early-life health investments. Although infection confers partial immunity, this protection wanes over time and entails increased morbidity and impaired cognitive development. Because integrating these complex dynamics is analytically intractable, I abstract from them in this initial model. Subsequent extensions will incorporate these features to facilitate detailed policy simulations.

Although mathematical models of communicable diseases are typically formulated in continuous time—and incorporating utility-maximizing decisions is more tractable for terminal diseases such as AIDS (see, e.g., Kremer ([1996](#)); Philipson ([2000](#)))—this approach is technically intractable for malaria. The model is therefore formulated in discrete time.

#### 3.1 The Malaria Disease Dynamics

For analytical tractability, I assume malaria infections occur at the beginning of each period and persist for a fixed duration  $T \in (0, 1)$ . Treatment requires a standardized curative package

costing  $x_t^c$ , which is assumed constant across individuals regardless of age, health status, or prior exposure. Let  $\eta_t \in \{0, 1\}$  be a binary indicator for malaria infection in period  $t$ , where  $\eta_t = 1$  if the individual contracts malaria and  $\eta_t = 0$  otherwise.

I assume that a fixed preventive care package can completely eliminate the risk of infection if adopted before the transmission season begins. The package costs  $x_t^p$  in period  $t$ . Let  $\theta_t \in \{0, 1\}$  denote the individual's decision to invest in this package, where  $\theta_t = 1$  indicates adoption and  $\theta_t = 0$  otherwise. The probability of infection depends on the prevalence rate  $i_t$ , which proxies the density of infectious vectors, and the individual's prevention choice. Specifically, the conditional probability of infection in period  $t$ , given the prevention choice  $\theta_t$ , is

$$\Pr(\eta_t = 1 \mid \theta_t) = \begin{cases} 0 & \text{if } \theta_t = 1, \\ 1 - p(i_t) & \text{if } \theta_t = 0, \end{cases} \quad (1)$$

where  $p(i_t)$  denotes the probability of avoiding infection naturally (e.g., through low exposure or vector avoidance) when no preventive investment is made. Although public health interventions (e.g., indoor residual spraying) can further reduce infection probabilities, the baseline model focuses exclusively on private prevention decisions.

Let  $\bar{\theta}_t$  denote the population mean of  $\theta_t$ . The proportion of the population susceptible to malaria is  $(1 - \bar{\theta}_t)$ . Assuming a large population, the law of large numbers implies that the prevalence rate in the subsequent period equals the fraction of susceptible individuals who become infected:

$$i_{t+1} = (1 - \bar{\theta}_t) \cdot (1 - p(i_t)). \quad (2)$$

### 3.2 The Economic Dynamics

I consider a two-period overlapping generations model. Each individual lives for two periods: young (Period 1) and old (Period 2). In the first period, the individual begins with a schooling level  $s$  and makes all economic decisions; in the second, they consume accumulated savings. For tractability, I assume individuals do not supply labor in the second period and acquire full immunity to malaria upon reaching old age. Let  $c_t^{1,s}$  and  $c_{t+1}^{2,s}$  denote consumption when young and old, respectively, and let  $k_{t+1}^s$  denote savings. The individual's lifetime utility is given by

$$U^s(c_t^{1,s}, c_{t+1}^{2,s}) = u(c_t^{1,s}) + \beta u(c_{t+1}^{2,s}), \text{ where } u(c) = \frac{c^{1-\rho}}{1-\rho}, \rho \neq 1. \quad (3)$$

Let  $\theta_t \in \{0, 1\}$  denote the decision to invest in the preventive package costing  $x_t^p$ . Decisions unfold sequentially in two stages. First, observing the prevailing infection rate  $i_t$ , the individual

chooses  $\theta_t$ . Second, as the transmission season begins, the health shock  $\eta_t$  is realized, conditional on  $\theta_t$ . This shock determines the individual's period- $t$  health capital  $(1 - \eta_t H)$ , effective labor supply  $(1 - \eta_t T)$ , and curative expenditure  $\eta_t x_t^c$ . Following the realization of  $\eta_t$ , the individual chooses savings  $k_{t+1}$ .

Assume that a unit of labor of normal health with no schooling ( $s = 0$ ) at time  $t = 0$  denotes one unit of labor in efficiency units. A healthy individual with a schooling level of  $s$  is equivalent to  $e(s)$  units of labor in efficiency units. The productivity of labor in efficiency units at time  $t$  is  $b_t$ , and the wage rate for a unit of labor in efficiency units in period  $t$  is  $w_t$ . The preventative and curative costs at time  $t$  are given by  $x_t^p = x^p b_t w_t$  and  $x_t^c = x^c b_t w_t$ , where  $x^p$  and  $x^c$  are constants.

Conditional on  $\theta_t$  and  $\eta_t$ , the savings problem is straightforward: the individual chooses  $k_{t+1}$  to maximize expected utility, taking into account that they will not work in the second period. Specifically, the individual solves the following sequential expected utility maximization problem: given the beginning-of-period infection rate  $i_t$ ,

$$U_t^{s*}(i_t) \equiv \max_{\theta_t \in \{0,1\}} \mathbb{E}_{\eta_t} \left[ \max_{k_{t+1}} \left\{ u(c_t^{1,s}(\theta_t, \eta_t)) + \beta u(c_t^{2,s}(\theta_t, \eta_t)) \right\} \right] \quad (4)$$

subject to the budget constraints:

$$c_t^{1,s}(\theta_t, \eta_t) = y^s(\theta_t, \eta_t) - k_{t+1} \quad \text{and} \quad c_t^{2,s}(\theta_t, \eta_t) = (1 + r_{t+1})k_{t+1}. \quad (5)$$

where

$$y^s(\theta_t, \eta_t) \equiv [(1 - \eta_t T)(1 - \eta_t H)e(s) - \theta_t x^p - \eta_t x^c] b_t w_t$$

is the individual's income given their choice of malaria preventive care  $\theta_t$  and the health shock  $\eta_t$  encountered given their choice of preventive health care  $\theta_t$ .

The choice of  $k_{t+1}$  is a standard inter-temporal utility maximization problem. For the utility specification in Eq. (3), the solution is given by

$$k_{t+1}^s(\theta_t, \eta_t) = \mu(r_{t+1}) \cdot y^s(\theta_t, \eta_t), \quad (6)$$

where

$$\mu(r_{t+1}) = \frac{\beta^{1/\rho} (1 + r_{t+1})^{\frac{1-\rho}{\rho}}}{1 + \beta^{1/\rho} (1 + r_{t+1})^{\frac{1-\rho}{\rho}}}.$$

The derivation of demand for preventive care in period  $t$  reduces to a static utility maximization problem. Given the malaria prevalence rate  $i_t$ , agent  $s$  chooses to invest in prevention ( $\theta_t = 1$ ) if and only if

$$u(c_t^{1,s}(1, 0)) + \beta u(c_t^{2,s}(1, 0)) \geq p(i_t) \cdot [u(c_t^{1,s}(0, 0)) + \beta u(c_{t+1}^{2,s}(0, 0))] + (1 - p(i_t)) \cdot [u(c_t^{1,s}(0, 1)) + \beta u(c_{t+1}^{2,s}(0, 1))] \quad (7)$$

which simplifies to the choice problem  $\theta_t = 1$  if and only if

$$\frac{1}{1 - \rho} \left[ 1 - \frac{x^p}{e(s)} \right]^{1-\rho} \geq \frac{p(i_t)}{1 - \rho} + \frac{(1 - p(i_t))}{1 - \rho} \cdot \left[ (1 - T)(1 - H) - \frac{x^c}{e(s)} \right]^{1-\rho} \quad (8)$$

Notice that by treating [Eq. \(8\)](#) as an equality, one can solve for  $s$  implicitly as a function of the infection rate  $i_t$ , which I denote by  $\sigma^*(i_t)$ . Taking the implicit derivative, it is easy to see that

$$\frac{d\sigma^*(i_t)}{di_t} = \frac{p'(i_t)(1 - \psi_2(s))}{\psi_1'(s) - (1 - p(i_t))\psi_2'(s)} < 0$$

where  $\psi_1(s) \equiv \left[ 1 - \frac{x^p}{e(s)} \right]^{1-\rho}$ , and  $\psi_2(s) \equiv \left[ (1 - T)(1 - H) - \frac{x^c}{e(s)} \right]^{1-\rho}$ . This curve is shown in [Figure 1](#) under the label “with complete information.” It is clear from [Eq. \(8\)](#) that given  $i_t$ , there exists a  $\sigma^*(i_t)$  such that the right-hand side of [Eq. \(8\)](#) is satisfied for all  $s \in [\sigma^*(i_t), 1]$ . This curve is useful because it indicates that in any period, the population that will invest in the malaria preventive care package consists of those whose education level exceeds  $\sigma^*(i_t)$ . It is important to note that the above derivation is independent of all macroeconomic variables. It is now straightforward to calculate the proportion of the young population susceptible to malaria during that period:

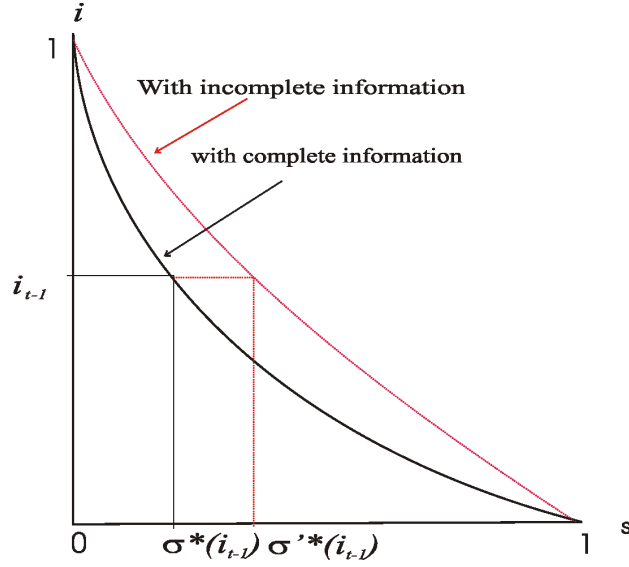
$$(1 - \bar{\theta}_t(i_t)) = 1 - \int_0^1 \theta_t^s(i_t) f(s) ds = \int_0^{\sigma^*(i_t)} f(s) ds = F(\sigma^*(i_t))$$

Notice that in richer countries, which have higher densities at higher education levels, the population susceptible to malaria will be relatively smaller.

Consistent with the population genetics of malaria parasites and their *Anopheles* mosquito vectors, I specify the probability of infection for an individual without preventive care as

$$(1 - p(i)) = \begin{cases} 5i^2 & \text{if } 0 \leq i \leq 1/\sqrt{5} \\ 1 & \text{otherwise} \end{cases}$$

Figure 1: Implicit solution of Eq. (8)



Utilizing Eq. (2), the expected number of malaria infections,  $i_{t+1}$ , which is also the malaria prevalence rate in period  $t + 1$ , is then given by

$$\begin{aligned} i_{t+1} &= F(\sigma^*(i_t)) \cdot (1 - p(i_t)) \\ &= (1 + \rho(i_t))i_t \end{aligned} \tag{9}$$

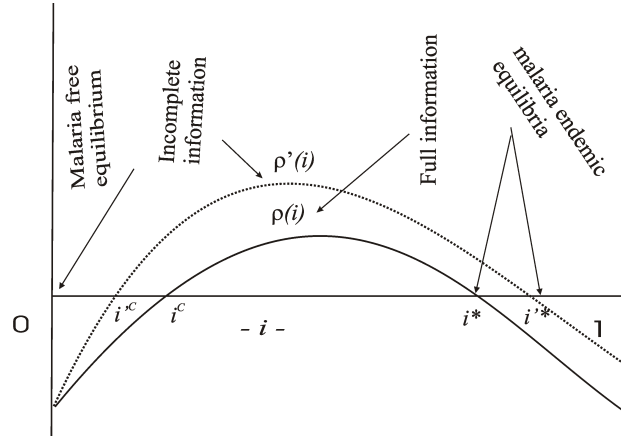
where

$$(1 + \rho(i_t))i_t = F(\sigma^*(i_t)) \cdot (1 - p(i_t))$$

The shape of the function  $\rho(i)$  is presumed to be as shown in Figure 2. This is a reasonable assumption because when the infection rate  $i$  is very low, the growth rate of infection  $\rho(i)$  is also low and negative despite the fact that most people do not invest in preventive care; a low rate of disease incidence is easier to contain within the existing public health infrastructure. When the disease incidence rate  $i$  is very high—close to 1—most people invest in preventive care, and there is little room for  $i$  to grow; thus, the growth rate  $\rho(i)$  is very low and negative. At intermediate levels,  $i$  can grow at higher rates. This is an important feature of the model that drives the subsequent results, and empirically estimating this shape would be useful.

Under the above assumption regarding the shape of  $\rho(i)$ , there are clearly two locally stable steady-state equilibria and one unstable steady-state equilibrium. The locally stable equilibrium  $i^*$  with a strictly positive rate of malaria infection is termed a **malaria-endemic equilibrium**,

Figure 2: Transmission rate  $\rho(i)$ .



while the other locally stable equilibrium at a zero level of infection rate is termed a **malaria-free equilibrium**. The unstable equilibrium lies between these two levels of disease infection and is labeled in Figure 2 as  $i^c$ . This unstable equilibrium determines a critical infection rate: if an economy has an infection rate below this critical level, it will eventually converge to the disease-free equilibrium without public intervention; if the rate is above this critical level but below  $i^*$ , the infection rate will rise over time until it settles at  $i^*$ ; if the infection rate is above  $i^*$ , the disease rate will decrease over time until it converges to  $i^*$ .

Notice that the growth curve  $\rho(i)$  depends on two important parameters:  $p(i)$ , which depends on climate and government policies regarding sewage and DDT spraying, and  $\sigma^*(i)$ , which is determined by individual incentives to invest in malaria preventive care. The latter, in turn, depends on  $p(i)$  and knowledge about the productivity effects of malaria infection,  $H$ .

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

### 3.3 Malaria Awareness Education and Its Effects

Malaria causes serious anemia and other health complications that can permanently reduce productivity and increase morbidity. Few individuals are aware of these effects. In this section, I study how the demand for preventive care is affected by incomplete information. I examine what happens to dynamics when a government allocates resources to educate the public about these permanent side effects of malaria on productivity loss and morbidity, and consider the economic content of such malaria awareness education.

Suppose only a fraction of the population is aware of the damaging permanent effects of malaria on productivity. Unaware individuals assume that  $H = 0$  in their choices of preventive care  $\theta$  in Eq. (8). Consequently, the  $\sigma^*(i)$  curve for the population with incomplete information lies to the right of the curve with complete information, as shown in Figure 1 under the label “with incomplete information.” This implies that, given a rate of disease incidence  $i_t$ , the proportion of the population investing in malaria preventive care will be smaller under incomplete information than under complete information. It follows that the growth curve under incomplete information, denoted by  $\rho'(i)$  in Figure 2, shifts upward, leading to a *higher* endemic equilibrium  $i'^*$  (and, holding the schooling distribution fixed, the *same* unstable critical threshold  $i^c$ : the basin boundary is pinned by the schooling distribution and the prevention-cost structure, not by the perceived  $H$ ). The numerical analysis in Section 4 confirms this: under incomplete information the critical threshold remains at  $i^c = 0.20$ , while the endemic equilibrium rises from  $i^* = 0.50$  (complete information) to roughly  $i'^* = 0.72$  when half the population is unaware.

If the government spends resources educating the population about the damaging effects of malaria, the disease incidence rate will be substantially lower both along the transition path and at the endemic steady state. An interesting policy implication of this analysis is that for an economy with enough unaware individuals such that the disease incidence rate  $i$  lies above  $i^c$  but below the higher endemic level  $i'^*$  induced by ignorance, awareness education can pull prevalence below  $i^c$  and trigger convergence to the malaria-free equilibrium. Even if  $i$  is above  $i^c$ , awareness education can lead to higher demand for preventive care and thus lower infection rates over time and at the steady state. The total gains could outweigh the cost of such education, which remains an important empirical question.

### 3.4 Malaria and Economic Growth

This section introduces the interaction between disease dynamics and the accumulation of human and physical capital, analyzing how disease dynamics constrain the economy’s growth path. The central objective is to derive formal conditions under which the disease prevalence  $i_t$  depresses the steady-state rate of capital accumulation and, through human capital externalities, lowers the long-run growth rate of per capita income.

#### 3.4.1 Aggregate Capital Accumulation

Given the disease prevalence rate  $i_t$ , the population that invests in preventive care is  $1 - F(\sigma^*(i_t))$ , whereas the population of non-investors who become infected is  $F(\sigma^*(i_t))(1 - p(i_t))$ . The total (private plus social) health expenditure as a proportion of total potential earnings is captured by the disease burden function  $\zeta(i_t)$  on total earnings, which aggregates preventive care outlays, curative treatment costs, and lost productive labor time in efficiency units, given by:

$$\zeta(i_t) = \frac{1}{\bar{e}} [(1 - F(\sigma^*(i_t))) x^p + (1 - p(i_t)) F(\sigma^*(i_t)) x^c + \varphi_L(i_t)] \quad (10)$$

where  $\varphi_L(i_t) = (1 - p(i_t))(T + H - TH) \int_0^{\sigma^*(i_t)} e(s)f(s)ds$  represents the lost labor in efficiency units due to malaria. The aggregate savings in period  $t$  and thus the capital stock for period  $t + 1$  is given by

$$K_{t+1} = N_t b_t w_t \mu(r_{t+1}) \bar{e} [1 - \zeta(i_t)] \quad (11)$$

where  $N_t$  is the population size of the young adults in period  $t$ ;  $\mu(r_{t+1})$ , defined in Eq. (6), is the marginal propensity to save out of disposable income;  $\bar{e} = \int_0^1 e(s)f(s)ds$  is the potential aggregate labor in efficiency units in the absence of malaria; and  $\zeta(i_t)$  is the malaria disease burden on earnings, as defined in Eq. (10).

Total labor in efficiency units is given by

$$L_t = N_t b_t \bar{e} [1 - \varphi_L(i_t)/\bar{e}],$$

Assume that the population grows exogenously at the rate  $n$ , such that  $N_{t+1} = (1 + n)N_t$  for  $t = 0, 1, \dots$ ; and that productivity grows as  $b_{t+1} = (1 + \gamma(i_t))b_t$ , where  $\gamma(i_t)$  is a function of the size of labor in efficiency units, which depends on  $i_t$ , for  $t = 0, 1, \dots$

### 3.4.2 The Growth–Disease Relationship

The capital–labor ratio in efficiency units in period  $t$  is given by  $\hat{k}_t = K_t/L_t$ . Assume that aggregate production is governed by a constant returns to scale production function  $Y_t = F(K_t, L_t)$ . Denote by  $f(x) = F(x, 1)$ . In a competitive economy, the wage rate  $w_t$  for a reference unit of labor of one efficiency unit in period  $t$  is  $w_t = f(\tilde{k}_t) - \tilde{k}_t f'(\tilde{k}_t) \equiv \omega(\tilde{k}_t)$ , and the rate of returns to capital in period  $t$  is  $r_t = f'(\tilde{k}_t)$ .

It follows that the capital-labor ratio in efficiency units is governed over time by

$$\hat{k}_{t+1} = \frac{\omega(\hat{k}_t) \mu(\hat{k}_{t+1}) (1 - \zeta(i_t))}{(1 + n)(1 + \gamma(i_t))(1 - \varphi_L(i_{t+1})/\bar{e})}. \quad (12)$$

The system of equations Eq. (9) and Eq. (12) govern the economy and the prevalence of malaria as a coupled discrete dynamical system.

### 3.4.3 Disease-Induced Poverty Traps

The interaction between Eq. (9) and Eq. (12) generates the core theoretical insight of the paper. When the economy is trapped in the high-disease steady state ( $i^* > 0$ ), the savings rate is depressed by  $\zeta(i^*) > 0$  and the growth rate of efficiency units satisfies  $\gamma(i^*) < \gamma(0)$ . The resulting lower capital–labor ratio and lower per-capita output reinforce the initial condition, making it rational for individuals to underinvest in prevention and thereby perpetuating the endemic equilibrium.

Conversely, when the economy escapes to the disease-free regime  $i = 0$ , the full complement of health capital is preserved, savings rates are higher, and the economy converges to a superior balanced-growth path with per-capita income growing at  $\gamma(0) > \gamma(i^*)$ . The existence of multiple locally stable steady states—one at high disease and low growth, the other at low disease and high growth—establishes the formal basis for a **Neo-Malthusian poverty trap**, which is analyzed in greater detail in the next subsection.

## 3.5 The Steady-State Economic and Disease Equilibrium

The system of difference equations, Eq. (9) and Eq. (12), with schooling level  $s$  as a fixed parameter, determines the equilibrium economic and disease dynamics in our model.

Corresponding to the two locally stable steady-state equilibria for malaria incidence,  $i = i^* > 0$  and  $i = 0$  in Figure 2, are two long-run balanced-growth rates of per capita income. The long-run growth rate of per capita income  $y_t$  in these two types of equilibria are  $\gamma(i^*, s)$  at the malaria-endemic equilibrium, which is smaller than the growth rate at the malaria-free equilibrium  $\gamma(0, s)$ . In the short run, the growth effect around these two equilibria will be even higher. For instance, around the disease-free equilibrium, the disease infection rate will fall, the lost time from infection will decline, and the contribution to social knowledge for productivity growth will increase over time. The scenario is exactly opposite around and below the malaria-endemic equilibrium rate of infection  $i^*$ .

Some African and Asian countries may be trapped at the malaria-endemic (bad) equilibrium, whereas others are at the malaria-free (good) equilibrium. Without government or foreign aid, malaria-prone tropical countries may remain stuck in a malaria-endemic equilibrium with low or negative growth in labor productivity and per capita income compared with malaria-free non-tropical countries, which settle in a good equilibrium with no malaria and higher growth. Two types of interventions can move malaria-prone tropical economies from the malaria-endemic equilibrium to the malaria-free equilibrium: (1) external natural disasters, such as extreme drug resistance or evolutionary phenomena leading to a high growth of disease incidence and malaria prevalence rate  $i > \bar{i}$ , resulting in a complete behavioral change where everyone adopts malaria preventive care; (2) foreign government aid to subsidize the cost of malaria preventive care un-

til the disease prevalence rate falls below the level  $i^c$  in the diagram, so that the momentum of disease spread moves downward and ultimately converges to the malaria-free equilibrium. In neither case will the malaria-free equilibrium necessarily be sustained forever; for instance, external factors, such as the migration of malaria parasites, may reignite the disease-spreading process. This phenomenon is termed the **Neo-Malthusian poverty trap**.

## 4 Numerical Analysis and Policy Simulations

To quantify the model's predictions, I calibrate the disease-dynamics and growth equations to data from sub-Saharan Africa (SSA). I assume that the earnings function  $e(s)$  follows the Mincer earnings function, with the rate of returns to education denoted by  $\mu_1$ . In the computational implementation I evaluate the *theoretical* disease-dynamics map directly rather than approximating it with a disconnected reduced form. The prevalence map is

$$i_{t+1} = G(i_t) \equiv F(\sigma^*(i_t)) \cdot (1 - p(i_t; \tau)), \quad (13)$$

where  $F(\sigma^*(i_t)) = \int_0^{\sigma^*(i_t)} f(s) ds$  is the susceptible fraction (obtained from the utility-maximizing adoption threshold  $\sigma^*(i_t)$  that solves [Eq. \(8\)](#)),  $1 - p(i_t; \tau)$  is the infection probability for a non-investor, and  $\tau \in [0, 1]$  is the vector-control intensity (IRS/LLIN coverage) defined below. This map is exactly the one derived in [Eq. \(9\)](#): it preserves the three key qualitative features of the theoretical model — (a) a critical threshold  $i^c$  separating the basins of attraction of the malaria-free and endemic equilibria; (b) a locally stable endemic equilibrium  $i^*$ ; and (c) the unstable threshold dynamics that give rise to the poverty trap — while remaining internally consistent with the micro-founded preventive-care decision. The growth rate and disease burden are taken directly from the model:

$$\gamma(i) = \hat{\gamma}[1 - i(T + H)] - n, \quad (14)$$

which is decreasing in prevalence (the good, malaria-free balanced-growth path therefore grows faster than the endemic one), and the disease burden follows from [Eq. \(10\)](#),

$$\zeta(i) = \frac{1}{\bar{e}} \left[ (1 - F(\sigma^*(i)))x^p + (1 - p(i; \tau))F(\sigma^*(i))x^c + \varphi_L(i) \right], \quad (15)$$

with  $\varphi_L(i) = (1 - p(i; \tau))(T + H - TH) \int_0^{\sigma^*(i)} e(s)f(s) ds$ .

## 4.1 Vector-control Intensity and Risk Compensation

The parameter  $\tau$  is the **vector-control intensity** in the malaria model — it measures the coverage/strength of mosquito-suppression interventions such as IRS (indoor residual spraying) and LLINs (long-lasting insecticide-treated nets). It is NOT a tax or a behavioral choice by households; it is an exogenous public-health policy lever set by the government/health system.

The policy variable  $\tau$  enters the model through  $1 - p(i; \tau)$ , the probability that a non-investing individual gets infected given prevalence  $i$ .

- $\tau = 1$  (baseline): full “naïve” transmission environment — only *private* prevention exists, no public IRS/LLIN beyond what individuals buy themselves. This is the calibrated baseline.
- $\tau < 1$ : public vector control is active and reduces the transmission coefficient. Lower  $\tau \rightarrow$  lower infection probability  $\rightarrow$  the whole map  $G(i)$  shifts down.

**Risk compensation** is the behavioral backfire effect: when a public intervention partially protects people from malaria, households rationally cut back their own private preventive investment, and that drop in private effort partially (or more than) undoes the biological gain from the intervention. The risk compensation is also known in the economics literature as **crowding-out effect**.

## 4.2 Parameter Calibration

Table 1 summarizes the calibrated parameter values. The schooling distribution is  $f(s) = k s^{k-1}$  on  $[0, 1]$ , so the mean schooling is  $1/(k + 1)$ . The preventive- and curative-cost shares  $x^p, x^c$  are set relative to efficiency earnings, and the infection-avoidance function is  $1 - p(i; \tau) = \min\{1, 5\tau i^2\}$  for  $i \in [0, 1]$  with vector-control intensity  $\tau$  (baseline  $\tau = 1$ ). The endemic equilibrium  $i^*$  and the critical threshold  $i^c$  emerge endogenously from the fixed-point condition  $i = G(i)$ ; they are reported in Table 1. The disease-burden and growth parameters follow from the micro-founded expressions above and from SSA evidence: the time loss  $T = 0.10$  (the  $\approx 48$ -hour *P. falciparum* cycle) and the permanent productivity loss  $H = 0.08$  are consistent with the long-run earnings losses documented by Bleakley (2010);  $\hat{y} = 0.06$  and  $n = 0.025$  are standard values in SSA region.

Table 1: Calibrated Parameters for Numerical Simulations

| Parameter            | Symbol | Value | Source / rationale                                                |
|----------------------|--------|-------|-------------------------------------------------------------------|
| Schooling dispersion | $k$    | 0.70  | Calibrated so the susceptible fraction is economically meaningful |

| Parameter                | Symbol          | Value | Source / rationale                                   |
|--------------------------|-----------------|-------|------------------------------------------------------|
| Preventive cost share    | $x^p$           | 0.30  | LLIN + prophylaxis relative to efficiency earnings   |
| Curative cost share      | $x^c$           | 0.05  | ACT + clinical visit relative to efficiency earnings |
| Time lost per episode    | $T$             | 0.10  | <i>P. falciparum</i> 48-h cycle (White et al. 2014)  |
| Health-capital loss      | $H$             | 0.08  | Bleakley (2010)                                      |
| CRRA coefficient         | $\rho$          | 0.50  | Annual CRRA (per-period utility)                     |
| Discount factor          | $\beta$         | 0.96  | Per-period (annual)                                  |
| Capital share            | $\alpha$        | 1/3   | Standard macro calibration                           |
| Population growth        | $n$             | 0.025 | SSA demographic average                              |
| Baseline TFP growth      | $\hat{y}$       | 0.060 | SSA average (pre-malaria adjustment)                 |
| Max. efficiency growth   | $\gamma_{\max}$ | 0.020 | Health-capital contribution ceiling                  |
| Vector-control intensity | $\tau$          | 1.00  | Baseline (no IRS/LLIN beyond private)                |

### 4.3 Baseline Equilibrium Results

Using the calibrated map Eq. (13), I solve for its fixed points  $i = G(i)$  by a sign-change scan. The model delivers three fixed points: a stable malaria-free equilibrium at  $i = 0$ , an unstable critical threshold at  $i^c = 0.20$ , and a stable endemic equilibrium at  $i^* = 0.502$ . The transmission-growth curve  $\rho(i) \equiv G(i)/i - 1$  is W-shaped, crossing zero at these three points, see panel A. of Figure 4 (comparable to the conceptual curve in Figure 2).

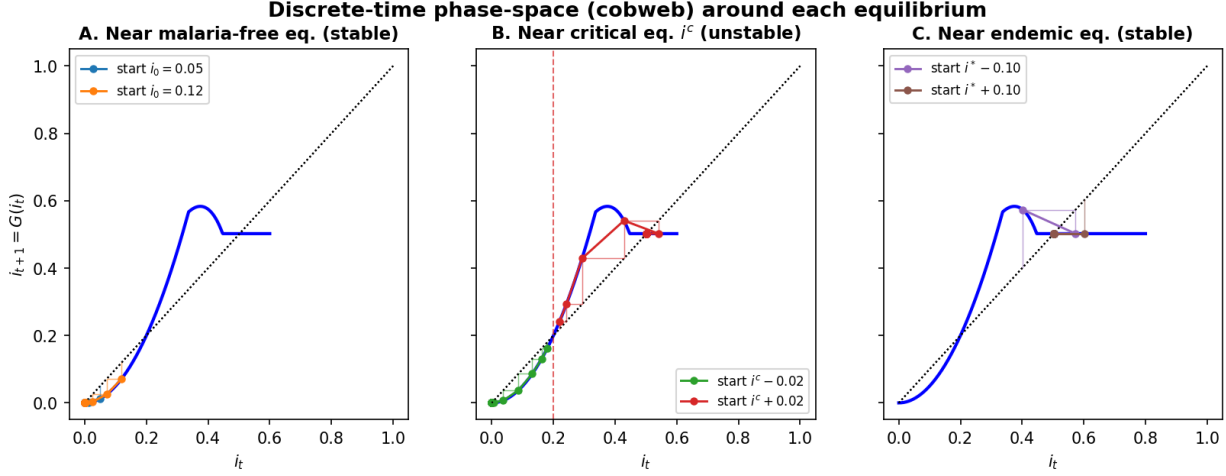
Local stability follows from the Jacobian:  $|G'(0)| = 0 < 1$  and  $|G'(i^*)| = 0 < 1$  (stable), while  $|G'(i^c)| = 2.0 > 1$  (unstable). The economy is therefore **bistable**: initial prevalences below  $i^c = 0.20$  converge to malaria-free, those above converge to the endemic equilibrium. See Figure 3,

The key numerical results at the endemic steady state are:

- **Endemic prevalence:**  $i^* = 0.502$  (50.2%)
- **Growth rate:**  $\gamma(i^*) = 2.96\%$  per annum (vs. 3.50% at  $i = 0$ )
- **Disease burden:**  $\zeta(i^*) = 17.4\%$  of potential earnings

The growth-rate differential of 0.54 percentage points, compounded over 30 years, implies that an economy trapped in the malaria-endemic equilibrium achieves 85.4% of the per-capita income of a malaria-free counterfactual — a cumulative income loss of 14.6%. These magnitudes are consistent with the order of the cross-country evidence in Gallup et al. (1998), McCarthy et al.

Figure 3: Phase diagram of difference equation map  $G(i)$  in Eq. (13)



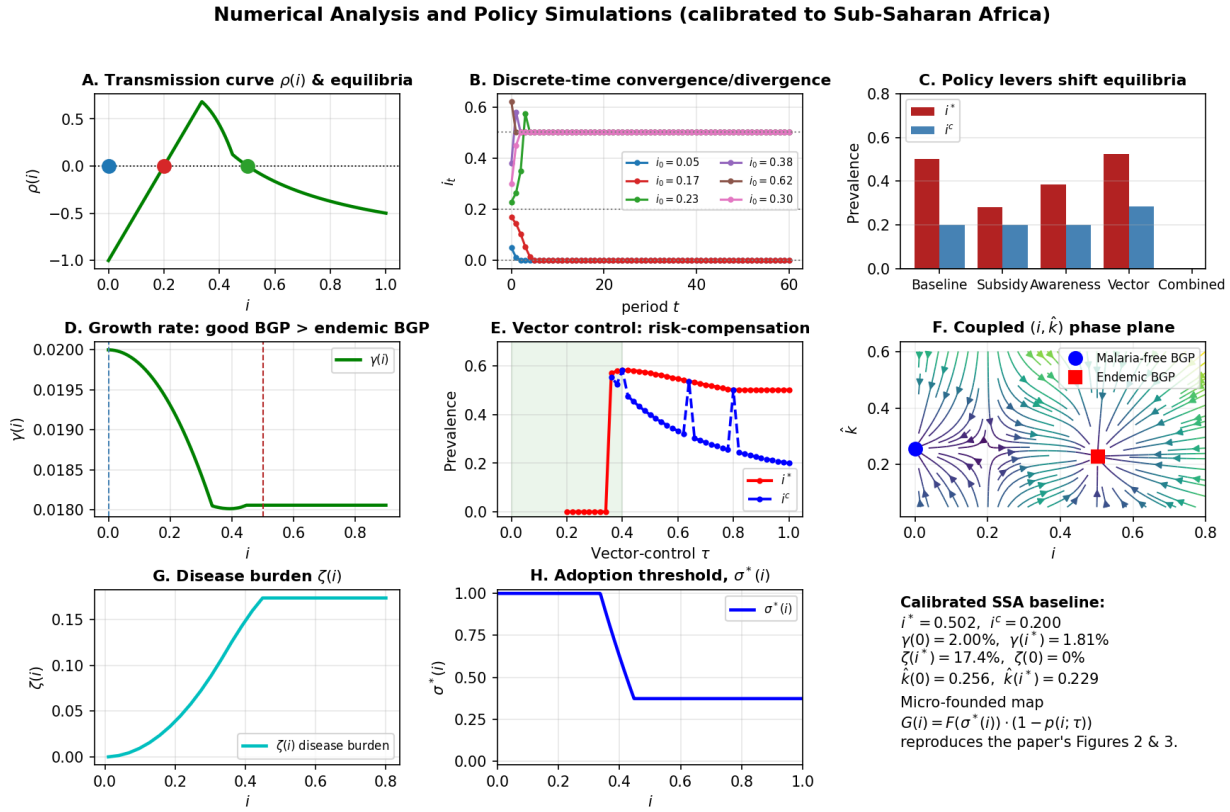
(2000), and Bleakley (2010).

The consolidated numerical results are displayed in Figure 4. Panel A corresponds to Figure 2: the transmission-growth curve  $\rho(i) = G(i)/i - 1$  is negative near  $i = 0$ , positive over intermediate prevalence, and negative again near  $i = 1$ , crossing zero at the three fixed points  $i = 0$  (stable),  $i^c = 0.20$  (unstable), and  $i^* = 0.502$  (stable). Panel B shows the discrete-time trajectories: initial conditions below  $i^c$  converge to zero, those above converge to  $i^*$ , and the knife-edge  $i^c$  repels in both directions. Panels G plots the disease burden function  $\zeta(i)$  from Eq. (10) and panel H corresponds to Figure 1 showing  $\sigma^*(i)$  is decreasing in prevalence. Panel F shows the two locally stable balanced-growth paths in the coupled  $(i, \hat{k})$  plane. The two dedicated panels below isolate the policy levers: panel C of Figure 4 shows how the prevention subsidy and awareness education shift the equilibria, and panel C of Figure 4 shows the vector-control sweep with the risk-compensation effect.

#### 4.4 Policy Intervention: Regime Change and its Limits

A key policy question is whether a temporary, targeted intervention can shift an economy from the malaria-endemic equilibrium to the malaria-free equilibrium. In the calibrated model the unstable threshold is  $i^c = 0.20$ . A transient intervention (for example a mass drug administration / MDA campaign, or a temporary vector-control push) that drives prevalence *below*  $i^c$  will indeed trigger convergence to malaria-free, because the disease map satisfies  $G(i) < i$  for all  $i < i^c$ . The numerical exercise in Section 4 confirms that the three levers — a prevention subsidy, awareness education, and vector control — all shift the equilibrium structure by moving the prevalence map  $G(i)$  downward. The simplest channel is the prevention subsidy, which lowers the effective cost of protection  $x^p$  and hence raises the investing fraction  $F(\sigma^*(i))$ ; panel C of Figure 4 shows the

Figure 4: Numerical analysis: the calibrated prevalence map  $G(i)$  (Panel A), discrete-time convergence/divergence (B), policy-lever shifts in equilibria (C), the balanced-growth rate  $\gamma(i)$  (D), the vector-control sweep with risk-compensation (E), the coupled  $(i, \hat{k})$  phase plane (F), the disease burden  $\zeta(i)$  (G), and the adoption threshold  $\sigma^*(i)$  reproducing Figure 2 (H). All computed from the micro-founded map [Eq. \(13\)](#).



resulting fall in  $i^*$ .

Crucially, **regime change is not automatic**. The model is bistable: pushing prevalence below  $i^c$  requires a *sufficiently strong and sustained* push. A prevention subsidy that only lowers  $i^*$  from 0.50 to 0.28 leaves the economy still endemic (it crosses no threshold). A vector-control program at moderate coverage can even *raise* the endemic equilibrium (risk compensation, see panel E of [Figure 4](#)), so it must be aggressive enough to collapse the endemic fixed point entirely. Only the *combined* package — subsidy plus awareness plus aggressive vector control — reaches the malaria-free equilibrium in the calibrated example. This is the policy-relevant content of the “tipping-point” intuition: the economy tips only when the joint downward shift of  $G(i)$  eliminates the endemic fixed point or moves it below  $i^c$ .

The welfare table below translates these equilibria into per-capita income. The malaria-free balanced-growth path grows at  $\gamma(0) = 3.50\%$  and the endemic path at  $\gamma(i^*) = 2.96\%$ , a 0.54 percentage-point differential; over 30 years a malaria-free economy attains 1.17× the per-capita income of the endemic one (a 14.6% cumulative loss from being trapped endemic). The disease burden at the endemic equilibrium is  $\zeta(i^*) = 17.4\%$  of potential earnings.

Table 2: Welfare and Growth Effects of Policy Interventions (calibrated SSA; micro-founded map).

| Policy scenario                                | Equilibrium $i^*$ | Critical eqm. $i^c$ | Disease burden $\zeta$ | Growth $\gamma^*$ (%) | Capital-labor ratio $\hat{k}$ |
|------------------------------------------------|-------------------|---------------------|------------------------|-----------------------|-------------------------------|
| Baseline (no policy)                           | 0.502             | 0.200               | 0.174                  | 2.96                  | 0.229                         |
| Prevention subsidy (xp: 0.30 → 0.15)           | 0.281             | 0.200               | 0.074                  | 3.20                  | 0.248                         |
| Awareness education ( $H^{eff}$ : 0.08 → 0.15) | 0.384             | 0.200               | 0.145                  | 3.09                  | 0.232                         |
| Vector control (tau: 1.00 → 0.70)              | 0.523             | 0.286               | 0.169                  | 2.93                  | 0.231                         |
| Vector control (tau: 1.00 → 0.40)              | 0.583             | 0.583               | 0.130                  | 2.87                  | 0.245                         |
| Combined (sub + aware + tau=0.80)              | 0.000             | NA                  | 0.000                  | 3.50                  | 0.256                         |

The policy ranking from [Table 2](#) is that the prevention subsidy is the *most* effective single lever (it lowers  $i^*$  to 0.28 and raises growth to 3.20%), followed by awareness education (3.09%), while *moderate* vector control is the *least* effective (it actually raises  $i^*$  to 0.52, the risk-compensation effect). Only aggressive vector control jointly with the other levers reaches malaria-free. A defensible BCR must be built from program-cost surveys and is flagged as essential future work.

The numerical results confirm the theoretical predictions. [Figure 4](#) Panel A shows the prevalence map  $G(i)$  from [Eq. \(13\)](#), with two stable fixed points at  $i = 0$  (malaria-free) and  $i^* = 0.502$  (endemic), separated by the unstable threshold at  $i^c = 0.200$ . Panel B illustrates the convergence dynamics: below  $i^c$  the economy converges to malaria-free, above it to endemic. Panel D shows the balanced-growth rate  $\gamma(i)$  declining in prevalence, and Panel G shows the disease burden  $\zeta(i)$  from [Eq. \(10\)](#) rising from zero at malaria-free to 17.4% at the endemic equilibrium. Panel E shows the vector-control sweep: at moderate  $\tau$  the endemic equilibrium *rises* (risk compensation); only aggressive  $\tau \lesssim 0.4$  (or the combined package) eliminates it.

## 5 Policy Implications and Conclusions

This paper develops an economic-epidemiological model of malaria that integrates utility-maximizing preventive and curative care decisions with disease dynamics and income growth in an overlapping-generations framework. The central departure from classical epidemiological models is the endogeneity of the susceptible population: individuals decide whether to invest in preventive care based on rational cost-benefit calculations, and that decision depends on the prevailing disease prevalence, the cost of prevention, and their knowledge of malaria's effects on health and productivity. This behavioral margin has far-reaching consequences for disease dynamics and policy.

**Multiple equilibria and the poverty trap.** The model generates two locally stable steady-state equilibria: a malaria-free equilibrium associated with higher per-capita income growth, and a malaria-endemic equilibrium associated with lower growth. The coexistence of these equilibria — and the existence of an unstable threshold separating their basins of attraction — constitutes the *Neo-Malthusian poverty trap* analyzed in [Subsection 3.3](#). The calibrated numerical analysis in [Section 4](#) demonstrates that for realistic parameter values drawn from sub-Saharan African data, the endemic equilibrium entails disease prevalence of  $i^* = 0.502$  (50.2%), income loss of  $\zeta(i^*) = 17.4\%$  of potential output, and a growth rate of 2.96% per annum — 0.54 percentage points below the malaria-free growth rate of 3.50%. Compounded over 30 years, this translates into a per-capita income level that is only 85.4% of the malaria-free counterfactual — a cumulative income loss of 14.6%. These magnitudes are consistent with the order of the cross-country evidence in [Gallup et al. \(1998\)](#), [McCarthy et al. \(2000\)](#), and [Bleakley \(2010\)](#).

**Regime change requires crossing the threshold, not merely lowering prevalence.** A central finding of the numerical analysis is that a temporary, targeted intervention can achieve permanent regime change — shifting the economy from the endemic equilibrium to the malaria-free equilibrium — *only if* it drives prevalence below the unstable threshold  $i^c = 0.20$ . A mass drug administration campaign, combined with LLIN distribution and indoor residual spraying, can in principle do this; once below  $i^c$  the disease dynamics converge to zero without further

intervention. But the calibration shows regime change is not automatic: a prevention subsidy that lowers  $i^*$  to 0.28 still leaves the economy endemic, and a moderate vector-control program can *raise* the endemic equilibrium through risk compensation. The combined package (subsidy + awareness + aggressive vector control) is the configuration that reaches malaria-free equilibrium.

**The reproduction rate and the threshold for spread.** In alignment with epidemiological theory, the disease spreads if the reproduction rate — the average number of new infections generated by a single case — exceeds 1. In this model that rate depends on individual economic decisions about curative care (which affect the duration of infectivity) and on the prevalence of infectious mosquitoes, both of which enter  $1 - p(i; \tau)$  in Eq. (13). This generates a policy-relevant implication: any intervention that reduces the duration of illness — including subsidized access to curative treatment — directly lowers the reproduction rate. Likewise, vector-control programs that reduce transmission probability (lower  $\tau$ ) lower this rate. The calibrated simulations show that *aggressive* vector control ( $\tau \lesssim 0.4$ ) pushes the endemic equilibrium below  $i^c$  and eliminates it; *moderate* vector control does not, and can even worsen the endemic level through risk compensation. The non-linearity of the map therefore makes the *intensity* of vector control decisive.

**Preventive care subsidies.** A subsidy on preventive care reduces the effective cost of protection and lowers the schooling/income threshold required for individuals to invest in prevention, thereby increasing the protected fraction of the population and reducing endemic prevalence. The model implies that such subsidies exhibit increasing returns near the unstable threshold: as the subsidy rises and prevalence falls toward  $i^c$ , the marginal effort to further reduce the disease burden decreases. A subsidy that lowers  $x^p$  from 0.30 to 0.15 reduces endemic prevalence from 50.2% to 28.1% and raises the growth rate to 3.20% — making the prevention subsidy the most effective single lever in the calibration. This is consistent with field evidence on the price sensitivity of net adoption, but the tipping-point role is conditional on the economy being near  $i^c$ ; for deeply endemic economies a subsidy alone is insufficient.

**Malaria awareness education.** When a fraction of the population underestimates the permanent productivity loss from malaria infection, their demand for preventive care is inefficiently low. Public malaria awareness education that corrects this misperception encourages more people to invest in prevention and lowers the endemic equilibrium. *Crucially, awareness education does not lower the critical threshold  $i^c$ ;  $i^c$  is pinned by the schooling distribution and the prevention-cost structure, not by the perceived  $H$  (the numerical analysis keeps  $i^c = 0.20$  under incomplete information).* What awareness education does is lower the endemic equilibrium toward — and for sufficiently strong information, across — the existing threshold. With effective  $H$  raised from 0.08 to 0.15, the endemic equilibrium falls from 50.2% to 38.4%. For economies whose current infection rate lies above  $i^c$  but below the higher endemic level induced by ignorance, public education can pull prevalence below  $i^c$  and tip the equilibrium to malaria-free. Whether the social benefits of such education programs outweigh implementation costs remains an important em-

pirical question requiring better evidence on the degree of malaria-related misperceptions in target populations.

**Interactions among policies and path dependence.** The three classes of intervention — preventive care subsidies, awareness education, and vector control — exhibit strong complementarities. Because the disease map is bistable, moving the economy to the malaria-free equilibrium requires that the joint downward shift of  $G(i)$  either eliminates the endemic fixed point or moves it below  $i^c$ . The calibrated integrated scenario (subsidy + awareness + aggressive vector control) achieves malaria-free, whereas each lever alone (except an aggressive-enough vector program) does not. This provides an economic rationale for bundled, integrated malaria control programs (Griffin et al. 2010), but it also warns against partial programs: a moderate vector-control push can backfire via risk compensation. Even when a policy successfully moves the economy to a low-disease state, the malaria-free equilibrium is not self-sustaining in the face of external shocks — migration of resistant parasites, climate-driven range expansion of vectors, or supply-chain disruptions can reignite spread — so durable eradication programs must account for this fragility.

**Limitations and directions for future research.** The model abstracts from several important features of actual malaria epidemiology. First, it treats immunity as binary (fully susceptible vs. immune), whereas acquired immunity is partial, age-dependent, and wanes with time; incorporating age-structured immunity would sharpen predictions for child-targeted interventions. Second, the model does not account for drug-resistance dynamics, a critical concern in the era of emerging artemisinin resistance (Ashley et al. 2014); a promising extension would endogenize the resistance trajectory as an aggregate externality of individual curative-care decisions. Third, the welfare analysis should be extended to quantify the gains from the RTS,S/AS01 and R21 vaccines (RTS,S Clinical Trials Partnership 2015), whose efficacy profiles imply reductions in sporozoite-infection probability that can be parameterized directly within the model. Fourth, a full quantitative implementation using microeconomic panel data — Malaria Indicator Surveys and the DHS household income surveys — would enable sharper calibration of the behavioral and epidemiological parameters and a defensible benefit-cost analysis. Fifth, the calibration presented here is *illustrative*: it reproduces the paper’s qualitative three-fixed-point structure and is anchored to SSA orders of magnitude, but the parameters (especially  $k$ ,  $x^p$ ,  $x^c$ ,  $\tau$ ) were chosen to match the theoretical fixed-point configuration rather than estimated from micro data; estimation is left for future work. These extensions are left for future work. These extensions are left for future work.

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