The Macroeconomic Consequences of Malaria Eradication in Sub-Saharan Africa

Minki Kim*

University of Mannheim

February 2026

Abstract

This paper quantifies the macroeconomic consequences of malaria eradication using a structural model in which individuals adjust fertility and educational investment in response to disease through the quantity–quality tradeoff. The model is disciplined by empirical evidence from an anti-malaria campaign in Tanzania. Eradication raises GDP per capita substantially, nearly five times larger than prior estimates. Gains arise because healthier children acquire more human capital per year of schooling, and parents increase investment per child by reducing fertility. The results imply a potentially large role for childhood disease in cross-country income differences and support accelerating the deployment of malaria vaccines.

Keywords: Malaria, fertility, childhood human capital, quantity-quality trade-off, cross-country income differences.

*I am deeply grateful to my advisors, David Lagakos and Tom Vogl. I thank Titan Alon, Natalie Bau, Julieta Caunedo, Doug Gollin, Joe Kaboski, Tatjana Kleineberg, Munseob Lee, Tim Lee, Craig McIntosh, Todd Schoellman, Michèle Tertilt, Mitchell VanVuren, David Weil, Anson Zhou, as well as seminar participants at UCSD macro seminar, Yonsei macro workshop, KAEA 2022 Job Market Conference, 2022 Yale Y-Rise conference, 2023 CSAE, 2024 STEG Annual Conference, York University, National University of Singapore, University of Edinburgh, University of Essex, Queen Mary University of London, Florida State University, Hunter College, FAU Nürnberg, and the University of Mannheim for helpful comments. Financial support from the Structural Transformation and Economic Growth (STEG) and the German Research Foundation (DFG) through CRC TR 224 (Project A03) is gratefully acknowledged. Earlier versions of this paper were circulated under the title *"How Will a New Malaria Vaccine Shape Africa's Economic Future? A Macroeconomic Analysis."* Contact: minki.kim[at]uni-mannheim.de.

1. Introduction

Despite substantial investments in preventive technologies and treatment, malaria remains a leading cause of child mortality and a barrier to human capital accumulation in sub-Saharan Africa. In 2020 alone, malaria claimed more than 600,000 lives, disproportionately among African children under five years old (World Health Organization, 2021). Even for surviving children, malaria infection can lead to lasting cognitive impairments, increased school absenteeism, and reduced classroom attention, all of which worsen learning outcomes. This raises an important question: How large are the potential income gains from eradicating malaria? Answering this question is crucial for evaluating the returns to health investments in developing countries and understanding the sources of cross-country income differences.

To accurately quantify the potential income gain, it is essential to understand how individuals respond to improved health. Since malaria adversely affects children's learning outcomes, eradicating malaria would directly influence parental decisions about educational investments, as healthier children learn more effectively in school. Changes in population size, also an important factor in determining per-capita income, depend on fertility responses to disease environment. Ignoring these behavioral adjustments can substantially bias estimates of the economic impact of health improvements. Previous macroeconomic studies relying on an accounting framework (Shastry and Weil, 2003; Weil, 2007; Bloom, Canning, Kotschy, Prettner, and Schünemann, 2024) typically overlook these endogenous behavioral responses.

This paper provides a quantitative general equilibrium framework to evaluate the macroeconomic gains from malaria eradication, explicitly accounting for how individuals respond to improved health. The model incorporates behavioral adjustments such as fertility decisions and investments in children's human capital. Aggregate outcomes, such as population size and educational attainment, are generated in equilibrium through aggregating individual responses. I find that eradicating malaria alone would increase per-capita income by 9.5%, roughly five times larger than estimates reported in previous studies. The model also suggests that failing to account for individual responses and general equilibrium effects significantly underestimates the economic benefits of health interventions, such as vaccination against malaria.

To this end, I build a general equilibrium, heterogeneous-agent, overlapping generations model featuring endogenous fertility, parental investment in children's human capital, and malaria. Malaria is introduced as an idiosyncratic childhood health shock

characterized by two key dimensions: mortality, reflecting deaths caused by malaria, and morbidity, representing cognitive impairment that dampens human capital accumulation. Unlike previous macroeconomic studies that used years of schooling as a sole measure of human capital, the model has a richer concept of human capital beyond just years of schooling. The epidemiological literature highlights that malaria negatively affects human capital not merely through reduced years of schooling, but also through cognitive impairments and reduced classroom attention. Reflecting this fact, the concept of human capital in the model incorporates both years of schooling and the amount of human capital acquired per year of schooling, referred to as the *quality* of human capital in Manuelli and Seshadri (2014). Thus, eradicating malaria enhances human capital accumulation not only by increasing schooling years but also by improving learning capacity per year spent in school. The model also features intergenerational dynamics, capturing that healthier children subsequently adjust their fertility and invest more in their own children's education (Daruich, 2026).

A key feature of the model is the interaction of childhood disease with parental decisions through a quantity-quality tradeoff (Barro and Becker, 1989). Suppose the risk of malaria is reduced, which is represented by lower mortality and morbidity in the model. This generates two opposing effects. On the one hand, reduced child mortality lowers the cost of child *quantity*, encouraging parents to have more children. Given limited household resources, this increase in fertility implies lower educational investment per child. On the other hand, higher returns to education due to reduced morbidity lower the cost of child *quality*, incentivizing parents to have fewer children and concentrate more educational investment in each child. While it is not ex-ante obvious which effect dominates, these competing mechanisms can be summarized by two sufficient statistics that can be empirically estimated: fertility and education elasticities with respect to malaria prevalence.

I validate the model's main behavioral mechanism using the empirical evidence from a large-scale antimalaria campaign in sub-Saharan Africa, the Roll Back Malaria (RBM) campaign (Kuecken, Thuilliez, Valfort et al., 2021). Initiated in 2003, the RBM campaign significantly reduced malaria prevalence by aggressively distributing preventive technologies against malaria, such as insecticide-treated bednets. The campaign successfully reduced the probability of infant mortality (1 p.p.). In response to that, women who were exposed to the program reduced the annual probability of birth (by 0.4 p.p.), and children exposed to the program experienced increased educational attainment by 0.4 years. I feed the empirically observed declines in malaria-related mortality and mor-

bidity during the RBM period into the model calibrated to Tanzania, and then compare the model's predicted changes in fertility and schooling to the corresponding empirical estimates. The simulated predictions from the model are well aligned with the reduced-form evidence.

Using the calibrated model, I then quantify the aggregate gains from malaria eradication. Recent scientific advancements have yielded two malaria vaccines that are up to 75% effective at preventing malaria infections among children under five, and the WHO has initiated partial rollouts in selected countries. Motivated by these developments, I compute the economic impact of a nationwide vaccination policy, assuming this reported vaccine efficacy (75%), on fertility, educational attainment, and per-capita income. The first exercise examines short-run effects, focusing on immediate, one-generational impacts reflecting direct benefits to vaccinated children. The second exercise evaluates the long-run consequences, accounting for intergenerational dynamics and long-run wage adjustments. Conceptually, this second exercise is more akin to an accounting exercise, providing a benchmark for the quantitative importance of childhood disease in explaining cross-country income differences. To this end, the long-run analysis also reports outcomes assuming a hypothetical 100% vaccine efficacy, to gauge the full potential benefit of complete malaria eradication.

The results show a sizable increase in per-capita income in both the short and long run, much larger than estimates from previous studies. In the short run, the model predicts a per-capita income gain of 6.9% within one generation. This income gain is primarily driven by improved childhood human capital accumulation, particularly through larger human capital acquired per year of schooling. The model suggests that the changes in the *quantity* of education are not the primary source of income gain. Instead, improved learning capacity alone accounts for 70% of the total income increase. This finding aligns well with the conclusions of macro-development literature, which emphasizes low schooling quality, rather than shorter schooling years, as the primary cause of low human capital in developing countries (Hanushek and Woessmann, 2008; Schoellman, 2012). The decomposition exercise also highlights the quantitative importance of the quantity-quality tradeoff. When malaria risk is reduced, parents respond by lowering fertility and increasing investment in their children's human capital. Shutting down this fertility adjustment channel reduces the income gain by half, to 3.09%.

While the short-run analysis is informative on gauging the immediate impacts of eradication, the long-run exercise sheds light on how heavy the burden of malaria is in explaining the income differences between high- and low-income countries. The model

predicts that complete eradication of malaria in the long run would increase per-capita income by 9.5% as intergenerational dynamics amplify the initial gains. These per-capita income gains are around five times larger than estimates reported in Ashraf, Lester, and Weil (2008). Even assuming the currently available vaccine with 75% efficacy generates substantial long-run benefits, increasing per-capita income by 6.7%. Decomposing the effects into mortality and morbidity components, the model reveals that the reduction in morbidity, rather than mortality, is the key driver of income gains. Reducing mortality alone—without alleviating malaria's adverse effects on learning capacity—leads per-capita income to *decline* by 0.4% due to higher population pressure. This finding aligns with Acemoglu and Johnson (2007), which documents that historical increases in life expectancy only raised population without boosting per-capita income.

I conclude by conducting a cost-benefit analysis of the vaccination policy, comparing model-implied income gains to estimated vaccination costs from the epidemiological literature. The model indicates that annual costs of vaccination program can be fully recovered through increased income. Given that the recently developed malaria vaccine has demonstrated 75% efficacy, these results strongly support the cost-effectiveness of mass vaccination. Vaccine rollout has been delayed due to financial challenges, primarily because malaria-endemic countries lack the fiscal capacity to independently finance deployment.[1][2] The findings of this paper suggest that delaying vaccine rollout not only costs lives but also postpones the realization of substantial macroeconomic benefits.

Related Literature This paper contributes to a vast literature examining the relationship between health, human capital, and cross-country income differences (Caselli, 2005; Schoellman, 2012, 2016). As Lagakos and Schoellman (2026) points out, evidence on the role of health in explaining cross-country income differences remains limited, and the existing development accounting estimates typically attribute to health a positive but modest share of cross-country income gaps. For instance, Shastry and Weil (2003) and Weil (2007) find modest impacts of eliminating health disparities on narrowing income gaps across countries, and Acemoglu and Johnson (2007) finds little evidence that improved health during the 1940s raised per-capita income in subsequent years. For malaria, Ashraf et al. (2008) argues that eradication in Zambia would raise only about 2% in GDP per capita. This paper advances the literature in two ways. First, I provide new quantitative results suggesting that eliminating childhood diseases in the

[1] The vaccine rollout heavily relies on foreign aid. Organizations such as Gavi, the WHO, and the U.S. President's Malaria Initiative have played central roles in financing malaria vaccine distribution.

[2] Duncombe, Elabd, and Sandefur (2024) estimates that, at the current funding pace, universal vaccination coverage for children under three will not be achieved until around 2035, resulting in approximately 2.5 million preventable child deaths in the meantime.

developing world can substantially narrow cross-country gaps in output per capita. Second, I provide a fully micro-founded structural model that explicitly incorporates individuals' behavioral responses to diseases, allowing aggregate outcomes to arise endogenously from optimizing behavior. While this paper focuses on malaria, the framework can easily be generalized to incorporate other childhood diseases. Previous studies typically abstract from these endogenous behavioral responses and instead extrapolate micro-level estimates through an aggregate production function; while this accounting approach is useful, ignoring behavioral adjustments can bias aggregate predictions.[3]

This paper also builds on a growing body of research in macroeconomic development that uses structural models to understand the general equilibrium effects of development policies, by combining the model with reduced-form empirical estimates (Buera, Kaboski, and Townsend, 2023; Todd and Wolpin, 2023). In this vein, the quantitative exercises of this paper are related to those of Brooks and Donovan (2020), which use the reduced-form evidence on the effects of rural bridge building to discipline the general equilibrium model of transportation infrastructure. Similarly, Buera, Kaboski, and Shin (2021) replicates the difference-in-differences estimates from microfinance initiatives within a general equilibrium model to investigate general equilibrium effects. Other studies using this approach have investigated power outages and firm productivity (Fried and Lagakos, 2022), managerial delegation (Akcigit, Alp, and Peters, 2021), publicly funded secondary education (Fujimoto, Lagakos, and Vanvuren, 2025), and rural-urban migration (Lagakos, Mobarak, and Waugh, 2023). This paper is the first to extend this methodology to study the macroeconomic effects of health improvement.

Lastly, the theoretical framework for the quantity-quality tradeoff in this paper is related to those in Kalemli-Ozcan, Ryder, and Weil (2000); Kalemli-Ozcan (2003); Soares (2005); Doepke (2005) and Aaronson, Lange, and Mazumder (2014), all of which focused on the role of mortality decline or improved schooling opportunity in human capital investment and fertility. I advance this literature by disciplining the quantitative magnitude of the quantity-quality tradeoff using reduced-form evidence from the RBM campaign. The quantitative model of this paper is also related to those in Zhou (2025) and Daruich (2026), who study the long-run effects of education subsidy and family policies, respectively, using a general equilibrium, heterogeneous-agent overlapping generations model. However, none of these previous studies considers diseases as a factor disrupting childhood human capital accumulation, which the model suggests to be quantitatively important.

[3]For example, Ashraf et al. (2008) assumes that population growth rates revert exogenously to pre-eradication levels following malaria eradication.

2. Model

This section introduces a quantitative overlapping-generations model designed to study the macroeconomic consequences of childhood disease through endogenous fertility and parental investments in children's human capital. Parents value their children's human capital out of altruism (Barro and Becker, 1989)[4]. I incorporate malaria as an exogenous health shock affecting children by increasing mortality risk and lowering returns to education.[5] Parents choose fertility and children's educational attainment, where these decisions are affected by children's health status. Additionally, motivated by evidence from Khanna (2023), I model workers with different education levels as imperfect substitutes, such that increasing the supply of educated workers depresses their relative wages. Section 2.4 introduces a simplified version of the model to highlight the core mechanisms.

2.1. Environment

Demographics and Environment Time is discrete, and each model period represents six years. The economy consists of L distinct locations/regions, each characterized by a different malaria prevalence. The population share of location $\ell \in 1, 2, \ldots, L$ is denoted by p_ℓ. This regional heterogeneity in malaria prevalence is introduced primarily to match observed spatial variations in malaria prevalence, which will later be exploited in parameter estimation. The economy is populated by overlapping generations of households, each living for 11 periods (66 years), consistent with life expectancy in Tanzania, the setting used for calibration. Figure 1 illustrates the lifecycle and family structure. An individual's age is denoted by $j \in 0, 1, \ldots, 12$.

Each household consists of a parent and their cohabiting children, if any. Children live with their parents and make no decisions until age 18, when they leave home and become independent adults with zero initial assets. Throughout adulthood, individuals make consumption and savings decisions. Borrowing is not permitted, but individuals

[4] A similar framework is adopted in Manuelli and Seshadri (2009) and Roys and Seshadri (2017).

[5] Other macro-development models have studied the interaction between human behavior and disease spread. For example, Greenwood, Kircher, Santos, and Tertilt (2019) and Alemán, Iorio, and Santaeulàlia-Llopis (2024) introduce models of the HIV epidemic in which sexual behavior endogenously influences disease transmission. In contrast, malaria is transmitted by mosquitoes, leaving less scope for human behavior to influence its spread.

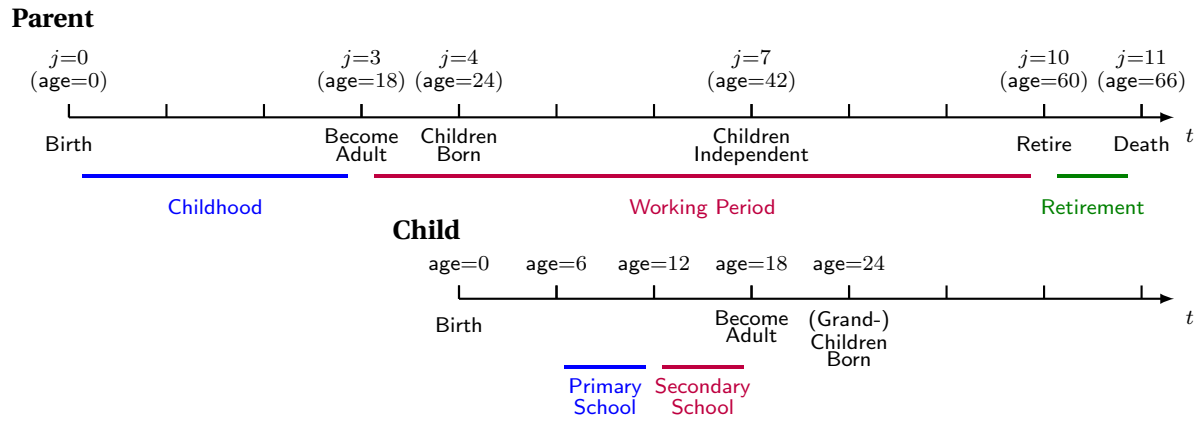


Figure 1: Life cycle, family structure, and stages of life

may accumulate savings at an exogenous interest rate r.[6] At age 24, individuals decide how many children to have and become parents, conditional on having children.

Parents choose their children's educational attainment by deciding whether to send them to school. Schooling decisions occur in two stages: primary school at age six and secondary school at age twelve. Children's initial human capital upon reaching adulthood thus directly depends on parental investment choices. Once children become independent adults, there is no further interaction between parents and children, and the children's human capital remains fixed throughout adulthood. Individuals retire at age 60 and consume all of their remaining assets until death at age 66, which is chosen to match the average life expectancy in many sub-Saharan African countries.

There are five exogenous sources of heterogeneity in the model. The first is a standard idiosyncratic, uninsurable labor productivity shock v_t, which introduces heterogeneity in earnings for working-age adults. I assume this shock is i.i.d. and log-normally distributed each period:

$$\log v_t \overset{\text{iid}}{\sim} N(0, \sigma_v).$$

The second is the fertility taste ϕ, which captures variations in fertility behavior not explained by the model's mechanisms. I assume that ϕ follows a standard Gumbel distribution with scale parameter θ. Third, children are born with heterogeneous learning ability z_k, which is imperfectly correlated with parental ability. Fourth, children face a health shock that determines whether they contract malaria. This shock is drawn once in early childhood (at age six) and reduces the returns from schooling in subsequent

[6] I abstract from domestic capital markets since many countries relevant to the model are small open economies with limited financial market development. I also omit endogenous labor supply and retirement decisions due to high labor participation rates and short retirement periods typical in developing countries.

periods of childhood, and the likelihood of the health shock depends on the location where the child is born, the last dimension of heterogeneity. Below, I illustrate in detail how learning ability and health shocks are drawn and how they interact with parental education decisions.

Learning Ability Children's learning ability is stochastic but partially inherited from their parents. Specifically, learning ability within a household follows an AR(1) process:

$$\log z_k = \rho_z \log z_p + \varepsilon_z,$$

where z_k and z_p denote the learning abilities of children and parents, respectively, and ε_z is an idiosyncratic i.i.d. shock. Parents observe their children's learning ability at age six, before deciding whether to send them to primary school. Learning ability remains constant throughout an individual's lifetime and is identical among siblings. Following the literature on early-childhood human capital accumulation (Cunha and Heckman, 2007; Lee and Seshadri, 2019), I interpret learning ability as an inherited capability capturing intergenerational persistence not explained by economic behavior.

Malaria in Early Childhood In addition to learning ability, children draw an idiosyncratic health shock at age six.[7] The health shock has two dimensions: mortality and morbidity. Mortality risk is equivalent to child mortality (death under age five), while morbidity reflects malaria's detrimental effects on human capital accumulation through cognitive impairment and comorbidities. Mortality risk is characterized by a survival probability; specifically, the probability that an age-six child born in location ℓ survives to the next period is denoted by χ^d_ℓ. This mortality risk encompasses deaths from all causes, not only malaria. Since the analysis focuses on reductions in mortality specifically due to malaria, I introduce the parameter μ, which represents the fraction of child mortality attributable to malaria: the child mortality rate due to malaria is $\mu \times \chi^d_\ell$.

Morbidity risk is represented by a proportional reduction in learning ability, reflecting that malaria infection worsens learning outcomes during and after bouts of illness (Fernando, Rodrigo, and Rajapakse, 2010; Chen, Clarke, Gosling, Hamainza, Killeen, Magill, O'Meara, Price, and Riley, 2016). Specifically, the morbidity shock is denoted by m:

$$m = \begin{cases} 1 & \text{w/ probability } 1 - \chi^m_\ell \\ \underline{m} & \text{w/ probability } \chi^m_\ell \end{cases}$$

[7] The assumption that the health shock is realized at age six captures that parents learn their children's health status by the time they enter primary school.

where ℓ denotes the location and $\underline{m} < 1$ indicates the reduced learning ability of a child hit by the adverse morbidity shock, and the status of a healthy child is normalized to one. The probability of experiencing the negative morbidity shock is denoted by χ_ℓ^m. I discuss how reduced learning ability affects children's human capital accumulation in the following subsection.

Since malaria is modeled as an exogenous shock, the model does not explicitly include parents' endogenous preventive measures against their children's malaria infection, such as purchasing bednets. Numerous studies document that more educated parents engage in greater preventive behavior, thereby lowering their children's malaria risk. To reflect this preventive behavior of educated parents, I assume that children of parents who received secondary education experience reductions in malaria-caused mortality and morbidity probabilities by a factor of $1-\xi$. This formulation parsimoniously captures how parental education mitigates malaria risks.[8]

Schooling and Human Capital After observing their children's learning ability z_k and the realized health shock m, parents decide whether to send their six-year-old children to primary school, and subsequently, their twelve-year-old children to secondary school. Schooling deterministically increases human capital as follows:

$$h_k = \begin{cases} m z_k \eta_S \eta_P & \text{if attend secondary school} \\ m z_k \eta_P & \text{if attend primary school} \\ 1 & \text{if no schooling} \end{cases}$$

where η_s is the deterministic increase in human capital from school $s \in \{P, S\}$. As the expression shows, children's human capital in adulthood depends jointly on their learning ability, disease status, and parental schooling investments. Schooling decisions are sequential: if parents choose not to send their child to primary school, the child loses the opportunity to attend secondary school in the subsequent period.

Sending children to school is costly, and these costs consist of two components. The first is the monetary cost, which includes tuition, uniforms, and supplies such as textbooks. These are represented in the model by per-child schooling fees p_P and p_S for primary and secondary schools, respectively, which enter the household budget constraint. The second component is the opportunity cost of schooling, which arises from

[8]To the best of my knowledge, Gollin and Zimmermann (2007) is the only paper that develops a macroeconomic model of parents' endogenous preventive behaviors against malaria. However, their focus is primarily on the adoption and efficacy of the preventive technology, rather than on the macroeconomic impacts of eradication.

forgone labor income when secondary-school-age children attend school rather than work, as children of that age are closer to entering the labor force. Parents must give up a period of child's work, and thus income, in order to send them to secondary school. This opportunity cost introduces parents a tradeoff between children's future human capital and current household income. It also implies that the effect of reducing malaria on secondary school enrollment is not straightforward, as healthier children are not only better students but also potentially more productive workers (Bau, Rotemberg, Shah, and Steinberg, 2024).

Production and Aggregation A representative firm operates competitively in the labor market, producing a single consumption good using skilled (secondary education completed) and unskilled (below secondary education) labor inputs. The following CES aggregator represents the production function:

$$Y = A\left[(H_U + H_P)^{\frac{\lambda-1}{\lambda}} + (H_S)^{\frac{\lambda-1}{\lambda}}\right]^{\frac{\lambda}{\lambda-1}}, \quad \lambda \in (0, \infty)$$

where H_s denotes aggregate efficiency units for the schooling groups U (uneducated), P (primary-completed), and S (secondary-completed), and λ is the elasticity of substitution between skilled and unskilled labor. Equilibrium wages per efficiency unit for each skill group are:

$$w_U = A\left[(H_U + H_P)^{\frac{\lambda-1}{\lambda}} + (H_S)^{\frac{\lambda-1}{\lambda}}\right]^{\frac{1}{\lambda-1}} (H_U + H_P)^{-\frac{1}{\lambda}}$$
$$w_S = A\left[(H_U + H_P)^{\frac{\lambda-1}{\lambda}} + (H_S)^{\frac{\lambda-1}{\lambda}}\right]^{\frac{1}{\lambda-1}} H_S^{-\frac{1}{\lambda}}$$

Given wages w_U and w_S are defined per efficiency unit, the labor income of an individual with skill level s, human capital h, and idiosyncratic productivity shock v is:

$$y(h, v, s) = hvw_s.$$

2.2. Household's Decision Problems

The model's individual's adulthood can be broadly divided into three stages: periods when they live without children, periods when they live with their children, and periods when they become alone again after their children become independent. Individuals solve a consumption-savings optimization problem throughout their lifetime. The util-

ity function for consumption is given as:

$$u(c) = \frac{c^{1-\gamma}}{1-\gamma}$$

When living with children, they also make educational investment decisions. In this subsection, I outline the individual's optimization problem for each period of life. Borrowing constraints apply in all periods ($a' \geq 0$). Throughout this section, child-specific variables are denoted by subscript k, and future variables by primes.

Age 18 ($j = 3$)**: Independence** Individuals leave their parents and form new households at age 18 with zero initial assets. Their state variables are human capital h, schooling level s determined during childhood, and learning ability z. Although learning ability does not directly affect their earnings, it is a state variable because their children's learning abilities will depend on it. An individual's income is determined by their human capital h, skill-specific wage rate w_s, and an idiosyncratic income shock v drawn at the beginning of the period. Since individuals do not yet have children at this stage, they solve a standard consumption-savings problem:

$$\begin{aligned} V_3(a,s,h,v) = \max_{c,a'} u(c) + \beta \mathbb{E}\Big[V_4\left(a',s,h,v'\right) \Big] \\ \text{subject to} \\ c + a' \leq w_s h v + (1+r)a \end{aligned} \tag{1}$$

where r is the period interest rate.

Age 24 ($j = 4$)**: Fertility** At this stage, individuals choose how many children to have, and those who choose to have children become parents. Fertility decisions are discrete; individuals choose the number of children n, where $n \in \{0, 1, 2, \cdots, \bar{N}\}$, by choosing the number n^* that provides the highest level of utility:

$$V_4 = \max\{V_4^0 + \phi_0, V_4^1 + \phi_1, \cdots, V_4^{\bar{N}} + \phi_{\bar{N}}\}$$

where V_4^n represents the value of having n number of children. For each number of children n, I also introduce a taste shock ϕ_n, which is drawn *i.i.d.* from a Gumbel distri-

bution with variance σ_n. The value of having n children can be expressed as follows:

$$
\begin{aligned}
V_4^n(a,s,h,z,v) = \max_{c,a'} u(c) + \beta \mathbb{E}\Big[V_5\left(a', s, h, v', z_k', m', n'\right) \Big] \\
\text{subject to} \\
c + a' \leq w_s h v (1 - t(n)) + (1+r)a \\
t(n) = 1 - e^{\omega n}
\end{aligned}
\tag{2}
$$

Raising children is costly in terms of time. Specifically, $t(n)$ represents parental time devoted to child-rearing, reducing income available for consumption and savings. The total time cost increases with n and h, reflecting that forgone work hours are more costly for high-income households than low-income ones. Lastly, note that the expectation is taken over the number of *surviving* children n' in the next period, as the mortality risk is realized at the beginning of the next period ($n' \leq n$).

Age 30 $(j = 5)$**: Ability, Health Shock and Primary Education** At the beginning of this period, parents observe their children's learning ability z_k, the realization of the malaria morbidity shock m, and an idiosyncratic income shock v.[9][10] Additionally, the mortality shock is realized at the start of the period, determining the number of *surviving children* n. Specifically, the probability that exactly n out of N children survive is:

$$
f(n;N) = \binom{N}{n} (\chi_\ell^d)^{N-n} (1 - \chi_\ell^d)^n
$$

Parents then decide whether or not to send their surviving children to primary school ($e = 1$ for schooling, $e = 0$ otherwise). If children attend school, a per-child primary schooling fee p_P is deducted from the household budget constraint. Children who do not attend school receive no education, leaving their human capital at the initial level. In contrast, if children attend school, their human capital in the next period increases according to the schooling efficiency parameter η_P, augmented by their learning ability z_k and the morbidity shock m. With n surviving children, a parent's value at this stage

[9] Malaria primarily affects children under age five, so the timing assumption is equivalent to parents observing their children's malaria infection up to age six, upon which they base schooling decisions.

[10] A considerable body of literature documents that parents in malaria-endemic countries are well aware of the causes, symptoms, and consequences of malaria. See Tarimo, Lwihula, Minjas, and Bygbjerg (2000) and Montgomery, Mwengee, Kong'ong'o, and Pool (2006) for studies conducted in Tanzania. In a survey conducted in the Democratic Republic of the Congo, all participants recognized that malaria has adverse effects on the health of schoolchildren, including anemia, school absenteeism, convulsions, and poor school performance (Matangila, Fraeyman, Kambulu, Mpanya, da Luz, Lutumba, Van Geertruyden, and Bastiaens, 2017).

can be expressed as follows:

$$
\begin{aligned}
V_5(a,s,h,v,z_k,m,n) = \max_{c,a',e\in\{0,1\}} u(c) + \beta\mathbb{E}\Big[V_6\left(a',s,h,v',s'_k,h'_k,z_k,m,n\right)\Big] \\
\text{subject to} \\
c + a' + enp_P \leq w_s hv(1-t(n)) + (1+r)a \\
h'_k = eh_k\eta_P z_k m + (1-e)h_k
\end{aligned}
\tag{3}
$$

Age 36 ($j = 6$): Secondary Education, Preferences over Child Quantity and Quality Parents who sent their children to primary school in the previous period now decide whether to continue sending them to secondary school. Due to the sequential nature of schooling, parents who did not send their children to primary school do not have the option to send them to secondary school ($e = 0$ for them). The value of parents with secondary school-age children is:

$$
\begin{aligned}
V_6(a,s,h,v,s_k,h_k,z_k,m,n) = \max_{c,a',e\in\{0,1\}} u(c) + \beta\mathbb{E}\Big[V_7\left(a',s,h,v'\right)\Big] + \beta b(n)\nu(h_k) \\
\text{subject to} \\
c + a' + np_S e \leq w_s hv(1-t(n)) + nw_U h_k v(1-e) + (1+r)a \\
h'_k = eh_k\eta_S z_k m + (1-e)h_k
\end{aligned}
\tag{4}
$$

The term $nw_{s_k}h_k\nu(1-e)$ in the budget constraint represents the opportunity cost of schooling in the form of lost labor income. If children do not attend school ($e = 0$), they work as low-skill workers and the earnings contribute to the household budget.

The last term in the value function represents parental altruistic preferences over child quantity and quality. I model altruism as in Barro and Becker (1989), where parents directly value the human capital their children will possess as adults (h_k) through function $\nu(\cdot)$, weighted by the discounting function $b(n)$.[11] I assume the utility for child quality has the following functional form: $\nu(x) = \frac{x^{1-\gamma}}{1-\gamma}$.

Age 42 to 66 ($j = 7-11$): After Children's Independence At the beginning of period 7, children become independent and leave their parents. After this point, there is no further interaction between parents and children, and parents solve a simple consumption-savings problem. The value function for working-age individuals after the child-rearing

[11] This approach to modeling altruism significantly reduces the computational burden. Earlier versions of the paper featured a full dynastic altruism structure and yielded similar quantitative results. Daruich and Kozlowski (2020) and Zhou (2025) also develop quantitative models with endogenous fertility, where altruism is introduced in a similar way.

stage is identical to (1) until age 60, when individuals retire and use their assets to finance consumption.

2.3. Competitive Equilibrium and Balanced Growth Path

In this economy, population growth is endogenous due to endogenous fertility. Therefore, I focus on a balanced growth path, defined as an equilibrium where the aggregate population growth rate and the distribution of households' states remain constant over time. Appendix B contains the formal definition of the recursive competitive equilibrium and balanced growth path.

2.4. Illustration: Impact of Malaria on Schooling and Fertility

The quantitative model features rich interactions between fertility, children's human capital, and malaria, captured by the quantity-quality tradeoff. This section presents a simplified version of the model that illustrates the core mechanism. Consider a model with continuous fertility choice, in which parents choose the number of children, denoted by b. Each birth is associated with a time cost of p, representing the fraction of forgone wages due to child-rearing. Given a survival probability of $1-\chi_d$, the number of surviving children n is $(1-\chi_d)b$.

Parents can invest in the education of their surviving children through educational spending e. A child's income is proportional to the level of human capital they possess, and the education provided by parents is converted into the child's human capital by a concave, increasing function $h(e)$. As in the quantitative model, parents have incentives to provide education for their children because they derive utility from their children's human capital in addition to their own consumption. The returns to education depend on whether the child contracts malaria. With probability χ_m, the child contracts malaria, and the amount of human capital accumulated is penalized by parameter m, where $m < 1$ represents the detrimental effects of malaria on children's human capital accumulation as in the quantitative model. The parents' utility can be described as follows:

$$
\begin{aligned}
V(a,h) &= \max_{c,e,b} u(c) + \beta b(n)\nu(h_k) \\
&\text{subject to} \\
n &= (1-\chi_d)b \\
c + en &= wh(1-pb) \\
h_k &= (1-\chi_m)h(e) + \chi_m m h(e)
\end{aligned}
$$

The simplified model provides useful intuition on how a reduction in malaria risk affects parents' fertility and educational investment, which carry over to the quantitative model. In the model, malaria eradication is represented by reductions in both mortality risk (lower χ_d) and morbidity risk (lower χ_m). Throughout this subsection, I assume $w = 10$, $\lambda = 0.5$, $\gamma = 0.5$, and $\tau = 0.2$. The detrimental effect of malaria, m, is set to 0.5, and the functional form used for $h(e)$ is $\sqrt{e}$. Figure 2 plots the optimal fertility levels for varying degrees of mortality and morbidity probabilities. The square dot denotes the baseline calibration, where the mortality rate (χ_d) is set to 0.2 and the morbidity rate (χ_m) is set to 0.7. The horizontal dotted line indicates a reduction in morbidity risk from its baseline value, while the vertical dotted line indicates a reduction in mortality risk.

Figure 2: Optimal Fertility Conditional on Malaria Prevalence

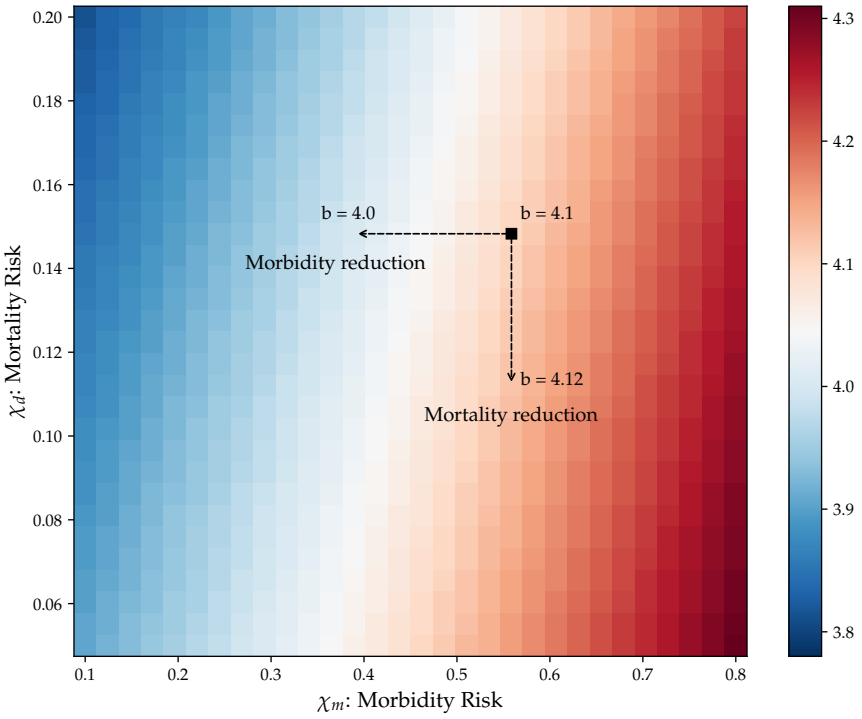


Figure 2 illustrates how fertility decisions respond to reductions in malaria risk. First, the vertical arrow indicates that lower mortality induces parents to have more children. The intuition behind this result is straightforward: since parents care about surviving children, reduced mortality lowers the effective cost of producing a surviving child. Because children are a normal good, this leads to higher fertility, as illustrated in the figure. Lower morbidity risk, however, has an offsetting effect. A reduced probability of contracting malaria implies higher returns to educational investment, as children acquire more human capital for a given level of parental investment. These higher returns en-

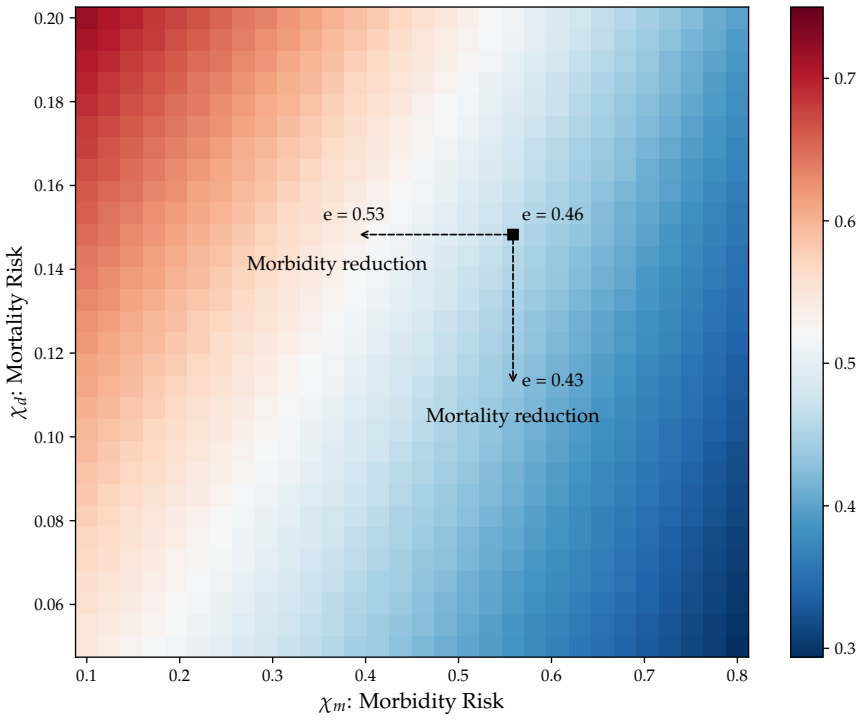
courage parents to reduce fertility and invest more in each child's education, resulting in lower fertility overall.

Figure 3a plots optimal educational investment for varying degrees of malaria prevalence. The educational investment response to changes in malaria prevalence is the mirror image of the fertility response shown previously. When the mortality rate declines, educational investment decreases; when the morbidity rate declines, educational investment rises. This occurs because child quantity and quality are substitutes within the household budget constraint. For instance, when parents decide to have more children, they allocate resources away from potential investments in education.

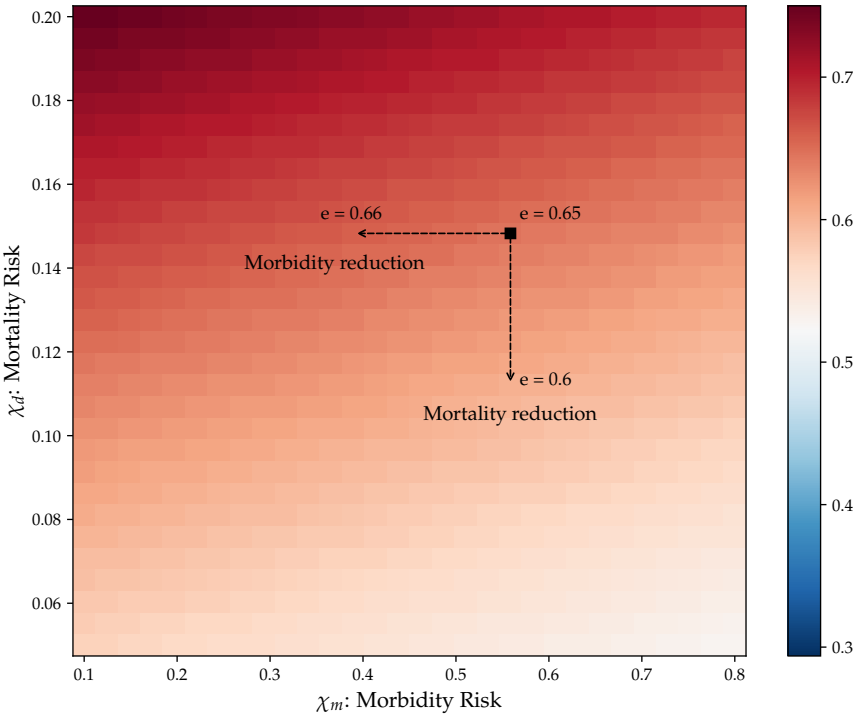
The main insight from the simplified model is that the extent to which fertility and educational investment respond to reduced malaria prevalence depends on the severity of malaria's adverse impact on human capital accumulation, captured by the parameter m. Figure 3b again plots optimal educational investment, but now assumes a lower severity of malaria's impact ($m = 0.9$) compared to the baseline ($m = 0.5$). Qualitatively, the responses to reductions in malaria prevalence remain consistent with the previous analysis: parents increase(decrease) educational investment in response to reduced morbidity(mortality) risk. However, the magnitude of these responses is significantly reduced. Intuitively, if contracting malaria has minimal negative consequences for children's human capital, then reducing malaria risk also has minimal effects on parental investment decisions. Thus, when the human capital penalty from malaria is small (high m), changes in morbidity risk have little influence on educational investment choices.

The simple model highlights how malaria eradication affects fertility and educational investment through the quantity-quality tradeoff. In the quantitative model, I set m to a reasonable value based on existing evidence on the human capital costs of malaria, and then let the model generate fertility and schooling responses to changes in malaria risk. Section 3 evaluates whether these model-implied responses are consistent with reduced-form evidence from the RBM campaign.

Figure 3: Optimal Educational Investment Conditional on Malaria Prevalence



(a) Optimal education, malaria highly detrimental ($m = 0.5$)



(b) Optimal education, malaria less detrimental ($m = 0.9$)

3. Parameterization

This section describes the parameterization of the quantitative model used to assess the macroeconomic consequences of malaria eradication. The model is parameterized to Tanzanian economy in 2002. Calibrating the model to a single economy ensures internal consistency because the target moments are drawn from a common demographic and institutional environment. Additionally, there are several reasons why Tanzania in the early 2000 is a suitable time and space for calibration. First, Tanzania is a representative economy whose fertility rate, malaria prevalence, and income level are close to the sub-Saharan African average. Figure A.4 and Table A.2 show that Tanzania is broadly representative of high-malaria countries in sub-Saharan Africa in terms of fertility, malaria prevalence, and GDP per capita.

Second, the choice of 2002 as the baseline year reflects the validation strategy. The model is validated by replicating the Roll Back Malaria (RBM) campaign, a large-scale antimalaria initiative launched in the early 2000s, by comparing the model's predicted responses of fertility and children's educational attainment to their empirical counterparts. Since the RBM campaign began in 2003, a pre-RBM period provides the appropriate baseline from which to introduce the observed reductions in malaria mortality and morbidity into the model. Tanzania is particularly well suited for this exercise, as it was among the earliest and most successful RBM recipients. Figures A.1 and A.2 show a sharp decline in malaria prevalence following the introduction of the campaign. Section 3.3 describes the RBM intervention and the empirical evidence in more detail.

The parameterization proceeds in three steps. First, a subset of parameters is taken from the existing literature. Second, the remaining parameters are calibrated to match key aggregate moments for Tanzania, using microdata when available and estimates from the literature otherwise. Third, I validate the model by comparing the model-implied fertility and schooling responses to the RBM intervention to the corresponding empirical estimates. These RBM responses are not targeted in calibration and are used only to assess whether the model generates quantitatively plausible behavior.

3.1. Exogenously Chosen Parameters

Table 1 summarizes the set of parameters that are chosen exogenously. These parameters fall into two categories. The first includes parameters that are standard in the macroeconomic literature. The second comprises malaria-related parameters. These parameters are either drawn from epidemiological and health studies or calculated from the data.

Table 1: Exogenously Chosen Parameters

Parameters	Description	Value				Source
A.Economic Parameters						
β	Annual discount rate	0.96				Standard value
r	Annual interest rate	1.02				Deposit interest rate
$\bar{N}$	Max number of children	6				DHS 1999
γ	Inverse of IES	0.5				See text
λ	Substitutability b/w skills	6				Bils et al. (2022)
B. Parameters Related to Malaria						
Parameters Common across Locations						
L	Number of locations	4				Epidemiological literature
ξ	Educ. disparity in malaria risk	0.62				Ogbo et al. (2019)
μ	Share of malaria-caused death	0.17				See text
$\underline{m}$	Severity of malaria morbidity shock	0.85				See text
Location(ℓ)-specific parameters		*Malaria prevalence (PfPR)*				
		<20%	20-50%	50-75%	>75%	
χ_ℓ^d	Under-five Mortality rate	0.097	0.137	0.128	0.156	DHS 1999
χ_ℓ^m	Prob. catching malaria	0.132	0.325	0.580	0.765	Malaria Atlas Project
p_ℓ	Population share	0.313	0.443	0.216	0.028	2002 Census

Standard Parameters Panel A of Table 1 presents the standard parameters. The discount factor is set to 0.96^6, a conventional value from the macroeconomic literature, adjusted to account for each model period corresponding to six years. The annual gross interest rate is 1.02, reflecting the low levels of financial access and household savings rates typical in low-income economies (Donovan, 2021). The maximum number of children an individual can choose in the model is set at $\bar{N} = 6$, corresponding to a maximum of 12 children per household (since each parent in the model represents a household of two parents). This cap is chosen based on data from the 2002 Tanzania Census, which shows that 95% of households have fewer than 12 children.

It is worth elaborating briefly on the role of the parameter γ, which represents the inverse of the intertemporal elasticity of substitution (IES). While commonly interpreted in the macroeconomic literature as governing trade-offs between current and future consumption, in this life-cycle model with intergenerational linkages, γ also captures the degree of *intergenerational* elasticity of substitution. Specifically, it influences how parents value their children's utility relative to their own. A higher value of γ implies that parents' marginal utility of consumption decreases faster as they become wealthier, increasing the relative value of children. A value of γ less than one ensures that the utility function is positive everywhere, meaning that parents always derive positive utility from

having children, and implicitly assign zero utility to childlessness or losing a child.[12] I set γ equal to 0.5.

The elasticity of substitution between skilled and unskilled workers, λ, determines how much an increase in the supply of educated workers depresses their relative wages in general equilibrium. I set this parameter to 6, following Bils, Kaymak, and Wu (2024), who estimate the elasticity of substitution across different schooling groups.

Parameters Related to Malaria I set the number of locations (L) to four, consistent with the empirical literature classifying locations based on pre-intervention malaria prevalence. Each location is assigned to one of the four locations based on the location-level average malaria prevalence (PfPR) in 2001: low prevalence (PfPR $< 20\%$), medium-low prevalence (PfPR between 20–50%), medium-high prevalence (PfPR between 50–75%), and high prevalence (PfPR $> 75\%$). This discretization follows standard classifications in the epidemiological and malaria literature and provides a parsimonious representation of heterogeneity in malaria risk. At the same time, working with a small number of locations keeps the computation manageable while preserving the cross-sectional variation in disease prevalence. The location ℓ's population share, p_ℓ, are then calculated for each location from the 2002 Census.

Two sets of parameters are required for each location: mortality (probability of death) and morbidity (probability of contracting malaria). The mortality parameters, χ^d_ℓ, correspond to the under-five mortality rate, calculated using the 1999 Demographic and Health Survey (DHS), the closest DHS wave to the RBM campaign's start year. The parameter χ^d_ℓ includes mortality from all causes, not only malaria. To isolate malaria-attributed mortality, I use an external estimate of the share of under-five deaths attributable to malaria. According to the Institute for Health Metrics and Evaluation (Institute for Health Metrics and Evaluation (IHME), 2026), approximately 17.09% of under-five deaths in Tanzania in 2002 were attributable to malaria, which sets the value of the parameter μ to 0.17.[13]

I use location-specific averages of malaria prevalence (PfPR) for morbidity probabilities. PfPR in 2001 comes from the Malaria Atlas Project (Hay and Snow, 2006; Malaria Atlas Project, 2026), which report gridded estimates of malaria prevalence rate. I construct location-level PfPR by averaging the MAP pixels within each location. The region

[12] Allowing negative utility requires additional assumptions regarding the utility of childlessness and child mortality. See Jones and Schoonbroodt (2010) for further discussion.

[13] Statistics from household surveys are consistent with this high share of malaria in childhood mortality. Data from the Tanzania National Panel Survey (waves 2008–2014) indicate that malaria consistently accounts for more than 40% of identifiable childhood illnesses leading to hospitalization; see Table A.1.

with the highest prevalence has 76.5% of children contracting malaria, while the lowest prevalence region has only 13.2%. Regarding educational disparities in malaria risk, Ogbo, Osita, Akorede, Ifegwu, Lawrence, Emmanuel, Deborah, and Kingsley (2019) reports that children under five whose mothers had primary education or less had a 38% higher mortality risk compared to those whose mothers had secondary education or more. Accordingly, I set the mortality rate for children of secondary-educated parents to be 38% lower relative to children of parents with lower education (primary completed or no education) levels.

The last parameter, $\underline{m}$, captures malaria's adverse effect on children's human capital accumulation conditional on schooling. Its magnitude governs how educational investments respond to changes in malaria risk and, through the quantity–quality tradeoff, how fertility adjusts. As shown in Section 2.4, a lower value of $\underline{m}$ amplifies the increase in educational investment and induces a sharper decline in fertility following malaria reduction.

I set $\underline{m} = 0.85$, implying that malaria reduces returns to schooling by about 15% for affected children. Evidence from both epidemiology and economics supports values of $\underline{m}$ meaningfully below one. Epidemiological studies document sizable cognitive and educational impairments from childhood malaria exposure: Fernando, Gunawardene, Bandara, De Silva, Carter, Mendis, and Wickremasinghe (2003) show that Sri Lankan children with fewer than three malaria episodes score at least 15% higher on school and special examinations than children with more frequent infections, and Al Serouri, Grantham-McGregor, Greenwood, and Costello (2000) find that even a single episode is strongly associated with poor school performance in Yemen. Previous empirical studies on historical malaria eradication episodes are consistent with these magnitudes. For instance, Bleakley (2010) shows that cohorts exposed to malaria in early life earn 10–20% less as adults even after controlling for years of schooling, implying persistent human capital losses beyond schooling quantity. Venkataramani (2012) documents 0.1–0.3 standard deviation declines in adult cognitive outcomes, and Barofsky, Anekwe, and Chase (2015) reports similar patterns from the malaria campaign in Uganda in the 1960s.

3.2. Internally Calibrated Parameters

The remaining parameters are internally calibrated to match targeted moments from aggregate data and empirical estimates from the RBM campaign. Specifically, I jointly

estimate the following ten parameters:

$$\{\eta_P, \eta_S, p_P, p_S, \sigma_v, \sigma_z, \rho_z, \theta, \omega, \lambda_n\}$$

Table 2 reports the estimated values of the parameters. The ten parameters are calibrated primarily to match aggregate statistics from the pre-RBM Tanzanian economy. The following subsections describe the calibration and model fit in detail.

Table 2: Parameters and Estimated Values

Parameter	Value	Description
η_P	1.78	Human capital gain from primary education
η_S	1.25	Human capital gain from secondary education
p_P	0.19	Goods cost of primary education
p_S	0.62	Goods cost of secondary education
σ_v	0.02	Standard deviation of idiosyncratic income shock
σ_z	0.03	Standard deviation of the learning ability draw
ρ_z	0.83	Intergenerational persistence of learning ability
θ	1.45	Gumbel scale parameter of the fertility taste shock
ω	0.7	Curvature of time cost of childcare
λ_n	0.20	Curvature of the altruism function

Table 3 summarizes the calibration targets calculated from aggregate data, along with the corresponding model fit. Primary and secondary school completion rates are computed as the share of individuals between the ages of 18 and 30 whose highest education level is primary or secondary school, respectively, based on the 2002 Census. Tanzania introduced a universal primary education initiative in 1978, but as seen in the table, actual primary school completion rates remain far below the universal coverage, even among younger cohorts. The two schooling cost parameters, p_P and p_S, directly influence these low completion rates. The estimated parameter values for primary (p_P) and secondary (p_S) schooling fees are 0.19 and 0.62, respectively. On the calibrated balanced growth path, per-child primary schooling fee is 11% of the average household income, and the per-child secondary schooling fee is 27% of the average household income.[14]

The education premia that primary- and secondary-educated workers earn relative to the uneducated group are taken from Table 3 of Leyaro, Twumasi Baffour, Morrissey,

[14]Total annual education-related cost as a fraction of annual household expenditure is around 43% for households with children, from the Tanzania National Panel Survey (Pooled for waves 2008, 2010, 2012, 2014).

Table 3: Targeted Moments and Model Fit

Moments	Source	Data	Model
Education			
Primary completion rate (%)	Tanzania Census 2002	69.6	69.9
Secondary completion rate (%)	Tanzania Census 2002	13.1	13.1
Primary ed. wage premium (%)	Leyaro et al. (2014)	59.9	54.7
Secondary ed. wage premium (%)	Leyaro et al. (2014)	115.2	119.7
Differential Fertility			
Total fertility rate	Tanzania DHS 1999	5.90	5.82
Total fertility rate, unskilled parents	Tanzania DHS 1999	6.06	6.12
Total fertility rate, skilled parents	Tanzania DHS 1999	3.08	3.04
% Secondary parents with 8 or more children	Tanzania DHS 1999	5.78	6.20
Intergenerational Mobility and Inequality			
Primary-Secondary IGM	Alesina et al. (2021)	13.9	12.9
Gini coefficient	Younger et al. (2016)	0.38	0.44

and Owens (2014), who estimated these premiums using the 2001/2006 Tanzania Integrated Labour Force Survey.[15] The parameters related to these moments are η_P and η_S, which govern the increase in human capital from completing primary and secondary schooling, respectively. The estimated values are 1.78 for primary education and 1.25 for secondary education.

Several parameters jointly influence fertility behavior in the model. First, the intergenerational discount function, $b(n) = n^{\lambda_n}$, includes parameters that determine the degree of intergenerational altruism with respect to the number of children. These parameters are central to both fertility and education decisions. The estimated parameter value for λ_n is 0.2, chosen to match the overall fertility rate. Another key fertility parameter is ω, which governs the curvature of the time cost of child-rearing relative to the number of children. A higher time cost leads high-skilled parents to have fewer children, generating a stronger negative relationship between income and fertility. Consequently, I estimate ω to match the observed fertility differential between skill groups; its estimated value is 0.7. Lastly, the estimated Gumbel scale parameter θ for fertility preference is 1.45. This parameter captures variation in fertility choices unexplained by economic in-

[15] Multiple sources provide similar estimates of wage premiums. Leyaro et al. (2014) relies on the Integrated Labour Force Survey (ILFS) and urban worker samples. Joseph (2020) also uses the ILFS, with primary-educated workers as the reference group. Similarly, Mlacha and Ndanshau (2018) uses the ILFS and reports comparable results.

centives. In the data, approximately 5.8% of highly educated women have more than eight children; the model produces a comparable number.

The remaining two parameters relate to intergenerational mobility and income inequality in the model. The first is the AR(1) persistence parameter of children's learning ability, ρ_z, and the second is the variance of the shock in the AR(1) process, σ_z. Intuitively, higher persistence reduces intergenerational mobility. To discipline these parameters, I target a measure of intergenerational upward mobility estimated by Alesina, Hohmann, Michalopoulos, and Papaioannou (2021). Specifically, I match primary-to-secondary intergenerational mobility, which is defined as the likelihood that children born to parents with only primary education complete secondary schooling. The estimated persistence parameter ρ_z is 0.83. Lastly, parameter σ_v governs the standard deviation of the idiosyncratic human capital shock in adulthood. This parameter is calibrated by matching Tanzania's 2010 income Gini coefficient, as reported by Younger, Myamba, and Mdadila (2016), calculated using the Tanzania Household Budget Survey.

3.3. Validation: Replicating the RBM Campaign in the Model

Up to this point, the parameterization has focused on matching key aggregate and distributional characteristics of the pre-RBM Tanzanian economy. These aggregate moments are informative in that they place restrictions on the potential macroeconomic effects of malaria eradication. This subsection provides an out-of-sample validation exercise by assessing whether the calibrated model reproduces the reduced-form responses of fertility and schooling observed during the Roll Back Malaria (RBM) campaign, a large-scale antimalarial campaign that took place in early 2000s in sub-Saharan Africa.

3.3.1. The Roll Back Malaria (RBM) campaign

The Roll Back Malaria (RBM) Partnership was launched by the WHO, the World Bank, and the United Nations in 1998. It aimed to halve the global malaria burden between 2000 and 2010. Relative to earlier initiatives, a distinguishing feature of RBM was its large external funding. Total funding was approximately $4.6 billion between 2003 and 2009, and 81 of 108 malaria-endemic countries received support during this period (Johansson, Cibulskis, Steketee et al., 2010). Because sub-Saharan Africa accounted for around 85% of the global malaria burden, a large share of this funding was directed to African countries. The RBM strategy emphasized distribution of insecticide-treated nets (ITNs) and indoor residual spraying (IRS), both of which reduce malaria transmission. Global malaria-related deaths declined substantially through 2014 relative to the

early 2000s (World Health Organization, 2016).[16]

The empirical benchmarks are drawn from Kuecken et al. (2021), who study the RBM campaign across sub-Saharan Africa. Their analysis compares outcomes before and after the campaign across locations with different baseline malaria prevalence. Areas with higher pre-campaign malaria risk experienced larger declines in fertility and larger increases in children's educational attainment. Their estimates imply that the RBM campaign reduced the probability of infant mortality by 1 percentage point, reduced the probability of birth by 0.4 percentage points, and increased educational attainment by 0.4 years. These estimates summarize household responses to an exogenous reduction in malaria risk induced by RBM and therefore provide a benchmark for the model's behavioral responses. I replicate RBM in the model, rescale these empirical moments to be consistent with simulated outcome measures, and compare the reduced-form effects in the data and in the model.

3.3.2. Implementing the RBM campaign in the model

I introduce the RBM campaign as an unexpected, location-specific reduction in malaria risk. Malaria prevalence declined by 72% on average between 2001 and 2012, and the largest decline occurred in the highest-prevalence location (Figure A.3). Guided by these magnitudes, I reduce the morbidity probability χ_ℓ^m and the malaria-attributed component of mortality, $\mu \times \chi_\ell^d$, by location-specific proportions. The observed reductions in malaria risk are $\{61\%, 75\%, 75\%, 77\%\}$ across the four locations.

Just like the RBM campaign was an exogenous reduction in malaria risk, households in the model do not anticipate the campaign. I first simulate the balanced growth path under pre-RBM malaria risk and then introduce the RBM intervention as an unanticipated shock. Households then adjust their fertility and education decisions based on the new decision rules under the new malaria environment. These treated households correspond to those exposed to the RBM campaign in the data. Households on the initial balanced growth path (without the RBM shock) correspond to households (and their children) born before the RBM campaign.

3.3.3. Mapping Empirical Estimates to Simulated Moments

Using simulated data from economies with and without the RBM intervention, I then estimate regressions that mirror the empirical specification, relating lifetime fertility

[16] Nevertheless, the RBM campaign did not eradicate malaria in sub-Saharan Africa. Progress has slowed in recent years due to antimalarial drug and insecticide resistance as well as funding constraints, and some countries have experienced resurgence in malaria prevalence (World Health Organization, 2025). This supports vaccination as a potentially more effective tool for achieving eradication.

and completed schooling to post-campaign exposure and baseline malaria risk.

$$y_{i\ell c} = \beta_1 + \beta_2 \text{Post}_c + \beta_3 \left(\text{Post}_c \times \text{ malaria }_{2000\ell}\right) + \delta_\ell + \epsilon_{i\ell c}$$

where i indexes households, ℓ indexes locations, and c indexes birth cohorts. The indicator Post_c equals one if cohort c is born after the RBM campaign was introduced and zero otherwise. The variable $\text{malaria}_{2000\ell}$ denotes malaria prevalence in location ℓ in the pre-RBM period, which is our baseline balanced growth path. Following Kuecken et al. (2021), I include location fixed effects δ_ℓ to control for baseline regional differences in malaria prevalence. The outcome $y_{i\ell c}$ denotes the three endogenous variables that households adjust in response to the changed disease environment: Child mortality (the number of children who died in household i), fertility (the number of children ever born to household i), or children's educational attainment (years of schooling completed by the children of household i).

I then compare coefficients from the above regression with simulated data to their data counterparts. In Kuecken et al. (2021), fertility and mortality responses are estimated as changes in the woman-year probability of birth and infant death. In the model, fertility and mortality are defined at the woman-lifetime level, as the total number of births and child deaths over the life cycle. I therefore convert the reduced-form estimates into woman-lifetime units. Appendix C provides details on this mapping. In these units, the point estimates imply 0.14 fewer births and 0.07 fewer infant deaths per woman over the life cycle. Schooling effects are directly comparable and correspond to 0.43 additional years of schooling for exposed children. Table 4 reports the resulting effects for both the data and the model. All coefficients are evaluated at the average pre-intervention malaria risk for ease of interpretation.

Evaluated at the average pre-RBM malaria risk, the model-implied effects of the RBM are of a reasonable magnitude relative to the empirical confidence intervals. The reduction in child mortality in the model is exactly aligned with the reduction in infant mortality observed during the RBM campaign, reflecting that the empirically observed declines in malaria-attributed mortality and morbidity during the RBM period are fed into the model. The model also reproduces the correct direction of the fertility and schooling responses, although it slightly overshoots the fertility decline and undershoots the increase in years of schooling. However, note that none of these estimates are directly targeted in the calibration. Overall, this validation exercise supports the view that the model's quantity-quality mechanism generates empirically plausible behavioral responses

Table 4: Validation: Effects of the RBM Campaign in the Data and the Model

Moments	**Data**	**Model**
RBM Effect on Child Mortality	-0.07 $(-0.13, -0.01)$	-0.07
RBM Effect on Gross Fertility	-0.14 $(-0.24, -0.07)$	-0.21
RBM Effect on Schooling (Years)	0.43 $(0.35, 0.51)$	0.31

Notes: The Data column reports the RBM reduced-form effects from Kuecken et al. (2021). Child mortality and gross fertility estimates are converted to women-lifetime units to make them comparable to the model moments. The Model column reports the corresponding coefficients estimated on simulated data using the regression specification above. Effects are evaluated at the average pre-RBM malaria risk (PfPR = 0.327). Parentheses contain 95% confidence intervals.

to malaria reduction.

4. Macroeconomic Consequences of Eradicating Malaria

Using the model, this section reports the results of two computational exercises that quantify the impact of malaria eradication on per capita income, educational attainment, and fertility. Malaria eradication is modeled by implementing a nationwide vaccination campaign. Vaccination has long been considered the ultimate tool to eradicate malaria, though progress toward an effective vaccine has been historically slow.[17] However, two recently developed malaria vaccine are known to provide up to 75% effective in preventing the disease among children under five.[18] Motivated by these factors, I adopt vaccination as the policy instrument to achieve malaria eradication in the model. Through the lens of the model, vaccination is represented as a 75% reduction in malaria mortality ($\mu \times \chi_\ell^d$) and morbidity shock probability (χ_ℓ^m) for all locations ℓ.

I report the results from two computational exercises. The first exercise examines the short-run effects of malaria eradication, focusing on the immediate, one-generational impact. This captures the direct benefits experienced by children who receive the vaccination. I keep the wages for each skill group unchanged in this exercise, to better isolate the mechanism. The second exercise considers the long-run effects by computing

[17] Large-scale spraying of DDT (dichloro-diphenyl-trichloroethane) and other pesticides was a primary method to eradicate malaria in the past. However, it is no longer recommended due to concerns about its harmful effects on the environment and human health.

[18] Demand for an effective vaccine is also high: in a nationally representative 2011 survey conducted in Tanzania, 95% of respondents expressed willingness to vaccinate their children against malaria, if available (Romore, Ali, Semali, Mshinda, Tanner, and Abdulla, 2015).

the new balanced growth path following vaccination. This analysis reflects intergenerational effects, where healthier parents respond by reducing fertility and increasing educational investments. In this framework, wages for each skill group are also allowed to adjust endogenously. For the long-run analysis, I also report outcomes for complete malaria eradication, assuming a hypothetical 100% vaccine efficacy. While these long-term gains may take time to materialize, the results shed light on the quantitative importance of childhood health in explaining cross-country income differences.

4.1. Short-Run Impact of Malaria Vaccine

The first exercise quantifies the short-run, one-generational impact of the malaria vaccine. Conceptually, this exercise is similar to simulating the RBM campaign within the model. The key difference is that while the RBM simulation uses the observed reduction in malaria risk from the data, the short-run vaccine simulation assumes a uniform 75% reduction in malaria risk. The RBM simulation is primarily used to discipline the behavioral responses in the model—Table 4 reports the corresponding coefficients from the *individual*-level regressions. In contrast, the short-run vaccine simulation provides a benchmark for the aggregate, economy-wide gains from malaria eradication in the short term by aggregating individual-level outcomes.

The first column of Table 5 presents the impact of malaria vaccination on aggregate educational attainment and per capita income. The first column reports the changes in the corresponding moments calculated from the cohorts born after the eradication. For per capita income, I measure income at age 18, the point at which individuals in the model enter the labor market. The results indicate that malaria eradication through vaccine distribution would increase the per capita income of vaccinated children by an average of 6.93%. This increase is driven by improved educational attainment during childhood, as parents respond to the enhanced health environment by recognizing the higher returns to education. The primary school completion rate rises by 1.5 percentage point. Despite the higher opportunity cost of secondary schooling—since children now acquire more human capital during primary school and thus can earn more if they enter the labor market instead—the secondary school completion rate still increases by 2.7 percentage points.

While the observed increases in primary and secondary school completion rates highlight the role of additional years of schooling in raising income, they also reflect other channels through which malaria eradication enhances children's human capital, such as improved learning *within* schools resulting from higher cognitive ability. The model

Table 5: Short-Run Effects of Malaria Vaccination

	Baseline	**No Improvement in Morbidity**	**Exogenous Fertility**
Primary completion rate (%)	+ 1.45	+ 1.45	+ 0.52
Secondary completion rate (%)	+ 2.74	+ 2.74	− 0.55
Δ Per capita income	+ 6.93%	+ 2.09%	+ 3.09%

Notes: This table shows the short-run, one-generational effects of the malaria vaccine on children's educational attainment and earnings in the first period of adulthood. The numbers in the second column are calculated from a simulation where I do not allow parents to make endogenous fertility choices and assign the number of children corresponding to the pre-vaccine balanced growth path. Parents still make the education investment choice in this case.

explicitly captures these broader effects: the malaria vaccine improves human capital not only by increasing school enrollment but also by enhancing learning within schools through eliminating morbidity shocks. The latter channel, which has not been considered in previous studies, turns out to be quantitatively important. For instance, Ashraf et al. (2008) assumes that malaria eradication raises human capital solely via increased years of schooling, ignoring within-school improvements. Consequently, their estimated long-run per capita income gain from malaria eradication is modest, around 2%. In contrast, even the short-run per capita income gain predicted by this model (6.93%) is more than three times as large.

To assess the quantitative implications of improved learning within schools, I calculate the short-run average gain in per capita income under a counterfactual scenario where educational attainment increases, but the amount of human capital acquired in school remains unchanged. In this scenario, children continue to suffer from the adverse effects of malaria on human capital, $\underline{m}$, yet attain higher levels of education anyway. The results are presented in the second column of Table 5. The model predicts that without improved learning in school, the predicted increase in earnings would be only 2.09%. This suggests that the increase in years of schooling alone accounts for just 30% of the total gain in human capital, underscoring the importance of enhanced learning outcomes as a key driver of higher per-capita income.

The finding that improved learning within schools is essential for generating income gains following malaria eradication also aligns with empirical evidence from historical malaria eradication episodes. For example, Bleakley (2010) examines malaria eradication campaigns in six Latin American countries around 1955 and finds that while the

campaign increased adult earnings of affected cohorts by over 20%, higher educational attainment accounted for only 25% of this increase. This further emphasizes that the gains in human capital following malaria eradication operate through channels beyond schooling alone, highlighting the importance of considering the *quality* of human capital improvement.

Another channel through which malaria eradication raises per capita income is the quantity-quality tradeoff. When malaria risk declines, households in the model respond by reducing fertility. This reduction in fertility, in turn, allows parents to allocate more resources toward educational investments for their fewer children. To quantify this mechanism, I generate the same moments under a counterfactual scenario in which households are not allowed to adjust their fertility. The results, reported in the last column of Table 5, underscore the importance of the quantity-quality tradeoff. In this scenario, the per capita income gain and the increase in primary school enrollment are both reduced by more than half, while the change in secondary school enrollment even becomes negative. These findings highlight the central role of fertility adjustments in amplifying the long-term gains from malaria eradication.

4.2. Long-Run Impact of Malaria Eradication

The second exercise computes the long-run macroeconomic impacts of malaria eradication on the economy's new balanced growth path. It is hard to believe that malaria eradication affects only one generation, without influencing subsequent generations' educational decisions. If children benefited from eradication further increase educational investment in their offspring, the long-run increase in per-capita output could be even larger. The overlapping-generations structure and intergenerational linkage embedded in the model explicitly capture this mechanism. The long-run computations also account for endogenous adjustments in both skilled and unskilled wages in response to the changing skill composition of the workforce. These long-run results may be less immediately relevant for policymakers evaluating vaccine distribution, given the time required for such effects to materialize. Nevertheless, the long-run exercises are useful in providing informative benchmarks for assessing the aggregate economic loss attributable to the burden of malaria.

Table 6 displays the baseline results for the long-run, general equilibrium effects of malaria eradication. The second column is the long-run steady state of the economy where both malaria mortality ($\mu \times \chi_\ell^d$) and morbidity probabilities (χ_ℓ^m) for all locations ℓ are lowered by 75%, the level of efficacy of currently available malaria vaccines. The

Table 6: Long-Run General Equilibrium Impact of Malaria Vaccines

	Baseline	Vaccine w/ 75% Efficacy	Full Eradication
Education			
Primary completion rate (%)	69.9	72.3	73.8
Secondary completion rate (%)	13.1	16.6	18.3
Differential Fertility			
Total fertility rate	5.82	5.35	5.17
Total fertility rate, unskilled parents	6.12	5.68	5.50
Total fertility rate, skilled parents	3.04	2.87	2.81
Intergenerational Mobility and Inequality			
Primary-Secondary IGM	12.9	17.0	18.0
Gini coefficient	0.44	0.43	0.42
Change in Per Capita Income and Prices			
Δ Per Capita Income		$+6.7\%$	$+9.5\%$
Δ Unskilled wage		$+0.4\%$	$+1.1\%$
Δ Skilled wage		-1.3%	-3.6%

Notes: This table reports the long-run, general equilibrium effects of nationwide malaria vaccination. The first column is the baseline calibrated economy. The second column is the economy where both malaria-attributed mortality ($\mu \times \chi_\ell^d$) and morbidity probabilities (χ_ℓ^m) are lowered by 75%, the level of efficacy of currently available malaria vaccines. The third column corresponds to the hypothetical vaccine with 100% efficacy, fully eradicating malaria.

third column corresponds to the steady state economy with a hypothetical vaccine with 100% efficacy, hence entirely eradicating malaria. In addition to the educational attainment and per capita income, Table 6 also reports outcomes related to fertility, intergenerational mobility, and inequality. It also presents how unskilled and skilled wages (w_U and w_S) respond to the changing skill composition of the workforce.

Focusing on the scenario of 75% efficacy vaccine, the long-run results on education and per capita income are *qualitatively* similar to the short-run outcomes, but the magnitudes are nearly twice as large. This larger long-run gain is driven by intergenerational dynamics: healthier children not only achieve higher income themselves, but also invest more in their own children's education while choosing to have smaller families. This amplification mechanism is similar to that of Daruich (2026), who show that early-childhood investments by the government improve parental background for the next generation, thereby reinforcing human capital accumulation over time.

In both the short and long run, improved educational attainment is the primary channel through which per capita income rises. Fertility declines across both skill groups, indicating that in the new steady state, households are having fewer children and allocating more resources to each child's education—an expression of the quantity-quality tradeoff also observed in the short-run results. As a result, population growth in the long-run steady state is 0.2 percentage points lower. Lastly, the relative wage of unskilled workers rises by 0.4 percent while the skilled wage falls by 1.3 percent, reflecting the larger supply of skilled workers in the post-vaccine steady state.[19]

The model also predicts improvements in intergenerational mobility and a reduction in inequality. Specifically, the probability that children of parents who completed primary school attend secondary school increases by 4.1 percentage points. When a child contracts malaria, unskilled parents are less likely than skilled parents to invest in the child's education. That is, more educated parents are better able to compensate for the adverse effects of malaria on their children. Vaccination narrows this gap in educational investment by reducing the incidence of malaria, leading to lower income inequality.

Turning to the complete eradication scenario, the 9.5% increase in long-run per capita income predicted by the model appears notably larger than estimates reported in the existing literature. Acemoglu and Johnson (2007) examines the impact of increased life expectancy on economic growth by exploiting substantial improvements in longevity driven by international health interventions in the 1940s. They find a relative decline in GDP per capita in countries that experienced large gains in life expectancy, suggesting that improved survival rates primarily contributed to population growth rather than economic expansion. At first glance, the reduction in per capita output seems inconsistent with the model's prediction of a substantial long-run income gain. However, the model also indicates that a decrease in mortality is not sufficient to generate transformative gains in per capita income if a decrease in morbidity does not accompany it. Column 2 of Table 7 presents long-run steady-state results for a scenario where only mortality declines, while the morbidity risk from malaria remains unchanged. This corresponds to a setting where life expectancy rises without improving health quality, particularly in dimensions that affect learning outcomes.[20] Consistent with the findings of

[19]The rise in unskilled wage and fall in skilled wage is consistent with Khanna (2023), who finds a large decline in the relative wages of skilled workers following an expansion in schooling in India.

[20]Several features of the period studied by Acemoglu and Johnson (2007) suggest that the international epidemiological transition did little to enhance children's learning outcomes. In the 1940s, schooling was not universal, preventing many children from taking advantage of improved health by attending school. Moreover, child labor was more prevalent due to the absence of strong legal restrictions, further limiting the potential for educational investments.

Acemoglu and Johnson (2007), the model predicts a 0.4% *reduction* in per capita income in the long run, alongside an increase in the population growth rate.

Table 7: Decomposition of the Long-Run Effects of Complete Eradication

	Pre-Vaccine BGP	**Lower Mortality**	**Lower Morbidity**	**Lower Both**
Population Growth Rate (%)	4.01	4.06	3.54	3.61
Primary Completion Rate (%)	69.9	67.3	77.1	73.8
Secondary Completion Rate (%)	13.1	13.6	18.2	18.3
Δ Per capita earnings		$-$ 0.4%	$+$ 10.9%	$+$ 9.5%

Notes: This table shows the long-run changes in educational attainment and per-capita output when mortality and/or morbidity are lowered. The second column contains the results from a simulation with a 100% reduction in malaria-attributed mortality ($\mu \times \chi_\ell^d$) while the morbidity shock probability (χ_ℓ^m) is unchanged. The third column contains the results from a simulation with no change in malaria mortality while the morbidity shock is removed. The last column is the baseline long-run simulation, where both mortality and morbidity probabilities are lowered.

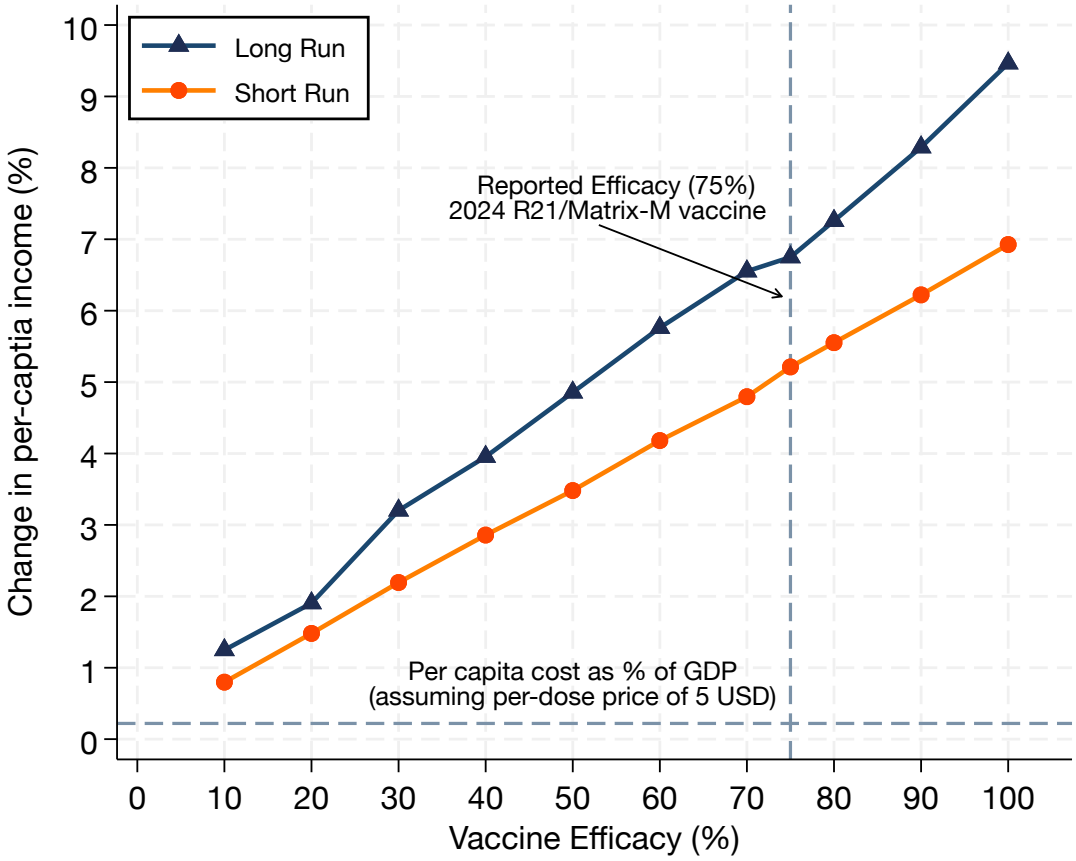
The reason that quantitatively similar reductions in mortality yield markedly different outcomes in per capita income lies in the endogenous responses of fertility and human capital investment. When a decline in mortality is not accompanied by a reduction in morbidity, the quantity–quality tradeoff predicts that parents will respond by having more children, as the risk of child loss has diminished. This, in turn, lowers per-child educational investment, resulting in reduced educational attainment and, consequently, lower per capita income. In contrast, when morbidity declines without a corresponding drop in mortality, per capita income rises even more, as parents reduce fertility further and allocate greater resources to each child's education. This mechanism is illustrated in the third column of Table 7. The calibrated model suggests that, in the case of malaria, the effect of reduced morbidity dominates. These findings underscore that a unidimensional, mortality-focused approach to modeling disease may lead to biased conclusions.

4.3. Cost-Benefit Analysis of Malaria Vaccines

Although the long-run increase in output per capita is substantial, producing and administering vaccines at scale can be costly. For instance, Sicuri, Yaya Bocoum, Nonvignon, Alonso, Fakih, Bonsu, Kariuki, Leeuwenkamp, Munguambe, Mrisho et al. (2019) estimates that, depending on the per-dose price, the total cost of administering a malaria vaccine ranges from 24 to 48 USD per child in Tanzania, including all associated ex-

penses.[21] Furthermore, the actual efficacy of the vaccine may fall short of the current 75% estimate. If efficacy is lower, the vaccination costs could potentially exceed the resulting benefits.

Figure 4: Vaccine Efficacy and the Cost of Vaccination



To assess whether the gains in per capita income are sufficient to justify the costs, I solve for the post-vaccination balanced growth path across a range of efficacy levels and compare the resulting income increases per year to the per-year cost of vaccination. The annual spending as a share of GDP under a steady rollout can be expressed as:

$$\text{Cost share of GDP} = \frac{(\text{doses}/_{\text{child}}) \times (\text{price}/_{\text{dose}}) \times (\%\ \text{population vaccinated}/_{\text{year}})}{\text{GDP}}$$

Following Sicuri et al. (2019), I assume a per-dose cost of 5 USD, implying a total vaccination cost of 24.9 USD per child in 2015 dollars. Given Tanzania's GDP per capita

[21] According to Datoo, Natama, Somé, Bellamy, Traoré, Rouamba, Tahita, Ido, Yameogo, Valia et al. (2022), complete vaccination requires four doses—three initial doses and a booster one year later.

in 2001 was 573 USD[22] and the crude birth rate of 41 in 2001, this amounts to approximately 0.18% of the annual GDP. Figure 4 presents the cost-benefit comparison across efficacy scenarios. The vertical line at 75% marks the reported efficacy of the R21/Matrix-M vaccine, while the horizontal line indicates the vaccination cost as a share of GDP per capita.

The figure illustrates that, at the currently reported efficacy of the R21/Matrix-M vaccine, both the short- and long-run increases in per capita income vastly outweigh the cost of vaccination. Given that the malaria vaccine rollout continues to be delayed by funding challenges, these results strongly support prioritizing investments to accelerate vaccine deployment. Duncombe et al. (2024) estimate that, at the current funding pace, universal vaccination coverage would only be achieved by 2035, resulting in approximately 2.5 million preventable child deaths. The cost-benefit analysis implies that substantial macroeconomic benefits may also be forgone due to delayed vaccine distribution.

The model also provides insights on which geographic areas should be prioritized. From a cost-effectiveness perspective, the model suggests that malaria vaccination should be recommended for children living in locations with moderate to high malaria prevalence. Figure 5 illustrates the heterogeneous effects of vaccination across regions with varying baseline malaria prevalence. While there is substantial variation in the per capita income gains, vaccination is cost-effective in the short run in all locations. In the highest-prevalence locations, the long-run income gains reach up to 28%, implying substantial economic benefits of geographically targeted vaccination efforts.

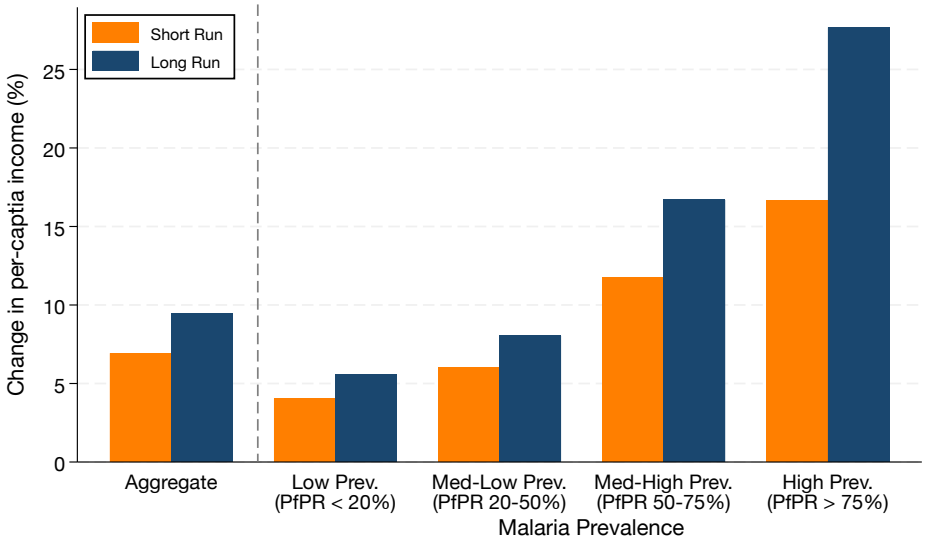
5. Conclusion

High mortality and poor health conditions have long been viewed as critical obstacles to economic development in poorer countries. Malaria, in particular, remains a major burden in sub-Saharan Africa, responsible for over 600,000 deaths annually. Beyond mortality, malaria infection leaves lasting cognitive impairments on surviving children, weakening educational outcomes and lowering labor productivity in adulthood.

This paper develops an integrated framework that combines a structural general equilibrium model with empirical evidence from a recent malaria-control intervention to credibly quantify the macroeconomic effects of malaria eradication. The model incorporates several relevant features, such as endogenous fertility, childhood human capital

[22] All figures are in 2015 constant dollars.

Figure 5: Post-Vaccine Change in Per Capita Income Across locations



accumulation through schooling, and malaria represented as a negative health shock. The calibrated model also replicates the reduced-form estimates obtained from Tanzania's Roll Back Malaria campaign.

The results indicate that the gains from eradicating malaria would be much larger than previously estimated in the macroeconomic literature. The model predicts a 9.5% increase in per-capita income from eradicating malaria. These gains primarily arise from improved learning capacity per year of schooling and adjustments in fertility decisions driven by the quantity-quality tradeoff. Evaluating the costs and benefits, the model suggests that a nationwide malaria vaccination policy would be highly cost-effective, supporting the roll-out of the recently developed vaccines with 75% efficacy.

I conclude by recognizing some limitations of this study and suggesting future avenues for research. First, health improvements may stimulate economic growth through additional channels not explored in this paper. As highlighted by Banerjee and Duflo (2005), increased savings and capital accumulation could be another important mechanism. Analyzing this channel would require more detailed data on household assets over a longer time horizon. Second, while the framework presented here can be extended to other diseases, this paper specifically focused on malaria. Studying a single disease has an advantage over examining health in general, because it is easier to obtain well-identified causal estimates. However, there are many other diseases prevalent in low-income countries, whose elimination could potentially improve living standards significantly. A challenge in studying multiple diseases simultaneously arises because

diseases often interact, and these interactions must be carefully accounted for. Future research could explore this avenue.

References

AARONSON, D., F. LANGE, AND B. MAZUMDER (2014): "Fertility Transitions Along the Extensive and Intensive Margins," *American Economic Review*, 104, 3701–24.

ACEMOGLU, D. AND S. JOHNSON (2007): "Disease and Development: the Effect of Life Expectancy on Economic Growth," *Journal of Political Economy*, 115, 925–985.

AKCIGIT, U., H. ALP, AND M. PETERS (2021): "Lack of Selection and Limits to Delegation: Firm Dynamics in Developing Countries," *American Economic Review*, 111, 231–275.

AL SEROURI, A. W., S. M. GRANTHAM-MCGREGOR, B. GREENWOOD, AND A. COSTELLO (2000): "Impact of Asymptomatic Malaria Parasitaemia on Cognitive Function and School Achievement of Schoolchildren in the Yemen Republic," *Parasitology*, 121, 337–345.

ALEMÁN, C., D. IORIO, AND R. SANTAEULÀLIA-LLOPIS (2024): "A Quantitative Theory of the HIV Epidemic: Education, Risky Sex and Asymmetric Learning," Unpublished Manuscript, NYU Abu Dhabi.

ALESINA, A., S. HOHMANN, S. MICHALOPOULOS, AND E. PAPAIOANNOU (2021): "Intergenerational Mobility in Africa," *Econometrica*, 89, 1–35.

ASHRAF, Q. H., A. LESTER, AND D. N. WEIL (2008): "When Does Improving Health Raise GDP?" *NBER Macroeconomics Annual*, 23, 157–204.

BANERJEE, A. V. AND E. DUFLO (2005): "Growth Theory Through the Lens of Development Economics," *Handbook of Economic Growth*, 1, 473–552.

BAROFSKY, J., T. D. ANEKWE, AND C. CHASE (2015): "Malaria Eradication and Economic Outcomes in Sub-Saharan Africa: Evidence from Uganda," *Journal of Health Economics*, 44, 118–136.

BARRO, R. J. AND G. S. BECKER (1989): "Fertility Choice in a Model of Economic Growth," *Econometrica*, 481–501.

BAU, N., M. ROTEMBERG, M. SHAH, AND B. STEINBERG (2024): "Human Capital Investment in the Presence of Child Labor," NBER Working Paper No. 27241.

BILS, M., B. KAYMAK, AND K.-J. WU (2024): "Labor Substitutability among Schooling Groups," *American Economic Journal: Macroeconomics*, 16, 1–34.

BLEAKLEY, H. (2010): "Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure," *American Economic Journal: Applied Economics*, 2, 1–45.

BLOOM, D. E., D. CANNING, R. KOTSCHY, K. PRETTNER, AND J. SCHÜNEMANN (2024): "Health and Economic Growth: Reconciling the Micro and Macro Evidence," *World Development*, 178, 106575.

BROOKS, W. AND K. DONOVAN (2020): "Eliminating Uncertainty in Market Access: The Impact of New Bridges in Rural Nicaragua," *Econometrica*, 88, 1965–1997.

BUERA, F. J., J. P. KABOSKI, AND Y. SHIN (2021): "The Macroeconomics of Microfinance," *The Review of Economic Studies*, 88, 126–161.

BUERA, F. J., J. P. KABOSKI, AND R. M. TOWNSEND (2023): "From Micro to Macro Development," *Journal of Economic Literature*, 61, 471–503.

CASELLI, F. (2005): "Accounting for Cross-Country Income Differences," *Handbook of Economic Growth*, 1, 679–741.

CHEN, I., S. E. CLARKE, R. GOSLING, B. HAMAINZA, G. KILLEEN, A. MAGILL, W. O'MEARA, R. N. PRICE, AND E. M. RILEY (2016): ""Asymptomatic" Malaria: a Chronic and Debilitating Infection that Should be Treated," *PLoS Medicine*, 13, e1001942.

CUNHA, F. AND J. HECKMAN (2007): "The Technology of Skill Formation," *American Economic Review*, 97, 31–47.

DARUICH, D. (2026): "The Macroeconomic Consequences of Early Childhood Development Policies," *Journal of Political Economy*.

DARUICH, D. AND J. KOZLOWSKI (2020): "Explaining Intergenerational Mobility: The Role of Fertility and Family Transfers," *Review of Economic Dynamics*, 36, 220–245.

DATOO, M. S., H. M. NATAMA, A. SOMÉ, D. BELLAMY, O. TRAORÉ, T. ROUAMBA, M. C. TAHITA, N. F. A. IDO, P. YAMEOGO, D. VALIA, ET AL. (2022): "Efficacy and Immunogenicity of R21/Matrix-M Vaccine Against Clinical Malaria After 2 years' Follow-up in Children in Burkina Faso: a Phase 1/2b Randomised Controlled Trial," *The Lancet Infectious Diseases*.

DOEPKE, M. (2005): "Child Mortality and Fertility Decline: Does the Barro-Becker Model Fit the Facts?" *Journal of Population Economics*, 18, 337–366.

DONOVAN, K. (2021): "The Equilibrium Impact of Agricultural Risk on Intermediate Inputs and Aggregate Productivity," *The Review of Economic Studies*, 88, 2275–2307.

DUNCOMBE, R., K. ELABD, AND J. SANDEFUR (2024): "Avoiding Another Lost Decade on Malaria Vaccines," Center for Global Development Policy Paper.

FEENSTRA, R. C., R. INKLAAR, AND M. P. TIMMER (2015): "The Next Generation of the Penn World Table," *American Economic Review*, 105, 3150–3182.

FERNANDO, S., D. GUNAWARDENE, M. BANDARA, D. DE SILVA, R. CARTER, K. MENDIS, AND A. WICKREMASINGHE (2003): "The Impact of Repeated Malaria Attacks on the School Performance of Children," *American Journal of Tropical Medicine and Hygiene*.

FERNANDO, S. D., C. RODRIGO, AND S. RAJAPAKSE (2010): "The 'Hidden' Burden of Malaria: Cognitive Impairment Following Infection," *Malaria Journal*, 9, 1–11.

FRIED, S. AND D. LAGAKOS (2022): "Electricity and Firm Productivity: A General-Equilibrium Approach," *American Economic Journal: Macroeconomics*.

FUJIMOTO, J., D. LAGAKOS, AND M. VANVUREN (2025): "Aggregate and Distributional Effects of 'Free' Secondary Schooling in the Developing World," *American Economic Journal: Macroeconomics*, Forthcoming.

GOLLIN, D. AND C. ZIMMERMANN (2007): "Malaria: Disease Impacts and Long-Run Income Differences," Unpublished Manuscript, Tufts University.

GREENWOOD, J., P. KIRCHER, C. SANTOS, AND M. TERTILT (2019): "An Equilibrium Model of the African HIV/AIDS epidemic," *Econometrica*, 87, 1081–1113.

HANUSHEK, E. A. AND L. WOESSMANN (2008): "The Role of Cognitive Skills in Economic Development," *Journal of Economic Literature*, 46, 607–68.

HAY, S. I. AND R. W. SNOW (2006): "The Malaria Atlas Project: Developing Global Maps of Malaria Risk," *PLOS Medicine*, 3, e473.

INSTITUTE FOR HEALTH METRICS AND EVALUATION (IHME) (2026): "GBD Results Tool," Interactive data visualization and download tool, accessed 2026-02-04.

JOHANSSON, E. W., R. E. CIBULSKIS, R. W. STEKETEE, ET AL. (2010): *Malaria Funding and Resource Utilization: the First decade of Roll Back Malaria*, World Health Organization.

JONES, L. E. AND A. SCHOONBROODT (2010): "Complements versus Substitutes and Trends in Fertility Choice in Dynastic Models," *International Economic Review*, 51, 671–699.

JOSEPH, C. (2020): "Education and Labour Market Earnings in Low Income Countries: Empirical Evidence for Tanzania," *Tanzania Journal for Population studies and Development*, 26.

KALEMLI-OZCAN, S. (2003): "A Stochastic Model of Mortality, Fertility, and Human Capital Investment," *Journal of Development Economics*, 70, 103–118.

KALEMLI-OZCAN, S., H. E. RYDER, AND D. N. WEIL (2000): "Mortality Decline, Human Capital Investment, and Economic Growth," *Journal of Development Economics*, 62, 1–23.

KHANNA, G. (2023): "Large-Scale Education Reform in General Equilibrium: Regression Discontinuity Evidence from India," *Journal of Political Economy*, 131, 549–591.

KUECKEN, M., J. THUILLIEZ, M.-A. VALFORT, ET AL. (2021): "Disease and Human Capital Accumulation: Evidence from the Roll Back Malaria Partnership in Africa," *The Economic Journal*.

LAGAKOS, D., A. M. MOBARAK, AND M. E. WAUGH (2023): "The Welfare Effects of Encouraging Rural–Urban Migration," *Econometrica*, 91, 803–837.

LAGAKOS, D. AND T. SCHOELLMAN (2026): "Accounting for Cross-Country Income Differences Revisited," Unpublished Manuscript, Boston University.

LEE, S. Y. AND A. SESHADRI (2019): "On the Intergenerational Transmission of Economic Status," *Journal of Political Economy*, 127, 855–921.

LEYARO, V., P. TWUMASI BAFFOUR, O. MORRISSEY, AND T. OWENS (2014): "Determinants of Urban Labour Earnings in Tanzania, 2000/01-06," Unpublished Manuscript, The University of Nottingham.

MALARIA ATLAS PROJECT (2026): "Malaria Atlas Project Data Explorer," Online database, accessed 2026-02-04.

MANUELLI, R. E. AND A. SESHADRI (2009): "Explaining International Fertility Differences," *The Quarterly Journal of Economics*, 124, 771–807.

——— (2014): "Human Capital and the Wealth of Nations," *American Economic Review*, 104, 2736–62.

MATANGILA, J. R., J. FRAEYMAN, M.-L. M. KAMBULU, A. MPANYA, R. I. DA LUZ, P. LUTUMBA, J.-P. VAN GEERTRUYDEN, AND H. BASTIAENS (2017): "The Perception of Parents and Teachers about Intermittent Preventive Treatment for Malaria in School Children in a Semi-Rural area of Kinshasa, in the Democratic Republic of Congo," *Malaria Journal*, 16, 1–7.

MINNESOTA POPULATION CENTER (2020): *Integrated Public Use Microdata Series, International: Version 7.3 [Machine-readable database]*, Minneapolis: University of Minnesota.

MLACHA, C. J. AND M. O. NDANSHAU (2018): "Education and Labour Earnings Inequality in Tanzania: Evidence from Quantile Regression Analysis," Unpublished Manuscript, University of Dar es Salaam.

MONTGOMERY, C. M., W. MWENGEE, M. KONG'ONG'O, AND R. POOL (2006): "'To help Them is to Educate Them': Power and Pedagogy in the Prevention and Treatment of Malaria in Tanzania," *Tropical Medicine & International Health*, 11, 1661–1669.

OGBO, F. A., K. E. OSITA, O. A. AKOREDE, K. I. IFEGWU, T. LAWRENCE, J. EMMANUEL, C. DEBORAH, AND E. A. KINGSLEY (2019): "Determinants of trends in neonatal, postneonatal, infant, child and under-five mortalities in Tanzania from 2004 to 2016," *BMC Public Health*.

ROMORE, I., A. M. ALI, I. SEMALI, H. MSHINDA, M. TANNER, AND S. ABDULLA (2015): "Assessment of Parental Perception of Malaria Vaccine in Tanzania," *Malaria journal*, 14, 1–6.

ROYS, N. AND A. SESHADRI (2017): "On the Origin and Causes of Economic Growth," Unpublished Manuscript, University of Wisconsin-Madison.

SCHOELLMAN, T. (2012): "Education Quality and Development Accounting," *The Review of Economic Studies*, 79, 388–417.

——— (2016): "Early Childhood Human Capital and Development," *American Economic Journal: Macroeconomics*, 8, 145–74.

SHASTRY, G. K. AND D. N. WEIL (2003): "How Much of Cross-Country Income Variation is Explained by Health?" *Journal of the European Economic Association*, 1, 387–396.

SICURI, E., F. YAYA BOCOUM, J. NONVIGNON, S. ALONSO, B. FAKIH, G. BONSU, S. KARIUKI, O. LEEUWENKAMP, K. MUNGUAMBE, M. MRISHO, ET AL. (2019): "The costs of implementing vaccination with the RTS, S malaria vaccine in five sub-Saharan African countries," *MDM Policy & Practice*, 4, 2381468319896280.

SOARES, R. R. (2005): "Mortality Reductions, Educational Attainment, and Fertility Choice," *American Economic Review*, 95, 580–601.

TARIMO, D. S., G. K. LWIHULA, J. N. MINJAS, AND I. C. BYGBJERG (2000): "Mothers' Perceptions and Knowledge on Childhood Malaria in the Holendemic Kibaha District, Tanzania: Implications for Malaria Control and the IMCI Strategy," *Tropical Medicine & International Health*, 5, 179–184.

TODD, P. E. AND K. I. WOLPIN (2023): "The Best of Both Worlds: Combining Randomized Controlled Trials with Structural Modeling," *Journal of Economic Literature*, 61, 41–85.

VENKATARAMANI, A. S. (2012): "Early Life Exposure to Malaria and Cognition in Adulthood: Evidence From Mexico," *Journal of Health Economics*, 31, 767–780.

WEIL, D. N. (2007): "Accounting for the Effect of Health on Economic Growth," *The Quarterly Journal of Economics*, 122, 1265–1306.

WORLD BANK (2026): "World Development Indicators," World Bank DataBank, accessed 2026-02-04.

WORLD HEALTH ORGANIZATION (2016): "World Malaria Report 2015," *www.who.int/teams/global-malaria-programme/reports/world-malaria-report-2015*.

——— (2021): "World Malaria Report 2020: 20 Years of Global Progress and Challenges," *www.who.int/teams/global-malaria-programme/reports/world-malaria-report-2020*.

——— (2025): "World Malaria Report 2025," *www.who.int/teams/global-malaria-programme/reports/world-malaria-report-2025*.

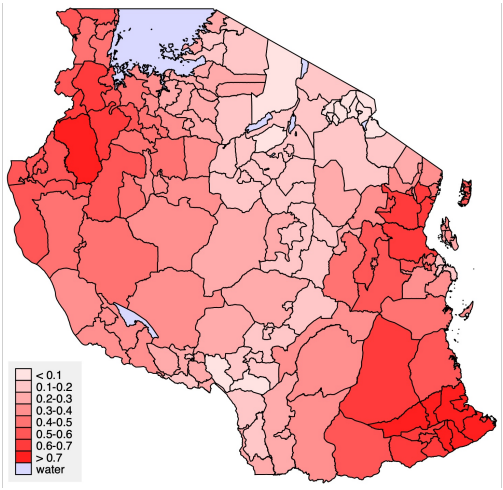
YOUNGER, S. D., F. MYAMBA, AND K. MDADILA (2016): "Fiscal Incidence in Tanzania," *African Development Review*, 28, 264–276.

ZHOU, A. (2025): "Building Future Generations: The Macroeconomic Consequences of Family Policies," Unpublished Manuscript, The University of Hong Kong.

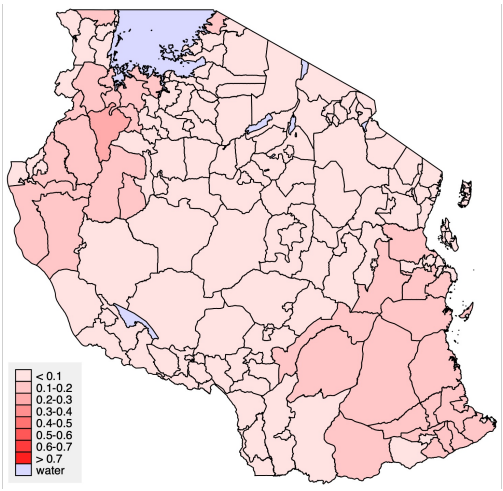
Appendix

A. Additional Tables and Figures

Figure A.1: Spatial distribution of malaria prevalence in Tanzania, pre- and post-RBM



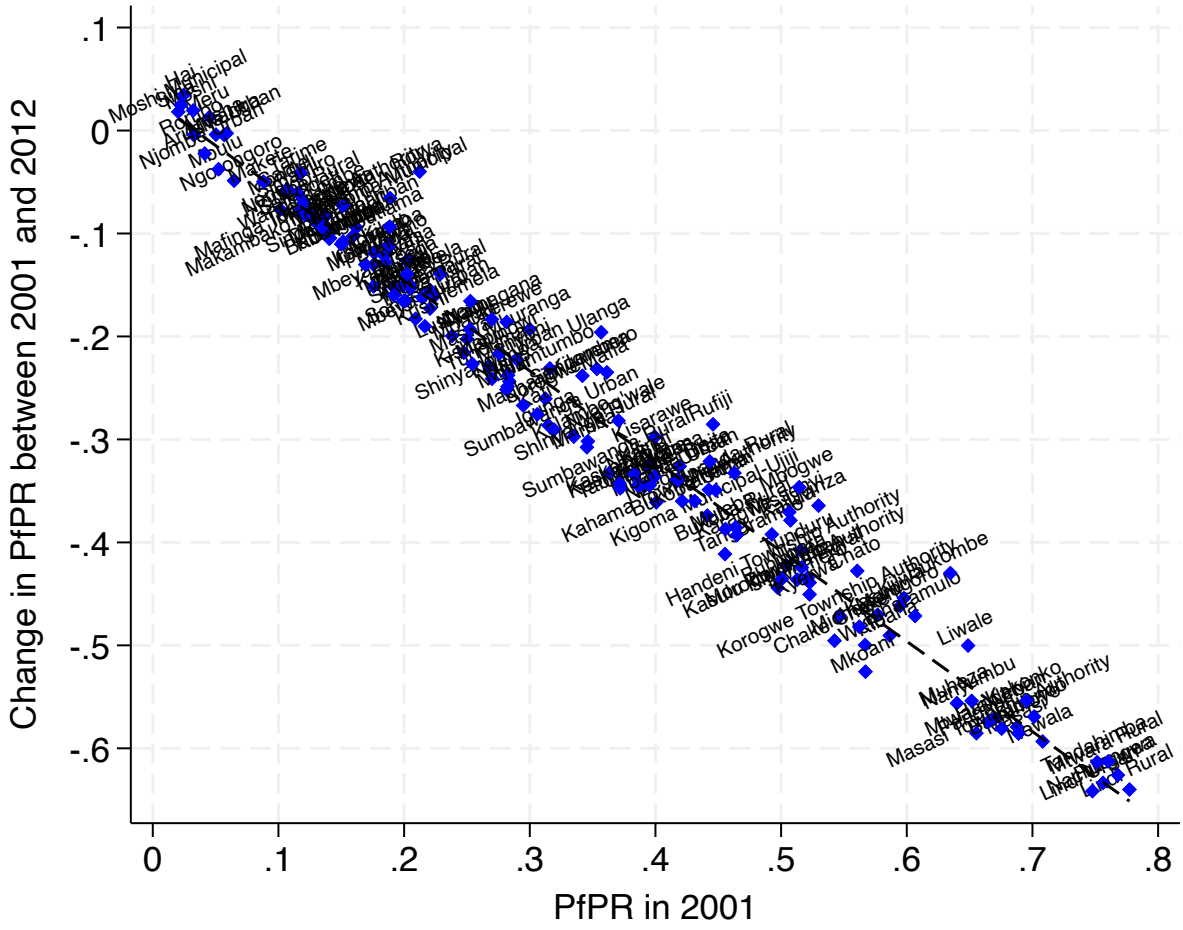
(a) PfPR in 2001



(b) PfPR in 2012

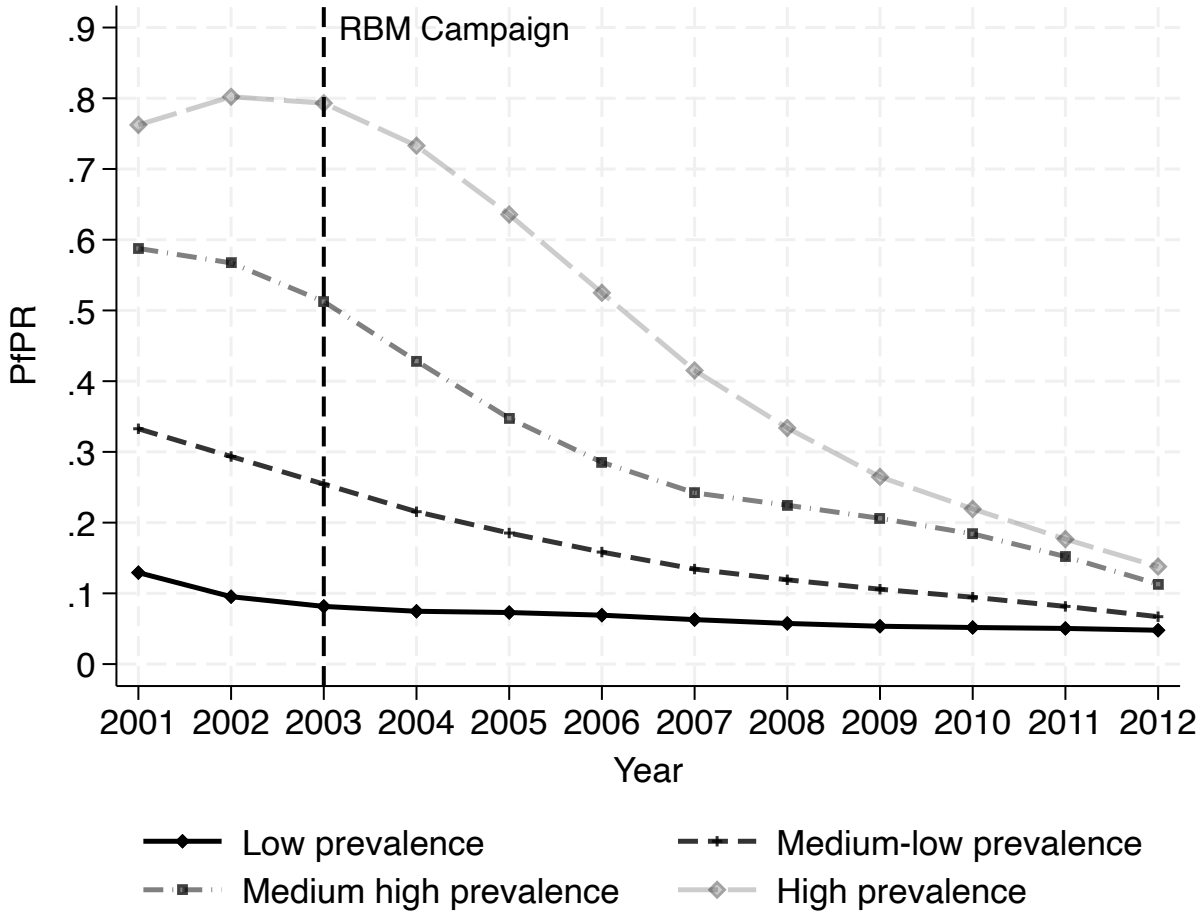
Notes: Geographic boundary is at the level of districts, which are the second-level administrative units in Tanzania. Boundaries were harmonized between 1988 and 2012 to account for political boundary changes across census years. Data downloaded from the IPUMS-International (Minnesota Population Center, 2020). PfPR data are taken from the Malaria Atlas Project (Hay and Snow, 2006; Malaria Atlas Project, 2026).

Figure A.2: Change in malaria risk for Tanzanian regions, conditional on malaria risk in 2001



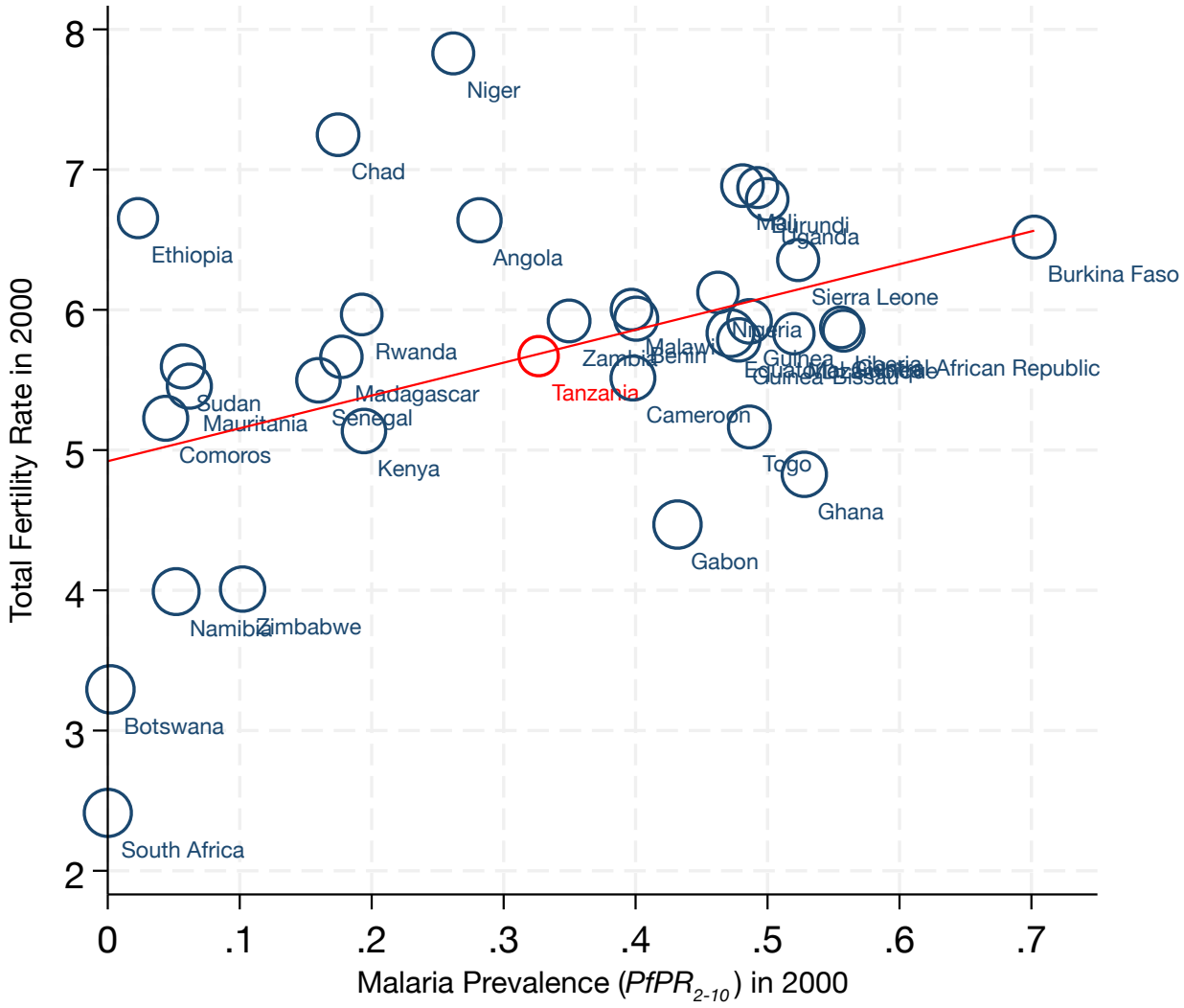
Notes: Each point represents a district at GIS-2 level. Yearly malaria risk (PfPR) is obtained from the Malaria Atlas Project (Malaria Atlas Project, 2026). The fitted line is calculated by regressing the changes in malaria risk over 2001 and 2012 on the initial malaria risk in 2001.

Figure A.3: Time trend of regional malaria prevalence in Tanzania based on the four pre-campaign malaria prevalence categories



Notes: Each point represents a within-category population-weighted mean of PfPR. Regional malaria prevalence data obtained from the Malaria Atlas Project (Malaria Atlas Project, 2026).

Figure A.4: Fertility, Malaria Prevalence, and Per Capita Income Across Sub-Saharan African Countries before the RBM campaign



Notes: Cross-country PfPR data are from the Malaria Atlas Project (Malaria Atlas Project, 2026). Cross-country GDP per capita data are from the Penn World Table (Feenstra, Inklaar, and Timmer, 2015). Fertility data are from the World Development Indicators (World Bank, 2026). Bubble size indicates log GDP per capita in 2000.

Table A.1: Malaria in Tanzania Among Children under age 10

Top 5 illnesses that led to hospitalization (%)					
	Wave 1	Wave 2	Wave 3	Wave 4	Average
Malaria	-	**41.21**	**49.1**	**39.62**	**43.27**
Fever	-	21.21	15.77	21.92	19.68
Stomach	-	7.58	3.58	4.23	5.29
Diarrhea	-	5.45	6.45	1.92	4.72
Headache	-	0.91	0	0.38	0.46

Note: From Tanzania Household Panel Survey, wave 1 (2008) – wave 4 (2014). The survey questions were "What is the 1st type of illness or injury did [NAME] had that led to his/her hospitalization?"

Table A.2: Fertility, Malaria Prevalence, and GDP per capita in 2000 for SSA countries

Country	**TFR**	**PfPR$_{2-10}$**	**GDP pc**
Population-weighted SSA Average	**5.73**	**0.299**	**2,467**
Tanzania	5.67	0.327	1,268
Angola	6.64	0.282	2,550
Benin	5.94	0.400	2,434
Botswana	3.29	0.002	12,417
Burkina Faso	6.52	0.702	1,563
Burundi	6.87	0.493	813
Cameroon	5.51	0.398	3,299
Central African Republic	5.85	0.558	1,090
Chad	7.25	0.175	1,442
Comoros	5.23	0.044	3,332
Equatorial Guinea	5.83	0.472	9,861
Ethiopia	6.65	0.023	638
Gabon	4.47	0.432	12,515
Ghana	4.83	0.528	3,766
Guinea	5.92	0.486	2,918
Guinea-Bissau	5.79	0.478	1,945
Kenya	5.14	0.194	2,725
Liberia	5.88	0.556	1,042
Madagascar	5.67	0.177	1,341
Malawi	6.00	0.397	1,020
Mali	6.89	0.481	1,440
Mauritania	5.46	0.062	3,588
Mozambique	5.83	0.520	962
Namibia	3.99	0.052	6,866
Niger	7.83	0.262	1,158
Nigeria	6.12	0.462	995
Rwanda	5.97	0.192	1,011
Senegal	5.50	0.160	2,909
Sierra Leone	6.36	0.523	1,193
South Africa	2.41	0.000	11,315
Sudan	5.60	0.057	2,542
Togo	5.17	0.486	1,569
Uganda	6.79	0.500	1,392
Zambia	5.92	0.350	1,506
Zimbabwe	4.01	0.102	3,817

Note: TFR data from the UN World Development Indicator (World Bank, 2026). PfPR data taken from the Malaria Atlas Project (Malaria Atlas Project, 2026)/ GDP and population data from the Penn World Table (Feenstra et al., 2015). Per capita GDP is expressed as constant 2021 USD. Year is 2000 for all three variables.

B. Definition of Equilibrium and Balanced Growth Path

B.1. Recursive Competitive Equilibrium

To simplify notation, denote the vector of an age-j individual's state variables $(a, s, v, s_k, h_k, z_k, m, n)$ by $\mathbf{X}_j$, and the distribution of these state variables at age j by $\mu(\mathbf{X}_j)$. A recursive competitive equilibrium consists of:

(a) Household value functions $V_j(\mathbf{X})$ and policy functions $c_j(\mathbf{X}), a'_j(\mathbf{X}), n_4(\mathbf{X}), e_5(\mathbf{X}), e_6(\mathbf{X})$

(b) Wages per efficiency unit for each skill group, w_U and w_S,

such that:

(i) The value and policy functions $(V, a', c, n_4, e_5, e_6)$ solve the individual's optimization problem, given prices w_U and w_S.

(ii) The representative firm maximizes profits, implying wages:

$$w_U = A\left[(H_U + H_P)^{\frac{\lambda-1}{\lambda}} + (H_S)^{\frac{\lambda-1}{\lambda}}\right]^{\frac{1}{\lambda-1}} (H_U + H_P)^{-\frac{1}{\lambda}},$$
$$w_S = A\left[(H_U + H_P)^{\frac{\lambda-1}{\lambda}} + (H_S)^{\frac{\lambda-1}{\lambda}}\right]^{\frac{1}{\lambda-1}} H_S^{-\frac{1}{\lambda}}.$$

(iii) Wages w_U and w_S clear the labor market.

B.2. Balanced Growth Path

A balanced growth path is a particular case of the recursive competitive equilibrium that satisfies additional conditions. Let P denote the aggregate population. A balanced growth path is a recursive competitive equilibrium in which:

(A) Aggregate population grows at a constant rate: $\frac{P'}{P} = \nu$ for some constant ν.

(B) The distribution of households is stationary: $\mu'(\mathbf{X}_j) = \mu(\mathbf{X}_j)$ for all j.

(C) Decision rules in (a) are stationary and independent of P.

C. Mapping Reduced-Form Estimates from Kuecken et al. (2021) into Model-Consistent Units

This appendix explains how we convert the reduced-form estimates in Kuecken et al. (2021) into quantities that are consistent with the units used in our quantitative model.[23]

C.1. Regression specification in Kuecken et al. (2021)

Kuecken et al. (2021) estimate the impact of the Roll Back Malaria (RBM) campaign on infant mortality and fertility using the demographic and health survey (DHS) microdata. For infant mortality, the baseline regression can be written as:

$$d_{i\ell ct} = \beta_0 + \beta_1 \cdot Post_c + \beta^{IM} \cdot (Post_c \times \text{malaria}_{2000\ell}) + \mathbf{X_{i\ell ct}}'\gamma + \mu_c + \lambda_\ell + \varepsilon_{i\ell ct}, \quad (5)$$

where $d_{i\ell ct}$ is an indicator equal to one if woman i, born in DHS cluster ℓ, belonging to cohort c and surveyed in year t, had experienced an infant death (death within the first year of life). malaria_{2000j} denotes pre-campaign malaria risk, $Post_c$ measures exposure to the RBM campaign, and X_{ijct} includes controls such as wealth and mother age. μ_c and λ_ℓ are cohort fixed effects and DHS cluster fixed effects, respectively. Because $d_{i\ell ct}$ is binary, β^{IM} captures the change in the probability of infant death per birth, expressed in percentage points. Fertility is estimated using an analogous specification:

$$b_{i\ell ct} = \beta_0 + \beta_1 \cdot Post_c + \beta^{F} \cdot (Post_c \times \text{malaria}_{2000\ell}) + \mathbf{X_{i\ell ct}}'\gamma + \mu_c + \lambda_\ell + \varepsilon_{i\ell ct}, \quad (6)$$

where $b_{i\ell ct}$ is an indicator equal to one woman i, born in DHS cluster ℓ, belonging to cohort c and surveyed in year t gave birth in year t. The coefficient β^F measures the change in the annual probability of giving birth.

C.2. Converting infant death probabilities to lifetime fertility

The fertility coefficient β^F represents a change in the probability that a woman gives birth in a given year. Let T^F denote the length of the reproductive lifespan, typically assumed as 34 years (from age 15 to 49) in demographics literature. Aggregating over the reproductive years, the implied change in lifetime fertility is

$$\Delta B = \beta^F \cdot T^F.$$

[23] The original specification has country dimension. For illustration purposes, I omit it here as the model is calibrated to a single country. See Kuecken et al. (2021) for the full specification.

The converted ΔB represents the change in the expected number of births per woman over her lifetime, which is the fertility object used in the model. Given that $\beta^F = -0.011$, average PfPR in Kuecken et al. (2021) = 0.374 and using $T = 34$, we have $\Delta B = -0.011 \times 0.374 \times 34 \approx -0.14$

C.3. Converting birth-level mortality to lifetime infant deaths

The infant mortality coefficient β^{IM} measures the change in the probability that a given birth results in an infant death. Because maternal age is controlled for in equation (5), β^{IM} reflects an average effect across births, conditional on maternal age and other covariates. The expected number of infant deaths per woman is the sum of infant death probabilities times the average number of children a woman would have if she lived through her childbearing years (age 15-49), which is typically measured as the total fertility rate (TFR). Therefore, the implied change in lifetime infant deaths is

$$\Delta D = \sum_{age=15}^{49} \Delta \Pr(\text{infant death}) = \text{TFR} \cdot \beta^{IM}.$$

Given that $\beta^{IM} = -0.032$, average PfPR in Kuecken et al. (2021) = 0.374 and using TFR in 2000 of 5.9, we have $\Delta D = -0.032 \times 0.374 \times 5.9 \approx -0.07$. This quantity can be interpreted as a change in expected infant deaths per woman. In the model, ΔD is directly comparable to the reduction in the total number of child deaths per woman.