The Macroeconomic Consequences of Malaria Eradication in Sub-Saharan Africa

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Abstract

Malaria is the primary cause of child mortality and a barrier to childhood human capital accumulation in sub-Saharan Africa. This paper quantifies the macroeconomic consequences of malaria eradication using a structural model, where individuals endogenously adjust fertility and educational investment in children in response to malaria. The model is disciplined by matching empirical estimates from an anti-malaria campaign in Tanzania. The estimated per-capita income gain from eradication is substantial—nearly three times larger than previously reported—as healthier children acquire more human capital per year of schooling, beyond simply attending school longer. Given the distribution of malaria vaccine remains delayed, the results support accelerating vaccine deployment.

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

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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 not only for evaluating the returns to health investments in developing countries, but also for understanding the sources of cross-country income differences.

Previous macroeconomic studies typically quantify the economic gains from health improvement or disease eradication using an accounting framework (Shastry and Weil, 2003; Weil, 2007; Bloom, Canning, Kotschy, Prettner, and Schünemann, 2024). In this approach, health is treated as a component of individual labor productivity, with its impact on productivity derived from micro-level estimates. The aggregate effects of health are then calculated by extrapolating these individual-level estimates through an aggregate production function. While this accounting approach provides useful initial estimates, it overlooks general equilibrium responses and endogenous behavioral adjustments. For instance, changes in population size, which is essential for understanding the change in per-capita income following health improvement, depend crucially on how individuals adjust their fertility behavior in response to improved health. Ignoring these endogenous adjustments can potentially bias estimates of the economic impact of health improvements.

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. Unlike extrapolation-based methods, 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 the per-capita income by 6.8%, roughly three times larger than estimates reported in previous macroeconomic studies. The model also suggests that failing to account for individual responses ane 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. What distin-

guishes this paper from previous macroeconomic studies is that I provide a richer measure of human capital, beyond just years of schooling. 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 captures intergenerational dynamics, capturing that healthier children to subsequently adjust their fertility and invest more in the education of their own children (Daruich, 2020).

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 in the model, represented by lower mortality and morbidity. This generates two opposing effects. On 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 by reduced morbidity lowers 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 discipline the mechanism by directly matching the empirical estimates of reduced malaria risk on women's fertility and children's educational attainment. I exploit a recent large-scale antimalarial campaign in sub-Saharan Africa, the Roll Back Malaria (RBM) campaign, as a source of exogenous variation in malaria prevalence. Initiated in 2003, the RBM campaign significantly reduced malaria prevalence through the aggressive distribution of insecticide-treated bednets. I focus on Tanzania, one of the earliest recipient of the campaign, where it was highly successful. Leveraging regional variation in the treatment intensity, I employ a difference-in-differences design to estimate the effects of reduced malaria prevalence on fertility and children's educational attainment. Consistent with previous empirical studies, I find that children exposed to the campaign attended an additional 0.56 years of schooling on average, and women in childbearing age reduced their fertility by 9.4%. I then simulate the RBM campaign within the model by implementing a comparable reduction in malaria risk. The predicted fertility and schooling responses from the model closely match the empirical estimates.

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 ini-

tiated 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 general equilibrium 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 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 3.9% that 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 predicts that the changes in the *quantity* of education is not the main source of income gain. Rather, improved learning capacity alone accounts for 74% of the total income increase.[1] 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, 2007; Schoellman, 2012). The decomposition exercise also highlights the quantitative importance of 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 1.85%.

While the short-run analysis is informative for gauge the immediate impacts of eradication, the long run exercise sheds light on how significant the burden of malaria is in explaining the income differences between rich and poor countries. The model predicts that, in the long run, full malaria eradication would increase per-capita income by 6.8% as intergenerational dynamics amplify the initial gains. The per-capita income gains are more than three times larger than estimates reported in Ashraf, Lester, and Weil (2008). Even distributing the currently available vaccine with 75% efficacy generates substantial long-run benefits, increasing per-capita income by 5.2%. 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. In fact, reducing mortality alone—without alleviating malaria's detrimental effects on learning capacity—leads per-capita income to *decline* by 0.4% due to increased population pressure. This finding aligns with Acemoglu and Johnson (2007), which documents that historical increases in life expectancy only raised population

[1] Bleakley (2010) makes a similar argument, although without explicitly considering the learning capacity.

without boosting per-capita income.

I conclude by conducting a cost-benefit analysis of the vaccination policy, comparing model-implied income gains to estimated costs from the epidemiological literature, including vaccine procurement, distribution, and administration. The model indicates that if vaccine efficacy exceeds 60%, costs per capita are fully recovered through increased income within one generation. Given the recently developed malaria vaccine has demonstrated 75% efficacy, these results strongly support the cost-effectiveness of mass vaccination. Despite these substantial benefits, however, vaccine rollout has been delayed due to financial challenges, primarily because malaria-endemic countries lack the fiscal capacity to independently finance deployment.[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. The findings of this paper suggests that delaying vaccine rollout not only costs lives but also postpones the realization of substantial macroeconomic benefits.

Related Literature This paper 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) replicate 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, 2023), and rural-urban migration (Lagakos, Mobarak, and Waugh, 2023). This paper is the first to extends this methodology to study general equilibrium effects of health intervention in the macro-development context.

This paper also contributes to a vast literature examining the relationship between health, human capital, and economic growth (Caselli, 2005). Empirical evidence on the role of health in explaining cross-country income differences remains mixed. Some studies, including Shastry and Weil (2003) and Weil (2007), find substantial impacts of eliminating health disparities on narrowing income gaps across countries. Similarly, Jones and Klenow (2016) highlights the importance of health in explaining cross-country welfare differences. In contrast, Acemoglu and Johnson (2007) exploit the large mortality declines during the

[2]The vaccine rollout heavily relies on the 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.

1940s international epidemiological transition and find little evidence that improved health raised per-capita income. For malaria specifically, cross-country regressions by Gallup and Sachs (2001) suggest malaria eradication could boost per-capita income growth in sub-Saharan Africa by 2.6% *per year*. Yet, Ashraf et al. (2008) argue that malaria eradication in Zambia would generate only modest gains of roughly 2% in per-capita GDP. These studies typically abstract from individuals' endogenous behavioral responses to health improvements. For example, Ashraf et al. (2008) assume that following malaria eradication, population growth rates revert exogenously to the pre-eradication levels. This paper extends the literature by developing a fully micro-founded structural model that explicitly incorporates individuals' behavioral responses, allowing aggregate outcomes to arise endogenously from optimizing behaviors. Since malaria predominantly affects children, accounting for how parents adjust investments in their children in response to malaria is essential (Schoellman, 2016).

The empirical results in this paper confirm previous findings on the effects of malaria eradication on children's education and women's fertility. Prior studies examining historical malaria eradication episodes consistently document positive impacts on children's educational attainment and some studies report higher adulthood earnings. (Cutler, Fung, Kremer, Singhal, and Vogl, 2010; Bleakley, 2010; Lucas, 2010; Venkataramani, 2012; Barofsky, Anekwe, and Chase, 2015; Shih and Lin, 2018). Closely related to this paper, Kuecken, Thuilliez, Valfort et al. (2021) analyzes the Roll Back Malaria campaign in 27 sub-Saharan African countries, finding increased educational attainment and reduced fertility. I complement these empirical analyses by providing a structural framework that leverages individual level estimates to quantify aggregate macroeconomic effects.

The remainder of the paper is structured as follows. Section 2 presents the quantitative model. Section 3 provides reduced-form empirical estimates of malaria's effects on fertility and educational investments. Section 4 calibrates the model using these empirical estimates. Section 5 examines the short- and long-run general equilibrium effects of malaria eradication through vaccination and evaluates the cost-effectiveness of this policy. Section 6 concludes.

2. Model

This section introduces the quantitative model. At its core, the model is an overlapping generations framework with endogenous fertility decisions, similar to those in Manuelli and Seshadri (2009) and Roys and Seshadri (2017), where parents value their children's human capital out of altruism (Barro and Becker, 1989). I incorporate malaria as an exogenous health shock affecting children by increasing mortality risk and lowering returns to

education.[3] Parents endogenously choose fertility and children's educational attainment, with these decisions directly 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 its core mechanisms.

2.1. Environment

Demographics and Environment Time is discrete, and each model period represents six years. The economy consists of N distinct locations, each characterized by different malaria prevalence. The population share of location $n \in 1, 2, \ldots, N$ is denoted by p_n. 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 12 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 $j \in 0, 1, \ldots, 12$.

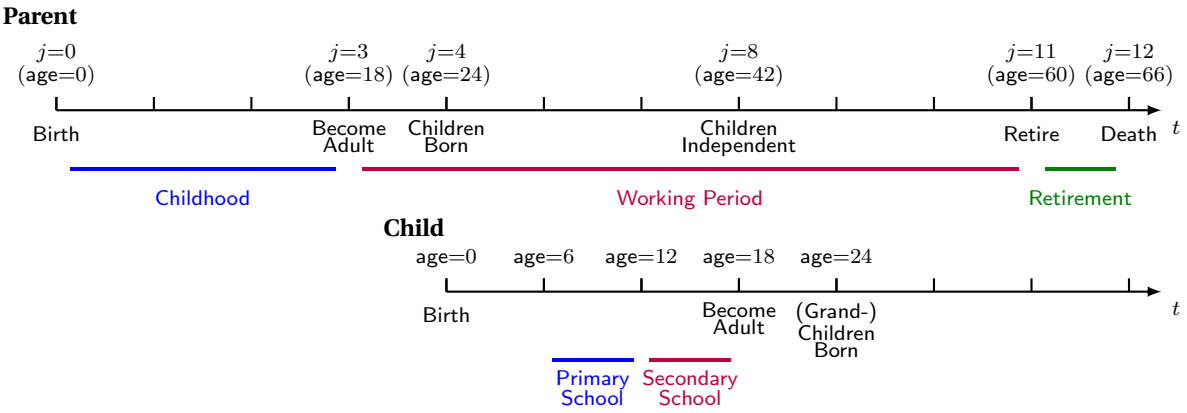


Figure 1: Life cycle, family structure, and stages of life

Each household consists of a parent and their cohabiting children, if any. Children live with their parents and make no independent 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 may

[3] 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 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.

accumulate savings at an exogenous interest rate r.[4] At age 24, individuals decide how many children to have and, conditional on having children, become parents.

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 live off their accumulated assets until death at age 66, which is chosen to match the average life expectancy in many sub-Saharan African countries.

There are four 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. Lastly, children face a health shock determining whether they contract malaria. This shock is drawn once in early childhood (at age six) and reduces the returns from schooling in subsequent periods of childhood. Below, I illustrate in detail how learning ability and health shocks are drawn, as well as 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.

[4] 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.

Malaria in Early Childhood In addition to learning ability, children draw an idiosyncratic health shock at age six.[5] 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 region j survives to the next period is denoted by χ_j^d. This mortality risk encompasses deaths from all causes, not only malaria. Since the analysis is focused 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 hence $\mu \times \chi_j^d$.

Morbidity risk is represented by a proportional reduction in learning ability, reflecting that malaria infection worsens learning outcomes both 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:

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

where j denotes the region and $\underline{m} < 1$ indicates the reduced health status 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_j^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 againsts their children's malaria infection, such as purchasing or using bednets. However, because the probability χ_j^m is parameterized using ex-post observed malaria prevalence, it implicitly accounts for preventive behaviors already adopted. Numerous studies document that more educated parents engage in greater preventive behavior, thereby lowering their children's malaria risk. To reflect this parental education-malaria gradient, I assume that children of parents who received secondary education experience reductions in both malaria-caused mortality and morbidity probabilities by a factor of $1 - \xi$. This formulation parsimoniously captures how parental education mitigates malaria risks.[6]

[5] 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.

[6] 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.

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 according to:

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

where η_s is the deterministic increase in human capital from school s, $s \in P, S$. As the expression shows, children's human capital in adulthood depends jointly on their learning ability, disease status, and parental schooling investments.

Sending children to school is costly. These costs are represented by per-child schooling fees p_P and p_S for primary and secondary schools, respectively, which enter the household budget constraint. Schooling fees encompass tuition, uniforms, and supplies such as textbooks, reflecting the goods cost of education.[7] Lastly, 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.

Production and Aggregation TA 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 production function is represented by the following CES aggregator:

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

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:

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

[7] I do not explicitly model child labor, as detailed data on child wages and child labor markets are not available. Earlier version of the paper (Kim, 2024) incorporated child labor by as lost labor income from school attendance under the assumption that children enter the same labor market as adults. The results are qualitatively similar.

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. Recursive Formulation of Decision Problems

After becoming independent, an individual's adulthood can be broadly divided into three stages: a short period when they live alone, periods when they live with their children, and periods where they become alone again after children become independent. Individuals solve a consumption-savings optimization problem throughout the lifetime. The utility function for consumption is given as:

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

When living with children, they also make fertility and 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 own earnings, it is included as 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 are drawn *i.i.d.* from a Gumbel distribution with variance σ_n. The value function corresponding to having n children can be expressed as follows:

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

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 for 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.[8][9] 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:

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

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

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

[9] 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 health of schoolchildren, including anemia, school absenteeism, convulsions, and poor school performances (Matangila, Fraeyman, Kambulu, Mpanya, da Luz, Lutumba, Van Geertruyden, and Bastiaens, 2017).

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 function at this stage 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 function for parents with secondary school-age children is:

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

As before, parents derive utility from their own consumption and their continuation value. 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): parents directly value the human capital their children will possess as adults (h_k) through function $\nu(\cdot)$, weighted by the discounting function $b(n)$.[10] I assume the utility for child quality has the following functional form: $\nu(x) = \frac{x^{1-\gamma}}{1-\gamma}$.

Age 42 to 66 ($j=7-12$): 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 stage is identical to (1). At age 60, individuals retire and no longer provide labor. The

[10] This approach to modeling altruism significantly reduces the computational burden. Earlier version of the paper featured a full dynastic altruism structure and yielded similar quantitative results. Daruich and Kozlowski (2020) and Zhou (2022) also develop quantitative models with endogenous fertility, where altruism is introduced in a similar way.

retirement-stage value function is:

$$V_j(a) = \max_{c,a'} u(c) + \beta V_{j+1}\left(a'\right)$$
$$c + a' \leq (1+r)a$$

2.3. Competitive Equilibrium and Balanced Growth Path

In this economy, population growth is endogenous due to endogenous fertility. I therefore focus on a balanced growth path, defined as an equilibrium where both the aggregate population growth rate and the distribution of households' states remain constant over time. Below, I formally define a recursive competitive equilibrium and a balanced growth path.

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.

Balanced Growth Path A balanced growth path is a particular case of the recursive competitive equilibrium that satisfies additional conditions. Let P denote 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.

2.4. Illustration: Impact of Malaria Eradication on Schooling and Fertility

The quantitative model features rich interactions between fertility, children's human capital, and malaria, captured by the quantity-quality tradeoff. To better illustrate the core mechanism, this section presents a simplified version of the model. Consider a model with continuous fertility choice, in which parents choose the number of children, denoted by b. It is assumed that each birth is associated with a time cost of p, representing the fraction of forgone wage due to child-rearing. Given a survival probability of $1-\chi_d$, the number of surviving children n is $(1-\chi_d)b$. In this simplified setup, parents derive utility from their own consumption and from the number of surviving children.

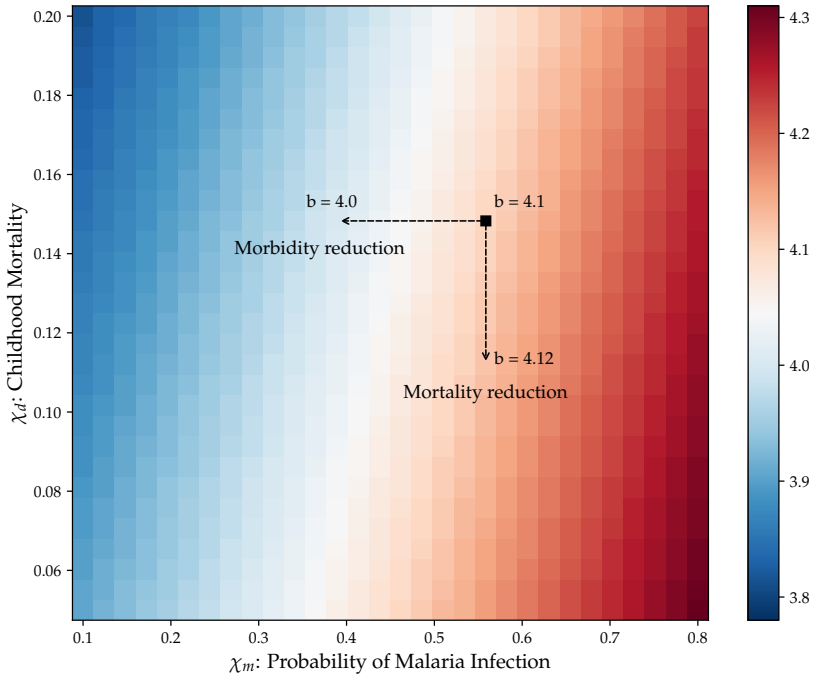
A parent can invest in the education of his/her surviving children through educational spending e. The motive for education is the same as in the quantitative model. A child's income is proportional to the level of human capital he/she possess, and the education provided by parents is converted into child's human capital by a concave, increasing function $h(e)$. The returns from education depends 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 parent's utility can be described as follows:

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

How does a reduction in malaria risk affect parents' fertility and educational expenditures? The simplified model provides useful intuition, which carries 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). 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. 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 $\sqrt{e}$. 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 illustrates how fertility decisions respond to reductions in malaria risk. First, 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 effec-

Figure 2: Optimal Fertility Conditional on Malaria Prevalence

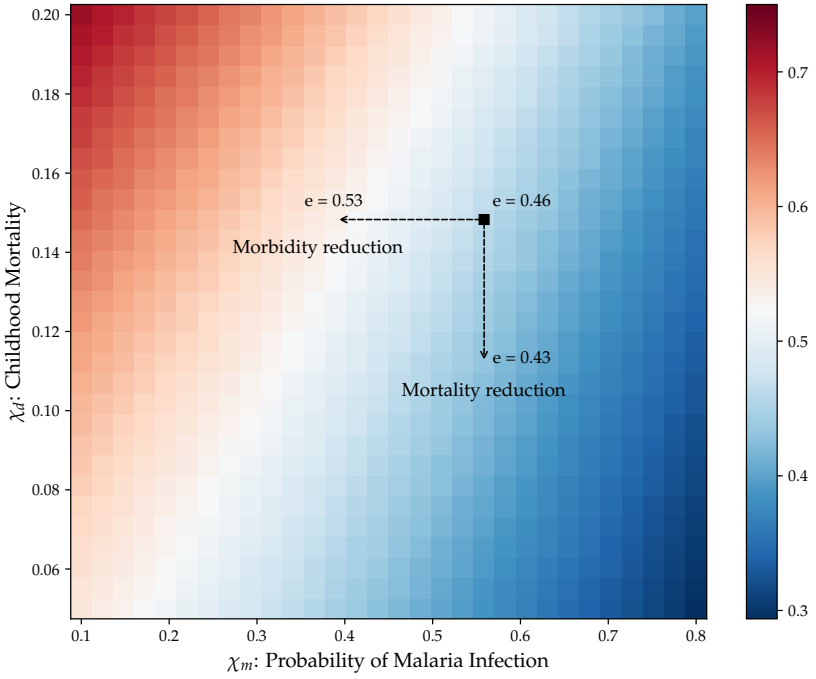


tive cost of producing a surviving child. Because children are a normal good, this leads to higher fertility, as illustrated in the figure. Lower morbidity, however, has an offsetting effect. A reduced probability of contracting malaria implies higher returns to educational investment, as children acquire more human capital for a given level of parental investment. These higher returns encourage parents to reduce fertility and invest more in each child's education, resulting in lower fertility overall.

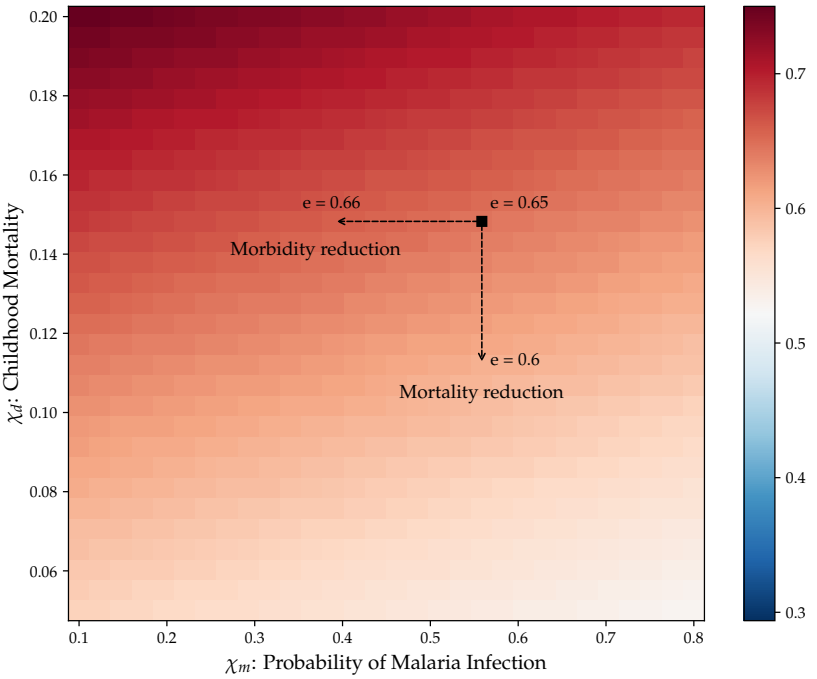
Figure 3a plots optimal educational investment for varying degrees of malaria prevalence. Qualitatively, the educational investment response to changes in malaria prevalence is the mirror image of the fertility response shown previously. When the mortality rate declines, educational investment decreases; 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 extent to which fertility and educational investment respond to reduced malaria prevalence depends on how severely malaria impacts human capital accumulation—that is, the parameter value of 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 economy ($m = 0.5$). In this alternative scenario, malaria still reduces the effectiveness of educational investment, but the effect is much smaller. Qualitatively, the responses to reductions in malaria prevalence remain consistent with the previous analysis: parents increase educa-

Figure 3: Optimal Educational Investment Conditional on Malaria Prevalence



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



(b) Optimal education, malaria highly severe ($m = 0.9$)

tional investment in response to reduced morbidity risk, and decrease it in response to reduced mortality risk. However, the magnitude of these responses is significantly diminished. 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 simplified model highlights how malaria eradication affects fertility and educational investment through the quantity-quality tradeoff. Importantly, the model indicates that aggregate data alone are insufficient to quantify the magnitude of these behavioral responses. A key reason is that the parameter governing malaria's detrimental effects on children's human capital accumulation, m, is inherently unobservable. To identify this latent parameter, one needs exogenous variation in malaria prevalence; observing how fertility and educational investment respond to such exogenous changes provides information about the magnitude of m. This motivates my approach of using quasi-experimental moments, in addition to aggregate moments, in estimating the parameters of the quantitative model. The following section empirically estimate the responses of fertility and children's educational attainment to an exogenous reduction in malaria prevalence, exploiting a large-scale anti-malaria campaign as a quasi-experimental setting.

3. Empirical Analysis of the Effects of Malaria on Fertility and Children's Human Capital

Motivated by the simplified model's implications, this section empirically estimates how fertility and children's educational attainment respond to an exogenous reduction in malaria prevalence. To this end, I exploit Tanzania's implementation of the Roll Back Malaria campaign, a recent large-scale anti-malaria intervention across sub-Saharan Africa. Using regional variation in pre-campaign malaria prevalence as the identifying source of variation, I employ a difference-in-differences design to estimate the causal effects of reduced malaria burden on fertility and children's human capital accumulation. The empirical estimates obtained in this section will serve as target moments for disciplining the model's key parameters in Section 4.

3.1. Background: The Roll-Back Malaria Campaign in sub-Saharan Africa

The Roll Back Malaria (RBM) Partnership was a large-scale anti-malaria initiative jointly 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. A distinguishing feature of the RBM compared to earlier anti-malaria efforts was its unprecedented external funding, totaling approximately $4.6 billion between 2003 and 2009. During this period, 81 of the 108 malaria-endemic countries received financial support from the global community for malaria

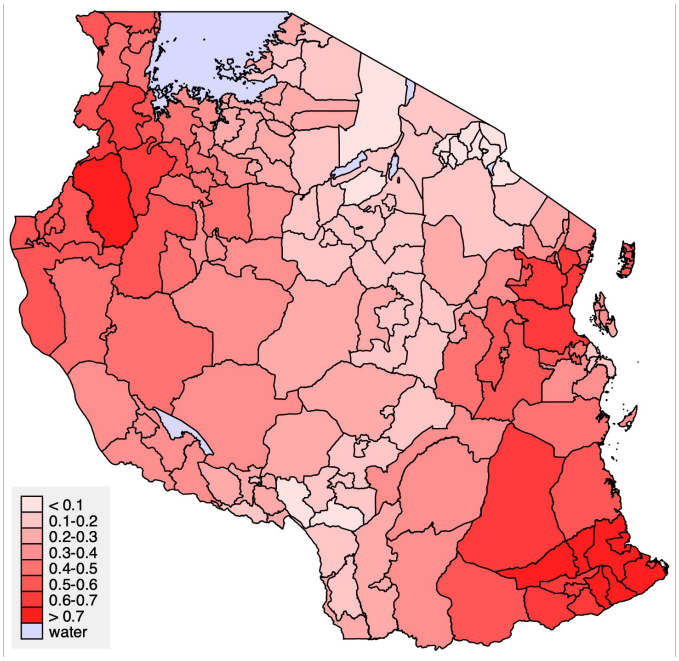
control (Johansson, Cibulskis, Steketee et al., 2010). Because sub-Saharan Africa accounted for around 85% of the global malaria burden, a significant share of this financial aid was directed toward African countries. The RBM strategy emphasized the massive distribution of insecticide-treated nets (ITNs) and indoor residual spraying (IRS), both proven to reduce malaria transmission. The intervention was highly successful; due to these coordinated international efforts, global malaria-related deaths had declined by half by 2014.

Among the recipient countries, Tanzania provides an ideal setting to study the effects of the RBM campaign, serving as a representative case for high-burden malaria countries in sub-Saharan Africa. Before the RBM campaign, more than 90% of Tanzania's population was at risk of malaria, categorizing it as a high-burden country. Malaria was a major contributor to childhood mortality; Tanzania had approximately 11 million clinical malaria cases annually, contributing to about 36% of all deaths among children under five (National Malaria Control Programme, 2010). Despite this substantial burden, malaria control efforts were minimal: prior to 2003, the coverage of ITNs was nearly zero nationwide. Tanzania was among the first twelve malaria-endemic countries to receive RBM funding starting in 2003, and the campaign was highly successful in reducing malaria prevalence.

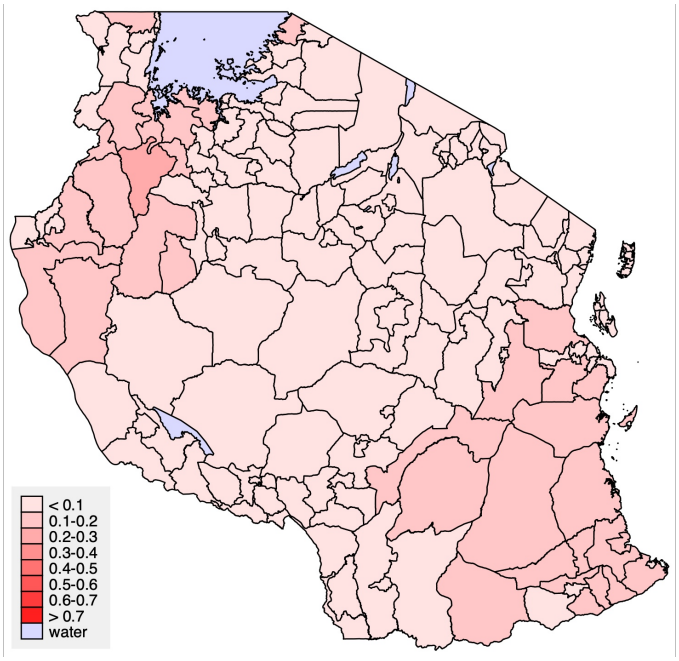
Financial support from the RBM enabled the Tanzanian government to scale up malaria interventions nationwide. ITNs began to be distributed to vulnerable groups in 2004, and free long-lasting insecticidal nets (LLINs) were provided to children under five starting in 2009. Indoor residual spraying was introduced in epidemic-prone areas in 2009 (National Malaria Control Programme, 2010). As a result of these coordinated efforts, Tanzania's malaria prevalence significantly decreased by 2012, approximately a decade after the campaign began. Figure 4 illustrates the reduction in malaria prevalence over this period. Malaria prevalence is measured by PfPR_{2-10}, which denotes the proportion of children aged 2 to 10 carrying *P. falciparum* parasites, the most common malaria parasite in sub-Saharan Africa, in their blood. PfPR_{2-10} is commonly used as an index of malaria prevalence and transmission intensity, and I continue using it as the main proxy for malaria prevalence in subsequent analyses.

Tanzania's successful reduction in malaria burden, combined with the availability of detailed micro-data, allow us to empirically estimate the effects of the RBM campaign on fertility and children's human capital outcomes. The primary dataset I use is the Population Census, with waves in 1988, 2002, and 2012. Unlike the frequently used Demographic and Health Surveys (DHS), Census data contain a broader set of variables, allowing better identification of the RBM campaign's effects on outcomes of interest. The following subsection describes the dataset's structure and highlights its advantages relative to other commonly used datasets.

Figure 4: Spatial distribution of malaria prevalence rate, pre- and post- campaign



(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 are 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 Malaria Atlas Project (MAP).

3.2. Data

I use two main datasets for the empirical analysis. First, malaria prevalence data are obtained from the Malaria Atlas Project (MAP). Second, household- and individual-level information on socioeconomic characteristics, mortality, fertility, and parental investment in children's human capital come from three waves of the Tanzania National Population Census. Below, I describe each dataset separately.

Malaria Atlas Project (MAP) I obtain information on malaria prevalence from the Malaria Atlas Project (MAP), an international academic organization which provides annual estimates of malaria prevalence for multiple sub-Saharan African countries.[11] Within each country, MAP reports regional prevalence estimates down to second-level administrative units (GIS-2 level). From this dataset, I construct the spatial distribution of malaria risk before and after the RBM campaign. I use region-level averages of the PfPR as the measure of malaria prevalence. Figure A.1 illustrates the changes in PfPR between 2001 (pre-campaign) and 2012 (post-campaign). As shown, regions with initially high malaria prevalence experienced greater reductions in malaria burden following the campaign.

I merge the regional malaria prevalence data from the MAP with the Tanzania National Census data based on the region of residence of census households. MAP uses administrative boundaries as defined by the Tanzania National Bureau of Statistics in 2012. For cases where administrative boundaries have changed over time, I harmonize the geographic boundaries using spatially harmonized boundaries for 1988–2012 provided by the IPUMS International.

Tanzania Population Census Individual-level data on fertility and educational attainment come from three waves of the Tanzania National Census (1988, 2002, and 2012). I focus on three outcome variables: mortality, fertility, and children's schooling (as a proxy for human capital). The Census collected complete birth histories from each female respondent, including the number and timing of all births and information about child mortality. I measure mortality using the number of children who had died by the time of the survey. For fertility, I use two measures: the total number of children ever born and the number of surviving children. The former captures gross fertility, including deceased children, while the latter represents net fertility. The baseline analysis of fertility and mortality outcomes restricts the sample to women aged 30–49. Lastly, I measure children's human capital by their years of schooling completed at the time of the survey.

The Tanzania Population Census offers advantages over the commonly used Demographic and Health Surveys (DHS) for estimating the effects of anti-malaria campaigns. First, unlike the DHS, the Census provides information on respondents' regions of birth as well as

[11] MAP also provides geographical data relating to malaria prevalence and related topics for the World Health Organization. See https://malariaatlas.org.

current residence. By restricting the sample to individuals residing in their birth region, I can control for internal migration, which could otherwise confound the estimates. Second, although the DHS covers multiple sub-Saharan African countries, focusing on a single country reduces estimation issues related to heterogeneous timing of interventions across countries.[12] Lastly, sample size is much larger in the Census data.

3.3. Empirical Specification and Identification

The estimation strategy closely follows Wilde, Apouey, Coleman, and Picone (2019) and Kuecken et al. (2021), which studied the RBM campaign for multiple countries in sub-Saharan Africa. Since the RBM campaign was targeted toward regions with high malaria prevalence, I exploit pre-campaign malaria prevalence as a proxy for campaign intensity. Specifically, I categorize each region into one of four groups based on their 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%).[13]

For mortality and fertility outcomes, which are count data (number of children ever born or died), I estimate Poisson regression models, specifying the logarithm of the expected number of child deaths or births experienced by a woman as the dependent variable.[14] Specifically, I estimate the following mortality and fertility equations:

$$M_{irt}^{c} = \beta_1^m \text{Post}_t + \sum_{j=2}^{4} \beta_j^m \text{Post}_t \times \text{Prev}_{r,2001}^{j} + \boldsymbol{X}_{ijct}{}' \cdot \boldsymbol{\Gamma} + \eta_r \tag{5}$$

$$F_{irt}^{c} = \beta_1^f \text{Post}_t + \sum_{j=2}^{4} \beta_j^f \text{Post}_t \times \text{Prev}_{r,2001}^{j} + \boldsymbol{X}_{ijct}{}' \cdot \boldsymbol{\Gamma} + \eta_r \tag{6}$$

where M_{irt}^{c} and F_{irt}^{c} are the logarithms of the expected numbers of child deaths or births experienced by woman i in age group c at time t, born and surveyed in region r. The variable Post$t$ is an indicator equal to one for observations in 2012 (post-treatment) and zero in 2002 (pre-treatment). Prev$^{j}r, 2001$ indicates whether region r belongs to prevalence group j, with $j \in 2, 3, 4$, where group 4 is the highest prevalence category (PfPR $>$ 75%). The vector $\boldsymbol{X}_{irct}$ includes control variables: age, years of schooling of the respondent, and urban-rural residence status. All specifications include region fixed effects (η_r).

[12] Recent econometric literature highlights that two-way fixed effects (TWFE) difference-in-differences estimates can be biased when treatment timing differs across groups (Callaway and Sant'Anna, 2021). Since the RBM campaign began nationwide in Tanzania in 2003, my estimates avoid such concerns.

[13] These thresholds align closely with standard epidemiological classifications, though epidemiologists typically label a region as low prevalence (hypoendemic) below 10%. I use a 20% cutoff to facilitate empirical analysis, but I also conduct robustness checks with alternative cutoff values. Robustness results are provided in Appendix C.

[14] Poisson regression is a commonly used method in the analysis of survival data. See Lu and Vogl (2022) for an application to the analysis of child mortality.

For education outcomes, I estimate the following OLS regression:

$$E^c_{irt} = \beta^e_1 \text{Post}_t + \sum_{j=2}^{4} \beta^e_j \text{Post}_t \times \text{Prev}^j_{r,2001} + \boldsymbol{X}_{ijct}{}' \cdot \boldsymbol{\Gamma} + \eta_r + \varepsilon_{ijt} \tag{7}$$

where E^c_{irt} denotes the years of schooling for child i in age group c at time t, born and surveyed in region r. Control variables remain the same as those for fertility and mortality, except that years of schooling is excluded. Unlike fertility and mortality regressions, this specification includes both male and female respondents.

In the main empirical analysis, I primarily focus on comparing low- and high-prevalence regions as control and treatment groups, respectively. As illustrated in Figure A.2, malaria prevalence remained consistently low from 2001–2012 in low-prevalence regions, whereas high-prevalence regions experienced substantial reductions following the RBM campaign onset in 2003. Although medium-low and medium-high prevalence regions also saw reductions, attributing these declines to the RBM campaign is challenging due to pre-existing secular trends in malaria prevalence.[15] Appendix Table B1 provides descriptive statistics for the full sample and separately for high- and low-prevalence regions.

The primary coefficient of interest in all specifications is β_4, the coefficient on the interaction term Post$t \times$ Prev$^4 r, 2001$. If the RBM campaign effectively reduced malaria-related child mortality and fertility in high-prevalence regions, we would expect $\beta_4^{m,f} < 0$. Similarly, if the campaign positively impacted children's educational attainment, we would expect $\beta_4^e > 0$.

3.4. Results and Interpretation

3.4.1. Child quality: RBM's effects on children's educational attainment

Figures A.5 and A.6 illustrate the parallel trends in years of schooling between high- and low-prevalence regions from 1998 to 2012. Table 1 presents the estimates of regression equation (7). Each column reports the coefficient β_4^e for different age groups, corresponding to varying levels of exposure to the RBM campaign. Columns 1 to 3 represent cohorts who are increasingly less likely to have benefited from the campaign. For example, column 1 reports results for children aged 10–15 in 2012, who were born after the campaign began and therefore likely received its full benefits. By contrast, the last column represents an older cohort unlikely to have benefited from the campaign, as these individuals had largely completed their schooling by its start. Hence, the final column serves as a placebo group. I report only the coefficients on the interaction term between the post-treatment indicator and the high-prevalence region dummy, as the analysis directly compares high- and low-prevalence regions as treatment and control groups.

[15] Results are robust to the exclusion of medium-low and medium-high prevalence groups and are available upon request.

Table 1: Effects of the RBM on Years of Schooling

	Age group in 2012			
	Age 10-15	**Age 15-20**	**Age 20-25**	**Age 25-30**
Dependent variable mean in 2012	4.29	6.91	6.86	6.06
$PfPR_{2-10}$ (75%+) × Post	0.689***	1.003***	0.562***	-0.046
	(0.096)	(0.131)	(0.107)	(0.119)
Observations	1,258,221	1,043,818	875,946	812,544

Notes: This table reports the estimation restuls from OLS regression (7). Brackets contain standard errors clustered at the region level. $PfPR_{2-10}$ (75%+) × Post indicates the interaction between the indicator of high-prevalence regions (PfPR in 2001 exceeding 0.75) and the post-treatment indicator. Samples are restricted to the individuals who were born and residing (surveyed) in the same region in 2012. Control variables included are age and urban-rural residential status. All columns include region fixed effects. Full table containing the estimates for other prevalence groups can be found in Table B2. *, **, and *** indicate significance at the 10, 5, 1% levels.

The positive and significant coefficients indicate that children born in regions with the highest malaria prevalence in 2001 (and therefore most exposed to the RBM campaign) experienced increased educational attainment.[16] Compared to low-prevalence regions, children from high-prevalence regions who were aged between 1 and 6 at the campaign's onset completed an additional 0.69 years of schooling. Children who were already in school at the start of the campaign also benefited; those aged 6–11 experienced an additional 1.00 years of schooling. However, the positive effects dissipate among the older cohort (ages 25–30 in 2012), as these individuals had largely completed their education before the campaign began.

The magnitude of these effects aligns with estimates from the existing literature. For instance, Lucas (2010) estimate that malaria eradication increased female educational attainment by as much as two years in the most heavily infected regions of Sri Lanka following its 1945 eradication campaign. Similarly, Bleakley (2010) document positive effects on children's educational attainment following historical malaria eradication episodes in six Latin American countries. More recently, Kuecken et al. (2021) estimate a 0.4-year increase in educational attainment among children exposed to the RBM campaign.[17]

3.4.2. Child quantity: RBM's effects on mortality and fertility

Figures A.3 and A.4 illustrate the parallel trends in mortality and fertility by plotting the predicted values from OLS versions of equations (5) and (6), conditional on observable control variables. Both mortality and fertility rates were declining across all regions; however, mortality rates decreased more sharply in high-prevalence regions following the introduction

[16] Results are similar for boys and girls. Appendix Table D2 reports separate regressions by gender.
[17] For a comprehensive review of the literature, see Currie and Vogl (2013).

of the RBM campaign.

Table 2: Effects of the RBM on Fertility

Dependent variable	Gross Fertility Children ever born	Mortality Children ever dead	Net Fertility Surviving children
Dependent variable mean in 2012	4.97	0.69	4.51
PfPR_{2-10} (>75%) × Post	–0.0613***	–0.0941***	–0.0321**
	(0.00978)	(0.0305)	(0.0160)
Observations	663898	633683	633567

Notes: This table reports the estimation results for the Poisson regression (5) and (6). PfPR_{2-10} (75%+) × Post indicates the interaction between the indicator of high-prevalence regions (PfPR in 2001 exceeding 0.75) and the post-treatment indicator. Samples are restricted to women between age 30 and 49 in 2012, and those who were born and residing (surveyed) in the same region in 2012. Control variables included are age and years of schooling of the respondents and urban-rural residential status. All columns include region fixed effects. Full table containing the estimates for other prevalence groups can be found in Table B3. *, **, and *** indicate significance at the 10, 5, 1% levels.

Table 2 reports the coefficients from regressions (5) and (6). Each cell presents results from a Poisson regression, with coefficients interpreted as changes in the log of the expected value of the dependent variable between high- and low-prevalence regions. Specifically, the coefficient β_4 implies that being in a high-prevalence region multiplies the mean of the dependent variable by $\exp(\beta_4)$. For example, a negative coefficient on the number of children ever born (β_4^f) indicates that women in high-prevalence regions reduced their fertility in response to the RBM campaign.

Gross Fertility Column 1 reports the results for gross fertility, measured by the number of children ever born. The coefficient β_4^f indicates that childbearing-age women in high-prevalence regions reduced fertility by 6.13%. This negative fertility response aligns with findings by Kuecken et al. (2021), who also documented reduced childbirth probabilities following the RBM campaign across multiple sub-Saharan African countries. Within the quantity-quality framework, the negative fertility response suggests that the RBM-induced reduction in the cost of child *quality* outweighed the reduction in the cost of child *quantity*.

Child Mortality Although the RBM campaign reduced gross fertility, it does not necessarily imply a decline in *net* fertility, as the campaign simultaneously reduced child mortality. If the decline in mortality exceeded the drop in gross fertility, net fertility could still increase. Column 2 in Table 2 shows the extent to which the RBM campaign reduced child deaths in high-prevalence regions. The coefficient β_4^m indicates that the RBM campaign

reduced the number of child deaths per woman by 9.4%, confirming the campaign's effectiveness in reducing child mortality.[18]

Net Fertility Column 3 combines the results from gross fertility and child mortality, showing the RBM campaign's effect on *net* fertility, measured by the number of surviving children. Although the coefficient is negative, the magnitude of reduction is smaller than that of gross fertility due to the offsetting effects of reduced child mortality. The results suggest that the RBM campaign lowered the number of overall births women have by 3.2%. [19]

3.4.3. Summary of Empirical Analysis

In summary, the empirical results indicate that the RBM campaign reduced women's fertility by 6.1% and child mortality by 10.7%. Together, these effects imply a 3.2% reduction in net fertility. The campaign also significantly improved children's educational outcomes, with treated cohorts obtaining between 0.56 and 1.0 additional years of schooling compared to untreated cohorts. These empirical findings are consistent with existing studies on anti-malaria interventions and historical malaria eradication episodes, which document positive impacts on children's education. The fertility results are consistent with existing studies on the RBM campaign in other countries/context, which document a negative response in fertility. In the next section, I use these empirical estimates to discipline the quantitative mechanisms of the structural model, replicating the RBM campaign within the model and targeting the regression coefficients estimated here.

4. Calibration

The goal of the quantitative model is to assess the macroeconomic consequences of malaria eradication. As discussed in Section 2.4, this requires quantifying malaria's deleterious effects on children's human capital accumulation and understanding how parents respond in their fertility and educational investment decisions. I achieve this by replicating the RBM campaign within the model and matching the estimated empirical effects on fertility and educational attainment. To this end, I calibrate the model to match the Tanzanian economy in 2002, one year before the RBM campaign was implemented. To further ensure the model is credible in other dimensions, I jointly estimate parameters to match other relevant aggregate moments of the Tanzanian economy, calculated from microdata when available or taken from existing literature otherwise.

[18] These results are consistent with Wilde et al. (2019) and Kuecken et al. (2021), who also found that the RBM campaign significantly reduced all-cause child mortality.

[19] Another interpretation is a possibility that the RBM induced women to delay their fertility. Although possible, it is not likely to change the conclusion because the sample is restricted to women with near-completed fertility. As the chance of pregnancy declines with age, it is unlikely that it will lead to an increase in net fertility.

4.1. Exogenously Chosen Parameters

A set of parameters is chosen exogenously, as summarized in Table 3. 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 3: 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 regions						
N	Number of regions	4				Section 3
ξ	Educ. disparity in malaria risk	0.62				Ogbo et al. (2019)
μ	Share of malaria-caused death	0.17				See text
Region(j)-specific parameters		*Malaria prevalence (PfPR)*				
		<20%	20-50%	50-75%	>75%	
χ_j^d	Under-five Mortality rate	0.097	0.137	0.128	0.156	DHS 1999
χ_j^m	Prob. catching malaria	0.132	0.325	0.580	0.765	MAP
p_j	Population share	0.313	0.443	0.216	0.028	2002 Census

Standard Parameters Panel A of Table 3 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 chosen as 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 de-

gree 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.[20] 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 regions to four, consistent with the empirical regression specification classifying districts based on malaria prevalence. Each district is assigned to one of the four regions based on the district-level 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%). The population shares, p_j, are then calculated for each region from the 2002 Census.

Two sets of parameters are required for each region: mortality (probability of death) and morbidity (probability of contracting malaria). The mortality parameters, χ_j^d, 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_j^d includes mortality from all causes, not only malaria. According to the Institute for Health Metrics and Evaluation (IHME), approximately 17% of under-five deaths in Tanzania in 2002 were attributable to malaria.[21]

For morbidity parameters, I use region-specific averages of malaria prevalence (PfPR). The region 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) report 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.

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

[21] An alternative estimate comes from household surveys. The Tanzania National Panel Survey (waves from 2008 to 2014) indicates malaria accounts consistently for around 51% of identifiable childhood deaths (see Table B4). However, this likely represents an upper bound, as only 5.9% of total deaths were diagnosed, meaning that the majority of people could not identify the exact cause of deaths. Since malaria is relatively easier to identify, it is likely to be over-represented.

4.2. 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 eleven parameters:

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

Table 4 reports the estimated values of the parameters. The first ten parameters are calibrated primarily to match aggregate statistics from the pre-RBM Tanzanian economy. The last parameter, $\underline{m}$, captures malaria's negative impact on children's human capital accumulation. The magnitude of this parameter determines how educational investments respond to reductions in malaria prevalence, and also influences fertility decisions through the quantity-quality tradeoff. Because human capital is inherently unobservable, this parameter is disciplined by matching the reduced-form empirical estimates from the RBM campaign. The following subsections describe the calibration approach and model fit in detail.

Table 4: 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	1.35	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
$\underline{m}$	0.88	Severity of malaria morbidity shock

4.2.1. Parameters Estimated from Aggregate Data

Table 5 summarizes the internal 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 age 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.

Table 5: Targeted Moments and Model Fit

Moments	Source	Data	Model
Education			
Primary completion rate (%)	Tanzania Census 2002	69.6	69.6
Secondary completion rate (%)	Tanzania Census 2002	13.1	13.8
Primary ed. wage premium (%)	Leyaro et al. (2014)	59.9	55.5
Secondary ed. wage premium (%)	Leyaro et al. (2014)	115.2	119.5
Differential Fertility			
Total fertility rate	Tanzania DHS 1999	5.90	5.73
Total fertility rate, unskilled parents	Tanzania DHS 1999	6.06	6.05
Total fertility rate, skilled parents	Tanzania DHS 1999	3.08	3.07
% Secondary parents with 8 or more children	Tanzania DHS 1999	5.78	5.19
Intergenerational Mobility and Inequality			
Primary-Secondary IGM	Alesina et al. (2021)	13.9	13.7
Gini coefficient	Younger et al. (2016)	0.38	0.44

The education premiums that primary- and secondary-educated workers earn relative to the uneducated group are taken from Table 3 of Leyaro, Twumasi Baffour, Morrissey, and Owens (2014), who estimated these premiums using the 2001/2006 Tanzania Integrated Labour Force Survey.[22] The parameters related to these moments, η_P and η_S, govern the increase in human capital from completing primary and secondary schooling, respectively. The estimated parameter 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

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

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 1.35. This parameter captures variation in fertility choices unexplained by economic incentives. 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 the primary-to-secondary intergenerational mobility, 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.

4.2.2. Parameters Estimated from the RBM Campaign

Until now, the calibration has sought parameters which, in equilibrium, replace important aggregate and distributional characteristics of the pre-RBM Tanzanian economy. These population moments are informative in that they place restrictions on the potential macroeconomic effects of malaria eradication. Beyond these distributional moments, the calibration also targets the estimated marginal effects of the RBM campaign on fertility and children's educational attainment at the microeconomic level. Matching this evidence is important because it disciplines how individual households, which constitutes the model's micro-foundation, respond to an exogenous improvement in malaria environment. In this analysis, these targeted moments correspond to the empirical estimates presented in Section 3.

To achieve this, I replicate the RBM campaign within the model as an unexpected, universal reduction in the probabilities of mortality and morbidity. Malaria prevalence declined by approximately 77% in the high-prevalence region and 61% in the low-prevalence region, compared to pre-campaign levels (see Figure A.2). Guided by this fact, I reduce the morbidity probability χ_j^m and the malaria-attributed component of mortality probability $\mu \times \chi_j^d$ by 77% in the high-prevalence region and and 1% in the low-prevalence region across all

schooling groups s. Although the proportional reduction in malaria risk is similar across the two regions, the level of reduction is much larger in the region with high malaria prevalence.

I also ensure that households in the model economy do not anticipate the reduction in malaria risk, thereby preventing behavior such as asset accumulation in preparation for increased educational investments post-campaign. To implement this, I first simulate the balanced growth path of the economy under pre-RBM malaria risks and subsequently introduce the RBM campaign an unexpected reduction in malaria risk. Households then adjust their fertility and education decisions based on the new decision rules under the changed malaria environment.

Specifically, I simulate two samples: one from the economy in which the RBM campaign is introduced, and another from the economy without the campaign, which serves as a control group. Using these simulated datasets, I regress individuals' fertility and their children's educational attainment on a dummy variable indicating exposure to the RBM campaign and its interactions with region dummies. In other words, I replicate the empirical regressions of Section 3 with simulated data, and compare the resulting coefficients to their empirical counterparts. The results are presented in Table 6, displaying the original empirical regression coefficients alongside those generated from the model-based regressions.

Table 6: Internal Calibration Targets on the Effects of the RBM Campaign

Moments for Indirect Inference	**Data**	**Model**
RBM Treatment Effect on Schooling (Years)	0.56 – 1.00	0.47
RBM Treatment Effect on Gross Fertility	−6.13%	−5.94%
RBM Treatment Effect on Child Mortality	−9.41%	−9.28%
RBM Treatment Effect on Net Fertility	−3.21%	− 4.74%

The empirical evidence primarily disciplines the calibration of $\underline{m}$ through indirect inference. Conditional on matching other aggregate moments, the average effects of an exogenous reduction in malaria risk on fertility and educational investment primarily depend on the latent parameter $\underline{m}$. As illustrated in Section 2.4, a lower value of $\underline{m}$ would generate a larger improvement in educational investment and a sharper decline in fertility. The estimated value of $\underline{m}$ is 0.88, indicating that children with malaria experience 12% lower returns from schooling. This estimate is consistent with epidemiological findings regarding malaria's detrimental impact on children's cognitive abilities and educational performance. For instance, in a study from Sri Lanka, Fernando, Gunawardene, Bandara, De Silva, Carter, Mendis, and Wickremasinghe (2003) find that children experiencing fewer

than three malaria attacks scored at least 15% higher in both special and school examinations compared to those who suffered more than five attacks during the same period. Similarly, a study conducted in Yemen by Al Serouri, Grantham-McGregor, Greenwood, and Costello (2000) concludes that experiencing at least one malaria attack was significantly associated with poor school performance (below the 50th percentile in class).

5. Macroeconomic Consequences of Eradicating Malaria

Using the estimated model, this section reports the results of two computational exercises which quantify the aggregate impact of malaria eradication on per capita income, educational attainment, and fertility. Malaria eradication is modeled as resulting from a nationwide vaccination campaign. Vaccination has long been considered the ultimate tool to eradicate malaria, though progress toward an effective vaccine had been historically slow. However, two recently developed malaria vaccine are known to provide up to 75% effective in preventing the disease among children under five.[23] 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 ($\mu \times \chi_j^d$) and morbidity shock probability (χ_j^m) for all region j.

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. The second exercise considers the long-run effects by computing 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 should be interpreted as an accounting exercise that provides benchmarks on the quantitative importance of childhood health in explaining cross-country income differences.

5.1. Short-Run Impact of Malaria Vaccine

The first exercise quantifies the short-run, one-generational impact of distributing the malaria vaccine. Empirical studies examining historical malaria eradication episodes — often leveraging cross-cohort variation in malaria exposure — consistently find that avoiding malaria in childhood is associated with a 10–20% increase in adult earnings at the individual level. The short-run exercise extends these individual-level findings by providing a benchmark for the *aggregate* gains from malaria eradication in the short term.

[23] 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 first column of Table 7 present 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 took place. 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 3.93%. 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. Specifically, the primary school completion rate rises by 1 percentage point, while the secondary school completion rate increases by 1.4 percentage points.

Table 7: Short-Run Effects of Malaria Vaccination

	Baseline	**No Improvement in Morbidity**	**Exogenous Fertility**
Primary completion rate (%)	+ 0.98	+ 0.98	+ 0.45
Secondary completion rate (%)	+ 1.39	+ 1.39	− 0.47
Δ Per capita income	+ 3.93%	+ 1.06%	+ 1.85%

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 that corresponds to the pre-vaccine balanced growth path. Parents still make education investments choice in this case.

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 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 the elimination of morbidity shocks. This is one channel that distinguishes this paper from previous macroeconomic studies. For instance, Ashraf et al. (2008) assume 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 (3.93%) is nearly twice 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 in which educational attainment increases, but the amount of human capital acquired in school re-

mains unchanged. In this scenario, children continue to suffer from the negative 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 7. The model predicts that without improved learning in school, the predicted increase in earnings would be only 1.06%. This suggests that the increase in years of schooling alone accounts for just 27% 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 the impact of this mechanism, I simulate 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 7, 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 half, while the change in secondary school enrollment becomes negative. These findings highlight the central role of fertility adjustments in amplifying the long-term gains from malaria eradication.

5.2. 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 debatable whether malaria eradication affects only one generation, without influencing subsequent generations' educational decisions. If children who benefit from eradication further increase educational investment in their own 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.

Nevertheless, unlike the short-run analysis, the long-run computations account for endogenous adjustments in both skilled and unskilled wages in response to the improved disease environment. While 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 that they provides informative benchmarks for assessing the aggregate economic loss attributable to the burden of malaria.

Table 8: Long-Run General Equilibrium Impact of Malaria Vaccines

	Baseline	**Vaccine w/ 75% Efficacy**	**Full Eradication**
Education			
Primary completion rate (%)	69.6	71.5	72.2
Secondary completion rate (%)	13.8	16.5	17.7
Differential Fertility			
Total fertility rate	5.73	5.38	5.26
Total fertility rate, unskilled parents	6.05	5.71	5.60
Total fertility rate, skilled parents	3.07	2.95	2.91
Intergenerational Mobility and Inequality			
Primary-Secondary IGM	13.7	16.4	17.7
Gini coefficient	0.44	0.43	0.43
Change in Per Capita Income and Prices			
Δ Per Capita Income		$+5.2\%$	$+6.8\%$
Δ Unskilled wage		$+0.6\%$	$+0.7\%$
Δ Skilled wage		-2.2%	-2.5%

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 mortality ($\mu \times \chi_j^m$) and morbidity probabilities (χ_j^d) 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.

Table 8 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 ($\mu \times \chi_j^m$) and morbidity probabilities (χ_j^d) are lowered by 75%, the level of efficacy of currently available malaria vaccines. The third column corresponds to the steady state economy with hypothetical vaccine with 100% efficacy, hence fully eradicating malaria. The table reports steady state changes in each of the main outcome variables following the implementation of malaria vaccine. In addition to the educational attainment and per capita income, Table 8 also reports outcomes related to fertility, intergenerational mobility and inequality. I also present how unskilled and skilled wages (w_U and w_S)

responds to the changing skill composition of workers.

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 (2020), 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, the primary channel through which per capita income rises is improved educational attainment. 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 0.7 percent while the skilled wage falls by 2.3 percent, reflecting the larger supply of skilled workers in the post-vaccine steady state.[24]

The model also predicts improvements in intergenerational mobility and a reduction in inequality. Specifically, the probability that children of parents who completed primary school go on to attend secondary school increases by 2.7 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 negative effects of malaria on their children's human capital by maintaining school attendance. By reducing the incidence of malaria, vaccination narrows this gap in educational investment, subsequently leading to lower inequality income inequality as well.

Turning to the full eradication scenario, the 6.8% increase in long-run per capita income predicted by the model appears notably larger than estimates reported in the existing literature. For example, Acemoglu and Johnson (2007) examine 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 to 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 calibrated model also indicates that a reduction in mortality is not sufficient to generate transformative gains in per capita income if it is not accompanied by a reduction in morbidity. Column 2 of Table 9 presents long-run steady-state results for a scenario in which only mortality declines,

[24]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.

while the morbidity risk from malaria remains unchanged. This corresponds to a setting where life expectancy rises without any improvement in health quality, particularly in dimensions that affect learning outcomes.[25] Consistent with the findings of Acemoglu and Johnson (2007), the model predicts a 1.3% *reduction* in per capita income in the long run, alongside an increase in the population growth rate.

Table 9: Decompositon of the Long-Run Effects of Complete Eradication

	Pre-Vaccine BGP	**Lower Mortality**	**Lower Morbidity**	**Lower Both**
Population Growth Rate (%)	3.95	4.02	3.62	3.66
Primary Completion Rate (%)	69.6	66.5	74.7	72.2
Secondary Completion Rate (%)	13.8	13.8	17.7	17.7
Δ Per capita earnings		$-$ 1.3%	$+$ 7.9%	$+$ 6.8%

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 an 100% reduction in malaria mortality ($\xi \times \chi_j^d$) while the morbidity shock probability (χ_j^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.

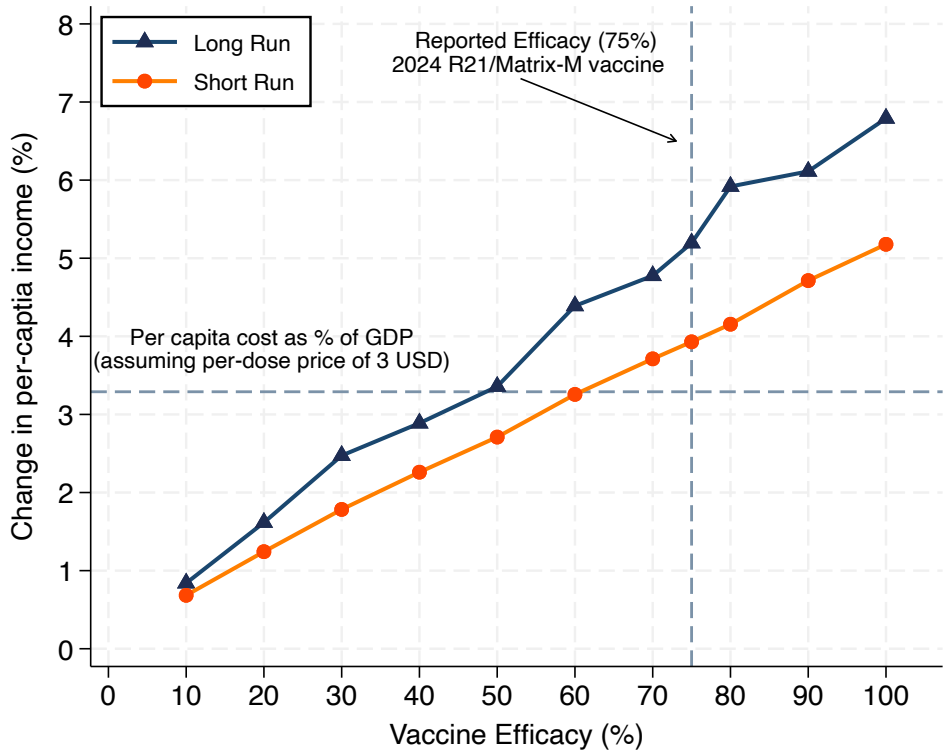
The reason that quantitatively similar reductions in mortality yield markedly different outcomes in per capita income lies in the endogenous responses of fertility and human capital investment. When a decline in mortality is not accompanied by a reduction in morbidity, the quantity–quality tradeoff predicts 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 9. The calibrated model suggests that, in the case of malaria, the effect of reduced morbidity dominates. These findings underscore that a uni-dimensional, mortality-focused approach to modeling disease impacts may lead to biased conclusions.

[25] Several features of the period studied by Acemoglu and Johnson (2007) suggest that the international epidemiological transition did relatively little to enhance children's learning outcomes. In the 1940s, schooling was not yet 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.

5.3. 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 expenses.[26] Furthermore, the actual efficacy of the vaccine may fall short of the current 75% estimate. If efficacy is lower, the costs of vaccination could potentially exceed the resulting benefits.

Figure 5: Vaccine Efficacy and the Cost of Vaccination



Notes: Y-axis is the long-run percentage change in per-capita output between the pre-and post-vaccination balanced growth paths. Orange dot at the 80% efficacy denotes the reported efficacy of the current vaccine.

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 to the per-child cost of vaccination. Following Sicuri et al. (2019), I assume a per-dose cost of 3 USD, implying a total vaccination cost of 18.9 USD per child in 2015 dollars. Given Tanzania's GDP per capita in 2001 was 573 USD[27], this

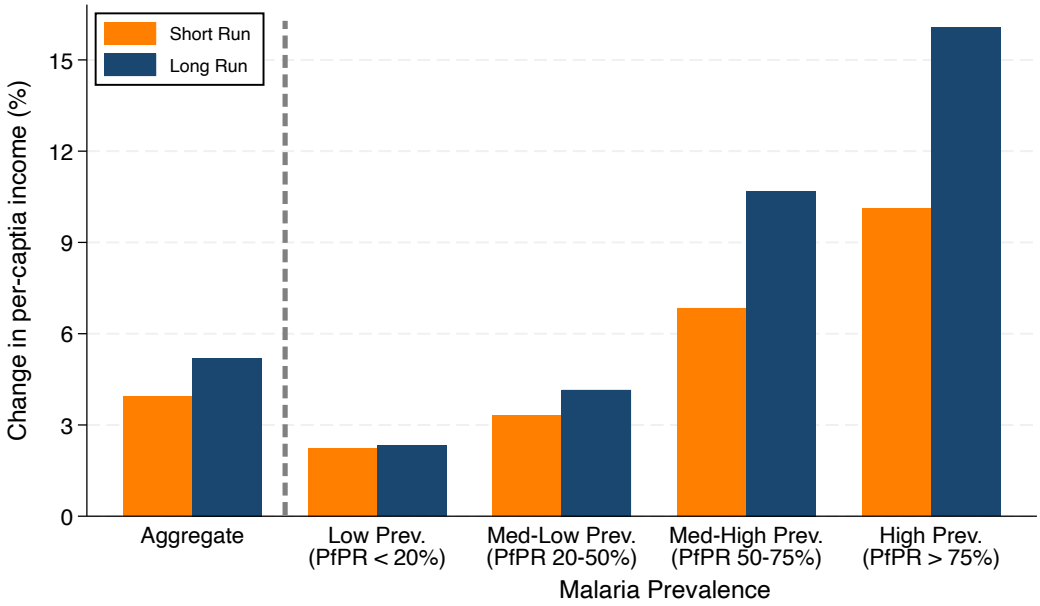
[26] According to Datoo, Natama, Somé, Bellamy, Traoré, Rouamba, Tahita, Ido, Yameogo, Valia et al. (2022), full vaccination requires four doses—three initial doses and a booster one year later.

[27] All figures are in 2015 constant dollar.

amounts to approximately 3.3% of per capita income. Figure 5 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.

As the figure illustrates, at the currently reported efficacy of the R21/Matrix-M vaccine, both the short- and long-run increases in per capita income clearly outweigh the cost of vaccination. The results indicate that vaccines with efficacy above 60% would be cost-effective in the short run—that is, the increase in per capita income exceeds the per-child cost of vaccination. In the long run, vaccines with efficacy above 50% would be cost-effective. Given that 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 presented here further implies that substantial macroeconomic benefits may be forgone due to delayed vaccine distribution.

Figure 6: Post-Vaccine Change in Per Capita Income Across Regions



Notes: This figure

Another practical consideration in vaccine distribution is determining which geographic areas to target. From a cost-effectiveness perspective, the model suggests that malaria vaccination should be recommended for children living in regions with moderate to high malaria prevalence. Figure 6 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 regions where malaria prevalence exceeds 20%—a threshold that covers 69% of the population in Tanzania. In the highest-prevalence regions, the long-run income gains reach up to 16%, implying substantial economic benefits of geographically targeted vaccination efforts.

6. Conclusion

High mortality and poor health conditions have long been viewed by policymakers as critical obstacles to economic development in poorer countries. Malaria, in particular, remains a major burden in sub-Saharan Africa, significantly impeding childhood human capital accumulation. In 2020 alone, malaria was responsible for over 600,000 deaths, mostly among children under the age of five in sub-Saharan Africa. Beyond mortality, malaria infection inflicts lasting cognitive impairments on surviving children, weakening educational outcomes and lowering labor productivity in adulthood. Given these consequences, understanding the economic impact of malaria eradication is crucial both for assessing the returns to health investments and for identifying the source of cross-country income differences.

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 accumulation through schooling, and malaria-induced health shocks. To quantify the macroeconomic impacts of malaria eradication, the calibrated model 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 bigger than previously estimated in the macroeconomic literature. The model predicts a 3.9% increase in per-capita income in the short run and a 5.2% increase in the long run. These gains primarily arise from improved learning capacity per year of schooling, as well as significant 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 if vaccine efficacy exceeds 60%, supporting the roll-out of current vaccines with reported efficacy around 75%.

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, I do not explicitly consider child labor and how changes in the malaria environment may interact with child labor practices. This omission is primarily due to the lack

of credible and detailed data on child labor markets in developing countries. The relationship between improved childhood health, educational outcomes, and child labor participation is not straightforward: healthier children are not only better students but also potentially more productive workers, complicating parental decisions on schooling versus labor (Bau, Rotemberg, Shah, and Steinberg, 2024). Subsequent research with improved data could further explore this avenue. Lastly, 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 poor countries, whose elimination could foster economic growth. A challenge in studying multiple diseases simultaneously arises from the fact that diseases often interact, and these interactions must be carefully accounted for. Future research could explore this avenue.

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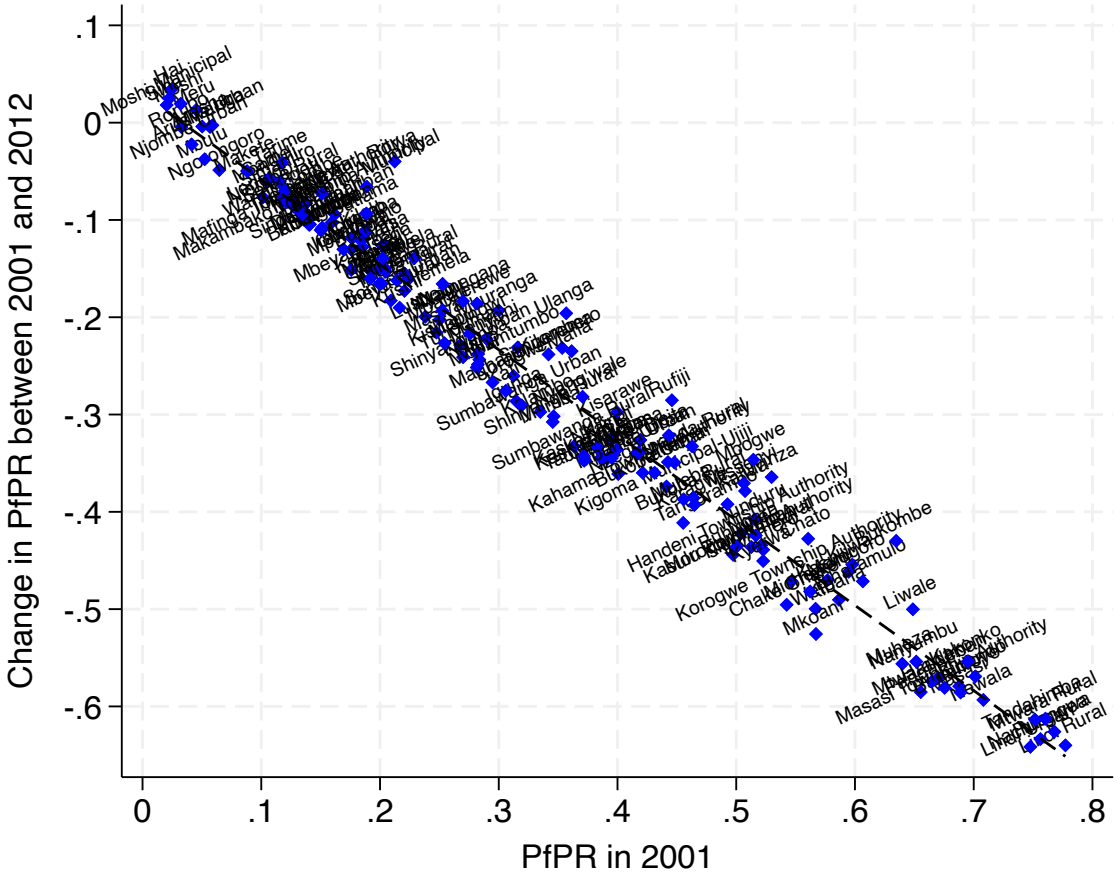
Appendix

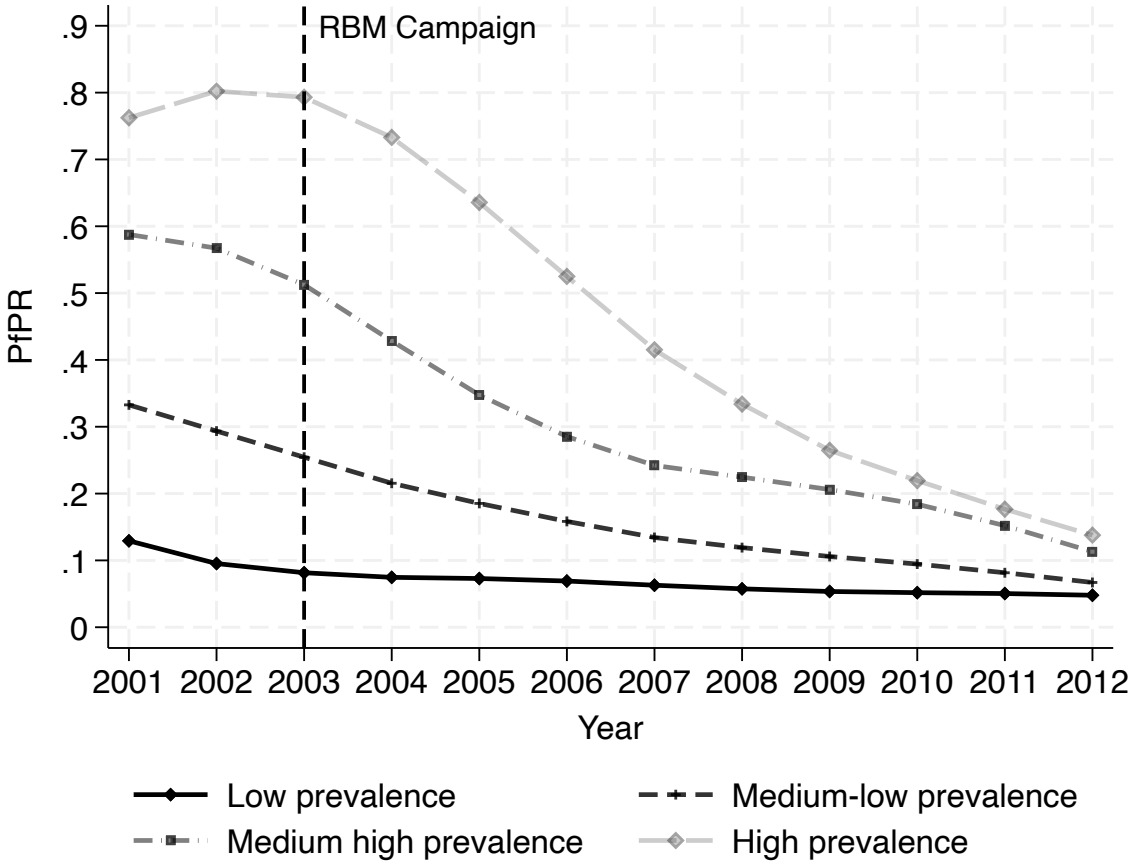
A. Appendix Figures

Figure A.1: Change in regional malaria risk conditional on malaria risk in 2001



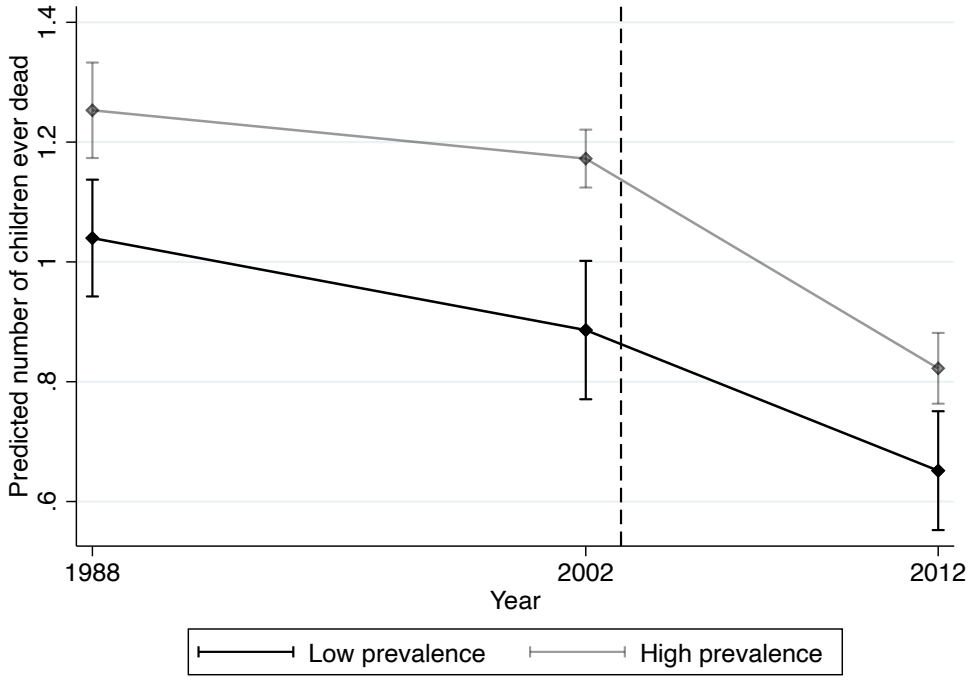
Notes: Each point represents a district at GIS-2 level. Yearly malaria risk (PfPR) is obtained from the Malaria Atlas Project (MAP). Fitted line is calculated by regressing the changes in malaria risk over 2001 and 2012 on initial malaria risk in 2001.

Figure A.2: Time trend of regional malaria prevalence based on the four pre-campaign malaria prevalence categories



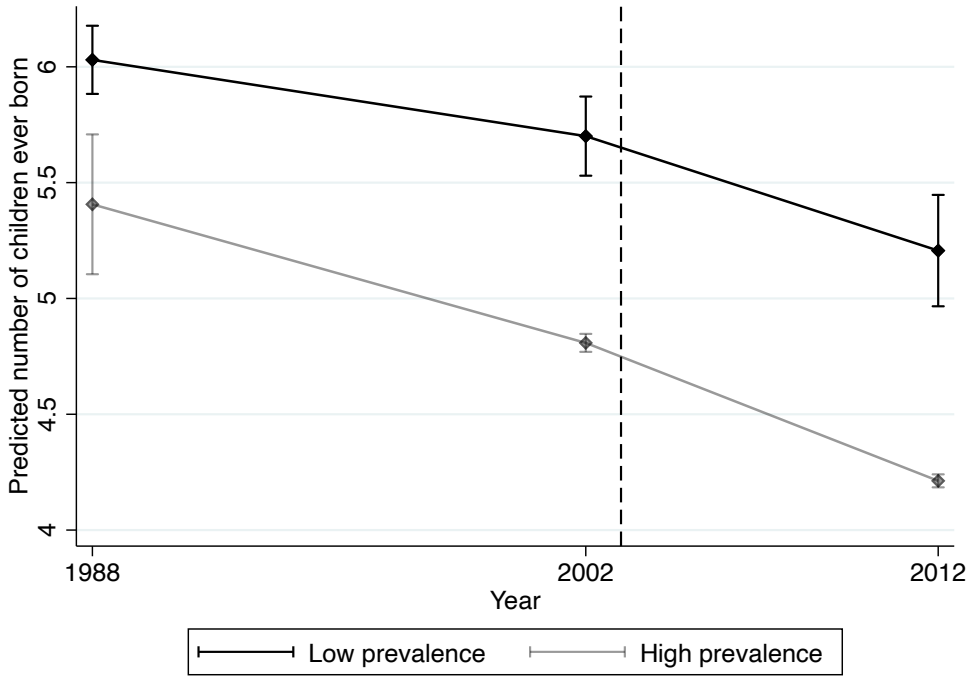
Notes: Each point represents a within-category population-weighted mean of PfPR. Regional malaria prevalence data obtained from the Malatia Atlas Project (MAP).

Figure A.3: Parallel trend in child mortality



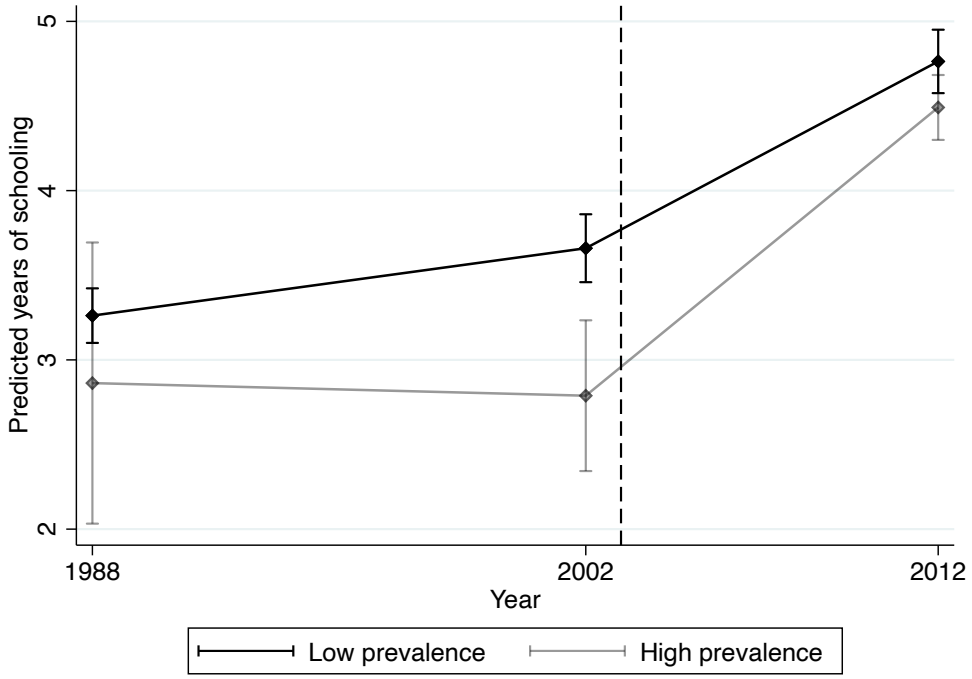
Notes: This figure illustrates the parallel trend between the high- and low- prevalence regions by plotting the mean of the number of children ever dead conditional on the covariates used in regression (5). Three waves of the Tanzania National Census (1988, 2002, 2012) are used. Samples are restricted to women between the ages 30 and 49 in 2012 and those who were born and residing (surveyed) in the same region in 2012. Control variables included are the respondents' age and years of schooling and urban-rural residential status. Standard errors are clustered at the region level. 95% confidence intervals are plotted. The vertical dashed line indicates the timing of the RBM campaign.

Figure A.4: Parallel trend in gross fertility



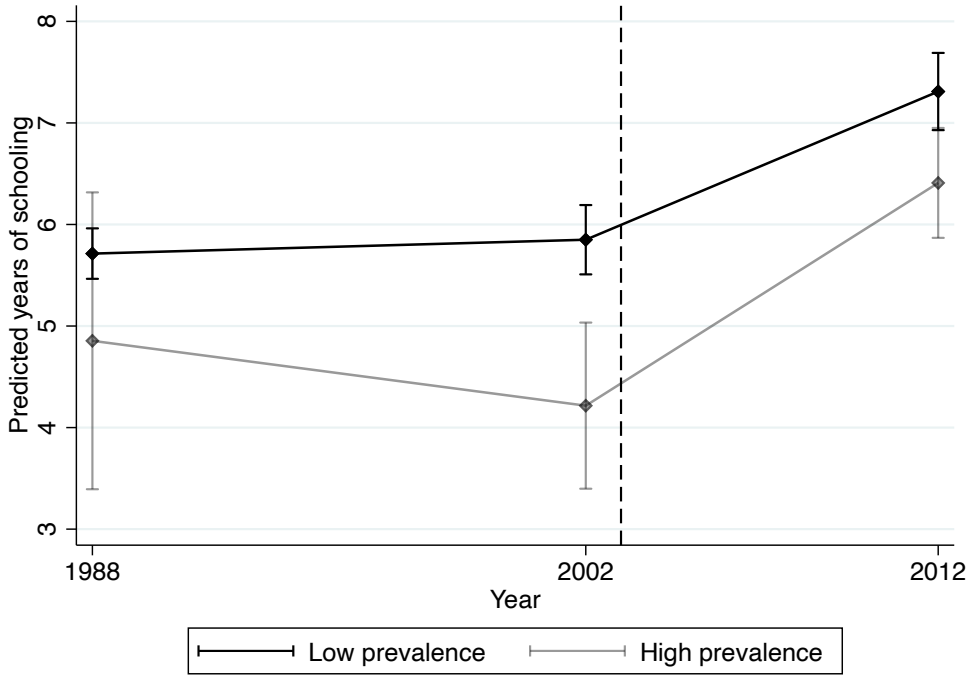
Notes: This figure illustrates the parallel trend between the high- and low- prevalence regions by plotting the mean of the number of children ever born conditional on the covariates used in regression (6). Three waves of the Tanzania National Census (1988, 2002, 2012) are used. Samples are restricted to women between the ages 30 and 49 in 2012 and those who were born and residing (surveyed) in the same region in 2012. Control variables included are the respondents' age and years of schooling and urban-rural residential status. Standard errors are clustered at the region level. 95% confidence intervals are plotted. The vertical dashed line indicates the timing of the RBM campaign.

Figure A.5: Parallel trend in children's years of schooling, children aged 10-15 in 2012



Notes: These figures illustrate the parallel trend between the high- and low- prevalence regions by plotting the mean of the number of years of schooling conditional on the covariates used in regression (7). Three waves of the Tanzania National Census (1988, 2002, 2012) are used. Samples are restricted to women between the ages 30 and 49 in 2012 and those who were born and residing (surveyed) in the same region in 2012. Control variables included are the respondents' age and urban-rural residential status. Standard errors are clustered at the region level. 95% confidence intervals are plotted. The vertical dashed line indicates the timing of the RBM campaign.

Figure A.6: Parallel trend in children's years of schooling, children aged 15-20 in 2012



Notes: These figures illustrate the parallel trend between the high- and low- prevalence regions by plotting the mean of the number of years of schooling conditional on the covariates used in regression (7). Three waves of the Tanzania National Census (1988, 2002, 2012) are used. Samples are restricted to women between the ages 30 and 49 in 2012 and those who were born and residing (surveyed) in the same region in 2012. Control variables included are the respondents' age and urban-rural residential status. Standard errors are clustered at the region level. 95% confidence intervals are plotted. The vertical dashed line indicates the timing of the RBM campaign.

B. Appendix Tables

Table B1: Descriptive Statistics

	Entire Sample	Low Prevalence Regions	High Prevalence Regions
Age	37.44	37.51	37.82
	(5.626)	(5.625)	(5.643)
Number of children ever born	5.465	5.319	4.567
	(3.202)	(3.026)	(2.887)
Number of children dead	0.978	0.825	1.215
	(1.380)	(1.294)	(1.519)
Years of schooling	4.427	4.772	3.922
	(3.509)	(3.428)	(3.388)
% Household has electricity	0.0752	0.0911	0.0234
	(0.264)	(0.288)	(0.151)
% Household has water supply	0.348	0.443	0.164
	(0.476)	(0.497)	(0.371)
PfPR in 2001	0.338	0.132	0.766
	(0.197)	(0.0534)	(0.00702)
Number of families in household	1.384	1.344	1.480
	(0.969)	(0.908)	(1.011)
Labor force participation	0.825	0.851	0.868
	(0.380)	(0.356)	(0.339)
Urban-rural status	0.364	0.355	0.434
	(0.481)	(0.479)	(0.496)
Observations	281,710	86,555	9,228

Notes: Calculated from 2002 Census data. Sample is restricted to women between age 30 and 49. Low-prevalence corresponds to the regions with PfPR lower than 10% in 2001, while high-prevalence corresponds to the regions with PfPR higher than 75% in 2001. Having water supply is defined as having access to piped water either within or outside the dwelling, including the public piped water. Mean coefficients; standard deviation in parentheses

Table B2: Effects of the RBM on Years of Schooling (Full Table)

	Age group in 2012			
	Age 10-15	**Age 15-20**	**Age 20-25**	**Age 25-30**
Dependent variable mean in 2012	4.29	6.91	6.86	6.06
Post	1.109***	1.400***	1.054***	0.398***
	(0.037)	(0.058)	(0.075)	(0.067)
$PfPR_{2-10}$ (20% − 50%) × Post	0.0196	0.0361	0.0371	-0.0868
	(0.055)	(0.081)	(0.100)	(0.091)
$PfPR_{2-10}$ (50% − 75%) × Post	0.0136	0.148	-0.0152	-0.225*
	(0.084)	(0.111)	(0.125)	(0.121)
$PfPR_{2-10}$ (75%+) × Post	0.689***	1.003***	0.562***	-0.0460
	(0.096)	(0.131)	(0.107)	(0.119)
Age	0.712***	0.139***	-0.0620***	-0.0747***
	(0.008)	(0.007)	(0.003)	(0.004)
Urban	0.807***	1.477***	1.745***	1.708***
	(0.041)	(0.071)	(0.076)	(0.074)
Observations	1,258,221	1,043,818	875,946	812,544

Notes: This table reports the estimation restuls from OLS regression (7). Brackets contain standard errors clustered at the region level. $PfPR_{2-10}$ (75%+) × Post indicates the interaction between the indicator of high-prevalence regions (PfPR in 2001 exceeding 0.75) and the post-treatment indicator. Other interaction terms are defined similarily. Samples are restricted to the individuals who were born and residing (surveyed) in the same region in 2012. Variable Urban indicates whether the respondent reside in the urban part within the region. All columns include region fixed effects. *, **, and *** indicate significance at the 10, 5, 1% levels.

Table B3: Effects of the RBM on Fertility (Full Table)

Dependent variable	Gross Fertility Children ever born	Mortality Children ever dead	Net Fertility Surviving children
Dependent variable mean in 2012	4.97	0.69	4.51
Post	–0.107***	–0.321***	–0.0766***
	(0.00832)	(0.0144)	(0.00933)
$PfPR_{2-10}$ (20% − 50%) × Post	0.0184*	0.0298	0.0173
	(0.0104)	(0.0192)	(0.0110)
$PfPR_{2-10}$ (50% − 75%) × Post	0.00895	0.00572	0.0152
	(0.0121)	(0.0223)	(0.0123)
$PfPR_{2-10}$ (>75%) × Post	–0.0613***	-0.0941***	-0.0321**
	(0.00978)	(0.0305)	(0.0160)
Age	0.026***	0.045***	0.022***
	(0.000)	(0.001)	(0.000)
Years of schooling	–0.015***	–0.058***	–0.008***
	(0.001)	(0.002)	(0.001)
Urban	–0.160***	–0.231***	–0.131***
	(0.006)	(0.014)	(0.006)
Observations	663,898	633,683	633,567

Notes: This table reports the estimation results for the Poisson regression (5) and (6). $PfPR_{2-10}$ (75%+) × Post indicates the interaction between the indicator of high-prevalence regions (PfPR in 2001 exceeding 0.75) and the post-treatment indicator. Other interaction terms are defined similarily. Samples are restricted to women between age 30 and 49 in 2012, and those who were born and residing (surveyed) in the same region in 2012. Control variables included are age and years of schooling of the respondents and urban-rural residential status. Variable Urban indicates whether the respondent reside in the urban part within the region. All columns include region fixed effects. *, **, and *** indicate significance at the 10, 5, 1% levels.

Table B4: Malaria in Tanzania Among Children under age 10

Panel A: 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
Panel B: Top 5 illnesses that caused death (%)					
Malaria	**55.56**	**42.39**	**60.62**	**46.81**	**51.35**
Diarrhea	7.78	15.22	0.00	4.26	7.69
Vomiting	0.00	1.63	0.00	0.00	0.62
Flu	0.00	0.54	0.62	0.00	0.42
Asthma	2.22	1.09	0.62	0.00	1.04

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?" for hospitalization, and "What was the illness that caused [NAME]'s death?" for the death. Responses from the parents who were unsure of the cause of deaths are excluded.

C. Robustness of Empirical Findings

Figure C.1: Gross Fertility

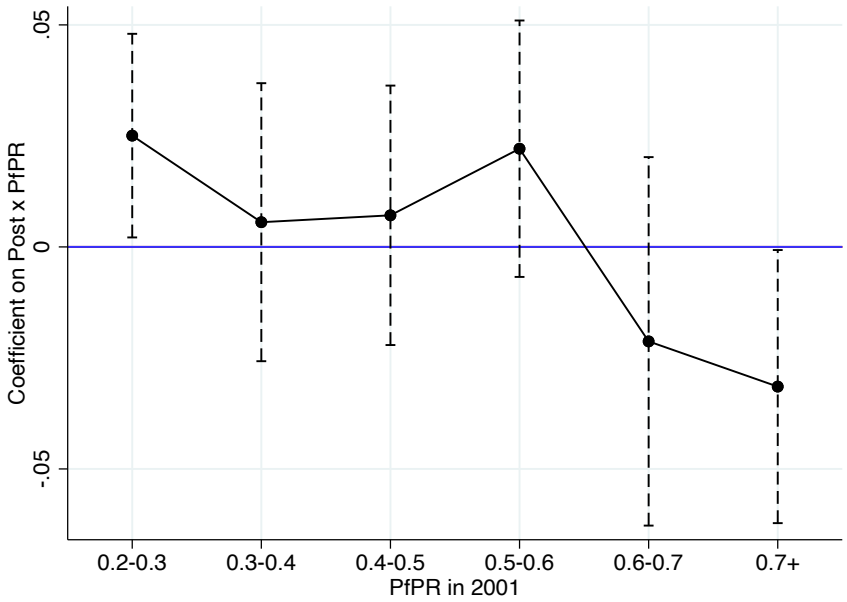


Figure C.2: Child Mortality

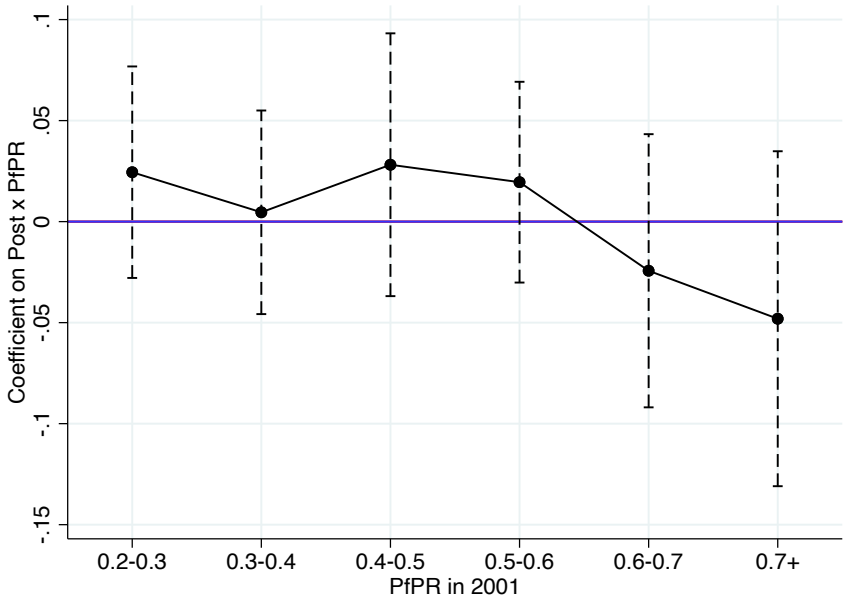


Figure C.3: Net Fertility

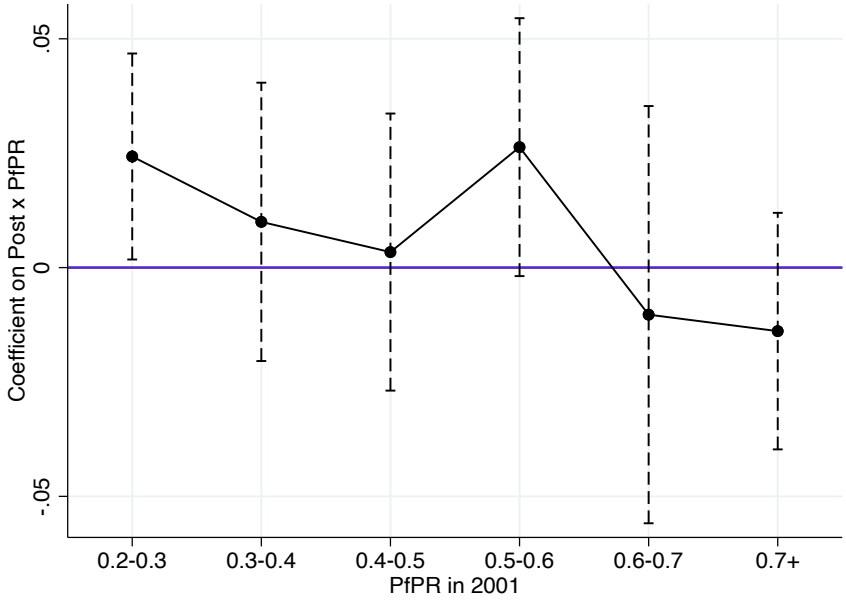


Figure C.4: Yrs of schooling 10-15

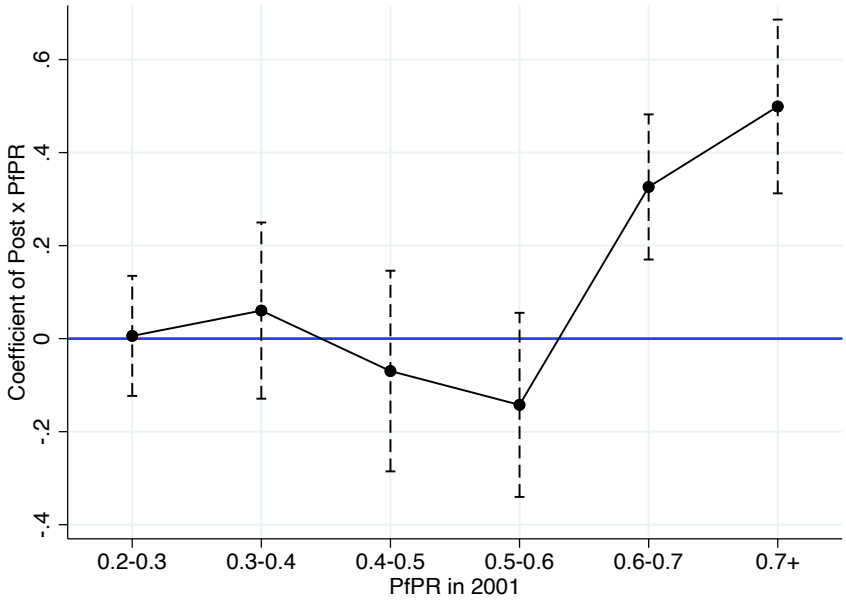


Figure C.5: Yrs of schooling 15-20

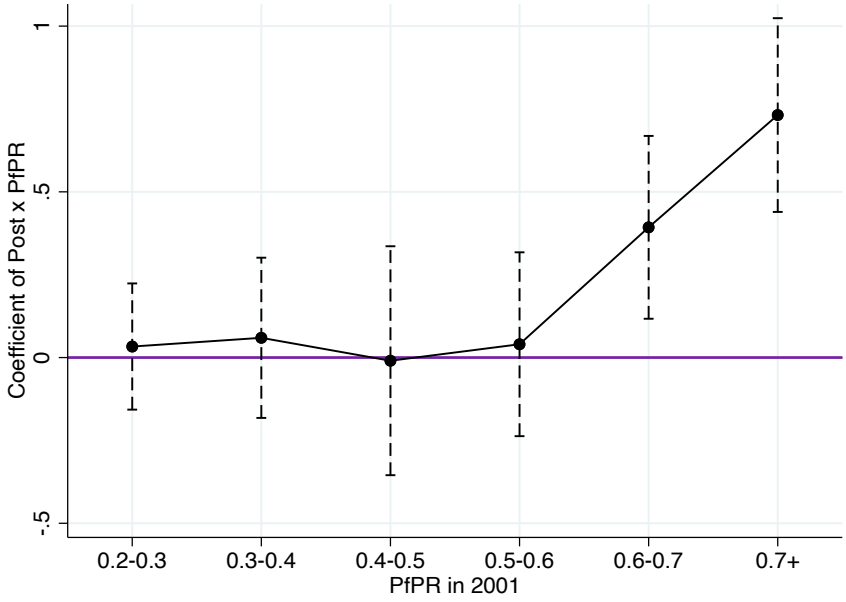
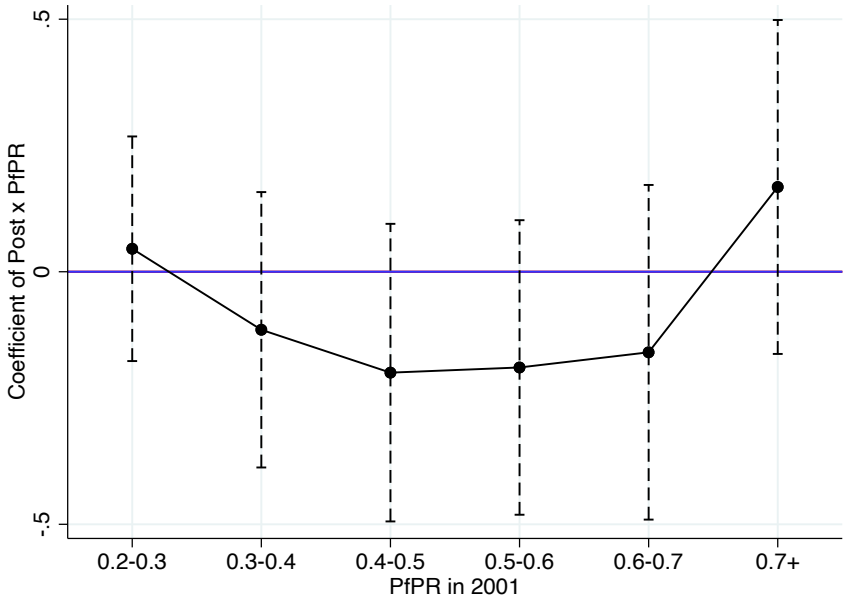


Figure C.6: Yrs of schooling 20-30



D. Additional Empirical Results

Table D1: Child Quantity Regression for Different Age Groups

	Age group of women in 2012			
	Age 30-39	Age 40-49	Age 50-59	Age 60-69
Panel A: Child Mortality				
Dependent variable: Number of children ever died				
Post	-0.399^{***}	-0.234^{***}	-0.233^{***}	-0.180^{***}
	(0.019)	(0.023)	(0.023)	(0.020)
PfPR_{2-10} (75%+) $\times$ Post	-0.160^{***}	-0.074	0.020	-0.025
	(0.047)	(0.064)	(0.038)	(0.063)
Panel B: Gross Fertility				
Dependent variable: Number of children ever born				
Post	-0.077^{***}	-0.137^{***}	-0.146^{***}	-0.054^{***}
	(0.008)	(0.010)	(0.012)	(0.013)
PfPR_{2-10} (75%+) $\times$ Post	-0.060^{***}	-0.053^{***}	0.004	0.007
	(0.009)	(0.014)	(0.018)	(0.028)
Panel C: Net Fertility				
Dependent variable: Number of surviving children				
Post	-0.034^{***}	-0.126^{***}	-0.137^{***}	-0.033^{**}
	(0.010)	(0.011)	(0.012)	(0.015)
PfPR_{2-10} (75%+) $\times$ Post	-0.007	-0.035^{**}	0.010	0.035^{*}
	(0.019)	(0.017)	(0.013)	(0.018)
Observations	355,644	231,192	133,687	90,455

Notes: This table reports the estimation results from Poisson regression (5) and (6) from different age groups for women. PfPR_{2-10} (75%+) $\times$ Post indicates the interaction between the indicator of high-prevalence regions (PfPR in 2001 exceeding 0.75) and the post-treatment indicator. Other interaction terms are defined similarily. Samples are restricted to women who were born and residing (surveyed) in the same region in 2012. Control variables included are age and years of schooling of the respondents and urban-rural residential status. Variable Urban indicates whether the respondent reside in the urban part within the region. All columns include region fixed effects. *, **, and *** indicate significance at the 10, 5, 1% levels.

Table D2: Heterogeneous Effects of the RBM on Years of Schooling by Gender

	Age group in 2012			
	Age 10-15	**Age 15-20**	**Age 20-25**	**Age 20-30**
Panel A: Male				
Dependent variable: Years of schooling				
Post	1.128***	1.358***	1.183***	0.404***
	(0.044)	(0.070)	(0.086)	(0.078)
$PfPR_{2-10}$ (75%+) × Post	0.561***	0.997***	0.639***	-0.006
	(0.091)	(0.107)	(0.120)	(0.102)
Observations	551,298	414,836	296,759	269,074
Panel B: Female				
Dependent variable: Years of schooling				
Post	1.161***	1.492***	0.914***	0.324***
	(0.043)	(0.073)	(0.089)	(0.085)
$PfPR_{2-10}$ (75%+) × Post	0.710***	0.947***	0.378***	-0.272
	(0.105)	(0.144)	(0.120)	(0.276)
Observations	544,976	441,917	377,984	338,902

Notes: This table reports the estimation restuls from OLS regression (7), run separately by male and female. Brackets contain standard errors clustered at the region level. $PfPR_{2-10}$ (75%+) × Post indicates the interaction between the indicator of high-prevalence regions (PfPR in 2001 exceeding 0.75) and the post-treatment indicator. Other interaction terms are defined similarily. Samples are restricted to the individuals who were born and residing (surveyed) in the same region in 2012. Variable Urban indicates whether the respondent reside in the urban part within the region. All columns include region fixed effects. *, **, and *** indicate significance at the 10, 5, 1% levels.