How Will a New Malaria Vaccine Shape Africa's Economic Future? A Macroeconomic Analysis

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Abstract

Malaria is the primary cause of death among children and a barrier to childhood human capital accumulation in sub-Saharan Africa. The macroeconomics literature thus far concludes that eradicating malaria would mainly increase populations but not substantially raise living standards. This paper reassesses this conclusion by modeling and quantifying the long-run macroeconomic effects of a successful malaria vaccine. To do so, I build a general-equilibrium, overlapping generations model of childhood human capital accumulation and endogenous fertility with malaria modeled as a health shock to children. To parameterize the model, I estimate the short-run effects of reduced malaria risk on women's fertility and children's human capital using difference-in-differences with a recent large-scale anti-malaria campaign in Tanzania. I use these estimates to calibrate the model's parameters and simulate the long-run general equilibrium impacts of malaria vaccines. The model suggests that a universal vaccination would increase per-capita GDP by 30% within 60 years, which is nearly ten times larger than previously estimated. The larger gains stem from higher human capital investments beyond simple increases in years of schooling, amplified over multiple generations.

JEL Classification: I15, I25, J11, J13, O11, O15, O20, O44, O55

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1. Introduction

Despite preventive technologies and treatment, malaria is still the leading cause of death and a barrier to children's human capital accumulation in sub-Saharan Africa. In 2020 alone, more than 600,000 people died from malaria, mostly African children under five years old (World Health Organization, 2021). Children who survive are also known to suffer from long-lasting cognitive impairments and co-morbidities (Fernando et al., 2010; Chen et al., 2016), which adversely affect their learning outcomes. In recent years, however, scientists have taken a major step towards eliminating malaria, as the newly developed vaccine is reported provide up to 80% protection against infections among young children (Datoo et al., 2022).

How will the new malaria vaccine change the macroeconomic outlook of sub-Saharan African countries? One view in the macroeconomics literature thus far is that eliminating malaria would mainly increase populations but not substantially raise living standards. For example, Acemoglu and Johnson (2007) study the effects of large improvements in life expectancy in the 1940s driven by international health interventions, more effective public health measures, and the introduction of new chemicals. The study concludes that the improvements in life expectancy had little impact on GDP per capita but only increased populations. Ashraf, Lester, and Weil (2008) corroborate this view, arguing that eradicating malaria in a typical sub-Saharan African country would increase GDP per capita only by two percent over a 60-year horizon.

This paper reassesses this conclusion by modeling and quantifying the long-run macroeconomic effects of a successful malaria vaccine and argues that the increase in long-run output per capita from eliminating malaria is much larger than the existing estimates. I focus on several new features that have been absent from previous macroeconomic studies of disease eradication. The first is a quantity-quality tradeoff parents face when making fertility and investment decisions in children. The second is a richer measurement of human capital than just years of schooling, as in Manuelli and Seshadri (2014). Using years of schooling as a measure of human capital implicitly assumes that one year of schooling delivers the same increase in human capital before and after eliminating malaria (Hanushek and Woessmann, 2008). However, eliminating malaria also allows children to learn more from schooling, increasing the amount of human capital gained per year of schooling. The third is the intergenerational dynamics, as healthier children may subsequently adjust their fertility and invest more in their own children's education.

I incorporate these features into a general equilibrium, heterogeneous-agent, overlapping generations model which allows for the interplay of fertility, childhood human capital accumulation, and childhood diseases. In the model, parents endogenously choose how many children

to have as well as the educational attainment of their children. Children are born with exogenously heterogeneous learning abilities inherited stochastically from their parents. Parents base their fertility decision on assets, income, and tastes for children. Parents also make a decision on whether to educate their children or send them to work by comparing the higher income and consumption today from child labor against the higher future utility their children will enjoy if they receive more education. Children's subsequent outcomes are then determined by their learning ability, the educational choice made by parents, and the skill-contingent wages determined in the competitive labor market.

I then introduce childhood disease into the model as an idiosyncratic health shock to children. Health shock in the model consists of two dimensions; mortality and morbidity. Mortality reflects the deaths caused by diseases, and morbidity represents the long-term cognitive damage on children, which dampens their human capital accumulation. A key feature of the model is that childhood disease interacts with parents' education and fertility decisions through the quantity-quality tradeoff (Barro and Becker, 1989). Within the quantity-quality framework, eliminating a disease has two opposing effects. On the one hand, lower mortality from the elimination lowers the price of child *quantity*, inducing parents to have more children and reducing per-child educational investment. On the other hand, reduced morbidity lowers the price of child *quality*, inducing parents to have fewer children and educate each child more. In the model, these two effects are summarized by two sufficient statistics which govern the long-run effects of disease elimination: fertility and education elasticities of disease.

To estimate the size of the fertility and education elasticities of malaria, I exploit a recent large-scale antimalarial campaign in sub-Saharan Africa, the Roll Back Malaria (RBM) campaign. Starting from 2003, the RBM campaign significantly reduced malaria prevalence through its aggressive distribution of preventive and treatment technologies, such as insecticide-treated nets (ITNs) and indoor residual spraying (IRS). I focus on Tanzania, one of the few countries where the campaign started at its earliest phase. Exploiting regional variation in the campaign's intensity, I estimate the effects of the reduced malaria risk on women's fertility and children's years of schooling through a difference-in-differences design. Consistent with the previous studies, I find that the reduced prevalence of malaria due to the campaign led to an average of 0.63 more years of schooling for the benefited children.[1] I also find that women in the highest malaria prevalence regions reduced their fertility by 5.9%. However, although fewer children were born, the campaign did not change the number of surviving children because

[1]For empirical studies found positive effects of reduced malaria risk on children's educational attainment, see Lucas (2010); Venkataramani (2012); Barofsky et al. (2015); Shih and Lin (2018); Kuecken et al. (2021). For studies focusing on adulthood outcomes such as income or consumption, see Cutler et al. (2010); Bleakley (2010). Unlike this paper, most of these studies used historical episodes of malaria eradication.

the campaign also reduced child mortality by 10%.

I quantitatively solve for the balanced growth path and bring the estimated elasticities to the model by replicating the Roll Back Malaria campaign within the model and matching the campaign's simulated causal effects to their empirical counterparts; a 0.63 year increase in children's schooling and a muted response of fertility. To ensure the model is credible in other dimensions, I jointly estimate parameters to match the empirical elasticities and other relevant aggregate moments of the Tanzanian economy, such as educational attainment and intergenerational mobility measures. Since the causal effects of reduced malaria risk on children's education outcomes and women's fertility are also estimated in Tanzania, all the moments used for calibration are from a single country.

Using the estimated model, I then simulate the long-run general equilibrium effects of a national malaria vaccine policy by solving for a new balanced growth path of the economy under the vaccination policy. The model predicts per capita income would rise by 34% within 60 years. Compared to the short-run, one-generational effects of policy, the long-run increase in per capita output is twice as large as the short-run counterpart, highlighting the importance of the intergenerational amplification channel. While the lowered mortality rate from vaccination is expected to put upward pressure on the population, the population growth rate on the post-vaccine balanced growth path is only modestly higher because households choose to have fewer children in the long run. The model also predicts a significant increase in both primary and secondary school completion rates and an improvement in intergenerational mobility in terms of education. While only 40% and 3% of the children born to uneducated parents complete primary and secondary education before the vaccination, the numbers increase to 52% and 5%, respectively, in the post-vaccination balanced growth path.

The results are surprising because they are nearly ten times larger than the literature's current best estimates from the influential work by Acemoglu and Johnson (2007) and Ashraf, Lester, and Weil (2008). Acemoglu and Johnson (2007) estimate the effects of life expectancy on economic growth, using the major international health improvement in the 1940s (the international epidemiological transition) as a natural experiment. They conclude that while improvement in life expectancy leads to an increase in population, it does not lead to an increase in GDP per capita. A key difference between the international epidemiological transition in the 1940s and malaria vaccines is that while the former mostly lowered mortality without increasing the returns to education, the latter would lower the mortality *and* raise the returns to education. I simulate a counterfactual long-run balanced growth path where only the mortality is lowered. Consistent with Acemoglu and Johnson (2007), the long-run increase in per capita output shrinks from 34% to a mere 2%. The result suggests that the long-run growth effects

of a health improvement hinge on whether it facilitates the accumulation of human capital.

Ashraf, Lester, and Weil (2008) specifically focus on malaria in sub-Saharan Africa. Using a standard neoclassical framework, they simulate the effects of eradicating malaria in a typical sub-Saharan African country and conclude that it would raise GDP per capita only by two percent in the long run. The underlying reasons for these different results are the difference in the measures of human capital and the omission of intergenerational dynamics. First, Ashraf et al. (2008) treat malaria eradication as a one-time increase in human capital, rather than considering intergenerational dynamics that amplify the effects in the long run. Second, following contemporary best practice, they employ years of schooling as a measure of human capital. An implicit assumption behind this is that a year of schooling delivers the same increase in human capital before and after eradication (Hanushek and Woessmann, 2008). However, eradicating malaria would also make children healthier and perform better *within* school (Fernando et al., 2010), an important margin that years of schooling cannot address. In the model, the increase in years of schooling only explains 30% of the increase in human capital for the children born after the vaccination, implying that focusing only on years of schooling could underestimate the increase in human capital. In Section 5.2.2, I explore the quantitative implications of omitting the intergenerational dynamics and using years of schooling as the human capital measure and find that without the two channels, the long-run increase in output per capita is reduced from 34% to 5%, much closer to the numbers found in Ashraf et al. (2008).

I conclude that eliminating malaria is not only a life-saving policy, but also a growth policy for sub-Saharan African countries. The estimated model implies that once the long-run intergenerational effects and the better learning in school are considered, the long-run increase in output per capita can be much larger than what is estimated in the literature thus far. This suggests that removing malaria should be a high priority in development strategy for policymakers. The results also imply that improving children's health conditions in the developing world can raise the living standard in the long-run, primarily through the higher human capital accumulation of children. Such implication is consistent with the conclusions of the macro-development literature, which have been emphasizing the role of human capital in explaining the cross-country income differences (Erosa, Koreshkova, and Restuccia, 2010; Schoellman, 2012; Hendricks and Schoellman, 2018; De Philippis and Rossi, 2021).

While this paper specifically focuses on malaria, the model can easily be generalized to other childhood infectious diseases and used as a framework to study the effects of improving health on long-run economic growth. In this vein, this paper builds on a long literature that studied the relationship between health, human capital, and economic growth (Shastry and Weil, 2003; Caselli, 2005). Weil (2007) shows that eliminating health differences across countries

has minor effects in narrowing the cross-country gaps in output per capita, while the quantitative results in this paper suggest that such effects can be potentially larger. The theoretical framework in this paper is related to those in Kalemli-Ozcan, Ryder, and Weil (2000); Kalemli-Ozcan (2003); Soares (2005) and Doepke (2005), all of whom focused on the role of mortality decline in human capital investment and growth. Lastly, the quantitative model of this paper is perhaps most closely related to those in Daruich (2020) and Zhou (2021), who study the long-run effects of education subsidy and family policies, respectively, using a general equilibrium, heterogeneous-agent overlapping generations model. However, none of these previous studies considers diseases as a factor disrupting childhood human capital accumulation, which the model suggests to be quantitatively important.

This paper also builds on a growing body of research in macroeconomic development that uses dynamic general equilibrium models to understand the potential long-run general equilibrium effects of development policies, using the short-run, partial equilibrium empirical evidence as ingredients. In this vein, the quantitative exercises of this paper are related to those of Fried and Lagakos (2022), who use a dynamic general equilibrium model to quantify the effects of power outages on productivity in developing countries. Similarly, Buera, Kaboski, and Shin (2021a) use a dynamic macroeconomic model of credit-constrained firms to study the general equilibrium effects of microfinance, using the partial equilibrium estimates of microfinance to discipline the model. Another example is Lagakos, Mobarak, and Waugh (2019), who quantify the aggregate effects of rural-to-urban migration subsidies compared to the partial equilibrium effects estimated from an RCT. Brooks and Donovan (2020) also use the reduced-form evidence on the effects of rural bridge building to guide the general equilibrium effects of transportation infrastructure.[2]

The rest of the paper is organized as follows. Section 2 lays out the quantitative model. Section 3 describes the data used to estimate key parameters and the estimation of empirical moments used to discipline the rest of the model parameters. Section 4 explains the calibration strategy and model validation results. Section 5 investigates the long-run general equilibrium effects of a national malaria vaccination policy and compares the results to the previous estimates. Section 6 concludes.

[2]See Buera, Kaboski, and Townsend (2021b) for a detailed review of the literature.

2. Model

In this section, I introduce a general equilibrium overlapping generations model with endogenous population dynamics, childhood human capital investment, and health risk. A household consists of parents and cohabiting children. Parents endogenously choose how many children to have as well as the educational level of their children, who are born with exogenously heterogeneous learning abilities. Children's endowment when they become adult are determined by their learning ability, the educational choice made by parents. Workers with different levels of education are imperfect substitutes, and their education-contingent wages are determined in a competitive labor market.

2.1. Environment

Demographics Time is discrete, and one model period is six years. The economy is populated by a large number of overlapping generations of households who live for 66 years (12 periods in total). Figure 1 shows the life cycle and family structure of a household. I use $j \in \{0, 1, \cdots, 12\}$ to denote the period of life.

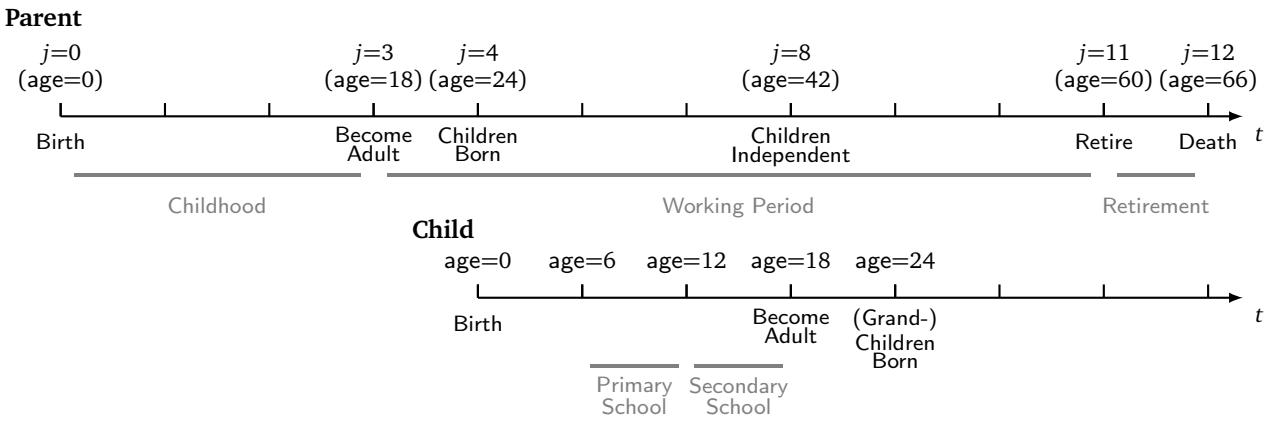


Figure 1: Life cycle, family structure, and stages of life

Children live with their parents and do not make any decisions on their own until they reach age 18, when they leave their parents and become independent with zero assets. Throughout their adulthood, individuals choose consumption expenditure and savings. Borrowing is not allowed but they can save through assets with exogenous interest rate r.[3] parents choose how

[3] I abstract from domestic capital market to focus on the main mechanism of the model. Most countries in this context are small open economy with under-developed capital market. I also omit the endogenous labor supply retirement choice, due to high hours worked and short retirement periods in the developing worlds.

many children to have at age 24, and conditional on having children, how much education their children will receive until they become adults. There are two periods of schooling; parents have to decide whether or not to send children to primary school when the children are six years old, and secondary school when they are twelve years old. The initial human capital their children possess is therefore directly influenced by the parents' educational choice. Once the children become independent, there is no further interaction between parents and children and the human capital is fixed throughout the adulthood. All individuals retire at age 60 and die at age 66. During the short retirement periods, individuals live off the assets they have accumulated in the working period.

There are four exogenous sources of heterogeneity in the model. The first one is the standard, idiosyncratic and uninsurable labor productivity shock ν_t for the working-age adults, which makes earnings stochastic. I assume that the idiosyncratic labor productivity shock is i.i.d. and drawn every period from a log-normal distribution:

$$\log \nu_t \overset{\text{iid}}{\sim} N(0, \sigma_\nu)$$

The second one is the fertility taste, which captures the fertility behaviors not attributed to the model's mechanism. I assume an extreme-value distribution-. The third one is the learning ability that every child is born with, which is imperfectly correlated between parents and children. The last one is the health shock that children face, which represent the childhood diseases, or malaria in our context. Health shock is drawn once at the early childhood when the children are six years old, and lowers the returns from schooling in subsequent childhood periods. I illustrate the way the ability and health shocks are drawn and how they interact with the parents' education decision in the next paragraphs.

Learning Ability Parents observe their children's learning ability at the beginning of the period $j = 5$, before they decide whether or not to send their children to primary school. The learning ability within a household follows a AR(1) process:

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

where z_k and z_p are the learning ability of children and parents and ε_z is the idiosyncratic i.i.d. shock. Therefore, learning ability is inherited across generations but only imperfectly.

Diseases in Early Childhood In addition to the learning abilities, children also draw a idiosyncratic health shock at age six.[4] The health shock has two dimensions: mortality and morbidity. Mortality reflects the deaths caused by the diseases, and morbidity represents the detrimental effects of the diseases on human capital accumulation. Mortality risk is manifested as a survival probability. Specifically, the probability an age-6 child survives to the next period is given as χ^d. Morbidity risk is represented by a proportional reduction in the learning ability, reflecting that children with malaria suffer from worse learning outcomes during and after bouts of malaria (Fernando et al., 2010). I discuss how the lowered learning ability affects children's human capital accumulation in the next paragraph. Specifically, I denote the morbidity shock as m:

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

where $\underline{m} < 1$. Here, $\underline{m}$ denotes the health status of a child hit by the adverse morbidity shock, while the status of a healthy child is normalized to one. The probability that a child is stricken with a negative morbidity shock is given as χ^m.

Schooling After observing their children's learning ability z_k and the health shock m they have drawn, parents decide whether to send their age-6 children to the primary school. Schooling increases human capital deterministically:

$$h_{k,t+1} = \begin{cases} \max\{mz_k\eta_s h_{k,t}, h_k\} & \text{if attend school} \\ h_k & \text{otherwise} \end{cases}$$

where η_s is the deterministic increase in human capital from school s, $s \in \{\text{Primary}, \text{Secondary}\}$. Sending children to school costs money, which are represented by the per-child schooling fee p_P, p_S for primary and secondary school, respectively. The schooling fee encompasses the tuition, uniforms, schooling supplies such as textbooks, etc., representing the goods cost of education. Children can work instead of going to school (child labor). Lastly, schooling decision is sequential; if parents do not send their primary school aged child to school and have them work instead, the child does not have the opportunity to attend the secondary school in the next period.

[4] The assumption that the health shock is realized at age 6 can be understood as parents learn about their children's health status by the time they enter primary school.

Production and Aggregation I assume that there is a profit-maximizing representative firm in the labor market. The representative firm uses skilled (secondary education completed) and unskilled (below secondary education) labor to produce the single consumption good with the following CES aggregate production function:

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

Here, H_s denotes the aggregate efficiency unit of schooling groups U (uneducated), P (primary-completed) and S (secondary-completed), and λ is the elasticity of substitution between the two skill groups. Equilibrium wages for each skill group are then determined as:

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

Since the wages w_U and w_S are per efficiency unit, the labor income of an individual with skill level s, human capital h, and idiosyncratic shock v is given as $y(h,v,s) = hvw_s$.

2.2. Recursive Formulation of Decision Problems

From independence, an individual's adulthood can be broadly divided into two stages depending on whether they are alone or live with their children. In both stages, individuals solve consumption-savings optimization problem. When with children, however, they make additional decisions on fertility and children's education. In this subsection, I explain the individual optimization problem in each period of life. Borrowing is limited in all periods ($a' \geq 0$), and I suppress the expression in following formulations to simplify the notations. Throughout this section, I will denote all child variables with subscript k, and future variables with primes.

Age 18 ($j = 3$)**: Independence** Individuals leave their parents and form a new household at age 18 with zero assets. Their initial states are human capital h, schooling level s which is determined in the childhood, and the learning ability z. While the learning ability is not relevant for themselves, it is still included as a state variable because their own children's learning abilities will depend on it. Their income is determined by their human capital h, wage rate w_s, and the idiosyncratic income shock v drawn at the beginning of the period.

Since they do not have children yet, they solve a standard consumption-savings problem:

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

where r is the period interest rate.

Age 24 ($j = 4$)**: Fertility** At this stage, individuals decide how many children to have, and those who choose to have children become parents. Fertility choice is discrete; individuals choose the number of children n, where $n \in \{0,1,2,\cdots,\bar{N}\}$, by choosing n^* that gives them 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 functions 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 haveing n children can be written as follows:

$$\begin{aligned} V_4^n(a,s,h,z,v) = \max_{c,a'} & u(c) + \beta \mathbb{E}\Big[V_5\big(a',s,h,v',z_k',m',n'\big) \Big] \\ & \text{subject to} \\ & c + a' \leq w_s h v (1 - t(n)) + (1+r)a \\ & t(n) = \omega_1 n^{\omega_2} \end{aligned} \tag{2}$$

where $\boldsymbol{\phi}$ is the vector of fertility taste shock draws. Raising children is costly. Specifically, $t(n)$ amount of parental time is taken away, reducing the available income for consumption and savings. The total time cost is increasing in h and n, reflecting the fact that the sacrificed working hour is more costly to high-income households than low-income ones. Lastly, note that the expectation is taken over the number of *surviving* children in the next period, n'. This is due to the realization of the health shock in the next period.

Age 30 ($j = 5$)**: Ability, Health Shock and Primary Education** At the beginning of this period, parents observe children's ability z_k and the realization of health shock m, and draw the idiosyncratic income shock v. The number of surviving children n is determined depending

on how many children are hit by the mortality shock. Specifically, the probability that 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 to send their children (if any) to primary school ($e=1$) or workplace ($e=0$). If children attend school, a per-child primary schooling fee p_P is deducted from the parents' budget constraint. If the children go to workplace instead (child labor), their income is added to the parents' budget. Since the working children did not receive any education, their have one (initial) level of human capital and receive unskilled wages.[5] A parent's value function with n number of surviving children at this stage is written as follows:

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

Age 36 ($j=6$): Secondary Education and Dynastic Altruism Parents who had sent their children to the primary school in the previous period decide whether to continue sending children to secondary school. Because of the sequential nature of the schooling system, parents who did not send their children to the primary school do not have option to send them to secondary school ($e=0$ for them). The value function of the parents with secondary school age children is:

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

Note that the value function at this stage now includes the continuation value of the children, $V_{4,k}$, discounted by the altruism function $b(n)$. Since the problem is written recursively, this continuation value captures the parental altruism toward their children; they take into account the utility value of all of their descendants and make decisions accordingly.

[5] I assume that the children's income is also subject to the same idiosyncratic income shock v that their parents have drawn; the income shock is common across the household members.

Age 42 to 66 ($j = 7-12$): Mature Parents with Grown-up Children At the beginning of the period 7, children become independent and leave their parents. Once the children become independent, there is no further interaction between parents and children and parents solve a simple consumption-savings problem. Value of working-age individuals after the child-rearing periods is the same as (1). At age 60, households retire and are assumed to provide no work. The value function after retirement is given as:

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

2.3. Competitive Equilibrium and Balanced Growth Path

In this economy, population is endogenous due to the endogenous fertility. Therefore, I focus on a balanced growth path of the economy where the population growth rate remains constant over time, as does the distribution of households' state variables. I introduce the concepts of the recursive competitive equilibrium and the balanced growth path below.

Recursive Competitive Equilibrium To save notations, define the vector of age-j individual's state variables $(a, s, v, s_k, h_k, z_k, m, n)$ as $\mathbf{X}_j$ and the distribution of the age-j state variables as $\mu(\mathbf{X}_j)$. 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) Prices for each skill group w_U and w_S

such that

(i) V, a', c, n_4, e_5, e_6 solve the individual's optimization problem conditional on w_U and w_S

(ii) The representative firm maximizes its profit:

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

(iii) Prices clear the labor market

Balanced Growth Path A balanced growth path is a particular case of recursive competitive equilibrium which satisfies further conditions and is defined below. Let P the aggregate population. A balanced growth path is a recursive competitive equilibrium that satisfies:

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

(b) The distribution of households is stationary: $\mu'(\mathbf{X}_j) = \mu(\mathbf{X}_j)\ \forall j$

(c) Decision rules (a) are stationary and do not depend on P

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

The model has a rich set of interactions between disease, fertility, and children's human capital, which is encapsulated by the quantity-quality tradeoff. Within this framework, eliminating a disease has two opposing effects. On the one hand, lower mortality from the elimination lowers the price of child *quantity*, inducing parents to have more children and reducing per-child educational investment. On the other hand, reduced morbidity lowers the price of child *quality*, inducing parents to have fewer children and educate each child more. In the model, these two effects can be summarized by sufficient statistics: fertility and education elasticities of disease. Since these two elasticities govern how parents adjust their fertility and education decisions in response to a better health environment, the sizes of these elasticities are essential to understand the long-run effects of disease elimination.

To ensure that the model's implied elasticities are indeed credible, I empirically estimate the elasticities and ask the model to replicate them. To this end, I use the Roll Back Malaria campaign, a recent large-scale anti-malaria campaign that took place in many sub-Saharan African countries. I focus on one country, Tanzania, and employ a difference-in-differences design to identify the causal effect of the reduction of malaria burden on fertility and children's human capital, exploiting the spatial variations in pre-campaign malaria prevalence as the identifying variations.

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

It's worth looking at the malaria situation in Tanzania and how the campaign was implemented. Before the Roll Back Malaria (RBM henceforth) campaign started, more than 90% of Tanzania's population was at risk of malaria, putting the nation in a high malaria burden category. Malaria was also a huge contributor to childhood deaths in Tanzania; there were, on average, around 11 million clinical malaria cases per year prior to the campaign (National Malaria Control

Programme, 2010), contributing to about 36% of all deaths in Tanzania in children under five. Despite such a high burden, little effort was taken to reduce malaria transmission. Prior to 2003, the coverage of insecticide-treated nets (ITNs) was nearly zero everywhere in the country.

The Roll Back Malaria Partnership was launched jointly by the WHO, the World Bank, and the United Nations in 1998, aiming to halve the malaria burden between 2000 and 2010. A major difference between the RBM from previous anti-malarial movements was its unprecedented level of external funding - approximately $4.6 billion between 2003–2009. Between 2003 and 2009, 81 of the 108 malaria-endemic received financial support from the global community for their malaria-control work (Johansson et al., 2010). As sub-Saharan Africa accounted for around 85% of the global malaria burden, a large fraction of the financial aid was concentrated in Africa. The strategy the RBM adopted was massive distribution of insecticide-treated nets (ITN) and indoor residual spraying (IRS), both proven methods of reducing malaria transmission. With coordinated actions across countries for a decade, worldwide malaria death were cut in half in 2014. Among the recipient countries, Tanzania provides an ideal setting to study the effects of the RBM campaign as a representative sub-Saharan African country with high malaria burden. It is one of the twelve malaria-endemic countries which received financial support from as early as 2003 (Johansson et al., 2010), and where the RBM campaign was highly successful in reducing the disease burden.

The financial support from the RBM enabled the Tanzanian government to scale up the malaria interventions to the entire country. For example, ITNs started to be distributed to the most vulnerable groups in 2004, and free long-lasting insecticidal nets (LLINs) were delivered to children under five years old starting in 2009. Indoor Residual Spraying (IRS) was introduced to epidemic-prone areas in 2009 (National Malaria Control Programme, 2010). As a result of the coordinated efforts, Tanzania's malaria prevalence had been reduced significantly by 2012, about a decade after the onset of the RBM campaign. Figure 2 shows the reduction in malaria prevalence during this period. Malaria prevalence is measured by PfPR_{2-10}, which represents the proportion of children between the age of 2 and 10 who are found to carry *P.falciparum* parasites in their blood. PfPR_{2-10} is a commonly used index to measure malaria prevalence and its transmission intensity, which I continue to use as a proxy of malaria prevalence in the subsequent sections.

Tanzania is an exemplary country where the RBM significantly reduced the malaria burden, and available microdata also allows us to estimate the effects of the campaign on fertility and child human capital outcomes. The main dataset I am leveraging is the Population Census, which has three waves: 1988, 2002, and 2012. Unlike the commonly used Demographic and

Health Survey (DHS) data, the Census data provides a broader range of variables and hence enables us to better identify the effects of the RBM on our outcome variables of interest. In the next subsection, I explain the dataset's structure and advantages over other commonly used datasets.

3.2. Data

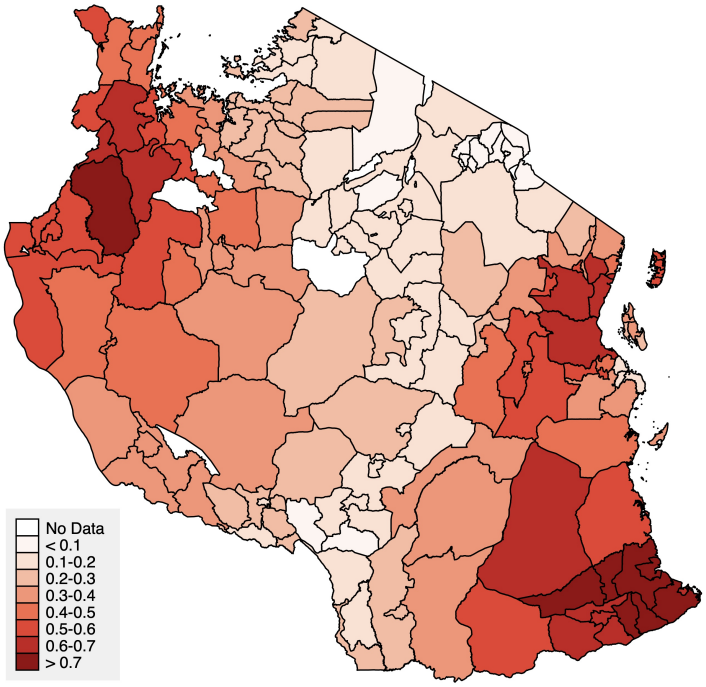
There are two main datasets I use for the empirical analysis. First, the information on malaria prevalence is derived from the Malaria Atlas Project (MAP). Second, household and individual level information on socioeconomic characteristics, mortality, fertility, and parental investment in children's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

Malaria Atlas Project (MAP) I obtain information on malaria prevalence prior to the intervention from the Malaria Atlas Project (MAP). The MAP provides annual estimates of malaria prevalence for a number of sub-Saharan African countries. Within each country, regional estimates are up to second-level administrative units (corresponding to the GIS-2 level). Using this data, I can recover the spatial distribution of malaria risk before and after the RBM campaign. I use the region-level average of the PfPR as a measure of malaria prevalence. Figure A.1 shows the changes in PfPR from 2001 (pre-campaign) and 2012 (post-campaign). As shown in the figure, regions with high malaria prevalence in 2001 experienced a larger reduction in malaria burden after the campaign.

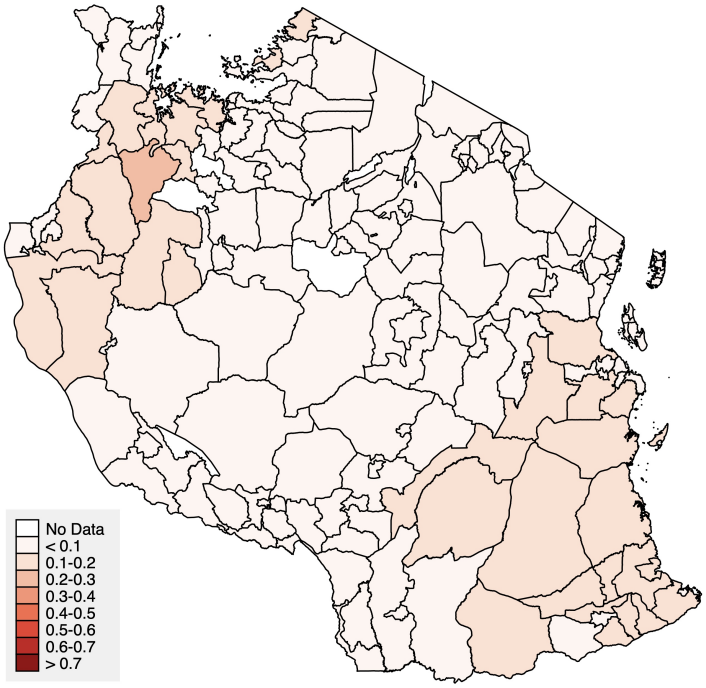
I merge the regional malaria prevalence data from MAP to the Tanzania National Census using the region of residence of the Census households. MAP follows the administrative boundaries announced by the Tanzania National Bureau of Statistics in 2012. In cases where administrative boundaries have changed over time, I harmonized them using the spatially harmonized geographic boundaries between 1988 and 2012 provided by the IPUMS-International.

Tanzania Population Census Household- and individual-level data on fertility and educational attainment are obtained from the Tanzania National Census, which has three waves: 1988, 2002, and 2012. Three outcome variables are of interest: mortality, fertility, and children's schooling (as a proxy for their human capital). The Census asked each female survey respondent for a complete birth history: the timing and number of every child they had given birth to, including those deceased at the time of the survey. I use the number of children who ever died to measure mortality. For fertility, I use the number of children ever born and the number of surviving children. The former is the number of all births a woman had given, in-

Figure 2: 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).

cluding children who died. Hence, this variable captures the gross fertility. On the other hand, the number of surviving children represents the net fertility. For mortality and fertility, I restrict the sample to women between the age of 35 and 49. Lastly, I used years of schooling, which is the total number of years a child had attended by the time of the survey for the children's human capital.

It is worth noting the advantages of using the Census data over the commonly used Demographic and Health Survey (DHS) data in estimating the effects of anti-malaria campaigns on our outcomes of interest. First, unlike the DHS data, the Census collects not only the regions of residence but also the region of birth. By restricting the sample to those born and residing in the same place, I can control for internal migration, which could be a potential confounding factor in estimation. Second, although the DHS covers many different sub-Saharan African countries, focusing on one specific country alleviates the problems coming from different timing of intervention across countries. Recent econometrics literature has pointed out that when there is heterogeneity in the timing of the treatment across groups, the two-way fixed effects (TWFE) difference-in-differences estimates can be biased (Callaway and Sant'Anna, 2021). Since the RBM campaign began to roll out at the national level in 2003, my estimates are free from such concerns.

3.3. Empirical Specification and Identification

The estimation strategy employed here is similar to those used in Wilde et al. (2019) and Kuecken et al. (2021). Since the RBM campaign was targeted toward the area with high malaria prevalence, I exploit the pre-campaign malaria prevalence as a proxy for the campaign intensity. I employ a difference-in-differences model with discrete treatment intensity, where each region falls into one of the four categories representing a different level of pre-campaign level of malaria prevalence. Specifically, I use the PfPR in the year 2001 as a criterion to allocate regions into the treatment category. The four campaign intensity categories are as follows: low prevalence regions (regions with PfPR $< 20\%$ in 2001), medium-low prevalence regions (PfPR between $20-50\%$ in 2001), medium-high prevalence regions (PfPR between $50-75\%$ in 2001), and high prevalence regions with PfPR $> 75\%$ in 2001.[6]

For mortality and fertility, where the dependent variables are count variables (number of chil-

[6]The thresholds used for the categorization are consistent with the standard cutoffs used in epidemiological studies of malaria, except that the malariometry literature label a region as low prevalence (hypoendemic) when the prevalence rate is less than 10%. However, the purpose of such classification is to facilitate easier communication between epidemiologist. For the purpose of the empirical analysis, I use the 20% cutoff as a baseline and conduct robustness analysis with different cutoff values. Appendix C contains the robustness results from using different grouping.

dren ever born or died), I estimate a Poisson regression model, in which the dependent variable is the logarithm of the expected number of child death/birth experienced by the woman.[7] Specifically, I estimate the following mortality and fertility equations:

$$M^c_{irt} = \beta^m_1 \text{Post}_t + \sum_{j=2}^{4} \beta^m_j \text{Post}_t \times \text{Prev}^j_{r,2001} + \boldsymbol{X}_{ijct}{}' \cdot \boldsymbol{\Gamma} + \eta_r \tag{5}$$

$$F^c_{irt} = \beta^f_1 \text{Post}_t + \sum_{j=2}^{4} \beta^f_j \text{Post}_t \times \text{Prev}^j_{r,2001} + \boldsymbol{X}_{ijct}{}' \cdot \boldsymbol{\Gamma} + \eta_r \tag{6}$$

where M^c_{irt} and F^c_{irt} is the logarithm of the expected number of child death or birth experienced by the woman i in age group c at time t, who was born and surveyed in the region r. The variable Post_t is the dummy indicating the pre-and post-treatment. It has a value of one in 2012 and zero in 2002. $\text{Prev}^j_{r,2001}$ is the indicator of whether a region r is in the prevalence group j, where $j \in \{2,3,4\}$ with 4 being the highest prevalence regions where PfPR exceeds 75%. Lastly, $\boldsymbol{X}_{ijct}$ is the vector of control variables, which are the age and years of schooling of the respondents and urban-rural residential status. I also include fixed effects for regions in all specifications.

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} is the years of schooling of a child i in age group c at time t, who were born and surveyed in the region r. The rest of the variables are the same as the fertility and mortality models, except that the years of schooling is now excluded from the control variables. Unlike the fertility and mortality regressions, the sample now includes both male and female respondents.

In the empirical analysis, I primarily focus on the the low- and high-prevalence regions as control and treatment groups. As seen in Figure A.2, malaria prevalence was consistently low throughout the 2001-2012 period in the low prevalence regions, and there is a clear reduction of prevalence in the high prevalence regions at the onset of the RBM program in 2003. Although the other regions in the medium-low and medium-high prevalence categories also experienced a reduction in malaria prevalence, it is unclear whether such a reduction was due

[7] 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.

to the RBM campaign because there is a pre-existing secular decline in the PfPR.[8] Appendix Table B1 reports the descriptive statistics for the entire sample as well as for the high- and low-prevalence regions.

The main parameter of interest in all specifications is β_4, which is on the interaction between Post_t and $\text{Prev}^4_{r,2001}$. For instance, if the RBM campaign was effective in reducing malaria-related mortality among children and women's fertility in the high malaria prevalence regions, we would expect $\beta_4^{f,m} < 0$. Similarly, if the RBM campaign's effect on children's educational attainment was positive, 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

Figure A.5 and A.6 illustrates the parallel trends in years of schooling between the high- and low-prevalence regions from 1998 and 2012, and Table 1 presents the results of the regression (7). Each column reports the estimate of β_4^e coefficients for different age groups. Columns 1 to 3 correspond to the cohorts of children likely to be affected by the RBM campaign, with descending intensity. For instance, the children between 10 to 15 years old in 2012 (column 1) represent the children born after the RBM campaign, hence likely to be fully benefited from the campaign. On the other hand, the last column represents an older cohort who are not likely to be benefited from the campaign, since these individuals were already beyond the school age and likely to have finished education. In that sense, the column can be interpreted as a placebo group. I only report the coefficients on the interaction of the post-treatment dummy and the indicator for the high-prevalence regions, since I am comparing the lowest and highest prevalence regions as control and treatment groups.

The positive and significant coefficients indicate that the children born in the regions with the highest malaria prevalence in 2001, and hence benefited the most from the RBM campaign, experienced an increase in years of schooling.[9] Compared to the low-prevalence regions, children in the highest prevalence regions who were between age 1 and 6 when the campaign began had additional 0.63 years of schooling. Children who were already in school when the campaign started had also benefited from it; those who were between 6 to 11 years old also experienced additional 0.97 years of schooling. However, the positive effects of the RBM dissipate for older age group (age 25-30 in 2012), as those individuals were likely to have completed schooling already when the campaign started in 2003.

[8] Results are robust to the exclusion of middle two prevalence groups and available upon request.

[9] Results are similar between boys and girls. Appendix Table D2 contains the results from the same regression run separately by gender.

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	4.28	6.85	6.68	5.78
$PfPR_{2-10}$ (75%+) × Post	0.633***	0.974***	0.473***	-0.172
	(0.098)	(0.122)	(0.096)	(0.181)
Observations	1,096,274	856,753	674,743	607,976

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 magnitude of the effects is in line with the existing literature's estimates. Lucas (2010) estimate that malaria eradication increased female educational attainment by as much as two years in the most heavily infected region, using Sri Lanka's national malaria eradication campaign in 1945. Bleakley (2010) examine historical episodes of malaria eradication in six Latin American countries and estimate positive effects on children's educational attainment in many countries. More recently, Kuecken et al. (2021) estimate 0.4 years increase in educational attainment among children who were exposed to the RBM campaign.[10]

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

Figure A.3 and A.4 plot the predicted values of the outcome variables in OLS version of equation (5) and (6), demonstrating the parallel trends for mortality and fertility conditional on observable control variables. As seen in the figures, both mortality and fertility rates were declining in all regions, but we observe a steeper decline in mortality in the high-prevalence regions upon the launch of the RBM campaign.

Table 2 reports the coefficients from regression (5) and (6). Each cell reports the coefficients from the Poisson regression, so the interpretation of the coefficient is the expected change in the log of the mean number of the dependent variable between the high-prevalence regions and the low-prevalence regions. In other words, being in the high-prevalence regions multiplies the mean of the dependent variable by a factor of $\exp(\beta_4)$. For example, when the dependent variable is the number of children ever born, a negative coefficient β_4^f is interpreted that women in the high-prevalence regions reduced fertility in response to the RBM campaign.

[10]For a comprehensive review of the literature, see Currie and Vogl (2013)

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	5.30	0.71	4.57
$PfPR_{2-10}$ (>75%) × Post	–0.0596***	–0.107***	–0.0255
	(0.008)	(0.035)	(0.017)
Observations	586,836	586,836	586,836

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.

Gross Fertility The first column summarizes the results for gross fertility, where the number of children ever born was used as a dependent variable. The coefficient β_4^f shows that childbearing-age women in the high-prevalence regions reduced their fertility by 5.96%. The negative response of fertility is consistent with Kuecken et al. (2021), which also found a reduced probability of childbirths in response to the RBM campaign in multiple sub-Saharan African countries. Within the framework of the quantity-quality tradeoff, the negative fertility response indicates that the drop in the price of child *quality* induced by the RBM campaign was larger than the drop in the price of child *quantity*.

Child Mortality Although the RBM campaign led the women in the high-prevalence regions to decrease their fertility, it does not immediately imply that the *net* fertility rate declined because the RBM campaign also lowered child mortality. If the reduction in gross fertility is smaller than the drop in child mortality, net fertility can still increase in response to the campaign. The second column of Table 2 shows to what extent the RBM campaign reduced the number of deaths among children in the high-prevalence regions. The coefficient β_4^m shows the RBM campaign reduced the number of child deaths per woman by 10.7%, indicating that the RBM campaign was indeed effective for reducing child mortality.[11]

[11]The results are consistent with Wilde et al. (2019) and Kuecken et al. (2021), which studied the effects of the RBM campaign on child mortality as well and found that the campaign reduced all-cause child mortality.

Net Fertility Combining the results from the gross fertility and child mortality regressions together, the last column shows the effects of the RBM campaign on *net* fertility. Instead of the number of children ever born, the number of surviving children was used as the dependent variable, which accounts for the immature deaths of children. Although the coefficient is negative, it is not statistically significant, indicating that although the RBM campaign induced the women have fewer children, it did not reduce the number of *surviving* children, at least after the ten years since the campaign started.

One interpretation for the muted response of net fertility is that the RBM campaign did not change the number of overall births women intended to have. In this interpretation, women reduced their fertility in response to the lowered mortality so that the number of surviving children remained the same. This interpretation can also explain the increase in children's educational attainment since having fewer children would have allowed parents to increase educational input in each child.

Another interpretation is a possibility that the RBM induced women to delay their fertility. Although possible, such an interpretation is not likely to change the conclusion that the net fertility did not change in response to the RBM campaign. The chance of pregnancy declines with age, so even if these women are delaying their fertility and planning to have more children at older ages, it is unlikely that it will lead to an increase in net fertility. Therefore, I conclude that the RBM campaign reduced gross fertility but did not change net fertility, although further investigation with extended data is needed.

3.4.3. Summary of Empirical Analysis

In summary, the RBM campaign is found to reduced women's fertility and child mortality by 5.9% and 10.7%, respectively. The campaign did not change the number of surviving children, suggesting that women reduced their fertility so that the number of surviving children remain constant.[12] The campaign also increased the years of schooling of the benefited children by 0.63 years. In the next section, I use these empirical estimates to discipline the model's quantitative mechanism.

[12]The muted response of net fertility is also consistent with the macroeconomic literature of demographic transition, which has been arguing that while a decline in child mortality is key for a decline in *gross* fertility, but is not sufficient to drive a decline in *net* fertility (Doepke, 2005).

4. Model Parameterization and Quantification

Guided by the empirical evidence in the previous section, I discipline the magnitude of the model's key mechanism by replicating the RBM campaign within the model and matching the empirical moments obtained in the previous section. The empirical analysis suggests that the reduction in malaria burden from the RBM campaign has induced the parents to increase educational investment to their children by sending them more to school, but the parents adjusted the fertility so that the net fertility does not change. I use these two moments which summarizes the effects of malaria on fertility behavior and children's human capital: the muted response of net fertility in (6) and increased educational attainment of children (β_4^e) in (7). To ensure the model is credible in other dimensions, I jointly estimate parameters to match the empirical elasticities and other relevant aggregate moments of the Tanzanian economy, such as educational attainment and intergenerational mobility measures. These moments are calculated from the microdata when possible, or taken from the existing literature otherwise.

4.1. Exogenously chosen parameters

A small set of parameters are chosen exogenously. Such parameters are summarized in Table 3. The exogenously chosen parameters can be classified into two broad categories. The first one is a set of parameters that are standard in the macroeconomics literature. Others are the parameters related to malaria, which are taken from epidemiological/health studies.

Standard Parameters The first panel of Table 3 shows the standard parameter values. The discount factor is chosen to be 0.96^6, consistent with the typical values in the literature, adjusted for the fact that the model period corresponds to six years. The exogenous gross interest rate is set to be 2 percent per year, representing the low financial access and low savings rate among households in low-income economies (Donovan, 2021).

It is worth mentioning the role of parameter γ, whose interpretation is the inverse of the intergenerational elasticity of substitution. This parameter is commonly interpreted as *intertemporal* elasticity of substitution in the macroeconomics literature, which governs the consumption tradeoffs between today's and tomorrow's consumption. In the life-cycle model with intergenerational linkage, γ also governs the degree of *intergenerational* elasticity of substitution because this parameter also affects how parents weigh their children's utility to their own utility. For instance, higher γ means that parents' marginal utility of consumption decreases faster as they become richer, which makes children relatively more valuable. I set the value of γ to 0.5, following Daruich and Kozlowski (2020). The fact that γ is less than one also ensures the

Table 3: Exogenously Chosen Parameters

Var	Description	Value	Source / Target
Economic Parameters			
β	Discount factor	0.96^6	Standard value
r	Exogenous gross interest rate	1.02^6	Deposit interest rate
γ	Inverse of IES	0.5	Daruich and Kozlowski (2020)
ω_2	Curvature of the time cost function	0.68	Folbre (2008)
$\bar{N}$	Maximum number of children	6	Tanzania Census 2002
λ	Elasticity of substitution between skill groups	4.0	Bils et al. (2022)
Epidemiological Parameters			
χ_U^d	Mortality rate, child from uneducated parents	0.109	Ogbo et al. (2019)
χ_P^d	Mortality rate, child from primary-educated parents	0.109	Ogbo et al. (2019)
χ_S^d	Mortality rate, child from secondary-educated parents	0.078	Ogbo et al. (2019)
χ_U^m	Prob. catching malaria, child from uneducated parents	0.79	Gonçalves et al. (2014)
χ_P^m	Prob. catching malaria, child from primary-educated parents	0.79	Gonçalves et al. (2014)
χ_S^m	Prob. catching malaria, child from secondary-educated parents	0.56	Gonçalves et al. (2014)

utility is positive everywhere. That is, parents always "enjoy" having children, and the implicit value of being childless or having a child die is zero.[13]

Fertility and Childcare Cost The maximum number of children parents can choose in the model is capped at $\bar{N} = 6$. Since a parent in the model corresponds to a household of two parents, a parent choosing $n = \bar{N}$ corresponds to a household having 12 children in reality. I choose $\bar{N} = 6$ because according to the Tanzania Census in 2002, 95% of the households have less than 12 children. The time cost function, $t(n) = \omega_1 n^{\omega_2}$, is an increasing and concave in the number of children. Typically, the literature estimates both the level and curvature parameters using the time-use data[14]. Unfortunately, detailed time-use data is not available in most low-income countries, and Tanzania is not an exception. Absent the available data, I follow the literature and set the value of the curvature parameter, ω_2 at 0.68, based on Table 6.4 in Folbre (2008).

[13] When the utility is allowed to be negative, we need extra assumptions on the value of being childless and having children die. See Jones and Schoonbroodt (2010) for discussion on this matter.

[14] See Lee and Seshadri (2019); Daruich and Kozlowski (2020), and Yum (2022) for the estimates of ω_2 from the US time use data.

Malaria Parameters related to child mortality and malaria are taken directly from epidemiological studies of malaria in the Sub-saharan Africa context. Two sets of parameters are needed: mortality (probabilities of deaths) and morbidity (probability of being sick from malaria). The mortality parameter, χ_s^d, corresponds to the under-age five mortality rate. I take this parameter from Figure 1 of Ogbo et al. (2019), which estimated the under-5 mortality rate in Tanzania between 2004 and 2016 using the Demographic and Health Survey. I use the estimates from the 2004-2005 DHS wave, which is the closest to the pre-RBM periods. Ogbo et al. (2019) also reports that children under five years whose mothers had primary or no education were 38% more likely to die before their fifth birthday compared to those whose mothers had a secondary or higher level of education (Table 2). Following their estimates, I set the mortality rate of children born from secondary-educated parents to be 38% lower than its lower-educated parents counterparts.

The parameter χ_s^d encompasses the probability of dying from all causes, including causes other than malaria. To calculate the fraction of the mortality rate attributed to malaria, I use the four waves of the Tanzania National Panel Survey (2008, 2010, 2012, and 2014) to calculate the fraction of malaria-caused deaths among all deaths. The survey asks the diagnosed cause of death for all deaths within the households, and malaria consistently accounts for about 51% of childhood deaths[15]. However, this number is likely to be an upper bound since only 5.9% of all deaths are identified, meaning that a lot of parents did not know which disease caused their child's death. If malaria is relatively easier to identify as a cause of death, then such a high fraction might be an overestimation. Instead, the Institute for Health Metrics and Evaluation (IHME) reports that about 17% of under-5 deaths were attributable to malaria in 2002 in Tanzania. I take the average of the two numbers and assume that 35% of all under-5 deaths are caused by malaria.

Parameters for morbidity probabilities, χ_d^m, are taken from epidemiological studies in Tanzania. From a study conducted between 2002 and 2005 among children in areas with intense malaria transmission in Tanzania, Gonçalves et al. (2014) concludes that the unconditional probability of experiencing malaria (either mild or severe) is 79%. Following this, I set 79.0% as the baseline probability of being hit by a morbidity shock in the model. I adjust the probability to 56% for children born from secondary-educated parents, as I did for mortality.

4.2. The RBM Campaign within the Context of the Model

I use the causal effects of the RBM campaign on fertility and children's educational attainment estimated in Section 3 to discipline the model's implied elasticities. To do so, I simulate the

[15] Exact numbers, along with the numbers for other causes of deaths, are reported in Table B4.

RBM campaign within the model and compare the model-generated moments to the estimated data moments. In this subsection, I describe how I interpret the RBM campaign from the perspective of the model and the procedure of the simulated method of moments.

I calibrate the baseline economy before the RBM campaign to the high-prevalence regions. When the region-specific parameters of moments are not available due to the data availability, I use the country-wide counterparts.[16] I then feed the RBM campaign to the model as an unexpected, universal reduction in the probability of negative health shocks. Compared to the pre-campaign periods, the malaria prevalence level was reduced by 70% (Figure A.2). Guided by this fact, I lower the morbidity probability χ_s^m and the malaria-attributed part of the mortality probability χ_s^d by 70% for all schooling groups s. Since the RBM was a nationwide campaign, all households in the model face the same reduction in malaria risk.

I also assure that the households in the economy do not expect a reduction in malaria risk and behave in anticipation. This approach prevents behaviors like accumulating assets in advance to increase educational investment when the campaign starts. To do so, I first simulate the balanced growth path of the economy with the pre-RBM level of malaria risks and then reduce the malaria risk unexpectedly. Households then adjust their fertility and education decisions according to the new decision rules under the changed disease environment. The simulated moments and their data counterparts are summarized in the last panel of Table 4, and the estimated parameter values are summarized in Table 5.

4.3. Parameters Estimated from the RBM Campaign

Aside from the exogenously calibrated parameters, the remaining 12 parameters of the model are calibrated to match the 14 moments, either from the RBM campaign or from the aggregate data. The two main outcomes of the RBM campaign are its treatment effects on fertility and children's education, measured by the number of children and years of schooling. Although all moments in the model are jointly determined, the parameter $\underline{m}$, which represents the negative effects of morbidity shock on returns from education, is most closely related to the two moments. The lower the $\underline{m}$ is, the more detrimental malaria is for a child's human capital formation. Hence, reducing the probability of malaria will have greater effects with lower $\underline{m}$. The estimated value of $\underline{m}$ is 0.76, suggesting that children with malaria receive 24% lower returns from schooling. This is broadly consistent with the epidemiological literature on malaria's effects on children's cognitive ability and school performance. For instance, in a study carried out in Sri Lanka, Fernando et al. (2003) find that children who experienced less than three

[16]The high-prevalence regions are relatively poorer than the rest of Tanzania and have smaller family sizes. However, the relationship between fertility, income, and parental investment is similar to the rest of the country.

attacks of malaria scored at least 15% more in both the special and school examinations than children who experienced more than five attacks of malaria during the same period. In another study that took place in Yemen, Al Serouri et al. (2000) conclude that having at least one attack of malaria was significantly associated with poor (below the 50th percentile for class/grade) school performance.

Table 4: Targeted Moments

Moments	Source	Data	Model
Education			
Primary completion rate (%)	Tanzania Census 2012	45.6	44.8
Secondary completion rate (%)	Tanzania Census 2012	11.5	10.3
Primary ed. wage premium (%)	Leyaro et al. (2014)	59.9	59.4
Secondary ed. wage premium (%)	Leyaro et al. (2014)	115.2	121.8
Intergenerational Mobility			
Uneducated-Primary intergenerational upward mobility	Alesina et al. (2021)	46.9	39.9
Uneducated-Secondary intergenerational upward mobility	Alesina et al. (2021)	4.7	3.3
Primary-Secondary intergenerational upward mobility	Alesina et al. (2021)	14.0	15.8
Differential Fertility			
Total fertility rate, uneducated parents	Tanzania Census 2012	5.00	4.94
Total fertility rate, primary completed parents	Tanzania Census 2012	4.67	4.56
Total fertility rate, secondary completed parents	Tanzania Census 2012	3.40	3.88
% of secondary-educated parents with 6+ children	Tanzania Census 2012	23.35	22.76
Inequality			
Gini coefficient	Younger et al. (2016)	0.38	0.42
Roll Back Malaria			
Treatment effect of the RBM on schooling (years)	Section 3	0.63	0.66
Treatment effect of the RBM on net fertility (%)	Section 3	0.00	0.01

4.4. Parameters Estimated from Aggregate Data

The remaining parameters jointly determine the targeted moments from aggregate data, as well as the moments from the RBM campaign. Primary and secondary school completion rates are calculated from the 2012 Census among adults younger than 30 years old. The low completion rates for both primary and secondary schools inform that child labor is quite prevalent. The two schooling fee parameters, p_P and p_S, pin down these two moments. The estimated parameter value of the primary school fee parameter p_P is -0.36. The negative sign of this

parameter suggests it is unlikely that the credit constraints are preventing the parents from sending their children to primary school because a negative p_P means parents receive money by sending kids to school. Instead, it means that child labor is an attractive alternative to educating children. On the other hand, the schooling fee for the secondary school, p_S, is positive at 1.18, implying a monetary cost of sending children to secondary school.

Table 5: Parameters and Estimated Values

Parameter	Value	Description
η_P	1.33	Human capital gain from primary education
η_S	1.64	Human capital gain from secondary education
p_P	-0.36	Goods cost of primary education
p_S	1.18	Goods cost of secondary education
σ_ν	0.16	Standard deviation of idiosyncratic income shock
σ_z	0.16	Standard deviation of the learning ability draw
ρ_z	0.92	Intergenerational persistence of learning ability
θ	0.39	Gumbel scale parameter of the fertility taste shock
ω_1	0.17	Level of time cost of childcare
δ	0.75	Level of intergenerational altruism
λ_n	0.72	Curvature of the altruism function
$\underline{m}$	0.76	Severity of malaria morbidity shock

The earnings premiums that primary- and secondary-educated workers enjoy (relative to the uneducated group), are taken from Table 3 of Leyaro et al. (2014), which estimated the education premiums with two the 2001/2006 Tanzania Integrated Labour Force Survey.[17] The two parameters related to these moments are η_P and η_S, which governs how much human capital grows from attending primary and secondary school, respectively. The estimated parameters are 1.33 and 1.64. Along with the low secondary school completion rate, the large η_S shape the sizable earnings premium the secondary-educated workers enjoy.

There are two parameters that govern intergenerational mobility in the model. The first is the AR(1) persistence parameters of the learning ability draw, ρ_z, and the other is the variance of the shock in the AR(1) process, σ_z. Intuitively, a high level of persistence would reduce inter-

[17]There are multiple sources for wage premium, all reporting similar estimates. Leyaro et al. (2014) use both IFS and the urban worker sample. Joseph (2020) used the Integrated Labor Force Survey, using primary educated workers as a reference group. Mlacha and Ndanshau (2018) also use the integrated labor force survey and get similar estimates.

generational mobility, while a high level of variance of the shock increases intergenerational mobility by making the learning ability more random. Given this intuition, I target three measures of intergenerational upward mobility, estimated in Alesina et al. (2021). Each moment represents the likelihood that children born to parents that have a certain level of education manage to attain a higher level of education. In the model, these moments are constructed from parent-children pairs from the balanced growth path of the model.

Several parameters jointly affect the fertility behaviors in the model. First, there are two parameters in the intergenerational discount function, $b(n) = \delta n^{\lambda_n}$, which govern the level of intergenerational altruism and its curvature to the number of children. These parameters are closely related to fertility decisions, although they also affect education decisions through intergenerational altruism. The estimated parameter values for δ, and λ_n are 0.75 and 0.72, matching the overall fertility rate. Another parameter that governs fertility behavior is ω_1, which represents the time cost of child-rearing. If the time cost is high, then high-skilled parents would be more reluctant to have children, generating a stronger negative relationship between income and fertility. I hence use ω_1 to match the differential fertility across the three skill groups. The estimated value of ω_1 is 0.17, and the fertility declines with the parental school level. Lastly, I use the Gumbel scale parameter of the fertility taste shock θ to match the distribution of fertility at the tail.

The remaining parameter σ_ν governs the standard deviation of the idiosyncratic shock to adult human capital. Given that this is also related to the variance of adult income, I discipline this parameter by matching the Gini coefficient in the year 2010, measured in Younger et al. (2016) using the Tanzania Household Budget Survey.

5. Long-Run General Equilibrium Effects of Malaria Vaccine

Using the estimated model, I simulate the long-run effects of a nationwide distribution of a successful malaria vaccine. A recent epidemiological study reports that the new malaria vaccine is up to 80% effective at preventing the disease in young children. Through the lens of the model, this is interpreted as an 80% reduction in malaria mortality (χ_s^d) and morbidity shock probability (χ_s^m). Since malaria accounts for 35% of all under five mortality in the model, the resulting reduction in mortality rate is sizable. To see the long-run effects, I simulate the new balanced growth path of the economy with the reduced malaria parameters and compare the aggregate moments between the pre-and post-vaccine balanced growth paths.

5.1. Quantitative Results

Table 6 summarizes the long-run, general equilibrium effects of a nationwide distribution of a successful malaria vaccine. The primary school completion rate increases significantly, from 45 percent in the pre-vaccine balanced growth path to almost 60 percent in the post-vaccine balanced growth path. The increase in the secondary school completion rate is relatively smaller. The smaller increase in secondary completion rate stems from the higher returns from child labor from secondary education-aged children. The higher school completion rates mean that the parents on the post-vaccine balanced growth path possess higher human capital. As a result, the negative income-fertility relationship becomes stronger. As seen in the second panel, fertility (the number of children ever born) falls in all skill groups. What is notable here is that despite the falls in gross fertility, the population growth rate *increases* in the post-vaccine balanced growth path, although the change is modest. The higher population growth implies that the decline in gross fertility rates is insufficient to compensate for the drop in mortality rate, as argued in Acemoglu and Johnson (2007).

The second panel of Table 6 demonstrates the changes in the skilled and unskilled wages and the increase in GDP per capita. GDP per capita increases significantly, up to 34% in the long run. Given that the population growth rate is also higher in the post-vaccine balanced growth path, such a high increase in GDP per capita suggests that the increase in aggregate production is more than enough to compensate for the larger population size. In the next subsection, I conduct several decomposition exercises to break down the sources of the GDP per capita gain. Lastly, the relative wage of unskilled workers rises by 4.4 percent while the skilled wage falls by 8.8 percent, reflecting the larger supply of skilled workers in the post-vaccine world.[18]

The last panel of Table 6 reports the changes in intergenerational upward mobility in terms of educational attainment. The fraction of children born to uneducated parents who manage to receive primary education increases by 12 percentage points. Upward mobility measures for higher education levels also increase, but with smaller magnitudes. The model implies that the improvement in intergenerational upward mobility is larger in households with less educated parents, even though the vaccine was universally distributed. Mass vaccination is pro-poor because when a child has malaria, unskilled parents are less likely to send the child to schools than skilled parents. Figure 3 illustrates the probability that a healthy (no malaria shock) and an unhealthy (malaria shock) attend primary school, depending on their parent's education level, after controlling for the number of children within the household. Two patterns are observed here. First, in all three groups of education level, parents are more likely to send their

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

Table 6: Long-Run General Equilibrium Effects of National Malaria Vaccine Policy

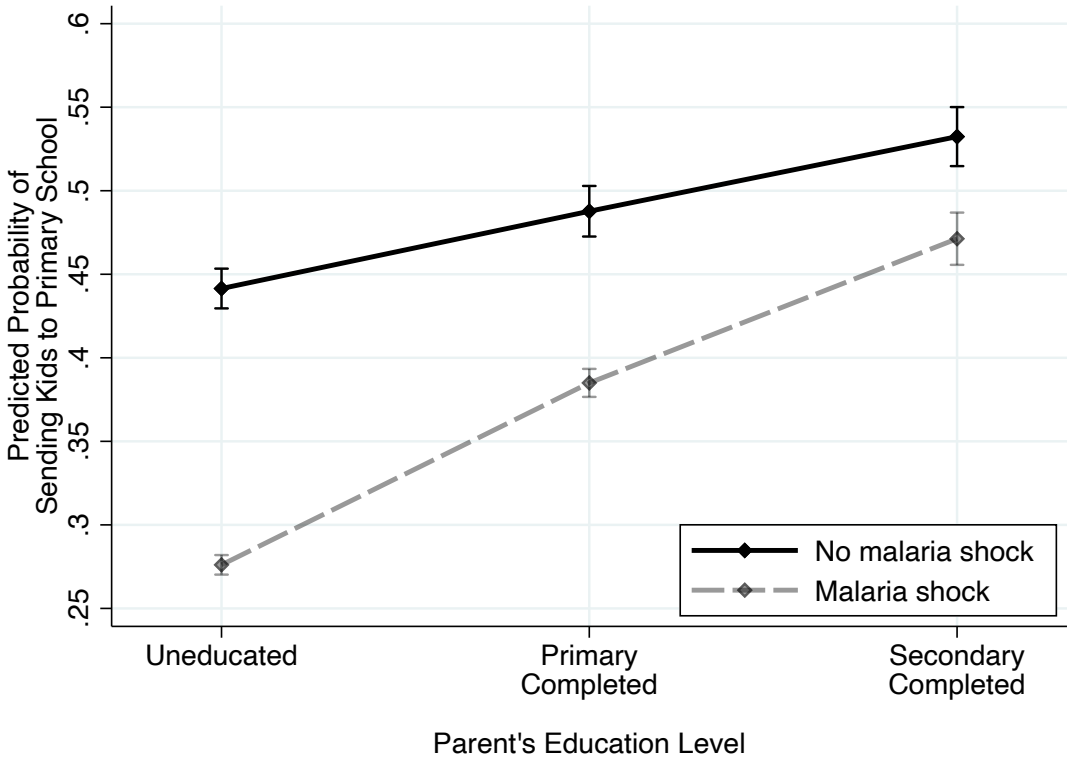
	Pre-Vaccine BGP	Post-Vaccine BGP	Change from Pre-Vaccine BGP
Education			
Primary completion rate (%)	44.8	58.3	+ 12.4 p.p.
Secondary completion rate (%)	10.3	14.4	+ 4.1 p.p.
Output per Capita and Prices			
Unskilled wage			+ 4.5%
Skilled wage			− 8.9%
Output per capita			+ 34.3%
Fertility			
Total fertility rate, uneducated parents	4.94	4.91	− 0.7%
Total fertility rate, primary completed parents	4.56	4.49	− 1.7%
Total fertility rate, secondary completed parents	3.88	3.77	− 2.8%
Population growth rate (%)	3.13	3.15	+ 2.0p.p.
Intergenerational Mobility			
Uneducated-Primary intergenerational upward mobility	39.9	51.6	+ 13.5 p.p.
Uneducated-Secondary intergenerational upward mobility	3.3	4.9	+ 1.6 p.p.
Primary-Secondary intergenerational upward mobility	15.8	18.1	+ 2.3 p.p.

Notes: This table reports the long-run, general equilibrium effects of nationwide malaria vaccination. The efficacy of the vaccine is assumed to be 80%.

children to school. In other words, parents tend to *reinforce* the adverse effects of the malaria shock on their child's human capital by not sending them to school. The second pattern is the heterogeneity of the reinforcing behaviors across different groups of education levels. Although parents from all educational backgrounds are less likely to send their children to school when they are sick, it is more so for the less educated parents. In other words, more educated parents are likely to *compensate* for the negative effects of malaria on children's human capital by sending them to school more, compared to the less educated parents.[19] As a consequence, the model predicts that vaccination is pro-poor by benefiting the children from less educated parents more.

[19]The terms "reinforce" and "compensate" are commonly used in the literature, but more commonly in a context of intra-household allocation of resources between children with different abilities or health conditions.

Figure 3: Child's Probability of Attending Primary School



Notes: This figure illustrates the likelihood that children born to parents with different levels of educational attainment complete primary schooling, depending on their health status at age six on the pre-vaccination balanced growth path. I simulated 50,000 parent-children pairs from the balanced growth path distribution and ran probit regression where the dependent variable is a binary variable of whether a parent sends children to primary school. Control variables include the idiosyncratic productivity shock, the number of children within the household, and children's learning abilities. Regression coefficients are plotted with 95% confidence intervals.

5.2. Sources of the Long Run Gains

The 34% increase in long-run GDP per capita is much larger than what previous literature has found. In this subsection, I formally compare this number to the numbers found in the previous literature and investigate the sources of the model's predicted large increase in per capita GDP.

5.2.1. Comparison to Acemoglu and Johnson (2007)

Acemoglu and Johnson (2007) examined the effect of longer life expectancy on economic growth by exploiting large the improvements in life expectancy driven by international health interventions in the 1940s, namely the international epidemiological transition. They found that a 1% improvement in life expectancy leads to a 1.7–2% increase in population but found

no evidence of growth in per capita income following a substantial increase in life expectancy. They even found relative *decline* in GDP per capita in countries that experienced large increases in life expectancy, suggesting that longer life expectancy contributes to population growth rather than improvement in economic growth.

The small or even negative changes in per capita output in Acemoglu and Johnson (2007) are seemingly contradictory to the model's large long-run increase in per capita output. However, their findings are perfectly consistent with the model's predictions once we interpret the international epidemiological transition through the lens of the model. Generally speaking, the model says whether eradicating diseases can decrease the long-run net fertility depends on if the eradication increases the children's human capital. In this sense, if the international epidemiological transition only lengthened the life expectancy and did not facilitate children's human capital accumulation, then simulating the transition within the model would generate the similar results as in Acemoglu and Johnson (2007).[20]

Table 7: Decompositon of the Long-Run Effects

	Lower Both	**Lower Mortality**	**Lower Morbidity**
Population Growth Rate (%)	3.15	3.26	3.02
Primary Completion Rate (%)	58.3	46.0	58.5
Secondary Completion Rate (%)	14.4	11.0	14.7
Δ Per capita GDP	+ 34.3%	+ 1.6%	+ 31.7%

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 80% reduction in malaria mortality (χ_s^d) while the morbidity shock probability (χ_s^m) is unchanged. The third column contains the results from a simulation with no change in malaria mortality while the morbidity shock probability is reduced by 80%. The first column is the baseline long-run simulation, where both mortality and morbidity probabilities are lowered.

To confirm this idea, I simulate a counterfactual long-run balanced growth path where I only lower either the mortality or morbidity to the post-vaccine level while keeping the other dimension of the disease at the pre-vaccine level. Table 7 contains the results from this counterfactual exercise. Although this is not the perfect way to replicate the epidemiological transition within the model, it is an informative exercise to check whether the model can nest the empirical ob-

[20]There are several reasons to believe that the epidemiological transition did little in increasing children's human capital. The most obvious observation is that schooling was not universal back in the 1940s, and many children were not able to take advantage of better health conditions by attending schools. Child labor was also more prevalent due to the lack of legal restrictions against it.

servations in the previous literature. The second column is the closest to the long-run effects of the epidemiological transition; fewer children die, but their returns from schooling are still low because morbidity shock is unchanged. The per capita output gain shrinks to a mere 1.6 percent. Moreover, the increase in primary school completion rate becomes very modest, compared to 45% in the pre-vaccine balanced growth path. Most importantly, a large increase in population growth rate (+ 0.13 p.p.compared to the benchmark post-vaccine balanced growth path) suggests that any individual-level income gains are dwarfed by the larger population size, resulting in a negative per capita income gain.

The key for a public health intervention to have a growth impact is whether it can facilitate education. Overall, the calibrated model suggests that even a significant reduction in mortality is not enough to generate a transformative increase in education when it's not accompanied by a reduction in morbidity. A key takeaway here is that uni-dimensional, mortality-focused modeling of disease could lead to biased results when calculating the economic effects.

5.2.2. Comparison to Ashraf, Lester, and Weil (2008)

Ashraf et al. (2008) simulate the effects of eradicating malaria using a standard neoclassical framework. In their framework, eradicating malaria raises life expectancy and years of schooling. Hence, they considered both the mortality and morbidity aspects of malaria. Nevertheless, they conclude that the effect of eradicating malaria in a typical sub-Saharan African country would be to raise GDP per capita by only about 2% in the long run.

There are two main differences between the simulation in this paper and that of Ashraf et al. (2008). The first one is the channels through which disease eradication (or reduction of disease prevalence in the current context) affects the human capital. Both assume that schooling increases human capital, but Ashraf et al. (2008) assume that eradicating malaria increases human capital only through increased years of schooling. This approach does not capture the channels other than years of schooling through which malaria eradication increase children's human capital, such as better learning in school due to higher cognitive ability, or better physical human capital.[21] The measurement of human capital in the model capture overcomes this weakness because, in the model, the malaria vaccine increases human capital not only through more schooling (more children sent to primary and secondary school) but also through improved learning in school.

[21] Bleakley (2010), for example, uses the malaria eradication in six Latin American countries circa 1955 as a natural experiment and studies how much adulthood earnings increased for the children who were born after the eradication. The study finds that 100% eradication of malarial infections in those countries increased subsequent adult income by >40%, and an increase in years of schooling only accounts for 25% of the earnings increase. This suggests that malaria eradication might have partially affected people's income through channels other than years of schooling.

The second difference of Ashraf et al. (2008) from the quantitative exercise of this paper is the lack of intergenerational dynamics. Ashraf et al. (2008) treat malaria eradication as a one-time increase in human capital, which does not affect future generations. However, it is debatable that eradicating malaria only affects one generation, without inducing the subsequent generations to change their education decisions for their children. If the children who benefited from eradication further increase educational investment for their own children, the increase in per capita output will be even larger in the long run. The overlapping generational structure and the intergenerational linkage embedded in the model allow us to capture this channel and investigate its quantitative importance.

Table 8: Decomposition of the Short-Run Effects of the Vaccination

	Benchmark Model	**Exogenous Fertility**	**Exogenous Schooling**
Children born right after the vaccination			
Primary completion rate (%)	55.2	55.1	44.8
Secondary completion rate (%)	11.0	10.9	9.4
Earnings at age 18 vs. parents	+ 18.0%	+ 17.6%	+ 12.8%

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. For the numbers in the third column, I do not allow parents to change their educational investment choice in response to the vaccination while letting them make fertility choices.

To understand the quantitative implications of the two departures mentioned above, I calculate the short-run effects of the malaria vaccine from two alternative scenarios where parents are not adjusting fertility or schooling decisions. Specifically, in each scenario, I do not allow the parents to have fewer children (exogenous fertility) or send children to school more (exogenous schooling). The second and third columns of Table 8 summarize the results of this exercise.

First, comparing the benchmark model where both fertility and schooling decisions are endogenous (column 1) manifests the importance of the amplifying effects of the intergenerational linkage. Comparing the 18% short-run partial equilibrium increase in per capita output with 34% of the long-run general-equilibrium counterpart reveals that the long-run general equilibrium effects are about 1.9 times larger. This implies that ignoring the intergenerational compounding effects could potentially underestimate the long-run gains by almost 50%.

Second, comparing the benchmark simulation (column 1) and the exogenous schooling sce-

nario (column 3) uncovers the quantitative importance of using years of schooling as the sole measure of human capital. The model predicts that even if we did not observe any increase in years of schooling among the children who receive the vaccines, their earnings in adulthood would have increased by 12.8 percent. In other words, an increase in years of schooling only accounts for 29% of the total increase in human capital. This is consistent with Bleakley (2010), who found that cohorts born after malaria eradication had higher adult income in six American countries. Of all six countries, years of schooling accounted for only less than a quarter of the effect of income. The model implies that measuring human capital through years of schooling would significantly underestimate the effects of malaria on human capital.

Combined together, we can do a back-of-the-envelope calculation of how much the long-run gains in output per capita will be reduced when we mimic the analysis in Ashraf et al. (2008). Since years of schooling accounts for only 29% of the human capital gain and intergenerational channel amplifies the short-run effects by 1.75 times, the long-run increase in per capita output in this scenario would be $34 \times 0.29 \times (1/1.9) \approx 5.2$ percent. This number is much closer to the 2 percent long-run gain that Ashraf et al. (2008) found.

5.3. Policy Counterfactual: Vaccine Efficacy and the Cost of Vaccination

Although the increase in output per capita is large in the long run, producing and administering vaccines to the mass population can be quite costly. For instance, Sicuri et al. (2019) estimate the per-child cost of administering malaria vaccine to range from $25 to $37 in a typical sub-Saharan African country, including all associated costs and assuming a vaccine price of $5 per dose.[22] Moreover, the vaccine's efficacy might turn out to be lower than the current 80%, and if so, the costs of vaccinating people might exceed the benefits.

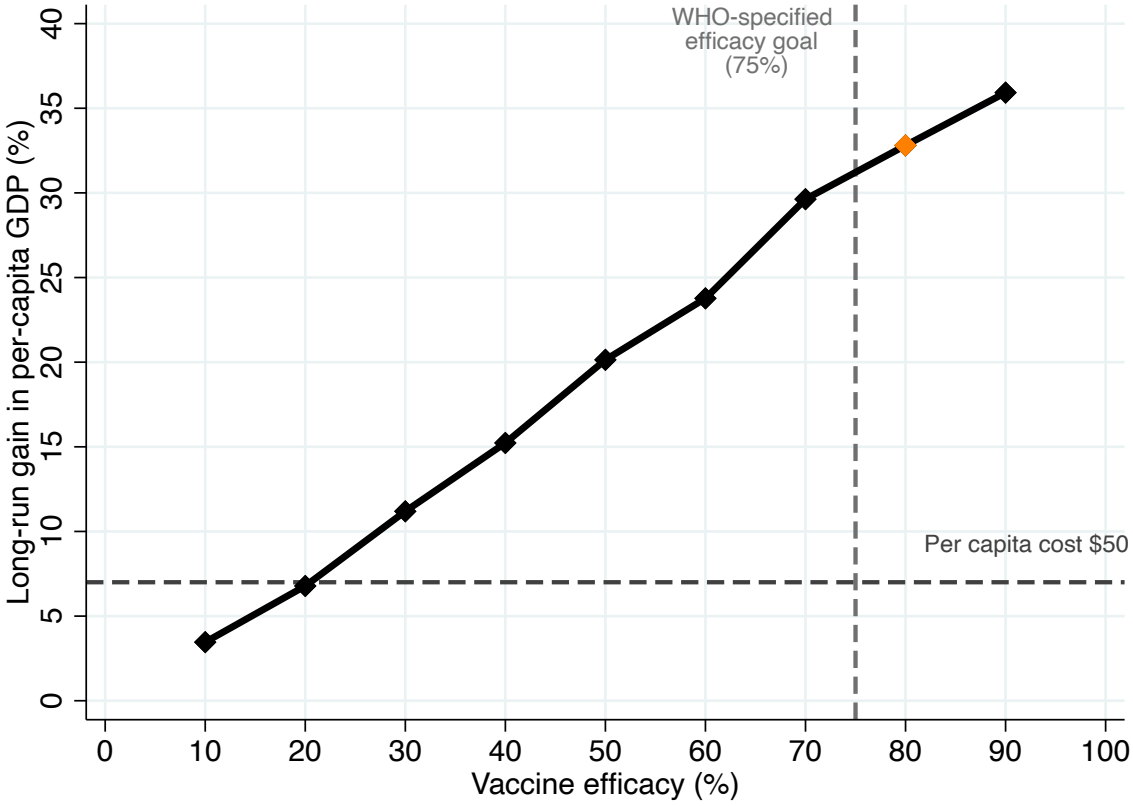
To see whether the long-run benefits are large enough to compensate for the costs of the vaccination policy, I solve the post-vaccine balanced growth path with a lower level of efficacy and compare the per-capita output gain to the cost of per-child vaccination. I take a conservative stance on the cost of vaccination and assume that per-child cost is $50, which roughly corresponds to a vaccine price of $10 per dose. Figure 4 shows the cost-benefit comparison of various efficacy scenarios. The vertical line at the 75% efficacy level represents the WHO-specified efficacy threshold for funding. The horizontal line indicates the $50 per-child cost of vaccination as a fraction of GDP per capita, using the GDP per capita in 2005 as a reference.

As seen in the figure, the long-run increase in output per capita clearly dominates the cost of vaccination, even though the costs used were conservatively high. Considering that the long-

[22] According to Datoo et al. (2022), four doses (three initial doses and a booster dose one year later) are needed to fully vaccinate one child.

run gains are about twice as large as the gains for the first generation who would receive the vaccines, the results imply that any vaccines with at least 40% efficacy would be cost-efficient, which is far below the WHO-specified efficacy level for financial support.

Figure 4: Vaccine Efficacy and the Cost of Vaccination



Note: 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.

6. Conclusion

High mortality and poor health conditions have been widely thought of as major development obstacles by policymakers in the developing world. Yet the macroeconomics literature thus far has found only small growth effects of health improvement. Using a quantitative dynamic macroeconomic model informed by reduced-form empirical evidence, this paper analyzes the long-run macroeconomic impacts of eliminating malaria, one of the deadliest diseases with a long-lasting cognitive damage for children in sub-Saharan Africa. In contrast to the previous literature, this paper argues that eliminating malaria in a typical sub-Saharan African country would generate large increases in output per capita, almost ten times as large as previously

estimated. Through the lens of the model, eliminating malaria cause parents and children to undertake higher investments in human capital, which drives substantial increase in living standards in the long run.

Developing countries, especially sub-Saharan African countries under the current context, are characterized by low fiscal and state capacity. While the vaccine will undoubtedly save many lives, whether the distribution of vaccines should be prioritized over other imperative development objectives depends on the size of economic benefits it generates. The results in this paper support prioritizing removing malaria as a core development objective. Moreover, since the long-run gains are materialized through the better education of healthier children, eliminating malaria can be complementary to policies aiming to improve the education system. Aside from the policy relevance, this paper also suggests that children's poor health conditions are one of the main reasons income per capita remains low in the developing world.

The model has several simplifying assumptions. Most prominent is perhaps the absence of physical capital. Improvements in health conditions may lead to a higher stock of physical capital since healthier workers can save more and increase the capital's marginal product. In this sense, including physical capital might further increase the long-run gains. However, it is not straightforward to expect that the physical capital would amplify the long-run gains because financial frictions might dampen the accumulation of capital, especially in developing countries where disease eradication is most needed (Banerjee and Duflo, 2005). Considering other channels, including the physical capital, through which health improvement affects long-run growth remains a topic for future research.

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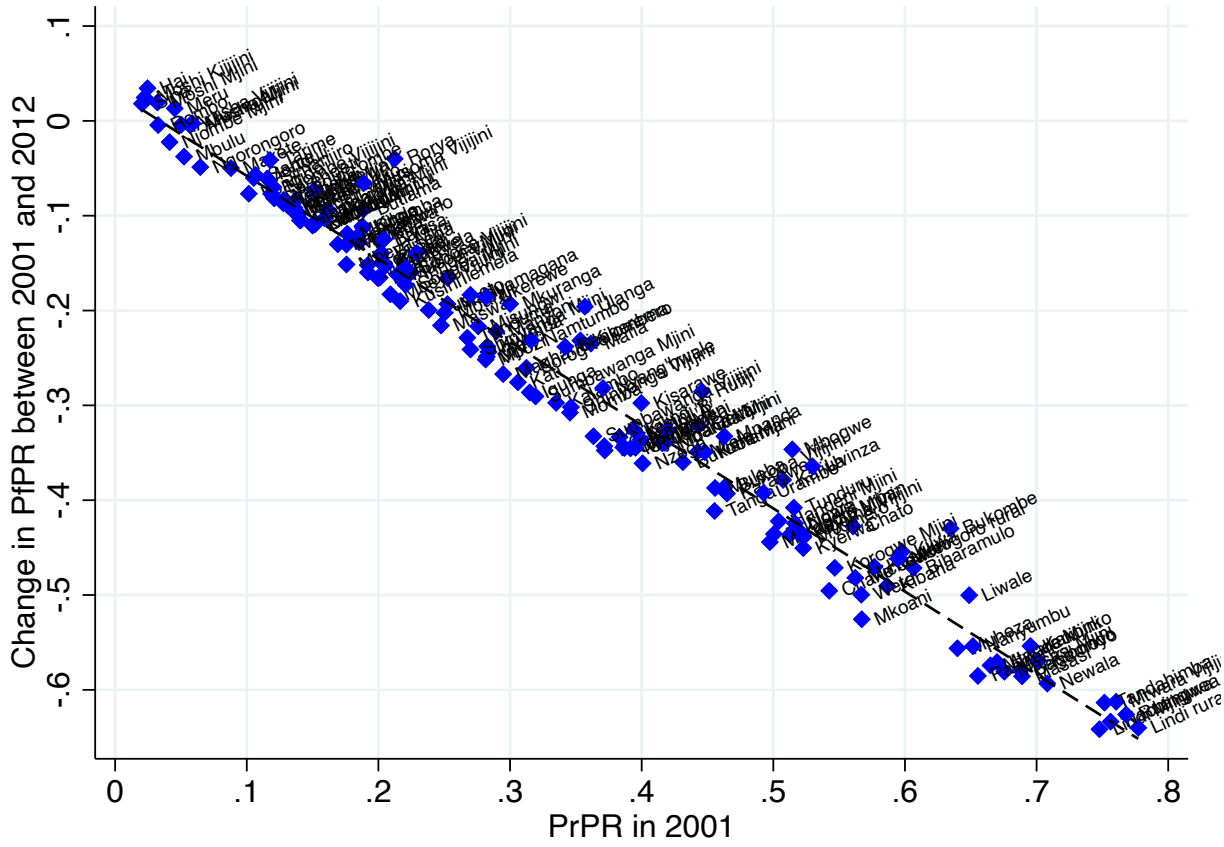
Appendix

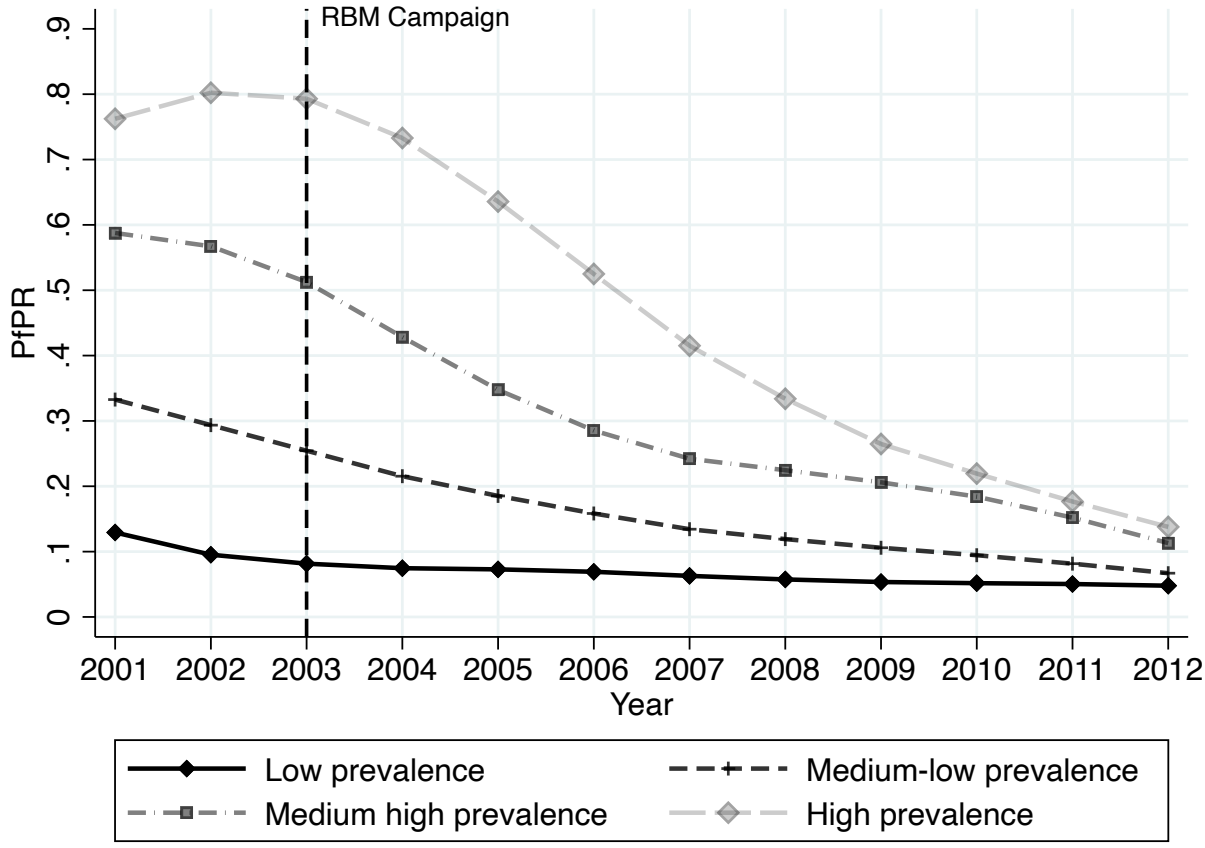
A. Appendix Figures

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



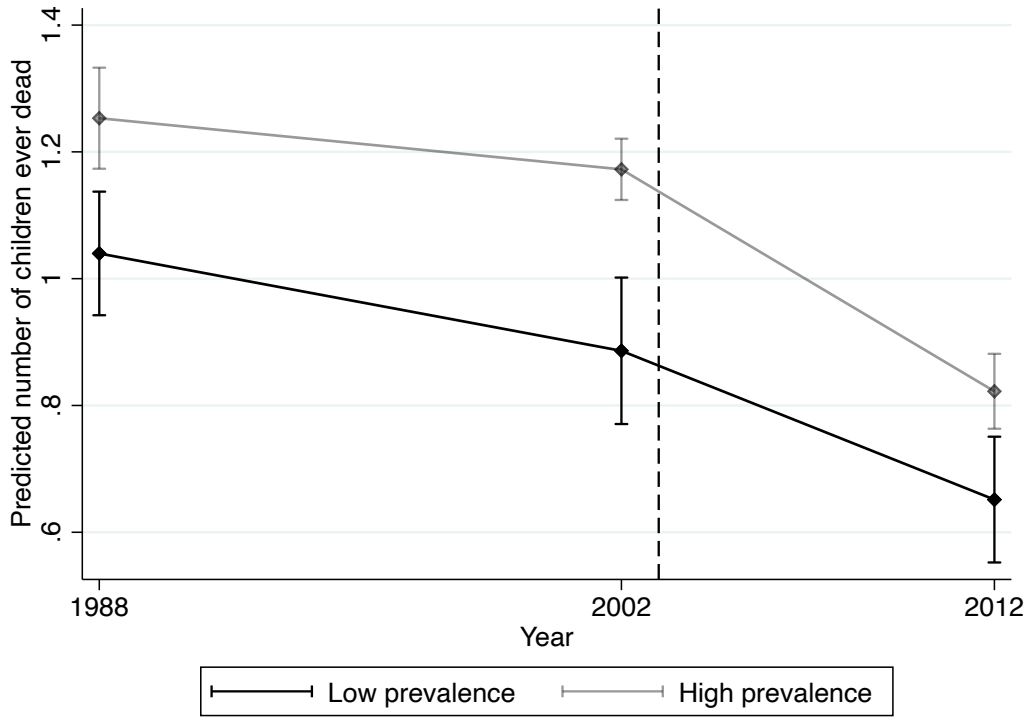
Notes: Each point represents a district at GIS-2 level. Yearly malaria risk (PfPR) is obtained from the Malaria Atlas Project (MAP). Fitted line is calculated by regressing the changes in malaria risk over 2001 and 2012 on initial malaria risk in 2001.

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



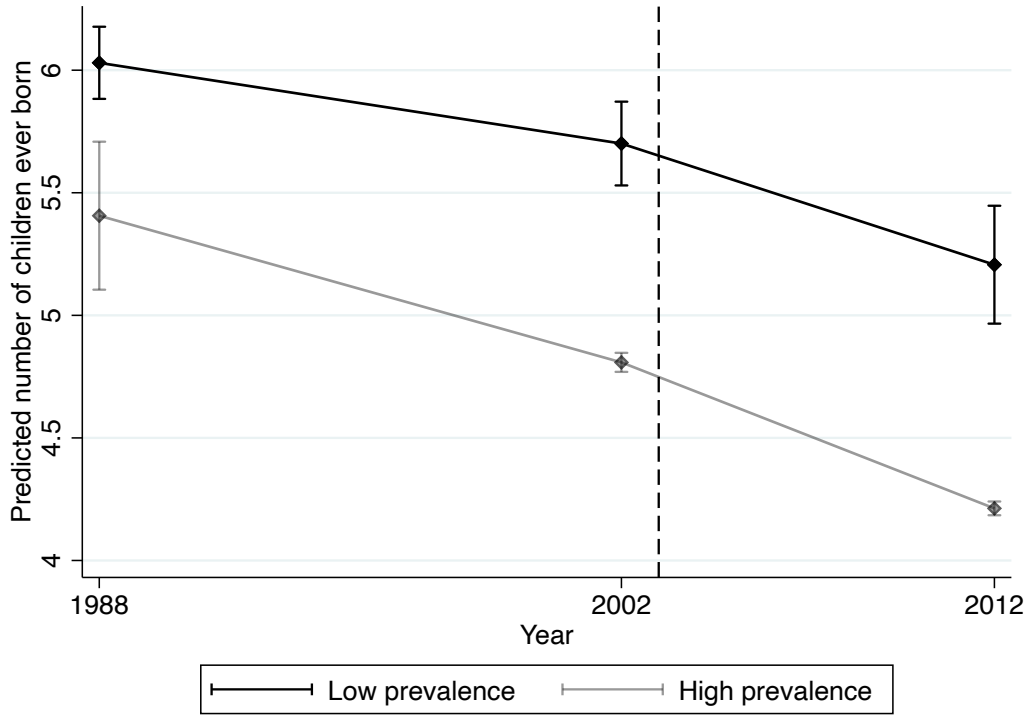
Notes: Each point represents a within-category population-weighted mean of PfPR. Regional malaria prevalence data obtained from the Malatia Atlas Project (MAP).

Figure A.3: Parallel trend in child mortality



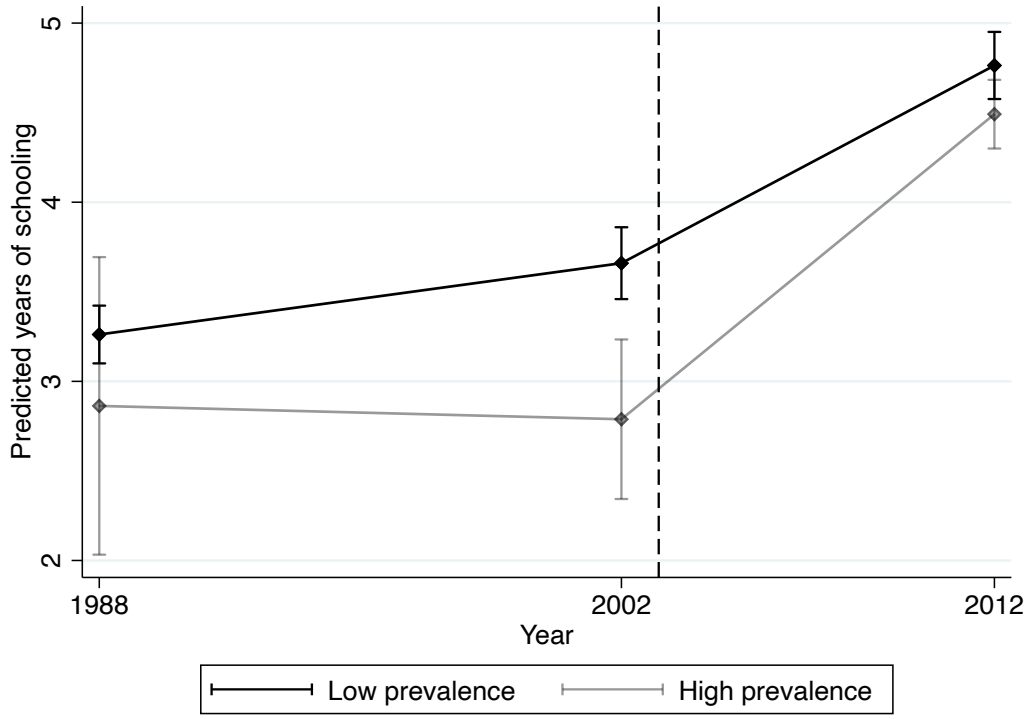
Notes: This figure illustrates the parallel trend between the high- and low- prevalence regions by plotting the mean of the number of children ever dead conditional on the covariates used in regression (5). Three waves of the Tanzania National Census (1988, 2002, 2012) are used. Samples are restricted 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 fertility



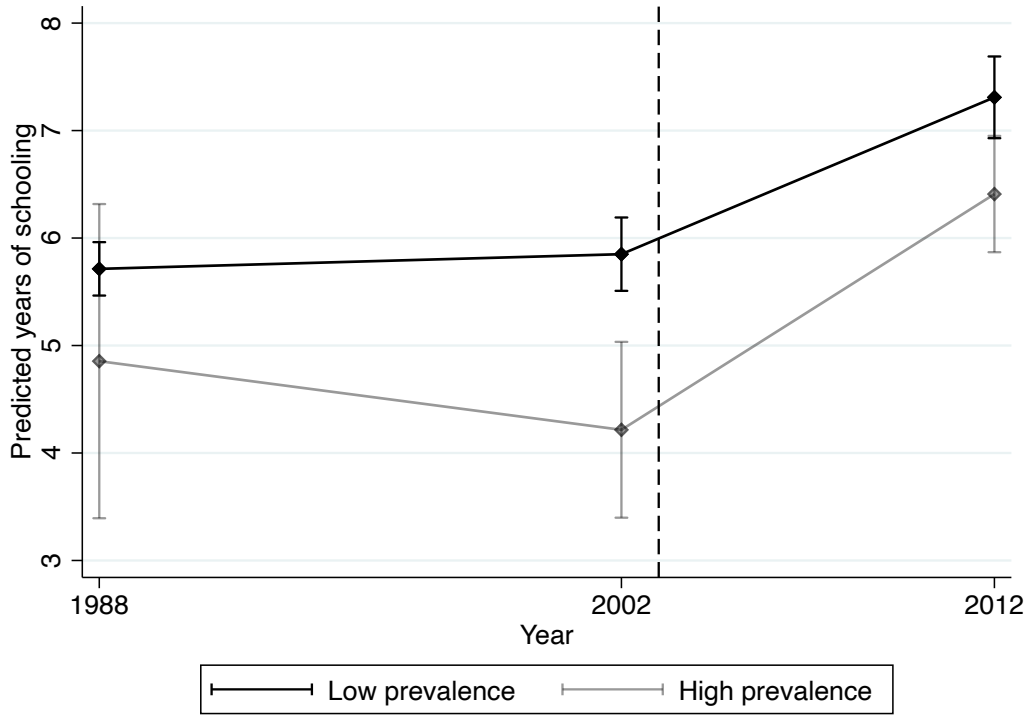
Notes: This figure illustrates the parallel trend between the high- and low- prevalence regions by plotting the mean of the number of children ever born conditional on the covariates used in regression (6). Three waves of the Tanzania National Census (1988, 2002, 2012) are used. Samples are restricted 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	37.48	37.55	37.81
	(5.624)	(5.621)	(5.666)
Number of children ever born	5.751	5.622	4.855
	(3.018)	(2.887)	(2.697)
Number of children dead	0.985	0.856	1.204
	(1.386)	(1.316)	(1.550)
Years of schooling	4.407	4.672	3.973
	(3.484)	(3.420)	(3.362)
% Household w/ electricity	0.0695	0.0791	0.0288
	(0.254)	(0.270)	(0.167)
% Household w/ water supply	0.347	0.421	0.185
	(0.476)	(0.494)	(0.388)
PfPR in 2001	0.336	0.135	0.763
	(0.194)	(0.0510)	(0.00664)
Number of families in household	1.365	1.323	1.492
	(0.936)	(0.866)	(1.041)
Labor force participation	0.832	0.855	0.880
	(0.374)	(0.352)	(0.325)
Urban-rural status	0.355	0.340	0.400
	(0.479)	(0.474)	(0.490)
Observations	244,343	76,836	6,209

Notes: Calculated from 2002 Census data. Sample is restricted to women between age 35 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	4.28	6.85	6.68	5.78
Post	1.144***	1.431***	1.040***	0.358***
	(0.041)	(0.068)	(0.085)	(0.079)
$PfPR_{2-10}$ (20% − 50%) × Post	0.006	0.032	0.012	-0.130
	(0.059)	(0.088)	(0.109)	(0.099)
$PfPR_{2-10}$ (50% − 75%) × Post	-0.019	0.140	-0.027	-0.290**
	(0.093)	(0.122)	(0.137)	(0.140)
$PfPR_{2-10}$ (75%+) × Post	0.633***	0.974***	0.473***	-0.172
	(0.098)	(0.122)	(0.096)	(0.181)
Age	0.711***	0.130***	-0.081***	-0.075***
	(0.008)	(0.008)	(0.004)	(0.005)
Urban	0.759***	1.383***	1.566***	1.433***
	(0.040)	(0.068)	(0.071)	(0.067)
Observations	1,096,274	856,753	674,743	607,976

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	5.30	0.71	4.57
Post	–0.104***	–0.316***	–0.073***
	(0.008)	(0.015)	(0.009)
PfPR_{2-10} (20% − 50%) × Post	0.012	0.019	0.016
	(0.010)	(0.021)	(0.011)
PfPR_{2-10} (50% − 75%) × Post	0.009	0.012	0.017
	(0.013)	(0.022)	(0.013)
PfPR_{2-10} (>75%) × Post	–0.0596***	–0.107***	–0.0255
	(0.008)	(0.035)	(0.017)
Age	0.026***	0.044***	0.022***
	(0.000)	(0.001)	(0.000)
Years of schooling	–0.016***	–0.057***	–0.008***
	(0.001)	(0.002)	(0.001)
Urban	–0.141***	–0.234***	–0.126***
	(0.005)	(0.015)	(0.006)
Observations	586,836	586,836	586,836

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

	Wave 1	Wave 2	Wave 3	Wave 4	Average
Panel A: Top 5 illnesses that led to hospitalization (%)					
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

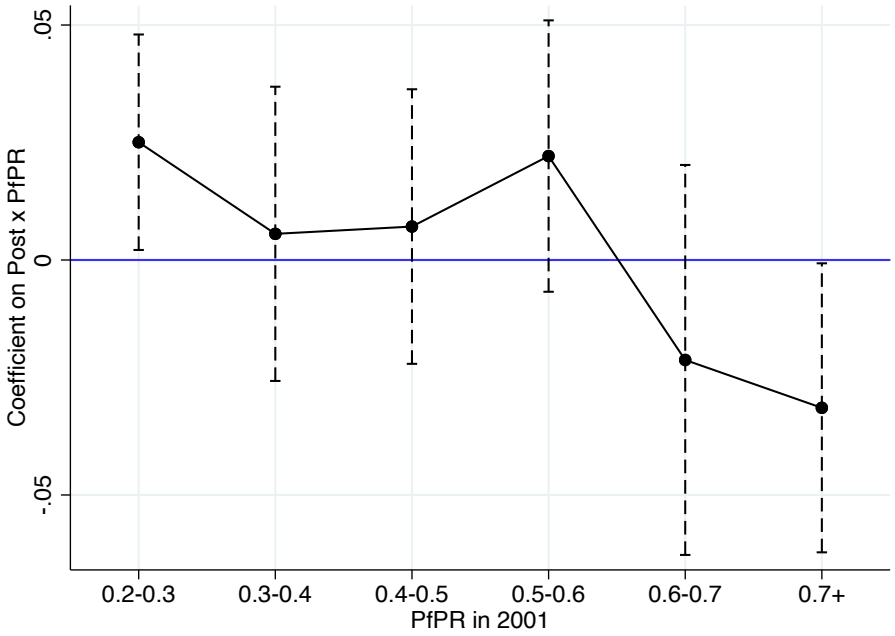


Figure C.2: Child Mortality

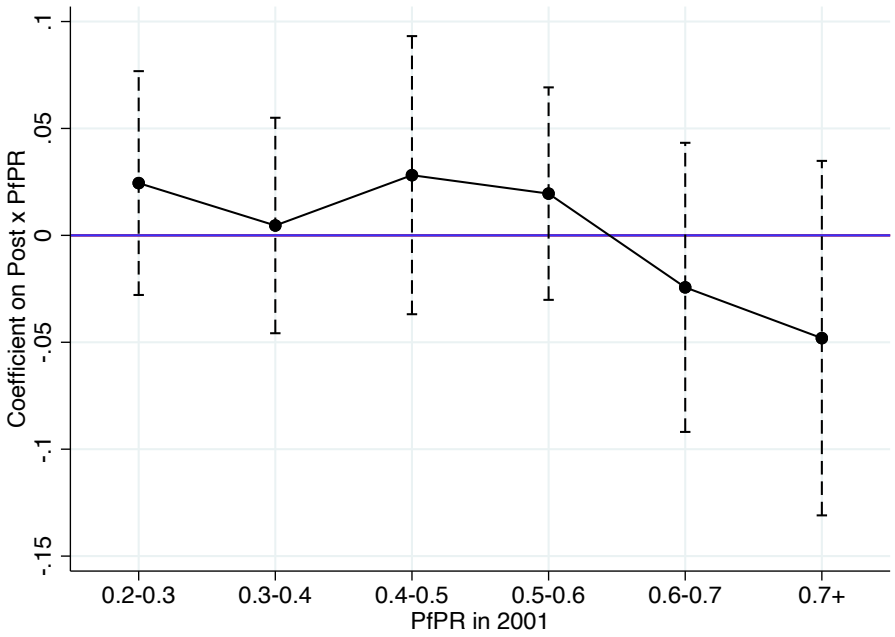


Figure C.3: Net Fertility

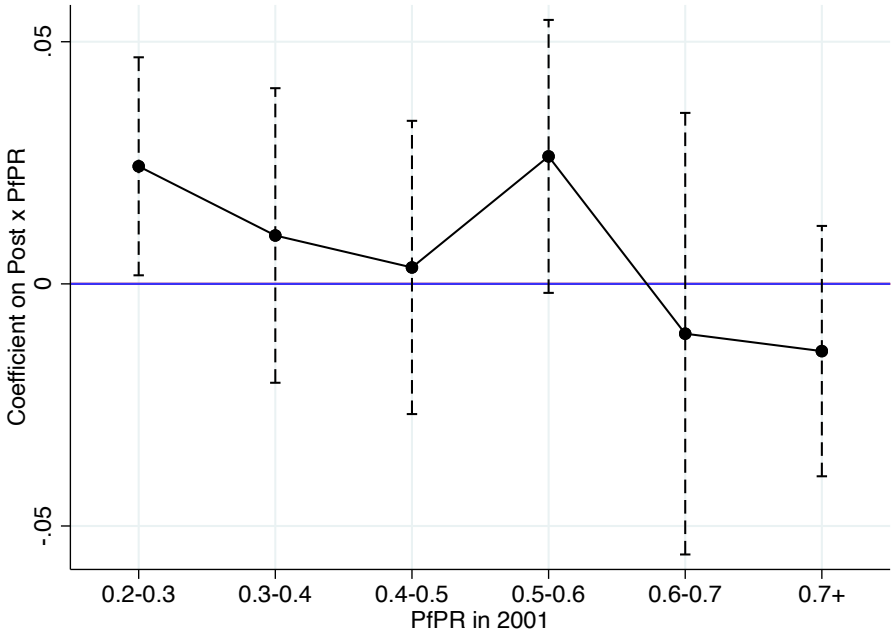


Figure C.4: Yrs of schooling 10-15

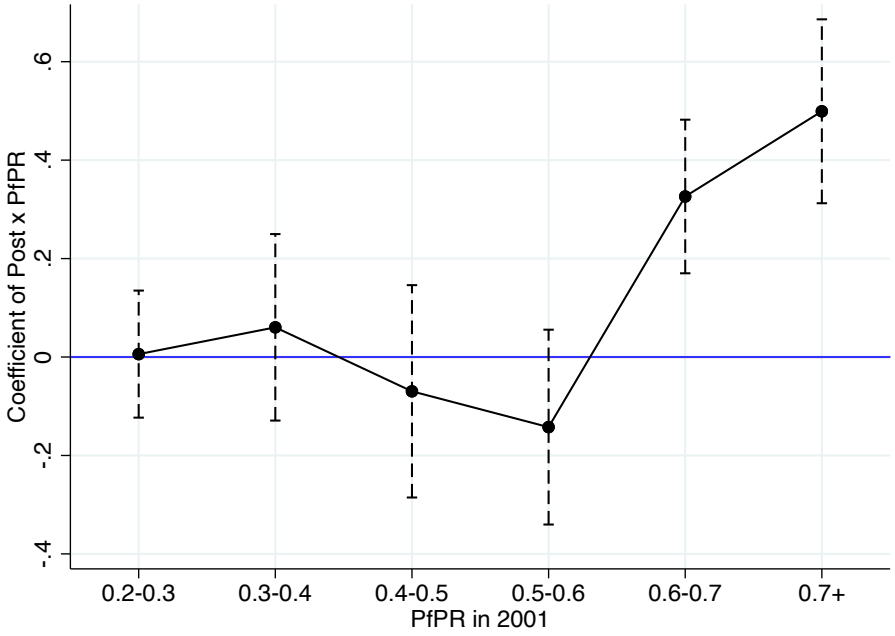


Figure C.5: Yrs of schooling 15-20

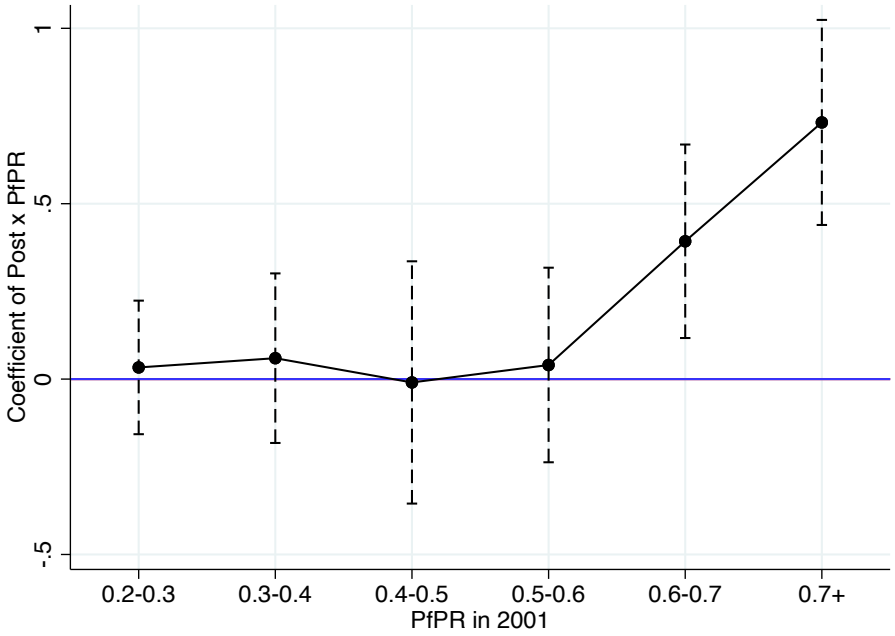
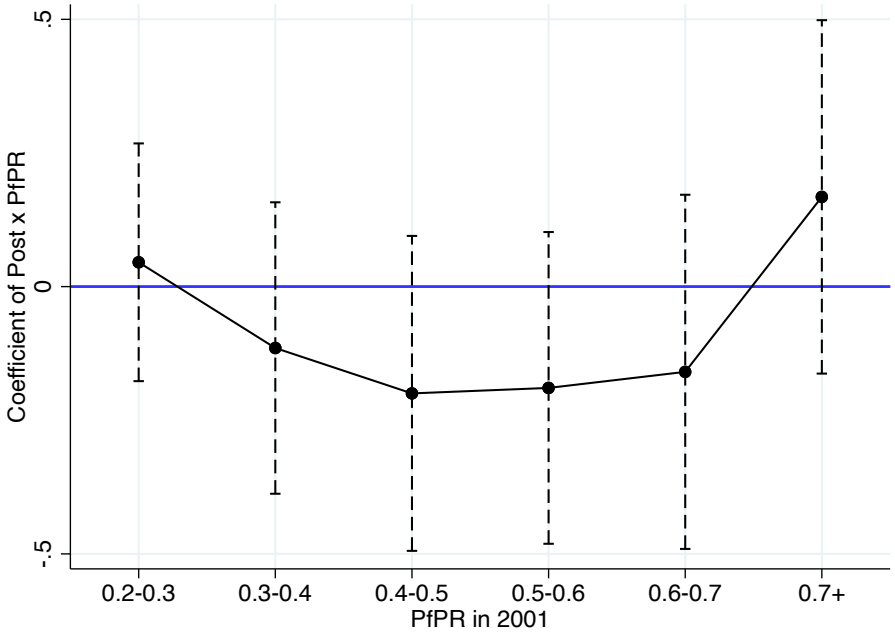


Figure C.6: Yrs of schooling 20-30



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%+) × 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%+) × 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%+) × 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%+) × 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				
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 for 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.