The Impact of Malaria Eradication on Fertility

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I. Introduction

Despite the prevalence of malaria in developing countries, a consensus has not emerged about its effects on growth and development. In the growth literature, Acemoglu and Johnson (2007) found that the "international epidemiological transition" did not increase gross domestic product (GDP) per capita, even though Gallup and Sachs (2001) found a negative correlation between malaria and economic growth.[1] Microeconomists have found that malaria eradication increased the lifetime schooling and/or productivity of those in utero or very young at the time of eradication (e.g., Barreca 2010; Bleakley 2010; Cutler et al. 2010; Lucas 2010). Further, Conly (1975) found that malaria eradication increased contemporaneous agricultural productivity. Therefore, while the full benefit of malaria eradication might not be realized until those born after eradication start production, some benefits to GDP should appear immediately. However, if malaria eradication expanded the size of the dependent population through increased fertility and child survival, then the full benefits to GDP per capita could be further delayed. This article provides convincing evidence of the effect of malaria on fertility and offspring survival and reconciles the seemingly incongruous findings across the two types of literatures.

Since live births are a function of both fecundity, the ability to have a live birth, and preferences about the target number of live births, a priori the sign of the net effect of malaria, combining both direct and indirect effects, on the total number of live births per woman is uncertain. The direct effect of malaria infections on fecundity is negative: increased probability of spontaneous

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[1] As a part of the "international epidemiological transition," Acemoglu and Johnson (2007) included public health improvements for malaria and 14 other diseases. They found no net effect on GDP per capita from the sum of the health improvements.

abortions and still births, reduced coital frequency, and a decrease in general maternal health.[2] These direct effects are more severe for women pregnant for the first time, primigravidae, than women of higher-order parity, multigravidae. Primigravidae exhibit higher parasite prevalence, are almost twice as likely to have placental malaria, and have higher rates of malaria-related anemia than multigravidae (McGregor 1984; Tako et al. 2005).[3] Because of the higher rate of infection and parasite load, primigravidae are at increased risk for spontaneous abortions and still births (Archibald 1956, 1958; Brabin et al. 1998; Brabin and Rogerson 2001). Therefore, on average, fewer first pregnancies result in live births than higher-order pregnancies. This epidemiology predicts that the direct effect of maternal malaria could be to delay the first birth while having less of an effect on subsequent birth spacing, as higher-order pregnancies are less susceptible to the most severe malaria complications.

The sign of the indirect effect of malaria that operates through fertility preferences is less clear. Models of fertility predict that lower income and an increased price of a surviving child, as was the case in the preeradication period, would suppress fertility. Conversely, the lower survival probability of children prior to eradication could result in higher fertility if households engaged in precautionary childbearing or preferred quantity over quality (see models of fertility in Barro and Becker [1989], Galor and Weil [2000], Kalemli-Ozcan [2003], and Doepke [2005]). These indirect channels are unlikely to vary by parity, unlike the direct effects of maternal infections discussed above.[4]

As with fertility, the net effect of malaria on the survival of live births is the sum of both direct and indirect effects. In the case of survival, the effects are negative in both cases. Directly, maternal malaria infections while the fetus is in utero cause a reduction in low birth weight, and postnatal malaria in-

[2] All women, even those with acquired immunity prior to pregnancy, are at risk for malaria infections and related complications.

[3] These differences by parity emerge with the creation of the first placenta, a new nonimmune organ that the malaria parasite attacks. Upon the creation of future placentas, women who were infected during a prior pregnancy have antibodies that can at least partially prevent parasites from adhering to the placenta, resulting in less severe symptoms.

[4] For example, increasing fertility by continuing fertility for a longer duration or decreasing birth spacing for all births would not exhibit the parity-specific pattern. I cannot empirically distinguish between a change in behavior that mimics the expected biological response and the direct malaria effect. The increase in survival for the first born (presented in Sec. V) provides additional suggestive evidence. Additionally, some of the direct effects of malaria are the same regardless of birth order. Similar outcomes for all parities would not be sufficient to reject the presence of a direct biological effect.

fections can cause infant or child mortality (Duffy and Desowitz 2001; Duffy and Fried 2001).[5] Because of more severe maternal infections, low birth weight and infant mortality are more likely for first births than for higher-order births (Archibald 1956, 1958; Brabin et al. 1998; Brabin and Rogerson 2001). Indirectly, expenditures on malaria treatment or illness-related forgone adult income could reduce money available for nutrition or other inputs into health production. The total effect could make survival less likely prior to eradication. Direct effects should affect first-born offspring disproportionately, while income and postnatal malaria effects would most likely be uniform by birth order.

I treat the national malaria eradication campaign in Sri Lanka as a quasi experiment and estimate the relationship between the exogenous decline in malaria, the fertility of women of childbearing age around the eradication period, and the survival of their offspring. I combine data from the nationally representative World Fertility Survey (WFS) with regional malaria rates from Newman (1965). To identify the effects of malaria, I exploit temporal and spatial variation in malaria exposure induced by the combination of heterogeneity of preexisting malaria rates and Sri Lanka's national malaria eradication campaign that reduced all malaria rates to zero.[6] The difference-in-difference estimates suggest that for those of childbearing age around the time of malaria eradication, the fall in malaria caused an increase in fertility and a younger maternal age at first birth. The effect of malaria on subsequent birth spacing is inconclusive. Further, I find that malaria eradication resulted in an increase in the probability of survival of the first-born offspring. Taken together the results suggest that the direct effects of maternal (or in utero) malaria infections were important but potentially not the only avenue through which malaria eradication increased fertility and survival. The increase in fertility and child survival provide a simple explanation for the seemingly contradictory findings of the growth and development literatures. The immediate increase in population size would only gradually be offset by additional educational attainment among the posteradication cohorts. Therefore, while others have found positive effects of malaria eradication on schooling, an increase in GDP per capita might not be expected immediately.

[5] Based on a meta-analysis of studies published from 1985 to 2000 on malaria infection in pregnancy, Steketee et al. (2001) summarized that malaria infections are responsible for 8%–14% of full-term, low-birth-weight babies, 8%–36% of preterm, low-birth-weight babies, and 3%–8% of infant mortality in areas in which malaria is endemic.

[6] Bleakley and Lange (2009) apply a similar identification strategy to hookworm eradication in the American South and find fertility decreased upon eradication.

II. Background

The malaria parasite is transmitted to humans through the bites of infected female *Anopheles* mosquitoes. Transmission requires adequate but not excessive rainfall, warm temperatures, humans, *Anopheles* mosquitoes, and the presence of the malaria parasite. These environmental and geographical constraints of the mosquito and the parasite greatly influenced the preeradication geographic malaria prevalence within Sri Lanka. Sri Lanka's precipitation pattern can be classified into three zones: wet, dry, and intermediate (Visvalingam 1961). Malaria prevalence in Sri Lanka was the highest in the northern and eastern dry zone and lowest in the wet zone around Colombo, where the 100 inches of rainfall per year washed away suitable breeding sites (Newman 1965). Between these two zones is the intermediate zone that received between 80 and 100 inches of rain per year.[7] As can be seen in figure 1, the preeradication malaria spleen rates, the portion of school children with enlarged spleens, a common symptom of a long-standing malaria infection, are highly correlated with this rainfall pattern.[8]

The World Health Organization (WHO) malaria eradication campaign began in Sri Lanka in 1945 and achieved nationwide coverage in 1947. As with other WHO campaigns, the goal of the campaign was to eliminate malaria by disrupting contact between infected mosquitoes and people. In the absence of transmission between people and mosquitoes, the malaria parasite is unable to sustain itself and dies. In practice, centrally trained and instructed teams sprayed the interior walls of dwellings with diluted DDT, a method referred to as indoor residual spraying. Mosquitoes that rested on treated walls would die for up to 6 months after the pesticide application, breaking the cycle of transmission. The spraying cycle was repeated once every 10–12 weeks, depending on the severity of the malaria transmission (Wickremesinghe 1953). Figure 2 plots the malaria spleen rate by region over time.[9]

III. Empirical Strategy

The primary difficulty in identifying the effect of malaria on fertility is the potential correlation between unobservable regional characteristics and the

[7] According to the zones as classified by Rajendram and Jayewickreme (1951), the districts of Kalutara and Galle lay entirely in the Wet Zone as does the majority of Colombo and parts of Negombo, Kegalla, Ratnapura, and Matara. The Intermediate Zone consists of parts of Colombo, Negombo, Kegalla, Matale, Nuwara Eliya, Ratnapura, and Matara, with the rest of the country in the Dry Zone.

[8] Jaffna Peninsula in the far north of the country appears to be an outlier in this geographic allocation of malaria. Newman (1965) notes the collection problems and nonrepresentative samples collected in that district. A figure similar to fig. 1 appears in my earlier paper, Lucas (2010).

[9] A similar figure appears in Lucas (2010).

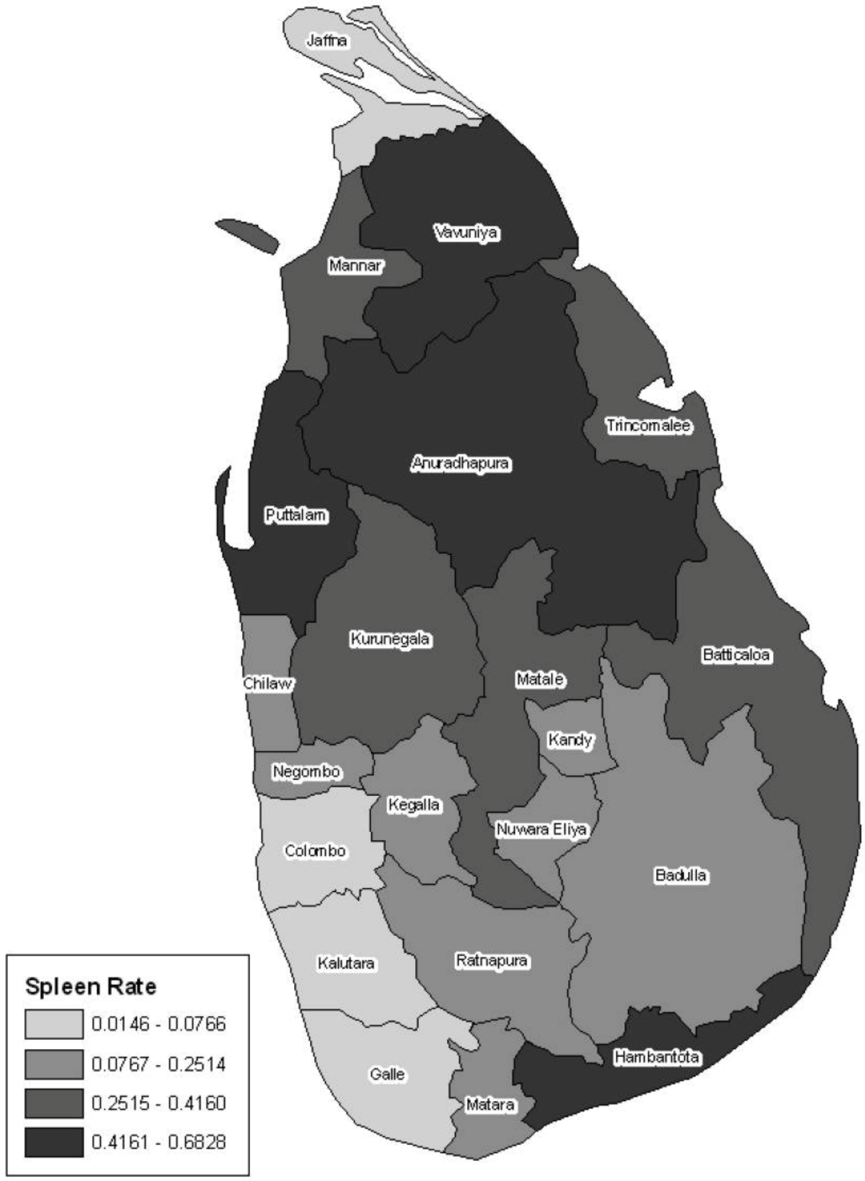

Figure 1. Sri Lanka average spleen rates, 1937–41

level of malaria. Further, decreases in malaria levels are often the result of improving incomes that could also have an effect on fertility and survival. To overcome these potential biases, I use the exogenous change in malaria from the malaria eradication campaign as a natural experiment that provides both temporal and spatial variation in malaria exposure.

I estimate the effect of malaria on the probability of a live birth using a difference-in-differences approach:

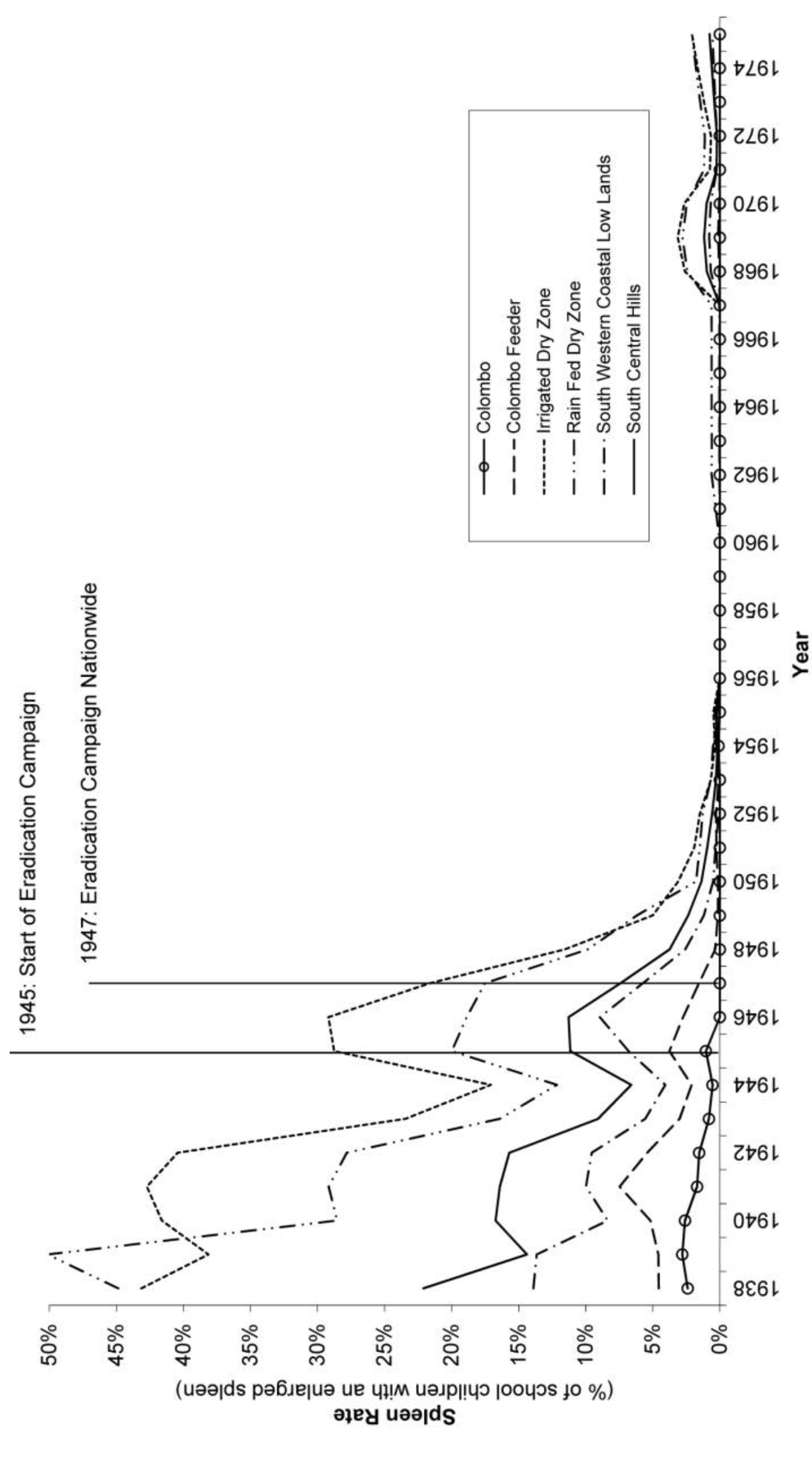

Figure 2. Spleen rates by World Fertility Survey region. Spleen rates are aggregated from Newman (1965)

$$\text{birth}_{ijt} = \alpha + \beta(\text{malaria}_j \times \text{pre}_t) + X'_{ij}\Gamma + \delta_j + \delta_t + \gamma_j t + \varepsilon_{ijt}, \qquad (1)$$

where birth_{ijt} is an indicator equal to one if woman i in region j gave birth in year t, malaria_j is the regional preeradication malaria spleen rate (see Sec. IV for additional details), pre_t is a dummy variable equal to one if year t was prior to eradication, X'_{ij} is a vector of individual-level controls, δ_j are region fixed effects, δ_t are year fixed effects, and $\gamma_j t$ are region-specific linear trends. I define $\text{pre}_t = 1$ for $t \leq 1947$, following Lucas (2010). The eradication campaign reached nationwide coverage in 1947, and 1948 was the first full year of national coverage. Thus, for $t > 1948$, $\text{pre}_t = 0$. I include region-specific linear trends to control for the possibility of regions being on different paths, that is, converging, in the absence of the eradication program. In this specification, β is the effect of malaria on the probability of a live birth net of these controls.

Because malaria symptoms are more severe for women pregnant for the first time, any change in fertility could be differential by parity. To estimate this birth order effect, I estimate equation (1) as a hazard model separately for first births and for second births.

As with other differences-in-differences specifications, the possibility of serial correlation makes an error correction method necessary. The typical cluster-robust solution suggested by Bertrand, Duflo, and Mullainathan (2004) could lead to inappropriately small standard errors because of the small number of regional categories available in the WFS data. I present these standard errors and calculate the p-values following Cameron, Gelbach, and Miller's (2008) wild cluster bootstrap-T method in all estimations.

In addition to differentially changing fertility timing by parity, the direct biological effects of malaria on pregnant women could differentially affect survival by birth order, with first births having the worst outcomes. I estimate, separately by birth order, the effect of malaria in the year of birth on survival to age 1 and age 5:

$$\text{survival}_{bijt} = \alpha + \beta(\text{malaria}_j \times \text{pre}_t) + X'_{bij}\Gamma + \delta_j + \delta_t + \gamma_j t + \varepsilon_{bijt}, \qquad (2)$$

where survival_{bijt} is the survival of birth b born to woman i in region j in year t and X'_{bij} are birth-specific covariates; other notation and error correction are as in equation (1). In this specification, β is the effect of malaria on survival.

In all empirical specifications, the estimates of the effect of malaria rely on both spatial and temporal variation for identification. First, even though the eradication was national in scope, regions with high preeradication malaria levels benefited relatively more from the eradication campaign than those regions with historically low malaria levels. The effective intensity of the program is the change in the malaria rates from the eradication program (i.e., the preeradication malaria level, since all malaria levels were reduced to zero). Second, the timing of the eradication campaign resulted in higher malaria levels prior to 1948, the first full year of the nationwide malaria eradication campaign.

The key assumption in the identification strategy is that in the absence of the eradication program there were no changes in fertility or survival concurrent to the eradication campaign and correlated with the preeradication malaria intensity. For example, an eradication campaign that targeted regions in order to increase child survival or change fertility would violate this assumption. According to Wickremesinghe (1953), DDT spraying was based on malaria levels, not other regional attributes. A second concern could be that the regions that benefited the most from malaria eradication would have converged toward the less malarious regions even in the absence of the campaign. In consideration of this possibility, I include regional trends in all specifications to control for the potential of preexisting regional convergence. By also including region and year fixed effects, the estimates of the malaria effect are net of any time-invariant differences between regions, uniform nationwide changes in outcomes, or underlying convergence.

IV. Data

To undertake this analysis, I combine individual-level survey data with preeradication regional malaria levels. The individual survey data are from the WFS conducted in 1975 (Department of Census and Statistics 1975). The survey was designed to be a nationally representative survey of ever-married women aged 12–50.[10] In order to estimate the probability of a live birth in a given year, I transform the retrospective fertility histories into a panel with one observation for each woman for each year from age 13 to the time of the survey, including an indicator for the years in which a live birth occurred.[11] The sample of 6,810 women had 20,911 live births at least 5 years prior to the survey. For the survival analysis, I used the same retrospective fertility histories

[10] The oldest women included in the sample were born in 1925. Ideally, survey data would be available that included women with earlier birth dates. The statistical significance of some of the point estimates in Sec. V could be affected by the small sample size in the preeradication period.
[11] Retrospective fertility histories can suffer from recall bias, which in this case should not be related to the malaria level. Because the identification strategy relies on variation from both year and geographic location, recall bias from only one source of variation (e.g., older respondents are more likely to suffer from recall bias) would not be sufficient to invalidate identification. Recall errors systematically related to the level of malaria and period of recall could bias the results. Nationwide contemporaneous fertility surveys, an alternative source for fertility estimates, are not available for the time period under study in Sri Lanka.

that included information on the age of death of each live birth to estimate the effect of malaria on survival of live births to ages 1 and 5. To compare the same cohorts for survival to ages 1 and 5, I limit the sample to include only live births that occurred at least 5 years prior to the survey.

The preeradication malaria levels are 1937 malaria spleen rates reported for each district in Newman (1965). District-level spleen rates were the percentage of school children with enlarged spleens, a measure of long-standing malaria, and were used as a convenient measure of malaria prevalence before the wide availability of alternative low-cost tests. Since adults living in areas with continual malaria transmission can develop acquired immunity, a high malaria spleen rate, indicating continual reinfection of a population, could signal some acquired immunity in the adult population. Nonpregnant adults with such immunity exhibit very mild or no malaria symptoms even when infected. Pregnant women, even those who had acquired immunity prior to becoming pregnant, are subject to more virulent malaria symptoms than the nonpregnant population. Therefore, a region with a high spleen rate indicates a higher probability of symptomatic infection among pregnant women than in an area with lower or zero spleen rates (Recker et al. 2009). I aggregate the district-level spleen rates to the regional level, the smallest level of geographic designation available in the WFS data. The six mutually exclusive regional designations are Colombo, Colombo Feeder, South Western Coastal Low Lands, South Central Hills, Irrigated Dry Zone, and Rain Fed Dry Zone.

V. Results

To estimate the effects of malaria on fertility, I use several specifications. The estimates of the effect of malaria on the probability of birth in a given year address the magnitude and direction of the total malaria effect. Estimates of hazard models and child survival by parity distinguish one possible mechanism driving this change in fertility.

Table 1 and figure 3 provide prima facie evidence of a limited malaria effect. In table 1, each region is classified as either "highly malarious" or "less malarious" based on preeradication malaria prevalence, with three regions in each classification. The means contained in the table are calculated for each group, separately for the preeradication (1938–47) and posteradication (1948–75) years. Square brackets contain the number of observations in each cell. The standard error, clustered at the regional level, from a simple regression that replicates each panel of the table, is presented in parentheses below the difference-in-differences estimate. Concurrent to malaria eradication, regions with the highest levels of malaria had similar changes to fertility as the less

TABLE 1

DIFFERENCES IN MEANS

	Preeradication (1)	Eradication (2)	Increase (3)
A. Probability of birth:			
Highly malarious	.1009	.2020	.1011
	[4,687]	[80,224]	
Less malarious	.0674	.1689	.1015
	[3,918]	[58,203]	
Difference-in-differences			-.00045
			(.022)
B. Probability of survival until age 1:			
Highly malarious	.863	.934	.071
	[489]	[12,575]	
Less malarious	.900	.941	.041
	[270]	[7,577]	
Difference-in-differences			.030
			(.019)
C. Probability of survival until age 1, first births only:			
Highly malarious	.856	.923	.067
	[285]	[2,677]	
Less malarious	.920	.935	.015
	[163]	[1,825]	
Difference-in-differences			.052
			(.037)
D. Probability of survival until age 5:			
Highly malarious	.800	.901	.102
	[489]	[12,575]	
Less malarious	.852	.914	.062
	[270]	[7,577]	
Difference-in-differences			.040
			(.021)
E. Probability of survival until age 5, first births only:			
Highly malarious	.793	.894	.101
	[285]	[2,677]	
Less malarious	.883	.914	.031
	[163]	[1,825]	
Difference-in-differences			.070
			(.055)

Note. Preeradication years are 1938–47. Eradication years are 1948–75. Highly malarious regions are South Central Hills, Irrigated Dry Zone, and Rain Fed Dry Zone. Less malarious regions are Colombo, Colombo Feeder, and South Western Coastal Low Lands. In panel A, the number of woman years are presented in square brackets. Because the number of women of each age are not the same in the preeradication and posteradication periods, the change in fertility cannot be interpreted as a change in age-specific fertility. In panels B–E, the number of live births are presented in square brackets. Sample is limited to live births at least 5 years prior to the survey. Standard errors clustered at the regional level, shown in parentheses, are from a simple regression that replicates the table.

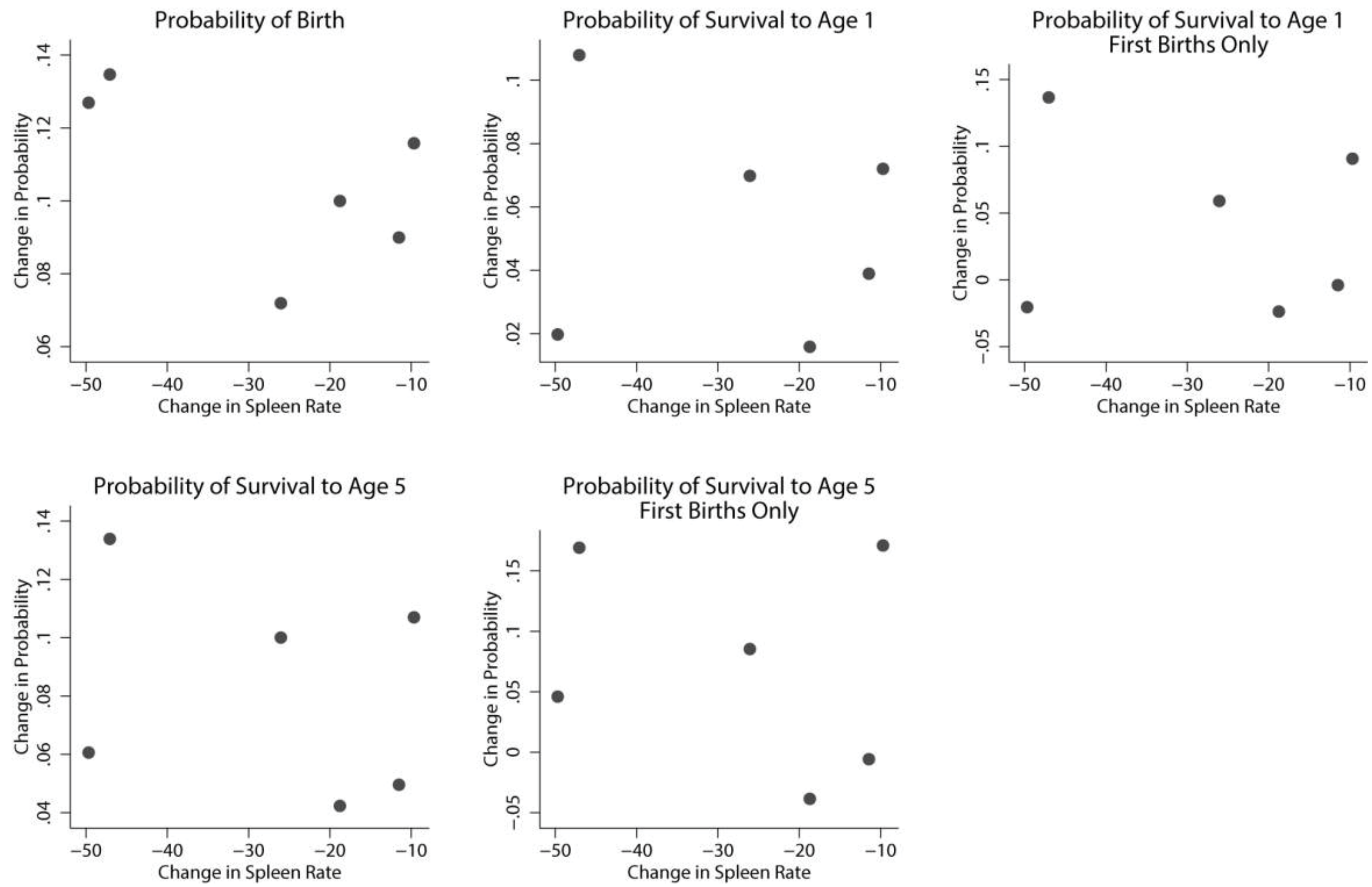

Figure 3. Differences in outcomes by region and eradication period. Change in spleen rate is the percentage point change in the spleen rate from 1937 to complete eradication. The changes in probabilities compare outcomes from 1938–47 (preeradication) to those from 1948−75 (posteradication). Each dot represents one region. Calculations are based on Newman (1965) and World Fertility Survey data.

malarious regions (panel A) but did have the largest increases in survival probabilities (panels B–E).[12] In all cases the difference-in-differences estimates are imprecisely measured.

These crude aggregations could mask important differences between regions in the same group. Within the highly malarious grouping, the spleen rate in 1937 was between 26% and 50%, while in the less malarious grouping the rate was between 10% and 19%. The regression analysis discussed below allows a more refined analysis.

Figure 3 contains the plots of the same outcomes as in table 1, disaggregated to the regional level. Each plot demonstrates the negative relationship between malaria and the probability of birth and survival of live births.[13]

Figure 4 displays the year-specific coefficients from a less restrictive version of equation (1) with the probability of a live birth as the dependent variable, including a separate interaction term between the preeradication regional malaria level and each year instead of $\beta(\text{malaria}_j \times \text{pre}_t)$. The coefficients on the interaction terms of the years prior to 1948 are insignificantly different from the 1938 omitted year, with the exception of 1947. After 1948, all of the coefficients on the interaction terms are positive and significant, indicating an increase in the probability of a live birth in the most infected regions after malaria eradication. The statistical significance in the years after 1948 bolsters the choice of 1948 as the first posteradication year.

Column 1 of table 2 contains the coefficients from the estimates of equation (1) as a linear probability model. Cluster-robust standard errors are presented in parentheses, and the two tailed p-values associated with the wild cluster bootstrap-T method appear in square brackets. The eradication of malaria increased the probability that a woman would have a birth in a particular year, controlling for regional trends and nationwide changes in fertility. The highest regional malaria spleen rate in the sample was 49.7% in the Rain Fed Dry Zone. In the absence of other nationwide changes in fertility patterns or regional convergence, eradication of malaria from this level would have increased the probability of a live birth in a particular year by 11 percentage points,

[12] Because the age structure of the sample changes between the pre- and posteradication periods, the increase in fertility between the two periods in panel A cannot be interpreted as a change in age specific fertility.

[13] As can be seen in the third plot in fig. 3 ("Probability of Survival to Age 1, First Births Only"), the probability of survival decreased in the region with the largest change in the spleen rate, the Rain Fed Dry Zone. This decline is not statistically different from zero (p-value = .65) and is at least in part due to the small preeradication sample of first births in this region. In the preeradication period, 43 live first births occurred, and 40 survived to age 1 (93.0%). Had one fewer survived (90.7% survival rate), the survival probability in this region would have increased between the two periods.

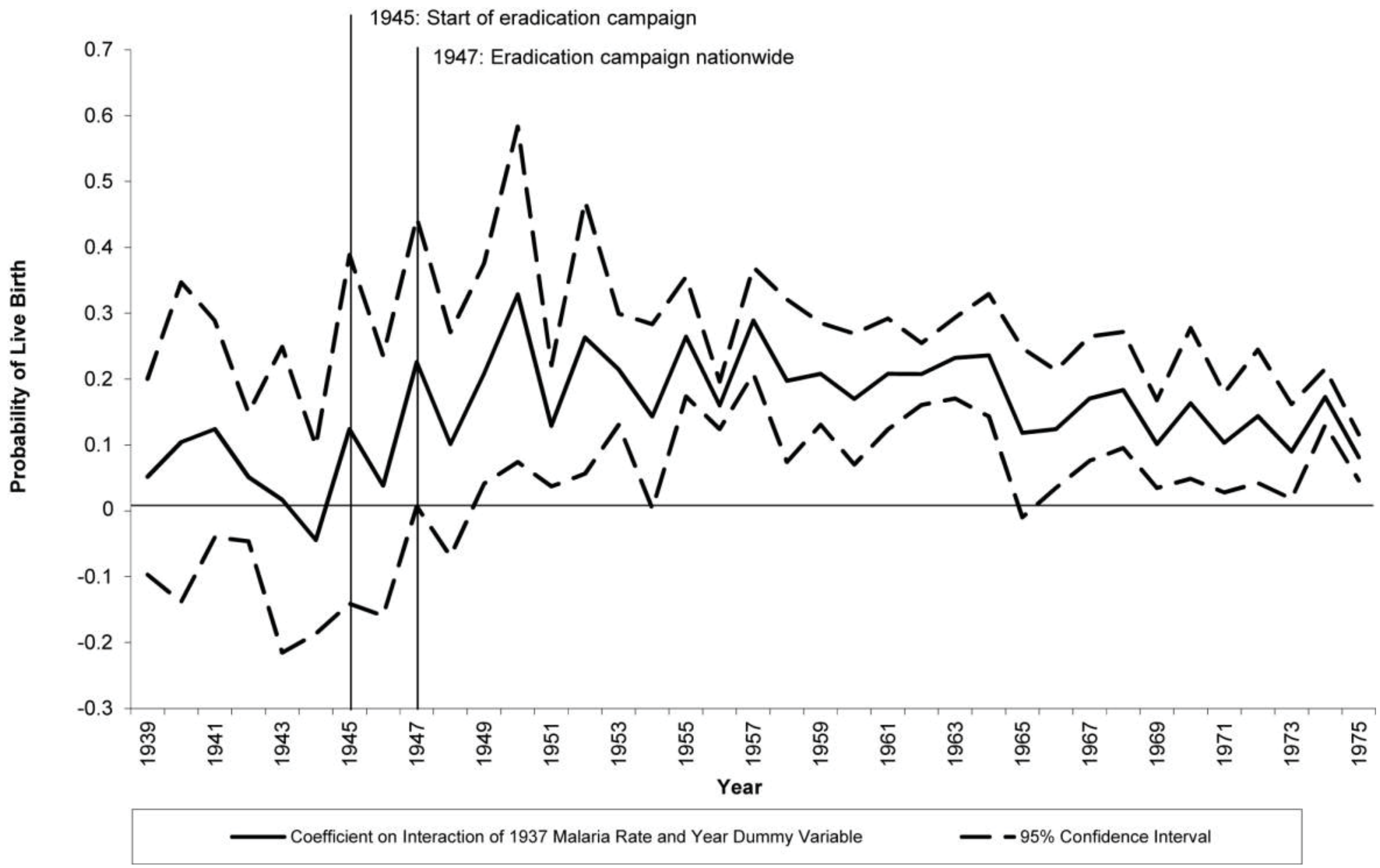

Figure 4. Timing of the malaria effect

TABLE 2
FERTILITY

		Hazard of Birth		
		First Birth		
	Probability of Birth (1)	All Women (2)	Women with at Least One Live Birth (3)	Second Birth (4)
Preeradication × 1937 malaria rate	−.223***	−.180***	−.177***	−.136*
	(.052)	(.023)	(.024)	(.060)
	[.187]	[.056]	[.123]	[.235]
Additional covariates:				
Current residence (omitted = urban)				
Rural residence	.015	.007	.008	.002
	(.009)	(.006)	(.006)	(.005)
Estate residence	−.029	−.015	.004	−.0453**
	(.015)	(.020)	(.017)	(.015)
Ethnicity (omitted = Sinhala):				
Sri Lanka Tamil	.005	.009	.013	−.020
	(.014)	(.016)	(.013)	(.014)
Indian Tamil	.0306**	.036	.0356**	−.022
	(.008)	(.018)	(.013)	(.019)
Sri Lanka Moor	.0365***	.0330*	.0426**	.013
	(.008)	(.013)	(.016)	(.025)
Other ethnicity	.0259**	.014	.0325***	.069
	(.010)	(.015)	(.004)	(.045)
Observations	147,031	62,948	54,206	16,793
R^2	.07	.05	.11	.05

Note. Standard errors clustered at the regional level appear in parentheses. Two-tailed *p*-values associated with the wild cluster bootstrap-*T* method appear in square brackets. The unit of observation is a woman-year, starting at the age of 13 for all columns. All columns are linear probability models and include age, region, and year fixed effects and region-specific linear trends. Cols. 2–4 are hazard models.
* Significant at the 10% level.
** Significant at the 5% level.
*** Significant at the 1% level.

approximately doubling the preeradication probability of a live birth in that zone. Within the entire sample, the median level of regional malaria for a woman-year in the preeradication period was 26.1%. Eradication of malaria from this level would increase the probability of a life birth by 5.8 percentage points. While sizable, the total change in fertility over this period would be the effect of malaria plus other nationwide and regional changes in fertility unrelated to eradication and controlled for with time fixed effects and regional trends. For example, in the Rain Fed Dry Zone, after taking into account year fixed effects and regional trends, the net increase in the probability of a live birth between 1945 and 1955 would be 3.4 percentage points. An increase in fertility is consistent with a change in preferences or a biological change in fecundity.

Because of the differential effect of malaria on first pregnancies, a purely biological response could decrease the transition time to a first live birth with less of an effect on subsequent transition times. To test if biology dominates preference-based changes in fertility, I estimate equation (1) as a hazard model, where the hazard of having a live birth starts at age 13.[14] The results from the estimation for all women appear in table 2, column 2. Malaria exerted a negative and significant effect on the transition to initial parity, decreasing the probability of transitioning to having had a live birth by 8.9 percentage points if the malaria spleen rate was at its highest level in the sample compared to complete eradication. The sample in column 3 is limited to those with at least one live birth to ensure that the difference between the results by parity is not being driven by sample selection. The result for those women with at least one live birth is statistically indistinguishable from the full sample estimation with a point estimate of smaller absolute magnitude.

This increase in fertility from eradication could be due to the elimination of the biological constraint or a preference- or income-induced change in fertility. Because of the epidemiology of malaria, the biological effects should dissipate with higher-order pregnancies while other changes might not.

Malaria levels had a weaker effect on the transition to a second live birth (table 2, col. 4).[15] The point estimate is smaller in absolute magnitude and statistically significant at the 10% level when standard errors are clustered at the regional level, indicating that both direct and indirect effects of malaria on pregnancy could be important determinants of the change in fertility. Based on the estimates in columns 2 and 4, malaria suppressed the total fertility rate by approximately 25% in the preeradication period.[16]

The sign and statistical significance of the finding in table 2, column 4, is not robust to the specification checks presented in table 4. The point estimate is positive, instead of negative, and it is insignificant when the posteradication sample is limited to the first 10 years after the nationwide eradication program, a modification under which the other estimates in table 2 are robust. The coefficient fails to be statistically significant across the other robustness checks.

[14] Formally, I estimate eq. (1) as a linear probability model in which a woman appears in the sample from age 13 until the year of the first birth. This is equivalent to estimating a discrete time proportional hazard model (Allison 1984).

[15] The earliest higher-order birth occurred in 1940.

[16] Formally, I use the estimates in table 2, col. 2, prior to the first birth and the estimates in col. 4 for subsequent births to calculate an age-specific fertility rate without malaria. The malaria-free total fertility rate is the sum of the average age-specific fertility rates. This is compared to the actual total fertility rate over the sample, the sum of the true age-specific fertility rates.

Because of the change in sign and significance, the second birth result does not conclusively support or refute the importance of biology over other latent causes for fertility changes. Malaria had a negative effect on the transition to initial parity, with a less clear effect on the transition to higher-order parity.[17] Because of the high degree of uncertainty regarding the effect on the timing of the second birth, the relative importance of biology versus preferences is unresolved. The findings are consistent with both a preference-based and biological change in fertility. Any shift in preferences should not result in parity-specific changes in the survival of live births.

An additional test of the parity-specific effects of malaria is to estimate the effect of malaria on child survival. If malaria had been delaying the initial live birth through an increase in spontaneous abortions and still births, then the other symptoms of malaria in pregnancy that are differential by birth order (i.e., prematurity and low birth weight) should also have been present. Therefore, if the parity-specific results were being driven by the direct effects of malaria infections, then there should be differential survival probabilities by birth order. Table 3 contains the estimates of equation (2) as a linear probability model, separately by birth order and survival from birth to age 1 and from birth to age 5.[18] Surprisingly, the effect of malaria on survival to age 1 for all births, first births, and second births is not statistically significant. One possible explanation is that the malaria parasite prevalent in Sri Lanka is *Plasmodium vivax*, a milder strain of malaria than the one that is the most common in Africa. Additionally, many other health insults can occur in the first year of life, unrelated to malaria, that could confound finding a malaria effect. Also, selective fertility in the preeradication period through which only fetuses with high health endowments resulted in a live birth or only healthier women had live births could contribute to this result. Finally, the lack of a significant finding could be because of the small sample size in the preeradication period.

Compared to various observational studies in the public health literature that attribute 3%–30% of infant mortality in malaria-endemic areas to malaria, the point estimate for all births, while imprecisely estimated, is within this range (Greenwood et al. 1992; Brabin and Piper 1997; Steketee et al.

[17] Survival of the first born could mechanically delay a second birth due to breastfeeding. Therefore, the estimated effect could reflect the change in survival probabilities. When survival of the first birth is an additional covariate, the point estimate of the coefficient is -0.175, with a standard error of 0.081. As with the baseline specification in table 2, col. 4, this finding is not robust to the alternative specifications in table 4.

[18] Each pregnancy that resulted in a live birth is counted as one. For example, if the first birth resulted in twins, then both twins would be considered in the first birth estimations. The next live birth to the same respondent would be considered the second birth.

TABLE 3
SURVIVAL

	Survival until Age 1			Survival until Age 5		
	All Births (1)	First Births (2)	Second Births (3)	All Births (4)	First Births (5)	Second Births (6)
Preeradication × 1937 malaria rate	−.060	−.314	.123	−.145	−.4482**	.032
	(.090)	(.167)	(.154)	(.078)	(.148)	(.155)
	[.594]	[.594]	[.594]	[.356]	[.291]	[.851]
Additional covariates:						
Multiple birth	−.255***	−.297***	−.276**	−.2417***	−.2910***	−.2473**
	(.044)	(.040)	(.080)	(.044)	(.029)	(.079)
Sex:						
Female	.0121***	.0279***	−.000284	.0091***	.0238***	−.0025
	(.003)	(.004)	(.008)	(.002)	(.006)	(.005)
Unknown	.0466***			.0747***		
	(.008)			(.006)		
Current residence (omitted = urban residence):						
Rural residence	−.000863	−.0307**	.00898	−.0286***	−.0428***	.0029
	(.005)	(.009)	(.005)	(.006)	(.004)	(.009)
Estate residence	−.00572	−.0416***	.00959	−.0505**	−.0509***	−.0201
	(.012)	(.010)	(.015)	(.016)	(.008)	(.020)
Ethnicity (omitted = Sinhala):						
Sri Lanka Tamil	−.009	−.007	−.019	−.018	−.019	−.002
	(.009)	(.015)	(.026)	(.015)	(.026)	(.015)
Indian Tamil	−.0753***	−.0743**	−.0493***	−.0555**	−.0493***	−.0628**
	(.012)	(.020)	(.007)	(.016)	(.007)	(.020)
Sri Lanka Moor	−.019	−.007	−.010	−.016	−.010	−.005
	(.011)	(.007)	(.017)	(.017)	(.017)	(.008)
Other ethnicity	−.021	.035	.007	−.008	.007	.0705*
	(.034)	(.022)	(.034)	(.032)	(.034)	(.028)
Observations	20,911	4,950	4,185	20,911	4,950	4,185
R^2	.04	.05	.04	.03	.05	.04

Note. Standard errors clustered at the regional level appear in parentheses. Two-tailed *p*-values associated with the wild cluster bootstrap-*T* method appear in square brackets. The unit of observation is a live birth. All columns include region and year of birth fixed effects and region-specific time trends. Cols. 1 and 4 include fixed effects for the number of prior pregnancies.
* Significant at the 10% level.
** Significant at the 5% level.
*** Significant at the 1% level.

2001). The point estimate on first births, again imprecisely estimated, implies a higher burden of mortality than found in the other studies, which could be explained by the estimate's imprecision or the other studies not focusing on first births.

Based on the estimates for survival from birth until age 5 (table 3, cols. 4–6), malaria negatively affected survival probabilities for first births but not second births. The difference in results between the first birth and all births suggests that malaria's effect on survival was most likely operating through the direct prenatal health effects, since once an infant is born the direct effect of malaria exposure should not vary by birth order. This differential result by birth order adds additional evidence to support the likelihood that the biological effect was an important contributor to the fertility increase.

VI. Robustness

A. Specification Checks

Tables 4 and 5 present several specification checks of the fertility and survival results. One potential concern with the identification strategy is that women who have live births in the preeradication period are somehow different than women whose fertility occurs only after eradication, even after controlling for age. The maternal fixed effects models in column 2 of both tables control for any time-invariant differences between women. In the survival specification, the inclusion of these fixed effects results in a negative and significant estimation of the effect of malaria on survival until age 5 for all births. This estimate is only 40% of the magnitude of the first birth baseline result. When the sample is limited to the 10 years before and 10 years after eradication (1938–58, col. 3), all fertility results except the hazard of second birth are similar to the baseline. This specification results in a positive and insignificant point estimate for the hazard of a second birth. The loss in precision could be due to the decrease in the sample size. The change in the sign of the effect casts doubt on the initial result. The survival results are of similar magnitude to the baseline, with some loss of precision with the decrease in the sample size.

Tables 4 and 5 column 4 limits the sample to those women (and their live births) who have always lived in the same locality (i.e., a nonmover sample). Because of limitations with the survey questionnaire, in the main specifications I assigned women and live births to their region of residence, which could be different from the region in which they gave birth or were born. Column 4 limits the sample to women who have " always lived in the same locality." The similarity of these results to the full sample reduces concern about selective migration. As with the other robustness checks, the hazard of a second birth is no longer statistically significant.

Malaria transmission continued for a number of years after the nationwide malaria eradication campaign (fig. 2). In the main estimations, all years after 1947 are classified as posteradication, potentially misclassifing the years in which malaria transmission continued. Tables 4 and 5 column 5 removes the observations for the first 5 years after the start of the eradication campaign (1948–52) from the sample. The point estimates and significance levels are similar to the baseline, with the exception of the hazard of the second birth, which is not statistically different from zero.

TABLE 4
FERTILITY ROBUSTNESS

Dependent Variable	Baseline (Table 2) (1)	Maternal Fixed Effects (2)	Years 1938–58 Only (3)	Nonmovers (4)	Excluding Early Eradication, Years 1948–52 (5)	Fake Intervention, 1943 (1938–53 Only) (6)	Fake Intervention, 1958 (1948–68 Only) (7)
Probability of birth:							
Preeradication × 1937 malaria rate	−.223***	−.189**	−.115***	−.199**	−.251***	−.046	.016
	(.052)	(.052)	(.027)	(.061)	(.059)	(.030)	(.036)
Observations	147,031	147,031	42,593	67,773	135,092	23,657	90,943
R^2	.066	.065	.085	.063	.065	.090	.065
Hazard of first birth:							
Preeradication × 1937 malaria rate	−.180***		−.132***	−.169***	−.175***	.030	−.0488*
	(.023)		(.018)	(.033)	(.031)	(.035)	(.019)
Observations	62,948		27,934	28,697	54,731	17,787	44,476
R^2	.054		.046	.053	.055	.049	.041
Hazard of first birth (women with at least one live birth):							
Preeradication × 1937 malaria rate	−.177***		−.139***	−.136*	−.166***	.040	−.0443*
	(.024)		(.017)	(.053)	(.036)	(.034)	(.017)
Observations	54,206		26,440	24,211	46,392	16,999	39,944
R^2	.111		.052	.114	.119	.054	.052
Hazard of second birth:							
Preeradication × 1937 malaria rate	−.136*		.075	−.078	−.126	.248	.038
	(.060)		(.056)	(.128)	(.063)	(.357)	(.069)
Observations	16,793		5,056	8,116	15,793	2,678	10,659
R^2	.047		.018	.058	.049	.026	.030

Note. Standard errors clustered at the regional level appear in parentheses. The unit of observation is a woman-year. All columns include region, age, and year fixed effects and region-specific time trends. Cols. 1 and 3–7 contain the coefficient estimates of four regressions. Col. 2 contains one regression and includes maternal fixed effects. Col. 3 limits the sample to the 10 years before and 10 years after eradication. Col. 4 limits the sample to women who have always lived in the same locality. Col. 5 excludes 1948–52, the first 5 years after eradication. Cols. 6 and 7 estimate the effect of fake eradication campaigns at dates when no eradication campaigns occurred.

* Significant at the 10% level.

** Significant at the 5% level.

*** Significant at the 1% level.

TABLE 5

SURVIVAL ROBUSTNESS

Dependent Variable	Baseline (Table 2) (1)	Maternal Fixed Effects (2)	Years 1938–58 Only (3)	Nonmovers (4)	Excluding Early Eradication, Years 1948–52 (5)	Fake Intervention, 1943 (1938–53 Only) (6)	Fake Intervention, 1958 (1948–68 Only) (7)
Survival to age 1, all births:							
Preeradication × 1937 malaria rate	−.060	−.077	−.101	.032	−.051	−.167	.006
	(.090)	(.079)	(.090)	(.116)	(.085)	(.399)	(.024)
Observations	20,911	20,911	7,281	9,620	19,681	3,443	17,931
R^2	.04	.03	.06	.04	.04	.03	.04
Survival to age 1, first births:							
Preeradication × 1937 malaria rate	−.314		−.239	−.375	−.365*	−.011	.005
	(.167)		(.211)	(.287)	(.162)	(.173)	(.046)
Observations	4,950		2,320	2,301	4,476	1,401	4,062
R^2	.05		.05	.08	.05	.05	.04
Survival to age 1, second births:							
Preeradication × 1937 malaria rate	.123		.249	.227	.058	.374	.058
	(.154)		(.168)	(.353)	(.156)	(1.592)	(.109)
Observations	4,185		1,781	1,921	3,833	972	3,576
R^2	.04		.13	.07	.05	.07	.04

Survival to age 5, all births:							
Preeradication × 1937 malaria rate	−.145	−.185**	−.204*	−.067	−.109	−.392	−.008
	(.078)	(.066)	(.100)	(.130)	(.075)	(.287)	(.038)
Observations	20,911	20,911	7,281	9,620	18,824	3,443	17,931
R^2	.03	.02	.04	.04	.03	.03	.03
Survival to age 5, first births:							
Preeradication × 1937 malaria rate	−.4482**		−.414	−.518	−.523**	−.004	−.144*
	(.148)		(.247)	(.336)	(.153)	(.307)	(.061)
Observations	4,950		2,320	2,301	4,165	1,401	4,062
R^2	.05		.05	.08	.05	.05	.03
Survival to age 5, second births:							
Preeradication × 1937 malaria rate	.032		.189	−.072	−.059	−.239	.066
	(.155)		(.187)	(.279)	(.114)	(1.278)	(.137)
Observations	4,185		1,781	1,921	3,589	972	3,576
R^2	.04		.09	.07	.04	.07	.03

Note. Standard errors clustered at the regional level appear in parentheses. The unit of observation is a live birth. All columns include region and year of birth fixed effects and region-specific time trends. "All births" specifications include fixed effects for the number of prior pregnancies. Cols. 1 and 3–7 contain the coefficient estimates of six regressions. Col. 2 contains two regressions and includes maternal fixed effects. Col. 3 limits the sample to the 10 years before and 10 years after eradication. Col. 4 limits the sample to women who have always lived in the same locality. Col. 5 excludes 1948–52, the first 5 years after eradication. Cols. 6 and 7 estimate the effect of fake eradication campaigns at dates when no eradication campaigns occurred.

* Significant at the 10% level.

** Significant at the 5% level.

Tables 4 and 5 columns 6 and 7 assign placebo treatments separately prior to and after the actual eradication campaign, times in which there was not a malaria eradication campaign. As expected, these fake assignment rules do not have the strength of the baseline findings. The exceptions are the effect of the 1958 fake campaign on transition to first birth and survival to age 5 of first births (col. 7). The point estimates are at most one-third of the baseline, and they are significant only at the 10% level. While much lower than prior to 1948, the spleen rate was still positive from 1948 to 1955. Therefore, this fake intervention was during a time of falling malaria.

B. *Additional Threats to Validity*

Concurrent health or nutrition programs that targeted the most heavily infected regions could lead to biased estimates of the malaria effect.[19] Through year fixed effects, I control for any nationwide changes in each year that affected all regions in a uniform way. Any time-invariant differences among regions are controlled for with regional fixed effects. Region-specific linear trends control for underlying convergence (or divergence) among regions. Therefore, in order to invalidate the estimation strategy, any program would have to differentially affect regions in a way that was both correlated with preeradication malaria and the timing of the eradication program.

Prior to eradication, public health availability in the highly malarial regions was equal or superior to that in the low malaria regions, as measured by the population per hospital, the population per hospital bed, the population-adjusted admission rates, and the coverage of the central dispensaries with inpatient care. "There is no evidence for an unbalanced improvement in medical services" in the period after 1945 (Gray 1974).[20] Furthermore, there were not differential changes in smallpox vaccination rates (Langford 1996). Continual health improvements uniformly applied nationwide would not bias the results.

Similarly, nutritional intake in the preeradication period, as measured by daily consumption of calories, carbohydrates, protein, and minerals and the lower levels of malnutrition, was superior in the villages of the highly malarious regions (various studies as summarized in Gray [1974]). Based on more limited data, nutrition in Colombo does not appear to be superior to

[19] This section draws on similar literature to that in Lucas (2010).

[20] Unfortunately, during the relevant period I am not aware of information on district-level health service provision. Other authors have used maternal mortality as a proxy for improvement in health service provision, but without additional data, the relative importance of malaria eradication versus improved health services cannot be estimated.

that available in the highly malarious regions. The late 1950s shift in the Dry Zone away from production of agricultural products for consumption to a wage-based labor structure led to a deterioration of nutritional intake. Finally, the introduction of high-yield-variety (HYV) rice to Sri Lanka was of relatively small magnitude into the early 1970s (2.5% of rice seed was HYV in 1973), and it did not lead to differential increases in income correlated with malaria reduction (Brown 1970; Pearse 1980).

VII. Discussion and Conclusions

Using the malaria eradication campaign in Sri Lanka as a natural experiment, I find that malaria eradication increased fertility. Economic theory suggests a number of reasons why disease eradication could increase fertility. Because malaria infections are more detrimental to first pregnancies than subsequent pregnancies, through an analysis of birth timing by birth order, I find that the source of this increase in fertility is most likely an elimination of a previously binding biological constraint but it could be from a change in preferences. I confirm that these more serious sequelae for first pregnancies also affect offspring survival probabilities by birth order, with the first born experiencing the largest increases in survival.

Fertility increases can cause a reduction in GDP per capita as the size of the nonproductive segment of the population increases. Despite the increase in cohort size, in Lucas (2010), I estimated that malaria eradication increased female educational attainment by as much as 2 years in the most heavily infected region, based on estimates from the same eradication episode in Sri Lanka. Eventually these educated individuals will enter their productive years. The net effect on GDP per capita of the education and fertility effect should be positive, but it will not be realized immediately. The duration of the transitory population increase reconciles the two contradictory views in the growth and microeconomic literatures of the relative importance of health for GDP per capita and GDP per capita growth. The relative sizes of the initial increase in population and the increase in education will determine the duration of a potential decrease or stagnation in GDP per capita.[21]

[21] Barlow (1968) used a simulation to estimate the effect of malaria eradication on the Sri Lankan economy. Based on his simulation, eradication caused the real gross national income (GNI) per equivalent consumer to increase in the 10 years after eradication, fall for 10 years, and be below the noneradication real GNI 20 years after eradication. The effect of increased fertility swamped the positive effects of increased size and quality of the labor force as the larger cohorts entered into the calculation of effective consumers. Barlow used malaria-induced increases in fertility based on the crude birth rate (effectively including compositional effects and changes in fertility).

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