The Impact of Malaria Eradication on Fertility and Education*

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Abstract

From 1935 to 1963 a malaria eradication campaign in Sri Lanka reduced incidence from 97% of the population to 17 cases. This paper combines this exogenous malaria eradication campaign and the pre-existing heterogenous malaria levels in Sri Lanka with two household surveys to identify the effect of malaria eradication on fertility, survival, and human capital accumulation across two successive generations. Contrary to theories of the demographic transition, a movement along the quantity-quality trade-off, and sequential or replacement fertility, the initial effect of malaria eradication was an *increase* in fertility. To separate the direct health effects from other potential causes of the increase in fertility, I exploit the particular epidemiology of malaria: the symptoms are more severe for primigravidae, women pregnant for the first time, than for multigravidae, women of higher order parity. Since malaria eradication induced a larger increase in survival probabilities for first born children and quickened the transition to initial parity while the transition time to higher order parity did not change, I conclude that the source of the increase in fertility was the elimination of the biological constraint. In the second generation, those born after eradication in the previously most heavily infected regions accumulate more human capital as measured by years of education or literacy. They also have lower fertility. Therefore, while the initial population growth might be detrimental to GDP per capita, increased education and lower subsequent fertility can mitigate this initial negative growth effect.

Keywords: malaria, fertility, human capital

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1 Introduction

Malaria is endemic in ninety-one countries. Almost 40% of the world's population is at risk for malaria infection, and the disease infects more than 300 million people annually. In Africa, malaria accounts for 10% of the overall disease burden, 40% of the public health expenditure, and 30 to 50% of inpatient admissions [Roll Back Malaria (2005)]. The effects of malaria on the quality of life and economic growth and development in sub-Saharan Africa have recently received renewed attention from international organizations. The reduction of malaria is one of the United Nations Millennium Development Goals and a large scale eradication program funded by the Gates Foundation is ongoing in Zambia. The Roll Back Malaria Program and the Malaria Vaccine Initiative are also focusing on the reduction of the malaria disease burden in Africa. Understanding the effect malaria has on economic growth and development is crucial for policy evaluation and identifying the sources of tropical underdevelopment.

Those at the highest risk for adverse outcomes from malaria infections are those with the weakest immune responses: pregnant women and children. This paper will focus on these two populations during the Sri Lanka malaria eradication campaign, exploring the relationship between the changing disease environment and fertility of those of childbearing age during the eradication campaign and the survival, eventual educational attainment, and fertility of their offspring.

In order to identify the multigenerational effects of malaria (and malaria eradication) I rely on the first wave of international interest in malaria eradication: the WHO malaria eradication program that followed the Second World War. I use the malaria eradication campaign in Sri Lanka to estimate the effect of malaria (or malaria eradication) on fertility, child survival, and lifetime human capital accumulation across two successive generations. I combine data from two separate national surveys (the World Fertility Survey and the Demographic and Health Surveys) with measures of sub-national malaria incidence. The source of identification is the heterogeneity in indigenous malaria rates within Sri Lanka based on climatic and geographic factors and the exogenous elimination of malaria during the national malaria eradication campaign. This identification strategy isolates the malaria effect from other nationwide trends and regional fixed effects.

I find that for those of childbearing age around the time of malaria eradication, the fall in malaria caused an *increase* in fertility, an increase in the probability of survival of their first born offspring, and a lower age of first birth. For those born after eradication, educational attainment was higher and their fertility was lower than those born prior to eradication. Therefore, while there

are positive effects of malaria eradication on educational attainment, there will be a lag between eradication and an increase in GDP per capita because of the initial population increase.

The remainder of this paper is organized as follows: Section 2 provides background on malaria generally and specific to Sri Lanka, Section 3 addresses the competing theoretical predictions about fertility, child survival, and education with malaria reduction, Section 4 contains the identification strategy, Section 5 contains the estimates of the effect of malaria on the outcomes of interest, Section 6 allows for alternative hypotheses, and discussions and conclusions are contained in Section 7.

2 Background

2.1 Epidemiology of Malaria

Malaria is a parasitic disease transmitted by female *Anopheles* mosquitoes. Certain climatic and geographic conditions are necessary for vector reproduction and parasite transformation and transmission. Broadly, harsher winters and colder temperatures are less hospitable for the vector and the *Plasmodium*. Transmission rates are the highest with temperatures above 64^0 F (18^0 C) and no parasite incubation can occur at temperatures below 60^0 F (16^0 C). A minimum amount of rainfall is also necessary to provide the standing water essential for vector breeding, but too much rainfall (100 inches or more) can eliminate suitable breeding sites. At altitudes above 3281 ft. (1000 m.) there is at most minimal malaria incidence. These environmental and geographical limitations of the mosquito and the parasite result in heterogenous geographic malaria incidence within Sri Lanka in the pre-eradication period.

Malaria directly reduces fecundity, the ability to have a live birth, through an increase in the probability of spontaneous abortions and stillbirths, a lowering of coital frequency due to its other debilitating symptoms, and a decrease in general health. All pregnant women living in malarial zones, even those who have acquired immunity prior to pregnancy, are at risk of severe malarial illness. Starting from conception, malaria infections can reduce the survival probability of live births through decreased birth weight from both fetal growth retardation and premature delivery [Duffy and Desowitz (2001); Duffy and Fried (2001)]. Malaria symptoms are more severe for primigravidae, women pregnant for the first time, than multigravidae, women with at least one prior pregnancy, resulting in higher rates of neonatal and infant mortality and low birth weight live births among primigravidae [Brabin et al. (1998); Brabin and Rogerson (2001); Archibald (1956); Archibald (1958); McGregor (1984)]. The biological source of this differential is the

additional potential for a virulent malaria infection upon creation of the first placenta [Greenwood et al. (1992)] Survival probability throughout infancy and childhood is further reduced through malaria infections occurring after birth [Mcdermott et al. (1996)]. Repeated infections increase the susceptibility of children to other illness such as respiratory infections and diarrhea [World Health Organization (2003)]. Indirectly, malaria infections reduce labor capacity, limiting the amount of resources available for expenditure on nutritional intake, further reducing survival probabilities. All effects beyond the direct in utero effects will not differentially change parity specific survival.

For educational attainment, malaria can have dire effects on cognitive and physical development starting from conception. The increased incidence of low birth weight among babies born to infected mothers is of particular interest to human capital accumulation. Low birth weight can lead to reduced or delayed cognitive, physical, and neurosensory development resulting in lower total human capital accumulation [McCormick et al. (1992); Behrman and Rosenzweig (2004); Black et al. (2005); Holding and Snow (2001)]. Also, low birth weight is associated with physical stunting, developmental delay, and poor health into adolescence. Besides low birth weight, the health of the mother during pregnancy can have profound effects on later infant and child heath and development. In all children under the age of five, malaria may develop rapidly. Survivors of severe childhood malaria may suffer learning impairments, speech disorders, behavioral disorders, blindness, hearing impairment, epilepsy, and cerebral palsy [Holding and Snow (2001)]. Even without severe symptoms, nutritional intake is interrupted in the presence of a malaria infection, impairing cognitive development [Rowland et al. (1977); Shiff et al. (1996); McKay et al. (1978)]. Since advanced cognitive development depends on prior development, any disease related interruption can affect all subsequent development even in non-severe malaria cases. The full developmental effects of early life malaria infections may not be realized until higher order functions are required of individuals during schooling age [Holding and Snow (2001)]. Even if a child is able to remain malaria free, there can be negative effects on a child's total education due to expenditure on treatment and forgone employment income reducing the total income available to be spent on nutrition and schooling. School aged infections can further reduce educational attainment. These permanent physical and mental impairments adversely affect an individual's likelihood of advancing through or attending school. While malaria can affect educational attainment throughout the course of schooling, following Case et al. (2005) and Almond (2006) I will focus on the importance of prenatal and early life malaria on educational attainment.[1] Because of data availability, both direct

[1]The malaria rate at the year of birth could be a proxy for total malaria exposure age 0-18. Lucas and Weil

and indirect effects are combined in the estimation strategy.

2.2 Malaria in Sri Lanka

Historically, Sri Lanka suffered from endemic malaria in the Dry and Intermediate Zones and epidemic malaria in the Wet Zone. Map 1 displays the average district level pre-eradication malaria spleen rates. The spleen rate reflects the percentage of school children displaying an enlarged spleen, a common indication of long-standing malaria infections. Sri Lanka can be divided into three climatic zones: Dry, Intermediate, and Wet. The Dry Zone in the north and south east receives less than 80 inches of rain per annum and had the highest historical malaria incidence rates.[2] The area around Colombo in the southwest comprises the Wet Zone with rainfall in excess of 100 inches per year. This abundance of rainfall washes away suitable vector breeding sites; malaria in the wet zone is the lowest in the country. Between these two Zones geographically and climatically is the Intermediate Zone with levels of malaria between the two extremes.[3] Limited malaria control measures including pyrethrum spraying began in 1936. With a firm belief in the capability of dichloro-diphenyl-trichloroethane (DDT) to eliminate a sufficient number of disease carrying mosquitoes to halt malaria transmission, the national malaria eradication campaign in Sri Lanka began in 1947. Spray teams targeted the entire country with interior residential spraying of DDT on a tri-annual basis. Following the commencement of the campaign, there was a drastic reduction in nation-wide malaria incidence from a high of 98 cases for every 100 population in 1935 to a low of 0.002 cases for every 100 population in 1963 (17 cases on the entire island). Two highly correlated measures of malaria, the spleen rate and the incidence rate, are plotted in Figure 1. Detailed information on these time series can be found in Appendix A. The incidence rate is the number of infections per year divided by the total population. The malaria incidence rate increased from its low point in 1963 to 5 cases for every 100 population in 1975. The increase of malaria incidence is due to a number of factors including a loss of international interest and financial support, limited DDT supplies, natural selection of mosquitoes that were either exophilic (resting outdoors after feeding) or resistant to DDT, and discouragement at the realization that eradication

(2005) estimate that the timing of the eradication effect on education is primarily in the first three years of life. All results are robust to using a three year average instead of the year of birth.

[2] In addition to ideal climate for mosquito breeding, the Dry Zone also had an ancient irrigation system with dilapidated earthen water tanks that provided further mosquito breeding grounds.

[3] 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 non-representative samples collected in that district. Because of civial disturbances, the DHS sample was not collected on the Peninsula; individuals in this region care not included in the analysis.

was going to prove more difficult than originally anticipated. The incidence level has not reached the levels seen in the 1930s and 1940s, but rates in the post-eradication era are comparable to those in the early 1950s. Figure 2 plots the decline in regional spleen rates during the eradication campaign.

The eradication campaign funding was exogenous to regions within a country. The primary sources were bilateral and international, with some national funding used. Budgets were planned in advance and unable to react to variations in local experiences.

2.3 Related Literature

The growth literature contains contradictory estimates regarding the effect of disease on GDP per capita. Gallup and Sachs (2001) found that the eradication of malaria would result in an increase in GDP per capita growth of 1.3% based on cross country growth regressions. Acemoglu and Johnson (2005) instrumented for life expectancy and used similar cross country growth regressions, but they found that disease eradication does not increase GDP per capita or average levels of education. Using on a cost-of-illness analysis, Conly (1975) found that individual instances of malaria resulted in decreased rural labor productivity in Paraguay. Similar findings from across Africa are summarized in Shepard et al. (1991).

Similar to the present study are Bleakley (2007) who showed that malaria eradication in the Americas impacts adult incomes through an increase in labor market productivity and Lucas (2007) and Cutler et al. (2007) who found an increase in literacy and completed education with malaria eradication in India, Paraguay, Sri Lanka, and Trinidad. Jayachandran and Lleras-Muney (2007) found an increase in literacy with a decrease in all cause maternal mortality in Sri Lanka. Bleakley and Lange (2004) addressed fertility changes from hookworm eradication in the American South, finding a decrease in fertility upon eradication. My study expands on prior work though the multigenerational approach and emphasis on the relationship between malaria and fertility.

3 Conceptual Framework

Since malaria infections can have both contemporaneous and long lasting effects, the total malaria effect occurs across at least two generations: those of childbearing age during the eradication program and those born during the eradication campaign.

3.1 Instantaneous Effects

Fertility: Since live births are a function of both fecundity and the target number of live births, *a priori* the direction of the effects of malaria on the total number of live births per woman is uncertain. Biologically, the effect of malaria on fecundity is negative: reduced coital frequency, increased spontaneous abortions, higher probability of still births, and a decrease in general maternal health. The effect of malaria on the target number of live births is less clear. Economic theories predict that increased income and the decreased price of a surviving child will increase fertility while increased survival certainty and preferences for quality over quantity will decrease fertility [Galor and Weil (2000); Doepke (2005); Kalemli-Ozcan (2003); Barro and Becker (1989)]. Since the net result of these potentially competing effects is ambiguous, the total effect of malaria on fertility is an empirical question.

Finding a positive overall relationship between malaria and fertility would indicate a dominance of the positive preference effect over the negative biological effect. Finding an overall negative effect could be from both effects being negative or the dominance of the negative biological effect. The unique epidemiology of malaria provides a source of identification to untangle this uncertainty.

The transition to initial and higher order parity provides additional insight into the importance of biological versus other factors in the changes of fertility with malaria eradication. Malaria infections are more severe among women pregnant for the first time than among those with prior pregnancies, leading to a higher probability of spontaneous abortions or still births among primigravidae. Mechanically, if the biological effects of malaria dominate the other potential sources of increased fertility, then malaria eradication should reduce the time of the transition to initial parity and have less of an effect on the transition to higher order parity. In a hazard model framework, if the biological effects dominate, then the eradication of malaria would increase the probability of a first birth and have a smaller effect on the probability of later births. Preference or income induced changes in fertility from malaria eradication should be uniform across all parities.[4]

Survival: The effect of malaria on infant and child survival is unequivocally negative. Those born during the post eradication period should be healthier and more likely to survive infancy and childhood. Because of the more severe symptoms of malaria in primigravidae than multigravidae, the survival outcomes for first born should increase more than the survival outcomes of those of

[4] I cannot empirically rule out a change in behavior that mimics the expected biological response. The additional survival among the first born reinforces the biological claim. Some of the biological effects are uniform across parities. The lack of differential parity specific outcomes would not reject the dominance of a uniform biological effect.

higher birth order with the eradication of malaria. Other changes in survival probabilities induced by malaria eradication should be uniform regardless of birth order.

3.2 Second Generation Effects

Education: Whether from the direct effects of increased birth weight and health or the indirect effects from an improved provision of nutrition and increased survival expectations, malaria eradication should increase educational attainment. Because of data availability, both direct and indirect effects on education are combined in the estimation strategy.[5]

Fertility: Members of the second generation completed their childbearing after nationwide malaria eradication. The treatment and control groups are determined by birth cohort and region. Women born after eradication in the previously high malaria regions would have been healthier, leading to higher fecundity. In contrast, their additional education increased their opportunity cost of time, potentially lowering total fertility. The net effect on age-specific fertility combines these counteracting forces.

4 Identification Strategy

The primary conceptual challenges in identifying the effects of changing health environments on fertility and child survival are the direction of causation and both measures' correlation with unobservable regional characteristics. The exogenous change in the malaria rate that occurred with the malaria eradication campaigns combined with heterogenous pre-eradication levels of malaria allows for proper identification.

4.1 Data

Two types of data are used for the analysis: individual survey level data and regional malaria data.

Survey Data: The individual data on first generation fertility and second generation child survival are from the World Fertility Survey conducted in Sri Lanka in 1975. It is a retrospective fertility survey of ever married women aged 12 to 50 (born 1925 to 1963), designed to be nationally

[5] A further test of the dominance of the biological effect would be to test educational gains by birth order. Unfortunately, the data on education do not contain this information.

representative. From this cross section of 6,810 women born in Sri Lanka there are 27,076 live births. Of these births, 25,811 were at least one year old prior to the survey.

The individual data on educational outcomes and second generation fertility outcomes are from the Demographic and Health Survey of ever married women aged 15-49 conducted in Sri Lanka in 1987. The resulting sample of 5,859 women was drawn from areas containing 86% of the 1986 Sri Lankan population. The eastern coastal belt and northern province were excluded due to civil disturbances. After eliminating all women born abroad and those under 19 at the time of the survey, the primary sample for estimation consists of 5,822 ever married women born in Sri Lanka between 1937 and 1968.[6]

Each woman is assigned a birth year and region of residence based on her responses. Based on the epidemiology of malaria, the malaria rate in the region of birth at the time of birth should be used. Because of data limitations, I am unable to ascertain a woman's birth location unless she remained a resident of that city or village until the time of the survey. Individuals remaining in their village or city of birth constitute 36% of the sample. Women are assigned a malaria rate based on their current region of residence. Since malaria rates are assigned at the regional level, mismeasurement should be minimal. I also present the very similar results separately for those who have never moved from their birthplace.[7] Means for the dependent variables of interest both before and after eradication can be found in Table 1. The largest changes in the mean values occur in the regions with the highest level of pre-eradication malaria levels.

Regional Malaria Data: I use the level of malarial prevalence in a region to capture both the direct and indirect effect of an individual's malaria exposure. Precisely, from Newman (1965) I use a district level time series of malaria spleen rates, a measure of long-standing malaria, aggregated to the regional level to match the finest level of geographical disaggregation in the WFS and DHS Sri Lanka data. These series are plotted in Figure 2.[8] Details about the exact construction of the series appear in Section A. The geographic distribution of malaria in Sri Lanka is primarily due to climatic and geographic differences within the country [Newman (1965); Meegama (1986);

[6] Since this sample will be used for completed fertility and total education, limiting the sample to aged 19 and older will reduce any bias resulting from those who are married and younger than 19 being a selected sample of their cohorts.

[7] Since the results are robust to limiting the sample to those that have not moved from their city of village of birth, this concern of particular migration patterns resulting in bias is ameliorated.

[8] The early 1940s fall in malaria and subsequent rebound is primarily due to rainfall: excessive rain during the monsoon seasons in 1943 and 1944 reduced the number of breeding sites in the regions with the typically higest malaria. There were also limited eraducation attempts concurrent to this weather shift.

Konradsen et al. (2000)].

4.2 Estimation Strategy

Cross country evidence shows that countries with lower levels of GDP per capita also have higher malaria rates, on average. Empirically identifying the ultimate cause, therefore, is impossible on a cross country basis. Combining an exogenous change in the malaria rate from the malaria eradication program with individual level survey data, I can identify malaria's total effect on fertility, survival, and education. The malaria eradication program in Sri Lanka, an exogenous change in the malaria rates that brought malaria levels across all regions of the country to zero from various levels, provides the quasi-experiment necessary for identification.

During the eradication program in Sri Lanka all zones were uniformly treated with DDT. The difference in exposure to the treatment is based on the pre-eradication malaria infection levels: those zones with the highest pre-eradication malaria levels gained more from malaria eradication than the zones that had low levels of malaria prior to eradication. $Malaria_{jt}$ is the malaria rate in zone j at time t (as discussed above). Y_{ijt} denotes outcome Y (e.g., the probability of a live birth) for individual i in region j at time t. The general specification is then

$$Y_{ijt} = \alpha + \beta Malaria_{jt} + \delta_j + \delta_t + X_{ijt}^{'}\Gamma + \varepsilon_{ijt} \tag{1}$$

where δ_j are region fixed effects that control for any region specific heterogeneity, and δ_t are time fixed effects that control for the average nationwide changes in a given year. X_{ijt} is a vector of individual level covariates, specific to each outcome of interest. β, the coefficient on the malaria rate, is the primary coefficient of interest.

Two aspects of the malaria eradication program provide for the identification of β: (1) exogenous implementation of the program and (2) heterogeneous indigenous rates of malaria infection.

Exogenous implementation of the eradication program: The eradication program was exogenous to the specific regions within a country. The campaign was instituted on a national scale under the guidelines and direct supervision of the WHO with the explicit purpose of nationwide malaria eradication. Spraying teams were centrally and uniformly trained with explicit instructions as to the geographic region to spray and precise concentrations of DDT to use. Each dwelling was sprayed on a tri-annual basis. The nationwide rollout occurred within six months with the goal complete nationwide eradication. Within the rigid framework there was no provision for

sub-national decision making. "Local" decision making was undertaken at the national level and was unable to react to local conditions. The primary source of funding was either bilateral or multilateral aid. Local or district level resources did not determine spraying allocations.

Heterogeneous indigenous rates of malaria infection: The treatment and control groups are determined by the pre-eradication malaria rates. While the entire country was treated with DDT interior residual spraying, individuals living in regions with low levels of indigenous malaria received relatively less benefit from the spraying (that is, less reduction in their exposure to malaria) than those living in regions in which there was endemic malaria. The control group (regions with low levels of indigenous malaria) prevents annual nationwide changes in survival and fertility from being attributed to malaria.

Regional variations in initial malaria levels could be due one of two situations (or a combination of the two): (a) time invariant climatic and geographic factors and (b) initial underdevelopment. In Sri Lanka, the initial concentration of malaria closely reflects region-specific climatic and geographic peculiarities, suggesting that the pre-treatment levels of malaria were due to regional fixed effects.

The areas of the most intense malaria transmission were also some of the most underdeveloped. Regardless of the cause, identification is not precluded. If underdevelopment is the ultimate cause of malaria and not ecology, then an exogenous change in the malaria rates, uncorrelated with other development shocks, creates the non-linearity necessary for identification. The use of regional fixed effects will control for any time invariant heterogeneity between the zones. An additional set of specifications include a set of regional trends to control for potential linear time varying heterogeneity. Results are robust to this inclusion.

In practice, the malaria rates did not fall immediately to zero, but in each region drastic reductions were achieved quickly. Figure 2 shows the regional time series of malaria spleen rates. Eradication campaigns were implemented almost simultaneously throughout the country, but because of the density of the mosquitoes, malaria eradication was not achieved instantaneously. As long as the speed of the regional fall in malaria is uncorrelated with region-cohort unobservables, the fixed effects estimator of the malaria effects remains unbiased and consistent. Since the regional decline in malaria closely parallels the national decline, this appears to be the case.[9]

By comparing changes in fertility, survival, and education among those in the regions with the highest indigenous malaria (treatment group) to those with low levels of indigenous malaria

[9] The adjusted R-squared from a regression of the regional rates and regional indicator variables on the national malaria rate is 0.92.

(control group) through the varying levels of $Malaria_{jt}$, I am able to isolate the effect of malaria eradication.

5 Estimation

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. For the effect on human capital accumulation, I estimate the effect of malaria on years of completed education and literacy.

5.1 Fertility

Figure 3 provides prima facie evidence that malaria eradication increases total fertility. Between 1937 and 1953, the increase in the crude birth rate per 1000 population was the most pronounced in districts with the largest decrease in malaria. This could be the result of increased fertility or changing demographics. Individual level regressions will further explore this relationship.

To estimate the effect of malaria on instantaneous fertility, I estimate the following equation:

$$P(B_{ijt}) = \alpha + \beta^{PB} malaria_{jt} + \delta_j + \delta_t + \delta_a + X'_{ijc}\Gamma + \varepsilon_{ijt} \tag{2}$$

where $P(B_{ijt})$ is the probability of respondent i in region j having a live birth at time t, $malaria_{jt}$ is the malaria rate in region j at time t, δ_j are region fixed effects, δ_t are time fixed effects, δ_a are maternal age fixed effects, and the X_{ijt} are individual level controls including the number of years of education and indicators for current residence type, childhood residence type, ethnicity, and birth control knowledge. The sample includes all women-years from age fifteen to the time of the survey. Standard errors are allowed to be correlated within a village or urban sample point, but are assumed to be uncorrelated between them. The remainder of the estimates use the same error structure. The estimation results appear in Column (1) of Table 2.[10] In contrast to the theories presented in Section 3.1 of a movement towards quality or a shift towards fewer children accompanying increased certainty of survival, the number of live births increased as the malaria

[10] Because of the incidential parameters problem with non-linear estimation procedures that include fixed effects [see Lancaster (2000)], the estimates that appear in all Tables are least squares estimates. The marginal effects evaluated at the mean from non-linear specifications are similar.

rate fell. The probability of a live birth in a particular year was a negative function of the malaria rate; as malaria eradication occurred in the previously heavily infected regions the probability of a live birth increased. The first births in the sample occurred in 1940. The highest regional spleen rate in a year during the sample was 42.7%. Therefore, reducing malaria from this level to zero would increase the probability of a live birth by 5.21 percentage points. Over the entire sample the average probability of a live birth was 0.198. If malaria had been eradicated prior to the start of the sample, the probability of birth would have been 0.199, equivalent to an extra 144 children over $133,428$ woman-years, an increase in the number of children of 0.5%. Over those years in which the malaria rate was greater than 5%, eradication would have increased the average probability of a live birth from 0.149 to 0.165. Based on age-specific fertility rates, for a decrease in the malaria spleen rate from 16.0% (the average level for the sample in 1940) to 0% over the entire reproductive lifespan the total fertility rate would have increased by 0.83 live births. The point estimates are robust to the inclusion of regional time trends or maternal fixed effects.[11]

To test if biology dominates preference based changes in fertility, Equation (2) is re-estimated as a hazard model where the hazard of having a live birth starts at age fifteen and the woman is removed from the sample upon a live birth. For this model, the covariates are the same as those in Equation (2). The results from the estimation appear in Columns (2) - (4) of Table 2.[12] Malaria exerted a negative and significant effect on the transition to initial parity (Columns (2) and (3)) decreasing the probability of transitioning to having had a live birth by 3.54 percentage points if the malaria spleen rate was reduced from its highest level in the sample (42.7%) to to 0%. The sample is 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 larger absolute magnitude. In contrast with the findings for the first live birth, malaria levels have an insignificant effect on the transition to a second live birth (Column (4)). The coefficient is positive and highly insignificant. Malaria had a negative effect on the transition to initial parity, but no effect on the transition to higher order parity.[13] Of the potential mechanisms that caused

[11] Formally, regional time trends are included for all regions except one and the year dummy variables remain in the model: $P(B_{ijt}) = \alpha + \beta^{PB} malaria_{jt} + \delta_j + \delta_t + \delta_a + \delta_j * timetrend_t + X'_{ijc}\Gamma + \varepsilon_{ijc}$. Regional trends are included similarly for all other estimates. The coefficient on the malaria rate is -0.298 (0.074) with the inclusion of regional trends and -0.296 (0.067) with maternal fixed effects.

[12] Equation (2) is estimated as a linear probability model as it is equivalent to estimating a discrete time proportional hazard model [Allison (1984)]. The finding in Column (2) is robust to the inclusion of regional time trends with the estimated coefficient on $malaria_{jt}$ of $-0.245(0.063)$.

[13] The initial realization of a second birth is 1942.

the increase in fertility following eradication, only the elimination of a biological constraint would have this parity specific attribute as all preference based fertility changes would be uniform across parity levels.

5.2 Child Survival

Parity specific malaria effects could be evident in birth order specific child survival. The child survival estimation is a linear probability model:

$$P(survival_{ijc}) = \alpha + \beta^S malaria_{jc} + \delta_j + \delta_c + X'_{ijc}\Gamma + \varepsilon_{ijc}. \tag{3}$$

The additional individual controls in X_{ijc} are maternal education and separate indicators for being a part of a multiple birth, female or missing sex, type of residence, ethnicity, birth order, and maternal birth year. Separate models are estimated to establish the effect of malaria on the probability of survival to age one and to age five on two samples: all births and first births. The point estimates appear in Table 3. While negative, the malaria rate's effect on child survival to age one or five is insignificant for the sample including all births in Columns (1) and (4). When the sample is limited to only the first born, the malaria rate in the individual's year of birth has a negative and significant impact on child survival to age one and five (Columns (2) and (5)).[14] When the sample is limited to non-first births (Columns (3) and (6)), then the malaria rate at birth has a positive and insignificant effect on survival that is statistically distinguishable from the first birth estimates. The highest spleen rate over the sample of live births is 42.73%, therefore eliminating malaria from this level would, in expectation, increase the probability of survival to age one by 18.3 percentage points and the probability of survival to age five by 25.7 percentage points for the first born. The difference in results between the first birth and all births suggests that malaria's effect on survival was operating through the direct pre-natal health effects since once an infant is born their post-birth malaria exposure should not vary by birth order. This differential result by birth order confirms the primacy of the biological effect causing the fertility increase.

[14] The point estimates are robust to the inclusion of regional time trends (the Column (2) analogue is -0.478 (0.216) and the Column (5) analogue is -0.628 (0.256)).

5.3 Second Generation Education

The general model estimated is

$$education_{ijc} = \alpha + \beta^E malaria_{jc} + \delta_j + \delta_c + X'_{ijc}\Gamma + \varepsilon_{ijc} \quad (4)$$

where $malaria_{jc}$ is the malaria rate in region j at the time of the respondent's birth (member of cohort c) and the X_{ijc} matrix includes indicators for type of current residence, type of childhood residence, and ethnicity indicators to control for potential within region racial segregation. Educational attainment is measured four ways: years of completed education, years of completed primary education, the ability to read a newspaper easily (high literacy), and the ability to read a newspaper or letter easily or with difficulty (minimal literacy).

Table 4 contains the separate estimates of Equation (4) with years of completed primary education, at least minimal literacy, total years of completed education, and high literacy as the dependent variables.[15] As expected the estimate of the coefficient on the malaria rate at birth is negative; the years of completed education or literacy increased the most for those who accrued the greatest benefited from malaria eradication. The robustness checks demonstrate a noteworthy pattern: total years of completed education is not robust to the inclusion of regional time trends (Column (8)) and high literacy is not robust to the limited non-mover sample (Column (2)). In contrast, the malaria effect on dependent variables that capture a more basic educational achievement (years of primary schooling completed and at least a minimal level of literacy) are robust to the inclusion of regional time trends or limiting the sample to non-movers. Malaria had a more robust effect on lower levels of education and literacy than on higher levels of education or literacy. The importance of early life malaria exposure manifests itself in early educational attainment.

Based on Column (1) a reduction in the malaria rate from 100% to 0% would increase expected years of completed primary schooling by 1.79 years. The highest regional malaria rate in Sri Lanka over the period under study is 59.7% in the Irrigated Dry Zone. Based on the point estimates, a reduction of this rate to 0% would lead to an increase in the expected number of years of completed primary education of 1.07 years. If there had been no malaria in the Irrigated Dry Zone for the cohorts born 1937 - 1939, the average number of years of completed primary education would have been 3.68 instead of 2.68 years. Those born in the Irrigated Dry Zone in 1967 - 1969 completed an average of 4.50 years of primary education. Therefore, malaria accounts for 54.9% of the increase

[15] Under the Sri Lankan schooling system, primary school lasted seven years.

in primary schooling over that period. Of those born 1937 - 1939, 75.0% had at least minimal levels of literacy. Without malaria that number is projected to be 90.8%. Of those born in the same region 1967 - 1969, 100% had at least minimal levels of literacy. Malaria accounts for 63.2% of this 25 percentage point increase.

5.4 Second Generation Fertility

The preceding section showed that malaria reduction increases primary education, and malaria reduction should also increase maternal health; I estimate the reduced form effect of early life malaria exposure on total fertility. The higher level of education that resulted from malaria eradication raised the opportunity cost of children and increased knowledge about contraception, both of which would lower fertility. Counteracting this effect, reducing a woman's malaria exposure in childhood led her to be healthier, with higher fecundity as an adult and have higher income from better health and more education. Finally, growing up in a low-malaria environment may change a woman's perception about the probability of child survival, leading to a reduction in precautionary childbearing. The effect of malaria eradication on fertility that I find will be a composite of all of these effects.

The two measures that I use are the probability of birth and the percentage of births who survive to their fifth birthday. I estimate

$$P(B_{ijt}) = \alpha + \beta^F malaria_{jc} + \delta_j + \delta_t + \delta_a + X'_{ijt}\Gamma + \zeta_{ijt} \tag{5}$$

where $P(B_{ijt})$ is the probability of a live birth occurring to respondent i living in region j at time t and all other notation and controls are the same as that from Equation (4). A higher malaria rate in infancy results in increased female fertility as can be seen in Columns (1) and (2) of Table 5. Therefore, the effect from increased education or a changed perception of survival dominate the pure health effects. The point estimates are quite similar for both the full and non-mover sample.[16] Eradication of malaria from the highest observed level at maternal birth (59.7%) reduces the probability of a live birth in a given year by 7.2 percentage points. The point estimate in Section 5.1 based on the contemporaneous shift in age-specific fertility was -0.122. The initial effect of malaria eradication is the increase of age-specific fertility. The second generation effect of decreasing age-specific fertility with eradication is of the same absolute magnitude as the

[16] The inclusion of regional trends results in a larger estimate of the malaria effect with a point estimate of 0.232 (0.022).

initial increase. Thus, the net effect will be for fertility in the second generation to return to the pre-eradication levels, absent other nationwide changes in fertility.

Increased maternal health and education could also be reflected in child survival. For the percentage of children who survive to age 5 I estimate

$$alive5_{ijc} = \alpha_0 + \beta^A malaria_{jt} + \delta_j + \delta_c + X'_{ijc}\Gamma + u_{ijc} \tag{6}$$

where $alive5_{ijc}$ is the percentage of children per woman who had survived until their fifth birthday.[17] The other variables and all subscripts are the same as those that appear in Equation (5). Reduction in the malaria rate during the infancy of the mother does not affect the percentage of live births who survive to age 5. Since the survival probability among live births across different cohorts changed very little even in the most heavily infected region, this lack of a significant result is not surprising. Of those born to 1937-1939 cohorts in the Irrigated Dry Zone, 96.0% of live births per woman survived to age 5. There is no change in the number for the 1955-1957 cohorts with 95.4% of live births per woman surviving to age 5.[18]

6 Alternative Explanations

Other authors have noted malaria's role in causing underdevelopment and the key role DDT spraying played in the reduction of malaria. When Gill (1940) divided Sri Lanka into five different zones by degree of malaria endemicity in 1940, he noted the high degree of correlation between the level of the malaria rate and high death and infant mortality rates, asserting that "it is reasonable to suppose that the variations in the intensity of endemic malaria...are mainly responsible for this [high correlation] circumstance...it has not been found possible to account for the facts on any other hypothesis." Coale and Hoover (1958) attribute the convergence of the death rates among the districts in Sri Lanka from 1945 to 1958 to residual interior spraying of DDT. Even with these claims the implementation of a public health intervention that coincided exactly with the malaria eradication campaign would bias the results towards finding a spurious relationship between malaria and fertility and survival. Public health availability in the endemic region was superior to that in the

[17] $alive5$ is calculated for each respondent as the total number of children of that respondent who reached age five divided by the total number of births to that respondent that occurred more than five years prior to the survey. Women will only be included in this regression if they gave birth at least once more than five years prior to the survey.

[18] In a setting with higher child mortality, the malaria eradication induced increase in education would be expected to be reflected in an increase in child survival.

less malarious regions prior to eradication: the population per hospital was lower, the population per hospital bed was similar, the population adjusted admission rates were higher, and the coverage of the central dispensaries with in-patient care was better in the highly malarious area. In the post-war era "there is no evidence for an unbalanced improvement in medical services" [Gray (1974)]. The malaria effect is also not due to increased smallpox vaccination. Primary smallpox vaccinations were widespread prior to malaria eradication with between 72% and 89% of live births vaccinated within one year of birth from 1937 to 1943. Administration of this vaccination declined during World War II and failed to top 80% into the late 1950s even though small pox still appeared on the island until 1974 [Langford (1996)]. Continual health improvements uniformly applied nationwide are not sufficient for spurious results. There is also no evidence of differential nutritional improvements. Instead, individuals in the highly malarious zone had higher nutritional value in their diets than their peers in villages of lower malaria endemicity in the pre-war period as measured by daily consumption of protein, carbohydrate, calories, and minerals and the lower prevalence of malnutrition [various studies as summarized in Gray (1974)]. Based on more limited data, nutrition in Colombo does not appear to be superior to that available in the highly endemic zone. The post-war nutritional improvements did not favor the endemic zone: in the late 1950s the nutrition of the Dry Zone inhabitants deteriorated as individuals shifted to a wage based labor structure away from production of agricultural products for consumption. Even though the timing of the decrease in malaria rates in Sri Lanka is partially coincidental with the introduction of high yield variety (HYV) rice, its introduction did not lead to differential increases in income correlated with malaria reduction. While the "take off" of rice yields in Sri Lanka is dated 1967, into 1973 only 2.5% of rice seed was of the HYV. Furthermore, concurrent to the shift to the HYV, declines in owner operator holdings, increases in the costs of consumer goods and "livelihood necessities," a lack of availability of machinery, and the system of village elders retaining the seed for themselves prevented cultivators' real income from increasing [Brown (1970); Pearse (1980)].

There were no differentially applied education programs that targeted regions where malaria was the highest [Ekanayake (1982)]. There is evidence that there were some educational improvements in the Colombo region where malaria was lower than the national average at the start of the program. If anything, these educational improvements will bias the results towards zero.

There is inconsistent evidence about the effect of DDT interior residual spraying on infant and child mortality. Studies on animals have found a negative correlation between similar insecticide residuals and adverse reproductive outcomes. In humans, the results are less conclusive, but there

is agreement that it does not induce an increase in fecundity or child survival. Since the entire country was treated with DDT, any effects will be uniform across the country and captured by time fixed effects.

7 Discussion and Conclusions

The multigenerational approach used here allows a richer understanding of the effect of malaria beyond immediate outcomes. Fertility among those of childbearing age increased as malaria eradication occurred. This does not appear to be driven by a significant change in the probability in child survival. Instead, the dominant mechanism is the shortened time to have an initial birth, indicating a biological response to a previously binding constraint on the ability to have the first live birth. The increased fertility is transitory as the subsequent generation attained more human capital than those born before eradication in the regions with the previously highest levels of malaria and had lower fertility. The decrease in second generation fertility would cause age-specific fertility to revert to the pre-eradication levels absent other country-wide changes in fertility.

Lasting a generation, the initial fertility increase can cause a reduction in GDP per capita as the size of the non-productive segment of the population increases. The full evolution of fertility back to the lower pre-eradication fertility levels will take an entire generation. During this transition, more highly educated individuals will enter their productive years. The net effect on GDP per capita of the education and fertility effect will be positive, but will not be realized immediately. The duration of the transitory population increase also provides a reconciliation between the two contradictory views in the growth literature of the relative importance of health for GDP per capita and GDP per capita growth. The relative sizes of the initial increase in population, the subsequent reduction in fertility, and the increase in education will determine the duration of a potential decrease or stagnation in GDP per capita.

A Appendix - Data Construction

Regional level data are the spleen rates from Newman (1965) Table A4. The spleen rates were collected by measuring the spleens of all children in a chosen sample school who were present on the day of the survey. This procedure produces downward bias in the reported spleen rate of a district since children who were too ill to attend school would not have been surveyed. This bias should not be systematically related to the average levels of malaria in a district.

These twenty-two district rates are aggregated on a population weighted basis into the seven geographical regions defined in the DHS-I data: metro Colombo, Colombo feeder areas, south-west lowlands, lower-south central hills, upper-south central hills, dry zone irrigated, and dry zone rain fed. The aggregation was performed based on the maps in Newman (1965) and Department of Census and Statistics (1988). When possible, the exact population from a given district was assigned to the correct region. Otherwise, the population of a district was divided evenly between all regions of which it was a part in order to create a population weighted spleen rate. These assignments assume a homogenous spleen rate among a population within a given district.

In order to get the most complete regional malaria spleen rates possible, I used the following algorithm:

1. Actual data when available are used. District level spleen rates are available 1937-1941 and 1946-1955. After 1955 spleen rates were not collected as the continued eradication of malaria rendered the values for all districts approximately zero.

2. I estimated the following regression individually for each region to predict the regional spleen rate from the national incidence rate:

$$spleen_{jt} = \beta_0 + \beta_1 incidencerate_t + \beta_2 incidencerate_t^2 + \varepsilon_{jt}$$

   using the twelve years for which the regional spleen rates and the national incidence rate overlap. Based on this regression, I predicted nine additional years of spleen rates (1942-1945, 1956, 1962-1969).[19] I constrain the predictions to be greater than or equal to 0.

3. For the remaining five years of data (1957-1961) I linearly interpolate the regional rates. Qualitative assessments over this period do not indicate disruptions in the malaria eradication program that would lead to significant non-linearities [Newman (1965)].

[19] Because of the quality of the predictions (adjusted $R^2 > 0.90$ and $var(spleen_{jt} - \widehat{spleen}_{jt}) = 0.00029$) and the large magnitude of the t-statistics, I do not adjust the standard errors in Section 7.

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Figure 1: Incidence and Spleen Rates

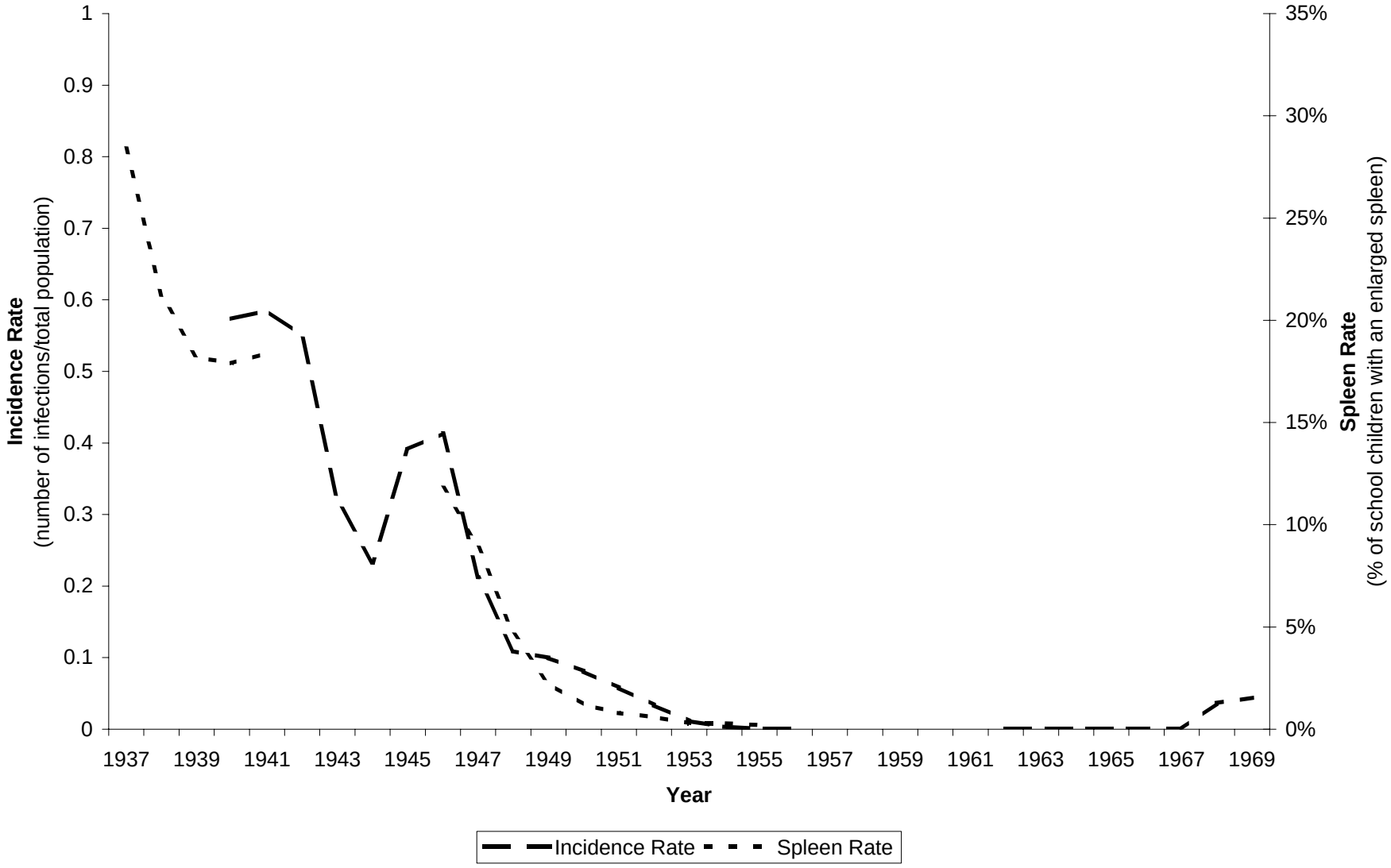

Figure 2: Spleen Rates by DHS Region

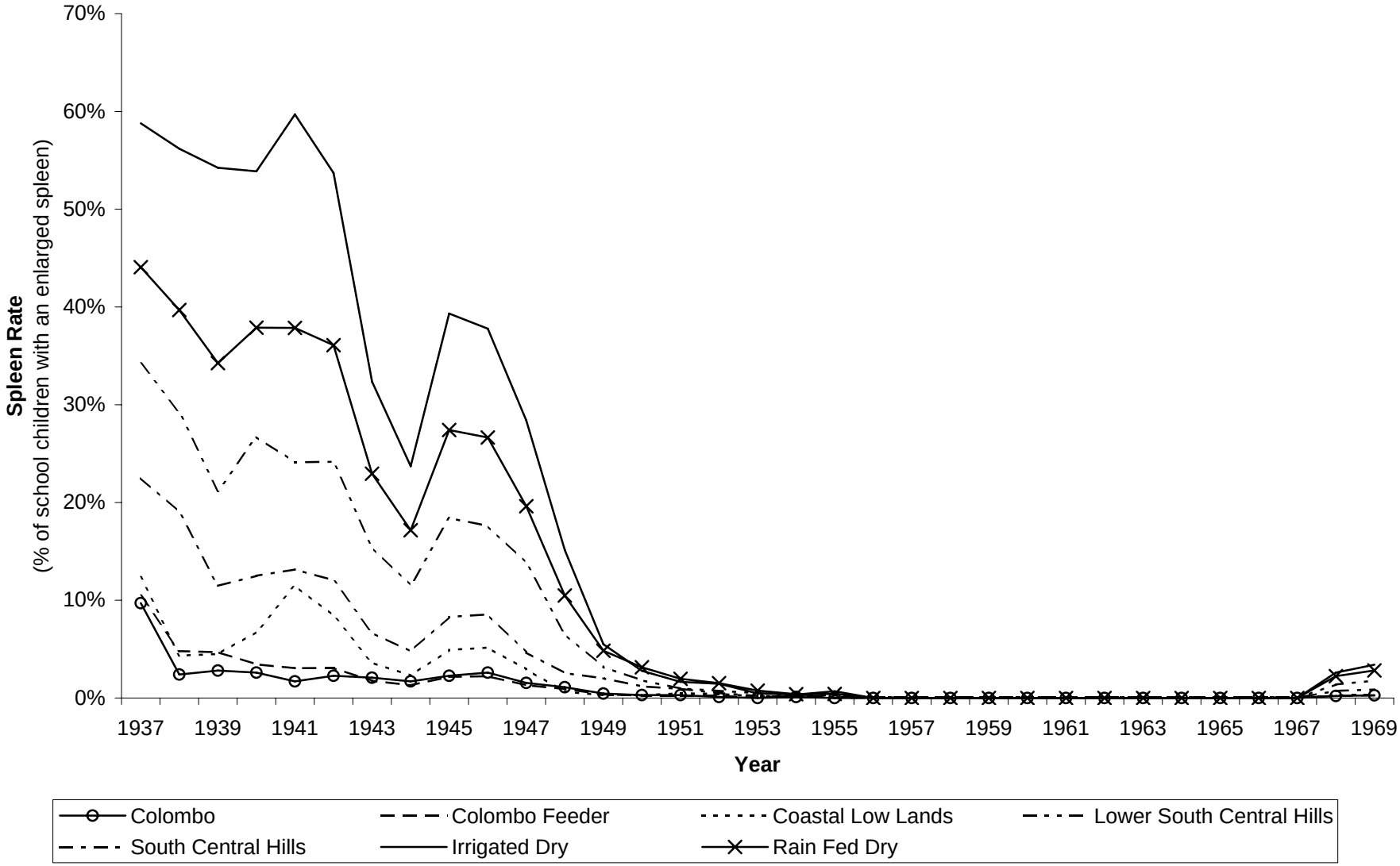

Figure 3: Crude Birth Rate

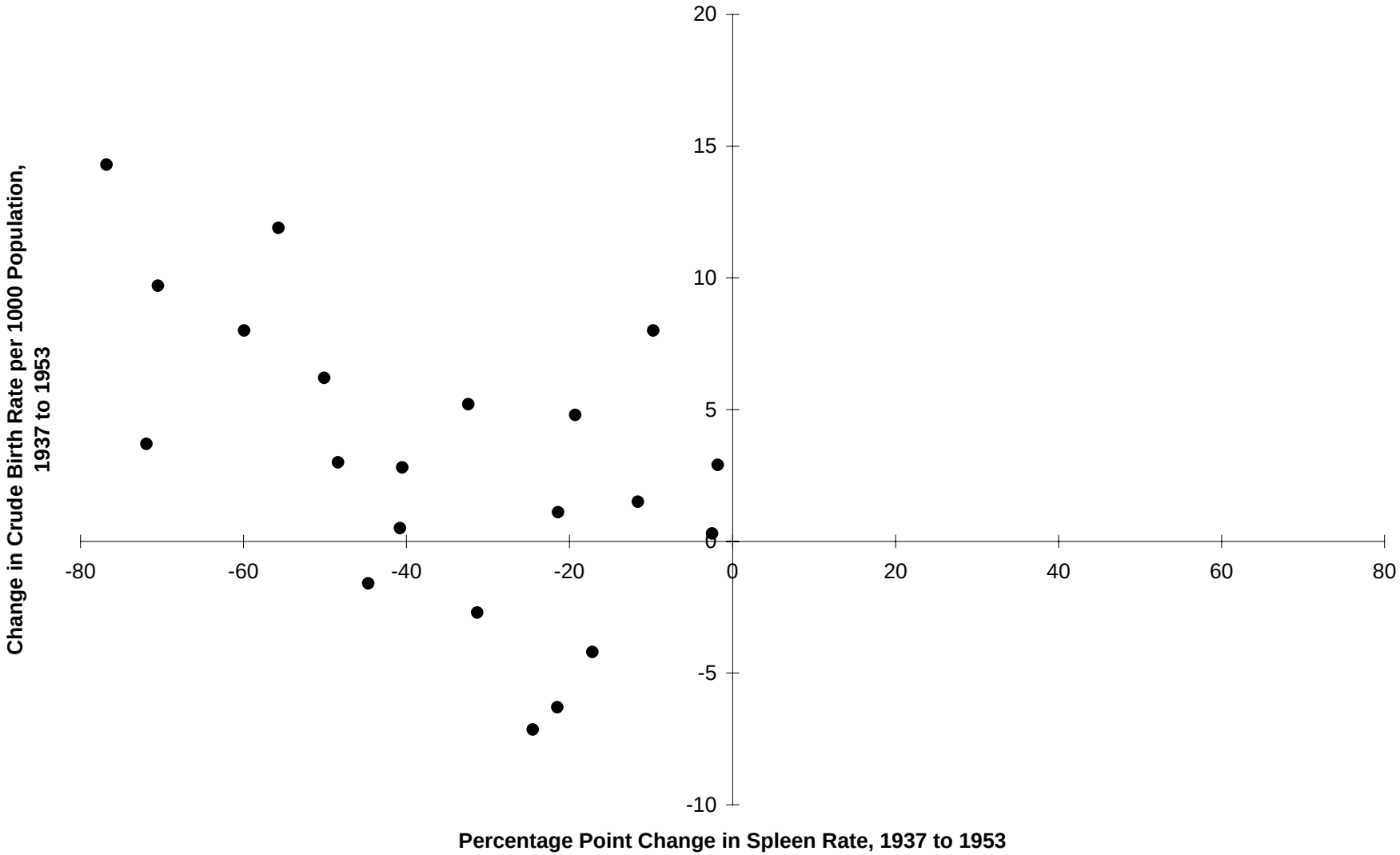

Table 1 - WFS and DHS Sample Means

Probability of Birth	**1937 - 1951**	**1952 - 1975**	**Increase**
Sri Lanka	0.127	0.190	0.063
Colmobo (Low Malaria)	0.099	0.171	0.072
Colombo Feeder (Low Malaria)	0.094	0.160	0.066
South Western Coastal Low Lands (Intermediate Malaria)	0.133	0.187	0.053
South Central Hills (Intermediate Malaria)	0.146	0.184	0.038
Irrigated Dry Zone (High Malaria)	0.135	0.215	0.080
Rain Fed Dry Zone (High Malaria)	0.157	0.233	0.076
Survival Until Age 1			
Sri Lanka	0.909	0.939	0.030
Colmobo (Low Malaria)	0.932	0.950	0.018
Colombo Feeder (Low Malaria)	0.928	0.951	0.023
South Western Coastal Low Lands (Intermediate Malaria)	0.902	0.929	0.027
South Central Hills (Intermediate Malaria)	0.900	0.933	0.034
Irrigated Dry Zone (High Malaria)	0.911	0.948	0.037
Rain Fed Dry Zone (High Malaria)	0.896	0.922	0.026
Survival Until Age 5			
Sri Lanka	0.861	0.908	0.047
Colmobo (Low Malaria)	0.888	0.926	0.039
Colombo Feeder (Low Malaria)	0.892	0.928	0.035
South Western Coastal Low Lands (Intermediate Malaria)	0.834	0.891	0.057
South Central Hills (Intermediate Malaria)	0.847	0.903	0.056
Irrigated Dry Zone (High Malaria)	0.871	0.919	0.047
Rain Fed Dry Zone (High Malaria)	0.852	0.880	0.028
Birth Year:	**1937 - 1951**	**1952 - 1968**	
Years of Completed Education			
Sri Lanka	5.73	6.52	0.79
Colmobo (Low Malaria)	7.29	7.57	0.28
Colombo Feeder (Low Malaria)	7.57	7.70	0.13
South Western Coastal Low Lands (Intermediate Malaria)	6.30	6.77	0.47
Lower South Central Hills (Intermediate Malaria)	5.63	6.82	1.19
South Central Hills (Intermediate Malaria)	3.84	5.09	1.24
Irrigated Dry Zone (High Malaria)	5.11	6.86	1.75
Rain Fed Dry Zone (High Malaria)	4.70	5.80	1.10
Ability to Read Easily			
Sri Lanka	0.71	0.75	0.04
Colmobo (Low Malaria)	0.85	0.86	0.01
Colombo Feeder (Low Malaria)	0.87	0.87	0.00
South Western Coastal Low Lands (Intermediate Malaria)	0.79	0.79	0.00
Lower South Central Hills (Intermediate Malaria)	0.72	0.78	0.06
South Central Hills (Intermediate Malaria)	0.44	0.55	0.11
Irrigated Dry Zone (High Malaria)	0.71	0.82	0.11
Rain Fed Dry Zone (High Malaria)	0.63	0.71	0.08
Ability to Read Easily or with Difficulty			
Sri Lanka	0.81	0.84	0.03
Colmobo (Low Malaria)	0.92	0.92	-0.01
Colombo Feeder (Low Malaria)	0.94	0.94	0.00
South Western Coastal Low Lands (Intermediate Malaria)	0.89	0.87	-0.02
Lower South Central Hills (Intermediate Malaria)	0.82	0.87	0.05
South Central Hills (Intermediate Malaria)	0.58	0.69	0.11
Irrigated Dry Zone (High Malaria)	0.81	0.89	0.08
Rain Fed Dry Zone (High Malaria)	0.77	0.82	0.05
Fertility by Age 30			
Sri Lanka	2.70	2.33	-0.37
Colmobo (Low Malaria)	2.45	1.84	-0.61
Colombo Feeder (Low Malaria)	2.05	2.19	0.14
South Western Coastal Low Lands (Intermediate Malaria)	2.07	1.96	-0.11
Lower South Central Hills (Intermediate Malaria)	2.90	2.32	-0.58
South Central Hills (Intermediate Malaria)	2.77	2.32	-0.45
Irrigated Dry Zone (High Malaria)	3.32	2.80	-0.52
Rain Fed Dry Zone (High Malaria)	3.57	2.93	-0.64
Fraction of Live Births Who Survive to Age 5			
Sri Lanka	0.950	0.956	0.006
Colmobo (Low Malaria)	0.971	0.961	-0.010
Colombo Feeder (Low Malaria)	0.961	0.962	0.001
South Western Coastal Low Lands (Intermediate Malaria)	0.956	0.956	0.000
Lower South Central Hills (Intermediate Malaria)	0.956	0.945	-0.011
South Central Hills (Intermediate Malaria)	0.925	0.949	0.024
Irrigated Dry Zone (High Malaria)	0.938	0.953	0.016
Rain Fed Dry Zone (High Malaria)	0.950	0.968	0.018

Table 2 - Fertility

Dependent Variables:	Probability of Birth	Hazard of Birth		
		First Birth	First Birth (women with at least one live birth)	Second Birth
Independent Variables	(1)	(2)	(3)	(4)
Malaria Rate	-0.122	-0.083	-0.117	0.109
	(0.062)**	(0.048)*	(0.049)**	(0.200)
Additional Covariates				
Years of Maternal Education	-0.01	-0.011	-0.013	0.016
	(0.000)***	(0.000)***	(0.001)***	(0.002)***
Current Residence (omitted = Urban)				
Rural Residence	0.009	-0.006	-0.007	0.034
	(0.004)**	(0.005)	(0.005)	(0.015)**
Estate Residence	-0.011	-0.011	0.013	-0.027
	(0.012)	(0.014)	(0.018)	(0.041)
Chilhood Residence (omitted = Urban)				
Rural Childhood Residence	-0.006	0.003	0.002	-0.04
	(0.004)	(0.004)	(0.005)	(0.013)***
Estate Childhood Residence	-0.044	-0.03	-0.033	-0.038
	(0.010)***	(0.010)***	(0.012)***	(0.030)
Missing Childhood Residence	-0.079	-0.031	0.031	-0.057
	(0.035)**	(0.048)	(0.022)	(0.162)
Ethnicity (omitted = Sinhala)				
Sri Lanka Tamil	0.012	0.018	0.016	-0.024
	(0.006)*	(0.007)***	(0.008)**	(0.017)
Indian Tamil	0.017	0.017	0.007	-0.004
	(0.010)*	(0.011)	(0.014)	(0.031)
Sri Lanka Moor	0.019	0.023	0.026	-0.015
	(0.007)***	(0.008)***	(0.009)***	(0.018)
Other Ethnicity	0.033	0.024	0.036	0.066
	(0.012)***	(0.016)	(0.017)**	(0.041)
Knowledge of Birth Control (omitted = no known method)				
Knowledge of Inefficient Method Only	0.01	0.009	-0.023	0.091
	(0.013)	(0.017)	(0.022)	(0.040)**
Knowledge of Efficient Method	0.036	0.042	0.022	0.050
	(0.006)***	(0.006)***	(0.007)***	(0.015)***
Region Fixed Effects	YES	YES	YES	YES
Maternal Age Fixed Effects	YES	YES	YES	YES
Year Fixed Effects	YES	YES	YES	YES
Regression Statistics				
Observations	133,428	49,559	41,967	23,075
Adjusted Rsquared	0.06	0.05	0.10	0.24

Notes:

* significant at 10%; ** significant at 5%; *** significant at 1%

Absolute values of robust standard errors appear in parenthesis. The sample includes non-foreign born women aged 15-49 at the time of the Sri Lanka World Fertility Survey in 1975. The unit of observation is a woman year starting at the age of 15 for Columns (1) - (3) and in the year after the first live birth in Column (4).

Table 3 - Survival

Dependent Variable:	Survival Until Age 1			Survival Until Age 5		
	All Births	First Births	Non-First Births	All Births	First Births	Non-First Births
Independent Variables	(1)	(2)	(3)	(4)	(5)	(6)
Malaria Rate at Birth	-0.226	-0.427	0.186	-0.317	-0.600	0.246
	(0.186)	(0.203)**	(0.262)	(0.196)	(0.228)***	(0.278)
Additional Covariates						
Twin	-0.231	-0.149	-0.241	-0.245	-0.206	-0.252
	(0.027)***	(0.067)**	(0.028)***	(0.031)***	(0.083)**	(0.033)***
Sex						
Female	0.012	0.024	0.008	0.009	0.025	0.005
	(0.004)***	(0.007)***	(0.004)*	(0.005)*	(0.009)***	(0.006)
Unknown	0.031		0.029	0.06		0.051
	(0.013)**		(0.014)**	(0.016)***		(0.018)***
Years of Maternal Education	0.003	0.004	0.002	0.004	0.006	0.005
	(0.001)***	(0.001)***	(0.001)***	(0.001)***	(0.001)***	(0.001)***
Current Residence (omitted = Urban Residence)						
Rural Residence	0.006	-0.022	0.015	0.000	-0.032	0.012
	(0.005)	(0.009)**	(0.006)**	(0.007)	(0.011)***	(0.008)
Estate Residence	-0.014	-0.046	-0.002	-0.014	-0.04	-0.004
	(0.015)	(0.026)*	(0.019)	(0.019)	(0.032)	(0.025)
Ethnicity (omitted = Sinhala)						
Sri Lanka Tamil	-0.005	-0.011	-0.004	-0.005	-0.010	-0.003
	(0.008)	(0.013)	(0.01)	(0.011)	(0.017)	(0.013)
Indian Tamil	-0.057	-0.037	-0.062	-0.049	-0.027	-0.054
	(0.013)***	(0.028)	(0.017)***	(0.015)***	(0.032)	(0.021)***
Sri Lanka Moor	-0.011	-0.007	-0.014	-0.004	0.007	-0.010
	(0.009)	(0.015)	(0.01)	(0.011)	(0.019)	(0.011)
Other Ethnicity	-0.013	-0.012	-0.013	-0.012	0.000	-0.016
	(0.035)	(0.034)	(0.042)	(0.04)	(0.039)	(0.046)
Region Fixed Effects	YES	YES	YES	YES	YES	YES
Maternal Age Fixed Effects	YES	YES	YES	YES	YES	YES
Birth Year Fixed Effects	YES	YES	YES	YES	YES	YES
Regression Statistics						
Observations	25,823	5,900	19,923	20,911	4,912	15,999
Adjusted Rsquared	0.03	0.03	0.04	0.03	0.03	0.03

Notes:

* significant at 10%; ** significant at 5%; *** significant at 1%

Absolute values of robust standard errors appear in parenthesis. The sample includes births to non-foreign born women aged 15-49 at the time of the Sri Lanka World Fertility Survey. All columns have one observation for each birth. Columns (1) - (3) include births at least one year prior to the survey. Columns (4) - (6) include births at least five years prior to the survey. Columns (2) and (5): all births had a reported sex.

Table 4 - Educational Attainment

Dependent Variable:	Years of Completed Primary Schooling			At Least Minimally Literate			Years of Completed Schooling			Highly Literate		
	Full Sample		Non-Movers	Full Sample		Non-Movers	Full Sample		Non-Movers	Full Sample		Non-Movers
Independent Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Malaria Rate at Birth	-1.790	-1.387	-1.464	-0.265	-0.259	-0.252	-3.757	-1.464	-3.409	-0.325	-0.321	-0.043
	(0.407)***	(0.730)*	(0.860)*	(0.084)***	(0.146)*	(0.184)	(0.807)***	(1.496)	(1.642)**	(0.100)***	(0.173)*	(0.231)
Additional Covariates												
Current Residence												
Rural	-0.248	-0.256	-0.352	-0.029	-0.031	-0.04	-0.763	-0.78	-0.899	-0.056	-0.058	-0.089
	(0.113)**	(0.114)**	(0.200)*	(0.023)	(0.023)	(0.038)	(0.280)***	(0.282)***	(0.506)*	(0.028)**	(0.028)**	(0.054)
Estate	-0.602	-0.603	-1.2	-0.109	-0.109	-0.18	-1.473	-1.478	-2.885	-0.16	-0.16	-0.24
	(0.360)*	(0.360)*	(0.645)*	(0.069)	(0.07)	(0.147)	(0.653)**	(0.652)**	(1.014)***	(0.067)**	(0.067)**	(0.145)*
Chilhood Residence (omitted = City)												
Town	-0.268	-0.271	-0.149	-0.039	-0.040	-0.013	-0.825	-0.834	-0.274	-0.072	-0.073	-0.033
	(0.061)***	(0.062)***	(0.142)	(0.012)***	(0.012)***	(0.030)	(0.159)***	(0.160)***	(0.339)	(0.016)***	(0.016)***	(0.040)
Countryside	-1.117	-1.127	-0.174	-0.201	-0.203	-0.001	-2.567	-2.596	-0.373	-0.262	-0.264	-0.058
	(0.231)***	(0.229)***	(0.570)	(0.055)***	(0.055)***	(0.123)	(0.453)***	(0.450)***	(0.901)	(0.050)***	(0.049)***	(0.132)
Missing Childhood Residence	0.494	0.489		0.086	0.084		0.731	0.682		0.128	0.132	
	(0.261)*	(0.228)**		(0.046)*	(0.040)**		(0.266)***	(0.239)***		(0.055)**	(0.049)***	
Ethnicity (omitted = Low Sinhalese)												
Up Sinhalese	-0.211	-0.21	-0.164	-0.034	-0.034	-0.038	-0.463	-0.462	-0.425	-0.046	-0.045	-0.063
	(0.080)***	(0.080)***	(0.150)	(0.016)**	(0.016)**	(0.031)	(0.166)***	(0.167)***	(0.292)	(0.020)**	(0.020)**	(0.038)
Sri Lanka Tamil	-0.592	-0.594	-0.559	-0.108	-0.109	-0.123	-1.389	-1.383	-1.283	-0.15	-0.151	-0.215
	(0.281)**	(0.280)**	(0.438)	(0.050)**	(0.050)**	(0.08)	(0.543)**	(0.539)**	(0.886)	(0.059)**	(0.059)**	(0.104)**
Indian Tamil	-1.092	-1.086	-1.383	-0.247	-0.246	-0.339	-2.227	-2.211	-2.414	-0.293	-0.292	-0.382
	(0.289)***	(0.287)***	(0.464)***	(0.066)***	(0.065)***	(0.107)***	(0.509)***	(0.505)***	(0.780)***	(0.056)***	(0.055)***	(0.098)***
Sri Lanka Moor	-0.401	-0.394	-0.591	-0.101	-0.1	-0.172	-1.165	-1.143	-1.342	-0.104	-0.103	-0.162
	(0.161)**	(0.161)**	(0.301)*	(0.034)***	(0.034)***	(0.068)**	(0.361)***	(0.361)***	(0.556)**	(0.032)***	(0.032)***	(0.066)**
Burgher	0.542	0.52	0.729	0.106	0.1	0.115	0.963	0.917	1.437	0.173	0.166	0.176
	(0.219)**	(0.222)**	(0.304)**	(0.022)***	(0.022)***	(0.062)*	(0.588)	(0.598)	(1.137)	(0.026)***	(0.027)***	(0.065)***
Malay	-0.429	-0.445	0.056	-0.101	-0.103	-0.088	-0.885	-0.944	-0.194	-0.186	-0.189	-0.176
	-0.461	(0.460)	(0.456)	(0.100)	(0.100)	(0.146)	(0.926)	(0.921)	(1.136)	(0.123)	(0.124)	(0.186)
Other Ethnicity	-1.294	-1.296	-0.422	-0.37	-0.367	-0.131	-2.417	-2.436	-1.085	-0.304	-0.303	-0.063
	-0.869	(0.879)	(0.94)	(0.218)*	(0.218)*	(0.189)	(1.515)	(1.553)	(1.506)	(0.221)	(0.222)	(0.187)
Missing Value	-2.234	-2.229	0.393	-0.463	-0.456	0.074	-4.433	-4.49	-1.406	-0.339	-0.331	0.167
	-1.459	(1.468)	(0.174)**	(0.308)	(0.308)	(0.038)*	(1.473)***	(1.542)***	(0.406)***	(0.268)	(0.264)	(0.050)***
Region Fixed Effects	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Birth Year Fixed Effects	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES
Regional Time Trends		YES			YES			YES			YES	
Regression Statistics												
Observations	5,822	5,822	2,079	5,822	5,822	2,079	5,822	5,822	2,079	5,822	5,822	2,079
Adjusted Rsquared	0.16	0.14	0.16	0.13	0.12	0.11	0.17	0.17	0.14	0.14	0.13	0.11

Notes:

* significant at 10%; ** significant at 5%; *** significant at 1%

Absolute values of robust standard errors appear in parenthesis. The sample includes non-foreign born women aged 18-49 at the time of the Sri Lanka Demographic and Health Survey in 1987. All columns are cross sectional data.

Table 5 - Second Generation Fertility

Dependent Variable:	Probability of Birth		Fraction of Live Births Who Survive to Age 5	
	Full Sample	Non-Movers	Full Sample	Non-Movers
Independent Variables:	(1)	(2)	(3)	(4)
Malaria Rate at Maternal Birth	0.122	0.151	-0.022	0.014
	(0.016)***	(0.029)***	(0.028)	(0.050)
Additional Covariates				
Current Residence (omitted = City Residence)				
Rural	0.008	-0.004	-0.008	-0.014
	(0.004)*	-0.009	(0.009)	(0.017)
Estate	-0.009	-0.048	0.019	0.043
	(0.008)	(0.020)**	(0.015)	(0.029)
Chilhood Residence (omitted = City Childhood				
Town	0.007	0.006	0.013	0.016
	(0.004)**	(0.009)	(0.007)*	(0.017)
Countryside	0.005	0.034	0.022	0.005
	(0.009)	(0.016)**	(0.015)	(0.031)
Missing Childhood Residence	0.069	0.000	0.045	0.000
	(0.029)**	(0.000)	(0.020)**	(0.000)
Ethnicity (omitted = Low Sinhalese)				
Up Sinhalese	0.009	0.007	-0.005	0.005
	(0.004)**	(0.006)	(0.005)	(0.009)
Sri Lanka Tamil	0.046	0.064	-0.041	-0.037
	(0.009)***	(0.011)***	(0.020)**	(0.026)
Indian Tamil	0.021	-0.003	-0.098	-0.102
	(0.010)**	(0.020)	(0.022)***	(0.043)**
Sri Lanka Moor	0.026	0.015	-0.016	-0.023
	(0.006)***	(0.006)***	(0.011)	(0.016)
Burgher	0.023	-0.021	0.018	0.039
	(0.020)	(0.018)	(0.018)	(0.019)**
Malay	0.003	-0.029	-0.021	0.028
	(0.023)	(0.019)	(0.037)	(0.015)*
Other Ethnicity	0.016	0.023	-0.037	-0.046
	(0.013)	(0.029)	(0.049)	(0.077)
Missing Value	0.044	0.027	0.023	0.017
	(0.003)***	(0.004)***	(0.010)**	(0.013)
Region Fixed Effects	YES	YES	YES	YES
Age Fixed Effects	YES	YES	YES	YES
Year Fixed Effects	YES	YES	YES	YES
Regression Statistics				
Observations	120,186	41,315	4,185	1,471
Adjusted Rsquared	0.19	0.20	0.01	0.02

Notes:

* significant at 10%; ** significant at 5%; *** significant at 1%

Absolute values of robust standard errors appear in parenthesis. The sample includes non-foreign born women aged 18-49 at the time of the Sri Lanka Demographic and Health Surveys. Columns (1) and (2): Panel data with one observation for each woman-year starting at age 15. Columns (3) and (4): cross section data.

Map 1 - Sri Lanka Average Spleen Rates 1937 - 1941