The Multigeneration Effects of Malaria Eradication on Economic Growth*

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Abstract

The malaria eradication campaign in Sri Lanka in the mid-twentieth century reduced malaria incidence from almost 97% in 1935 to 17 cases in 1963. This paper combines this campaign with two household surveys to identify the effect of this eradication on fertility and human capital accumulation on two successive generations. Contrary to theories of the demographic transition and a movement along the quantity-quality trade-off, the initial effect of this disease eradication is an increase in fertility. However, these larger cohorts also have fewer births and accumulate more human capital despite the congestion effects. Therefore, while the initial effect might be detrimental to economic growth, increased education and lower subsequent fertility will at least partially mitigate this negative growth effect.

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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 30-50% of inpatient admissions.[1] 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, exploring the relationship between the changing disease environment and fertility in those of child bearing age during the eradication campaign and the survival, eventual educational attainment, and fertility of their offspring. Through the use of a repeated cross section this multigenerational approach provides a more complete view on the short and longer terms effects of malaria eradication (and continued malaria endemicity). I find a transitory fertility *increase* from malaria eradication; focusing only on a single generation would miss the subsequent increased education and fertility decline and lead to incorrect conclusions about the total malaria effect.

The effects of malaria on the quality of life and economic growth and development in sub-Saharan Africa have recently received renewed attention from both international organizations and in the economics literature. In addition to its reduction being one of the United Nations Millennium Development Goals, the Roll Back Malaria Program (a joint project of the World Health Organization, United Nations Development Program, United Nations Children's Fund, and the World Bank) and the Malaria Vaccine Initiative (partially funded by the Bill & Melinda Gates Foundation and The Wellcome Trust) are also focusing on the reduction of the malaria disease burden in Africa.

Within the economics literature there is a substantial body of work at the microeconomic and macroeconomic level quantifying malaria's effects. At the microeconomic level, cost-of-illness methodology is used to create a per person accounting of the costs of malaria.[2] The unifying theme in this literature is that malaria exerts a significant burden at the individual level. These studies fail to capture non-instantaneous costs of malaria infection and the externalities from malaria in the community since by definition they focus on specific instances of malaria infection. Alternatively, the macroeconomic literature combines cross-country growth regressions with various measures of malarial intensity within a country, in many cases finding a strong correlation between the level of malaria infection in a country and the growth or level of a county's GDP per capita, and in one case finding no relationship. Gallup and Sachs (2001) estimate that severe malaria in 1965 is

[1] Roll Back Malaria (2005).

[2] Shepard, Ettling, Brinkmann, and Sauerborn (1991) contains a survey of work in this field in Africa. Conly (1975).

associated with 1.3% lower GDP growth per annum from 1965 to 1990. In direct contrast to these results, Acemoglu and Johnson (2005), also using cross country methodology, find no discernible gain in either the average levels of education or log per capita GDP with the improvements in the disease environment that resulted from international health interventions. My multigenerational approach reconciles the seemingly contradictory findings of Acemoglu and Johnson (2005) and the cost of illness methodology: while there are long run gains to education, the magnitude of these gains may not be of a sufficient size to quickly overcome the population increase that accompanies eradication.

In order to identify the multiple effects of malaria (and malaria eradication) I rely on the first international interest in malaria eradication: the WHO malaria eradication program that followed the Second World War. Departing from both the existing growth and development literatures, in this paper 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. 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 a country based on climatic and geographic factors and the exogenous reduction of malaria during the national malaria eradication campaigns. This identification strategy isolates the malaria effect from other nationwide trends and regional fixed effects.

This study contributes to the broader literature on the importance of health in human capital accumulation and the debate on the sources of underdevelopment in sub-Saharan Africa. Furthermore, it offers additional support to the importance of the Roll Back Malaria campaign and other undertakings to reduce malaria incidence throughout the world.

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 estimation strategies, 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. These environmental and geographical limitations of the vector and the parasite combine

to form a portion of the identification strategy. 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 (approx. 80 inches or 2000 mm.) of rainfall is also necessary to provide the standing water essential for vector breeding, but too much rainfall can eliminate suitable breeding sites. At altitudes above 3281 ft. (1000 m.) there is at most minimal malaria incidence.

In a given region, if malaria is present it can either be *epidemic* or *endemic*. In epidemic areas malaria transmission is seasonal or less frequent, with breaks within the year or between years in the transmission cycle of sufficient duration so that the majority of parasites in infected individuals die without being replaced by new parasites.[3] These breaks in transmission are typically due to climatic conditions that are either not conducive to vector breeding (insufficient or abundant rain) or parasite incubation (temperatures below 60^0 F). With each new transmission season, malaria-free individuals are infected resulting in severe illnesses and death. This cycle of infection, elimination of infection, and re-infection does not provide the length of continuous exposure necessary for individuals to develop acquired immunity. In these communities any malaria outbreak can be deadly for individuals of all ages.

Where malaria is endemic, infections occur throughout the year or breaks in transmission are not sufficient for individuals to rid themselves of the parasite. In endemic regions most of the adult population has complete acquired immunity that confers upon them resistance to severe malaria symptoms but does not prevent an individual from being a carrier of the parasite or experiencing the resultant lethargy, anemia, and exhaustion from a long-standing infection.[4] Children and pregnant women in endemic regions do not have acquired immunity, and thus remain at high risk for severe symptoms and death with any infection.

The most common manifestation of malaria is relapsing fever with spikes on alternative days. Anemia, lethargy, tremors, headaches, and general aches and pains are also common symptoms with abdominal pain and diarrhoea appearing less commonly. Severe malaria has many more serious complications: unrousable coma, convulsions, hypoglycemia, respiratory distress, renal failure, abnormal bleeding, and jaundice. In pregnant women malaria often results in low birth weight full-term births or pre-term labor and can result in spontaneous abortions and still-births.

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

[3] Malaria parasites rarely live longer than 150 days, but relapses 52 years after the last known infection have been recorded.

[4] Pampana (1969).

rates. The spleen rate reflects the percentage of school children displaying an enlarged spleen, a common indication of long-standing malaria infections. The Wet Zone is the area immediately surrounding Colombo on the South West coast of the country. the Dry Zone is in the North and on the South East coast.[5] 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). Detailed information on these time series can be found in Section A. Two highly correlated measures of malaria, the spleen rate and the incidence rate, are plotted in Figure 1. The infection rates 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 was going to prove more difficult that 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.

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

3 Conceptual Framework

Since malaria infections impact fertility and child survival and health, in order to quantify the total malaria effect, two generations must be considered: those of child bearing age during the malarious period and those born during the malarious period.

[5] 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 cannot be included in the analysis.

3.1 Instantaneous Effects

3.1.1 Infant and Child Survival

Starting from conception, malaria infections can reduce the survival probability of live births through decreased birth weight.[6] All pregnant women living in epidemic or endemic malarial zones, even those who have acquired immunity prior to pregnancy, are at risk of severe malarial illness. Malaria that was acquired but non-symptomatic before pregnancy can become active once the infected is gravidae. Malaria can occur at any time during pregnancy. Pregnancy malaria can cause anemia and low birth weight from both fetal growth retardation and premature delivery.[7] A significant reduction in birth weight can occur even if the mother exhibits no outward symptoms of malaria, since the malaria parasite disrupts nutritional transmission from the mother to the fetus. The incidence of low birth weight, weight of less than 2500 grams (5.5 lbs), can increase by over 50% in areas of intense malaria transmission.[8] Malaria infections are correlated with a reduction in birth weight of 290 to 669 grams (0.6 to 1.5 lbs.).[9] These effects are of larger magnitude for the primigravidae than those of higher order parity.[10]

Malaria also effects survival well into infancy and childhood. In all children under the age of five, malaria may develop rapidly. Infection rates tend to be higher in children than in adults, and in endemic regions malaria symptoms are concentrated in pre-school children.[11] In children, the serious malaria symptoms of cerebral malaria, severe anemia, respiratory distress, and hypoglycemia are more common than in adult suffers. In five percent of childhood malaria infections the plasmodium infects the brain and central nervous system, obstructing blood vessels and causing cerebral malarial that if untreated results in certain death. In an area of Tanzania of intense malaria transmission, infant infection rates of 2.1 malaria episodes per child-year were recorded, indicating that in regions of similarly intense transmission, for each child there is a 10.5% probability of cerebral malaria per year.[12] Survivors of severe childhood malaria may suffer epilepsy and cerebral palsy, increasing their risk for premature mortality. Those under the age of three are especially susceptible to severe malaria complications. Additionally, repeated infections increase the susceptibility of children to other illness such as respiratory infections and diarrhoea.[13] All

[6] In a holoendemic area of rural Malawi, malaria infections while in utero and after birth contributed to 45.7% of preinatal deaths (fetal or infant deaths occurring from 28th week of pregnancy up to the seventh day after birth), 80% of all neonatal deaths (infant deaths occurring within the first two months after birth), and 37.8% of infant mortality.(Mcdermott, Wirima, Steketee, Breman, and Heymann (1996))

[7] Duffy and Desowitz (2001).

[8] Duffy and Fried (2001).

[9] See summaries of Goth (1881), Chairleoni (1886), and Le Dantec (1929) in Duffy and Desowitz (2001).

[10] McGregor (1984).

[11] Onwujekwe, Chima, and Okonkwo (2000) estimate 70% of all malaria cases in five holoendemic rural communities in Nigeria are contracted by children aged 0 to 14 years. Holding and Snow (2001).

[12] Kitua, Smith, Alonso, Masanja, Urassa, Menendez, Kimario, and Tanner (1996).

[13] World Health Organization (2003).

health effects beyond those in utero will not differentially change parity specific survival.

Indirectly, malaria infections reduce labor capacity, limiting the amount of resources available for expenditure on nutritional intake. Lack of nutrition can also reduce survival probabilities.

3.1.2 Fertility

A priori the direction of the effects of malaria on the total number of live births per woman is unclear. Malaria eradication increases income, reduces the price of a surviving child, and lifts prior biological constraints on total fertility. From these three mechanisms, malaria should increase the total fertility rate. Conversely, the increased certainty about child survival and movement along the quality-quantity trade-off would serve to reduce the total fertility rate.

Malaria reduction increases household income as individuals no longer suffer forgone income due to absenteeism or anemic lethargy nor are expenditures on treatments required. The most important times in the agricultural calendar, the planting and harvesting seasons, are also the time of the highest levels of malaria in Sri Lanka. Since children are a normal good, the pure income effect increases the demand for children.[14]

A second component of this increase in income is an increase in the opportunity cost of time. Since child rearing is a time intensive activity, the increase in income causes children to become relatively more expensive versus other consumption goods. The resulting substitution effect could result in a reduction in the desired number of children.

As stated in Section 3.1.1, reductions in malaria increase the survival probability of children both directly through the elimination of in utero and childhood infections and indirectly through increased income available for nutrition. Since parents target the number of surviving children, the increase in the survival probability reduces the price of each survivor. In the Barro and Becker (1989) framework in which altruistic parents optimize over their own consumption, their number of surviving children, and the utility of each child, a decrease in malaria decreases the price of each child. Since children are a normal good, households, therefore, have a higher number of surviving children. Extensions of this model to include stochastic survival probabilities and sequential fertility choice in Doepke (2005) yield a similar prediction for a an increase in the surviving fertility rate with falling child mortality, but also predict a decrease in total fertility. In contrast with these models, the "hoarding" fertility model of Kalemli-Ozcan (2003) predicts a strong negative effect on fertility when there is more certainty regarding child survival. The removal of the impetus of precautionary fertility yields this hoarding effect. This change in survival has an unclear net effect on fertility.

Malaria directly reduces fecundity, the ability to have a live birth, through an increase in the

[14] The effect of changing income on survival, fecundity, and quality preferences are addressed in the remainder of this section.

probability of spontaneous abortions and stillbirths.

A final effect that malaria can have on fertility is through a move towards preferring quality over quantity of children. This is intrinsically linked to the reduction in mortality that accompanies malaria eradication. As mortality falls, the certainty associated with child survival increases the return to an investment in child quality for altruistic parents who value the future utility of their children or who rely on them for old age support.

The relative magnitudes of these potentially counteracting effects determine the direction of the net malaria effect on fertility.

3.2 Second Generation Effects

3.2.1 Education

Previous studies have quantified the importance of other disease eradications on school attendance. Looking at hookworm in Kenya, Miguel and Kremer (2004) found a seven percentage point increase in primary school participation and a one quarter decrease in absenteeism when students were treated for hookworm infections. Bleakley (2004) used hookworm and education data from the American South to show that schooling enrollment increased by five percentage points when hookworm was eradicated from a county with a 50% infection rate, and that a student infected with hookworm is 23% less likely to attend school.

In the case of malaria, there are two periods in which it can directly affect educational accumulation: (1) pre-school infections: infection of the mother while an individual is in utero and infection in early childhood and (2) school aged infection. Indirect effects of malaria on education occur throughout the pre-school and school aged years. Because of data availability, both direct and indirect effects are combined in the estimation strategy. Those born after eradication should, in expectation, have higher human capital accumulation.

Malaria's Effects in Utero and Early Childhood 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.[15] Based on data from monozygotic twins, Behrman and Rosenzweig (2004) find that an increase in birth weight of 1 lb. results in 0.3 more years of schooling and Black, Devereux, and Salvanes (2005) find an increase in the probability of high school completion of 3 percentage points with an increase in birth weight of 2.2 lbs.. Specific to premature birth, regardless of the cause, children born prematurely (less than 37 weeks) are 2 -

[15] McCormick, Brooks-Gunn, Workman-Daniels, Turner, and Peckman (1992).

4 times more likely than full term children to experience failure in school.[16] 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. The nutritional, hormonal, and metabolic environment of the mother may permanently "program" the physiology of the offspring resulting in lifelong effects. Undernutrition has been found in animal studies to reduce total cell numbers and alter organ structures, hormone response levels, and metabolism.[17] These permanent physical and mental impairments adversely affect an individual's likelihood of advancing through or attending school.

In all children under the age of five, malaria may develop rapidly. Infection rates tend to be higher in children than in adults, and in endemic regions malaria symptoms are concentrated in pre-school children.[18] In children, the serious malaria symptoms of cerebral malaria, severe anemia, respiratory distress, and hypoglycemia are more common than in adult suffers. In five percent of childhood malaria infections the plasmodium infects the brain and central nervous system, obstructing blood vessels and causing cerebral malarial. In an area of Tanzania of intense malaria transmission, infant infection rates of 2.1 malaria episodes per child-year were recorded, indicating that in regions of similarly intense transmission, for each child there is a 10.5% probability of cerebral malaria per year.[19] Among the survivors of cerebral malaria, 16% had evidence of neurological damage upon hospital discharge.[20] Survivors of severe childhood malaria may suffer learning impairments, speech disorders, behavioral disorders, blindness, hearing impairment, epilepsy, and cerebral palsy. Those under the age of three are especially susceptible to severe malaria complications. Even without severe symptoms, nutritional intake is interrupted in the presence of a malaria infection, impairing cognitive development.[21] Additionally, repeated infections increases the susceptibility of children to other illness such as respiratory infections and diarrhoea that can also interrupt nutritional intake and make a child too prone to illness to attend school.[22]

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 develop-

[16] Holding and Snow (2001), includes an limited number of additional controls.

[17] Barker (1994).

[18] Onwujekwe, Chima, and Okonkwo (2000) estimate 70% of all malaria cases in five holoendemic rural communities in Nigeria are contracted by children aged 0 to 14 years. Holding and Snow (2001).

[19] Kitua, Smith, Alonso, Masanja, Urassa, Menendez, Kimario, and Tanner (1996).

[20] Holding and Snow (2001). Those who have cerebral malaria and are not admitted to a hospital face almost certain death.

Based on a rough calculation, in areas of intense transmission, 1.68% of young children are expected to suffer this damage annually (assuming that all children with cerebral malaria receive timely hospital care and survive).

[21] Rowland, Cole, and Whitehead (1977). Shiff, Checkley, Winch, Minijas, and Lubega (1996). McKay, Sinisterra, McKay, Gomez, and Loreda (1978).

[22] World Health Organization (2003).

mental effects of early life malaria infections may not be realized until higher order functions are required of individuals during schooling age.[23]

Malaria's Effects on School Aged Children Malaria can also be detrimental to human capital accumulation during the school aged years. A series of studies performed in areas of endemic malaria found that children aged 5 to 9 have between 0.25 and 2.3 malaria attacks per annum and individuals aged 10 to 20 have on average 0.1 to 1.3 attacks per annum.[24]

Individuals with low level anemia, a common symptom of mild or long-standing malaria infections, exhibit lethargy and an inability to concentrate making attending and staying in school much more difficult. Individuals with severe anemia or other malaria symptoms are unable to attend school. Extensive absenteeism or lethargy while at school can cause children to fall behind, making dropping out inevitable. A study in Kenya found primary school students on average miss 20 school days (11% of the school year) due to malaria and secondary school children miss 8 days (4% of the school year) due to malaria. In Nigeria students on average miss 3 to 12 days (2% to 6% of the school year) due to malaria.[25] Other studies in Africa find malaria accounts for 3% - 8% of school absenteeism and 13 - 50% of preventable medical absenteeism.[26] Among school aged children, severe malaria is associated with depression, psychotic disorders, memory impairments, irritability, and violence.[27] The presence of malaria parasites, even when the individual is asymptomatic has been shown to be correlated with reduced performance in a test of fine motor skills when controlling for age, socioeconomic background, and nutritional status. Once treated, those who previously had the highest parasite load improved the most in the fine motor and memory tests.[28]

Effects Throughout Infancy and Childhood Even if a child is able to remain malaria free, there can be negative effects on a child's education due to illness in the family or community. These effects are common throughout infancy and childhood. At the community level, the increased level of infant and child mortality from malaria can result in uncertainty about the number of live births who will survive to adulthood. "Child hoarding" and an increased dependency ratio leave fewer resources available for each child. Expenditure on treatment and forgone employment income reduce the total income available to be spent on nutrition and schooling. If the one of the primary financial supporters of the family falls ill, the family cannot always fully compensate for this loss in income. Studies in Kenya and Nigeria found lost income and health care costs related to

[23] Holding and Snow (2001).
[24] Brooker, Guyatt, Omumbo, Shretta, Drake, and Ouma (2000).
[25] Leighton and Foster (1993).
[26] Brooker, Guyatt, Omumbo, Shretta, Drake, and Ouma (2000).
[27] Holding and Snow (2001).
[28] Serouri, Grantham-McGregor, Greenwood, and Costello (2000).

malaria amount to 5% to 19% of total household income.[29] Specifically in Sri Lanka, the annual timing of the highest malaria transmission coincides with the important agricultural planting and harvesting season, greatly impacting a family's income if a member becomes too ill to work or suffers from a decrease in work efficiency from the disease. Therefore, even if not directly afflicted, the ambient malaria incidence rate can have dire effects on income available for early nutritional requirements. Furthermore, because of the appreciable probability of death of children, parents may find investment in formal human capital accumulation an unaffordable luxury. Children might also be kept from school in order to care for an ill family member or take up some of the tasks of an ill adult in lieu of school attendance.[30]

3.2.2 Fertility

Malaria eradication in childhood exerts two opposing forces on total lifetime fertility: (1) because of the negative correlation between total fertility and education, increased education could reduce fertility and (2) biologically, healthier women are more fecund.

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 allows for proper identification.

4.1 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.

In order to properly identify the effects of malaria one cannot simply compare the outcomes from two regions with different levels of malaria since there could be unobservables that are correlated with the malaria levels, biasing the estimate of the malaria effect. Adding a time dimension is not a sufficient solution for identification since region-cohort unobservables could again be biasing the results. The malaria eradication program in Sri Lanka, an exogenous change in the malaria

[29] Leighton and Foster (1993). Onwujekwe, Chima, and Okonkwo (2000).

[30] Minders in Nigeria lost 5 to 9 days of productivity per month to care for malarial family members. (Onwujekwe, Chima, and Okonkwo (2000)).

rates that brings malaria across all regions to zero, provides the quasi-experiment necessary for identification. The assumptions necessary for identification are (1) heterogeneous indigenous rates of malaria infection, (2) a well defined treatment period, and (3) exogenous implementation of the program.[31]

Heterogeneous indigenous rates of malaria infection: The treatment and control groups are determined by the pre-existing 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. Map 1.1 demonstrates this distribution.

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. Section 6 contains estimates including regional time trends to control for any regional linear convergence.

A well defined treatment period: The precise timing of the success of intervention is based on rates of infection. In practice, the malaria rates did not fall immediately to zero, but in each region drastic reductions we 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.[32]

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

[31] Section B provides a more formal derivation of the identification strategy.

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

as to the location to spray and precise concentrations of DDT to use. Within the rigid framework there was no provision for sub-national decision making. "Local" decision making was undertaken at the national level. The primary source of funding was either bilateral or multilateral aid.

Since these conditions are met, a variation of the following equation is estimated

$$Y_{ijt} = \alpha + \beta malaria_{jt} + \delta_j + \delta_t + X'_{ijt}\Gamma + \varepsilon_{ijt}$$

at the individual level for each individual i in region j at time t with region, δ_j, and time, δ_t, fixed effects, replacing the additional covariates, X_{ijt}, and outcome of interest, Y_{ijt}, as appropriate. The outcomes estimated are the total number of live births, the total number of surviving births, the probability of a live birth, hazard model of first birth, child survival to ages one and five, years of completed education, literacy, and fertility by age 30. The coefficient of interest is β, measuring the incremental change in the outcome from the changes in the malaria rates over time and across different regions. 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 (control group) through the varying levels of $malaria_{jt}$, I am able to isolate the effect of malaria eradication from other country-wide and time specific effects.

4.2 Data

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

4.2.1 Survey Data

The individual data on fertility and child survival are from the World Fertility Survey conducted in 1975. It is a retrospective fertility survey of ever married women aged 12 to 50, designed to be nationally representative. From this cross section of 6,810 women there are 27,076 live births. Of these births, 25,811 were at least one year old prior to the survey. Select summary statistics appear in Table 1.

The individual data on educational outcomes and second generation fertility outcomes are from the DHS-I 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 5,843 ever married women born in Sri Lanka between 1937 and 1968. The sample of non-movers is 2,086 ever married women.

Each woman is assigned a birth year and region of residence based on her responses. 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. Since malaria rates are assigned at the regional level, mismeasurement should be minimal. I also present the results separately for those who have never moved from their birthplace. Means for the dependent variables of interest can be found in Table 2.

4.2.2 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.[33] Precisely, 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. 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.[34] 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 has the highest historical malaria incidence rates. 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. The pre-eradication district level spleen rates appear in Map 1.1.

5 Estimation

To estimate the effects of malaria on fertility, I use several specifications. The estimates of the effect of malaria on total live births, surviving births, and the probability of birth in a given year address the magnitude and direction of the total malaria effect. Estimates of 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 total years of completed education and literacy.

[33] Within a given region there will be variation in the true realized exposure. Since this is classical measurement error, uncorrelated with the other independent variables, the coefficient estimate on the malaria measure will be biased towards 0.

[34] Newman (1965), Meegama (1986), and Konradsen, Amerasinghe, can der Hoek, and Amerasinghe (2000).

5.1 Fertility

To estimate the effect of malaria on the total number of live births, I estimate the following equation:

$$B_{ijc} = \alpha + \beta^B \left(\frac{\sum_{t=1}^{T} malaria_{jt}}{T} \right) + \delta_j + \delta_c + X'_{ijc}\Gamma + \varepsilon_{ijc} \tag{1}$$

where B_{ijc} is the cumulative number of live births to woman i in region j as a member of cohort c, δ_j are region fixed effects, δ_c are cohort fixed effects, and X_{ijc} are individual level controls including the number of years in a marriage, and indicators for birth control knowledge, birth control use, residing in an urban setting, ethnicity, and being born in an urban setting.[35] The average annual malaria rate, $\frac{\sum_{t=1}^{T} malaria_{jt}}{T}$, is the sum of the region j malaria rates from the year of marriage, 1, to the year of the survey or dissolution of the marriage, T, divided by the total number of years in a marriage, T. 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 are assumed to have the same error structure.

In contrast to a theory of a quality/quantity trade-off or a shift towards fewer children with increased certainty of survival in Section 3.1.2, the number of live births increases as the malaria rate falls as can be seen in Column 1 of Table 3.[36] While the point estimate is quite large, the maximum average malaria rate experienced by a woman in the sample over her years in a union is 18.8%, reduction of malaria from this level to zero would increase the total number of live births per woman by 2.57. This increase in fertility indicates that the biological, income, or substitution effects from increased infant survival are dominating other potential responses to malaria eradication.

Intuitively, households instead target the number of surviving children instead of live births. When Equation (1) is re-estimated replacing live births with births who survive until age 10, an approximate measure of the number of surviving children,

$$N_{ijc} = \alpha + \beta^N \left(\frac{\sum_{t=1}^{T} malaria_{jt}}{T} \right) + \delta_j + \delta_c + X'_{ijc}\Gamma + \varepsilon_{ijc}, \tag{2}$$

the malaria rate also has a negative effect on this measure as can be seen in Column 2 of Table 3.[37]

[35] While for the majority of cases women have a single continuous span of time in a union, for those 145 cases in which there is a break in union duration, the malaria rate is averaged only over those years in which the respondent was in a union. Of the 26,698 total births, less than 0.5% took place when the respondent was not in a union. These births are included in the total number of live births and the malaria rate in that year is also included in the malaria incidence average.

[36] The estimates that appear in Table 2 are least squares estimates because of the incidential parameters problem with non-linear estimation procedures with fixed effects (see Lancaster (2000)). Marginal point estimates from a non-linear specification are provided as robustness checks in Section 6.

[37] Survival until the age of ten is used because of data limitations. Those who died prior to the survey but aged 10 years or older are all coded in the same manner.

A fall in the malaria rate over the reproductive period from 18.8% to zero increases the number of surviving births by 2.25. This negative relationship again points to the dominance of biological, substitution, or income effects.

To further explore this increase in fertility, I also estimate a probability of birth model. For this specification the unit of observation is a woman-year. I use two separate samples: all women-years above the age of thirteen and only those women-years in which the woman was in a marriage, since marriage formation could be a response to malaria induced changes in fecundity. Formally,

$$P(B_{ijt}) = \alpha + \beta^{PB} malaria_{jt} + \delta_j + \delta_t + \delta_c + X'_{ijc}\Gamma + \varepsilon_{ijt} \tag{3}$$

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_c are maternal cohort fixed effects, and the X_{ijt} term includes fixed effects for the total number of prior child births and deaths, indicators for birth control knowledge and usage, the number of years in a union, indicators for births and deaths in the previous year, and the level of maternal education. The results of the linear probability estimation appear in Columns 3 and 4 of Table 3. Consistent with the total number of live births being a decreasing function of the malaria rate, the probability of a live birth in a particular year is also a negative function of the malaria rate for both samples; the point estimate is -0.170 for the thirteen and older sample and -0.342 for the married sample. The highest regional spleen rate in a year over both samples was 50.1%. Therefore, reducing malaria from this level to zero would increase the probability of a live birth by 8.5 percentage points for an individual over the age of thirteen and by 17.1 percentage points for an individual who was married.

To estimate the effect on transition to initial parity, Equation (3) is re-estimated as a hazard model where the hazard of having a live birth starts at either thirteen or marriage and the woman is removed from the sample upon a live birth. For this model, the additional covariates included besides the fixed effects are birth control knowledge and usage, an indicator for being married, the number of years in the marriage, and maternal education. The results from the linear probability estimation appear in Columns 1 and 2 of Table 4. In both samples, malaria exerts a negative and significant effect on the transition to initial parity; malaria eradication shortens the transition to initial parity. The magnitudes are smaller than those from the more generalized probability of birth model.

The sample size shrinks by 2,580 because women whose first child was born after ten years prior to the survey were eliminated as they could not have a "surviving child" at the time of the survey.

5.2 Child Survival

The child survival estimation is a linearized probability model:

$$P(survival_{ijc}) = \alpha + \beta^S malaria_{jc} + \delta_j + \delta_c + X'_{ijc}\Gamma + \varepsilon_{ijc}. \quad (4)$$

The additional individual controls in X_{ijc} are maternal education and separate indicators for the sex of the child, the birth year of the mother, the birth order, if the birth was part of a multiple birth, and urban or rural residence. Separate models are estimated to establish the effect of malaria on the probability of survival to age one and to age five and by birth order. The point estimates from the linearized probability model appear in Table 4. Surprisingly, the malaria rate's effect on child survival is insignificant for the sample including all births with negative and insignificant point estimates in Columns 1 and 3. 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. The highest spleen rate over the sample is of live births is 44.9%, therefore eliminating malaria from this level would, in expectation, increase the probability of survival to age one by 19.2 percentage points and the probability of survival to age five by 26.7 percentage points for the first born. The difference in results between the first birth and all births suggests that malaria's effect on survival is primarily coming through the direct pre-natal health effects since once an infant is born there is no difference in their post birth malaria exposure.

5.3 Second Generation Education

The general model estimated is

$$education_{ijc} = \alpha + \beta^E malaria_{jc} + \delta_j + \delta_c + X'_{ijc}\Gamma + \varepsilon_{ijc} \quad (5)$$

where $malaria_{jc}$ is the regional spleen rate in the region and time of the respondent's birth and the X_{ijc} matrix includes ethnicity indicators to control for within region racial segregation, childhood place of residence (city, town, countryside, and missing) indicators, and sector (urban, rural, and estate) indicators. Educational attainment is measured three ways: years of completed 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 6 Columns 1 and 2 contain the tobit estimates of the marginal effects of Equation (5) with years of education as the dependent variable for the full and non-mover samples respectively. The point estimates for the two samples are similar. As expected the estimate of the coefficient on the regional spleen rate is negative and significant. A reduction in the spleen rate from 100% to 0% would increase expected years of completed education by 3.71 years. The highest regional spleen 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 the spleen rate from 59.7% to 0% would lead to an increase in the expected number of years of completed education of 2.21 years. In the Irrigated Dry Zone, the average number of years of completed education increased from 3.20 years for those born 1937 - 1939 to 7.36 years for those born 1967 - 1969. Malaria eradication therefore accounts for 53.2% of the increase in completed education over that period.

Table 6 Columns 3 - 6 contain the marginal effects from probit estimations of Equation (5) with the two different literacy measures as dependent variables. Similar to the results for education, the regional spleen rate has a negative and significant effect on literacy attainment. The result is not distinguishable from 0 for the non-mover sample, Columns 4 and 6. Based on the estimates in Columns 3 and 5 and the 59.7% maximum regional spleen rate, the expected probability of being able to read easily increases by 14.0 percentage points and that of being able to read easily or with some difficulty by 9.5 percentage points. Comparing the cohorts born in the irrigated dry zone in 1937 - 1939 to those born in 1967 - 1969, there was a 40.4 percentage point increase in the probability of being able to read easily and a 25.0 percentage point increase in the probability of being able to read easily or with some difficulty; I attribute 34.6% and 38.0% respectively of that gain to malaria eradication.

5.4 Second Generation Fertility

Others have noted the negative relationship between levels of maternal education and fertility. Since I have shown that malaria reduction increases education, I will estimate a reduced form effect of early life malaria exposure on future fertility.[38] The higher level of education that results from malaria eradication will raise the opportunity cost of children and increase knowledge about contraception, both of which tend to lower fertility. Counteracting this effect, reducing a woman's malaria exposure in childhood will lead her to be healthier, with higher fecundity as an adult. 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 we use are total number of children born to a respondent by age 30 and the percentage of births who survive to their fifth birthday. I estimate

$$fertilityby30_{ijc} = a + \beta^F malaria_{jt} + \delta_j + \delta_c + X'_{ijc}\Gamma + \zeta_{ijc} \tag{6}$$

where $fertilityby30_{ijc}$ is the total number of live births by age 30 to respondent i living in region j born in cohort c and all other notation and controls are the same as that from Equation (5).[39] A

[38] See Lucas (2006) for the effects of the contemporaneous malaria rate on fertility.

[39] We look at fertility to age 30 rather than completed fertility, typically 45, because using the latter variable in our 1987 sample would require excluding women born after 1942, thus eliminating all women born after the eradication

higher regional childhood malaria rate results in increased female fertility as can be seen in Table 7. Reduction of the spleen rate from the highest observed to 0% reduces the total number of births by 1.28 child per woman by the age of 30. Cohorts born between 1937 and 1939 in the dry irrigated zone had an average of 3.90 live births by age 30. In contrast, those born in the same region between 1955 and 1957 had an average of 2.64 live births. Our results suggest that this entire drop in fertility can be attributed to malaria. Therefore the increased levels of fertility found in the first generation in Section 5.1 do not continue for those born during the eradicated period.

For the percentage of children who survive to age 5 we estimate

$$alive5_{ijc} = \alpha_0 + \beta^A malaria_{jt} + \delta_j + \delta_c + X'_{ijc}\Gamma + u_{ijc} \tag{7}$$

where $alive5_{ijc}$ is the percentage of children per woman who had survived until their fifth birthday.[40] The other variables and all subscripts are the same as those that appear in Equation (6). 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 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.

6 Robustness

To test for robustness of the above estimates, I use three different techniques: non-linear estimation, the inclusion of regional time trends, and the inclusion on maternal fixed effects where there is more than one observation per woman.

6.1 Non-Linear Estimation

The estimates presented above were based on linear estimates of both probabilities and truncated variables. An alternative, and potentially biased, estimation method is to use non-linear maximum likelihood techniques in lieu of the also potentially biased least squares estimation. Tables 8 - 10 replicate Tables 3 - 5 providing the marginal effects of the tobit estimation in Columns 1 and 2 of Table 8 and all of Table 10 and marginal effects of logit estimation for the remaining columns of Table 8 and Table 9. While the magnitudes of the marginal effects are smaller for the total

of malaria.

[40] $alive5$ 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.

number of live births, the marginal non-linear effects are quite similar for the other outcomes of interest. These results are consistent with the findings above of an increase in the total number of live births, surviving children, and the probability of birth with a decrease in the malaria rate. For the hazard of birth estimations (Table 9), the point estimates for the sample that is limited to those who are married are statistically the same as those in the linear probability model. For the sample that includes all maternal ages above thirteen, the magnitudes of the coefficients are smaller.[41] The differential effects by parity are confirmed with these non-linear estimates. Finally, for the survival estimates, the magnitudes are approximately one half the size, but have the same significance as the linear probability models.

I also re-estimate equations Equations (6) and (7) using tobit estimation. The results are extremely similar for the total number of live births by 30. When the malaria effect on the fraction of live births surviving was only suggestively negative in the original specification, it is now twice the magnitude (within one standard error of the first estimate) and significantly different from zero for the full sample. For non-movers the point estimate remains imprecisely estimated. These estimates appear in Table 11.

6.2 Convergence Between Regions

After allowing for convergence between regions with the inclusion of a region-specific time trend, the direction of malaria's effects and differential outcomes by parity in almost all cases remain as above. Tables 3 - 5 are replicated including a set of $\delta_j * timetrend_t$ or $\delta_j * timetrend_c$ as appropriate as additional covariates. These results appear in Tables 12 - 14. All point estimates maintain their significance (or insignificance). The hazard and survival estimates are quite similar in magnitude with the inclusion of the regional time trends.

After allowing for convergence between regions with the inclusion of a region-specific time trend in the replication of human capital results in Table 15, the effect of malaria on education remains negative and significant. Region specific time trends are added to Equation (5) to estimate

$$education_{ijc} = \alpha + \beta malaria_{jc} + \delta_j + \delta_c + \delta_j * trend_c + X'_{ijc}\Gamma + \varepsilon_{ijc} \tag{8}$$

over the DHS sample. The first six Columns of Table 15 contain the results. While the point estimates remain negative, the standard errors are quite large. The high level of correlation between the sixty five included independent variables results in these large standard errors. An alternative estimation replacing δ_c with $trend_c$, a national level time trend, as is more typical in papers with similar estimation techniques, restores the significant of the point estimates on the malaria spleen rate as can be seen in the final six columns of Table 15. Table 15b contains an additional estimation

[41] Since age at marriage is not censored, Column 7 of Table 3.3 is not estimated using a non-linear technique.

alternative: using a binary variable to indicate malaria eradication instead of the full time series. The six columns in Table 1.7b contain the estimation of

$$education_{ijc} = \gamma_0 + \gamma_1 malarious_c * malaria_{j1937} + \delta_j + \delta_c + \delta_j * trend_c + X'_{ijc}\Gamma + v_{ijc}$$

where $malarious_c = \{1 \text{ if } c \leq 1950, 0 \text{ otherwise}\}$ and $malaria_{j1937}$ is the spleen rate in region j in 1937. The results continue to indicate the importance of malaria for human capital accumulation.

Finally, the results from the inclusion of regional level time trends in Equations (6) and (7) appear in Table 16. The fertility by thirty estimates are well within one standard error of the original estimates, but the point estimate for the non-mover sample is now insignificant. As with the original specification, reduced regional malaria when the mother was young does not have an effect on the survival of her children to the age of 5.

6.3 Maternal Fixed Effects

A final robustness check includes maternal fixed effects in estimates where there are multiple observations per woman to control for potential time invariant maternal heterogeneity. Tables 3.11 - 3.13 contain these estimates. The estimate of malaria's effect on the probability of birth is of similar magnitude as above. For child survival, the point estimates are quite similar to the non fixed effect estimates and remain insignificant when all parities are considered together.

6.4 Sample Size

Since the measures of malaria are at the regional level, having a sample size of between two and five thousand can cause a downward bias in the standard errors The results of aggregating the data into region- (or ethnicity-) year cells and estimating the analogue to Equation (5) appear in Table 20. The results are robust to this aggregation.

6.5 Alternative Explanations

Other authors have noted malaria's role is 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 spleen 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."[42] 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

[42] Gill (1940) p. 215.

claims the implementation of a public health intervention that coincided exactly with the malaria eradication campaign would negatively bias the results towards finding a spurious relationship between malaria and fertility and survival. However, there were no such education programs in the areas in which malaria was the highest.[43] Public health availability in the endemic region was superior to that in the 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."[44] 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.[45] 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.[46] 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.[47] 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.[48] There is inconsistent evidence about the effect of DDT on infant and child mortality. Studies on animals have found a negative correlation between Organochlorine 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. At the most the DDT effect

[43] Ekanayake (1982).
[44] Gray (1974).
[45] Langford (1996).
[46] Various studies as summarized in Gray (1974).
[47] Brown (1970). Pearse (1980).
[48] Pearse (1980).

biases the fertility and child survival lower and should not differentially effect parities. Therefore, explanations of differential improvements in the provision of public health and nutrition causing the malaria reduction are unfounded.

7 Discussion and Conclusions

The multigenerational approach used here draws attention to the folly of basing long run conclusions on immediate outcomes. Fertility among those of child bearing age increased as the malaria eradication occurred whether measured by the probability of birth or total fertility. This does not appear to be driven by a significant change in the probability in child survival. Instead, a driving mechanism is the more rapid progression to initial parity, indicating a biological response to a previously binding constraint on the ability to have a live birth. These effects are only transitory as the subsequent generation attains more human capital than those born before eradication in the regions with the previously highest level of malaria, despite potential congestion effects of being a member of a larger cohort. These cohorts also had lower fertility than those who were exposed to a higher malaria incidence rate in their infancy.

While transitory, this fertility increase can cause a reduction in GDP per capita as the size of the non-productive segment of the population increases. This effect could last an entire generation until the more highly educated members of the larger cohorts enter into the labor force. The relative magnitudes of these offsetting effects on GDP per capita will be a avenue for further study. The duration of the transitory population increase also provides a partial reconciliation between the two contradictory views in the growth literature of the relative importance of health in 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 sample school 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 is 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).[49] 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.[50]

B Appendix - Identification Strategy

Formally, ignoring covariates (or assuming that they have already been conditioned out), an individual level outcome is determined by the level of malaria and regional and cohort fixed effects. For simplicity, assume that this relationship is linear such that

$$Y_{ijc} = \beta M_{jc} + \delta_j + \delta_c + U_{ijc} \tag{9}$$

where Y_{ijc} is the outcome of interest for individual i born in region j as a member of cohort c, δ_j are regional fixed effects, and δ_c are cohort fixed effects. M_{jc} is the malaria level of each region-cohort. The error term, U_{ijc}, is a composite of both a region-cohort component (η_{jc}) and individual random shocks (ε_{ijc}):

$$U_{ijc} = \eta_{jc} + \varepsilon_{ijc}. \tag{10}$$

[49] 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.

[50] Newman (1965).

Therefore, the OLS estimator for the average effect of malaria, after controlling for region and cohort fixed effects, is

$$\text{plim } \widehat{\beta}^{FE} = \beta + \frac{cov(\eta_{jc}, M_{jc})}{var(M_{jc})} \tag{11}$$

Because the malaria rates are observed at the region-cohort level, using region-cohort fixed effects to control for this error structure is not feasible. Therefore, if actual malaria rates are correlated with region-cohort unobservables, the OLS estimate of β is biased and inconsistent.

Consider a country in which there were two regions, one with highly endemic malaria and one without any malaria, with an instantaneous universal eradication campaign at time T. Therefore, all cohorts with $c \geq T$ received treatment and those born in cohorts $c < T$ were too old to receive treatment. Let Z_c be a binary variable that is equal to 1 for those affected by the treatment and equal to 0 for those unaffected, that is

$$Z_c = \left\{ \begin{array}{l} 1 \text{ if } c < T \\ 0 \text{ otherwise} \end{array} \right\}. \tag{12}$$

Let M_{j0} be the malaria level in region j before treatment. In this simple example, therefore,

$$M_{j0} = \left\{ \begin{array}{l} 1 \text{ if } j \ = \text{highly endemic region} \\ 0 \text{ if } j = \text{ malaria free region} \end{array} \right\}. \tag{13}$$

Combing definitions (12) and (13), therefore,

$$\begin{aligned} M_{jc} &= M_{j0} * Z_c \\ &= \left\{ \begin{array}{l} 1 \text{ if } j \ = \text{highly endemic region and } c < T \\ 0 \text{ otherwise} \end{array} \right\}. \end{aligned}$$

Assuming that there are two cohorts, $c' < T$ and $c'' > T$ such that

1. There is a monotonic relationship between treatment assigned and treatment received:

$$M_{jc'} - M_{jc''} > 0 \text{ for any such } c' \text{ and } c''.$$

2. The exclusion restriction of treatment assignment at this margin holds:

$$E\left(\eta_{jc} | M_{j0}, c = c'\right) - E\left(\eta_{jc} | M_{j0}, c = c''\right) = 0.$$

Then

$$\text{plim}\widehat{\beta}^{FE} = \beta$$

making an OLS estimate of β both unbiased and consistent. Therefore even if the presence of malaria in a region in the initial period is endogenous to its level of (under)development, the exogenous implementation of the program across all regions simultaneously creates a non-linearity in the malaria rate that allows identification. As observed in Paraguay, Sri Lanka, and Trinidad, initial malaria levels were highly correlated with climatic and geographic features of each country.

The assumptions necessary for identification are therefore, (1) heterogeneous indigenous rates of malaria infection, (2) a well defined treatment period, and (3) exogenous implementation of the program. Each of the conditions are addressed fully in Section 4.

References

Acemoglu, D., and S. Johnson (2005): "Disease and Development: The Effect of Life Expectancy on Economic Growth," MIT Mimeo.

Barker, D. J. P. (1994): *Mothers, Babies, and Disease in Later Life*. BMJ Publishing Group, London.

Barro, R., and G. Becker (1989): "Fertility Choice in a Model of Economic Growth," *Econometrica*, 57(2), 481–501.

Behrman, J. R., and M. R. Rosenzweig (2004): "Returns to Birthweight," *The Review of Economics and Statistics*, 86, 585–601.

Black, S. E., P. J. Devereux, and K. G. Salvanes (2005): "From the Cradle to the Grave? The Effect of Brith Weight on Adult Outcomes of Children," UCLA Mimeo.

Bleakley, H. (2004): "Disease and Development: Evidence from Hookworm Eradication in the American South," UCSD mimeo.

Brooker, S., H. Guyatt, J. Omumbo, R. Shretta, L. Drake, and J. Ouma (2000): "Situation Analysis of Malaria in School-aged Children in Kenya - What Can Be Done?," *Parasitology Today*, 16(5), 183–6.

Brown, L. R. (1970): *Seeds of Change: The Green Revolution and Development in the 1970's*. Praeger Publishers, New York.

Chairleoni, G. (1886): "Malaria e atti funionali della generazoine nella donna," *Annali Universali di Medicina e Chirurgia*, 275, 249–305.

Coale, A. J., and E. M. Hoover (1958): *Population Growth and Economic Development in Low-Income Countries: A Case Study of India's Prospects*. Princeton University Press, Princeton.

Conly, G. N. (1975): "The Impact of Malaria on Economic Development: A Case Study," Scientific Publication Number 297, Pan American Health Organization Pan American Sanitary Bureau, Regional Office of the WHO.

Department of Census and Statistics (1988): *Sri Lanka Demographic and Health Survey 1987*. Institute of Resource Development/Westinghouse, Maryland.

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

Duffy, P. E., and R. S. Desowitz (2001): "Pregnancy Malaria Throughout History: Dangerous Labors," in *Malaria in Pregnancy: Deadly Parasite, Susceptible Host*, ed. by P. E. Duffy, and M. Fried. Taylor and Francis.

Duffy, P. E., and M. Fried (eds.) (2001): *Malaria in Pregnancy: Deadly Parasite, Susceptible Host*. Taylor and Francis, London.

Ekanayake, S. B. (1982): "National Case Study – Sri Lanka," in *Multiple Class Teaching and Education of Disadvantaged Groups: National Studies India, Sri Lanka, Philippines, Republic of Korea*. Unesco Regional Office for Education in Asia and Pacific, Thailand.

Gallup, J. L., and J. D. Sachs (2001): "The Economic Burden of Malaria," *Am. J. Trop. Med. Hyg.*, 64((1,2)S), 85–96.

Gill, C. (1940): "The Influence of Malaria on Natality with Special Reference to Ceylon," *Journal of the Malaria Institute of India*, 3, 201–252.

Goth, E. (1881): "Ueber den Einfluss der Malariainfection auf Schwagerschaft, Geburt and Wochenbett," *Zeitschrift fur Geburtshilfe und Gynskologie*, 6, 17–34.

Gray, R. H. (1974): "The Decline of Mortality in Ceylon and the Demographic Effects of Malaria Control," *Population Studies*, 28, 205–229.

Holding, P., and R. Snow (2001): "Impact of Plasmodium Falciparum Malaria on Performance and Learning: Review of the Evidence," *Am. J. Trop. Med. Hyg.*, 64((1,2)S), 68–75.

Kalemli-Ozcan, S. (2003): "A Stocastic Model of Mortality, Fertility, and Human Capital Investment," *Journal of Development Economics*, 70(1), 103–118.

Kitua, A., T. Smith, P. Alonso, H. Masanja, H. Urassa, C. Menendez, J. Kimario, and M. Tanner (1996): "Plasmodium falciparum Malaria in the First Year of Life in an Area of Intense and Perennial Transmission," *Tropical Medicine and International Health*, 1(4), 475–484.

Konradsen, F., F. A. Amerasinghe, W. can der Hoek, and P. H. Amerasinghe (2000): *Malaria in Sri Lanka: Current Knowledge on Transmission and Control*. International Water Management Institute, Colombo.

Lancaster, T. (2000): "The Incidental Parameter Problem Since 1948," *Journal of Econometrics*, 95, 391–413.

Langford, C. (1996): "Reasons for the Decline in Mortality in Sri Lanka Immediately After the Second World War: A Re-Examination of the Evidence," *Health Transition Review*, 6, 3–23.

Leighton, C., and R. Foster (1993): "Economic Impacts of Malaria in Kenya and Nigeria," Major Applied Research Paper Number 6, Office of Health, Health Services Division, Bureau of Reserach and Development Agency for International Development.

Lucas, A. M. (2006): "Behavioral and Biological Response to Disease Reduction: Evidence from the Malaria Eradication Campaign Sri Lanka," Brown University Mimeo.

McCormick, M. C., J. Brooks-Gunn, K. Workman-Daniels, J. Turner, and G. J. Peckman (1992): "The Helath and Development Status of Very Low-Birth-Weight Children at School Age," *Journal of the American Medical Association*, 267(16).

Mcdermott, J., J. Wirima, R. Steketee, J. Breman, and D. L. Heymann (1996): "The Effect of Placental Malaria Infection on Preinatal Mortaliry in Rural Malawi," *American Journal of Tropical Medicien and Hygiene*, 55, 61–65.

McGregor, I. (1984): "Epidemiology, Malaria, and Pregnancy," *American Journal of Tropical Medicine and Hygiene*, 33, 517–525.

McKay, H., L. Sinisterra, A. McKay, H. Gomez, and P. Loreda (1978): "Improving Cognitive Ability in Chrinically Deprived Children," *Science*, 200, 270–278.

Meegama, S. (1986): "The Mortality Transition in Sri Lanka," in *Determinants of Mortality Change and Differentials in Developing Countries*. United Nations, New York.

Miguel, E., and M. Kremer (2004): "Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities," *Econometrica*, 72(1), 159–217.

Newman, P. (1965): *Malaria Eradication and Population Growth With a Special Reference to Ceylon and British Guiana*. School of Public Health University of Michigan, Ann Arbor.

Onwujekwe, O., R. Chima, and P. Okonkwo (2000): "Economic Burden of Malaria Illness of Households Versus that of all Other Illness Episodes: A Study in Five Malaria Holo-Endemic Nigerian Communities," *Health Policy*, 54, 143–159.

Pampana, E. (1969): *A Textbook of Malaria Eradication*. Oxford University Press, London.

Pearse, A. (1980): *Seeds of Plenty, Seeds of Want: Social and Economic Implications of the Green Revolution*. Clarendon Press, Oxford.

Roll Back Malaria (2005): "Malaria in Africa," .

Rowland, M., T. Cole, and R. Whitehead (1977): "A Quantitative Study into the Role of Infection in Determining Nutritional Status in Gambian Village Children," *British Journal of Nutrition*, 37, 441–450.

Serouri, A. A., S. Grantham-McGregor, B. Greenwood, and A. Costello (2000): "Impact of Asymptomatic Malaria Parasitaemia on Cognitive Function and School Achievement of Schoolchildren in the Yemen Republic," *Parasitology*, 121, 337–45.

Shepard, D., M. Ettling, U. Brinkmann, and R. Sauerborn (1991): "The Economic Cost of Malaria in Africa," *Trop. Med. Parasitol.*, 42(3), 199–203.

Shiff, C., W. Checkley, P. Winch, J. Minijas, and P. Lubega (1996): "Changes in Weight Gain and Anaemia Attributable to Malaria in Tanzanian Children Living under Holoendmic Conditions," *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 90, 262–265.

World Health Organization (2003): *Africa Malaria Report 2003*. World Health Organization/UNICEF, Geneva.

Map 1.1 - Malaria in Sri Lanka

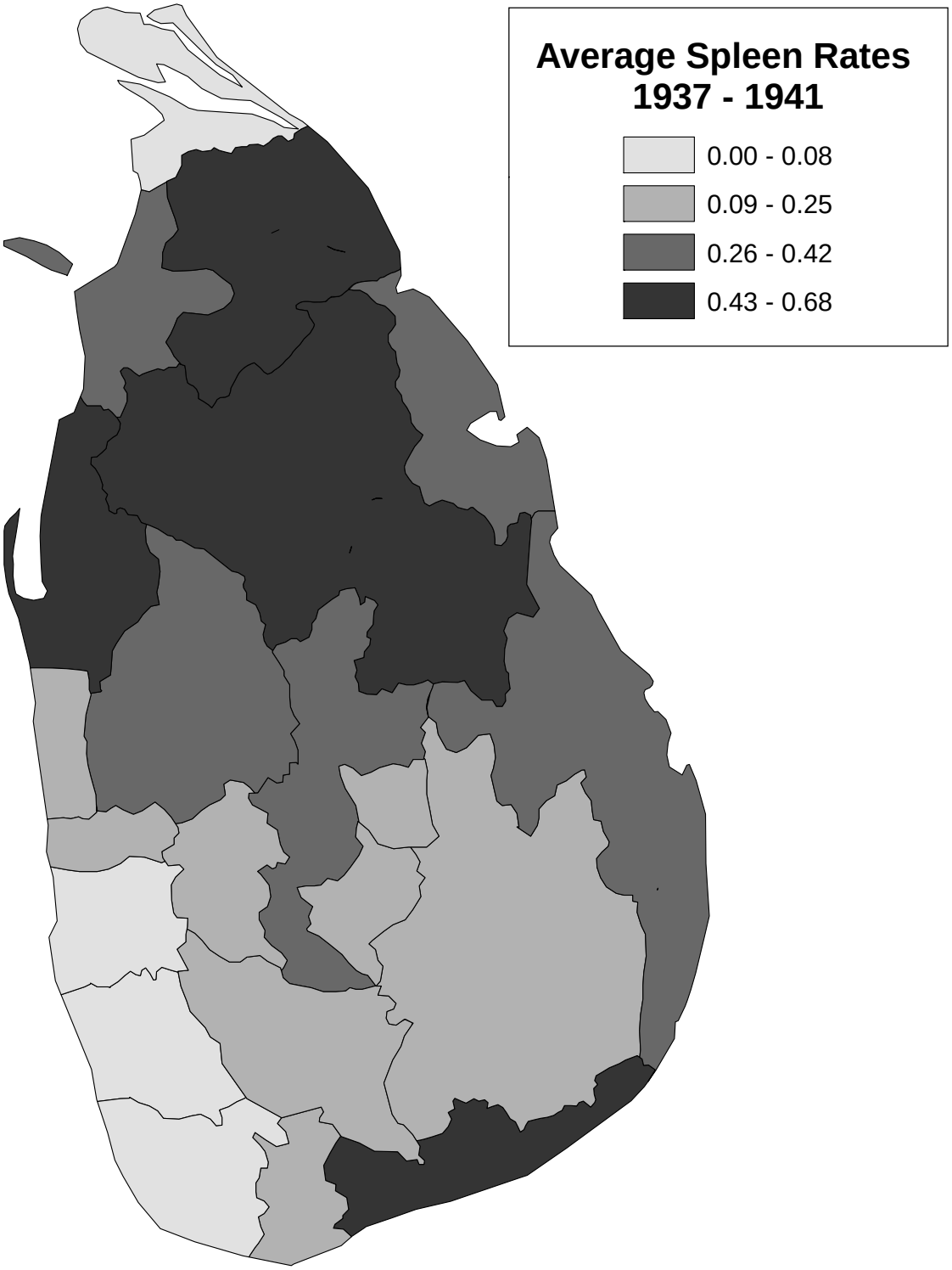

Figure 1 - Incidence and Spleen Rates

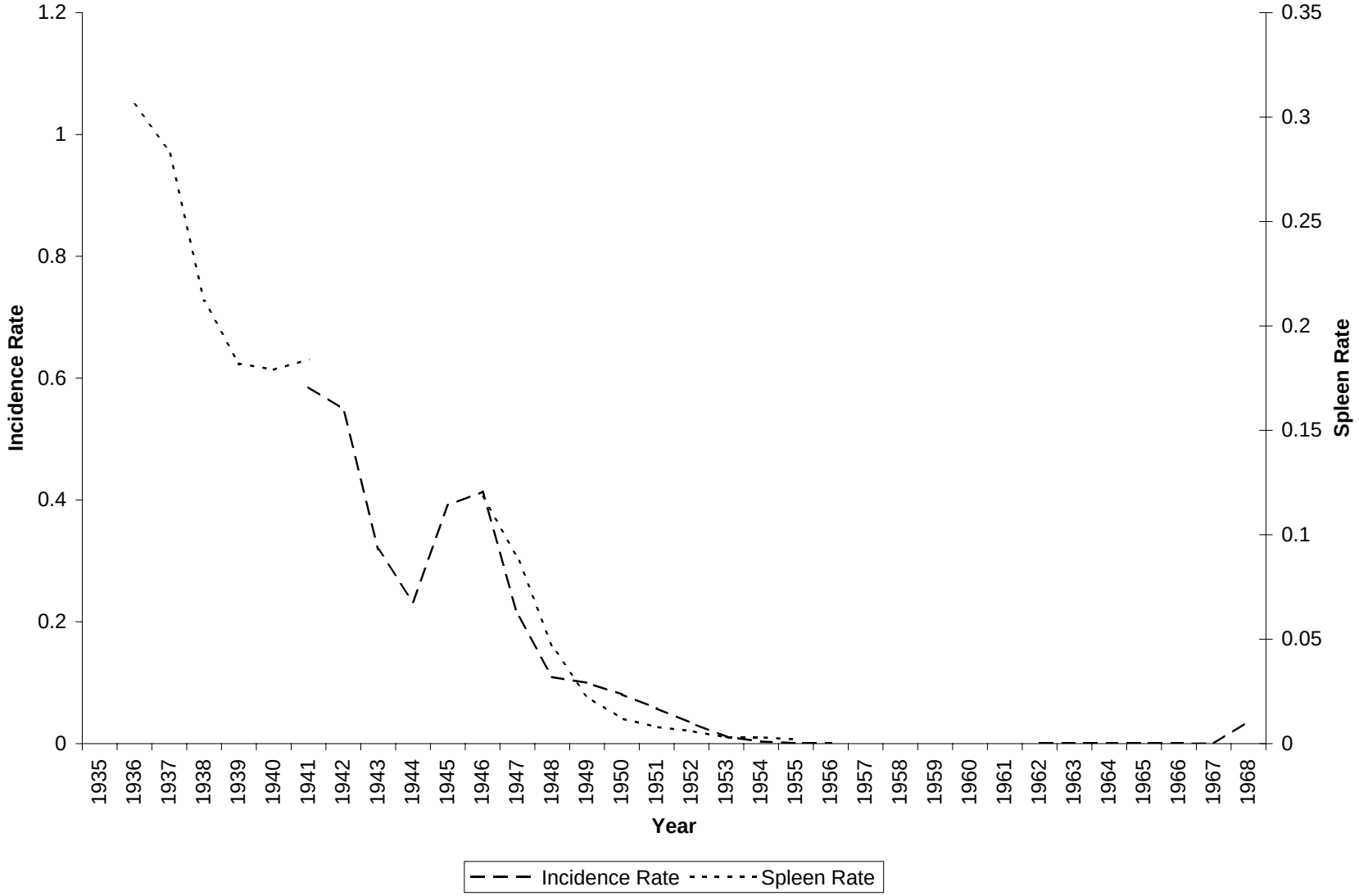

Figure 2:

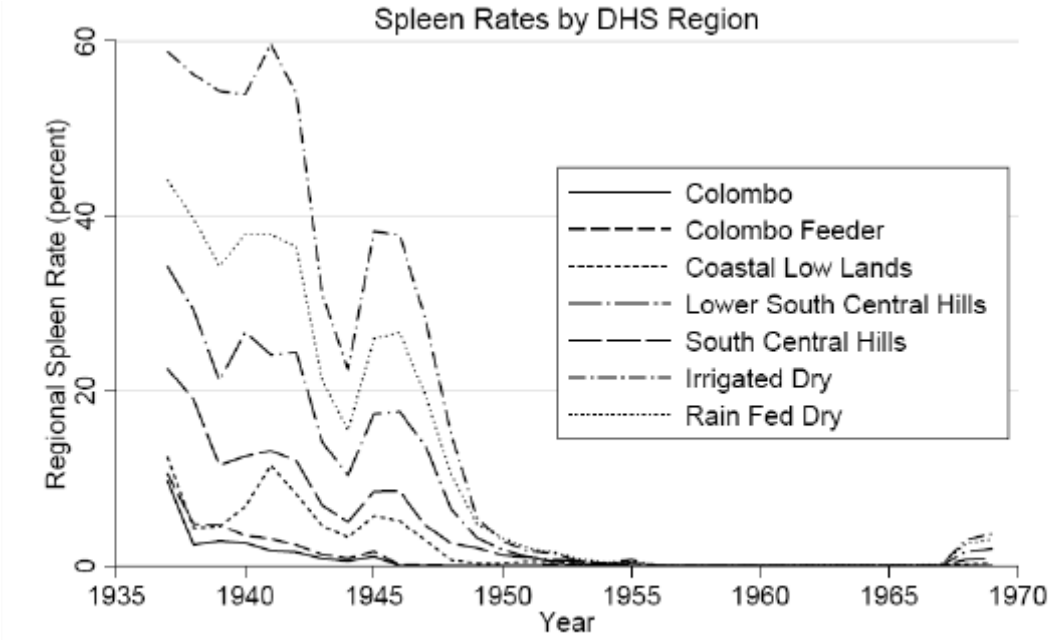
Spleen Rates by DHS Region
Regional Spleen Rate (percent)
0
20
40
60
1935
1940
1945
1950
1955
1960
1965
1970
Year
Colombo
Colombo Feeder
Coastal Low Lands
Lower South Central Hills
South Central Hills
Irrigated Dry
Rain Fed Dry

Table 1 - World Fertlity Survey Summary Statistics

	Mean
Total Number of Live Births	3.981
Total Number of Surviving Births	3.025
Probablity of Birth	
Age 13 and higher	0.178
Married	0.272
Age at First Marriage	19.168
Survival Until Age 1	
All births	0.936
First Births	0.931
Survival Until Age 5	
All births	0.903
First Births	0.897

Table 2 - Demographic and Health Surveys Sample Means

	All Respondents		Non-Movers	
Birth Year:	**1937 - 1951**	**1952 - 1968**	**1937 - 1951**	**1952 - 1968**
Years of Completed Education				
Sri Lanka	5.73	6.52	5.40	6.13
Colmobo	7.29	7.57	6.68	7.10
Colombo Feeder	7.57	7.70	7.14	7.12
South Western Coastal Low Lands	6.30	6.77	5.89	6.51
Lower South Central Hills	5.63	6.82	4.87	6.07
South Central Hills	3.84	5.09	3.68	4.67
Irrigated Dry Zone	5.11	6.86	5.15	6.50
Rain Fed Dry Zone	4.70	5.80	4.42	5.87
Ability to Read Easily				
Sri Lanka	0.706	0.706	0.673	0.715
Colmobo	0.854	0.854	0.755	0.839
Colombo Feeder	0.868	0.868	0.840	0.804
South Western Coastal Low Lands	0.793	0.793	0.756	0.792
Lower South Central Hills	0.722	0.722	0.652	0.687
South Central Hills	0.440	0.440	0.440	0.525
Irrigated Dry Zone	0.712	0.712	0.696	0.800
Rain Fed Dry Zone	0.632	0.632	0.600	0.696
Ability to Read Easily or with Difficulty				
Sri Lanka	0.809	0.843	0.781	0.816
Colmobo	0.924	0.917	0.858	0.902
Colombo Feeder	0.938	0.938	0.927	0.899
South Western Coastal Low Lands	0.891	0.873	0.840	0.877
Lower South Central Hills	0.824	0.872	0.772	0.793
South Central Hills	0.577	0.688	0.567	0.649
Irrigated Dry Zone	0.807	0.891	0.804	0.852
Rain Fed Dry Zone	0.771	0.820	0.720	0.830
Fertility by Age 30				
Sri Lanka	2.70	2.33	2.72	2.39
Colmobo	2.45	1.84	2.61	1.99
Colombo Feeder	2.05	2.19	2.03	2.43
South Western Coastal Low Lands	2.07	1.96	2.17	2.20
Lower South Central Hills	2.90	2.32	3.23	2.53
South Central Hills	2.77	2.32	2.65	2.20
Irrigated Dry Zone	3.32	2.80	3.09	2.61
Rain Fed Dry Zone	3.57	2.93	3.54	2.80
Fraction of Live Births Who Survive to Age 5				
Sri Lanka	0.950	0.956	0.950	0.951
Colmobo	0.971	0.961	0.971	0.949
Colombo Feeder	0.961	0.962	0.951	0.956
South Western Coastal Low Lands	0.956	0.956	0.960	0.947
Lower South Central Hills	0.956	0.945	0.942	0.914
South Central Hills	0.925	0.949	0.924	0.972
Irrigated Dry Zone	0.938	0.953	0.952	0.951
Rain Fed Dry Zone	0.950	0.968	0.962	0.963

Table 3 - Fertility

			Probability of Birth	
	Total Number of Live Births	**Total Number of Surviving Births**	**Maternal Age Thireteen or Higher**	**Married**
Independent Variables	(1)	(2)	(3)	(4)
Malaria Rate	-13.681 (4.327)***	-11.946 (3.306)***	-0.170 (0.030)***	-0.342 (0.095)***
Additional Covariates				
Years in Marriage	YES	YES	YES	YES
Years in Marriage Squared	YES	YES	YES	YES
Region Fixed Effects	YES	YES	YES	YES
Mom Birth Year Fixed Effects	YES	YES	YES	YES
Birth Control Knowledge	YES	YES	YES	YES
Birth Control Usage	YES	YES	YES	YES
Year of Marriage	YES	YES		
Marriage Indicator			YES	
Year Fixed Effects			YES	YES
Death in Previous Year			YES	YES
Birth in Previous Year			YES	YES
Dummies for Parity			YES	YES
Dummies for Number of Previous Child Deaths			YES	YES
Regression Statistics				
Observations	6,810	4,317	147,045	100,410
Adjusted Rsquared	0.60	0.59	0.14	0.05

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

Table 4 - Hazards

	Hazard of First Birth	
Independent Variables	**Maternal Age Thirteen or Higher** (1)	**Married** (2)
Malaria Rate	-0.055 (0.021)**	-0.223 (0.105)**
Additional Covariates		
Maternal Education	YES	YES
Years in Marriage	YES	YES
Years in Marriage Squared	YES	YES
Region Fixed Effects	YES	YES
Year Fixed Effects	YES	YES
Mom Birth Year Fixed Effects	YES	YES
Birth Control Knowledge	YES	YES
Birth Control Usage	YES	YES
Marriage Indicator	YES	
Regression Statistics		
Observations	62,957	20,011
Adjusted Rsquared	0.24	0.02

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

Table 5 - Survival

	Survival Until Age 1		Survival Until Age 5	
	All Births	**First Births**	**All Births**	**First Births**
Independent Variables	(1)	(2)	(3)	(4)
Malaria Rate	-0.232	-0.428	-0.320	-0.595
	(0.186)	(0.204)**	(0.195)	(0.227)***
Additional Covariates				
Region Fixed Effects	YES	YES	YES	YES
Birth Year Fixed Effects	YES	YES	YES	YES
Mom Birth Year Fixed Effects	YES	YES	YES	YES
Birth Order Fixed Effects	YES		YES	
Twin Indicator	YES	YES	YES	YES
Sex Indicator	YES	YES	YES	YES
Urban Indicator	YES	YES	YES	YES
Regression Statistics				
Observations	25,823	5,900	20,911	4,912
Pseudo Rsquared	0.03	0.03	0.03	0.03

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

Table 6 - Human Capital Accumulation

	Years of Completed Education		Can Read a Newspaper Easily		Can Read a Newspaper Easily or With Difficulty	
	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only
Independent Variables	(1)	(2)	(3)	(4)	(5)	(6)
Regional Spleen Rate	-3.707 (0.896)***	-3.710 (1.928)**	-0.234 (0.097)**	0.049 (0.223)	-0.159 (0.072)**	-0.124 (0.159)
Regression Statistics						
Observations	5,822	2,079	5,803	2,072	5,797	2,072
Pseudo Rsquared	0.04	0.04	0.12	0.11	0.13	0.13

Notes:
* significant at 10%; ** significant at 5%; *** significant at 1%
All columns include year of birth, region, place of childhood residence, ethnicity, and sector (urban, rural, estate) as additional regressors.
Columns (1) and (2): Maginal effects of tobit estimates are presented; absolute values of robust standard errors appear in parentheses.
Columns (3) - (6): Maginal effects of probit estimates are presented; absolute values of robust standard errors appear in parentheses.
Column (3): The sample size is reduced by 19 individuals because all individuals identified as ethnic Burghers are literate (17) as are all individuals with unknown childhood places of residence (2).
Columns (4) and (6): The sample size is reduced by 7 individuals because all individuals identified as nonmover ethnic Burghers are literate as is the one nonmover with unknown ethnicity.
Column (5): The sample size is reduced by 25 individuals because all individuals identified as ethnic Burghers are literate (17) as are all individuals with unknown childhood places of residence (2) and those born in 1969 (6).

Table 7 - Second Generation Ferility and Third Generation Survival

	Total Fertility by Age 30		Fraction of Live Births Who Survive to Age 5	
Independent Variables	**All Respondents** (1)	**Non-Movers Only** (2)	**All Respondents** (3)	**Non-Movers Only** (4)
Regional Spleen Rate	2.265 (0.563)***	3.487 (1.107)***	-0.030 (0.029)	0.023 (0.059)
Additional Control Variables				
Place of Childhood Residence Indicators	YES	YES	YES	YES
Ethnicity Indicators	YES	YES	YES	YES
Regional Indicators	YES	YES	YES	YES
Year of Birth Indicators	YES	YES	YES	YES
Regression Statistics				
Observations	4,053	1,372	4,185	1,471
Adjusted Rsquared	0.11	0.12	0.01	0.02

Notes:
* significant at 10%; ** significant at 5%; *** significant at 1%
Maginal effects of tobit estimates are presented; absolute values of robust standard errors appear in parentheses.

Table 8 - Fertility - Non-Linear Estimation

	Total Number of Live Births	Total Number of Surviving Births	Probability of Birth	
			Maternal Age Thireteen or Higher	Married
Independent Variables	(1)	(2)	(3)	(4)
Malaria Rate	-6.877 (4.093)*	-12.016 (2.931)***	-0.076 (0.029)***	-0.299 (0.095)***
Additional Covariates				
Years in Marriage	YES	YES	YES	YES
Years in Marriage Squared	YES	YES	YES	YES
Region Fixed Effects	YES	YES	YES	YES
Mom Birth Year Fixed Effects	YES	YES	YES	YES
Birth Control Knowledge	YES	YES	YES	YES
Birth Control Useage	YES	YES	YES	YES
Year of Start of Marriage	YES	YES		
Year Fixed Effects			YES	YES
Marriage Indicator			YES	YES
Death in Previous Year			YES	YES
Birth in Previous Year			YES	YES
Dummies for Parity			YES	YES
Dummies for Number of Previous Child Deaths			YES	YES
Regression Statistics				
Observations	6,810	4,317	146,955	100,359
Pseudo Rsquared	0.18	0.19		

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

Table 9 - Hazards - Non-Linear Estimation

	Hazard of First Birth	
Independent Variables	**Maternal Age Thirteen or Higher** (1)	**Married** (2)
Malaria Rate	-0.007 (0.004)	-0.253 (0.122)**
Additional Covariates		
Years in Marriage	YES	YES
Years in Marriage Squared	YES	YES
Region Fixed Effects	YES	YES
Year Fixed Effects	YES	YES
Mom Birth Year Fixed Effects	YES	YES
Birth Control Knowledge	YES	YES
Birth Control Useage	YES	YES
Marriage Indicator	YES	
Death in Previous Year		
Birth in Previous Year		
Dummies for Parity		
Dummies for Number of Previous Child Deaths		
Regression Statistics		
Observations	62,918	20,008
Adjusted or Pseudo Rsquared		

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

Table 10 - Survival - Non-Linear Estimation

	Survival Until Age 1		Survival Until Age 5	
	All Births	**First Births**	**All Births**	**First Births**
Independent Variables	(1)	(2)	(3)	(4)
Malaria Rate	-0.121 (0.103)	-0.221 (0.104)**	-0.179 (0.124)	-0.337 (0.138)**
Additional Covariates				
Region Fixed Effects	YES	YES	YES	YES
Birth Year Fixed Effects	YES	YES	YES	YES
Mom Birth Year Fixed Effects	YES	YES	YES	YES
Birth Order Fixed Effects	YES		YES	
Twin Indicator	YES	YES	YES	YES
Sex Indicator	YES	YES	YES	YES
Urban Indicator	YES	YES	YES	YES
Regression Statistics				
Observations	25,811	5,894	20,903	4,909
Pseudo Rsquared	0.05	0.07	0.04	0.06

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

Table 11 - Second Generation Ferility and Third Generations Survival - Non Linear Estima

	Total Fertility by Age 30		Fraction of Live Births Who Survive to Age 5	
Independent Variables	**All Respondents** (1)	**Non-Movers Only** (2)	**All Respondents** (3)	**Non-Movers Only** (4)
Regional Spleen Rate	2.227 (0.525)***	3.218 (1.035)***	-0.060 (0.027)**	0.015 (0.033)
Additional Control Variables				
Place of Childhood Residence Indicators	YES	YES	YES	YES
Ethnicity Indicators	YES	YES	YES	YES
Regional Indicators	YES	YES	YES	YES
Year of Birth Indicators	YES	YES	YES	YES
Regression Statistics				
Observations	4,053	1,372	4,185	1,471
Pseudo Rsquared	0.11	0.12	0.04	0.07

Notes:
* significant at 10%; ** significant at 5%; *** significant at 1%
Maginal effects of tobit estimates are presented; absolute values of robust standard errors appear in parentheses.

Table 12 - Fertility Robustness - Regional Trends

			Probability of Birth	
	Total Number of Live Births	**Total Number of Surviving Births**	**Maternal Age Thireteen or Higher**	**Married**
Independent Variables	(1)	(2)	(3)	(4)
Malaria Rate	-12.422 (4.304)***	-15.329 (3.494)***	-0.071 (0.037)*	-0.242 (0.099)**
Additional Covariates				
Years in Marriage	YES	YES	YES	YES
Years in Marriage Squared	YES	YES	YES	YES
Region Fixed Effects	YES	YES	YES	YES
Mom Birth Year Fixed Effects	YES	YES	YES	YES
Birth Control Knowledge	YES	YES	YES	YES
Birth Control Useage	YES	YES	YES	YES
Year of Start of Marriage	YES	YES		
Death in Previous Year			YES	YES
Birth in Previous Year			YES	YES
Dummies for Parity			YES	YES
Dummies for Number of Previous Child Deaths			YES	YES
Year Fixed Effects			YES	YES
Marriage Indicator			YES	YES
Region Fixed Effect x Time Trend	YES	YES	YES	YES
Regression Statistics				
Observations	6,810	4,317	146,955	100,359
Adjusted or Pseudo Rsquared	0.58	0.55	0.21	0.09

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

Table 13 - Hazard Robustness - Regional Trends

	Hazard of First Birth	
Independent Variables	**Maternal Age Thirteen or Higher** (1)	**Married** (2)
Malaria Rate	-0.072 (0.030)**	-0.212 (0.123)*
Additional Covariates		
Years in Marriage	YES	YES
Years in Marriage Squared	YES	YES
Region Fixed Effects	YES	YES
Year Fixed Effects	YES	YES
Mom Birth Year Fixed Effects	YES	YES
Birth Control Knowledge	YES	YES
Birth Control Useage	YES	YES
Marriage Indicator	YES	
Death in Previous Year		
Birth in Previous Year		
Dummies for Number of Previous Child Deaths		
Region Fixed Effect x Time Trend	YES	YES
Regression Statistics		
Observations	62,957	20,011
Adjusted or Pseudo Rsquared	0.24	0.02

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

Table 14 - Survival Robustness - Regional Trends

	Survival Until Age 1		Survival Until Age 5	
	All Births	**First Births**	**All Births**	**First Births**
Independent Variables	(1)	(2)	(3)	(4)
Malaria Rate	-0.192 (0.187)	-0.481 (0.217)**	-0.255 (0.202)	-0.625 (0.256)**
Additional Covariates				
Region Fixed Effects	YES	YES	YES	YES
Birth Year Fixed Effects	YES	YES	YES	YES
Mom Birth Year Fixed Effects	YES	YES	YES	YES
Birth Order Fixed Effects	YES		YES	
Twin Indicator	YES	YES	YES	YES
Sex Indicator	YES	YES	YES	YES
Urban Indicator	YES	YES	YES	YES
Region Fixed Effect x Time Trend	YES	YES	YES	YES
Regression Statistics				
Observations	25,823	5,900	20,911	4,912
Pseudo Rsquared	0.03	0.03	0.03	0.03

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

Table 15 - Education Robustness - Regional Trends

	National Year of Birth Indicators						National Time Trend					
	Years of Completed Education		Can Read a Newspaper Easily		Can Read a Newspaper Easily or With Difficulty		Years of Completed Education		Can Read a Newspaper Easily		Can Read a Newspaper Easily or With Difficulty	
	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only
Independent Variables	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Regional Spleen Rate	-1.073	-0.295	-0.173	0.064	-0.106	-0.041	-6.242	-6.930	-0.621	-0.815	-0.475	-0.636
	(1.593)	(2.972)	(0.185)	(0.36)	(0.149)	(0.294)	(1.130)***	(2.178)***	(0.114)***	(0.236)***	(0.093)***	(0.186)***
Additional Control Variables												
Year of Birth Indicators	YES	YES	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO
National Time Trend	NO	NO	NO	NO	NO	NO	YES	YES	YES	YES	YES	YES
Regression Statistics												
Observations	5,822	2,079	5,803	2,072	5,797	2,072	5,822	2,079	5,803	2,075	5,803	2,075
Pseudo Rsquared	0.04	0.04	0.13	0.11	0.14	0.13	0.04	0.04	0.12	0.10	0.13	0.11

Notes:
* significant at 10%; ** significant at 5%; *** significant at 1%
All columns include year of birth, region, place of childhood residence, ethnicity, sector (urban, rural, estate), and six regional time trends as additional regressors.
Columns (1), (2), (7), and (8): Maginal effects of tobit estimates are presented; absolute values of robust standard errors appear in parentheses.
Columns (3) - (6) and (9) - (12): Maginal effects of probit estimates are presented; absolute values of robust standard errors appear in parentheses.
Columns (3) and (9): The sample size is reduced by 19 individuals because all individuals identified as ethnic Burghers are literate (17) as are all individuals with unknown childhood places of residence
Columns (4) and (6): The sample size is reduced by 7 individuals because all individuals identified as nonmover ethnic Burghers are literate as is the one nonmover with unknown ethnicity.
Column (5): The sample size is reduced by 25 individuals because all individuals identified as ethnic Burghers are literate (17) as are all individuals with unknown childhood places of residence (2) and those born in 1969 (6).
Column (10): The sample size is reduced by 4 individuals because all individuals identified as ethnic Burghers are literate (3) as are all individuals with unknown ethnicity (1).

Table 15b - Education Robustness - Regional Trends (cont'd)

	Pre-Eradication and Post-Eradication Indicators					
	Years of Completed Education		Can Read a Newspaper Easily		Can Read a Newspaper Easily or With Difficulty	
	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only	All Respondents	Non-Movers Only
Independent Variables	(1)	(2)	(3)	(4)	(5)	(6)
Pre-Eradication x spleen rate 1936	-1.550 (0.817)**	-1.104 (1.358)	-0.127 (0.097)	-0.169 (0.182)	-0.167 (0.085)*	-0.025 (0.149)
Additional Control Variables						
Year of Birth Indicators	YES	YES	YES	YES	YES	YES
National Time Trend	NO	NO	NO	NO	NO	NO
Regression Statistics						
Observations	5,822	2,079	5,803	2,072	5,797	2,072
Pseudo Rsquared	0.04	0.04	0.13	0.11	0.14	0.13

Notes:

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

All columns include year of birth, region, place of childhood residence, ethnicity, sector (urban, rural, estate), and six regional time trends as additional regressors.

Columns (1) and (2): Maginal effects of tobit estimates are presented; absolute values of robust standard errors appear in parentheses.

Columns (3) - (6): Maginal effects of probit estimates are presented; absolute values of robust standard errors appear in parentheses.

Table 16 - Second Generation Ferility and Third Generation Survival Robustness - Region

	Total Fertility by Age 30		Fraction of Live Births Who Survive to Age 5	
Independent Variables	**All Respondents** (1)	**Non-Movers Only** (2)	**All Respondents** (3)	**Non-Movers Only** (4)
Regional Spleen Rate	3.096 (1.330)**	3.134 (2.605)	0.063 (0.071)	0.175 (0.156)
Additional Control Variables				
Place of Childhood Residence Indicators	YES	YES	YES	YES
Ethnicity Indicators	YES	YES	YES	YES
Regional Indicators	YES	YES	YES	YES
Year of Birth Indicators	YES	YES	YES	YES
Regression Statistics				
Observations	4,053	1,372	4,185	1,471
Adjusted Rsquared	0.12	0.12	0.02	0.03

Notes:
* significant at 10%; ** significant at 5%; *** significant at 1%
Maginal effects of tobit estimates are presented; absolute values of robust standard errors appear in parentheses.

Table 17 - Fertility - Maternal Fixed Effects

	Probability of Birth	
Independent Variables	**Maternal Age Thireteen or Higher** (1)	**Married** (2)
Malaria Rate	-0.159 (0.053)***	-0.293 (0.108)***
Additional Covariates		
Years in Marriage	YES	YES
Years in Marriage Squared	YES	YES
Death in Previous Year	YES	YES
Birth in Previous Year	YES	YES
Dummies for Parity	YES	YES
Dummies for Number of Previous Child Deaths	YES	YES
Year Fixed Effects	YES	YES
Marriage Indicator	YES	
Maternal Fixed Effects	YES	YES
Regression Statistics		
Observations	147,031	100,397
Number of Respondents	6,809	6,810
Adjusted Rsquared	0.01	0.04

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

Table 18 - Hazards - Maternal Fixed Effects

	Hazard of First Birth	
Independent Variables	**Maternal Age Thirteen or Higher** (1)	**Married** (2)
Malaria Rate	-0.381 (0.097)***	-1.276 (0.903)
Additional Covariates		
Years in Marriage	YES	YES
Years in Marriage Squared	YES	YES
Region Fixed Effects	YES	YES
Year Fixed Effects	YES	YES
Death in Previous Year		
Birth in Previous Year		
Dummies for Number of Previous Child Deaths		
Maternal Fixed Effects	YES	YES
Regression Statistics		
Observations	62,957	20,011
Number of Respondents	6,748	6,753
Adjusted Rsquared	0.16	0.10

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

Table 19 - Survival - Maternal Fixed Effects

Independent Variables	Survival Until Age 1 (1)	Survival Until Age 5 (2)
Malaria Rate	-0.207	-0.283
	(0.196)	(0.210)
Additional Covariates		
Birth Year Fixed Effects	YES	YES
Birth Order Fixed Effects	YES	YES
Twin Indicator	YES	YES
Sex Indicator	YES	YES
Maternal Fixed Effects	YES	YES
Regression Statistics		
Observations	25,823	20,911
Number of Respondents	5,900	4,912
Adjusted Rsquared	0.02	0.02

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

Table 20 - Robustness Checks - Aggregated Sample

Independent Variables	Years of Completed Education (1)	Can Read a Newspaper Easily (2)	Can Read a Newspaper Easily or With Difficulty (3)
Regional Spleen Rate	-4.193 (0.636)***	-0.348 (0.090)***	-0.265 (0.081)***
Regression Statistics			
Observations	231	231	2970
Pseudo Rsquared	0.42	0.53	0.63

Notes:
* significant at 10%; ** significant at 5%; *** significant at 1%
Columns (1) - (3) inlcude regional, year of birth, and childhood place of residence dummie
Columns (4) - (6) include year of birth, region, place of childhood residence, ethnicity, and
Columns (7) - (9) include year of birth, ethnicity, St. George, and religion dummy
Columns (1), (4), and (7): Maginal effects of tobit estimates are presented; absolute
Columns (2) - (3), (5) - (6), and (8) - (9): Maginal effects of probit estimates are