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Mosquitoes: The Long-term Effects of Malaria Eradication in India
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ABSTRACT

We examine the effects of malaria on educational attainment and income by exploiting geographic variation in malaria prevalence in India prior to a nationwide eradication program in the 1950s. We find that the program led to modest increases in income for prime age men. This finding is robust to using very localized sources of geographic variation and to instrumenting for pre-eradication prevalence with climate factors. We do not observe improvements in income for women, suggesting that observed effects are likely driven by increased labor market productivity. We find no evidence of increased educational attainment for men, and mixed evidence for women.

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1 Introduction

Malaria has afflicted humans for more than 10,000 years (Dunn 2003). Today, malaria is endemic in over 100 countries and affects 40% of the world's population. The World Health Organization (2001) estimates that 300 million people suffer from malaria in the world each year, with almost one million deaths from malaria. Faced with this huge global burden, international organizations have redoubled their efforts to combat the disease. The United Nations has made combating malaria one of its Millennium Development Goals. The Roll Back Malaria Global Partnership, formed by the WHO, United Nations Children's Fund, United Nations Development Program, and the World Bank, aims to halve the malaria burden relative to 2000 levels by 2010.

Many argue that improving health, while important in itself, can also lead to higher economic growth and development. Gallup and Sachs (2001) use cross-country growth regressions to show that countries with intensive malaria grew 1.3% less per year and that a 10% reduction in malaria was correlated with 0.3% higher growth.[1] In contrast, Acemoglu and Johnson (2007) argue that the wave of international health innovations that began in the 1940s did not lead to a disproportionate increase in log per capita GDP in the areas with high pre-intervention disease burden.

In this paper, we examine some of the channels through which health may have a causal impact on development by studying the effects of childhood exposure to malaria eradication on human capital accumulation and income in adulthood. We use the national malaria eradication program in India in the 1950s as a quasi-experiment and exploit geographic variation in malaria prevalence prior to the eradication campaign. We compare gains for cohorts born before and after the program in areas with varying pre-eradication prevalence. These differences-in-differences estimates show no gains in literacy or primary school completion for areas that experienced large reductions in malaria to those that experienced small reductions.[2] We do, however, observe modest relative increases in income (proxied by per capita household expenditure) for prime age men. This effect is robust to using quite localized sources of geographic variation and to instrumenting for pre-eradication prevalence using geographic and climate factors.[3] We do not observe increases in expenditure as a result of the program for women, who have much lower labor force participation

[1] Other macroeconomic studies, such as those by Bloom and Canning (2005) and Alleyne and Cohen (2002), also conclude that improvements in health can lead to higher economic growth.

[2] These results differ from an earlier working paper draft. This version corrects errors discovered in the original program files and extends the empirical analysis along a number of dimensions.

rates than men, suggesting that the primary channel underlying the observed effects for men may be improved labor market productivity.

Our findings relate most closely to two recent papers that examine the effect of malaria eradication campaigns in other parts of the world. Bleakley (2007) studies the effect of malaria eradication campaigns on the income and education of native males in the United States, Brazil, Colombia, and Mexico. Using malaria mortality rates and an ecology index to identify pre-eradication disease prevalence, Bleakley finds that childhood exposure to malaria lowers labor productivity and leads to lower adult income. Results for years of schooling are mixed. Lucas (2009) studies ever-married women in Paraguay and Sri Lanka in the 1940s to 1960s and finds that malaria eradication leads to increases in female education and literacy rates.[4] Two other studies use weather conditions to instrument for malaria exposure in the United States and examine the effects on long run health and economic outcomes. Hong (2007) finds that malarial risk leads to adverse long run health outcomes, lower labor force participation, and lower wealth. Barecca (2009) finds that in utero and postnatal malaria exposure leads to lower educational attainment. We discuss our findings relative to this literature in Section 5.

The paper proceeds as follows. Section 2 provides an overview of the epidemiology of malaria and the mechanisms through which malaria may affect educational attainment, describes malaria in India in the pre-eradication era, and discusses the National Malaria Control Program in India and its effectiveness. Section 3 outlines our empirical strategy. Section 4 describes the data. Section 5 presents our results, and Section 6 concludes.

2 Malaria in India

> "The problem of existence in very many parts of India is the problem of malaria. There is no aspect of life in this country which is not affected either directly or indirectly by this disease. It constitutes one of the most important causes of economic misfortune, engendering poverty, diminishing quantity and quality of food supply, lowering the physical and intellectual standards of the nation and hampering increased prosperity in every way."
>
> - *John Sinton, Director of the Malaria Survey of India, 1936*

[3] The IV results do suggest improvements in educational attainment resulting from the program for women; we discuss these results in detail below.

[4] Lucas (2007) finds that malaria eradication in Sri Lanka led to an initial increase in fertility followed by lowered fertility in the second generation.

2.1 Overview of Malaria Epidemiology

Malaria is a protozoal infection transmitted to human beings by mosquitoes. The classic symptom of malaria is bouts of fever with spikes on alternating days. Headaches, malaise, fatigue, nausea, and anemia are also common. Severe forms of the disease can result in organ failure, delirium, impaired consciousness, and generalized convulsions, followed by persistent coma and death.[5] Infants and children suffer the worst from malaria and high morbidity and mortality rates. The other high-risk group is pregnant women, for whom malaria often results in pre-term labor or low birth weight full-term births, as well as spontaneous abortions and still-births.

Malaria has been hypothesized to have lifelong effects on skill acquisition through at least three channels: effects on cognitive activity, school absenteeism, and fetal development (Sachs and Malaney, 2002). Epidemiological studies have associated malaria with anemia, epileptic convulsions, and growth faltering during the first three years of life (Shiff et al., 1996), potentially leading to learning disabilities and negative effects on cognitive development (Boyle et al., 1994). Malaria in pregnancy can cause low birth weight because of fetal growth retardation or premature delivery (Duffy and Desowitz, 2001). This can in turn reduce the physical, cognitive, and neurosensory development of the child, resulting in lower human capital accumulation (McCormick et al., 1992). Randomized evaluations have documented effects of malaria on school absenteeism,[6] but no randomized evaluation has so far, to the best of our knowledge, examined the effect of malaria prevention or treatment programs on longer run outcomes.

Importantly, malaria's various sequelae do not necessarily imply that its eradication will lead to improvements in schooling outcomes in the population. Eradication may lead to the survival of children with poorer health and weaker cognitive skills. This is unlikely to be the case here because, as described below, the most prevalent form of malaria in India is generally non-fatal. Second, as emphasized by Bleakley (2007), in a country with widespread child labor, the effect of improved childhood health on the labor-schooling decision is ambiguous because malaria could affect children's productivity not just in education but also in work. Similarly, the cognitive gains from eradication can lead to increased or decreased schooling investment depending on the balance of income and substitution effects. The conventional wisdom is that cognitive ability is complementary to schooling (Card 2001), but this is an empirical claim, not a theoretical prediction.

[5] WHO, http://www.who.int/malaria/faq.html.
[6] See, for example, Leighton and Foster (1993), Aikins (1995), and Brooker, et al. (2000).

In addition, malaria may have effects on other outcomes, even for a given level of schooling. Fewer school absences could lead to greater learning, leading to improvements in literacy and earnings holding the years of schooling constant (Bleakley, 2007). The health benefits of malaria reduction could also result in improved physical and mental condition later in life and therefore higher labor market productivity.

2.2 The Pre-Eradication Era

References to malaria can be found in Vedic writings dating to 1600 B.C. (Desowitz, 1991), and two classical books on Ayurveda describe malaria as the "king of diseases" (Rao, 1959). Efforts to control malaria date back to the early 1900s but were revolutionized in the mid-1940s with the advent of DDT (dichlorodiphenyl trichloroethylene).[7] DDT was effective, non-toxic to humans, and "dirt-cheap to manufacture" (Desowitz, 1991). Aggressive campaigns using DDT were launched almost simultaneously around the world, leading to the rapid eradication of malaria in Taiwan, much of the Caribbean, the Balkans, parts of northern Africa, northern Australia, and large parts of the South Pacific (Davis, 1956).

DDT was first used in India by the military in 1944 and became available for civilian anti-malaria operations in 1945. During the late 1940s, a number of pilot programs and trials took place throughout the country. International organizations such as WHO, UNICEF, and the Rockefeller Foundation also sponsored demonstration projects. These pilot projects were very successful, and in 1951, the national Planning Commission endorsed the development of a comprehensive, nationwide malaria control program. In April of 1953, the National Malaria Control Program (NMCP) was launched. Funding for the program was primarily from bilateral and international sources, and program implementation was overseen by the WHO. The timing of the program is plausibly exogenous, since it was driven by the advent of DDT.

Prior to the eradication program, malaria was considered the greatest health problem facing India. Survey evidence estimates that immediately after partition in 1947, India suffered from 75 million cases of malaria (doubled during epidemic years) and 800,000 deaths directly attributable to malaria annually (Sinton, 1935, 1936; Rao, 1959). The population of India in 1947 was 344 million,

[7] Early experiments prior to 1910 focused on breeding control. These attempts were generally considered failures. From 1910 to 1944, various actors undertook measures such as drainage and the filling up of breeding places. The use of larvicidal chemicals such as oil, Paris green, and later pyrethrum also achieved limited success (NMEP 1986, p. 2).

implying an annual incidence rate of 22%.

Of the four human malaria parasites (*Plasmodium falciparum, P. vivax, P. malariae,* and *P. ovale*), two are endemic in India: *P. vivax* and *P. falciparum*.[8] *P. falciparum* is associated with the most severe forms of malaria and accounts for most malaria fatalities. It is the primary cause of malarial infections in Africa, where 90% of malaria deaths currently occur. Data on the relative prevalence of these parasites in India during the pre-eradication era are unfortunately unavailable; data from the immediate post-eradication period suggest that approximately 30% of cases were due to *P. falciparum* (NMEP, 1996).

2.3 National Malaria Control Program

The National Malaria Control Program's main operational activity was to spray DDT in human dwellings and cattle sheds. Two rounds of spraying were conducted per year, usually between May and September, the peak transmission times.

A five-year plan was formed with the goal of establishing 125 Malaria Control Units by 1956, each covering a population of one million (NMEP, 1986). The program was successful in achieving this target (*Table 1*). By 1956, 134 units had been established and 112 million people were estimated to be protected. By 1958, almost 200 units had been established and 165 million people were under protection.

The program was so successful that in 1958 it was reformulated as the National Malaria Eradication Program, with the goal of completely eradicating malaria from the nation. This effort was adopted in coordination with a WHO campaign to eradicate malaria from the entire region, launched after the Eighth World Health Assembly in 1955 (WHO, 1967). By 1960-61, the entire country was brought under the program.

Figure 1 illustrates the rapid geographic expansion of coverage as districts were phased into the program. Once a district was incorporated into the program, it remained in the program in all subsequent years. The statement of the Planning Commission indicated that priority targeting of areas should be based on endemicity and food producing capacity. The timing of the phase-in for particular districts may therefore not be exogenous.

Large urban areas were relatively free from malaria prior to the eradication era (League of Nations, 1930). In fact, urban malaria was considered to be a negligible problem, so the NMCP left

malaria control efforts to local governments.[9] Prevalence of malaria in urban areas increased only later (although only to much lower levels than pre-eradication rural malaria), and the government launched an Urban Malaria Scheme in 1971 to address the growing problem of urban malaria. We therefore focus our analysis on rural areas.

2.4 Program Effectiveness

While the campaign was unsuccessful in eradicating malaria from India, it did achieve tremendous reductions in malaria prevalence. The NMEP, which began tracking malaria prevalence from 1961 using blood smear data, estimates the 1965 national malaria caseload at 100,000 per year, compared to 75 million annual cases in the pre-eradication era (*Table 2*).

Although the 1965 figures are likely to underestimate true malaria prevalence, there is no doubt that there was a dramatic reduction in malaria prevalence over this period. *Table 3* shows state level child spleen rates (the percentage of examined children aged 2-9 with an enlarged spleen) over the eradication era.[10] We observe substantial declines in this measure in all states over this period. Furthermore, vital statistics data on causes of death indicate that the number of malaria deaths between 1952 and 1963 dropped by 91.2% and 98.3% in Uttar Pradesh and West Bengal, respectively (*Figure 2*). *Figure 2* focuses on these two states because they are large states with relatively complete data, and their boundaries remained stable through the reorganization of Indian states in 1956; the malaria death rate for these states prior to the program is similar to the nationwide death rate implied in *Table 1*.

Malaria prevalence remained low throughout the 1960s but experienced a slight resurgence in the 1970s, peaking in 1976. However, even at the peak of the resurgence, the prevalence rate was only 1.1% (*Table 2*). Reported prevalence decreased again, although not to the low levels seen in the immediate post-eradication period. This may partially be a result of increased accuracy in reported caseloads over time.

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[8] *P. malariae* also exists, but is confined to tribal areas of the country (NMEP, 1986).

[9] The following quote describes the treatment of urban malaria during this time: "As per the plan of operations formulated at the time of launching of the National Malaria Eradication Programme, all the roofed structures in the rural areas received indoor residual insecticidal spray except urban areas with a population of over 40,000. In such urban areas, the indoor residual insecticidal spray was confined only to the peripheral belt to a depth of 1 to 1.5 km. Antilarval measures were recommended in towns and cities. The implementation of antilarval operations was made the responsibility of the local bodies. Due to financial constraints many local bodies failed to implement the control measures. Though malaria epidemics were recorded earlier in Bombay, Delhi, Lucknow, etc., these could be immediately contained. Hence, malaria in urban areas was not considered as a major problem" (NMEP 1996, p. 251).

3 Empirical Strategy

Our study focuses on the effects of early-life malaria exposure on subsequent human capital attainment and expenditure in adulthood. We use a differences-in-differences design, exploiting geographic variation in the prevalence of malaria prior to the eradication program.[11] We compare outcomes at a point in time for individuals in birth cohorts born before and after the eradication era in areas with varying pre-eradication malaria prevalence. Ideally, we would know each individual's district of birth, but our outcomes data report only the district of current residence. An identifying assumption of our analysis is therefore that district of residence is a good proxy for district of birth. In the 1991 Census of India, only 7.5% of rural residents reported living in districts other than their districts of birth.

We focus on the effects of malaria exposure in very early life for two reasons, one conceptual and one practical. First, as suggested in Section 2.1, malaria likely exerts its most powerful influence on cognitive development and educational attainment during infancy and childhood. Second, the outcomes data we use exhibit age heaping, preventing us from employing a dose-response model as in Bleakley (2007). Bleakley allows the effect size to vary with years of exposure to eradication in childhood, which requires precise age reporting. We take an approach that places fewer demands on the quality of the age data, using a binary treatment variable to separate pre- and post-eradication cohorts. We thus use malaria prevalence at birth as an approximation of an individual's malaria exposure during the first few years of life, when the effect of malaria is likely the strongest.

To study the effects of early-life malaria exposure, we run regressions of the following form, for individual *i* in birth cohort *c* in district *d*:

$$Outcome_{icd} = \beta(Post)_c * (Malaria)_d + \delta_d + \alpha_c + X'_{icd}\gamma_{pre} + (Post)_c * X'_{icd}\gamma_{post} + \varepsilon_{icd} \qquad (A)$$

where *Post* indicates whether the individual was born after the eradication era and *Malaria* is a measure of pre-eradication endemicity in individual *i*'s district. δ and α are district and birth cohort fixed effects. The vector X includes membership in a scheduled caste, membership in a scheduled

[10] The spleen rate is a commonly used measure of childhood malaria infection over a long period.

[11] Although districts were phased into the program over several years during the eradication era, the timing of phase-in may be related to malaria severity and other relevant factors. In addition, measures of malaria prevalence such as the child spleen rate show declines in both sprayed and unsprayed areas over this period, suggesting that even those in unsprayed areas may have benefited from the program (NMCP, 1986). Finally, the phase-in of the program was quite

tribe, and household religion. The influence of this vector, captured in γ, is permitted to vary across the pre and post periods. Our coefficient of interest is β, representing the difference-in-difference estimate of the effect of malaria eradication. We run specification (A) separately for men and women. In robustness checks, we add several other time-varying district-, state-, and region-level covariates to this specification, as well as district-specific linear trends.

To represent our results visually, we also plot cohort-specific relationships between pre-eradication malaria endemicity and our socioeconomic outcomes of interest. The cohort-specific relationships derive from regressions of the form:

$$Outcome_{icd} = \sum_{c} \beta_c * (Malaria)_d + \alpha_c + X'_{icd}\gamma + \varepsilon_{icd} \quad \text{(B)}$$

where β_c gives the cohort-specific relationship between pre-eradication endemicity and later-life outcomes. If malaria eradication affected the human capital accumulation and economic wellbeing of exposed cohorts, these effects should be visible in a break from preexisting trends in β_c. This method would also shed light on the partial effects of malaria exposure in late childhood (rather than at birth), if such effects exist. Due to age heaping on ages ending in the digits 0 and 5, we group individuals into 5-year birth cohorts for the graphical analysis, centered on years ending in the digits 2 and 7.

4 Data

4.1 Map of Pre-Eradication Endemicity

A central problem in assessing the impact of malaria is the identification of a suitable indicator for the prevalence of the disease. As Gallup and Sachs (2001) point out, the most severely affected countries often lack high-quality data on malaria prevalence or incidence. In their study, they use historical maps of the geographical distribution of malarial risk to derive an index of malaria prevalence.

In this paper, we use a 1948 government map that classifies areas of India into categories of malaria endemicity. The map was obtained from the Ministry of Health and Family Welfare, Government of India. The pre-eradication malaria map classifies areas into six endemicity categories: (1) areas above 5000 feet; non malarious, (2) known healthy plain areas; spleen rate

rapid, raising further difficulties in exploiting variation in timing of coverage.

under 10%, (3) variable endemicity associated with dry tracts; potential epidemic areas, (4) known areas liable to fulminant epidemic diluvial malaria, (5) moderate to high endemicity; fulminant epidemics unknown, and (6) hyperendemicity of jungly hill tracts and terai land. This map was based on spleen rate surveys and climate factors, although the exact mechanism by which category boundaries were constructed is not known.

Using geographic information system (GIS) software, we digitized the 1948 malaria endemicity map. *Figure 3* shows the digitized map, overlaid with district boundaries as defined by the 1991 Census. The National Sample Survey (NSS), which we use for our outcome measures, groups some of the 466 Census districts together, resulting in 431 NSS composite "districts." We follow the NSS district coding. We drop the island district of Lakshadweep, for which malaria prevalence data are unavailable, as well as fourteen further districts that lack observations in the NSS that satisfy our sample inclusion criteria (described below), leaving 416 districts in our main sample.

The digitization procedure subdivided districts into polygons of roughly equal size, so that some districts have more than one possible classification. To aggregate the polygons at the district level, we take two approaches. In the first approach, we average all polygon values (ranging from 1 to 6, as described above) within a district to generate a continuous measure of endemicity, which we call the malaria index. However, the effects of malaria eradication may be nonlinear, so our second approach uses a categorical classification of pre-eradication endemicity. To generate this classification, we first map the original six-category endemicity measure into a new three category variable, as specified in *Table 4*. This new variable classifies each polygon as non-malarious, potential epidemic, or malarious.[12] We then categorize each district by its modal polygon malaria category. 77 districts do not have unique modes. For example, some mountainous districts in northern India have equal numbers of non-malarious, high altitude polygons as malarious, low altitude polygons. To avoid classifying bimodal districts arbitrarily, we omit them from the analysis that uses this categorical classification.

The resulting measures of pre-eradication malaria endemicity are strongly correlated with the sequelae and ecological determinants of malaria. *Figure 4* plots the state-level child spleen rate

[12] In areas where malaria is endemic, individuals can acquire limited immunity over time through years of continued exposure and multiple infections. The effects of malaria are therefore most pronounced in childhood and youth, when individuals have not acquired immunity. Immunity may also be reduced during pregnancy. In areas where malaria is epidemic, individuals may have little or no acquired immunity. In these areas, malaria can affect both children and adults and can result in severe adverse health consequences.

against our map-based state-level malaria index, constructed in the same way as the district-level index. In 1953-54, just as the NMEP was starting its operations, the child spleen rate was strongly positively associated with the malaria index. By 1959-60, as the eradication program was nearing completion, states converge to very low child spleen rates, so that states with high pre-eradication levels of malaria experienced the largest reductions in malaria over the eradication era. Furthermore, Appendix *Table A1* reports regressions of the district-level malaria index on four known ecological determinants of malaria endemicity, also measured at the district level.[13] Consistent with the accumulated knowledge on malaria ecology (e.g., Sharma 2002), the malaria index is positively associated with precipitation and humidity and negatively associated with elevation. The coefficient on temperature is negative (and the coefficients of the quadratic in column 3 imply a negative relationship over relevant temperatures), but the unconditional correlation between the malaria index and temperature is strongly positive (unreported).[14]

The 416 districts in our sample are grouped into 75 regions according to the NSS definition of regions, and these regions are in turn grouped into 29 states. When we drop bimodal districts, we observe the following patterns. Of the 74 remaining regions, 51 (69%) have districts in only one category of malaria endemicity, 22 (30%) have districts in two malaria categories, and one region has districts belonging to all three malaria categories. Of the 28 remaining states, 13 (46%) have districts in only one malaria category, another 13 (46%) have districts in two malaria categories, and two (7%) have districts belonging to all three malaria categories.

4.2 Outcomes

We use data on human capital attainment and economic status from the 43rd round of the Indian National Sample Survey (NSS), conducted in 1987. The NSS is an all-India representative household consumer expenditure survey run by the Government of India starting in 1950. It includes a parallel employment and unemployment survey every five years. The NSS has four "thick" rounds that have the largest samples: namely, 1983, 1987, 1993, and 1999. We use the 43rd

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[13] The ecology data are drawn from the International Water Management Institute *World Water and Climate Atlas* (http://www.iwmi.org) and the Climatic Research Unit (http://www.cru.uea.ac.uk). The dataset consists of a 10' latitude/longitude mean monthly climatology of surface climate over global land areas, and is interpolated from station means for the period 1961 to 1990. New et al. (2002) provide a detailed description of the dataset. We use GIS to overlay the ecology data with district boundaries.

[14] Ordered probits of the modal malaria category on the same ecological variables yield similar conclusions.

round because it is the earliest thick round that contains district identifiers. Choosing an early round mitigates possible mortality bias, and using the district identifiers allows us to examine outcomes at a very local level. The NSS reports district of current residence but not district of birth.

The human capital analyses draw on literacy and primary school attainment data, whereas the economic status analyses use household-level expenditure data.[15] Past research has used earnings or occupational wage data to estimate the productivity effects of childhood malaria exposure (e.g., Bleakley, 2007), under the implicit assumption that employers pay workers their marginal product. However, only seven percent of the NSS sample has a non-zero wage, and three-quarters of workers aged 20-60 (68% of men, 78% of women) report one of two occupations (out of 463 in the classification), both agricultural. Given the unsuitability of the labor force data, we use the survey's rich data on household consumption (including goods produced in the household) to measure the effects of eradication on economic status. We construct two income measures: log household monthly per capita expenditure (measured in 1987 rupees) and poverty, using the 1987 all-India rural poverty line of 115.70 rupees per person per month. We trim the top and bottom one percent of the expenditure data to remove implausible values.

Because we are primarily interested in the productivity effects of malaria exposure in childhood, we restrict our consumption sample to adults of the ages with the highest labor force participation rates.[16] Appendix *Figure A1* shows age profiles in labor force participation for men and women separately. Men aged 20-60 are far more likely to work than men in other age groups or women of any age. We therefore focus our attention on men in this age group, whom we call prime-age men. For completeness, we report estimates for women in the same age group.[17] Schedule 1 (consumer expenditure schedule) of the NSS gives information on household consumption, and Schedule 10 (employment schedule) gives information on education.

Table 5 provides summary statistics for our sample, which omits individuals born during the eradication era (1953 to 1961). For literacy and primary school completion, we analyze individuals

[15] We have also run our analyses using higher education outcomes, including middle school, secondary school, and college attainment. The results were substantively similar to those we report here for literacy and primary schooling.
[16] In theory, households might save to smooth consumption after a member's retirement, so that older adults could still be included in the sample. Although past research has found evidence of consumption smoothing in rural India (Townsend 1994), we feel uncomfortable assuming that this smoothing is complete, so we omit older adults from our sample.
[17] Many households have multiple workers, but we expect household consumption on average to be higher in households with workers who benefited from eradication. This approach is common in settings without good individual earnings data; in a recent example, Maccini and Yang (2009) regress a household-level asset ownership index on individual-level exposure to early-life economic shocks.

between the ages of 15 and 75. In this sample, individuals born during 1912-1952 thus comprise the pre-eradication cohorts, whereas those born during 1962-1972 comprise the post-eradication cohorts. As discussed above, to analyze expenditures and poverty, we restrict the sample to adults aged 20-60. Here, the pre-eradication era spans 1927-1952 and the post-eradication era includes 1962-1967. 77,071 households, containing 111,308 men and 107,642 women, satisfy our inclusion criteria for the human capital outcomes. Women in our sample are about half as likely as men to be literate or to have finished primary school. Roughly a third of our sample is poor, and 28 percent are members of a scheduled caste or tribe, which are at the bottom of the Indian social hierarchy. The mean age is 36. Finally, although the weighted proportion of our sample in non-malarious areas is merely two percent, the unweighted proportion is five percent, or roughly 10,000 people.

5 Results

5.1 Differences-in-Differences Analysis

We next examine the effects of the eradication program using the differences-in-differences specification described in Section 3. We examine effects separately by gender, and include controls for membership in a scheduled caste, membership in a scheduled tribe, and indicators for the two largest religious categories (Hindu and Muslim). We also interact these controls with the post-eradication dummy to allow their influence to vary across cohorts from the pre-eradication and post-eradication eras.

Table 6 shows the results of our baseline specification for literacy and primary school completion, followed by several robustness checks. Panel A presents results for men, and Panel B presents results for women. Each panel shows the treatment effects using first our three category district classification (where bimodal districts are excluded) and then using our continuous endemicity index.

The results show no robust evidence of an effect of malaria eradication on human capital attainment. For example, the first column of Panel A1 shows the effects of the program on male literacy. The coefficients on post*potential epidemic and post*malarious capture the effect of being born post-eradication versus pre-eradication in a district that was formerly potentially epidemic or malarious, relative to the effect of being born in a non-malarious district. If malaria reduction increased educational attainment, we would expect these coefficients to be positive. We also report F-tests for equality of the post*potential epidemic and post*malarious coefficients. We see no

significant differences in gains for those born in potential epidemic or malarious areas relative to those born in non-malarious areas. The baseline specification implies that those born in malarious areas experienced significantly smaller gains in literacy relative to those born in potential epidemic areas; however this effect is not robust to the inclusion of state*post controls (column 2), region*post controls (column 3), or the inclusion of region*post controls with district specific linear trends (column 4). We observe a similar pattern in Panel A2, which shows the effects using our continuous malaria index measure; again, positive treatment effects of the program would imply positive coefficients. The baseline specification implies a negative treatment effect, driven by the smaller gains in malarious areas relative to potential epidemic areas shown in Panel A1. However, this result is not robust to allowing differential trends by geographic area. Similarly, we observe no robust treatment effect on primary education for men.

Panel B presents results for women, also revealing little evidence that eradication increased human capital. The literacy estimates are not statistically significant, and they change sign across specifications. The primary education results imply that those in potential epidemic and malarious areas experienced smaller gains than those in non-malarious areas; the point estimates also indicate that the gains in malarious areas were larger than those in potential epidemic areas. However, the coefficients become insignificant and change sign in our most demanding specification (region*post controls and district specific linear trends). We discuss these results further in relation to our instrumental variables estimates in Section 5.3.

We next examine the effects of the program on income, measured by per-capita household expenditure and a poverty dummy, as described above (*Table 7*). Unlike the human capital results, these results indicate a positive effect of eradication on adult male income. In Panel A1, the baseline specification (column [1]) implies a positive, monotonic program effect across the three categories of malaria endemicity. The treatment effect estimate for potential epidemic areas relative to non-malarious areas changes sign in the robustness checks, but the differences-in-differences between potential epidemic and malarious areas, which account for over 95 percent of or sample, are always positive.

Panel A2 presents the results for men using the index measure of malaria endemicity. A one unit increase in the pre-eradication malaria index is associated with a 0.8% increase in per-capita household expenditure. To put this magnitude in context, we can convert the malaria index into an approximate measure of the corresponding spleen rate using the slope of the 1953-54 regression line

in Figure 4. If we assume that malaria levels were reduced to zero in the post eradication period, this estimate implies that a 40 percentage point reduction in the spleen rate, as was experienced in the most malarious states, is associated with a 2 percent increase in per-capita household expenditure. Stated somewhat differently, a move from the ninety-fifth to the fifth percentile of the district-level malaria index distribution increases per-capita expenditure by 3 percent. The effect is quite robust to using very localized sources of geographic variation: the point estimate remains significant, and in fact increases, when we include state*post and region*post effects. When we include both region*post controls and district-specific linear trends, the effect is no longer significant but the point estimate is identical to the baseline specification. Consistent with these findings, we observe negative (but insignificant) coefficients on the poverty dummies.

Notably, we do not observe significant effects of the program on income outcomes for women (Panel B). Given women's lower rate of participation in the extra-household labor market, this suggests that the effects for men may be driven by improvements in labor market productivity arising from the eradication program.[18] Note that even if improvements in income are driven through this channel, we might have expected to see improvements for women if they are married to treated men. However, the average age gap among married couples in our sample is five years: women defined to be in our treatment group are, on average, married to men who were born during rather than after the eradication era. We have examined the effects for women using a five year lag, and we still observe no significant effects on income (unreported). This is likely a result of the fact that the "treated" women are now in the 1967-1972 birth cohorts, making them age 15-20 at the time of the survey. Only 41% of this treated group is married, and those that are married are likely to be a quite selected group.

5.2 Cohort Analysis

In this section, we examine outcomes by birth cohort over time. Motivated by the differences-in-differences results, we focus on the income and poverty effects for men. We run regression (B), using 5-year birth cohorts as described in the empirical strategy section. If the program had a positive treatment effect, we would expect to see increases in the plotted coefficients for post-eradication cohorts relative to pre-eradication cohorts.

[18]The human capital results are similar if we restrict the sample to prime ages (20-60), and the expenditure results for men are similar in the unrestricted age sample (unreported).

The top two panels of *Figure 5* show the coefficients on birth cohort*malaria index; the bottom two panels show the coefficients on birth cohort*potential epidemic and birth cohort* malarious. We do observe relative improvements in per-capita expenditure for those post-cohorts born in more malarious areas. If anything, relative outcomes appear to be trending down in malarious areas prior to the program and rise sharply for those born after eradication. The cohort effects for poverty do not exhibit a clear break at the time of the eradication era, which is not surprising, given the differences-in-differences results.

Our concerns that improvements for those affected by the program might reflect pre-existing trends are also alleviated by the robustness of the differences-in-differences estimates to geographic controls. This implies that any spurious trends across high and low prevalence areas would have to be reflected not only at the national level, but within state and within region as well. In the next section, we consider several possible sources of bias in our estimates.

5.3 Robustness Analysis

The cohort analysis gives a clear visual representation to our results, suggesting that our main estimates are not spurious. In this section, we discuss sensitivity of our results to accounting for measurement error and confounding trends.

One potential concern is whether the classifications of districts used here reflect true geographic variation in malaria prevalence in the pre-eradication period. The most likely source of bias is attenuation of the coefficients resulting from measurement error in our prevalence classifications.[19] We therefore instrument for the map classifications using the ecological factors shown in *Table A1*. Specifically, we use the interaction of the *Post* dummy with the covariates in column (3) as our excluded instruments in the first-stage of our instrumental variables specifications. The second-stage equation is identical to equation (A).

Table 8 reports the results of the IV estimates for educational and income outcomes for both men and women. For simplicity, we report all results using the malaria index measure of prevalence. The effects for men generally reflect the OLS estimates presented in *Tables 6 and 7*. We see implied negative treatment effects for men on educational outcomes, but these effects are not robust to the choice of geographic controls. The point estimates for per-capita expenditure and

poverty are now slightly larger in magnitude than the OLS estimates, and the IV estimates imply significant reductions in poverty as a result of the program, consistent with the effects on per-capita expenditure.

For women, we now observe positive coefficients on the index measure for literacy and primary outcomes, although the significance is not robust to the inclusion of region*post controls. When we use the three category measure in an otherwise identical instrumental variables setup, we obtain a non-monotonic pattern as with the OLS results: malarious areas experienced improvements relative to potential epidemic areas, but both experienced smaller gains than non-malarious areas (unreported). In the OLS specifications, this non-monotonicity resulted in no overall effect when using the index measure. In the IV specifications, the difference between potential epidemic and malarious areas is more pronounced, which is likely what drives the positive net effect in the index measure. These results provide suggestive evidence that the eradication program led to improvements in educational outcomes for women in malarious areas relative to potential epidemic areas. We find no robust effects for household expenditure for affected women.

Two other potential concerns arise with evaluating the effects of changes in disease burden: selective mortality and migration. With a lower disease burden, the weakest members of affected cohorts may survive, leading to potential compositional biases when evaluating outcomes among survivors. Mortality bias is unlikely to be a problem in our experiment since the predominant form of malaria in India is *P. vivax*, which leads to morbidity but only rarely mortality. Consistent with this, the pre-eradication era estimates in *Table 2* indicate an annual death rate from malaria of only 0.2%. In regards to migration, as mentioned above, we unfortunately do not observe individuals' districts of birth. However, only 7.5% of individuals in rural areas are living outside their districts of birth. In addition, the robustness of our point estimates to the source of geographic variation suggests that the effects are unlikely to be driven by migration alone.

It is also unlikely that these effects can be explained by other programs whose targeting was correlated with pre-eradication endemicity. All of the results presented above control for interactions between individual level demographics and an indicator for being born in the post period. The expenditure results for men are also robust to controlling for an interaction between income at the district level, averaged over the pre-cohorts, and the post indicator (unreported). In

[19] The instrumental variables estimates will produce consistent estimates if the measurement error is classical. This is an approximation in our case, given the categorical nature of the underlying variable; see Kane, Rouse and Staiger (1999).

order to generate the observed results, targeting would have also had to be correlated with malaria prevalence within localized geographic areas. We also directly examine one potential confounder: the early adoption of new agricultural technologies, defined by the use of high-yielding variety (HYV) seeds and chemical fertilizers in 1970, early in the Green Revolution. We do not find evidence that HYV adoption is correlated with malaria endemicity: the correlation between the district-level map index and proportion of land cropped with HYV in 1970 is -0.0019 (p-value = 0.97) and the correlation with the intensity of fertilizer use in 1970 is 0.0475 (p-value = 0.44).[20]

As a final pair of robustness checks, we have conducted two falsification exercises using our baseline specification. In the first, we assume that eradication took place one decade earlier than in reality. If our estimates were driven by pre-existing differential trends across districts of varying malaria endemicity, then the estimate of this "placebo" treatment's effect on male income would be positive, significant, and similar in magnitude to our main estimate. However, the coefficient on the interaction of the malaria index and a post placebo treatment dummy is small and insignificant (coef. = 0.003, SE = 0.004). The second falsification test draws on the unimportance of malaria in urban areas during the pre-eradication era. Consistent with this unimportance, we find no evidence of a positive treatment effect on male income (coef. = -0.014, SE = 0.014).

5.4 Interpretation of the Findings

Our estimates shed light on whether malaria eradication had effects, but our use of the 1948 malaria endemicity classifications makes their magnitudes difficult to interpret. What do the estimates imply for individuals who grew up in India's most malarious areas? How do the results compare with the existing literature's findings on eradication programs in other countries (Bleakley 2007, Lucas 2009)?

As discussed above, one way to gain further understanding from our estimates involves focusing on differences between India's most and least malarious districts. The ninety-fifth percentile of the malaria index is 5.7, while the fifth percentile is 2. Therefore, a move from the ninety-fifth to the fifth percentile induces an effect equal to 3.7 times the coefficient on the interaction of post with the malaria index. The men's expenditure point estimates range from 0.008

for further discussion.

[20] To study agricultural technology adoption, we use the India Agriculture and Climate Data Set from the World Bank. For the 271 districts in the dataset, we relate the malaria index with the quantity of fertilizer used per hectare of gross

to 0.035, implying that a move between these two percentiles increases per-capita expenditure by 3 to 13 percent. The baseline OLS regressions for women's human capital yield imprecisely estimated zeros, but the instrumental variables procedure increases these point estimates to at least 0.013, signifying that a move between the two percentiles increases female literacy and primary schooling by nearly 5 percentage points. We can glean further insight into the estimates by comparing them with those of the existing literature. The malaria index does not lend itself to a clear comparison with results from other settings, but we can rescale the coefficients to represent the effect in terms of a more recognizable measure. In line with Lucas (2009), we rescale the coefficients to infer the effect of a ten percent decline in malaria incidence. Unfortunately, NMEP materials (1986, 1996) do not report pre-eradication incidence at the sub-national level, thus preventing us from directly estimating the relationship between malaria incidence and the malaria index. However, the post-eradication data are rich enough to allow us to estimate the relationship between incidence and the child spleen rate. If eradication did not alter the relationship between incidence and the spleen rate (which is plausible), we can supplement this with the information in *Figure 4* on the correlation between the child spleen rate and the malaria index. Regression estimates using these data sources indicate that a ten percentage point increase in state-level malaria incidence is associated with a 28.8 percentage point rise in the child spleen rate. Furthermore, a percentage point increase in the spleen rate is associated with an increase of 0.067 in the malaria index (*Figure 4*). If we multiply our coefficients by the product of these two numbers, 1.93, the result tells the impact of a ten percentage point decrease in incidence.

The rescaled OLS and IV point estimates indicate that a ten percentage point decrease in incidence raises per-capita expenditure of between 1.5 and 6.8 percent, similar in magnitude to Bleakley's estimates for male earnings in Latin America. For women's human capital, the OLS results do not imply positive treatment effects. However, the rescaled IV estimates imply that a ten percentage point decrease in incidence increases female primary school attainment and literacy by 2.5 to 5.6 percentage points, with the most demanding specification (column [7] of *Table 8*) yielding estimates at the bottom of this range. These estimates are slightly higher than both Bleakley (2007) and Lucas (2009), who find that a change in incidence of the same magnitude raises literacy 0.08 to 2 percentage points.

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cropped area and the proportion of the gross cropped area sown with high-yielding varieties (HYV), both in 1970.

Importantly, the primary malaria parasite in India differs from the primary parasite responsible for malaria in sub-Saharan Africa, where malaria is most prevalent today. The effects of present-day malaria control efforts on long-run outcomes may therefore differ from the effects estimated in this study. For instance, in the absence of a large offsetting fertility response, eradication of the more fatal *P. falciparum* in sub-Saharan Africa is more likely to result in population growth, as discussed in Acemoglu and Johnson (2007). As a result, effects on cohorts exposed to eradication in childhood may differ in general equilibrium.

6 Conclusion

This paper examines the effects of a large scale eradication program that drastically reduced malaria in India over a short time period. Exploiting the heterogeneity in indigenous malaria rates and the exogenous implementation of the eradication program, we find that malaria eradication resulted in improvements in income for males. We do not observe robust treatment effects for education, which may reflect the tradeoff between schooling and labor emphasized by Bleakley (2007). The program does not appear to have resulted in similar improvements in income for women, suggesting that the observed effects for men are likely driven by increased productivity in the labor market, where male participation rates much higher than female.

The estimated income gains for men who benefited from the program are similar when we exploit national, state, or regional sources of geographic variation in pre-eradication prevalence, alleviating concerns that the effects are driven by other omitted factors. Furthermore, the results are robust to instrumenting for pre-eradication prevalence with the ecological determinants of malaria endemicity.

Our results provide support for the belief that improvements in health and in the disease environment can have a causal effect on income. This may be an important causal channel linking health and economic growth.

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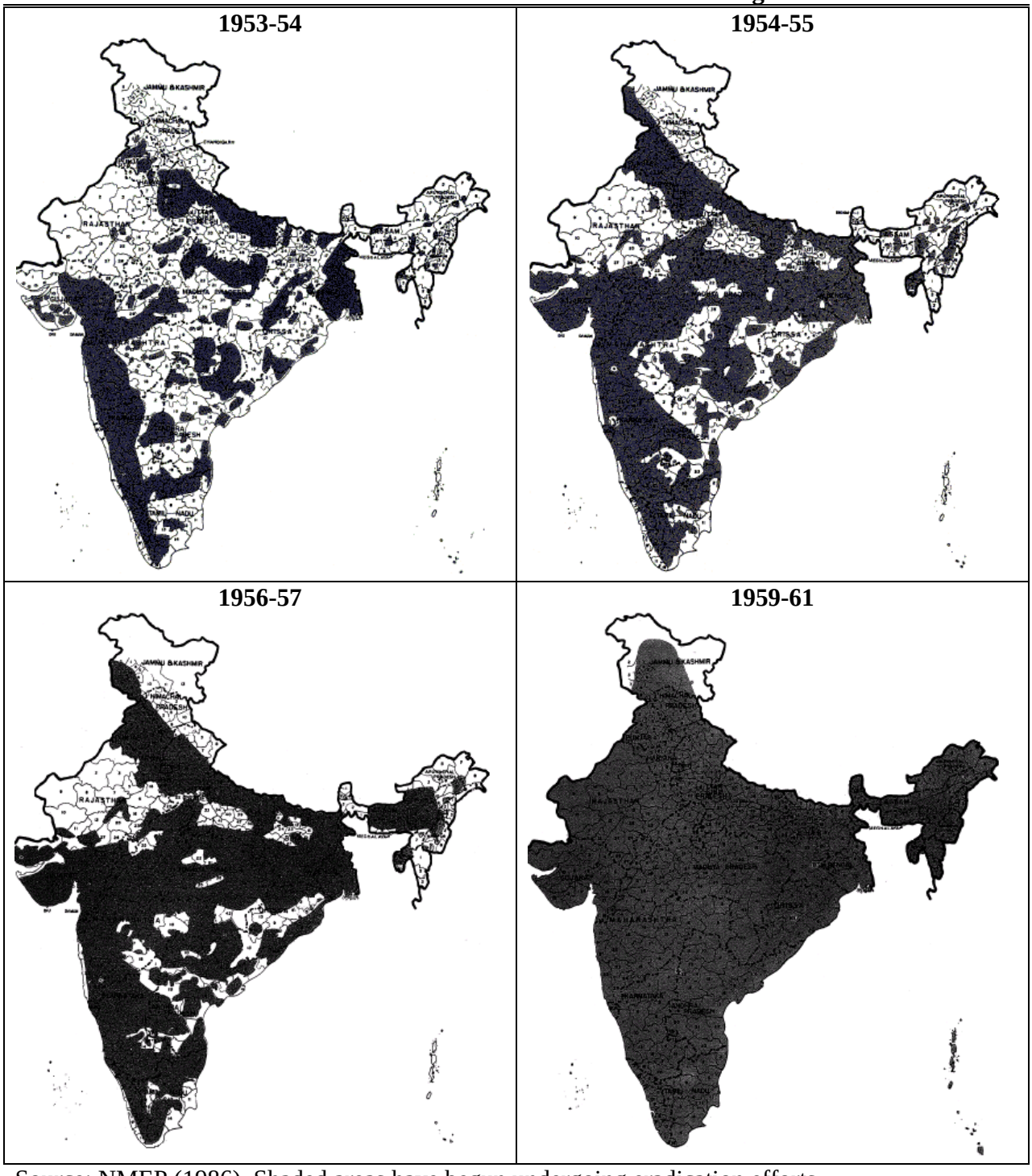
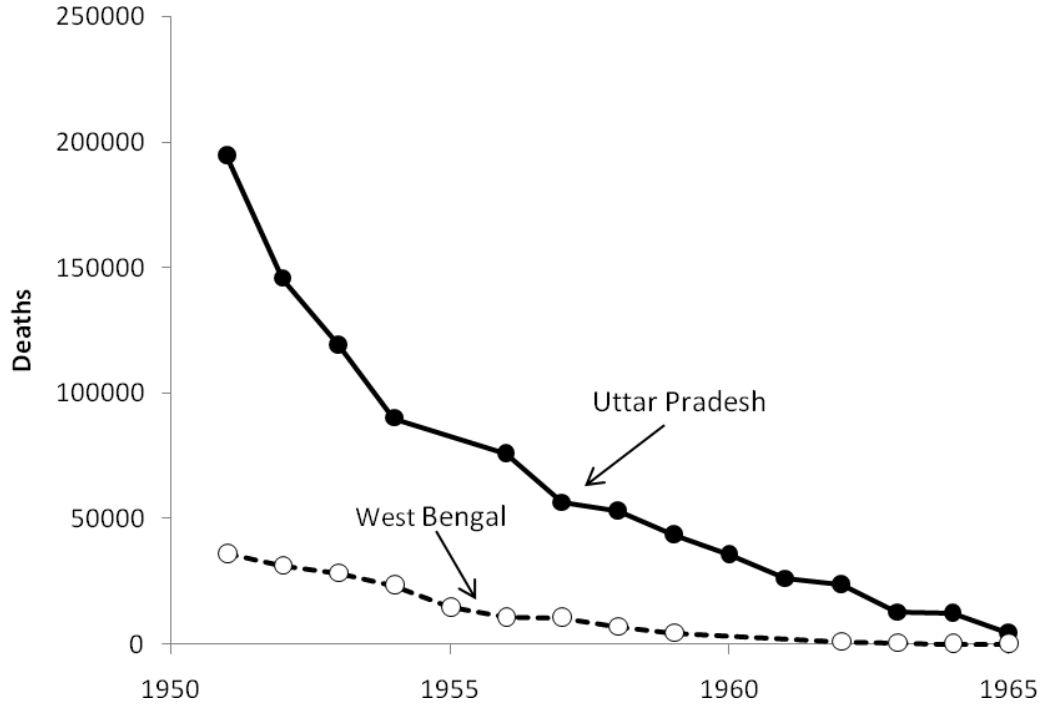
FIGURE 1
Phases of the National Malaria Eradication Program



Source: NMEP (1986). Shaded areas have begun undergoing eradication efforts.

FIGURE 2
Declines in Malaria Deaths



Source: *Health Statistics of India, 1951-1965*. We show West Bengal and Uttar Pradesh due to these states' relatively complete time series, as well as the stability of their borders through the States Reorganization Act of 1956. The figures above imply a ratio of malaria deaths to population of 0.3% in Uttar Pradesh and 0.1% in West Bengal in 1952. These rates are similar to the nationwide rate in 1947 (0.2%) implied by the NMCP figures in *Table 1*.

FIGURE 3
Malaria Endemicity Map

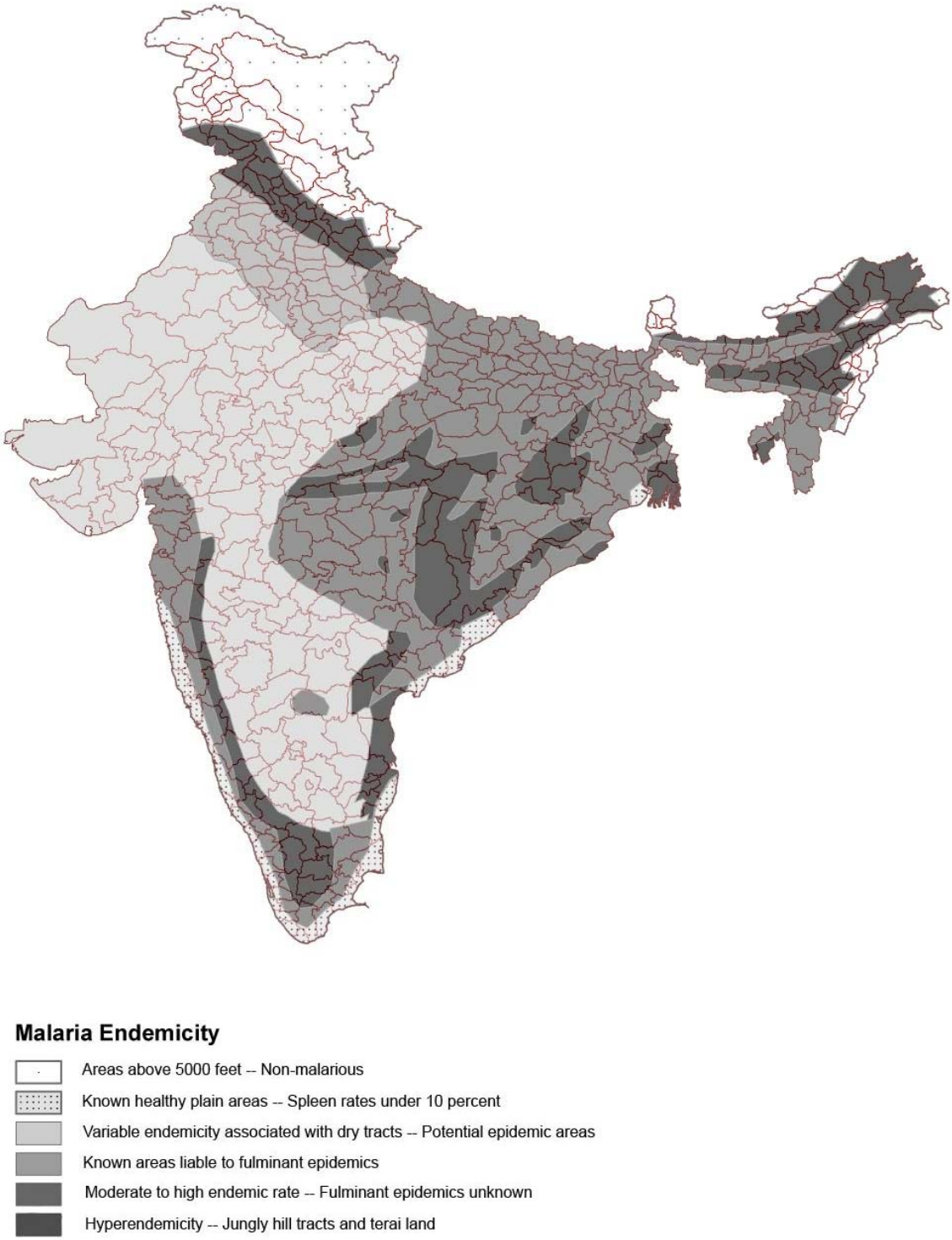
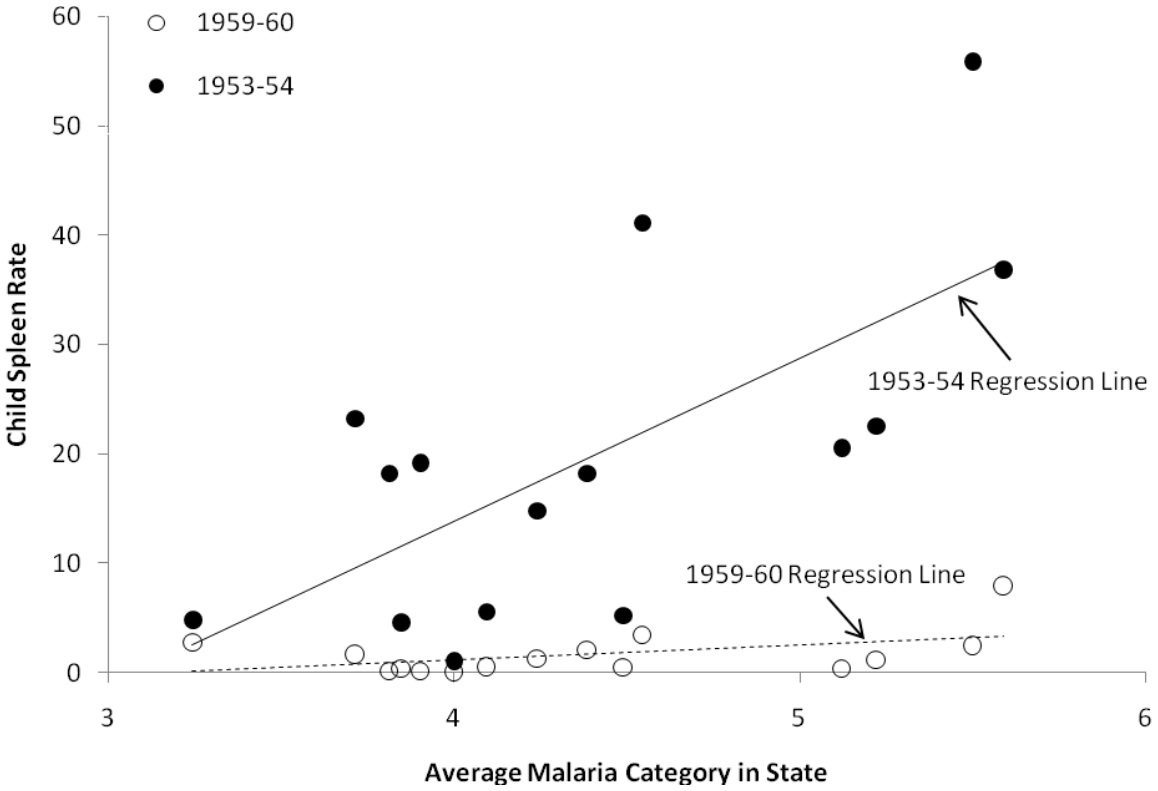


FIGURE 4
1948 Malaria Endemicity and Child Spleen Rates in 15 States and Territories

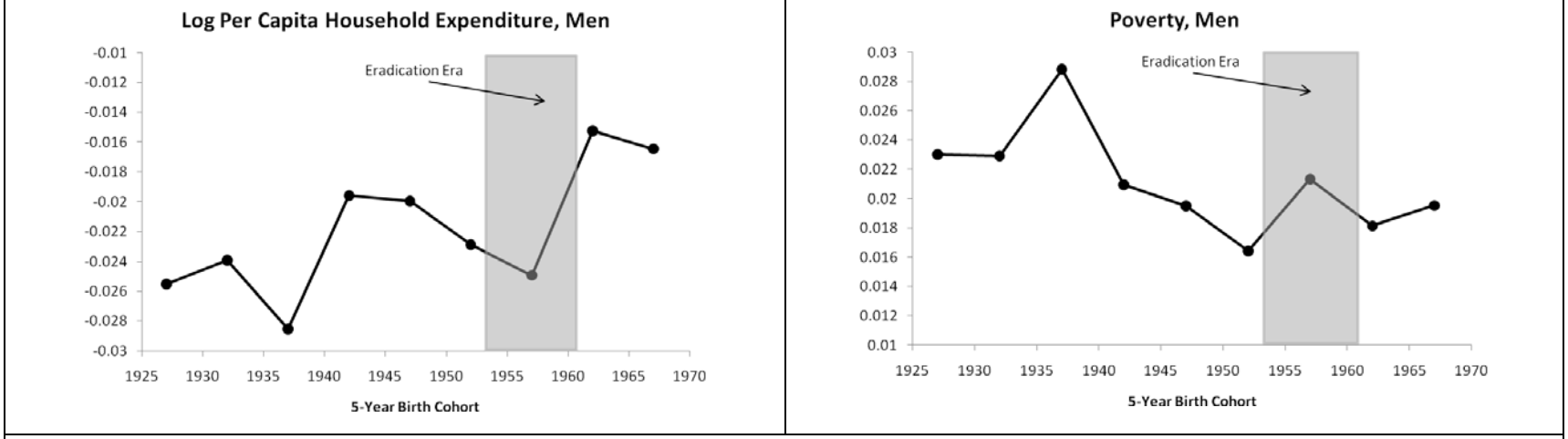


Source: NMCP (1986). Sample includes all states and territories with child spleen rate data for both 1953-54 and 1959-60. See notes to Table 3 for details on the construction of state-level spleen rates for 1953-54. We obtain the state-level average malaria category by averaging the categories of all GIS polygons within each state. The slopes of the 1953-54 and 1959-60 regression lines, respectively, are 14.9 and 1.4.

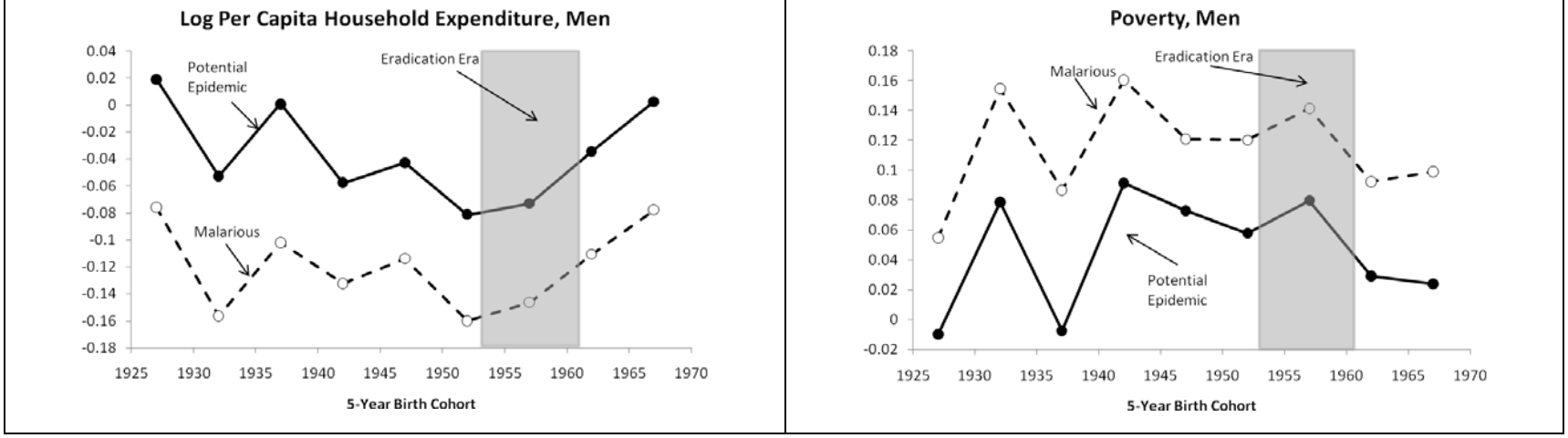
FIGURE 5

Pre-Eradication Malaria Endemicity and Adult Economic Status: Cohort-Specific Relationships

1. Districts Classified by Average Malaria Category



2. Districts Classified by Modal Malaria Category



Note: Relationships were estimated in regressions of economic outcomes on cohort fixed effects and interactions of cohort fixed effects with measures of malaria endemicity: modal malaria category (relative to non-malarious) in the top panel and average malaria category in the bottom. The 5-year birth cohorts are centered on birth years ending in 2 and 7 (ages ending in 5 and 10). To focus on individuals of relevant ages (20+), the last birth cohort in each panel is three years long. Regressions also included dummies for membership in a scheduled caste or tribe and household religion

TABLE 1
Expansion of the National Malaria Control Program

	# Units established	# Units established (cumulative)	Population protected (cumulative, in millions)	% of population protected
1953-54	84.00	84.00	49.50	13.2
1954-55	26.75	110.75	79.90	20.9
1955-56	23.00	133.75	112.00	28.7
1956-57	35.50	169.25	144.50	36.3
1957-58	23.25	192.50	165.57	40.8

Source: NMCP (1986). A "unit" is defined as "a contiguous area comprising of one million population for undertaking anti-malaria activities" (NMEP, 1986). Since units are defined by population covered, they are not always in integers. The population statistics used to calculate the percentage of the population protected are from the United Nations Demographic Yearbook Historical Supplement. We use the 1953 midyear population figure for 1953-54, etc.

TABLE 2
National Malaria Prevalence over Time

	Population (in millions)	Malaria cases (in millions)	% of population with malaria	# Deaths
1947	344	75	21.8	800,000
1965	483	0.1	0.02	0
1976	616	6.5	1.1	59
1984	735	2.2	0.3	247
1994	900	2.5	0.3	1122

Source: NMCP (1986), NMCP (1996). The population statistics used to calculate the percentage of the population with malaria are from the United Nations Demographic Yearbook Historical Supplement.

TABLE 3
Child Spleen Rates in 15 Indian States and Territories

	1953-54	1954-55	1955-56	1956-57	1959-60
States					
Andhra Pradesh	18.2	14.2	12.8	13.0	2.1
Bihar	22.5	23.2	18.2	11.8	1.2
Bombay	5.6	4.5	4.1	3.6	0.6
Kerala	19.2	5.3	4.6	4.6	0.2
Madhya Pradesh	41.1	22.7	12.6	12.3	3.5
Mysore	4.6	3.0	2.7	1.9	0.4
Orissa	37.0	29.0	19.0	—	8.0
Punjab	5.3	5.2	2.5	2.0	0.5
Rajasthan	4.9	4.4	21.2	18.0	2.8
Uttar Pradesh	14.8	19.6	13.5	13.9	1.3
West Bengal	20.6	16.5	6.9	4.0	0.4
Union Territories					
Delhi	1.1	0.5	0.3	0.5	0.1
Himachal Pradesh	18.2	14.1	2.7	1.2	0.2
Manipur	23.3	17.8	12.0	13.2	1.7
Tripura	55.9	61.2	18.1	3.9	2.5

Source: NMCP (1986). Sample includes all states and territories with child spleen rate data for 1953-54. These rates are based on tables that report the results of NMCP spleen surveys. For the entire eradication era, the tables use consistent administrative boundaries as defined by the States Reorganization Act of 1956. The tables for 1953-54 to 1956-57 divide areas within states and territories into those sprayed with DDT and those still unsprayed. We calculate the total number of enlarged spleens detected in each state/territory and divide by total number of examinations performed in the state/territory.

TABLE 4
Malaria Endemicity Classifications

Classification for Paper	1948 Map Category
Non-malarious	(1) Areas above 5000 feet; non-malarious.
	(2) Known healthy plain areas; spleen rate under 10%.
Potential Epidemic	(3) Variable endemicity associated with dry tracts; potential epidemic.
	(4) Known areas liable to fulminant epidemic diluvial malaria.
Malarious	(5) Moderate to high endemicity; fulminant epidemics unknown.
	(6) Hyperendemicity of jungly hill tracts and terai land.

TABLE 5
Summary Statistics

	Men		Women	
	Mean	(Std. Dev.)	Mean	(Std. Dev.)
Panel A: Outcomes				
Human Capital Sample (Ages 15-75)				
Literate	0.53	(0.50)	0.23	(0.42)
Primary school attainment	0.39	(0.49)	0.16	(0.37)
Economic Status Sample (Ages 20-60)				
Log per capita household expenditure	4.97	(0.48)	4.97	(0.48)
Poor	0.34	(0.47)	0.35	(0.48)
Panel B: Malaria Endemicity Measures (Combined Sample)				
Avg. malaria category in district (1-6)	4.38	(0.98)	4.39	(0.98)
Modal malaria category in district				
Non-malarious (1 and 2)	0.02	(0.14)	0.02	(0.14)
Potential epidemic (3 and 4)	0.24	(0.43)	0.23	(0.42)
Endemic malaria (5 and 6)	0.53	(0.50)	0.54	(0.50)
Bimodal	0.20	(0.40)	0.20	(0.40)
Panel C: Demographic Characteristics (Combined Sample)				
Age	36.29	(16.61)	36.39	(16.43)
Married	0.66	(0.47)	0.71	(0.45)
Scheduled caste	0.18	(0.39)	0.18	(0.39)
Scheduled tribe	0.10	(0.30)	0.10	(0.30)
Hindu	0.85	(0.36)	0.85	(0.36)
Muslim	0.09	(0.29)	0.10	(0.30)
Number of observations	111,218		107,551	

Number of states	29
Number of regions	75
Number of districts	417
Number of households	77,020

Note: Means and standard deviations are weighted using sampling weights. Sample includes rural residents and excludes those born during the eradication era (1953-1961). Panels B and C report summary statistics and sample sizes for the sample with non-missing data on at least one outcome.

TABLE 6
Childhood Malaria Exposure and Human Capital Attainment

Dependent Variable:	Literacy (Ages 15-75)				Primary School (Ages 15-75)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Men								
A1. Districts Classified by Modal Malaria Category *(Omitted Category: Post * Non-malarious)*								
Post * Potential Epidemic	0.035	-0.007	-0.030	-0.105	0.021	0.009	0.000	0.021
	(0.032)	(0.026)	(0.032)	(0.066)	(0.047)	(0.044)	(0.044)	(0.060)
Post * Malarious	-0.015	0.013	0.003	-0.045	-0.020	0.014	0.001	0.050
	(0.032)	(0.021)	(0.025)	(0.047)	(0.047)	(0.041)	(0.037)	(0.045)
F-test: Equal Treatment Effects (*p*-value)	<0.001	0.213	0.106	0.200	0.003	0.790	0.958	0.220
Observations	88,639	88,639	88,639	88,639	88,639	88,639	88,639	88,639
A2. Districts Classified by Average Malaria Category								
Post * Malaria Index	-0.017	0.004	-0.001	0.008	-0.016	0.002	-0.005	0.009
	(.006)***	(0.005)	(0.006)	(0.011)	(0.007)**	(0.006)	(0.008)	(0.010)
Observations	111,139	111,139	111,139	111,139	111,139	111,139	111,139	111,139
State*Post Fixed Effects		X				X		
Region*Post Fixed Effects			X	X			X	X
District-Specific Linear Trends				X				X

(Table continued on next page.)

Dependent Variable:	Literacy (Ages 15-75)				Primary School (Ages 15-75)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel B: Women								
B1. Districts Classified by Modal Malaria Category *(Omitted Category: Post * Non-malarious)*								
Post * Potential Epidemic	-0.053	-0.005	-0.016	0.052	-0.132	-0.071	-0.074	0.054
	(0.031)*	(0.028)	(0.034)	(0.066)	(.049)***	(0.040)*	(0.045)*	(0.038)
Post * Malarious	-0.026	0.016	-0.018	0.043	-0.101	-0.045	-0.066	0.043
	(0.030)	(0.023)	(0.020)	(0.062)	(0.049)**	(0.037)	(0.036)*	(0.033)
F-test: Equal Treatment Effects (*p*-value)	0.076	0.223	0.933	0.708	0.030	0.124	0.764	0.596
Observations	85,291	85,291	85,291	85,291	85,291	85,291	85,291	85,291
B2. Districts Classified by Average Malaria Category								
Post * Malaria Index	0.005	0.011	-0.006	0.008	-0.004	0.005	-0.012	0.002
	(0.006)	(0.006)*	(0.006)	(0.010)	(0.007)	(0.006)	(0.008)	(0.007)
Observations	107,472	107,472	107,472	107,472	107,472	107,472	107,472	107,472
State*Post Fixed Effects		X				X		
Region*Post Fixed Effects			X	X			X	X
District-Specific Linear Trends				X				X

Note: OLS coefficients, with standard errors clustered at the district level in parentheses. Sample includes rural residents from pre-eradication (1912-1952) and post-eradication (1962-1972) cohorts. The sample in Panels A1 and B1 omits individuals living in bimodal districts. All regressions include district and year of birth fixed effects, as well as demographic covariates and their interaction with post. Demographic covariates include membership in a scheduled caste, membership in a scheduled tribe, and household religion. * significant at 10%; ** significant at 5%; *** significant at 1%.

TABLE 7
Childhood Malaria Exposure and Adult Economic Status

Dependent Variable:	Log Per Capita Household Expenditure (Ages 20-60)				Poverty (Ages 20-60)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Men								
A1. Districts Classified by Modal Malaria Category *(Omitted Category: Post * Non-malarious)*								
Post * Potential Epidemic	0.011	0.011	-0.054	0.037	-0.012	-0.013	0.037	-0.069
	(0.015)	(0.024)	(0.032)*	(0.062)	(0.017)	(0.028)	(0.035)	(0.067)
Post * Malarious	0.033	0.034	0.018	0.082	-0.018	-0.016	-0.005	-0.091
	(.014)**	(0.021)	(0.027)	(0.052)	(0.016)	(0.024)	(0.028)	(0.048)*
F-test: Equal Treatment Effects (*p*-value)	0.055	0.080	< 0.001	0.187	0.563	0.807	0.037	0.622
Observations	59,906	59,906	59,906	59,906	59,906	59,906	59,906	59,906
A2. Districts Classified by Average Malaria Category								
Post * Malaria Index	0.008	0.011	0.019	0.008	-0.003	-0.001	-0.008	-0.014
	(0.004)**	(0.005)**	(.006)***	(0.011)	(0.004)	(0.005)	(0.007)	(0.013)
Observations	75,230	75,230	75,230	75,230	75,230	75,230	75,230	75,230
State*Post Fixed Effects		X				X		
Region*Post Fixed Effects			X	X			X	X
District-Specific Linear Trends				X				X

(Table continued on next page.)

Dependent Variable:	Log Per Capita Household Expenditure (Ages 20-60)				Poverty (Ages 20-60)			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel B: Women								
B1. Districts Classified by Modal Malaria Category *(Omitted Category: Post * Non-malarious)*								
Post * Potential Epidemic	0.011	-0.006	-0.016	-0.014	-0.035	-0.012	0.050	0.002
	(0.015)	(0.028)	(0.039)	(0.077)	(0.024)	(0.040)	(0.047)	(0.093)
Post * Malarious	-0.003	-0.014	-0.015	0.027	-0.008	0.006	0.026	-0.050
	(0.013)	(0.024)	(0.031)	(0.057)	(0.023)	(0.037)	(0.037)	(0.070)
F-test: Equal Treatment Effects (*p*-value)	0.163	0.570	0.967	0.425	0.018	0.218	0.440	0.396
Observations	59,617	59,617	59,617	59,617	59,617	59,617	59,617	59,617
B2. Districts Classified by Average Malaria Category								
Post * Malaria Index	-0.003	-0.003	0.004	0.011	0.006	0.006	-0.004	-0.017
	(0.004)	(0.004)	(0.005)	(0.014)	(0.005)	(0.005)	(0.006)	(0.014)
Observations	75,212	75,212	75,212	75,212	75,212	75,212	75,212	75,212
State*Post Fixed Effects		X				X		
Region*Post Fixed Effects			X	X			X	X
District-Specific Linear Trends				X				X

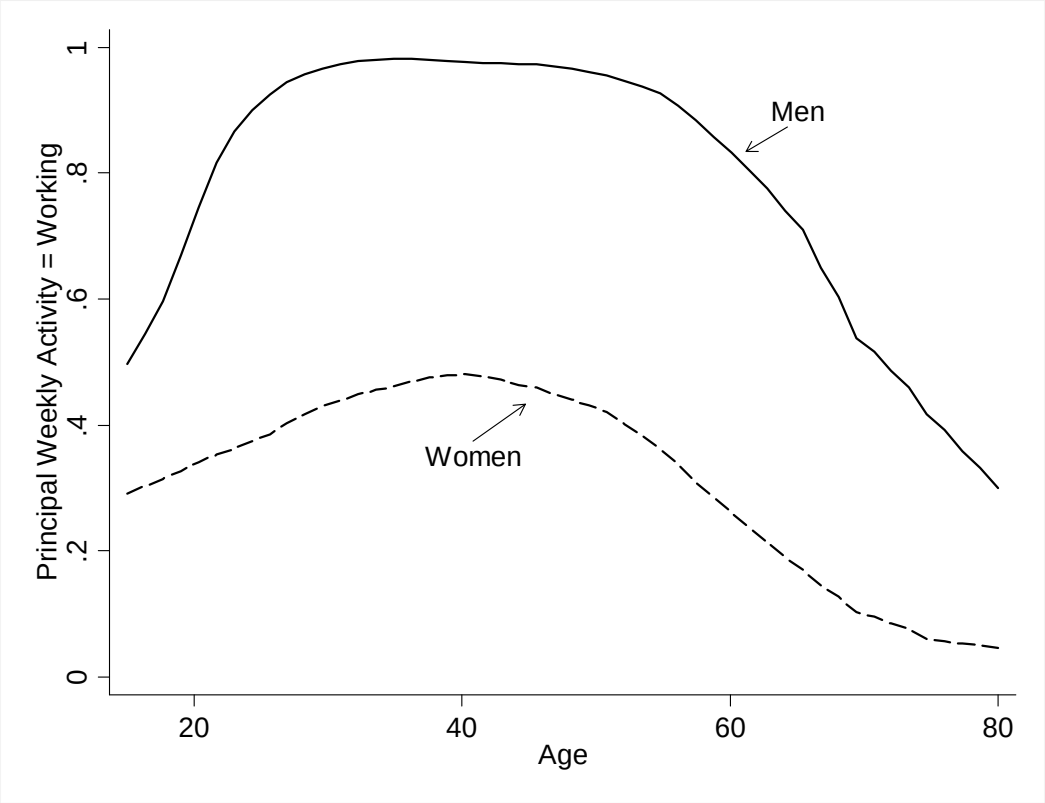
Note: OLS coefficients, with standard errors clustered at the district level in parentheses. Sample includes rural residents from pre-eradication (1927-1952) and post-eradication (1962-1967) cohorts. The sample in Panels A1 and B1 omits individuals living in bimodal districts. All regressions include district and year of birth fixed effects, as well as demographic covariates and their interaction with post. Demographic covariates include membership in a scheduled caste, membership in a scheduled tribe, and household religion. * significant at 10%; ** significant at 5%; *** significant at 1%.

TABLE 8
Instrumental Variables Estimates

	Men			Women		
	(1)	(2)	(3)	(5)	(6)	(7)
Dependent Variable:						
Literacy	-0.029	0.018	0.002	0.013	0.029	0.014
(111,000 men; 107,308 women)	(0.009)***	(0.010)*	(0.009)	(0.009)	(0.011)***	(0.012)
Primary schooling	-0.022	0.020	0.006	0.016	0.029	0.018
(111,000 men; 107,308 women)	(0.009)**	(0.011)*	(0.010)	(0.009)*	(0.011)***	(0.011)
Log per capita H.H. expenditures	0.0094	0.026	0.035	0.000	0.002	0.009
(*75.131 men; 75,102 women)*	(0.006)*	(0.010)***	(0.015)**	(0.006)	(0.006)	(0.009)
Poverty	-0.011	-0.016	-0.019	0.001	-0.003	-0.007
(*75.131 men; 75,102 women)*	(0.006)*	(0.008)**	(0.011)*	(0.006)	(0.007)	(0.007)
State*Post Fixed Effects		X			X	
Region*Post Fixed Effects			X			X

Note: Each cell shows the coefficient on the malaria index from a separate regression. The excluded instruments include average temperature, average elevation, average humidity, average precipitation, and squared terms in all four variables. The sample sizes differ from *Tables 6 and 7* because two districts did not have ecology data. The OLS results for the subsample used for this table are identical to those reported in earlier tables for the full sample. See column (3) of *Table A1* for the district-level first stage regression. Parentheses contain standard errors clustered at the district level. The human capital sample includes rural residents from pre-eradication (1912-1952) and post-eradication (1962-1972) cohorts, whereas the consumption sample includes only the 1927-1952 and 1962-1967 cohorts. All regressions include district and year of birth fixed effects, as well as demographic covariates and their interaction with post. Demographic covariates include membership in a scheduled caste, membership in a scheduled tribe, and household religion**.** * significant at 10%; ** significant at 5%; *** significant at 1%.

FIGURE A1
Age Profiles of Labor Force Participation



Note: Profiles are based on the entire rural sample of the NSS. The curves are from local linear regressions estimates with a bandwidth of 2 years. We define the following categories as working: worked in household enterprise (self-employed), worked as helper in household enterprise, worked as regular salaried wage employee, worked as casual wage labor in public works, worked as casual wage labor in other types of work.

TABLE A1
Ecological Determinants of Pre-Eradication Malaria Endemicity

	Summary Statistics, Ecology Measures	Malaria Index	
	(1)	(2)	(3)
Avg. temperature (°C)	24.05	-0.086	1.22
	[4.85]	(0.034)**	(0.302)***
(Avg. temperature)^2/100			-0.027
			(0.006)***
Avg. precipitation (mm/month)	106.68	0.002	0.011
	[64.16]	(0.001)	(0.003)***
(Avg. monthly precipitation)^2/100			-0.002
			(0.001)**
Avg. humidity (%)	62.44	0.02	0.519
	[9.37]	(0.007)***	(0.060)***
(Avg. humidity)^2			-0.004
			(0.0004)***
Avg. elevation (km)	0.50	-1.144	-1.425
	[0.74]	(0.209)***	(0.245)***
(Avg. elevation)^2			0.862
			(0.196)***
R^2		0.25	0.43
F-statistic (p-value)		23.16 (<0.0001)	41.12 (<0.0001)
Number of Districts		415	415

Note: Climatologic and topographic data are from the World Water and Climate Atlas (New et al. 2002). Column (1) lists means of the independent variables, with standard deviations in brackets. Columns (2) and (3) report coefficients from regressions of the average malaria category in a district (the malaria index) on the variables listed in the table, with robust standard errors in parentheses. * significant at 10%; ** significant at 5%; *** significant at 1%.