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Early-life Malaria Exposure and Adult Outcomes: Evidence from Malaria Eradication in India†

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We examine the effects of exposure to malaria in early childhood on educational attainment and economic status in adulthood 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 household per capita consumption for prime age men, and the effects for men are larger than those for women in most specifications. We find no evidence of increased educational attainment for men and mixed evidence for women. (*JEL* I12, I18, I21, 015, 018)

Malaria, a disease that has afflicted humans for more than 10,000 years (Frederick L. Dunn 2003), today infects some 300 million people and kills 1 million each year (Jeffrey D. Sachs 2001). Despite important advances in the control of malaria during the twentieth century, the disease remains stubbornly prevalent throughout much of the world. Faced with this huge global burden, international organizations have redoubled their efforts to combat the disease.

Many argue that improving health, while important in itself, can also lead to higher economic growth and development. John Luke Gallup and Sachs (2001) show that falciparum malaria endemicity is negatively correlated with economic growth across countries.[1] In contrast, Daron Acemoglu and Simon Johnson (2007) argue that the wave of international health innovations that began in the 1940s did not lead

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

to a disproportionate increase in log per capita gross domestic product (GDP) in areas with a high pre-intervention disease burden.

In this paper, we extend this literature by studying the effects of childhood exposure to malaria eradication on human capital accumulation and economic status in adulthood. Malaria has been hypothesized to have lifelong effects on skill acquisition through at least three channels: its effects on cognitive ability, school absenteeism, and fetal development (e.g., Sachs and Pia Malaney 2002).[2] These sequelae do not necessarily imply that eradication of the disease will always lead to improvements in schooling attainment. As emphasized by Hoyt Bleakley (2010), 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 in both education and work.[3] In this case, eradication could boost adult income even without affecting schooling attainment. The health benefits of malaria reduction could result in improved physical and mental condition later in life and therefore higher labor market productivity. Furthermore, a reduction in school absences could enhance learning, thus improving literacy and earnings even holding years of schooling constant.[4]

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 between areas that experienced large reductions in malaria and those that experienced small reductions in malaria.[5] We do, however, observe modest relative increases in economic status (proxied by household per capita household expenditure) for prime age men. This effect is robust to using localized sources of geographic variation and to instrumenting for pre-eradication prevalence using geographic and climate factors, although in our most demanding specification (identified using within-region variation and including district linear trends) the point estimates remain similar but become imprecise. We do not observe significant increases in expenditure as a result of the program for women, and these gender differences cannot be explained by differences in the household composition of treated men and treated women. In most, but not all, of our specifications, the gender differences are statistically significant. This

[2] Randomized evaluations have documented effects of malaria on cognitive ability and school absenteeism (Charlotte Leighton and Rebecca Foster 1993; Moses Aikins 1995; S. Brooker et al. 2000; Sian E. Clarke et al. 2008). Additionally, Matthew C. H. Jukes et al. (2006) find suggestive evidence of a positive long-run effect of childhood malaria prophylaxis on educational attainment.

[3] 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 (David Card 2001), but this is an empirical claim, not a theoretical prediction.

[4] Eradication may also lead to the survival of children with poorer health and weaker cognitive skills. This is unlikely to be the case in our setting because, as described below, the most prevalent form of malaria in India is generally nonfatal.

[5] These results differ from the draft of an earlier working paper. This version corrects errors discovered in the original program files and extends the empirical analysis along a number of dimensions. The IV results do suggest improvements in educational attainment resulting from the program for women. We discuss these results in detail below.

suggests that improved labor market productivity may underlie the observed effects for men, given their much higher rates of labor force participation.

Our findings relate most closely to two papers that examine the effects of malaria eradication campaigns in other parts of the world. Bleakley (2010) studies the effect of malaria eradication campaigns on the income and education of men in the United States, Brazil, Colombia, and Mexico. He finds that childhood exposure to malaria lowers labor productivity and leads to lower adult income. Results for years of schooling are mixed. Adrienne Lucas (2010) uses a similar strategy to study ever-married women in Paraguay and Sri Lanka, and she finds that malaria eradication increases female education and literacy rates.[6] We discuss our findings relative to this literature in Section IV. Together with these papers, our results suggest that tropical disease control generates important economic benefits, but that it is unlikely to be a major driver of economic growth.

The paper proceeds as follows. Section I describes malaria in India in the pre-eradication era and discusses the National Malaria Control Program in India and its effectiveness. Section II outlines our empirical strategy. Section III describes the data. Section IV presents our results, and Section V concludes.

I. Malaria in India

Efforts to control malaria in India date back to the early 1900s, but were revolutionized in the mid-1940s with the advent of DDT (dichlorodiphenyl trichloroethylene).[7] DDT was effective, nontoxic to humans, and "dirt-cheap to manufacture" (Robert S. 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 (Kingsley Davis 1956).

DDT was first used in India by the military in 1944 and became available for civilian anti-malaria operations in 1945. Successful pilot programs in the late 1940s led the National Planning Commission to endorse the development of a comprehensive, nationwide program, and the National Malaria Control Program (NMCP) was launched in April of 1953. 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 per year (double during epidemic years), and 800,000 deaths were directly attributable to malaria annually

[6] Lucas (2007) finds that malaria eradication in Sri Lanka led to an initial increase in fertility followed by lowered fertility in the second generation. 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. Sok Chul Hong (2007) finds that malarial risk leads to adverse long-run health outcomes, lower labor force participation, and lower wealth. Alan Barreca (forthcoming) finds that in utero and postnatal malaria exposure leads to lower educational attainment.

[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 (National Malaria Eradication Program (NMEP) 1986).

(J. A. Sinton 1935, 1936; B. A. Rao 1959). The population of India in 1947 was 344 million, implying an annual incidence rate of 22 percent.

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 percent 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 percent of cases were due to *P. falciparum* (NMEP 1996).

The NMCP's main operational activity was to conduct two annual rounds of DDT spraying of human dwellings and cattle sheds. By 1956, 112 million people were estimated to be protected (NMEP 1986). In 1958, the program was reformulated as the NMEP with the goal of completely eradicating malaria from the nation, and by 1960–1961, the entire country was brought under the program (Web Appendix Table 1).

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 later increased (although to much lower levels than pre-eradication rural malaria), and the government launched an Urban Malaria Scheme in 1971 to address this problem. We therefore focus our analysis on rural areas.

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 in 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 (Web Appendix 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. Both state-level data on child spleen rates and vital statistics data on malaria deaths show substantial declines during the eradication era (Web Appendix Table 3 and Web Appendix Figure 1).[10]

[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, 251).

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

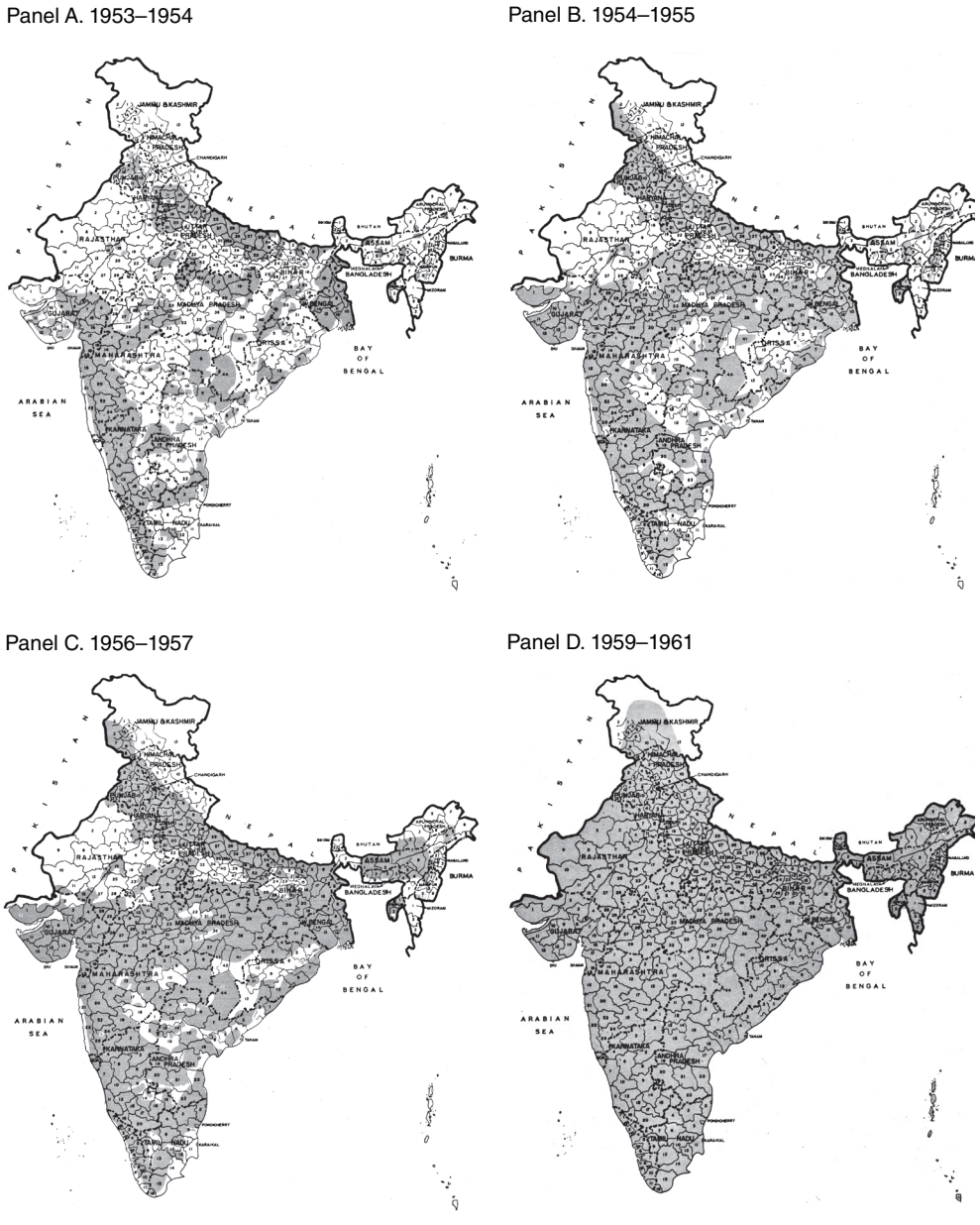

FIGURE 1. PHASES OF THE NATIONAL MALARIA ERADICATION PROGRAM

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

Malaria prevalence remained low throughout the 1960s, then experienced a slight resurgence in the 1970s, peaking in 1976. However, even at the peak of the resurgence, the incidence rate was only 1.1 percent. Reported prevalence then 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.

II. Empirical Strategy

Our study focuses on the effects of early-life malaria exposure on subsequent human capital attainment and consumption in adulthood. We use a differences-in-differences design, exploiting geographic variation in the prevalence of malaria prior to the eradication program. 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. Therefore, an identifying assumption of our analysis is that the district of residence is a good proxy for district of birth. In the 1991 Census of India, only 7.5 percent 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. Infants, children, and pregnant women are at high risk for malaria-related morbidity, and 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 (2010). 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. Thus, we 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:

$$(1) \qquad Outcome_{icd} = \beta(Post)_c \times (Malaria)_d + \delta_d + \alpha_c + \mathbf{X}'_{icd}\,\gamma_{pre} + (Post)_c \times \mathbf{X}'_{icd}\,\gamma_{post} + \varepsilon_{icd},$$

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.[11] δ and α are district and birth cohort fixed effects. The vector X includes membership in a scheduled caste, membership in a scheduled 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 (1) separately for men and women. In robustness checks, we add several other time-varying

[11] We exclude individuals born during the eradication era (1953–1961). 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 (NMEP 1986). Finally, the phase-in of the program was quite rapid, raising further difficulties in exploiting variation in timing of coverage.

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:

$$(2) \qquad Outcome_{icd} = \sum_{c} \beta_c \times (Malaria)_d + \alpha_c + \mathbf{X}'_{icd}\gamma + \varepsilon_{icd},$$

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 well-being 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 five-year birth cohorts for the graphical analysis, centered on years ending in the digits 2 and 7.

III. Data

A. *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 the disease burden of malaria. 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; (2) non-malarious; (3) known healthy plain areas, spleen rate under 10 percent; (4) variable endemicity associated with dry tracts, potential epidemic areas; (5) known areas liable to fulminant epidemic diluvial malaria; (6) moderate to high endemicity, fulminant epidemics unknown; and 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 2 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 14 other districts that lack observations in the NSS that satisfy our sample inclusion criteria (described below), leaving 417 districts in our main sample.

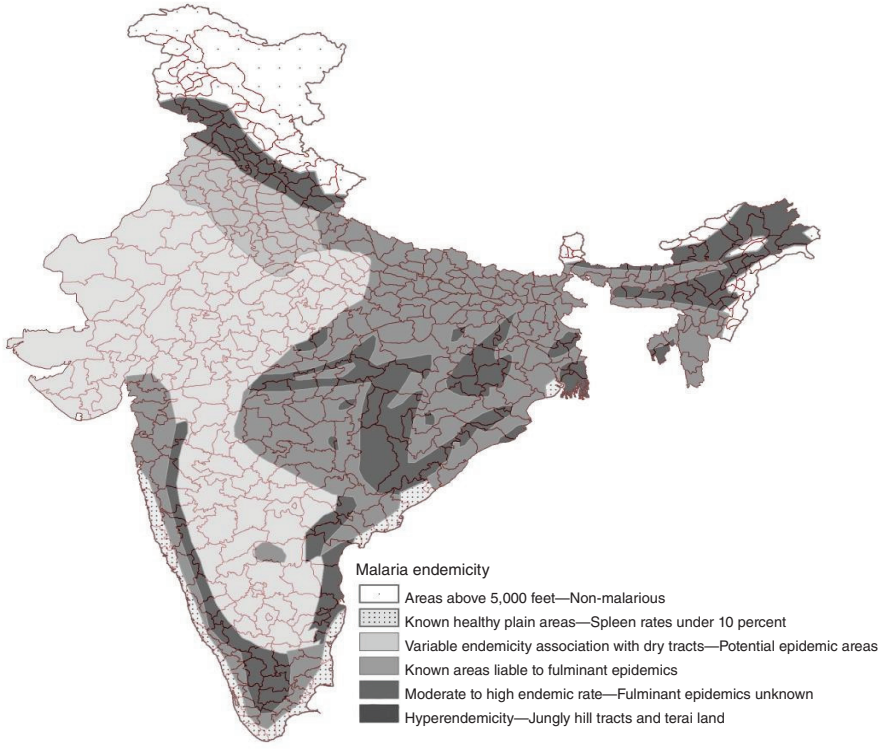

FIGURE 2. MALARIA ENDEMICITY MAP

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. Categories 1 and 2 (as described above) are classified as non-malarious, categories 3 and 4 are classified as potential epidemic, and categories 5 and 6 are classified as malarious.[12] We then categorize each district by its modal polygon malaria category. Seventy-seven districts do not have unique modes. For example, some mountainous districts in northern India have equal numbers of non-malarious, high-altitude polygons and malarious, low-altitude polygons.

[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 children and adults, and can result in severe adverse health consequences.

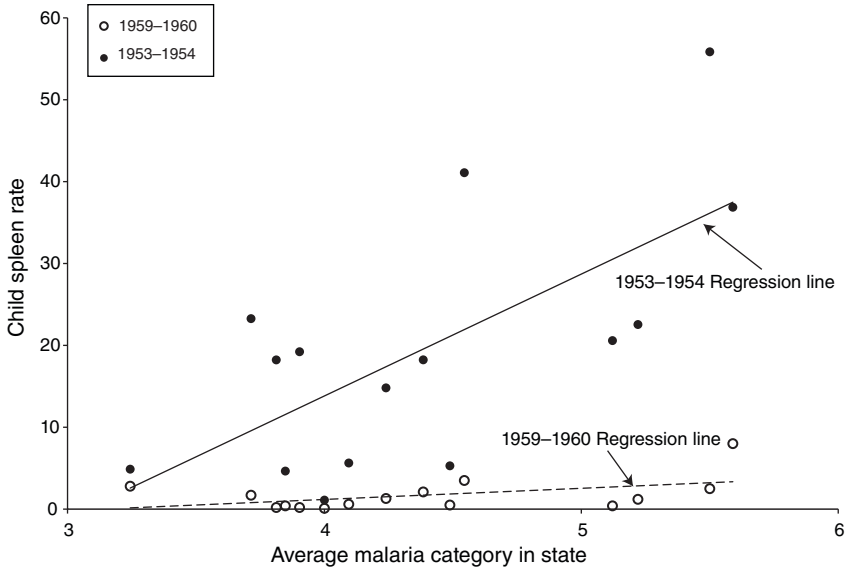

FIGURE 3. 1948 MALARIA ENDEMICITY AND CHILD SPLEEN RATES IN 15 STATES AND TERRITORIES

Notes: Sample includes all states and territories with child spleen rate data for both 1953–1954 and 1959–1960. See notes to Web Appendix Table 3 for details on the construction of state-level spleen rates for 1953–1954. We obtain the state-level average malaria category by averaging the categories of all GIS polygons within each state. The slopes of the 1953–1954 and 1959–1960 regression lines are 14.9 and 1.4, respectively.

Source: NMEP (1986)

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 3 plots the state-level child spleen rate against our map-based, state-level malaria index, constructed in the same way as the district-level index. In 1953–1954, just as the NMEP was starting its operations, the child spleen rate was strongly positively associated with the malaria index. By 1959–1960, 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. The district malaria index is also associated with known ecological determinants of malaria endemicity (precipitation, humidity, elevation, and temperature) in the expected directions (Web Appendix Table 4).

The 417 districts in our sample are grouped into 75 regions according to the NSS definition of regions, and these regions are grouped into 29 states. In the sample we use for the categorical analysis, we observe the following patterns. Of the 74 remaining regions, 51 have districts in only one category of malaria endemicity, 22 have districts in two malaria categories, and one region has districts belonging to all three malaria categories. Of the 28 remaining states, 13 have districts in only one malaria category, another 13 have districts in two malaria categories, and 2 have districts belonging to all three malaria categories.

B. *Outcomes*

We use data on human capital attainment and economic status from the forty-third 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. The NSS has four "thick" rounds that have the largest samples: namely, 1983, 1987, 1993, and 1999. We use the forty-third round (1987) 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.[13] Past research has used earnings or occupational wage data to estimate the productivity effects of childhood malaria exposure (e.g., Bleakley 2010), under the implicit assumption that employers pay workers their marginal product. However, only 7 percent of the NSS sample has a nonzero wage, and three-quarters of workers aged 20–60 (68 percent of men, 78 percent 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, measured by log household monthly per capita expenditure (measured in 1987 rupees). We trim the top and bottom 1 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. Web Appendix Figure 2 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. Men's labor force participation rates are well over 90 percent for most of this age interval, while women's labor force participation rates never rise above 50 percent. Therefore, we 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.[14] We also examine treatment effects at the household level, which we describe in more detail below. Schedule 1 (consumer expenditure schedule) of the NSS gives information on household consumption, and Schedule 10 (employment schedule) gives information on education.

Table 1 provides summary statistics for our sample, which omits individuals born during the eradication era (1953 to 1961). For literacy and primary school completion,

[13] 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.

[14] 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, Sharon Maccini and Dean Yang (2009) regress a household-level asset ownership index on individual-level exposure to early-life economic shocks.

Table 1—Summary Statistics

	Men		Women	
	Mean	SD	Mean	SD
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	4.98	(0.49)	4.96	(0.48)
Panel B. Malaria endemicity measures				
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				
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	77,015			

Notes: 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 nonmissing data on at least one outcome.

we analyze individuals between the ages of 15 and 75. In this sample, individuals born during the period 1912–1952 comprise the pre-eradication cohorts, whereas those born during the period 1962–1972 comprise the post-eradication cohorts. As previously discussed, to analyze expenditures, we restrict the sample to adults aged 20–60. Here, the pre-eradication era spans 1927–1952, and the post-eradication era spans 1962–1967.

IV. Results

A. *Differences-in-Differences Analysis*

We now examine the effects of the eradication program using the differences-in-differences specification described in Section II. 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

TABLE 2—CHILDHOOD MALARIA EXPOSURE AND HUMAN CAPITAL ATTAINMENT

	Literacy (ages 15–75)				Primary school (ages 15–75)			
Dependent variable:	(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	9.3 E-5	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
	(0.006)***	(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
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)	(0.049)***	(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

Notes: 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 the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

allow their influence to vary across cohorts from the pre-eradication and post-eradication eras.

Table 2 shows the results of our baseline specification for literacy and primary school completion, followed by several robustness checks. Panel A shows results for men, and panel B shows results for women. Each panel shows the treatment effects using 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 IVC.

We next examine the effects of the program on economic status, measured by per capita household expenditure, as described above (Table 3). Note that these coefficients should be interpreted as the effect of being treated on household per capita consumption for men versus women rather than the effect of treatment on male income and female income per se. Unlike the human capital results, these results indicate a positive effect of eradication per capita household consumption for treated men. 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 our 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 percent increase in per capita household expenditure. To put this magnitude in context, we can convert the malaria index into an approximate measure of the

TABLE 3—CHILDHOOD MALARIA EXPOSURE AND ADULT ECONOMIC STATUS

	Log per capita household expenditure (ages 20–60)			
Dependent variable:	(1)	(2)	(3)	(4)
Panel A. Households classified by male birth cohort				
A1. districts classified by modal malaria category (Omitted category: post × non-malarious)				
Post × potential epidemic	0.011	0.011	–0.054	0.037
	(0.015)	(0.024)	(0.032)*	(0.062)
Post × malarious	0.033	0.034	0.018	0.082
	(.014)**	(0.021)	(0.027)	(0.052)
F-test: equal treatment effects (*p*-value)	0.055	0.080	< 0.001	0.187
Observations	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
	(0.004)**	(0.005)**	(0.006)***	(0.011)
Observations	75,230	75,230	75,230	75,230
State × post fixed effects		X		
Region×post fixed effects			X	X
District-specific linear trends				X
Panel B: households classified by female birth cohort				
B1. Districts classified by modal malaria category (Omitted category: post × non-malarious)				
Post × potential epidemic	0.011	–0.006	–0.016	–0.014
	(0.015)	(0.028)	(0.039)	(0.077)
Post × malarious	–0.003	–0.014	–0.015	0.027
	(0.013)	(0.024)	(0.031)	(0.057)
F-test: equal treatment effects (*p*-value)	0.163	0.570	0.967	0.425
Observations	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
	(0.004)	(0.004)	(0.005)	(0.014)
Observations	75,212	75,212	75,212	75,212
State × post fixed effects		X		
Region × post fixed effects			X	X
District-specific linear trends				X

Notes: OLS coefficients, with standard errors in parentheses. The 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 in 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 the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

Table 4—Household-level Analysis of Malaria Exposure and Economic Status

Dependent variable:	Log per capita household expenditure
1. Districts classified by modal malaria category	
Potential epidemic × (# males in treatment cohorts)	0.040
	(0.018)**
Malarious × (# males in treatment cohorts)	0.055
	(0.018)**
Potential epidemic × (# females in treatment cohorts)	0.031
	(0.022)
Malarious × (# females in treatment cohorts)	0.018
	(0.021)
F-test: equal treatment effects (*p*-values)	
Males: potential epidemic vs. malarious	0.004
Females: potential epidemic vs. malarious	0.158
Potential epidemic: males vs. females	0.773
Malarious: males vs. females	0.113
Number of households	63,219
2. Districts classified by average malaria category	
Malaria index × (# males in treatment cohorts)	0.014
	(0.003)***
Malaria index × (# females in treatment cohorts)	0.002
	(0.003)
F-test: equal treatment effects (*p*-values)	0.022
Number of households	79,500

Notes: OLS coefficients, with heteroskedasticity-robust standard errors in parentheses. The regressions include the main effects of the number of males and females in treatment cohorts, district fixed effects, and demographic covariates. Demographic covariates include household size, membership in a scheduled caste, membership in a scheduled tribe, and household religion.

***Significant at the 1 percent level.
**Significant at the 5 percent level.
*Significant at the 10 percent level.

corresponding spleen rate using the slope of the 1953–1954 regression line in Figure 3. 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 for treated men. 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.

Notably, we do not observe significant effects of the program on per capita household expenditure for treated women (panel B). The coefficients on *post* × *malaria* index are significantly higher for men in all specifications other than the one with district linear trends. 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.[15]

Note that even if the improvements in household economic status 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 in our treatment group are, on average, married to men 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 household expenditure (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.[16] Only 41 percent of this group is married, and those that are married are likely to be a selected group.

The possibility remains that at least part of the apparent gender differential may be driven by differences in household composition between men and women. For example, given the marital age gap, men are more likely to be married to treated women than women are to be married to treated men. We explore this possibility by running a household-level regression of per capita household expenditure on pre-eradication prevalence interacted with the number of household men and women in treatment birth cohorts (1962–1967), controlling for the main effect of the number of men and women in treatment cohorts and household size (Table 4). The results imply that having more treated men in the household, controlling for the number of treated women, results in significantly higher per capita household consumption. This is true when using the categorical or index measure of malaria prevalence. Having more treated women in the household, controlling for the number of treated men, has, at most, a small, insignificant effect. The difference between the index coefficients for men and women is significant at the 5 percent level.

B. *Cohort Analysis*

In this section, we examine outcomes by birth cohort over time. Motivated by the differences-in-differences results, we focus on consumption effects for men. We run regression (2), using five-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.

Figure 4 shows the coefficients on *birth cohort* × *malaria* index and the coefficients on *birth cohort* × *potential epidemic* and *birth cohort* × *malarious*. We 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 rising sharply for those born after eradication.

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

[16] Note that in all the other specifications presented in the paper, in which our goal is to measure direct effects of treatment on women, treatment birth cohorts are 1962–1967. This specification is designed simply to test whether women benefit from eradication through the spousal income channel.

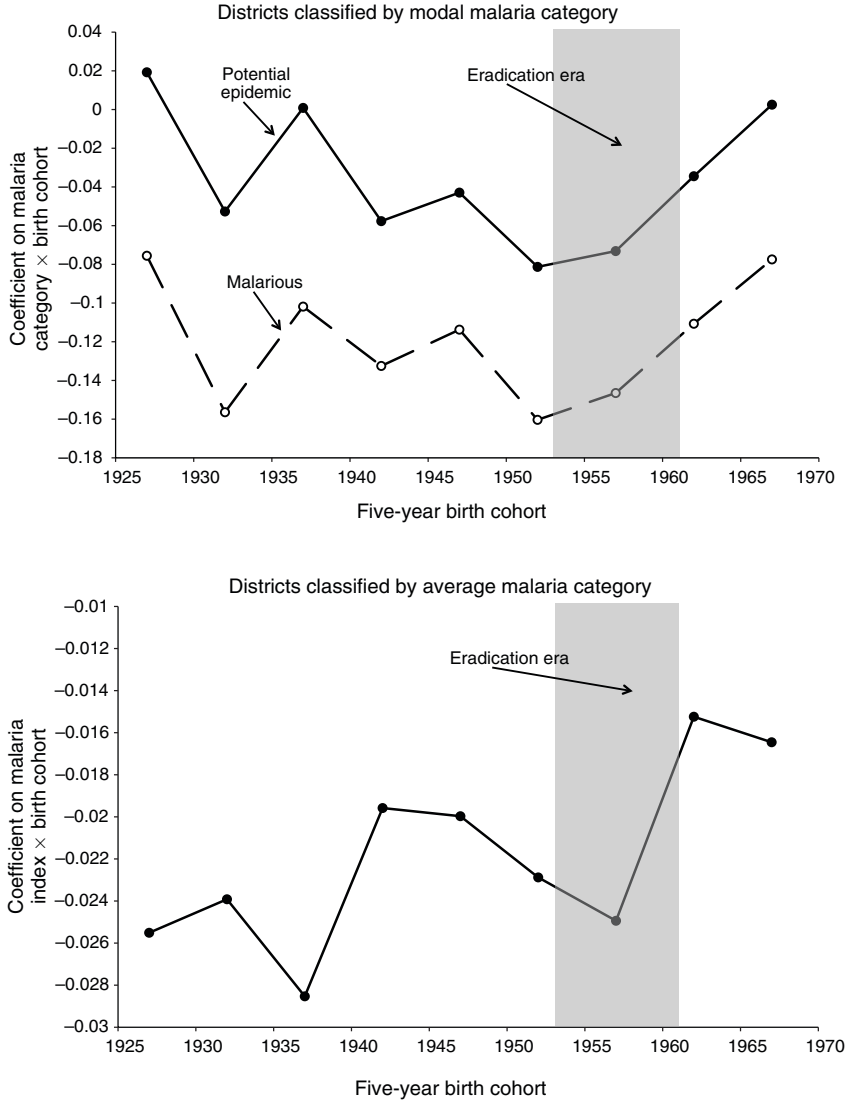

FIGURE 4. PRE-ERADICATION MALARIA ENDEMICITY AND ADULT ECONOMIC STATUS: COHORT-SPECIFIC RELATIONSHIPS
(*Households Classified by Male Birth Cohort*)

Notes: 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 five-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.

Our concerns that improvements for those affected by the program might reflect preexisting 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.

C. *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.[17] We therefore instrument for the map classifications using the ecological factors shown in Web Appendix Table 4.[18] Specifically, we use the interaction of the *Post* dummy with the district's average temperature, average elevation, average humidity, average precipitation, and squared terms in all four variables as our excluded instruments in the first-stage of our instrumental variables specifications. The second-stage equation is identical to equation (1).

Table 5 reports the results of the IV estimates for educational and income outcomes for 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 2, 3, and 4. 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 are now slightly larger in magnitude than the OLS estimates.

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* $\times$ *post* controls. When we use the three category measure in an otherwise identical instrumental variables setup, we obtain a nonmonotonic 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 nonmonotonicity 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

[17] 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 Thomas J. Kane, Cecilia Elena Rouse, and Douglas Staiger (1999).

[18] 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). Mark New et al. (2002) provide a detailed description of the dataset. We use GIS to overlay the ecology data with district boundaries.

Table 5—Instrumental Variables Estimates

	Men/male birth cohort			Women/female birth cohort		
Dependent variable:	(1)	(2)	(3)	(5)	(6)	(7)
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	0.009	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)
State × post fixed effects		X			X	
Region × post fixed effects			X			X

Notes: 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 2 and 3 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 Web Appendix Table 4 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 the 1 percent level.

**Significant at the 5 percent level.

*Significant at the 10 percent level.

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 indicate an annual death rate from malaria of only 0.2 percent (NMEP 1986). In regard to migration, as mentioned above, we unfortunately do not observe individuals' districts of birth. However, only 7.5 percent 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 for which 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 order to generate the observed results, targeting would have also had to be correlated with malaria prevalence within localized geographic areas. We 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).[19]

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 per capita household consumption for treated men 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 in urban areas (coef. = −0.014, SE = 0.014).

D. *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 they compare with existing findings on eradication programs in other countries (Bleakley 2010; Lucas 2010)?

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 to 0.035, implying that a move between these two percentiles increases per-capita expenditure by between 3 percent and 13 percent. These estimates are somewhat lower than Bleakley's (2010) estimates for male earnings in Latin America, which may be partially reconciled by the fact that our treated cohorts are relatively young. See Bleakley (2010) for further discussion. 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.

Another way to compare our results with other estimates is to rescale our coefficients to infer the effect of a 10 percentage point decline in malaria incidence, as in Lucas (2010). 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

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

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 3 on the correlation between the child spleen rate and the malaria index. Regression estimates using these data sources indicate that a 10 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 3). If we multiply our coefficients by the product of these two numbers, 1.93, the result tells the impact of a 10 percentage point decrease in incidence.

The rescaled OLS and IV point estimates indicate that a 10 percentage point decrease in incidence raises per-capita expenditure of between 1.5 and 6.8 percent. For women's human capital, the OLS results do not imply positive treatment effects. However, the rescaled IV estimates imply that a 10 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 4) yielding estimates at the bottom of this range. These estimates are slightly higher than both Bleakley (2010) and Lucas (2010), who find that a change in incidence of the same magnitude raises literacy 0.08 to 2 percentage points.

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 an offsetting fertility response, eradication of the more fatal *P. falciparum* in sub-Saharan Africa is more likely to result in population growth. As a result, effects on cohorts exposed to eradication in childhood may differ in general equilibrium.

V. 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 males exposed to malaria eradication in early childhood enjoyed higher per capita household consumption as adults. We do not observe robust treatment effects for education, which may reflect the trade-off between schooling and labor emphasized by Bleakley (2010). The results generally imply larger treatment effects on consumption for men than for women. One possibility is that the observed effects for men are driven by increased productivity in the labor market, where male participation rates are much higher than female.

The estimated 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. Specifications that include district-specific time trends yield positive but imprecise estimates.

Our results and those in the accompanying articles provide support for the belief that improvements in health and in the disease environment can have a causal effect

on economic well-being. This effect is large enough to conclude that investments in malaria control technologies such as insecticide-treated nets, which cost less than $10 and have been proven medically efficacious, likely have very high returns even without taking into account the large direct utility benefits of better health. However, the estimated effects are not large enough to provide support for the view that malaria control is a major driver of economic growth.

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