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Disease eradication, infant mortality and fertility response :Evidence from malaria eradication in India

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Abstract

Disease environment and demographic change plays a critical role in determining the size and quality of human capital that drives the growth path of an economy. While broad patterns of demographic transition are understood there is a mixed evidence on the role of disease eradication in expediting demographic change. Using the massive malaria eradication program in India during the 1950's as a natural experiment, we examine the effect of disease environment on infant mortality and fertility response at household level. We harmonize a rich database on malaria endemicity with fertility histories of women to exploit the cohort level variation in exposure to the program. We find that the program leads to a significant decline in infant and neonatal mortality and leads to a significant increase in probability of birth in high malaria-endemic regions. We confirm the mechanism of fall in mother's age at first birth in post eradication period drives the fertility response.

JEL codes: I12 I15 I18 J13

Keywords: Malaria, Selection Bias, Disease eradication, Infant Mortality, Fertility

1 Introduction

There is mixed evidence on the role of disease eradication in harnessing demographic transition which has a significant role in shaping the human capital and growth trajectory of a country. While a set of studies highlight negative relation between disease environment and growth (Bloom et al. (2019),Ashraf et al. (2008), Gallup and Sachs (2001)), an improvement in population health with decline in mortality can slow down the rate of growth (Acemoglu and Johnson (2007), Young (2005)). Existing evidence on the effects of malaria eradication

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on human capital and other economic outcomes is also mixed, with the effects ranging from positive to zero (Apouey et al. (2018),Barofsky et al. (2015),Venkataramani (2012), Bleakley (2010), Cutler et al. (2010), Lucas (2010)). In addition to the economic outcomes, disease eradication can also affect demographic outcomes such as infant mortality and fertility(Lucas (2013)). This can make the estimation of human capital gains to disease eradication difficult if relatively weak individuals survive after the eradication. Theoretically the direction in which fertility would move is not apparent, in response to an improvement in the disease environment. On one hand decrease in malaria incidence should improve maternal health leading to reduced miscarriages, higher sexual activity and female fecundity, which results in increased fertility. On the other hand,reduction in malaria deaths among children can lead to lower demand for children through the channel of replacement effect and precautionary demand as risk from disease environment goes down(Kalemli-Ozcan (2002),Galor and Weil (1999), Becker and Barro (1988)). This makes it an empirical question.

In this paper we ask how does child mortality and fertility outcomes respond to an exogenous change in the disease environment in the context of a developing country. In particular we examine effects of the massive public health intervention in India during the 1950's which led to a sharp drop in malaria. We exploit the quasi-experimental setup offered by the malaria eradication program in India to look at its impacts on infant mortality and fertility. Using cohort level variation in exposure to the malaria eradication program during 1950's we test whether the districts that were most burdened by malaria in the pre-eradication era experienced the largest changes in fertility and child health outcomes. Our main estimation strategy aligns with the difference in difference approach as used in Bleakley (2010), Cutler et al. (2010), Lucas (2010).

In contrast to the existing literature which largely abstracts from the issue of selection due to infant mortality, we bring out the effects of the eradication campaign on neonatal and infant mortality and also examine the fertility response to these health gains. Using detailed fertility histories from National Family Health Survey of India (NFHS-I) conducted in 1992-1993 we find a significant decline in both infant and neonatal mortality in post eradication decade for high malaria endemic districts. Secondly, we find that the probability of birth increases in the post eradication period for these districts. The results hold true only in the

rural areas where both the disease burden and the program were mostly focused. Further, we find the mechanism of a fall in mother's age at first birth drives the female fertility response in the post eradication period for high malaria-endemic districts.

Our study has several contributions. The existing micro economic literature on malaria eradication, which has mostly focused on the causal impacts of early exposure to malaria on later-life socio-economic outcomes, has given scant attention to selection bias arising out of neonatal and infant mortality from malaria infections citing rarity of such events [1].To the best of our knowledge this is one of the first papers to challenge this conventional wisdom and provide the reduced form estimates of the impact of malaria eradication on both neonatal and infant mortality, which so far has been largely ignored in the literature[2] Additionally, our findings have broader normative implications in improving the evaluation framework for calculation of "economic gains" of eradication on human capital returns (viz. education and labour market outcomes) where mortality bias plays a critical role. This mechanism can possibly reconcile the apparent puzzle of a weak effect of disease eradication on growth and human capital outcomes such as found in (Acemoglu and Johnson (2007)) and (Cutler et al. (2010)).The paper most closely related to our work is Lucas (2013) which finds the effect of malaria eradication resulting in increase in fertility in the context of Sri Lanka. However the paper found an imprecise effect of malaria eradication on infant mortality. Together with Lucas (2013) our finding pushes the existing literature forward in recognizing the impact of eradication on demographic factors, viz. neonatal and infant mortality, along with fertility in the calculation of returns to malaria eradication programs. Furthermore, this informs the literature on the relationship between mortality decline on fertility outcomes and enriches the quality-quantity framework of fertility choice behaviour.

The rest of the paper is organised as follows. In section 2 we describe the conceptual framework and the relevant literature. Section 3 includes a brief description of the eradication program and section 4 discusses the data used for analysis.In section 5 we describe the estimation strategy used in the paper.We explain the results in section 6.Section 8 concludes.

[1]This is especially true for the set of studies carried in South Asia and America with milder forms of malaria as compared to sub-saharan Africa

[2]A recent set of working papers (Wilde et al. (2019); Cogneau and Rossi (2019)) look at the impact of malaria intervention on infant mortality in the context of Africa.

2 Conceptual Framework

Theoretical models emphasize the link between health and fertility in mainly two ways. Increases in life expectancy can potentially reduce the precautionary demand for children due to reduction in survival uncertainty thereby lowering fertility. Schultz (1997) analyzes data on mortality and fertility rates for 70 developing countries (from 1972-1989) and finds that for every percentage point reduction in the fraction of children who die before five years, women had 0.25 fewer births. Health improvements can also translate into better labour market outcomes. Thus parents can increase educational investment and substitute the quantity with better quality children. Bhalotra et al. (2014) uses the introduction of antibiotics in America that lowered both infant and maternal mortality to look at fertility response. The paper finds that the fall in infant mortality and improvements in child health reduced fertility in the white population.

Importantly, this linkage between mortality decline and fertility will be perhaps most strong when couples are consciously choosing the size of their family with access to contraceptives. There seems to be little use of contraceptive practices during the implementation of the malaria eradication program in India (Freymann (1963)), which we consider in this paper. On the other hand, disease prevalence might imply lower fertility and thus eradication might lead to an increase in fertility. Young (2005) simulates the impact of AIDS epidemic on future living standards in South Africa that suggests the economic gains from lower fertility during widespread community infection dominates the loss in human capital from reduced educational attainment.

Malaria eradication can also lead to higher fertility by removing the previous biological constraint in the form of reduction in in-utero complications and still births and reduced maternal mortality. Lucas (2013) argues the direct effect of malaria on fecundity is positive (increased probability of still births and spontaneous abortion, reduced coital frequency and maternal health risk) and this health burden is higher for first time mothers. As a result fewer first pregnancies result in live births in a high disease environment. Thus, reduction in malaria incidence can result in a younger maternal age at first birth if probability of survival of first born increases post eradication. Additionally, there may be an unmet demand for

children in the presence of a biological constraint. Apart from these two channels, disease eradication can also have a bearing on work capacity of parents thus impacting household income. This may also have an independent effect on fertility preference. This channel is perhaps less important as most malaria related deaths are concentrated at young ages, and kills people before they have begun working (Depetris-Chauvin and Weil (2017)). The above discussion seems to imply that, if the eradication results in reduced neonatal and infant mortality, a priori it is difficult to predict the direction of fertility change.

There is also a big literature on the long term effects of malaria eradication on human capital outcomes and economic growth. Bleakley (2010) finds that early exposure to malaria lowers labour productivity and income using information on malaria eradication campaigns in US, Brazil, Colombia and Mexico. Lucas (2010) finds malaria eradication increased years of educational attainment and literacy for women in Sri Lanka and Paraguay. In Indian context, Cutler et al. (2010) examines the effects of early exposure to malaria 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 1950. They use the digitized version of 1948 government map of India that classifies areas under six different categories of malaria endemicity to compare gains for cohorts before and after the program in areas with varying pre-eradication prevalence. They find modest albeit imprecise increases in economic status for men and no gains on educational attainment for men and mixed results for women.

Important to note, if malaria eradication resulted in bringing down infant mortality rates and increased female fecundity it might result in attenuation bias in the estimates of any "economic gains". Harmonising detailed fertility records of women with the digitized malaria endemicity record data from Cutler et al. (2010) we bring out the first reduced form estimates of the eradication program on infant mortality and fertility. To the best of our knowledge we are the first to present estimates of malaria eradication campaign on neonatal, infant mortality and fertility in the Indian context. The following section discusses the eradication program and lays out the econometric framework for evaluation.

3 The Malaria Eradication Program

Malaria is considered one of the important causes of infant, child and adult mortality in India (Kumar et al. (2007)). Before independence the estimated death toll caused by malaria was 1 million during normal years and 2 million during epidemic years. There was a dramatic decline in malaria related mortality after the National Malaria Eradication Program was launched in 1958. The timing of the intervention is plausibly exogenous, primarily driven by the advent of DDT in mid 1940's. The national Planning Commission endorsed the development of a comprehensive nation wide program,the National Malaria Control Program (NCMP) in 1953. The program was reformulated in 1958 as the National Malaria Eradication Program with the goal of completely eradicating malaria from the nation, and by 1960-61, the entire country was brought under the program. Figure 1 depicts the dramatic success of the eradication campaign which resulted in a sharp drop in malaria case rates.

Infants and children died mostly from the indirect effects of malaria in the form of premature birth and malnutrition and in later childhood died from direct or secondary infections (Sinton et al. (1936)). Steketee et al. (2001) mentions a range of adverse effects of malaria infection faced by pregnant women in malarious areas including maternal anemia, placental accumulation of parasites, low birth weight from prematurity and intrauterine growth retardation, fetal parasite exposure and congenital infection, and infant mortality linked to low birth weight. Overall, there is a disproportional cost of malaria infection on children and pregnant women including spontaneous abortions, still births,low birth weight in neonates and maternal mortality.

Cutler et al. (2010) mentions that post malaria-eradication data in India suggests about 30 per cent of malaria cases were due to P.falciparum, the parasite associated with most of malaria related fatalities (NMEP (1996)).Importantly for our identification strategy, there was a dramatic decline in malaria incidence following the eradication drive. There was a major exogenous shift shows in the epidemiological environment with reduction in spleen rates by 91 percent, child parasite rates by 79 percent and 63 percent reduction in infant parasite rate. The report (GOI (1996)) argues that about 45 million malaria cases were less in 1957-58 as compared to 1953. The proportional case rate (percentage of cases of clinical

malaria to total number of cases due to all diseases) drastically came down from 10.8 percent in 1953-54 to 2.4 percent in 1959-60. The areas with highest incidence of malaria were given priority under the program.

The primary activities under the control program were to spray DDT on wall surfaces and roofs of all houses and cattle sheds twice a year. The spraying time was adjusted according to the local transmission season and the interval between two sprays was two and a half months. During the first year of the operations, 75 malaria control units were allotted to various states, priority being given to areas where the incidence of malaria was of the highest order. This involved intensified spray operations in every roofed structure, twice in endemic areas throughout the country for a varying number of years. Once a district got the program it continued to remain in the program in subsequent years Cutler et al. (2010). Urban areas were largely free from malaria in the pre-eradication era and we use it for our falsification tests. The low prevalence of malaria sustained throughout 1960's until a resurgence around 1975(with a much lower prevalence rate).

4 Data

The micro-data used in this survey are derived from the first round of the National Family Health Survey of India (NFHS-I) conducted in 1992-1993. This data-set contains complete fertility histories for ever-married women aged 13-49 years in 1992-93, including the retrospective data on the year of birth and time and incidence of child deaths. Unfortunately, the data does not contain anthropometric outcomes for all children due to which we are limited in our scope to look at objective health outcomes beyond mortality. The data has information the district of residence of the household during the time of the survey. While it is possible that the district of birth of the child might not be same as the district of the residence of the household in 1992-93, this is unlikely to be an issue in India. Spatial mobility is low in India(Munshi and Rosenzweig (2006); Deshingkar and Anderson (2004); Cutler et al. (2010)) and this is particularly true for women after marriage. Migration at the time of marriage is the main reason for geographical movement among women in India (Deshingkar and Akter (2009),Rosenzweig and Stark (1989)). We have used this data-set to construct indicators of fertility at the level of individual woman and mortality at the level of

individual child. The estimation sample contains children born to more than 66,000 mothers born over the period 1954-1991.

Data on malaria endemicity comes from replication data set of Cutler et al. (2010).The pre-eradication malaria map classifies areas into six endemicity categories[3] Their paper digitizes the endemicity map which subdivided districts into polygons of approximately equal size with some districts having more than one possible classification. They categorize each district by its modal polygon malaria category and drop the districts that do not have unique mode. We classify the districts according to their maximum value of malaria prevalence and conduct robustness tests using alternative measures of prevalence such as maximum modal value. We argue that this is in line with the roll out feature of the intervention which gave a higher focus on areas with maximum endemicity. A report from the planning commission indicates 230 units were planned to operate in hyper-meso-endemic areas in 1958-59 and 160 units in hypo-endemic areas from 1959-60.

Tables 1, 2 and 3 report the summary statistics for variables used in the child health outcomes regressions, fertility regressions and the age at first birth regressions respectively. It can be seen that from Table 1 that both neonatal and infant mortality is quite high in India during the sample period. Neonatal mortality was 69 per 1000 and infant mortality was 111 per 1000. Mortality outcomes are worse in the rural areas than in the urban areas. Fertility outcomes, measured by probability of birth in a year is also higher in rural areas. In order to identify the mechanisms, we find the effects of the malaria eradication program on the mother's age of first birth. The summary statistics of the variables used in these regressions are presented in Table 3. The mean age at first birth in India was 19 years during the sample period and is lower in rural areas compared to the urban areas.

[3]This map classifies areas based on child spleen rates and climatic factors into the following categories (1) areas above 5000 feet; non malarious, (2) known healthy plain areas; spleen rate under 10 percent (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.

5 Estimation Strategy

We use a difference in differences estimation strategy closely in line with (Cutler et al. (2010)) to look at the effect of malaria eradication on child mortality and women fertility where we exploit the geographic variation in the prevalence of malaria in the pre-eradication period. One of the key challenges in identifying the effect of malaria on child health and fertility is the potential correlation between unobservable regional characteristics and malaria levels. Improvement in income can lead to lower incidence of malaria as well as affect child survival and fertility. To overcome these endogeneity concerns, we use the exogenous change in malaria incidence from the eradication drive to look at its causal impacts on child survival and women's fertility. We argue that the areas burdened with high malaria-endemicity are likely to see the highest declines in child mortality[4]. Thus we examine the causal effect of the program on infant and neonatal mortality using detailed information on child deaths constructed from fertility histories of women from NFHS. Since urban areas were relatively unaffected by this eradication program where malaria was not much a concern in this time period we mainly focus on the rural sample and use urban areas for falsification tests.

The key assumption in the identification strategy is that in the absence of the eradication program there would be no changes in infant /child mortality and fertility that is correlated with the pre-eradication malaria intensity. The inclusion of district fixed effects and time fixed effects account for time invariant differences between regions and secular improvements in disease environment over time. A potential concern could be be that regions with high malaria endemicity may have attracted other public health interventions which affects child health and fertility independently. We address this concern by including district specific time trends over and above district and year fixed effects. The district specific time-trends also control for the possibility of mean reversion or convergence of outcomes for high malarious regions with that of less malarious areas in the absence of the eradication drive.

The main estimating equation for estimating the effect on infant and neonatal mortality outcomes is

[4]For child health outcomes, ideally we would like to see the effect of the intervention on both mortality and morbidity outcomes. However the data does not contain the anthropometric outcomes for*all* children due to which we are limited in our scope to look at objective health outcomes beyond mortality

$$Y_{idt} = \alpha + \beta post_t \times BaseMalaria_d + \gamma X_{idt} + \delta_d + \tau_t + \delta_d T + \epsilon_{idt} \quad (1)$$

where Y_{idt} is a dummy variable equal to 1 if the child i born in district d and year t died by the age of 1 month (neonatal) and 12 months[5] (Infant mortality). $post_t$ is a dummy variable equal to 1 if t is a post eradication period. The eradication program covered the whole of India by 1960-61. The graph (Trends of Malaria 1947-2007, NMEP) shows the dramatic fall in malaria cases post eradication. For mortality outcomes we compare children born after 1961 with children born before 1960 in order to allow full exposure. Children born in 1960 or 1961 are likely to have been conceived in 1959, 1960 or 1961. Since infant and in particular neonatal mortality depends on prenatal care, we only consider children born after 1961 as children born in the post period. We also exclude children born in 1960 and 1961 since they do not have full exposure to either the pre or the post period.

FollowingCutler et al. (2010) we exploit the geographic variation of the disease prevalence prior to the program to study the effect of this program on mortality and fertility outcomes. $BaseMalaria_d$ is the maximum endemicity category for a district.

X_{idt} denotes the various controls used including dummies for the sex of the child, dummies for the order of birth, age of the mother at birth, parental education and dummies for membership in Scheduled Caste, Scheduled Tribe and a Muslim dummy. δ_d, and τ_t denote district and time respectively. $\delta_d T$ denotes district specific linear time trends.

The estimating equation for fertility is similar to equation 1

$$Y_{idt} = \alpha + \beta post_t \times BaseMalaria_d + \gamma X_{idt} + \delta_d + \tau_t + \delta_d T + \epsilon_{idt} \quad (2)$$

where Y_{idt} is a dummy variable indicating whether a woman i in district d gave birth in the year t. The controls include her age in year t, education, husband's education, existing number of children, and dummies for membership in Scheduled Caste, Scheduled Tribe and religion in addition to district and time fixed effects and district specific linear time trends. To detect the fertility response and change in the mother's age at first pregnancy, we allow

[5]There can be concerns of age heaping which comes from the fact that ages are often rounded off to the nearest 6 months (Bhalotra (2010)). Thus we have also estimated the infant mortality results excluding the 12th month. The results are presented in Appendix Table 1. We see that the results are unchanged.

for a lag of 5 years and compare child births in and after 1965 with births before 1960. If the change is driven by the biological channel, we expect that disease eradication should affect fertility around the same time as infant mortality. We verify that our results are robust to the inclusion of shorter lags[6]. However, in our main specification, we work with a lag of 5 years to distinguish between the biological channel and the quality-quantity trade-off channel. It is usually stressed in literature that the quality quantity channel comes into effect with a lag (Kalemli-Ozcan (2002),Palloni and Rafalimanana (1999)).

6 Results

6.1 Effect on Child Health Outcomes

Table 4 presents the results on the impact of the malaria eradication program on the mortality outcomes separately for rural and urban areas. Since the program primarily targeted the rural areas, we expect the effect to be more pronounced in rural areas. Columns 1 and 2 report the results for neonatal mortality and infant mortality in rural areas. Our results suggest that, in response to a one unit increase in malaria index, both infant and neonatal mortality are lower by about 4.5 percentage points in the post period compared to the pre-period in rural areas. The results imply that complete malaria eradication for the most malarious districts will reduce infant and neonatal mortality by 27 percentage points. Columns 3 and 4 present the results for neonatal and infant mortality in the urban areas. The magnitude of the effect is much smaller and the estimate is statistically insignificant for the urban sample.

One problem with our specification is that the post period is relatively long compared to the pre-period. Even though we include district specific time trends, one might worry that our results are driven by the post period observations and not by the program itself. In order to ensure that this is not the case, we restrict our sample with births till 1975 and estimate the effects. The results are reported in Table 5. The results obtained from this reduced sample is same in sign, magnitude and significance as our baseline results.

[6]The results are presented in Appendix Table 2. Column 1 presents the results excluding children born in 1960 and 1961 similar to the mortality specifications. Column 2 excludes children born between 1960 and 1962 and Column 3 excludes children born between 1960 and 1963.

Mothers less than 30 years of age in 1992-93 (the survey year) have no exposure to the pre-malaria period and hence ideally their exclusion should not affect our results. We next test whether this is indeed the case. In Table 6 we exclude women less than 30 years of age and estimate the regressions again. We thereby confirm that our results are not affected by the exclusion of these women.

In Table 7, we impose both the restrictions of limiting our sample to births till 1975 and considering women aged 30 and above in 1992-93. Again our results are similar to the baseline estimates.

Next we do a placebo test, assuming that the eradication program took place in the 1970s and treat the period after 1980 as the post period.We have already verified that urban areas did not experience any change in terms of child heath outcomes. We drop observations with birth before 1970. In the baseline model, we dropped children born in 1960 and 1961 and so in the placebo test we drop children born in 1980 and 1981. Using this specification, we estimate the effect of the malaria eradication program on infant and neonatal mortality. Table 8 presents the results. All the estimates turn out to be small in magnitude and statistically insignificant. In table 9, we do the same robustness check but we restrict our sample to women who are above 30 in 1992-93. We again find that the effects are statistically insignificant.

We further verify that the reduction in neonatal and infant mortality are not due to any change in the gender composition of children post eradication to rule out any gender discrimination channel for mortality reduction. Although the period under consideration had little access to sex detection techniques we check to see whether the program had any impacts on the sex ratio at birth. To estimate the effects of the program on sex ratio, we estimate an equation similar to equation 1, with the dependent variable being a dummy variable equal to 1 if the child born is a female. Table 10 presents the results. As expected, the results are all statistically insignificant. In Table 11, we do the same robustness checks restricting the sample to post 1970, assuming that the eradication took place in the 1970s. Columns 1 and 2 estimate the effects for all children and columns 3 and 4 restrict the analysis to children born to women aged 30 and above in 1992-93. We can see that the estimated coefficients are all statistically insignificant.

6.2 Effect on Fertility

Table 12 presents our results on the effect of the malaria eradication program on fertility or the probability of birth. Panel A reports the results for rural areas and Panel B reports the results for urban areas. Column 1 presents the results for the entire sample. Column 2 shows the results if the sample is restricted to the pre-1975 period. Column 3 restricts the sample to women aged above 30 in 1992-93. Column 4 adds both the restrictions in columns 2 and 3. We see that the probability of birth increases in the post eradication period for high malaria endemic districts. A unit increase in malaria index results in a increase of probability of births by 0.55 to 1 percentage points. This means that elimination of malaria in the most malarious regions will increase fertility by 3-6 percentage points. The magnitude of the effect is modest compared to the effects found in Lucas (2013), who find that complete elimination of malaria in the most malarious regions will increase fertility by 11 percentage points. The results again hold true only in the rural areas. In urban areas the effects are statistically insignificant.

We do the placebo test for the probability of birth in Table 13. Assuming that the eradication program took place in the 1970's, we compare the probability of birth before 1980 with the probability of birth after 1985. We again drop observations with birth years before 1970. Columns 1 and 2 estimates the effects for all women and columns 3 and 4 restrict the analysis to women aged 30 and above in 1992-93. We can see that the estimated coefficients are all statistically insignificant.

7 Mechanism: Effect on Age at First Birth

Our results suggest that fertility increased even when child health outcomes improved. The increase in fertility despite an improvement in child health indicates that the result might be driven by the rise in female fecundity following the eradication program. In order to identify the mechanisms, we try to find the effect of the eradication program on the childbearing age of women. In particular, we estimate the effect of the program on the age of women at the time of their first birth. The specification is the same and is given by

$$Y_{idt} = \alpha + \beta post_t \times BaseMalaria_d + \gamma X_{idt} + \delta_d + \tau_t + \delta_d T + \epsilon_{idt} \quad (3)$$

where Y_{idt} is the age at first birth of woman i in district d whose year of first birth is year t. X_{idt} is the set controls including her education, husband's education and dummies for membership in Scheduled Caste, Scheduled Tribe and religion in addition to district and year of first birth fixed effects and district specific linear time trends. $post_t$ is a dummy variable equal to 1 if the year of first birth was after 1961.

Table 14 presents our results where the dependent variable is the age at first birth. Panel A presents the results for the rural areas while panel B presents the results for the urban areas. Column 1 presents the results for the entire sample, column 2 restricts the sample to women with year of first birth before 1975, column 3 estimates the results for women who were more than 30 years of age in 1992-93. Column 4 introduces both the restrictions. We find that the age at first birth for the mother falls in the post eradication period. The effect, once again, exists consistently only in the rural India.

In order to test the robustness of the results, we again do the same placebo test for the age at first birth. Table 15 presents the results. We can see that the estimated coefficients are all statistically insignificant.

8 Conclusion and Discussion

The existing literature is divided on the role of disease eradication in expediting demographic transition that has important bearing on size and quality of human capital and labour productivity. This is true with regard to malaria eradication in particular, where impacts on human capital range from positive to null effects. Further, scant attention has been given in the literature on the impact of malaria eradication on infant mortality and fertility, particularly in settings outside Africa, where malaria related infant deaths were thought to be negligible. We present one of the first reduced form estimates of a hugely successful malaria eradication drive in India on neonatal and infant mortality and further check fertility response at the household level. We find the eradication program led to a significant decrease in infant and neonatal mortality by around 4 percentage points. The results imply that

complete malaria eradication for the most malarious districts would have reduced infant and neonatal mortality by 27 percentage points.

Important to note, that positive impact on infant survival is net of any potential negative effect of DDT on infant mortality. Interestingly, we find the eradication resulted in significant increase in probability of birth and a fall in the maternal age at first birth. We estimate that for the most malarious regions, the probability of birth in a certain year goes up by 3-6 percentage points along with reduction in mother's age at first birth by a quarter of an year. Our results on fertility align with the findings from Lucas (2013) which documents an increase in fertility in the post eradication in Sri Lanka. However, in contrast to their paper we find a significant negative impact of malaria eradication campaign on infant and neo-natal mortality that is robust to various specifications.

Evidence from a recent set of papers has examined cyclicality in infant mortality (Bhalotra (2010)) and fertility (Chatterjee and Vogl (2018)). Bhalotra (2010) documents that infant mortality in rural India is counter cyclical despite the fact that relatively high-risk women aver birth or suffer fetal loss in recessions. A potential concern in our estimates could be selection of mothers, if relatively poorer or weaker mothers are able to survive and give births post intervention. In that case, the improvements in child health is net of any negative effect from selection of mothers post intervention, in which case it is a possible lower bound of the actual improvements in child health. We further argue that the potential concern of a positive selection of households in accessing the benefits of eradication is limited in our setting, by the sheer nature of this intervention, which involved macro scale spraying of DDT in household dwellings and cattle sheds in most malarious regions.

(Chatterjee and Vogl (2018) which finds fertility is pro cyclical in the short run and declines with long run economic growth raise important questions about mechanisms and highlight how the underlying source of that growth may influence the development trajectory. Acemoglu and Johnson (2007) argue that increasing life expectancy raises population growth, that reduces per capita capital and slows down income growth. This is empirically supported using the global epidemiological revolution as an instrument for life expectancy. However, Cervellati and Sunde (2011) argue that results from Acemoglu and Johnson (2007) might be relevant for less-developed countries that have not yet undergone the demographic

transition. Specifically, they find the effect of life expectancy on growth is positive for post–demographic transition countries and negative but insignificant for pre–demographic transition countries. On the economic return on health Bloom et al. (2019) mentions that typically the micro-based approach tends to find smaller effects than the macro-based approach in the economics literature. Simulation exercise in Ashraf et al. (2008) finds a positive impact of health improvement on growth and highlights that the mismatch between their results from simulation with that of Acemoglu and Johnson (2007) can perhaps be explained by the increase in fertility with health improvement.

The findings from our paper complements the strand of literature on development gains from health innovations. Our findings highlight the selection issue of disease induced infant mortality which has largely been ignored in the literature. We provide suggestive evidence on the mechanism behind fertility rise and highlight to what extent factors beyond volition or conscious fertility choice,like fecundity or in-utero survival, might also play a role in this debate, especially relevant in the context of developing countries that has not surpassed demographic transition.Together with Lucas (2013) our findings extend the literature on understanding the role of disease eradication on economic growth and demographic transition. We argue that recognising the impacts of eradication on demographic outcomes has important bearing in understanding the returns to human capital. The increase in number of dependents, reduction in mother's age are all suggestive of weaker health capital in the cohort of children who are born in post eradication period. This has important policy insights particularly for developing countries that are transitioning through demographic transition. Any health innovation or intervention that leads to an improvement in reducing disease burden may result in increased fertility despite an improvement in child health. While it may take some time for these gains to show up as measured by"economic growth" in the short run, it may have first order implications on the desired family size through replacement effect and precautionary channel, particularly with access to modern contraceptives and economic development. This could help in moving parents along the quality-quantity frontier which has important consequences for investments in human capital and long run growth. We are however limited in our scope to examine the subsequent impact on child level investments due to data limitations in our set up. We believe this is an important area for future research.

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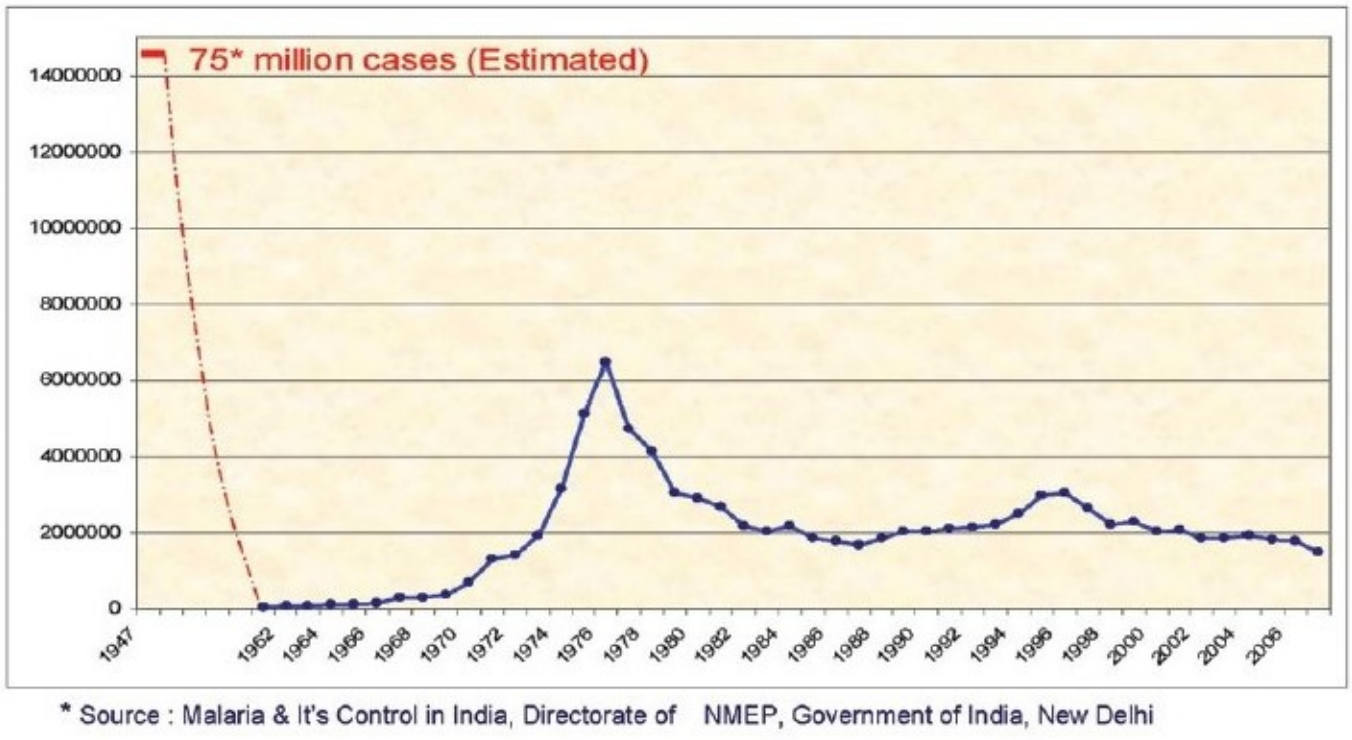
Figure 1: Malaria Eradication Program



Notes: The graph presents the dramatic success of the eradication campaign which resulted in a sharp drop in the number of malaria cases post 1960. The vertical axis plots the estimated case rates of malaria and the horizontal axis plots the calendar years. Source: (Narain et al., 2011)

Table 1: Summary statistics: Mortality Outcomes

All			
Neonatal Mortality	0.069	0.253	214482
Infant Mortality	0.111	0.314	214482
Maximum Malaria Endemicity	4.962	1.1	214482
Proportion of Females	0.483	0.5	214482
Proportion of Birth Order 1	0.283	0.45	214482
Proportion of Birth Order 2	0.237	0.425	214482
Proportion of Birth Order 3	0.179	0.383	214482
Proportion of Birth Order 4	0.122	0.327	214482
Proportion of Birth Order 5	0.078	0.268	214482
Proportion of Birth Order 6	0.047	0.211	214482
Proportion of Birth Order 7	0.027	0.163	214482
Proportion of Birth Order 8	0.015	0.121	214482
Proportion of Birth Order 9	0.007	0.086	214482
Proportion of Birth Order 10	0.006	0.075	214482
Mother's Age at Birth	23.233	5.326	214482
Mother's Years of Schooling	2.041	3.606	214482
Father's Years of Schooling	4.8	4.834	214482
Muslim Household	0.138	0.345	214482
Scheduled Caste Household	0.132	0.338	214482
Scheduled Tribe Household	0.085	0.279	214482
Rural			
Neonatal Mortality	0.075	0.263	160433
Infant Mortality	0.121	0.326	160433
Maximum Malaria Endemicity	4.997	1.061	160433
Proportion of Females	0.482	0.5	160433
Proportion of Birth Order 1	0.274	0.446	160433
Proportion of Birth Order 2	0.231	0.421	160433
Proportion of Birth Order 3	0.179	0.383	160433
Proportion of Birth Order 4	0.125	0.33	160433
Proportion of Birth Order 5	0.082	0.274	160433
Proportion of Birth Order 6	0.05	0.218	160433
Proportion of Birth Order 7	0.029	0.169	160433
Proportion of Birth Order 8	0.016	0.126	160433
Proportion of Birth Order 9	0.008	0.089	160433
Proportion of Birth Order 10	0.006	0.077	160433
Mother's Age at Birth	23.201	5.437	160433
Mother's Years of Schooling	1.338	2.815	160433
Father's Years of Schooling	4.06	4.465	160433
Muslim Household	0.118	0.322	160433
Scheduled Caste Household	0.14	0.347	160433

Table 1 Continued: Summary statistics: Mortality Outcomes

Scheduled Tribe Household	0.102	0.302	160433
Urban			
Neonatal Mortality	0.049	0.216	54049
Infant Mortality	0.078	0.268	54049
Maximum Malaria Endemicity	4.847	1.212	54049
Proportion of Females	0.484	0.5	54049
Proportion of Birth Order 1	0.311	0.463	54049
Proportion of Birth Order 2	0.257	0.437	54049
Proportion of Birth Order 3	0.179	0.383	54049
Proportion of Birth Order 4	0.111	0.315	54049
Proportion of Birth Order 5	0.065	0.247	54049
Proportion of Birth Order 6	0.036	0.186	54049
Proportion of Birth Order 7	0.02	0.141	54049
Proportion of Birth Order 8	0.01	0.102	54049
Proportion of Birth Order 9	0.005	0.073	54049
Proportion of Birth Order 10	0.004	0.066	54049
Mother's Age at Birth	23.339	4.94	54049
Mother's Years of Schooling	4.378	4.774	54049
Father's Years of Schooling	7.263	5.186	54049
Muslim Household	0.205	0.404	54049
Scheduled Caste Household	0.104	0.306	54049
Scheduled Tribe Household	0.031	0.172	54049

Table 2: Summary statistics: Fertility

Variable	Mean	Std. Dev.	N
All			
Birth Dummy	0.107	0.309	1970748
Maximum Malaria Endemicity	4.972	1.113	1970748
Year Specific Age	17.406	10.784	1970748
Number of Existing Children	1.105	1.811	1970748
Years of Schooling	2.82	4.246	1970748
Husband's Years of Schooling	5.503	5.071	1970748
Muslim Household	0.118	0.323	1970748
Scheduled Caste Household	0.121	0.326	1970748
Scheduled Tribe Household	0.083	0.276	1970748
Rural			
Birth Dummy	0.11	0.313	1421785
Maximum Malaria Endemicity	5.014	1.068	1421785
Year Specific Age	17.199	10.768	1421785
Number of Existing Children	1.133	1.851	1421785
Years of Schooling	1.827	3.328	1421785
Husband's Years of Schooling	4.563	4.664	1421785
Muslim Household	0.102	0.303	1421785
Scheduled Caste Household	0.132	0.338	1421785
Scheduled Tribe Household	0.102	0.302	1421785
Urban			
Birth Dummy	0.095	0.294	548963
Maximum Malaria Endemicity	4.852	1.227	548963
Year Specific Age	18.002	10.806	548963
Number of Existing Children	1.024	1.687	548963
Years of Schooling	5.676	5.199	548963
Husband's Years of Schooling	8.205	5.222	548963
Muslim Household	0.164	0.37	548963
Scheduled Caste Household	0.091	0.287	548963
Scheduled Tribe Household	0.029	0.168	548963

Table 3: Summary statistics: Age at First Birth

Variable	Mean	Std. Dev.	N
Age at First Birth	19.144	3.344	74007
Maximum Malaria Endemicity	4.98	1.108	74007
Years of Schooling	2.783	4.205	74007
Husband's Years of Schooling	5.481	5.058	74007
Muslim Household	0.12	0.324	74007
Scheduled Caste Household	0.12	0.325	74007
Scheduled Tribe Household	0.089	0.285	74007
Rural			
Age at First Birth	18.805	3.16	51043
Maximum Malaria Endemicity	5.031	1.068	51043
Years of Schooling	1.783	3.269	51043
Husband's Years of Schooling	4.518	4.638	51043
Muslim Household	0.104	0.306	51043
Scheduled Caste Household	0.132	0.338	51043
Scheduled Tribe Household	0.108	0.311	51043
Urban			
Age at First Birth	20.097	3.649	22964
Maximum Malaria Endemicity	4.837	1.203	22964
Years of Schooling	5.597	5.161	22964
Husband's Years of Schooling	8.192	5.204	22964
Muslim Household	0.162	0.369	22964
Scheduled Caste Household	0.088	0.283	22964
Scheduled Tribe Household	0.034	0.181	22964

Table 4: Mortality and Disease Exposure

	(1)	(2)	(3)	(4)
	Rural		Urban	
	Neonatal Mortality	Infant Mortality	Neonatal Mortality	Infant Mortality
Malaria Intensity X Post	-0.0467***	-0.0431**	-0.0155	-0.0156
	(0.0158)	(0.0216)	(0.0193)	(0.0247)
Observations	160433	160433	54049	54049
r2	0.0228	0.0380	0.0293	0.0382

Notes: Each column represents separate equations. The dependent variable in columns (1) and (3) is neonatal mortality and the dependent variable in columns (2) and (4) is infant mortality. The sample consists of children born between 1954 and 1991, excluding children born in 1960 and 1961. Columns (1) and (2) correspond to rural sample and columns (3) and (4) correspond to urban sample. Malaria max corresponds to the maximum of malarial intensity index in a district. Post is a dummy variable equal to 1 if the child is born after 1961. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, a dummy for the gender of the child, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 5: Mortality and Disease Exposure (Children born in or before 1975)

	(1)	(2)	(3)	(4)
	Rural		Urban	
	Neonatal Mortality	Infant Mortality	Neonatal Mortality	Infant Mortality
Malaria Intensity X Post	-0.0456***	-0.0551**	-0.0227	-0.0245
	(0.0165)	(0.0228)	(0.0215)	(0.0269)
Observations	41610	41610	14700	14700
r2	0.0362	0.0560	0.0597	0.0730

Notes: Each column represents separate equations. The dependent variable in columns (1) and (3) is neonatal mortality and the dependent variable in columns (2) and (4) is infant mortality. The sample consists of children born between 1954 and 1975, excluding children born in 1960 and 1961. Columns (1) and (2) correspond to rural sample and columns (3) and (4) correspond to urban sample. Malaria max corresponds to the maximum of malarial intensity index in a district. Post is a dummy variable equal to 1 if the child is born after 1961. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, a dummy for the gender of the child, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 6: Mortality and Disease Exposure (Mother's Age 30 and Above)

	(1)	(2)	(3)	(4)
	Rural		Urban	
	Neonatal Mortality	Infant Mortality	Neonatal Mortality	Infant Mortality
Malaria Intensity X Post	-0.0438***	-0.0426*	-0.0113	-0.0111
	(0.0162)	(0.0218)	(0.0195)	(0.0244)
Observations	115213	115213	40523	40523
r2	0.0260	0.0419	0.0363	0.0460

Notes: Each column represents separate equations. The dependent variable in columns (1) and (3) is neonatal mortality and the dependent variable in columns (2) and (4) is infant mortality. The sample consists of children born to women 30 and above between 1954 and 1991, excluding children born in 1960 and 1961. Columns (1) and (2) correspond to rural sample and columns (3) and (4) correspond to urban sample. Malaria max corresponds to the maximum of malarial intensity index in a district. Post is a dummy variable equal to 1 if the child is born after 1961. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, a dummy for the gender of the child, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 7: Mortality and Disease Exposure (Born before 1976 to Women Aged 30 and Above)

	(1)	(2)	(3)	(4)
	Rural		Urban	
	Neonatal Mortality	Infant Mortality	Neonatal Mortality	Infant Mortality
Malaria Intensity X Post	-0.0456***	-0.0550**	-0.0227	-0.0245
	(0.0165)	(0.0228)	(0.0215)	(0.0269)
Observations	41605	41605	14700	14700
r2	0.0363	0.0560	0.0597	0.0730

Notes: Each column represents separate equations. The dependent variable in columns (1) and (3) is neonatal mortality and the dependent variable in columns (2) and (4) is infant mortality. The sample consists of children born to women 30 and above between 1954 and 1975, excluding children born in 1960 and 1961. Columns (1) and (2) correspond to rural sample and columns (3) and (4) correspond to urban sample. Malaria max corresponds to the maximum of malarial intensity index in a district. Post is a dummy variable equal to 1 if the child is born after 1961. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, a dummy for the gender of the child, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 8: Robustness Check: Mortality Outcomes

	(1)	(2)	(3)	(4)
	Rural		Urban	
	Neonatal Mortality	Infant Mortality	Neonatal Mortality	Infant Mortality
Malaria Intensity X Post	-0.00512	-0.00627	-0.000663	0.000730
	(0.00345)	(0.00463)	(0.00399)	(0.00413)
Observations	127018	127018	42608	42608
r2	0.0229	0.0360	0.0285	0.0348

Notes: Each column represents separate equations. The dependent variable in columns (1) and (3) is neonatal mortality and the dependent variable in columns (2) and (4) is infant mortality. The sample consists of children born between 1971 and 1991, excluding children born in 1980 and 1981. Columns (1) and (2) correspond to rural sample and columns (3) and (4) correspond to urban sample. Malaria max corresponds to the maximum of malarial intensity index in a district. Post is a dummy variable equal to 1 if the child is born after 1981. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, a dummy for the gender of the child, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 9: Robustness Check: Mortality Outcomes (Mother's Age 30 and Above)

	(1)	(2)	(3)	(4)
	Rural		Urban	
	Neonatal Mortality	Infant Mortality	Neonatal Mortality	Infant Mortality
Malaria Intensity X Post	-0.00222	-0.00355	0.00333	0.00573
	(0.00396)	(0.00500)	(0.00434)	(0.00493)
Observations	83838	83838	29598	29598
r2	0.0274	0.0412	0.0378	0.0447

Notes: Each column represents separate equations. The dependent variable in columns (1) and (3) is neonatal mortality and the dependent variable in columns (2) and (4) is infant mortality. The sample consists of children born to mothers aged more than 30 between 1971 and 1991, excluding children born in 1980 and 1981. Columns (1) and (2) correspond to rural sample and columns (3) and (4) correspond to urban sample. Malaria max corresponds to the maximum of malarial intensity index in a district. Post is a dummy variable equal to 1 if the child is born after 1981. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, a dummy for the gender of the child, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 10: Probability of Female Birth

	(1)	(2)	(3)	(4)
	All Sample	Restricted Sample	Mother's Age $\geq$ 30	Restricted Sample and Mother's Age $\geq$ 30
Panel A: Rural				
Malaria Intensity X Post	0.00580	0.0166	0.00340	0.0167
	(0.0277)	(0.0274)	(0.0275)	(0.0274)
Observations	160433	41610	115213	41605
r2	0.00577	0.0208	0.00753	0.0208
Panel B: Urban				
Malaria Intensity X Post	0.0227	0.0488	0.0268	0.0488
	(0.0391)	(0.0471)	(0.0400)	(0.0471)
Observations	54049	14700	40523	14700
r2	0.0109	0.0381	0.0154	0.0381

Notes: Each column represents separate equations. The dependent variable is a dummy equal to 1 if he child is a female. Panel A presents the results for the rural sample and panel B for the urban sample. The sample in column (1) consists of children born between 1954 and 1991, excluding children born in 1960 and 1961. Column (2) restricts the analysis only to children born in or before 1975. Column (3) presents results for children born to mothers aged 30 and above. Column (4) presets the results for children born to women 30 and above between 1954 and 1975. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 11: Robustness Check: Probability of Female Birth

	(1)	(2)	(3)	(4)
	All Women		Age $\geq$ 30	
	Rural	Urban	Rural	Urban
Malaria Intensity X Post	0.00241	-0.00756	-0.00362	-0.00796
	(0.00709)	(0.00952)	(0.00803)	(0.0123)
Observations	127018	42608	83838	29598
r2	0.00708	0.0131	0.0103	0.0200

Notes: Each column represents separate equations. The dependent variable is a dummy equal to 1 if the child born in year t is a female. The sample in columns (1) and (2) consists of years between 1971 and 1991, excluding the years 1980-1984. Columns (3) and (4) restricts the analysis children born to women aged 30 and above. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, parental years of schooling, mother's year specific age, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 12: Fertility and Disease Exposure

	(1)	(2)	(3)	(4)
	All Sample	Restricted Sample	Age $\geq$ 30	Restricted Sample and Age $\geq$ 30
Panel A: Rural				
Malaria Intensity X Post	0.00554***	0.00574**	0.0102***	0.00616**
	(0.00187)	(0.00222)	(0.00297)	(0.00302)
Observations	1421785	567989	803066	386298
r2	0.0516	0.158	0.0603	0.137
Panel B: Urban				
Malaria Intensity X Post	0.00136	-0.00417	0.00136	-0.00649
	(0.00190)	(0.00360)	(0.00299)	(0.00475)
Observations	548963	230896	345177	165945
r2	0.0427	0.148	0.0566	0.134

Notes: Each column represents separate equations. The dependent variable is a dummy equal to 1 if the woman gives birth in the year t. Panel A presents the results for the rural sample and panel B for the urban sample. The sample in column (1) consists of years between 1954 and 1991, excluding the years 1960-1964. Column (2) restricts the analysis only to 1975 and before. Column (3) presents results for women aged 30 and above. Column (4) presets the results to women aged 30 and years 1975 and before. Each regression includes district fixed effects, year fixed effects, district specific linear time trends, year specific age, existing number of children, parental years of schooling, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 13: Robustness Check: Fertility

	(1)	(2)	(3)	(4)
	All Women		Age $\geq$ 30	
	Rural	Urban	Rural	Urban
Malaria Intensity X Post	0.000587	-0.00222	-0.000198	-0.000770
	(0.00228)	(0.00342)	(0.00347)	(0.00461)
Observations	830801	313747	416768	179232
r2	0.0300	0.0239	0.0371	0.0420

Notes: Each column represents separate equations. The dependent variable is a dummy equal to 1 if the woman gives birth in the year t. The sample in columns (1) and (2) consists of years between 1971 and 1991, excluding the years 1980-1984. Columns (3) and (4) restricts the analysis only to women aged 30 and above for the years 1971-1991. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, parental years of schooling, year specific age, existing number of children, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 14: Age at First Birth and Disease Exposure

	(1)	(2)	(3)	(4)
	All Sample	Restricted Sample	Age $\geq$ 30	Restricted Sample and Age $\geq$ 30
Panel A: Rural				
Malaria Intensity X Post	-0.374***	-0.244**	-0.436***	-0.243**
	(0.0905)	(0.112)	(0.122)	(0.111)
Observations	51043	16786	29114	16782
r2	0.180	0.223	0.408	0.223
Panel B: Urban				
Malaria Intensity X Post	-0.152	-0.149	-0.243*	-0.149
	(0.138)	(0.148)	(0.140)	(0.148)
Observations	22964	7418	14859	7418
r2	0.290	0.295	0.500	0.295

Notes: Each column represents separate equations. The dependent variable is the age of first birth. Panel A presents the results for the rural sample and panel B for the urban sample. The sample in column (1) consists of women with first births between 1954 and 1991, excluding the years born in 1960 and 1961. Column (2) restricts the analysis only to women with first births in or before 1975. Column (3) presents results for women aged 30 and above. Column (4) presets the results for women 30 and above with their year of first births between 1954 and 1975. Each regression includes district fixed effects, year of first birth fixed effects, district specific linear time trends, parental years of schooling, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 15: Robustness Check: Age at First Birth

	(1)	(2)	(3)	(4)
	All Women		Age $\geq$ 30	
	Rural	Urban	Rural	Urban
Malaria Intensity X Post	0.0213	0.0558	0.0586	0.128
	(0.0653)	(0.0856)	(0.0724)	(0.102)
Observations	41873	19032	19897	10873
r2	0.165	0.270	0.405	0.466

Notes: Each column represents separate equations. The dependent variable is the age of first birth. The sample in columns (1) and (2) consists of women with year of first birth between between 1971 and 1991, excluding children born in 1980 and 1981. Columns (3) and (4) restricts the analysis only to women aged 30 and above for the years 1971-1991. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, parental years of schooling, mother's year specific age, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Appendix Table 1: Mortality and Disease Exposure

	(1)	(2)	(3)	(4)
	All Sample	Restricted Sample	Mother's Age $\geq$ 30	Restricted Sample and Mother's Age $\geq$ 30
Panel A: Rural				
Malaria Intensity X Post	-0.0441**	-0.0535**	-0.0438**	-0.0535**
	(0.0207)	(0.0219)	(0.0208)	(0.0219)
Observations	160433	41610	115213	41605
r2	0.0354	0.0528	0.0394	0.0527
Panel B: Urban				
Malaria Intensity X Post	-0.0166	-0.0277	-0.0129	-0.0277
	(0.0249)	(0.0277)	(0.0247)	(0.0277)
Observations	54049	14700	40523	14700
r2	0.0360	0.0687	0.0433	0.0687

Notes: Each column represents separate equations. The dependent variable is a dummy equal to 1 if the child died by the age of 11 months. Panel A presents the results for the rural sample and panel B for the urban sample. The sample in column (1) consists of children born between 1954 and 1991, excluding children born in 1960 and 1961. Column (2) restricts the analysis only to children born in or before 1975. Column (3) presents results for children born to mothers aged 30 and above. Column (4) presets the results for children born to women 30 and above between 1954 and 1975. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Appendix Table 2: Probability of Birth with Varying Lags

	(1) Lag 2	(2) Lag 3	(3) Lag 4
Panel A: Rural			
Malaria Intensity X Post	0.00467***	0.00532***	0.00540***
	(0.00164)	(0.00173)	(0.00184)
Observations	1505594	1479528	1451669
r2	0.0558	0.0547	0.0536
Panel B: Urban			
Malaria Intensity X Post	0.00122	0.00135	0.00131
	(0.00172)	(0.00178)	(0.00185)
Observations	584783	573633	561734
r2	0.0465	0.0455	0.0443

Notes: Each column represents separate equations. The dependent variable is a dummy equal to 1 if he child is a female. Panel A presents the results for the rural sample and panel B for the urban sample. The sample in column (1) consists of children born between 1954 and 1991, excluding children born in 1960 and 1961. Column (2) excludes children born in 1960, 1961 and 1962. Column (2) excludes children born in 1960-1963. Each regression includes district fixed effects, year of birth fixed effects, district specific linear time trends, order of birth fixed effects, parental years of schooling, dummies for Scheduled Caste, Scheduled Tribe and Muslim households. The Standard errors are reported in parentheses. Errors are clustered at district level.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$