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Life expectancy and human capital investments: Evidence from maternal mortality declines

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Abstract

Is high mortality a major impediment to human capital accumulation? A shorter time horizon decreases the value of investments that pay out over time, so lower life expectancy could reduce the incentive to invest in human capital. The empirical importance of this life-expectancy effect is unresolved, though, partly because previous research has been unable to isolate it from the many other effects that health has on education. We examine a sudden drop in maternal mortality risk in Sri Lanka between 1946 and 1953 and use variation across districts, time and gender to identify the effects of longevity on education. We find that the 70% reduction in maternal mortality risk over the sample period increased female life expectancy at age 15 by 1.5 years (4.1%) and increased female literacy by 2.5%.

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1. Introduction

A person will invest more if the stream of returns on the investment lasts longer, all else equal. An important implication is that improvements in life expectancy will increase investment in human capital, which in turn may spur economic growth.

A large literature has explored this idea theoretically (Ben Porath 1967, Kalemli-Ozcan, Ryder and Weil 2000, Soares 2005, Murphy and Topel 2005). Most previous empirical research measures the cross-country relationship between life expectancy and growth, education or investment (Shastry and Weil 2003, Lorentzen, McMillan and Wacziarg 2005, Acemoglu and Johnson 2006).[1] One limitation of this approach is that much of the cross-country variation over time in life expectancy is driven by changes in infant mortality, or deaths that occur before schooling begins. Infant mortality might affect education but not by changing the expected period over which returns to education will be earned. Also, the identifying variation in mortality is primarily from reductions in tuberculosis, malaria, and pneumonia, which also dramatically lowered morbidity rates.

Another branch of the literature presents evidence on the effects of health on education, but does not aim to disentangle incentive effects of a longer life from direct effects of healthier children being more able to attend school. For example, Miguel and Kremer (2004) and Bleakley (2007) show that deworming interventions led to increased school attendance. The main interpretation of their findings is that sickness had been preventing children from attending or succeeding in school.

This paper's main contribution is to obtain estimates of how health affects education specifically through the channel of increased life expectancy. We use reductions in maternal mortality, a source of variation that lends itself to better isolating life-expectancy effects on behavior. The setting we study, Sri Lanka between 1946 and 1953, is particularly well suited for the analysis.[2] In 1946, the maternal mortality ratio (MMR) in Sri Lanka was 1.8 maternal

[1] The evidence is mixed on the quantitative importance of improvements in life expectancy on growth and human capital. Shastry and Weil (2003) and Lorentzen, McMillan and Wacziarg (2005) find large effects, while Acemoglu and Johnson (2006) find small effects. See also Weil (2007), Young (2005), Rodriguez and Sachs (1999), and Barro and Sala-i-Martin (1995).

[2] We investigated other countries and time periods for which MMR declined rapidly (and mortality statistics exist to study the decline). In the other cases, MMR improvements increased life expectancy by much less. For example, MMR fell dramatically in the US around 1936 after the introduction of sulfa drugs (Thomasson and Treber 2004). However, life table calculations suggest that the elimination of maternal mortality in the US could have at most increased female life expectancy by less than 0.5 years (Retherford 1972).

deaths per 100 live births, or 1.8%. By 1953, MMR had fallen by two thirds to 0.5%. These declines were large, not just in percentage terms but also in levels; they had a substantial effect on life expectancy for women. The dramatic nature of the decline is also helpful for identification since one can hope to separate the effects caused by the MMR decline from slower secular changes in education. It is also advantageous that within Sri Lanka there is considerable geographic variation in the declines.

Maternal mortality has several advantages in terms of empirical identification. First, maternal mortality occurs after major human capital investments are made. Therefore, investment decisions will depend on this risk, unlike infant mortality risk which is realized *before* educational decisions are made. Second, because maternal mortality occurs early in adult life, an averted maternal death translates into a large increase in life expectancy. Third, this cause of death directly affects only women, so men can serve as a comparison group. Finally, maternal mortality is not associated with large morbidity rates among the school-age population.[3] This fact is important because if mortality declines were associated with declines in morbidity among those in school, as is the case, for example, with hookworm and malaria, the incentive effects of increased life expectancy would be confounded with the effects of morbidity reductions that facilitated school attendance.[4] These features make maternal mortality an attractive source of variation for isolating the incentive effects of longer life expectancy.[5]

[3] Pregnancy, childbirth and complications during the postpartum period are estimated to result in subsequent disability or illness (such as fistulae) for about 15% of women today (WHO 1994). Reductions in maternal mortality could be associated with higher or lower incidence of these complications.

[4] Lucas (2005) studies several countries that underwent malaria eradication and finds that cohorts born after malaria eradication are more educated than those born before. This pattern could reflect the higher incentive to invest in human capital when life expectancy is higher, but also could reflect morbidity reductions.

[5] Using HIV/AIDS risk to examine how education responds to life expectancy is confounded by HIV morbidity among school-age children, and the larger problem of parents' health being affected along with the child's expected health. A child's HIV risk, as proxied by current HIV rates, is correlated with her parent's actual and anticipated morbidity and mortality from HIV. MMR has several advantages over HIV. Income effects from parents' affliction will be higher for HIV both because a long illness usually precedes an HIV death, and because HIV, unlike maternal mortality, also affects fathers. Fathers' contribution to household income is typically higher and, moreover, both of a child's parents might be stricken with HIV. Also, in high-prevalence settings, HIV has more severe community-wide effects that could affect education (e.g., effects on teacher supply). In principle, variation in the individual's risk, conditional on parental or community risk, would address these problems, but such a strategy (whether for HIV or MMR) is not empirically viable with most data sets or in most settings. Fortson (2007) finds a negative effect of HIV prevalence on school attendance in Africa. She interprets the results as partly explained by direct effects from morbidity and income effects due to sick parents, but due mainly to lower expected lifetime returns to education due to mortality risk.

Our estimation strategy is a difference-in-difference-in-difference (DDD): we use variation across districts, over time and by gender to identify the effects of longevity on education and other outcomes. We use mortality and birth data from Sri Lanka's vital statistics, which is based on a registration system and has been shown to be close to complete (United Nations 1976, World Bank 2003). Thus our life expectancy measures are considerably more accurate than country-level measures, which differ in their accuracy and often are based solely on infant mortality (Deaton 2006).

We find that the 70% reduction in maternal mortality risk that occurred between 1946 and 1953 increased female life expectancy at age 15 (censored at 65) by 1.5 years, or 4.1%, and can account for the entire difference in life expectancy gains between men and women.[6] These declines in maternal mortality increased female literacy among the affected cohorts by 2.5% (one percentage point off a base of 44%). Thus the estimated elasticity of literacy with respect to life expectancy is 0.6.

The estimation strategy makes three main assumptions. First, the underlying behavioral model is that people's choices depend on their subjective life expectancy, and our analysis assumes that people rationally update their subjective life expectancy based on changes in objective life expectancy.[7] The total fertility rate in Sri Lanka was 5 births over a lifetime, so MMR of 1.8% implies a lifetime risk of dying in childbirth on the order of 9%. It seems likely that people would have noticed the rapid near-elimination of a large risk, particularly since complications from childbirth are a readily identifiable cause of death. (We do not assume they notice the reduction instantly; we allow there to be a lag.)

Second, the analysis compares outcomes for females and males. Our estimates would be biased if maternal mortality were correlated with other factors that had gender-specific effects on human capital investment. Maternal mortality declines in Sri Lanka were driven by improved availability of health care and transportation to hospitals at the time of delivery. Eradication of malaria, which is a risk factor for maternal death, also contributed to the declines. These policies very likely affected morbidity rates among school-age children. The estimation strategy allows for other gender-neutral effects of these policies within a district-

[6] We use expected years of life between ages 15 and 65 because these are the years over which returns on human capital are likely to mainly accrue. A more complete description is given later in the text and the exact formulae used for computations can be found in the data appendix.

[7] Hurd and McGarry (2002) have shown evidence of such updating elsewhere. There are no data on subjective expectations to verify the finding in our setting.

year, but the assumption is that their differential effect on females was due to their large impact on maternal mortality risk. Using males as a comparison group also assumes that there are no appreciable spillover effects on boys when girls' life expectancy and education increase. For example, effects such as a family budget constraint causing an increase in a girl's education to crowd out her brother's education are assumed to be small.

Another potential limitation is that children's education can be affected when their mother is less likely to die in childbirth. In our framework, a decline today in maternal mortality affects girls' education because expectations about the girl's future maternal mortality risk are revised. The mother is also at lower risk of dying in childbirth now, and there might be effects on children's education from the mother's survival. If these effects are gender-neutral, they do not threaten the validity of our estimates, but one might worry that there are gender-specific effects. However, as we show in the paper, the decrease in orphanhood is much too small to explain our estimates.

The paper is organized as follows. The next section describes the theoretical predictions that we test in the data. Section 3 gives background information on Sri Lanka. Section 4 describes the data. In Section 5 we discuss our empirical strategy. Section 6 quantifies the effects of maternal mortality on life expectancy. Section 7 examines the effects of MMR on literacy, and Section 8 presents the effects on fertility and other behaviors. Section 9 concludes.

2. Conceptual framework

This paper tests the hypothesis that changes in life expectancy affect human capital investments and other behavioral choices. To lay out the empirical predictions, we present a simple model of schooling and fertility choices, and examine comparative statics when mortality rates change.

Consider a unitary household consisting of a woman and man who make two decisions, whether to have a child and how much schooling to give their child. The decisions depend, in part, on the risk of maternal mortality. For the fertility decision, the risk of maternal mortality is a cost to the (potential) mother, and also affects the utility derived from a daughter. For the schooling decision, a daughter's maternal mortality risk will affect her returns to schooling. In the model, and also in the empirical analysis, it is assumed that a

reduction in mortality risk today changes beliefs about both current risk and future risk. For the mother (older cohorts), education is predetermined but fertility is affected, and for the daughter (younger cohorts), her educational attainment is also affected.

We use a standard Mincerian model of returns to education: each year of schooling leads to a certain percentage increase in earnings.[8] Earnings are just one, and perhaps not the most important, benefit of education for females, particularly in the context we study (Haveman and Wolfe 1984). Other potential benefits of education are that it improves her health; enables her to match with a higher "quality" husband; increases her bargaining power in the household; improves her effectiveness in using contraceptives and controlling her fertility; and improves the quality (e.g., education or health) of her children (Rosenzweig and Schultz 1989, Thomas, Strauss, and Henriques 1991, Glewwe 1999, Peters and Siow 2002). We model earnings because it is the most standard outcome to model, but the model should be thought of as also encompassing other benefits of education that provide a stream of utils during post-schooling years.

Consider a unitary household that maximizes the sum of the present discounted value of income of the wife, the husband, and the (potential) child. The subscript w is used to denote wife, h denotes husband, b denotes boy and g denotes girl. The household makes a binary choice C_w about whether to have a child and then chooses the years of schooling s of the child after observing the child's gender. We treat the next generation's childbearing decision as exogenous; a daughter will have a child with probability C_g.[9]

Having a child occurs at time τ in the woman's lifetime. The decision problem we model occurs at this moment of childbearing for the wife. We assume that childbearing results in the mother's death with probability μ, and that this is the only uncertainty in life expectancy. Conditional on surviving childbirth, the wife lives until time T_w. The husband

[8] The empirical analysis and hence the model focus on education, but the reasoning could apply to health investments as well. As one mortality risk (maternal mortality) declines for daughters, parents would have an incentive to invest in preventing other competing mortality risks or to make health investments that give a flow of payoffs throughout their daughter's life. Dow, Philipson, and Sala-i-Martin (1999) discuss theoretically how health investments respond to increased longevity and provide evidence that other health measures improved in response to vaccination availability. Oster (2007) finds that HIV/AIDS avoidance behavior is more pronounced when there is lower non-HIV mortality, e.g., due to lower malaria prevalence or maternal mortality risk.

[9] We make this assumption, rather than considering infinite generations, because it simplifies the model without qualitatively changing the comparative statics that the model lays out.

faces no longevity uncertainty and lives until T_h. Households have a discount rate δ. The return to schooling is γ, and (instantaneous) income is y for someone with no schooling.

The household's maximization problem is

$$\max_{s_b, s_g, C_w} \left[Y_w(C_w) + Y_h + \frac{C_w}{2}(Y_b(s_b) + Y_g(s_g)) \right]$$

where

$$Y_w = (1 - C_w\mu)\int_{\tau}^{T_w} e^{-\delta(t-\tau)} y e^{\gamma s_w} dt, \quad Y_h = \int_{\tau}^{T_h} e^{-\delta(t-\tau)} y e^{\gamma s_h} dt,$$

$$Y_g = \int_{s_g}^{\tau} e^{-\delta t} y e^{\gamma s_g} dt + (1 - C_g\mu)\int_{\tau}^{T_g} e^{-\delta t} y e^{\gamma s_g} dt, \text{ and } Y_b = \int_{s_b}^{T_b} e^{-\delta t} y e^{\gamma s_b} dt,$$

and the factor of ½ represents the (approximately accurate) assumption that there is equal probability of having a boy or a girl. Note that the schooling level is already determined for the wife and husband, and they are currently earning at the time of the decision. We abstract from the foregone earnings of parents who are raising a child since that would not affect the comparative statics of interest. For the child, the income stream begins in the future, upon completion of his or her schooling.

Working backwards, conditional on having a girl, the schooling decision is determined by:

$$\max_{s_g} \int_{s_g}^{\tau} e^{-\delta t} y e^{\gamma s_g} dt + (1 - C_g\mu)\int_{\tau}^{T_g} e^{-\delta t} y e^{\gamma s_g} dt$$

The optimal schooling level is

$$s_g^* = \frac{1}{\delta}\left(\ln\frac{\gamma - \delta}{\gamma} - \ln\left[C_g\mu e^{-\delta\tau} + (1 - C_g\mu)e^{-\delta T_g} \right] \right),$$

yielding the comparative static that girls obtain more schooling when the risk of maternal mortality falls (μ decreases):[10]

$$\frac{\partial s_g^*}{\partial \mu} = -\frac{C_g}{\delta}\frac{e^{-\delta\tau} - e^{-\delta T_g}}{C_g\mu e^{-\delta\tau} + (1 - C_g\mu)e^{-\delta T_g}} < 0 \tag{2.1}$$

[10] Another result is that the effect given in (2.1) is larger when mortality reductions occur at younger ages (when τ is lower). We do not empirically test the cross derivative, but it is a reason why maternal mortality, which occurs early in post-schooling years, is advantageous in terms of statistical power. The earlier in productive life the mortality risk occurs, the larger the incentive effects on investment from reduced risk.

As modeled, the reduction in maternal mortality risk does not affect boys' education.

$$\frac{\partial s_b}{\partial \mu} = 0 \tag{2.2}$$

Under different assumptions, one might find a positive or negative effect on boys' education. For example, if we incorporated credit constraints and extended the model to allow for multiple children per household, then higher returns to a daughter's education might crowd out her brothers' education.

Maternal mortality risk also affects the decision to have a child:[11]

$$\frac{\partial C_w}{\partial \mu} \leq 0 \tag{2.3}$$

The household will have a child if the benefit (utility from the child) outweighs the cost (risk of utility loss from the mother's death). Maternal mortality risk affects this tradeoff through two channels, both of which operate in the same direction. First, higher μ raises the cost of childbearing because of the risk to the mother. Second, it lowers the benefit of childbearing since, if the child is a girl, she will have a shorter expected life and generate less utility for the household. This second effect illustrates an important point that fertility choice will be affected by *any* change in the expected longevity of children (Soares 2005). In our case, the change in longevity (change in μ) also applies to those making the fertility decision, and it applies conditional on having a child.

Other effects

As modeled, choosing between zero and one child presents no "quantity-quality" tradeoff. Households respond to the maternal mortality decline by (weakly) having more children because maternal mortality risk, in essence, raises the price of quantity. If one also modeled the intensive margin for fertility, households might shift from quality to quantity, having more children and educating each of their children less. This effect, though, would not necessarily change gender differentials in education, if quality falls for both boys and girls. Since the expected lifetime returns to female education increase when MMR falls, parents might shift instead from quantity to quality, having fewer children but educating each one more (Becker, Murphy, and Tanamura 1990, Galor and Weil 2000, Bleakley and

[11] The derivative for a discrete variable is of course not defined. The notation is shorthand for the comparative static: as μ increases, all else held constant, the household is weakly more likely to choose C_w=0.

Lange 2006).[12] This incentive would create an offsetting effect to the higher fertility induced by greater maternal survival modeled above. But again this would not necessarily imply differences in how parents educate boys versus girls.

Finally, for parsimony we do not incorporate into the model the effect of MMR on age at marriage, which is one of the outcomes we examine empirically. MMR has effects on education and fertility, both of which could affect the timing of marriage, and the overall effect of MMR on age at marriage is ambiguous.[13]

3. Background on Sri Lanka

Sri Lanka was and is a poor country, but on many dimensions of human development it is quite advanced. Its progress against maternal mortality sixty years ago is one example. Sri Lanka during 1946-1953 also had higher educational participation and gender equality in education than most poor countries. The education system was organized into three levels: primary school (ages 5-11), secondary school (ages 12-18) and higher learning. In 1945, fees in state-assisted schools, which made up the majority of schools and were open to both genders, were abolished. There was also a shift away from English toward local languages (Sinhalese or Tamil) as the medium of instruction during this period. School enrollment significantly increased between 1946 and 1953: the proportion of children ages 5 to 14 in school went up from 58% to 72% (Jayaweera 1969).[14]

Decisions about how much education to obtain should respond to MMR declines only if there are returns to education that accrue over time. While no solid causal estimates of the returns to education exist for the cohorts we study, Mincerian estimates for Sri Lanka suggest a return to a year of education of 7% for both males and females (Psacharopoulos 1994). Consistent with the hypothesis that education also has benefits outside of the labor

[12] Bleakley and Lange (2006) find that fertility declined in response to the eradication of hookworm in the U.S. South, consistent with a shift from quantity to quality.

[13] First, if girls delay marriage until education is complete, then a reduction in MMR, by increasing female education, would increase female age at marriage. Second, because in Sri Lankan society, widowers (men) typically remarried but widows (women) did not, a decline in female mortality may have caused a "marriage squeeze" for women because of the reduced supply of widowers seeking wives (Dixon 1970, Fernando 1975). This effect also would lead MMR declines to increase female age at marriage. Third, a reason to delay marriage in a society with limited birth control is to delay the onset of fertility and reduce total fertility. A reduction in MMR would increase the demand for childbearing, causing female age at marriage to decrease. Hence, on net, it is ambiguous whether lower MMR would lead women to get married earlier or later.

[14] The enrollment rate for 5-14 year olds is available for Sri Lanka as a whole. District-gender data on enrollment is aggregated for 5-24 year olds.

market, unreported OLS regressions using the 1987 Demographic and Health Survey for Sri Lanka suggest that more education for a woman is associated with marriage to a more educated man and with lower infant mortality among her children.

The total fertility rate (average number of children a woman gave birth to over her lifetime) was approximately 5 (Langford 1981). The birth rate increased between 1946 and 1953 and then decreased between 1953 and 1963, so Sri Lanka appears to have entered its fertility transition (period of declining fertility) in the late 1950s, after the study period.[15] The average age of marriage was 21 for females and 28 for males.[16]

Sri Lanka's reduction in maternal mortality was driven by several policies related to health. Several main factors related to access to health care are commonly cited (World Bank 2003). The first was the expansion of health care services, mostly concentrated on improving maternal and infant health. The number of hospitals, clinics and health centers in the country rose considerably, and many of these were specifically used for maternal and child services. The number of trained birth attendants also increased. Importantly, most of the services were provided for free. Second, to increase access to healthcare, transportation to health facilities improved: a system of free ambulances was developed, and if ambulances were not available, then transportation in cases of emergencies would be reimbursed by the government (World Bank 2003). Figure 1 shows Sri Lanka's increase in its number of ambulances, health centers, government midwives, and hospital beds. The proportion of women delivering at health clinics or hospitals, rather than at home, increased from 20% in 1945 to 55% in 1960, suggesting that access to care indeed improved (World Bank 2003). Third, Sri Lanka adopted recently-developed technologies from the West, most importantly sulfa drugs, penicillin and blood transfusions, technologies that had markedly reduced maternal mortality in the West (Loudon 2000a, 1991, 1988, Paxton et al 2004, De Brouvere and Lerberge 2001).

Finally another factor that contributed to MMR declines was malaria eradication. In 1945 Sri Lanka began a successful malaria control program centered on DDT spraying, and malaria death rates fell sharply. Reports on maternal mortality at the time linked malaria to

[15] Note that family planning activities did not start in earnest in Sri Lanka until 1965, with the initiation of the National Family Planning Program (World Bank 2003).

[16] Sri Lanka is primarily a Buddhist society, so its marriage customs differ from the rest of South Asia, which is primarily Hindu or Muslim. During the period we study, about two thirds of marriages were arranged, and dowry played a limited role in Sri Lanka (Caldwell 1999).

maternal mortality (De Silva 1943). Malaria is thought to cause anemia and increase the likelihood of death from hemorrhage at birth (WHO 2007).

Finally, we note that for most other factors that may affect maternal mortality (such as malnutrition, parity, or mother's age), the consensus in the literature is that such factors are in fact not as important in magnitude as access to proper care at the time of delivery.[17]

The factors described above seem to have caused bigger improvements in MMR in places with initially higher levels of MMR. This pattern is shown in Figure 2, which plots for Sri Lanka's 19 districts the decline in MMR between 1946 and 1953 versus the 1946 level. (The data will be described in detail in the next section.) The slope of the relationship is -0.7. This strong β-convergence combined with the across-the-board declines in MMR imply that the initially-high-MMR districts essentially caught up to the initially-low-MMR districts by 1953. Note that each data point is a three-year running average, which should minimize measurement error and the extent to which the pattern could be due to mean reversion.

A final and important piece of background information is that Sri Lanka became independent from Britain in 1948, during the study period. In 1931 self-governance was instituted, so the transition to independence was peaceful and the ruling politicians continued to hold power after independence. Therefore, although independence was of course momentous, it was not associated with dramatic changes in government or policy in 1948 (Peebles 2006). For the purposes of our empirical analysis, which uses district-gender variation, countrywide effects of independence are not a confounding factor, nor are district-level or gender-level effects. The same point applies to other national events that occurred, such as the changes in education policy mentioned above.

[17] Prenatal care does not seem to be a major determinant of maternal mortality because most complications at birth cannot be predicted (Maine et al 1991). Recent work is also skeptical about the relationship between nutrition and maternal deaths (Loudon 2000a, Maine 2000). Loudon (2000a, p. 241S) states this view most strongly: "the main determinant of maternal mortality was the overall standard of maternal care provided by birth attendants. Poverty and associated malnutrition played little part in determining the rate of maternal mortality." Maternal age and parity also seem to have a relatively small quantitative impact on maternal mortality. Maternal mortality is highest for very young and old mothers, and it is also higher for first born and for high-parity births. Hence, changes in the number and timing of births may affect maternal mortality. However, Trussell and Pebley (1984) calculate that, in most settings, eliminating all births by women under 20 and over 39, as well as all births parity six or higher would reduce maternal mortality by only about 25%. Thus, even large changes in fertility behavior could not explain the dramatic declines in MMR in Sri Lanka.

4. Data and descriptive statistics

The data for the analysis were collected primarily from annual Vital Statistics reports and the Census of Population for 1946 and 1953. The data are disaggregated geographically into 19 districts. See the data appendix for details.

Vital statistics data on the maternal mortality ratio (ratio of maternal deaths to births) are available by district and year from 1941 onwards.[18] MMR for 1925 to 1939, which we use in Figure 3a, are from De Silva (1943) who calculated them from vital statistics data for those years. Figure 3a shows MMR over time, by district, from 1925 to 1964. As can be seen, MMR fell considerably over the period. Figure 3b focuses on 1939 to 1955 and shows MMR averaged across districts. The sharpness of the decline after 1946 can be seen clearly, as well as the fact that most of the decline occurred by 1950. A test for a trend break in MMR between 1930 and 1960 identifies 1947 as the break year.[19] Also evident is a spike in MMR in 1946, thought to be caused by a malaria epidemic that year. To ensure that the trend break in 1947 is not spuriously created by the spike, we repeated the trend break test excluding 1945 and 1946 (or only 1946), and again the data choose a trend break in 1947.

For variables available annually, the regression analysis uses three-year running averages (e.g., for 1946, we average 1945 to 1947 and for 1953 we average 1952 to 1954). The main purpose is to reduce measurement error, but it has the added advantage that the results are less sensitive to variation driven by the 1946 spike. Table 1a shows that the mean of three-year-averaged MMR is 1.80 maternal deaths per 100 births or 1.8% in 1946 and 0.5% in 1953. We also use MMR lagged by 3 years, which also declined by 1.3 percentage points over the study period.

Total deaths are available by district, year, gender and age (5-year groups) and are used to construct age-specific death rates (by using interpolated population counts from the Census), which are shown in Table 1b. Death rates exhibit the usual age profile, with high infant mortality and increasing mortality after age 40. In 1946 females show larger death rates than males up until age 45, but lower mortality rates for older ages. The ratio of female to male deaths is the largest for ages 15 to 45, a noteworthy pattern since these are the

[18] MMR data are also in the 1940 Vital Statistics but we were unable to obtain the 1940 Vital Statistics.
[19] Using district-year level data from 1930 to 1960, we regress MMR on a continuous year variable, a year dummy, and the interaction of year*dummy. We repeat the procedure, allowing the year dummy to vary from 1939 to 1953. The highest R-squared is obtained for 1947 (even if we drop 1935 and 1946 or 1935, 1945 and 1946, years with malaria epidemics).

childbearing years. Mortality rates at all ages fell significantly during the period. The mortality decline in combination with an increase in the birth rate resulted in a population increase from 6.7 million in 1946 to 8.3 million in 1953.

The mortality data are believed to be of excellent quality[20] and allow us to construct life expectancy measures for each district-year-gender (World Bank 2003). We construct measures of life expectancy at age *a* censored at age *b*, which we denote as e(*a*-*b*) following the notation in demography. For example, our main measure is e(15-65), which is life expectancy at 15 censored at 65, or the expected years alive between ages 15 and 65, conditional on survival until 15. We censor life expectancy at age 65 because the death rates in the early vital statistics reports are not reported for older ages, and life expectancy calculations are in general very sensitive to assumptions about the distribution of deaths among those censored.

The life expectancy measures are calculated in the standard way from age-specific mortality rates, measured at a particular point in time. The data appendix describes the procedure in detail. Ages 15 to 65 are a plausible span over which returns to education are mainly earned, so we use e(15-65) as the life expectancy metric to which educational decisions should be responsive. We construct e(45-65) to show that MMR reductions are not significantly correlated with relative female life expectancy gains in the post-childbearing years (although because of competing risks, MMR could have an effect on mortality rates at post-childbearing ages). We also construct e(0-15) to verify that MMR reductions were not correlated with gender-specific reductions in mortality in the pre-childbearing years. Finally, we construct e(0-65), which is used as a control variable in some specifications.

Table 1a shows that there was a large improvement in life expectancy from 1946 to 1963, with e(15-65) increasing by 7.4 years for women and 6.6 years for men. Life expectancy also shows convergence across gender: the difference between men and women was 2.3 years in 1946 but 1.5 years by 1953. Published life expectancy measures by gender for the entire country show very similar patterns (Nadarajah 1983, United Nations 1976).

Data on deaths broken down by cause are available from the vital statistics by district, year, and gender, but not age. Cause of death is reported in fine categories that we

[20] Sri Lanka's registration system began in 1867 and is highly regarded (Levine 2007). Studies of the completeness of birth and death records show very high completeness (United Nations 1976).

aggregate up to broader causes of death for use as control variables. Table 1b reports the means for the diseases we use in the analysis. We arrived at the list of diseases using two distinct criteria. First, we selected diseases that a priori seem likely to have been affected by the major health interventions in the period, which centered on malaria, nutrition and helminths. Second, we collected cause-specific mortality rates for almost all causes of death, and then selected the diseases with the highest death rates among school-age children, under the assumption that diseases with high mortality rates also had high morbidity rates.[21] The two criteria yielded almost the same set of diseases; these diseases—malaria, vitamin deficiencies, diarrhea, helminths, and anemia–will be used as control variables.[22]

Vital statistics are also the data source for births and marriages. They report total number of births by district and year. Birth registration was almost 100% complete (United Nations 1976). As shown in Table 1a, the birth rate (births per 1000 females ages 15 to 45) increased from 179 to 202 between 1946 and 1953. We also use the number of births, combined with mortality data, to calculate the infant mortality rate. The 1946 infant mortality rate was 17 infant deaths per 100 live births, and by 1953 it had fallen by roughly 50%. Statistics on marriages are available every year by district and gender, and they include mean age at marriage and percent illiterate at marriage. Although the number of marriages is available for all ethnic groups, a consistent series on age and education is available only for civil marriages and not for Muslim or traditional Sinhalese (Kandyan) ones. The marriage data therefore cover only 76% of marriages.

Data on population, literacy, and student population are available from the census in 1946 and 1953. Literacy rates, which are for five-year age cohorts, by gender and district, are the main educational outcome we examine. An individual was considered literate if "the person is able to write a short letter and read the reply to it." As seen in Table 1a, literacy rates rise at young ages, which reflects the fact that a child's likelihood of becoming literate (necessarily) increases as she becomes older (age effects). Literacy rates then fall for older

[21] We collected data on the causes of death that were large in 1946 and for which consistent series could be obtained. These constitute 78% of all deaths in 1946. For selecting which diseases were most common for children, we use data from 1950, which is the earliest year with cause-specific mortality rates by age (for the country as a whole).

[22] The one exception is pyrexia which was a leading cause of death of school-age children. Pyrexia is a catch-all category: the decedent had a fever and the cause was otherwise unknown. We exclude pyrexia as a covariate because maternal mortality was frequently classified as pyrexia; puerperal pyrexia is one of the common causes of maternal death (De Silva 1943, Loudon 2000b, Deneux-Thauraux et al 2005).

ages, reflecting a secular increase in literacy across birth cohorts (cohort effects). The turning point is a good way to infer the ages at which people are on the margin of becoming literate, a piece of information that is important to our empirical analysis. The turning point for males is 25-29 years and, for females, 20-24 years, suggesting that people become literate up until age 25 or age 19. It may seem surprising that individuals are still becoming literate at age 19 (or even older), but in Sri Lanka, as well as most developing countries, a child might attend school for several years without becoming literate, and many children start school late and interrupt their schooling.

The empirical analysis defines a set of "treated" age cohorts—those who, as observed in 1953, are in the age range such that their literacy could and should have been affected by the MMR declines. Note that we are using *age* cohorts; we cannot (accurately) follow birth-year cohorts across the two censuses because of the 5-year age bins, combined with the 7-year gap between the 1946 and 1953 censuses. We use two alternative definitions of the treated cohorts: 5 to 19 years, and 5 to 24 years. (The latter grouping still assumes that literacy is determined by 19, but in the data we observe people several years after the treatment effect occurs.) On average for the 5-19 year old cohorts, male literacy was 58% in 1946 and 69% in 1953. For females, it was 44% in 1946 and 58% in 1953. Literacy is lower for females but it increased by 3 percentage points more than male literacy during 1946-53.

The percent in school variable is a proxy measure: it is the percent of individuals ages 5-24 who reported their occupation as "student." The census did not ask directly about school enrollment. The student population is not broken down by age; it is available as an aggregate count for 5 to 24-year-olds. The percent in school increased more for males than females, while, as seen above, literacy increased more for females. This pattern most likely reflects relative female gains in primary-school enrollment and relative male gains in secondary and post-secondary enrollment.

There are some variables that we would have liked to have used for which we do not have data. Years of education or other measures of completed schooling were unfortunately not recorded in the censuses. To measure how health investments respond to life expectancy, data on height or vaccination rates would have been valuable, but we have been unable to obtain them. Another limitation is that there are no further breakdowns of the data within districts, either geographically or by characteristics besides age and gender. Lastly, we

attempted to obtain district-level data on education and health services, such as the number of schools, or the number of hospitals and ambulances. These data, however, are not available for the period we study.

5. Empirical strategy

Our empirical strategy uses differential declines in maternal mortality across districts (and gender) in Sri Lanka as a source of variation in life expectancy. This approach is a difference-in-difference-in-difference (DDD). The first difference is over time, since maternal mortality fell between 1946 and 1953. The second difference is across geographic areas. The magnitude of the MMR declines varied considerably across Sri Lanka's 19 districts. The third difference is between genders; maternal mortality is quite unique among major causes of death in that it exclusively pertains to women.

With two time periods, the estimation is equivalently a DD where each observation measures the change in variables between the two time periods. Consider life expectancy, (denoted by *e*) as the dependent variable. The estimating equation is

$$\Delta e_{dg} = \beta_0 + \beta_1 \Delta MMR_d * female + \gamma_d + \nu_g + \varepsilon_{dg} \quad (5.1)$$

where *d* stands for district and *g* for gender. The coefficient of interest is β_1, which measures the effect of MMR, imposing the restriction that there is no effect of MMR on males (an assumption we discuss below). The specification includes district fixed effects (γ_d) and gender fixed effects (ν_g), which absorb the main effect of MMR and female, and ε_{dg} is a random disturbance term. The identifying assumption is that there are no unobserved district-gender specific changes that (1) are correlated with changes in maternal mortality in the district and (2) are correlated with district-gender specific changes in the outcome.

We examine two distinct types of outcomes: those that MMR affects mechanically, such as life expectancy; and those that we hypothesize MMR will have a causal effect on due to behavioral responses, such as educational attainment and fertility. For the mechanical effects, MMR should have immediate effects and thus the specification uses current MMR, while with behavioral outcomes, the correct specification will depend on how quickly people update beliefs and change their behavior. We allow for a three-year lag between the declines and when people respond, which seems like a plausible duration before behavior

would change.[23] We test the sensititivity of our results using other lags below. Another important reason for using a lag is to minimize the potential for reverse causality. Note that the 3-year lag also implies that the variation in MMR used excludes the 1946 spike.

We have age-specific literacy data, so we can examine whether MMR has effects specifically on age cohorts who during 1946-53 are in the age range when people become literate. Figure 4 lays out the timing of events and explains how one determines which age cohorts are the treated ones. Our time period is 1946 to 1953. Because we assume a three-year lagged response, the changes in MMR that began in 1946 could first affect behavior in 1949. The cohorts whose education could respond to the MMR declines, therefore, are those who were 19 years old or younger at some point between 1949 and 1953, assuming that 19 is the maximum age at which people become literate. These cohorts would be ages 23 and younger in 1953, the year for which we observe their outcomes. Data are in 5-year age cohorts such as 5-9, 10-14, and so on. We show results for two specifications: one where the treated cohorts are ages 5-24, and another where we use the more conservative age range of 5-19. One reason to not include ages 20-24 is that they would have been ages 13-17 in 1946. Their fertility rate would be low but non-zero, so there is a possibility of reverse causality, with their fertility decisions having an effect on the MMR regressor.

As a falsification test, we also verify that MMR has no effect on older cohorts whose literacy was already determined at the start of the study period. For the control group, we use ages 25 to 54.[24] Rather than estimating separate equations for treated and control groups, to improve efficiency we estimate a single regression, where the unit of observation is a district, gender, and 5-age category:

$$\begin{aligned} \Delta literacy_{adg} = \; & \beta_0 + \beta_1 \Delta LaggedMMR_d * female * TreatedAges_a + \\ & \beta_2 \Delta LaggedMMR_d * female * ControlAges_a + \lambda_{ad} + \theta_{ag} + \varepsilon_{adg} \end{aligned} \tag{5.2}$$

[23] A practical consideration is that our second observation is for 1953, so to observe effects of the steep MMR declines that occurred from 1946 to 1950, three years is the maximum behavioral lag for which our data allow us to observe the full effects. In principle, people could have anticipated the MMR decline in which case a lead would be appropriate, but it seems unlikely that the declines were anticipated.

[24] We exclude ages 55 and above since their literacy rates appear to be affected by selective mortality.

The specification includes fixed effects for treatment/control dummy*district (λ_{ad}), and treatment/control dummy*gender (θ_{ag}).[25] β_1 gives the effect of MMR on female-male literacy gaps for the treated cohorts. The hypothesis is that $\beta_1<0$ or that when MMR fell, female literacy saw relative increases. The coefficient β_2 is predicted to be 0, since MMR declines could not have affected these older cohorts' literacy. A significant coefficient for the older ages would imply that either there is reverse causality (if female education lowered MMR) or there were pre-existing trends in literacy correlated with MMR declines.

The birth rate regression is special because only women bear children, so we cannot use gender as a third difference. Instead we make comparisons across districts:[26]

$$\Delta birthrate_d = \beta_0 + \beta_1 \Delta LaggedMMR_d + \upsilon_d \tag{5.3}$$

We augment the regression with control variables that capture other changes in the district, but nevertheless, the estimates for fertility are more tentative because of the stronger identification assumptions needed.

6. Effect of MMR on life expectancy

This section quantifies the impact that MMR reductions had on female life expectancy. This exercise is useful later for interpreting the magnitudes of MMR's effect on behaviors. Examining life expectancy outcomes also allows us to test for omitted variable bias by examining whether MMR declines are correlated with relative female gains in life expectancy outside of childbearing ages.

Figure 5 previews graphically the regression results. We plot the difference in e(15-65) between men and women and MMR over time, by district. As MMR declined, e(15-65) of women relative to men rose (with a few exceptions). One can see that districts where the initial life expectancy disadvantage of women was the greatest were the districts with initially high maternal mortality (i.e., the correlation between MMR and the gender gap in

[25] We estimate coefficients for both treated and control groups; there is no omitted category for treated/control. District*gender fixed effects would be uncorrelated with the regressors of interest.

[26] Data for the birth rate and some other variables are available every year, but we still restrict attention to the two census years, 1946 and 1953. It seems less probable that high-frequency changes are perceived by people as permanent, so behavior is less likely to respond to such variation, especially if people respond with a lag. We also time-average data that are available annually to reduce measurement error, which rules out using high-frequency changes for identification.

e(0-65) holds in levels). In addition, the relative female gains in e(15-65) were larger where MMR declines were larger (i.e., the correlation holds in changes). Figure 6 shows additional evidence consistent with excess female mortality being mostly due to maternal deaths. The figure divides districts into those with above-median and below-median MMR in 1946, and plots the ratio of female to male death rates by age, and also the birth rate by age. (Data on births by age of mother are from 1952, the earliest year for such data.) Excess female mortality was highest at the ages when the birth rate was highest, particularly in the high-MMR districts. This is precisely what one would expect if maternal mortality were responsible for excess female mortality.

One can quantify the effect of MMR declines on e(15-65) using the standard demographic method, which consists of calculating what e(15-65) would have been if MMR declined by a given amount, but all other death rates remain unchanged. Maternal deaths declined by 67% from 1946 to 1953. We do not know the age distribution of these deaths, except that they occurred between ages 15 and 44, and we know that they accounted for 26% of deaths in 1946 for this age group. If one recomputes e(15-65) for 1946 with mortality rates for women ages 15 to 44 reduced by 17% (.67*.26), then one finds that declines in MMR increased female life expectancy by 1.4 years.

Another way to quantify the effect of maternal mortality on life expectancy is to estimate (5.1). The direct calculation above must make assumptions about MMR by age and about independence between MMR risk and other mortality risk, whereas the regression analysis does not need to invoke such assumptions. More importantly, regression analysis also allows us to probe the importance of omitted variables by testing for an "effect" of MMR on life expectancy outside of childbearing ages. We regress the change in life expectancy on ΔMMR*female and gender and district dummies. The results are reported in Table 2. The first column shows the results from the main specification. The effect of MMR on relative female e(15-65)—the life expectancy measure that MMR *should* affect —is negative and significant. When MMR fell, life expectancy rose. Since MMR fell by 1.3 percentage points between 1946 and 1953 (from 1.80 to 0.53), the estimate implies that MMR declines resulted in an increase in female life expectancy of 1.5 years. Reassuringly, this estimate is very close to the direct calculation of 1.4 years presented above. Female e(15-65) increased by 7.4 years over the period, so MMR declines can explain about 20% of

the increase in female life expectancy. Female e(15-65) increased by 0.8 years more than male e(15-65) from 1946 to 1953, which is a little over half of the female-male convergence predicted by maternal mortality declines. There seem to have been other factors causing relative male life expectancy gains, and absent them, MMR declines would have enabled women to catch up even more in terms of life expectancy.

The next rows show the effect of maternal mortality on other measures of life expectancy. The coefficient of ΔMMR*female on e(45-65) is close to zero and insignificant, and for e(0-15) it is marginally significant but small (about 5 percent of the change in female e(0-15) during the period). Both of these age ranges are outside of the primary childbearing ages, so large negative and significant effects would have suggested other female-specific health gains that were correlated with MMR declines.

Finally, we examine the correlation between MMR and the infant mortality rate (IMR). There are at least two distinct reasons that IMR might be correlated with MMR. First, health programs for mothers and children plus the malaria eradication efforts that contributed to MMR declines are likely to have improved IMR as well. Second, maternal mortality could have a causal effect on infant mortality, since motherless infants may be more at risk. The primary concern for our identification strategy is a correlation between MMR and *gender differentials* in IMR. Nevertheless, it is helpful to begin by discussing the correlation between MMR and total IMR. In a regression of changes in IMR on changes in MMR (N=19), with both variables measured in percentage points, the coefficient is 3.73 and statistically significant at the 1% level. A 1 point reduction in MMR is associated with a 3.7 point reduction in IMR. A back-of-the envelope calculation suggests that the upper bound on what the *causal* effect of MMR on IMR could be is 0.8, implying that much of the correlation between MMR and IMR is due to third factors such as health care improvements. This calculation uses the upper bound in the literature that IMR could be as much as 6 times higher for motherless infants as for infants with mothers (Loudon 1991).[27]

Again note, though, that the key issue is whether health programs had gender-specific effects, and whether maternal mortality presents a higher mortality risk for female

[27] A 1 point decline in MMR would decrease the number of motherless infants by about 1 percentage point. One can use the IMR ratio of 6 to back out the initial-period IMR for those orphaned due to MMR (93.6%) and non-orphans (15.6%) that gives back the population-average IMR, and then simulate the change in IMR from a 1 point decline in the proportion of orphans.

infants. Table 2 shows the effect of MMR on IMR for girls relative to boys. The coefficient on ΔMMR*female of 0.13 is small and insignificant.[28]

One potential threat to the validity of the research design is that declines in MMR could be correlated with other health improvements that affected outcomes such as literacy. For example, expansion of maternal and child health programs are one reason that MMR declined, and a concern is that these programs directly improved child health and, in turn, education. The strength of the identification strategy, in this regard, is that such improvements likely helped both boys and girls, and we identify MMR effects based on differential improvements for girls. Another potential confounding factor is the successful malaria control program during the period. There also were two other major programs that we are aware of that targeted health: nutrition programs, such as free milk distribution, and intestinal worm eradication. The concern is that these programs reduced morbidity among children, encouraging school attendance. We address these concerns by controlling for gender-specific malaria and nutrition-related death rates (we refer to anemia, diarrhea, vitamin deficiencies and helminths jointly as nutritional diseases hereafter; our controls include death rates for each of these diseases separately).

These controls address potential omitted variable bias, but they are not our preferred specification for two reasons. First, the control variables could be endogenous in some specifications. For example, school enrollment rates could determine nutrition-related diseases since the government provided food in school. Second, we could be over-controlling by including these diseases because malaria and nutrition deficiencies could increase the likelihood of maternal deaths. Nonetheless, we view the results with these controls as useful checks. As seen in columns 2 to 4 of Table 2, the controls do not have an appreciable impact on the coefficients for e(15-65). The coefficients for e(0-15) and IMR are somewhat more sensitive to the inclusion of the control variables, which is sensible since these diseases were chosen because they are prevalent among children.

More detailed results are presented in Appendix Table 2, which examines the change in age-specific death rates and shows the coefficient for ΔMMR*female. MMR is positively and significantly associated with death rates for ages 15-19, 20-24, 25-29 and 30-34 year-

[28] The 0.13 percentage point decrease in relative female IMR that is associated with a 1 percentage point decline in MMR represents less than a 1% effect.

olds, with the largest effects at age 20-24, which is consistent with birth rate patterns. The effects on deaths rates below age 15 are sometimes statistically significant, but the magnitudes are small. The results for ages 35 are small and are insignificant except for one case.

Maternal mortality resulted in an increase in female e(15-65) of 1.5 years between 1946 and 1953. We now turn to the effects of MMR on behaviors that should respond to increased life expectancy such as educational investment and fertility.

7. Effect of MMR on literacy

The main results of the paper are on the effect of MMR on literacy. As explained in Section 5, the age cohorts whose literacy should be affected by the 1946-53 MMR declines are ages 5-24 or 5-19 years old. Figure 7 shows the identifying variation graphically. We plot the female-male gap in literacy for the treated cohorts (using the ages 5-19 definition) and MMR for each district. The prediction we test is that the female-male literacy gap narrowed more in districts where MMR fell by more.

Table 3 presents the regression results. Panel A uses the age 5-19 definition of treated cohorts, and panel B uses the age 5-24 definition. Column 1 of panel A presents the basic regression results with no covariates besides age-district and age-gender fixed effects. Each observation is a district-gender-5 year age group, and standard errors are clustered at the district-gender level. We use the 3-year lag of MMR to allow for a delay before people notice the mortality decline and adjust their behavior. The coefficient on Δ(lagged MMR)* female is -0.87. As predicted, declines in MMR (health improvements) increased female literacy. The p-value for the coefficient is .06. The coefficient on the control cohorts, which is hypothesized to be 0, is statistically insignificant and positive. For the corresponding specification in panel B, the effect of MMR on literacy is -1.05 for the treated cohorts, with a p-value of .02. The control cohorts are the same in Panels A and B, and the model is fully interacted with respect to the treatment/control dummies (each is interacted with gender and with district), so the coefficient for the control cohorts is the same across panels.

To be conservative, we use the specification in which ages 5-19 are the treatment group when interpreting the effect size. The coefficient of -0.87 means that the decline by 1.3 in MMR led to an increase in relative female literacy of 1.1 percentage point. The

female-male gap literacy gap among 5-19 year olds was 14.3 percentage points in 1946 and narrowed to 10.9 percentage points by 1953. MMR declines explain a third of the relative gains that girls made over the period.

The results can also be interpreted as an elasticity. Female literacy among 5-19 year olds was 44% in 1946. Therefore, MMR declines led to a 2.5% increase in female literacy. As shown in the previous section, MMR led to a 1.5 year or 4.1% increase in female e(15-65), the life expectancy measure that proxies for the years over which returns to education are earned. Dividing the two numbers gives an elasticity of literacy with respect to e(15-65) of 0.6.[29]

Specification checks

The final three columns of Table 3 present robustness checks. Column 2 controls for nutritional diseases and malaria. The coefficient on ΔMMR*female for the treated cohorts becomes more negative but less precise. For reasons laid out earlier, the specification without the covariates is our preferred specification. Column 3 presents the basic specification but the control cohorts are narrowed to 25-44 year olds, which might represent a more comparable control group and a better group with which to test for pre-trends. Here the coefficient for the control group is essentially 0. (The coefficient on the treated group is unchanged from column 1, again because of the saturated model.)

Finally, column 4 drops the district fixed effects and controls for male e(0-65) interacted with the treatment-cohort and control-cohort dummies. The purpose of this specification is less as a robustness test of the literacy results and more as a way to shed light on the validity of the fertility estimates presented later. To the extent that controlling for male e(0-65) versus district fixed effects gives similar results for literacy, this increases confidence about controlling for male e(0-65) as a way of arriving at causal estimates of the effect of MMR on the birth rate, where district fixed effects are impossible to include.

[29] One can do a very rough translation of the estimated 1 percentage point increase in female literacy into years of schooling. In the 1987 DHS, the average education of literate women is 7.8 years and the average education of illiterate women is 1.6 years (excluding the young cohorts who were educated much after the study period). On the one hand, some increases in schooling will be inframarginal to literacy, and on the other hand, tiny increases in education can tip those on the margin to become literate, so the bounds on the changes in years of education are large. Nonetheless, as an approximation, if the 1 percentage point increase in literacy corresponded to that proportion of the population gaining 6.2 years of schooling (7.8 minus 1.2), the implied increase in (relative) female education is 0.06 years from a base of 4.8 years, or 1.3%.

Comparing columns 1 and 4, one sees that the precision falls considerably. In panel A, the coefficient becomes about 50% larger, while the coefficient is more stable in panel B. Controlling for male e(0-65) seems to do a good but not excellent job of absorbing district-level omitted factors that affect literacy.

Table 4 presents the results separately estimating coefficients for each 5-year age group. The coefficients are much less precise, but the patterns are similar. MMR declines are associated with female literacy gains for young cohorts but not older cohorts. Table 5 presents other robustness checks. It shows, first, that the standard errors are unchanged if we cluster on treated cohort-gender-district instead of gender-district, and second, that weighting by population gives a larger but imprecise coefficient. It also shows the results for different lags where the MMR regressor is a single-year measure, rather than the 3-year average. In Appendix Table 5 we test for the influence of outlier districts, re-running the basic specification dropping one district (out of 19) at a time. Not surprisingly given the relatively small sample size, the coefficients fluctuate, but the results are not overly sensitive to any particular district. Overall these specification checks confirm our main results.

Other potential threats to validity

One might worry that less advanced districts catch up on all fronts over time, and the process of development entails both health improvements and progress in girls' education. Districts with initially higher maternal mortality risk experienced bigger MMR declines over the period, as shown earlier (Figure 2). We can test then whether gender gaps in education are initially larger in places with more maternal mortality. If this were the case, and if districts naturally tend to catch up over time with respect to gender gaps in education, then this might generate a spurious correlation between MMR and female education. Figure 8 shows, however, that initial MMR and the initial gender gap in literacy in fact have a very weak correlation. While one cannot directly test the DDD identification assumption, which is about changes over time in these measures, this finding is reassuring: the fact that high-MMR districts were not laggards in terms of girl's education suggests that the results are not simply driven by the fact that poor places catch up on all dimensions.

In addition, Sri Lanka as a whole underwent other changes during the study period, e.g., the end of colonialism. Arguably, education-related policies, such as the elimination of

school fees, are of greatest concern since education is the outcome we examine. There were no major gender-specific or district-specific educational policy changes that we are aware of during the period. If the national education policies had gender- or district-level effects, these would not bias our estimates, which use district-gender variation. The identification only would be threatened if national policies had gender-specific effects that varied at the district level in a manner correlated with MMR.

Another concern is that the death of a mother might differentially affect girls' education. Mothers might be more "pro-daughter" than fathers. Or girls might be a closer substitute for a deceased mother's home labor, so maternal death might cause girls, more than boys, to drop out of school to work in the home. Under this alternative, MMR has a causal effect on girls' education, but one different from the incentive effect that we focus on that arises from girls' rising life expectancy. The concern is an important one, and what assuages it in practice is a quantitative exercise showing that it is unlikely to account for the empirical results. Consider the following rough calculation. Relative to 1946, the risk of a mother dying per childbirth was on average 1.0 percentage point lower during the subsequent seven years up to 1953. The average number of births per woman during the seven-year interval was 1.2 births (17% fertility rate per year). Thus, the likelihood of a child being motherless fell by about 1.2 percentage points (1.2*1.0). Consider the extreme scenario where every girl whose mother had died has 0% chance of becoming literate while boys' literacy is unaffected by maternal death. The population average literacy for the relevant cohorts of girls was 44%, so in this stark scenario, girls' literacy would increase by 0.5 percentage points (.44*1.2). Our estimate of the increase in girl's literacy caused by the 1946-53 MMR declines is over twice this magnitude, so even under the starkest assumptions—no effect on boys and every motherless girl is illiterate—the effects of orphanhood cannot explain our estimated effect size.

The potential for spillovers of MMR raises the question of whether males are a valid control group. Our assumption is that MMR has a direct effect on only female life expectancy and therefore schooling. However, MMR could have an indirect effect on males if their schooling is affected by their siblings' or future spouses' life expectancy or schooling, for example. One can imagine these spillovers being either positive or negative. For example, if men prefer to be as educated as their wives, then an increase in girls'

education might induce an increase in boys' education. Conversely, spillovers within the family might be negative if a family faces a credit constraint. Higher returns to education for girls might shift resources away from boys' education. Unfortunately, we cannot empirically assess the relative magnitude of direct versus spillovers effects, but spillovers on boys seem unlikely to be of the same order of magnitude as the direct effects on girls.

In addition, the underlying behavioral model has people rationally updating their beliefs about life expectancy. This assumption is not unique to our study; it is a standard assumption in the literature. If this were a wrong assumption, however, then it could be one explanation for null results. If we found that people do not change their behaviors in response to MMR declines, we would not be able to distinguish between, on the one hand, a zero elasticity of behavior with respect to subjective life expectancy and, on the other hand, subjective life expectancy not tracking objective life expectancy. We in fact do not find null results, making this concern somewhat less forceful. When calculating the elasticity of behavior with respect to life expectancy, one needs to make the more bold assumption that individuals get the *magnitudes* of life expectancy gains exactly correct. The calculations are still valid as an elasticity with respect to objective life expectancy, but interpreting them as with respect to subjective life expectancy requires stronger assumptions.

In sum, our estimates indicate that MMR declines caused girls to obtain more education. The 1 percentage point increase in female literacy attributable to MMR declines translates into a 0.6 elasticity of literacy with respect to e(15-65). Human capital investment seems quite responsive to life expectancy.

8. Effect of MMR on other behaviors

Fertility

We next test the theoretical prediction that when the risk of dying in childbirth falls, the propensity to have children increases. Because we cannot make use of gender as a third difference, we estimate a difference-in-difference model where there is a larger concern of omitted variable bias. However, one variable that might capture much of the potentially confounding district-specific factors is male life expectancy. Maternal mortality should not have a causal effect on male life expectancy (except through infant mortality as discussed above), so a correlation between MMR and male life expectancy is likely driven by

unobserved factors such as overall health improvements.[30] Using male e(0-65) as a control variable seems promising because it gets close to the female-male comparison that one can use with dependent variables, such as life expectancy or literacy, that permit the DDD design.

The fertility results are in the first row of Table 6.[31] Column 1 controls for male e(0-65), or life expectancy at birth censored at 65. The magnitude of -5 for the coefficient on ΔMMR implies that the decline of 1.3 in MMR over the period caused the birth rate (births per 1000 women age 15-45) to increase by 6.5 from a base of 178.9. In other words, the birth rate seems to have increased by 4% in response to the 67% decrease in MMR. Column 2 shows that the estimates are stable when malaria and nutritional diseases are added as covariates. The point estimates are also stable when IMR (total for males and females) is included (Column 3), though the coefficient is no longer statistically significant.

We used IMR as a control variable because it could affect the birth rate. As discussed in Section 6, IMR declines (total, not gender-specific) indeed were correlated with MMR declines. On the one hand, the fact that IMR is a confounding factor makes us cautious in interpreting the fertility results. On the other hand, the fact that, conditional on e(0-65) for males, the results are robust to the inclusion of other covariates gives some reassurance about the results. Thus, we interpret the results as providing suggestive evidence that when the risk of dying in childbirth decreased, individuals responded by increasing their fertility. The birth rate increased by 13% between 1946 and 1953, and lower MMR seems to account for one third of the increase.

Percent in school, percent illiterate at marriage, and age at marriage

Table 7 examines two other human capital measures and also age at marriage. In the first row, the dependent variable is the percent in school among those ages 5 to 24. (More precisely, the variable is the percent who report student as their occupation.) ΔMMR*female has a negative effect that is marginally significant. The coefficient of -0.9, given the 1.3 point decline in MMR, suggests that the MMR decline caused the percent in school to increase disproportionately for females by 1.2 percentage points. In 1946, 34.6 out of 100

[30] If we regress changes in e(0-65) for males on changes in maternal mortality (19 observations), the coefficient on maternal mortality is -2.1 and significant at the 1% level.

[31] MMR could be affecting either the numerator or the denominator of the birth rate; the next two rows present results separately for log of births and log of female population as the dependent variables.

girls were students, so the effect size corresponds to a 3.4% increase, somewhat larger than the 2.5% increase in female literacy presented above. When disease rates are added as covariates, the coefficient becomes insignificant, although its magnitude remains similar.

The second human capital measure presented in Table 7 is the percent illiterate at marriage. The coefficient on ΔMMR*female has the expected positive sign (when MMR falls, the percent illiterate at marriage falls disproportionately for females) but it is insignificant. The point estimate in column 1 suggests that the decline in MMR by 1.3 caused the percent illiterate to fall by 1.9 percentage points or 7%. The estimates using percent illiterate at marriage are imprecise probably partly because reporting of marriages is incomplete, so the data are of lower quality than the births, deaths, and literacy data. Also, sample selection is a potential problem since this measure of literacy is conditional on marriage.

Finally, we examine age at marriage. As mentioned in Section 2, it is theoretically ambiguous whether there will be a positive or negative effect. The estimated coefficient is small and positive and essentially 0. Based on the coefficient of 0.015 in column 1, the MMR decline could account for an increase in the female age of marriage of only 0.02 years. Moreover, the coefficient becomes negative (but still small) with covariates added.

9. Conclusion

Over the past fifty years, longevity has improved dramatically, particularly in developing countries. During this period, the welfare gains from longer life expectancy were comparable to the welfare gains as traditionally measured by income per capita, and moreover, there was convergence in longevity, with poor countries beginning to catch up to rich countries (Becker, Philipson, and Soares 2005).

Besides being welfare-improving per se, longevity gains could spur human capital accumulation and growth. A longer horizon gives stronger incentives to obtain schooling and undertake other investments. This paper presented empirical evidence in support of the hypothesis that life expectancy gains lead to higher human capital. We identified these effects using changes in maternal mortality, a cause of death that is particularly well-suited to isolating this life-expectancy channel from other effects that health has on education.

We examined a period of rapid MMR decline in Sri Lanka. MMR fell by 70% (from 1.8 to 0.5 deaths per 100 births) between 1946 and 1953, causing female adult life expectancy to increase by 1.5 years. We used geographic variation in this decline to estimate the corresponding change in human capital, as measured by literacy. The MMR decline caused female literacy to increase by 1 percentage point (relative to changes in male literacy), which represents a 2.5% increase. This change resulted from a 4.1% increase in female life expectancy at age 15 (censored at 65). These estimates imply an elasticity of literacy with respect to life expectancy of 0.6. Human capital investment appears to be quite responsive to lifespan. We also found suggestive evidence that MMR declines caused fertility to increase, which is as predicted, both because childbearing became less risky, and because having a child is probably more valued when the child (daughter) is expected to live longer.

While Sri Lanka made great strides against maternal mortality 60 years ago, maternal mortality continues to be an important health risk in most developing countries. MMR today is on average 0.4 maternal deaths per 100 births in developing countries, with several countries, mainly in sub-Saharan Africa, facing rates over 1 maternal death per 100 births. But the findings of this paper speak to improvements in life expectancy more broadly, for example those that might accrue if a vaccine against tuberculosis or malaria were able to eliminate those diseases. The sizeable effects we find suggest that the extra benefit of higher human capital accumulation from life expectancy gains is an important component of cost-benefit analyses of such public health interventions. Of course the reasoning also works in reverse, and suggests that recent declines in life expectancy in many sub-Saharan African countries, brought about by HIV/AIDS and civil war, have an important additional deleterious effect of dampening the incentive to invest.

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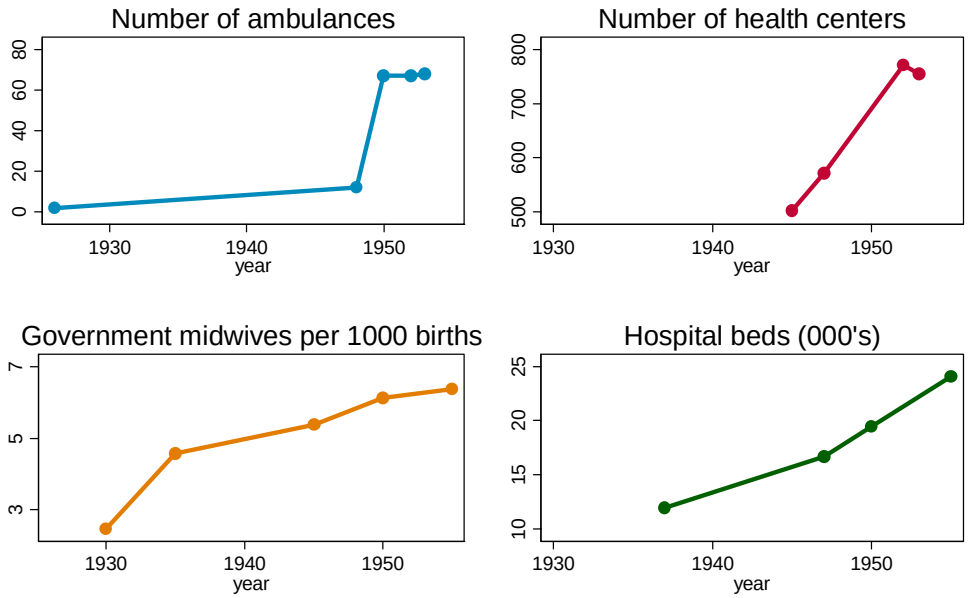
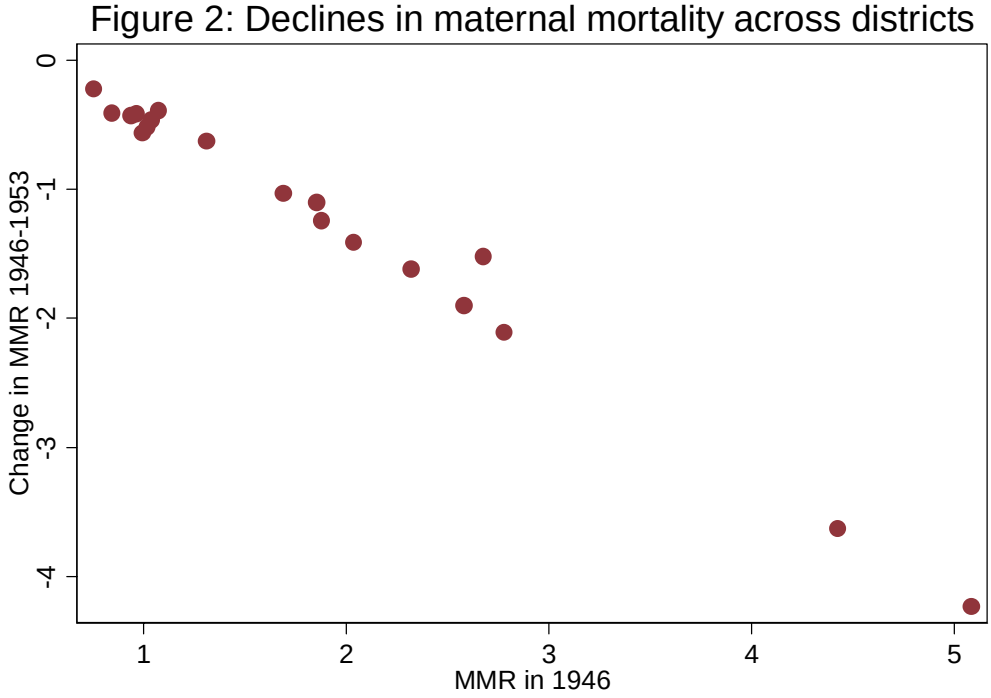
Figure 1: Expansion of health services in Sri Lanka



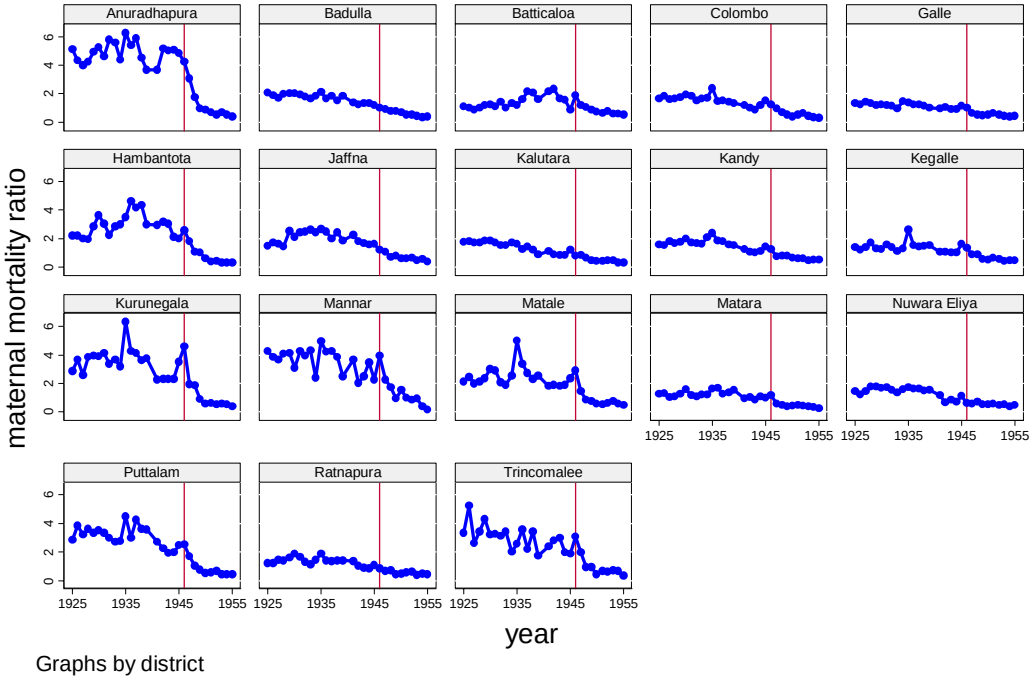
Sources: Data on ambulances, health centers and hospital beds are from the report of the Director of Health Services Administration Reports (various years). Data on government midwives are from World Bank (2003).

Figure 2: Declines in maternal mortality across districts



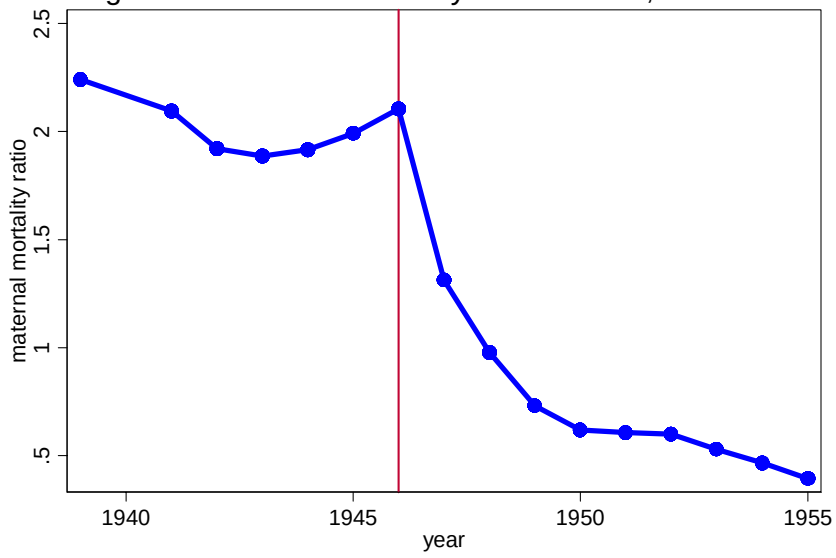
Note: Each dot represents a district. Maternal mortality is the number of deaths per 100 live births. In a univariate regression of maternal mortality changes between 1946 and 1953 on in the initial 1946 level, the coefficient on initial MMR is -0.70 and is statistically significant at the 5% level.

Figure 3a: Maternal mortality by district, 1925-1955



Note: Maternal mortality is the number of deaths per 100 live births. Vavuniya is excluded because of scale: due to small numbers, its female/male ratio is noisy and takes on large values. When plotting all districts on the same scale, including Vavuniya makes the patterns difficult to observe.

Figure 3b: Maternal mortality for Sri Lanka, 1939-1955



Note: Figure reports unweighted averages across districts. Maternal mortality is the number of deaths per 100 live births.

Figure 4
Cohorts affected by MMR declines

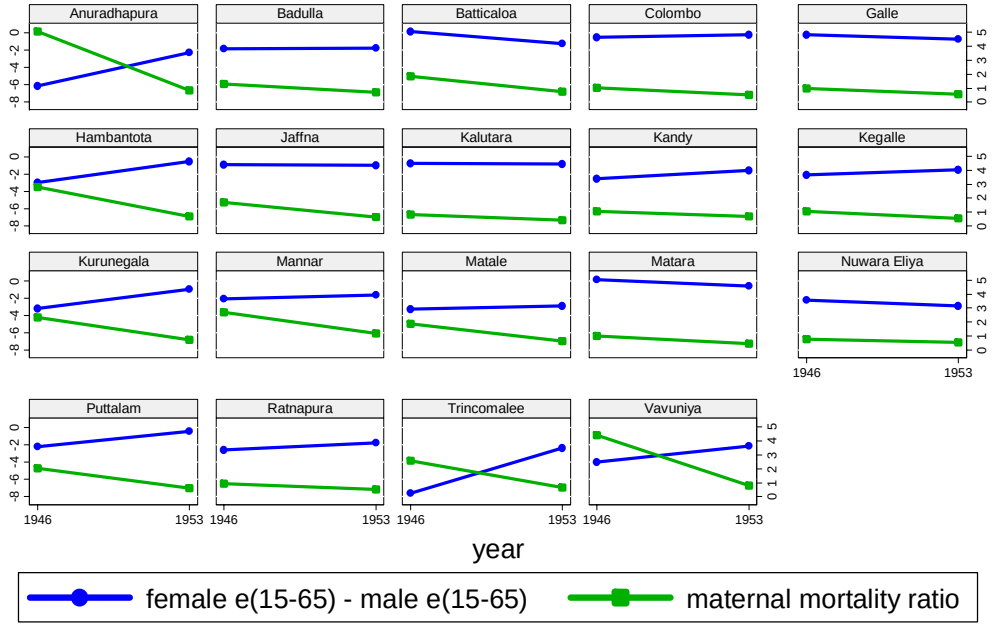
Year	**1946**	**1949**		**1953**
	MMR declines begin	Behavior assumed to respond to MMR declines with 3-year lag		Outcomes observed
Ages for whom literacy could be affected by MMR declines		≤Age 19	→	≤Age 23
Ages that were not childbearing in 1946	≤Age 15		→	≤Age 22
Treated age cohorts				**≤22 to 23**

Notes: This figure lays out the criteria for determining which age cohorts comprise the treatment group, that is, the age groups, *as observed in 1953*, whose literacy is hypothesized to be affected by the MMR declines.

(a) The timeline lays out the 3 key dates: the start date of MMR declines (1946), the year in which behavioral responses would begin, assuming a 3-year lag (1949), and the date for which we have outcome data (1953).
(b) The first criterion for inclusion as a treated cohort is that at the date when behavioral responses begin (1949), the cohort was young enough to be on the margin of becoming literate; we assume that no one become literate after age 19. Therefore, as observed in 1953, age cohorts 23 and younger are treated.
(c) The second criterion is that the cohort was not childbearing at the time of the MMR declines. The purpose is to avoid reverse causality (literacy affecting MMR).* The cohorts who meet the criterion are those no older than 15 in 1946. As observed in 1953, these cohorts are those younger than 22.
(d) Combining the two criteria implies that the maximum age for the treated cohorts is 22 or 23 years old. Therefore, we use ages 5-24 or ages 5-19 as 2 alternative definitions of the treated group. The data are not disaggregated beyond 5-year age categories.

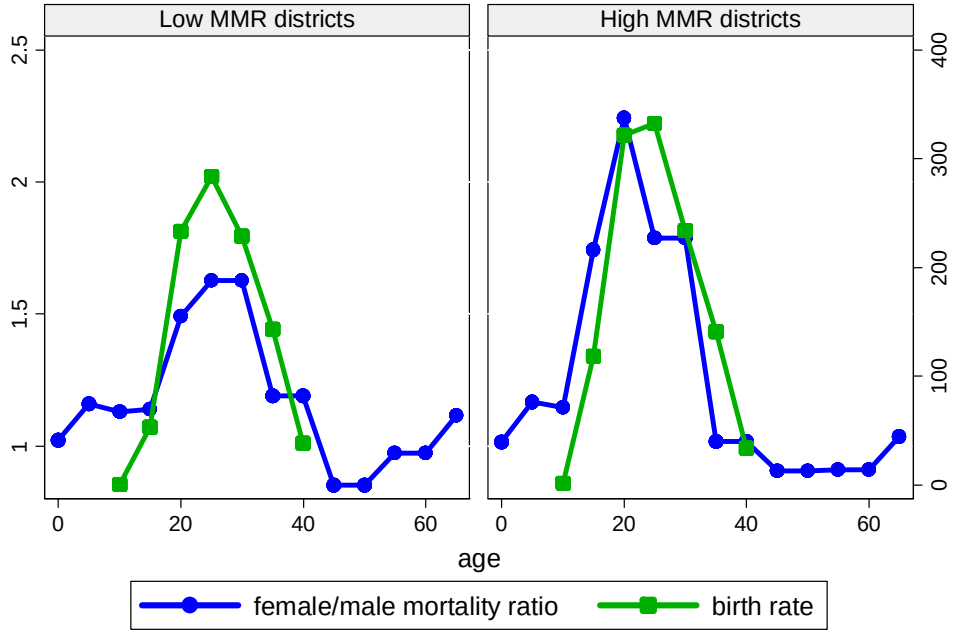
* If we omitted cohorts who were of childbearing age when the MMR declines finished in 1950, the cutoff rule in 1953 would be <age 18. We excluded this nuance from the figure because, first, there was in fact, very little childbearing even at age 16 and, more importantly, given the 5-year-aggregated data, the age range chosen for the treated group would not change.

Figure 5: Changes in female-male life expectancy and MMR



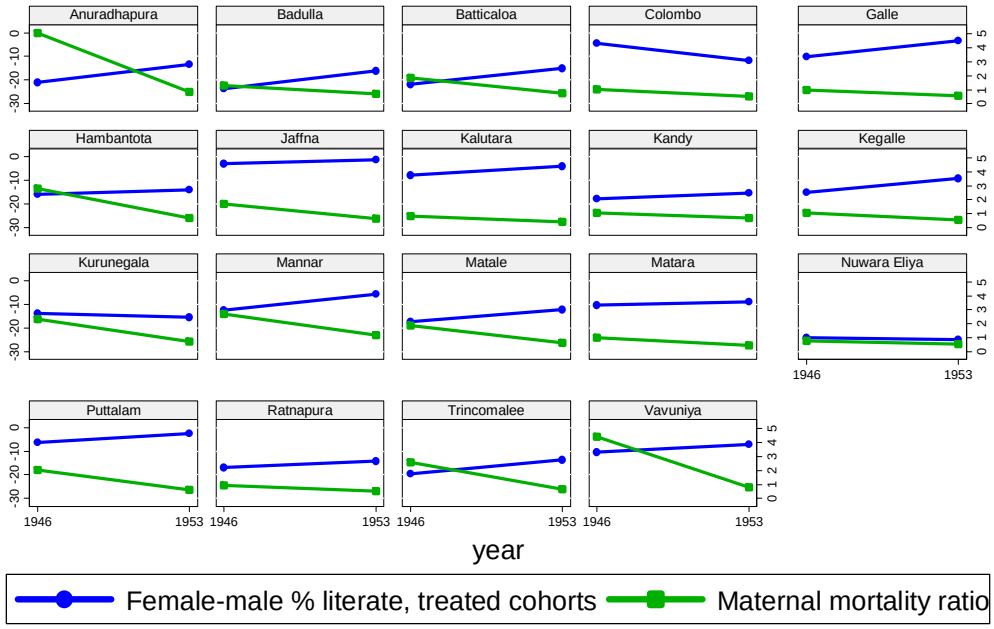
Note: Maternal mortality is the number of deaths per 100 live births. e(15-65) is life expectancy at age 15, censored at age 65.

Figure 6: Birth rate and excess female mortality by age



Note: Birth rate is measured in number of births per 1000 women ages 15-45.

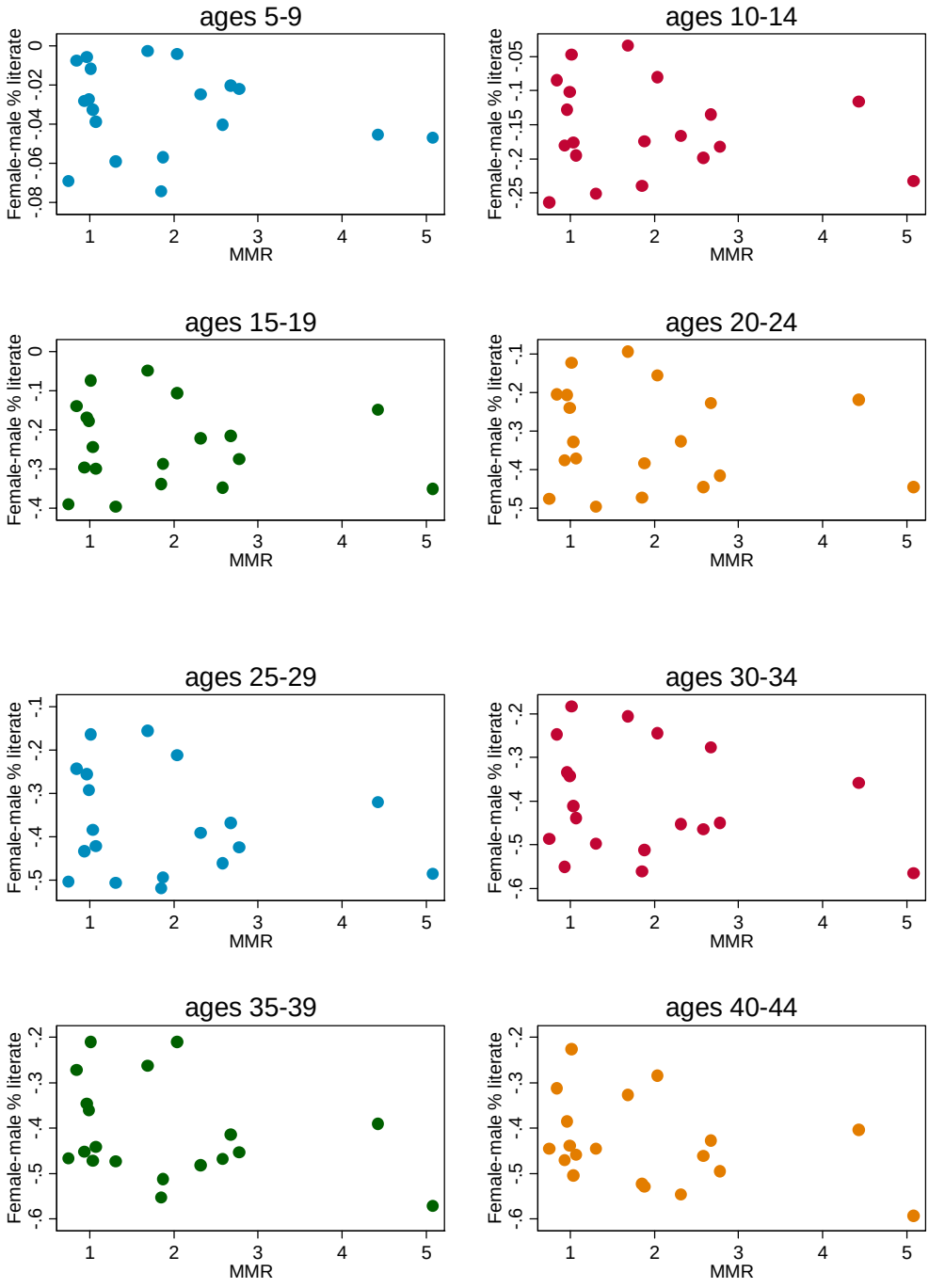
Figure 7: Changes in female-male literacy and changes in MMR



Graphs by district

Note: Maternal mortality is the number of deaths per 100 live births. Female-male % literate is % literate among females minus % literate among males.

Figure 8: Relationship between maternal mortality and literacy gender gaps in 1946



Each dot represents a district.

Note: MMR is the number of deaths per 100 live births. Female-male % literate is % literate among females minus % literate among males. Both MMR and literacy differences are measured in levels for 1946.

Table 1a: Summary Statistics

	Males		Females	
	1946	1953	1946	1953
Mortality and Life expectancy				
Maternal mortality ratio (current)*			1.80	0.53
Maternal mortality ratio (lagged 3 yrs)			1.91	0.65
e(15-65)**	39.1	45.7	36.8	44.2
e(45-65)	15.7	18.2	16.1	18.3
e(0-15)	10.9	12.8	10.8	12.9
e(0-65)	37.12	50.26	35.27	49.37
Infant mortality rate (current)*	17.7	8.1	16.6	6.8
Fertility* **				
Birth rate			178.9	202.4
Log(Number of births)			9.11	9.39
Log(Female population 15-45)			10.83	11.02
Marriage				
Mean age at marriage	27.8	27.8	21.3	21.6
% illiterate at marriage	6.4	6.8	26.8	23.7
Education				
% literate				
Treated cohorts: ages 5-19	58.17	69.35	43.88	58.43
Alternative treated cohorts: ages 5-24	64.13	73.42	45.50	58.83
Control cohorts: ages 25-54	76.04	79.78	34.13	41.67
Age 5-9	28.43	44.62	25.17	41.82
Age 10-14	67.55	81.06	51.81	69.26
Age 15-19	78.55	82.37	54.65	64.21
Age 20-24	81.99	85.63	50.36	60.03
Age 25-29	81.83	85.36	44.78	54.99
Age 30-34	80.06	84.37	40.14	49.38
Age 35-39	77.20	80.83	36.04	42.74
Age 40-44	75.07	78.71	31.52	37.76
Age 45-49	72.20	75.50	27.79	34.31
Age 50-54	69.91	73.93	24.53	30.86
Age 55-59	69.28	70.19	23.36	27.67
Age 60-64	64.65	67.64	19.53	23.13
% in school (ages 5-24)****	36.30	44.80	34.60	39.71

Notes: N=19 districts. Maternal mortality, life expectancy, fertility and marriage statistics are 3-year averages centered on 1946 and 1953 (e.g. the 1946 birth rate is the average for 1945, 1946 and 1947).

* Maternal mortality ratio and infant mortality rate are number of deaths per 100 live births.

** e(15-65) denotes the expected years of life between ages 15 and 65, conditional on surviving until age 15, and so forth. Note that life expectancy, because it involves conditioning on survival to a certain age is not additive, so e(0-65) is not the sum of e(0-15) and e(15-65).

*** Birth rate=[births/female pop ages 15-45]*1,000. Number of births and female population are in units of 1000 before taking the logarithm.

**** % in school is percent who report their occupation as student.

Table 1b: Summary Statistics
Death rates by disease and by age

Year	1946	1953	1946	1953
	Males		Females	
Disease mortality rates				
Vitamin	0.99	0.58	1.51	0.79
Malaria	1.44	0.10	1.64	0.12
Diarrhea	0.91	0.39	0.98	0.45
Helminths	0.35	0.32	0.46	0.45
Anemia	0.37	0.21	0.36	0.25
Age-specific death rates				
ages 0-4	72.75	34.92	74.20	33.31
ages 5-9	6.75	3.04	7.72	3.48
ages 10-14	3.57	1.46	4.20	1.56
ages 15-19	5.10	1.68	8.01	2.67
ages 20-24	6.58	2.16	12.87	3.94
ages 25-29	8.19	2.55	13.44	4.91
ages 30-34	8.19	2.78	13.44	5.10
ages 35-39	13.13	3.67	13.44	5.48
ages 40-44	13.13	4.58	13.44	5.44
ages 45-49	21.55	6.42	19.29	6.50
ages 50-54	21.55	9.29	19.29	8.46
ages 55-59	39.78	13.92	35.20	11.91
ages 60-64	39.78	20.68	35.20	19.09
ages 65+	99.46	68.69	107.22	76.96

Notes: Disease-specific and cause-specific rates are per 1000. The 1946 data are available only by 10 year age groups for ages 25-34, 45-54 and 55-64. All statistics in this table are averages over three years, centered on 1946 and 1953 (e.g. the 1946 death rate is the average for 1945, 1946 and 1947).

Table 2: Effect of maternal mortality on life expectancy and infant mortality
Difference-in-difference-in-difference estimates

	(1)	(2)	(3)	(4)
Dependent variable:	Basic	Add malaria death rates	Add nutritional diseases death rates	Add nutritional diseases and malaria death rates
Δe(15-65)				
ΔMMR*female	-1.204***	-1.302***	-1.135***	-1.369***
	[0.198]	[0.307]	[0.181]	[0.444]
R-squared	.97	.97	.97	.97
Δe(45-65)				
ΔMMR*female	0.054	-0.033	0.115	-0.041
	[0.089]	[0.120]	[0.090]	[0.204]
R-squared	0.94	0.95	0.94	0.95
Δe(0-15)				
ΔMMR*female	-0.088*	-0.081	-0.072**	-0.022
	[0.050]	[0.065]	[0.031]	[0.050]
R-squared	0.99	0.99	1	1
ΔIMR				
ΔMMR*female	0.133	0.081	0.306*	0.228
	[0.164]	[0.192]	[0.159]	[0.247]
R-squared	0.99	0.99	0.99	0.99

Variables are measured in changes between 1946 and 1953. All regressions include district and gender fixed effects. Additional controls are measured in changes. The notation e(15-65) is the expected years of life between ages 15 and 65, conditional on surviving until age 15, and so forth. MMR is the maternal mortality ratio, and IMR is the infant mortality rate. Both are measured as deaths per 100 live births. Nutritional diseases are helminths, anemia, diarrhea and vitamin deficiencies. Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. N=38 (19 districts, 2 genders).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 3: Effect of maternal mortality on literacy
Difference-in-difference-in-difference estimates

	(1)	(2)	(3)	(4)
	Basic	Add nutritional diseases and malaria death rates	Drop ages 45-54 from control group	Drop district FE & control for male e(0-65)
Panel A:				
Ages 5-19 as treated				
Ages 25-54 as control				
Δ (lagged MMR)	-0.873*	-1.617**	-0.873*	-1.408
*female*treated	[0.446]	[0.721]	[0.455]	[1.682]
Δ(lagged MMR)	0.329	0.620	-0.035	-0.072
*female*control	[0.471]	[0.563]	[0.453]	[1.189]
Observations	342	342	266	342
R-squared	0.54	0.56	0.56	0.32
Panel B:				
Ages 5-24 as treated				
Ages 25-54 as control				
Δ (lagged MMR)	-1.051**	-1.869***	-1.051**	-1.342
*female*treated	[0.446]	[0.549]	[0.453]	[1.531]
Δ(lagged MMR)	0.329	0.620	-0.035	-0.072
*female*control	[0.468]	[0.558]	[0.448]	[1.188]
Observations	380	380	304	380
R-squared	0.46	0.48	0.48	0.26

Variables are measured in changes between 1946 and 1953. MMR is lagged by 3 years. All regressions include district-treated, gender-treated, district-control and gender-control fixed effects. Treated is a dummy for being age 5-19 in Panel A or age 5-24 in Panel B. Control is a dummy for being age 25-54 in columns 1, 2, and 4 or age 25-44 in column 3. All observations in the regressions are either treated=1 or control=1. Additional controls are measured in changes. Nutritional diseases are helminths, anemia, diarrhea and vitamin deficiencies. These diseases are district- and gender-specific, and they are interacted with treated and control when they are included. Standard errors clustered within a district-gender are reported in brackets. Each observation is a district-gender-5-year age group (19 districts, 2 gender, 9 age groups in Panel A, column 1, but varies by panel and column).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 4: Age-specific effects of maternal mortality on literacy
Difference-in-difference-in-difference estimates

	(1)	(2)
Dependent variable: % literate by age	Basic	Add nutritional diseases and malaria
Ages 5-9*		
Δ(lagged MMR)*female	-0.499	-1.318
	[0.648]	[1.339]
Ages 10-14*		
Δ(lagged MMR)*female	-0.768	-1.806
	[0.852]	[1.526]
Ages 15-19*		
Δ(lagged MMR)*female	-1.352	-1.728
	[0.976]	[1.385]
Ages 20-24*		
Δ(lagged MMR)*female	-1.586	-2.625***
	[0.947]	[0.948]
Ages 25-29*		
Δ(lagged MMR)*female	-1.224	0.417
	[0.726]	[0.954]
Ages 30-34*		
Δ(lagged MMR)*female	-0.844	-0.331
	[0.815]	[1.309]
Ages 35-39*		
Δ(lagged MMR)*female	0.062	0.159
	[0.872]	[0.810]
Ages 40-44*		
Δ(lagged MMR)*female	1.865*	1.864
	[1.072]	[1.160]
Ages 45-49*		
Δ(lagged MMR)*female	1.424	0.873
	[1.145]	[1.169]
Ages 50-54*		
Δ(lagged MMR)*female	0.691	0.740
	[1.346]	[1.509]
Observations	380	380
R-squared	0.87	0.91

Variables are measured in changes between 1946 and 1953. MMR is lagged by 3 years. The reported coefficients are for the triple interaction of dummy for the specified age group * ΔMMR * female. All regressions include district-age group and gender-age group fixed effects. Additional controls are measured in changes. Nutritional diseases are helminths, anemia, diarrhea and vitamin deficiencies. The disease death rates in column 2 are district- and gender-specific, and they are interacted with a dummy for each age group. Standard errors, clustered by district-gender, are reported in brackets. Each observation is a district-gender-5-year age group (19 districts, 2 gender, 10 age groups).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 5: Effect of maternal mortality on literacy: Specification checks
Difference-in-difference-in-difference estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Basic (MMR lagged 3 years)	Clustered at district-gender-treated	Population weights	MMR lagged 1 year	MMR lagged 2 year	MMR lagged 3 year	MMR lagged 4 year
Δ (lagged MMR) *female*treated	-0.873*	-0.873*	-1.449	-0.210	-0.683*	-0.731	-1.160**
	[0.446]	[0.443]	[0.976]	[0.528]	[0.381]	[0.435]	[0.453]
Δ(lagged MMR) *female*control	0.329	0.329	0.056	-0.344	0.101	0.292	0.168
	[0.471]	[0.468]	[0.915]	[0.487]	[0.343]	[0.451]	[0.559]
Observations	342	342	342	342	342	342	342
R-squared	0.54	0.54	0.45	0.54	0.54	0.54	0.54

Variables are measured in changes between 1946 and 1953. MMR is lagged by 3 years in columns 1-3. In columns 4-7, MMR is not a 3-year running average, but the single-year value. All regressions include district-treated, gender-treated, district-control and gender-control fixed effects. Treated is a dummy for being age 5-19, and control is a dummy for being age 25-54. All observations in the regressions are either treated=1 or control=1. Standard errors clustered within a district-gender are reported in brackets (except for column 2). Each observation is a district-gender-5-year age group (19 districts, 2 gender, 9 age groups).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 6: Effect of maternal mortality on fertility
Difference-in-difference estimates

	(1)	(2)	(3)
Dependent variable:	Basic: D-D with male e(0-65) as a control	Male e(0-65), malaria and nutritional death rates as controls	Male e(0-65), malaria and nutritional death rates and lagged IMR as controls
ΔBirth rate			
Δ(lagged MMR)	-5.15*	-4.43*	-4.34
	[2.55]	[2.45]	[2.67]
R-squared	0.92	0.96	0.96
Δ log(# of births)			
Δ(lagged MMR)	-0.08***	-0.09***	-0.10**
	[0.02]	[0.03]	[0.03]
R-squared	0.92	0.94	0.94
Δlog (Female population age 15 -45)			
Δ(lagged MMR)	-0.07***	-0.08**	-0.09**
	[0.02]	[0.03]	[0.04]
R-squared	0.74	0.81	0.82

Variables are measured in changes between 1946 and 1953. MMR is lagged by 3 years. The regressions do not contain any additional controls other than those specified in the column heading (which are all in changes). Nutritional diseases are helminths, anemia, diarrhea and vitamin deficiencies. Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. Number of births and female population ages 15 to 45 are in units of 1000. For consistency with the MMR measure, IMR in column 3 is lagged by 3 years. N=19 districts.
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 7: Effect of maternal mortality on other education measures & age at marriage
Difference-in-difference-in-difference estimates

	(1)	(2)
Dependent variable:	Basic	Add nutritional diseases and malaria death rates
Δ% in school (ages 5-24)		
Δ(lagged MMR)*female	-0.904*	-0.707
	[0.458]	[0.515]
Δ% illiterate at marriage		
Δ(lagged MMR)*female	1.445	2.011
	[0.906]	[1.328]
ΔMean age at marriage		
Δ(lagged MMR)*female	0.015	-0.095
	[0.043]	[0.091]

Variables are measured in changes between 1946 and 1953. MMR is lagged by 3 years. All regressions include district and gender fixed effects. Additional controls are measured in changes. Nutritional diseases are helminths, anemia, diarrhea and vitamin deficiencies. Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. N=38 (19 districts, 2 genders).
* significant at 10%; ** significant at 5%; *** significant at 1%

Data Appendix

District definitions

Two districts that merged over the study period (Puttalam and Chilaw) are treated as a merged district from the outset (named Puttalam). Also, Colombo and Negombo are treated as one district in the censuses (named Colombo), and therefore in our study, despite being separate administrative districts throughout the period. This yields 19 districts.

Conversion from annual data to 1946 and 1953 time periods

Vital statistics data (births, deaths) are available annually. The values we use for 1946 are the average of 1945, 1946, and 1947, and the values for 1953 are the average of 1952, 1953 and 1954. We construct the other variables in a consistent manner. For example, MMR lagged 3 years from 1946 is constructed as the average of the 1942, 1943, and 1944 rates.

Interpolation between census years

To calculate death rates and birth rates, we use annual vital statistics data on deaths and births in the numerator. For the denominator, we linearly interpolate population between census years.

Life expectancy calculation

To calculate life expectancy from mortality tables, we consider an individual who has survived until age a and calculate the probability of surviving each subsequent year. The death rate data are for a 5-year age band. Our calculation uses 1 year as the age increment that we iterate over, and we assume the death rate is constant for each age in the 5-year-band. The formula is:

$$e(a-b) = \left(\sum_{t=a,a+1,\ldots}^{b} (t+1/2) * {}_{t}p_{a} * q_{t} \right) \quad + \quad b * {}_{b}p_{a} * (1-q_{b}) \quad - \quad a$$

where q_t is the probability of dying at age t (the mortality rate for age t), and ${}_tp_a$ is the probability of surviving from age a to age t. The summation accumulates the expected years of life at successive ages for individuals who die at that age. The factor of 1/2 is because we treat the death as taking place at the midpoint of the discrete one-year period. The term that follows the summation accounts for individuals who survive past age b, that is, those for whom the censoring at age b is binding. The subtraction of a is because the measure is of *subsequent* years of life, conditional on surviving until a. The original data are death rates, so ${}_tp_a$ is itself constructed as follows:

$${}_{t}p_{a} = \prod_{\tau=a}^{t-1} (1-q_{\tau})$$

Notes on other variables

IMR: The infant mortality rate is calculated as the number of deaths in a calendar year among those 0 to 1 divided by the number of births in the calendar year. Some individuals born in one calendar year will die as infants in the next calendar year, but this approximation should net out on average.
% Literate: The census defined people as literate if they reported that they could both read and write in at least one language. The standard was that "the person is able to write a short letter and read the reply to it." Literacy was asked of individuals age 5 and over.
% ages 0-24 in school: The census reports the student population among those ages 5-24. Students are those whose response to the occupation question is "student." The most likely category that would be used for children not in school is "other dependent." No further instructions are given for the occupation question.
% illiterate at marriage: This measure defines illiteracy as being unable to sign the marriage certificate.

Appendix Table 1: Classification if diseases 1945-1954

Disease	line in table	1945-1949 classification	line in table	1950-1954 classification
Malaria	28	Malaria	37	Malaria (110-117)
		(a) Benign tertian		
		(b) Quartan		
		(c) Tropical malignant tertian		
		(d) Blackwater fever		
		(e) Malarial cachexia		
		(f) Other and unspecified malaria		
Helminths	42	Other disease due to helminths	42	Other diseases due to Helminths (124, 126, 128, 130)
		(a) Round worms		
		(b) Tapeworms		
		(d) Others		
Vitamin		Vitamin deficiency diseases	64	Avitaminoses & other deficiency states (280, 286)
	67	Scurvy		
	68	Beriberi		(a) Mandama
	69	Pellagra (except alcoholic)		(b) Others
	70	Rickets	134	(a) Mandama
	71	Other vitamin deficiency diseases		
		(a) Mandama		
		(b) Others		
Anemia	73	Anemias (except splenic anemia)	65	Anemias (290-293)
		(a) Pernicious		
		(b) Others (excluding hookworm anemia and malarial cachexia)		
Diarrhea	119	Diarrhea enteritis and ulceration of the intestines (under 2 years of age)	104	Gastro-enteritis and colitis, except diarrhea of the newborn (571, 572)
		(a) Diarrhea and enteritis		
		(b) Ulceration of the intestines (except duodenum)		
	120	Diarrhea enteritis and ulceration of the intestines (2 years of age and over)		
		(a) Diarrhea and enteritis		
		(b) Ulceration of the intestines (except duodenum)		

Notes: This table reports the causes of death from the Reports of the Registrar (Table XXIV) that we use in the analysis. In 1950, Sri Lanka switched to a new classification of diseases (ICD 6) when reporting causes of death. The table shows how we constructed diseases with a consistent definition over the time period. All tables from 1945 to 1949 are identical; each disease is on a separate line in the reports (which is the number we report above in the even columns). The 1950 table was unique but comparable to tables later on. Tables from 1951 to 1954 are identical. The ICD 6 disease codes are shown in parentheses for the 1950-4 data. The first column reports the name of the disease as it is used in the paper.

Appendix Table 2: Effect of maternal mortality on age-specific mortality rates

Dependent variable: ΔAge-specific death rate	Basic	Add nutritional diseases and malaria death rates
Age 0-4		
ΔMMR*female	1.670	0.391
	[1.116]	[1.339]
Age 5-9		
ΔMMR*female	0.323*	0.429***
	[0.174]	[0.138]
Age 10-14		
ΔMMR*female	0.578**	0.167
	[0.223]	[0.344]
Age 15-19		
ΔMMR*female	2.616***	2.479***
	[0.379]	[0.688]
Age 20-24		
ΔMMR*female	5.391***	4.527***
	[0.987]	[0.793]
Age 25-29		
ΔMMR*female	2.055***	2.559***
	[0.597]	[0.632]
Age 30-34		
ΔMMR*female	2.323***	2.991***
	[0.554]	[0.744]
Age 35-39		
ΔMMR*female	-0.829	0.197
	[1.016]	[0.829]
Age 40-44		
ΔMMR*female	-0.915	0.310
	[0.846]	[0.748]
Age 45-49		
ΔMMR*female	0.258	0.678
	[0.689]	[1.301]
Age 50-54		
MMR*female	0.625	-0.167
	[1.023]	[1.198]
Age 55-59		
ΔMMR*female	-4.305	1.190
	[2.559]	[2.558]
Age 60-64		
ΔMMR*female	-5.308**	0.317
	[2.275]	[2.938]
Age 65+		
ΔMMR*female	-2.860	-3.193
	[2.797]	[2.403]

Variables are measured in changes between 1946 and 1953. All regressions include district and gender fixed effects. Additional controls are measured in changes. Nutritional diseases are helminths, anemia, diarrhea and vitamin deficiencies. Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. N=38 (19 districts, 2 genders). * significant at 10%; ** significant at 5%; *** significant at 1%

Appendix Table 3: Test for outliers: results dropping one district at a time
Dependent variable: % literate by age

	Basic	**Drop district 1**	**Drop district 2**	**Drop district 3**	**Drop district 4**
Δ (lagged MMR) *female*treated	-0.873*	-0.486	-1.003**	-0.929**	-0.457
	[0.446]	[0.563]	[0.443]	[0.448]	[0.375]
Δ(lagged MMR) *female*control	0.329	0.680	0.502	0.377	0.063
	[0.471]	[0.666]	[0.472]	[0.477]	[0.442]
R-squared	0.54	0.52	0.55	0.54	0.58
	Drop district 5	**Drop district 6**	**Drop district 7**	**Drop district 8**	**Drop district 9**
Δ (lagged MMR) *female*treated	-1.037**	-1.001**	-0.855*	-0.921*	-0.862*
	[0.447]	[0.445]	[0.452]	[0.470]	[0.477]
Δ(lagged MMR) *female*control	0.086	-0.001	0.291	0.289	0.433
	[0.450]	[0.406]	[0.475]	[0.495]	[0.497]
R-squared	0.54	0.51	0.54	0.54	0.54
	Drop district 10	**Drop district 11**	**Drop district 12**	**Drop district 13**	**Drop district 14**
Δ (lagged MMR) *female*treated	-0.999**	-0.966**	-0.855*	-0.877*	-0.819*
	[0.454]	[0.442]	[0.446]	[0.447]	[0.473]
Δ(lagged MMR) *female*control	0.363	0.440	0.333	0.337	0.200
	[0.496]	[0.481]	[0.471]	[0.471]	[0.480]
R-squared	0.53	0.55	0.53	0.55	0.54
	Drop district 15	**Drop district 16**	**Drop district 17**	**Drop district 18**	**Drop district 20**
Δ (lagged MMR) *female*treated	-0.730	-0.872*	-0.876*	-0.785*	-1.174**
	[0.473]	[0.446]	[0.474]	[0.456]	[0.465]
Δ(lagged MMR) *female*control	0.615	0.332	0.393	0.385	0.231
	[0.460]	[0.472]	[0.498]	[0.487]	[0.586]
R-squared	0.55	0.54	0.54	0.54	0.54

This first regression reported in this table reproduces the regression in Table 3, Panel A, column 1 (N=342). Thereafter, the results are reported dropping one district at a time in alphabetical order (N=324). Variables are measured in changes between 1946 and 1953. MMR is lagged by 3 years. All regressions include district-treated, gender-treated, district-control and gender-control fixed effects. Treated is a dummy for being age 5-19, and control is a dummy for being age 25-54. All observations in the regressions are either treated=1 or control=1. Standard errors clustered within a district-gender are reported in brackets. Each observation is a district-gender-5-year age group (19 districts in the first regression, and 18 districts thereafter; 2 genders; 9 age groups). Results not reported here: As best seen in Figure 2, there are 2 main outliers for MMR which are Anuradhapura and Vavuniya (districts 1 and 19 above). Dropping them simultaneously (N=306) gives a coefficient of -.865, almost exactly as in the main specification.
* significant at 10%; ** significant at 5%; *** significant at 1%