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

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Abstract

Many economists argue that high mortality is a major restraint on human capital accumulation and, in turn, growth. A short time horizon makes a person less likely to invest. Therefore, having a lower life expectancy reduces the incentive to obtain schooling. However, there is controversy over whether this theoretical effect is empirically important. There is good evidence that improvements in health make children more capable of obtaining schooling, but previous research has been less able to estimate whether health improvements encourage human capital accumulation via life-expectancy effects. This paper uses a type of mortality that lends itself to isolating life-expectancy effects. We examine maternal mortality declines in Sri Lanka between 1946 and 1963. Maternal mortality was a major killer of prime-age women and was a common and visible risk. Its elimination (driven by improvements in availability of health care and transportation to hospitals at the time of delivery) resulted in large increases in the life expectancy of women relative to men in a very short period of time. We use variation across districts, over time and by gender to identify the effects of longevity on education and other outcomes. We find that the 80% reduction in maternal mortality risk increased female adult life expectancy by 1.7 years (a 5% increase), and increased female literacy by 5%. Lower maternal mortality risk also appears to have increased the birth rate.

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I. Introduction

A person will invest more if the stream of returns on that investment last for longer, all else equal. One of the potentially most powerful implications of this simple reasoning is that improvements in life expectancy will increase investment, for example in human capital, which in turn will 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). Much of the empirical evidence on this question measures the cross-country relationship between health improvements and growth, as well as channels such as education and investment. This literature has found mixed evidence on the quantitative importance of improvements in life expectancy on growth. Shastry and Weil (2003) and Lorentzen, McMillan and Wacziarg (2005) find that the effects are large, while Acemoglu and Johnson (2006) find small effects.[2] One limitation of that approach is that much of the between-country variation over time in life expectancy is driven by changes in the infant mortality, or deaths that occur before schooling begins. Infant mortality might have effects on education but not because it affects the period over which returns to education are earned.

Another branch of the literature examines the effects of health on education, but does not disentangle incentive effects from direct effects of healthier children being more able to attend school. For example, both Miguel and Kremer (2004) and Bleakley (2007) find that deworming interventions led to increased school enrollment. The main interpretation of their results is that sickness had been preventing children from attending or succeeding in school.

This paper uses reductions in maternal mortality, a source of variation that lends itself to better isolating life-expectancy effects on behavior. We examine Sri Lanka over the twenty-year period from 1945 to 1964. In 1946, the maternal mortality ratio (MMR) was 1550 maternal deaths per 100,000 live births, or 1.55%. By 1953, this had fallen to 490 and by 1963 to 240. In other words, there was a 70% reduction over the seven-year time span of 1946-53 and another 50% decline over 1953-63. These declines were large and occurred in a very short period, with larger declines in districts where maternal mortality was larger to begin with.

[2] See also Young (2005) and Weil (2007).

Maternal mortality declines have several other advantages as a way to measure the effects of life expectancy. This type of mortality importantly occurs after major human capital investments are made and early in adult life so that a deterred maternal death translates into a large increase in life expectancy. Because maternal mortality is not a large morbidity source, we can isolate the incentive effects of longer life expectancy. Finally maternal mortality directly affects only women, so men can serve as controls.

Maternal mortality is also an important type of health improvement per se, because while Sri Lanka made great strides sixty years ago, most developing countries still face high maternal mortality. MMR is on average 400 per 100,000 births in developing countries, with several countries mainly in sub-Saharan Africa facing rates of over 1000 maternal deaths per 100,000 births.

Our estimation strategy is a difference-in-difference-in-difference: 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 very complete. Thus our life expectancy measures are considerably more accurate than country level measures, which differ in their accuracy and are often based on infant mortality alone (Deaton 2006).

We find that the 80% reduction in maternal mortality risk increased female adult life expectancy by 1.7 years (a 5% increase), and accounted for all of the difference in life expectancy gains between men and women. These declines in maternal mortality increased female literacy by 5%. Lower maternal mortality risk also appears to have increased the birth rate.

This paper is organized as follows. The next section describes the theoretical predictions that we test in the data. Then in section 3 we discuss our empirical strategy, its advantages and potential caveats. The data and some general background on Sri Lanaka are presented in section 4. Section 5 shows the effects of maternal mortality on life expectancy and Section 6 looks at its effects on outcomes. Section 7 concludes.

II. Conceptual framework

The hypothesis that this paper tests empirically is that changes in life expectancy affect human capital investments as well as other behavioral choices. To lay out the predictions that we test, we present in this section a simple model of schooling and fertility choices, and examine comparative statics when mortality rates change.

We consider a unitary household consisting of a woman and man who make two linked 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.

We model the returns to schooling in a standard Mincerian way: each year of schooling leads to a certain percentage increase in earnings. It is important to note that earnings are just one, and perhaps not the most important, benefit of education for females, particularly in the context we study, Sri Lanka fifty years ago (Haveman and Wolfe 1984). Other potential benefits for a woman of being better educated are being healthier, matching with higher "quality" husbands, having more bargaining power in the household, being able to use contraceptives and control fertility better, and having higher "quality" children (e.g., better educated or healthier) (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. The model can be thought of as also encompassing other benefits of education that provide a stream of utils during post-schooling years.

It is also worth noting that 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 et al 1999, Oster 2007).

Another important point to make about the model which is also relevant for the empirical work is about belief formation regarding mortality risk. We assume that a reduction in mortality changes people's beliefs contemporaneously. This implies both

that people do not anticipate reductions (they are not too sophisticated) and that they take note of contemporaneous reductions and correctly calculate how life expectancy changes (they are not too unsophisticated).

We model the household as making a binary choice C of whether to have a child and then choosing the years of schooling s of the child (after observing the child's gender). We assume the household's maximand is the sum of the woman's, the man's and the child's discounted income. This is a simplification but the basic comparative statics we illustrate hold more generally. Having a child occurs at time τ in the woman's lifetime. The decision we model occurs at the moment of (potential) childbearing for the mother. 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, a woman lives until time T. The man always lives until T. Households have a discount rate δ. We assume the labor market returns to schooling is γ. We use the subscript f to denote father, m to denote mother, b to denote boy and g to denote girl.

The household's maximization problem is

$$\max_{sb,sg,C}\left[U_f(C)+U_m+\frac{C}{2}(U_b(s_b)+U_g(s_g))\right]$$

where

$$U_m=(1-C\mu)\int_\tau^T e^{-\delta(t-\tau)}ye^{\gamma s_m}dt,\quad U_f=\int_\tau^T e^{-\delta(t-\tau)}ye^{\gamma s_f}dt,$$

$$U_g=\int_{s_g}^{\tau} e^{-\delta t}ye^{\gamma s_g}dt+(1-C\mu)\int_\tau^T e^{-\delta t}ye^{\gamma s_g}dt,\ \text{and}\ \ U_b=\int_{s_b}^{T} e^{-\delta t}ye^{\gamma s_b}dt$$

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 mother and father and they are currently earning at the time of the decision. We also 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:

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

The optimal schooling level is

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

This gives the comparative static that girls obtain more schooling when the risk of maternal mortality falls (μ decreases):

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

This effect is larger when reductions occur earlier (τ is lower):

$$\frac{\partial^2 s_g^*}{\partial \mu \partial \tau} > 0 .$$

We do not empirically test this cross derivative, but it motivates our use of maternal mortality as a source of identification, since the earlier in productive life the mortality risk is, the larger the incentive effects on investment if it is reduced.

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

$$\frac{\partial s_b^*}{\partial \mu} = 0$$

Under different assumptions, one might find a positive or negative effect on boy's education. For example, if one incorporated credit constraints into the problem, 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. Conversely, the shadow of the marriage market might create a ratcheting effect, so that boys' education increases.

The decision to have a child is also affected by the maternal mortality rate with

$$\frac{\partial C^*}{\partial \mu} < 0$$

This is driven by two effects: the risk to the mother's health (and income stream) and the lower utility from a child, if the child is a girl since girls face higher mortality risk compared to boys.

We do not incorporate into the model one choice that we examine empirically: age at marriage. It is worth discussing in words how the model might be extended to incorporate this choice. There are three main hypothesized channels through which MMR affects age at marriage for females. First, if girls get more education when MMR decreases and they typically delay marriage until education is complete, then a reduction in MMR would lead to an older age of marriage for girls. Second, because in Sri Lankan society, widowers typically remarried but widows did not, a decline in female mortality may create a "marriage squeeze" for women because of the reduced supply of widowers seeking wives (Dixon 1970, Fernando 1975). This also would lead MMR declines to cause a higher age at marriage for women. Third, a reason to delay marriage in a society with limited birth control is to delay the onset of fertility and reduce total fertility. Here MMR would have the opposite effect on age at marriage. A reduction in MMR would increase the demand for childbearing, and might also shift births to earlier ages since maternal mortality risk is particularly high for young women. Hence, on net, it is theoretically ambiguous whether lower MMR would lead women to get married younger or earlier. Also note that there is likely to be a spillover effect on men's age at marriage because of positive assortative matching on age between wife and husband.

Another aspect of this decision problem not in the model is the "quantity-quality" tradeoff. If a household responds to the maternal mortality decline by having more children—MMR in essence raises the price of quantity—then households might choose to have more but lower quality children. The household might educate each of their children less. This effect, though, would not necessarily affect gender differentials in education, if quality falls for both boys and girls. (Note that another channel through which child quality falls is if there is cohort crowding and school quality falls, though again this would primarily predict a level drop in educational attainment.) Alternatively, if the expected lifetime returns to female education increase when MMR falls, then parents might shift from quantity to quality and have fewer children (Becker, Murphy, and Tanamura 1990, Galor and Weil 2000, Bleakley and Lange 2006). This would create

an offsetting effect to the higher fertility induced by greater maternal survival modeled above.

III. 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, in essence, a difference-in-difference-in-differences (DDD). The first difference is over time, since maternal mortality declined over the time period 1946 to 1963. The second difference is across geographic areas. In practice, places with initially high maternal mortality experienced larger declines. The third difference is between gender; maternal mortality is quite unique among major causes of death since it exclusively affects women.

The estimating equation is

$$(1) \qquad \begin{aligned} Y_{dtg} = \; & \beta_0 + \beta_1 MMR_{dt} * female + \\ & \gamma_{dt}(district * year) + \delta_{dg}(district * female) + \mu_{tg}(year * female) + \varepsilon_{dtg} \end{aligned}$$

where *d* stands for district (19), *t* for year (3) and *g* for gender (2). The main specification contains district*year fixed effects, district*female fixed effects, and year*female fixed effects (thus the main effect of *MMR* is absorbed by the district*year interactions and the main effect of *female* is absorbed by the year*female and district*female interactions). 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).[3]

Even though for some outcomes data is available every year, we restrict attention to the three census years, 1946, 1953 and 1963 because behavior is more likely to respond to low-frequency changes in MMR than to year-to-year changes; high-frequency changes are less likely to be perceived by people contemporaneously, and are less likely to be viewed as permanent. Sharp declines in MMR over 7 or 10-year periods are the type of objective change in life expectancy that are likely to lead to changes in subjective life

[3] We re-estimated models clustering standard errors at the district and gender levels (to account for possible serial correlation), although since there are only 19 districts we prefer our main specification. We also re-estimated models using population weights. The results are in Appendix Table 4. Our conclusions are qualitatively very similar either way.

expectancy.[4] The use of long differences also minimizes the effect of measurement error in th presence of fixed effects, since measurement error is likely to be small relative to the changes that took place over the 20 year studied here.

The gender-specificity of maternal mortality is one of several advantages of maternal mortality as a source of variation in life expectancy. A second advantage is that it affected prime-age adults (primarily 20 to 40 year-olds), as opposed to children or older adults. The channel that has been emphasized theoretically as a reason that mortality reductions could lead to more investments is that individuals have longer horizons over which to reap the returns to investment. However, much of the historical improvement in life expectancy which previous research has used to identify the impact of mortality reductions on investments has been driven by declines in infant and child mortality, that is, mortality before the age at which human capital investments are even made. Conversely, improvements in elderly death rates will lead to modest gains in expected life-years compared to improvements in early adult mortality.

A third useful feature of maternal deaths is that this type of mortality does not correspond to a major source of morbidity. The implication is that reductions in maternal mortality isolate life expectancy effects on, say, schooling rather than being intertwined with mechanical effects of reduced morbidity on the ability to attend school. Consider malaria, in contrast, where interventions that reduced malaria deaths simultaneously reduced morbidity from malaria, so that a statistical analysis of the interventions' effect on education confounds life expectancy and mechanical effects.

A fourth important aspect of the maternal mortality declines in Sri Lanka is that there were large, rapid declines. Therefore, it seems plausible that the reductions were salient to the population, so that not just objective life expectancy, but subjective life expectancy increased. The rapidness of the decline is also helpful for identification purposes since one can hope to separate the effects of the rapid decline from slower secular changes in outcomes such as literacy or age of marriage that occurred over time.

[4] It is possible that even though we allow for several years in between observations, there is an even longer lag between the change in maternal mortality and the change in behavioral outcomes. We re-estimated models using MMR lagged 3 or 5 years instead of contemporaneous values. We find that lagged changes have a much smaller effect on behavior.

Threats to validity

The empirical strategy treats between-district variation in maternal mortality declines as exogenous, and the corresponding effects on female outcomes as caused by these declines. One potential threat to the validity of this research design is if declines in maternal mortality are correlated with other health improvements that also affected outcomes such as literacy or age at marriage. For example, expansion of maternal and child health programs are one reason maternal mortality 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 the effects based on differential improvements among girls. Another potential confound is that Sri Lanka made dramatic gains in malaria eradication over the time period of 1946 to 1953. This concern is addressed, first, by controlling for malaria death rates and, second, by taking advantage of the gender comparison, since again malaria affected both males and females.

Another type of concern is that there might be differential gender-specific trends in certain districts, with the same districts that reduce maternal mortality also seeing gains in female education for independent reasons. For example, one might worry that certain less advanced districts catch up over time, and the process of development entails both health improvements and progress for girls. We can test whether initially gender gaps in education are larger in places with more maternal mortality. Figures 1a and 1b show that these two measures in fact have a very weak correlation in the initial period. While one cannot directly test the DDD identification assumption, which is about changes over time in these measures, the fact that high-MMR districts were not particularly behind 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.

The death of a mother might also directly affect girls' education relative to boys' education if mothers are more pro-daughter than fathers are. This is an alternative way that MMR could have a causal effect on girls' education, different from the incentive effects that we focus on which arise from the daughter's life expectancy rising.
To gauge whether this direct effect could account for the results, consider that the total fertility rate was about 5 during this period. The likelihood of a child having a mother

who has died will vary with parity (because one's mother might die during a subsequent childbirth), but a rough calculation suggests that on average, a child's mother survives childbearing with 96.8% probability when there is a 1.6% maternal mortality risk (the 1946 level). A reduction to 0.3% maternal death risk (1963 level) increases the likelihood of a mother surviving childbearing to 99.4%, or a 2.6 percentage point increase. To explain, say, a 2.6 percentage point increase in girl's literacy caused by MMR declines from 1946-63 (to preview an actual result), every girl whose mother has died would have to have a 0% chance of becoming literate and every girl whose mother is alive would have to become literate. The overall literacy rate was 50%, however. Therefore, even in the extreme case where girls without mothers are certain to be illiterate, and girls with mothers have the population-average probability, the direct effects could only account for half of the estimated effects. Moreover, the probability that a motherless girl becomes literate is almost surely significantly higher than 0%, particularly since some high birth-order girls would have become literate before their mother subsequently died in childbirth.

Finally, the potential for spillovers or general equilibrium effects of MMR raise the question of whether males are a valid control group. Our assumption is that MMR has a direct effect on women's life expectancy and therefore schooling, but MMR could have an indirect effect on men if their schooling is affected either by their siblings' or future spouses' life expectancy or schooling. One can imagine these spillovers being either positive or negative. For example, the dynamics of the marriage market might create positive spillovers if male education is more valuable in the marriage market when females are more educated. Conversely, the 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 these effects, but we think these are not likely to be of the same order of magnitude as the main effects we examine here.

IV. Background on Sri Lanka and Data

While Sri Lanka today remains a poor country (about $4000 annual per capita income), on many dimensions of human development, it is quite advanced. Its progress against maternal mortality sixty years ago is one example.

Sri Lanka in the 1940's as today had much higher educational participation and gender equality in educational participation than most poor countries.[5] The education system in this period was organized into three levels: primary school (ages 5-11), secondary school (ages 12-18) and higher learning. School attendance was compulsory from ages 5 to 14 since early in the 20th century but not strictly enforced. In 1945, all fees in state, assisted schools (which made up the vast majority of schools and were opened to both genders) and at the university were abolished. There were no major gender-specific educational policy changes during the 1945-1963 period. Enrollment at primary and secondary levels increased tremendously during this period, in large part because of a transition from English-medium to Sinhalese- and Tamil-medium education: the percent of children ages 5 to 14 enrolled in school in Sri Lanka went up from 57.6 in 1946 to 71.6 in 1953 and 75.1 in 1963. Secondary enrollment increased by 36.1% from 1953 to 1963. Although females had lower literacy rates than males, the literacy gender gap was substantially reduced during the period (see summary statistics). By 1967, 37% of university students were female. (By comparison at that time, females made up 38% of university students in UK and 40% in US.) (Siriwardena, 1973).

The total fertility rate in Sri Lanka in 1946 was 5. The birth rate (births per 1000 females ages 15 to 45) increased from 164 to 186 between 1946 and 1953, and then decreased between to 180 in 1963. Sri Lanka appears to have entered its "fertility transition" (period of declining fertility) toward the end of our study period. Sri Lanka is primarily a Buddhist society, so its marriage customs differ from the rest of South Asia which is primarily Hindu or Muslim. About one third of marriages in Sri Lanka were "love marriages" rather than arranged marriages at this time, and dowry plays a less important role in Sri Lanka, for example (Caldwell 1999). The mean age of marriage was 22.4 for females and 28.3 for males.

[5] We refer to Sri Lanka, but during the study period (and until 1972), the country was named Ceylon. Also note that the year of independence from Great Britain was 1948.

In Sri Lanka, the reduction in maternal mortality was driven by several policies related to health. Three factors 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. Importantly most of the services were provided for free. Second, to increase access to health care, transportation to health care 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). If the mother's (or newborn's) health was at risk due to a delivery complication, a woman could be rushed to a health facility. Figure 2 shows for the entire country that there was a large increase in the number ambulances, health centers, and hospital beds per capita (the number of hospitals per capita increased but population increased more in the 1950s). The number of women delivering at government institutions rather than at home increased dramatically during this period (from 20% in 1945 to 55% in 1960, World Bank 2003), suggesting that access to care did indeed improve. Third, like other developing countries, Sri Lanka adopted recently developed technologies from the West, most importantly sulfa drugs, penicillin and blood transfusions. These technologies had been proved to dramatically reduce maternal mortality in the West (Loudon 2000, 1991, 1988, Paxton et al 2004, Lerberge and De Brouvere 2001). Causes of maternal mortality are very difficult to determine and are often misdiagnosed (Ceylon Administration Reports, 1945). Nevertheless the existing data shows that the main causes of maternal deaths in 1945 were hypertension/eclampsia (46%), sepsis (24%), and other.[6]

Although the literature suggests that access to proper care at the time of delivery is the single most important determinant of maternal mortality, other factors affect maternal mortality rates. Access to prenatal care appears not to matter much because most complications at birth cannot be predicted (Maine et al 1991). Maternal mortality is highest for very young and very old mothers, and it is also higher for first born and for higher order births (4^{th} and above)—thus changes in the number and timing of births may affect maternal mortality. These factors, however, appear to have a relatively small

[6] We calculated these numbers using data from Table 50 of the Vital Statistics report for 1945.

impact on maternal mortality. Trussell and Pebley (1984) calculate that, in general, 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 very large changes in fertility behavior could not explain the dramatic declines in MMR in Sri Lanka. It is also worth noting that family planning activities only started in Sri Lanka in earnest in 1965, with the initiation of the National Family Planning Program (World Bank 2003).

While no solid causal estimates of the returns to education for the cohorts we study exist, Mincerian estimates suggest a return to a year of education of 7% for both males and females (Psacharopoulos 1994). Returns calculated conditional on labor force participation might seriously overestimate ex ante returns, particularly for women. However, as discussed earlier, the labor market is not the only or arguably even the most important type of returns to education for women. Consistent with the hypotheses that education has returns in the marriage market and in terms of children's health, unreported OLS regressions using the 1987 Demographic and Health Survey (DHS) for Sri Lanka suggest that more education for a woman is associated with being married to a more educated man and with lower infant mortality among her children.

Data

The data for the analysis were collected from multiple sources, primarily annual Vital Statistics reports and the Census of Population for 1946, 1953, and 1963. The data are disaggregated geographically by district. For districts that divided over the study period, we aggregate up to the original, larger district, and for districts that merged over the study period, we use the merged district from the outset. This yields 19 districts. (See the data appendix for more details.)

The main explanatory variables are from the vital statistics. Data on total deaths are available by district, year, gender and age (5 year groups). These are used to construct overall age-specific death rates (by using interpolated population counts from the Census). These are shown in Table 1b. Death rates exhibit the usual J-shape, with very high infant mortality and increasing mortality after age 40. It is interesting that in 1946 females show larger death rates than males up until age 45, but lower mortality rates

thereafter. The ratio of female to male deaths is the largest for ages 15 to 45. The maternal mortality rate is reported at the same level of aggregation.

For deaths broken down by cause, data are reported by district, year, gender, but not age (although some diseases are denoted as affecting only children). Because deaths by cause were reported in great detail, we only collected information on the causes of death that were large in 1946 and for which consistent series could be obtained (the reported causes of death changed in 1950 when Ceylon adopted the new International Classification of Diseases—see data appendix for details). The final data includes 18 broad causes of death and constitute 78% of all deaths in 1946. Two common diseases reported in the vital statistics are unique to Sri Lanka. Pyrexia is a catch-all category; the cause of death is said to be pyrexia if the person had a fever and the cause is otherwise unknown, which is particularly common in rural areas. Rathe is a disease that affects only infants; it was not part of the international classification of diseases at the time, but the registrar of Sri Lanka used it because it was commonly reported as the cause of death (Vital Statistics of Ceylon 1945, page 28). Table 1b shows that death rates for infant diseases (rathe, convulsions and congenital debilities) were very high. Among diseases that affect adults, pyrexia (fever), pneumonia, malaria and nutritional deficiencies were the highest in 1946. In 1963, however, malaria deaths were virtually non-existent.

These data are believed to be of excellent quality (World Bank 2003)[7] and allow us to construct life tables for each district-year-gender during the period. Although it would be preferable to use age-specific death rates with 1-year age categories, the data are not available, so we make assumptions about the distribution of deaths within age categories (see Appendix for details). One advantage of the 5-year grouping is that it minimizes errors in the distribution of ages, which tend to be misreported in single digits (United Nations 1976). We construct three measures of life expectancy: life expectancy at age 15 and censored at age 65, life expectancy at age 15 and censored at age 45 and life expectancy at age 45 and censored at age 65. Note that this last measure covers ages after the years of childbearing, so it is primarily a placebo variable (but is also affected by MMR reductions due to competing risks). We censor life expectancy at age 65 because

[7] Studies of the completeness of births and deaths records, for example, show very high completeness (United Nations 1978).

the death rates in the early years are not reported for older ages, and because life expectancy calculations are in general very sensitive to assumptions about the distribution of deaths among censored individuals (in 1946 only relatively few are censored but the number is much larger in 1963). We focus on life expectancy at age 15 since our intent is to look at changes in longevity in the post-investment years of a person's life. Table 1a shows that there was a very substantial increase in life expectancy at age 15, about 8.2 years for women and 6.6 years for men. Life expectancy at 15 shows convergence: the difference between men and women was 2.2 years in 1946, but about 0.6 of a year in 1963.

The vital statistics are also the source for births and marriages. They report total number of births by district and year. Breakdowns by age of mother are also available starting in 1952. Birth registration was almost 100% complete (United Nations 1978).

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 groups, age and education are available only for all marriages other than Muslim and Kandyan marriages (data for these groups are not available in the early reports). Out of 44,325 marriages, 6,001 were Kandyan and 4,641 were Muslim. Therefore our statistics only cover 76% of marriages in 1946.

Data on population, literacy and school enrollment are available from the census in 1946, 1953 and 1963. School enrollment, unfortunately, is not broken down by age; it is an aggregate number for 5 to 24-year-olds. We also collected district level information on percent living in urban areas, population density and employment.

The main limitation of the data is that we do not have any further variation within districts and gender. Also there are a few outcomes that we would like to examine that we do not have in the data. Completed years of education is not available because it was not recorded in the earlier censuses. To measure how child health investments respond to life expectancy, we would also like to have measures such as height or vaccination rates but we have been unable to obtain them (Dow, Philipson, Sala-i-Martin 1999).

V. Patterns of MMR reduction and effect on life expectancy

This section presents descriptive evidence on MMR reductions in Sri Lanka and quantifies the impact the reduction had on life expectancy. Figure 3a shows MMR over time by district from 1941 to 1964. As can be seen, MMR fell considerably over the time period, particularly between 1946 and 1953, with substantial variation across districts. It is also evident that there is a peak in 1946, thought to be caused by the 1946 malaria epidemic. We average data from 1945 and 1947 to avoid using this variation.

An important question is what drove the reductions. As described above, there were several policies such as expanded hospital births, ambulances, and maternal health programs that contributed. A summary way of characterizing the changes is that the places with initially higher levels of MMR saw larger improvements. In other words, there was strong convergence in MMR, as shown in Figure 3b. Appendix Table 2 reports results from regressions where the change in MMR is regressed on the level in the base period. The results very strongly support the convergence hypothesis—the initial level of MMR is highly significant and the r-squared in the regression is higher than .99 for both periods, thus suggesting that differences in initial levels across districts almost perfectly predict subsequent declines, although not as well in the 1953-63 period.

Figure 4 shows the trends in life expectancy at age 15 (censored at age 65), by gender and district. They show that life expectancy was rising rapidly for both men and women, but women were catching up to men. Also interestingly, the districts where the initial disadvantage of women was the greatest were also the districts with initially high maternal mortality. Figure 5 shows this directly. For each census year we plot the difference in life expectancy between men and women at age 15 and the MMR. As maternal mortality declined, life expectancy of women relative to men rose (although there are a few exceptions for which MMR and LE move in the same direction). Also as expected, the relative increases in life expectancy were larger when MMR declines were larger, especially in the 1946 to 1953 period. When we plot life expectancy differences at age 45 (rather than 15) against MMR we find, consistent with our hypothesis, that MMR declines are either not associated with or are *positively* associated with gender differentials in life expectancy at age 45. A positive association might be expected if the

marginal women who survive childbearing years have higher risk of dying between the ages of 45 and 65.

This evidence suggests that maternal mortality declines were perhaps responsible for the initially lower life expectancy of women and might explain much of the convergence between men and women. Figure 6 shows some additional evidence that suggests that excess female mortality was mostly related to maternal deaths. The figure plots the ratio of female to male death rates by age (when the ratio is larger than one, female death rates are larger than males), and the birth rate by age. It shows that excess female mortality was highest at the ages when the birth rate is highest as well. This is what we would expect since mortality from maternal causes is only a risk for pregnant women, and the higher the birth rate the higher the associated risk. Unfortunately these data are only available in 1952 onwards.

To quantify the effect of maternal mortality on life expectancy we estimate equation (1). We regress life expectancy on MMR*female and a full set of double interactions of gender, district, and year. The results are reported in Table 2. The first column shows the results from the main specification. The effect of MMR on life expectancy at 15 is negative and significant: when MMR fell, life expectancy at 15 rose. The coefficient implies large effects: Since MMR fell from 1.65 in 1946 to .31 in 1963, the estimate implies that MMR declines resulted in an increase in female life expectancy of 1.69 years, 1.47 of which occurred during the 1946-53 years. Women's life expectancy at 15 increased by 8.23 years over the full period so maternal mortality declines can explain about 20% of the increase in life expectancy. Male life expectancy at 15 during the same period increased by 6.63 years, so maternal mortality can explain 100% of the convergence between men and women.

An alternative way of assessing the impact of declines in maternal mortality is to calculate what life expectancy in 1946 for women would have been if mortality rates for ages 15 to 45 were at there 1963 levels, but all other rates were at their 1946 level. Although we do not have maternal deaths by age, we know that the total number of maternal deaths and we can calculate that they account for 25% of deaths in the relevant age range. We find that lowering mortality rates for age 15-45 by 25% (essentially eliminating maternal mortality) results in an increase in life expectancy by 1.95. This

estimate (which is an upper bound since MMR was still positive in 1963) is consistent with our regression results.

The next rows show instead what the effect of maternal mortality is on life expectancy between ages 15 to 45 and from ages 45 to 65. As expected there is a large and significant negative effect on the 15 to 45 measure, and a positive and insignificant effect on the 45 to 65 measure. Imposing that district fixed effects are the same for men and women or that districts do not have their own specific trends has no effect on our estimates.

The remaining columns in the table report the results controlling for death rates from malaria or nutrition-related diseases, two types of disease targeted by health interventions during the period under study. These controls are meant to test the potential for omitted variable bias. However, they are not our preferred specification for two reasons. First, these controls could be endogenous: for example, percent in school (an outcome we examine later) could determine nutrition-related diseases since the government provided food in school. The second issue is more subtle. In some sense we could be over-controlling by including these diseases because malaria and nutrition deficiencies increased the likelihood of maternal deaths. At the time in Sri Lanka, reports on maternal mortality always linked nutrition of the mother and malaria to maternal mortality because poorly nourished mothers or mothers with anemia are more likely to die at birth (e.g. De Silva et al 1943). More recent work on maternal mortality is much more skeptical about the relationship between nutrition and maternal deaths, though (Loudon 2000, Maine 2000). Loudon (2000) in particular strongly suggest that "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." Nonetheless, we view these results with these controls as useful checks. These controls also have no significant impact on our coefficients. In short, maternal mortality is a significant predictor of female life expectancy.

More detailed results are presented in Table 3, which shows the coefficient on MMR*female on age-specific death rates, using various specifications. These results show that maternal mortality is positively and significantly associated with death rates for ages 15-19, 20-24, 25-29 and 30-34 year-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 small and statistically insignificant. The results for ages 35 and above are all negative suggesting that when MMR falls, death rates at older ages increase.

In Table 4 we look at whether MMR*female predicts differential declines in death rates for other causes of death. Recall that cause of death data is available by gender but not age, but some diseases are exclusively confined to those under 5. We estimate separate linear regressions for each major cause of death, although these results are somewhat hard to interpret since these are competing risks (thus when mortality from one cause of death falls at least one other must increase). Also the errors will be correlated across equations, which we ignore.

Panel A looks at diseases that only affect children under 5. Here we find that MMR*female is significant, although the direction of the effect varies, for some causes declines in maternal mortality are correlated with disproportionately large declines for girls (e.g. convulsions and congenital debilities) whereas in other cases, boys benefited relatively more (rathe). The coefficients almost offset each other, which explain why in the previous table, there appear to be no effect of MMR on deaths rates for ages 0 to 4. These results are consistent with the fact that health services were concentrated on women and children. Although among children, there was no differential effect overall on mortality, boys benefited more than girls when they started with initial levels of the disease that were higher, and vice versa (this is confirmed by looking at the mean of cause-specific death rates). Appendix Table 3 shows in fact that controlling for the initial level of the disease, there are no differences in the trends for males and females (the interaction between the initial level and the dummy for female is never significant).

Panel B looks at death rates for other diseases, they appear in order of importance, with the first cause (pyrexia) being the largest in 1946. Ideally maternal mortality should not be correlated with declines in other causes of death for women relative to men. Two out of the 10 diseases we examine have significantly correlated with MMR*female: pyrexia and tuberculosis. Pyrexia, however, is most likely to be correlated with maternal mortality because maternal mortality is often associated with fever and misdiagnosed—this is true both in Sri Lanka at this time (De Silva et al 1943) and in most countries today (Deneux-Thauraux et al 2005). Death rates from pyrexia are indeed higher among women

than men in 1946 and the difference is indeed smaller in 1963. As additional suggestive evidence, Figure 7 plots the ratio of female to male deaths for the entire country in 1950, the first year for which vital statistics reports deaths by cause and age for the entire country (these are never reported by district). It shows that excess female mortality from pyrexia increases during childbearing years, which is consistent with the misdiagnosed maternal mortality hypothesis. We also find MMR*female to be negatively correlated with tuberculosis. We cannot explain this finding, although as we mentioned above the estimation here does not take into account the competing risk framework and, of course, some statistically significant coefficients will arise through chance.

Overall the evidence presented in this section suggests that declines in maternal mortality resulted in an increase in female life expectancy at age 15 of about 1.7 years and were mostly responsible for the convergence in life expectancy between men and women in Sri Lanka between 1946 and 1963. We now examine the effects of maternal mortality on behaviors such as educational investment.

VI. Effect of MMR on behaviors

Tables 5a and 5b show the estimates of the declines in MMR on a variety of outcomes. We also report results controlling for gender-specific malaria and nutrition related death rates. We start by looking at the birth rate (Table 5a). We predicted that when the risk of dying in childbirth falls, the number of pregnancies and births should increase. This is indeed what we find: the coefficient on maternal mortality is negative and significant. But because when examining the birth rate, we cannot make use of gender as a third difference, the potential for omitted variable bias is larger in these simple difference-in-difference estimates. Thus, we control for malaria and nutrition related diseases. Malaria in particular is hypothesized to have affected the fertility rate (Langford 1981). We can also control for male life expectancy at age 15. Maternal mortality should not have an effect on males, so any correlation between MMR and male life expectancy must be driven by unobserved factors. When we include other controls, the coefficient falls quite considerably. In the last column (including all controls), it is insignificant and considerably smaller, less than a sixth of the original estimate. The magnitude of the effect is also small: a 10% decline in MMR results in less than 1%

increase in the birth rate. That said, according to the point estimate (final column), the decline in MMR from 1945 to 1963 led to a 3% increase in the birth rate, explaining more than half of the overall increase in the birth rate for the period.

Because the birth rate is computed as the number of births per 1,000 women ages 15 to 45, it potentially confounds two effects: when MMR falls more women are alive, and the incentives to give birth change. We therefore also report regressions of the log of births. We find that when MMR falls the number of birth increases—a 10% change in MMR increases births by about 1 to 2%, although this estimate is also sensitive to specification.[8]

Overall the results suggests that births increase when MMR falls but also that the difference-in-difference estimates are subject to a substantial amount of omitted variable bias and thus should be interpreted with caution. The last row of Table 5a confirms this. It shows the difference-in difference estimates of the effects of MMR of male life expectancy. Absent omitted variables at the district-year level, the coefficient should be 0 since MMR is not a cause of male mortality, but the coefficient is instead statistically significant even when controlling for other causes of death. This pattern motivated our use of male life expectancy as a control variable in the fertility estimates reported in the preceding rows.

In Table 5b we examine the effects of MMR on additional outcomes that are available by gender and thus permit a DDD strategy. We start by looking at the mean age at marriage. As mentioned above, it is not theoretically clear whether we expect a positive or a negative effect, and indeed we find that although the coefficient is positive, suggesting that when MMR falls age at marriage falls disproportionately for girls, it is not significant in any of our specifications. It is also very small in magnitude: a 0.046 coefficient implies that a 10% decline in MMR during the period results in a decrease in the age at marriage of 0.074, which is very small relative to the mean or the change in this period (mean age at marriage rose by about 2 years).

Turning to the effects of MMR on education, we start with the percent of individuals who were illiterate at marriage. The coefficient on MMR*female has the

[8] We also estimated models using the crude birth rate (births divided by population) and estimates of the birth rate from Langford. The results are very similar to those presented here.

expected positive sign (when MMR falls the percent illiterate at marriage falls disproportionately for females) but it is insignificant and small: a 10% decrease in MMR (0.16) results in a 0.3 decrease in the percent illiterate at marriage, about 1.2 percent of the mean for women in 1946.

Another educational outcome that we examine is percent in school among individuals ages 5 to 24. MMR*female has a negative and insignificant effect on this measure. The largest coefficient we estimate (-0.01) implies that a 10% decrease in MMR increases the percentage of girls in school by 0.0016, relative to a mean of 34.6 and a change of 16.9 during the period. This is a very small effect. But it is possible that this variable underestimates changes in education because it includes everyone up to ages 24: only a very small percentage of individuals entered post-secondary school, while the vast majority of individuals obtained some primary or secondary schooling, and the greatest increases in enrollment in the country were observed for lower levels. The limitations of the data, i.e., the lack of a breakdown in school attendance by age, prevent us from drawing strong conclusions about this outcome.

Next we look at literacy by age. This measure is more precise in that it is available by age, but does not tell us directly about enrollment or total years of schooling. We expect the coefficients to be negative mostly for those ages 30 and below. The reasons these ages should be most affected is that their education occurs after the life-expectancy gains have occurred, whereas for older cohorts, there education is determined prior to the health improvement. Our assumption is that literacy is predetermined by the time someone is about 20 to 23 years old (since, for example, the 23-year-olds in 1946 would be 30 in 1953), so that older ages are a "placebo" test. If people become literate at later ages, then older age groups might not be a valid placebo test.

We estimate two different specifications. The first follows our strategy so far and estimates a separate regression for each group. In addition, to improve the precision of the estimates we stack the data and estimate models were we impose the restriction that the gender*district, year*district and female*year dummies are the same across ages, but include age*district, age*year and age*female dummies.

The results (Table 6) are not particularly sensitive to the inclusion of covariates, although they are somewhat smaller in magnitude when death rates from other diseases

are included. When we estimate a single regression the coefficients are almost identical but with smaller standard errors.

We find that a reduction in maternal mortality causes literacy increases for ages 15 through 39 (or 44 in the joint the specification). Theoretically, we might have also expected to find an effect for the lowest ages, but the absence of such a finding may be explained by the fact that in 1946 the literacy rates and therefore the gender gap in literacy was small for very young ages. The 1946 gender gap in literacy is about 3 percentage points for ages 5-9 whereas it is 16 percentage points for ages 10-14, and it grows with age.

It is surprising to find effects for women above age 30. As mentioned above, if literacy is determined prior to age of 20 to 25, then the literacy of these cohorts could not have been affected by the MMR declines that we are using for identification. This finding suggests that there may be gender-specific trends correlated with MMR. To address this, in the third column, we re-estimate the regressions for ages 30 and below controlling for the literacy rates for ages 35 through 65 and over (we add 7 explanatory variables). We find that the results are qualitatively unchanged: the coefficients for ages 15-19, 20-24 and 25-29 are of the same magnitude. We interpret this as evidence that the results for the younger cohorts are unaffected by controlling for pre-period trends.

On average the coefficients in the first column imply that a 10% decrease in MMR increases literacy rates by a bit less than 1%. Declines in MMR can explain about 9% of the change in literacy during the period. A more intuitive way to interpret the coefficients is to consider an elasticity of literacy with respect to adult life expectancy. The coefficient on MMR*female for life expectancy from 15 to 65 is -1.29. The base period life expectancy of women at age 15, censored at 65, is 37.3 years, so a 1 unit decrease in MMR (1 fewer death per 100 births) increases adult life for women by 3.5%. The same MMR*female regressor has a coefficient of about -0.020 for literacy rates of young women, off of a base rate of about .50. Thus a 1 unit decrease in MMR increases literacy rates by 4%. Combining these two calculations, the elasticity of literacy with respect to adult life years is about 1.16. In short, our results for literacy, the educational outcome we can measure at the finest level of disaggregation, suggest that human capital investments are quite responsive to life expectancy.

MMR fell by 1.3 between 1946 and 1963, so the estimate implies a 2.5 percentage point increase in literacy. To get a sense of the increase in schooling corresponding to this literacy increase, we use data from the 1987 DHS in which the average education of literate women is 7.8 years and the average education of illiterate women is 1.6 years. 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 a rough approximation, if the 2.5 point increase in literacy corresponded to that proportion of the population gaining 6.2 years of schooling (7.8 minus 1.2), this corresponds to an average increase in (relative) female education of 0.15 years (from a base of 4.8 years). It is worth noting that this magnitude of schooling gains from a life-expectancy gain of 1.7 years (0.088 years of school per additional life-year) is quite similar to the prediction of a calibrated model by Gan and Gong (2004) in a very different context (black-white gaps in the US) that a 5.9 year gap in life expectancy leads to 0.5 years more of school (0.084 years of school per additional life-year).[9]

VII. Conclusion

To be written

[9] Investments decisions should be affected by *discounted* life expectancy. Given that black-white mortality differences in the U.S. occur at later ages than maternal mortality in Sri Lanka, on average, this adjustment would make the two estimates even closer to one another.

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Data Appendix

District definitions

Over the period studied, some districts divided in which case we aggregate up to the original, larger district, and some districts merged in which case we use the merged district from the outset. In addition, Colombo and Negombo are treated as one district in the censuses, and therefore in our study, despite being separate administrative districts throughout the period. This yields 19 districts, shown in bold.

1946	1953	1963
Colombo	Colombo	Colombo
	Negombo	Negombo
Kalutara	Kalutara	Kalutara
Kandy	Kandy	Kandy
Matale	Matale	Matale
Nuwara Eliya	Nuwara Eliya	Nuwara Eliya
Galle	Galle	Galle
Matara	Matara	Matara
Hambantota	Hambantota	Hambantota
Jaffna	Jaffna	Jaffna
Mannar	Mannar	Mannar
Vavuniya	Vavuniya	Vavuniya
Batticaloa	Batticaloa	Batticaloa (1958)
		Amparai (1958)
Trincomalee	Trincomalee	Trincomalee
Kurunegala	Kurunegala	Kurunegala
Puttalam	Puttalam	**Puttalam (1958)**
Chilaw	Chilaw	
Anuradhapura	Anuradhapura	Anuradhapura (1958)
		Pollonaruwa (1958)
Badulla	Badulla	Badulla
		Monaragala
Ratnapura	Ratnapura	Ratnapura
Kegalia	Kegalia	Kegalia

Conversion from annual data to 1946, 1953, and 1963 time periods

Vital statistics data (births, deaths) is available annually. The values we use for 1946 are the average of 1945 and 1947; the values for 1953 are the average of 1952 and 1954; and the values for 1963 are the average of 1962 and 1964. We average to reduce measurement error, and we exclude the actual year because 1946 was an abnormal year for mortality because of a malaria outbreak.

Interpolation between census years

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

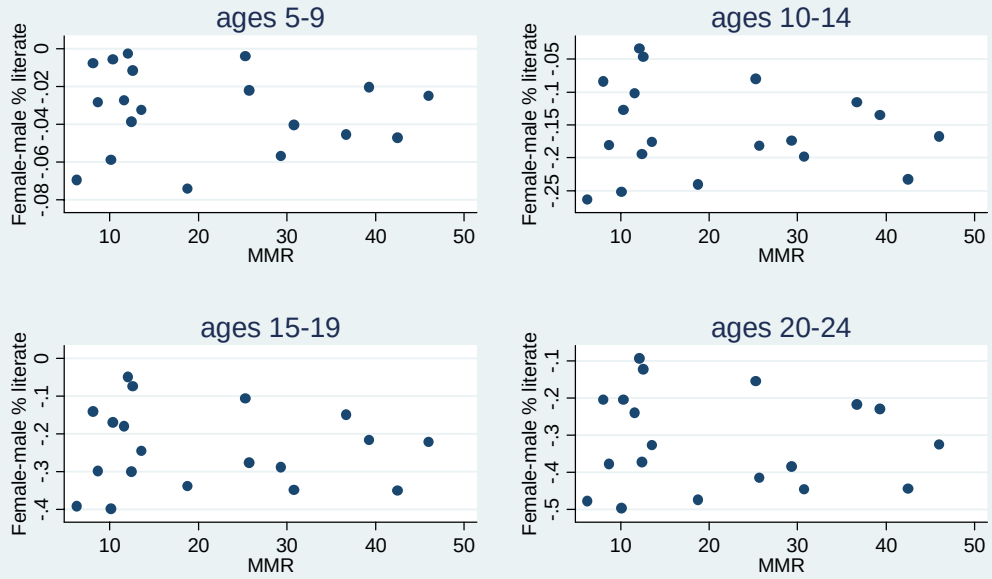
Life expectancy calculation

To calculate life expectancy from mortality tables, we consider an individual who has survived until age 15 and calculate the probability of surviving each subsequent year. The death rate data are for a 5-year age band, and we assume the rate is constant for each age in the band. We treat the deaths as taking place at the midpoint of the year.

Percentage in school ages 5-24

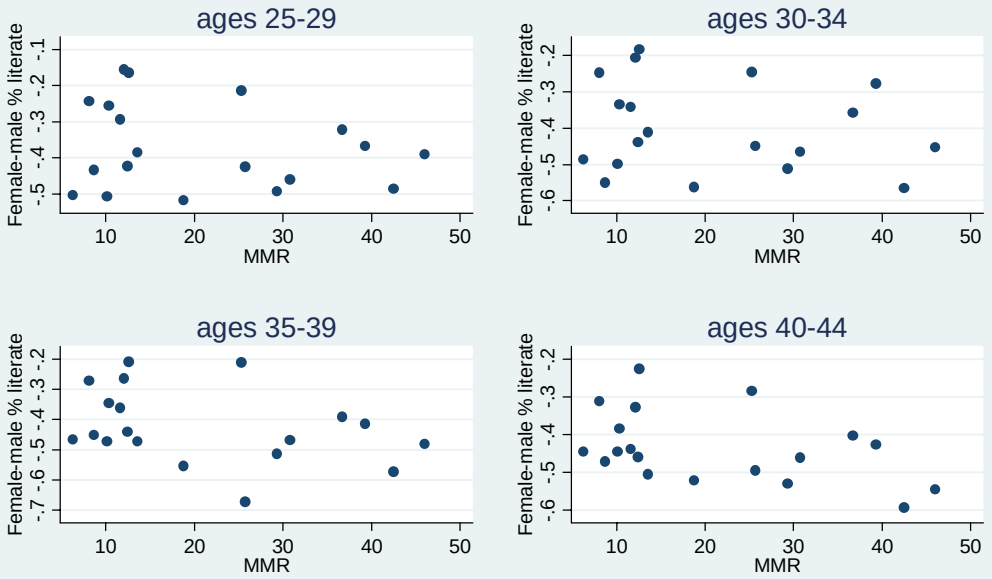
This variable was not reported in the 1963 census tabulations. What was reported instead was the percentage of individuals ages 5 and above who were in school. Breakdowns by age were not available by district, but are available for the entire country. These suggest that only 0.75% of students were above age 25.

Figure 1a: Correlation between maternal mortality and literacy gender gaps in 1946



each dot represents a district

Figure 1b: Correlation between maternal mortality and literacy gender gaps in 1946



each dot represents a district

Figure 2: Expansion in health services in Sri Lanka 1945-1965

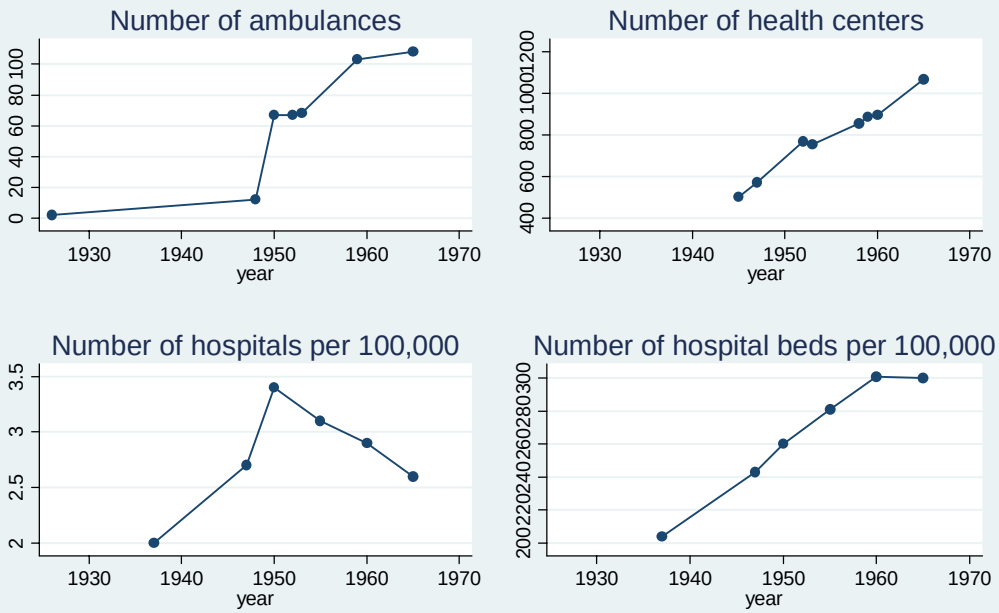
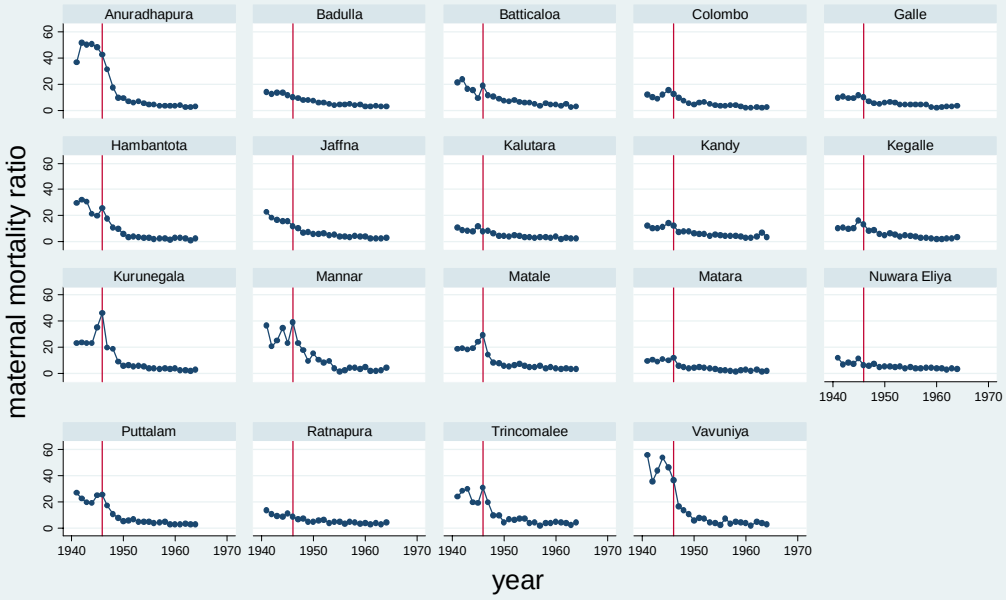


Figure 3a: Trends in maternal mortality 1941-1964 by district



Graphs by district

Figure 3b: Convergence in maternal mortality across districts

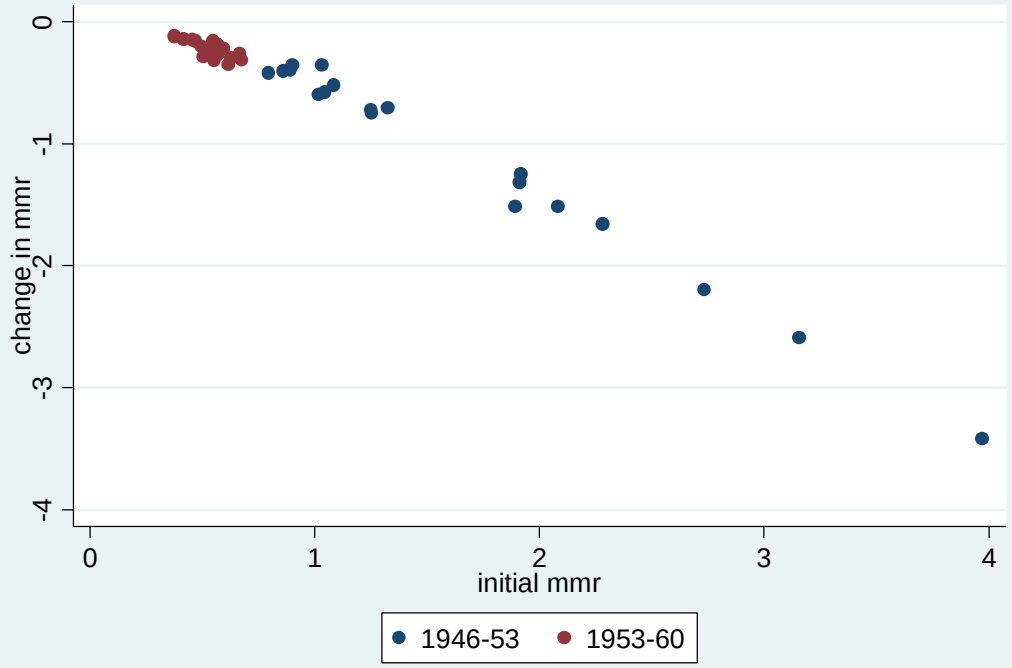
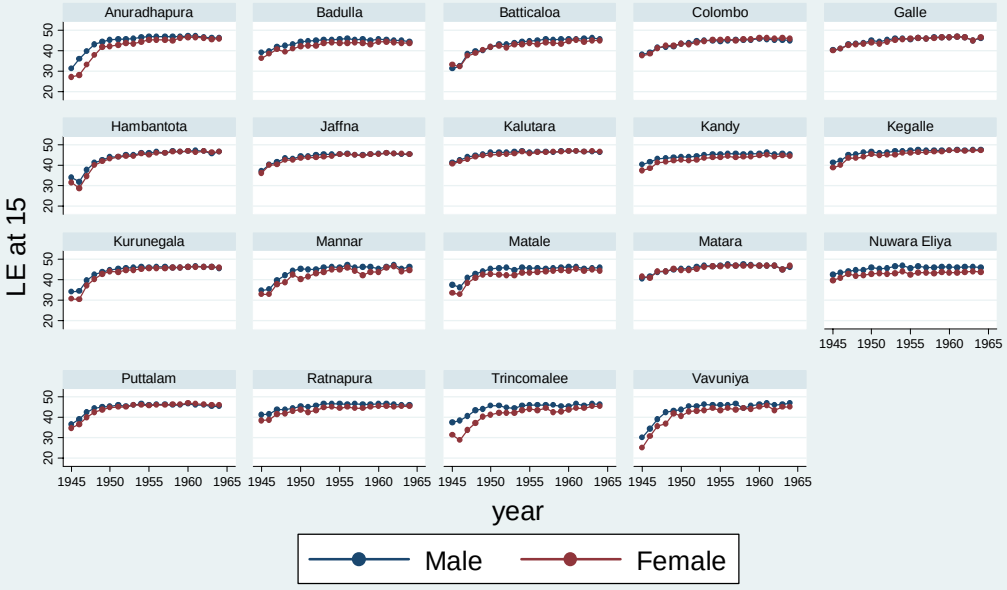
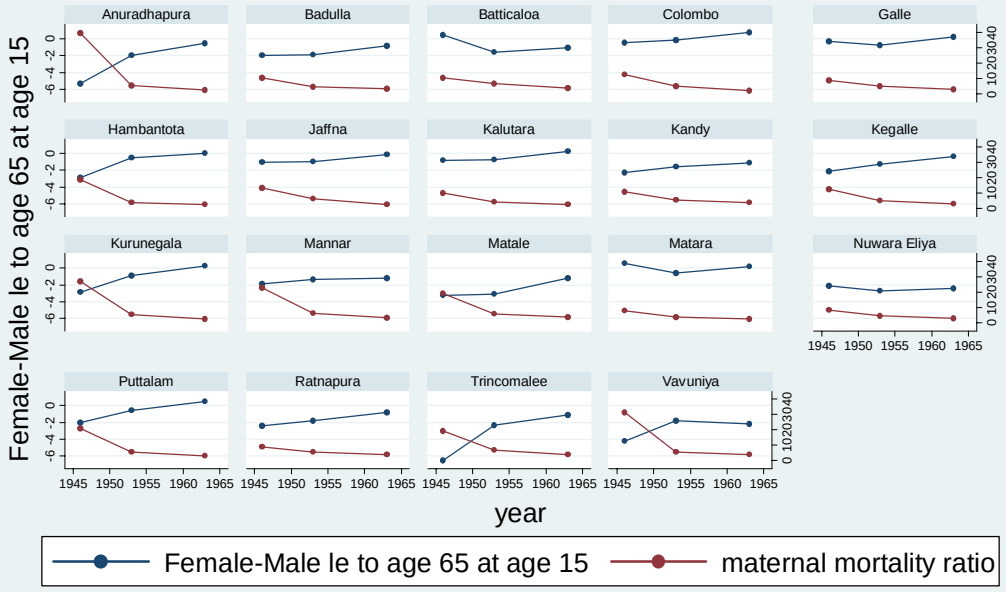


Figure 4: trends in LE at age 15 by district, 1945-1964



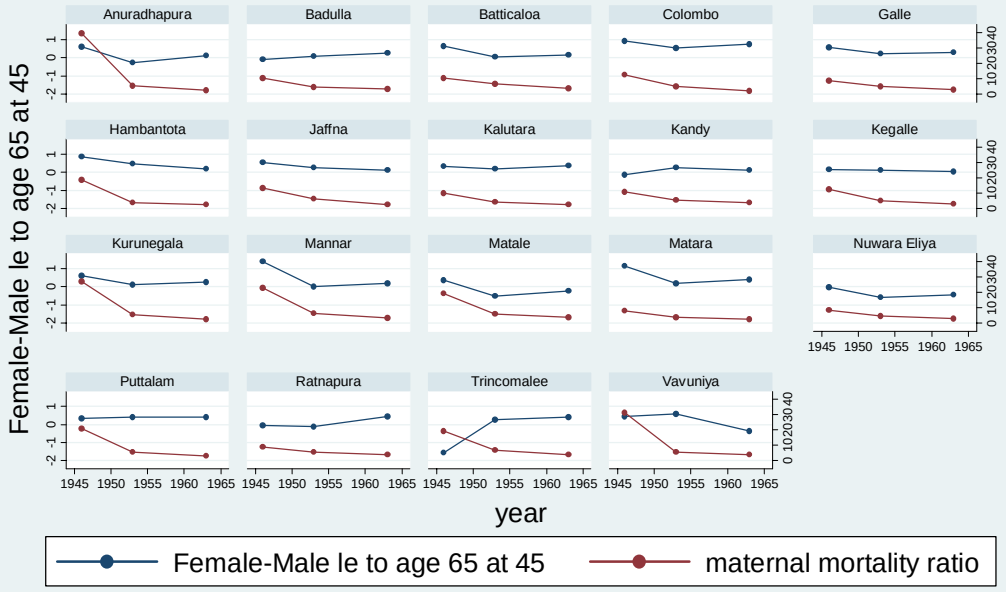
Graphs by district

Figure 5a: trends in female-male LE at 15 and MMR by district, 1945-1964

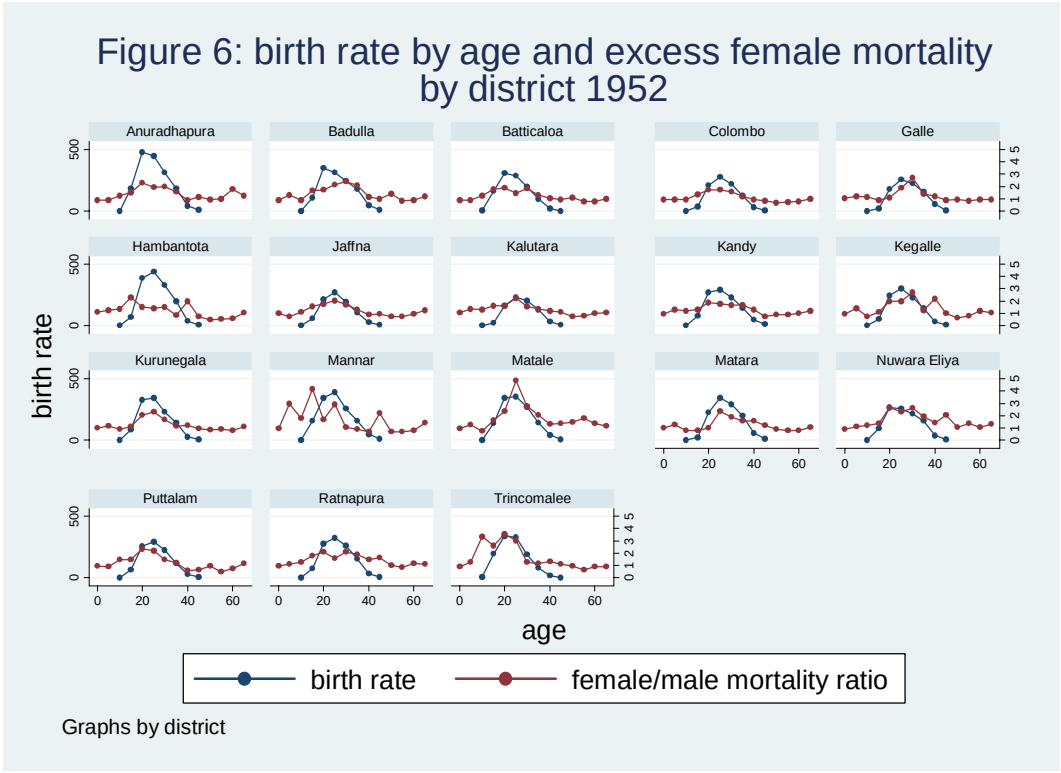


Graphs by district

Figure 5b: trends in female-male LE at 45 and MMR by district, 1945-1964



Graphs by district



Note: Vavuniya is excluded because of scale: due to small numbers, its female/male ratios are very large. When plotting all districts on the same scale, including Vavuniya makes the patterns very difficult to observe.

Figure 7: ratio of female/male death rates from pyrexia by age, Ceylon 1952

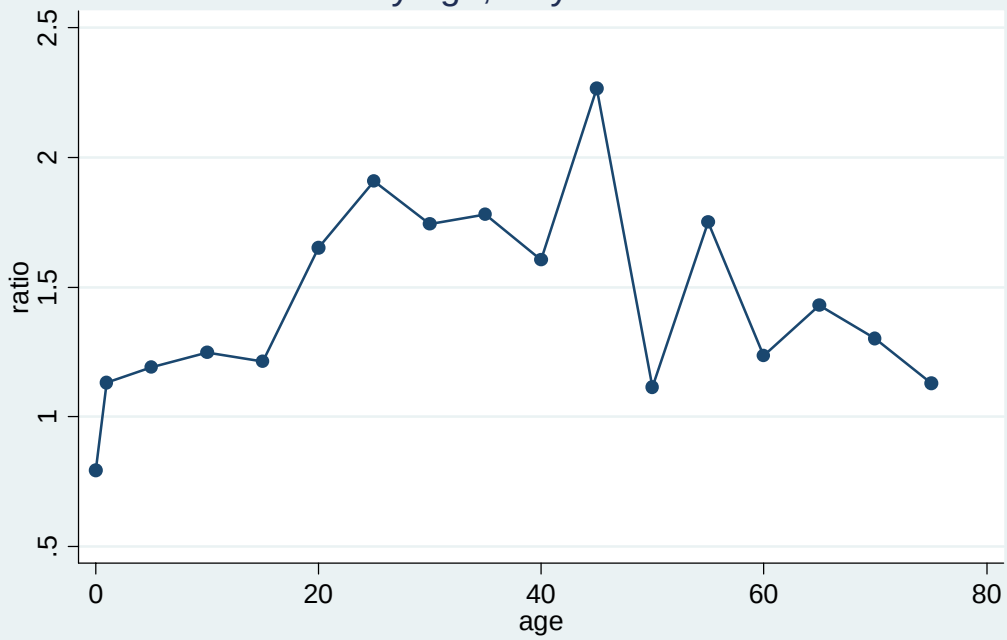


Table 1a: Summary Statistics
Unweighted means across districts

	Males			Females		
	1946	1953	1963	1946	1953	1963
MMR*				1.652	0.533	0.311
LE at 15 (censored at 65)	39.46	45.65	46.14	37.29	44.24	45.57
LE at 15 (censored at 45)	27.04	28.95	29.10	25.62	28.25	28.76
LE at 45 (censored at 65)	15.88	18.23	18.37	16.25	18.36	18.55
Fertility						
Birth rate**				178.90	202.45	187.06
Number of births				13,413	16,248	19,272
Female population 15-45				82,634	95,115	115,611
Marriage						
Mean age at marriage	27.71	27.86	29.18	21.16	21.57	23.17
% illiterate at marriage	6.48	6.68	2.91	26.66	23.64	13.39
Education						
% in school (ages 5-24)	0.37	0.45	0.56	0.35	0.40	0.51
% literate						
Age 5-9	0.28	0.45	0.43	0.25	0.42	0.43
Age 10-14	0.68	0.81	0.85	0.52	0.69	0.80
Age 15-19	0.79	0.82	0.89	0.55	0.64	0.79
Age 20-24	0.82	0.86	0.90	0.50	0.60	0.74
Age 25-29	0.82	0.85	0.88	0.45	0.55	0.66
Age 30-34	0.80	0.84	0.87	0.40	0.49	0.63
Age 35-39	0.77	0.81	0.85	0.35	0.43	0.55
Age 40-44	0.75	0.79	0.84	0.32	0.38	0.50
Age 45-49	0.72	0.75	0.81	0.28	0.34	0.45
Age 50-54	0.70	0.74	0.79	0.25	0.31	0.40
Age 55-60	0.69	0.68	0.76	0.23	0.28	0.37
Age 60-64	0.65	0.68	0.72	0.20	0.23	0.31
Age 60 and above	0.61	0.63	0.55	0.17	0.20	0.21
District level characteristics						
% urban	11.78	11.2	14.54	11.78	11.2	14.54

*#deaths per 100 live births
**Birth rate=[births/female pop(15-45)]*1,000
Number of districts: 19

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

year	1946	1953	1963	1946	1953	1963
	Males			Females		
Disease rates						
Rathe*	8.15	2.32	0.65	7.70	2.00	0.64
Pyrexia	1.84	0.45	0.34	2.26	0.62	0.44
Pneumonia	1.93	0.79	0.44	2.10	0.97	0.52
Diseases of the nervous system**	1.85	1.15	0.69	2.05	1.15	0.65
Vitamin	0.95	0.44	0.20	1.51	0.65	0.29
Malaria	1.17	0.10	0.00	1.28	0.12	0.00
Congenital debilities	1.18	0.71	0.15	1.25	0.64	0.24
Diarrhea	0.83	0.40	0.49	0.89	0.46	0.51
Helminths	0.33	0.33	0.14	0.45	0.46	0.19
Diseases of the circulatory system	0.44	0.34	0.50	0.34	0.27	0.32
Tuberculosis	0.45	0.26	0.13	0.27	0.21	0.08
Anemia	0.34	0.22	0.19	0.32	0.26	0.27
Rheumatic fever	0.30	0.14	0.06	0.31	0.17	0.06
Influenza	0.20	0.09	0.02	0.27	0.11	0.03
Dysentery	0.24	0.09	0.04	0.18	0.10	0.05
Bronchitis	0.19	0.09	0.05	0.19	0.11	0.05
Premature birth*	0.0058	0.0042	0.0038	0.0049	0.0033	0.0030
Pregnancy***	-	-	-	0.0030	0.0010	0.0005
Age-specific rates						
ages 0-4	68.39	35.09	20.07	69.39	33.63	18.80
ages 5-9	6.37	2.98	2.05	7.51	3.54	2.37
ages 10-14	3.46	1.48	1.15	3.97	1.56	1.15
ages 15-19	4.94	1.64	1.38	7.83	2.54	1.75
ages 20-24	6.51	2.20	1.88	12.20	4.06	2.77
ages 25-29	7.92	2.59	2.22	12.63	4.93	3.41
ages 30-34	7.92	2.79	2.34	12.63	5.07	3.49
ages 35-39	12.70	3.74	3.30	12.77	5.53	4.32
ages 40-44	12.70	4.60	4.02	12.77	5.52	4.04
ages 45-49	20.57	6.17	6.20	18.46	6.26	5.37
ages 50-54	20.57	9.34	8.10	18.46	8.59	7.21
ages 55-59	37.92	13.87	12.34	33.64	11.60	10.59
ages 60-64	37.92	20.63	17.83	33.64	18.68	17.39
ages 65+	96.96	67.47	64.69	103.63	76.08	69.34

*denominator is ages 0-4
**mostly convulsions, which are for children under 5.
***denominator is ages 15-45.
Disease-specific and cause-specific rates are per 1,000

Table 2: Effect of maternal mortality on life expectancy

Dependent variable:	year*district, female*district and female*year fe	Drop female*district	Drop year*district	Add malaria death rates	Add nutrition diseases death rates	Add nutritional diseases and malaria death rates
LE 15-65						
MMR*female	-1.288***	-1.469***	-1.288**	-1.289***	-1.355***	-1.359***
	[0.181]	[0.220]	[0.535]	[0.203]	[0.178]	[0.205]
Observations	114	114	114	114	114	114
R-squared	1	0.99	0.96	1	1	1
LE 15-45						
MMR*female	-0.957***	-1.036***	-0.957***	-0.958***	-0.931***	-0.921***
	[0.061]	[0.104]	[0.219]	[0.067]	[0.062]	[0.070]
Observations	114	114	114	114	114	114
R-squared	1	0.99	0.96	1	1	1
LE 45-65						
MMR*female	0.139	0.077	0.139	0.117	0.150*	0.128
	[0.093]	[0.094]	[0.192]	[0.098]	[0.078]	[0.095]
Observations	114	114	114	114	114	114
R-squared	0.99	0.97	0.92	0.99	0.99	0.99

Nutrition diseases are helminths, anemia and vitamin deficiencies.
Robust standard errors reported in brackets.
Each cell reports the coefficient from a separate regression.
In addition to the controls listed in the column heading, the regressions control for a female dummy.
N=114 (19 districts, 2 genders and 3 years).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 3: The effect of maternal mortality on age-specific mortality rates

Dependent variable: age specific death rate	year*district, female*district and female*year fe	Drop female*district	Drop year*district	Add malaria death rates	Add nutritional diseases and malaria death rates
Age 0-4					
MMR*female	1.536	1.62	1.536	1.62	0.07
	[1.350]	[1.524]	[3.311]	[1.524]	[0.598]
Age 5-9					
MMR*female	0.259	0.204	0.259	0.204	0.318***
	[0.238]	[0.253]	[0.301]	[0.253]	[0.107]
Age 10-14					
MMR*female	0.173	0.065	0.173	0.065	0.025
	[0.198]	[0.192]	[0.290]	[0.192]	[0.268]
Age 15-19					
MMR*female	3.156***	3.305***	3.156***	3.305***	2.450***
	[0.586]	[0.611]	[0.757]	[0.611]	[0.231]
Age 20-24					
MMR*female	5.483***	5.506***	5.483***	5.506***	4.712***
	[0.596]	[0.661]	[0.756]	[0.661]	[0.264]
Age 25-29					
MMR*female	1.953***	1.903***	1.953**	1.903***	2.572***
	[0.499]	[0.499]	[0.772]	[0.499]	[0.321]
Age 30-34					
MMR*female	2.276***	2.304***	2.276***	2.304***	2.700***
	[0.340]	[0.378]	[0.721]	[0.378]	[0.379]
Age 35-39					
MMR*female	-0.853	-1.025	-0.853	-1.025	0.06
	[0.958]	[1.071]	[1.002]	[1.071]	[0.495]
Age 40-44					
MMR*female	-0.793	-0.892	-0.793	-0.892	0.178
	[0.872]	[0.982]	[0.972]	[0.982]	[0.490]
Age 45-49					
MMR*female	-0.386	-0.28	-0.386	-0.28	-1.156**
	[0.798]	[0.847]	[1.346]	[0.847]	[0.539]
Age 50-54					
MMR*female	-0.201	-0.073	-0.201	-0.073	-1.186**
	[0.967]	[1.058]	[1.226]	[1.058]	[0.537]
Age 55-59					
MMR*female	-4.488**	-4.075*	-4.488*	-4.075*	-0.828
	[2.027]	[2.268]	[2.402]	[2.268]	[1.288]
Age 60-64					
MMR*female	-5.478**	-5.069*	-5.478**	-5.069*	-1.141
	[2.689]	[2.961]	[2.686]	[2.961]	[1.905]
Age 65+					
MMR*female	-1.679	-1.433	-1.679	-1.433	-4.041*
	[3.661]	[4.180]	[4.073]	[4.180]	[2.273]

Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. In addition to the controls listed in the column heading, the regressions control for a female dummy. N=114 (19 districts, 2 genders and 3 years).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 4: The effect of maternal mortality on disease-specific mortality rates

Dependent variable: cause-specific death rate	year*district, female*district and female*year fe	Drop female*district	Drop year*district
Panel A: Diseases affecting children under 5 only			
Rathe			
MMR*female	-0.354**	-0.354	-0.389**
	[0.140]	[2.407]	[0.183]
Diseases of the nervous system (convulsions)			
MMR*female	0.134**	0.134	0.208***
	[0.053]	[0.197]	[0.058]
Congenital debilities			
MMR*female	0.125***	0.125	0.172***
	[0.036]	[0.255]	[0.056]
Premature birth			
MMR*female	0	0	0
	[0.000]	[0.000]	[0.000]
Panel B: Diseases for adults			
Pyrexia (fever)			
MMR*female	0.327***	0.327	0.400***
	[0.106]	[0.404]	[0.143]
Pneumonia			
MMR*female	0.249	0.249	0.31
	[0.156]	[0.799]	[0.206]
Vitamin			
MMR*female	0.305	0.305	0.285
	[0.285]	[0.516]	[0.330]
Malaria			
MMR*female	0.058	0.058	0.067
	[0.054]	[0.154]	[0.060]
Diarrhea			
MMR*female	0.022	0.022	0.038
	[0.039]	[0.094]	[0.026]
Helminths			
MMR*female	0.009	0.009	0.007
	[0.024]	[0.069]	[0.027]
Diseases of the circulatory system			
MMR*female	0.004	0.004	0.050**
	[0.028]	[0.045]	[0.022]
Tuberculosis			
MMR*female	-0.056***	-0.056*	-0.071***
	[0.016]	[0.029]	[0.022]
Anemia			
MMR*female	0.035	0.035	0.014
	[0.026]	[0.042]	[0.030]
Rheumatic fever			
MMR*female	-0.019	-0.019	-0.009
	[0.015]	[0.033]	[0.018]

Table 4 continued

	year*district, female*district and female*year fe	Drop female*district	Drop year*district
Influenza			
MMR*female	0.004	0.004	0.002
	[0.018]	[0.074]	[0.017]
Dysentery			
MMR*female	-0.021	-0.021	-0.01
	[0.016]	[0.022]	[0.012]
Bronchitis			
MMR*female	-0.023*	-0.023	-0.029*
	[0.012]	[0.029]	[0.016]

Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. In addition to the controls listed in the column heading, the regressions control for a female dummy. N=114 (19 districts, 2 genders and 3 years).

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

Table 5a: Effect of maternal mortality on outcomes
Difference in difference estimates for fertility

	Year and district fe	Add malaria death rates	Add nutrition diseases death rates*	Add nutritional diseases and malaria death rates	Add male life expectancy 15-65	Add male life expectancy 15-65, malaria and nutrition death rates
Birth rate						
MMR	-28.216***	-18.790**	-19.438**	-11.996	-2.713	-4.27
	[7.974]	[8.845]	[8.661]	[8.547]	[8.805]	[8.340]
Observations	57	57	57	57	57	57
R-squared	0.81	0.84	0.88	0.9	0.88	0.91
Log(# Births)						
MMR	-0.241***	-0.254***	-0.204***	-0.226***	-0.111**	-0.162***
	[0.037]	[0.053]	[0.037]	[0.050]	[0.046]	[0.045]
Observations	57	57	57	57	57	57
R-squared	0.99	0.99	0.99	0.99	0.99	0.99
Male LE 15-65						
MMR	-2.504***	-1.813***	-1.997***	-1.516**		
	[0.401]	[0.626]	[0.452]	[0.583]		
Observations	57	57	57	57		
R-squared	0.94	0.96	0.96	0.97		

Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. N=57 (19 districts and 3 years).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 5b: Effect of maternal mortality on outcomes
Difference in difference in difference estimates for marriage and enrollment

	year*district, female*district and female*year fe	Drop female*district	Drop year*district	Add malaria death rates	Add nutrition diseases death rates	Add nutritional diseases and malaria death rates
Mean age at marriage						
MMR*female	0.046	-0.306	0.046	0.001	0.066	0
	[0.099]	[0.184]	[0.302]	[0.086]	[0.107]	[0.118]
Observations	114	114	114	114	114	114
R-squared	1	0.99	0.96	1	1	1
Percent illiterate at marriage						
MMR*female	1.927	-0.576	1.927	1.387	2.495**	1.688
	[1.187]	[2.809]	[1.891]	[1.220]	[1.051]	[1.288]
Observations	114	114	114	114	114	114
R-squared	0.99	0.87	0.96	0.99	0.99	0.99
Percent in school (ages 5-24)						
MMR*female	-0.008	0.011	-0.008	-0.005	-0.01	-0.003
	[0.007]	[0.009]	[0.027]	[0.008]	[0.008]	[0.009]
Observations	114	114	114	114	114	114
R-squared	0.99	0.98	0.84	0.99	0.99	0.99

Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. In addition to the controls listed in the column heading, the regressions control for a female dummy. N=114 (19 districts, 2 genders and 3 years).
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 6: Effect of maternal mortality on literacy rates

	Individual regressions by age			Single regression, controlling for age*district age*year and age*female fe (cluster female*district*year)
	year*district, female*district and female*year fe	Add nutritional diseases and malaria	Control for literacy at older ages (35 up to 65 and above)	
Ages 5-9				
MMR*female	-0.007*	-0.006*	-0.004	0.015
	[0.004]	[0.003]	[0.004]	[0.010]
Ages 10-14				
MMR*female	-0.008	-0.008	-0.019	-0.011
	[0.015]	[0.016]	[0.015]	[0.010]
Ages 15-19				
MMR*female	-0.020	-0.022*	-0.021*	-0.020*
	[0.012]	[0.012]	[0.011]	[0.010]
Ages 20-24				
MMR*female	-0.018	-0.026***	-0.023	-0.023**
	[0.013]	[0.009]	[0.014]	[0.009]
Ages 25-29				
MMR*female	-0.026***	-0.019*	-0.018***	-0.031***
	[0.009]	[0.010]	[0.006]	[0.007]
Ages 30-34				
MMR*female	-0.017	-0.02		-0.024***
	[0.011]	[0.013]		[0.008]
Ages 35-39				
MMR*female	-0.032**	-0.027**		-0.023**
	[0.015]	[0.012]		[0.009]
Ages 40-44				
MMR*female	-0.012	-0.016		-0.014**
	[0.015]	[0.012]		[0.007]
Ages 45-49				
MMR*female	-0.009	-0.014		-0.013
	[0.014]	[0.010]		[0.008]
Ages 50-54				
MMR*female	-0.008	-0.009		-0.01
	[0.016]	[0.011]		[0.010]
Ages 55-59				
MMR*female	0.009	-0.001		-0.016
	[0.018]	[0.013]		[0.010]
Ages 60-64				
MMR*female	-0.015	-0.011		0.001
	[0.014]	[0.010]		[0.010]
Ages 65+				
MMR*female	-0.007	-0.005		-0.004
	[0.010]	[0.009]		[0.012]
Observations	114	114	114	1482
R-squared	NA	NA	NA	0.98

Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. In addition to the controls listed in the column heading, the regressions control for a female dummy. * significant at 10%; ** significant at 5%; *** significant at 1%

Appendix Table 1: Classification if diseases 1945-1965

Disease	line in table	1945-1949 classification	line in table	1950-1965 classification
TB	13	Tuberculosis of respiratory system	1	Tuberculosis of respiratory system (001-008)
Dysentery	27	Dysentery (a) Bacillary (b) Amoebic (c) Other and Unspecified forms of dysentery	16	Dysentery all forms (045-048)
Malaria	28	Malaria (a) benign tertian (b) quartan (c) tropical malignant tertian (d) Blackwater fever (e) Malarial cachexia (f) other and unspecified malaria	37	Malaria (110-117)
Influenza	33	Influenza (a) with respiratory complications specified (b) without respiratory complications specified	88	Influenza (480-483)
Helminths	42	Other disease due to helminths (a) Round worms (b) Tapeworms (d) Others	42	Other diseases due to Helminths (124, 126, 128, 130)
Rheumatic fever	58	Rheumatic fever (a) Acute rheumatic pericarditis (b) Acute rheumatic endocarditis (c) Acute rheumatic myocarditis (d) Other forms, including acute articular rheumatism and rheumatic pleurisy (e) Rheumatic chorea (f) Others	79	Rheumatic fever (400-402)

Appendix Table 1 continued

Disease	line in table	1945-1949 classification	line in table	1950-1965 classification
Vitamin		Vitamin deficiency Diseases	64	Avitaminoses and other deficiency states (280, 286)
	67	Scurvy		(a) Mandama
	68	Beriberi		(b) Others
	69	Pellagra (except alcoholic)		
	70	Rickets		
	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)		
Diseases of the nervous system	83	Intra-cranial lesions of vascular origin	78	All other diseases of central nervous system and sense organs (341-344, 350-352, 354-369, 380-384, 386, 388-390, 394-398)
		d-Hemiplegia and other paralysis of unstated origin		(a) Hemiplegia and other paralysis
	86	convulsions in children under 5 years of age		(b) Convulsions (under 5 years)
Diseases of the circulatory system	VII	Diseases of the circulatory System		
			80	Chronic Rheumatic heart disease (410-416)
			81	Arteriosclerotic and degenerative heart disease (420-422)
			82	Other diseases of heart (430-434)
			83	Hypertension with heart disease (440-443)
Bronchitis	106	Bronchitis	93	Bronchitis chronic and unqualified (501, 502)
		(b) Chronic		

		(c) Bronchietiectasis		
		(d) Unspecified		
Pneumonia		Pneumonia		Pneumonia
	108	Lobar pneumonia	89	Lobar pneumonia (490)
	109	Pneumonia unspecified	91	Primary atypical pneumonia, other and unspecified onemonia (492-493)

Appendix Table 1 continued

Disease	line in table	1945-1949 classification	line in table	1950-1965 classification
Diarrhea	119 120	Diarrhea enteritis and ulceration of the intestines (under 2 years of age) (a) Diarrhea and enteritis (b) Ulceration of the intestines (except duodenum) Diarrhea enteritis and ulceration of the intestines (2 years of age and over) (a) Diarrhea and enteritis (b) Ulceration of the intestines (except duodenum)	104	Gastro-enteritis and colitis, except diarrhea of the newborn (571, 572)
Pregnancy	XI	Diseases of pregnancy, childbirth and the puerperium	115 116 117 118 119 120	Sepsis of pregnancy, Childbirth and the puerperium (640, 641, 681, 682, 684) (a) puerperal sepsis (b) others Toxaemias of pregnancy and the puerperium (642, 652, 685, 686) (a) puerperal eclampsia (b) Others Haemorrage of pregnancy and childbirth (643, 644, 670-672) Abortion without mention of sepsis or toxaemia (650) Abortion with sepsis (651) Other complications of pregnancy, childbirth and the puerperium (645-649, 673-680, 687-689)
Congenital debilities	158	Congenital debility (cause not stated)		(b) congenital debility
Premature birth	159	Premature birth (cause not stated)		(a) Immaturity

Rathe	161	Other diseases peculiar to the first year of life e-Rata	126	Other disease of skin and musculoskeletal system (700-716, 731-736, 738-744) (a) Rathe erythematous conditions)
Pyrexia	200	Ill-defined and Unknown causes of death c-Pyrexia	137	Ill-defined and unknown causes (780-793, 795) (b) Pyrexia

Appendix Table 2: Convergence in MMR across districts

Dependent Variable: MMR change				
	1946-1953	1953-1963	All	All
Panel A: no other covariates except year				
MMR level	-0.9719***	-0.6399***	-0.9687***	-0.6399***
	[0.0168]	[0.0822]	[0.0171]	[0.0822]
mmr_early				-0.3320***
				[0.0839]
R-squared	0.99	0.58	0.99	0.99
Panel B: Control for initial level of other diseases				
MMR level	-1.0977***	-0.3742**	-1.0412***	
	[0.0673]	[0.1635]	[0.0754]	
R-squared	0.99	0.77	0.99	

There are 19 districts, thus regressions contain either 19 or 38 observations. Robust standard errors are reported in parenthesis. The regressions do not include any other controls.

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

Appendix Table 3: Convergence by disease: are there gender differences?

Dependent variable: Change in death rate for disease report Initial level*female	1946-1953	1953-1963	All
Rathe	-0.022	0.0555	-0.023
	[0.0685]	[0.0614]	[0.0631]
Diseases of central nervous system	-0.0905	-0.0543	-0.0692
	[0.0717]	[0.0781]	[0.0542]
Congenital debilities	-0.0282	0.1946***	-0.0646
	[0.1192]	[0.0321]	[0.1091]
Premature Birth	-0.0713	0.1662	-0.0337
	[0.2167]	[0.4189]	[0.1955]
Pyrexia	0.0346	-0.1164	0.0394
	[0.0328]	[0.1236]	[0.0299]
Pneumonia	0.0453	-0.0579	0.0337
	[0.0942]	[0.0955]	[0.0951]
Vitamin deficiencies	-0.1324	0.0062	-0.1225
	[0.1951]	[0.0702]	[0.1608]
Malaria	-0.0077	0.0014	0.0029
	[0.0398]	[0.0144]	[0.0229]
Diarrhea	-0.0507	-0.2869	0.0059
	[0.2668]	[0.4447]	[0.1284]
Helminths	-0.0085	-0.0299	-0.0354
	[0.1218]	[0.1360]	[0.1696]
Diseases of the circulatory system	0.019	0.0193	0.0774
	[0.1152]	[0.2986]	[0.1748]
Tuberculosis	0.0001	-0.077	0.0073
	[0.0774]	[0.1219]	[0.0805]
Anemia	-0.2514	0.037	-0.155
	[0.1588]	[0.2042]	[0.1290]
Rheumatic fever	0.0232	0.0287	0.0304
	[0.2489]	[0.1339]	[0.1694]
Influenza	0.0005	0.0099	-0.001
	[0.0344]	[0.0221]	[0.0414]
Dysentery	0.2412	0.1351	0.2397**
	[0.1747]	[0.1631]	[0.1056]
Bronchitis	0.074	-0.033	0.0619
	[0.0912]	[0.2677]	[0.0937]

Appendix Table 4: Specification checks

	population weights	Standard errors clustered by district and gender
LE 15-65	-1.354***	-1.288***
	[0.153]	[0.236]
LE 15-45	-0.931***	-0.957***
	[0.054]	[0.084]
LE 45-65	0.146*	0.139
	[0.079]	[0.104]
Mean age at marriage	0.164	0.046
	[0.161]	[0.097]
%illiterate at marriage	2.468**	1.927
	[1.115]	[1.436]
Birth rate	-34.044***	-28.216**
	[8.781]	[11.204]
% in school (ages 5-24)	-0.015	-0.008
	[0.010]	[0.010]
% literate (joint regression)		
Ages 5-9	0.027	0.015
	[0.021]	[0.011]
ages 10-14	-0.007	-0.011
	[0.016]	[0.013]
ages 15-19	-0.025**	-0.02
	[0.012]	[0.012]
Ages 20-24	-0.038***	-0.023***
	[0.012]	[0.008]
Ages 25-29	-0.038***	-0.031***
	[0.010]	[0.006]
Ages 30-34	-0.038***	-0.024***
	[0.010]	[0.007]
Ages 35-39	-0.030***	-0.023***
	[0.010]	[0.008]
Ages 40-44	-0.030**	-0.014**
	[0.011]	[0.006]
Ages 45-49	-0.028**	-0.013
	[0.011]	[0.009]
Ages 50-54	-0.026*	-0.016
	[0.013]	[0.011]
Ages 55-59	-0.021*	-0.01
	[0.012]	[0.012]