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. Most previous research designs have been unable to isolate whether health improvements encourage human capital accumulation specifically via life-expectancy effects. This paper uses a type of mortality well-suited to this task. We examine maternal mortality declines in Sri Lanka between 1946 and 1963. Maternal mortality was a major killer of prime-age women, and 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 life expectancy by 1.7 years (a 4.5% increase in prime-age years), and increased female literacy by 7.2%. 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 the investment lasts 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 can 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 health improvements and growth, education or investment. This literature has found mixed evidence on the quantitative importance of improvements in life expectancy on growth and other outcomes. Shastry and Weil (2003) and Lorentzen, McMillan and Wacziarg (2005) find that the effects are large, while Acemoglu and Johnson (2006) find small effects.[1] 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 presents microempirical evidence on the effects of health on education, but does not 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 enrollment. 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 hone in empirically on 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 is Sri Lanka between 1946 and 1963. In 1946, the maternal mortality ratio (MMR) was 1650 maternal deaths per 100,000 live births, or 1.65%. By 1953, this had fallen to 530 and by 1963 to 310. In other words, there was a 70% reduction over the seven-year time span of 1946-53 and another 40% decline over the

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

next decade. These declines were large, occurred over a very short period, and varied considerably across Sri Lanka.

Maternal mortality has several advantages as a way to identify the effects of life expectancy. First, maternal mortality occurs after major human capital investments are made. Therefore, investment decisions will depend on this mortality risk, unlike infant mortality. 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 controls. Finally, there is not much morbidity corresponding to maternal mortality, in contrast to, say, malaria and HIV/AIDS which cause both mortality and substantial morbidity.[2] This feature makes maternal mortality well-suited to isolating the incentive effects of longer life expectancy.

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 that occurred between 1946 and 1963 increased female adult life expectancy by 1.7 years (a 4.5% increase in expected prime-age years[3]), and accounted for all of the difference in life expectancy gains between men and women. These declines in maternal mortality increased female literacy by 7.2%. 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. In section III we discuss our empirical strategy, its advantages and potential caveats. The data and some general background on Sri Lanka are presented in section IV. Section V shows the effects of maternal mortality on life expectancy, and section VI looks at its effects on outcomes. Section VII concludes.

[2] Fortson (2007) finds a negative effect of HIV prevalence on school attendance in sub-Saharan Africa that extends beyond the direct effects from morbidity or income effects due to sick parents. She interprets the results as due in large part to lower expected lifetime returns to education due to mortality risk.

[3] We use the term prime-age years to denote years between the ages of 15 and 65. These are the years over which we assume that returns on human capital mainly accrue.

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 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 (Haveman and Wolfe 1984). Other potential benefits for a woman of being better educated 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 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).[4]

[4] Dow, Philipson, and Sala-i-Martin (1999) discuss theoretically how health investments respond to an exogenous improvement in longevity and provide evidence that other health measures improved in response to vaccination availability. Oster (2007) finds evidence that HIV/AIDS avoidance behavior is more

An 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, or in our context they did not anticipate the increases in access to health care) 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_w about whether to have a child and then choosing the years of schooling s of the child (after observing the child's gender). We use the subscript w to denote wife, h to denote husband, b to denote boy and g to denote girl. We abstract from the next generation's childbearing decision and assume girls have a child with probability C_g, which we treat as exogenous. 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. We assume the household's maximand is the sum of the wife's, the husband's and the child's present discounted value of income. This maximand 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, the wife lives until time T_w. The husband faces no longevity uncertainty and lives until T_h. Households have a discount rate δ. We assume the labor market return to schooling is γ, and (instantaneous) income is y for someone with no schooling.

The household's maximization problem is

$$\max_{s_b, s_g, C_w} \left[U_w(C_w) + U_h + \frac{C_w}{2}(U_b(s_b) + U_g(s_g)) \right]$$

where

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

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pronounced when there is lower non-HIV mortality, e.g., due to lower malaria prevalence or maternal mortality risk.

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

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 mother and father 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

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

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

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

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

$$\frac{\partial^2 s_g}{\partial \mu \partial \tau} > 0 \,. \tag{2.2}$$

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.

(2.3) $$\frac{\partial s_b}{\partial \mu} = 0$$

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 maternal mortality rate also affects the decision to have a child:

(2.4) $$\frac{\partial C_w}{\partial \mu} < 0$$

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). The maternal mortality rate 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 so will 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 (dμ) also applies to those making the fertility decision, and it applies conditional on having a child. The fertility effect, therefore, will be stronger.

Another implication of the model is that with heterogeneity in women's risk of other causes of death, the marginal women who survive past childbearing years when MMR falls will have a relatively shorter lifespan, i.e., they will be the relatively less healthy ones. To see this, consider that for those who expect to live longer (higher T_w), childbearing will be more responsive to the maternal mortality rate,

(2.5) $$\frac{\partial^2 C_w}{\partial \mu \partial T_w} < 0 .$$

Having more children when MMR falls ($\partial C_w/\partial \mu<0$) undoes some of the survival benefit of the MMR decline. Because the women who expect to live longer if they survive childbearing will have a bigger fertility increase in response to an MMR decline, they will experience a smaller net effect on their probability of surviving childbearing (given by 1-

$C_w\mu$). As long as $\partial C_w/\partial\mu$ is not so large that MMR declines cause life expectancy to *fall*, then

$$\frac{\partial^2(1-C_w\mu)}{\partial\mu\partial T_w} > 0. \tag{2.6}$$

In words, the probability of surviving childbearing years is less sensitive to changes in maternal mortality rates (less negative) when lifespan post-childbearing is longer. The empirical prediction implied by (2.6) is that when MMR falls, the deaths rates for women at older ages will increase, and that the death rates increases will be especially pronounced relatively early in the post-childbearing ages.

Other effects

An aspect of this decision problem not in the model is the "quantity-quality" tradeoff. As modeled, the choice of how much to educate a child is separable from the choice of whether to have a child. However, if a household responds to the maternal mortality decline by having more children—because MMR in essence raised the price of quantity—then under alternative assumptions, households might shift from quality to quantity. The household might have more children and 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.[5] On the other hand, if the expected lifetime returns to female education increase when MMR falls, then parents might shift from quantity to quality and have fewer children but educate each one more (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.[6] But again this would not necessarily imply differences in how parents educate boys versus girls.

Also, 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

[5] Another channel through which child quality could fall is if there is cohort crowding and school quality falls, though again this would primarily predict a level drop in educational attainment.
[6] 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.

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 (men) typically remarried but widows (women) 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 earlier or later. Also note that there may be a spillover effect on men's age at marriage because of positive assortative matching on age between wife and husband.

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. The magnitude of the MMR declines varied considerably across Sri Lankan districts. The third difference is between genders; maternal mortality is quite unique among major causes of death in that it exclusively affects women.

The estimating equation is

$$(3.1)\qquad \begin{aligned} Y_{dtg} = \; & \beta_0 + \beta_1 MMR_{dt} * female + \\ & \gamma_{dt}(district * year) + \delta_{dg}(district * female) + \nu_{tg}(year * female) + \varepsilon_{dtg} \end{aligned}$$

where *d* stands for district (19), *t* for year (3) and *g* for gender (2). The specification contains district*year fixed effects, district*female fixed effects, and year*female fixed effects (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).[7]

Even though for some outcomes data are 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 most likely to lead to changes in subjective life expectancy.[8] The use of long differences also minimizes the effect of measurement error in the presence of fixed effects, since measurement error is likely to be small relative to the changes that took place over the 20 years 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 35 year-olds), as opposed to children or older adults. The theoretical effect of life expectancy that the empirical literature has aimed to test is that mortality reductions lead to more investment because individuals have longer horizons over which to reap the returns on investment. However, much of the historical improvement in life expectancy which previous research has used to identify the impact of mortality reductions on investments was driven by declines in infant and child mortality, that is, mortality before the age at which human capital investments are even made. A reduction in prime-age mortality is also preferable to a reduction in elderly mortality, for reasons of statistical precision; the latter 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

[7] We re-estimated models clustering standard errors at the district-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.

[8] 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 contemporaneous MMR predicts behavior much more than lagged MMR.

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.

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 that declines in maternal mortality could be 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 for girls. Another potential confound is that Sri Lanka made dramatic gains in malaria eradication over the time period of 1946 to 1953. There were two other important programs that we are aware of that targeted health: nutrition programs, such as free milk distribution, and intestinal worm eradication. Again, these diseases affected both males and females, but we also address this concern by controlling for gender-specific malaria and nutrition-related death rates (we refer to anemia and vitamin deficiencies and helminthes—which also cause anemia and malnutrition—jointly as nutrition diseases hereafter).

Another 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. In particular, 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. Districts with initially higher maternal mortality risk experienced bigger MMR declines over the period; in other words, there was convergence, as we show below. We can test whether gender gaps in education are initially larger in places with more maternal mortality. If this were the case, and if districts also tend to converge over time with respect to gender gaps in education, then this might generate a spurious correlation between MMR and female education. 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, 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.

The death of a mother might also directly affect girls' education relative to boys' education if mothers are more "pro-daughters" 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 mother survives childbearing with 96.8% probability when there is a 1.6% maternal mortality risk (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. It is mathematically impossible to explain a 3.6 percentage point increase in girl's literacy caused by MMR declines from 1946-63 (to preview an actual result) even under the extreme assumptions that every girl whose mother had died had a 0% chance of becoming literate and every girl whose mother was alive had a 100% chance of being literate. The overall literacy rate was 50%, and one also wants such a calculation to match this aggregate statistic. In the extreme case where girls without mothers are certain to be illiterate, and girls with mothers have (just above) the population-average probability, the direct effects could only account for a third 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 raises 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, for example. One can imagine these spillovers being either positive or negative. 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 direct effects and spillovers effects, but spillovers on boys seem unlikely to be of the same order of magnitude as the direct effects on girls.

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 during 1945-1963 as today had much higher educational participation and gender equality in education than most poor countries.[9] Importantly, there were no major gender-specific educational policy changes during the period we study. The education system 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 open to both genders) and at the university were abolished. Enrollment at primary and secondary levels increased tremendously during this period, in large part because of a transition from

[9] 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.

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 the United Kingdom and 40% in the United States.) (Siriwardena, 1973).

The total fertility rate in Sri Lanka in 1946 was approximately 5. The birth rate (births per 1000 females ages 15 to 45) increased from 179 to 202 between 1946 and 1953, and then decreased between to 187 in 1963. Sri Lanka appears to have entered its "fertility transition" (period of declining fertility) toward the end of the 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 limited role in Sri Lanka (Caldwell 1999). The mean age of marriage in 1946 was 22.4 for females and 28.3 for males.

Sri Lanka's 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. Investigations into causes of maternal mortality suggest that hospital delivery was particularly helpful in preventing deaths from hemorrhage (De Silva 1943). Figure 2 shows that for the entire country there was a large increase in the number of ambulances, health centers, and hospital beds per capita (the number of hospitals per capita increased but population increased more in the 1950s). The proportion of women

delivering at health clinics or hospitals rather than at home increased dramatically 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. These technologies had been proved to dramatically reduce maternal mortality in the West (Loudon 2000a, 1991, 1988, Paxton et al 2004, Lerberge and De Brouvere 2001).

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 may affect maternal mortality rates. Access to prenatal care, which one would think is an important factor, does not appear 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 high-order births (4th and above). Hence, changes in the number and timing of births may affect maternal mortality. These factors, however, seem to have a relatively small impact on maternal mortality. For example, 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).

Theory predicts changes in investments as a result of MMR declines only when there are returns on these investments. While no solid causal estimates of the returns to education exist for the cohorts we study, 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 in section 2, 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 benefits in the marriage market and for 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 subdivided 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 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) which are shown in Table 1b. Death rates exhibit the usual J-shape, with very 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 thereafter. The ratio of female to male deaths is the largest for ages 15 to 45 which is noteworthy since these are the childbearing years. The maternal mortality rate is reported at the same level of aggregation. The maternal mortality rate for 1925 to 1939 (which we use in some specifications to control for lagged MMR) is from De Silva (1943) who calculated them from vital statistics data for those years.[10]

For deaths broken down by cause, data are reported by district, year, and gender, but not age (although some diseases are denoted as affecting only children). Because deaths by cause were reported in fine 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 Sri Lanka adopted the new International Classification of Diseases. See Appendix Table 1 for details). In the analysis we aggregate up to 18 broad causes of death which 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. Maternal mortality is thought to have often been classified as pyrexia, since puerperal pyrexia is one of the

[10] We were unable to locate the data for 1940.

common causes of maternal death (De Silva 1943, Loudon 2000b, Deneux-Thauraux et al 2005). 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 and allow us to construct life tables for each district-year-gender during the period (World Bank 2003).[11] Although it would be preferable to have age-specific death rates for every 1-year age category, the data are not disaggregated beyond 5-year age groups, 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 may also affected by MMR reductions as our model predicts). We censor life expectancy at age 65 because the death rates in the early vital statistics reports 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 and because MMR is (nearly) inapplicable to those under 15. Table 1a shows that there was a very substantial improvement in life expectancy at age 15 from 1946 to 1963, about 8.2 years for women and 6.6 years for men. Life expectancy at 15 shows convergence across gender: the difference between men and women was 2.2 years in 1946, but about 0.6 of a year in 1963. Published life expectancy

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

measures by gender for the entire country show very similar patterns (Nadarajah 1983, United Nations 1976).

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

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.

The main limitation of the data is that we do not have any further variation within districts and gender. In addition, there are some outcomes that we would like to examine for which we do not have data. Completed years of education is not available because it was not recorded in the 1946 to 1963 censuses. To measure how child health investments respond to life expectancy, it also would be valuable to have measures such as height or vaccination rates, but we have been unable to obtain them. Lastly, we do not have any data on migration.

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 reductions had on female life expectancy. Figure 3a shows MMR over time by district from 1925 to 1964. As can be seen, MMR fell considerably over the time period, particularly between 1946 and 1950, 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 and discard the 1946 data to avoid using this variation. For consistency we also use the average of 1952 and 1954 for 1953 and the average of 1962 and 1964 for 1963.

An important question is what drove the MMR reductions. As described above, there were several policies such as expanded hospital births, ambulances, and maternal health programs that contributed. The spatial variation in MMR declines is characterized by the places with initially higher levels of MMR experiencing 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; 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. 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 versus MMR. As maternal mortality declined, life expectancy of women relative to men rose (although there are a few exceptions for which MMR and life expectancy 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—i.e., after childbearing years—against MMR we find that MMR declines are either not associated with or are *positively* associated with gender differentials in life expectancy at age 45. As predicted by the model, 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 is suggestive that maternal mortality declines were 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 consistent with excess female mortality being mostly due 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. Excess female mortality was highest at the ages

when the birth rate was highest as well. This is what we would expect if maternal mortality was responsible for excess female morality; maternal mortality is only a risk for pregnant women, and the higher the birth rate the higher the associated risk. Unfortunately data on births by age of mother are only available for 1952 onwards.

To quantify the effect of maternal mortality on life expectancy we estimate equation (3.1). We regress life expectancy on MMR*female and a full set of double interactions of gender, district, and year dummies. 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. Female 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 overall childbearing age range. We find that lowering female mortality rates for age 15-45 by 20% (reducing maternal mortality by 80%, as occurred over the period) results in an increase in life expectancy by 1.6 years. This estimate is nearly identical to our regression results.

The next rows show what the effect of maternal mortality is on life expectancy between ages 15 to 45 and between ages 45 to 65. As expected there is a large and significant negative effect on the 15 to 45 measure, the primary childbearing years. There is a positive and insignificant effect on the 45 to 65 measure. Note that a positive coefficient is consistent with competing risks if the marginal survivors of childbirth then die between age 45 and 65 from other causes.

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 address potential omitted variable bias. However, they are not our preferred specification for two reasons. First, the control variables 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 that we could be over-controlling by including these diseases because malaria and nutrition deficiencies could increase 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 1943). More recent work on maternal mortality is much more skeptical about the relationship between nutrition and maternal deaths, however (Loudon 2000a, Maine 2000). Loudon (2000a, pg. 241S) in particular strongly suggests 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 the results with these controls as useful checks. As seen in columns 2 to 4 of Table 2, the controls 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 (but mostly insignificant), suggesting that when MMR falls, if anything, death rates at post-childbearing ages increase. In proportional terms, the deaths particularly increase for relatively young ages (55 to 64 in column 1 or 45 to 54 in column 3). This is consistent with the theoretical prediction given in (2.6) that the marginal survivors will be relatively less healthy because their childbearing decisions, which offset the life expectancy impact of MMR declines, are least responsive to MMR.

In Table 4, as a way of assessing whether there are omitted variables, we look at whether MMR*female predicts differential declines in death rates for other causes of death. Cause of death data are 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. 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 exactly offset each other, which explains why in the previous table, there appears to be no effect of MMR on gender gaps in deaths rates for ages 0 to 4. These results are consistent with the fact that health services were concentrated on women and children. Among children there was no gender-differentiated effect on overall 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 prevalence, 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 are significantly correlated with MMR*female: pyrexia and tuberculosis. Pyrexia is most likely correlated with maternal mortality because maternal mortality often entails fever, so it is commonly miscategorized on death certificates as pyrexia. This is true both in Sri Lanka at this time (De Silva 1943) and in most countries today (Loudon 2000b, Deneux-Thauraux et al 2005). Death rates from pyrexia are indeed higher among women than men in 1946, and the difference is smaller in 1963. As additional suggestive evidence, Figure 7 plots the ratio of female to male pyrexia 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.

The evidence presented in this section suggests that declines in maternal mortality resulted in an increase in female life expectancy at age 15 of 1.7 years and were responsible for essentially all of 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 several outcomes. We start by looking at the birth rate (Table 5a). Since this outcome is applicable only to females, the estimates are a difference-in-difference, without the third difference of gender. 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 a concern. Therefore, 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 quite small: a 10% decline in MMR results in less than 1% increase in the birth rate. That said, according to the point estimate (column 6), although the decline in MMR from 1945 to 1963 only led to

a 3% increase in the birth rate, it explains 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 (and perhaps different) women are alive, and the incentives to give birth change. We therefore also report regressions that isolate the effects on the numerator by using log of births as the dependent variable. 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.[12]

Overall the results suggest 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 hence should be interpreted with caution. The last row of Table 5a confirms the cautiousness one must have in interpreting the results in the rows above. It shows the difference-in-difference estimates of the effect 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 is what 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 outcomes that are available by gender and thus permit a DDD strategy. We start by looking at the mean age at marriage. As discussed in section 2, it is not theoretically clear whether there will be 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 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

[12] We also estimated models using the crude birth rate (births divided by population) and estimates of the birth rate using data from Langford (1981). 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. The estimates for age at marriage and percent illiterate at marriage are imprecise probably partly because reporting of marriages is incomplete so these variables (from the vital statistics) are less reliable (compared to births and deaths in the vital statistics or outcomes from the censuses).

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 age 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 outcome is better measured in that it is available by age. While it does not tell us directly about enrollment or total years of schooling, literacy was typically obtained through schooling. A nice aspect of having literacy rates for a range of cohorts is that it enables us to run "placebo tests" to test the validity of our identification strategy. If a decline in MMR affects current and future investment, then one expects to find effects of MMR*female on literacy mostly for those ages 30 and below. These ages should be affected because their education occurs after the life-expectancy gains have occurred. For example, someone who is observed as a 20-year-old in the 1953 census would have been 13 years old in 1946 when MMR began declining. Note that in stating that one expects effects for individuals who are roughly under 30 in the census, the 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). For cohorts above these ages, literacy is most likely determined prior to the timing of Sri

Lanka's maternal health improvements. A significant effect of MMR*female on their literacy would suggest that unobserved factors are influencing the estimates. Such a pattern also might be driven by reverse causality, with higher female literacy causing lower maternal mortality rates.

It is worth noting that using older cohorts as a placebo group would be invalid if people become literate at later ages, for example through adult education programs or through "upward" spillovers from their children. (Completed schooling would be less subject to these problems, but it is not available.) Another concern in assuming that there will be no effect of MMR on older cohorts is that there might have been changes in MMR when *they* were young which affected their educational investments. Figure 3a suggests that for a few districts there was a continuous downward trend in MMR since 1925 (although for the districts with the largest declines there is a clear drop in levels in the post-WW2 years). We can address this potential omitted variable by controlling for lagged MMR. The appropriate lag will depend on the age group (with, for example, a longer lag for 55 year olds than 45 year olds). We present results using a 10-year-lag, but the results are similar using a longer lag.[13]

We estimate the age-specific effects of MMR in two different ways. First, to improve the precision of the estimates, we create one observation per gender-district-year-age, stack the data, and estimate a single regression where 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 estimating equation is

$$(6.1)\quad \begin{aligned} Y_{dtga} = \; & \beta_0 + \sum_a \beta_{1a} MMR_{dt} * female * (age = a) + \\ & \gamma_{dt}(district * year) + \delta_{dg}(district * female) + \nu_{tg}(year * female) \\ & \lambda_{ad}(age * district) + \theta_{ag}(age * female) + \psi_{at}(age * year) + \varepsilon_{dtg} \end{aligned}$$

The coefficients β_{1a} give the age-specific effect of MMR on female-male literacy gaps. We use the 5-year age groups from age 5 to age 59, so there are 11 observations per gender-

[13] .Ideally one would match individuals to MMR at the time when the investment decisions are being made. Since we do not know precisely at which age this occurs, we use the same lag for each age group when we estimate (6.1). Also, MMR by district is unavailable before 1925 which limits how long of lags we can use.

district-year in the regression.[14] Second, we follow our strategy so far and estimate (3.1) as a separate regression for each age group.

The first three columns of Table 6 presents the results with the age-specific coefficients estimated jointly (each column reports coefficients from a single regression). We find that a reduction in maternal mortality causes literacy increases for ages 15 through 44 with the magnitude being largest for the 25 to 29 year-olds. Theoretically, one might also expect an effect for the lowest ages, but the absence of such a finding may be explained by the fact that in 1946 the gender gap in literacy was small for very young ages (3 percentage points for ages 5-9 but 16 percentage points for ages 10-14, and larger for older ages).

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. The results suggest that there may be omitted variables. A likely candidate is that, through the very same mechanism, MMR when these older cohorts were young might be affecting their literacy. Therefore, we re-estimate the regression controlling for lagged MMR*female*age dummies. (MMR is lagged by 10 years, but the results are similar with longer lags as shown in Appendix Table 5, and as expected, the longer the lag, the older the cohort it has an effect on). As seen in column 2, pre-trends in MMR were driving the coefficients for older cohorts. We now find effects of MMR*female for ages 15 to 29 and insignificant and mostly *positive* coefficients for older ages. This pattern of coefficients is consistent with the predictions: the large changes in MMR occurred around 1946, so those age 15 to 29 in 1953 would have been 8 to 22, ages when females are obtaining literacy. Column 3 adds as additional controls the age- and gender-specific mortality rates for nutritional diseases and malaria. The results are almost identical with slightly smaller standard errors.[15]

Columns 4 to 6 of Table 6 present the results with separate regressions by age (each coefficient is from a separate regression). The results are similar but less precise. For

[14] Note that we exclude ages 60 and above since literacy rates appear to be affected by selective mortality (for example, when we follow a cohort across 2 censuses, the cohort's literacy rate often falls over time.) The results are similar if we include these older ages.

[15] Note that pre-trends are most relevant when examining older cohorts whose outcomes were determined in the past, and they are less relevant for contemporaneous outcomes such as the birth rate. That said, all of the other results we have shown are essentially unchanged if we include lagged MMR as a control, as shown in column 3 of Appendix Table 4.

example, in column 6, we find significant effects of MMR*female on those ages 15 to 24 and the coefficient for the 25-29 year olds is insignificant.

The declines in MMR can explain about 13% of the change in female literacy during the period for women. (For this calculation and those below, we average the coefficient across the three age groups comprising ages 15-29 and the six specifications in Table 6, which gives an effect of -0.0275.) A second way to interpret the coefficients is to calculate the 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. Therefore, a 1.3 unit decrease in MMR (1.3 fewer death per 100 births), which is the change that occurred between 1946 and 1963, increases adult life for women by 4.5%. The MMR*female average coefficient of -.0275 is relative to a base literacy rate of about .50. Thus, a 1.3 unit decrease in MMR increases literacy rates by 7.2%. Combining these two calculations, the elasticity of literacy with respect to adult life years is about 1.6. These results for literacy suggest that human capital investments are quite responsive to life expectancy.

One can also do a back-of-the-envelope calculation to translate the literacy estimates into years of schooling. The MMR drop of 1.3 deaths per 100 births implies a 3.6 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 3.6 percentage point increase in literacy corresponded to that proportion of the population gaining 6.2 years of schooling (7.8 minus 1.2), this yields an average increase in (relative) female education of about 0.22 years from a base of 4.8 years, or 4.6%. This corresponds to an elasticity of 1.0.[16]

[16] Since the life-expectancy gain was 1.7 years, there were 0.13 years of school per additional life-year. As a comparison, the calibrated model by Gan and Gong (2004) in a very different context (black-white gaps in the US) finds that a 5.9 year gap in life expectancy leads to 0.5 years more of school (0.08 years of school per additional life-year). Note that the black-white mortality differences occur at much later ages than maternal mortality, so accounting for time discounting would make the two estimates close to one another.

VII. Conclusion

Over the past fifty years, longevity has improved dramatically, particularly in developing countries. Over 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 hold promise of spurring 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. Maternal mortality occurs after human capital decisions yet while a woman still potentially has many prime-age years to live. Moreover, the fact that maternal mortality only affects women enables one to use a comparison to men to aid with causal identification.

We examined an episode of rapid MMR decline in Sri Lanka that began in 1946, a decline that, importantly, affected some regions in Sri Lanka much more than others. Over a twenty-year period, MMR declined by 80% (from 1650 to 310 deaths per 100,000 births), and this caused female adult life expectancy to increase by 1.7 years, eliminating a previous deficit between female and male life expectancy. We use this decline to estimate the corresponding change in human capital, as measured by literacy. The MMR decline caused female literacy to increase by 3.6 percentage points (relative to changes in male literacy), which represents a 7.2% increase. This change resulted from a 4.5% increase in female prime-age life-years. Thus, human capital investment appears to be highly responsive to lifespan. We also find some suggestive evidence that MMR declines caused fertility to increase, which is as predicted both because the risk of childbearing decreased 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 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. However, 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 eliminated 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, because of HIV/AIDS and civil war, have an important additional deleterious effect of dampening the incentive to invest.

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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) are 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.

We construct the other variables in a consistent manner. For example, for lagged maternal mortality rates, the 10-year-lag for 1946 (nominally 1936) is the average of 1935 and 1937 rates.

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.

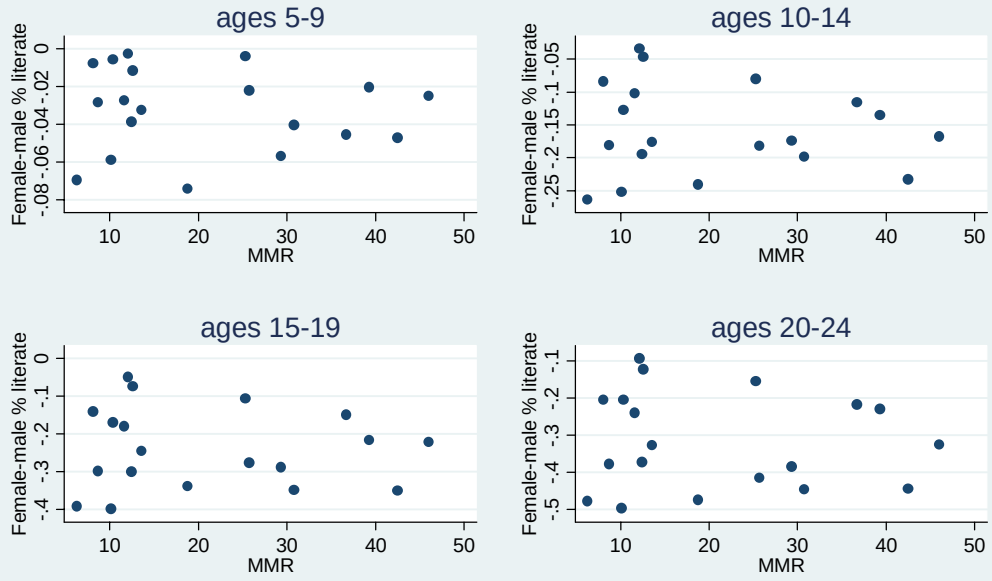
Life expectancy calculation

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

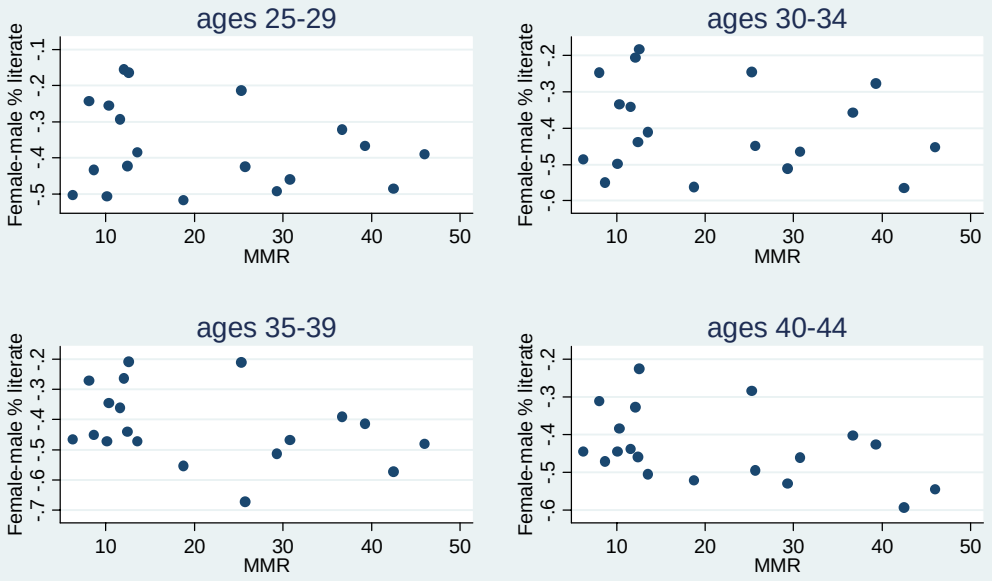
This variable was not reported in the 1963 census tabulations. What was reported instead was the percentage of individuals ages 5 and above who were in school. Breakdowns by age were not available by district, but are available for the entire country. These suggest that only 0.75% of students were above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

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



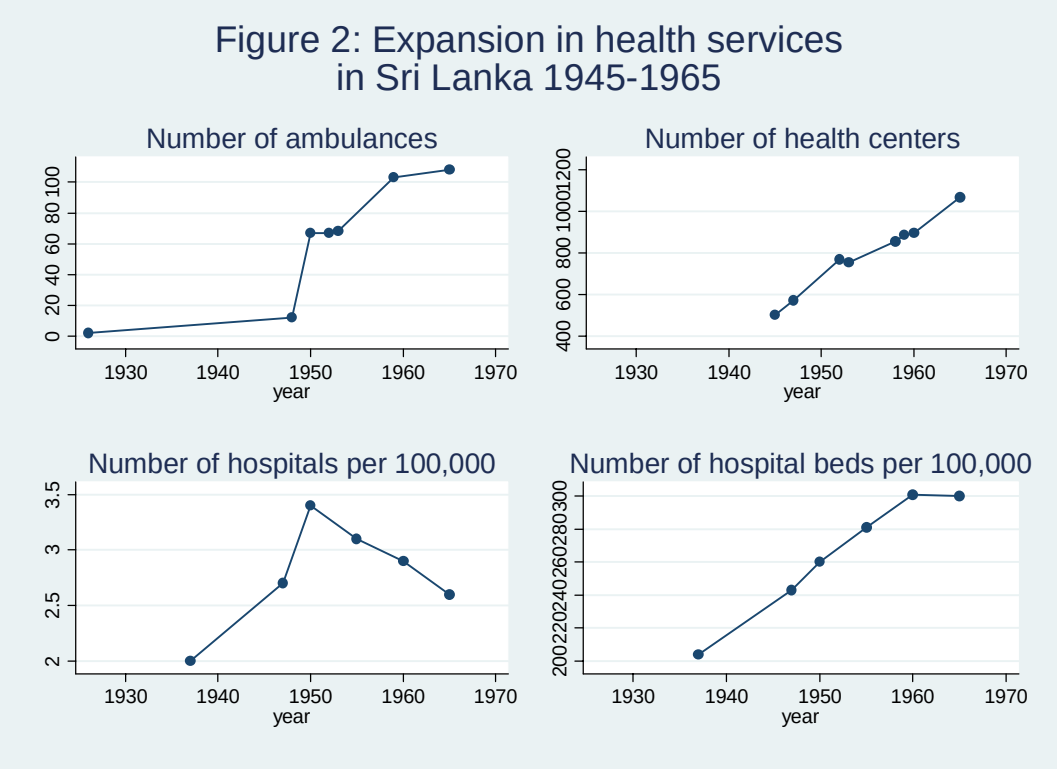
each dot represents a district

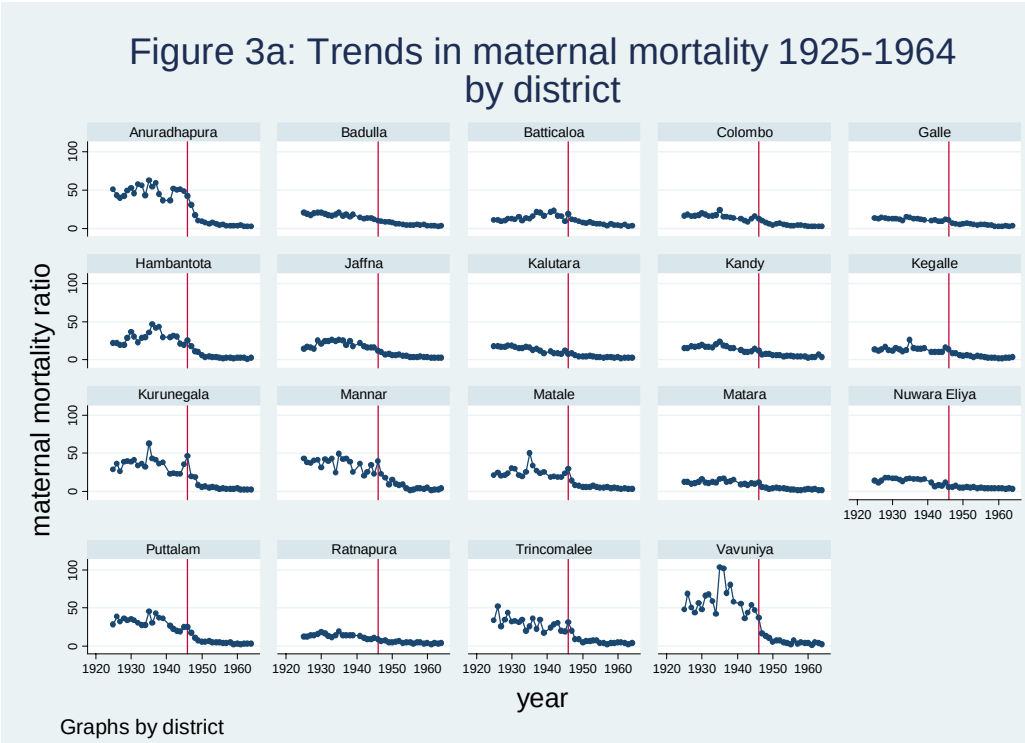
Figure 1b: Correlation between maternal mortality and literacy gender gaps in 1946



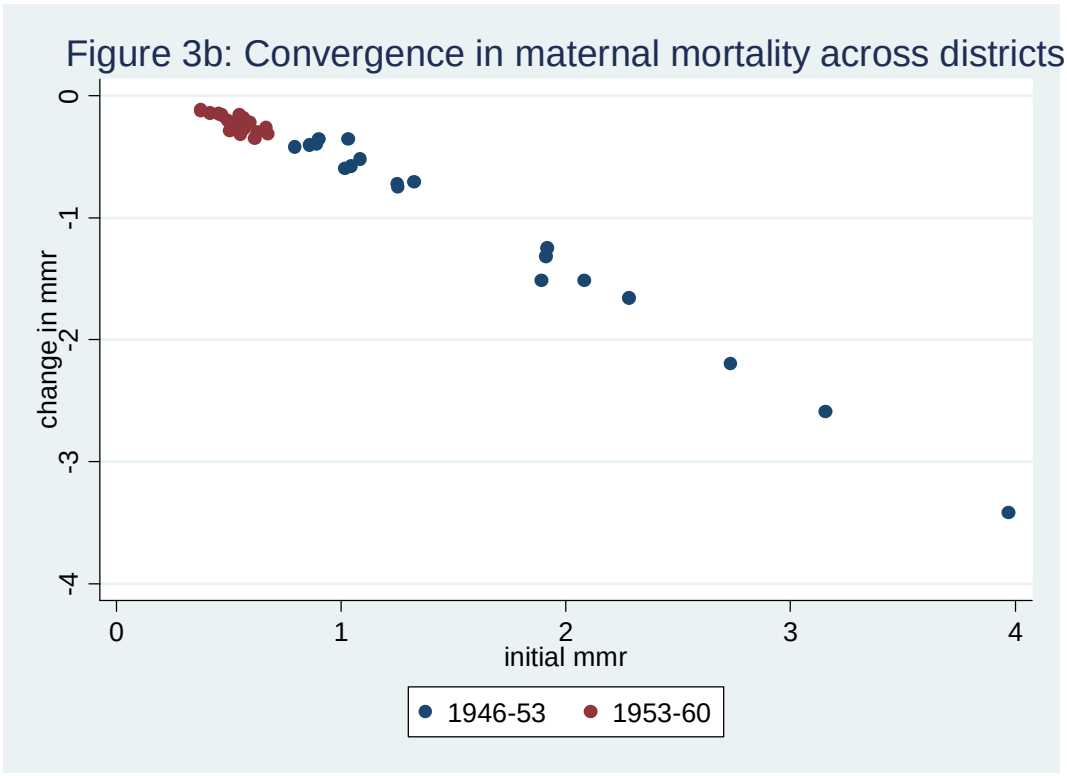
each dot represents a district

Note: Maternal mortality is reported here as the number of deaths per 1000 live births.





Note: Maternal mortality is reported here as the number of deaths per 1000 live births.



Note: Maternal mortality is reported here as the number of deaths per 100 live births.

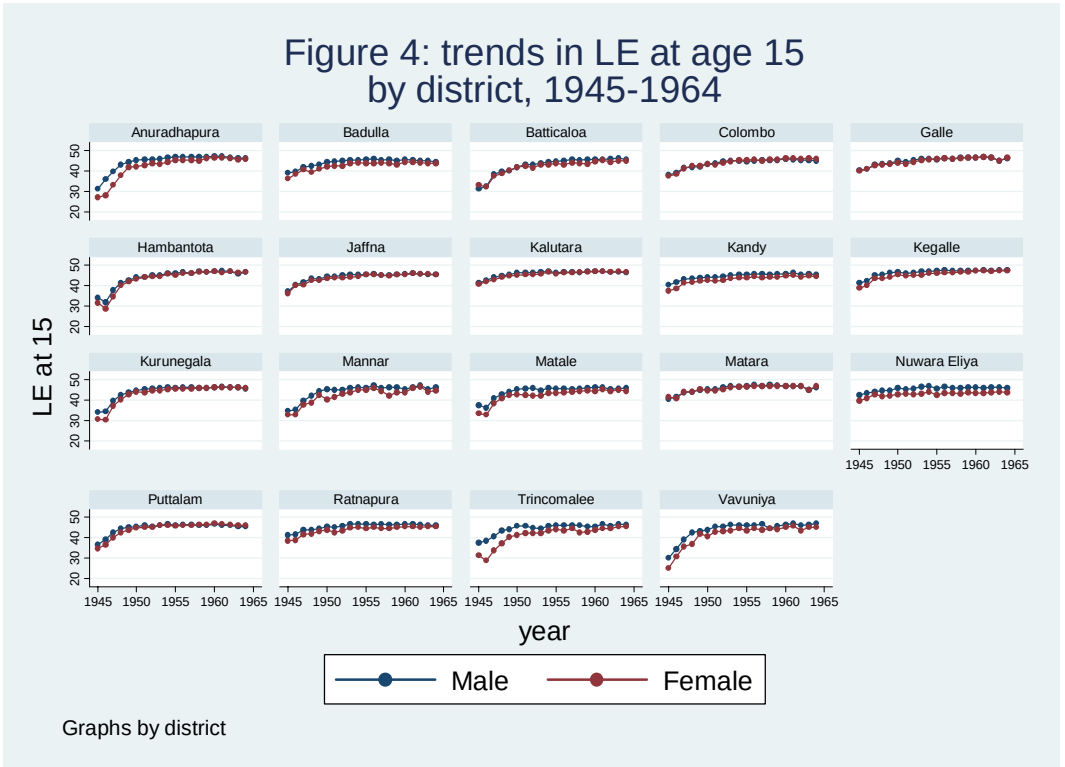


Figure 5a: trends in female-male LE at 15 and MMR by district, 1945-1964

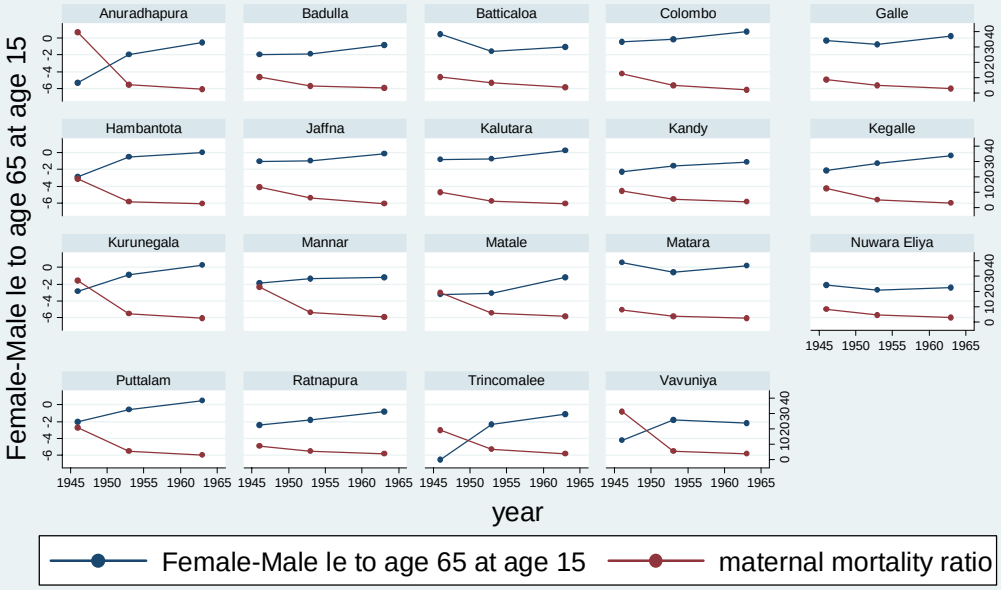
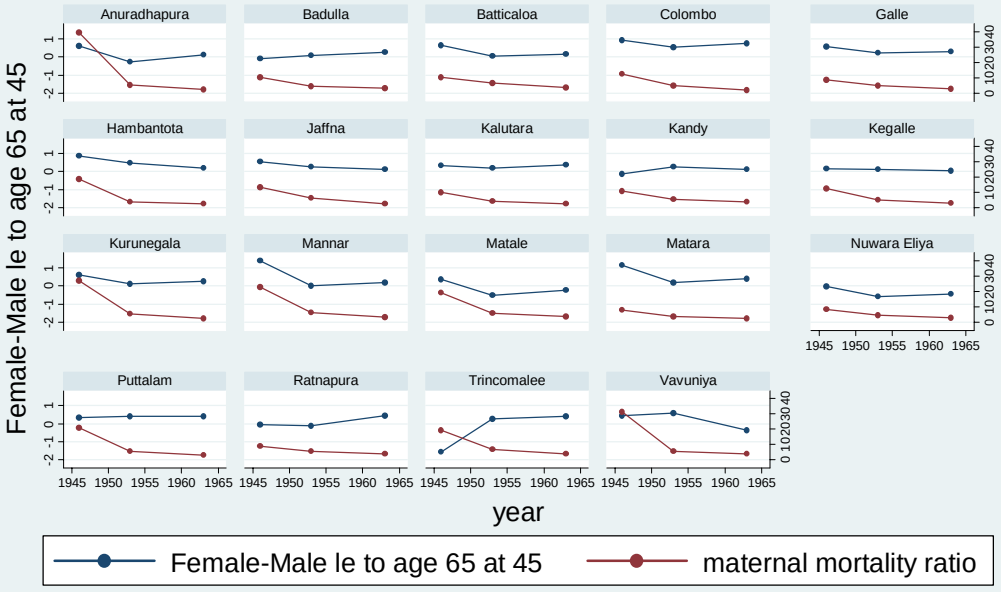
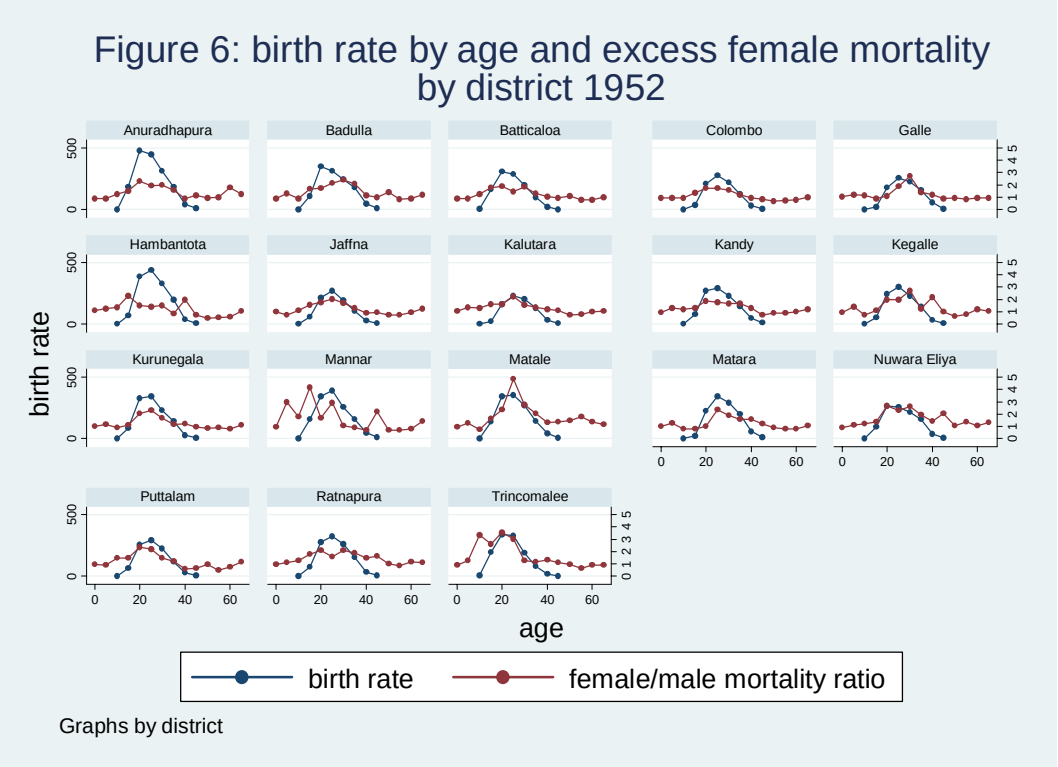


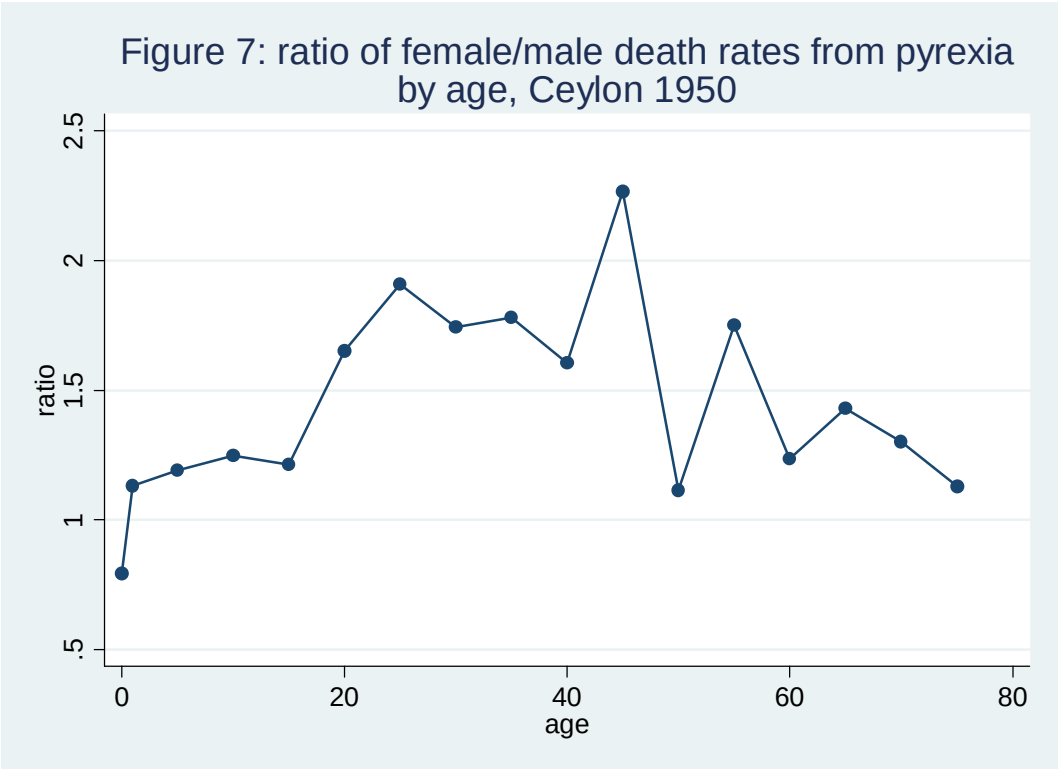
Figure 5b: trends in female-male LE at 45 and MMR by district, 1945-1964



Note: Maternal mortality is reported here as the number of deaths per 1000 live births.



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 difficult to observe.



Note: 1950 is the first year for which tabulations by cause and age are available from the Reports of the Registrar. The breakdowns by cause and age are reported only for the country as a whole.

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	27.7	27.9	29.2	21.2	21.6	23.2
% 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

*#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 death 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 population 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.
In 1946, we only have data by 10 year groups for ages 25-34, 45-54 and 55-64.

Table 2: Effect of maternal mortality on life expectancy

	(1)	(2)	(3)	(4)
Dependent variable:	year*district, female*district and female*year fe	Add malaria death rates	Add nutrition diseases death rates	Add nutritional diseases and malaria death rates
LE 15-65				
MMR*female	-1.288***	-1.289***	-1.355***	-1.359***
	[0.181]	[0.203]	[0.178]	[0.205]
R-squared	1	1	1	1
LE 15-45				
MMR*female	-0.957***	-0.958***	-0.931***	-0.921***
	[0.061]	[0.067]	[0.062]	[0.070]
R-squared	1	1	1	1
LE 45-65				
MMR*female	0.139	0.117	0.150*	0.128
	[0.093]	[0.098]	[0.078]	[0.095]
R-squared	0.99	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. 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

	(1)	(2)	(3)
Dependent variable: age specific death rate	year*district, female*district and female*year fe	Add malaria death rates	Add nutritional diseases and malaria death rates
Age 0-4			
MMR*female	1.536	1.62	0.070
	[1.350]	[1.524]	[0.598]
Age 5-9			
MMR*female	0.259	0.204	0.318***
	[0.238]	[0.253]	[0.107]
Age 10-14			
MMR*female	0.173	0.065	0.025
	[0.198]	[0.192]	[0.268]
Age 15-19			
MMR*female	3.156***	3.305***	2.450***
	[0.586]	[0.611]	[0.231]
Age 20-24			
MMR*female	5.483***	5.506***	4.712***
	[0.596]	[0.661]	[0.264]
Age 25-29			
MMR*female	1.953***	1.903***	2.572***
	[0.499]	[0.499]	[0.321]
Age 30-34			
MMR*female	2.276***	2.304***	2.700***
	[0.340]	[0.378]	[0.379]
Age 35-39			
MMR*female	-0.853	-1.025	0.060
	[0.958]	[1.071]	[0.495]
Age 40-44			
MMR*female	-0.793	-0.892	0.178
	[0.872]	[0.982]	[0.490]
Age 45-49			
MMR*female	-0.386	-0.280	-1.156**
	[0.798]	[0.847]	[0.539]
Age 50-54			
MMR*female	-0.201	-0.073	-1.186**
	[0.967]	[1.058]	[0.537]
Age 55-59			
MMR*female	-4.488**	-4.075*	-0.828
	[2.027]	[2.268]	[1.288]
Age 60-64			
MMR*female	-5.478**	-5.069*	-1.141
	[2.689]	[2.961]	[1.905]
Age 65+			
MMR*female	-1.679	-1.433	-4.041*
	[3.661]	[4.180]	[2.273]

Nutrition diseases are helminths, anemia and vitamin deficiencies. Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. 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

Panel A: Diseases affecting children under 5 only		**Panel B: Diseases for adults**	
Rathe		**Pyrexia (fever)**	
MMR*female	-0.354**	MMR*female	0.327***
	[0.140]		[0.106]
Diseases of the nervous system (convulsions)		**Pneumonia**	
MMR*female	0.134**	MMR*female	0.249
	[0.053]		[0.156]
Congenital debilities		**Vitamin**	
MMR*female	0.125***	MMR*female	0.305
	[0.036]		[0.285]
Premature birth		**Malaria**	
MMR*female	0	MMR*female	0.058
	[0.000]		[0.054]
		Diarrhea	
		MMR*female	0.022
			[0.039]
		Helminths	
		MMR*female	0.009
			[0.024]
		Diseases of the circulatory system	
		MMR*female	0.004
			[0.028]
		Tuberculosis	
		MMR*female	-0.056***
			[0.016]
		Anemia	
		MMR*female	0.035
			[0.026]
		Rheumatic fever	
		MMR*female	-0.019
			[0.015]
		Influenza	
		MMR*female	0.004
			[0.018]
		Dysentery	
		MMR*female	-0.021
			[0.016]
		Bronchitis	
		MMR*female	-0.023*
			[0.012]

Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. 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

	(1)	(2)	(3)	(4)	(5)	(6)
	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	-4.270
	[7.974]	[8.845]	[8.661]	[8.547]	[8.805]	[8.340]
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]
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]		
R-squared	0.94	0.96	0.96	0.97		

Nutrition diseases are helminths, anemia and vitamin deficiencies. 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

	(1)	(2)	(3)	(4)
	year*district, female*district and female*year fe	Add malaria death rates	Add nutrition diseases death rates	Add nutritional diseases and malaria death rates
Mean age at marriage				
MMR*female	0.046	0.001	0.066	0
	[0.099]	[0.086]	[0.107]	[0.118]
R-squared	1	1	1	1
Percent illiterate at marriage				
	1.927	1.387	2.495**	1.688
	[1.187]	[1.220]	[1.051]	[1.288]
R-squared	0.99	0.99	0.99	0.99
Percent in school (ages 5-24)				
	-0.008	-0.005	-0.010	-0.003
	[0.007]	[0.008]	[0.008]	[0.009]
Observations	114	114	114	114

Nutrition diseases are helminths, anemia and vitamin deficiencies. Robust standard errors reported in brackets. Each cell reports the coefficient from a separate regression. 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 by age

	(1)	(2)	(3)	(4)	(5)	(6)
	Joint regression for all ages *(each column is from a separate regression)*			Separate regressions by age *(each coeff is from a separate regression)*		
	All 2-way interactions of year, district, gender, & age fixed effects (female*year, female*dist, year*dist, age*dist, age*year & age*female fe)	Control for pre-trends (add 10-year lag of MMR*female *age dummies)	Add nutritional diseases and malaria	year*district, female*dist. & female*year fe	Control for pre-trends (add 10-year lag of MMR*female)	Add nutritional diseases and malaria
Ages 5-9						
MMR*female	0.017	-0.004	-0.005	-0.007*	-0.003	-0.003
	[0.010]	[0.017]	[0.016]	[0.004]	[0.006]	[0.005]
Ages 10-14						
MMR*female	-0.009	-0.023*	-0.024*	-0.008	-0.01	-0.009
	[0.010]	[0.014]	[0.014]	[0.015]	[0.017]	[0.017]
Ages 15-19						
MMR*female	-0.018*	-0.035***	-0.036***	-0.020	-0.028*	-0.028*
	[0.009]	[0.009]	[0.009]	[0.012]	[0.015]	[0.016]
Ages 20-24						
MMR*female	-0.022**	-0.027***	-0.029***	-0.018	-0.035***	-0.037***
	[0.008]	[0.010]	[0.010]	[0.013]	[0.012]	[0.010]
Ages 25-29						
MMR*female	-0.029***	-0.032***	-0.033***	-0.026***	-0.022	-0.020
	[0.006]	[0.010]	[0.010]	[0.009]	[0.014]	[0.014]
Ages 30-34						
MMR*female	-0.022***	-0.014	-0.015	-0.017	-0.015	-0.016
	[0.008]	[0.011]	[0.010]	[0.011]	[0.015]	[0.015]
Ages 35-39						
MMR*female	-0.021**	-0.004	-0.005	-0.032**	-0.020	-0.014
	[0.008]	[0.012]	[0.011]	[0.015]	[0.017]	[0.010]
Ages 40-44						
MMR*female	-0.012*	0.017	0.016	-0.012	0.012	0.009
	[0.007]	[0.012]	[0.011]	[0.015]	[0.016]	[0.013]
Ages 45-49						
MMR*female	-0.011	0.006	0.005	-0.009	-0.003	-0.002
	[0.008]	[0.012]	[0.011]	[0.014]	[0.013]	[0.009]
Ages 50-54						
MMR*female	-0.008	0.022**	0.021**	-0.008	0.012	0.006
	[0.011]	[0.009]	[0.009]	[0.016]	[0.015]	[0.011]
Ages 55-59						
MMR*female	-0.015	0.002	0.001	0.009	0.021	0.011
	[0.011]	[0.014]	[0.013]	[0.018]	[0.024]	[0.018]
Observations	1254	1254	1254	114 per reg	114 per reg	114 per reg
R-squared	0.98	0.98	0.98	varies	varies	varies

Nutrition diseases are helminths, anemia and vitamin deficiencies. Robust standard errors reported in brackets, clustered by district-year-gender in columns 1-3. In columns 1-3, the variable corresponding to a reported coefficient is a dummy for the specified age interacted with MMR*female. * 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	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)		
Pregnancy	XI	Diseases of pregnancy, childbirth and the puerperium	115	Sepsis of pregnancy, Childbirth and the puerperium (640, 641, 681, 682, 684)
				(a) puerperal sepsis
				(b) others
			116	Toxaemias of pregnancy and the puerperium (642, 652, 685, 686)
				(a) puerperal eclampsia
				(b) Others
			117	Haemorrage of pregnancy and childbirth (643, 644, 670-672)
			118	Abortion without mention of sepsis or toxaemia (650)
			119	Abortion with sepsis (651)
			120	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	126	Other disease of skin and musculoskeletal system (700-716, 731-736, 738-744)
		e-Rata		(a) Rathe erythematous conditions)
Pyrexia	200	Ill-defined and Unknown causes of death	137	Ill-defined and unknown causes (780-793, 795)
		c-Pyrexia		(b) Pyrexia

Appendix Table 1 Notes: This table reports the causes of death that we coded from the Reports of the Registrar. These were reported in Table XXIV each year. In 1950 the new classification of diseases (ICD 6) was used for the first time 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 reported on a separate line (which is the number we report here). The 1950 table was unique but comparable to tables later on. Tables from 1951 to 1965 are identical. In these tables each disease is reported on a separate numbered line (as reported here) and the codes corresponding to the diseases are shown in parentheses. The first column reports the name of the disease as it is used in the paper.

Appendix Table 2: Convergence in MMR across districts

Dependent Variable: MMR change				
	(1)	(2)	(3)	(4)
	1946-1953	1953-1963	All	All
Panel A: no other covariates except year				
MMR level	-0.972***	-0.640***	-0.960***	-0.640***
	[0.017]	[0.082]	[0.017]	[0.082]
MMR*1946-53 period				-0.332***
				[0.084]
R-squared	0.99	0.58	0.99	0.99
Panel B: Control for initial level of other diseases				
MMR level	-1.098***	-0.374**	-1.041***	
	[0.067]	[0.164]	[0.075]	
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			
	(1)	(2)	(3)
Coefficient on 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

	(1) Standard errors clustered by district and gender	(2) population weights	(3) Control for lagged values of MMR (10 years)
LE 15-65	-1.288***	-1.354***	-1.598***
	[0.236]	[0.153]	[0.364]
LE 15-45	-0.957***	-0.931***	-0.993***
	[0.084]	[0.054]	[0.123]
LE 45-65	0.139	0.146*	0.065
	[0.104]	[0.079]	[0.144]
Mean age at marriage	0.046	0.164	-0.038
	[0.097]	[0.161]	[0.235]
%illiterate at marriage	1.927	2.468**	2.561
	[1.436]	[1.115]	[1.563]
Birth rate	-28.216**	-34.044***	-21.156**
	[11.204]	[8.781]	[9.354]
% in school (ages 5-24)	-0.008	-0.015	-0.009
	[0.010]	[0.010]	[0.008]
% literate (joint regression)			
Ages 5-9	0.031	0.017	
	[0.022]	[0.012]	
Ages 10-14	-0.004	-0.009	
	[0.016]	[0.013]	
Ages 15-19	-0.021*	-0.018	
	[0.012]	[0.011]	
Ages 20-24	-0.034***	-0.022***	
	[0.011]	[0.007]	
Ages 25-29	-0.034***	-0.029***	
	[0.009]	[0.005]	
Ages 30-34	-0.034***	-0.022***	
	[0.009]	[0.007]	
Ages 35-39	-0.026***	-0.021**	
	[0.009]	[0.008]	
Ages 40-44	-0.026**	-0.012*	
	[0.011]	[0.007]	
Ages 45-49	-0.024**	-0.011	
	[0.011]	[0.010]	
Ages 50-54	-0.017	-0.008	
	[0.013]	[0.013]	
Ages 55-59	-0.022	-0.015	
	[0.015]	[0.013]	

Appendix Table 5: Effect of MMR on literacy using different lags of MMR

	(1)	(2)	(3)	(4)	(5)	(6)
	Joint regression for all ages *(each column is from a separate regression)*			Separate regressions by age *(each coeff is from a single regression)*		
	Control for 10 year lag of MMR	Control for 15 year lag of MMR	Control for 20 year lag of MMR	Control for 10 year lag of MMR	Control for 15 year lag of MMR	Control for 20 year lag of MMR
Ages 5-9						
MMR*female	-0.004	0.006	0.015	-0.003	-0.008	-0.006
	[0.017]	[0.011]	[0.011]	[0.006]	[0.006]	[0.004]
Ages 10-14						
MMR*female	-0.023*	-0.009	-0.013	-0.01	-0.002	-0.008
	[0.014]	[0.014]	[0.011]	[0.017]	[0.017]	[0.015]
Ages 15-19						
MMR*female	-0.035***	-0.025**	-0.025***	-0.028*	-0.02	-0.018
	[0.009]	[0.009]	[0.009]	[0.015]	[0.013]	[0.012]
Ages 20-24						
MMR*female	-0.027***	-0.024***	-0.027***	-0.035***	-0.021	-0.015
	[0.010]	[0.008]	[0.008]	[0.012]	[0.013]	[0.011]
Ages 25-29						
MMR*female	-0.032***	-0.032***	-0.034***	-0.022	-0.027**	-0.025**
	[0.010]	[0.008]	[0.006]	[0.014]	[0.010]	[0.009]
Ages 30-34						
MMR*female	-0.014	-0.015*	-0.024***	-0.015	-0.016	-0.013
	[0.011]	[0.008]	[0.007]	[0.015]	[0.012]	[0.010]
Ages 35-39						
MMR*female	-0.004	-0.011	-0.021**	-0.02	-0.028	-0.027*
	[0.012]	[0.011]	[0.009]	[0.017]	[0.019]	[0.016]
Ages 40-44						
MMR*female	0.017	0.007	-0.006	0.012	0.003	-0.01
	[0.012]	[0.008]	[0.007]	[0.016]	[0.014]	[0.015]
Ages 45-49						
MMR*female	0.006	0.004	-0.005	-0.003	0	-0.008
	[0.012]	[0.009]	[0.008]	[0.013]	[0.015]	[0.014]
Ages 50-54						
MMR*female	0.022**	0.01	0.001	0.012	0.004	-0.011
	[0.009]	[0.010]	[0.010]	[0.015]	[0.016]	[0.016]
Ages 55-59						
MMR*female	0.002	0.002	-0.004	0.021	0.028	0
	[0.014]	[0.011]	[0.010]	[0.024]	[0.020]	[0.019]
Observations	1254	1254	1254	114 per reg	114 per reg	114 per reg
R-squared	0.98	0.98	0.98	varies	varies	varies

Robust standard errors reported in brackets, clustered by district-year-gender in columns 1-3. In columns 1-3, the variable corresponding to a reported coefficient is a dummy for the specified age interacted with MMR*female.
* significant at 10%; ** significant at 5%; *** significant at 1%