Does Increased Access Increase Equality? Gender and Child Health Investments in India

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Abstract

Policy makers often argue that increasing the level of development and access to health care are crucial to addressing gender inequality in the developing world. This paper analyzes the relationship between access to child health investments and gender inequality in those investments in India. The first part of the paper explores the proximate causes of the gender imbalance in mortality in India. I find that a large share of the gender imbalance (about 30%) can be explained by differential access to vaccination. The second part of the paper estimates the effect of changes in access to vaccination on gender inequality. I argue that the direction of these effects is not obvious. A simple model of (gender-biased) parental investments, and empirical work using variation in access to vaccination, both suggest that initial increases in vaccination availability from low levels will increase gender inequality; further increases will then decrease inequality. This non-monotonic pattern is also reflected in differences in mortality. This result may shed light on the contrast between the cross-sectional and time series evidence on gender and development and may provide guidance about policy.

1 Introduction

Gender inequality in mortality in Asia and the Middle East is an important policy issue. Fifteen years ago, Sen (1990, 1992) coined the phrase "missing women" to describe the gender imbalance in populations, arguing that discrimination had led to more than 100 million premature female deaths. The issue remains salient: the 2006 World Bank Development

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Report focused on equity, with gender equity as a central issue (World Bank, 2006). Simultaneously, decreases in the female-male sex ratio (number of women divided by number of men) over time in India, South Korea, China and elsewhere – whether due to sex-selective abortion or other changes – have caused concern about the trend and the consequences for societies (see, for example, Hudson and den Boer (2004)).

Policy makers have argued that the increasing the level of development is one of the key factors in ameliorating gender inequality. In 2001, a World Bank report on gender and development begins with the statement that poverty and gender inequality are closely linked: "Large gender disparities in basic human rights, in resources and economic opportunity ... are pervasive around the world... And these disparities are inextricably linked to poverty" (World Bank, 2001). One of the crucial aspects of development cited is access to health services (World Bank, 1991; Hill and Upchurch, 1995). It has been argued that increasing the level of health care will benefit women and reduce gender inequality (Grown, Gupta and Pande, 2005), although the link between development and inequality is not limited to health (see, for example, Dulfo, 2005). This argument is particularly salient in India, where poverty is often linked to gender ratios by region and, by extension, excess female mortality (World Bank, 1991; Chatterjee, 1990).

These conclusions are largely motivated by the cross-country relationship between gender inequality and development – gender inequality is highest in countries and regions that are poor. The poverty-inequality relationship is, however, is often not supported in the time series. Figure 1, for example, shows the female-male sex ratio (number of women in the population divided by number of men) in India over the twentieth century. Although India has experienced a dramatic increase in income over this period, the population has become increasingly male-dominated.[1] In this paper I attempt to resolve some of this puzzle, arguing that, both in theory and in fact, improvements in health inputs may have ambiguous effects on gender inequity.

I focus in this paper on the case of India, and I proceed in two parts. I first analyze the underlying proximate causes of the gender imbalance in India, and then I explore the

[1]This pattern is even more surprising when we consider that as life expectancy overall increases we would generally expect the sex ratio to *increase* since women tend to live longer.

relationship between gender inequality and access to health investments. Although the primary goal of the paper is a deeper understanding of the inequality-access relationship, knowing the proximate causes of the gender inequality is crucial for understanding which health investments are the most important to explore.

In Section 2 I use microdata on children (from the National Family and Health Surveys) to explore the proximate causes of the gender imbalance in mortality.[2] I focus on childhood, in particular on children under 5. The data indicate that this is the most crucial time period: the analysis here shows that girls experience significant excess mortality between the ages of 1 and 4, and the gender imbalance by age 5 is large enough to explain virtually all of the imbalance in the population.

I consider the role of gender differences in immunization, nutrition and medical treatment in explaining the excess mortality, using two primary empirical strategies. I first use a simple regression to estimate how much the gender imbalance in mortality is reduced by including controls for child health investments. Second, I combine estimates of the gender inequality in each investment with estimates of the effect of that investment on mortality. Together, these allow me to calculate the total contribution of that investment to the gender bias. The results indicate that vaccination is the most important factor, explaining about 30% of the excess female mortality. Nutritional differences and medical care play a role, but a smaller one. Together, all causes considered explain about half of the total imbalance in mortality.[3]

I then turn to the primary focus of the paper – the connection between access to health investments and gender inequality. Based on the results in Section 2, I concentrate on

[2]Elsewhere (Oster, 2005) I argue that a naturally occurring lower sex ratio at birth (more boys) resulting from hepatitis B can explain a fraction of the gender bias. However, in the case of India, this fraction is small – only around 20% – and most of the literature suggests that excess female mortality is the primary cause of the gender imbalance. In this paper, I will be able to abstract away from a biased sex ratio at birth by using information on deaths for children conditional on birth.

[3]The focus on childhood here is consistent with an existing literature on the determinants of excess female mortality; the gender difference in vaccinations, nutrition and medical treatment has also been noted in that literature, but there has been little attempt to quantify the importance of these factors (DasGupta, 1987; Basu, 1989; Griffiths et al, 2002; Borooah, 2004; Pande, 2003; Mishra et al, 2004). It is also worth noting here that this paper is concerned with the *proximate* causes of excess female mortality. With this focus I mean to distinguish this work from a large existing literature about the underlying determinants of gender imbalance in mortality – female education, kinship patterns, etc (see, for example, Rosenzweig and Shultz, 1982; Agnihotri, 2000; Agnihotri et al, 2002; Murthi et al, 1995; Rahman and Rao, 2004).

vaccinations.[4] Section 3 presents a simple model of gender-biased parental investment in which parents choose whether or not to vaccinate their children; vaccination is a costly activity with positive effects on survival. The model suggests that the effect of access on gender inequality is non-monotonic: in particular, starting from a situation with little access (equivalently, high costs) increases in access will increase inequality. Continued increases in access, however, will eventually reduce inequality. Intuitively, when vaccination is first made available (equivalently, made cheaper) the most valuable children – i.e. boys – will get access first. This means that there is some range of increasing access over which more boys are getting vaccinated and there is no change for girls, which makes the gender imbalance worse. Further improvements in access, however, will lead to a decrease in the gender imbalance as the society moves to a situation in which all children are vaccinated.[5]

I test this prediction in Section 4 using (primarily) data on the recent availability of "Health Camps" in villages; the availability of these camps serves to increase access to vaccination. Consistent with the theory, the data suggest that initial increases in the number of camps increase the gender imbalance in vaccination, but that further increases decrease the imbalance. This non-monotonic effect is stronger for families with a stronger reported gender bias and stronger in areas where the health camps provide a larger share of total vaccinations. The results are not obviously driven by non-random camp placement. As a robustness analysis, I also show a non-monotonic relationship between the gender imbalance in vaccination and distance to the nearest government health center, as well as a non-monotonic relationship at the regional level.

In the final section of the paper, I consider whether this non-monotonicity is reflected in changes in mortality over time. I first use retrospective information on child mortality from the microdata to construct a short panel of child mortality over the period from 1982-1993. Consistent with the theory, excess female mortality increases over this period in areas where initial vaccination levels are low. However, during the same period, excess female mortality

[4]The possibility that increases in vaccination availability might combat the gender imbalance has been discussed in both policy and academic work (Hill and Upchurch, 1995; World Bank, 2001).

[5]There are interesting parallels between this theoretical result and an older literature on wealth and intrahousehold inequality. Kanbur and Haddad (1994), for example, argue that an intrahousehold bargaining framework can predict this type of non-monotonic relationship between wealth and inequality within the household.

decreases in areas with initially high levels of vaccination. I then explore whether there is any evidence for this non-monotonicity in mortality differences over a longer period of time in India. Using data on life expectancy by gender in India for the last 100 years, I show that average life expectancy increases while women initially lose relative to men and then rebound. Although this evidence is only suggestive, it leaves open the possibility that non-monotonicities might appear in investments other than vaccination, and may have impacted overall changes in the sex ratio over time.

The results in this paper may be informative about policy. First, the basic results in Section 2 underscore the importance of universal vaccination. A policy that would ensure vaccination of all children would have a dramatic impact on overall mortality (a fact which is well known) *and* would significantly affect the gender bias. The results on the relationship between access and inequality, however, suggest that *how* vaccination is introduced may be meaningful. A program that puts two vaccination camps in every village may have a similar overall effect to one that puts four in half of the villages, but the impact on gender inequality is likely to be quite different. Ultimately, the choice of policy depends on whether we are concerned only about overall mortality or whether the gender imbalance is a direct input to the social utility function.

2 Proximate Causes of Excess Female Mortality

This section analyzes the proximate causes of the gender imbalance in mortality in India. I first briefly address the issue of when in life the gender imbalance arises, and argue that early childhood is the crucial period. I then analyze the role of a number of child health investments – vaccination, nutrition and medical care for illness – and argue that vaccination is the most important. I ultimately conclude that vaccination can explain roughly 30% of the gender bias in mortality. This conclusion will be important in focusing the remainder of the paper, which will then consider the effects of increasing access to vaccination in particular on gender inequity. Section 2.1 discusses the methodology used here. Section 2.2 discusses the data and Section 2.3 presents results.

2.1 Methodology for Estimating Proximate Causes

The goal of this section is to calculate the importance of any given health investment in explaining differences in mortality. To illustrate the basic concept, define D as the differences between genders in some investment (for example, the difference in the chance of measles vaccination). Define μ as the importance of this investment in mortality (for example, the difference in mortality probability if vaccinated and unvaccinated) and Ψ as the overall excess female mortality. The share of the overall difference that is explained by this investment is, therefore, simply

$$\frac{D\mu}{\Psi} \tag{1}$$

That is, the overall contribution is simply the expected excess mortality from this particular cause ($D\mu$) divided by the total excess mortality. The challenge, then, is estimating D, μ and Ψ.

Consider first the estimation of overall differences in mortality (Ψ from equation 1 above). This variable is, intuitively, the difference between actual and expected probability of death for girls. In other words, Ψ measures how much more likely a girl is to die relative to what we would expect based on mortality of boys. The most obvious way to estimate this would be to simply calculate the difference between male and female mortality in India and assume that Ψ is equal to that difference. The problem with this, however, is that there may well be difference between genders even in non-discriminatory environments.[6] To solve this problem, I employ a "difference in difference" technique, in which I use data on India and a comparison region to evaluate the difference in mortality in India relative to the "expected" difference.[7] The equation estimated is below:

$$\Pr(die) = (\alpha) + (\beta_0)(girl) + (\beta_1)(India) + (\beta_2)(girl \times India) + \mathbf{\Phi X} \tag{2}$$

The coefficient of interest is β_2, the interaction between being a girl and living in India. The coefficient on this interaction is the gender imbalance in mortality. If India is similar to the

[6] It is very frequently observed that men are more likely to die at all ages in non-discriminatory environments, but the reasons are not obvious. Wells (2000) provides good links to the literature on the existence of this effect, and presents one potential explanation.

[7] The comparison region used is Sub-Saharan Africa. This is discussed in more detail in the data section.

comparison region, then we should find $\beta_2 = 0$. If girls are disadvantaged, we should find $\beta_2 > 0$. In the language of equation 1, $\beta_2 = \Psi$.[8]

The second methodological issue is identification of $D\mu$. I focus on two primary analyses: individual regression (which will estimate the entire quantity $D\mu$) and direct calculation of D and μ. Consider first the individual regression. Imagine that I have an individual-level panel in which I observe, for children, their level of health investment and their mortality outcomes. I can then estimate the quantity $D\mu$ by comparing the coefficient on $girl \times India$ in two difference in difference regressions – the first without controls for the health investment, and the second with these controls. In particular, denoting the health investment as Z, I first estimate equation 2 above, and then equation 3 below.

$$\Pr(die) = \delta + \gamma_0 (girl) + \gamma_1 (India) + \gamma_2 (girl \times India) + \gamma_3 (Z) + \Phi X \tag{3}$$

Given these regressions, $D\mu = \beta_2 - \gamma_2$.

Perhaps the easiest way to see the intuition behind this calculation is to think of Z as an omitted variable in equation 2. β_2 captures the effect of many investments, one of which is Z. By not controlling directly for Z, β_2 is "upward biased". Controlling for Z will decrease β_2, with the amount depending on how important Z is in explaining the mortality imbalance. More concretely, model that the relationship between Z and the interaction by equation 4 below:

$$Z = \eta + \upsilon_1 (girl) + \upsilon_2 (India) + \upsilon_3 (girl \times India) \tag{4}$$

We then note, based on the omitted variable intuition, that $\beta_2 = \gamma_2 + (\gamma_3)(\upsilon_3)$. This means, however, that $\beta_2 - \gamma_2 = (\gamma_3)(\upsilon_3)$. From this, however, it is straightforward to see why this is an estimate of $D\mu$: γ_3 is just a measure of the effect of the health investment on mortality (μ) and υ_3 is a measure of the gender bias in that investment (D). The product of these two will give us the share explained by that particular investment. As noted, this analysis will require an individual-level panel dataset (or enough information to construct one). This will mean that, in practice, this methodology will only be possible for vaccination, where information in the NFHS is sufficient to create a panel on investment and mortality.

[8] It is important to note that, while I will continue to refer to this as a difference-in-difference regression, that does not connote anything about identification. What is done here is simply a mechanical adjustment for baseline differences between the genders.

A significant concern with this approach is that including controls for the elements of Z "over-controls", and soaks up some of the effect of parental preferences. If Z measures vaccination, but differences in vaccination are simply a proxy for preferences and are perfectly correlated with all other forms of discrimination, then the difference between β_2 and γ_2 will capture much more than just the effect of vaccination. This will only be an issue if vaccination overall is correlated with parental gender preferences *and* if mortality is correlated with gender preferences.[9] However, this does not seem to be the case. Regressing mortality and vaccination on the parental reported ideal sex ratio (parents are asked about their ideal number of sons and daughters in the later survey waves) yields insignificant and small coefficients (results available from the author). Given this, the concerns about "over-controlling" may be less valid. Nevertheless, in general we may still worry about omitted parental preferences. To adjust for this, one option is to include some simple preference controls – in particular, reported ideal sex ratio. When I do this, the results do not change. Of course, this control may not fully capture preferences and it remains a concern. One advantage of the second methodology discussed below is that these concerns will be largely avoided.

The second methodology used to calculate $D\mu$ is direct estimation of D and μ. In particular, I first use the NFHS to directly calculate the gender differences in treatment. This is done by estimating equation 3 above – the estimate of D is simply γ_2. I then obtain estimates of μ from the existing literature, based on studies where mortality outcomes are observed for children with varying levels of health investment. There are two advantages to this approach. First, because the estimates of the effect of treatment on mortality come from other surveys, there is less concern about bias based on this particular sample. Second, this technique will allow me to get estimates for the effect of nutrition and medical treatment, as well as for vaccination. As a final robustness check, I replicate the individual-level analysis using data at the regional level. Although this is likely to be the least appealing methodology, it does allow me to control for all of the elements of mortality simultaneously.

[9] To see this, consider the same setup as the footnote above. To say that we "over-control" implies that the adjustment between the two regressions is too large – in other words, $(\gamma_3)(\upsilon_3)$ is too big. This will be the case if $\hat{\gamma_3}$ – the relationship between vaccination and mortality – is over-estimated. Based on the standard omitted variable bias arguments, omitting parental preferences will be a problem if measles vaccination is correlated with preferences and mortality is correlated with preferences.

Before moving on to the data and results, it is worth briefly discussing how the methodology used here differs from that in the existing literature. There are basically two differences. First, most existing literature (for example, Das Gupta, 1987) uses only a difference approach – comparing the death rates of boys and girls in India. This will generally underestimate true excess female mortality because boys are more likely to die in non-discriminatory environments. Second, the existing literature on proximate causes generally focuses only on estimating the differences in treatment by gender (i.e. D from the discussion above) and not the effect of these treatments on mortality (Basu, 1989; Griffiths et al, 2002; Pande, 2003; Borooah, 2004; Mishra et al 2004). Without adjusting for differences in μ it is very difficult to say anything conclusive about which inputs are more important in explaining the gender differences.[10]

2.2 Data on Child Mortality

The analyses here will be run using individual-level microdata on child survival and health investments.[11] For India, the data used are from two waves of the National Family and Health Survey (NFHS) (1992-1993 and 1998-1999), which covers approximately 90,000 women in each wave. Women are asked about their birth history, including children ever born, dates of birth, if the children are alive and, if not, when they died. In addition, for children under 5, information is collected on vaccination, medical treatment and malnutrition.

As discussed in the methodology section, the size of the gender imbalance in mortality and investments in India is evaluated relative to the size of this imbalance in a comparison area. This will allow me to difference out any differences across genders (favoring boys or girls) that occur in apparently non-discriminatory environments. The literature on the "missing women" suggests two natural comparisons: Sub-Saharan African (Sen, 1990; Sen, 1992) and demographer's life tables (Coale, 1991; Klasen, 2002).[12] Gender differences in

[10] In the existing literature there is also frequently a lot of focus on the differences between North and South India. I will not focus on these separately below. However, if I do separate into the two regions I find that gender imbalances are higher in North India in virtually all of the inputs and in excess mortality, but the conclusions about patterns by age in childhood and about which proximate causes are most important will hold.

[11] This is in contrast to much of the literature on this topic, which relies on district-level data on sex ratios. The advantage of using the individual-level data is that we observe directly the relationship between health investments and mortality.

[12] Life tables are widely used by demographers. Among other things, they give death probabilities by age

mortality in Sub-Saharan Africa are similar to those predicated in the life tables, suggesting that either comparison will give similar results. The advantage of using Sub-Saharan Africa, as I do here, is that the same type of microdata on children is available from a number of countries. The Demographic and Health Surveys (DHS) in Africa mirror the NFHS, so the difference-in-difference analysis can be run at the level of the individual child. The comparison countries are Kenya, Namibia, Zambia, Tanzania and Zimbabwe.

The three child investments analyzed are vaccination, malnutrition and treatment for disease. There are seven possible vaccinations (three DPT vaccines, two polio vaccines, a measles vaccine and a BCG vaccine). In general, I will use two measures of vaccination: the total number of vaccinations reported by the mother and the total number marked on the child's health card. The results are extremely similar if I control for dummies for each vaccination.

Information on malnutrition is based on actual height and weight measurements. Living children under 4 in each household are measured and weighed and their percentile weight-for-age is reported (weight-for-height and height-for-age are also reported, and all are very closely linked). I define children as severely malnourished if their percentile weight-for-age is less than 60% of the reference median for their age and gender. I use this indicator rather than a continuous measure since research on the effect of malnutrition on mortality indicates that mortality is largely unaffected by malnutrition above 60% of the reference median, but increases sharply after that (Chen et al, 1980).

To evaluate differences in medical treatment, parents were asked whether each of their (living) children under 4 had diarrhea or symptoms of a respiratory infection in the last two weeks. If the answer is yes, they are asked about what treatment was provided. I report children as having been treated if their parents report having given the child any treatment (including doctor visit, home remedies, etc). In these data, differentiating by treatment type has little effect on the gender difference.

for populations at different levels of development. The data are based on historical data from different areas of the world, extrapolated using regressions. They can be used to predict, for example, the age structure of a population given the fertility rate and overall life expectancy. Details on life table construction and use can be found in Coale, Demeny and Vaughan (1983).

2.3 Results on Proximate Causes of Mortality

Baseline Mortality Differences

The first set of results here estimate the baseline excess female mortality. The primary analysis below will focus on proximate causes of mortality for children ages 18 months to 4 years (this is the group for which we have good data on health investments). For this analysis I will use an estimate of excess female mortality based on that age group. However, to get a better sense of the patterns in mortality over childhood, I first estimate equation 2 for smaller age groups ranging from birth to ten years. The dependent variable is a series of indicators for having died within a particular age group. For example, the first variable is a 0-1 dummy for whether a child born in the last ten years died before the age of 6 months; the second variable is a 0-1 dummy for whether the child died between 6 months and 1 year, *conditional on* having lived to six months. The additional age groups are 1-2 years, 2-4 years, 4-6 years, 6-8 year and 8-10 years.

The results of this analysis can be seen graphically in Figure 2, which plots actual and expected mortality for India by age group. By the age of 10, the actual probability of female deaths is almost 12%, compared with an expected probability of slightly less than 10%. Nearly all of this imbalance seems to arise between the ages of 1 and 4, when expected mortality is around 1.4% and actual mortality is a full 2.4%. The regression analog to this figure appears in Table 1, where the difference-in-difference estimate is the coefficient on $girl \times India$. Consistent with the picture, the difference is statistically significant between 6 months and 6 years, but not in the youngest or oldest groups.

Controls in this regression include child age, maternal age, maternal education, birth order dummies and total number of siblings. In general, these enter with the expected sign and are unremarkable. However, the control for total number of siblings is worth a brief discussion. It is frequently suggested that one of the major reasons why female mortality is higher is that girls are, on average, in larger families (due to some form of gender-biased stopping rule). Although this seems to be somewhat true (i.e. the India-girl interaction is larger when I exclude controls for total siblings), the effect of vaccination is independent of this. Since I have controlled for total siblings in the regression here, it is clear that there is

some discrimination in vaccination that goes on independent of differences in family size.

The results in Table 1 give a sense of the magnitude of excess female mortality in childhood and the periods of childhood which are most crucial. A related question is how important childhood is in explaining the overall gender imbalance. To get a sense of this issue I calculate the predicted sex ratio in the population (based on life tables) taking the sex ratio at age five as given. If the predicted sex ratio in the population based on this calculation is much lower than the actual sex ratio then it suggests that any excess mortality up to age 5 is probably unimportant in the overall gender bias. If, in contrast, the predicted and actual sex ratios are similar then this suggests the excess mortality up to age 5 explains nearly all of the overall gender imbalance.

The result of these calculations appears in Table 2 (details of the calculation are in Appendix A). The results suggest that a very large share of the gender bias can be explained by events occurring up to age 5. This, in turn, suggests that understanding the proximate causes of mortality in this age bracket may go very far in helping us understand the overall problem. This is, perhaps, not surprising. Mortality rates among young children are much higher than among prime-age adults, so we would expect this to contribute a large share of the gender imbalance simply because the level is higher. It is worth noting, however, that the relationship is not mechanical. Even though mortality from zero to six months is much higher than mortality later in childhood, that period does not contribute very much to the gender imbalance.

Individual Level Regression

I turn now to estimating the importance of different health investments in explaining this excess female mortality in childhood, beginning with the individual regression methodology. This analysis will only be possible with vaccinations, and only using surveys from the early 1990s. Information on malnutrition and medical treatment is not collected for children who have died, and later surveys did not ask about vaccinations for deceased children.

Table 3 shows the regressions evaluating the effect of vaccinations by comparing the results of estimating equations with and without controls for vaccination. The regression is limited to children born 4-5 years ago, and the dependent variable is a dummy for having died

between 18 months and 4 years, conditional on having lived to 18 months. The sample size is smaller than for the similar age group in Table 1 because we use only children born 4-5 years ago, not all children born in the last 10 years. Column 1 estimates equation 2 and Column 2 estimates equation 3; as discussed in the methodology section, the share explained is calculated as the difference in the interaction coefficient divided by the interaction coefficient in Column 1. Vaccinations have a significant negative effect on mortality. Moving from zero vaccinations to a full set decreases the probability of dying between ages 1 and 4 by a full 1.8 percent. In addition, vaccinations seem to explain a large share of the gender imbalance, around 30%. The standard errors are sufficiently large that we cannot reject equality of the coefficients (i.e. we cannot reject that the amount explained is equal to zero). However, the size of the point estimate is certainly economically significant.

One possible concern with this analysis is recall bias. As mentioned, I consider both vaccinations reported on the health card and vaccinations only reported by the mother. If mothers in India are less likely to remember vaccinations for girls who have died, relative to boys who have died, then this could introduce some bias. Effectively, this would be like omitting a measure of true vaccination status, while including a measure of reported vaccination status. If true vaccination status (controlling for reported vaccination status) is correlated with the $girl \times India$ interaction, and with mortality, then the coefficient may be biased. This concern is ameliorated, at least somewhat, by the inclusion of both measures of vaccination. Marks on the health card are likely to be a much better measure of actual vaccination status than maternal reports, and the closer they get to the true vaccination the less of a concern the omitted variable bias is. Further, including only the control for vaccinations reported on the health card makes relatively little difference in the results.

There may also be concern that the effect of vaccinations varies by gender. If the benefit for girls from vaccination is larger than that for boys then this may understate the share of the biased explained by vaccination. Although there is some evidence on gender differences in the nonspecific protective effect of vaccinations (Aaby et al, 2002), these do not seem to be consistent across vaccines. As a sensitive analysis, I repeat the regressions in Table 3, allowing for the effect of vaccination to differ by gender. The results (available from the author) are virtually identical.

Direct Calculation of Medical Effects

The second methodology here relies on direct evidence about the effect of child investments on mortality. In contrast to the regression framework, this analysis will be possible for all of the investments considered: malnutrition, treatment for diarrhea, treatment for respiratory infections and vaccinations. I consider only measles vaccination because this is the illness for which we have the best estimates of the effectiveness of vaccination; obviously, the effect of measles alone will be an understatement of the total vaccine effect.

The calculations here require two elements: the difference in treatment by gender (D) and the effect of the treatment on mortality (μ). The first element is estimated in Table 4, which shows the gender bias in an indicator for severe malnutrition (Panel A), treatment for diarrhea (Panel B), treatment for respiratory infections (Panel C) and measles vaccination (Panel D). Controls are listed at the bottom of the table. The results indicate that boys in India are about 1 percentage point less likely to be malnourished, and that this effect is significant. The results on medical treatment are mixed: boys are significantly more likely to be treated for respiratory infections, but not any more likely to be treated for diarrhea. The largest observed effects are for measles vaccination; boys are approximately 7 percentage points more likely to be vaccinated.

Information on the second element – the effect of treatment on mortality – is presented in Appendix B. The details of the calculations appear in the Appendix but, in general, I use one of two techniques. In the case of malnutrition, I take advantage of studies in which nourishment levels of children were observed and then they were followed over time and mortality outcomes reported. The difference in mortality by nutritional status provides an estimate of the effect. In the case of treatment and vaccination, the effect is the product of the probability of dying from the illness (either diarrhea, ALRI or measles) and the protective effect of treatment. So, in the case of measles, the effect of vaccination on mortality is the chance of dying from measles in India during this period multiplied by the effect of measles vaccination on measles mortality. The studies suggest that the protective effect of being well nourished is the largest, although the effect of measles vaccination is much larger than treatment for illnesses.[13] The studies used here are based on information from the developing

[13]The larger effect of malnutrition does not seem to be an artifact of the difference in methodology. Using the

world, or from India directly, so they should capture the experience of South Asia reasonably closely.

Table 5 brings together the results from Table 4 and Appendix B and presents them with reference to the size of the gender imbalance. The first row of the table shows the excess female mortality between 1 and 4 years, and the share explained is simply the gender difference multiplied by the mortality effect, divided by this baseline difference. The results here suggest that food plays a sizable role in the gender imbalance (explaining around 16%), but that treatment for diarrhea and respiratory infections plays only a limited one. The reason for this is straightforward. In the case of diarrhea, there is virtually no difference in treatment propensity. In the case of respiratory infections, there *is* a large difference in treatment propensity, but the chance of dying from that cause is simply not that large and the protective effect of treatment is not that great. The effect of the measles vaccine provides a supportive robustness check on the earlier estimates of the effectiveness of vaccination form the individual-level regressions. Measles vaccination alone explains about 21% of the gender imbalance. Although this is less than the 28% estimated in Table 3, it is an estimate for only one of many vaccinations.

Regional Level Analysis

The results in Tables 3 and 5 suggest that around 50% of the gender imbalance up to age 5 can be explained by vaccination, food intake and medical treatment. One issue, however, is that these variables may not be independent. If malnutrition makes children more likely to die from measles, then the effect of malnutrition in Appendix B is also partially an effect of measles vaccination. This may lead the results in Table 5 to overstate the total explanatory power of the investments considered. Without an individual-level panel in which we observe all elements of food and treatment over time, this is a difficult problem to solve.

One option, however, is to try collapsing the data to the area level within India and then running regional-level equivalents to equations 2 and 3. Doing the same analysis

DHS data from Africa, it is possible to get an estimate of the effect of measles vaccination on mortality which effectively parallels the estimate of malnutrition. The result suggests around a 3 percentage point decrease in death probability with measles vaccination, similar to what is seen in Appendix B. Although I do not use this estimate, since the goal is to use estimates from outside these data, it does provide some comfort.

specified for the individual regression we can infer the share of mortality explained by different causes. There are clear issues with this approach. States within India differ on many dimensions and it may be difficult to fully control for these differences (I attempt to do so with controls for education, income and parental preferences, but fully controlling will be virtually impossible). However, the advantage of the approach is that we can consider the effect of all investments simultaneously, so it provides a useful robustness check.

The results of this analysis are in Table 6.[14] I show only the regression with no components of Z and the regression will all of the components of Z. What we will be able to conclude, therefore, is what share of the bias is explained by all of these elements together. The regression includes controls for average education level, average income and average ideal sex ratio reported (these are also at the gender-region level); the data here are limited to 1992. The explanatory power is similar to what we would expect based on the other analyses. Around 50% of the gender imbalance is explained by these components together.

There are obvious cautions with this approach. Nevertheless, the results are roughly consistent with the previous ones. At least some significant share of the gender imbalance – as much as 50 percent, perhaps – seems to be explained by two factors: vaccination and food intake. Of course, this result implies that at least half of the imbalance remains unexplained. One possibility is that, with more accurate data, more would be understood. Another is that there is an important element not considered here – for example, direct parental intervention. From a policy analysis standpoint, the fact that a large share of the gender bias remains unexplained is not necessarily problematic. Evaluation of vaccination intervention can still provide insight. If these results are correct, then eliminating the gender differences in vaccination could reduce the total population gender bias by around 30%, which is a significant change. I turn now to considering, both theoretically and empirically, how increases in vaccination availability in particular might affect the gender inequality.

[14]This analysis is run using India only. The sample sizes for Africa are much smaller, so there are only a very limited number of regions possible, making the comparison difficult. For simplicity, I assume that the coefficient on "girl" in the regression should be zero, understanding that this is not exactly correct.

3 Theoretical Framework of Parental Investment

This section analyzes a simple model of (gender-biased) parental investments in children. The question of interest here is how increases in access to health investments will affect the gender equality in those same investments. The previous section argues that vaccination is crucial in explaining the gender imbalance in mortality, and I therefore focus here on vaccination in particular. The general framework, however, may be applicable to other health investments.

Families have either a male or a female child, with measure 1 of each type of family. There is a unitary family utility function, which is separable over money and children. The utility of a girl is ϕ_g and of a boy is ϕ_b; boys are preferred, so $\phi_b > \phi_g$.[15] For simplicity, we will assume that utility of income is linear. The overall utility function for each family type is therefore:

$$\begin{aligned} U_g &= Y + \phi_g \\ U_b &= Y + \phi_b \end{aligned}$$

Parents have an opportunity to invest in vaccination for their children. Without vaccination, a child will live with probability p. With vaccination, they live with probability $\hat{p} > p$. The cost of vaccination for family i is $v + \varepsilon_i$, where $\varepsilon \sim N(0, \sigma^2)$. In this section I focus on this special case of normally distributed costs. However, the central conclusion holds true for a wider set of distributions; in Appendix C I discuss general results. Vaccination will be chosen for boys and girls, respectively, if the following inequalities hold.

$$\begin{aligned} \phi_b(\hat{p} - p) - v &> \varepsilon_i \\ \phi_g(\hat{p} - p) - v &> \varepsilon_i \end{aligned}$$

The share of boys vaccinated is, therefore, $F(\phi_b(\hat{p} - p) - v)$ and the share of girls is $F(\phi_g(\hat{p} - p) - v)$, where $F(.)$ is distributed normally. The gender inequality in investment is

[15] The assumption of one child families is obviously a simplification. However, it is not crucial. In an earlier version of this paper (available from the author) I developed a version of the model in which families were endowed with existing boy and girl children. In this model I find that discrimination against girls is predicted to decrease with the number of existing male children and increase with the number of existing female children. However, the results on access (below) hold as long as, on average, boys are preferred to girls. Note that this can hold even if the only reason girls are discriminated against is that they are in larger families.

measured by the difference in these shares: $F\left(\phi_b\left(\hat{p}-p\right)-v\right)-F\left(\phi_g\left(\hat{p}-p\right)-v\right)$. The analysis focuses on the change in this quantity as v changes.[16]

Denote the gender difference Θ. Under the assumption of normally distributed costs, this difference is simply

$$\Theta=\int_{\phi_g(\hat{p}-p)-v}^{\phi_b(\hat{p}-p)-v}\frac{1}{\sqrt{2\pi\sigma^2}}\exp\left(\frac{-\left(x\right)^2}{2\sigma^2}\right)dx$$

Integrating out, and differentiating with respect to v implies that

$$\frac{d\Theta}{dv}=\frac{1}{\sqrt{2\pi\sigma^2}}\left(-\exp\left(\frac{-\left(\phi_b\left(\hat{p}-p\right)-v\right)^2}{2\sigma^2}\right)+\exp\left(\frac{-\left(\phi_g\left(\hat{p}-p\right)-v\right)^2}{2\sigma^2}\right)\right)$$

The sign of this differential, however, changes based on v. The result is summarized in Proposition 1.

Proposition 1. *When vaccination costs are high on average, decreases in the cost result in increased pro-male bias. As average vaccination costs decrease, the sign of this effect switches and further decreases results in decreases in gender bias.*

Proof. The proposition claims that $\frac{dD}{dv}$ is negative for high v and positive for low v and moves from negative to positive as v decreases. To prove, we show a sufficient set of conditions: $\frac{dD}{dv}>0$ when $v=0$, $\frac{dD}{dv}<0$ when $v=\infty$ and $\frac{dD}{dv}$ is decreasing everywhere as v increases. To show this, note that the derivative suggests that $\frac{dD}{dv}<0$ when $\left(\phi_b\left(\hat{p}-p\right)-v\right)^2-\left(\phi_g\left(\hat{p}-p\right)-v\right)^2<0$. When $v=0$, $\left(\phi_b\left(\hat{p}-p\right)\right)^2-\left(\phi_g\left(\hat{p}-p\right)\right)^2>0$, so $\frac{dD}{dv}>0$. When $v=\infty$, the difference is negative, so $\frac{dD}{dv}<0$. Differentiating with respect to v, we find that the object is decreasing when $-2\phi_b+2\phi_g<0$, which will hold everywhere since $\phi_b>\phi_g$. □

The proposition suggests that beginning from a situation with very high vaccination costs (hence, limited vaccination), increases in access will actually make gender inequality worse. Further increases, however, are predicted to decrease gender inequality again.

To see the graphical intuition behind the result, consider Figure 3. This figure graphs two possible cost distributions with different levels of v; the dotted line represents a distribution with better access to vaccination (lower v). The cutoffs W_1, M_1 and W_2, M_2 represent two sets of vaccination cutoffs (W_x is the cutoff for women, M_x for men). The mass

[16] To see why this is the quantity of interest, consider that the overall sex ratio (given equal shares of boys and girls born) is equal to $\frac{p+(\hat{p}-p)F(\phi_b(\hat{p}-p)-v)}{p+(\hat{p}-p)F(\phi_g(\hat{p}-p)-v)}$. As F_b-F_g increases, this will increase; as it decreases, this will decrease. The difference between the two therefore maps into the ultimate object of interest, which is the sex ratio.

of the distribution under the cutoff is vaccinated, so the W_1, M_1 cutoffs represent a world with overall higher vaccination levels. Consider what happens to the gender difference in vaccination when we move from the solid to the dotted distribution, which represents a decrease in v. For the case of W_2, M_2 this movement causes a greater increase in the share vaccinated for men than for women, because both lines are on the increasing part of the distribution. In contrast, for the case of W_1, M_1, the increase causes a greater improvement for women, because both lines are on the decreasing part of the distribution. It is this intuition that is central to the result.

In Appendix C I discuss the generality of this result. Although it will not be true for all cost distributions, the intuition in Figure 3 is robust, and the fact that this is negative at high values of v and positive at low values will be true in general for single peaked distributions. If we take this framework seriously, it suggests that improvements in access to health care are not always the path to decreased gender inequality, at least in the short run. The next sections focuses on testing the theory in the context of vaccinations and connecting this intuition with results on mortality imbalances over time.

4 Results on Gender Inequality and Access to Vaccination

The theoretical framework above points to possible non-monotonic effects of access to health investments on the gender balance of these investments. This section focuses on testing this theory in the context of vaccination.

Health and Welfare Camps

The primary test focuses on the relationship between gender differences in vaccination and availability of "vaccination camps" in villages. I take advantage of a question in the 1998 NFHS which asked about "Family Health and Welfare Camps" in villages in the past year. Beginning in the mid-1990s, the government of India financed a campaign to bring better maternal and child health care to rural areas. This came at least partly in the form of health

camps, where childhood immunizations, prenatal care etc, were offered. In the 1998-1999 NFHS survey, the village representative (in rural areas only) was asked about the number of health and welfare camps in the village in the previous year.

The number of camps varied quite a bit, which allows us to ask a simple question: is the difference in vaccination by gender non-monotonically related to the number of camps? To the extent that we can view increases in the number of camps as changes in access to vaccination, this seems like a reasonable test of the theory. As a first pass, the relationship is shown graphically in Figure 4, which reports the difference in vaccinations for children 6 months to 2 years graphed against the number of vaccination camps last year, as well as the average number of vaccinations for each gender.[17] The figure points to a non-monotonic relationship. Moving from zero or one camp to two camps causes a large increase in the gender difference. This increase continues up to three camps, after which girls begin to gain again. The maximum gender difference is at 3 camps, and the gender difference is similar at zero or one camp, and at 5 or more. Of course, the average number of vaccinations is generally increasing for both boys and girls as the number of camps increase, so children are, on average, better off in areas with four or five camps than areas with none.

Table 7 explores the relationship in a regression context, with controls (including individual demographic controls, as well as fixed effects for state of residence). All standard errors are clustered at the village level. Column 1 assumes that the relationship is quadratic, and estimates the coefficient on the interaction between girl and number of camps, and the interaction between girl and the number of camps squared. The number of camps is top-coded at 5. Although some areas report as many as 40 camps in the last year, 98% report 5 or fewer. The coefficient estimates do point to a non-monotonic relationship. The linear interaction term is negative (increases in vaccination camps increase discrimination), but the squared term is positive. The magnitudes suggest that the effect is zero around 4.2 vaccination camps per year.

In Columns 2 and 3 of Table 7 I consider an alternative to the assumption of a quadratic functional form – estimating separately for areas with few camps and areas with

[17] I restrict to children in this age group since they are the ones who would have needed vaccinations in the previous year. Consistent with this, the results are less strong for older children.

many. Column 2 estimates the effect of vaccination camps in areas with fewer than 3 camps, and Column 3 estimates the effects in areas with more than 3. The results are consistent with Column 1. In areas with limited vaccination camps, increases seem to make girls worse off relative to boys. In areas with more camps, increases improve the relative position of girls.[18]

To further test the theory, I consider two sample splits. First, I divide the sample based on women's reported ideal sex ratio. Women in the survey were asked about their ideal number of male and female children. Since the conclusions in Proposition 1 apply only if male children are preferred to female children, the results should be stronger for the set of women who report wanting more male then female children.[19] I estiamte the regression in Column 1 of Table 7 separately for women who report wanting equal numbers of male and female children, or more female children and for women who report wanting more male children.

The regressions are shown in Columns 1 and 2 of Table 8. Column 1 includes women who do not want a male-biased sex ratio among children; Column 2 includes those who do. The results are consistent with the theory. In Column 1 neither interaction between gender and number of camps is significant. In Column 2, however, the interactions are strongly significant and large. The contrast suggests that, as predicted, this relationship holds more consistently for families with more gender-biased preferences.

A second sample split-based test of the theory relies on the observation that vaccination camps are not the only way that children get vaccinated. Given this, the non-monotonicity should be stronger in areas where vaccination camps are more important. In the NFHS, women who have a young child are asked where most of the vaccinations of the child took place. The majority of women report either a Primary Health Center (PHC), a Community Health Center (CHC) or a Government Hospital. For women with easy access to these sources, it seems likely that vaccination camps will be less important.

I divide the sample roughly in half based on the distance to the closest PHC, CHC or

[18]The magnitude of the interaction, relative to the level effect of camps, would suggest that initial increases in vaccination camps actually make girls *worse* off in an absolute sense, which is not consistent with the pattern in Figure 4. This is due to the inclusion of state fixed effects, which are highly correlated with the number of camps. When these are excluded the interaction results remains the same, but the level effect of camps is much larger.

[19]Obviously reported ideal number of male and female children is not a perfect measure of gender preferences. However, it should provide some proxy. In addition, it is certainly the case that the gender inequity in vaccination is larger for families where parents report wanting more male children (results available from the author).

Government Hospital and explore whether the relationship between gender inequality and number of camps differs by the distance to these other sources. Columns 3 and 4 of Table 8 show these results. In Column 3 (nearest other source less than 5 km away), the interactions are not significant and are small. In contrast, in Column 4 (nearest other source at least 5 km away) the nonlinear relationship is large and significant.[20]

Both sample splits in Table 8 bolster the conclusions in Table 7. The effect is largest for families with limited access to other sources of vaccination, and for those with stronger male-biased preferences. Another obvious test would be to explore whether the inequality-access relationship holds less strongly for families where there are two children of opposite gender close in age. Intuitively, in families where a female child needs vaccination at the same time as a male child we may not get these relationships with access. Unfortunately, the sample sizes are too limited (given the necessary closeness in age – a year or less) to do this test.

One possible issue with the entire analysis is the non-random placement of camps. Although I have controlled for a number of possible correlates of camp placement in Table 7, I have not ruled out the possibility that the camps were placed in areas with an eye to gender inequality. Although it is not obvious why this would produce the non-linear relationship, it seems worth considering a test of this claim. If we aggregate at the state level, it is possible to compare the 1992 and 1998 surveys. To test the possibility of placement related to gender differences, I calculate the gender imbalance in vaccines by state in 1992 and regress average vaccination camps by state on this measure.

These results are shown in Table 9. In Column 1 the dependent variable is the number of vaccinations camps truncated at 5 (as in the earlier regressions). In Column 2 it is simply the number of camps, without truncation. In neither case does the difference in vaccination in 1992 influence the number of vaccination camps. In addition to being insignificant, the difference is small. Moving from the 25th to the 75th percentile of vaccination difference in 1992, we would predict an increase of 0.1 camps in 1998, on a base of around 2 camps. Although obviously this does not rule out non-random placement, it does

[20] An alternative way to do this would be to separate the sample based on the share of women in the area who report vaccination camps as the primary source of vaccination. If I do this, the results are extremely similar – the quadratic relationship is stronger and more significant in areas where vaccination camps are more important.

provide some comfort that the placement isn't obviously related to the variable of interest. Although camp placement does not seem to be closely related to some simple state-level demographics (i.e. income and education) there is significant variation across states. In Andrha Pradesh, villages sampled had an average of 3.55 camps in the previous year, whereas in Jammu the average is around 0.1. To the extent that state-level characteristics influenced the number of camps that arrived in a village, this points to the importance of state-level fixed effects, which are included in all regressions above.

Alternative Proxies for Vaccination Access

A significant advantage of the analysis above is that vaccination camps are likely to operate in large part as shocks to the availability of vaccination in the village. This makes them less obviously correlated with existing conditions and they provide a relatively clean test. In addition, their discrete nature makes the exploration of non-monotonic effects relatively straightforward. The theoretical framework, of course, is not specific to vaccination camps (or even to vaccination). In general, we expect discrimination to be non-monotonic in any measure of access to vaccination. I therefore consider also the relationship between the level of vaccination and the gender difference using other proxies for access.

I first proxy for access using the reported distance from the nearest Primary Health Center, Community Health Center or Government Hospital, as reported in the village survey in the 1998 NFHS. Approximately 50% of women report this as their source of immunization, so it seems like a good proxy for access. Of course, access to a these centers has other implications and is presumably correlated with unobservables. However, there is no obvious bias that would produce a non-monotonic relationship in gender imbalance.

Table 10 shows the relationship between total vaccinations and gender, interacted with both distance and distance squared. In this case the theory would predict that the interaction with distance to be negative, and with distance squared to be positive.[21] A sizable fraction of people report having a health facility in their village. These are coded as zero distance although, of course, this may not be strictly correct. In Column 1 of Table 10 I show

[21] These predicted interactions have the opposite sign from the interactions on number of camps because increases in the number of camps imply increases in access and increases in distance imply decreases in access.

the regression with all observations; in Column 2 I restrict to people who report a non-zero distance, to avoid any issues with the in-village measure. The coefficients have the expected sign and are significant (at least at the 10% level) in both columns. If anything, the results are stronger in Column 2, when we leave out people who report having a health facility in their village. The relationship switches sign at around 30 km in distance.

As a final test, I consider the cross-regional relationship between the level of vaccination and the gender difference in vaccination. In this case, the level of vaccination is the proxy for vaccination access. Even more than the analysis with Primary and Community Health Centers, this is subject to bias. However, it *does* provide the only opportunity to consider this relationship outside of rural villages.

I take advantage of the cluster design of the NFHS. I aggregate the data to the cluster level and calculate the average number of vaccinations and the difference between this average for boys and girls. The primary regression will consider the shape of the relationship between the level and the difference. The results are shown in Table 11, where the dependent variable is the gender difference in average number of vaccines received; Column 1 considers a monotonic relationship between the level and the difference and Column 2 considers a non-monotonic relationship. The results seem consistent with a nonlinear relationship; the coefficient in Column 1 is small, and not significant. In Column 2, however, both the average and the average squared are significant and have the expected sign. The number of vaccinations ranges from 0 to 7. The magnitude of the coefficients suggests that the gender imbalance is increasing up to an average of 5 vaccinations and decreasing thereafter.[22]

5 Non-Monotonicities in Mortality over Time

The evidence in Section 4 focuses on vaccination directly. Together with the results in Section 2, this suggests that the gender imbalance in mortality may change non-monotonically over time. The evidence above, however, does not directly establish this relationship. In this section I discuss direct evidence on non-monotonicities in mortality. The first subsection

[22]This result is in contrast to the results in Pande and Yazbeck (2003), who argue that gender differences in immunization across states do not seem to be related to levels. This may underscore the importance of considering the regional relationship at a less aggregated level, or relying on the vaccination camp analysis.

discusses mortality among children in the data here; the second takes a broader view and considers overall life expectancy in India over time.

5.1 Childhood Mortality 1982-1993

Given the focus on childhood and child health investments in the sections above, it seems reasonable to first consider non-monotonic changes in gender inequity in mortality in this age group. Ideally, we would have relatively long time-series in which child mortality by gender is observed. These type of data are not, however, generally available. It is possible, however, to create a short time series using retrospective reports on child mortality in the two survey waves of the NFHS.

I consider the mortality outcomes for children born five to ten years before each survey year (1992 and 1998). This effectively creates a time series of child death rates from 1982 through 1993. The outcome of interest is mortality between 18 months and five years. I do not consider very early life mortality, since it will be generally unaffected by vaccinations. In addition, I limit to children under the age of ten to avoid (as much as possible) the chance that mothers have forgotten to list children who were born many years ago and died early in life.

The ideal analysis would look at the existence of non-monotonic changes in the gender balance of mortality over time within a particular area. Unfortunately, with such a short time series, there are no areas that have gone through a sufficiently large change. What I do instead is consider whether the change in the gender imbalance in mortality over this short period is different in regions with different initial levels of vaccination.

Using information on average number of vaccinations by state in the 1992 and 1998 I first restrict the analysis to states in which there was an increase of at least 0.1 vaccinations between the two survey years.[23] I then divide the remaining states into two groups based on their initial level of vaccination and using information from Figure 4 on the level of vaccination at which the relationship flips sign. There are relatively few areas with many vaccinations, and I restrict this group to states where the average number of vaccinations by 1998 is at least

[23]This eliminates Rajasthan, Bihar, Uttar Pradesh, Haryana, Gujarat, Arunachal Pradesh, Assam, New Delhi and Mizoram.

3 (Tripura, Jammu, Himachal Pradesh, Kerala and Goa).[24] All remaining states are assigned to the other group. Recall that the theory would suggest increases in relative female mortality in areas with initially low vaccinations and decreases in areas with initially high levels.

The results from the regressions in the two groups are shown in Table 12. Column 1 shows the regression in the low initial vaccination group and Column 2 in the high vaccination group. The coefficient of interest is that on the interaction between girl and time of birth. The dependent variable is an indicator for having died between 18 months and 5 years, so the theory suggest a positive coefficient in Column 1 and a negative coefficient in Column 2. In addition to the simple demographic controls shown, I have controlled for birth order dummies and dummies for each region, as well as these regional dummies interacted with gender. This controls for the possibility that low (or high) levels of vaccination are correlated with overall levels of gender discrimination.

The results in Table 12 are supportive of the theory. In Column 1, the coefficient on the interaction is positive. The magnitude suggests that during the ten year period considered, female mortality increased relative to male mortality by 0.74 percentage points. In contrast, the coefficient in Column 2 is negative. During the same ten year period, female mortality in these regions decreased relative to male mortality by 0.96 percentage points. These are both quite big effects, since the average mortality in this age range is around 3 percent.

5.2 Life Expectancy in India, 1870-2000

Given the effects on child mortality, it seems interesting to consider whether it is possible to see these non-monotonicities in wider data – in particular, in data on life expectancy in India over the last century. This moves away from the earlier analysis in two respects. First, life expectancy and child mortality are not identical, although they are closely linked, since a major component of increases in life expectancy at low levels is decreases in infant mortality. Second, the issue of vaccinations is important later in the century, but not in the early part.

[24] The evidence from Figure 4 would suggest that the cutoff is higher than this. However, what we observe in Figure 4 is the average vaccination level *after* the vaccination clinics. Presumably the vaccination level was lower before, making three a not-unreasonable cutoff. In addition, because this is done at the state level (due to the fact that I cannot link smaller areas in the data over time), there are virtually no states with very high levels of vaccination. It is worth noting that these issues will only make it more difficult to find evidence of non-monotonicities.

The analysis here looks for a more general correlation between level of health investments and the gender difference in mortality. Evidence for such a relationship might suggest that there are non-monotonicities across genders in other health investments.

The Indian Census (formerly the British Census of India) reports life expectancy for men and women over the period from 1871 to the present. Using these data, it is possible to construct series of the average and the difference between genders for this relatively long period. These series are graphed in Figure 5. In these data, particularly after 1910, we see evidence for a U-shaped relationship in the difference, accompanied by an overall increase in the average (the general pattern of changes for men and women over the century is very similar). In the 1910-1920 period, female life expectancy exceeds men by around 1.5 years, although the average is very low, at around 20. By the 1960-1970 time frame, female life expectancy is actually 1.5 years *less* than men, but the average has increased to around 45. Women seem to rebound by the end of the period – women in 1980-1990 have a life expectancy about 1 year greater than men, and the average has climbed to almost 60 years.

This may shed some light on overall changes in the sex ratio in India over the last hundred years. It is well known that population female-male sex ratios (the ratio of women to men in the population) have been falling over time. This is consistent with increases in discrimination, but it is also potentially consistent with a constant level of discrimination and the non-monotonic effect discussed above. The evidence on life expectancy in Figure 5 would suggest that, if this is true, the sex ratio in the population should not continue to decline, but should rebound toward the end of the century.[25] Interestingly, as can be seen in Figure 1, we do potentially see some evidence of a rebound, or at least a flattening of the trend. Although, as noted in the Introduction, the general movement over the century is sharply downwards, there does seem to be a flattening in the trend after 1970 and there is actually some increase between 1990 and 2000. The timing, at least, is similar to the trend in life expectancies.

The drop in sex ratio over time has caused significant policy concern (Mayer, 1999). The graphs here do not necessarily demonstrate that changes over time are related to non-monotonic changes in the gender imbalance in health investments. However, taken

[25]This ignores the introduction of sex-selective abortion, which has become increasingly important recently (see, for example, Jha et al, 2006). Obviously this would push things in the other direction, which is important to keep in mind.

together with the evidence on vaccinations, the connection is suggestive. If true, it may lead us to expect further changes in the patterns of mortality over time – moving back into a situation where the imbalance is similar to what it was at the start of the century, but the level of mortality is much lower.

6 Conclusion

There has been much policy focus, in particular in India, on increasing access to health services as a way to decrease gender inequality. This paper explores the evidence supporting such interventions. I argue increasing access to vaccination is an obvious intervention of this type, since differences in vaccination appear to explain about 30% of the excess female mortality in the Indian population. However, theory and evidence indicates the effect of increases in vaccination is not clear. At low levels of immunization, increasing access is likely to make the gender imbalance worse, although further increases are likely to improve it.

The analysis in the sections above is primarily positive. I focus on establishing the relationship between vaticination availability and gender imbalance. This leads to the question of whether there are normative conclusions to be made here. Do these results suggest a particular policy approach?

It is worth noting, first, that technological change, in the form of sex-selective abortion, may increasingly render this issue moot. It has been recently argued that as many as ten million female fetuses were aborted in the last fifteen years (Jha et al, 2006). In the limit, if use of this technology increases, it could be the case that virtually all discrimination is moved to the period before birth. In this case, conditional on being born, there should be limited discrimination and we should see no non-monotonic effect of increases in vaccination access. This scenario seems unlikely, in part because Indian authorities have moved to prevent increases in the use of these technologies and in part because abortion may actually *not* be less costly than neglect. Nevertheless, it may temper any possible policy ramifications here.

Abstracting away from recent changes, the potential policy conclusions here depend largely on what welfare function we hope to maximize. These results suggest that increases in access to health inputs will make everyone better off; they just make girls better off more

slowly than boys. If the primary concern is decreasing mortality, then a policy that seeks to increase access to health investments may be optimal. If, on the other hand, the policy-maker cares independently about the sex ratio, then it may be prudent to think more carefully about the mechanics of health interventions.

To be more concrete, the non-monotonicity would suggest that if policymakers care directly about the sex ratio, interventions to increase access to health inputs should focus on saturating one area rather than doing some in all areas. If we take the results in this paper quantitatively seriously, it is possible to directly evaluate these tradeoffs. In particular, combining the results behind Figure 4 with the effect of vaccination estimated in Table 3 can tell us the effect of different methods of vaccination camp dispersion. Consider the difference between moving from zero to three camps in all areas of the country, versus moving from zero to some kind of saturation (i.e. at least 4) in half of the regions. The calibration suggests that moving from zero to three camps in all areas would increase survival by, on average, 10 lives per 10,000 children. However, it would increase the sex ratio by 11 for every 20,000 children. In contrast, moving from zero to saturation in half the regions would also increase survival, on average (including non-affected areas), by 10 lives per 10,000. However, in this case the sex ratio would be virtually unchanged. Depending on how the policymaker wants to trade off gender discrimination versus spatial discrimination, at least in this example, will give guidance about modes of introduction.

Appendix A: Calculating the Importance of the Childhood Gender Imbalance

The calculation behind Table 2 uses life tables from Coale et al (1983). In order to do this it is necessary to pick a life table that corresponds to the mortality level in the country and to the reproductive rate. The first piece (the mortality level) provides information about the likelihood of death for each gender at each age group. The second piece (the reproductive rate) combines with the mortality rate to give information on what share of the population is in each age group at any given time.

I use the West Model life tables, with a mortality level of 17 and a gross reproductive rate of 4. This corresponds relatively closely with the experience of India in the mid 1990s. The table below shows the mortality rate and the share of men and women in each age group based on this choice of life table and reproductive rate.

Age	**Male Mort. Prob.**	**Female Mort. Prob**	**Sh. Men**	**Sh. Wom**
0-1	8.62%	7.07%	4.99%	4.90%
1-5	3.50%	3.32%	17.03%	16.88%
5-10	1.18%	1.10%	17.18%	17.04%
10-15	0.88%	0.85%	13.68%	13.57%
15-20	1.38%	1.25%	10.87%	10.80%
20-25	1.95%	1.65%	8.60%	8.56%
25-30	2.09%	1.91%	6.78%	6.76%
30-35	2.38%	2.19%	5.33%	5.33%
35-40	2.89%	2.55%	4.17%	4.18%
40-45	3.75%	3.02%	3.25%	3.27%
45-50	5.01%	3.79%	2.50%	2.54%
50-55	7.04%	5.23%	1.89%	1.95%
55-60	9.94%	7.27%	1.39%	1.47%
60-65	14.42%	10.93%	0.98%	1.08%
65-70	20.66%	16.33%	0.65%	0.75%
70-75	29.78%	25.08%	0.40%	0.48%
75-80	42.31%	37.29%	0.21%	0.27%
80+			0.11%	0.16%

I first calculate the expected sex ratio (women divided by men) in the population using the observed sex ratio at birth. To do this, I assume that at birth there are 1000 women for 1069 men (the observed sex ratio at birth is 0.936) and calculate the expected sex ratio at the end of each age group based on the mortality patterns by age. I then weight the sex ratios by the share in that age group, which results in the total sex ratio. This calculation is shown in the table below.

Age	# Men	# Women	Sex Ratio (F/M)
0	1069.000	1000.000	0.935
1	976.842	929.340	0.951
5	942.691	898.532	0.953
10	931.577	888.675	0.954
15	923.370	881.086	0.954
20	910.646	870.090	0.955
25	892.888	855.734	0.958
30	874.253	839.423	0.960
35	853.481	821.040	0.962
40	828.816	800.128	0.965
45	797.760	775.932	0.973
50	757.768	746.532	0.985
55	704.406	707.459	1.004
60	634.402	656.026	1.034
65	542.915	584.310	1.076
70	430.754	488.874	1.135
75	302.471	366.255	1.211
80	174.496	229.697	1.316
Sex Ratio Weighted by Age Shares		**0.959**	

The observed sex ratio in the population is around 0.927, quite different from the 0.959 predicted based on normal mortality patterns and the observed sex ratio at birth.

The second step is to consider the same calculation, except assuming that the sex ratios observed up to age 5 are naturally occurring. This calculation appears in the next table.

Age	# Men	# Women	Sex Ratio (M/F)
0	1069.000	1000.000	0.935
1	1071.000	1000.000	0.934
5	1094.000	1000.000	0.914
10	1081.102	989.030	0.915
15	1071.577	980.584	0.915
20	1056.811	968.346	0.916
25	1036.203	952.368	0.919
30	1014.578	934.216	0.921
35	990.471	913.757	0.923
40	961.847	890.483	0.926
45	925.806	863.555	0.933
50	879.395	830.835	0.945
55	817.468	787.349	0.963
60	736.228	730.109	0.992
65	630.057	650.293	1.032
70	499.893	544.081	1.088
75	351.020	407.615	1.161
80	202.504	255.635	1.262
Sex Ratio Weighted by Age Shares		**0.925**	

The predicted sex ratio is now 0.925, quite close to the actual sex ratio of 0.927. This suggests that the observed population sex ratio is actually very close to what we would expect *if* the sex ratio at age 5 was naturally occurring.

Appendix B: Estimating the Effect of Medical Treatment on Mortality

Study	Effect	Methodology
Panel A: Food		
Chen et al, 1980	14.61%	Children were weighed and measured and followed for two years. Mortality rate is calculated based on an extension of the 2-year window to the entire period.
Sommer and Lowenstein, 1975	20.40%	Children were weighed and measured and followed for 18 months; this study is divided up by age group. I calculate the one year mortality rate for each age, and add them together to get the total mortality rate for the entire period.
Panel B: Diarrhea		
Rahaman et al, 1979	1.80%	The study used reported the chance of dying in a village with 80% treatment and the chance of dying in a village with 40% treatment. I used this information to interpolate the chance of dying with no treatment, and the chance of dying with treatment, and calculate a protective effect. I then calculate the share of deaths attributed to diarrhea in this area from Murray et al (1996) and multiply that by the chance of dying in this age group, which gives the total chance of dying from diarrhea. I assume this is the chance of dying if not treated and calculate the chance of dying if treated, and subtract.
Panel C: Respiratory Infections		
Ali et al, 2001	0.92%	The study used reported the chance of dying in places with ALRI treatment and non-treatment programs. I used this information calculate a protective effect. I then calculate the share of deaths attributed to ALRI in this area from Murray et al (1996) and multiply that by the chance of dying in this age group, which gives the total chance of dying from ALRI. I assume this is the chance of dying if not treated and calculate the chance of dying if treated, and subtract.

continued on the next page

Study	Effect	Methodology
Fauveau et al, 1992	1.02%	The study used reported the chance of dying in places with ALRI treatment and non-treatment programs. I used this information calculate a protective effect. I then calculate the share of deaths attributed to ALRI in this area from Murray et al (1996) and multiply that by the chance of dying in this age group, which gives the total chance of dying from ALRI. I assume this is the chance of dying if not treated and calculate the chance of dying if treated, and subtract.
Panel D: Measles Vaccination		
Clemens et al, 1988	2.52%	Case control study of measles vaccination in Bangladesh. Compared to the matched controls, vaccinated children had 36% lower mortality. I combine this with the baseline probability of dying after the first six months but before age 5 in regions with little or no vaccination, which I estimate to be 7%. Multiplying yields the result.
Koenig et al, 1990	3.22%	Case-control study of measles vaccination in Bangladesh. Compared to the matched controls, vaccinated children had 46% lower mortality. I combine this with the baseline probability of dying after the first six months but before age 5 in regions with little or no vaccination, which I estimate to be 7%. Multiplying yields the result.

Appendix C: General Theoretical Results

This appendix discusses the general form of the result in Section 3. As there, we note that the conditions for vaccinations for boys and girls are

$$\begin{aligned} \phi_b(\hat{p}-p)-v &> \varepsilon_i \\ \phi_g(\hat{p}-p)-v &> \varepsilon_i \end{aligned}$$

and that the difference of interest is $F(\phi_b(\hat{p}-p)-v)-F(\phi_g(\hat{p}-p)-v)$. In the text I focus on the case where $F(.)$ is normal and show results indicating that at high v this difference will increase with decreases in v and at low v the difference will decrease with decreases in v. Here, I discuss what must generally be true about the distribution for this to hold.

This difference Θ can be represented

$$\Theta = \int_{\phi_g(\hat{p}-p)}^{\phi_b(\hat{p}-p)} f(x-v)\,dx$$

Differentiating this with respect to v yields:

$$\frac{d\Theta}{dv} = -\int_{\phi_g(\hat{p}-p)}^{\phi_b(\hat{p}-p)} f'(x-v)\,dx$$

In order for this to be negative at high v and positive at low v, we simply require that $f(.)$ be increasing in the left tail, and decreasing in the right tail. Any single-peaked distribution will have this property.

In contrast to the case of the normal, where this is decreasing everywhere in v, we will not be able to prove that generally. However, the intuition that girls become relatively worse off with decreases in v at high v and relatively better off with decreases in v at low v will be true for a wide class of distributions.

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Table 1. ***Gender Imbalance in Death by Age, All of India***

	Dependent Variable: Child Died in Given Age Range (0/1)						
	<6mons	6mons-1yr	1-2yrs	2-4yrs	4-6yrs	6-8yrs	8-10yrs
Explanatory Variables:							
Girl	−.0104***	.0002	−.0011*	−.0014***	0	0	.0007
	(−6.97)	(.33)	(−1.81)	(−2.66)	(−.04)	(−.02)	(1.21)
Girl × India	.0016	.0021***	.0055***	.0041***	.0026***	.0005	−.0006
	(.91)	(2.86)	(6.48)	(5.57)	(3.23)	(.7)	(−.91)
India	−.0077***	−.0135***	−.0175***	−.0098***	−.0088***	−.0032***	−.0008
	(−5)	(−16.12)	(−18.61)	(−12.65)	(−10.29)	(−4.58)	(−1.16)
Income (durables)	−.0049***	−.0012***	−.0019***	−.002***	−.0029***	−.0015***	−.0012***
	(−12.77)	(−6.96)	(−9.21)	(−11.14)	(−13.98)	(−8.19)	(−6.63)
Child Age	−.0003***	−.0004***	0	0	.0007***	.0003**	.001***
	(−6.61)	(−3.89)	(.42)	(−.39)	(5.56)	(2.12)	(4.64)
Mother's Age	−.0026***	−.0004***	−.0005***	−.0002***	−.0003***	−.0001	0
	(−27.69)	(−7.42)	(−8.9)	(−4.95)	(−6.28)	(−1.31)	(−.46)
Mother's Educ	−.0014***	−.0004***	−.0007***	−.0003***	−.0003***	−.0002***	0
	(−13.27)	(−7.46)	(−10.85)	(−6.43)	(−6.33)	(−4.61)	(−.24)
# Siblings	.0257***	.0039***	.0045***	.0028***	.0025***	.0008***	.0005***
	(67.13)	(19.33)	(20.05)	(15.42)	(12.69)	(5.26)	(3.53)
Year	−.0007***	−.0004***	−.0001	.0001	−.0001	0	0
	(−4.57)	(−5.97)	(−1.03)	(1.5)	(−1.18)	(.16)	(−.42)
Number of Observations	338,943	294,407	278,396	243,496	211,896	150,801	86,604

Notes: All regressions are limited to children born in the last ten years. The dependent variable is a dummy equal to one if the child died within the specified age range *conditional on* having lived up until that age category. The measure of income is a measure of the number of durable goods owned, where the durable goods are radio, television, refrigerator, motorcycle, bicycle and car. Controls for birth order (dummies) are also included in all regressions. India is observed in 1992 and 1998. The African countries included are Ethiopia (2000), Kenya (1998, 2003), Malawi (2000), Namibia (1992, 2000), Tanzania (1996, 1999) and Zambia (1996, 2001).

[a] t-statistics in parenthesis

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

Table 2. ***Share of Overall Imbalance Explained by Age 5***

	All India
Observed Sex Ratio (F/M) Age 5	0.914
Predicted Sex Ratio, Normal Mortality after 5	0.925
Empirical Population Sex Ratio	0.932

Notes: Sex ratio is the number of women divided by the number of men. Observed sex ratio at age 5 is from the NFHS. The predicted population sex ratio is calculated by assuming that the sex ratio at age 5 is naturally occurring and then assuming "normal mortality" after age 5. Mortality is taken from the Coale, Demeny and Vaughn (1983) life tables, using the West Model mortality level 17 with a GRR of 4.0. Empirical population sex ratio is from the 1991 Census. The full calculations behind the table are described in Appendix C.

Table 3. ***Impact of Vaccines on Excess Female Mortality***

Dependent Variable: Child Died 1 year - 4 years		
	(1)	(2)
Explanatory Variables:		
Girl	$-.0013$	$-.0007$
	$(-.44)$	$(-.29)$
Girl × India	$.0091^{**}$	$.0065^{**}$
	(2.33)	(2.03)
India	$-.0233^{***}$	$-.0393^{***}$
	(-5.58)	(-8.13)
# Vacc. Rep. by Mom		$-.0009^{***}$
		(-3.72)
# Vacc. on Health Card		$-.0026^{***}$
		(-6.57)
Obs.	13,817	13,817
Share Expl.	**28.4%**	

Notes: Controls in all regressions include dummies for child size at birth, maternal age, maternal education, child age, income (durables), total number of children and dummies for birth order. The two measures of vaccines capture both the total number of vaccines reported by the mother and the total number marked on the health card (3 DPT vaccines, 2 polio vaccines, measles and BCG). The share explained is the difference between the coefficients on the interactions in Columns 1 and 2, divided by the interaction in Column 1.

[a] t-statistics in parenthesis

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

Table 4. ***Gender Imbalance in Malnutrition and Treatment of Illness***

Panel A: Malnutrition	
Dependent Variable: Child is Severely Malnourished	
Explanatory Variables:	
Girl	−.0025
	(−1.17)
India	.0342***
	(18.86)
Girl × India	.0116***
	(4.39)
Number of Observations	80819
Panel B: Diarrhea	
Dependent Variable: Child received treatment if had Diarrhea	
Girl	−.0176
	(−1.46)
India	−.1057***
	(−8.64)
Girl × India	−.0027
	(−.16)
Number of Observations	13666
Panel C: Respiratory Infections	
Dependent Variable: Child received treatment if had Cough or Fever	
Girl	−.0010
	(−.13)
India	.0666***
	(7.65)
Girl × India	−.0429***
	(−3.51)
Number of Observations	23154
Panel D: Measles Vaccination	
Dependent Variable: Child is Vaccinated for Measles	
Girl	.0356*
	(1.66)
India	−.411***
	(−27.64)
Girl × India	−.0698***
	(−2.98)
Number of Observations	15,120

Notes: Controls in all regressions include income, age of child, dummies for birth order, number of children in the household, maternal education and age.
[a] t-statistics in parenthesis
* significant at 10%; ** significant at 5%;*** significant at 1%

Table 5. ***Share of Missing Girls Explained by Food, Treatment and Vaccination***

	All India
Baseline Difference	-0.0115
Food (% Explained)	16.2%
Diarrhea Treatment (% Explained	0.51%
ALRI Treatment (% Explained)	4.4%
Measles Vaccination (% Explained)	21.0%

Notes: Baseline differences are drawn from Table 1. The share of the puzzle explained by each indicator is equal to the difference from Table 4 multiplied by the effect on mortality from Appendix Table 1 and then divided by the baseline difference.

Table 6. ***Regional Analysis of Proximate Causes***

Dependent Variable: Share of Girls/Boys in Region Who Died Ages 1-5		
	Without Controls	**With Controls for Vacc, Malnour.**
Explanatory Variables:		
Girl	.0156***	.0091**
	(4.03)	(2.52)
Vaccination		.0003
		(.27)
% Severely Malnourished		.0273
		(.89)
Vacc × Malnour.		.0018
		(.19)
Averge Mother Education	−.0015	−.0018*
	(−1.22)	(−1.67)
Average Income (durables)	−.0029	−.0032
	(−.68)	(−.83)
Ave. Ideal Sex Ratio	.0411***	.0113
	(3.73)	(1.07)
constant	−.03	.018
	(−1.6)	(1.01)
Number of Observations	339	338
R^2	.09	.06
Share explained	**46.3%**	

Notes: Each observation represents a gender-region and the dependent variable is the share of children born 5-10 years ago in that gender-region group who died between the ages of 1 and 4 years (conditional on reaching 1). The measure of vaccination represents the first principal component of indicators for having gotten six vaccines: measles, DPT(1,2,3) and Polio(1,2). Columns (3) and (4) are limited to 1992 data. "Ideal Sex Ratio" is the average ideal number of boys divided by the average ideal number of girls.
[a] t-statistics in parenthesis, * significant at 10%; ** significant at 5%; *** significant at 1%

Table 7. ***Vaccination Camps and Gender Imbalance in Vaccination***

Dependent Variable: Number of Vaccinations Child Has			
	(1)	(2)	(3)
	Entire Sample	**<3 Camps**	**≥ 3 Camps**
Explanatory Variables:			
Girl	−.1025**	−.0991**	−1.0272**
	(−2.25)	(−2.18)	(−2.1)
Girl × # Camps	−.1841**	−.1197**	.2316**
	(−2.14)	(−1.93)	(1.94)
Girl × # Camps Sq.	.0434**		
	(2.17)		
# Camps	.0761	.0194	−.1209
	(1.08)	(.37)	(−1.24)
# Camps Sq.	−.0152		
	(−.94)		
Child Age	1.7117***	1.6498***	2.2904***
	(6.79)	(6.16)	(3.1)
Child Age Sq.	−.5551***	−.534***	−.763**
	(−5.48)	(−4.96)	(−2.59)
# Older brothers	−.0232**	−.0194*	−.0335
	(−2.3)	(−1.78)	(−1.26)
# Older Sisters	.0231**	.0192*	.0425*
	(2.22)	(1.71)	(1.69)
constant	1.219***	1.61***	.816**
	(6.25)	(6.29)	(2.2)
State Fixed Effects	YES	YES	YES
Number of Observations	10854	9415	1439
R^2	.29	.29	.29

Notes: This table estimates the effect of vaccination camps on the gender imbalance in vaccination. An observation is a child aged 6 months to 2 years and the dependant variable is the number of vaccinations the child has received (0-6). # Camps is the number of Family Health and Welfare Camps reported in the village last year – 0, 1, 2, 3, 4 or 5 or more. Column 1 includes all areas. Column 2 includes only areas where the number of camps is 0, 1 or 2. Column 3 includes only areas in which there were three or more camps. Other controls: maternal age, maternal education, family income, a dummy for being Hindu and birth order.

[a] t-statistics in parenthesis, standard errors clustered at the village level

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

Table 8. ***Effect of Vaccination Camps by Gender Preference and Proximity to Other Sources of Vaccination***

	Dependent Variable: Number of Vaccinations Child Has			
	(1)	(2)	(3)	(4)
	Ideal: Equal/More Girls	**Ideal: More Boys**	**Other Source < 5 km**	**Other Source >= 5 km**
Explanatory Variables:				
Girl	$-.1324^{**}$	$-.1039$	$-.09$	$-.1011^{*}$
	(-2.22)	(-1.38)	(-1.33)	(-1.66)
Girl × # Camps	$-.0384$	$-.393^{***}$	$-.1261$	$-.2331^{*}$
	$(-.36)$	(-2.79)	(-1.05)	(-1.9)
Girl × # Camps Sq.	.0087	$.0939^{***}$	.0288	$.0546^{*}$
	(.35)	(2.93)	(1.05)	(1.88)
# Camps	$-.0075$	$.1963^{*}$	.0023	.0995
	$(-.09)$	(1.92)	(.02)	(.95)
# Camps Squared	.0061	$-.0455^{**}$	.0038	$-.0256$
	(.32)	(-1.98)	(.18)	(-1.06)
# Older Brothers	$-.0118$	$-.0359^{**}$	$-.0302^{**}$	$-.0186$
	$(-.89)$	(-2.32)	(-2)	(-1.39)
# Older Sisters	$.0225^{*}$	.0161	$.0301^{**}$	.0158
	(1.63)	(1.02)	(2.03)	(1.1)
Constant	1.687^{***}	$.564^{*}$	1.312^{***}	1.179^{***}
	(7.07)	(1.93)	(4.55)	(4.49)
Number of Obs.	6445	4409	5092	5762
R^2	.29	.22	.28	.29

Notes: An observation is a child aged 6 months to 2 years and the dependant variable is the number of vaccinations the child has received (0-6). # Camps is the number of Family Health and Welfare Camps reported in the village last year. Column 1 includes only women who report wanting equal numbers of boys and girls or more girls; Column 2 includes women who report wanting more boys. Column 3 includes only areas where there is a Primary Health Center, Community Health Center or Government Hospital less than 5 km away. These will be places where camps are likely to be a less important source of vaccination. Column 2 includes only areas where the closes PHC, CHC or Government Hospital is 5 or more km away. Other controls: maternal age, maternal education, family income, a dummy for being Hindu, birth order and a quadratic in child age.

[a] t-statistics in parenthesis, standard errors clustered at the village level

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

Table 9. ***Falsification Test: Placement of Camps***

Dependent Variable:	*Number of Camps, Top-Coded*	*Number of Camps*
Explanatory Variables:		
Gender Difference in Vacc. 1992	.4865	.2036
	(.63)	(.17)
Average Vacc, 1992	.1755	.3707
	(.81)	(1.08)
Ave. Income (durables)	−.2568	−.5061
	(−.69)	(−.86)
Average Mom Educ	.0589	−.0692
	(.52)	(−.39)
Constant	.356	.86
	(.73)	(1.12)
Number of Observations	25	25
R^2	.1	.12

Notes: The table estimates whether there is any evidence for camp placement being based on existing gender differences in vaccination. An observation is a state. Dependent variable in Column 1 is the average number of camps, top-coded at 5; in Column 2, simply the number of camps. Independent variable is the difference in vaccination rates across genders in that state in 1992.

[a] t-statistics in parenthesis

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

Table 10. ***Access to Health Facility and Gender Imbalance in Vaccination***

Dependent Variable: Number of Vaccinations Child Has		
	All	**Excluding Facility in Village**
Explanatory Variables:		
Girl	−.0586	−.0106
	(−1.15)	(−.15)
Girl × Distance	−.0153*	−.0213**
	(−1.8)	(−2.03)
Girl × Dist. Sq.	.0004**	.0005**
	(2.04)	(2.32)
Distance	−.0127*	−.0147*
	(−1.73)	(−1.62)
Dist. Sq.	−.0001	−.0001
	(−.71)	(−.32)
Child Age	.2784	.329
	(1.29)	(1.35)
Child Age Sq.	−.0904*	−.0943
	(−1.64)	(−1.51)
# Older Brothers	−.0241***	−.0184*
	(−2.66)	(−1.84)
# Older Sisters	.0176*	.0142
	(1.94)	(1.39)
constant	2.279***	2.201***
	(10.02)	(8.35)
Number of Observations	14447	11492
R^2	.27	.26

Notes: This table estimates the effect of distance to a primary health center, a community health center or a government hospital on the gender imbalance in vaccination. An observation is a child aged 1 to 4 years and the dependant variable is the number of vaccinations the child has received (0-6). Distance is the minimum distance reported in the village survey to either a Primary Health Center, a Community Health Center or a Government Hospital. Column 1 includes all observations and Column 2 limits to those without one of these health facilities in the village. Other controls: maternal age, maternal education, family income, a dummy for being Hindu and birth order.

[a] t-statistics in parenthesis, standard errors clustered at the village level

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

Table 11. ***Regional Relationship Between Levels and Difference in Vaccination***

	Dependent Variable: Boy Vacc - Girl Vacc	
	(1)	(2)
Explanatory Variables:		
Average Vacc.	−.0294	.135**
	(−1.44)	(2.29)
Average Vacc. Sq.		−.0262***
		(−2.97)
Ave. Mother Educ.	−.013	−.0072***
	(−1.07)	(−.59)
Ave. Income (durables)	.0639	.0616
	(1.53)	(1.48)
Year	−.001	−.0088
	(−.07)	(−.58)
Urban Type	.1012***	.11
	(3.36)	(3.64)
constant	1.962	17.231
	(.07)	(.57)
Number of Observations	3506	3506
R^2	.01	.01

Notes: This table estimates the relationship between average vaccination level and gender difference in vaccination by region in India. An observation is a cluster in the survey. The dependent variable is the vaccination average for boys minus that for girls. The independent variables of interest are the average vaccination level and that variable squared. These are intended to proxy for the cost of these investments.
[a] t-statistics in parenthesis
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 12. ***Changes in Gender Bias in Mortality, 1982-1992***

Dependent Variable: Child Died 18 mons - 5 years

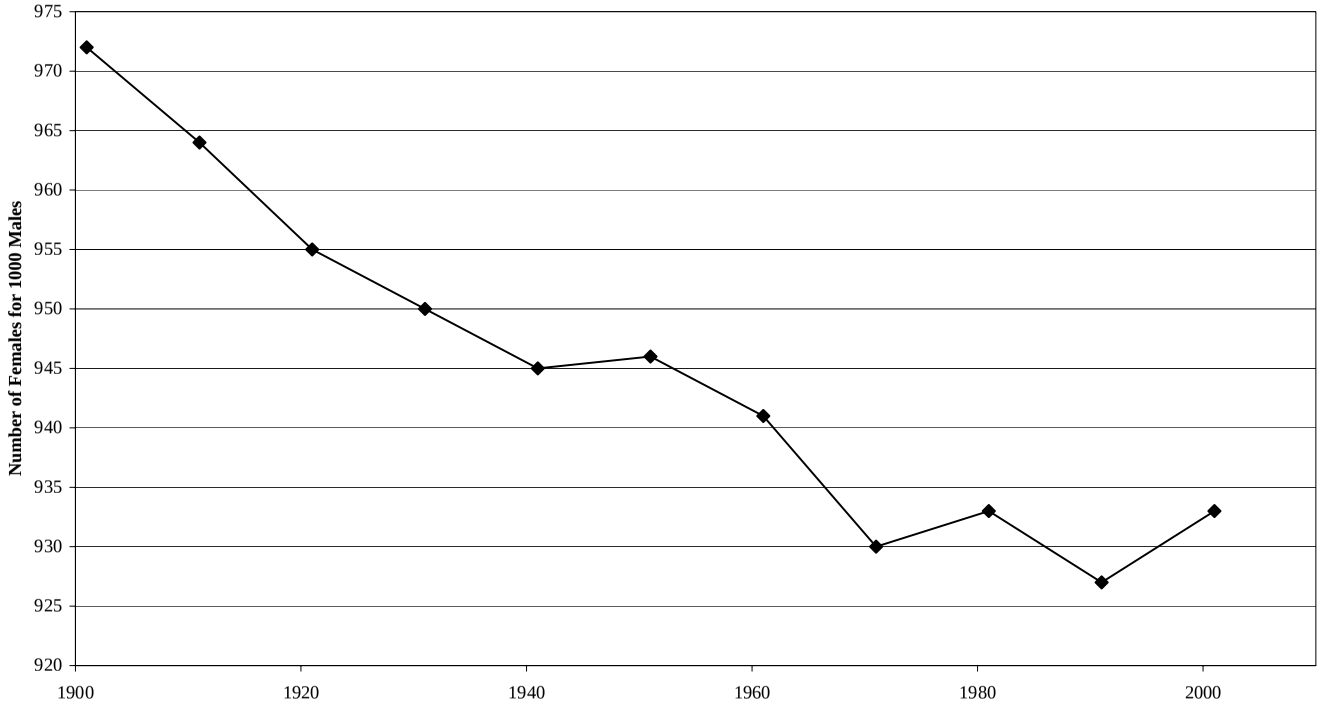
	(1) Low Initial Vaccination	(2) High Initial Vaccination
Explanatory Variables:		
Girl × date of birth	.00068**	−.00088**
	(2.25)	(−2.24)
Date of birth (yr)	−.0004*	−.0001
	(−1.72)	(−.2)
Family Income	−.005***	−.0029***
	(−9.4)	(−4.84)
Mother Age	−.0006***	−.0001
	(−4.15)	(−.57)
Child Age	.0001	−.0006
	(.36)	(−1.31)
Mother Educ.	−.0004***	0
	(−3.12)	(.49)
# Kids in Family	.0062***	.0036***
	(12.8)	(6.03)
Number of Observations	53420	14994

Notes: This table estimates a probit model of the evolution of gender inequality in mortality over the period from 1982 to 1993, using a created panel based on children of different ages. The dependent variable is an indicator for whether the child died between 18 months and 5 years, conditional on having reached 18 months. The regression is limited to children born 5 to 10 years before the survey. Column 1 includes states with initially low vaccination levels, where we would expect increases in gender discrimination over time. Column 2 includes states with initially high vaccination levels where we would expect decreases over time. Other controls include dummies for birth order and dummies for region, interacted with female. The main effect of *girl* is not reported since it is captured in the effect for each region.

[a] t-statistics in parenthesis

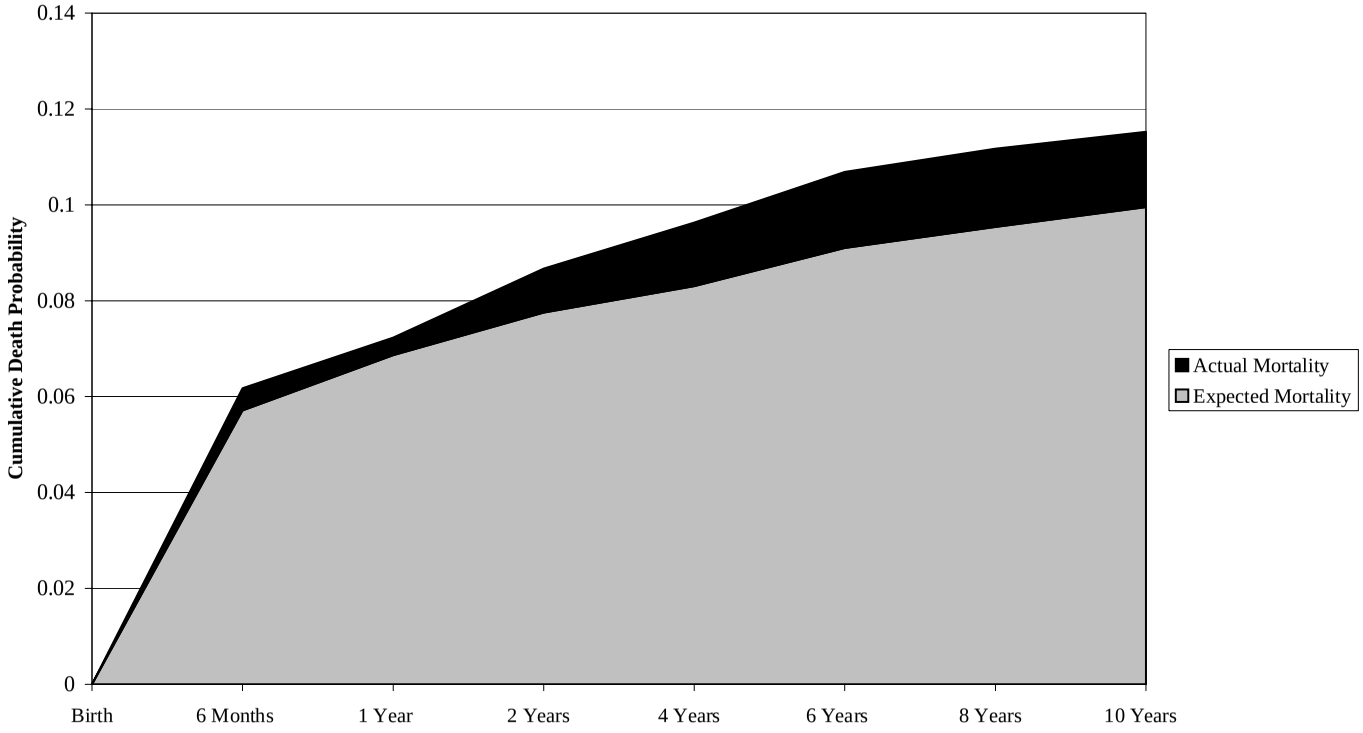
* significant at 10%; ** significant at 5%; *** significant at 1%

Figure 1:
Female to Male Sex Ratio in India, 1901-2001



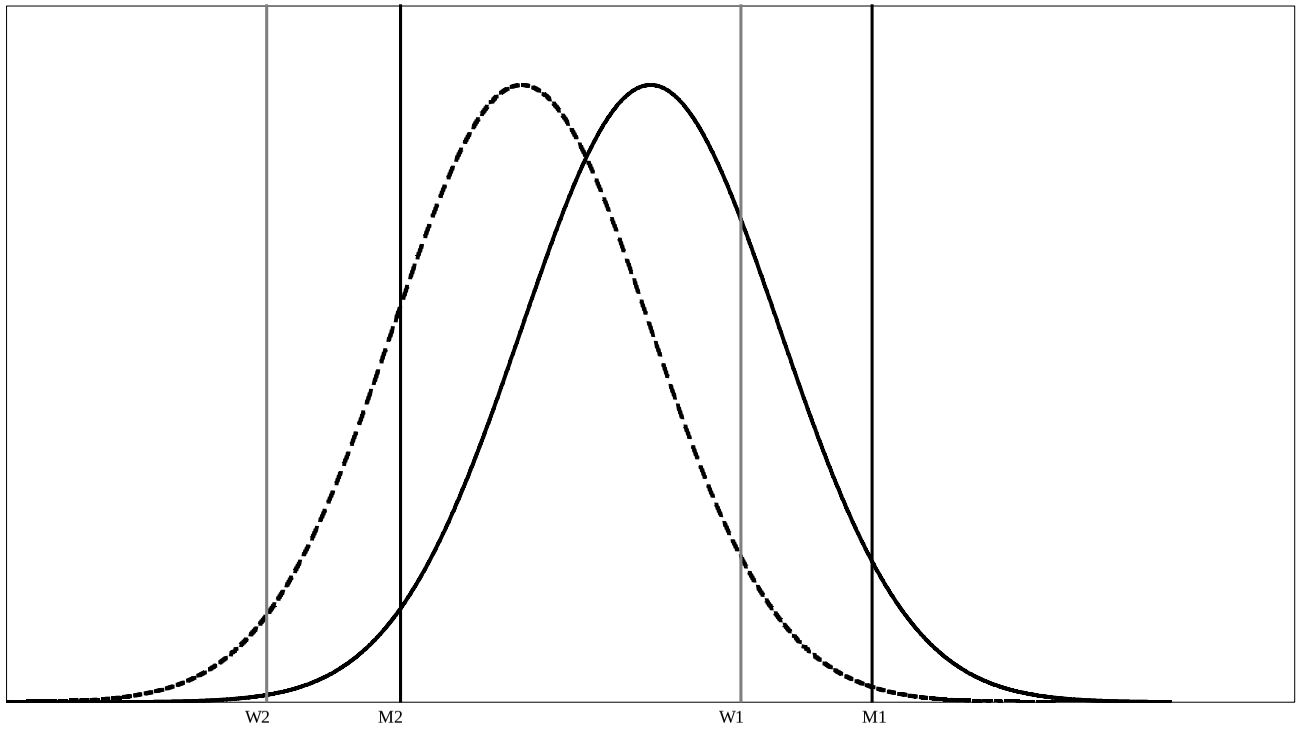
Notes: Sex ratio (# of women per 1000 males) as reported in either the British Census of India or the Indian Census. Sex ratio refers to the ratio in the overall population. This captures a combination of the sex ratio at birth and mortality patterns over the life cycle.

Figure 2:
Actual and Expected Female Mortality in India



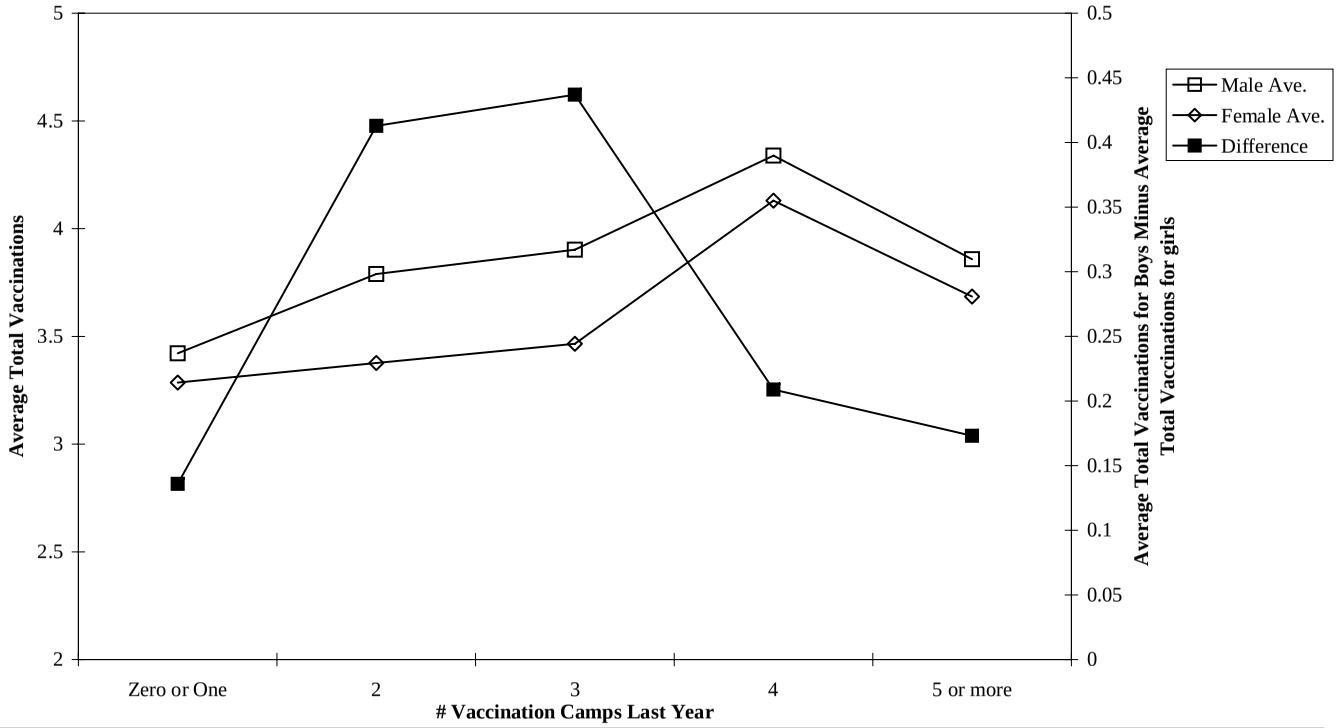
Notes: The sample is limited to children born in the ten years before the survey, and estimates are based on the regressions in Table 2. Expected mortality is the predicted mortality from the regression for girls in India *if* they did not have the negative effect of being a girl in India (i.e. if the value of the value of the interaction term was zero). Actual moratlity is actual predicted mortality.

Figure 3:
Theoretical Framework Vaccination Cutoffs



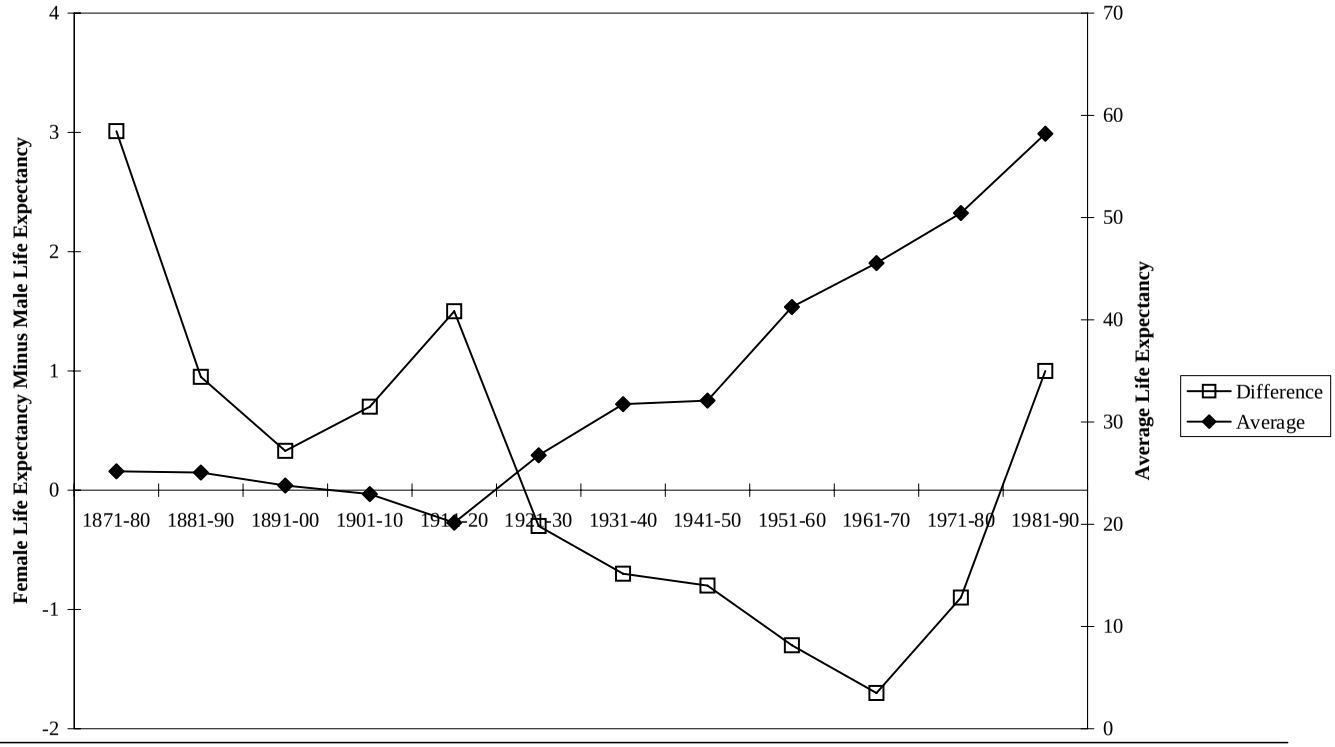
Notes: This figure shows the effect of changing the average vaccination cost (moving the distribution from the solid to the dotted line) on the gender difference in vaccination for a high vaccination environment (W1,M1) and a low vaccination environment (W2,M2). Everyone with costs below the cutoff line vaccinates. In the high vaccination situation, moving the distribution increases female vaccination relative to male. In the low vaccination environment the converse is true.

Figure 4:
Vaccination Camps and Gender Difference in Vaccination



Notes: Figure reports differences in total number of vaccinations by gender and the average number of vaccinations graphed against the reported number of vaccination camps in the village last year. A value of 0.3 on the y-axis means that boys are getting an average of 0.3 more vaccinations than girls (out of a total of 6).

Figure 5:
Difference in Life Expectancy at Birth in India, 1871-1990



Notes: Life expectancy at birth is from the Indian Census (the British Census of India before independence). The average simply represents the straight (not population weighted) average of male and female life expectancy. The difference is female life expectancy minus male life expectancy. In developed Western countries females outlive males by around five years.