

Is Sex Imbalance in Early Age Mortality Driven by Pre-birth Environmental Factors, Child Biology, or Parental Preferences? Evidence from Male-Female Twin Pairs

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I thank Anna Aizer, Kenneth Chay, Oded Galor, Andrew Foster, Emily Oster, Marie Patience Bayab Pongou, Louis Putterman, Yona Rubinstein, Valerie Wilson, and participants at the Applied Micro lunch for useful comments on an earlier version of this paper. I acknowledge the Compton Foundation for generous financial support.

Abstract: Sex differences in early age mortality have been explained by sex differences in biological make-up and sex-selective discrimination in the allocation of household resources. Studies estimating the effects of these factors generally assume that sex is exogenous, which is an implausible assumption in view of a recent evidence that child sex is endogenous to several pre-birth environmental factors which may also determine child health and survival *in utero* and after birth. We propose a methodology that decomposes sex differences in mortality into the effects of pre-birth environmental factors, child biology, and parental preferences. Exploiting variation in sex differences in mortality among twins and within male-female twin pairs, and variation in gender bias in sub-Saharan Africa and India, we show that : (1) pre-birth environmental factors account for a large fraction of the usual excess mortality rates of male children; (2) the biological make-up of male children contributes to this excess mortality only during infancy, but its effect has been previously overstated; (3) parental discrimination against female children in India negatively affects their survival; but failure to adjust for pre-birth and biological effects leads the conventional methodological approach to understate its effect by 160 percent during infancy, and 33 percent during childhood.

Keywords: Sex differences in mortality, pre-birth environment, child biology, sex-selective discrimination.

1 Introduction

This paper investigates the origins of sex imbalance in early age mortality. It has long been observed that girls have a better survival chance than boys (Graunt, 1662). The survival advantage of female children however diminishes and eventually reverses by age five in some countries of South and East Asia. Two theories have been advanced to explain sex imbalance in early age mortality. From a biological point of view, male children have a weaker immune system than female children, which increases their susceptibility to infectious diseases, and lowers their survival chance (Waldron (1983)). From an economic point of view, *rational* and *optimizing* parents may favor children of one gender over the other in the allocation of household resources, because of expected gender wage differentials in the labor market, gender price differentials in human capital investments, the dowry system, or because they simply value one sex over the other given identical outcomes and costs. This theory of sex-selective discrimination in child health investments has been severally validated in countries of South and East Asia like India and China, where parents place a much higher cultural and economic value on sons than daughters, leading to the neglect of the latter and to the reversal of their survival advantage by age five (Kynch and Sen (1983), Sen(1989)).

A common assumption made in all studies testing the biological and the economic theories of sex imbalance in early age mortality is that child sex is random. A recent literature however shows that offspring sex ratios are partly predetermined by parental circumstances and levels of hormones at the time of conception¹. According to James (1996), the likelihood of siring a son is increased by high concentrations of testosterone and estrogen, and the likelihood of siring a daughter is increased by high concentrations of gonadotrophins and progesterone. Levels of parental hormones are in turn related to parental stresses, illnesses and occupations (James (1998)). To the

¹This literature is reviewed in section 2.3.

extent that these parental pre-conceptual factors have a direct or indirect impact on a baby's health and survival chance *in utero* or after birth, the estimates of sex differences in early age mortality produced by the conventional methodological approach adopted in the available biological and economic literature are biased. In this study, we attempt to correct for this bias, and propose a methodology that allows the estimation of the distinct effects of child biology, pre-birth environmental factors², and sex-selective discrimination on sex differences in early age mortality.

Our identification strategy exploits variation in sex differences in mortality in a sample of twins and within male-female twin pairs. We posit that sex difference in mortality rates is the additive effects of male-female differential immune system or biological make-up, pre-birth environmental factors, and parental discrimination. This difference is obtained by estimating a cross-sectional linear probability model (LPM) regression of mortality on sex using the sample of all twins. To adjust for pre-birth environmental factors, we estimate a twins fixed effect LPM regression.³ The estimate thus obtained is the additive effects of differential immune system and parental discrimination, and subtracting this estimate from the cross-sectional LPM estimate yields an estimate of the effect of differential pre-birth environmental factors. To separate out the effects of differential immune system and parental discrimination, we assume that there exist two types of environments, non-discriminatory and discriminatory⁴, and that the effect of male-female differential immune system is the same across the two environments⁵. In a non-discriminatory environment, parents do not discriminate against children of a particular sex in the allocation of parental and household resources, implying that the effect of parental discrimination is zero, which

²By pre-birth environmental factors, we mean factors that are external to a child and that occur before conception or before birth. These factors might be pure environmental factors such as parental exposure to chemicals, or medical factors such as parental illnesses. Note also that despite the fact that these factors occur before birth, they might persist long after birth.

³Note that this rightly controls for the confounding effect of pre-birth environmental factors since twins have the same exposure to these factors.

⁴See Sen (1990b), Sen (1992) and Oster (2006) for a similar assumption with respect to sub-Saharan Africa considered as non-discriminatory, and South and East Asia considered as discriminatory. The plausibility of this assumption is also established by Garenne (2003) for sub-Saharan Africa and numerous studies including for instance Sen (1990), Sen (1992), Basu (1989) and Ebeinstein (2006) for South and East Asia, most notably India and China.

⁵See Oster (2006) for a similar assumption.

in turn implies that in such an environment, the twins FE estimate only measures the effect of differential immune system. In a discriminatory environment however, the twins FE estimate is the additive effects of immune system and parental discrimination, but the effect of parental discrimination is obtained by subtracting from the twins FE estimate the estimate of the effect of child immune system obtained for non-discriminatory environments.⁶ Our methodology therefore allows to estimate the distinct effects of child immunity, pre-birth environmental factors, and sex-selective discrimination on sex differences in mortality.

We implement this methodology using nationally representative household surveys from sub-Saharan African countries and India. There are numerous evidences in the literature that parents do not treat boys and girls asymmetrically in the allocation of parental and household resources in sub-Saharan Africa (Garenne (2003), Sen (1990b), Sen (1992))⁷, while widespread discrimination against female children has been severally documented in India (Sen (1990), Sen (1992), Borooah (2004), Basu (1989), Pande (2003), Coale and Banister (1994)). Figure 1 provides an additional piece of descriptive evidence of discrimination against female children in India as opposed to sub-Saharan Africa. We note that mortality is higher in male children compared to female children in the first year of life in both regions, but while male children continue to die at a higher rate between the first and the fifth birthday in sub-Saharan Africa, the pattern is reversed in India. Despite the fact that this figure shows evidence of discrimination against girls in India, it should be noted that the extent of such discrimination has hardly been quantified. By not accounting for the effects of male-female differential immune system and pre-birth environmental factors, previous studies have produced a biased estimate of the mortality effect of discrimination in India and other Southern and Eastern Asian countries. The descriptive illustration provided by figure 1 for instance has led many scholars to wrongly conclude that parental discrimination against female children in India is negligible during infancy, and has damaging effects on girls only in the period between the first

⁶Note that by assumption, the effect of male-female differential immune system is the same for non-discriminatory and discriminatory environments.

⁷Also see Deaton (2001) for evidence of the symmetrical treatment of boys and girls in Côte d'Ivoire.

and the fifth birthday (see e.g., Sen (1999), Osmani and Sen (2003)). In a recent study, Oster (2006) tries to control for the effect of male-female differential biological make-up by assuming this effect to be the same in sub-Saharan Africa and India, and then estimates the effect of discrimination in India net of the effect of biology, but she finds no effect of parental discrimination against girls in the first six months of life.⁸ While the methodological approach adopted in Oster (2006) is an improvement over the approach traditionally used in studies of gender bias, it is still the case that Oster (2006), as all other studies, treats child sex as exogenous, and therefore does not account for the confounding effect of pre-birth environmental factors. That these studies have found the effect of parental discrimination against girls to be negligible in early life is therefore not surprising, but such findings are obviously inconsistent with the long documented existence of widespread phenomena such as female infanticide, neglect and abandonment that take place in very early stages of life in most countries of South and East Asia (see e.g., Sudha and Rajan (1999), Coale and Banister (1994)). These findings are also hardly defensible because they imply a non-plausible discontinuity in parental preferences.⁹ Implementing the methodology described in the previous paragraph allows us to estimate the distinct effects of child immunity, pre-birth environmental factors, and sex-selective discrimination on sex differences in mortality in early ages in sub-Saharan Africa and India. Our findings, which we preview in the following paragraphs, particularly show that the effects of discrimination against girls in India have been grossly underestimated in previous studies.

The estimation of the cross-sectional LPM regression shows that infant mortality is respectively 45 and 27 per thousand points higher in male children compared to female children in sub-Saharan Africa and India. Estimating the twins FE regression shows that infant mortality is 27 per thousand points higher in males in sub-Saharan Africa, and is 10 per thousand point lower in this sex in India, but the estimate for India

⁸Oster (2006) pools data from India and selected sub-Saharan African countries, and estimate a linear probability model regression of mortality on gender, while controlling for a dummy indicator for India as well as an interaction term between gender and the India dummy, but the coefficient on the interaction term is not statistically different from zero, leading her to conclude that the effect of parental discrimination against females in the first six months of life is negligible.

⁹In fact, if there is no discrimination against female children in very early ages, this would imply that discrimination that starts in the prenatal period in the form of sex-selective abortion, stops right after birth for a few months, then arises again afterwards, which is very hard to understand.

is not statistically significant at the 10 percent level. Applying our methodology for the decomposition of sex differences in mortality implies that pre-birth environmental factors raise the infant mortality rate of boys by 18 and 37 per thousand points in sub-Saharan Africa and India, respectively; the immune system of boys contributes 27 per thousand points to their excess mortality in both regions; and parental discrimination against girls in India increases their mortality rate by 37 per thousand points. We replicate this analysis in the neonatal period (period covering the first month of life after birth) and the postneonatal period (period between the first month and the first birthday), to take into account the fact that factors contributing to male-female differential mortality rates might differ across ages. Our conclusions are qualitatively the same with respect to the decomposition of the sex gap. These conclusions particularly challenge previous studies that have been unable to detect any effect of parental discrimination against girls in early ages in India, especially during the neonatal period.

Finally, we replicate this exercise in the childhood period, which is the period between the first and the fifth birthday. The estimation of the cross-sectional LPM regression shows that child mortality is 4 per thousand points higher in boys compared to girls in sub-Saharan Africa, but is 16 per thousand lower for boys in India. Estimation of twins FE regression shows that child mortality is respectively 8 and 31 per thousand points lower among boys than girls in sub-Saharan Africa and India. These results imply that pre-birth environmental factors raise the mortality rate of boys by 12 and 15 per thousand points in sub-Saharan Africa and India, respectively, while the biological make-up of boys lowers their mortality rate by 8 per thousand points in both regions. Parental discrimination against girls in India increases their mortality rate by 23 per thousand points.

We extend our analysis to singletons. We note that the sex ratios of same-sex twins is similar to that of singletons in sub-Saharan Africa and India, implying that pre-birth environmental factors that partly determine offspring sex ratios are similar for single births and multiple births. We therefore assume that the contribution of these factors to sex differences in mortality is the same (in relative terms) for singletons and twins, which allows us to estimate the distinct effects of child immunity, pre-birth

environmental factors, and sex-selective discrimination on sex differences in mortality among singletons. Our results are qualitatively similar to those obtained for twins.

The findings of this study lead to at least three important conclusions: (1) unobserved pre-birth environmental factors account for a large fraction of the higher mortality rates of male children observed in most populations; (2) the biological make-up of male children contributes to their excess mortality rates during infancy only, but its effect has been previously overestimated by about 50 percent due to failure to account for pre-birth effects; but contrary to the long-held biological theory of sex gap in morbidity and mortality, male biological make-up favors male survival in the childhood period; (3) and parental discrimination against girls in India increases their mortality rates; however usual estimates of sex differences in mortality grossly underestimate its effects by about 160 percent during infancy and 33 percent during childhood, failing to detect any effect of parental discrimination in early ages.

The remainder of this paper is organized as follows. Section 2 provides some background on the determinants of sex imbalance in early age mortality, summarizes the literature on the pre-birth environmental determinants of child sex, and discusses the potential confounding effect of these factors in the traditional approach to estimating sex differences in mortality. Section 3 presents the methodology and the empirical strategy respectively developed and adopted in our study. Data are described in Section 4, and results follow in Section 5. The extension of our analysis to singletons follows in Section 6, and Section 7 summarizes the key findings and concludes our study.

2 Background on the determinants of sex imbalance in early age mortality

2.1 Child biology

The explanation for the excess mortality of male children partly relies on the chromosomal XY sex-determination system discovered by Stevens (1905) and Wilson

(1905).¹⁰ Males have XY chromosomes and females have XX chromosomes. Waldron (1983) explains that XY chromosomes are more susceptible to X-linked recessive disorders, implying that male children are less likely to be healthy than their female counterparts.¹¹ Studies based on experimental animal models also show that sex hormones have physiological and pathological effects on the immune system (Ansar Ahmed et al. (1985)). Male hormones seem to inhibit T and B lymphocyte maturation, two major components of the immune system (Ansar-Ahmed and Talal (1990)). Females therefore have a more active and stronger immune response than males (Ansar Ahmed et al. (1985), Chao (1996), Bouman et al. (2005)).

This biological literature implies that in a world in which male and female children are treated equitably, male children should suffer a higher incidence of infectious and non-infectious diseases resulting in a lower survival rate. This is indeed the pattern observed worldwide, especially in regions where parents do not discriminate against children of a particular sex in the allocation of household resources. However, whether the excess mortality of male children is solely attributable to their sex chromosomes or their weaker immune system still requires further investigation. In fact, while females have a stronger immune system, they also suffer a higher incidence of autoimmune diseases compared to males (Ansar Ahmed et al. (1985), Chao (1996), Bouman et al. (2005)). Analyzing national data from the World Health Organization, Garenne (1992) finds that mortality from measles is higher for females compared to males. This study is consistent with Preston (1976) who finds an excess female mortality from certain diseases including for instances tuberculosis at age 5-29, influenza-pneumonia-bronchitis at age 5-14, and certain infectious and parasitic diseases at age 1-14. These findings seem to imply that the biological explanation of excess male mortality is inconclusive.

2.2 Parental gender bias

Parental gender bias in the allocation of household resources has been documented in a large body of literature, especially in South and East Asia. Studies show that

¹⁰Also see Wilson (1909).

¹¹Also see Waldron (1985, 1998).

boys are favored over girls in the allocation of food and health care (Alderman and Gertler (1997), Basu (1992), Basu (1989), Behrman (1988), Chen, Huq and D’Souza (1981), Borooah (2004), Hazarika (2000), Pande (2003), Sen (1984), Sen and Sengupta (1983), Croll (2001), Preston and Weed (1976)). This discriminatory treatment of female children translates into excess female mortality, exacerbating the “missing women” problem noted in these countries (Sen (1990b), Sen (1992), Coale and Banister (1994)).

Several economic factors including female labor-market participation and education have been shown to explain excess female mortality in South and East Asia. Higher parental investments in sons relative to daughters in India respond to differential labor-market returns by sex (Rosenzweig and Schultz (1982), Sen (1990a)). Consistent with this finding, other studies have shown that female labor market participation, higher female income and education decrease the relative mortality of girls (Rose (1999), Qian (2005), World Bank (2001), Drèze and Sen (1996)). While some of these studies suggest that excess female mortality due to lower investments in female children only result from parental optimizing behavior, Behrman (1988) shows that parents have pro-male preferences in the allocation of nutrients that do not just reflect differential expected labor market returns by sex. This study suggests that non-economic factors also explain the discriminatory treatment of girls.

2.3 Pre-birth environment

According to a recent literature, pre-conceptual and pre-birth environmental factors¹² determine offspring sex ratios and affect child health *in utero* and after birth (see e.g., James (1996), Garry et al. (2002)). But studies testing the biological and the economic theories of sex differences in morbidity and mortality generally assume that sex is exogenous. This is an implausible assumption in view of new evidences showing that parental circumstances at the time of conception may increase or decrease the likelihood of conceiving a child of a particular sex. James (1996) hypothesizes that the likelihood of having a son is increased by high concentrations of testosterone and

¹²By pre-conceptual and pre-birth environmental factors, we mean factors that are external to a child and that occur before conception or before birth.

estrogen, while the likelihood of having a daughter is increased by high concentrations of gonadotrophins and progesterone. Levels of parental hormones are in turn related to parental stresses, illnesses and occupations (James (1995)). About the impact of illnesses and occupations on offspring sex ratio, James (1998) writes:

“I have cited evidence that male patients with multiple sclerosis, non-Hodgkins’s lymphoma and prostatic cancer, respectively, sire excesses of daughters, daughters and sons; that men engaged in professional driving, professional diving and carbon-setting produce excesses of daughters; that men of high status produces excesses of sons; that prostatectomy of male rodents is associated with their subsequently producing excesses of sons; and that men treated with gonadotrophin or methyltestosterone both produce excesses of sons (James 1996). It is also reported that excesses of daughters are produced by men exposed to the nematocide DBCP, dioxin, borates, vinclozolin, and high voltage installations.”

Further evidence from Seveso, Italy, shows that high parental concentrations of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), one of the most toxic man-made substances, increase the likelihood of siring a female child (Mocarelli et al. (1996), Mocarelli et al. (2000)). Following an explosion at a plant that manufactured the herbicide 2,4,5-trichlorophenol (TCP) near Seveso in 1976, more than 30 kilograms of dioxin were released into the environment. Mocarelli et al. (2000) show that higher serum TCDD concentrations in fathers were associated with a decrease in the proportion of male births in the period 1977-1996. The probability of a male birth was significantly lower for exposed fathers compared to non-exposed fathers (odd ratio 0.555 [95% CI 0.35–0.88]), but TCDD concentrations in serum samples from mothers were not a significant predictor of the probability of having a male birth (Mocarelli et al. (2000)). This study supports the hypothesis that dioxin has a permanent effect on the human epididymus from the time of exposure. This study is also consistent with findings from several other studies on the effect of parental exposure to environmental toxicants such as trichlorophenolate, the nematocide dibromochloropropane and other pesticides, borates, alcohol, lead, solvents, waste anesthetic gases and air pollution from incinerators on the sex ratios of their offspring (see, e.g., Dimid-Ward et al. (1996), James (1997), Williams et al. (1992)). Garry et al. (2002) also found

a higher exposure to fungicide among residents of the Red River Valley in Minnesota to be associated with a lower sex ratio at birth. In most industrialized countries, the proportion of male births has significantly declined in the past decades, and this has been attributed to increased exposure to toxic chemicals (Davis et al. (1998)). According to James (2001), these adverse chemical, occupational and environmental paternal exposures lower offspring sex ratio by causing the ratios of testosterone to gonadotropin in men to be low or/and by disrupting the endocrine system.¹³

The effects of parental medical conditions on offspring sex ratios are also documented. Hesser et al. (1975) and Drew et al. (1986) show that parents who are carriers of the Hepatitis B virus sire excesses of boys, due to a high probability of female fetuses miscarriages.¹⁴ Multiple sclerosis has been found to be associated with higher offspring sex ratios in female patients and lower offspring sex ratios in male patients (James (1994)).

Parental environmental, medical and occupational circumstances that determine offspring sex ratios also affect the health of these children *in utero* and after birth. Mocarrelli et al. (1986) show that children who were exposed to 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) released following the environmental accident that occurred near Seveso showed alterations in their Y-glutamyltransferase and alanine aminotransferase activity compared to the control group. Garry et al. (1996) and Garry et al. (2002) found the prevalence of birth defects (circulatory/respiratory, urogenital, and musculoskeletal/integumental) to be higher among children born to male pesticide applicators in the Red River Valley, Minnesota, USA, with male children suffering a higher burden. But among children born to parents who were exposed to fungicide, female children were more likely to suffer a birth defect. Parental circumstances might also affect children by affecting household income. Most occupations that expose parents to adverse environmental conditions are likely to have a negative effect on their health, reducing their productivity and consequently their income, with negative consequences on children's health and survival.

That parental environmental, medical and occupational circumstances determine

¹³Also see Moller (1998) and Jacobsen et al. (2000) on the sex ratios of patients who suffer endocrine diseases.

¹⁴Also see Oster (2005) for cross-country evidence.

offspring sex ratios at birth and affect the health and survival of these children *in utero* and after birth simply implies that sex is clearly not an exogenous variable. This complicates the interpretation of results found in studies that test the biological and the economic theories of sex differences in morbidity and mortality. The fact that in standard household surveys conducted in developed and developing countries, data on these pre-birth factors are generally not collected poses further challenge in addressing this issue. The methodological approach adopted in the current study, which mainly consists of comparing the mortality rates of male children and female children within male-female twin pairs, corrects for this bias, and yields findings that show that the contribution of the biological make-up of male children to their excess mortality is generally overstated, due to failure to account for pre-birth environmental effects. These latter effects indeed contribute a large fraction of excess male mortality, which seems to imply that pre-conceptual parental circumstances which cause excess male births in almost all populations might also contribute to their excess mortality. We also find that failing to account for the effect of biology and environment results in an underestimation of the effect of parental discrimination against females in societies where such practices take place. In India particularly, studies have failed to find any effect of discrimination against female children in the neonatal period, but we show that discrimination has a negative effect on their survival during this period.

3 Econometric model

3.1 Estimating sex differences in mortality

The sex gap in mortality is usually estimated using the following specification.

$$M_{iht} = \theta Male_i + X_{ht}\pi + \epsilon_{iht} \quad (1)$$

where M_{iht} is a dummy variable indicating whether child i , born to parents h died at time t (M_{iht} takes on value 1 if i died at time t and 0 if not); $Male_i$ is a dummy indicator for whether child i is male; X_{ht} is a vector of observed parental and

household characteristics thought to be correlated with sex and mortality¹⁵; and ϵ_{iht} is an error term usually assumed to be uncorrelated with sex. The parameter of interest θ , which measures male-female difference in mortality rates, is generally interpreted as the effect of inherent biological differences between boys and girls, or/and the effect of parental discrimination against females. Its interpretation however is very ambiguous in the literature. When θ is positive, meaning that the probability of dying is greater for boys compared to girls, this is generally interpreted as the effect of the weak immune system of boys, and when θ is negative, this is interpreted as the effect of parental discrimination against girls. As discussed in the introduction, the observation that infant mortality is higher for boys than girls in both sub-Saharan Africa and India (Figure 1) has led many studies to conclude that parental discrimination has a negligible effect on girls mortality during infancy in India. This conclusion stems from the fact that sex is treated as exogenous in those studies.

The assumption made in most studies that ϵ_{iht} is uncorrelated with child sex is not plausible in view of the evidences provided in Section 2.3, that child sex is partly determined by pre-birth factors that might also affect child health and survival, implying that sex is an endogenous variable. Any estimate of θ that does not address this issue of endogeneity is therefore likely to be biased, although the direction of the bias is difficult to pin down.¹⁶ Our goal in this study is to correct for this bias, and decompose θ into the effects of pre-birth environmental factors, child biology and parental preferences.

Write $\epsilon_{iht} = u_h + v_{ht} + w_{iht}$ where u_h , v_{ht} and w_{iht} are respectively parental time-invariant unobservables, parental time-variant unobservables, and a child random unobserved shock (not correlated with sex). u_h and v_{ht} are interpreted as parental

¹⁵Note however that since sex has been traditionally treated as exogenous, controlling for the vector X_{ht} is irrelevant in most studies.

¹⁶It is possible for instance that factors such as prostate cancer or hepatitis B that cause fathers to sire excess of boys also contribute to kill them once born, through lowering the economic status of their parents for instance, then resulting in higher mortality rates in this sex. If this is the case, then the share of excess male mortality generally attributed to male biological make-up is exaggerated. But if boys are also more likely to be born to high socioeconomic status parents (see James (1998)), which is also a contributing factor to child survival, then the share of excess male mortality attributed to biology is underestimated. Also note that failure to take into account the effect of pre-birth factors and child biology will result in an underestimate of the effect of discrimination in discriminatory settings.

pre-birth circumstances and gender bias. We can explicitly write v_{ht} as the sum of time-variant parental pre-birth circumstances (p_{ht})¹⁷ and parental bias (b_{ht}); that is, $v_{ht} = p_{ht} + b_{ht}$. They are correlated with child sex and mortality. We posit that a cross-sectional LPM estimate of θ is the additive effects of child biology (or immune system), pre-birth factors, and parental preferences.¹⁸ The effect of parental time-invariant unobservables can be netted out by comparing the mortality of male-female siblings (children born to the same parents). This is done by estimating within siblings fixed effect regression as follows.

Let (i, j) be a pair of siblings. Re-writing Equation (1) for i and j yields respectively Equations (2) and (2)' below.

$$M_{iht} = \theta_{SFE} Male_i + X_{ht}\pi + u_h + p_{ht} + b_{ht} + w_{iht} \quad (2)$$

$$M_{jht'} = \theta_{SFE} Male_j + X_{ht'}\pi + u_h + p_{ht'} + b_{ht'} + w_{jht'} \quad (2)'$$

Taking (2)-(2)' yields:

$$M_{iht} - M_{jht} = \theta_{SFE}(Male_i - Male_j) + (X_{ht} - X_{ht'})\pi + p_{ht} - p_{ht'} + b_{ht} - b_{ht'} + w_{iht} - w_{jht'} \quad (3)$$

Estimating Equation (3) using within siblings fixed effect regression yields an estimate of θ_{SFE} . Note that θ_{SFE} still includes the effect of parental time-variant factors as long as $p_{ht} - p_{ht'}$ for instance is correlated with child sex. Parental environmental and health circumstances for instance are likely to vary over time, making such a correlation very likely. To net out the effect of pre-birth factors, we compare a male twin with his female counterpart, by estimating a male-female twins fixed effect regression. Let $(i, -i)$ be a pair of male-female twins. Equation (1) can be re-written for each of them as follows.

$$M_{iht} = Male_i \theta_{TFE} + X_{ht}\pi + u_h + p_{ht} + b_{ht} + w_{iht} \quad (4)$$

$$M_{-iht'} = Male_{-i} \theta_{TFE} + X_{ht'}\pi + u_h + p_{ht'} + b_{ht'} + w_{-iht'} \quad (4)'$$

Since pre-birth factors are the same for a pair of twins (that is, $p_{ht} = p_{ht'}$), taking (4)-(4)' yields:

¹⁷Parental pre-birth circumstances determining offspring sex ratios such as environmental conditions (e.g., dioxin exposure) or occupation might vary over.

¹⁸Note that if u_h and v_{ht} were observed and controlled in equation (1), θ would only measure the effect of male biological make-up.

$$M_{iht} - M_{-iht} = \theta_{TFE}(Male_i - Male_{-i}) + b_{ht} - b_{ht'} + w_{iht} - w_{-iht'} \quad (5)$$

$b_{ht} - b_{ht'}$ is still correlated with $Male_i - Male_{-i}$, implying that estimating equation (5) using within male-female twins fixed effect regression yields as estimate of θ_{TFE} that is the additive effects of child biology and parental bias. Also note that subtracting θ_{TFE} from the cross-sectional LPM estimate of θ yields an estimate of the effect of pre-birth factors. It is also important to notice that in environments where parents do not discriminate against any specific sex in the allocation of household resources (that is, $b_{ht} - b_{ht'} = 0$), θ_{TFE} measures the effect of male biology since $w_{iht} - w_{-iht'}$ is uncorrelated with $Male_i - Male_{-i}$ by assumption.

We first estimate θ and θ_{TFE} using the same sample of twins, but θ_{TFE} is also estimated based only on the restricted sample of male-female twin pairs. Since the sample of mothers with at least two twin pairs such that at least one pair is a pair of opposite sex twins is generally very small, we do not estimate θ_{SFE} using the sample of twins. Estimating θ and θ_{TFE} allows us to separate out the effects of pre-birth factors, child biology and parental preferences (see Section 3.2) in the population of twins.

3.2 Decomposing sex differences in mortality into the effects of pre-birth factors, child biology and parental preferences

We posit that θ estimated from equation (1) is the additive effects of pre-birth factors, child biology and parental preferences. That is,

$$\theta = \theta_1 + \theta_2 + \theta_3 \quad (6)$$

where θ_1 is the effect of pre-birth factors, θ_2 the effect of child biology, and θ_3 the effect of parental preferences.

We assume that parental sex-selective discrimination varies from one environment to another. More precisely, we assume two types of environments, non-discriminatory (ND) and discriminatory (D). The effect of parental discrimination in a non-discriminatory

environment is zero by definition.¹⁹ We also assume that the effect of male immune system on sex differences in mortality is the same in discriminatory and non-discriminatory environments.²⁰ Both assumptions can be formally expressed as follows.

$$\begin{cases} \theta_3^{ND} = 0 \\ \theta_2^D = \theta_2^{ND} \end{cases} \quad (7)$$

Plugging the first and the second equation of System (7) into Equation (6) for non-discriminatory and discriminatory environments, respectively, and re-writting Equation (6) for each of these environments yields:

$$\begin{cases} \theta^{ND} = \theta_1^{ND} + \theta_2^{ND} \\ \theta^D = \theta_1^D + \theta_2^D + \theta_3^D \end{cases} \quad (8)$$

The question now is how do we separate θ_1^{ND} and θ_2^{ND} on the one hand, and θ_1^D , θ_2^D and θ_3^D on the other hand. Estimating Equation (1) yields θ^{ND} and θ^D for non-discriminatory and discriminatory environments, respectively. θ_{TFE} , obtained from estimating a male-female twin fixed effect regression (Equation (5)) is solely the additive effects of biology and parental preferences since twins are exposed to the same pre-birth factors; and since the effect of parental preferences is zero in non-discriminatory environments, θ_{TFE} in these environments solely measures the effect of biology. That is,

$$\begin{cases} \theta_{TFE}^{ND} = \theta_2^{ND} \\ \theta_{TFE}^D = \theta_2^D + \theta_3^D \end{cases} \quad (9)$$

Plugging the first and second equation of System (9) into the first and second equation of System (8), respectively, and re-arranging allows to extract the effect of pre-birth factors in non-discriminatory and discriminatory environments, respectively, as follows:

$$\begin{cases} \theta_1^{ND} = \theta^{ND} - \theta_{TFE}^{ND} \\ \theta_1^D = \theta^D - \theta_{TFE}^D \end{cases} \quad (10)$$

¹⁹See section 3.3.2 for a justification of this assumption.

²⁰Given that biology might interact with the disease environment in determining mortality, this assumption might not be valid if discriminatory and non-discriminatory settings have different diseases environments. In our study, we choose sub-Saharan Africa as a non-discriminatory setting and India as a discriminatory setting (also see Oster (2006) for a similar choice). It has also been shown that in sub-Sahara Africa and South and East Asia, children suffer and/or die from similar diseases (Black et al. (2003)), making our assumption plausible.

We have completely separated out the effects of differential pre-birth factors and biology in non-discriminatory environments. For discriminatory environments, it remains to separate out the effects of child biology and parental preferences. We first note that the effect of biology is assumed to be the same across non-discriminatory and discriminatory environments; that is, $\theta_2^D = \theta_2^{ND} = \theta_{TFE}^{ND}$. Plugging this latter equation into the second equation of System (9) and re-arranging yields the effect of parental preferences:

$$\theta_3^D = \theta_{TFE}^D - \theta_{TFE}^{ND} \quad (11)$$

System (12) below summarizes the results obtained from System (7) through Equation (11).

$$\left\{ \begin{array}{l} \theta_1^{ND} = \theta^{ND} - \theta_{TFE}^{ND} \\ \theta_2^{ND} = \theta_{TFE}^{ND} \\ \theta_3^{ND} = 0 \\ \theta_1^D = \theta^D - \theta_{TFE}^D \\ \theta_2^D = \theta_{TFE}^{ND} \\ \theta_3^D = \theta_{TFE}^D - \theta_{TFE}^{ND} \end{array} \right. \quad (12)$$

System (12) completely separates out the roles of pre-birth factors, child biology and parental preferences in determining sex differences in child mortality in discriminatory and non-discriminatory environments.

3.3 Empirical strategy

3.3.1 Estimating the effects of pre-birth factors, child biology and parental preferences across ages

It is possible that the effects of pre-birth factors, child biology and parental preferences on sex differences in mortality varies with age. Understanding and measuring the contribution of these factors to the determination of the sex gap in mortality are likely to improve policies aimed at closing this gap. In this study, we examine sex differences in mortality among children under five years of age. We distinguish between the infancy (I) period and the childhood period (CH). The infancy period is the period

between birth and the first birthday. Infant mortality is therefore measured as the probability of dying during this period conditional on being born alive. The infancy period is further divided into the neonatal (NN) period (that is, the period between 0 and 28 days or 1 month after birth) and the postneonatal (PNN) period (that is, the period between 1 month and 12 months after birth). Neonatal mortality is measured as the probability of dying during the neonatal period conditional on being born alive, and postneonatal mortality is the probability of dying during the postneonatal period conditional on surviving the neonatal period. In most developing countries, neonatal mortality accounts for a large fraction of under five mortality (Black et al. (2003)), making it important to distinguish the neonatal period from subsequent periods in the study of the contribution of pre-birth factors, child biology and parental preferences to sex differences in mortality. The childhood period is the period between the first and the fifth birthday. As for infant mortality, childhood mortality is measured as the probability of dying during this period conditional on surviving the infancy period.

We estimate Equations (1) and (5) for each of the periods previously defined. When estimating the twins fixed effect regression (Equation (5)) during the postneonatal period, we consider only twin pairs who survived the neonatal period. Similar, when estimating the twins fixed effect regression during the childhood period, we consider only twin pairs who survived postneonatal period. We therefore drop from the sample surviving twins whose counterparts did not survive the previous period. Our decomposition of the sex gap leads to the derivation of parameters in Equation (12) for each period (P) as follows.

$$\left\{ \begin{array}{l} \theta_{1,P}^{ND} = \theta_P^{ND} - \theta_{TFE,P}^{ND} \\ \theta_{2,P}^{ND} = \theta_{TFE,P}^{ND} \\ \theta_{3,P}^{ND} = 0 \\ \theta_{1,P}^D = \theta_P^D - \theta_{TFE,P}^D \\ \theta_{2,P}^D = \theta_{TFE,P}^{ND} \\ \theta_{3,P}^D = \theta_{TFE,P}^D - \theta_{TFE,P}^{ND} \end{array} \right. \quad \text{with } P = I, NN, PNN, \text{ or } CH \quad (13)$$

3.3.2 Choice of discriminatory and non-discriminatory environments

Our choice of discriminatory and non-discriminatory environments is based on studies conducted in different regions of the world. Most Southern and eastern Asian countries are known as countries where parents have strong preferences for male children, therefore discriminating against female children in the allocation of foods and health care (See section 2.2). On the contrary, sub-Saharan Africa is known as a region where sex-selective parental discrimination is almost non-existent. While most African societies are patriarchal, studies have been unable to detect discrimination against children of a particular sex in these societies. Based on household data from sub-Saharan African countries, Garenne (2003) finds that the probability of dying before the fifth birthday is higher for boys compared to girls, but investment in health care such as immunization does not significantly differ between the two sexes. The findings of this study supports the assumption that sub-Saharan Africa is non-discriminatory, as also recognized by other scholars (Sen (1990b), Sen (1992), Oster (2006)). Further evidence for the symmetrical treatment of boys and girls in a sub-Saharan African country is provided by Deaton (2001). Using household expenditure data from Côte d'Ivoire, Deaton (2001) finds no gender bias in the allocation of goods, while finding a statistically insignificant pro-male bias in Thailand.

For our analysis, we use data from India, considered as a discriminatory environment, and from sub-Saharan African countries, considered as non-discriminatory. Sub-Saharan African countries are chosen solely based on the availability of data, and are listed in Table A-1 in appendix.

4 Data

4.1 Data sources

We use Demographic and Health Surveys data collected in thirty sub-Saharan African countries, and two National Family Health Surveys conducted in India. Information on years of surveys is provided in Table A-1 in appendix. The DHS and the NFHS surveys are conducted by the same organization (Measure DHS), and are standardized

and comparable across countries and years for most variables. In each survey, a two-stage probabilistic sampling technique is used to select clusters or census enumeration zones at the first stage, and household at the second stage. In each household, data are collected on household characteristics including household durable assets and facilities (e.g., car, TV, radio, access to clean water, toilet facilities, etc.). Information on the demographic and socioeconomic characteristics of each household member is also collected. Selected women in the household provide complete information on their fertility history. In particular, information on each live birth is collected, including date of birth, whether the birth is a singleton or a multiple birth, whether the person is still alive or not, and when the person died if dead. In this study, we use the file of all live births reported by mothers in each survey. The number of these files is 75 for sub-Saharan Africa, and two for India. Sub-Saharan African countries' files are merged and analysed as a single file, and so are the two files from India. The total sample size of all live births is 1,670,477 for sub-Saharan Africa, and 543,981 for India. Detailed information on the sample size of all births for each country and survey year can be found in Table A.1 in Appendix.

4.2 Data descriptions: Comparing twins and singletons

Comparing twins and singletons along common demographic and socioeconomic variables shows the extent to which results obtained from analysing twins samples are generalizable to the entire population.

4.2.1 Sex ratios

Information on whether a birth was single or multiple is provided in each survey. We identified and matched twins based on: (1) whether they were declared as twins by their mothers, (2) their mother's identification number, and (3) their month and year of birth. Triplets and quadruplets are dropped from the sample. Table 1 shows that the sample size of twins is 50,994 for sub-Saharan Africa and 6,920 for India. They represent respectively 3.05 percent and 1.27 percent of the sample of all live births in these settings. The proportion of twins for sub-Saharan Africa is comparable

to that found in the United States by Almond, Chay and Lee (2005). However this proportion seems inexplicably low for India. In sub-Saharan Africa, male-male, female-female, and male-female twins represent respectively 31%, 30% and 39% of all twins. In India, these figures are respectively 35%, 33% and 32%. The moderately high proportion of male-female twins in sub-Sahara Africa may be explained by high levels of gonadotropin among black women, which is thought to increase dizygotic twinning rates (Milham (1964), James (1984)), and perhaps the proportion of male-female twin pairs.

We note that the proportion of male births is respectively 0.508 and 0.504 for singletons and twins in sub-Saharan Africa, and respectively 0.520 and 0.514 in India. This indicates a slightly lower proportion of male among twins in both settings. However the relevant comparison of sex ratios should be between singletons and same sex twins. The proportion of males among same sex twins is 0.506 and 0.521 in sub-Saharan Africa and India, respectively, figures that are similar to the proportion of males among singletons in these regions (0.508 and 0.520, respectively). This shows that male-female relative differences in fetal death are similar for twins and singletons, and so are the pre-birth environmental factors that determine child sex.

For the pooled sample of twins and singletons, these figures imply a sex ratio at birth (the ratio of males to females at birth) of 1.032 in sub-Saharan Africa and 1.08 in India. The figure for sub-Saharan Africa is similar to that found by Garenne (2002) using both Demographic and Health Surveys and World Fertility Surveys from this region. A lower sex ratio among Africans has also been noted in the United States and the United Kingdom. It has been advanced that higher levels of circulating gonadotropin in black women increase their probability of conceiving girls, resulting in lower sex ratios (James (1984)). The figure for India is the same as that found in the 2001 Indian Census, and by Rosenzweig and Schultz (1982) based on a nationally representative sample of rural households in India, and is close to the sex ratio of 1.09 found by Pakrasi and Halder (1971) using the 1961-62 Indian National Sample Survey. High sex ratios at birth in India have been explained by the abortion of female fetuses, a persistent form of discrimination against female children prevalent in most countries of South and East Asia (Sen (1990b); Sen (1992), Ebenstein (2007)). Sex

ratios in sub-Saharan Africa and India are significantly different from the world sex ratio of 1.055.

4.2.2 Socioeconomics

Table 2 shows the summary statistics of common demographic and socioeconomic variables for twins and singletons. In sub-Saharan Africa and India, twins and singletons are similar along several characteristics such as maternal age, marital status and education, and paternal education. Twins tend to be born to slightly older mothers than singletons in both regions, and in sub-Saharan Africa, singletons are more likely to be born to single mothers. In India, a slightly higher proportion of twins than singletons are born to mothers or fathers with a secondary or higher level education. With respect to household characteristics, we note that twins live in slightly larger size households than singletons. Twins and singletons do not significantly differ along other household characteristics such as the level of wealth, here measured by the possession of facilities and assets such as electricity, radio, TV, and car. This comparison of twins and singletons shows that the sample of twins is not a selected sample based on common demographic and socioeconomic characteristics. Other studies have also found that twins largely mirror the entire population along several demographic and socioeconomic variables (see e.g., Almond, Chay and Lee (2005)).

4.2.3 Mortality

Table 2 shows that twins die at a higher rate compared to singletons. In sub-Saharan Africa, the probability of dying before the fifth birthday is 163 per thousand for singletons and 405 per thousand for twins. These figures are respectively 115 and 329 per thousand in India. Twins-singletons differences in mortality rates are high in early life, but decreases with age. The twins-singletons mortality rates ratio in the neonatal period is close to 5 in sub-Saharan Africa (193 vs. 41 per thousand), and is close to 6 in India (287 vs. 50 per thousand). But in the childhood period, this ratio falls below 2 in both regions. This simply shows that while twins are not selected based on

common demographic and socioeconomic factors as previously demonstrated, being a twin has a multiplicative effect on mortality.

5 Results

We first describe sex differences in mortality, and second, we estimate these differences in a multivariate framework using the methodology developed in section 3.1. Based on the multivariate results, we decompose sex differences in mortality into the effects of pre-birth environmental factors, child immune system, and parental preferences, following the methodology presented in section 3.2.

5.1 Sex differences in mortality

Table 3 shows male-female differences in mortality rates at different ages in sub-Saharan Africa and India. We note that during infancy, male children die at a higher rate compared to female children in both regions. The sex gap in infant mortality rate is 46 per thousand points in sub-Saharan Africa and 27 per thousand points in India. This female advantage is generally attributed to sex difference in biological make-up. In the sample of male-female twins, the sex gap drops to 27 per thousand points in sub-Saharan Africa, and completely reverses in India, where mortality is now 10 per thousand points higher among girls than boys. Since male-female twins have the same exposure to pre-birth environmental factors, the smaller sex gap found in the sample of male-female twins in sub-Saharan Africa clearly rules out the effect of these factors, and the reversed gap in India additionally shows the effect of discrimination against female children.

In the neonatal, postneonatal and childhood periods, the results are qualitatively the same as in the infancy period. We however note that while female children have a survival advantage in the neonatal period, they die at a higher rate in subsequent periods in India, while still keeping their advantage in sub-Saharan Africa.

5.2 Decomposing sex differences in mortality into the effects of pre-birth factors, child biology and parental preferences

5.2.1 Infant mortality

We estimate equations (1) and (5). The dependent variable is a dummy indicator taking on the value 1 if the child died before his/her first birthday, and 0 if not. Results are presented in Panel A of Table 4. Columns (I)-(IV) show the results for sub-Saharan Africa, and Columns (V)-(VIII) show the results for India. In Columns (I) and (V), the dependent variable is regressed on a dummy indicator taking on the value 1 if the child is male, and 0 if the child is female, using a linear probability model on the full sample of twins. As shown in descriptive analysis, the probability of dying before the first birthday is 47 and 27 per thousand points higher among males compared to females both in sub-Saharan Africa and India, respectively. In Columns (II) and (VI), we control for child, parental and household characteristics as well as country and year of survey fixed effects.²¹ This changes little to the estimates obtained in Column (I) and (VI). The male-female difference in infant mortality decreases to 45 per thousand points in sub-Saharan Africa, but remains the same in India.

The existence of unobserved pre-birth environmental factors that determine both child sex and health as implied by the biological literature surveyed in section 2 implies that the estimates of the sex gap in infant mortality obtained in Columns (I)-(II) and (V)-(IV) are biased. We correct for this bias by estimating a within male-female twin fixed effect regression in Columns (III)-(IV) for sub-Saharan Africa and Columns (VII)-(VIII) for India. The estimation of Columns (III) and (VII) is based on the full sample of twins and that of Columns (IV) and (VIII) is based on the restricted sample of male-female twin pairs. Infant mortality is now only 27 per thousand points higher among boys compared to girls in sub-Saharan Africa, and is 10 per thousand points lower in boys compared to girls in India. However the estimate

²¹Child, parental and household characteristics include child's year of birth, maternal age at survey, education and marital status, husband education, household size, possession of assets and facilities such as car, television, radio and electricity, a linear control for year of survey, and country fixed effect.

for India is not statistically significant at the level 10%.

Table 5, Panel A shows the decomposition of the sex difference in infant mortality into the effects of pre-birth environmental factors, child biology and parental preferences. These estimates are computed based on the point estimates obtained in Columns (II) and (III) for sub-Saharan Africa, and Columns (VI) and (VII) for India. It results from this calculation that pre-birth environmental factors play a significant role in sex differential mortality rates. These factors raise boys infant mortality rate by (45-27) 18 per thousand points in sub-Saharan Africa and by (27 -(-10)) 37 per thousand points in India. Male-female differential immune system is also an important factor in the sex gap in mortality as supported by the biological literature (Waldron (1983)), but its role is much less important than previously thought, due to failure to account for pre-birth factors in previous studies. The biological make-up of male children increases their infant mortality rate by 27 per thousand points in sub-Saharan Africa and India. Finally, discrimination against female children in India increases their mortality rate by 37 per thousand points. This finding contradicts most studies that found that the effect of parental discrimination against girls in early age in India and other southern and eastern Asian countries had negligible effect on their mortality. As already mentioned, such studies derive their conclusion from the fact that during infancy, boys die at a higher rate compared to girls as shown by Figure 1 for India; but our analysis shows that if we adjust for pre-birth environmental effects and biology, we clearly see a huge effect of discrimination against girls, which is consistent with the well documented phenomena of female neglect, abandonment, and infanticide which take place in very early life (Sudha and Rajan (1999)).

5.2.2 Neonatal mortality

We replicate the previous analysis for neonatal mortality. The results are presented in Table 4, Panel B. Columns (I) and (V) show that neonatal mortality is respectively 37 and 43 per thousand points higher among male children than female children in sub-Saharan Africa and India. After controlling for child, parental and household characteristics as well as country and year fixed effects in Columns (II) and (VI), the coefficient on male decreases to 36 per thousand points in sub-Saharan Africa,

and increases to 45 per thousand points in India. Estimation of twins fixed effects regression in Columns (III)-(IV) and (VII)-(VIII) shows that neonatal mortality is respectively 22 and 9 per thousand points higher among males in sub-Saharan Africa and India, respectively, but the estimate for India is not statistically different from zero.

Table 5, Panel B shows the decomposition of sex differences in neonatal mortality. Pre-birth environmental factors increase the neonatal mortality rate of male children by respectively 14 and 36 per thousand points in sub-Saharan Africa and India. The biological make-up of male children contributes 22 per thousand points to their elevated mortality, and parental discrimination against female children in India increases their mortality rate by 13 per thousand points. Again, this latter finding shows that the detrimental effect of female discrimination in India is apparent in very early life.

5.2.3 Postneonatal mortality

The estimation of sex imbalance in postneonatal mortality is shown in Table 4, Panel C. Regressing a dummy indicator for postneonatal death on child sex (Columns (I) and (V)) shows that postneonatal mortality is 18 per thousand points higher among boys than girls in sub-Saharan Africa, but is 14 per thousand points lower among boys in India, although the estimate for India is not statistically significant at the level 10%. Adding controls change little to those estimates (Columns (II) and (VI)). The estimation of the twins fixed effects regression (Columns (III)-(IV) and (VII)-(VIII)) results in an important reduction of female survival advantage in sub-Saharan Africa, and increases their disadvantage in India. Postneonatal mortality is now only 10 per thousand points higher among boys compared to girls in sub-Saharan Africa, and is 32 per thousand points higher among girls in India.

The results of the decomposition exercise for postneonatal mortality is presented in Table 5, Panel C. We note that pre-birth environmental factors increase the mortality of male children by 8 and 18 per thousand points in sub-Saharan Africa and India, respectively, the biological make-up of male children contributes 10 per thousand points to their elevated mortality rate, and parental discrimination against female children in India raises their mortality by 42 per thousand points.

5.2.4 Child mortality

The results for child mortality are presented in Table 4, Panel D. A dummy indicator for whether a child who survived the first year of life died during the childhood period is regressed on a dummy indicator for male (Columns (I) and (V)). We note that child mortality is 4 per thousand points higher among males in sub-Saharan Africa (results not statistically significant), but is 17 per thousand points lower in this sex in India. Adding controls change little to the estimate of the sex gap for sub-Saharan Africa, and increases the estimate for India by 1 per thousand point (Columns (II) and (VI)). Estimating the twins fixed effects regression (Columns (III)-(IV) and (VII)-(VIII)), child mortality is now 8 and 31 per thousand points lower among male children compared to female children in sub-Saharan Africa and India, respectively.

Table 5, Panel D presents the decomposition of the sex gap in the childhood period. We note that pre-birth environmental factors increase the mortality of male children by 12 and 15 per thousand points in sub-Saharan Africa and India, respectively. But contrary to the long-held biological theory of sex differences in morbidity and mortality, male biological make-up now favors male survival during the childhood period. The biological make-up of male children reduces their mortality rate by 8 per thousand points. Parental discrimination against female children in India increases their mortality rate by 23 per thousand points.

6 Extending the decomposition of the sex gap in mortality to singletons

We extend to singletons the decomposition of sex differences in mortality into the effects of pre-birth environmental factors, child biology, and parental preferences. This extension mainly relies on the fact that pre-birth environmental factors that determine offspring sex ratios are the same for twins and singletons, as the sex ratios at birth of same-sex twins and singletons are similar (see Table 1). Given that the sample of twins is not selected on common demographic and socioeconomic factors as demonstrated in section 4.2.1, we therefore only need to adjust for the higher mortality of twins

when extending the decomposition of the sex gap to singletons. For this purpose, we simply assume that the relative contribution of pre-birth environmental factors to sex differences in mortality is the same for twins and singletons. This assumption relies on the fact that mechanisms through which these factors affect health are largely non-behavioral and do not depend on family size. The methodology for the extension is detailed below.

6.1 Methodology

We estimate a linear probability model regression as well as a within siblings fixed effects regression (equations (1) and (3)) based on the sample of singletons, which yields θ and θ_{SFE} , respectively. Following our assumption that the relative contribution of pre-birth environmental factors to sex differences in mortality is the same for singletons and twins (that is, $\frac{\theta_1}{\theta}|_{\text{singletons}} = \frac{\theta_1}{\theta}|_{\text{twins}}$), we are able to calculate the absolute contribution of pre-birth factors to sex differences in mortality among singletons (θ_1) by multiplying θ for singletons by $\frac{\theta_1}{\theta}$ for twins (that is, $\theta_1|_{\text{singletons}} = \theta|_{\text{singletons}} * \frac{\theta_1}{\theta}|_{\text{twins}}$). This yields $\theta_1^{ND}|_{\text{singletons}}$ and $\theta_1^D|_{\text{singletons}}$ for non-discriminatory and discriminatory environments, respectively. For non-discriminatory environments, the absolute contribution of child biology to sex differences in mortality (θ_2^{ND}) is therefore obtained by subtracting θ_1^{ND} for singletons from θ^{ND} for singletons (that is, $\theta_2^{ND}|_{\text{singletons}} = \theta^{ND}|_{\text{singletons}} - \theta_1^{ND}|_{\text{singletons}}$). For discriminatory environments, we assume, as for twins, that the effect of child biology is the same in non-discriminatory and discriminatory environments (that is, $\theta_2^D|_{\text{singletons}} = \theta_2^{ND}|_{\text{singletons}}$), and since we know $\theta_1^D|_{\text{singletons}}$ and $\theta_2^D|_{\text{singletons}}$ (because we know $\theta_2^{ND}|_{\text{singletons}}$), we extract the effect of parental discrimination by subtracting $\theta_1^D + \theta_2^D$ from θ^D (that is, $\theta_3^D|_{\text{singletons}} = \theta^D|_{\text{singletons}} - \theta_1^D|_{\text{singletons}} - \theta_2^D|_{\text{singletons}}$). This completely extends the decomposition of the sex gap to singletons. Note that there is no need to do the same for the entire population because the population of singletons largely proxies the general population because of the small fraction of twins.

6.2 Extension results

We estimate equations (1) and (3) and present the results in Table 6. As for twins, Panels A, B, C and D show the results for infant mortality, neonatal mortality, post-neonatal mortality, and child mortality, respectively. Similarly, Columns (I)-(III) and Columns (IV)-(VI) of each panel show results for sub-Saharan Africa and India, respectively. In Columns (I) and (IV) of each panel, mortality is regressed on sex without controls, in Columns (II) and (V), controls are included, and siblings fixed effects are added in Columns (III) and (VI). We note that all these specifications yield similar results for each mortality indicator. Infant mortality is 13 and 6-7 per thousand points higher for males compared to females in sub-Saharan Africa and India, respectively. Similarly, neonatal mortality is 11 and 9-11 per thousand points higher for males compared to females in sub-Saharan Africa and India, respectively. As for postneonatal mortality, it is 3-4 per thousand points higher for males in sub-Saharan Africa, but is 3 per thousand lower in this sex in India. Sex differences in child mortality show similar patterns. Child mortality is 2-3 per thousand points higher for boys in sub-Saharan Africa, but is 10-11 per thousand points lower in this sex in India. We note that patterns of male-female mortality are qualitatively similar to those found for twins.

We now proceed to the extension of the sex gap in mortality to singletons following the methodology outlined in section 6.1, and using the point estimates of Table 6, Column (II) for sub-Saharan Africa, and Table 6, Column (V) for India. The results are presented in Table 7. Panels A, B, C and D show the results for infant mortality, neonatal mortality, postneonatal mortality, and child mortality, respectively. We note that pre-birth environmental factors increase the infant mortality rate of male children by 5 and 8 per thousand points in sub-Saharan Africa and India, respectively. The biological make-up of male children contributes 8 per thousand points to their excess mortality rates, and parental discrimination against female children in India increases their neonatal mortality rate by 10 per thousand points. The results are qualitatively similar for neonatal and postneonatal mortality. For child mortality, we note that pre-birth environmental factors increase the mortality of male children

by 6 and 9 per thousand points in sub-Saharan Africa and India, respectively, the biological make-up of male children reduces their mortality rate by 4 per thousand points, and parental discrimination against female children in India increases their mortality rate by 15 per thousand points.

The results in Table 7 shows that failing to account for pre-birth effects results in an upward bias in the estimate of the biological effect. In sub-Saharan Africa, we found that infant mortality is 13 per thousand points higher among males than females, and that only 8 per thousand points of this elevated male mortality can be attributed to biology, while 5 per thousand points is attributed to pre-birth environmental factors. This implies that studies that do not adjust for pre-birth effects overestimate the effect of biology on sex differences in infant mortality by $\left(\frac{13-8}{8}\right)*100$ 62.5 percent. In the childhood period, we found that the elevated mortality of male children is solely attributable to pre-birth environmental factors, and that their biological make-up favors their survival, contrary to the long-held biological theory of sex differences in morbidity and mortality.

Failing to account for the effects of pre-birth factors and child biology also implies that in discriminatory environments such as India, the effect of parental discrimination against girls is usually grossly underestimated. The results for the decomposition exercise for India implies that failing to adjust for pre-birth and biological factors results in underestimating the effect of female discrimination on mortality by 160 percent during infancy, 350 percent during the neonatal period, 67 percent during the post-neonatal period, and 33 percent during childhood. Adjusting for the effect of biology, but failing to adjust for the effects of pre-birth factors result in underestimating the effect of female discrimination by 80 percent during infancy, 175 percent during the neonatal period, 44 percent during the postneonatal period, and 20 percent during childhood. These underestimates have wrongly led to the conclusion that discrimination against female children in India has negligible effect in very early life (see e.g., Sen (1990), Oster (2006)). Our findings that discrimination against girls in this country has important effect on their mortality are consistent with frequent observation of widespread practices such as female infanticide, neglect, and abandonment that occur in early stage of postbirth life.

7 Conclusion

Sex imbalance in early age mortality raises a fundamental issue of equity that has long attracted the attention of scholars and policymakers. Understanding the origins of this imbalance is essential in designing policies that efficiently address this crucial issue. It has been observed in almost all populations that male children die at a higher rate compared to female children. But the survival advantage of female children diminishes and eventually reverses by age five in some countries of South and East Asia. It has been long advanced that male-female differential biological make up accounts for the higher mortality of male children, and that parental preferences for boys in Southern and Eastern Asian countries leading to the neglect of female children cause them to lose their initial survival advantage after a few months of life. In this study, we argue that in all studies that have examined sex and gender differences in mortality, sex has been treated as an exogenous variable. Evidences from the biological literature however show that sex is endogenous to pre-conceptual and pre-birth environmental factors that are also likely to affect the health and survival of a baby *in utero* and after birth. This implies that conventional estimates of sex imbalance in early age mortality are biased, due to failure to account for these factors. Using samples of twins from sub-Saharan Africa and India has allowed us to correct for this bias. We have decomposed sex differences in early age mortality into the distinct effects of pre-birth environmental factors, child biology, and parental preferences. The findings of our analysis lead to at least three important conclusions: (1) pre-birth unobserved environmental factors account for a large fraction of the higher mortality rates of male children; (2) the biological make-up of male children contributes to their excess mortality rates only during infancy, however its effect is usually overestimated due to failure to control for pre-birth factors; but contrary to the long-held biological theory of sex imbalance in morbidity and mortality, the biological make-up of male children favor their survival during childhood; (3) in India, parental discrimination against female children has a sizeable effect on their mortality; failure to adjust for pre-birth and biological effects has however led conventional estimates of sex and gender differences in mortality to underestimate its effect by about 160 percent during infancy,

and 33 percent during childhood. This bias has prevented scholars from estimating the true effect of discrimination in very early life, wrongly leading some to conclude that female discrimination is mostly important during childhood. Our findings that discrimination against girls in India has important effect on their mortality in very early ages are consistent with frequent observation of widespread phenomena such as female infanticide, neglect, and abandonment that occur in early stage of postbirth life.

That male-female differential immune system has long been advanced as the unique explanation for the higher mortality of male children in almost all populations has left the impression that little can be undertaken to improve their survival chance. The demonstrated role of pre-conceptional and pre-birth environmental factors in this excess mortality certainly calls for new investigations of mechanisms through which these factors affect life. In this respect, our analysis is obviously limited, due to the fact that these factors are unobserved in the data we use, as in most household surveys from developed and developing countries. Our analysis also demonstrates the role of discrimination in increasing the mortality of female children in India. That this effect has previously been grossly underestimated simply means that new efforts should be undertaken by researchers, governments and policymakers to combat this very crucial problem that unjustly prevents million of women from life, leaving on its trail numerous issues that jeopardize the social and economic stability of countries.

References

Alderman, H., and P. Gertler (1997): “Family Resources and Gender Differences in Human Capital Investments: The Demand for Children’s Medical Care in Pakistan,” in *Intrahousehold Resource Allocation in Developing Countries: Models, Methods, and Policy*, ed. by L. Haddad, J. Hoddinott, and H. Alderman. Baltimore: Johns Hopkins University Press.

Ansar Ahmed, S., W.J. Penhale, and N. Talal (1985): “Sex Hormones, Immune Responses, and Autoimmune Diseases. Mechanisms of Sex Hormone Action,” *American Journal of Pathology*, 121(3), 531-51.

Ansar Ahmed, S., and N. Talal (1990): “Sex Hormones and the Immune System—part 2: Animal Data,” *Baillière’s Clinical Rheumatology*, 4(1), 13-31.

Almond, D., K. Chay, and D. Lee (2005) “The Costs of Low Birth Weight,” *Quarterly Journal of Economics*, 120(3), 1031-1083.

Basu, A.M. (1989): “Is Discrimination in Food Really Necessary for Explaining Sex Differentials in Childhood Mortality?,” *Population Studies*, 43 (2), 193–210.

Basu, A.M. (1992): *Culture, the Status of Women, and Demographic Behaviour*. Oxford: Clarendon Press.

Berhman, J.R. (1988): “Intrahousehold Allocation of Nutrients in Rural India: Are Boys Favored? Do Parents Exhibit Inequality Aversion?” *Oxford Economic Papers*, 40(1), 32-54.

Black R.E., S.S. Morris, and J. Bryce (2003): “Where and Why are 10 million Children Dying every Year? *Lancet*, 361, 2226-2234.

Borooah, V. (2004): “Gender Bias among Children in India in their Diet and Immunisation against Disease,” *Social Science and Medicine*, 58, 1719–1731.

Bouman, A., and M.J. Heineman, and M.M. Faas (2005): “Sex Hormones and the Immune Response in Humans,” *Human Reproduction Update*, 11(4), 411-23.

Chao, T.C. (1996): “Female Sex Hormones and the Immune System,” *Chang Gung Medical Journal*, 19(1), 95-106.

Chen, L.C., E. Huq, and S. D’Souza (1981): “Sex Bias in the Family Allocation of Food and Health Care in Rural Bangladesh,” *Population and Development Review*, 7, 55-70.

Coale, A., and J. Banister (1994): "Five Decades of Missing Women in China," *Demography*, 31(3), 459-479.

Croll, E. (2001): *Endangered daughters: Discrimination and development in Asia*. London: Routledge.

Davis, D.L., M.B. Gottlieb, and J.R. Stampnitzky (1998): "Reduced Ratio of Male to Female Births in Several Industrial Countries: A Sentinel Health Indicator?," *Journal of the American Medical Association*, 279, 1018-1023.

Deaton, A. (2001): "Looking for Boy-Girl Discrimination in Household Expenditure Data," *The World Bank Economic Review*, 3(1), 1-15.

Dimid-Ward, H., C. Hertzman, K. Teschke, R. Hershler, S.A. Marion, and A. Ostry (1996): "Reproductive Effects of Paternal Exposure to Chlorophenolate Wood Preservatives in the Sawmill Industry," *Scandinavaian Journal of Work, Environment and Health*, 22, 267-273.

Drew, J.S., B.S. Blumberg, and J. Robert-Lamblin (1986): "Hepatitis B Virus and Sex Ratio of Offspring in East Greenland," *Human Biology*, 58, 115-20.

Drèze, J., and A. Sen (1996): *India: Economic Development and Social Opportunity*. Oxford: Clarendon Press.

Ebenstein, A.Y. (2007): "Fertility Choices and Sex Selection in Asia: Analysis and Policy," *Mimeo*, University of California Berkeley.

Garenne, M. (1992): "Sex Differences in Measles Mortality: A World Review," *Harvard Center for Population and Development Studies Working Paper*, No.92.04.

Garenne, M. (2002): "Sex Ratios at Birth in African Populations: A Review of Survey Data," *Human Biology*, 74 (6), 889-900.

Garenne, M. (2003): "Sex Differences in Health Indicators among Children in African DHS Surveys," *Journal of Biosocial Science*, 35, 601-614.

Garry, V.F., D. Schreinemachers, M.E. Harkins, J. Griffith (1996): "Pesticide Appliers, Biocides, and Birth Defects in Rural Minnesota," *Environmental Health Perspectives*, 104(4), 394-399.

Garry, V.F., M.E. Harkins, L.L. Erickson, L.K. Long-Simpson, S.E. Holland, and B.L. Burroughs (2002): "Birth Defects, Season of Conception, and Sex of Children Born to Pesticide Applicators Living in the Red River Valley of Minnesota, USA,"

Environmental Health Perspectives, 110, s3, 441-449.

Graunt, J. (1662): *Natural and Political Observations mentioned in a following index and made upon the Bills of Mortality*. eprinted by the Johns Hopkins University Press, ed. by Walter F. Wilcox. Baltimore, 1939.

Hazarika, G. (2000): "Gender Differences in Children's Nutrition and Access to Health Care in Pakistan," *Journal of Development Studies*, 37, 73-92.

Hesser, J.E., J. Economidou, and B.S. Blumberg (1975): "Hepatitis B Surface Antigen (Australia Antigen) in Parents and Sex Ratio of Offspring in a Greek population," *Human Biology*, 47 (4), 415-425.

James, W.H. (1984): "The Sex Ratios of Black Births," *Annals of Human Biology*, 11(1), 39-44.

James, W.H. (1994): "The Sex Ratios of Offspring of Patients with Multiple Sclerosis," *Neuroepidemiology*, 13, 216-219.

James, W.H. (1995): "What Stabilizes the Sex Ratio?," *Annals of Human Genetics*, 59, 243-249.

James, W.H. (1996): "Evidence that Mammalian Sex Ratios at Birth are Partially Controlled by Parental Hormone Levels at the Time of Conception," *Journal of Theoretical Biology*, 190, 271-286.

James, W.H. (1997): "The Sex Ratio of Offspring Sired by Men Exposed to Wood Preservatives Contaminated by Dioxin," *Scandinavian Journal of Work, Environment and Health*, 23, 69.

James, W.H. (1998): "Hypotheses on Mammalian Sex Ratio Variation at Birth," *Journal of Theoretical Biology*, 192, 113-116.

James, W.H. (2001): "Sex Ratios at Birth as Monitors of Endocrine Disruption," *Environmental Health Perspectives*, 109(6), A250-A251.

Jacobsen, R., E. Bostofte, G. Engholm, J. Hansen, N.E. Skakkebaek, and H. Moller H (2000): "Fertility and Offspring Sex Ratio of Men who Develop Testicular Cancer: A Record Linkage Study," *Human Reproduction*, 15, 1958-1961.

Kynch, J., and A. Sen (1983): "Indian Women: Well-being and Survival," *Cambridge Journal of Economics*, 7, 363-380.

Milham, S. (1964). "Pituitary Gonadotrophin and Dizygotic Twinning," *Lancet*,

ii, 566.

Moller H. (1998): "Trends in Sex Ratio, Testicular Cancer and Male Reproductive Hazards: Are They Connected?" *Acta Pathologica et Microbiologica Scanddinavica*, 106, 232–239.

Mocarelli, P., P. Brambilla, P.M. Gerthoux, D.G. Patterson Jr., and L.L. Needham (1996): "Change in Sex Ratio with Exposure to Dioxin," *Lancet*, 348, 409.

P. Mocarelli, P.M. Gerthoux, E. Ferrari, D.G. Patterson Jr., S.M. Kieszak, P. Brambilla, N. Vincoli, S. Signorini, P. Tramacere, V. Carreri, E.J. Sampson, W.E. Turner, and L.L. Needham (2000): "Paternal Concentrations of Dioxin and Sex Ratio of Offspring," *Lancet*, 355, 1858–1863.

Osmani, S., and A. Sen (2003): "The Hidden Penalties of Gender Inequality: Fetal Origins of Ill-health," *Economics and Human Biology*, 1(1), 105-121.

Oster, E. (2005): "Hepatitis B and the Case of the Missing Women," *Journal of Political Economy*, 113(6), 1161-1216.

Oster, E. (2006): "Proximate Causes of Population Gender Imbalance in India," *Mimeo*, University of Chicago.

Pakrasi, K., and A. Halder (1971): "Sex Ratios and Sex Sequences in India," *Journal of Biosocial Science*, 1971, 3, 377-87.

Pande, R. (2003): "Selective Gender Differences in Childhood Nutrition and Immunization in Rural India: The Role of Siblings," *Demography*, 40 (3), 395–418.

Preston, S.H. (1976): *Mortality Patterns in National Populations*. Academic Press, New-York.

Preston, S.H., and J.A. Weed (1976): "Causes of Death Responsible for International and Intertemporal Variation in Sex Mortality Differential," *World Health Statistics Reports*, 30(4), 144-88.

Qian, N. (2005) "Missing Women and the Price of Tea in China: The Effect of Relative Female Income on Sex Imbalance," *Mimeo*, Brown University.

Rose, Elaine (1999): "Consumption Smoothing and Excess Female Mortality in India," *The Review of Economics and Statistics*, 81(1), 41-49.

Rosenzweig, M.R., and T.P. Shultz (1982): "Market Opportunities, Genetic Endowments and Intrafamily Resource Distribution: Child Survival in India," *American*

Economic

Review, 72 (4), 803–815.

Sen, A. (1984): “Family and Food: Sex Bias in Poverty,” in *Resources, Value and Development* ed. by A. Sen. London: Blackwell.

Sen, A. (1989): “Women’s survival as a development problem,” *Bulletin of the American Academy of Arts and Sciences*, 43, 14-29.

Sen, A. (1990a): “Gender and Cooperative Conflict,” in *Persistent Inequalities: Women and World Development* ed. by I. Tinker. New York: Oxford University Press.

Sen, A. (1992): “Missing Women,” *British Medical Journal*, 304, 587–588.

Sen, A. (1990b): “More than 100 Million Women are Missing,” *New York Review of Books*, 37(20).

Sen, A. (1999): *Development as Freedom*. Knopf/Oxford University Press, New York/Oxford.

Sen, A., and S. Sengupta (1983): “Malnutrition of Rural Children and the Sex Bias,” *Economic and Political Weekly*, 18, 855-864.

Stevens, N.M. (1905): “Studies in Spermatogenesis, with Especial Reference to the Accessory Chromosome,” *Carnegie Institute Wash.*, 36,1-32.

Sudha, S., and S.I. Rajan (1999): “Female Demographic Disadvantage in India 1981-1991: Sex Selective Abortions and Female Infanticide,” *Development and Change*, 30(3), 585-618.

Waldron, I. (1983): “Sex Differences in Human Mortality: the Role of Genetic Factors,” *Social Science and Medicine*, 17(6), 321–333.

Waldron, I. (1985): “What Do We Know about Causes of Sex Differences in Mortality? A Review of the Literature,” *Population Bulletin of the United Nations*, 18, 59–76.

Waldron, I. (1998): “Sex Differences in Infant and Early Childhood Mortality: Major Causes of Death and Possible Biological Causes,” in *Too Young To Die: Genes or Gender?* New York: United Nations, 1998.

Williams, F.L.R., A.B. Lawson, and O.L. Lloyd (1992): “Low sex ratio of births in areas at risk from air pollution from incinerators as shown by geographical analysis

and 3-dimensional mapping,” *International Journal of Epidemiology*, 21, 311–319.

Wilson, E.B. (1905): “Studies on Chromosomes 1. The Behavior of the Idiochromosomes,” *The Journal of Experimental Zoology*, 2, 371-405.

Wilson, E.B. (1909): “Recent Researches on the Determination and Heredity of Sex,” *Science*, 29, 52-70.

World Bank (2001): *Engendering Development: Through Gender Equality in Rights, Resources, and Voice*. Washington, DC: The World Bank.

Table 1: Sex ratios at birth of singletons and twins in sub-Saharan Africa and India

	Africa		India	
	Sample size	% boys (S.D)	Sample size	% boys (S.D)
Singletons	1,619,483	0.508 (0.500)	537,061	0.520 (0.500)
All twins	50,994	0.504 (0.500)	6,920	0.514 (0.500)
Boy-Girl	20,154	0.500 (0.500)	2,232	0.500 (0.500)
Boy-Boy	15,610	1 (0)	2,442	1 (0)
Girl-Girl	15,230	0 (0)	2,246	0 (0)
Same sex twins	30,840	0.506 (0.500)	4,688	0.521 (0.500)

Table 2: Summary statistics

Variables	Sub-Saharan Africa						India					
	Singletons			Twins			Singletons			Twins		
	N	Mean	S.D	N	Mean	S.D	N	Mean	S.D	N	Mean	S.D
Child is male	1,619,483	0.508	0.500	50,994	0.504	0.500	537,061	0.520	0.500	6,920	0.514	0.500
Maternal characteristics												
Age	1,619,483	35.104	8.062	50,994	36.343	7.521	537,061	34.772	8.040	6,920	35.225	7.804
Marital status												
Single	1,619,432	0.022	0.148	50,994	0.015	0.122	537,061	0.000	0.000	6,920	0.000	0.000
Married	1,619,432	0.769	0.422	50,994	0.771	0.420	537,061	0.943	0.232	6,920	0.942	0.233
Widowed	1,619,432	0.047	0.211	50,994	0.050	0.218	537,061	0.047	0.212	6,920	0.047	0.211
Living with a partner	1,619,432	0.097	0.296	50,994	0.096	0.295	537,061	0.000	0.000	6,920	0.000	0.000
Not living with a partner	1,619,432	0.034	0.181	50,994	0.037	0.188	537,061	0.008	0.090	6,920	0.008	0.090
Divorced or separated	1,619,432	0.031	0.173	50,994	0.030	0.172	537,061	0.002	0.042	6,920	0.003	0.051
Education												
Not Educated	1,619,404	0.554	0.497	50,990	0.558	0.497	536,070	0.624	0.484	6,908	0.597	0.491
Primary	1,619,404	0.335	0.472	50,990	0.335	0.472	536,070	0.171	0.376	6,908	0.184	0.387
Secondary or higher	1,619,404	0.111	0.314	50,990	0.107	0.309	536,070	0.206	0.404	6,908	0.219	0.414
Father's education												
Not Educated	1,548,881	0.579	0.494	49,038	0.580	0.493	536,465	0.623	0.485	6,906	0.597	0.491
Primary	1,540,365	0.352	0.478	48,576	0.351	0.477	535,280	0.171	0.376	6,888	0.184	0.388
Secondary or higher	1,512,371	0.119	0.323	47,740	0.114	0.318	535,115	0.206	0.404	6,886	0.220	0.414
Household characteristics												
Household size	1,619,483	7.993	4.795	50,994	8.447	4.728	537,061	7.229	3.539	6,920	7.427	3.772
Has electricity (0/1)	1,519,492	0.170	0.376	47,648	0.167	0.373	537,061	0.596	0.491	6,920	0.610	0.488
Has radio (0/1)	1,584,591	0.551	0.497	49,820	0.556	0.497	536,867	0.415	0.493	6,920	0.427	0.495
Has TV (0/1)	1,532,985	0.126	0.332	47,972	0.122	0.327	536,918	0.304	0.460	6,920	0.309	0.462
Has car (0/1)	1,527,477	0.042	0.201	47,950	0.039	0.193	536,897	0.016	0.127	6,920	0.019	0.137
Child outcomes												
Infant mortality (0/1)	1,619,483	0.090	0.287	50,994	0.300	0.458	537,061	0.082	0.275	6,920	0.380	0.485
Neonatal mortality (0/1)	1,619,483	0.041	0.199	50,994	0.193	0.394	537,061	0.050	0.218	6,920	0.287	0.453
Postneonatal mortality (0/1)	1,552,795	0.051	0.220	41,175	0.133	0.340	510,302	0.034	0.181	4,932	0.130	0.337
Child mortality (0/1)	1,473,364	0.073	0.260	35,686	0.105	0.306	492,928	0.033	0.180	4,289	0.049	0.217

Table 3: Mortality rates of boys and girls in different age intervals in sub-Saharan Africa and India

	Sub-Saharan Africa				India			
	Boys		Girls		Boys		Girls	
	Mean	S.D	Mean	S.D	Mean	S.D	Mean	S.D
Infant mortality								
All twins	0.323	0.468	0.277	0.447	0.393	0.489	0.366	0.482
Male-female twins	0.307	0.461	0.280	0.449	0.338	0.473	0.348	0.476
Neonatal mortality								
All twins	0.211	0.408	0.174	0.379	0.308	0.462	0.265	0.441
Male-female twins	0.202	0.401	0.180	0.384	0.260	0.439	0.251	0.434
Postneonatal mortality								
All twins	0.143	0.350	0.124	0.330	0.123	0.329	0.138	0.345
Male-female twins	0.132	0.339	0.122	0.328	0.105	0.307	0.129	0.336
Child mortality								
All twins	0.107	0.309	0.103	0.304	0.041	0.198	0.058	0.234
Male-female twins	0.095	0.293	0.101	0.301	0.031	0.174	0.063	0.243

Table 4: Linear probability model estimates of sex differences in mortality based on twins data from sub-Saharan Africa and India

	Sub-Saharan Africa				India			
	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)	(VIII)
Panel A: Infant mortality								
Male	0.047*** [0.004]	0.045*** [0.004]	0.027*** [0.005]	0.027*** [0.005]	0.027** [0.012]	0.027** [0.011]	-0.010 [0.016]	-0.010 [0.016]
# Observations	50,994	50,994	50,994	20,154	6,920	6,920	6,920	2,232
Panel B: Neonatal mortality								
Male	0.037*** [0.003]	0.036*** [0.003]	0.022*** [0.004]	0.022*** [0.004]	0.043*** [0.011]	0.045*** [0.011]	0.009 [0.013]	0.009 [0.013]
# Observations	50,994	50,994	50,994	20,154	6,920	6,920	6,920	2,232
Panel C: Postneonatal mortality								
Male	0.018*** [0.003]	0.018*** [0.003]	0.010** [0.004]	0.010** [0.004]	-0.014 [0.010]	-0.014 [0.010]	-0.032** [0.015]	-0.032** [0.015]
# Observations	41,175	41,175	37,958	14,976	4,932	4,932	4,324	1,450
Panel D: Child mortality								
Male	0.004 [0.003]	0.004 [0.003]	-0.008* [0.005]	-0.008* [0.005]	-0.017*** [0.007]	-0.016** [0.007]	-0.031*** [0.011]	-0.031*** [0.011]
# Observations	35,686	35,686	30,176	11,928	4,289	4,289	3,418	1,158
Twins FE	NO	NO	YES	YES	NO	NO	YES	YES
Controls	NO	YES	NO	NO	NO	YES	NO	NO
All twins sample	YES	YES	YES	NO	YES	YES	YES	NO
Male-female twins sample only	NO	NO	NO	YES	NO	NO	NO	YES

In Columns (II) and (VI) of each panel, controls include child's year of birth; mother's characteristics including age at survey, education, and marital status; husband's education; household's characteristics including household size, possession of assets such as car, television, radio, and electricity; and a linear control for year of survey, and country fixed effect. Standard errors are in brackets, and are corrected for clustering of observations within twin-pairs for Columns (III), (IV), (VII) and (VIII).

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

Table 5: Decomposition of sex differences in mortality into the effects of pre-birth environmental factors, child biology and parental preferences based on twins data

	Sub-Saharan Africa			India		
	Sex differences in mortality attributable to:			Sex differences in mortality attributable to:		
	Pre-birth factors	Child biology	Parental preferences	Pre-birth factors	Child biology	Parental preferences
Infant mortality	0.018	0.027	0	0.037	0.027	-0.037
Neonatal mortality	0.014	0.022	0	0.036	0.022	-0.013
Postneonatal mortality	0.008	0.010	0	0.018	0.010	-0.042
Child mortality	0.012	-0.008	0	0.015	-0.008	-0.023

The decomposition of sex differences in mortality is based on the point estimates of Columns (II) and (III) of Table 4 for sub-Saharan Africa and Columns (VI) and (VII) of the same table for India.

Table 6: Linear probability model estimates of sex differences in mortality based on singletons data from sub-Saharan Africa and India

	Sub-Saharan Africa			India		
	(I)	(II)	(III)	(IV)	(V)	(VI)
Panel A: Infant mortality						
Male	0.013*** [0.000]	0.013*** [0.000]	0.013*** [0.001]	0.007*** [0.001]	0.006*** [0.001]	0.007*** [0.001]
# Observations	1,619,483	1,619,483	1,390,238	537,061	537,061	436,440
Panel B: Neonatal mortality						
Male	0.011*** [0.000]	0.011*** [0.000]	0.011*** [0.000]	0.010*** [0.001]	0.009*** [0.001]	0.011*** [0.001]
# Observations	1,619,483	1,619,483	1,390,238	537,061	537,061	436,440
Panel C: Postneonatal mortality						
Male	0.003*** [0.000]	0.003*** [0.000]	0.004*** [0.000]	-0.003*** [0.001]	-0.003*** [0.001]	-0.003*** [0.001]
# Observations	1,552,795	1,552,795	1,331,798	510,302	510,302	413,431
Panel D: Child mortality						
Male	0.003*** [0.000]	0.002*** [0.000]	0.002*** [0.000]	-0.010*** [0.001]	-0.010*** [0.001]	-0.011*** [0.001]
# Observations	1,473,364	1,473,364	1,261,345	492,928	492,928	398,062
Siblings FE	NO	NO	YES	NO	NO	YES
Controls	NO	YES	YES	NO	YES	YES

In Column (II) and (V) of each panel, controls include child's year of birth; mother's characteristics including age at survey, education, and marital status; husband's education; household's characteristics including household size, possession of assets such as car, television, radio, and electricity; and a linear control for year of survey, and country fixed effect. In Columns (III) and (VI), controls include child's year of birth and year of survey. Only the sample of children born to mothers who gave birth to at least one male child and one female child is used to estimate the model of Columns (III) and (VI). Standard errors are in brackets, and are corrected for clustering of observations within mothers for Columns (III) and (VI).

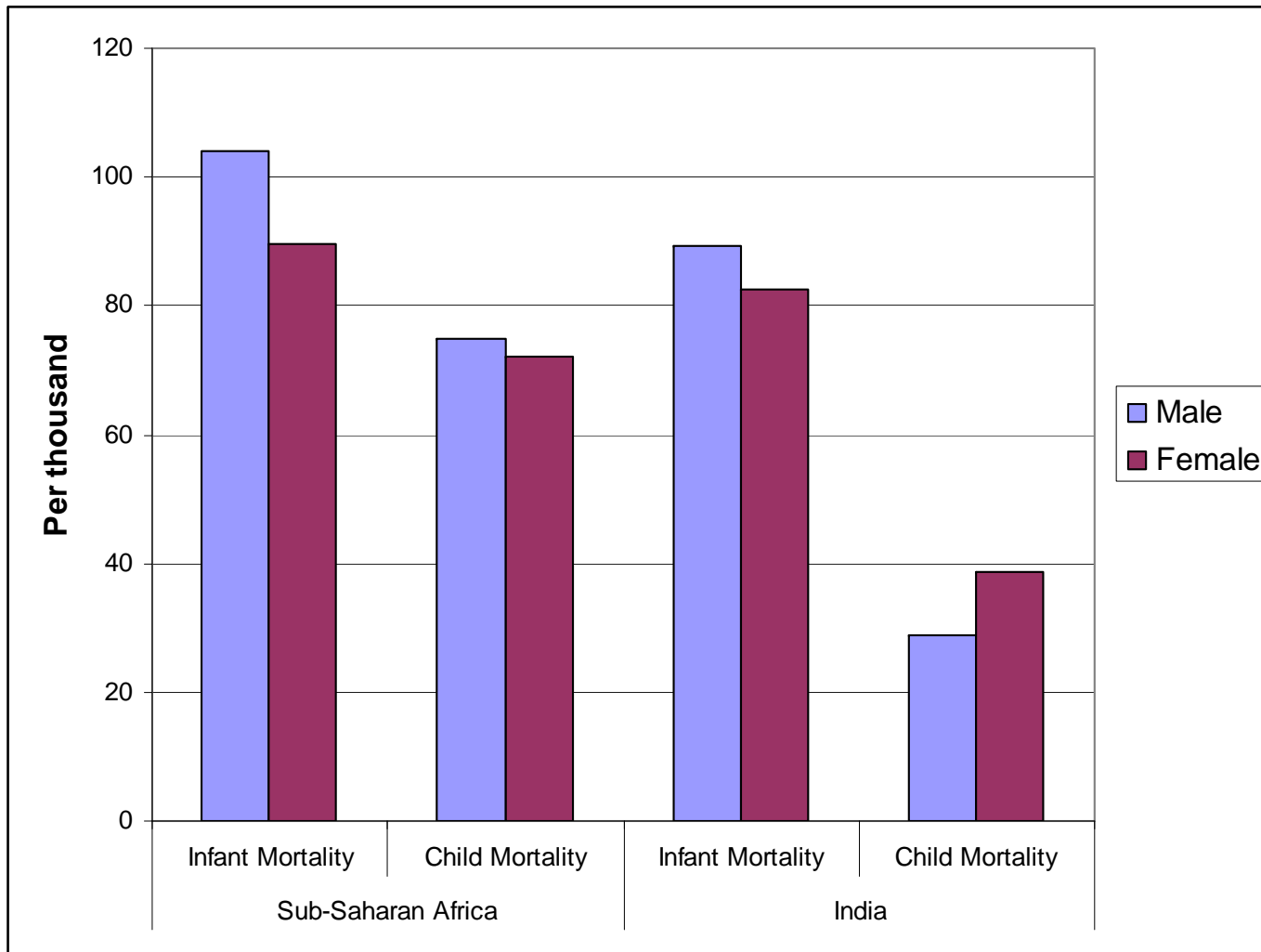
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 7: Extension of the decomposition of sex differences in mortality into the effects of pre-birth environmental factors, child biology and parental preferences to singletons

	Sub-Saharan Africa			India		
	Sex differences in mortality attributable to:			Sex differences in mortality attributable to:		
	Pre-birth factors	Child biology	Parental preferences	Pre-birth factors	Child biology	Parental preferences
Infant mortality	0.005	0.008	0	0.008	0.008	-0.010
Neonatal mortality	0.004	0.007	0	0.007	0.007	-0.005
Postneonatal mortality	0.001	0.002	0	0.004	0.002	-0.009
Child mortality	0.006	-0.004	0	0.009	-0.004	-0.015

The decomposition of sex differences in mortality is based on the point estimates of Column (II) of Table 6 for sub-Saharan Africa and Column (V) of the same table for India.

Figure 1: Sex differences in infant and child mortality in Sub-Saharan Africa and India



Appendix

Table A1

Countries	Years of Survey	Total sample size of live births	Sample size of twins	Sample size of singletons
India	1992/93, 1998/99	543,981	6,920	537,061
Sub-Saharan African Countries				
Benin	1996, 2001	38,703	1,880	36,823
Burkina Faso	1992/93, 1998/99, 2003	84,278	2,520	81,758
Burundi	1987	11,880	198	11,682
Central African Republic	1994/95	16,933	444	16,489
Cameroon	1994, 1998, 2004	56,218	2,116	54,102
Chad	1996/97, 2004	47,175	1,350	45,825
Comoros	1996	7,907	294	7,613
Côte d'Ivoire	1994, 1998/99, 2005	45,779	1,486	44,293
Ethiopia	2000, 2005	84,040	1,740	82,300
Gabon	2000	16,862	532	16,330
Ghana	1988, 1993, 1998, 2003	55,743	1,890	53,853
Guinea	1999, 2005	50,021	1,900	48,121
Kenya	1989, 1993, 1998, 2003	94,460	2,572	91,888
Lesotho	2004	14,699	422	14,277
Liberia	1986	17,261	698	16,563
Madagascar	1992, 1997, 2003/04	61,362	1,282	60,080
Malawi	1992, 1996, 2000, 2004	92,571	3,584	88,987
Mali	1987, 1995/96, 2001	98,535	2,788	95,747
Mozambique	1997, 2003	63,157	2,086	61,071
Namibia	1992, 2000	28,309	684	27,625
Niger	1992, 1998	52,702	1,558	51,144
Nigeria	1990, 1999, 2003	74,387	2,628	71,759
Rwanda	1992, 2000, 2005	77,087	1,702	75,385
Senegal	1986, 1992/93, 1997, 1999, 2005	102,487	2,608	99,879
South Africa	1998	22,905	558	22,347
Sudan	1990	25,793	684	25,109
Tanzania	1992, 1996, 2004	96,491	3,228	93,263
Togo	1988, 1998	37,009	1,532	35,477
Uganda	1988, 1995, 2000/01	62,203	1,618	60,585
Zambia	1992, 1996, 2001/02	70,702	2,334	68,368
Zimbabwe	1988, 1994, 1999, 2005/06	62,818	2,078	60,740