Demography (2013) 50:421–444
DOI 10.1007/s13524-012-0161-5

# Why Is Infant Mortality Higher in Boys Than in Girls? A New Hypothesis Based on Preconception Environment and Evidence From a Large Sample of Twins

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Published online: 14 November 2012


**Abstract** Infant mortality is higher in boys than girls in most parts of the world. This has been explained by sex differences in genetic and biological makeup, with boys being biologically weaker and more susceptible to diseases and premature death. At the same time, recent studies have found that numerous preconception or prenatal environmental factors affect the probability of a baby being conceived male or female. I propose that these environmental factors also explain sex differences in mortality. I contribute a new methodology of distinguishing between child biology and preconception environment by comparing male-female differences in mortality across opposite-sex twins, same-sex twins, and all twins. Using a large sample of twins from sub-Saharan Africa, I find that both preconception environment and child biology increase the mortality of male infants, but the effect of biology is substantially smaller than the literature suggests. I also estimate the interacting effects of biology with some intrauterine and external environmental factors, including birth order within a twin pair, social status, and climate. I find that a twin is more likely to be male if he is the firstborn, born to an educated mother, or born in certain climatic conditions. Male firstborns are more likely to survive than female firstborns, but only during the neonatal period. Finally, mortality is not affected by the interactions between biology and climate or between biology and social status.

**Keywords** Preconception origins hypothesis · Sex differences in mortality · Preconception environment · Child biology · Twins

## Introduction

The long-observed greater chance of survival for female children than for male children (Graunt 1662) has been explained by sex differences in genetic and biological makeup, with male children being biologically weaker and more susceptible to diseases than their female counterparts (e.g., Naeye et al. 1971; Waldron 1983). A

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common assumption in studies testing this biological hypothesis is that the offspring sex ratio[1] is randomly assigned across and within families. Recent studies, however, have revealed that it is not random. For example, girls are more likely to be born to smoking parents (Fukuda et al. 2002); to mothers with low gestational weight (Cagnacci et al. 2004); to aged parents (Jacobsen et al. 1999; Juntunen et al. 1997); and to parents with certain medical conditions, such as type 1 diabetes (James 1998a; Rjasanowski et al. 1998). An offspring sex ratio biased toward males has been observed among wealthy parents (Almond and Edlund 2007; James 1998b; Trivers and Willard 1973), men with multiple sclerosis, and men exposed to certain environmental toxins (James 1998b).

As discussed later in the article, the effects of these preconception or prenatal factors on sex ratio are large enough to constitute a source of bias in studies examining the determinants of sex differences in child outcomes. As a result, an explanation of the sex imbalance in morbidity and mortality that is based only on sex differences in genetic and biological makeup is limited. Moreover, conventional methodological approaches to quantifying the effect of child biology on these outcomes are likely to produce biased estimates.

In this article, I propose a methodology for estimating the distinct effects of preconception or prenatal environment and child biology on sex differences in infant mortality.[2] My methodological approach controls for the effects of unobserved preconception factors by exploiting variation in sex differences in mortality across same-sex twins, opposite-sex twins, and all twins (same-sex twins and opposite-sex twins). I apply this methodology to a large sample of twins extracted from 75 Demographic and Health Surveys (DHS) conducted in 31 countries in sub-Saharan Africa, a region where sons and daughters are treated equally (e.g., Garenne 2003; Sen 1990, 1992). The results show that both the prenatal environment and child biology are important contributing factors to sex differences in infant mortality, but the effect of biology is much less important than the literature suggests. Unobserved prenatal factors explain 40 %–52 % of male excess mortality, and biology accounts for only 48 %–60 % of this outcome, indicating that conventional methodological approaches overestimate the effect of the latter.

The results reveal little interaction between biology and some observed environmental factors in determining this outcome. These environmental factors include birth order within a twin pair (a proxy for intrauterine environment), social status, and climate. I find that a twin is more likely to be male if he is the firstborn, born to an educated mother, or born in certain climatic conditions. Twin firstborns have a greater chance of surviving through infancy. Male firstborns are more likely to survive than female firstborns, but only during the neonatal period. However, the interactions between biology and climate or between biology and social status do not affect infant mortality.

[1] *Offspring sex ratio*, which is the ratio of male children to female children born to a parent, should not be confused with *population sex ratio* (at birth), which is the ratio of male children to female children born in a population. The world population sex ratio is estimated to be about 1.05, but offspring sex ratio varies widely across parents and population subgroups. This article is mostly concerned with offspring sex ratio.

[2] By *preconception environment*, I mean factors that are external to a child and that occur around the time of conception. These factors might be pure environmental hazards (such as parental exposure to chemicals) or medical factors (such as parental illnesses). Throughout the article, I use the terms *preconception environment* and *prenatal environment* interchangeably.

To the best of my knowledge, the current study represents the first attempt to estimate the distinct effects of biology and preconception environment on sex differences in infant mortality using data on twins. This study also makes a data contribution: it is the first to extract data on twins from the DHS to carry out such an analysis.

The remainder of this article is organized as follows. The second section reviews the role of child biology in sex differences in mortality and lays the groundwork for the main hypothesis that preconception environment is also a contributing factor. The exposition of the methodology follows in the third section. The fourth section discusses the data used for the analysis, and the fifth section contains the main findings. The interaction between biology and some observed environmental factors is explored in the subsequent section. In the final section, I discuss the study and offer conclusions.

## The Theory

In this section, I review the role of biology in the determination of sex differences in mortality. I also review the effects of preconception environment on offspring sex ratio, which lays the groundwork for the main hypothesis that preconception environment is also instructive in explaining the sex gap in mortality.

### 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, 1909). Waldron (1983) explained that XY chromosomes, which are present in males, are more susceptible to X-linked recessive disorders than are XX chromosomes, which are present in females; thus, male children are less likely to be healthy than their female counterparts (see also Waldron 1985, 1998). 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; Bouman et al. 2005; Chao 1996).[3]

This biological literature implies that when male and female children are treated equally, male children should suffer a higher incidence of infectious and noninfectious diseases and thus 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, their weaker immune system, or biological makeup requires further investigation. In fact, while females have a stronger immune

[3] Clearly, sex differences in mortality cannot be solely the result of the sex chromosomes XY and XX. A human cell has 22 homologous chromosome pairs in addition to the sex chromosomes, and interactions between them likely play a role in determining mortality. I therefore view sex chromosomes as well as their likely interactions with non–sex chromosomes as being entirely part of the biological process hypothesized to explain male-emale differences in mortality.

system, they also suffer a higher incidence of autoimmune diseases compared with males (Ansar Ahmed et al. 1985; Bouman et al. 2005; Chao 1996). Analyzing national data from the World Health Organization, Garenne (1992) found that mortality from measles is higher among females than among males. Similarly, Preston (1976) found excess female mortality from certain diseases, including tuberculosis at ages 5–29 years, influenza-pneumonia-bronchitis at ages 5–14 years, and certain infectious and parasitic diseases at ages 1–14 years. These findings seem to imply that the biological explanation for excess male mortality is inconclusive.

### Preconception Environment and Offspring Sex Ratio

The mechanisms determining offspring sex ratio in humans have been investigated in several studies. Recent studies have shown that the offspring sex ratio is related to parental circumstances and levels of hormones at the time of conception. Levels of parental hormones are in turn related to parental stresses, illnesses, and occupations (James 1994, 1995, 1996, 1997, 1998b, 2001). James (1998b) provided evidence that men with multiple sclerosis or non-Hodgkins lymphoma are more likely to bear female children. Similarly, men engaged in professional diving, and those exposed to the nematocide dibromochloropopane (DBCP), dioxin, borates, vinclozolin, or high-voltage installations bear excesses of daughters. On the contrary, men suffering from prostate cancer or treated with gonadotrophin or methyltestosterone are more likely to produce sons. The male-to-female sex ratio at birth of children born to mothers with type 2 diabetes was estimated to be 1.37 by Moller et al. (1998), and 1.39 by Paterson (1998). Rjasanowski et al. (1998) found that the male-to-female sex ratio at birth of children born to mothers with type 1 diabetes was 0.47.

These findings are consistent with those of many other studies on the effects of parental exposure to environmental toxicants, such as 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), fungicide, trichlorophenate, alcohol, lead, solvents, waste anesthetic gases, and air pollution from incinerators, on sex ratio (see, e.g., Davis et al. 1998; Dimid-Ward et al. 1996; Garry et al. 1996, 2002; Jacobsen et al. 2000; James 1997; Mocarelli et al. 1996, 2000; Moller 1998; Williams et al. 1992). A sex ratio biased toward females has been observed among the offspring of smoking parents. In a study conducted in a community in Japan, Fukuda et al. (2002) found that the male-to-female sex ratio of children born to parents who smoked more than 20 cigarettes per day around the time of conception was 0.823, compared with 1.026 for parents who smoked fewer than 20 cigarettes per day and 1.214 for nonsmoking parents. These numbers were significantly different from the population sex ratio of 1.043 in the study area. Also, Mocarelli et al. (2000) documented that parents exposed to high concentrations of TCDD following a plant explosion in Seveso, Italy, when they were younger than 19 years of age later had significantly more girls than boys, with a male-to-female sex ratio of 0.38.

The determination of offspring sex ratio has also been studied in animals. In a diet-controlled experiment of roe deer, Wauters et al. (1995) found that females fed a high-energy diet had an offspring sex ratio of 3, whereas those that fed a low-energy diet had an offspring sex ratio of 0.72, with both estimates being significantly different from the natural sex ratio of 1. In another diet-controlled experiment, Rivers and Crawford (1974) found that female mice that were fed a low-fat diet were 3 times more likely to give birth to a female than a male, whereas those in the control group

had an offspring sex ratio of 1. This result is consistent with a study by Rosenfeld et al. (2003), who, in another controlled-diet experiment of mice, found that the fraction of male births was 67 % for mothers on a very high-fat diet and only 39 % for mothers on a low-fat diet.[4]

The effects of these preconception environmental factors on the offspring sex ratio are large enough to constitute a source of bias in studies examining the determinants of sex differences in child outcomes after birth. The proximal mechanisms are not well understood, but a variety of possibilities have been proposed. There is no testing of proximal mechanisms in this study.

In the next section, I show how my methodological approach overcomes the potential bias introduced by preconception environmental determinants of child sex when estimating the determinants of sex differences in mortality. More precisely, I show how sex differences in mortality can be decomposed into the distinct effects of preconception environment and child biology.

## Model: Estimating Sex Differences in Mortality

The sex gap in mortality is generally estimated in the literature using the following specification:

$$M_{ihyt} = \theta Male_i + \mathbf{X}_{hyt}\pi + \varepsilon_{ihyt}, \quad (1)$$

where $M_{ihyt}$ is a dummy variable indicating whether child *i,* born to parents *h* in year *y*, died at time *t* ($M_{ihyt}$=1 if *i* died at time *t*, and 0 otherwise); $Male_i$ is a dummy indicator for whether child *i* is male; $\mathbf{X}_{yht}$ is a vector of observed parental and household characteristics thought to be correlated with sex and mortality[5]; and $\varepsilon_{ihyt}$ is an error term that is usually assumed to be uncorrelated with sex. The parameter of interest, θ, which measures the male-female difference in the probability of death, is generally interpreted as the effect of inherent biological differences between boys and girls. This interpretation, however, does not take into account the effect of preconception environment. Also, the assumption made in most studies that $\varepsilon_{ihyt}$ is uncorrelated with child sex is not plausible in view of the aforementioned evidence showing that the sex of a child is determined by preconception factors that might also affect child health and survival. Any estimate of θ that does not address this issue of omitted variable bias is therefore likely to be misinterpreted and biased, although the direction of the bias is difficult to determine.[6] The goal in this study is to overcome this bias. More precisely, I decompose θ into the effects of preconception environment and child biology.

[4] See also Rosenfeld and Roberts (2004) for a review of the literature on the effect of maternal diet on offspring sex ratio.

[5] Note, however, that because sex has traditionally been treated as random, controlling for the vector $\mathbf{X}_{yht}$ is irrelevant in most studies.

[6] Exposures to particular diseases or treatments, for instance, may not only lead to excess male births but also contribute to male mortality. If this is the case, then the share of male excess mortality generally attributed to biology is exaggerated. But if boys are also more likely to be born to parents of higher socioeconomic status, which would also favor their survival, then the share of male excess mortality attributed to biology is underestimated.

Write the error term $\varepsilon_{ihyt}$ as follows:

$$\varepsilon_{ihyt} = u_h + p_{hy} + w_{ihyt},$$

where $u_h$, $p_{hy}$, and $w_{ihyt}$ are, respectively, parental time-invariant unobservables, parental time-variant unobservables, and a child random unobserved shock (not correlated with sex). $u_h$ and $p_{hy}$ are interpreted as parental preconception circumstances.[7] They are correlated with child sex and mortality. I posit that a cross-sectional linear probability model (LPM) estimate of θ captures the additive effects of child biology and preconception factors.[8] The effect of parental time-invariant unobservables can be netted out by comparing the mortality of male and female siblings (opposite-sex children born to the same parents). This is done by estimating a sibling fixed-effect regression as follows.

Let $(i,j)$ be a pair of siblings. Rewriting Eq. (1) for $i$ and $j$ yields, respectively, Eqs. (2) and (2)':

$$M_{ihyt} = \theta_{SFE} Male_i + X_{hyt} \beta + u_h + p_{hy} + w_{ihyt} \tag{2}$$

$$M_{jhy't} = \theta_{SFE} Male_j + X_{hy't} \beta + u_h + p_{hy'} + w_{jhy't}. \tag{2}'$$

Taking (2) – (2)' yields the following:

$$M_{ihyt} - M_{jhy't} = \theta_{SFE}(Male_i - Male_j) + (X_{hyt} - X_{hy't})\beta + p_{hy} - p_{hy'} + w_{ihyt} - w_{jhy't}. \tag{3}$$

$\theta_{SFE}$ measures the difference in the probability of death between male and female siblings. Estimating Eq. (3) using a sibling fixed-effect regression yields an estimate of $\theta_{SFE}$. Note that $\theta_{SFE}$ still includes the effect of parental time-variant factors as long as $p_{hy} - p_{hy'}$, for instance, is correlated with child sex. For example, parental environmental and health circumstances are likely to vary over time, making such a correlation very likely. To net out the effect of preconception factors, I compare a male twin with his female co-twin by estimating a twin fixed-effect regression. Let $(i,-i)$ be a pair of male-female twins. Equation (1) can be rewritten for each of them as follows:

$$M_{ihyt} = Male_i \theta_{TFE} + X_{hyt} \pi + u_h + p_{hy} + w_{ihyt} \tag{4}$$

$$M_{-ihy't} = Male_{-i} \theta_{TFE} + X_{hy't} \beta + u_h + p_{hy'} + w_{-ihy't}. \tag{4}'$$

$\theta_{TFE}$ is the effect of child biology. Because observed parental factors and unobserved preconception factors are the same for a pair of twins (i.e., $X_{hyt} = X_{hy't}$ and $p_{hy} = p_{hy'}$), taking (4) – (4)' yields

[7] Parental prenatal circumstances that determine offspring sex ratios, such as occupation or exposure to dioxin, might vary over time.

[8] Note that if $u_h$ and $p_{hy}$ were observed and controlled in Eq. (1), θ would measure only the effect of male biology.

$$M_{ihyt} - M_{-ihy't} = \theta_{TFE}(Male_i - Male_{-i}) + w_{ihyt} - w_{-ihy't}\,. \quad (5)$$

Estimating (5) yields an estimate of the effect of child biology $\theta_{TFE}$. Subtracting this from the cross-sectional LPM estimate of θ yields an estimate of the effect of preconception environmental factors. I estimate θ and $\theta_{TFE}$ using samples of twins, which allows me to separate out the effects of preconception environment and child biology.

To summarize the methodology, the main assumption is that the sex difference in the probability of death for all twins (denoted A) is the result of the additive effects of child biology and preconception environment.[9] The sex difference in the probability of death estimated from the sample of male-female twin pairs (denoted B) is the effect of biology only.[10] Subtracting B from A thus yields the effect of preconception environment.

## Data

### Data Sources

I use Demographic and Health Surveys (DHS) data collected in 31 sub-Saharan African countries. Information on years of survey is provided in Table 10 in the Appendix. The DHS 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 households at the second stage. Data are collected on characteristics of each household, including its durable assets and facilities (e.g., car, TV, radio, access to clean water, and toilet facilities).

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 is provided on each live birth, including date of birth; whether the birth is a singleton or a multiple birth; whether the child is still alive; and, if dead, when the child died. In this study, I use the file of all live births (Children Recode File) reported by mothers in each survey, for a total of 75 files. I merge these 75 files into a single file. The total sample size of all live births is 1,670,477.

### Twins

I use the DHS Children Recode File to identify and match twins based on (1) whether they were declared as twins by their mother; (2) their mother's identification number; and (3) their month and year of birth. Triplets and quadruplets are dropped from the sample. As shown in Table 1, the sample size

[9] Additivity is consistent with models generally used by biologists and geneticists to disentangle the effects of genetic and environmental factors on health outcomes (e.g., Evans et al. 2002; Neale and Cardon 1992).
[10] Indeed, estimating the effect of child sex on mortality over a sample of opposite-sex twins allows me to control automatically for the effect of preconception environment. This is because within an opposite-sex twin pair, sex is perfectly uncorrelated with preconception environment, since there is exactly 1 male for 1 female.

**Table 1** Population sex ratios at birth of singletons and twins in sub-Saharan Africa

| | Sample Size | % Boys (SD) |
|---|---|---|
| Singletons | 1,619,483 | 50.8 (50.0) |
| All Twins | 50,994 | 50.4 (50.0) |
| Opposite-Sex Twins | 20,154 | 50.0 (50.0) |
| Same-Sex Twins | 30,840 | 50.6 (50.0) |

*Note:* Standard deviations (SD) are in parentheses.

of twins is 50,994, representing 3.05 % of the sample of all live births. Twinning rates vary across sub-Saharan Africa (see Table 10 in the Appendix), but the reasons are not entirely known. The proportion of twins in sub-Saharan Africa is smaller than in the United States, where twins represent 3.2 % of all births (Martin et al. 2009).[11] Male-male, female-female, and male-female twins represent, respectively, 31 %, 30 % and 39 % of all twins.

The proportion of male births is .508 among singletons and .506 among same-sex twins, which suggests that male-female relative differences in fetal death and the prenatal determinants of child sex are similar in the two populations. For the pooled sample of twins and singletons, these figures imply a sex ratio (males to females) at birth of 1.032, which is similar to the figure found by Garenne (2002) using both DHS and World Fertility Surveys.

Turning to the summary statistics of socioeconomic variables (Table 2), note that twins and singletons are comparable along several dimensions, including maternal age, marital status, and education; paternal education; and household wealth. Thus, twins largely mirror the entire population along these characteristics.

With respect to mortality, the probability of dying in the first year is 3.4 times higher for twins than for singletons (305 vs. 90 per thousand). This is not a surprising finding. In the United States, where mortality rates are much lower than in developing countries, twins are 6 times more likely than singletons to die in their first year (Almond et al. 2005). The higher mortality of twins is mainly attributed to low birth weight. But as Table 3 shows, boys are 1.166 (= 0.323 / 0.277) and 1.161 (= 0.097 / 0.083) times more likely to die before their first birthday than girls among twins and singletons, respectively; in relative terms, then, twins and singletons are similar in this respect.

[11] It is well known that twinning rates are high in the United States because of assisted reproduction. Globally, the population of twins was estimated to be 125 million in 2006 (Oliver 2006—about 1.9 % of the world population.

**Table 2** Summary statistics

| Variables | Singletons | | | Twins | | |
|---|---|---|---|---|---|---|
| | *N* | Mean | SD | *N* | Mean | SD |
| Child Is Male | 1,619,483 | 0.508 | 0.500 | 50,994 | 0.504 | 0.500 |
| Maternal Characteristics | | | | | | |
| Age | 1,619,483 | 35.104 | 8.062 | 50,994 | 36.343 | 7.521 |
| Marital status | | | | | | |
| Single | 1,619,432 | 0.022 | 0.148 | 50,994 | 0.015 | 0.122 |
| Married | 1,619,432 | 0.769 | 0.422 | 50,994 | 0.771 | 0.420 |
| Widowed | 1,619,432 | 0.047 | 0.211 | 50,994 | 0.050 | 0.218 |
| Living with a partner | 1,619,432 | 0.097 | 0.296 | 50,994 | 0.096 | 0.295 |
| Not living with a partner | 1,619,432 | 0.034 | 0.181 | 50,994 | 0.037 | 0.188 |
| Divorced or separated | 1,619,432 | 0.031 | 0.173 | 50,994 | 0.030 | 0.172 |
| Education | | | | | | |
| Not educated | 1,619,404 | 0.554 | 0.497 | 50,990 | 0.558 | 0.497 |
| Primary | 1,619,404 | 0.335 | 0.472 | 50,990 | 0.335 | 0.472 |
| Secondary or higher | 1,619,404 | 0.111 | 0.314 | 50,990 | 0.107 | 0.309 |
| Father's Education | | | | | | |
| Not educated | 1,548,881 | 0.579 | 0.494 | 49,038 | 0.580 | 0.493 |
| Primary | 1,540,365 | 0.352 | 0.478 | 48,576 | 0.351 | 0.477 |
| Secondary or higher | 1,512,371 | 0.119 | 0.323 | 47,740 | 0.114 | 0.318 |
| Household Characteristics | | | | | | |
| Household size | 1,619,483 | 7.993 | 4.795 | 50,994 | 8.447 | 4.728 |
| Has electricity (0/1) | 1,519,492 | 0.170 | 0.376 | 47,648 | 0.167 | 0.373 |
| Has radio (0/1) | 1,584,591 | 0.551 | 0.497 | 49,820 | 0.556 | 0.497 |
| Has TV (0/1) | 1,532,985 | 0.126 | 0.332 | 47,972 | 0.122 | 0.327 |
| Has car (0/1) | 1,527,477 | 0.042 | 0.201 | 47,950 | 0.039 | 0.193 |

## Results

### All Twins Versus Opposite-Sex Twins

In this section, I compare sex differences in infant mortality across all twins and opposite-sex twins. Table 3 shows that among all twins, the probability of dying in the first year is 46 per thousand points higher among males than females (323 versus 277 per thousand). Among opposite-sex twins, the male-female difference in mortality is only 27 per thousand points (307 versus 280 per thousand). These figures clearly indicate that preconception environmental factors play an important role in the determination of sex differences in infant mortality.

Table 4 shows estimated sex differences in infant mortality based on a multivariate framework. The dependent variable is a dummy indicator equal to 1 if the child died before his/her first birthday, and 0 otherwise. This variable is regressed on a

**Table 3** Probability of dying in the first year by sex for all twins, opposite-sex twins, and same-sex twins

| | Boys | | Girls | |
|---|---|---|---|---|
| | Mean | SD | Mean | SD |
| Singletons | .097 | .296 | .083 | .276 |
| All Twins | .323 | .468 | .277 | .447 |
| Opposite-Sex Twins | .307 | .461 | .280 | .449 |
| Same-Sex Twins | .334 | .477 | .274 | .446 |

binary variable equal to 1 if the child is male and 0 if the child is female. In the sample of all twins, when no controls are included (column 1), infant mortality is 47 per thousand points higher among males than among females, confirming the descriptive statistics presented in Table 3. When controls are added (column 2), the male-female difference in mortality drops by only 2 per thousand points to 45 per thousand points.[12]

The existence of unobserved preconception factors that might affect both a child's sex and health, as suggested by the earlier-reviewed literature, implies that the estimates of the sex gap in infant mortality shown in columns 1–2 are likely biased. I correct for this bias by estimating a twin fixed-effect regression, shown in column 3. Infant mortality is now only 27 per thousand points higher for boys than for girls.

As per the methodology, I conclude that sex differences in biology account for 27 per thousand points in higher male than female infant mortality. The effect of preconception environment is obtained by subtracting the effect of biology from the overall sample estimate (in column 2). The calculation implies that preconception factors increase the mortality of boys relative to that of girls by 45 – 27 = 18 per thousand points. It follows from this decomposition that both the preconception environment and child biology are important contributing factors to higher male than female infant mortality, but the effect of biology is much less important than the literature suggests. Indeed, biology would entirely account for the difference of 45 per thousand points in the mortality of boys and girls according to the conventional methodological approach, which would therefore overestimate its effect by 40 %.

## Same-Sex Twins Versus Opposite-Sex Twins

I now compare sex differences in infant mortality in same-sex twins versus opposite-sex twins. This comparison has two motivations. First, prenatal environmental factors determine offspring sex ratio only among same-sex twins,

[12] Controls include the child's year of birth; mother's age at survey, education and marital status; husband's education; household size; possession of household assets and facilities; a linear control for year of survey; and country-year fixed effects.

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

| | (1) | (2) | (3) | (4) | (5) |
|---|---|---|---|---|---|
| Male | 0.047** | 0.045** | 0.027** | 0.059** | 0.056** |
| | (0.004) | (0.004) | (0.005) | (0.005) | (0.000) |
| Number of Observations | 50,994 | 50,994 | 50,994 | 30,840 | 30,840 |
| All Twins | Yes | Yes | Yes | No | No |
| Same-Sex Twins Only | No | No | No | Yes | Yes |
| Twins Fixed Effect | No | No | Yes | No | No |
| Country-Year Fixed Effect | No | Yes | Yes | No | Yes |
| Controls | No | Yes | No | No | Yes |

*Notes:* Controls include child's year of birth; mother's characteristics (age at survey, education, and marital status); husband's education; household's characteristics (household size; and possession of assets, such as car, television, radio, and electricity); a linear control for year of survey and country–survey year fixed effects. Standard errors, shown in parentheses, are corrected for clustering of observations within mothers.

**$p < .01$

given that child sex is perfectly uncorrelated with these factors among opposite-sex twins. Second, the fact that the sex ratio of same-sex twins is very close to that of singletons suggests that the prenatal determinants of offspring sex ratio in these distinct populations are similar; thus, results based on a comparison between same-sex twins and opposite-sex twins are more likely to be generalizable to the larger population of singletons.

Table 4, column 4, shows the estimated effect of being male on infant mortality among same-sex twins. Infant mortality is 59 per thousand points higher for boys than for girls. After other factors are controlled for in column 5, the male-female difference in mortality decreases to 56 per thousand points. Comparing this latter figure to the estimate of the male-female difference in mortality in opposite-sex twins (column 3), I calculate that prenatal environmental factors increase boys' mortality by 29 per thousand points, which is 52 % of the male-female difference in mortality, and is higher than the estimate obtained by comparing all twins with opposite-sex twins. In either case, prenatal environment is an important contributing factor to males' mortality.

## Time-Variant Versus Time-Invariant Parental Factors

I quantify the distinct effects of time-variant and time-invariant unobserved parental factors on sex differences in infant mortality by comparing twin, singleton, and sibling estimates. The results are presented in Table 5. Column 1 shows the estimated effect of sex on infant mortality among singletons. The probability of dying in the first year is 13 per thousand points higher for boys than for girls. In column 2, I control for the sibling fixed effect, thus controlling for fixed parental characteristics. The estimate is not different from that in column 1,

**Table 5** Linear probability model estimates of sex differences in infant mortality based on singletons and twins

| | (1) | (2) | (3) | (4) |
|---|---|---|---|---|
| Male | 0.013** | 0.013** | 0.052* | 0.050* |
| | (0.000) | (0.000) | (0.022) | (0.022) |
| Number of Observations | 1,619,483 | 1,619,483 | 1,982 | 1,982 |
| Same-Sex Twins | No | No | Yes | Yes |
| Singletons | Yes | Yes | No | No |
| Sibling Fixed Effect | No | Yes | No | Yes |

*Notes:* Standard errors, shown in parentheses, are corrected for clustering of observations within siblings. Column 3 compares the mortality of a male from a male-male twin pair to that of a female from a female-female twin pair, and column 4 compares the mortality of a male from a male-male twin pair to that of a female sibling from a female-female twin pair.

*$p < .05$; **$p < .01$

suggesting that time-invariant parental factors do not matter in determining sex differences in mortality.[13]

I replicate the analysis in columns 1–2 on same-sex twins: male-male and female-female twin pairs. When sibling fixed effects are controlled for, this sample restriction ensures that a male in a male-male twin pair is compared with a female sibling in a female-female twin pair. In column 3, I regress infant mortality on sex, and find that mortality is 52 per thousand points greater for boys than girls. After controlling for sibling fixed effects in column 4, I find that the coefficient on sex decreases by only 2 per thousand points. This finding suggests that time-variant or pregnancy-specific factors matter most for sex differences in mortality, confirming the conclusion reached for singletons. It is consistent with the fact that most determinants of offspring sex ratio (e.g., smoking) are likely to vary over time for an individual.

## Intrauterine Environment, External Environment, and Biology-Environment Interactions

So far, I have relied on an additive model to disentangle the distinct effects of environment and biology on sex differences in infant mortality, without any consideration of potential interactions between those factors. In this section, I estimate the interacting effects of biology and environment, focusing on proxies for intrauterine and external environmental factors that are likely to be correlated with the sex of a child. These factors include birth order, climate, and maternal education as a measure of parental social status. Birth order has been used as a proxy for intrauterine environment. Indeed, twins do not have the same position in the womb, which

[13] However, I cannot fully ascertain that children born at different times to the same mother also have the same father, which implies that the sibling fixed-effect regressions mostly control for time-invariant maternal factors.

implies that they are exposed to different intrauterine environmental conditions. Moreover, it has been shown that twin firstborns have a higher survival probability than twin second-borns (e.g., Buekens and Wilcox 1993; Smith et al. 2002), strongly suggesting that firstborns enjoy more favorable intrauterine environmental conditions than second-borns.

Climate and maternal education proxy external environments. These factors have been shown to affect offspring sex ratio (James 1998b; Mysterud et al. 2000). Climate is measured by rainfall variability in the period 1960–1993. The information on rainfall is provided by the Sustainable Development Department of the Food and Agriculture Organization (http://www.fao.org/), which has identified eight major climate zones for African Countries.[14] I categorize my data into climate zones, using only countries for which data are available, and obtain the first seven groups.

To assess the role of these factors on offspring sex ratio in the data, in Table 6, I regress a binary indicator for whether the child is a male on birth order within a twin pair (columns 1 and 5), climate zones (columns 2 and 6), and maternal education (columns 3 and 7) using different subsamples, further controlling for all these variables in columns 4 and 8.[15] I find that twin firstborns are more likely to be male. With respect to climate, the proportion of boys in the Sahel and Sudan climatic zone is .507, corresponding to a sex ratio of 1.03, the population sex ratio of sub-Saharan Africa. I therefore consider that group of countries to be the reference. Compared with this reference group, a population sex ratio biased toward males is observed in the East and the West Gulf of Guinea, whereas a population sex ratio biased toward girls is observed in the Central Gulf of Guinea and the Great Lakes region. The analysis also confirms that boys are more likely to be born to educated mothers.

It is not clear whether these environmental determinants of sex ratio interact with child biology in determining sex differences in mortality. To answer this question, in Table 7, I regress infant mortality on child sex, each of the environmental factors just covered, and an interaction term between the two variables, controlling for other factors. These variables are included incrementally. I restrict the analysis to the sample of opposite-sex twins, given that the estimation of the effect of child biology on sex differences in mortality is based only on this sample. I find that infant mortality is 47 per thousand points lower in twin firstborns than in twin second-borns (column 1), which is consistent with earlier findings by Smith et al. (2002) and Buekens and Wilcox (1993). Furthermore, male firstborns have a higher survival rate

[14] Those eight zones are (1) Sahel and Sudan (Burkina Faso, Cape Verde, Chad, Gambia, Guinea-Bissau, Mali, Mauritania, Niger, Senegal, and Sudan); (2) southern-central Africa and Madagascar (Madagascar, Malawi, Mozambique, Namibia, Zambia, and Zimbabwe); (3) Central Gulf of Guinea countries and Tanzania (Benin, Côte d'Ivoire, Ghana, Tanzania, and Togo); (4) East and West Gulf of Guinea (Cameroon, Central African Republic, Equatorial Guinea, Gabon, Guinea, Liberia, Nigeria, and Sierra Leone); (5) southern Africa (Botswana, Lesotho, South Africa, and Swaziland); (6) Horn of Africa and Kenya (Djibouti, Ethiopia, Kenya, and Somalia); (7) Great Lakes countries (Burundi, Rwanda, and Uganda); and (8) central-west Africa (Angola, Congo, and Democratic Republic of Congo).

[15] Analyzing the effect of birth order on child sex using all twins (Table 6, column 1) likely understimates the effect of this variable because only the variation generated by the sample of opposite-sex twins is being used in this estimation. It is therefore better to estimate the effect of birth order using the sample of opposite-sex twins, as I do in column 5. Similarly, estimating the effects of climate and parental social status on child sex using the entire sample (columns 2–3) is likely to underestimate the true effects of these variables, given that variations are obtained only from same-sex twins. I therefore also estimate the effects of these variables using only the sample of same-sex twins (columns 6–8).

**Table 6** Linear probability model estimates of the effects of birth order, climate zone, and maternal education on child sex (the dependent variable is a binary indicator for being male)

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
|---|---|---|---|---|---|---|---|---|
| Firstborn | 0.058** | | | 0.058** | 0.146** | | | |
| | (0.004) | | | (0.004) | (0.007) | | | |
| Africa 2 | | −0.002 | | −0.004 | | −0.004 | | −0.006 |
| | | (0.006) | | (0.006) | | (0.008) | | (0.008) |
| Africa 3 | | −0.01 | | −0.011† | | −0.017* | | −0.018* |
| | | (0.007) | | (0.007) | | (0.009) | | (0.009) |
| Africa 4 | | 0.015* | | 0.013† | | 0.027* | | 0.023* |
| | | (0.008) | | (0.008) | | (0.010) | | (0.010) |
| Africa 5 | | −0.008 | | −0.011 | | −0.013 | | −0.018 |
| | | (0.017) | | (0.017) | | (0.022) | | (0.022) |
| Africa 6 | | 0.002 | | 0.001 | | 0.004 | | 0.002 |
| | | (0.009) | | (0.009) | | (0.011) | | (0.011) |
| Africa 7 | | −0.022* | | −0.023* | | −0.036* | | −0.037* |
| | | (0.009) | | (0.009) | | (0.012) | | (0.012) |
| Mother Has Secondary Education | | | 0.016* | 0.014† | | | 0.027* | 0.023* |
| | | | (0.007) | (0.007) | | | (0.009) | (0.009) |
| Number of Observations | 50,994 | 50,994 | 50,990 | 50,990 | 20,154 | 30,840 | 30,836 | 30,836 |
| All Twins | Yes | Yes | Yes | Yes | No | No | No | No |
| Same-Sex Twins Only | No | No | No | No | No | Yes | Yes | Yes |
| Opposite-Sex Twins Only | No | No | No | No | Yes | No | No | No |

*Notes:* Standard errors are shown in parentheses. The different climate zones are obtained based on rainfall variability in the period 1960–1993, and are provided by the Sustainable Development Department of the Food and Agriculture Organization. In the data used here, those zones are the following: **Sahel and Sudan (Africa 1):** Burkina Faso, Chad, Guinea-Bissau, Mali, Mauritania, Niger, Senegal, and Sudan; **southern-central Africa and Madagascar (Africa 2):** Comoros, Madagascar, Malawi, Mozambique, Zambia, and Zimbabwe; **Central Gulf of Guinea countries and Tanzania (Africa 3):** Benin, Côte d'Ivoire, Ghana, Tanzania, and Togo; **East and West Gulf of Guinea (Africa 4):** Cameroon, Central African Republic, Gabon, Liberia, and Nigeria; **southern Africa (Africa 5):** Lesotho and South Africa; **Horn of Africa and Kenya (Africa 6):** Ethiopia and Kenya; **Great Lakes countries (Africa 7):** Burundi, Rwanda, and Uganda.

†$p < .10$; *$p < .05$; **$p < .01$

than female firstborns (columns 2–3). However, this is true during the neonatal period (i.e., the first month after birth) (Table 8), but not during to the postneonatal period (i.e., between months 1 and 12) (Table 9). This finding implies that for most of the first year after birth, sex and birth order do not interact in determining mortality. As for climate, I find that the probability of dying in the first year varies significantly across climate zones (column 4), but there is no interaction between sex and climate (columns 5–6). Finally, in column 7, I find that children born to educated mothers have a higher survival rate. However, the interaction between child sex and maternal

**Table 7** Linear probability model estimates of the interacting effects of child sex and birth order, climate zone, and maternal education on infant mortality

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
|---|---|---|---|---|---|---|---|---|---|
| Male | 0.034** | 0.018* | 0.015† | 0.027** | 0.041** | 0.041** | 0.027** | 0.027** | 0.027** |
| | (0.006) | (0.009) | (0.009) | (0.006) | (0.013) | (0.013) | (0.006) | (0.007) | (0.007) |
| Firstborn | −0.047** | −0.063** | −0.066** | | | | | | |
| | (0.006) | (0.009) | (0.009) | | | | | | |
| Male × Firstborn | | 0.032* | 0.038** | | | | | | |
| | | (0.013) | (0.013) | | | | | | |
| Africa 2 | | | | −0.044** | −0.038** | −0.045 | | | |
| | | | | (0.009) | (0.013) | (0.061) | | | |
| Africa 3 | | | | 0.046** | −0.032* | 0.082 | | | |
| | | | | (0.009) | (0.013) | (0.055) | | | |
| Africa 4 | | | | 0.064** | −0.063** | 0.111* | | | |
| | | | | (0.010) | (0.015) | (0.055) | | | |
| Africa 5 | | | | 0.070* | −0.04 | 0.042 | | | |
| | | | | (0.023) | (0.033) | (0.050) | | | |
| Africa 6 | | | | 0.060** | −0.05** | 0.022 | | | |
| | | | | (0.013) | (0.018) | (0.056) | | | |
| Africa 7 | | | | 0.014 | 0.001 | 0.041 | | | |
| | | | | (0.014) | (0.020) | (0.052) | | | |
| Male × Africa 2 | | | | | −0.014 | −0.014 | | | |
| | | | | | (0.018) | (0.018) | | | |
| Male × Africa 3 | | | | | −0.028 | −0.028 | | | |
| | | | | | (0.019) | (0.019) | | | |
| Male × Africa 4 | | | | | −0.002 | −0.002 | | | |
| | | | | | (0.022) | (0.021) | | | |
| Male × Africa 5 | | | | | −0.061 | −0.061 | | | |
| | | | | | (0.046) | (0.046) | | | |
| Male × Africa 6 | | | | | −0.019 | −0.019 | | | |
| | | | | | (0.026) | (0.025) | | | |
| Male × Africa 7 | | | | | −0.031 | −0.031 | | | |
| | | | | | (0.028) | (0.028) | | | |
| Mother Has Secondary Education | | | | | | | −0.118** | −0.120** | −0.035* |
| | | | | | | | (0.010) | (0.015) | (0.016) |
| Male × Secondary Education | | | | | | | | 0.003 | 0.003 |
| | | | | | | | | (0.021) | (0.020) |
| Number of Observations | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 |
| Controls | No | No | Yes | No | No | Yes | No | No | Yes |

*Notes:* Controls are those listed in Table 4. Standard errors, shown in parentheses, are corrected for clustering of observations within mothers.

†$p < .10$; *$p < .05$; **$p < .01$

education has no effect on mortality (columns 8–9). When replicating the analysis for neonatal and postneonatal mortality separately, I still find that the interactions between biology and climate and between biology and social status have no significant effect (Tables 8 and 9). Apart from being interesting on their own, these different results, which show little interaction between biology and environment, lend support to the additive model used to estimate the distinct effects of these factors.

## Discussion and Conclusions

I proposed the "preconception origins hypothesis" to explain sex differences in infant mortality. This hypothesis complements the biological hypothesis according to which the excess mortality of male children is due to their weaker biological makeup. My methodology for decomposing sex differences in infant mortality into the distinct effects of preconception environment and child biology accounts for the fact that the *offspring sex ratio* is not random, but instead is partly determined by preconception factors that also affect child health and survival in utero and after birth. My approach overcomes the bias in previous estimates of the effect of child biology on sex differences in mortality. More precisely, using large samples of twins from sub-Saharan Africa, I found that unobserved factors in the preconception environment increase the mortality risk of male children, and that the conventional methodological approach overestimates the effect of biology by 40 %–52 %. I also found that twin firstborns have a higher survival probability than twin second-borns, but the interaction between birth order and child sex has no significant effect on infant mortality in most months of the first year after birth. Furthermore, boys are more likely than girls to be born in certain climatic conditions and to educated mothers, but I found little interaction between those external environmental factors and child biology. My findings suggest that biology may be best viewed as a mechanism, not the (distal) cause, of sex disparities in infant survival, which is consistent with other studies viewing low birth weight and other aspects of child health to be reflective of social processes.

My estimates rely on comparing sex differences in mortality across all twins, same-sex twins, and male-female twins. Same-sex twins are often identical, while male–female twins may be mostly fraternal.[16] Identical twins often have perinatal problems (e.g., resulting from sharing the same placenta) that fraternal twins do not have, which often results in lower birth weight in the former. In this study, I focused most of my attention on the *preconception* environment, which precedes intrauterine environment and which, because it determines child sex, can be viewed as a distal determinant of monozygosity among same-sex twins. It will therefore be interesting in future research to determine how much of the effect of the preconception environment is mediated by monozygosity. The data I used, like most data, do not contain information on monozygosity.

Also, as with most analyses based on twins, my analysis is likely to suffer from a sample selection issue. To address this concern, I compared twins and

[16] The notion that opposite-sex twins are always fraternal has been challenged in recent studies showing that such twins could be identical (see, e.g., Wachtel et al. 2000).

**Table 8** Linear probability model estimates of the interacting effects of child sex and birth order, climate zone, and maternal education on neonatal mortality

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
|---|---|---|---|---|---|---|---|---|---|
| Male | 0.026** | 0.005 | 0.004 | 0.022** | 0.03* | 0.03* | 0.022** | 0.022** | 0.022** |
| | (0.006) | (0.008) | (0.008) | (0.006) | (0.011) | (0.011) | (0.006) | (0.006) | (0.006) |
| Firstborn | −0.031** | −0.052** | −0.053** | | | | | | |
| | (0.006) | (0.008) | (0.008) | | | | | | |
| Male × Firstborn | | 0.043** | 0.045** | | | | | | |
| | | (0.011) | (0.011) | | | | | | |
| Africa 2 | | | | −0.042** | −0.038** | −0.014 | | | |
| | | | | (0.008) | (0.011) | (0.053) | | | |
| Africa 3 | | | | −0.02* | −0.009 | 0.086† | | | |
| | | | | (0.008) | (0.012) | (0.048) | | | |
| Africa 4 | | | | −0.028* | −0.028* | 0.117* | | | |
| | | | | (0.009) | (0.013) | (0.048) | | | |
| Africa 5 | | | | −0.063* | −0.043 | 0.036 | | | |
| | | | | (0.020) | (0.028) | (0.044) | | | |
| Africa 6 | | | | −0.029* | −0.022 | 0.071 | | | |
| | | | | (0.011) | (0.016) | (0.049) | | | |
| Africa 7 | | | | 0.004 | 0.008 | 0.066 | | | |
| | | | | (0.012) | (0.017) | (0.045) | | | |
| Male × Africa 2 | | | | | −0.007 | −0.007 | | | |
| | | | | | (0.016) | (0.016) | | | |
| Male × Africa 3 | | | | | −0.022 | −0.022 | | | |
| | | | | | (0.016) | (0.016) | | | |
| Male × Africa 4 | | | | | 0.000 | 0.000 | | | |
| | | | | | (0.019) | (0.019) | | | |
| Male × Africa 5 | | | | | −0.04 | −0.04 | | | |
| | | | | | (0.040) | (0.040) | | | |
| Male × Africa 6 | | | | | −0.013 | −0.013 | | | |
| | | | | | (0.022) | (0.022) | | | |
| Male × Africa 7 | | | | | −0.007 | −0.007 | | | |
| | | | | | (0.024) | (0.024) | | | |
| Mother Has Secondary Education | | | | | | | −0.066** | −0.066** | −0.018 |
| | | | | | | | (0.009) | (0.013) | (0.014) |
| Male × Secondary Education | | | | | | | | 0.000 | 0.000 |
| | | | | | | | | (0.018) | (0.018) |
| Number of Observations | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 | 20,154 |
| Controls | No | No | Yes | No | No | Yes | No | No | Yes |

*Notes:* Controls are those listed in Table 4. Standard errors, shown in parentheses, are corrected for clustering of observations within mothers.

†$p < .10$; *$p < .05$; **$p < .01$

**Table 9** Linear probability model estimates of the interacting effects of child sex and birth order, climate zone, and maternal education on postneonatal mortality

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) |
|---|---|---|---|---|---|---|---|---|---|
| Male | 0.013* | 0.017* | 0.015† | 0.01† | 0.02† | 0.02† | 0.01† | 0.01† | 0.01† |
| | (0.005) | (0.008) | (0.007) | (0.005) | (0.010) | (0.010) | (0.005) | (0.006) | (0.005) |
| Firstborn | −0.025** | −0.021* | −0.024** | | | | | | |
| | (0.005) | (0.007) | (0.007) | | | | | | |
| Male × Firstborn | | −0.008 | −0.002 | | | | | | |
| | | (0.011) | (0.010) | | | | | | |
| Africa 2 | | | | −0.011 | −0.006 | −0.036 | | | |
| | | | | (0.007) | (0.010) | (0.049) | | | |
| Africa 3 | | | | −0.036** | −0.029* | 0.012 | | | |
| | | | | (0.008) | (0.011) | (0.045) | | | |
| Africa 4 | | | | −0.05** | −0.048** | 0.015 | | | |
| | | | | (0.009) | (0.012) | (0.045) | | | |
| Africa 5 | | | | −0.019 | −0.003 | 0.015 | | | |
| | | | | [0.018) | (0.026) | (0.040) | | | |
| Africa 6 | | | | −0.043** | −0.037* | −0.044 | | | |
| | | | | (0.011) | (0.015) | (0.046) | | | |
| Africa 7 | | | | −0.022† | −0.006 | −0.016 | | | |
| | | | | (0.012) | (0.016) | (0.042) | | | |
| Male × Africa 2 | | | | | −0.01 | −0.01 | | | |
| | | | | | (0.015) | (0.015) | | | |
| Male × Africa 3 | | | | | −0.013 | −0.014 | | | |
| | | | | | (0.016) | (0.015) | | | |
| Male × Africa 4 | | | | | −0.005 | −0.006 | | | |
| | | | | | (0.018) | (0.018) | | | |
| Male × Africa 5 | | | | | −0.032 | −0.033 | | | |
| | | | | | (0.037) | (0.037) | | | |
| Male × Africa 6 | | | | | −0.011 | −0.011 | | | |
| | | | | | (0.021) | (0.021) | | | |
| Male × Africa 7 | | | | | −0.032 | −0.033 | | | |
| | | | | | (0.023) | (0.023) | | | |
| Mother Has Secondary Education | | | | | | | −0.071** | −0.071** | −0.02 |
| | | | | | | | (0.008) | (0.011) | (0.013) |
| Male × Secondary Education | | | | | | | | 0.001 | 0.001 |
| | | | | | | | | (0.016) | (0.016) |
| Number of Observations | 16,312 | 16,312 | 16,312 | 16,312 | 16,312 | 16,312 | 16,312 | 16,312 | 16,312 |
| Controls | No | No | Yes | No | No | Yes | No | No | Yes |

*Notes:* Controls are those listed in Table 4. Standard errors, shown in parentheses, are corrected for clustering of observations within mothers.

†$p < .10$; *$p < .05$; **$p < .01$

singletons along several observable characteristics (Table 2) and found little difference between the two samples. I also found the proportion of males among same-sex twins and singletons to be very close (.506 vs. .508), suggesting that the preconception determinants of sex, most of which cannot be observed in these data, are similar for the two populations. Twins therefore largely mirror the general population and cannot be regarded as being selected along those observable and unobservable characteristics.

In results not shown, I also determined whether the sample of opposite-sex twins is selected. For this purpose, I considered parents who have twins and singletons, and examined whether the propensity to bear singleton males (or females) affects the probability that a twin pair is male-female. I measured the propensity of a parent to have singleton males as the proportion of singleton males that he/she has; a value of zero means that all singleton children are female, and a value of 1 means that all singleton children are male. I found that this variable has no effect on the probability of a twin pair being male-female (the coefficient is .000), which suggests that opposite-sex twins are "randomly" distributed across parents and hence are not selected with respect to the preconception determinants of a baby's sex. This result provides additional support for my comparisons of sex differences in infant mortality across all twins, same-sex twins, and opposite-sex twins.

I also showed that infant mortality is much higher for twins than for singletons, raising the issue of whether my results are generalizable. In relative terms, males are 1.166 (= .323 / .277) and 1.161 (= .097 / .083) times more likely to die in their first year than females among twins and singletons, respectively. Obviously, these figures are very similar. Also, although it seems quite plausible that intrauterine mortality of twins in Africa might be substantially higher than that of singletons, the similarity of the sex ratio of same-sex twins with that of singletons in the general population implies that intrauterine mortality is not sex-specific. In addition, because twins cannot be viewed as a selected population along several important characteristics, as discussed earlier, one could argue that the findings are generalizable to a certain extent, but only in relative terms. That is, the finding that 40 %–52 % of sex differences in infant mortality are attributable to preconception factors and only 48 %–60 % to biological factors among twins is generalizable to singletons. Furthermore, the sibling fixed-effect estimates, which control for maternal fixed factors but not for pregnancy-specific factors, imply that most of the preconception factors responsible for higher male than female infant mortality are pregnancy-specific or time-variant.

An alternative explanation of our empirical findings might be inspired by the literature on intrauterine hormone influences in opposite-sex twin pairs (see, e.g., Hines and Collaer 1993; Miller 1994). This literature argues that girls in opposite-sex twin pairs receive a higher dose of androgen or male hormones in utero, as compared to either singleton girls or girls in same-sex twin pairs, and that this results in slightly more "masculine" girls. Similarly, there might be some "feminization" of boys following exposure to estrogen hormones released by their twin sisters. While the hormone transfer theory has caught the interest of several biologists and behavioral scientists, it has not always been validated empirically (see, e.g., Rodgers et al. 1998). If the mechanism proposed by this theory is important in this setting, then the fixed-effect estimates based on opposite-sex twins provide an incorrect estimate of the population male-female

difference in mortality. Although the lower male mortality and higher female mortality in opposite-sex twins (shown in Table 3) is consistent with such a mechanism, the effect is not strong enough to change the main interpretation of my results; the mortality difference between girls in female-female twin pairs and girls in opposite-sex twin pairs is very small and is not statistically significant. Also, in results not shown, I found that in the Sahel and Sudan climate zone (Africa 1 in Table 6), the probability of dying in the first year is higher for boys in male-female twin pairs than boys in male-male twin pairs, and lower for girls in male-female twin pairs than girls in female-female twin pairs; those differences are all statistically significant. These findings do not seem to support the alternative explanation of our empirical results based on hormone influences in opposite-sex twin pairs.

To conclude, it seems important to note that from a theoretical perspective, my methodology for decomposing sex differences in mortality into the effects of preconception environment and child biology does not imply that preconception environmental factors always increase the mortality of male children. It simply offers guidance as to how to compare and interpret estimates of the sex difference in mortality estimated over a sample of all twins ($\hat{\theta}$), sibling fixed-effect estimates ($\hat{\theta}_{SFE}$), and twin fixed-effect estimates ($\hat{\theta}_{TFE}$). Empirically, I found $\hat{\theta}_{TFE}$ to be smaller than $\hat{\theta}$, implying that preconception environment plays a role in higher male than female infant mortality. A finding that $\hat{\theta}_{TFE}$ is equal to $\hat{\theta}$ would not have meant that preconception environment doesn't matter, but it would have meant that its effect on boys and girls is the same. Finally, a finding that $\hat{\theta}_{TFE}$ is greater than $\hat{\theta}$ would have implied that preconception environment increases the mortality of girls relative to that of boys. I also found $\hat{\theta}$ and $\hat{\theta}_{SFE}$ to be very similar, implying that time-variant parental factors in the prenatal environment play a more important role than time-invariant factors in determining sex differences in mortality. Nothing in my study indicates that it is impossible to find $\hat{\theta}_{TFE} = \hat{\theta}$ or $\hat{\theta}_{TFE} > \hat{\theta}$, or $\hat{\theta} \neq \hat{\theta}_{SFE}$ in other settings or contexts, especially given that environmental factors are likely to vary substantially across countries and continents, as well as over time. In this respect, it would be premature to claim that my empirical results based on data from sub-Saharan Africa generalize, for instance, to the United States. More caution is warranted in interpreting the empirical findings. However, my framework, which I view as a theoretical contribution, can certainly be used beyond the African context, especially in regions such as Europe and the United States, where there is little parental or societal discrimination against girls or boys in the allocation of resources. Also, I view this study as making a data contribution: it is the first to extract data on twins from Demographic and Health Surveys to study the role of biology and environment in determining an important demographic outcome. A similar approach can be followed to study a variety of child outcomes using the DHS.

Understanding the origins of sex imbalance in mortality is essential in designing policies that will efficiently address this crucial issue. Sex differences in biology have long been advanced as the main explanation for the excess mortality of male children in nondiscriminatory societies, leaving the impression that little could be done to improve their survival. The demonstrated role of preconception environment in male excess mortality means that actions can be taken to solve this problem.

**Acknowledgments** I am grateful to the Editor and three anonymous referees for insightful comments and suggestions that have helped improve the paper. I also thank Anna Aizer, Jason Boardman, Ken Chay, Jakina Debnam, Parfait Eloundou-Enyegue, Andrew Foster, Jeffrey Greenbaum, Blessing Mberu, Mark Pitt, Marie Patience Pongou, Yona Rubinstein, Olumide Taiwo, and Valerie Wilson for encouragement and useful comments on earlier versions of this article. I acknowledge the generous financial support of the Brown University Population Studies and Training Center, the Compton Foundation, the Hewlett Packard Foundation, and the University of Ottawa.

## Appendix

**Table 10** Sample size by country

| Countries | Years of Survey | Total Sample Size of Live Births | Sample Size of Twins | Sample Size of Singletons |
|---|---|---|---|---|
| Benin | 1996, 2001 | 38,703 | 1,880 | 36,823 |
| Burkina Faso | 1992/1993, 1998/1999, 2003 | 84,278 | 2,520 | 81,758 |
| Burundi | 1987 | 11,880 | 198 | 11,682 |
| Cameroon | 1994, 1998, 2004 | 56,218 | 2,116 | 54,102 |
| Central African Republic | 1994/1995 | 16,933 | 444 | 16,489 |
| Chad | 1996/1997, 2004 | 47,175 | 1,350 | 45,825 |
| Comoros | 1996 | 7,907 | 294 | 7,613 |
| Côte d'Ivoire | 1994, 1998/1999, 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-Bissau | 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, 2003/2004 | 61,362 | 1,282 | 60,080 |
| Malawi | 1992, 1996, 2000, 2004 | 92,571 | 3,584 | 88,987 |
| Mali | 1987, 1995/1996, 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, 1992/1993, 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, 2000/2001 | 62,203 | 1,618 | 60,585 |
| Zambia | 1992, 1996, 2001/2002 | 70,702 | 2,334 | 68,368 |
| Zimbabwe | 1988, 1994, 1999, 2005/2006 | 62,818 | 2,078 | 60,740 |

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