Excess Male Infant Mortality: The Gene-Institution Interactions

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Abstract: Excess male mortality at early ages is an important source of child inequality in most societies. It mirrors a significant early-life human capital disadvantage that has long been regarded as a case of genetic fatality. Yet, an unresolved fundamental question is whether this problem can be minimized through appropriate policy interventions. Using data on twins in combination with a natural experiment on the development of African institutions, we quantify the distinct effects of biology and preconception environmental factors on the infant mortality sex gap, and examine how they respond to the quality of political institutions. We find that these effects are important only in poor institutions. The analysis implies that improved institutions constrain genetic expression and mitigate preconception influences on excess male infant mortality, which is an optimistic finding with pragmatic implications.

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1. Introduction

Sex differences in early-age mortality continue to be an important source of child inequality around the world. Among children who died before their fifth birthday between 2005 and 2010 globally, boys outnumbered girls by over 5 million (United Nations, Department of Economic and Social Affairs 2013). Excess male mortality mirrors a significant disadvantage in early-life human capital development that has long been regarded as an instance of biological and genetic fatality (Naeye et al. 1971; Waldron 1983). Perhaps owing to this biological hypothesis, this important issue has been overlooked by researchers and policymakers altogether.[4] Yet, an unresolved fundamental question is whether the male survival disadvantage can be mitigated through appropriate policy interventions. This paper answers this question for the first time.

Addressing this question is important for several reasons. Besides being a fundamental equity problem, the sex gap in early-age mortality leads to a demographic imbalance that has broad social and economic consequences. Excess male mortality at early ages results in a deficit of male adults in many regions of the world, and this has negative consequences for women on the marriage market and their bargaining power in the household (see, e.g., Abramitzky, Delavande and Vasconcelos (2011), Pongou and Serrano (2016)).[5] Also, sex differences in early-life conditions may result in differential economic outcomes later in life, given the well-documented evidence for the effect of early-life circumstances on later-life outcomes (see, e.g., Almond and Currie (2011), Bozzoli, Deaton, and Quintana-Domeque (2009)). These crucial issues however can be minimized if good institutions and policies can mitigate sex differences in early-life morbidity and mortality.

[4] The economics literature on this topic has mostly focused on the effect of discrimination against females in South and East Asia (see, e.g., Sen (1990) and Ebenstein and Leung (2010) and some of the references therein).

[5] Lower female bargaining power also implies that women are more severely punished for sexual infidelity than men in a two-sided fidelity mating economy. Pongou and Serrano (2013) show that this type of discrimination leads to the formation of sexual networks in which sexually transmitted diseases are more prevalent in women than in men.

Our analysis of this question relies on the two available theoretical explanations for excess male mortality at early ages, which are the aforementioned *biological hypothesis* and the *preconception origins hypothesis* (Pongou 2013, 2015). The biological hypothesis affirms that XY chromosomes, which are present in males are more susceptible to X-linked recessive disorders than XX chromosomes, which are present in females. Males are consequently more susceptible to certain diseases and premature death than females (Naeye et al. 1971; Waldron 1983).[6] The preconception origins hypothesis holds that certain preconception environmental factors that determine the sex of a child, such as parental stresses, occupations, diet, exposure to environmental hazards, and medical conditions around the time of conception, also affect child health in utero and after birth, and therefore partly determine sex differences in infant morbidity and mortality (Pongou 2013, 2015). Following these two theories, we quantify the distinct roles of preconception environment and biological factors in excess male infant mortality, and analyze how each of these effects responds to improvement in the quality of political institutions.

Our methodology is a twin-based decomposition approach which compares the mortality sex gap across all twins and opposite-sex twins. It exploits the fact that co-twins are exposed to the same preconception environment. Therefore, in circumstances where there is no gender bias in the allocation of food and other resources likely to impact survival, the difference in mortality between a male and his female co-twin is only due to biological and genetic differences between the sexes. The effect of preconception environment is obtained by subtracting the effect of biology from the total sex difference in mortality estimated over the entire sample of twins.

We apply this methodology to a sample of over 50,000 twins extracted from 75 nationally representative Demographic and Health Surveys conducted in 31 sub-Saharan African countries, a region where boys and girls have been found to receive equal treatment in the allocation of foods,

[6] See Pongou (2015) for a more detailed explanation of this hypothesis as well as a discussion of its limitations.

health care, and other resources likely to affect child survival (Sen 1990). We measure institutional quality using a widely used historical natural experiment on the development of national and regional institutions in sub-Saharan Africa. This experiment has resulted in sub-Saharan Africa being classified into four institutional regions (see Section 3 for a detailed explanation).

Our main finding is that both preconception and biological factors contribute to excess male infant mortality, but these effects become economically and statistically insignificant in relatively good institutions. The analysis therefore implies that improved institutions constrain the expression of genes and mitigate preconception influences on excess male mortality, resulting in an overall smaller mortality sex gap. To the best of our knowledge, our paper is the first to obtain these findings.

The rest of this paper is organized as follows. Section 2 presents our twin-based methodology. Section 3 describes the data. Section 4 presents the main findings, and Section 5 briefly discusses the mechanism and concludes.

2. Methodology: Estimating the Sex Difference in Infant Mortality

We estimate the distinct contributions of child biology and preconception environment to the sex difference in infant mortality using the following general equation, which slightly extends the model in Pongou (2015):

$$M_{ihryt} = \theta Male_i + X_{hyt}\pi + u_h + p_{hy} + v_{ry} + \varepsilon_{ihryt} \quad [1]$$

where M_{ihryt} is a binary variable indicating whether child i, born to parents h in region (or institution) r in year y, died at time t (M_{ihryt} = 1 if i died at time *t (within the first year of life* in this paper*)*, and 0 otherwise); $Male_i$ is a binary variable for whether child i is male; X_{hyt} is a vector of observed parental and household characteristics thought to determine child sex and mortality; u_h captures parental time-invariant unobservables, p_{hy} parental time-variant unobservables, v_{ry} regional unobservables, and

ε_{ihryt} a child random unobserved shock uncorrelated with sex. Our parameter of interest, θ, measures the difference in infant mortality between boys and girls.

We estimate equation [1] using a linear probability model, obtaining an estimate of θ which, conditional on the institutional setting, represents the additive effects of preconception environment and child biology on the sex difference in infant mortality. In order to isolate the effect of biology, we compare the mortality of opposite-sex twins because they have the same preconception environment. This is pertinent in situations where preconception factors are not observed like in most data. Let (*i*,*j*) be a pair of opposite-sex twins. Rewriting equation [1] for each yields the equations:

$$M_{ihryt} = \theta_{TFE} Male_i + X_{hyt}\pi + u_h + p_{hy} + v_{ry} + \varepsilon_{ihyt} \quad [2]$$

$$M_{jhry't} = \theta_{TFE} Male_j + X_{hy't}\pi + u_h + p_{hy'} + v_{ry'} + \varepsilon_{jhy't} \quad [2']$$

Taking [2] – [2'] yields equation [3] below:

$$M_{ihyt} - M_{jhy't} = \theta_{TFE}(Male_i - Male_j) + (X_{hyt} - X_{hy't})\pi + (p_{hy} - p_{hy'}) + (v_{ry} - v_{ry'}) + (\varepsilon_{ihyt} - \varepsilon_{jhy't}) \quad [3]$$

Since *i* and *j* are twins, they are born at the same time (*y*=*y'*), and they have the same parental characteristics ($X_{hyt} = X_{hy't}$), the same institutional exposure ($v_{ry} = v_{ry'}$), and the same exposure to parental time-variant preconception factors ($p_{hy} = p_{hy'}$). Equation (3) therefore reduces to:

$$M_{ihyt} - M_{jhyt} = \theta_{TFE}(Male_i - Male_j) + \varepsilon_{ihyt} - \varepsilon_{jhyt} \quad [4]$$

We estimate θ_{TFE} using a twin fixed effect (TFE) linear probability model. It measures the effect of sex differences in biology on the sex difference in infant mortality. Subtracting the estimate of θ_{TFE} from that of θ yields an estimate of the effect of preconception environment.

3. Data

Twins. We use 75 Demographic and Health surveys (DHS) collected in 31 sub-Saharan African countries during the period 1987-2005. The DHS are nationally representative and administer

standardized questionnaires across years and countries. Information is collected on household and individual characteristics, with selected women also providing information on all their children, their date of birth, their living status, and their date of death if dead. Using the DHS Birth Recode File, we identified and matched twins based on: (*i*) whether they were declared as twins by their mother; (*ii*) their mother's identification number; and (*iii*) their month and year of birth. Details on the surveys used in this paper are in Appendix Table A.1.

Our pooled sample has a total of 50,994 twins, representing 3.05 % of the sample of all live births. Male-male twins represent 31% of the sample, female-female twins 30%, and male-female twins 39 %. The summary statistics of the variables utilized in our analysis are shown in Table 1.

The proportion of male births is 50.6% among like-sex twins and 50.6% among singletons, which represents a male-to-female sex ratio of 1.032. This suggests that twins and singletons do not differ much in terms of the male-female relative differences in foetal death and the preconception determinants of child sex.

A Natural Institutional Experiment. We measure the quality of institutions by exploiting a widely used natural historical experiment on the development of African institutions. Following Acemoglu, Johnson and Robinson (2001), variation in the quality of political institutions across African countries can be partially explained by variation in the degree to which the local disease environment was favorable to European colonizers. In more favorable environments, colonizers settled in greater numbers, also setting up more "inclusive" institutions. Based on this experiment, sub-Saharan African countries can be grouped into four institutional regions: Central Africa, Western Africa, Eastern Africa, and Southern Africa. Colonizers settled more in the latter regions than in the former. Other historical factors explaining why these regions might have developed different institutions include colonization policies, which greatly differed across countries, also depending on the colonizer's identity. Most countries of Central and Western Africa were colonized by the French, whereas those in Eastern and Southern Africa were mostly colonized by the British. Furthermore, the

fact that the countries belonging to each region now form an "official" regional body implies a lower degree of institutional heterogeneity within regions than across them.

To ascertain that the aforementioned regions effectively differ in the quality of their institutions, we consider four classical institutional variables-corruption index, democratic accountability, and law and order- used in the literature on the relationship between institutions and infant mortality (e.g., Zweifel and Navia 2000). Data are from the World Bank Development Indicators and the Political Risk Services/International Country Risk Guide. Figure 1 shows the average value of these variables, coded on a 0-4 scale, over the period between 1960 and 2009 for each region. We note that the four regions can effectively be ranked on the basis of these indicators, with Central Africa and Southern Africa experiencing the worst and the best situations, respectively.

4. Biology, Preconception Environment, and Institutions: Regression-based Results

We apply the regression-based decomposition approach described in Section 2 to distinguish the effects of biological and preconception factors on the male-female gap in infant mortality, and analyze how these effects respond to improvement in the quality of institutions. The findings are presented in Table 2 and summarized in Figure 2 below.

Table 2 has four panels corresponding to the institutional regions. For each panel, Column (1) estimates the total infant mortality sex gap. Column (2) adds controls. Column (3) controls for twin fixed effects, thus estimating the effect of biology. Column (4) estimates the effect of preconception environment by subtracting the effect of biology from the total effect estimated in Column (1).

Mortality is larger for boys than girls in all regions, but the gap is smaller in regions with better institutions; it even loses its statistical significance in Southern Africa (Column (1)). Weaker male biology increases male infant mortality by 29, 32, 23 and 17 per thousand points in Central, Western, Eastern and Southern Africa, respectively. Preconception factors increase it respectively by 44, 16 and

20 per thousand points in the first three regions, but decrease it by 5 per thousand points in Southern Africa, with the latter effect being statistically insignificant.

5. Possible Mechanisms and Conclusions

In results not presented here, we explore several mechanisms for our findings that better institutions mitigate preconception and biological influences on excess male infant mortality. Better institutions translate into higher income and higher public health expenditures, which in turn positively affect health care facilities, child care, and maternal health. We find evidence that boys respond more positively to these factors than girls. Indeed, we find that mothers are taller in Southern Africa than in other sub-Saharan African regions, reflecting higher income and public health expenditures per capita in the former region, and that maternal height has a larger effect on boys' survival. Birthweight also has a larger impact on boys, but birthweight is not greater in Southern Africa than other regions. The findings therefore suggest that better institutions reduce the infant mortality sex gap partially by improving environmental conditions and parental human capital.

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Figure 1: Quality of institutions by sub-Saharan African region

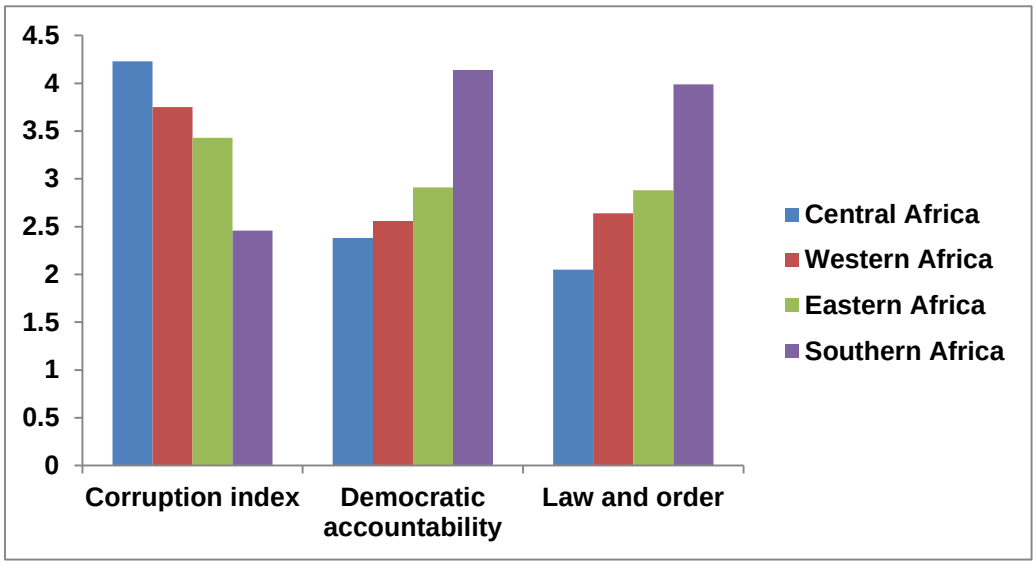
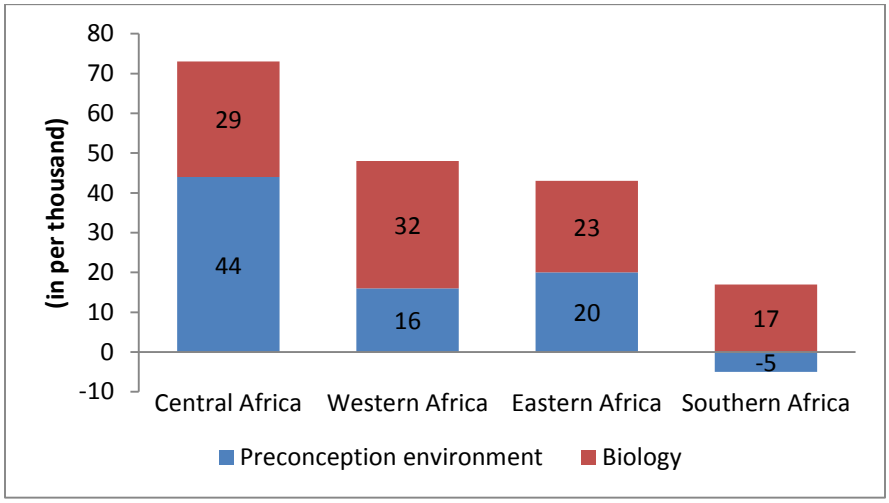


Figure 2: Regression-based decomposition of the infant mortality male-female difference into the distinct effects of biology and preconception environment among twins



Note: Figure 2 summarizes the findings of Table 2. It shows the total sex difference in infant mortality (Column (1) of Table 2) and its decomposition into the distinct effects of biology and preconception environment (Columns (3) and (4)). Clearly, these effects are smaller in regions with improved institutions, and lose their statistical significance in Southern Africa (see Table 2).

Table 1: Summary statistics of twins in sub-Saharan Africa by institutional region

	Eastern Africa			Central Africa			Southern Africa			Western Africa			All twins		
	N	**Mean**	**SD**	***N***	**Mean**	**SD**	***N***	**Mean**	**SD**	***N***	**Mean**	**SD**	***N***	**Mean**	**SD**
Child is Male	23,400	0.499	0.500	4,442	0.520	0.500	1,664	0.484	0.500	21,488	0.507	0.500	50,994	0.504	0.500
Maternal Characteristics															
Age	23,400	36.332	7.585	4,442	35.306	7.746	1,664	37.704	7.258	21,488	36.464	7.398	50,994	36.343	7.521
Marital status															
Single	23,400	0.012	0.111	4,442	0.025	0.155	1,664	0.139	0.346	21,488	0.006	0.079	50,994	0.015	0.122
Married	23,400	0.718	0.450	4,442	0.667	0.471	1,664	0.573	0.495	21,488	0.867	0.340	50,994	0.771	0.420
Widowed	23,400	0.113	0.316	4,442	0.164	0.370	1,664	0.112	0.315	21,488	0.063	0.243	50,994	0.096	0.295
Living with a partner	23,400	0.065	0.246	4,442	0.054	0.225	1,664	0.083	0.276	21,488	0.031	0.175	50,994	0.050	0.218
Not living with a partner	23,400	0.045	0.207	4,442	0.026	0.159	1,664	0.024	0.153	21,488	0.016	0.125	50,994	0.030	0.172
Divorced or separated	23,400	0.047	0.213	4,442	0.064	0.245	1,664	0.069	0.253	21,488	0.016	0.127	50,994	0.037	0.188
Mother's education															
Not educated	23,396	0.406	0.491	4,442	0.443	0.497	1,664	0.167	0.373	21,488	0.778	0.415	50,994	0.558	0.497
Primary	23,396	0.484	0.500	4,442	0.349	0.477	1,664	0.565	0.496	21,488	0.151	0.358	50,994	0.335	0.472
Secondary or higher	23,396	0.110	0.313	4,442	0.208	0.406	1,664	0.268	0.443	21,488	0.070	0.255	50,994	0.107	0.309
Father's Education															
Not educated	21,940	0.260	0.438	4,124	0.371	0.483	1,362	0.310	0.463	19,754	0.700	0.458	47,180	0.455	0.498
Primary	21,940	0.530	0.499	4,124	0.297	0.457	1,362	0.433	0.496	19,754	0.153	0.360	47,180	0.349	0.477
Secondary or higher	21,940	0.211	0.408	4,124	0.331	0.471	1,362	0.257	0.437	19,754	0.147	0.354	47,180	0.196	0.397
Household Characteristics															
Household size	23,400	7.350	3.451	4,442	8.965	5.019	1,664	7.356	4.242	21,488	9.620	5.537	50,994	8.447	4.728
Has electricity (0/1)	22,446	0.110	0.313	4,300	0.313	0.464	1,632	0.268	0.443	19,696	0.200	0.400	48,074	0.170	0.376
Has radio (0/1)	23,106	0.487	0.500	4,300	0.574	0.495	1,634	0.671	0.470	21,206	0.619	0.486	50,246	0.556	0.497
Has TV (0/1)	21,978	0.082	0.274	4,300	0.194	0.396	1,632	0.222	0.416	20,488	0.146	0.353	48,398	0.124	0.329
Has car (0/1)	21,952	0.026	0.159	4,298	0.047	0.213	1,628	0.124	0.330	20,500	0.044	0.206	48,378	0.039	0.193

Table 2: Linear probability model estimates of sex differences in infant mortality based on twins data by institutional region

	Effects of biology + preconception environment	**Effects of biology + preconception environment after Controls**	**Effect of biology**	**Effect of preconception environment**
Panel A: Central Africa	**(1)**	**(2)**	**(3)**	**(4) = (1) – (3)**
Male	0.073***	0.072***	0.029*	0.044**
	(0.013)	(0.013)	(0.015)	(0.019)
Observations	4,442	4,442	4,442	4,442
Panel B: Western Africa				
Male	0.048***	0.044***	0.032***	0.016
	(0.006)	(0.006)	(0.008)	(0.010)
Observations	21,488	21,488	21,488	21,488
Panel C: Eastern Africa				
Male	0.043***	0.041***	0.023***	0.020**
	(0.006)	(0.006)	(0.007)	(0.009)
Observations	23,400	23,400	23,400	23,400
Panel D: Southern Africa				
Male	0.012	0.017	0.017	-0.005
	(0.021)	(0.021)	(0.023)	(0.031)
Observations	1,664	1,664	1,664	1,664
Twin Fixed Effect	**No**	**No**	**Yes**	**-**
Controls	**No**	**Yes**	**Yes**	**-**

Robust standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Note: 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 assets, such as car, television, radio, and electricity); and country-survey-year fixed effects.

ONLINE APPENDIX (NOT TO BE PRINTED)

Table A.1: Classification of African countries into institutional regions and years of survey and sample size for each country

East Africa	Years of Survey	Sample size of twins
Burundi	1987	198
Comoros	1996	294
Ethiopia	2000, 2005	1,740
Kenya	1989, 1993, 1998, 2003	2,572
Madagascar	1992, 1997, 2003/04	1,282
Malawi	1992, 1996, 2000, 2004	3,584
Mozambique	1997, 2003	2,086
Rwanda	1992, 2000, 2005	1,702
Sudan	1990	684
Tanzania	1992, 1996, 2004	3,228
Uganda	1988, 1995, 2000/01	1,618
Zambia	1992, 1996, 2001/02	2,334
Zimbabwe	1988, 1994, 1999, 2005/06	2,078
Central Africa		
Central African Republic	1994/95	444
Cameroon	1994, 1998, 2004	2,116
Chad	1996/97, 2004	1,350
Gabon	2000	532
Southern Africa		
Lesotho	2004	422
Namibia	1992, 2000	684
South Africa	1998	558
Western Africa		
Benin	1996, 2001	1,880
Burkina Faso	1992/93, 1998/99, 2003	2,520
Côte d'Ivoire	1994, 1998/99, 2005	1,486
Ghana	1988, 1993, 1998, 2003	1,890
Guinea-Bissau	1999, 2005	1,900
Liberia	1986	698
Mali	1987, 1995/96, 2001	2,788
Niger	1992, 1998	1,558
Nigeria	1990, 1999, 2003	2,628
Senegal	1986, 1992/93, 1997, 1999, 2005	2,608
Togo	1988, 1998	1,532
Total		50,994