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**WORKING PAPERS**

# Missing Twins: Fetal Origins, Institutions, and Twin-singleton Mortality Convergence

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# Missing Twins: Fetal Origins, Institutions, and Twin-singleton Mortality Convergence

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## Abstract

An important number of twins are missing because of their substantially greater mortality risk in early ages relative to singletons. This paper has a twofold goal. First, it investigates whether, as children age, the twin-singleton inequality in mortality rates vanishes, and if yes, when. Second, it analyzes how the timing of mortality convergence is affected by the quality of political institutions. We use a sample of more than 3 million births from numerous countries in sub-Saharan Africa. Twins represent 3.2% of the sample, and children are followed up to the age of 25. We find that mortality is substantially higher for twins, but the difference persists only to around the age of 5. Importantly, the timing of mortality convergence is shortened by better quality political institutions. The findings yield two major conclusions. First, biology-induced disadvantages can be partially remedied through appropriate policy interventions. Second, the fetal origins hypothesis, which holds that the risk of adult morbidity and mortality is positively affected by intrauterine growth retardation, is not universally valid. In particular, better institutions are likely to offset the short- and long-term consequences of poor intrauterine conditions, attenuating the “missing twins” problem.

*Key words:* Mortality, twins, singletons, convergence, institutions, sub-Saharan Africa.

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## 1. Introduction

Twin-singleton mortality differences in early ages are an important source of child inequality in all societies. The risk of death within the first year of life is higher for twins compared to singletons by a factor ranging from above three in Gambia to five in the United Kingdom and six in the United States.<sup>1</sup> These mortality inequalities do not only mean that a large number of twins are missing<sup>2</sup>, but they also raise the possibility that surviving twins may experience important health and developmental problems which compromise their ability to accumulate human capital as they age. This constitutes a fundamental equity issue, which evidently raises the question of how long the twin-singleton difference in mortality rates persists.

This paper addresses this question for the first time. More precisely, we identify the timing of twin-singleton mortality convergence. In addition, we analyze how the timing of convergence is affected by the quality of political institutions. In this sense, our study contributes to the small but growing literature that seeks to integrate biology into public policy (e.g., Boardman and Fletcher, 2015). This literature tends to show that, contrary to received wisdom, “biology” is not synonymous with “fatality”: biology-induced disadvantages can be partially or entirely remedied through appropriate policy interventions. The analyses also allow us to shed light on the validity of the fetal origins hypothesis, which holds that intrauterine growth retardation significantly increases the risk of adult morbidity and mortality (Almond and Currie, 2011; Almond, Currie,

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<sup>1</sup> The infant mortality risk of twins is five times higher in the United Kingdom (ONS, 2012) and in Sweden (Cheung et al., 2000), 3.46 times higher in Gambia (Miyahara et al., 2016), and six times higher in the United States (Almond, Chay, and Lee, 2005). This gap is observed in all sub-Saharan African countries (Pongou, 2013; Smits and Monden, 2011).

<sup>2</sup> There were about 125 million individuals who were born as twins in the world in 2006 (Oliver, 2006). This figure represents about 1.6% of the world population, which is far below the proportion of twin births in most societies, which is around 3% (Pison et al., 2015; Smits and Monden, 2011). These figures indicate that at least 100 million twins are missing; that is, if there were no differences in mortality rates between twins and singletons, there would be at least 100 million more twins than the world currently has.

and Meckel, 2014; Barker, 1990; Barker and Osmond, 1986; Hales and Barker, 2001). Our results are complementary to the excellent recent studies by Almond and Currie and by Almond, Currie, and Meckel.

We conduct these analyses using individual-level data from 99 Demographic and Health Surveys collected in 34 sub-Saharan African countries. The data show that 3.2% of all live births in this setting are twins, a figure comparable to those found in the United States and many European countries (Almond, Chay, and Lee, 2005; Pison et al., 2015; Smits and Monden, 2011). Sub-Saharan Africa is also pertinent for our study because it presents a great deal of institutional heterogeneity, which makes it possible to study the effect of political institutions on the timing of mortality convergence between twins and singletons. The underlying idea is that countries with better institutions have better governance and provide better health support and health care infrastructure (Pongou et al., 2017).

We document twin-singleton mortality differences from birth to the age of 25. We show that twins are at a disadvantage in early ages, but this disadvantage gradually decreases and disappears after age 5. The twin-singleton difference in the probability of death falls from 210 per thousand points in the first year of life to less than 3 per thousand points after age 5. Further analyzing mortality differences within the first year of life, we find that these differences are mostly concentrated in the neonatal period. These results show that the “missing twins” phenomenon mainly originates very early in life.

These descriptive findings raise the unavoidable question of whether being a twin has a causal effect on mortality. While twin conception has generally been considered as a random event, recent research shows that a number of maternal factors are likely to increase its probability (Bhalotra and Clarke, 2016). These factors include genetics, diet, and age. If these factors have a

direct effect on child survival, not controlling for them will prevent us from interpreting the effect of twinning on mortality as causal. In a multivariate regression analysis, we directly control for mother's age at child birth since this information is available in the data. However, information on genetics and dietary habits is lacking. We account for these factors by controlling for mother fixed effects, which also controls for all other time-invariant unobserved maternal factors likely to affect both twinning and mortality. These controls have little effect on the magnitude of the twin-singleton differences in mortality.

Next, we answer the question of whether the quality of political institutions affects the timing of mortality convergence between twins and singletons. We exploit a natural historical experiment on the development of political institutions in Africa. This experiment, described in section 2.2, allows us to partition sub-Saharan African countries into four groups, each having a distinct institutional quality. We find that mortality convergence occurs earliest in countries with better institutions. This finding implies that investment in the treatment of twin and other low birthweight children is higher in countries with higher quality institutions.

Overall, our findings highlight some limitations of the fetal origins hypothesis, as initially formulated by Barker and colleagues (Barker, 1990; Barker and Osmond, 1986; Hales and Barker, 2001). According to this hypothesis, intrauterine growth retardation increases the risk of adult diseases, including cardiovascular diseases, diabetes mellitus, and hypertension. Because these diseases significantly increase the risk of mortality and twins have important growth retardation as evidenced by their low birthweight, the fetal origins hypothesis implies that the mortality risk of twins should be higher than that of singletons after childhood. We do not find that to be the case in our data. Christensen et al. (1995) reach a similar conclusion using data from Sweden. We differ

from their study in that we use data from different institutional settings, obtaining new findings about how the quality of institutions affects the timing of the twin-singleton mortality convergence.

Our paper contributes to the literature on health and survival inequalities. However, we focus on a question that has been little studied. Indeed, our study is the first to document the timing of mortality convergence between twins and singletons across different institutional settings, and to show that the “missing twins” phenomenon originates in very early ages. In addition, by showing that this phenomenon is likely to be reduced by better institutions, our analysis proves that biology-induced disadvantages can be partially remedied through appropriate policies. In this sense, our results are consistent with recent studies of the effect of institutions on genetic influences. For instance, Boardman et al. (2012) find that more restrictive school policies and those designed to prevent obesity decrease genetic influences on body mass. Boardman (2009) shows that high taxes on cigarettes moderate genetic influences on smoking in the United States. Pongou et al. (2017) show better political institutions mitigate biological and genetic influences on the sex gap in early-age mortality in sub-Saharan Africa. Our analysis suggests that countries with better institutions invest more in the treatment of twins, mitigating their biological disadvantage.

The rest of this paper is organized as follows. The next section describes the data and the methods, including a description of the African institutional heterogeneity and groups formed. Section 3 presents the results, and the last section provides concluding remarks.

## **2. Data and methods**

We now present the data on live births and institutions and the methods we use for the analyses.

### **2.1 Data**

#### **Twins and singletons**

We use data on all live births recorded in 99 Demographic and Health Surveys collected in 34 sub-Saharan countries between 1990 and 2013 (an Appendix table with data on the countries, surveys, and sample sizes is available from the lead author on request)). For each birth, the survey provides information on the age of the child at the time of the survey if the child is alive, the age at death if the child is dead, birth order, biological sex, twinning status, and various characteristics of the mother. The data are on 3,048,923 deliveries, of which 1.7% are twin deliveries. The corresponding number of children is 3,099,780, of which 3.2% are twins.

#### **2.2 Political institutions**

As mentioned in the Introduction, we also study the effect of political institutions on the timing of the twin-singleton mortality convergence. Our measure of the quality of institutions is based on a historical natural experiment. Acemoglu, Johnson, and Robinson (2001) suggested that differences in the quality of political institutions among countries in sub-Saharan Africa are in part attributable to differences in the extent to which European colonizers were favored by the local disease environment. Colonizers settled in greater numbers in more favorable environments, and in the process set up more “inclusive institutions.” This constituted a natural experiment: choices driven in part by the disease environment resulted in different institutional settings.

The United Nations regions of sub-Saharan Africa (United Nations, 2017) are Western, Middle (Central), Eastern, and Southern Africa, and these correspond to distinct institutional regions. Figure 1 shows the proportion of countries within each region where the mortality rate of



Europeans exceeded the median value<sup>3</sup>, and the proportion exceeding 235 per thousand, which corresponds to about three times the median value.

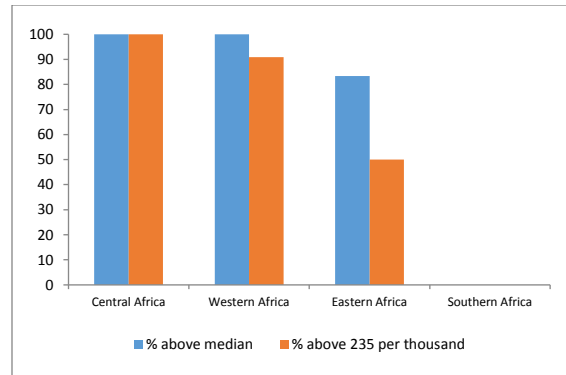


Fig. 1. The Proportion of Countries where the Mortality Rate of European Settlers was Above the Median Value and Above 235 per thousand (three times the median value)

Source: Pongou et al. (2017)

Southern Africa and Eastern Africa have very low and comparatively low disease burdens, respectively, and these regions were settled more heavily than the two high-disease-burden regions, Central and Western Africa. The case for region-specific institutions is buttressed in considering the principal colonizers. Most countries in Central Africa and many in Western Africa were colonized by the French, while in Eastern and Southern Africa the British were the principal colonizers. Colonization policies of these two countries were very different. For example, the French put less emphasis on providing schooling to women than did the British: as shown by data in Shapiro and Tenikue (2015), in a sample of nearly 30 countries in the region, mean years of schooling exceeded 6 in the Anglophone countries, and 19% of women had no schooling; while mean schooling in the Francophone countries was only 3.9 years, and 44% of women had never been to school.

<sup>3</sup> The median value of the mortality rate, 78.1 per thousand, is for the base sample of ex-colonies in Acemoglu et al. (2001) and Albouy (2012).

To assess the degree to which the quality of institutions differs among these four regions, we use three variables that have been used in the literature: the corruption index, democratic accountability, and law and order. Data are from the World Bank Development Indicators (2011) and from the Political Risk Services (2011).

The average values of these three indicators for each region, over the period between 1980 and 2009, are shown in Figure 2. Each indicator tells the same story: Southern Africa has the best institutions, followed by Eastern Africa, then Western Africa, and Central Africa has the worst institutions.

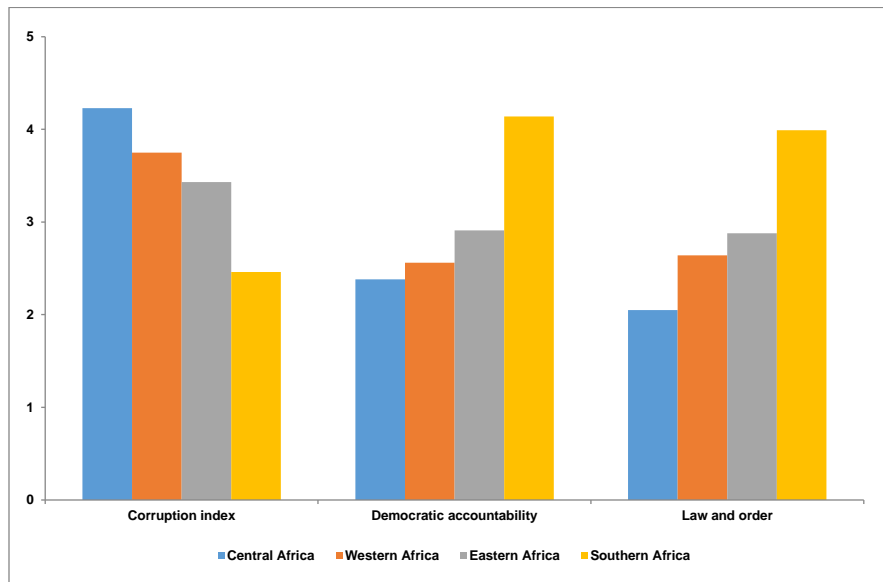


Fig. 2. Quality of Institutions by sub-Saharan African Region

Source: Pongou et al. (2017)

Institutional quality translates into health investment. In analyses not shown here, we find that public health expenditure per capita is greatest in Southern Africa and lowest in Central Africa. Figure 3 below also shows the proportion of children immunized against measles during the period 1960-2011. Clearly, regions with better institutions have higher proportion of immunized children.

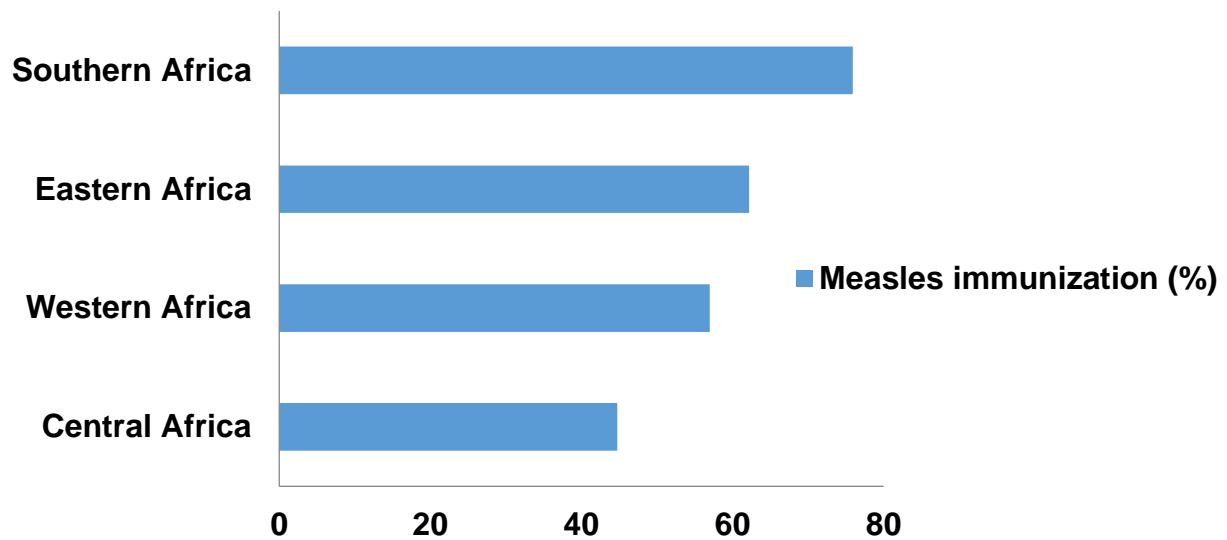


Fig. 3: Percentage of children immunized against measles by sub-Saharan African institutional region

Source: Data are from the World Bank Development Indicators (2011).

### 2.3 Methods

We first use duration analysis methods to document differences in survival rates of twins and singletons by age, measured in months. The maximum age considered is 300 months (25 years). In a second step, we discretize the data in years. A child is included in the sample of year  $t$  if she has survived to year  $t$ . If a child dies in year  $t$ , she is not included in the sample in year  $t+1$ . For every year, we run a mother fixed effect regression to estimate the twin effect on mortality as children age. We consider that mortality converges when the difference in twin and singleton mortality rates falls below 3 per 1000<sup>4</sup> and remains below this value as children age.

We use a mother fixed effect model<sup>5</sup> as indicated in Eq. 1 below to control for family background characteristics that are constant over time. This model helps to account for mother's

<sup>4</sup> The Millennium Development Goal target of child mortality for developed countries is 5 per 1000.

<sup>5</sup> We also estimated the equation by OLS and added mother's characteristics as control variables. Results (available upon request) were qualitatively similar to those presented in this paper.

unobserved heterogeneity with a time-invariant effect on child mortality. It also helps to account for mother’s genetic factors that may affect twinning and mortality. These factors are unmeasured in the survey.

For a given year  $t$ , a child ( $i$ ) born to a mother ( $j$ ), the following mother fixed effect regression is estimated:

$$Y_{ijt} = \alpha_t + \beta_t Twin_{ijt} + \gamma_t X_{ijt} + F_{jt} + \varepsilon_{ijt} \quad 1$$

In Eq. 1,  $Y_{ijt}$  is a dummy variable that takes on the value 1 if the child died in year  $t$  conditional on surviving to year  $t-1$ ;  $Twin_{ijt}$  is a dummy variable that takes the value of 1 if the child is a twin;  $X_{ijt}$  is a vector of child covariates (sex and birth order);  $F_{jt}$  is the mother fixed effect term; and  $\varepsilon_{ijt}$  is the error term, assumed to be uncorrelated with  $Twin_{ijt}$  given the controls as well as the mother fixed effect. In this equation, only the dependent variable ( $Y$ ) changes over time (as children age) and the estimated coefficients at a given age reflect the effect of the covariates at that age.

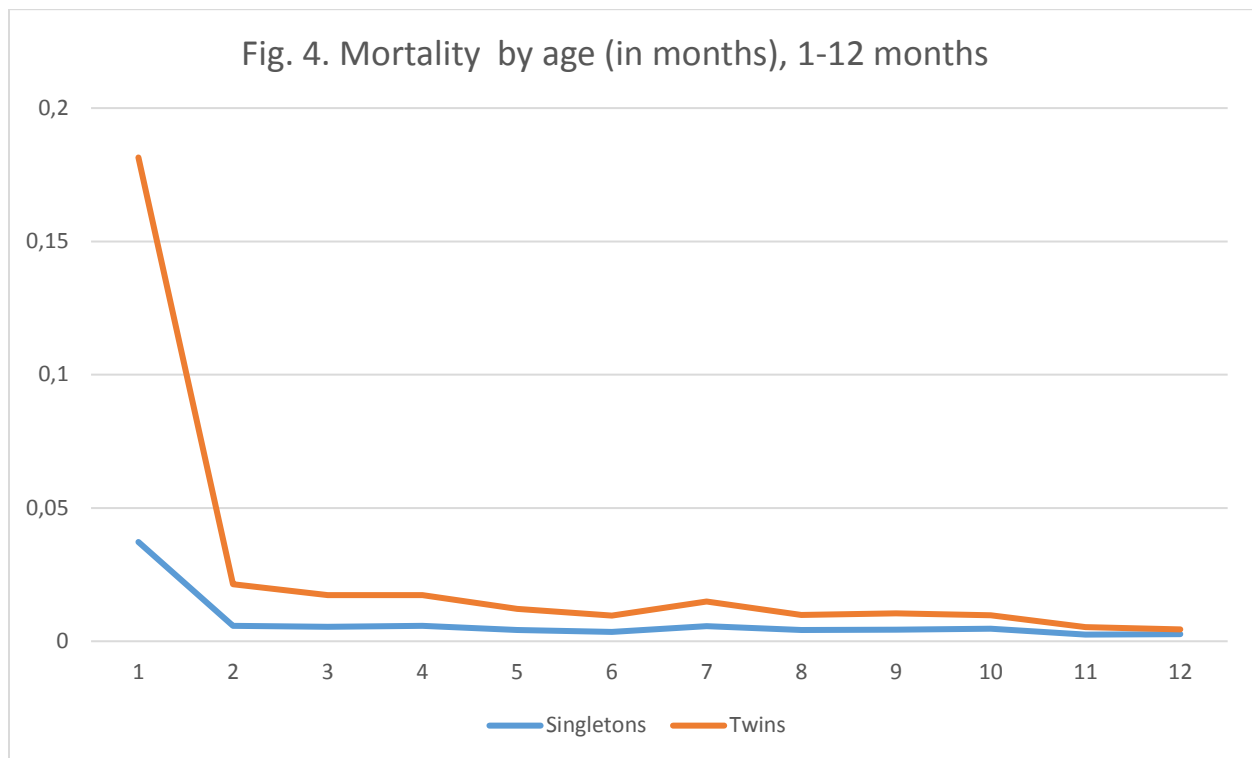
### 3. Results

#### 3.1 Descriptive analyses

##### Mortality and survival of twins and singletons by age

Figure 4 displays mortality rates by age (in months) for the first year of life. An important twin-singleton mortality difference is evident during the first twelve months of life. Twins have lower survival throughout these first 12 months, with the differences being especially marked in the first few months of life. At this early age, mortality of twins is nearly five times as high as that of singletons in the first month, presumably due to a greater incidence of biological problems (e.g., birth defects) among twins. The ratio of mortality rates of twins to those of singletons decreases

almost monotonically from about five in the first month to about three in the next few months, reaching 1.7 in the 12<sup>th</sup> month.



Mortality of twins and singletons by age in years is shown in Figure 5. The substantial difference in the first year, already noted, is apparent. And the diminishing differences are evident up through age four.

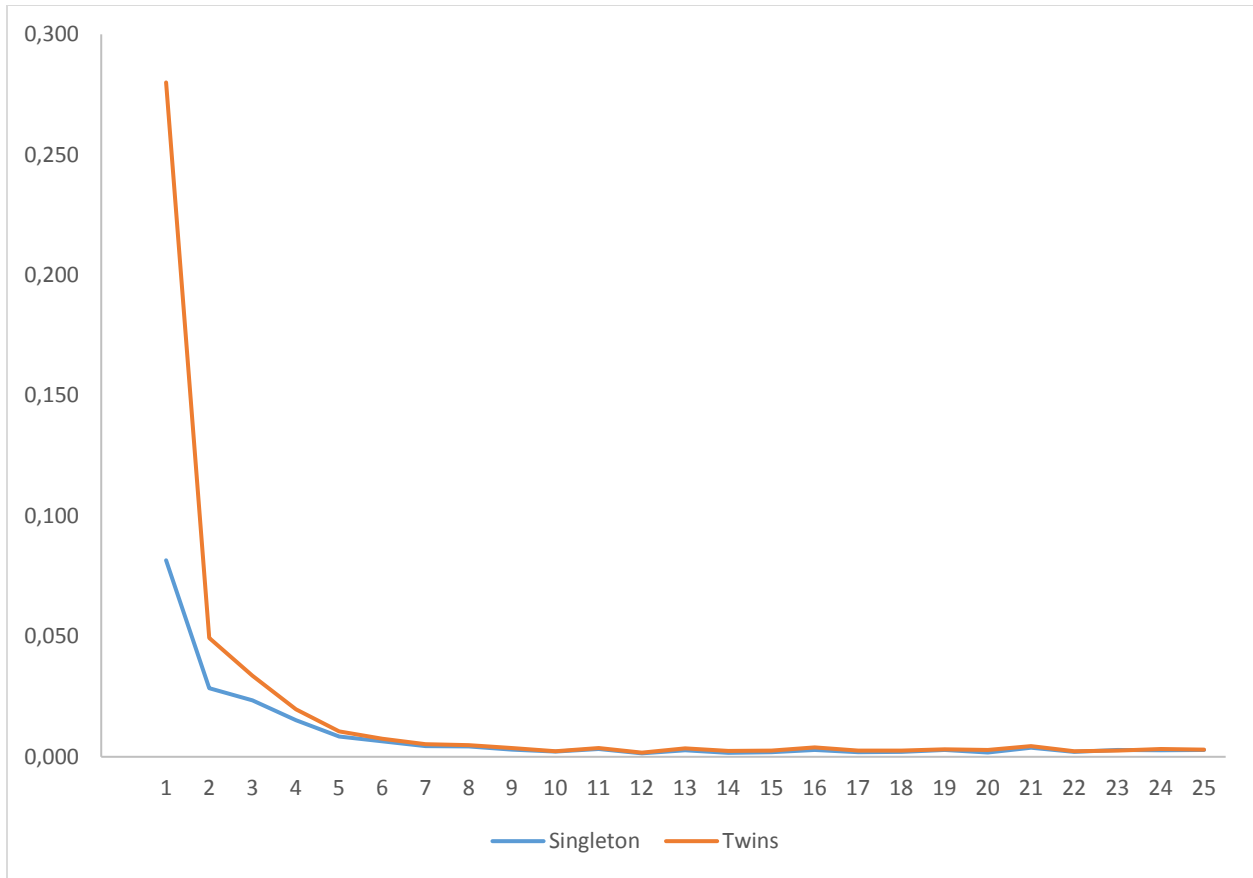


Fig. 5: Mortality of twins and singletons by age (in years)

As children age, the difference in mortality rates between twins and singletons decreases, but at a non-monotonic rate<sup>6</sup>. The cumulative data by years show that mortality of twins is more than three times that of singletons during the first year of life, but this factor falls to 1.7 and 1.3 in years 2 and 3, respectively. From age 5 to age 10, twin mortality remains slightly higher (by about 10%), but the overall mortality has dropped to an average of 5 per 1000. Beyond this age, the overall mortality is below 4 per 1000.

<sup>6</sup> The log-log curves of the survival rates of twins and singletons (available upon request) are slightly parallel over different periods of time. This indicates that the twin effect on mortality is not constant as children age.

## **Cohort trends in mortality and survival of twins and singletons**

We now analyze time trends in the twin-singleton mortality difference. We use data collected since 1990 from women aged 15-49 at the time of the survey. These data therefore provide detailed information on births that date back more than 34 years prior to the survey, which allows us to monitor mortality over time.

It is well known that child survival in sub-Saharan Africa has markedly improved over the years<sup>7</sup>, mainly due to important improvements in neonatal care and vaccination against infectious diseases (United Nations 2015). Prior studies have not, however, looked at whether these improvements differ for twins and singletons. We investigate this question. Figure 6 displays the survival of twins and singletons by decade of birth (1960s, 70s, 80s, 90s, 2000s, and 2010-13). It shows that survival has improved for both twins and singletons over the years. Also, the twin-singleton mortality difference has narrowed, but the higher mortality of twins has persisted. For instance, during the infant period, when children are at their highest mortality risk, a twin had a 30-percentage point greater risk of dying in the 1960s, about a 20-percentage point higher risk of dying in the 1970s to 1990s, and only a 15-percentage point greater risk of dying after the year 2000.

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<sup>7</sup> The annual rate of reduction of the infant mortality rate increased from 1.6% in the 1990s to 4.1% in 2000–2015 (United Nations, 2015).

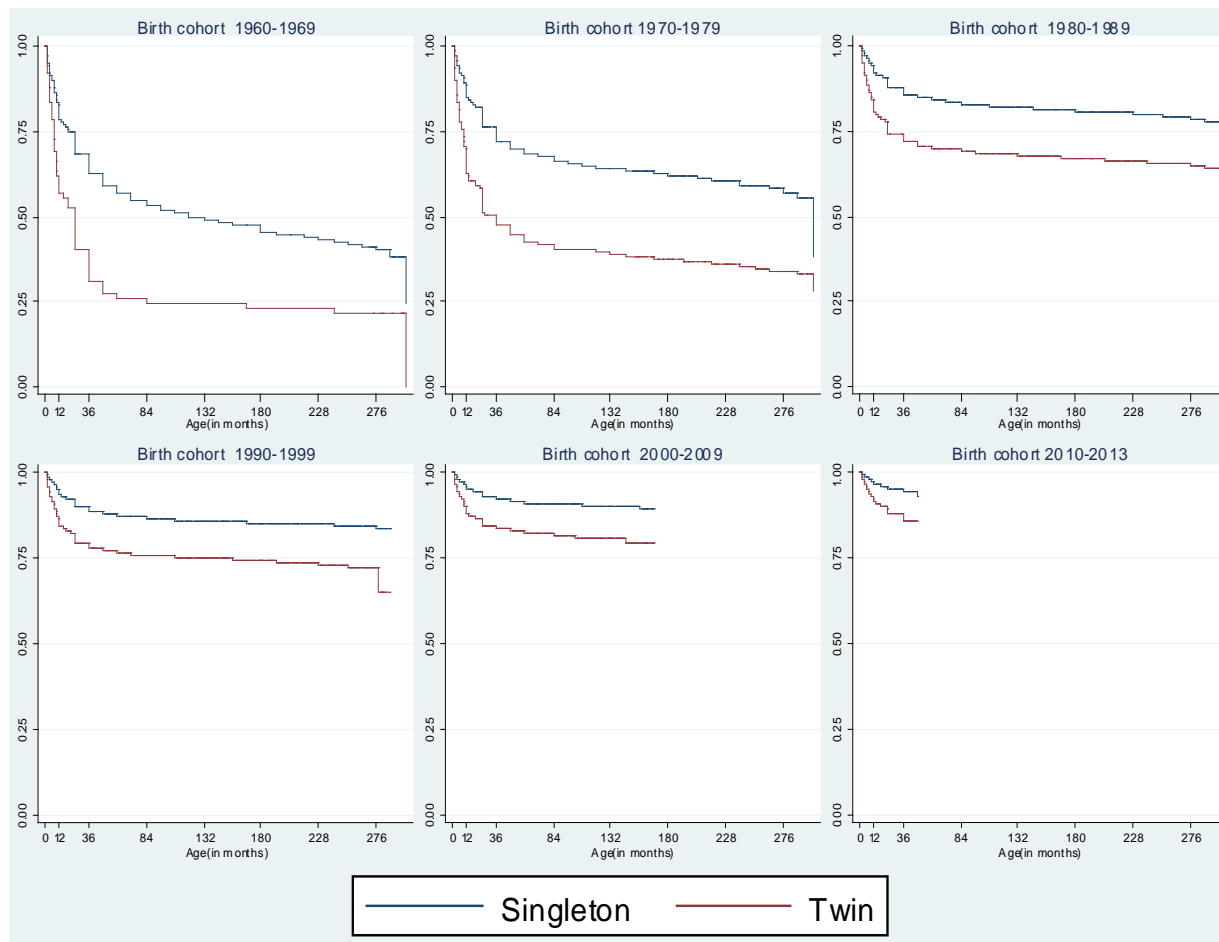


Fig. 6: Kaplan-Meier Survival rate of twins and singletons by birth cohort

### 3.2 Regression-based analyses

We estimate equation 1 to determine the twin-singleton mortality difference at each age after controlling for a child's baseline characteristics (sex and birth order) and mother fixed effect. The estimated twin effect on mortality by age shows that twins have significantly higher mortality for the first five years of life. In addition, the mortality difference between twins and singletons is greatest in the first year of life (by about 21%), and declines steadily up through five years of age. After the age of 5, the twin effect remains positive, sometimes statistically significant, up to the age of 20, but its value is around 0.002 or below. The twin effect turns negative, smaller than 0.002



in absolute terms, but not statistically significant, at the age of 21. From this age, the twin effect fluctuates around zero but remains below 0.001. Based on the convergence criterion defined earlier, we state that twin and singleton mortality rates converge at the age of 5. Or strictly speaking, the twin and singleton difference in mortality rates falls to an acceptable level at the age of 5.

As documented in the literature, male children face a higher mortality rate than do females because of differences in the preconception environment and the biological make-up (Pongou, 2013; Pongou, 2015). Also, earlier-born children are exposed to higher mortality risk (Table A in appendix). We investigate whether gender and birth order effects on mortality are reinforced by twinning. The results show that male twins display a higher mortality risk during their first year of life (about 10% higher than for female twins) but not much afterwards (Table 1). As regards birth order, the interaction of birth order and twinning displays a very moderate effect on mortality. If anything, it is within the first three years of life (Table 2).

**Table 1: Effect of the interaction between child sex and twin status on mortality by age**

The dependent variable is a dummy that takes on the value one if the child died during the corresponding age											
VARIABLES	Age (in years)										
	1	2	3	4	5	6	7	8	9	10	
Twin	0.201*** (0.001)	0.026*** (0.001)	0.016*** (0.001)	0.005*** (0.001)	0.002*** (0.001)	0.002** (0.001)	0.001 (0.001)	0.001 (0.001)	0.001 (0.001)	0.001 (0.001)	0.000 (0.001)
Male	0.013*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001** (0.000)	0.000* (0.000)	0.000** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.000*** (0.000)
Male * Twin	0.022*** (0.002)	0.001 (0.002)	-0.002 (0.001)	0.003*** (0.001)	0.002 (0.001)	-0.000 (0.001)	0.000 (0.001)	0.001 (0.001)	0.000 (0.001)	0.001 (0.001)	0.001 (0.001)
Birth Order	-0.008*** (0.000)	-0.003*** (0.000)	-0.003*** (0.000)	-0.002*** (0.000)	-0.001*** (0.000)	-0.001*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)
Observations	3,098,118	2,650,000	2,411,296	2,199,996	2,010,447	1,843,286	1,697,511	1,540,996	1,401,063	1,267,105	

Mother fixed effect regression analysis of annual mortality by age. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

**Table 2: The effect of the interaction between birth order and twin status on mortality by age**

The dependent variable is a dummy that takes on the value one if the child died during the corresponding age										
VARIABLES	Age (in years)									
	1	2	3	4	5	6	7	8	9	10
Twin	0.202*** (0.002)	0.021*** (0.002)	0.012*** (0.002)	0.004** (0.002)	0.002 (0.001)	0.002 (0.001)	-0.001 (0.001)	0.000 (0.001)	0.001 (0.001)	0.000 (0.001)
Male	0.014*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.000** (0.000)	0.000** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.000*** (0.000)	0.000*** (0.000)
Birth Order	-0.008*** (0.000)	-0.003*** (0.000)	-0.003*** (0.000)	-0.002*** (0.000)	-0.001*** (0.000)	-0.001*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)
Birth Order * Twin	0.002*** (0.000)	0.001*** (0.000)	0.001* (0.000)	0.001* (0.000)	0.000 (0.000)	-0.000 (0.000)	0.000* (0.000)	0.000 (0.000)	0.000 (0.000)	0.000 (0.000)
Observations	3,098,118	2,650,000	2,411,296	2,199,996	2,010,447	1,843,286	1,697,511	1,540,996	1,401,063	1,267,105

Mother fixed effect regression analysis of annual mortality by age. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

### **3.3 The role of political institutions in the timing of the twin-singleton mortality convergence**

As shown in the first panel of Figure 7, during the first year of life, twin mortality is higher than singleton mortality in all parts of sub-Saharan Africa. However, twins are more vulnerable in Eastern, Western, and Central Africa compared to Southern Africa. A similar pattern is observed during the second year of life, but with smaller values of the twin-singleton differences. Starting in year 3, there is no mortality risk difference between twins and singletons in Southern Africa, while for the other regions, the convergence does not occur until the fifth year of life. This analysis therefore shows that the twin-singleton mortality convergence occurs earlier when institutions are better. Higher quality institutions provide better prenatal and postnatal care, which benefits the most vulnerable children. This likely explains why the mortality risk of twins and singletons converges earlier in Southern Africa.

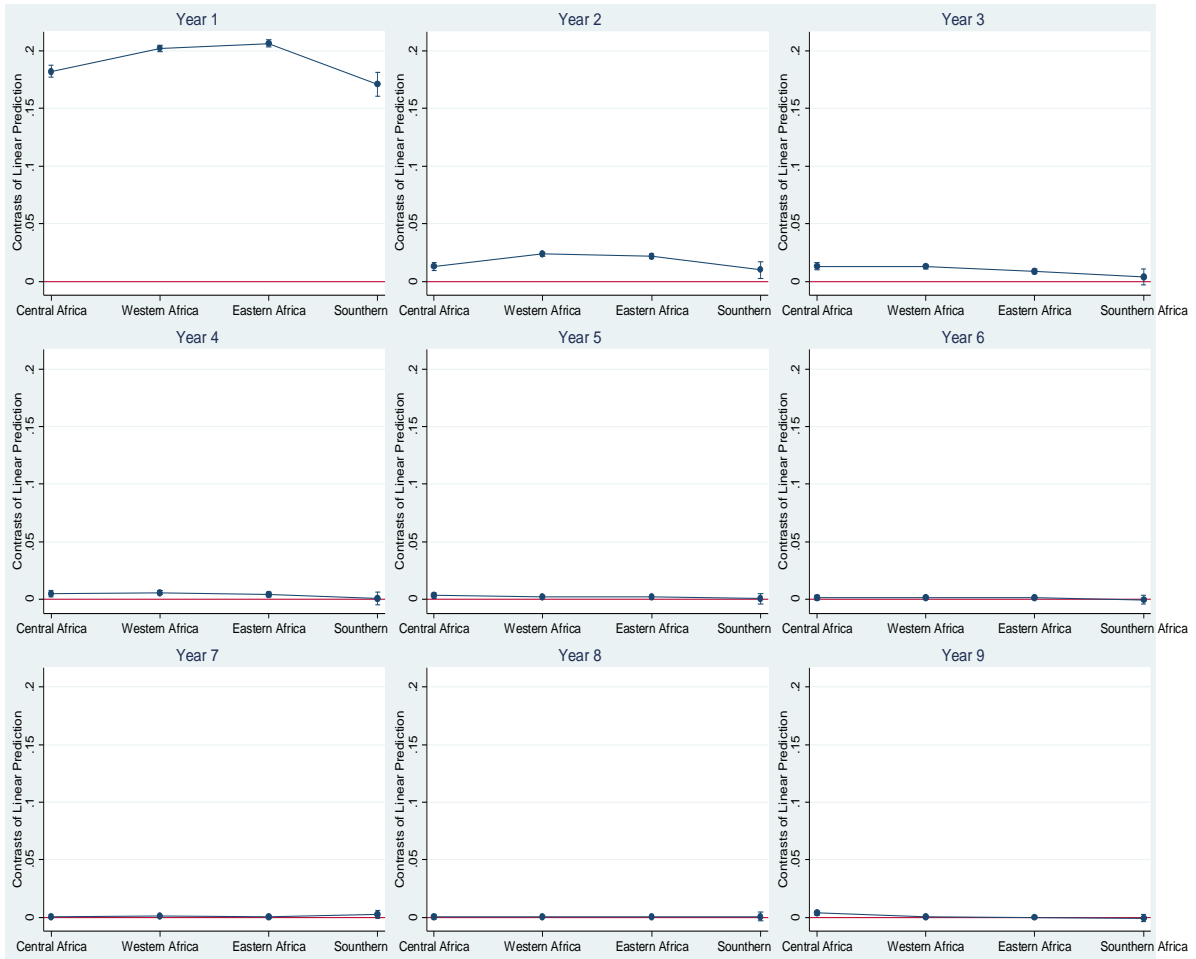


Fig. 7: Twin-Singleton difference in (predicted) mortality rates by region (regression-based analyses)

## **4. Conclusion**

This paper examines twin-singleton differences in mortality risk through the age of 25, as well as the effect of political institutions on the timing of convergence. We find an important difference in the first year of life, but the difference decreases with age and practically disappears after age 5. This convergence at the age of 5 and after persists even after child characteristics are controlled for. After the age of 5 all children are exposed to a very low annual mortality risk (about 7 deaths per 1000 children). This is the outcome of both the selection effect, because most vulnerable children died before age 5, and the fact that by that age many children are already immunized for some infectious diseases and are less vulnerable to health problems. We also note that the fact that the mortality of twins and singletons is not significantly different after childhood shows some limitation of the fetal origins hypothesis, according to which the risk of adult morbidity and mortality is positively affected by intrauterine growth retardation. We also find that this convergence occurs earlier in countries with better political institutions. This finding implies that the “missing twins” phenomenon can be mitigated through appropriate policy interventions.

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**Appendix Table A: Mother fixed effect regression analysis of annual mortality by age**

The dependent variable is a dummy that takes on the value one if the child died during the corresponding age												
VARIABLES	Age(in years)											
	1	2	3	4	5	6	7	8	9	10	11	12
Twin	0.212*** (0.001)	0.027*** (0.001)	0.015*** (0.001)	0.007*** (0.001)	0.003*** (0.001)	0.002*** (0.001)	0.001** (0.000)	0.002*** (0.000)	0.001* (0.000)	0.001 (0.000)	0.001 (0.000)	0.001** (0.000)
Male	0.014*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.001*** (0.000)	0.000** (0.000)	0.000** (0.000)	0.000*** (0.000)	0.001*** (0.000)	0.000*** (0.000)	0.000*** (0.000)	0.000** (0.000)	0.000*** (0.000)
Birth Order	-0.008*** (0.000)	-0.003*** (0.000)	-0.003*** (0.000)	-0.002*** (0.000)	-0.001*** (0.000)	-0.001*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000** (0.000)
Constant	0.102*** (0.000)	0.038*** (0.000)	0.033*** (0.000)	0.021*** (0.000)	0.011*** (0.000)	0.008*** (0.000)	0.005*** (0.000)	0.006*** (0.000)	0.004*** (0.000)	0.002*** (0.000)	0.004*** (0.000)	0.001*** (0.000)
Observations	3,098,118	2,650,000	2,411,296	2,199,996	2,010,447	1,843,286	1,697,511	1,540,996	1,401,063	1,267,105	1,155,549	1,028,447
R-squared	0.019	0.002	0.002	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

The dependent variable is a dummy that takes on the value one if the child died during the corresponding age													
VARIABLES	Age (in years)												
	13	14	15	16	17	18	19	20	21	22	23	24	25
Twin	0.001* (0.001)	0.000 (0.000)	0.002*** (0.001)	0.000 (0.001)	0.001 (0.001)	0.001 (0.001)	0.001 (0.001)	0.001* (0.001)	-0.000 (0.001)	0.000 (0.001)	0.001 (0.001)	-0.000 (0.002)	-0.001 (0.002)
Male	0.000** (0.000)	0.000** (0.000)	0.000 (0.000)	0.000* (0.000)	-0.000 (0.000)	-0.001*** (0.000)	0.000 (0.000)	-0.000 (0.000)	0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)
Birth Order	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000*** (0.000)	-0.000** (0.000)	-0.000*** (0.000)	-0.000 (0.000)	-0.001*** (0.000)	-0.000 (0.000)	-0.000** (0.000)	-0.000 (0.000)	-0.000** (0.000)
Constant	0.003*** (0.000)	0.002*** (0.000)	0.002*** (0.000)	0.004*** (0.000)	0.002*** (0.000)	0.003*** (0.000)	0.004*** (0.000)	0.002*** (0.000)	0.006*** (0.000)	0.002*** (0.000)	0.004*** (0.000)	0.003*** (0.000)	0.004*** (0.000)
Observations	935,707	828,780	734,789	648,162	567,182	494,646	430,386	364,140	314,530	256,268	216,381	176,158	142,442
R-squared	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

## Missing Twins: Fetal Origins, Institutions, and Twin-singleton Mortality Convergence

### Supplemental Materials

**Table A: List of countries, survey years and sample size**

Country	Years of DHS surveys (Number of surveys)	Number of children	% of twin
Angola	(1) 2011	25,857	3.00
Burkina Faso	(4) 1993, 1999, 2003, 2010	140,498	3.01
Benin	(4) 1996, 2001, 2006, 2012	143,141	4.81
Burundi	(1) 2012	30,051	2.44
Congo Democratic Republic	(2) 2007, 2014	88,824	3.57
Central African Republic	(1) 1995	16,936	2.64
Congo (Brazzaville)	(2) 2005, 2012	48,635	4.03
Ivory Coast	(3) 1994, 2005, 2012	66,439	3.66
Cameroon	(4) 1991, 1998, 2004, 2011	98,566	3.93
Ethiopia	(2) 1997, 2003	129,595	2.27
Gabon	(2) 1997, 2003	39,987	3.44
Ghana	(4) 1994, 1999, 2003, 2008	53,460	3.56
Guinea	(3) 1999, 2005, 2012	77,741	3.88
Kenya	(3) 1993, 2003, 2009	68,507	2.88
Liberia	(2) 2009, 2013	71,717	3.72
Lesotho	(2) 2005, 2010	29,137	2.87
Madagascar	(5) 1992, 1997, 2004, 2009, 2013	123,210	2.06
Mali	(3) 1996, 2001, 2006	138,468	3.09
Malawi	(4) 1992, 2005, 2010, 2012	167,607	3.91
Mozambique	(3) 1997, 2004, 2011	101,179	3.36
Nigeria	(5) 1990, 1999, 2003, 2008, 2013	318,276	3.53
Niger	(4) 1992, 1998, 2006, 2012	131,290	3.02
Namibia	(3) 1992, 2000, 2007	47,840	2.50
Rwanda	(4) 1992, 2005, 2008, 2013	132,019	2.38
Senegal	(2) 2008, 2013	68,528	3.46
Malawi	(4) 1997, 2005, 2009, 2011	189,084	2.86
Swaziland	(1) 2007	11,410	2.76
Chad	(2) 1997, 2004	47,187	2.89
Togo	(1) 1998	26,269	4.05
Tanzania	(5) 1992, 1999, 2005, 2010, 2012	164,843	3.50
Uganda	(4) 1995, 2001, 2009, 2011	118,724	2.86
South Africa	(1) 1998	22,934	2.56
Zambia	(4) 1992, 1996, 2002, 2007	92,092	3.40
Zimbabwe	(4) 1994, 1999, 2006, 2011	69,729	3.22
<b>Total</b>	<b>(99)</b>	<b>3,099,780</b>	<b>3.26</b>

**Table B: Means of the main variables by age of child**

VARIABLES	Age (in years)									
	1	2	3	4	5	6	7	8	9	10
Twin	0.03	0.03	0.02	0.02	0.02	0.02	0.02	0.02	0.02	0.02
Male	0.51	0.50	0.50	0.50	0.50	0.50	0.50	0.50	0.50	0.51
Birth Order	3.37	3.34	3.31	3.29	3.25	3.22	3.18	3.12	3.06	3.00
Mother education (years)	2.91	2.95	2.95	2.95	2.95	2.93	2.91	2.90	2.90	2.89
Observations	3,098,118	2,650,000	2,411,296	2,199,996	2,010,447	1,843,286	1,697,511	1,540,996	1,400,196	1,267,105

