The Effects of Intrauterine Malnutrition on Birth and Fertility Outcomes: Evidence from the 1974 Bangladesh Famine

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Abstract

This paper uses the Bangladesh famine of 1974 as a natural experiment to estimate the impact of intrauterine malnutrition on sex of the child and infant mortality. In addition, we estimate the impact of malnutrition on post-famine pregnancy outcomes. Using the 1996 Matlab Health and Socioeconomic Survey (MHSS), we find that women who were pregnant during the famine were less likely to have male children. Moreover, children who were *in utero* during the most severe period of the Bangladesh famine were 32 percent more likely to die within one month of birth compared to their siblings who were not *in utero* during the famine. Finally, controlling for pre-famine fertility, we find that women who were pregnant during the Famine experienced a higher number of stillbirths in the post-Famine years. This increase appears to be driven by an excess number of male stillbirths.

1 Introduction

Famines have been associated with increased mortality in several regions of the world and in many different time periods. Although this association has been documented for all age groups, increased rates of mortality have been especially striking among infants. For instance, Lindeboom et al. (2010) estimated that approximately 25 percent of children below one year of age died during the Dutch Potato famine of 1847-1864. Infant mortality rates also increased substantially during the Finnish famine of 1866-1868, the 1941-1944 siege of Leningrad, and the World War II Dutch famine (Kannisto et al. 1997; Hart 1993).

Large-scale famine events have also offered researchers the opportunity to study the impact of intrauterine nutrition on the health and survival probabilities of infants (Stein and Susser 1975; Razzaque et al. 1990; Lumey and Stein 1997; Almond et al. 2008). This is particularly important in light of Barker's (1990) "fetal origins" hypothesis, which argues that *in utero* malnutrition adversely impacts the health of the fetus and leads to increased risk for future diseases (Almond and Currie 2011). Although serious famines are relatively rare, they enable researchers to circumvent factors such as household environment and socioeconomic background that might confound the results of studies relying on self-reports of maternal nutrition. Thus, they present a unique method of analyzing the impacts of fetal nutrition on child and adult outcomes.

The most comprehensive set of evidence on the impact of intrauterine malnutrition on infant and adult outcomes comes from the 1944-46 Dutch famine (Almond et al. 2008). Stein and Susser (1975) found that pregnant women exposed to the Dutch famine in their third trimester had lower postpartum weight while their infants had lower head circumference

and reduced length at birth compared to non-exposed women. Lumey and Stein (1997) found that baby girls who were exposed to the Dutch famine during their third trimester of pregnancy had lower birth weight, while Roseboom et al. (2001a) found that late-term exposure was associated with increased mortality among male infants. Hart (1993), on the other hand, compared the outcomes of infants who were exposed to different levels of famine intensity. She found no evidence that children in areas most affected by the famine had higher mortality. Exposure to the Dutch famine is also associated with adult outcomes such as increased risk of coronary heart disease (Rosenboom et al. 2001b; Painter et al. 2005) and higher rates of antisocial personality disorders (Neugebauer et al. 1999). Studies of other famines corroborate these results. Razzaque et al. (1990) found that children conceived during the 1974-75 Bangladesh famine were at a higher risk of dying before the age of one month but had a lower risk of death during their second year of life compared to children who were not conceived during the famine. Scott et al. (1995) found that a 1623 famine that occurred in northern England increased infant mortality, especially among those who were exposed during the later stages of pregnancy. More recently, economists have documented a link between fetal malnutrition and subsequent adult economic outcomes. For instance, Almond et al. (2008) found that Chinese cohorts exposed to intrauterine malnutrition during the Chinese famine of 1959-1962 had a higher probability of being illiterate as adults and were less likely to work.

Beyond their effects on health and economic outcomes, famines have also been hypothesized to increase the ratio of female-to-male births. Such a link can be interpreted in light of the Trivers-Willard (1973) hypothesis that male infants will fare worse when parental conditions, such as health, are bad. From an evolutionary perspective, the reproductive ad-

vantage that females have during bad conditions enables mothers to maximize their number of grandchildren (Rosenfeld and Roberts 2004). If mothers in good conditions are able to have high-quality sons, then they can obtain more grandchildren through sons rather than daughters as those sons would be able to acquire multiple mates. In contrast, mothers in bad conditions will obtain more grandchildren through daughters than sons (Cameron 2004; Almond and Edlund 2007).

Although this pattern has been observed among some mammalians (Huck et al. 1986; Meikle and Drickamer 1986; Rosenfeld and Roberts 2004), its existence among human populations is controversial, with studies continuing to provide mixed evidence (Cameron 2004). For example, Almond and Edlund (2007) found that married mothers in the United States are more likely to give birth to a son compared to unmarried mothers, and also that poorly educated women are more likely to give birth to daughters. Further supporting this hypothesis, Almond et al. (2008) found an excess number of females among middle-aged Chinese cohorts who had been exposed *in utero* to the Chinese famine. In contrast, neither Stein et al. (2004) nor Anderson and Bergström (1998) found an association between intrauterine exposure to malnutrition and an excess of female births.

Even when researchers have found an impact on the sex ratio, it has been difficult to pin down the mechanism for this adjustment (Cameron 2004). One possibility ia that pre-natal determination of an infant's sex could lead parents to selective abortions, thus changing the sex ratio at birth. However, without the ability to determine the sex of the infant, higher rates of infant male mortality is another mechanism that may lead to an imbalance in the sex ratio. Consistent with this argument, Almond and Edlund (2007), using U.S. data, found that being married lowered the probability that a deceased infant was male. It is not clear,

however, whether similar patterns emerge in the context of a developing country, particularly in one where there may be a pronounced preference for sons.

Intrauterine malnutrition has also been shown to impact long-term reproductive outcomes. Lumey and Stein (1997) provided evidence that women who were *in utero* during the Dutch famine experienced a higher risk of stillbirths and perinatal deaths later in life, especially those who were affected in the third trimester of pregnancy. This is despite the fact that no association was found between intrauterine malnutrition and completed fertility, age at first pregnancy, or child spacing. More recently, Almond et al. (2008) provided evidence that intrauterine nutritional deprivation is linked to the reproductive outcomes of the next generation. Specifically, they found that infants born to mothers who were exposed *in utero* to the Chinese famine were less likely to be male. Existing studies, however, do not examine whether exposure to malnutrition during pregnancy will also impact the future outcomes of the mother herself in addition to those of her exposed offspring.

Although this specific hypothesis has not been previously investigated, studies have documented a negative link between episodes of eating disorders and future pregnancy outcomes. Bulik et al. (1999), for instance, found that women with a history of anorexia nervosa had significantly more miscarriages and were more likely to deliver premature babies compared to a control group of women with no previous history of eating disorders. These findings are consistent with the findings of retrospective studies which relied on women's own reporting of nutrition and pregnancy outcomes (Katz and Vollenhoven 2000).

Despite this growing literature, some important questions remain unanswered, while others need to be reexamined. First, do women exposed to malnutrition during pregnancy have a higher probability of a female live birth? Second, conditional on being a live birth, do

male infants exposed to fetal malnutrition have a higher probability of early mortality? We use the Bangladesh famine of 1974 (henceforth the Famine) as a natural experiment to answer these questions. Although Razzaque et al. (1990) also analyzed mortality outcomes in the context of the Bangladesh famine, our analysis compares the outcomes of siblings, only of whom one was exposed to the Famine. This within-family analysis is an important methodological contribution because it controls for all time-invariant confounding factors at the family level and unlike existing studies, does not rely on geographical variation in the intensity of famines or on comparing children exposed at different stages of gestation.[1] We also examine a third question: Does severe undernutrition during pregnancy impact the reproductive outcomes of exposed women in future pregnancies after the Famine event is over? In particular, are women who experience a famine while pregnant more likely to have future miscarriages and stillbirths? Do these reproductive outcomes vary by the sex of the child? Despite the importance of understanding whether nutritional deprivation during pregnancy impacts future reproductive outcomes, this is the first study that uses a natural experiment to link maternal malnutrition with subsequent fertility outcomes.

The remainder of the paper is organized as follows. Section 2 provides a brief background of the Famine. In section 3, we discuss our data sources and construction of the main variables of interest. We describe the analytical framework in section 4 and present the results in section 5. We discuss the findings in section 6.

[1] There are several other smaller differences between our studies. Razzaque et al. (1990) defined the period of the famine-born as July 1974 through June 1975 and compares the outcomes during that period to those during a famine-conceived period of July 1975-1976 and a non-famine period between July 1976 and March 1977. Our study uses a different definition of the famine, August 1974 to October 1975, based on the months where the price of rice was more than 50 percent higher than the pre-famine price. Our study also uses a wider window of time as a control group and has other minor differences, such as our use of a multivariate logistic regression, instead of a univariate one.

2 Famine background

Bangladesh, formerly a part of India (known as East Bengal then East Pakistan), became independent in 1971. While part of the Indian colony, Bangladesh experienced massive death and suffering during the Great Bengal Famine of 1943. The causes and severity of this famine have been studied by both the India Famine Inquiry Commission (1976) and by Sen (1981). Bangladesh suffered another significant famine in 1974 following a smaller one in 1971. Toward the middle of 1974, severe flooding led to a sharp rise in unemployment, particularly among rural farmers and laborers (Sen 1981). Although food availability remained unaffected until the harvest period later in the year, food prices began to rise immediately, toward an eventual increase of 500 percent (Dyson 1991). While the precise cause of these increased prices remains unclear, the most likely factors appear to be macroeconomic and include a combination of inflation and speculation about future price increases (Sen 1981). Regardless, the unemployment and dramatic price increases placed a severe burden on rural areas.

The Famine itself began in March of 1974, reaching its peak between July and October. It began to subside by the end of that year, although the impact on people due to high food prices and increased mortality lasted well into 1975 (Alamgir 1980). Figure 1 depicts the price of medium rice in Bangladesh between July 1972 and June 1976 (Bangladesh Institute of Development Studies 1977). The nationwide price of rice did not peak until February of 1975, returning to pre-Famine levels by the end of that year.

The Famine and high food prices led to increased mortality rates that persisted through 1976 (Razzaque et al. 1990). Estimates of fatalities range from 450,000 to well over a

million (Alamgir 1980), which comprises approximately 0.6-1.3 percent of the estimated national population (United Nations 2002). Dyson (1991) uses data from Matlab, the region of particular interest in this study, to plot mortality rates in Bangladesh before, during, and after the Famine (see Figure 8, page 287), showing a peak death rate in early 1975 that remained high well after the Famine ended, as many remained weakened and sick. In fact, the death rate for both infants and all ages was higher in 1975 than in 1974 (Dyson 1991).

Although female child mortality rates tend to be higher than male rates in Matlab during normal years (Choe and Razzaque 1990, Fauveau and Chakraborty 1994, Langsten 1981), the Famine resulted in significant but similar increases in child mortality rates among both male and female children (Koenig and D'Souza 1986; Bairagi 1986). Moreover, fertility rates declined by about 34 percent during 1974-1975, before increasing by 17 percent in the post-Famine years, thereby partially offsetting the Famine's effect (Razzaque 1988).

3 Data

The 1996 Matlab Health and Socioeconomic Survey is a cross-sectional data set sampling 4,364 households in Matlab district, a poor, rural, agricultural area of Bangladesh approximately 55 km southeast of Dhaka (Rahman et al. 1999). These households cover 2,687 baris (groups of households living and working together, sharing a common outdoor space), which comprise a one-third random sample of all Matlab baris. All women aged 15 and older in the sample were asked about their fertility history, including any subsequent mortality outcomes for their children. From this information, we are able to gather the date, sex, birth outcome (miscarriage, stillbirth, or live birth), and neonatal and infant mortality outcomes for 24,916

children born between 1919 and 1996. We then supplement this information on births with demographic characteristics of the mother, including her age at the pregnancy outcome, age at first marriage, years of education (in years), and adult height (in centimeters), and the number of male and female children she had prior to 1974. We also use information about the relative size of the infant at birth compared to other infants (i.e. much bigger, bigger than, smaller then, or same size as other babies), number of prenatal visits that the mother had during a given pregnancy, whether the infant was breastfed, whether the child was born in a hospital, a clinic, or at home, and whether there was a skilled care provider present at the time of birth. These variables are important to account for in the empirical analysis, as they could have an impact on the health and survival rates of the infants.

The reliability of the women's fertility histories depends on their ability to recall every fertility event, but the events are cross-checked against birth records in the vital events database of Matlab. Thus, the mortality records should be unaffected by any recall bias. However, in terms of prenatal outcomes such as miscarriages or stillbirths that would not be recorded in the vital events database, there remains a small possibility that women are intentionally biased in the recall of their fertility history toward (or against) remembering stillborn male children compared to stillborn females, or they may have better recall for instances that happen near dramatic events such as the Famine. We discuss the potential implications of any such recall bias later in our results section.

Our goal is to examine the effects of the 1974 famine on birth and subsequent fertility outcomes. Examining the prices of rice in Figure 1 suggests that the most severe period of the Famine occurred between August 1974 and October 1975, when prices were more than 50 percent above pre-Famine levels. Thus, we first compare the birth outcomes for all live

births occurring between September 1974 and December 1975 to live births occurring in other periods between 1970 and 1980.[2] Children born within the September 1974 – December 1975 treatment window were *in utero* during the most severe part of the famine for at least one full month of their third trimester. A child born in September 1974 would have experienced the Famine for the full month of August 1974. Assuming an average gestation length of 38 weeks (266 days), a child born in December 1975 would have experienced the Famine for at least one full month of their third trimester. Thus, we exclude from this initial treatment group infants who were exposed to the Famine only in their first trimester and infants who were exposed only in their first and second trimesters. This definition of the treatment is guided by the famine literature that has provided evidence that malnutrition may be particularly harmful to fetuses during the last trimester of pregnancy (Lumey and Stein 1997). The timeline in Figure 2 shows the span of this treatment, as well as alternate treatment windows which we discuss in detail in section 4.

Our outcomes of interest include whether the child is male, whether the child died during the first 29 days of life (neonatal mortality), and whether the child died during the subsequent 1-12 months after birth (post-neonatal mortality). Table 1a presents the mean values of these key outcomes for children who were *in utero* during the Famine, the means for children who were *in utero* in other periods, and the difference between them. Children *in utero* during the Famine have significantly higher mortality rates in the first month after birth but have

[2]When the specific month of birth was not remembered or unavailable, fieldworkers often coded the birth as occurring during January. Thus, the data records an inaccurately large number of January births. The main specifications of this paper are estimated while including these January births. However, all of the results (available upon request) are qualitatively robust to the exclusion of all January births from the sample. In addition, 1970-1972 were also associated with bad crops, high food prices, and political turmoil. We estimate similar regressions where we limit the sample to the years 1974-1980 effectively eliminating the war, earlier famine, and hardship from the control group, and find similiar qualitative and quantitative results. These are available upon request.

similar post-neonatal mortality rates compared to non-affected children. Children exposed *in utero* are less likely to be male, although this difference is not statistically significant. The rates of miscarriages and stillbirths, however, are not statistically different between children who were in utero during the Famine and children who were not. There is no other statistically significant difference between children on any other observable characteristic, such as mother's age at first marriage or mother's age at birth.

In order to determine whether the Famine had a lasting impact on subsequent fertility, Table 1b compares key post-Famine fertility outcomes of women who became pregnant after a long period of exposure to the Famine (had a live birth, stillbirth, or miscarriage between January 1976 and July 1976) to all other women aged 15 and older who were not pregnant during the Famine. Women who experienced a pregnancy outcome between January 1976 and July 1976 would have become pregnant after at least nine months of exposure to the Famine but before the Famine itself was over. Thus, in contrast to the immediate effects we expect to see on children *in utero*, we hypothesize that malnutrition may take longer to impact the adult mothers themselves.

After separating women who were fertile during the Famine according to whether they became pregnant before the famine ended but still following at least nine months of Famine exposure, we then examine their post-Famine fertility outcomes for every pregnancy outcome that occurred subsequent to 1977. Limiting the post-Famine pregnancy outcomes to only those occurring after 1977 ensures that these outcomes are not affected by any direct impacts of the Famine itself, which had been over for a year by that time. Specifically, we are interested in whether the post-Famine pregnancies resulted in miscarriages or stillbirths. As seen in Table 1b, we find little evidence that women who became pregnant after exposure

to the Famine were more likely to have a post-Famine miscarriage. In contrast, the simple mean comparison suggests that women who were pregnant during the Famine were more likely to have a stillbirth in a future, post-Famine pregnancy and particularly more likely to have a male stillbirth.

The descriptive statistics also suggest that women who were pregnant during the Famine are positively selected on observables. Specifically, they have higher educational levels, are married to more educated husbands, and married at a younger age. Moreover, we find that they had a larger number of sons before the Famine. Although our empirical specifications control for these factors, Section 4.3 presents results that decompose the sample by spouse's education and land holdings. This analysis allows us to examine whether the results vary by socioeconomic background.

4 The Empirical Model

4.1 Children specifications

We start by estimating the following probit regression for children born between 1970 and 1980:

$$C_i = \alpha + \beta_1 \text{Famine}_i + \beta_2 YOB_i + X_i\delta + \epsilon_i \tag{1}$$

where C_i is the outcome of infant i. To investigate the impact of exposure to the Famine on the sex of the child, C_i takes the value of 1 if the child is male and zero otherwise. For neonatal mortality, C_i takes the value of 1 if the child died within the first 29 days after birth and zero otherwise, and for post-neonatal mortality, it takes the value of 1 if the child

died during the first 1-12 months of life and zero otherwise. Famine$_i$ is an indicator variable that equals 1 if the child was exposed to the Famine while *in utero* (i.e. was born between September 1974 and December 1975). In Table 4, we present estimates allowing for this exposure window to vary.

The vector X includes demographic characteristics of the mother, as listed in the data section. It also contains variables controlling for the relative birth weight of the infant and measures of prenatal and postnatal care, again as described in the data section.[3] In addition, we include in vector X controls for season of birth and whether the mother's village is part of the treatment group of a Maternal and Child Health and Family Planning services program operating in the area.[4] Furthermore, including a linear year of birth trend, YOB_i, ensures that β_1 measures the difference in outcomes for children born in the treatment window separate from the cohort trend (Almond 2006). Standard errors are clustered at the bari level to account for any serial correlation.

It is likely, however, that women who were pregnant during the Famine are different from women who did not get pregnant during the Famine on some important unobservable dimensions. Since many of the women in our sample gave birth to more than one child during the period of 1970-1980, we are able to include mother fixed effects, m_j, in equation

[3]Birth weight and access to prenatal care are potential channels through which *in utero* exposure to Famine may impact infant mortality. The results presented are similar in magnitude and significance when these variables are excluded.

[4]We include a dummy variable for the villages where the Maternal and Child Health Family Planning would be present even though that project did not begin until 1978 to account for potential differences between these villages even before the project began. We also estimate regressions without including these dummy variables and find nearly identical results, which are available upon request. Season of birth dummies include whether the child was born during the monsoon season (June-October) or the dry winter season (November-February), with the omitted category being the pre-monsoon hot season (March-May). Infant deaths are significantly higher during the winter season, largely due to agricultural cycles.

(1) and estimate the following OLS regression:

$$C_{ij} = \alpha + \beta_1 \text{Famine}_{ij} + \beta_2 YOB_{ij} + X_{ij}\delta + m_j + \eta_{ij} \tag{2}$$

where C_{ij} is the outcome of infant i born to woman j. The vector X in equation (2) includes similar variables included in equation (1), with the exception of mother's education, her age at first marriage, and her height. In order to ensure that our results are not spurious, we also report results from estimating equations (1) and (2) using births between 1960-1970 and between 1980-1990, where we assign 1964-65 and 1984-85 as placebo treatment years, respectively.

4.2 Maternal specification

The impact of exposure to the Famine on future fertility outcomes is estimated from the following probit regression:

$$M_{ij} = \alpha + \beta_1 FamMother_j + \beta_2 YOB_i + Z_j\gamma + X_{ij}\delta + \varepsilon_{ij} \tag{3}$$

where M is an indicator variable that equals 1 if a post-Famine pregnancy i resulted in a miscarriage or stillbirth for woman j, and zero otherwise. Our main treatment group in this specification differs from the one we considered for the child's outcomes. Specifically, the indicator variable $FamMother$ takes the value of 1 if the mother had any pregnancy outcome between January of 1976 and July of 1976 (miscarriage, stillbirth, or live birth). This birth window implies that the mother conceived between April of 1975 and October of 1975 and

would have been exposed to at least 9 months of the Famine before becoming pregnant, assuming an average gestation length of 38 weeks. Thus, we compare the likelihood of a miscarriage and stillbirth for women who became pregnant after a lengthy period of exposure to the Famine to women who did not become pregnant after their exposure to malnutrition. The vector X includes similar controls to those included in equation (1), with the exception of prenatal and postnatal healthcare indicators, which cannot be included in a sample of pregnancy outcomes that are not all carried to term. In addition to the total number of pre-Famine (prior to 1974) live male and female births for woman j included in X, vector Z includes post-Famine live births by sex (births after 1976), and spouse's education. In Table 6, we reproduce the results using different periods of exposure to Famine. Because $FamMother$ does not vary by pregnancy outcome, we cannot include mother fixed effects in this specification.

5 Results

5.1 Children findings

Table 2 presents the estimation results for equations (1) and (2). Looking at the implied marginal effects, children born between September of 1974 and December of 1975 are 2 percentage points less likely to be male (column 2), although this estimate is not significant at conventional levels. As one would expect, since the sex of a child can be considered random, the coefficient estimates for most of the controls listed in the previous section (not reported in the Table) are small and not statistically significant.

Columns 4 and 7 of Table 2 show estimation results of equation (1) using infant mortality at 1 month and between 1 month and 1 year as the outcomes. Marginal effects evaluated at the mean are shown in columns 5 and 8. Children born during the treatment period are 2 percentage points more likely to die within their first month after birth (a 32 percent increase over the mean) but have no difference in life expectancy between one month and one year. Consistent with the findings of Razzaque et al. (1990), exposure to the Famine *in utero* increased the probability of neonatal mortality. Although not presented in the paper, the controls in the regressions have expected signs. For instance, mother's age at birth is negatively correlated with mortality within 1 month of birth, and children who were reported as being smaller than average at birth have a higher likelihood of mortality.

It could be the case that mothers who opt to get pregnant during famine periods are unobservably different from mothers who do not. For instance, maybe more cautious mothers who are concerned for their child's health avoid becoming pregnant during food shortages.[5] To control for such traits, we include mother fixed effects in the regressions in columns 3, 6, and 9, thereby limiting the sample to children whose mothers had both a Famine-affected birth and a birth that was not affected within the sample period. The infant mortality results remain similar in both magnitude and significance to our original probit models when mother fixed effects are included. However, in contrast to the findings of Razzeque et al. (1990), the fixed effects specifications indicate a clear and significant decrease in the probability of male birth. Specifically, women who were pregnant during the Famine are four percent less likely to have a male birth, compared to women who were pregnant during the surrounding

[5] This type of selection implies that women who choose to become pregnant during a Famine are negatively selected. Based on observable measures, the descriptive statistics from Table 1b suggest the opposite; though the magnitude of the difference is not large, women who became pregnant during the famine were more educated and married spouses who were more educated, compared to women who avoided pregnancy.

years (significant at the five percent level). Thus, our results provide evidence in favor of the Trivers-Willard hypothesis that women are less likely to give birth to sons during lean times.

While the mother fixed effects capture relevant time-invariant characteristics of the mother, one lingering concern may be the possibility of non-random migration by males during the famine. We are unable to observe the presence of the father or to account for the pregnancies and births that did not take place because the father was absent. However, Kuhn (2005) finds that it is *single* men in Matlab who are most likely to migrate for work (rather than married men), as are men who own less land. We would be most concerned about this type of selection bias if our data showed larger famine effects on children from families with fewer landholdings. In a later section of the paper we show that it is *wealthier* families who face the largest impacts during the Famine, which leads us to conclude that selective migration is likely not a significant mechanism driving our results. Another concern could be that the famine changed access or availability of family planning methods. Although this is possible, it is unlikely in the Bangladeshi context. Access to family planning clinics and availability of contraceptives was extremely limited in Matlab prior to 1978 (Janowitz et al. 1997) and the government-run clinics providing counseling on family planning were mostly run by male workers and were often dirty and unsterile (Joshi and Schultz 2007).

To test whether our results are spurious, we conduct two sets of placebo tests. We estimate equations (1) and (2) using the same outcomes as the previous regressions, but redefining the treatment and sample around the years 1964 and 1984. Neither 1964 nor 1984 were affected by famine, war, monsoon, or other catastrophic events. The sample for the 1964 placebo test includes births between 1960 and 1970. For the 1984 placebo test, the

sample includes births between 1980 and 1990. The results of these analyses are presented in Table 2. Unlike our results for the Famine cohort, we find no significant difference between the likelihood of a male birth in the placebo treatment year compared to the other birth years in the samples. In addition, estimates from the infant mortality regressions remain small and insignificant, further supporting our findings that the 1974-75 famine is the driving factor behind the increased infant mortality observed in our main results. We do not believe that recall bias is a concern as our outcomes of interest are verifiable using the vital statistics registries.

As an alternative to placebo years, we also estimate each of our main specifications after incorporating decade-of-birth dummy variables interacted with the famine treatment window, using the full record of births covering 1919-1996. Results are qualitatively similar to our findings for the placebo years and are available upon request. We also estimate a regression that limits the sample to births between 1974 and 1980, since Bangladesh suffered a war and a lesser famine between 1970-1973. Results from those regressions are nearly identical to those presented here and are also available upon request. In other results not presented, we estimate whether the Famine increased the likelihood of miscarriage or stillbirth and find a small (2 percent) increase in the likelihood of miscarriage but no relationship with stillbirths.

Table 3 presents infant mortality results separately for male and female infants. The coefficient estimates suggest that the results from Table 2 are in fact driven by male mortality. The results given in columns 2 and 3 imply that male infants who were exposed to the Famine *in utero* were 3-4 percentage points more likely to die within the first month after birth. Female infants, on the other hand, do not have significantly different survival rates associated with famine exposure. The estimates using 1964 or 1984 as placebo famine years

do not indicate any positive relationship with infant mortality rates.

Table 4 varies the window of treatment in order to better understand the impact of exposure to malnutrition through the different stages of pregnancy. The first row repeats the main results from Table 2, while the second and third rows alter the length of the treatment, effectively increasing the amount of time the infant is exposed to the famine while in utero. The treatment window of births in the next row, between December 1, 1974 and October 31, 1975, means that the child would have been exposed to the famine for the full third trimester *and* at least one month of the second trimester. The treatment window of April 1, 1975 through October 31, 1975 means that the child would have been exposed to the famine during the entire nine months *in utero*. The results from Table 4 suggest that as the time of exposure to famine lengthens, the adverse impacts of malnutrition become worse, but possibly at a decreasing rate; the marginal effects for infant mortality at one month become slightly larger in magnitude when we include possible second-trimester exposure but become statistically insignificant when we examine the impact of being exposed for the full 9 months of pregnancy. Moreover, the likelihood of a male birth is consistently negative and statistically significant across the different exposure windows, but increases (in absolute value) only when we include possible second-trimester exposure. Thus, the results suggest that male fetuses are especially vulnerable to malnutrition during their first and second trimesters *in utero*.

Taken together, the results of Tables 2-4 suggest that famine exposure significantly decreases the likelihood of a male birth while increasing the likelihood of infant mortality by one month, particularly for male infants. However, the adverse effects of the famine appear to fade as the child reaches one year of age, since the famine coefficients on the likelihood of

death by one year are statistically insignificant.

5.2 Maternal findings

The previous estimations focused on the effect of the famine on children *in utero*. However, a famine pregnancy could also affect the mother. The next regressions estimate how experiencing a pregnancy after famine exposure is related to women's long-term fertility outcomes. The results from estimating equation (3) are reported in Table 5. As can be seen, we find no evidence of a relationship between exposure to Famine and future miscarriages. However, the results suggest that becoming pregnant after exposure to famine increases the likelihood of a stillbirth by about 2 percentage points (a 61 percent increase over the mean; see column 4). Since it is possible to identify the sex of a stillborn child, we estimate the number of stillbirths separately for male and female stillbirths. Though future female stillbirths are more common among women who became pregnant during exposure to Famine, this difference is not significant. In contrast, male stillbirths (which are always more common than female stillbirths) become significantly even more common among these women.

In a culture that prefers male children, it could be the case that this difference is driven by a bias in recall: male stillbirths may be more often remembered than female ones. To alleviate this concern, we estimate the same regression using placebo famines in 1964 and 1984, finding no evidence of an increased likelihood of male stillbirth among these cohorts. Furthermore, we do not believe that any recall bias in the timing of the stillbirths would be correlated with the Famine, since all these stillbirths took place after the Famine. It could be the case that there may be some recall bias on whether the mother experienced a

post-Famine pregnancy; however, since any such bias would mis-assign some treated women to the comparison group, it would only place a downward bias on our results.

To check the sensitivity of the results to the treatment window chosen in Table 5, Table 6 presents estimates of equation (3) where we vary the definition of exposure to the Famine. The first row repeats the original specification from Table 5 where treated women are those who had a pregnancy outcome between January 1, 1976 and July 31, 1976. The next two rows follow the same rule that the woman must become pregnant before the Famine is over, but reduces Famine exposure to 6 months (pregnancy outcome between October 1, 1975 and July 31, 1976), while the third row reduces time of exposure to the Famine to 3 months (pregnancy outcome between July 1, 1975 and July 31, 1976). The effect of the Famine on future stillbirths is consistently significant at the 1 or 5 percent level, with a marginal effect around 2 percentage points. Moreover, this effect becomes stronger when the fetus is male. For purposes of comparison, the last row of Table 6 restricts the treatment to the same window used for infants in Table 2. This window does not generate significant results, which suggests that malnutrition due to famine may take longer to affect adults than children. This result is consistent with the medical literature on malnutrition among adults (Collins 1995; Davis 1996).

5.3 The role of demographic characteristics

Finally, we investigate whether the results vary by family's wealth or education level. As reported earlier, the descriptive statistics suggest that women who became pregnant during the Famine may have been positively selected on observable characteristics. Unfortunately,

our data set does not provide direct information on wealth or income at the time of birth. However, our data includes information on land holdings by household in 1996 (the year of the survey). Since land is the largest household asset in Matlab, it provides a good proxy for wealth. Moreover, the inactive nature of land exchanges in the South Asian land market (at least during the time period of our data) enables us to make the reasonable assumption that households which report owning land at the time of the survey were also land holders at the time of their child's birth (see Pitt and Khandker 1998; Binswinger and Rosenzweig 1986; and Rosenzweig and Wolpin 1985 for more detailed descriptions of the land market). In fact, several papers have found turnover in land ownership to be so low that they have argued land may be used as an exogenous variable (Pitt and Khandker 1998).

Table 7 reports results on the impact of exposure to the Famine *in utero* by land holdings and years of schooling of the husband, another proxy for socioeconomic status. To facilitate the comparison, the first row repeats the results from Table 2. The second and third rows report the results for the top and bottom tercile in land holdings (measured in decimals), respectively. Although the results in column 3 suggest a negative association between exposure to *in utero* malnutrition and the likelihood of a male birth across the different socioeconomic groups, the results are statistically insignificant (perhaps due to the significantly reduced sample size). The results for infant mortality, however, do vary by land holdings. Specifically, we find that children born to mothers in the top tercile of land holdings are more likely to die within one month of birth compared to children born to mothers in the bottom tercile of the land holdings distribution. This pattern of results is consistent with the finding reported earlier that women who became pregnant during the Famine are more educated (when surveyed in 1996) and are married to more educated husbands compared to women

who avoid getting pregnant during the Famine. The results for infant mortality by husband's years of schooling are generally not statistically significant, but the magnitude of the results suggests that children of women with more educated spouses faced a higher likelihood of mortality, further supporting the idea that women who became pregnant during the Famine are positively selected in terms of wealth and education.

We also repeat the analysis of post-Famine pregnancy outcomes by land holdings and education. The results suggest that women from households in both parts of the land holdings distribution are more likely to have post-Famine stillbirths. Similar to the results in the full sample, the excess stillbirths are mainly driven by males. The magnitude of the results are slightly larger for women in the bottom tercile of the land holdings distribution, but the estimates between the two terciles are not statistically different from each other. Similar patterns are found when we analyze the results by terciles of the education distribution.

6 Discussion

Male infants who were exposed to the 1974 Bangladesh famine for at least one full month of their third trimester experienced higher rates of neonatal mortality as compared to their siblings who were not exposed. This result is robust to variations in the length of exposure to the Famine and is not present in alternative "placebo" famine years. There are, however, explanations aside malnutrition that could have contributed to the increase in neonatal mortality. For instance, it is possible that women who choose to become pregnant during a famine have unobserved factors that contribute to increased infant mortality. The siblings comparison, however, accounts for time-invariant factors related to the mother or the family

that could impact the health of the infant. Moreover, the effects for infant mortality are stronger for women with more landholdings, suggesting positive selection into motherhood during the Famine.

It remains possible that other factors such as limited access to quality care, increased maternal stress, increased burdens of infectious disease, and selective migration of spouses during pregnancy could have impacted infant mortality. However, the results with regard to infant mortality are robust to the inclusion of a set of variables that measure access to health care. Unfortunately, our data contain no information about retrospective stress measures, infectious diseases, or about the presence of the spouse during pregnancy. Thus, we remain unable to definitely rule out these alternative explanations or document how their impact interacts with the impact of reduced access to food.

Pregnant mothers during the 1974 Famine were also less likely to give birth to a male child compared to other live births they had during the period of 1970-1980. This result supports the Trivers-Willard (1973) hypothesis and contributes to a growing literature about its relevance to human populations. Although we are unable to identify the exact mechanism through which intrauterine malnutrition impacts the sex of the infant, the results suggest that male infants are particularly vulnerable to the *in utero* environment they face. Of course, the generalizability of this finding is limited, since it may be driven by specific factors unique to Bangladesh. Thus, further research about the sensitivity of male infants to intrauterine factors as well as the mechanisms through which they operate is warranted.

Finally, exposure to the Famine during pregnancy impacted not only the exposed infants, but also the post-Famine reproductive outcomes of mothers. Controlling for pre-Famine fertility, women who conceived after at least 9 months of exposure to the Famine experienced

a higher likelihood of a future male stillbirth compared to other fertile women who did not become pregnant during the Famine. As with the infant-related findings, these results are robust to variations in the length of exposure to the Famine and are not present in alternative "placebo" famine years. To our knowledge, this is the first study that documents a long-run impact of intrauterine exposure to malnutrition on subsequent pregnancy outcomes. These results, however, should be interpreted cautiously. In contrast to the analysis of infant outcomes, we are unable to account for time-invariant factors about the mother or the household. Instead, we rely on a comparison between women who conceived after a lengthy period of malnutrition and women who did not. Thus, we cannot rule out the possibility that at least part of the effects we document may be related to unobserved traits of the mother that impact her reproductive outcomes. Moreover, miscarriages are typically difficult to identify because they depend on the age at which a pregnancy is recognized which could also be impacted by the Famine. Nonetheless, these findings are relevant to the literature on the reproductive outcomes of women with eating disorders as well as for relief agencies operating in areas facing nutritional constraints.

In summary, the results of this paper suggest that malnutrition during famine is likely to have an especially adverse effect on both male infants and pregnant women. These impacts may be long-lasting, as they appear to affect women not only in their current pregnancy, but in their future pregnancies as well. An important area for future research may be to explore the mechanisms through which malnutrition impacts infant mortality rates and future fertility outcomes, as well as how it interacts with other factors such as access to health and maternal stress. A deeper understanding of the importance of different channels, whether biological or behavioral, would provide insight into how to mitigate the health effects

of famine episodes in developing countries.

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Figure 1: Average Retail Price of Coarse Rice in Bangladesh

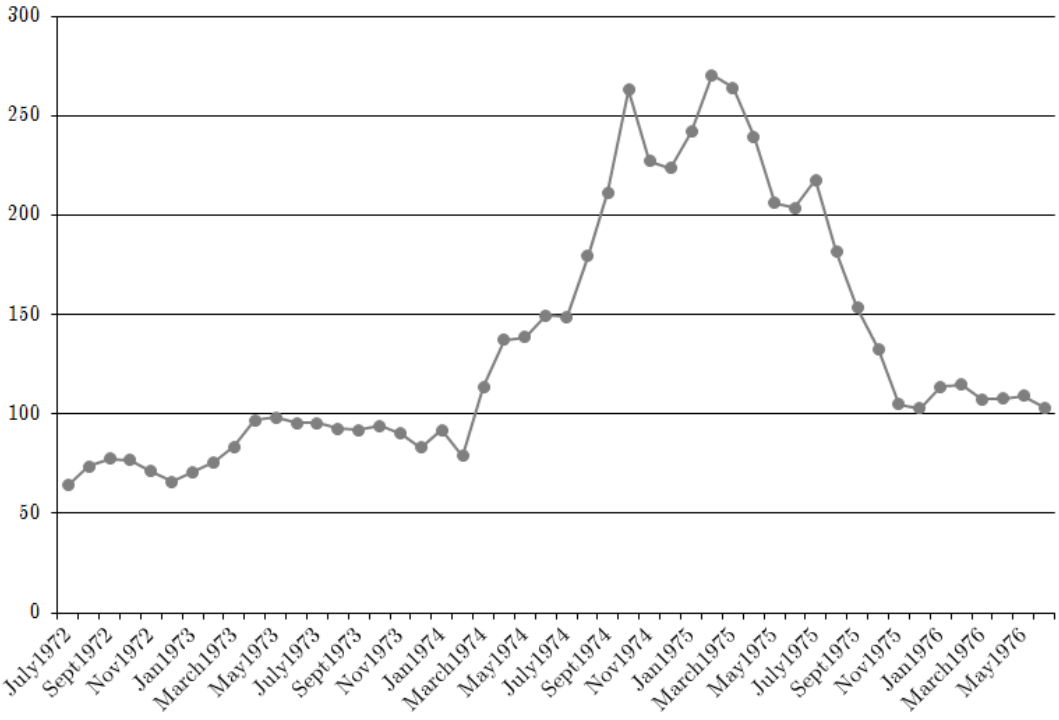

Figure 2: Famine and Treatment Timeline

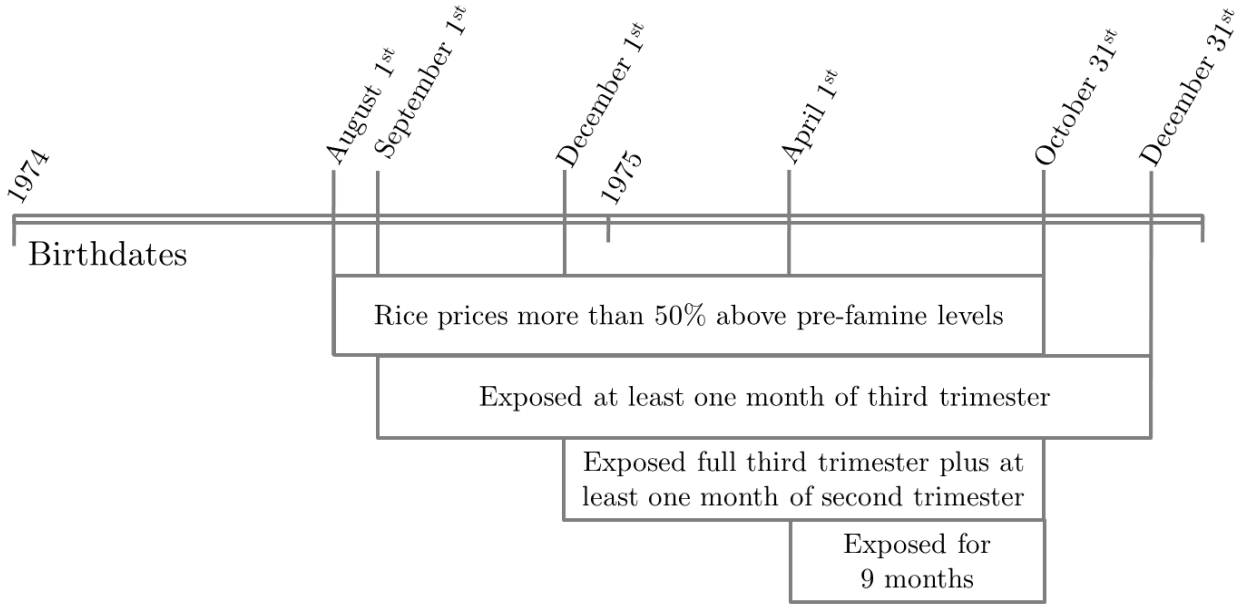

Table 1a. Descriptive statistics on the children

Child Outcome	In utero during Famine	N	In utero Other Years	N	Difference
Male	0.53	539	0.55	4,510	-0.02
	(0.50)		(0.50)		(0.02)
Death by 1 month	0.08	633	0.06	5,523	**0.02****
	(0.27)		(0.23)		**(0.01)**
Death between	0.05	581	0.05	5,202	0.00
1 mo. and 1 yr.	(0.22)		(0.21)		(0.01)
Miscarriage	0.04	588	0.03	4,825	0.01
	(0.19)		(0.16)		(0.01)
Stillbirth	0.03	588	0.02	4,825	0.01
	(0.16)		(0.14)		(0.01)
Mother's Education	1.43	539	1.36	4,510	0.07
	(2.45)		(2.36)		(0.11)
Mother's Age at	14.35	539	14.24	4,510	0.11
First Marriage	(4.69)		(3.40)		(0.16)
Mother's Age	25.71	539	26.25	4,510	-0.54
when Child born	(6.73)		(7.04)		(0.32)
Mother's adult	148.74	539	148.85	4,510	-0.10
height (cm)	(5.99)		(6.02)		(0.27)
Number of older	1.60	539	1.59	4,510	0.01
brothers	(1.53)		(1.60)		(0.07)
Number of older	1.45	539	1.49	4,510	-0.03
sisters	(1.50)		(1.50)		(0.07)

Standard deviations in parentheses for means. Standard error in parentheses for the difference.

Child outcomes include all live births 1970-1980. "Born during famine" = born between September 1974 and December 1975. * Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level.

The number of older brothers and sisters include only live births.

Table 1b. Descriptive statistics on the mothers

Maternal Outcomes	Experienced Famine Pregnancy	N	No experience of Famine Pregnancy	N	Difference
Post-Famine Pregnancy	0.06	712	0.06	4,760	-0.01
resulted in Miscarriage	(0.23)		(0.25)		(0.01)
Post-Famine Pregnancy	0.05	712	0.03	4,760	**0.02*****
resulted in Stillbirth	(0.54)		(0.54)		**(0.02)**
Post-Famine Pregnancy	0.06	356	0.03	2,204	**0.03*****
resulted in Male Stillbirth	(0.24)		(0.17)		**(0.01)**
Post-Famine Pregnancy	0.05	356	0.03	2,556	**0.02***
resulted in Female Stillbirth	(0.21)		(0.17)		**(0.01)**
Years of Education	1.88	712	1.31	2,760	**0.57*****
	(2.70)		(2.34)		**(0.10)**
Age at First Marriage	14.53	712	14.46	2,760	0.07
	(2.29)		(3.35)		(0.13)
Age when Child born	26.73	712	27.35	2,760	**-0.62****
	(6.69)		(6.97)		**(0.28)**
Spouse's Educ. (yrs)	4.13	712	3.52	2,760	**0.61*****
	(4.18)		(3.88)		**(0.16)**
Adult height (cm)	148.92	712	149.18	2,760	-0.26
	(5.36)		(6.13)		(0.24)
Number of sons	1.01	712	0.91	2,760	**0.61*****
born before Famine	(1.27)		(1.21)		**(0.16)**
Number of daughters	0.82	712	0.74	2,760	**0.10****
born before Famine	(1.02)		(1.14)		**(0.05)**
Number of sons	1.91	712	1.99	2,760	-0.08
born after Famine	(1.50)		(1.36)		(0.06)
Number of Daughters	1.80	712	1.98	2,760	**-0.18*****
born after Famine	(1.31)		(1.43)		**(0.06)**

Standard deviations in parentheses for means. Standard error in parentheses for the difference.

"Famine Pregnancy"= birth outcome (live, stillbirth, or miscarriage) between January 1976- July 1976.

*Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level.

The number of older brothers and sisters include only live births.

Table 2. Impact of Famine on sex ratio and infant mortality

	Likelihood of male birth			Death during first 0-29 days			Death during first 1-12 months		
	(1) Probit	(2) m.e.	(3) f.e.	(4) Probit	(5) m.e.	(6) f.e.	(7) Probit	(8) m.e.	(9) f.e.
In utero during Famine	-0.05	-0.02	**-0.04****	**0.19****	**0.02**	**0.02***	0.02	0.00	-0.01
(1970-1980 cohorts)	(0.06)		**(0.02)**	**(0.08)**		**(0.01)**	(0.01)		(0.01)
N	5,049		4,441	6,156		5,601	5,783		5,195
Placebo famines:									
In utero during 1964 "famine"	-0.05	-0.02	-0.04	-0.09	-0.01	-0.01	-0.06	-0.01	-0.01
(1960-1970 cohorts)	(0.08)		(0.03)	(0.10)		(0.01)	(0.1)		(0.01)
N	2,963		2,495	4,809		4,460	4,474		4,133
In utero during 1984 "famine"	-0.00	-0.00	-0.01	-0.04	-0.00	-0.00	-0.18	-0.01	-0.01
(1980-1990 cohorts)	(0.05)		(0.01)	(0.01)		(0.01)	(0.11)		(0.01)
N	5,998		5,254	6,349		5,611	6,034		5,256

*Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level. "*In utero* during Famine" means born between September 1, 1974 and December 31, 1975. Standard errors corrected for clustering at the Bari level are in parentheses. Marginal effects, labeled m.e., are evaluated at the mean. Regressions labeled f.e. include mother fixed effects.

All regressions include controls for year of birth linear trend, mother's education, age at marriage age at birth, age at birth squared, height in 1996, number of older brothers and sisters, indicators for size at birth, indicators for season at birth, the number of prenatal checkups, whether the child was breastfed, whether the child was born in a hospital, a clinic, or at home, whether there was a skilled care provider present, and an indicator for whether the mother's village is in the treatment group of a Maternal and Child Health Planning services program.

Table 3. Impact of Famine on infant mortality, by sex

	Death during 0-29 days						Death during first 1-12 months					
	Male			Female			Male			Female		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Probit	m.e.	f.e.	Probit	m.e.	f.e.	Probit	m.e.	f.e.	Probit	m.e.	f.e.
In utero during Famine	**0.34*****	**0.04**	**0.04*****	0.05	0.01	0.01	0.06	-0.00	-0.00	0.07	0.01	0.01
(1970-1980 cohorts)	**(0.108)**		**(0.16)**	(0.13)		(0.02)	(0.13)		(0.02)	(0.12)		(0.02)
N	3,236		2,296	2,965		2,068	3,014		2,080	2,740		1,863
Placebo famines:												
In utero during 1964 "famine"	-0.05	-0.01	-0.00	-0.16	-0.01	**-0.05****	-0.14	-0.01	-0.01	0.01	0.00	0.00
(1960-1970 cohorts)	(0.12)		(0.02)	(0.17)		**(0.02)**	(0.15)		(0.02)	(0.13)		(0.03)
N	2,486		1,874	2,297		1,737	2,286		1,696	2,141		1,584
In utero during 1984 "famine"	0.07	0.01	0.01	-0.19	-0.01	**-0.03****	**-0.41****	**-0.02**	-0.01	-0.02	-0.00	-0.00
(1980-1990 cohorts)	(0.11)		(0.02)	(0.12)		**(0.02)**	**(0.20)**		(0.02)	(0.12)		(0.02)
N	3,191		2,275	2,943		2,048	2,979		1,916	2,961		1,871

*Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level. "*In utero* during Famine" means born between September 1, 1974 and December 31, 1975. Standard errors corrected for clustering at the Bari level are in parentheses. Marginal effects evaluated at the mean are reported in columns 2, 4, 6, and 8. See notes to Table 2 for list of controls.

Table 4. Impact of Famine on sex ratio and infant mortality: Alternative measures of exposure

	Likelihood of male birth			Death during first 0-29 days			Death during first 1-12 months		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Famine exposure *in utero*:	Probit	m.e.	f.e.	Probit	m.e.	f.e.	Probit	m.e.	f.e.
At least 1 month of 3rd trimester	-0.05	-0.02	**-0.04****	**0.19****	**0.02**	**0.02***	0.02	0.00	-0.01
	(0.06)		**(0.02)**	**(0.08)**		**(0.01)**	(0.01)		(0.01)
Third trimester + at least 1 month of second trimester	-0.08	-0.03	**-0.06****	**0.26****	**0.03**	**0.03***	0.01	0.00	-0.00
	(0.07)		**(0.02)**	**(0.09)**		**(0.01)**	(0.11)		(0.01)
Full 9 months	-0.06	-0.03	**-0.06***	0.20	0.02	0.02	-0.13	-0.01	-0.02
	(0.09)		**(0.03)**	(0.15)		(0.02)	(0.19)		(0.02)
N	5,049		4,441	6,156		5,601	5,783		5,195

*Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level. Standard errors corrected for clustering at the Bari level are in parentheses. Samples include all births occurred between 1970-1980. Marginal effects evaluated at the mean are reported in columns labeled m.e. Mother fixed effects are included in columns labeled f.e. See notes to Table 2 for list of controls.

Table 5. Impact of Famine on post-Famine pregnancy outcomes

	Miscarriage		Stillbirth		Stillbirth male fetus		Stillbirth female fetus	
	(1) Probit	(2) m.e.	(3) Probit	(4) m.e.	(5) Probit	(6) m.e.	(7) Probit	(8) m.e.
Preg. outcome Jan 76 - July 76	-0.10	-0.01	**0.27*****	**0.02**	**0.35*****	**0.03**	0.20	0.01
(9 months of exposure, and then became pregnant)	(0.09)		**(0.01)**		**(0.13)**		(0.14)	
N	5,472		5,472		2,560		2,558	
Placebo famines:								
Preg. outcome Jan 66 - July 66	-0.02	-0.00	-0.17	-0.01	-0.11	-0.01	-0.19	-0.01
(1960-1970 cohorts)	(0.10)		(0.11)		(0.15)		(0.19)	
N	5,502		5,502		2,669		2,568	
Preg. outcome Jan 86 - July 86	0.02	0.00	0.01	0.00	-0.13	-0.01	0.20	0.01
(1980-1990 cohorts)	(0.10)		(0.12)		(0.18)		(0.17)	
N	3,862		3,862		1,765		1,799	

Robust standard errors in parentheses, clustered by bari. *Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level.

Marginal effects evaluated at the mean are reported in columns labeled m.e.

Controls include education, at at first marriage, age at child's birth, age at birth squared, height, indicator for whether mother resides in a treatment group village of a Maternal and Child Health and Family Planning services program, total sons born before famine (live births), total daughters born before famine (live births), spouse's years of education, total sons born after famine (live births), and total daughters born after famine (live births).

Table 6. Impact of Famine on post-Famine pregnancy outcomes: Alternative measures of exposure

	Miscarriage		Stillbirth		Stillbirth male fetus		Stillbirth female fetus	
	(1) Probit	(2) m.e.	(3) Probit	(4) m.e.	(5) Probit	(6) m.e.	(7) Probit	(8) m.e.
Preg. outcome Jan 76 - July 76	-0.10	-0.01	**0.27*****	**0.02**	**0.35*****	**0.03**	0.20	0.01
(9 months of exposure, and then became pregnant)	(0.09)		**(0.01)**		**(0.13)**		(0.14)	
Preg. outcome Oct 75 - July 76	-0.02	-0.00	**0.16****	**0.01**	**0.19***	**0.01**	**0.18***	**0.01**
(6 months of exposure, and then became pregnant)	(0.06)		**(0.08)**		**(0.11)**		**(0.11)**	
Preg. outcome July 75 - July 76	-0.11	-0.01	**0.22*****	**0.02**	**0.31*****	**0.02**	0.15	0.01
(3 months of exposure, and then became pregnant)	(0.07)		**(0.08)**		**(0.11)**		(0.12)	
Preg. outcome Sept 74 - Dec 75	0.03	0.00	0.07	0.00	0.12	0.01	0.08	0.00
(treatment window used for child outcomes)	(0.07)		(0.08)		(0.12)		(0.11)	
Obs	5,472		5,472		2,560		2,558	

Robust standard errors in parentheses, clustered by bari. *Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level.

Sample: all women fertile during the 1975 famine.

Controls include education, at at first marriage, age at child's birth, age at birth squared, height, indicator for whether mother resides in a treatment group village of a Maternal and Child Health and Family Planning services program, total sons born before famine (live births), total daughters born before famine (live births), spouse's years of education, total sons born after famine (live births), and total daughters born after famine (live births).

Table 7. Impact of Famine on sex ratio and infant mortality: subsamples by landholdings and spouse's education

	Likelihood of male birth			Death during first 0-29 days			Death during first 1-12 months		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
In utero during Famine:	Probit	m.e.	f.e.	Probit	m.e.	f.e.	Probit	m.e.	f.e.
Full sample	-0.05	-0.02	**-0.04****	**0.19****	**0.02**	**0.02***	0.02	0.00	-0.01
	(0.06)		**(0.02)**	**(0.08)**		**(0.01)**	(0.01)		(0.01)
N	5,049		4,441	6,156		5,601	5,783		5,195
Top tercile in land holdings	0.00	0.00	-0.03	**0.32***	**0.04**	**0.04****	-0.09	-0.01	-0.00
	(0.06)		(0.02)	**(0.13)**		**(0.02)**	(0.19)		(0.02)
N	1,809		1,609	2,155		1,991	1,989		1,849
Bottom tercile in land holdings	-0.00	-0.00	-0.14	0.18	0.02	0.03	0.19	0.02	-0.01
	(0.11)		(0.10)	(0.16)		(0.02)	(0.13)		(0.02)
N	1,451		1,239	1,827		1,626	1,717		1,505
Top tercile in spouse's schooling	0.10	0.04	-0.02	**0.30***	**0.03**	0.03	-0.00	-0.00	-0.00
	(0.09)		(0.04)	**(0.17)**		(0.02)	(0.17)		(0.02)
N	1,510		1,318	1,614		1,553	1,640		1,458
Bottom tercile in spouse's schooling	-0.10	-0.04	-0.04	0.12	0.01	0.01	-0.05	-0.00	-0.01
	(0.09)		(0.11)	(0.11)		(0.02)	(0.14)		(0.02)
N	2,160		1,911	2,751		2,529	2,565		2,326

*Statistically significant at the 0.10 level; **statistically significant at the 0.05 level; *** statistically significant at the 0.01 level.
Standard errors corrected for clustering at the Bari level are in parentheses. Samples include all births occurred between 1970-1980. Marginal effects evaluated at the mean are reported in columns labeled m.e.
Mother fixed effects are included in columns labeled f.e.
See notes to Table 2 for list of all other controls.

Table 8. Impact of Famine on post-Famine pregnancy outcomes: subsamples by landholdings and spouse's education

	Miscarriage		Stillbirth		Stillbirth male fetus		Stillbirth female fetus	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Preg. outcome Jan 76 - July 76:	Probit	m.e.	Probit	m.e.	Probit	m.e.	Probit	m.e.
Full sample	-0.10	-0.01	**0.27*****	**0.02**	**0.35*****	**0.03**	0.20	0.01
	(0.09)		**(0.01)**		**(0.13)**		(0.14)	
N	5,472		5,472		2,560		2,558	
Top tercile in landholdings	-0.24	-0.03	**0.30****	**0.02**	**0.37***	**0.02**	0.26	0.01
	(0.15)		**(0.15)**		**(0.21)**		(0.24)	
N	1,837		1,837		854		856	
Bottom tercile in landholdings	-0.07	-0.01	**0.40****	**0.04**	**0.55****	**0.05**	0.28	0.02
	(0.17)		**(0.18)**		**(0.22)**		(0.26)	
N	1,795		1,795		821		851	
Top tercile in spouse's schooling	0.14	0.02	0.19	0.01	**0.54****	**0.04**	-0.35	-0.01
	(0.15)		(0.17)		**(0.22)**		(0.32)	
N	1,555		1,555		751		697	
Bottom tercile in spouse's schooling	-0.18	-0.02	**0.31***	**0.02**	**0.61*****	**0.03**	0.09	0.00
	(0.15)		**(0.16)**		**(0.22)**		(0.24)	
N	2,426		2,426		1,120		1,157	

Robust standard errors in parentheses, clustered by bari. *Statistically significant at the 0.10 level;
statistically significant at the 0.05 level; * statistically significant at the 0.01 level.
Marginal effects evaluated at the mean are reported in columns labeled m.e.
Controls include education, at at first marriage, age at child's birth, age at birth squared, height, indicator for whether mother resides in a treatment group village of a Maternal and Child Health and Family Planning services program, total sons born before famine (live births), total daughters born before famine (live births), total sons born after famine (live births), and total daughters born after famine (live births). With the exception of regressions by spouse's education, all regressions also control for spouse's years of schooling.