LUND UNIVERSITY
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Master in Economic Development and Growth

The impact of *in utero* malnutrition on "lost" births and neonatal mortality:

Evidence from the 2002 food shortage in Malawi

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Abstract: This study aims to look at the link between *in utero* malnutrition and the survival probabilities of fetuses and newborns, using the 2002 food shortages in Malawi as an instrument for malnutrition. Concretely, it looks at differences in the probability of a born child being a male and in neonatal mortality between children *in utero* affected by food scarcity and those born right before and after the food shortages. The obtained results show that there exist a link between intrauterine nutrition and early mortality. Moreover, they suggest that malnutrition may lead to a process of positive selection as early as *in utero*. Firstly, children are less likely to be males when belonging to food shortage-affected cohorts. Secondly, children affected by food scarcity are less prone to die within their first month of life. Both phenomena suggest that the cohort of children born after having been affected by intrauterine malnutrition represent a positive selection of what the cohort would have been in absence of the nutrition shock. These results have several implications, the most relevant one being that nutritional interventions targeting pregnant women may be a very cost-effective way of enhancing the life paths of many individuals, especially in those countries that are still threatened by food insecurity today.

Key words: Food shortage, *in utero* malnutrition, neonatal mortality, maternal health

EKHM52
Master thesis, second year (15 credits ECTS)
May 2014
Supervisor: Luciana Quaranta
Examiner: Erik Green

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I. Motivation and Introduction

The importance of nutrition for human development is widely accepted. Malnutrition represents a real burden for societies suffering from it, placing a huge impediment to the fulfillment of the human potential and hindering the social and economic progress of nations. For that reason, the first of the Millennium Development Goals focuses on the eradication of extreme poverty and hunger. Achieving this goal is imperative to onset economic progress in less developed nations, but it is also of crucial importance to achieve other development goals like reductions in child mortality or improvements in maternal health (UN, 2000).

Fortunately, the 20th century saw the disappearance of famine in almost every spot of the globe. However the least developed regions of the world, basically South Asia and Africa, are still being threatened by food insecurity (O'Grada, 2007). Famines in the 20th and 21st century have been moderate by historical standards. Nonetheless, Devereux & Berge (2000) estimate that they have cost roughly 70 million lives and have resulted in millions of still-births and "lost" births. Moreover, these famines have had significant effects on the physical and psychological development of survivors (O'Grada, 2007).

Although famines are less common in nowadays world, episodes of food shortage are much more frequent in the least developed nations. Consequently, the levels of child and maternal malnutrition are still unacceptably high in many developing countries. According to UNICEF (2009), as much as 195 million children under five in the developing world are undernourished.

Nutritional deficiencies are especially harmful during a woman's pregnancy and during the first two years of a child's life (UNICEF, 2009). According to Black et al (2008), maternal and child undernutrition indirectly account for more than one third of all child deaths. Moreover, undernourished children who survive may become trapped in a vicious cycle of poor health and low development. Concretely, they are likely to have lower education attainments, worse labor market outcomes (Almond, 2006; Black, Devereux & Salvanes, 2007) and less physical development (Chen & Zhou, 2007).

Therefore, efforts should be directed to improve the nutritional status of these subpopulations.

The present study aims to look at the link between malnutrition and early health in poorly developed societies. Concretely, it looks at the impact of malnutrition during pregnancy on fertility outcomes, measured by the probability that a child born is a male, which has been used in the literature as an indicator of fetal selection, and on neonatal mortality for children that have been undernourished *in utero*. For that purpose, an acute food shortage that occurred in Malawi in 2002 is used as a natural experiment.

The specific features of Malawi make the country an interesting case of study. On the one hand, Malawi is among the poorest countries in nowadays world, presenting very low living standards and still being threatened by food insecurity. According to the UNDP (2007), malnutrition remains a problem in Malawi, with around 35% of the population being undernourished and undernutrition being the underlying cause of more than 50% of deaths among children under five. Therefore, Malawi is a good representation of the countries at risk of suffering the negative impacts of *in utero* malnutrition in the present, as well as a good proxy for past societies that have lived in the edge of the surviving line. On the other hand, Malawi is among the group of countries that have suffered acute food shortages during the 21st century. Moreover, given that the food shortage that affected Malawi in 2002 was highly concentrated in time and that its intensity varied significantly among districts, the Malawian food shortage appears as an ideal exogenous nutritional shock in order to derive causal inferences of its impacts.

In 2002, Malawi was ranked 165th out of 177 countries according to Human Development Index (HDI). The HDI considers three dimensions; income, health and education. The Malawian income per capita in 2002 was around 580 US$ per year (United Nations, 2002)[1]. This number translates to around 1.6 US$ per capita per day, an amount that is alarmingly close to the international poverty line set at 1.25 US$ per capita per day (Ravallion, Chen & Sangraula, 2009)[2]. In the health dimension, Malawi was among the worst performers of the world with a life expectancy at birth of only 37.8 years. Such life expectancy is extremely low even when compared with other

[1] The income per capita is reported in Purchasing Parity Power using US$ (PPP US$) of 2002.
[2] The poverty line is reported in Purchasing Parity Power using US$ (PPP US$) of 2005.

countries with a similar development status. For instance, in 2002 the set of countries categorized by the United Nations (UN) as low income countries had a life expectancy of 59.1 years. Moreover, Sub-Saharan Africa, the region with the lowest life expectancy in the world had an average life expectancy of 46.3 (UN, 2002). To put it in an historical context, it is worth pointing that 37.8 years of life expectancy at birth is below the life expectancy that was enjoyed in England back in 1841, when English men had a life expectancy at birth of 40.6 years (LSAP, 2012). Thus, Malawi is a good representative of the countries that are prone to suffer food disasters nowadays, as well as a good approximation of past societies with very low living standards.

In utero malnutrition has been shown to have negative impacts in many dimensions of the individual, such as survival, health, cognitive development or productivity (Almond and Currie, 2011; Almond, 2006; Black, Devereux & Salvanes, 2007), both in the short term as well as in the long term. The present research focuses on the issue of malnutrition and health. Concretely, it looks at the link between malnutrition on the one hand, and fertility outcomes and neonatal mortality on the other. An extensive body of research had shown that in past societies a strong link between malnutrition, proxied by high food prices, and demographic outcomes such as mortality and fertility existed in the short term (Allen, Bengtsson & Dribe, 2005). However, much less is known on this link in contemporaneous societies, which face a completely different situation due to increasing market integration and the presence of international aid which may significantly lower the severity of the nutritional shocks (O'Grada, 2007).

With the purpose of analyzing the link between undernutrition during pregnancy and fertility outcomes, birth trends are analyzed to see if it is the case that during the period of famine there was any fertility decline. Theoretically, two main mechanisms could be driving a fertility decline during periods of hardship. On the one hand, it could be the case that individuals decide to reduce or postpone fertility whenever they anticipate that a period of hardship is approximating. On the other hand, involuntary fertility reductions could also take place, through increases in the number of involuntarily interrupted pregnancies or reductions in fecundity (Allen, Bengtsson & Dribe, 2005). Then, the probability of a male birth among the children born alive is assessed, which can provide some idea on whether the sex ratio at birth is unbalanced in favor of girls for cohorts affected by intrauterine malnutrition. This second question is motivated by

the fact that several studies suggest that male fetuses are more vulnerable to negative shocks than female fetuses while *in utero* (Fukuda et al, 1998; Lyster, 1974; Zorn et al, 2002). Therefore, knowing how the sex ratio behaved for the cohort of children exposed to the food shortages can potentially provide a hint on whether there was positive selection taking place during the gestation stage. The presented results show a significant fertility decline during the food shortage, followed by a decreased probability of a child born being a male for the cohorts that were *in utero* exposed to malnutrition. Together, both results suggest that positive selection may have occurred for the cohorts that were *in utero* during the period of food shortage.

Following, the focus is shifted to mortality outcomes of born alive children to assess the impact of *in utero* malnutrition on early death among newborns. More specifically, the neonatal mortality is studied given that it has been shown to be less influenced by environmental factors than infant or child mortality, and therefore differences are more likely to be purely due to deficient *in utero* development (Behar et al, 1958). According to the results, exposure to malnutrition seems to have had a negative impact on neonatal mortality, with cohorts exposed to food shortages being significantly less likely to die within their first 28 days of life. Moreover, these effects are more prominent among male infants, especially in the areas that were hit the hardest by the food shortages. Again, this result is consistent with the idea of positive selection for the cohorts *in utero* exposed to malnutrition.

While looking at the impact of malnutrition during pregnancy on fertility and on neonatal mortality of *in utero* exposed offspring, this research also indirectly addresses an unresolved problem that arises in the literature assessing the long term effects of intrauterine shocks. Studies analyzing the relation between shocks *in utero* and later outcomes usually suffer from sample selection bias, as only those individuals that survived to adulthood can be studied. Intuitively, one would expect the survivors to be a positive selection of the whole cohort, potentially leading to underestimation of the real effect of intrauterine shocks. The results obtained in this study indicate that such positive selection could be taking place as early as *in utero*

The rest of the paper is organized as follows. Section II is devoted to review previous work concerning the link between *in utero* development and subsequent outcomes. Section III focuses more specifically on the mechanisms that drive the relations

analyzed in this study. First, it presents potential mechanisms that may relate food crisis to declines in fertility. Secondly, the link between intrauterine undernourishment and the probability of the *in utero* affected child being born a male is theoretically and empirically analyzed. Finally, a third subsection addresses the link between inadequate development during gestation and neonatal mortality. Section IV describes the evolution of the Malawian food shortages of 2002, as well as the validity of this nutritional shock as an instrument for undernutrition. Section V and VI describe the data and methodology used in the analysis, and Section VII presents the main results. A sensitivity analysis is then performed in section VIII. Finally, section IX presents the main limitations of the analysis, followed by its main conclusions and findings which are summarized in Section X.

II. Previous work

In the mid 20th century, epidemiologists though that the placenta protected fetuses from any nutritional damage that mothers could suffer (Almond & Currie, 2011). However, this belief was frontally challenged in the 1990s by David Barker when he pointed that, in order to learn about mortality patterns, efforts should be directed towards understanding the intrauterine environment (Barker, 1990).

The new approach triggered by David Barker is known as the "fetal origins hypothesis", which states that malnutrition during gestation may result on permanent damaging effects on the health of individuals. Concretely, it declares that lack of nutrition during the second and third trimesters of gestation leads to low birth weight and increasing probabilities of chronic diseases in adulthood (Barker, 1995). Following the same line, Ben-Shlomo and Kuh (2002) state in their "critical period model" that exposure to negative shocks while *in utero* cause irreversible damage in an individual's health, given that the gestation stage is the period with the fastest development of cells and organs.

The empirical work surrounding these theories has shown that early life conditions, and concretely *in utero* conditions, actually have permanent effects on the health of individuals along its whole life (Almond & Currie, 2011). Much of the work concerning the impact of early life conditions has focused on adulthood outcomes. Negative shocks suffered during gestation have been associated with lower education attainments and

worse labor market outcomes (Almond, 2006; Black, Devereux & Salvanes, 2007), lower socioeconomic status (Almond, 2006), more propensity to develop chronic diseases (Barker, 1995) and less physical development (Chen & Zhou, 2007).

Another approach has been to look at how negative shocks during fetal stages affect the reproductive health of *in utero* undernourished women. Using low birth weights as a measure of inadequate intrauterine nutrients supply, Elkhom et al (2005) find that insufficient nutrition in early stages is related to higher age at first birth for affected women. Lumey and Stein (1997) find that exposure to famine leads to lower birth weights and higher perinatal death for the offspring of *in utero* exposed women. Rickard et al (2010) find that crop yield during the year of birth reduces the viability and the survival chances of offspring of women that experienced high prices while *in utero*. Conversely, Quaranta (2013) finds no relation between high prices during the fetal stage of women and the sex ratio of their offspring.

Thus, the literature has convincingly shown that having proper nutrition during gestation may significantly improve the physical and psychological development of individuals, suggesting that probably the best time to intervene to improve individuals' life is during the gestation stage (Almond & Currie, 2011).

Besides affecting the long term health and survival prospects of individuals (i.e. scarring), negative shocks experienced during gestation may also increase fetal and early life mortality. Several studies have analyzed the short run effects of malnutrition during gestation.

For instance, using the 1944/1945 Dutch famine as an exogenous shock, Lummey (1992) looks at the relation between malnutrition *in utero* and birth weight of the exposed child, which is a good predictor of surviving probabilities for infants (Mosley, 1987). His findings reveal that *in utero* exposure to famine reduces birth weight for newborn females. Interestingly, the effect is restricted to those female children who were exposed to famine during the second or the third trimester of gestation, while no effect is found for children exposed during the first trimester. Hernandez et al (2013) look at the impact of intrauterine malnutrition on the sex ratio at birth and on infant mortality for famine-exposed cohorts, using the Bangladesh famine of 1974 as an exogenous shock. They find that children who were *in utero* during the famine had

higher probabilities of dying within its first year of life, while no significant effect is found for the probability of a born child being a male. De Waal et al (2006) use the Ethiopian drought of 2002/2003 to look at the impact of malnutrition on infant and child mortality. Although they find that both infant and child mortality were higher in drought affected areas, they conclude that the difference is due to underlying vulnerability of the affected zones, but not directly due to the drought. Akresh and Verwimp (2006) look at the effect of crop failure and civil war at the time of birth on height for age z-score for children under five in Somalia. They find that girls born after a negative shock exhibit lower height for age z-scores, especially in poor households, while boys do not show any significant variation. Finally, Hartwig and Grimm (2012) look at the short run effects of the Malawian famine of 2002 looking at child mortality and anthropometric measures of children under five. They do not find any significant effect of the food crisis on child mortality. However, their findings show that children from affected districts had higher weight-for-age and height-for-age z-scores in the aftermath of the famine, indicating that potentially a positive selection took place in earlier stages.

The present study aims to complement the above literature by assessing the relation between being exposed to nutritional stress while *in utero* and the survival chances of affected children, looking at the probability of a child born from a food shortage-affected cohort being a male, as a proxy for selective fetal mortality, and at neonatal mortality outcomes.

Indirectly, this research also addresses the potential problem of positive selection that studies looking at long term outcomes of intrauterine shocks may suffer from. In presence of a severe nutritional shock, survivors are likely to be a positively selected sample as those with poor health are the most prone to succumb to early death. Looking at birth outcomes and neonatal mortality can provide some insights on whether is it the case that positive selection works as early as *in utero* and during the first days of life. If positive selection takes place, when looking at adult outcomes, and even at outcomes of children, what one will be looking at is indeed at the outcomes of a selected sample consisting of the strongest individuals who managed to survive. As a result, the conclusions drawn from such analyses may underestimate the overall damages of intrauterine undernutrition. Additionally, in order to find significant effects of *in utero*

malnutrition, the impact on adult outcomes will need to be strong enough to overwhelm the effect of positive selection.

III. The mechanisms

III. A. "Lost" births

An extensive literature has shown that, historically, poor societies have reacted demographically to short term economic stress. In most extreme cases, sudden rises of food prices have lead to increases in mortality (Breschi, Fornasin & Gonano, 2005; Bengston & Dribe, 2005). However, adjustments in marriage and fertility have also been part of the mechanisms to overcome short term fluctuations (Allen, Bengtsson and Dribe, 2005). For instance, Oris, Alter and Neven (2005) find that in poor rural areas of Belgium, fertility was highly responsive to increases in prices during the 19th century. Campbell and Lee (2005) find the same relation between rising prices of rice and declining fertility when looking at different regions of Liaoning during the 18th century. Finally, Bengtsson and Dribe (2006) looking at Southern Sweden before the onset of the fertility transition find that fertility dropped among landless and semilandless families in years of moderate and high increases in prices. Moreover, the time pattern indicates that the mechanism driving the drop in fertility was deliberate fertility control when families expected a period of hardship.

Interestingly, Lee (2002) finds that poor developing societies in the 20th century followed similar patterns in terms of demographic responses to economic shocks as those found in poor historical societies. Looking at data on the major famines in Bangladesh 1974 and China 1959-1961, as well as on less severe food crisis in India, Japan and Taiwan, he finds a negative association between rising prices of grain and both nuptiality and marital fertility, as well as a positive relation between high prices and mortality in times of economic crisis. In the same line, Lindstrom and Berhanu (1999) find a decline in the probability of conception during years of famine and economic instability looking at the Ethiopian experience of the 20th century, while Agadjanian and Prata (2008) find that fertility drop in Angola during war times with the magnitude of the impact highly varying with the socioeconomic status of the women. So apparently, today's poorest societies seem to behave similarly as how poor historical societies did in the past in terms of demographic responses to short-term fluctuations.

In a situation of economic stress, understood as a period of rising prices with consequent lost of purchasing power, a decline in fertility could be driven by two different phenomena. On the one hand, individuals expecting a coming period of hardship may decide to intentionally control fertility, through postponement of birth and deliberated abortions. On the other hand, fertility could decline unintentionally due to the effects of the negative economic shock. Firstly, an increase in prices could lower fecundity through cessation of ovulation, reduction in sperm production or loss of libido, the effect of malnutrition appearing at least nine months after the nutrition shock. Secondly, fetal loss and spontaneous abortions could also rise as a response to food shortage, the effect appearing with at least a six month lag this time (Bengtsson and Dribe, 2006). Usually, it is difficult to disentangle deliberate from unintentional fertility reductions[3]. However, whether one or the other mechanism is the one driving the decline in fertility may have different implications for the composition of the cohort born during the period of economic stress.

Empirical evidence shows that individuals from higher socioeconomic groups are more likely to know about contraception methods as well as more likely to have easier access to them. Consequently, they are more prone to use contraception more effectively leading to more successful control of fertility than its poorer counterparts (Cleland, 1985; Gage, 1994; Livi-Bacci, 1986). On the other hand, empirical evidence from historical studies has also shown that individuals from higher socioeconomic strata are less likely to demographically respond to short term fluctuations than its poorer counterparts (Allen, Bengtsson and Dribe, 2005). Usually richer social groups enjoy greater adjustment mechanisms when facing a period of hardship. For instance, before having to resort to demographic adjustments they could sell available assets or borrow money. On the contrary, landless families and day laborers have traditionally been the strata with higher demographic responses to rises in food prices, as not much adjustment mechanisms are available to them (Bengtsson, Campbell & Lee, 2009). So even with lower accessibility to effective contraception, one could expect to be the lower social strata the ones controlling fertility more intensively. Therefore, if Malawians increased fertility control as a response to the expectations of a bad harvest

[3] Looking at the time-lag between the economic shock and the demographic response can be a way of knowing whether the reaction is driven by deliberate fertility control or if it is unintentional. However, the explicit separation of both phenomena is beyond the extent of this study.

in the months preceding the period of food scarcity, it is difficult to predict how the socioeconomic composition of the cohort of children born during and slightly after the crisis would be in comparison with the other cohorts.

In sharp contrast, if the mechanism in action is unintentional abortions and decreasing fecundity, one would expect the individuals from the lower socioeconomic strata to be unequivocally the more affected ones, as they are likely to have poorer underlying health conditions as well as to be the most affected by rising food prices. As a result, the cohort born during and slightly after a period of hardship should proportionally contain more children with richer background if the decrease in the number of births is unintentional.

Given the findings of previous studies, I expect fertility to be lower during the period of rising prices and food scarcity than in the previous and following years. Moreover, I expect the composition of the cohort to depend on the mechanisms driving the fertility decline. In presence of deliberate fertility control, I could expect the cohorts born during the period of food shortage to be either relatively poorer or relatively richer when compared to other cohorts, depending on which are the socioeconomic groups controlling fertility more intensively. In contrast, if the decline is driven by excessive fetal mortality, spontaneous abortions and declining fecundity I expect the resulting cohort to have a higher proportion of children from high socioeconomic status families. Finally, a mixed result could indicate that both mechanisms are cohabiting. It is therefore difficult to formulate a priori expectations.

III. B. Fetal mortality

Epidemiological studies have repeatedly shown that there exists a close link between fetal mortality and the secondary sex ratio of a society (i.e. the sex ratio at birth), through fetal loss being more common among male fetuses. The specific sex ratio at birth of a society depends on the primary sex ratio, which stands for the sex ratio at conception, and on the sex-specific fetal mortality (Tremblay, Vézina & Houde, 2003). On average, the sex ratio at birth for humans is 105 males per 100 females, (Sieff et al, 1990). However, it has been estimated that the sex ratio at conception is as high as 120 males per 100 females (McMillen, 1979). As a consequence, it is widely accepted that fetal mortality has a sex-specific dimension, with spontaneous abortions being relatively more common among male than female fetuses.

Apparently, events that put stress on individuals result in lower sex ratios at births for the affected populations. Fukuda et al (1998) report a significant decline in the sex ratio of live-born infants after the Kobe earthquake of 1995. Lyster (1974) finds sex ratios unbalanced towards girls when looking at environmental disasters and Zorn et al (2002) findings suggest that war can also lead to lower sex ratios looking at the 10 days War of Slovenia. In the same line, Catalano (2003) finds that economic depression may also result in secondary sex ratio reductions, looking at the economic collapse of East Germany in 1991.

Two different mechanisms have been pointed out as responsible for the link between stress and lower sex ratios at birth. The first one relates to the primary sex ratio and states that stress reduces sperm mobility in men, which results in reduced probabilities of their offspring being males (Fukuda et al, 1996). The second mechanism, focus on sex-specific patterns of spontaneous abortions. Spontaneous abortions are more common among male fetuses both in absolute number (Catalano & Bruckner, 2005) as well as in terms of sex-specific death rates (Mizuno, 2000).

Focusing on the second mechanism, it is thought that hormones induced by stress increase the probabilities of spontaneous abortions during the first stages of pregnancy (Hobel et al, 1999). Moreover, it has been argued in the literature that women who go through a gestation in stressful contexts spontaneously abort weak male embryos and fetuses more frequently than female embryos and fetuses (Catalano et al, 2005).

Two theories have been proposed to explain the link between maternal stress and higher *in utero* death rates for males. Trivers and Willard (1973) argue that it is a matter of natural selection. Pregnant women in stressful contexts would affect the gender of their child by aborting weak male fetuses to improve their chances of grandchildren, as weak sons produce fewer offspring that weak daughters. Although the idea of women being able to affect the gender of their offspring through selective abortion has been highly questioned by the academia, the statistical model beyond this interpretation is very useful. This model assumes that pregnant women abort fetuses which fall under a hypothetic threshold in a normal distribution of survivability of fetuses, and that presumably the males' distribution lies to the left of the females' distribution. In a situation of stress, the threshold shifts to the right, and therefore given the relative position of the males' and females' distributions, relatively more males are aborted. In

that scenario, the loss of male fetuses would have two main implications. Firstly, the sex ratio at birth should decline as the sex ratio of abortions rises. And secondly, males born from cohorts with lower sex ratios should survive longer on average as the weaker males of this cohort would have died already *in utero* (Catalano & Bruckner, 2005).

The second theory rejects the idea of maternal manipulation of the criterion for abortion and argues in favor of the sex ratio being determined by the damage caused by the stressful situations to mothers and consequently to their fetuses (Hobel et al, 1999). Under this framework, it is not the abortion threshold what shifts to the right, but the normal distribution of survivability of both females and males what shifts to the left. Again, the males' distribution presumably lies to the left of the females' distribution, and therefore, with the shift produced during the period of stress, a higher proportion of males fall below the abortion criterion. Consequently, the sex ratio of fetal death rates rises, making the sex ratio at birth to decline. As opposed to the previous model, in this case the predictions of further survival should be lower for males born from a cohort with lower sex ratios at birth, as the whole survivability distribution shifts to the left for fetuses gestated under stress (Catalano & Bruckner, 2005). Thus, the affected cohorts would be relatively weaker and would experience more early deaths when compared to non-affected cohorts. Given that the males' survivability distribution is thought to lie to the left of the females' distribution, the effect is expected to be more intense in males.

Therefore, and according to the existing evidence and theories, I expect the probabilities of a born child being a male to be lower for the cohort affected by intrauterine malnutrition in relation to the other cohorts, resulting in lower sex ratios at births. This relation would be justified by the fact that individuals facing food shortage are expected to be under more stress, as well as children *in utero* during the period of food scarcity to be worse nourished. Additionally, in presence of reduced sex ratios, I could expect either a higher or a lower survivability for males in the affected cohort, depending on the mechanisms behind the relative decrease of male births.

III. C. Neonatal mortality

A recurrent problem that arises when assessing the link between *in utero* malnutrition and later life outcomes is that in many contexts it is difficult to disentangle the effects of *in utero* negative shocks from the effects of the environment in which a child spend its

first years of life (Almond and Currie, 2011). In a lower extent, the same problem arises when looking at short term outcomes like child mortality.

Using neonatal mortality as the outcome of interest partly solves this problem. Neonatal mortality is defined by the World Health Organization (WHO) as deaths that occur from the first day of life but before the 28th day. As shown in Behar et al (1958), neonatal mortality is much less sensitive to environmental factors than infant or child mortality, and therefore it is a better indicator to analyze the link between *in utero* shocks and early life health. In fact, neonatal mortality is highly affected by the health of the mother during pregnancy, including its nutrition, as well as the maternal care during the gestation stage. Concretely, early neonatal deaths, defined as death occurring within the first seven days of live, are the most affected by complications rooted in pregnancy (WHO, 2006).

Another characteristic of neonatal mortality that has been repeatedly stated in the literature is that it is higher among male newborns than among females (McMillen, 1979; Naeye et al, 1972). Even in societies where there are sex preferences towards boys, neonatal mortality rates for females are lower than for males, while mortality rates of females exceed those of male in all other ages (Ruzicka & Chowdhury, 1978).

One of the explanations that have been proposed is that sex differentials in early neonatal mortality come from the postponement of late fetal mortality, which was already argued to be more persistent among male fetuses (Teitelbaum, 1971). Unlike adult mortality, sex differentials in early neonatal mortality are basically dominated by biological factors much more than by behavioral factor, with biological factors being highly influenced by feto-maternal risks (Drevenstedt et al, 2008). For instance, Waldron (1983) states that females may be less vulnerable to shocks in early life due to the immunoregulatory gens linked to the X-chromosome providing greater resistance to infectious diseases. Moreover, males are likely to be born in earlier gestational stages (Hall&Carr-Hill, 1982) and with respiratory systems less developed for a given gestational age (Torday et al, 1981), also influencing negatively its survivability chances. Other hypothesis state that the larger size and head circumference of male fetuses difficult the labor and delivery (Eogan et al, 2003), what could exacerbate the disadvantage that biologically males present in front of females in terms of neonatal

mortality. Therefore I expect the neonatal mortality rates in my sample to be higher for males when comparing them to the females.

Finally, and according to the literature, neonatal mortality should be higher for those babies that suffered a worse environment during its gestations. Consequently, a priori one would expect the cohorts that were *in utero* affected by the food shortage to present higher neonatal mortality rates that their non-affected counterparts. However, if the negative shock was so drastic that it significantly increased the spontaneous abortions and stillbirths, it could be the case that live births represent a positive selection of what the cohort would have been in absence of the negative food shock. But even in this scenario it is not clear whether one needs to expect a negative or a positive impact of the food shortage in neonatal mortality rates. As argued in the previous subsection, neonatal mortality is expected to be lower for the affected cohorts if the selection process of live births worked under a shift to the right of the abortion threshold. Conversely, an increase in neonatal mortality rates would be consistent with the surviving curves shifting to the left.

IV. Malawi: the food shortage of 2002

IV. A. The timing of the food shortage

In the 2001/2002 season, a minor maize production shortfall led to the most severe food shortage episode of Malawi's history, even when compared to the major drought of 1991/1992 and the 1949 famine of Nyasaland (Devereux, 2002a) [4].

The trigger of the food shortage was localized flooding in the Southern and Central regions of the country that resulted in a 32% reduction of the national maize production with respect to the previous season, which was an exceptionally good one (Devereux, 2002a). However, the maize deficit was not exceptionally sharp. Indeed, the production of maize of 2001 was 6% higher when compared to the 10-years national average (Devereux, 2009). The reasons why a minor production shock resulted in the major food crisis in the history of the country are rooted in the increasing vulnerability of the Malawian population together with a bad functioning of the institutions in place (Devereux, 2002b).

[4] Nyasaland was the name of Malawi before the independence of the country from Britain in 1964.

Still in April 2001, just before the harvest started, the official maize production estimates suggested that national consumption needs would be met with the coming harvest. As a consequence and based on the available information, during April and May 2001 the National Food Reserve Agency (NFRA) sold 35,000 metric tons to Mozambique and Kenya under the advice of the International Monetary Fund (IMF) (Devereux, 2002b).

In June 2001, after the failure of the harvest was materialized, the Famine Early Warning System Network (FEWSNET) predicted that Malawi would suffer a maize deficit of 273,000 metric tons that year (FEWSNET, 2001). Despite the good previsions of cassava and potato production, the government declared that it would buy 220,000 tons of maize from local producers and resell it to the population at affordable prices to cover part of the gap. However, when the Agricultural Development and Marketing Corporation (ADMARC) entered the market in July 2001 to purchase maize at an initial offer of MK3/kg it was already too late[5]. Private traders had already bought the little local supply of maize available in the market, making impossible for the ADMARC to find farmers willing to sell them the maize at the offered price. Even when the offer rose to MK12/kg the ADMARC was unable to find local sellers (Devereux, 2002a).

By August 2001, the maize reserve of the NFRA was already virtually zero, so the government decided to order 134,000 metric tons of maize to South Africa to be delivered from October to December 2001. However, by mid-March only 83,000 metric tons had reached the country (FEWS, 2002b). Additionally, the assistance call of the Ministry of Agriculture and Irrigation to international donors was declined (Devereux, 2000b). In absence of donor's aid, lack of local supply and the delay of the imports, Malawi was out of public food reserves until November 2001 (Devereux, 2002a).

Already in August 2001 NGOs working on rural areas reported unusually high maize prices and reduced food security. Peasants were selling livestock to buy food. In October, Save the Children (UK) provided evidence of food prices rising in Mchinji District (Taifour, 2002). But the government and the international donors did not react to these reports. In fact, in January 2002 the FEWSNET reported that Malawi would experience food surplus in the current season (FEWSNET, 2002a). It was not until

[5] MK stands for Malawian Kwacha, the currency of Malawi

February 27th 2002 that President Muluzi declared a state of disaster and international donors started to act (Devereux, 2002a).

But the response was late. Until the next harvest arrived, local markets continuously faced food supply shortages and rising prices, while ADMARC markets were rationed so only 10 to 15 kg per person per day could be purchased from them. Therefore, the population was restricted to two bad alternatives: the ADMARC markets where supply was erratic or local markets with unaffordable high prices (FEWS, 2002c).

To sum up, a minor production shortage in the maize harvest of 2001 resulted in the worst situation of food insecurity in the history of Malawi, which lasted from August 2001 until June 2002 when the next harvest started to be collected. International reports estimate that during the period of food shortage millions of Malawians faced starvation, especially in the interior districts of the Central and Southern regions (Menon, 2007). The World Food Programme estimated that 2.1 to 3.2 million people were severely affected by the food shortage, with the intensity of the impact having a remarkable socioeconomic gradient. Although official statistics of hunger-related deaths are not available, the estimates of independent researchers range from 300 to 3000 hunger-related deaths during the period of food shortage (Taifour, 2002; Devereux, 2002a). However, this crisis is likely to have had other effects beyond the rise in mortality given its severity and wideness.

IV. B. The food shortage as an instrument for malnutrition

In most cases, when testing an economic theory, it is not enough to show correlation. To the extent that the mechanisms are willing to be understood, the goal of the researcher should be to prove that one variable has a causal effect on another variable. In order to be able to isolate the causal effect of a variable, one needs to have a *ceteris paribus* situation (i.e. any possible third variable which can influence the relation between the independent variable and the outcome variable of interest needs to be held constant) (Wooldridge, 2012). Experimental designs probably represent the best strategy to prove causality between variables. However, experiments are rare and costly. A good alternative is to use natural experiments and an instrumental design. This methodology requires finding an exogenous event which can be used to instrument the independent variable, avoiding the endogeneity problems caused by omitted variables.

The potential endogeneity between malnutrition and survival chances of fetuses and infants is obvious. For instance, one would expect wealthier or more educated families to be better nourished, as well as being able to take better care of their children. More importantly, it could also be the case that certain unobservable characteristics of individuals, like special interest in health issues, make them more likely to have a better nutrition themselves as well as to have children less likely to die within its first days of life. Consequently, in order to obtain unbiased estimates of the causal impact of intrauterine nutrition on the survival probabilities of infants, the independent variable needs to be instrumented. Here, I argue that the food shortage of 2002 in Malawi can be used for that purpose.

In order to be valid as an instrument, the situation of food scarcity needs to be correlated to the intrauterine nutrition of the fetuses, while not being correlated to the outcome variable (i.e. probability of a born child being a male or neonatal mortality) except for the correlation driven through maternal malnutrition or *in utero* malnutrition respectively.

To start with, food scarcity is very likely to be correlated with the nutrition status of individuals, especially in contexts where the population have extremely low living standards as it is the case of Malawi. With lack of access to food at affordable prices, one would expect the nutrition status of individuals to deteriorate during the food shortage. A second condition is that the only channel through which the lack of affordable food would affect the probabilities of dying of fetuses and infants is its intrauterine nutrition. According to the revised literature, it seems to be the case that both sex ratio at birth and mortality within the first 28 days of life are highly determined by the intrauterine environment enjoyed by the fetus.

Using the food shortage of 2002 as an instrument has an additional advantage. Given that the food crisis was highly concentrated in time, it is possible to clearly distinguish between cohorts of individuals that were *in utero* affected by the food shortage from those that were not, which can be used as a comparison group in the analysis.

V. Data

The main data for the analysis come from the 2010 Malawi Demographic and Health Survey (2010 MDHS). The survey was implemented from June to November 2010 and among its objectives the most relevant one is providing data on fertility and early childhood mortality. The survey consists in a representative sample of 27,345 households, of which 25,311 were successfully interviewed. Within each selected household, all eligible women age 15 to 49 were interviewed.

The 2010 MDHS contains detailed information regarding the birth history of each eligible woman. For every child ever born alive, there is information on date of birth, sex of the child, age of the mother at birth, whether the child was part of a multiple birth and whether he was alive at the time of the interview. If the child died before the interview, the date of death is also provided. In addition, the survey contains information regarding sociodemographic characteristics of the mothers and the household they belong to.

The analysis focuses on children born between October 1999 and September 2004. Only those children for whom it is possible to identify its region of birth are included in the analysis. This selection includes every child born within the chosen time span, except for those whose mother migrated after they were born which account for 4031 birth and represent a 20% of the whole sample. To the extent that the two groups of children significantly differ among them, this could lead to sample selection bias, and consequently the results could not be generalized for the whole population. Table 1 compares the two groups.

The children from the two groups have radically different backgrounds. According to the statistics in Table 1, the migrants are a positive selection of the whole population. The mothers are more educated and more literate, younger and got married later. Moreover, migrants belong to relatively richer households, and in 2010 they were more likely to live in cities than non-migrants, suggesting that a great share of the migration can be considered rural exodus.

Unfortunately, the children whose mothers migrated need to be excluded from the analysis as there is no way to find out where they were born. However, it is worth keeping in mind that a subsample of relatively wealthier individuals was debarred. After

excluding them, the final sample consists of 14,566 children born between October 1999 and September 2004 for whom it is possible to identify their place of birth.

Table 1. Descriptive statistics for non-migrants versus migrants

	Non-migrants	Migrants	Difference	T-test	
Rural household	90.87%	67.75%	-23.12	**0.000**	***
Male headed household	75.42%	80.23%	4.81	**0.000**	***
Poor household	46.14%	27.87%	-18.27	**0.000**	***
Middle income household	22.18%	14.76%	-7.42	**0.000**	***
Rich household	31.68%	57.37%	25.69	**0.000**	***
Mother years of education	3.69	5.48	1.79	**0.000**	***
Literate mother	45.30%	59.56%	14.26	**0.000**	***
Age at first marriage	17.10	17.49	0.39	**0.000**	***
Age of the mother at birth	25.25	23.90	-1.35	**0.000**	***
N	**14,566**	**3,807**	**Total**	**18,373**	

"Migrant"= child from a mother who migrated after his birth

P-values of the T-test for difference in means under the heading of T-test.

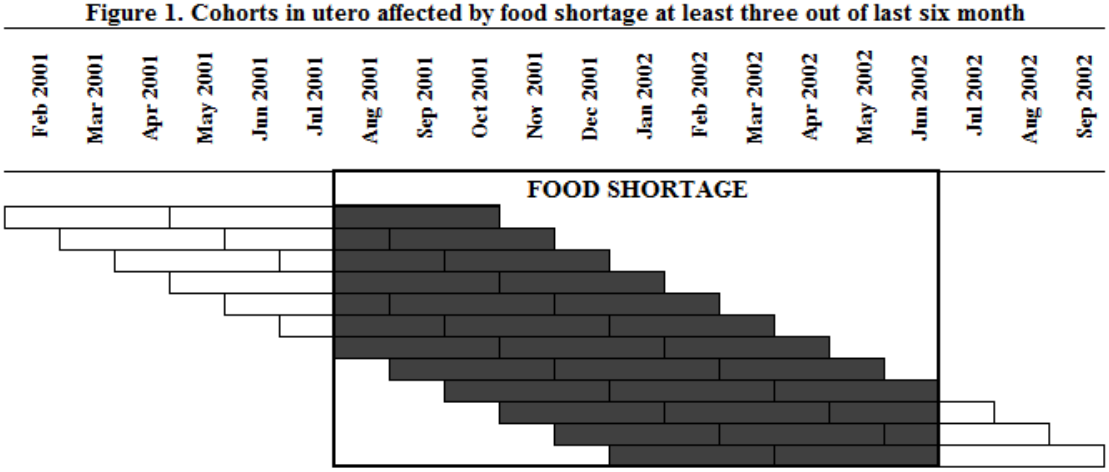
*** significant at 1%, ** significant at 5%, * significant at 10%

The children who are considered to have been malnourished while *in utero* are those who were exposed to the situation of food scarcity for at least three out of the last six month of gestation. The choice is motivated by the conclusions of epidemiological studies that affirm that the last two trimesters are the most important for the proper development of the fetus (Barker, 1995). As the period of food shortage lasted from August 2001 until June 2002, only children born between October 2001 and September 2002 lay within the definition of *in utero* affected by intrauterine malnutrition (Figure 1)[6]. Children born before and after these boundaries are used as comparison groups.

Thus, the selected sample contains the treatment group (i.e. the cohorts of children who were affected by the situation of food shortage while *in utero*) and two control groups. The control groups consist of children born within the two years preceding the births of the affected cohorts (i.e. born from October 1999 to September 2001) and those born during the two following years (from October 2002 to September 2004). Only children born in the years surrounding the period of food scarcity episode are considered because it is desirable that the cohorts to be compared are as similar as possible.

[6] This classification is made assuming that pregnancy lasts 9 months.

Figure 1. Cohorts in utero affected by food shortage at least three out of last six month



This selection of the treatment and control groups has some advantages as well as some disadvantages. Among the positive elements, none of the children born within the considered time span and for which it is possible to know its place of birth is excluded from the analysis. This has a positive impact on the sample size, improving the power of the analysis. Additionally, it assures that treatment and control groups are as similar as possible.

However, this classification also has some disadvantages. First of all, the control group includes children that although not exposed during three full months within the last two trimesters of gestation, they are in fact exposed during other stages, what potentially could contaminate the outcomes of the control group. For instance, children born between July and September 2001 were exposed to the food shortage during its first month of life, what could increase its probabilities of neonatal death. On top of that, cohorts of children born in the three months following September 2001 were exposed to food scaricuty during their first trimester of gestation, when fetal selection is the strongest, what could affect both the number of births and the sex ratio at birth. Secondly, within the treatment group there are individuals who were exposed during the whole gestation and others who were only exposed for three months. In this scenario, it is plausible that individuals with different time of exposure have different impacts. For these reasons, in Section VIII the specification of the groups is modified to see whether the results are sensitive to the definition of the treatment window or to the restriction of the control group.

Table 2 compares socioeconomic characteristics for the cohorts affected by the food shortage while *in utero* versus the non-affected cohorts. As shown, the affected children are not systematically different from those children who were not affected, at least in observable characteristics. Both were born predominantly in rural areas and belonged mostly to families headed by males. Regarding the characteristics of the mother, in both groups mothers have little more than 3.5 years of education and got married at around the age of 17. Concerning the specific birth, children born from the affected cohorts do not significantly differ from children of other cohorts in terms of their birth order or the age of the mother at their birth.

The only socioeconomic variable in which the cohorts significantly differ is the percentage of born children belonging to the lowest socioeconomic status. For the affected cohort, only 43.88% are from the lowest socioeconomic group, while for the non-affected groups this percentage is 46.62%. In contrast, the children affected by malnutrition while *in utero* are more likely to belong to the highest socioeconomic group, although the difference is not statistically significant in this case.

Table 2. Descriptive statistics for children *in utero* non-affected versus *in utero* affected

	In utero non-affected	*In utero* affected	Difference	T-test	
Rural household	91.07%	89.89%	-1.18	0.216	
Male headed household	75.53%	74.87%	-0.66	0.556	
Poor household	46.62%	43.88%	-2.74	**0.024**	**
Middle income household	21.97%	23.17%	1.20	0.267	
Rich household	31.41%	32.94%	1.53	0.213	
Mother years of education	3.70	3.65	-0.05	0.511	
Literate mother	45.65%	43.62%	-2.03	0.114	
Age at first marriage	17.10	17.10	0.00	0.947	
Age of mother at birth	25.55	25.62	0.07	0.63	
Multiple birth	3.93%	3.88%	-0.05	0.943	
Number of dead siblings	0.40	0.43	0.02	0.232	
Birth order	3.56	3.55	-0.01	0.916	
Male child	50.37%	49.75%	-0.62	0.665	
Neonatal death	25.03 ‰	17.16‰	-7.78	**0.025**	**
N	**11,989**	**2,577**	**Total**	**14,566**	

"*In utero* affected" = children born between October 2001 and September 2002

P-values of the T-test for difference in means under the heading of T-test.

*** significant at 1%, ** significant at 5%, * significant at 10%

Regarding the outcome variables of interest, it is worth noting that children are less likely to be males when belonging to the affected cohorts, even though this difference is not statistically significant. In contrast, the two cohorts significantly differ in what neonatal mortality respects, with the children from the affected cohorts being less likely to die within their first 28 days of life.

The presented results insinuate that the food shortage may have driven a positive selection process among fetuses while *in utero*. First of all, the children from the affected cohorts are less likely to be from the poorer socioeconomic group. This phenomenon could be basically due to two different reasons. On the one hand, it could be the case that individuals in the lower part of the wealth distribution voluntarily decide to control fertility when they expect a bad harvest. In a very poor country like Malawi, this subpopulation is always living critically near to subsistence levels and therefore being aware that they are unable to cope with intense negative shocks, they could decide to postpone fertility whenever the harvest expectation are specially bad as it was the case in Malawi in 2001. On the other hand, it could also be a matter of natural selection. Under this hypothesis, the weaker fetuses would not survive gestation and therefore only the healthier children would survive until birth. Intuitively, one would expect the women from lower economic status to have worse health and its fetuses to be weaker and worse nourished, and consequently less likely to survive the whole gestation period.

Secondly, and related to the hypothesis of natural selection, the fact that children in the affected cohort are less likely to be males could potentially be an indicator of selection. As it has been already argued, males are thought to be more vulnerable than females during while *in utero*. Consequently, a population showing sex imbalance in favor of girls could indicate that it has existed some natural selection in the gestation stage.

Finally, in a context of food shortage, one would expect the health of the population to deteriorate and thus the premature mortality to increase. However, the children from the affected cohorts are significantly less likely to die within the first month of live when compared to their counterparts in unaffected cohorts. Therefore, this also insinuates that the final affected cohort could be a positive selection of what the cohort would have actually been in absence of the negative nutrition shock.

To have a better insight of the effects of the nutrition shock on the health of fetuses and infants, the whole country can be divided between districts highly affected by food scarcity and districts in which the food shortage was more moderate. This division is done by looking at the average price of maize in 23 markets from 20 different districts during the season 2001/2002 obtained from the National Statistics Office of Malawi. Whenever data on the price of maize in the main food market was available, this price was imputed to the whole district. Secondly, in cases in which the price of the main market was not reported, the price of secondary markets was used instead. Finally, for the remaining 8 districts, for which there was no data, the price of the closest market from the region's main food market was used.

Once every district had an imputed average price of maize for the season 2001/2002, a threshold of 18MK/kg was used to divide the districts according to the severity of the food shortage. All the districts with a seasonal average price of maize over 18MK/kg are classified as highly affected districts, while districts with average price under 18MK/kg are considered moderately affected districts. The cut-off used to divide the two groups is justified by the fact that the NFRA was selling maize at a price of MK17.49/Kg, which was considered to be an adequate price in order to make the food accessible for the general population (Devereux, 2000a). Appendix A provides a map of Malawi in which the districts are marked depending on whether they are considered as treatment (i.e. highly affected) or as control (i.e. moderately affected) areas according to this criterion.

The resulting distribution is reasonable. First of all, it matches qualitative reports on the food crisis (Menon, 2007). Secondly, the districts classified as control zones are concentrated in the Northern region, which was the least affected by the floods, districts containing the main cities of the Central and Southern regions (Lilongwe and Blantyre respectively) and the districts in the Central and Southern regions which border with Mozambique and Zambia and therefore have lower transport costs for the imports, making the access of food to the area easier.

Even if this classification looks reasonable, the choices made to construct it have implications. First of all, to the extent that maize prices are not substantially correlated over space, the methodology to impute prices to districts with missing data according to the proximity to other markets may be deficient. Secondly, although the threshold

chosen to divide the districts between highly and moderately affected areas seems reasonable and justifiable, it is interesting to see whether the results depend significantly on the chosen cut-off. Consequently, other classifications in which either the threshold or the classification of critical districts is slightly varied would also be considered in Section VIII, which contains the sensitivity analysis.

Finally, it is also important to compare the children from the treatment and control districts in order to know if the underlying conditions of both groups differ, as most likely the consequences of food shortage would highly depend on the initial situation of the population.

Table 3. Descriptive statistics for moderately affected versus highly affected districts

	Moderately affected districts	Highly affected districts	Difference	T-test	
Rural household	82.72%	96.59%	13.87	**0.000**	***
Male headed household	77.66%	73.84%	-3.82	**0.008**	***
Poor household	38.97%	51.18%	12.21	**0.000**	***
Middle income household	20.66%	23.25%	2.59	**0.062**	*
Rich household	40.37%	25.56%	-14.81	**0.000**	***
Mother years of education	4.24	3.31	-0.93	**0.000**	***
Literate mother	47.99%	43.40%	-4.59	**0.043**	**
Age at first marriage	17.30	16.96	-0.34	**0.001**	***
Male child	51.33%	49.50%	-1.83	**0.078**	*
Neonatal death	22.97‰	24.12‰	1.16	0.733	
N	**6,677**	**7,889**	**Total**	**14,566**	

"Highly affected region"= season mean price of maize over 18MK

P-values of the T-test for difference in means under the heading of T-test.

*** significant at 1%, ** significant at 5%, * significant at 10%

Table 3 compares children from severely affected districts with children from moderately affected districts, independently on whether they belong to an affected or an unaffected cohort. The results show that children from the districts that were hit the hardest by the food shortage are significantly poorer and have lower socioeconomic status than those children in the districts that were only moderately affected. Children from the treatment areas are more likely to live in rural areas and to be part of households headed by females. Also, their mothers are significantly less literate, have less years of education and got married earlier. All these indicators insinuate that the districts that were affected by food scarcity are also the poorest districts of the country.

In fact, when looking at the socioeconomic status of the household to which the children belong, it results that in highly affected districts the children are much more likely to belong to the lowest social strata and much less likely to belong to the richest classes. The fact that both areas are so different would have implications for the choice of the model as well as for the interpretation of some results.

Another interesting result from comparing the two groups of districts is that in highly affected districts children are less likely to be males. This provides some support to the hypothesis that looking at the sex ratio at birth can be a good indicator of the impact of bad economic conditions for Malawi in the period under analysis, because it is the case that poorer areas have sex ratios that favor girls. No difference in neonatal mortality is found between the two sets of districts.

VI. Methodology

To analyze the impact of *in utero* malnutrition on subsequent short term outcomes of the fetuses, I will basically look at sex ratios at birth, through analyzing the probability of a newborn being a male, and neonatal mortality outcomes. In order to be able to draw casual relations, the food shortage of 2001/2002 will be used as an instrument for *in utero* malnutrition. The food shortage is considered as an exogenous negative shock on nutrition that only affected those children that were *in utero* during the food crisis, while children born right before or after remained untreated. Therefore, by using the food shortage as a measure of malnutrition instead of other indicators such as weight at birth it is possible to overcome endogeneity problems arising from correlation between unobservable characteristics of the children and the outcome variables.

As a preliminary analysis of the data, I look at how the number of births evolved within the period under study, thus from October 1999 to September 2004, to see whether the nutrition shock resulted in a fertility reduction. If this was the case, it could indicate that some kind of selection may have taken place for the affected cohort.

Afterwards, I look at whether there was a sex imbalance in favor of girls for the cohorts of children that were *in utero* during the food shortage. With that purpose, I estimate the following logit model on the probability of a born child being a male:

$$M_i = \alpha + \beta_1 \, Food_i + \gamma \, YOB_i + X_i \, \delta + \varepsilon_i \qquad (1)$$

In this model Mi equals one if the child is a male, and zero otherwise. Thus, the model indicates how a set of independent variables affects the odds of a child being a boy. $Food_i$ is a variable which equals one if the child belongs to a cohort that was exposed to food shortage while *in utero*, and zero if the contrary is true. Therefore, β_1 is the parameter of interest, as it captures the effect of malnutrition on the odds of the born child being a boy. In order to control for other characteristics that could influence the probability of a child being boy, a vector of controls (X) is added to the regression. The set of controls includes household characteristics, such as whether the household belongs to a rural area, through a dummy variable that equals one if the child was born in a rural area, whether the district is considered to have been highly affected by the food shortage, with the variable being equal to one if the district was highly affected and zero otherwise, the sex of the head of the household, a variable that takes value one if the head is a male and zero if the head is a female, and the economic status of the family, which is a categorical variable that equals one for the poorest individuals, two for the individuals with medium income and three for the richest strata. In addition, it includes variables referring to specific characteristics of the mother, such as whether she is literate, through a dummy equaling one for literate women, the number of years of education that she received, and its age at first marriage in years, which is an additional indicator of the socioeconomic status of the mother given that women in Malawi tend to get married earlier the poorer they are. Finally, also some controls are included referring to the specific birth: the birth order of the child, the age of the mother at birth and its square in order to control for non-linear effects, how many sibling have died before the birth of the child and a dummy variable indicating whether the child was part of a multiple birth. Finally, a linear year of birth trend (YOB_i) is included to control for cohort trends.

Then, another model is estimated with the purpose of seeing if the relation between being from a food shortage-affected cohort and the probability of being born a boy depends on the severity of the shock. To see the potential heterogeneous effects among differently affected areas, an interaction variable between being from a food shortage-affected cohort and being from a highly affected district is included in the model:

$$M_i = \alpha + \beta_1 Food_i + \beta_2 High_i + \beta_3 (Food_i * High_i) + \gamma YOB_i + X_i \delta + \varepsilon_i \qquad (2)$$

In principle, whether a district was highly or moderately affected by the famine should not depend on the characteristics of its population, and thus no correlation should exist between being from a highly affected area and individual characteristics. Periods of acute food shortage can be considered as exogenous shocks as they normally depend on extreme weather-related events, wars or institutional failures, and therefore being treated (i.e. being exposed to the situation of food shortage) should not depend on the specific characteristics of individuals but on an event that occurs at a macro level. Nonetheless, in the case under study the individuals from the highly affected areas are significantly different from their counterparts in moderately affected areas. As was shown in the previous section when looking at the descriptive statistics in Table 3, children from highly affected areas are likely to belong to households with a poorer background, to live in rural areas and to have mothers with lower literacy and educational attainment.

The fact that the affected and unaffected districts are systematically different has two implications. First of all, it implies that the treatment and control groups are not statistically equal, and therefore that it is difficult to use a difference-in-difference approach in order to estimate the impact of *in utero* malnutrition on the health of children. Secondly, it also implies that when looking at the heterogeneous effects among districts, it is possible that some of the differing effects are due to different underlying conditions between affected and unaffected districts, and not purely to the intensity of the food shortage. In order to minimize such confounding effects, it is necessary to include socioeconomic variables in the regressions in order to control for the different underlying characteristics, and thus be able to isolate as much as possible the impact of the nutrition shock.

Once the problem of potential sample selection before birth has been analyzed, the next model looks at the link between suffering malnutrition while *in utero* and the subsequent health of the child, proxied by its probability to die within its first month of life. For that purpose the following logit model is estimated:

$$D_i = \alpha + \beta_1 \, Food_i + \gamma \, YOB_i + X_i \, \delta + \varepsilon_i \qquad (3)$$

where D_i is an indicator of neonatal death for child i, which takes the value of 1 if the child i died within its first 28 days of life and zero otherwise. With regard to the

independent variables, the ones included are exactly the same as in the model assessing the probability of a male birth with the exception that this model also includes a variable indicating whether the child is a male or a female, through a dummy that equals one for male children. Thus, β_1 is again the parameter of interest, as it captures the effect of being affected by the acute food shortage on the probability of a child dying within its first month of life.

Then, a series of models are constructed with the purpose of identifying regional and gender heterogeneous effects of being *in utero* affected by malnutrition on the probability of dying within the first 28 days of life. The first of these models includes an interaction term between belonging to an affected cohort and living in a highly affected area:

$$D_i = \alpha + \beta_1 \, Food_i + \beta_2 \, High_i + \beta_3 \, (Food_i * High_i) + \gamma \, YOB_i + X_i \, \delta + \varepsilon_i \qquad (4)$$

in which β_3 shows whether the impact of food shortage on the probability of neonatal death depends on the intensity of the food crisis in the district of birth.

The second model aims to look at distinct effects depending on the sex of the child. With that intention, an interaction between gender and food scarcity is added in the original model:

$$D_i = \alpha + \beta_1 \, Food_i + \beta_2 \, Male_i + \beta_3 \, (Food_i * Male_i) + \gamma \, YOB_i + X_i \, \delta + \varepsilon_i \qquad (5)$$

A last model aims to look at how these heterogeneous effects combine with each other, which requires a set of four interactions to be included:

$$D_i = \alpha + \beta_1 \, Food_i + \beta_2 \, High_i + \beta_3 \, Male_i + \beta_4 \, (Food_i * High_i) + \beta_5 \, (Food_i * Male_i) + \beta_6 \, (High_i * Male_i) + \beta_7 \, (Food_i * High_i * Male_i) + \gamma \, YOB_i + X_i \, \delta + \varepsilon_i \qquad (6)$$

The main problem with the specified models is that interpretation can be hard, especially for interaction terms. Interaction terms in non-linear models are difficult to interpret, as the intuition from linear models is not extensible to them. This means that in a logit model the marginal effect of a change in both interacted variables does not equal the marginal effect of simply changing the interaction term. Even more, the sign of the marginal effect may differ for different observations and the statistical significance cannot be derived from the z-statistic linked to the coefficient of the

interaction term. As a consequence, in non linear models in order to compute the magnitude of the interaction effect the cross derivative of the expected value of the dependent variable needs to be computed. Moreover, the significance test of the interaction term must be based on the estimated cross-partial derivative (Norton, Wang & Ai, 2004).

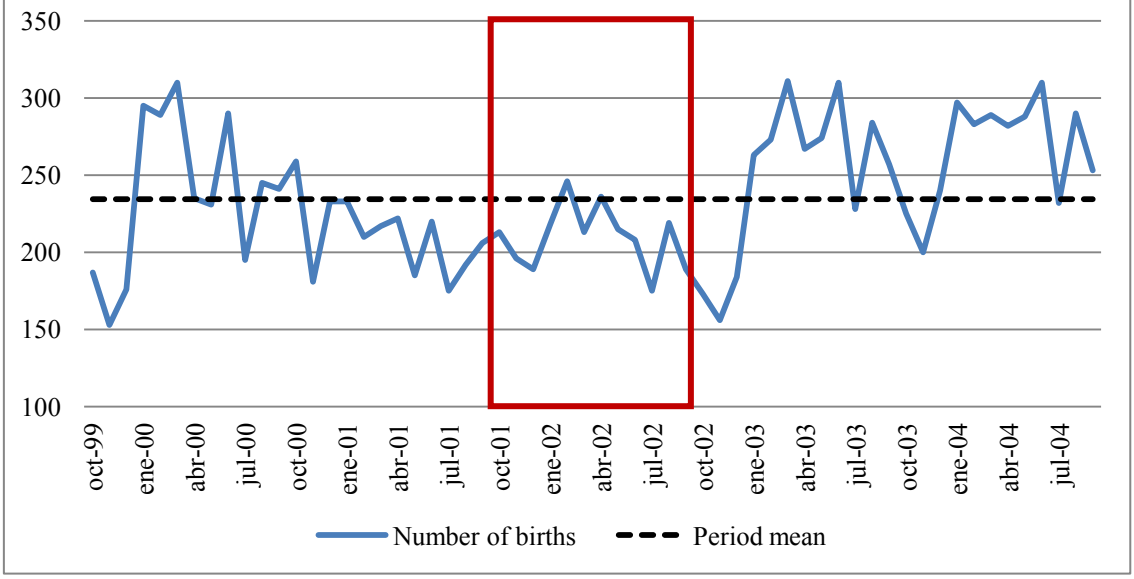
For that reason, after estimating each model, an interpretation of the interactions is presented in terms of marginal effects. Based on multiplicative effects, what will be shown is whether the effect of having been exposed to the food shortage on the probability of a newborn being a male and on neonatal mortality differs between subgroups. For instance, when the variable representing food scarcity is interacted with the type of districts where the child was born, the multiplicative effects will show if the effect of exposure to food shortage differs between highly and moderately affected districts. Given that all the variables participating in the interaction terms are categorical, the marginal effects of being exposed to intrauterine malnutrition will be calculated by subtracting the probability of a born child being a male (or of neonatal death) for non-exposed cohorts to the same probability for exposed cohorts for each subpopulation.

VII. Results

VII. A. "Lost" births

The cohorts that were *in utero* affected by malnutrition at least three out of the last six months of gestation (i.e. cohorts of children born between October 2001 and September 2002) have a lower number of live births than the cohorts born within the two years preceding or following the food shortage. Figure 2 shows how the number of births within the window of affected children is almost always laying below the average of births for the whole period under consideration. In fact, for every 100 children who were born in the control years only 87 children are born in the affected cohorts. Thus, during the period of food shortage a decline of 13% relative to the average number of birth in years of non severe food scarcity occurred. When considering the highly affected and moderately affected areas separately, the patterns look really similar as when considering the country as a whole, being slightly more pronounced in highly affected districts (Appendix B).

Figure 2: Fertility evolution from October 1999 to September 2004



As argued above, the decline in fertility could be due to two different phenomena. On the one hand, it could have been the case that because of the expectation of a bad harvest, families decided to postpone fertility through intentional birth control. On the other hand, it could also be that due to the exogenous nutrition shock, weaker fetuses died during the gestation stage, leading to a lower number of live births. According to the literature, higher socioeconomic strata are more effective in controlling fertility than its poorer counterparts. However, they are also less likely to be forced to respond demographically to short terms economic stress. On the contrary, although poorer individuals can have worse access to contraception methods, they may be the ones more interested in controlling fertility in front of a negative shock. Therefore, if fertility control is the force driving the fertility decline, it is not clear in which socioeconomic strata the reduction in the number of births should be observed. Conversely, premature death of fetuses is unequivocally more persistent in weaker and unhealthier individuals.

In the descriptive statistics it was shown that the only observable variable in which the cohorts affected by *in utero* malnutrition differed from the non-affected cohorts was that they were significantly less likely to be from the poorest households. This could indicate that higher social groups are not forced to respond demographically to the rise in prices through reduction in fertility. And that in fact, the poorest individuals are the ones experiencing the drop in fertility during the period of food scarcity. As already argued, this contraction in the number of births could be due either to the fact that

weaker fetuses from the poorest families did not succeed in surviving the whole gestation period, implying that the drop in fertility would have been driven by excess fetal mortality during the food crisis, or due to intentional fertility control.

Luckily, looking at the outcomes of children born alive from the food shortage-affected cohorts can provide extra information on which were the causes behind the decline in fertility during the food crisis. If selection of the strongest individuals occurred in the gestation stage, one would expect the sex ratio to be unbalanced in favor of girls. Additionally, one would also expect the neonatal mortality to be lower for the affected cohorts as the weakest proportion of infants that would have died within its first month of live already died *in utero*. In contrast, these effects should not be observed if the decline in fertility was due to intentional birth control.

VII. B. Probability of a male birth

On average, the sex ratio at birth for humans is 105 males per 100 females, (Sieff et al, 1990). However, this ratio is lower in Malawi, with only 102 males born per 100 females (CIA Factbook, 2013). Within my sample of children born between October 1999 and September 2004, the sex ratio is almost even, with only 100.8 males born per 100 females.

Table 4 presents the estimations of the logit model for the probability that a child born is a male, using odds ratios for an easier interpretation. The first column shows the results of the basic model, while the second column includes an interaction term between being from an affected cohort and being born in a district highly hit by the food shortage.

According to the results, the sex ratio at birth does not significantly differ between affected and unaffected cohorts, with the odds of the newborn child being a male being very similar between both types of cohorts. In contrast, being from the affected cohort in a district that was severely hit by the famine reduces the odds of the child being male, with only 94 males born per 100 females. Nevertheless, this difference is not statistically significant.

Table 4. Logit estimations for the probability of a male birth: odds ratios

	(1)	(2)
Year of birth trend	0.996	0.996
	(0.0148)	(0.0148)
Rural	0.899	0.900
	(0.0958)	(0.0958)
Male head	0.943	0.943
	(0.0445)	(0.0444)
Middle income	0.995	0.995
	(0.0503)	(0.0503)
Richer	0.981	0.981
	(0.0486)	(0.0486)
Mother education	0.989	0.989
	(0.0100)	(0.0100)
Mother literacy	**1.116***	**1.116***
	(0.0674)	(0.0674)
Age at first marriage	**1.018****	**1.018****
	(0.00842)	(0.00842)
Age of mother at birth	0.997	0.997
	(0.0249)	(0.0249)
Age of mother at birth^2	1.000	1.000
	(0.000435)	(0.000435)
Multiple birth	0.837	0.837
	(0.0965)	(0.0964)
Number of dead siblings	0.973	0.973
	(0.0323)	(0.0323)
Birth order	1.011	1.011
	(0.0206)	(0.0206)
In utero affected	0.978	1.012
	(0.0559)	(0.0976)
Highly affected region	0.940	0.949
	(0.0396)	(0.0447)
In utero affected x Highly affected region		0.943
		(0.112)
N	**14,472**	**14,472**

"*In utero* affected" = children born between October 2001 and September 2002
"Highly affected region"= season maize mean price over 18MK
Standard errors corrected by cluster effects in parenthesis
*** significant at 1%, ** significant at 5%, * significant at 10%

In order to better understand the effect of intrauterine malnutrition on the probability of a child being a male the marginal effects of being from a food shortage-affected cohort are calculated (Table 5). When considering the country as a whole, the probability of being a male is 0.5 percentage points lower for the children from affected cohorts, with the probability of being a male being 50.3% for unaffected cohorts and 49,8% for affected cohorts, this difference not being statistically significant. If the effect of the food shortage is disaggregated by type of district, it shows up that the decline in the sex

ratio for the malnourished cohorts comes from the highly affected areas, where food scarcity reduced by 1.2 percentage points the probabilities of the infant being a male. However, as it was pointed out before, one cannot be sure whether this bigger impact is due to the severity of the food shortage or conversely due to the underlying vulnerability of highly affected areas.

Table 5. Marginal effects of intrauterine malnutrition on the odds of a child being a male

	Affected cohorts	Unaffected cohorts	Marginal effect	P-value
Whole country [a]	0.498	0.503	-0.005	0.706
Highly affected districts [b]	0.485	0.497	-0.012	0.488
Moderately affected districts [b]	0.516	0.512	0.004	0.883

[a] Computed from the logit model (1)
[b] Computed from the logit model (2)

To sum up, cohorts that suffered intrauterine malnutrition present lower sex ratios than its non-affected counterparts, especially in those districts that were hit the hardest by the food shortage. But the marginal effect of being from affected cohorts is not large enough in order to be statistically significant[7]. These finding suggest that fetal mortality may be driving at least part of the fertility reduction observed in the previous subsection.

VII. C. Neonatal mortality

The results from the two previous sections show that a fertility decline occurred during the time of the acute food shortage and suggest that the drop in the number of births may due to the weaker fetuses not surviving until birth. On the one hand, the drop in fertility coincides with children being less likely to belong to the poorest social strata. On the other, children that were born during the period of food shortage in highly affected areas are less likely to be males, even if the difference is not statistically significant. Given these results, I expect neonatal mortality to be lower for the *in utero* affected infants. Moreover, I expect the effect to concentrate among males in highly affected areas.

[7] Separate models for highly and moderately affected areas cohorts were run to see if the results changed. However, the obtained impacts perfectly match the ones exposed here.

Table 6. Logit estimations for neonatal mortality: odds ratios

	(3)	(4)	(5)	(6)
Year of birth trend	0.969	0.969	0.969	0.969
	(0.0428)	(0.0429)	(0.0428)	(0.0428)
Rural	1.296	1.301	1.302	1.308
	(0.475)	(0.478)	(0.479)	(0.484)
Male head	0.832	0.830	0.833	0.831
	(0.122)	(0.122)	(0.123)	(0.123)
Middle income	0.840	0.840	0.841	0.839
	(0.168)	(0.168)	(0.169)	(0.169)
Richer	0.692	0.691	0.691	0.690
	(0.167)	(0.167)	(0.167)	(0.167)
Mother education	1.051	1.051	1.052	1.052
	(0.0401)	(0.0401)	(0.0401)	(0.0403)
Mother literacy	**0.661***	**0.662***	**0.659***	**0.660***
	(0.157)	(0.157)	(0.156)	(0.157)
Age at first marriage	**1.079*****	**1.078*****	**1.079*****	**1.078*****
	(0.0262)	(0.0262)	(0.0261)	(0.0262)
Age of mother at birth	**0.609*****	**0.609*****	**0.609*****	**0.610*****
	(0.0497)	(0.0497)	(0.0496)	(0.0499)
Age of mother at birth^2	**1.007*****	**1.007*****	**1.007*****	**1.007*****
	(0.00143)	(0.00143)	(0.00143)	(0.00144)
Multiple birth	**8.235*****	**8.215*****	**8.252*****	**8.206*****
	(1.855)	(1.858)	(1.855)	(1.862)
Number of dead siblings	1.195	1.194	1.193	1.192
	(0.135)	(0.135)	(0.136)	(0.135)
Birth order	**1.176****	**1.176****	**1.178****	**1.179****
	(0.0837)	(0.0838)	(0.0841)	(0.0840)
In utero affected	**0.682***	0.782	0.793	0.912
	(0.136)	(0.240)	(0.207)	(0.375)
Highly affected region	0.914	0.943	0.912	1.120
	(0.142)	(0.164)	(0.142)	(0.294)
Male child	**1.430*****	**1.428*****	**1.482*****	**1.771****
	(0.189)	(0.188)	(0.213)	(0.477)
In utero affected x Highly affected region		0.785		0.801
		(0.317)		(0.428)
In utero affected x Male child			0.762	0.776
			(0.261)	(0.420)
Highly affected region x Male child				0.749
				(0.234)
In utero affected x Highly affected region x Male child				0.902
				(0.623)
N	**14,472**	**14,472**	**14,472**	**14,472**

"*In utero* affected" = children born between October 2001 and September 2002
"Highly affected region"= season maize mean price over 18MK
Standard errors corrected by cluster effects in parenthesis
*** significant at 1%, ** significant at 5%, * significant at 10%

Table 6 presents the results of the estimated logit models. As expected given the previous results, which suggest that some positive selection may have taken place in the gestation stage, the cohorts affected by malnutrition while *in utero* are less likely to die within its first 28 days of life, even if this effect is only statistically significant at 10% level for model (3) which does not include any interaction term to allow for heterogeneous effects among districts or gender. Focusing on the models that include interactions, it is shown that being from an affected cohort plus being born in a highly affected area or being a male further reduces the probability of neonatal death. However, interactions are not easy to interpret in non-linear models, and therefore the marginal effects of being malnourished *in utero* are computed for a better interpretation.

Table 7 shows the marginal effects calculated from the estimations above. Panel A presents the marginal effects of being *in utero* affected by the nutrition shock considering both sexes together. As it was already suggested by the estimated coefficient, *in utero* affected cohorts are less likely to suffer from neonatal death when compared to the other cohorts. More concretely, neonatal mortality is 9 deaths per 1000 children born alive lower for the affected cohorts, this difference being statistically significant at a 5% confidence level. Moreover, it is reaffirming that this effect is basically driven by a reduction in neonatal deaths in the highly affected areas. In these areas, *in utero* malnourished cohorts present 11,6 deaths per 1000 children born alive less than the comparison cohorts, with the difference being again statistically significant at a 5% confidence level. Neonatal mortality also drops in moderately affected areas, the difference not being significant in this case.

Panel B and Panel C also look at the marginal effect of being exposed to intrauterine malnutrition on the odds of dying in the first month of live, but only considers male infants in the former panel and female infants in the latter. As argued above, if selection is driving the decline in fertility and male are more vulnerable *in utero*, one would expect the effects to concentrate in male infants. And in fact that is what is found when dividing the effects according to gender. Considering the whole country, males affected by food shortages are significantly less likely to die in the neonatal period, with 12.7 less male children per 1000 male born alive dying in the affected cohorts with respect to their unaffected counterparts, the difference being statistically significant at a 5% level. Moreover, the effect is concentrated in severely affected areas, where as much as 16.5

male neonates per 1000 male born alive die, this time the difference being significant at a 1% level. Males in moderately affected areas also die less, but the difference is not statistically significant in this case. On the contrary, although girls are also less likely to die within the first 28 days of life, the differences are not statistically significant, neither looking at the country as a whole nor at the highly and lowly affected separately.

Table 7. Marginal effect of *in utero* malnutrition on neonatal mortality

	Affected cohort	Unaffected cohort	Marginal effect	p-value	
Panel A - Full sample					
Whole country [a]	0.0181	0.0271	**-0.009**	**0.024**	**
Highly affected districts [b]	0.0164	0.0280	**-0.0116**	**0.016**	**
Moderately affected districts [b]	0.0206	0.0256	-0.005	0.451	
Panel B - Subsample of males					
Whole country [c]	0.0194	0.0321	**-0.0127**	**0.024**	**
Highly affected districts [d]	0.0152	0.0317	**-0.0165**	**0.007**	***
Moderately affected districts [d]	0.0251	0.0326	-0.0075	0.477	
Panel C - Subsample of females					
Whole country [c]	0.0167	0.0220	-0.0053	0.260	
Highly affected districts [d]	0.0174	0.0244	-0.007	0.271	
Moderately affected districts [d]	0.0158	0.0185	-0.0027	0.683	

[a] Computed from the logit model (3)
[b] Computed from the logit model (4)
[c] Computed from the logit model (5)
[d] Computed from the logit model (6)
*** significant at 1%, ** significant at 5%, * significant at 10%

All together, the presented results suggest that the part of the impact of the nutrition shock on affected individuals is throughout an increase in sex-selective fetal mortality. Therefore, the resulting cohorts are likely to consist on a selection of stronger individuals. This idea is supported by the three assessed measures: a decline in overall fertility, cohorts unbalanced in favor of girls and neonatal mortality being significantly lower among the *in utero* affected individuals, especially in highly affected districts and among males. However, it is worth keeping in mind that fertility control should be also driving part of the results obtained.

VIII. Sensitivity analysis and Robustness checks

As it was argued when defining the control and treatment groups, the chosen classification presents some advantages like not ignoring any observation of children born in the considered time span, making sure that both groups are as similar as possible or only considering as treated those individuals that were exposed during the most critical period of the gestation. However, the definition of the groups also presents some disadvantages. In this section I test for different specifications of the treatment and control groups in order to see whether the results presented above are sensitive to changes in the definition of the groups, or if in contrast they are robust to significant changes in the definition of treatment status.

First of all, the chosen control group (children born from October 1999 to September 2001 and from October 2002 to September 2004) contains children that, although not having been exposed to food shortage during at least three out of the last six month of gestation, were exposed during other stages of the gestation or during their first month of life. This exposure could be somehow contaminating the outcomes of the control group both in terms of the probability of an offspring being a male as well as in terms of the probability of dying within the first month of life. For instance, the literature has stated that the critical months for spontaneous abortion are the first three month of gestation (Wood, 1994), and therefore children exposed during this period could be also less likely to survive the whole gestation. Additionally, although neonatal mortality is thought to be less influenced by environmental factors than infant or child mortality, being exposed to the period of food shortage during the first month of life could affect the surviving probabilities of a neonate.

For that reason, here the control group is redefined excluding from it any child who was exposed to the food crisis during at least one month, either *in utero* or during its first month of life. Consequently, infants born between July and September 2001 are excluded from the control group as they were exposed to food shortages during their first month of life or during the last month or two months of gestation, depending of the cohort. On top of that, children born between October 2002 and March 2003 were also exposed to food shortages during part of their gestation, ranging from five months to one month of exposure time. These individuals are also excluded from the control group. The restriction of the control group allows for a reduction in the potential

contamination. But unluckily, this diminution is carried out at the expense of a reduction in the sample size.

Tables 8 and 9 present the marginal effects of being exposed to food shortage on the probability of a born child being a male and on neonatal mortality respectively, considering the redefined control group. If it is the case that there are significant effects beyond those of being exposed during one out of the last two trimesters of gestation, the impacts should be more pronounced once the partly exposed children are excluded from the control group.

Table 8. Marginal effect of in utero malnutrition on the probability of a male birth

	Affected cohorts	Unaffected cohorts	Marginal effect	P-value
Whole country	0.498	0.505	-0.007	0.613
Highly affected districts	0.485	0.497	-0.012	0.489
Moderately affected districts	0.516	0.517	-0.001	0.960

Table 9. Marginal effect of in utero malnutrition on noenatal mortality

Panel A - Full sample

	Affected cohorts	Unaffected cohorts	Marginal effect	P-value	
Whole country	0.018	0.027	**-0.009**	**0.032**	**
Highly affected districts	0.016	0.028	**-0.012**	**0.021**	**
Moderately affected districts	0.021	0.025	-0.004	0.515	

Panel B - Subsample of males

	Affected cohorts	Unaffected cohorts	Marginal effect	P-value	
Whole country	0.019	0.031	**-0.012**	**0.043**	**
Highly affected districts	0.015	0.031	**-0.016**	**0.013**	**
Moderately affected districts	0.025	0.031	-0.006	0.594	

Panel C - Subsample of females

	Affected cohorts	Unaffected cohorts	Marginal effect	P-value
Whole country	0.017	0.022	-0.005	0.235
Highly affected districts	0.017	0.025	-0.008	0.249
Moderately affected districts	0.016	0.019	-0.003	0.653

*** significant at 1%, ** significant at 5%, * significant at 10%

As shown in table 8, the results on the probability of a newborn being a male do not change at all with the modification of the control group. Again, children are less likely to be males when belonging to the affected cohorts, especially in most affected areas, but the difference is far from significant. The same stands for neonatal mortality. After restricting the control group, the marginal effects of being *in utero* exposed to food shortage remain exactly the same in magnitude for all the samples under analysis. The only difference is that the level of significance is slightly reduced with the new specification, probably due to the reduction in the sample size that makes the standard errors to increase. Thus, the results suggest that in the main results the control group is not significantly contaminated by the fact that some individuals in it are partly exposed to food scarcity. At least, it is not sufficiently contaminated in order to affect the results. Therefore, not restricting the control group appears as a better option as it allows for a bigger sample size without significantly affecting the outcomes.

A second problem with the initially defined groups is that the treatment group contains children that widely vary in terms of time of exposure. While children born within April 2002 and June 2002 were exposed during the whole gestation, individuals born in September and October 2001 were only exposed during three or four months during the initial stage of the famine. Intuitively, one would expect the children to be more affected the more time they have been exposed to the food shortage. Therefore heterogeneous effects could exist among children with different exposure time. If the effects observed in the main results are actually due to *in utero* malnutrition, one would expect the effects to be larger the more time the individual was exposed to the food crisis. Here, the treatment window is significantly modified to identify whether the intensity of the impacts depends on the time of exposure to malnutrition.

To start with, the same logit models for the probability of a child being a male and for neonatal mortality are estimated considering that any child who was exposed to food shortages for at least one month belongs to the treatment group (children born between July 2001 and March 2003). As a consequence, the control group consists of those children that were never exposed to the famine neither *in utero* nor during their first month of life (children born from October 1999 to June 2001 and from April 2002 to September 2004). If malnutrition is the cause of the decreased neonatal mortality for the affected cohorts, one would expect the effect to diminish in magnitude as the mean time

of exposure falls for the treatment group. Table 10 and 11 present the results under the expanded treatment window.

Table 10. Odds ratio of being a male

	Affected cohort	Unaffected cohort	Marginal effect	P-value
Whole country	0.494	0.505	-0.011	0.490
Highly affected districts	0.489	0.497	-0.008	0.600
Moderately affected districts	0.503	0.517	-0.014	0.524

As shown in table 10, the expansion of the treatment does not affect the results on the probability of a male birth, with this probability remaining lower for the cohorts exposed to food scarcity relatively to the non-exposed cohorts. Conversely, the results on neonatal mortality are significantly affected (Table 11).

Table 11. Marginal effect of *in utero* malnutrition on neonatal mortality

Panel A - Full simple				
	Affected cohort	Unaffected cohort	Marginal effect	P-value
Whole country	0.022	0.027	-0.005	0.201
Highly affected districts	0.020	0.028	-0.008	0.106
Moderately affected districts	0.024	0.025	-0.001	0.876
Panel B - Subsample of males				
	Affected cohort	Unaffected cohort	Marginal effect	P-value
Whole country	0.026	0.031	-0.005	0.423
Highly affected districts	0.022	0.032	-0.010	0.156
Moderately affected districts	0.032	0.031	0.001	0.885
Panel C - Subsample of females				
	Affected cohort	Unaffected cohort	Marginal effect	P-value
Whole country	0.018	0.023	-0.005	0.262
Highly affected districts	0.019	0.025	-0.006	0.301
Moderately affected districts	0.016	0.019	-0.003	0.614

*** significant at 1%, ** significant at 5%, * significant at 10%

When expanding the definition of affected by malnutrition to every child who has been exposed for at least one month, the impact of exposure to food shortage diminishes in magnitude. Even more relevant, the marginal effect of *in utero* exposure to malnutrition becomes statistically insignificant. These results suggest that in fact the intensity of exposure affects the magnitude of the impact of *in utero* malnutrition, providing support

to the argument that the marginal effects of exposure to food shortage presented in Section VII are in fact driven by intrauterine undernutrition

Interestingly, excluding the partly affected children from the control group does not make any difference in the results. However, including them in the treatment group dissolves the marginal effect of being exposed to malnutrition. Together, these two results suggest that, as argued in the literature, the impacts are concentrated among these children who were undernourished during the last two trimesters of gestation.

To corroborate what the above results insinuate, the same models are re-run considering as treated only those children who were exposed to food shortages during the whole two last trimesters of gestation. If it is true that the intensity of exposure to the food shortage affects the magnitude of the impacts, one would expect the effects to be greater once the treatment window is shrunk, and consequently the mean time of exposure of the treatment group increased. This would provide extra evidence supporting the argument that the mechanism driving the drop in neonatal mortality for exposed cohorts is in fact their condition of *in utero* malnourished. In order to make the results of this model comparable to the results of the specification presented in the Section VII, the control group consists of the cohorts born right before and right after the birth of those exposed for the full six last months. Thus, the control contains children partly exposed, as it was also the case in the control group for the specifications in Section VII[8]. Table 12 and Table 13 show the marginal effects of exposure to malnutrition after the redefinition of the treatment and control groups.

Table 12. Odds ratio of being a male

	Affected cohort	Unaffected cohort	Marginal effect	P-value
Whole country	0.504	0.502	0.002	0.896
Highly affected districts	0.491	0.495	-0.004	0.872
Moderately affected districts	0.523	0.512	0.011	0.706

Again, the marginal effect of exposure to *in utero* undernutrition on the probability of a child born being a male is unaffected, remaining insignificant for all the samples analyzed. On the contrary, the results on neonatal mortality change substantially in

[8] Restricting the control group to exclude those individuals party exposed to the food shortage does not affect the results, nor in magnitude neither in significance.

terms of magnitude and significance, in the expected direction. When considering males and females together and the country as a whole, the marginal effect of being exposed to the famine rises by 3 deaths per mil children born alive (from 9 death per mil children in the initial specification to 12 death per mil children once the treatment window is redefined). The change is more moderate in highly affected districts, with the marginal effect increasing only by 1 death per mil infants. Moreover, the statistical significance of both effects improves. Interestingly the impact on moderately affected areas also increases notably, although remaining statistically insignificant.

Table 13. Marginal effect of *in utero* malnutrition on neonatal mortality

Panel A - Full simple					
	Affected cohort	Unaffected cohort	Marginal effect	P-value	
Whole country	0.015	0.027	**-0.012**	**0.010**	***
Highly affected districts	0.014	0.027	**-0.013**	**0.018**	**
Moderately affected districts	0.017	0.026	-0.009	0.237	
Panel B - Subsample of males					
	Affected cohort	Unaffected cohort	Marginal effect	P-value	
Whole country	0.012	0.032	**-0.020**	**0.000**	***
Highly affected districts	0.010	0.031	**-0.021**	**0.002**	***
Moderately affected districts	0.015	0.033	**-0.018**	**0.042**	**
Panel C - Subsample of females					
	Affected cohort	Unaffected cohort	Marginal effect	P-value	
Whole country	0.018	0.021	-0.003	0.651	
Highly affected districts	0.018	0.024	-0.006	0.480	
Moderately affected districts	0.019	0.018	0.001	0.877	

If males and females are considered separately, the results with this specification confirm what was found with the initial group definition: the effect observed over the whole population is driven by significant drops in the neonatal mortality of *in utero* affected males. The marginal effect over females stays statistically insignificant, while the marginal effect of exposure over the male children appears again negative, this time higher in magnitude and with its statistical significance rising dramatically. A final change derived from the modification of the treatment and control groups is that with the new specification the marginal effect of having been exposed to malnutrition while

in utero becomes significant for males also in moderately affected regions. These results confirm that increasing the mean time of exposure to the food shortages for the treatment group leads to a more intense drop on neonatal mortality. Moreover, they show that although not being that severe, the Northern region and the border districts which belong to the group of moderately affected areas were also affected by the period of food scarcity.

Finally, other classifications of the districts between highly affected and moderately affected areas were also considered to look at whether the results in Section VII are robust to the modification of the groups. The obtained results were consistent with the results in Section VII for the three alternative classifications considered[9].

IX. Caveats and Limitations

The presented results suffer from some limitations that, even if tried to reduce at maximum, could not be eliminated. Starting with the characteristics of the database, given that in order to make the analysis it is necessary to know where each child was living during the gestation stage as well as its place of birth, only the subsample of children whose mothers did not migrate after their birth can be used for the analysis. As it was shown in the descriptive statistics section, children whose mothers migrated are significantly different from those whose mothers did not migrate. In fact, they represent a wealthier and more educated subsample. For that reason, the obtained results cannot be generalized to the whole population, but are only valid for the subsample of non-migrants. As migrants are richer, and according to the results obtained the richest seem to be the least affected ones, the effects for the migrants are likely to be milder.

Another shortcoming of this research concerning generalization of the results is inherent to the estimation strategy used. In order to be able to quantify the effect of *in utero* exposure to food shortage, the cohorts affected by the 2001/2002 period of acute food scarcity were compared to those cohorts born just before and just after the crisis. This strategy requires the use of an exceptional shock in order to be able to clearly identify a treatment and a control group. Therefore, it may be difficult to generalize the results to other contexts in which these kind of severe exogenous events are absent. This shortcoming has also a policy implication. Almond and Currie (2011) suggest that most

[9] Tables are not presented, but can be obtained from the author under request

likely policy measures aimed to prevent famines or severe food shortages would not pass a cost-benefit analysis. Consequently, policy makers may be more interested in looking at the impact of less severe malnutrition shocks, which are possible to prevent in an inexpensive and effective way, like providing extra nutrients to pregnant women. However, the estimates provided in this research may be useful as an upper bound of the impact of malnutrition on reproductive health of women and early health of infants.

Additionally, it was shown that the most highly affected districts were also the poorest and least developed ones. Thus, it is difficult to know whether the strongest effect that was found in severely affected areas is due to the intensity of the food shortage or contrary to the underlying vulnerability of those areas. In order to be able to isolate the impact of the nutrition shock, many socioeconomic variables were included in the models, all of them showing coefficients with the expected sign when statistically significant. However, to the extent that prevalent vulnerability is not absolutely controlled for, the impacts imputed to the food shortage could be partially due to the underlying characteristics of the affected population.

Finally, even if the results suggest that some selection took place before birth leading to the children from cohorts affected by intrauterine malnutrition being relatively stronger (i.e. suffering from lower neonatal mortality) than the non-affected children, it is difficult to assure whether the selection is through deliberate fertility control from the feeblest individuals or through increases in fetal mortality affecting the weakest fetuses. Looking at the time lag between the nutrition shock and the apparition of the response could help differentiating both effects. However, this exercise is left for further research.

X. Conclusions and Contribution

By looking at the Malawian food shortage of 2002, this study aimed to assess the impact of *in utero* malnutrition on the early health of affected children, through its survival probabilities. With that purpose, fertility trends, the probability of a born child being a male and the probability of neonatal death were studied, comparing cohorts *in utero* affected by malnutrition to non-affected cohorts.

The results insinuate that food scarcity may have driven a positive selection process among the cohorts of children which were *in utero* during the food shortage. To start with, it was found that for every 100 children born in control years only 87 children were born in affected cohorts. Thus, during the food shortage a decline of 13% relative to the average number of birth in control years occurred. Moreover, children from affected cohorts were less likely to be from the poorest socioeconomic strata, what could be explained either by arguing that individuals in the lower part of the wealth distribution voluntarily decided to control fertility when they expected a bad harvest after the flooding of February 2001, or by a process of *in utero* positive selection.

If the second mechanism is dominating, this should be reflected in the cohort of children exposed to the food shortage being unbalanced in favor of females, as male fetuses are thought to be more vulnerable than female fetuses during gestation, as well as more prone to spontaneous abortion (Fukuda et al, 1998: Lyster, 1974; Zorn et al, 2002). In contrast, these effects should not be observed if the decline in fertility was due to intentional birth control. The obtained results show that in cohorts affected by food shortage the probability of a newborn being a male is reduced, although the reduction was not found statistically significant. This could indicate that although natural selection may have played a role, deliberated fertility control is also likely to exist.

Regarding neonatal mortality, in a context of food shortage one would expect the health of the population to deteriorate and thus the premature mortality of children exposed *in utero* to malnutrition to increase. Nevertheless, the previous results show that a fertility decline occurred during the time of the food shortage and suggest that the drop in the number of births may be partly due to the weaker fetuses not surviving until birth. Consequently, a drop in neonatal mortality for exposed children is expected under this scenario, as only the stronger individuals are expected to be born (Catalano and Bruckner, 2005). And that is what was actually found. Children from affected cohorts were shown to be significantly less likely to die within their first month of live when compared to their counterparts in unaffected cohorts. Thus, the hypothesis of positive selection is further supported by the results on the probability of early death, as lower neonatal mortality for exposed children may be an indicator of the fact that the weakest proportion of infants which would have died within their first month of life already died *in utero*.

In order to strengthen the argument of intrauterine malnutrition being the cause behind the differences observed in neonatal mortality, heterogeneous impacts by gender and region of birth were also analyzed. If malnutrition is the driving force behind natural selection and the consequent reduction in neonatal mortality, one would expect children born in the most severely affected regions to show stronger impacts. Moreover, the effects should be concentrated in male infants, as the literature states that male fetuses are more vulnerable than female fetuses. As expected, males born in highly affected regions are the ones showing the greatest impact, with neonatal mortality being 16.5 deaths per mil male children born alive lower for exposed cohorts when compared to non-exposed children, this difference being statistically significant at a 5% confidence level. Conversely, the decline in neonatal mortality for males in moderately affected areas and females in both types of districts are not statistically significant.

Another way of corroborating that it is in fact the lack of food while *in utero* what drives neonatal mortality declines is by demonstrating that an increase in the time of exposure to the food shortage accentuates the impacts. The sensitivity analysis showed that there is a significant positive correlation between the mean time of exposure to the food shortage of the treatment group and the intensity of the effects. Therefore, intrauterine malnutrition is further enhanced as the cause of diverging probabilities of neonatal death.

Finally, it is worth mentioning that the results are robust to significant changes in the definition of the control group as well as to modifications in the classification between severely and moderately affected areas of critical districts.

All together, the discussed results suggest that the mechanism through which the nutritional shock affected individuals was through an increase in fetal mortality. Therefore, the surviving cohorts consist on a selection of stronger individuals. This idea is supported by the three assessed measures: a decline in overall fertility, cohorts unbalanced in favor of girls and neonatal mortality being significantly lower among the individuals affected *in utero*, especially in highly affected districts and among males.

These findings have several implications. Regarding the theoretical contributions, probably the most important one is that it provides additional evidence of an existing causal link between the nutritional level experienced by pregnant women, and

consequently by its offspring while *in utero,* and both the reproductive health of these women and the early health of the children.

In turn, proving the existence of this relation has important policy implications. First of all, it reaffirms the gestation stage as critical for the health of both mothers and offspring, suggesting that most probably interventions that focus on the pregnancy period may be highly effective (Almond & Currie, 2011). Secondly, given that many nutritional interventions do not have very high costs it places nutritional maternal care as a very cost-effective way of improving the health of children (UNICEF, 2009). Finally, by improving the health of children as early as *in utero*, an enhanced intrauterine nutrition is likely to have a critical impact on the life paths of these individuals, making them healthier and more productive (UNDP, 2000). Having more capable inhabitants indubitably would have a positive macroeconomic impact on countries, like Malawi, which are still threatened by food insecurity.

Coming back to the contribution of this study for the academia, it provides further evidence for theories arguing for the vital disadvantages of males as fetuses and as infants, as well as those proposing the sex ratio at birth as an indicator of intrauterine unfavorable conditions (Catalano & Bruckner, 2005; Naeye et al, 1971; Waldron, 1983). Additionally, it has an indirect implication for studies looking at the long term effects of intrauterine negative shocks. By showing that positive selection may be taking place as early as *in utero,* it implies that studies focusing on adults, as well as on children, may be underestimating the effects of intrauterine negative shocks. The underestimation would be due to the fact that the population they are analyzing could be a positively selected sample of what this population would have been in absence of the adverse shock.

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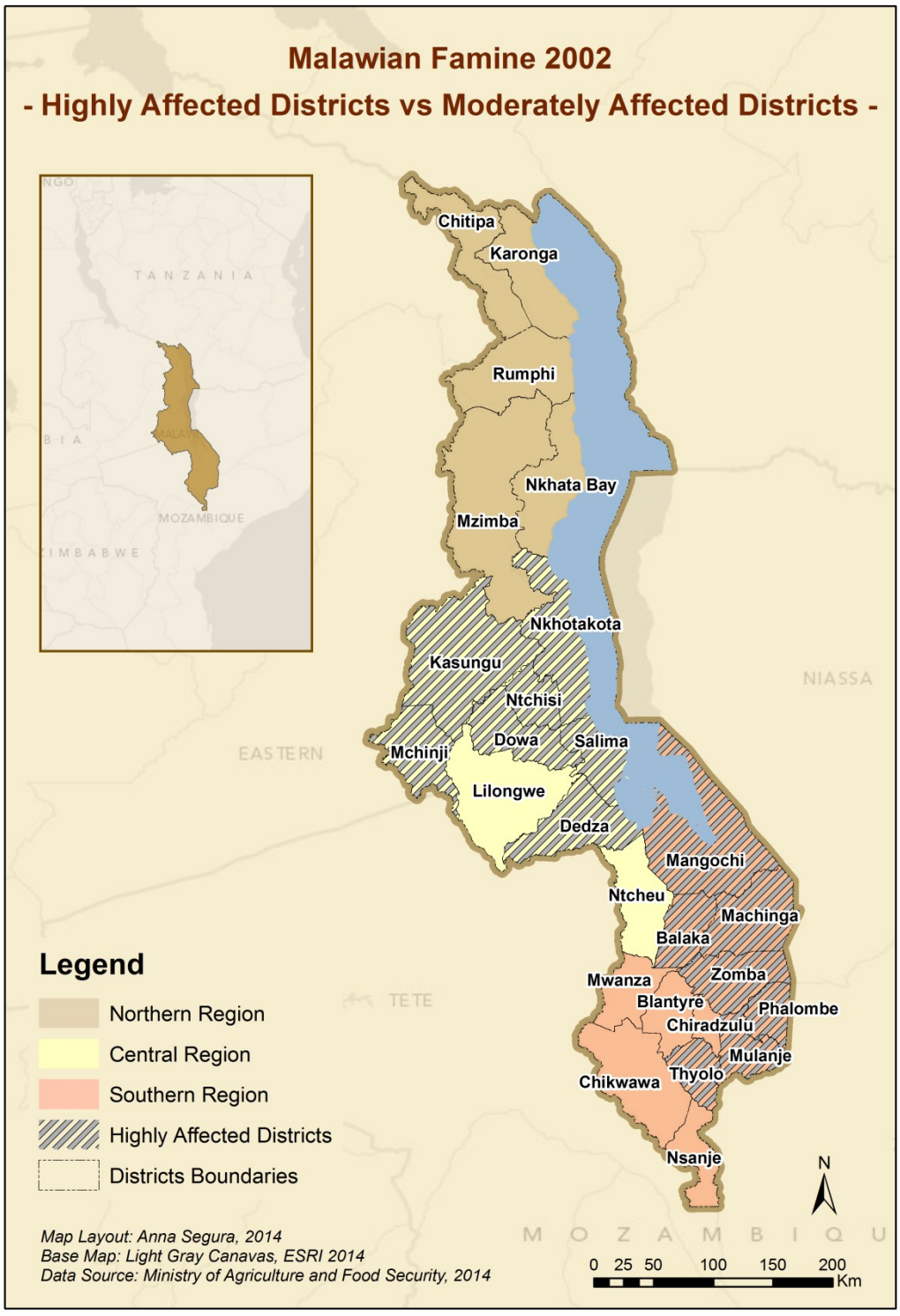
Appendixes

Appendix A – Map of Malawi showing highly affected districts vs moderately affected districts



Appendix B – Fertility trends considering highly affected and moderately affected areas separately

Figure x: Fertility evolution from October 1999 to September 2004
Highly affected districts

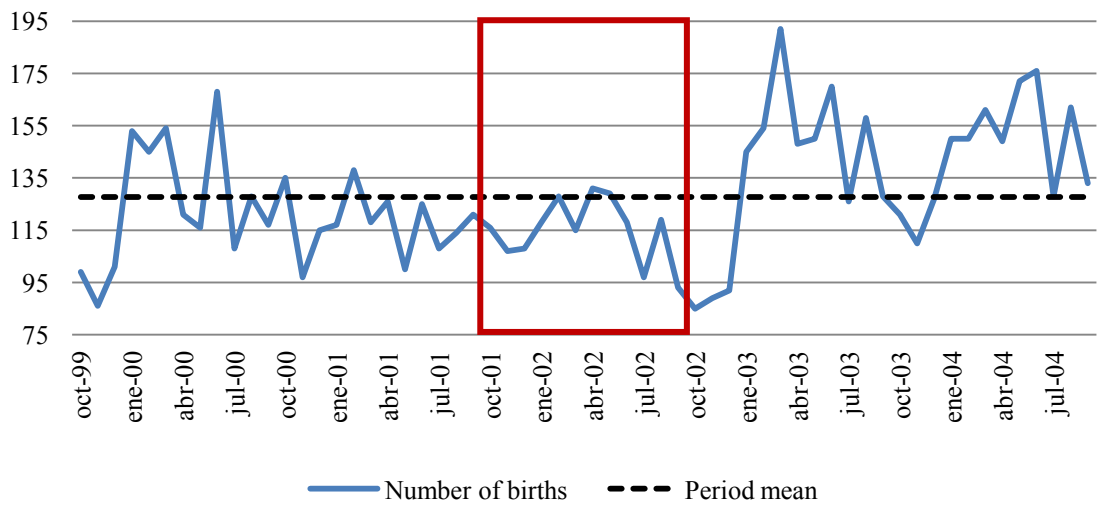


Figure 3: Fertility evolution from October 1999 to September 2004
Moderately affected districts