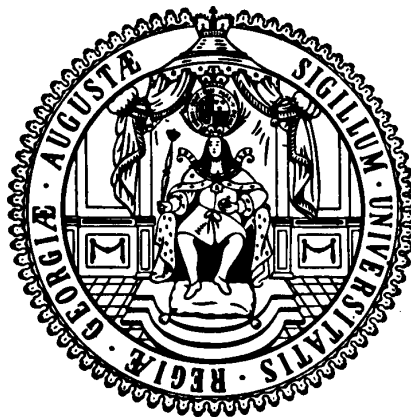# Courant Research Centre
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An Assessment of the Effects of the 2002 Food Crisis on Children's Health in Malawi

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An Assessment of the Effects of the 2002 Food Crisis on Children's Health in Malawi[1]

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Abstract

In 2002 Malawi experienced a serious shortage of cereals due to adverse climatic conditions. The World Food Programme assumed that about 2.1 to 3.2 million people were threatened of starvation at that time. However, not much research has been undertaken to investigate the actual consequences of this crisis. In particular, little is known about how the crisis affected the health status of children. Obviously, quantifying the health impact of such a crisis is a serious task given the lack of data and the more general problem of relating outcomes to specific shocks and policies. In this paper a difference-in-difference estimator is used to quantify the impact of the food crisis on the health status of children. The findings suggest that at least in the short run, there was neither a significant impact on child mortality nor on malnutrition. This would suggest that the shock might have been less severe than initially assumed and that the various policy interventions undertaken at the time have been effective or at least sufficient to counteract the immediate effects of the crisis.

Keywords: Child Mortality, Malnutrition, Food Crisis, Malawi.

1. Introduction

The 2002 food crisis in Malawi is often said to be the worst in the recent history of the country. However, the actual consequences of the crisis have so far not been investigated in a quantitative and rigorous way. Estimates of the related excess mortality, for example, range from 300 to 500 as lower bound[2] to estimates, in particular of civil society groups, of about 1,000 to 3,000 cases. Some NGOs even estimated up to 10,000 to 15,000 deaths on the basis of hospital records (Devereux, 2000b; Taifour, 2002). Although, the latter estimate is by many considered as largely overestimated, the WHO (2002) too assumed in some districts of the country a doubling of child mortality rates, what would roughly imply at least 9,000 additional deaths.[3]

While most previous studies have explored the causes of the food shortfall (see, for example, Devereux, 2002b; Stevens et al., 2002; and Kydd et al., 2002), only few researchers have investigated the effectiveness of policy interventions undertaken at the time (except Dorward and Kydd (2004)). Moreover, no efforts have been made to quantify exactly the health and mortality effects of the crisis.

The World Food Programme (WFP) estimated that during the crisis period 2.1 to 3.2 million Malawians were threatened by starvation. The large number of potentially undernourished people, in particular children, and the exposure to further nutrition-related diseases, is said to have adversely affected the health of children — often with fatal outcomes. But even if the effect was not fatal, it is well known that temporary undernutrition can seriously hamper the development of cognitive skills of children in the long run. Although the long-term consequences cannot be studied now, we will analyse some of the potential short-run effects of the food crisis, in particular the impact on child mortality and children's anthropometric measures.

Obviously, providing a rigorous quantitative analysis is impeded by the lack of longitudinal data that would allow tracking children's characteristics before, during and after the crisis. However, we think that cross-sectional survey data before and after the crisis and the fact that children in different regions were differently exposed to the shock, can be used to identify the effects of the crisis. We use a difference-in-difference strategy and suggest various ways to deal with potential selection problems and confounding factors.

On the basis of this analysis, we do not find any evidence that the 2002 food crisis caused an increase in child mortality and acute undernutrition. The estimates rather suggest that children from affected regions had on average 0.14 standard deviations (SD) higher weight-for-age and 0.15 SD higher height-for-age z-scores in the aftermath of the crisis, implying that their nutritional status had slightly improved. Even if the results for acute undernutrition have to be interpreted with caution given that two years had elapsed between the peak of the crisis and the post-impact data collection, the improvements in underweight and chronic malnutrition suggest that the policy interventions undertaken at that time have been effective in the sense that they improved the nutritional status of children living in affected areas.

This article contributes to a better understanding of the consequences of the 2002 food crisis in Malawi. It builds on existing qualitative studies and offers a rigorous quantitative analysis of the effects on child mortality and nutrition. More generally, the

study contributes to the debate on the potential health effects of a global food crisis similar in magnitude as seen in 2008 in a typical poor and largely agrarian African country.

The remainder of the paper is structured as follows: Section 2 briefly reviews the relevant literature. Section 3 discusses the context of the 2002 Malawian food crisis. Section 4 describes the empirical strategy and the data used for the analysis. Section 5 presents the results and tests their robustness. Section 6 concludes.

2. A Review of the Relevant Literature

Akresh and Verwimp (2006) examine the effects of crop failure and civil war on children's health in Rwanda. Similar to what we will do, they use variation over space and time in the intensity of these shocks to identify the effects. More precisely, they look at the significance and magnitude of province and birth-cohort interaction terms to measure impact. While the authors find no impact on the health status of boys, they find height-for-age z-scores of girls in affected regions to be 0.72 SDs lower. Hoddinott and Kinsey (2001) and Hoddinott (2006) investigate the impact of various droughts on child growth in Zimbabwe. While they find no effects on children older than two, their results suggest, a (probably permanent) loss of 1.5-2 cm of growth for children aged between 12 to 24 months. They also show that children from poorer households suffered disproportionately more from such crises, not only in terms of nutrition but also in terms of schooling. Again for Zimbabwe, Alderman et al. (2004) study the long-term consequences of early childhood malnutrition. They use an IV strategy and control for mother-fixed effects for identification and conclude that children lost 3.4 cm of height, 0.85 grades of schooling and half a year of school attendance by the time they reached adolescence. Negative effects on schooling are also confirmed by Grimm (2008). Similar to the approach chosen in this paper, he uses two repeated cross-sections from Burkina Faso to analyze the effect of higher food prices on real household income and children's schooling. An IV estimator is used to deal with the potential endogeneity of household income in the schooling equation. The findings suggest that food-prize inflation induced declines in real income had a substantial effect on parental investments in children's schooling. Grimm estimates that following the drought in 1997/98 more than 100,000 children under 12 were withdrawn from or not enrolled in school. A similar study on Côte d'Ivoire (Jensen, 2000) examines if children that live in regions that were exposed to adverse weather shocks knew lower investments in their education and lower well-being. In order to also assess the impact on health, Jensen analyses differences in the nutritional status measured by the child's weight-for-height z-score and the use of medical services. Based on household data collected between 1985 and 1988, Jensen concludes that investments in children, in particular nutrition, are significantly affected by adverse agricultural conditions. However, due to the short period under study, this research cannot answer the question whether these effects are likely to be permanent.

Finally, Yamano et al. (2005) also examine the effects of harvest failure on children's growth. For Ethiopia, they find similar effects than the cited studies on Zimbabwe. When looking at the effectiveness of policy interventions, they emphasize that food aid can in

principle compensate for such effects, but that inflexible targeting, endemic poverty and low maternal education often keep stunting at high levels despite such interventions. Ruel et al. (2008) are even more pessimistic on ex-post interventions. They show for Haiti that stunting, wasting and underweight were on average 4-6 percentage points lower in communities that participated in preventative child health and nutrition programs compared to communities that participated in recuperative programs.

However, there are also examples where climatic shocks had only moderate or even insignificant effects on children's outcomes. De Waal et al. (2006) for instance, looked at child survival during the 2002/2003 drought in Ethiopia, which was probably the worst in the modern history of the country with more than 13.2 million affected people. Although, the data from the Ethiopian Child Survival Survey of 2004 suggests that child mortality was higher in affected areas, in a more detailed analysis using multivariate regression, the authors come to the conclusion that the stated differences are rather attributable to the persistently adverse conditions in these areas than to the immediate impact of the 2002/2003 drought (de Waal et al., 2006). Finally, the study by Strauss et al. (2002) that investigates the impact of the 1997 economic crisis in Indonesia (which also led to increasing food prices) on child health outcomes, indicates that three years after the crisis, children were not substantially worse off in respect to their health or income poverty than they were before the crisis. Contrary to expectations, some seem to have been even better off.

3. The 2002 Food Crisis in Malawi

Due to climatic fluctuations countries in Sub-Saharan Africa frequently experience harvest failure, food shortages and food price inflation. Over the period 1970 to 2006, Malawi experienced about 40 weather-related disasters, 16 of which occurred after 1990 (Roshni, 2007). The most serious ones in the country's history were the *Nyasaland*[4] famine in 1949, the drought of 1991/1992 and the two major food crises in 2002 and 2005. The 2002 food crisis is judged by many as the worst in the country's recent history.[5] The adverse weather conditions are largely seen as the immediate trigger of the crisis. Strong rainfalls in February 2001 caused flooding in 13 out of the 27 Malawian districts (FEWSNET, 2001a), which led to a fall in the national maize production by 32 percent to 1.7 million metric tons in the 2000/2001 season (FEWSNET, 2001b). Looking at the production figures over a longer time period (figure 1), it can be seen that the 2000/2001 harvest fell below the five- and ten-year moving averages. In absolute figures, however, the production loss was less severe than the ones faced in 1992 and 2005. Hence, compared with the pre- and succeeding scenarios, the shortfall in the 2001 harvest should not have resulted in a major food crisis, even if an average population growth rate of 2 percent per year and therefore a required steady augmentation in output is taken into consideration. But, the situation was aggravated by the fact that Malawi was already in economic recession for some time with a commercial agricultural sector, that suffered from low commodity prices, as well as, declining employment opportunities and both formal and informal sector wages falling in real terms due to high inflation and the devaluation of the Malawian currency, the Kwacha (Kw). Given the economic downturn

not much *ganyu*[6] work was offered either. Moreover, anecdotal evidence suggests that, at the climax of the crisis people that managed to find occasional work, requested to be paid directly in maize instead of cash, which can be seen as an indicator for the high insecurity on maize prices and maize availability (Bryceson, 2006). Therefore, despite a bumper harvest in 1999/2000 the fall in maize production in 2000/2001 hit an increasingly vulnerable rural economy where people had very little to fall back on. A closer look at the price developments (figure 2)[7] shows that even when adjusted for inflation, the average maize market price peaked in 2002 indicating a major food shortage.

[Insert figure 1]

Various other factors further worsened the situation: First, due to 60,000 metric tons of maize held in stock by the National Food Reserve Agency (NFRA) and an estimated high roots and tuber production, the approximate maize shortfall of 272,975 metric tons was considered severe, but the total food availability assessed to be more than adequate. However, the complacency of the food situation was based on a lack of information (and partly wrong information). In fact, the roots and tuber production at that time had been largely overestimated and even though the first reports of a looming food crisis circulated already as early as August 2001 (Devereux, 2002b), the Famine Early Warning Systems Network (FEWSNET) (2002a), for example, still, in January 2002, predicted an overall food surplus in the country for the season 2001/2002. Only by the end of February 2002 the President of Malawi finally declared a state of disaster. Another important factor driving the food crisis was the mismanagement of the Strategic Grain Reserve (SGR) run by the National Food Reserve Agency (NFRA). In August 2000, the SGR held about 175,000 metric tons of maize in stock. Based on advice from the IMF and World Bank, which considered the size of the SGR as excessive at the time, the NFRA sold significant parts of its maize stock to Kenya and Mozambique to avoid the distortion of the local market. To make things worse, a significant amount of maize, namely the before mentioned 60,000 metric tons, which were not officially exported, 'disappeared' from the SGR.[8] One year later, at the onset of the crisis in August 2001, the SGR was left with nearly zero reserves.

[Insert figure 2]

In consequence, the Cabinet Committee on the Economy asked the NFRA to import 150,000 metric tons of maize from South Africa. Due to import delays, price increases and exchange rate movements, finally only 134,000 tons could be purchased. The ordered maize was expected to be delivered at a rate of 50,000 metric tons per month and thus, should have arrived by December 2001 at the latest. But, since imports were deferred by logistical constraints and competition with the neighbouring food deficit countries, Zimbabwe and Zambia, only 94,000 tons arrived in Malawi by April 2002. Experts argued that if the food imports would have arrived at the planned rate, the food crisis could have been prevented. Finally, tensions between the government and donors on

governance issues at the time led to a rather hesitant response with donors only starting to offer unconditional food aid by mid 2002.

[Insert figure 3]

Anecdotal evidence suggests that due to the initial absence of food aid and other support mechanisms, households started to implement various coping strategies, what in Malawi usually means the rationing of meals, increased labour supply and the depletion of assets, such as livestock. Although the latter strategy is usually not observed for poor and very poor households. Moreover, due to the increased supply, prices for cattle, goats and chicken plummeted by more than half between July 2001 and February 2002. For example, while in July prices for goats and chicken ranged between 700-1,000 and 90-200 Kw per animal respectively, they dropped to 250-500 and 30-100 Kw in February (FEWSNET, 2001, 2002b). Thus, selling livestock was not very effective to overcome the crisis. From figure 2 it can be seen, that, prices after 1997 show an enormous volatility. In a poor country such as Malawi, price volatility of that extent is widely seen as detrimental to economic activity and living standards of the poor as they typically have only limited access to appropriate smoothing devices to protect their consumption.

Hence, in sum, the 2002 food crisis cannot only be attributed to a production shock but rather to a number of additional events that were directly or indirectly related to the crisis. In addition the agricultural sector suffered under a more general political neglect of smallholders, declining soil fertility, restricted access to inputs (from the 1990s on), the generally high market power of traders that have the capacity to keep producer prices low and push consumer prices up, and, last but not least a pronounced HIV/AIDS epidemic together with its social and demographic consequences (see Devereux, 2002; Rubey, 2003). However, it is important to note that there was a substantial regional variation in the severity of the food crisis. Due to the exposure to bad weather, lack of diversity in agricultural production and trade restrictions, the effects of the crisis were much more pronounced in the central and southern regions than in the northern regions which, for example, benefited from cross-border trade with Tanzania and a higher roots and tuber production.

As mentioned, there are no officially approved estimates of how many people died due to the shock under study. The same is true with respect to malnutrition, where data is also only sporadically available. The nutrition surveys commissioned by *Save the Children UK* in December 2001 and February 2002 stated an alarming deterioration in malnutrition rates in the Salima district (Central Region) from 9.3 to 19.0 percent in just two months (Devereux, 2002a). However, due to the combined effect of prevention and treatment, malnutrition decreased again from March 2002 onwards, and in June 2002 rates seem to have been down again at 9.7 percent (Taifour, 2002). A further reduction was recorded by September 2002 when malnutrition dropped to 3.8 percent (Taifour, 2002). This, however, is probably a biased estimate, since the month September is in the middle of the post-harvest season.

However, more recently, the country seems to have made further progress in reducing malnutrition rates. The latest global hunger index rating (2009) shows a drop from 32.2

points in 1990 to 18.5 points in 2009 (von Grember et al., 2009). Henceforth, the country moved from the group of extremely alarming countries to the group of serious countries (von Grember et al., 2009), still indicating that malnutrition and its effects remain a problem in Malawi, with about 35 percent of the total population undernourished (UNDP, 2007) and causing child death in more than 50 percent of the cases (see Pelletier, 1994; Pelletier, et al. 1995). The 2006 Poverty and Vulnerability Assessment showed that there is not much dietary diversity in Malawi. 93% of the total cereal consumption is accounted for by maize. The Central Region, despite being the area with the highest calorie availability, has also the highest incidence of chronic child malnutrition (Republic of Malawi, 2006). However, here it is important to note that there are different types of malnutrition - the most common being protein-energy malnutrition, which is commonly reported during famines (Kloos and Lindtjorn, 1994). The health consequences of protein-energy malnutrition include stunted growth, body wasting, retarded mental development and high mortality of young children. Anthropometric measures, such as height-for-age (HAZ), weight-for-height (WHZ) and weight-for-age (WAZ)[9], are typically not perfect indicators to measure nutritional shortfalls (Kloos and Lindtjorn, 1994), nevertheless they are commonly used and are also used in this article. Looking at these indicators for Malawi, one can state that hardly any significant progress has been made over the period 1992 to 2004 (table 1).

[Insert table 1]

Moreover, the figures in table 1 seem to suggest that boys are slightly more suffering from malnutrition compared to girls. Studies by Guha-Khasnobis and Hazarika (2007), Hardenbergh (1997) and Kabubo-Mariara et al. (2006) in different contexts confirm this finding. Others find a female bias (see Klasen, 1996). But, many studies in the literature do not find any gender bias – particularly not in Sub-Saharan Africa (see e.g. DeRose et al., 2000; Gunderson et al., 2007). Hence, the evidence is mixed and no generalization can be made.

4. Empirical Strategy and Data
4.1 Specification and Variables

This study is an attempt to identify the short run effects of the 2002 food crisis on child health in Malawi. Since there was significant regional disparity in the severity of the crisis across the districts of the country, we will use this source of variation to identify the causal relation between child health and food shortage. The regional disparities in crisis severity provide an 'exogenous variation' to determine the causal effects of the food crisis. On the basis of this spatial variation and the variation over time retrieved from the data, we can construct a difference-in-difference (DID) estimator, i.e. we compare the differences in child health in affected ('treatment group') and unaffected districts ('control group') before and after the crisis. We use data from 2000 and 2004. The

information from 2000 will serve as the baseline. The assumption is that while the treatment group has been exposed to a 'treatment' in 2004, i.e. in this case was suffering from food shortages in 2002, the control group has not been exposed to it.

If the sample units observed in each time period were the same, i.e. if panel data was used for estimation, the average effect in the control group would simply be subtracted from the average effect in the treatment group. This would ensure that biases are removed in the second period comparison between the treatment- and control group, which could result from permanent differences between the two groups. In the present case, the data set at hand is not a panel but consists of repeated cross sections. The basic principle remains the same but econometric estimation is slightly different as panel properties cannot be fully exploited. More precisely, panel data allows tracing the individual behaviour over time while repeated cross-sectional observations only allow making a statement on average. Hence, we estimate:

$$y_{i_{n_t}} = \beta_0 + \delta_1 dT_{in} + \delta_2 d2_{in} + \delta_3 d2_{in} \cdot dT_{in} + u_{\text{int}} \tag{1}$$

where y is either the discrete observation if a child died or not which is assumed to represent the latent variable of the probability of mortality, or the different z-scores, which serve as an indicator for the prevalence of malnutrition. The subscript i indicates the observation unit or the individual, n the region and t the time period. With T representing the treatment group, the dummy dT captures possible permanent differences between the treatment- and control group, the dummy $d2$ is a time effect and absorbs aggregate factors that would cause changes in the outcome variable over time for all observation units even in the absence of a shock or intervention. The effect of interest is the so-called treatment effect, δ_3, which is associated with the interaction between belonging to the treated group and the time effect. The treatment effect can also be represented as:

$$\delta_3 = (\bar{y}_{T,2} - \bar{y}_{T,1}) - (\bar{y}_{C,2} - \bar{y}_{C,1}) \tag{2}$$

and captures the difference in means within the treatment and control group before and after the crisis. This difference provides an estimate of the impact of the food shortage on the respective outcome variable.

Considering that the basic estimation equation only includes variables directly related to the shock, the results might be suffering from an omitted variable bias. Therefore, additional variables will be included in the estimation equation to account for possible differences between the treatment and control group:

$$y_{int} = \beta_0 + \beta_1' C_{int} + \beta_2' M_{int} + \beta_3' H_{int} + \delta_1 dT_{in} + \delta_2 d2_{in} + \delta_3 d2_{in} \cdot dT_{in} + u_{int} \tag{3}$$

where C is a vector of child characteristics, including gender, birth size, if the child was a twin and the months breast fed. In M we include information on the mothers, i.e. the years of education, the marital status of the mother, and her age at birth of the child. A

number of studies (e.g. by Miller et al., 1992; Mwabu, 2009; and Sastry, 1997), have found a U-shaped relationship between maternal age at birth and child mortality. Young and old mothers have shown to exhibit higher risk of mortality due to immature reproductive systems and declining maternal resources. The parabolic relationship could not be confirmed for our data. The anthropometric measures of the mother are included in *M* in the regressions for the nutritional outcomes. The rational is that children that were breastfed during the period of the crisis may have been less exposed to the crisis by relying on their mothers resources. Breastfeeding mothers in turn may have suffered more than other women by sharing their 'resources' with their babies. Hence, it is important to control for both, breastfeeding status of the children and mothers z-score. The vector *H* contains household specific information, i.e. whether the household is rural or urban, whether the household is engaged in agriculture, whether the head of the household is a female, the number of household members and the number of children under five. The economic living standard of a household is represented by a dummy variable, based on an index which has been constructed using principal component analysis (see e.g. Filmer and Pritchett, 2001), which allows to measure the household wealth from the possession of household consumer durables such as a bicycle, a radio or a television set.[10]

Since child mortality is represented as a binary variable, i.e. taking the value one if the child died and zero otherwise, a probit model is used for estimation in order to obtain consistent estimates. The z-scores of the children have been calculated based on the 2006 WHO reference standards for child growth. The z-score for height-for-age is obtained by subtracting the median height in the reference population of a child of the same sex and age in months from the child's height, and dividing it by the standard deviation of the height in the reference population, also for a child of the same sex and age. The weight-for-age or weight-for-height z-scores are defined analogously, except that the standardization is done using the reference population median and standard deviation of the of weight of children of the same sex, age and height. Since the z-score of the anthropometric measures are continuous variables with a distribution close to normal the standard OLS technique is used for estimation.

It should be noted that due to potential measurement and reporting errors, the estimates on child mortality are likely to be downward biased. The incidence of child mortality is usually underreported. The anthropometric z-scores, and in particular the weight-for-age and weight-for-height z-scores, are probably also affected by measurement error. The data used for calculation of the z-scores does for instance not include information on the existence of oedema at the time of the measurement, which means that for the calculation it was assumed that the children had no oedema and, hence, z-scores on WAZ and WHZ are potentially overestimated as oedemas cause a weight increase.

4.2 Identification

The separation of the treatment and control groups is obviously a central issue when using difference-in-difference estimation (see e.g. Angrist and Kruger, 1999). Particularly

important is the appropriate choice of the control group (see e.g. Abadie et al., 2007; Bertrand et al., 2002; Kubik and Moran, 2003). To identify the treatment and control group we would ideally rely on an indicator measuring the availability of food across space in Malawi at the time of the crisis. In absence of such a measure, one may use maize production levels as a proxy, but as argued in Section 2 looking at the production levels over the years, the 2002 food crisis was probably not a crisis in terms of overall output. Therefore, production levels do not seem to be a useful indicator in this context. Other papers investigating the effects of droughts on children's health often used rainfall data (e.g. Akresh and Verwimp, 2006). For two reasons, we refrained from using such data for identification: First, the available rainfall data is incomplete and, second, not all areas that were flooded early 2001 experienced food shortages, as a number of other factors, such as the possibility for cross-border trade, mitigated the effect of the food crisis. Instead we decided to use the consumer price for maize as an indicator. This is considered to be a suitable indicator to measure food shortages across regions in this case because, first, prices can be seen as a measure of scarcity if no major price distortions in the market exist; second, there is significant variation in price levels across the districts which do largely tie in with a number of qualitative reports on food shortages (see figure 4).

[Insert figure 4]

For instance on the market in Chitipa in the North of the country (see figure 4 above) maize was sold at much lower prices during the crisis than in the other two locations and indeed the majority of the northern districts have not reported any severe food shortages.

To identify the areas in crisis, we computed the annual average of the monthly maize market prices for selected markets throughout the country. We set the threshold average price at 20 Kw/Kg for 2002, as it was reported to be the highest price to which the NFRA would buy maize. On the basis of this limit, we coded districts with an average maize market price above 20Kw/Kg as belonging to the 'treatment group', while we coded the others as belonging to the 'control group'. Through this mechanism, the following regions have been identified to be affected by the food crisis: Nkhotakota, Ntchisi, Dowa, Mchinji, Salima, and Dedza in the Central Region and Mangochi, Machinga, Zomba, Phalombe, Mulanje, Thyolo, Balaka and Mwanza in the Southern Region. The other districts predominantly in the Northern Region are classified as not-affected.

To test the sensitivity of our results, we use various alternative ways to distinguish between treatment and control groups. First, we vary the above mentioned price threshold. Second, we sort districts by distinguishing whether they are mainly maize or tuber and roots-producing, assuming that the predominately tuber and roots-producing areas were less affected by food shortages due to a good harvest in the 2001/2002 season. Third, we use the classification from the emergency assessment carried out by the FAO and WFP in May 2002 immediately after the high point of the crisis was reached.

4.3 Data

In order to implement the strategy described above, we use pooled cross-sectional data from the Demographic and Health Surveys (DHS) conducted in Malawi in 2000 and 2004, which are representative at national, regional and the rural and urban level. We use predominantly the sub-sample containing the information on children less than five years of age and the respective household and mother characteristics. The combined dataset of both years includes in total 22,840 observations, 11,926 from 2000 which serve as baseline and 10,914 from 2004. The response rate for the underlying data on the anthropometric indicators was 77.5 percent in 2000 and 74.8 percent in 2004. We did not find any systematic bias when regressing a dummy for missing values on household and mother's characteristics. There are no missing values in the module on child mortality.

Comparing the affected and non-affected districts over the years, on the basis of the descriptive statistics presented in the appendix (table A1) allows getting a first naïve estimate of the health effect of the crisis, i.e. of the difference-in-difference. It can be seen that the percentage of children that died before the age of 5 dropped from 13.8 percent to 10.4 percent in affected districts and from 12.1 percent to 8.7 percent in non-affected districts. Thus the reduction in child mortality was in both areas 3.4 points. The anthropometric measures WAZ, HAZ and WHZ show diverging trends. While the WAZ has improved over time in both groups, even slightly more so in affected areas (although from a lower initial level), the HAZ fell in the affected districts, but increased in the non-affected districts. Acute malnutrition, i.e. WHZ, improved quite substantially, again in both affected and non-affected districts. In the base year both acute malnutrition (WHZ) and chronic malnutrition (HAZ) are slightly higher in affected areas. The multivariate analysis will show how both groups compare over time if potentially confounding factors are controlled.

The descriptive statistics show to what extent children differ in affected and non-affected districts and, in particular, whether their characteristics developed differently over time. There is no difference in the share of boys in both areas. Breastfeeding increased over the years: Children in non-affected areas were on average fed 0.39 months and 0.83 months longer in 2000 and 2004 respectively. The size of the children at birth seems to have increased over time, with children in the non-affected districts being slightly larger. Mother's education improved in both groups by about 0.4 years of schooling, which is probably a pure cohort effect. The lower average number of years at school in affected districts suggests again that households in these areas are on average slightly poorer than in non-affected districts. This is also reflected by the larger share of households falling into the group of the 40 percent poorest households in terms of the possession of assets in affected regions. Mother's anthropometric measures are not significantly different in both years but are slightly worse in affected areas. Interestingly, the BMI shows that on average the population still lies within the normal range of 18.5 to 25 points. In 2004 more households are located in rural areas and are engaged in agriculture compared to 2000. Average household size declined in both groups over time and is slightly lower in affected districts compared to non-affected ones. The structural differences between both groups highlight the importance to include control variables in the difference-in-difference estimation. Regarding the unobservable factors that are

unrelated to the crisis but correlated with health outcomes, we must assume that these did not change differently for both groups in the period under study.

A further issue that needs attention is whether the sample size is large enough to detect excess mortality of the order of magnitude mentioned in the media during and after the crisis. Taking the standard significance level of 95% (one-sided) and making the most conservative assumption about the variance in mortality in the total population, we find that we can detect excess mortality between the affected and non-affected areas of 8,360 death events and more. Smaller differences would need a larger sample to be detected with certainty. Hence, the sample is at least large enough to confirm or not the high estimates put forward by several NGOs. Smaller difference in turn would imply that the crisis was by far less severe as often assumed.

5. Discussion of the Results
5.1 Effect of the Food Crisis on Child Mortality

The results of the estimated probit models specified as described in Section 4.1 are presented in table 2. Column (1) shows that, as already suggested by the descriptive evidence above, those areas presumably affected by the food crisis, have not experienced a higher child mortality than the non-affected areas controlling for general time and area effects. This result does also not change if we introduce a large set of control variables in the regression (see columns (2) and (3)). This suggests that the food crisis did not have an impact on child mortality in the order of magnitude put forward by some NGOs. Before, we analyze the effects on children's anthropometric measures; we briefly discuss the estimated coefficients of the control variables.

[Insert table 2]

The majority of the control variables are statistically significant. As expected, twin birth is positively related to the incidence of child mortality, hence, increasing the probability of child death in the early years of live. Boys also have a higher risk to die before the age of five, although this effect is very small in quantitative terms. Moreover, the results indicate that breastfeeding increases the chance of child survival. The results obtained for the educational attainment and marital status of mothers are also as expected. Both coefficients are inversely related with child death, thus education and being married increase the chances of child survival. In the present case an additional year of education at the sample mean (3.8 years of schooling) reduces the probability of child mortality by 0.3 percentage points. This finding is consistent with other results from the literature. Katahoire et al. (2004), for instance, find for south-eastern Uganda that maternal schooling, while not protecting the children from malnutrition and morbidity had a positive impact on child survival, i.e. the risk of mortality was higher among children of mothers without any formal education. Living in rural areas is associated with higher child mortality. Interesting are the coefficient of the dummy variables for female headed

households and the number of children under five in the household (column 3). Both variables are negatively related to child mortality. It is hard to say why living in a female headed household reduces the probability of a child's death. On the one hand, one could argue that female headed households are found to be poorer than male headed households. On the other hand, there is an extensive literature confirming that women put more emphasis on food, health and education expenditure than men, thus, benefiting the children. Moreover, the concept of a female headed household is very controversial. Female headed households are typically a very heterogeneous group, in the sense, that they are not necessarily poorer or exposed to more vulnerability because the male had died for example, but instead moved away and supports the family through remittances, which in turn makes the household less vulnerable to shocks. For instance, Kennedy and Haddad (1994) investigate if pre-schoolers from female headed households were less malnourished using weight-for-age z-scores comparing Ghana and Kenya and find a substantial positive effect for Kenya and a lower effect for Ghana. In Ghana, in turn, income has been identified as an important determinant of children's nutritional status.

The coefficient of the number of children under five in the household (see column (3)) indicates that, the more children live in the household, the smaller the probability of a child's death. More specifically, one additional child under five reduces the probability of child mortality at the sample mean (1.7) by 7.5 percentage points. However, it should be noted that this coefficient is probably upward biased, since households with many children under five are probably those households that experienced less child mortality in the past, thus this variable has to be considered as endogenous and its coefficient should be interpreted with caution.

5.2 Effect of the Food Crisis on Child Anthropometry

Table 3 shows the results for all three anthropometric measures: WAZ, HAZ and WHZ. In the first three columns we present again first the results of the regressions, in which we just use the time and area effects and the corresponding interaction term. Looking at the interaction term, i.e. the 'treatment effect', we see that children from treatment regions experienced slightly larger improvements in their nutritional status than children in control regions (significant at 1 percent for specification 2). More precisely, children from the affected districts exhibit 0.14 SD higher weight-for-age and 0.15 SD higher height-for-age z-scores than children in non-affected districts. Only the measure of acute malnutrition (weight-for-height), does not show any significant difference.

As before, we now check whether the results are robust to the introduction of further control variables. We now also include the corresponding z-scores of the mother in the set of covariates, for the reasons outlined in section 4.1. In general the used variables can better explain WAZ and HAZ than WHZ. In particular household level characteristics are only a poor predictor of weight-for-height z-scores. The latter seem to a large part driven by the child's own endowment regarding the size at birth or being a twin birth, being breastfed, as well as, the mothers anthropometric status. The coefficient of mothers' z-

scores is positive, suggesting that better nourished mothers can more easily breastfed and protect their children from starving.

In sum the above assessment shows that the crisis did not have a dramatic impact on mortality and the nutritional status of children, at least not of the order of magnitude reported by some NGOs. The slight nutritional improvement stated for children in affected districts suggests that food aid or other interventions were probably quite effective. This possibility and the general robustness of our results will be discussed next.

[Insert table 3]

5.3 Caveats

In this section we discuss critically some of the assumptions underlying the approach used in the previous section. Thereafter, we test, where possible, the robustness of our results using alternative assumptions.

First, when using a difference-in-difference estimator one has to assume that the changes in the outcome variable in the absence of a shock would have been exactly the same in both, the affected and non-affected districts; or in other words, that the treatment and control group follow constant and parallel trends in absence of treatment. Yet, to account for the possibility of a potentially diverging trend a number of control variables have been included in the regressions. However, of course we cannot exclude the possibility that there are other unobservable factors that are systematically different between the treatment and control group and are related to changes in children's health.

Second, it is difficult to measure what is due to the direct effects of the drought and what is due to other shocks that occurred at the same time but dissimilar in both areas or due to policy interventions that responded to the drought. For example, if in the period of study the HIV/AIDS epidemic evolved differently in both regions, then these effects may be confounded with the effects of the drought. Although such a development is rather unlikely, the approach we have chosen cannot exclude that possibility. Food aid that was targeted to affected districts in response to the crisis would also mitigate the health impact in affected regions compared to non-affected regions.[11] Hence, our analysis just allows looking at the net effect in 2004, i.e. after some coping mechanisms have already been implemented. We cannot construct a counterfactual that looks at the potential impact without such mechanisms. But this would anyway not be helpful to answer the question what the actual impact on mortality and children's health has been. Relying on such a counterfactual, would mean to overestimate the actual health impact.

Third, from the descriptive statistics in table A1, we know that households in the control area are slightly richer and more educated. Although, we control for these variables in the regressions, it is possible that these characteristics help households in control groups also to cope easier with shocks than households in treatment groups. This would mean that part of the difference we see between the control and treatment group could stem from the fact that households in the treatment area are particularly vulnerable

to such shocks. Since vulnerabilities to food shocks are not random and neither are coping strategies, in the sense that wealthier households would probably be better prepared, through production-, saving-, and insurance decisions, poorer households in areas with frequent food insecurities are likely to be less able to smooth out income shocks. In either case the treated and non-treated would respond differently to the crisis. Moreover, shocks like a drought may actually not be fully exogenous, but rather endogenous in a sense that regions prone to natural disasters and disease "attract" mainly the poor, while the non-poor sort to more hospitable environments. Hence, the impact of the crisis is likely to be higher in affected areas than in the control areas, as the control population is more capable to deal with covariate shocks and is less exposed to such shocks. In other words, the endogeneity of the 'severity' of the food crisis may lead to a biased estimate of its effects on child mortality and children's health. However, it would mean that we rather over than underestimate the consequences of the food crisis. Given that we find only limited if not no evidence for a severe impact of the food crisis on children's health this potential source of bias seems not to be very important. However, to see whether such endogeneity problems are a concern, the estimations for child mortality and anthropometry have also been carried out for the poor and non-poor population separately (see Section 5.4 below). The results confirm the findings obtained in the previous sections; no substantial impact on child mortality and rather better nutritional outcomes for children in the affected areas compared to the control areas.

Fourth and last, our identification strategy would be problematic if there was substantial migration between the treatment and the control area. If such migration was important and if migrants had unobservable characteristics different from the population at destination and if these characteristics were correlated with the observed health outcomes, our estimation would be biased. The data source used in this paper does not allow studying migration, since it only provides information on women and children under five and the total number of household members. An assessment just based on household size is difficult and might be misleading. Anyway, we do not find a systematic decline in household size in treated districts and an increase in control districts, which if it had happened could be a sign of child fostering. A study by Makoka (2008) shows that migration is not a widely used strategy to cope ex-post with drought or increased food crop prices in the context of Malawi and hence we assume that the potential bias from that source is negligible.

5.4 Sensitivity Analysis and Robustness Checks

To address some of the potential problems discussed above, the following robustness checks were performed. First, we re-estimated the results using alternative identification strategies, e.g. by classifying the regions into tuber and non-tuber growing areas with the underlying argument, that the areas producing and consuming tuber as a staple food would not be affected by a food crisis resulting from shortages in maize. Other identification strategies used to check for the robustness of the results were a separation of the treatment and control group based on the food security and vulnerability

assessments carried out alternatively by the Malawi National Vulnerability Assessment Committee (MNVAC) in September 2002 and the FAO and WFP in May 2002. The former two identification strategies were found to have a number of drawbacks and did not yield conclusive results, mainly because the separation into tuber and non-tuber growing areas was not sharp enough and the vulnerability assessment by the MNVAC was carried out too late and thus overlapped with the start of the next harvest. Hence, the FAO and WFP assessment is the most convincing instrument. In fact, the FAO and WFP assessment was carried out very close to the high point of the food crisis. The FAO and WFP (2002) have classified seven districts as facing a high severity of the food crisis: Nkhotakota, Salima, Lilongwe, and Ntcheu in the Central Region and Mangochi, Blantyre and Zomba in the Southern Region. For the estimation these districts were defined as the treatment group, while the rest of the country was defined as the control group. The results obtained confirm the main conclusions drawn in section 5.1 and 5.2. They also show no impact of the shock on the probability of child mortality, as well as, no significant disadvantage of the affected areas with respect to malnutrition.

Second, we re-estimated the difference-and-difference specification for various sub-groups: poor and non-poor, rural and urban, male and female, households engaged in agricultural activity and non-agricultural households, the education level of the mother, and further sub-combinations such as by gender and poverty status. In most cases we obtained qualitatively the same results as in Section 5.1 and 5.2, i.e. no impact on child mortality and acute malnutrition and a slight improvement in chronic malnutrition in affected areas. However, when analyzing the differential impact by gender and poverty status, we found that boys from poor households in the affected areas have a weight-for-height z-score 0.31 SD higher relative to their non-affected counterparts, while girls from poor households in the affected areas lost in their weight-for-height z-score (0.23 SD lower) relative to their control group. So there seem to be minor differences in the impact if the sample is disaggregated by gender and income, without however changing our overall result. Separating the sample with respect to the educational attainment of the mother revealed no differential impact of the food crisis on the child anthropometry when mothers had no formal education. However, we found a positive impact with respect to chronic malnutrition and underweight. More precisely, children of mothers with primary education in the 'treatment group' gained on average 0.18 SD more in their weight-for-age z-scores and 0.21 SD more in their height-for-age z-scores than children of mothers without education. Hence, mother's education may help to better cope with such shocks.

Third, we have also re-estimated the model including district dummies to account for district fixed effects. However, these were not significant and did not change the results presented in Sections 5.1 and 5.2.

A fourth and final variation in the estimation strategy was made by replacing the treatment dummy dT with the market price information combined with district fixed effects. That creates more variation in the treatment variable. The results obtained have, again, not significantly changed with respect to mortality, the degree of underweight and chronic malnutrition. For acute malnutrition, measured by the weight-for-height z-score, we found the interacted time and treatment variable to be significant and positive at 10 percent. The positive sign of the coefficient indicated, as the results above, that children

in the affected area were actually experiencing relative gains in their weight-for-height z-score (0.02 SD higher) in the period around and following the crisis.

6. Concluding Remarks

We analyzed the impact of the 2002 Malawi food crisis on child mortality and child malnutrition using the Malawian Demographic and Health Surveys undertaken in 2000 and 2004. For identification of the impact we used a difference-in-difference estimator defined over affected and non-affected districts. Districts were grouped in one or the other group according to the level of local market prices for maize during the period of the crisis. Our results suggest that the food crisis has not led to a significant increase in child mortality or acute undernutrition in affected areas. We even find some evidence for a slight improvement in children's underweight and chronic malnutrition. Our estimates imply in affected districts on average 0.14 standard deviations higher weight-for-age scores and 0.15 standard deviations higher height-for-age z-scores relative to non-affected districts. These results have withstood to a large extent our sensitivity analysis and various robustness checks. It is difficult to say what was behind these improvements, but food aid and other aid following the crisis could be an important factor.

The finding of a limited impact on child mortality does in fact well tie in with the results from Howe and Devereux (2004) who classified the Malawian food crisis of 2002 as a minor famine compared to the events in Sudan in 1998 and Ethiopia in 2000 which caused, so the estimates, proportionately more deaths by starvation and hunger related diseases. Regarding the impact on malnutrition, it should be noted that the weight-for-height z-score, used to estimate acute undernutrition, is in principle the best indicator to measure the short or better very short-term impact of food shortages. However, the observations used here are based on data collected two years after the crisis and hence it is likely that other events, as well as, the gradual disappearance of the crises introduce noise in this measure. This is less an issue for underweight and chronic malnutrition and of course for mortality, which is based on retrospective information, but the impact of the food crisis on these measures is anyway expected to be smaller as these measures are by definition less volatile over a short period of time.

Again, one potential explanation for our results could be that the interventions and policy measures taken at the time were actually successful to tackle the negative impact of the crisis. And this would be good news. Obviously it is impossible to assess the effectiveness of any particular project with the data at hand. Moreover, probably it was rather the combination of different projects that helped to mitigate the effects of the crisis.[12] However, it is worth to focus briefly on the two major programs initiated in response to the crisis – the Joint Emergency Food Aid Program (JEFAP) and the Extended Targeted Input Program (ETIP). The JEFAP was based on the Emergency Operation (EMOP) of the WFP. A consortium of NGOs implemented and distributed food aid in the districts. The EMOP started in June 2002 with the initial objective to distribute 56,500 metric tons of food commodities to 2.1 million targeted beneficiaries in 18 districts by September 2002 (FEWSNET, 2002c). The main target groups were

children under five, pregnant and lactating women and the elderly. Because of ongoing reports of households facing food shortages even after the 2002 harvest, food aid, funded mainly by USAID and the EU was extended beyond its initial period and in some areas provided assistance until early 2004. Moreover, to increase the maize production after the crisis, the Government with financial support from the World Bank, DFID, and Norwegian Aid, extended the subsidized input program to reach three instead of formerly one million households (FEWSNET, 2002d). The ETIP was sustained at this level for several seasons after the crisis. Considering that both of these large-scale programs were targeted and extended over a long period of time and well beyond the end of the crisis, nutritional improvements in affected districts are plausible and would be in line with the conclusion drawn by Yamano et al. (2005) in their study on Ethiopia that food aid can be very effective if the targeting is appropriate.

A final explanation for the results could be 'adaptation'. As mentioned in section 3 Malawi is frequently hit by adverse weather shocks which typically result in serious production shortages and hikes in the consumer price of food crops. For instance, in the period from 1990 to 2006, Malawi faced about 16 of these shocks. Furthermore, during the pre-harvest period starting around January food rationing is common for most of the agricultural households in rural areas. Therefore, their bodies might be more able to adapt to periods of food shortage without showing any immediate negative effects. Work in this area is spares and inconclusive. Ferro-Luzzi et al. (2001), for example, studied the magnitude of seasonal undernutrition in two areas in Ethiopia and even though their results are very mixed, the nutritional status of school aged children does not seem to be affected by seasonality. However, this hypothesis needs further research before any generalization can be made.

We end this analysis with a final word of caution. We have considered only the potential short and medium impact of the food crisis, but, for the moment, had to ignore the potential long-term effects. It is well known that even temporary food shortages can affect children's cognitive development. These effects will only be visible if affected and non-affected cohorts enter the labor market. Future research should investigate this issue.

Appendix

[Insert table A1]

Notes

[1] We thank Arjun Bedi and Robert Sparrow for useful comments on an earlier draft of this paper.

[2] Even assuming the lowest estimates, the 2002 crisis would have already been more severe than the Nyasaland famine of 1949 with estimated deaths of 200 'only'.

[3] More precisely, the WHO estimated child mortality rates in some of the districts most severely hit at ca. 3.9/10,000 per day (i.e. a doubling of the usual mortality rates of 2/10.000 per day) lasting at least from

April until September 2002. Based on the census data, that would imply an excess mortality of 156 per day or about 9,000 over two months (WHO, 2002).

[4] Former name of Malawi.

[5] The 2005 crisis might actually have been more severe. According to estimates 4.7 million people were affected and in immediate need of food aid (compared to 3.2 million in 2002 (Malawi National VAC, 2002)). But, there is not enough information available to assess the situation (Roshni, 2007).

[6] *Ganyu* is a term commonly used in Malawi to describe a variety of ad hoc and short-term casual labour relationship in rural areas.

[7] Prices displayed in figure 2 are consumer prices. Producer prices are much lower in particular due to powerful traders who make use of information asymmetries and high transaction costs. Maize market prices in Malawi follow a cyclical pattern with a drop in prices after the harvest in June/July and a significant increase at the beginning of the year in January/February, the high point of the lean season.

[8] Anecdotal evidence suggests that high-rank politicians have been involved selling off the stocks to local traders at high returns (Devereux, 2002b).

[9] Height-for-age (HAZ) measures skeletal growth and is considered to be a reliable indicator of long-standing malnutrition in childhood. Weight-for-height (WHZ) is a measure of the deficit in tissue and fat mass and is sensitive to temporary food shortages and episodes of illness. Weight-for-age (WAZ) is a broader measure and does not well discriminate between temporary and more permanent malnutrition (WHO, 1995).

[10] The use of such wealth indices is often criticised: Asset indexes can be biased particularly when comparing rural and urban areas, because they might not correctly reflect income differentials in varying locations due to differences in prices, the supply of assets and durable goods, and the variation in preferences between regions (see e.g. Grimm et al., 2008).

[11] Note that major agricultural and health programmes like the Input Factor Programmes (IFP) have been rolled out nationwide with similar coverage and targeting across all districts. However, we cannot exclude the possibility that smaller NGO projects were more concentrated in affected districts.

[12] See Taifour (2002) for examples of successful feeding programmes in the Salima and Mchinji districts.

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Tables and Figures

Table 1: Anthropometric Indicators for Child Health in Malawi

		Height-for-age ("stunting")		Weight-for-height ("wasting")		Weight-for-age	
		Percentage below -3 SD ("severe")	Percentage below -2 SD ("moderate")	Percentage below -3 SD ("severe")	Percentage below -2 SD ("moderate")	Percentage below -3 SD ("severe")	Percentage below -2 SD ("moderate")
1992	Male	24.5	50.9	2.0	6.0	8.5	28.3
	Female	21.4	46.5	0.9	4.9	6.7	26.1
	Total	**22.9**	**48.7**	**1.4**	**5.4**	**7.6**	**27.2**
2000	Male	25.3	50.5	1.2	5.1	6.0	25.7
	Female	23.0	47.6	1.3	6.0	5.7	25.1
	Total	**24.4**	**49.0**	**1.2**	**5.5**	**5.9**	**25.4**
2004	Male	23.8	50.0	1.9	5.5	4.5	22.4
	Female	20.7	45.6	1.4	4.8	4.5	21.6
	Total	**22.2**	**47.8**	**1.6**	**5.2**	**4.5**	**22.0**

Source: Elaborated, based on NSO and ORC Macro (1993, 2001, 2005)

Table 2: Child Mortality – Probit Regression (marginal effects)

Variable	(1) Marginal Effects	SE	(2) Marginal Effects	SE	(3) Marginal Effects	SE	X^a
Dummy child is a twin			0.122	0.013***	0.196	0.018***	0.038
Dummy child is male			0.009	0.003***	0.007	0.002***	0.501
Breast fed (months)			-0.010	0.000***	-0.007	0.000***	15.592
Dummy small size at birth			-0.022	0.005***	-0.012	0.004***	0.120
Dummy average size at birth			-0.038	0.006***	-0.019	0.005***	0.534
Dummy large size at birth			-0.033	0.005***	-0.018	0.004***	0.202
Dummy very large size at birth			-0.023	0.006***	-0.012	0.005**	0.091
Mothers education (years)			-0.003	0.000***	-0.003	0.000***	3.779
Dummy married			-0.010	0.005**	-0.011	0.005**	0.862
Age at birth			-0.002	0.000***	-0.001	0.000**	25.912
Dummy rural					0.012	0.004***	0.862
Dummy agr. HH					-0.000	0.003	0.537
Dummy medium HH (40th percentile)					0.001	0.003	0.417
Dummy rich HH (20th percentile)					-0.000	0.004	0.174
Dummy electricity							
Dummy female headed HH					-0.007	0.003**	0.189
No. of household members					0.003	0.001***	5.546
No. of children under 5					-0.075	0.003***	1.690
Dummy 2004	-0.037	0.007***	-0.030	0.005***	-0.017	0.004***	0.464
Dummy treatment group	0.016	0.006	0.004	0.004	0.000	0.003	0.576
Time and treatment indicator interacted	0.003	0.009	0.001	0.007	0.002	0.005	0.278
N	*22,840*		*22,838*		*21,799*		
Log pseudo likelihood	*-8085.594*		*-6,325.384*		*-4,907.001*		
Pseudo R²	*0.005*		*0.222*		*0.370*		

Note: * significant at 10%, ** significant at 5%, *** significant at 1%.

[a] X refers to the mean value of the variable. The mean has been calculated based on the sample used for the estimation of specification (1) (*N*=21,799).

Table 3: Anthropometric Measures – OLS

	(1)						(2)					
	WAZ		HAZ		WHZ		WAZ		HAZ		WHZ	
	Coef.	SE	Coef.	SE	Coef.	SE	Coef.	SE	Coef.	SE	Coef.	SE
Dummy child is a twin							-0.808	0.063***	-0.833	0.074***	-0.249	0.072***
Dummy child is male							-0.112	0.019***	-0.228	0.026***	0.036	0.023
Breast fed (months)							-0.027	0.001***	-0.061	0.002***	0.011	0.001***
Dummy small size at birth							-0.006	0.057	-0.112	0.074	0.046	0.068
Dummy average size at birth							0.310	0.051***	0.148	0.066**	0.179	0.059***
Dummy large size at birth							0.476	0.053***	0.271	0.069***	0.275	0.063***
Dummy very large size at birth							0.639	0.058***	0.281	0.076***	0.514	0.068***
Mothers education (years)							0.021	0.003***	0.019	0.004***	-0.001	0.004
Dummy married							0.025	0.031	0.139	0.043***	-0.005	0.038
Mothers respective z-score							0.064	0.003***	0.230	0.013***	0.246	0.014***
Dummy rural							-0.128	0.031***	-0.153	0.044***	-0.040	0.039
Dummy agr. HH							-0.042	0.021**	-0.093	0.028***	0.022	0.026
Dummy medium HH (40th per.)							0.093	0.021***	0.085	0.029***	0.013	0.027
Dummy rich HH (20th per.)							0.203	0.032***	0.355	0.044***	-0.037	0.040
Dummy electricity												
Dummy female headed HH							-0.048	0.027*	-0.010	0.038	-0.078	0.033**
No. of household members							0.012	0.005***	0.024	0.006***	0.012	0.006**
No. of children under 5							-0.012	0.014	-0.061	0.019***	0.010	0.017
Dummy 2004	0.045	0.029	-0.058	0.042	0.089	0.035***	0.031	0.029	-0.024	0.040	0.059	0.036
Dummy treatment group	-0.153	0.026***	-0.241	0.037***	0.020	0.031	-0.101	0.026***	-0.155	0.035***	0.027	0.031
Time and treatment indicator interacted	0.088	0.039**	0.099	0.055*	0.006	0.046	0.144	0.038***	0.147	0.052***	0.043	0.047
Constant	-0.923	0.019***	-1.797	0.027***	0.239	0.022***	-2.242	0.106***	-0.693	0.107***	-0.037	0.094
N	18,690		17,725		17,768		17,719		16,820		16,828	
R^2	0.003		0.003		0.001		0.115		0.138		0.033	

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

Table A1: Descriptive Statistics

|	**2000**							**2004**						
	Affected			**Non-affected**				**Affected**			**Non-affected**			
Variable	N	**Mean**	**SD**	N	**Mean**	**SD**	**p-value**[*]	N	**Mean**	**SD**	N	**Mean**	**SD**	**p-value***
Dummy child died	6,622	0.138	0.345	5,304	0.121	0.326	0.005	6,547	0.104	0.305	4,367	0.087	0.281	0.003
Height-for-age SD	5,176	-2.037	1.777	4,231	-1.800	1.792	0.000	4,875	-1.997	1.772	3,443	-1.855	1.840	0.000
Weight-for-age SD	5,377	-1.076	1.330	4,413	-0.923	1.276	0.000	5,276	-0.943	1.302	3,624	-0.878	1.307	0.021
Weight-for-height SD	5,171	0.255	1.500	4,251	0.239	1.464	0.505	4,913	0.354	1.555	3,433	0.328	1.561	0.440
Dummy child is a twin	6,622	0.039	0.193	5,304	0.045	0.207	0.091	6,547	0.031	0.175	4,367	0.037	0.188	0.125
Dummy child is male	6,622	0.500	0.500	5,304	0.498	0.500	0.893	6,547	0.510	0.500	4,367	0.500	0.500	0.350
Breast fed (months)	6,622	15.106	9.111	5,304	15.487	8.966	0.022	6,547	15.455	9.225	4,367	16.285	9.321	0.000
Dummy very small size at birth	6,622	0.041	0.199	5,304	0.041	0.197	0.850	6,547	0.035	0.183	4,367	0.041	0.198	0.088
Dummy small size at birth	6,622	0.126	0.332	5,304	0.113	0.317	0.029	6,547	0.122	0.328	4,367	0.109	0.311	0.028
Dummy average size at birth	6,622	0.543	0.498	5,304	0.627	0.484	0.000	6,547	0.502	0.500	4,367	0.455	0.498	0.000
Dummy large size at birth	6,622	0.168	0.374	5,304	0.160	0.367	0.264	6,547	0.224	0.417	4,367	0.266	0.441	0.000
Dummy very large size at birth	6,622	0.114	0.318	5,304	0.051	0.221	0.000	6,547	0.083	0.275	4,367	0.112	0.315	0.000
Mothers education (years)	6,622	3.027	3.168	5,304	4.472	3.473	0.000	6,547	3.517	3.357	4,367	4.818	3.551	0.000
Marital status	6,622	0.846	0.361	5,304	0.892	0.311	0.000	6,547	0.793	0.405	4,367	0.854	0.353	0.000
Age at birth	6,622	25.886	7.054	5,304	25.628	6.610	0.420	6,547	25.812	6.802	4,367	25.736	6.663	0.563
Mother Height-for-age SD	6,543	-1.358	0.974	5,243	-1.279	0.991	0.000	6,252	-1.360	1.004	4,194	-1.269	1.025	0.000
Mother Weight-for-age SD	6,517	-0.708	0.822	5,227	-0.591	0.850	0.000	6,242	-0.708	0.848	4,183	-0.619	0.913	0.000
Mother BMI	6,548	22.001	2.951	5,237	22.276	2.840	0.000	6,252	21.928	2.900	4,194	22.181	3.109	0.000
Dummy rural	6,622	0.876	0.330	5,304	0.762	0.426	0.000	6,547	0.951	0.216	4,367	0.813	0.390	0.000
Dummy agricultural household	6,497	0.504	0.500	5,181	0.471	0.499	0.000	6,052	0.584	0.493	4,071	0.587	0.492	0.741
Dummy poor HH (40th percentile)	6,622	0.416	0.493	5,304	0.372	0.483	0.000	6,547	0.482	0.500	4,367	0.335	0.472	0.000
Dummy med. HH (40th percentile)	6,622	0.429	0.495	5,304	0.355	0.479	0.000	6,547	0.420	0.494	4,367	0.455	0.498	0.000
Dummy rich HH (20th percentile)	6,622	0.155	0.362	5,304	0.273	0.446	0.000	6,547	0.097	0.297	4,367	0.210	0.407	0.000
Dummy electricity	6,472	0.034	0.182	5,113	0.060	0.238	0.000	6,456	0.035	0.183	4,294	0.072	0.259	0.000
Dummy female headed household	6,622	0.232	0.422	5,304	0.173	0.378	0.000	6,547	0.212	0.409	4,367	0.147	0.354	0.000
No. of household members	6,622	5.484	2.532	5,304	5.799	2.455	0.000	6,547	5.366	2.126	4,367	5.694	2.312	0.000
No. of children under 5	6,622	1.661	0.871	5,304	1.696	0.872	0.032	6,547	1.710	0.796	4,367	1.707	0.861	0.876

Note: * The *p*-value reported gives an indication about the potential equality of the means (null hypothesis).

Figure 1: Maize Production in Malawi (1986-2006)
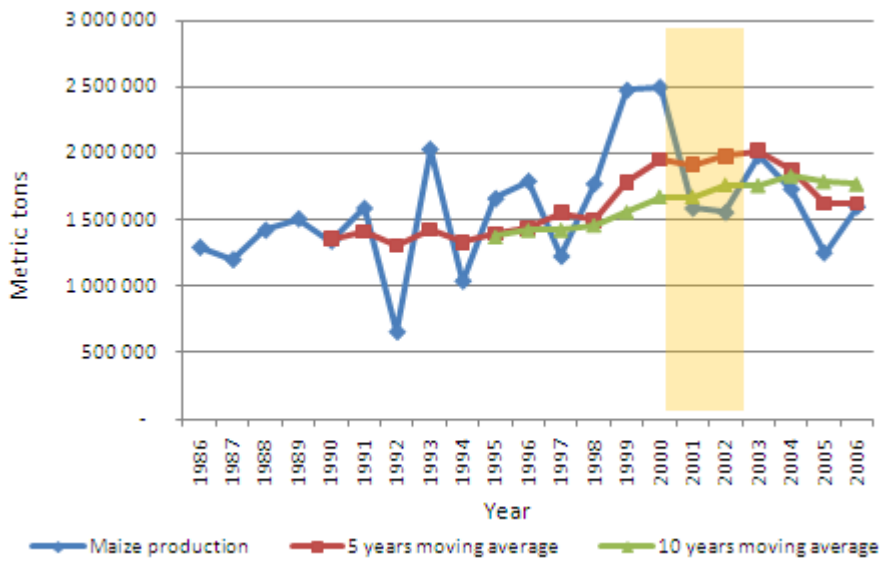
Source: Data from FAO Statistics Division (2008).

Figure 2: Real National Average Maize Market Price 1990 – 2007 and Monthly Average Prices during the Crisis
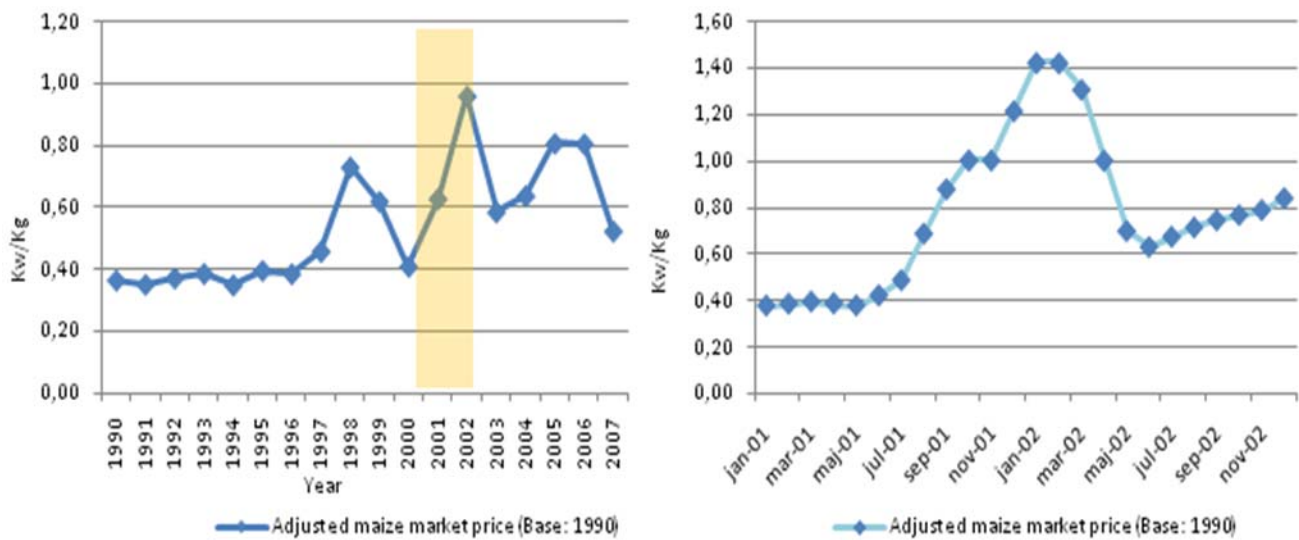
Source: Data from FEWSNET and NSO (2008)
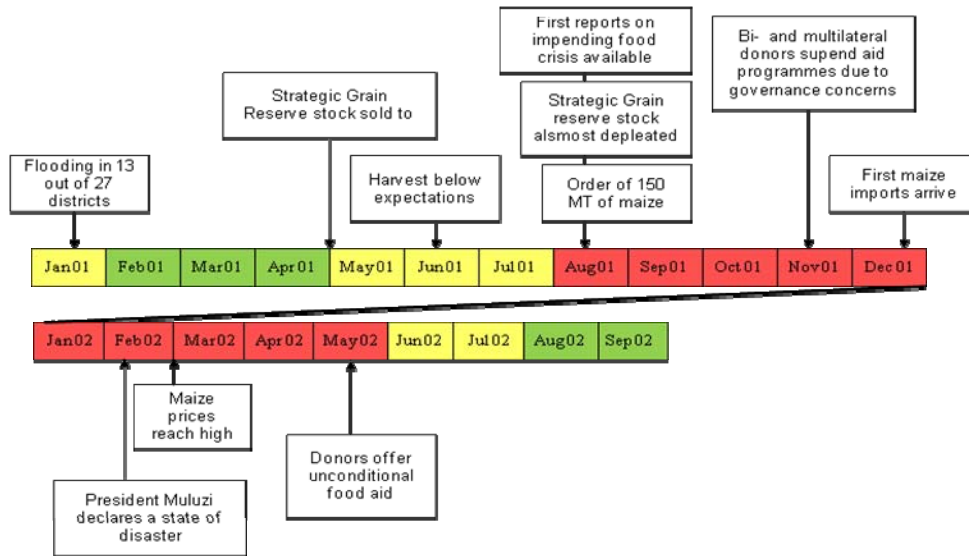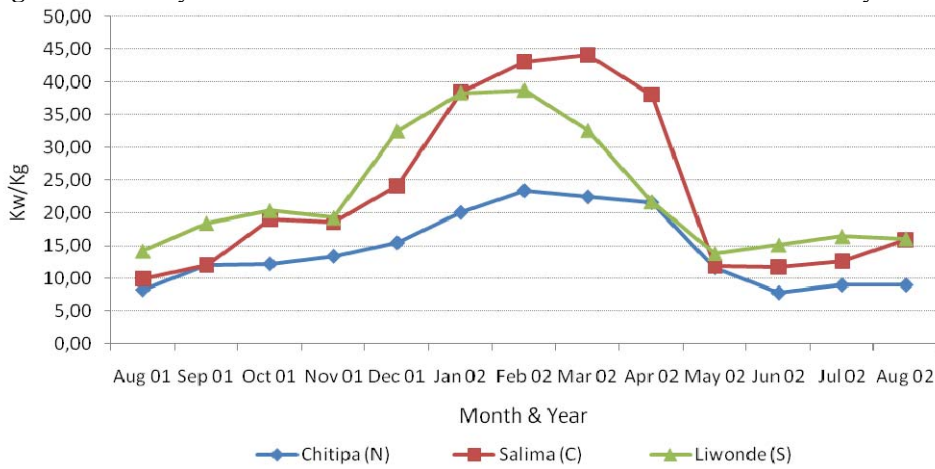
Figure 3: Timeline of the Crisis

Figure 4: Monthly Maize Market Prices from Selected Markets across the Country

Source: Data from FEWSNET (personal communication)