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The Power of Markets: Impact of Desert Locust Invasions on Child Health

Abstract

This paper investigates the consequences of a locust plague that occurred in Mali in 2004. We argue that in agricultural economies with a single harvest per year, this type of shock can affect households through two channels: first, a *speculative/anticipatory effect* that kicks in during the growing season, followed by a local *crop failure effect* after harvest. We show that, in terms of health setbacks, children exposed in utero only to the former suffered as much as those exposed to the latter. We also document a substantial impact of the plague on crop price inflation before the harvest, as well as a stronger *crop failure effect* for children born in isolated areas.

JEL-Codes: O120, I150, Q120, Q180.

Keywords: desert locust swarms, agricultural shocks, local markets, child health.

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Introduction

Climate change is receiving considerable attention both from the media and the academic community. One of its consequences is an increase in the likelihood and intensity of extreme weather conditions, which in turn can lead to severe agricultural shocks. Disruptions in agricultural production often lead to detrimental effects for many households in developing countries, with serious repercussions for young children. This amplifies the negative impact of such shocks since conditions experienced early in life have long-lasting effects on various socioeconomic outcomes. In particular, it is well established that harsh conditions experienced in utero can have detrimental and persistent effects on health throughout the whole life cycle (Lavy et al., 2020; Almond and Currie, 2011; Maccini and Yang, 2009; Stein et al., 1975). This concept, known as the fetal origins hypothesis, implies that it is harder to remedy bad fetal health later on in life.[1]

In this paper, we study the impact, on child health, of exposure to a specific type of agricultural shock that is indirectly linked to extreme weather conditions and climate change: the damage caused by desert locust plagues.[2] Locust swarm invasions are destructive events that recurrently put food supply in many developing countries at risk (Brader et al., 2006). They are caused by a specific species of grasshoppers that usually live, in their solitary phase, around the Sahara desert. Under favorable breeding conditions (excess rainfall), these grasshoppers go through a gregarization process with substantial changes in their behavior, morphology, and physiology. They become more voracious and can grow into huge swarms that travel to less arid

[1] On the one hand, Almond and Currie (2011) provide an excellent review of the fetal hypothesis theory and its theoretical relation to later life outcomes. On the other hand, the empirical literature has successfully documented the impact of adverse in utero conditions on infant mortality (Kudamatsu et al., 2016; Dagnelie et al., 2018), child health (Koppensteiner and Manacorda, 2016; Akresh et al., 2011; Bundervoet et al., 2009), adult health (Akresh et al., 2012; Maccini and Yang, 2009), and adult socioeconomic outcomes such as literacy, educational attainment, income, and labor market status among others (Almond et al., 2007; Alderman et al., 2006; Lavy et al., 2020).

[2] The direct consequence of extreme weather conditions and climate change on agricultural production is crop failure due to droughts or floods.

areas to feed and reproduce causing devastating effects on agricultural production.

We study how early-life exposure to the most recent plague that occurred in Mali, between July and October 2004, led to detrimental effects on child health. While the impact of early life shocks on child health has been explored extensively in the literature, uncovering the channels through which such shocks affect children is still a standing and relevant question. We make a step in this direction, arguing that agricultural shocks due to pest invasion, such as locust plagues, can affect households living in areas with a single harvest per year through two main channels. The first channel is a *speculative/anticipatory effect* that kicks in immediately during the growing season in which the plague is occurring, in anticipation of the upcoming harvest failure. At this point, households and markets are still relying on the harvest from the previous agricultural season. Yet, crop destruction by the pest in the ongoing season could lead to an anticipation of a future crop production decline. This "bad news" may affect the supply and demand of crops on the markets before the actual shock.[3] The second channel is the actual *crop failure effect* that would constitute an income shock for farmers and a supply shock for markets after the harvest of affected crops. This effect should last at least until the following harvest.[4]

The key contribution of our paper is that we are able to explore in detail these mechanisms, by making use of a minutely identified agricultural shock. Specifically, we rely on the exact timing and the precise location of locust swarm events to identify the temporal and spatial variation in the exposure of different cohorts of children to the 2004 locust plague in Mali. We also link this data to local agricultural crop price data from the Malian Agricultural Market Observatory (Observatoire du Marche Agricole - OMA) to tease out potential local price effects.

[3]Part of the *speculative/anticipatory* effect could drive up prices in local markets if the supply is more elastic to the "bad news" compared to the demand for crops. This price effect is in line with the pricing theory of seasonally produced commodities with storage by forward–looking agents and news about future production (Osborne, 2004; Chambers and Bailey, 1996; Deaton and Laroque, 1992; Williams et al., 1991).

[4]Locust plagues can also have a persistent effect if, for instance, the failed harvest in a given agricultural season affects the quality of seeds used for the next one. We found no evidence of such a long–term effect.

We use geocoded household survey data with detailed information on the birth history and health outcomes of children from the Demographic and Health Surveys (DHS). Data on the timing and location of locust swarm invasions comes from the locust monitoring system run by the Food and Agriculture Organization (FAO) Desert Locust Information Service (DLIS).

Using a Difference-in-Differences identification strategy, we first show that children exposed in utero to the adverse effects of the locust plague suffer major health setbacks. They have, on average, a height-for-age Z-score 0.42 points lower than non-exposed children. This represents around 30% of the average height-for-age Z-score. Our estimates suggest an increase in the average stunting rate by more than 20%. We find no impact on cohorts of children exposed to the shock after birth.

We then study the timing of the estimated effect by the quarter of birth. Our results show that cohorts of children that were subject only to the *speculative/anticipatory effect* in utero suffer as much as those exposed to the actual *crop failure effect*.[5] Using data on local crop prices, we show that the plague led to significant inflation of crop prices (average increase of 8%) in affected areas, compared to non-affected areas, during the growing season of 2004.[6] We take this as empirical evidence of speculative price effect in treated markets: during that period, local markets were still relying on the previous harvest that has not been affected by the plague. Therefore, we infer that the estimated price inflation is caused by the expectations of agents about a potential failure of the upcoming harvest. The magnitude of this price inflation is consistent with the impact of market interventions on crop prices. Gross et al. (2020) show that the random scaling–up of a food security program that buys grain from outside sources and sells it locally in poor and

[5]We rule out the possibility that this effect is driven by exposure to the *crop failure effect* after birth.

[6]The agricultural season in Mali happens every year according to the following calendar: planting of seeds happens in May-June followed by the growing season from July to September and the harvest in October/November.

isolated areas of Burkina Faso decreased crop prices by 6 % on average. The magnitude of our price inflation is also consistent with the impact of other agricultural shocks such as droughts on crop prices. Kudamatsu et al. (2016) shows for instance that a severe lack of growing-season rainfall raises staple crop prices by 7 to 10% in Sub-Saharan Africa.

Importantly, we argue that the health effects of the exposure (in utero) to the speculative/anticipatory effect go beyond the local crop price inflation effect for two reasons. First, it includes any precautionary consumption smoothing effect: a decrease in the demand for crops (consumption) driven by households smoothing the impact of the anticipated consumption shock over time. Second, it also captures the impact of in utero exposure to the stress/anxiety that the news of an imminent shock (harvest failure in our case) can bring to households (Torche, 2011; Talge et al., 2007; Tapsoba, 2020). We are not able to tease out these channels with our data. However, the price data does allow us to provide suggestive evidence on the importance of the price inflation channel. Indeed, we show that, as expected, the treatment effect during the speculative/anticipatory effect is only partly absorbed when we account for the price variation.

The extent to which local markets are isolated from other sources of agricultural supply also plays a crucial role in this context. In particular, we find that exposed children born in isolated areas, with limited access to crops from other areas, suffer more compared to those born in well-connected areas. This pattern is driven by the *crop failure effect*. We found no differential effect of treatment by the level of isolation of local markets for children exposed only to the *speculative/ anticipatory effect*.[7]

Our results are robust to specifications that include region-specific time trends, household and mother characteristics, climate shocks, and mother fixed effects. We also argue that they

[7]This could be because economic agents (intermediaries and/or consumers) may have over-predicted the impact or magnitude of the shock during the growing season, at least in non-isolated areas.

are less likely to be biased by potential migration, or pre-existing differences in trends between treated and non-treated areas. Results are also robust to restricting the analysis to male-female and rural-urban sub-samples.

The findings of this paper have relevant policy implications. In particular, we provide evidence of the existence of a strong *speculative/anticipatory effect* that operates differently than the actual *crop failure effect* when agricultural shocks such as locust plagues occur. This calls therefore for different types of policy reactions. Fighting the speculative behavior of intermediaries is crucial during the growing season when the overall crop supply on markets is at its lowest level. Conversely, after the harvest period, policy action should focus on coping with the local crop failure shocks. Our findings also suggest that easy and diversified access to agricultural production from non-affected areas can effectively mitigate this effect.

Related Literature

This paper belongs to three main strands of the economic literature. First, it contributes to the literature on the importance of early–life conditions (Lavy et al., 2020; Maluccio et al., 2009; Black et al., 2007; Behrman and Rosenzweig, 2004; Stein et al., 1975). A substantial part of it focused on identifying the effect on child health of exposure to weather shocks (Maccini and Yang, 2009), violence and civil wars (Tapsoba, 2020; Dagnelie et al., 2018; Quintana-Domeque and Ródenas-Serrano, 2017; Koppensteiner and Manacorda, 2016; Valente, 2015) or adverse institutional setup (Kudamatsu, 2012). We complement this literature by investigating the impact of desert locust plagues on child health. One of the novelties of our analysis relies on the use of a shock characterized by a clear-cut spatial and temporal variation, i.e. geolocated locust swarm invasions. Moreover, this is the first paper, to the best of our knowledge, to shed light on the channels through which such pest invasion can affect households. In particular, we provide evidence of a clear distinction between the purely speculative/anticipatory effect and the actual crop failure effect of the exposure to the swarm invasions. We also show that this shock has long-lasting health effects on children exposed in utero, but no effects after birth. Through several alternative specifications of our model, we show that the adverse effects are present for both male and female children. This contrasts with recent evidence in the literature on early life development, which generally finds larger adverse effects on females (Dagnelie et al., 2018; Lavy et al., 2020; Akbulut-Yuksel, 2017).

Second, this paper is also related to the literature on the effects of news about future production of seasonally produced commodities on competitive storage behavior and prices (Osborne, 2004; Chambers and Bailey, 1996; Deaton and Laroque, 1992; Williams et al., 1991). We borrow our theoretical framework from this literature. Deaton and Laroque (1992) present a supply and demand model for commodities with competitive speculators who hold inventories in the expectation of making extra profits when selling in the future. Osborne (2004) uses a structural model to explain dramatic seasonal price swings and a high degree of serial correlation in commodity price data. They show that the fact that markets incorporate news about future production lowers variation in prices without substantially increasing the mean price. We contribute to this literature by showing how news about a one–time shock to future production can affect current consumption, storage behavior, and prices. Moreover, irrespective of efficiency concerns in the market behavior, this type of anticipation may still affect time–sensitive investments and lead to long–term damages. In our case, this effect comes from the fact that in utero conditions get worse for an entire cohort of children that would otherwise be exposed to the shock only after birth and hence not suffer any detectable effect in the medium/long run.

Finally, this paper adds to the literature that studies the consequences of negative agricultural shocks caused by pest invasions. Baker et al. (2020) show that the boll weevil's pest invasion that affected US cotton production from 1892 to 1922 led to an increase in educational attainment due to reduced opportunity cost for schooling. Banerjee et al. (2010) show that the phylloxera invasion in 19th century France affected wine production and led to substantial effects on adult height for people born in affected areas in that period. De Vreyer et al. (2014) used locust plague invasion in 1987-1989 in Mali to show that it had a long term effect on educational attainment.[8] We contribute to this literature by providing evidence on the channels through which pest invasion may affect household welfare in developing countries. This paper is the first one to document evidence of a strong speculative/anticipatory effect besides the crop failure effect in this type of shock.

The remainder of this paper is organized as follows. Section 1 provides some background on the 2003-05 locust plague in Western Africa and its relation to food shortages and child health in Mali. Section 2 presents the data used for the empirical analysis. Section 3 discusses the conceptual framework and the channels through which locust invasions can affect the well-being of households. Section 4 presents the empirical strategy used and Section 5 shows the results obtained. Section 6 concludes.

1 Background

In this section, we describe the context of our analysis. We first provide a general description of desert locusts, their habitat, and their link to climatic conditions that can turn them into agricultural plagues. We then give some details of the locust plague that took place in the Sahel region in the mid–2000s, stressing its consequences in Mali.

[8] In an independent paper, Linnros (2017) estimates the aggregate effect of locust infestations on child health using data from Senegal, Burkina Faso, Mali, and Niger.

1.1 Desert Locust Plagues

Desert locusts are insects that live in the arid and hyper-arid zones of the Sahel region, northern Africa, Middle East, and Southeast Asia, as shown in Figure 1. They pertain to the family of *grasshoppers* and normally inhabit desert zones, called *recession areas* (bounded by the black solid line in Figure 1), in a solitary, harmless and integrated way with the local ecology. What makes them different from traditional grasshoppers is their capacity to mutate physically and change behavior under certain conditions. In particular, if specific areas called *breeding areas* (green and ocher areas in Figure 1) experience periods of excessive rainfall, followed by periods of relatively mild temperatures, a process of fast reproduction takes place. The high density of locusts combined with a relative shortage of vegetation induce them to a *gregarious stage*: the locusts mutate physically and start behaving as a unique group, known as a *swarm*.

Upon gregarization, these groups become more and more voracious and reproduce faster. If needed, locust swarms will fly away to find a location with vegetation for feeding and appropriate conditions for reproducing. Within a few weeks after settling in a suitable new location, there can be a new generation of gregarious locusts. The increase in the size of the swarm can be remarkable: the incorporation of locusts from the new generation can lead up to a ten-fold increase in the size of the swarm (FAO, 2004). If breeding conditions are favorable and there is no human intervention, the density of a swarm can become extremely high, exceeding a billion insects.

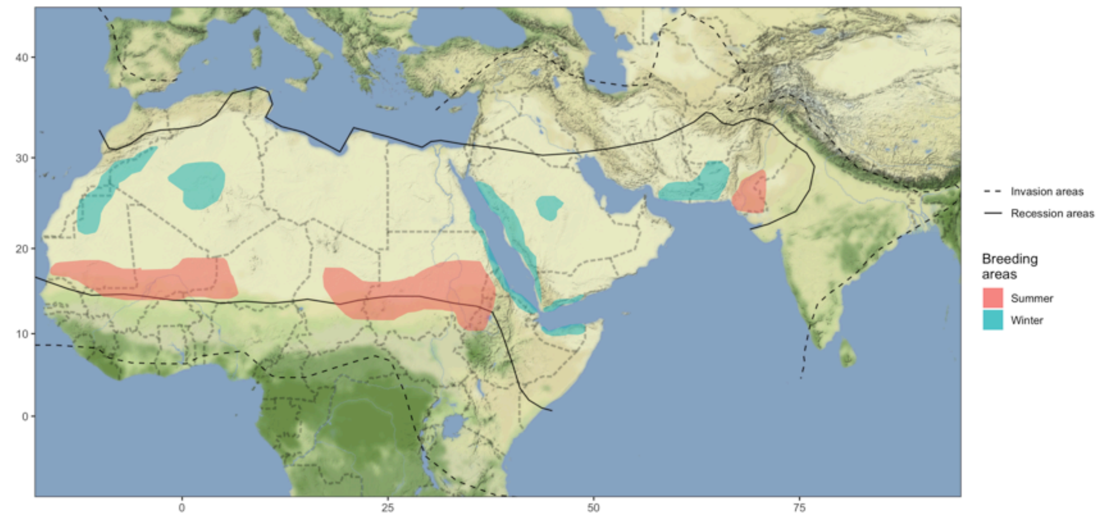
Desert locusts swarms are very threatening because, while flying in search of new locations for feeding and reproducing, they end up following winds that move them away from the desert areas of the Sahara. Usually, the winds blow them towards the central Sahelian and tropical areas in the South or the Mediterranean regions in the North. These *invasion areas*, shown in Figure 1 by the dashed red line, span over more than 50 countries and have a total surface of about 29 million square km (Herok et al., 1995). These zones are more densely populated and are used for agricultural production. Thus, crop yields and/or pasture vegetation can be partially or totally consumed by the swarms, potentially threatening the food supply of entire regions.[9] Moreover, the fact that the swarms can fly for very long distances, over hundreds of kilometers in a single day, implies that favorable conditions for gregarization in one place can create severe repercussions in relatively far away locations.

If climate conditions remain favorable for a long time within large geographic areas, the swarming, breeding, and migration behavior of locusts can create a regional plague, i.e. when locust swarms multiply exponentially in size and number and spread over several countries. If so, food security in multiple countries can be at risk. Moreover, fighting the plague in these circumstances is extremely difficult and costly.

Fortunately, such events are not very recurrent nowadays – as seen in Figure 2, there have been relatively fewer regional plagues since 1970 compared to the previous period. This reduction is associated with active human preventive interventions (Cressman and Stefanski, 2016). The plagues that managed to take place since 1970, however, happened to be extremely severe. This is the case of the plague that occurred between 2003 and 2005, whose consequences we analyze in this paper.

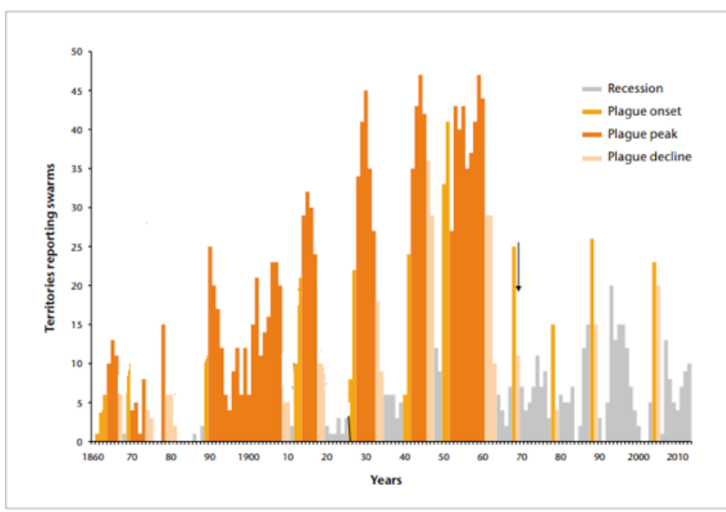
[9] A locust swarm can cover between less than one to several hundreds of square km, and each square km of the swarm has at least 40 million locusts. Each square km of a swarm can consume daily the equivalent food consumption of 35,000 people (Symmons et al., 2001).

Figure 1: Desert Locust Breeding and Invasion Areas



Note: Authors' calculation based on Cressman and Stefanski (2016)

Figure 2: History of Locust Plagues



Source: Cressman and Stefanski (2016)

1.2 The 2003-2005 Desert Locust Plague

The 2003-2005 desert locust plague started from optimal climate conditions in late 2003 which led to a massive plague throughout entire Western Africa in 2004 (Ceccato et al., 2007), peaking in the third quarter of that year. Importantly, it developed from independent outbreaks that took place in different locations, taking the international prevention community by surprise (Cressman and Stefanski, 2016). It affected multiple countries, mostly in West Africa and North Africa, but also in central and eastern territories.

The 2003-2005 locust plague created a regional food crisis that involved more than eight million people in the whole Sahel region, implying a huge cost. On top of its direct cost (that includes the agricultural lands and the crops damaged), of which there is not a precise estimate, there was a substantial collateral cost of more than 400 million dollars needed to control the invasion (Brader et al., 2006). This loss does not include the indirect cost that the plague entailed, such as forgone investment for other national development activities or significant increases in food prices (Brader et al., 2006; FAO, 2005a).

1.3 The Case of Mali

Mali is a landlocked country in West Africa with a surface of about 1.2 million square km, spread in latitude across three different climate zones: the desert zone (Saharan), the transition zone (Sahel), and the tropical zone. As Figure 1 depicts, a substantial part of its territory is a locust breeding area, which makes it very likely to experience (or be invaded by) locust swarm outbreaks.

This country is a relevant case study for our analysis for several reasons: first, as mentioned above, Mali is located in a very risky area in terms of the likelihood of experiencing locust plague invasions. Second, the pattern of the 2003-2005 swarm invasions was unevenly spread over its territory, allowing us to exploit this spatial variation together with variation in the timing of the plague in our empirical exercise. Third, this is a poor country that relies mostly on agriculture.[10]

[10]Other countries with similar characteristics to Mali could not be investigated in this study because of data limitations.

More specifically, Mali is classified as a low-income country, with a GDP per capita ranked 160^{th}, with more than 40% of its population below the poverty line. Malian population, of approximately 19 million individuals, is largely present in rural areas (about 60%). Mali is strongly dependent on agriculture, which accounts for about 40% of its GDP (in 2004 this share was 30%).[11] Its agricultural sector is largely composed of small-scale subsistence farming. As such, a large share of its population is vulnerable to agricultural hazards such as droughts and plagues.

Not surprisingly, Mali was among the countries that have been most damaged by the locust plague of 2003-2005. According to Brader et al. (2006), 1 million people were affected by this plague. Exact estimates of the agricultural losses are not available, but the existing evidence suggests that the plague had devastating effects. Brader et al. (2006) states that in Mali, the losses caused by the 2003-2005 plague have been evaluated at 90% of the expected cereal production in the affected areas. On top of this, around one-third of the pasture was also lost. By mid-2005, FAO (2005b) reported alarming increases in cereal prices and a deterioration of conditions for livestock production in the region. The poorest households suffered the most from this situation. The report states that by mid–2005, the "*access to main food staples [was] increasingly difficult for vulnerable households and pastoralists. Severe child malnutrition [was] increasing rapidly.*" (FAO, 2005b).

2 Data and Descriptive Statistics

In order to study how exposure to locust invasions in Mali impacted child health and the mechanisms behind it, we collect data from four different sources: (i) information on locust swarms are

[11] This information is obtained from the World Bank Development Indicators.

obtained from a rich database of worldwide locust monitoring, (ii) data on children anthropometrics is gathered from DHS, a widely used household survey, (iii) geographically disaggregated crop price data provided by the Malian Agricultural Markets' authority, finally (iv) climate data and other geographical characteristics at different levels of geographical disaggregation and sources complement these data.

2.1 Locust Swarm Data

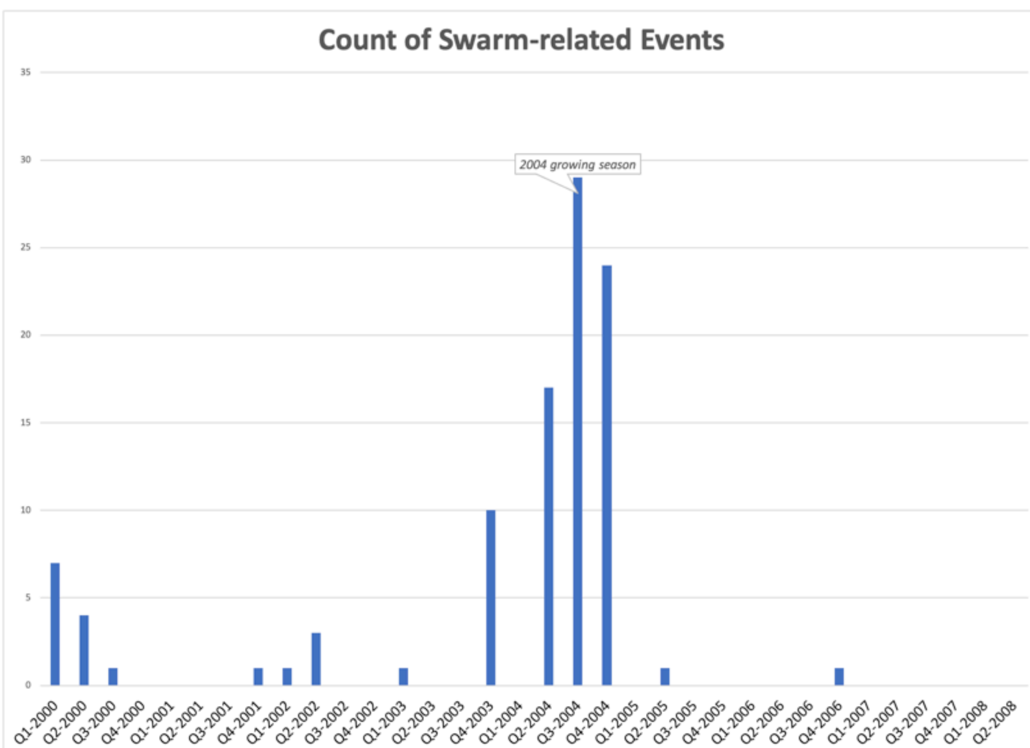
We collect geographical and temporal incidence of locust swarms from the SWARMS database (Cressman, 1997). SWARMS contains historical geocoded information on many "locust parameters" including local breeding conditions, the incidence of adult locusts, hopper bands, swarms, and many others. For this application, we select information exclusively on locust swarms. Specifically, for each event related to the presence of a locust swarm, we collect the date and the geographic coordinates in which the event occurred.

The SWARMS system is held and maintained by the FAO Desert Locust Information Service (DLIS) Unit. This unit is monitoring, preventing, and controlling locust incidence in Sub Saharan Africa for over 60 years. For this, a national office in each Sub Saharan country conducts field activities. These activities include field incursions into areas prone to locust incidence and reproduction to search for (and code if found) locust bands and swarms. The coding is done in-field with a satellite-based technology; the information is automatically sent to the FAO-DLIS headquarters to be further cleaned if necessary. The data is complemented with information from local villages which self-report to field officers and/or the national DLIS office. The resulting data on the incidence of locust swarms spans from 1985 to 2016.

Figure 3 shows the distribution of locust swarm incidence between 2000 and 2008 in Mali. Within this time window, most of the locust swarm–related events occurred during the second half of 2004 and picked during the third quarter of this year. As discussed in detail in Section 3, this period coincides with the growing season. Conversely, nothing happened during the growing season of 2003.[12] This means that, in Mali, the plague we study damaged the harvest of a single agricultural campaign, i.e. the one of 2004. In our main specification, we use all swarm events that happened in 2004.[13] Our results are robust to considering only those that occurred during the growing season of 2004.

Potential measurement error issues in the locust data are attenuated by the fact that during our period of study, the National Desert Locust Office in Mali has not faced any drastic changes or other difficulties that could affect its field operations.[14] The current political instability and terrorist threats in Mali started in 2012, long after our period of study.

Figure 3: Quarterly Number of Swarm Events in Mali



Source: SWARMS data base from FAO Desert Locust Information Service.

[12]The locust started swarming in arid areas of the North after the end of the harvest period of 2003.

[13]Those events started in June 2004 mostly in arid and desert areas in the North.

[14]This information was confirmed unofficially by an Information Officer at the DLIS office at FAO, Rome.

2.2 Children Anthropometrics Data

Information on child health in Mali is obtained from the Demographic and Health (DHS) Surveys. DHS surveys are being conducted in many developing countries since the late 1990s with a standardized and nationally representative data collection methodology. The interviews target female individuals, aged from 15 to 49 years old. For a subsample of respondents' children (less than) 5 years old, anthropometric data such as height and weight are collected by trained surveyors. Importantly, children's height is provided in Z-scores, i.e. the difference between the child's height and the mean height of the same-aged international reference population, adjusted by the standard deviation of the reference population.[15]

Moreover, DHS surveys provide GPS coordinates of where each household cluster is located.[16] These coordinates are published with a random noise for privacy reasons (set to up to 2 km for clusters located in urban areas, 5 km for those in rural areas, and 10 km for 1% of the latter). The locations of the clusters are drawn independently – thus differ – in each DHS wave. In order to have a geographical unit that is comparable across waves, we also collect the coordinates of the 0.5 x 0.5 decimal degrees PRIO-GRID cell (see Section 2.4) in which each enumeration area lays over.

In this analysis, we use wave V and wave VI of the DHS survey, whose interviews took place in 2006 and 2012/13 respectively. Therefore, children who were measured during the interviews cover all birth cohorts ranging from 2001 to 2013, which includes the plague period in the middle. In particular, children measured in the first wave are those whose cohort of birth can potentially be exposed to the plague early in life. Children from the second wave, instead, are too young

[15]Unfortunately data on birth weight is of poor quality in the DHS surveys that we are using. Birth weight is reported for less than 30% of observations with a lot of bunching due to the fact that most respondents provide rough guesses from what they could recall.

[16]Empirical work using DHS data also call the clusters enumeration areas or locations. We use the terminology of DHS cluster or enumeration area without distinction.

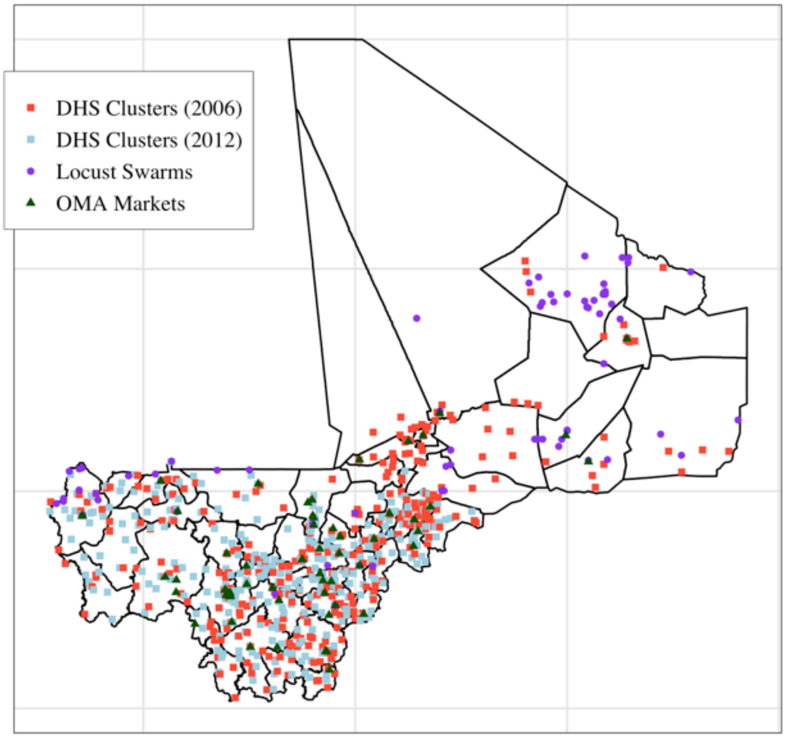
to have been exposed to it. In our main specification, we use the information on children born before the 2006 DHS data collection period. Data from the 2012 wave are used for robustness and placebo tests. To enrich the set of variables available, the data of each child is linked to other household characteristics.

To have a broad picture of the socio–economic conditions of our population of interest, Table A1 provides some descriptive statistics of the children, mothers, households, and clusters characteristics. Children are on average 2 years old, and the gender composition of the sample is quite balanced. Mothers are on average poorly educated, and about 1,6 meters tall. Households are on average fairly large (about 7 members) and have a male individual as their head/chief (92 percent). The households are mostly in clusters located in rural (65 percent) and isolated areas (about 3,5 hours from the nearest large town, on average). Moreover, in Table A2 we compare treated and non–treated households and find no remarkable differences in terms of family/household characteristics.[17] The distributions of mother characteristics, as well as of the age of household head, rural/urban, and wealth are fairly even across the two groups.

Figure 4 depicts the spatial distribution of household clusters and locust swarm events in Mali. The potentially treated clusters are mainly located in the eastern part of the country, where most of the swarm events occurred. The remaining potentially treated clusters are located in the western and central regions of the country. The affected areas are closer to the breeding area depicted in Figure 1.

[17] A household is defined as exposed if located within 30 km from at least one locust swarm event during the plague. Moreover, the source of some of the characteristics discussed is explained in Section 2.4.

Figure 4: DHS Clusters, Locust Swarms and Local Markets in Mali



Source: DHS waves of Mali from 2006 and 2012, SWARMS–FAO database, and OMA market prices' data, respectively.

2.3 Crop Price Data

We collect official data on crop prices from the Malian Agricultural Markets' authority, Observatoire du Marché Agricole (OMA prices henceforth). The data provides the monthly retail consumer prices, in national currency, of the 4 cereal crops: maize, millet, sorghum and, rice (the latter is available for 4 different sub-varieties). The data covers 74 local markets in Mali from 2000 to 2015. These markets are depicted in Figure 4 (green triangles). We link the market data to households by looking for the nearest OMA market located in the same PRIO-GRID cell where the DHS clusters lay on, and analogously to the weather data (SPEI index).

Table A3 documents the descriptive statistics of the OMA data. It contains more than 50 thousand data points. On average, a market–crop series covers about 9 years of time. About 24 percent of the markets are located in treated locations and are located on average 1.6 hours from a large town.

2.4 Additional Spatial Data

The georeferenced feature of the household and locust swarms datasets allows us to aggregate more information for our analysis. We add to the data described above geographical information from the PRIO-GRID dataset (Tollefsen et al., 2012). This is a geocoded dataset, whose statistical unit is spatial cells of 0.5×0.5 degrees, hereafter called PRIO-GRID cells. Moreover, we collect weather data from the Standardized Precipitation-Evapotranspiration Index (SPEI), a multiscalar drought index that considers the joint effects of precipitation, potential evaporation, and temperature (Vicente-Serrano et al., 2010). This allows us to take into account the fact that the impact of rainfall on the growing cycle of a plant depends on the extent to which water can be retained by the soil.[18] This dataset is available at the 0.5×0.5 degrees, and so matched to the household coordinates.

We also collect data at finer levels of disaggregation that are linked to the DHS coordinates. We obtain data on access to transportation networks and, as a consequence, access to large cities and markets, from the Global Accessibility Map (GAM; Uchida and Nelson, 2010) project. This is a high–resolution raster data which provides information on the travel time to the nearest 50,000 inhabitants town in 1km^2 geographical units, in the year of 2000.

3 Impact of Locust Invasions: Potential Channels

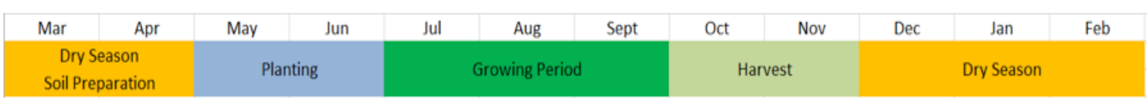
In this paper, we are interested in understanding how early–life exposure to the 2004 desert locust plague could have affected child health using data from Mali. The relation between the timing of the exposure to the plague and food shortage is, however, not straightforward. Agricultural

[18] See Appendix A.2 for a description of how we construct an index of local weather shocks using SPEI index.

production has a cyclical nature, following a specific seasonal calendar. Crop damages caused by locust swarms in a period might not affect the food supply at that moment if markets are still relying on the previous season's production. Therefore, to understand how the 2004 plague could have affected households in Mali, we first need to understand which harvest households and markets were relying on in each period.

Figure 5 shows the seasonal agricultural calendar in Mali (FEWS-NET, 2005). It starts with soil preparation between March and May followed by planting in May-June. The growing season happens between July and September; the harvest period is in October/November. The 2003–2005 locust plague infested parts of Mali mostly during the third quarter of 2004 (see Figure 3). This means that it has affected the planting and the growing season of the 2004 agricultural season, scheduled to be harvested in October of that year. Therefore, food supply on local markets before October 2004 came from the harvest of the previous year, which, as shown in Figure 3, has not been affected by the plague. Given this, we identify three potential channels through which locust invasion could have affected households: (i) a speculative/anticipatory effect, (ii) a local crop failure effect, and (iii) a potential long–term effect on agricultural production.

Figure 5: Seasonal Calendar



Source: FEWS-NET (2005)

3.1 Speculative/Anticipatory Effect

Between July and October 2004, i.e., during the peak of the plague, households, and markets were still relying on agricultural production from the previous season, harvested by the end of 2003. Hence, there was no threat to the food supply in this time window. However, there could have been a speculative/anticipatory effect in expectation of the imminent harvest failure in locust–affected areas. This could affect households in two different ways. First, through the supply and demand elasticity to the news about future harvest shock. Second through the potential stress/anxiety that such news can induce on vulnerable household members such as pregnant women.

Supply and demand elasticity to the news about future harvest shock: The mere exposure to the plague could cause severe inflation of crop prices and harm households with limited resources, especially because the plague happened during the lean season.[19] The conceptual framework to understand this price effect comes from the pricing theory of seasonally produced commodities with storage by forward–looking agents and news about future production (Osborne, 2004; Chambers and Bailey, 1996; Deaton and Laroque, 1992; Williams et al., 1991). The bad news about future harvest in a spot market (no futures) with intermediaries can lead to a decrease in the supply of crops on the market at time t if intermediaries withhold inventories for future sales in expectation of higher profits. It can also affect demand at time t if households try to smooth the impact of the shock over time by anticipation. If the supply is more elastic to the news, this will lead to an increase in crop prices at time t.[20] In the present context, this effect is exacerbated by the fact that African farmers often sell their crops at a low price after harvest and buy them for consumption at a high price during the lean season (the growing season before the next harvest). These farmers fail to exploit optimally such regular price variation because of credit market imperfections (Burke et al., 2019) and non–monetary storage cost (Gross et al., 2020).

This is a pure speculative/precautionary effect since the actual quantities of grains available

[19] The lean season is a period of seasonal poverty in rural areas between planting and harvest periods when stocks of food are the lowest, there is no income and families often miss meals.

[20] If supply and demand are equally elastic to the news shock, there will be no price effect but the equilibrium quantities will still decrease and this could still affect the well-being of households. The case in which the elasticity of the demand would be higher is not realistic in settings with imperfect competition and important market power for intermediaries such as those in Sub-Saharan Africa (Bergquist and Dinerstein, 2020).

(including those in storage) remain the same. In Section 5.5.1 we document the impact of this speculative effect on crop prices using monthly market–level crop price data. Specifically, we show that the plague caused significant inflation of crop prices in treated areas during the growing season of 2004 (before the affected harvest). However, the speculative effect goes beyond this price inflation effect since equilibrium crop quantities consumed by households can drop without a price increase (as argued earlier).

Stress/anxiety due to the news: The news about future harvest failure could also create a stressful environment in budget-constrained households. This may have substantial effects for children in utero through maternal stress. The literature has shown indeed that maternal exposure to stressful events such as earthquakes leads to worse birth outcomes (Torche, 2011; Talge et al., 2007). Therefore, the children that were in utero during the third quarter of 2004 were potentially affected by this speculative/anticipatory effect even though markets and households were still relying on harvests not affected by the plague. Consequently, cohorts belonging to the third quarter of 2004 were exposed only to the speculative/anticipatory effect in utero. We use this information to disentangle the impact of the speculative/anticipatory effect, on child health, from other potential effects described below.

3.2 Local Crop Failure Effect

The harvest that was directly affected by the locust swarms was gathered between October and November 2004. Households and local markets had to rely on this potentially bad harvest until the next one, gathered in October 2005. We can, therefore, expect that areas affected by locust invasions have been treated during all this period by the actual local crop supply shock. This agricultural production shock could have negatively affected all households that are net buyers of local crops through the increase in market prices. It could have also harmed farming households that had their fields invaded by the swarms (a direct income shock).[21]

Therefore, in terms of exposure of children, only the cohorts from the third quarter of 2005 onward were exposed exclusively to the crop failure effect. Children born between the fourth quarter of 2004 and the second quarter of 2005 were potentially exposed to both effects, as their 9-month pregnancy period overlaps with the third quarter of 2004, i.e., what we define as the speculative/anticipatory effect period. We will exploit such distinction when separating the latter from the crop failure effect.

3.3 Potential Long Term Effect on Agricultural Production

The adverse effects of the plague should have disappeared after the harvest of November 2005, unless the crop failure in 2004 affected the quality of seeds used for the next harvest, or the income shock led to a depletion of productive assets (livestock, for instance). This would have led to a persistent effect, in the medium run, on household food availability. We found no evidence suggesting that subsequent harvests suffered from the locust swarm invasions. First, there is no account of such effects from reports produced by agencies like FAO. Moreover, we will show empirically in the next sections that there are no differences in health status between children conceived in the locust affected areas and children conceived in non-affected areas, after the harvest of 2005.

4 Empirical Strategy

We adopt a Difference-in-Differences approach to estimate the causal impact of locust invasions on child health. First, we define treatment and control groups at the enumeration area level by

[21]Locust swarms can also affect livestock farmers through a decrease in the available pasture.

setting as treated the clusters within 30 kilometers of a locust swarm event. Then, we exploit all the available variation in childhood exposure to treatment across enumeration areas and birth cohorts.

We estimate the following model:

$$y_{i(h,t,e)} = \gamma T_{i(t,e)} + \mu_e + \beta_t + \theta_{hh} X_h + \theta_1 X^{(1)}_{i(h,t,e)} + \theta_2 X^{(2)}_{i(t,e)} + \epsilon_{i(h,t,e)}, \tag{1}$$

where i denotes a child belonging to cohort t (year-month), born in household h who lives in enumeration area e.[22] The dependent variable $y_{i(h,t,e)}$ measures height-for-age Z-score of child i in our main specification. The treatment variable is $T_{i(t,e)}$, a dummy that takes a value of one if child i belongs to an enumeration area in the treatment group and has been exposed in utero to the adverse effects of the plague, i.e. born between July 2004 and June 2006 in a locust affected area.[23]

We control for μ_e and β_t that are enumeration area and birth cohort fixed effects respectively. $X^{(1)}_{i(h,t,e)}$ and $X^{(2)}_{i(t,e)}$ are observable characteristics at individual level (gender, birth order among siblings, age gap with direct older and younger siblings, etc.) and enumeration area level (Standardized Precipitation-Evapotranspiration Index (SPEI) drought index), respectively. Finally, X_h is a vector that includes household–level controls, such as gender and age of household head, wealth index of household, education of the mother. Standard errors are clustered at 0.5 x 0.5 decimal degrees PRIO-GRID cell level (see section 2.2).

In the baseline specification of the model in equation 1, we control only for enumeration area

[22]We omit from the notation, for neatness, the grid-cell g where enumeration area e lays over (see section 2.2). When estimating the model (1) with the two DHS waves, we replace the cluster fixed effects by grid cell fixed effects in order to compare geographical units consistent across waves. In that case, we add to (1) a dummy for treatment status at the cluster e level.

[23]This treatment dummy will be split below into different time bins to properly explore the timing and channels of this adverse shock.

fixed effects to account for permanent unobserved characteristics of the place of residence and cohort fixed effects to account for cohort–specific shocks, whereas the full version of the model includes controls for the relevant household and child characteristics.

Our coefficient of interest is γ, which measures the average difference in changes of height-for-age Z-scores of children born in locust–infested areas and children born in non-infested areas, holding constant all the other relevant characteristics. The implicit assumption behind this identification strategy is that, after controlling for cohort and enumeration area fixed effects, household characteristics, and other relevant exogenous covariates, changes in height-for-age Z-scores would be similar across locust infested areas and non-infested areas in absence of the plague. Given that we control for cohort and enumeration area fixed effects, the coefficient γ does not represent the national impact of locust plague but the average effect with respect to local and cohort averages.

5 Empirical Results

5.1 Average Impact of Locust Plague on Child Health

Table 1 displays the point estimates of γ in Equation 1. Column (1) shows the estimated coefficient of the baseline specification of our model, in which we control only for location fixed effects and cohort fixed effects. As explained in Section 4, the treatment variable is a dummy equal to one for children born in locust affected areas between July 2004 (beginning of locust plague) and June 2006, i.e. the last cohort of children that relied on the 2004-2005 agricultural campaign harvest while in utero.[24] The estimated impact is negative and statistically significant. The estimate is robust and remains significant after progressively adding controls for child

[24]The youngest children used in this regression were born in March 2006.

characteristics in column (2) and family characteristics in column (3). Our main specification in column (3) suggests that exposed children have, on average, a Z-score 0.42 points smaller than non-exposed children. This represents approximately a 30% decrease in the average Z-score for the children in our sample. Column (5) shows an alternative way to quantify the magnitude of this effect. It shows that this plague increased stunting rates by 7.4 percentage points. This represents more than 20% of the average stunting rate in our sample.[25] The estimates of the impact of locust plague on height-for-age z-score and stunting rates are also robust to the inclusion of region-specific time trends (columns (4) and (6)). In Table A6, we also show that this plague invasion did not have any detectable effect on neonatal mortality (death less than a month after birth) and infant mortality (death less than a year after birth).

5.2 Robustness of the Average Effect

To validate the results presented above, we start by confirming that the estimated effect is not sensitive to the radius of 30 km used in our main specification. To do so, we run several alternative specifications of our baseline model described in Equation 1 (Section 4); in each iteration, we change the definition of *treatment group*. In particular, in the first specification, an area is defined as *locust affected* if it is located at most 20 km from at least one swarm event during the 2004 plague. In the subsequent specifications, we add progressively 2 km to the threshold that defines what is a treatment area, up to 70 km. Figure A1 plots the estimates of γ for each performed specification, together with their 95% confidence bands. It can be noticed that the adverse effect of locust invasions on child health is both negative and statistically significant for thresholds up to 40 km. After that, the estimates approach zero and lose significance. This is consistent with

[25] Stunting rate is defined as the share of children with height-for-age Z-score smaller than -2 standard deviations.

Table 1: Impact of Locust Plague on Child Health

Dependent variable	Height-age Z-score				Stunted	
	(1)	(2)	(3)	(4)	(5)	(6)
In utero treatment	-0.476***	-0.432***	-0.421***	-0.429***	0.074**	0.079***
	(0.112)	(0.115)	(0.112)	(0.092)	(0.031)	(0.023)
Observations	9,173	9,173	9,173	9,173	9,173	9,173
R-squared	0.200	0.220	0.239	0.245	0.183	0.189
Cohort FE	YES	YES	YES	YES	YES	YES
Location FE	YES	YES	YES	YES	YES	YES
Child characteristics	NO	YES	YES	YES	YES	YES
Family characteristics	NO	NO	YES	YES	YES	YES
Region specific time trend	NO	NO	NO	YES	NO	YES
Mean dependent variable	-1.425	-1.425	-1.425	-1.425	0.365	0.365

Source: SWARMS data base from FAO Desert Locust Information Service and household survey data from 2006 DHS wave in Mali. Full set of controls includes mother's height, education, gender and age of household head, household wealth index, SPEI index, birth order, time gap between conception and the previous and following pregnancies. Robust standard errors in parentheses are clustered at PRIO cell grid level. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable in columns (1)-(4) is height for age z-score. Column (1) controls only for cohort and location fixed-effects. Column (2) and Column (3) include progressively child characteristics and family characteristics. Column (4) adds to the previous controls region-specific time trends. Columns (5) and (6) use a dummy equal to 1 if child is stunted (height-for-age Z-score<-2) as dependent variable.

the idea that locusts invasions are events that have adverse *local* effects.

In Table A5 we present results of robustness checks performed on alternative empirical specifications and data samples. Column (1) shows robustness to very demanding mother fixed-effects specification where we are only relying on variation across siblings in exposure to the locust plague. The magnitude of the impact does not change substantially and the effect remains significant. Columns (2), (3), (4), and (5) show the robustness of the main results presented in Table 1 when using, respectively, survey sample weights, spatial HAC standard errors, treatment at the municipality level, and including events that occurred beyond Mali country borders. Column (6) runs the main specification using data from both 2006 and 2012/2013 DHS waves and the estimated coefficient is still negative and significant. Column (7) uses both survey waves but restricts the sample to children born after the plague outbreak. This means that treated children belonging to the older cohorts and younger children are not treated.[26] To check that the estimated effect is not capturing any pre-existing time trends between treated and non-treated areas, we run a first placebo test in column (8). We use only data from the 2012/2013 survey and the placebo treatment is a dummy equals to 1 for children born in locust affected areas after 2010. The estimated coefficient is small, positive, and not significant.[27]

5.3 Impact of Locust Plague on Child Health across Space

We have established that the 2004 locust plague in Mali led to major health setbacks for children exposed in utero. In this section, we explore whether and how the average impact on child health changes with the distance from the locust swarm events. To do so, we run an alternative specification of the model presented in column (3) of Table 1, in which we define as non-treated areas located at least 60 km away from any swarm event. We then split the treated areas into 6 groups using 10 km-wide intervals. Therefore, we have 6 treatment groups, the first one includes children belonging to households located at most 10 km away from the locust event, the second group includes children belonging to households located between 11 and 20 km away from the locust event, and so forth.

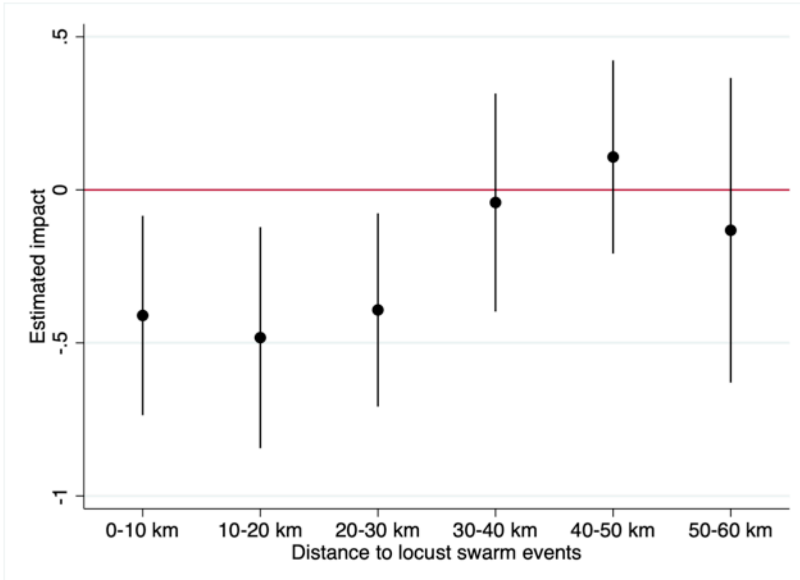
Results shown in Figure 6 suggest that the estimated effect is negative, significant, and fairly constant if the impacted child belongs to a household located within the first 30 km from the locust plague event. This means that the plague affected entire localities by impacting their local markets of reference, and not just those households or small communities that have experienced a bad harvest (or completely lost it) because of the invasion. The role of local markets is a key finding of our paper that we explore further in the next sections. The estimated coefficient fades

[26] In our main specification in Table 1, treated children belong to younger cohorts, and all the older children are not affected in utero by the plague.

[27] We discuss more evidence of absence of pre-existing trends in Section 5.4.

out after 30 km, confirming the idea that locust invasions have local effects.

Figure 6: Impact of Locust Plague on Child Health across Space



Source: SWARMS data base from FAO Desert Locust Information Service and household survey data from 2006 DHS wave in Mali. This figure depicts the estimated coefficient of locust invasion on height-for-age Z-scores splitting the treatment effect in the main specification into average effects by groups of 10 km rings around each location. Non-treated areas are those with no swarm event within a 60 km radius. The first coefficient plots the average effect for affected children with at least 1 swarm event within a 10 km radius. Each coefficient is plotted together with its 95% confidence bands.

5.4 Timing of the Estimated Effect, Channels and Placebo Test

The fact that the 2004 locust invasion impacted Mali within a relatively narrow time window allows us to investigate the effect of such shock for different cohorts of children. Moreover, as discussed in Section 3, for each life-stage, we can differentiate between *speculative/anticipatory effect* and *crop failure effect* as potential channels through which locust plagues affect child health. To do so, we estimate the impact of being born in a locust–affected area for different birth cohorts.

Figure 7 depicts the estimated impact of being born in locust infested areas by the quarter of birth between years 2003 and 2008, together with the 95% confidence bands of each coefficient.[28]

[28]The 2006 DHS survey data was collected between May and December so we restrict the analysis to

The dashed line represents the quarter when the locust plague started in Mali. This figure shows that children born in impacted areas before the plague, i.e., between the first quarter of 2003 and the second quarter of 2004 both included, have height-for-age Z-scores comparable to children belonging to the control group. This means that the children that were already born when the plague hit did not suffer any effect perceptible in their height-for-age Z-scores.[29] It also confirms the hypothesis that the estimated impact that we documented in the previous section is not capturing pre-existing trend differences between treated and non-treated areas.

Moreover, between the third quarter of 2004 and the first quarter of 2006, there is a clear drop in our coefficient of interest, which becomes negative and statistically significant (estimates for quarters four of 2004 and one and two of 2005 are significant at 90% confidence level). This corroborates our finding that all the cohorts of children that have been exposed in utero to the plague suffered substantial health setbacks.

Note that the estimated coefficient turns negative and significant for children born in July, August, or September 2004, i.e. the first three months of the locust invasion in Mali. According to the seasonal calendar reported in Figure 5, these children, during the onset of the plague, were still relying on the previous harvest that has not been affected by locusts. Thus, this evidence suggests that the negative impact on their health status was driven by speculations on the agricultural campaign that was starting. Note that this cohort was exposed also to the crop failure effect, but only after birth, and we find no effect of the plague after birth (see older cohorts of children in Figure 7). Yet, this negative effect could be explained by differences between the very first 2-3 months of life and the subsequent months in early childhood, which, to the best of our knowledge, has never been documented in the literature. Nevertheless, we rule out this

children born before MAY 2006 to prevent survey timing biases from contaminating the estimated effect. The oldest cohorts with anthropometric measures from the 2012-2013 survey were born in 2008.

[29]This justifies also our focus on in utero exposure in most of our analysis.

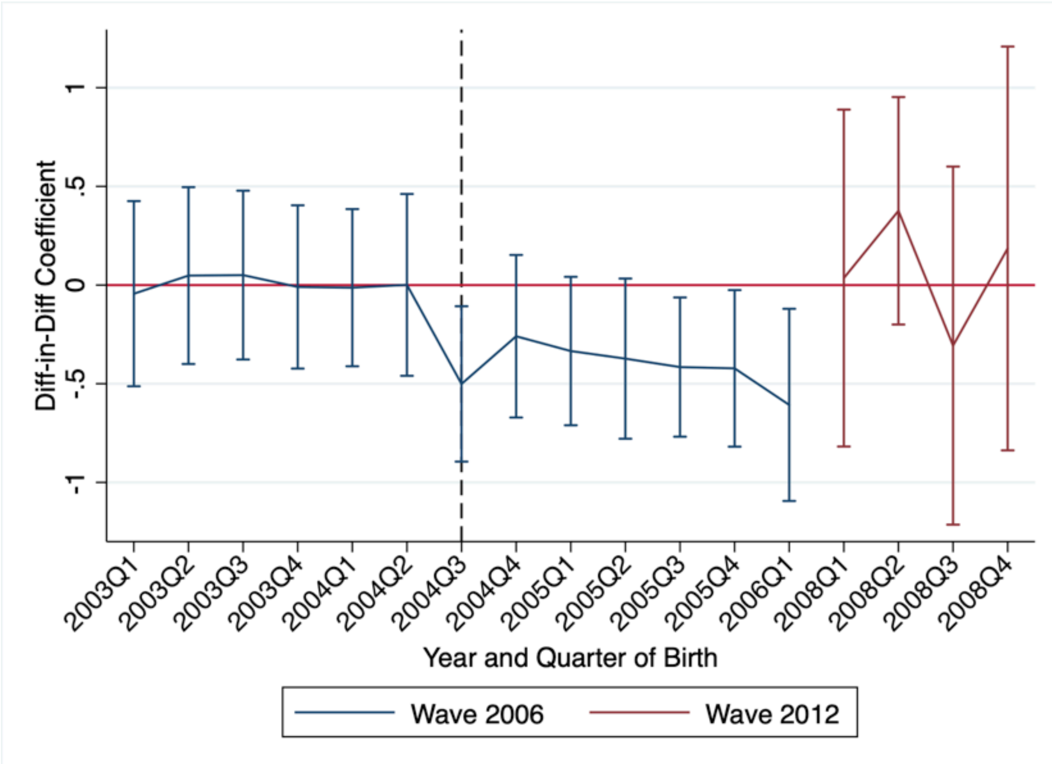
alternative explanation in Section 5.5 by first showing evidence of a significant crop price increase during the third quarter of 2004 due to the plague. Later on, we show that when we control for local prices, the estimated effect for the impacted children born during this time window (i.e., exposed only to the speculative price effect) drops significantly.

Figure 7 shows negative coefficients also for children born between September 2004 and March 2006. These groups were relying on the harvest of the 2004-2005 season, i.e. the harvest that was directly damaged by locust swarms in treated areas. Therefore, these children were potentially subject to the crop failure effect due to the failed harvest while in utero. The negative and significant impact found for these cohorts supports this idea. Children born between October 2004 and June 2005 are potentially exposed in utero to both the *speculative price effect* and the *crop failure effect*. Those born after June 2005 are exposed only to the crop failure effect.

Finally, Figure 7 also depicts the estimated coefficients on younger cohorts, born after the plague ended, namely children born between January and December 2008. Those children were 4 to 5 years old at the time of the survey when they were measured so estimated coefficients are noisier and more volatile from quarter to quarter. In any case, there is no evidence of a clear trend and the estimated impact is oscillating around zero.[30] Given that the health status of children born in the locust affected areas (after the plague ended) is comparable to the health status of children born in non-affected areas, we can infer that the locust invasion did not have any perceptible long-lasting effects. The harvest gathered during the 2005-2006 season seems not to have been impacted by the damages which took place during the previous agricultural season.

[30] We show in column (8) of Table A4 (Appendix A.3) a similar result in a placebo test where we consider older cohorts of children in the 2012 wave as not affected by the plague and younger cohorts of children as potentially affected by the plague.

Figure 7: Estimated Impact of 2004 Locust Invasion in Mali



Source: SWARMS data base from FAO Desert Locust Information Service and DHS data, Mali waves 2006 and 2012. This figure depicts the estimated coefficient of locust invasion on height-for-age Z-scores for each quarter-year cohort born in locust invaded areas between January 2003 and December 2008. Each coefficient is plotted together with its 95% confidence bands. The blue colored coefficients are obtained using height-for-age Z-scores computed during the 2006 DHS survey, while the red ones are obtained using height-for-age Z-scores computed during the wave held in 2012. The black dashed vertical line shows the quarter of the onset of the locust plague in Mali.

5.5 The Power of Markets: Understanding their Role

We explore in detail the mechanisms through which the locust plague led to the estimated health setbacks. In Section 5.3 we have shown that the plague's impact spreads over entire localities by affecting their local markets of reference. In this Section, we first take a closer look at what happens to the local crop prices when an area is hit by the plague. We then look at the impact of the plague on child health, accounting for price effects (Subsection 5.5.1). Next, we study the importance of local crop market isolation on the impact of locust plagues on child health (Subsection 5.5.2).

5.5.1 Locust Plague and Crop Prices

We rely on a Difference-in-Differences approach, similar to Equation (1), to investigate how market prices evolved during the plague and among treated and non-treated locations. To keep our analysis consistent with the discussion in Section 5.4, we split the exposure to the plague into three distinct time windows: (i) the period in which children have been exposed, in utero, only to the speculative/anticipatory effect, (ii) the period with both speculative/anticipatory and crop failure effects, and (iii) the period with crop failure effect only. In practice, we use OMA monthly prices (years 2000-2015) of maize, millet, rice, and sorghum as the dependent variable in the following specification:

$$\log(p_{m(c,t)}) = \theta_1 T_m \mathbb{1}(t \in \text{2004Q3}) + \ \theta_2 T_m \mathbb{1}(t \in \text{2004Q4 to 2005Q2}) + \\ \theta_3 T_m \mathbb{1}(t \in \text{2005Q3 to 2006Q1}) + \beta X_{m(t)} + \mu_m + \delta_c + \xi_t + \epsilon_{m(c,t)}. \quad (2)$$

Variable $\log(p_{m(c,t)})$ stands for the prices (in logs) of crop c, in month-year period t, at market m. T_m is a treatment dummy equal to 1 if market m was exposed to a locust event within 30 km during the invasion of 2004. θ_1 is the main coefficient of interest: it measures the difference-in-differences impact of locust invasion on crop prices during the speculative/anticipatory period (third quarter of 2004). Similarly, θ_2 and θ_3 quantify analogous estimates for the other exposure periods. We account for possible confounding factors by controlling for weather conditions (SPEI) in the 12 months previous to t in market m, $X_{m(t)}$, and by adding market, crop and time fixed effects, μ_m, δ_c and ξ_t, respectively.

Table 2 presents the results. In Column (1), we report the estimates of θ_1, θ_2, and θ_3 not adding the set of weather controls, which is done in column (2). The results suggest that crop prices in locust–affected markets were significantly higher, compared to non-affected areas, only during the speculative/anticipatory effect period. During the third quarter of 2004, the plague

Table 2: Impact of Locust Plague on Crop Prices

	Log crop price			
	(1)	(2)	(3)	(4)
Speculative/anticipatory effect (θ_1)	0.085**	0.085**	0.088***	0.081***
	(0.034)	(0.034)	(0.029)	(0.028)
Both effects (θ_2)	0.019	0.019	0.008	0.001
	(0.016)	(0.016)	(0.013)	(0.014)
Crop failure effect (θ_3)	0.001	0.000	-0.010	-0.016
	(0.018)	(0.018)	(0.016)	(0.014)
Observations	51,166	51,166	51,166	51,166
R-squared	0.871	0.871	0.912	0.915
Location FE	YES	YES	YES	YES
Time FE	YES	NO	NO	NO
Crop FE	YES	NO	NO	NO
Time-Crop FE	NO	YES	YES	YES
Location Time Trend	NO	NO	NO	YES

Source: SWARMS data base from FAO Desert Locust Information Service and OMA crop prices at monthly level from 2000 to 2015. *** p<0.01, ** p<0.05, * p<0.1. Dependent variable is log crop price for maize, millet, sorghum and rice in local currency. Weather controls include SPEI index in the previous 6 months, separated in two distinct quarters. Columns (1) and (2) report the results of the estimation of Equation 2 respectively with and without weather controls. Columns (3) and (4) report the results of the estimation with weather controls, adding sequentially market-specific time trends and crop–time fixed effects.

led to an average increase of prices of more than 8%. This result is robust to the addition of market–specific time trends (column (3)) and to the inclusion of more demanding fixed effects at crop–time level (column (4)).

We now use the three different exposure time windows previously presented, together with data on local prices, to investigate the importance of different channels through which locust plagues impact child health. To do so, we build on our benchmark model of Equation (1) by splitting in utero exposure treatment into three dummies that account for the three different phases of the plague.

The results are reported in Table 3. In column (1), we estimate the impact of the locust invasion on child health, allowing for a different impact for each of these 3 windows.[31] Results show that the health setback implied by the locust invasion is statistically significant and similar in magnitude during the three periods of exposure to the plague. This result corroborates our findings presented in Section 5.4, where we document the existence of a speculative/anticipatory effect of locust plague on child health.

In column (2) we run the same specification of column (1) on the sub-sample of children for which we have market price data as a benchmark because, as explained in Section 2, we observe local crop prices only for a subset of areas in our study. We get the same pattern as in column (1) but with slightly higher magnitudes for the coefficients. In particular, children exposed to the speculative/anticipatory effect have, on average a Z-score 0.72 points below that of non-exposed children. This effect is close in magnitude to that of children exposed to both effects (average decrease of Z-score of 0.56) and that of children exposed only to the crop failure effect (average decrease of Z-score of 0.75).

To check that a significant part of the negative effect of the plague during the speculative/anticipatory period is driven by local inflation, we control for average local prices during pregnancy (column (3)).[32] The comparison between column (2) and column (3) points to a drop

[31] This specification will be our main specification for everything that follows.

[32] As explained in Section 2, in order to control for average local prices during pregnancy, we first match each DHS cluster to the closest market located in the prio–cell where the DHS cluster belongs to. Then,

of the effect suffered by children exposed only to the speculative/anticipatory effect, but not for other children that have been exposed to the crop failure effect. Indeed, the estimated coefficient for the speculative/anticipatory effect goes from -0.72 to -0.42, whereas the coefficients of the other two effects remain unchanged. Importantly, we reject the equivalence of the speculative/anticipatory effect coefficients between columns (2) and (3) (p-value equal 0.03). On the contrary, we do not reject the equivalence of the coefficients related to the remaining two phases of the plague (p-value equal to 0.58 and 0.99). This suggests that price inflation accounts for a significant part of the speculative/anticipatory effect.[33]

As expected, after controlling for prices, the speculative/anticipatory effect does not vanish completely. This confirms that the speculative/anticipatory effect goes beyond the price inflation effect (see Section 3). This effect also captures other factors. First, it includes the effect of any precautionary decrease in household consumption in anticipation of the future shock. Second, it also captures any effect of in utero exposure to maternal stress due to the news of a future harvest failure. Panel B of Table 3 shows that all the results in Panel A are robust to the exclusion of region-specific time trends.[34]

for each child belonging to a given DHS cluster, we compute the average crop price (from the matched market) during his/her pregnancy period. Since the OMA price dataset is unbalanced, we are only able to match the average prices of maize, millet, sorghum, and local rice (one of the four varieties of rice shown in Table A3).

[33]We test the statistical equivalence of the estimates across models in columns (2) and (3) with a Hausman test. The motivation for using this test for this purpose is that the model with more controls (prices) nests the one with fewer (the latter restricts the coefficients of the additional controls to be equal to zero). As such, the restricted model is more efficient if the restrictions are correct, but inconsistent if they are incorrect. In practice, we estimate simultaneously the two models with the correct specification of standard errors and test the equivalence of the cross-model parameters. This procedure is comparable to the traditional Hausman test but allows for more flexible standard errors.

[34]The analogous Hausman tests to compare coefficients between column (5) and column (6) p-values equal to 0.04 for the speculative/anticipatory effects, and 0.59 and 0.99 for the remaining two effects.

Table 3: Impact of Locust Plague on Child Health: Price Inflation Channel

	Panel A			Panel B		
	(1)	(2)	(3)	(4)	(5)	(6)
In utero treatment with speculative/anticipatory effect	-0.492***	-0.716**	-0.415	-0.496***	-0.589**	-0.311
	(0.176)	(0.276)	(0.259)	(0.168)	(0.268)	(0.261)
In utero treatment with both	-0.346***	-0.565***	-0.540**	-0.329**	-0.368	-0.339
	(0.127)	(0.205)	(0.211)	(0.144)	(0.271)	(0.286)
In utero treatment with crop failure effect	-0.494***	-0.749***	-0.767***	-0.489***	-0.496**	-0.495*
	(0.128)	(0.144)	(0.146)	(0.153)	(0.227)	(0.247)
Observations	9,173	2,910	2,910	9,173	2,910	2,910
R-squared	0.245	0.265	0.270	0.239	0.255	0.261
Cohort FE	YES	YES	YES	YES	YES	YES
Location FE	YES	YES	YES	YES	YES	YES
Child characteristics	YES	YES	YES	YES	YES	YES
Family characteristics	YES	YES	YES	YES	YES	YES
Region specific time trend	YES	YES	YES	NO	NO	NO
Control for prices	NO	NO	YES	NO	NO	YES
Mean dependent variable	-1.425	-1.273	-1.273	-1.425	-1.273	-1.273

Source: SWARMS data base from FAO Desert Locust Information Service, household survey data from 2006 DHS wave in Mali, local crop data from OMA. Dependent variable is child height-age Z-score. Full set of controls includes mother's height, education, gender and age of household head, household wealth index, SPEI index, birth order, time gap between conception and the previous and following pregnancies. Robust standard errors in parentheses are clustered at PRIO cell grid level. *** p<0.01, ** p<0.05, * p<0.1. Column (1) uses the full sample. Column (2) restricts the sample to observations with price data without controlling for prices. Column (3) controls for prices. We control for average prices using pregnancy using OMA monthly data of maize, millet, rice, and sorghum. Column (4), (5) and (6) are equivalent to Columns (1), (2) and (3), respectively, but excluding region specific time trends.

5.5.2 Locust Plague and Level of Isolation of Local Markets

The evidence presented so far suggests that local markets play a key role in the timing of the adverse impact of agricultural shocks such as locust plagues. The extent to which a given local market is trading crops with other markets can also play a crucial role in the magnitude of the shock faced by households. Local and minor agricultural shocks can be easily absorbed by well–connected markets. Major shocks may however exacerbate the effect of food shortage at the local level because crops are sold fast to a larger market (Burgess and Donaldson, 2010; Townsend, 1995).

Going back to the distinction between speculative/anticipatory effect and crop failure effect, market openness can have very different consequences. On the one hand, well–connected areas hit by the crop failure effect can rely on crop supply from non-affected areas in their network, to mitigate the effects of the shock. Conversely, rural areas that are isolated and rely mostly on their own agricultural production may be more affected when hit by such shock. On the other hand, a high level of trading may imply that local crops are sold fast, putting vulnerable households at higher risk. This may exacerbate the inflation in rural areas with a single harvest per year.

In order to investigate how market openness affects the impact of locust plagues on child health, we use the travel time to the nearest major city to capture the level of isolation of different locations. For each household cluster coordinate from the DHS database, we get the minimum travel time to the nearest town of at least 50,000 inhabitants in 2000 from Uchida and Nelson (2010).[35] The assumption behind the use of this metric is that large towns work as local trade hubs. Therefore, differently from rural and isolated areas, places that are close to main cities have easier access to a well connected and diversified set of markets.

In this perspective, we focus the analysis that follows on the most vulnerable areas, i.e., the sub-sample of households located in rural areas. We build on the specification used in column (1) of Table 3 by adding the interaction terms between travel time and the three treatment variables. Results are reported in Table 4. Column (1) shows that children born in more isolated areas suffer stronger health setbacks when exposed in utero to the crop failure effect. In particular, a one standard deviation increase of travel distance to the nearest 50,000 inhabitants city implies, for exposed children, an average decrease in Z-scores of 0.16 on top of the baseline effect (0.45 decrease in Z-score). This is not the case for children exposed only to the speculative/anticipatory effect. Indeed, the estimated coefficient of the interaction term between speculative/anticipatory effect and travel time is not statistically significant and, in magnitude, it is approximately one–third of the estimated coefficient of interaction between crop failure effect and travel time. This result is robust to the inclusion household wealth index (column (2)) and region-specific time

[35] This metric is derived from a global high-resolution (1 km^2 pixel) raster map of accessibility. It is the result of network analysis using a combination of several sources, most of them collected between 1990 and 2005. The original pixel value is the estimated travel time in minutes by land transportation from the pixel to the nearest major city. In our analysis we use standardized values of the travel time, inferring sample average and standard deviation of travel time from the sample of enumeration areas.

Table 4: Impact of Locust Plague on Child Health and Level of Isolation of Rural Areas

	Height-age Z-score		
	(1)	(2)	(3)
Treatment with speculative/anticipatory effect	-0.462**	-0.471**	-0.452*
	(0.217)	(0.221)	(0.236)
Treatment with both	-0.266*	-0.267*	-0.244*
	(0.142)	(0.142)	(0.129)
Treatment with crop failure effect	-0.454***	-0.446***	-0.418***
	(0.146)	(0.148)	(0.131)
Treatment with speculative/anticipatory effect × Travel time	-0.051	-0.045	-0.035
	(0.075)	(0.075)	(0.078)
Treatment with both × Travel time	-0.120***	-0.119***	-0.101***
	(0.015)	(0.018)	(0.030)
Treatment with crop failure effect × Travel time	-0.155***	-0.159***	-0.139***
	(0.024)	(0.026)	(0.039)
Observations	6,479	6,479	6,479
R-squared	0.239	0.240	0.245
Cohort FE	YES	YES	YES
Location FE	YES	YES	YES
Child characteristics	YES	YES	YES
Family characteristics	YES	YES	YES
HH Wealth	NO	YES	YES
Region time trend	NO	NO	YES
Mean dependent variable	-1.607	-1.607	-1.607

Source: SWARMS data base from FAO Desert Locust Information Service and household survey data from 2006 DHS wave in Mali. *** p<0.01, ** p<0.05, * p<0.1. The dependent variable in all regression are the children's height-for-age Z-scores. Full set of controls includes mother's height, education, gender and age of household head, household wealth index, SPEI index, birth order, time gap between conception and the previous and following pregnancies. Travel time is the standardized travel time to the nearest city of more than 50,000 inhabitants. Column (2) controls for household-level wealth. Column (3) controls for region-specific time trends.

trends (column (3)).[36] Overall, this suggests that isolation itself plays a role and these results are not likely to be biased by the fact that more isolated households might be poorer, and thus more vulnerable, than better–connected ones.

To conclude, the fact that, in each specification, the interaction term between travel time and the speculative/anticipatory effect treatment variable is small and not significant suggests that the adverse effect during the speculative/anticipatory period is rather homogeneous across space,

[36] In each of these specifications, we can reject the statistical equivalence of these interaction terms, with a p-value between 0.07 and 0.05.

irrespective of how isolated local markets are. This is not the case for the crop failure effect, since it gets more severe as we move to more isolated areas. This could be due to economic agents (intermediaries and/or consumers) over-predicting the impact or magnitude of the shock during the growing season, at least in non-isolated areas.

5.6 Further Heterogeneity: Migration, Residence, and Gender

In this section, we discuss potential threats to identification due to migration, and how the main results of the paper vary by type of place of residence and gender. Investigating whether the estimated impact is driven by specific sub-groups of the population can be useful to further understand the channels through which this effect is operating. We run our main specification presented in column (1) of Table 3 on several sub-samples. Results are shown in Table 5.

First, we focus on migration. One plausible issue concerning our results is that anthropometric measures for children in our sample were measured few years after the plague took place. Some of these children may have migrated to other places of residence after the plague. This makes it impossible to attribute them to the right treatment group. If this is the case, our estimates might be subject to an attenuation bias. Moreover, a subgroup of those that have migrated across treatment areas might have been positively or negatively selected, leading to additional biases in the estimates. For instance, richer households may move out of the treated areas after the plague leading to an upward bias in the estimated coefficients.

We use the information on the number of years of residence in the locations where each household has been surveyed and we run our main regression restricting the sample to those that lived at the same location for more than 2 years (before the plague since households were surveyed in 2006) in column (1) and those that have always lived there in column (2) of Table 5. Results are robust in both cases: the point estimates are statistically significant and range from -0.31 to -0.51, close to the benchmark estimates (between -0.34 and -0.49; see Table 3). Thus, it suggests that our estimates are not affected by potential migration.

Furthermore, columns (3) and (4) display the outcomes of the main regression estimated on urban and rural sub-samples. The results suggest that children living in both areas suffer major health setbacks. The reduced sample size for the urban sample affects the precision of the estimates but the magnitudes are comparable to those from the rural sample.[37]

Finally, we explore the degree of heterogeneity of our results in terms of gender. Usually, the literature on in utero shocks finds a large negative bias against girls (Dagnelie et al., 2018; Valente, 2015) because female fetuses are more resilient and this may lead to a stronger selection effect for male children born alive. Columns (5) and (6) of Table 5 show, instead, a negative impact of the plague on children of both genders.[38] In both subsamples, we cannot reject the statistical equivalence of the three treatment dummies (the F-test yields a p-value of 0.51 (0.15) for the male (female) sample).

[37] Table A4 documents the estimation results using a unique dummy variable for treatment, analogously to Table 5. The results in both rural and urban subsamples are comparable in magnitude to our benchmark effect (between -0.33 and -0.48) and statistically significant.

[38] In Table A4 we show that the average treatment effect (with a single treatment dummy), for both genders, are large, statistically significant, and comparable to the benchmark effects (between -0.33 to -0.54).

Table 5: Heterogeneity: Impact of Locust Plague on Child Health

	(1)	(2)	(3)	(4)	(5)	(6)
	Years of residence		Place of residence		Gender	
	2 + years	Always	Urban	Rural	Male	Female
In utero treatment with speculative/anticipatory effect	-0.498***	-0.521**	-0.587	-0.505**	-0.837***	-0.296
	(0.170)	(0.220)	(0.356)	(0.208)	(0.268)	(0.350)
In utero treatment with both	-0.316**	-0.517***	-0.297	-0.344**	-0.496**	-0.149
	(0.151)	(0.174)	(0.153)	(0.168)	(0.209)	(0.176)
In utero treatment with crop failure effect	-0.463***	-0.485***	-0.287	-0.594***	-0.468**	-0.537***
	(0.152)	(0.183)	(0.159)	(0.169)	(0.186)	(0.204)
Observations	8,811	4,995	2,694	6,479	4,654	4,519
R-squared	0.241	0.268	0.209	0.238	0.269	0.300
Cohort FE	YES	YES	YES	YES	YES	YES
Location FE	YES	YES	YES	YES	YES	YES
Child characteristics	YES	YES	YES	YES	YES	YES
Family characteristics	YES	YES	YES	YES	YES	YES
Mean dependent variable	-1.431	-1.449	-0.986	-1.607	-1.484	-1.364

Source: SWARMS data base from FAO Desert Locust Information Service and household survey data from 2006 DHS wave in Mali. Dependent variable is height-for-age Z-score. Full set of controls includes mother's height, education, gender and age of household head, household wealth index, SPEI index, birth order, time gap between conception and the previous and following pregnancies. Robust standard errors in parentheses are clustered at PRIO Cell grid level. *** p<0.01, ** p<0.05, * p<0.1. Column (1) and (2) report the results of the baseline specification (column (3) of Table 1) restricting the sample to households that have reported living in the location where they were interview for at least 2 years, or since ever, respectively. Columns (3) and (4) split the sample between urban and rural. Column (5) restricts the sample to male children while Column (6) uses only female ones.

6 Concluding Remarks

In this paper, we study how in utero exposure to desert locust plagues can lead to substantial health setbacks. We estimate the causal impact of a plague that occurred in Mali in 2004. We first find that children exposed in utero have, on average, a height-for-age Z-score 0.42 points lower than non-exposed children. We show that the detrimental effects of locust invasions have broad repercussions that involve entire localities, by impacting their local crop markets.

We then consider the mechanisms that led to these health setbacks. We provide evidence of a strong speculative/anticipatory effect of the locust invasion that kicked in during the plague itself, and a local crop supply shock that lasted at least until the subsequent harvest. Cohorts of children that were subject only to the speculative price effect suffered as much as those exposed to the actual crop failure effect. Only a part of this speculative/anticipatory effect can be explained by local inflation. The rest captures other effects related to stress/anxiety or precautionary decrease in crop consumption in anticipation of the effects of the shock. A core empirical finding along this dimension is the role of access to markets: our results suggest that market openness dampens the adverse health effects of exposure to crop failure effect.

This study has relevant implications from a policy perspective. In particular, our results suggest that it is important to address expectations during pest invasions that affect agricultural production. It is crucial to put efforts in two main directions: (i) prevent inflation and impeding any type of anticipation that might have adverse effects; (ii) provide safety nets to the most vulnerable households to dampen the consequences of these shocks on young children.

With the alarming climate change prospects, one can expect more frequent extreme weather conditions in the breeding areas of the desert locusts, which could lead to more plagues in the future. More recently (since 2019), ongoing locust invasions have been affecting several countries in the Horn of Africa, the Middle East, South Asia, and Latin America. As of April 2020, 23 countries in Africa and Asia, from Pakistan to Tanzania, have been affected (World Bank, 2020). This is a global outbreak that is threatening to reach the level of a plague. Preparing to limit the consequences of such locust invasions is thus necessary for policymakers that operate in the potential invasion areas.

Appendix

A.1 Descriptive Statistics and Balance Table

Table A1: Descriptive statistics: children, mothers, households, and clusters.

	Obs.	Mean	St. Dev.	Min	Pctl(25)	Median	Pctl(75)	Max
Children characteristics								
Height (z-scores)	9,173	−1.42	1.79	−5.99	−2.59	−1.45	−0.36	6.00
Treated	9,173	0.07	0.25	0	0	0	0	1
Age in years	9,173	2.07	1.36	0	1	2	3	4
Female	9,173	0.49	0.50	0	0	0	1	1
Gap from previous birth	9,173	2.77	1.42	0	2	2	4	5
Gap to next birth	9,173	3.95	1.54	0	2	5	5	5
SPEI[1]	9,173	−0.21	0.49	−1.75	−0.54	−0.12	0.16	1.09
Respondent (Mother) charact.								
Height (meters)	6,953	1.61	0.07	1.00	1.57	1.61	1.65	1.92
Education (years)	6,953	0.93	2.52	0	0	0	0	17
Years living in the cluster	6,953	26.23	15.12	0	10	39	39	39
Measured children	6,953	1.32	0.50	1	1	1	2	4
Household characteristics								
Household members	6,184	6.82	3.29	2	4	6	8	29
Age household head	6,184	41.79	12.14	16	33	40	49	98
Female household head	6,184	0.08	0.27	0	0	0	0	1
Measured children	6,184	1.48	0.70	1	1	1	2	6
Poor dweling[2]	6,184	0.39	0.49	0	0	0	1	1
Number of interviewed women	6,184	1.12	0.36	1	1	1	1	4
Clusters characteristics								
Rural	385	0.65	0.48	0	0	1	1	1
Distance to nearest town (hours)	385	4.07	4.48	0.18	1.64	3.51	5.41	56.70
Households by cluster	385	16.06	4.57	4	13	16	19	31

Notes: [1]SPEI stands for the weigthed average of the SPEI index during the period in utero; see section A.2 for details. [2]Poor dweling stands for those in the 2 bottom quintiles of the wealth index distribution. The wealth index is built based on household ownership of selected assets, such as televisions and bicycles; materials used for housing construction; and types of water access and sanitation facilities.

Table A2: Balance table: differences in average characteristics between treated and control groups.

	Control group	Treatment Group	Difference
Respondent (mother) characteristics			
Height (meters)	1.611***	1.618***	0.008**
	(0.001)	(0.003)	(0.003)
Years of education	0.884***	0.959***	0.075
	(0.060)	(0.154)	(0.155)
Years living in the cluster	26.192***	26.177***	−0.015
	(0.358)	(0.965)	(0.954)
Household/Cluster characteristics			
Household members	7.473***	7.190***	−0.283
	(0.079)	(0.208)	(0.211)
Age of household head	41.878***	41.131***	−0.747
	(0.198)	(0.752)	(0.732)
Female household head	0.071***	0.108***	0.037**
	(0.005)	(0.019)	(0.018)
Rural household	0.706***	0.710***	0.004
	(0.023)	(0.061)	(0.060)
Poor dweling[1]	0.403***	0.359***	−0.043
	(0.016)	(0.043)	(0.042)
Travel distance to nearest town (hours)	4.037***	5.394***	1.357
	(0.181)	(1.109)	(1.012)
Observations	8,569	604	9,173

Note: *p<0.1; **p<0.05; ***p<0.01. [1]Poor dweling stands for those in the 2 bottom quintiles of the wealth index distribution. The wealth index is built based on household ownership of selected assets, such as televisions and bicycles; materials used for housing construction; and types of water access and sanitation facilities.

Table A3: Descriptive statistics of OMA prices: all sample, by markets, and market–crops.

	Obs.	Mean	St. Dev.	Min	Pctl(25)	Median	Pctl(75)	Max
All sample								
Year	51,166	2,007.78	4.62	2,000	2,004	2,008	2,012	2,015
Maize	51,166	0.16	0.36	0	0	0	0	1
Millet	51,166	0.21	0.41	0	0	0	0	1
Sorghum	51,166	0.20	0.40	0	0	0	0	1
Rice (local)	51,166	0.20	0.40	0	0	0	0	1
Rice (white)	51,166	0.10	0.30	0	0	0	0	1
Rice (red)	51,166	0.11	0.31	0	0	0	0	1
Rice (paddy)	51,166	0.03	0.18	0	0	0	0	1
Market–crop								
Length of series	432	118.44	76.95	1	24.8	160	190	192
Min(Year)	432	2,003.04	5.17	2,000	2,000	2,000	2,004	2,015
Max(Year)	432	2,014.13	2.34	2,000	2,015	2,015	2,015	2,015
Average SPEI	432	−0.10	0.16	−0.67	−0.15	−0.08	−0.04	0.49
Average price (local currency)	432	234.60	96.53	97.17	156.45	201.37	302.09	563.83
Market								
Number of crops	74	5.84	1.06	2	5	6	7	7
Treated	74	0.24	0.52	0	0	0	0	3
Distance to Nearest Town (hours)	74	1.64	1.94	0.00	0.11	1.16	2.38	7.98

Notes: The OMA price data is a monthly panel of crop prices at the market–crop–year–month level. [1]"Maize"refers to a dummy for observations in the panel for that crop. Its mean stand for the share of observations in the sample for maize prices. The same stands for the other crop dummies. [2]Length in months. [3]Average SPEI during the previous 12 months of each observation.

A.2 Climate Data

To account for potential weather-related omitted variables in our analysis, we collect data from the Standardized Precipitation-Evapotranspiration Index (SPEI, see Vicente-Serrano et al., 2010). The SPEI is a multi–scalar index that jointly maps rainfall, temperature, and potential evaporation into a standardized index of drought. Importantly, it is measured in units of standard deviations from the long–term average (1901 – 2015 in version 2.5) and has zero average by construction. It is available in a 0.5 x 0.5 grid, which is matched to the household coordinates available from the DHS surveys.

We want to control for exposure of individual pregnancies to food supply shocks due to weather conditions in our empirical specification. To do so we need to determine how weather affects each mother's nutritional intake during the 9 months in utero. We follow Kudamatsu et al. (2016) and focus on variations in the SPEI index during the relevant growing seasons, as summarized by a simple index constructed as follows.

The relevant growing seasons of an individual birth depend on its timing relative to local harvest time. We weigh the last 3 harvests before the birth of cohort t (year-month) by the number of months that a pregnant woman will spend relying on each of them.

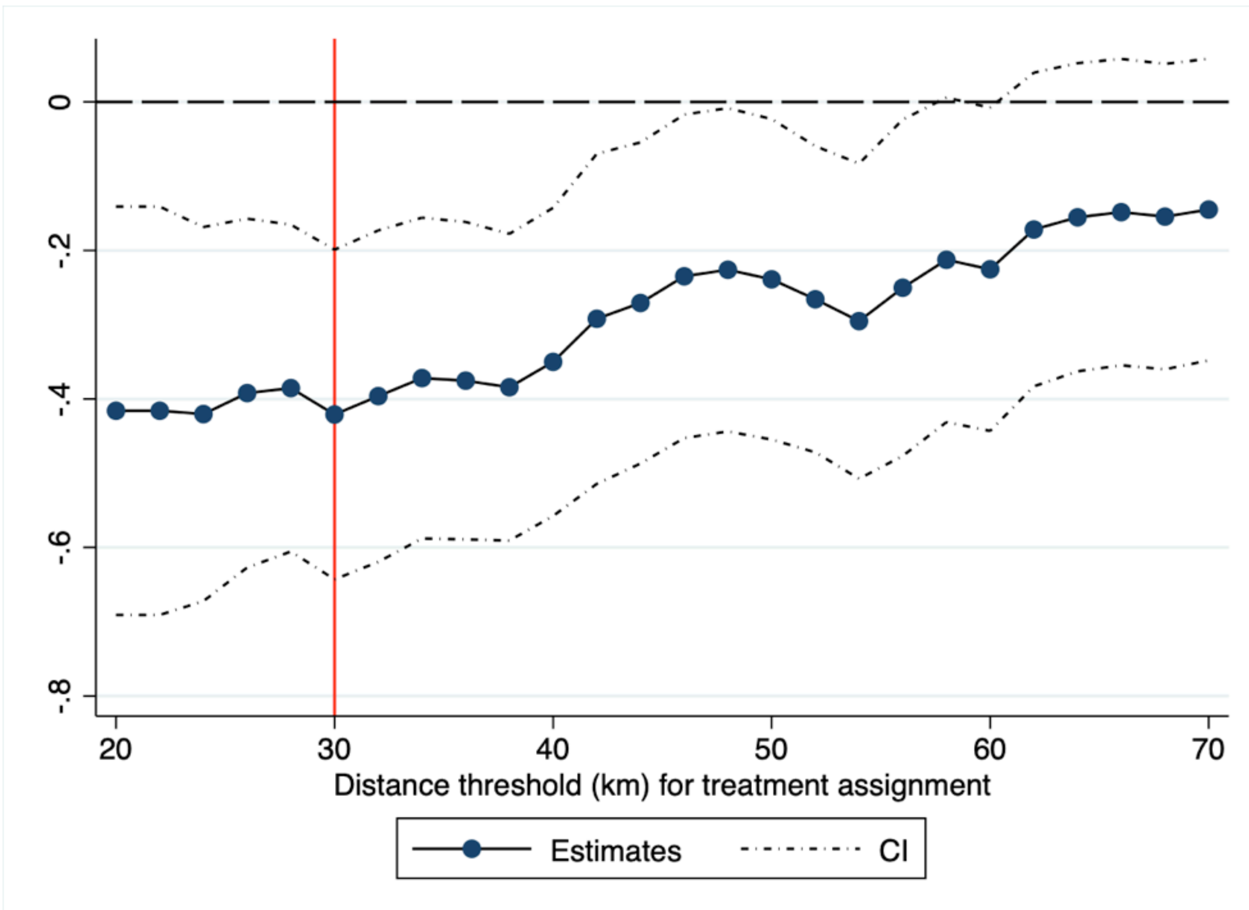
$$SPEI^{g,t} = \sum_{i=1}^{3} \omega_i S^{g,t}_{a(t)-i},$$

where $\sum_i \omega_i = 1$, $S^{g,t}_{a(t)}$ is average SPEI index in cell g during the growing season of the year in which children of cohort t are born. For example, children born in January of a given year a (conceived in April of the previous year), spend 3 months relying on the harvest from year $a-1$ (October to December) and 6 months relying on the harvest of year $a-2$ (April till September) so the weights are 0/9, 3/9 and 6/9 for $\omega_0, \omega_1, \omega_2$, respectively. The results are robust to using SPEI for the last 3 years as separate variables. They also do not change when we consider SPEI for the entire year.

A.3 Robustness of the Average Impact of Locust Plagues

In this Appendix, report the results of the robustness described in Section 5. We first test our definition of *treatment group*. We allow the threshold that defines treated areas to vary between 20km to 70km, in steps of 2k; results are shown in Figure A1. Moreover, we test the robustness of the results presented in Table 1 performing several robustness checks on alternative empirical specifications and data samples (described in Section 5). We present our results in Table A4.

Figure A1: Local Impact of Locust Plague on Child Health



Source: SWARMS data base from FAO Desert Locust Information Service and DHS data, Mali wave 2006. The dependent variable in each specification is height-for-age Z-score. In each iteration we change the definition of treatment group: in the first specification an area treated if it is located at most 20 km from at least one swarm event; in the subsequent specifications we add progressively 2 km to the threshold that defines what is a treatment area, up to 70 km. For each of these specifications, we plot the estimates of the impact of locust plague on height-for-age Z-score (i.e., γ in Equation 1) together with their 95% confidence bands.

Table A4: Heterogeneity: Average Impact of Locust Plague on Child Health

	(1)	(2)	(3)	(4)	(5)	(6)
	Years of residence		Gender		Place of residence	
	2 + years	Always	Male	Female	Urban	Rural
In utero treatment	-0.405***	-0.504***	-0.536***	-0.337**	-0.331*	-0.473***
	(0.111)	(0.129)	(0.136)	(0.156)	(0.173)	(0.127)
Observations	8,811	4,995	4,654	4,519	2,694	6,479
R-squared	0.241	0.268	0.269	0.300	0.209	0.238
Cohort FE	YES	YES	YES	YES	YES	YES
Location FE	YES	YES	YES	YES	YES	YES
Child characteristics	YES	YES	YES	YES	YES	YES
Family characteristics	YES	YES	YES	YES	YES	YES
Mean dependent variable	-1.431	-1.449	-1.484	-1.364	-0.986	-1.607

Source: SWARMS data base from FAO Desert Locust Information Service and household survey data from 2006 DHS wave in Mali. Dependent variable is height-for-age Z-score. Full set of controls includes mother's height, education, gender and age of household head, household wealth index, SPEI index, birth order, time gap between conception and the previous and following pregnancies. Robust standard errors in parentheses are clustered at PRIO Cell grid level. *** p<0.01, ** p<0.05, * p<0.1. Column (1) and (2) report the results of the baseline specification (Column (3) of Table 1) restricting the sample to households that have reported living in the location where they were interview for at least 2 years, or since ever, respectively. Columns (3) restricts the sample to male children while column (4) uses only female ones. Columns (5) and (6) split the sample between urban and rural.

A.4 Additional Analyses

Table A5: Additional Analyses: Impact of Locust Plague on Child Health

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
In utero treatment with 30 km ring	-0.31**	-0.336***	-0.476***			-0.333***	-0.287***	
	(0.150)	(0.120)	(0.085)			(0.101)	(0.106)	
In utero treatment at municipality level				-0.327***				
				(0.117)				
In utero treatment including swarms beyond country borders					-0.392***			
					(0.113)			
Placebo treatment								0.150
								(0.195)
Observations	9,173	9,173	9,173	9,173	9,173	14,209	9,059	4,182
R-squared		0.2352	0.2	0.238	0.239	0.226	0.257	0.346
Number of mother	6,953							
Cohort FE	YES	YES	YES	YES	YES	YES	YES	YES
Mother FE	YES							
Child characteristics	YES	YES	NO	YES	YES	YES	YES	YES
Location FE		YES	YES	YES	YES	YES	YES	YES
Family characteristics		YES	NO	YES	YES	YES	YES	YES
Mean dependent variable	-1.425	-1.425	-1.425	-1.426	-1.425	-1.284	-1.117	-1.276

Source: SWARMS data base from FAO Desert Locust Information Service and household survey data from 2006 DHS wave in Mali. The dependent variable in all regression are the children's height-for-age Z-scores. Robust standard errors in parentheses are clustered at enumeration area level. *** p<0.01, ** p<0.05, * p<0.1. Column (1) to (4) use data from the 2006 wave. Column (1) shows results for a specification with mother fixed-effects. Coulumn (2) is equivalent of our main specification in Column (3) Table 1, but using DHS survey sample weights. Column (3) shows baseline resutls with no controls allowing for spatial HAC standard errors as in Conley (1999). Column (4) defines treatment area at municipality level. Column (5) adds events that occurred beyond the borders of Mali to define treatment areas. Column (6) uses data from 2006 and 2012 DHS survey waves. Column (7) restrict the previous sample to children born after June 2004. Column (8) is a placebo specification that uses only data from the 2012 DHS survey wave and treated children are those born in locust affected areas after 2010 (in 2011 or 2012).

Table A6: Impact of Locust Plague on Neo-natal and Infant Mortality

	death < 1 month		death < 12 month	
	(1)	(2)	(3)	(4)
In utero treatment	0.007	0.007	-0.005	-0.003
	(0.010)	(0.010)	(0.019)	(0.018)
Observations	24,189	24,189	22,638	22,638
R-squared	0.073	0.074	0.112	0.113
Cohort FE	YES	YES	YES	YES
Location FE	YES	YES	YES	YES
Child characteristics	YES	YES	YES	YES
Family characteristics	YES	YES	YES	YES
Region specific time trend	NO	YES	NO	YES
Mean dependent variable	0.0499	0.0499	0.122	0.122

Source: SWARMS data base from FAO Desert Locust Information Service and household survey data from 2006 DHS wave in Mali. Dependent variable is dummy equal 1 for children who die within the first month for n'eo-natal mortality and within the first year for infant mortality. Full set of controls includes mother's height, education, gender and age of household head, household wealth index, SPEI index, birth order, time gap between conception and the previous and following pregnancies. Robust standard errors in parentheses are clustered at PRIO Cell grid level. *** p<0.01, ** p<0.05, * p<0.1.

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