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

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

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Abstract

This paper investigates the consequences of the 2004 locust plague in Mali. 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 document a substantial impact of the plague on crop price inflation before the harvest. Regarding health setbacks, children subject only to the *speculative/anticipatory effect* suffered as much as those exposed to the actual *crop failure effect*. The latter is more severe for children born in isolated areas.

Keywords: Desert Locust Swarms, Agricultural Shocks, Local Markets, Child Health.
JEL Codes: O12, I15, Q12, Q18.

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Introduction

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 these 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).

In this paper, we study the impact, on child health, of exposure to an agricultural shock that is indirectly linked to extreme weather conditions and climate change: the damage caused by desert locust plagues. Locust swarm invasions are destructive events that recurrently put food supply in many developing countries at risk (especially in sub-Saharan Africa, see 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 areas to feed and reproduce.

We focus on how this shock operates in areas with a single harvest per year.[1] We argue that agricultural shocks due to pest invasion can affect households living in this type of economy 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.[2] 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.[3] We can separately identify these two effects because

[1] This is the case in the Sahel region which covers more than a dozen countries, stranded between the Sahara desert to the north and the belt of humid savannas to the south. It is one of the most vulnerable areas to desert locust invasions due to its proximity to the breeding zones.

[2] 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).

[3] 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

they kick in at different points in time.

We explore in detail these two effects, by making use of a sharp episode of locust plague invasion and tracing out how it affected the health of children exposed to it in utero. For that, we rely on the timing and location of locust swarm events that occurred between June and December 2004 in Mali to identify the temporal and spatial variation in the exposure of different cohorts of children to the plague. Data on locust swarm invasions come from the locust monitoring system run by the Food and Agriculture Organization (FAO) Desert Locust Information Service (DLIS). Geocoded household survey data with detailed information on the timing of birth and health outcomes of children is taken from the Demographic and Health Surveys (DHS). 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.

In the first part of our empirical analysis, we start by showing that the plague led to significant inflation of crop prices (average increase of 8.4%) in affected areas, compared to non-affected areas, during the growing season of 2004.[4] We take this result as empirical evidence of the 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 can 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 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.

We then turn to the core of our analysis and explore the impact of locust invasion on child health. First, using a Difference-in-Differences identification strategy, we show that children exposed in utero to the adverse effects of the plague suffered major health setbacks. They had, 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.

In the second part of this analysis, we study the timing of the estimated effect by

long-term effect.

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

quarters 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] Importantly, we argue that the health effects of the exposure (in utero) to the *speculative/anticipatory effect* go beyond the documented local crop price inflation effect for two main reasons. First, it includes (potentially) any precautionary consumption smoothing effect: a decrease in grain 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, 2023). We are not able to tease out these mechanisms with our data (this is beyond the scope of this paper). We argue nonetheless that the documented significant inflation effect in anticipation of the future crop failure cannot be explained by any of these two alternative mechanisms or by other alternative factors such as increased use of pesticides, a devotion of local resources to controlling the invasion, limited access to health care, etc.[6] Moreover, we show that the estimated treatment effect (on child health) of exposure to the *speculative/anticipatory effect* is 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, household fixed effects, and mother fixed effects. We also argue that they are not likely to be biased by potential migration, pre-existing differences in trends between treated and non-treated areas, and exposure to anti-locust pesticide spraying. Results are also robust to restricting the analysis to male-female and rural-urban sub-samples.

The distinction between the *speculative/anticipatory effect* and the *crop failure effect* is crucial when we consider how this shock affects time-sensitive health investments such as those that households make when they have children in utero. The anticipation of future negative shocks such as a crop failure at the next harvest may lead to persistent health effects for entire cohorts of children that would otherwise not suf-

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

[6] The spray of pesticides was limited in Mali during the plague due to limited resources for flying aircrafts and most of the resources used to fight the plague came from international donors (through FAO) and (to a much smaller extent) central government resources.

[7] This could be because 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.

fer any detectable effect. The locust plague episode studied in this paper provides a setup that allows us to document this *speculative/anticipatory effect* separately from the actual *crop failure effect*. This effect is also potentially present in other types of agricultural shocks such as droughts or floods since agents often form strong beliefs about the next harvest outcome early in the rainy season.[8] A common illustration of such behavior is when farmers decide to replant some seeds in the middle of the season hoping to receive the right amount of rainfall for the new crops to produce as well and help limit the damage of the anticipated shock. This legitimate anticipation of potential negative shock may however lead to persistent health consequences for vulnerable household members such as children in utero.[9]

The findings of this paper also have important 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 contributes to two 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 this literature focused on identifying the effect on child health of exposure to weather shocks (Adhvaryu et al., 2019; Maccini and Yang, 2009), violence and civil wars (Tapsoba, 2023; Dagnelie et al., 2018; Quintana-Domeque and Ródenas-Serrano, 2017; Koppensteiner and Manacorda, 2016; Valente, 2015) or adverse institutional setup (Kudamatsu, 2012). The health impact of desert locust swarms has also been investigated more recently in this literature.[10] Le and Nguyen (2022) analyze the impact of prenatal exposure to locust events in 39 countries in Africa and Asia between 1990 and 2018. Linnros (2017) estimates the aggregate effect of locust infestations on child health using data from Senegal, Burkina Faso, Mali, and

[8]Measuring these types of news or beliefs is difficult in the case of rainfall shocks compared to the presence of locust swarms during the growing season.

[9]There is also the issue that the shock may turn out not to be as bad as anticipated leading to an important cost in terms of time-sensitive investments but no upside effect ex-post from the anticipation.

[10]The consequences of pest invasions on education and adult height have been studied in Baker et al. (2020), Banerjee et al. (2010) and De Vreyer et al. (2014).

Niger. Importantly, our contribution goes beyond documenting the average impact of locust swarms: it lies in the careful investigation of the underlying channels through which pest invasions affect household welfare in developing countries. Our paper is the first one to document evidence of a *speculative/anticipatory effect* on top of the *crop failure effect* for this type of shock.

Second, this paper is related to the literature on the effects of news about the 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-off 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.

The paper proceeds as follows. Section 1 provides some background on locust plagues. Section 2 discusses the conceptual framework and the channels through which locust invasions can affect the well-being of households. Section 3 presents the data. In Section 4 we study the impact of locust plague on local crop price, while in Section 5 we analyze its impact on child health. Section 6 concludes.

1 Background: Desert Locust Plagues

Desert locusts are insects that live in the arid and hyper-arid zones of the Sahel region, northern Africa, the Middle East, and Southeast Asia, as shown in Figure A.1. They pertain to the family of *grasshoppers* and normally inhabit desert zones, called *recession areas* (bounded by the black solid line in Figure A.1), in a solitary, harmless, and integrated way with the local ecology. Under favorable breeding conditions (high rainfall followed by periods of relatively mild temperatures), they undergo a gregarization process with substantial changes in behavior, morphology, and physiology. They start behaving as a group that can then turn into a large swarm.

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 A.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.[11] 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. Fortunately, such events are not very recurrent nowadays, especially if compared to the period before 1970 (see fig. A.2). 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 relatively severe. This is the case of the plague that occurred between 2003 and 2005, whose consequences we analyze in this paper. The most recent locust outbreak of 2020 affected more than 23 countries in Africa, the Middle East, and South Asia (The Economist, 2020; World Bank, 2020).

The 2003-2005 Desert Locust Plague. This 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.

The case of Mali. Mali 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.

[11] 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).

Third, this is a poor country that relies mostly on agriculture.[12]

2 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 child development is, however, not straightforward. It may happen through different channels that are important to disentangle.

Agricultural production has a cyclical nature, following a specific seasonal calendar. Figure 1 shows this seasonal calendar for 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 from October to November. The 2004 locust plague infested parts of Mali from June to December (see Figure 2, Panel A). 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 Panel A of Figure 2, has not been affected by the plague.[13] 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.

2.1 Speculative/Anticipatory Effect

Between June and October 2004, i.e., during the peak of the plague, households and local 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 house-

[12]Other countries with similar characteristics to Mali could not be investigated in this study because of the lack of data around the year of the shock (2004).

[13]There were a few isolated locust events that happened in 2003 in the North of Mali but they all occurred after the harvest in the last quarter. We provide more details on these events in Section 3.

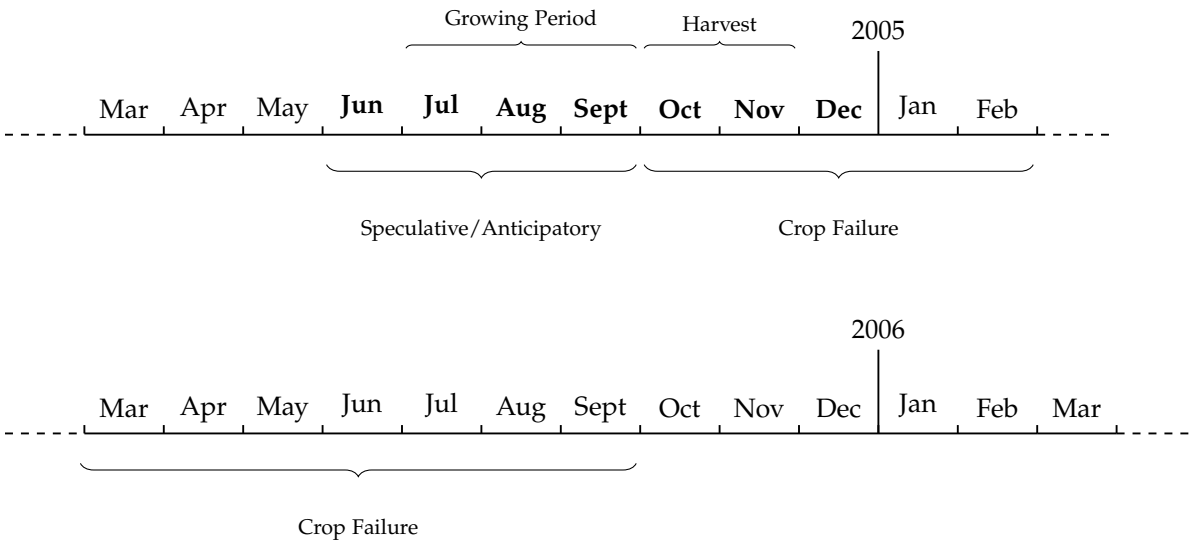

Figure 1: Seasonal Agricultural Calendar in Mali and Exposure of Agricultural Markets to the 2004 Locust Plague.

Note: The timeline starts in March 2004 and illustrates the temporal overlapping of the 2004 plague and the agricultural calendar in Mali as of FEWS-NET (2005). The plague took place between June and December 2004 (months in boldface), coinciding with the growing and harvest seasons of 2004. The underbrackets stand for the type of effects that agricultural markets were exposed to. Figure A.3 provides an analogous timeline but with respect to the exposure of cohorts born before, during, and after the plague.

holds with limited resources, especially because the plague happened during the lean season.[14] 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.[15] 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

[14]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.

[15]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).

optimally such regular price variation because of credit market imperfections (Burke et al., 2019) and non-monetary storage costs (Gross et al., 2020).

This is a pure speculative/precautionary effect since the actual quantities of grains available (including those in storage) remain the same. In Section 4 we document the impact of this 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 planting and growing seasons of 2004, i.e. when local markets were still relying on a non-affected harvest. Yet, as argued above, the speculative effect goes beyond this price inflation effect since equilibrium crop quantities consumed by households can drop without a price increase.

Stress/anxiety due to the news. News about future harvest failure could create a stressful environment in budget-constrained households that anticipate an imminent consumption shock. This may impact children in utero through maternal stress, in line with the empirical literature that shows that maternal exposure to stressful events (e.g. earthquakes) leads to worse birth outcomes (Torche, 2011; Talge et al., 2007).

In what follows, we consider children born during the third quarter of 2004 as exposed in utero to the speculative/anticipatory effect. Specifically, we expect to find a detectable impact of in-utero exposure on the health of children that have been exposed to the plague for at least one month while in utero. Children born in June 2004 are exposed in utero for less than a month (or even not exposed at all depending on their birth date). Therefore, since their in-utero exposure to the plague is marginal, we do not consider them as potentially impacted by the speculative/anticipatory effect in our main specification.[16]

2.2 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 this entire 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).[17]

Therefore, in terms of in-utero exposure of children, only the cohorts born from

[16] In our sample, 206 children were born in June 2004 (2% of the sample), 24 of which from treated areas. Removing these children from the analysis does not affect our results as shown in Table A.9.

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

the third quarter of 2005 onward (conceived in October 2004 or later) 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, which includes the speculative/anticipatory effect period (Figure 1). We will exploit such distinction when separating the latter from the crop failure effect.[18]

2.3 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, in Section 5.3 we show empirically that there are no differences in health status between children conceived in treated versus non-treated areas after November 2005.

3 Data and Descriptive Statistics

Locust Swarm Data. We collect geographical and temporal incidence of locust swarms from the SWARMS database (Cressman, 1997).[19] 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 each event related to the presence of a locust swarm, we collect information on its date and the geographic coordinates in which the event occurred.

Panel A of Figure 2 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. As discussed in detail in Section 2, this period encompasses the growing season. Conversely, nothing happened during the growing season of 2003.[20] This means that the plague episode that we study damaged

[18]Figure A.3 shows the set of cohorts exposed to each of these exposure effects or to both effects while in utero.

[19]See Appendix Appendix A.2.1 for more details on swarm data collection.

[20]Some locust events occurred in 2003 but after the growing season (Q4; see fig. 2). These relate to the beginning of the swarming in very remote areas in the desertic areas of Northern Mali, fairly far from most of the inhabited settlements. In fact, only about 0.2 percent of observations are exposed to those events. We also show in our robustness checks that they do not influence our results.

A. Monthly Number of Swarm Events in Mali.

B. DHS Clusters, Locust Swarms and Local Markets in Mali.

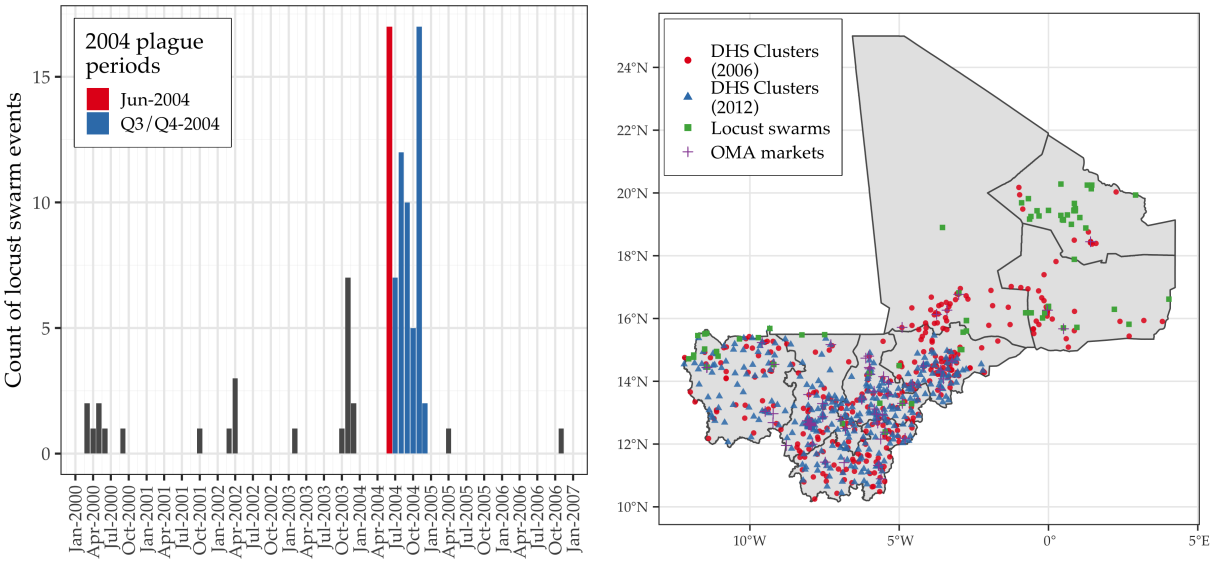

Figure 2: Monthly Number of Swarm Events (Panel A) and Locations of DHS Clusters, Locust Swarms and Local Markets (Panel B) in Mali.

Source: DHS waves of Mali from 2006 and 2012, SWARMS-FAO database, and OMA market prices.

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 to discern among treated and control areas. Our results are robust if considering only those that occurred during the growing season of 2004 (Q3-2004).

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.[21] The current political instability and terrorist threats in Mali started in 2012, long after our period of study.

Children Anthropometrics and Birth Cohort Data. Information on child health in Mali comes from the Demographic and Health (DHS) Surveys. The survey provides data on height-for-age Z-scores for children below age 5.[22] We use waves V and 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, including 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

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

[22] 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 because most respondents provide rough guesses from what they could recall.

young to have been exposed to it. In our main specification, we use information about children born before the 2006 DHS data collection period. Data from the 2012 wave are used in robustness and placebo tests. We define the in-utero period of each child by inferring their conception period as 9 months before the year-month information available in the DHS survey. Moreover, we enrich our set of variables available by linking each child to her household characteristics.

To have a broad picture of the socio-economic conditions of our sample, Table A.1 provides descriptive statistics of children, mothers, households, and clusters characteristics. Panel B of Figure 2 plots 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. Besides, Tables A.2 and A.3 compare children, mothers, and household characteristics between treated and control groups and find no remarkable differences between the two groups.[23]

Crop Price Data. We use 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 four cereal crops: maize, millet, sorghum, and rice (the latter is available for 4 different subvarieties). The data covers 74 local markets in Mali, shown in Figure 2 Panel B, from 2000 to 2015. We link them to DHS clusters if located in the same PRIO-GRID cell, and analogously so for the weather data (SPEI index).

Table A.4 documents the descriptive statistics of the OMA data. It contains 51,166 data points and, on average, a market-crop series covers about 9 years. About 24 percent of the markets are located in treated locations and are, on average, 1.6 hours distant from a large town.

Additional Spatial Data. We use the coordinates of our dataset to add more information to it. In particular, we match DHS clusters to geographical information from the PRIO-GRID dataset (Tollefsen et al., 2012), whose statistical unit is spatial cells of 0.5×0.5 degrees (hereafter PRIO-GRID cells). Analogously, we collect weather data from the Standardized Precipitation-Evapotranspiration Index (SPEI Vicente-Serrano et al., 2010), a spatial (0.5×0.5 degrees), multiscalar drought index that considers the joint effects of precipitation, potential evaporation, and temperature. With it, we account for 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.[24]

[23] An area is treated if it was exposed to at least one locust event within 30 km during the plague in 2004.

[24] In particular, we retrieve weather data for both OMA markets and children in DHS clusters. For

We complement our data with information on transportation networks and access to large cities and markets from the Global Accessibility Map project (GAM; Uchida and Nelson, 2010). This is a high-resolution raster data with information on the travel time to the nearest 50,000 inhabitants town in 1km^2 geographical units, in 2000. We match the DHS clusters to it by overlaying the latter onto the former.

4 Locust Plague and Local Crop Prices

We investigate the impact of the locust plague on crop prices with a Difference-in-Differences identification strategy. The timing of this price effect is particularly important given that the speculative channel described in Section 2.1 should kick in right away when the swarm events occur and before the harvest of October/November 2004. We provide evidence of the existence of this speculative price inflation effect by comparing the evolution over time of crop prices across treated and non-treated areas in an event study framework. In particular, we use OMA crop prices (years 2001-2006) for maize, millet, rice, and sorghum as the dependent variable in the following specification:

$$\log(p_{m(c,t)}) = \sum_{j=2003Q4}^{2006Q1} \theta_j T_m \times Q_j(t) + X_{m(t)}\beta + \mu_m + \delta_{c,t} + \xi_r \times t + \epsilon_{m(c,t)}, \qquad (1)$$

where $\log(p_{m(c,t)})$ stands for market m's (log) price of crop c, in period t (month-year). T_m is a treatment dummy equal to 1 if market m lies within 30 km from a locust swarm event from the 2004 invasion. We interact it with $Q_j(t)$, a dummy for each quarter around the plague period, starting with the first quarter after the harvest of 2003 (2003Q4) until the quarter of birth of the youngest cohort of children in our main sample (2006Q1). We account for market fixed effects (μ_m) and crop $\times$ year-month fixed effects ($\delta_{c,t}$). We also control for weather conditions (SPEI index) in quarter t and $t-1$ ($X_{m(t)}$) and region-specific time trends ($\xi_r \times t$). Standard errors are clustered at the market level.

Figure 3 depicts the point estimates and 95% confidence bands of each θ_j. It shows that the plague gave rise to sharp surges in the prices of crops sold in markets impacted by the invasion. The peak of the locust invasion (2004Q3; see Figure 2 Panel A) coincides with the peak of the increase in local crop prices. During that period, crop prices in exposed markets are approximately 8.4% higher than in non-exposed markets. There is an analogous effect during the second quarter of 2004, but smaller

the latter, Appendix A.2.2 describes how we construct an index of in-utero weather shocks using the SPEI index.

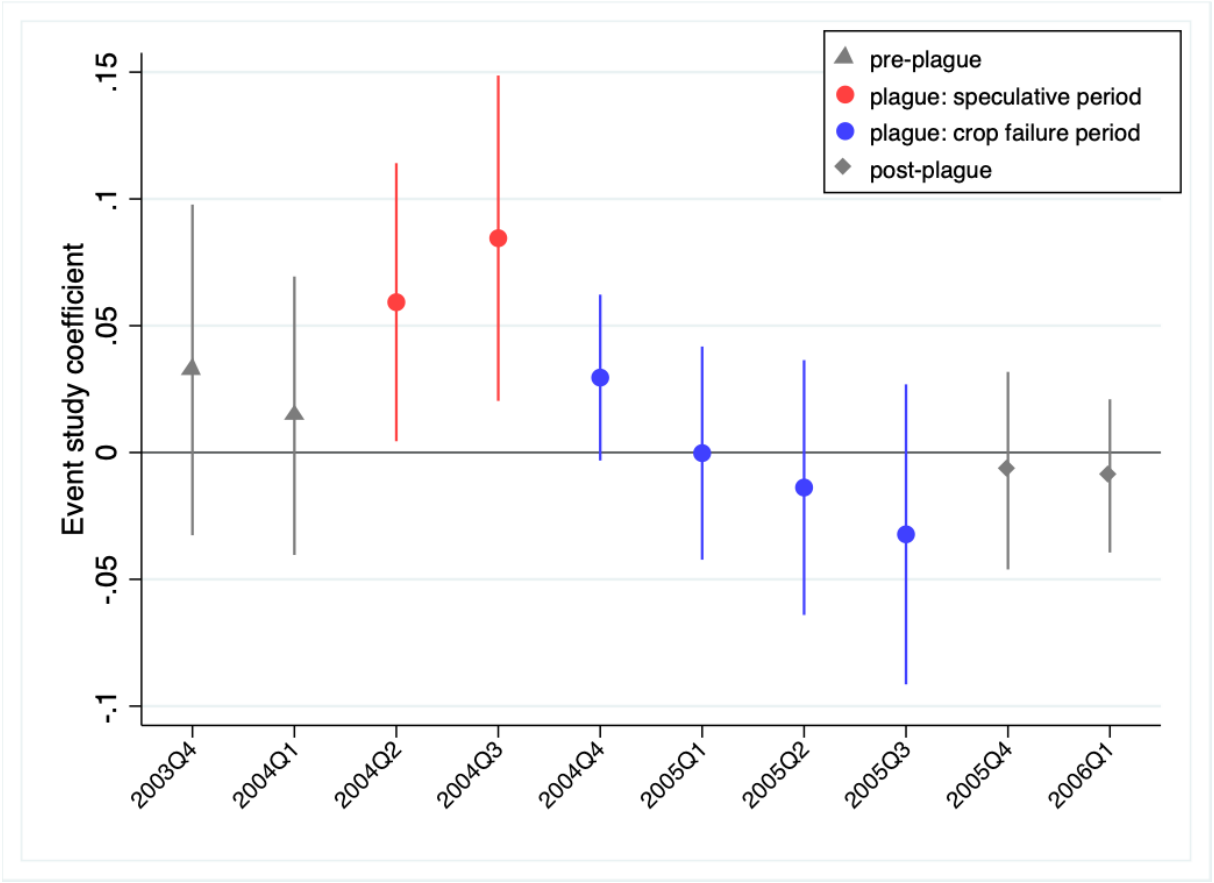

Figure 3: Impact of Locust Plague on Crop Prices.

Note: Estimated θ_j coefficients and 95% confidence bands of the impact of locust invasion on crop prices (maize, millet, rice, and sorghum) between 2003Q4 and 2006Q1. The gray triangles and diamonds mark respectively the quarters before and after the locust plague. The red dots mark the speculative period. The blue dots mark the crop failure period.

in magnitude because only one month (June) out of three has been impacted by the plague in this quarter (see Figure 2 Panel A). The plague has no detectable effect on crop prices in exposed markets after the impacted harvest of 2004 (crop failure period in blue) and after the next harvest in 2005. Figure 3 also shows that there is no differential pre-trend between treated and non-treated areas before the plague. The estimated difference is close to zero for the first two quarters after the harvest of 2003 (2003Q4 and 2004Q1) and this was the last harvest gathered before the plague.

Table A.7 documents analogous estimates averaged during the whole speculative period (June to September 2004) and the crop failure effect period (October 2004 to September 2005). Column (1) shows the average effects for a basic specification with crop and time fixed effects separately (thus without region-specific trends and crop-time fixed effects). Column (2) adds the region-specific trends and column (3), our preferred specification, adds crop-time fixed effects. The estimated coefficients are

relatively stable across all specifications, and the underlying message is sound: crop prices in exposed markets are higher during the speculative period, and not so afterward.[25]

5 Locust Plague and Child Health

5.1 Empirical Strategy

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

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

where i denotes a child belonging to cohort t (year-month), born in household h who lives in enumeration area e.[26] The dependent variable $y_{i(h,t,e)}$ measures the 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. This means being born in a locust-affected area between July 2004 (shortly after the plague outbreak) and June 2006 (the last cohort that relied on the 2004 harvest while in utero).[27]

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 the individual level (gender, birth order among siblings, age gap with direct older and younger siblings, etc.) and enumeration area level (weather conditions from the SPEI drought index), respectively. X_h is a vector that includes household-level controls, such as gender and

[25] As for robustness, Column (4) of Table A.7 clusters standard errors at the PRIO-GRID level rather than the market level, and Column (5) uses the entire price sample (from 2000 to 2015) rather than only 2001 to 2006, the relevant period for the children in our main sample.

[26] We omit from the notation, for neatness, the grid-cell g where enumeration area e lays over (see section 3). When estimating model (2) with the two DHS waves, we replace the cluster fixed effects with grid cell fixed effects to compare geographical units consistent across waves. In that case, we add to (2) a dummy for treatment status at the cluster e level.

[27] The first locust events happened in June so children born in July 2004 are the first cohort of children potentially exposed to locusts for at least one month in utero. We expect to see detectable effects of the plague starting from this cohort and we show below that this is the case. The youngest children used in our main sample were born in March 2006.

age of household head, wealth index of household, and mother education. Standard errors are clustered at the PRIO-GRID cell level.[28]

Our coefficient of interest is γ, which measures the average difference in changes of height-for-age Z-scores for 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.

5.2 Results

Average Effect. Table 1 displays the point estimates of γ in Equation 2. 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. The estimated impact is negative and statistically significant. The estimate is robust and remains significant after progressively adding controls for child 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 0.426 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.[29] The estimates of the impact of locust plague on height-for-age Z-scores and stunting rates are also robust to the inclusion of region-specific time trends (columns (4) and (6)). Table A.10 shows no detectable effect of the plague on neonatal mortality (death less than a month after birth) and infant mortality (less than a year after birth).

Le and Nguyen (2022) document that exposure to swarm events decreases height-for-age by 0.16 standard deviations, about one-third of our estimate (-0.426). This

[28]Clustering standard errors in coarser units than our treatment makes the results more conservative. However, we do test our specification if setting both treatment and clustering at the same unit: grid cells of 30 km. We find no qualitative changes in the results; see column (3) of Appendix Table A.9. Moreover, we follow related literature and we do not use sample weights in the baseline regressions (Adhvaryu et al., 2019; Akresh et al., 2012b,a; Kudamatsu, 2012; Rieger et al., 2019; Valente, 2015). We do check for the robustness of our results if using sample weights and find no qualitative changes; see column (5) of Appendix Table A.8.

[29]Stunting rates stand for the share of children whose height-for-age Z-score lies below 2 standard deviations.

Dependent variable	Height-age Z-score				Stunted	
	(1)	(2)	(3)	(4)	(5)	(6)
In utero treatment	-0.477***	-0.436***	-0.426***	-0.434***	0.074**	0.079***
	(0.112)	(0.112)	(0.110)	(0.091)	(0.030)	(0.023)
Observations	9,194	9,194	9,194	9,194	9,194	9,194
R-squared	0.200	0.220	0.238	0.244	0.183	0.189
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	-1.422	-1.422	-1.422	-1.422	0.364	0.364

Table 1: Impact of Locust Plague on Child Health

Note: All regression include cohort fixed effect (FE) and location FE. 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. Household survey data comes from 2006 DHS wave in Mali. Robust standard errors in parentheses are clustered at PRIO cell grid level. *** p<0.01, ** p<0.05, * p<0.1.

difference in magnitude is due to two main reasons. First, we focus on Mali, one of the poorest countries in Africa while Le and Nguyen (2022) look at several countries with most of them being less vulnerable to pest invasions. These countries also have better child nutrition and health levels. The average impact over this larger sample will therefore give a much smaller effect. Second, we also focus on an acute plague episode while Le and Nguyen (2022) look at exposure to any swarm event including very minor and/or isolated events that did not pose a threat to agricultural production.[30]

Treatment Radius and Impact across Space. We show that the estimated effect is not sensitive to the radius of 30 km used in our main specification. To do so, we first run several alternative specifications of our baseline model of Equation (2) with different distance thresholds for the *treatment group*. We start by defining an enumeration area e as treated if within 20 km from at least one swarm event during the 2004 plague. We then progressively increase it by 2 km up to 70 km. Figure A.4 plots the point estimates and 95% confidence bands of γ for each distance threshold. It shows that the effects of locust invasions on child health are both negative and statistically significant for thresholds up to 40 km. After that, they approach zero and lose significance. This provides evidence for the adverse *local* effects of the locust invasions, which is corroborated in Figure A.5. There, we define as non-treated the enumeration areas at least 60 km away from any swarm event. We then split the treated areas into 6 groups of 10 km distance bins.[31] The estimated effects are negative, significant, and

[30]The magnitude of our estimates remains smaller than what is reported in other related studies in the literature. For instance, Akresh et al. (2011) estimate that a crop failure episode in Southern Rwanda led exposed girls to be, on average, 0.855 standard deviations shorter. They also find similar magnitudes for in-utero exposure to the civil war for both boys and girls.

[31]Thus, there are six different treatment dummies, the first one for exposed children of households

fairly constant if the impacted child belongs to households located within the first 30 km from locust plague events. Thus, 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 estimated coefficient fades out after 30 km, confirming the idea that locust invasions have local effects.

Robustness Checks. In Tables A.8 and A.9, we present the results of robustness checks performed on alternative empirical specifications and data samples. Column (1) of Table A.8 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 point estimate decreases in magnitude (-0.337) but remains statistically significant ($p<0.01$). Importantly, its sample size differs from the baseline because the variation that identifies the treatment effects in this specification comes only from the children whose mothers had at least two children sampled by the DHS survey. As a reference, column (2) reports the result of the baseline specification restricted to that subsample, and the magnitude of the estimated effect is very similar to the one of column (1).[32] Columns (3), (4), (5), and (6) show the robustness of the main results presented in Table 1 when using, respectively, household fixed effects, survey month fixed effects, survey sample weights, and spatial HAC standard errors (Conley, 1999).

In Table A.9, columns (1) and (2) exclude children that are born respectively in 2003Q4 and in June 2004. The estimated impact remains stable and this confirms that these children play a minor role in our sample, irrespective of whether they were affected by the locust events that happened in these periods or not. Column (3) redefines the treatment (and clusters standard errors) at grid cells of 30 km. Columns (4), (5), and (6) refer to, respectively, treatment at the municipality level, including events that occurred beyond Mali country borders, and using data from both 2006 and 2012/2013 DHS waves. Column (7) uses both survey waves but restricts the sample to children born after the plague outbreak. This means that treated children belong to the older cohorts and younger children are not treated.[33]

Finally, column (8) checks that the estimated effect does not capture any pre-existing time trends between treated and non-treated areas with a placebo test. We use only data from the 2012/2013 survey and the placebo treatment is a dummy for children born in the locust-affected areas after 2010. The estimated coefficient is small,

located at most 10 km away from locust events, the second for children from households located between 11 and 20 km away from locust events, and so forth.

[32] In particular, we repeat the specification of Equation (2) with child and family controls; i.e. the results of Table 1, column (3).

[33] 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.

positive, and not significant.

5.3 Timing of the Estimated Effect and Further Placebo Tests

The fact that the 2004 locust invasion impacted Mali within a relatively narrow time window allows us to investigate the effect of such shock on different cohorts of children. As discussed in Section 2, 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. Specifically, we run the following regression using the 2006 DHS wave:

$$y_{i(h,t,e)} = \sum_{j=2003Q1}^{2006Q1} \gamma_j L_{i(e)} \times Q_j(t) + \mu_e + \beta_t + \theta_{hh} X_h + \theta_1 X^{(1)}_{i(h,t,e)} + \theta_2 X^{(2)}_{i(t,e)} + \epsilon_{i(h,t,e)}, \quad (3)$$

where $L_{i(e)}$ is a dummy for being born in a treatment area. We interact it with $Q_j(t)$, a dummy for each quarter of birth around the period of exposure to the shock for the youngest children in this survey (2003Q1 to 2006Q1). Moreover, we estimate an analogous regression with the 2012 DHS wave but focus on the oldest children of the 2008 cohorts (i.e. those born the closest to the exposure period of the plague):[34]

$$y_{i(h,t,e)} = \sum_{j=2008Q1}^{2008Q4} \gamma_j L_{i(e)} \times Q_j(t) + \mu_e + \beta_t + \theta_{hh} X_h + \theta_1 X^{(1)}_{i(h,t,e)} + \theta_2 X^{(2)}_{i(t,e)} + \epsilon_{i(h,t,e)}. \quad (4)$$

In both eqs. (3) and (4), γ_j is the coefficient of interest. It measures the average differences in height-for-age Z-scores between treated and control children of a specific quarter j cohort. Moreover, we separate cohorts into three groups based on when their in-utero life happened: (i) before the plague (2003Q1-2004Q2 cohorts), (ii) during the plague (2004Q3-2006Q1 cohorts), and (iii) after the plague (2008 cohorts). Their point estimates and 95% confidence bands are shown in Figure 4 with triangles, circles, and diamonds, respectively.[35]

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

[35]Note that this setting, which compares offspring conceived both before the plague outbreak, during the outbreak, and after it, does not address possible selectivity in fertility mechanisms put in place after the plague onset. In Figure A.6, we explore whether net of seasonality in births, there is no systematic difference in conception patterns between poor and rich households. In this perspective, we perform a Difference-in-Difference analysis along the lines of Equation 3, where the dependent variable is a children-level dummy for being born in a household with a rich or medium wealth level. Our results suggest that our estimates are not inflated or deflated by selectivity in fertility mechanisms. Furthermore, we do not find evidence that the plague impacted cohort size, e.g., through abortion or stillbirth. Finally, since our data come from waves V and VI of the DHS survey, whose interviews took

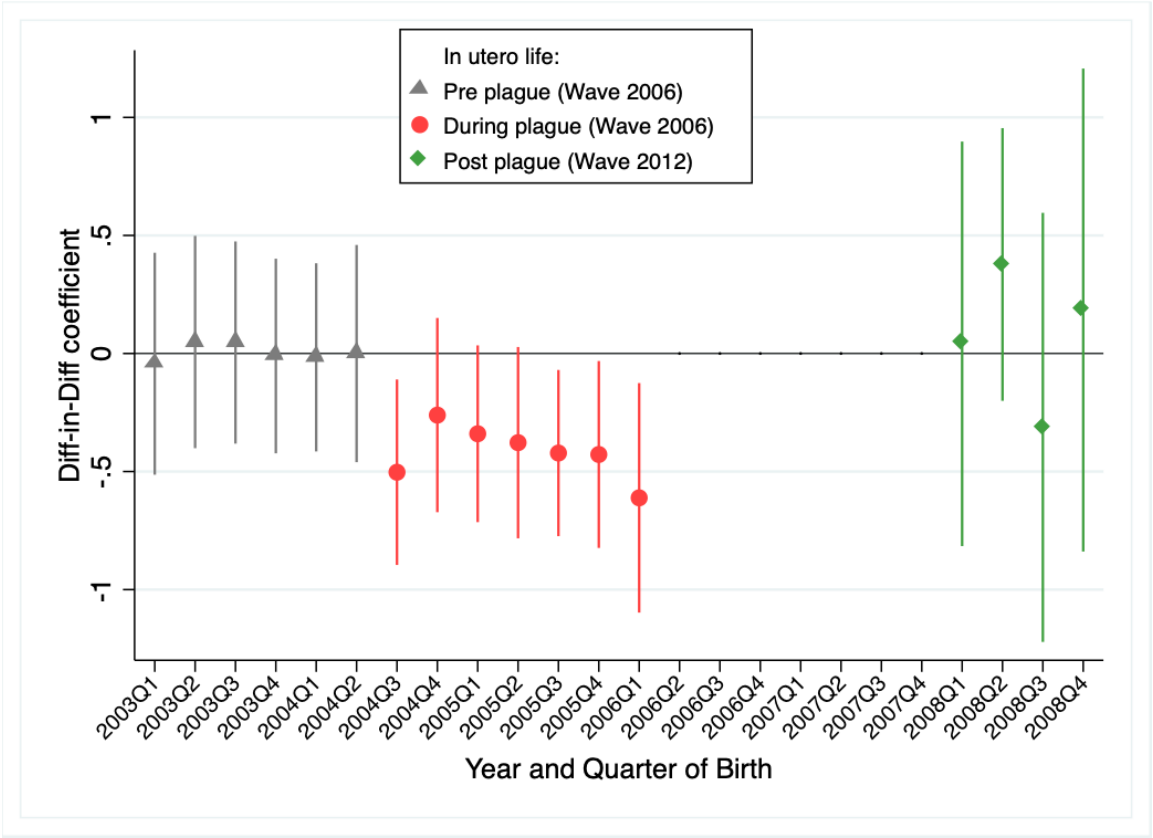

Figure 4: Timing of the Impact.

Note: 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 grey triangles refers to children born before the plague onset. The red dots denotes children that are exposed to the plague while in utero. The grey diamonds corresponds to children born after the plague ended.

First, we find no systematic differences in height-for-age Z-score between treated and control children of the pre-plague cohorts. Thus, the children that were already born when the plague hit did not suffer any perceptible effect in their height-for-age Z-scores.[36] Importantly, being able to identify the post-natal effects of the plague (which are negligible) allows us to pin down the distinct effects of the in-utero exposure to the plague phases. Moreover, the pre-plague results rule out possible pre-existing trend differences between treated and non-treated areas in the average results shown before. Finally, the clear drop in the γ_j coefficients from 2004Q3 to 2006Q1 corroborates our previous finding that all the cohorts of children exposed in utero to the plague, for at least a month, suffered sizeable health setbacks.

The effect of the plague turns negative already for children of the first plague co-

place in 2006 and 2012/13 respectively, we are not able to find short-term maternal health indicator that corresponds to the time periods right before, during, and right after the plague. Therefore we are not able to check whether adult health might have been adversely affected by the plagues too.

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

hort, 2004Q3. According to the seasonal calendar in Figure 1, their mothers were still relying on the unharmed harvest collected in 2003. Thus, this evidence suggests that the negative impact of the plague was driven by speculations about the agricultural campaign that was starting. This cohort was exposed also to the crop failure effect, but only after birth, and as argued previously, we find no after-birth plague effects for older cohorts (see pre-plague cohorts in Figure 4). 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. To the best of our knowledge, such differential sensitivity to early life shocks has never been documented in the literature. Moreover, this unlikely alternative story cannot explain the substantial plague-induced crop price inflation that happened during the third quarter of 2004 (see Section 4). We also show in Section 5.4 that, when we account for local price effects, the estimated impact for treated children of the 2004Q3 cohort (exposed only to the speculative price effect) drops significantly.

The mothers of the children born between October 2004 and March 2006 were relying on the harvest of the 2004-2005 season, which was damaged by locust swarms. 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/anticipatory effect and the crop failure effect. Those born after June 2005 are exposed only to the crop failure effect.

Figure 4 also shows no effects on treated children of the post-plague younger cohorts of 2008, born after the plague. Those children were 4 to 5 years old at the time of the survey when they were measured. Thus, their related γ_j coefficients are noisier and more volatile. Nevertheless, there is no evidence of a clear trend and the point estimates oscillate around zero.[37] 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.

5.4 Channels of the Estimated Effect and other Robustness

We explore in detail the mechanisms through which the locust plague led to the estimated health setbacks. We have shown that the plague's impact spreads over

[37] We show in column (8) of Table A.9 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.

entire localities by affecting their local markets of reference (Figure A.5) and that the plague gave rise to sharp surges in the prices of crops sold in the impacted markets (Section 4). Thus, we first look at the impact of the plague on child health accounting for price effects. Next, we study the importance of local crop market isolation on the impact of locust plagues on child health.

5.4.1 Price Inflation Channel

We use the three different exposure time windows introduced in Section 2 and 5.3, together with data on local prices, to investigate the importance of different channels through which locust plagues impact child health. To do so, we split the treatment dummy of Equation (2) into three in-utero exposure groups: (i) in-utero exposure to the speculative/anticipatory effect (children born in 2004Q3), (ii) in-utero exposure to both effects (children born between October 2004 and June 2005), and (iii) in-utero exposure to the crop failure effect (children born between July 2005 and March 2006).[38]

The estimation results, reported in Table 2 column (1), show that the health setbacks implied by the locust invasion are statistically significant and similar in magnitude during the three periods of exposure. This is consistent with the stability of the estimated impact for the treated cohorts shown in Section 5.3.

In column (2) we run the same specification of column (1) on the sub-sample of children matched to market price data. The sample decreases because not all DHS clusters have an OMA market nearby to be matched to in the price dataset (see Section 3). 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 0.66 points lower than non-exposed children. This effect is close in magnitude to that of children exposed to both effects (-0.52) and that of children exposed only to the crop failure effect (-0.73).

Next, we check whether the negative plague effects during the speculative/anticipatory period are driven by local inflation. We do that by controlling for average local prices during pregnancy in column (3).[39] The comparison between column (2) and column (3) points to a drop in 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 -0.66 to -0.35, whereas the coefficients of the other two effects remain un-

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

[39] As explained in Section 3, to control for average local prices during pregnancy, we first match each DHS cluster to the closest market located in the PRIO-GRID cell where the DHS cluster belongs to. Then, 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.

	Panel A			Panel B		
	(1)	(2)	(3)	(4)	(5)	(6)
In utero treatment with speculative / anticipatory effect	-0.509***	-0.659**	-0.348	-0.507***	-0.528*	-0.236
	(0.176)	(0.263)	(0.256)	(0.169)	(0.272)	(0.267)
In utero treatment with both	-0.355***	-0.515**	-0.502**	-0.336**	-0.356	-0.330
	(0.125)	(0.197)	(0.202)	(0.142)	(0.258)	(0.270)
In utero treatment with crop failure effect	-0.490***	-0.732***	-0.757***	-0.489***	-0.528**	-0.537**
	(0.127)	(0.139)	(0.139)	(0.149)	(0.210)	(0.225)
Observations	9,194	2,859	2,859	9,194	2,859	2,859
R-squared	0.244	0.268	0.273	0.238	0.257	0.263
Region specific time trend	YES	YES	YES	NO	NO	NO
Control for prices	NO	NO	YES	NO	NO	YES
Mean dependent variable	-1.422	-1.268	-1.268	-1.422	-1.268	-1.268

Table 2: Impact of Locust Plague on Child Health: Price Inflation Channel

Note: All regressions include location FE, cohort FE, child and family characteristics: 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.

changed. Importantly, we reject the equivalence of the speculative/anticipatory effect coefficients between columns (2) and (3) (p-value equal 0.02).[40] Panel B of Table 2 shows that all the results in Panel A are robust to the exclusion of region-specific time trends.[41] 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 0.76 and 0.55). This suggests that price inflation accounts for a significant part of the speculative/anticipatory effect.

As expected, the speculative/anticipatory effect does not vanish completely after controlling for prices. This corroborates the prior that other factors, on top of the price inflation, determine the speculative/anticipatory effect (see Section 2). These factors could be a precautionary decrease in household consumption in anticipation of future shocks or in-utero exposure to maternal stress due to the news of a future harvest failure.

5.4.2 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

[40]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.

[41]The analogous Hausman tests comparing the coefficients in columns (5) and (6) convey p-values equal to 0.03 for the speculative/anticipatory effects, and 0.63 and 0.88 for the remaining two effects.

	Height-age Z-score		
	(1)	(2)	(3)
Treatment with speculative/anticipatory effect	-0.461**	-0.470**	-0.458*
	(0.218)	(0.222)	(0.237)
Treatment with both	-0.260*	-0.261*	-0.250*
	(0.141)	(0.142)	(0.129)
Treatment with crop failure effect	-0.440***	-0.431***	-0.413***
	(0.143)	(0.145)	(0.129)
Treatment with speculative/anticipatory effect × Travel time	-0.059	-0.052	-0.041
	(0.082)	(0.082)	(0.086)
Treatment with both × Travel time	-0.133***	-0.132***	-0.112***
	(0.017)	(0.020)	(0.033)
Treatment with crop failure effect × Travel time	-0.172***	-0.177***	-0.154***
	(0.027)	(0.029)	(0.042)
Observations	6,479	6,479	6,479
R-squared	0.239	0.240	0.244
Household wealth	NO	YES	YES
Region time trends	NO	NO	YES
Mean dependent variable	-1.607	-1.607	-1.607

Table 3: Impact of Locust Plague on Child Health and Level of Isolation of Rural Areas

Note: All regressions include location FE, cohort FE, child and family characteristics: 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. Robust standard errors in parentheses are clustered at PRIO Cell grid level. *** p<0.01, ** p<0.05, * p<0.1.

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).

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's coordinates 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).[42] 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,

[42]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.

i.e., the sub-sample of households located in rural areas. We build on the specification used in column (1) of Table 2 by adding the interaction terms between travel time and the three treatment variables. Results are reported in Table 3. 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, for children exposed only to the crop failure effect, we find that a one standard deviation increase of travel distance to the nearest town implies an average decrease in height-for-age Z-scores of 0.17 on top of the baseline effect (0.44 decrease in Z-score). For children exposed to the crop failure effect and the speculative/anticipatory effect, we find that a one standard deviation increase of travel distance implies an extra decrease in height-for-age Z-score of 0.16 (baseline effect equal to 0.26 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 of household wealth index (column (2)) and region-specific time trends (column (3)).[43] Overall, this suggests that isolation itself plays a role and that results are not likely to be biased by more isolated households being potentially poorer, and 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, 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 happen because economic agents, like intermediaries and/or consumers, over-predicted the impact or the potential magnitude of the shock during the growing season, at least in non-isolated areas.[44]

5.4.3 Heterogeneity: Migration, Rural-Urban Residence, Gender, and Exposure to Sprayed Pesticides

We explore the heterogeneity of our results with respect to migration, urban/rural residence, and gender by estimating an analogous regression model of Table 2 with subsamples restricted along those dimensions (See Table A.5 and Appendix A.4.1 for further details). First, we find that our results are robust to restricting our sample

[43] We reject the statistical equivalence of the interaction terms in all of these specifications (the maximum p-value is 0.0018).

[44] We test the robustness of these results using Euclidean distance (in kilometers) instead of travel distance (Table A.11). Results do not change qualitatively.

to people that have not migrated from their place of birth. Second, we find that the estimated effect is present for both boys and girls sub-samples. We also show that treated children living in both urban and rural areas suffer major health setbacks.[45]

Finally, in Appendix A.5 we rule out the hypothesis that our results are driven by the exposure to anti-locust pesticides carried out by spraying campaigns based on two reasons. First, most of the anti-locust spraying during the plague was conducted with ground spraying, which is relatively harmless if compared to airplane spraying.[46] Second, we find no differences between the estimates for sub-samples exposed to different intensities of anti-locust spraying.[47]

6 Concluding Remarks

This paper shows the importance of markets in understanding the impact of agricultural shocks on child health. 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/anticipatory effect suffered as much as those exposed to the actual crop failure effect. Moreover, market openness dampens the adverse health effects of exposure to crop failure effect.

We believe these results have 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 any type of anticipation that might have adverse effects on time-sensitive investments; (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 to deal with in the coming years.

[45] Table A.6 documents the estimation results using a unique dummy variable for treatment, analogously to Table A.5

[46] Intuitively, air spraying is more harmful because it consists of deploying much larger quantities of pesticides that can be potentially carried around by the wind. Ground spraying, if harmful, affects the spray operators. Thus, it should not affect most pregnant women in locust-exposed locations.

[47] For instance, we find that anti-locust spraying takes place mostly in rural areas. That suggests that spraying cannot be the main driver of our results, as we do find comparable treatment effect patterns for treated children in both rural and urban households. In the same spirit, more isolated rural areas are less likely to be exposed to spraying and yet our estimated effects are stronger in these areas. We convey these arguments in detail in Appendix A.5.

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Appendix

A.1 Additional Figures

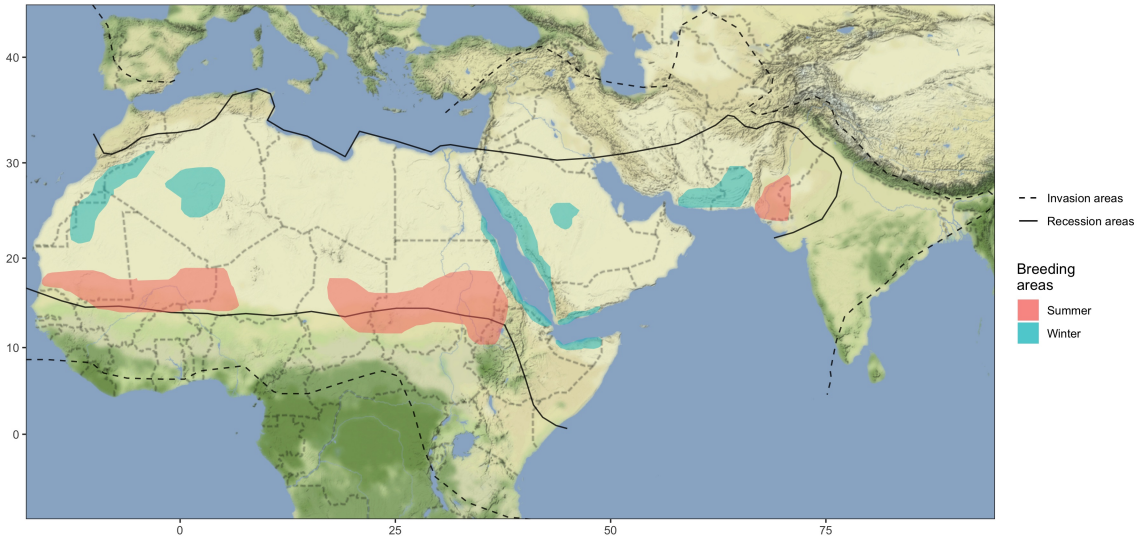

Figure A.1: Desert Locust Breeding and Invasion Areas (based on Cressman and Stefanski (2016)).

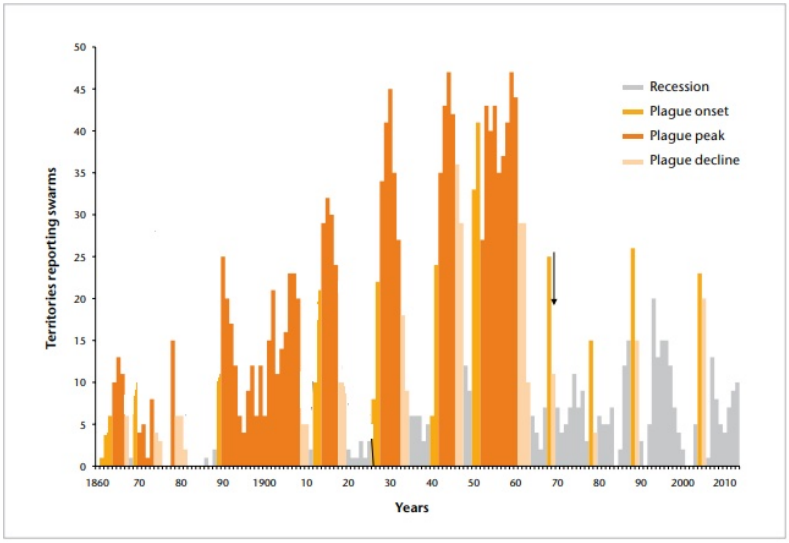

Figure A.2: History of Locust Plagues (based on Cressman and Stefanski (2016)).

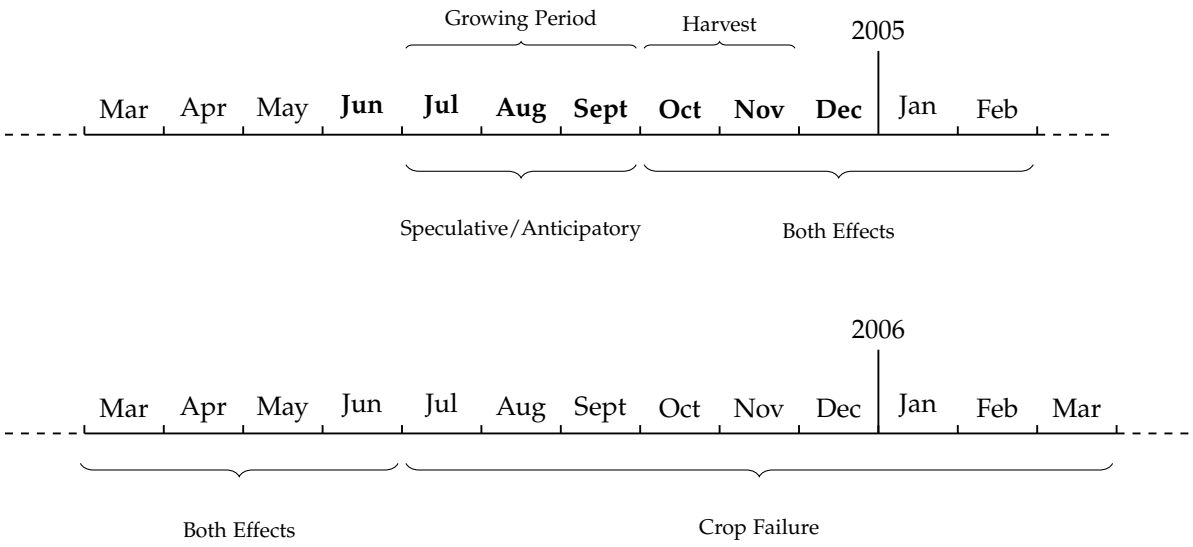

Figure A.3: Seasonal Agricultural Calendar in Mali and Exposure of Cohorts to the 2004 Locust Plague by Type of Effect.

Notes: The timeline starts in March 2004 and illustrates the temporal overlapping of the 2004 plague and the agricultural calendar in Mali as of FEWS-NET (2005). The plague took peaked during the second semester of 2004 (months in boldface), coinciding with the growing and harvest seasons of 2004. The underbrackets stand for the type of effects the covered cohorts were exposed to while in utero. The June 2004 cohort is not considered here given the little time that they were exposed in utero.

A.2 Data

A.2.1 Swarm Data Collection by SWARMS System

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.

A.2.2 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 Descriptive Statistics and Balance Tables

	Obs.	Mean	St. Dev.	Min	Pctl(25)	Median	Pctl(75)	Max
Children characteristics								
Height (z-scores)	9,194	−1.42	1.79	−5.99	−2.59	−1.44	−0.35	6.00
Treated	9,194	0.15	0.36	0	0	0	0	1
Age in years	9,194	2.06	1.36	0	1	2	3	4
Female	9,194	0.49	0.50	0	0	0	1	1
Gap from previous birth	9,194	2.77	1.42	0	2	2	4	5
Gap to next birth	9,194	3.95	1.54	0	2	5	5	5
SPEI[1]	9,194	−0.15	0.37	−1.55	−0.44	−0.14	0.13	1.24
Respondent (Mother) charact.								
Height (meters)	6,968	1.61	0.07	1.00	1.57	1.61	1.65	1.92
Education (years)	6,968	0.93	2.52	0	0	0	0	17
Years living in the cluster	6,968	26.23	15.12	0	10	39	39	39
Measured children	6,968	1.32	0.50	1	1	1	2	4
Household characteristics								
Household members	6,195	6.82	3.30	2	4	6	8	29
Age household head	6,195	41.77	12.14	16	33	40	49	98
Female household head	6,195	0.08	0.27	0	0	0	0	1
Measured children	6,195	1.48	0.70	1	1	1	2	6
Poor Dweling[2]	6,195	0.39	0.49	0	0	0	1	1
Number Interviewed Women	6,195	1.12	0.36	1	1	1	1	4
Clusters characteristics								
Rural	386	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	386	16.05	4.57	4	13	16	19	31

Table A.1: Descriptive statistics: children, mothers, households, and clusters.

Notes: [1]SPEI stands for the weigthed average of the SPEI index during the period in utero; see appendix A.2.2 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.

Children characteristics	Control group	Treatment Group	Difference
Height–for–age Z–score	−1.431	−1.373	0.058
	(0.036)	(0.077)	(0.084)
Age in years	2.064	2.068	0.003
	(0.014)	(0.028)	(0.032)
Female	0.493	0.492	−0.001
	(0.006)	(0.012)	(0.013)
Gap from last birth (in years)	2.775	2.725	−0.051
	(0.021)	(0.048)	(0.052)
Gap to next birth (in years)	3.970	3.871	−0.098**
	(0.017)	(0.039)	(0.042)
Birth order in the family	1.376	1.405	0.029*
	(0.006)	(0.015)	(0.016)
SPEI[1]	−0.152	−0.135	0.017
	(0.009)	(0.023)	(0.025)
Observations	7,774	1,420	9,194

Table A.2: Balance table – differences in average children characteristics between treated and control groups.

Note: *p<0.1; **p<0.05; p<0.01. [1]SPEI stands for the weigthed average of the SPEI index during the period in utero; see appendix A.2.2 for details.

	Control group	Treatment Group	Difference
Respondent (mother) characteristics			
Height (meters)	1.610	1.620	0.010***
	(0.001)	(0.003)	(0.003)
Age in years	28.750	28.639	−0.111
	(0.117)	(0.231)	(0.257)
Years of education	0.878	0.933	0.055
	(0.064)	(0.140)	(0.153)
Age at first birth	18.191	18.311	0.119
	(0.069)	(0.125)	(0.142)
Births in the past 5 years	1.745	1.789	0.044
	(0.011)	(0.026)	(0.028)
Children ever had	4.513	4.545	0.032
	(0.050)	(0.101)	(0.112)
Total living children	3.698	3.775	0.077
	(0.036)	(0.076)	(0.084)
Years living in the cluster	26.120	26.625	0.506
	(0.381)	(0.910)	(0.980)
Sons living in the household	1.680	1.692	0.011
	(0.023)	(0.047)	(0.052)
Daughters living in the household	1.573	1.575	0.002
	(0.018)	(0.040)	(0.044)
Household/Cluster characteristics			
Household members	7.474	7.386	−0.088
	(0.085)	(0.170)	(0.189)
Age of household head	41.792	41.923	0.131
	(0.204)	(0.671)	(0.696)
Female household head	0.069	0.106	0.036**
	(0.005)	(0.016)	(0.017)
Number of children up to 5 years old	2.263	2.318	0.055
	(0.028)	(0.063)	(0.068)
Rural household	0.701	0.723	0.022
	(0.025)	(0.059)	(0.064)
Poor dweeling	0.405	0.363	−0.043
	(0.017)	(0.043)	(0.046)
Matched to an OMA market	0.456	0.556	0.100
	(0.029)	(0.070)	(0.075)
Distance to water (minutes)	7.534	7.743	0.209
	(0.295)	(0.700)	(0.755)
Travel distance to the nearest town (hours)	3.881	5.470	1.589
	(0.165)	(1.083)	(1.087)
Altitude (meters)	325.226	270.046	−55.180***
	(4.782)	(16.073)	(16.652)
Observations	7,774	1,420	9,194

Table A.3: Balance table – differences in average mother and household characteristics between treated and control groups.

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. [2] Dummy for whether the cluster is located close enough to an OMA market and thus have crop prices available.

	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[1]	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
Crops[2]								
Maize prices	8,092	151.01	42.84	40	125	150	175	343
Millet prices	10,612	167.27	51.85	41	131.2	167.3	200	350
Sorghum prices	10,075	157.17	58.04	40	125	152	187	3,630
Rice (local) prices	10,010	310.75	59.28	146	262.7	300	352	562
Rice (white) prices	5,052	300.21	88.85	125	250	275	325	1,138
Rice (red) prices	5,557	354.95	126.90	100	256	308.3	487.5	625
Rice (paddy) prices	1,768	150.16	41.44	68	125	145	167	400
Market–crop								
Length of series[3]	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[4]	432	−0.10	0.16	−0.67	−0.15	−0.08	−0.04	0.49
Average price[2]	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

Table A.4: Descriptive statistics of OMA prices: all sample, by crops, markets, and market–crops.

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]Prices in local currency. [3]Length in months. [4]Average SPEI during the previous 12 months of each observation.

A.4 Impact of Locust Plague: Robustness

A.4.1 Migration, Residence, and Gender

In this appendix, 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 2 on several sub-samples. Results are shown in Table A.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 in 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 A.5. Results are robust in both cases: the point estimates are statistically significant and range between -0.33 and -0.51 (column (1)) and between -0.49 and 54 (column (2)), close to the benchmark estimates (between -0.36 and -0.51; see Table 2). Thus, it suggests that our estimates are not affected by potential migration.

Furthermore, columns (3) and (4) 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 (3) and (4) of Table A.5 show, instead, a negative impact of the plague on children of both genders.[48] In both sub-samples, we cannot reject the statistical equivalence of the three treatment dummies (the F-test yields a p-value of 0.5 (0.15) for the male (female) sample). Table A.6 shows the equivalent average effects for Table

[48] In Table A.6 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 -0.35 to -0.53).

	(1)	(2)	(3)	(4)	(5)	(6)
	Years of residence		Gender		Place of residence	
	2 + years	Always	Male	Female	Urban	Rural
Treatment with speculative/anticipatory effect	-0.509***	-0.542**	-0.837***	-0.313	-0.643*	-0.514**
	(0.171)	(0.223)	(0.269)	(0.346)	(0.369)	(0.208)
Treatment with both	-0.325**	-0.518***	-0.498**	-0.163	-0.325	-0.347**
	(0.149)	(0.174)	(0.208)	(0.173)	(0.212)	(0.157)
Treatment with crop failure effect	-0.464***	-0.485***	-0.461**	-0.544***	-0.294	-0.589***
	(0.147)	(0.180)	(0.186)	(0.197)	(0.245)	(0.181)
Observations	8,832	5,009	4,663	4,531	2,715	6,479
R-squared	0.240	0.267	0.269	0.299	0.203	0.238
Mean dependent variable	-1.428	-1.447	-1.481	-1.361	-0.981	-1.607

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

Note: All regressions include location FE, cohort FE, child and family characteristics: 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.

A.5 without distinguishing between the 3 treatment phases.

Finally, columns (5) and (6) 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 magnitudes are smaller in urban areas, in line with our results on the level of isolation of local markets, but this heterogeneity is not statistically significant. The reduced sample size for the urban sample affects the precision of the estimates in this sub-sample.[49]

	(1)	(2)	(3)	(4)	(5)	(6)
	Years of residence		Gender		Place of residence	
	2 + years	Always	Male	Female	Urban	Rural
In utero treatment	-0.410***	-0.508***	-0.534***	-0.348**	-0.343*	-0.471***
	(0.109)	(0.129)	(0.136)	(0.153)	(0.173)	(0.127)
Observations	8,832	5,009	4,663	4,531	2,715	6,479
R-squared	0.240	0.267	0.269	0.298	0.205	0.238
Mean dependent variable	-1.428	-1.447	-1.481	-1.361	-0.981	-1.607

Table A.6: Heterogeneity: Average Impact of Locust Plague on Child Health

Source: All regressions include location FE, cohort FE, child and family characteristics: 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. 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. Robust standard errors in parentheses are clustered at PRIO Cell grid level. *** p<0.01, ** p<0.05, * p<0.1.

[49]Columns (5) and (6) of Table A.6 documents the estimation results using a unique dummy variable for treatment, analogously to Table A.5.

A.4.2 Additional Robustness

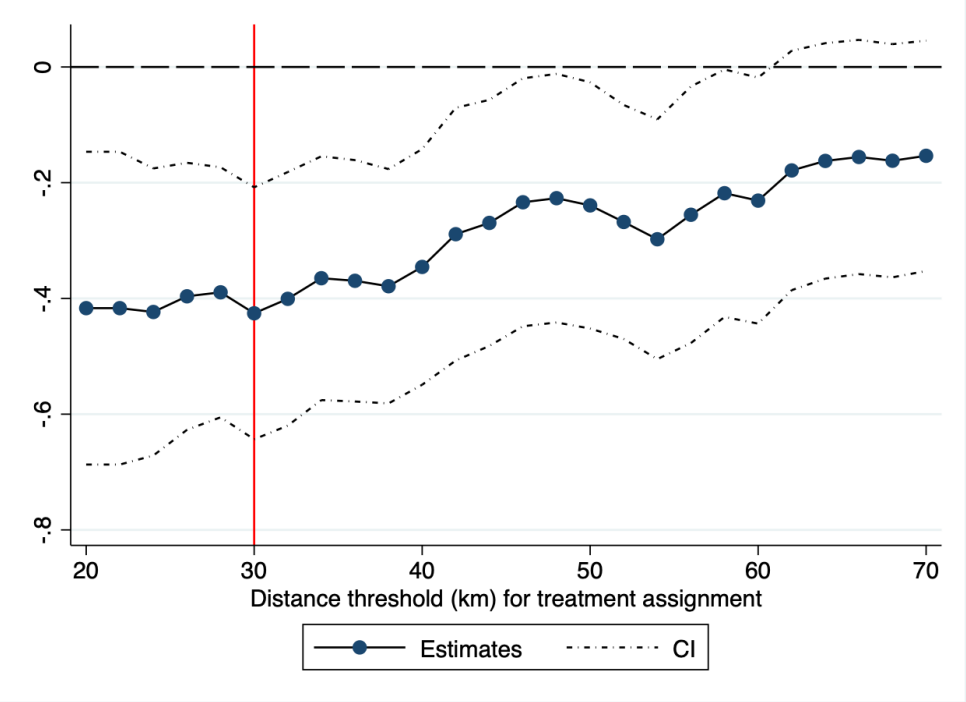

Figure A.4: Local Impact of Locust Plague on Child Health

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 2) together with their 95% confidence bands.

	Log crop price				
	(1)	(2)	(3)	(4)	(5)
Treated × speculative period	0.061*	0.066*	0.076***	0.076**	0.083***
	(0.033)	(0.033)	(0.028)	(0.029)	(0.028)
Treated × crop failure period	-0.005	0.003	-0.007	-0.007	-0.001
	(0.021)	(0.023)	(0.021)	(0.027)	(0.017)
Observations	21,128	21,128	21,128	21,128	51,166
R-squared	0.839	0.840	0.911	0.911	0.913
Market FE	YES	YES	YES	YES	YES
Crop FE	YES	YES	NO	NO	NO
Year-month FE	YES	YES	NO	NO	NO
Region specific time trend	NO	YES	YES	YES	YES
crop-year-month FE	NO	NO	YES	YES	YES
Mean dependent variable	5.139	5.139	5.139	5.139	5.306

Table A.7: Impact of Locust Plague on Crop Prices

Note: 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. Robust standard errors in parentheses are clustered at market level except in column (4) where they are clustered at PRIO-GRID cell level. Column (5) uses all the sample of crop price data (from 2000 to 2015). All other columns use data until 2006Q1. *** p<0.01, ** p<0.05, * p<0.1.

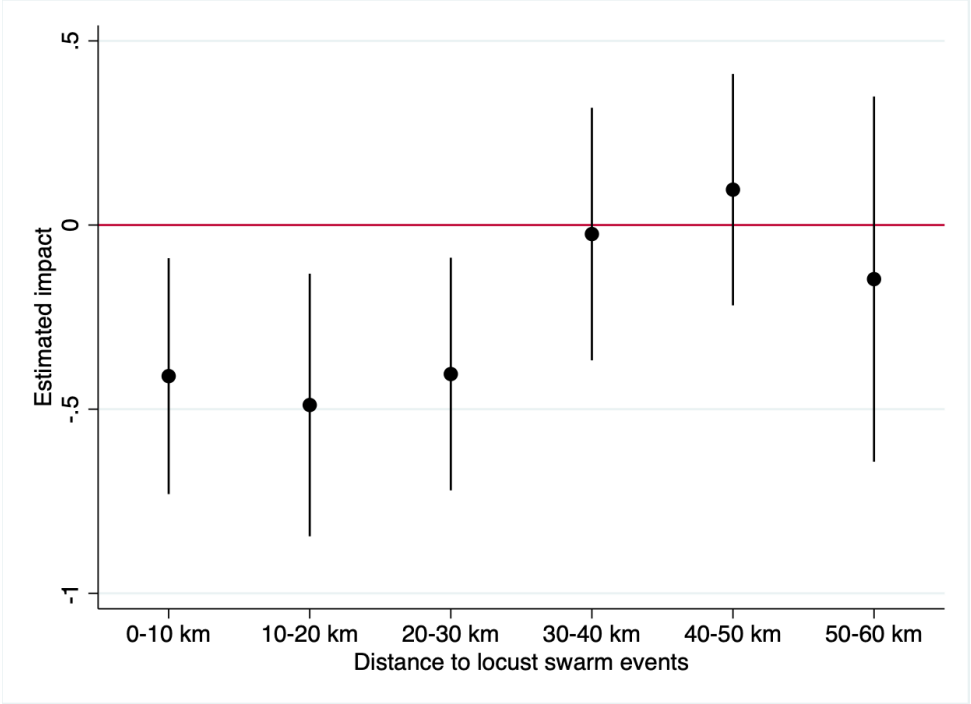

Figure A.5: Impact of Locust Plague on Child Health across Space.

Note: Estimated impact 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.

	(1)	(2)	(3)	(4)	(5)	(6)
In utero treatment	-0.337***	-0.322**	-0.313**	-0.427***	-0.337***	-0.477***
	(0.126)	(0.141)	(0.121)	(0.110)	(0.117)	(0.085)
Observations	4,346	4,346	5,401	9,194	9,194	9,194
R-squared	0.714	0.314	0.666	0.240	0.247	0.004
Number of mothers/households	2120	2,120	2402			
Cohort FE	YES	YES	YES	YES	YES	YES
Child characteristics	YES	YES	YES	YES	YES	YES
Mother FE	YES	NO	NO	NO	NO	NO
Household FE	NO	NO	YES	NO	NO	NO
Location FE	NO	YES	NO	YES	YES	YES
Family characteristics	NO	YES	NO	YES	YES	YES
Mean dependent variable	-1.294	-1.294	-1.422	-1.422	-1.422	-1.422

Table A.8: Additional Robustness I: Impact of Locust Plague on Child Health

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 PRIO grid cell level unless otherwise specified. *** p<0.01, ** p<0.05, * p<0.1. Column (1) shows results for a specification with mother fixed-effects restricted to the sample of children with at least one sibling measured by the 2006 survey wave. Column (2) estimates, with that same subsample, the main specification of Column (3) of Table 1. Column (3) estimates a household fixed effect specification. Column (4) adds survey year-month fixed effects to the main specification. Column (5) shows robustness to using survey weights. Column (6) shows baseline results with no controls allowing for spatial HAC standard errors as in Conley (1999).

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
In utero treatment	-0.478***	-0.462***				-0.334***	-0.277**	
	(0.112)	(0.112)				(0.101)	(0.108)	
Treatment at 30km grid cell			-0.400***					
			(0.115)					
Treatment at municipality level				-0.332***				
				(0.116)				
Treatment with swarms outside Mali					-0.386***			
					(0.111)			
Placebo treatment								0.152
								(0.198)
Observations	9,179	9,187	9,194	9,141	9,194	14,239	9,080	4,187
R-squared	0.200	0.200	0.244	0.238	0.238	0.225	0.256	0.346
Mean dependent variable	-1.420	-1.422	-1.422	-1.422	-1.422	-1.283	-1.115	-1.277

Table A.9: Additional Robustness II: Impact of Locust Plague on Child Health

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 PRIO cell grid level unless otherwise specified. *** p<0.01, ** p<0.05, * p<0.1. Columns (1) and (2) exclude from the baseline specification, respectively, children born in locations that are also exposed to locust events in Q42003 and in June 2004. Column (3) defines the treatment at the 30 km grid cell level and clusters standard errors within these units. 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).

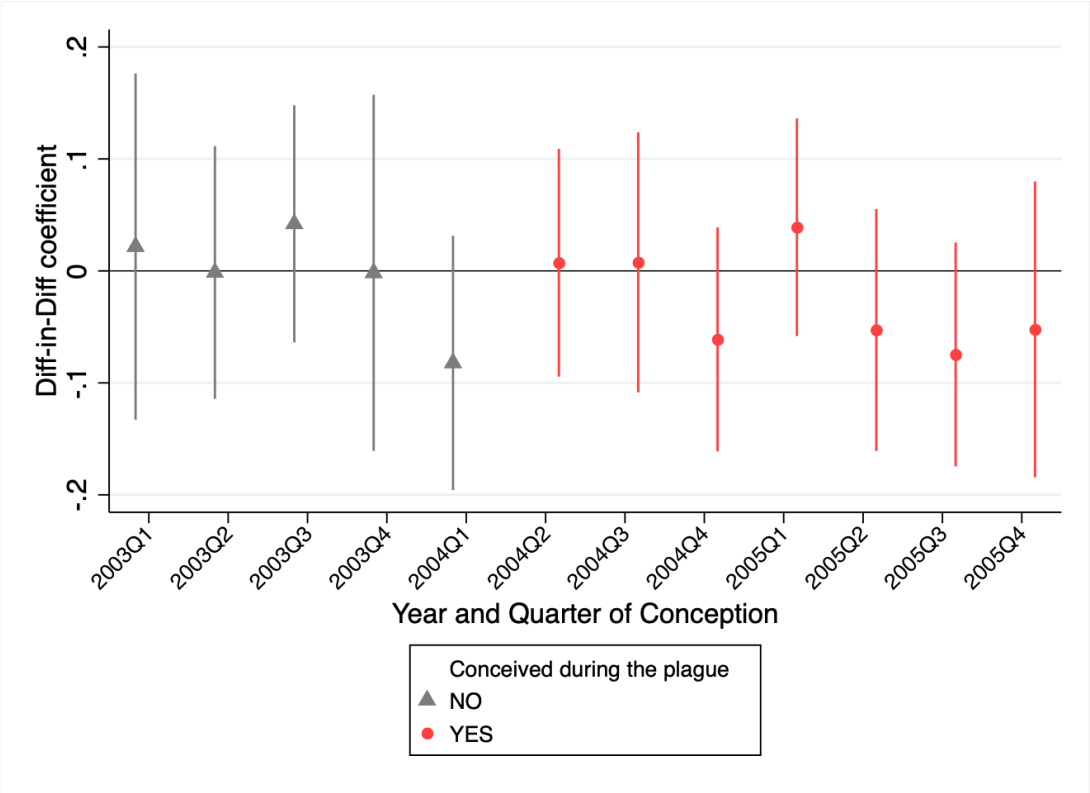

Figure A.6: Impact of Locust Plague on Fertility Decision.

Note: This figure depicts the estimated coefficient of locust invasion on the probability of being conceived in a rich or middle-income household versus being conceived in a household with poor level of wealth. This regression estimates one coefficient for each quarter-year cohort conceived between 2003 and 2005. Each coefficient is plotted together with its 95% confidence bands. The grey triangles refers to children conceived before the plague onset. The red dots denotes children that are conceived during the plague.

	death < 1 month		death < 12 month	
	(1)	(2)	(3)	(4)
In utero treatment	0.007	0.007	-0.005	-0.002
	(0.010)	(0.010)	(0.019)	(0.018)
Observations	24,233	24,233	22,677	22,677
R-squared	0.074	0.074	0.112	0.113
Region specific time trend	NO	YES	NO	YES
Mean dependent variable	0.0499	0.0499	0.122	0.122

Table A.10: Impact of Locust Plague on Neo-natal and Infant Mortality

Source: All the regressions include location fixed effects, cohort fixed effects, child and family characteristics. 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 neo-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.

A.5 The potential role of anti–locust spraying campaigns

Anti–locust spraying is a mitigating tool that can be harmful if done under specific circumstances. Spraying toxic insecticides over large areas can contaminate entire plantations, harvests, livestock, and water. This is mostly the case with aerial spraying, which consists of spraying pesticides over large areas with airplanes, potentially leading to harmful consequences on biodiversity.[50] A second known effect of these spraying campaigns comes from the direct human exposure to the pesticides. That affects mostly the operators of ground spraying (with trucks) when the spraying is done under weak security measures.[51]

In fact, FAO deployed spraying operations in 2004 to potentially mitigate the effects of the locust plague. Thus, depending on the circumstances under which these operations were conducted, there could have been a direct health effect on exposed

[50] See, for instance, https://www.nationalgeographic.com/environment/article/locust-plague-hit-east-africa-pesticide-solution-may-have-dire-consequences (last accessed 28.06.2022)

[51] Indeed, this is argued to be the case in Western Africa by early 2000's, where resources to face the locust plague were very limited compared to the dimension of the plague. In particular, FAO and CRC (Comission for Controlling the Desert Locust in the Central Region) both document the direct harm of the ground spraying to operators under poor conditions. See https://www.fao.org/ag/locusts/common/ecg/347_en_DLG4e.pdf and http://desertlocust-crc.org/App_Data/App_Uploads/Bulletins/Files/191121101130Human%20health%20and%20environmental%20safety%20in%20desert%20locust%20control%20operations%20en.pdf, for instance (last access 28.06.2022).

	Height-age Z-score		
	(1)	(2)	(3)
Treatment with speculative/anticipatory effect	-0.477*	-0.482*	-0.478*
	(0.242)	(0.247)	(0.262)
Treatment with both	-0.274**	-0.273**	-0.266**
	(0.129)	(0.129)	(0.120)
Treatment with crop failure effect	-0.454***	-0.445***	-0.432***
	(0.129)	(0.130)	(0.123)
Treatment with speculative/anticipatory effect × Distance	-0.076	-0.074	-0.034
	(0.141)	(0.143)	(0.149)
Treatment with both × Distance	-0.223**	-0.226***	-0.150
	(0.086)	(0.085)	(0.098)
Treatment with crop failure effect × Distance	-0.345***	-0.356***	-0.258***
	(0.070)	(0.070)	(0.098)
Observations	6,479	6,479	6,479
R-squared	0.239	0.240	0.244
Household wealth	NO	YES	YES
Region time trends	NO	NO	YES
Mean dependent variable	-1.607	-1.607	-1.607

Table A.11: Robustness of the sensitivity of treatment effects with respect to the level of isolation measured with euclidean distance (kilometers)

Source: All regressions include cohort fixed effects, location fixed effects, child and family characteristics. 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. Distance stands for the standardized euclidean distance (km) 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.

individuals. In other words, spraying could be a confounder in our results. In what follows, we provide evidence against this hypothesis. In particular, we describe the characteristics of the spraying campaigns around the period of the 2004 plague in Mali and argue that the exposure to these events is not an identification threat to our main empirical results.

In this perspective, we retrieve data on the incidence of the FAO spraying events between 2000 and 2007 from the SWARMS database. It provides information on the GPS coordinates and time periods of the anti–locust spray events in that period. Figure A.7 shows the distribution of these events across space and time. Anti–locust spray events are obviously spatially correlated with the locust swarm events. They also took place mostly at the beginning of the upsurge in late 2003 and during the plague peak in 2004. Unfortunately the information about the type of insecticides used is available for less than 1 per cent of the sample. Thus, we cannot exploit variation between the usage of not–/harmful insecticides during the 2004 campaign. Yet, we are able to discern the typology of the spraying events (e.g. aerial or ground). Such a distinction is of interest because aerial spraying events should be the most concerning ones for the validity of our main results. The reason is that, as long as pregnant

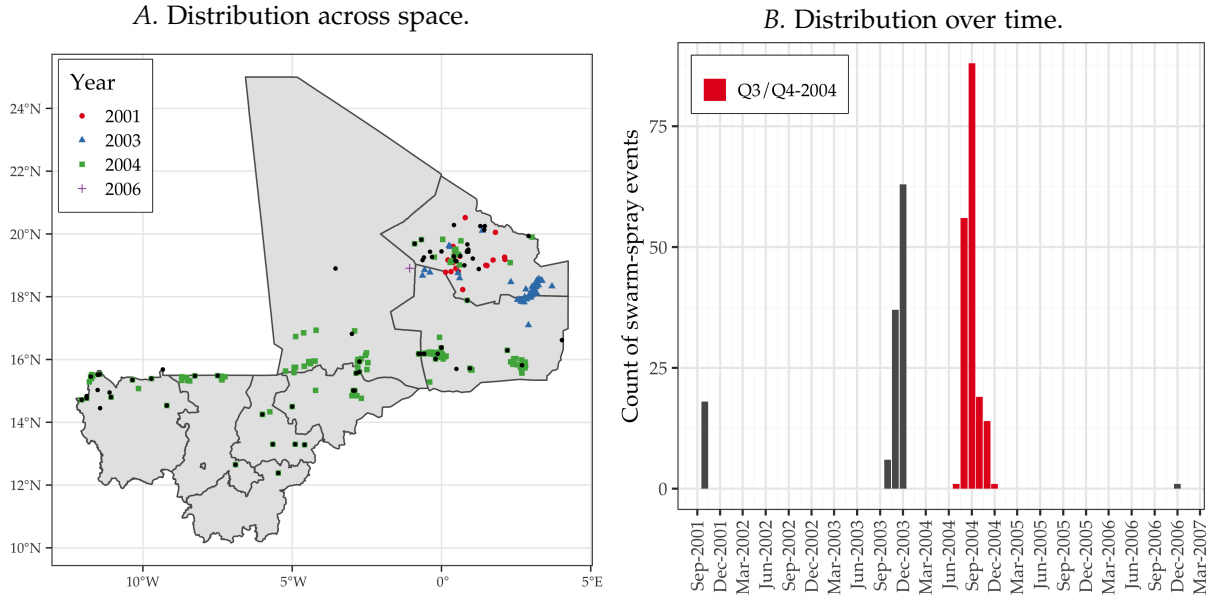

Figure A.7: Distribution of swarm and spraying events across space (Panel A) and time (Panel B) in Mali between 2000 and 2007.

Source: SWARMS–FAO database. Black dots in Panel A refer to locust swarm events in 2004, whereas the colored ones to anti–locust spraying events.

women did not systematically work as spraying operators in 2004, only aerial spraying should have affected the households located in locust-affected areas through their impacts on harvests, livestock, etc.

We find that aerial spraying events were fewer compared to ground spraying events. This is documented in Figure A.8: a large share of the spraying sample stands for ground spraying (about 60 per cent of the events between 2003 and 2004), whereas a smaller one stands for aerial (about 18 per cent). The rest of the events do not contain information on the typology of the implementation method. Thus, we conclude that, if present, the effects of the spraying on the treated children should not be large.

To be conservative, we also exploit the variation between exposure to locusts and spraying at the household level. To do that, we first assign exposure to spray events analogously to the exposure to locusts (i.e. if the household is located within 30 km of a spraying event between 200 and 2007, disregarding its type). The ideal experiment would be to verify whether the treatment effects of the in–utero exposure to the locust hold for the subsample of children that are not exposed to any spraying event. However, most of the spray–exposed children are in the treatment group, in line with the high spatial correlation between locust and spray events shown before. This is shown in Panel A of Table A.12: more than 90 per cent of the treated children (550/604) belong to the sub-sample of exposed to spray events. Thus, we do not have sufficient power and representativeness of the not exposed sample (bottom row) to estimate our main specification.

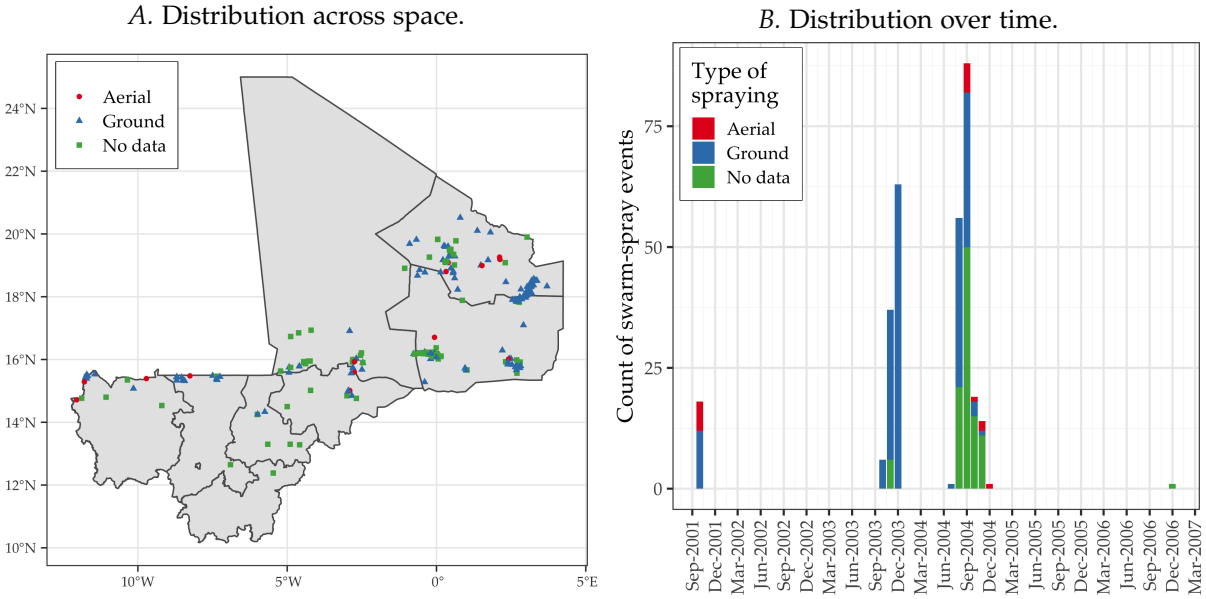

Figure A.8: Distribution of swarm and spraying events across space (Panel A) and time (Panel B) in Mali between 2000 and 2007 by types of spraying.

Source: SWARMS–FAO database. Black dots in Panel A refer to locust swarm events in 2004, whereas the colored ones to anti–locust spraying events.

Nevertheless, we exploit other dimensions of heterogeneity in our sample and results to argue that anti–locust spraying should not be driving the treatment effects we identify. In particular, Panel B of Table A.12 shows the distribution of not–/exposed samples along the rural–urban dimension. The exposed sample (first row) consists mostly of children living in rural households, in line with the idea that locusts invade plantations (not located in urban areas). If spraying were confounding our baseline treatment effects, children located in urban households should not be affected by the in–utero exposure to the locust plague. We do find, however, evidence for the contrary: treated children in both rural and urban households are estimated to suffer health setbacks, as shown in tables A.5 and A.6. An analogous argument holds when we compare treated children born in more and less isolated households. Table 3 shows that the former suffer larger health impacts from the in-utero exposure to the locust plague. However, we find that most of the spray-exposed children belong to less isolated households, suggesting that spray campaigns are not the main driver of our estimated health effects.

	Panel A		Panel B		Panel C	
	Control	Treated	Rural	Urban	Isolated	Not isolated
Exposed to spray	1102	550	1229	423	519	1133
Not exposed	7488	54	5250	2292	1790	5752

Table A.12: Exposure to spraying campaigns between 2000 and 2006 and other characteristics of the main estimating sample.

Notes: Tabluation of subsets of the main sample of 9,194 children used in the regressions. Exposed to spray stands for children born in DHS clusters located within 30 km or less from a FAO spraying campaign event between 2000 and 2006.