Protecting Infants from Natural Disasters: The Case of Vitamin A Supplementation and a Tornado in Bangladesh*

Snaebjorn Gunnsteinsson Teresa Molina Achyuta Adhvaryu Parul Christian
Alain Labrique Jonathan Sugimoto Abu Ahmed Shamim Keith P. West, Jr.

April 28, 2022

Abstract

Environmental disasters have increased in frequency and intensity as a result of climate change. Can timely intervention help protect against the health impacts of these disasters? We study this question by leveraging data from a double-blind cluster-randomized controlled trial of at-birth vitamin A supplementation, which boosts immune system functioning, in Bangladesh. During the trial, a large tornado swept through the study area, affecting both treatment and control clusters. Tornado exposure in infancy decreased physical growth and increased the incidence of severe fevers. But infants who received supplementation were protected from these negative effects.

JEL: I18, J13, Q54
Keywords: natural disasters, early childhood, climate change, micronutrient supplementation, vitamin A, Bangladesh

*Gunnsteinsson: University of Maryland; sgunnste@umd.edu; snaebjorn.com; Molina: University of Hawaii at Manoa and IZA; tmolina@hawaii.edu; Adhvaryu: University of Michigan, NBER, BREAD, Good Business Lab, William Davidson Institute; adhvaryu@umich.edu; Christian, Labrique and West: Johns Hopkins Bloomberg School of Public Health. Sugimoto: Fred Hutchinson Cancer Research Center; Shamim: JiViTa Research Project. Thanks to Marcella Alsan, Nava Ashraf, Prashant Bharadwaj, Hoyt Bleakley, Andy Foster, Paul Gertler, Jess Goldberg, Pam Jakiela, Anant Nyshadham, Rafael Perez-Escamilla and Atheen Venkataramani, as well as seminar participants at the NBER (CH & DEV), Michigan, Yale, USC, Maryland, Barcelona GSE, NEUDC, PopPov, and the CDC for helpful discussions. Adhvaryu gratefully acknowledges funding from the NIH/NICHD (5K01HD071949). This paper was previously titled "Resilience to Early Life Shocks."

1 Introduction

A child's environment in early childhood shapes her survival and wellbeing in profound ways (Almond and Currie, 2011; Currie, 2000; Heckman, 2007; Jayachandran and Pande, 2017). Shocks to this environment are all too common and can create life-long disadvantage in terms of health and economic wellbeing, especially in low-income contexts (Almond et al., 2018; Currie and Vogl, 2013). Natural disasters in particular can have devastating short- and long-run effects. From 1996 to 2015, natural disasters were responsible for 1.35 million deaths; the vast majority of this impact was borne by low- and middle-income countries (CRED/UNISDR, 2016), and these numbers belie much broader, if insidious, impacts on health and economic outcomes for survivors (Cas et al., 2014; Currie and Rossin-Slater, 2013; Frankenberg et al., 2008; Hornbeck, 2012; Karbownik and Wray, 2019). Moreover, these shocks are growing in frequency and intensity due to climate change, disproportionately affecting climate-vulnerable populations in low-income countries (Burke et al., 2015). In this paper, we ask: can preventative health investments effectively protect vulnerable populations, especially young children, from the negative impacts of these shocks? And can such intervention help those who have been affected to recover? Answering these questions rigorously is not a straightforward task. Billions of dollars are invested annually to aid recovery efforts in communities affected by natural disasters (Linnerooth-Bayer et al., 2005). Measuring the effectiveness of these investments entails identifying both the causal effects of a natural disaster as well as the causal mitigating effects of a particular investment. While it is plausible that exposure to some types of environmental shocks is as good as random, measures taken to buffer against negative impacts – and, analogously, measures to mitigate impacts once shocks occur – are most likely not random at all. They are deliberate choices made by households, communities, relief organizations, and governments. Comparing outcomes after a shock across different levels of investment would typically yield biased estimates of the protective or remediating effects of that investment.

The aim of this study is to identify these effects by leveraging a unique situation in which a natural disaster affected several areas of northwest Bangladesh that were involved in a large double-blind cluster-randomized controlled trial (RCT) of newborn vitamin A supplementation. The RCT was evaluating the public health impacts of a large, one-time oral dose of vitamin A immediately after birth on infant health and survival.

On March 20, 2005, while the RCT was ongoing, a tornado tore through the study area, generating substantial property damage in 1007 of the approximately 18,000 households in our sample; killing 56 people; and injuring nearly 4000 (Gunnsteinsson et al., 2010; Sugimoto et al., 2011). The quasi-random "selection" of localities by the tornado allows for the assessment of effects on various birth outcomes, which were being evaluated as part of the ongoing trial, for infants exposed to the stress of the tornado *in utero*). We document that *in utero* exposure to the tornado increased the probability of low birth weight (< 2.5kg) by 8 percentage points (15% on the mean), and decreased birth length by half a centimeter.

Having documented negative effects on birth size (determined before the vitamin A supplementation took place), we then move on to examine outcomes measured at 3 and 6 months. The overlap in tornado exposure and RCT treatment status – both treatment and control localities were affected in a balanced way – enables us to estimate the remediating effects (for infants exposed to the tornado while *in utero*) and protective effects (for infants exposed after birth) of vitamin A supplementation on anthropometric and survey-based outcomes for infants at 3 and 6 months. We detail the identification assumptions necessary to obtain unbiased estimates of these effects, and provide empirical support for each of these assumptions.

Our examination of infant outcomes suggests that vitamin A had significant protective effects. In control localities, tornado exposure in the first three months of life had large negative impacts on chest circumference and mid-upper arm circumference (reliable correlates of child mortality) and on the incidence of severe fevers measured at both 3 and 6 months. This was likely due to the detrimental effect of the tornado on the disease and nutrition environment. In treatment localities, however, in which all infants were dosed with vitamin A at birth, these impacts were nonexistent. We argue this is due to role of vitamin A in strengthening the immune system and protecting from infection. Effects were entirely driven by impacts on boys; girls were largely unaffected by the tornado, even in control areas. The results were not driven by differential rates of miscarriage, stillbirth, or attrition from the sample after birth.

Large environmental shocks, like the one studied in this paper, and insidious exposures (for example, to air pollution or heat stress) can have sizable long-run impacts – particularly for exposures at critical periods of fetal and early childhood development. Impacts that are generated early in infancy, if not corrected, tend to persist over the life cycle (Almond and Currie, 2011; Almond

et al., 2018; Currie and Vogl, 2013). Measuring these early impacts, and evaluating whether certain interventions might mitigate negative effects, is therefore critically important from both the academic and policymaking perspectives (Currie and Vogl, 2013). Our contribution to this space is to demonstrate that it is possible to substantially dampen the impacts of exposure to a severe shock via vitamin A, which is known to enhance immune function (Cunningham-Rundles et al., 2009; Huang et al., 2018; Semba, 1994; Stephensen, 2001; West, 1991).

Our results also shed light on the mixed evidence regarding the effectiveness of vitamin A supplementation in reducing infant and child mortality (Awasthi et al., 2013; Haider and Bhutta, 2011; West Jr et al., 1995; Klemm et al., 2008; Mayo-Wilson et al., 2011). Another interpretation of our main finding (negative health effects of a tornado can be mitigated by vitamin A supplementation) is that vitamin A supplementation was more effective in tornado-affected regions. That is, the effects of vitamin A supplementation are very context-dependent, even within the same geographic location. Our analysis therefore highlights that differences in natural disaster risk by location may be an important factor to consider when interpreting the range of estimated impacts from newborn vitamin A supplementation observed in different contexts. As such, our results suggest that wide-scale supplementation policies would generate potential health benefits in areas where immune systems are weak or risk of infection is high, like disaster-prone areas of low-income countries.

These results are relevant in context of the renewed focus on curbing rates of infant mortality in low-income countries (Bhutta et al., 2013, 2012). Despite significant progress over the last decade (Lozano et al., 2011), more than 3 million children still die each year from "preventable" causes (Liu et al., 2015). This study suggests a vital role for vitamin A as protection from the increasingly devastating health effects of natural disasters (Field, 2012).

Finally, we also contribute to the literature on the early formation of health and human capital (Almond and Currie, 2011; Heckman, 2006, 2007; Jayachandran and Pande, 2017). The question of protecting infants from shocks via supplementation relates to the interaction between existing endowments and investments at different stages of life (Almond and Mazumder, 2013; Almond et al., 2018; Cunha et al., 2010). In line with our results, several studies from diverse contexts find evidence that early-life interventions often yield larger benefits for more disadvantaged children (Adhvaryu et al., 2018; Bitler et al., 2014; Cornelissen et al., 2018; Rossin-Slater and Wüst, 2018).[1]

[1]One recent paper, Duque et al. (2018), using administrative data on children from Colombia, finds the oppo-

2 Context, RCT, and Tornado

2.1 Vitamin A and Infant Health

Vitamin A is vital to the proper functioning of neutrophils, macrophages, and natural killer cells – essential components of the body's immune system. It also helps prevent infections by maintaining epithelial integrity (Thurnham et al., 2000) and restores innate immunity after infection by promoting the normal regeneration of mucosal barriers (Stephensen, 2001).

In vitamin A-deficient contexts, supplementation at birth can reduce infant mortality (Haider and Bhutta, 2011; Klemm et al., 2008). Multiple randomized trials of infant vitamin A supplementation in South Asia have found a reduction in infant mortality in excess of 10% (Haider and Bhutta, 2011; Humphrey et al., 1996; Klemm et al., 2008; Mazumder et al., 2015; Rahmathullah et al., 2003).[2] This includes the trial we study, which found a 15% reduction. We hypothesize that this reduction is due in part to the ability of vitamin A to prevent or mitigate the impacts of shocks that the infant experiences shortly after birth. Vitamin A supplementation in post-infancy (6 months to 5 years) has been shown to improve child survival based on evidence from a wide variety of contexts (Sommer et al., 1996).

Vitamin A supplementation has also been found to improve other measures of infant and child health. For example, Humphrey et al. (1996) document that vitamin A supplementation at birth reduced the incidence of cough and fever in the first four months of life. For older children, vitamin A supplementation has been found to reduce the incidence of diarrhea and measles (Mayo-Wilson et al., 2011; Zeba et al., 2008). Related studies have shown that vitamin A supplementation can lower the severity – specifically, the case fatality rates – of measles, diarrhea, and fever (Barclay et al., 1987; Hussey and Klein, 1990; Tielsch et al., 2007).

Due to the bidirectional relationship between infection and malnutrition (Dewey and Mayers, 2011; Macallan, 2009), the protective effects of vitamin A can also lead to improved growth. Studies have found improvements in child weight and arm circumference due to vitamin A supplementation (Donnen et al., 1998; West et al., 1988).

site pattern of results, i.e., larger long-run effects of a CCT program for children who were *not* exposed to early disadvantage.

[2]Similar effects have not been observed in Sub-Saharan Africa (Bhutta et al., 2013), for reasons that remain unknown.

Despite dramatic improvements in infant and child health in Bangladesh over the last 3 decades (Wang et al., 2014), the survival and health of Bangladeshi children lies well below the global mean, with the majority of neonatal and infant deaths due to treatable causes such as diarrheal disease and pneumonia (Liu et al., 2015). Micronutrient deficiencies are common in the Bangladeshi setting. In a comprehensive review of the medical and public health literature, Bhutta et al. (2013) cite the potential gains from large-scale micronutrient supplementation – in particular, with vitamin A, iron/folic acid, and zinc – in low-income countries.

2.2 RCT Design

The RCT we study was part of a nested double-blind placebo-controlled cluster randomized trial of maternal and newborn vitamin A supplementation in Bangladesh, conducted from 2001 to 2007. In the maternal trial there was also an arm providing β-carotene. These trials and the tornado survey referred to below were all approved by the Institutional Review Board of the Bloomberg School of Public Health, Johns Hopkins University, and the Ethics Committee of the Bangladesh Medical Research Council. Each of the trials was pre-registered at clinicaltrials.gov; the identifiers are NCT00198822 (maternal trial) and NCT00128557 (infant trial). These trials are part of the JiVitA Bangladesh international nutrition research project on maternal and child health.

Both trials were conducted in a contiguous 435 square kilometer area in northwest Bangladesh, in Rangpur Division, with an estimated population of about 600,000 (see Appendix Figure A1 for a map of the study region). The study area was subdivided into 596 sectors, each of which was populated with 107 to 377 households at baseline. These sectors were randomized using a 3 x 2 cluster randomized factorial design with three different groups for pregnant women and 2 groups for their newborn children. The 3-group randomization (maternal trial) used a geographic block randomization, which is described in detail in West et al. (2011). The 2-group randomization (infant trial) was also done by geographic block randomization, where each block was defined within one of the three earlier groups, as described in Klemm et al. (2008).

All married women in the study area in 2001 and newlywed women, ages 13-45, were surveilled for pregnancy over the next six years. In total, over 60,000 pregnancies were identified and, if consent was given (>99% of cases), the pregnant woman was enrolled in the maternal supplementation study. The two treatment groups in the maternal trial received the recommended weekly allowance

of vitamin A, either in the form of vitamin A or β-carotene (which the body converts into vitamin A), as weekly supplements from first trimester through 12 weeks postpartum, while the control group received a placebo supplement.

In this paper we focus the analysis solely on the newborn supplementation trial, which was nested within the maternal trial and was conducted between January 2004 and December 2006. Live-born infants in each sector were randomized to receive either 50,000 International Units (IU) of vitamin A or a placebo once as oral oil drops from a capsule shortly after birth (50,000 IU are equivalent to 15,000 μg retinol (U.S. Department of Agriculture, 2011)). Adequate intake, based on a diet of breast milk from a healthy mother, is 400 μg retinol equivalent per day (Institute of Medicine , US)). For further information on field procedures and other details, we refer the reader to Labrique et al. (2011), West et al. (2011) and Klemm et al. (2008).

Of the approximately 19,000 infants that were part of the newborn trial, around 16,000 received supplementation or placebo directly at birth (or shortly thereafter) and were followed until 6 months after birth. Previous work has documented that the at-birth supplement reduced mortality at 6 months by 15% (Klemm et al., 2008).[3]

The reason we focus on the newborn supplementation trial is that maternal supplementation with vitamin A or β-carotene in this setting had no impact on maternal, fetal, or infant mortality (West et al., 2011), nor on gestational length or birth anthropometry (Christian et al., 2013). In a set of analyses not reported here, we do not find any protective or mitigating effects of maternal supplementation, in line with the lack of main effect of this treatment on mortality.

On the night of March 20th, 2005, a tornado swept through Gaibandha District, affecting about 7% of the study area (Sugimoto et al., 2011) (see top left panel of Figure A2). Between August and October 2005 each household in the affected areas was visited by a survey enumerator, who asked questions on mortality and morbidity of household members as well as damage to homes as a result of the tornado. Based on this survey, the tornado resulted in 56 deaths, injured 3,710 people, and destroyed 3,540 houses (Sugimoto et al., 2011). Out of 596 study sectors, at least one house was destroyed in 41 sectors, and in 24 sectors more than 20% of houses were destroyed.

Our evidence suggests that the tornado had no effect on the timing of supplementation or

[3]Though large in relative terms, these effects do not appear to have substantially changed the sample composition of the treatment group relative to control group (as we show later), likely because overall infant mortality was low.

anthropometric measurement and surveying. For instance, among infants in their second or third trimesters *in utero* during the tornado, those in the tornado area were supplemented within 24 hours at the rate of 71.4% while those outside of this area were dosed at the rate of 72.6%. Birth anthropometry for this same population was obtained within 7 days in the tornado area at the rate of 82.9% and outside this area at the rate of 84.1%. These differences are small and not statistically different from 0.

In addition, tornado intensity was similar across vitamin A and placebo households. A Kolmogorov-Smirnov test cannot reject the null that the distributions of tornado intensity were the same in vitamin A and placebo sectors. Figure 1 illustrates the cumulative distribution functions for each group. We restrict to households within 1.6 km of the tornado path (described in more detail below), in order to allow for a closer examination of the non-zero portion of the distribution since the majority of households were not hit by the tornado.

3 Research Design

3.1 Defining the Sample, Cohorts and Tornado Exposure

We begin with all infants in the infant supplementation trial (all infants that the study intended to dose, whether they were ultimately dosed or not) for whom consent was obtained for supplementation ($> 99\%$), save for 154 observations for which we do not have data on the date of the last menstrual period (and are therefore unable to construct our exposure cohorts in a consistent way), and another 154 observations for which we do not have latitude and longitude coordinates (necessary for defining tornado exposure). After these adjustments, the sample is 18,879 live births.

We approximate the path of the tornado based on damages to homes in the area using the survey conducted in Sugimoto et al. (2011). We split the study area into 50 even sized vertical bands and calculate the average latitude of houses destroyed within the band. These coordinates (for bands that have any damages), along with the longitudinal midpoint of each band creates a series of knots that maps the approximate path of the tornado. We then define tornado exposed households as those who are within 1km of one of these knots (depicted in lower left corner of Figure A2). By this definition 450 out of the 458 houses destroyed by the tornado (98%) are within the tornado path.

Second, we construct dummies for three main time periods of early exposure: the prenatal period (i.e., the infant was *in utero* during the tornado event), the first 3 months of life, and the second 3 months of life (i.e., the infant was 0-3 months or 3-6 months during the tornado). About half of the sample falls into one of these three cohorts, and the remainder is comprised of those born more than 6 months before or more than 9 months after the tornado.[4] Throughout the paper, we define the *in utero* period as the time between our best guess of the date of conception and birth. The best-guess date of conception is determined via a combination of information on the woman's last menstrual period (self-reported) and a urine test-based confirmation of pregnancy.[5]

Third, we use randomized variation in the allocation of vitamin A to newborns by sector. Accordingly, we construct a dummy for whether the infant was born in a treatment sector, meaning that he was assigned to receive vitamin A as opposed to a placebo supplement at birth. As explained earlier, supplementation at birth in the RCT was cross-randomized with prenatal supplementation and was balanced across the newborn supplementation trial, and thus we do not need to control for prenatal supplementation status.

In Table 1, we compare the characteristics of infants in tornado and non-tornado areas, restricting to infants who were born at least 7 months before the tornado, which means they had no tornado exposure in early life (up to 6 months of age). Differences between the two groups in birth outcomes, health at 3 months, health at 6 months, maternal characteristics, and timing of dosing are small in magnitude and statistically insignificant.

In addition, Table 1 shows that differences between Vitamin A and placebo groups, for this same sample of pre-tornado infants, are also minimal. The vast majority of the treatment-placebo differences are small in magnitude – less than 3% of the placebo group means. One exception is the 6-month mortality rate, which is about 17% lower in the treatment group. Although this difference is not statistically significant, the magnitude is in line with the results reported in Klemm et al. (2008).[6] Though large in relative terms, these differences in mortality rates correspond to only small differences in the number of infant deaths across the two groups. This is likely why these mortality

[4] Around 5000, 2000, and 2500 were exposed *in utero*, 0-3 months, and 3-6 months, respectively.

[5] Note that women were under active pregnancy surveillance. If amenstrual, women were offered a urine-based pregnancy test. Once pregnant, women were monitored closely and visited soon after birth for measurements and data collection.

[6] Note that the sample in Klemm et al. (2008) differs from the sample in Table 1 in two ways: Klemm et al. (2008) restricts to infants who were visited for dosing and does not restrict to those born at least 7 months before the tornado (as Table 1 does).

differences did not substantially change the composition of the treatment group compared to the placebo group (see 6-month outcomes in Table 1, which are only available for infants who survived to 6 months, as well as Table B1 and the accompanying discussion).

3.2 Estimation

This section outlines the empirical specification and discusses the assumptions required to identify the causal effect of the tornado and its interaction with the vitamin A supplemenation. Our basic specification is

$$Y = \alpha + \beta_1 T + \beta_2 T \cdot E + \beta_3 E + \beta_4 C + \beta_5 C \cdot E + \beta_6 C \cdot T + \beta_7 C \cdot T \cdot E + u \tag{1}$$

where T is vitamin A treatment, E is being in an area exposed to the tornado (regardless of whether an infant is in an affected cohort) and C is an indicator for being in an affected cohort (regardless of whether an infant is in the tornado affected area). This equation generalizes easily to incorporate multiple cohorts. In most specifications, we include several different cohort dummies as follows:

$$Y = \alpha + \beta_1 T + \beta_2 T \cdot E + \beta_3 E + \sum_j \beta_{4j} C_j + \sum_j \beta_{5j} C_j \cdot E + \sum_j \beta_{6j} C_j \cdot T + \sum_j \beta_{7j} C_j \cdot T \cdot E + u, \tag{2}$$

where j is an index representing a different period of life: in-utero, first 3 months of life, or second 3 months of life. C_j indicates whether or not an infant was in period j when the tornado hit.

Under the three assumptions discussed in detail in section B, β_{5j} provides us with the causal effect of being exposed to the tornado during time period j for those without vitamin A supplementation, while β_{7j} provides the differential effect of tornado exposure (during time period j) for those who received vitamin A supplementation.

Several pieces of evidence support the validity of this empirical strategy. First, we show in Table B1 that within each of the four sub-samples defined by tornado exposure and cohort ($E = 0$ and $C = 0$, $E = 1$ and $C = 0$, $E = 0$ and $C = 1$, $E = 1$ and $C = 1$), characteristics are similar across treatment and control even after restricting to infants who remain in the sample by the time outcomes are measured. The largest differences are in mother's age at enrollment, but this difference is only significant for one group ($E = 0$, $C = 0$), where the magnitude translates to less

than 4 months in the mother's age. While the random assignment of treatment, along with the independence of the tornado (E) and vitamin A treatment (T), would guarantee balance across treatment and control groups in the original sample of infants, this need not necessarily be true after conditioning on the observed sample (due to potential selective mortality resulting from the 15% reduction in infant mortality in the vitamin A group). The fact that we find balance in the surviving sample suggests that the infant mortality effects did not lead to large changes in the sample composition, likely because overall infant mortality was quite low (6%).

We also find little evidence of differential pre-trends across tornado and non-tornado areas. Figure B1 shows cohort trends in health outcomes for children born before the tornado, separately for those inside and outside the tornado-affected areas. Table B2 reports regression estimates of these pre-trends, allowing for different trends in vitamin A and control areas. We detect no evidence that pre-trends differ across tornado and non-tornado areas, or that this difference in pre-trends (across tornado and non-tornado areas) varies across groups that recevied vitamin A versus placebo. See section B for a more detailed description of the identification assumptions and associated tests.

3.3 Estimation of Standard Errors

To account for the clustered design of the RCT and the spatial correlation in tornado exposure, we implement a randomization inference procedure to construct confidence limits and estimate statistical significance. Fisherian randomization inference, which continues to become more common in the economics literature (see, for example, Heß (2017) and the papers cited therein), involves generating a large number of permutations of the treatment assignment and comparing the treatment effect estimates obtained from the actual treatment assignment to the distribution of estimates obtained from the hypothetical permutations.

Because our empirical strategy relies on two sources of exogenous variation, each "placebo scenario" we construct involves a new vitamin A randomization allocation (according to the original location stratified randomization procedure), as well as a new a "placebo tornado" – that is, a random area somewhere within the study area of a similar size and shape as the original tornado. Unlike the assignment of vitamin A supplementation (and unlike in other typical applications of Fisherian randomization inference), the assignment rule for tornados is unknown. Other work using randomization inference for rainfall shocks draws placebo scenarios from 70 years of historical

rainfall patterns in the U.S. (Cooperman, 2017), but our setting is not well-suited for this approach given the more infrequent nature of tornados and limited data availability. Instead, we generate placebo tornados by choosing a random house in the study area and a random angle (between 0 and 360 degrees) defining a direction from this house. We then define knots along a linear path from the chosen house in the direction of the chosen angle up to a distance that equals the distance traveled by the original tornado. The households affected by the placebo tornado are defined (similarly to the original tornado) as those within a 1km radius of one of those knots. If a placebo tornado lands substantially outside the study area or substantially overlaps with the original tornado then we exclude it and instead compute a new one for the given iteration.[7]

The middle and right panels of Figure A2 in the appendix show four examples of these "placebo tornados". For each scenario we estimate our main specification using the placebo exposure definitions and treatment indicators. We repeat this process 5,000 times to obtain a distribution of "placebo" coefficient estimates for each coefficient in our specification. We use the range of this distribution (distance between the 2.5th and the 97.5th percentile) as the width of our 95% confidence intervals and compute p-values by identifying where our original estimate falls on this distribution.[8]

4 Results

In this section, we begin by documenting that *in utero* exposure to the tornado negatively affected birth outcomes. We then move on to examine the effect of *in utero* and early life exposure to the tornado on health outcomes measured at three and six months of age. Because vitamin A supplementation took place immediately after birth, we are also able to examine the extent to which the vitamin A treatment mitigated any effects the tornado had on these three-month and six-month outcomes. We then discuss robustness checks.

[7] If the number of households outside the tornado area covered by the placebo tornado is less than 90% of the number of houses covered by the original tornado then this placebo tornado is excluded. Although the above rules imply that the center of the study area is more likely to be in a placebo tornado region than the edge of the study area, we argue that the relevant set of tornados are those which covered enough of the study area to make this empirical approach possible.

[8] Standard errors are calculated by dividing the confidence interval width by 2*1.96.

4.1 Birth Outcomes

Table A1 reports impacts of the tornado on birth outcomes. To do this, we estimate a difference-in-differences specification equivalent to equation (1) excluding all vitamin A treatment indicators (because supplementation took place after birth). We report β_5, the coefficient on the interaction between tornado location and affected cohort, as the tornado effect.

The first column in this table reports impacts on birth weight, measured in kilograms (kg). Tornado exposure *in utero* had a statistically significant negative impact on birth weight. In addition, infants exposed *in utero* were about 8 percentage points more likely to have low birth weight (under 2.5kg), from a baseline of 54 percent (column 2). We observe this effect throughout the lower end of the birthweight distribution, as can be seen in column 3, which reports that infants exposed *in utero* were 7 percentage points more likely to be born under 2kg (from a baseline of 14%). We also find significant negative impacts of the tornado on length at birth, another summary measure of newborn health. The last two columns show no significant effects on gestational age or prematurity (born before 37 weeks).

4.2 Outcomes Measured at 3 and 6 Months

Next, we estimate the impacts of tornado exposure *in utero* and in early life on infants' outcomes at 3 and 6 months, using specification (2), which allows us to identify the protective effects of vitamin A supplementation at birth. We show results for mid-upper arm circumference (MUAC), chest circumference, head circumference, and the number of severe fever episodes (over the past three months). The latter is reported by the infant's mother and likely subject to some recall bias. Fever in particular is an important potential mediator of impacts on anthropometry because of the crucial role of vitamin A in providing barriers to infection and supporting a healthy immune system.

Table 2 reports tornado impacts on these outcomes at 3 and 6 months, for both *in utero* and early life (0-3 month and 4-6 month) exposure, by vitamin A treatment status. There are no statistically significant effects of *in utero* tornado exposure on any outcomes for the control group (with the exception of a marginally significant effect on fevers at 3 months), or any differential effect in the vitamin A treatment group. This is also true of 4-6 month tornado exposure: no impact in

the control group (except for fevers) and no differential impact for the treatment group.

There is however, a clear pattern for 0-3 month tornado exposure. For both 3 and 6 month outcomes, the tornado had large deleterious effects on MUAC, chest circumference, and fever incidence in the control group, but essentially no impacts whatsoever in the vitamin A group. The difference across these two groups is, in general, statistically different from 0 (with the exception of 4-6 month fevers). The coefficients in the head circumference regressions display a similar pattern as those in the chest circumference regressions, but are not statistically significant.

In short, tornado exposure does not always generate negative effects, but exposure during the sensitive early period between 0 and 3 months of life does have substantial impacts on infant health. Impacts are quite large – e.g., 0.35-0.37 cm for MUAC, which translates to approximately 0.4 SD, and between 0.4- 0.5 additional fevers on a mean of just under 1 severe fever episode within each three-month measurement period. More importantly, these effects were almost completely mitigated by vitamin A supplementation at birth.

In Table 3, we report regressions estimated separately for each gender. Tornado exposure in the first three months of life had large deleterious impacts on both 3-month and 6-month MUAC, chest circumference, and fever episodes for male infants in the control group. In contrast, female infants in the control group were largely unaffected. Gender differences in these coefficients are statistically significant for all 3-month outcomes and for 6-month MUAC and head circumference (estimates and standard errors of the gender differences are not reported but available upon request).

For male infants supplemented with vitamin A, the negative impacts all but disappear. The coefficients on the vitamin A interaction for 0-3 month exposure are the opposite sign of the main tornado effect and large in magnitude for all outcomes; they are statistically significant for 3-month MUAC, chest circumference, and fevers, as well as 6-month MUAC. With one exception (6-month MUAC), these interaction terms are all significantly different from the corresponding estimates in the female regression, where there is no evidence of significant tornado effects in either the treatment or control group. The substantial gender differences in tornado impacts as well as vitamin A interactions may represent a manifestation of the "fragile male," the finding consistent across a wide variety of studies that boys tend to be much more susceptible to negative shocks *in utero* and in early life than girls (Catalano et al., 2006; Kraemer, 2000; Pongou, 2013).

4.3 Checks

We perform a variety of checks on potential threats to internal validity, which are discussed below. Corresponding tables are relegated to the appendix.

4.3.1 Attrition

There are two forms of attrition that are relevant in our study context. First, since we are able to observe and track every pregnancy from its inception, we can identify attrition from the sample due to fetal death (miscarriage or abortion) and stillbirth. Second, for live births, there is additional attrition due to infant death or loss to follow up (e.g., if the household could not be located at 3 or 6 months following the infant's birth). If either of these types of attrition is affected by tornado exposure or its interaction with vitamin A treatment status, it is possible that this differential sample selection could be driving our results.

In Table A2, we examine the first type of attrition by estimating the effect of *in utero* exposure to the tornado on miscarriages, abortions, and live births.[9] The main takeaway from this table is that *in utero* exposure to the tornado did not significantly affect the probability of miscarriage or abortion, and thus (since the live birth dummy is nearly collinear with the sum of miscarriage and abortion) also did not affect live births.

Next, to investigate the second type of attrition, we estimate specification (1), using dummies for whether 3-month and 6-month measures were missing as the outcome variables. This allows us to estimate whether exposure to the tornado (in any early life period – *in utero*, first three months, or first six months) affected attrition, and whether this effect differed across Vitamin A and placebo locations. In Table A3, we find that attrition of live births is not significantly different across tornado-exposed and unexposed infants, nor is it different by vitamin A group interactions with tornado exposure. Taken together, the evidence in Tables A2 and A3 suggests that our estimates are not being driven by selective attrition.

[9]Note that we do not separately estimate selection due to stillbirth because less than 3 percent of pregnancies resulted in stillbirth; however, this variation is captured in the "0" category of the live birth dummy.

4.3.2 Dosing

According to the trial protocol, infants were to be dosed within hours of birth with either treatment (vitamin A) or placebo. 56 percent of infants were dosed within 12 hours of birth, but the dose timing distribution has a long right tail, with 24 percent of infants dosed more than 7 days after birth. Because the trial was double blind, the implementation teams did not know whether they were dosing infants with treatment or placebo.

Table A4 investigates whether dosing timing was affected by the tornado and whether this differed for Vitamin A and control infants. We report the results of specification (1), focusing on *in utero* exposure. Our first outcome variable is an indicator for whether the infant was dosed at all. Next, restricting to infants who were dosed, we investigate whether dosing timing (measured by age in days at dosing) was affected. Neither the probability of dosing nor dosing timing was significantly affected by tornado exposure or its interaction with vitamin A treatment status.

The fact that there is no difference in dosing timing across tornado exposure categories is reassuring, given the possible concern that the tornado may have caused delays in trial administration. The fact that there are no significant interactions with vitamin A treatment reflects the double-blind nature of the trial.

5 Discussion

This study demonstrates that a health intervention at birth can strengthen resilience to exposure to natural disasters in early life. Our results support a novel role for vitamin A, given at birth as a single large dose, in strengthening the physiological resilience of infants exposed to a devastating tornado. We were not able to assess precisely through what mechanism the observed effects may have occurred, but they are likely due to stronger resistance to infection.

This is important because improving the health and survival of infants, particularly in low-income countries, is a primary goal for global health policy. Moreover, a growing literature in economics shows that in addition to these immediate impacts, early life insults have far-reaching long run consequences. Disease (Almond, 2006; Bleakley, 2007, 2010; Cutler et al., 2010), natural disasters (Currie and Rossin-Slater, 2013), income shocks (Maccini and Yang, 2009), and conflict (Akresh et al., 2012) all leave lasting scars on health, human capital, and wellbeing that persist over

the lifecourse. The role of public policy in mitigating these impacts or protecting against them is widely recognized but poorly understood. In large part, the dearth of rigorous evidence on policy levers is due to the difficulty in finding overlapping episodes of early life trauma and orthogonal variation that changes investments in children.

Taking a step toward filling this gap, this study demonstrates strong protective effects of one-time vitamin A supplementation at birth. Our findings suggest that much of the impact of supplementation, at least on reliable predictors of infant and child mortality such as MUAC, can be attributed to the large benefits accruing to the most distressed infants (in this case, to tornado-affected infants). To enhance their impact, supplementation policies should thus target distressed infants, particularly those living through traumatic experiences in the first few months of life.

References

Adhvaryu, Achyuta, Anant Nyshadham, Teresa Molina, and Jorge Tamayo, "Helping children catch up: Early life shocks and the progresa experiment," Technical Report, National Bureau of Economic Research 2018.

Akresh, Richard, Sonia Bhalotra, Marinella Leone, and Una Okonkwo Osili, "War and Stature: Growing Up during the Nigerian Civil War," *The American Economic Review*, 2012, *102* (3), 273–277.

Almond, Douglas, "Is the 1918 Influenza pandemic over? Long-term effects of in utero Influenza exposure in the post-1940 US population," *Journal of Political Economy*, 2006, *114* (4), 672–712.

_ and Bhashkar Mazumder, "Fetal Origins and Parental Responses," *Annual Review of Economics*, 2013, *5* (1), 37–56.

_ and Janet Currie, "Killing me softly: The fetal origins hypothesis," *The Journal of Economic Perspectives*, 2011, *25* (3), 153–172.

_ , _ , and Valentina Duque, "Childhood circumstances and adult outcomes: Act II," *Journal of Economic Literature*, 2018, *56* (4), 1360–1446.

Awasthi, Shally, Richard Peto, Simon Read, Sarah Clark, Vinod Pande, Donald Bundy, DEVTA (Deworming, Enhanced Vitamin A) team et al., "Vitamin A supplementation every 6 months with retinol in 1 million pre-school children in north India: DEVTA, a cluster-randomised trial," *The Lancet*, 2013, *381* (9876), 1469–1477.

Barclay, AJ, Allen Foster, and Alfred Sommer, "Vitamin A supplements and mortality related to measles: a randomised clinical trial.," *Br Med J (Clin Res Ed)*, 1987, *294* (6567), 294–296.

Bhutta, Zulfiqar A, Jai K Das, Arjumand Rizvi, Michelle F Gaffey, Neff Walker, Susan Horton, Patrick Webb, Anna Lartey, Robert E Black, The Lancet Nutrition Interventions Review Group et al., "Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost?," *The Lancet*, 2013, *382* (9890), 452–477.

_ , Sergio Cabral, Chok wan Chan, and William J Keenan, "Reducing maternal, newborn, and infant mortality globally: an integrated action agenda," *International Journal of Gynecology & Obstetrics*, 2012, *119*, S13–S17.

Bitler, Marianne P, Hilary W Hoynes, and Thurston Domina, "Experimental evidence on distributional effects of head start," Technical Report, National Bureau of Economic Research 2014.

Bleakley, Hoyt, "Disease and development: evidence from hookworm eradication in the American South," *The Quarterly Journal of Economics*, 2007, *122* (1), 73–117.

_ , "Malaria Eradication in the Americas: A Retrospective Analysis of Childhood Exposure," *American Economic Journal: Applied Economics*, 2010, *2* (2), 1–45.

Burke, Marshall, Solomon M Hsiang, and Edward Miguel, "Global non-linear effect of temperature on economic production," *Nature*, 2015, *527* (7577), 235.

Cas, Ava Gail, Elizabeth Frankenberg, Wayan Suriastini, and Duncan Thomas, "The impact of parental death on child well-being: evidence from the Indian Ocean tsunami," *Demography*, 2014, *51* (2), 437–457.

Catalano, Ralph, Tim Bruckner, Amy R Marks, and Brenda Eskenazi, "Exogenous shocks to the human sex ratio: the case of September 11, 2001 in New York City," *Human reproduction*, 2006, *21* (12), 3127–3131.

Christian, P, R Klemm, A A Shamim, H Ali, M Rashid, S Shaikh, L Wu, S Mehra, A Labrique, J Katz, and K P West, "Effects of vitamin A and beta-carotene supplementation on birth size and length of gestation in rural Bangladesh: a cluster-randomized trial," *American Journal of Clinical Nutrition*, January 2013, *97* (1), 188–194.

Cooperman, Alicia Dailey, "Randomization inference with rainfall data: Using historical weather patterns for variance estimation," *Political Analysis*, 2017, *25* (3), 277–288.

Cornelissen, Thomas, Christian Dustmann, Anna Raute, and Uta Schönberg, "Who benefits from universal child care? Estimating marginal returns to early child care attendance," *Journal of Political Economy*, 2018, *126* (6), 2356–2409.

CRED/UNISDR, "Poverty and death: Disaster mortality 1996–2015," 2016.

Cunha, Flavio, James J Heckman, and Susanne M Schennach, "Estimating the technology of cognitive and noncognitive skill formation," *Econometrica*, 2010, *78* (3), 883–931.

Cunningham-Rundles, Susanna, Hong Lin, Deborah Ho-Lin, Ann Dnistrian, Barrie R Cassileth, and Jeffrey M Perlman, "Role of nutrients in the development of neonatal immune response," *Nutrition reviews*, 2009, *67* (suppl_2), S152–S163.

Currie, Janet, "Child health in developed countries," *Handbook of health economics*, 2000, *1*, 1053–1090.

_ and Maya Rossin-Slater, "Weathering the storm: Hurricanes and birth outcomes," *Journal of health economics*, 2013, *32* (3), 487–503.

_ and Tom Vogl, "Early-Life Health and Adult Circumstance in Developing Countries," *Annu. Rev. Econ*, 2013, *5*, 1–36.

Cutler, David, Winnie Fung, Michael Kremer, Monica Singhal, and Tom Vogl, "Early life Malaria Exposure and Adult Outcomes: Evidence from Malaria Eradication in India," *American Economic Journal: Applied Economics*, 2010, *2* (2), 72–94.

Dewey, Kathryn G and Daniel R Mayers, "Early child growth: how do nutrition and infection interact?," *Maternal & child nutrition*, 2011, *7*, 129–142.

Donnen, Philippe, Daniel Brasseur, Michele Dramaix, Francoise Vertongen, Mweze Zihindula, Mbasha Muhamiriza, and Philippe Hennart, "Vitamin A supplementation but not deworming improves growth of malnourished preschool children in eastern Zaire," *The Journal of nutrition*, 1998, *128* (8), 1320–1327.

Duque, Valentina, Maria Rosales-Rueda, Fabio Sanchez et al., "How do early-life shocks interact with subsequent human-capital investments? Evidence from administrative data," in "IZA World of Labor Conference" 2018.

Field, Christopher B, *Managing the risks of extreme events and disasters to advance climate change adaptation: special report of the intergovernmental panel on climate change*, Cambridge University Press, 2012.

Frankenberg, Elizabeth, Jed Friedman, Thomas Gillespie, Nicholas Ingwersen, Robert Pynoos, Iip Umar Rifai, Bondan Sikoki, Alan Steinberg, Cecep Sumantri, Wayan Suriastini et al., "Mental health in Sumatra after the tsunami," *American journal of public health*, 2008, *98* (9), 1671–1677.

Gunnsteinsson, Snaebjorn, Alain B Labrique, Keith P West, Parul Christian, Sucheta Mehra, Abu Ahmed Shamim, Mahbubur Rashid, Joanne Katz, and Rolf D W Klemm, "Constructing indices of rural living standards in Northwestern Bangladesh.," *JOURNAL OF HEALTH POPULATION AND NUTRITION*, October 2010, *28* (5), 509–519.

Haider, Batool A and Zulfiqar A Bhutta, "Neonatal vitamin A supplementation for the prevention of mortality and morbidity in term neonates in developing countries," *Cochrane Database Syst Rev*, 2011, *10.*

Heckman, James J, "Skill formation and the economics of investing in disadvantaged children," *Science*, 2006, *312* (5782), 1900–1902.

_ , "The economics, technology, and neuroscience of human capability formation," *Proceedings of the national Academy of Sciences*, 2007, *104* (33), 13250–13255.

Heß, Simon, "Randomization inference with Stata: A guide and software," *The Stata Journal*, 2017, *17* (3), 630–651.

Hornbeck, Richard, "The enduring impact of the American Dust Bowl: Short-and long-run adjustments to environmental catastrophe," *American Economic Review*, 2012, *102* (4), 1477–1507.

Huang, Zhiyi, Yu Liu, Guangying Qi, David Brand, and Song Guo Zheng, "Role of vitamin A in the immune system," *Journal of clinical medicine*, 2018, *7* (9), 258.

Humphrey, Jean H, Tina Agoestina, Lee Wu, Ali Usman, Muhammad Nurachim, Dedi Subardja, Syarief Hidayat, James Tielsch, Keith P West, and Alfred Sommer, "Impact of neonatal vitamin A supplementation on infant morbidity and mortality," *The Journal of Pediatrics*, April 1996, *128* (4), 489–496.

Hussey, Gregory D and Max Klein, "A randomized, controlled trial of vitamin A in children with severe measles," *New England journal of medicine*, 1990, *323* (3), 160–164.

Institute of Medicine (US) Panel on Micronutrients, "Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc," Technical Report, Washington (DC) 2001.

Jayachandran, Seema and Rohini Pande, "Why are Indian children so short? The role of birth order and son preference," *American Economic Review*, 2017, *107* (9), 2600–2629.

Jr, Keith P West, Joanne Katz, Sharada Ram Shrestha, Steven C LeClerq, Subarna K Khatry, Elizabeth K Pradhan, Ramesh Adhikari, LS Wu, Ram Prasad Pokhrel, and Alfred Sommer, "Mortality of infants< 6 mo of age supplemented with vitamin A: a randomized, double-masked trial in Nepal," *The American journal of clinical nutrition*, 1995, *62* (1), 143–148.

Karbownik, Krzysztof and Anthony Wray, "Long-run consequences of exposure to natural disasters," *Journal of Labor Economics*, 2019, *37* (3), 949–1007.

Klemm, R D W, A B Labrique, P Christian, M Rashid, A A Shamim, J Katz, A Sommer, and K P West, "Newborn Vitamin A Supplementation Reduced Infant Mortality in Rural Bangladesh," *PEDIATRICS*, July 2008, *122* (1), e242–e250.

Kraemer, Sebastian, "The fragile male," *British Medical Journal*, 2000, *321* (7276), 1609.

Labrique, Alain B, Parul Christian, Rolf DW Klemm, Mahbubur Rashid, Abu A Shamim, Allan Massie, Kerry Schulze, Andre Hackman, and Keith P West, "A cluster-randomized, placebo-controlled, maternal vitamin A or beta-carotene supplementation trial in Bangladesh: design and methods," *Trials*, 2011, *12* (1), 102.

Linnerooth-Bayer, Joanne, Reinhard Mechler, and Georg Pflug, "Refocusing disaster aid," *Science*, 2005, *309* (5737), 1044–1046.

Liu, Li, Shefali Oza, Daniel Hogan, Jamie Perin, Igor Rudan, Joy E Lawn, Simon Cousens, Colin Mathers, and Robert E Black, "Global, regional, and national causes of child mortality in 2000–13, with projections to inform post-2015 priorities: an updated systematic analysis," *The Lancet*, 2015, *385* (9966), 430–440.

Lozano, Rafael, Haidong Wang, Kyle J Foreman, Julie Knoll Rajaratnam, Mohsen Naghavi, Jake R Marcus, Laura Dwyer-Lindgren, Katherine T Lofgren, David Phillips, Charles Atkinson et al., "Progress towards Millennium Development Goals 4 and 5 on maternal and child mortality: an updated systematic analysis," *The Lancet*, 2011, *378* (9797), 1139–1165.

Macallan, Derek, "Infection and malnutrition," *Medicine*, 2009, *37* (10), 525–528.

Maccini, Sharon and Dean Yang, "Under the Weather: Health, Schooling, and Economic Consequences of Early-Life Rainfall," *The American Economic Review*, June 2009, *99* (3), 1006–1026.

Mayo-Wilson, Evan, Aamer Imdad, Kurt Herzer, Mohammad Yawar Yakoob, and Zulfiqar A Bhutta, "Vitamin A supplements for preventing mortality, illness, and blindness in children aged under 5: systematic review and meta-analysis," *Bmj*, 2011, *343.*

Mazumder, Sarmila, Sunita Taneja, Kiran Bhatia, Sachiyo Yoshida, Jasmine Kaur, Brinda Dube, G S Toteja, Rajiv Bahl, Olivier Fontaine, Jose Martines, and Nita Bhandari, "Efficacy of early neonatal supplementation with vitamin A to reduce mortality in infancy in Haryana, India (Neovita): a randomised, double-blind, placebo-controlled trial," *The Lancet*, April 2015, *385* (9975), 1333–1342.

Pongou, Roland, "Why is infant mortality higher in boys than in girls? A new hypothesis based on preconception environment and evidence from a large sample of twins," *Demography*, 2013, *50* (2), 421–444.

Rahmathullah, Lakshmi, James M Tielsch, R D Thulasiraj, Joanne Katz, Christian Coles, Sheela Devi, Rajeesh John, Karthik Prakash, A V Sadanand, N Edwin, and C Kamaraj, "Impact of supplementing newborn infants with vitamin A on early infant mortality: community based randomised trial in southern India," *Bmj*, July 2003, *327* (7409), 254–0.

Rossin-Slater, Maya and Miriam Wüst, "Parental responses to child support obligations: Evidence from administrative data," *Journal of Public Economics*, 2018, *164*, 183–196.

Semba, Richard D, "Vitamin A, immunity, and infection," *Clinical Infectious Diseases*, 1994, *19* (3), 489–499.

Sommer, Alfred, Keith P West, James A Olson, and A Catherine Ross, *Vitamin A deficiency: health, survival, and vision*, Oxford University Press, USA, 1996.

Stephensen, Charles B, "Vitamin A, infection, and immune function*," *Annual review of nutrition*, 2001, *21* (1), 167–192.

Sugimoto, Jonathan D, Alain B Labrique, Salahuddin Ahmad, Mahbubur Rashid, Abu Ahmed Shamim, Barkat Ullah, Rolf D W Klemm, Parul Christian, and Keith P West, "Epidemiology of tornado destruction in rural northern Bangladesh: risk factors for death and injury.," *Disasters*, April 2011, *35* (2), 329–345.

Thurnham, D I, C A Northrop-Clewes, F S W McCullough, B S Das, and P G Lunn, "Innate Immunity, Gut Integrity, and Vitamin A in Gambian and Indian Infants," *Journal of Infectious Diseases*, September 2000, *182* (Supplement 1), S23–S28.

Tielsch, James M, Lakshmi Rahmathullah, R D Thulasiraj, Joanne Katz, Christian Coles, S Sheeladevi, Rajeesh John, and Karthik Prakash, "Newborn vitamin A dosing reduces the case fatality but not incidence of common childhood morbidities in South India.," *The Journal of Nutrition*, November 2007, *137* (11), 2470–2474.

U.S. Department of Agriculture, "Composition of Foods Raw, Processed, Prepared USDA National Nutrient Database for Standard Reference, Release 24," September 2011.

Wang, Haidong, Chelsea A Liddell, Matthew M Coates, Meghan D Mooney, Carly E Levitz, Austin E Schumacher, Henry Apfel, Marissa Iannarone, Bryan Phillips, Katherine T Lofgren et al., "Global, regional, and national levels of neonatal, infant, and under-5 mortality during 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013," *The Lancet*, 2014, *384* (9947), 957–979.

West, Keith P, "Dietary vitamin-A deficiency: Effects on growth, infection, and mortality," *Food and nutrition bulletin*, 1991, *13* (2), 1–12.

_ , Edi Djunaedi, Akbar Pandji, I Tarwotjo, and Alfred Sommer, "Vitamin A supplementation and growth: a randomized community trial," *The American journal of clinical nutrition*, 1988, *48* (5), 1257–1264.

_ , Parul Christian, Alain B Labrique, Mahbubur Rashid, Abu Ahmed Shamim, Rolf DW Klemm, Allan B Massie, Sucheta Mehra, Kerry J Schulze, and Hasmot Ali, "Effects of vitamin A or beta carotene supplementation on pregnancy-related mortality and infant mortality in rural Bangladesh," *JAMA*, 2011, *305* (19), 1986.

Zeba, Augustin N, Hermann Sorgho, Noël Rouamba, Issiaka Zongo, Jeremie Rouamba, Robert T Guiguemdé, Davidson H Hamer, Najat Mokhtar, and Jean-Bosco Ouedraogo, "Major reduction of malaria morbidity with combined vitamin A and zinc supplementation in young children in Burkina Faso: a randomized double blind trial," *Nutrition journal*, 2008, *7* (1), 1–7.

Tables and Figures

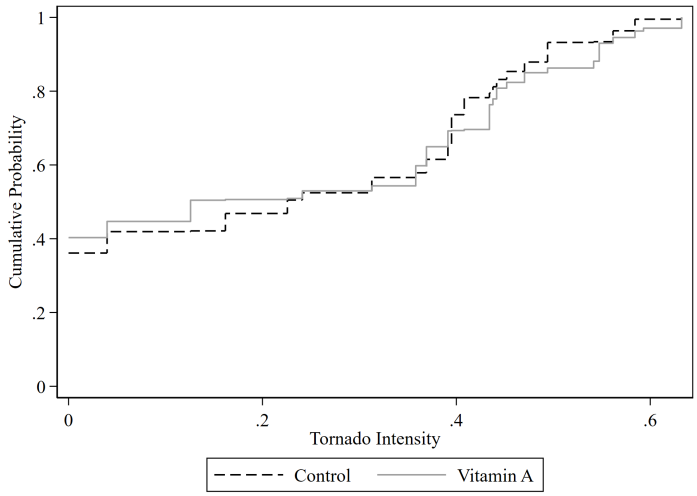


Figure 1: Tornado Intensity by Treatment Group

Tornado intensity is equal to zero for households further than 1 km from the tornado path (defined in section 3). For households within 1 km of the tornado path, tornado intensity is equal to the share of houses destroyed within the 1km radius area to which the household belongs. This figure restricts to households within 1.6 km of the tornado path.

Table 1: **Summary Statistics of Infants in the Pre-tornado Cohorts**

	All	Tornado	Non-Tornado	Diff.	Vitamin A	Placebo	Diff.
Weight at birth (kg)	2.50	2.51	2.50	0.01	2.50	2.49	0.01
	(0.43)	(0.42)	(0.43)	(0.03)	(0.43)	(0.44)	(0.02)
Height at birth (cm)	46.66	46.66	46.66	-0.00	46.71	46.62	0.09
	(2.37)	(2.23)	(2.38)	(0.19)	(2.39)	(2.34)	(0.10)
MUAC at birth (cm)	9.43	9.47	9.43	0.04	9.42	9.45	-0.03
	(0.86)	(0.91)	(0.86)	(0.07)	(0.84)	(0.87)	(0.03)
Head circumference at birth (cm)	32.68	32.70	32.68	0.02	32.71	32.65	0.06
	(1.62)	(1.73)	(1.61)	(0.13)	(1.61)	(1.63)	(0.06)
Chest circumference at birth (cm)	30.75	30.76	30.74	0.02	30.76	30.73	0.02
	(2.13)	(2.08)	(2.14)	(0.17)	(2.12)	(2.14)	(0.09)
MUAC at 3 mon. (cm)	12.39	12.42	12.39	0.03	12.40	12.40	0.00
	(1.07)	(1.10)	(1.07)	(0.07)	(1.07)	(1.08)	(0.03)
Head circumference at 3 mon. (cm)	38.73	38.65	38.74	-0.09	38.72	38.76	-0.04
	(1.50)	(1.53)	(1.50)	(0.10)	(1.46)	(1.53)	(0.05)
Chest circumference at 3 mon. (cm)	38.90	38.99	38.89	0.10	38.91	38.89	0.01
	(2.25)	(2.29)	(2.25)	(0.15)	(2.21)	(2.29)	(0.07)
Fever Episodes: 0-3 mon.	1.00	0.95	1.01	-0.06	0.99	1.02	-0.03
	(1.08)	(1.01)	(1.09)	(0.07)	(1.06)	(1.10)	(0.03)
MUAC at 6 mon. (cm)	13.09	13.16	13.09	0.07	13.11	13.08	0.03
	(1.06)	(1.06)	(1.06)	(0.07)	(1.07)	(1.05)	(0.03)
Head circumference at 6 mon. (cm)	40.88	40.98	40.87	0.11	40.88	40.88	0.00
	(1.43)	(1.38)	(1.43)	(0.09)	(1.43)	(1.42)	(0.05)
Chest circumference at 6 mon. (cm)	41.31	41.39	41.30	0.09	41.33	41.29	0.04
	(2.14)	(2.12)	(2.14)	(0.14)	(2.16)	(2.12)	(0.07)
Fever Episodes: 4-6 mon.	0.84	0.91	0.84	0.07	0.83	0.85	-0.02
	(0.98)	(1.02)	(0.98)	(0.06)	(1.00)	(0.96)	(0.03)
Male	0.51	0.54	0.51	0.03	0.52	0.51	0.01
	(0.50)	(0.50)	(0.50)	(0.03)	(0.50)	(0.50)	(0.01)
Died by 6 mon. of age	0.06	0.05	0.06	-0.00	0.05	0.06	-0.01
	(0.23)	(0.22)	(0.23)	(0.01)	(0.23)	(0.24)	(0.01)
Parity	1.36	1.30	1.36	-0.06	1.31	1.41	-0.11
	(2.51)	(1.54)	(2.57)	(0.15)	(2.49)	(2.54)	(0.07)
Living Standards Index	-0.07	-0.11	-0.07	-0.04	-0.08	-0.06	-0.02
	(0.98)	(0.97)	(0.98)	(0.06)	(0.97)	(0.99)	(0.03)
Maternal height	149.30	149.13	149.31	-0.19	149.23	149.37	-0.15
	(5.13)	(5.13)	(5.13)	(0.30)	(5.12)	(5.15)	(0.15)
Maternal MUAC	22.60	22.61	22.60	0.01	22.64	22.57	0.07
	(1.93)	(1.97)	(1.92)	(0.11)	(1.90)	(1.95)	(0.06)
Mother's Education	3.54	3.49	3.54	-0.06	3.57	3.53	0.04
	(4.01)	(3.92)	(4.02)	(0.24)	(4.01)	(4.02)	(0.12)
Age at dosing (days)	3.15	2.62	3.19	-0.57	3.09	3.20	-0.11
	(7.72)	(6.79)	(7.78)	(0.56)	(7.73)	(7.69)	(0.28)
Observations	4659	303	4356		2307	2352	

Sample restricts to infants born at least 7 months before the tornado. Tornado and Non-Tornado refer to inside vs. outside the tornado area. Observation counts report the total number of individuals in each group; the number of non-missing observations varies across variables. OLS standard errors and associated p-values reported.
Significance: * < 0.1; ** < 0.05; *** < 0.01.

Table 2: Impact of Tornado and Vitamin A on Infant Health

	Outcomes assessed at 3 months				Outcomes assessed at 6 months			
	MUAC	Head Circ.	Chest Circ.	Fevers	MUAC	Head Circ.	Chest Circ.	Fevers
In tornado area X ...								
In utero	−0.04	0.13	−0.02	0.20*	−0.01	0.16	0.06	0.07
	(0.13)	(0.15)	(0.25)	(0.12)	(0.12)	(0.15)	(0.25)	(0.10)
Age 0-3 months	−0.35***	−0.17	−0.99***	0.50***	−0.37***	−0.28	−0.65**	0.45***
	(0.13)	(0.19)	(0.32)	(0.13)	(0.14)	(0.21)	(0.32)	(0.17)
Age 4-6 months					−0.08	0.14	−0.28	0.24*
					(0.15)	(0.19)	(0.34)	(0.13)
In tornado area X Vitamin A X ...								
In utero	0.09	−0.07	0.00	−0.01	0.12	−0.19	0.10	0.15
	(0.16)	(0.21)	(0.33)	(0.15)	(0.16)	(0.21)	(0.35)	(0.15)
Age 0-3 months	0.40**	0.24	0.88*	−0.47***	0.49**	0.41	0.80*	−0.26
	(0.20)	(0.28)	(0.47)	(0.18)	(0.21)	(0.29)	(0.45)	(0.20)
Age 4-6 months					0.02	−0.19	0.28	0.05
					(0.20)	(0.28)	(0.47)	(0.20)
Dependent variable mean	12.19	38.44	38.62	0.91	13.02	40.82	41.29	0.94
Observations	16509	16487	16003	16811	16247	16156	15610	16636

This table reports the results of the triple-difference specification in (2). All regressions control for age at time of measurement. MUAC, head circumference, and chest circumference are measured in cm. Fevers measure the number of fever episodes in months 0-3 (for 3 months) and 4-6 (for 6 months), top coded at 4. Standard errors are computed using the randomization inference procedure described in section 3.3.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 3: Impact of Tornado and Vitamin A by Gender

	Males								Females							
	Outcomes assessed at 3 months				Outcomes assessed at 6 months				Outcomes assessed at 3 months				Outcomes assessed at 6 months			
	MUAC	Head Circ.	Chest Circ.	Fevers	MUAC	Head Circ.	Chest Circ.	Fevers	MUAC	Head Circ.	Chest Circ.	Fevers	MUAC	Head Circ.	Chest Circ.	Fevers
In tornado area X ...																
In utero	-0.13	0.15	0.15	0.30*	-0.06	0.14	0.07	0.08	0.04	0.05	-0.26	0.07	0.02	0.12	-0.07	0.07
	(0.16)	(0.19)	(0.32)	(0.16)	(0.16)	(0.18)	(0.33)	(0.15)	(0.15)	(0.18)	(0.34)	(0.15)	(0.16)	(0.20)	(0.34)	(0.14)
Age 0-3 months	-0.61***	-0.38	-1.35***	0.85***	-0.57***	-0.49*	-0.85**	0.53**	-0.03	0.17	-0.52	0.14	-0.12	0.03	-0.38	0.39**
	(0.20)	(0.32)	(0.44)	(0.20)	(0.17)	(0.29)	(0.42)	(0.24)	(0.19)	(0.24)	(0.42)	(0.17)	(0.21)	(0.28)	(0.41)	(0.19)
Age 4-6 months					-0.12	0.24	-0.29	0.30					-0.03	0.02	-0.27	0.16
					(0.21)	(0.28)	(0.44)	(0.19)					(0.23)	(0.24)	(0.48)	(0.17)
In tornado area X Vitamin A X ...																
In utero	0.30	0.09	-0.12	-0.09	0.11	-0.13	-0.43	0.14	-0.09	-0.09	0.39	0.08	0.17	-0.13	0.78*	0.16
	(0.21)	(0.28)	(0.47)	(0.20)	(0.22)	(0.26)	(0.47)	(0.22)	(0.21)	(0.28)	(0.43)	(0.22)	(0.21)	(0.29)	(0.46)	(0.21)
Age 0-3 months	0.76***	0.60	1.31**	-0.71**	0.51*	0.43	0.40	-0.43	0.04	-0.11	0.48	-0.20	0.43	0.30	1.11*	-0.09
	(0.28)	(0.38)	(0.60)	(0.28)	(0.27)	(0.38)	(0.59)	(0.31)	(0.29)	(0.37)	(0.60)	(0.25)	(0.30)	(0.38)	(0.60)	(0.26)
Age 4-6 months					-0.20	-0.60	-0.38	-0.17					0.17	0.02	0.71	0.32
					(0.27)	(0.39)	(0.63)	(0.28)					(0.28)	(0.36)	(0.59)	(0.25)
Dependent variable mean	12.41	38.93	39.22	0.94	13.24	41.38	41.93	0.99	11.96	37.93	37.99	0.88	12.79	40.25	40.64	0.89
Observations	8400	8392	8123	8577	8248	8210	7922	8462	8109	8095	7880	8234	7999	7946	7688	8174

This table reports the results of the triple-difference specification in (2). All regressions control for age at time of measurement. MUAC, head circumference, and chest circumference are measured in cm. Fevers measure the number of fever episodes in months 0-3 (for 3 months) and 4-6 (for 6 months), top coded at 4. Standard errors are computed using the randomization inference procedure described in section 3.3.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Online Appendix

A Appendix Figures and Tables

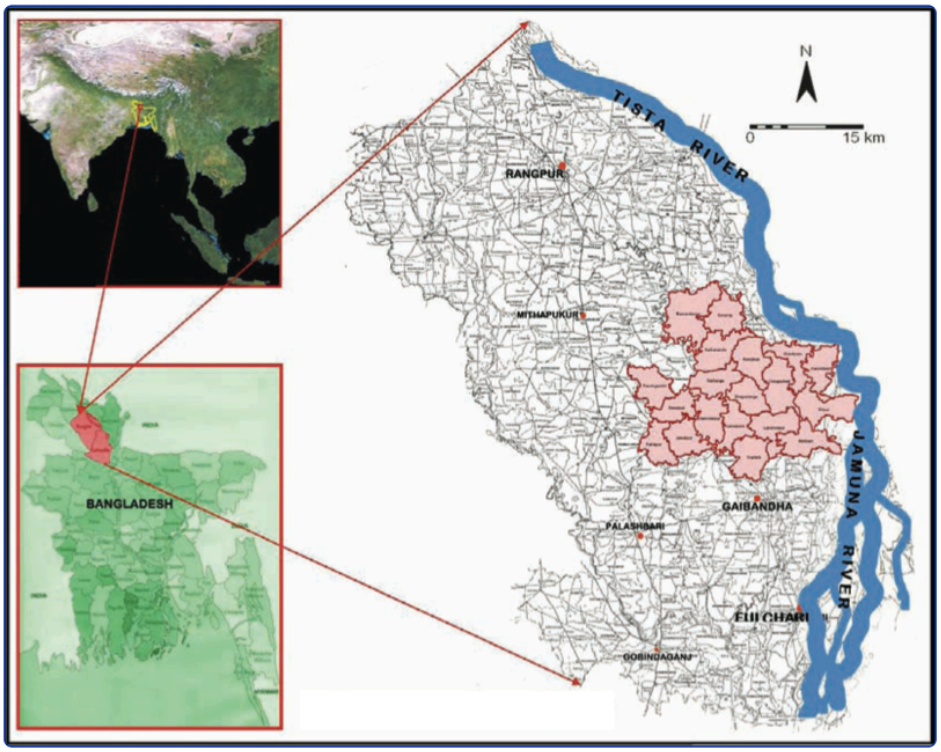


Figure A1: Location of the study area. The figure was produced by the JiVitA GIS Unit.

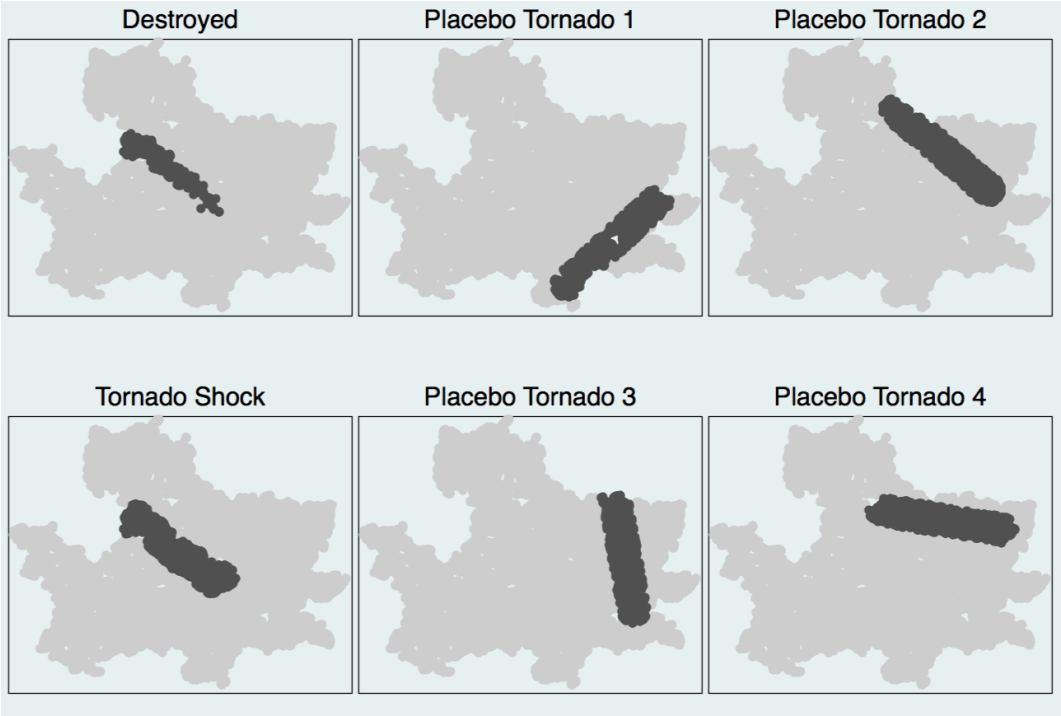


Figure A2: This figure depicts the houses destroyed in the tornado (top left), the definition of tornado exposed (bottom left) and four examples of placebo tornados, each in dark gray (other houses in the study are depicted in light gray).

Table A1: Impact of Tornado on Birth Outcomes

	Birth Weight (kg)	BW < 2.5kg	BW < 2kg	Birth Length (cm)	GA (weeks)	Premature
In tornado area x in utero	-0.07*	0.08**	0.06**	-0.44**	-0.02	0.02
	(0.04)	(0.04)	(0.03)	(0.22)	(0.23)	(0.03)
Dependent variable mean	2.43	0.55	0.14	46.44	37.64	0.29
Observations	13909	13909	13909	13545	18879	18879

This table reports the coefficient on the interaction between E and $C_{inutero}$ in a regression that controls for the main effects of E and $C_{inutero}$. GA denotes gestational age in weeks and Premature is an indicator for births before 37 weeks. Standard errors are computed using the randomization inference procedure described in section 3.3.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table A2: Impact of Tornado on Miscarriage and Stillbirth

	Miscarriage	Abortion	Live birth
In tornado area X in-utero	0.005	-0.016	0.017
	(0.013)	(0.016)	(0.022)
Dependent variable mean	0.11	0.16	0.69
Observations	25842	25842	25842

This table reports the coefficient on the interaction between E and $C_{inutero}$ in a regression that controls for the main effects of E and $C_{inutero}$. Cohorts are defined differently from other parts of the paper (since we cannot rely on birthday). The infant is defined as being in-utero if the tornado happened after the last menstrual period and before the date of pregnancy outcome. The sample for these regressions includes pregnancies, as opposed to the sample of live births used in other tables and figures. We limit the sample to pregnancies of mothers who had their last menstrual period after July 1st, 2003 (before this date the infant is unlikely to end up in the infant trial, which started in January 2004, and an exact match between the two samples is not possible given that gestational length determines in part inclusion in the infant trial around the start of the trial). Standard errors clustered at the sector level.
Significance: * $<$ 0.10; ** $<$ 0.05; *** $<$ 0.01.

Table A3: Impact of Tornado and Vitamin A on Attrition

	Missing	
	3 month measures	6 month measures
In tornado area X ...		
Ever Exposed	0.019	0.027
	(0.037)	(0.045)
In tornado area X Vitamin A X ...		
Ever Exposed	−0.004	−0.047
	(0.041)	(0.046)
Dependent variable mean	0.11	0.13
Observations	18879	18879

This table reports the results of the triple-difference specification in (1). Ever Exposed is an indicator for cohorts who were either *in utero* or between 0-6 months when the tornado hit. The dependent variables are dummies indicating missing values for 3-month and 6-month anthropometry. Standard errors are computed using the randomization inference procedure described in section 3.3.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table A4: Impact of tornado and vitamin A on dosing timing

	Dosed	Age at Dosing (Days)
In tornado area X ...		
In utero	0.01	−0.35
	(0.02)	(0.78)
In tornado area X Vitamin A X ...		
In utero	−0.04	0.79
	(0.03)	(1.16)
Dependent variable mean	0.88	3.63
Observations	18879	16704

This table reports the results of the triple-difference specification in (1), focusing on *in utero* exposure. Standard errors are computed using the randomization inference procedure described in section 3.3.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

B Identification Assumptions

In this section, to formalize the assumptions underlying our double- and triple-difference estimation, we describe the research design in a potential outcomes framework. Let Y_i^{tec} be the potential outcome for infant i given treatment ($T = t$), tornado exposure ($E = e$) and cohort ($C = c$). We define τ_i as the causal effect of the vitamin A supplementation on individual i, ω_i as the causal effect of tornado exposure, γ_i as the causal effect of the interaction of vitamin A supplementation and tornado exposure and ν_i as the causal effect of being part of cohort $C = 1$ relative to $C = 0$ due to aggregate changes (e.g., seasonality or other aggregate conditions affecting all individuals in the study area). We assume that this cohort effect, ν_i, is independent of location. The potential outcomes are

Treatment Location	Tornado Location	Cohort measured before the tornado	Exposed cohort
No	No	$Y_i^{000} = \alpha + u_i$	$Y_i^{001} = \alpha + \nu_i + u_i$
Yes	No	$Y_i^{100} = \alpha + \tau_i + u_i$	$Y_i^{101} = \alpha + \nu_i + \tau_i + u_i$
No	Yes	$Y_i^{010} = \alpha + u_i$	$Y_i^{011} = \alpha + \nu_i + \omega_i + u_i$
Yes	Yes	$Y_i^{110} = \alpha + \tau_i + u_i$	$Y_i^{111} = \alpha + \nu_i + \tau_i + \omega_i + \gamma_i + u_i$

In this 2 x 2 x 2 research design we observe 8 moments that map to the OLS parameters (from equation (1), reproduced below):

$$Y = \alpha + \beta_1 T + \beta_2 T \cdot E + \beta_3 E + \beta_4 C + \beta_5 C \cdot E + \beta_6 C \cdot T + \beta_7 C \cdot T \cdot E + u$$

$$
\begin{aligned}
E[Y_i|T=0,E=0,C=0] =& \alpha && + E[u_i|T=0,E=0,C=0] \\
E[Y_i|T=1,E=0,C=0] =& \alpha+\beta_1 && + E[u_i|T=1,E=0,C=0] \\
E[Y_i|T=0,E=1,C=0] =& \alpha+\beta_3 && + E[u_i|T=0,E=1,C=0] \\
E[Y_i|T=1,E=1,C=0] =& \alpha+\beta_1+\beta_2+\beta_3 && + E[u_i|T=1,E=1,C=0] \\
E[Y_i|T=0,E=0,C=1] =& \alpha+\beta_4 && + E[u_i|T=0,E=0,C=1] \\
E[Y_i|T=1,E=0,C=1] =& \alpha+\beta_1+\beta_4+\beta_6 && + E[u_i|T=1,E=0,C=1] \\
E[Y_i|T=0,E=1,C=1] =& \alpha+\beta_3+\beta_4+\beta_5 && + E[u_i|T=0,E=1,C=1] \\
E[Y_i|T=1,E=1,C=1] =& \alpha+\beta_1+\beta_2+\beta_3 & & \\
& \quad +\beta_4+\beta_5+\beta_6+\beta_7 && + E[u_i|T=1,E=1,C=1]
\end{aligned}
$$

The treatment was randomized and the path of the tornado was certainly independent of this treatment. Actual exposure to the tornado is independent of treatment aside from possible sample selection due to the treatment. The RCT was double blind and it is unlikely parents would be able to deduce from the health of their or neighbors children whether their area was in treatment or control. The main sample selection is through mortality since the vitamin A treatment reduced mortality by 15%. The random assignment and independence of the treatment (T) and living in the tornado exposed area (E) imply that for each $\chi_i \in \{\omega_i, \nu_i, u_i\}$:

$$
\begin{aligned}
E[\chi_i|T=1,E=0,C=0] &= E[\chi_i|T=0,E=0,C=0] && (I) \\
E[\chi_i|T=1,E=1,C=0] &= E[\chi_i|T=0,E=1,C=0] && (II) \\
E[\chi_i|T=1,E=0,C=1] &= E[\chi_i|T=0,E=0,C=1] && (III) \\
E[\chi_i|T=1,E=1,C=1] &= E[\chi_i|T=0,E=1,C=1] && (IV)
\end{aligned}
$$

Given that we will not observe 3 and 6 months outcomes for those infants that die before that time, we introduce a fourth variable S that is 1 if infant i is in the sample. We assume that equations (I) - (IV) hold when conditioning on the observed sample. We examine later evidence for this

assumption. That is, we assume (Assumption 1) that

Identification Assumption 1 *For each* χ_i *in* $\{\tau_i, \omega_i, \nu_i, u_i\}$*:*

$$E[\chi_i|T=1,E=0,C=0,S=1] = E[\chi_i|T=0,E=0,C=0,S=1] \qquad (I')$$

$$E[\chi_i|T=1,E=1,C=0,S=1] = E[\chi_i|T=0,E=1,C=0,S=1] \qquad (II')$$

$$E[\chi_i|T=1,E=0,C=1,S=1] = E[\chi_i|T=0,E=0,C=1,S=1] \qquad (III')$$

$$E[\chi_i|T=1,E=1,C=1,S=1] = E[\chi_i|T=0,E=1,C=1,S=1] \qquad (IV')$$

We now link the observed moments to the main structural parameters of interest: τ_i, ω_i and γ_i. We start with the identification of the causal effect of the vitamin A supplementation (τ_i). We have

$$\begin{aligned} &E[Y_i|T=1,E=0,C=0,S=1] - E[Y_i|T=0,E=0,C=0,S=1] \\ &\qquad = \beta_1 + \underbrace{E[u_i|T=1,E=0,C=0,S=1] - E[u_i|T=0,E=0,C=0,S=1]}_{=0 \text{ by Assumption 1 (I')}} \end{aligned}$$

Therefore, by (I'),

$$\begin{aligned} \beta_1 &= E[\alpha+\tau_i+u_i|T=1,E=0,C=0,S=1] - E[\alpha+u_i|T=0,E=0,C=0,S=1] \\ &= E[\tau_i|E=0,C=0,S=1] \end{aligned}$$

That is, β_1 identifies the causal effect of the vitamin A on infants outside of the tornado affected area in the pre-tornado period. Similarly $\beta_1 + \beta_2 = E[\tau_i|E=1,C=0,S=1]$ identifies this causal effect within the tornado affected area in the pre-tornado period.

Now we consider the identification of the causal effect of the tornado on infant outcomes, ω_i. To identify this structural parameter we use the four moments (among the eight in the 2 x 2 x 2 research design) that are derived from the population that did not get supplementation and we rely on a standard parallel trends assumption for difference-in-difference estimation. Specifically, we assume that, absent the tornado the outcomes in the tornado area would have followed the same trend as the outcomes outside this area:

Identification Assumption 2

$$E[u_i|T=0,E=1,C=1,S=1] - E[u_i|T=0,E=1,C=0,S=1]$$
$$= E[u_i|T=0,E=0,C=1,S=1] - E[u_i|T=0,E=0,C=0,S=1]$$

We have

$$\begin{aligned}
A :=& E[Y_i|T=0,E=1,C=1,S=1] - E[Y_i|T=0,E=0,C=1,S=1] \\
=& \beta_3 + \beta_5 + E[u_i|T=0,E=1,C=1,S=1] - E[u_i|T=0,E=0,C=1,S=1] \\
B :=& E[Y_i|T=0,E=1,C=0,S=1] - E[Y_i|T=0,E=0,C=0,S=1] \\
=& \beta_3 + E[u_i|T=0,E=1,C=0,S=1] - E[u_i|T=0,E=0,C=0,S=1]
\end{aligned}$$

Computing $A - B$ and re-arranging terms gives

$$\begin{aligned}
\beta_5 =& E[Y_i|T=0,E=1,C=1,S=1] - E[Y_i|T=0,E=0,C=1,S=1] \\
& - \{E[Y_i|T=0,E=1,C=1,S=1] - E[Y_i|T=0,E=0,C=0,S=1]\} \\
& - \Big[E[u_i|T=0,E=1,C=1,S=1] - E[u_i|T=0,E=0,C=1,S=1] \\
& - \{E[u_i|T=0,E=1,C=0,S=1] - E[u_i|T=0,E=0,C=0,S=1]\}\Big]
\end{aligned}$$

$$\begin{aligned}
=& E[\omega_i|T=0,E=1,C=1,S=1] \\
& - \Big[E[u_i|T=0,E=1,C=1,S=1] - E[u_i|T=0,E=0,C=1,S=1] \\
& - \{E[u_i|T=0,E=1,C=0,S=1] - E[u_i|T=0,E=0,C=0,S=1]\}\Big] \\
=& E[\omega_i|T=0,E=1,C=1,S=1] \text{ by Assumption 2}
\end{aligned}$$

The OLS coefficient β_5 therefore identifies the average causal impact of the tornado $E[\omega_i|T=0,E=1,C=1,S=1]$.

Finally we link the observed moments to the structural parameter for the interaction of the vitamin A supplementation and the tornado shock, γ_i. To identify this parameter we employ all eight moments defined by the 2 x 2 x 2 research design. Taking the first difference of treatment versus control for the four cases of inside or outside the tornado area factored with cohorts measured before versus affected by the tornado we have:

$$
\begin{aligned}
C :=& E[Y_i|T=1,E=1,C=1,S=1] - E[Y_i|T=0,E=1,C=1,S=1] \\
=& \beta_1 + \beta_2 + \beta_6 + \beta_7 + E[u_i|T=1,E=1,C=1,S=1] - E[u_i|T=0,E=1,C=1,S=1] \\
=& \beta_1 + \beta_2 + \beta_6 + \beta_7 \text{ by Assumption 1 (IV')} \\
D :=& E[Y_i|T=1,E=0,C=1,S=1] - E[Y_i|T=0,E=0,C=1,S=1] \\
=& \beta_1 + \beta_6 + E[u_i|T=1,E=0,C=1,S=1] - E[u_i|T=0,E=0,C=1,S=1] \\
=& \beta_1 + \beta_6 \text{ by Assumption 1 (III')} \\
E :=& E[Y_i|T=1,E=1,C=0,S=1] - E[Y_i|T=0,E=1,C=0,S=1] \\
=& \beta_1 + \beta_2 + E[u_i|T=1,E=1,C=0,S=1] - E[u_i|T=0,E=1,C=0,S=1] \\
=& \beta_1 + \beta_2 \text{ by Assumption 1 (II')} \\
F :=& E[Y_i|T=1,E=0,C=0,S=1] - E[Y_i|T=0,E=0,C=0,S=1] \\
=& \beta_1 + E[u_i|T=1,E=0,C=0,S=1] - E[u_i|T=0,E=0,C=0,S=1] \\
=& \beta_1 \text{ by Assumption 1 (I')}
\end{aligned}
$$

Then $C - D - (E - F) = \beta_7$. For the OLS coefficient β_7 to map to the structural interaction parameter γ we need one final assumption, Assumption 3, which is:

Identification Assumption 3

$$
\begin{aligned}
&E[\tau_i|T=1,E=1,C=1,S=1] - E[\tau_i|T=1,E=1,C=0,S=1] \\
&\qquad = E[\tau_i|T=1,E=0,C=1,S=1] - E[\tau_i|T=1,E=0,C=0,S=1]
\end{aligned}
$$

This is a parallel trends assumption on the causal effect of vitamin A inside and outside the tornado area. That is, we assume that the causal effect of vitamin A for infants in the tornado area would have followed the same trend as the causal effect outside the tornado area in the absence of a

tornado. With this assumption, along with Assumption 1, we now have

$$
\begin{aligned}
\beta_7 = \quad & E[\alpha+\nu_i+\tau_i+\omega_i+\gamma_i+u_i|T=1,E=1,C=1,S=1] \Big\} \ u_i,\ \nu_i \text{ and } \omega_i \text{ cancel out} \\
& -E[\alpha+\nu_i+\omega_i+u_i|T=0,E=1,C=1,S=1] \qquad\qquad \Big\} \ \text{by Assumption 1 (IV')} \\
& +E[\alpha+\nu_i+\tau_i+u_i|T=1,E=0,C=1,S=1] \Big\} \ u_i \text{ and } \nu_i \text{ cancel out} \\
& -E[\alpha+\nu_i+u_i|T=0,E=0,C=1,S=1] \qquad\quad \Big\} \ \text{by Assumption 1 (III')} \\
& +E[\alpha+\tau_i+u_i|T=1,E=1,C=0,S=1] \Big\} \ u_i \text{ cancels out} \\
& -E[\alpha+u_i|T=0,E=1,C=0,S=1] \qquad\quad \Big\} \ \text{by Assumption 1 (II')} \\
& +E[\alpha+\tau_i+u_i|T=1,E=0,C=0,S=1] \Big\} \ u_i \text{ cancels out} \\
& -E[\alpha+u_i|T=0,E=0,C=0,S=1] \qquad\quad \Big\} \ \text{by Assumption 1 (I')}
\end{aligned}
$$

This is equal to

$$
\begin{aligned}
& E[\tau_i+\gamma_i|T=1,E=1,C=1,S=1]-E[\tau_i|T=1,E=0,C=1,S=1] \\
& -\Big[E[\tau_i|T=1,E=1,C=0,S=1]-E[\tau_i|T=1,E=0,C=0,S=1]\Big] \\
& =E[\gamma_i|T=1,E=1,C=1,S=1] \text{ by Assumption 3}
\end{aligned}
$$

In summary, under Assumptions 1 - 3, the OLS coefficients β_5 and β_7 identify the average causal effect of the tornado, $E[\omega_i|T=0,E=1,C=1,S=1]$, and the average causal effect of the interaction of the tornado and the vitamin A supplementation, $E[\gamma_i|T=1,E=1,C=1,S=1]$, respectively.

B.1 Evidence on Assumptions 1-3

Assumption 1 refers to three structural parameters, ω_i (the causal impact of the tornado), ν_i (the cohort effect; or causal impact of changes in the aggregate environment over time) and u_i (unobservables and those observables not included in the estimation). We believe that assuming that Assumption 1 holds for the cohort effect is reasonable and we do not test it specifically. For the causal impact of the tornado we only have to worry about the tornado area in the affected period (A1-IV'). The main worry here is if, by random happenstance, the tornado affected treatment

and control areas with different intensity. Figure 1, which graphs the intensity of the tornado (as measured by the share of houses destroyed by treatment sector) by treatment status, shows that this is not the case.

With respect to u_i we can test for balance in observables. In Table B1 we test balance across the four subsamples (tornado exposure versus cohort) corresponding to parts I' through IV' of Assumption 1. These are remarkably well balanced across treatment and control. The largest difference is in mother's age at enrollment but the larger coefficients are not statistically significant and the one significant coefficient means only a difference of less than 4 months in the mother's age. Based on this, it appears that Assumption 1 is reasonable.

In Figure B1 we examine the validity of Assumption 2: that in the absence of the tornado, outcomes in the tornado area would have followed a similar trend as outcomes outside this area. We show average outcomes within 20 bins and an associated regression line up to the time of the tornado for birth outcomes, up to 90 days before the tornado for 3 month outcomes and up to 180 days prior for 6 month outcomes. As the graphs show, there is little evidence of substantial differences in pre-trends. To formally test these trends and the trends in vitamin A impact we estimate:

$$Y_i = \alpha_0 + \alpha_1 E_i + \alpha_2 b_i + \alpha_3 E_i \cdot b_i + \alpha_4 T_i + \alpha_5 T_i \cdot E_i + \alpha_6 T_i \cdot b_i + \alpha_7 T_i \cdot E_i \cdot b_i + v_i \qquad (3)$$

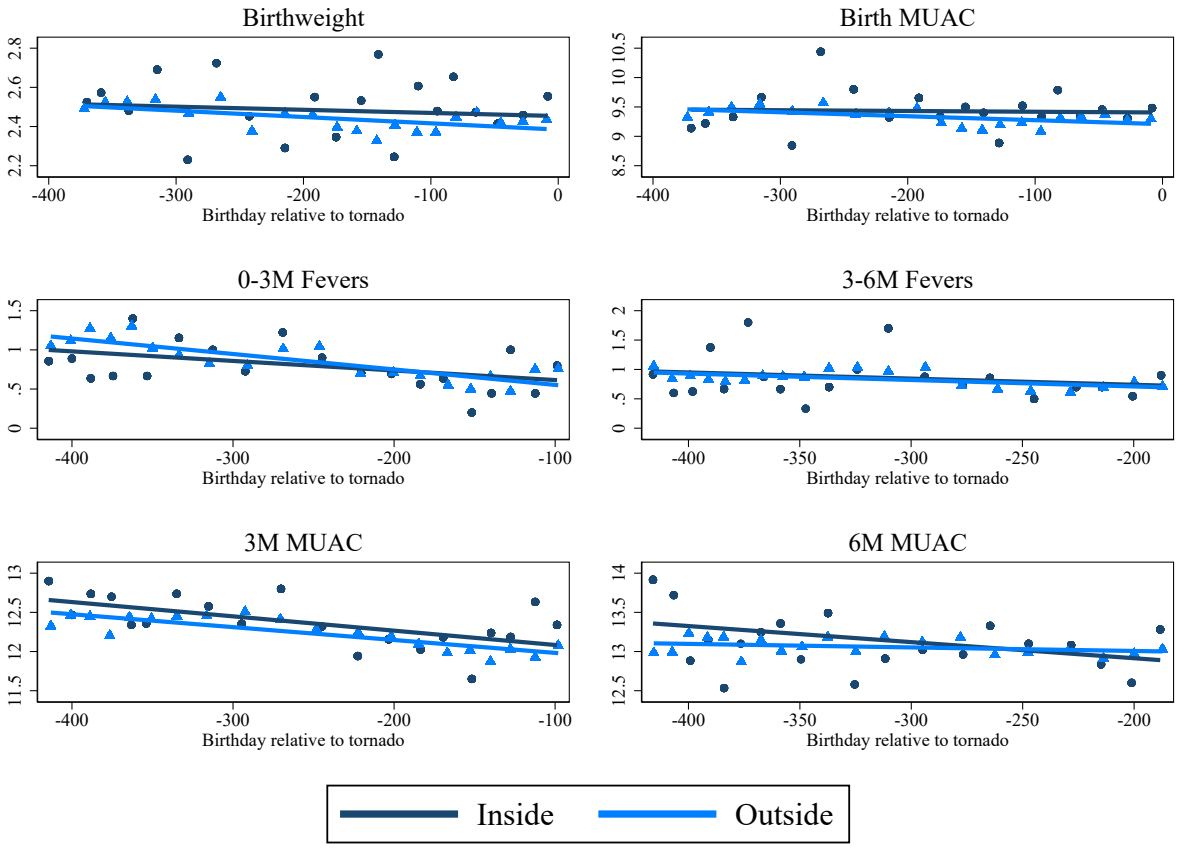
where b_i is the infants birthday coded as number of days since January 1st, 1960, and we limit the regression to those who were born before the tornado for birth outcomes, and those born more than 90 or 180 days before the tornado for 3 and 6 month outcomes. The top panel of Table B2 shows the coefficient for the difference in trends in the control group (α_3) and the second panel shows the difference in trends of the estimated treatment effect (α_7). The estimated differences in trends are in all cases less than 10% of the dependent variable standard deviation with one exception: For MUAC at 6 months the estimate for α_3 (Assumption 2) is 15% of the standard deviation and α_7 (Assumption 3) is 23% of the standard deviation. In both cases and for all outcome variables the estimates are far from reaching statistical significance. Based on this data, Assumptions 2 and 3 seem reasonable, with the possibility of some violation in the case of 6 month MUAC.

Table B1: Tests of Assumption 1 (I' - IV')

	Assumption 1 (I'): E=0,C=0			Assumption 1 (II'): E=1,C=0			Assumption 1 (III'): E=0,C=1			Assumption 1 (IV'): E=1,C=1		
Variable	Treat.	Cont.	Difference	Treat.	Cont.	Difference	Treat.	Cont.	Difference	Treat.	Cont.	Difference
Weight at birth (kg)	2.46	2.46	0.00	2.41	2.48	-0.07**	2.42	2.43	-0.01	2.45	2.47	-0.02
	(0.41)	(0.42)	(0.01)	(0.42)	(0.44)	(0.03)	(0.42)	(0.41)	(0.02)	(0.39)	(0.36)	(0.05)
MUAC at birth (cm)	9.32	9.33	-0.01	9.29	9.39	-0.10	9.23	9.27	-0.04	9.49	9.37	0.12
	(0.83)	(0.84)	(0.02)	(0.86)	(0.91)	(0.07)	(0.85)	(0.83)	(0.03)	(0.82)	(0.85)	(0.13)
Chest circ. at birth (cm)	30.49	30.48	0.01	30.39	30.66	-0.28	30.31	30.37	-0.06	30.47	30.51	-0.04
	(2.01)	(2.08)	(0.05)	(2.07)	(2.29)	(0.19)	(2.03)	(1.98)	(0.08)	(1.97)	(1.85)	(0.27)
Head circ. at birth (cm)	32.48	32.41	0.06	32.45	32.60	-0.15	32.39	32.37	0.02	32.65	32.52	0.13
	(1.56)	(1.60)	(0.04)	(1.50)	(1.70)	(0.11)	(1.59)	(1.60)	(0.06)	(1.52)	(1.39)	(0.18)
Height at birth (cm)	46.65	46.57	0.08	46.28	46.61	-0.34*	46.36	46.39	-0.03	46.32	46.52	-0.20
	(2.32)	(2.38)	(0.06)	(2.25)	(2.42)	(0.19)	(2.42)	(2.38)	(0.10)	(2.20)	(1.93)	(0.29)
Length of gestation (weeks)	37.93	37.83	0.10*	37.71	37.73	-0.02	37.75	37.82	-0.07	37.58	37.66	-0.08
	(2.83)	(2.85)	(0.06)	(2.80)	(3.02)	(0.19)	(2.90)	(2.87)	(0.10)	(3.05)	(2.82)	(0.39)
Living Standards Index	0.12	0.16	-0.04	0.07	0.04	0.02	0.08	0.09	-0.01	0.15	0.01	0.14
	(1.00)	(1.02)	(0.03)	(1.05)	(0.97)	(0.11)	(0.99)	(1.01)	(0.04)	(0.94)	(0.95)	(0.13)
Maternal MUAC	22.95	22.95	0.00	22.89	23.06	-0.17	23.06	23.01	0.06	23.01	22.79	0.22
	(1.96)	(2.03)	(0.05)	(2.13)	(2.11)	(0.17)	(2.05)	(1.99)	(0.07)	(1.93)	(1.80)	(0.26)
Maternal height	149.45	149.44	0.00	149.27	149.67	-0.40	149.59	149.53	0.05	149.72	149.32	0.40
	(5.15)	(5.13)	(0.10)	(5.13)	(5.23)	(0.40)	(5.11)	(5.25)	(0.18)	(4.96)	(4.88)	(0.60)
Mother's age at enrollment	19.59	19.80	-0.22**	19.84	19.39	0.45	19.93	19.95	-0.02	19.88	20.64	-0.76
	(4.98)	(5.15)	(0.11)	(5.20)	(4.84)	(0.40)	(5.32)	(5.33)	(0.18)	(5.86)	(5.47)	(0.62)
Mother's years of education	4.13	4.16	-0.03	4.25	3.85	0.39	4.00	4.03	-0.03	3.87	3.69	0.19
	(4.03)	(4.08)	(0.10)	(4.19)	(3.82)	(0.30)	(4.06)	(4.01)	(0.14)	(4.07)	(4.04)	(0.46)
Father's years of education	3.99	4.11	-0.12	3.90	3.98	-0.08	3.87	3.83	0.05	3.61	3.42	0.19
	(4.61)	(4.65)	(0.11)	(4.71)	(4.39)	(0.36)	(4.64)	(4.60)	(0.17)	(4.37)	(4.60)	(0.49)
Observations	5,794	5,637	11,431	387	440	827	1,837	1,855	3,692	157	140	297

Sample restricts to infants observed in sample at 6 months of age. Significance levels: $* < 0.1$; $** < 0.05$; $*** < 0.01$.

Figure B1: Test of Assumption 2



Figures show pre-trends in outcomes for control sectors, comparing tornado-affected and unaffected areas.

Table B2: Tests of Assumptions 2 and 3

	Birth outcomes					At 3 months		At 6 months	
	Weight (KG)	MUAC (cm)	Chest (cm)	Head (cm)	Height (cm)	Fever	MUAC (cm)	Fever	MUAC (cm)
Test of parallel trends in control areas (Assumption 2)									
Trend differential per 100 days (α_3)	0.02	0.05	0.05	0.06	0.04	0.07	-0.02	0.01	-0.16
	(0.02)	(0.05)	(0.12)	(0.09)	(0.14)	(0.07)	(0.07)	(0.12)	(0.11)
Test of parallel trends in the treatment effect (Assumption 3)									
Trend differential per 100 days (α_7)	0.01	0.08	0.12	0.10	-0.02	0.09	0.06	-0.01	0.24
	(0.03)	(0.07)	(0.15)	(0.13)	(0.19)	(0.09)	(0.10)	(0.16)	(0.17)
Dependent variable mean	2.44	9.32	30.46	32.47	46.43	0.85	12.25	0.83	13.08
Dependent variable SD	0.43	0.87	2.13	1.67	2.45	1.02	1.08	0.98	1.05
Observations	6,740	6,667	6,601	6,675	6,517	6,840	6,688	4,683	4,516

This table reports estimates of α_3 and α_6 from equation 3 to test the validity of Assumptions 2 and 3. We restrict to those who were born before the tornado for birth outcomes, and those born more than 90 (or 180) days before the tornado for 3 (or 6) month outcomes. Robust standard errors reported.
Significance levels: $* < 0.1$; $** < 0.05$; $*** < 0.01$.