Resilience to Early Life Shocks

Evidence From the Interaction of a Natural Experiment and a Randomized Control Trial*

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September 16, 2016

Abstract

Can health investments engender resilience to early life shocks? We address this question by leveraging the confluence of two independent sources of variation: exposure to a large-scale natural disaster (a tornado) and randomized access to vitamin A supplementation at birth. Tornado exposure *in utero* and in infancy increased the frequency of fevers and decreased birth size and physical growth. But infants who received vitamin A supplementation, which boosts immune system functioning, were protected from these effects. Tornado impacts and protective effects were larger for boys. Our results provide support for wide-scale supplementation policies in disaster-prone areas of low-income countries.

JEL: I12, J13, O15
Keywords: early childhood development, human capital formation, resilience, natural disasters, Bangladesh, vitamin A

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A child's environment *in utero* and in early life shapes her survival and wellbeing in profound ways (Almond and Currie, 2011; Currie, 2000; Heckman, 2007). Shocks to this environment are all too common and can create life-long disadvantage, especially in low-income contexts (Currie and Vogl, 2013).[1] To what extent is it possible to protect children against these impacts, and to remediate negative outcomes for children who have been harmed?

The existence of such protective or remediating effects may appear self-evident. Indeed, these effects implicitly form the basis of our understanding of the complex process of health and human capital formation in childhood.[2] But empirically demonstrating their existence is not straightforward (Almond and Mazumder, 2013). While it is plausible that exposure to many types of insults is effectively random, measures taken to prevent or buffer against negative impacts – and, analogously, measures to mitigate impacts once a shock has occurred – are likely not random at all. They are deliberate choices. The extent to which parents, communities, and governments invest in promoting a child's wellbeing is likely correlated with unobserved characteristics that also determine his outcomes. Thus, demonstrating heterogeneity in children's outcomes after a shock across different levels of investment does not constitute a valid estimate of the protective or remediating effect of that investment.

We address this issue by leveraging two independent sources of variation, combining an exogenous negative shock with randomized health investment in early life. On March 20, 2005, a tornado tore through several areas of northwest Bangladesh that were involved in a large, double-blind cluster-randomized controlled trial (RCT) of newborn vitamin A supplementation. The tornado killed 56 people and injured almost 4000, and generated substantial property damage in about 7 percent of the villages under study (Sugimoto et al., 2011). Both treatment and control villages were affected.

Using this overlap – along with detailed anthropometric measurement and survey-based outcomes for infants at 0 to 6 months – we test whether vitamin A supplementation mitigates the deleterious effects of experiencing a natural disaster in early life. Vitamin A promotes the functioning of neutrophils, macrophages, and natural killer cells – vital components of the body's immune system. It also helps prevent infections by maintaining epithelial integrity (Thurnham et

[1] Shocks that cannot be fully insured, such as aggregate crises or shocks in areas with incomplete insurance markets, are particularly harmful (Bharadwaj and Vogl, 2015).

[2] See, e.g., the discussion of "static substitutability" in Heckman and Mosso (2014) and Cunha et al. (2010).

al., 2000), and restores innate immunity after infection by promoting the normal regeneration of mucosal barriers (Stephensen, 2001). And in vitamin A-deficient contexts, supplementation at birth can reduce infant mortality (Haider and Bhutta, 2011; Klemm et al., 2008). We therefore hypothesized that supplementation, through its role in maintaining developing immunity and restoring it after infection, may affect infants' abilities to cope with assaults to the early life environment. Specifically, we ask: does supplementation at birth remediate the effects of *in utero* shocks, or protect against shocks after birth, or both? If yes, then which role of vitamin A is most salient in remediation and/or protection? Finally, what do the answers to these questions tell us about the overall effect of supplementation?

To estimate the effects of the tornado, we compare the health outcomes of cohorts exposed *in utero* and in early life (0-3 and 3-6 months)[3] to unexposed cohorts, across localities ("sectors") falling within and outside the tornado's path. Then, to identify the remediating and protective effects of vitamin A supplementation, we add a third difference, across treatment and control sectors of the RCT.

We show that exposure to the tornado, particularly in the second trimester of pregnancy and at 0-3 months, increased the frequency of severe fevers, and negatively impacted infants' nutritional status in the RCT control sectors. In particular, mid-upper arm circumference (MUAC), a reliable indicator of thinness and predictor of child mortality (Briend et al., 1987), declined dramatically, by more than 0.3 standard deviations. A standardized index of anthropometric outcomes (Kling et al., 2007) fell by roughly the same amount. But these differences all but vanish for exposed cohorts in vitamin A treatment sectors, indicating both a remediating and a protective role for early vitamin A supplementation. Consistent with much previous evidence on the "fragile male" hypothesis (Kraemer, 2000), we estimate large tornado impacts – as well as large remediating and protective effects of vitamin A – for boys, and small impacts for girls. Impacts on fevers do not demonstrate the same pattern of remediation and protection. We argue in the paper that, taken together, these results suggest that the tornado's impact on infants was primarily through changes in the disease environment, which spurred an increased probability of infections, and that vitamin A's role in remediation and protection was via the regeneration of immunity after infection.

[3]The 0-3 month cohort is defined as those infants who were born 1 - 90 days before the tornado and the 3-6 month cohort are those born 91 to 180 days before the tornado.

Our study adds to the understanding of human capital formation at very early stages (Almond and Currie, 2011; Heckman, 2006, 2007). Learning how to protect children from the negative consequences of early life shocks is an essential undertaking for academics and policymakers alike (Currie and Vogl, 2013). The question of resilience relates to the shape of the human capital production function: do early investments (or shocks) complement or substitute for each other (Almond and Mazumder, 2013; Cunha et al., 2010)? In line with what we find, several recent studies from diverse contexts suggest that substitution (in these cases, remediation of early life disadvantage) prevails (Adhvaryu et al., 2015; Bitler et al., 2014; Rossin-Slater and Wüst, 2015).[4]

This paper also contributes to the literature demonstrating that early intervention – for example, to prevent disease (Bhalotra and Venkataramani, 2015; Bleakley, 2007; Fink et al., 2015); increase access to adequate nutrition (Adhvaryu et al., 2016b; Almond et al., 2011; Hoynes et al., 2012); expand health care coverage (Almond et al., 2006; Miller and Wherry, 2014); and raise household income (Adhvaryu et al., 2016a; Aizer et al., 2014; Dahl and Lochner, 2012; Hoynes and Patel, 2015) – has profound impacts on wellbeing that can last well into adulthood. Our results reveal one potential mechanism for these effects: investments in early life may protect against or help to remediate the negative impacts of large shocks.

Finally, our findings are also 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 against the risk of mortality from natural disasters, which are increasingly common as a result of climate change (Field, 2012). Our results provide support for wide-scale infant supplementation with vitamin A in disaster-prone areas of low-income countries.

The remainder of the paper is organized as follows. Section 1 provides contextual details regarding the vitamin A supplementation RCT and the tornado event. Section 2 describes our data, and section 3 provides details on our empirical strategy. Section 4 describes the results, and section 5 reports checks on internal validity. Section 6 concludes.

[4] A recent paper by Malamud et al. (2016) estimates no significant interaction of two policies affecting the welfare of Romanian children.

1 Context

1.1 Vitamin A Supplementation and Infant Health in Bangladesh

Rates of infant and child mortality in Bangladesh have declined dramatically over the last 3 decades. Between 1980 and 2015 infant mortality fell from 137 to 31 per thousand and child mortality from 198 to 38 per thousand (Wang et al., 2014). Still, 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, and leave infants vulnerable to a variety of potentially mortal "insults." In a recent 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. 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 (West Jr, 1996). Supplementation at (or shortly after) birth has consistently been shown to reduce infant mortality by 10% or more in South Asia (Humphrey et al., 1996; Mazumder et al., 2015; Rahmathullah et al., 2003) although similar effects have not been observed in Sub-Saharan Africa (Bhutta et al., 2013), for reasons that remain unknown.

1.2 The RCT

The randomized field experiment 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.[5] 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. The study site is typical of rural Bangladesh, lying at approximately the 35th percentile of the distribution of economic and quality of life indicators among rural areas

[5] 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; Identifiers: NCT00198822 (maternal trial) and NCT00128557 (infant trial).

in Bangladesh. See Figure 1 for a representation of the study's location within South Asia and Bangladesh. We direct the reader to Labrique et al. (2011) for a more detailed discussion of the study area and how it relates to the context of rural Bangladesh.

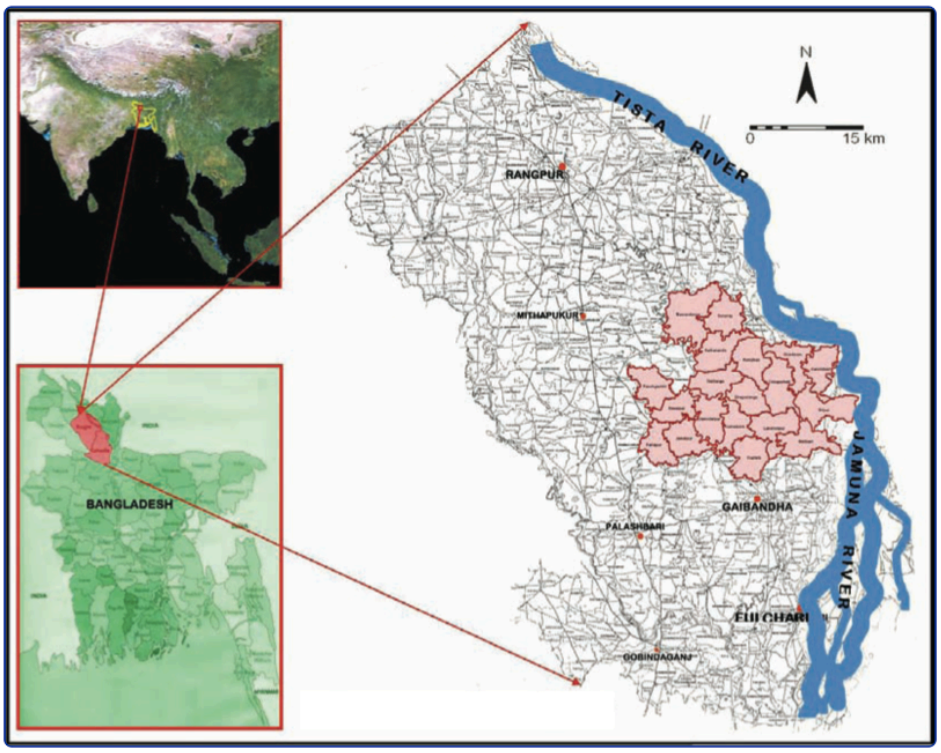


Figure 1: Location of the study area. The figure was produced by the JiVitA GIS Unit.

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 (totaling 102,769) and newlywed women (during the study, totaling 27,711), ages 13-45, were surveilled for pregnancy. In total, 60,294 pregnancies were identified and, if consent was given (>99% of cases), the pregnant woman was enrolled in the maternal supplementation study. The infant trial was nested within the maternal trial and

was conducted between January 2004 and December 2006. A total of 15,937 infants received supplementation or placebo directly at birth or shortly thereafter and were followed until 6 months after birth.

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. Live-born infants in each sector were randomized to receive either 50,000 IU[6] of vitamin A or a placebo once as oral oil drops from a capsule shortly after birth. 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).

Our study focuses analysis solely on the newborn supplementation trial. The primary reason is that maternal supplementation with vitamin A or β-carotene in this context had no impact on maternal, fetal, or infant mortality (West et al., 2011), nor on gestational length or birth anthropometry (Christian et al., 2013). Given the lack of effectiveness of maternal supplementation, we do not find it relevant to focus on this type of investment as a potential contributor to resilience to shocks.

The at-birth supplement, in contrast, did have significant impacts on mortality: mortality at 6 months was 15 percent lower for infants who were supplemented with vitamin A at birth compared to those supplemented with placebo (Klemm et al., 2008). These impacts suggest exploring the hypothesis that at-birth supplementation confers resilience to shocks experienced *in utero* and during infancy.

1.3 The Tornado

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 Figure 2). 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

[6]International Units. 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).

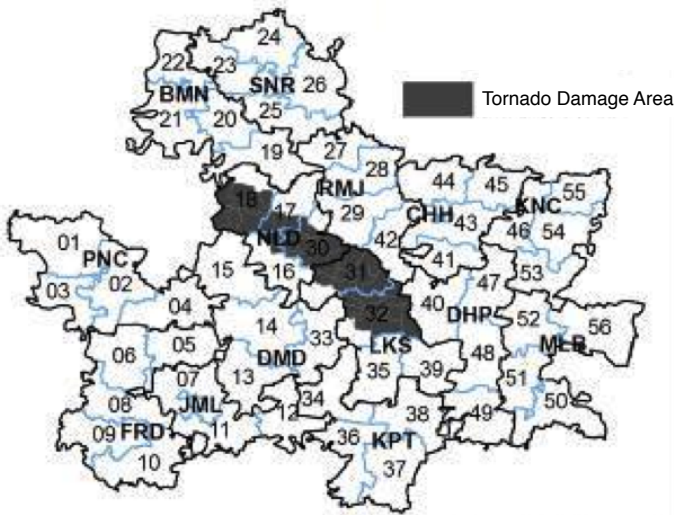


Figure 2: Area damaged by the tornado. The figure was produced by the JiVitA GIS Unit.

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 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 73.5% while those outside of this area were dosed at the rate of 72.5%. Birth anthropometry for this same population was obtain within 7 days in the tornado area at the rate of 84.5% and outside this area at the rate of 83.9%.

For balance to hold, it must be the case that the tornado hit vitamin A and placebo sectors equally hard. This can be checked in the data. In fact, the average number of houses destroyed in the tornado hit vitamin A sectors was 33.7% compared to 47.6% in the tornado hit control sections. In Section 5.4 we show that the results are consistent in subsamples that have a more equal tornado exposure and based on this conclude that this imbalance is not driving our vitamin A interaction results.

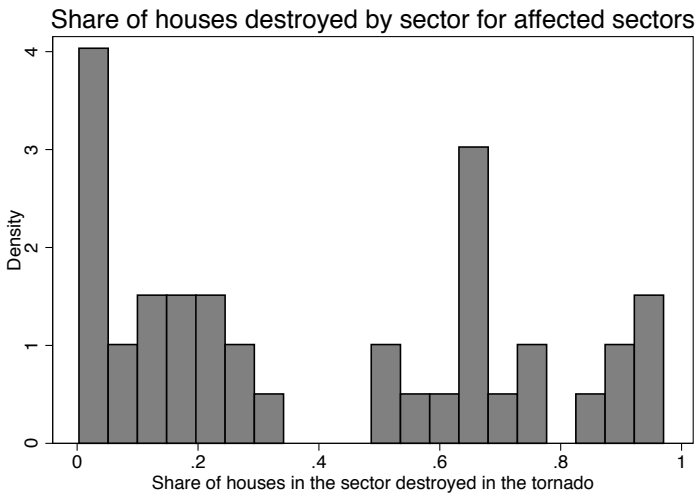


Figure 3: Histogram of the share of houses destroyed by sector (only for the 41 sectors with $> 0\%$ damage).

2 Data

2.1 Sample

We include 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 the same way as for other observations). After these adjustments, the final sample is 19,033 live births.

2.2 Summary Statistics

Table 1 reports means and standard deviations of important outcomes and control variables for the sample of infants that were at least 9 months of age at the time of the tornado. We code as missing birth measures taken after 7 days and 3 and 6 month measures taken more than 8 weeks after the target date (in our regressions we also control for the date of measurement). We report means for the whole sample, as well as within and outside of tornado-affected sectors, and across treatment and control sectors within the tornado area. We also report differences in means across these subsamples. Asterisks denote statistically significant differences (standard errors were calculated via ordinary least squares regressions).

Infants in this area of Bangladesh are small relative to reference populations. The mean weight is 2.5 kg, exactly at the threshold for classification as low birth weight. Average length at birth in cm is approximately 46.7, a full 3 cm less than the reference US population. Head circumference is 32.7 cm at birth, which is 3 cm less than the same measurement for the reference US population. This difference (with respect to the reference population) shrinks slightly by 6 months: head circumference at 6 months is 40.89 cm as compared to 43.5 cm for reference infants.[7]

2.2.1 Comparisons across affected and unaffected areas and across study arms within tornado sectors

Means of health outcomes at birth and at 3 and 6 months are balanced across the tornado and non-tornado areas for pre-tornado cohorts. There is some evidence that infants in tornado-affected sectors were slightly healthier, particularly by 6 months: 9 out of the 11 anthropometric measurements recorded are larger in the tornado area; three of these differences–MUAC, CC, and AI, all at 6 months–are statistically significant, though the differences are small in magnitude.

Next, we compare means across treatment sectors (infants who received vitamin A supplementation at birth) and control (placebo) sectors within the tornado-affected area. Reassuringly, most birth outcomes (weight and anthropometry) and maternal characteristics are balanced across the treatment arms within the tornado area. Finally, anthropometric measures at 3 and 6 months are not significantly different across the two groups.

3 Empirical Strategy

3.1 Sources of variation

We leverage three sources of variation to identify the protective and remediating effects of vitamin A. The first two identify the impacts of the tornado, and the third identifies the vitamin A effect: 1) spatial variation in tornado exposure; 2) temporal overlap between the tornado event and key early life periods; and 3) the randomized allocation of vitamin A to newborns.

With regard to spatial variation, we compare infants born in sectors that were in the tornado's

[7] Data for reference populations are from the Centers for Disease Control and Prevention Growth Charts for the United States (Kuczmarski et al., 2000).

path with those born in sectors outside this path. Our baseline definition of spatial exposure classifies a particular sector as exposed if there was any tornado damage – that is, at least 1 home was destroyed – in the sector.[8] Under this definition, 41 sectors, or 7 percent of all sectors involved in the RCT, were exposed. We face the following tradeoff in defining tornado exposure this way. On one hand, since several sectors experienced "minor" damage (less than 20 percent of homes were destroyed in 17 sectors), we may be misclassifying these sectors as "exposed." On the other hand, a small percentage of sectors were affected, creating a relatively small number of exposed infants. The more restrictive the definition of exposure is, the smaller the number of exposed infants becomes. We chose to keep a wide definition of exposure to expand the group of exposed infants as much as possible. Though not reported here, we have estimated the models varying the definition of exposure and find that our results are qualitatively unchanged.

Second, we construct dummies for two main time periods of early exposure: the prenatal period (i.e., the infant was *in utero* during the tornado event) and early life (i.e., the infant was either 0-3 months or 3-6 months during the tornado). 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.

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 dosed with 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.

3.2 Empirical Specification

We estimate a triple difference across the three dimensions described above to identify the protective effect of vitamin A. We assess the impact of the tornado by comparing outcomes for infants across sectors affected by the tornado v. unaffected sectors and for those whose prenatal and early life

[8] We chose to use a binary classification rather than a continuous variable to avoid the possibility that housing stock (e.g., the type of roofing used) may determine the intensity of damage and thus of exposure, as well.

periods coincided with the tornado timing v. those for whom these periods did not. We then take a third difference across treatment v. control sectors, to estimate the protective or remediating effect of vitamin A supplementation at birth.

We estimate the following specification via ordinary least squares (OLS):

$$\begin{aligned} O_{ijk} \quad = \quad & \alpha + \sum_{k=1}^{K} \left(\beta_1^k C_i^k + \beta_2^k C_i^k \cdot T_{ij} + \beta_3^k C_i^k \cdot VitA_{ij} + \beta_4^k C_i^k \cdot T_{ij} \cdot VitA_{ij} \right) \\ & + a_i + \lambda_j + \epsilon_{ijk} \end{aligned} \tag{1}$$

Here, i denotes infant, j denotes sector and k denotes cohort (e.g., being in the 2nd trimester at the time of the tornado). O_{ijk} is a health outcome measure. T_{ij} is a dummy for tornado-exposed sector. $VitA_{ij}$ is an indicator for treatment status of sector j in the infant supplementation trial. C_i^k is a dummy that is 1 if the infant was in cohort k during the tornado event. a_i is the age of anthropometric measurement (to control for late measurement). The λ_i's are sector fixed effects (these absorb T_{ij}, $VitA_{ij}$ and $T_{ij} \cdot VitA_{ij}$, which would otherwise be included in the regression) and ϵ_{ijk} is a mean-zero error term.

For birth outcomes, we exclude all vitamin A interactions (since supplementation at birth does not impact birth anthropometry) and as a result the specification reduces to the following specification, capturing the impact of the tornado on birth outcomes:

$$\begin{aligned} O_{ijk} \quad = \quad & \alpha + \sum_{k=1}^{K} \left(\beta_1^k C_i^k + \beta_2^k C_i^k \cdot T_{ij} \right) \\ & + a_i + \lambda_j + \epsilon_{ijk} \end{aligned} \tag{2}$$

Standard errors are clustered at the level of variation of the main explanatory variables, that is, within categories defined by the interaction of T_j, $VitA_j$, and year x month of birth.

4 Results

All of our main results are reported in figures, with supporting tables (with the same results) provided for reference. All figures report point estimates and standard error bars corresponding to

95% confidence intervals. All standard errors have clustering corrections applied.

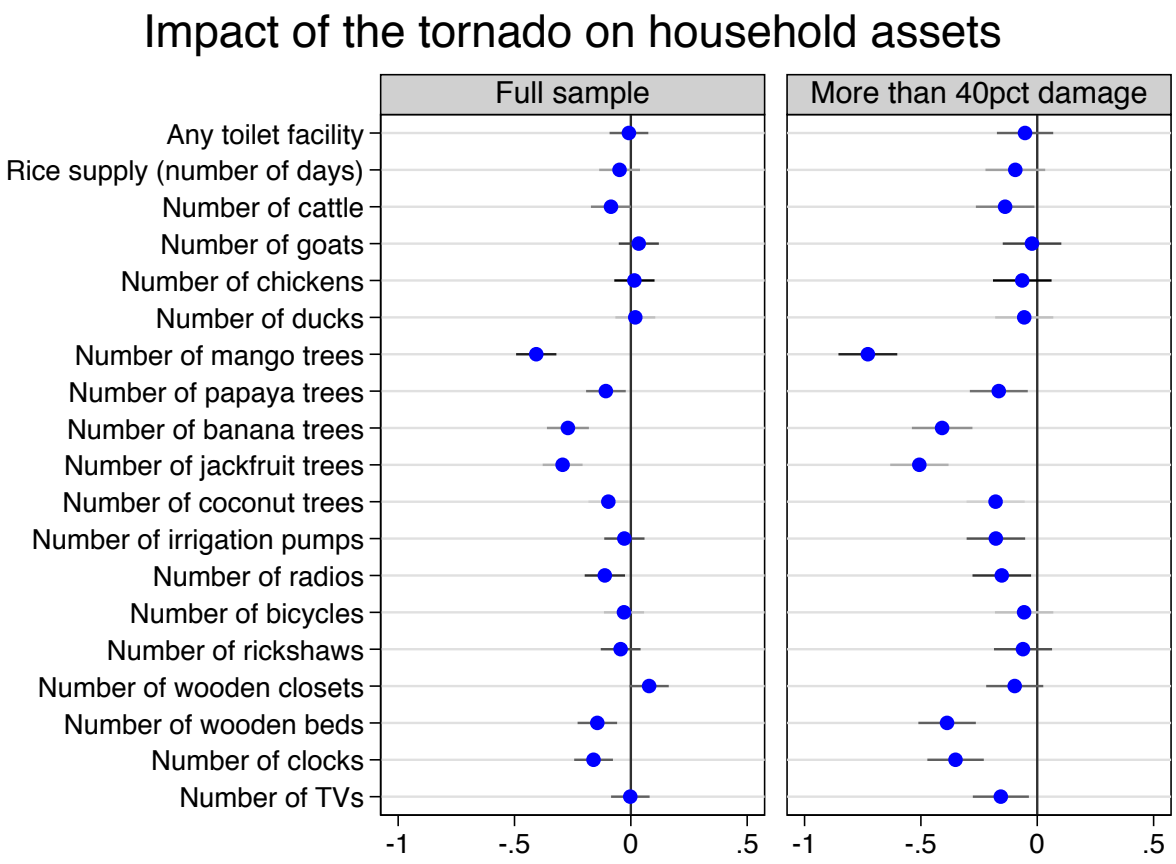


Figure 4: This figure shows the impact of the tornado on households' assets using a double difference strategy in which we compare asset ownership of households within and outside the tornado area, and those surveyed about assets before or after the tornado. Each row presents results for one type of asset. Standard errors are clustered at the sector level.

4.1 Impacts of the Tornado on Household Assets

The tornado likely affected health in many ways, through maternal stress, changes in the disease environment, maternal malnutrition, and decreased access to health care services. We cannot separate these mechanisms. Instead, we show some results on physical assets that were damaged below, and, later in the paper, argue that the pattern of our results is quite consistent with the primary mechanism of impact being shifts in the disease environment.

Figure 4 show the impacts of the tornado on household assets. The left panel depicts impacts using the full sample of households whereas the right panel excludes tornado affected sectors in

which less than 40% of houses were destroyed. Each row depicts a difference in differences estimate of this impact, comparing household assets inside and outside of tornado areas for households who happened to be surveyed before and after the date of the tornado. We document significant impacts on agricultural property, particularly large reductions in the numbers of mango, papaya, banana, jackfruit, and coconut trees. Several physical assets were also affected, including radios, beds, and clocks.

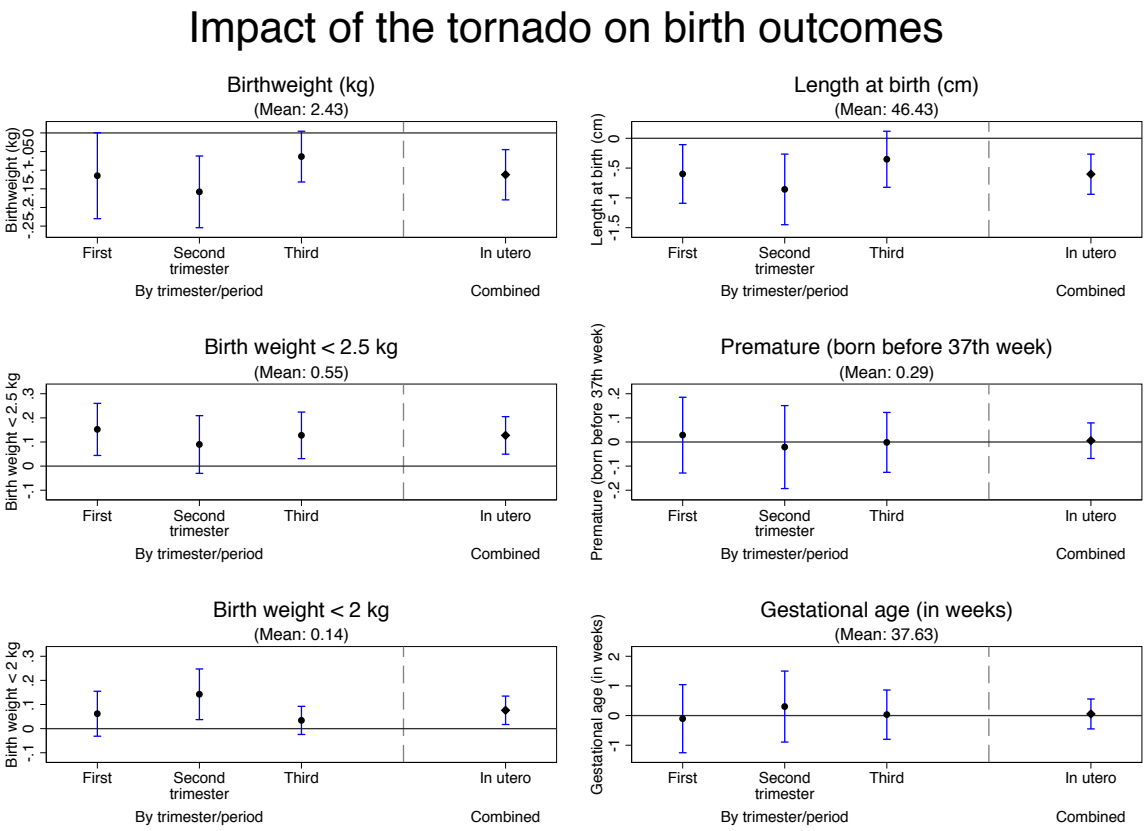


Figure 5: This figure shows the impact of the tornado on birth outcomes using the double-difference strategy described by Equation 2. Each panel presents results for one outcome variable. In each panel, we show point estimates and confidence intervals broken up by trimester of exposure (to the left of the dashed line) as well as results for combined *in utero* exposure (to the right of the dashed line). These two parts of the panel represent two different regressions.

4.2 Impacts of the Tornado on Birth Outcomes

Figure 5 reports impacts of the tornado on birth outcomes. Each panel in the figure reports one outcome. The first three point estimates correspond to impacts by first, second, and third trimester

exposure. The fourth point estimate, shown after the dashed vertical line in each panel, corresponds to the overall impact of *in utero* exposure (an aggregate of the impacts by trimester).

The top left panel of Figure 5 shows impacts on birth weight, measured in kilograms (kg), and the middle left panel shows impacts on a dummy for low birth weight (an indicator for birth weight less than 2.5 kg). Tornado exposure has a large and statistically significant impact on birth weight, with most pronounced impacts for first and second trimester exposure. Table 3 reports the corresponding coefficients. Infants exposed *in utero* were more than 10 percentage points more likely to have low birth weight from a baseline of 55 percent among unexposed infants. We observed this effect throughout the lower end of the birthweight distribution as can be seen in the bottom left panel, which reports that infants exposed *in utero* were 7 percentage points more likely to be born less than 2kg (from a baseline of 14%).

The top right panel in this figure reports impacts on length at birth, another summary measure of newborn health. Again, we find significant negative impacts of the tornado, especially in the first and second trimesters: exposed newborns were approximately 0.6 cm shorter.

The middle right and bottom right panels show impacts on prematurity (born before 37 weeks) and gestational age in weeks, respectively. These panels show that the tornado did not have any discernible impacts on length of gestation, and our estimates of no effects have a fairly high degree of precision. In sum, the tornado significantly negatively affected size at birth, but did not change gestation length.

4.3 Outcomes 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. We estimate these separately by vitamin A treatment status, allowing us to identify the protective effects of vitamin A supplementation at birth. We use regression models of the form shown in equation 1. We show results for mid-upper arm circumference (MUAC), a reliable early predictor of infant mortality, and for the number of severe fever episodes as reported by the infant's mother. Fever in particular is an important potential mediator of impacts on anthropometry because of the crucial role vitamin A plays in the body.

Results are shown in Figure 6. For 3-month outcomes, we report impacts of exposure in each trimester of pregnancy as well as in the first three months of life. For 6-month outcomes, we add

the impact of exposure in months 3-6 of life. For each time period, two coefficients are reported, one for the impact of tornado exposure in the vitamin A group, and one for the impact in the placebo group. Each coefficient has two standard error bars, one for the 95% confidence interval (whiskers), and one for the 83% confidence interval (solid lines). The outer confidence interval (whiskers) can be used to visually assess the hypothesis of a non-zero point estimate, while the inner confidence interval (solid lines) can be used to assess the hypothesis of a difference between the two point estimates across vitamin A v. placebo. If the inner confidence intervals do not overlap, then the two point estimates are significantly different at the 5% level.

We begin by looking at results on MUAC, shown in the top two panels of Figure 6. We find similar results for MUAC measured at 3 and 6 months. Namely, second trimester exposure and exposure at 0-3 months significantly decreases MUAC in the control group. Impacts are quite large – between 0.4 and 0.5 cm, which also translates to between 0.4 and 0.5 SD.[9] But in the vitamin A treatment group, there is essentially no negative tornado impact on MUAC. Moreover, second trimester and 0-3 month impacts are significantly different across treatment and control sectors.

Next, we discuss results on severe fever episodes. Here, the conclusion is slightly different. For number of severe fevers reported between 0 and 3 months, *in utero* tornado exposure in every trimester increases fevers in the control group (significant at the 10% level), and also increases fevers for the treatment group by roughly the same amount in the first and second trimesters. But at 0-3 months, while exposure in the control group generates a sharp increase in fever episodes, infants in the treatment group are protected by vitamin A (the difference in impact across the two groups for 0-3 month exposure is statistically significant). For fevers measured at 3-6 months, second trimester and early life (0-3 and 3-6 month) exposures matter for the control group, and early life exposure matters for the treatment group, as well. In this case, there are no significant differences in impact across vitamin A and placebo groups. The corresponding point estimates and errors are reported in Table 4.

Taken together, we interpret these results in the following way. Tornado exposure, both during gestation and in the first few months of life, has a clear negative impact on the infant's immune system, which increases the number of infections (and thus severe fevers) experienced early in life.

[9]The MUAC measures reported in Figure 6 are in measured in centimeters. However, as can be seen in Table 1, the standard deviation of these measures are 1.04-1.06 cm so an approximate impact in standard deviations can be read from the figure.

This is consistent with a shift in the disease environment as a result of the tornado. This pattern holds for both the treatment and control groups for nearly every period of tornado exposure, suggesting that vitamin A has a limited role in mitigating the deleterious effects of shocks on immune system robustness. The only counterexample to this pattern is for exposure at 0-3 months. Here, for fevers at 0-3 months, we see a fairly substantial *protective* effect of vitamin A. This may be due to the fact that the shock occurred after supplementation was administered (at birth), or because of the fact that serum retinol levels remain elevated for several weeks after supplementation, delivering a direct protective effect.

The results on MUAC – showing large negative effects on anthropometry in early life for exposure in the second trimester and at 0-3 months in the control group but not in the treatment group – are interesting in their divergence from the pattern in the fever results. These results suggest substantial mitigating *and* protective effects of vitamin A for anthropometry. We suggest that these results relate to the role of vitamin A in quickening the regeneration of immune system function (e.g., rebuilding of mucosal barriers) *after* infection, creating a smaller likelihood that infection translates into differences in anthropometry.

This nuance is important. It suggests that vitamin A works through a particular immune system-based mechanism – the regeneration of immune function after infection – to cause health differences for children who have experienced negative shocks. It also suggests that vitamin A exposure would not necessarily be useful against shocks that do not affect the immune system in a meaningful way.

As one example, take the fluctuations in MUAC seen across birth months, plotted here in Figure 7. This relationship, between month of birth and health in childhood (as well as later in life), has been documented in many contexts (see, e.g., Buckles and Hungerman (2013)). In poor agrarian contexts, it often reflects the fact that the lean season, when resources from the previous harvest have run thin, is one of widespread food insecurity (Basu and Wong, 2015). Babies whose gestation and early life periods overlap with the lean season thus often experience persistently negative health outcomes.

This can be seen clearly in Figure 7. For both male and female infants, MUAC at 6 months is substantially lower for babies born in the October, November, and December. The point of showing these patterns is to note the striking lack of differences across vitamin A treatment and placebo

groups. That is, for large nutritional shocks, vitamin A appears to have no role in mitigation or protection of negative effects on infant health. This is consistent with the mechanism suggested above, which emphasizes the role of vitamin A in immune system function.[10]

4.4 Results by Gender

Next, we estimate heterogeneous effects across gender. The manyfold innate physiological differences across male and female infants, particularly as relate to vulnerability to shocks, suggest that both the extent of the negative impacts of tornado exposure, as well as the resilience generated by vitamin A supplementation, might vary across gender. We test this hypothesis by estimating impacts the same way as above, separately for boy and girl infants.

The results, presented in Figures 8 and 9 reveal meaningful heterogeneity. The tornado had large deleterious impacts on MUAC and fever episodes for male infants in the control group, especially in the second trimester and in early life. But for male infants supplemented with vitamin A, those negative impacts all but disappear, particularly for MUAC at 3 and 6 months. In contrast, there are few significant impacts of tornado exposure on female infants (in fact, MUAC shows no significant effects), and effects are essentially 0 across both treatment and control groups for girls.[11] The corresponding point estimates and errors are reported in Tables 5 and 6.

The substantial heterogeneity in tornado impacts as well as vitamin A interactions seen in boys v. girls may represent a manifestation of the "fragile male," the finding consistent across a wide variety of studies that boys are much more innately susceptible to insults *in utero* and in early life than girls (Kraemer, 2000).

5 Checks

In this section, we check for potential concerns related to internal validity.

[10] Of course, nutrition and immune function are intimately linked, but it is likely true that direct shocks to the disease environment, such as that conferred by tornado exposure, would have a larger propensity to damage the immune system than more insidious immune system effects generated by malnutrition during gestation or in early life.

[11] We do find that for fever at 3 months, girls in the treatment group actually reported more fevers if exposed in the first or second trimester. This might be a spurious result due to the small numbers of girls per cell in the regression, or it maybe real, and related to a finding from previous RCTs showing that girls sometimes react negatively to early supplementation with vitamin A (Jørgensen et al., 2013).

5.1 Attrition

We begin with a discussion of 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 loss to follow up (i.e., the household could not be located at 3 or 6 months following the infant's birth and thus anthropometry and survey responses are not recorded) or due to death of the infant. If either of these types of attrition is affected by tornado exposure or (after birth) by vitamin A interactions with exposure, it is possible that this differential sample selection could be driving our results. We thus estimate the relationship between both of these types of attrition and exposure (and vitamin A supplementation) to test for sample selection bias in our estimates.

In Table 7, we look at the first type of attrition by studying miscarriages, abortions, and live births. (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). The main result in Table 7 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) live births were also not significantly affected (there is a marginally significant effect on live birth in the second and third trimesters but this may be an artefact of the small sample given that almost all pregnancies that survive into the second trimester result in a live birth; in this case only 3 and 1 pregnancy exposed in the second and third trimester, respectively, did not result in a live birth, whereas, based on rates outside the tornado area, we would have expected 7.6 and 4.6, respectively).[12]

Next, we look at the second type of attrition, namely attrition at 3 or 6 months for live births. Here we use an identical set of right-hand side variables as in our baseline specification, but use as outcomes dummies for whether measures were missing, or "missing or late" (where late refers to measurement 8 weeks or more after the target date), for 3 and 6 month measurements. The results of this analysis are reported in Table 8. We find overall that attrition of live births is not

[12] We have to cluster standard errors in a slightly different way in this analysis than any of the other analysis in the paper, because the date of birth is obviously not defined for pregnancies that did not result in a live birth. Please see the table notes for clarification on clustering in this situation.

significantly different across exposed and unexposed infants, nor is it different by vitamin A group interactions with tornado exposure.

Taken on the whole, the evidence on attrition strongly suggests that our estimates are not affected by sample selection bias.

5.2 Dosing

According to the trial protocol, infants were to be dosed within hours of birth with either treatment (vitamin A) or placebo. The trial was double blind, so the implementation teams did not know whether they were dosing infants with treatment or placebo. 41 percent of infants were dosed within 6 hours of birth. 56 percent were dosed within 12 hours, and 67 percent by 24 hours. The dose timing distribution has a long right tail: 24 percent of infants were dosed more than 7 days after birth.

Table 9 reports results for dummies indicating dosing occurred within 1 day and within 7 days. Overall, the results in this table show that the distribution of dosing timing was not significantly different across infants exposed and unexposed to the tornado, and across vitamin A and placebo interactions with tornado exposure. 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: there is no reason to suspect differential delays in dosing across treatment status given the fact that trial administrators did not know which sectors were assigned to receive vitamin A and which were assigned to receive placebo supplementation.

5.3 Restricting the Control Group to Pre-Tornado Cohorts

In our main analysis, infants conceived after the tornado are considered part of the (temporal) control group. It is possible that these infants were affected by the aftermath of the tornado; for example, sanitation and health infrastructure likely took time to rebuild in affected areas, so infants born in some window well after the tornado could still have been exposed to its negative impacts.

To account for this possibility, we include additional interaction terms that remove the cohort conceived after the tornado from the control group. Thus all cohorts are now compared only to the

cohort born more than 3 months before the tornado. The results are reported in Figure 10. We find that the main results (on MUAC and fever) are effectively unchanged. The inclusion of the cohort conceived after the tornado largely serves to make the estimates of tornado impact in early life and the protective effect of vitamin A stronger.

5.4 Changing the Definition of Spatial Tornado Exposure

One possible concern for our estimates, highlighted previously, is that 6 of the most damaged sectors happened to all be control sectors. As a result, among the sectors affected by the tornado, 47.6% of houses in control sectors were destroyed compared to 33.5% of houses in vitamin A sectors. To examine whether this drives the results we performed a robustness exercise by estimating the baseline model on different subsets of the tornado sectors. For each $k = 1, ..., 10$ we re-estimated the baseline model 1000 times excluding k randomly selected tornado affected sectors. Then we collected the double and triple difference estimates and plot them up on the y-axes of figure 11. The x-axis of each figure is R, the ratio of the percent damage in treatment sectors over the percent damage in control sectors within the sample used for each estimation. On each figure we draw a line for the average value of the double or triple difference estimate over values of R using a locally weighted regression smoother. Our estimates are remarkably consistent over the range of values of R and in particular appear to be essentially unchanged in the neighborhood of $R = 1$, where treatment and control sectors are damaged equally. It thus appears unlikely that our findings are driven by control sectors being especially hard-hit.

In our main specifications we use all unaffected sectors as our comparison sectors for tornado impact. It may be more appropriate to compare the tornado affected sectors to the sectors that are geographically close but unaffected. To examine robustness of the findings to this restriction we ordered the sectors by their distance from the tornado and included as comparison sectors only the 20% that were closest to the tornado. Our results are very similar to the main specifications.

5.5 Changing the Definition of Temporal Tornado Exposure

Another possible concern, first highlighted in a recent study on hurricanes and birth outcomes (Currie and Rossin-Slater, 2013), is that there may be a mechanical correlation between the length of gestation and the likelihood of *in utero* exposure, given that infants with relatively long periods

of gestation had more time *in utero* to potentially be exposed.

We deal with this eventuality by using data on the best-guess date of conception, which is determined through a combination of a urine test for pregnancy and the woman's self-reported date of last menstrual period. We construct uniform gestation lengths for the entire sample by predicting a date of birth for each infant in the following way: take the infant's best-guess date of conception and add the median length of gestation in our sample. This ensures that gestation length is effectively held as fixed given date of conception. While this procedure will give us estimates free from the potential mechanical correlation described above, it will, of course, introduce measurement error in temporal (cohort-based) tornado exposure, given that the predicted and actual dates of birth are different.

Results of this estimation for birth outcomes (similar to the outcomes reported Currie and Rossin-Slater (2013)) are reported in Figure 13. We find that the pattern of results is qualitatively unchanged. Coefficients become slightly smaller under this revised definition of gestation, suggesting that measurement error is indeed at play.

6 Conclusion

Infants are highly vulnerable to a variety of insults *in utero* and in early life. Quantifying the deleterious effects of disease, environmental factors, income and nutritional scarcity, and natural disasters on infant health and survival is the focus of a rapidly expanding set of studies in economics. We know from this work that impacts, particularly in low-income contexts, can be large and long-lasting. But we have little rigorous empirical evidence on whether intervening in early life can change outcomes for children exposed to trauma.

In this study, we leverage the co-occurrence of a natural disaster and an RCT to estimate the negative impacts of tornado exposure on outcomes at birth and in early infancy, as well as the remediating and protective effects of vitamin A supplementation at birth. We find significant impacts of the tornado on birth outcomes as well as anthropometry at 3 and 6 months with quite large estimated impact sizes. But infants who received a one-time dose of vitamin A at birth did not experience the same drops in anthropometric measures.

Our results support a novel role for vitamin A, given at birth as a single large dose, in strength-

ening the physiological resilience of infants born to mothers who experienced a devastating tornado, or experienced themselves the event and stresses that followed. These effects have been observed in a population where a randomized trial reported an overall reduction of 15% in all-cause infant mortality following newborn vitamin A versus placebo receipt, consistent with multiple other trials showing similar effects in the South Asian region. In one (Tielsch et al., 2007), the design allowed investigators to discern significant reductions in infant fatality due to diarrhea and, important for our results here, severe fever. Results on the incidence of fever episodes in infancy reinforce the findings on anthropometry and shed some light on a potential mechanism through which the remediating and protective role of vitamin A may operate. We were not able to assess precisely through what mechanism the observed effects may have occurred, but they may be due to stronger resistance to infection, or possibly other sources of stress and inflammation that may accompany severe trauma.

This study demonstrates, to our knowledge for the first time, that a health intervention at birth can strengthen resilience to trauma in early life. 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 war (Akresh et al., 2012) all leave lasting scars on health, human capital, and welfare that persist well into adulthood. 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 the incentives for investing in children.

Our study takes a step toward filling this gap. Our results demonstrate strong effects of one-time vitamin A supplementation at birth. We interpret this as evidence that, at least in very early life, endowments (as proxied for by tornado exposure) and investments (vitamin A) are substitutes. Whether this remains true when outcomes are measured in later childhood and adulthood is an open question. Although our findings hold up to a variety of checks of internal validity, their strength is somewhat limited by the relatively small share of infants in the study affected by the tornado. Our results hopefully offer a valuable start and suggest that more research on the role of

micronutrient deficiencies in infants' resilience to shocks is likely to be very valuable.

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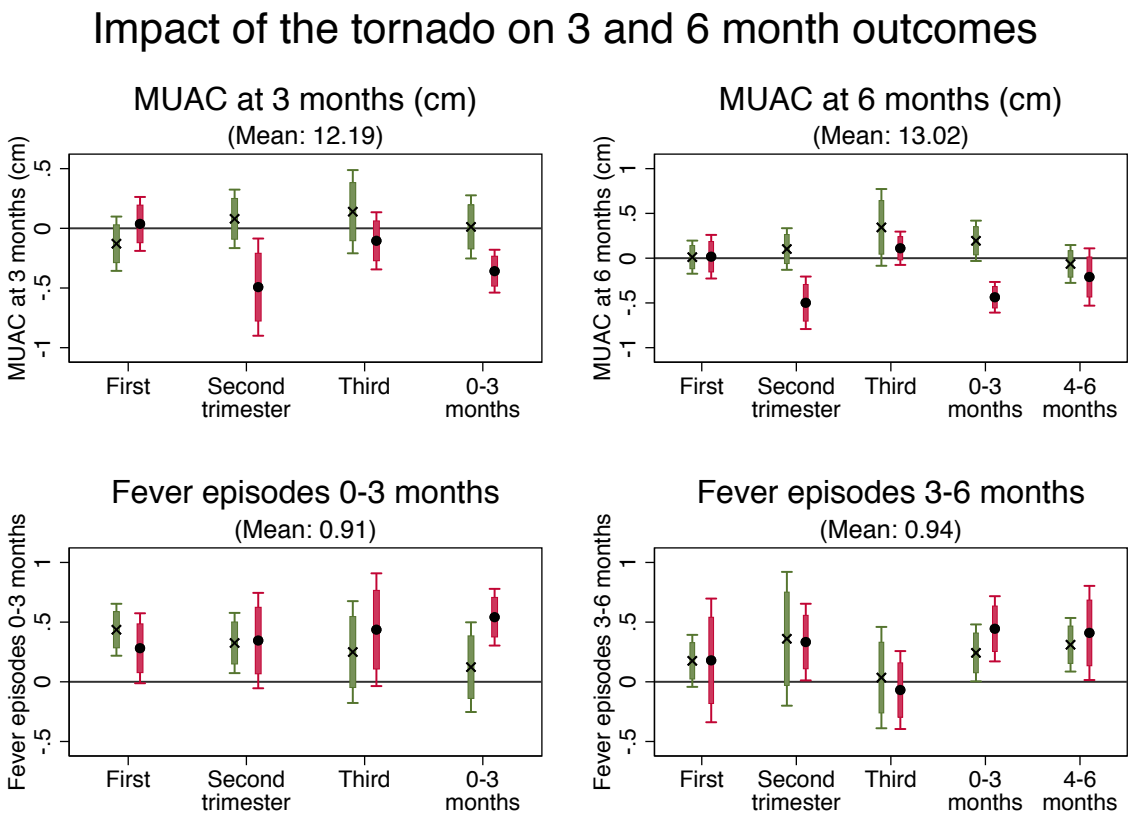


Figure 6: This figure shows the impact of the tornado on 3 and 6 month outcomes using the triple-difference strategy described by Equation 1. The newborn vitamin A group is depicted in green with a point estimate marked by an 'X' and the control group in red with a point estimate marked by a dot. Each panel presents results for one outcome variable. We show point estimates and confidence intervals for each cohort that is affected (from first trimester through 3 or 6 months after birth). In each case we present both an inner 83% (solid lines) and an outer 95% (whiskers) confidence interval. The outer confidence interval can be used to visually assess the hypothesis of a non-zero point estimate while the inner confidence interval can be used to assess the hypothesis of a difference between the two point estimates – if the inner confidence intervals do not overlap then the two point estimates are significantly different at the 5% level.

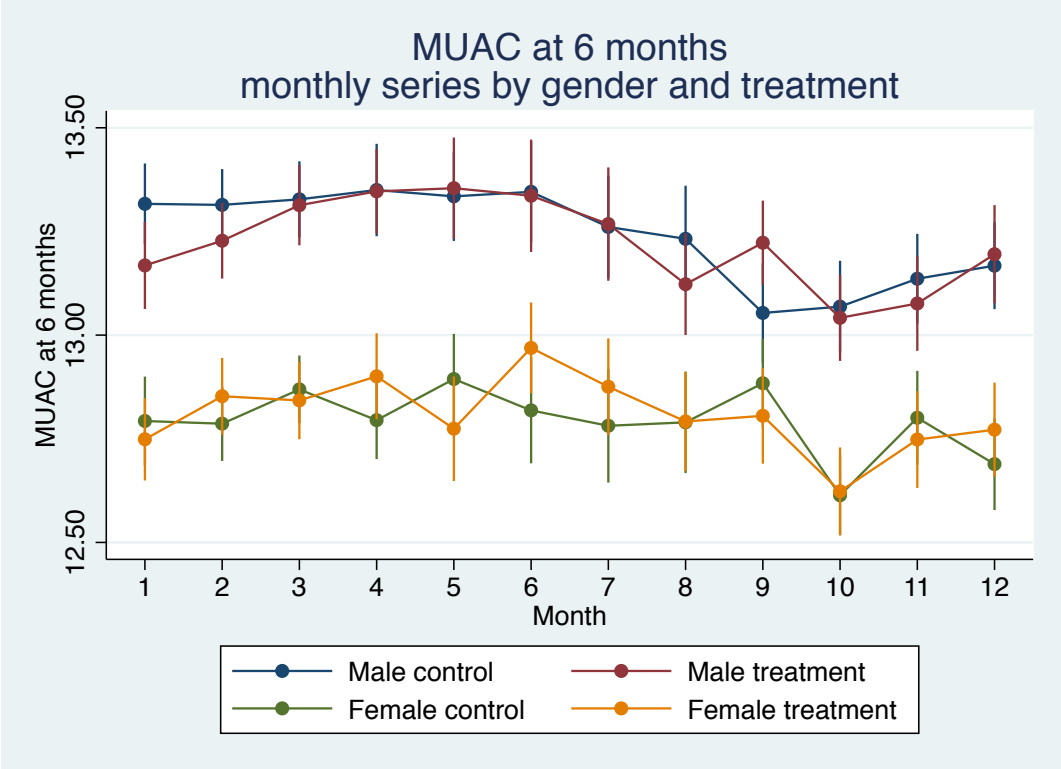


Figure 7: Average 6 month MUAC by birth month. The figure shows four series, one for each combination of gender and treatment. Standard errors are clustered at the sector level.

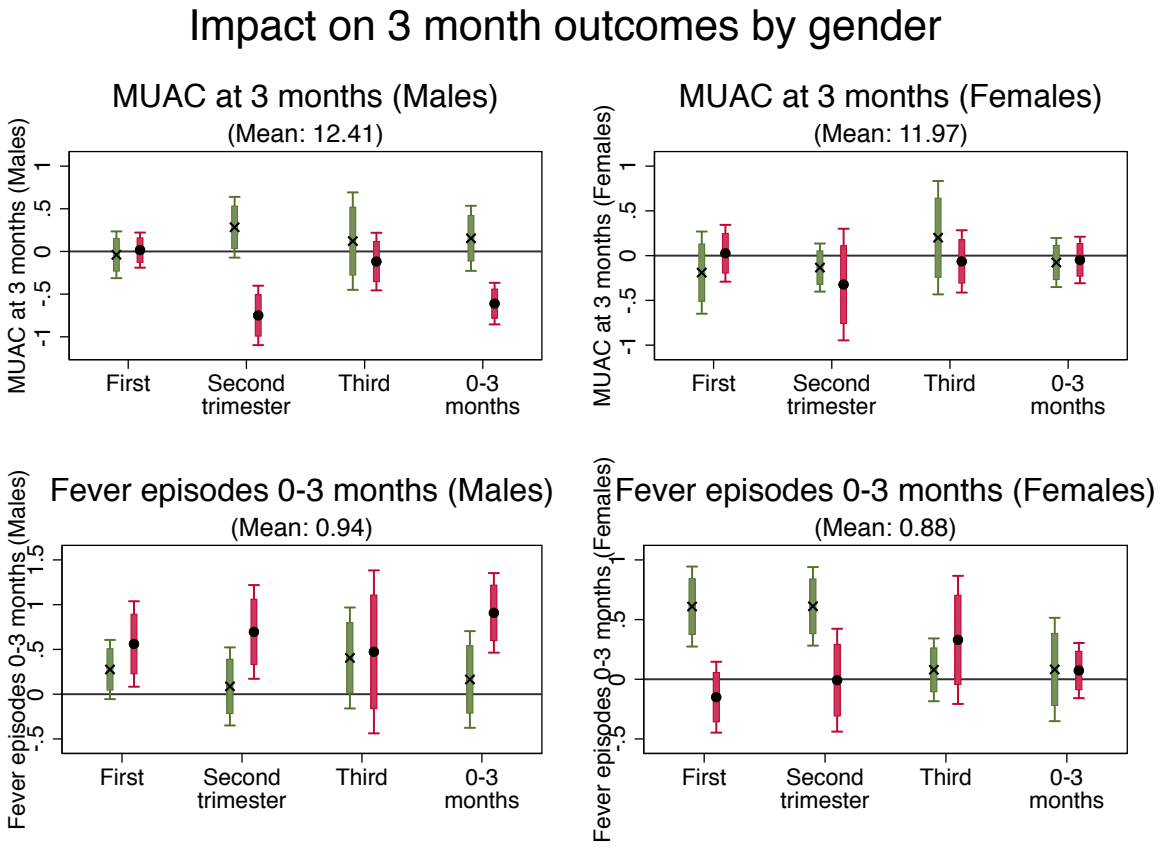


Figure 8: This figure shows the impact of the tornado on 3 month outcomes using the same triple-difference strategy and presentation as in Figure 6 but separately for males (left panels) and females (right panels). Refer to caption for Figure 6 for further details.

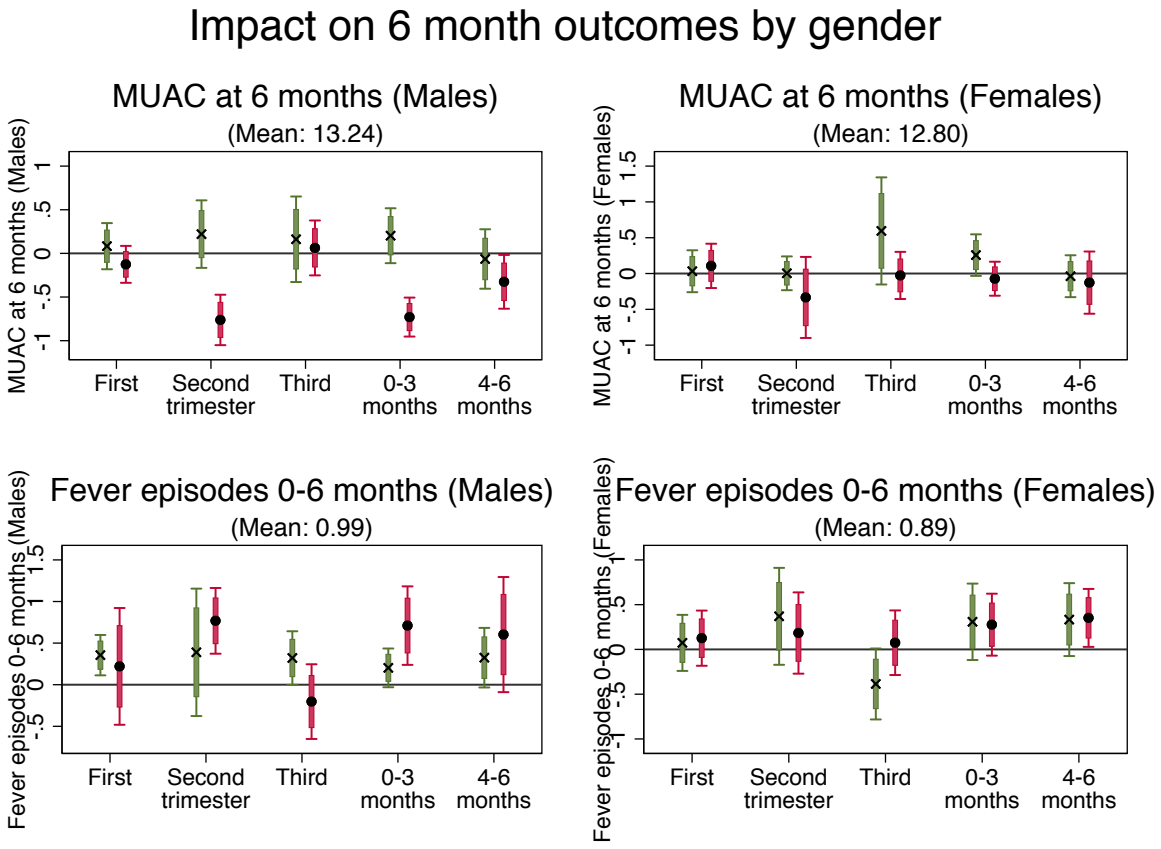


Figure 9: This figure shows the impact of the tornado on 6 month outcomes using the same triple-difference strategy and presentation as in Figure 6 but separately for males (left panels) and females (right panels). Refer to caption for Figure 6 for further details.

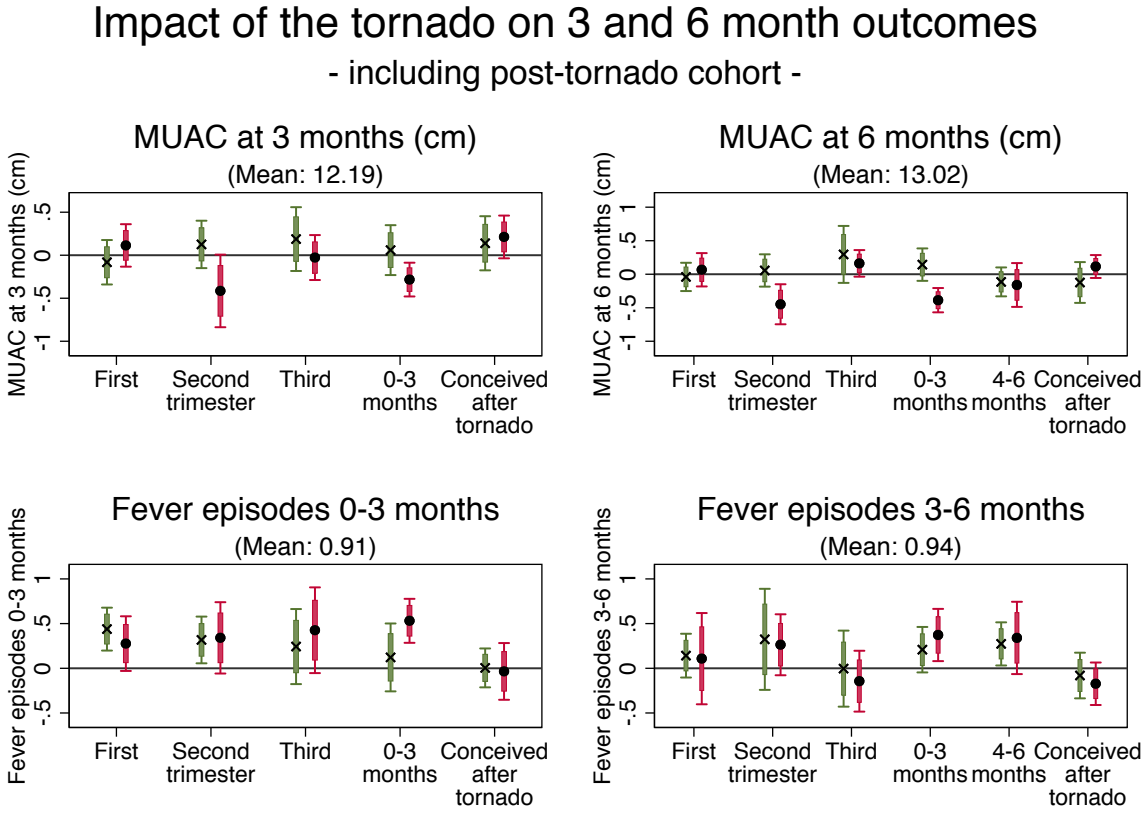


Figure 10: This figure shows the impact of the tornado on 3 and 6 month outcomes using the triple-difference strategy described by Equation 1. We include a cohort dummy for infants conceived after the tornado (they are therefore removed from the comparison group for the other cohorts). Refer to caption for Figure 6 for further details.

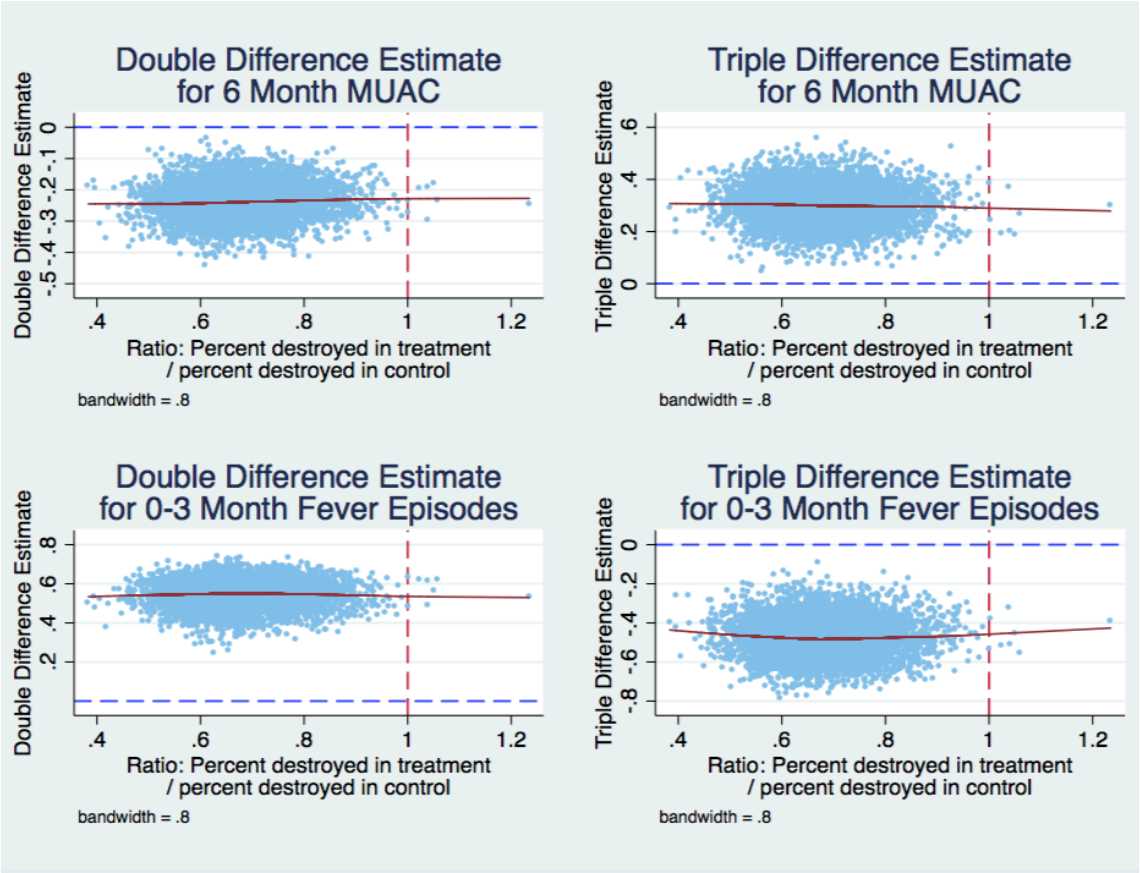


Figure 11: This figure shows the double (on the left) and triple (on the right) difference estimates from Equation 1 for 6 month MUAC (in the top two panels) and 0-3 month fever (in the bottom two panels). Each point in a figure is taken from a separate regression where we exclude from 1 to 10 sectors at random from within the tornado area. The X-axis is the ratio of the share of houses destroyed in treatment sectors to the share of houses destroyed in the control sectors for the particular sample that is used for the estimate being reported.

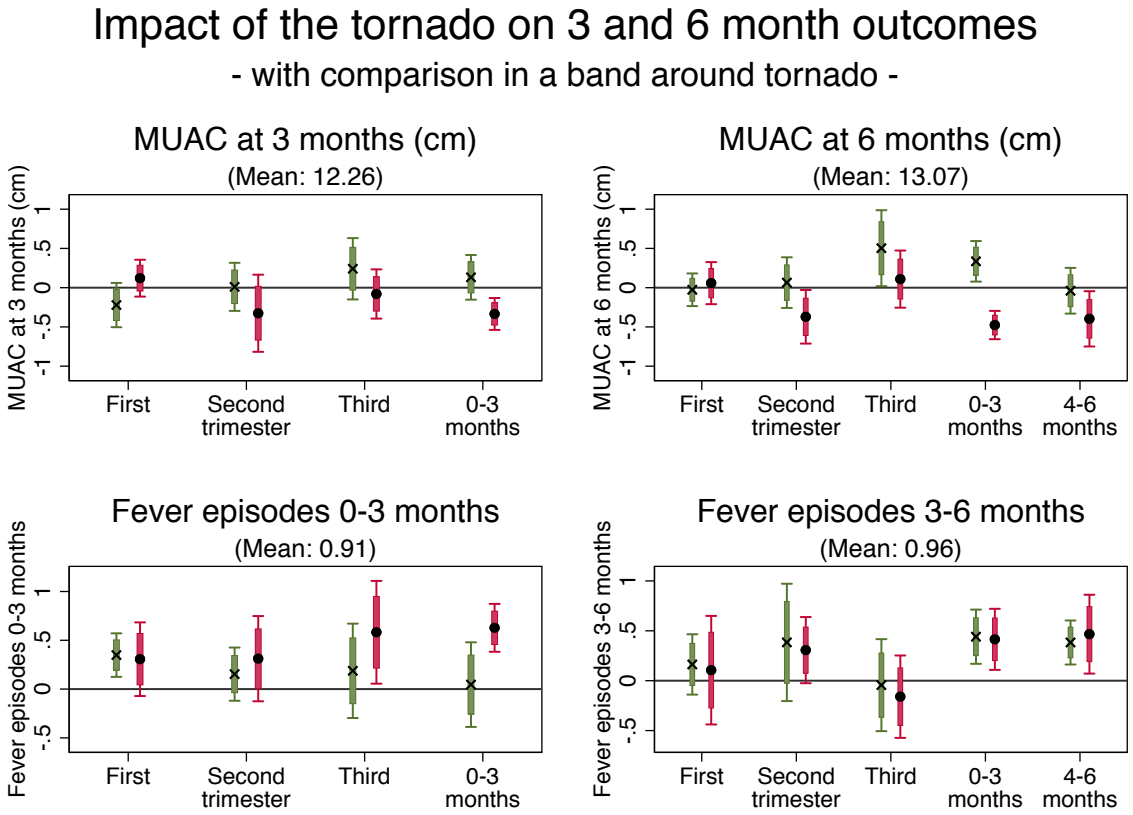


Figure 12: This figure shows the impact of the tornado on 3 and 6 month outcomes using the triple-difference strategy described by Equation 1. To construct this figure we sorted the unexposed sectors by their distance to the tornado and include only sectors that are among the 20% that are closest to the tornado. Refer to caption for Figure 6 for further details.

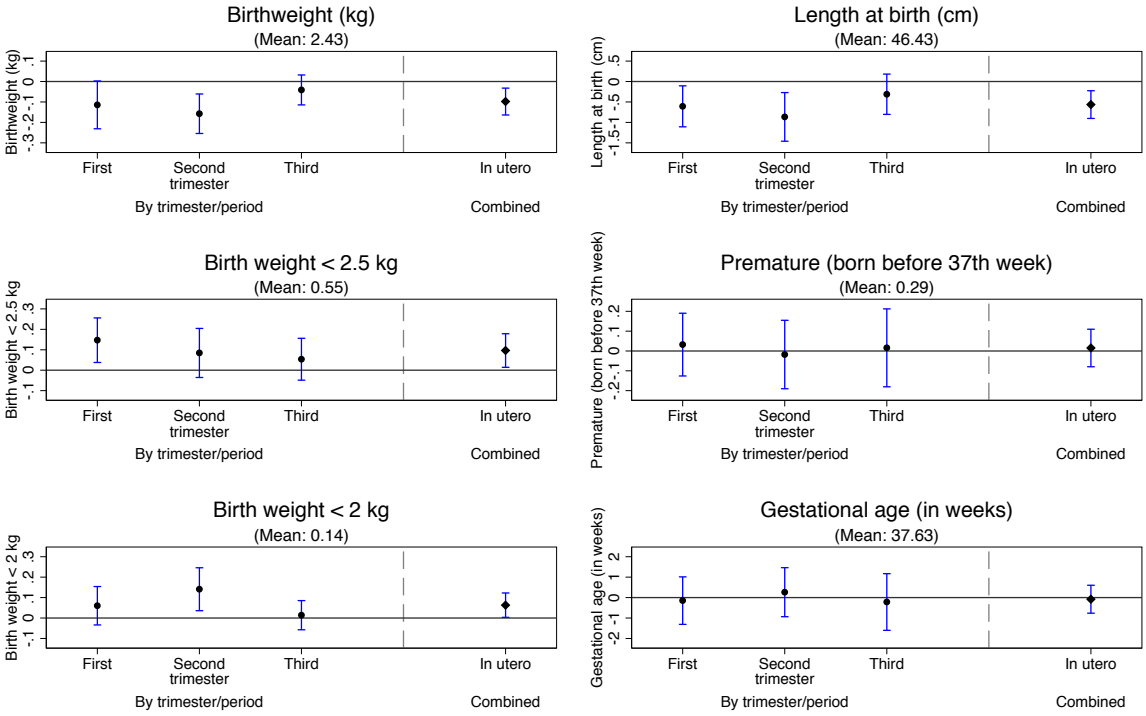


Figure 13: This figure shows the impact of the tornado on birth outcomes using the double-difference strategy described by Equation 2 except that the cohorts are defined by counting forward from the likely date of conception (ignoring data on birth date) instead of our normal cohort definition. Each panel presents results for one outcome variable. In each panel, we show point estimates and confidence intervals broken up by trimester of exposure (to the left of the dashed line) as well as results for combined *in utero* exposure (to the right of the dashed line). These two parts of the panel represent two different regressions.

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

									Within Tornado Area					
	All N = 5269		**Tornado N = 347**		**Non-tornado N = 4924**		**Difference**		**Vitamin A N = 186**		**Placebo N = 161**		**Difference**	
	Mean	SD	Mean	SD	Mean	SD	Mean	SE	Mean	SD	Mean	SD	Mean	SE
Infant birth anthropometry														
Weight (kg)	2.49	0.44	2.51	0.41	2.49	0.44	0.03	0.03	2.54	0.38	2.49	0.43	0.04	0.06
Height (cm)	46.64	2.39	46.61	2.23	46.64	2.40	−0.03	0.17	46.80	2.09	46.40	2.37	0.40	0.31
MUAC (cm)	9.43	0.86	9.48	0.81	9.42	0.86	0.05	0.06	9.50	0.73	9.44	0.89	0.06	0.11
Head Circumference (cm)	32.65	1.64	32.73	1.71	32.65	1.63	0.09	0.12	32.82	1.58	32.63	1.84	0.19	0.24
Chest Circumference (cm)	30.70	2.14	30.77	1.96	30.70	2.15	0.07	0.15	30.80	1.74	30.73	2.18	0.07	0.27
Infant anthropometry at 3 months														
MUAC (cm)	12.37	1.07	12.43	1.07	12.36	1.07	0.06	0.06	12.43	1.02	12.42	1.14	0.01	0.12
Head Circ. (cm)	38.70	1.49	38.58	1.45	38.71	1.49	−0.13	0.09	38.51	1.33	38.67	1.59	−0.16	0.17
Chest Circumference (cm)	38.87	2.24	38.98	2.17	38.86	2.25	0.12	0.13	38.82	2.15	39.18	2.18	−0.36	0.25
Anthropometric Index	0.17	0.98	0.17	0.97	0.17	0.99	0.00	0.06	0.13	0.91	0.22	1.05	−0.09	0.11
Infant anthropometry at 6 months														
MUAC	13.08	1.05	13.20	1.06	13.07	1.05	0.12	0.06**	13.26	1.05	13.12	1.07	0.14	0.12
Head Circumference (cm)	40.88	1.42	41.00	1.39	40.87	1.43	0.13	0.08	40.96	1.41	41.05	1.36	−0.09	0.16
Chest Circumference (cm)	41.32	2.13	41.58	2.11	41.30	2.13	0.28	0.13**	41.49	2.13	41.67	2.10	−0.18	0.24
Anthropometric Index	0.04	0.99	0.16	1.00	0.03	0.98	0.13	0.06**	0.17	1.02	0.16	0.99	0.01	0.11
Other infant outcomes														
Gender is Male	0.51	0.50	0.54	0.50	0.51	0.50	0.03	0.03	0.51	0.50	0.57	0.50	−0.06	0.05
Fever Incidence, 0-3 months	0.59	0.49	0.56	0.50	0.59	0.49	−0.03	0.03	0.56	0.50	0.55	0.50	0.01	0.06
Fever Incidence 0-6 months	0.55	0.50	0.57	0.50	0.55	0.50	0.02	0.03	0.58	0.50	0.57	0.50	0.00	0.06
Mortality 0-24 weeks	0.06	0.23	0.04	0.20	0.06	0.24	−0.02	0.01	0.05	0.22	0.04	0.19	0.01	0.02
Maternal characteristics														
Parity	1.33	2.40	1.22	1.48	1.34	2.46	−0.11	0.13	1.36	1.64	1.07	1.26	0.29	0.16*
LSI	−0.04	0.99	−0.11	0.93	−0.04	0.99	−0.07	0.05	−0.09	0.96	−0.13	0.90	0.03	0.10
Height (cm)	149.32	5.15	148.98	5.09	149.35	5.15	−0.37	0.29	148.67	4.97	149.33	5.20	−0.67	0.55
MUAC (cm)	22.65	1.93	22.61	2.00	22.66	1.92	−0.05	0.11	22.58	2.07	22.65	1.91	−0.06	0.22
Education (years)	3.61	4.03	3.54	3.93	3.62	4.04	−0.08	0.22	3.91	4.24	3.11	3.50	0.80	0.42*
Dosing														
Dosed ≤ 6 hours	0.34	0.47	0.40	0.49	0.34	0.47	0.06	0.03**	0.37	0.48	0.43	0.50	−0.07	0.05
Dosed ≤ 12 hours	0.44	0.50	0.49	0.50	0.44	0.50	0.06	0.03**	0.48	0.50	0.51	0.50	−0.03	0.05
Dosed ≤ 18 hours	0.49	0.50	0.54	0.50	0.49	0.50	0.05	0.03*	0.52	0.50	0.56	0.50	−0.04	0.05
Dosed ≤ 24 hours	0.52	0.50	0.56	0.50	0.52	0.50	0.04	0.03	0.55	0.50	0.57	0.50	−0.02	0.05
Dosed ≤ 7 days	0.59	0.49	0.63	0.48	0.59	0.49	0.04	0.03	0.65	0.48	0.62	0.49	0.02	0.05

Summary statistics for the study sample (infant sample), limited to infants born at least 9 months before the tornado. Tornado and Non-Tornado refer to inside vs. outside the tornado area. The last three columns restrict the sample to only within the tornado area. OLS standard errors and associated p-values reported.
Significance: * < 0.1; ** < 0.05; *** < 0.01.

Table 2: **Summary Statistics of Infants in the Pre-tornado Cohorts Excluding Sectors Further Away from the Tornado**

									Within Tornado Area					
	All N = 2557		**Tornado N = 347**		**Non-tornado N = 2210**		**Difference**		**Vitamin A N = 186**		**Placebo N = 161**		**Difference**	
	Mean	SD	Mean	SD	Mean	SD	Mean	SE	Mean	SD	Mean	SD	Mean	SE
Infant birth anthropometry														
Weight (kg)	2.50	0.43	2.51	0.41	2.50	0.43	0.01	0.03	2.54	0.38	2.49	0.43	0.04	0.06
Height (cm)	46.67	2.36	46.61	2.23	46.68	2.38	−0.06	0.18	46.80	2.09	46.40	2.37	0.40	0.31
MUAC (cm)	9.47	0.83	9.48	0.81	9.47	0.83	0.00	0.06	9.50	0.73	9.44	0.89	0.06	0.11
Head Circumference (cm)	32.67	1.65	32.73	1.71	32.66	1.64	0.08	0.12	32.82	1.58	32.63	1.84	0.19	0.24
Chest Circumference (cm)	30.74	2.17	30.77	1.96	30.73	2.20	0.04	0.16	30.80	1.74	30.73	2.18	0.07	0.27
Infant anthropometry at 3 months														
MUAC (cm)	12.45	1.09	12.43	1.07	12.46	1.09	−0.03	0.07	12.43	1.02	12.42	1.14	0.01	0.12
Head Circ. (cm)	38.73	1.50	38.58	1.45	38.76	1.50	−0.17	0.09*	38.51	1.33	38.67	1.59	−0.16	0.17
Chest Circumference (cm)	38.96	2.25	38.98	2.17	38.95	2.27	0.03	0.14	38.82	2.15	39.18	2.18	−0.36	0.25
Anthropometric Index	0.22	1.00	0.17	0.97	0.23	1.01	−0.06	0.06	0.13	0.91	0.22	1.05	−0.09	0.11
Infant anthropometry at 6 months														
MUAC	13.15	1.06	13.20	1.06	13.14	1.06	0.06	0.06	13.26	1.05	13.12	1.07	0.14	0.12
Head Circumference (cm)	40.87	1.46	41.00	1.39	40.85	1.47	0.15	0.09*	40.96	1.41	41.05	1.36	−0.09	0.16
Chest Circumference (cm)	41.40	2.13	41.58	2.11	41.37	2.13	0.21	0.13	41.49	2.13	41.67	2.10	−0.18	0.24
Anthropometric Index	0.08	1.00	0.16	1.00	0.07	1.00	0.09	0.06	0.17	1.02	0.16	0.99	0.01	0.11
Other infant outcomes														
Gender is Male	0.51	0.50	0.54	0.50	0.50	0.50	0.04	0.03	0.51	0.50	0.57	0.50	−0.06	0.05
Fever Incidence, 0-3 months	0.59	0.49	0.56	0.50	0.59	0.49	−0.03	0.03	0.56	0.50	0.55	0.50	0.01	0.06
Fever Incidence 0-6 months	0.56	0.50	0.57	0.50	0.55	0.50	0.02	0.03	0.58	0.50	0.57	0.50	0.00	0.06
Mortality 0-24 weeks	0.06	0.23	0.04	0.20	0.06	0.24	−0.02	0.01	0.05	0.22	0.04	0.19	0.01	0.02
Maternal characteristics														
Parity	1.31	2.43	1.22	1.48	1.32	2.55	−0.10	0.14	1.36	1.64	1.07	1.26	0.29	0.16*
LSI	−0.04	1.01	−0.11	0.93	−0.03	1.02	−0.08	0.06	−0.09	0.96	−0.13	0.90	0.03	0.10
Height (cm)	149.34	5.06	148.98	5.09	149.40	5.06	−0.42	0.29	148.67	4.97	149.33	5.20	−0.67	0.55
MUAC (cm)	22.66	1.97	22.61	2.00	22.67	1.97	−0.06	0.11	22.58	2.07	22.65	1.91	−0.06	0.22
Education (years)	3.54	4.01	3.54	3.93	3.54	4.02	0.00	0.23	3.91	4.24	3.11	3.50	0.80	0.42*
Dosing														
Dosed $\leq$ 6 hours	0.36	0.48	0.40	0.49	0.35	0.48	0.04	0.03	0.37	0.48	0.43	0.50	−0.07	0.05
Dosed $\leq$ 12 hours	0.45	0.50	0.49	0.50	0.45	0.50	0.05	0.03	0.48	0.50	0.51	0.50	−0.03	0.05
Dosed $\leq$ 18 hours	0.50	0.50	0.54	0.50	0.50	0.50	0.04	0.03	0.52	0.50	0.56	0.50	−0.04	0.05
Dosed $\leq$ 24 hours	0.53	0.50	0.56	0.50	0.52	0.50	0.03	0.03	0.55	0.50	0.57	0.50	−0.02	0.05
Dosed $\leq$ 7 days	0.60	0.49	0.63	0.48	0.59	0.49	0.04	0.03	0.65	0.48	0.62	0.49	0.02	0.05

Summary statistics for the study sample (infant sample), limited to infants born at least 9 months before the tornado. The sample is further restricted from the sample used in Table 1 by excluding sectors where the distance of the centroid from the tornado destruction area is less than the median for such distances (to include only sectors in a band around the tornado). Tornado and Non-Tornado refer to inside vs. outside the tornado area. The last three columns restrict the sample to only within the tornado area. OLS standard errors and associated p-values reported.

Significance: * < 0.1; ** < 0.05; *** < 0.01.

Table 3: **Birth outcomes**

	Birth weight	Low birth weight	Very low birth weight (< 2kg)	Height at birth	Gestational length	Premature
In tornado area X ...						
In utero	−0.10 ∗ ∗∗	0.13 ∗ ∗∗	0.07 ∗ ∗	−0.53 ∗ ∗∗	0.01	0.01
	(0.03)	(0.04)	(0.03)	(0.16)	(0.04)	(0.26)
In tornado area X ...						
First trimester	−0.09	0.14 ∗ ∗	0.05	−0.45∗	0.02	0.01
	(0.06)	(0.06)	(0.05)	(0.26)	(0.08)	(0.62)
Second trimester	−0.15 ∗ ∗∗	0.10∗	0.13 ∗ ∗	−0.81 ∗ ∗∗	−0.01	0.02
	(0.05)	(0.06)	(0.05)	(0.30)	(0.10)	(0.72)
Third trimester	−0.06∗	0.13 ∗ ∗∗	0.03	−0.35	0.01	−0.05
	(0.03)	(0.05)	(0.03)	(0.23)	(0.06)	(0.42)
Dependent variable mean	2.43	0.55	0.14	46.43	0.29	37.63
Observations	14017	14017	14017	13652	19033	19033

Impact of the tornado on birth outcomes using the double-difference strategy described in Equation 2. In parenthesis are standard errors clustered within categories defined by the interaction of "In Tornado Area", "VitA", and year and month of birth. Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 4: **Impact on number of fevers and anthropometry**

	At 3 months			At 6 months		
	Fever episodes	AI	MUAC	Fever episodes	AI	MUAC
In tornado area X ...						
First trimester	0.28*	0.06	0.04	0.19	0.09	0.01
	(0.15)	(0.12)	(0.11)	(0.26)	(0.13)	(0.12)
Second trimester	0.35*	−0.59***	−0.49**	0.35**	−0.65***	−0.50***
	(0.20)	(0.16)	(0.21)	(0.16)	(0.14)	(0.15)
Third trimester	0.44*	−0.05	−0.10	−0.07	0.14	0.11
	(0.24)	(0.13)	(0.12)	(0.17)	(0.10)	(0.09)
Age 0-3 months	0.54***	−0.33***	−0.36***	0.45***	−0.35***	−0.43***
	(0.12)	(0.10)	(0.09)	(0.14)	(0.09)	(0.08)
In tornado area X Vitamin A X ...						
First trimester	0.15	−0.24	−0.17	−0.01	−0.22	0.00
	(0.18)	(0.16)	(0.16)	(0.28)	(0.15)	(0.15)
Second trimester	−0.02	0.68***	0.57**	0.01	0.72***	0.61***
	(0.24)	(0.20)	(0.24)	(0.33)	(0.20)	(0.19)
Third trimester	−0.19	0.22	0.24	0.10	0.13	0.25
	(0.32)	(0.18)	(0.21)	(0.27)	(0.20)	(0.23)
Age 0-3 months	−0.42*	0.35**	0.37**	−0.20	0.48***	0.63***
	(0.22)	(0.16)	(0.16)	(0.18)	(0.16)	(0.14)
Dependent variable mean	0.91	0.00	12.19	0.94	0.00	13.02
Observations	16942	16490	16636	16765	16226	16370

Regression models of infant development measured by number of fever episodes and anthropometry at 3 and 6 months of age. The outcome variables are: In columns 1 and 4, fever episodes in 0-3 months and 4-6 months, top coded at 4 (>4 episodes are coded as 4); In columns 2 and 5, an anthropometric index (AI) that is a standardized (zero mean, unit SD) average of three anthropometric measurements (mid-upper arm circumference, head circumference and chest circumference) after each has been standardized to zero mean and unit standard deviation. Each anthropometric variable is winsorized at 1%. "Vit A" is an indicator that is 1 if infants in the sector were given vitamin A and zero if they were in the placebo group. "In Tornado Area" is an indicator defined as 1 if any households in the sector were destroyed in the tornado. Each regression contains randomization sector fixed effects (this absorbs main effects of the tornado area and treatment indicators). In parenthesis are standard errors clustered within categories defined by the interaction of "In Tornado Area", "VitA", and year and month of birth.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 5: **Impacts by gender at 3 months**

	Anthropometry				Fever	
	Males		Females		Males	Females
	AI	MUAC	AI	MUAC		
In tornado area X ...						
First trimester	0.09	0.02	−0.01	0.03	0.56 ∗ ∗	−0.15
	(0.11)	(0.10)	(0.14)	(0.16)	(0.24)	(0.15)
Second trimester	−0.67 ∗ ∗∗	−0.75 ∗ ∗∗	−0.52∗	−0.32	0.70 ∗ ∗∗	−0.01
	(0.13)	(0.18)	(0.29)	(0.31)	(0.26)	(0.22)
Third trimester	−0.02	−0.12	−0.08	−0.06	0.47	0.33
	(0.18)	(0.17)	(0.14)	(0.18)	(0.46)	(0.27)
Age 0-3 months	−0.51 ∗ ∗∗	−0.61 ∗ ∗∗	−0.03	−0.05	0.91 ∗ ∗∗	0.07
	(0.12)	(0.12)	(0.13)	(0.13)	(0.22)	(0.12)
In tornado area X Vitamin A X ...						
First trimester	−0.14	−0.05	−0.20	−0.22	−0.28	0.76 ∗ ∗∗
	(0.14)	(0.17)	(0.24)	(0.28)	(0.29)	(0.23)
Second trimester	1.01 ∗ ∗∗	1.03 ∗ ∗∗	0.35	0.19	−0.61∗	0.62 ∗ ∗
	(0.21)	(0.25)	(0.31)	(0.34)	(0.34)	(0.27)
Third trimester	0.20	0.24	0.28	0.26	−0.07	−0.25
	(0.23)	(0.34)	(0.29)	(0.37)	(0.54)	(0.30)
Age 0-3 months	0.68 ∗ ∗∗	0.76 ∗ ∗∗	−0.01	−0.03	−0.74 ∗ ∗	0.01
	(0.23)	(0.23)	(0.19)	(0.19)	(0.35)	(0.25)
Dependent variable mean	0.30	12.41	−0.31	11.97	0.94	0.88
Observations	8395	8467	8095	8169	8645	8297

Specifications and variable descriptions are identical to Table 4. Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 6: **Impacts by gender at 6 months**

	Anthropometry				Fever	
	Males		Females		Males	Females
	AI	MUAC	AI	MUAC		
In tornado area X ...						
First trimester	−0.02	−0.12	0.16	0.09	0.23	0.14
	(0.13)	(0.11)	(0.17)	(0.16)	(0.35)	(0.16)
Second trimester	−0.65 ∗ ∗∗	−0.76 ∗ ∗∗	−0.59 ∗ ∗∗	−0.34	0.78 ∗ ∗∗	0.20
	(0.16)	(0.15)	(0.23)	(0.29)	(0.20)	(0.23)
Third trimester	0.11	0.05	0.04	−0.03	−0.20	0.08
	(0.15)	(0.16)	(0.14)	(0.17)	(0.23)	(0.19)
Age 0-3 months	−0.52 ∗ ∗∗	−0.72 ∗ ∗∗	−0.07	−0.07	0.72 ∗ ∗∗	0.28
	(0.11)	(0.11)	(0.14)	(0.12)	(0.24)	(0.17)
Age 3-6 months	−0.16	−0.33 ∗ ∗	−0.16	−0.14	0.61∗	0.37 ∗ ∗
	(0.15)	(0.16)	(0.16)	(0.22)	(0.34)	(0.16)
In tornado area X Vitamin A X ...						
First trimester	0.01	0.21	−0.24	−0.06	0.14	−0.06
	(0.17)	(0.17)	(0.23)	(0.22)	(0.37)	(0.22)
Second trimester	0.84 ∗ ∗∗	0.98 ∗ ∗∗	0.58 ∗ ∗	0.35	−0.39	0.18
	(0.23)	(0.24)	(0.26)	(0.31)	(0.44)	(0.36)
Third trimester	−0.02	0.13	0.45	0.63	0.50∗	−0.46∗
	(0.25)	(0.29)	(0.35)	(0.41)	(0.28)	(0.28)
Age 0-3 months	0.65 ∗ ∗∗	0.93 ∗ ∗∗	0.27	0.33∗	−0.52∗	0.04
	(0.19)	(0.19)	(0.21)	(0.19)	(0.27)	(0.28)
Age 3-6 months	0.04	0.28	0.13	0.11	−0.29	−0.04
	(0.23)	(0.24)	(0.21)	(0.26)	(0.39)	(0.26)
Dependent variable mean	0.34	13.24	−0.35	12.80	0.99	0.89
Observations	8241	8311	7985	8059	8529	8236

Specifications and variable descriptions are identical to Table 4. Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 7: **Impacts on miscarriage and stillbirth**

	Miscarriage	Abortion	Live birth
	Panel A: Whole in-utero period		
In tornado area X in-utero	0.01	-0.01	0.02
	(0.03)	(0.04)	(0.07)
Dependent variable mean	0.11	0.16	0.69
Observations	26099	26099	26099
	Panel B: By trimesters		
In tornado area X first trimester	0.03	-0.03	-0.01
	(0.03)	(0.05)	(0.08)
In tornado area X second trimester	-0.01	-0.01	0.06*
	(0.02)	(0.02)	(0.03)
In tornado area X third trimester	0.01	-0.01	0.05*
	(0.01)	(0.02)	(0.03)
Dependent variable mean	0.11	0.16	0.69
Observations	26099	26099	26099

This table reports impacts of the tornado using a similar double-difference strategy as in other parts of the paper except that cohorts are defined in an alternative way from other parts of the paper (since we can't 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 three trimesters are defined as the 0-90, 91-180 and 181-270 days after the last menstrual period, respectively, or up to the date of outcome (whichever comes earlier). 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)). Three percent of pregnancies ended in stillbirth and the remaining possible outcomes (mom died, multiple births and other) accounted for two percent. Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 8: **Attrition by 3 and 6 months**

	3 month measures		6 month measures	
	Missing	Missing or late	Missing	Missing or late
In tornado area X ...				
First trimester	−0.02	−0.04	−0.01	−0.00
	(0.03)	(0.03)	(0.03)	(0.03)
Second trimester	0.04	0.06	0.12	0.12
	(0.09)	(0.09)	(0.08)	(0.08)
Third trimester	0.05	0.05	0.06	0.07
	(0.03)	(0.05)	(0.05)	(0.05)
Age 0-3 months	0.02	0.01	0.02	0.01
	(0.04)	(0.04)	(0.03)	(0.03)
In tornado area X Vitamin A X ...				
First trimester	0.06	0.06	0.06	0.05
	(0.05)	(0.06)	(0.05)	(0.05)
Second trimester	−0.03	−0.06	−0.11	−0.13
	(0.10)	(0.09)	(0.09)	(0.09)
Third trimester	0.01	−0.03	−0.08	−0.12∗
	(0.05)	(0.06)	(0.07)	(0.07)
Age 0-3 months	−0.05	−0.06	−0.03	−0.02
	(0.05)	(0.05)	(0.05)	(0.05)
Dependent variable mean	0.76	0.76	0.77	0.77
Observations	19033	19033	19033	19033

Attrition in the data by cohort. The dependent variable in columns 1 and 3 is a dummy indicating missing values for 3-month and 6-month anthropometry. The dependent variable in columns 2 and 4 is the same as the odd columns except that infants measured late (8 weeks after the target date) are also coded as missing. Our main outcome measures used in the paper are set to missing after these cutoff dates so the even numbered columns correspond to the attrition for those main outcome measures. Each regression includes fixed effects for the randomization cluster (sector). In parenthesis are standard errors clustered within categories defined by the interaction of "In Tornado Area", "VitA", and year and month of birth.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 9: **Timing of dosing relative to birth**

	Dosed at <= 24 hours	Dosed at <= 7 days
In tornado area X ...		
First trimester	0.00	0.01
	(0.10)	(0.10)
Second trimester	0.18*	0.14
	(0.10)	(0.10)
Third trimester	−0.12	−0.05
	(0.10)	(0.11)
Age 0-3 months	0.14	0.07
	(0.09)	(0.10)
Age 3-6 months	−0.01	0.03
	(0.09)	(0.10)
In tornado area X Vitamin A X ...		
First trimester	0.01	−0.06
	(0.16)	(0.16)
Second trimester	−0.21	−0.25
	(0.15)	(0.16)
Third trimester	0.08	0.04
	(0.14)	(0.16)
Age 0-3 months	−0.16	−0.12
	(0.15)	(0.16)
Age 3-6 months	0.05	−0.04
	(0.14)	(0.16)
Dependent variable mean	0.67	0.76
Observations	19033	19033

Regression models of time at dosing using our main specifications. Each regression includes fixed effects for the randomization cluster (sector). In parenthesis are standard errors clustered within categories defined by the interaction of "In Tornado Area", "VitA", and year and month of birth.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table 10: **Impact on number of fevers and anthropometry (Excluding the most damaged sectors)**

	At 3 months			At 6 months		
	Fever episodes	AI	MUAC	Fever episodes	AI	MUAC
In tornado area X ...						
First trimester	0.26∗	−0.01	0.03	0.08	0.08	0.00
	(0.15)	(0.12)	(0.11)	(0.25)	(0.09)	(0.08)
Second trimester	0.55 ∗ ∗∗	−0.48 ∗ ∗∗	−0.29	0.59 ∗ ∗∗	−0.54 ∗ ∗∗	−0.26
	(0.19)	(0.18)	(0.22)	(0.20)	(0.18)	(0.19)
Third trimester	0.53∗	−0.20	−0.21	−0.08	0.14	0.14
	(0.29)	(0.16)	(0.16)	(0.21)	(0.15)	(0.15)
Age 0-3 months	0.50 ∗ ∗∗	−0.47 ∗ ∗∗	−0.47 ∗ ∗∗	0.52 ∗ ∗∗	−0.53 ∗ ∗∗	−0.55 ∗ ∗∗
	(0.11)	(0.10)	(0.10)	(0.09)	(0.10)	(0.09)
In tornado area X Vitamin A X ...						
First trimester	0.18	−0.17	−0.16	0.10	−0.20	0.01
	(0.19)	(0.16)	(0.16)	(0.28)	(0.13)	(0.12)
Second trimester	−0.23	0.57 ∗ ∗∗	0.36	−0.23	0.61 ∗ ∗∗	0.37
	(0.23)	(0.22)	(0.26)	(0.35)	(0.22)	(0.23)
Third trimester	−0.28	0.37∗	0.35	0.10	0.13	0.22
	(0.36)	(0.20)	(0.24)	(0.30)	(0.23)	(0.26)
Age 0-3 months	−0.38∗	0.50 ∗ ∗∗	0.48 ∗ ∗∗	−0.27∗	0.66 ∗ ∗∗	0.75 ∗ ∗∗
	(0.22)	(0.16)	(0.16)	(0.15)	(0.16)	(0.15)
Dependent variable mean	0.91	0.00	12.19	0.94	0.00	13.02
Observations	16782	16332	16477	16605	16070	16214

Regression models of infant development measured by number of fever episodes and anthropometry at 3 and 6 months of age. In this table we exclude the sectors with more than 80% damage. The outcome variables are: In columns 1 and 4, fever episodes in 0-3 months and 4-6 months, top coded at 4 (>4 episodes are coded as 4); In columns 2 and 5, an anthropometric index (AI) that is a standardized (zero mean, unit SD) average of three anthropometric measurements (mid-upper arm circumference, head circumference and chest circumference) after each has been standardized to zero mean and unit standard deviation. Each anthropometric variable is winsorized at 1%. "Vit A" is an indicator that is 1 if infants in the sector were given vitamin A and zero if they were in the placebo group. "In Tornado Area" is an indicator defined as 1 if any households in the sector were destroyed in the tornado. Each regression contains randomization sector fixed effects (this absorbs main effects of the tornado area and treatment indicators). In parenthesis are standard errors clustered within categories defined by the interaction of "In Tornado Area", "VitA", and year and month of birth.
Significance: * < 0.10; ** < 0.05; *** < 0.01.