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

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ABSTRACT

Severe environmental shocks have grown in frequency and intensity due to climate change. Can policy protect against the often devastating human impacts of these shocks, particularly for vulnerable populations?We study this question by leveraging data from a situation in which a tornado tore through an area involved in a double-blind cluster-randomized controlled trial of at-birth vitamin A supplementation in Bangladesh. Tornado exposure in utero and in infancy decreased birth size and physical growth, and increased the incidence of severe fevers. But infants who received vitamin A supplementation, which boosts immune system functioning, were protected from these effects. Tornado impacts and protective effects were both substantially larger for boys. Our results suggest that wide-scale supplementation policies would generate potential health benefits in disaster-prone areas of low-income countries.

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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). 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 (Currie and Vogl, 2013). Natural disasters in particular take an enormous toll on the survival and wellbeing of children. 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). Can policy intervention effectively protect vulnerable populations, especially young children, from the negative impacts of these shocks? Can such intervention help those who have been affected to recover?

Answering this set of questions rigorously is not straightforward. Governments, international organizations, and NGOs invest billions of dollars 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 mitigation 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 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.[1] As reported previously, this newborn vitamin A supplementation reduced infant mortality to six months of age by 15% (Klemm et al.,

[1]The newborn supplementation trial was actually nested into a larger trial evaluating the impacts of vitamin A and beta-carotene supplementation for pregnant mothers (Labrique et al., 2011). No impacts of prenatal supplementation on serum retinol (a biomarker of vitamin A deficiency) or maternal or infant health and mortality were found in this trial (West et al., 2011). The treatment was orthogonal to both the newborn supplementation treatment as well as to tornado exposure.

2008). The primary biological mechanism for this mortality reduction is vitamin A's crucial role in the development and functioning of infants' immune systems.[2]

On March 20, 2005, while the RCT was ongoing, a tornado tore through the study area, generating substantial property damage in 1007 of the 14893 households with live births 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, all of which were participating in the RCT and thus being longitudinally followed, allows for the assessment of effects on birth size (for infants exposed to the stress of the tornado *in utero*) and post-natal stress outcomes such as growth faltering and severe illness, both of which were being evaluated as part of the ongoing trial. 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 (for infants exposed to the tornado while *in utero*) and protective (for infants exposed after birth) effects of vitamin A supplementation on anthropometric and survey-based outcomes for infants at 0-6 months. We detail the identification assumptions necessary to obtain unbiased estimates of these effects, and provide empirical support for each of these assumptions.

In utero exposure 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. In control localities, tornado in the first three months of life had large negative impacts on mid-upper arm circumference (a reliable correlate of child mortality) and the incidence of severe fevers measured at both 3 and 6 months. But in treatment localities, in which all infants were dosed with vitamin A at birth, these impacts are nonexistent. These effects are entirely driven by impacts on boys; girls are largely unaffected by the tornado, even in control areas. The results are not driven by differential rates of miscarriage, stillbirth, or attrition from the sample after birth.

Environmental shocks can have devastating short- and long-run effects (Cas et al., 2014; Currie and Rossin-Slater, 2013; Frankenberg et al., 2008, 2011; Hornbeck, 2012; Karbownik and Wray, 2016). Moreover, these shocks will likely grow in frequency and intensity due to climate change, disproportionately affecting climate-vulnerable populations in low-income countries (Burke et al.,

[2]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).

2015). Insidious exposures (for example, to air pollution or heat stress) can have sizable long-run impacts as well – particularly for exposures at critical periods of fetal and early childhood development – and impacts that are generated early in infancy tend, if not corrected, to persist over the life cycle (Almond and Currie, 2011; Almond et al., 2017; Currie and Vogl, 2013). Measuring these early impacts, and evaluating the role for specific interventions that might mitigate negative effects so that they do not result in persistently poor health stocks, is therefore critically important from both the academic and policymaking perspectives. 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 supplementation, which both reduces the probability of early infection and decreases the extent to which infections translate into slower physical growth.

We also contribute to the literature on the early formation of health and human capital (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 protecting infants from shocks via supplementation 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, protection against early life disadvantage) seems to prevail (Adhvaryu et al., 2015; Bitler et al., 2014; Rossin-Slater and Wüst, 2015).

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 the increasingly devastating effects of natural disasters (Field, 2012). 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 disaster-prone areas of low-income countries.

The rest of the paper is organized as follows. Section 2 provides background on the Bangladeshi

context, the RCT, and the tornado event. Section 3 lays out our quasi-experimental research design, details assumptions needed to identify treatment effects, and reports on tests of those assumptions. Section 4 reports the main results, and section 5 concludes.

2 Context, RCT, and Tornado

2.1 Vitamin A and Infant Mortality Declines

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).[3] 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 either *in utero* or 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 (Keith P. West Jr, 1996).

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.

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

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; Identifiers: 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. 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 in Bangladesh. See Figure A1 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.

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 of vitamin A or a placebo once as oral oil drops from a capsule shortly after birth (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)). 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).

In this paper we focus the analysis solely on the newborn supplementation trial. As previously reported, the 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). The at-birth supplement, in contrast, had substantial 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). Consistent with these overall trial findings, we find that at-birth vitamin A supplementation promotes resilience in infancy. 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.

2.3 Tornado Event

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 S3). 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 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 obtained within 7 days in the tornado area at the rate of 84.5% and outside this area at the rate of 83.9%. These differences are small and not statistically different from 0. In Figure S2, we show balance in tornado damage intensity across vitamin A and placebo areas, statistically confirmed via a Kolmogorov-Smirnov test.

3 Research Design

3.1 Defining the Sample, Cohorts and Tornado Exposure

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.

To define tornado exposure we approximate the path of the tornado based on damages to homes in the area. 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 S3). By this definition 459 out of the 467 houses destroyed by the tornado (98%) are within the tornado path.

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 Identification

This section illustrates the research design and the assumptions required to identify the effect of the tornado and its interaction with the vitamin A supplementation. 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 treatment, E is being in an area exposed to the tornado (whether or not an infant is in an affected cohort) and C is an indicator for the affected cohort (whether or not an infant is within or outside the tornado affected area). This equation generalizes easily to multiple cohorts such as including impact both on the in-utero period and the period after birth. To formalize the assumptions underlying our double- and triple-difference estimation we describe the research design in a potential outcomes framework in this section. Let Y_i^{tec} be the potential outcome for infant i given treatment ($T = t$), tornado exposure ($E = e$) and cohort ($C = c$), where C is equal to 1 if the infant is part of the exposed cohort (whether he or she is in an area hit by the tornado or not). Here τ_i is the causal effect of the vitamin A supplementation on individual i, ω_i is the causal effect of tornado exposure, γ_i is the causal effect of the interaction of vitamin A supplementation and tornado exposure and ν_i is 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 2x2x2 research design we observe 8 moments that map to the OLS parameters:

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

The treatment was randomized and the path of the tornado was certainly independent of this treatment (we also show later that the tornado impact, in terms of infrastructure, was balanced across 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 $\chi_i \in \{\omega_i, \nu_i, u_i\}$:

$$E[\chi_i|T=1,E=0,C=0] = E[\chi_i|T=0,E=0,C=0] \quad (I)$$

$$E[\chi_i|T=1,E=1,C=0] = E[\chi_i|T=0,E=1,C=0] \quad (II)$$

$$E[\chi_i|T=1,E=0,C=1] = E[\chi_i|T=0,E=0,C=1] \quad (III)$$

$$E[\chi_i|T=1,E=1,C=1] = E[\chi_i|T=0,E=1,C=1] \quad (IV)$$

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 $\{\tau_i, \omega_i, \nu_i, u_i\}$:*

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

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

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

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

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 2x2x2 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

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

which, given that by construction $E[\nu_i|T = 0, E = 1, C = 1, S = 1] = E[\nu_i|T = 0, E = 0, C = 1, S = 1]$, is equivalent to

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

Now 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 2x2x2 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:

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

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 area that was affected by the tornado. 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

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

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.

3.3 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 S2 in the Supplementary Materials, which graphs

the intensity of the tornado (as measured by the share of houses destroyed by treatment sector) by treatment status, show that this is not the case. With respect to u_i we can test for balance in observables. In Table 1 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 1 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:

$$Y_i = \alpha_0 + \alpha_1 E_i + \alpha 2 b_i + \alpha 3 E_i \cdot b_i + \alpha_4 T_i + \alpha_5 T_i \cdot E_i + \alpha_6 T_i \cdot b_i + \alpha_7 T_i \cdot E_i \cdot b_i + v_i \qquad (2)$$

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 2 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 1: Evidence on the validity of Assumption 1 (I' - IV') based on observables

	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)	2.50	2.51	-0.00	2.50	2.50	-0.00	2.42	2.43	-0.01	2.45	2.47	-0.02
	(0.42)	(0.42)	(0.02)	(0.40)	(0.45)	(0.07)	(0.42)	(0.41)	(0.02)	(0.39)	(0.36)	(0.05)
MUAC at birth (cm)	9.43	9.47	-0.05	9.48	9.49	-0.01	9.23	9.27	-0.04	9.49	9.37	0.12
	(0.82)	(0.85)	(0.04)	(0.79)	(0.93)	(0.11)	(0.85)	(0.83)	(0.03)	(0.82)	(0.85)	(0.13)
Chest circ. at birth (cm)	30.77	30.79	-0.02	30.80	30.75	0.04	30.31	30.37	-0.06	30.47	30.51	-0.04
	(2.06)	(2.05)	(0.09)	(1.64)	(2.32)	(0.25)	(2.03)	(1.98)	(0.08)	(1.97)	(1.85)	(0.27)
Head circ. at birth (cm)	32.70	32.71	-0.01	32.72	32.62	0.10	32.39	32.37	0.02	32.65	32.52	0.13
	(1.59)	(1.56)	(0.07)	(1.39)	(1.94)	(0.20)	(1.59)	(1.60)	(0.06)	(1.52)	(1.39)	(0.18)
Height at birth (cm)	46.74	46.70	0.03	46.72	46.59	0.13	46.36	46.39	-0.03	46.32	46.52	-0.20
	(2.35)	(2.27)	(0.10)	(2.02)	(2.52)	(0.35)	(2.42)	(2.38)	(0.10)	(2.20)	(1.93)	(0.29)
Length of gestation (weeks)	37.86	37.73	0.13	38.00	37.54	0.46	37.75	37.82	-0.07	37.58	37.66	-0.08
	(2.96)	(2.94)	(0.10)	(2.81)	(3.03)	(0.31)	(2.90)	(2.87)	(0.10)	(3.05)	(2.82)	(0.39)
Living Standards Index	-0.02	-0.01	-0.01	-0.09	-0.04	-0.05	0.08	0.09	-0.01	0.15	0.01	0.14
	(0.99)	(0.99)	(0.04)	(0.98)	(0.96)	(0.12)	(0.99)	(1.01)	(0.04)	(0.94)	(0.95)	(0.13)
Maternal MUAC	22.71	22.62	0.09	22.62	22.75	-0.13	23.06	23.01	0.06	23.01	22.79	0.22
	(1.91)	(1.96)	(0.06)	(2.05)	(1.91)	(0.24)	(2.05)	(1.99)	(0.07)	(1.93)	(1.80)	(0.26)
Maternal height	149.41	149.41	-0.00	148.70	149.49	-0.80	149.59	149.53	0.05	149.72	149.32	0.40
	(5.12)	(5.07)	(0.16)	(4.75)	(5.32)	(0.53)	(5.11)	(5.25)	(0.18)	(4.96)	(4.88)	(0.60)
Mother's age at enrollment	20.27	20.57	-0.30*	20.44	19.34	1.10	19.93	19.95	-0.02	19.88	20.64	-0.76
	(5.22)	(5.44)	(0.16)	(5.65)	(5.02)	(0.69)	(5.32)	(5.33)	(0.18)	(5.86)	(5.47)	(0.62)
Mother's years of education	3.70	3.61	0.09	3.97	3.59	0.37	4.00	4.03	-0.03	3.87	3.69	0.19
	(4.04)	(4.06)	(0.14)	(4.28)	(3.70)	(0.52)	(4.06)	(4.01)	(0.14)	(4.07)	(4.04)	(0.46)
Father's years of education	3.69	3.68	0.01	3.71	3.34	0.36	3.87	3.83	0.05	3.61	3.42	0.19
	(4.53)	(4.54)	(0.15)	(4.46)	(4.19)	(0.50)	(4.64)	(4.60)	(0.17)	(4.37)	(4.60)	(0.49)
Observations	2,115	2,089	4,204	148	164	312	1,837	1,855	3,692	157	140	297

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

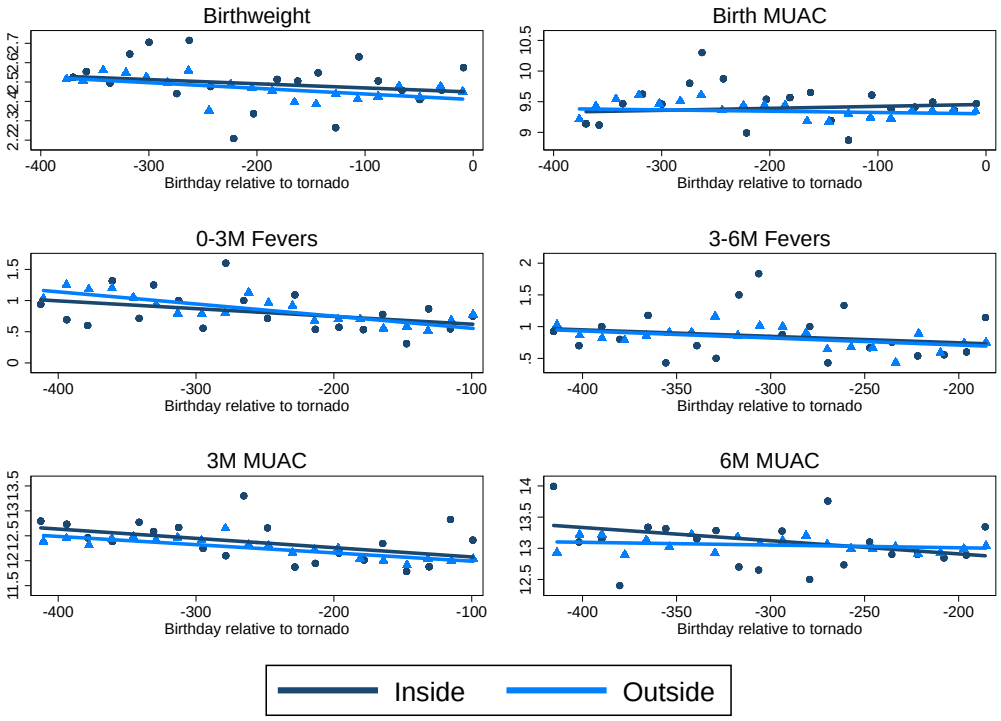

Figure 1: Evidence on the validity of Assumption 2. The figures show pre-trends in outcomes for control sectors in the tornado affected area.

Table 2: 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
	(0.03)	(0.05)	(0.13)	(0.10)	(0.15)	(0.07)	(0.07)	(0.11)	(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
	(0.04)	(0.07)	(0.18)	(0.14)	(0.21)	(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 shows estimates based on Equation 2 to test the validity of Assumptions 2 and 3. Significance levels: $* < 0.1$; $** < 0.05$; $*** < 0.01$.

3.4 Estimation

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. Please see details of this procedure in section A1.3.

4 Results

4.1 Illustration of Research Strategy

Table 3: Group Means for Severe Fever Incidence, 0-3 Months

	Control		Vitamin A	
	Mean	(SE)	Mean	(SE)
	Age > 6 Months at Time of Tornado			
Not in Tornado Area	0.98	(0.02)	0.95	(0.02)
In Tornado Area	0.88	(0.08)	0.87	(0.08)
Difference[a]	−0.09	(0.09)	−0.08	(0.09)
	Age 0-3 Months at Time of Tornado			
Not in Tornado Area	0.84	(0.03)	0.86	(0.03)
In Tornado Area	1.30	(0.14)	0.97	(0.13)
Difference[b]	0.45 ∗ ∗∗	(0.13)	0.11	(0.12)
Difference (b) - (a)	0.55 ∗ ∗∗	(0.16)	0.19	(0.15)
Difference VA - PL			−0.36	(0.22)

This table shows group means and differences of the incidence of severe fever in the first 3 months of life, by tornado exposure, birth cohort, and vitamin A treatment status, as an illustration of the research strategy. Conventional (OLS) standard errors are reported in parenthesis. Significance levels: ∗ < 0.1; ∗∗ < 0.05; ∗ ∗ ∗ < 0.01.

To illustrate our estimation strategy, in Table 3 we report group means for one of our primary outcomes, (mothers') self-reported incidence of severe fevers from 0 to 3 months. In the top panel, we report means for children aged > 6 months at the time of the tornado. There should be no impacts of the tornado on outcomes at 3 months for this cohort, since these outcomes were recorded months before the tornado struck. We report group means for infants born in tornado areas v. unaffected areas, and in vitamin A treatment v. placebo (control) villages.

As hypothesized, the results in this first panel show 0 differences in means across these groups.

The mean incidence of severe fevers in all groups is fairly close to 1; differences across infants born in tornado v. unaffected areas are small and statistically insignificant for both vitamin A and placebo groups.

In the second panel, we report the same means but for the cohort of infants aged 0-3 months at the time of the tornado. For these infants, the group means reveal a very different story from the results of the slightly older cohort described above. In the placebo group, infants born in tornado-affected areas had substantially higher incidence of severe fever (1.3 fevers by 3 months postpartum) compared to those born in unaffected areas (0.84 fevers). This mean difference of 0.45 fevers is statistically significant and large – more than half the mean in unaffected areas. In vitamin A treatment villages, this difference is much smaller (0.11).

Below the second panel, we report the difference in differences across cohorts. Consistent with the results above, the difference in differences is large and statistically significant for placebo villages (0.55), while smaller and insignificant for vitamin A treatment villages (0.19). The triple difference (-0.36), reported in the last row of the table, again reflects the same patterns.

4.2 Birth Outcomes

Table 4 reports impacts of the tornado on birth outcomes. 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 (dummy for birth weight < 2.5 kg). Infants exposed *in utero* were about 8 percentage points more likely to have low birth weight from a baseline of 54 percent among unexposed infants (column 2 results). We observed 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 less than 2kg (from a baseline of 14%).

Column 4 in this table reports impacts on length at birth (cm), another summary measure of newborn health. Again, we find significant negative impacts of the tornado, especially in the second and third trimesters: exposed newborns were 0.43 cm shorter than unexposed newborns. The last two columns (columns 5 and 6, respectively) show impacts on prematurity (born before 37 weeks) and gestational age in weeks, respectively. These panels show that the tornado did not appear to have large impacts on length of gestation.

Table 4: Impact of the Tornado on Birth Outcomes

	Birth Weight (kg)	BW < 2.5kg	BW < 2kg	Birth Length (cm)	Premature	GA (weeks)
	Exposure in Utero					
Tornado effect	$-0.07**$	$0.08**$	$0.07***$	$-0.43**$	$0.04*$	-0.08
	(0.04)	(0.04)	(0.03)	(0.21)	(0.03)	(0.24)
Outcome mean	2.44	0.54	0.14	46.5	0.28	37.7
Observations	10,372	10,372	10,372	10,073	14,191	14,191

This table shows the impact of the tornado on birth outcomes. Standard errors are computed using a randomization inference procedure described in the Supplementary Materials.

4.3 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. We estimate these separately by vitamin A treatment status, allowing us to identify the protective effects of vitamin A supplementation at birth. We show results for mid-upper arm circumference (MUAC), an 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 of vitamin A in maintaining epithelial integrity (providing barriers to infection) and supporting a healthy immune system. For example, Tielsch et al. (2007) find that supplementation with vitamin A reduces the case-fatality rate of fevers and diarrhea.

Table 5 reports tornado impacts on two key outcomes – mid-upper arm circumference (MUAC) and the incidence of severe fevers – at 3 and 6 months, for both *in utero* and early life (0-3 month and 4-6 month) exposure, by vitamin A (treatment) and placebo (control) groups. In Panel A, we report results for *in utero* exposure. Overall, there do not appear to be statistically significant impacts of the tornado on MUAC or fever episodes at 3 or 6 months, suggesting the tornado did not have substantial impacts on either group (vitamin A or placebo) for fetal exposure.[4] Panel B shows impacts for 0-3 month tornado exposure. Here, the pattern is clear: for both 3 and 6 month outcomes, the tornado had large deleterious effects on infant health in the control group, but essentially no impacts whatsoever in the vitamin A group. The difference across these two

[4]It is worth noting that when exposure is divided by trimester, the results show consistently that second trimester exposure does seem to generate impacts on early life health, and that vitamin A at birth mitigates this impact. See Table S2 for these results.

Table 5: Impact of Tornado and Vitamin A on 3 and 6 month outcomes

	Outcomes assessed at 3 months		Outcomes assessed at 6 months	
	Mid-Upper Arm Circumference (cm)	Fever Episodes In Months 0-3	Mid-Upper Arm Circumference (cm)	Fever Episodes In Months 4-6
	Panel A: Exposure in Utero			
Tornado Impact:				
By Treatment Group:				
Vitamin A	0.04	0.22	0.11	0.15
	(0.13)	(0.15)	(0.11)	(0.12)
Control	−0.04	0.21	−0.01	0.06
	(0.13)	(0.15)	(0.12)	(0.12)
Difference	0.09	0.01	0.12	0.09
	(0.16)	(0.16)	(0.16)	(0.17)
	Panel B: Exposure in Age 0-3 Months			
Tornado Impact:				
By Treatment Group:				
Vitamin A	0.05	0.08	0.12	0.13
	(0.14)	(0.16)	(0.15)	(0.18)
Control	−0.35 ∗ ∗	0.51 ∗ ∗∗	−0.37 ∗ ∗∗	0.44 ∗ ∗∗
	(0.13)	(0.16)	(0.14)	(0.19)
Difference	0.40∗	−0.43 ∗ ∗	0.49 ∗ ∗	−0.31
	(0.21)	(0.20)	(0.21)	(0.21)
	Panel C: Exposure in Age 4-6 Months			
Tornado Impact:				
By Treatment Group:				
Vitamin A			−0.07	0.22
			(0.15)	(0.14)
Control			−0.08	0.22
			(0.16)	(0.14)
Difference			0.02	−0.00
			(0.20)	(0.20)
Outcome mean	12.2	0.87	13.0	0.92
Observations	16,636	13,321	16,370	13,211

This table shows the impact of the tornado on 3 and 6 month outcomes by vitamin A treatment status. Standard errors are computed using a randomization inference procedure described in the Supplementary Materials.

groups is, in general, statistically different from 0 (with the exception of 4-6 month fever). In Panel C, we report analogous estimates for 4-6 month tornado exposure, again showing little impact of the tornado in both experimental groups.

The takeaway from Table 5 is that tornado exposure does not always generate negative effects, but exposure during the sensitive early period between 0 and 3 months of life had substantial impacts on infant health. Impacts are quite large – e.g., 0.35-0.37 cm on MUAC, which also translates to approximately 0.4 SD[5], and between 0.4 and 0.5 additional fevers on a mean of just less than 1 severe fever episode within each three-month measurement period. Moreover, these effects were all but mitigated by vitamin A supplementation at birth: in the vitamin A group, we see no such tornado exposure effects at 3 and 6 months.

In Tables S3 and S4, we report results on MUAC and severe fever incidence by gender. The main conclusion from these results is that 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. 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. 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).

We perform a variety of checks for potential concerns related to on internal validity with results reported in the A1.5, including discussions of attrition (section A1.5.1), dosing timing (section A1.5.2), changing the definition of the control group (section A1.5.3), and the changing the definition of tornado exposure (section A1.5.4). Our results are generally robust to these potential concerns.

5 Discussion

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

[5]The MUAC measures reported here are in centimeters. The standard deviation of these measures are 1.04-1.06 cm, so an approximate impact in standard deviations can also be read from the figure.

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 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 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. 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 important.

Our results suggest that much of the impact of supplementation, at least on infant mortality, 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 – natural disasters,

disease outbreaks, war, and the like – in the first few months of life.

References

Adhvaryu, Achyuta, Teresa Molina, Anant Nyshadham, and Jorge Tamayo, "Helping Children Catch Up: Early Life Shocks and the Progresa Experiment," 2015.

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," Technical Report, National Bureau of Economic Research 2017.

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.

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.

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.

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.

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.

_ , Thomas Gillespie, Samuel Preston, Bondan Sikoki, and Duncan Thomas, "Mortality, the family and the Indian Ocean tsunami," *The Economic Journal*, 2011, *121* (554), F162–F182.

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.

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.

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.

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.

Jørgensen, Mathias J, Ane B Fisker, Erliyani Sartono, Andreas Andersen, Christian Erikstrup, Ida M Lisse, Maria Yazdanbakhsh, Peter Aaby, and Christine S Benn, "The effect of at-birth vitamin A supplementation on differential leucocyte counts and in vitro cytokine production: an immunological study nested within a randomised trial in Guinea-Bissau," *British Journal of Nutrition*, 2013, *109* (03), 467–477.

Jr, Alfred Sommer Keith P. West, *Vitamin A Deficiency: Health, Survival, and Vision*, Oxford: Oxford University Press, 1996.

Karbownik, Krzysztof and Anthony Wray, "Long-run consequences of exposure to natural disasters," 2016.

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.

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.

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.

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, "Are Different Early Investments Complements or Substitutes? Long-Run and Intergenerational Evidence from Denmark," 2015.

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, 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.

A1: Appendix

A1.1: Figures

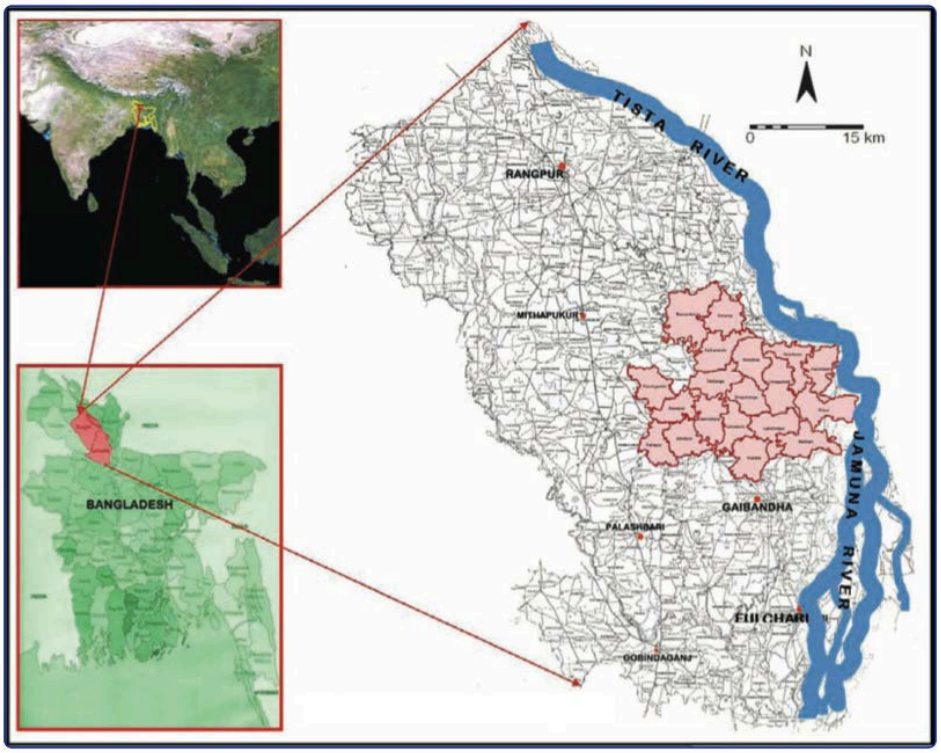

Figure A1: Location of the study area. The figure was produced by the JiVitA GIS Unit.

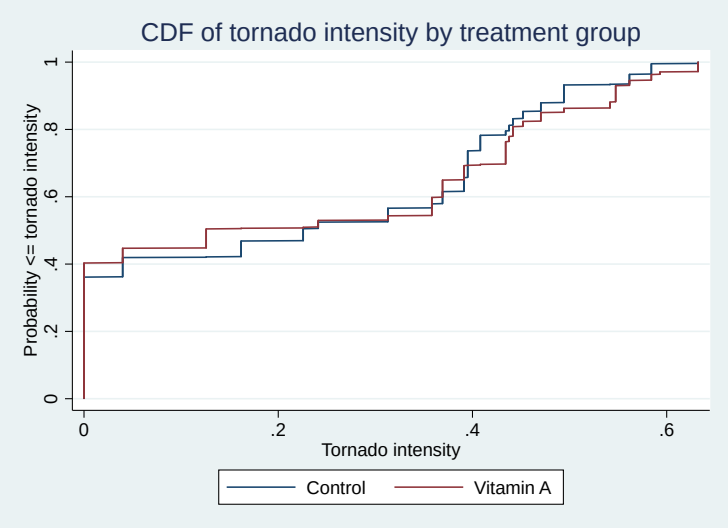

Figure A2: A CDF of the intensity of tornado damage by vitamin A treatment group.

A1.2: Tables

Table S1: Summary Statistics of Infants in the Pre-tornado Cohorts

	All N = 5306		Tornado N = 350		Non-tornado N = 4956		Difference		Within Tornado Area: Vitamin A N = 173		Placebo N = 177		Difference	
	Mean	SD	Mean	SD	Mean	SD	Mean	SE	Mean	SD	Mean	SD	Mean	SE
Infant birth anthropometry														
Weight (kg)	2.49	0.44	2.49	0.44	2.49	0.44	0.00	0.04	2.48	0.42	2.50	0.45	−0.02	0.07
Height (cm)	46.64	2.39	46.53	2.32	46.65	2.39	−0.12	0.22	46.52	2.20	46.54	2.46	−0.02	0.38
MUAC (cm)	9.43	0.86	9.47	0.88	9.42	0.86	0.05	0.09	9.44	0.84	9.51	0.92	−0.06	0.13
Head Circumference (cm)	32.65	1.64	32.65	1.77	32.65	1.62	−0.01	0.13	32.63	1.64	32.66	1.91	−0.03	0.23
Chest Circumference (cm)	30.70	2.14	30.70	2.09	30.70	2.14	0.00	0.21	30.62	1.87	30.78	2.31	−0.16	0.29
Infant anthropometry at 3 months														
MUAC (cm)	12.37	1.07	12.40	1.10	12.37	1.07	0.04	0.13	12.34	1.03	12.46	1.17	−0.13	0.13
Head Circ. (cm)	38.70	1.49	38.64	1.52	38.70	1.49	−0.07	0.12	38.51	1.36	38.76	1.65	−0.25	0.19
Chest Circumference (cm)	38.87	2.24	38.98	2.27	38.86	2.24	0.12	0.20	38.71	2.18	39.25	2.34	−0.55	0.25**
Anthropometric Index	0.17	0.98	0.18	1.02	0.17	0.98	0.01	0.09	0.08	0.92	0.28	1.10	−0.20	0.11*
Infant anthropometry at 6 months														
MUAC	13.08	1.05	13.15	1.07	13.08	1.05	0.07	0.10	13.16	1.07	13.14	1.07	0.02	0.12
Head Circumference (cm)	40.88	1.42	40.97	1.38	40.87	1.43	0.10	0.09	40.87	1.33	41.06	1.43	−0.20	0.16
Chest Circumference (cm)	41.32	2.13	41.47	2.15	41.30	2.13	0.17	0.20	41.21	2.14	41.70	2.14	−0.49	0.24**
Anthropometric Index	0.04	0.99	0.12	1.01	0.03	0.98	0.08	0.07	0.05	1.01	0.17	1.02	−0.12	0.10
Other infant outcomes														
Gender is Male	0.51	0.50	0.54	0.50	0.51	0.50	0.03	0.02	0.51	0.50	0.56	0.50	−0.06	0.04
Fever Incidence, 0-3 months	0.59	0.49	0.57	0.50	0.59	0.49	−0.01	0.06	0.56	0.50	0.58	0.49	−0.02	0.06
Fever Incidence 0-6 months	0.55	0.50	0.59	0.49	0.55	0.50	0.04	0.05	0.62	0.49	0.57	0.50	0.05	0.04
Mortality 0-24 weeks	0.06	0.23	0.05	0.22	0.06	0.24	−0.01	0.01	0.06	0.25	0.04	0.20	0.02	0.03
Maternal characteristics														
Parity	1.33	2.40	1.25	1.50	1.34	2.46	−0.09	0.17	1.43	1.67	1.07	1.29	0.36	0.16**
LSI	−0.04	0.99	−0.10	0.95	−0.04	0.99	−0.06	0.09	−0.14	0.96	−0.05	0.95	−0.09	0.11
Height (cm)	149.32	5.15	149.08	5.20	149.34	5.14	−0.26	0.40	148.54	5.06	149.62	5.29	−1.07	0.44**
MUAC (cm)	22.65	1.93	22.66	1.97	22.65	1.93	0.00	0.15	22.56	2.07	22.75	1.87	−0.20	0.24
Education (years)	3.61	4.03	3.59	3.97	3.61	4.03	−0.01	0.33	3.75	4.23	3.45	3.69	0.30	0.54
Dosing														
Dosed $\leq$ 24 hours	0.52	0.50	0.57	0.50	0.52	0.50	0.05	0.03*	0.58	0.49	0.56	0.50	0.02	0.06
Dosed $\leq$ 7 days	0.59	0.49	0.64	0.48	0.59	0.49	0.05	0.03*	0.68	0.47	0.61	0.49	0.07	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 S2: 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	0.13	0.20	0.17	0.04	0.22*	0.15
	(0.15)	(0.13)	(0.16)	(0.13)	(0.13)	(0.15)
Second trimester	0.18	−0.35**	−0.31	0.21	−0.49***	−0.41**
	(0.18)	(0.16)	(0.21)	(0.15)	(0.16)	(0.18)
Third trimester	0.35*	0.07	−0.02	−0.06	0.30*	0.19
	(0.18)	(0.16)	(0.18)	(0.18)	(0.17)	(0.19)
Age 0-3 months	0.50***	−0.31**	−0.33***	0.45***	−0.31**	−0.35***
	(0.13)	(0.12)	(0.13)	(0.17)	(0.13)	(0.13)
Age 3-6 months				0.23*	−0.04	−0.07
				(0.13)	(0.13)	(0.15)
In tornado area X Vitamin A X ...						
First trimester	−0.01	−0.39**	−0.24	0.17	−0.36*	−0.13
	(0.20)	(0.19)	(0.21)	(0.20)	(0.19)	(0.20)
Second trimester	0.16	0.65***	0.52*	0.22	0.70***	0.55**
	(0.24)	(0.23)	(0.27)	(0.22)	(0.24)	(0.26)
Third trimester	−0.27	−0.12	−0.01	0.08	−0.22	−0.01
	(0.26)	(0.22)	(0.25)	(0.25)	(0.25)	(0.27)
Age 0-3 months	−0.47**	0.31*	0.38*	−0.26	0.40**	0.48**
	(0.18)	(0.18)	(0.20)	(0.20)	(0.19)	(0.20)
Age 3-6 months				0.05	−0.02	0.02
				(0.20)	(0.19)	(0.20)
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 for tornado exposure as described in the text. Standard errors are computing using randomization inference as described in the text.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table S3: Impacts by gender at 3 months

	Anthropometry				Fever	
	Males		Females		Males	Females
	AI	MUAC	AI	MUAC		
In tornado area X ...						
First trimester	0.14	0.09	0.22	0.20	0.26	−0.04
	(0.20)	(0.23)	(0.18)	(0.19)	(0.19)	(0.20)
Second trimester	−0.28	−0.39	−0.34∗	−0.15	0.39	−0.05
	(0.22)	(0.25)	(0.20)	(0.25)	(0.26)	(0.23)
Third trimester	0.07	−0.15	0.01	0.09	0.31	0.37
	(0.20)	(0.22)	(0.20)	(0.25)	(0.26)	(0.25)
Age 0-3 months	−0.51 ∗ ∗∗	−0.58 ∗ ∗∗	−0.03	−0.02	0.85 ∗ ∗∗	0.14
	(0.20)	(0.19)	(0.16)	(0.19)	(0.20)	(0.17)
In tornado area X Vitamin A X ...						
First trimester	−0.24	−0.04	−0.40	−0.34	0.05	−0.00
	(0.27)	(0.30)	(0.25)	(0.27)	(0.25)	(0.31)
Second trimester	0.84 ∗ ∗∗	0.89 ∗ ∗∗	0.29	−0.01	−0.35	0.73 ∗ ∗
	(0.31)	(0.34)	(0.30)	(0.36)	(0.34)	(0.35)
Third trimester	−0.24	0.02	0.13	0.03	−0.06	−0.47
	(0.31)	(0.34)	(0.31)	(0.35)	(0.37)	(0.36)
Age 0-3 months	0.62 ∗ ∗∗	0.73 ∗ ∗∗	0.02	0.03	−0.71 ∗ ∗	−0.20
	(0.24)	(0.27)	(0.24)	(0.27)	(0.28)	(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 **??**. Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table S4: Impacts by gender at 6 months

	Anthropometry				Fever	
	Males		Females		Males	Females
	AI	MUAC	AI	MUAC		
In tornado area X ...						
First trimester	0.13	0.09	0.24	0.15	0.14	−0.09
	(0.20)	(0.21)	(0.19)	(0.21)	(0.20)	(0.19)
Second trimester	−0.41∗	−0.46∗	−0.48 ∗ ∗	−0.31	0.36∗	0.08
	(0.22)	(0.24)	(0.21)	(0.25)	(0.21)	(0.22)
Third trimester	0.23	0.13	0.28	0.21	−0.41	0.34
	(0.23)	(0.25)	(0.22)	(0.26)	(0.26)	(0.25)
Age 0-3 months	−0.47 ∗ ∗∗	−0.55 ∗ ∗∗	−0.10	−0.10	0.53 ∗ ∗	0.39 ∗ ∗
	(0.16)	(0.16)	(0.17)	(0.20)	(0.24)	(0.19)
Age 3-6 months	−0.04	−0.13	−0.04	0.01	0.30	0.16
	(0.20)	(0.20)	(0.18)	(0.22)	(0.19)	(0.17)
In tornado area X Vitamin A X ...						
First trimester	−0.24	−0.01	−0.28	−0.12	0.12	0.31
	(0.26)	(0.29)	(0.27)	(0.28)	(0.30)	(0.30)
Second trimester	0.64 ∗ ∗	0.63∗	0.62 ∗ ∗	0.38	−0.04	0.45
	(0.31)	(0.33)	(0.30)	(0.35)	(0.29)	(0.32)
Third trimester	−0.44	−0.24	0.10	0.25	0.50	−0.40
	(0.35)	(0.36)	(0.32)	(0.36)	(0.37)	(0.35)
Age 0-3 months	0.38	0.51 ∗ ∗	0.38	0.40	−0.43	−0.09
	(0.24)	(0.26)	(0.26)	(0.28)	(0.31)	(0.26)
Age 3-6 months	−0.28	−0.15	0.14	0.11	−0.17	0.32
	(0.26)	(0.26)	(0.25)	(0.27)	(0.28)	(0.25)
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 **??**. Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table S5: Impacts on miscarriage and stillbirth

	Miscarriage	Abortion	Live birth
	Panel B: By trimesters		
In Tornado Area X First Trimester	0.01	-0.03	-0.00
	(0.02)	(0.02)	(0.03)
In Tornado Area X Second Trimester	-0.01	-0.01	0.04
	(0.01)	(0.01)	(0.03)
In Tornado Area X Third Trimester	0.02	-0.01	0.05***
	(0.01)	(0.01)	(0.02)
Dependent variable mean	0.11	0.16	0.69
Observations	25842	25842	25842

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 S6: 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	−0.01	−0.02	0.00	0.01
	(0.06)	(0.07)	(0.07)	(0.07)
Second trimester	0.05	0.05	0.09	0.09
	(0.05)	(0.05)	(0.06)	(0.06)
Third trimester	0.07	0.08	0.10	0.12
	(0.05)	(0.05)	(0.08)	(0.08)
Age 0-3 months	−0.00	−0.01	0.00	−0.01
	(0.04)	(0.04)	(0.07)	(0.07)
Age 3-6 months			0.00	0.01
			(0.05)	(0.05)
In tornado area X Vitamin A X ...				
First trimester	0.07	0.06	0.06	0.04
	(0.07)	(0.07)	(0.07)	(0.07)
Second trimester	−0.00	0.01	−0.08	−0.09
	(0.07)	(0.08)	(0.08)	(0.08)
Third trimester	−0.02	−0.06	−0.11	−0.15
	(0.07)	(0.07)	(0.09)	(0.09)
Age 0-3 months	−0.05	−0.05	−0.05	−0.07
	(0.06)	(0.06)	(0.07)	(0.07)
Age 3-6 months			−0.08	−0.11
			(0.07)	(0.08)
Dependent variable mean	0.11	0.13	0.13	0.14
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. Standard errors are computing using randomization inference as described in the text.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

Table S7: Timing of dosing relative to birth

	Dosed at <= 24 hours	Dosed at <= 7 days
In tornado area X ...		
In utero	−0.03	−0.03
	(0.03)	(0.03)
Age 0-3 months	0.07	0.00
	(0.05)	(0.04)
Age 4-6 months	0.02	0.00
	(0.04)	(0.04)
Dependent variable mean	0.67	0.76
Observations	19033	19033

Regression models of time at dosing a double difference specification. Standard errors are computing using randomization inference as described in the text.
Significance: * < 0.10; ** < 0.05; *** < 0.01.

A1.3: Randomization Inference Procedure

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. To do this we construct "placebo scenarios" where each scenario involves generating a new vitamin A randomization allocation (according to the original location stratified randomization procedure) and generating a "placebo tornado" – that is, defining a random area somewhere within the study area of a similar size and shape as the original tornado. We implement this by choosing a random house in the study area and a random angle (between 0 and 360 degrees) 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 travelled 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. Technically, 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.

The middle and right panels of Figure S3 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.

A1.4: 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.

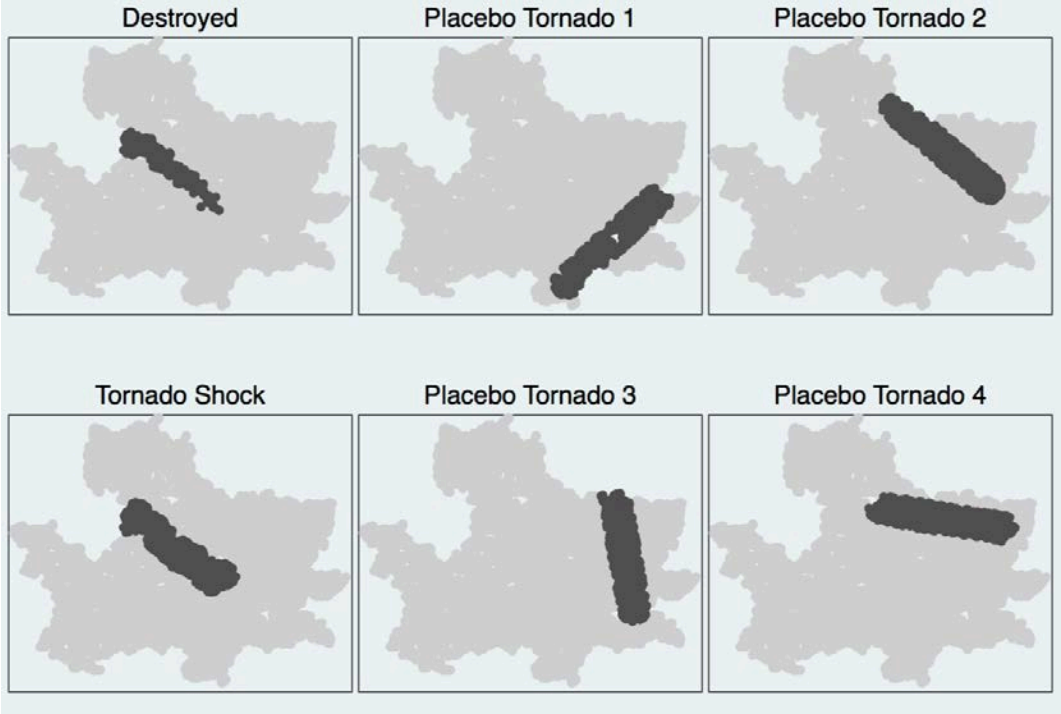

Figure A3: This figure depicts the houses destroyed in the tornado (top left), the definition of tornado exposed (bottom left) and four examples of placebo tornados, each in dark gray (other houses in the study are depicted in light gray).

The results, presented in Figures S4 and S5 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. 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). The corresponding point estimates and errors are reported in Tables S4 and S5.

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).

A1.5: Checks

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

A1.5.1: 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 S6, 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 S6 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). 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.

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 S7. We find overall that attrition of live births is not 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.

A1.5.2: 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 S8 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.