Early-life Shocks, Birth Endowments, and Family Responses

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Abstract

This paper studies how *in utero* exposure to extreme hot temperatures affects parental investments in Colombia. Using a sibling-fixed effects strategy, we show that children who were exposed *in utero* to heat stress during second trimester are more likely to receive necessary vaccines and are breastfed for longer. A variety of evidence is presented in favor of the interpretation that this household behavior reflects a compensatory health response to shifts in children's endowments.

Keywords: Early-life shocks; Health Conditions; Capabilities; Family responses.

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1. Introduction

There is increasing recognition of the importance of early-life conditions in determining socio-economic and health outcomes. It has been documented, for example, that individuals prenatally exposed to maternal stress or diseases such as malaria and influenza have lower cognitive abilities, lower educational attainment and worse health outcomes. (Aizer et al., *Forthcoming*; Almond, 2006; Persson and Rossin-Slater, *Forthcoming*; Venkataramani, 2012). While this body of research suggests strong evidence that prenatal conditions matter, we know very little to date about the role of parents' behavior in shaping these relationships. A better understanding of the direction, magnitude, and scope of parental responses to these shocks would allow to better understand and interpret the broad literature on the persistent impact of early life health deficits. Knowing these relationships can also provide useful information for guiding the targeting of policies intended to remedy inequalities originated in early-life. From an empirical stand point, the question has received little attention, despite the long theoretical debate on how parents' investments respond to shifts in child endowments (Becker and Tomes, 1976; Behrman et al., 1982).

The early studies by Becker and Tomes (1976) and Behrman et al. (1982) provide ambiguous predictions on the direction of these parent behaviors. According to Becker and Tomes (1976), parents are likely to devote less resources in the worse endowed children if poor endowments imply lower returns on investments. Behrman et al. (1982) argue parents make more human capital investments in the less-endowed child if they care about sibling inequality. More recently, Yi et al. (2015) reconcile these two arguments by exploring the implications of multidimensionality of human capital and the cross-productivity of different types of human capital. By doing so, they show that parents are likely to compensate an early health shock by making more health investments, but reinforce the health shock in terms of education investments. Using Colombian data, we document evidence highly consistent with this framework. We do so by investigating how health investments respond to prenatal high temperatures, an environmental shock that is hazardous for health and that has been shown to increase the incidence of health deficits at birth. The focus on prenatal heat waves is particularly compelling to understand the importance of parental responses because temperature shocks are

unable to cause disruption of physical infrastructure, limiting the scope of other different mechanisms such as changes in the supply of health services.

A pregnant woman is especially susceptible to high temperatures due to the additional physical strain and the reduced capacity to lose heat by sweating (Strand et al., 2011; Wells and Cole, 2002). Medical literature generally cites maternal stress and reduced placental growth as potential mechanisms by which exposure to extremely high temperatures during pregnancy results in poorer health at birth. Randomized experiments with animals are consistent with this view, showing that exogenous exposure to high temperatures *in utero* negatively affects health of offspring (Shiota and Kayamura, 1989; Strong et al., 2015). While such experiments are unavailable in humans, a set of studies also documents that high temperatures *in utero* has adverse consequences on fetal health (Deschenes et al., 2009; Strand et al., 2011). Recent work has investigated the long-term consequences of these weather shocks (Adhvaryu et al., 2015; Isen et al., 2015), finding that prenatal heat stress is associated with lower income and increased risk of poor mental health. However, these results represent reduced-form estimates and the role played by family investments remain unclear.

Understanding the impacts of exposure to heat shocks is of particular interest for policy in view of projections indicating that extreme temperature episodes will increase in the next decades (IPCC, 2007). A growing body of recent work has tried to quantify the effects of such climatic shocks on several dimensions, including health and income (Dell et al., 2012; Deschênes and Moretti, 2009). However, most of studies of this literature focus on the short-term impacts of extreme temperatures. Remarkably, the pathways on how weather events could have long-term impacts are not well-studied. Understanding the parental responses to shifts in endowments induced by prenatal extreme hot temperatures would provide important insights.

Our identification strategy exploits plausibly exogenous variation in temperature over time within municipalities. We construct a municipality-by-month weather dataset, which then is combined with microdata by using date and place of birth to identify the prevailing temperature conditions during pregnancy. The empirical approach then compares health investments on children who were prenatally exposed to extreme hot temperatures relative to those who experienced less extreme temperature conditions *in utero*. Since the occurrence of a temperature shock at a given moment in time and place is unpredictable, prenatal exposure to heat waves can be considered as

good as randomly assigned. In addition, we can control for sibling-fixed effects to address the issue that different types of families may change their fertility decisions based on temperature around the time of conception (Barreca et al., 2015). This research design is particularly suitable for the Colombian context. As hydro-meteorological patterns are affected by a recurrent climatic event, temperature records in Colombia vary widely year to year across municipalities. Furthermore, since agricultural production account for a low share of Colombia GDP, the potential for general equilibrium effects is diminished.

Using the sibling-fixed effects strategy, we document that children who were exposed to heat waves while *in utero* are more likely to receive necessary vaccines and are breastfed for longer. These results are not driven by time-series correlation in temperature, selective mortality, migration, or changes in local economic activity. Furthermore, we find that the quantity and spacing of births are not significantly affected. Therefore, we believe that it may be reasonable to attribute the effects to variations in health endowments. This interpretation is made somewhat more plausible by the evidence that prenatal heat stress has adverse consequences on offspring endowments. As such, our findings point out that parental health investments are an unexamined mechanism by which extreme hot temperatures could affect long-term outcomes. Our findings imply that the effects of prenatal heat stress on mental health documented in Adhvaryu et al (2015) are likely to represent a lower bound of biological effects.

Our study complements the recent contribution by Adhvaryu and Nyshadham (2014), who find that children with higher exposure to an iodine supplementation program during pregnancy received more health investments in Tanzania. These results therefore indicate that parents responded by reinforcing health investments. The major difference between their study and ours is that they were concerned with the effects of an intervention that affected only cognition endowments. In contrast, we focused on an "intervention" that is more likely to affect the health endowment dimension of human capital. These differences in parental responses across different dimensions of human capital are consistent with the theoretical framework developed by Yi et al. (2015), and further confirm that is important to explore different dimensions of initial endowments to better understand investment responses to *in utero* shocks.

This study also contributes to a small but growing body of knowledge on the links between *in-utero* exposure to environmental shocks and human capital investments (Almond et al., 2009;

Parman, 2013; Venkataramani, 2012). While studies in this area typically focus on uncommon and severe historical events, we focus on an environmental shock that is less drastic but occurs with higher frequency. We see our results as a first attempt to show the systematic importance of heat stress *in utero* on parental investments. Furthermore, most of existing historical studies use limited measures of investments and have used a variety of indirect strategies to infer parental responses. For example, Almond, Edlund, and Palme (2009) argue that parents adopt reinforcing strategies because the effect of fetal exposure to the radioactive fallout on cognitive skills was greater in children from poor families. This evidence is compelling, but requires corroboration.

This paper is also related to a number of previous studies that link parental investments to proxy variables for endowment, such as birth weight. This literature is not conclusive.[1] While some studies find evidence for reinforcement (Aizer and Cunha, 2012; Datar et al., 2010), others find that parents respond with compensating behavior (Del Bono et al., 2012). In part, clear stylized facts are not developed due to the endogeneity issues. Prenatal and postnatal unobserved investments could create a correlation between birth endowments and parental investment, even in the absence of a behavior response. In contrast, we use a cleaner identification strategy that allows us to provide important insights on parental responses to *in utero* shocks.

The rest of the paper is structured as follows. In section 2, we provide background information on the relationship between heat stress and offspring outcomes. In sections 3 and 4, we describe our data and empirical strategy, respectively. In section 5, we present our empirical findings, including robustness checks. Section 6 concludes.

2. Background on Heat-Stress and Endowments

Exposure to high temperatures is one of the most stressful events. Medical literature indicates that prenatal heat stress increases mother's levels of cortisol, a hormone that plays a critical role in fetal health (Davis and Sandman, 2010; Wadhwa et al., 1993). An early study by Vaha-Eskeli et al. (1991) investigates the effect of moderate heat stress on levels of cortisol in three groups of women: 1) non-pregnant women, (2) women 13-14 weeks pregnant, and (3) women 36-37 weeks pregnant. Blood samples were taken every 5–10 minutes during a resting period followed by the

[1] See Almond and Mazumder (2013) for an inventory.

heat stress intervention. Although this study uses a relatively small sample, the authors found that exposure to moderate heat stress increased significantly cortisol levels in pregnant women. While this study does not examine offspring outcomes, the documented evidence is still important in view of the growing consensus that prenatal exposure to increased cortisol levels negatively impacts offspring outcomes. Aizer et al. (*forthcoming*), for example, show that prenatal exposure to increased levels of cortisol is associated with worse health conditions.

In addition to maternal stress, there are likely also other physiological mechanisms at play. It has been shown that heat stress affects fetal and placental growth, and in the extreme, hyperthermic conditions can cause intra-uterine growth restriction (Hansen, 2009; Regnault et al., 2002). One less direct mechanism includes the effects of temperature on the mother's disease exposure. Increases in temperature may favor the development of specific mosquitoes capable of carrying malaria (Barreca, 2010). In turn, malaria can affect fetal health through direct transmission from the mother (Menendez and Mayor, 2007; Poespoprodjo et al., 2010), and through restricted nutrient intake and oxygen deprivation associated with anemia (Crimmins and Finch, 2006). Despite this potential pathway, we believe this mechanism is likely to be less prominent in the Colombian context since the incidence of malaria is not high. However, we do not rule out the possibility that it plays a role.

Randomized studies based on animals support the idea that prenatal exposure to hot temperatures has adverse consequences on fetal health. These works generally exogenously expose pregnant animals to high temperatures. Examples include Shiota and Kayamura (1989) who exposed mice to high temperatures during pregnancy and observed retardation in brain growth of offspring. Strong et al (2015) likewise exposed pregnant cows to hot temperatures and found that the offspring of exposed cows were more likely to have a damaged immune system. This established link between *in utero* exposure to extreme hot temperatures and poor offspring outcomes are keys to extrapolate findings based on animal experiments to humans where similar experiments are simply unavailable for a variety of ethical or practical reasons. Much of the evidence in humans comes from epidemiological literature. In general, this literature finds that exposure to higher temperatures is associated with increased risk of prematurity and low birth weight.[2] But these

[2] A full review of the epidemiological literature can be found in Strand, Barnett, and Tong (2011).

studies suffer from problems of endogeneity as they are based on cross-sectional or time series comparisons. Surprisingly, the most convincing evidence comes from recent work in economics. For instance, Deschenes et al. (2009) exploit plausible exogenous variation in temperature within counties in U.S and find that prenatal heat stress during second trimester reduces birth-weight. Other studies using a similar approach find also negative impacts on APGAR scores and prematurity (Andalón et al., 2016).

3. Data

Our analysis is based on children who are under five years of age. Below, we describe the weather, investment, and supplementary data that we will analyze. Investment outcomes data are available for cohorts born 1990–2010. To identify exposure to heat waves during pregnancy, these data are matched to the weather measures based on the date of birth and the mother's municipality of residence. Summary statistics of these data are presented in Table 1.

3.1. *Weather Dataset*

We have built a series for temperature and precipitation using data from the Terrestrial Air Temperature and Terrestrial Precipitation: 1900–2010 Gridded Monthly Time Series, version 3.02, respectively (Matsuura and Willmott, 2012). This dataset provides worldwide estimates for weather conditions at the 0.5 *x* 0.5 degree latitude/longitude grid.[3] Using an interpolation algorithm, Matsuura and Willmott (2012) compute values for each grid node from several nearby weather stations. Since some years did not have weather stations over the entire period, the data for missing years are imputed using a meteorological model. To minimize any potential bias from this measurement error, we focus on the period 1970-2010, as most of the weather stations were established in Colombia from 1970 and onwards. Using a strategy similar Rocha and Soares (2015), we construct a municipality-by-month of weather panel. We begin by computing the centroid for each of the 1,120 municipalities in Colombia. Using the centroid, we located the four closest nodes to build a monthly series of temperature and precipitation as the weighted average of estimates related to these four nodes. We use the inverse of the distance to each node as

[3] 0.5 degree correspond to 56 kilometers.

weights. The mean per municipality per month of temperature in our sample is 21.5 ºC, with a standard deviation of 4.7 ºC.

Using this consolidated dataset, we define a heat wave for a given month as temperature above the 90th percentile of distribution for that calendar month within the municipality. Since we are not comparing municipalities, the "extreme" hot temperature should not be taken in an absolute sense. These are simply extreme high temperature months for each municipality within the given time frame. We also investigate the effects of less severe heat waves by defining heat wave as temperature above 85th, 80th, and 75th percentiles.

Prenatal exposure to heat waves through pregnancy is measured by *first trimester*, *second trimester*, and *third trimester*. If, for example, a child was born on October, then *first trimester* is calculated as the number of extreme high temperature months that occurred in their municipality of birth during the months of February, March and April. Naturally, the s*econd trimester* is computed by the number of extreme high temperature months that occurred in their municipality of birth during the months of May, June, and July; and *third trimester* is computed using these criteria during the months of August, September, and October.

This measure represents the exposure that would have occurred if each pregnancy had lasted exactly nine months. Using this measure, we address the issue that children who have longer gestations are mechanically more likely to be exposed to a heat shock at some point during the pregnancy.[4] Since that we do not have information on conception date in our parental investment dataset, we count exposure backwards from the date of birth. Either counting backwards from time of the birth or counting forwards from the date of conception would not matter for children who had nine months of gestation. However, counting backwards nine months will induce measurement error into the assignment of exposure in the second trimester for premature children, which might attenuate the impacts of heat shocks. To examine this potential issue, we use birth certificate data where gestation length is available to construct measures that count backwards and forwards. The means are quite similar for the two variables and we cannot reject

[4] Our exposure measure is similar in spirit to the source of exogenous variation used by Currie and Rossin-Slater (2013) to estimate the effects of exposure to hurricanes during pregnancy.

equality of means. Given the enormous sample size (approximately 8 million birth records), this suggests that in practice such measurement error is unlikely to introduce important biases.

Figure 1 shows the spatial distribution of the incidence of heat waves. The figure reveals that the incidence of high temperatures varies sharply across municipalities within a given month. Episodes of extreme heat occur, on average, in 10 percent of the Colombian municipalities. Yet, there are periods with pervasive heat waves hitting almost 80 percent of the municipalities as well as periods with no municipality experiencing a heat wave.

3.2.*Main Outcomes*

Our empirical analysis uses the 1995, 2000, 2005 and 2010 waves of the Demographic and Health Survey (DHS) of Colombia, a nationally representative survey of women ages 15 to 49. The DHS contains detailed information on early-life health investments for all children under five. For our analysis, we pooled these DHS waves into one dataset. We restricted the sample to mothers with at least two children given that we used family fixed effects models. We also focus on children who were more than 12 months old at time of the survey. Our basic sample consists of 8,949 children. We use the municipality of residence as a proxy for child's municipality of birth. The use of this variable is likely to introduce measurement error, although it is likely to be small given the low migration rates of infants. In section 5.3.2, we provide evidence consistent with this argument.

The inputs we have examined are breastfeeding and vaccination. Vaccinations such as polio and measles have been shown to be effective in preventing ill health and mortality. Given the limited access to medical treatment in developing countries, vaccinations become an important health inputs. Likewise, breastfeeding plays a central role in nutrition, especially in environments characterized by unsafe drinking water and limited supply of food. A large body of work has documented that breastfeeding is predictive of later cognitive outcomes.[5]

Available measures of vaccination reported consistently across the four waves of the DHS include: polio, DPT (diphtheria, pertussis and tetanus combination), and measles. In Colombia, the recommended vaccination schedule is: polio at two months, four months, and six months;

[5] See, for example, Del Bono et al. (2012).

DPT at two months, four months, and six months; measles at 11 months. Our analysis investigates the effect of prenatal heat on the likelihood of being vaccinated for specific diseases. In terms of breastfeeding, we use a dummy variable that equals to one if the child was breastfed for more than six months. This is the minimum length of breastfeeding recommended by the World Health Organization. In the 2005 DHS, breastfeeding duration is only recorded for the youngest child born to a surveyed mother. Therefore, we exclude children from the 2005 DHS for the breastfeeding analysis.

3.3.*Other Data*

As a complementary analysis, we use the birth certificate microdata for the period 1998-2010 from the Colombian Department of Statistics (DANE). We obtained these administrative data for all the municipalities in Colombia- approximately 8 million birth records. This register provides date of delivery, information on gestation length, weight, and APGAR scores. In Colombia, there are between 400,000 and 700,000 births per year.[6] Using this information, we construct a municipality-by-month of birth data set for the 1998-2010 period. The municipality of reference in this panel is the one in which the mother lives. Our outcomes of interest are rate of low 5 minute APGAR (<8), rate of very low birth-weight (≤1,500 gr.), rate of low birth-weight (≤2,500 gr.), rate of birth via Caesarian section and rate of prematurity (less than 37 weeks of gestation).[7]

For further analysis, we also use microdata from the 2005 demographic census (the most recent available). The Integrated Public Use Microdata Series (IPUMS) provides a one percent sample. Although the census does not collect information on parental investments, we can assess whether selective migration may drive our main results. Finally, we use other data sources for supplementary analysis. To assess whether our main results may be driven by changes in the local economic activity, we collected data on: *i*) municipality-year level information on local

[6] In the birth certificate data we used, there is no unique mother identifier so that subsequent births by the same mother cannot be identified. This precludes the use of the sibling-fixed effects estimator.

[7] APGAR score is a clinical test that is given to the newborn in which five parameters are assessed. The parameters evaluated are muscle tone, respiratory effort, heart rate, reflexes and skin color. The test provides a total score between 0 and 10, where a higher score means healthier.

public revenue and spending collected by the Economics Research Center at Andes University for the period 1993-2010; *ii*) *departmento*-year level data on Gross Domestic Product (GDP) and Agricultural production (available for the period 1990-2010) from the DANE.[8]

3.4. *Variation in Prenatal Heat Stress Within Families*

An important concern about the sibling analysis is that siblings may experience "too similar" prenatal exposure to extreme hot temperatures. This may weaken the within-sibling relation between parental investments and prenatal heat stress. However, Figure 2 reveals that prenatal exposure to heat waves varies widely across children in our sample. The standard deviation in the number of months exposed to hot temperatures during pregnancy is 1.58 (relative to a mean of 0.94). More importantly, mother fixed-effects explain only about 48 percent of the variation in the number of months exposed to heat waves while *in utero*, leaving a fair amount of within-sibling variation. This wide within-sibling variation is the basis of our identification strategy.

4. Empirical Strategy

Equation (1) relates each parental investment, *y*, of the child (*i)* born from mother (*j)* in municipality (*k)* to the three measures of prenatal heat waves discussed above:

$$y_{ijktm} = \alpha + \beta_1(1sttrimester)_{ijktm} + \beta_2(2ndtrimester)_{ijktm} + \beta_3(3rdtrimester)_{ijktm} + \delta' X_{ijktm} + \eta_j + \lambda_t + \mu_m + \xi_{ijktm} \quad (1)$$

where the vector X_{ijktm} includes indicators for child's gender, birth order, age in months, and municipal-rainfall for each trimester. λ_t, and μ_m are fixed effects for year of birth and month of birth, respectively. Because η_j is included on the right-hand side of the equation, it is used only within-sibling variation to identify the parameters β_1 through β_3.

Model (1) essentially uses sibling differences in prenatal exposure to extreme hot temperatures, and the timing of which is plausibly exogenous to identify prenatal heat stress impacts. Thus, our strategy compares parental investments of children prenatally exposed to greater extreme hot temperature months against the parental investments for siblings exposed to less extreme hot

[8] *Departamentos* is a first-order administrative unit similar to U.S States. In Colombia, there are 33 *departamento.*

temperature months. The reason why one of them ended up with a greater exposure and the other one did not can be, for all practical purposes, considered as random. Given the evidence of previous studies that heat stress during pregnancy has negative consequences on initial endowments, positives values for β_1, β_2, and β_3 are interpreted as compensatory parental responses and negative values are interpreted as reinforcing responses.

Using this empirical approach, we are able to identify the causal impact of prenatal extreme hot temperatures on parental responses. A potential problem pervading our analysis is the one related to recent evidence that parents may be changing fertility decisions based on temperature around the time of conception. Barreca et al. (2015) convincingly show that parents are likely to postpone conception by one month in response to additional extreme high temperature. To the extent that this is important and that these parents may differ in ways that could affect parental inputs, between-family estimates of the effect of heat stress during first trimester may be biased. Our approach deals with this issue as it relies on within-family comparisons, thereby controlling any time-invariant family qualities. The use of this strategy would be biased if the specific-shift in the timing of conception is directly related to future family postnatal investments. There is no reason to believe that this is plausible. More generally, one could be concerned if there are other unobserved changes within a family that would lead also to postpone conception. However, such specific unobserved changes within family would bias our approach only if they are correlated with the occurrence of temperature shocks. This is highly unlikely since the exact timing of a heat shock is exogenous. Thus, any differences we observe in terms of health investments between siblings can be plausibly attributed to prenatal heat exposure. We discuss other potential issues related to our empirical approach and interpretation of results below.

5. Results

5.1. *Main Results*

Our main results are presented in Table 2. Columns (1)-(4) look at vaccination during infancy. We use dummy variables indicating whether the child has the recommended vaccination doses for specific diseases. Column (4) uses a dummy variable that equals one if the child has all recommended vaccination doses. The results for breastfeeding are presented in columns (5).

The results from (1)-(4) show that exposure to heat waves during second trimester significantly increases the likelihood of being vaccinated. The magnitudes of the effects vary depending on the vaccination. One additional month of exposure to heat waves during the second trimester increases the probability of receiving the recommended vaccination schedule for polio, DPT, and measles by 2, 3, and 1.5 percentage points, respectively. In column (5), we find that exposure to prenatal heat stress during second trimester is significantly associated with increases in probability of having been breastfed for more than six months (point estimate of 0.03).

Overall, the evidence suggests that prenatal heat stress increases health investments. One way to assess the size of the effects is to compare them to the impacts of early-life interventions. For example, Attanasio et al. (2005) show that *Familias en Accion* (FA), a conditional cash transfer program in Colombia, increases the probability of DPT vaccination by 9 percentage points. This shows that the estimated effect of second trimester exposure on DPT vaccination is one third of the effect of the FA program. In other words, the child from a mother who was exposed to three extreme high temperature months during the second trimester would have a similar probability of receiving DPT vaccination as a child of a mother enrolled in the FA program.

5.2.*Heterogeneity by severity of intensity*

Our baseline specifications estimate the impacts of prenatal exposure to very extreme hot temperatures. A natural extension is to assess the presence of heterogeneous effects with respect to the severity of the shock. Table 3 explores this question by using measures of prenatal exposure that define heat waves as temperature above 85^{th}, 80^{th}, and 75^{th} of distribution. In general, we find in fact that exposure to less extreme hot temperatures has significant smaller effects. For example, the marginal effect of exposure during second trimester ranges on the likelihood of total vaccination ranges from 3.5 percentage points in the baseline estimate to 1.6 percentage points in specification that uses the least extreme measure of heat wave. This analysis highlights the usefulness of an intensity-specific analysis when assessing the effects of environmental shocks *in utero*.

5.3. *Potential Mechanisms and Robustness Checks*

Next, we explore potential mechanism by which maternal heat stress affects parental investments. The results from this section suggest that variations in child endowments is a plausible explanation to our results. While the evidence is supportive of this idea, alternative interpretations may be also consistent with the patterns in parental investments. As we shall see, such alternative hypothesis have little empirical support.

5.3.1 Heat stress and child endowments

Studies based largely on animal experiments suggest that exogenous exposure to heat stress *in utero* negatively affects offspring endowments. This suggests that variations in birth endowments could be an important mediator between maternal heat stress and parental investments. While there is extensive literature documenting that prenatal heat stress has adverse consequences on fetal health (see, for example, Deschenes et al. 2009), we also test for the relationship using birth certificate data for the period 1998-2010. The results are presented in Table 4. We weight observations by the number of births per month in the municipality.

Panel A uses a specification that adjusts only for the baby's sex, for municipality-rainfall in each trimester and for fixed effects for municipality of residence at birth, year of birth and month of birth. We find a positive and significant effect of prenatal exposure to heat stress during first trimester on very low birth weight. Increasing the number of extreme high temperature months by 1 increases the probability of very low birth weight by 0.009 percentage points (*P*-value=0.052). Compared to the mean of 0.8 percent, the effect is 1.1 percent. We also find that prenatal heat stress during second trimester has a positive and large effect on the likelihood of having a low APGAR score. One additional month exposed during second trimester increases the incidence of low 5 minute APGAR by 0.98 percentage points. Relative to the mean low 5 minute APGAR rate of 2.3 percent, the effect is substantial at 40 percent. Almond et al. (2005) show that 5 minute APGAR may be a more reliable measure of fetal health than birth weight.

The remaining columns examine the effect on other proxies for fetal health. In column (4) we show that an additional heat wave in the second trimester increases the probability of having a caesarian section by 0.16 percent points (*p*-value =0.04). We consider that this variable

potentially reflects the presence of problems at birth, which may be correlated with an increased risk of poor infant health. The specific timing of the effect is consistent with Currie and Rossin-Slater (2013) who find that exposure to stress induced by an extreme weather event during the second trimester increases the probability of having a caesarian section in U.S. In column (5) we also find that exposure to prenatal heat in the third trimester leads to preterm birth.

Panel B corresponds to specifications that include maternal characteristics as control variables. The estimated coefficients are insensitive to adding such additional variables. In general, the estimates are significant and imply that prenatal exposure to heat waves is associated with poorer health at birth. Furthermore, in many cases, their precision improves. This provides reassuring evidence on the validity of the empirical approach.

Panel C presents the results of a natural falsification test: we repeat the baseline analysis, but also include exposure to heat waves during the first trimester after birth as an independent variable. To the extent that our empirical approach captures the effect of prenatal heat waves, and not the influence of unobserved factors, future heat waves should not predict current birth outcomes. The results indicate that our baseline estimates are robust to including this additional control variable.

Overall, the findings support the notion that heat waves have adverse consequences on infant health, confirming the evidence from prior studies. It is important to recognize that this analysis may underestimate the impact of heat stress, as we do not use other more direct measures of infant health. For example, Currie and Rossin-Slater (2013) find that prenatal stress induced by extreme weather events has a substantial larger effect on the probability of complications of labor and delivery, and of abnormal conditions such as meconium aspiration syndrome. In view of this evidence and that these variables could be more direct proxies for health at birth, our estimates can be interpreted as lower bounds of the effect of maternal heat stress on infant health.

5.3.2 Migration

Given that we use the municipality of residence as a proxy for child's municipality of birth, a bias could drive our results if municipality-migration is related to extreme hot temperatures. It is hard to argue that this is the case since we have used temporary variations in temperature and they are unable to cause disruption of physical infrastructure (unlike others weather events such as

storms). To asses this more formally, we have examined the 2005 census and analyzed differences in prenatal heat waves between migrant and non-migrant children. In Table 5 we regress prenatal exposure to heat waves on a dummy variable that equals 1 if the child was born in the survey municipality. Consistent with the view that heat waves are unlikely to be related to migration, we find no differences in prenatal exposure to extreme hot temperatures between migrant and non-migrant children.

Yet, the use of municipality of residence as a proxy for municipality of birth most likely introduces a random measurement error that attenuates our estimates. To investigate the magnitude of this potential bias, in Table 6 we estimate the investment regressions based on a sample that includes only mothers with children who were born in the municipality of residence. While in general the estimated coefficients of second trimester exposure are larger in magnitude, they are very similar to those of the baseline estimates. Collectively, these findings are consistent with the presence of a random measurement error and suggest that the resulting attenuation bias in our sample is small.

5.3.3 Selective mortality

As our analysis is based on surviving (and presumably stronger) children, an important concern with the results is selective mortality, either during gestation or in early infancy. While most miscarriage happens in the first trimester, there is possibility of late miscarriage and stillbirth. If heat exposure in the second trimester affects this culling process, any estimated impacts after birth would need to be a combination of selection and a direct treatment effect. Similarly, if mortality during early infancy is affected by prenatal temperature exposure, then any impacts on health investments will again be a combination of direct impacts and selection. Kudamatsu et al. (2012) show that infants are more likely to die when exposed *in utero* to higher temperatures, which is consistent with the presence of this selection bias. In our setting, this type of bias is particularly relevant. Suppose that parents make in reality less health investments in children who were exposed to high temperatures *in utero*. Suppose also that prenatal heat stress leads to increases in mortality rates during early infancy so that surviving children have better health endowments. Ignoring this selection bias would lead us to conclude erroneously that parents making compensating health investments.

We address this mortality selection in two ways. First, we exploit the feature of the DHS data that mothers are asked to report information on investments even for children who had died before the interview. Therefore, we can examine the selection issue by simply including these deceased children in our estimation sample. Our second approach to address the mortality selection issue is to calculate bounds by imputing the missing information for infants who did not survive before the investments was possible. Specifically, lower bounds assume that all dead children would have received investments and upper bounds assume exactly the opposite.

The results from these exercise are presented in Table 7. When including deceased children in the sample, the coefficients of interest are almost identical in magnitude relative to the baseline estimates. Although the lower bounds of the coefficient on *second trimester exposure* are imprecise in some cases, the point estimates remain similar to the baseline results. Upper estimates of the effect of *second trimester exposure* are always positive and significant, with point estimates close to those implied by our results in Table 2. Overall, these checks indicate that mortality selection is not driving our main findings.

5.3.4 Subsequent fertility

Some studies argue that child endowments can affect future fertility decisions. This would be a potential channel explaining the link between heat stress, initial endowments and parental investments. The seminal study by Becker and Tomes (1976) suggests ambiguous predictions on the direction of how variations in child endowments may affect future fertility. On the one hand, if less healthy children increase the cost of child quantity, then it would lead to a reduction in fertility. Alternatively, if returns to child quality are lower in less endowed children, then this would increase fertility in response to the higher shadow price of child quality. However, we are unable to find any significant effect of heat waves on fertility. We do this in Table 8 by checking whether prenatal heat stress affects the quantity and spacing of children. The estimates are small and tightly bound around zero, suggesting that having a child who was prenatally exposed to heat waves did not alter subsequent fertility decisions significantly.

5.3.3. Other hypothesis

Perhaps an obvious objection to the interpretation of our results comes from evidence that high temperatures shocks lead to economic downturns (Dell et al., 2012). Therefore, one may argue that the patterns in parental investments are in fact not so much determined by child endowment shifts, but by parents' reactions to reductions in the cost of opportunity, given that the inputs we have used are time intensive. This hypothesis is somewhat consistent with Miller and Urdinola (2010) who show that time-intensive investments are higher during economic downturns. Yet, for this alternative interpretation to make sense, agricultural sector should be an important sector in the Colombian economy. This does not seem to be the case. Indeed, the agricultural value-added only accounts for 11 percent of GDP across the 1990-2010 period, which contrasts with countries like Uganda where agriculture importance ranges from 30 to 60 percent of GDP. Furthermore, the available evidence indicates that higher temperature leads to economic downturns only in poor countries. Even if temperature shock lead to economic downturn, it is hard to think of reasons why this would explain significant effects on parental investments in urban children since such economic shocks would be presumably important in rural areas. In fact, we find little evidence of differential impacts between rural and urban children.[9]

In any case, we can directly investigate this alternative hypothesis by estimating the relationship between heat waves and production, using *departmento*-year level data on GDP and Agricultural production for the period 1990-2010, and municipality-level data on local public finance for years 1993 through 2010. We use data on public finance as proxies for local economic activity. The variable independents of interest are the number of extreme high temperature months that occurred in the *departamento*/municipality in a given year, along with a one-year lag. The results are presented in Table 9, with dependent variables shown in the first column of each row. Considering the discussion above, it is not surprising to find insignificant estimates on these regressions. Moreover, the estimated coefficients are very small in magnitude. We take these results as evidence that our main findings are in fact not driven by changes in local economic activity.

[9] See column (3) from Table A3.

Other alternative hypothesis is that heat waves this month may be correlated with heat waves next months. If so, our estimates may not represent the effects of heat waves while *in utero*. While this is a reasonable hypothesis, we can test it by including in the same regressions the variables of heat exposure after birth. We do this in Table 10. The coefficients associated with heat stress *in utero* during second trimester remain virtually identical, casting doubt on this alternative explanation.

One could argue that was not just child endowment shifts that affect parental investments, but also reductions in time allocated to labor to minimize the potential health impacts of warmer temperatures. This idea is made more plausible in view of evidence from Zivin and Neidell (2014), who find a moderate short-run (within few weeks after the shock) decline in time allocated to labor at high temperature. This alternative hypothesis may explain why parents increase health investments, as vaccination and breastfeeding inputs are made early in life. However, the evidence from Table 2 that exposure during the third trimester does not affect parental investments weights against this alternative interpretation, as one would expected to observe an effect of exposure around the time of birth. Note also that, to the extent that this explanation is important, we should see changes in our estimates when controlling for heat waves after birth. We do not. Although we cannot completely rule out this possibility, the evidence suggests that it is unlikely to be the main mechanism driving the patterns in parental investments.

5.3.4. Further robustness checks

In the appendix A, we conduct additional robustness checks. Table A1 examines the relationship between prenatal heat stress and parental investments by using specifications that control for municipality-specific linear time trends. In general, the use of this more demanding specification produces estimated coefficients of the second trimester exposure that are very similar and that remain statistically significant. In Table A2, we exclude twin children given that prenatal exposure between them does not vary. Our results are broadly similar. Finally, we also assess whether there are heterogeneous effects by child's sex, rural/urban location, and mother's education. Table A3 presents estimates interacting second trimester exposure with these characteristics. We find differential impacts that are statistically significant only in a few cases. For instance, the compensating investment behavior in terms of breastfeeding is more pronounced in boys than girls. Still, there are no significant differences between boys and girls in

vaccinations. We conclude that there is little evidence of a consistent interaction between prenatal heat exposure and these characteristics.

6. Conclusion

In this paper we estimate the impacts of *in utero* exposure to heat waves on parental investments using Colombian data. We find that prenatal exposure to heat waves is associated with more postnatal health investments. We interpret these findings as evidence highly consistent with the model of intrahousehold resource allocation from Yi et al (2015). In particular a plausible interpretation of our findings is that under substitutability between health endowments and investment in health, endowment shifts induced by prenatal heat stress would increase the returns to child health quality. As a result, parents responded by devoting more resources in health. While there are alternative interpretations that could be consistent with the patterns in health investments, the paper is able to show that migration, changes in income, selective mortality, subsequent fertility, time-series correlation in temperature, and other potential concerns do not seem to be influencing the results.

Our findings have implications for the long-term consequences on human capital of prenatal exposure to heat waves. Recent studies indicates that prenatal exposure to heat waves results in increased risk of poor mental health and reduced income (Adhvaryu et al., 2015; Isen et al., 2015). Our results combined with this previous evidence imply that increased health investments in infancy do not remedy the baseline effects of prenatal heat stress. These findings should be taken into account in the cost-benefit analyses of climate change mitigation policies. In particular, interventions that shield pregnant women from the consequences of temporary environmental shocks become a natural policy recommendation.

The findings in this paper also can be used to understand other contexts. Previous studies have documented that individuals prenatally exposed influenza pandemic have increased rates of physical disabilities and are more likely to have health problems such as kidney diseases and diabetes (Almond, 2006; Lin and Liu, 2014). The lack of data on investments has prevented researchers to assess the importance of parental responses to this shock. To the extent that influenza pandemic affected mainly health endowments, our findings suggest that estimates from

these studies represent lower bounds of biological effects of this prenatal shock on health conditions later in life.

The relationship between prenatal heat stress and other human capital investments, such as cognitive and non-cognitive investments, poses an interesting direction for future research. The direction of these investment responses may be different to that documented here if these human capital investments and health endowments are complements in the function production for child quality. Reliable estimates of these parameters are crucial to understand the role of household behavior in determining the long-run effects of prenatal conditions.

Compliance with Ethical Standards:

Conflict of Interest: The authors declare that they have no conflict of interest.

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Table 1. Descriptive statistics, Demography Health Survey (1995, 2000, 2005 and 2010 rounds)

	Mean	Standard deviation
Complete schedule of vaccination (Polio, DPT and Measles doses)	0.60	0.48
Complete schedule of Polio vaccination	0.72	0.44
Complete schedule of DPT vaccination	0.80	0.40
Measles vaccination	0.83	0.37
Breastfed for more than six months	0.68	0.46
Child is male	0.51	0.50
Child's age in months	35.50	1.45
First born	0.21	0.41
Month of birth:		
January	0.08	0.27
February	0.08	0.27
March	0.09	0.28
April	0.08	0.27
May	0.08	0.27
June	0.08	0.27
July	0.09	0.28
August	0.09	0.28
September	0.09	0.29
October	0.09	0.28
November	0.08	0.27
December	0.09	0.28

Note. Survey weights are used.

Table 2. Effects of Heat Waves *In Utero* on Parental Investments

	Health investments				
	Vaccinations				
	Polio	DPT	Measles	Total vaccination	Breastfed for more than six months
	(1)	(2)	(3)	(4)	(5)
1st Trimester	-0.000	-0.005	-0.010	-0.001	0.006
	[0.011]	[0.011]	[0.008]	[0.011]	[0.016]
2nd Trimester	0.022	0.029	0.015	0.035	0.031
	[0.009]**	[0.010]***	[0.009]*	[0.012]***	[0.015]**
3rd Trimester	0.014	-0.003	-0.008	-0.004	0.002
	[0.010]	[0.008]	[0.009]	[0.009]	[0.015]
N	8,949	8,949	8,949	8,949	5,932

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. All regressions are based on the mother-effects fixed estimator, and include as controls dummies for age in months, precipitation in each trimester, dummies for year of birth, dummies for month of birth, and child's sex and birth order. Survey weights are used.

Table 3. Effects of Heat Waves *In Utero* on Parental Investments (Heterogeneity by severity of intensity)

	Temperature above:			
	90th percentile	*85th percentile*	*80th percentile*	*75th percentile*
	(1)	(2)	(3)	(4)
	Panel A: Dependent Variable is Polio vaccination			
2nd Trimester heat exposure	0.022	0.025	0.016	0.019
	[0.009]**	[0.008]***	[0.007]**	[0.007]**
N	8,949	8,949	8,949	8,949
	Panel B: Dependent Variable is DPT vaccination			
2nd Trimester heat exposure	0.029	0.018	0.015	0.010
	[0.010]***	[0.008]**	[0.006]**	[0.005]*
N	8,949	8,949	8,949	8,949
	Panel C: Dependent Variable is Measles vaccination			
2nd Trimester heat exposure	0.015	0.014	0.009	0.005
	[0.009]*	[0.007]**	[0.006]	[0.006]
N	8,949	8,949	8,949	8,949
	Panel D: Dependent Variable is Total vaccinations			
2nd Trimester heat exposure	0.035	0.027	0.019	0.016
	[0.012]***	[0.011]**	[0.009]**	[0.007]**
N	8,949	8,949	8,949	8,949
	Panel E: Dependent Variable is Breastfed for more than six months			
2nd Trimester heat exposure	0.031	0.017	0.009	0.005
	[0.015]**	[0.012]	[0.010]	[0.009]
N	5,932	5,932	5,932	5,932

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. Column (1) produces baseline results. Columns (2) through (4) report estimates by using alternative definitions of prenatal heat waves.

Table 4. Effects of Heat Waves *In Utero* on Birth Outcomes

	Rate of very low birth weight (1)	Rate of low birth weight (2)	Rate of low 5 minute APGAR (3)	Rate of C-Section (4)	Rate of preterm birth (5)
			Panel A: Baseline specification		
1st Trimester in utero	0.0099	0.0004	-0.200	0.1161	0.0036
	[0.0050]**	[0.0258]	[0.441]	[0.1271]	[0.0405]
2nd Trimester in utero	-0.0060	-0.0008	0.9880	0.1662	-0.0776
	[0.0044]	[0.0201]	[0.5770]*	[0.0839]**	[0.0589]
3rd Trimester in utero	0.0010	0.0131	0.3370	0.03762	0.0580
	[0.0046]	[0.0223]	[0.3120]	[0.1140]	[0.0332]*
			Panel B: Controlling for maternal characteristics		
1st Trimester in utero	0.0099	-0.0009	-0.2050	0.1321	0.0044
	[0.0049]**	[0.0255]	[0.4461]	[0.1272]	[0.0401]
2ndTrimester in utero	-0.0061	-0.0024	0.9890	0.1711	-0.0792
	[0.0045]	[0.0196]	[0.5781]*	[0.0836]**	[0.0591]
3rd Trimester in utero	0.0010	0.0117	0.3311	0.0508	0.0585
	[0.0046]	[0.0218]	[0.3091]	[0.1161]	[0.0328]*
			Panel C: Controlling for exposure after birth		
1st Trimester in utero	0.0099	-0.0010	-0.2081	0.1312	0.0041
	[0.0049]**	[0.0254]	[0.4501]	[0.1270]	[0.0402]
2ndTrimester in utero	-0.0061	-0.0023	0.9911	0.1710	-0.0790
	[0.0045]	[0.0197]	[0.5850]*	[0.0833]**	[0.0586]
3rd Trimester in utero	-0.0002	0.0158	0.4170	0.0667	0.0681
	[0.0046]	[0.0225]	[0.4091]	[0.0959]	[0.0325]**
1rd Trimester after birth	0.0040	-0.0126	-0.2621	-0.0482	-0.0291
	[0.0052]	[0.0157]	[0.3782]	[0.0733]	[0.0355]
Mean of dependent variable	0.82	7.31	2.32	30.23	14.15
N	168,692	168,692	167,800	169,113	168,743

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. Panel A is a specification that controls for municipality of birth, year of birth, and month of birth fixed effects, and for precipitation in each trimester, and baby's sex. In addition, Panel B and Panel C include dummy for mother age under 20 years, dummy for mother age over 45 years, dummy for mother's education (some college), dummy for mother's marital status (married). Panel C includes exposure to heat temperatures during first trimester after birth as a control variable. All regressions are weighted by the number of births per month in the municipality.

Table 5. Heat Waves *In Utero* between migrant and non-migrant children

	Number of prenatal heat waves during:		
	1st trimester	2nd trimester	3rd trimester
	(1)	(2)	(3)
Child was born in the survey municipality	0.021	0.001	-0.012
	[0.0158]	[0.014]	[0.016]
N	1,222,311	1,222,311	1,222,311

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. Each coefficient is from a different regression. All regressions are based on the mother-effects fixed estimator, and include controls for precipitation in each trimester, dummies for municipality of birth, dummies for year of birth, dummies for month of birth, and child's sex and age. Survey weights are used.

Table 6. Effects of Heat Waves *In Utero* on Parental Investments (Excluding migrant families)

	Health investments				
	Vaccinations				
	Polio	DPT	Measles	Total Vaccination	Breastfed for more than six months
	(1)	(2)	(3)	(4)	(5)
	Panel A: Baseline estimates				
1st Trimester	-0.000	-0.005	-0.010	0.001	0.006
	[0.011]	[0.011]	[0.008]	[0.013]	[0.016]
2nd Trimester	0.022	0.029	0.015	0.035	0.031
	[0.010]**	[0.010]***	[0.009]*	[0.012]***	[0.015]**
3rd Trimester	0.014	-0.003	-0.008	-0.004	-0.002
	[0.010]	[0.007]	[0.009]	[0.009]	[0.015]
N	8,949	8,949	8,949	8,949	5,932
	Panel B: Excluding migrant families				
1st Trimester	0.000	-0.008	-0.006	0.003	0.010
	[0.012]	[0.011]	[0.008]	[0.013]	[0.018]
2nd Trimester	0.029	0.039	0.014	0.043	0.040
	[0.009]***	[0.010]***	[0.010]	[0.014]***	[0.015]***
3rd Trimester	0.018	-0.009	-0.008	-0.005	-0.008
	[0.012]	[0.009]	[0.011]	[0.011]	[0.014]
N	7,150	7,150	7,150	7,150	4,714

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. Panel A produces baseline results. Panel B presents results based on a sample of children who were born in the municipality of residence. Survey weights are used.

Table 7. Effects of Heat Waves *In Utero* on Parental Investments (Selective mortality)

	Health investments				
	Vaccinations				
	Polio	DPT	Measles	Total vaccination	Breastfed for more than six months
	(1)	(2)	(3)	(4)	(5)
			Panel A: Baseline estimates		
1st Trimester	-0.000	-0.005	-0.010	-0.001	0.006
	[0.011]	[0.011]	[0.008]	[0.011]	[0.016]
2nd Trimester	0.022	0.029	0.015	0.035	0.031
	[0.009]**	[0.010]***	[0.009]*	[0.012]***	[0.015]**
3rd Trimester	0.014	-0.003	-0.008	-0.004	0.002
	[0.010]	[0.008]	[0.009]	[0.009]	[0.015]
N	8,949	8,949	8,949	8,949	5,932
			Panel B: Including deceased infants		
1st Trimester	-0.000	-0.005	-0.010	-0.001	0.014
	[0.011]	[0.011]	[0.007]	[0.011]	[0.016]
2nd Trimester	0.022	0.029	0.015	0.035	0.034
	[0.009]**	[0.010]***	[0.009]*	[0.012]***	[0.014]**
3rd Trimester	0.014	-0.003	-0.008	-0.004	0.003
	[0.010]	[0.007]	[0.009]	[0.009]	[0.014]
N	9,243	9,243	9,243	9,243	6,391
			Panel C: Lower bounds		
1st Trimester	-0.003	-0.009	-0.009	-0.005	0.007
	[0.011]	[0.011]	[0.007]	[0.011]	[0.016]
2nd Trimester	0.0164	0.023	0.012	0.028	0.026
	[0.009]*	[0.010]**	[0.009]	[0.013]**	[0.016]
3rd Trimester	0.010	-0.007	-0.010	-0.008	0.002
	[0.010]	[0.007]	[0.009]	[0.104]	[0.015]
	9,642	9,642	9,642	9,642	
N					6,523
			Panel D: Upper bounds		
1st Trimester	0.005	0.000	0.000	0.003	0.012
	[0.010]	[0.010]	[0.010]	[0.010]	[0.016]
2nd Trimester	0.026	0.033	0.022	0.038	0.038
	[0.009]***	[0.009]***	[0.010]**	[0.012]***	[0.014]***
3rd Trimester	0.016	-0.001	-0.005	-0.002	0.004
	[0.011]	[0.008]	[0.010]	[0.010]	[0.014]
N	9,642	9,642	9,642	9,642	6,523

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. All regressions are based on the mother-effects fixed estimator, and include as controls dummies for age in months, precipitation in each trimester, dummies for year of birth, dummies for month of birth, and child's sex and birth order. Lower bounds assume that all dead children would have received investments and upper bounds assume exactly the opposite. Survey weights are used.

Table 8. Effects of Heat Waves *In Utero* on Quantity and Spacing of Births

	Subsequent births	Succeeding birth interval
	(1)	(2)
1st Trimester	-0.000	-0.600
	[0.004]	[0.366]
2nd Trimester	0.001	0.335
	[0.005]	[0.39]
3rd Trimester	0.002	0.484
	[0.005]	[0.360]
N	8,949	4,923

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. All regressions are based on the mother-effects fixed estimator, and include controls for mother's age at birth, precipitation in each trimester, dummies for year of birth, dummies for month of birth, and child's sex and birth order. Dependent variable in column (1) is a dummy indicating whether the number of subsequent births to a mother is greater than zero. Dependent variable in column (2) is the succeeding birth interval in months. Survey weights are used

Table 9. Relationship between Heat Waves and Economic Activity

	$(\text{Heat waves})_t$		$(\text{Heat waves})_{t-1}$		
	Coefficient Estimate	Standard Error	Coefficient Estimate	Standard Error	N
Agricultural production	-0.001	[0.010]	0.005	[0.008]	640
GDP	0.005	[0.004]	0.004	[0.003]	660
Total Income (Indirect+ direct taxes)	-0.002	[0.004]	-0.003	[0.003]	17,257
Capital Income	-0.005	[0.006]	0.002	[0.006]	16,304
Transfer	0.005	[0.004]	0.000	[0.007]	16,791
Total Spending	-0.001	[0.004]	-0.003	[0.004]	17,316
Investment Spending	-0.004	[0.005]	0.002	[0.007]	11,676
Operational Spending	0.000	[0.002]	-0.007	[0.005]	17,236
Housing Spending	-0.001	[0.017]	-0.010	[0.020]	10,211
Education Spending	-0.008	[0.012]	0.000	[0.010]	11,662
Health Spending	-0.001	[0.007]	0.006	[0.007]	11,648

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the *departamento* level. Coefficients in each row are from a different regression. Regressions for agricultural production and GDP use data at *departamento* level and include *departamento* and year fixed effects, and control for precipitation and *departamento*-specific time trends. Regressions on local public finance use data at municipality level and include municipality and year fixed effects, and controls for precipitation and *departamento*-specific time trends. All dependent variables are in logs.

Table 10. Effects of Heat Waves *In Utero* on Parental Investments (controlling for heat waves after birth)

		Controlling for heat waves:				
	Baseline	*1 trimester after birth*	*2 trimester after birth*	*3 trimester after birth*	*4 trimester after birth*	*5 trimester after birth*
	(1)	(2)	(3)	(4)	(5)	(6)
		Panel A: Dependent Variable is Polio vaccination				
1st Trimester heat exposure	-0.000	-0.000	-0.001	-0.002	-0.002	-0.001
	[0.011]	[0.011]	[0.011]	[0.011]	[0.011]	[0.011]
2nd Trimester heat exposure	0.022	0.022	0.024	0.022	0.022	0.022
	[0.009]**	[0.009]**	[0.009]***	[0.009]**	[0.009]**	[0.009]**
3rd Trimester heat exposure	0.014	0.014	0.014	0.015	0.014	0.014
	[0.010]	[0.011]	[0.011]	[0.011]	[0.011]	[0.011]
		Panel B: Dependent Variable is DPT vaccination				
1st Trimester heat exposure	-0.005	-0.005	-0.005	-0.006	-0.006	-0.006
	[0.011]	[0.011]	[0.011]	[0.011]	[0.011]	[0.011]
2nd Trimester heat exposure	0.029	0.029	0.029	0.028	0.028	0.028
	[0.010]***	[0.010]***	[0.010]***	[0.010]***	[0.010]***	[0.010]***
3rd Trimester heat exposure	-0.003	-0.005	-0.005	-0.005	-0.005	-0.005
	[0.008]	[0.008]	[0.008]	[0.008]	[0.008]	[0.008]
		Panel C: Dependent Variable is Measles vaccination				
1st Trimester heat exposure	-0.010	-0.010	-0.010	-0.011	-0.011	-0.011
	[0.008]	[0.008]	[0.008]	[0.008]	[0.008]	[0.008]
2nd Trimester heat exposure	0.015	0.015	0.016	0.016	0.017	0.016
	[0.009]*	[0.009]*	[0.010]*	[0.010*]	[0.010]*	[0.009]*
3rd Trimester heat exposure	-0.008	-0.009	-0.009	-0.009	-0.008	-0.008
	[0.009]	[0.011]	[0.011]	[0.010]	[0.010]	[0.010]
		Panel D: Dependent Variable is Total vaccinations				
1st Trimester heat exposure	-0.001	-0.002	-0.002	-0.003	-0.003	-0.002
	[0.011]	[0.012]	[0.012]	[0.011]	[0.011]	[0.011]
2nd Trimester heat exposure	0.035	0.035	0.036	0.034	0.034	0.034
	[0.012]***	[0.012]***	[0.012]***	[0.012]***	[0.012]***	[0.012]***
3rd Trimester heat exposure	-0.004	-0.006	-0.006	-0.005	-0.005	-0.005
	[0.009]	[0.010]	[0.010]	[0.010]	[0.010]	[0.010]
		Panel E: Dependent Variable is Breastfed for more than six months				
1st Trimester heat exposure	0.006	0.006	0.006	0.006	0.0055	0.0055
	[0.016]	[0.016]	[0.016]	[0.017]	[0.017]	[0.017]
2nd Trimester heat exposure	0.031	0.031	0.031	0.028	0.028	0.028
	[0.015]**	[0.015]**	[0.014]**	[0.014]*	[0.014]*	[0.014]*
3rd Trimester heat exposure	0.002	-0.000	-0.000	-0.001	-0.002	-0.002
	[0.015]	[0.014]	[0.014]	[0.014]	[0.014]	[0.014]

Notes. Robust standard errors in brackets (***p < 0.01, **p < 0.05, *p < 0.1) are clustered at the municipality level. Column (1) produces baseline results. Columns (2) through (6) report estimates controlling for heat waves after birth.

Figure 1. % of municipalities with temperature over 90th percentile

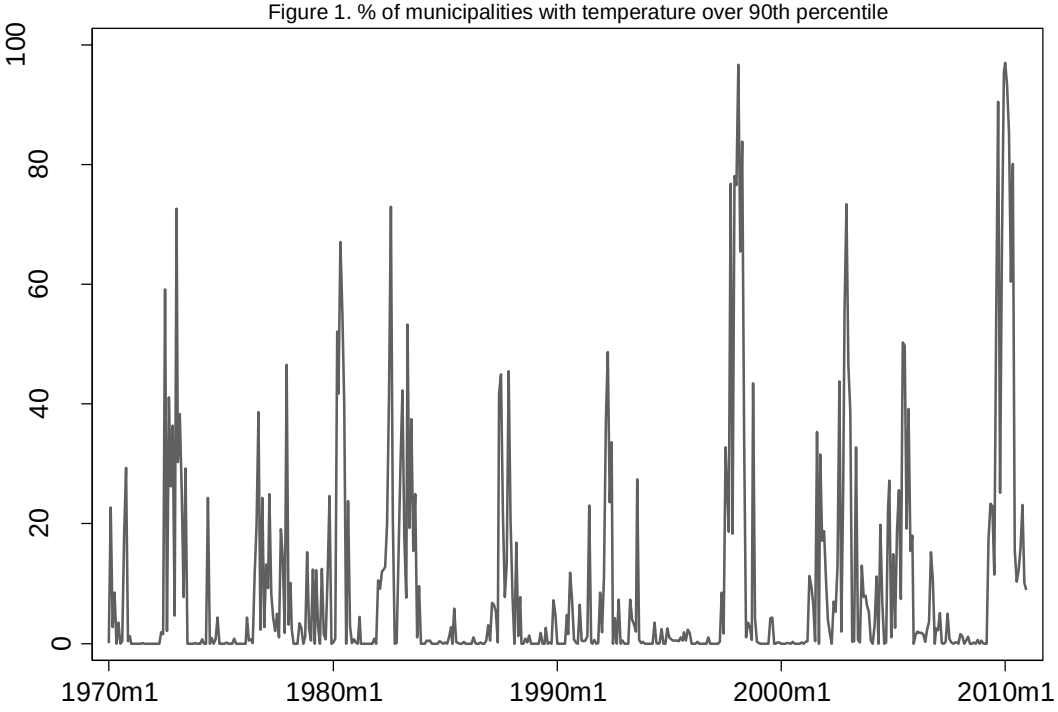

Figure 2. Distribution of in utero exposure to heat waves