Does a mother's exposure to drought in utero increase the resistance of her offspring to in utero shocks?

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Abstract

This paper seeks to determine whether, by being exposed to a drought in utero, a mother can transfer resistance of in utero exposure to drought to her offspring. Results of the difference-in-difference model show that there is a form of resistance transfer from mother to child, with double exposed children having better weight-for age and weight-for height measures than children whose mothers were not exposed in utero. In addition to the above, the paper also shows that by using a difference-in-difference model and identifying two shocks, the effect of in utero drought on child health and the intergenerational transfer of in utero shocks from mother to child can also be estimated.

JEL classification: J13, Q54, C49

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1 Introduction

Over the last 3 decades, we have seen an increase in extreme weather events, such as droughts, floods and hurricanes, across the globe. As a result, we are beginning to witness not only an increase in the number of people that were exposed to a shock in utero, but also an increase the number of those double exposed. These are people who were not only exposed in utero, but also had a mother exposed in utero. Studies have shown that people who experience an adverse shock in utero are more likely to have poorer health in infancy and childhood, and are also more likely to suffer from diabetes and cardiovascular diseases in later life. A more recent set of studies shows that these negative health effects can be transferred from mother to child.

This paper seeks to answer the question whether resistance to an adverse in utero shock can be passed on from mother to offspring. Although studies show that in utero shocks have adverse effects on both the parent generation (F1) and subsequent generation (F2), what is not clear is whether an F2 also experiencing a shock in utero would acquire resistance inherited from the F1. Developing resistance would help the F2 to have better health indicators at birth than an infant who experiences the same shock, but without having the parent transfer the "resistance". This paper also shows that in answering the above question, the separate

strands of the literature, immediate and intergenerational effects of adverse shocks can be combined in the same model and asnwered simultaneously.

Studies on the effect of in utero shocks on child and adult health outcomes take their cue from the Fetal Origins Hypothesis put forward by Barker in his paper, "The fetal and infant origins of adult disease" (Barker, 1990). The hypothesis proposes that, early life conditions of a fetus can have long lasting effects that can be seen in adulthood. Though controversial at the time, the theory has been tested by epidemiologists and economists. Papers such as Nelson (2010), Wells (2003), Bhalotra and Rawlings (2011) and Currie (2008) provide evidence the supports the fetal origins hypothesis. The paper by Almond and Currie (2011) gives a broad and more structured overview of the fetal origins hypothesis and the literature testing this hypothesis.

Related to the Fetal Origins Hypothesis is the Thrifty Phenotype Hypothesis, put forward by Hales and Barker (1992). The Thrifty Phenotype Hypothesis states that the environment the fetus finds itself in provides cues for what type of environment it will find itself in after birth. As a result, if a fetus finds itself in a hostile environment, it will be programmed to thrive in a hostile environment, thus having a "thrifty phenotype". If the fetus survives and finds itself in a resource rich environment, then over time it is likely to suffer from diseases more commonly found in people who are obese/overweight.

Animal studies (Ozanne et al., 2005; Park et al., 2008; Ferland-McCollough et al., 2012) and event studies by economists (Lee, 2009; Li and An, 2015; Kim et al., 2010) have shown that adverse effects of in utero shocks can be passed on from mother to offspring. These results offer strong evidence that having a thrifty phenotype can be passed on from mother to offspring, thus making the offspring also thrifty; better suited to harsh environments in utero and outside. The hypothesis put forward by this paper is that if a fetus has a thrifty mother, then it will have a resistance to an adverse in utero shock similar to the one the mother was exposed to, and therefore we can say that resistance to the shock has been passed from mother to fetus (offspring if it survives to birth).

By being exposed to an adverse shock in utero, the mother has now adapted to surviving in a nutrition deprived environment. The effect upon coming into a resource rich environment would be to have lower health measures and be more likely to fall ill later in life. Due to acquiring a thrifty phenotype and passing it on to her offspring, two things happen. First, the offspring develops a thrifty phenotype, making it resistant to adverse in utero environments. The second, a susceptibility to chronic illnesses in later life. The

second can be easily seen with an intergenerational study such as those by Painter et al. (2008) and Li and An (2015). The first effect cannot be easily seen unless the offspring were to experience an adverse shock in utero. Given that the adverse shocks considered in the literature are short infrequent events, a thrifty offspring is most likely to experience a resource rich environment both in utero and after birth. As a result current methodologies would not be able to observe the transfer of resistance from mother to offspring. But if the offspring is faced with a hostile environment, the transfer of resistance from mother to offspring would lead to the offspring thriving in the hostile environment. What we would see in this case is that, children exposed to an adverse shock in utero, who also have a mother who experienced one in utero, would have better health measures than children whose mother did not experience the same shock in utero. This paper proposes that a logical extension of the thrifty phenotype hypothesis is that resistance to in utero shocks can be passed on from mother to offspring.

This paper uses a difference-in-difference estimator to determine what effect in utero exposure to a drought by both a mother (F1) and her child (F2) would have on the child's health. This paper will show that by using this approach, not only can the question of whether resistance to in utero shocks be transferred from mother to child be answered, but the first and second generation effects of the shock can be estimated as well. The first generation effect being the immediate impact of the shock on the fetus, similar to Almond (2006) and Kumar et al. (2014), whilst the second generation effect is the effect on the next generation (F2) who do not experience the shock in utero as in Painter et al. (2008), Li and An (2015) and Caruso (2015). In short, this paper answers the question of whether resistance to shocks in utero can be passed on from mother to child whilst also combining the separate literature on the effects of adverse shocks in utero to child health and the intergenerational transfer of shocks in utero into a single paper.

Results of the difference-in-difference show that the immediate impact of a drought on a child is to reduce weight-for-age and weight-for-height Z scores by 0.307 and 0.211 standard deviations respectively. If a child and her mother are exposed to a drought in utero, the child will have a higher weight-for-age and weight for-height than an exposed child whose mother was not exposed by 0.347 and 0.271 standard deviations respectively. In short, resistance to the drought in utero is passed on from mother to child and can only be observed if both the mother and child experience a drought in utero. The resistance is enough to wipe out the negative intergenerational effect passed on from mother to child. The results obtained are robust to different estimation techniques and robustness checks. The results show no transfer of resistance for height-for-age in the difference-in-difference model, but a significant positive effect (0.419) is found in the fixed effects model.

This paper adds to the literature in two significant ways. First, to the author's knowledge, this is the first study to look into the effect of multiple in utero shocks across generations to answer the question, whether resistance to adverse in utero environments can be transferred from mother to offspring. The second is to show that the separate strands of the literature, effect of in utero shocks on child and later life outcomes, along with effects of adverse shocks in utero on subsequent generations, can be merged into one model and answered simultaneously.

The rest of the paper is structured as follows, Section 2 provides a review of the existing literature on the first and second generation effects of in utero exposure to shocks. Section 3 gives a brief explainer on the 2 drought periods in Senegal that mothers and their children were exposed to. Section 4 covers the methodology used in this paper. Sections 5 and 6 deal with the descriptive statistics and results obatined respectively. Finally, section 7 concludes the paper.

2 Empirical Literature

There has been more ground covered in the area of in utero shocks regarding its effects on the F1 in, childhood, teenage years and adulthood, than its effect on the offspring (F2). These groups of papers I call the first generation papers due to their focus on a shock's effect on the individual themselves over the course of their lifecycle, as opposed to their offspring. In addition, the first generation papers focus goes beyond the typical health measures such as birth weight, wasting, stunting and occurrence of illness. First generation papers have also looked at the effect of in utero shocks on educational outcomes (Maccini and Yang, 2008; Caruso, 2015), wages (Chen and Zhou, 2007; Cutler et al., 2010), outcomes in the marriage market (Almond et al., 2010), just to name a few. The papers by Almond and Currie (2011) and Currie and Vogl (2012) give an excellent and more detailed review of the literature on in utero shocks and the challenges faced in the estimation of different types of shocks.

The literature on the intergenerational transfer of adverse shocks in utero is relatively limited. This is surprising given that the first paper on intergenerational transfer of adverse shock by Lumey (1992), was over 20 years ago. This was followed by another paper by Stein and Lumey (2000), focusing on the relationship between the birth weights of the maternal and offspring generations of the cohort that experienced the

1944 Dutch famine. After 2000, it seems there was lull in the study of intergenerational transfers, with the literature focusing more on the effect of in utero shocks to child outcomes (Pörtner, 2010; Kumar et al., 2014; Almond et al., 2015; Datar et al., 2013; Rocha and Soares, 2015) and later life outcomes (Almond, 2006; Maccini and Yang, 2008; Boekelheide et al., 2012; Chen and Zhou, 2007). Since 2008, there has been a resurgent interest in understanding the longer term consequences of in utero shocks for the offspring of those who experienced it in utero. The resurgence of this topic can be traced to the paper by Painter et al. (2008) which also focuses on the Dutch famine of 1944.

Since Painter et al. (2008), the studies on the intergenerational transfer of in utero shocks largely focus on 3 events in history; the Influenza pandemic of 1918-19, the Dutch famine of 1944 to 1945 and the China great famine of 1959 to 1961. The paper by Richter and Robling (2013) focuses on the 1918 influenza pandemic which killed an estimated 50 million people out of 500 million infected people world wide (Taubenberger and Morens, 2006). The Influenza pandemic was an exogenous shock that spread quickly across countries and lasted for 2 years. Richter and Robling (2013) look at the effect of the influenza pandemic on the children of women who were in utero during the 1918 influenza pandemic. The results of the study show that the offspring (F2) of the women (F1) who experienced the pandemic in utero experienced higher levels of chronic illnesses during adulthood. The main weakness of this paper (and other papers focusing on the dutch famine) is the possible confounding effect of World War 1, which took place over the same period. The effect of the War was to reduce the pool of able bodied men in the countries taking part in the War and thereby affecting the pool of men left at home that women could marry. Without controlling for this, any effects found could be due to the poorer (gene) quality of the woman's partner. It should be noted that the results above still hold for countries that were neutral during the first World War; Taiwan (Lin and Liu, 2014) and Switzerland (Neelsen and Stratmann, 2012).

The studies focusing on the intergenerational transmission of health shocks for the Dutch famine (Painter et al., 2008; Stein and Lumey, 2000) found similar results to the 1918 influenza pandemic. They show that the effects were also present in childhood, with child health measures such as birth weight lower for those whose parents experienced the Dutch famine in utero. As in the influenza pandemic studies, studies on the Dutch famine are plagued with the same confounding effects issues, in that the period in question (1944 to 1945) coincided with the second World War and thus also potentially had an effect on the gene pool. The parental characteristics at the time of the War were not the same as would be found in normal (non war) times and thus the separate effects of the War and the famine would be difficult to disentangle. In addition

to the above, the famine came about due to a shortage of food, caused by an embargo by Germany, which cannot be described as a random event. As a result, there was a possibility that certain couples choose not to have a baby during this period, leading to a selection bias problem.

The third and more recent set of studies, Kim et al. (2010), Fung and Ha (2009) and Li and An (2015), look at the 1959 to 1961 great famine in China. Li and An (2015), show that the great famine in China has led to the children of those who experienced the famine in utero to be shorter by 0.3 standard deviations, compared to those whose parents did not. The effect they find extends to age 18 for those in their sample, with the greatest impact for children between 8 and 12 years. Results from Li and An (2015) confirm those of Fung and Ha (2009). Although the great China famine did not coincide with any other major shocks which could lead to confounding effects, there is a selection bias issue resulting from the parents' decision to have a child. Li and An (2015), as well as Fung and Ha (2009) show that although fertility decisions were affected by the famine, (with wealthier households choosing to have less births) the strict controls on rural migration meant that the bulk of the rural area, at the time were in the low SES group. Therefore, by focusing the study in the rural areas, in the case of Li and An (2015), fertility decisions would not bias results. The main contribution of the Li and An (2015) paper is to address the issue of paternal influence on the child. Prior studies have omitted the father of the F2 generation focusing only on the mother. Medical studies have shown that the fathers who experience shocks in utero can pass on certain genetic traits to their children. Li and An (2015) argue that omitting the father's attributes can lead to omitted variable bias (OVB). On the other hand, due to assortative mating, the correlation between the characteristics of the mother and father would lead to issues of collinearity. Given that OVB is a bigger problem than multicollinearity, Li and An (2015) include the father's attributes in the model.

There is a fourth group of studies that also looks at intergenerational transmission of in utero shocks. These papers, to my knowledge, are the only papers to look at the intergenerational transmission of shocks outside of the 3 events above (Lee, 2009; Caruso, 2015; Caruso and Miller, 2015). Lee (2009) looks at the effect of the Kwanju uprising in South Korea (which took place within an eleven day period in 1980) on the offspring of women who experienced the uprising in utero. The paper found that the offspring were more likely to be born with a low birth weight and to be born preterm. The papers by Caruso (2015) and Caruso and Miller (2015), look at the great flood of Tanzania in 1993 and the 1970 Ancash earthquake in Peru. Both papers find intergenerational transmission of health shocks for mothers to offspring, but not father to offspring. Children of mothers who experienced the 1993 flood in Tanzania were found to have lower height-for-age Z

scores, whilst children in Peru whose mothers experienced the Ancash earthquake had less education (0.4 years) than their peers in the control group.

3 A General Background on Senegal and the Shocks

Senegal is a country of 15 million people in West Africa. The country ranks 163 on the 2015 HDI rankings, with 52% of the population termed as multidimensionally poor[1] (UNDP, 2015). Senegal has had an average GDP growth rate of 3.9% between 2000 and 2014 (WB, 2015), without a single year of negative growth. Inequality at the national level is not pronounced, with a gini coefficient of 0.43, but results of the Oxford Poverty and Human Initiative study on multidimensional poverty show a significant difference in poverty incidence between the capital Dakar (46%) and the poorest department, Kolda (92%). The country's main exports are fishing, tourism and groundnuts, which make up the bulk of the country's foreign exchange earnings. As with many of its neighbors, agriculture is the main livelihood of the population, with an estimated 75% of the labor force engaged in the sector, both as a form of subsistence, and an income generating activity. Most of the farming in the country takes place in the central, southern and the eastern departments. Although farming does take place in the north, soil degradation, and increased desertification has meant that raising livestock has become a more profitable activity than farming. As in most of the Sahel region of West Africa, Senegal has seen an increase in the frequency of droughts since the 1960s.

Over the last 40 years, Senegal has experienced a number of droughts, mostly in the 1980s and the 2000s. None of these were as severe as droughts of 1983/84 and 2011, with 1983/84 being the worst by far. Other droughts within this period did not have a deep effect on the country's economy or lead to a humanitarian emergency. For example, although precipitation figures show that 2000/01 and 2006/7 were drought years, the lack of official data on the events and the fact that the Government of Senegal did not request assistance from the international community leads many to believe these two events were not as severe as the 1983/84 and 2011 episodes.

It is estimated that 1.2 million people were affected by the 1983/84 drought (USAID, 2013), with agricultural output estimated to be between 20 and 30 percent of the previous year's output. The effect on groundnut,

[1]This is the current measure used by UNDP to measure poverty. The measure is based on the deprivation of 10 basic necessities and the intensity of that deprivation.

then a major export and foreign exchange earner, only made the situation worse for the country. GDP fell by 5.3%, the biggest fall in the last 40 years. The same year saw agricultural worker value added fall by 24%, the largest fall in the country's history to date. The severity of the drought led to a concerted effort by the international community to help meet the shortfall in production, from countries such as the United States, France and Japan and international donors such as the World Food Program (WFP) and Christian Relief Services (CRS). The most affected regions were Diourbel, Fleuve (now Saint-Louis), Louga and Thies, situated in the west and northern parts of the country. It is important to note that, in 2011, these 3 regions were amongst the least food insecure and therefore more likely to be resistant to the 2011 drought as reported by WFP. The lack of data and reports on the 1983/84 drought means that little is known about the event beyond the monetary value of contributions by international donors (found in a USAID report), GDP figures from the World Bank and precipitation figures collected by weather stations at the time.

By comparison, the 2011 drought was less severe than the 1983/84 drought, but still significant, affecting an estimated 806,000 people (WFP, 2012). Unlike 1983/84, the effect on economic output was not severe given GDP growth was positive for that year. This is can be attributed to the diversification of the economy over that period towards manufacturing, mining, tourism and fishing, which make up the bulk of output, whilst agriculture, which employs almost 75% of the population makes up less than 20% of output. An increased reliance on imports of rice to meet the growing demand for the country's staple food meant the number of people affected by the drought was smaller than 1983/84. Farming communities in the south and central parts of the country, where fishing, and mining do not play a larger role saw a significant decline in agricultural output and therefore felt the effects of the 2011 drought more strongly.

As explained above, the 1983/84 and 2011 droughts were the two most severe droughts experienced by Senegal in the past 50 years. The growth figures for GDP and agricultural worker value added hide the fact that the severity of 1983 meant that the economy was coming from a very low base, and growth would occur as long as the following year was not as bad. As a result this paper treats both years as one drought, with the assumption that the occurrence of drought for each of the two years to be random and unpredictable from the perspective of households. Past evidence of rainfall patterns shows this to be true, with high rainfall years equally likely to be followed by low or high rainfall years. This assumption is tested during the robustness tests. We see no significant change in the results when only 1983 is considered the drought year. Due to this assumptions, this paper will treat all mothers born between November 1983 and August 1985 as having been exposed to the 1983/84 drought in utero and children born between November 2011 and August 2012

exposed to the 2011 drought in utero.

4 Methodology

4.1 Identification Challenges

The aim of this paper is to develop a model that can answer not only the effect of multiple shocks across generations, but also as a by-product, breakdown the effect of shocks into first (immediate) and second generation (the intergenerational transmission) effects. By merging both first generation and second generation studies, this paper has to deal with the challenges faced by both types of studies in attempting to identify the parameters of interest; confounding events, fertility selection and selection (culling).

The studies on the intergenerational transmission of in utero shocks are usually plagued with issues of confounding, with two of the 3 events mentioned in the Literature section (Influenza pandemic and the Dutch famine) occurring during world wars. As a result, it is difficult to tease out the separate effects of the war and the shock. This paper, by focusing on two droughts, natural disasters, that did not occur during any world or regional event that could impact child health, is free from the issues of confounding that we see in other papers on intergenerational transmission of in utero shocks.

Another challenge faced by papers on in utero shocks are issues of fertility selection by parents. This is expected to happened if parents have foreknowledge of the shock and adjust their fertility decisions. This is usually the case in papers that deal with shocks that are fairly predictable, or shocks that go on for a long period of time. Events such as the Dutch famine can be said to be among the former category, whilst the great China famine is an example of the latter. The Dutch famine, due to an embargo by the Germans during the second world war, could be said to have been anticipated by the population, and thus have led to a decision by parents to delay having children. In the case of the great China famine, the famine, which went on for nearly 3 years, meant that even if parents could not predict the onset of the famine, the extended duration led to delays in having children, something that has been shown to have occurred (Kim et al., 2010; Fung and Ha, 2009; Li and An, 2015). The two droughts that are the focus of this paper were both unpredictable, and also did not last as long. The 2011 drought started in the third quarter of that year, and by the first quarter of 2012, the government of Senegal had taken steps, along with the donor community to

deal with the crop failures and thus reducing the length and impact of the drought. The 1983/84 drought was actually two successive years of droughts, both unpredictable. The unpredictability of the second drought, along with its severity leads me to believe that first, fertility decisions were not altered by the drought, and second, although the Government of Senegal had sought assistance in September of 1983, the severity of the next drought more than outweighed any effects to tackle the widespread crop failures of the 1983.

Selection (culling) is a common challenge faced by both the first and second generation studies on in utero shocks. This is similar to the sample selection problem, with a reduction in the number of children who survive to age 5. This occurs for two reasons, the first being an increase in the number of stillbirths and abortions (natural) and the second an increase in neonatal (and infant) mortality. Meng and Qian (2009) and Gørgens et al. (2012) show that famines lead to an increase in neonatal mortality by killing off those that are too weak to survive the famine; had the famine not occurred, the infant would have survived. The result is only those that have strong constitutions would be present in our dataset; those too weak would never be observed. Given those who survived are the strongest (have stronger constitutions) of the cohort, any results obtained should be seen as a lower bound. Those not observed, if included, would increase the effect of the shock on our outcomes in absolute value terms. Generally, studies dealing with in utero shocks ignore the effect of child survival and interpreting the results obtained as lower bounds. This makes sense as it has been shown by Gørgens et al. (2012), Almond et al. (2010) and Bozzoli et al. (2009) that during periods of adverse shocks, there is an increase in selection (culling) which is more likely to affect those with weaker genes and as a result lead to an underestimation of the true impact of the shock.

This paper, by using information on all reported pregnancies, not only those that led to a live birth but also those that were terminated, would make it possible to estimate a Heckman two stage model to determine how sample selection affects our estimates of interest. This method of testing and adjusting the estimates is possible due to our dataset having information on the birth history of all women in the sample. Women were asked about not only their live births, but also pregnancies that were terminated and the month of termination. The data being used, the Senegal Continuous DHS dataset, contains information on all pregnancies for women in the sample, whether the child was alive during the survey or not. As a result, this paper uses this information to estimate a Heckman two stage model to determine whether sample selection (culling) for fetuses, neonates or infants has a significant effect on the baseline results. This is done by treating all children whose anthropometry data was collected as observed and those not alive at the time of the survey as unobserved. Results of the Heckman show that the difference-in-difference model does underestimate the

effect of the drought on child health (immediate effect), but does not underestimate that for the intergenerational and resistance transfer. The results are in line with the literature on how the selection (culling) effect impacts our estimates when selection is not accounted for.

It is important to note that the selection criteria in this paper follows that by Neelsen and Stratmann (2011), which shows that using children born before the drought as part of the control group would muddle the results obtained; in essence we would be comparing apples to oranges. The reason for this being that children born before the drought would experience the drought as children (or infants) and any childhood outcomes used would be a comparison of the effect of drought on children (the control group) versus its effect in utero, the comparison group. To avoid this issue, only children born from November 2011 onwards are used in the analysis with children born after August 2012 (after the end of the drought) serving as the control group. It should also be noted that this is not a requirement when the outcome measure is birthweight. Although not shown in the paper, including children who were born before the 2011 drought yields similar result to only including children born after October 2011.

A second point to note is the use of all women within the sample. Unlike the children, the paper by Kim et al. (2010) has shown that intergenerational transfer of in utero shocks only applies to women who experience the shock in utero. There is no intergenerational transfer if a woman experienced the drought as a child or infant. As a result, all children born after September 2011 will be included in the sample, regardless of when their mother was born.

Finally, we have to deal with the fact that even though there are systemic differences in the characteristics of both groups of mothers, there should not be a difference in fertility decisions of mothers in terms of their children. In short, there should be no systemic difference in the fertility decision of mothers when we compare treated and untreated children. As will be shown in the results section, although the mothers exposed to the 1983/84 drought in utero have their first birth a year later than non exposed mothers, there is no difference in age at first birth when the mothers of treated children are compared to those of the untreated children.

4.2 Identification Strategy

This paper looks at whether a mother (F1) experiencing a shock in utero, would increase resistance of her child (F2) to shocks in utero. The study does this by choosing two events that occurred in Senegal, the first

in 1983/84, the second in 2011. The data used is the Senegal Continuous Demography and Health Survey (CDHS) an ongoing DHS survey in Senegal which started at the end of 2012 and ended in late 2016. The data used in this paper is from the first two rounds of the survey, which took place from end 2012 to mid 2014. The data set has a total of 5,209 children born after October 2011, with information on the attributes, anthropometry measures and their mother's characteristics. The women in the sample are between the ages of 15 and 49 whilst all children are under the age of 5.

The paper identifies the parameters of interest by using a difference-in-difference type model, where the children in the sample are grouped in to 4 categories. The first group of children (the control) are children born after August 2012 whose mothers were not in utero during the 1983/84 drought in Senegal. The second group (intergenerational effect group) are children born after August 2012, but whose mothers were in utero during the 1983/84 drought. The third group (immediate effect group) are children born between November 2011 and August 2012 and thus exposed to the drought in utero for at least 3 months, but whose mother was not exposed to the 1983/84 drought. The fourth group (both effect group) are children born between November 2011 and August 2012 who had a mother who was in utero during the 1983/84 drought.

Comparing groups 2, 3 and 4 to the control group would allow us to estimate the immediate effect of exposure in utero on child health (first generation effect), the intergenerational transmission effect (second generation effect) and transfer of resistance from mother to child respectively. Results for the immediate effect are comparable to results from studies on impact of droughts (or extreme weather effects) on child health such as Almond et al. (2010), Caruso (2015) and Kumar et al. (2014), whilst results for the intergenerational transmission are comparable to the second generation models such as Lee (2014), Richter and Robling (2013) and Li and An (2015). The interaction term will answer the question of interest for this paper; whether a mother's exposure to an in utero shock would increase the resistance of her offspring to in utero shocks.

$$y_{is} = \beta_0 + \delta_0 d_0 + \delta_1 d_1 + \delta_2 d_0 d_1 + C'\phi_c + X'\phi_x + V'\phi_v + u_i, \tag{1}$$

Model 1 shows the base model estimated using difference-in-difference, where y_{is} is the child health outcome of interest. The outcomes of interest are birth weight[2] (BW), weight-for-age z-score (WAZ), height-for-age

[2]Only birth weight that was verified with an antenatal card shown to the interviewer was used in the analysis. Birth weight figures obtained through recall were not used. This was done as recall birth weights are significantly higher than official birth weight figures.

z-score (HAZ) and weight-for-height z-score (WHZ). On the right hand side, δ_0 is the effect of exposure to a drought in utero on child health (immediate effect), δ_1 the effect of having a mother exposed to a drought in utero (intergenerational transmission) and δ_2 the transfer of resistance from mother to child; the parameter of interest. The variable d_0 is a dummy for whether child was exposed to the 2011 drought in utero, whilst d_1 is a dummy for whether child's mother was exposed to the 1983/4 famine in utero. The vectors C, X and V are controls for child, mother and settlement respectively. The controls for the child are, the child's sex ($female$) and whether child was born during lean season[3] ($lean$). The mother controls are whether she went for antenatal care during her first trimester (anc), her age at last birth(age) and whether she is a farmer ($farmer$). There is only one control for the settlement, whether the settlement is in an urban area ($urban$).

A second model to determine whether there was a differential effect for length of exposure is also estimated. Model 2 is similar to Model 1 with the addition of exposure by trimester, where the variables tr_1, tr_2 and tr_3, refer to first, second or third trimesters respectively. Each is a dummy variable for whether the child was exposed to the 2011 drought for that particular trimester in utero. The controls used in Model 2 are the same as those in Model 1.

$$y_{is} = \beta_0 + \delta_0 d_0 + \delta_1 d_1 + \delta_2 d_0 d_1 + \gamma_1 tr_1 + \gamma_2 tr_2 + \gamma_3 tr_3 + C'\phi_c + X'\phi_x + V'\phi_v + u_i, \tag{2}$$

5 Descriptive Statistics

Table 1 shows the summary statistics of the 4 outcomes of interests, by category (Region, Wealth and Educational Attainment of Mothers). As is expected, urban children have better health outcomes across the board when compared to rural children. Children who live in the urban areas have higher birth weights, and better anthropometric measures, although we see that the mean anthropometric figures are below zero. A similar pattern is seen when we look at mean health outcomes based on the wealth quintile of the household the child lives in. Children from the bottom fifth exhibit the poorest health outcomes across the board, with health outcomes improving as we move to higher wealth quintiles. This is in line with the literature on child health in general and reports on Senegal in particular. The results for educational attainment of mothers is not as straight forward as those for urban/rural and wealth quintile. The general trend is that higher

[3]The lean season refers to the months when farming households' food stocks are at their lowest. Children born during this period would have been in utero during the period when food stocks were high. Studies show these child weight more than children born outside the lean months. In Senegal, the lean months are from March to September.

educated mothers have children with better health outcomes, but the relationship is not linear, for example children of women who completed primary school have lower health outcomes when compared to children whose mother did not complete primary school.

Tables 2 and 3 show the difference in means outcome for treated and non treated (children, Table 2 and mothers, Table 3). For Table 2 columns 2 to 4 are the measures based on the treatment status of the children, and columns 5 to 7 are based on the treatment status of the mother of the children. A priori, we would expect there to be differences in means for children when we compare them based on the exposure of their mothers. For our parameters to be identified, this is not necessary. As long as there is balance between the children based on their exposure (columns 2 to 4) then our estimates will be unbiased.

The first 4 variables of Table 2 shows the difference in outcomes variables (*birth weight*, *weight-for-age* z score, *weight-for-height* z score and *height-for-age* z score). We observe no difference for *birth weight* but the other three anthropometry outcomes show a difference when we compare children exposed to the 2011 drought in utero to those that did not. This is to be expected and is in line with the literature. When we compare the same variables by exposure of the mother, we see no difference in anthropometry measures between the children. As we will see in the Section 6, this is due to the double exposed effect running counter to the intergenerational effect.

Focusing on the variable *child alive* in Table 2, this refers to whether the child is observed in the data. In both cases, comparing mother and child exposure, we see no difference in the probability that the child is alive. This tells us that the 2011 drought did not significantly impacted neonatal deaths as was would be expected. Even though the results show that child survival differences between the drought and non drought periods are not statistically different, a Heckman two step procedure is shown in Table 7 in the Appendix of this paper.

Focusing on the age at which the child's mother first gave birth, we observe that children of mothers who were exposed in utero get married almost a year and half later than their counter parts (21.51 versus 19.99 years) which is statistically significant. As mentioned in Section 4 this is not a problem since we observe no significant difference in the age during first birth when comparing treated and untreated children.

Moving to Table 3, the characteristics of the women in the sample are compared based on their exposure to

the 1983/84 drought in utero. We observe no difference in characteristics between treated and non treated mothers in terms of education (in years) and antenatal care visit within the first trimester. The results for education should be interpreted with caution. The age range of women in the sample is 15 to 49 years, whilst women exposed to the 1983/84 drought are between 28 and 31; which puts them at the center of the age distribution. The trend for education is that younger cohorts have higher education levels compared to older cohorts, and as a result exposed women, being in the middle of the distribution have a mean education outcome equal to all cohorts combined. In short, we cannot tease out the cohort effect from the drought effect even when we control for year of birth. The last two rows of Table 3 show the likelihood of a woman giving her child's birth weight by recall and by ANC card respectively. No difference in the likelihood is observed in both cases when comparing based on woman's exposure.

6 Results

6.1 Difference-in-difference Results

This section shows the results for the 4 outcomes of interest using a difference-in-difference estimator. Table 4 gives a condensed table for the 3 regressions models, focusing on the 3 variables of interest; *child exposed*, *mother exposed* and *both exposed*. The variable *child exposed* gives the immediate impact of in utero exposure to drought on child health. *Mother exposed*, measures the intergenerational (second generation) effect of in utero exposure to the 1983/84 drought on the children of those exposed. *Both exposed*, the variable of interest, measures the transfer of resistance to in utero exposure to drought from mother (F1) to offspring (F2).

The DiD model, has controls for child attributes, mother attributes and the settlement. All standard errors are clustered at the settlement level. The results for the DiD show that the 2011 drought had a negative effect on child health outcomes, with children exposed to the drought having lower weight-for-age, weight-for-height and height-for-age Z scores (-0.307, -0.211 and -0.363 respectively) when compared to the control group. The results echo those of the literature such as Kumar et al. (2014), Caruso (2015) and Neelsen and Stratmann (2011). The immediate (first generation) effect is significant at 1 percent for all the anthropometry outcomes. The results for birth weight are not significant for all 3 variables. This is also seen in the Pooled and Cluster Fixed Effects models discussed below.

The intergenerational effect of in utero exposure is negative, as is seen in the literature, although not significant at 10 percent. The intergenerational effect is -0.215 for WAZ (under weight), -0.165 for WHZ (wasting) and -0.190 for HAZ (stunting). The parameter of interest (both exposed) is consistently positive in all 3 regressions; 0.347 for WAZ, 0.271 for WHZ and 0.217 for HAZ. The intergenerational transfer of resistance is only significant in the WAZ and WHZ (under weight and wasting) regressions, being significant up to 5 percent. This is not the case with the stunting regression, suggesting that the effect of the transfer works more through weight than height. Focusing on the magnitude of the both exposed parameter, we see that the resistance transfer is larger than the intergenerational effect. In short, the benefit of the resistance transfer to in utero shocks more than wipes out the negative effect of the intergenerational effect, leading to children who have in essence been "double exposed" having better health outcomes than G2 group (mother exposed only) and G3 group (child exposed only) children. The resistance transfer from mother to child, in the case of weight-for-age z score, leads to a net effect (when compared to the control group) of -0.175 for "both exposed" children compared to -0.307 for child exposed (G3) and -0.215 for mother exposed (G2) children.

6.2 Robustness Checks

The second set of parameters in Table 4 are from the Pooled cross sectional regression. A Pooled OLS was estimated to determine whether there are significant differences between children in the different rounds of the Senegal Continuous DHS survey, and whether these differences would have a significant effect on the results. Compared to the DiD results above, we see little movement among the 9 parameters of interest, with a notable difference in the height-for-age regression for child exposed (-0.363 versus -0.651). The signs still stay the same for all 9 parameters, as well as the significance level. A Hausman specification test (not shown in paper) where DiD is the efficient estimator and Pooled OLS the consistent estimator, shows that the consistent model should be chosen. In addition, a simple Z test (not shown in the paper), to determine whether there is a significant difference between the parameters when DiD is compared to Pooled, shows that for all 9 parameters, there is no statistical difference. In essence, choosing between the DiD and Pooled is a matter of preference.

To check for the possibility of unobserved village or neighborhood fixed effects, a cluster fixed effects was estimated. If there is an unobserved fixed effect, at either of these levels, we should see a significant change in the estimates. Due to the time period used, August 2011 to August 2014, and that only the measurement of the youngest child is taken; no child in our sample has height and weight measurements for their sibling

taken, we therefore cannot estimate a family fixed effect model. In addition, estimating a family fixed effects would wipe out the parameter for mother exposed, which is vital. Due to these two challenges, a cluster fixed effect model is estimated instead. A cluster in this case is a group of households within a settlement (city, town or village) that are near each other.

A cluster is composed of a maximum of 500 people. If there are any unobserved fixed effects either at the cluster level or higher (neighborhood, village or region), we would expect to see a significant change in our estimates. The lower part of Table 4 shows that, once we control for the cluster fixed effects, the magnitude of the estimates are higher than those for the difference-in-difference across all 9 estimates, although the signs are still the same. The change in magnitude is not statistically significant (test not shown), but we see that mother exposed is now significant at 10 percent in all 3 regressions and so is the both exposed estimate in the height-for-age (stunting) model (0.419). As in the DiD, the transfer of resistance from mother to child (both exposed) is strong enough to wipe out the negative intergenerational effect. A Hausman specification test shows that the consistent estimator, Fixed Effect, should be chosen over the efficient estimator, DiD.

The consistency of the results across different models points to a strong case for the transfer of resistance to in utero shocks from mother to child, an effect strong enough to wipeout the negative intergenerational effect and part of the immediate drought effect. How this occurs, whether through a gene effect, an environment effect or a combination of the two, is beyond this paper, but the results of this paper point to a strong case for further study in this area.

Below we see a side by side comparison of the results for under weight (weight-for-age), Table 5 and wasting (weight-for-height), Table 6 for ease of comparison. It can be seen that in the case of under weight, the difference in estimates when we compare the results for Heckman, Pooled and FE to DiD are not that significant. The biggest difference is between DiD and FE, with DiD under estimating the true impact of in utero exposure. The same pattern is observed when we focus on Table 6. As with Table 5, the signs and significance are consistent across all 3 models, providing a strong case for transfer of resistance to in utero shocks.

As mentioned in sections 2 and 4, the issue of selection (culling) is mostly side stepped since it has now been shown that any results obtained would be a lower bound of the true effect of in utero drought exposure drought. To test this, I use information provided in the DHS on terminated pregnancies to test whether including pregnancies that were terminated (unborn children) and children who were born alive but died

prior to the survey taking place, would significantly impact the results. Table 7 shows the results of a Heckman two stage regression. We can observe that in the case of all 3 anthropometry measures, the Heckman results for the effect on child health are larger than the DiD; which is in line with the literature. The positive selection has led to the DiD underestimating the true effect of the drought. The difference between the Heckman results and the DiD end there. We see no difference in the case of the other two parameters, with the DiD and the Heckman having virtually the same estimates for Mother exposed and Both exposed.

All four regressions, DiD, Pooled, FE and Heckman show no difference in birthweight between the treated and untreated children. Even after accounting for selection, we see no statistical difference between the groups. I believe that this could be due either to sample size (approximately 5,000) being too small to detect the effect of the drought on birthweight, or our measure of terminated pregnancies is a poor proxy for unborn children. Given that similar results are observed in all four regressions for the 3 anthropometry outcomes, the case can be made that the former is a more likely reason for the results observed for birthweight.

In this paper we treat both the 1983 and 1984 droughts as one random shock arguing that the randomness of both events is a reasonable assumption to make. If families in 1984 after experiencing the 1983 drought changed their fertility decisions, then we should expect to see different results if only 1983 was used as the shock rather than both years. A separate set of regressions were estimated, using DiD, Pooled and Fixed Effects and in this case only women exposed to the 1983 drought in utero for at least 3 months were categorised as exposed. Table 9 shows results very similar to those in Table 4. The magnitudes of the coefficients are statistically the same for all 3 estimation techniques, although their significance is weaker in the DiD and Pooled models now. The Fixed Effects models, as before gives the stronger results and is also the preferred model of the 3. These results lends credibility to the use of both 1983 and 1984 as the exposure years.

7 Conclusion

This is the first paper that looks at whether resistance to in utero exposure to shocks (specifically droughts) can be passed on from mother to child. This question is answered by identifying two drought events that occurred in Senegal, the first in 1983/84 and the second in 2011. In addition, this paper also shows that the separate strands of the literature on in utero shocks, impact of in utero shocks on child health and intergenerational transfer of in utero shocks, can be estimated together within the same model. Using a

cross sectional data set, the Continuous DHS for Senegal, and information on date of birth for children and mothers, children in the sample are put into one of four groups. These groups are, the control group which was not exposed to the 2011 drought, nor had a mother exposed to the 1983/84 drought, the mother exposed group, the child exposed group and the both exposed group (double exposed group).

Using a difference-in-difference model, the results show that there is a transfer of resistance to in utero shocks from mother to child. The magnitude of the transfer is strong enough to wipe out the negative intergenerational transfer of in utero shock from mother to child, and also a portion of the immediate impact of the child's exposure to the drought. As a result, children who are "double exposed" have better health measures (a net effect of -0.175) than children who are either directly exposed (-0.307) to the 2011 drought or whose mother was exposed (-0.215) to the 1983/84 drought in utero with the control children as the benchmark group.

The significance of the "double exposed" estimates for weight-for-age and weight-for-height, point to the resistance working through weight more than height, although the results of the Fixed Effects shows a significant effect on height. The robustness of the results to other estimation techniques and models points to a strong case for a transfer of resistance to in utero exposure to shocks from mother to child. This paper also shows that selection (culling) has an effect on the estimates for the child effect, but not intergenerational and resistance transfer effects. Although the results are robust to different techniques, this paper does not delve into the transmission mechanism; how this resistance is transferred, whether by genes, environment or a combination of both. It is our hope that this paper will serve as the catalyst for further research into how this in utero resistance to shocks is transferred from mother to child.

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8 Tables

Table 1: General Statistics, Outcome Variables

	BW	HAZ	WAZ	WHZ
Rural				
Urban	3105.84	-0.51	-0.27	-0.59
Rural	3068.05	-0.78	-0.40	-0.87
Wealth index				
Poorest	2976.27	-0.95	-0.54	-1.02
Poorer	3060.65	-0.78	-0.36	-0.93
Middle	3107.13	-0.53	-0.27	-0.62
Richer	3131.46	-0.53	-0.25	-0.57
Richest	3131.86	-0.21	-0.14	-0.29
Educational attainment				
No education	3080.29	-0.78	-0.42	-0.85
Incomplete primary	3107.19	-0.56	-0.31	-0.68
Complete primary	3146.32	-0.72	-0.31	-0.98
Incomplete secondary	3048.69	-0.38	-0.04	-0.56
Complete secondary	3205.88	-0.32	-0.14	-0.45
Higher	3128.33	-0.14	-0.09	-0.08
Total	3084.79	-0.70	-0.36	-0.79
Observations	2662	4865	4844	4849

[1] BW, Birth Weight; HAZ, Height-for-Age; WAZ, Weight-for-Age; WHZ, Weight-for-Height

[2] Wealth Index is in quintiles, with Poorest the bottom quintile and Richest the top. Calculated using principal components analysis.

[3] The table gives the descriptive statistics, breaking down the outcomes of interest (BW, WAZ, HAZ and WHZ) by the characteristics of the mother and household. Rural refers to whether the household the mother resides in is in the urban or rural areas. Wealth index is the wealth quintile group the household falls under and educational attainment refers to the level at which the mother stopped schooling. Values in table are mean of outcome variable for the group.

Table 2: Test of Means by Child and Mother Exposure

	Children Exposure in utero			Mother Exposure in utero		
	(1) No Mean	(2) Yes Mean	t-test (1) - (2) p-value	(1) No Mean	(2) Yes Mean	t-test (1) - (2) p-value
Child's Birth weight	3081.648	3091.994	0.923	3089.784	3066.425	0.995
Weight-for-Age Z score	-0.413	-0.699	0.000***	-0.609	-0.503	0.227
Weight-for-Height Z score	-0.177	-0.371	0.000***	-0.309	-0.251	0.161
Height-for-Age Z score	-0.483	-0.836	0.000***	-0.720	-0.668	0.190
Child is Alive	0.971	0.967	0.481	0.968	0.965	0.733
Child Female	0.502	0.497	0.915	0.494	0.568	0.748
Age of Mother during 1st birth	20.049	20.125	0.659	19.994	21.505	0.000***
Mother's education (years)	2.362	2.152	0.203	2.215	2.334	0.792
ANC First Trimester	0.535	0.520	0.491	0.524	0.542	0.447
Age of Mother during last birth	27.612	27.444	0.815	27.427	28.490	0.000***
Mother is a farmer	0.171	0.177	0.864	0.177	0.141	0.310
Wealth Index Score household	1.25e+05	1.01e+05	0.635	95500.051	2.92e+05	0.013**

* 10%; ** 5%; *** 1%

[1] This table shows the test of means using the children's dataset. it looks at the attributes of the children and their mothers.

[2] Table tests differences in the left hand variables by exposure to drought for child (2011) (columns 2 to 4) and mother (1983/84) (columns 5 to 7).

[3] In all instances standard errors were clustered at the cluster level and sample weights were used.

Table 3: Test of Means by Mother's Attributes

	Woman Exposure in utero		
	(1)	(2)	t-test
	No	Yes	(1) - (2)
	Mean	Mean	p-value
Woman's education (years)	2.188	2.355	0.776
ANC First Trimester	0.550	0.555	0.530
Age of Woman during last birth	27.506	28.504	0.000***
Woman is a farmer	0.178	0.142	0.317
Wealth Index Score household	98986.245	3.05e+05	0.008***
Birth weight given is from recall	0.369	0.372	0.556
Birth weight given if from ANC card	0.631	0.628	0.556

* 10%; ** 5%; *** 1%

[1] The table above shows the test of means focusing on the mothers of the children. For this analysis, the Women's dataset is used.
[2] The two groups of women are, those not exposed to the 1983/84 drought in utero (column 2) and those exposed in utero (column 3).
[3] In all instances standard errors were clustered at the cluster level and sample weights were used.

Table 4: Results for DiD, Pooled and Fixed Effects

Results of DiD Regression

	Birth weight	Weight-for-Age	Weight-for-Height	Height-for-Age
Child Exposed	-7.673	-0.307	-0.211	-0.363
	[27.860]	[0.052]***	[0.049]***	[0.058]***
Mother Exposed	-91.15	-0.215	-0.165	-0.19
	[93.889]	[0.137]	[0.109]	[0.152]
Both Exposed	144.174	0.347	0.271	0.217
	[131.106]	[0.171]**	[0.128]**	[0.207]
R-Squared	0.04	0.05	0.02	0.05

Results of Pooled OLS

	Birth weight	Weight-for-Age	Weight-for-Height	Height-for-Age
Child Exposed	-9.743	-0.451	-0.219	-0.651
	[33.895]	[0.047]***	[0.054]***	[0.055]***
Mother Exposed	-91.133	-0.206	-0.164	-0.172
	[93.911]	[0.137]	[0.109]	[0.153]
Both Exposed	144.271	0.354	0.271	0.243
	[131.084]	[0.170]**	[0.128]**	[0.206]
R-Squared	0.04	0.06	0.02	0.1

Results of Fixed Effects at Settlement cluster Level

	Birth weight	Weight-for-Age	Weight-for-Height	Height-for-Age
Child Exposed	-25.242	-0.486	-0.225	-0.701
	[38.204]	[0.052]***	[0.055]***	[0.055]***
Mother Exposed	-63.562	-0.301	-0.222	-0.306
	[95.622]	[0.158]*	[0.121]*	[0.161]*
Both Exposed	121.905	0.418	0.335	0.419
	[146.100]	[0.195]**	[0.135]**	[0.224]*
R-Squared	0.03	0.04	0.01	0.07
Observations	1,870	4,865	4,844	4,849

* 10%; ** 5%; *** 1%

[1] Table shows condensed results for the difference-in-difference, Pooled OLS and cluster Fixed Effects. Each column is a separate regression with all controls included in the regression .
[2] All 4 models were regressed using the survey's sample weights and standard errors were clustered at the cluster level.
[3] A cluster in the Senegal Continuous DHS data refers to an area with approximately 500 people; can be viewed as a neighborhood.

Table 5: Comparison of Results, Weight-for-Age

	DiD	Heckman	Pooled	FE
Child Exposed	-0.307	–0.319	-0.451	-0.486
	[0.052]***	[0.052]***	[0.050]***	[0.052]***
Mother Exposed	-0.215	-0.216	-0.206	-0.301
	[0.137]	[0.136]	[0.137]	[0.158]*
Both Exposed	0.347	0.345	0.354	0.418
	[0.171]**	[0.169]**	[0.170]**	[0.195]**
R-Squared	0.05		0.06	0.04
Observations	4,865	5,725	4,865	4,865

* 10%; ** 5%; *** 1%

[1] Table shows side by side the results for outcome Weight-for-Age for the 4 regression techniques used in this paper. These results in the second, fourth and fifth columns are the same as those in Table 4 above.

Table 6: Comparison of Results, Weight-for-Height

	DiD	Heckman	Pooled	FE
Child Exposed	-0.211	–0.219	-0.219	-0.225
	[0.049]***	[0.049]***	[0.051]***	[0.055]***
Mother Exposed	-0.165	-0.165	-0.164	-0.222
	[0.109]	[0.109]	[0.109]	[0.121]*
Both Exposed	0.271	0.271	0.271	0.335
	[0.128]**	[0.128]**	[0.128]**	[0.135]**
R-Squared	0.02		0.02	0.01
Observations	4844	5725	4844	4844

* 10%; ** 5%; *** 1%

[1] Table shows side by side the results for outcome Weight-for-Height for the 4 regression techniques used in this paper. These results in the second, fourth and fifth columns are the same as those in Table 4 above.

Table 7: Results of Heckman Two step Regression

	Birth weight	Weight-for-Age	Weight-for-Height	Height-for-Age
dc_1	16.488	-0.319	-0.219	-0.38
	[30.590]	[0.050]***	[0.051]***	[0.058]***
dm_4	-22.233	-0.216	-0.165	-0.197
	[100.818]	[0.136]	[0.109]	[0.148]
T1	83.504	0.345	0.271	0.216
	[127.805]	[0.169]**	[0.128]**	[0.208]
p value, rho=0	0.0122	0.0002	0.2156	0.0000
Observations	5,663	5,725	5,725	5,725

* 10%; ** 5%; *** 1%

[1] Table shows results from the Heckman two step regression. Each column shows a separate regression for each of the outcomes.
[2] In all instances standard errors were clustered at the cluster level and sample weights were used.

Table 8: Results DiD, Trimester of Exposure for Child

	Birth weight	Weight-for-Age	Weight-for-Height	Height-for-Age
Child Exposed	-79.375	-0.338	-0.262	-0.435
	[69.995]	[0.100]***	[0.088]***	[0.093]***
MotherExposed	-89.002	-0.215	-0.165	-0.191
	[93.831]	[0.137]	[0.110]	[0.152]
Both exposed	152.641	0.342	0.26	0.208
	[128.113]	[0.171]**	[0.128]**	[0.207]
Trimester 1	-46.621	-0.021	0.066	0.078
	[59.462]	[0.072]	[0.066]	[0.080]
Trimester 2	14.248	0.148	0.119	0.048
	[60.531]	[0.075]**	[0.078]	[0.085]
Trimester 3	125.793	-0.101	-0.124	-0.031
	[54.912]**	[0.076]	[0.078]	[0.082]
R-Squared	0.05	0.05	0.02	0.05
Observations	1,870	4,865	4,844	4,849

* 10%; ** 5%; *** 1%

[1] All 4 models were regressed using the survey's sample weights and standard errors were clustered at the cluster level.
[2] DiD regressions with trimester of exposure for child. Variables trimester1, trimester2 and trimester 3 are dummy variables for whether child was exposed to 2011 drought in utero during that trimester. The same controls are used as those in the base model.

Table 9: Results using only 1983 drought as exposure period

	Difference-in-Difference Results			Pooled OLS Results			Cluster Fixed Effects Results		
	Weight-for-Age	Weight-for-Height	Height-for-Age	Weight-for-Age	Weight-for-Height	Height-for-Age	Weight-for-Age	Weight-for-Height	Height-for-Age
Child Exposed	-0.304***	-0.207***	-0.362***	-0.448***	-0.215***	-0.649***	-0.483***	-0.220***	-0.699***
	(0.051)	(0.049)	(0.058)	(0.047)	(0.055)	(0.055)	(0.052)	(0.056)	(0.055)
MotherExposed	-0.198	-0.155	-0.169	-0.189	-0.155	-0.152	-0.279*	-0.215*	-0.275*
	(0.138)	(0.118)	(0.148)	(0.138)	(0.118)	(0.150)	(0.163)	(0.130)	(0.159)
Both exposed	0.334*	0.226	0.216	0.339*	0.227	0.237	0.409**	0.296**	0.413*
	(0.180)	(0.141)	(0.210)	(0.179)	(0.141)	(0.211)	(0.204)	(0.148)	(0.229)
R-squared	0.049	0.017	0.048	0.063	0.017	0.098	0.039	0.010	0.067
Observations	4,865	4,844	4,849	4,865	4,844	4,849	4,865	4,844	4,849

* 10%; ** 5%; *** 1%

[1] All models were regressed using the survey's sample weights and standard errors were clustered at the cluster level.
[2] Table shows the results when only 1983 is used as the exposure period for mothers. These results have the same controls as those in Table 4.