INTERGENERATIONAL CONSEQUENCES OF IN-UTERO AND EARLY-LIFE CONDITIONS

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Abstract

This thesis comprises three self-contained chapters in applied microeconometrics, with an overall underlying theme of exploring the intergenerational transmission of effects of early-life conditions on human capital outcomes.

Chapter 2 traces the impacts of in-utero exposure to negative rainfall shocks on the outcomes of grandchildren of those affected using representative survey data from the India Human Development Survey (IHDS). I find that (a) exposure to rainfall shock during pregnancy has strong negative effects that pass down to health and cognitive ability of the third generation; and (b) biological explanation, plausibly genetic and epigenetic inheritance, may be the key transmission mechanism of these effects.

Chapter 3 investigates the gender-differential response of parents' expenditure to changes in child's health due to negative rainfall shocks in early childhood. I link the rural sample of Young Lives survey for Andhra Pradesh, India with the district-level monthly rainfall data, and use an instrumental variable framework. Findings show that when negative rainfall shocks adversely affect children during early childhood, parents compensate by investing more in their education. My results uncover gender discriminatory investment behaviour, where parents invest more in education of a boy as compared to a girl.

Chapter 4 analyses the intergenerational effects following the positive changes in women's inheritance rights. I employ a difference-in-differences strategy and exploit the state level variation in a woman's exposure to the Hindu Succession Amendment Act in India. Using the IHDS data, I find that the property rights reform significantly increased the health of children whose mothers were exposed to the amendment. Further analysis reveals a substantive gender-differential pattern; the health of daughters is significantly worse than that of sons, but is only visible in children with less educated mothers. I discuss mothers' improved bargaining power in household decisions as the main channel through which the reform operates. To my family.

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I declare that this thesis is a presentation of original work and I am the sole author of the three self-contained chapters. This work has not previously been presented for an award at this, or any other university. All sources are acknowledged as references. Funding for my studies was partially provided by a three year PhD Studentship by the Department of Economics and Related Studies and a Scholarship for Overseas Students at University of York. Finally, I declare that the funders and data creators have no responsibility for the contents of this thesis.

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Maha Khan York, June 2021

Chapter 1

Introduction

This thesis comprises three self-contained chapters in applied microeconometrics, with a special focus on areas of family economics, early child development, and economics of gender and health. They all straddle the intergenerational transmission of effects of early-life conditions on human capital outcomes. The underlying theoretical foundation is based on a model of "fetal origins hypothesis", which suggests that the intrauterine environment is not a protected state and that any adverse conditions in utero can cause modifications in the fetus including also epigenetic changes which, even though remain invisible for most of the life course, may result in serious morbidities later in life (e.g., Almond & Currie, 2011b). The importance of this phase can be gauged from Barker's (1990) argument that "[t]he womb may be more important than the home". This garnered interest from several empirical studies from medical literature and other social sciences that found evidence for correlations between an array of in-utero and early-life circumstances and later-life outcomes, but oftentimes failed to establish a robust, causal association.

The connection between early-life conditions and adult outcomes has raised and continues to raise considerable individual and societal interest, especially because of the potentially severe economic consequences. Empirical evidence from studies in economics shows that in utero (and early-life) shocks appear to adversely affect adult health (e.g., Almond, 2006; Almond & Mazumder, 2011; Bhalotra & Rawlings, 2011; Lin & Lui, 2014; Van den Berg et al., 2006) as well as investment behaviour (Cronqvist et al., 2016) and other human capital and labour market outcomes (e.g. Almond et al., 2009; Bhalotra & Venkataramani, 2016; Nelson, 2010). Unfavourable early life conditions seem to be prominent drivers of economic inequality, some of which may be preventable if the damages are compensated by suitable interventions and policies early on.

The main objective of this thesis is to focus on giving new econometric insights into understanding the long-term risks of early life shocks and their impact, along with proposing potential ways to remediate them. This can offer policy makers with the knowledge and tools to provide individuals a more level playing field from childhood, which is of paramount interest in developing countries where the effect is already likely to be larger due to poorer socioeconomic conditions and constraints in spending on mitigating strategies. Chapter 2 addresses the thesis's central question regarding the relationship between adverse exposure to in-utero shocks and human capital outcomes; more specifically, the effect on outcomes of the third generation (i.e. on grandchildren of women who experience shock while pregnant) and the mechanisms driving these effects. Once the relationship is established, Chapters 3 and 4 explore ways to potentially mitigate the effects of shocks through parental investment behaviour and policy reforms, respectively. The main econometric challenge when studying the impact of early life shocks is to isolate its causal effect. Thus, to provide results closer to a causal interpretation, I rely on "quasi-experiments" and instrumental variable techniques as substitutes for random experiments. What follows, outlines more detailed motivation and contribution of each of the three chapters that make up this thesis.

Climate change related increases in temperatures have led to a rise in the frequency and intensity of extreme weather shocks such as storms, heat waves, droughts, and floods. Poorer communities in much of the developing countries end up facing worse economic and social costs as they are already under-resourced to shoulder the burden of environmental shocks. The worst hit by these shocks are people who subsist on agriculture. Extreme weather shocks can cause a reduction in consumption and nutritional intake of children, which can lead to adverse consequences for child human capital, adult health (e.g. in the form of reduced longevity) and labour market outcomes (e.g., loss in productivity) (Almond, 2006; Currie, 2011; Maccini & Yang, 2009; Shah & Steinberg, 2017).¹ Children who are exposed to these shocks in their formative period (normally the first 1000 days – in utero and two years post birth) face lasting negative effects (Caruso & Miller, 2015) in terms of impaired cognitive development and physical health deficits in childhood and adulthood (Almond & Mazumder, 2011; Petronis, 2010). Nutritional stress in the pre- and post-natal period can cause epigenetic and physiological changes that determine an individual's developmental trajectory. This is particularly the case with women because a female is born with all the reproductive eggs she will ever produce in her lifetime; thus, an exposure to a prenatal shock has the potential to directly affect three generations (Almond & Mazumder, 2011; Osmani & Sen, 2003). These

¹ The most important factor determining longevity and quality of adult health is fetal nutrition of the mother when she was pregnant with the child and also of the grandmother when she was pregnant with the mother.

include not only herself and her unborn child (the fetus), but also the immature germ cells within the fetus, if that fetus is a girl.

Chapter 2 focuses on the multigenerational effects of in-utero shocks on human capital. Over the last two decades there has been an influx of research on the relationship between early-life shocks and various child and adult outcomes, however much less is known about the intergenerational role of these shocks. This chapter aims to fill the research gap by making two main contributions. The first contribution is to present the first estimates of the effects of an adverse shock experienced by a grandmother during her pregnancy on her grandchildren's outcomes in the context of India. The shocks in focus are negative rainfall shocks that tend to be more common and much more relevant in India, which relies on rainfed agriculture; thus, this study can provide immediate policy relevance compared to some other studies that have leveraged more severe and less frequent shocks (such as famine and influenza pandemic) as exogenous sources of variation.²

According to Almond and Currie (2011), most of the literature in "early origins" relies on reduced form estimation, but it leaves unanswered, the question about which channel – biological or environmental – explains the effect. Thus, the second contribution is to address that by providing new insights on two main types of potential transmission mechanisms: a) genetic and epigenetic inheritance, and b) child home environment. I attempt to disentangle between these two mechanisms by using mediation analysis. I use survey data from the India Human Development Survey (IHDS) to identify mother-child pairs and combine it with historical, district-level rainfall records data for over 100 years. I exploit exogenous variation in rainfall over time and geographical areas and find that grandmother's exposure to negative rainfall shocks during her pregnancy has a statistically significant negative total effect on her grandchildren's cognitive ability z-scores at age 8-11 and on height-for-age z-scores at age 0-5, but not on height-for-age z-scores at age 8-11. Results from the mediation analysis highlight that the key transmission mechanism of the shocks may be the genetic and epigenetic inheritance. This suggests that delayed interventions to alleviate the damage that is already done in-utero might be less effective, but that there is still a possibility to reverse some of the repairable damage if it is targeted in-utero or early-life. Therefore, in

² See, for example, Painter et al. (2008), Stein and Lumey (2000), and Richter and Robling (2016) for evidence from developed countries and Tafere (2017), Li and An (2015), and Fung and Ha (2009) for evidence from developing countries.

light of these results, I shift the focus in Chapters 3 and 4 to emphasise the strategies to redress the health disadvantages caused by negative shocks in early-life – Chapter 3 looks at parental investments, while Chapter 4 explores policy reforms as targeted strategies. Both chapters also explore the results of these strategies differentially by gender of the child.

Chapter 3 complements the findings of my previous chapter by investigating how parents' investment behaviour could help ease a child's health disadvantage that was caused by negative rainfall shocks in-utero. In an attempt to do that, I start by answering the question of whether in a low resource setting like India parents compensate or reinforce the difference in children's health caused by negative rainfall shocks. If a negative shock leads to an adverse impact on a child's health, a reinforcing strategy would compel parents to respond to the negative effects by investing even less in human capital of that child and harming their health further, while a compensatory strategy would mean that they would try to make up for the negative impact of the shock by investing more in the adversely affected child, thereby improving their overall health. More specifically, I focus on parents' response in terms of educational investments. In order to pin down the causal effect, this chapter makes a methodological contribution by applying an instrumental variable method to isolate the exogenous variation in child health endowment. I use the first two rounds of the rural sample of Young Lives survey for the state of Andhra Pradesh in India and district-level monthly rainfall data predating the start of Young Lives sampling process. The state of Andhra Pradesh experienced a drought during the South-West monsoon period of the year 2002 – I exploit fluctuation in the timing and magnitude of this deficit caused by the drought for an exogenous variation in child's health. I find that on average parents tend to invest more in a child with poor health, thereby compensating for some of the disadvantages to their health in early childhood.

Despite the infamously strong son preference in India, studies have not addressed the gender aspect of parental investment responses to shock induced changes in child health. This is particularly important in the case of India where investment decisions of already constrained households could be determined by efficiency motives, where parents tend to direct more household resources toward sons due to higher potential returns on investments in them and end up, sometimes inadvertently, discriminating against daughters. Hence, the main contribution of Chapter 3 is to provide one of the first estimates of the gender-differential response of parental investments to changes in child health due to early life rainfall shocks. Even though parents in rural households show an overall compensatory strategy for both boys and girls, I find that parents follow a genderdiscriminatory investment strategy – they seem to invest in education of a boy more as compared to investment in a girl child, possibly in expectation of higher future returns from investments in a son. These results build on the findings of Chapter 2 and can help us understand ways to reduce the inequality in child outcomes due to negative rainfall shocks by emphasising the need for support to disadvantaged families. The gender differences in investment strategy seem driven by families that are resource constrained. This suggests that access to additional support would mean that parents' otherwise compensatory behaviour would not have to be influenced by gender norms when faced with resource constraints and that the financial support would help them invest in children in a more equitable manner.

Chapter 4 shifts the focus to policy reforms, exploring the effects of an inheritance rights reform on child health. . Women in developing countries have faced a longstanding legal inability to inherit ancestral property and since land is typically acquired through inheritance, women continue to remain "asset-poor" in comparison to men (Bhalotra et al., 2018). Closing this gender gap in inheritance rights presents an opportunity to improve women's economic outcomes and has the potential to be a powerful instrument for their empowerment (Hallward-Driemeier & Hasan, 2012). In an attempt to promote gender equality, five states in India equalised inheritance rights for women with those of men by enacting legislative reforms between 1976 and 1994, while the federal legislation imposed equal rights in all states in 2005. Again, the data are from IHDS and relying on a difference-in-difference strategy, I use staggered implementation of amendments to the Hindu Succession Act (HSA) to discover the impact of women's exposure to the reform on children's height-for-age using a difference-in-difference strategy. Height-for-age is a compound measure of overall childhood health and reflects the dietary history of the child. Only a handful of studies have exploited the staggered implementation of these rights on different aspects of women's status in India and the causal evidence is somewhat mixed and often weakly identified.

Existing literature shows that financial empowerment of women through more control of resources benefits children as mothers have stronger preferences than father to invest in children (Baranov et al., 2017; Bobonis, 2009; Duflo & Udry, 2004; Lundberg & Pollack, 1993; Lundberg et al., 1997; Thomas, 1990; Ward-Batts 2008). Despite the universally recognised direct benefit of women empowerment for their children's outcomes, quantitative evidence on the intergenerational effects of inheritance rights reform remains largely unexplored for India. There is scant literature that looks at the effects of amendments to the HSA reform on educational outcomes of children and offers mixed findings. While Deininger et al. (2019) find a positive effect of mothers' exposure to HSA reform on their children's education attainment, Bose and Das (2017) do not find any effect on children's education outcomes. By looking at the impact of HSA reform on long term health outcomes instead, this chapter not only adds to the scarce existing knowledge, but at the same time also provides one of the first estimates for the effect of the reform on child health outcomes. It additionally explores the effects of the reform on child outcomes by gender and household composition and contributes to explaining the persisting gender discrimination and son preference norms in India. A final contribution of this chapter is to explore women's bargaining power as a potential mechanism for better child outcomes.

The empirical results show a significant improvement in health of children whose mothers were exposed to the HSA reform, but with a substantial gender bias against girls for children whose mothers are less educated. I also find that the enhancement in child health can be explained by mother's improved bargaining power, which I measure by her autonomy in household decisions regarding perinatal health care utilisation and freedom of mobility. The main challenge in this literature is to prove the validity of the differencein-difference identification strategy and I show several falsification tests and robustness checks to support my analysis.

Finally, Chapter 5 concludes the thesis by summarising the findings of each of the three main chapters, including the significance of the results for policy, and by touching upon suggestions and avenues for future research.

Chapter 2

Multi-generational effects of adverse inutero shocks on health and cognitive outcomes

Multi-generational effects of adverse in-utero shocks on health and cognitive outcomes

Abstract

This paper is the first to evaluate whether an adverse shock experienced by a grandmother while pregnant has a negative effect on her grandchildren in the context of India. Using district-by-month-by-year rainfall information matched with the India Household Development Survey, I find that negative rainfall shocks during the grandmother's pregnancy have negative effects on her grandchildren's height-for-age and cognitive ability z-scores. I also explore the mechanisms that can potentially explain these multigenerational effects and find that the adverse effects of rainfall shocks on grandchildren do not decrease once controlled for the mother's education, consumption expenditure and body mass index. These results seem to suggest that the main mechanism of transmission of negative in-utero shocks from grandmothers to grandchildren is through the biological channel – plausibly genetic or epigenetic inheritance. This implies that interventions to remedy for the damage that is already done in-utero might be less effective.

Keywords Multigenerational effects, rainfall shocks, human capital, fetal-origins hypothesis, India

JEL I12, I15, J13, O15, Q54

2.1 Introduction

Biomedical literature shows that prenatal health shocks to animals in one generation lead to adverse health outcomes for several subsequent generations, suggesting that outcomes in any generation may have biological roots in adverse shocks that would have occurred many generations earlier.¹ Do strong multigenerational effects of shocks also exist for humans? Does exposure of a pregnant woman to adverse shocks negatively affect her child as well as her grandchildren? According to the Barker's fetal origins hypothesis in epidemiology, nutritional deficiencies during pregnancy can impair the fetal development with consequences that continue to persist after birth and through adulthood (Barker, 1990, 1995).

This paper addresses two main research questions on multigenerational transmission of in-utero shocks. First, I evaluate whether a negative shock experienced by a grandmother while pregnant has a negative effect on the health and cognitive outcomes of her grandchildren. Second, I explore the mechanisms that can potentially explain the multigenerational effects of these in-utero rainfall shocks. In the following I will refer to the grandmother that was exposed to the shock while pregnant as the first-generation, to the mother that was exposed to the shock while in utero as the second-generation and to the child who was never directly exposed to the shock as the third-generation.

Although an extensive and continually growing literature has taken cue from fetal origins hypothesis and has shown evidence of life-long causal effects of exposure to adverse environments in-utero on child and adult outcomes,² evidence of transmission of the negative effect of in-utero insults across multiple generations is still scant in the economic literature. The few human studies on multigenerational effects tend to consider only severe in-utero shocks, such as famine or influenza pandemic.³ On the contrary, in this paper I evaluate the third-generation effects of less severe adverse in-utero events, rainfall shocks, in a developing country, India. Focusing on such rainfall shocks that are less severe and less rare, I can provide evidence that is more generalizable to the future in

¹ See Drake and Walker (2004) and Drake and Liu (2010) for comprehensive review of biomedical and epidemiological literature.

² See Almond and Currie (2011a, 2011b) and Almond, Currie and Duque (2018) for an extensive and structured review of this literature.

³ Examples of studies for developed countries are Painter et al. (2008) and Stein and Lumey (2000) who examined the Dutch Famine in 1944 to 1945, and Richter and Robling (2016) who looked at the effects of influenza pandemic in 1918-19. Among the very few studies focusing on developing countries there are Tafere (2017) who considered the Ethiopian Famine in 1983-85, and Li and An (2015) and Fung and Ha (2009) who looked at the effect of the Great Chinese Famine in 1959-61.

India and to other developing countries. Adverse in-utero events in developing countries are likely to have a larger effect because of the poorer socioeconomic conditions and fewer public funding for remedial interventions. Because approximately 70% of Indian working population directly or indirectly subsist on agriculture, which is mainly rain-fed, negative rainfall shocks can have adverse consequences in terms of food availability and household income and this can ultimately lead to maternal and fetal malnutrition (Kumar et al., 2014; Shah & Steinberg, 2017).⁴

My first contribution is to provide the first empirical evidence on the effects of in-utero rainfall shocks on the third generation. As in some of previous studies (Maccini & Yang, 2009; Shah & Steinberg, 2017; Krutikova & Lilleør, 2015; Rocha & Soares, 2015; Leight, 2017) I identify the effect of adverse in-utero rainfall shocks by exploiting exogenous variation in rainfall over time and across geographical areas (Indian districts); but contrary to these previous papers I focus on the effect of the number of months the grandmother was exposed to negative rainfall shocks during her pregnancy on her grandchildren. For each month of pregnancy for which the recorded amount of monthly rain in the district where the grandmother lived was below 1.65 standard deviations from its historical monthly mean, i.e. had a 5% probability of being below the historical mean,⁵ I say that the grandmother was exposed to a negative rainfall shock.

My second contribution is to provide new insights on two main types of mechanisms through which in-utero rainfall shocks get transmitted from the first to the third-generation. The rainfall shock when the grandmother (first-generation) was pregnant can cause: (i) genetic and epigenetic changes for the fetus (second-generation) that ultimately can be transmitted directly to the third-generation through genetic and epigenetic inheritance;⁶ (ii) changes in the utero environment that can lead to fetal developmental issues for the mother (second-generation) with potential long term adverse consequences on her health and socioeconomic outcomes, which ultimately can indirectly affect her child (third-generation) through the child home environment. I call

⁴ See Duflo (2003) and Jensen (2000) for evidence confirming that household income does affect the nutritional status of children.

⁵ I define historical mean and standard deviation at district level using monthly records on rainfall over the years 1900 to 2002 available for 384 districts of India.

⁶ Van Den Berg and Pinger (2016) explain how malnutrition can be transmitted across multiple generations and they find that the multigenerational biological effects on health and education outcomes are driven by epigenetic mechanisms. Considering the Dutch Hunger Winter and comparing siblings of the same sex and exposed and non-exposed to the famine while in-utero, Heijmans et al. (2008) find evidence that in-utero famine leads to methylation in the second generation, i.e. to epigenetic marks that can be inherited.

these two mechanisms the genetic and epigenetic inheritance and the child home environment mechanisms.⁷

In order to identify the effects of these two mechanisms, I use mediation analysis to disentangle between the child home environment effect and the genetic and epigenetic inheritance effect on the child's (third-generation) by comparing the effect of in-utero adverse shocks on the child outcome controlling and not controlling for mother's attributes, such as education, that characterise the child home environment. The effect of in-utero shocks net of these mother's characteristics is then interpreted as the genetic and epigenetic direct inheritance effect; while the difference between the total and the net effect, i.e. the difference between the effect without and with controls for mother's characteristics, is interpreted as the effect operating through the child home environment.⁸

By using the Indian Human Development Survey, I am able to consider three main outcomes for the third-generation, which are cognitive skills at age 8-11 and health at age 0-5 and at age 8-11, which I measure considering the height-for-age z-score⁹. I find a statistically significant negative total effect of in-utero rainfall shocks on the thirdgeneration cognitive skills at age 8-11 and on health at age 0-5, but not on health at age 8-11. Results from the mediation analysis indicate that the main mechanism explaining these adverse effects of in-utero rainfall shocks may be the genetic and epigenetic inheritance. This would suggest that mother's ability to provide a good home environment for her children may not be compromised by rainfall shocks to which they were exposed while in-utero, but they may still transmit a negative effect through the genetic and epigenetic inheritance.

⁷ It must be noted, however, that there could be an interaction effect between these two mechanisms (gene-environment interaction) and therefore disentangling the contribution of the two can be quite challenging. To address this issue, in my additional analysis in Section 6, I allow for a heterogeneous effect of in-utero rainfall shocks by different type of child home environment defined as urban vs. rural households, rural landowners vs. no landowners, those living below the poverty line, and those with more or less number of children.

⁸ It must be noted that if there are omitted variables that explain both the child's outcome and mother's characteristics, then there is an issue of endogeneity. For more details on this endogeneity issue see, e.g. Imai, Keele, Tingley, and Yamamoto (2011), Heckman, Pinto, and Savelyev (2013), Heckman and Pinto (2015), Acharya, Blackwell, and Sen (2016), and Aklin and Bayer (2017). However, due to lack of genetic and epigenetic information in survey data, it is almost standard in the few existing studies to use mediation analysis to explore the transmission mechanisms. Some of the previous studies that have used this strategy include Akresh et al. (2017), Tafere (2017), Van Den Berg and Pinger (2016), Richter and Mazumder (2016) and Richter and Robling (2016).

⁹ The z-score measure the height-for-age in standard deviations below or above the reference world mean value of the same age and gender (see de Onis et al., 2006).

Lab experiments on mammal animals that are able to control more thoroughly for the environment of the third-generation can cleanly isolate the effect of genetic and epigenetic inheritance from the effect through a change in the third-generation environment (see Drake & Walker, 2004; and Drake & Liu, 2010). They find that in-utero shocks lead to adverse outcomes for the third-generation even when all third-generation offspring grow in a perfectly identical environment. This evidence supports the presence of strong genetic or epigenetic inheritance effect of in-utero shocks on the third generation. On the contrary, these lab experiments find that the genetic and epigenetic inheritance effect seems to cancel out when considering shocks that occur after pregnancy. Similar to these animal experiments, I find that while in-utero rainfall shocks adversely affect third generation outcomes, shocks after the birth of the mother do not have an adverse consequence for her child (third-generation) outcomes.

The remainder of the paper is organised as follows. Section 2.2 discusses the related literature and where this paper lies among the existing studies. Section 2.3 describes the data used in the analysis and defines the key variables. In Section 2.4, I describe the empirical identification strategy and in Section 2.5 I present the main results. Section 2.6 reports the additional analyses for heterogeneity in my estimates, factors mitigating the effects and mechanisms driving the effects, and finally, section 2.7 concludes.

2.2 Related Literature

There is widespread literature analyzing the effects of physical insults to the pregnant woman on the outcomes of her children during their early years (Pörtner, 2010; Kumar et al., 2014; Almond et al., 2015; Datar et al., 2013; Rocha & Soares, 2015) and even adulthood (Almond et al., 2018; Shah & Steinberg, 2017). In line with how I define generations in this paper, I call this large group of papers as the 'second generation' papers due to their focus on effects on outcomes of the offspring of those mothers who faced the shock during pregnancy. The papers by Almond and Currie (2011b) and Currie and Vogl (2013) provide a more detailed and structured review of literature on second generation effects of the in-utero shocks and the challenges in estimating the effects of different types of shocks.

Existing evidence for mutigenerational impact of health conditions is more common in epidemiological and biomedical literature than in the economic literature.¹⁰ Most insight on persistent, intergenerational effects of prenatal health shocks stems from experiments on animals. Animal studies have shown that if a female mouse is exposed to a shock while pregnant, her fetus as well as the reproductive cells of the fetus will be affected. This means that three generations are always connected – a pregnant mother's female fetus already contains all the eggs she will have in her lifetime. Thus, the impact of multigenerational inheritance can only truly be seen on the third generation – if the grandmother is affected by a shock, the grandchild, though never directly exposed to it, will be indirectly affected as well (Heard & Martienssen, 2014). According to a recent systematic review of this literature, Aiken and Ozanne (2014) finds that out of 48 published animal experiments, 44 found effects on the third generation. These experiments involve a treated group of pregnant animals who are exposed to some form of stress (e.g. under- or malnutrition, or excessive exercise) and a control group with not exposure. Multiple generations of offspring are then observed and their health outcomes are compared between the treatment and control groups. For instance, studies have documented that rats that are malnourished before or during pregnancy produce offspring with smaller brains and reduced cognition, even after the restoration of a normal diet post-birth. More importantly, these effects are not only seen in the immediate offspring, but are present in the next generation as well.¹¹ Stewart, Preece and Sheppard (1975) and Stewart, Sheppard, Preece and Waterlow (1980) followed rats over twelve generations and found negative health effects of in-utero malnourishment over three subsequent generations, even if the offspring received sufficient nutrition after birth. This particularly confirms the importance of uterine period for multigenerational effects.

There are only very few studies which observe multigenerational effects in human populations. The interest in studying the effect of in-utero shocks on child and then adult life outcomes has moved recently toward understanding the multigenerational consequences for children of those who suffered in-utero insults.¹² This shift in interest came about after the research by Painter et al. (2008) that investigated multigenerational

¹⁰ See Drake and Walker (2004) and Drake and Liu (2010) for comprehensive reviews.

¹¹ Recent reviews, of the literature on transgenerational epigenetic inheritance, include Daxinger and Whitelaw (2010), Daxinger and Whitelaw (2012), Grossniklaus (2013), and Heard and Martienssen (2014). ¹² In this review I do not include papers on third generation effects of shocks experienced in phases other than the uterine period. E.g. the effects of shocks in the period around age 9, on children and grandchildren, have been examined by Van den Berg & Pinger (2016).

impact of the Dutch Hunger Winter 1944-45. In the following, I review the few papers in economics that have estimated the multigenerational effect of in-utero shocks.

The literature on multigenerational impact of adverse shocks in utero, so far, has largely focused on three historical events: the Dutch Hunger Winter of 1944 to 1945, the influenza pandemic of 1918 to 1919, and the Chinese Great Famine of 1959 to 1961. The papers that study the multigenerational impact of extreme nutritional deprivation as a result of the *Dutch Hunger Winter of 1944-45* (Painter et al, 2008; Stein & Lumey, 2000), show that children of mothers who were in-utero during the Dutch famine experienced worse health in later life – children of prenatally insulted parents had lower birth weight than those whose parents did not experience the Dutch famine in utero. Jallow (2017) points out a weakness of these papers in terms of a possible confounding effect of World War II that took place around the same time period. For the countries that did take part in the War, able-bodied men were chosen to fight, reducing the remaining pool of men at home that women could marry. Thus, it would be difficult to disentangle the separate effects of Dutch famine from that of the Second World War Additionally, the famine was a result of an embargo by Germany; an embargo is not any random event (Jallow, 2017).

The second group of studies focuses on the multigenerational effect of disease exposure due to the *influenza pandemic of 1918-19*. Richter and Robling (2016) find that the children of those who were exposed to the 1918-1919 influenza pandemic (colloquially known as the Spanish flu) in-utero are prone to having chronic illnesses in adulthood. Furthermore, Richter and Robling (2016) find that exposure to Spanish flu lowers the education attainment of next generation children in Sweden. Jallow (2017) highlights that there is a similar problem with these studies as with the papers studying the Dutch Hunger Winter, due to this pandemic coinciding with the First World War Thus, the resulting effects for countries that took part in the War would be confounded due to poorer gene quality of women's partner and not just the Spanish Flu. On the contrary, studies based on countries that remained neutral during the First World War (Jallow, 2017) – e.g. Taiwan (Lin & Liu, 2014) or Switzerland (Neelsen & Stratmann, 2012) – are unaffected by this bias.

The third group of papers, which studies the multigenerational impact of the *Chinese Great Famine 1959-61,* is not subjected to the same criticism as the above two groups of papers, but studies using famine as an exogenous shock are subjected to criticism because their results cannot be generalized to starvation in normal periods as

people behave differently during famine, e.g. they change their fertility and family formation decisions. Li and An (2015) confirms the results of Fung and Ha (2009), and show that children of those who experienced the Chinese Great Famine of 1959-61 inutero were shorter in height compared to those who did not and the adverse effect of the shock persisted for the third generation children up to age 18.

Moreover, there are few studies that look at other "historical" events as shocks in-utero to study their multigenerational impact. Black et al. (2019) find that Norwegian cohorts exposed to radioactive fallout from nuclear weapon testing during the in-utero period had children with lower cognitive ability. The papers by Caruso (2015) and Caruso and Miller (2015) look at the impact of the great flood of Tanzania in 1993 and the 1970 Ancash earthquake in Peru, respectively. These papers found that children of those mothers who experienced the Tanzanian flood in utero gave birth to children with lower height-for-age z-scores, while children of those mothers who suffered from the Ancash earthquake in Peru had less education. Akresh et al. (2017) study the intergenerational impacts of the 1967-1970 Nigerian Civil War, and find that war exposed mothers (but not fathers) have adverse impacts on child growth, survival and education.

A recent paper by East et al. (2017) looks at multigenerational effects of a positive, policy-driven health intervention during utero and early-life.¹³ They focus on the effect of 1980s US Medicaid Programme exposure during pregnancy and early-life on the next generation's health outcomes at birth. They find strong evidence that health benefits associated with in-utero access to Medicaid Programme extend to children in the next generation in terms of better average birth weight and decreased incidence of very low birth weight. However, they find that these multigenerational results only hold for in-utero exposure to the Programme and not for exposure during later childhood, again proving that the in-utero period is the most critical period for multi-generational analysis.

Finally, there is a large strand of the literature that has looked at the effects of inutero rainfall shocks, but none of these studies has looked at multigenerational impacts. Examples of these papers include Hoddinott and Kinsey (2001), Yamano et al. (2005), Alderman et al. (2006), Maccini and Yang (2009), Neelsen and Stratmann (2011), Ampaabeng and Tan (2013), Dercon and Porter (2014), Bertoni (2015) and Abiona

¹³ Hoynes, Schanzenbach, and Almond (2016) provide useful evidence of a positive intervention as an inutero and early-life shock and find that access to the U.S. Food Stamp program led to a large reduction in the incidence of "metabolic syndrome" and, among women, an increase in economic self-sufficiency. However, it does not look at the multigenerational effect of the programme.

(2017). Before moving on with the second generation analysis, it is important to understand the channels through which rainfall shocks could have an effect on health of children of the mother exposed to these shocks during her pregnancy. The two main channels that have been identified in literature include the income effect and price effect. Income effect suggests that a rainfall shock is likely to cause a reduction in availability of food as a result of crop failure, negatively affecting the source of income of those households who subsist on agricultural output. Thus, in a financially constrained household, if a pregnant mother's nutrition is hampered due to food shortage, it could have a severe effect on her unborn child's health during the developmental stage in gestation. The price effect entails that crop failure caused by a rainfall shock will reduce the supply of agricultural output, consequently increasing prices of staple foods. The crops that are sown in rainy season are called 'Kharif' crops (also known as the monsoon crop) and include staples like rice, maize and cotton. When staple crops become more expensive due to supply shortage, it could again result in the pregnant woman not being able to afford them in order to fulfil her nutritional requirements, causing an adverse effect on health of her unborn child. In addition, financial constraints and the associated stress due to lower incomes or higher prices could also affect an expectant mother's behaviour as she may not be able to obtain health inputs like medicines, vaccinations or medical care in a timely manner, impairing her child's health. Ahmed (2016), Kumar et al. (2015), Shah and Steinberg (2013), and Burgess et al. (2011) provide a detailed discussion on the potential pathways through which rainfall affects child health.

A related concern could be the timing of the shock and how the nutritional effects described above are likely to vary across the key periods around a child's birth. More specifically, within the annual cycle of wet or dry seasons, what periods of rainfall will have the most impact on crop yields, potentially harming a child's health. Particularly in case of Kharif crops like rice, it is normally planted about one month after the start of the wet season, with the next three to four months representing the grow-out phase, followed by harvest. There should be plentiful food around harvest season and in the months following it, but scarce at other times, particularly when stores are depleted prior to the next harvest. The effect of this cycle has been well-documented in development literature and is referred to as the phenomenon of the hungry season – where food shortages in the months leading up to harvest can have quite sizeable health effects (Moore et al., 1997). The period of gestation, or the nine months prior to a child's birth, is where development of the foetus is solely dependent on the mother's health. After

birth infants tend to be exclusively breast-fed at first, with solids introduced from about four months of age. Empirical evidence suggests the critical stages are actually closer to birth – gestation and the first few months after birth – seeing no significant effects beyond six months. This justifies my hypothesis to look at the effects of rainfall shocks in-utero.

2.3 Data

Data and sample selection

In this analysis, I use household-level survey data and historical rainfall records to create a unique panel data set. The household data comes from the nationally representative India Human Development Survey (IHDS) of 2004-05, which is jointly conducted by the National Council of Applied Economic Research and the University of Maryland (Desai &Vanneman, 2011). The data covers 41,554 households located in 384 districts of India. I use this multi-topic survey to identify mother-child pairs, restricting the sample to women of reproductive age i.e. 15 to 49 years and their children between 0 and 11 years in 326 districts of 23 states in India.

The design of IHDS leads to a natural choice of what constitutes the three generations. The second generation includes mothers born between 1955 and 1990, while the third generation includes their children. The youngest mother in the sample is 15 years old upon being observed in 2004-05. Overall, the third generation includes children between 0 and 11 years, born to the mothers in the second generation. I use two main child's age-specific estimation samples for the two outcomes that I am studying: (a) Due to the importance of the first five years in shaping a child's long-term outcomes, my first sample for health outcomes includes children 0 to 5 years of age; (b) IHDS contains information on cognitive ability only for children 8-11, which is what makes my second sample. Anthropometric information is also available for this group, making the sample of children 8 to 11 years old the only age group for which information on both, cognitive and health outcomes, is available.¹⁴

For rainfall data, I use monthly, district-level historical records (available from year 1900 to 2002) from the India Water Portal to create a district-by-month of rainfall

¹⁴ Data on cognitive ability of children is not collected for the sample of children 0 to 5 years old.

records. I use the full time series of rain fall data to compute the district-specific monthly average and standard deviation.

The empirical strategy I undertake requires linking the rainfall data during the time the mother (second generation) was in utero with the third generation, child-level observations from IHDS. I am able to match these two datasets as IHDS allows me to use the exact date of birth and the district of residence of the mother to identify the district-level rainfall during her perinatal period.

Rainfall measure: In-utero shock exposure

In the rainfall data, the amount of rainfall is recorded monthly as millimetre per acre for each of the districts. I use the following steps to create district-level extreme rainfall deficit¹⁶:

I start by calculating the rainfall z-score (rain-z) in order to determine the drier and wetter months in the record (Asfaw et al., 2018; Agnew & Chappel, 1999; Woldeamlak & Conway, 2007; Eiste et al., 2012; Gebre et al., 2013). The rain-z represents z-scores of rainfall in district d and month t and is computed as:

$$rain - z_{dt} = \frac{(X_{dt} - \bar{X}_d)}{\sigma_d}$$
(2.1)

where X_{dt} is the observed rainfall in district d month t, and \overline{X}_d and σ_d are the historical mean and standard deviation for the district d computed using the monthly rainfall in district d from 1900 to 2002.

Then for each month, I create a measure for rainfall deficit. This is simply a dummy that equals one if the rain z-score falls below -1.65 for that month. Agnew and Chappel (1999) defines severity classes of rainfall, with the rain z-score < -1.65 representing extreme drought (or rainfall deficit). Assuming that the rain z-score be distributed as a Normal with mean 0 and variance 1, the probability of a rainfall deficit in a district is equal to 5 per cent.

I measure in-utero exposure to an extreme rainfall deficit or simply what I define as "in-utero shock exposure" as the number of months that a rainfall deficit occurs in the 9 months prior to the individual's birth. For instance, if a mother was born in November,

¹⁶ As in Viste et al. (2013), in this study, the term rainfall deficit is reserved for rainfall deficits that are outside of the normal range. It does not take into account that some districts may have generally dry or frequently varying conditions.

then in-utero rainfall shock exposure is computed as the number of months she was exposed to rainfall deficit between February and October.

Using information from IHDS on mother's date of birth and district of residence, I link the IHDS data with the constructed rainfall data and measure the in-utero shock exposure for all mothers born between 1955 and 1990. This exposure can theoretically range from 0 months, for mothers who were exposed to no rainfall shock while in utero, to 9 months, for mothers who were exposed to a rainfall shock in each of the gestation months. In my sample, the maximum number of months of in-utero negative rainfall shock is actually only 3. I also create variables for the rainfall shock exposure in the year after birth, in years when the child is aged between 1 and 5 and also between years 6 and 9 by simply adding up the number of months that the mother was exposed to the rainfall shocks in her first year of life, in years 1 to 5 and in years 6 to 9, respectively.¹⁷

Outcome variables

I estimate the impact of exposure to extreme rainfall shock on outcomes of third generation children whose mothers (second generation) were exposed to shocks in utero. These child-level outcomes are measured in terms of health and cognitive ability.

Health outcomes are measured by Height-for-Age z-score (HAZ) for two samples, namely the sample of children 0 to 5 as well as 8 to 11 years of age. The choice of HAZ as a measure of child health is due to the established literature showing that it is a good summary indicator of childhood growth, nutrition and environmental factors (Guven & Lee, 2013; Case & Paxson, 2008a, 2008b). A child whose HAZ is below minus two standard deviations is considered stunted and chronically malnourished. I calculate HAZ for these children using the World Health Organisation's (WHO) 2006 growth standards (de Onis et al., 2006). While absolute height may indicate child growth, it may not mean anything in overall sample; however, the z-scores provide information on how the anthropometric characteristic of a child (in this case, height-for-age) compares to the WHO reference population of the same age and gender. So a z-score less than -2 means that a child's height-for-age is 2 standard deviations below the median in reference population.

In order to create the cognitive ability z-score, I first start by creating a standardised composite index using the actual test scores that IHDS reports for math,

¹⁷ I also check for serial correlation of rainfall shocks and my results (not mentioned here) show lack of it, as proven by p-values of the Breusch-Godfrey test (Bertelli, 2005).

reading and writing abilities.¹⁸ I then calculate the cognitive ability z-score by same age and gender for the sample of children 8-11 years, because IHDS only administered these learning tests for this particular age sample. These test scores are administered and gathered by a third party and are more reliable than measures of skills self-assessed by children themselves or their parents.

Main control variables

I include basic controls variables for the child (third generation) and second generation control variables as mediators in some specifications. Basic controls for the child include age in months and a dummy for male child. Second generation mothers' outcomes that I add as additional covariates in the third generation regressions include: mother's years of education, her monthly consumption expenditure and her Body Mass Index z-score (BMI).

A mother's completed education level, which also represents the financial resources spent on her, is one of the potentially most important channels, reflecting her cognitive ability, parenting skill, social class and earnings potential, all of which are essential for health and cognitive outcomes of her children (Van Den Berg & Pinger, 2016). IHDS reports the consumption expenditure to measure a household's current economic level. The consumption expenditure was constructed using household's reports of quantity consumed and price of both market and home-produced consumption items. Mother's BMI (kg/m^2) is a continuous variable which is constructed using height and weight information for samples of children 0 to 5 years and between 8 and 11 years. It is calculated as weight in kilograms divided by height in metres squared. Literature shows evidence of the association between mother's BMI and child's growth and development (Nie et al., 2016). BMI of the mother is indicative of her long-term and cumulative nutrition history, which as a result also determines the health of her fetus for the third generation child. BMI is interpreted for all adults in the same way, regardless of their age, height or gender. Typically, a BMI under 18 is considered underweight and possibly malnourished. There are approximately 3 per cent mothers in the IHDS sample with

¹⁸ The math test scores are recorded in the following discrete variables: cannot count (=0), number (=1), subtraction (=2), and division (=3). The reading test scores are recorded in five discrete variables: cannot read (=0), letter (=1), word (=2), paragraph (=3), and story (=4). Finally, the writing test scores are recorded in two discrete variables: cannot write (=0) and write with two or less mistakes (=1).

missing observations for anthropometric data; therefore, I use Predictive Mean Matching (PMM) to perform multiple imputation for 3 per cent missing BMI data.¹⁹

Descriptive statistics

Table 2.1 reports descriptive statistics of all the variables of interest for the analysed samples. Panel A shows descriptive statistics for sample of 12,696 children (born to 9,122 mothers) aged 0 to 5 years for whom I only observe the HAZ outcomes as IHDS only collects learning outcomes for children aged 8 to 11 years. Panel B shows descriptive statistics for sample of 9,278 children (born to 7,550 mothers) aged 8 to 11 years for whom I observe both the cognitive skills and HAZ outcomes. Column (1) shows the number of observations, column (2) shows the sample mean, column (3) shows standard deviation of the sample, while columns (4) and (5) show the minimum and maximum values, respectively, for each of the variables.

Panel A of Table 2.1 shows that the sample of children 0 to 5 years of age have a mean HAZ of -1.330 standard deviations; a negative HAZ suggests that on average, children are malnourished compared to WHO's reference population of the same age and gender. Additional statistics (not shown here) show that approximately 39 per cent of the sample children are stunted i.e. their HAZ is below minus two standard deviations. The rainfall shock was faced by the maternal grandmother before and during pregnancy and by the mother herself after her birth. Maternal grandmothers faced approximately 0.04 months of exposure to extreme rainfall deficit during their pregnancy. In the sample, any woman faced a maximum of 3 months of extreme rainfall deficit during the 9 months of when she was in gestation herself. After the mother was born, she experienced a maximum of 3 months, 5 months and 4 months in the first year after her birth, in years 1 to 5 and in 6 to 10, respectively. Basic child controls show that the average age for grandchildren is 2.8 years and approximately 53 per cent of them are boys. Controls for maternal characteristics show that the average completed education of mothers is 4 years. Their per capita monthly consumption is about 651 Indian Rupees on average.²⁰ Mean BMI of mothers of this group of children is 20.4 kg/m^2 .

¹⁹ For this prediction, I use all variables in the analysis model that are related to the mother and provide information about the true value of the missing data for mother's BMI i.e. her completed education of mothers and her per capita monthly consumption, as well as the dependent variable i.e. the in-utero rainfall shock exposure.

²⁰ 1 Indian Rupee (INR) = 0.0138 US Dollar (USD) and 0.0105 British Pound (GBP) (accessed from <u>http://www.xe.com/currencyconverter/convert/?Amount=1&From=INR&To=GBP</u> on Sept 18, 2017).

VARIABLES	(1)	(2)	(3)	(4)
VARIADLES	Mean	Std. Dev.	Min.	Max.
Panel A: Sample of children 0-5 years				
Outcome variables:				
Height-for-age z-score (HAZ)	-1.330	2.154	-4.990	4.993
Main independent variable: No. of months of exposure to negative rainfall shock:				
in-utero period	$0.037 \\ 0.042$	$0.193 \\ 0.213$	0	3 3
in the year after birth in the years $1 \text{ to } 5$	0.042	$0.213 \\ 0.497$	0 0	ы 5
in the years 6 to 9	0.240	0.497 0.545	0	4
Basic child controls:				
Dummy for male	0.530	0.499	0	1
Age (in months)	33.930	18.748	0	60
Maternal characteristics:				
Mother's completed years of education	4.060	4.645	0	15
Monthly consumption expenditure in Indian Rupees (2004)	650.924	551.135	0	12020
Mother's BMI z-score	-0.134	0.964	-2.617	5.514
Mother's BMI	20.382	3.162	12.782	39.232
No. of children born to the mother	2.850	1.615	0	13
No. of mothers: 9,122 No. of children: 12,696				
Panel B: Sample of children 8-11 years				
Outcome variables:				
Height-for-age z-score (HAZ)	-1.568	1.450	-4.995	4.950
Cognitive ability z-score	0.012	1.000	-2.606	3.250
Main independent variable: No. of months of	-	-		
in-utero period	0.049	0.225	0	3
in the year after birth in the years 1 to 5	$\begin{array}{c} 0.058\\ 0.254\end{array}$	$\begin{array}{c} 0.243 \\ 0.544 \end{array}$	0 0	3 4
in the years 6 to 9	0.189	$0.344 \\ 0.460$	0	4
Basic child controls:				
Dummy for male	0.528	0.499	0	1
Age (in months)	113.565	12.822	96	132
Maternal characteristics:				
Mother's completed years of education	3.399	4.355	0	15
Monthly consumption expenditure in Indian Rupees (2004)	717.041	603.993	0	13119
Mother's BMI z-score	-0.0995	0.980	-2.349	4.798
Mother's BMI	21.002	3.513	13.047	38.409
No. of children born to the mother	3.504	1.6445	0	13
No. of mothers: 7,550				
No. of children: 9,278				

Table 2.1: Descriptive Statistics

Panel B shows that for the sample of third generation children aged 8 to 11 years, the cognitive ability z-score is negative (-0.012 standard deviations) and their HAZ is also negative (-1.568 standard deviations) suggesting that children on average are malnourished compared to WHO's reference population of the same age and gender. These children have a worse average height-for-age compared to the 0 to 5 sample. On average, mothers of these children were exposed to slightly more months of extreme rainfall deficit in utero (0.05 months) compared to mother of children 0-5 years; but similar to the 0-5 sample, any maternal grandmother faced a maximum of 3 months of extreme rainfall deficit during her pregnancy. After she gave birth to the mother, the mother herself experienced a maximum of 3 months of the extreme negative rainfall shock till her first birth, 4 months in the years 1 to 5 and 4 months in the years 6 to 10. Basic child controls show that the grandchildren are around 9 years old with approximately 52.8 per cent of them being males. Controls for maternal characteristics show that the average completed years of education of mothers is 4. On average, mothers of children aged 8 to 11 years are 34 years old (born in year 1970). The per capita monthly consumption is about 717 Indian Rupees on average. Mean BMI of mothers of this group of children is 21.1 kg/m².²¹

2.4 Empirical Strategy

I evaluate whether there is a causal effect of a mother's exposure to an extreme negative rainfall shock when she was in-utero on outcomes of her children. The validity of my estimation rests on the assumption that the extreme rainfall shock is a historical, quasirandom event and that its occurrence is beyond the control of the pregnant woman, making it an exogenous shock that is uncorrelated with any omitted determinants of laterlife outcomes for the next generations.

2.4.1 First to third generation effects

To assess whether the shock that a grandmother faces during her pregnancy has a long term impact on outcomes of her grandchild who was never directly exposed to the shock, I estimate the following linear equation, where Y is the child health or cognitive outcome:

²¹ A BMI between 18.5 and 24.9 is considered normal and represents satisfactory nutritional status of adults.

$$Y_{idmt} = \alpha_1 + \alpha_2 S_{dmt} + \alpha_3 X_{idmt} + \eta_d + \mu_m + \gamma_t + \varepsilon_{idmt}$$
(2.2)

The subscripts i, d, m, and t index a grandchild i, born to a mother who was born in month m and year t and resident in district d.

I estimate separate models for two different outcomes (Y_{idmt}) , which are: (a) child's cognitive ability z-score and (b) child's height-for-age z-score. The independent variable of interest (S_{dmt}) is the number of months of exposure to negative rainfall shock in-utero, i.e. during the 9 months prior to a mother's birth when the mother is still in the grandmother's fetus. The coefficient of interest α_2 measures the causal total impact of negative rainfall shock exposure, when the second generation mother was in-utero, on her child's health and cognitive ability outcomes. X_{idsmt} is a vector which includes controls for child's gender and age.

The identification strategy relies on rainfall shock variation across two dimensions: *spatial* (variation across 384 different districts of residence of the mother) and *temporal* (birth cohort).²² Specifically, IHDS data allows me to use information on a second generation mother's district of residence and her date of birth to calculate the number of months the mother was exposed to rainfall shocks while she was in-utero.

To make sure that the variation in rainfall shocks across time and district is exogenous I also include fixed effects for the mother's district of residence (η_d), which control for any unobservable time-invariant determinants of outcomes that differ across districts. These could include changes in the geographical conditions or any districtspecific risks of diseases during pregnancy (Carillo, 2018). I also include mother's monthof-birth (μ_m) and year-of-birth fixed effects (γ_t) to capture any variation that may be cohort/birth month and year specific; thus, it will account for any time-variant, but district-invariant characteristics such as seasonal fluctuations, macroeconomic conditions or national policies. ε_{idmt} denotes a random, idiosyncratic error term – I still consider the correlation in the error across time for the district. I assume the error components to be identically distributed across districts, but correlated within them; hence, all standard errors are clustered by mother's district of residence to account for any serial correlation. I include two-way clustering of all standard errors at the *district* and at the *household* level. Standard errors are also clustered at the household level because they may be correlated

²² Examples of papers that have used a similar spatial and temporal variation in rainfall shocks are Carrillo (2018), East et al. (2017) and Akresh et al. (2014).

within the household. This is because there are some households (approximately 25 per cent) that have more than one sample child born to the mother who was exposed to the in-utero shock.

Even though my main concern is to understand what happens in-utero, through my benchmark estimation in Equation (2.2), but I will also compare this basic specification with a specification where this shock is experienced in various phases after pregnancy to see potentially if some channels of transmission are stronger in the utero phase.

2.4.2 Transmission mechanisms – mediated third generation effects

Few of the studies that look at in-utero shocks, have tried to distinguish between biological and environmental channels by re-estimating the third generation effects via inclusion of maternal adult outcomes as regressors (see for instance, Akresh et al., 2017; Richter & Robling, 2016). In line with the specification used in these studies, I re-estimate the third generation effects of children's health and cognitive outcome regressions by including maternal characteristics as regressors in order to net out the effect of human capital of the mother from the total effect (α_2) in Equation (2.2):

$$Y_{idmt} = \delta_0 + \delta_N S_{idmt} + \delta_E E duc_{idmt} + \delta_C Cons_{idmt} + \delta_B B M I_{idmt} + \delta_1 X_{idmt} + \eta_d + \mu_m + \gamma_t + \varepsilon_{idmt}$$
(2.3)

 $Educ_{idmt}$, $Cons_{idmt}$ and BMI_{idmt} represent the three maternal characteristics: mother's completed years of education, her economic status that is measured by household monthly per capita consumption expenditure and her health that is measured by z-score of her Body Mass Index (BMI). δ_N is the effect of the shock that is netted out of the maternal human capital. All other right hand side controls and their subscripts remain the same as in Equations (2.2).

To capture the mediated effects of the shocks and explore the mechanisms of transmission, I approach this part of the analysis by re-estimating two separate models for child's cognitive and health outcomes(Y_{idmt}), adding the mediating factors in three steps: initially I only control for mother's education, then her monthly consumption

expenditure and then I also add her BMI z-score. These maternal controls are added as regressors in steps to look at the role of each of them separately in grandmother-to-grandchild shock transmission.

Maternal adult outcomes serve as inputs in child outcome equations because they determine the resource-environment available for development of the child – a prenatally insulted mother could possibly have lower educational attainment, lower earnings and poorer reproductive health, which could indirectly also affect her fetus, parenting behaviour and the human capital investments on her children resulting in poorer outcomes for them (Richter & Robling, 2016). As all three of them provide the environment the third generation child grows up in, I can say that by adding the mediators I am potentially controlling for the effects of the socioeconomic environment. If the third generational effect is purely driven by genetic/epigenetic effects, inclusion of maternal controls as mediators should not affect the main estimates. Thus, by controlling for the maternal characteristics more broadly, I intend to cautiously comment on the role of genetic/epigenetic and socioeconomic environment as potential mechanisms.

A potential concern regarding a mediated analysis is that there is a risk of these regressors being bad mediators as they can be endogenous – there could be unobserved factors explaining both, maternal adult outcomes and her child's human capital (see Heckman & Pinto, 2013). Despite including maternal characteristics as mediators, I cannot completely rule out other multigenerational mechanisms that I am unable to account for; thus, this part of the analysis should be looked at with caution.

My analysis will be valid under two main assumptions. First, I am assuming that any in-utero effect of the shock that is not genetic or epigenetic passes down the next generation through an environmental channel, by having a mediated effect on mother's long term characteristics. Second, the effect of environment on the child is captured by the three mother's characteristics that I am controlling for in my mediated analysis, namely her education, consumption expenditure and her BMI. The second assumption may appear a bit crude, but it is in line with what other studies have done. For instance, in order to distinguish between direct and indirect effects, Richter and Robling (2016) controls for education and earnings.
VARIABLES	(1)	(2)	(3)
	HAZ	HAZ	Cog. Ability
	0-5	8-11	8-11
Maternal grandmother shock in-utero	-0.197^{**} (0.095)	-0.004 (0.076)	-0.097^{**} (0.045)
Child' sex (male)	$\begin{array}{c} 0.092^{***} \\ (0.035) \end{array}$	$\begin{array}{c} 0.086^{***} \\ (0.028) \end{array}$	-0.007 (0.024)
Child's age (in years)	-0.030***	-0.005^{***}	-0.0005
	(0.002)	(0.001)	(0.001)
Constant	-0.358^{***} (0.047)	-1.011^{***} (0.143)	$\begin{array}{c} 0.079 \ (0.097) \end{array}$
Fixed effects	Yes	Yes	Yes
R-squared	0.133	0.137	0.171
Observations	12,696	9,278	9,278

Table 2.2: Third generation effects of exposure to negative rainfall shocks in-utero

Notes: Standard errors in parentheses clustered two-way at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for: mother's year of birth, month of birth, and district of residence.

2.5 Main Empirical Results

This section presents estimates of the multigenerational effects of in-utero exposure to negative rainfall shocks. Along with my baseline model (2.2), I present specifications which control for maternal outcomes to show their potential role as mediators as in equation (2.3).

2.5.1 Third generation effects: grandchild's outcomes

In Table 2.2, I show the main estimation results for the baseline specification outlined in equation (2.2). The columns show effects of grandmother's in-utero exposure to the extreme negative rainfall shock on two dimensions of her grandchild's human capital: health and cognitive ability. The first two columns show results for grandchildren's health as measured by their height-for-age z-score (HAZ). Anthropometric data is available for a sample of grandchildren 0-5 and 8-11, the results for both of which I separately present in the first two columns. The third column shows estimates for cognitive ability z-scores for grandchildren 8-11 years old.²³ Standard errors in all columns are clustered two-ways at the district and household level.

²³ I only have cognitive ability data for a sample of children 8 to 11 years old as IHDS only gathered learning test scores for this age range.

In case of health, the period below age five is considered a critical one in terms of predicting adult human capital and economic outcomes for individuals. Any health insults in this period are considered largely irreversible and have long term consequences. Column 1 in Table 2.2 shows that for those below age 5, the coefficient for maternal grandmother's exposure to shock during pregnancy is negative and statistically significant at 5 per cent level of significance. Every additional month of exposure to a negative rainfall deficit by the grandmother while she was pregnant, leads to a reduction in her grandchild's HAZ by 19.7 per cent of a standard deviation. This direct, causal effect of the in-utero shock on grandchild's health seems to disappear in column 2 for children 8 to 11 years old. This could be attributable to the fact that as children age, they catch up in terms of physical health as a result of better nutritional intake. This is not inconceivable because "catch-up" growth is possible in children if the factors responsible for impaired growth are eradicated.²⁴ However, we still cannot disregard the fact that even if they catch up by age 8, the bad health at age 0-5, as seen in column 1, may still have long-term impact in terms of adverse adult health, cognitive and economic outcomes.²⁵

In the third column of Table 2.2, I present results for grandmother's in-utero shock exposure on her grandchild's cognitive ability outcome for the sample aged 8 to 11. Similar to health outcomes, there is a negative and statistically significant, causal effect of the in-utero shock on grandchild's cognitive ability z-score. Every additional month of exposure to a negative rainfall shock in-utero reduces cognitive ability z-score of a third generation child by approximately 10 per cent of a standard deviation. Following the literature in Almond et al. (2018), one could possibly comment that if children's cognitive ability suffers during this school going age bracket, they may continue to perform poorly in school and hence, may even suffer from extensive negative effects in terms of their long-term socioeconomic outcomes.

Overall, even though we see a negative relationship between a rainfall deficit shock in utero and the health and cognitive development of the grandchildren of the woman affected by the shock, there could be a potential concern with this analysis if there was possibly a time-lag in the effect of the shock experienced in-utero. I empirically

²⁴ For instance, Graham and Adrianzen (1972) find that severely-malnourished infants who after birth have a good environment reach the 25th US centile for height by approximately seven years of age. Early-life literature also shows that the effect of shock experienced by children in early childhood largely "fades out" by age eight or nine (also referred to as the "missing middle"), before they resurface in adulthood (Almond and Currie, 2011a; Almond et al., 2018).

²⁵ An exhaustive review of literature by Almond et al. (2018) shows that human capital is produced very early in life. Even relatively mild shocks seem to have substantive negative effects on adult outcomes.

test the effect of time-lag by looking at the effect of the shock that the grandmother experienced in the pre-pregnancy phase i.e. in the 12 months prior to she conceived on her grandchildren's health and cognitive ability outcome. Results in Appendix Table A9 seem to convince that there is no effect of the pre-pregnancy shocks experienced by the grandmother on her grandchildren's health. I do not find any empirical evidence for it, strengthening my existing analysis.

2.5.2 Exploratory analysis of potential channels

Multigenerational effects of in-utero shocks can have an effect through direct biological (i.e. genetic and epigenetic) changes and through changes in a child's home environment. In order to explore whether biological or environmental channels are responsible for effects of these in-utero shocks, some of the studies that look at extreme shocks, have tried to re-estimate the third generation effects by including maternal adult outcomes as regressors (see for instance, Akresh et al., 2017; Richter & Robling, 2016) to characterise the home environment of the child. In line with the specification used in some studies, I estimate equation (2.3) which has maternal characteristics as additional explanatory variables. As I only found a causal effect of grandmother's in-utero shock on child's outcomes in the first and third column of Table 2.2, i.e. for HAZ at age 0-5 and cognitive ability at age 8-11, I focus only on these two outcomes for this analysis.

Panel A of Table 2.3 shows results for child's HAZ at age 0-5, while Panel B shows results for the child's cognitive skills when 8-11 years old. Columns (1) in Table 2.3 Panel A reports again the results of Table 2.2 for the child's HAZ at 0-5 with no controls for mother's characteristics. Column (2) reports the results when including maternal education as explanatory variable, column (3) additionally controls for the mother's monthly consumption expenditure and finally column (4) additionally controls for the mother's BMI z-score. Columns (5) to (8) in Panel B follow the same type of model specification as in columns (1) to (4), but considering as dependent variable the child's cognitive skills at 8-11 rather the HAZ at 0-5. By comparing the results across columns, I explore the effect of including each of the three mother's characteristics as a potential channel of shock transmission form the first generation (grandmother) to the third generation (child).

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Moving from column 1 to column 2, when I add mother's number of years of education, the coefficient for in-utero shock remains unchanged and statistically significant at 5 per cent level of significance, suggesting that maternal education is not a channel that explains the third generational effect of the in-utero shock. Further, moving from column 2 to column 3 in Table 2.3, when I add mother's monthly consumption expenditure, the negative effect of the in-utero shock slightly attenuates from 19.7 to 19.2 per cent of a standard deviation, but still remains statistically significant at 5 per cent level of significance. In column 4, I explore whether a mother's BMI z-score (an indicator of her own general health) could be explaining the third-generational linkages between the shock she experienced while she is in-utero and HAZ outcomes of her children. After the mother's BMI z-score is included, the coefficient for maternal in-utero shock hardly moves, it is 10.1 and still statistically significant at 5 per cent level of significance.²⁶ This means that none of these mediators – mother's characteristics – can be considered as a main channel in explaining the third generational effect of the negative in-utero shock on the child's health.

Similarly, I find that the negative effect of in-utero shocks on the child's cognitive skills is not mediated by mother's education, monthly consumption expenditure and BMI z-score (see Table 2.3 panel B).

Theoretically, after a mother is born, her genes do not change and any shock that she experiences post-birth should not affect human capital of her children, unless it is through her own human capital that she accumulates over her lifecycle and which defines the environment for development of her child. Therefore, even though all of these three mediating factors affect the environment of development of the child, the fact that their inclusion does not affect the main estimates suggests that none of these seem to be conclusively explaining the effect of in-utero shock on third generation outcomes. Nonetheless, as mentioned earlier, this analysis must be looked at cautiously as there is a risk of these mediators being bad (endogenous) controls, but this is the best that can be done given the data limitations.

²⁶ Even though the 'reghdfe' command in STATA does not allow me to test for whether the coefficients for in-utero shock in columns 1 and 4 (and later, 5 and 8) are statistically different from each other, the strong overlap in standard errors clearly suggest that the coefficients are not different from each other.

	Panel A: HAZ (0-5)			Panel B: Cog. Ability (8-11)				
VARIABLES	(1) No mother's controls	(2) controlling for mother's educ.	(3) + mother's cons. expenditure	(4) + mother's BMI z-score	(5) No mother's controls	(6) controlling for mother's educ.	(7) + mother's cons. expenditure	(8) + mother's BMI z-score
Maternal grandmother shock in-utero	-0.197^{**} (0.095)	-0.197^{**} (0.095)	-0.192^{**} (0.095)	-0.191^{**} (0.094)	-0.097^{**} (0.045)	-0.090^{**} (0.041)	-0.084^{**} (0.041)	-0.080^{*} (0.041)
Child' sex (male)	$\begin{array}{c} 0.092^{***} \\ (0.035) \end{array}$	$\begin{array}{c} 0.088^{**} \ (0.035) \end{array}$	$\begin{array}{c} 0.083^{**} \\ (0.035) \end{array}$	$\begin{array}{c} 0.085^{**} \ (0.035) \end{array}$	-0.007 (0.024)	-0.006 (0.023)	-0.009 (0.023)	-0.009 (0.023)
Child's age (in years)	-0.030*** (0.001)	-0.029^{***} (0.001)	-0.029*** (0.001)	-0.030^{***} (0.001)	-0.001 (0.001)	$7.65e-05 \\ (0.001)$	-0.0002 (0.0001)	-0.0001 (0.001)
Mother's education		$\begin{array}{c} 0.051^{***} \\ (0.005) \end{array}$	$\begin{array}{c} 0.042^{***} \\ (0.005) \end{array}$	$\begin{array}{c} 0.039^{***} \\ (0.005) \end{array}$		0.076^{***} (0.003)	$\begin{array}{c} 0.067^{***} \\ (0.003) \end{array}$	$\begin{array}{c} 0.065^{***} \\ (0.003) \end{array}$
Mother's consumption expenditure			0.0002^{***} (4.39e-05)	0.0002^{***} (4.31e-05)			0.0002^{***} (2.36e-05)	0.0002^{***} (2.30e-05)
Mother's BMI z-score				0.087^{***} (0.022)				0.052^{***} (0.012)
Constant	-0.358^{***} (0.047)	-0.579^{***} (0.050)	-0.676^{***} (0.055)	-0.643^{***} (0.055)	$\begin{array}{c} 0.079 \ (0.097) \end{array}$	-0.248^{***} (0.090)	-0.317^{***} (0.090)	-0.303^{***} (0.090)
Fixed effects R-squared Observations	Yes 0.133 12,696	Yes 0.141 12,696	Yes 0.143 12,696	Yes - 12,696	Yes 0.171 9,278	Yes 0.256 9,278	Yes 0.264 9,278	Yes - 9,278

Table 2.3: Third generation effects of exposure to negative rainfall shocks, controlling for maternal characteristics

Notes: Standard errors in parentheses clustered at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects include: mother's year of birth, month of birth, and district of residence.

Is biological channel an explanation?

For the effect to be purely driven by biological effects, the inclusion of maternal characteristics should not significantly affect the main estimates. In line with this hypothesis, the main observation from the analysis so far has been that after netting out the effect of the mediators, the main effect only slightly attenuates in magnitude, but still survives. This suggests that the explanation for what remains of the total effect, is maybe more genetic and epigenetic. The presence of these biological effects is supported by evidence from lab experiments on animals where they are able to fully isolate direct genetic and epigenetic inheritance effects from indirect effects through changes in socioeconomic environment (Drake & Liu, 2010).

By being able to put all third generation offspring in exactly the same environment, biological evidence shows that exposure to shocks in-utero has negative effects that are transmitted epigenetically, beyond the treated generation, for at least three generations.²⁷ An explanation for this biological pattern is that the precursors of the ovaries in women and sperm cells in men are already present in utero, and therefore any insults during the utero phase will also affect the germ cells, which will eventually produce the next generation. It potentially reassures the importance of gene development during the time a mother is in-utero and its impact on her children (third generation). However, it still does not completely eliminate the possibility of socioeconomic environment interacting with epigenetic or other genetic channels.

Biological evidence from these lab experiments on animals further finds that the genetic and epigenetic multigenerational effect seems to be the strongest for shocks during pregnancy.²⁸ In order to put that hypothesis to test, I include effects of exposures to negative rainfall shock during various phases around birth on health and cognitive outcomes. These phases include: a) the shock that the maternal grandmother faced while she was pregnant i.e. in-utero shock; b) maternal shock from birth till age 1; c) shock faced by the mother between ages 1 and 5; and d) shock faced by the mother from year 6 to 9. In Table 2.4, I show separate effects of each of these phases in each of the four columns. The last column shows results for estimation when including all four phases of shock exposure as explanatory variables.

²⁷ For evidence from experiments on animals and epidemiological studies in humans, see Gluckman et al. (2007), Jirtle and Skinner (2007), and Nomura (2008).

²⁸ For some of the reviews of the corresponding literature, see for example, Franklin and Mansuy (2010), Jablonka and Raz (2009) and Aiken and Ozanne (2014).

VARIABLES	(1)	(2)	(3)	(4)	(5)			
Panel A: Dependent variable is HAZ for children 0-5 year old								
Maternal grandmother shock in-utero	-0.197^{**} (0.095)				-0.200^{**} (0.097)			
Maternal shock in year 0-1	L	$0.110 \\ (0.096)$			$\begin{array}{c} 0.083 \ (0.100) \end{array}$			
Maternal shock in year 1-5			-0.025 (0.046)		-0.040 (0.051)			
Maternal shock in year 6-10				-0.041 (0.048)	-0.047 (0.050)			
Child' sex (male)	$\begin{array}{c} 0.092^{***} \ (0.035) \end{array}$	0.092^{***} (0.035)	0.092^{***} (0.035)	0.092^{***} (0.035)	0.091^{***} (0.035)			
Child's age (in years)	-0.030*** (0.001)	-0.030*** (0.001)	-0.030*** (0.001)	-0.030*** (0.001)	-0.030*** (0.001)			
Constant	-0.358^{***} (0.047)	-0.370^{***} (0.047)	-0.359^{***} (0.049)	-0.355^{***} (0.049)	-0.339^{***} (0.052)			
Fixed effects	Yes	Yes	Yes	Yes	Yes			
R-squared	0.133	0.132	0.132	0.132	0.133			
Observations	$12,\!696$	$12,\!696$	$12,\!696$	$12,\!696$	$12,\!696$			
VARIABLES	(1)	(2)	(3)	(4)	(5)			
Panel B: Dependent variable	is Cognitive	e ability z-s	core for ch	ildren 8-11	year old			
Maternal grandmother shock in-utero	-0.097^{**} (0.045)				-0.107^{**} (0.046)			
Maternal shock in year 0-1		-0.063 (0.044)			-0.074 (0.046)			
Maternal shock in year 1-5			-0.002 (0.028)		-0.016 (0.029)			
Maternal shock in year 6-10				-0.008 (0.028)	-0.016 (0.029)			
Child' sex (male)	-0.007 (0.024)	-0.007 (0.024)	-0.007 (0.024)	-0.007 (0.024)	-0.008 (0.024)			
Child's age (in years)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)	-0.001 (0.001)			
Constant	(0.079) (0.097)	0.077 (0.097)	$0.074 \\ (0.097)$	$0.075 \\ (0.097)$	0.092 (0.098)			
Fixed effects	Yes	Yes	Yes	Yes	Yes			
R-squared	0.171	0.171	0.171	0.171	0.171			
Observations	9,278	$9,\!278$	9,278	9,278	9,278			

Table 2.4: Third generation effects of exposure to negative rainfall shocks in utero and post-birth

Notes: Standard errors in parentheses clustered at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects include: mother's year of birth, month of birth, and district of residence.

Panel A in Table 2.4 shows results for HAZ of children 0 to 5 years old, while Panel B shows the estimation results for cognitive ability z-scores of children 8 to 11 years old. Both Panels A and B show that there is no statistically significant effect of a negative rainfall shock experienced by the mother in various phases post-birth, i.e. when she is an infant (column 2), when she is between 1 and 5 years old (column 3) and when she is 6 to 9 years old (column 4), on her child's outcomes. There is a clear demarcation in results between the shocks in utero and other phases on both health and cognitive ability outcomes (also clearly seen in column 5 of Table 2.4 where all phases around birth are included together). Both Panels A and B show that the total effect completely disappears after birth and it is only the shock faced by the maternal grandmother when she is pregnant with the mother that passes down two generations. Thus, my finding that there are no negative effects of adverse shocks after pregnancy on third generation's outcomes is in line with evidence from animal experiments.

In animal experiments the difference between the effect of in-utero shocks and post-birth shocks on the third generation outcomes can be attributed exclusively to the genetic and epigenetic transmission, given that the environment for the third generation is completely controlled. On the contrary in my study, the difference in the effect of inutero shocks and post-birth shocks can be explained by both a reduction in the genetic and epigenetic transmission and a potential reduction of the effect on the second generation (mother) outcomes, which could be important to characterise the third generation (child) environment. Because we cannot control for all mother's characteristics which are relevant for child development, we cannot exclude that the larger effect of inutero shocks be explained in part by a larger effect on unobserved mother's characteristics we do not control for.

2.6 Further Analysis

In this section, I run a series of additional regressions to examine: (i) heterogeneity in my estimates, (ii) factors mitigating the effects of the third-generational effect that I find, and (iii) mechanisms that could be driving these effects. This additional analysis also helps me shed light on the exploratory analysis presented earlier, particularly on the plausibility of the presence of a genetic and epigenetic channel. These results are very similar to the estimates produced by my benchmark specification outlined in Equation (2.2). This section briefly describes them below.

2.6.1 Heterogeneity in estimates

One might expect third generation effects to differ by specific trimesters in which the grandmother faced the shock. This not only determines whether there is a differential effect for length of exposure to the shock, but it also helps identify critical periods in which the fetus could be more sensitive to the rainfall shocks. My third generation analysis seems to show that the effect for grandchild's general health (as measured by their HAZ) is mainly driven by their grandmother's exposure to the negative rainfall shock in the first trimester. Panel A of Table A1 in Appendix shows that the first trimester effect remains the same and highly statistically significant at 5 per cent level of significance throughout the columns, even after controlling for maternal characteristics. On the other hand, the trimester-specific results for grandchild's cognitive ability are much less precisely estimated and do not seem to be driven by a shock in any particular trimester (for details, see Panel B, Table A1 in Appendix).

My results so far show that there is a third-generational effect, despite controlling for maternal adult characteristics as mediators. Do I find a statistically significant netted out, third-generational effect because these characteristics are not affected by the shock in the first place? The answer is, no. I conducted a second generation analysis which looks at the effect of exposure to the shock in-utero on maternal adult outcomes in terms of number of years of education, monthly consumption expenditure and BMI z-score for samples of mothers of children 0-5 and 8-11. In Appendix Table A2, I find that exposure to an extreme negative rainfall shock causes a negative impact on mother's monthly consumption expenditure and her general health, represented by her BMI z-score. There is not much of an effect on education because number of years of education is a rough measure of cognitive ability and the effect of shock is not large enough to have any effect on number of years of education.

I also conduct a trimester-specific analysis for the second generation. There is discrepancy in literature regarding the relative importance of different periods of gestation for different outcomes. Most studies have confirmed that the first trimester is critical for brain development, because it is a stage during which the epigenetic programming of the endocrine system occurs. Conversely, the third trimester is known to be critical for general health, because that is the time when the fetus gains the most weight (Duchoslav, 2017). My second generation results – for the mother who experienced the shock in-utero – follow this hypothesis for health outcomes and show

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that shock experienced in the third trimester causes a negative impact on mother's general health, represented by her BMI z-score. This effect is highly statistically significant for both samples (see Panel A and B of Appendix Table A2 for trimester-specific second generation results).

I also explore whether the third generation effect is different for boys and girls by looking at the estimates of the interaction of gender and maternal rainfall shock variable for child's HAZ and cognitive ability outcomes in Tables A3 in the appendix. I do not find any evidence of gender differences.

2.6.2 Factors mitigating the negative effect of the shocks

My analysis considers rainfall shocks over a span of 35 years; the period during which my sample mothers were born i.e. from year 1955 to 1990. It is useful to see if there is still an effect on third generation outcomes in a more recent cohort and if the presence of any government support programmes could act as an insurance against bad weather conditions to reduce the negative effect of the shock. In India, 3 of the 6 major poverty alleviation programmes were launched in or after the year 1980; the rest were launched after the year 1990, which is the period beyond the scope of my analysis.²⁹ In all of these programmes there was some form of support available to households to cope with poverty, e.g. a subsidy to small farmers, providing employment to those unemployed or under-employed, or wages to landless labourers paid partly in money and partly in food grains. These programmes may remediate the negative effects of rainfall shocks. In Table A4 in the Appendix, I interact the in-utero rainfall shock variable with a dummy variable Post1979, which takes the value 1 if the mother was born in 1980 or later and 0 otherwise. Column 1 - in which I use the same controls as in Table 2.2 - shows that without any government support programmes, for every additional month of exposure to extreme negative rainfall shock by the grandmother, the child's HAZ reduces by 30% of a SD. The presence of programmes cancels out this negative effect and improves health of grandchildren 0 to 5 years of age by about 10 per cent of a SD (-0.300+0.397=0.097). Column 2 shows that there is no negative effect of the in-utero shock on health of children 8-11 years, with or without any programmes again due to the possibility of catching up effect in nutritional intake as children grow older. This confirms the previous results from Table 2.2. Column 3 shows that without presence of any programmes, every

²⁹ See Mondal, P. (n.d.). Top 6 Major Poverty Alleviation Programmes in India. Retrieved from <u>http://www.yourarticlelibrary.com/poverty/top-6-major-poverty-alleviation-programmes-in-india/32152</u>

additional month of exposure to extreme rainfall shock in-utero reduces cognitive ability z-score of grandchildren 8-11 years old by approximately 10 per cent of a SD. However, there is a positive sign for the effect of rainfall shock in presence of government programmes on cognitive ability outcomes, but it is not statistically significant. Thus, an important takeaway from these results is that there is margin for improvement in health outcomes of children 0-5 years old if there is support available through government poverty alleviation programmes at the time when the grandmother was pregnant. An improvement in health of children in the critical period of 0-5 years is likely to have a positive impact on their long-term outcomes as well.

I move now to test whether the negative effect of rainfall shocks is amplified for grandchildren in a low socioeconomic background or where there is less wealth available to be spent on them. I use two ways to test for this: a) I look at the heterogeneous effects on third generation children in households that are poor and b) I look at heterogeneous effects of the shocks on third generation children by the total number of children that their parents have. First, the negative effect of the shock may be amplified if the child is living in poor circumstances and has fewer resources to remediate the negative effects of the shock. In Appendix Table A5, I interact a dummy for poor households with the shock in-utero. The dummy takes on a value of 1 for those households who live below the poverty line, which is created based on consumption expenditures. I find an interactive effect for health outcomes of children for both 0-5 and 8-11 samples, but no effect of shock for poor households on cognitive ability outcomes. In columns (1) and (2) it seems that the effect on health outcomes is significant and more negative for households living below the poverty line. Second, number of children is another way by which the effect of the shock may be intensified – a negative rainfall shock can have a larger effect on bigger sized families because time and income investments each child gets depends on the number of total children a family has, especially in a developing country like India. I interact number of children a mother has with the shock that she experienced while in-utero. Appendix Table A6 shows that even though the effect for number of children is negative and statistically significant, every additional child has a negative effect of health and cognitive ability outcomes, the effect of the rainfall shock does not seem to be amplified by the number of children a family has. There could, however, be a potential issue with these results because poverty status of the households and the number of children could be endogenous i.e. caused by the shock. And even though I assume that the shock that was experienced so long ago by the grandmother should not continue to

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have an impact on the number of children and the poverty status of the future generations, it is important to highlight that these must be looked at with caution.

There are two other ways in which the grandmothers may be insured and it is important to see if the effect of the shock may be attenuated once those are taken into account: a) using marriage migration as a way of consumption smoothing, and (b) using marriage migration as insurance against climatic shocks. Both of these can act as a way to protect households from income or nutritional shocks. I do not have data to test them empirically, but I justify my analysis using evidence from literature. Firstly, as there is great geographical variation in yields from agriculture, households use marriage of females as a way to smooth consumption by co-insuring each other (Rosenzweig & Stark, 1989). This means if one family has a good harvest, but their daughter's does not, they may transfer their resources to her family, leading to lower consumption volatility overall. In order to test this, Fulford (2015) uses the same data that I use in this analysis (IHDS) to investigate if households are making any transfers to actually provide insurance. He finds that the reported transfers from or to a married daughter, sister or niece are as low as 0.05% across households in India. There could be a possibility of underreporting due to respondents forgetting about making or receiving any transfers, but the same respondents reported receiving 26 times more from a married son, brother or nephew. Without any transfers between households, I reject the hypothesis of households using marriage migration as a means of creating consumption smoothing links. Secondly, rainfall is one of the most important determinants of income in rural India (Jayachandran, 2006) and higher rainfall volatility means greater income volatility. This geographically correlated income shock means that parents, in general, may try to send their daughter farther away to mitigate effects of such climatic shocks. Fulford (2015) tests this smoothing strategy through the assumption that the marriage migration should be higher in areas that are exposed to additional rainfall volatility. He finds the opposite of what one would expect if consumption smoothing were an important factor in marriage migration – households in districts with higher rainfall volatility have stronger incentive to find ways of insuring themselves, yet they are found less likely to marry their daughters outside the village. Thus, based on all these results, it is safe to say that my results are not overestimated.

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2.6.3 Mechanisms driving the results

Further, I examine the effects of maternal grandmother's in-utero rainfall shock on third generation's outcomes by urban vs. rural areas in India. It appears that in-utero exposure to extreme negative rainfall shocks has a multigenerational impact on child's health and cognitive ability that is driven by residence in rural areas (for details see Table A7 in Appendix). The effect of in-utero exposure to the shock on the grandchild in rural areas is even more statistically significant and its magnitude is twice as large as in urban areas. It is likely that households in rural areas have a greater reliance on agriculture for their income; thus if rainfall shocks affect agriculture, they are expected to have a larger negative impact of these shocks on exposed mothers, and later on their children as well.

Within rural areas, it is worthwhile to investigate households' mobility post-shock. After exposure to an extreme negative rainfall shock, do families stay in the same district or move away from the locus of the shock to some other district? One reason why they may be more affected by the shock is if they own and subsist on land and are directly affected by its negative effects on crop production and consumption expenditures. Table A8 in Appendix shows a statistically significant interactive effect of the shock on cognitive ability outcome for grandchildren in landowning households in rural areas. For those rural households who own any land, grandmother's exposure to extreme rainfall shock is negatively related with the cognitive ability of grandchildren. This means that some level of immobility associated with ownership of land can make it more difficult for families to relocate to other areas and offset the adverse effects of the shock through other sources of income e.g. maybe through a job elsewhere. In contrast, there is no statistically significant effect of the shock on rural landless households. One reason for this effect may be that after being affected by the shock, households without land ownership might find it easier to take jobs elsewhere and move away from the district; thus, this may be a reason why their children's outcomes, in comparison to the landowning households, are not seen to be affected by the shock exposure.

2.7 Conclusions

In this paper, I present novel evidence of multigenerational effects of in-utero insults. I use birth cohorts spanning 384 districts of India over 35 years (1955-1990) to estimate the multigenerational effects of maternal in-utero exposure to negative rainfall shock on health and cognitive outcomes of children. To identify a causal effect of an in-utero rainfall shock on outcomes of the third generation, my identification strategy exploits variation in historical rainfall over time and within geographical space. I link the districtby-month-by-year rainfall data with India Household Development Survey (2004-05) using the date of birth and place of residence of the mother to construct the potential rainfall shock during the nine months prior to her birth.

This study generates two broad conclusions about the effect of shocks: (a) any exposure to shocks during the utero phase has effects that pass down to the third generation's health and cognitive ability outcomes, and (b) genetic and epigenetic channels may be the main responsible for this multigenerational transmission beside the potential indirect transmission through unobserved mother's characteristics which may affect the child's home environment and which I cannot control for.

This is a first study that shows the effects of in-utero rainfall shocks on outcomes of the third generation. I find that there are strong negative effects of maternal grandmother's exposure to a negative rainfall shock during pregnancy on height-for-age of her grandchildren below age five, but not for grandchildren 8-11 years old – every additional month of in-utero exposure to rainfall shock reduces height-for-age z-scores by 19.7 per cent of a standard deviation for those 0-5 years old. Even though grandchildren seem to catch-up in terms of their physical health by age 8-11, studies confirm that the damage done to their health in the first few years of their life remains largely irreversible, with effects potentially hampering the quality of their adult life. Grandchild's cognitive ability z-score at age 8-11 reduces by approximately 10 per cent of a standard deviation with every additional month of maternal grandmother's exposure to rainfall shock during her pregnancy.

The implications of these results can be far-reaching because early childhood (first 60 months of a child's life) HAZ is a good predictor of not only their adult health, but also of their cognitive, non-cognitive and labour market outcomes (Maluccio et al., 2009; Maccini & Yang, 2009; Hoddinott et al., 2008; Case & Paxson, 2008a; Alderman et al., 2006). Similarly, a significant effect on cognitive ability of children till age 8-11 means that children affected by the mother's in-utero shock are more likely to continue to suffer from the negative effects later in life as well in terms of other human capital and economic outcomes.

As previously mentioned, most of the evidence on persistence of negative effects of prenatal shocks into future generations comes from experiments on animals. While these experiments are able to questionably isolate the direct biological mechanisms from the indirect effects, similar evidence in human populations is inexistent. In human studies these effects capture not only the direct genetic and epigenetic effects of the shocks, but also the indirect effects through changes in socioeconomic environment, where a mother who is insulted in utero and grows up to attain lower education and poorer health, raises a child in low-resource environment as well. The genetic and epigenetic component of the multigenerational effect may not be remediable after the damage is done, but we do know that some of the transmittable effects of rainfall shocks are transferred through maternal outcomes (see for instance, Caruso & Miller, 2015; Tan et al., 2014). By controlling for broad maternal adult characteristics, I partially account for the child's socioeconomic environment and I find that third generation effect remains almost unaltered. This seems to suggest that the effect of maternal in-utero shocks on the third generation is driven potentially more by unexplained genetic and epigenetic channel than by the environmental channel.

Additional analysis comparing the effects of in-utero and post-birth shocks shows that the only in-utero shocks have effects that pass down to the child. Using the evidence from human studies that epigenetic effect is the strongest in-utero and weak or even absent after birth and from animal experiments that in-utero shocks effects on third and following generations are driven by epigenetics and not environment, I suggest that the multigenerational transmission of the effects of in-utero rainfall shocks is likely to be explained mainly by biological channels and plausibly the result of epigenetic inheritance.

Moreover, it is interesting to note that if a woman is exposed to the shock in presence of a government support programme, it mitigates the negative effect on health outcomes of her future generations. Therefore, in terms of policy implications, it is crucial to develop poverty alleviation programmes and health interventions to help reduce the impact of rainfall shocks especially on pregnant women to minimise the adverse effects of rainfall shocks on two subsequent generations. Moreover, for an economy that is predominantly based on agriculture, the effects of rainfall shocks are stronger in rural areas than in urban areas and are amplified for rural landowning households. Thus, targeting government interventions at these subgroups may be a possible solution to assuage the negative effects of the multigenerational shock. Chapter 3

Parental investment responses to child's health: Are there gender-differences in Andhra Pradesh, India?

Parental education investment responses to child's health: Are there gender-differences in Andhra Pradesh, India?

Abstract

In this paper, I use instrumental variable approach to investigate gender-differential response of parents' education expenditure to changes in child's health due to negative rainfall shocks in early childhood. I use the first two rounds of the rural sample of Young Lives survey for Andhra Pradesh, India and district-level monthly rainfall data predating the start of Young Lives sampling process. I find that parents in rural areas, on average, compensate for changes in child's health by increasing their education expenditures. I also find gender-differential investment behaviour; even though parents show a compensatory strategy for both boys and girls, they seem to invest more in education of a boy as compared to a girl child. This suggests that when hit by a shock, parents protect their son's health more than they protect their daughter's.

Keywords Education, child health, rainfall shock, parental investment, son preference, India

JEL I00; I14; I24; D19; D91; J16

3.1 Introduction

There is mounting evidence emphasising the importance of early childhood experiences and investments in shaping children's early life human capital, subsequent development and therefore also their later life outcomes.¹ In theory, through their investments, parents can choose to either amplify (reinforce) or attenuate (compensate) the effect of early life experiences and shocks on long-term human capital outcomes. The relationship between human capital development and investments also has an often ignored gender dimension to it. Evidence of human capital differences by gender emerges in early years of life; thus, any differences in parental investments by their child's gender may also translate into the well-documented boy-girl gaps in adult life outcomes.²

In this chapter, the main questions guiding my analysis are whether parental education investments reinforce or compensate for changes in child's health due to negative shocks in early life and whether there is any heterogeneity in their investment responses across boys and girls. Specifically, I investigate gender-differential response of parents' education expenditure when their child is 4 to 6 years old to child's health (measured by height-for-age) observed between ages 5 and 19 months in India. In other words, in a low resource setting like India, if a negative shock leads to an adverse impact on a child's health, do parents respond by investing even less in human capital of that child (i.e. adopting a reinforcing investment strategy) or do they make up for the negative impact of the shock by investing more in the adversely affected child (i.e. make compensatory education investments) and is this response motivated by the gender of the affected child.

More than 200 million children across developing countries are potentially at a risk of being unable to reach their full potential of human capital due to poverty and other environmental challenges (Currie & Vogl, 2013; Grantham-McGregor, et al., 2007). This makes it relevant to explore this pattern of parental investment responses in the context of a country like India where such challenges are much more heightened and are also coupled with pervasive son preference that is one of the strongest manifestations of gender inequality in the country (Pande & Astone, 2007). Investigating education expenditure in Indian context is important in its own right as according to Education for

¹ See, Almond and Currie (2011a) for a synthesis of this literature.

² For example, even though Baker and Milligan (2013) study parental time investments in Canada, they find that gender-differences in these investments start to emerge as early as nine months.

All Global Monitoring Report, India has the highest population of illiterate adults, standing at 37 per cent of global statistics. India is also expected to be more than 50 years late in achieving the 2030 Sustainable Development Goals (SDGs) deadline for universal primary education (UNESCO, 2015). Moreover, crude statistics from my rural sample (see Table 3.3) show that the mean difference in the education expenditures between boys and girls is highly statistically significant with more monetary resources spent on boys compared to girls.³ Hence, it is worthwhile to probe further the gender-differential response in these education expenditures.

I use the first two rounds of the longitudinal dataset of the Young Lives study conducted in Andhra Pradesh, a largely rural state in India. Measuring the response of parental education expenditure to child health introduces empirical challenges due to reverse causality and unobserved factors affecting both child's health and parental investments, which unless accounted for, will lead to biased estimates. As lagged variable for child health is used, potential endogeneity in my analysis is not caused by reverse causality, but by presence of unobserved confounding factors, rendering the child health measure endogenous. In order to correct for the bias, I use an instrumental variable approach. I use exposure to a rainfall deficit as an instrument for child's physical health. Furthermore, to assess whether parental strategy of education expenditure varies across gender, I allow the effects of child health on education expenditure to differ between boys and girls. Finally, I test the validity of my instrumental variable estimation by (i) checking whether my estimation results change for households that have access to irrigation facilities; (ii) checking if my results change in presence of government poverty alleviation programme; (iii) testing if there is serial correlation in my rainfall deficit measure in the last 30 years.

Overall, my paper adds to the small, but recently expanding subset of literature on the role of parental investments in reinforcing or compensating for changes in child human capital due to early-childhood shocks and interventions.⁴ My paper makes a methodological contribution in how it addresses endogeneity of child's health.⁵ Andhra Pradesh experienced a Drought in the South-West monsoon period of 2002 (just before

³ Sample statistics show that the p-value of the difference in education expenditure, which is in favour of boys, is 0.004 (i.e. < 0.01) making the difference statistically significant at 1 per cent level of significance. ⁴ See, for example, Restrepo (2016); Aizer and Cunha (2012); Akresh et al. (2012); and Venkataramani

⁽²⁰¹²⁾ for studies that try to answer a similar research question. ⁵ See for example, Adhvaryu and Nyshadham (2014); Venkataramani (2012); Kelly (2011); Almond,

Edlund, and Palme (2009) for methodology used in related literature to address endogeneity issues.

the data was collected for these children in the first round of Young Lives study) causing a rainfall deficit in all sample districts - I exploit the timing and variation in the extent of this deficit to purge child health of its endogenous component to estimate its impact on education expenditure.

Moreover, research that uses shocks to study this empirical question is mostly part of sibling-rivalry literature, where it is common to use twins for analysis, in which they crudely assume that the shocks occur randomly within twin pairs and do not address potential endogeneity of endowments for children born as singletons. Instead, my identification strategy of using a plausibly exogenous source of instrument for variation in child health allows me to identify parental responses more convincingly, because it allows me to control for any other factors that could affect parental investment responses other than through the channel of child's health. While other studies using such a strategy have focused more on extreme and often less generalizable policies and events experienced in childhood (see, for example, Adhvaryu & Nyshadham, 2016; Halla et al., 2014), I focus on negative rainfall shocks, which are considered more frequent and "normal" to childhood experiences (Maccini & Yang, 2010).⁶

Finally, my main contribution is related to exploring heterogeneity in parental investment responses across boys and girls. Most studies either analyse gender preferences or the dissension in reinforcing or compensating behaviour; however, combining the two could better explain if gender lies at the root of these investment differences. This paper allows for a more nuanced understanding of how investment responses vary by severity of the rainfall shock and the role of subgroups (e.g. by gender) in mitigating or aggravating such a behaviour. Instead of relying on using endowments at birth (e.g. birthweight) as a proxy for child health, Leight (2017) claims to be the first to employ climatic shocks such as grain yield in infancy as an instrument for child health to compare siblings in China. However, Leight (2017) does not address the gender aspect at all even though China is as infamous for strong son preference as India (Jayachandran, 2015). Thus, to the best of my knowledge, this is the first analysis that looks at genderdifferential responses of parents in India to changes in child's health due negative rainfall shocks in early life.

⁶ Other examples of studies using less severe natural shocks include Carrillo, Lima, and Trujillo (2016) who study in-utero exposure to heat waves in Colombia. Leight (2017) relies on using grain yield in early-childhood as an instrument for height-for-age in China.

To preview my results, I find that if a rainfall deficit adversely affects a child's health, parents respond by investing more in that child in terms of education expenditure, thereby compensating for an adverse shock to his/her health in early childhood. While I do find a compensatory behaviour on average, parental investment reaction also seems to differ between boys and girls. Parents seem to compensate for a boy's health, but they show no such strategy for changes in a girl's health. This suggests that when hit by a shock, resource constrained parents, in my rural sample follow an efficiency investment strategy by protecting their son's health more than they protect their daughter's. This could be related to parents expecting higher utility and returns from investment in a son.

My results hold important implications for policymakers. In terms of social policy, they emphasise the need for support to disadvantaged families, which would assure a sufficient level of early childhood investment. The support should particularly be targeted to those families living below the poverty line, in which case resource constraints might force them to choose to invest more in children with higher returns, possibly resulting in discrimination against girls as parents view them as a cost to the family. Moreover, policies that help promote greater investments in early childhood, especially targeted at those families hit by a shock, might also result in improving their children's long run outcomes as well.

The remainder of the paper is organised as follows. Section 2 discusses the related literature and where my paper fits into that. Section 3 introduces key features of the sample, the measurement variables and the instrumental variables chosen for the analysis. Section 4 lays out in detail the empirical strategy that I use. I report my main results in Section 5 and present empirical evidence for the validity of my instrumental variable approach in Section 6. Finally, Section 7 provides some conclusions.

3.2 Related Literature

The insights of this paper are derived from combining and building upon two strands of literature – first strand relates to parental investment reaction to differences in child human capital in early childhood, while the second strand is related to gender bias in parental investment decisions. Both these subsets are interconnected, but studies mostly only focus on either one of them. In this section, I summarise literature related to each, elaborating on potential explanations for heterogeneity in investment behaviour by gender.

Burgeoning empirical literature has explored whether parents reinforce or compensate for child human capital; however, the results of this literature are still inconclusive and indicate that parental response to child human capital is heavily contextspecific. Most studies have found evidence of reinforcing parental responses (e.g. Aizer & Cunha, 2012; Adhvaryu & Nyshadham, 2016; Behrman et al., 1994; Datar et al., 2010; Ermisch and Francesconi, 2000; Frijters et al., 2013; Grätz & Torche, 2016; Hsin 2012); several have found that parents compensate in their responses (Ashenfelter and Rouse, 1998; Behrman et al., 1982; Bharadwaj et al., 2018; Cabrera-Hernandez, 2016; Del Bono et al., 2012; Griliches, 1979; Halla et al., 2014; Leight, 2017); a few have found that parents neither compensate nor reinforce (e.g. Almond and Currie 2011a, Lynch and Brooks 2013, Royer 2009); while there are some that have also found both behaviours in different measures of endowments. For example, both Ayalew (2005) and Yi et al. (2014) present evidence that parents reinforce endowment differences with respect to educational investments, but compensate with respect to health investments. However, to date, only a small, but expanding subset of literature has looked at these responses for early-childhood human capital differences caused by shocks and interventions as opposed to birth endowments (e.g. Aizer and Cunha, 2012; Akresh et al., 2012; Restrepo, 2016; Venkataramani, 2012).

To take this discussion further, it is important to explore the factors that drive parental investment behaviour and the potential explanations for why this behaviour is expected to differ between boys and girls. Recent, seminal research on this topic postulates that there could be at least three conceptually distinct, yet non-mutually exclusive explanations for parents investing differently in their children's human capital: (i) cost of investment, (ii) beliefs and (iii) preferences. The first explanation relates to the cost of investing in a child. These costs will result in different investment patterns if we assume that all children are identical and parents are only different in terms of the budget constraints that they face (Cunha, 2014). On this basis, poorer families faced with greater cost of investments would be forced to spend less on children with low endowments and for whom the costs of investments are higher (see for example, Dahl & Lochner, 2012; Becker & Tomes, 1986).⁷ It bears mention that my research is in a low-income country setting where these budget constraints are more binding than in higher-income countries.

⁷ This concept is also well-evinced in literature on sibling-rivalry, where higher quantity of children implies fewer resources available to each child i.e. resource dilution. This implies that parents follow a quantity-quality tradeoff, suggesting that if families choose more children, they also choose lower average quality (see Becker & Lewis, 1973 and Becker & Tomes, 1976).

Under this assumption, the cost of investing in a child with better health will be lower, inducing parents to reinforce human capital (Becker and Tomes, 1976; Grätz and Torche, 2016). Additionally, within family, cost of investments would also force parents to respond to systematic differences by gender (Behrman, Pollak, & Taubman, 1986). For example, parents in India are culturally expected to pay dowry upon their daughter's marriage. Since dowry is considered a financial cost of having daughters, parents may find raising sons less costly. This would encourage them to promote inequality by investing less in the relatively more expensive daughters (Jayachandran, 2015; Bharadwaj, Dahl, & Sheth, 2014).

A second plausible reason for differential investment behaviour is parental beliefs (also known as expectations) about productivity of their investments in terms of future labour market outcomes (Cunha, 2014). Returns to investments may differ across gender and are generally believed to hold true more for sons as compared to daughters (Garg & Morduch, 1998). Parents perceive lower returns to human capital investments in girls in terms of lower future earnings for them and end up investing lesser in them compared to boys (Attanasio and Kaufman, 2009; Cunha, 2014). Even though it is difficult to assess parental beliefs about returns to human capital investments, there is growing evidence that this is indeed part of the explanation, particularly in Asian context (Rosenzweig and Schultz, 1982). For instance, in India, opening of female-oriented call centres and other labour market opportunities in the community signalled availability of higher-paying jobs targeted at educated women. This increase in returns to schooling led parents to increase investments in girls' education (Jensen, 2012; Oster & Steinberg, 2013). Also, Jayachandran and Lleras-Muney (2010) in case of Sri Lanka find that schooling of girls increases as female adult mortality declines. So improvement in life expectancy increases the incentives for parents to invest in children's education, because longer life span increases the value of investments that pay out over time. Similarly, in China, higher returns in terms of higher price of female-intensive crops and accordingly, female incomes resulted in improvement in mortality rates and education for girls (Qian, 2008).

Third, parental preferences, in terms of child outcomes, child quality and the value parents place on certain skills in their child among other things, shape their investment behaviour (Francesconi & Heckman, 2016; Cunha, 2014). Parents consider certain dimensions of human capital (e.g. better health, cognitive ability or socio-emotional skills) as essential according to their standards of childrearing. Preferences are

heterogeneous and parents prefer to invest more, altruistically or paternalistically, in the qualities they consider more valuable in their child. Altruistic parents would invest more in a child based on maximization of child's utility, while paternalistic parents would evaluate investments in their children by maximizing their own utility function.⁸ For example, altruistic parents may prefer to minimise inequality between their children by compensating for a child's low endowments, while paternalistic parents would invest in a better endowed child who can maximize parents' utility which they may expect in terms of old age financial support and future care from that child. Like the previous two explanations, parental preferences may also differ by a child's gender. Parental preferences may favour boys or girls, by valuing identical outcomes at identical costs more for one gender than for the other. Preferences that are neutral between boys and girls exhibit "equal concern", where all children receive equal weight in parents' utility function, while those that favour one gender over the other exhibit "unequal concern" (Behrman et al., 1986; Lundberg, 2007). For instance, assumed that productivity and costs are same across gender, son preference may still induce parents to invest more in boys' human capital as they are more likely to stay with their parents even after marriage.

Due to a combination of these explanations at play, it is difficult to estimate parental investment behaviour empirically. There is growing evidence that parents are not fully informed about production technology or even about the skills of their own children, when making these investment decisions (Dizon-Ross, 2016; Cunha et al., 2013). Yet, most parental investment models typically assume that parents are fully informed (see, for instance, Cunha, 2014; Del Boca et al., 2014; Caucutt & Lauchner, 2012). Thus, how parents change human capital investments in their children in response to the gender or to the price of human capital depends critically on the relative importance of all of the three aforementioned explanations.

Gender inequality is a phenomenon not just common to developing countries, but essentially pervasive in some form in all societies. Even though gender gaps in human capital tend to be larger in developing countries, they are less explained by economic development and more by the preference of sons over daughters. Numerous studies have documented worse outcomes in developing countries for girls in many domains including education and health (Jayachandran, 2015). Despite their rapid economic growth, son preference has cultural roots in countries like India and China. Son preference causes

⁸ See Francesconi & Heckman (2016) and Doepke & Zilibotti (2012) for detail on paternalistic vs. altruistic preferences.

gender gaps in investments even if parents derive the same utility from a boy's and girl's quality. This is obvious from parents' son-biased fertility stopping behaviour (e.g. Clark 2000, Jensen 2003, Yamaguchi 1989) or the fact that mothers stop breastfeeding girls sooner to regain their fertility to try for a son (e.g. Jayachandran & Kuziemko 2011). Gender inequality in many cases is function of lower investments in girls as compared to boys (Barcellos, Carvalho, and Lleras-Muney, 2014). Lower investments in girls can be due to cultural preferences, but can also represent efficiency concerns. For instance, if parents anticipate more transfers from sons as opposed to daughters or if labour market returns on nutrition vary by gender then parents may discriminate against daughters in terms of nutrition allocation within families, irrespective of inherent cultural differences in gender preferences.

Pande and Astone (2007) analyse the determinants of long-standing social and cultural norm of son-preference in rural India and conclude that it is one of the strongest indicators of gender inequality in Indian context. There are several reasons for parental preference for sons, which motivate me to study this heterogeneity with respect to gender.⁹ First, India is predominantly a patriarchal society where only sons are allowed to carry forward a family's legacy and have a right to the lineage. Second, sons are more likely to enter into the labour market and are supposed to provide financial support and care to parents in old age. On the contrary, daughters physically and financially leave the family upon marriage and so less may be spent upon them due to the belief that parents will not reap potential benefits like old age financial support from them and in fact would have to pay dowry upon their marriage (Jayachandran, 2015; Garg & Morduch, 1998). In addition, there is also religious importance given to sons because in Hinduism (dominant religion in India) a dead parent's soul can only attain salvation if the funeral pyre is lighted by a son (Vlassoff, 1990; Pande & Astone, 2007). India also has an established history of distorted sex ratios, where birth of a daughter means that parents would continue to try for sons.¹⁰

Thus, after reviewing the literature, such strong cultural preference for sons in India compels me to hypothesise that parents in India, particularly in more resource constrained families, might not just prefer to invest in children with better physical

⁹ See Jayachandran (2015); Kugler & Kumar (2017); Pande & Astone (2007); Mishra et al. (2004); Vlassoff (1990) for reasons of why parents prefer sons over daughters.

¹⁰ Sex ratio in India is defined as the number of women per 1,000 men. The sex ratio declined from 972 females per 1,000 males in 1901 to 933 females per 1,000 males in 2001 (Banthia, 2001). The sex ratio in 2015 was 900 females per 1,000 males (Census of India, 2015).

Table 3.1: Cohort profile						
	(1)	(2)	(3)	(4)		
Round	1	2	3	4		
Year	2002	2007	2009-10	2013-14		
Younger Cohort						
Approximate age	5-19 months	4-6 years	7-8 years	11-12		
Older Cohort						
Approximate age	8 years	12 years	15 years	19 years		

Source: Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

health, but may also vary their investments by gender, potentially investing relatively more in their sons.

3.3 Data

3.3.1 Data and sample selection

My paper uses two sources of data. First, I use data from the Indian survey of the Young Lives project. Young Lives is a unique, longitudinal study of 12,000 children, in younger and older cohorts, in Ethiopia, India (Andhra Pradesh), Peru and Vietnam. The study spans over 15 years focusing on investigating childhood poverty. Young Lives survey consists of four rounds completed in years 2002 (Round 1), 2006-07 (Round 2), 2009-10 (Round 3) and 2013-14 (Round 4).

I focus on 2,011 children from the younger cohort, observing them approximately from age 5 months, in 2002, through age 12 in year 2013-14 (see, Table 3.1 for a summary of the cohort profile). In my sample, I only use information from first two rounds of data from Andhra Pradesh, India. As shown in Table 3.1, the same 2,011 children who were approximately 5-19 months in 2002 (Round 1) were tracked and surveyed in 2006-07 (Round 2) at age 4-6 years. Between these two rounds, the attrition rate due to household mobility or mortality was only 0.9 per cent across the whole sample (Boo, 2009).¹¹

Due to their aim to document child poverty, Young Lives deliberately over sampled poor communities, making the sample not a completely representative one. This

¹¹ The attrition rate between baseline and Round 4 was less than 3 per cent for the younger cohort.

is an advantage for me, because my research question of studying parental investment responses is more relevant in resource constrained households in which parents usually have to choose to make one form of expenditure by sacrificing other expenditures. However, this pro-poor sample still covers households from various socioeconomic backgrounds and is also caste representative (Boo, 2009). Young Lives survey only follows one child per household through various rounds, so each child represents a different household. I restrict the sample to households in rural areas only.¹² I focus on parental responses in terms of monetary education expenditure, the majority of which is school related, so one may argue that this expenditure may be zero for children below the official school starting age. Since there is no official school starting age in India, I consider all children enrolled in school.¹³

Second, in order to create a variable for rainfall deficit as an instrumental variable, I also use district-level, monthly rainfall data from year 1901 to 2002 for the state of Andhra Pradesh from India Water Portal (variable generation is explained in detail later in this section).

3.3.2 Setting

The state of Andhra Pradesh is one of the 29 states of India, situated in the south-eastern part of the country. It is the eighth largest and fifth most populated state in India.¹⁴ It borders with the Bay of Bengal on the east side and is known for its second longest coastline after the state of Gujarat.

Before June 2014, the state comprised of 23 districts spread across three distinct agro-climatic regions: Coastal Andhra, Rayalaseema and Telangana (see, Figure 3.1). After

¹² Since I am looking at investments in children as my outcome, it is important to consider investments towards which a significant share of parents' resources is directed. Education and entertainment expenses are considered on the basis that in rural India, one third of household expenditures are directed towards these two categories of expenses (Banerjee & Duflo, 2008; Fakir, 2016). Thus, restricting the sample to rural households will allow me to get a more accurate analysis of parents' reinforcing or compensating investment behaviour. Moreover, approximately 75% of the children in Andhra Pradesh live in rural areas and thus it makes sense to focus my analysis on this subsample. However, my main results are robust to including both rural and urban household and can be seen in Appendix Table B3.

¹³ Young Lives Survey asks a question about the type of school that their child goes to and gives the following options to choose from: private (unaided), NGO/Charity/not-for-profit, public (government), informal or non-formal community, charitable trust, bridge school, mix of public and private (private aided), and other. Parents are then asked about how much they spent on any of these options for their child and that makes up their education expenditure. Some children are never even enrolled in a structured, institutional setting, but that does not mean that there is no education expenditure on them.

¹⁴ Census Organization of India. (2011). Indian States Census 2011. Retrieved from. Retrieved from http://www.census2011.co.in/census/state/andhra+pradesh.html.





June 2014, Telangana region, comprising of 10 districts, was given the status of an autonomous state, leaving the new state of Andhra Pradesh with 13 districts only. However, since the data for Young Lives Survey was collected before 2014, in this paper I refer to the old state of Andhra Pradesh before it underwent restructuring in 2014. Young Lives survey considers 7 out of the 23 districts of Andhra Pradesh for the data.¹⁵

There is clear geographical disparity in India, with northern states generally exhibiting a much more pronounced gender bias and worsening trend in sex ratio compared to the southern states. In comparison to its northern counterparts, Andhra Pradesh has more educated women and lower rates of malnutrition (Boo, 2009). Thus, the generalisability of my results may be limited by the fact that Young Lives Survey for India is not a nationally representative sample. However, I do expect my research to be

¹⁵ The seven districts were selected based on the classification of poor/non-poor given by their relative levels of development.

even more relevant in case of the northern states, which I cannot study due to data limitations.

3.3.3 Measurement of variables

Outcome variable: Parental education expenditure

In order to understand parental responses to child's health, investment is considered at age 4-6 years (Round 2) in terms of direct monetary education expenditure. I consider logarithm of monthly expenditure in child's education. Education expenditure includes spending on school uniform, payment for schooling fees (registration/examination), payment for tuition, school books and stationary (pens, erasers, paper) and transport to school.

Young Lives survey includes data on total food and non-food consumption expenditures on household members. The survey initially asks about the exact share (in Indian Rupees)¹⁶ of the total non-food consumption expenditure spent on various items for all groups of all household males and females, children and adults separately. The items I consider for education expenditure include payments for school uniform, school fees, tuition, books and stationary and transport to school. It then has a follow-up question on how much of the total non-food consumption expenditures was spent on the child's school uniform, fees, tuition, books and stationary, and transport to school, asking them to choose from the following categories: 'none of it', 'less than half', 'almost half', 'more than half', or 'all of it'. To calculate an approximate value of expenditure on the child, I create new variables for each of the item, by multiplying the amount spent on each item with the share that the parents said they spent on the child: if the parents said they spent 'none of it' ('it' being the amount spent on each item e.g. tuition fees) on the child, then I multiply the share by 0 to get the child's share; if they said they spent 'less than half', I multiply the share with 0.25 (i.e. 25% of the share); if they said they spent 'almost half', I multiply the share with 0.5; if they said they spent 'more than half', I multiply the share with 0.75; and lastly if they said they spent 'all of it', I multiply the share by 1. This gives me an annual expenditure on each category of education for each child. I add up all of them to obtain total annual education expenditure for each child,

¹⁶ Indian Rupee (INR) = 0.0155 US Dollar (USD) and 0.0121 British Pound (GBP) (accessed from <u>http://www.xe.com/currencyconverter/convert/?Amount=1&From=INR&To=GBP</u> on June 15, 2017).

which I then divide by 12 to obtain a monthly expenditure on child's education. The final outcome variable is a log of the monthly expenditure on education.

Main explanatory variable: Child's health

In my analysis, I focus on the direct measure of a child's physical health as a dimension of human capital. Health is measured by considering standardised z-scores for height-forage of child in Round 1, when the child is 5-19 months old. Height-for-age z-scores¹⁷ are normalised according to World Health Organization (WHO) standards, representing the number of standard deviations (SD) a sample child's height-for-age is from the median of international reference population of the same age and gender. The z-scores range from - 5 to 5 SD, with less than -2 SD indicating stunting in a child (de Onis & Blössner, 2003). There is widespread consensus in literature to consider height-for-age as a measure of human capital and a summary indicator of physical robustness because it is correlated with a range of physical and cognitive indicators (Borga, 2016; Grantham-McGregor et al., 2007). Evidence also suggests that it reflects a long-term stock of nutrition or health prior to age three; hence, a robust relationship is expected between height-for-age and early childhood shocks (Leight, 2017).

Instrumental variable: Rainfall deficit

Rainfall deficit is used as an instrumental variable for child health. The use of this measure is motivated by the fact that rainfall is a critical factor in determining agricultural production across the state of Andhra Pradesh. Overall, the state depends on rain-fed agriculture for more than half of its total cropped area, while heavily relies on tube-wells and wells for irrigation for the other half. Timing and quantity of rainfall is crucial for both of these sources (Ahmed, 2015). Heavy reliance on rain-fed agriculture means a quasi-random rainfall deficit may greatly affect livelihoods in the state. Availability of reliable rainfall data preceding the start of Young Lives sampling process means that for my analysis I will not have to rely on the self-reported measures of household shocks in the data.

Moreover, I only focus on rainfall deficit in the South-West monsoon period in the year 2002. Andhra Pradesh experiences two monsoon seasons: the South-West monsoon during the months of June-September and the North-East monsoon during the months of October to December. South-West monsoon season contributes 66 per cent

¹⁷ Children's height is standardized according to the following formula: $Z = (x - \mu)/\sigma$, where x is the raw score and μ and σ are the mean and standard deviation, respectively (World Health Organization, 2010).

to the total annual rainfall of the state (Ahmed, 2015). I use monthly, district-level rainfall data during these four months for the state. Rainfall is significantly heterogeneous during this monsoon period across the three regions of Andhra Pradesh, with Coastal Andhra generally receiving the highest average rainfall, followed by modest rainfall in Telangana, and finally followed by precarious rainfall in Rayalaseema (EPTRI, 2012).

I use a continuous variable for rainfall deficit as an instrumental variable.¹⁸ It is referred to as a deficit because in the year 2002, India was hit by a severe drought affecting all districts of the state of Andhra Pradesh and causing the average rainfall to fall below the long term average, but still varying in intensity across all seven districts. This deficit is constructed as shown below, using monthly, district-wise rainfall data for the drought period. It is a district level indicator, measuring deviance of cumulative rainfall for the South-West monsoon period (in months June to September) of 2002 ($\Sigma_m R$) from each district's long term average rainfall \overline{R} over the same four months and divided by the standard deviation (σ^R). District-wise long term average rainfall and its deviations are estimated based on historical rainfall patterns from the year 1901 to 2000. Thus, for example, if an area is always dry, it is not a concern; rather, if it gets ever drier, that is when it would be a shock to the district.

$$RD_{d} = \frac{\sum_{m=1}^{4} R_{d_{m}}^{2002} - \overline{R_{d}}}{\sigma^{R_{d}}}$$
(3.1)

where m=1 for June, 2 for July, 3 for August and 4 for September; d is an index for district and goes from 1 to 7;

$$\overline{R_d} = \sum_{t=1901}^{2001} R_d^t \; ; \; R_d^t = \sum_{m=1}^{4} R_d^t_m$$

$$\sigma^{R_d} = \sqrt{\frac{\sum_{t=1901}^{2001} (R_d^{\ t} - \overline{R_d})^2}{99}}$$

where t denotes year. RD_d is the rainfall deficit z-score; $\sum_{m=1}^4 R_d_m^{2002}$ is the cumulative rainfall for the SW monsoon period of 2002; $\overline{R_d}$ denotes each district's long term average rainfall (1901-2001) for SW period; σ^{R_d} denotes standard deviation.

¹⁸ Most studies only use a dummy for occurrence of a rainfall shock. Three other studies that use a continuous variable for rainfall fluctuations include Maccini and Yang (2009), Björkman-Nyqvist, Martina (2013) and Ahmed (2015). However, none of them use rainfall shock as an instrument like I do in this paper.

	(1)	(2)	(3)	(4)	(5)
DISTRICT	Rainfall (mm) (2002)	Mean (mm) (1901-2001)	S.D. (1901-2001)	Rainfall Deficit (%)	Rainfall Deficit (z-score)
Anantapur	235.677	352.570	94.360	-33.150	-1.239
Cuddapah	262.280	367.101	102.554	-28.550	-1.022
Hyderabad	510.950	663.696	162.836	-23.010	-0.938
Karimnagar	621.219	763.767	161.022	-18.660	-0.885
Mahbubnagar	359.937	476.802	115.850	-24.510	-1.009
Srikakulam	496.704	603.716	122.120	-17.730	-0.876
West Godavari	423.366	631.688	150.217	-32.980	-1.387

Table 3.2: District-wise rainfall z-scores during the South-West monsoon

period of the Drought of 2002

Source: Author's calculations using data from India Water Portal accessed on 30th Sept, 2017.

Table 3.2 shows the calculations for standardized z-scores of rainfall exposure which are negative for all the districts, showing a deficit ranging from -0.87 to -1.38 (see column 6) and indicating that all districts were hit by the drought, but the effect varied in level of intensity. The higher negative number is indicative of a severer shock.

Younger children are likely to be more vulnerable to negative weather shocks. I expect negative rainfall shocks to affect children's health through income or price effect.¹⁹ Shortage of rainfall could adversely affect households, particularly in rural areas, who subsist on agricultural activity, due to a possible crop failure, and thus reduced incomes. This crop failure induced by a rainfall deficit could also cause a shortage of agricultural output, consequently increasing the prices of staple foods. Both of these effects, reduced incomes and increased prices, could cause parents to reduce investments in their children. There is consensus in medical literature that negative shocks, during prenatal stage and between birth and age three, have a substantial negative and irreversible impact on a child's physical and cognitive development (see, Grantham-Mcgregor and Ani, 2001; Pollitt et al., 1993). I use interactions between a continuous variable for a rainfall deficit and dummy variables that take value of 1 if child belonged to a certain age group (5-12 months or 13-19 months) at the occurrence of the deficit. In order to see if one age

¹⁹ I further elaborate upon this under the relevance condition in Section 3.6.1.



Age group (year 2002)	Observations	Per cent	Min	Max
Group 1: Child is aged >5 and \leq 12 months	548	47.9	5.063	12.427
Group 2: Child is aged >12 and \leq 19 months	596	52.1	12.493	18.805

Figure 3.2: Distribution of child's age in months

Source: Author's calculations based on data from Young Lives Survey round 1 (year 2002) for Andhra Pradesh, India.

group of children is more vulnerable than the other to the rainfall deficit, I define these particular age categories by dividing the sample in two comparable groups based on the distribution of child's age as shown in Figure 3.2. The figure shows two similarly distributed groups, 5-12 months and 13-19 months respectively.

Control variables

Control variables are measured in Round 1, when the child is between 5 and 19 months old. I include controls for child, mother and household level characteristics. Controls for child characteristics include age, age squared, gender, and dummy for the eldest child in Round 1.Child's age is measured in months and gender is a dummy variable for a male child.

Control for mother's characteristics include mother's years of completed education, mother's age in years and mother's age squared and mother's height in centimeters. Mother's education is ordinal variables where the categories of level of education are ordered and ranked from 0 to 14; '0' indicates no education, while '14' indicates having a graduate degree. I control for household characteristics as well by adding variables for the number of people from specific gender and age composition present in the same household as the sample child in order to act as a proxy for the structure of the family.

These categories are the following eight quantitative variables: number of males 6-12 years old; number of males 13-17 years old; number of males 18-60 years old; number of males 61+ years old; number of females 6-12 years old; number of females 13-17 years old; number of females 18-60 years old; and number of females 61+ years old.²⁰ Presence of other people in the household implies that parents' resources and time spent on the sample child could be diluted and could also be indicative of the support system available for the child. I also control for wealth index of the household which ranges from 0 to 1. It is a weighted average of (a) housing quality index; (b) access to services index; and (c) consumer durables index. Housing quality index is based on the number of rooms per person in a household (indicating crowding) and the quality of material used for the walls, floors and roofs within a household. Access to services index captures whether the household has access to electricity, sanitation facility, source of drinking water and the type of cooking fuel used. Finally, the consumer durables index is based on ownership of durable assets like radio, fridge, bike, television, motorbike, car, mobile phone, telephone and fan. Wealth index is measured in Round 1, when the child is 5-19 months old so that there is no reverse causality between investment and wealth.

3.3.4 Description of the data

Table 3.3 shows descriptive statistics of the entire sample (column 1) and separately for boys (column 2) and girls (column 3). Column (4) shows a 2-sample t-test to determine if the difference in means for boys and girls is significant. The p-value appears in parenthesis of column (4) and a value less than 0.05 shows that the mean difference between boys and girls is statistically significantly different from zero.

²⁰ I also had variables for males 0-5 years old and females 0-5 years old, but I do not include those because these variables would not be predetermined at the time of the shock in 2002 when the observed child is still between 5-12 months of age.

	(1)	(2)	(3)	(4)
VARIABLES	Full sample	Boys	Girls	Mean
	Mean $(S.D)$	Mean	Mean	difference
		(S.D)	(S.D)	(p-value)
Investments	2 2 2 2	0.100	0.001	
Log(monthly expenditure in education) (at 4-6 years old)	$2.263 \\ (1.873)$	$2.409 \\ (1.904)$	2.081 (1.819)	0.327^{***} (0.003)
education) (at 4-0 years old)	(1.073)	(1.904)	(1.019)	(0.003)
Monthly expenditure in	48.180	53.710	41.330	12.377**
education in Indian Rupee (at 4-6 years old)	(103.400)	(109.000)	(95.660)	(0.044)
+ o years ord)				
Child health				
HFA z-score (at 5-19 months)	-1.334	-1.462	-1.176	-0.285***
× /	(1.487)	(1.552)	(0.387)	(0.001)
Instruments Rainfall Deficit in year 2002	-1.024	-1.020	-1.030	0.009
Raman Dencit in year 2002	(0.157)	(0.157)	(0.157)	(0.324)
	× ,			
Rainfall deficit * dummy variable for child was 5-12	-0.453 (0.514)	-0.453 (0.513)	-0.455 (0.515)	-0.002 (0.949)
months old in 2002	(0.514)	(0.013)	(0.010)	(0.949)
Rainfall deficit * dummy variable for child was 13-19	-0.571 (0.528)	-0.568 (0.525)	-0.575	0.007
months old in 2002	(0.328)	(0.525)	(0.532)	(0.8181)
Child characteristics				
Dummy=1 if male	0.554	1	0	1
,	(0.497)	(0)	(0)	(.)
Child's age (months)	12.506	12.592	12.400	0.191
	(0.100)	(0.136)	(0.147)	(0.342)
Child's age squared	167.889	170.316	164.879	5.437
	(2.456)	(3.359)	(3.595)	(0.271)
Dummy=1 if child's age is	0.447	0.447	0.447	(0.000)
5-12 months	(0.497)	(0.498)	(0.498)	(0.999)
Dummy=1 if child's age is	$\begin{array}{c} 0.553 \ (0.497) \end{array}$	$\begin{array}{c} 0.553 \ (0.498) \end{array}$	$\begin{array}{c} 0.553 \ (0.498) \end{array}$	$0.000 \\ (0.999)$
13 - 19 months Dummy=1 if child is eldest	0.537	0.557	0.512	0.046
Dunning—1 if child is eldest	(0.499)	(0.497)	(0.512)	(0.123)
Mother's characteristics	· · /	× /	× /	· · /
	0 = 1 =	0.015	0 500	0.0=0
Mother's years of completed education	$2.747 \\ (3.884)$	$2.915 \\ (4.009)$	$2.539 \\ (3.716)$	$\begin{array}{c} 0.376 \ (0.103) \end{array}$
	27.510	(4.003) 27.430	(3.710) 27.600	-0.165
Mother's age (years)	(4.334)	(4.475)	(4.156)	(0.522)
Mother's age squared	775.500	772.700	779.000	-6.318
	$(264.300)\ 151.600$	$(276.200) \\ 151.600$	$(249.000) \\ 151.500$	$(0.688) \\ 0.157$
Mother's height	(6.354)	(5.905)	(6.875)	(0.678)

Table 3.3 : Descriptive Statistics

No. of males 6-12 years	0.405	0.395	0.416	-0.021
	(0.620)	(0.618)	(0.623)	(0.573)
No. of males 13-17 years	0.105	0.106	0.105	0.000
	(0.345)	(0.364)	(0.320)	(0.998)
No. of males 18-60 years	1.696	1.661	1.738	-0.077
	(1.131)	(1.121)	(1.142)	(0.253)
No. of males $61 + years$	0.223	0.243	0.199	0.043^{*}
	(0.429)	(0.443)	(0.409)	(0.089)
No. of females 6-12 years	0.535	0.513	0.563	-0.049
	(0.732)	(0.754)	(0.702)	(0.259)
No. of females $13-17$ years	0.146	0.159	0.131	0.028
	(0.409)	(0.422)	(0.391)	(0.246)
No. of females 18-60 years	1.745	1.680	1.826	-0.146**
	(0.999)	(0.916)	(1.089)	(0.014)
No. of females $61 + years$	0.187	0.191	0.182	0.009
	(0.392)	(0.393)	(0.391)	(0.702)
Wealth Index in year 2002	0.348	0.357	0.338	0.019**
	(0.165)	(0.172)	(0.155)	(0.049)
No. of observations	$1,\!147$	635	512	

Notes: The table shows sample means with standard deviations appearing in parentheses for columns (1), (2) and (3). For column (4), the table shows difference in means of the two groups of gender, with p-values of the t-test appearing in parentheses.

Source: Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

It is important to note from t-test p-values of the mean difference between subsample of boys and girls in Column (4) of Table 3.3 that both groups are comparable in terms of their means for almost all variables – as shown by a p-value greater than 0.05 – except for education expenditure and child health. The p-value of the t-test of difference in education expenditure and health of boys and girls is less than 0.05, making the difference statistically significant and signifying that parents invest differently between boys and girls, investing more in boys.

Parents on average spend approximately 48.180 Indian Rupees (approximately less than £1) per month on education of a child. They spend above the mean on a boy's and below the mean on a girl's education. Education expenditure for a boy is approximately 30 per cent more than it is for a girl. The descriptive statistics exhibit that the average height-for-age z-score of boys is lower relative to that of the girls and the difference is statistically significant. It is possible that the difference is driven by the shock itself because all sample districts in the state of Andhra Pradesh experienced a rainfall deficit in the year 2002, which just varied in intensity across different districts. The difference in means of HAZ across boys and girls would have warranted a measure
of height-for-age that is gender-specific, but this should not be a problem in my case as the HAZ measure is already standardised separately for boys and girls.

On average, the rainfall deficit z-score is -1.024 and it does not differ between samples for boys and girls. Child level controls show that 55.4 per cent of the sampled children are boys while 44.6 per cent are girls. On average, a child is 12.506 months old in Round 1. Approximately 44.7 per cent of the sample children were between 5 and 19 months old while 55.3 per cent of them were between 13 and 19 months old at the time of the rainfall deficit in 2002. On average, 53.7 per cent of the sample children are the eldest children in the family.

Descriptive statistics for mother's characteristics show that mothers of sample children have only completed roughly 3 years of education and are about 27 years old. An average mother is 151.6 centimetres in height.

Statistics for household characteristics mainly show the gender and age composition of all of the household members. Each control shows the number of household members who belong to the ten age and gender categories. I also control for the wealth index, which ranges from 0 to 1; a higher index is indicative of wealthier households. On average, households in my sample have a wealth index of 0.348.

3.4 Estimation Strategy

3.4.1 Parental investment responses to child's health

I estimate the response of parental education expenditure to child's health as measured by height-for-age z-score using the following Ordinary Least Squares (OLS) specification:

$$E_{i,2007} = \beta_0 + \beta_1 HFA_{i,2002} + \beta_2 C_{i,2002} + \beta_3 M_{i,2002} + \beta_4 H_{i,2002} + \epsilon_{i,2007}, \qquad (3.3)$$

where the dependent variable, denoted $E_{i,2007}$, is the reported expenditure on the education of child *i* in year 2007 when he/she is 4 to 6 years old. The independent variable, denoted $HFA_{i,2002}$, is child's health as measured by height-for-age in 2002 when the child is between 5 and 19 months of age. $C_{i,2002}$, $M_{i,2002}$ and $H_{i,2002}$ represent

vectors of child, mother and household level controls, respectively.²¹ The child-specific error term is denoted $\epsilon_{i,2007}$.

My parameter of interest in Equation (3.3) is β_1 , which captures whether parents invest more or less in children with lower height-for-age. A positive (negative) sign indicates that parental education investment response is reinforcing (compensating).

However, a potential issue in estimating the causal relationship between child health and parental investment response is that $HFA_{i,2002}$ in Equation (3.3) is likely to be endogenous, because it may be influenced by unobserved factors affecting the parental investment response. For instance, parental investment preferences are likely to be correlated over time. More committed parents may have a preference for higher prenatal investments, which can be correlated with both the child's height-for-age z-score in early life and parental later investments such as education expenditure, causing an upward bias in the OLS estimation of the effect of child's physical health (Borga, 2016). In a country like India, poorer households may be financially constrained and would have fewer resources to spend on both pre- and post-natal investments; not taking into account this cumulative lack of investments results in biased OLS estimates.

In order to address the endogeneity in child's height-for-age and estimate a causal link, I rely on Two Stage Least Squares (2SLS) as my benchmark estimation. To instrument for height-for-age in 2002, I use interactions between a continuous variable for intensity of rainfall deficit in 2002 and dummies for whether the child belonged to a certain age group (5-12 months or 13-19 months) at the onset of the deficit. These interactions with age groups will allow me to see if one group of children is more vulnerable than the other to the rainfall deficit. Intensity of rainfall deficit as an instrument allows me to capture exogenous variation in child health caused by random variation in rainfall across time and geographic areas in Andhra Pradesh. The first and second stage regressions are given by:

$$HFA_{i,2002} = \alpha_0 + \alpha_1 RD * Age5 - 12_{i,2002} + \alpha_2 RD * Age13 - 19_{i,2002} + \alpha_3 C_{i,2002} + \alpha_4 M_{i,2002} + \alpha_5 H_{i,2002} + u_{i,2002},$$
(3.4)

²¹ *Child controls include*: child's age in months; child's age squared; dummy variable taking value one if the child was 5-12 months old in 2002 and zero if he/she was 13-19 months old; and dummy variable for being the eldest child. *Mother's controls include*: mother's years of completed education; mother's age in years; mother's age squared; and mother's height in cm. *Household controls include*: Separate variables for the number of males present in the household in the age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; separate variables for the number of females present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; wealth index of the household in year 2002.

$$E_{i,2007} = \beta_0 + \beta_1 \widehat{HFA}_{i,2002} + \beta_2 C_{i,2002} + \beta_3 M_{i,2002}$$
(3.5)
+ \beta_4 H_{i,2002} + \varvarbox_{i,2007},

where $RD * Age5-12_{i,2002}$ and $RD * Age13-19_{i,2002}$ denote the instruments, i.e. interactions of rainfall deficit z-score with dummy variables that take value 1 if child *i* was between 5 and 12 months old and when he/she was between 13 and 19 months old at the time of the shock in year 2002, respectively. $\widehat{HFA}_{i,2002}$ is the predicted value of height-for-age computed using the estimated coefficients from the first stage regression (3.4). Child, mother and household level variables are included as controls in both regressions.

3.4.2 Gender differential investment reaction

I extend the analysis to explore gender heterogeneity in parental expenditure reaction to differences in child's health. I use 2SLS estimation again, but I modify my main independent variables in equations (3.4) and (3.5) by interacting height-for-age z-score with dummies for male and female child – this means I now have two endogenous variables. This specification allows me to investigate how parents react to differences in child's health given that the child is a boy or a girl. I now have two first stage regressions for height-for-age and its interaction male and female (equations 6 and 7, respectively). I further interact my instruments from equations (3.4) with dummies for gender of the child. The two first stage and the second stage regressions now look like:

$$HFA_{i,2002} * Male = \theta_0 + \theta_1 (RD * Age5 - 12 * Male)_{i,2002} + \theta_2 (RD * Age13 - 19 * Male)_{i,2002} + \theta_3 (RD * Age5 - 12 * Female)_{i,2002} + \theta_4 (RD * Age13 - 19 * Female)_{i,2002} (3.6) + \theta_5 Male + \theta_6 C_{i,2002} + \theta_7 M_{i,2002} + \theta_8 H_{i,2002} + e_{i,2002}$$

$$HFA_{i,2002} * Female = d_0 + d_1(RD * Age5 - 12 * Male)_{i,2002} + d_2(RD * Age13 - 19 * Male)_{i,2002} + d_3(RD * Age5 - 12 * Female)_{i,2002} + d_4(RD * Age13 - 19 * Female)_{i,2002} + d_5Male + d_6C_{i,2002} + d_7M_{i,2002} + d_8H_{i,2002} + \mu_{i,2002}$$

$$(3.7)$$

$$E_{i,2007} = \gamma_0 + \gamma_1 HF\widehat{A * Male_{i,2002}} + \gamma_2 HF\widehat{A * Female_{i,2002}} + \gamma_3 Male$$

$$+ \gamma_4 C_{i,2002} + \gamma_5 M_{i,2002} + \gamma_6 H_{i,2002} + \varepsilon_{i,2007}$$
(3.8)

where $(RD * Age5-12 * Male)_{i,2002}, (RD * Age13-19 * Male)_{i,2002}, (RD * Age5-12 * Female)_{i,2002}, (RD * Age13-19 * Female)_{i,2002}$ denote the instruments, i.e. triple interaction terms of rainfall deficit z-score, dummies for if child *i* was between 5 and 12 or 13 and 19 months old at the time of the deficit in year 2002 and dummies for child *i*'s gender. $HFA * Male_{i,2002}$ and $HFA * Female_{i,2002}$ are the predicted values of heightfor-age and gender interactions computed using the estimated coefficients from the first stage regressions (3.6) and (3.7). *Male* is a dummy for gender of the child. Child, mother and household level variables are included as controls in these regressions as well.

3.5 Validity of the instrumental variable estimation

For rainfall deficit to be a valid instrument for child's height-for-age, it must satisfy two key conditions of relevance and exclusion restriction.

3.5.1 Relevance condition

This first condition requires rainfall deficit to be highly correlated with height-for-age, conditional on all other exogenous variables in the model – that is,

 $Cov(RD_{2002}, HFA_{2002}|C_i, M_i, H_i) \neq 0$. Rainfall deficit is likely to be a relevant instrument in areas which are economically dependent on rain-fed agriculture (Sarsons, 2015). Two main channels have been identified in literature, through which a rainfall deficit may affect a child's physical growth as measured by height-for-age: (a) income effect and (b) price effect.²² Income effect entails that for households who subsist on agricultural activity as a source of income, a shortage of rainfall is likely to lead to a

²² See e.g. Ahmed (2016), Kumar et al. (2015), Shah and Steinberg (2013), and Burgess et al. (2011), for a greater discussion on the potential pathways through which rainfall affects child health. There is also a potential channel of 'time substitution' that might have an opposite, positive effect on child health. It may make my IV less relevant if the positive effect cancels out the negative effect of the rainfall deficit. For instance, a rainfall deficit may reduce work opportunities for households dependent on agricultural activity leading to caretakers having more time available to invest in a child's care, positively affecting his/her wellbeing. However, this threat is not relevant for my strategy because additional time spent may possibly affect a child's cognitive ability, but may not be able to compensate for lack of availability of nutrients and resources caused by a rainfall deficit. Ahmed (2016) does not find any significant impact of time substitution effect on a child's height and weight.

reduction in availability of food as a result of crop failure and dwindling incomes. Thus, for a rural household that is already financially constrained, a rainfall deficit and the consequent reduction in food availability is expected to hamper a child's physical growth.²³ The price effect suggests that a crop failure due to a rainfall deficit will reduce the supply of agricultural output, as a result increasing prices of staple foods. Thus, height-for-age may be adversely affected in rural families due to their inability to afford more expensive staple foods to meet the caloric intake requirements.²⁴ Both these channels have a negative effect and support the direction in which my instruments affect child health. These channels also support the relevance of rainfall deficit as an instrument because in my analysis I only look at households from the rural areas of the state of Andhra Pradesh, where the main economic activity is agriculture. So a rainfall deficit in a given year is expected to be directly related to crop yield, food prices and ultimately nutritional intake, thereby affecting health of children in that year.

Two possible concerns for the relevance of my IV is the availability of irrigation facilities and a government safety net programme referred to as the Indira Kranthi Pratham (IKP). Sarsons (2015) highlights that in settings where there is development in irrigation and other public services, rainfall as an IV should be used with caution because the presence of irrigation facilities weakens dependence of crop yield on rainfall and presence of public services weakens dependence on agricultural activity as a source of income. I address both these concerns below.

The state of Andhra Pradesh heavily relies on rain-fed agriculture, implying that drought periods would lead to crop losses, adversely affecting households' livelihood (Ahmed, 2016). To test empirically that the IV is also relevant for areas where there are some irrigation facilities, I test whether the estimated effect of rainfall deficit on child's health (in the first stage regression) is significantly different from zero for the subsample of households which report the presence of irrigation facilities. Column (1) of Table 3.4 shows that even in presence of irrigation facilities, rainfall deficit is a relevant instrument

²³ In addition to reduction in food availability, financial constraint could also affect a caregiver's behaviour as there may not be enough resources to obtain health inputs like medicines, vaccinations or medical care in a timely manner, impeding a child's physical health.

²⁴ It may be argued that in certain country context a monsoon rainfall may be interpreted as too much rain, which may also be detrimental to the crop yield. However, in case of India, Kumar et al. (2004) corroborates that increase in aggregate food grain and production of major crops like wheat, rice, sugarcane, groundnut and sorghum is positively and significantly dependent on an increase in monsoon rainfall. Hence, a rainfall deficit is likely to negatively affect production of these crops, increasing their overall prices and making them less affordable for rural households.

	(1)	(2)
Sample:	With irrigation	With IKP support
VARIABLE	$1^{\rm st}$ stage	$1^{\rm st}$ stage
Rainfall deficit $*$ dummy for child was 5-12 months old in 2002	-3.423^{***} (0.675)	$\begin{array}{c} \textbf{-2. } 395^{***} \\ (0.614) \end{array}$
Rainfall deficit $*$ dummy for child was 13-19 months old in 2002	-1.287^{**} (0.571)	-1.173^{***} (0.493)
Male (yes=1)	-0.254^{**} (0.115)	-0.198 (0.137)
Constant	-8.184^{***} (2.130)	-7.136 (2.290)
Child controls	Yes	Yes
Mother controls	Yes	Yes
Household controls	Yes	Yes
First stage F-statistic	15.132	9.652
Sargan-Hansen test p-value	0.764	0.894
Observations	547	489

Table 3.4: First-stage results for investment responses to child HFA – with availability of irrigation facilities and IKP programme Dependent variable: Log (monthly expenditure in education) in year 2007 when the child is 4-6 years old

Notes: Robust standard errors appear in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The sample includes children enrolled in school and living in rural households only. The regressions are run on different subsamples represented by those living in areas with irrigation facilities in column (1) and those households living in areas with presence of IKP programme in column (2). Child controls include: child's age in months; child's age squared; dummy variable for whether the child was 5-12 months old in year at the time of shock in year 2002; and dummy variable for the eldest child. Mother's controls include: mother's years of completed education; mother's age in years; mother's age squared; and mother's height in cm. Household controls include: Separate variables for the number of males present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; separate variables for the number of females present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; wealth index of the household in year 2002.

as the first stage F-statistic is greater than 10 and the p-value Sargan-Hansen test is 0.764, which confirms that my instruments are valid.

Next, I address the second possible concern of presence of public service programme. The government of Andhra Pradesh, in collaboration with World Bank, launched the IKP programme.²⁵ This programme could be a potential issue for relevance condition if for households who benefitted from IKP there is a weaker dependence of height-for-age on rainfall deficit, because now they have additional government support

²⁵ The main objectives of IKP programme were to empower communities, provide financial support to projects (identified by local self-help groups) that may deliver missing facilities in the community and improve service delivery of institutions in order to include marginalised groups in the community (Ahmed, 2016).

to mitigate negative effects of the weather shock. To test empirically that the IV is relevant also for areas where there is presence of IKP programme, I test whether the estimated effect of rainfall deficit on child's health (in the first stage regression) is significantly different from zero for both the subsample of households living with and without the presence of IKP programme. Column (2) of Table 3.4 shows the first stage regressions of households with IKP support. This first stage results show that even in case of lesser dependence of height-for-age on rainfall deficit for households with availability of IKP, the coefficients for rainfall deficit variables are negative and still statistically significant at 1 per cent level of significance. The first stage F-statistic in Column (2) is 9.652, which is not greater than 10 and suggests we should be cautious in attaching too much importance to an economic interpretation of the magnitudes of these coefficients. The p-value Sargan-Hansen test is also 0.894, which corroborates the relevance of my instruments even in presence of IKP programme.

3.5.2 Exclusion restriction

The second key assumption for validity of my instrument is that it has to satisfy the exclusion restriction. The rainfall deficit in 2002 must be uncorrelated with education expenditure in 2007, other than through an effect on child health in 2002, conditional on all other exogenous variables in the model – that is, $Cov(HFA_{2002}, v_{i,2007}|C_i, M_i, H_i) = 0$. A random rainfall deficit experienced by a child in early childhood when he/she was 5-19 months old should be unlikely to affect education expenditure decisions taken a few years later in 2007 when the child was 4-6 years old. So I expect a deficit in year 2002 to affect expenditure decisions in 2007 only through its impact on child height-for-age in 2002.

As pointed out by Bertelli (2015), serial correlation of weather shocks could represent a threat, making the rainfall deficit of 2002 not random. The effect of this shock could be overestimated if, for example, areas historically affected by shortage of rainfall are more likely to also experience a deficit in year 2002. However, I address this concern in three ways. First, rainfall deficit is random across time and geographic location in the way it is defined. Using deviation in severity of district-level rainfall deficit in South-West monsoon period in 2002 from a long term district average since year 1901 introduces more exogeneity at the district level. Second, exogeneity of my instrument can also be explained by the fact that I am looking at the deficit in four specific four months

VARIABLES	(1)	(2)	(3)	(4)	(5)	(6)
	Yearly SW Monsoon Rainfall Deficit (z-score) in th last 30 years (1972-2002)					
Lagged rainfall deficit	-0.078 (0.196)	-0.281 (0.183)	$\begin{array}{c} \textbf{-0.367 } * \\ (0.179) \end{array}$	-0.249 (0.183)	-0.351^{*} (0.182)	-0.185 (0.188)
Serial correlation test: H_{ρ} : no serial correlation						
Breusch-Godfrey test p-value (df=1)	0.392	0.251	0.288	0.594	0.133	0.205
Breusch-Godfrey test p-value (df=2)	0.573	0.509	0.547	0.863	0.139	0.432
Observations	30	30	30	30	30	30

Table 3.5: Evidence for lack of serial correlation in rainfall

Notes: Each column represents a different district of AP. Robust standard errors appear in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

(South West monsoon from July to September) of a particular year, indicative of a completely random shock, which is unrelated to the historical performance of the district. Thus, through my instrument I exploit the variation across time and within a particular geographical location (i.e. district). Third, I also empirically test this, as done in Bertelli (2015), by looking at serial correlation in the rainfall deficit measure in the previous 30 years. The lack of serial correlation is confirmed in Table 3.5 as the p-values of the Breusch-Godfrey test at degrees of freedom 1st and 2nd order correlation are above 0.05 across all districts.

Furthermore, the potential issue of non-random mobility caused by the rainfall is not an issue for my sample because I argue that the use of my IV is supported by the timing of both the rainfall deficit and the collection of data for the first round of the survey. The rainfall deficit occurred in the South West monsoon season from June 2002 to September 2002, while the data for the first round was collected starting from September 2, 2002 up until December 31, 2002. As my endogenous variable (height-forage) was measured right after the occurrence of the rainfall deficit, there seem to be hardly any gaps between the two time periods, suggesting that households may not have enough time to have relocated as a result of the deficit. Thus, my results should not be affected by selective sampling due to relocation of households.

Finally, rainfall shock can also have a direct response on education investment of the child through the fact that it reduced parental income or employment status. This means that the instrument may affect the outcome through a pattern that does not go

	(1)	(2)	(3)	(4)	(5)
	(1)	OLS	(3)		SLS
		OLS			
VARIABLES				${1^{ m st}} { m stage} { m HFA}$	$2^{ m nd}~{ m stage}$
HFA z-score (at 5-19 months)	303.089^{***} (73.986)	$\begin{array}{c} 192.154^{***} \\ (72.521) \end{array}$	117.152^{*} (69.150)		$\begin{array}{c} 453.757 \\ (317.936)) \end{array}$
Rainfall deficit * dummy variable for child 5-12 months old in 2002				-2.401*** (0.403)	
Rainfall deficit * dummy variable for child 13-19 months old in 2002				-1.394^{***} (0.305)	
Male (yes=1)	$\begin{array}{c} 43.410 \\ (209.816) \end{array}$	-45.401 (205.460)	-38.509 (198.368)	-0.291^{***} (0.082)	$74.103 \\ (244.112)$
Constant	$3,050.4^{*}$ (1,588.8)	$-8,074.1^{***}$ (3,103.9)	$-6,471.8^{**}$ (2,975.7)	-8.384*** (1.544)	-3,317.6 (3,440.8)
Child controls	Yes	Yes	Yes	Yes	Yes
Mother controls	No	Yes	Yes	Yes	Yes
Household controls	No	No	Yes	Yes	Yes
First stage F-statistic					27.022
Endogeneity test p-value	e				0.333
Sargan-Hansen test p-va	lue				0.233
Observations	$1,\!147$	$1,\!147$	$1,\!147$	$1,\!147$	$1,\!147$

Table 3.6: Evidence f	or lack of threat to	identification due to	the income channel

Dependent variable: Total monthly household expenditure in year 2007 when the child is 4-6 years old

Notes: Robust standard errors appear in parentheses. *** p < 0.01, ** p < 0.05, * p < 0.1. The sample includes children enrolled in school and living in rural households only. Child controls include: child's age in months; child's age squared; dummy variable for whether the child was 5-12 months old in year at the time of shock in year 2002 (for columns 6 and 7); and dummy variable for the eldest child. Mother's controls include: mother's years of completed education; mother's age in years; mother's age squared; and mother's height in cm. Household controls include: Separate variables for the number of males present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; separate variables for the number of females present in the household in year 2002.

Source: Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

through the physical health of the child, rendering the instrument invalid. In theory, I could control for the income of the parents or their employment status, but the issue in doing that is that they are bad controls. Instead, I show that for my benchmark result in Table 3.7, rather than having monthly education expenditure as outcome, I use the total monthly household expenditure as a proxy for parental income. New results presented in

Column 5 of Table 3.6 show that the coefficient for HAZ once I instrument it by the rainfall shock is not statistically significant, confirming that the effect of the rainfall shock when the child was 5-12 months does not have a long term impact on income or the expenditure of the family. Thus, I presume that any reduction in income is only temporary and does not pose a threat to my identification.

3.6 Main empirical results

3.6.1 Do parents reinforce or compensate for child's health?

I report OLS and 2SLS results for the main coefficients of investment, the effect of HFA and the effect of gender dummy in Table 3.7 (see Appendix Table B1 for remaining coefficients), which shows the impact of child health at age 5-19 months on investment responses in education at age 4-6 years. In column (1), I present results for my OLS specification in equation (3.3), with only child controls. Positive sign for height-for-age coefficient shows that better child health is positively correlated with investments in education. In other words, if a child has better health, it will induce parents to invest more in that child's education and vice versa in case of poor health. As I control for more covariates from column (1) through column (3), the height-for-age coefficient remains positive, but reduces in magnitude.

Next, in columns (4) and (5) of Table 3.7, I present the results for first and second stages of my benchmark specification in equations (3.4) and (3.5), respectively. Interestingly, when I address the bias in OLS results through 2SLS estimation (as shown by second stage results in column (5), height-for-age coefficient remains significant, but becomes negative. The reason for the coefficient to become negative is that column (1) is also picking up the effect of unobserved factors like prenatal investments or parental beliefs or preferences for better quality children, which could be biasing the estimates upwards. When I move from columns (2) to (3), controlling for more covariates reduces the upward bias and results in a coefficient in Column (3) which is very close to zero. Hence, when I use an IV approach to deal with the omitted variable bias, the coefficient in Column (5) decreases further, rather becoming negative. This indicates that parents in fact compensate in their investments, by investing more in children with worse health. This seems a plausible result as in presence of a rainfall deficit; a 1 standard deviation

	(1)	(2)	(3)	(4)	(5)
	(1)		(3)	. ,	. ,
		OLS			SLS
VARIABLES				1 st stage HFA	$2^{ m nd}$ stage
HFA z-score (at 5-19 months)	$\begin{array}{c} 0.203^{***} \\ (0.038) \end{array}$	$\begin{array}{c} 0.107^{***} \\ (0.036) \end{array}$	$\begin{array}{c} 0.055 \ (0.038) \end{array}$		-0.549^{***} (0.183)
Rainfall deficit * dummy variable for child 5-12 months old in 2002				-2.401^{***} (0.403)	
Rainfall deficit * dummy variable for child 13-19 months old in 2002				-1.394^{***} (0.305)	
Male (yes=1)	$\begin{array}{c} 0.338^{***} \\ (0.107) \end{array}$	0.257^{**} (0.100)	$\begin{array}{c} 0.233^{**} \ (0.098) \end{array}$	-0.291^{***} (0.082)	$0.049 \\ (0.125)$
Constant	$\begin{array}{c} 0.901 \ (0.728) \end{array}$	-4.525^{***} (1.549)	-4.780^{***} (1.520)	-8.384^{***} (1.544)	-8.905*** (2.048)
Child controls	Yes	Yes	Yes	Yes	Yes
Mother controls	No	Yes	Yes	Yes	Yes
Household controls	No	No	Yes	Yes	Yes
First stage F-statistic					27.022
Endogeneity test p-value					0.0001
Sargan-Hansen test p-	value				0.5007
Observations	$1,\!147$	$1,\!147$	$1,\!147$	$1,\!147$	$1,\!147$

Table 3.7: Parental response of education expenditure to child's height-for-age (HFA) Dependent variable: Log (monthly expenditure in education) in year 2007 when the child is 4-6 years old

Notes: Robust standard errors appear in parentheses. *** p < 0.01, ** p < 0.05, * p < 0.1. The sample includes children enrolled in school and living in rural households only. Child controls include: child's age in months; child's age squared; dummy variable for whether the child was 5-12 months old in year at the time of shock in year 2002 (for columns 6 and 7); and dummy variable for the eldest child. Mother's controls include: mother's years of completed education; mother's age in years; mother's age squared; and mother's height in cm. Household controls include: Separate variables for the number of males present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; separate variables for the number of females present in the household in year 2002.

Source: Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

decrease in height-for-age z-score causes parents to increase education expenditure by approximately 55 per cent, ceterus paribus.²⁶

²⁶ To explore an alternative measure of parental educational investment, in Appendix Table B4, I look at the impact on whether the child aged 4-6 years is currently enrolled in pre-school. I have enough percentage of children who are not enrolled in school at this age i.e. 44.4 % are currently not enrolled in preschool. My results are robust and coincide with the benchmark results in Table 3.7, where I find that the child who experienced a negative early-life health shock receives more educational investment in terms of enrolment in school. The effect on enrolment represents parents' decision to invest.

Coefficient for the dummy variable for male child is positive and statistically significant in columns (1) through (3). These positive correlations indicate an advantage for boys relative to girls. The advantage to boys disappears in IV estimation as shown in column (5) in which the coefficient for male dummy is positive, but not statistically significant.

I instrument child's height-for-age by rainfall deficit of year 2002 and its interaction with dummy variables which takes value 1 if the child belonged to that age group (5 to 12 months or 13 to 19 months) at the time of the deficit in 2002. Results of the first stage in Column (4) confirm the validity and strength of my IV. Coefficients of the instruments are significant at 1 per cent level of significance and show that for both age groups (children between ages 5 and 12 months and those between 13 and 19 months), a rainfall deficit decreases child's height-for-age. Higher magnitude of interaction of rainfall deficit and dummy variable which takes value 1 if child was between 5 and 12 months old during the deficit illustrates that younger children who are below 1 year old are much more vulnerable to the effect of the rainfall deficit. A one standard deviation decrease in rainfall is expected to decrease a child's height-for-age by 2.401 of a standard deviation for a child who was below 1 year old at the time of the 2002 rainfall deficit. Similarly, for a child between age 13 and 19 months, a standard deviation reduction in rainfall during the South-West monsoon season is expected to worsen a child's height-for-age by approximately 1.394 of a standard deviation.

The validity of my two instruments for height-for-age can be confirmed from the overidentification test. A p-value of the Sargan-Hansen test is 0.5007, which confirms that my instruments are valid. The F-statistic from the first stage is above 10 (i.e. 27.022) and suggests that the rainfall deficit instruments are a relevant measure of child's height-for-age. Furthermore, p-value of the endogeneity test is 0.0001, which indicates that the results of OLS estimates in Column (3) are statistically different from 2SLS results in Column (5). I reject the null hypothesis that height-for-age in OLS model is exogenous; thus, making 2SLS my preferred estimation.

	(1)	(2) OLS	(3)	(4)	(5) 2SLS	(6)
VARIABLES				$1^{ m st} { m stage} \ { m HFA} \ ({ m males})$	$1^{\rm st}$ stage HFA (females)	$2^{ m nd}$ stage
HFA for 5-19 months old * Male	$\begin{array}{c} 0.163^{***} \\ (0.048) \end{array}$	$\begin{array}{c} 0.051 \ (0.045) \end{array}$	-0.004 (0.046)			-0.635^{***} (0.210)
HFA for 5-19 months old * Female	0.266^{***} (0.061)	$\begin{array}{c} 0.192^{***} \\ (0.056) \end{array}$	$\begin{array}{c} 0.143^{***} \\ (0.056) \end{array}$			-0.426^{*} (0.259)
Rainfall deficit * Dummy for child 5-12 months old in 2002 * Male				-2.630^{***} (0.489)	-0.047 (0.183)	
Rainfall deficit *Dummy for child 5-12 months old in 2002 * Female				-0.238 (0.261)	-1.832*** (0.348)	
Rainfall deficit *Dummy for child 13-19 months old in 2002 * Male				-1.457^{***} (0.359)	-0.171 (0.155)	
Rainfall deficit * dummy for child 13-19 months old in 2002 * female				0.378^{**} (0.191)	-1.480*** (0.355)	
Male (yes=1)	$0.205 \\ (0.154)$	$\begin{array}{c} 0.074 \ (0.141) \end{array}$	$\begin{array}{c} 0.041 \\ (0.140) \end{array}$	-3.61^{***} (0.367)	2.74^{***} (0.348)	-0.223 (0.390)
Constant	$\begin{array}{c} 0.980 \\ (0.730) \end{array}$	-4.457^{***} (1.55)	-4.838^{***} (1.586)	-3.349^{***} (1.298)	-4.766^{***} (0.846)	-8.827^{***} (2.087)
Child controls Mother controls Household controls	Yes No No	Yes Yes No	Yes Yes Yes	Yes Yes Yes	Yes Yes Yes	Yes Yes Yes
F-statistic 1st stage: F-statistic 1st stage: Endogeneity test p-v Sargan-Hansen test p P-value for t-test HF	HFA(Male HFA(Fema calue p-value YA (Male)=) ale) HFA(Fema	le)			$\begin{array}{c} 14.26 \\ 21.49 \\ 0.0003 \\ 0.672 \\ 0.465 \end{array}$
Observations	1,147	1,147	1,147	1,147	1,147	1,147

Table 3.8: Heterogeneous effects by child's gender Dependent variable: Log (monthly expenditure in education) in year 2007 when the child is 4-6 years old

Notes: Robust standard errors appear in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The sample includes children enrolled in school and living in rural households only. Child controls include: child's age in months; child's age squared; dummy variable for whether the child was 5-12 months old at the time of shock in year 2002; and dummy variable for the eldest child. Mother's controls include: mother's years of completed education; mother's age in years; mother's age squared; and mother's height in cm. Household controls include: Separate variables for the number of males present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; wealth index of the household in year 2002.

Source: Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

3.6.1 Is there an evidence of gender bias?

In Table 3.8 (see Appendix Table B2 for remaining coefficients), I explore heterogeneity in parental allocation of education expenditure with respect to child's gender. I keep adding same controls as in Table 3.3 from Columns (1) through (3); positive correlations from the OLS results of the second row of Column (3) with all covariates show that parents seem to reinforce education expenditure for girls. In a rural setting like Andhra Pradesh, this implies that if a girl's height-for-age worsens by one standard deviation, parents would reduce education expenditure by approximately 14 per cent for that girl; on the other hand parents show a neutral and a statistically non-significant reaction to boys' height-for-age due the coefficient for height-for-age and its interaction with a dummy for male being statistically insignificant in Column (3).

Columns (4) to (6) show 2SLS estimation results in which I now have two endogenous variables (interactions of height-for-age with male and female dummy variables) to test for gender-differential response of expenditures to child's health. Columns (4) and (5) represent promising first stage results of height-for-age for males and for females, respectively. P-value of the Sargan-Hansen test is 0.6726, which confirms that my instruments are valid. The F-statistics from both the first stages is above 10, suggesting that my instruments are a relevant measure of child's height-for-age and gender interactions. Furthermore, p-value of the endogeneity test is 0.0003, which makes 2SLS as my preferred estimation over the OLS estimation.

A comparison of first stage results for male and female child show that for both age groups (5 to 12 months and 13 to 19 months at the time of deficit), a boy's health is more adversely affected by a rainfall deficit than a girl child's health. Columns (4) and (5) show that for children under one year of age, a one standard deviation reduction in rainfall would result in reduction in height-for-age of a boy by 2.630 of a standard deviation while the height-for-age of a girl child would only decrease by 1.832 of a standard deviation. Similarly, for children between 13 and 19 months of age at the time of deficit, a one standard deviation reduction in rainfall would cause a reduction in heightfor-age which is 1.457 of a standard deviation for a boy, while it the reduction is only 0.171 of a standard deviation for a girl. This could be attributed to the fact that male children, particularly in a rural setting, are more susceptible to weather and consequently nutritional shocks compared to female children.²⁷

Column (6) represents second stage results, which reveal that parents compensate for male child's poor health by investing more in him, while they show no significant reaction toward a girl child. This is an important result because in a rural setting of a developing country like India, when hit by a common weather shock such as a rainfall deficit, a one standard deviation reduction in a boy's height-for-age induces parents to compensate by increasing their allocation of educational expenditure on a boy by approximately 64 per cent. Conversely, parents' behaviour towards a daughter's worse health is shown by coefficient for height-for-age interaction with a female dummy, which is statistically insignificant at 10 per cent level of significance – parents seem to compensate less for girls. These results should be interpreted with caution because the p-value of the equality of the test is 0.465, which suggests that height-forage for males and females is not statistically significantly different from each other and that parents may be behaving similarly toward their male or female children.

3.7 Conclusions

The burgeoning literature attempting to understand whether parental investment responses serve to reinforce or compensate differences in child human capital that arise from early life shocks is far from conclusive (see Almond & Mazumder, 2013 for a review of this debate). I add to this debate by adding a gender dimension to it, which is particularly relevant in case of India due to its longstanding household preference for the number of sons versus daughters.

Using the first two rounds of the Young Lives survey for Andhra Pradesh, India, I investigate gender-differential response of parents' education expenditure to changes in child health as measured by height-for-age. In order to estimate a causal link and to purge height-for-age of its endogenous component, I use an instrumental variable approach, by exploiting exposure to a rainfall deficit caused by the Drought in year 2002 as an instrument for child's physical health.

²⁷ Kim (2010) looks at the impact of rainfall shock on survival of male and female children in West Africa. She finds that given a child is born in the rainy season a positive rainfall shock in the first year has a positive and statistically significant impact on the probability of survival of a male child. This supports the argument that whether it is a positive or a negative weather shock, male children in their early life are more susceptible to these shocks as they have weaker immune systems compared to female children.

My results confirm that parents compensate for changes in child's health caused by negative rainfall shocks during early childhood in Andhra Pradesh. Even though there is no pervasive evidence of gender-differential behaviour, there seems to be a preference to invest more in boys compared to girls in rural households. When faced with a shock, parents protect boys more than girls. This indicates that when hit by a rainfall shock, rural households who subsist on agricultural production may be forced to protect boys more because of the higher future returns that they expect from their sons. This suggests that parents may have an efficiency investment strategy only when seriously resource constrained.

These results could have strong implications for policymaking. If more investments in sons in the face of shocks are a consequence of cultural norms, policymakers may have limited ability to influence. However, if these preferences are explained by resource constraints, there may be opportunities to influence by affecting intra-household resource allocations. Policy interventions could aim to improve position of girls in households through awareness programmes, to improve financial returns for women's education allowing parents to see higher benefits in educating girls and by targeting the poor through income support programmes. My results suggest that better child health induces parents to increase their investments; thus, policies that help in improving child health may also indirectly result in higher parental investments and consequently, better adult life outcomes. Chapter 4

Do inheritance rights empower women and affect their offspring's health?

Do inheritance rights empower women and affect their offspring's health?

Abstract

I estimate the intergenerational impact of women's exposure to the legislative amendments in inheritance law on their children's health. In 2005, there was a national constitutional amendment to the Hindu Succession Act of 1956 in India, which gave females an equal right as males to inherit their parents' ancestral property. However, five states had amended the same Act earlier than the national amendment. I use this exogenous, state-level variation and employ a difference-in-difference estimation strategy. Using the India Human Development Survey, I find a significant improvement in health of children whose mothers were exposed to the amendment, but that even after the reform, substantial gender bias persists. The height-for-age z-score of daughters is significantly lower than that of sons. This pattern of discrimination is only seen for those children whose mothers have less than primary level of education and worsens with the number of children she has. I further explore the mechanisms through which the reform operates and I find that the improvement in child health can be explained by mothers' improved bargaining power in household decisions regarding perinatal health care utilisation and her freedom of movement.

Keywords Inheritance, Gender, Household bargaining, Intergenerational transfers, Child health, India

JEL D23; J16; D13; D64; I14; O53

4.1 Introduction

Women constitute approximately 43% of the agricultural labour force in developing countries and this figure is as high as 70% in South Asia alone (FAO, 2011). Despite a huge contribution in production and income, globally only 15% of the land is owned by women (FAO, 2020).¹ Land is possibly the most important household asset that supports production and provides for nutrition and income security for rural areas in developing countries. One of the main ways for women to own land is through inheritance, but the legal inability to inherit property (as has prevailed in many societies; see Cooper (2008)) not only harms them directly, but may also adversely affect the welfare of their children. This may especially be important in the Indian context where women's lack of inheritance rights, particularly over agricultural land,² may have serious implications for their own as well as their children's welfare³. Thus, there is a need to study the relationship between inheritance rights and their corresponding implications for the next generation.

In this paper, I use an exogenous change in inheritance legislation to estimate the intergenerational impact of Indian inheritance rights reform, which was aimed at improving women's access to ancestral property. In 2005, India introduced a major constitutional amendment to the long-standing Hindu Succession Act of 1956 (hereafter the HSA), which gave women a legal right as men to inherit their parents' ancestral property. Five states had amended this Act prior to the national amendment. Using this legislative change in India's inheritance laws, I seek to answer three main questions. First, does women's exposure to property rights reform have an impact on their children's health relative to those whose mothers were not impacted by the reform?⁴ Second, there exists an inherent bias in South Asia as girls tend to fare worse than boys⁵ thus begging

¹ According to the 2000-2001 Agricultural Census, women make up for 11% of all landholders in India.

² Lack of inheritance rights over agricultural land is a major cause of women's economic disadvantage relative to men in India (Agarwal, 1994).

³ Concentration of resources in women's hands could benefit their children more than those concentrated in men's hands (Lundberg & Pollack, 1993; Thomas, 1990; Ward-Batts, 2008).

⁴ India carries a substantial burden in terms of child health because the improvement in child health outcomes is still not at par with the international targets set by the Millennium Development Goals, affecting social and capital growth in the country (UN, 2018).

⁵ For example, in India, infant and child mortality is higher for girls (D'Souza & Chen, 1980; Rosenzweig & Schultz, 1982); girls in India also seem be at a disadvantage in terms of anthropometric indicators (see, Sen, 1984; Sen & Sengupta, 1983; Behrman, 1988) and in the allocation of nutrients to them within the household (see, e.g., Chen et al. (1981) for Bangladesh; Rosenzweig & Schultz (1982) and Behrman & Deolalikar (1989) for India; and Evenson et al. (1980) and Senauer et al. (1988) for the Philippines). In Pakistan, Alderman and Gertler (1989) found income and price elasticity of the demand for health care to be larger for girls compared to boys. Additionally, Subramanian and Deaton (1990) found that parents allocated more household expenditures toward boys compared to girls.

the question of the evidence for differences by gender in the intergenerational impact of women's exposure to the property rights reforms.⁶ Third, improvement in women's bargaining power⁷ is known to result in better human capital outcomes for their children (Doss, 2006; Quisumbing & Maluccio, 2003); therefore, I evaluate if 'amendments' to the HSA (hereafter the HSAA) improve mothers' bargaining power over household decisions, making it a possible mechanism through which the reform operates.

In the context of developing countries, the existing evidence on benefits of this reform for women is mixed with regard to the impact of inheritance rights. Changes in inheritance laws and their impacts have been studied for Indonesia (Carranza, 2012) and Kenya (Harari, 2017). A nation-wide land registration effort has also been studied for Rwanda (Ali, Deininger, & Goldstein, 2014). Studies that explore the first generation impact of the HSA amendment for India find both positive and negative effects on women who were directly exposed to them. An obvious positive impact includes the HSAA resulting in women's increased likelihood of inheriting ancestral land (Deininger, et al., 2013). Other positive effects include an improvement in alternative transfers to women in the form of more educational attainment and the amounts of dowries given to them (Roy, 2015); improved involvement in more high-paying jobs (Heath & Tan, 2016); and reduced domestic violence (Amaral, 2015). However, even if these amendments challenge the long-standing cultural norms in India, their impacts have not been unarguably positive. Parents often ended up circumventing the new land rights by "gifting" their share of land to their sons (Roy, 2015). The legislation to improve women's inheritance led to increased stress which was often expressed in wife beating and increased suicide levels (Anderson & Genicot, 2015). These inheritance rights also inadvertently increased the costs of having girls; Bhalotra, Brule, and Roy (2018) and Rosenblum (2015) argue that the HSAA reform prompted parents to decrease investments in their daughters, thus increasing female infant mortality and son-selective fertility stopping behaviour.

⁶ Even though more secure inheritance rights should reduce long-standing gender discrimination (Cooper & Bird, 2012), India still fares poorly on the Global Gender Gap Index – its performance is particularly dismal on the health and survival parameter of the Global Gender Gap Index of 2019-2020, ranking 150th out of 153 countries (Ghosh & Sen, 2020).

⁷ Based on the definition for empowerment used in extensive literature, I consider a woman having "bargaining power" over decision-making within her household if she has the freedom to influence the decisions regarding her own and household welfare and regarding her mobility, without other household members' input.

Evidence of such mixed results of the HSAA provides strong basis to explore if its effects persist beyond the exposed first generation mothers into outcomes of their offspring as well. It will be difficult to make a case for such reforms if any positive effects on the exposed mothers are either fully or partly offset by negative effects on their children. Empirical studies that look at the direct or indirect effects of the inheritance rights on women's outcomes are few and often weakly identified, and those that further look at persistence of these effects into next generation's outcomes are scarce. This paper fills this gap in the literature by making two main contributions. Firstly, to the best of my knowledge, this is one of the first studies to explore the effect of mothers' exposure to HSAA on long term health outcomes of their children, measured by height-for-age zscore.⁸ Deininger et al. (2019) and Bose and Das (2017) are the two exceptions, but not only do they just focus on the educational outcomes, there is no consensus in their findings either. While Deininger et al. (2019) find a positive and sustained effect of mothers' exposure to HSAA reform on their children's educational attainment, Bose and Das (2017) do not find any effect on children's education. This discrepancy in results shows that the impact on next generation seems inconclusive and needs to be explored further. Besides, instead of educational outcomes, I focus on child's height-for-age, which is an important measure of child development because parental influences regarding socio-economic background and demographic and behavioural factors can be observed in long term health outcomes. Thus, I provide first insight into persistence of the impact of HSAA into next generation's health outcomes, which can be crucial for policy recommendations. Moreover, by exploring the effects of the reform on child outcomes by gender and household composition, I also contribute to the literature on gender discrimination and son preference in India.

My second main contribution comes from exploring potential mechanisms through which the reform affects child outcomes. It has already been well-established that a mother's bargaining power in household decisions has significant benefits for her

⁸ The very few studies that have looked at outcomes of the offspring of mothers exposed to the reform, focus on education as opposed to health, and because education is considered an alternative to transfer of wealth, the argument in those studies becomes about compensating for disinheritance of land by providing more education to daughters (for papers looking at educational outcomes of next generation, see Deininger et al. (2019) and Bose & Das (2017)). Deininger et al. (2019) also claims to look at health outcomes of children, but lacks detailed information on health status and only looks at it in terms of resources spent on treating diseases that could have been prevented with proper care.

children.⁹ However, for bargaining power to act as a mechanism, it needs to be proven that reforms in inheritance rights increase mothers' bargaining power. Causal evidence proving this relationship is still understudied and the existing studies are somewhat mixed and dependent on the type of empowerment measures used. I contribute to this strand of literature by providing insight into how the bargaining dynamics within the household act as potential mechanisms through which the reform materialises. To this end, I specifically focus on bargaining power in terms of decision-making within the household, using measures related to women's freedom of mobility and autonomy.¹⁰ Overall, most studies have explored the effects of the reform on women without investigating the mechanisms – this study builds on that literature by not only further exploring persistence of the impact of the reform into their children's outcomes, but also proposing a strong case for bargaining power as a mechanism through which those outcomes are affected.¹¹

My identification relies on a difference-in-difference estimation strategy to evaluate the causal impact of legal amendments introduced to the HSA, interpreted as an improvement in mothers' access to property rights. I make use of two sources of variation of mothers' exposure to the reform. The first source of variation comes from five states that passed the reform prior to the national reform in 2005 – these states form part of my treatment group, while the rest of the country is part of the control group. The second source of variation comes from the mothers' timing of marriage. Mothers who were unmarried at the time the reform was passed in their state are part of the treatment group, while already married mothers were excluded from the effects of the reform and are part of my control group. The main underlying assumption is that in the absence of the reform, any difference between outcomes of children born to mothers

⁹ For instance, if mothers placed a greater value on human capital outcomes, they would use their bargaining power (as a result of property rights reform) to direct more resources towards their children's human capital.

¹⁰ When looking at the impact of inheritance rights on bargaining power, other papers that use similar measures for bargaining power include: Heath and Tan (2014) who mainly focus their analysis on labour force participation; Roy (2008) discusses variables related to freedom of movement only, while I also focus on variables representing participation in healthcare related decisions; Harari (2014) studies the impact of women's inheritance rights on similar bargaining power measures in the context of Kenya; Mookerjee (2019) is the only other study for the case of India, which looks at how inheritance rights reform affects similar measures for bargaining power.

¹¹ The only other study that empirically looks at measures of empowerment as mechanisms through which the HSAA affected mothers and their children's education is Deininger et al. (2019), but they use completely different measures of empowerment and different child outcomes. For empowerment measures, they consider mother's education, assets she brought into marriage, and improved access to bank accounts. Unlike the measures I use, these measures do not directly indicate her bargaining power over household decisions. Prior research has shown that different measures of empowerment can be differently related to outcomes (Bloom et al. 2001).

across cohorts would have been the same on average across reform and non-reform states. Similar identification strategy has been used by Anderson and Genicot (2012), Card (2001), Deininger et al. (2013), Lemieux and Card (2001), Rosenblum (2015), and Roy (2015) to estimate the impact on mothers. However, instead of looking at the impact on just mothers, I explore the persistence of the impact on their children employing an extension of this strategy as in Bose and Das (2017) and Deininger et al. (2019). I complement my identification strategy by various additional checks. I estimate the effects of "placebo" reforms on unexposed cohorts and find zero effects.

Drawing upon a rich set of outcomes from the 2004-05 round of the nationally representative Indian Human Development Survey (IHDS), I find that mothers' exposure to amendments to the HSA significantly improved their children's health by approximately 0.234 standard deviations. Looking at the gender differential impact on children of mothers who are eligible for inheritance rights, I find that there are gender differences in the way the household resources are allocated to child health. The heightfor-age z-score of daughters of eligible mothers with less than primary level of education is significantly lower than that of sons. This pattern of discrimination is not seen for children of eligible mothers with at least primary level of education. Additionally, I find that the discrimination against daughters is only visible for children of less educated mothers in three children families, but not in two children families. This finding may be explained by the fact that parents have fewer resources to spend on each additional child and they may favour investing in sons over daughters due to expectation of higher returns in future (Jayachandran & Pande, 2017). More educated mothers do not seem to discriminate between sons and daughter, even if the family size increases. These results are compatible with the findings in previous papers that show that there are gender differences in the allocation of household resources to child health, which vary by gender and level of education of the parent (Lundberg & Pollack, 1993; Thomas, 1994).^{12 13}

¹² According to Lundberg and Pollack (1993), resources concentrated in the hands of mothers do more for children than those concentrated in the hands of fathers. Similarly, according to Thomas (1994), non-labour income concentrated in the hands of mothers has a positive effect on daughter's height, but no significant effect on that of a son, pointing to the fact that gender of the parent is a factor in gender-differential allocation towards child health. Results from Thomas (1994) also show that level of education of the mother has a positive impact on the health of the daughters. This is also supported by Afridi (2010), who posits that mothers with a higher level of education (indicative of her higher status) would have better knowledge and control over household resources and would thus be less inclined to discriminate against their daughters.

¹³ Mother's education and family size may also be affected by HSA, which could pose problems for my analysis. However, I only consider women with completed education and hence it remains unaffected by HSA, but family size could be an issue as the data precludes me to consider women with completed

While some studies have shown that the HSAA did not actually result in more land inheritance for women, the improvement in their children's health could be due to an improvement in their bargaining power as a direct or indirect result of the reform. According to Deininger et al. (2019), the sizeable effects, beyond those linked to HSAAinduced direct resource transfers to women, could reflect a better "fall-back position" of women empowered through the HSAA. I attempt to examine bargaining power in household decisions as a mechanism by considering proxies for women's household bargaining power, based on survey questions on decisions regarding perinatal health care utilisation and mobility. The role of the reform in determining women's bargaining power can help explain its relationship to their children's health outcomes. I find suggestive evidence that the reform makes mothers more likely to receive antenatal and post-natal check-ups and to have their child delivered at a healthcare facility by a trained health professional. I also find that HSAA induced mothers to have more autonomy in decisions regarding her mobility when it comes to travelling unaccompanied, supporting the interpretation that mothers' bargaining power is indeed improved.

The rest of the paper is structured as follows. Section 2 describes the Hindu Succession Act of 1956 and the subsequent amendment to it, providing additional background information on the Indian context. Section 3 outlines the identification strategy. In Section 4, I detail the data and provide a descriptive analysis. Section 5 discusses the results, also highlighting evidence for the possible mechanisms that may underpin my estimations. In Section 6, I present falsification tests to test the validity of the difference-in-difference strategy and discuss additional robustness checks. Finally, I conclude in Section 7.

4.2 Background

4.2.1 Hindu Succession Act (HSA) 1956

The rights for inheritance of property vary by religion in India and apply to all Hindus, Jains, Sikhs and Buddhists.¹⁴ Before the Hindu Succession Act of 1956 (HSA), in the precolonial India, property rights laws were governed by two systems of doctrines,

childbearing. In additional robustness checks in Section 4.6.2, I elaborate on the potential role of fertility in the reform and child health linkage.

¹⁴ Muslims, Christians, Parsis and Jews have their own property laws.

Mitakshara' and *Dayabhaga*' (Agarwal, 1994). The two systems differed in how they categorised an individual's separate property and ancestral property.

Separate property is acquired by an individual on his own, during his or her lifetime. This also includes any property that is not purchased or acquired patrilineally (i.e. inherited from the person's father, paternal grandfather or paternal greatgrandfather). Ancestral property consists of any property that is essentially inherited patrilineally: property, including ancestral homes and land that is passed from the paternal great-grandfather to grandfather to father and so on. It also includes any property that was acquired separately by them, but was merged into the joint ancestral property (Agarwal, 1994).¹⁵

Mitakshara system differentiated between the rights regarding these two types of properties. For rights regarding separate property, the system allowed the patriarch to divide the property among his children as per his wishes because he had the sole right over the separate property. However, the rights to joint ancestral property were limited to a group, called the coparcenary, which includes sons who had a birth right to a share of the joint property. Daughters had no access to their family's joint property (Agarwal, 1994). On the other hand, the Dayabhaga system did not differentiate between the two types of property and considered all property as private property. It allowed the patriarchs to distribute the property as per their will. Under this system, all heirs (both sons and daughters) could have a right over the property, excluding land – daughters still faced discrimination in this regard. This shows that under both systems, women experienced tremendous discrimination with respect to inheriting property, particularly land. As land is the most common form of joint ancestral property in India, these systems were seen to heavily promote gender bias in inheritance rights.

In an attempt to have a more unified system of doctrines and to promote gender equality in inheritance of property, the federal legislation imposed the Mitakshara system under the Hindu Succession Act (HSA) of 1956 (Bloom et al., 1991) and mandated that daughters and sons of Hindu males who died intestate (i.e. without a will or settlement)¹⁶ would have an equal right to inherit their father's separate property only. Daughters were still precluded from any direct inheritance rights to the joint ancestral property (including

¹⁵ The family does not have to be cohabiting in the same household in order to share the ancestral property Agarwal (1994).

¹⁶ The proportion of people who die without a written will in India is as high as around 65%, with rural areas presumed to experience an even higher percentage (Agarwal, 1994; Bhalotra et al., 2018; Deininger et al., 2013; Deininger et al., 2015).



Figure 4.1: The figure shows five reform states (coded in colour) that passed amendments to the HSA 1956. The legend shows the names of the reform states along with the years when they passed those amendments. In 2014, the state of Andhra Pradesh was split into two (Andhra Pradesh and Telangana), but for the purpose of this analysis, the two states are treated as one undivided state of Andhra Pradesh which experienced reform in 1986 as a whole.

land), while sons continued to be entitled to both separate and ancestral property. In India, particularly in rural areas, land is the most common form of joint ancestral property (Roy, 2015). Thus, despite any progress made under the HSA 1956 (which followed the Mitakshara system), not being able to inherit joint ancestral property meant that daughters still suffered from discrimination and barriers to equal inheritance rights.

4.2.2 State Amendments to the HSA 1956

To better the inadequacies of the HSA 1956 and promote gender equality, some states started to amend the HSA 1956 by passing substantively similar amendments.¹⁷ In addition to having an equal share in the separate property, these amendments allowed daughters to have an equal share in the joint ancestral property (including land) as well, so

¹⁷ Both the central and the state governments have legislative authority over inheritance in India.

long as they were unmarried at the time of the reform. The state of Kerala amended the HSA in 1976, followed by the states of Andhra Pradesh in 1986, Tamil Nadu in 1989, and Karnataka and Maharashtra in 1994 (see Figure 4.1).

4.3 Identification Strategy

To understand the causal impact of mothers' access to property rights on child health, I employ a difference-in-difference strategy using the staggered implementation of the amendments to the HSA reform, known as the HSAA in my analysis.¹⁸ I exploit two sources of variation in a woman's property rights reform. The first source of variation comes from the timing of amendment: reform states passed the amendments between 1976 and 1994, prior to the national amendment in 2005. The four treated states are Andhra Pradesh (1986), Tamil Nadu (1989), Karnataka (1994) and Maharashtra (1994).¹⁹²⁰ The second source of variation comes from the woman's timing of marriage: I restrict my sample to Indian women who got married before 2005. Therefore, all sample women who at the time of the reform were not residing in Andhra Pradesh, Tamil Nadu, Karnataka and Maharashtra belong to the control group, while those women residing in the above four states belong to the treatment group. These women are considered treated if they were unmarried by the year of the introduction of the reform in their state and untreated if they were already married by the reform year.²¹

First, I look at the impact of the HSAA reforms on children born to mothers who were exposed as opposed to those who were not exposed to the HSAA reform. Using the difference-in-difference estimation strategy, I use the following benchmark equation to analyse the intergenerational effects:

$$y_{ist} = \beta_0 + \beta_1(HSAA_{ist}) + \mu_d + \delta_t + \gamma X_i + \varepsilon_{ist}$$
(4.1)

¹⁸ Other studies that have employed a difference-in-difference strategy using the state reforms as an exogenous variation include e.g. Roy (2008, 2015), Anderson and Genicot (2015), Deininger et al. (2019), Bose and Das (2017), Rosenblum (2014), Mookerjee (2019), Heath and Tan (2014) and Calvi (2019).

¹⁹ Even though Kerala passed the amendments before 2005, in 1976, it is excluded from my main estimation because the amendments it passed were different in nature and were passed long before they were passed in other treated states, which could give me biased estimates (Sen, 1990). As a robustness check, I re-estimate my main estimation results by including the state of Kerala.

²⁰ According to Bose and Das (2017), there may be a caveat with using variation across reform states if the women migrated from the HSAA state to a non-HSAA state. However, this should not be a concern for me because only approximately 3% of the women migrate, which is close to negligible (Roy, 2015).

²¹ Bose and Das (2017) raise a potential problem with using mother's year of marriage to identify the treated group: gender progressive parents could delay their daughters' marriage while those who want to avoid transferring property to their daughters could advance their marriage. So I include age at marriage as a control in order to account for this potential bias.

where y_{ist} is the outcome variable for height-for-age of child *i* born to mother in state *s* who was married in the year *t*. *HSAA*_{st} is a treatment indicator that takes on the value 1 if the child's mother belongs to the reform state *s* and was unmarried in the year of the reform in state *s* and 0 otherwise. μ_d and δ_t are district and mother's year of marriage fixed effects, respectively. District fixed effects are included to account for differences among regions with comparable socioeconomic characteristics.²² X_i is a vector of individual and household characteristics, which includes child's gender, age and age squared, mother's education, mother's age and her age at marriage, rural vs. urban status, landowning status, scheduled caste or tribe, and below poverty line (BPL) status. ε_{ist} is the error term and all standard errors are clustered at the district level. It must be noted that the estimated effect of the reforms is an average intent-to-treat effect of implementing equal inheritance rights. Thus, these estimates show the effects on children of having a mother who is eligible to the reform amendments.

Given the issue of gender inequality in India, particularly in terms of parental human capital investments, I also analyse the differential impact of the HSAA reform on girls by interacting the $HSAA_{st}$ variable with a dummy for a female child. I estimate the following equation:

$$y_{ist} = \beta_0 + \beta_2 (HSAA_{st} * girl_i) + \beta_3 (HSAA_{st}) + \beta_4 (girl_i) + \mu_d + \delta_t + \gamma X_i + \varepsilon_{ist}$$
(4.2)

where $girl_i$ is a dummy that is equal to 1 for a female child and 0 otherwise. The coefficient of the interaction term (β_2) gives the differential impact of securing property rights on girls' health and β_3 gives the impact of the HSAA on boys' health. $\beta_2 + \beta_3$ is the total effect of HSAA on girls. All other right-hand-side variables and fixed effects remain the same as in equation (4.1).

Next, I explore the mechanisms through which the HSAA reform operates. Better control over income or assets may give women greater bargaining power over household decisions, which is known to result in better health and educational outcomes for their children (Allendorf, 2007; Mishra & Sam, 2016; Rangel, 2006). I hypothesise that securing inheritance rights empowers a woman by giving her more bargaining power within the household. Based on the literature on empowerment, a woman is known to

²² As Bose and Das (2017) point out, controlling for district fixed effects controls for state level unobservable variation as well because districts are administrative divisions of the states.

have "bargaining power" within her family if she has the ability to influence decisions regarding her own and her household's welfare. When she can make those decisions without the input of another household member, she is deemed to have bargaining power over those decisions (Mishra & Sam, 2016).

I use Linear Probability Model to test this hypothesis by estimating the following equation:

$$y_{ist} = \beta_0 + \beta_6(HSAA_{ist}) + \mu_d + \delta_t + \gamma X_{ist} + \varepsilon_{ist}$$
(4.3)

where the outcome variable y_{ist} now denotes a binary outcome measuring empowerment, which takes value 1 if the mother *i* can take independent decisions and zero otherwise. More precisely, I focus mainly on two measures of empowerment, one for the mother's decisions regarding neonatal care (which includes her own and her child's healthcare) and another one for her decisions regarding mobility. I discuss my outcome variables and the motivation behind them more explicitly in the Data section (Section 4). To summarise, outcomes (y_{ist}) based on questions related to neonatal care decisions are dummies denoting (i) whether the woman had antenatal check-up, (ii) whether she received postnatal check-up, (iii) if her child was delivered by a skilled doctor and (iv) if the delivery took place at home versus at a government clinic or private nursing home. For outcomes regarding mobility decisions, y_{ist} takes the value 1 if the woman is allowed to go alone (i) to her friend's house, (ii) to the corner shop, and (iii) to a health centre, and 0 otherwise. Following Mookerjee (2019), both of these measures broadly represent a woman's decision-making ability regarding her health-seeking behaviour and mobility and hence, her associated bargaining power within the family. Moreover, it is important to note that securing inheritance rights could also have an impact on women's empowerment through other possible mechanisms such as mother's education and her fertility, both of which could have an effect on her children's outcomes. I do not explore mother's level of education as a mechanism because my sample only consists of mothers who have already completed their education and so the reform cannot have an effect on it. I do consider a change in fertility as a consequence of inheritance rights as a mechanism. Whether a woman's fertility is a mechanism or not is more of an empirical question and will depend on the direction of the effect. As in previous equations, the term $HSAA_{ist}$ is a dummy variable equal to 1 if the woman *i* was treated by the reform i.e. if she belongs to reform state s and was unmarried at the time

of reform in her state; μ_d are the district fixed effects; δ_t are the year of marriage fixed effects; X_{ist} is a vector of individual and household characteristics; and ε_{ist} is the error term. Standard errors clustered at the district level.

4.4 Data and Descriptive Analysis

I use the 2004-05 wave of the India Human Development Survey (IHDS), which is a nationally representative sample, consisting of 41,554 households from 25 states and Union Territories of India and covers 1,503 villages and 971 urban neighbourhoods. I restrict the sample to Hindu, Sikh, Jain and Buddhist households. My sample of states does not include Jammu & Kashmir, Kerala and the North Eastern states.

From the subsample of married women between 15 and 49 years of age, I use information on their year of marriage to put them in treatment and control groups and refer to this sample as *'sample of women with children'*. The woman has to be in the reform state and unmarried by the year the reform passed in her state for her to be part of the treatment group. The reform states are the ones that passed the amendment before the national amendment was passed in 2005 and include: Kerala (1976), Andhra Pradesh (1986), Tamil Nadu (1989), Karnataka (1994) and Maharashtra (1994). The non-HSAA (control) states include Bihar, Chhattisgarh, Gujarat, Haryana, Himachal Pradesh, Jharkhand, Madhya Pradesh, Orissa, Punjab, Rajasthan, Uttaranchal, and Uttar Pradesh.²³ For instance, Maharashtra introduced the reform in 1994. So, any woman in the reform state of Maharashtra who were already married by the year 1994 are put in the control group. This is done for all states to form treatment and control groups.

For my analysis of estimating the impact of improved inheritance on children's health, I use information on children of the subsample of married women across reform and non-reform states and I refer to this sample as *'sample of children'* in my tables and future analysis. This sample of children consists of 17, 786 males and females who are

²³ In addition to excluding Kerala from the main sample, I also exclude Jammu & Kashmir (J&K), West Bengal and the North-eastern states. I drop West Bengal from the control group because it passed a successful redistributive land reform in the 1970s (around the same time the property rights amendment was passed in the other five states) and followed the Dayabhaga system of property rights that allowed daughters an equal share in all types of property, making it unsuitable as a valid control. I exclude J&K because it was never a part of the HSA 1956. Union Territories are also removed from the sample as they are different from the rest of the states in India in terms of political and administrative nature (Bose & Das, 2017).

between ages 0 and 14 years.²⁴ The dependent variable is child health, which is measured by the height-for-age z-score (HAZ). HAZ is a standard measure of child health and indicates childhood growth, nutrition and environmental factors (Guven & Lee, 2013; Case & Paxson, 2008a, 2008b). The z-score provides information on how a child's height-for-age compares to the world reference population of the same age and gender. I use height and weight information for children under 5 and adolescents to calculate ageand sex-adjusted z-scores according to the UK growth reference charts (Vidmar et al., 2004). HAZ of -2, which means that the child is two standard deviations below the reference population, is considered the cut-off for being stunted and chronically malnourished.

For further empirical analysis of the mechanisms that can possibly explain the impact on child health, I look at the impact of HSAA reform on a woman's bargaining power over household decisions through the following indicators: (a) neonatal care, which includes decisions regarding her own and her child's healthcare, and (b) decisions regarding mobility. The outcomes variables that are based on questions related to neonatal care are dummy variables denoting: (i) whether woman had antenatal check-up, (ii) whether she, her child or both had postnatal check-up, (iii) whether the delivery took place by a skilled doctor, and (iv) whether the delivery took place at home as opposed to at a government clinic or a private health centre. In patriarchal societies like India, husband's supportive input in decision-making is considered a key component in increasing utilisation of maternal and child care services (Chattopadhyay, 2011). Gender inequality and poor communication between the couple constrain women's access to healthcare services. Thus, I use her decision-making regarding healthcare utilisation as a measure of bargaining power in the household because it reflects reduced inequality and better communication between the couple. Another dimension of women's bargaining power is whether she can visit certain places alone without being escorted (Heath & Tan, 2016). For outcomes based on questions related to mobility decisions, I use the woman's self-reported answers to (i) weather she can visit her friend's house alone, (ii) whether she can visit the corner shop alone, and (iii) whether she can visit the health centre alone²⁵. These dummy variables represent if she has a say in any of these decisions. Besides, these locations also present opportunities to spend money, hence the fact that she can visit

²⁴ I dropped some observations which have missing information on height, weight, age of the children and on parental education, age and household characteristics.

²⁵ Visiting the health centre alone indicates her decision-making ability regarding her own health and regarding her mobility.

these alone also reflects her ability to control household purchases, and hence is indicative of her intra-household bargaining power as well. For decisions regarding a woman's reproductive choices, I look at her fertility, which is measured by the total number of children borne by her. I restrict my sample to children aged between 0 and 14, but it is possible that an eligible woman has children younger or older than 0-14 years, and hence I include her total fertility i.e. the total number of children borne by her.²⁶

Main control variables in all my estimations include mother's age, her age at marriage and her number of years of completed education. I include dummy variables for being a Hindu and whether they belong to a low caste (i.e. Scheduled Caste or Scheduled Tribe in India). Religion and caste are known to play critical roles in societal and legal systems in South Asia, especially when it comes to women's property ownership and decision-making roles (see Basu & Koolwal, 2005; Kabeer, 1999; Trommlerová et al., 2015). I also control for whether she belongs to a household that owns any land, is located in a rural area, and whether they live below the poverty line. I control for woman's residence in a rural or urban area because conventional barriers to women's empowerment tend to be stronger and more difficult to challenge in rural areas where women are often relegated to subservient roles compared to urban areas (see Kabeer, 1999; Kishor & Gupta, 2004; Trommlerová et al., 2015).

Table 4.1 shows descriptive statistics for full sample, non-reform and reform states. All non-reform states are part of the control group, while reform states are further divided into treatment and control groups, based on the year of marriage of the women.²⁷

Panel A consists of descriptive statistics for *sample of children* 0-14 years born to married women in the reform and non-reform states who have at least one child. It shows the statistics for the key outcome variable (i.e. HAZ) and main control variables in my analysis. Children in non-reform states seem to have slightly worse health (as measured by HAZ) compared to children in the reform states. Gender mix is comparable in both reform and non-reform states, with approximately 48% girls in both.

Column 1 in Panel A of Table 4.1 shows that about 11 per cent of children have mothers who were exposed to the HSAA reform in my total sample of 16,746 children.

²⁶ For example, suppose an eligible woman has three children aged 9, 11 and 16 years. My estimation sample would only include 9 and 11 year old children, but the total fertility variable will also be able to account for the 16 year old child.

²⁷ Unmarried women by the year of the reform are part of the treatment group, while those married by that time are part of the control group.

	(1)	(2)	(3)	(4)	(5)
	Full sample	Non-reform states –	Reform State		
D. 14 M. 14 14 14	-		All	Treated	Contro
Panel A: Main outcome and		· · · · =	,	1.055	1 505
HAZ	-1.449 (1.848)	-1.510 (1.891)	-1.444 (1.703)	-1.255 (1.948)	-1.595 (1.460
Mother married post- HSAA	$\begin{array}{c} 0.106 \ (0.308) \end{array}$	$\begin{pmatrix} 0\\(0) \end{pmatrix}$	$\begin{array}{c} 0.445 \ (0.497) \end{array}$	$\begin{pmatrix} 1\\(0) \end{pmatrix}$	$\begin{pmatrix} 0\\(0) \end{pmatrix}$
Girl	$\begin{array}{c} 0.478 \ (0.500) \end{array}$	$\begin{array}{c} 0.477 \ (0.499) \end{array}$	$\begin{array}{c} 0.481 \ (0.500) \end{array}$	$\begin{array}{c} 0.480 \\ (0.500) \end{array}$	$0.483 \\ (0.500$
Child's age (in months)	$72.929 \\ (42.408)$	$70.898 \\ (42.750)$	$79.381 \\ (42.949)$	$58.519 \ (38.018)$	96.092 (34.487)
Child's age squared	$\begin{array}{c} 49.423 \\ (43.080) \end{array}$	$47.596 \\ (42.961)$	$55.226 \\ (42.949)$	$33.813 \ (37.077)$	72.378 (39.498
Mother's level of education (years)	$3.819 \\ (4.528)$	$3.735 \\ (4.587)$	$4.085 \\ (4.327)$	$\begin{array}{c} 4.179 \\ (4.343) \end{array}$	4.009 (4.314
Mother's age (years)	$30.686 \\ (5.834)$	$30.740 \\ (6.034)$	$30.513 \\ (5.141)$	$27.418 \\ (4.343)$	32.992 (4.684
Mother's age at marriage (years)	$17.307 \ (3.103)$	$17.177 \ (3.049)$	$17.722 \\ (3.236)$	$18.583 \\ (3.395)$	17.033 (2.926
No. of children	$3.062 \\ (1.466)$	$3.179 \\ (1.506)$	$2.689 \\ (1.259)$	$2.492 \\ (1.148)$	2.846 (1.322
Hindu $(0/1)$	$\begin{array}{c} 0.955 \ (0.206) \end{array}$	$0.951 \\ (0.216)$	$0.969 \\ (0.173)$	$\begin{array}{c} 0.984 \ (0.124) \end{array}$	$0.957 \\ (0.203)$
Rural $(0/1)$	$\begin{array}{c} 0.726 \ (0.446) \end{array}$	$0.736 \\ (0.441)$	$\begin{array}{c} 0.692 \\ (0.462) \end{array}$	$\begin{array}{c} 0.655 \\ (0.476) \end{array}$	$0.721 \\ (0.448)$
Own Land $(0/1)$	$\begin{array}{c} 0.501 \\ (0.500) \end{array}$	$\begin{array}{c} 0.519 \ (0.500) \end{array}$	$\begin{array}{c} 0.442 \ (0.497) \end{array}$	$\begin{array}{c} 0.370 \\ (0.483) \end{array}$	$0.500 \\ (0.500)$
Low caste (SC/ST) $(0/1)$	$\begin{array}{c} 0.401 \ (0.490) \end{array}$	$\begin{array}{c} 0.423 \ (0.494) \end{array}$	$\begin{array}{c} 0.333 \ (0.472) \end{array}$	$\begin{array}{c} 0.318 \\ (0.466) \end{array}$	$0.346 \\ (0.476$
Below poverty line $(0/1)$	$\begin{array}{c} 0.300 \ (0.458) \end{array}$	$\begin{array}{c} 0.317 \ (0.465) \end{array}$	$\begin{array}{c} 0.247 \ (0.433) \end{array}$	$\begin{array}{c} 0.231 \\ (0.421) \end{array}$	$0.261 \\ (0.439)$
Observations	16,746	12,737	4,009	1,783	2,226
Panel B: Women's outcome	variables (Sample of wom	en with children)	
Antenatal care	$\begin{array}{c} 0.764 \ (0.425) \end{array}$	$\begin{array}{c} 0.715 \ (0.451) \end{array}$	$0.947 \\ (0.224)$	$\begin{array}{c} 0.964 \ (0.187) \end{array}$	0.909 (0.288)
Postnatal care	$\begin{array}{c} 0.317 \ (0.465) \end{array}$	$0.277 \\ (0.447)$	$\begin{array}{c} 0.470 \ (0.499) \end{array}$	$\begin{array}{c} 0.479 \ (0.500) \end{array}$	0.448 (0.498
Delivery by a skilled doctor	$\begin{array}{c} 0.396 \\ (0.489) \end{array}$	$\begin{array}{c} 0.332 \\ (0.471) \end{array}$	$0.635 \\ (0.482)$	$\begin{array}{c} 0.674 \\ (0.469) \end{array}$	0.548 (0.498
Delivery at home	$\begin{array}{c} 0.575 \ (0.494) \end{array}$	$0.636 \\ (0.481)$	$0.346 \\ (0.476)$	$\begin{array}{c} 0.299 \\ (0.458) \end{array}$	0.452 (0.498
Can visit friend's house alone?	$\begin{array}{c} 0.741 \\ (0.438) \end{array}$	$\begin{array}{c} 0.705 \ (0.456) \end{array}$	$\begin{array}{c} 0.827 \ (0.378) \end{array}$	$\begin{array}{c} 0.799 \ (0.401) \end{array}$	0.838 (0.368)
Can visit corner shop alone?	$\begin{array}{c} 0.750 \ (0.433) \end{array}$	$\begin{array}{c} 0.718 \ (0.450) \end{array}$	$0.826 \\ (0.379)$	$\begin{array}{c} 0.804 \ (0.397) \end{array}$	$0.838 \\ (0.371)$
Can visit health centre alone?	$\begin{array}{c} 0.727 \ (0.446) \end{array}$	$\begin{array}{c} 0.673 \ (0.469) \end{array}$	$0.843 \\ (0.353)$	$\begin{array}{c} 0.820 \ (0.384) \end{array}$	0.868 (0.339
Observations	6,208	6,208	6,208	6,208	6,208

Table 4.1: Descriptive Statistics on relevant outcomes in different samples

Source: Author's analysis based on IHDS. *Notes*: The table reports sample means followed by standard deviations in parentheses for each variable indicated in the first column.

Children in reform and non-reform states differ in terms of their mothers' number of average years of education; within the reform states, there is no clear difference in the number of years of education between the treatment and control group – mothers who were exposed to the HSAA as well as those in the control group have about 4 years of education. Deininger et al. (2013), Roy (2015) and Bose and Das (2017) find that there is an increase in women's education due to the HSAA, plausibly because instead of devolving physical capital (like land) to these women, they are compensated with more human capital. It may also be because the average income in the household with a treated woman may be higher than the average income in a household without a treated woman (Bose & Das, 2017). I do not see the same in my descriptive statistics because I have removed from my sample all those women who had not yet completed their education. Mothers in reform and non-reform states are similar in average age (30 years) and age at marriage (17 years), as well as in terms of proportion of Hindus. Reform states have 97% of Hindu women compared to 95% in non-reform states. Approximately 52% of households in the non-reform states own any land, while 42% own land in the reform states. The proportion of women who belong to a low caste (i.e. a Scheduled Caste or a Scheduled Tribe in India) is slightly higher in non-reform states (42%), compared to 33% in reform states. There seems a difference in the proportion of households who live below the poverty line -32% live in poverty in the non-reform states, while 25% do so in the reform states.²⁸

If actual or potential increases in inheritance due to the HSAA translated into better household bargaining power for females, my variables measuring intra-household bargaining power should capture some systematic differences between reform and nonreform states. There is again some descriptive evidence to support this hypothesis. Panel B in Table 4.1 shows descriptive statistics for the *sample of women with children*. It shows the proportion of women 15-49 years old who report a positive outcome for their bargaining power in household decision-making (e.g. in health-seeking behaviour and in mobility decisions as well as their fertility decisions). On average, all women in reform states (column 3) have access to better neonatal care, enjoy more freedom of mobility and more

²⁸ Table C1 in the Appendix further shows that the households in the treatment group are statistically significantly different in comparison to the control group in terms of mean household characteristics, as shown by Column 3.

unrestricted visits as well as have reduced fertility in comparison to those in non-reform states (column 2).²⁹

4.5 Results

In this section, I look at the impact of mothers' exposure to amendments in the property rights reform on their children's health. I further explore whether mothers' improved bargaining power in household decisions due to the reform acts as a mechanism for better health outcomes of their children.

4.5.1 Intergenerational impact of property rights reform: effect on children's health

Does the impact of the amendment to the HSA reform on women translate to their children's outcomes? Table 4.2 shows empirical results for the relationship between mother's improved property rights status on her child's health outcome as measured by the HAZ. These results focus on children between ages 0 and 14.

In Table 4.2, column 1, I start by estimating equation (4.1) to evaluate the average intergenerational impact of a mother's exposure to the HSA reform status on her child's HAZ outcome, without any mother or household controls. Column 1 shows that on average, there is a 0.268 standard deviation statistically significant increase in HAZ for children born to mothers exposed to the HSAA reform in comparison with those not. As I control incrementally for covariates (starting with observable individual and household characteristics in column 2 to adding mother's year of marriage and district fixed effects in column 3), the impact on child HAZ slightly attenuates in magnitude, but remains positive and statistically significant throughout. Once I have included all controls in addition to mother's year of marriage and district fixed effects (column 3), I find that amongst households where mothers are exposed to the reform, children see a significant increase of 0.227 standard deviations in their health. These results seem to be better for more educated mothers and worse for those households who live in rural areas and those who live below the poverty line. This suggests an overall improvement in health of

²⁹ It must however be noted that I only observe the use of care and the decisions taken regarding their mobility and fertility, but not who makes decisions about them.

neight-for-age z-score (TIAZ)							
	(1)HAZ	(2) HAZ	(3)HAZ				
Reform State*Mother married post-HSAA	0.268^{***} (0.079)	0.252^{***} (0.071)	0.227^{***} (0.073)				
Child's age (in months)	(0.013)	-0.038^{***} (0.002)	-0.037^{***} (0.002)				
Child's age squared		0.032^{***} (0.002)	0.031^{***} (0.002)				
Child's gender (girl=1)		-0.105^{***} (0.026)	-0.100^{***} (0.025)				
Mother's education		0.040^{***} (0.004)	0.041^{***} (0.004)				
Mother's age		0.016^{***} (0.003)	-0.015 (0.035)				
Mother's age at marriage		-0.006 (0.009)	0.023 (0.038)				
Hindu (yes=1)		-0.187^{**} (0.086)	-0.152 (0.094)				
Rural (yes=1)		-0.187^{***} (0.051)	-0.175^{***} (0.054)				
Own land $(yes=1)$		0.054 (0.034)	0.059^{\star} (0.035)				
Low caste (yes=1)		-0.039 (0.036)	-0.038 (0.035)				
Below poverty line status (yes=1)		-0.129^{***} (0.047)	-0.131^{***} (0.047)				
Constant	-1.523^{***} (0.021)	-0.487^{*} (0.248)	0.599 (1.086)				
Controls	No	Yes	Yes				
Fixed effects	No	No	Yes				
R-squared	0.002	0.066	0.076				
Observations	16,746	16,746	16,746				

Table 4.2: Effect of the Hindu Succession Act Amendment (HSAA) on children's height-for-age z-score (HAZ)

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. Robust standard errors are clustered at the district level and appear in parentheses. The last column includes district and mother's year of marriage fixed effects. This table considers the *sample of children* 0-14 years old with mothers who had already completed their education by the year of reform in their state.

children, as a result of the reform, but that a woman's own education and the family's economic status also have a role to play.³⁰

Next, to understand if the amendment-induced impact differed by gender, I estimate equation (4.2). In Table 4.3, once I have controlled for covariates and fixed effects, I find that the HAZ of sons in treated households is 0.268 standard deviations higher when compared to HAZ of sons in untreated households (Column 1, Row 1). The interaction term shows that the difference between sons and daughters (Column 1, Row 2) even though negative, is not statistically significant, and implies that there is no

³⁰ These results are also robust to including district-specific time trends, as shown in Column 4 of the Appendix Table C8.
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
VARIABLES	Main effect	Any children	2 children	3 children	Any children	2 children	3 children
		Mother's	education: less	than primary	Mother's educ	cation: at leas	st primary
Reform State*Mother married post-HSAA	$\begin{array}{c} 0.268^{***} \\ (0.083) \end{array}$	$\begin{array}{c} 0.387^{***} \\ (0.110) \end{array}$	0.260^{**} (0.122)	$\begin{array}{c} 0.557^{***} \ (0.190) \end{array}$	$0.110 \\ (0.120)$	$\begin{array}{c} 0.120 \\ (0.183) \end{array}$	$\begin{array}{c} 0.048 \ (0.310) \end{array}$
Girl*Reform State*Mother married post-HSAA	-0.0839 (0.0872)	-0.144 (0.129)	-0.194 (0.204)	-0.365^{*} (0.213)	-0.0234 (0.149)	$\begin{array}{c} 0.0970 \ (0.228) \end{array}$	-0.121 (0.283)
Girl	-0.0908^{***} (0.0259)	-0.0992^{***} (0.0273)	-0.0636 (0.0495)	-0.0149 (0.0525)	-0.0599 (0.0584)	-0.114 (0.102)	-0.0443 (0.103)
Constant	0.603 (1.084)	$\begin{array}{c} 0.418 \\ (1.577) \end{array}$	$\begin{array}{c}(10100)\\1.590\\(3.524)\end{array}$	$\begin{array}{c} (0.0520) \\ 2.949 \\ (2.859) \end{array}$	-0.0250 (1.403)	-0.651 (2.678)	(3.336) (2.657)
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.085	0.062	0.082	0.073	0.111	0.134	0.131
Observations	17,786	11,375	2,869	$3,\!492$	6,411	2,741	1,615

Table 4.3: Gender differential impact of the Hindu Succession Act Amendment (HSAA) on children's height-for-age z-score (HAZ)

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. Col. (1) considers the *sample of children 0-14 years*; while the remaining columns consider different types of subsamples as defined at the top of each column. All samples include children with mothers who had already completed their education by the year of the reform in their state.

differential impact of HSAA between HAZ of sons and daughters. This seems to suggest that on average the households are gender neutral.

At first glance, my results indicate no evidence of gender discrimination in intergenerational transmission of property rights reform. However, due to overwhelming evidence of son preference in Indian households, it is important to probe into it further.³¹ Based on the role of mother's education in reducing the effect of son preference and the bias against daughters, I look at heterogeneity in my results by mother's level of education (Bose, 2012). In Table 4.3, column 2 shows the gender differential impact of the HSAA on HAZ of those children whose mothers have not completed primary level of education (i.e. grade five) while column 5 shows results for children whose mothers have completed at least primary level of education. As expected, the results in columns (2) and (5) do not indicate gender disparity by mother's level of education, suggesting that mother's education level is not a channel through which the HSAA impacts children.

Furthermore, as family size grows in size with more number of children, parents may become unwilling to spend scarce resources on a daughter given that there will be no future economic returns for her birth family, particularly after her marriage (Banerjee 1998). To see if this expectation uncovers a different pattern from the one previously observed for full sample households, I separately compare homogenous households with two and three children within the 0 and 14 age group born to less or more educated mothers.³² Interestingly, for households where the mother does not have primary education, the discrimination between sons and daughters can be noticed in families with three children, but not in those with just two children – for these households, HAZ of treated daughters is statistically significantly lower than HAZ of treated sons by 0.365 standard deviations (Table 4.3, Column 4, Row 2). I do not find any pattern of discrimination by the size of family for more educated mothers. This kind of discrimination in larger families can be explained by unequal allocation of resources within the household. With each additional child, parents may have lesser resources available to spend on their children's food and healthcare. Thus, the share spent on boys may be higher than girls due to expectation of higher returns on investments in the

³¹ Health outcomes and parental investments seem to be in favour of sons compared to daughters in South Asia and it has been frequently documented in e.g. Bhalotra & Attfield (1998), Biswas & Rose (2010), Dancer et al. (2008), Gupta (1987), Hussain et al. (2000), Jayachandran & Pande (2017), Mishra et al. (2004), and many others.

³² Das & Gupta (2017) and Jayachandran & Pande (2017) find that the modal number of children in India is between 2 and 3 children. Thus, I restrict my sample to reflect this family size.

wellbeing of sons as compared to daughters in India. Column 4 shows that the magnitude of the height-for-age deficit is also much bigger for daughters compared to sons in three children families. This is supported by Jayachandran and Pande (2017) who find that the height deficit in children increases with higher birth order and that the investments in successive pregnancies also decline in India. However, this pattern of allocation seems to be dictated by mothers' level of education – more educated mothers have better knowledge and more control over allocation of household resources (Afridi, 2010).

4.5.2 Evidence of bargaining power as a mechanism

According to the literature, better bargaining power of women in household decisions leads to better intergenerational outcomes;³³ more bargaining power in decisions is known to come from women having higher education and making a greater contribution to household wealth. Women's ability to inherit could also bring about an improvement in their bargaining power, which they could use to shift human capital investments in children toward their own preferences. Previous papers have found that mothers tend to attach higher value to children's welfare and therefore an increase in their bargaining power leads to an increase of investments in children's human capital (Behrman, 1990; Doepke & Tertilt, 2014; Strauss et al., 2000). Building on this pre-existing evidence, I aim to look at the effect of the amendments to the HSA on bargaining power of the mothers exposed to the reform, making it a potential mechanism through which the reform affects child health.

First, I start by investigating the impact of HSAA on mother's own health-seeking behaviour, which includes decisions regarding receiving antenatal and post-natal checkup, receiving trained assistance at child birth and choosing a safe delivery place versus delivering at home. All these decisions represent out-of-pocket costs, which are still unaffordable for many. In such circumstances, women's access and utilisation of perinatal healthcare also reflects her bargaining power in the household. The regression results reported respectively in columns 1, 2, 3 and 4 of Table 4.4 show that mothers exposed to the benefits of the HSAA reform have better bargaining power in household when it comes to decisions regarding perinatal healthcare utilization. Column 1 shows that women are 19 percentage points more likely to receive antenatal check-ups if they were

³³ For some papers that look at this relationship, see Afridi, Mukhopadhyay and Sahoo (2016), Bruins (2017), Duflo (2003), Lépine and Strobl (2013), Lundberg, Pollak and Wales (1997), Qian (2008), and Ward-Batts (2008).

	permutan mea	unicale admoath		
	(1)	(2)	(3)	(4)
VARIABLES	Antenatal check-up	Postnatal check-up	Delivery by a skilled doctor	Delivery at home
Reform State*Mother married post-HSAA	0.190^{***} (0.021)	$\begin{array}{c} 0.146^{***} \\ (0.032) \end{array}$	$\begin{array}{c} 0.266^{***} \ (0.028) \end{array}$	-0.261^{***} (0.026)
Education	0.018^{***} (0.002)	$\begin{array}{c} 0.014^{***} \\ (0.002) \end{array}$	0.023^{***} (0.001)	-0.024^{***} (0.002)
Age	$0.014 \\ (0.011)$	$\begin{array}{c} 0.017^{*} \ (0.009) \end{array}$	$0.015 \\ (0.011)$	-0.018 (0.012)
Age at marriage	-0.011 (0.011)	-0.007 (0.009)	-0.005 (0.012)	$\begin{array}{c} 0.005 \ (0.012) \end{array}$
Hindu	-0.092^{***} (0.022)	-0.050 (0.031)	-0.040 (0.026)	$\begin{array}{c} 0.063^{**} \\ (0.030) \end{array}$
Rural	-0.057^{***} (0.016)	-0.036 (0.025)	-0.193^{***} (0.020)	0.210^{***} (0.020)
Own any land	-0.009 (0.015)	-0.031^{**} (0.013)	-0.047^{***} (0.011)	0.047^{***} (0.014)
Low caste	-0.022^{*} (0.013)	-0.029^{*} (0.016)	-0.053^{***} (0.012)	0.048^{***} (0.012)
Below poverty line	-0.042^{**} (0.018)	-0.001 (0.013)	-0.093^{***} (0.015)	0.099^{***} (0.013)
Constant	-0.143 (0.401)	-0.532^{*} (0.308)	-0.111 (0.417)	$1.233^{***} \\ (0.424)$
Fixed effects	Yes	Yes	Yes	Yes
R-squared	0.216	0.116	0.282	0.297
Observations	7,449	7,449	7,449	7,449

Table 4.4: Effect of the Hindu Succession Act Amendment (HSAA) on decisions about perinatal healthcare utilisation

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. Table considers *sample of women with children*, with no missing data on variables about perinatal healthcare utilization. The sample only includes women who had already completed their education by the year of the reform in their state.

exposed to the reform. There is also a higher probability (approximately 15 percentage points) for treated mothers to get post-natal check-ups (Column 2, Table 4.4). These two results seem to reflect a direct association with child health – better access and utilisation of health care before and after birth is associated with better child health. Furthermore, there is about a 27 percentage points higher likelihood for mothers, secured by the reform, to have their child delivered by a trained medical professional (column 3, Table 4.4). The chances of mothers delivering at home instead of at a medical institution or health centre – also known as an institutional delivery – reduce by approximately 26

percentage points. Both, institutional delivery and institutional or home delivery assisted by a skilled medical professional are termed as 'safe delivery' (World Health Organization, 2006). Again, safe delivery practices should have a positive impact on child health, thereby making improved bargaining power in health-seeking decisions a credible mechanism.

Second, I look at women's autonomy regarding her mobility as another evidence of her bargaining power in the household. Permission regarding mobility decisions captures the aspect of female autonomy. Existing literature provides evidence for why female autonomy can have a positive impact on child health. Mothers who are not physically constrained in terms of being escorted are known to ensure suitable and timely treatment and vaccination for their children; this has a positive effect on overall health of children.³⁴ Women's freedom of mobility allows social interactions, which may increase their exposure to new healthcare knowledge and healthy practices (Smith et al., 2003). It can also give her access to local markets, medicine shops, and health centres that can indirectly impact her own as well as her children's health. However, in a gender-stratified society like India, one of the ultimate ways to curtail women's autonomy is by means of controlling their physical movement. Women, regardless of their age and marital status, are still not allowed to leave the house without the permission of an elderly family member and/or husband. In the events when they are allowed to leave the house, they are rarely unescorted and their movement is restricted in duration, distance and purpose (Chakrabarti, 2019; Mandelbaum, 1986). Women's freedom of mobility is closely linked to their bargaining power within the household and an improvement in their status in society. Table 4.5 shows the impact of HSAA reform on outcomes related to a woman's ability to decide if she wants to go to her friend's house, to the corner shop or to a health centre by herself. The coefficients for the treated sample are positive and statistically significant for all the outcomes, with women exposed to the reform being 14.1 percentage points more likely to be able to visit a friend's house, 12.3 percentage points more likely to visit the market or the corner shop and 17.7 percentage points more likely to visit a health centre alone.

Lastly, women's fertility is another mechanism through which inheritance rights could possibly have an impact on their bargaining power within the household. In

³⁴ For instance, Nobi (2018) finds that Nigerian mothers' mobility, represented through permission indices, has a positive impact on their children's chances of receiving vaccination for the six killer diseases and in participating in the Vitamin A drive.

	(1)	(2)	(3)
VARIABLES	Can visit friend's house alone?	Can visit corner shop alone?	Can visit health centre alone?
Reform State*Mother married post-HSAA	0.141^{***} (0.023)	0.123^{***} (0.022)	0.177^{***} (0.017)
Education	$0.009^{stst} (0.001)$	0.005^{***} (0.001)	0.010^{***} (0.001)
Age	$0.011 \\ (0.010)$	0.024^{**} (0.011)	-0.005 (0.008)
Age at marriage	-0.002 (0.010)	-0.011 (0.011)	0.021^{***} (0.008)
Hindu	-0.031 (0.020)	$egin{array}{c} 0.022 \ (0.030) \end{array}$	-0.058^{***} (0.019)
Rural	-0.015 (0.020)	-0.032 (0.023)	-0.046^{**} (0.021)
Own any land	-0.040^{***} (0.012)	-0.061^{***} (0.013)	-0.069^{***} (0.012)
Low caste	$0.009 \\ (0.010)$	$0.020^{st} (0.009)$	$0.009 \\ (0.008)$
Below poverty line	$0.037^{stst} \ (0.013)$	0.028^{**} (0.014)	-0.022^{**} (0.010)
Constant	$\begin{array}{c} 0.412 \\ (0.348) \end{array}$	$\begin{array}{c} 0.0755 \ (0.381) \end{array}$	$\begin{array}{c} 1.107^{***} \\ (0.289) \end{array}$
Fixed effects	Yes	Yes	Yes
R-squared	0.089	0.088	0.127
Observations	$19,\!250$	$19,\!250$	$19,\!250$

Table 4.5: Effect of Hindu Succession Act Amendment (HSAA) on
decisions regarding mobility

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. Table considers *sample of women with children*, with no missing data on variables about mobility. The sample only includes women who had already completed their education by the year of the reform in their state.

Appendix Table C9 I look at the effect of HSAA reform on women's total fertility and I find a statistically significant reduction in the total number of children borne by the woman. This result is in line with Dyson and Moore (1983) who find that women's control over productive resources results in drastic reduction in fertility. Fewer children and the associated bargaining power, would mean that women could concentrate more resources towards each child, as models of the quantity-quality trade-off predict, eventually having a positive impact on their health.

Overall, this analysis suggests that there is a positive impact of the reform across both dimensions of a woman's decision-making – perinatal healthcare utilisation and freedom of mobility and a negative impact on her fertility. Women with greater bargaining power could eventually also improve the health of their children through better human capital and health investments in them. This makes mothers' bargaining power in household decisions a plausible mechanism through which the reform affects child health outcomes.

4.6 Robustness checks

4.6.1 Robustness checks of the parallel trend assumption

The identifying assumption for this analysis is that the parallel trends assumption holds true. This assumption requires that in the absence of the HSAA reform, the trend in HAZ of children born to the treatment group of women – includes Hindu, Sikh, Buddhist, and Jain women in the reform states who were unmarried at the time of the reform – should not be any different from those born to the control group of women.

One challenge in this analysis, as is also pointed out by Rosenblum (2014) and Bose and Das (2017) for the case of India, is that the treatment and control groups are substantially different from each other in terms of mean household characteristics (see Table C1 in the Appendix for differences between treatment and control groups in the non-reform and reform states in panel A and between treatment and control groups within the reform states in panel B); however, in order to control these baseline differences, I use district and year of marriage fixed effects that account for cross-place and cross-time differences. Even though the identifying assumption is not that the two groups are similar in mean characteristics, it is important to test the validity of the difference-in-differences strategy which requires that the parallel trend assumption holds true.

Based on the strategy used by Deininger et al. (2013), I conduct a test for parallel trend in the pre-reform period by allowing for lag and lead effects of the reform, i.e. by including three dummy variables, a first dummy taking value 1 for treated mothers who married *between 1 and 6 years before* the introduction of reform in their state, a second dummy taking value 1 for treated mothers who married *between 0–5 years after* the reform introduction, and a third dummy taking value 1 for treated mothers who married *at least 6*

years after the reform (the left-out baseline dummy is taking value 1 for treated mothers married more than 6 years before the reform introduction). Because there is no reform effect for treated women who got married between 1 and 6 years before the reform and for treated women who married more than 6 years before the reform, the differential effect of the first dummy with respect to the baseline dummy can capture only a difference in pre-reform trends between the treated and control states. Results in Appendix Table C2 show no statistically significant effect for the first dummy and therefore support the hypothesis of parallel trends in the pre-reform period. This placebo test substantiates our reliance on using the difference-in-difference strategy to estimate a causal effect. For the post-reform effects captured by the second and third dummy variables, I find statistically significant positive effects. Children born to mothers who were exposed to the reform in the five and six or more years after the reform see an improvement in their HAZ. The F statistic for equality of pre- and post-coefficients is 4.57 (p = 0.0142), suggesting that the coefficients before and after the reform are statistically different from each other. But the effect in both periods post-HSAA (second and third dummy effects) is comparable with the *F* statistic for equality of coefficients between the two postperiods equal to 0.79 (with a p - value of 0.3784), suggesting that the coefficients are not statistically different from each other supporting my benchmark model (4.1) where I assumed that the reform effect be the same for all mothers in treated states who got married after the introduction of the reform.

As additional support for my identification strategy, I report three more falsification tests. First, in the pre-treatment period, when none of the observations were affected by the reform yet, HAZ should be the same between the treated and control groups. Following Rosenblum (2014) and Bose and Das (2017), in Appendix Table C3, column 1, I give the treatment group in the four reform states a false reform start date of January 1983 and restrict the data until the start of actual reforms in their respective states. The year 1983 represents a pre-reform period because no inheritance reforms were introduced in any states before the year 1986. Using an equation analogous to equation (1), I find that the coefficient on the treatment variable is much smaller and negative, but not statistically significant. This shows that HAZ is not different between the treatment and control groups in the pre-treatment period.

Second, I estimate equation (4.1) only for children in Muslim households who, by definition, should be unaffected by the Hindu Succession Act. This test will allow me to

rule out any possibility of coefficient estimates erroneously picking up time trends in patterns of inheritance which should be independent of the amendments to the reform Deininger et al. (2013). My results in Appendix Table C3 (Row 2, Column 2) show that the coefficient is smaller and positive, but lacks statistical significance, confirming that there was no impact of the HSAA reform on HAZ of non-Hindu children.

Third, I use Hindus in the non-reform states, with three different cut-offs for reform years.³⁵ This is a double falsification test as it uses a) a group (non-reform states) that was unaffected by the treatment and b) by using placebo years for reforms as the entire period before and after these placebo years is a pre-treatment period for these non-reform states.³⁶ Appendix Table C4 shows that for children in households that own any land, the timing of mother's marriage either before or after either 1986 (Column 1), or 1989 (Column 2) or 1994 (Column 3) has no effect on their HAZ outcomes.

Thus, findings of a common pre-trend in all these tests provide support for the use of a difference-in-differences strategy and allay any concerns regarding unobserved factors driving my main results.

4.6.2 Additional robustness checks

To check the robustness of my results, I re-estimated them by adding the state of Kerala to the reform states. I had initially omitted Kerala from my main estimation because a) it introduced a different kind of reform in the form of the Kerala Joint Hindu Family System (Abolition) Act and b) it introduced the reform in 1976, which was much earlier compared to the reforms first introduced in other states in 1986. Kerala abolished the joint family property system altogether in favour of the system where all family members, irrespective of their gender, could have separate shares in joint property (Agarwal, 1994). This was considered very gender-progressive at the time. However, since the spirit of this amendment passed in Kerala was similar to those passed by other reform states, and could be expected to have a favourable effect on women's inheritance, I re-estimate my results which are robust to inclusion of Kerala, as shown in Appendix Table C5.

³⁵ These three reform years of 1986, 1989 and 1994 represent the years when the actual reform was made effective in the reform states, with Andhra Pradesh introducing the reform in 1986, Tamil Nadu in 1989, and Karnataka and Maharashtra in 1994. This robustness check is similar to the one conducted by Deininger et al. (2013).

³⁶ The non-reform states passed the reform in the year 2005, while the IHDS finished collecting the data just before the year 2005.

Additionally, I test the robustness of my results for a subsample of a different age group of children. IHDS defines children as individuals between 0 and 14 years, so my sample includes individuals in this entire age group.³⁷ Since I am not considering educational outcomes, I do not have to restrict the sample to school going children and I can use the entire sample. Even though the z-scores that I consider as outcomes, provide information of how a child compares to the reference population of the same age and gender, fifteen years is still a long period to consider. Thus, in Appendix Table C6, I reestimate equation (4.1) restricting my sample to only children under the age of 5 years (as is standard in most studies that look at HAZ outcomes) and my results are robust to this sample restriction. I also restrict my sample to children 6 to 14 years old and again, my results are robust to this sample restriction, as shown in Appendix Table C7.

Finally, my model posits that the link between mother's exposure to the HSA reform and her child's health is her bargaining power within the household. However, a mother's exposure to the reform likely determines other outcomes within the household and these outcomes (rather than her bargaining power) are mechanisms linking the mother's reform exposure to her child's health. Mother's education and her fertility are two such factors. Mother's education is not a problem as I only include women who have already completed their education; hence their level of education is pre-determined and will not be affected by the reform. The same is not true for her fertility; the reform could have an effect on reproductive decisions. Appendix Table C9 confirms that the HSA had impacts on fertility – the HSA reduced fertility by 0.17 children. Heath and Tan (2016) find that HSA decreased women's fertility by 0.52 children per woman. This result coincides with other research that finds that women in developing countries tend to prefer fewer children than men and can translate that increased bargaining power into lower fertility ((Klawon and Tiefenthaler 2001; Rasul 2008). One possible explanation is that when women have fewer children, they are able to invest more in each child. If this effect of the HSA (women's fertility) improved child health, it could still be a link between the HSA and child's health. To provide evidence for this, Appendix Table C10 looks at the relationship between the woman's fertility and her child's health.³⁸ I find that the number of children is negatively correlated with child's health, conditional on other controls, and therefore could possibly work in the direction of obtaining an effect of the

³⁷ According to The Census of India, children are defined as anyone below the age of 14.

³⁸ Ideally, I would estimate causal effects of a woman's fertility on child health in order to estimate the effects of exogenous changes in them due to the HSA, but because women have not completed their fertility, the data does not allow that.

HSA, which reduced fertility. I do not claim that these are the only other factors linking women's exposure to HSA with their child's health and while these results may only be an approximation due to data limitations, they are nonetheless a useful thought exercise.

4.7 Conclusions

In this paper I provide estimates of the impact of improving women's property inheritance rights, by using state-level amendments to the Hindu Succession Act in India. These amendments allowed unmarried women to have an equal share in property as their brothers, particularly with respect to ancestral and agricultural property. Five states in India passed these reforms prior to 2005, which is when there was a national amendment to the HSA. Using this quasi-experimental framework, I look at the intergenerational effects of the reform on health of children whose mothers were eligible for the reform. The legal amendments to women's inheritance rights in India offer an exogenous source of variation, which has been widely used to study the impact of the reform on women themselves. However, the direction of the effect is theoretically ambiguous and the empirical evidence shows mixed results, which provides a strong basis to investigate the persistence of these reform effects beyond the exposed generation of women into outcomes of their children as well.

Using the 2004-05 round of the Indian Human Development Survey (IHDS), I employ a difference-in-differences strategy where the treated group of individuals are children whose mothers belonged to the treatment states that introduced the reform before 2005 and were unmarried at the time of the reform in their state. I find that the height-for-age z-score is (HAZ) significantly higher for treated children. While it may initially seem from my results that households are gender-neutral, I do uncover a genderdifferential impact of the reform in households where the mother is less educated. For families where the mother has less than primary level of education, HAZ of treated daughters is statistically significantly lower than HAZ of treated sons by 0.213 standard deviations. This may be due to inherent culture of son preference and discrimination against daughters in Indian households (see for example, Bhalotra & Attfield, 1998; Biswas & Rose, 2010; Dancer et al., 2008; Gupta,1987; Hussain et al., 2000; Jayachandran & Pande, 2017; and Mishra et al., 2004). I find that this discriminatory effect is visible in larger families – mothers with less than primary education discriminate against treated daughters only in three children families, but not in two children families. This could be explained by resource constraints compelling some parents to favour sons over daughters as the number of children in the household increases (Jayachandran & Pande, 2017).

I argue that improvement in child health could be an indirect result of the reform raising mothers' bargaining power in household decisions. To show this I focus on mothers' decisions regarding their health-seeking behaviour around child birth and their freedom of mobility. I find that mothers exposed to the reform are more likely to receive antenatal and post-natal check-ups and to have their child delivered at a healthcare facility by a trained medical professional. I also find that the reform induced mothers to have more autonomy in decisions regarding their mobility when it comes to travelling unaccompanied. These findings provide a coherent picture of the reform improving women's "fall-back" position in terms of giving them more bargaining power in household decision-making. Increase in women's bargaining power, due to their ability to inherit, could eventually also improve the health of their children through better human capital and health investments in them. More bargaining power means that women can steer resources and decisions regarding human capital investments in children towards their own preferred direction. Previous literature found that women have stronger preferences for investments in children than men (e.g. Ward-Batts 2008) and therefore an increase in their bargaining power is likely to lead to an improvement in children's outcomes. Furthermore, the reform might also reduce the resource limitations which I found to be related to gender gaps in investments against daughters.

Taken together, these findings suggest that legal reforms at the state level are likely to have a positive impact despite the persistence of inherent social and cultural norms. Even in contexts where customs are very difficult to change, more gender egalitarian legislations such as the amendment to the HSA can have long term effects which can benefit both the exposed women and their daughters.

Chapter 5

Conclusions

This thesis sets out to explore the impact on intergenerational outcomes of negative inutero shocks, and analyses two coping responses to ameliorate their effects. In all chapters I examine the impact on child outcomes of in-utero and early-life conditions, but priority is given to answering in Chapter 2 the relatively understudied question of whether the impact of these conditions persists into outcomes of multiple generations. Chapters 3 and 4 then look at the ways to assuage the adverse intergenerational effects of shocks in-utero, primarily through material resources in terms of parental investments and policy reforms.

In Chapter 2, I build on the "fetal-origins" literature by studying whether adverse rainfall shocks in utero causally affect the third generation descendants in India. In a predominantly agricultural economy like India, fluctuations in rainfall out of the norm may affect crop yields and cause income losses; these are known to be major pathways through which weather shocks affect child outcomes. I present the first evidence that maternal grandmother's exposure to a negative rainfall shock during pregnancy has negative effects on health and cognitive ability outcomes of her grandchildren. I find that the number of months that the grandmother is exposed to the rainfall shock during her pregnancy is inversely proportional to the cognitive ability of her grandchildren – every additional month of maternal grandmother's exposure during her pregnancy reduces her grandchild's cognitive ability z-score by approximately 10 per cent of a standard deviation. I also find that every additional month of grandmother's exposure to the rainfall shock reduces the height-for-age z-scores (HAZ) by approximately 19.7 per cent of a standard deviation for her grandchildren below age five, but not for those 8-11 years old. This result is not surprising because the early-life literature finds that the effect of initial shocks tends to "fade out" by age eight or nine (also commonly known as the "missing middle"), but then resurface in adulthood (Almond and Currie, 2011a; Almond et al., 2018). This reappearance is a strong indication that the damage done to the health of the developing fetus remains largely irreversible and has far-reaching implications in terms of potentially hampering not only their own but also their children's quality of adult life.

While it is clear that in-utero shocks have long-lasting multi-generational effects, it is unclear whether the effects are driven by changes in direct biological (i.e. genetic and epigenetic) effects or whether they act mainly through changes in a child's home environment. One of the drawbacks of the work to date is that it is often impossible to distinguish between the exact channels that lead to these linkages. Animal experiments are able to provide biological evidence that the effects of in-utero shocks extend beyond the exposed generations, but in human studies such corresponding experiments are not possible. I take account that some of the transferrable effects of rainfall shocks are mediated through changes in child home environment by controlling for broad maternal adult characteristics. Results from my mediation analysis show that the third generation effect remains almost unaltered, suggesting that the key transmission mechanism of the third generation effect of the shocks may be the genetic and epigenetic inheritance.

A related concern arises whether the shocks in-utero matter more than shocks in the early postnatal period and my additional analysis from a comparison between the fetal and postnatal period confirms that only the effect of in-utero shocks passes down to the grandchild. This result in conjunction with evidence from human studies that epigenetic effect is the strongest in-utero and weak or even absent after birth and from animal experiments that effects of in-utero shocks on third and following generations are driven by epigenetics and not environment, strengthens my conclusion that the biological channels (plausibly the epigenetic inheritance) have a key role in the multigenerational transmission of the effects of in-utero rainfall shocks. This result certainly advocates that the phase in-utero is one of the most critical for development; this has key implications for the timing of public health interventions devised for mitigating damages - there is low-hanging fruit in terms of targeting interventions toward expectant mothers and women of child bearing age. Building on these results, I break the in-utero phase down further to learn if certain stages of pregnancy matter more. My trimester-specific third generation analysis shows that the effect for grandchild's health is mainly driven by their grandmother's exposure to the rainfall shocks in the first trimester.

My results so far show that even relatively mild shocks such as rainfall deficit, if experienced during the utero phase, can have lasting adverse effects on the developing fetus. While delayed interventions to alleviate the damage already done in-utero might be less effective, there is still a possibility to redress some of the repairable damage if it is targeted in-utero or early-life. Therefore, the idea implicit in the structure of the other two chapters is to focus on strategies that remedy the disadvantages caused by negative shocks in early-life. Chapter 3 answers the question of how parents' investment behaviour could help reduce a child's health disadvantage due to adverse rainfall shocks in-utero. I find that, in the circumstances when an extreme rainfall deficit negatively affects a child's health, parents compensate for some of the disadvantages of early life events by investing more in education of that child. A 1 standard deviation decrease in HAZ causes parents to increase education expenditure by approximately 52 per cent. Despite an overall compensatory behaviour, I also find that parental investment response differs between their sons and daughters; while they seem to compensate for a boy's health, no such strategy appears for changes in a girl's health. In the event of a rainfall shock, resource constrained parents, in my rural sample, follow an efficiency investment strategy and protect their son's health more than they protect their daughter's. There seems no definite answer to whether parents compensate or reinforce the effects of initial shocks, but it is apparent that, due to an expectation of higher utility and future returns from investment in a son, parents are likely to compensate their differences more in lowresource settings. This indicates that the behaviours observed at least partly respond to binding budget constraints and not just their preferences. These results hold important implications for policymakers – there is a need to target support to poorer families, whose discriminatory investment preferences are more likely to be influenced by resource constraints. Future research should then focus on better understanding how shocks and disadvantages interact, and the role of parents in responding to them.

Chapter 4 explores an inheritance rights reform as another targeted way to remediate the repairable damage caused by early-life shocks on child health. I find that the HAZ is significantly higher for treated children – in households where mothers are exposed to the Hindu Succession Amendment Act (HSAA) reform, children see a significant increase of 0.234 standard deviations in their health. Mother's level of education is also known to play an important role in reducing the bias against daughters and I see this pattern in my results. While I find no discrimination by more educated mothers, I find that HAZ of treated daughters is statistically significantly lower than HAZ of treated sons by 0.213 standard deviations for mothers with less than primary level of education.

I investigate women's bargaining power in household decisions as one of the mechanisms behind improvement in child health: women's legal ability to inherit ancestral property leads to an improvement in their bargaining power in intra-household decisions, associated with better child health outcomes. This presumed mechanism is consistent with my findings that uncover the pivotal role of the reform in increasing the likelihood of treated mothers receiving antenatal and post-natal check-ups and having their child delivered at a healthcare facility by a trained medical professional. I also find that the reform gave mothers more autonomy in decisions regarding their mobility, especially when it comes to travelling unaccompanied. More bargaining power allows women to steer resources and decisions regarding human capital investments in children towards their own preferred direction. We already know that women have stronger preferences for investments in children than men (e.g. Ward-Batts 2008), so an increase in their bargaining power is likely to lead to an improvement in child outcomes.

Related literature suggests that shocks and interventions can have differing intergenerational effects on boys and girls, but there is a dearth of evidence on whether the gender differences are biological or if they reflect differential parental responses caused by son preferences or other cultural beliefs. This thesis has provided detailed evidence on a) gender-specific heterogeneity in the effects of shocks, b) how mitigating strategies affect boys and girls differently, and c) the possible reasons for the gender differences given the cultural context. These findings can help formulate a case for targeted policies for improvement of human capital, while simultaneously spurring further questions for future research.

Clearly, there is still much to be learned about what matters and why. In this thesis, one limitation in studying child human capital is that some of the most commonly used measures such as height-for-age z-scores are at best only proxies for a whole range of subtle damages that a fetus may have suffered in-utero. More progress can be achieved if some of the measurement problems could be addressed. There is a need for more precise measurement of child health and for more information on the mechanisms through which a child is harmed and on interventions to mitigate this harm. Another area that could benefit from more specific measurement is related to the development of non-cognitive or "soft skills", which are now known to matter for outcomes such as education and employment.

This thesis focuses on the role of mothers and how the effects of shocks and interventions are transmitted along the maternal line. Due to the required design of birth cohorts across multiple generations, I have information about mothers, but not about fathers. There is a need for future analysis to start also to explore the neglected role of fathers.

Finally, identifying cost-effective ways to intervene in order to improve child outcomes is still an open question and has great room for future research, especially given the surmises on effectiveness of variegated policies. For example, evidence of little effect of income transfers on child outcomes caused a steer toward in-kind transfers instead. However, more recent evidence on the effectiveness of cash transfers should cause a reevaluation of the preferred policy, particularly considering the inefficiencies involved in offering in-kind transfers. Similarly, to date, most of the literature showed negative effects of maternal employment in the early years, but it should be thrown into question by recent evidence on changes in maternity leave policies affecting maternal employment without having any effect on long-term child outcomes. Continued evolvement in the understanding of human capital development in the early childhood is a stimulating frontier for intergenerational research in economics. Appendix A

Appendix to Chapter 2

A.1 Appendix tables

		controlling for m		
	(1)	(2)	(3)	(4)
VARIABLES	no mother's	controlling for	+ controlling	+ controlling
	$\operatorname{controls}$	mother's	for mother's	for mother's
		education	consumption	BMI z-score
		Danol	expenditure 1: HAZ (0-5 yea	
			1. 11AZ (0-5 yea	15)
In-utero rainfall shock:				
1^{st} trimester	-0.325^{**}	-0.320^{**}	-0.313^{**}	-0.317^{**}
2^{nd} trimester	(0.143) -0.200	(0.143) -0.222	(0.143) -0.219	(0.142) -0.214
2 trimester	(0.161)	(0.160)	(0.161)	(0.160)
$3^{\rm rd}$ trimester	-0.018	-0.001	0.004	0.016
	(0.218)	(0.218)	(0.217)	(0.216)
Child' sex (male)	0.092***	0.088**	0.083**	0.084**
O(1, 1) V $(1, 1)$	(0.035) - 0.030^{***}	(0.035) - 0.029^{***}	(0.035) - 0.030^{***}	(0.035) - 0.030^{***}
Child's age (in years)	(0.001)	(0.029 (0.001)	(0.001)	(0.001)
Mother's education	(0.001)	0.051***	0.042***	0.039***
		(0.005)	(0.005)	(0.005)
Mother's monthly			0.0002***	0.0002***
consumption expenditure			(4.39e-05)	(4.35e-05)
Mother's BMI z-score				0.131^{***}
Constant	-0.357***	-0.579***	-0.675***	(0.037) - 0.644^{***}
Constant	(0.047)	(0.050)	(0.055)	(0.055)
Observations	$12,\!696$	$12,\!696$	ì2,696	12,696
	Panel	B: Cognitive abi	litv z-score (8-1	1 vears)
In-utero rainfall shock:		0	0	0 /
$1^{\rm st}$ trimester	-0.075	-0.071	-0.061	-0.060
1 trimester	(0.071)	(0.068)	(0.068)	(0.068)
$2^{ m nd}$ trimester	-0.112	-0.124*	-0.117	-0.117
	(0.079)	(0.072)	(0.072)	(0.072)
$3^{\rm rd}$ trimester	-0.105	-0.080	-0.076	-0.067
	$(0.081) \\ -0.007$	(0.071) -0.006	(0.070) - 0.009	(0.070)
Child' sex $(male)$	(0.024)	(0.023)	(0.023)	-0.009 (0.023)
Child's age (in years)	-0.001	8.44e-05	-0.0002	-0.0001
Child's age (in years)	(0.001)	(0.001)	(0.001)	(0.001)
Mother's education		0.076***	0.067***	0.065***
		(0.003)	$(0.003) \\ 0.0002^{***}$	(0.003)
Mother's monthly			(2.36e-05)	0.0002^{***} (2.30e-05)
consumption expenditure			(2.30e-00)	0.052***
Mother's BMI z-score				(0.0123)
Constant	0.078	-0.249***	-0.317***	-0.304***
	(0.097)	(0.090)	(0.091)	(0.090)
Observations	9,278	`9,278 [´]	9,278	9,278

Table A1: Trimester-specific third generation effects of in-utero rainfall shocks on HAZ and cognitive ability outcomes, controlling for maternal characteristics

Notes: Standard errors in parentheses clustered at the district and household level *** p<0.01, ** p<0.05, * p<0.1. All columns include fixed effects for: mother's year of birth, month of birth, and district of residence.

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES	Education	Education	Monthly	Monthly	BMI z-score	BMI z-score
			Consumption	Consumption		
			Expenditure	Expenditure		
		Panel A: Mothers	of sample of children	0-5 years old		
In-utero shock	-0.561**	-0.100	-98.370***	-44.100*	-0.089**	-0.054
	(0.269)	(0.230)	(26.330)	(23.800)	(0.035)	(0.034)
Trimester-specific shocks:				· · · · ·		
$1^{\rm st} { m trim shock}$	-0.561	-0.098	-126.700***	-65.110*	-0.054	-0.010
	(0.458)	(0.388)	(37.340)	(35.860)	(0.055)	(0.054)
$2^{\rm nd}$ trim shock	-0.194	0.064	-63.180	-16.440	-0.072	-0.051
	(0.499)	(0.428)	(49.940)	(44.630)	(0.065)	(0.060)
$3^{\rm rd} { m trim shock}$	-Ò.948*´*	-0.274	-97.300**	-44.660	-0.154* ^{**}	-0.116**
	(0.412)	(0.414)	(38.850)	(41.010)	(0.044)	(0.046)
Fixed effects	No	Yes	No	Yes	No	Yes
Observations	9,122	$9,\!122$	$9,\!122$	$9,\!122$	9,122	9,122
		Panel B: Mothers of	of sample of children	8-11 years old		
In-utero shock	-0.270	-0.192	-112.6***	-61.170**	-0.132***	-0.087***
	(0.258)	(0.238)	(25.200)	(23.700)	(0.028)	(0.028)
Trimester-specific shocks:						
$1^{\rm st} { m trim shock}$	0.003	0.063	-126.3***	-62.490*	-0.125**	-0.066
	(0.431)	(0.401)	(32.480)	(33.390)	(0.051)	(0.053)
$2^{\rm nd}$ trim shock	-0.211	-0.238	-109.8**	-51.340	-Ò.109*´*	-0.068
	(0.440)	(0.437)	(47.060)	(43.510)	(0.044)	(0.046)
$3^{ m rd} { m trim shock}$	-0.625*	-0.424	-100.7* [*]	-69.660*	-0 .164***	-0.128***
	(0.342)	(0.340)	(40.480)	(40.210)	(0.038)	(0.038)
Fixed effects	No	Yes	No	Yes	No	Yes
Observations	$7,\!550$	$7,\!550$	$7,\!550$	$7,\!550$	7,550	$7,\!550$

Table A2: Second generation results for mothers who were exposed to the shock while in-utero

Notes: Robust standard errors clustered at the district level appear in parentheses. *** p < 0.01, ** p < 0.05, * p < 0.1. When specified, regressions include mother's year of birth, month of birth and state of residence fixed effect.

	ind 5 gender	
VARIABLES	$ \begin{array}{c} \hline (1) \\ \text{HAZ} (0-5) \end{array} $	(2) Cog. Ability (8-11)
In-utero shock	-0.186 (0.124)	-0.123^{*} (0.064)
In-utero shock*Male	-0.021 (0.189)	$\begin{array}{c} 0.054 \ (0.089) \end{array}$
Child' sex (male)	$0.093^{stst} \\ (0.035)$	-0.010 (0.024)
Child's age (in years)	-0.030^{***} (0.001)	-0.001 (0.001)
Constant	-0.358^{***} (0.047)	$0.079 \\ (0.097)$
Observations R-squared	$12,696 \\ 0.133$	$9,278 \\ 0.171$

Table A3: Third generation effects of exposure to negative rainfall shocks in-utero: heterogeneous effects by child's gender

Notes: Standard errors in parentheses clustered at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects include: mother's year of birth, month of birth, and district of residence.

VARIABLES	(1) HAZ 0-5	(2) HAZ 8-11	(3) Cog. Ability 8-11
In-utero shock	-0.300^{***} (0.112)	-0.011 (0.074)	-0.104^{**} (0.044)
In-utero shock * Post1979	0.397^{**} (0.201)	$0.264 \\ (0.514)$	$0.267 \\ (0.295)$
Post1979	-	-	-
Child' sex (male)'	0.092^{***} (0.035)	0.085^{***} (0.028)	-0.008 (0.024)
Child's age (in years)	-0.030**** (0.001)	-0.005^{***} (0.001)	-0.001 (0.001)
Constant	-0.358^{***} (0.047)	-1.010^{***} (0.143)	$0.080 \\ (0.097)$
District FEs	Yes	Yes	Yes
Mother's MOB & YOB FEs	Yes	Yes	Yes
R-squared Observations	$12,696 \\ 0.133$	$9,278 \\ 0.137$	$9,278 \\ 0.171$

Table A4: Factors mitigating the third generation effects of exposure to extreme rainfall shocks in-utero: effects by mother's year of birth pre and post 1979

Notes: Standard errors in parentheses clustered two-way at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects (FEs) include: mother's year of birth (YOB), month of birth (MOB), and district of residence. Controls also include a dummy variable for *Post1979*, which takes the value of 1 if time period t>1979 (i.e. for t 1980-1989) and takes a value of 0 if t<=1979 (i.e. for t 1955-1979). The variable *Post1979*, is omitted due to collinearity with mother's year of birth fixed effects, because all years from 1955 to 1990 are already controlled for, making the inclusion of the variable redundant.

	(1)	(2)	(3)
VARIABLES	HAZ (0-5)	HAZ (8-11)	Cog. Ability (8-11)
In-utero shock*Poor	-0.354^{*} (0.199)	-0.383^{***} (0.135)	$\begin{array}{c} 0.083 \ (0.082) \end{array}$
In-utero shock	-0.070 (0.125)	$0.125 \ (0.081)$	-0.115^{**} (0.056)
Dummy for poor (yes=1)	-0.254^{***} (0.048)	-0.240^{***} (0.044)	-0.477^{***} (0.034)
Child's sex (male)	0.088^{**} (0.035)	0.076^{***} (0.028)	-0.021 (0.023)
Child's age (in years)	-0.030^{***} (0.001)	-0.005^{***} (0.001)	-0.001 (0.001)
Constant	-0.270^{***} (0.051)	-0.920^{***} (0.143)	$\begin{array}{c} 0.256^{***} \ (0.094) \end{array}$
Observations R-squared	$12,696 \\ 0.135$	$9,278 \\ 0.143$	$9,278 \\ 0.207$

 Table A5: Factors mitigating the third generation effects of exposure to extreme rainfall

 shocks in-utero: effects for those living below the poverty line

Notes: Standard errors in parentheses clustered at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for: mother's year of birth, month of birth, and district of residence.

	(1)	(2)	(3)
VARIABLES	HAZ $(0-5)$	HAZ (8-11)	Cog. Ability (8-11)
In-utero shock $*$ No. of children	-0.025 (0.055)	-0.060 (0.046)	-0.016 (0.027)
In-utero shock	-0.104 (0.214)	$0.256 \\ (0.177)$	-0.008 (0.119)
No. of children	-0.039** (0.018)	-0.093^{***} (0.014)	-0.144^{***} (0.010)
Child's sex (male)	0.089^{**} (0.035)	0.059^{**} (0.029)	-0.049^{**} (0.025)
Child's age (in years	-0.030^{***} (0.001)	-0.006*** (0.002)	-0.001 (0.001)
Constant	-0.248^{***} (0.074)	-0.630^{***} (0.153)	0.668^{***} (0.106)
Observations	12,686	9,273	9,273
R-squared	0.133	0.144	0.203

Table A6: Factors mitigating the third generation effects of exposure to negative rainfall shocks in-utero: effects by number of children

Notes: Standard errors in parentheses clustered at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects for: mother's year of birth, month of birth, and district of residence.

	Panel A: Rural sample		Panel I	B: Urban sample
	(1)	(2)	(3)	(4)
VARIABLES	$\begin{array}{c} \text{HAZ} \\ (0\text{-}5) \end{array}$	Cog. Ability (8-11)	HAZ (0-5)	Cog. Ability (8-11)
In-utero shock	-0.281^{***} (0.107)	-0.106^{**} (0.046)	$0.132 \\ (0.219)$	-0.052 (0.100)
Child' sex (male)	0.091^{**} (0.041)	$0.042 \\ (0.028)$	$0.086 \\ (0.071)$	-0.124^{***} (0.037)
Child's age (in years)	-0.030*** (0.002)	$\begin{array}{c} 0.0003 \\ (0.001) \end{array}$	-0.029*** (0.002)	-0.001 (0.001)
Constant	-0.465^{***} (0.057)	-0.153 (0.119)	-0.106 (0.079)	$0.491^{***} \\ (0.161)$
Fixed effects Observations	Yes 9,068	Yes 6,571	Yes 3,628	Yes 2,707

Table A7: Mechanisms driving the third generation effects of exposure to extreme rainfall shocks in-utero: effects by area of residence

Notes: Standard errors in parentheses clustered at the district and household level *** p < 0.01, ** p < 0.05, * p < 0.1. Fixed effects for: mother's year of birth, month of birth, and district of residence.

SHOCKS III-	utero. effects by fail	d owners in rufal are	eas
VARIABLES	$\begin{array}{c} \hline (1) \\ HAZ \\ (0-5 \text{ yrs}) \end{array}$	(2) HAZ (8-11 yrs)	(3)Cog. Ability (8-11 yrs)
In-utero shock	-0.267 (0.185)	-0.064 (0.128)	0.019 (0.075)
In-utero shock*own land	-0.013 (0.218)	0.059 (0.162)	-0.188** (0.089)
Own land (yes=1)	0.102^{*} (0.054)	0.171^{***} (0.050)	0.277^{***} (0.033)
Male	0.091^{**} (0.041)	$\begin{array}{c} 0.107^{***} \\ (0.033) \end{array}$	0.047^{st} (0.027)
Child's age (in years)	-0.030^{***} (0.001)	-0.006^{***} (0.001)	$9.12e-05 \ (0.001)$
Constant	-0.532^{***} (0.068)	-1.180^{***} (0.156)	-0.311^{***} (0.119)
Observations	9,068	$6,\!571$	$6,\!571$
R-squared	0.131	0.151	0.193

Table A8: Mechanisms driving the third generation effects of exposure to extreme rainfall shocks in-utero: effects by land owners in rural areas

Notes: Standard errors in parentheses clustered two-way at the district and household level *** p<0.01, ** p<0.05, * p<0.1. Fixed effects (FEs) for: mother's year of birth (YOB), month of birth (MOB), and district of residence. Controls also include a dummy variable for **Own land**, which takes the value of 1 if the household owns any land and 0 otherwise. The variable *In utero shock * own land*, is an interaction term between the variable *in utero shock* and the dummy for *own land*.

VARIABLES	(1)	(2)				
Panel A: Dependent variable is HAZ for children 0-5 year old						
Maternal grandmother shock in 12 months before pregnancy	$0.017 \\ (0.095)$	$\begin{array}{c} 0.004 \ (0.094) \end{array}$				
Maternal grandmother shock in-utero		-0.197^{**} (0.095)				
Child' sex (male)	$\begin{array}{c} 0.091^{***} \ (0.035) \end{array}$	0.092^{***} (0.035)				
Child's age (in years)	-0.030^{***} (0.001)	-0.030^{***} (0.001)				
Constant	-0.377^{***} (0.047)	-0.358^{***} (0.047)				
Fixed effects	Yes	Yes				
R-squared	0.132	0.133				
Observations	$12,\!696$	$12,\!696$				
VARIABLES	(1)	(5)				
Panel B: Dependent variable is Cognitive ability	z-score for childre	en 8-11 year old				
Maternal grandmother shock in 12 months before pregnancy	$0.055 \\ (0.042)$	$\begin{array}{c} 0.051 \ (0.042) \end{array}$				
Maternal grandmother shock in-utero		-0.094^{**} (0.045)				
Child' sex (male)	-0.007 (0.024)	-0.007 (0.024)				
Child's age (in years)	-0.001 (0.001)	-0.001 (0.001)				
Constant	$egin{array}{c} 0.071 \ (0.097) \end{array}$	$\begin{array}{c} 0.077 \ (0.097) \end{array}$				
Fixed effects	Yes	Yes				
R-squared	0.171	0.171				
Observations	9,278	9,278				

Table A9: Third generation effects of exposure to negative rainfall shocks
experienced in pre-pregnancy and in-utero phase

Notes: Standard errors in parentheses clustered at the district and household level *** p < 0.01, ** p < 0.05, * p < 0.1. Fixed effects include: mother's year of birth, month of birth, and district of residence.

Appendix B

Appendix to Chapter 3

B.1 Appendix tables

	(1)	(2)	(3)	(4)	(5)	
	OLS			2SLS		
VARIABLES				1 st stage HFA	$2^{ m nd}$ stage	
HFA (at 5-19 months)	$\begin{array}{c} 0.202^{***} \\ (0.038) \end{array}$	$\begin{array}{c} 0.105^{***} \ (0.036) \end{array}$	$\begin{array}{c} 0.058 \ (0.038) \end{array}$		-0.517^{**} (0.173)	
Rainfall deficit * dummy variable for child was 5-12 months old in 2002				-2.510^{***} (0.417)		
Rainfall deficit * dummy variable for child was 13-19 months old in 2002				-1.543^{***} (0.315)		
Male (yes=1)	$\begin{array}{c} 0.332^{***} \ (0.108) \end{array}$	0.260^{**} (0.101)	0.248^{**} (0.100)	-0.291^{***} (0.083)	$\begin{array}{c} 0.067 \ (0.125) \end{array}$	
Child's age (months)	$\begin{array}{c} 0.091 \ (0.126) \end{array}$	$\begin{array}{c} 0.122 \ (0.117) \end{array}$	$\begin{array}{c} 0.130 \ (0.116) \end{array}$	-0.008 (0.102)	$\begin{array}{c} 0.099 \\ (0.130) \end{array}$	
Child's age squared	$\begin{array}{c} 0.001 \ (0.005) \end{array}$	-0.001 (0.005)	-0.002 (0.005)	-0.003 (0.004)	-0.002 (0.005)	
Dummy for child's age $5 - 12$ months				-0.825 (0.523)	$\begin{array}{c} 0.101 \ (0.218) \end{array}$	
Dummy for eldest child	0.531*** (0.108)	$\begin{array}{c} 0.356^{***} \\ (0.113) \end{array}$	$\begin{array}{c} 0.083 \\ (0.136) \end{array}$	-0.163 (0.109)	-0.051 (0.154)	
Dummy for premature birth	$\begin{array}{c} 0.357^{*} \ (0.210) \end{array}$	0.226 (0.196)	0.251 (0.186)	-0.349** (0.148)	0.108 (0.204)	
Mother's years of completed education		$\begin{array}{c} 0.159^{***} \\ (0.014) \end{array}$	0.122^{***} (0.015)	$\begin{array}{c} 0.020 \\ (0.0121) \end{array}$	$\begin{array}{c} 0.137^{***} \\ (0.017) \end{array}$	
Mother's age (years)		-0.003 (0.062)	-0.005 (0.062)	$0.056 \\ (0.081) \\ 0.0005$	0.027 (0.078)	
Mother's age squared		$\begin{array}{c} 0.0001 \ (0.001) \ 0.035^{***} \end{array}$	$\begin{array}{c} 0.0002 \ (0.001) \ 0.033^{***} \end{array}$	-0.0005 (0.001) 0.031^{***}	-0.0001 (0.001) 0.051***	
Mother's height No. of males 0-5 years		(0.008)	(0.033) (0.008) 0.051	(0.031) (0.008) 0.032	(0.051) (0.010) 0.066	
No. of males 6-12 years			(0.106) -0.182**	(0.082) (0.085) -0.251^{***}	(0.117) - 0.332^{**}	
No. of males 13-17 years			(0.087) -0.365**	(0.077) -0.275**	(0.105) -0.483***	
No. of males 18-60 years			(0.169) -0.028	(0.118) 0.193^{***}	(0.182) 0.070	
No. of males 61+ years			(0.062) 0.084	(0.047) 0.114	(0.069) 0.138	
No. of females 0-5 years			(0.121) -0.043 (0.096)	(0.107) -0.080 (0.082)	(0.139) -0.088 (0.104)	

Table B1: Parental response of education expenditure to child's height-for-age (HFA) Dependent variable: Log (monthly expenditure in education) in year 2007 when the child is 4-6 years old

No. of females 6-12 years			-0.274^{***} (0.073)	-0.203^{***} (0.058)	-0.398^{***} (0.088)
No. of females 13-17 years			-0.057 (0.132)	-0.264^{***} (0.098)	-0.193 (0.143)
No. of females 18-60 years			0.168^{**} (0.069)	-0.127^{**} (0.050)	$\begin{array}{c} 0.118 \ (0.075) \end{array}$
No. of females 61+ years			$\begin{array}{c} 0.202 \ (0.136) \end{array}$	-0.168 (0.109)	$\begin{array}{c} 0.072 \ (0.150) \end{array}$
Wealth Index in year 2002			2.044^{***} (0.353)	1.384^{***} (0.271)	2.828^{***} (0.442)
Constant	$\begin{array}{c} 0.747 \ (0.742) \end{array}$	-5.055^{***} (1.565)	-5.290^{***} (1.560)	-8.117^{***} (1.592)	-8.939^{***} (2.002)
First stage F-statistic Endogeneity test p-value					$29.030 \\ 0.0001$
Sargan-Hansen test p-value					0.580
Child controls Mother controls	Yes No	Yes Yes	Yes Yes	Yes Yes	Yes Yes
Household controls	No	No	Yes	Yes	Yes
Observations R-squared	$1,116 \\ 0.091$	$\begin{array}{c}1,\!116\\0.210\end{array}$	$1,116 \\ 0.255$	1,116	$1,116 \\ 0.075$
Adjusted R-squared	0.086	0.203	0.241	-	0.056

Notes: Robust standard errors appear in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The sample includes children enrolled in school and living in rural households only. Source: Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

	(1)	(2)	(3)	(4)	(5)	(6)
VARIABLES				$^{ m 1st}$ stage HFA males	1 st stage HFA females	$2^{ m nd}~{ m stage}$
HFA z-score * Male (at 5-19 months old)	$0.156^{***} \\ (0.048)$	$\begin{array}{c} 0.045 \ (0.045) \end{array}$	-0.004 (0.047)			-0.605^{***} (0.196)
HFA z-score * Female (at 5-19 months old)	$0.265^{***} \\ (0.061)$	$\begin{array}{c} 0.198^{***} \\ (0.057) \end{array}$	0.154^{***} (0.057)			-0.372 (0.256)
Rainfall deficit * dummy variable for child was 5-12 months old in 2002 * Male				-2.733^{***} (0.501)	-0.152 (0.182)	
Rainfall deficit * dummy variable for child was 5-12 months old in 2002 * Female				-0.166 (0.274)	-1.879^{***} (0.357)	
Rainfall deficit * dummy variable for child was 13-19 months old in 2002 * Male				-1.695^{***} (0.378)	-0.188 (0.163)	
Rainfall deficit * dummy variable for child was 13-19 months old in 2002 * Female				$\begin{array}{c} 0.292 \\ (0.191) \end{array}$	-1.442^{***} (0.359)	
Male (yes=1)	$0.199 \\ (0.155)$	$0.064 \\ (0.142)$	$0.042 \\ (0.142)$	-3.770^{***} (0.383)	2.680^{***} 0.359)	-0.233 (0.388)
Child's age (months)	$0.081 \\ (0.126)$	$0.121 \\ (0.117)$	$0.134 \\ (0.119)$	$\begin{array}{c} 0.001 \\ (0.088) \end{array}$	-0.002 (0.056)	$0.098 \\ (0.130)$
Child's age squared	(0.001) (0.005)	-0.001 (0.005)	-0.002 (0.005)	-0.002 (0.003)	-0.001 (0.003)	-0.002 (0.005)
Dummy for child's age $5 - 12$ months			$\begin{array}{c} 0.036 \ (0.198) \end{array}$	-0.713^{*} (0.431)	-0.105 (0.313)	$0.097 \\ (0.217)$
Dummy for eldest child	0.535^{***} (0.108)	$\begin{array}{c} 0.357^{***} \\ (0.113) \end{array}$	0.080 (0.135)	-0.088 (0.083)	-0.067 (0.074)	-0.056 (0.152)
Dummy for premature birth	()	0.232 (0.194)	0.257 (0.184)	-0.242^{*} (0.124)	-0.124 (0.097)	0.116 (0.202)
Mother's years of completed education		0.160^{***} (0.014)	0.123^{***} (0.015)	0.020^{**} (0.010)	-0.0002 (0.008)	0.140^{***} (0.017)
Mother's age (years)		-0.003 (0.062)	-0.002 (0.062)	0.052 (0.080)	0.004 (0.027)	0.031 (0.082)
Mother's age squared		0.0001 (0.001)	0.0002 (0.001)	-0.0005 (0.001)	8.55e-05 (0.0004)	-0.0002 (0.001)
Mother's height		0.036*** (0.008)	0.033*** (0.008)	0.020^{***} (0.007)	0.012*** (0.004)	0.051^{***} (0.010)
No. of males 0-5 years		()	0.042 (0.106)	-0.016 (0.063)	0.058 (0.063)	0.053 (0.117)

Table B2: Heterogeneous effects by child's gender Dependent variable: Log (monthly expenditure in education) in year 2007 when the child is 4-6 years old

No of the C10 or a			0 100**	0 177***	0.000	0.941***
No. of males 6-12 years			-0.189^{**} (0.087)	-0.177^{***} (0.063)	-0.069 (0.056)	-0.341^{***} (0.103)
No. of males 13-17 years			-0.361**	-0.163*	-0.124*	-0.473***
, i i i i i i i i i i i i i i i i i i i			(0.167)	(0.097)	(0.073)	(0.180)
No. of males 18-60 years			-0.022 (0.062)	0.153^{***}	$0.038 \\ (0.031)$	0.078
No. of males $61 + years$			(0.002) 0.092	$(0.039) \\ 0.126$	(0.031) -0.009	$(0.068) \\ 0.151$
No. of males $01 + years$			(0.120)	(0.094)	(0.061)	(0.131)
No. of females $0-5$ years			-0.039	-0.041	-0.041	-0.085
			(0.097)	(0.062)	(0.061)	(0.103)
No. of females 6-12 years			-0.284^{***} (0.074)	-0.175^{***} (0.047)	-0.025 (0.039)	-0.411^{***} (0.091)
No. of females 13-17 years			-0.033	-0.014	-0.254***	-0.156
			(0.131)	(0.085)	(0.057)	(0.154)
No. of females 18-60 years			0.166**	-0.092^{**}	-0.035	0.116
			(0.069)	(0.040)	(0.036)	(0.074)
No. of females 61+ years			$0.194 \\ (0.136)$	-0.169^{**} (0.0855)	-0.003 (0.073)	$0.061 \\ (0.149)$
Wealth Index in year 2002			2.028***	0.835***	0.570***	2.802***
Wearth mack in year 2002			(0.353)	(0.214)	(0.173)	(0.447)
Constant	0.904	-4.988***	-5.298***	-3.208**	-4.554***	-8.811***
	(0.740)	(1.570)	(1.623)	(1.334)	(0.881)	(2.042)
F-statistic 1st stage: HFA (ma	ale)					15.21
F-statistic 1st stage: HFA (fer	male)					22.54
Endogeneity test p-value						0.0003
Sargan-Hansen test p-value						0.5636
P-value for t-test						0.4170
HFA(male)=HFA(female)						0.1110
Child controls	yes	yes	yes	yes	yes	yes
Mother controls	no	yes	yes	yes	yes	yes
Household controls	no	no	yes	yes	yes	yes
Observations	1,116	1,116	1,116	1,116	1,116	1,116
R-squared	0.090	0.214	0.259	-	-	0.080
Adjusted R-squared	0.0852	0.206	0.243	-	-	0.0608

Notes: Robust standard errors appear in parentheses. *** p < 0.01, ** p < 0.05, * p < 0.1. The sample includes children enrolled in formal or informal school and living in rural households only.

Source: Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

	(1)	(2)	(3)	(4)	(5)
	OLS			2SLS	
VARIABLES				1 st stage HFA	$2^{ m nd}$ stage
HFA z-score (at 5-19 months)	0.256^{***} (0.037)	$\begin{array}{c} 0.109^{***} \\ (0.034) \end{array}$	$\begin{array}{c} 0.041 \\ (0.034) \end{array}$		-0.849*** (0.229)
Rainfall deficit * dummy variable for child 5-12 months old in 2002				-1.547^{***} (0.304)	
Rainfall deficit * dummy variable for child 13-19 months old in 2002				-1.102^{***} (0.249)	
Male (yes=1)	$\begin{array}{c} 0.293^{***} \\ (0.102) \end{array}$	0.179^{*} (0.091)	0.159^{**} (0.085)	-0.232^{***} (0.068)	-0.058 (0.10)
Constant	2.259^{***} (0.696)	1. 10	-4.111^{***} (1.345)	-9.574^{***} (1.348)	-11.488^{***} (2.503)
Child controls	Yes	Yes	Yes	Yes	Yes
Mother controls	No	Yes	Yes	Yes	Yes
Household controls	No	No	Yes	Yes	Yes
First stage F-statistic					22.416
Endogeneity test p-value					0.0000
Sargan-Hansen test p-value	<u>è</u>				0.0942
Observations	$1,\!534$	$1,\!534$	1,534	$1,\!534$	$1,\!534$

Table B3: Parental response of education expenditure to child's height-for-age (HFA) – Rural and Urban Sample

Dependent variable: Log (monthly expenditure in education) in year 2007 when the child is 4-6 years old

Notes: Robust standard errors appear in parentheses. *** p < 0.01, ** p < 0.05, * p < 0.1. The sample includes children enrolled in school and living in rural and urban households. Child controls include: child's age in months; child's age squared; dummy variable for whether the child was 5-12 months old in year at the time of shock in year 2002 (for columns 6 and 7); and dummy variable for the eldest child. Mother's controls include: mother's years of completed education; mother's age in years; mother's age squared; and mother's height in cm. Household controls include: Separate variables for the number of males present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; separate variables for the number of females present in the household in year 2002. *Source:* Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

(4)	(5)
	2SLS
1 st stage HFA	$2^{\rm nd}$ stage
	-0.849^{***} (0.229)
-2.649^{***} (0.420)	
-1.315^{***} (0.315)	
-0.351^{***} (0.086)	$\begin{array}{c} 0.0047 \\ (0.036) \end{array}$
-8.796^{***} (1.618)	1.0090^{*} (0.581)
Yes	Yes
Yes	Yes
Yes	Yes
	27.548
	0.0013
	0.4791
1,031	1,031
	1st stage HFA 2:649*** (0.420) 1:315*** (0.315) 0:351*** (0.086) -8.796*** (1.618) Yes Yes Yes Yes Yes Yes Yes Yes

Table B4: Parental response of education investment to child's height-for-age	(HFA)
Dependent variable: Is the child 4-6 years old currently enrolled in preschool $(0/1)$	

Notes: Robust standard errors appear in parentheses. *** p<0.01, ** p<0.05, * p<0.1. The sample includes children living in rural households only. Child controls include: child's age in months; child's age squared; dummy variable for whether the child was 5-12 months old in year at the time of shock in year 2002 (for columns 6 and 7); and dummy variable for the eldest child. Mother's controls include: mother's years of completed education; mother's age in years; mother's age squared; and mother's height in cm. Household controls include: Separate variables for the number of males present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; separate variables for the number of females present in the household in age groups 6-12 years, 13-17 years, 18-60 years and 61+ years, respectively; wealth index of the household in year 2002.

Source: Author's calculations based on data from Young Lives Survey rounds 1 (year 2002) and 2 (year 2007) for Andhra Pradesh, India.

Appendix C

Appendix to Chapter 4

C.1 Appendix tables

Table C1: Descriptive Statistics: Mean Household Characteristics By HSAA Reform

	Control group	Treatment group	Difference
Panel A: Full sample			
Mother's education (years)	$3.776 \\ (0.037)$	$4.179 \\ (0.103)$	-0.404^{***} (0.113)
Mother's age (years)	$31.075 \ (0.048)$	$27.418 \\ (0.091)$	3.657^{***} (0.143)
Mother's age at marriage (years)	$17.155 \\ (0.025)$	$18.583 \\ (0.080)$	-1.428^{***} (0.077)
No. of children	$3.129 \\ (0.012)$	$2.492 \\ (0.027)$	$\begin{array}{c} 0.637^{***} \ (0.036) \end{array}$
Hindu $(0/1)$	$0.952 \\ (0.002)$	$0.984 \\ (0.003)$	-0.032^{***} (0.005)
Rural $(0/1)$	$0.734 \\ (0.004)$	$\begin{array}{c} 0.655 \ (0.011) \end{array}$	0.080^{***} (0.011)
Own any Land $(0/1)$	$\begin{array}{c} 0.517 \ (0.004) \end{array}$	$\begin{array}{c} 0.370 \ (0.011) \end{array}$	$\begin{array}{c} 0.147^{***} \\ (0.012) \end{array}$
Low caste (SC/ST) $(0/1)$	$\begin{array}{c} 0.411 \\ (0.004) \end{array}$	$\begin{array}{c} 0.318 \ (0.011) \end{array}$	0.093^{***} (0.012)
Below poverty line $(0/1)$	$\begin{array}{c} 0.308 \ (0.004) \end{array}$	$\begin{array}{c} 0.231 \ (0.010) \end{array}$	0.078^{***} (0.011)
Observations	$14,\!963$	1,783	
Panel B: Reform States			
Mother's education (years)	$4.009 \\ (0.091)$	$\begin{array}{c} 4.179 \\ (0.103) \end{array}$	-0.170 (0.138)
Mother's age (years)	$32.992 \\ (0.993)$	$27.418 \\ (0.091)$	5.574^{***} (0.138)
Mother's age at marriage (years)	$17.033 \\ (0.062)$	$18.583 \\ (0.080)$	-1.550^{***} (0.100)
No. of children	$2.846 \ (0.028)$	$2.459 \\ (0.027)$	$\begin{array}{c} 0.353^{***} \\ (0.040) \end{array}$
Hindu $(0/1)$	$0.957 \\ (0.004)$	$0.984 \\ (0.003)$	-0.027^{***} (0.005)
Rural $(0/1)$	$0.721 \\ (0.010)$	$0.655 \\ (0.011)$	$\begin{array}{c} 0.067^{***} \ (0.015) \end{array}$
Own any Land $(0/1)$	$\begin{array}{c} 0.500 \ (0.011) \end{array}$	$\begin{array}{c} 0.370 \ (0.011) \end{array}$	$\begin{array}{c} 0.131^{***} \\ (0.016) \end{array}$
Low caste (SC/ST) $(0/1)$	$\begin{array}{c} 0.346 \ (0.010) \end{array}$	$\begin{array}{c} 0.318 \ (0.011) \end{array}$	$\begin{array}{c} 0.028^{*} \\ (0.015) \end{array}$
Below poverty line $(0/1)$	$0.261 \\ (0.009)$	$\begin{array}{c} 0.231 \ (0.010) \end{array}$	0.030^{**} (0.014)
Observations	2,226	1,783	

Notes: Entries present sample means with standard deviations reported in parentheses. Source: Author's analysis based on IHDS. Control group includes all those who were not exposed to the reform, while those in treatment group were exposed to the reform. Col. (1) and (2) in Panel (A) show control and treatment groups in the total sample of 16,746 children in reform and non-reform states. Col. (1) and (2) in Panel (B) show control and treatment groups only within the reform states sample of 4,009 children.

	(1)	(2)
	HAZ	HAZ
Reform State*Mother married post-HSAA	0.227^{***} (0.073)	
Reform State*Mother married pre-1–6 years	· · · ·	-0.015
Reform State*Mother married post-0–5 years		(0.058) 0.154^{**}
Reform State*Mother married post-6+ years		(0.072) 0.262^{**} (0.120)
Child's age (in months)	-0.037***	-0.037***
Child's age squared	(0.002) 0.031^{***}	(0.002) 0.031^{***}
Child's gender (girl=1)	(0.002) -0.100***	(0.002) -0.100***
Mother's education	(0.025) 0.041^{***}	(0.025) 0.041^{***}
Mother's age	(0.004) -0.015	(0.004) -0.015
Mother's age at marriage	$(0.035) \\ 0.023$	$(0.036) \\ 0.022$
Hindu	(0.038) -0.152	(0.039) -0.151
Rural	(0.094) -0.175***	(0.094) -0.174***
Own any land	(0.054) 0.059^{*}	(0.054) 0.059 (0.025)
Low caste	(0.035) - 0.038 (0.035)	(0.035) -0.039 (0.036)
Below poverty line status	(0.033) -0.131*** (0.047)	(0.030) -0.130^{***} (0.047)
Constant	(0.047) 0.599 (1.086)	(0.047) 0.589 (1.112)
R-squared	0.085	0.084
Observations	16,746	16,746

Table C2: Parallel Trends Assumption: Intergenerational Effect of the Hindu Succession Act Amendment (HSAA) on Children's Height-for-Age Z-Scores (HAZ)

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include all controls and district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. This table considers the *sample of children* 0-14 years old with mothers who had already completed their education by the year of reform in their state. *Mother married post-HSAA* is an indicator variable for whether the mother of the individual got married after the reform. *Mother married pre-1*–6 years is an indicator variable for whether the child's mother had married in the six years leading up to the reform. *Mother married post-0–5 years* is an indicator variable for whether the reform and *Mother married post-6+ years* is an indicator variable for whether the mother got married in the sixth year after the reform and beyond.
	(1)	(2)
VARIABLES	HAZ	HAZ
Ref state*Mother was unmarried by the year of reform in 1983	-0.006 (0.059)	
Muslim*Ref state*Mother was unmarried by the reform year		$\begin{array}{c} 0.180 \\ (0.256) \end{array}$
Child's age (in months)	-0.038^{***} (0.002)	-0.031^{***} (0.005)
Child's age squared	$\begin{array}{c} 0.032^{***} \ (0.002) \end{array}$	$\begin{array}{c} 0.026^{***} \ (0.005) \end{array}$
Child's gender (girl=1)	-0.095^{***} (0.026)	$\begin{array}{c} 0.080 \ (0.060) \end{array}$
Mother's education	$\begin{array}{c} 0.043^{***} \\ (0.005) \end{array}$	0.037^{**} (0.015)
Mother's age	$0.009 \\ (0.037)$	$0.160^{st} (0.091)$
Mother's age at marriage	$0.002 \\ (0.040)$	-0.115 (0.078)
Hindu	-0.147 (0.102)	N/A
Rural	-0.190^{***} (0.057)	-0.324^{**} (0.160)
Own any land	0.086^{**} (0.034)	-0.080 (0.110)
Low caste	-0.031 (0.037)	-0.390 (0.258)
Below poverty line	-0.127^{**} (0.051)	-0.251^{**} (0.089)
Constant	-0.375 (1.170)	-4.538 (3.108)
Fixed effects	Yes	Yes
R-squared	0.078	0.098
Observations	$14,\!675$	2,713

Table C3: Falsification Test: Effect of False Reforms on Height-for-Age Z-Scores (HAZ) of Children across Reform and Non-Reform States

Notes: ***, ** denote significance at 1%, 5% and 10% respectively. All regressions include district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. The sample consists of male and female children, 0-14 years old with mothers who had already completed their education by the year of reform in their state. The sample is restricted to Hindus, Sikhs, Jains and Buddhists. Sample of states does not include Jammu & Kashmir, Kerala and the North Eastern states. Column 1 reports pre-treatment results for a "false" reform date of January 1983. Column 2 reports results for children of Non-Hindu i.e. Muslim women.

	Any land owned		
	(1)	(2)	(3)
	1986	1989	1994
Reform State [*] Mother was unmarried by the reform year	$1.325 \\ (2.096)$	$0.767 \\ (2.207)$	$1.406 \\ (2.131)$
Child's age (in months)	-0.044^{***} (0.003)	-0.044^{***} (0.003)	-0.042^{***} (0.003)
Child's age squared	0.039^{***} (0.003)	$\begin{array}{c} 0.039^{***} \\ (0.003) \end{array}$	$\begin{array}{c} 0.037^{***} \ (0.003) \end{array}$
Child's gender (girl=1)	-0.126^{***} (0.042)	-0.121^{***} (0.043)	-0.113^{***} (0.041)
Mother's education	0.049^{***} (0.006)	$\begin{array}{c} 0.049^{***} \\ (0.006) \end{array}$	$\begin{array}{c} 0.052^{***} \ (0.007) \end{array}$
Mother's age	-0.006 (0.074)	-0.022 (0.077)	-0.004 (0.074)
Mother's age at marriage	$\begin{array}{c} 0.005 \ (0.073) \end{array}$	$\begin{array}{c} 0.021 \ (0.075) \end{array}$	$\begin{array}{c} 0.0001 \ (0.073) \end{array}$
Hindu	-0.376^{*} (0.216)	-0.444^{**} (0.186)	-0.474^{**} (0.188)
Rural	-0.278^{*} (0.141)	-0.353^{**} (0.154)	-0.296^{*} (0.162)
Low caste	$\begin{array}{c} 0.004 \ (0.057) \end{array}$	$\begin{array}{c} 0.010 \ (0.054) \end{array}$	-0.015 (0.058)
Below poverty line	-0.112 (0.068)	-0.068 (0.069)	-0.035 (0.075)
Constant	$egin{array}{c} 0.615 \ (2.238) \end{array}$	$1.291 \\ (2.388)$	$\begin{array}{c} 0.409 \ (2.252) \end{array}$
Fixed effects	Yes	Yes	Yes
Observations	6,336	$6,\!294$	6,285
R-squared	0.096	0.097	0.094

Table C4: Falsification Test: Effect of False Reforms in control States on Height-For-Age Z-Scores (HAZ) amongst Children of Hindu Women in Non-Reform States

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. The sample consists of male and female children, 0-14 years old with mothers who had already completed their education by the year of reform in their state. 1986, 1989 and 1994 are years when the reform was passed in the reform states, but in this table they represent placebo years in the non-reform states. The three columns report results for any landowning Hindus, Sikhs, Jains and Buddhists in non-reform states. Sample of states does not include Jammu & Kashmir, Kerala and the North Eastern states.

	(1)	(2)	(3)
	HAZ	HAZ	HAZ
Reform State*Mother married post-HSAA	0.267^{***} (0.046)	0.251^{***} (0.071)	0.228^{***} (0.072)
F	(0.0 - 0)	(0.0.2)	(1111)
Child's age (in months)		-0.038^{***} (0.002)	-0.037^{***} (0.002)
Child's age squared		$\begin{array}{c} 0.032^{***} \\ (0.002) \end{array}$	0.031^{***} (0.002)
Child's gender (girl=1)		-0.106^{***} (0.026)	-0.100^{***} (0.025)
Mother's education		0.040^{***} (0.004)	0.041^{***} (0.004)
Mother's age		0.016^{***} (0.003)	-0.015 (0.036)
Mother's age at marriage		-0.006 (0.009)	$0.023 \\ (0.038)$
Hindu (yes=1)		-0.188** (0.086)	-0.152 (0.093)
Rural (yes=1)		-0.187^{***} (0.051)	-0.175^{***} (0.054)
Own land (yes=1)		$\begin{array}{c} 0.054 \ (0.034) \end{array}$	$\begin{array}{c} 0.059^{*} \ (0.035) \end{array}$
Low caste (yes=1)		-0.040 (0.036)	-0.039 (0.035)
Below poverty line status $(yes=1)$		-0.129^{**} (0.047)	-0.131*** (0.047)
Constant	-1.523***	-0.483*	0.613
	(0.0151)	(0.247)	(1.090)
Controls	No	Yes	Yes
Fixed effects	No 0.000	No	Yes
R-squared	0.002	0.066	0.076
Observations	16,758	16,758	16,758

Table C5: Robustness Check: Effect of the Hindu Succession Act Amendment (HSAA) on children's height-for-age z-score (HAZ); sample includes the reform state, Kerala

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. Table considers the *sample of children* 0-14 years with mothers who had already completed their education by the year of reform in their state, but also including the state of Kerala.

	(1)	(2)	(3)
	HAZ	HAZ	HAZ
Reform State*Mother married post-HSAA	$\begin{array}{c} 0.278^{***} \ (0.067) \end{array}$	$\begin{array}{c} 0.314^{***} \ (0.105) \end{array}$	0.237^{**} (0.105)
Child's age (in months)		-0.122^{***} (0.005)	-0.124^{***} (0.005)
Child's age squared		0.205^{***} (0.010)	0.208^{***} (0.010)
Child's gender (girl=1)		-0.093** (0.041)	-0.084** (0.041)
Mother's education		0.050^{***} (0.006)	0.050^{***} (0.006)
Mother's age		0.012^{***} (0.004)	$0.017 \\ (0.046)$
Mother's age at marriage		-1.98e-05 (0.011)	-0.007 (0.050)
Hindu (yes=1)		-0.053 (0.162)	-0.002 (0.158)
Rural (yes=1)		-0.221^{***} (0.064)	-0.215^{***} (0.067)
Own land (yes=1)		0.022	0.028
Low caste (yes=1)		(0.051) -0.124** (0.050)	(0.050) -0.127**
Below poverty line status (yes=1)		(0.050) - 0.046 (0.059)	(0.048) - 0.045 (0.059)
Constant	-1.414^{***} (0.025)	0.355 (0.362)	-0.585 (1.565)
Controls	No	Yes	Yes
Fixed effects	No	No	Yes
R-squared	0.002	0.137	0.152
Observations	8,387	8,387	8,387

Table C6: Robustness Check: Effect of the Hindu Succession Act Amendment (HSAA) on children's height-for-age z-score (HAZ); subsample of children aged 0-5 years old

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. Table considers the subsample of children aged between 0 and 5 with mothers who had already completed their education by the year of reform in their state.

	(1)	(2)	(3)
	HAZ	HAZ	HAZ
Reform State*Mother married post-HSAA	$\begin{array}{c} 0.135^{**} \ (0.063) \end{array}$	0.205^{**} (0.084)	$\begin{array}{c} 0.205^{**} \ (0.091) \end{array}$
Child's age (in months)		$0.019 \\ (0.012)$	0.025^{st} (0.013)
Child's age squared		-0.014^{**} (0.007)	-0.019** (0.008)
Child's gender (girl=1)		-0.108^{***} (0.033)	-0.097^{***} (0.033)
Mother's education		0.031^{***} (0.004)	0.032^{***} (0.004)
Mother's age		0.019^{***} (0.004)	-0.060 (0.047)
Mother's age at marriage		-0.015 (0.010)	$0.066 \\ (0.048)$
Hindu (yes=1)		-0.318^{***} (0.085)	-0.267^{***} (0.091)
Rural (yes=1)		-0.179^{***} (0.065)	-0.152** (0.067)
Own land (yes=1)		0.094**	0.102**
Low caste (yes=1)		$(0.039) \\ 0.021$	$(0.039) \\ 0.031$
Below poverty line status (yes=1)		(0.035) - 0.219^{***} (0.061)	(0.035) - 0.235^{***} (0.062)
Constant	-1.624^{***} (0.017)	-2.513^{***} (0.745)	-0.885 (2.058)
Controls	No	Yes	Yes
Fixed effects	No	No	Yes
R-squared	0.001	0.026	0.047
Observations	8,359	8,359	8,359

Table C7: Robustness Check: Effect of the Hindu Succession Act Amendment (HSAA)
on children's height-for-age z-score (HAZ); subsample of children aged 6-14 years old

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. Table considers the subsample of children aged between 6 and 14 with mothers who had already completed their education by the year of reform in their state.

	(4)			
	(1)	(2)	(3)	(4)
	ĤÁZ	Η̈́́́AZ	HÁZ	HÁZ
Reform State*Mother married post-	0.268***	0.252^{***}	0.227***	0.220***
HSAA	(0.079)	(0.071)	(0.073)	(0.071)
Child's age (in months)	()	-0.038***	-0.037***	-0.038***
china's age (in months)		(0.002)	(0.002)	(0.002)
Child's age squared		0.032^{***}	0.031^{***}	0.031***
- 0 I		(0.002)	(0.002)	(0.002)
Child's gender (girl=1)		-0.105***	-0.100***	-0.097***
6 (6)		(0.026)	(0.025)	(0.025)
Mother's education		0.040***	0.041^{***}	0.041***
		(0.004)	(0.004)	(0.004)
Mother's age		0.016^{***}	-0.015	-0.014
<u> </u>		(0.003)	(0.035)	(0.035)
Mother's age at marriage		-0.006	0.023	0.022
		(0.009)	(0.038)	(0.038)
Hindu (yes=1)		-0.187**	-0.152	-0.138
		(0.086)	(0.094)	(0.092)
Rural (yes=1)		-0.187***	-0.175***	-0.168***
		(0.051)	(0.054)	(0.053)
Own land $(yes=1)$		0.054	0.059*	0.059
-		(0.034)	(0.035)	(0.035)
Low caste $(yes=1)$		-0.039	-0.038	-0.039
		(0.036)	(0.035)	(0.036)
Below poverty line status (yes= 1)		-0.129***	-0.131***	-0.138***
		(0.047)	(0.047)	(0.047)
Constant	-1.523***	-0.487*	0.599	0.861***
	(0.021)	(0.248)	(1.086)	(0.067)
Controls	No	Yes	Yes	Yes
Fixed effects	No	No	Yes	Yes
R-squared	0.002	0.066	0.076	0.082
Observations	16,746	16,746	16,746	16,746

Table C8: Effect of the Hindu Succession Act Amendment (HSAA) on children's height-for-age z-score (HAZ)

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. Robust standard errors are clustered at the district level and appear in parentheses. Column 3 includes district and mother's year of marriage fixed effects, while in addition, column 4 also includes district-specific time trends. This table considers the *sample of children* 0-14 years old with mothers who had already completed their education by the year of reform in their state.

	(1)
VARIABLES	No. of children
Reform State*Mother married post-HSAA	-0.167^{***} (0.037)
Education	-0.033^{***} (0.003)
Age	$0.055 \\ (0.044)$
Age at marriage	-0.040 (0.045)
Hindu	-0.248^{***} (0.093)
Rural	0.083^{*} (0.042)
Own any land	$\begin{array}{c} 0.042 \ (0.034) \end{array}$
Low caste	0.087^{***} (0.031)
Below poverty line	0.287^{***} (0.038)
Constant	10.890^{***} (1.560)
Fixed effects	Yes
R-squared	0.601
Observations	6,208

Table C9: Effect of Hindu Succession Act Amendment (HSAA) on women's fertility

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. All regressions include district and year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. The dependent variable represents women's fertility i.e. the total number of children borne by her. Table considers *sample of women with children*, with no missing data on variables about mobility. The sample only includes women who had already completed their education by the year of the reform in their state.

	(1)
VARIABLES	HAZ
Number of children	-0.0272^{*} (0.015)
Child's age (in months)	-0.037^{***} (0.002)
Child's age squared	$\begin{array}{c} 0.031^{***} \ (0.002) \end{array}$
Child's gender (girl=1)	-0.093^{***} (0.026)
Mother's education	0.038^{***} (0.004)
Mother's age	-0.009 (0.036)
Mother's age at marriage	$\begin{array}{c} 0.019 \ (0.039) \end{array}$
Hindu (yes=1)	-0.136 (0.095)
Rural (yes=1)	-0.170^{***} (0.054)
Own land (yes=1)	$\begin{array}{c} 0.050 \ (0.036) \end{array}$
Low caste (yes=1)	-0.042 (0.036)
Below poverty line status (yes=1)	-0.127^{***} (0.047)
Constant	$\begin{array}{c} 0.527 \ (1.090) \end{array}$
Controls	Yes
Fixed effects	Yes
R-squared	0.075
Observations	16,744

Table C10: Mother's fertility and her child's height-for-age (HAZ)

Notes: ***, **, * denote significance at 1%, 5% and 10% respectively. Regression includes district and mother's year of marriage fixed effects. Robust standard errors are clustered at the district level and appear in parentheses. This table considers the *sample of children* 0-14 years old with mothers who had already completed their education by the year of reform in their state.

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