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Three Essays on the Social Determinants of Early Childhood Health and Development

Andrew Barenberg
University of Massachusetts - Amherst

THREE ESSAYS ON THE SOCIAL DETERMINANTS OF EARLY CHILDHOOD HEALTH AND DEVELOPMENT

A Dissertation Presented

by

ANDY BARENBERG

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Approved as to style and content by:

Arindrajit Dube, Chair

Mwangi wa Gĩthĩnji, Member

Ning Zhang, Member

Michael Ash, Department Chair
Economics

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ABSTRACT

THREE ESSAYS ON THE SOCIAL DETERMINANTS OF EARLY CHILDHOOD HEALTH AND DEVELOPMENT

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ANDY BARENBERG

B.A., UNIVERSITY OF MISSOURI-KANSAS CITY

M.A., UNIVERSITY OF MASSACHUSETTS AMHERST

Ph.D., UNIVERSITY OF MASSACHUSETTS AMHERST

Directed by: Professor Arindrajit Dube

This three-paper dissertation examines the social determinants of early childhood and in-utero health. The first chapter examines the impact of early childhood stunting on educational outcome in Tanzania. Using the extent of third-trimester overlap with the Tanzania hunger season to create an exogenous variation in stunting, I find that a one standard deviation stunting decreases educational achievement by .88 school years compared to a child's siblings. A placebo group not affected by the hunger season is used to confirm that in-utero nutrition deprivation is the cause of the education differences. The second paper utilizes the food price shocks and price increases to examine the impact of nutritional sufficiency on child development in four

sub-Saharan countries. I find adverse effects of third-trimester and early-childhood exposure to food price increases, but get inconsistent results on infancy that requires additional research. The final paper uses an instrumental variable method to determine the impact of public health spending on infant mortality in India. The results imply that a one percent of state-level GDP increase in public health prevents seven children deaths for every 1,000 live births. Together the three papers highlight the possible role investments in early childhood health could have in increasing human capabilities and well-being.

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INTRODUCTION

This dissertation consists of three papers on the social determinants of early childhood and in-utero health of children and the impacts that has on their later health outcomes. Although all three papers focus on less developed countries, I believe the lessons are more broadly applicable. My research here, and the growing body of existing literature on early childhood, highlight the extent that the implications of early childhood health has been vastly underestimated.

An example of the underestimation is the current political debates about inner-city violence, which I believe provides a valuable parable of the motives and themes of my research. Inner-city crime became a key focus at Donald Trump's 2016 Republican National Convention. Since then numerous commentators have noted that violent crime in the United States has actually decreased dramatically since the 1990s (Marshall Project, 2016), but few have discussed the influence of early childhood health in why crime has decreased. The causes of the decreases highlight the importance of research on early childhood health, its role in the wealth and inequality of a nation, and the depravity of our tendency to blame the victims of societies inequities.

During the higher crime rates of the 1990s Trump's current electoral opponent, Hillary Clinton, sounded the same themes as he does today: "They are often the kinds of kids that are called super-predators," she said (Gearan and Phillip, 2016). "No conscience, no empathy, we can talk about why they ended up that way, but first we have to bring them to heel." In 1994, her husband, President Bill Clinton, pushed through a crime bill that focused on increasing police forces and incarcerations. Similarly, cities and mayors focused on getting tough on crime policies -most notably from Rudy Giuliani in New York City. Policies of mass incarcerations followed the trends started in the 1980s in a failed effort to thwart the growing crime epidemic. By 1990, one in four black males in their twenties were under some version of criminal justice supervision. The Sentencing Project estimated the government cost of the incarceration at $6 billion, a number that leaves out the incredible toll on the lives of those involved (Mauer and Huling, 1995). Today, the move to mass incarceration keeps over 400,000 individuals behind bars, over ten times higher than in 1980.

While the costs of these policies piled up, evidence of their success was not forthcoming. As Manzi (2012) notes in reviewing the period, criminology regressions struggled to explain 10-20% of variation, and both regression analysis and randomized controlled trails of criminal policies rarely found same results when replicated. Recent research on incarceration finds that the increased recidivism of those imprisoned outweighs the deterrent and detainment effects to increase crime (Mueller-Smith, 2014). However, as the numerous efforts seemed fruitless, suddenly and unexpectedly, the crimerate started to go down, seemingly unconnected to any standard explanation of causes of crimes rates.

In 2000, Rick Nevin proposed a new explanation for the sudden turnaround in crime trends. Looking at data going back to 1900, Nevin found that trends in crime rates correlated with exposure to lead and gasoline paint (Nevin, 2000). Lead exposure in early childhood had been known to reduce IQ and the ability to control emotions. It was plausible that children exposed to lead would be more likely to be predisposed to violence later in life. Seven years after Nevin's publication, Jessica Wolpaw Reyes published her dissertation linking lead to crime. Armed with state-level data, she was able to show that drops in crime corresponded to when leaded gasoline was phased out at the state level, with a 20-30 year lag (Reyes, 2007). Her estimates implied that decreases in lead explained more than half of the reduction in crime. That year, Nevin returned with a second paper, showing that the relationship between lead exposure and early childhood and murder produced a similar strong fit in Britain, Canada, France, Australia, Finland, Italy, West Germany, and New Zealand, in addition to the United States (Nevin, 2007). When researchers looked at the neighborhood level in New Orleans (Mielke and Zahran, 2012) or followed several hundred individuals through time in Cincinnati (Wright et al., 2008), they found the same trends: exposure to high concentrations of lead in infancy were correlated to higher rates of aggravated assault in adulthood.

As I stated at the start of this introduction, I find this story to be a parable of my motives for the research contained in this dissertation. In the face of a seemingly intractable social ill of violent crime, politicians had reached for simple answers: more police, increased incarceration, bringing "superpredators to heel," and so on. These purported solutions not only failed to control crime, but they also came at an incredible cost, ripped at the fabric of society, and put the poor and the marginalized in conflict with the authorities. However, the trends did reverse, not because of any crime policy but because of a decision to protect the health of children.

To me, the question of early childhood health goes beyond the broad impacts of wealth and well-being of a nation as a whole, but also directly tied to the ethical question of equality. For example, the concentrations of lead dust in the air rose

higher in urban areas where the poor and black lived, and the resulting crime struck their communities harder than the rest (Mielke and Zahran, 2012). As the wealthier are better able to protect their young from the environmental harm and invest in their health, early childhood health becomes and important means via which inequality is transmitted from generation to generation. Janet Currie (2011) notes that the majority of differences in adult wages that economists can explain can be explained by a few variables on childhood background and score on standardized tests at age seven. Despite our love of pull-yourself-up-by-your-bootstraps stories, adult outcomes are largely determined by inequities in early childhood.

Compounding the injury of health disparities is the insult that the resulting differences in outcomes are the fault of the victims. At the height of the crime wave anxiety of the 1990s, Herrnstein and Murray published the Bell Curve (1996), arguing that IQ is mostly immutable and genetically determined. Their argument was that exogenously determined IQ explains the vast disparity of incomes and that social policy needed to accept that there was little that could be done. The same theory has also been given more scholarly treatment. Cunha and Heckman (2007) note that in Becker's (1993) comparison of human capital models to ability models, he assumes thatability is created purely by nature.

Herrnstein and Murray's argument was that the lower incomes of certain families and racial groups that persist from generation to generation are the result of inferior innate ability. My contention is that causation flows in the other direction, that inequality and poverty shape capabilities, and that any comments on the role of genetics are, at best, beyond the current state of our knowledge. To support their argument, Herrnstein and Murray used studies of identical twins raised separately to argue that genetics play a larger part than environment. My research in this dissertation helps illuminate the fatal flaw in such arguments. In Chapters 1 and 2, I show how the in-utero environment is a key determinant of the health and development of a child. If a shared in-utero environment plays a significant role in the formation of cognitive capabilities, as I argue in Chapter 1, these studies would be systematically flawed. Since adoptions commonly follow an unplanned and resource-strained pregnancy, this could play a significant influence in biasing these studies. The differences in outcomes are likely the result of inequities during gestation and claims that they show genetics as a scurrilous return to the claims of eugenics.

The tendency to incorrectly explain the outcomes of the social environment as being genetic extends to the key measure I use in Chapters 1 and 2, the children's Height-for-Age Z-score (HAZ). To create this score, a child's height is compared with the 2006 WHO growth standards. For the creation of these standards, the WHO collected data on 8,440 healthy breastfed infants and young children from Brazil,

Ghana, India, Norway, Oman, and the U.S. In order to ensure the children grew to their genetic potential, before birth pregnant mother's were selected that would be raising the child in a clean (non-smoking and open defecation-free) and nutritionally sufficient environment.

It could be thought - and it is most people's intuition - that the reason why some countries' height distributions are below the WHO standards is the result of genetic– not nutritional– differences. Many may find it implausible that there are no genetic differences that explain the differences in the average height of the Guatemalan Maya population and the US population, but this is indeed the case. Mayan children forced to flee from Guatemala to the US in the 1980s saw their growth accelerate (Smith et al., 2003). After eight years, their average height was near that of the US population. On average, Mayan children that grew up in the US were 10.2 cm taller than their Guatemalan counterparts,and only 13.7% were stunted compared to 71.6% in Guatemala.

A recent defense of the genetic argument in scholarly literature is foundin Panagariya (2013), who argued that WHO standards were inappropriate to assessthe Indian sub-continent. His concern was that the utilization of this standard was maligning the neo-liberal reforms that had brought growth but little change inthe HAZ scores. His argument was one of incredulity; surely, India's lower height distribution compared to poor Sub-Saharan African nations could not be the result of malnutrition. Panagariya argued it was implausible that India had worse nutrition when India outperformed these countries in per-capita GDP, female literacy, and health indicators such as infant mortality and life expectancy.

"[W]hat is needed," he argued, "is evidence that some sub-populations of children born and raised in India have managed to eliminate the gap with the WHO 2006 reference population." The evidence that Panagariya demanded could be found by reading the WHO report on the creation of the standards (Onis, 2007). The report includes the results from 1791 Indian children included in the study, and in no age group were Indian children (or children from any other countries) significantly different from the pooled averages. Over 70% of the variation in heights was due to differences between individuals within a site, and only 3% of the variation from came from differences between sites. Of the 54 country and age group categories, the report considers that no country's mean is greater than .5 SD from the mean, and only 10 are greater than .3 SD. The report notes that these differences "are relatively minor and are likely of little, if any, practical and/or clinical importance"(Onis, 2007, p. 61).

Pangariya's argument on the role of genetics is by residual, he assumes that "if we cannot think of anything else, it must be genetics. There is no direct evidence on

genetics anywhere in the paper" Tarozzi et al. (2013). But in six separate responding paper numerous alternative explanations are provided. Regional differences within India of rates of stunting correspond with rates of open defecation, which, due to population density, would have a much higher impact than in African countries (Spears et al., 2013; Buttenheim, 2008; Tarozzi et al., 2013). First born Indian children are taller than their African counterparts, it is the children who are lower in the birth order who drive the differences (Jayachandran and Pande, 2013; Coffey, 2013). This result is possibly driven by declining female bargaining power and, hence, lower nutritional status after the birth of the first child. Contrary to Panagriya's implication that India's growth should have eliminated widespread malnutrition, 70% of Indian children suffer from anemia, and micro-nutrient deficiencies are widespread (Lodha et al., 2013; Gupta et al., 2013). Similar to the Mayan case, Tarozzi (2008) finds that "children of Indian ethnicity who live in the UK have anthropometric outcomes comparable to [WHO] standards."

The key point here is not the specifics of the Indian case but the overall conclusion that regional genetic differences do not explain regional height differences. It is likely that genetics plays a significant role in defining an individual's potential height, but there is no evidence these genetic differences are distributed geographically. Research has consistently found that when any sufficiently large population is given a healthy, growing environment, it takes on a distribution resembling the international WHO standard. As such, the finding of a height distribution systematically below the WHO standard should be interpreted as evidence of nutritional or health barriers preventing the achievement of potential. That such extreme height differences seem so obviously linked to regional genetic differences but are, in fact not, should give pause to any other claim that differences are driven by genetics. Instead, it highlights the extreme difference in outcomes that arise from children's social environment and the potential that remains for substantial improvements in human development.

This point is magnified by examining the consequences of this stunting. A substantial and growing literature has established that a low HAZ score is a good indicator of not only physical growth stunting but also impaired cognitive development. Several studies from Brazil focusing on the role of diarrhea in early childhood cognitive development found a relationship between HAZ scores and aptitude tests and school outcomes (Niehaus et al., 2002; Alderman et al., 2006b; Dillingham and Guerrant, 2004). From 1960 to 1998, the Institute of Nutrition of Central America and Panama (INCAP) conducted experiments that gavenutritionally at-risk children nutritional supplementsandcomparedthemwith control groups with follow-up anthropometric measurements and cognitive tests. The study found that lower physical development stemmingfrom malnutrition strongly correlated with lower cognitive

development, which seemingly persists permanently (Engle and Fernández, 2010). Similarly, Alderman et al. (2006a) havefound that "improvements in height-for-age in preschoolers are associated with increased height as a young adult and number of grades of schooling completed."

The existing economic literature provides two philosophical frameworks to consider investments in children's health. The first is Human Capital theory, as developed by Becker (1993) and Grossman (1972). The second is the capabilities approach first promoted by Sen (1980, 1993) and given mathematical treatment by Cunha and Heckman (2007). I follow the latter approach. There are multiple reasons I prefer the capability approach. First, I believe the word capital should not be applied to something that is embedded in an individual, and can not be utilized except via their labor. Unlike physical capital, human capital can earn wealth while one is on vacation, nor can it be liquefied and transferred to new industries when markets are preferences change. Without these characteristics, "capital" is reduced from the vibrant role it played in Marx's theories to meaning just "something that will increase output in the future." A second concern is the limited utilitarian view of human health. As Sen (1980) argued, human health is not just a means to an end, but key end onto itself. A person's health is an essential aspect of wellbeing, and should not be regard simply as a tool for increasing wealth. A final and related philosophical complaint against the human capital theory is its moral nihilism: the moral question of if a child is entitled to food should not rest on the same calculus that determines if a robot is to be oiled.

In addition to the philosophical differences between human capital and capability approaches, there are also fundamental differences that arise from the mathematical model used to represent the theories. These differences are critical to understanding the implication of my research. In the standard human capital model of health first presented in Grossman (1972) the stock of human capital is given by the equation $H_t = (1-\delta)H_{t-1}+I_t$ where I is investment in human capital and δ is the depreciation rate. Almond and Currie (2011) note that, according to this model, even with low rates of depreciation the effects of differences at birth would be trivial by the time the individual reached working age. Recently economists have begun to reconsider the importance of early childhood in later life outcomes in ways that cannot be captured by Grossman's model.

Cunha and Heckman (2007) provides an alternative conceptual model for analyzing the development of human capabilities. We can consider a vector (H) of capabilities of a person-which can include cognitive, non-cognitive and physical health-as being determined in a point of time by environmental and background variables (E), parental factors (P), and by constant elasticity of substitution model between

investments in different ages.

$$H_{t+1} = f(E, P, A[\lambda I_{t-1}^{\rho} + (1-\lambda) I_t^{\rho}]^{1/\rho})$$

Cunha and Heckman define a sensitive period as

$$\frac{\partial H_{t+1}}{\partial I_t *} > \frac{\partial H_{t+1}}{\partial I_s}$$

for all s that is not t*. A critical period on the other hand is defined as

$$\frac{\partial H_{t+1}}{\partial I_s} = 0$$

for all periods not t* but for t*

$$\frac{\partial H_{t+1}}{\partial I_t *} > 0$$

.

According to the fetal origins hypothesis, the time in utero is, for some aspects of the vector H, a particularly sensitive period. Between earlier and later semesters of the pregnancy the later periods could be a sensitive period despite a high level of substitution between the periods. When a shock happens early in the pregnancy, the remaining time in utero can compensate for the effect; but, when shocks come later, there is no time to compensate. In other words, since the early trimesters are followed by periods with a high level of substitution (still being in utero), highly responsive compensating investments remain possible. On the other hand, later in the pregnancy, the post-natal period that follows has lower levels of substitution. Hence, compensating for a shock becomes more difficult at this stage of pregnancy. The impact of a shock when investments are fixed would be:

$$\frac{\partial H_{t+1}}{\partial \mu} = \lambda A[\lambda(\bar{I}_{t-1} + \mu)^{\rho} + (1-\lambda)\bar{I}_t^{\rho}]^{1-\rho/\rho}(\bar{I} + \mu)^{\rho-1}$$

Currie and Almond (2011) and Almond and Mazumder (2013) note that this implies the effects of a shock on a lower income family are likely to be higher, because the family's baseline investments will generally be lower and, hence, on a steeper section of the production function.

Cunha and Heckman call lambda (λ) the skill multiplier because through it investment at earlier age influences current health not only directly but also by influ-

encing the returns of investment at a later time. Because of this, the model can have dynamic complementaries. This is when

$$\frac{\partial^2 H_t(H,I)}{\partial H_t I_t} > 0$$

investments (or shocks) can increase (or decrease) the ability of an individual to learn or finish school.

We cannot observe this productivity of later investments directly but we can observe the outcomes that result. The observed impact ($\frac{\partial H_{t+1}}{\partial \mu}$) is not merely the result of biology but also the response of later investments in response to the shock ($\frac{\partial I_{t+1}}{\partial \mu}$). These later investments can be compensatory ($\frac{\partial I_{t+1}}{\partial \mu} > 0$) or reinforcing ($\frac{\partial I_{t+1}}{\delta \mu} < 0$). If the investment response is compensatory, and the compensating investment is not costless, an examination of the later effects would underestimate the cost of the initial shocks.

The parents' response function to early childhood shock will depend on the production function, their own preferences, and their resources and access to liquidity. Even where parents hold strong inequality aversion, a low level of substitution in the production function could make reinforcing investments optimal. Almond and Mazumder (2013) argue that the "bulk of empirical evidence to date suggest that parental investments reinforce initial endowment differences." But other studies find parental investments to be compensating. For example Behrman (1988); Pitt et al. (1990) demonstrate, in separate studies, that inequality aversion is a major feature in the allocation of food in households in India. Almond and Mazumder (2013) note that "parental responses could differ across these dimensions [of the vector H of health capabilities]. For example, it could be that parents might prefer to compensate for health endowments but reinforce cognitive ability endowments."

In Chapter One I am examining what Cunga and Heckman refer to as self-productivity. Self-productivity is the ability of health and capabilities to result in greater levels of health and productivities in later period. Formally self productivity is:

$$\frac{\partial^2 H_t(H_t, I_t, E, P)}{\partial H_{t-1}} > 0$$

The key problem in empirically estimating self productivity is that the various environmental and background factors (E) and parental (P) factors that influence H_{t-1} will also independently influence H_t. To solve this we need an exogenous source of variation in H_{t-1} that is not correlated with E and P.

Separating out these environmental, parental, and background factors is the motivation for examining the effects of third trimester overlap with the hunger season in the first chapter. In order to correctly estimate the impact of early childhood stunting it is necessary to have an exogenous variation not correlated with these other factors. I show that third trimester exposure to the hunger season results in negative shock in health that can be seen in under-5 height for age z-scores, a finding replicated in second chapter. The use of these shocks allows estimation of impacts that are exogenous to the conditions of the household. By combining this instrument with household fixed effects I purge any correlation between it and environmental, background and parental factors that is fixed through time. There could be concern that timing of birth could be correlated with other factors (for example, start of the school year) which could also influence educational achievement. I will show through a falsification test using regions that do not have seasonal hunger that this is not the case.

Previous efforts to research the impact of childhood stunting on educational outcomes have struggled to overcome the requirement to have an exogenous source of variation. For example, Alderman et al. (2009) analyze the impact of childhood malnutrition on schooling by instrumenting early childhood nutrition with famines and civil wars in Zimbabwe. They defend the excludability of this shock through tests of over-identification, but the validity of the instrument has some inherent implausibility. Certainly growing up after a civil war or famine as opposed to during one will effect future education in more ways than just nutritional status. Yamauchi (2012), like my model in the first paper, uses season of birth in Indonesia to instrument birth weight; he finds that higher birth weight correlates with an earlier start to school and fewer grades repeated. However, his paper does not incorporate a household fixed effect (possibly due to small sample size), requiring greater faith that month of birth is not correlated with socio-economic factors. I will show that this assumption is not valid for the Tanzanian case. By combining season of birth and household fixed effects, and providing a test of possible falsification I provide clear identification that effects are causal then previous research.

That sensitivity of the third trimester in utero to nutritional deprivation is reinforced by the second chapter, which finds a negative impact on children's height-for-age when the third trimester overlaps with food price crisis or food price rises. In the second chapter, I find a log point increase in local prices in the third-trimester in-utero corresponds with a 1.20 decrease in height-for-age Z-scores. In Tanzania the hunger season often sees local prices increases of up to 30 percent, according to the estimate in chapter one this should result in a .31 standard deviation decrease in the height-for-age. This effect is nearly identical to .29 finding in chapter one.

In Ethiopia and Malawi, which had significant local food price increases, third-trimester overlap with the crises caused decreases in height of .69 and .82 standard deviations respectively. These decreases are indicative that these two food price crises took a substantially larger toll than what we see from the seasonal hunger in Tanzania, but similar when scaled by the extent of the price swings. If the results from chapter one are externally valid for Ethiopia and Malawi, then they would imply that children whose third-trimester in-utero overlapped with the local crises would see decreased educational achievement of .61 and .72 grades completed, respectively. The worst hit cohort would be children born in August 2008 in Ethiopia. These children would experience the food price crisis both in third-trimester in-utero and throughout infancy. They will on average be a full standard deviation shorter and will on average complete .88 fewer grades of school. This is an incredible and substantial cost for failing to ensure food security for the population.

The comparability of the outcomes found in chapter one and chapter two are interesting in that causes were rather different in nature. The food price shocks studied in chapter two were a sudden, unprecedented shock on the global scale. In contrast, the seasonal shock of chapter one is a predictable yearly event. Indeed, a remarkable aspect of these effects is that they are an additional effect on top of preexisting levels of stunting in these populations. For children in Ethiopia born during the crisis the average height-for-age is below 2 standards deviations below the healthy population mean. That is, the mean of this population meets the medical definition of being stunted. But even if we look at the population not affected by the crises in Ethiopia the average HAZ scores are 1.5 standard deviations below the mean of a non-deprived population. This baseline needs to be kept in mind when considering the potential impact investment in child health could have. Efforts that just lower the day-to-day hunger or small seasonal variations in food access, could have potentially significant impacts on health and life outcomes.

Often the criticism is made that while it is easy to find the harms of ill-health, creating solutions is far more challenging. Even if ill-health has substantial negative impacts, government failure might make seemingly self-evident policies to fail cost effectiveness evaluation. This was the conclusion that Filmer and Pritchett (1999) came to in analyzing the impact of public health spending in developing countries. My third chapter directly counters this view. Co-authored with Deepankar Basu and Ceren Soylu, the paper examines the role that public health expenditures can play in decreasing infant mortality. Using a panel dataset of Indian States from 1983-84 to 2011-2012,we examine public health expenditures using a measure fiscal space available to the state. We find that an increase in state expenditure of one percent of the states domestic product causes a decrease of approximately seven infant deaths

per 1000 live births. These results were subjected to a large battery of robustness checks that ruled out other possible pathways between fiscal space and decreased infant mortality.

Our results imply an elasticity of infant mortality the health spending of -.36. In contrast, Filmer and Pritchett's study (1999) found an elasticity of only -.07. Our higher estimate would dramatically change Filmer and Pritchett's cost benefit analysis. But any cost benefit analysis should take in more than infant mortality rates. Although our paper shows public health spending is effective at lowering mortality, there is so much more to health than preventing death. The public health spending included spending family welfare and nutrition. This spending will not only prevent death but also enhance the well being of lives, and development of the children's capabilities.

The lesson of that final chapter is that we should not be so quick to seize evidence that there is nothing to be done, that government is powerless to alleviate the harms of poverty and malnutrition. Given what I have found on the relationship of early childhood health and education, countries that give up on early childhood health to focus on budgets do so at their peril. Such shortsighted policies will not only adversely affect their population today, but by limiting the development of the capabilities of their population, stunt the future growth of the nation.

CHAPTER 1

EFFECTS OF EARLY CHILDHOOD STUNTING ON SCHOOL ACHIEVEMENT: EVIDENCE FROM THE TANZANIA HUNGER SEASON

1.1 Abstract

This paper uses seasonal variation in food availability in Tanzania and the month of a childs birth to examine the impacts of early childhood stunting on later health and educational outcomes. Utilizing a combination of Two-Sample 2SLS models with and without household fixed effects, I derive estimates for the impact of early childhood stunting on later growth, body mass index, and educational achievement. I find that having third trimester in-utero overlap with the hunger season causes early childhood height to be decreased by .3 standard deviations. Of this stunting, only a statistically insignificant 21% persist into later childhood but with a higher resulting body mass index. These results give evidence that later investments can help compensate for earlier stunting. Turning to education outcomes, I find that malnutrition associated with a one standard deviation decrease in under-5 height causes a statistically significant .88 year decrease in future grades completed. To support the argument that the decrease in education is caused by in utero malnutrition, I show that in the Zanzibar & coastal urban regions that do not have seasonal hunger there is no decrease in educational achievement. These results imply that

the decreases in under-5 stunting that occurred between 1990 and 2010 will result in 6.6% increase in years of education for the Tanzanian children. If this trend were to continue to where the mean child in Tanzania was the same height as the mean child raised without nutritional constraints, they would on average complete 1.22 more years of schooling, a 15% increase.

1.2 Introduction

In the standard model of human capital accumulation the effect of a negative shock on human capital depreciates over time. For any non-trivial rate of depreciation, the effect of a shock in early childhood or while in-utero would be largely non-existent by the time they reach adulthood. Recently this model has come under question, as economists have begun to consider not only how shocks directly affect the capacity of a child, but also how they can influence the effectiveness of future developments of these capabilities. Additionally, economists are considering the possibility that certain periods are particularly sensitive or critical to human development and that the effects of mal-development may not be possible to reverse in later periods.

This paper adds to our understanding of the importance of early childhood development by using the hunger season in Tanzania to show that the effects of stunting in early childhood can influence the ability to acquire skills later in life. I show that a one standard deviation stunting in under-five height results in .88 fewer years of education completed compared to non-stunted siblings. I show that this effect on education persists despite the children largely being able to eliminate the height gap

with catch-up growth. I derive these results using time of birth and the extent of third trimester overlap with the Tanzania hunger season to get an exogenous source of variation in stunting. To confirm that the results are not driven by a spurious correlation, I create a falsification test using regions that do not experience the seasonal hunger. By utilizing individuals who migrated from regions of seasonal hunger to areas without seasonal hunger in comparison to the local populations of the latter regions, I show that the results are driven solely by the experience of in-utero malnutrition. I also use a bounding procedure to rule out the possibility that the stunting effect of the seasonal shock in the first stage is driven by sample selection from selective mortality.

Recent papers such as Majid (2013); Schultz-Nielsen et al. (2014) and Almond and Mazumder (2011) have used Ramadan to study the impact of in-utero malnutrition on later life outcomes. This paper confirms these results with the added value of examining a unique source of variation. But this paper also studies the impact of under-five stunting, a broadly used indicator of physical health and nutrition. Previous efforts to estimate the relationship between stunting and education used either simple OLS style controls (Hoddinott et al., 2013) or questionable instruments (Alderman et al., 2006a). The Ramadan studies lacked data on early childhood heights to address these questions. Here, I use Two Sample 2SLS methods (TS2SLS) to bridge across the different data sets, and consistently estimate the impact of stunting on schooling while accounting for differences in the characteristics of the two samples. Importantly, I am able to show that these results are similar when we limit our comparisons to treated versus untreated siblings within the same household-ruling

out an important source of unobserved heterogeneity. Finally, I explicitly consider, and rule out a negative spillover effect on siblings arising from compensatory investments by parents. By comparing within households, providing evidence on sibling spillovers, and utilizing multiple placebo groups for falsification tests, I am able to generate a much cleaner identification of the causal effect of childhood stunting on skill formation than had previously been possible.

1.3 Literature Review

A particular change has taken place on how both the medical profession and economists consider the impact of in-utero conditions. Up to late 1980s the prevailing research had suggested that the fetus acted as a perfect parasite and filter, taking in necessary nutrients while filtering out any toxins. Under such a schema, the mothers health status had little impact on either the immediate or long-term health of the fetus. In a series of articles in the 1980s and 1990s the British physician D.J. Barker (Barker, 2001, 1995; Barker et al., 1989; Barker and Osmond, 1986) argued that conditions in utero determined later life health outcomes. Barker argued that conditions such as diabetes, heart disease, and obesity had what he termed fetal origins. While Barkers fetal origins hypothesis gained traction through the 1990s, malnourishment was still thought to slow only the physical development of the fetus, while preserving resources to protect cognitive development. It has only been in the last 15 years that extensive medical and economics research has demonstrated that fetal shocks can and do impact cognitive development (Gluckman and Hanson, 2004; Almond and Mazumder, 2013).

The existing research indicates that shocks to the fetus during the third trimester of pregnancy in particular can result in significant impacts later in life. Barker (2001, p. 70) notes that studies of animals show that a fetus that grows slowly throughout gestation may have the same size at birth as a fetus whose growth was arrested for a period and then caught up. This is also found in humans. Research by Currie and Schwandt (2013) associates May births in the United States with lower birth weights, presumably due to the fact that flu season falls while the child is close to full term. Lokshin and Radyakin (2012a) observe similar seasonal effects, observing that children born during the monsoon season in India have height-for-age and weight-for-age z-scores that are .2 to .4 standard deviations lower.

A more singular, and more extreme, case is discussed by A.D. Stein, A.C. Ravelli, and L.H. Lumey (1995). Their research on the Dutch Hunger at the end of WWII finds a strong correlation between third trimester famine exposure on birth weight and future health outcome, with less impact from similar exposure in other trimesters. Because this famine hit all sectors of society in a definite geographical area, the event allowed Stein et al. to distinguish the effects of acute malnutrition from other socio-economic factors.

Schultz-Nielsen et al.(2014) provide a snapshot of the impact of cyclical periods of fasting in their study of Muslims living in Norway. They find that children born at the time corresponding to the seventh month of pregnancy during the fasting season of Ramadan results in worse labor market outcomes. This research corresponds with that of Majid (2013), who shows that children exposed in utero to Ramadan fasting in Indonesia have lower birth weight, score lower on general aptitude tests,

spend fewer hours studying at school, and work fewer hours as adults. For the results relating to school achievement, he consistently finds that third trimester exposure has the strongest effect. For adult outcomes, the relative effect of the various trimesters varies according to the specification used. (Conversely Almond and Mazumder (2011) find a negative result of Ramadan fasting corresponds to first trimester exposure.)

A substantial and growing literature has established that a low HAZ score is a good indicator of not only physical growth stunting but also impaired cognitive development. Several studies from Brazil focusing on the role of diarrhea in early childhood cognitive development found a relationship between HAZ scores and aptitude tests and school outcomes Niehaus et al. (2002); Alderman et al. (2006b); Dillingham and Guerrant (2004). From 1960 to 1998, the Institute of Nutrition of Central America and Panama (INCAP) conducted experiments that gavenutritionally at-risk children nutritional supplementsandcomparedthemwith control groups with follow-up anthropometric measurements and cognitive tests. The study found that lower physical development stemmingfrom malnutrition strongly correlatedwith lower cognitive development, which seemingly persists permanently (Engle and Fernández, 2010). Similarly, (Alderman et al., 2006a) havefound that "improvements in height-for-age in preschoolers are associated with increased height as a young adult and number of grades of schooling completed."

Research on the impacts of the early childhood stunting is complicated by fact that the environmental, parental, household and community aspects that determine a child's health almost certainly affects other life outcomes in other ways. A requirement for causal estimation is to find an exogenous source of variation in health. This

is the motivation for examining the effects of third trimester overlap with the hunger season. I will show in this papers that third trimester exposure to the hunger season results in negative shock in health that can be seen in under-5 height for age z-scores. By using household fixed effects I can purge from this instrument any correlation between it and environmental, background and parental factors that is fixed through time. The use of these shocks allows estimation of impacts that are exogenous to the conditions of the household. There could be concern that timing of birth could be correlated with other factors (for example, start of the school year) which could also influence educational achievement. I will show through a falsification test using regions that do not have seasonal hunger that this is not the case.

1.4 The Data

The data for this paper comes from two distinct survey sets. In the language of the two-sample 2SLS model I will develop later on I will refer to these two surveys as the first stage data and the second stage data, respectively. The first stage data comes from five rounds of the Demographic & Health Surveys conducted in 1991, 1996, 1999, 2006 and 2010. These surveys are focused on women, but for each mother the survey includes height data for all children under the age of five. The number of children under five in each survey round with their birth year is shown in Table 1.1.

The data for the second stage comes from the third round of the Tanzania National Panel Survey conducted in 2012-2013 as part of the World Banks Living Standard Measurement Survey. The first round ran from October 2008 to September 2009 and the second round ran from October 2010 to September 2011. The original

sample included 3265 households separated in 409 enumeration areas. The second round re-interviewed 97% of the original households (90% of individuals) and followed split off households for a total sample of 3924 households. There are 3198 household represented in both surveys. The units of observation used here are individuals in the household. Except were explicitly noted, references to TNPS survey will mean specifically the third round of data.

The TNPS section on anthropometrics was limited to individuals under 20 years old. It includes each individuals weight, height and if they were measured while standing or laying down. Given the requirement for the individual to be present during the survey, and some peoples refusal to be measured, only 83% of individuals in the study were measured. To calculate the HAZ score, each persons height is compared to the 2006 WHO growth standards chart, if they are under 5, and to the 2007 WHO growth standards chart if they are ages 5-19. The score is based upon height, age, gender and if they were measured while standing. Over the past 20 years, Tanzania has seen significant decreases in the extent of stunting. Between the 1990 DHS survey and the 2012-2013 survey, under-5 Height-For-Age Z-scores rose from -1.99 to -1.4, an improvement of .59 standard deviations. This improvement is substantial but the rate leaves much to be desired. At this rate of .03 standard deviations a year, it will take 47 years for Tanzania under-5 population to match the distribution of the non-nutritionally deprived chilren found from the WHO Standards studies. The extent of stunting, defined as having a height for age score of -1 or below, has on the other hand, been dropping rapidly in recent years. In the 2008-2009 LSMS survey, 38% of Tanzanians under the age of twenty were stunted, and in the 2010-

2011 survey 33% of youths were stunted. By the 2012-2013 study this had fallen to 28%.

The surveys differed in what they recorded regarding household conditions and assets. To create a common index valid across samples I used principle component analysis to reduce the multiple variables into a single wealth index. This procedure is widely used for DHS data and has been shown to be a consistent and valid estimator of household social and economic status. To construct the wealth index I used variables for the number of rooms in the household, the material used for the roof, floors, and ceilings, the source of water and type of latrine or toilet, and the households ownership of a range of assets. To prevent differing units of scale from biasing the results each variable was first converted into a z-score before calculating the index from the first component. The index was then divided into five quintiles. Because household size is correlated with wealth of the households the sample quintiles are not evenly divided into 20 percent each.

1.5 The Empirical Model

I will first explain the concept behind the two sample two stage least squares (TS2SLS) model, then derive the particular specification used to answer specific questions. To understand the TS2SLS model, consider a case where we desire to estimate the equation $Y = X\beta + \epsilon$, where in our case X is childrens height for age z-score along with various controls and Y is years of education achieved or another variable of interest. For an OLS estimate to be unbiased, X would have to be uncorrelated with the error term ϵ. That is obviously not true. The numerous determinants

Table 1.1. Summary Statistics for DHS Suveys

	Male		Female		All	
	Mean	SD	Mean	SD	Mean	SD
Height-Age Zscore	-1.91	1.47	-1.73	1.43	-1.82	1.45
Hunger Season Birth	0.23	0.42	0.25	0.44	0.24	0.43
Rural	0.77	0.42	0.82	0.38	0.79	0.40
Female	0	0	1	0	0.49	0.50
Birth Order	3.88	2.57	3.88	2.58	3.88	2.58
Poor	0.18	0.38	0.19	0.39	0.18	0.39
Middle Class	0.23	0.42	0.23	0.42	0.23	0.42
Richer	0.20	0.40	0.19	0.39	0.19	0.39
Richest	0.15	0.35	0.14	0.35	0.14	0.35

Mean and standard deviation of key variables from the DHS surveys. Data is of children under the the age of five for which we have height data.

of nutrition and health are also likely determinants of school achievement. To get at the exogenous variation in X we need an instrument Z that is correlated with X but, other than its impact on X, has no effect on Y.

Unfortunately we need both early age height for age scores and later age school outcome, which are not combined in the sample survey. Instead, we can consider two samples that include the instrument, endogenous, and control variables in the first sample, but where the endogenous variable is unobserved in the second sample. So we can consider a model where

$$X_2 = Z_2\Pi + \eta_2$$

and

Table 1.2. Summary Statistics For TNPS 2012-2013 Survey

	Male		Female		All	
	Mean	SD	Mean	SD	Mean	SD
Height-Age Zscore	-1.55	1.13	-1.26	1.06	-1.39	1.10
Education	4.28	2.75	4.59	2.86	4.44	2.81
bmi	16.8	2.27	17.4	3.43	17.1	2.96
Hunger Season Birth	0.25	0.43	0.25	0.43	0.25	0.43
Rural	0.71	0.45	0.69	0.46	0.70	0.46
Female	0	0	1	0	0.53	0.50
Birth Order	2.58	1.57	2.55	1.56	2.57	1.56
Poor	0.19	0.39	0.22	0.41	0.21	0.40
Middle Class	0.24	0.43	0.22	0.42	0.23	0.42
Richer	0.22	0.41	0.20	0.40	0.21	0.41
Richest	0.18	0.39	0.20	0.40	0.19	0.39

Mean and standard deviation of key variables from the LSMS surveys. Data is of children born between 1985-2005 to match the sample from the DHS.

$$X_1 = Z_1\Pi + \eta_1$$

but X_2 is unobserved. Despite being unable to observe X_2 we can still created a $\hat{X_2}$ of instrumented values of X_2 by utilizing the first stage relationship:

$$\hat{X_2} = Z_2(Z_1'Z_1)^{-1}Z_1'X_1$$

From this we can estimate the instrumented values of $\hat{\beta}_{ts2sls}$ via:

$$\hat{\beta}_{ts2sls} = (\hat{X_2}'\hat{X_2})^{-1}(\hat{X_2}'Y_2)$$

This can be rearranged to the simpler form of

$$\hat{\beta}_{ts2sls} = (Z_1'X_1/n_1)^{-1}C(Z_2Y_2/n_2)$$

where

$$C = (Z_1'Z_1/n_1)^{-1}(Z_2'Z_2/n_2)$$

This is the two sample 2SLS estimator from Inoue and Solon (2010).

A similar but different two stage IV estimator was presented in Angrist and Krueger (1992) where $\hat{\beta}_{ts2sls} = (Z_1'X_1/n_1)^{-1}(Z_2Y_2/n_2)$. Unlike the single sample case for TS2SLS, it is not equivalent to an IV estimator. The TS2SLS differs by the inclusion of a C matrix. This matrix provides a correction for any difference between the two samples. Important for this paper is that this correction maintains consistency when samples differ in their sampling stratification.

To account for household fixed effects, all the variables are transformed in each sample, taking out the household-specific means. Estimating the regression with the transformed variables, removes any household heterogeneity from the estimation yet maintains the standard TS2SLS framework. However, this procedure would require a degree of freedom adjustment to calculate appropriate standard errors. Since proper procedure for this where the degrees of freedom used differ in the two stages has not been derived I utilized bootstrapped standard errors in all the TS2SLS models.

I will use this TS2SLS estimator to answer three questions.

First: What is the impact of congenital stunting on later height achieved? Do children stunted by hunger season birth later experience catch-up growth and ultimately match the height of their peers? Specifically I will estimate:

$$H_{2013} = \beta_1 * \hat{H}_{U-5} + \beta_2 * BirthYear + \beta_3 * BORD + \beta_4 * Female + \sum \gamma_i * W_i + \mu_j + \epsilon \quad (1.1)$$

In this model $\hat{H}_{U-5}$ is the under-5 Height-for-Age Z-score as predicted from the first stage regression with the hunger season birth variable included as an instrument:

$$\hat{H}_{U-5} = \beta_1 * HS\_Birth + \beta_2 * BirthYear + \beta_3 * BORD + \beta_4 * Female + \sum \gamma_i * W_i + \mu_j + \epsilon \quad (1.2)$$

Where HS_Birth is the percent of third trimester that overlapped with the hunger season. The results of this first stage regression were presented in the previous section in Table 1.3. The first stage estimation uses DHS surveys, while the other variables in this equation are from the LSMS as explained in the two stage model above. H_{2013} is the Height-for-Age Z-score of children under the age of twenty as recorded in the 2012- 2013 LSMS survey. $BirthYear$ and $BORD$ are the childs birth year and order of birth relative to siblings respectively. W_i is four dummy variables for each of wealth quantiles created from the wealth index. μ_j are region level fixed effects. To create a household fixed effects for each variable I took the deviation from the household mean as described above. The two hypotheses I am interested in are:

1. $H_0 : \beta_1 > 0$ Do individuals stunted in utero maintain that stunting or does it disappear over time?
2. $H_0 : \beta_1 == 1$ If the stunting does not completely disappear can we say that some catch-up growth has occurred?

Second: What impact does childhood stunting have on later body mass index. Here I am estimating:

$$BMI_{2013} = \beta_1 * \hat{H}_{U-5} + \beta_2 * BirthYear + \beta_3 * BORD + \beta_4 * Female + \sum \gamma_i * W_i + \mu_j + \epsilon \quad (1.3)$$

Third: What impact does early childhood stunting have on the number of years of schooling completed? The model is as before:

$$S = \beta_1 * \hat{H}_{U-5} + \beta_2 * BirthYear + \beta_3 * BORD + \beta_4 * Female + \sum \gamma_i * W_i + \mu_j + \epsilon \tag{1.4}$$

where S is number of years of schooling achieved and the rest of the variables stay as specified above. The hypothesis I am testing here is if $H_0 : \beta_1 > 0$. Note that the variable $\hat{H}_{U-5}$ is height and so the expected sign here is positive.

1.6 The Lean Season

The instrument of quarter of birth depends on the seasonal variation in access to food. Moore et al. (1997); Lokshin and Radyakin (2012b); Moore et al. (2004); Yamauchi (2012) have all previously explored the impacts of seasonal variations in food availability on children. These studies, with the exception of Yamauchi (2012), focus on higher levels of mortality later in life. In this section I will present some evidence of the relationship between season of birth and health and educational outcomes and presenting evidence of the validity of season of birth as an instrument for stunting. In addition to arguing that season of birth provides a strong predictor of stunting I will address potential threats to the validity of season of birth being properly excludable.

1.6.1 Effect of Season of Birth on under-5 Stunting

Agriculture in Tanzania is shaped by two growing seasons: the masika, or long rainy season where food is planted in the spring and harvested from July to early September, and the vuli, or short rainy season with planting in October and November and harvesting in February. Prices for staples food tend to reach their lowest point each August and then rise to a high point from November through January. During these months, respondents to the LSMS survey are also most likely to say they fear hunger.

In Figure 1.1 I present a local polynomial graph of the relationship between height for age and month of birth from the LSMS. Those born in the first quarter, when their late pregnancy would have overlapped with the hungry season, have a mean height for age z-score of less than negative two. As the time between hunger season and birth grows the average height also increases, reaching its peak after the harvest in August. This pattern is consistent with the view that the in utero period is a sensitive period and time spent in utero after a shock helps to compensate for the earlier shock.

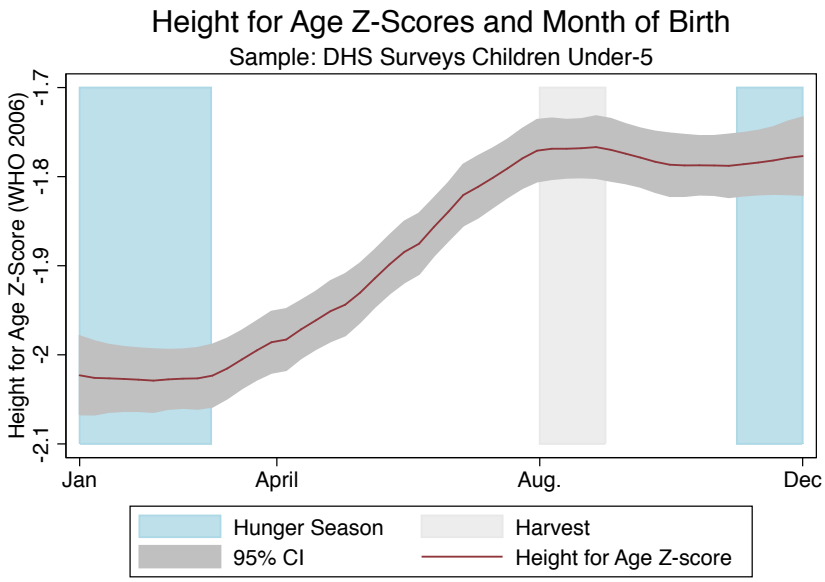
The first stage regressions are shown in Table 1.3 with both male and female children, the genders separated and a household fixed effects model. Robust standard errors clustered at the sampling cluster level are reported. In the difference of being born with all of the third trimester overlapping with the hunger season compared to there being no overlap is roughly .3 standard deviations. In all four models the result is statistically significant at the $p < .001$ level.

This third trimester effect of the hunger season seems to affect individuals at all levels of the society. As shown in Table 1.4, when these first stage regressions are run for each wealth quintile separately the hunger season variable is statistically significant in each one. However the effect is somewhat muted in the first and fifth quintile-both the poorest and the richest-and highest in the middle quintiles. This pattern is consistent with what we would expect from a seasonal hunger explanation. The lowest quintile likely faces year around deprivation while the upper classes have some ability to protect themselves from seasonal hunger. That the middle income levels would have the highest seasonal variation is to be expected. That the upper incomes levels are not able to completely protect their children from seasonal hunger is not as surprising as it may first seem. Stunted birth weight has resulted from even relatively minor nutritional deficiencies. For stunting to occur there need not be absolute starvation but merely relative deprivation. For example, Scharber (2014) found that periods of unemployment for mothers in Texas correlates with lower birth weights.

1.7 Empirical Findings

In this section I present the results of models explained in Section 1.5. In all the models here the standard errors reported are bootstrapped with clustering at the sample cluster level. To replicate the two-sample survey the sampling for the bootstrap was stratified by first and second stage sample, with the sample size in each bootstrap set to its respective sample size. One thousand replications were used in each model.

Figure 1.1. Height for Age Z Scores by Month of Birth



Local polynomial estimate of height for age z-score. Data from DHS survey of children under the age of five.

Table 1.3. Effects of Third Trimester in Hunger Season on Under-5 Height

	(1) All	(2) Females Only	(3) Males Only	(4) HH Fixed Effects
Hunger Season	-0.302***	-0.304***	-0.294***	-0.291***
	(0.0424)	(0.0555)	(0.0605)	(0.0589)
Birth Year	0.0118**	0.0124*	0.0110*	0.0239**
	(0.00449)	(0.00527)	(0.00554)	(0.00856)
Female	0.157***	0	0	0.188***
	(0.0249)	(.)	(.)	(0.0321)
Birth Order	-0.00239	-0.00370	-0.00184	0.0190
	(0.00531)	(0.00720)	(0.00741)	(0.0113)
Wealth Quantiles:				
Poor	0.0781	0.145*	0.00586	
	(0.0452)	(0.0581)	(0.0604)	
Middle Class	0.115*	0.180**	0.0469	
	(0.0478)	(0.0610)	(0.0599)	
Richer	0.393***	0.434***	0.350***	
	(0.0528)	(0.0605)	(0.0708)	
Richest	0.524***	0.641***	0.409***	
	(0.0549)	(0.0639)	(0.0769)	
Constant	-25.94**	-27.06*	-24.32*	-0.116***
	(8.931)	(10.48)	(11.03)	(0.00512)
Observations	15461	7703	7758	15688

Standard errors in parentheses, Standard errors are bootstrapped with clustering at sampling cluster level. Fixed effects for each region is included in each regression but not reported. Wealth quintiles were created by principle component analysis of household assets. Sample includes children under five but over 1 at the time of survey from five rounds of DHS surveys from 2005 to 2007. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.4. First Stage Regression - Effects of Hunger Season Birth on Under-5 height - by Wealth Quintiles.

	(1) Height (Poorest)	(2) Height (Poor)	(3) Height (Middle)	(4) Height (Richer)	(5) Height (Richest)
Hunger Season	-0.234**	-0.317**	-0.353***	-0.352***	-0.209*
	(0.0803)	(0.115)	(0.0921)	(0.0948)	(0.106)
Birth Year	-0.00688	-0.00426	0.0198**	-0.0174*	0.0701***
	(0.00831)	(0.00919)	(0.00715)	(0.00814)	(0.00809)
Female	0.0623	0.208***	0.200***	0.147*	0.236***
	(0.0506)	(0.0617)	(0.0539)	(0.0592)	(0.0623)
Birth Order	0.00608	-0.00264	0.0197	-0.0208	-0.0190
	(0.0106)	(0.0109)	(0.0107)	(0.0117)	(0.0150)
Constant	11.11	6.031	-41.80**	32.72*	-141.6***
	(16.55)	(18.35)	(14.24)	(16.21)	(16.09)
Observations	4152	2584	3582	2913	2230

Standard errors in parentheses

Regional fixed effects included but not reported.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.5. Effects of Early Childhood stunting on later childhood stunting

	(1) All	(2) Females Only	(3) Males Only	(4) HH Fixed Effects
Height-Age Zscore	0.646*	0.432	1.077	0.214
	(0.256)	(0.352)	(0.559)	(0.211)
Birth Year	0.00837	-0.0368***	0.0609***	0.00289
	(0.00786)	(0.0102)	(0.0133)	(0.00687)
Female	0.238***			0.223***
	(0.0618)			(0.0563)
Birth Order	-0.0218	-0.0191	-0.0132	-0.0274
	(0.0157)	(0.0195)	(0.0201)	(0.0159)
Wealth Quantiles:				
Poor	-0.0375	-0.153	0.105	
	(0.0824)	(0.103)	(0.128)	
Middle Class	0.174*	0.121	0.226	
	(0.0815)	(0.114)	(0.128)	
Richer	0.171	0.148	0.105	
	(0.143)	(0.208)	(0.261)	
Richest	0.493**	0.494	0.430	
	(0.169)	(0.257)	(0.265)	
Constant	-17.22	72.93***	-121.3***	-0.0448
	(15.99)	(20.74)	(27.16)	(0.0247)
KP F-Stat	52.99***	27.14***	28.51***	29.83***
	(8.014)	(5.327)	(6.405)	(5.216)
Observations	23577	11839	11738	23804

Standard errors in parentheses, Standard errors are bootstrapped with clustering at sampling cluster level and stratified by first stage and second stage samples. Fixed effects for each region is included in each regression but not reported. Wealth quintiles were created by principle component analysis of household assets. First stage sample included children under five but over 1 at the time of survey from five rounds of DHS surveys from 2005 to 2007. Second stage sample comes from the 2012-2013 Living Standards Measurement Survey of individuals born from 1985 to 2007. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1.6. Effects of Sibling's Hunger Season Birth on height, BMI and Education

	Height	Height	Height	Height
Hunger Season Birth For:				
Individual	-0.236**	-0.253**	-0.146	-0.142
	(0.0873)	(0.0963)	(0.109)	(0.131)
Other Siblings	-0.00317			
	(0.125)			
Older Siblings		0.0379		-0.0813
		(0.133)		(0.191)
Younger Siblings			-0.148	-0.0702
			(0.132)	(0.157)
Observations	1574	1286	887	616
	BMI	BMI	BMI	BMI
Individual	0.356*	0.311	0.366*	0.212
	(0.144)	(0.162)	(0.170)	(0.206)
Other Siblings	-0.337			
	(0.209)			
Older Siblings		-0.0416		-0.0804
		(0.212)		(0.290)
Younger Siblings			-0.393	-0.427
			(0.204)	(0.243)
Observations	2155	1449	1438	755
	Education	Education	Education	Education
Individual	-0.396***	-0.545***	-0.263	-0.479**
	(0.114)	(0.125)	(0.144)	(0.182)
Other Siblings	-0.0275			
	(0.179)			
Older Siblings		-0.153		-0.114
		(0.179)		(0.256)
Younger Siblings			0.0411	0.226
			(0.181)	(0.214)
Observations	3152	2012	2200	1089

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Robust standard errors clustered at the sample cluster level reported in parenthesis. Controls included but not reported are dummies of female, birth order and rural household; a birth year trend, wealth index quintiles and regional fixed effects.

Table 1.7. Two-Sample 2SLS Estimates Congenital Stunting on Years of Education

	(1) All	(2) Females Only	(3) Males Only	(4) HH Fixed Effects
Height-Age Zscore	1.183***	1.504**	0.927	0.878*
	(0.343)	(0.526)	(0.532)	(0.404)
Birth Year	0.300***	0.330***	0.265***	0.270***
	(0.00989)	(0.0136)	(0.0126)	(0.0142)
Female	-0.0765			-0.0368
	(0.0863)			(0.0972)
Birth Order	0.0406*	0.0970***	-0.0260	0.0119
	(0.0199)	(0.0277)	(0.0281)	(0.0280)
Wealth Quantiles:				
Poor	0.443***	0.178	0.649***	
	(0.122)	(0.168)	(0.160)	
Middle Class	0.812***	0.575**	1.018***	
	(0.121)	(0.177)	(0.155)	
Richer	1.352***	1.035***	1.639***	
	(0.186)	(0.265)	(0.256)	
Richest	1.979***	1.468***	2.426***	
	(0.225)	(0.367)	(0.298)	
Constant	-599.6***	-659.6***	-530.7***	0.0360
	(20.13)	(27.91)	(25.78)	(0.0465)
KP F-Stat	52.99***	27.14***	28.51***	29.83***
	(8.395)	(5.841)	(6.004)	(5.671)
Observations	23577	11839	11738	23804

Standard errors in parentheses, Standard errors are bootstrapped with clustering at sampling cluster level and stratified by first stage and second stage samples. Fixed effects for each region is included in each regression but not reported. Wealth quintiles were created by principle component analysis of household assets. First stage sample included children under five but over 1 at the time of survey from five rounds of DHS surveys from 2005 to 2007. Second stage sample comes from the 2012-2013 Living Standards Measurement Survey of individuals born from 1985 to 2007. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

1.7.1 Effect of early childhood stunting on stunting and BMI later in life

In Table 1.5 the results from the TS2SLS estimates on effect of early childhood stunting on later stunting are presented. They show interesting differences between the non-fixed effects model, male- and female-only models, and the household fixed effects model. In the model without household fixed effects (Model 1), 65% of the childhood stunting is maintained. Looking at the female- and male-only models, it appears that the gains are entirely attributable to female children, who maintain only 43% of their stunting, while for males the later stunting is 1.08 times the earlier stunting. It should be noted, however, that for models such as these, with only half the sample, the standard errors are large and not significantly different from each other or zero. It has been previously hypothesized that females in Tanzania suffer fewer effects of food deprivation because the higher levels of indoor and food preparation work give them greater access to food.

More interesting are the results in the household fixed effects model. The estimate of .21 is both much smaller than the non-household fixed effects model and is not significantly different then zero. In other words, with the inclusion of the household fixed effects there is no evidence stunting is preserved into later years. Since the first stage of the household fixed effects model was essentially identical to the non-fixed effects first stage; this result is not driven by different first stage results. So when some siblings are born in the hunger season and some are not there is a difference of under-5 stature; however, this difference dissipates to statistical non-significance over time. But when two children from different families are born in different seasons there is less collapsing of the difference over time.

We see similar differences between the models with and without household fixed effects when examining body mass index. In the model without household fixed effects there was no relationship (coefficient of -.038 and standard error of .47). But in the household fixed effects model, a one standard deviation in stunting is correlated with an 1.8 unit increase in the body mass index (with standard error of .85). This is consistent with the idea that unobserved household heterogeneity correlates with households with more hunger season births to lower availability of food or other determinants of health.

Another possible interpretation of this is that there might be spillover effects between siblings. The presence of a non-hunger season born child in the same household as a hunger season born child would make the stunting of the latter child more apparent, and quite possibly induce the parents to initiate compensatory food provision.

The question of if parents engage in compensatory or reinforcing behavior is related to but distinct from the question of if there are spillover effects on siblings. To review, compensatory investments is when investments increase in response to shock ($\frac{\delta I_{t+1}}{\delta \mu} > 0$) and reinforcing investment is when it fall in response to a negative shock ($\frac{\delta I_{t+1}}{\delta \mu} < 0$). These are comparing the investments in single comparison to the hypothetical case where their exposure differed. These compensating or reinforcing investment patterns become spillovers when they affect the investment made in a sibling. A spillover effect would be $\frac{\delta I_{t+1,i}}{\delta \mu_j} \neq 0$ where j is any child in the family other then i.

If spillover effects exist they would bias the results of the household fixed effects models. Luckily we can test for spillover effects directly. To do this we use the second stage data and set up a the reduced form model but include in addition a variable of the average third trimester exposure of siblings:

$$Y = \beta_1 * HS\_Birth_i + \beta_2 * HS\_Birth_j + \beta_3 * BirthYear + \beta_4 * BORD + \beta_5 * Female + \sum \gamma_i * W_i + \mu_j + \epsilon$$

In this model HS_Birth_j is the average third trimester exposure to siblings. I include variation where j is limited to only older or only younger siblings. For the dependent variable Y I consider all three of my key dependent variables, height for age in 2013 survey, BMI, and education achievement. The results from this test for the hunger season exposure variables are shown in Table 1.6. In all specifications the effects of siblings exposure are statistically insignificant. There is no evidence of spillover effects between siblings.

I will show in Section 1.8.2 that, over time, upper income households have become less likely to have children born during the hunger season; hence, low income households are overrepresented among those stunted from the hunger season. Some similar unobserved heterogeneity could be driving the differences between the models with fixed effects and those without. Although compensating investments are still a possible explanation the finding that siblings exposure has no effect assure us that the differences are not driven by decreases in siblings height. Whether driven by household heterogeneity or compensating investments the results are indicative that

when a stunted child is given adequate resources they are able to make early stunting dissipate with catch-up growth.

1.7.2 Effects of early childhood Stunting on Educational Outcomes.

The results of the TS2SLS estimates on the effects of congenital stunting on educational outcomes are presented in Table 1.7. In the full-sample non-household fixed effects model (Model 1) a one standard deviation in increase in early childhood increases the years of schooling achieved by 1.183 years. This result is significant at the $p < 0.001$ level. The household fixed effects model (Model 4) shows that a one standard deviation increase in childhood height relative to siblings increases years of school attained by .898 years. This is significant at the $p < .05$ level. So even if, among siblings, stunted children are able to reduce the height difference, there still remain statistically and socially significant differences in years of education achieved.

In the female and male models (Models 2 & 3, respectively) the female's education decreases by 1.5 years for a 1 standard deviation stunting ($p < .01$) while for boys there is a non-significant decrease of .93 years of schooling for one standard deviation unit of childhood stunting. While keeping in mind that these are not statistically significant differences, we might note the differences between these results and the previous section on the extent of catch-up growth. Assuming the results are not just driven by sampling error, females seem to recover from the physical effects of early childhood stunting better than their male counterparts, but suffer worse effects in the extent of school achieved. It is possible that while females have more access to food to compensate for early stunting, there might be more of a reinforcing investment pattern for female educational investments.

In the next section I will discuss evidence of household heterogeneity in the timing of births. This would bias the models without household fixed effects. On the other hand there is not evidence of spillover effects on siblings. Because of these reason he household fixed effects model is the preferred model. The result that a one standard deviation difference in stunting causes a .898 years difference in education needs to be put in perspective. Using the 2012-2013 LSMS survey we find that adults over the age of 16 have on average 7.9 years of education. A .898 year difference in education is equivalent to 11% of the average adult level of education. Between the 1990 DHS survey and the 2012-2013 survey, under-5 Height-For-Age Z-scores rose from -1.99 to -1.4, an improvement of .59 standard deviations. This improvement over time implies that, due to their superior nutritional status, children born in the period of 2005-2010 will likely complete .52 more years of school, a 6.6% difference, compared to children born from 1985-1990. If this upward trend were to continue to where the mean child in Tanzania was the same stature of the non-nutritionally deficient children in the WHO Standards, the gains from nutrition would cause 21% in the overall average educational achievement in Tanzanian adults.

1.8 Possible Sources of Bias

I consider two additional threats to identification that might bias the results above. They are 1) sample selection due to selective mortality and 2) month of birth be correlated with determinants of health and schooling.

1.8.1 Sample Selection

The DHS survey include a complete birth history for every woman in the survey. The birth history includes the month of birth of all children, even those that have passed away. From this data we are able to calculate the infant mortality rate by season of birth. Of children born during the hunger season 10.5% have passed away by their fifth birthday. In comparison for those born outside the hunger season there is a 9.2% under-5 mortality rate. This difference is significant at the $p < .01$ level.

A simple method for considering the possible range of the selection effect is to use a trimming procedure (Lee, 2009). The essential concept of is to take the portion of sample that is missing data due to mortality and to take the distribution of an equivalent proportion of the non-missing sample at both the top and bottom of the distribution. Using this we can calculate the counter-factual case if the children from the additional mortality had survived and were either among the tallest or the shortest and get the ultimate potential bounds of the bias.

The standard assumption in the literature on height and selection is that health can be conceived as a latent variable which is observed as height if above some level and death if below that level. If this is the case then the lower bound is likely to be correct. On the other hand there is the potential that naturally shorter people might be better suit for conserving energy and hence might survive deprivation better. In this case the bias would be in the direction of the upper bound.

In a t-test of the difference in height by the binary hungry season birth indicator (if the birth is in January, February, or March, the three months with the most third trimester overlap with the hunger season) find a statistically significant difference

in Height-for-Age for hunger season birth by -.18 standard deviations. Using Lee's (2009) trimming to create upper and lower bounds for changes in mortality might have effected this outcome we find an upper bounds of -.12 and lower bound of -.25. The upper bound maintains a statistically significant difference from zero. So even in the unlikely case that additional mortality came exclusively from the tallest portion of the population the hunger season births would still be significantly shorter than non-hunger season births. The lower bound, which accords to the more generally accepted latent variable concept of health, shows that mortality might be shaving up to .07 standard deviations from the seasonal differences.

Unfortunately given the second stage sample incomplete information on mortality we can not conduct a similar exercise to determine the potential effects on education outcomes. A more vital question is what is the possible impact on our two-sample 2SLS estimates of the impact of early child hood stunting on later stature and later educator outcomes. Consider two families that are identical on observed characteristics but one family scores lower in some unobserved vector Q of qualities that helped children recover from shocks. We would expect from this that the family with lower Q would both have higher mortality and a higher response to shocks. If this is the case the results would be biased downward.

1.8.2 Month of Birth

For the season of birth to be a valid instrument it needs to be uncorrelated with other determinants of educational outcomes. This is likely to be the case if when the child is born is essentially random. However it is quite possible that this is not the case. It could be that the agricultural season or migrant work season effect times

of conception. Or they could be correlated with religious holidays. There might be different seasonal differences for younger or first time parents due to wedding or school seasons.

Another possible bias is if the timing of children's birth are not randomly distributed by correlated to other household characteristics. If this is the case it would bias the models without household fixed effects. To check for this I conducted multiple t-tests and linear probability model (LPM) tests of the DHS sample to search for possible trends in season of birth. None were found when the data was limited to the birth years I use for my regression: 1985 - 2005. Using a binary indicator for if the child was born in the three months (January, February, March) that would have the most third trimester over lap with hunger season I tested if any wealth quintile's proportion of hunger seasons births differed from the 1/4 proportion expected from random distribution. None did. Nor did any wealth quintile proportion of hunger season birth differ from the other quintiles. Using a LPM with controls for region, birth year, birth order the wealth quintiles still had no relationship. These results were robust to both including and excluding children who had passed away of limiting to first born or not first born. Similar t-tests and LPM found no relationship between being first born and a hunger season birth.

To check the robustness of this I repeated the exercise including in the sample people who fell outside birth years I include in my sample. Including people born before 1985 had no effect on the results. However including children born after the 2005 cut off induced substantial changes however. With these children included all quintiles of wealth except for the lowest saw a decrease in portions of births born

during the hunger season. For the second to fifth quintile portion of births during the hunger season fell to 20%. With this change the lowest quintile now has a statistically significant higher portion of births in the hunger season. A possible explanation is that increased access to birth control is allowing families to plan pregnancies to avoid the hunger season. In 1992 Tanzania initiated a National Population Policy that called for universal access to modern contraceptives. From 1996 to 2004 the use of modern contraceptives increased from 18.4% to 26 percent.

Limiting the sample to before 2006 and then iteratively adding prior years I tested for each of upper four wealth quintiles when its proportion of hunger season birth became statistically different than 1/4. For the second and fourth quintile only the 2005 birth set by itself was significant, for the third quintile everything after 1994 was significant and for richest quintile everything after 1996 was significant. Despite these trends in the third and fifth quintiles iteratively adding years prior to 2005 was unable to find any subset of years in the sample where the poorest quintile was statistically significantly different than the other quintiles.

To summarize the above there appears to be in the second half my sample (those born after the mid 1990's) a lower portion of babies born during the hunger season for all but the poorest households. While the trends on observed characteristics can controlled for there is still the possibility that these trends are correlated with non-observed differences. Because of this, the models estimated without household fixed effects should be considered with skepticism and I report the household fixed effect models to be the preferred ones.

1.8.3 Relationship between Hunger Season and Later School Outcomes

A possible bias to my results could arise if season of birth influence educational achievement by means other than in-utero nutrition. Here we will look more closely at the relationship between birthdate and education and consider other possible pathways. Most notably there could be a relationship between the third trimester overlap and being born after the start of the school year. I will argue that the likely effect if any of a school year effect would be to create a small positive effect for those with third trimester exposure on education. This would have an effect of biasing my results downward. In the section that follows I will attempt to confirm this analysis using a falsification test from regions without seasonal hunger.

Figure 1.2 shows the relationship between the age when a child begins school and their month of birth. Most interesting about this graph is that there is not a linear trend across the year but instead almost a perfect mirror image of the distribution of height in Figure 1.1. Those born in the first quarter, when the third trimester would have overlapped with the hunger season, start the latest. This fact is important, as one potential source of bias could be if the timing of the start of the school year drove early in year births to start school at younger age. There is some evidence that starting younger, when children are not as emotionally or mentally prepared for the challenges of school, leads to worse school outcomes. The later starting age for hunger season births indicates that this is not driving the results here and might, in fact, be muting the actual effects. These results could be the outcome of parents subjectively trying to decide when their children are ready and thus holding congenitally stunted

children back a year. Again, one interesting observation is that the children born following the harvest in August start the earliest.

Conversely we can consider the opposite side of potential start of the school year effects. Angrist and Krueger (1992) consider the effects of the month of birth as driving total school achievement in the U.S. by assuming a certain percent will drop out once achieving the legal age to do so. For this population, those born in months where they would start at later age would achieve less education then those born in other months. The institutional context here is important: the results in the U.S. are driven by compulsory education being limited to a specific age range. Although the 1978 Tanzania law stated that [i]t shall be compulsory for every child who has attained the age of seven years but has not attained the age of thirteen years to be enrolled for primary education, a 1995 amendment rephrased the requirement to [i]t shall be compulsory for every child who has reached the age of seven years to be enrolled for primary education. With this change, the number of complete years of school replaced age as determining the end of compulsory education. Since my regression reported later will be limited to children born after 1985, none would have reached the 13 year cut off of the 1978 law prior to it being amended. Hence, the law that would apply to children in my sample would require achieving seven years of education regardless of the age at which they started school.

If, somehow, there was still some start of school year effect that was driving the results we would expect the children born in months that start school later to either leave school at a parallel later age or for there to be no season difference between season of birth and age leaving school. In Figure 1.3 I present the age leaving school

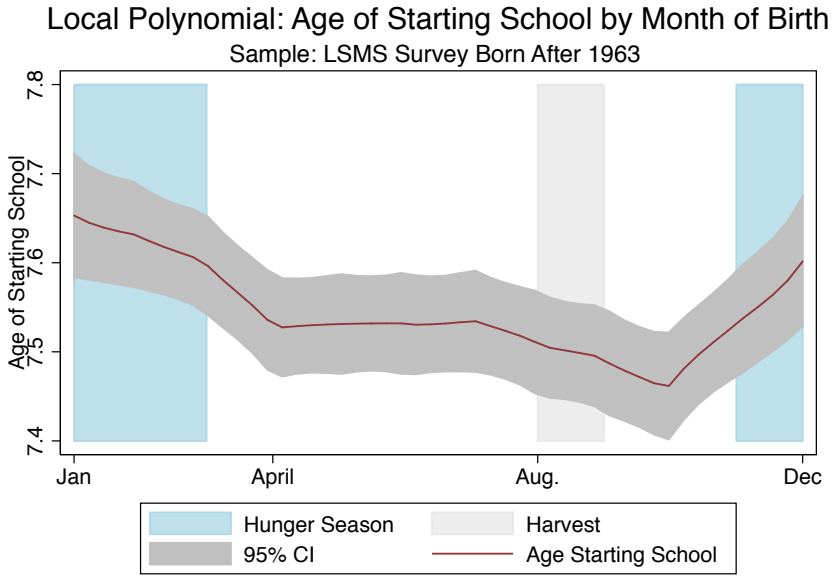
by month of birth for those born since independence (1963) and who are of at least 15 years of age. Instead of the pattern we would expect from a timing of the start of the school year we see that those starting later (those born in the first quarter) leave at a younger age, while those starting earlier (those born after the harvest) leave school at a later age.

Using the same sample as in Figure 1.3 we can look at the percent that attempt to take the Primary School Leaving Exam, necessary to go on to secondary education. In Figure 1.4 we see that those born at the start of the year are less likely to attempt the test, with the probability of taking the test rising for those born after the harvest. To summarize this section: children born when the third trimester overlaps with the hunger season have lower height for age z-scores, they start school at an older age, they leave school at an older age and they are less likely to take the PSLE test to go on to secondary school.

1.8.4 Falsification Test with Zanzibar and Coastal Urban Areas.

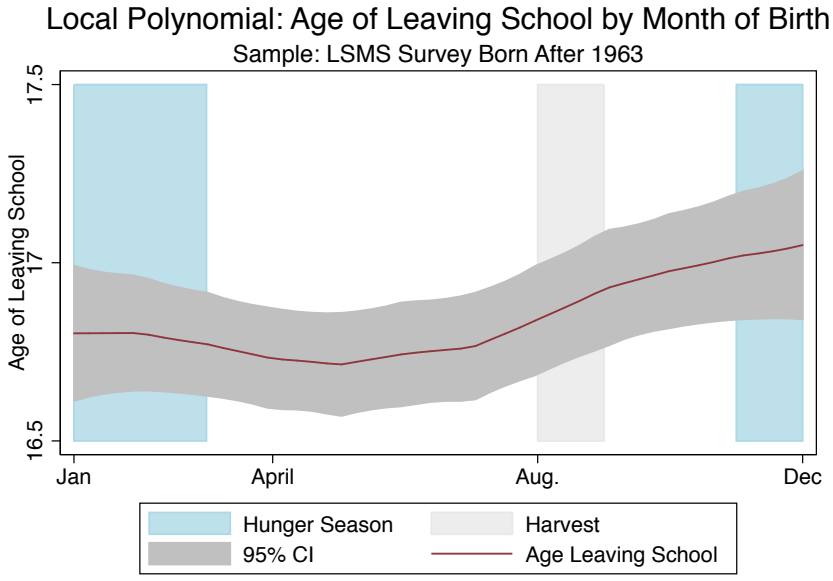
In the previous section I presented evidence that overlap between the third trimester of pregnancy and the hunger season is correlated with under-5 stunting. Additionally, I presented arguments that this stunting determines later educational outcomes and that any relationship between season of birth and the school year was likely to bias the estimates towards underestimating the relationship. Here I will provide a more formal falsification test of this argument. If the relationship with school year was driving the results this relationship should be found in all areas of the country regardless of if there was a strong hunger season stunting effect or not. If we find evidence of a season of birth effect on education in areas that do not have

Figure 1.2. Age of Starting School by Month of Birth



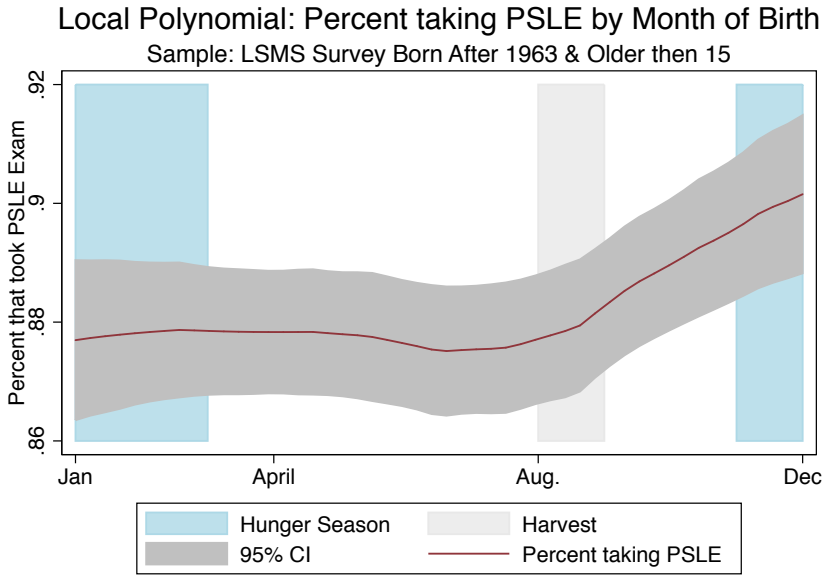
Local polynomial estimate of age of starting school by month of birth. Data from LSMS, limited to people born since Tanzanian Independence in 1963.

Figure 1.3. Age of Leaving School by Month of Birth



Local polynomial estimate of age of leaving school by month of birth. Data from LSMS, limited to people born since Tanzanian Independence in 1963 and who have left school.

Figure 1.4. Percent taking PSLE by Month of Birth



Local polynomial estimate of age of start school by month of birth. Data from LSMS, limited to people born since Tanzanian Independence in 1963 and over age of 15.

a season of birth effect on stunting this falsify my assumption. On the other hand, finding no effects on education there would provide evidence that the relationship is purely nutritional.

Two potential candidate regions are Zanzibar and the coastal urban areas. Zanzibar is a chain of islands that were united with the mainland in 1964. Although agriculture still makes up over a third of the regions domestic product compared to the mainland, the agricultural product tends to be export oriented. Fishing and tourism play a large role in the economy. The coastal urban areas share with Zanzibar a traditional diet that differs from the interior of the country both due to the presence of fish and the history of Arab colonization. Anthropologists have categorized distinct "traditional-coastal" and "traditional-inland" diets (Keding 2011). Keding et al. note that the coastal diet is "characterized by fruits, nuts, starchy plants, and fishcharacterized by fruits, nuts, starchy plants, and fish" as opposed to the inland diet of "cereals, oils and fats, and vegetables." Due to a lower reliance on subsistence agriculture and foods effect by the seasonal rainfall it is expected that the hunger seasonal would have a muted or nonexistent pattern in Zanzibar and coastal urban areas. A third possible area is Kagera, next to Lake Victoria, which is known (PrinciplesOfMedicineInAfrica:2004, 2004) as having lower seasonal hunger due to fishing.

To test the appropriateness of the excludability of season of birth I provide here the results of first stage and reduced form regressions for three regions of interior Tanzania (where we expect to find a hunger season effect) and the coastal urban and Zanzibar regions (where we do not expect to find a hunger season effect). Due to

the small sample, I do not include separate estimates for Kagera. For both the first stage and second stage models I present the household fixed effects model. As above, the first stage regression on the effect of season of birth on height is taken from DHS data of children under the age of five. The reduced form regression of the effect of season of birth on education uses data from the LSMS surveys. In both regressions the samples are limited to the years 1985 to 2005.

The first stage is in the form of

$$H_{U-5} = \beta_1 * HS\_Birth + \beta_2 * BirthYear + \beta_3 * BORD + \beta_4 * Female + \alpha_H + \epsilon$$

and the reduced form regression is:

$$S = \beta_1 * HS\_Birth + \beta_2 * BirthYear + \beta_3 * BORD + \beta_4 * Female + \alpha_H + \epsilon$$

where α is the household fixed effects.

The results of these tests are shown in Table 1.8. The first stage regressions show the expected patterns with a strong hunger season birth effect in the interior of Tanzania but with a muted and statistically insignificant effect in Zanzibar and no effect in coastal urban areas. The reduced form regression also follow this pattern; a strong statistically significant effect of the hunger season on education in the interior but statistically insignificant effects in the coastal urban and Zanzibar areas. Looking at the coefficients for the interior and Zanzibar it is interesting to note that they moved in parallel with an almost 1-to-1 relationship between stunting and fewer years of education. Although the difference are very small and statistically insignificant

we do not see this almost 1-to-1 relationship between stunting and education in the coastal urban areas. This could be due to small sample size and random sample selection. Alternatively this could be the result of in migration to urban areas. Luckily the LSMS data used in the reduced form includes information on whether the subjects moved from another region, and from where they moved.

In Table 1.9 I present the results of separating the population of the coastal urban areas into the those natively born in the area and those who migrated to the area. The coefficient for those born in the area is now small and statistically insignificant, but positive like the first stage regression. Using a one tail test of if the native coastal urban hunger season effect is smaller than the that for the interior of Tanzania we find it is significant at the 5% level. In column 4 we see that the school achievement of those who migrated into coastal urban areas is similar to the those who were born and remained in the interior. While the small sample sizes makes it impossible for us to be sure that these relationships are not driven by random sample selection they do support the argument that the results are driven by nutrition and congenital stunting.

To solve the issue of low sample sizes we can expand the sample to include those born before 1985. In Table 1.10, I separate out three samples; those born in areas without seasonal hunger; those born in areas in which we have not found evidence of seasonal hunger; and those born in areas of seasonal hunger currently living in areas without seasonal hunger. Areas without seasonal hunger are defined as being coastal urban areas, Zanzibar, and Kagera. All other regions are included in estimates of areas with seasonal hunger. The table also reports the results of across model

tests of the equivalence of the hunger season coefficients. The coefficient on hunger season for those born in areas without seasonal hunger is positive with a statistically insignificant difference from zero. There is no effect for those who did not experience seasonal in-utero nutritional constraints. This non-effect is statistically significantly different from both those born and living in areas with seasonal hunger and from those living with them in the non-seasonal hunger areas but having migrated from areas of seasonal hunger.

These results show that there is an effect of the hunger season on education only on people born in areas where the seasonal hunger pattern exists. For those who migrate from areas with seasonal hunger to areas without the result show a relationship between education and season of birth that resembles that of their birth region and not their adopted region. Altogether these tests provide strong evidence that the relationship between season of birth and educational outcomes is driven solely by in-utero nutritional constraints.

1.9 Conclusion

There are three important implications of the findings presented in this paper. First, consistent with a small but growing body of literature, I find in-utero environments matter, and economic shocks affect this environment. This can have lasting impact, both biological and economic. Second, the good news is that congenital stunting seems to be responsive to investments, as only 2/3 of the stunting seems to persist (even less when comparisons are made within households). Moreover, this catch-up does not seem to come with a penalty for other siblings there is no clear

Table 1.8. Effects between Siblings on Hunger Season Birth between Siblings on Height & Education in Interior Tanzania, Zanzibar & Coastal Urban Areas

	(1)	(2)	(3)	(4)	(5)	(6)
	Tanzania Interior		Zanzibar		Coastal Urban	
	Height	Education	Height	Education	Height	Education
Hunger Season	-0.305***	-0.332**	-0.118	-0.101	0.0283	-0.0761
	(0.0595)	(0.107)	(0.213)	(0.283)	(0.238)	(0.184)
Birth Year	0.0220*	0.178***	0.142***	0.124***	0.0350	0.169***
	(0.00874)	(0.00487)	(0.0331)	(0.0133)	(0.0222)	(0.00895)
Birth Order	0.0172	-0.114***	-0.193***	-0.0177	0.0596	0.122
	(0.0115)	(0.0266)	(0.0491)	(0.0678)	(0.0473)	(0.0672)
Female	0.194***	0.197**	0.221*	0.303*	0.0395	-0.260
	(0.0327)	(0.0679)	(0.0905)	(0.126)	(0.157)	(0.135)
Constant	-0.120***	-0.287***	-0.0857***	-0.174	-0.0541*	-0.493***
	(0.00551)	(0.0335)	(0.0144)	(0.0987)	(0.0227)	(0.0710)
Observations	13918	5758	996	878	774	1469

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Robust standard errors clustered at the sample cluster level reported in parenthesis. Sample limited to those born between 1985 and 2005. Household fixed effects used in all models reported here.

Table 1.9. Effects between Siblings on Hunger Season Birth on Education in Interior Tanzania, Zanzibar & Coastal Urban Areas

	(1)	(2)	(3)	(4)
	Tanzania Interior	Coastal Urban		
	Education	All Education	Native Born Education	Migrant Education
Hunger Season	-0.332**	-0.0761	0.105	-0.271
	(0.107)	(0.184)	(0.212)	(0.344)
Birth Year	0.178***	0.169***	0.170***	0.149***
	(0.00487)	(0.00895)	(0.0129)	(0.0130)
Birth Order	-0.114***	0.122	0.0403	0.202
	(0.0266)	(0.0672)	(0.0806)	(0.115)
Female	0.197**	-0.260	-0.167	-0.312
	(0.0679)	(0.135)	(0.184)	(0.171)
Constant	-0.287***	-0.493***	-0.346**	-0.548***
	(0.0335)	(0.0710)	(0.119)	(0.101)
Observations	5758	1469	784	729

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Robust standard errors clustered at the sample cluster level reported in parenthesis. Sample comes from second stage data from the LSMS surveys of those born between 1985 and 2005. Household fixed effects used in all models reported here.

Table 1.10. Effects between Siblings on Hunger Season Birth on Education by Region of Birth, Expanded Sample

	(1) Born in Areas Without Seasonal Hunger	(2) Born in Areas With Seasonal Hunger	(3) Migrated From Areas With Seasonal Hunger
	Education	Education	Education
Hunger Season	0.137	-0.294**	-0.488
	(0.155)	(0.106)	(0.285)
Birth Year	0.122***	0.137***	0.0920***
	(0.00522)	(0.00298)	(0.00809)
Female	-0.179	-0.300***	-0.917***
	(0.117)	(0.0585)	(0.132)
Birth Order	0.0380	-0.0237	0.233**
	(0.0566)	(0.0280)	(0.0876)
Constant	-0.0417	-0.0657***	-0.196***
	(0.0294)	(0.0111)	(0.0345)
Observations	2930	9198	1523
Test of if Hunger Season Coefficient Differnt From Areas without Seasonal Hunger:			
Chi-Sq.		5.171	3.852
P-Value		0.0230	0.0497

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Robust standard errors clustered at the sample cluster level reported in parenthesis. Sample comes from second stage data from the LSMS surveys and is limited to those born before 2006 but includes those born before 1985. Household fixed effects used in all models reported here.

negative spillover on sibling health or education. Third, the bad news is that the physical catch up does not erase losses in skill formation. Instead, these shocks appear to leave a permanent mark via reduced school attainment, and hence likely on economic and social opportunities.

Additionally, these results suggest that nutritional security could have a significant impact on macroeconomic outcomes. The results from the more conservative estimates from the comparison of siblings imply that the decreases in under-5 stunting that occurred between 1990 and 2010 will result in 6.6% increase in years of education for the Tanzanian children. If this trend were to continue to where the mean child in Tanzania was the same height as the mean child raised without nutritional constraints, they would on average complete 1.22 more years of schooling, a 15% increase. If the human capital argument that education increases productivity is correct these nutritional gains could have a significant impact on growth.

This persistent effect could reflect a permanent negative effect of the in-utero shock upon cognitive development. Or it could be that the cognitive effect is reversible, but too little (or not appropriate) investment is provided to reverse it. Future research will be important to discern these channels which have important policy implications.

CHAPTER 2

EFFECTS OF THE 2007-2008 FOOD PRICE SPIKES ON IN-UTERO AND EARLY CHILDHOOD DEVELOPMENT

2.1 Introduction

Throughout 2007 and the first two quarters of 2008 the world saw rapid increases in the prices of food. During these 18 months, the FAO's world food price index rose 60% with an even higher increase for the main staple foods such as maize (Arndt et al., 2012b). For much or the world's poor who are net consumers of food products, the price increases decreased their ability to afford food and other necessities. Researchers following the effects of the crisis have found negative effect on the food crisis on the height of the young. This paper adds to this research by studying how the effects differ according to the age of the child.

In this study, I use the 2007-2008 food price crisis impacts on four sub-Saharan African countries - Ethiopia, Malawi, Uganda, and Tanzania - to examine the impact on the children's development from pre-conception to early childhood. There are three goals of this study. The first goal is to discover the impact that the food price crisis had on children's development. As government stockpiles of food stores has decreased over the past two decades, and expectations of increased variation in harvest driven by a changing climate, understanding the potential impact of movements in staple food prices becomes more critical. The second goal is to identify

differences in sensitivity of children's development to a shock at different stages of development. Greater understanding of how and when a shock affects development can help guide future event studies examining in-utero shocks. The third goal is to review how the food price shocks differed in their impacts across the four countries. Looking at the heterogeneity of the country experiences can help guide research in countries can protect their population from future food prices crises.

Exisiting research has found that crisis had negative effects in sub-Saharan African countries despite high portions of the population being involved in agriculture. For example, Anríquez et al. (2013) find "food price spikes not only reduce the mean consumption of dietary energy but also worsen the distribution of food calories, further deteriorating the nutritional status of populations." In a study of children in Mozambique Arndt et al. (2012a) note that "the prevalence of underweight amongst children rises in response to a higher inflation rate for basic food products. The very high food inflation during 2008/09 was responsible for an extra 39,000 moderately underweight and 24,000 severely underweight children." Brinkman et al. (2010) note "that energy consumption declined during 2006-2010 in nearly all developing regions, resulting potentially in an additional 457 million people (of 4.5 billion) at risk of being hungry and many more unable to afford the dietary quality required to perform, develop, and grow well." Brinkman et al. (2010) also note that nutritional diversity can fall as family cope by substituting their diet to the cheapest per calorie foods and resulting in "increased odds of child stunting and chronic energy deficiency among mothers." Cornia et al. (2012) also find that in Malawi child visits to feeding centers tripled during the crisis.

Darnton-Hill and Cogill (2010); Brinkman et al. (2010); Kumar and Quisumbing (2013) all note that nutritional deficiency from the spike tend to hit hardest on women and children, and that pregnant women and infants are especially vulnerable. This vulnerability can have long term implications as a growing body of research is finding that in-utero and early childhood development is a sensitive period which can effect life outcomes (Gluckman and Hanson, 2004; Almond and Mazumder, 2013). A key indicator used to identify if a population of children has had their development stunted is their height, measure in height-for-age zscores, as compared to the 2006 WHO child growth standards. This indicator taken on greater importance in recent years as research has shown that maldevelopment in height due to deprivations is an indicator of maldevelopment of cognitive and noncognitive capabilities (Niehaus et al., 2002; Alderman et al., 2006b; Dillingham and Guerrant, 2004). In addition to the immediate toll on human welfare we now know that the deprivations of a shock such as food price crisis can inhibit the development of the children's human capabilities and hence their life outcomes.

2.2 The Data

The data for this research comes from Demographic and Health Surveys (DHS) and Living Standard and Measurement Surveys (LSMS). Both DHS and LSMS surveys have large nationally representative samples and include for every household in the survey the height of every child under the age of five. Since the height data is limited to children under five only surveys taken with-in five years of the crisis are included. That a country had both a Living Standard Measurement Survey and

Demographic and Health Survey taken within five years after the 2007-2008 food price crisis was the criteria used to determine inclusion of the country in study. This criteria was driven by the need to separate out the effect of the food price shock on a birth cohort of children from the effect of their age at the time of the survey. This requirement is necessary as age-at-the time of survey can be correlated with a child's height-for-age Z-score, and to separate out the effects of environment in which cohort was born from this timing artifact it is necessary to have measurements at multiple ages for each cohort. In Section 2.3, I will lay out the strategy for utilizing the multiple studies for controlling this timing artifact.

From these requirements, there were five candidate countries: Ethiopia, Malawi, Uganda, Tanzania, and Nigeria. The last one, Nigeria, was excluded from the study, as a lack of information on age at the time of the survey made calculating the children's height-for-age z-score impossible. Excluding Nigeria left the study with four countries: Ethiopia, Malawi, Uganda, and Tanzania. Ethiopia and Malawi each had two Living Standard Measurement Surveys that covered the period of interest. Tanzania and Uganda had three. In each survey height was measured for children under five years of age. Key variables selected from these surveys were the height and age in months for the children, their dates of birth, whether they lived in a rural or urban area, gender, region of the country, and whether they were the first child of the mother. A wealth index was calculated for each household using principle component analysis on a variety of assets listed in the surveys. From the height and age of the children, each child's height-for-age z-score relative to the 2006 WHO growth standards survey was calculated.

Table 2.1. Food Price Data

Country	Crop	% of Calories	Data Start Date
Ethiopia	Maize	16.7	Jan 2000
	Wheat	12.6	Jan 2000
	Teff	10.9	Jan 2000
	Sorgrum	14.1	March 2001
Uganda	Maize	10	Jan 2006
	Rice	2	Jan 2006
Malawi	Rice	4	Jan 2007
	Maize	54	May 2007
Tanzania	Maize	24.3	Jan 2006
	Rice	9.1	Jan 2006
	Beans	6.1	Jan 2006

Notes: Price data from FAOstata (http://faostat3.fao.org/home/). Percent of Calories each crop represents comes from FAO reports from each country (Leete, 2013b,a; Kikafunda, 2010; Ahmed, 2011)

The means and standard deviations of these variables are listed in Tables 2.9-2.12 in the appendix.

An additional step was to calculate a staple food price index for each country using price data from FAOStats access via http://faostat3.fao.org. For each country, I weighed each crop for what data was available by the percent of calories each crop constitutes in the local diet, as reported in FAO dietary reports for each country (Leete, 2013b,a; Kikafunda, 2010; Ahmed, 2011). In Table 2.1 the crop prices that were available for each countries, and the series start date, are listed.

2.3 Research Design

This paper uses two strategies to look at the impact of food price increases on in-utero and early childhood development. The first strategy is to define periods of crisis and to examine the impact that those periods have on children at various stages of development during the crisis. The second strategy is estimate the relationship between increases in local food prices and fetal and early childhood development. This section lays out and reports the finding of the former strategies.

For the first strategy I define a set of variables for exposure to the crisis at various stages of development. Each exposure variable is the portion of months of each period that overlapped with the crisis. The developmental periods are early childhood (7 to 24 months old), infancy (0 to 6 months old), and the four trimesters prior to birth. Including the four trimester prior to birth logically implies the first trimester is prior to pregnancy. This control is included because hunger during the time could influence fertility, the resulting male to female ratio, or influence the paragenome of the future zygote. The term trimester is used to indicate months prior to birth. Since we do not have data on if the children were born prematurely, this can differ from the actual stage of the pregnancy. An alternative division of the ages after birth that divides the periods into first and second year of life also reported as a robustness test.

The complete estimating equation is :

$$HAZ_i = \sum_{t=-4}^{2} \beta_t E_t + \beta X_i + \beta S + \epsilon$$

Where E_t is the exposure to the crisis in each developmental stage. The subscript t indicates each of the four trimester prior to birth and the two periods (infancy and early childhood) after birth. The vector of controls, X, include if the household is in a rural area, if the child is first born or female, the month of birth, region of the country and the wealth quintile of the household. Additionally, a linear time control is included and the child's age at date of the survey is controlled by a series of cubic splines (S). In all regressions the standard errors are clustered at the household level.

In the case of Malawi, I also include a dummy variable for the DHS survey, as there is a known discrepancy in the HAZ reported by it and the LSMS surveys. The assumption underlying this is that after controlling for covariates, any difference in the average HAZ in the DHS survey is due to measurement error. Because there is a risk that fertility is endogenous - that the most vulnerable households might have fewer children during a crisis- the household fixed effects models are considered the preferred specification.

To correct for the differences in timing of the international and local price spikes I created a local food price proxy by predicting local crop prices with international crop prices with nine lags for each crop, and then detrending the resulting series. As with the international food price crisis is defined as being one standard deviation above the trend. An alternative definition, based on the price index being 20% above the mean, is reported in the appendix. Figure 2.2 shows the resulting definition. For Ethiopia a continuous period of crisis results from May 2008 to February 2009, and in Malawi continuous period of crisis (June 2008 to March 2009) follow lone months of high prices in December 2007 and March 2008. Uganda has two periods of crisis

(April 2008 to July 2008 and April 2009 to November 2009) with two additional months of high prices in-between. Tanzania, which as previously noted, there are essentially no price spike, has 9 months labeled as crisis, but never more then two months in row. I repeated the estimation strategy of examinining exposure to a crisis utilizing this definition of crisis.

Lastly, to estimate the broader effect of food price rises regardless of if it constitutes a crisis, I utilizied a similar equation as above, but with the exposure variables replaced with the log of the average of the local food prices, P_t^L, for each period of a child's development, indicated with the subscript t.

$$HAZ_i = \sum_{t=-4}^{2} \beta_t log(P_t^L) + \beta X_i + \epsilon_2$$

As before, the vector of controls, X, include if the household is in a rural area, if the child is first born or female, the month of birth, the region of the country, and the wealth quintile of the household. Additionally, a linear time control is included, and the child's age at the time of the survey is controlled by a series of cubic splines (S). As in the previous regressions, in the case of Malawi, I also included a dummy variable for the DHS surveys as there is a known discrepancy in the HAZ reported from that survey and the DHS survey. The assumption underlying this is that after controlling for covariates, any difference in average HAZ in the DHS survey is due to a measurement error. In all regressions, the standard errors are clustered at the household level.

A key difficulty for estimating the effect of a shock on children is separating out the effect of age on height-for-age Z-scores. While the WHO growth standards

are labeled as being a measure of height for the child's age, they are intended as a measure of development that would occur in a nutritionally and environmentally healthy setting that does not hinder the child's development. For children in an environment that will stunt growth it is common to find that children in nutritionally or environmentally limiting situations appear close to mean height for age levels at birth, rapid decay in Z-score over first 18-20 months, and then slowly start to recover. Using the WHO standard without appropriately controlling for this effect would find a spurious correlation with stunted growth during a period of 18-20 months before the survey. Later in this section, I will lay out the strategy for utilizing the multiple studies for controlling this timing artifact.

This timing artifact is highlighted in Figure 2.3 in the appendix, which shows the average height-for-age Z-score for the children in the four countries by the age in months. The children show very little stunting for several months and then deteriorate rapidly from months 3-18 before then stabilizing and slowly recovering. As Figure 2.3 shows, during the months from 20-60, there are occasional small drops in Z-scores, roughly at ages where children born during the food price spikes were at times of various surveys. Accurate estimation requires separating out this potential signal from the noise of the broader deterioration and recovery of height, which are standard in these conditions.

To prevent a timing artifact from this relationship between height and age from creating an omitted variable bias in a regression, Cummin (2013) recommends using a cubic spline specification. A cubic spline model splits the range of a variable with knots between the splines. Between adjacent knots, the function is allowed to take on

the form of a cubic polynomial, yet at the knots, the function is linear with smooth first and second derivatives.

To create this functional form, a series of covariates are defined as a function of the variable x and the locations of the knots(k).

The first covariate is simply $x_1 = x$. Subsequent covariates are defined as

$$x_j = (x - t_{j-1})_+^3 - \frac{(x - t_{k-1})_+^3 (t_k - t_{j-1})}{(t_k - t_{k-1})} + \frac{(x - t_k)_+^3 (t_{k-1} - t_{j-1})}{(t_k - t_{k-1})}$$

for $j = 2, ..., k-1$ where

$$u+ = \begin{cases} 0 \text{ if } u < 0 \\ u \text{ if } u > 0 \end{cases}$$

These covariates can then be used in linear regression:

$$y = a + x_1 b_1 + x_2 b_2 + ... + x_{k-1} b_{k-1}$$

To control the height-age relationship, I create splines with knots at 0, 3, 12 and 24 months. Figure 2.3 shows the resulting predicted Z-scores from this model, along with a local polynomial of the actual height-for-age Z-score by age in months.

2.4 Empirical Findings

2.4.1 Effects of Crisis Exposure

Figure 2.1 shows the international price trend of the staple crops of rice, wheat and maize combined into a single index. Defining a crisis as a time where the price level is one standard deviation above the trend, there is distinct crisis existing from

March to August 2008. Figure 2.1 also shows a local polynomial height-for-age Z-score - after partialling out the restricted cubic spline as explained later in this section - by the month the children were born for children from all four countries. From the graph we see that the modified height-for-age Z-score decreasing as the birth date approaches the crisis - with the lowest heights for those born during the crisis. More distinct is the immediate improvement in height-for-age for those born after the crisis.

Table 2.4 reports the findings for each country, with and without fixed effects, and for all countries pooled together. When the countries are pooled the effects in both with and without household fixed effects are significant in the first and third trimesters, and in early childhood and infancy. An optimistic take is that the coefficients on these estimates are small. Focusing on the household fixed effects model, going from no exposure to complete exposure in early childhood decreases height-for-age by just .09 standard deviations, .15 deviations during infancy, .23 standard deviations for exposure in the third trimester, and .27 standard deviations in the first trimester. In comparison children born during periods of seasonal hunger in Tanzania are, in general, a third of a standard deviation shorter than their peers.

There is less consistency in the individual country regressions. In Ethiopia we see a negative relationship between exposure and stature in the developmental periods following birth, and the third and second trimesters, both with and without household fixed effects. In Malawi, we see small negative effects in the model without fixed effects in the post birth periods, and in the first trimester. With household fixed effects, the effects of infancy and early childhood become insignificant, but a

statistically significant effect can be observed in the third and trimester and trimester before pregnancy. As I discuss below, there was relatively little pass through of the international price increases to Uganda and Tanzania. As a result, we find fewer statistically significant impact of exposure for these two countries, except of a positive relationship for infancy for Uganda with fixed effects, and for second trimester for Tanzania with household fixed effects.

A possible explanation of the limited impact of the international price crisis on children's development is a result of delays or limitations in the transmission of the international prices to the local context. In Figure 2.4 I show each country's price trend over a local polynomial of that country's children's modified height-for-age Z-scores. I retain from Figure 2.1 the lines indicating the timing of the international food price crisis and each countries regression discontinuity lines. For Ethiopia Malawi, and Uganda we observe a local food price spike but with a delay between the timing of the international food price spike and the local food price spike. In Uganda, a second and more substantial food price spike occurs a few months later. In Tanzania there are essentially no spikes in prices and little deviation from the longer trend.

To correct for the differences in timing of the international and local price spikes, I created a local food price proxy by predicting local crop prices with international crop prices with nine lags for each crop and then detrending the resulting series. As with the international food price crisis is defined as being one standard deviation above the trend. An alternative definition, based on the price index being 20% above the mean, is reported in the appendix. Figure 2.2 shows the resulting definition. For

Ethiopia, a continuous period of crisis results from May 2008 to February 2009, and in Malawi continuous period of crisis (June 2008 to March 2009) follow lone months of high prices in December 2007 and March 2008. Uganda has two periods of crisis (April 2008 to July 2008 and April 2009 to November 2009) with two additional months of high prices in-between. Tanzania, where as previously noted, there is essentially no price spike, has 9 months labeled as a crisis, but never more than two months in row.

I repeated the estimation of exposure to the food prices crisis but now with using this definition of the local food price crisis. The results are reported in Table 2.3. When the countries are pooled a consistent effect appears with negative effects for exposure during early childhood, infancy, and third-trimester in-utero. Except the household fixed effects model estimate on exposure in infancy, which is significant at the $p < .01$ level, these estimates are all significant at the $p < .001$ level across both the fixed effects and non-fixed effects models.

None of the other exposure coefficients are significant in the pooled regressions. Most notably, the findings from the international price crisis regression that there was a negative effect of the first trimester exposure has disappeared despite the standard errors being smaller. The result in the international food price crisis regression was likely a result of the delay in the transmission of the food price crisis, particularly in Malawi. In the local food price regression Malawi instead now shows a weakly significant positive relationship with the exposure to the food price crisis. For both Ethiopia and Malawi, the two countries with the most distinct crisis, exposure during the third trimester is consistently significant. In both countries the household fixed

effects model is showing a substantially larger effect from the third trimester effects than the second, jumping from -.37 to -.69 in Ethiopia and from -.45 to -.82 for Malawi. Ethiopia also shows an effect for early childhood and infancy, but this effect does not appear for Malawi or any of the other countries.

Table 2.4 reports robustness regressions for local food price crisis model with all the countries pooled. Columns 1 and 2 repeat the base model discussed above, with and without fixed effects. Using quadratic time controls (columns 3 and 4) or no time controls (columns 5 and 6) does not substantially change the results. Using an alternative definition of crisis (columns 7 and 8) of where the detrended prices rise 20% above the mean produces a small, weakly significant adverse effect of first-trimester exposure. In the fixed effects model of the alternative definition exposure in infancy is no longer statistically significant. Besides these differences, the results of the alternative crisis definition are similar to the base model. Lastly, an alternative breakdown of child ages is provided in columns 9 and 10, with exposure to first or second year of life. Full tables with individual country results for these robustness models and with breakdowns by gender and rural or urban are provided in the appendix.

2.4.2 Effects of Food Price Rises

The second strategy is to examine the impact of food prices on children's height directly, regardless of if the price constitutes a crisis or not. In these models, the exposure variables are replaced with the log of local prices for each period of a child's life. The pooled models show negative effects for early childhood and the third trimester with and without fixed effects. In the model without household fixed

Figure 2.1. International Prices and Modified Height for Age

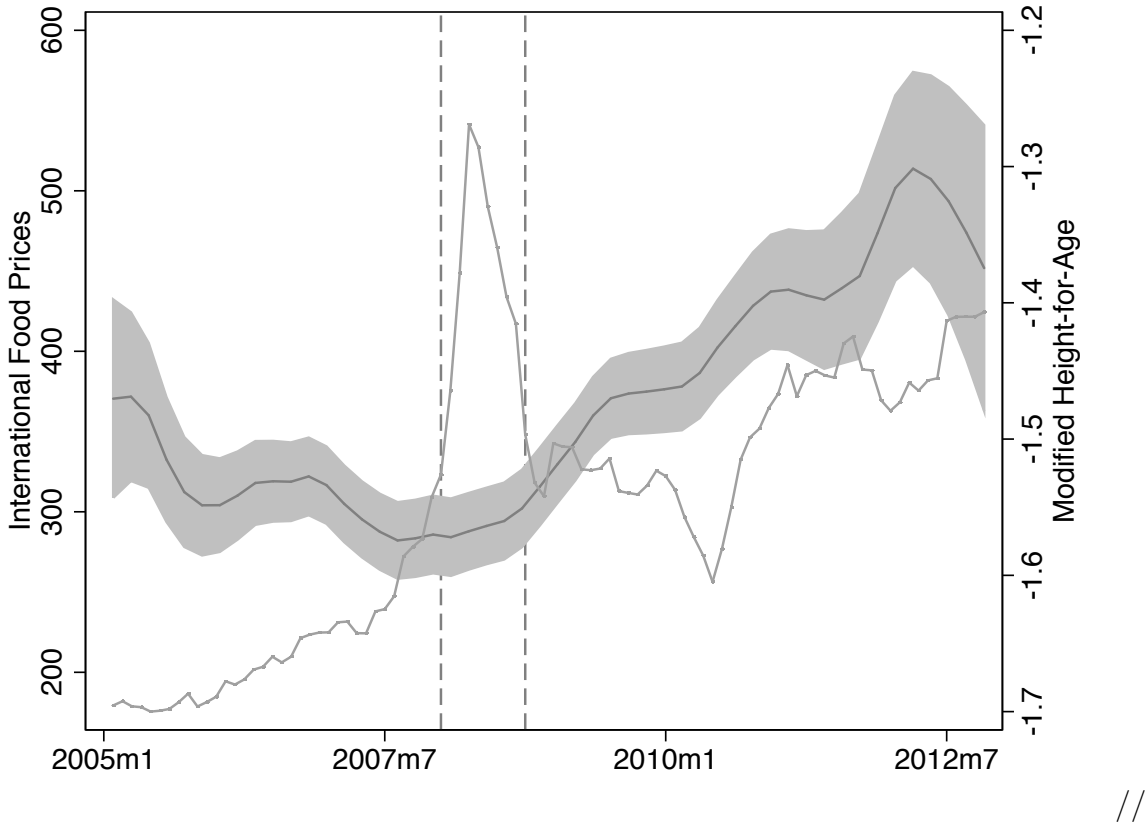


Figure 2.1 notes: Black line shows international prices, dashed vertical lines indicate the months of the international food price crisis. Gray lines are local polynomial of Height-for-age by month of birth, after cubic spline of age in months have been partialled out.

Table 2.2. Effects of Exposure to International Food Price Crisis

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2) FE	(3)	(4) FE	(5)	(6) FE	(7)	(8) FE	(9)	(10) FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Exposure to Crisis During:										
-Early Childhood (7-24mo)	-0.11***	-0.09**	-0.47***	-0.37**	-0.09*	-0.05	0.14	0.19	-0.10	-0.17
	(0.02)	(0.04)	(0.09)	(0.11)	(0.04)	(0.07)	(0.13)	(0.13)	(0.07)	(0.11)
-Infancy (0-6mo)	-0.15***	-0.15**	-0.43***	-0.38***	-0.13*	-0.11	0.26*	0.12	-0.11	-0.21
	(0.04)	(0.05)	(0.08)	(0.11)	(0.06)	(0.12)	(0.12)	(0.14)	(0.13)	(0.20)
-Third Trimester	-0.19**	-0.23*	-0.32**	-0.40*	-0.14	-0.48*	-0.14	0.13	-0.25	-0.35
	(0.06)	(0.10)	(0.11)	(0.19)	(0.10)	(0.19)	(0.16)	(0.21)	(0.17)	(0.27)
-Second Trimester	-0.12	-0.08	-0.68***	-0.66**	-0.02	0.34	0.21	0.08	0.49*	0.42
	(0.08)	(0.11)	(0.14)	(0.22)	(0.13)	(0.20)	(0.20)	(0.27)	(0.20)	(0.30)
-First Trimester	-0.19*	-0.27*	-0.06	-0.09	-0.27*	-0.65***	-0.23	-0.21	-0.16	-0.35
	(0.08)	(0.12)	(0.14)	(0.22)	(0.13)	(0.19)	(0.19)	(0.27)	(0.20)	(0.33)
-Trimester Before Conception	0.01	0.05	-0.02	-0.09	0.08	0.25**	-0.04	0.05	0.15	0.11
	(0.04)	(0.06)	(0.08)	(0.11)	(0.07)	(0.10)	(0.10)	(0.15)	(0.10)	(0.16)
Household Fixed Effects		Yes		Yes		Yes		Yes		Yes
Observations	48500	48500	14366	14366	14479	14479	6620	6620	13055	13055
R-Squared	0.10	0.15	0.16	0.19	0.08	0.11	0.10	0.12	0.18	0.22

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Crisis is defined as period when international prices 1 SD above detrended price mean. Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled for. Standard Errors are clustered at household level.

Figure 2.2. Local Food Prices & Crisis Months

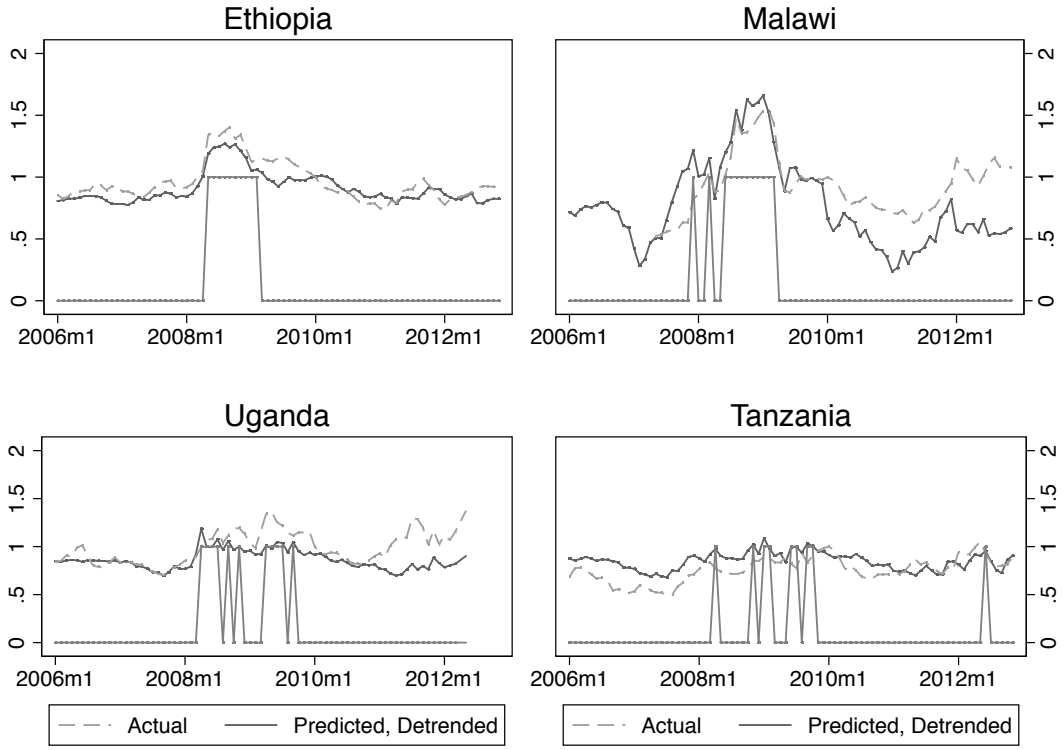


Figure 2.2 notes: Dashed line shows actual local prices relative to 2010 levels. Black line shows detrended local prices predicted from international prices. Gray line shows months where the predicted prices are 1 SD above the trend and considered a crisis.

Table 2.3. Effects of Exposure to Local Food Price Crisis

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2) FE	(3)	(4) FE	(5)	(6) FE	(7)	(8) FE	(9)	(10) FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Exposure to Crisis During:										
-Early Childhood (7-24mo)	-0.15***	-0.17***	-0.18**	-0.32**	-0.08	0.01	0.15	0.23	-0.24	-0.13
	(0.03)	(0.05)	(0.07)	(0.10)	(0.05)	(0.09)	(0.13)	(0.13)	(0.14)	(0.33)
-Infancy (0-6mo)	-0.19***	-0.21**	-0.17*	-0.30*	-0.02	0.09	0.22	0.07	-0.17	-0.07
	(0.05)	(0.07)	(0.09)	(0.14)	(0.11)	(0.20)	(0.14)	(0.16)	(0.21)	(0.41)
-Third Trimester	-0.25***	-0.28***	-0.37*	-0.69**	-0.45*	-0.82**	-0.16	0.25	-0.14	-0.02
	(0.06)	(0.08)	(0.17)	(0.27)	(0.18)	(0.30)	(0.18)	(0.23)	(0.15)	(0.33)
-Second Trimester	-0.01	-0.04	0.09	0.11	-0.02	0.15	0.21	-0.20	0.01	0.25
	(0.06)	(0.08)	(0.20)	(0.31)	(0.20)	(0.33)	(0.18)	(0.23)	(0.15)	(0.34)
-First Trimester	-0.04	-0.06	-0.04	-0.41*	0.38*	0.53*	-0.31	0.15	-0.11	0.02
	(0.05)	(0.08)	(0.14)	(0.21)	(0.15)	(0.25)	(0.17)	(0.23)	(0.15)	(0.34)
-Trimester Before Conception	-0.01	-0.02	0.23***	0.09	-0.08	0.07	0.20	0.10	-0.23	-0.25
	(0.04)	(0.06)	(0.07)	(0.10)	(0.08)	(0.12)	(0.10)	(0.13)	(0.15)	(0.34)
Household Fixed Effects		Yes		Yes		Yes		Yes		Yes
Observations	48500	48500	14366	14366	14479	14479	6620	6620	13055	13055
R-Squared	0.10	0.15	0.16	0.19	0.08	0.11	0.10	0.12	0.18	0.23

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Crisis is defined as period when local prices 1 SD above detrended price mean.Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled for. . Standard Errors are clustered at household level.

Table 2.4. Robustness Regressions for the Effects of Exposure to Local Food Price Crisis

	Base		Quadratic		No Trend		Alt. Crisis Definition		Alternative Periodization	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
-Second Year									-0.10**	-0.14**
									(0.03)	(0.05)
-First Year									-0.22***	-0.23***
									(0.04)	(0.06)
-Early Childhood (7-24mo)	-0.15***	-0.17***	-0.13***	-0.21***	-0.20***	-0.10*	-0.14***	-0.21***		
	(0.03)	(0.05)	(0.04)	(0.05)	(0.03)	(0.05)	(0.02)	(0.03)		
-Infancy (0-6mo)	-0.19***	-0.21**	-0.16**	-0.30***	-0.23***	-0.18*	-0.13**	-0.10		
	(0.05)	(0.07)	(0.06)	(0.09)	(0.05)	(0.07)	(0.04)	(0.06)		
-Third Trimester	-0.25***	-0.28***	-0.23***	-0.32***	-0.29***	-0.23**	-0.18***	-0.30***	-0.23***	-0.26**
	(0.06)	(0.08)	(0.06)	(0.09)	(0.06)	(0.08)	(0.05)	(0.08)	(0.06)	(0.08)
-Second Trimester	-0.01	-0.04	0.01	-0.08	-0.04	0.00	-0.07	-0.08	0.00	-0.01
	(0.06)	(0.08)	(0.06)	(0.09)	(0.06)	(0.08)	(0.06)	(0.08)	(0.06)	(0.09)
-First Trimester	-0.04	-0.06	-0.02	-0.12	-0.07	-0.03	-0.12*	-0.20*	-0.03	-0.05
	(0.05)	(0.08)	(0.06)	(0.09)	(0.05)	(0.08)	(0.06)	(0.08)	(0.06)	(0.08)
-Trimester Before Conception	-0.01	-0.02	0.01	-0.07	-0.03	-0.00	-0.04	-0.13*	-0.01	-0.02
	(0.04)	(0.06)	(0.04)	(0.06)	(0.04)	(0.06)	(0.04)	(0.06)	(0.04)	(0.06)
Observations	48500	48500	48500	48500	48500	48500	48500	48500	48500	48500
R-Squared	0.10	0.15	0.10	0.15	0.10	0.15	0.10	0.15	0.11	0.15
Household Fixed Effects		Yes		Yes		Yes		Yes		Yes
Linear Trend	Yes	Yes	Yes	Yes			Yes	Yes	Yes	Yes
Quadratic Time Control			Yes	Yes						
Alternative Crisis Definition							Yes	Yes		
Alternative Age Periodization									Yes	Yes

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. Standard Errors are clustered at household level. Columns 1 & 2 are the base model, columns 3 & 4 include quadratic time control, columns 5 & 6 have no time trend. Columns 7 & 8 use the alternative definition of crisis based on prices rising 20%. Columns 9 & 10 use alternative periodization of early childhood, breaking early childhood into first and second year.

effects, there is also a small negative effect on the quarter before pregnancy and a positive effect on prices during the first trimester.

One counter-intuitive result stands out. Local prices during infancy – 0 to 6 months old – have a substantial and statistically significant positive impact on the stature of children. This result is both counter-intuitive and contradicts the results of the crisis models. Robustness checks for alternative periodization are included in Table 2.8. Columns 7 and 8 report breaking ages into the first and second year. The first year coefficient in the model without household fixed effect is smaller the infant (0-6 months old) coefficient in the base model, and it is statistically insignificant in household fixed effects model. A third possible periodization is reported in columns 9 and 10, with age divided into infant (0-6 months old), toddler (7-12 months old), and second year of life. The infant coefficient is still positive and strongly statistically significant both with and without household fixed effects. The toddler coefficients are negative but statistically insignificant. A possible hypothesis for further study is if this outcome is the result of childcare practices responding to prices. Some studies have found the duration of exclusive breastfeeding has been shown to be endogenous to the conditions at the time and the health of the child (Simondon and Simondon, 1998). In return, the duration of exclusive breastfeeding has been found– after controlling for conditions and health at birth– to be positively correlated with a child's stature (Simondon et al., 2001; Onyango et al., 1999).

Although the results are not precise in the separate country regressions, we can note a consistent negative relationship, with the sole exception of Uganda in the model without the household fixed effects. In the pooled household fixed effects

model, the result is a highly significant (p <.001) estimate of -1.20. This result implies that a 30% increase in prices would decrease a child's z-score by .315 standard deviations. This estimate is similar to what is seen in Tanzania during the hunger season both in terms of increase in price and decrease in height-for-age Z-scores. During the Ethiopian food price crisis, local prices were roughly 50% higher than before the crisis. According to this estimate, this should cause a decrease in stature of .48 standard deviations. This result is not quite as large as our finding of a .69 standard deviation effect of being born during the crisis in the household fixed effects model of the local price crisis (Table 2.3). In Malawi the prices roughly doubled during the crisis. According to our estimate of Z-scores decreasing 1.2 times a log in prices that a doubling in prices would be expected to reduce the height by .83 standard deviations. This result is almost identical to the -.82 found for Malawis household fixed effects model in Table 2.3.

Looking at the regressions decomposed by gender (Table 2.6), we see that females appear to have less sensitivity to effects of price movement than males if third trimester and after birth, however, these difference are small and statistically insignificant. The female pooled regressions we see statistically significant adverse effect of prices before pregnancy then a positive effect from the first trimester. Existing literature has found that stress before conception increases the probability of a female child (Grant and Chamley, 2010). For example, the ratio of males to females dropped in the United States 40 weeks after the September 11 attacks (Catalano et al., 2005). To test this explanation, I ran a linear probability model of the likelihood of a child being female compared with prices during and before pregnancy, with controls for the

region, rural, wealth, and birth month. The only statistically significant predictor was the prices for the quarter before pregnancy, which indicated that a 30% increase in prices during the trimester would cause a 1.3% increase in the number of females. Looking at the country specific models of the effect of prices on height (Table 2.6) we see only an effect for Uganda males. This effect is positive and very large. Looking at the country specific linear probability model of a child being female, we see that only Uganda has a statistically significant effect from prices before pregnancy. The coefficient implies that a 30% increase in prices increases the probability of a child being female 6.4 percentage points. Future research will be necessary to examine why this effect is found in Uganda and not in other countries in the sample.

Table 2.5. Effect of (Log) Local Prices on Height

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2) FE	(3)	(4) FE	(5)	(6) FE	(7)	(8) FE	(9)	(10) FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Log of Proxy Prices for:										
-Early Childhood (7-24mo)	-0.73***	-1.17***	-0.49	-1.04	-0.52	-0.52	2.96	2.69	0.03	-0.57
	(0.17)	(0.26)	(0.35)	(0.54)	(1.02)	(2.24)	(1.89)	(2.84)	(0.76)	(1.60)
-Infancy (0-6mo)	0.75***	0.84***	0.29	0.43	0.64	0.57	-0.73	-0.61	0.22	0.11
	(0.14)	(0.23)	(0.29)	(0.45)	(0.47)	(0.92)	(0.71)	(1.04)	(0.44)	(0.90)
-Third Trimester	-0.88***	-1.20***	-0.66	-0.82	-0.09	-1.92*	0.02	-0.61	-0.37	-0.10
	(0.15)	(0.26)	(0.38)	(0.58)	(0.42)	(0.77)	(0.39)	(0.60)	(0.28)	(0.49)
-Second Trimester	0.19	0.50	0.18	-0.15	-0.07	1.39	-0.27	0.50	0.13	0.21
	(0.18)	(0.31)	(0.39)	(0.64)	(0.44)	(0.93)	(0.42)	(0.60)	(0.29)	(0.57)
-First Trimester	0.35	0.06	0.67	1.33*	-0.00	-0.23	-0.15	-0.36	-0.08	-0.29
	(0.18)	(0.32)	(0.40)	(0.64)	(0.43)	(0.94)	(0.44)	(0.62)	(0.28)	(0.53)
-Trimester Before Pregnancy	-0.23	-0.30	0.13	-0.66	0.29	-0.43	0.82*	0.62	0.00	-0.07
	(0.12)	(0.21)	(0.28)	(0.44)	(0.39)	(0.78)	(0.37)	(0.51)	(0.22)	(0.42)
Observations	37937	37937	13893	13893	7489	7489	5899	5899	10656	10656
Adjusted R^2	0.137	0.187	0.168	0.217	0.069	0.097	0.110	0.141	0.182	0.247

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled forStandard Errors are clustered at household level.

Table 2.6. Effect of (Log) Local Prices on Height by Gender

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1) Male b/se	(2) Female b/se	(3) Male b/se	(4) Female b/se	(5) Male b/se	(6) Female b/se	(7) Male b/se	(8) Female b/se	(9) Male b/se	(10) Female b/se
Log of Proxy Prices for:										
-Early Childhood (7-24mo)	-1.01***	-0.42	0.08	-1.06*	-2.20	1.14	3.96	2.48	-0.53	0.36
	(0.24)	(0.25)	(0.48)	(0.51)	(1.43)	(1.44)	(2.70)	(2.52)	(1.10)	(1.03)
-Infancy (0-6mo)	0.92***	0.59**	0.15	0.45	0.79	0.45	-0.74	-0.86	0.83	-0.15
	(0.21)	(0.19)	(0.41)	(0.41)	(0.68)	(0.65)	(1.03)	(0.95)	(0.65)	(0.60)
-Third Trimester	-1.01***	-0.79***	-0.56	-0.76	0.27	-0.37	0.13	-0.09	-0.47	-0.39
	(0.22)	(0.21)	(0.53)	(0.54)	(0.59)	(0.59)	(0.58)	(0.52)	(0.41)	(0.37)
-Second Trimester	0.30	0.08	0.06	0.25	0.48	-0.60	-0.47	-0.08	0.29	-0.07
	(0.26)	(0.24)	(0.55)	(0.55)	(0.61)	(0.62)	(0.64)	(0.56)	(0.42)	(0.40)
-First Trimester	0.19	0.54*	0.59	0.81	-0.92	0.73	-0.81	0.67	0.03	-0.14
	(0.26)	(0.24)	(0.57)	(0.56)	(0.63)	(0.59)	(0.68)	(0.58)	(0.39)	(0.39)
-Trimester Before Pregnancy	-0.12	-0.36*	0.36	-0.15	0.38	0.35	2.01***	-0.41	-0.16	0.10
	(0.17)	(0.16)	(0.40)	(0.40)	(0.56)	(0.54)	(0.57)	(0.50)	(0.31)	(0.30)
Observations	19095	18842	7099	6794	3757	3732	2960	2939	5279	5377
Adjusted R^2	0.130	0.144	0.165	0.172	0.068	0.063	0.114	0.103	0.155	0.194

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled forStandard Errors are clustered at household level.

2.5 Conclusion

A key finding of this study is the difference in outcome for Ethiopia and Malawi, which had substantial domestic food price spikes, and Uganda and Tanzania, which had more modest price increases. Both Ethiopia and Malawi saw a decrease in the height-for-age Z-scores of children that were in the final two trimesters of pregnancy or under two years of age. Neither Uganda or Tanzania had statistically significant decrease in height in any of the periods of development. This story is strengthened when we looked at the period of high prices domestically. For those in third-trimester in-utero during the local crisis children's height-for-age Z-score compared to their siblings dropped .69 standard deviations in Ethiopia and .82 standard deviations for Malawi. Again we failed to find a statistically significant effect in Uganda or

Table 2.7. Effect of (Log) Local Prices on Height by Location

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1) Rural b/se	(2) Urban b/se	(3) Rural b/se	(4) Urban b/se	(5) Rural b/se	(6) Urban b/se	(7) Rural b/se	(8) Urban b/se	(9) Rural b/se	(10) Urban b/se
Log of Proxy Prices for:										
-Early Childhood (7-24mo)	-0.56**	-1.27**	-0.63	0.09	-0.32	-1.41	4.55*	-3.84	0.36	-1.73
	(0.19)	(0.40)	(0.39)	(0.78)	(1.13)	(2.44)	(2.12)	(3.93)	(0.84)	(1.74)
-Infancy (0-6mo)	0.70***	0.99**	0.24	0.82	0.56	1.04	-1.39	2.28	0.29	0.09
	(0.16)	(0.33)	(0.32)	(0.64)	(0.52)	(1.13)	(0.80)	(1.50)	(0.49)	(1.00)
-Third Trimester	-0.86***	-1.08**	-0.32	-2.71**	-0.13	0.08	0.10	-0.46	-0.31	-0.56
	(0.17)	(0.35)	(0.42)	(0.83)	(0.47)	(1.02)	(0.43)	(0.86)	(0.31)	(0.58)
-Second Trimester	0.15	0.36	-0.13	1.83*	0.01	-0.62	-0.02	-1.13	-0.06	0.68
	(0.20)	(0.41)	(0.43)	(0.89)	(0.49)	(1.02)	(0.46)	(1.01)	(0.32)	(0.64)
-First Trimester	0.39*	0.17	0.67	0.72	-0.02	0.11	-0.36	0.57	0.08	-0.57
	(0.20)	(0.43)	(0.44)	(0.96)	(0.46)	(1.13)	(0.48)	(1.04)	(0.31)	(0.62)
-Trimester Before Pregnancy	-0.23	-0.20	0.24	-0.36	0.32	0.24	1.10**	-0.10	0.03	-0.21
	(0.13)	(0.28)	(0.32)	(0.64)	(0.43)	(0.95)	(0.41)	(0.89)	(0.24)	(0.49)
Observations	31232	6705	11743	2150	6299	1190	4837	1062	8353	2303
Adjusted R^2	0.132	0.129	0.155	0.184	0.072	0.050	0.107	0.074	0.172	0.179

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled forStandard Errors are clustered at household level.

Table 2.8. Robustness Regressions for the Effects of Local Food Price Rises

	Base		Quadratic		No Trend		Alt. Periodization		Alt. Periodization 2	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Log of Proxy Prices for:										
-Early Childhood (7-24mo)	-0.73***	-1.17***	-0.72***	-0.99***	-0.52**	-1.09***				
	(0.17)	(0.26)	(0.18)	(0.27)	(0.17)	(0.26)				
-Second year							-0.68***	-0.91***	-0.61***	-0.72**
							(0.15)	(0.24)	(0.15)	(0.25)
–Toddler (7mo-12mo)									-0.05	-0.49
									(0.17)	(0.28)
-First Year							0.57***	0.38		
							(0.14)	(0.24)		
-Infancy (0-6mo)	0.75***	0.84***	0.73***	0.60*	0.85***	0.77***			0.64***	0.90***
	(0.14)	(0.23)	(0.15)	(0.26)	(0.14)	(0.23)			(0.17)	(0.27)
-Third Trimester	-0.88***	-1.20***	-0.88***	-1.15***	-1.01***	-1.17***	-0.73***	-0.97***	-0.86***	-1.21***
	(0.15)	(0.26)	(0.15)	(0.26)	(0.15)	(0.26)	(0.14)	(0.25)	(0.15)	(0.26)
-Second Trimester	0.19	0.50	0.19	0.49	0.26	0.49	0.20	0.54	0.20	0.50
	(0.18)	(0.31)	(0.18)	(0.31)	(0.18)	(0.31)	(0.18)	(0.31)	(0.18)	(0.31)
-First Trimester	0.35	0.06	0.35	0.04	0.23	0.08	0.23	-0.06	0.31	0.08
	(0.18)	(0.32)	(0.18)	(0.32)	(0.18)	(0.32)	(0.18)	(0.32)	(0.18)	(0.32)
-Trimester Before Pregnancy	-0.23	-0.30	-0.24*	-0.37	0.04	-0.33	-0.15	-0.22	-0.20	-0.31
	(0.12)	(0.21)	(0.12)	(0.21)	(0.12)	(0.21)	(0.12)	(0.21)	(0.12)	(0.21)
Observations	37937	37937	37937	37937	37937	37937	37937	37937	37937	37937.00
R-Squared	0.14	0.19	0.14	0.19	0.14	0.19	0.14	0.19	0.14	0.19
Household Fixed Effects		Yes		Yes		Yes		Yes		Yes
Linear Trend	Yes	Yes	Yes	Yes			Yes	Yes	Yes	Yes
Quadratic Time Control			Yes	Yes						
Alternative Age Periodization							Yes	Yes		

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. Standard Errors are clustered at household level. Columns 1 & 2 are the base model, columns 3 & 4 include quadratic time control, columns 5 & 6 have no time trend. Columns 7 & 8 use alternative periodization of early childhood, breaking early childhood into first and second year. Columns 9 & 10 use another alternative periodization that breaks ages into first six months, from 6mo-12mo, and second year of life.

Tanzania. These results give hope that if countries can prevent international food price spike from rising local food prices, they can prevent harm to their population.

Looking at the effect of food prices on children's development reinforced the finding that the third trimester seems to be sensitive period in-utero. I found that a log increase in prices decreased the height by 1.2 standard deviations. This result implies that a 30% increase in prices would reduce the stature of a child that is in the third trimester by .315 standard deviations. Urban areas the are less likely to have land to produce their own food were especially sensitive to price changes.

Two additional findings call for future research. First, an unexpected finding was that infants zero to six months old had a positive response in their development to food prices. Exisiting literature finding that breastfeeding duractions responds to child health and the duration positively impact catch up growth could provide a possible explanation, but confirmation requires additional research. Second, consistent with there was evidence that stress prior to pregnancy reduces conception of male children, I find that in Uganda prices before pregnancy increase the probability of child being female. Examining why this effect is only found in Uganda is left for future research.

2.6 Appendix

Table 2.9. Summary Statistics for Ethiopia

	LSMS		DHS	
	2011	2013	2011	Total
Height-For-Age Zscore	-1.69	-1.48	-1.61	-1.60
	(1.94)	(1.84)	(1.76)	(1.80)
Rural	0.93	0.78	0.84	0.84
	(0.26)	(0.41)	(0.37)	(0.36)
First Born	0.70	0.35	0.19	0.30
	(0.46)	(0.48)	(0.39)	(0.46)
Female	0.48	0.49	0.49	0.49
	(0.50)	(0.50)	(0.50)	(0.50)
Age in Months	32.41	33.21	29.41	30.54
	(15.10)	(15.70)	(17.26)	(16.75)
Year of Birth:				
2006	0.00	0.00	0.16	0.11
	(0.07)	(0.02)	(0.37)	(0.31)
2007	0.20	0.00	0.21	0.17
	(0.40)	(0.05)	(0.41)	(0.38)
2008	0.23	0.01	0.20	0.17
	(0.42)	(0.10)	(0.40)	(0.38)
2009	0.23	0.19	0.19	0.39)
2010	0.22	0.22	0.21	0.21
	(0.41)	(0.41)	(0.41)	(0.41)
2011	0.12	0.22	0.04	0.08
	(0.32)	(0.42)	(0.18)	(0.27)
2012	0.00	0.21	0.00	0.04
	(0.04)	(0.41)	(0.00)	(0.19)
2013	0.00	0.15	0.00	0.03
	(0.00)	(0.35)	(0.00)	(0.16)
Wealth Index				
Poorest	0.16	0.23	0.31	0.27
	(0.37)	(0.42)	(0.46)	(0.44)
Poorer	0.21	0.24	0.18	0.20
	(0.40)	(0.43)	(0.39)	(0.40)
Middle	0.23	0.23	0.17	0.19
	(0.42)	(0.42)	(0.37)	(0.39)
Richer	0.22	0.16	0.16	0.17
	(0.42)	(0.36)	(0.37)	(0.38)
Richest	0.18	0.14	0.18	0.17
	(0.38)	(0.35)	(0.38)	(0.38)
Observations	2209	2545	9611	14365

Table 2.10. Summary Statistics for Malawi

	LSMS		DHS	
	2011	2013	2011	Total
Height-For-Age Zscore	-1.34	-1.12	-1.78	-1.44
	(1.64)	(1.60)	(1.58)	(1.63)
Rural	0.84	0.78	0.90	0.85
	(0.36)	(0.41)	(0.30)	(0.36)
First Born	0.79	0.81	0.18	0.60
	(0.40)	(0.40)	(0.39)	(0.49)
Female	0.50	0.50	0.50	0.50
	(0.50)	(0.50)	(0.50)	(0.50)
Age in Months	32.67	32.03	29.38	31.52
	(15.06)	(15.89)	(16.70)	(15.80)
Year of Birth:				
2006	0.22	0.00	0.19	0.18
	(0.42)	(0.02)	(0.39)	(0.38)
2007	0.24	0.00	0.20	0.19
	(0.43)	(0.00)	(0.40)	(0.39)
2008	0.24	0.09	0.22	0.20
	(0.42)	(0.28)	(0.41)	(0.40)
2009	0.25	0.22	0.29	0.26
	(0.43)	(0.41)	(0.45)	(0.44)
2010	0.06	0.21	0.10	0.09
	(0.23)	(0.41)	(0.30)	(0.29)
2011	0.00	0.23	0.00	0.04
	(0.01)	(0.42)	(0.00)	(0.19)
2012	0.00	0.22	0.00	0.04
	(0.00)	(0.41)	(0.00)	(0.19)
2013	0.00	0.04	0.00	0.01
	(0.00)	(0.19)	(0.00)	(0.08)
Wealth Index				
Poorest	0.20	0.22	0.20	0.20
	(0.40)	(0.41)	(0.40)	(0.40)
Poorer	0.21	0.23	0.23	0.22
	(0.41)	(0.42)	(0.42)	(0.41)
Middle	0.21	0.20	0.23	0.22
	(0.41)	(0.40)	(0.42)	(0.41)
Richer	0.21	0.20	0.20	0.20
	(0.41)	(0.40)	(0.40)	(0.40)
Richest	0.17	0.14	0.15	0.16
	(0.37)	(0.35)	(0.35)	(0.36)
Observations	7499	2396	4586	14481

Table 2.11. Summary Statistics for Uganda

	LSMS			DHS	
	2009	2010	2011	2011	Total
Height-For-Age Zscore	-1.39	-1.41	-1.39	-1.38	-1.39
	(1.68)	(1.51)	(1.47)	(1.54)	(1.55)
Rural	0.82	0.84	0.85	0.79	0.82
	(0.39)	(0.37)	(0.36)	(0.41)	(0.38)
First Born	0.60	0.67	0.66	0.17	0.50
	(0.49)	(0.47)	(0.47)	(0.38)	(0.50)
Female	0.48	0.50	0.51	0.50	0.50
	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)
Age in Months	26.98	32.35	31.77	27.88	29.55
	(12.72)	(14.80)	(15.20)	(16.98)	(15.33)
Year of Birth:					
2006	0.25	0.16	0.00	0.04	0.11
	(0.43)	(0.36)	(0.06)	(0.21)	(0.31)
2007	0.28	0.23	0.13	0.19	0.21
	(0.45)	(0.42)	(0.34)	(0.39)	(0.41)
2008	0.28	0.24	0.23	0.19	0.23
	(0.45)	(0.43)	(0.42)	(0.39)	(0.42)
2009	0.18	0.21	0.21	0.21	0.20
	(0.39)	(0.41)	(0.41)	(0.40)	(0.40)
2010	0.01	0.14	0.21	0.21	0.15
	(0.08)	(0.35)	(0.40)	(0.41)	(0.35)
2011	0.00	0.01	0.20	0.16	0.10
	(0.00)	(0.11)	(0.40)	(0.37)	(0.30)
2012	0.00	0.00	0.02	0.00	0.00
	(0.00)	(0.00)	(0.13)	(0.00)	(0.06)
Wealth Index					
Poorest	0.21	0.23	0.21	0.25	0.23
	(0.41)	(0.42)	(0.41)	(0.44)	(0.42)
Poorer	0.21	0.21	0.20	0.20	0.21
	(0.41)	(0.41)	(0.40)	(0.40)	(0.41)
Middle	0.21	0.19	0.22	0.18	0.20
	(0.41)	(0.39)	(0.41)	(0.38)	(0.40)
Richer	0.21	0.19	0.19	0.17	0.19
	(0.40)	(0.39)	(0.39)	(0.37)	(0.39)
Richest	0.16	0.17	0.18	0.20	0.18
	(0.37)	(0.38)	(0.38)	(0.40)	(0.38)
Observations	1565	1496	1492	2070	6623

Table 2.12. Summary Statistics for Tanzania

	LSMS			DHS	
	2008	2010	2012	2010	Total
Height-For-Age Zscore	-1.65	-1.35	-1.36	-1.62	-1.52
	(1.46)	(1.53)	(1.58)	(1.44)	(1.50)
Rural	0.74	0.77	0.76	0.82	0.79
	(0.44)	(0.42)	(0.43)	(0.39)	(0.41)
First Born	0.31	0.24	0.33	0.19	0.24
	(0.46)	(0.43)	(0.47)	(0.39)	(0.43)
Female	0.53	0.50	0.50	0.50	0.51
	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)
Age in Months	27.46	28.98	29.36	28.28	28.55
	(14.44)	(17.50)	(17.44)	(17.23)	(17.03)
Year of Birth:					
2006	0.25	0.13	0.00	0.19	0.15
	(0.44)	(0.33)	(0.00)	(0.40)	(0.35)
2007	0.26	0.19	0.00	0.19	0.16
	(0.44)	(0.39)	(0.07)	(0.39)	(0.36)
2008	0.18	0.19	0.14	0.21	0.19
	(0.38)	(0.39)	(0.35)	(0.41)	(0.39)
2009	0.31	0.21	0.18	0.36	0.29
	(0.46)	(0.40)	(0.39)	(0.48)	(0.45)
2010	0.00	0.22	0.21	0.04	0.11
	(0.00)	(0.42)	(0.41)	(0.20)	(0.31)
2011	0.00	0.06	0.19	0.00	0.05
	(0.00)	(0.24)	(0.39)	(0.00)	(0.23)
2012	0.00	0.00	0.21	0.00	0.05
	(0.00)	(0.00)	(0.41)	(0.00)	(0.21)
2013	0.00	0.00	0.06	0.00	0.01
	(0.00)	(0.00)	(0.23)	(0.00)	(0.11)
Wealth Index					
Poorest	0.20	0.24	0.24	0.20	0.22
	(0.40)	(0.43)	(0.43)	(0.40)	(0.41)
Poorer	0.23	0.24	0.25	0.23	0.24
	(0.42)	(0.43)	(0.43)	(0.42)	(0.43)
Middle	0.22	0.22	0.20	0.21	0.21
	(0.41)	(0.42)	(0.40)	(0.41)	(0.41)
Richer	0.20	0.15	0.15	0.21	0.18
	(0.40)	(0.36)	(0.36)	(0.40)	(0.39)
Richest	0.15	0.14	0.16	0.15	0.15
	(0.36)	(0.35)	(0.36)	(0.35)	(0.35)
Observations	1721	2652	3258	6792	14423

Figure 2.3. Height-for-Age Zscore by Month Age

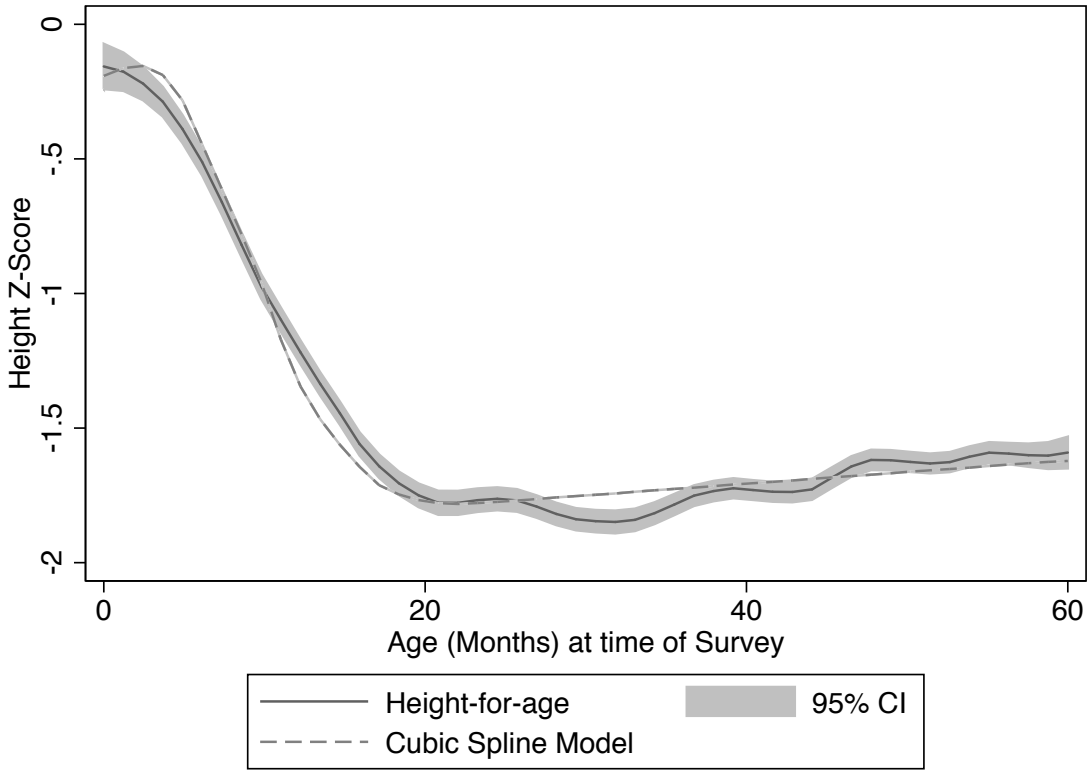


Figure 2.3 notes: Solid gray line shows a local polynomial of height-for-age by age in months, for a countries surveys pooled together, with its confidence interval. The dashed line shows the predicted height for age from a restricted cubic spline model with knots at months 3, 12 and 24.

Figure 2.4. Local Polynomials of Modified Height for Age

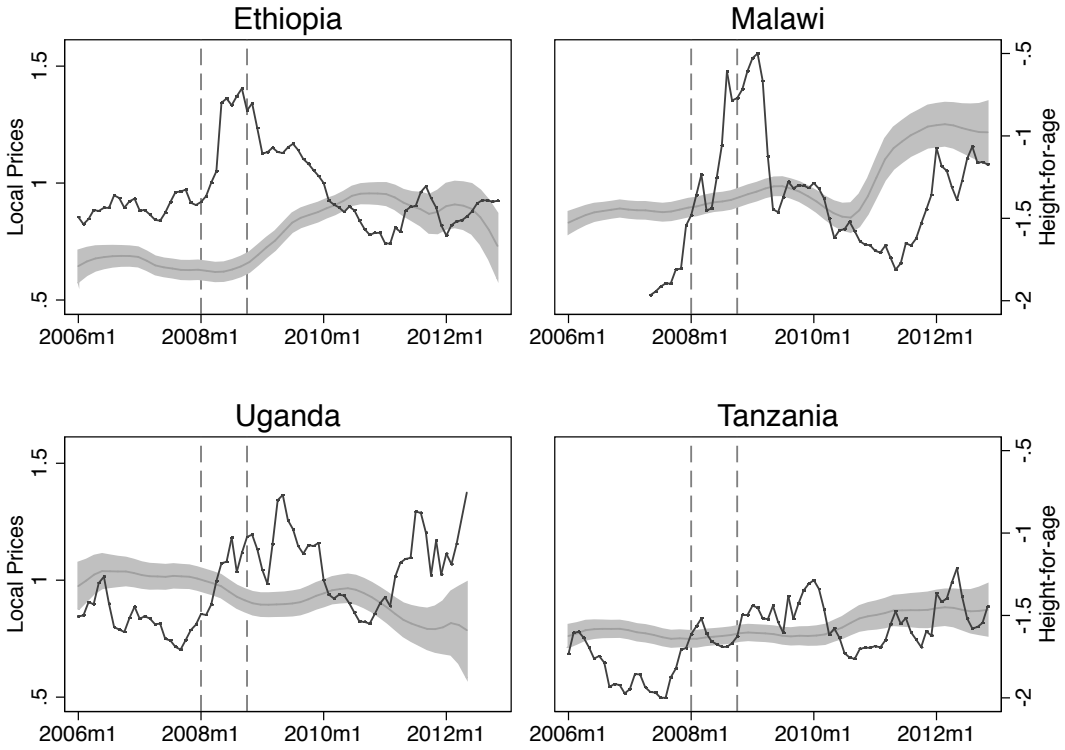


Figure 2.4 notes: Black line shows actual local prices, dashed vertical lines indicate the months of the international food price crisis. Gray lines are local polynomial of height-for-age by month of birth after partialling out cubic spline of age in months.

Table 2.13. Effects of Exposure to Local Food Price Crisis

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
		FE		FE		FE		FE		FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Exposure to Crisis During:										
-Early Childhood (7-24mo)	-0.14***	-0.21***	-0.24***	-0.38***	-0.09	-0.04	0.18	0.24	-0.15*	-0.08
	(0.02)	(0.03)	(0.07)	(0.10)	(0.08)	(0.13)	(0.13)	(0.12)	(0.07)	(0.13)
-Infancy (0-6mo)	-0.13**	-0.10	-0.22**	-0.34**	-0.29*	-0.01	0.15	0.11	0.13	0.33
	(0.04)	(0.06)	(0.08)	(0.12)	(0.12)	(0.21)	(0.13)	(0.15)	(0.11)	(0.17)
-Third Trimester	-0.18***	-0.30***	-0.34*	-0.58*	0.11	0.15	-0.07	0.02	0.07	0.09
	(0.05)	(0.08)	(0.15)	(0.23)	(0.18)	(0.33)	(0.14)	(0.18)	(0.12)	(0.20)
-Second Trimester	-0.07	-0.08	-0.28	-0.46	-0.13	-0.31	0.12	0.10	0.24	0.39
	(0.06)	(0.08)	(0.19)	(0.29)	(0.21)	(0.35)	(0.14)	(0.20)	(0.15)	(0.23)
-First Trimester	-0.12*	-0.20*	0.22	0.17	-0.19	-0.28	-0.19	-0.02	0.13	0.06
	(0.06)	(0.08)	(0.16)	(0.24)	(0.20)	(0.32)	(0.15)	(0.19)	(0.17)	(0.25)
-Trimester Before Conception	-0.04	-0.13*	0.04	-0.23*	0.03	0.40*	0.17	0.14	0.05	0.12
	(0.04)	(0.06)	(0.07)	(0.10)	(0.13)	(0.19)	(0.11)	(0.14)	(0.14)	(0.21)
Household Fixed Effects		Yes		Yes		Yes		Yes		Yes
Observations	48500	48500	14366	14366	14479	14479	6620	6620	13055	13055
R-Squared	0.10	0.15	0.16	0.19	0.08	0.11	0.10	0.12	0.18	0.23

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Crisis is defined as period when local prices 20% above detrended price mean. Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled forStandard Errors are clustered at household level.

Table 2.14. Effects of Exposure to Local Food Price Crisis by Gender

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1) Male b/se	(2) Female b/se	(3) Male b/se	(4) Female b/se	(5) Male b/se	(6) Female b/se	(7) Male b/se	(8) Female b/se	(9) Male b/se	(10) Female b/se
Exposure to Crisis During:										
-Early Childhood (7-24mo)	-0.11*	-0.18***	-0.09	-0.28**	-0.08	-0.09	0.05	0.27	-0.27	-0.26
	(0.05)	(0.05)	(0.09)	(0.11)	(0.08)	(0.08)	(0.15)	(0.20)	(0.22)	(0.19)
-Infancy (0-6mo)	-0.14	-0.24***	-0.07	-0.27*	-0.11	0.09	0.29	0.16	-0.19	-0.17
	(0.07)	(0.07)	(0.13)	(0.12)	(0.15)	(0.15)	(0.19)	(0.20)	(0.31)	(0.28)
-Third Trimester	-0.27**	-0.22**	-0.38	-0.35	-0.42	-0.51*	-0.33	0.04	-0.16	-0.14
	(0.08)	(0.08)	(0.24)	(0.25)	(0.25)	(0.25)	(0.25)	(0.24)	(0.22)	(0.19)
-Second Trimester	0.01	-0.02	0.28	-0.10	0.25	-0.31	0.10	0.33	-0.04	0.04
	(0.08)	(0.08)	(0.28)	(0.29)	(0.29)	(0.27)	(0.26)	(0.24)	(0.23)	(0.19)
-First Trimester	-0.02	-0.05	-0.12	0.03	0.23	0.53*	-0.43	-0.18	-0.04	-0.24
	(0.08)	(0.08)	(0.20)	(0.20)	(0.22)	(0.21)	(0.24)	(0.22)	(0.23)	(0.20)
Household Fixed Effects										
Observations	24379	24140	7340	7026	7211	7368	3323	3297	6505	6550
R-Squared	0.10	0.11	0.16	0.17	0.08	0.08	0.11	0.10	0.18	0.19

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Crisis is defined as period when local prices 1 SD above detrended price mean. Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled forStandard Errors are clustered at household level.

Table 2.15. Effects of Exposure to Local Food Price Crisis by Location

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1) Rural b/se	(2) Urban b/se	(3) Rural b/se	(4) Urban b/se	(5) Rural b/se	(6) Urban b/se	(7) Rural b/se	(8) Urban b/se	(9) Rural b/se	(10) Urban b/se
Exposure to Crisis During:										
-Early Childhood (7-24mo)	-0.17***	-0.02	-0.18*	-0.24	-0.13*	0.22	0.15	0.17	-0.34*	0.16
	(0.04)	(0.08)	(0.08)	(0.15)	(0.06)	(0.16)	(0.14)	(0.24)	(0.15)	(0.35)
-Infancy (0-6mo)	-0.25***	0.10	-0.18	-0.14	-0.09	0.30	0.05	1.07***	-0.23	0.11
	(0.05)	(0.12)	(0.10)	(0.20)	(0.12)	(0.32)	(0.15)	(0.30)	(0.23)	(0.47)
-Third Trimester	-0.23***	-0.34**	-0.33	-0.60	-0.56**	0.07	-0.04	-0.76*	-0.19	0.09
	(0.06)	(0.13)	(0.19)	(0.36)	(0.19)	(0.49)	(0.20)	(0.36)	(0.16)	(0.36)
-Second Trimester	-0.06	0.19	0.03	0.33	0.04	-0.55	0.20	0.32	-0.10	0.39
	(0.06)	(0.13)	(0.22)	(0.44)	(0.22)	(0.52)	(0.19)	(0.40)	(0.16)	(0.35)
-First Trimester	-0.07	0.15	-0.08	0.21	0.28	0.94*	-0.38*	0.10	-0.16	0.14
	(0.06)	(0.13)	(0.16)	(0.32)	(0.17)	(0.43)	(0.18)	(0.35)	(0.16)	(0.36)
Household Fixed Effects										
Observations	40561	7959	12099	2267	12332	2147	5440	1180	10690	2365
R-Squared	0.10	0.10	0.15	0.17	0.08	0.07	0.10	0.09	0.17	0.20

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Crisis is defined as period when local prices 1 SD above detrended price mean. Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A linear time trend is also controlled forStandard Errors are clustered at household level.

Table 2.16. Effects of Exposure to Local Food Price Crisis-Quadratic Time Control

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2) FE	(3)	(4) FE	(5)	(6) FE	(7)	(8) FE	(9)	(10) FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Exposure to Crisis During:										
-Early Childhood (7-24mo)	-0.13***	-0.21***	-0.12	-0.22*	-0.01	0.03	0.14	0.03	-0.28	-0.12
	(0.04)	(0.05)	(0.07)	(0.10)	(0.06)	(0.10)	(0.15)	(0.16)	(0.21)	(0.40)
-Infancy (0-6mo)	-0.16**	-0.30***	-0.24**	-0.35*	0.20	0.16	0.20	-0.37	-0.18	-0.07
	(0.06)	(0.09)	(0.09)	(0.14)	(0.13)	(0.23)	(0.20)	(0.27)	(0.21)	(0.42)
-Third Trimester	-0.23***	-0.32***	-0.39*	-0.70**	-0.34	-0.79*	-0.18	-0.11	-0.14	-0.02
	(0.06)	(0.09)	(0.17)	(0.27)	(0.18)	(0.31)	(0.22)	(0.29)	(0.15)	(0.34)
-Second Trimester	0.01	-0.08	0.01	0.03	0.13	0.20	0.20	-0.52	-0.00	0.26
	(0.06)	(0.09)	(0.20)	(0.31)	(0.21)	(0.35)	(0.21)	(0.28)	(0.16)	(0.35)
-First Trimester	-0.02	-0.12	-0.12	-0.49*	0.57***	0.59*	-0.32	-0.13	-0.13	0.02
	(0.06)	(0.09)	(0.15)	(0.21)	(0.17)	(0.26)	(0.19)	(0.26)	(0.17)	(0.36)
-Trimester Before Conception	0.01	-0.07	0.16*	0.01	0.08	0.12	0.19	-0.06	-0.25	-0.25
	(0.04)	(0.06)	(0.07)	(0.11)	(0.09)	(0.14)	(0.12)	(0.16)	(0.19)	(0.38)
Household Fixed Effects		Yes		Yes		Yes		Yes		Yes
Observations	48500	48500	14366	14366	14479	14479	6620	6620	13055	13055
R-Squared	0.10	0.15	0.16	0.19	0.08	0.11	0.10	0.12	0.18	0.23

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Crisis is defined as period when international prices 1 SD above detrended price mean. Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A quadratic time trend is also controlled forStandard Errors are clustered at household level.

Table 2.17. Effects of Exposure to Local Food Price Crisis-No Time Control

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2) FE	(3)	(4) FE	(5)	(6) FE	(7)	(8) FE	(9)	(10) FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Exposure to Crisis During:										
-Early Childhood (7-24mo)	-0.20***	-0.10*	-0.31***	-0.32***	-0.30***	0.04	0.38***	0.39***	-0.25	-0.13
	(0.03)	(0.05)	(0.05)	(0.09)	(0.04)	(0.09)	(0.10)	(0.10)	(0.14)	(0.33)
-Infancy (0-6mo)	-0.23***	-0.18*	-0.27***	-0.29*	-0.20	0.11	0.35**	0.15	-0.17	-0.07
	(0.05)	(0.07)	(0.08)	(0.13)	(0.11)	(0.20)	(0.13)	(0.16)	(0.21)	(0.41)
-Third Trimester	-0.29***	-0.23**	-0.46**	-0.69**	-0.61***	-0.80**	-0.05	0.33	-0.14	-0.02
	(0.06)	(0.08)	(0.17)	(0.27)	(0.18)	(0.30)	(0.17)	(0.23)	(0.15)	(0.33)
-Second Trimester	-0.04	0.00	0.01	0.12	-0.18	0.15	0.28	-0.16	0.00	0.25
	(0.06)	(0.08)	(0.20)	(0.31)	(0.20)	(0.33)	(0.18)	(0.23)	(0.15)	(0.34)
-First Trimester	-0.07	-0.03	-0.12	-0.41*	0.25	0.54*	-0.26	0.18	-0.12	0.02
	(0.05)	(0.08)	(0.14)	(0.21)	(0.15)	(0.25)	(0.16)	(0.23)	(0.15)	(0.34)
-Trimester Before Conception	-0.03	-0.00	0.18**	0.09	-0.14	0.06	0.19	0.10	-0.24	-0.25
	(0.04)	(0.06)	(0.07)	(0.10)	(0.08)	(0.12)	(0.10)	(0.13)	(0.15)	(0.34)
Household Fixed Effects		Yes		Yes		Yes		Yes		Yes
Observations	48500	48500	14366	14366	14479	14479	6620	6620	13055	13055
R-Squared	0.10	0.15	0.16	0.19	0.08	0.11	0.10	0.12	0.18	0.23

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Crisis is defined as period when international prices 1 SD above detrended price mean. Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. No time trend is used. Standard Errors are clustered at household level.

Table 2.18. Effect of (Log) Local Prices on Height-Quadratic Time Controls

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2) FE	(3)	(4) FE	(5)	(6) FE	(7)	(8) FE	(9)	(10) FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Log of Proxy Prices for:										
-Early Childhood (7-24mo)	-0.72***	-0.99***	-0.19	-0.60	-0.20	-1.10	3.03	2.62	0.01	-0.19
	(0.18)	(0.27)	(0.48)	(0.72)	(1.02)	(2.27)	(1.90)	(2.86)	(0.75)	(1.60)
-Infancy (0-6mo)	0.73***	0.60*	0.43	0.69	0.16	1.11	-0.59	-0.71	0.20	0.37
	(0.15)	(0.26)	(0.32)	(0.51)	(0.50)	(1.00)	(0.75)	(1.09)	(0.47)	(0.92)
-Third Trimester	-0.88***	-1.15***	-0.78	-1.05	-0.25	-1.72*	-0.02	-0.58	-0.39	0.07
	(0.15)	(0.26)	(0.40)	(0.63)	(0.43)	(0.79)	(0.39)	(0.59)	(0.28)	(0.49)
-Second Trimester	0.19	0.49	0.24	-0.04	-0.06	1.47	-0.22	0.47	0.13	0.29
	(0.18)	(0.31)	(0.40)	(0.65)	(0.44)	(0.93)	(0.43)	(0.59)	(0.29)	(0.57)
-First Trimester	0.35	0.04	0.73	1.45*	0.17	-0.33	-0.14	-0.37	-0.08	-0.32
	(0.18)	(0.32)	(0.41)	(0.65)	(0.43)	(0.94)	(0.44)	(0.62)	(0.28)	(0.53)
-Trimester Before Pregnancy	-0.24*	-0.37	0.25	-0.48	0.43	-0.75	0.86*	0.58	-0.02	0.25
	(0.12)	(0.21)	(0.31)	(0.47)	(0.39)	(0.83)	(0.38)	(0.54)	(0.23)	(0.45)
Observations	37937	37937	13893	13893	7489	7489	5899	5899	10656	10656
Adjusted R^2	0.137	0.187	0.168	0.217	0.069	0.099	0.110	0.141	0.182	0.248

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. A quadratic time trend is also controlled for. Standard Errors are clustered at household level.

Table 2.19. Effect of (Log) Local Prices on Height-No Time Trend Controls

	All		Ethiopia		Malawi		Uganda		Tanzania	
	(1)	(2) FE	(3)	(4) FE	(5)	(6) FE	(7)	(8) FE	(9)	(10) FE
	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se	b/se
Log of Proxy Prices for:										
-Early Childhood (7-24mo)	-0.52**	-1.09***	-1.60***	-1.48**	2.06***	-3.42	-0.84	-2.00	2.21***	-0.57
	(0.17)	(0.26)	(0.32)	(0.53)	(0.41)	(1.98)	(1.62)	(2.35)	(0.56)	(1.60)
-Infancy (0-6mo)	0.85***	0.77***	0.73**	0.63	-0.32	1.57	0.20	0.75	-0.51	0.11
	(0.14)	(0.23)	(0.28)	(0.44)	(0.30)	(0.87)	(0.66)	(0.93)	(0.41)	(0.90)
-Third Trimester	-1.01***	-1.17***	-0.83*	-0.90	0.21	-2.18**	-0.27	-1.14*	-0.23	-0.10
	(0.15)	(0.26)	(0.38)	(0.58)	(0.41)	(0.77)	(0.38)	(0.57)	(0.27)	(0.49)
-Second Trimester	0.26	0.49	0.19	-0.12	-0.51	1.68	-0.23	0.75	0.43	0.21
	(0.18)	(0.31)	(0.39)	(0.64)	(0.40)	(0.93)	(0.42)	(0.60)	(0.29)	(0.57)
-First Trimester	0.23	0.08	0.75	1.38*	0.33	-0.53	-0.24	-0.49	-0.30	-0.29
	(0.18)	(0.32)	(0.40)	(0.64)	(0.41)	(0.92)	(0.44)	(0.62)	(0.28)	(0.53)
-Trimester Before Pregnancy	0.04	-0.33	0.00	-0.72	0.84*	-1.08	0.12	-0.25	0.49**	-0.07
	(0.12)	(0.21)	(0.28)	(0.44)	(0.34)	(0.74)	(0.33)	(0.44)	(0.18)	(0.42)
Observations	37937	37937	13893	13893	7489	7489	5899	5899	10656	10656
Adjusted R^2	0.134	0.186	0.167	0.217	0.068	0.090	0.107	0.137	0.180	0.247

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Controls not reported include birth month, first born, female, region, wealth index and cubic spline of age in months. No time trend is used. Standard Errors are clustered at household level.

Table 2.20. Local Prices and Probability of Child being Female

	(1) Pooled	(2) Ethiopia	(3) Malawi	(4) Uganda	(5) Tanzania
-Trimester Before Pregnancy	0.0531*	-0.120	0.0231	0.245*	0.0484
	(0.0235)	(0.0732)	(0.0780)	(0.116)	(0.0598)
-First Trimester	-0.0384	0.208	-0.0457	0.0635	-0.0997
	(0.0348)	(0.119)	(0.112)	(0.165)	(0.0921)
-Second Trimester	0.0145	0.00988	0.0333	-0.223	-0.00833
	(0.0345)	(0.114)	(0.104)	(0.158)	(0.0908)
-Third Trimester	-0.0192	-0.108	-0.0707	0.0901	0.0686
	(0.0250)	(0.0697)	(0.0830)	(0.106)	(0.0613)
Observations	38874	14366	7666	5899	10943

Notes: * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ Controls not reported include birth month, region, and wealth index. Standard Errors are clustered at household level.

CHAPTER 3

THE EFFECT OF PUBLIC HEALTH EXPENDITURE ON INFANT MORTALITY: EVIDENCE FROM A PANEL OF INDIAN STATES, 1983–1984 TO 2011–2012

Using a panel data set of Indian states between 1983–1984 and 2011–2012, this paper studies the impact of public health expenditure on the infant mortality rate (IMR), after controlling for other relevant covariates like political competition, per capita income, female literacy, and urbanization. We find that public expenditure on health care reduces the IMR. Our baseline specification shows that an increase in public health expenditure by one per cent of state-level net domestic product is associated with a reduction in the IMR by about seven infant deaths per 1000 live births. We also find that political competition, female literacy and urbanization reduces the IMR. s JEL Codes: E12, E20.

Keywords: development; healthcare; mortality; south Asia.

3.1 Introduction

Despite rapid economic growth over the past three decades, India has witnessed very slow improvements in average health indicators of its population. Thus, India's position with respect to key indicators of health are below what would be expected on the basis of its income level. According to data for 187 countries available in the

2014 Human Development Report, India is ranked 130th in terms of gross national income per capita (2011 PPP $) but 136th in terms of life expectancy at birth (LEB, years), 139th in terms of under-five mortality rate (U5MR, deaths before the age of five years for every 1000 live births), and 140th in terms of the infant mortality rate (IMR, deaths before the age of one year for every 1000 live births).

One of the possible reasons for India's relatively worse performance on health indicators could be the low level of public health expenditure (Rao and Choudhury, 2012). India has not only one of the lowest levels of public health expenditure, but it also has one of the most privatised health care systems in the world (Sengupta, 2013). Drawing on data for the period 1995–2013 from the World Development Indicators, Figures 3.1 and 3.2 highlight these important facts about India's health care system. In Figure 3.1, we see that India has much lower public health expenditure (as a share of GDP) than most other regions of the world, including sub-Saharan Africa. Moreover, it has remained relatively stagnant over the last two decades, hovering around one per cent of GDP, whereas sub-Saharan Africa and the Arab World both have more than double that amount of public health expenditure for the same period. Figure 3.2 highlights the other important fact about India's health care system: its reliance, to a predominant extent, on a privatised system. For the period between 1995 and 2013, public expenditure has accounted for much lower than 40 per cent of total expenditure on health care in India. This is far lower than what is seen in other parts of the world, including sub-Saharan Africa.

In poor countries like India, public health expenditure is an important component of progressive policy. Large hospitals with expensive equipment and facilities,

proper sanitation, safe drinking water, nutritional programs, and similar interventions with impact on average health status can only be properly supported by public policy. Moreover, where household incomes are low and credit market imperfections high, the vast majority of the population are unable to provide adequate health care through private expenditure. Recent studies have noted the poverty deepening impact of out-of-pocket payments for health care (Garg and Karan, 2009; Shahrawat and Rao, 2012), which points to an important distributional aspect of public health expenditure in a poor country like India. Hence, public expenditure can be an important mechanism to ensure health care services to the population, especially the poorer sections of the population.

If public health expenditure in the context of a poor country like India has any positive impact on health outcomes, which seems likely, then India's exceptionally low level represents a potentially large opportunity. If it could increase its public health expenditure to the level of sub-Saharan Africa or Latin America, and if increases in public health expenditure have a positive impact on health outcomes, then India could rapidly improve the health status of its population. Such considerations have often been noted by Indian policy makers. The recently released Draft National Health Policy 2015 notes that perhaps 'the single most important policy pronouncement of the National Health Policy 2002 articulated in the 10th, 11th and 12th Five Year Plans, and the National Rural Health Mission (NRHM) framework was the decision to increase public health expenditure to 2 to 3 % of the NDP' (MoHFW, 2015). A similar target was also recommended by an earlier High Level Expert Group on Universal Health Coverage (Rao and Choudhury, 2012).

While the need for increasing public health expenditure seems necessary from a policy perspective, existing studies on the issue present at best a mixed picture. While some report that public health expenditure has very little effect, if at all, on health outcomes (Filmer and Pritchett, 1999), others find some positive impact (Anand and Ravallion, 1993). This is also true for the few studies that focus on India. Studies that use state-level panel data sets usually report that there is no significant relation between public health expenditure and health outcomes (Kaur and Misra, 2003; World Bank, 2004; Makela et al., 2013). On the other hand, some studies that have used individual level cross-sectional data from the National Family and Health Surveys do find some effect of public health expenditure on the IMR (Bhalotra, 2007; Farahani et al., 2010).

In this paper, we revisit the issue of the effect of public health expenditure on health outcomes in the context of Indian states, focusing on a single health outcome, namely the IMR.

There are two reasons for focusing on states in India. First, as we explain in greater detail below, even though health is a joint domain of state and central governments, the former play a much larger role in funding health service provision. Under India's constitution, State governments are dominantly responsible for health provision, both in terms of health care and public health measures (Gupta and Rani, 2004). This brings in variation in public health spending across states that can be exploited to estimate its effect on health outcomes, as we do in this paper. Second, the advantage of studying states within India is that data on public health expenditure and other key covariates is largely consistent and comparable. Hence, our statelevel

analysis allows us to avoid many difficult issues related to data comparability that becomes important in cross country settings.

We use the IMR as the key health outcome for our study because it is widely accepted among demographers, health economists, and policy makers as one of the most important indicators of the well-being of a population. In India, the acceleration of the rate of growth after the economic reforms in the early 1990s was accompanied by a slowdown in the rate of decline in infant mortality (Dreze and Sen, 2002). Following a period of rapid decline in the 1970s, the IMR in India stagnated for close to a decade, before declining again. For instance, the IMR in India fell from 80 (per 1000 live births) in 1990 to 68 in 2000 and further down to 50 in 2009. Even as average IMR for India has declined over the past few decades, states have displayed large variation in their performance. While some states like Kerala, Maharashtra and West Bengal have shown rapid improvement and are on their way to meet the Millenium Development Goals by 2015, others like Bihar, Gujarat, Orissa and Uttar Pradesh have lagged far behind (NIMS et al., 2012).

Using this observation as the point of departure, this paper aims to investigate the factors that determine the spatial and temporal variation of the IMR across Indian states, with a specific focus on the effects of public expenditure on health. Building on the empirical framework in Pritchett and Summers (1996), Filmer and Pritchett (1999), and NIMS et al. (2012), we use a panel data set for 31 Indian states and union territories between 1983–1984 and 2011–2012 to analyse the impact of public health expenditure (as a share of state-level net domestic product, NDP) on the IMR, after controlling for other relevant covariates like the female literacy rate, per

capita net state domestic product, political competition (which can affect the quality or effectiveness of policy), sex ratio (females per 1000 males), and urbanization. We find that public health expenditure has a significant effect on the IMR, that is, states that have higher public expenditure on health, as a share of their NDP, have lower IMR. Our preferred specification shows that an increase in public health expenditure by 1 per cent of state-level NDP can reduce the IMR by about seven infant deaths per 1000 live births. Our results also show, in line with existing results, that political competition, female literacy and urbanization reduce the IMR. Our preferred specification shows that an increase in the effective number of political parties (our measure of political competition) by one unit would reduce the IMR by about 1.8; an increase in the female literacy rate by 10 percentage points would reduce the IMR by 9.5; and an increase in the share of urban population by 10 percentage points would be associated with an IMR reduction of 3.3.

In discussing how our results compare with previous findings in the literature that point to a weak effect of public health expenditure on the IMR, we focus on the possibility of simultaneity bias as a potential problem with such findings. While it is intuitively plausible that higher public health expenditure will reduce the state-level IMR, it is also possible that there is a causation running in the opposite direction. States (or countries) with sub-par health outcomes are likely to increase their public health expenditure. In fact, it has been documented that, in India, the states that perform worse in terms of health outcomes also register a larger public health expenditure as a share of state GDP (Rao and Choudhury, 2012). If the bi-directional causality between public health expenditure and the IMR holds, then the OLS esti-

mate of the effect of the former on the latter will be biased. Another key contribution of this paper is to address this possible problem of endogeneity in two ways.

First, we use a simultaneous equation model to capture the bi-directional causality between public health expenditure and the IMR. Using this model, we show that the asymptotic bias in the OLS estimate of the effect of public health expenditure on the IMR is likely to be positive. To the extent that researchers have been unable to adequately address the problem of endogeneity, this can partly explain why many studies report insignificant effects of public health expenditure on the IMR: since the expected sign is negative (because higher public health expenditure is expected to reduce IMR), a positive bias will push the OLS estimate towards zero.

Second, using a panel of 31 Indian states and union territories between 1983–1984 and 2011–2012, we estimate the causal effect of public health expenditure on the IMR using an instrumental variables strategy. We use two fiscal variables as instruments for public health care expenditure: a state's own tax revenue and own non-tax revenue. Both of these fiscal variables can be expected to increase the 'fiscal space' of the states, and thereby enable them to devote more resources to improving the health status of its population (Heller, 2006). Thus, while it is clear that the instruments would be correlated with the endogenous regressor, public health expenditure, we go through a series of robustness checks to ensure that they meet the exogeneity condition. In addition, we include a full set of controls that have been highlighted as important determinants of IMR in existing studies: political competition (Chibber and Nooruddin, 2004; Besley and Kudamatsu, 2006; Fumagalli et al., 2013; Ashworth et al., 2014), female literacy (Subbarao and Raney, 1995; Caldwell,

1986, 1990; Anand and Barnighausen, 2004), per capita income (Pritchett and Summers, 1996), urbanization (Van de Poel et al., 2009; NIMS et al., 2012), sex ratio (Jayaraj, 2009), and state and year fixed effects (World Bank, 2004).

In addition to addressing long-standing debates in health economics and demography, our research also has immediate policy relevance. Even as India has grown rapidly over the past three decades, health indicators (infant mortality rate, life expectancy at birth, calorie intake, child malnutrition, prevalence of anemia in women, and so forth) of the vast majority have improved, if at all, sluggishly. Policy makers have been puzzled by this apparent disconnect between economic growth and improvement in living standards. A clue to this puzzle is provided by the results of this paper: public health expenditure has been stagnant over this period of growth. This could potentially explain why India has not witnessed big health improvements despite rapid economic growth.

The rest of the paper is organised as follows. In section 3.2, we introduce the empirical model and discuss the issue of simultaneity bias; in section 3.3, we describe our data sources; in section 3.4, we discuss our main results; and, section 3.5, concludes the discussion. Details about data sources are collected in the appendix.

3.2 Empirical Model

To motivate the empirical investigation in this paper, let us start with a discussion of Figure 3.3. It is a scatter plot of public health expenditure (as a percentage of state-level NDP) against the infant mortality rate for a pooled sample of 31 India states and union territories over the period 1983–1984 to 2011–2012. The scatter

plot also includes the line from a bivariate regression of the IMR on public health expenditure and a constant. The line slopes downward, suggesting that state-years with higher public health expenditure witness lower IMR. Our primary interest in this paper is to investigate whether the effect of public health expenditure on IMR, as depicted by the regression line in Figure 3.3, can be interpreted as a causal effect. To address the question of causal effect, we will need to address two sets of issues.

First, the scatter plot and regression line in Figure 3.3 leaves out many other covariates that could be correlated with both public health expenditure and IMR. To account for the possible effect of such determinants of IMR, we can use the following structural relationship:

$$y_{it} = z_{it}\alpha_1 + \mathbf{x'}_{it}\boldsymbol{\beta_1} + \mu_i + \delta_t + \sum_r \eta_r t + \epsilon_{1,it}, \tag{3.1}$$

where $i = 1, 2, \ldots n$ indexes states, $t = 1, 2, \ldots T$ indexes years (so that we have a total of $N = n \times T$ observations), y_{it} denotes average IMR, z_{it} denotes public health expenditure (expressed as a percentage of state-level GDP), $\mathbf{x'}_{it}$ is a $((k-1)\times 1)$ vector of controls that include the female literacy rate, per capita net state domestic product (expressed in 2004–2005 prices), the degree of political competition (measured by the effective number of parties in state governments), the sex ratio (measured by the number of females for every 1000 males) and urbanization rate, μ_i denotes a state-level fixed effect, δ_t is a year fixed effect, and $\sum_r \eta_r t$ are regional time trends.

The set of controls corresponds to important determinants of the IMR that have been highlighted by existing studies. Most studies find female literacy rate to be important because standard public health interventions that can reduce the IMR is

enhanced by the ability of the mother to read and follow basic instructions (Caldwell, 1986, 1990; Subbarao and Raney, 1995; Anand and Barnighausen, 2004). Per capita real income is an important determinant because it acts as a proxy for the level of private expenditure that can complement public expenditure in improving health status (Pritchett and Summers, 1996). Effective political competition is often found to have beneficial impacts on health status of the population (Besley and Kudamatsu, 2006; Fumagalli et al., 2013). The sex ratio (females per 1000 males) is a proxy for the level of patriarchal attitudes in society, with a lower sex ratio corresponding to a more onerous burden of patriarchy (Jayaraj, 2009). Urbanization is meant to capture the relative difference in the availability of health infrastructure, such as hospitals, primary health centers, doctors, nurses, between rural and urban areas (NIMS et al., 2012). Finally, state fixed effects control for unobserved state-level factors that change slowly over time, like cultural norms or attitudes of government officials, and time fixed effects control for factors that impact all states over time, like technological change.[1]

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[1]We could not include any health infrastructure variable or efficiency measure in our model due to lack of data, but our variables incorporate the determinants of differences in such measures between the states. There are few state-level studies on aspects of health care efficiency in India (Kathuria and Sankar, 2005; Shetty and Pakkala, 2010; De et al., 2012; Prachitha and Shanmugam, 2012). These studies mainly focus on the major states, and, although their findings agree on some degree of heterogeneity with regards to the relative efficiency of health care at the state-level, they differ to a great extent in terms of their models and understanding of the determinants of efficiency. The distinction that is most relevant for the purposes of our analysis is whether the variables in our specification (mainly public health expenditure, female literacy, income and urbanization) are considered as directly affecting the efficiency of the health care system, that is, considered as input variables in the production function, or as exogenous variables that affect the relative efficiency but not the production function. In the former case, these variables are used in estimating the efficiency level, whereas in the latter case, the level of efficiency is measured focusing on infrastructure variables (such as the number of primary health centers, sub-centers, doctors, and

The second issue that we need to address in interpreting the observed relation between public health expenditure and IMR (Figure 3.3) relates to dealing with the possibility of bi-directional causality and, by implication, the problem of simultaniety bias. In the next section, we present a discussion of the simultaneity bias and how we address it.

3.2.1 Simultaneity Bias

To fix ideas, let us posit the bi-directional causality in terms of two structural relationships. The first is a re-written version of (3.1), and captures the causal effect of public health expenditure (PHE), z, on the IMR, y,

$$y_{it} = z_{it}\alpha_1 + \mathbf{u'}_{it}\boldsymbol{\beta_1} + \epsilon_{1,it}, \tag{3.2}$$

and the second captures the causal relationship running in the opposite direction from IMR (y) to PHE (z)

$$z_{it} = y_{it}\alpha_2 + \mathbf{v'}_{it}\boldsymbol{\beta_2} + \epsilon_{2,it}, \tag{3.3}$$

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so forth), and the efficiency measure is then regressed on non-health system determinants of health (including our control variables) to find out the factors that affect the relative efficiency (Kinfu and Sawhney, 2015). The discussion of the appropriate approach to measure efficiency of the health care system is beyond the scope of our paper; suffice it to say that our control variables are shown to have significant effect (direct or indirect, depending on the study) on the efficiency level, and their effect appear to dominate the effects of various infrastructure variables when used in combination in an efficiency estimation model (see, for example, Prachitha and Shanmugam (2012)). Hence, these results suggest that our variables, along with state and year fixed effects, capture the effect of differences between states in terms of efficiency of the health care.

where $i = 1, 2, \ldots n$ indexes states, $t = 1, 2, \ldots T$ indexes years, $\mathbf{u}'_{it} = (1, u_{1,it}, u_{2,it}, \ldots, u_{k-1,it},)$ and $\mathbf{v}'_{it} = (1, v_{1,it}, v_{2,it}, \ldots, v_{k-1,it},)$ are k-vectors of strictly exogenous variables (including a constant), $\boldsymbol{\beta_1}, \boldsymbol{\beta_2}$ are vectors of parameters, and $\epsilon_{1,it}, \epsilon_{2,it}$ are structural errors with

$$\begin{pmatrix} \epsilon_{1,it} \\ \epsilon_{2,it} \end{pmatrix} \sim \begin{pmatrix} \sigma_1^2 & 0 \\ 0 & \sigma_2^2 \end{pmatrix}.$$

Basic economic theory and intuition suggests that the partial effect of PHE on the IMR will be negative so that $\alpha_1 < 0$; similarly, the partial effect of the IMR on PHE is likely to be positive, so that $\alpha_2 > 0$. Since the expected sign of α_1 is negative, a positive bias might lead to an estimate of α_1 that is close to zero. Thus, models that do not address the simultaneity bias will produce weak estimates for the effect of PHE on the IMR, and this may offer a partial explanation for the predominance of such findings in this literature. To deal with the possible problem of endogeneity of public health expenditure and the resulting simultaneity bias in (3.1), we will use an instrumental variables estimation strategy.

3.2.2 Identification through Instrumental Variables

We will use two different fiscal variables as instruments for public health expenditure: a state's own tax revenue, and a state's own non-tax revenue. In India, total revenue of a state is the sum of tax and non-tax revenue (in the main, the latter are user fees for various services offered by the government). The tax revenue, in turn, is the sum of *own* tax revenue (tax revenue collected by the state government) and tax

revenue provided by the Central government.[2] Similarly, the non-tax revenue is the sum of *own* non-tax revenue and grants from the Central government. Thus, the sum of a state's *own* tax and non-tax revenue can be used to capture the 'fiscal space' of a state.[3] The larger the tax and non-tax revenue a state can collect, the easier will it be for the state to finance public health expenditure. We will discuss possible concerns about the exogeneity of these two instrumental variables extensively when we discuss robustness checks of our main results in section 3.4.3. Let us first present the data set used for the analysis in this paper.

3.3 Data: Variables and Summary Statistics

We use an unbalanced panel data set covering the period between 1983–1984 and 2011–2012 for 31 Indian states and union territories: Andhra Pradesh, Arunachal Pradesh, Assam, Bihar, Chandigarh, Dadra & Nagar Haveli, Delhi, Goa, Damand & Diu, Gujarat, Jammu & Kashmir, Karnataka, Kerala, Lakswadeep, Madhya Pradesh, Maharashtra, Manipur, Meghalaya, Mizoram, Nagaland, Orissa, Puducherry, Pun-

[2]In India's federal structure, tax collection is divided between the central and state governments. The central government collects direct taxes like income, corporation & personal, dividend and wealth tax, and indirect taxes like central excise, customs, and service tax. On the other hand, state governments collect the value added tax, excise on alcohol, luxury tax, electricity duty, entertainment tax, stamp duty, property tax and professional tax.

[3]Heller (2006, pp. 75) defines fiscal space as 'the capacity of government to provide additional budgetary resources for a desired purpose without any prejudice to the sustainability of its financial position.'

jab, Rajasthan, Tamil Nadu, Uttar Pradesh, and West Bengal.[4] Here, we provide a brief description of the data set; for more details, see the appendix.

3.3.1 Variables

The outcome variable of interest in our analysis is the state-level infant mortality rate (IMR). The IMR is the probability of death faced by infants of age one year or lower, and is measured as the number of deaths of infants under one year old per 1000 live births. The key explanatory variable for our study is state-level public health expenditure (PHE). We measure PHE as the sum of the state-level expenditure on medical & public health, family welfare and nutrition.

In India, public health expenditure is undertaken at all three levels of government: central (federal), state, and local. The central government undertakes expenditures in two forms: direct expenditure, and grants-in aid to state governments. State governments incur expenditures out of the grants-in aid and other resources, for example, tax revenues, available to them. Some state-level expenditure also takes the form of transfers to local government bodies. Local government bodies, in turn, incur expenditures out of these transfers and from other resources that they have. The total of all the expenditures incurred at the three tiers of government provides an estimate of public health expenditure in India. In this paper, we are interested in analyzing the effect of state-level public health expenditure on state-level IMR. Hence, the data for our research project relates only to state-level expenditure on

[4]In the early 2000s, the states of Jharkhand, Chhattisgarh and Uttarakhand were carved out of Bihar, Madhya Pradesh and Uttar Pradesh, respectively. For comparability, we keep them as part of the larger states all through.

health care. This includes expenditures incurred by state governments from Central grants-in aid and from other resources available to the state government (for example, through taxation).[5]

An important source of variation that might be correlated with both PHE and the IMR is the general quality of policy making and implementation of public welfare schemes at the state level. Previous studies have documented that political competition improves policy making (Besley and Case, 1995; Rodgers and Rodgers, 2000; Besley et al., 2005). Perhaps more pertinent for this paper, many recent studies have found that the degree of political competition has beneficial effects on health status of the population (Besley and Kudamatsu, 2006; Fumagalli et al., 2013). The link in question seems intuitively clear: intense political competition between political parties can lead to an increase in the effectiveness of governance and accountability

[5]Thus, our data set does not include direct central government expenditure and local-level expenditure funded by resources other than state-level transfers. According to NHAC (2009), the local-level expenditure funded by resources other than state-level transfers is a negligible part of total expenditure. While we would have liked to include each state's portion of total central government expenditure on health, we are unable to do so because of lack of easily available data on the state-wise distribution of central government expenditure on health. According to data compiled by Gupta and Chowdhury (2014), the bulk of health care spending is contributed by states. In 2010–2011, about 64 per cent of total public health care spending came from expenditure by states, about 31 per cent came from expenditure by the central government (direct expenditure and grants-in aid to states), and the rest was accounted for by expenditure of local government bodies. If we count the grants-in aid as part of state-level expenditure, then the contributions of the central government varies between 20 and 30 per cent of total public health expenditure (Choudhury and Nath, 2012). Thus, the figures for state-level public health expenditure used in this paper covers between 70 and 80 per cent of total public expenditure on health care in India. Yet, because our health outcome of interest is the infant mortality rate, which is likely to respond to even low levels of increases in public expenditure, and because, as stated above, under India's constitution, state governments, rather than the central government, are primarily responsible for health provision, both in terms of health care and public health measures (Gupta and Rani, 2004), our data is expected to cover the part of total health expenditure at the state-level that is most pertinent to our analysis.

of the organs of the state, both of which can lead to an improvement in policy making. This can not only force state governments to devote more resources to welfare activities (like health care), but also improve the effectiveness of existing delivery mechanisms that have a direct impact on health status for every level of resource allocation. Moreover, higher level of political competition may provide incentives for the political parties to increase public expenditure on education, healthcare, nutrition and so forth, to increase their chances of getting re-elected.

Borrowing from the political science literature, we use an index of the index of effective number of parties in the government as a proxy for the degree of political competition in the state government, a process that is accentuated by the growth of coalition governments over the last two decades in India (Laasko and Taagepera, 1979). For state level assembly election years, the index of the effective number of parties in any state government is computed as

$$N = \frac{1}{\sum_{i=1}^{n} p_i^2}$$

where N is the effective number of parties in a state government, $i = 1, 2, \ldots, n$ indexes parties in the state government, and p_i is the share of party i in the government.[6] The value of the index remains unchanged until the next election year, when a new government is formed and a new configuration of parties emerge as the

[6]This measure of the effective number of political parties was first proposed by Laasko and Taagepera (1979). Although many alternatives have been proposed over the years, it is still considered as the standard for comparative political research (Kline, 2009). Chamon et al. (2009, p.4) note that starting with Laasko and Taagepera (1979), the political science literature has used the effective number of parties as 'the main measure of political competition.'

governing coalition. If a single party forms a government, the value of N is unity, and as the number of parties increase, the value of N increases.

While we have highlighted the possible positive impact of a higher number of effective political parties through more intense political competition, it is worth pointing out that it might also have an opposite effect. If the effective number of political parties is understood as a measure of the 'hyper-fractionalization' of political power, then a higher value of the index can lead to higher rent seeking behaviour, and militate against expenditures that are in the long term benefit of the general population. For instance, coalition governments with a higher number of parties in the ruling coalition make the coalition unstable and increase the probability of dissolution of the government before the end of the full five year term. This increases the incentive for each party in the coalition to make expenditure that cater to their narrow support bases, rather than undertake expenditure that would have long term benefits for the population. Thus, depending on the strength of these opposite effects, the effective number of parties might have a positive or negative effect on the IMR.

The additional controls used in the model are the following: per capita real income, population sex ratio, (adult) female literacy, and urbanization.[7]

3.3.2 Summary Statistics

Table 3.1 provides summary statistics, mean and standard deviation, for the variables used for the analysis in this paper. Summary statistics are reported roughly every five years roughly spanning our sample period from 1983–1984 to 2011–2012.

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[7]Details of the definitions of all variables and data sources are available in Appendix.

The average IMR has declined secularly over time, falling from 91.18 in 1985–1986 to 36.35 in 2010–2011. The average public health expenditure (which includes medical & public health, family welfare, and nutrition) as a share of state-level NDP increased over the decade of the 1980s, but has more or less declined since then. It started rising again from the mid-2000s.

The degree of political competition, as captured by the index of effective number of parties in state governments, has increased over the sample period. The mean of the effective number of parties increased from 2.13 in 1985–1986 to 2.37 in 1990–1991, and remained relatively unchanged for the next five years. Since then, it has increased almost continuously: the mean of the effective number of parties was 2.36 in 1995–1996, 2.60 in 2000–2001, and 2.94 in 2005–2006.

Female literacy and per capita real NSDP have both increased secularly over the two and a half decade period from 1983–1984 to 2011–2012. The (adult) female literacy has nearly doubled over this period, increasing from 42.12 per cent in 1985–1986 to 70.96 in 2010–2011. Average per capita real NSDP has increased by about 3.2 times over the sample period (from 13227 rupees in 1983–1984 to 42617 rupees in 2011–2012, both expressed in 2004–2005 rupees), growing at an annual compound rate of 4 per cent per annum.

The average sex ratio follows a U-shaped pattern: it worsened from 922.12 in 1985–1986 to 917.20 in 2000–2001, and then improved to 925.83 in 2011–2012. Urbanization has increased steadily from 1983–1984 onwards, other than a small dip since the mid-2000s. Average values of both own tax revenue and own non-tax rev-

enue (measured as share of state-level NDP) has been stagnant over the period of analysis.

3.4 Empirical Results

3.4.1 Main Results

The main results of our empirical analysis are summarised in Table 3.2. The dependent variable in the model is the infant mortality rate; the independent variables are public health expenditure (as a share of state-level NDP), effective number of political parties in state governments, log of per capita net state domestic product (at constant prices), the female literacy rate, the sex ratio of the population, the urbanization rate, and state and year fixed effects.

In the first column of the table, we report OLS estimates of the bivariate regression of IMR on the public health expenditure with state and year fixed effects. The estimate of the coefficient on public health expenditure is -1.75, but it is imprecisely estimated. In the next column, we report 2SLS estimates for the same model (with own tax revenue, own non-tax revenue and the effective number of parties in government as instrumental variables for PHE). The coefficient on PHE becomes -7.17 and is significant at the five per cent level. Given that the OLS estimate is likely to be biased upwards, this is expected.

As we move from column 2 through 6 in Table 3.2, we keep adding controls: effective number of parties in state governments, log of per capita NSDP, the female

literacy rate, the sex ratio, and the rate of urbanization. The coefficient on PHE does not change much, and remains statistically significant at standard levels of significance.

Our preferred specification is the model in column 6 in Table 3.2, which includes all the controls, and state and year fixed effects. We will use the estimates in this model for interpretation. The coefficient of -7.19 on PHE means that an increase in PHE by 1 per cent of state-level NDP would reduce the infant mortality rate by about seven (deaths of infants per 1000 live births). This is an economically meaningful and statistically significant effect and suggests that increasing public health expenditure would have a large effect on the reduction of IMR in India.[8]

In Table 3.2, we also see that the effective number of parties, female literacy rate and urbanization emerge as significant and strong determinants of IMR. An increase in the index of effective number of parties by 1 unit reduces the IMR by 1.84 per 1000 live births. This suggests that an increase in the degree of political competition, as captured by an increase in the effective number of political parties

[8]In their important work, Filmer and Pritchett (1999) report an elasticity of public health expenditure on the IMR of -0.078 (Filmer and Pritchett, 1999, Table 2). Our estimates suggest a much larger effect of public health expenditure: the elasticity of public health expenditure (as a share of NDP) at mean values of IMR and public health expenditure is -0.366 ($= (-7.19) * (1.85/36.35)$), where we use the mean value of IMR (36.35) and public health expenditure (1.85) for 2011–2012 (see Table 1 in this paper). The fact that Filmer and Pritchett (1999) get such a low estimate of the elasticity might be because their instruments, used for the 2SLS estimation, are weak. They do not report first stage results to argue otherwise. Moreover, the imprecisely estimated 2SLS coefficients in Filmer and Pritchett (1999) are consistent with weak instrument problems. In line with Filmer and Pritchett (1999), state-level analysis in India mostly report weak effect of public health expenditure on the IMR (Kaur and Misra, 2003; World Bank, 2004; Makela et al., 2013). One study that reports a significant effect is Bhalotra (2007), who restricts the sample to rural households, allows for lagged effects, and finds a significant effect of health expenditure on the IMR, with a long run elasticity of about -0.24.

in the government, improves the health status of the population. An increase in the female literacy rate by 10 percentage points would reduce the IMR by 8.9, and an increase in the share of urban population by 10 percentage points would reduce the IMR by 2.8. The direction of these results is in line with existing studies. For instance, Filmer and Pritchett (1999) and Saurabh et al. (2013) find a significant inverse relationship between IMR and female literacy. World Bank (2004) finds a negative effect of adult female literacy on the infant mortality rate only beyond a threshold per capita GSDP level. Using data from three rounds of the National Family and Health Surveys, NIMS et al. (2012) find significant effect of the location of residence (rural/urban) on neonatal and post-neonatal mortality, but weak effect on child mortality. This suggests two things. First, increasing female literacy must figure as one of the important elements of any strategy to improve the health status of the population in India. Second, urbanization improves the IMR, possibly through better access to basic health care facilities that are lacking in remote and rural areas.

The identification of the causal effect of public expenditure on the infant mortality rate rests on the validity of our instruments. To assess the validity of our instruments, we conduct standard tests, and carry out an extensive set of additional robustness checks (reported in Table 3.4 and discussed below in Section 3.4.3). The standard tests relate to the strength of the first stage regression, which we capture through the Kleibergen-Papp (Wald) F-stat, and the overidentification test, captured through the J-statistic. Both these statistics are reported for all specifications estimated by 2SLS in Table 3.2. All specifications have F-stats that are significantly larger than 10, the rule of thumb value for ruling out weak instrument problems with a single

endogenous regressor. The p-values associated with the over-identification tests are generally larger than 0.01, which suggests that the instruments pass the exogeneity test.[9]

3.4.2 Reduced Form and First Stage Regression

In Table 3.3, we report estimates from the reduced form model, where the dependent variable (IMR) is regressed on the excluded instruments and the included regressors, and the first stage model, where the endogenous regressor (public health expenditure) is regressed on the excluded instruments and the included regressors. The parameter estimates for the reduced form model are meaningful in magnitude and sign. For instance, both the own tax revenue and the own non-tax revenue have negative coefficients (though the former is not precisely estimated); both female literacy and urbanization reduce the IMR. In the first stage regression, the two instruments, own tax revenue, and own non-tax revenue, have positive coefficients that are also statistically significant. This implies that increases in own revenue of states, both tax and non-tax, have a significant effect in increasing fiscal space, and lead to a reduction in the IMR through this channel.

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[9]Model 7 in Table 3.2 reports results of estimating the model by Limited Information Maximum Likelihood (LIML) estimation. It is well-known that LIML is less precisely estimated but is also less biased. Thus, comparing the overidentified model 6 in Table 3.2 (that has been estimated with 2SLS) with model 7 in Table 3.2 (that has been estimated with LIML) is useful. There is no significant change in the coefficient on public health expenditure, going from -7.19 to -7.20. Moreover, the coefficients on effective number of parties, female literacy and urbanization also remain stable. This increases the confidence in the overidentified 2SLS results of the basic model (column 6 in Table 3.2).

3.4.3 Robustness Checks

While the KP (Wald) F-stat and the J-stat reported in Table 3.2, in conjunction with the results of the reduced form and first stage regression reported in Table 3.3, indicate towards the validity of our estimation strategy, here we discuss some additional concerns that might arise about the crucial exogeneity assumption of the instruments. Since the exoegneity of the instrumental variables means that they have an impact on the IMR only through their impact on public health expenditure (PHE), we need to be reasonably sure that there are no other channels through which the instrumental variables might have an impact on IMR.

A potential cause for our instrument to be non-excludable would be if it was associated with more efficient or effective government. We limit this possibility in two ways: (a) we include state fixed effects to control for time-invariant, state-level factors that might impact effectiveness of government; and (b) we control the time varying factors that impact effectiveness of government by including the index of effective number of parties in the state government as a regressor. This measure of political competition, that is, effective number of parties in state governments, is well known to be correlated with government efficiency and performance (Besley and Case, 1995; Rodgers and Rodgers, 2000; Besley et al., 2005). Along the lines of the earlier papers, Ashworth et al. (2014) find that number of parties has a significant positive effect on the productive efficiency of municipal policy. Similarly Guyvoronskiy (2015) find that electoral competition improves efficiency of public health care systems in Russia. In the Indian context, Chibber and Nooruddin (2004) find a relationship between political competition and state government effectiveness.

Hence, we believe that including the index of effective number of parties in the state government as a regressor in our regression model addresses the concern about effectiveness of government being an omitted variable that could lead to a violation of the exogeneity of our instruments.

Another concern might be that rainfall shocks could operate as an omitted variable. For instance, an adverse rainfall shock might reduce agricultural output and incomes, and thereby lead to higher mortality of infants, and also lead to lower state-level tax revenue. This is not a problem for our identification strategy because agricultural income is not taxed in India. A second concern would be that state-level negative demand shocks would reduce the state-level NDP, leading to higher IMR and also lower state-level tax revenue. We block off this possible channel by explicitly controlling for state-level real NDP in Table 3.2.

In Table 3.4, we report additional results that provide robustness checks. Column 1 reproduces the results for our preferred specification for comparison, and in columns 2 through 6, we make one change at a time with respect to the preferred specification to compare the effect of that change on our results.

First, we check for the possibility of own tax and nontax revenue to be correlated with other items of state level expenditure, such as food subsidy, transfer to the poor, sanitation & drinking water, that are determinants of state level IMR. We address this possible concern in two ways. First, we use a broad definition of public health expenditure that includes expenditure on medical & public health, family welfare, and nutrition. Second, in column 2 in Table 3.4, we include log aggregate state level

social sector expenditure as an additional control. In column 3, we include log of total non-public health expenditure (total expenditure less public health expenditure) as an additional control. Neither of these additional controls changes lead to a significant change in our results in column 1: the coefficient on PHE changes to -7.19 and -7.84 (the latter is less precisely estimated than would be desirable).

Our second robustness check considers the possibility that the ideological bent of state governments is correlated with the error term. This might be driven by the fact that left parties are more likely to favour public over private expenditure on health care. Political ideology might also have an independent effect on the IMR through other non-public health spending channels like a generally more pro-people orientation of policies that improve the quality of services conditional on the quantity of expenditure. To block this possible channel, we include an index of political ideology in column 4. To compute this index we first assign an ideology score ranging from one to five to each political party, with one denoting a Left ideology and five a Right ideology. The index of political ideology of the government is the weighted average of ideology scores of the parties in the government, with share of seats won by parties used as weights. Inclusion of this regressor does not change the results from column 1 too much with the coefficient on PHE becoming -7.40.

Third, it is possible that states that offer higher quality services might be able to collect more user fees (and hence, earn more non-tax revenue). If some of these services improve state-level IMR, then one of our instrument, own non-tax revenue, would be correlated with the error term. We address this problem by including lags of PHE (which capture the provision of government services) in column 5. The

results are more or less unchanged, and the coefficient on PHE increases a little to -12.29. The long run impact of PHE on IMR, that is, the sum of the coefficients on all the lags of PHE, is -6.60 and statistically significantly different from zero (p-value $= 0.01$).

The last robustness check that we report in Table 3.4 is to account for a possible 'south India' effect. It is well known that states in south India, like Kerala and Tamil Nadu, are generally better performing in terms of human development indicators. To make sure that our results are not driven by rapid improvements in the south Indian states, we include a south India time trend (a dummy for south India interacted with a linear time trend) in column 6. Again, the results remain relatively unchanged, with the coefficient on PHE estimated as -7.04.

3.4.4 Some Caveats

While the results reported in this paper present interesting evidence regarding the link between public health expenditure and the infant mortality rate, we would like to draw attention to some possible limitations of our work.

The first area of concern is that the effect of public health expenditure is statistically quite weak when we restrict our analysis to a sub-sample of 15 major states–Andhra Pradesh, Assam, Bihar, Gujarat, Jammu & Kashmir, Karnataka, Kerala, Madhya Pradesh, Maharashtra, Orissa, Punjab, Rajasthan, Tamil Nadu, Uttar Pradesh and West Bengal. It is common in the literature to see if results of such cross-state analyses are valid for different sub-samples, like poor versus the non-poor states (World Bank, 2004), or the 15 major states (that account for the vast majority of the population, area and economic activity in the country) versus

all states. In Table 3.5, we report results of estimating our basic model for the full sample of 31 states and a sub-sample of the 15 major states. In addition to investigating robustness for this sub-sample of 15 major states, we also investigate robustness for different definitions of public health expenditure by reporting results for three increasingly comprehensive ways of measuring public health expenditure: PHE1 = expenditure on medical & public health (% of state NDP); PHE2 = PHE1 + expenditure on water & sanitation (% of state NDP); PHE3 = PHE2 + expenditure on nutrition (% of state NDP).

Columns 1 through 3 in Table 3.5 report results for the sample of all states, with column 1 using PHE1, column 2 using PHE2 and column 3 using PHE3 as the measure of public health expenditure. It is reassuring to see that the results are robust to different ways of measuring public health expenditure: the coefficient on public health expenditure remains stable, and the coefficients on the three key regressors, namely, political competition (index of effective number of parties in government), female literacy and urbanization, remain more or less unchanged across columns 1 through 3.

Columns 4 through 6 in Table 3.5 report analogous results for a sub-sample of the 15 major states. There are two issues worth highlighting. First, the coefficient on public health expenditure increases in magnitude but is no longer statistically significant. Looking at the KP-stat for the first stage regression we see a probable source of the problem of imprecise estimates: the KP-stat is much smaller than the rule of thumb cut-off of 10, and so the instruments are too weak to predict the

variation in public health expenditure. Second, the statistical significance of two key regressors is also adversely affected: effective number of political parties and urbanization are no longer significant explanatory variables. This issue needs further investigation.

The second area of concern relates to the way we have measured the public health expenditure, that is, as a share of state-level NDP rather than in terms of per capita real public health expenditure. Since many existing studies (World Bank, 2004; Bhalotra, 2007; Farahani et al., 2010) have used the second measure, we report results using this measure - logarithm of per capita real public health care expenditure - in Table 3.6.[10] Following the pattern in Table 3.2, we start with a stripped down model in column 1 of Table 3.6 (where the log of per capita real public health care expenditure and effective number of political parties are the only two regressors), and then add controls one by one. As we move down the columns, we see that the coefficient on the log of per capita real public health care expenditure remains negative and statistically significant. In column 7, we add state fixed effects and that reduces the statistical significance of the coefficient on the log of per capita real public health care expenditure dramatically. While we suspect that this is a problem of weak instruments (as suggested by the low value of the KP-stat), we would also like to flag this issue for further investigation.

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[10]We compute real per capita public health expenditure by deflating the nominal per capita public health expenditure by a state-level NDP deflator. The deflator is computed as the ratio of real net state domestic product (at 2004–2005 prices) and the nominal net state domestic product.

3.5 Conclusion

In this paper, we investigated the relationship between public health expenditure and the infant mortality rate using an unbalanced panel of 31 Indian states from 1983–1984 to 2011–2012. To investigate whether the relationship can be interpreted in a causal manner, we focused on the possibility of bi-directional causation between public health expenditure and the infant mortality rate. We argued that the simultaneity bias arising from bi-directional causation is likely to be positive. Since the association between public health expenditure and IMR is expected to be negative, a positive simultaneity bias might explain why many existing studies do not find any significant negative effect of public health expenditure on the infant mortality rate.

In our empirical analysis, we addressed the issue of simultaneity bias by using an instrumental variables strategy. We used a state's own tax revenue, and own non-tax revenue as instruments for public health expenditure. Our estimation results show that public health expenditure as a share of state-level net domestic product is negatively associated with the state-level infant mortality rate. Our baseline specification suggests that an increase in public health expenditure by 1 per cent of state-level NDP will reduce the infant mortality rate by about seven deaths per 1000 live births. Our estimate is far more stronger than what has been previously reported in the literature, for instance in Filmer and Pritchett (1999), and suggest that Indian states can reduce the infant mortality rate rapidly by increasing the level of public health expenditure (as a share of state-level NDP), which has been extremely low in the last decades. We also find that an increase in the degree of political competition, female literacy and urbanization reduces infant mortality rates.

Appendix: Data Sources

Infant Mortality Rate

The main source of data on the IMR, and other vital health statistics, in India is the Sample Registration System.There are 4 main sources of vital statistics in India: (a) Sample Registration System (SRS), (b) Civil Registration System (CRS), (c) indirect estimates from the decennial Census, and (d) indirect estimates from the National Family Health Surveys (NFHS). Among these 4 sources, the SRS remains the most important source for reliable data at an annual frequency (Mahapatra, 2010). The Sample Registration System (SRS) is an annual demographic survey covering 1.2 million households, one of the largest, continuous demographic surveys in the world, conducted since 1970 by the Registrar General of India and provides reliable data on fertility and mortality indicators for the country and larger states (Mahapatra, 2010). We downloaded state-level IMR data compiled from annual reports of the SRS by the Government of India and made available through the Open Government Data Platform India.[11]

Public Health Expenditure

The state-level public health expenditure data comes from the Economic and Political Weekly Research Foundation's *India Time Series* data base, which, in turn, uses data from the *Handbook of Statistics on State Government Finances* published by the *Reserve Bank of India* in 2010.For details see `http://www.epwrf.res.in/home.aspx`

[11]See `https://data.gov.in/`

Broadly, total expenditure of a state government can be broken up into expenditures on three types of services: (a) social services, (b) economic services, and (c) general services. Expenditures on (a) and (b) combined is called 'development expenditure', and expenditure on (c) is called 'non-development expenditure'. Each of the first two expenditure categories can be further broken down into revenue expenditure (current expenditure including interest payments) and capital expenditure (capital outlays like construction of roads, buildings, irrigation projects). Revenue expenditures are financed through current income, the main source of which are taxes and duties. Capital expenditures are not financed from current income, but instead rely on loans, surplus revenue from previous years, and so forth.

The category that is relevant for this paper is 'social services' (category (a) above). Expenditure on social services is often referred to as 'social sector expenditure' and includes the following sub-categories: education, sports, arts and culture; medical and public health; family welfare; water supply and sanitation; housing; urban development; welfare of scheduled castes (SCs), scheduled tribes (STs), and other backward castes (OBCs); labour and labour welfare; government servants; social security & welfare; nutrition (expenditure relating to the nutrition program under child welfare); relief on account of natural calamities; others. Thus, public health expenditure falls under 'social services' and we measure it in the most expansive manner by including expenditure on medical and public health, family welfare and nutrition.

Expenditure on 'medical and public health' can be broken down, in turn, into the following sub-categories, with the figures in parenthesis referring to percentages

in 2012–2013: general (4.52%); public health (12.43%); medical education, training & research (13.2%); rural health services–other systems of medicine (3.03%); rural health services–allopathy (22.3%); urban health services–allopathy (39.26%); urban health services–other systems of medicine (5.26%) (NHAC, 2012).

In a similar manner, the components of expenditure on 'family welfare' are: rural family welfare services (45.5%); urban family welfare services (3.28%); maternity & child health (11.46%); assistance to local bodies (10.35%); training (6.84%); direction and administration (5.49%). As before, the figures in parenthesis refer to the percentage in 2012–2013 (NHAC, 2012).

Expenditure on 'nutrition' is mainly incurred on account of the Integrated Child Development Scheme (ICDS). This program includes supplementary nutrition, immunization, health check-up, referral services, pre-school non-formal education, nutrition & health education. The ICDS is a centrally-sponsored scheme implemented by State governments. For details, see `http://wcd.nic.in/icds.htm`

Political Competition

The index of effective number of parties and the index of the ideology of a state government is constructed from state-level election data available from the website of the Election Commission of India.For details, see `http://eci.nic.in/eci/eci.html`

Other Covariates

The controls in the model are the following: per capita real income, population sex ratio, women's education, and urbanization. Our measure of per capita real income

at the state level is per capita real net state domestic product (NSDP). The source for data on per capita NSDP (at 2004-2005 prices) is the *Handbook of Statistics on Indian Economy*, an annual publication of the Reserve Bank of India. We use the sex ratio (females per 1000 males) as a proxy for the strength of patriarchal attitudes; data on state-level sex ratios come from various issues of the *Economic Survey*. For details, see `http://indiabudget.nic.in/survey.asp` We measure women's education by the (adult) female literacy rate (percentage of females aged 15 and above who can read, write and carry out simple arithmetic calculations), and use data from the Open Government Data Platform India. Data on the instruments, state-level own tax and non-tax revenue, is downloaded from IndiaStat.[12]

[12]For details, see `http://www.indiastat.com/default.aspx`

Figure 3.1. Time series plot of public health expenditure (% of gross domestic product) for major regions of the world and India, 1995-2013. Source: World Development Indicators, World Bank.

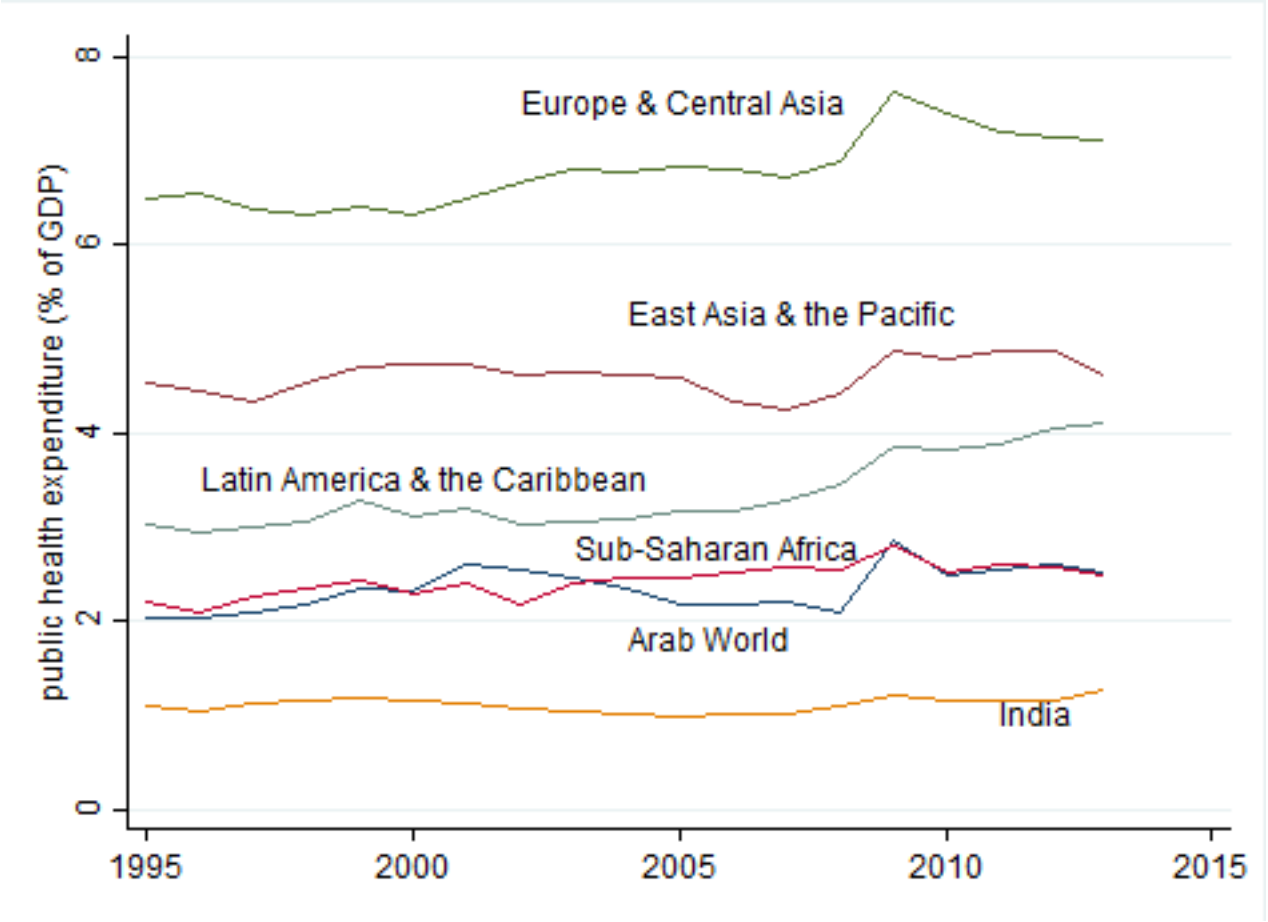

Figure 3.2. Time series plot of public health expenditure (% of total health expenditure) for major regions of the world and India, 1995-2013. Source: World Development Indicators, World Bank.

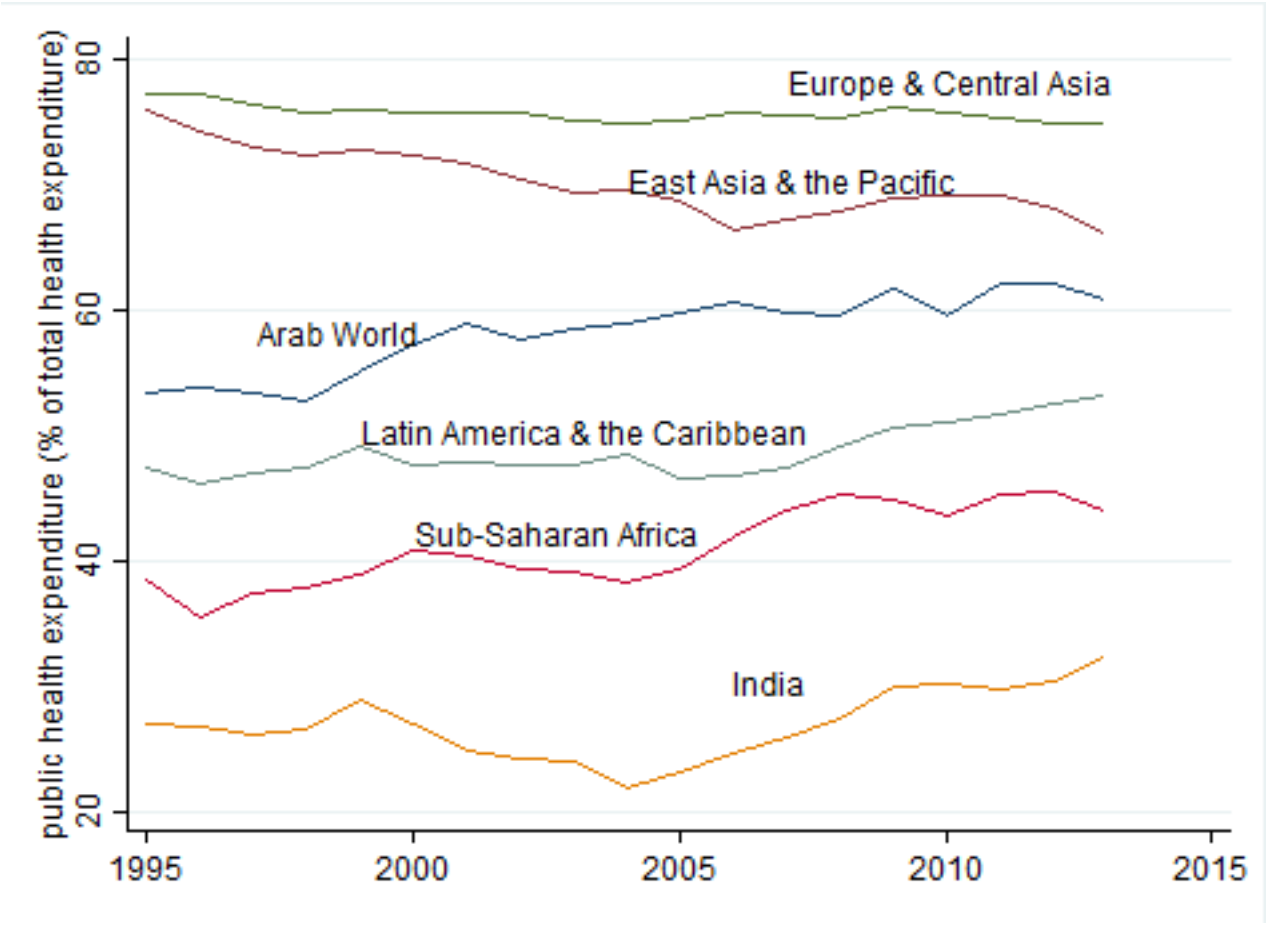

Figure 3.3. Scatter plot of public health expenditure (% of state-level net domestic product) and infant mortality rate (IMR) across India states, 1981-2012, with a linear regression line. Source: Authors' calculations.

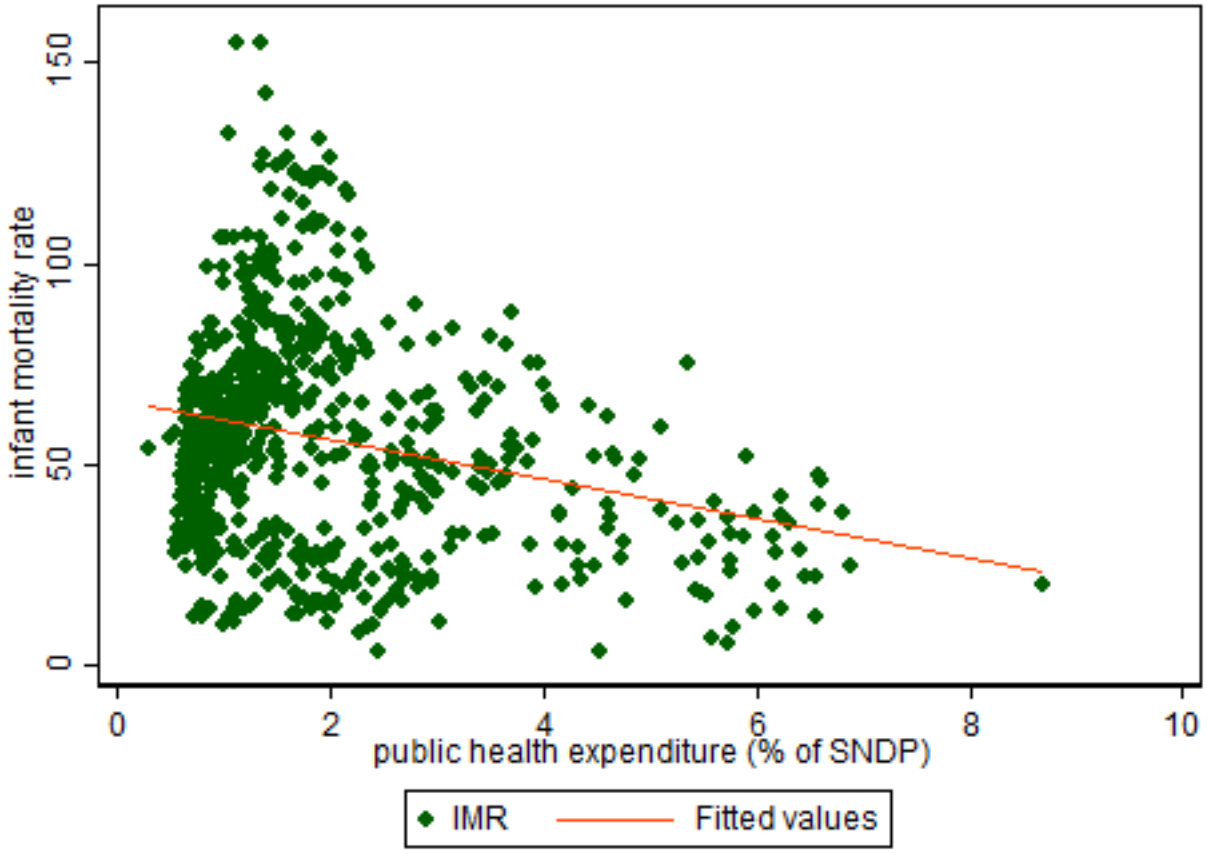

Table 3.1. Descriptive Statistics

	1985–1986	**1990–1991**	**1995–1996**	**2000–2001**	**2005–2006**	**2010–2011**
IMR	91.18	58.01	58.58	49.98	41.78	36.35
	(27.15)	(28.04)	(21.95)	(22.03)	(18.11)	(13.93)
PHE (% of SNDP)	2.04	2.84	2.19	2.19	1.74	1.85
	(1.18)	(2.07)	(1.30)	(1.54)	(1.47)	(1.62)
PCPHE	2.57	3.94	3.67	5.12	6.51	9.60
	(1.73)	(3.36)	(2.72)	(4.09)	(8.99)	(11.79)
Female Literacy	42.12	47.51	53.40	59.64	65.36	70.96
	(17.30)	(17.43)	(15.72)	(14.07)	(12.30)	(10.76)
Log PCNSDP	9.55	9.74	9.88	10.07	10.29	10.66
	(0.43)	(0.41)	(0.43)	(0.47)	(0.50)	(0.52)
Sex Ratio	922.12	921.19	919.31	917.20	920.80	925.83
	(62.22)	(55.04)	(54.48)	(65.37)	(71.45)	(76.38)
Urbanization	0.32	0.29	0.28	0.25	0.24	0.35
	(0.25)	(0.21)	(0.17)	(0.13)	(0.14)	(0.19)
Own Tax Revenue	0.06	0.07	0.06	0.06	0.05	0.05
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Own Non-Tax Revenue	0.03	0.03	0.06	0.07	0.04	0.03
	(0.02)	(0.02)	(0.14)	(0.24)	(0.11)	(0.04)
Parties in Govt	2.13	2.37	2.36	2.60	2.94	2.60
	(1.06)	(0.89)	(0.77)	(1.11)	(1.49)	(0.88)
Observations	31	31	31	31	31	31

This table reports mean and standard deviations (in parentheses below the mean) for key variables for a selected number of years. IMR = infant mortality rate (deaths of infants under one year old per 1000 live births); PHE (% of SNDP) = public health expenditure, that is, expenditure on medical & public health, water & sanitation, and nutrition, measured as a percentage of the state-level NDP; PCPHE = per capita real PHE (at $2004-2005$ prices); Female Literacy = proportion of women aged 15 years and more who can read, write and carry out simple arithmetic calculations; PCNSDP = per capita net state domestic product (at $2004-2005$ prices); Sex Ratio = females per 1000 males; Urbanization = proportion of population living in urban areas; Own tax revenue = total tax revenue less the tax revenue transferred by the central government (measured as % of state NDP); Own non-tax revenue = total non-tax revenue less the amount transferred from the central government (measured as % of state NDP); Parties in the government = index of the effective number of political parties in the state government (a proxy for the degree of political competition).

Table 3.2. Basic Regression Results

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
PHE	-1.75	-7.17*	-8.33**	-6.85**	-6.92**	-7.19**	-7.20**
	(0.198)	(0.089)	(0.033)	(0.044)	(0.039)	(0.030)	(0.030)
Parties in Govt		-2.52**	-2.50**	-2.11**	-2.04*	-1.84*	-1.84*
		(0.035)	(0.049)	(0.039)	(0.071)	(0.086)	(0.086)
Log PCNSDP			-2.69	-2.51	-2.50	-2.04	-2.04
			(0.387)	(0.397)	(0.399)	(0.461)	(0.461)
Female Literacy				-0.86***	-0.85***	-0.89***	-0.89***
				(0.000)	(0.001)	(0.000)	(0.000)
Sex Ratio					0.03	0.04	0.04
					(0.847)	(0.737)	(0.737)
Urbanization						-28.43***	-28.44***
						(0.007)	(0.007)
State Fixed Effects	Y	Y	Y	Y	Y	Y	Y
Year Effects	Y	Y	Y	Y	Y	Y	Y
KP (F-stat)		242.612	207.323	197.124	182.776	190.297	190.297
P-value (J-Stat)		0.709	0.553	0.827	0.836	0.895	0.895
N	677	548	540	532	532	532	532

The dependent variable in all regressions is the state-level infant mortality rate. PHE (% of SNDP) = expenditure on medical & public health, water & sanitation, and nutrition (% of state NDP); Parties in Govt = effective number of parties in the government; Female Literacy = proportion of women aged 15 years and more who can read, write and carry out simple arithmetic calculations; PCNSDP = per capita net state domestic product (at 2004-2005 prices); Sex Ratio = females per 1000 males; Urbanization = proportion of population living in urban areas. P-values (clustered by state) appear in parenthesis below estimates; *p<0.10, **p<0.05, ***p<0.01. Specification 1 is estimated by OLS. For all other specifications public health expenditure has been instrumented with own tax revenue, and own non-tax revenue. Models 1 through 6 have been estimated by 2SLS; model 7 has been estimated by LIML. The KP (F-stat) refers to the Kleibergen-Paap rk Wald statistic for weak identification; the J-stat refers to Hansen's overidentification test.

Table 3.3. Reduced Form and First Stage Estimates

	(1)	(2)
Own Tax Revenue	-46.68	8.27*
	(0.701)	(0.055)
Own Non-Tax Revenue	-14.72**	1.84***
	(0.017)	(0.000)
Parties in Govt	-1.87*	0.00
	(0.076)	(0.854)
Female Literacy	-0.85***	-0.01
	(0.002)	(0.466)
Log PCNSDP	-1.78	-0.02
	(0.435)	(0.924)
Sex Ratio	0.05	-0.00
	(0.712)	(0.773)
Urbanization	-21.59*	-0.94*
	(0.076)	(0.068)
State Fixed Effects	Y	Y
Year Fixed Effects	Y	Y
Observations	532	532

Specifications (1) and (2) refer to the reduced form and first stage regressions, respectively, for the model referred in specification (6) in Table 3.2. All variables as defined in Table 3.2. P-values (clustered by state) in parenthesis; *p<0.10, **p<0.05, ***p<0.01.

Table 3.4. Regression Results: Robustness Checks

	(1)	(2)	(3)	(4)	(5)	(6)
PHE	-7.19**	-7.19**	-7.84	-7.40**	-12.29**	-7.04**
	(0.030)	(0.030)	(0.158)	(0.029)	(0.010)	(0.027)
Parties in Govt	-1.84*	-1.83*	-1.73	-1.71	-1.48*	-1.75
	(0.086)	(0.092)	(0.132)	(0.110)	(0.097)	(0.104)
Female Literacy	-0.89***	-0.90***	-0.79***	-0.91***	-0.87***	-0.74***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.004)
Log PCNSDP	-2.04	-2.10	2.72	-1.71	-1.41	-3.48
	(0.461)	(0.464)	(0.777)	(0.543)	(0.543)	(0.201)
Sex Ratio	0.04	0.04	0.03	0.02	0.07	0.00
	(0.737)	(0.735)	(0.796)	(0.871)	(0.471)	(0.977)
Urbanization	-28.43***	-28.86***	-34.08***	-27.83***	-25.75**	-34.42***
	(0.007)	(0.008)	(0.000)	(0.009)	(0.026)	(0.000)
State Fixed Effects	Y	Y	Y	Y	Y	Y
Year Effects	Y	Y	Y	Y	Y	Y
Log SPHE	N	Y	N	N	N	N
Log NPHE	N	N	Y	N	N	N
Index of Ideology of Govt.	N	N	N	Y	N	N
Pub Health Exp (2 Lags)	N	N	N	N	Y	N
South India Time Trend	N	N	N	N	N	Y
KP (F-stat)	190.297	189.882	14.498	182.647	49.582	218.232
P-value (J-Stat)	0.895	0.893	0.336	0.936	0.947	0.823
N	532	532	507	532	497	532

The dependent variable in all regressions is the state-level infant mortality rate. SPHE = total state-level social sector expenditure less public health expenditure; NPHE = total state expenditure less public health expenditure; all other variables are as defined in Table 3.2. P-values (clustered by state) appear in parenthesis below estimates; *p<0.10, **p<0.05, ***p<0.01. For all specifications, public health expenditure is instrumented with own tax revenue, and own non-tax revenue. The models have been estimated by 2SLS.

Table 3.5. Regression Results with Three Definitions of Public Health Expenditure and in 15 Major States

	All States			15 Major States		
	(1)	(2)	(3)	(4)	(5)	(6)
PHE1	-10.84*			-28.25		
	(0.099)			(0.275)		
Parties in Govt	-1.90*	-1.79*	-1.84*	-1.85*	-1.61	-1.73
	(0.059)	(0.083)	(0.086)	(0.079)	(0.154)	(0.203)
Female Literacy	-0.96***	-0.94***	-0.89***	-1.18***	-1.15***	-1.01***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.004)	(0.006)
Log PCNSDP	-2.78	-2.09	-2.04	-6.54	-5.94	-8.98
	(0.245)	(0.451)	(0.461)	(0.208)	(0.397)	(0.632)
Sex Ratio	0.04	0.04	0.04	-0.11	-0.11	-0.14
	(0.756)	(0.721)	(0.737)	(0.433)	(0.465)	(0.558)
Urbanization	-30.29***	-29.53***	-28.43***	-52.95*	-43.78	-59.53
	(0.008)	(0.008)	(0.007)	(0.099)	(0.316)	(0.594)
PHE2		-7.93*			-16.91	
		(0.083)			(0.544)	
PHE3			-7.19**			-25.97
			(0.030)			(0.711)
State Fixed Effects	Y	Y	Y	Y	Y	Y
Year Effects	Y	Y	Y	Y	Y	Y
KP (F-stat)	118.302	91.882	190.297	2.336	1.832	0.247
P-value (J-Stat)	0.661	0.756	0.895	0.585	0.258	0.215
N	532	532	532	415	415	415

The dependent variable in all regressions is the state-level infant mortality rate. PHE1 = expenditure on medical & public health (% of state NDP); PHE2 = PHE1 + expenditure on family welfare (% of state NDP); PHE3 = PHE2 + expenditure on nutrition (% of state NDP); all other variables are as defined in Table 3.2. P-values (clustered by state) appear in parenthesis below estimates; *p<0.10, **p<0.05, ***p<0.01. For all specifications, public health expenditure is instrumented with own tax revenue, and own non-tax revenue. The models have been estimated by 2SLS.

Table 3.6. Regression Results with Key Independent Variable as Log of per capita Real Public Health Expenditure

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log PCPHE	-38.35***	-19.64***	-14.04***	-14.03***	-15.74***	-16.79***	-3.48
	(0.000)	(0.002)	(0.004)	(0.004)	(0.010)	(0.008)	(0.821)
Parties in Govt	-12.87***	-10.83***	-6.17***	-6.18***	-6.36***	-6.28***	-1.96*
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.056)
Log PCNSDP		-17.83***	0.07	0.03	2.97	6.68	1.77
		(0.006)	(0.989)	(0.995)	(0.675)	(0.502)	(0.895)
Female Literacy			-0.89***	-0.89***	-0.88***	-0.81***	-0.84***
			(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Sex Ratio				-0.00	0.00	-0.00	0.04
				(0.992)	(0.983)	(0.989)	(0.743)
Urbanization					-20.78	-29.95	-23.00*
					(0.351)	(0.210)	(0.099)
State Fixed Effects	N	N	N	N	N	N	Y
Year Effects	N	N	N	N	N	Y	Y
KP (F-stat)	19.112	10.076	8.898	7.711	6.995	5.645	4.594
P-value (J-Stat)	0.019	0.804	0.689	0.667	0.587	0.615	0.064
N	541	541	533	533	533	533	533

The dependent variable in all regressions is the state-level infant mortality rate. Log PCPHE = logarithm of per capita expenditure on medical & public health, family welfare, and nutrition deflated by the state-level NDP deflator; all other variables as defined in Table 3.2. P-values (clustered by state) appear in parenthesis below estimates; *p<0.10, **p<0.05, ***p<0.01. For all specifications, public health expenditure is instrumented with own tax revenue, and own non-tax revenue. The models have been estimated by 2SLS.

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