

Childhood Health and Prenatal Exposure to Seasonal Food Scarcity in Ethiopia*

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Abstract

This paper analyzes the impact of prenatal exposure to seasonal food scarcity on childhood health in Ethiopia. I construct a novel measure of seasonal exposure based on reported months of relative food scarcity in the local community. I find that exposure has a significant negative impact on height by age five that strengthens by age eight. Effects decrease with household wealth and maternal education and are stronger during the first trimester of gestation. In contrast to height, effects on child body mass are only identified closer to birth and when exposure is concentrated in the second trimester. Overall, results highlight that in addition to the effects of severe famine conditions identified in many studies, more typical variation in prenatal food availability can have lasting impacts on health in the developing world.

JEL classification: I12, O13, O15

Keywords: health, nutrition, Ethiopia, child development, prenatal

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1 Introduction

An estimated 805 million people globally—roughly one out of every nine—still suffer from chronic undernourishment (FAO 2014). In many of the poorest developing countries, including Ethiopia, estimates are more than one out of every three. Despite a long history of attempts to address food security, an estimated 40% of households in Ethiopia are still classified as food energy deficient by the World Food Programme (2014). A heavy reliance on small-scale rainfed agriculture combined with highly localized agricultural markets make Ethiopia’s erratic climatic conditions a significant source of food uncertainty. Moreover, even in years of fairly typical seasonal patterns of cultivation, lack of storage capacity and costly transport can lead to measurable differences in food availability over the agricultural cycle (FAO 2004; WFP 2014). While seasonal effects on nutritional intake may be mild in comparison to more extreme weather phenomenon (i.e. drought, flood, monsoon), they could still have a substantial impact among vulnerable groups of the population. In this paper I focus on one such group—unborn children.

There is growing support in the biomedical literature for the hypothesis that poor maternal nutrition during pregnancy can lead to permanent fetal adaptations that affect health throughout a child’s life (Gluckman and Hanson 2005). This “fetal origins” hypothesis has recently garnered interest among economists, who have attempted to establish and quantify the casual impact of such a mechanism.¹ These studies have often used uncommon and arguably exogenous events such as famine or disease epidemic to identify the causal effects of prenatal nutritional environment. While many find significant effects from such environmental shocks, less is known about the magnitude of effects due to more normal variations in food availability.

In this paper, I use a unique longitudinal data set to examine the effects of prenatal exposure to seasonal variations in food scarcity on childhood health in Ethiopia. My exposure measure is derived by combining individual date of birth with survey data collected shortly after birth at the local community level. Importantly, the survey contains explicit data on months when food becomes harder to obtain or more expensive within each community. Identification relies on the assumption that prenatal exposure to reported months of food scarcity, conditional on community and month of birth fixed effects, is uncorrelated with any unobserved determinants of examined child health outcomes. Under this assumption, I am able to identify the impact of in-utero exposure to reported seasonal food scarcity on health outcomes measured at age one, five, and eight for a cohort of Ethiopian children born between May 2001 and May 2002. My main finding is that exposure has a significant negative impact on height by age five that strengthens by age eight. Moreover, effects decrease with household wealth and maternal education and are stronger when exposure occurs during the first trimester of gestation. In contrast, effects on child body mass are only identified at age one and

¹For a review of the literature, see Currie (2009); Almond and Currie (2011); Currie and Almond (2011); Currie and Vogl (2013).

when exposure is concentrated in the second trimester.

There are many studies of developed countries that have found month or season of birth to be robustly correlated with health outcomes such as birthweight, life expectancy, and height.² A smaller but growing body of literature has established similar patterns in the developing world. For example, using a sample of Indian children under three years of age, Lokshin and Radyakin (2012) find those born during monsoon months are significantly shorter than children born in fall or winter months. In Gambia, Rayco-Solon et al. (2005) show the incidence of small-for-gestational age was higher among children born at the end of the “hungry season,” while the peak incidence of preterm births paralleled increases in agricultural labor demand and malaria infections. Moore et al. (2004) show that birth during the hungry season resulted in increased infant mortality rates in both Gambia and Bangladesh. The association between season of birth and health outcomes also appears to persist into adulthood in the developing world. For example, McEniry and Palloni (2010) find in-utero exposure to the hungry season was associated with higher probabilities of heart disease and diabetes among a sample of older Puerto Ricans. Researchers have most commonly argued that prevalence of disease, seasonal maternal labor supply, or nutritional intake associated with agricultural output are likely channels through which calendar time of birth may affect health outcomes in the developing world. While much of the season of birth literature is suggestive that the timing of birth in relation to the agricultural cycle is important, it is difficult to disentangle prenatal nutritional effects from exposure to disease or other seasonal factors.

There is a growing body of related economic literature that examines the effects of early exposure to localized weather shocks. These studies have most commonly relied on changes in annual rainfall or ambient temperature patterns as exogenous sources of variation. While this literature arguably implements stronger identification strategies than the season of birth literature, it still has trouble isolating the relevant mechanisms at work. Maccini and Yang (2009), for example, find higher rainfall in early life is associated with better health outcomes for Indonesian women, but not for men. They attribute the positive association to the influence of rainfall on increased agricultural output and lower food prices. Rocha and Soares (2015), on the other hand, attribute a positive relationship between rainfall and birth outcomes in Brazil to increased access to safe drinking water and consequently lower prevalence of disease. Still other studies find increased rainfall early in life has negative consequences for later outcomes (e.g. Kim 2010; Aguilar and Vicarelli 2011). Such negative correlations are generally attributed to the disease environment, increased maternal labor supply, or a negative impact of excessive rain on agricultural production. In Africa, for example, Kudamatsu et al. (2014) find that increased rainfall can negatively affect infant mortality in regions with epidemic malaria while drought shocks can have a negative impact in arid areas. Similarly for Mexico, Skoufias et al. (2011) find that weather shocks associated with

²See, for example, Doblhammer and Vaupel (2001); Kihlbom and Johansson (2004); Tanaka et al. (2007); Strand et al. (2011).

rainfall and temperature can have substantial negative as well as positive effects that vary across geographic regions.

In this paper, I make strides towards isolating the impact of seasonal changes in prenatal nutrition on health outcomes by using a treatment measure that is both localized and explicitly based on exposure to food scarcity. A complication with using environmental shocks for identification is the presence of multiple channels through which weather changes have been argued to effect health outcomes. Moreover, even when weather patterns are convincingly linked to changes in agricultural production, it is not inherently clear how effects travel through the supply chain and ultimately impact food availability and/or prices. To my knowledge, this is the first study to use an explicit measure of food scarcity to circumvent the ambiguity surrounding the use of environmental shocks, such as rainfall or temperature, as instruments for nutritional deprivation. This is also the first study to use a localized instrument to examine how season of gestation impacts later health outcomes. Localization of the measure allows me to control for seasonal trends that occur at the country level but are unrelated to food availability. Finally, beyond improved isolation of prenatal nutritional environment, having health outcomes collected repeatedly over a period of rapid physical growth adds novel evidence on gestation being a “sensitive” period of development. Specifically, the pattern of effects across time shed light on how easy or hard it is to make up for poor nutrition during early development with increased health inputs at later stages.

Consistent with the view of gestation as a “sensitive” period of development, I find that exposure has a significant negative impact on child height that strengthens as children age. Specifically, I estimate that an additional month of prenatal exposure to reported seasonal food scarcity decreases height by at least 0.31 cm by age five and 0.42 cm by age eight. The magnitude of these effects are similar to a one-third standard deviation decrease in the household wealth index used in my benchmark specification. As an outside comparison, Dercon and Porter (2014) estimate that infant exposure to the 1984 Ethiopian famine decreased height at least 5 cm by early adulthood and was accompanied by an estimated annual income loss of 5%.³ I also find that effects decrease with household wealth and maternal education and are stronger during the first trimester of gestation. In contrast to height, effects on child body mass are statistically significant only at age one for exposure during the second trimester. This is consistent with height and weight being measures of health variation in the long-run and short-run, respectively. I find no evidence that seasonal variation of water quality or maternal labor supply are driving results, suggesting that I am indeed capturing the effects of seasonal changes in nutritional intake on child health outcomes. Likewise, I find no evidence that results are driven by seasonal fertility patterns within local communities or selective mortality of children on the basis of exposure to seasonal food

³Dercon and Porter (2014) estimated a significant impact only on those aged 12-36 months during the peak of the famine. Effects are not identify for children in-utero, although the authors cannot rule out that potentially severe mortality selection dominates scarring for this group.

scarcity. Overall, my results highlight that in addition to the effects of severe famine conditions identified in many studies, more typical variation in prenatal nutritional environment can have lasting impacts on health in the developing world.

The remainder of this paper is presented as follows. Section 2 begins by discussing the data and construction of the prenatal exposure measure. Section 3 describes the empirical strategy, and Section 4 presents the results. Section 5 assesses potential alternate channels of correlation including seasonal patterns of fertility, water quality, seasonal labor supply, and selective mortality. Finally, Section 6 concludes.

2 Data

I use unique data from the Young Lives Study (YLS) to conduct an empirical analysis of the impact of prenatal exposure to relative food scarcity on later child health outcomes in Ethiopia. The YLS conducted surveys for a cohort of 2,000 children born between May 2001 and May 2002 in twenty sites across the country. Currently data is available from three rounds of surveys conducted in 2002, 2006, and 2009—when children were approximately one, five, and eight years old. The study collects detailed information on household and child characteristics, including anthropometric markers such as height and weight. In addition, a community level survey was conducted during the first wave of data collection, when children were 6-18 months old. These data were obtained on a variety of topics through interviews with key community leaders such as government officials, municipal leaders, and village headmen.

2.1 A Measure of Prenatal Exposure to Seasonal Food Scarcity

Regional agroecosystems across Ethiopia are quite diverse, particularly in terms of rainfall and elevation. This can result in substantial variations in crop yield patterns across geographic regions. Moreover, agricultural markets in Ethiopia consist primarily of small farmers and traders who produce and sell product in local markets. According to a 2004 report from the Food and Agriculture Organization of the United Nations, “these [local] markets function in relative isolation and grain movements from surplus to deficit areas are constrained by high transport costs due to poor road infrastructure, weak market information systems, and a quasi monopoly in the transportation sector.” The combination of a limited agricultural market and varied agroclimatic conditions results in meaningful variations in food availability patterns across geographic regions of Ethiopia.

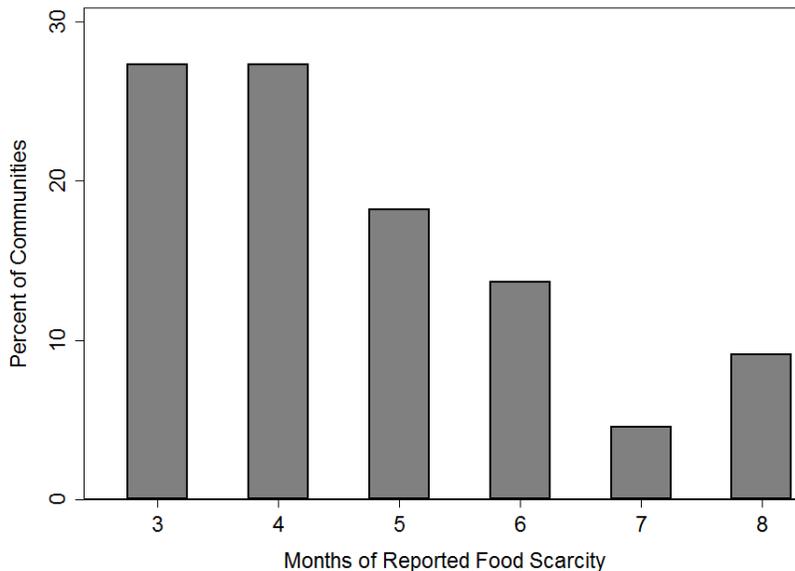
In light of these geographic diversities, I use relevant data collected at the local community level to construct my measure of prenatal exposure to seasonal food scarcity. A community survey was conducted at each of the sites selected to participate in the YLS. While poor and food-poor areas were oversampled by the study, the communities span Ethiopia geographically, and are contained in the regions where almost 97% of the population reside. Specifically, communities were sampled from the capital city of

Addis Ababa and the regional states of Amhara, Oromia, Tigray, and the Southern Nations, Nationalities, and Peoples' Region (SNNPR).

My exposure measure is constructed on the basis of the following community survey question:

In which months of the year does food become harder to obtain / more expensive?

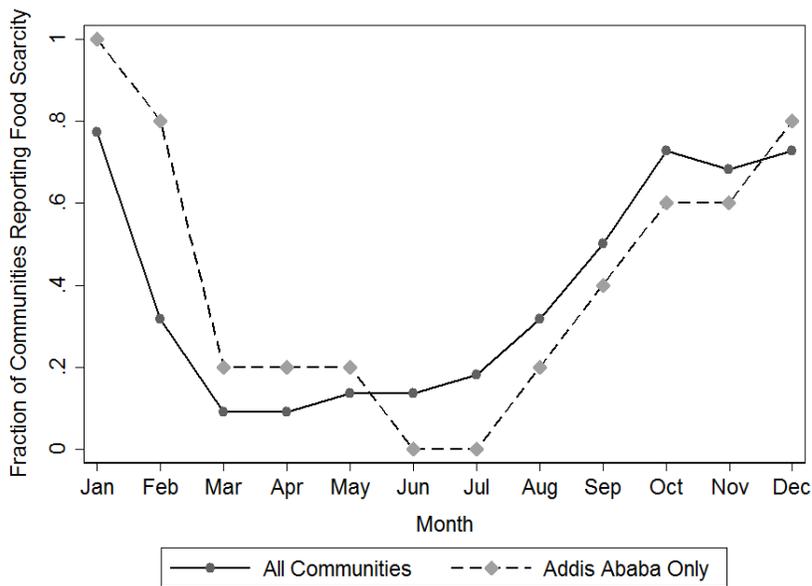
Data collectors recorded responses to this question by ticking 'yes' or 'no' for each month of the year. I use the survey responses for 22 of the 23 local communities, with the last community excluded from analysis due to missing food scarcity data. On average, non-missing communities reported just over 4.5 months of relative food scarcity, with a range of three to eight months (see Figure 1). Figure 2 shows the percent of all communities reporting food scarcity by calendar month. In anticipation of later analysis, data is also reported for the subset of five communities in or around the Ethiopian capital of Addis Ababa. On aggregate, more communities reported relative scarcity from October to January while less reported scarcity from March to June. This corresponds to a reported average increase in food availability following what is considered Ethiopia's main harvest season, which typically runs from October to February.



Source: Author's calculations using data from Young Lives Study, Ethiopia.

Figure 1: Number of Reported Months of Seasonal Food Scarcity

The community surveys were conducted during the last few months of 2002, shortly after the youngest children in the cohort of interest were born. While it was not explicitly specified that respondents answer the food scarcity question in relation to



Source: Author's calculations using data from Young Lives Study, Ethiopia.

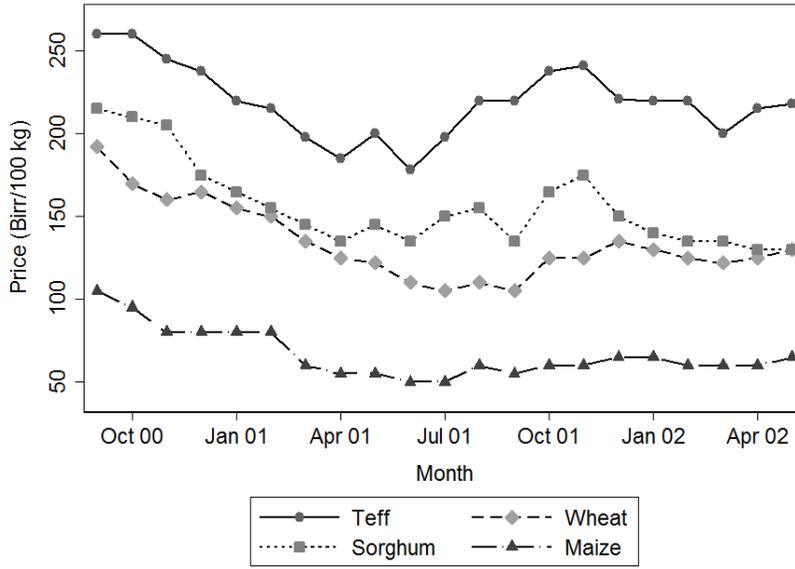
Figure 2: Reported Seasonal Food Scarcity by Calendar Month

the most recent year or years, responses are consistent with available food pricing data while sample children were in-utero. Figure 3 shows the monthly price in Addis Ababa of the four major grains harvested in Ethiopia from September 2000 to May 2002. As sample children were born between May 2001 and May 2002, the examined time frame completely spans when the children were in-utero. The observed pattern of prices over the time frame is generally consistent with the data on relative food scarcity reported by the Addis Ababa survey communities shown in Figure 2—higher prices from September to February and lower from March to August. Thus, the pricing data provides collaborative evidence that responses from the community survey measure seasonal food scarcity while sample children were in-utero.

I combine the community level food scarcity data with individual date of birth to compute the days of prenatal exposure to relative food scarcity for each child. Conception for a full-term birth is estimated precisely 270 days prior to birth allowing division of the gestational period into three trimesters of 90 days each. For preterm births, gestation length and corresponding exposure days are adjusted based on the reported number of weeks premature.⁴ The density of the exposure measure is shown in Figure 4. On average, children were exposed to an estimated 103 days of reported food scarcity in-utero, with a standard deviation of 49 days.

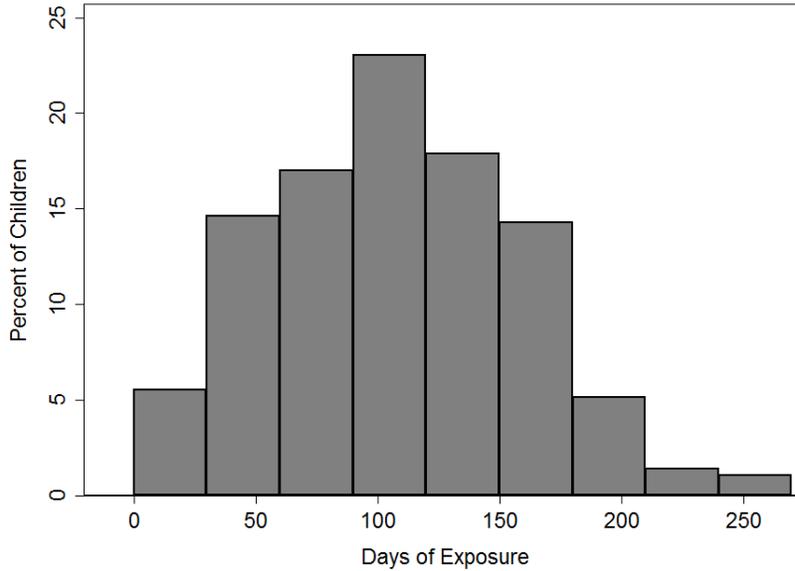
As later detailed, a possible concern for identification of exposure effects is a systematic correlation between the exposure measure and seasonal patterns of fertility

⁴8% of all births were reported as preterm. Of these, 75% reported the number of weeks premature. To construct exposure for the remaining 25%, I assign them the median of reported weeks premature (two weeks).



Source: FAO (2004).

Figure 3: Monthly Average Prices of Main Cereals, Addis Ababa



Source: Author's calculations using data from Young Lives Study, Ethiopia. Histogram with 30 day window for each bin.

Figure 4: Prenatal Days Exposed to Reported Seasonal Food Scarcity

within communities. However, it is important to note that the pattern of food scarcity summarized in Figure 2 is somewhat uncharacteristic for Ethiopia due to above average harvest seasons in both 2000 and 2001. Specifically, there were atypically low

food prices following harvest season (from March to July) and a delay in the start of pre-harvest peak food prices (from beginning in July to beginning in October). These atypical seasonal patterns combine with randomness in individual date of conception to provide exogenous variation in prenatal exposure to food scarcity that is far less extreme than weather phenomenon such as floods or droughts, yet more direct than rainfall or ambient temperature patterns. As such, the exposure measure sheds light on the magnitude of effects from explicit variation in food availability that is much similar in scale to typical seasonal fluctuations in Ethiopia and much of the developing world.

2.2 Health Outcomes and Other Data

In my empirical analysis I focus on two child health outcomes—height and body mass index.⁵ Height captures a child’s restricted growth potential associated with the chronic or long-term effects of malnourishment. In contrast, body mass is more sensitive to short-term health changes as it captures weight loss associated with acute undernutrition. Outcomes were measured at each round of data collection—when children were approximately one, five, and eight years old. Repeated observations during this period of rapid physical development is one of the benefits of the YLS data. Specifically, the pattern of effects across time can shed light on how easy or hard it is to make up for poor nutrition during early development with increased health inputs at later stages. Panel A of Table 1 gives descriptive statistics for the outcomes at each age. For ease of interpretation, I express height in centimeters and I standardize the body mass index to have mean zero and variance one. By either measure, malnutrition is quite severe among the sample population of children—stunting and wasting prevalence were both over 20% by age eight.⁶

In addition to prenatal exposure to reported food scarcity, a number of household and child characteristics are used in the empirical analysis to help control for demographic and socioeconomic effects on child health outcomes. These include gender, number of older siblings, a household wealth index, mother’s height, mother’s education, child ethnicity, and weeks premature at birth.⁷ In a robustness analysis, I also make use of other YLS data in attempt to evaluate alternate mechanisms that may be driving results. First, I use individual level data on the household’s main source of drinking water. Reported responses were grouped into four sources—unprotected (e.g. river, pond, unprotected well), piped directly into private dwelling/yard, public stand-pipe/tubewell, or other source. Second, I use community survey data to construct a

⁵Body mass index is calculated as weight (kg) divided by the square of height (m).

⁶Prevalence of child stunting is the percentage of children whose height-for-age is more than two standard deviations below the median for the international reference population. Wasting is defined analogously using weight-for-height.

⁷YLS provides a constructed household wealth index for each of the three survey rounds. After taking the log and standardizing each to have mean zero and variance one, I use the standardized average across non-missing values as my household wealth index.

measure of prenatal exposure to seasonal increases in local labor demand. Specifically, I use responses to the following survey question:

In which months of the year is there relatively more work to do?

With this data, I construct the new exposure measure in an analogous fashion as my measure of prenatal exposure to seasonal food scarcity. Descriptive statistics for household and child characteristics are presented in Panel B of Table 1.

Table 1: Descriptive Statistics: Health Outcomes and Other Data

	Mean	S.D.	Obs.		Mean	S.D.	Obs.
<i>Panel A — Child Health Outcomes</i>							
Height (cm)				Body Mass Index (BMI)			
Age 1	70.8	5.48	1849	Age 1	0.0	1.00	1756
Age 5	103.7	5.38	1810	Age 5	0.0	1.00	1810
Age 8	120.4	6.58	1784	Age 8	0.0	1.00	1784
<i>Panel B — Household and Child Characteristics</i>							
Male	0.53	0.50	1899	Wealth Index	0.00	1.00	1899
Mother's Height (cm)	158.6	5.88	1675	Older Siblings	2.45	2.27	1899
Mother's Education				Weeks Premature			
None	0.60	0.49	1890	None	0.92	0.28	1899
1 to 4 years	0.17	0.37	1890	1 to 2 weeks	0.04	0.20	1899
5 to 8 years	0.16	0.36	1890	>2 weeks	0.02	0.14	1899
>8 years	0.08	0.27	1890	Unknown	0.02	0.15	1899
Child Ethnicity				Water Source			
Amhara	0.29	0.45	1899	Unprotected	0.44	0.50	1899
Gurage	0.08	0.27	1899	Private Pipe	0.11	0.31	1899
Oromo	0.22	0.42	1899	Public Standpipe/Well	0.40	0.49	1899
Tigrian	0.23	0.42	1899	Other	0.05	0.21	1899
Hadiya	0.05	0.21	1899	Prenatal Days Exposed to High Labor Demand	121.0	67.8	1899
Other	0.13	0.33	1899				

Source: Young Lives Study, Ethiopia, young cohort. Sample of children without missing community data on seasonal food scarcity (n = 1,899).

3 Empirical Strategy

Empirical analysis of the effects of prenatal exposure to seasonal food scarcity on child health outcomes is based on the following benchmark specification:

$$Y_{idc} = \alpha + \delta Exp_{dc} + \beta X_{idc} + \theta_c + \mu_m + u_{idc}, \quad (1)$$

where Y_{idc} is a health outcome for individual i , born on date d , in community c ; Exp_{dc} is my measure of prenatal exposure to seasonal food scarcity; X_{idc} are household and child characteristics; θ_c is a fixed effect for community of residence; μ_m is a fixed effect for month of birth; and u_{idc} is a random error term. I use equation-by-equation OLS to estimate the system separately for each child height and body mass outcome measured at approximately one, five, and eight years of age.⁸ The coefficient of interest is that on prenatal exposure to relative food scarcity δ . Note that the exposure measure varies by date of birth d and community of residence c . Throughout the analysis, exposure days are expressed in thirty day units so coefficients can be interpreted as approximate monthly effects. Household and child characteristics include age of the child in months when the outcome was measured, a household wealth index, number of older siblings, mother’s height, and dummies for gender, mother’s education, child ethnicity, and weeks premature at birth.⁹

Identification relies on the assumption that prenatal exposure to reported months of food scarcity is uncorrelated with any unobserved determinants of examined child health outcomes. It is clearly the case that community of residence is likely to be correlated with both health outcomes and the exposure measure, as climatic conditions and household demographics vary considerably across Ethiopia. However, the inclusion of community dummy variables ensures that effects associated with geographic area are controlled for. An additional concern is the existence of unobserved socioeconomic or demographic determinants of child health that correlate with seasonal patterns of fertility, and hence the exposure measure. However, the inclusion of month of birth dummies controls for seasonal effects that occur at the country level and are not related to exposure to food scarcity.¹⁰ It is still conceivable that seasonal fertility patterns could correlate with unobserved characteristics within communities. However, as detailed in later robustness analysis, I find no evidence of correlation between exposure and seasonal patterns of fertility based on the rich set of observable household characteristics available. Moreover, sample children were exposed to atypical seasonal patterns of food availability while in-utero, providing an exogenous and unexpected source of variation in addition to estimated date of conception.

⁸Allowing for correlation of the error terms across outcome equations by jointly estimating the system using seemingly unrelated regressions (SUR) produces very similar results as equation-by-equation OLS.

⁹Approximately 11.8% of individuals were missing data on mother’s height. These data were imputed using the MI multiple imputation package in Stata (2015). Dependent and independent variables in the benchmark specification were included in the imputation equation.

¹⁰Month of birth dummies also help control for nonlinear growth in children that may not be captured by the continuous age of child control.

Even under the identifying assumption, interpretation of the coefficient of interest δ requires careful consideration. Due to collinearity, δ is a measure of the effect of exposure during the approximately nine months prior to birth relative to the first three months after birth (i.e. the first three months after birth is the reference period). However, imposing a relatively mild assumption can allow for further interpretation of the empirical estimates. Specifically, I assume that the effects of exposure are weakly negative regardless of whether exposure occurs during pregnancy or the months just after birth. Under this assumption, I interpret the empirical δ estimates as the minimum total effect of prenatal exposure to food scarcity according to the following proposition:

Proposition. *If the effects of exposure to relative food scarcity the year following conception are weakly negative during pregnancy and after birth, then δ is the minimum effect (in absolute terms) of prenatal exposure to food scarcity. Proof: see Appendix A.*

In addition to the benchmark specification, I examine heterogeneity of effects across a number of dimensions to shed further light on the relevant mechanisms at work. I analyze how effects vary over household wealth, maternal education, geographic region, and the timing of exposure over stages of gestation. Results are compared with previous findings in the medical and economic literatures. I also examine heterogeneity across sources of water supply and exposure to seasonal variation in work availability to assess if results may be partially operating through access to clean water or changes in maternal labor supply in congruence with food availability. Lastly, I discuss and evaluate the potential influence of selective mortality on the basis of prenatal exposure to seasonal food scarcity. This may be a particular concern because of the high infant mortality rates in Ethiopia.

4 Results

4.1 Benchmark Results

The main results from the benchmark specification are presented in Table 2. Under the assumptions specified in the previous section, I interpret reported coefficients as lower (absolute) bounds on the total effects of exposure to seasonal food scarcity on corresponding health outcomes. Panel A reports the estimated exposure effect on child height measured at approximately age one, five, and eight. Panel B gives analogous results for the standardized measure of body mass.

A lower bound on the effect of exposure on age one height is not identified at conventional significance levels under the benchmark specification. However, by age five, the estimated coefficient has greatly increased in magnitude and is statistically significant. The coefficient implies, holding other independent variables constant, an additional month of prenatal exposure to food scarcity leads to an estimated decrease in height of at least 0.31 cm. Moreover, the magnitude of the estimated effect increases to 0.42 cm by age eight. This pattern is consistent with a divergence of heights between

ages one and eight on the basis of prenatal exposure to food scarcity. This would imply that parents are unable and/or unwilling to fully make up for the early effects of exposure. This is consistent with the view of gestation as a “sensitive” period of child development in which nutritional and other inputs are difficult to substitute for in later stages of life (Cunha and Heckman 2007).

Table 2: Effects of Prenatal Exposure to Seasonal Food Scarcity on Child Health Outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
<i>Panel A — Height (cm)</i>						
	<i>Height₁</i>		<i>Height₅</i>		<i>Height₈</i>	
<i>Exp</i>	-0.085 (0.233)	-0.054 (0.214)	-0.316 (0.212)	-0.311* (0.177)	-0.460*** (0.160)	-0.421*** (0.127)
<i>Obs</i>	1,847	1,840	1,807	1,804	1,781	1,776
<i>R</i> ²	0.364	0.396	0.185	0.266	0.138	0.218
<i>Panel B — Body Mass Index</i>						
	<i>BMI₁</i>		<i>BMI₅</i>		<i>BMI₈</i>	
<i>Exp</i>	-0.030 (0.026)	-0.033 (0.029)	-0.015 (0.029)	-0.008 (0.028)	0.009 (0.022)	0.014 (0.021)
<i>Obs</i>	1,754	1,748	1,807	1,804	1,781	1,776
<i>R</i> ²	0.267	0.282	0.120	0.152	0.071	0.106
Controls (<i>X</i>)	No	Yes	No	Yes	No	Yes
Birth Month FE (μ)	Yes	Yes	Yes	Yes	Yes	Yes
Community FE (θ)	Yes	Yes	Yes	Yes	Yes	Yes

Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: height and body mass at age one, five, and eight. Reported independent variable: prenatal exposure to seasonal food scarcity (*Exp*). Additional independent controls (*X*): age of child in months, wealth index, number of older siblings, mother’s height, and dummies for gender, mother’s education, ethnicity, and weeks premature.

Panel B of Table 2 show that non-zero lower bounds on the effects of exposure on body mass index are not identified at any age. As body mass is essentially a measure of weight-for-height, these estimates are consistent with height and weight being measures of the variation of health inputs in the long-run and short-run, respectively. Specifically, prenatal exposure has a long-term impact on health as demonstrated by the effects on childhood height but, after controlling for height, has no significant impact on the short-term health measure (weight). Furthermore, the life-cycle impact of early malnourishment on body mass is not fully understood. In general, studies have found that poor early nutrition can lead to obesity later in life (Black et al. 2013). Such

a mechanism is also consistent with the weak effects on body mass as compared to height found in this paper. However, I will return to this discussion as further insights are revealed when examining how the timing of exposure during the gestational period affects health outcomes at alternate ages.

4.2 Socioeconomic and Geographic Heterogeneity

In order to gain further insight on the mechanism at work in my baseline specification, I next examine how the effects of prenatal exposure to seasonal food scarcity vary with socioeconomic status and geographic area. Specifically, I estimate heterogeneity in effects across household wealth, level of maternal education, and regional state of residence. As the benchmark specification identified no significant relationships between overall exposure and body mass, I limit my analysis here to child height outcomes.

4.2.1 Household Wealth

Panel A of Table 3 reports results when adding an interaction between the exposure measure and the household wealth index to the benchmark specification. Children in wealthier households were significantly less adversely affected by prenatal exposure to reported food scarcity. Moreover, the heterogeneity is quantitatively substantial. For example, the estimated coefficients imply the minimum decrease in height by age five from a month of exposure is 0.65 cm for a child from the tenth percentile of the wealth distribution compared to 0.09 cm from the ninetieth percentile. By age eight, the estimated magnitude of the effects has increased to 0.78 cm and 0.19 cm, respectively. These findings are consistent with the related empirical literature which generally find stronger long term effects of early health shocks on poor households (e.g. Currie and Hyson 1999). In the context of this study, results suggest that poor families may be particularly vulnerable to variation in food availability in Ethiopia.

There are several possible channels through which household wealth may influence the effects of exposure. First, wealthier families may be better equipped to smooth consumption during pregnancy over fluctuations in food prices or availability. As a result, children from these households may directly experience a smaller exposure shock in-utero. Alternatively, wealthier parents may have the financial means to make additional remedial investments after birth. In this instance, even if actual in-utero exposure is similar across wealth levels, exposed children from wealthier families may “catch up” to unexposed children through remedial health investments by the time the outcomes are measured. In either of these two cases, it is important to note that the reported results may underestimate the total costs of exposure as there may be additional utility costs due to the reallocation of resources in response to the shock. However, in contrast to the above channels which focus on responsive investments by parents, the relationship between wealth and exposure could partially be a mechanical feature of child health “production.” For example, many empirical and theoretical

Table 3: Heterogeneity of Effects by Wealth and Maternal Education

	<i>Height</i> ₁	<i>Height</i> ₅	<i>Height</i> ₈
	(1)	(2)	(3)
<i>Panel A — Heterogeneity by Household Wealth</i>			
<i>Exp</i>	-0.070 (0.231)	-0.344* (0.199)	-0.454*** (0.147)
<i>Exp</i> × <i>Wealth</i>	0.110 (0.451)	0.219*** (0.072)	0.233* (0.122)
<i>Panel B — Heterogeneity by Mother’s Education</i>			
<i>Exp</i>	-0.137 (0.226)	-0.408* (0.196)	-0.551*** (0.162)
<i>Exp</i> × <i>MomEd</i>			
1 to 4 years	0.086 (0.120)	0.127 (0.225)	0.126 (0.160)
5 to 8 years	0.291 (0.189)	0.126 (0.253)	0.297 (0.337)
>8 years	0.110 (0.233)	0.467* (0.237)	0.496* (0.255)
<i>Obs</i>	1,840	1,804	1,776

Robust standard errors (clustered at the community level) in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Dependent variable across columns: height at age one, five, and eight. Additional independent variables in all regressions: age of child in months, wealth index, number of older siblings, mother’s height, and dummies for gender, mother’s education, ethnicity, weeks premature, month of birth, and community.

studies argue that there are diminishing marginal returns to health investments. Thus, all else equal, children with higher baseline levels of investment would be less adversely affected by an equivalent decrease in health inputs. In the context of this study, it seems plausible that wealthier children could have higher baseline levels of prenatal health inputs compared to their less wealthy counterparts (e.g. wealthier mothers have more substantial or varied diets). If so, even if all mothers respond to exposure with equivalent level declines in health inputs, wealthier children could be less adversely affected by exposure.

4.2.2 Maternal Education

I next examine the heterogeneity of effects by level of maternal education based on four schooling categories—no schooling, lower primary (1-4 years), upper primary (5-8 years), and secondary plus (>8 years). Results are reported in Panel B of Table 3.

The estimated minimum effect of an additional month of exposure on a child born to a mother with no schooling (the reference group) is -0.41 cm by age five and -0.55 cm by age eight. Children of the most highly educated mothers were significantly less adversely affected by exposure. Interactions were also positive for mothers in the middle education groups, though not statistically significant. However, although not precisely estimated, there is a clear pattern of diminishing effects moving up the full set of maternal education indicators for height at age five and eight. To the extent that more highly educated mothers come from wealthier families, similar channels as detailed above could be partially explaining these results. However, it could also be the case that maternal cognitive capacity directly influences the effects of exposure. For example, more highly educated mothers may have partially shielded their fetus from the effects of external food scarcity. This would be consistent with much of the empirical evidence that maternal education strongly impacts child health outcomes through improved child-care practices and attitudes towards reproductive behavior (e.g. Thomas et al. 1991; Glewwe 1999).

4.2.3 Geographic Region

Lastly, I examine the heterogeneity of effects by geographic region. Table 4 shows the results when adding an interaction between exposure and regional dummy indicators to the benchmark specification. As the strongest effect of exposure occurred for children in the SNNPR, this region is set as the reference group. Compared to the SNNPR, only children from the agriculturally productive Amhara region were significantly less adversely effected by exposure at age five. By age eight, a positive interaction is also identified for Addis Ababa and nearby communities of Oromia. The only area that did not significantly differ from the SNNPR was the food deficit region of Tigray.

The SNNPR is the most culturally and ecologically diverse region in Ethiopia. However, much of the rural population are characterized by very small land holdings that are fully cultivated 2-3 times per year. This structure leaves many in the region particularly food vulnerable as households lack the capacity to alter future planting areas in response to unexpected seasonal fluctuations in crop yields. In contrast, Amhara is one Ethiopia's most agriculturally productive regions, resulting in less vulnerability to seasonal food availability. The region accounts for almost a third of the country's main grain harvest, often producing a regional surplus.

While Addis Ababa is densely populated, geographically small, and has relatively little agriculturally productive land, it is also the capital city and economic heart of the country. It features the most well-developed regional and international agricultural markets in Ethiopia, providing relative food stability over the agricultural cycle. In contrast, as the largest region in the country, Oromia features diverse ecologies and sub-regional crop yield patterns. However, the region also entirely encompasses Addis Ababa and the particular sites sampled by the YLS were clustered relatively closely around the city, perhaps allowing the communities favorable access to the aforementioned agricultural markets. Finally, the far northern region of Tigray is generally

Table 4: Heterogeneity of Effects by Region

	<i>Height</i> ₁	<i>Height</i> ₅	<i>Height</i> ₈
	(1)	(2)	(3)
<i>Exp</i>	-0.250 (0.279)	-0.682*** (0.284)	-0.914*** (0.179)
<i>Exp</i> × <i>Region</i>			
Addis Ababa	0.223 (0.323)	0.399 (0.253)	0.708*** (0.246)
Amhara	0.404 (0.422)	0.771*** (0.164)	0.794*** (0.270)
Oromia	0.171 (0.267)	0.212 (0.151)	0.611** (0.291)
Tigray	0.132 (0.295)	0.454 (0.267)	0.263 (0.172)
<i>Obs</i>	1,840	1,804	1,776

Robust standard errors (clustered at the community level) in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Dependent variable across columns: height at age one, five, and eight. Additional independent variables in all regressions: age of child in months, wealth index, number of older siblings, mother’s height, and dummies for gender, mother’s education, ethnicity, weeks premature, month of birth, and community.

classified as a food deficit area due to its semi-arid climate and dense population. After depleting harvested grain stocks, households in the region are heavily reliant on regional markets leading to vulnerability to seasonally rising food prices.

4.3 Timing of Prenatal Exposure

In addition to the benchmark measure of total prenatal exposure, it is also insightful to analyze how effects are influenced by the timing of exposure over gestational periods of development. In analyzing these differences, I follow the common practice of delineating the prenatal period into three trimesters of pregnancy. These trimesters roughly coincide with embryogenesis (first trimester), fetal development (second trimester), and a perinatal period (third trimester). The empirical medical and economic literature examining prenatal shocks during alternate gestational periods is substantial and quite varied. However, there is considerable evidence that long-term health outcomes such as diabetes and heart disease may be particularly sensitive to insults during the first trimester of gestation.¹¹ In regards to short-term effects, studies have shown significant impact on birthweight from shocks during all stages of prenatal development, though a majority are focused around mid to late term shocks (Currie and Almond 2011).

¹¹See, for example, the summary of studies in Almond and Mazumder (2011), Table A1.

In order to empirically examine the importance of the timing of prenatal exposure to seasonal food scarcity in Ethiopia, I translate my baseline exposure measure into distinct trimester measures. In practice, I drop the total exposure measure (*Exp*) from the benchmark specification and add three measures indicating the number of exposure days during each trimester. Results from this specification are reported in Table 5. The first three columns show the effects of exposure by trimester of gestation on child height at ages one, five, and eight. For example, exposure to an additional month of reported food scarcity during the first trimester reduces child height by an estimated 0.44 cm by age five. While coefficients are negative for all trimesters at age five, the magnitude of the estimate is considerably larger and statistically significant only for the first trimester. By age eight, effects are significant from exposure during all stages of gestation, but remain strongest for the first trimester. As age five and eight measures were taken several years after gestation, I view this as consistent with the empirical findings that health insults during the first trimester have stronger effects on long-term health outcomes.

Table 5: Effects of Exposure by Trimester of Gestation

	<i>Height</i> ₁	<i>Height</i> ₅	<i>Height</i> ₈	<i>BMI</i> ₁	<i>BMI</i> ₅	<i>BMI</i> ₈
	(1)	(2)	(3)	(4)	(5)	(6)
1st Trimester	-0.193 (0.186)	-0.438** (0.183)	-0.633** (0.262)	0.017 (0.042)	0.043 (0.037)	0.061 (0.056)
2nd Trimester	-0.033 (0.247)	-0.299 (0.205)	-0.364** (0.139)	-0.049* (0.027)	-0.026 (0.031)	-0.005 (0.019)
3rd Trimester	-0.032 (0.162)	-0.247 (0.120)	-0.480** (0.213)	-0.004 (0.039)	0.028 (0.041)	0.053 (0.033)
<i>Obs</i>	1,840	1,804	1,776	1,748	1,804	1,776
<i>R</i> ²	0.397	0.267	0.219	0.283	0.153	0.108

Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: height and body mass at age one, five, and eight. Reported independent variables: exposure to seasonal food scarcity by trimester. Additional independent variables in all regressions: age of child in months, wealth index, number of older siblings, mother's height, and dummies for gender, mother's education, ethnicity, weeks premature, month of birth, and community.

The last three columns of Table 5 give the estimated trimester effects on child body mass. At age one, estimates on the last two trimesters are negative, though the coefficient is only statistically significant for the second trimester. This implies that an additional month of exposure to food scarcity during the second trimester reduces child body mass at age one by an estimated 0.05 standard deviations. As age one measurements are taken relatively soon after birth, I view these results as consistent with the empirical evidence that mid to late term shocks have a stronger impact on birthweight. However, unlike effects on child height, this result does not intensify with

age, as the magnitude and statistical significance fades away by age five. This is again consistent with body mass being a measure of health inputs in the relatively short-term.

5 Assessing Alternate Channels

5.1 Water Accessibility and Maternal Labor Supply

Due to the explicit nature of the survey data used to construct my exposure measure, I argue that my results are primarily operating through nutritional intake fluctuations due to seasonal variations in food availability and/or prices. However, it is conceivable that my results could stem from other channels as well. For example, if seasonal variation in food scarcity is highly correlated with water supply quality for some households, results could be driven by the adverse disease environment that is known to accompany limited access to clean water. In order to evaluate the evidence on the commonly proposed confounding channels in developing countries—water quality and maternal labor supply—I examine heterogeneity of effects by household sources of drinking water and prenatal exposure to increased labor demand.

I begin by re-estimating the benchmark specification with the inclusion of an indicator for household source of drinking water as well as its interaction with prenatal exposure to reported food scarcity. I also include an interaction between the exposure measure and the wealth index to help ensure that water source interactions are not simply reflecting exposure effect heterogeneity by household wealth. Panel A of Table 6 reports results for the altered specification. The water source reference group are those families who access water through unprotected sources (e.g. river, pond, unprotected well). If access to clean drinking water was indeed a confounding channel, we would expect households with better access to protected water sources to be less adversely affected by the exposure measure (i.e. positive interaction terms). Results indicate no statistically significant difference in the effects of exposure between the reference group and those who access water through private pipes, public standpipes/wells, or other sources. I will note, however, that in comparison to the main exposure effect, the magnitude of several interaction coefficients are positive and substantial for the private pipe and other sources categories. However, the coefficients are imprecisely estimated as these groups comprise only about 11% and 5% of the sample, respectively. Moreover, as these categories are highly correlated with wealth, interaction coefficients could also be reflecting socioeconomic heterogeneity not fully captured by the wealth index interaction. A majority of sample households that did not obtain water through unprotected sources did so through public standpipes or wells. For this group, interaction estimates are both statistically insignificant and small in comparative magnitude. Thus, at least tentatively, I find little evidence suggesting that results are being substantially driven by seasonal variations in water supply quality.

An alternative concern is that agricultural production cycles may be correlated with maternal labor supply. Researchers have argued that seasonal changes in maternal labor supply during pregnancy could impact fetal development (e.g. Strand et al.

Table 6: Heterogeneity by Water Source and Exposure to Seasonal Labor Demand

	<i>Height</i> ₁	<i>Height</i> ₅	<i>Height</i> ₈
	(1)	(2)	(3)
<i>Panel A — Heterogeneity by Source of Water</i>			
<i>Exp</i>	0.043 (0.265)	-0.386* (0.221)	-0.498** (0.204)
<i>Exp</i> × <i>Wealth</i>	0.159 (0.102)	0.184** (0.084)	0.199 (0.133)
<i>Exp</i> × <i>WaterSource</i>			
Private Pipe	-0.245 (0.216)	0.254 (0.285)	0.234 (0.299)
Public Standpipe/Well	-0.181 (0.146)	0.022 (0.205)	-0.030 (0.203)
Other	-0.100 (0.403)	-0.018 (0.482)	0.351 (0.372)
<i>Panel B — Effects of Exposure to Seasonal Labor Demand</i>			
<i>Exp</i>	-0.058 (0.211)	-0.328* (0.180)	-0.457*** (0.139)
<i>Labor</i>	-0.012 (0.123)	-0.069 (0.140)	-0.148 (0.126)
<i>Obs</i>	1,840	1,804	1,776

Robust standard errors (clustered at the community level) in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Dependent variable across columns: height at age one, five, and eight. Reported independent variables: prenatal exposure to seasonal food scarcity (both panels), interaction with wealth and water source (Panel A), and exposure to increased seasonal labor demand (Panel B). Additional independent variables: age of child in months, wealth index, number of older siblings, mother's height, and dummies for water source (Panel A only), gender, mother's education, ethnicity, weeks premature, month of birth, and community.

2011). This may be especially true in developing countries where labor is often concentrated in the physically demanding agricultural sector. Although I do not have a direct measure of maternal labor decisions throughout pregnancy, I use my measure of prenatal exposure to seasonal increases in local labor demand (*Labor*) as a proxy for increases in maternal labor supply. As shown in Panel B of Table 6, adding this proxy to the benchmark specification has little impact on estimated effects of exposure to seasonal food scarcity. If anything, the magnitude of impacts increase slightly suggesting benchmark results may be biased towards zero due to correlations with maternal labor supply. This is perhaps unsurprising as it is quite plausible that maternal labor supply is negatively correlated with food scarcity as well as child health outcomes.

It may be of interest to note that all point estimates on the maternal labor supply proxy are negative and increasing in magnitude over time. As such, results could still be consistent with the conjecture of maternal labor decisions as a separate channel through which prenatal environment affects long-term health trajectories in Ethiopia. However, the further analysis of such a channel is outside the scope of this paper.

5.2 Seasonal Patterns of Fertility

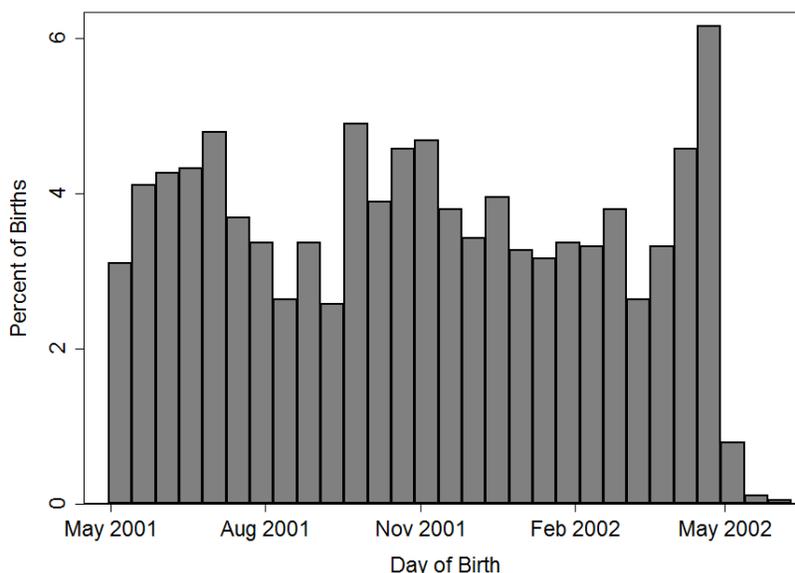
A remaining concern is the possibility that seasonal patterns of fertility within communities could be biasing the results of the empirical analysis. Recall that while inclusion of month of birth dummies controls for seasonal effects that occur at the country level, it is still conceivable that seasonal fertility patterns could correlate with unobserved characteristics within communities. If, for example, the pregnancies of wealthier women correlate with periods when food is relatively plentiful, then results could be attributed to differences in resources available to the child as opposed to exposure to food scarcity. Moreover, studies have documented seasonal patterns of fertility across a variety of countries (e.g. Rajagopalan et al. 1981; Panter-Brick 1996; Artadi 2005; Buckles and Hungerman 2013). In the developing world, these patterns have been most commonly linked to the influence of agricultural cycles on female labor supply, seasonality of marriage, and male migration. Although exposure is derived from atypical seasonal patterns of food scarcity, helping alleviate concern over some such confounding channels, this section examines the data for evidence of any remaining fertility bias within communities.

In order to evaluate the role of seasonal fertility patterns across the study sample, I begin by examining the timing of births by calendar date (see Figure 5). There is a small decline in births in August-September 2001 and January-March 2002, both followed by a period of somewhat higher birthrates. However, there is no discernible correlation between these birthrate patterns and the aggregated community food scarcity data shown in Figure 2. Nonetheless, to more rigorously evaluate the influence of fertility patterns associated with family demographics, I estimate the relationship between my measure of exposure to food scarcity and household characteristics. Specifically, I estimate the following equation:

$$ExpDays_{idc} = \alpha + \beta X_{idc} + \theta_c + u_{idc} \quad (2)$$

where X are the same set of child and household characteristics used in the baseline specification and θ are community of residence indicators.

Panel A of Table 7 reports the estimated coefficients on observed characteristics from equation 2. Virtually none of the child or household characteristics are related with the number of exposure days at conventional significance levels. The only exception is a negative correlation with premature birth. However, this is expected given the gestation period for preterm births is by definition shorter than full-term births. An F -test cannot reject the null-hypothesis that reported coefficients, other than those on premature birth, are equal to zero. As such, based on observed characteristics, there



Source: Author’s calculations using data from Young Lives Study, Ethiopia. Histogram with 2 week window for each bin.

Figure 5: Date of Birth

is no evidence of substantial selection based on demographic or socioeconomic seasonal patterns of fertility.

An alternate source of seasonal fertility bias could emerge as a result of unplanned pregnancies. Suppose, for example, that parents attempt to plan pregnancies around seasonal variations in food availability because they *believe* exposure is “bad” for their unborn child. In this case, children exposed to heavy amounts of food scarcity are more likely to be the result of unplanned pregnancies. Moreover, several studies have linked unplanned pregnancies to negative child outcomes including health status (e.g. Kost et al. 1998; Do and Phung 2010; Lokshin and Radyakin 2012). Under this scenario, the correlation between exposure and child health could be the result of a higher proportion of unplanned pregnancies during times of relative food scarcity.

In relation to unplanned pregnancies, the YLS survey asked participants the following question:

At the time you became pregnant with ‘NAME’ did you want to become pregnant?

Based on the replies, about 35% of pregnancies were “unwanted” with another 5% of responses missing due to the mother not being present for the interview. I use these data to evaluate the extent to which undesired pregnancies could be biasing my empirical results. Specifically, I re-estimate equation 2 but also include an indicator variable that takes a value of one if the pregnancy was reportedly “unwanted”. As shown in Panel B of Table 7, the relationship between undesired pregnancies and days

Table 7: Dependent Variable: Prenatal Days of Exposure to Seasonal Food Scarcity

<i>Panel A — Coefficients on Observed Characteristics</i>					
Male	-1.941	(1.292)	Wealth Index	-0.654	(1.425)
Mother's Height	-0.084	(0.190)	Mother's Education		
Older Siblings	-0.243	(0.443)	1 to 4 years	-0.276	(2.060)
Child Ethnicity			5 to 8 years	1.973	(2.326)
Amhara	-0.555	(5.035)	>8 years	4.905	(3.374)
Gurage	-1.204	(5.446)	Weeks Premature		
Oromo	3.962	(4.469)	1 to 2 weeks	-1.166	(4.061)
Tigrian	-2.497	(5.989)	>2 weeks	-15.540*	(8.870)
Hadiya	-1.766	(35.929)	Unknown	-16.984*	(8.669)
<i>Obs</i>	1890		<i>F</i> (11, 19)	0.96	<i>p</i> < 0.512
<i>R</i> ²	0.537				
<i>Panel B — Coefficient on Unplanned Pregnancy</i>					
Unplanned	2.388	(1.501)			
<i>Obs</i>	1807				
<i>R</i> ²	0.538				

Standard errors in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. All regressions include community fixed-effects. Reported *F-test* for joint significance of all independent variables in Panel A except weeks premature. Additional independent variables in Panel B: age of child in months, wealth index, number of older siblings, mother's height, and dummies for gender, mother's education, ethnicity, weeks premature, month of birth, and community.

of exposure to seasonal food scarcity is not statistically significant. The point estimate is also quantitative small—an undesired pregnancy is correlated with an increase in exposure of about two days. As such, I find little evidence of selection based on seasonal concentrations of unplanned pregnancies.

5.3 Mortality Selection

A final concern is the possibility of bias due to selective mortality on the basis of prenatal exposure to seasonal food scarcity. The basic problem is that a given outcome is only observed for children that survive to the age of measurement. If prenatal exposure has differentiated mortality effects on children, the composition of survivors may be different than it would have been in absence of exposure. However, mortality selection from a negative in-utero health shock will generally result in estimates that understate the magnitude of effects (Almond and Currie 2011). For example, if only the healthier or more robust of the exposed children survive, then selective mortality of unhealthy children would be biasing results towards zero. Nonetheless, while I cannot

directly evaluate the magnitude of selective mortality that occurred in-utero, I can examine the evidence of selection bias between rounds of data collection.

Approximately 4.7% of children were missing health outcomes at age five, and 6.1% at age eight. Unfortunately, I cannot identify the proportion of missing data that occurs as an explicit result of child mortality. However, the estimated child mortality rate between ages one and five for Ethiopia in 2006 was 3.7%, suggesting a potentially significant role for mortality in generating missing survey data (World Bank 2015). Moreover, while receiving less attention in the literature, other proximate causes of missing data could bias results through similar mechanisms as mortality selection.

In order to evaluate the potential role of this type of selection bias on results, I estimate the probability of having missing data at ages five or eight using a simple linear probability model:

$$Miss_{idca} = \alpha + \delta Exp_{dc} + \pi H_{idc} + \kappa (Exp_{dc} \times H_{idc}) + \beta X_{idc} + \mu_m + \theta_c + u_{idc},$$

where $Miss_{idca}$ is an indicator for a missing health outcome at age a , H_{idc} is a measure of child health during the first round of data collection, and other explanatory variables are as in the benchmark specification.¹² In practice, I use height and body mass at age one as alternate proxies for child health during the first round. The coefficient of interest is that on the interaction between prenatal exposure and age one health. A significant coefficient would reject the null hypothesis that exposure has no differentiated effects on the probability of missing outcomes based on early health status.

Table 8 shows the relevant results from the probability estimates using first round height or body mass as early measures of child health. All interaction coefficients are statistically insignificant at conventional levels, providing no evidence of substantial selection bias due to mortality or other forces resulting in missing health outcomes. Moreover, although imprecisely estimated, point estimates for all interaction coefficients are negative. This tentatively suggests that, if anything, healthier children are more likely to survive exposure than their less healthy counterparts. This would be consistent with the results from the benchmark specification underestimating the effects of prenatal exposure to seasonal food scarcity.

6 Conclusions

This paper presents novel evidence on the impact of prenatal nutritional environment on later childhood health outcomes. Among a cohort of Ethiopian children, I find that prenatal exposure to months of reported seasonal food scarcity had a significant negative effect on height by age five. Furthermore, these effects strengthen by age eight and are stronger when exposure is concentrated in the first trimester, supporting early gestation as a “sensitive” period of child development. In contrast, effects on

¹²Significance of results are robust to the use of a logistic regression. I present the linear probability model for simplicity in interpreting interaction terms.

Table 8: Heterogeneity of Effects of Exposure on Probability of Missing Outcomes

	<i>Miss</i> ₅	<i>Miss</i> ₅	<i>Miss</i> ₈	<i>Miss</i> ₈
	(1)	(2)	(3)	(4)
<i>Exp</i> × <i>Height</i> ₁	-0.031 (0.052)		-0.028 (0.056)	
<i>Exp</i> × <i>BMI</i> ₁		-0.004 (0.005)		-0.001 (0.005)
<i>Obs</i>	1,840	1,748	1,840	1,748
<i>R</i> ²	0.055	0.054	0.050	0.046

Coefficients of linear probability model reported. Robust standard errors (clustered at the community level) in parentheses. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Dependent variable across columns: missing outcomes at age one and five. Reported independent variables: interaction between prenatal exposure to seasonal food scarcity and round one height (meters) or body mass. Additional independent variables in all regressions: height (round one), body mass (round one), age of child in months (round one), wealth index, number of older siblings, mother’s height, and dummies for gender, mother’s education, ethnicity, weeks premature, month of birth, and community.

child body mass are strongest when exposure is concentrated in the second trimester and tend to fade with time. Consistent with other empirical studies, effects are also stronger for poorer children and those born to less educated mothers. I also find no evidence that results are driven by commonly proposed seasonal factors other than prenatal nutritional environment.

The impact of seasonal food scarcity on prenatal development has important policy implications in Ethiopia and throughout much of the developing world. In the long-run, addressing the source of seasonal food insecurity likely involves substantial public investment in transport infrastructure, storage and processing technologies, promotion of alternate crop varieties, and agricultural market organization (World Bank 2012). In the meantime, social safety net programs can serve to combat the impact of seasonal food scarcities in the short-run. However, these programs come at the cost of diverting limited public funds from long-term investments and potentially creating a chronic dependency on food aid. As such, understanding which populations are particularly vulnerable to seasonal food insecurity can help efficiently target relief interventions.

In Ethiopia, policies such as the Productive Safety Net Programme (PSNP), launched in 2005, already aim to provide predictable support for seasonal variations in food availability. Further targeting benefits towards pregnant women or those of child rearing age could be a low-cost but effective modification to such a program. Information or family planning campaigns could also be modified to emphasize the impact seasonal variations in maternal diets can have on fetal development. Other low-cost interventions such as distribution of nutrient rich season-specific recipes or improved home-based preservation technologies are being piloted in other developing countries (Wijesinha-Bettoni et al. 2013). My findings suggest such policies to mitigate the effects of seasonal food scarcities on the prenatal nutritional environment could have significant and long last-

ing impacts on child health.

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Appendix A: Proof of Proposition

Proof. For ease of exposition, I suppress controls (X, μ) and error terms (u) below. Due to collinearity, $Exp_{dc} + Exp_{dc}^* = K_c$, where Exp_{dc}^* denotes exposure during the three months after birth and K_c is some constant within each community. Denoting the total effect of prenatal exposure to food scarcity γ and the total effect of exposure during the three months after birth ϕ , the empirical specification can be derived:

$$\begin{aligned} Y_{idc} &= \alpha + \gamma Exp_{dc} + \phi Exp_{dc}^* \\ &= \alpha + \gamma Exp_{dc} + \phi (K_c - Exp_{dc}) \\ &= \alpha + (\gamma - \phi) Exp_{dc} + \phi K_c \\ &= \alpha + \delta Exp_{dc} + \theta_c \end{aligned}$$

where $\delta = (\gamma - \phi)$ and the constant K_c is absorbed by the empirical community dummies θ_c . The assumption that the effects of exposure to relative food scarcity are weakly negative during pregnancy and the three months after birth implies $\gamma, \phi \leq 0$. Together with the identity $\delta = (\gamma - \phi)$, this assumption implies that $max \gamma = \delta$, or alternatively $min |\gamma| = |\delta|$, where $|\cdot|$ denotes the absolute value. \square