The World Bank Economic Review, 0(0), 2022, 1–17
https://doi.org10.1093/wber/lhac009
Article

OXFORD

Food Inflation and Child Health

Andinet Woldemichael, Daniel Kidane, and Abebe Shimeles

Abstract

Malnutrition is one of the most important early life shocks that have lasting effects on health. An often neglected cause of malnutrition and hidden hunger is high food inflation, particularly in developing countries. This study uses the Ethiopian Demographic and Health Survey data, matching each child's early life age in months from the time of conception with the corresponding local monthly food price data to examine the medium-term and long-term impacts of exposure to food inflation during the critical early life window—pregnancy and infancy—on child health. Exposure to one percentage point higher month-to-month food inflation while in utero increases the risk of under-five stunting by 0.95 percent. The impacts are heterogeneous depending on the month of exposure, highlighting the complicated biological mechanisms through which malnutrition during early life affects human growth. The results are robust to various empircal specifications and potential biases arising from survivor sample selection and age misreporting.

JEL classification: D13, E31, I10, I15

Keywords: in utero, height, stunting, macroeconomic instability, malnutrition, low-income, Africa

1. Introduction

Shocks during pregnancy and early childhood—the first 1,000 days after conception—have lasting consequences on health, educational and cognitive achievements, labor market outcomes, and social behavior (Case and Paxson 2010; Currie and Almond 2011). One of the most critical early life shocks in developing countries is malnutrition. It is detrimental to healthy brain development, physical stature, and the immune system and is linked to the onset of chronic diseases during adult life (Barker 1995; Barker and Clark 1997; Victora et al. 2008; Ford et al. 2018). More importantly, the effects of malnutrition during early life are the most difficult to reverse through *ex post* remediation (Currie and Almond 2011). Using a rich dataset from Ethiopia, this paper investigates how exposure to malnutrition caused by food inflation, while in utero and during infancy, affects under-five stunting.

The impacts of severe nutrition shocks such as famine and drought are widely recognized in the literature and in policy spheres, and responses through, for instance, emergency food aid, are relatively prompt. However, the channels through which exposure to high food inflation impacts child health are not well

Andinet Woldemichael (corresponding author) is a principal research economist in the Development Research Department at African Development Bank, Abidjan, Côte d'Ivoire; his email address is a.woldemichael@afdb.org. Daniel Kidane is an economist at the Centers for Medicare and Medicaid Services, Bethesda, MD, USA; his email address is dakidane@gmail.com; Abebe Shimeles is a director of research at the African Economic Research Consortium (AERC) Nairobi, Kenya; his email address is abebe.shimeles@aercafrica.org. The authors wish to thank Professor Eric Edmonds and three anonymous reviewers for their extensive comments and suggestions, which helped improve the paper considerably. A supplementary online appendix is available with this article at *The World Bank Economic Review* website.

studied. There are several possible channels through which child health can be affected by high food inflation. It results in malnutrition in low-income households as they respond by rationing available food and reducing the quantity and dietary diversity of their food, putting children and pregnant women at greater risk of malnutrition (D'Souza and Jolliffe 2011; Tandon and Landes 2014). Household non-food-related strategies to mitigate high food inflation, such as selling productive assets, reducing productive investment, and borrowing at higher interest rates, negatively impact future income-generating capacity while also increasing the risk of future poverty and malnutrition (Barrett, Garg, and McBride 2016). Moreover, coping with high food prices by forgoing critical medical care affects health adversely during childhood and adult life (Tandon and Landes 2014). Therefore, childhood malnutrition due to food inflation is a crucial welfare issue that is often neglected in the literature.

This study examines the causal effects of exposure to food inflation during early life on the health status of children. The paper uses stunting, a standard measure of impaired growth due to poor nutrition, as an indicator of child health status. It uses data from the Ethiopian Demographic and Health Survey (DHS) and monthly local retail food price data, matching each child's early life calendar month since conception to the corresponding local monthly food inflation data. The econometric analysis implemented different empirical strategies and performed robustness checks to establish causality.

Two factors motivated the focus on Ethiopia. The first is that food inflation there from 2000 to 2010 was unprecedented compared to past inflationary episodes (Admassie 2013). For instance, month-to-month inflation was much higher for major staple crops and surpassed 100 percent in July 2008. This is particularly important because food accounts for more than 50 percent of the Consumer Price Index (CPI) consumption basket (Central Statistical Agency 2009). The full extent of the impacts of such a dramatic surge in food prices on children's health is not well understood. The second factor is that Ethiopia has made significant progress in reducing child malnutrition, with under-five stunting decreasing from 57 percent in 2000 to 44 percent in 2011. High food inflation during this period might have reversed some of these gains. This paper aims to provide some evidence on its effects on children's health.

The present paper is closely related to that of Arndt et al. (2016), who investigate the contemporaneous impacts of food inflation in Mozambique in 2008–2009 on child health and found the prevalence of underweight children falls by about 40 percent when food inflation is low compared to when it is high. In a similar study, Alderman, Hoddinott, and Kinsey (2006) found that exposure to the 1982–1984 drought in Zimbabwe as a preschooler resulted in being 2.3 cm shorter, starting school 3.7 months later, and being 0.4 of a year lower in completed grade. Yamano, Alderman, and Christiaensen (2005) found that exposure to malnutrition due to crop failure in early childhood impeded childhood growth in regions where crop losses were high. More recently, Dercon and Porter (2014) investigated the long-term impacts of the severe famine shock in Ethiopia in 1984 on the height of young adults twenty years after experiencing the shock while in utero and as infants. They found that children who were three years old at the peak of the crisis were at least 3 cm shorter than older cohorts when they reached adulthood, less likely to have completed primary school, and more likely to have recently experienced illness. Similarly, De Waal, Taffesse, and Carruth (2006) found that the droughts in 2002–2003 substantially increased child mortality in affected regions.

While these studies focus on the impacts of supply-side nutritional shocks, such as drought and crop failure, little attention has been paid to the impacts of food inflation. Furthermore, there is little evidence of potential heterogeneity due to the complicated biological mechanisms through which exposure to malnutrition at different stages of early life affects human growth, an issue to which the literature does not give enough attention. There is also a noticeable gap in the literature and the policy arena regarding food inflation as an important cause of malnutrition in developing countries. Considering the recent trend of high food inflation in many developing countries, the lack of evidence about its impacts on long-term welfare and human capital is concerning.

This paper fills these gaps by quantifying the causal impacts of exposure to high food inflation during each month of early life on human growth from conception to age five. From a policy perspective, the paper brings fresh evidence to global nutrition policies and goals pertaining to the Sustainable Development Goal of eliminating all forms of hunger by 2030. It underscores that food market volatility in general, and food inflation in particular, are not just short-term nominal phenomena but are also important macroeconomic issues with lasting consequences on children's health. The results show that exposure to one percentage point of higher month-to-month inflation while in utero increases the likelihood of stunting by at least 0.95 percent among children under five years of age. There is also considerable heterogeneity depending on the month of exposure, with a pronounced long-term impact on child health from exposure during the second and third trimesters of pregnancy. The results are robust to different econometric specifications and potential biases. The study also performs a battery of robustness checks and rules out potential biases due to survivor sample selection problems, the potential misreporting of children's age, and unobserved family-level heterogeneity. The paper also addresses the issue of false discovery (false positives) arising from multiple hypothesis testing, a statistical problem that is often neglected in the literature.

The rest of the paper is organized as follows. Section 2 describes the biological mechanisms through which exposure to malnutrition during different stages of early life affects long-term health outcomes. Section 3 describes the data. Section 4 presents the study's empirical estimation strategy. Section 5 discusses the results, and section 6 concludes the paper with some policy recommendations.

2. Biological Mechanisms

Maternal undernutrition before and during pregnancy affects fetal growth and alters the fetal genome, which is referred to as "fetal programming," through different channels (Barker and Clark 1997; Wu et al. 2004). Fetal nutrition is determined by the mother's dietary intake and nutrient stores, nutrient delivery to the placenta, and placental transfer capabilities (Owens, Owens, and Robbinson 1989; Barker and Clark 1997). Factors such as maternal nutrient stores and anemia can also shape the in utero environment well before conception. The in utero environment—fetal nutrition and oxygen supply—can change the concentration of growth-influencing hormones such as insulin and insulin-like growth factors (IGFs) and the sensitivity of early embryonic growth to nutrients. Thus, fetal growth and the fetal growth trajectory are especially susceptible to maternal dietary deficiencies prior to implantation and during rapid placental development (Wu et al. 2004).

Similarly, undernutrition in the last trimester affects fetal development, resulting in fetal wasting and fetal amino acid consumption by the placenta. Such downward pressure on the fetal growth trajectory during the early gestation period alters subsequent nutritional demand (Barker and Clark 1997). Moreover, depending on the trimester of exposure, the effects of undernutrition on birth weight, adult health outcomes, and death are heterogeneous.[1] Barker and Clark (1997) also suggest that low nutrient and oxygen supply in different trimesters of pregnancy impair growth during fetal life and could permanently affect the physiological structure of critical organs and tissues such as the endocrine pancreas, liver, and blood vessels.

Food availability affects newborn and infant growth by affecting the mother's lactation capacity and milk production (Rasmussen 1992). Studies show the mother's nutritional history has a greater impact on her lacation capacity than other factors such as age, breast enlargement during pregnancy, and genetics (Hytten 1954; Rasmussen 1992, in Rasmussen and McGuire, 1996; Rosso et al. 1981, in Rasmussen 1992).[2] Maternal malnutrition, therefore, determines lactation performance through its impact on

1 See table S3.2 in the supplementary online appendix, which is available at *The World Bank Economic Review* website.

2 A simplified channel linking maternal malnutrition to infant growth is shown in fig. S3.1 in the supplementary online appendix which is available at *The World Bank Economic Review* website.

maternal nutrient stores and nutrient mobilization. In undernourished women, milk production is limited by lactation capacity, the availability of substrates for milk biosynthesis, or both. Evidence shows that seasonal food shortages, as opposed to the general undernutrition of women, are associated with a significant decrease in infant milk intake (Rasmussen 1992). This study's empirical analysis recognizes such heterogeneity and quantifies the effects of exposure during a specific month of early life.

3. Data and Descriptive Results

Data are collected from two rounds of the Ethiopian DHS: 2005 and 2010. The DHS is one of the most comprehensive surveys conducted in developing countries that focuses on children and women. The survey collects detailed demographic, anthropometric, health, parental, household, geographic, and population information. Moreover, it is one of the few nationally representative household surveys on children's and women's health in Ethiopia. Close to 20,000 children under the age of five were covered in the 2005 and 2010 waves. The present study follows the literature and excludes infants younger than six months (see, for instance, Duflo 2000). Thus, the final pooled sample consists of 12,296 children between the ages of 6 and 59 months who were born between 2000 and 2010.

The key outcome variable of interest is the stunting status of children under the age of five. The study constructs stunting variables based on the height-for-age (HAZ) standardized score calculated using the World Health Organization (WHO) standard reference, which takes children's age, sex, and country into account.[3] The policy variable of interest is month-to-month staple food inflation. Given that the DHS does not collect monthly local food price information, monthly retail food price data are used from the Ethiopian Central Statistical Agency (CSA). The food price survey regularly gathers price information from 119 local markets and is one of the most comprehensive surveys that it periodically conducts and uses to construct regional and national consumer price indices (CPIs). The survey covers thousands of goods and services traded in the markets, and the data spans July 2001 to November 2012. First, the study spatially merged the DHS and CSA price survey datasets using a GIS-based nearest neighbor match algorithm.[4] Then, the study matched each child's early life months from the time of conception with the corresponding month-to-month inflation rate in the nearest market. Based on the literature, the study assumes a normal gestation period of 40 weeks (Almond and Mazumder, 2011; Persson and Rossin-Slater 2018).

One of the limitations of the GIS-based nearest neighbor matching technique is that it does not consider topography or road conditions as it is based simply on geodesic distance, the shortest possible line between two coordinates. Therefore, the actual ground travel distance between two linked points could be different depending on the topography, availability and type of roads, and weather conditions, given the poor road network and rugged terrain in the country. Also, it is important to note that the DHS randomly displaces GPS coordinates of clusters up to 2 kilometers for urban areas and 10 kilometers for rural areas (Burgert et al. 2018).

Households in Ethiopia spend a significant proportion of their income on food, with a few staple crops constituting a major share.[5] This study focuses on six main staple crops—*teff*, wheat, maize, barley, sorghum, and *enset*—which are widely consumed in different parts of the country and are important

3 A child is considered stunted if their HAZ is below –2.00. A detailed description of the methodology and the software (Stata) are available on the WHO's website (WHO 2006).

4 See fig. S3.3 in the supplementary online appendix, which is available at *The World Bank Economic Review* website.

5 For instance, food expenditures account for more than 83.1 and 71 percent of rural and urban households' consumption budget, respectively, with the combined share of *teff*, wheat, and maize being 25 percent and 20 percent of their total consumption budget in rural and urban areas, respectively (Shimeles and Woldemichael 2013).

sources of daily caloric intake, micronutrients, minerals, and carbohydrates.[6] To account for dietary differences based on geography, a single composite staple price index is constructed using the average calorie shares for each region as weights, which were obtained from 2000 and 2005 Ethiopian Household Income and Consumption Expenditure Survey (HICE) data. The HICE is a nationally representative consumption and income survey that is conducted every five years. The composite price index also rules out possible substitution effects across different crops arising from price changes that could confound the results.

Variation in food inflation can come from different sources, including change in consumption bundles over time and across regions, change in inflation for a fixed consumption bundle, or both. Therefore, it is important to establish that the variation comes primarily from change in food inflation. To do that, the study set the initial caloric weights constant at the level recorded in 2000, which was the first year the HICE was conducted. In the time-variant consumption bundle, the caloric weights are allowed to change over time using the 2000 weights for prices observed in 2001–2004 and the 2005 weights for prices observed in 2005–2010. Generally, the variable consumption bundle's standard deviation is expected to be higher than that of the fixed bundle since the variation comes from changes in the bundle and changes in inflation. This study's assessment shows that about 84 percent of the variation comes from changes in inflation and the rest from changes in the consumption bundle.[7] The analysis attempts to control for the small amount of variation (16 percent) coming from the change in the consumption bundle by including region and survey year interaction terms in the regression.

A simple plot shows strong correlation between early-life exposure to food inflation and stunting (see fig. 1). Children exposed to higher food inflation early in their lives are more likely to be stunted. However, the slope is much steeper for exposure while in utero than exposure during infancy. The correlation also varies depending on the month of exposure. Moreover, it varies by observable characteristics such as sex.[8]

Table 1 presents the summary statistics of control variables, including child-level, parental, household, geographic, and temporal factors that are controlled for in the regression analysis.[9] Child-level factors include age and age squared to capture the geometric relationship between age and height or stunting variables. The study also controls for sex, birth order, and subjective assessment of the child's birth weight as recalled by the mother. Furthermore, the analysis controls for *ex ante* prevention and *ex post* remedial parental behavior in response to actual or perceived nutrition and health outcomes, which include receipt of prenatal care, vaccination, Vitamin A supplementation, and the duration of breastfeeding as recalled by the mother or recorded on a vaccination card. The analysis also includes birth month in the regression to control for seasonal food availability and the outbreak of seasonal diseases such as flu, diarrhea, and malaria.

The medical literature shows that genetic factors predispose a child to certain diseases and a certain physical stature, possibly confounding the results. Such issues are addressed by controlling for maternal characteristics such as age, age at first birth, height, body mass index (BMI), and anemia status, which are good indicators of maternal health history and genetic factors. Additional controls that capture maternal

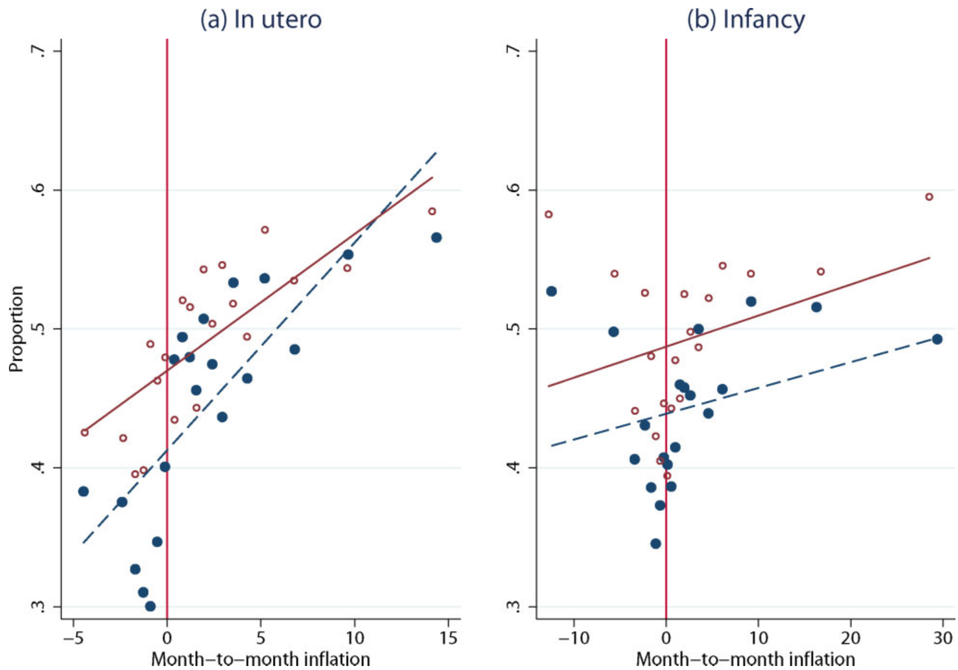
6 For instance, *teff*, a gluten-free grain, is a good source of essential minerals (iron, magnesium, and calcium), carbohydrates/fiber, Vitamin B-12, and protein. While *teff*, maize, and sorghum are widely consumed, there is significant regional variation in diet composition, including wheat, barley, and *enset*. For example, *teff* accounts for about 24 percent of total household caloric intake in Addis Ababa but only 0.8 percent in the Somali region. See table S3.3 in the supplementary online appendix which is available at *The World Bank Economic Review* website.

7 See fig. S3.4 in the supplementary online appendix, which is available at *The World Bank Economic Review* website, for plots of the standard deviation of change in food inflation for fixed and variable consumption bundles.

8 For plots corresponding to each month of exposure from conception, see fig. S3.2 in the supplementary online appendix, which is available at *The World Bank Economic Review* website.

9 The full list of variables included in the regression is available in table S3.1 in the supplementary online appendix which is available at *The World Bank Economic Review* website.

Figure 1. Average Month-to-Month Food Inflation while in Utero and during Infancy (6 Months after Birth) and Stunting.



Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS).
Note: Solid dots and solid lines correspond to girls; dashed lines and hollow circles correspond to boys. The x-axis measures the month-to-month inflation that children in the sample were exposed to during the j^{th} month since inception. The y-axis measures the proportion of stunted children between the age of 6 and 59 months.

Table 1. Summary Statistics of Control Variables: Children Aged 6–59 Months

	2005	2010	Pooled
Exposure			
Month-to-month price change while in utero (%)	1.411	2.375	2.177
	(3.238)	(4.505)	(4.293)
Month-to-month price change during infancy (%)	1.094	2.794	2.444
	(5.066)	(9.219)	(8.559)
Average NDVI value while in utero	104.1	110.7	109.2
	(32.29)	(33.63)	(33.45)
Average NDVI value during infancy	100.2	103.2	102.5
	(13.63)	(17.61)	(16.86)
Child health outcomes and characteristics			
HAZ score	−1.909	−1.786	−1.822
	(1.857)	(1.666)	(1.725)
Stunted (%)	50.0	45.9	47.1
Age (months)	32.51	32.50	32.50
	(15.72)	(15.50)	(15.57)
Boy (%)	50.10	50.80	50.60
No. of observations	3,607	8,685	12,292

Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS), the monthly Ethiopian Central Statistical Agency (CSA) retail food price data from July 2001 to November 2012, and the monthly Normalized Difference Vegetation Index (NDVI) for Ethiopia between February 2000 and December 2011.
Note: Standard deviations are in parentheses. The full list of variables included in the regression is shown in table S3.1 in the supplementary online appendix, available with this article at *The World Bank Economic Review* website.

health behavior and preferences about the pregnancy are prenatal and postnatal visits and whether the pregnancy was planned. The study also controls for the number of living and deceased children the mother ever gave birth to as well as parental education and occupation, which are important factors in the child health production function. Furthermore, the analysis controls for household-level factors such as the age of the household head, the household size, the number of children under five in the household, the sex of the household head, religion, wealth index, and the size of the land owned. To account for village- and region-level factors, the study includes geospatial characteristics—a dummy for an urban area, altitude, geodesic distance to the market hubs, estimated travel time (in minutes) to the nearest city of more than 50,000 people, population density per square kilometer, and the number of drought episodes between 1980 and 2000. In addition, the analysis accounts for regional and subregional (zone) variations by including region and zone dummy variables and the interaction between region and survey year to capture region-specific changes over time. Finally, in all specifications, the study includes monthly Normalized Difference Vegetation Index (NDVI) values to control for area-level drought and food availability.[10]

4. Estimation Strategy

The primary interest of this study is to estimate the effects of exposure to food inflation while in utero and during infancy on the stunting status of children aged 6 to 59 months. The analysis regresses stunting status on the month-to-month food inflation that the child was exposed to while in utero (nine months in utero) and during infancy (six months after birth).[11] To formally describe the model, let h_{ijkv} denote the stunting status of child i born to mother j in household k and village v, which is located near market q. Then, the linear probability model can be written as:

$$h_{ijkv} = \alpha_0 + \sum_{m=-9}^{+6} \phi_m \pi_{k,q(m)} + \mathbf{\Theta W}_{ijkv} + \mu_i + \varphi_j + \omega_k + g_v + \delta_c + \epsilon_{ijkv}, \tag{1}$$

where $\{\phi_m\}$ is a set of coefficients that capture the impacts of exposure during the m^{th} month of early life ($m = [-9, +6]$ is the month of exposure), $\mathbf{\Theta}$ is a vector of coefficients to be estimated, $\mathbf{W}_{ijkv}$ is a vector of observed child, maternal, household, and area-level factors, μ_i, φ_j, ω_k, g_v, and δ_c denote child, mother, household, village, and cohort (year of birth) fixed effects, respectively, and ϵ_{ijkv} is the error term. Standard errors are clustered at the market level, q.

The analysis starts by determining a baseline of a simple ordinary least squares (OLS) estimate, ignoring child (μ_i), mother (φ_j), and household (ω_k) fixed effects, which are assumed to be absorbed in the idiosyncratic error term. However, in the OLS estimation, the analysis controls for potentially confounding village-level factors by including the history of drought and rainfall shocks in the village over the previous 30 years. More important, the study includes monthly NDVI values covering a 50-kilometer radius from the center of the village. The study also includes the interaction terms of region and survey year dummies to control for region-specific changes over survey waves. Finally, the baseline specification includes cohort dummies to account for cohort fixed effects.

Mother Fixed Effects Estimation

The OLS estimation of equation (1) assumes there is no unobserved heterogeneity arising from factors such as biological or genetic predispositions, perceptions, behavior, preferences towards child health, shocks in

10 Details on how the study calculated the NDVI values while in utero and during infancy for each cluster are discussed in S2 of the supplementary online appendix, which is available at *The World Bank Economic Review* website. The NDVI data were obtained from https://modis.gsfc.nasa.gov/data/dataprod/mod13.php.

11 A detailed conceptual framework that guides the empirical specification is presented in S1 of the supplementary online appendix, which is available at *The World Bank Economic Review* website.

family preferences, differences in child health production technology, and differences in intra-household food allocation behavior. Such unobserved heterogeneity could potentially bias the results if it is correlated with food inflation (i.e., $E(\epsilon_{ijkq} \cdot \pi_{k,q}) \neq 0$). Moreover, although market prices enter in the household optimization problem as given, and identification in the baseline model relies on exogenous variation in market-level food prices, there is potential endogeneity arising from omitted variables such as area-level policy changes and environmental factors. For example, factors such as child nutrition interventions by the government or by nongovernmental organizations could be correlated with child health outcomes. Such issues can be addressed using the panel fixed effects model. Unfortunately, because the data are a cross-section of only two rounds, it is possible to net out unobserved heterogeneity only at the family level using the mother fixed effects specification for children i and i' who were born to the same mother.[12] Household- and geographic-level unobserved heterogeneity are also differenced out.

The mother fixed effects model can be written as:

$$\Delta h_{ijkv} = \sum_{m=-9}^{+6} \widetilde{\phi_m} \Delta\pi^{f}_{k,q(m)} + \tilde{\mathbf{\Theta}}\Delta\mathbf{W}_{ijkv} + \mu_i + \delta_c + \Delta\epsilon_{ijkq}, \tag{2}$$

where $\Delta\, h_{ijkv} = h_{ijkv} - h_{i'jkv}$, $\Delta\, \pi_{k,\, q(m)} = \pi_{k,\, q(m_i)} - \pi_{k,\, q(m_{i'})}$, $\Delta\mathbf{W}_{ijkv} = \mathbf{W}_{ijkv} - \mathbf{W}_{i'jkv}$, $\Delta\epsilon_{ijkq} = (\epsilon_{ijkq} - \epsilon_{i'jkq})$, $\{\widetilde{\phi_m}\}$, and $\tilde{\mathbf{\Theta}}$ are coefficients, while mother, household, and geographic fixed effects drop out. Standard errors are clustered at the market level, q. Mother-level characteristics are not included in $\mathbf{W}_{ijkv}$. Although equation (2) is an improvement over (equation 1), it still does not difference out child-level fixed effects, μ_i. However, the study includes observed characteristics that capture some of the heterogeneity at the child level. The child-level controls are age, gender, birth month, birth weight, birth order, duration of breastfeeding, prenatal care, vaccination status, and receipt of vitamin A supplementation, which should capture some variations in unobserved child-specific endowments and biological predispositions.

Survivor Sample Selection Bias

Another empirical challenge that potentially biases the results is the survivor sample selection problem. The fertility history in the data shows that 11 percent of all pregnancies end up being terminated, and 9.5 percent of live births die before their fifth birthday. Some of these fetal and infant mortality cases could be driven by exposure to malnutrition.[13] Therefore, the sample could suffer from systematic incidental truncation, also referred to as survivor sample selection bias, resulting in attenuation of the effects towards zero (Wooldridge 2002).

There are several possible approaches to address this issue. The first approach is the inverse probability weighting method, which is a technique commonly used to correct for survivor sample selection bias in the literature. However, it has limitations in the context of fixed effects specification as the weights are invariant. Instead, this study applies an alternative approach that involves treating deceased ("missing") children as stunted and adding them back into the data. To do this, the analysis creates an auxiliary outcome variable called "missing or stunted" and estimates the model on controls available for both surviving and deceased children. If there is no significant survivorship bias, the magnitude and significance of the coefficients from this auxiliary regression would be expected to be the same as those in specifications (1) and (2).

12 Different terms are used in the literature, including "family fixed effects," "parent fixed effects," and "mother fixed effects." Given that survey responses about pregnancy and children are given by mothers, such information is used to difference out "mother-level" fixed effects. While "sibling fixed effects" and "twin fixed effects" are common approaches, this study's data are not in a sibling-pair panel form.

13 The impact of food inflation on mortality is documented in a recent study that uses the same dataset (Kidane and Woldemichael 2020).

5. Results and Discussion

This study's primary interest is to estimate the effects of exposure to food inflation while in utero and during infancy on the stunting status of children aged 6–59 months measured at survey time. This section presents and discusses the results. The first set of results are from the OLS estimation of the baseline specification shown in equation (1), including average month-to-month inflation while in utero and during infancy. The second set of results are from the mother fixed effects estimation of equation (2), which addresses possible bias due to unobserved heterogeneity at the mother, household, and geographic levels. Although this study has a small panel of two siblings, which potentially inflates the standard errors, the fixed effects estimation addresses a more pressing threat to identification—unobserved heterogeneity. The analysis then presents the results from the estimation that addresses heterogeneous effects depending on the month of exposure. Monthly NDVI values are included, which are a good proxy for food shortage and drought. Finally, this article discusses the robustness of the results to false discovery and potential threats to identification. The study reports robust standard errors clustered at the market level throughout all specifications.

Baseline Results

Table 2 presents the results from the baseline model.[14] The results in column 1 show that exposure to 1 percentage point higher month-to-month inflation while in utero increases the likelihood of childhood stunting by 0.009 probability points. Incrementally controlling for child, parental, and household characteristics in columns 2 through 6 slightly decreases the magnitude of the coefficient, which remains statistically significant. Controlling for birth cohort and age interaction in column 8 decreases the magnitude by more than 40 percent, to 0.0046, suggesting considerable cohort effects. It can be concluded from the results in the full specification (column 6) that exposure to 1 percentage point higher food inflation while in utero increases childhood stunting by 0.0046 probability points. In other words, a 1 percent increase in month-to-month food inflation results in a 0.96 percent increase in the probability of childhood stunting.[15] However, the study finds no evidence that exposure to food inflation during infancy affects childhood stunting, which could be due to the widespread practice of breastfeeding among Ethiopian mothers, which seems to provide some cushion against food inflation. Furthermore, the higher NDVI (lower drought risk) value during infancy is associated with a lower risk of stunting, although the coefficient is insignificant for exposure while in utero.

Results from Fixed Effects Estimation

The analysis addresses potential threats to identification from unobserved heterogeneity by estimating the mother fixed effects model specified in equation (2). Children with no siblings drop out of the sample, reducing the sample size to 8,300, and the panel is short, as 95 percent of children have only one sibling under five. Thus, the study expects the fixed effects estimate to be inefficient. Table 3 presents the results.[16] The coefficient on exposure while in utero without added controls is 0.0119 (column 1).

14 The study also estimates the regression using continuous HAZ scores. The results are reported in table S3.4 in the supplementary online appendix which is available at *The World Bank Economic Review* website.

15 To put this into perspective, children who were in utero during the inflationary episode of 2008–2009—during which average month-to-month food inflation was 5.9 percent—have a 0.027 probability point (or a 5.65 percent) higher risk of stunting. This result is obtained by dividing the probability point increase for a 5.9 percent inflation increase by the average stunting rate, i.e., 100*(5.9*0.0046)/0.4803.

16 The analysis also estimates the model using random effects in which the unobserved child-level heterogeneity is assumed to be normally distributed. The magnitude of the coefficient on in utero exposure is between the mother fixed effects and OLS estimates, and the standard error is much smaller than the mother fixed effects estimation. Overall, the estimates remain consistent across OLS, fixed effects, and random effects estimations. The results of random effects estimates are reported in table S3.6 in the supplementary online appendix, which is available at *The World Bank Economic Review*

Table 2. OLS Estimates of the Effects of Exposure to Food Inflation while in Utero and during Infancy on Stunting

	(1)	(2)	(3)	(4)	(5)	(6)
Month-to-month inflation while in utero	0.0094***	0.0081***	0.0080***	0.0080***	0.0081***	0.0046***
	(0.0014)	(0.0014)	(0.0014)	(0.0014)	(0.0014)	(0.0014)
Month-to-month inflation during infancy	−0.0007	0.0000	0.0001	0.0001	0.0002	−0.0004
	(0.0008)	(0.0007)	(0.0008)	(0.0007)	(0.0007)	(0.0007)
Average NDVI while in utero	0.0006	0.0005	0.0004	0.0003	0.0003	0.0002
	(0.0004)	(0.0004)	(0.0004)	(0.0004)	(0.0004)	(0.0004)
Average NDVI during infancy	−0.0021***	−0.0019***	−0.0016***	−0.0014***	−0.0014***	−0.0009**
	(0.0005)	(0.0005)	(0.0004)	(0.0005)	(0.0005)	(0.0005)
Observations	10,581	10,558	10,506	10,462	10,462	10,462
R-squared	0.0264	0.0608	0.1156	0.1249	0.1297	0.1385
Age and month of birth dummies	X	X	X	X	X	X
Child characteristics	–	X	X	X	X	X
Household characteristics	–	–	X	X	X	X
Mother's characteristics	–	–	X	X	X	X
Father's characteristics	–	–	X	X	X	X
Geographic characteristics	–	–	–	X	X	X
Survey year–region interaction	–	–	–	–	X	X
Age–birth cohort interaction	–	–	–	–	–	X

Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS), the monthly Ethiopian Central Statistical Agency (CSA) retail food price data from July 2001 to November 2012, and the monthly Normalized Difference Vegetation Index (NDVI) for Ethiopia between February 2000 and December 2011.

Note: Robust standard errors are in parentheses and clustered at market level. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. The month of birth is 11 calendar month dummy variables. "Age and month of birth dummies" is a vector of age, age squared, and 11 dummies for the month of birth. "Child characteristics" is a vector of child-level control variables including a sex dummy, birth order, a dummy if the child has siblings, dummies for the mother's categorical subjective assessment of birth weight, a dummy for whether the mother received prenatal care, a dummy if the child was ever vaccinated, a dummy if they received Vitamin A, and duration of breastfeeding in months. "Household characteristics" is a vector of household characteristics including household size, a dummy for sex of household head, number of under-five children in the household, household wealth index, land size in hectares, and a dummy for the religion of the household head. "Mother's characteristics" is a vector of mother-level characteristics including marital status, age, height, level of education, age at first birth, BMI, a dummy for anemia status, number of live children ever gave birth to, number of dead children, occupation, a dummy if antenatal care received, and a dummy if the pregnancy was planned. "Father's characteristics" includes an interaction term of marital status of the mother with the husband's education and occupation. "Geographic characteristics" is a vector of geographic characteristics including a dummy for being an urban area, altitude in meters, distance to the nearest market center in kilometers, a dummy if the distance to a health care facility is a "big problem," slope, population density, drought episodes since 1981, and a set of dummy variables for regions. Age–birth year cohort interactions are age in months interacted with an integer value of birth years. Standard errors are clustered at the market level.

However, when controlling for child-level factors (column 2), the coefficient decreased slightly, and adding birth cohort dummies further reduces the magnitude by more than half. Despite the short panel that inflates the standard error by a factor of two compared to the OLS estimates, the coefficient remains statistically significant.

As for exposure during infancy, the coefficient remains insignificant even after controlling for child-level characteristics and birth cohort fixed effects (columns 2 and 3). Children in villages with better vegetation have a lower risk of stunting. Overall, the fixed effects estimation results are consistent with the OLS estimates, although the standard errors are larger.

Heterogeneous Effects

To unpack the heterogeneity that depends on month of exposure, the analysis runs the models by including the month-to-month food inflation that the child was exposed to from conception. This means adding 15 month-to-month inflation variables corresponding to the specific months of exposure—the 9 months of the normal gestation period and 6 months after birth.[17] Figure 2 shows the coefficients from the OLS and

website. The corresponding HAZ score results are reported in table S3.5 in the supplementary online appendix, which is available at *The World Bank Economic Review* website.

17 The analysis also reports the results from OLS and fixed effects estimation with the adjusted *p*-values (q-values) in table S3.7 in the supplementary online appendix which is available at *The World Bank Economic Review* website.

Table 3. Mother Fixed Effects Estimates of the Effects of Exposure to Average Inflation while in Utero and during Infancy on Stunting

	(1)	(2)	(3)
Month-to-month inflation while in utero	0.0119***	0.0111***	0.00406*
	(0.00263)	(0.00258)	(0.00270)
Month-to-month inflation during infancy	−0.00316**	−0.00141	−0.00251
	(0.00124)	(0.00114)	(0.00118)
Average NDVI while in utero	0.000425	0.000329	0.000121
	(0.000740)	(0.000719)	(0.000729)
Average NDVI during infancy	−0.00320***	−0.00307***	−0.00221***
	(0.000879)	(0.000871)	(0.000837)
Observations	10,581	10,558	10,558
R-squared	0.052	0.123	0.139
Number of siblings	8,322	8,308	8,308
Age and month of birth dummies	X	X	X
Child characteristics	–	X	X
Age–birth cohort interaction	–	–	X

Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS), the monthly Ethiopian Central Statistical Agency (CSA) retail food price data from July 2001 to November 2012, and the monthly Normalized Difference Vegetation Index (NDVI) for Ethiopia between February 2000 and December 2011.

Note: Robust standard errors are in parentheses and clustered at market level. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. The month of birth is 11 calendar month dummy variables. "Age and month of birth dummies" is a vector of age, age squared, and 11 dummies for the month of birth. "Child characteristics" is a vector of child-level control variables including a sex dummy, birth order, a dummy if the child has siblings, dummies for the mother's categorical subjective assessment of birth weight, a dummy for whether the mother received prenatal care, a dummy if the child was ever vaccinated, a dummy if they received Vitamin A, and duration of breastfeeding in months.

fixed effects estimations, including the full list of control variables. It shows that the OLS and mother fixed effects estimation coefficients have similar magnitudes. The OLS estimation's coefficients on exposure during the fourth month (i.e., five months before birth, or B−5), the fifth month (B−4), the seventh month (B−2), and the eighth month (B−1) of pregnancy are all statistically significant. These months correspond to the second and third trimesters. In the fixed effects estimation, only the coefficient on the eighth month of pregnancy is statistically significant. Regardless, the results underscore considerable heterogeneity arising from the timing of exposure. It is important to note that the study did not find significant effects from exposure during infancy.

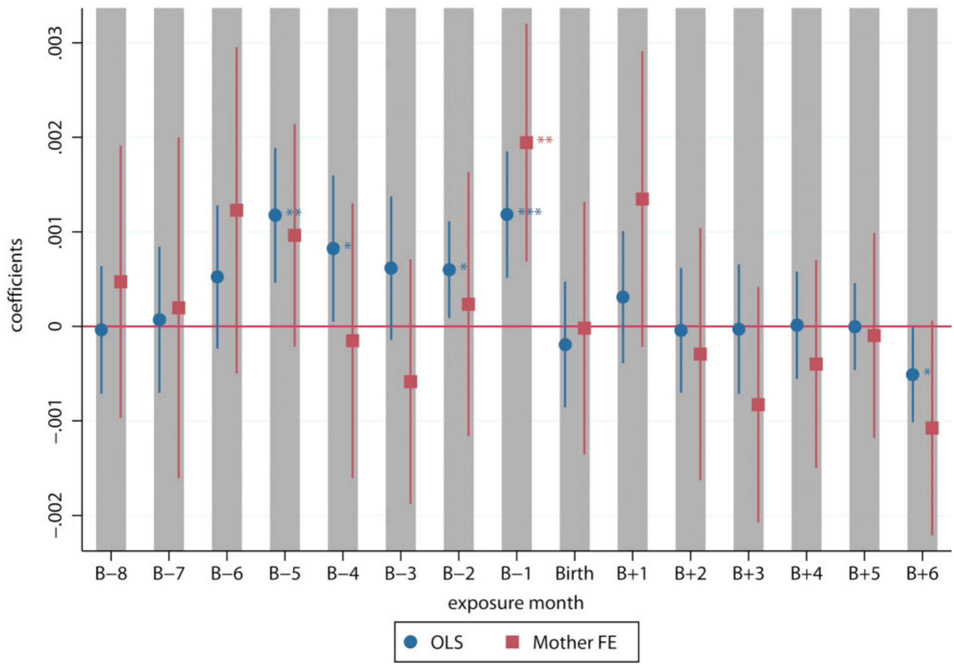
It is difficult to rely on the results in fig. 2, however, due to the problem of false positives (false discovery) associated with testing multiple hypotheses simultaneously (Benjamini and Hochberg 1995, 2000; Benjamini and Yekutieli 2001). In this study, 15 hypotheses are tested, corresponding to each month of exposure simultaneously, resulting in the possibility of false discovery in some of the coefficients. More specifically, with $k = 15$ hypotheses tested simultaneously at a confidence level of α, there is a $1 - (1 - \alpha)^k$ chance of false discovery (Rouam 2013). This implies that at an $\alpha = 10$ percent level of significance, there is a 79 percent chance that the analysis incorrectly rejects the null of at least one hypothesis.[18] To identify the coefficients for which the null hypothesis is incorrectly rejected due to false discovery, the false discovery rate (FDR) is computed for each estimate.

Figure 3 shows the FDR-adjusted *p*-values (also referred to as *q*-values) and the unadjusted *p*–values on the y-axis for the OLS (panel A) and fixed effects (panel B) models.[19] Although a 10 percent cut-off point was used to reject the null hypothesis, the FDR-adjusted *p*-values are much lower, at 0.77 percent.

18 Thanks go to the editor for pointing out the issue and for his helpful suggestions that informed the FDR corrections for this study's multiple hypothesis testing.

19 The study uses Benjamini and Yekutieli's (2001) method—a commonly used FDR control technique—to calculate the *q*-values. The **multproc** procedure in Stata is used.

Figure 2. Fixed Effects and OLS Estimates of the Effects of Exposure to Month-to-Month Inflation on Stunting.



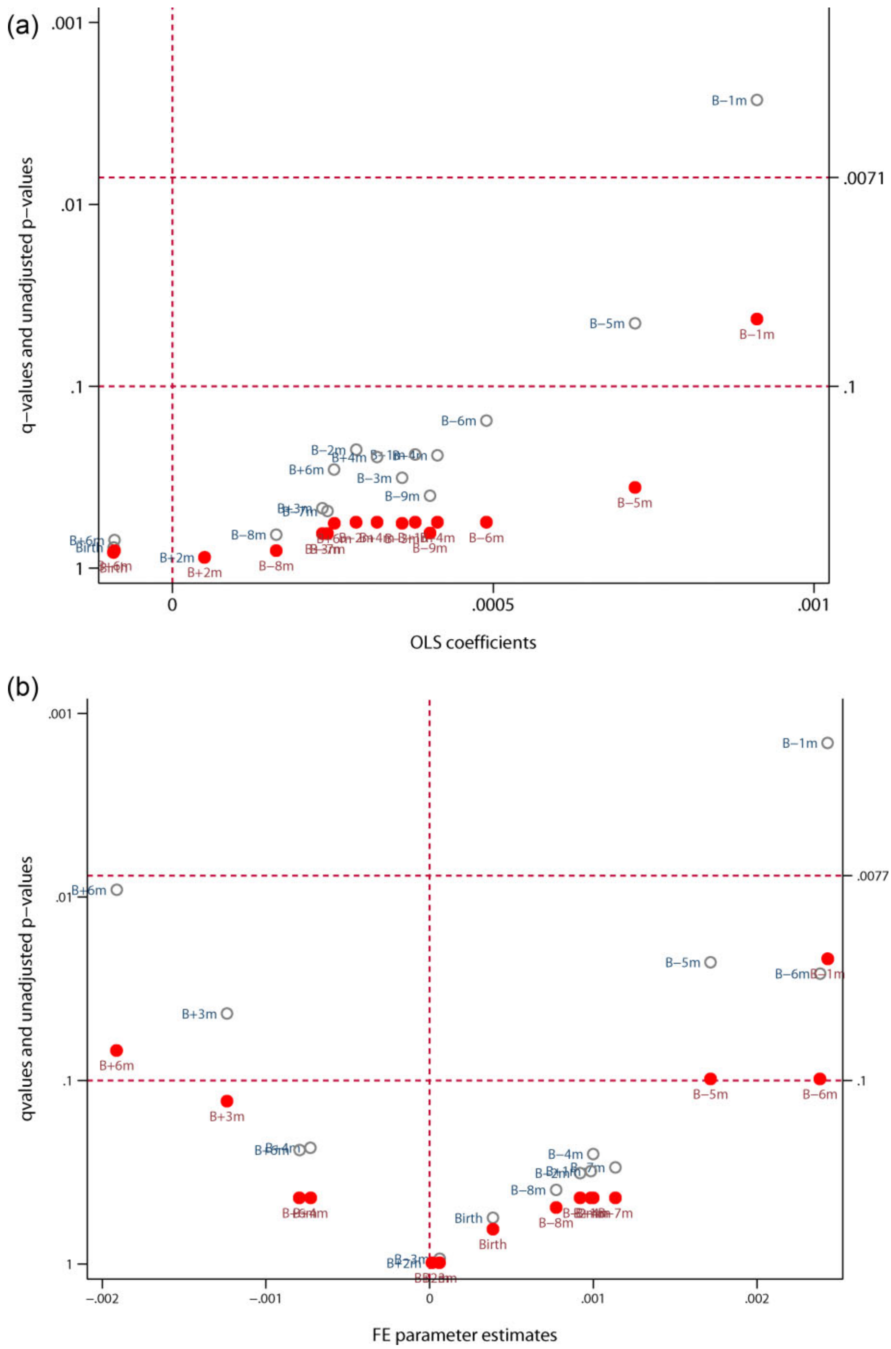
Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS) and the monthly Ethiopian Central Statistical Agency (CSA) retail food price data from July 2001 to November 2012.
Note: Robust standard errors are in parentheses and clustered at market level. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. The y-axis shows the estimated coefficient on the month-to-month staple food inflation of the mth month. The x-axis shows the month of exposure between the first month of pregnancy and six months after birth. The OLS estimates include all the controls in table 2, specification (6), whereas the mother fixed effects estimates include all the controls in table 4, specification (2). B−1 denotes 1 month before birth (eighth month of pregnancy), B−2 denotes two months before birth (seventh month of pregnancy), B−3 denotes three months before birth, etc. And B + 1 denotes one month after birth, B + 2 denotes two months after birth, etc.

Therefore, based on the 0.78 percent criteria, the analysis accepted the null for the OLS coefficient on the seventh month (B−2) and the fifth month (B−4) of pregnancy as well as the sixth month after birth, the latter of which was incorrectly rejected based on the unadjusted *p*–value. However, the study finds no fixed effects coefficients incorrectly rejected. Hence, it can be confidently inferred that the OLS and fixed effects coefficients on the fourth month (B−5) and eighth month (B−1) of pregnancy are statistically significant.

As discussed in section 4, survivor sample selection is another potential source of bias. Table 4 presents the results after adjusting for survivor sample selection bias by reclassifying deceased children as "missing or stunted." It shows that the magnitude of the adjusted coefficients on in utero exposure is much higher than that of the unadjusted coefficients in the main OLS and fixed effects specifications. For instance, the magnitude of the adjusted OLS coefficient on exposure while in utero is larger than that of the unadjusted coefficient reported in table 2. Similarly, in the fixed effects estimation of the "missing or stunted" sample, the magnitude of the coefficient is larger—almost 60 percent larger—compared to the coefficient in table 3, which does not address bias arising from survivors' sample selection. Therefore, survivor sample selection bias is an important factor that attenuates exposure to food inflation towards zero.

The analysis re-estimates the OLS and mother fixed effects models and includes month-to-month exposure to check whether the estimates are sensitive to survivor sample selection bias. The magnitude and significance of the coefficients are indeed sensitive to survivor sample selection. The coefficient on exposure remains stable up to the eighth month in utero. Moreover, coefficients that were previously not

Figure 3. q- Values and Unadjusted *p*-Values for the OLS and Fixed Effects Coefficients on Specific Month of Exposure on Stunting.



Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS) and the monthly Ethiopian Central Statistical Agency (CSA) retail food price data from July 2001 to November 2012.

Note: Robust standard errors are in parentheses and clustered at market level. The x-axis shows the OLS and fixed effects coefficients. The y-axis shows *q*-values and unadjusted *p*-values. The solid red dots represent the unadjusted *p*-values. The hollow gray circles represent the *q*-values (adjusted *p*-values). The parallel horizontal lines show the critical unadjusted *p*-value of 10 percent and the FDR adjusted *p*-value (*q*-values) of 0.7 percent for the OLS and 0.78 for fixed effects estimation. The OLS model includes all the controls in table 2, specification (6), whereas the mother fixed effects model includes all the controls in table 3, specification (3). B−1 denotes 1 month before birth (i.e., the eighth month of pregnancy), B−2 denotes two months before birth (i.e., the seventh month of pregnancy), B−3 denotes three months before birth, etc. And B + 1 denotes one month after birth, B + 2 denotes two months after birth, etc.

Table 4. Effects of Exposure to Average Inflation while in Utero and during Infancy on Stunting

	OLS: "missing or stunted"	Mother fixed effects: "missing or stunted"
Month-to-month inflation while in utero	0.0050***	0.0064**
	(0.0013)	(0.0026)
Month-to-month inflation during infancy	−0.0003	−0.0030*
	(0.0007)	(0.0010)
Average NDVI while in utero	0.0002	0.0002
	(0.0003)	(0.0006)
Average NDVI during infancy	−0.0008**	−0.0020***
	(0.0004)	(0.0007)
Observations	11,748	11,741
R-squared	0.1950	0.1220
Age and month of birth dummies	X	X
Child characteristics	X	X
Household characteristics	X	–
Mother's characteristics	X	–
Father's characteristics	X	–
Geographic characteristics	X	–
Survey year–region interaction	X	–
Age–birth cohort interaction	X	X
Number of siblings	–	8,866

Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS), the monthly Ethiopian Central Statistical Agency (CSA) retail food price data from July 2001 to November 2012, and the monthly Normalized Difference Vegetation Index (NDVI) for Ethiopia between February 2000 and December 2011.

Note: Robust standard errors are in parentheses and clustered at market level. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. "Age and month of birth dummies" is a vector of age, age squared, and 11 dummies for the month of birth. "Child characteristics" is a vector of child-level control variables including a sex dummy, birth order, a dummy if the child has siblings, dummies for the mother's categorical subjective assessment of birth weight, a dummy for whether the mother received prenatal care, a dummy if the child was ever vaccinated, a dummy if they received Vitamin A, and duration of breastfeeding in months.

significant (e.g., exposure during the third month of pregnancy) become statistically significant in the fixed effects estimation that addresses survivor sample selection bias. Survivor sample selection creates bias by attenuating the coefficients downwards, and the unadjusted coefficients can be considered the lower bound.

Robustness Checks of Survey Timing and Age Misreporting

The results above could still be sensitive to potential age misreporting and survey timing.[20] The analysis observes that children measured during the first and last months of the survey periods indeed have a higher weight and a lower proportion of stunting in both waves. There seems to be a systematic relationship between stunting status and birth month. Children born in the rainy season (June, July, and August) have relatively low stunting rates.[21] In addition, given that Ethiopia follows a different, 13-month calendar

20 Agarwal et al. (2017) show that there is a spurious correlation between month of birth, age-at-measurement, and child growth patterns using DHS data. Similarly, Larsen, Headley, and Masters (2018) find that the misreporting of age attenuates the estimates on HAZ scores and stunting. In particular, it results in lower HAZ scores for children born earlier in the calendar year, around December and January, and children whose age is just below a rounded age due to rounding. These reporting problems introduce a nontrivial amount of bias on estimated stunting.

21 For the sake of brevity, this study does not show the figures reporting the correlation between survey timing and stunting status, but they are available from the authors upon request. Ethiopian DHS data field collection took place between April 27, 2005, and August 20, 2005, and between December 27, 2010, and June 3, 2010, for the 2005 and 2010 waves, respectively.

Table 5. Robustness Checks of Survey Timing and Month-Of-Birth Effects (OLS)

	(1) Control for interview month	(2) Control for interview month + excluding survey start and end months	(3) Control for interview month + excluding children born in June–November
Month-to-month inflation while in utero	0.00449***	0.00439***	0.00394**
	(0.00139)	(0.00142)	(0.00169)
Month-to-month inflation during infancy	−0.000375	−7.63e-05	−0.00186
	(0.000748)	(0.000780)	(0.00127)
Average NDVI while in utero	0.000178	1.81e-05	−0.000251
	(0.000358)	(0.000352)	(0.000728)
Average NDVI during infancy	−0.000897**	−0.000687	−0.000483
	(0.000447)	(0.000471)	(0.000738)
Observations	10,462	9,472	4,918
R-squared	0.140	0.140	0.165
Age and month of birth dummies	X	X	X
Child characteristics	X	X	X
Household characteristics	X	X	X
Mother's characteristics	X	X	X
Father's characteristics	X	X	X
Geographic characteristics	X	X	X
Survey year–region interaction	X	X	X
Age–birth cohort interaction	X	X	X

Source: Authors' analysis based on data from the 2005 and 2010 Ethiopian Demographic and Health Survey (DHS) and the monthly Ethiopian Central Statistical Agency (CSA) retail food price data from July 2001 to November 2012.
Note: Robust standard errors are in parentheses and clustered at market level. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. The OLS estimates include all the controls in table 2, specification (6).

system that begins in September and ends in August, with an additional month called "Puagme," it is possible that the age of children born in Puagme was misreported or rounded. Therefore, not controlling for the birth month and survey timing could introduce additional bias.

To check whether the results are sensitive to these problems, the analysis runs the model excluding children interviewed at the beginning and end of the survey periods: April and August 2005, and December and May 2010. It also excludes from the analysis children born in June–November. Table 5 presents the results. The coefficients and the standard errors remained the same (column 1), suggesting that the results are not biased due to possible measurement error arising from survey timing.

Similarly, when children born at the start and end of the survey period are excluded, the coefficients remain unchanged (column 2), although the magnitude decreases slightly (column 3) when the interview month is controlled for and children born in June–November are excluded. The standard error also increased slightly, presumably because about half of the observations were lost. Regardless, the main results still hold and remain robust to both survey-timing and month-of-birth bias.

6. Concluding Remarks

High food prices erode the purchasing power of poor households and undermine dietary quality and total energy intake, compromising child growth and cognitive development. This paper investigates the health consequences of exposure to food inflation during early life using data from the Ethiopian DHS and monthly local retail food price data. The study matches local food inflation with each child's early life months since conception. It finds that exposure to food inflation during early life has detrimental medium-term and long-term consequences on child health. The effects vary considerably depending on the month of exposure, with exposure to high inflation while in utero having a significant impact. The results also vary by observable factors such as the location of residency and land ownership.

The findings highlight that food inflation is not limited to being a traditional monetary policy concern. It has far-reaching consequences on child health, potentially undermining future health, educational achievement, job market outcomes, family formation, social behavior, and many other later-life outcomes. Given that many developing countries continue to experience high food inflation, this study underscores that these inflationary episodes are not just transitory and have lasting impacts on health. This has important implications for global policy dialogue on nutrition and the drivers of childhood stunting. If low-income countries in the developing world are to achieve the United Nations' Sustainable Development Goal of zero hunger for all by 2030, the negative impacts of food inflation must be considered, and remedial interventions must be put in place.

Considering the resource constraints that low-income countries face, the findings can help formulate well-targeted social safety net programs during periods of high food inflation. More specifically, inflation-targeted nutrition programs could be put in place to help pregnant women and infants. Such targeted interventions are common in developed countries. For instance, the United States Department of Agriculture—Food and Nutrition Service's Special Supplemental Nutrition Program for Women, Infants, and Children provides supplemental foods, health care referrals, and nutrition education for low-income pregnant, breastfeeding, and non–breastfeeding postpartum women as well as infants and children up to age five who are at nutritional risk. Although it is unrealistic to implement such wide-scale interventions or wait for the market to correct the problem in resource-poor countries, targeted nutrition-related intervention for pregnant women during inflationary episodes could be impactful.

7. Data Availability Statement

The data underlying this article are available at DOI:10.5281/zenodo.6543736.

References

Admassie, A. 2013. "The Political Economy Of Food Price: The Case Of Ethiopia." Working Paper 2013/001. UNU-WIDER. Helsinki, Finland.

Agarwal, N., A. Aiyar, A. Bhattacharjee, J. Cummins, C. Gunadi, D. Singhania, and M. Taylor. 2017. "Month of Birth and Child Height in 40 Countries." *Economics Letters* 157: 10–13.

Alderman, H., J. Hoddinott, and B. Kinsey. 2006. "Long Term Consequences of Early Childhood Malnutrition." *Oxford Economic Papers* 58 (3): 450–74.

Almond, D., and B. Mazumder. 2011. "Health Capital and the Prenatal Environment: The Effect of Ramadan Observance during Pregnancy" *American Economic Journal: Applied Economics* 3 (4): 56–85.

Arndt, C., M. A. Hussain, V. Salvucci, and L. P. Østerdal. 2016. "Effects of Food Price Shocks on Child Malnutrition: The Mozambican Experience 2008/2009." *Economics & Human Biology* 22: 1–13.

Barker, D. J. 1995. "Fetal Origins of Coronary Heart Disease." *British Medical Journal* 311 (6998): 171–74.

Barker, D. J. P., and P. M. Clark. 1997. "Fetal Undernutrition and Disease in Later Life." *Reviews of Reproduction* 2 (2): 105–12.

Barrett, C. B., T. Garg, and L. McBride. 2016. "Well-Being Dynamics and Poverty Traps." *Annual Review of Resource Economics* 8:303–27.

Benjamini, Y., and Y. Hochberg. 1995. "Controlling the False Discovery Rate: A Practical and Powerful Approach to Multiple Testing." *Journal of the Royal Statistical Society: Series B (Methodological)* 57 (1): 289–300.

———. 2000. "On the Adaptive Control of the False Discovery Rate in Multiple Testing with Independent Statistics." *Journal of Educational Behavior Statistics* 25 (1): 60–83.

Benjamini, Y., and D. Yekutieli. 2001. "The Control of the False Discovery Rate in Multiple Testing under Dependency." *Annals of Statistics* 29:1165–88.

Burgert, C. R., J. Colston, T. Roy, and B. Zachary. 2018. " Geographic Displacement Procedure and Georeferenced Data Release Policy for the Demographic and Health Surveys." Calverton, Maryland, USA: ICF International; 2013 (No. 7). DHS Spatial Analysis Report.

Case, A., and C. Paxson. 2010. "Causes and Consequences of Early-Life Health." *Demography* 47 (1): S65–S85.

Central Statistics Agency. 2009. Country and Regional Level Consumer Price Indices. Addis Ababa, Ethiopia

Currie, J., and D. Almond. 2011. "Human Capital Development Before Age Five." *Handbook of Labor Economics* 4: 1315–486.

De Waal, A., A. Taffesse, and L. Carruth. 2006. "Child Survival during the 2002–2003 Drought in Ethiopia." *Global Public Health* 1 (2): 125–32.

D'Souza, A., and D. Jolliffe. 2011. "Rising Food Prices and Coping Strategies: Household-Level Evidence From Afghanistan." *Journal of Development Studies* 48 (2): 282–99.

Dercon, S., and C. Porter. 2014. "Live Aid Revisited: Long-Term Impacts of the 1984 Ethiopian Famine on Children." *Journal of the European Economic Association* 12 (4): 927–48.

Duflo, E. 2000. "Child Health and Household Resources in South Africa: Evidence from the Old-Age Pension Program." *American Economic Review* 90 (2): 393–98.

Ford, N.D., J.R. Behrman, J.F. Hoddinott, J.A. Maluccio, R. Martorell, M. Ramirez-Zea, and A.D. Stein 2018. "Exposure to Improved Nutrition from Conception to Age 2 Years and Adult Cardiometabolic Disease Risk: A Modelling Study." *Lancet Global Health* 6 (8): e875–e884.

Hytten, F.E. 1954. "Clinical and Chemical Studies in Human Lactation—VIII." *British Medical Journal* 2 (4892): 844.

Kidane, D., and A. Woldemichael. 2020. "Does Inflation Kill? Exposure to Food Inflation and Child Mortality." *Food Policy* 92: 101838.

Larsen, A., D. Headey, and W.A. Masters. 2018. "Misreporting Month of Birth: Implications for Research on Nutrition and Early Childhood in Developing Countries." *Demography* 56 (2): 707–28.

Owens, J. A., P. C. Owens, and J. S. Robinson. 1989. Experimental Fetal Growth Retardation: Metabolic and Endocrine Aspects. In *Advances in Fetal Physiology. Reviews in Honor of G.C. Liggin*. Edited by P. Gluckman, B. Johnston and P. Nathanielsz, 263–86. Ithaca, NY: Perinatology Press.

Persson, P., and M. Rossin-Slater. 2018. "Family Ruptures, Stress, and the Mental Health of the Next Generation." *American Economic Review* 108 (4–5): 1214–52.

Rasmussen, K.M. 1992. "The Influence of Maternal Nutrition on Lactation." *Annual Review of Nutrition* 12: 103–7.

Rasmussen, K.M., and M.K. McGuire. 1996. "Effects of Breastfeeding on Maternal Health and Well-Being." *Food and Nutrition Bulletin* 17 (4): 1–7.

Rosso, P., G. Keyou, J.A. Bassi, and W.M. Slusser. 1981. "Effect of Malnutrition during Pregnancy on the Development of the Mammary Glands of Rats." *Journal of Nutrition* 111 (11): 1937–41.

Rouam, S. 2013. "False Discovery Rate (FDR)." In *Encyclopedia of Systems Biology*. Edited by W. Dubitzky, O. Wolkenhauer, K-H. Cho, and H. Yokota, 262–28. New York: Springer.

Shimeles, A., and A. Woldemichael. 2013. "Rising Food Prices and Household Welfare in Ethiopia: Evidence from Micro Data." *Working Paper* 182. African Development Bank. Abidjan, Côte d'Ivoire.

Tandon, S., and M. Landes. 2014. "Coping Strategies in Response to Rising Food Prices: Evidence from India." Economic Research Report 177. Economic Research Service. U.S. Department of Agriculture. Washington, DC: USA.

Victora, C.G., L. Adair, C. Fall, P.C. Hallal, R. Martorell, L. Richter, and H.S. Sachdev. 2008. "Maternal and Child Undernutrition Study Group." *Lancet* 371 (9609): 340–57.

WHO. 2006. " WHO Child Growth Standards: Length/Height-for-age: Weight-for-age, Weight-for-length, Weight-for-Height, and Body Mass Index-for-Age: Methods, and Development. Geneva, Switzerland: World Health Organization. https://apps.who.int/iris/handle/10665/43413.

Wooldridge, J.M. 2002. "Inverse Probability Weighted M-Estimators for Sample Selection, Attrition, and Stratification." *Portuguese Economic Journal* 1 (2): 117–39.

Wu, G., F.W. Bazer, T.A. Cudd, C.J. Meininger, and T.E. Spencer 2004. "Maternal Nutrition and Fetal Development." *Journal of Nutrition* 134 (9): 2169–72.

Yamano, T., H. Alderman, and L. Christiaensen. 2005. "Child Growth, Shocks, and Food Aid in Rural Ethiopia." *American Journal of Agricultural Economics* 87 (2): 273–88.