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Research Article

Child anemia and the 2008 food price crisis in Senegal

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Jesse McDevitt-Irwin[1]

Abstract

BACKGROUND
In 2008, world food prices skyrocketed. There is little consensus on the effect of the 2008 food price crisis on poverty, food security, and population health.

OBJECTIVE
To estimate the effects of the 2008 crisis on maternal nutrition and child anemia in Senegal.

METHODS
Child hemoglobin reflects in utero iron deposition, making it a biomarker for maternal nutrition. By comparing the hemoglobin of children in utero during the 2008 crisis to the hemoglobin of those who were breastfeeding, I estimate the impact of the 2008 crisis on maternal nutrition and child anemia.

RESULTS
The 2008 crisis caused child hemoglobin measures to deteriorate in urban Senegal. The effect was largest in Dakar, where the magnitude (a 10% drop in hemoglobin) would imply an increase in the prevalence of childhood anemia from one in three children to three in four children. I find little to no impact in rural areas.

CONCLUSIONS
The 2008 food price crisis had a large negative impact on child hemoglobin in urban Senegal, likely through a deterioration of maternal nutrition. There was no offsetting improvement in rural areas, meaning that the net effect of the 2008 crisis on Senegal was to substantially increase child anemia.

CONTRIBUTION
There is continued debate over the effect of food price spikes on the world's poor. With a novel empirical framework, I leverage child hemoglobin as a biomarker of iron nutrition during pregnancy, finding clear evidence of a large negative impact of the 2008 food price crisis on maternal nutrition and child anemia in Senegal. This methodology could be applied more generally, as Demographic and Health Survey data on child hemoglobin is available for a wide range of populations.

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1. Introduction

In 2008, food prices around the world skyrocketed. On the international market, the price of rice tripled from January to April (FRED 2021).[2] In poor countries, where food expenditures often make up more than half of household expenditures (Seale Jr., Regmi, and Bernstein 2003), purchasing power collapsed as prices rose. The 2008 world food price crisis has drawn attention from the natural sciences (Moseley, Carney, and Becker 2010; Swinnen and Squicciarini 2012), economics (Benson, Mugarura, and Wanda 2008; D'Souza and Jolliffe 2014), demography (Alexandratos 2008; Naylor and Falcon 2010), food studies (McMichael 2009b; Kumar and Quisumbing 2013), nutrition (Brinkman et al. 2010; Martin-Prevel et al. 2012), peasant studies (Hossain and Kalita 2014; Bohstedt 2016), and development studies (Rosset 2008; Wodon and Zaman 2010). Recent food price spikes, such as those caused by the COVID-19 pandemic (Laborde et al. 2020) and the Russian invasion of Ukraine (Mottaleb, Kruseman, and Snapp 2022), have renewed interest in the 2008 crisis.

The populations of low- and middle-income countries (LMICs) were particularly affected by the 2008 crisis. The sudden nature of the crisis, impacting much of the world at the same time, has made it difficult for researchers to estimate the effect of the crisis on health and poverty. This lack of clear evidence has left a debate over the severity of the 2008 crisis for the world's poor. While some emphasize its negative impact on the urban poor (Moseley, Carney, and Becker 2010), others argue that the crisis raised income in rural areas, under the assumption that households in these areas typically sell excess foodstuffs (Aksoy and Isik-Dikmelik 2008).

I develop a novel empirical strategy, using child hemoglobin as a biomarker of iron deposition during gestation, allowing me to quantify the effects of the 2008 crisis on maternal nutrition and child anemia in Senegal. The 2008 crisis had a large effect on child hemoglobin in urban Senegal, likely reflecting maternal iron depletion due to worsening diets. The effect of the 2008 crisis was large enough to cause a doubling of child anemia in Dakar, from 35% to more than 70%. In Senegal as a whole, the effect implies a 30% increase in child anemia, from 47% to 64%, with the effects concentrated in urban areas.

Senegal is emblematic of today's challenges for population health and food security in West Africa. It has a young, rapidly urbanizing population, without substantial economic growth or industrialization. As urbanization continues, Senegal's vulnerability to such crises as 2008 will only increase.

[2] The exact causes of the crisis are unclear, but they include financial speculation and export restrictions (Headey and Fan 2008).

2. Background

2.1 Senegal

Senegal is in the Sahel, on the west coast of Africa. With a GDP per capita of less than $2,000 (current USD; World Bank 2024), Senegal is a low-income country and is highly dependent on imported foodstuffs. Over the last 15 years, Senegal has seen limited economic growth but substantial demographic expansion.[3] Half of Senegal's population lives in rural areas (World Bank 2024), where subsistence agriculture predominates (WFP 2018). Senegalese society is marked by a high degree of mobility, including both internal migration (Delaunay et al. 2016; Herrera-Almanza and Sahn 2020) and emigration (Willekens, Zinn, and Leuchter 2017). Malnutrition is widespread in Senegal. More than half of women aged 15–49 suffer from anemia (Demographic and Health Survey data), and more than a fifth of children are stunted (Nene 2018).

Senegal imports 70% of its food supply (USDA Foreign Agricultural Service 2019), and the 2008 food price crisis led to widespread protests and unrest in Senegal and across West Africa (Le Monde 2008). This dependence on imported foodstuffs stems from French colonial and neocolonial policy, with a characteristic pattern of trade – importing rice and exporting peanuts – established by the turn of the 20th century (Bonnefond and Couty 1988). Today, urbanization drives Senegal's continued increase in food imports.[4] In 1960, one in five Senegalese lived in cities; that number is one in two today (World Bank 2024). This transformation has been accompanied by a "nutrition transition" (Popkin 1999), in which the population has shifted away from locally produced coarse cereals (especially millet) toward imported foodstuffs, especially rice (Ndoye et al. 2001) and wheat (Tikum 2022). This shift away from agriculture has reduced Senegal's vulnerability to drought,[5] as fewer people depend on rain-fed agriculture for their livelihood. At the same time, this urbanization has increased Senegal's vulnerability to commodity price volatility, as a growing number of people depend on international markets to meet their basic food needs.

The price of rice doubled in Dakar over the first half of 2008 (Figure 1). Most households in Senegal spend over half their budget on food (Figure A-1). In a typical household, rice purchases alone make up about 20% of food expenditures (about 10% of total expenses). Rice is a highly valued staple, whose importance has increased over time at the expense of more traditional crops, such as millet (Ndoye et al. 2001). Rice consumption increases with income but decreases as a share of food expenditures. Richer

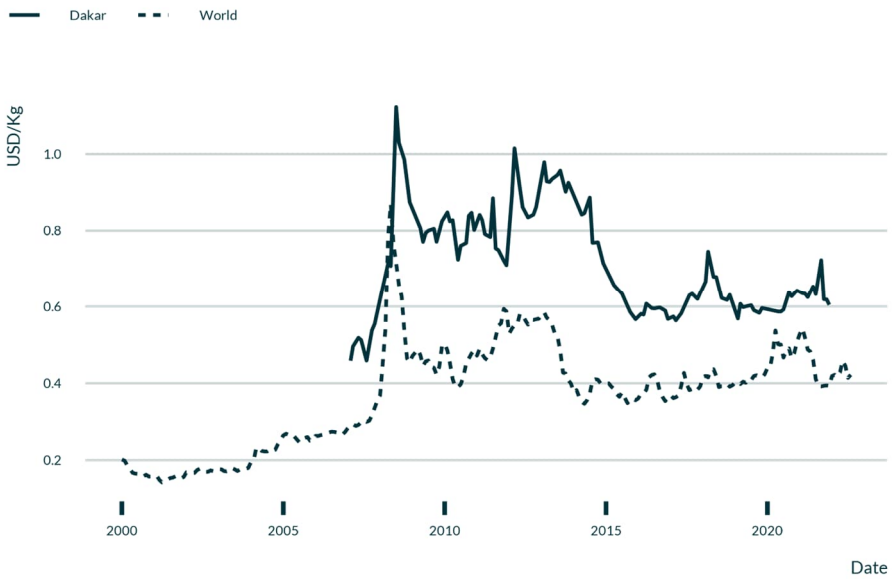
[3] According to the World Bank, Senegal's GDP per capita went from $1,420 in 2008 to $1,598 in 2022. Total GDP grew by 50%, driven largely by population growth.

[4] The specific pattern of urbanization in Senegal – largely concentrated in Presqu'île du Cap-Vert (Dakar) – is also due to French colonial policies. For discussion, see, for example, Wane (1985), Nelson (2007), and Harris (2011).

[5] See Grace et al. (2021), for example, on the effect of local weather shocks on child health in neighboring Mali.

households diversify their diets into more expensive foods like meat and fish (Ndoye et al. 2001). The 2008 price spike thus likely had the greatest effect on the urban poor, who lack both the financial security of richer urban households and the home-produced food of rural classes.

Figure 1: Rice prices in Dakar and the World Market



Notes: World price data from FRED Economic Data, world price of rice (series PRICENPQUSDM), accessed September 19, 2022. Dakar price data from FAO FPMA tool, accessed September 19, 2022.

2.2 Literature

There has been more attention than consensus on the consequences of the 2008 food price crisis for the world's poor. Contemporary journalism has focused overwhelmingly on the negative consequences of the 2008 price spike (e.g., Adam 2008; Bernard and Tuquoi 2008). Similarly, Moseley, Carney, and Becker (2010) take the negative impacts of the food price spike as given. They blame decades of "neoliberal policy reform" for reducing support for West African farmers, leading to further urbanization and dependence on imports. This perspective is shared by Rosset (2008), Mittal (2009), and McMichael (2009a, 2009b).

This view is supported by evidence from food security surveys and child anthropometric data. Food security surveys show a deterioration of diet quality over the course of 2008 in several countries around the world, with a shift away from nutrient-rich foods but no change in quantity (overall calories) consumed.[6] Households protected their consumption of basic staples like wheat and rice by reducing consumption of expensive, nutritious foods like fish and meat. Child anthropometrics also show a negative effect of the 2008 crisis on child health. De Brauw (2011), Galab and Reddy (2013), Arndt et al. (2016), Lazzaroni and Wagner (2016), and Woldemichael, Kidane, and Shimeles (2017) all find that food price increases early in childhood were associated with increased wasting and/or stunting.

In contrast, results from household welfare analyses of the 2008 crisis are less clear.[7] Some authors highlight negative impacts of the 2008 price increase on household welfare (Ivanic and Martin 2008; Wodon and Zaman 2010; De Hoyos and Medvedev 2011; Ferreira et al. 2013; Balagtas et al. 2014; Hasan 2017). Others argue that the *net* effects of the 2008 crisis on poverty, once we account for gains to rural food producers, were small (Mghenyi, Myers, and Jayne 2011; Dessus, Herrera, and De Hoyos 2008; Benson, Mugarura, and Wanda 2008). Furthermore, because rural food producers are on average poorer than city dwellers, the 2008 food price increase may have been "progressive," raising rural income from the sale of excess foodstuffs and reducing the gap between rural and urban households (Aksoy and Isik-Dikmelik 2008; Dimova and Gbakou 2013; Minot and Dewina 2015; Van Campenhout, Pauw, and Minot 2018). Dimova and Gbakou (2013) query whether the 2008 price increase was a "crisis" at all or instead an "opportunity" or a "non-event." This question has created a setting of mixed messages around food prices and poverty (Swinnen and Squicciarini 2012).[8]

Quantifying the health impacts of the 2008 crisis could resolve some of the debate over its impacts on the well-being of the world's poor. Because the 2008 crisis affected entire populations, we cannot easily define a treated and a control group, and existing empirical evidence is adjustment based. My approach does not require such adjustments, as I leverage child hemoglobin as a biomarker of maternal iron nutrition during pregnancy and compare children in utero during the crisis to those already born.

Understanding the impacts of the 2008 crisis on maternal nutrition and child health is particularly important because of the importance of the first 1,000 days for life cycle health and human capital. A wide body of research has shown that gestation and early childhood are critical periods of human development (Almond and Currie 2011; Stein et al. 1975; Lynch and Smith 2005; Gillman 2005). Any negative impacts of the 2008 crisis would be felt throughout the life cycle of children who were exposed in utero.

[6] See Hadley et al. (2011) and Kumar and Quisumbing (2013) for evidence from Ethiopia, Martin-Prevel et al. (2012) for Burkina Faso, and D'Souza and Joliffe (2012, 2014) for Afghanistan.
[7] By "household welfare analysis," I mean the style of household budget analysis stemming from Deaton (1989).
[8] Welfare analysis based on budget data does not easily translate to health, as its methods assume the equivalence of income losses and gains, and ignore the intra-household effects of price changes.

2.3 Iron and pregnancy

Iron is essential to the formation of hemoglobin, the oxygen-transport protein that facilitates aerobic respiration. Anemia is characterized by low levels of hemoglobin and is most often due to iron deficiency (Kassebaum et al. 2014). For women, anemia causes fatigue and increases the probability of death during childbirth (Camaschella 2015). For infants, iron is a critical nutrient, and anemia can impair cognitive and motor development (Walter 2003).[9]

The amount of hemoglobin in blood is limited by iron availability, both for children and adults.[10] During pregnancy, the placenta transports iron to the fetus (Gambling, Lang, and McArdle 2011). The development of fetal hemoglobin is sensitive to maternal hemoglobin, with anemic mothers giving birth to anemic infants (Akhter et al. 2010). There is little iron in breast milk (Domellöf 2007), and the breastfed infant relies on stores of iron accumulated during gestation (Friel, Qasem, and Cai 2018). The amount of iron in breast milk is insensitive to maternal hemoglobin (Loh and Sinnathuray 1971), except in cases of severe anemia (Kumar et al. 2008).

A change in maternal diet, and therefore iron availability, should therefore influence iron stores of infants in utero during this change but not iron stores of infants who were breastfeeding. Breastfeeding is nearly universal in Senegal, as seen in Table 1. However, exclusive breastfeeding typically lasts only three months, and predominant breastfeeding lasts around seven months. Those cohorts born just before the 2008 crisis, and therefore breastfeeding during the crisis, should be, in terms of iron, insulated from its effects. The cohorts in utero during and after the crisis should have been affected, as depletion of maternal iron stores would result in less iron being transported to the fetus. This difference offers an empirical framework for evaluating the effect of the 2008 crisis on maternal nutrition. Because the breastfeeding children would have had some complementary foods introduced, they may still have been impacted by the crisis as the family cut back on nutritious food. If this is the case, it would mean that my results underestimate the true effect of the crisis on child hemoglobin, as the control (breastfeeding) group was also affected, but less so than the treated (in utero*)* group.

[9] Maternal and infant hemoglobin have been used as outcomes in a wide range of social science research. See, for example, Block et al. (2004), Kumar, Molitor, and Vollmer (2016), Sunder (2019), Von der Goltz and Barnwal (2019), or Yue et al. (2020).

[10] Lack of available iron can come from depletion of iron stores but also from increased iron sequestration due to inflammation (Camaschella 2015). In this paper I focus on the former.

Table 1: Breastfeeding patterns in Senegal, DHS data

	Children breastfeeding at 1 year (%)	Introduction of foods, 6–8 months (%)	Predominant breastfeeding, 0–5 months (%)	Mean duration of exclusive breastfeeding (months)	Mean duration of predominant breastfeeding (months)
Survey	Total	Total	Total	Total	Total
2012–13 DHS	97	65	86	2.8	6.8
2010–11 DHS	97	67	80	3.2	6.8
2005 DHS	93	59	76	3	6.7

Source: ICF 2015; DHS program STATcompiler, http://www.statcompiler.com, March 6, 2024.

3. Data

I use data from the Demographic and Health Survey (DHS) and the Malaria Indicator Survey (MIS) in Senegal, which give repeated cross sections of children under the age of 5 and women aged 15–49. For child hemoglobin data, I use standard DHS data (2005, 2010), MIS data (2008, 2020), and continuous DHS surveys (2012–2017). I exclude the 2018 and 2019 continuous DHS surveys because they lack anemia testing.

Table 2: Data sources for child anemia

Analytic Sample	Use	N (total)	N (analysis)
2005 DHS	Reference group	14,472	3,581
2008 MIS	Reference group	15,595	3,656
2010 DHS	Cohorts of interest and reference group	12,326	3,293
2012–2016 continuous DHS	Reference group	27,364	21,202
2017 continuous DHS	Reference group	12,185	9,558
2020 MIS	Reference group	6,395	2,562

For my analyses, I break the child sample into three groups: those who were in utero during the 2008 crisis, those who were breastfeeding, and everyone else. The first and second group are my cohorts of interest, and I infer the effect of the 2008 crisis from the difference between their hemoglobin levels. The rest of my sample acts as a reference group. The reference group is by far the largest (42,540 children) and allows me to include various controls in the regression (particularly child age) without using up the statistical power of my small subsample of interest (1,312 children). This greater sample size is particularly important when adjusting for the effect of child age on measured hemoglobin, which requires a flexible functional form.

Hemoglobin data are missing for a substantial part of the population, some 60,000 of the 100,000 children in all DHS and MIS surveys from 2005 to 2020. For all surveys, children under the age of six months were not tested. This precludes using the 2008 child sample for my cohorts of interest, although I do use the women's sample later. Note that

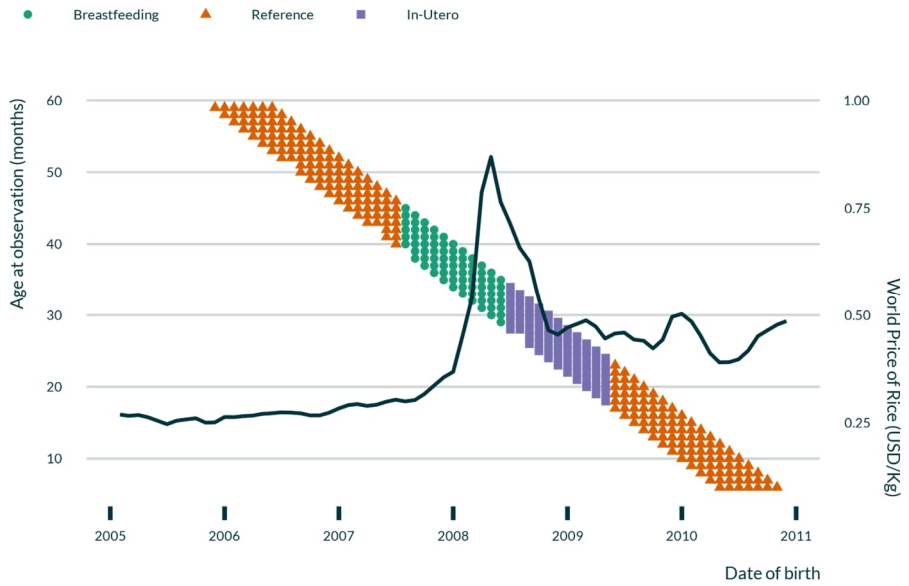
the 2005, 2008, 2010, and 2020 samples have many more missing values than other years due to subsampling. As noted in the 2010 DHS final report (ANSD and ICF 2012: 307), only a subsample of households was selected for anemia testing, the same as the men's survey. One household was selected for every two in the broader sample. The same was true for 2005, except that the subsample was one in three households rather than one in two (Ndiaye and Ayad 2006: 5 and 321). In the 2008 MIS survey, one in every three sampled households was tested for anemia (Ndiaye and Ayad 2009: 67). For 2018 and 2019, no anemia testing was performed. For 2020, only the southeast of the country was given hemoglobin tests, in accordance with malaria prevalence (ANSD and ICF 2022: Figure 4.3).

I also draw on household living standards surveys from the Senegalese government and local price data from the FAO FPMA to construct contextual variables. The household living standard surveys are the Enquête de suivi de la pauvreté (EPS) 2005 and 2010 and are nationally representative. I do not use these data in regression analyses. I use them to provide greater detail on the socioeconomic context of Senegal.

4. Empirical strategy

The hemoglobin of infants in utero during the crisis should have been sensitive to changes in maternal nutrition, unlike that of infants who were breastfeeding. I take the in utero cohort as my treated group and those who were breastfeeding as the control. These cohorts are mapped out with reference to international rice prices in Figure 2. I define in utero during the crisis as being born between June 2008 and April 2009. I define breastfeeding during the crisis as being born between July 2007 and April 2008. I use only observations from the 2010 survey for these cohorts of interest. They also appear in 2012, but after several years, the effect of the 2008 crisis on child hemoglobin likely had faded away. The rest of my sample – observations from the 2010 survey who do not fall in those birth windows, as well as observations from all other samples (2005, 2008, 2012–2017, and 2020) – is included as a reference group. I use this third group to calibrate the effect of relevant covariates, especially child age, as hemoglobin follows a strong age path (see Figure A-3). I also include such controls as household wealth and mother's age and education. As seen in Table 3, my cohorts of interest are balanced across these other covariates, but controlling for them should improve the precision of my estimates.

Figure 2: Birth month cohorts from Senegal 2010 DHS by exposure to 2008 crisis



Notes: Birth cohorts are colored by their month of birth relative to the 2008 crisis (see text for further discussion). Data from the 2010 Senegal DHS. The world price of rice is plotted against the right-side axis for reference.

Table 3: Balance of treatment; comparisons of those in utero during the crisis to those breastfeeding and to the reference group

Treatment	Urban	Wealth (Standardized)	Primary+	Child Age (Months)	Mother's Age (Years)
Reference	29%	–0.22	29%	31	30
In utero	31%	–0.09	27%	26	29
Breastfeeding	31%	0.00	25%	37	30

Notes: Wealth is the DHS household wealth index, standardized by subtracting the mean and dividing by the standard deviation. Primary+ is the proportion of children whose mothers have completed at least primary school.

In my main regression specification, I allow the effect of the 2008 crisis – the difference in hemoglobin between children who were in utero versus children who were breastfeeding – to differ by residence (Dakar, rest of urban Senegal, rest of rural Senegal). I define a categorical variable "treatment" and let the breastfeeding group be the left-out category.

$$hemo_i = \beta_{1,j} + \beta_{2,j} utero_i + \beta_{3,j} Ref_i + g(age_i) + X_i + \epsilon_i \quad (1)$$

where $\beta_{2,j}$ are the coefficients of interest, j denotes residence, $hemo_i$ is the hemoglobin of child i, $g(age_i)$ is a cubic spline of age in months, and X_i is a vector of covariates (household wealth, mother's education, and mother's age).

The assignment of in utero and breastfeeding during the crisis is, of course, arbitrary when using a binary variable. Some children were exposed in utero to the crisis, but for only one or two months. As an alternative to this binary measure of exposure, I also construct a data-driven continuous measure of in utero exposure to high rice prices, based on local market data available from the FAO FPMA tool.[11] This alternative specification also allows the timing of the 2008 crisis to differ by region, as it took time for the international price spike to disseminate into rural areas of Senegal. I find similar results using both the binary and continuous approaches. I present the binary results for ease of interpretation; see the appendix (Table A-1) for results from the continuous price exposure specification.

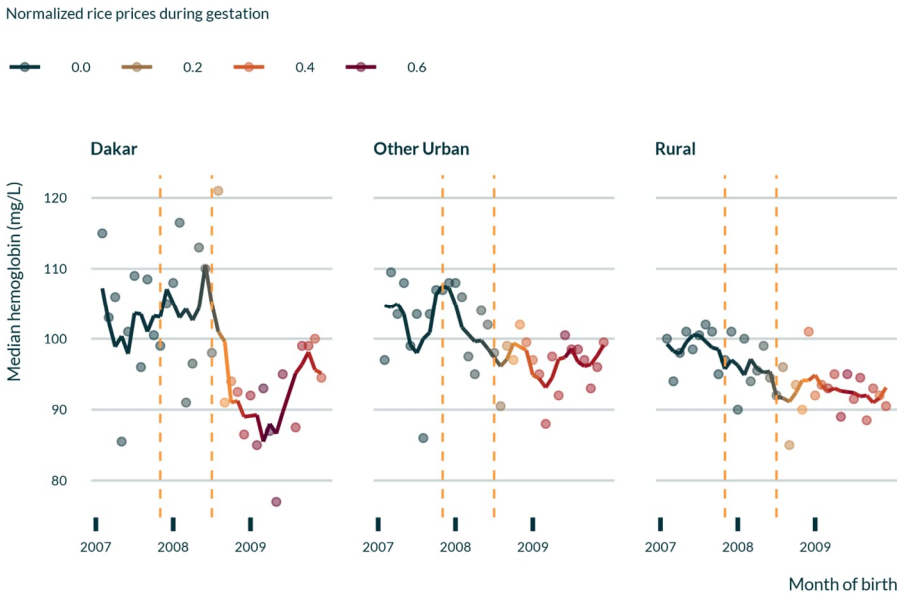
5. Results

I present median hemoglobin by birth cohort, split by residence (Dakar or rest of Senegal) in Figure 3. June 2008, when rice prices spiked in Dakar, is represented by a dashed vertical line. Another dashed line represents October 2007. The cohorts born between the dashed lines were breastfeeding during the crisis. The onset of the crisis coincides with a sharp decline in hemoglobin in the Dakar population. The points are colored by in utero price exposure (described above). We see that the deterioration of child hemoglobin in Dakar is strongest in the cohorts with the greatest in utero exposure to the 2008 rice price increases. The cohorts with the highest levels of hemoglobin are those who were breastfeeding during the crisis. The deterioration in the rest of Senegal is much smaller than in Dakar.

The data suggest that the 2008 crisis had a substantial effect on child hemoglobin in Dakar. To quantify this effect, I estimate Equation (1), allowing for the treatment effect to vary for three subsets of Senegal: Dakar, rest of Senegal (ROS) urban, and ROS rural. The estimated levels of hemoglobin across treatment status and effects are plotted in Figure 4. The 2008 crisis was associated with a large deterioration in child hemoglobin in Dakar, a smaller one in the rest of urban Senegal, and no effect in rural Senegal. In Table 4, we see that adding additional controls, household wealth and mother's age and education, does not meaningfully change the estimated effect of the 2008 crisis on child hemoglobin.

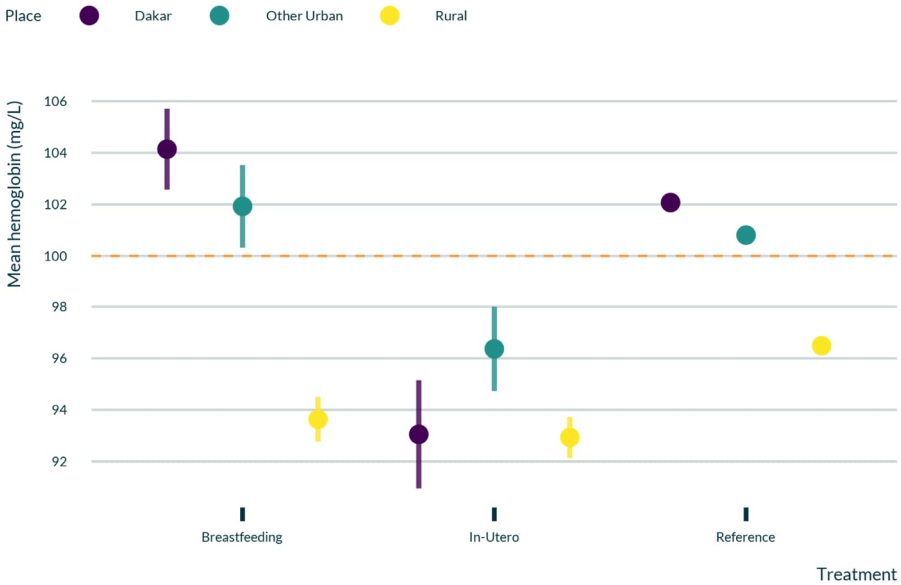
[11] Available at https://fpma.fao.org/giews/fpmat4/#/dashboard/home, accessed September 19, 2022.

Figure 3: Hemoglobin by birth cohort and residence in Senegal, 2010 DHS data



Notes: Median hemoglobin in Senegal 2010 by birth month cohort, divided by residence. Points are the values, and lines are the rolling mean, centered with a window of four months. Control cohorts (breastfeeding during crisis) are between the dashed lines; treated cohorts (in utero during crisis) are to the right of the second dashed line. Color represents mean local rice prices during gestation. Hemoglobin data are from the 2010 standard DHS in Senegal (see Table 2). Price data are from the FAO FPMA tool (see note 11).

Figure 4: Estimated hemoglobin by residence and treatment status



Notes: Mean hemoglobin by treatment group and residence, adjusted for child age. Vertical lines are standard errors. Data are from the Senegal DHS and MIS; see Table 2. The horizontal line at 100 mg/L is a cutoff for moderate to severe anemia. The breastfeeding and In utero groups come from the 2010 Senegal DHS and are defined by their age at the time of the 2008 crisis. The reference group contains observations from all other survey years, as well as those observed in 2010 who were more than nine months old at the time of the crisis. Those in the breastfeeding group were nine months old or younger when the crisis hit Senegal, and those in the in utero group were in gestation.

Table 4: Child hemoglobin regressed on binary treatment definition, interacted with residency

	Model 1	Model 2
In utero	–11.09 (2.63)	–10.94 (2.54)
Other urban: in utero	5.53 (3.50)	5.48 (3.39)
Rural: in utero	10.39 (2.89)	10.49 (2.79)
Reference	–2.07 (1.61)	–2.20 (1.58)
Wealth		2.47 (0.11)
Primary+		1.23 (0.22)
Mother's age		0.97 (0.18)
N	43,852	43,852

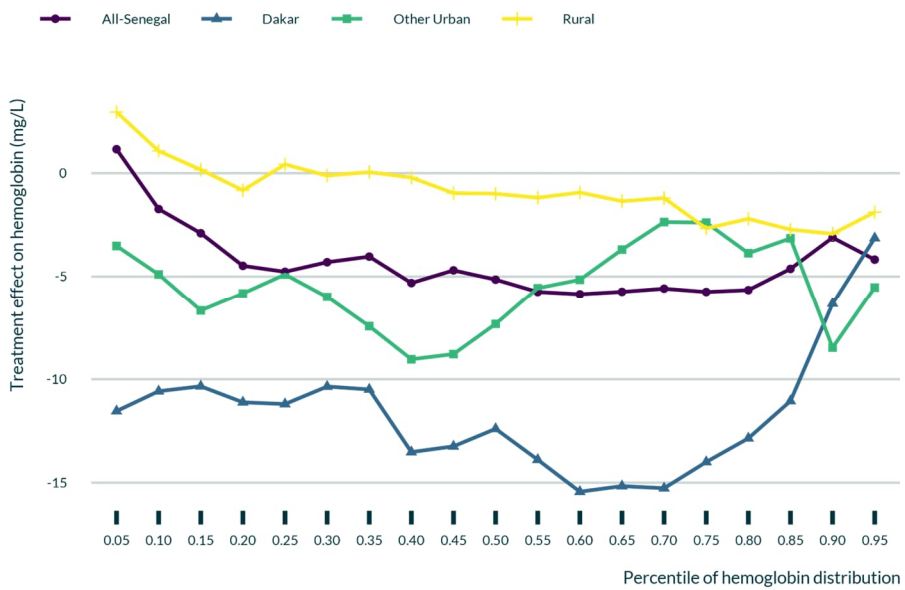
Notes: Child hemoglobin regressed on binary treatment definition, interacted with residency. Breastfeeding during crisis is the excluded group. Dakar is the default residency group. Least-squares estimation of Equation (1). Model 1 adjusts only for age, while Model 2 includes other controls. Data from Senegal DHS and MIS; see Table 2. Standard errors are heteroskedasticity robust.

5.1 Quantile regression and implications for anemia

To quantify the effect of the 2008 crisis on anemia, I use quantile regression. I estimate Equation (1) for Senegal as a whole and then separately for the Dakar, other urban, and rural groups. I remove all controls except for child age to make inferences about the effect of the 2008 crisis on anemia in the broader population.[12] I estimate the regression at each quantile.

The results follow the same patterns as above, with the effect of the 2008 crisis largest in Dakar, followed by urban Senegal, and with no clear effect in rural areas. The effects at the extremes of the distribution are smaller than in the center (Figure 5), likely because of attrition at the left tail (mortality) and the ability to consumption smooth at the right tail (as high-hemoglobin children are likely to come from food-secure families).

Figure 5: Treatment effect by quantile



Notes: Quantile regression results by residence. Effect of exposure to the 2008 crisis in utero on child hemoglobin by quantile of dependent variable. Conditioned on child age. Data from the Senegal DHS and MIS (see Table 2).

[12] I am interested in the treatment effect on the unconditional distribution rather than the conditional distribution. See Killewald and Bearak (2014) for a discussion of conditional versus unconditional quantile effects.

I then take the rest of my sample (2005 and 2012–2020) and sort observations into percentiles. For each observation I apply the estimated treatment effect from the corresponding quantile regression – so an observation in the 20th percentile of the anemia distribution in Dakar would be given the treatment effect of the 20th percentile of the Dakar regression (the second point from the right in the Dakar line in Figure 5).

Applying the effect of the 2008 crisis to the rest of my sample (2005 and 2012–2020) implies a 30% increase in child anemia in Senegal across these years: from 47% to 63%.[13] For Dakar, my estimates imply an increase in child anemia from 33% to 76%. Repeating this exercise in the rest of urban Senegal, I find a one-third increase in anemia, from 39% to 56%. In rural Senegal, the effect is small, with anemia increasing from 51% to 56%.

The 2008 crisis had a large effect on child hemoglobin in urban Senegal. This effect was strongest in Dakar, where it implied a 10% decline in mean hemoglobin. Based on quantile regression results, this translates into more than a doubling of the prevalence of anemia, from about one in three children to three in four. The effect was smaller in the rest of urban Senegal and was close to null in rural areas. Note that these calculations are based on the thought experiment of applying the effect of the 2008 crisis on those in utero to the entire age distribution. They do not imply that the actual prevalence of anemia increased by this much; rather they speak to how much anemia would have increased if the entire population were submitted to the shock experienced by children exposed in utero to the 2008 crisis.

5.2 Maternal hemoglobin

The affected children were in utero at the time of the crisis, suggesting that maternal iron depletion was responsible for their low hemoglobin. I cannot directly test for maternal iron depletion, as there is no continuous data on women's hemoglobin in Senegal over the period, but I compare hemoglobin levels in 2005 to those in 2008, 2010, and 2017.

Figure 6 plots median hemoglobin of women in Senegal aged 15–45 for 2005, 2008, 2010, and 2017. Women's hemoglobin was not recorded for the 2012–2016 DHS surveys. In 2005 women's hemoglobin was highest in Dakar, followed by other cities in Senegal, and was lowest in rural areas. Between 2005 and 2008, women's hemoglobin deteriorated in urban Senegal, particularly in Dakar, while improving slightly in rural areas. The 2005 order returns only in 2017. These patterns support the proposed mechanisms of maternal iron depletion leading to reduced iron accumulation in utero for affected cohorts and therefore lower iron stores in early childhood.

[13] I use 100 mg/L of hemoglobin as a cutoff for moderate to severe anemia, as in Wang et al. (2015).

Figure 6: Mean hemoglobin in Senegal: Women aged 15 to 45

Note: Mean hemoglobin among women aged 15–45 in Senegal, by residence. Data from DHS 2005, 2010, and 2017, and MIS 2008.

6. Discussion and conclusions

The legacy of the 2008 food price crisis remains contentious. Even the label "crisis" implies a critical juncture or choice, in contrast to more unambiguously negative terms like "disaster" or "catastrophe" (Koselleck 2006). As outlined above, existing academic writing on the 2008 crisis fits into two broad narratives: (1) that it had large deleterious effects on urban populations in LMICs and (2) that its net effects in poor countries were small, because urban losses were offset by rural gains. My results support the first narrative, as I find large negative impacts of the 2008 crisis on child hemoglobin in urban Senegal but no evidence of corresponding improvements in rural areas.

A key limitation of my approach is the degree of statistical power and precision. By focusing exclusively on children who were either very young or not yet born at the time of the 2008 crisis, I drastically limit the sample size of my comparison groups. In my regressions, the two groups of interest (breastfeeding and in utero) contain about 650 children each. Once broken apart by place of residence, the samples are even smaller.

Dakar, for example, has only 100 children in the groups of interest. Strictly limiting these groups allows me to estimate the effect of the 2008 crisis with minimal adjustments, as the only variable that differs systematically between my groups of interest is child age (see Table 3). But limiting my sample also reduces the precision of my estimates, raising the concern of magnitude error (Gelman and Carlin 2014): estimates that are much larger than the true effects due to a lack of statistical power. One way to address this concern would be to apply the same methodology across a broader set of countries affected by the 2008 crisis to see if the estimated effect size is similar across contexts.

My work complements existing studies on child height and weight during the 2008 crisis, which find that both stunting and wasting increased following price shocks in various LMICs (de Brauw 2011; Galab and Reddy 2013; Arndt et al. 2016; Lazzaroni and Wagner 2016; and Woldemichael, Kidane, and Shimeles 2017). I use a new indicator: child hemoglobin. Although child hemoglobin has been used as an indicator of maternal and child nutrition in other contexts (e.g., Block et al. 2004; Yue et. al 2020), mine is the first study to examine it in the context of the 2008 food price crisis.

Hemoglobin, being determined by iron intake, is particularly useful as an indicator of diet quality, not just quantity. This makes hemoglobin an ideal indicator for testing the implications of existing research on food security surveys, which find that during the 2008 crisis, households cut back on expensive, nutritious ingredients while maintaining overall caloric intake (Hadley et al. 2011; Martin-Prevel et al. 2012; Kumar and Quisumbing 2013; D'Souza and Jolliffe 2012, 2014). Indeed, my findings strongly support these studies, as I see a substantial deterioration of child hemoglobin for those who were exposed to high food prices in utero. This change was likely due to a reduction in heme iron intake among mothers, as families reduced consumption of meat and fish in response to higher rice prices.[14]

While my results speak directly to child anemia, they also have implications for life course health and human capital. The first 1,000 days are a crucial phase in human development (Almond and Currie 2011), and iron is a critical nutrient in motor and cognitive development (Lozoff et al. 1998; Walter 2003). The effects of the 2008 crisis are likely still felt today by those whose mothers were affected, contributing to the reproduction of social inequalities (Palloni 2006). Similar effects will likely be seen around the world due to recent food price crises. Child hemoglobin measures, widely available from the DHS, could shed light on the consequences of food price spikes for the nutrition and health of vulnerable mothers and children.

Existing work on household welfare has argued that higher food prices would increase incomes in rural areas, where food production takes place (Dessus, Herrera, and De Hoyos 2008; Benson, Mugarura, and Wanda 2008; Aksoy and Isik-Dikmelik 2008; Mghenyi, Myers, and Jayne 2011; Dimova and Gbakou 2013; Minot and Dewina 2015;

[14] Leport (2017) notes a similar process over a longer period, where households in Dakar respond to the increasing scarcity of fish by reducing its quantity in rice-based dishes without reducing the quantity of rice consumed.

Van Campenhout, Pauw, and Minot 2018). I find no evidence of any positive effects of the 2008 crisis on child hemoglobin in rural areas. This is likely because of the class structure of rural Senegal: The vast majority of rural households in Senegal do not sell staple goods, although they do produce them. According a 2010 household living standard survey conducted by the Senegalese government (EPS 2010), 20% of agricultural households in Senegal produce some rice. Only 30% of these households sell rice.[15] Sale of staple goods is simply not a part of most household livelihoods in Senegal, reflecting the moral economy of staple goods.[16] On the contrary, nearly all agricultural households in Senegal (93%) purchase rice, suggesting that they might have suffered from the 2008 rice price increase.

However, I also find no evidence of negative effects of the 2008 food crisis on rural households. This is likely because rural households in Senegal have some control over the means of food production – arable land – giving them relative autonomy. Urban classes, on the other hand, depend on market relations to meet their basic food needs, making them particularly sensitive to shifts in exchange relations, such as food price spikes.[17]

The ongoing class transformation in Senegal, with a growing proportion of urban laborers, will increase vulnerability to crises like that of 2008, as fewer people will control their own means of food production. The same transformation is occurring across much of sub-Saharan Africa, driven by structural economic change (Mercandalli and Losch 2017), demographic expansion (Fox 2012), and urban population growth (Parnell and Walawege 2011). Senegal, in being more urbanized than neighboring countries, is not an exception but a portend.

Senegal's vulnerability to world food price volatility is well-known by the local population, both in the streets and in government. After the 2008 crisis, the national government (under President Macky Sall) made massive investments in rice production in the Senegal River valley, hoping to reduce staple import dependency (Demont and Rizzotto 2012). While these investments have increased rice production, they have failed to reduce imports. Moreover, over the last 20 years, Senegal has developed a similarly large dependency on imported wheat (FAO 2020). These joint import dependencies leave the Senegalese food system even more vulnerable to international economic and geopolitical volatility today than it was in 2008.

Sen (1982: 173) observed half a century ago that "the phase of economic development after the emergence of a large class of wage laborers but before the development of social security arrangements is potentially a deeply vulnerable one." In Senegal, this set of class relations, with a large urban class of wage workers with limited

[15] Similarly, 70% of agricultural households in Senegal produce millet. Only one-quarter of these sell millet.
[16] Informally, I was told in the Casamance region of Senegal, famed for its rice production, that households do not sell their rice. Rice is kept for the family. Rice might be gifted, particularly to extended family members in urban areas or to local families who are struggling to make ends meet, but it is generally not sold.
[17] Sen (1982: 6) highlights this distinction in his classic treatment of class and food security.

social protections, does not seem like a phase. It is the new normal, as are the challenges to food security and population health that come along with it. These include world food price volatility but also climate change, which will undermine existing livelihoods across the Sahel (Sissoko et al. 2011). The people of Senegal are responsible for neither yet face the consequences of both.

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Data sources for figures and regressions

Demographic and Health Surveys for Senegal, available from https://www.dhsprogram.com/data/available-datasets.cfm. Used in regressions and figures throughout. See Table 2 for details on samples.

Agence Nationale de la Statistique et de la Démographie (ANSD) and ICF. 2005–2017 Demographic and Health Surveys (various; datasets). Funded by USAID. Rockville, MD: ICF (distributor).

Agence Nationale de la Statistique et de la Démographie (ANSD) and ICF. 2008 and 2020 Malaria Indicator Surveys (various; datasets). Funded by USAID. Rockville, MD: ICF (distributor).

Enquête de suivi de la pauvreté au Sénégal (EPS) 2005 and 2010–2011. Available from Archivage national des données du Sénégal (ANADS), https://anads.ansd.sn/index.php/home. Used for description of the socioeconomic context of Senegal.

FAO FPMA tool: https://fpma.fao.org/giews/fpmat4/#/dashboard/home. Used in Figure 1, Figure 3, Figure A-3, and alternative regression specification (See Table A-1).

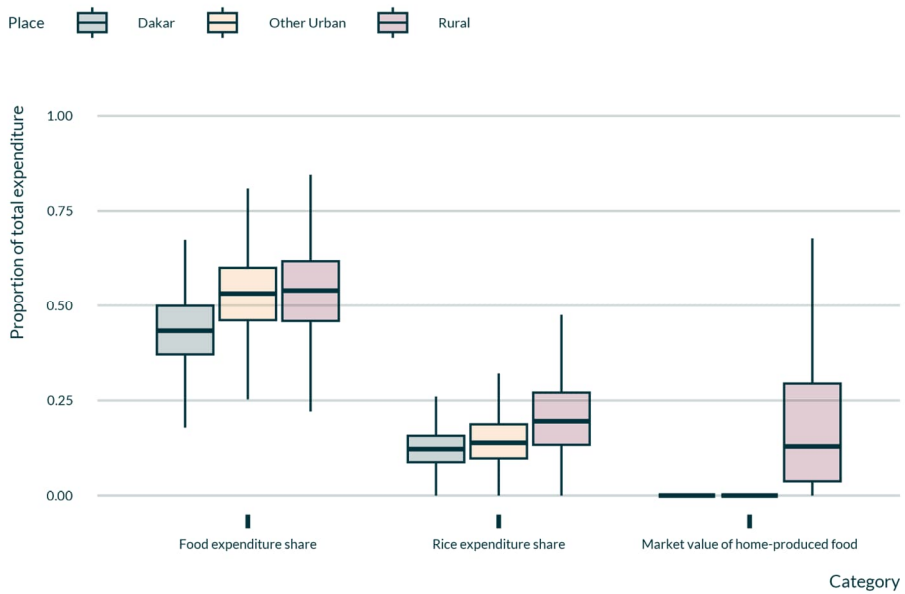
FRED Economic Data, world price of rice (series PRICENPQUSDM), https://fred.stlouisfed.org/series/PRICENPQUSDM, accessed September 19, 2022. Used in Figures 1 and 2.

Appendix

Budget shares

In Figure A-1, I plot household budget data from a 2005 living standards survey conducted by the government of Senegal (EPS 2005). In the first column, I take food expenditure as a share of total expenditure. In the middle, I take the share of rice within food expenditure. Therefore one can calculate the share of rice expenditure in the total budget by multiplying the values of the first and second columns. In the third column, I plot the (estimated) market value of home-produced food relative to total expenditure.

Figure A-1: Distribution of household budget share in Senegal (2005) by residence

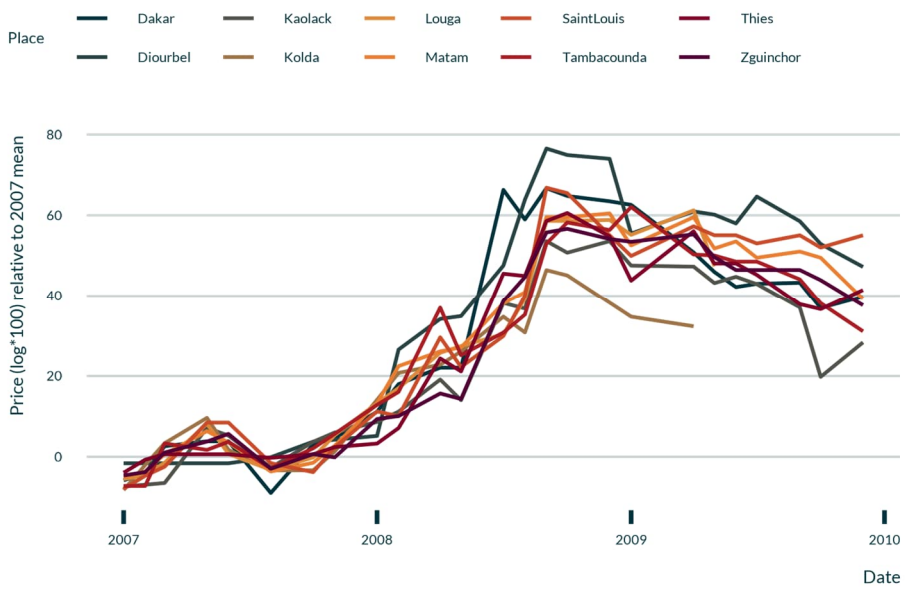


Notes: Budget shares from 2005 household survey data by residence, showing 25th and 75th percentiles, along with the median, and whiskers of 1.5 times the interquartile range. The left panel is the share of food in overall expenses; the middle is the share of rice in food expenses; the right is the value of home-produced food relative to total food expenses. Data from the *Enquête de suivi de la pauvreté* (EPS) 2005.

Local rice prices in Senegal

Here I plot the local price of rice from ten markets in Senegal, corresponding to 10 of the 11 regions of Senegal in 2007. I take 2007 as a reference period and then plot the relative price increase ($ln\left(\frac{p_{it}}{\hat{p}_i}\right)$) in each market. These data come from the FAO FPMA tool. They also underlie Figure 3 and the alternative regression specification below.

Figure A-2: Local rice prices in Senegal, 2007 to 2010

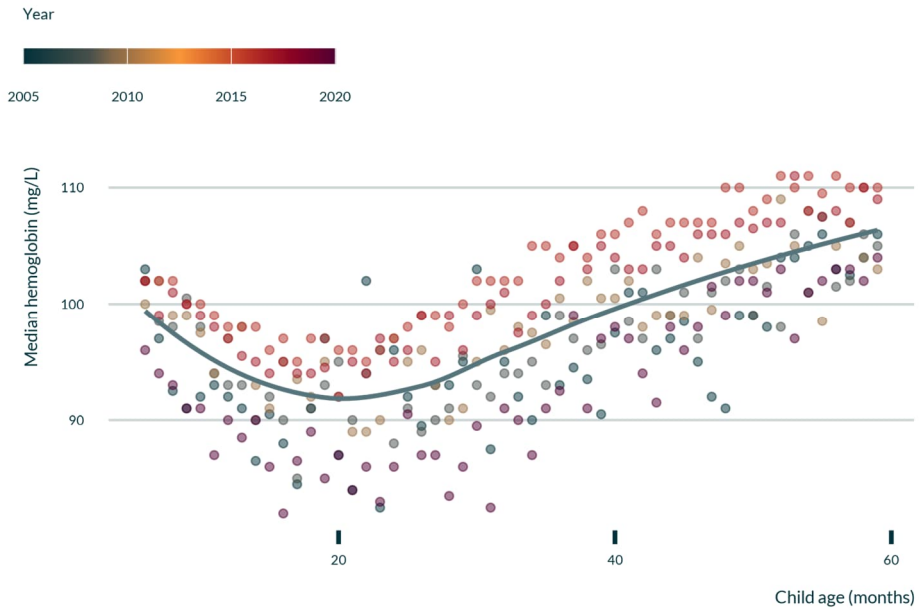


Notes: Retail rice prices in Senegal relative to 2007 average. I take the natural logarithm of the ratio of the monthly price to the 2007 mean for each market, multiplied by 100. Data from the FAO FPMA tool, accessed September 19, 2022.

Hemoglobin by age

Here I plot child hemoglobin by age for all my DHS and MIS data (see Table 2). Note the strong age path of hemoglobin as it falls over the first 18 months of life before slowly increasing thereafter. My empirical strategy depends on correctly adjusting for child age, meaning that properly characterizing the normal age path of hemoglobin is of vital importance.

Figure A-3: Child hemoglobin by age in Senegal



Note: Median hemoglobin by child age and year of survey, Senegal DHS and MIS data 2005, 2008, 2010, 2012–2017, 2020.

Alternative regression specification

I create a data-driven measure of in utero exposure to the price shock. I take retail rice price data from ten regional markets in Senegal (see Figure A-2) for plots of these data and calculate a pre-2008 average ($\acute{p}_i$). I take the rice price relative to the pre-2008 standard: $\Delta p_{t,i} = log\left(\frac{p_{t,i}}{\acute{p}_i}\right)$ for each market i and month t. For each cohort market, in

utero price exposure is the average of this normalized rice price during the nine months prior to birth, denoted by $\Delta_i = mean_t(\Delta p_{t,i})$. I estimate the following equation:

$$hemo = \alpha_1 + \alpha_{2,j}\Delta_i + \alpha_{3,j}Neither + \alpha_{4,j}(\Delta : Neither) + g(age) + X + \epsilon \quad (2)$$

The coefficients of interest are the effects of price exposure on those in either the treated or the control group ($\alpha_{2,j}$), which vary by residence j (Dakar, rest of urban Senegal, rest of rural Senegal).

Next I estimate Equation (2), allowing for an intensive margin of treatment and for the timing of treatment to differ across regions. A 2-σ increase in price exposure in utero results in a 16 mg/L decrease in hemoglobin in Dakar and an 8.5 mg/L decrease in the rest of urban Senegal. The effect in rural Senegal is close to null. My results are thus broadly consistent across the two specifications.

Table A-1: Child hemoglobin regressed on treatment and controls

	Binary treatment	Price exposure
In utero	–10.93 (2.53)	
Reference	–2.3 (1.59)	–2.52 (1.60)
Wealth	2.52 (0.11)	2.54 (0.11)
Primary+	1.48 (0.23)	1.45 (0.23)
Mother's age	1.03 (0.19)	1.01 (0.19)
Other urban: in utero	4.10 (2.60)	
Rural: in utero	4.50 (2.21)	
Rice price (in utero)		–15.71 (3.25)
Other urban: rice price (in utero)		7.24 (5.42)
Rural: rice price (in utero)		16.16 (3.96)
N	43,025	43,025

Notes: Child hemoglobin regressed on treatment and controls. Least-squares regression results for Equation (1) (column 1) and Equation (2) (column 2). All regressions adjusted for mother's age and education, child age, and household wealth. For binary treatment, exposure is defined as discussed in the empirical methods section. For price exposure, exposure is measured by normalized rice prices in the closest regional market during gestation. The coefficient compares children breastfeeding during the crisis to those who were in utero. Coefficient on price exposure allowed to vary by residence, where Dakar is the default group. Data from the DHS; see Table 2. Standard errors are heteroskedasticity robust.

Software used

Analysis done in *R* version 4.4.1 (2024–06–14), with the following packages:

Package	Loaded version	Date	Source
dplyr	1.1.4	2023-11-17	CRAN (R 4.4.0)
forcats	1.0.0	2023-01-29	CRAN (R 4.4.0)
ggplot2	3.5.1	2024-04-23	CRAN (R 4.4.0)
haven	2.5.4	2023-11-30	CRAN (R 4.4.0)
interactions	1.1.5	2021-07-02	CRAN (R 4.4.1)
jtools	2.2.2	2023-07-11	CRAN (R 4.4.0)
lubridate	1.9.3	2023-09-27	CRAN (R 4.4.0)
mediocrethemes	0.1.3	2024-05-08	Github (vincentbagilet)
purrr	1.0.2	2023-08-10	CRAN (R 4.4.0)
quantreg	5.97	2023-08-19	CRAN (R 4.4.0)
readr	2.1.5	2024-01-10	CRAN (R 4.4.0)
SparseM	1.81	2021-02-18	CRAN (R 4.4.0)
stringr	1.5.1	2023-11-14	CRAN (R 4.4.0)
tibble	3.2.1	2023-03-20	CRAN (R 4.4.0)
tidyr	1.3.1	2024-01-24	CRAN (R 4.4.0)
tidytable	0.11.0	2024-02-09	CRAN (R 4.4.0)
tidyverse	2.0.0	2023-02-22	CRAN (R 4.4.0)