Anemia in Senegal: The Effect of the 2008 World Food Price Crisis

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Abstract

I explore the effect of the 2008 world food price crisis on the health of Senegalese children who were *in-utero* at the time. While previous work has looked at the effect of the 2008 crisis on child health, mine is the first to use a fetal origins approach, which allows me to use data collected several years after the crisis. As predicted by the food-policy literature, I find that the 2008 crisis had a negative impact on fetal health in urban areas, but not in rural areas. Observing the same cohort over time, I find that the effect of exposure is apparent in 2010 but not in 2012, suggesting hemoglobin catch-up. Because iron is a critical nutrient in early-life development, this catch-up masks an important negative shock to health.

1 Introduction

In early 2008, world food prices rose sharply and suddenly. The prices of rice and wheat doubled over the course of a few months, while the prices of other staples increased as well. Rising prices diminished the purchasing-power of consumers around the world, resulting in unrest and riots in low-to-medium-income countries. The causes of the price increase are debated, but include American agriculture policy as well as the real-estate market crash(Rosset 2008). Basic necessities became objects of financial speculation, squeezing the budgets of the world's poor.

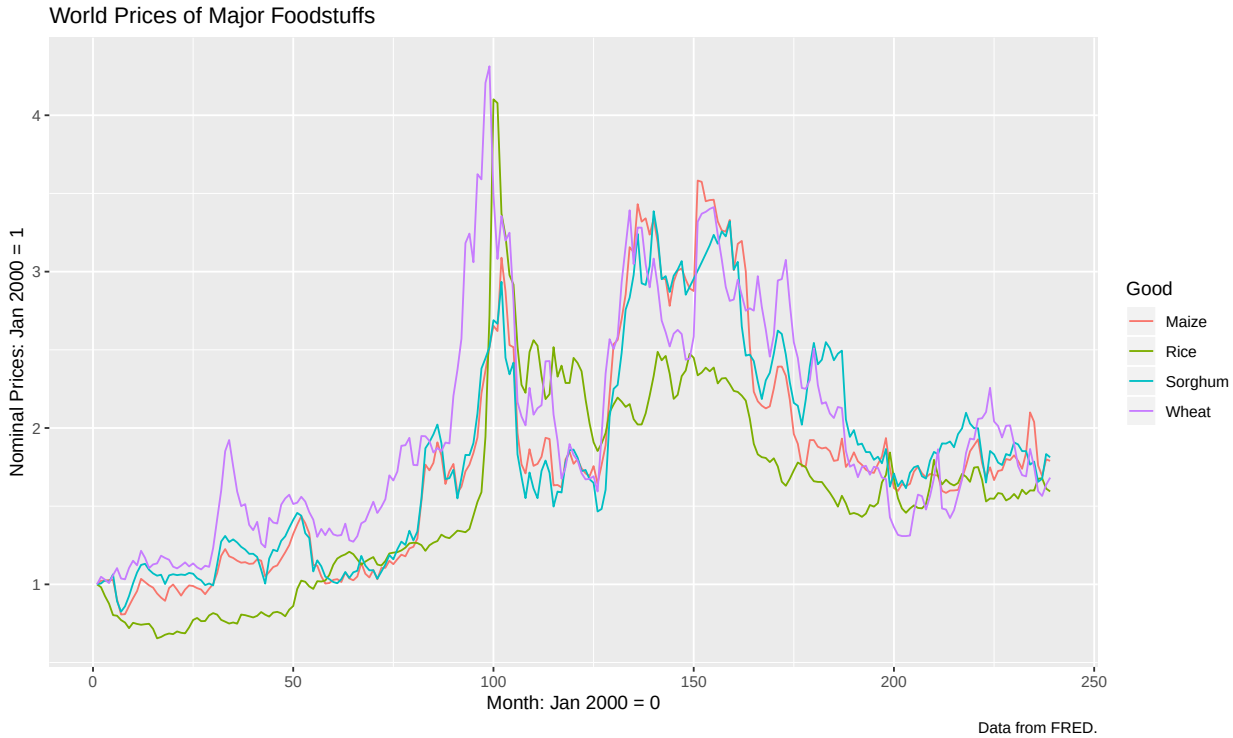


Figure 1: The 2008 Food Price Crisis

The 2008 crisis drew attention from major journals Science and Nature, as well as from the fields of peasant studies, nutrition, and development.[1] The crisis was viewed as evidence of the risk that poor countries take by specializing in cash-crops for export and relying on imported food. Much of the subsequent rhetoric in these fields advocated reduced integration into the world market, and more self-sufficiency in food production (Rosset 2008).

Despite the attention surrounding the crisis, there are few causal evaluations of its impact on nutrition and health. Identifying the effect of the crisis is difficult because it affects everyone in the world at a given time. If we are to use the standard measures of nutrition, like weight-for-height, then the data-collection must have been contemporaneous with the crisis. To observe the effect of the crisis, we must compare the same children before and after the crisis occurs. If we observe these children several years after the crisis, there is no control group or exposed group. Everyone was exposed to the crisis, unless they were born too late, in which case they are not comparable.

Arndt et. al. (2016) run into this problem explore the relationship of food-price inflation and weight-for-height of children in Mozambique. They employ a propensity-score matching approach to compare children observed during the peak of the crisis to those observed before-hand. While a useful paper, the methodology is limited by the dataset, and the lack of a true control group. Dimova et. al. (2013) take a different approach, examining the effect of the crisis on a synthetic measure of welfare. While interesting in its own right, their approach does not address the basic issue: was the 2008 food price crisis bad for nutrition in developing countries? If so, who for. Moreover, many outside of economics would find the result of direct impact on health more convincing than synthetic measure of "welfare" which relies on assumptions that non-economists find less than appealing. We would like to be able to say whether the 2008 was bad for health. If so, who for? This is exactly the question I answer.

To address these limitations, I bring to bear the biological mechanism of iron-transmission and hemoglobin formation in fetuses and infants. For simple, biological reasons I explain below, fetuses are relatively more sensitive than infants to nutritional iron-availability. Thus, we can compare the hemoglobin those who experience the 2008 crisis as infants to those who experienced the crisis *in-utero*, following the now-common fetal-origins literature (Almond and Currie 2011). I explore the effect of the 2008 crisis on maternal-fetal health in Senegal. Senegal imports most of its food supply and has nationally-representative surveys which measure hemoglobin 2 and 4 years after the crisis, making it an excellent candidate for this analysis.

Using a fetal origins approach opens a wider range of evidence, as data from 2-4 years post-crisis can be used. Having a broader sample allows me to test heterogeneity in the effect of the crisis, and to disentangle its effect from seasonal fluctuations, unlike Arndt et. al. (2016). By looking at hemoglobin, I gain improved sensitivity relative to weight-for-height, as hemoglobin is sensitive to both quality and quantity of food. Relative to Dimova et. al., my approach measures the direct effect of the 2008 crisis on maternal and fetal health, rather than a synthetic measure of welfare. My paper most closely resembles that of Block et. al. 2004, although the crisis of interest differs.

Moreover, the increased variation from the fetal origins approach allows me to test for spatial heterogeneity in the effect of the crisis. This will allow me to provide empirical evidence to bear on previous work, which has emphasized a rural-urban divide where the urban poor suffering from increased food prices, and the rural poor are minimally affected (Dimova and Gbakou 2013; Barrett 2010; Ruel et al. 2009).

My work also contributes to the literature on intergenerational transmission of anemia. The economic costs of anemia are well-understood, as are its biological causes and consequences (Zimmermann and Hurrell 2007). While it is known that maternal iron consumption affects fetal hemoglobin, the magnitude of this effect has not been quantified. Moreover, the extent to which any *in-utero* effects *persist* as the infant ages has not yet been addressed. Previous work has focused on small, controlled trials involving anemia and iron supplementation, while I use a large, nationally representative dataset.

I use the rapid, unforeseen increase of world food prices in early 2008 as a natural experiment to analyze the intergenerational effects of maternal nutrition on infant anemia. Using data from Senegal 2-4 years after the

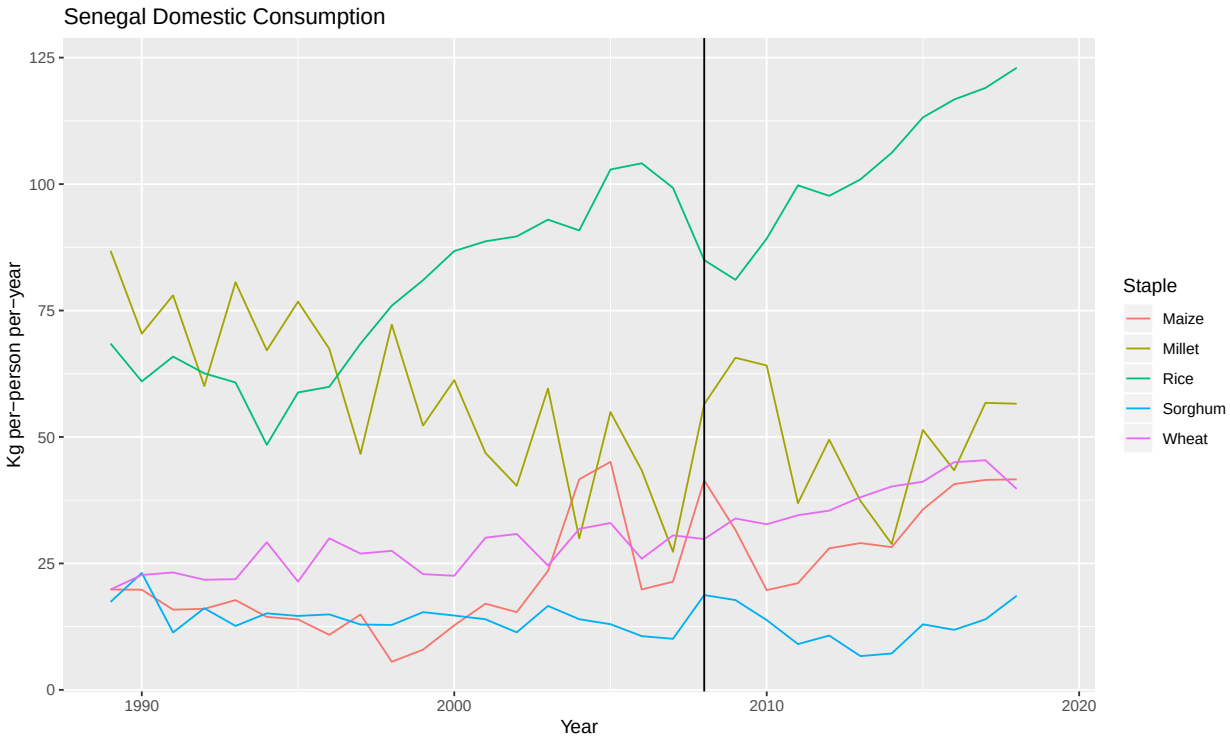
[1] See Cohen and Garrett (2010); Hadley et al. (2011); Martin-Prevel et al. (2012); Minot (2010); Ruel et al. (2009); Wodon and Zaman (2008); Von Braun (2008); Barrett (2010).

crisis, I am able to both quantify the effect of the 2008 crisis in Senegal and examine the effect of *in-utero* shocks on anemia in childhood. The causes of anemia and the consequences of the 2008 crisis have been explored separately by researchers. I am, to my knowledge, the first to connect the two.

1.1 Senegal in the 2008 Crisis

Senegal is a small, poor economy on the west coast of Africa, with a population of 16 million and a GDP per capita of $3,675 (PPP). The climate is Sahelian, with a rainy season starting in May. Average annual rainfall increases on a southward gradient. Dakar is the port and economic hub. The coastal areas practice horticulture and fishing, while the hinterlands close to Dakar engage in commercial groundnut production for export. The river basins in the north and south are farmed for rice, while millet, sorghum and maize are the staples in drier areas. Population is concentrated around Dakar, with smaller concentrations in the river basins in the north and south. See the appendix for more information about the regions of Senegal.

Although agriculture employs three-quarters of the population (CIA Factbook), imports make up a large part of the food supply. The main staples are rice, maize, sorghum, wheat and millet. As seen in the graph below, foods which are mainly imported, like wheat and rice, have increased in importance in Senegal over the past thirty years, while locally produced staples millet and sorghum have declined in per-capita consumption.



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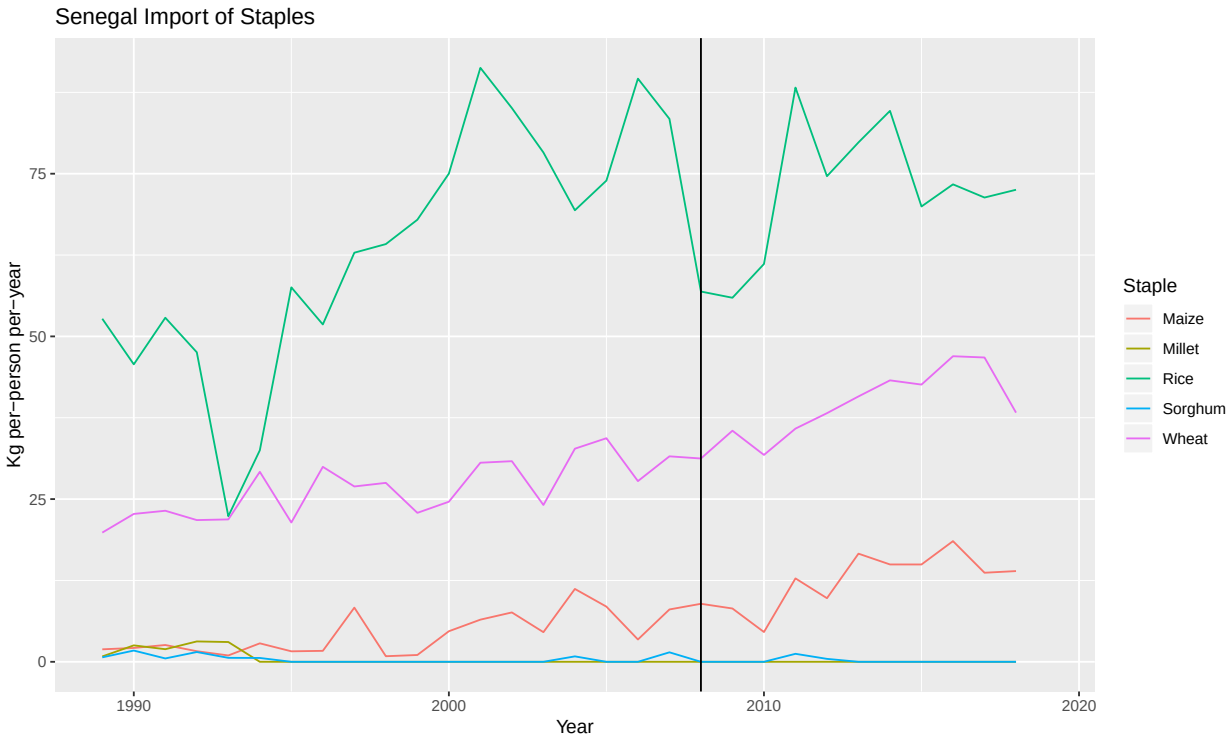

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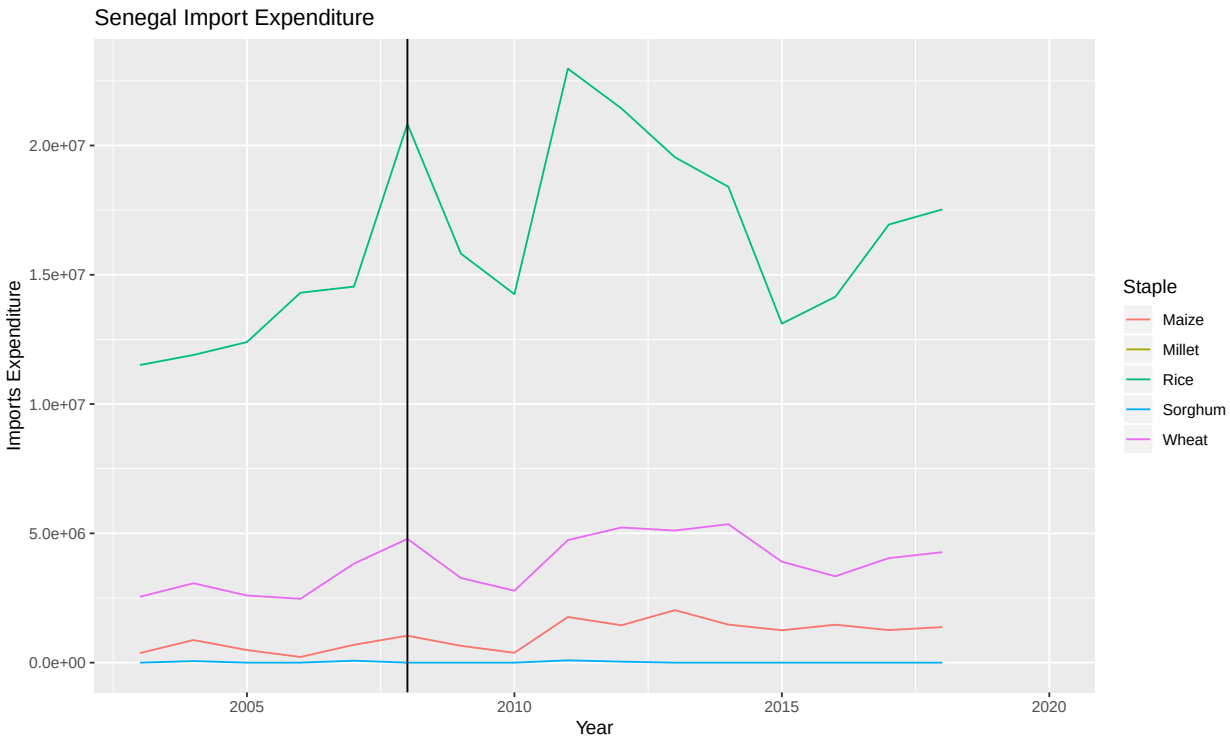


Figure 4:

We see that in 2008, when prices increased dramatically, consumption of millet and sorghum increased while that of rice decreased. This might reflect some substitution away from high-priced, imported goods. It would indicate some reserve capacity for local food production.

The world price spike is apparent in retail prices in Senegal. Here I plot the retail price of rice in Dakar. Rice prices follow a similar pattern to the world price, with a delay, shifted upwards by transportation costs and retail markup.

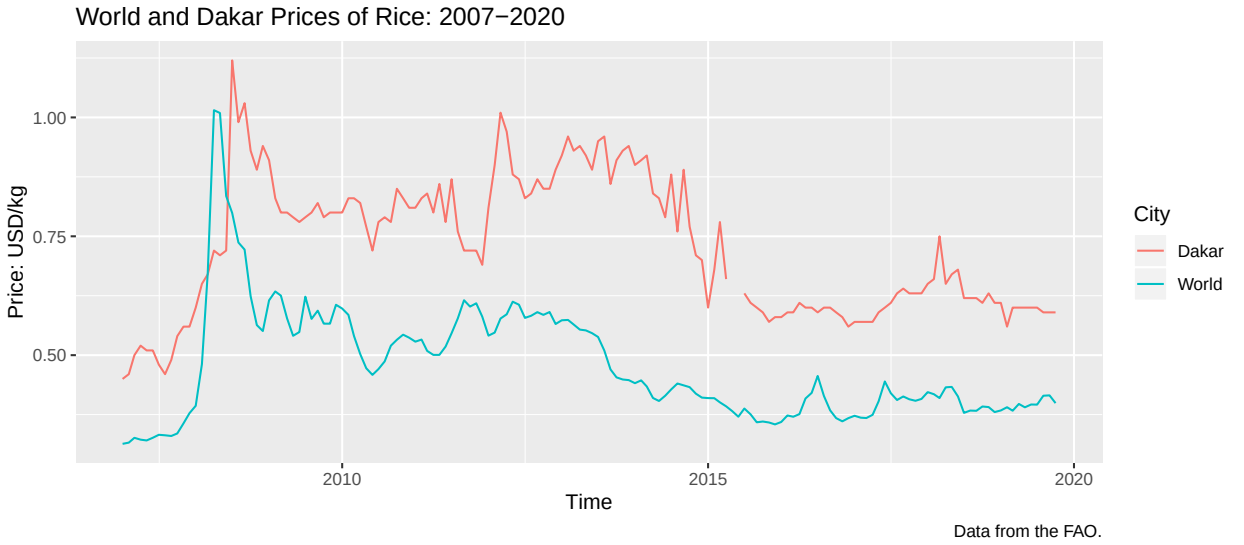


Figure 5: World Rice Price and Retail Price in Senegal

To construct a world price index that is relevant for anemia in Senegal, I use two weights. One, the amount of consumption of a particular staple in Senegal in 2008, and two, the iron per 100 grams of that staple.[2] I took the weighted average of the commodity prices in real USD, and multiplied by the CFA-USD exchange rate in order to get prices in terms of local currency. I plot the index in log-terms below, so that proportional changes can be easily read.

[2]The final weights were 0.39 0.13 0.18 0.30 for rice, maize, sorghum and wheat respectively. I exclude millet from the study because it has not been imported or exported since the early 1990's.

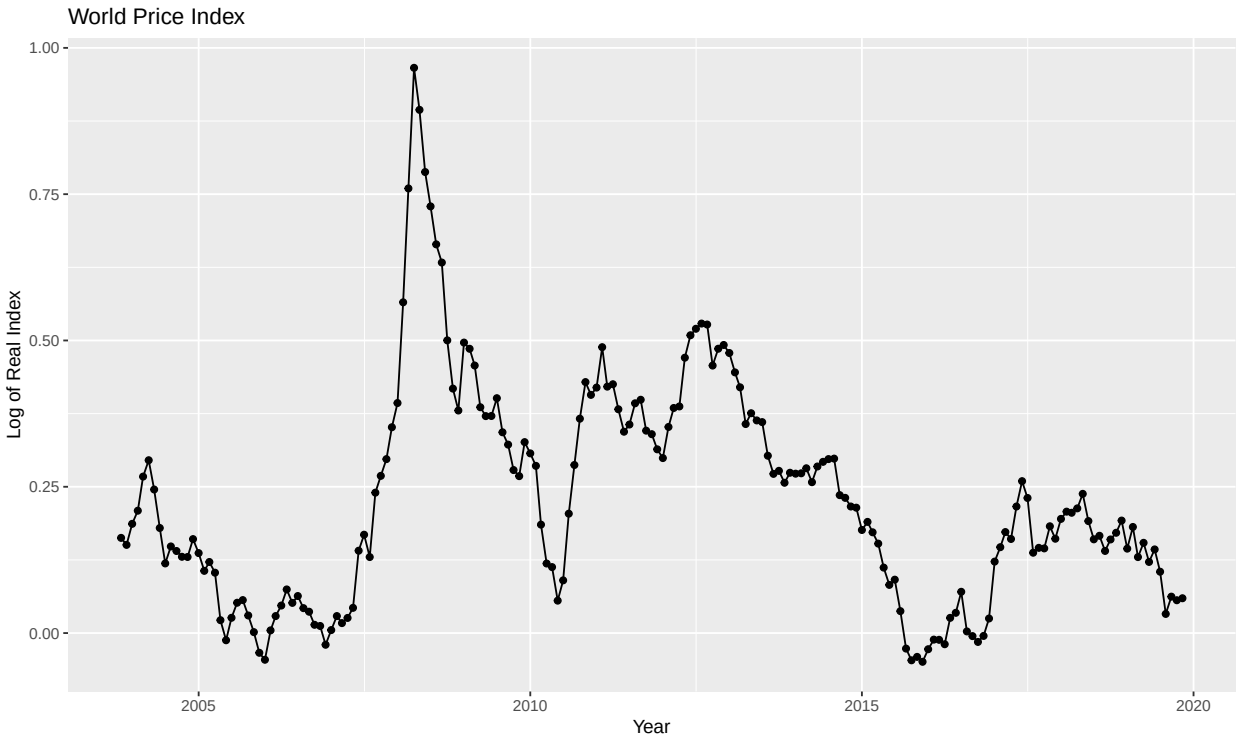


Figure 6: World Price Index, weighted by Senegal iron-consumption

2 Mechanisms

Given an increase in food prices *in-utero*, I expect a decrease in infant hemoglobin through the following chain of reasoning:

1. An increase in the world price of food leads to an increase in local prices in Senegal;
2. which causes food consumption to decrease in quality and/or quantity;
3. which causes iron intake to fall for pregnant women;
4. which reduces fetal iron stores;
5. leading to reduced hemoglobin as an infant and child.

Each of these mechanisms has specific caveats and mediators which will be considered in order to develop appropriate empirical analysis.

2.1 World Price, Local Price and Transportation Costs

The effect of world price on local price will depend on transportation costs and local supply (Minot 2010). Consider a two good model, with P as the price of food, and the non-food good set as the numeraire. Let P_a be the hypothetical price of food under autarky. Because Senegal is an small-open-economy (SOE), trade provides an infinitely elastic supply curve at price $P_w + t$, the world price plus transportation costs. Given an increase in the world price of food, there are three cases for how the local price will respond:

1. Food was not imported before the price increase, and it is not be imported after the increase. Price will be determined by local supply and demand, $\frac{\Delta P_l}{\Delta P_w} = 0$.

2. Food was imported before the price increase, but is not imported afterwards. In this case, the local supply and demand curves meet at a lower price than P_w. The economy moves up the local supply curve and $0 < \frac{\Delta P_l}{\Delta P_w} < 1$.
3. Food was imported before the price increase, and continues to be imported afterwards. In this case, the new price is still $P_w + t$ and $\frac{\Delta P_l}{\Delta P_w} = 1$.

Transportation costs also change the relative price of commodities faced by producers, affecting investment. Transportation costs make commercial, export-oriented production less profitable relative to local, subsistence production. Thus, we should also expect food-producing capacity to increase with transportation costs. Greater food-producing capacity would shift the local supply curve outward, making the world price less likely to bind.

2.2 Price and Consumption

As price increases there are substitution and income effects which lead to lower consumption. Given that the 2008 price increase affected all of the major, imported staples, I would expect the substitution effect to be relatively small.[3] As poor families spend a greater share of income on food than rich families, the income effect, and therefore total impact of the spike, should decrease with household wealth.

Iron, unlike calories, is a function of both food quality and quantity. As prices increase, the household must either reduce quantity or change the type of food they are consuming. In Burkina Faso, which neighbors Senegal, the 2008 price shock lead to decreased amounts and diversity of food (Martin-Prevel et al. 2012). As I am particularly interested in female iron consumption, gender inequality looms large. While there are a host of issues surrounding intra-household allocation and gender, I will not treat those in this paper. Instead, I will assume that female iron consumption decreases with prices in a similar way as household iron consumption.

Given a price shock, households try to smooth consumption across time, attenuating the effect of price on consumption. However, most households, especially the poor, are credit constrained. Thus, we would expect a nonlinear effect of price on consumption, with large increases in price having disproportionately large effect on consumption. As savings, and therefore ability to smooth in a credit-constrained world, are a stock, prices in previous months will also affect the ability to smooth consumption. If prices have been high for several months, then a household is less likely to be able to smooth consumption given another month of high prices.

All of this analysis assumes that the household is a net-buyer of food. If the household is a net-seller of food, then a price increase will be a windfall (Ivanic and Martin 2008). More generally, greater capacity to produce food will allow a household to mitigate the negative effects of an increase in food prices. The urban population of Senegal typically does not own their own land, and must purchase food. Therefore, we would expect the negative effects of a price increase to be greater in the cities than in the countryside.

2.3 Iron and Anemia in Mother and Child

Anemia is characterized by low levels of hemoglobin in the blood and is most often caused by iron-deficiency due to malnutrition (Camaschella 2015). By World Health Organization (WHO) estimates, 1.62 billion people around the world suffer from anemia, most of whom are poor women. Anemia causes fatigue and blindness in adults and the direct effect of anemia on productivity accounts for 4% of world GDP per year (World Bank, 2004).

For pregnant women, anemia increases the probability of death during childbirth, and maternal anemia has a direct effect on fetal and infant anemia (M. M. Rahman et al. 2016). In children, iron is a critical nutrient, and anemia may impair cognitive and motor development. An anemic infant is then more likely to develop into an anemic adult. Thus, an anemic mother begets anemic infant who becomes an anemic woman

[3] One possibility is that the increase in the price of imported goods lead to increased consumption of local crops, like millet. Millet is relatively high in iron compared to rice, so this would attenuate any results I find.

who begets an anemic child. This inter-generational aspect of anemia is well appreciated by the WHO and is recognized as an important issue at the nexus of public health, nutrition and female empowerment (Camaschella 2015).

Hemoglobin is limited by iron stores in the body, which depend on disease environment and the quality and quantity of food intake. A healthy human has iron stores roughly equal to the iron in use in their blood. There is a small, constant loss of iron through the small intestines. Because hemoglobin is determined by a stock, whereas iron intake is a flow, past iron consumption will affect current iron-stores and hemoglobin levels (Camaschella 2015).

Iron is passed from the mother to fetus, facilitating the development of fetal hemoglobin, which differs from adult hemoglobin. Over the first four months of life post-birth, the infant breaks down the fetal hemoglobin and synthesizes adult hemoglobin. Little iron can be transmitted through breast-milk, so the fetus must develop sufficient stores of iron in order to transition to adult hemoglobin as they grow and their blood volume increases. The additional iron required for pregnancy is around 1,000 mg– nearly half of the total iron stores in a healthy, non-pregnant woman, making iron a scarce nutrient for most pregnant women (Lactation 1990).

Infants, on the other hand, need relatively little iron, given that they develop sufficient stores *in-utero*. Breast-milk contains very little iron, around 0.1 mg per gram (USDA). As illustration, consider that a typical infant would consume less than 700 grams of breast milk a day in the first six months (Neville et al. 1988), meaning a total iron intake of around 54 mg over the first six months. This is trivial compared to the 1,000 mg needed during pregnancy. Moreover, as long as the woman is breastfeeding, the absence of menstruation will reduce her iron loss rate. Thus, during the first six months of life hemoglobin in infants should be relatively insensitive to maternal iron consumption and general iron-availability in the surrounding nutritional environment.[4] Here I list the CDC's recommended daily intake of iron for mother and infant across different periods, which illustrates this point.

Period	Maternal Iron Intake	Period	Infant Iron Intake
Pregnancy	27 mg		
Lactation	9 mg	0-6 mo	.27 mg
Menstruation	18 mg	7-12 mo	11 mg
		Childhood	7 mg

We see that iron availability is critical during the fetal period, but not during the breastfeeding period. This discontinuity in the response of infant hemoglobin to nutritional environment (very sensitive up until birth, and then insensitive) offers a clear identification strategy. Given a specific nutritional event, we can compare those who had already been born to those still *in-utero*.

3 Data

The DHS measures the hemoglobin of infants and children age 6 months to 5 years. There are two DHS surveys of Senegal which include children *in-utero* during the 2008 crisis: 2010 and 2012. The DHS data includes location and households characteristics. The variables which I will use for this study are hemoglobin, child month of birth, household wealth, whether a household lives in an urban *commune* and driving-distance to Dakar.

[4]Note that this all relies on women breastfeeding their children. In Senegal, breastfeeding is overwhelmingly common during the first six months, with very little cessation before the child reaches a year old (Mané et al. 2006).

3.1 Hemoglobin

At birth, a healthy infant will have a large amount of fetal hemoglobin which is then broken down and replaced by adult hemoglobin. In general, hemoglobin levels decrease after birth and then rebound after the first year of life. We see such a pattern in the DHS data, which I plot below. The yellow window indicates the 2008 crisis. The curves are the conditional mean of hemoglobin over month of birth, calculated via local linear regression. The grey banner is the 95% confidence interval of the mean. The highlighted region are those children exposed to the 2008 crisis *in-utero*. Note that I observe this cohort twice: first when they are 2 years old, and again when they are 4.

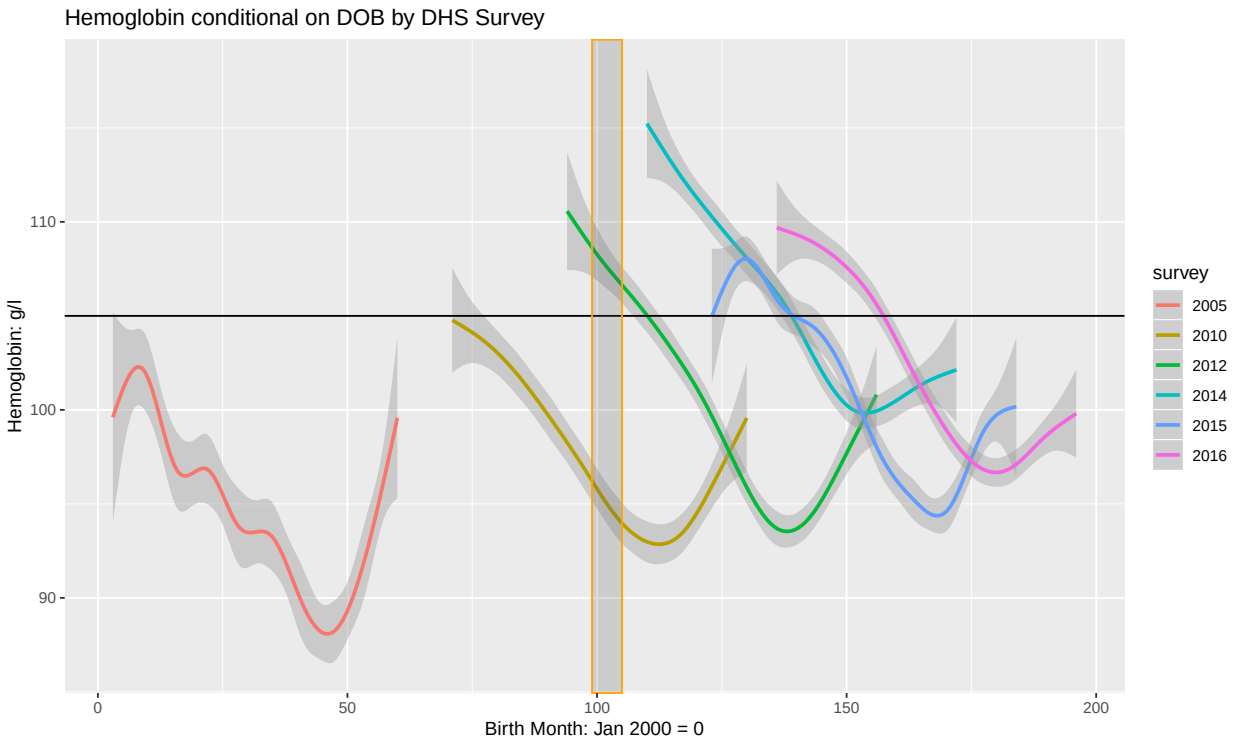


Figure 7: Senegal DHS Hemoglobin data, smoothed over time

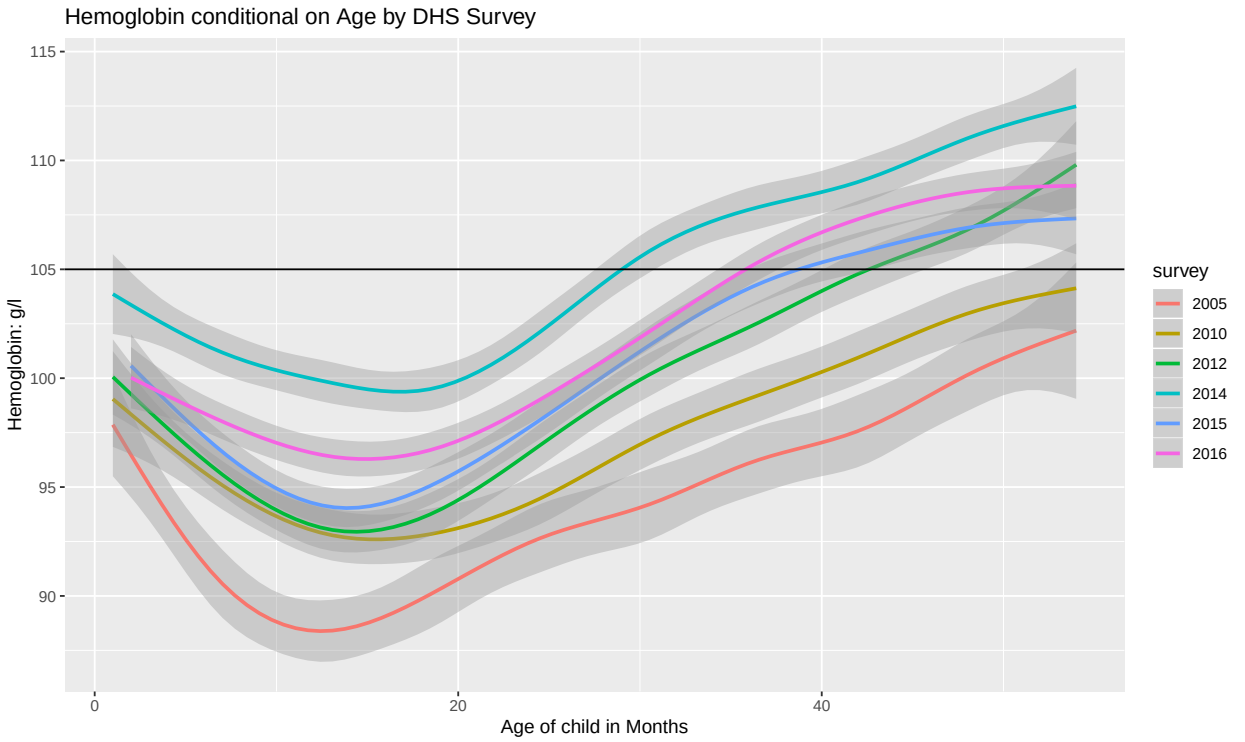


Figure 8: Senegal DHS Hemoglobin data, smoothed over time

Anemia is relatively rare in children, compared to in infants or adults. The cutoff for anemia in infants and children, 105 grams per liter of blood is shown as a black, horizontal line. Note that these curves are not a trajectory; rather, they are a single cross-section of children of multiple ages. Within an age group, hemoglobin follows a uni-modal distribution.

In order to account for age, I will use a z-score approach analogous to how child height and weight is treated by the DHS. We are interested in the 2008 price shock. The only surveys which provide information on the shock are 2010 and 2012, so I use the other 5 surveys (2005, 2014-17) to make z-scores for the surveys of interest. Taking the mean and standard deviation by age, I then normalize hemoglobin by age.

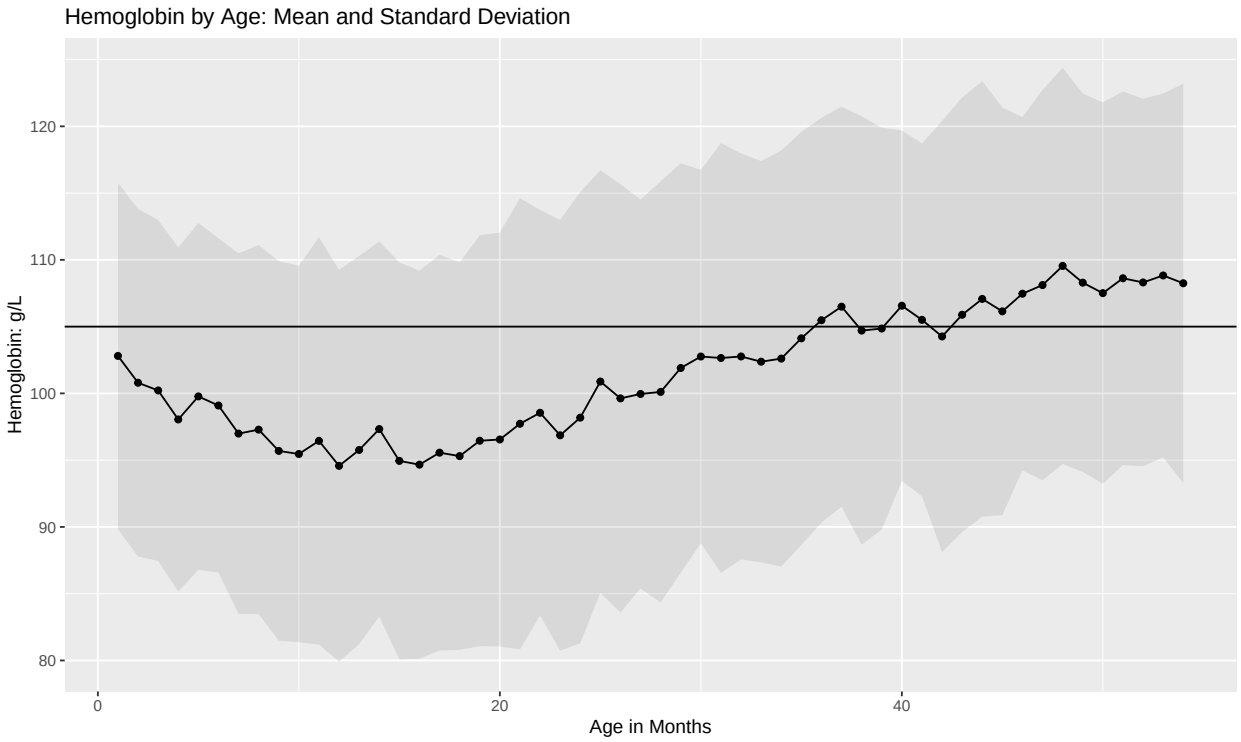


Figure 9: Hemoglobin Z-Score Transformation

The black line is the mean hemoglobin by age. We see that the mean hemoglobin during early-childhood falls below the anemia threshold during early childhood, suggesting that maternal iron-intake is generally insufficient for the fetus to develop the iron-stores it requires. The standard deviation of hemoglobin by age is 14-15 g/L.

3.2 Other Variables of Interest

Below I list some descriptive statistics of other variables of interest. Driving distance to Dakar is in terms of hundreds of kilometers, and land refers to the proportion of households who own their own land. Wealth and distance are reported as means.

Urban	Survey	Wealth	Distance	Land	N
Rural	2010	-0.0656	3.14	82%	2252
Rural	2012	-0.0680	3.01	84%	3492
Urban	2010	0.0928	2.25	24%	974
Urban	2012	0.0658	2.59	29%	1573

The DHS does not record income. It measures wealth as a principle-components analysis of observed household possessions. This index is then centered around zero for each survey. Thus it not a measure of absolute wealth, but a proxy for socio-economic status.

Each DHS observation is tied to a "cluster"– generally a village or neighborhood. These cluster are geo-located with displacement, creating neutral measurement error. I use the coordinates of each cluster to measure the driving-distance to Dakar through the OpenStreetMaps API. The DHS also records whether a household is urban or rural. This urban-rural distinction is inherited directly from the census demarcation

of the country being studied. In Senegal, urban is defined on the level of the *commune* (roughly translated as township). Below I plot the distribution of DHS clusters across Senegal, colored by whether they are urban or rural.

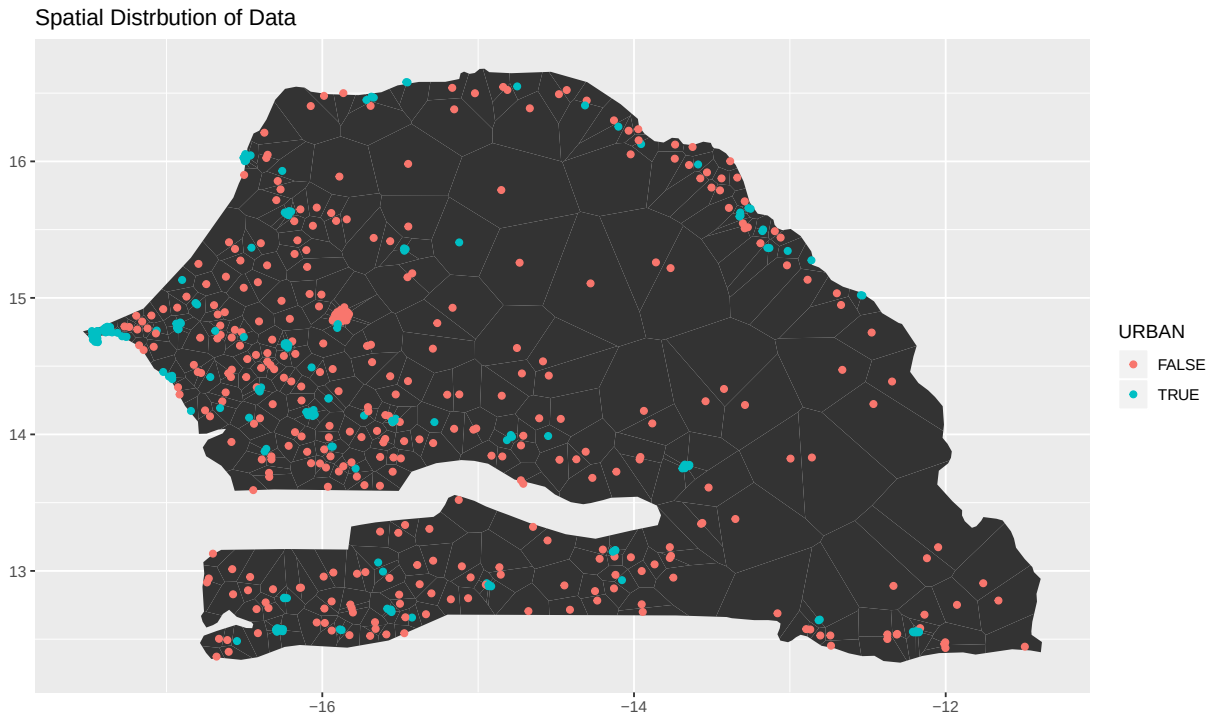


Figure 10: Distribution of DHS Clusters

We will see below that the variable of interest, "exposure", is generally balanced across observables. However, I will also be interacting exposure with other variables. These are not balanced with respect to each other. Urban areas are wealthier than rural areas, and urban dwellers are more likely to live near Dakar. For both the rural and urban populations, wealth is negatively correlated with distance to Dakar.

4 Methods

The 2008 crisis forms a contiguous 8-month period in which the price index is greater than at any other point in the 20 year span. I take all those *in-utero* during this period, February to September 2008, to be exposed. The degree of exposure is the proportion of months of gestation which fall during the crisis period. For anyone born before March 2008, or conceived after September 2008, exposure is zero. For someone born in November 2008, exposure is $\frac{8}{9}$, as they were *in-utero* for the entirety of the 8-month crisis period.

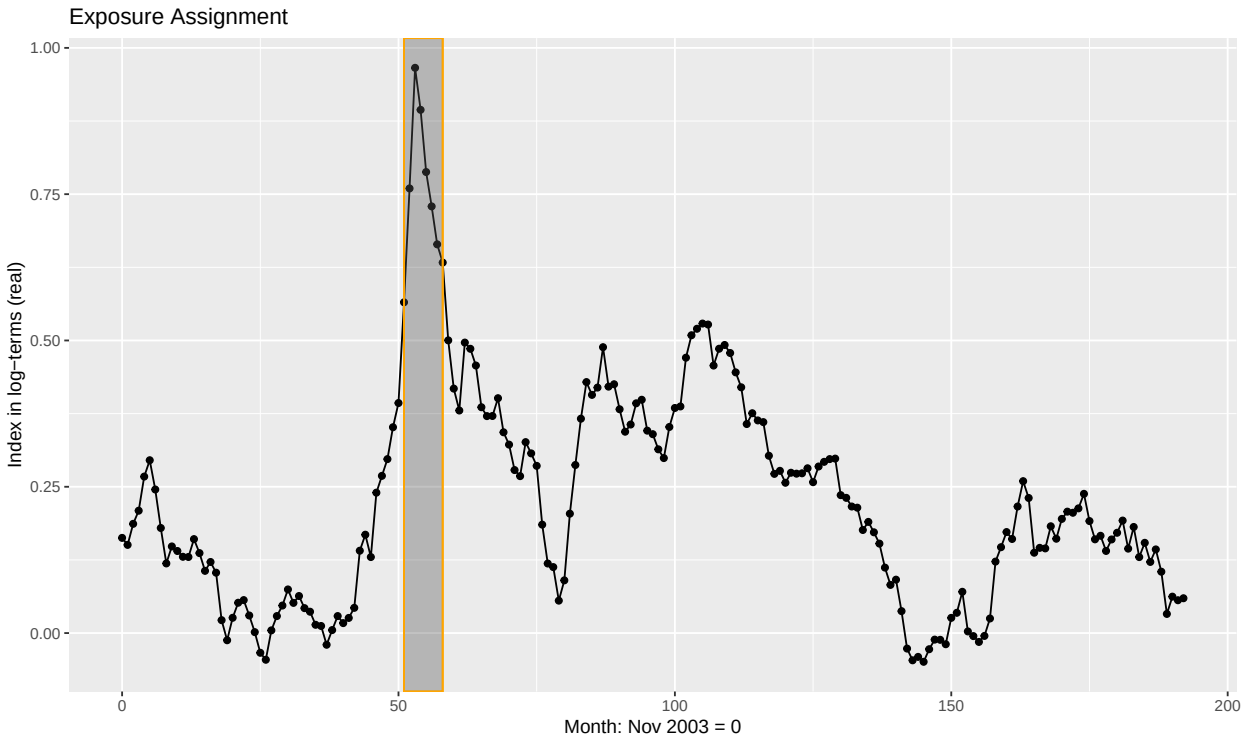


Figure 11: Definition of Exposure to the 2008 Crisis

My methodology follows that of Almond (2006) as we both analyze a cohort-level time shock. The effect should be visible in the raw data, and then this visual test is formalized with a regression characterizing deviation from a quadratic time trend. I then include additional interaction terms because I am interested in heterogeneous effects. I run these regressions separately for the 2010 and 2012 DHS surveys. These surveys identify the same cohorts, but at different points in the cohort life-cycle.

4.1 Identification

The 2008 crisis was caused by developments in large, rich economies and can be taken as external to Senegal. If the 2008 crisis indeed forms a "natural experiment," then it ought to be unrelated to any covariates of interest, just like the treatment in a controlled experiment: i.e. $Exp_i \perp X_i$ where X_i is a vector of covariates. Below we see that exposure is balanced across observables, and we fail to reject the hypothesis of $Exp_i \perp X_i$.

	Exposure	Exposure	Exposure	Exposure
Distance to Dakar	0.0000			
	(0.0000)			
Wealth		−0.0001		
		(0.0003)		
Urban			0.0023	
			(0.0064)	
Land				0.0086
				(0.0063)
R^2	0.0002	0.0000	0.0000	0.0002
Adj. R^2	0.0000	-0.0001	-0.0001	0.0001
Num. obs.	8291	8291	8291	8291
RMSE	0.2698	0.2698	0.2698	0.2698

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 3: Balance Test

The fetal origins strategy, which relies on a biological sensitivity to iron intake determined by whether an individual was *in-utero*, provides a strong argument that any effect I find reflects a change maternal health conditions. I would not, however, claim that my analysis identifies the effect of an increase in world prices. Rather, I am identifying the effect of being *in-utero* during a specific 8-month window, which included a price increase.

Although there is no reverse causation from developments in Senegal to world prices, my results are identified purely on the basis of time. Therefore, temporal coincidence could be responsible for my results. Although the price shock is *external* to Senegal, it is not necessarily *exogenous*, taking the distinction from Heckman (2000). Identification relies upon an understanding of developments within Senegal, and the argument that there was nothing exceptional during the period, save for the effects of the 2008 price crisis. For example, a drought in 2007-2008 might have caused reduced food-availability during the period. I have found no evidence of developments from *within* Senegal which would coincide with the 2008 price spike.

4.2 Specifications

As results, I present two kinds of evidence:

1. The deviation from a quadratic time trend[5] of the exposed cohorts, estimated separately by survey; and,
2. The median hemoglobin-for-age by birth-quarter.

I use median-regression, rather than least-squares, as it is less sensitive to heterogeneous response across the hemoglobin distribution.[6] I run the following regressions separately by survey and urban.

$$H_i = \alpha_0 + \beta Exp_i + \alpha_1 MOB_i + \alpha_2 MOB_i^2$$

Where H_i is hemoglobin-for-age, Exp_i is the number of months *in-utero* during the crisis, $Dist_i$ is the driving time to Dakar for the cluster of individual i.

I then test for heterogeneity, by allowing the effect of exposure to depend on wealth and distance-to-Dakar:

[5]The results are stable with cubic and quartic time trends as well.

[6]We would expect little to no response of hemoglobin to external shocks at the high and low ends of the distribution. At the high end, those in good health are presumably insulated from price shocks by SES. At the low end, a certain level of hemoglobin is required by the body, and it cannot diminish beyond some point without causing death.

$$H_i = \alpha_0 + \beta Exp_i + \delta Exp_i * Dist_i + \alpha_1 MOB_i + \alpha_2 MOB_i^2 + \rho Exp_i * Wealth_i + \alpha_3 Dist_i + \alpha_4 Wealth_i$$

My hypotheses are that $\delta > 0$ and $\rho > 0$. In the first regression, I expect that $\beta < 0$, because distance-to-Dakar must be non-negative. However, in the second specification, The sign of β will depend on the underlying distributions of wealth, which can be negative.[7]

5 Results

Turning to regression, we see a small, insignificant effects of *in-utero* exposure to the 2008 crisis on hemoglobin.

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.0191	−0.3333***	0.2995	0.0664
	(0.0708)	(0.0502)	(0.2144)	(0.2152)
Exposure	−0.0506	0.0342	−0.2669	−0.1306
	(0.2474)	(0.1652)	(0.4005)	(0.4163)
Num. obs.	696	1612	424	888
Percentile	0.5000	0.5000	0.5000	0.5000

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 4: Parsimonious Regression Results

Following the results of the above regression, I plot the median hemoglobin by birth quarter, split into rural and urban groups and only including observations from the 2010 survey.

[7] I do not include land-ownership in any of my analysis because it is so highly correlated with being from a rural *commune* with wealth. Once accounting for the urban-rural split, land-ownership mostly captures the wealth of a household. In future research, with a larger, more geographically diverse data-set, I would like to include land-ownership.

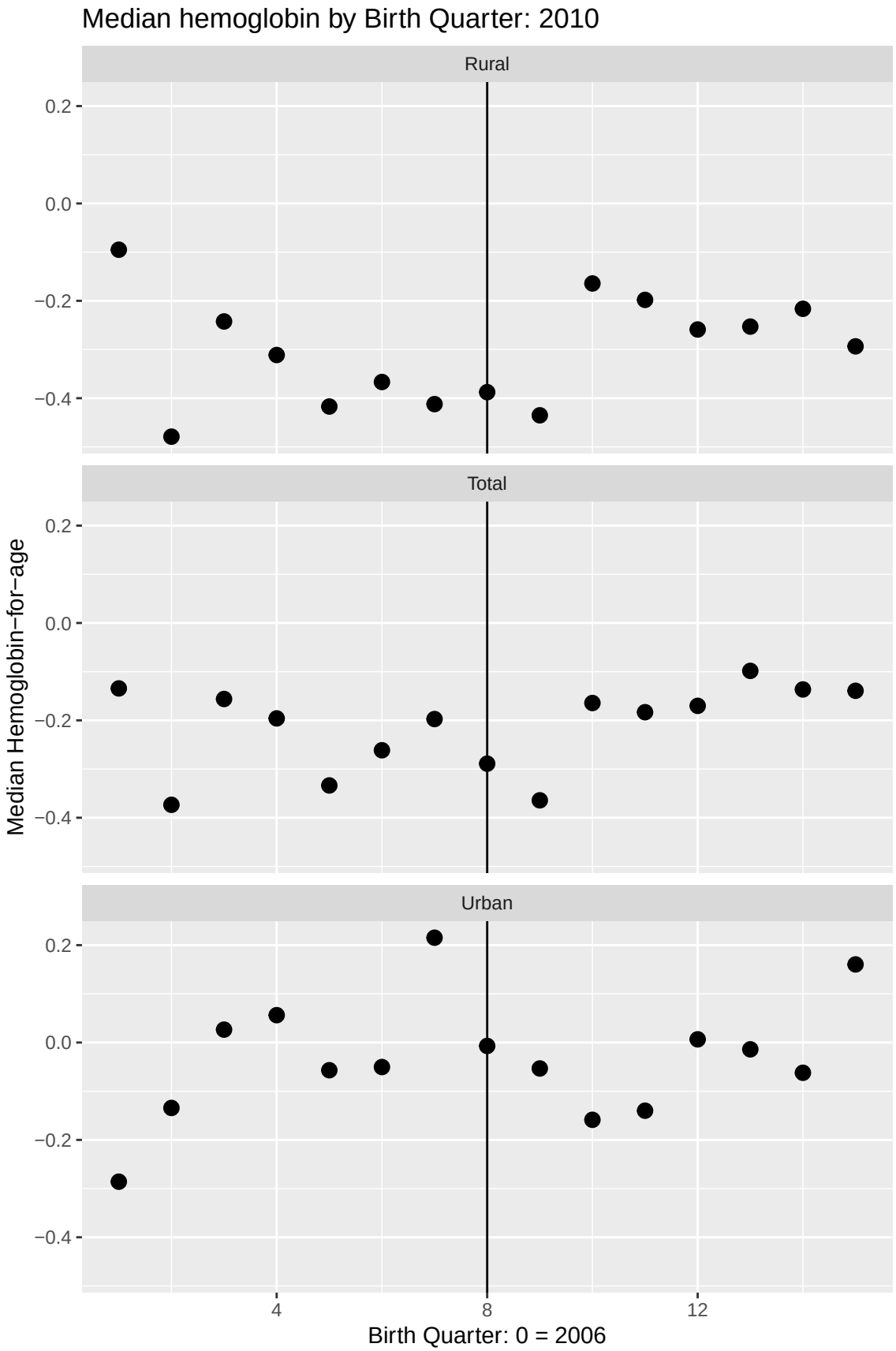


Figure 12: Hemoglobin-for-age by Birth Quarter

We would expect, however that within urban-dwellers the poor to be more affected by the crisis, and those who live closes to Dakar. Thus, I allow the effect of exposure to depend on these two variables. I find support for both of the predicted hypotheses. Wealth and distance-to-Dakar both attenuate the effect of exposure.

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.0317	−0.3515***	0.0106	−0.3154
	(0.1534)	(0.0910)	(0.3391)	(0.2676)
Exposure	−1.2562**	0.0700	−0.3105	0.8541*
	(0.4751)	(0.2265)	(0.6136)	(0.4667)
Distance to Dakar	−0.0192	0.0289	0.0707	0.0944*
	(0.0346)	(0.0246)	(0.0657)	(0.0499)
Wealth	1.4858	2.2794***	0.5930	1.4001
	(0.9519)	(0.4903)	(1.8959)	(1.0096)
Distance to Dakar * Exposure	0.2133*	0.0023	−0.0020	−0.1905**
	(0.0843)	(0.0540)	(0.1130)	(0.0798)
Wealth * Exposure	3.5569	0.2027	1.9335	2.1867
	(2.4572)	(1.5698)	(2.9552)	(1.8642)
Num. obs.	696	1612	424	888
Percentile	0.5000	0.5000	0.5000	0.5000

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 5: Regression Results by Survey and Urban-Rural

Here we see that exposure is negatively related to hemoglobin for urban-dwellers. The effect of exposure is greater for the poor across all specifications. For the rural population, the effect of the crisis on 2010 hemoglobin is noisy but slightly positive. In 2012, the results are attenuated, and the urban-rural difference has become almost undetectable. The attenuation is expected because hemoglobin reflects iron stocks. The stocks will be affected by various other shocks over time, masking the signal from the 2008 shock.

With many interaction terms, the magnitude of effects depends on the underlying distribution of the covariates. The estimated coefficient on exposure above is for someone with $wealth = 0$ and living in Dakar. As Dakar is the wealthiest part of the country, only a very small fraction of the sample will be expected to have such an effect. I calculate the predicted effect of the 2008 crisis given the coefficients estimated above multiplied by each individual's values of wealth and distance-to-Dakar. As we move to the right along the x-axis, the individuals are wealthier and live further from Dakar.

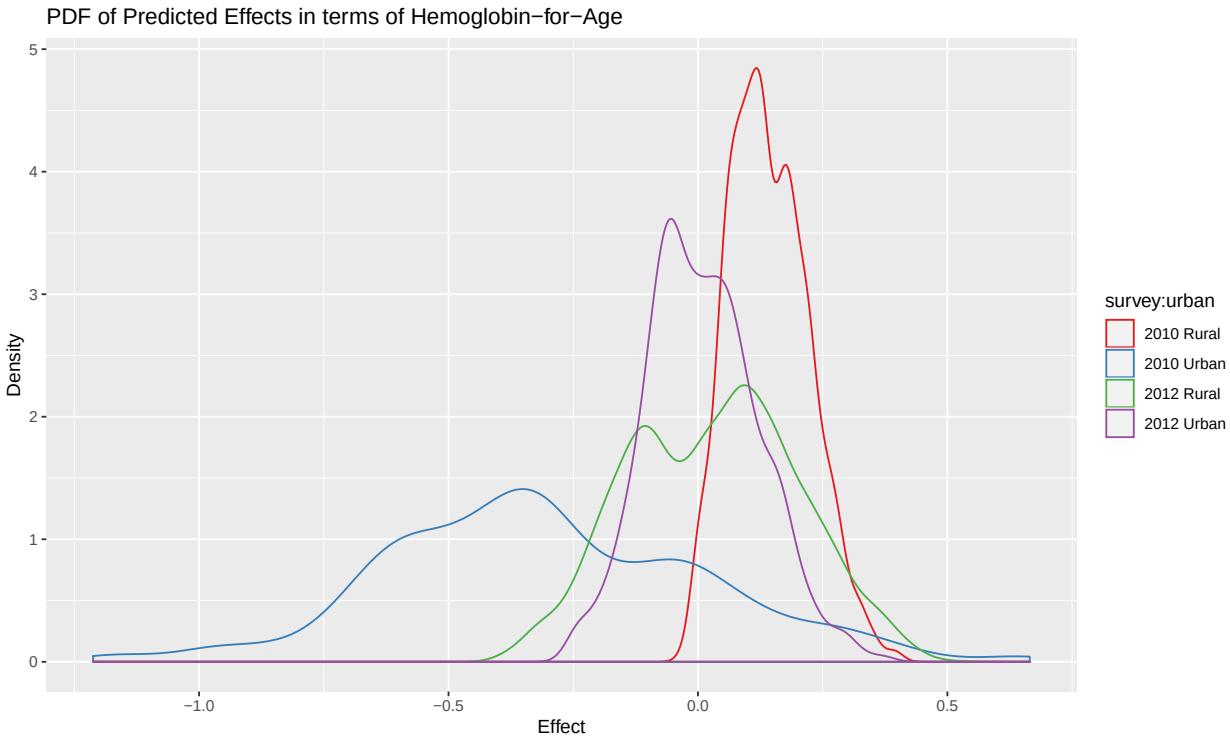


Figure 13:

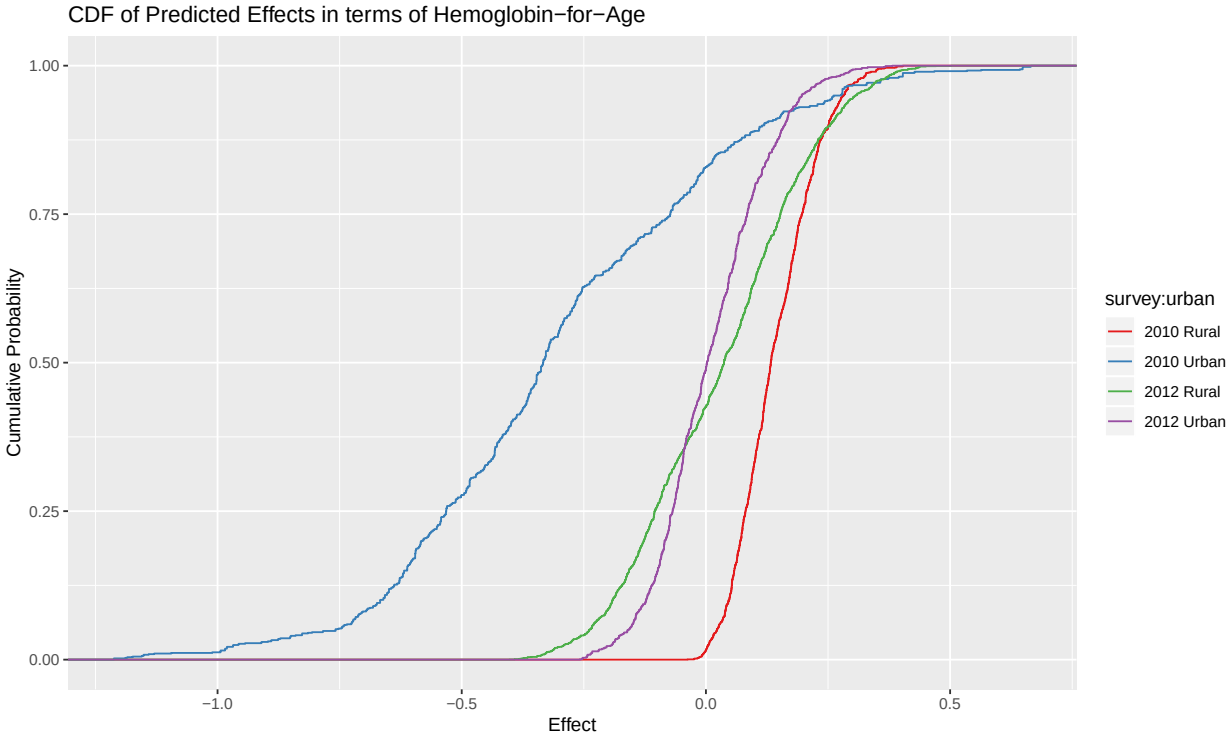


Figure 14:

The implied effects for the urban 2010 population are large. For the urban-poor, the magnitude ranges from 0.5 to greater than 1 standard deviations in 2010. The standard deviation of hemoglobin by age in Senegal is 14-15 g/L. To understand the magnitude of this shocks, consider a 10 g/L shock to hemoglobin. If every child in Senegal was exposed to such a shock, rates of anemia would be expected to increase from ~60% to ~85%. As a further illustration, consider a poor household in Dakar (10th percentile of the wealth distribution within Dakar). The expected effect of being *in-utero* during th 2008 crisis, would be negative 14 g/L of hemoglobin. For a child not to be driven into anemia after such a shock, they would have to have initial hemoglobin of 120 g/L, the 90th percentile of the overall distribution of hemoglobin in Senegal.

6 Discussion

6.1 The Effect of Exposure

The results above provide support for the following hypotheses:

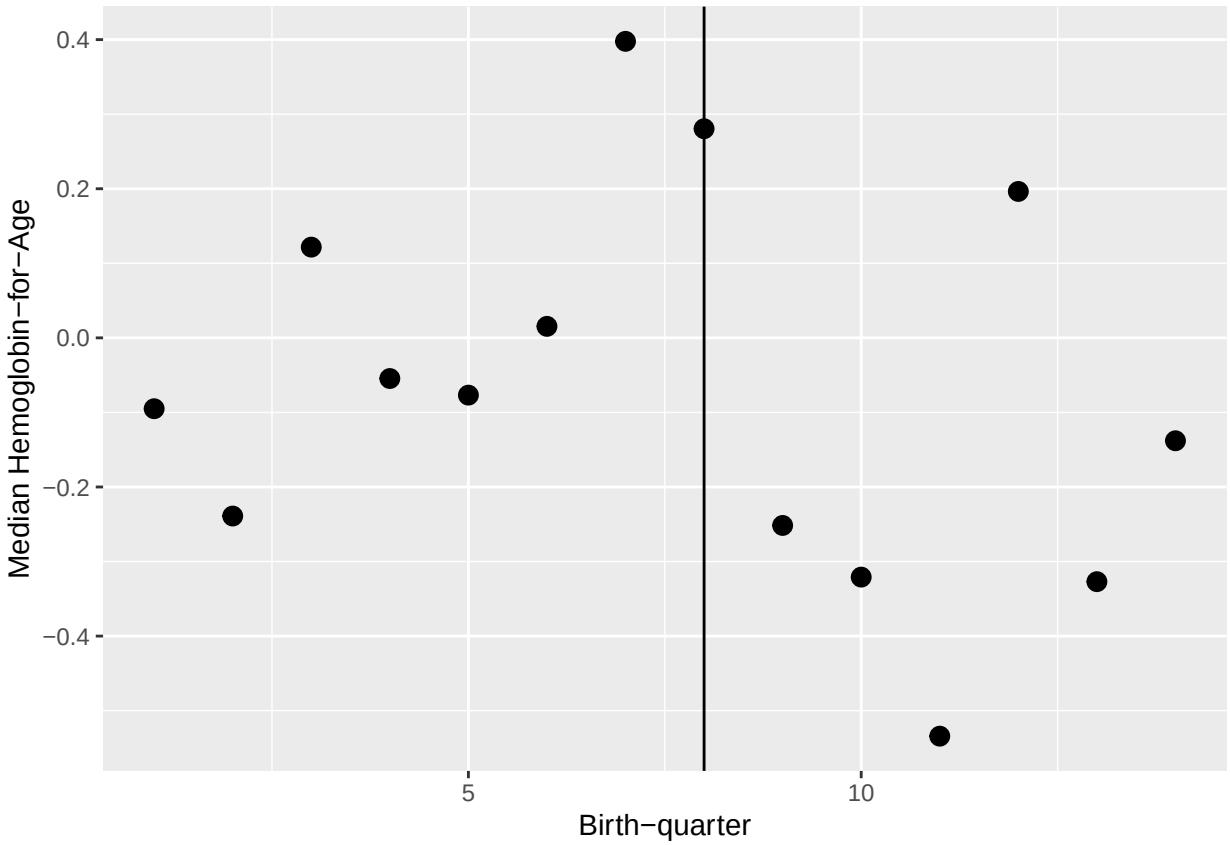
1. The 2008 crisis had a negative impact on the maternal nutrition of urban-dwellers, but not rural-dwellers;
2. The effect of the 2008 crisis on nutrition was greater for the poor than the rich;
3. The effect of the 2008 crisis on child hemoglobin was apparent in 2010, but not in 2012; and,
4. For urban dwellers, the effect of the 2008 crisis was greatest for those in and around to Dakar.

For those affected– the urban poor– the estimated effect of being exposed *in-utero* to the 2008 crisis is large: between 10-14 g/L of hemoglobin, where the cutoff for anemia is defined at 105 g/L. These effects are best understood as lower bounds. The fetal origins approach implicitly compares those *in-utero* to those already born, meaning the estimated effect of exposure is net any negative impact on children born before the crisis. Also, given the persistence in iron stocks and budget constraints, those conceived after the crisis also likely felt some negative effects. I run a more narrow regression in the Appendix, which discards half the data and focuses on those exposed and those born just before exposure. This estimated effect of the crisis is larger than in my main specification, further supporting the argument that my main results form a lower bound.

There is substantial attenuation of the effect of the 2008 crisis between 2010 and 2012. This should not be interpreted as recovery of health, but rather as the masking of an informative signal. Iron is a critical nutrient in human development, and deficiency impairs cognitive and motor development (Lozoff 2007). Looking at the effect of *in-utero* shocks too late in life can mask important differences. Two children who seem the same in 2012 in terms of hemoglobin, might have had very different hemoglobin in 2010, and therefore be on very different biological trajectories.

Although exposure was defined with respect to prices, the 2008 crisis is a coarse instrument. During the crisis there were protests, unrest and a change in the type of agricultural production, with an increase in staples like millet and sorghum. Thus, we cannot rule out that the estimated effect of the crisis has more to do with the general characteristics of the period than with prices *per se*. For example, the strong effect in Dakar might come from social unrest, as there was rioting in Dakar during the period.

Similarly, distance-to-Dakar should be considered as a general proxy for integration to the world market. Distance captures a broad set of socio-economic conditions. Areas further from Dakar are poorer, have less education and have more subsistence agriculture relative to commercial agriculture. Thus, the estimated effect will not be of transportation costs alone, but rather the combination general set of characteristics associated with being further from Dakar. Moreover, when I plot hemoglobin-by-birth-quarter for each region of Senegal, Dakar is the only place where an there is an obvious discontinuity in hemoglobin around exposure to the 2008



crisis.

This result suggests that distance to Dakar might be less important than the qualities of Dakar itself.

6.2 Connection to the Broader Literature

My results support the existing view that increases in food prices negatively affect urban households while having little effect on the rural populace (Cohen and Garrett 2010; Dimova and Gbakou 2013). While the results are too noisy to reject the null hypothesis that the crisis had no effect on rural households, I do find a slight improvement in hemoglobin for rural-dwellers exposed to the crisis *in-utero*. This might due to the increased income from sale of food, or to substitution toward locally produced, high-iron staples like millet and sorghum.

All of this discussion and analysis has been *specific to Senegal*. One would expect very different effects of the 2008 crisis in different countries. As shown above, millet production actually increased during 2008. This may reflect a reserve capacity for food production, or the good fortune of high yields coinciding with high price of imports. In either case, it suggests significant value in having reserve domestic food production capacity. Given a sudden increase in food prices, households may adapt by shifting production towards food. Even if adaptation isn't possible, the independence of world price and local weather conditions imply that having some domestic production and some imported food supply would shrink the variance of food consumption. A country which completely abandons local food production in order to specialize in cash crops, would be in a significantly more vulnerable position than Senegal was in 2008. Thus, my work may provide suggestive evidence to the arguments for "food-sovereignty" in the food-policy literature (Rosset 2008).

7 Conclusion

I find suggestive evidence that the 2008 world food crisis negatively affected the health of women, as shown by the health of their children, in urban Senegal. The effect is strongest and most consistent around Dakar. Within the context of the food-policy literature, my results are direct evidence to the hypothesis that food price shocks negatively affect urban households, without having a detectable effect on rural households.

My results suggest that the effect of a negative shock to maternal nutrition may have a large effect on the hemoglobin of infants who were *in-utero*, outweighing the effect of the same shock on children who are already born. This negative shock is apparent 2 years ex-post, but has dissipated 4 years ex-post. As iron is a critical micro-nutrient for fetal and infant neuro-development, although the shock has dissipated, its effect on long-run health and welfare will remain.

These results are evidence that the health of poor people in poor countries depends on the movement of international markets. The 2008 food price crisis is a specific episode which had a strong, negative impact on the urban-poor of Senegal. Since 2008, urbanization and food imports have continued to increase in Senegal. Incomes have also risen. Whether the country is more or less vulnerable today to a price shock like that of 2008 is difficult to answer.

We expect urban populations to be less vulnerable to drought and other natural disasters than the rural populace. There would then seem to be a trade-off where urban, well-connected populations are more vulnerable to the market, while rural, remote populations are vulnerable to nature. Here we must bring in that universal feature of social analysis: class. In either case, it is the poor who are vulnerable and the rich who are not. Any policy which considers mitigating the effect of these shocks, whether they come from the market or from nature, must keep poverty in the foreground.

This is a fetal origins paper. In general, the fetal-origins literature tends to concentrate on the fetus. I would like to draw attention to the mother. Poor fetal nutrition comes from poor maternal nutrition. Any discussion of infant health must consider gender inequality (Osmani and Sen 2003). The negative consequences of the 2008 crisis on child hemoglobin are direct evidence of the malnutrition of poor, urban women of Senegal.

8 Future Research

In this project, I have found the the 2008 crisis had a large, negative impact on the urban-poor of Senegal. I also find that the effect diminishes with distance-to-Dakar. The distance from a particular community to Dakar is related to a host of other characteristics, such as wealth, concentration of land-ownership, and local food production capacity. An inviting avenue for future research would be the systematic exploration of these relationships, and how each of these underlying variables is related to the effect of the 2008 crisis. Previous literature has focused on the effect of an increase of food prices on households who are net-sellers vs net-buyers of food. More coarsely, I could explore regional effects based on whether a particular region is a net-importer or net-exporter of food.

Another route would be to expand my analysis to include Senegal's neighbors like Burkina Faso, Ghana, Cote D'ivoire, Liberia and Guinea. While I have argued that my results are internally valid for Senegal, it is an open question whether they apply more broadly. Conducting the same test over multiple countries could provide evidence for or against a stable pattern. Given that my dataset is relatively small, expanding coverage to more countries would also improve the power of my tests and allow for a greater number of hypotheses to be tested, such as the effect of land-ownership.

More ambitiously, I would like to connect my research to the more general question of market integration and food-security. As outlined above, there appears to be a trade-off between vulnerability to shocks from nature and from the world market. In this paper, I have explored an extreme case of the latter. In future research, I hope to explore the effect of drought on urban and rural populaces in concert with analysis of the 2008 crisis, in order to determine the relative magnitude of each of these effects.

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10 Appendix 1: A guide to Senegal's Regions

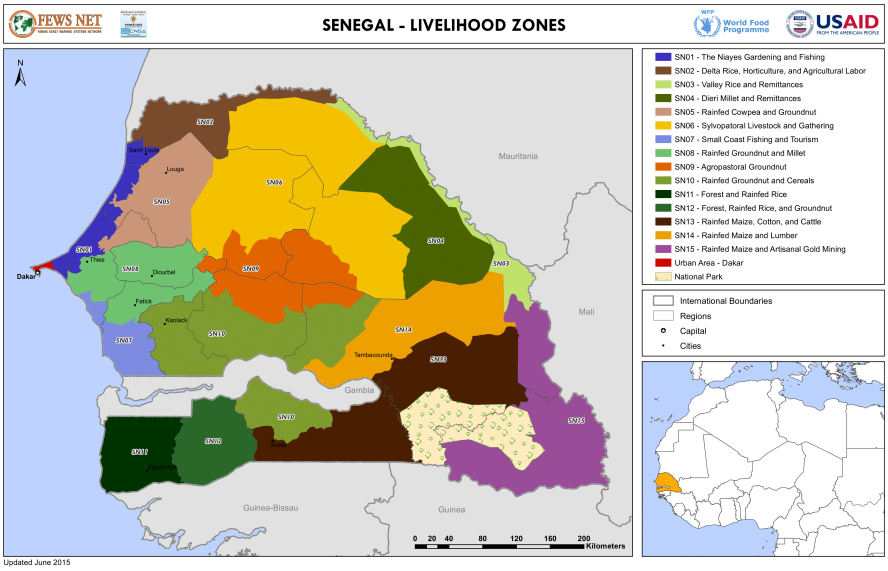

Figure 15: FEWs Livelihood Maps

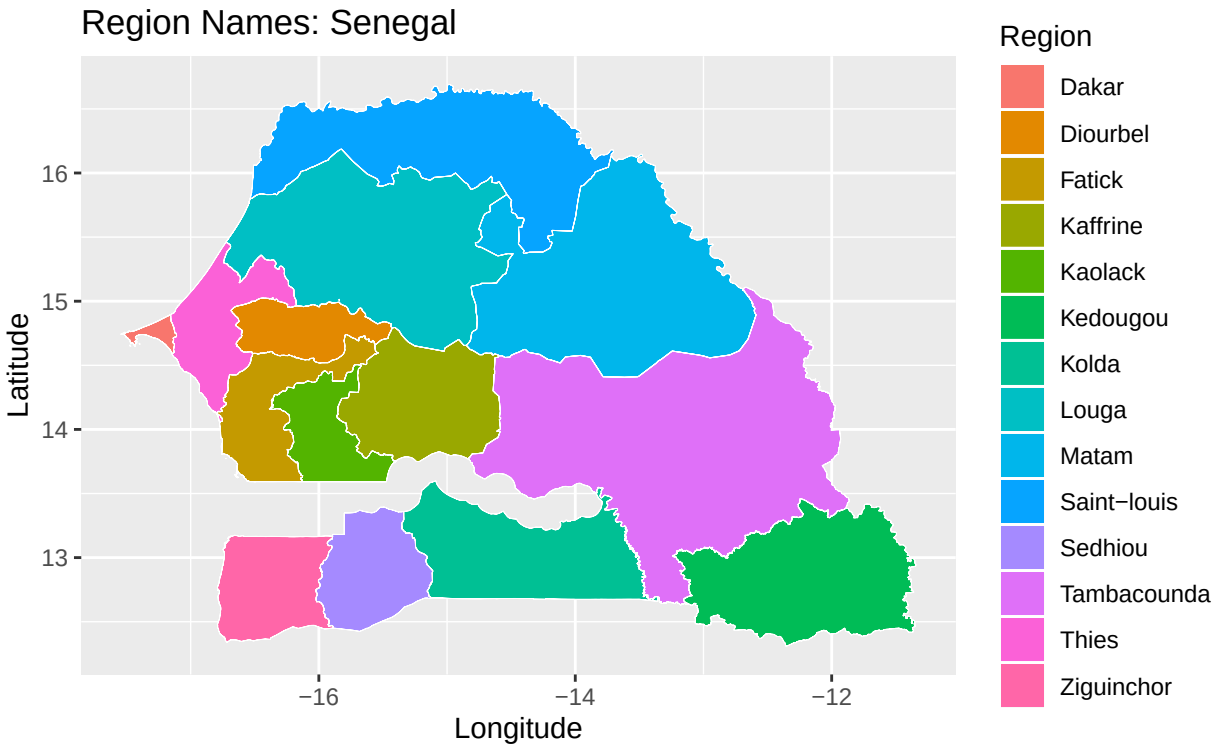

Figure 16:

region	Wealth	Land	Urban	N
Dakar	0.12	0.11	0.78	1444
Diourbel	0.03	0.38	0.30	2484
Fatick	-0.04	0.76	0.20	1947
Kaffrine	-0.07	0.84	0.14	2104
Kaolack	-0.02	0.68	0.28	2715
Kedougou	-0.05	0.80	0.23	1207
Kolda	-0.07	0.81	0.21	2074
Louga	-0.01	0.72	0.24	1996
Matam	-0.02	0.78	0.26	1913
Saint-louis	0.01	0.56	0.41	1751
Sedhiou	-0.05	0.83	0.24	1649
Tambacounda	-0.07	0.80	0.22	2049
Thies	0.03	0.55	0.38	2263
Ziguinchor	0.00	0.48	0.44	1029

10.1 Identification: Rainfal Anomaly in Senegal

Below I plot the rainfall anomaly for the Sahel region from 1950-2017. We see that 2007-08 was unremarkable. We have already seen in the introduction that domestic food production was relatively high in 2008.

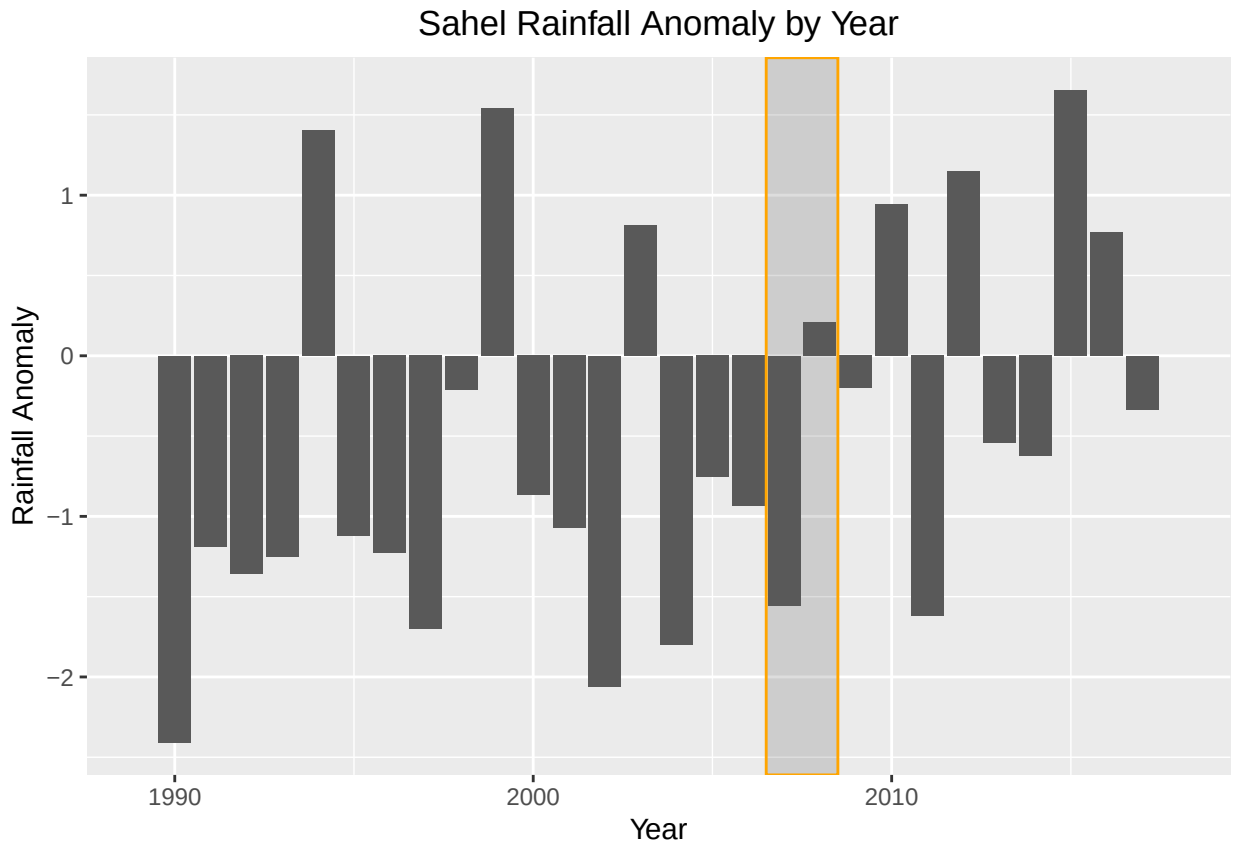

Figure 17: Searching for coincidence

11 Appendix 2: Alternate Specifications

Here I run several alternative specification to those reported above. They generally support my conclusions. Of particular importance in the narrow-window regression, which finds even stronger effects than my main specification.

11.1 Narrow Window

In the main specification, we are comparing infants exposed to the 2008 crisis *in-utero* to two control groups:

1. Those who are exposed in childhood; and,
2. Those who are conceived after the crisis.

For reasons discussed above, including breastfeeding and delayed transmission of shocks, we would expect both of these groups to also experience a negative effect of the crisis. Thus, the ideal control group is to narrow the regression to the window just around those who had just been born and those who were still *in-utero*. I did not do this as my main specification because it would involve throwing out much of the data set, and would reduce my power. Next I report a more narrow specification, where I have thrown out over half the data-set. The data are restricted to those who were born between April 2007 and July 2009. The

while the standard errors increase, the point estimates increase in magnitude, as expected if the effect is concentrated in these groups.

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.0088	−0.3547***	0.2995	0.0664
	(0.1317)	(0.0840)	(0.2102)	(0.2049)
Exposure	−0.0657	0.0877	−0.2669	−0.1306
	(0.3728)	(0.2038)	(0.3921)	(0.4028)
Num. obs.	446	1017	424	888
Percentile	0.5000	0.5000	0.5000	0.5000

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 7: Restricted Sample Results

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	0.2215	−0.4824***	0.0106	−0.3154
	(0.2167)	(0.1428)	(0.3218)	(0.2691)
Exposure	−1.5196**	0.2737	−0.3105	0.8541*
	(0.5727)	(0.2900)	(0.5726)	(0.4808)
Distance to Dakar	−0.0568	0.0522	0.0707	0.0944*
	(0.0397)	(0.0405)	(0.0608)	(0.0497)
Wealth	1.0099	2.3263**	0.5930	1.4001
	(1.3187)	(0.7614)	(1.8293)	(1.0525)
Distance to Dakar * Exposure	0.2574**	−0.0299	−0.0020	−0.1905**
	(0.0899)	(0.0761)	(0.1084)	(0.0805)
Wealth * Exposure	4.0122	0.0928	1.9335	2.1867
	(2.8503)	(1.9586)	(2.8799)	(1.8575)
Num. obs.	446	1017	424	888
Percentile	0.5000	0.5000	0.5000	0.5000

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 8: Restricted Sample Results

I take this as convincing evidence of the effect of the 2008 crisis. However, it was not the specification to which I pre-committed, so I have included it in the appendix as supporting evidence, rather than as my main specification.

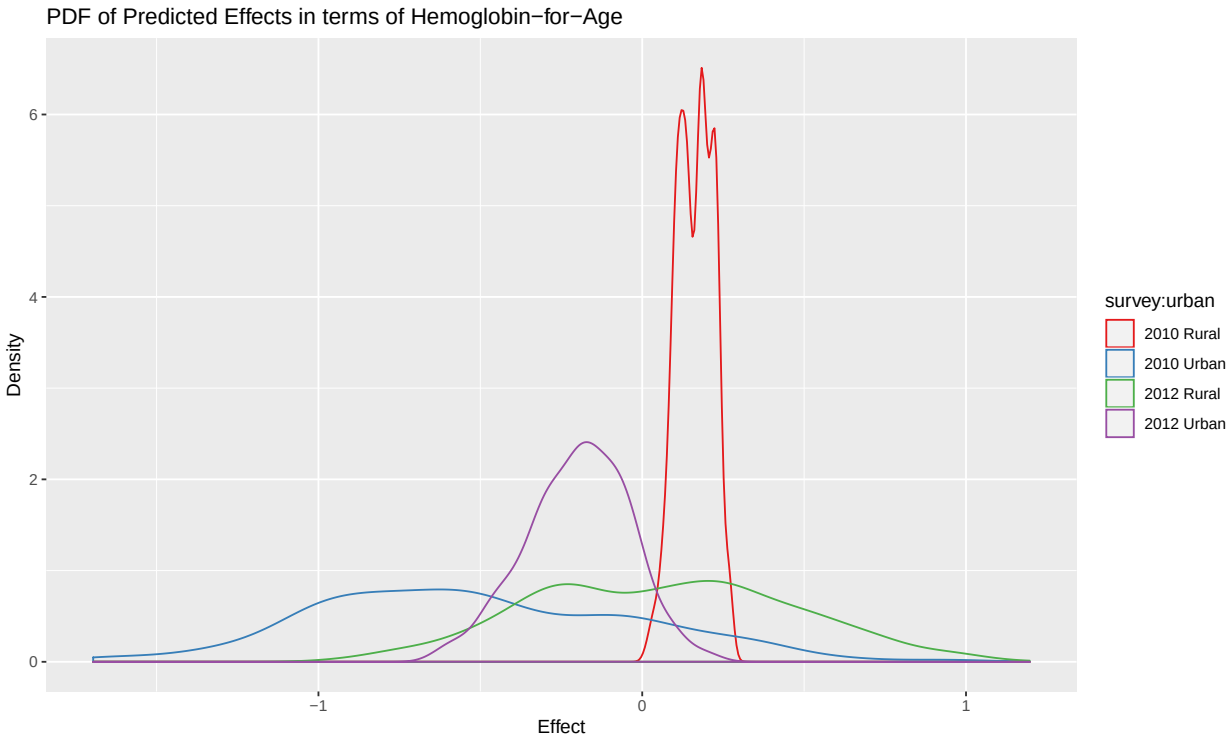

Figure 18:

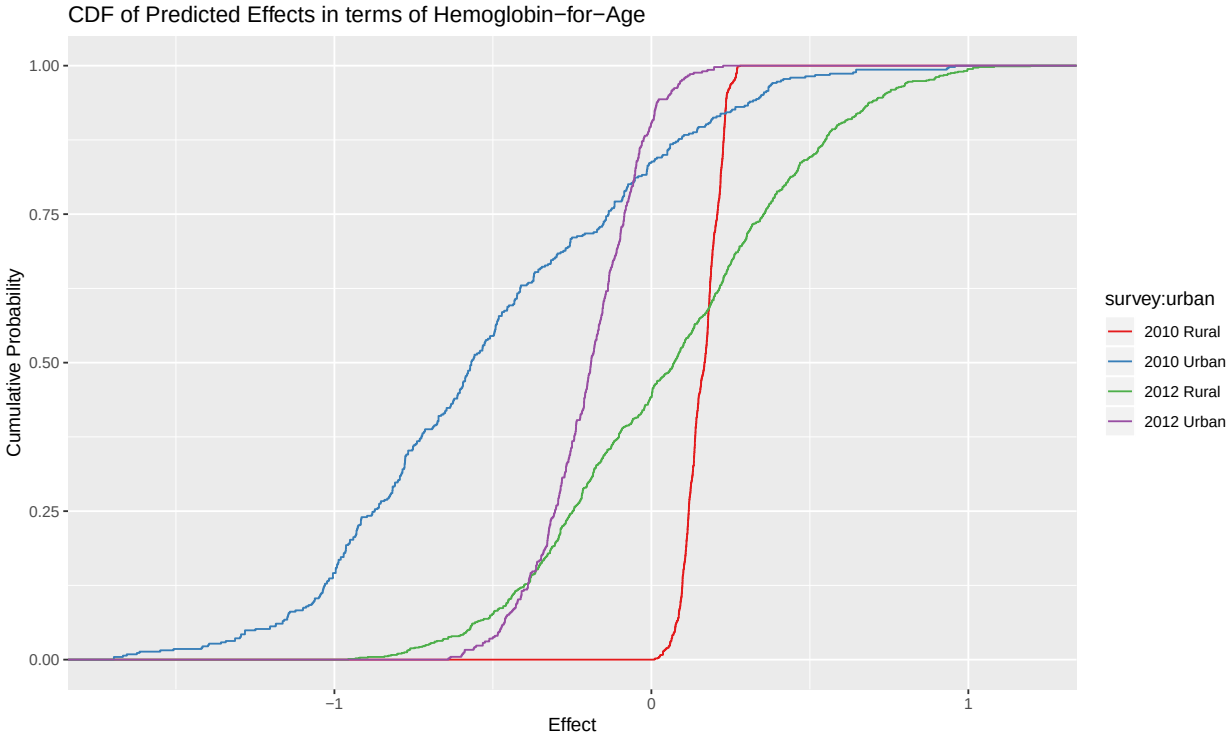

Figure 19:

Under the narrow specification, the median effect of the crisis on urban-dwellers in 2010 is greater than -.5 standard deviations. Thus, the typical urban dweller of Senegal suffered a -8 g/L shock to hemoglobin.

11.2 Spatial Distribution of Effects

The regressions above do not allow for spatial heterogeneity of response, except through distance-to-Dakar. Therefore, I run a more flexible regression, which allows the effect of exposure to vary by region:

$$H_i = \alpha_0 + \beta Exp_i + \delta Exp_i * Region + \alpha_1 MOB_i + \alpha_2 MOB_i^2$$

I report the predicted effect of exposure in each region. A guide to the regions of Senegal, with names and distribution of variables of interest, can be found in the Appendix.

I do not control for urban-rural or for wealth, as I want to capture the regional effects inclusive of differences in wealth and the proportion who are urban. We see that Dakar and its neighbor Thiès are most strongly affected, although both are relatively wealthy. Outside of these two regions, distance-to-Dakar seems to have no effect, indicating a nonlinear relationship of distance-to-Dakar and effect of the 2008 crisis. I include the results for 2012 in order to compare magnitudes. We see that for many regions the effect is inconsistent across years. For Dakar and Thiès, on the other hand, the effect of the crisis is consistent across years and persists into 2012.

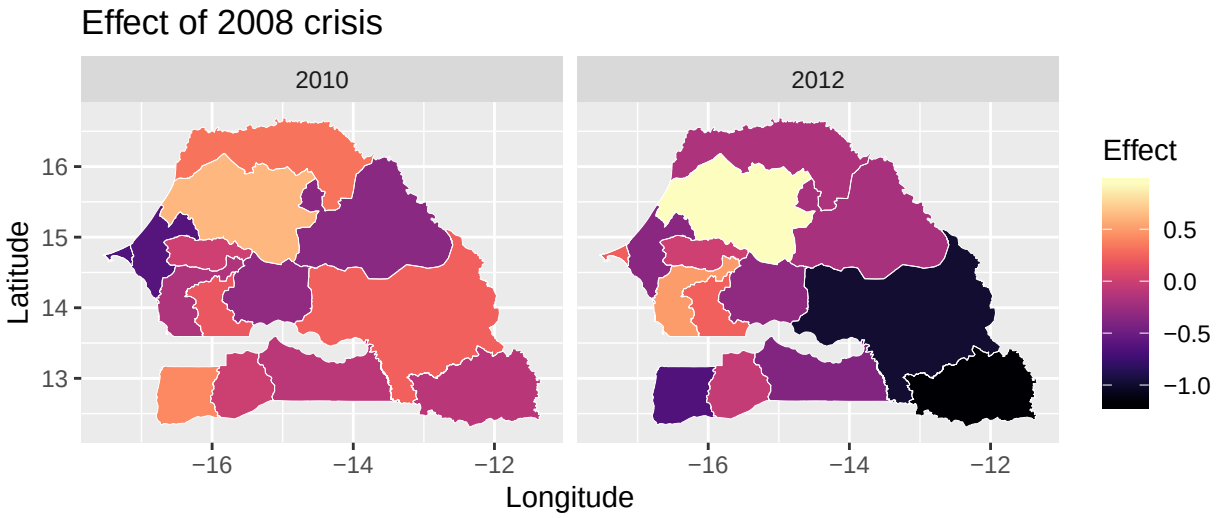

Figure 20: Effect of the 2008 crisis across Senegal by survey-year

11.3 Lead-lag

As noted in the prices section, it can take several months for world prices to diffuse to cities in Senegal. Thus one might expect the exposure variable to me mis-assigned, as those who were really exposed to the shock were born several months later. Therefore, I take the twice lagged values of exposure, and regress them in the same way as before. Interestingly, the effect of the crisis is now negative for both urban and rural dwellers. This suggests that the price effect takes longer to be transmitted to rural areas.

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.0653	−0.2955***	0.1718***	−0.1281***
	(0.0444)	(0.0353)	(0.0337)	(0.0241)
Exposure	0.2749*	−0.0152	−0.2041	0.0489
	(0.1175)	(0.0774)	(0.1088)	(0.0808)
Num. obs.	974	2250	1573	3492
Percentile	0.5000	0.5000	0.5000	0.5000

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 9: Test for Lagged Effects

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.2252	−0.2387**	−0.0743	−0.0854
	(0.1173)	(0.0744)	(0.0845)	(0.0450)
Exposure	−0.0358	−0.1130	0.0753	0.3918*
	(0.4015)	(0.1811)	(0.2456)	(0.1744)
Distance to Dakar	0.0201	0.0067	0.0503**	0.0159
	(0.0271)	(0.0186)	(0.0182)	(0.0130)
Wealth	1.5622*	1.9714***	1.4877**	2.2306***
	(0.6843)	(0.3867)	(0.5612)	(0.3299)
Distance to Dakar * Exposure	0.0678	0.0291	−0.0618	−0.1016*
	(0.0740)	(0.0446)	(0.0596)	(0.0558)
Wealth * Exposure	0.3240	−0.3998	−1.2766	−0.4449
	(2.1362)	(1.2297)	(1.7847)	(1.1000)
Num. obs.	974	2250	1573	3492
Percentile	0.5000	0.5000	0.5000	0.5000

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 10: Test for Lagged Effects

11.4 Selection

Next I test for fetal selection. Female fetuses are more robust than males, so if the 2008 crisis caused selection due to *in-utero* stress, we should see an increase in the number of girls. I find no such result.

	Girl
(Intercept)	1.4863***
	(0.0061)
exposure	−0.0007
	(0.0203)
R^2	0.0000
Adj. R^2	-0.0001
Num. obs.	8358
RMSE	0.4999

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 11: Test for Selection

11.5 Seasonal Fixed Effects

Here I include my main specification, with seasonal fixed effects included. The results remain largely unchanged.

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.0266	−0.2989**	0.0489	−0.2889
	(0.1469)	(0.1078)	(0.4356)	(0.3306)
Exposure	−1.2827**	0.0912	−0.4412	0.9013
	(0.4842)	(0.2426)	(0.7069)	(0.5201)
Distance to Dakar	−0.0163	0.0400	0.0585	0.0913*
	(0.0342)	(0.0247)	(0.0635)	(0.0481)
Wealth	1.6015	2.1964***	0.0502	1.0803
	(0.8661)	(0.4669)	(1.7799)	(1.0480)
Distance to Dakar * Exposure	0.2131*	−0.0225	0.0151	−0.1867*
	(0.0828)	(0.0569)	(0.1074)	(0.0772)
Wealth * Exposure	3.4741	0.4866	2.3134	1.4936
	(2.4041)	(1.5720)	(2.8668)	(1.9322)
Num. obs.	696	1612	424	888
Percentile	0.5000	0.5000	0.5000	0.5000

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Table 12: Seasonal Fixed Effects

11.6 Hemoglobin by Region by Birth-quarter

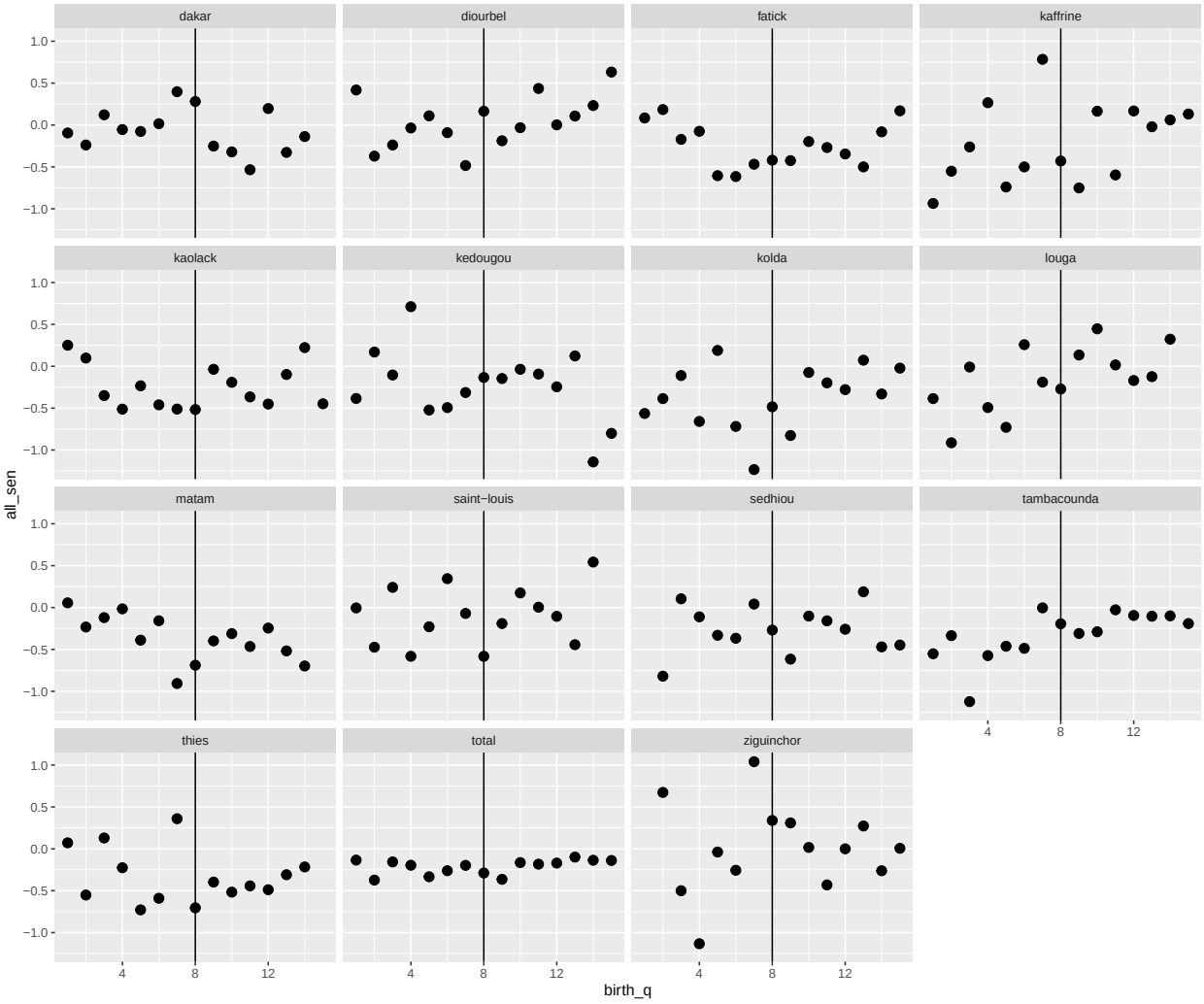

Figure 21: