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. As predicted by the food-policy literature, I find that the 2008 crisis had a large, negative impact on maternal health in urban areas, but a small, positive effect in rural areas. Observing the same urban cohort over time, I find that the effect of exposure is apparent in 2010 but not in 2012, suggesting catch-up in infant health. Iron is a critical nutrient in early-life development, suggesting that this catch-up masks an important negative shock to health. Further research is needed to determine the long-term effects of *in-utero* exposure to the 2008 crisis.

1 Introduction

The world's poor rely increasingly on local and global markets to provide their basic food needs. More than half the world's population now lives in cities, where providing your own food supply is near-impossible. In rural settings the self-reliant peasant has been replaced by the petty-commodity producer and wage laborer (Bernstein 2010). Food entitlements are mediated by market forces, as both rural and urban-dwellers must exchange their labor and produce in return for food. Following the reduction in trade-barriers over the last 40 years, most poor countries now rely on imported foodstuffs to meet domestic demand. While we assume that this integration has led to rising incomes, the resulting specialization has also left the world's poor vulnerable to fluctuations in world markets. A sudden increase in the price of food, for reasons external to the poor economy, may lead to a reduction in food purchasing-power and increased malnutrition.

In early 2008, such an increase was realized. The prices of rice and wheat doubled over the course of a few months, while the prices of other staples increased as well. The causes of the price increase are debated, but include both American agriculture policy and the sub-prime mortgage crisis (Rosset 2008). Rising prices decreased the purchasing-power of consumers around the world, resulting in unrest and riots in low-to-medium-income countries.

The 2008 world food price crisis, as it was named, drew attention from major journals Science and Nature, as well as from the fields of peasant studies, nutrition, and development.[1] Much of the literature is qualitative, as real-time measures of health and welfare are difficult to procure. My work, which ties the crisis to observed changes in maternal health, thus fills an important hole in the existing literature. 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).

[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); Compton, Wiggins, and Keats (2010); Headey (2013).

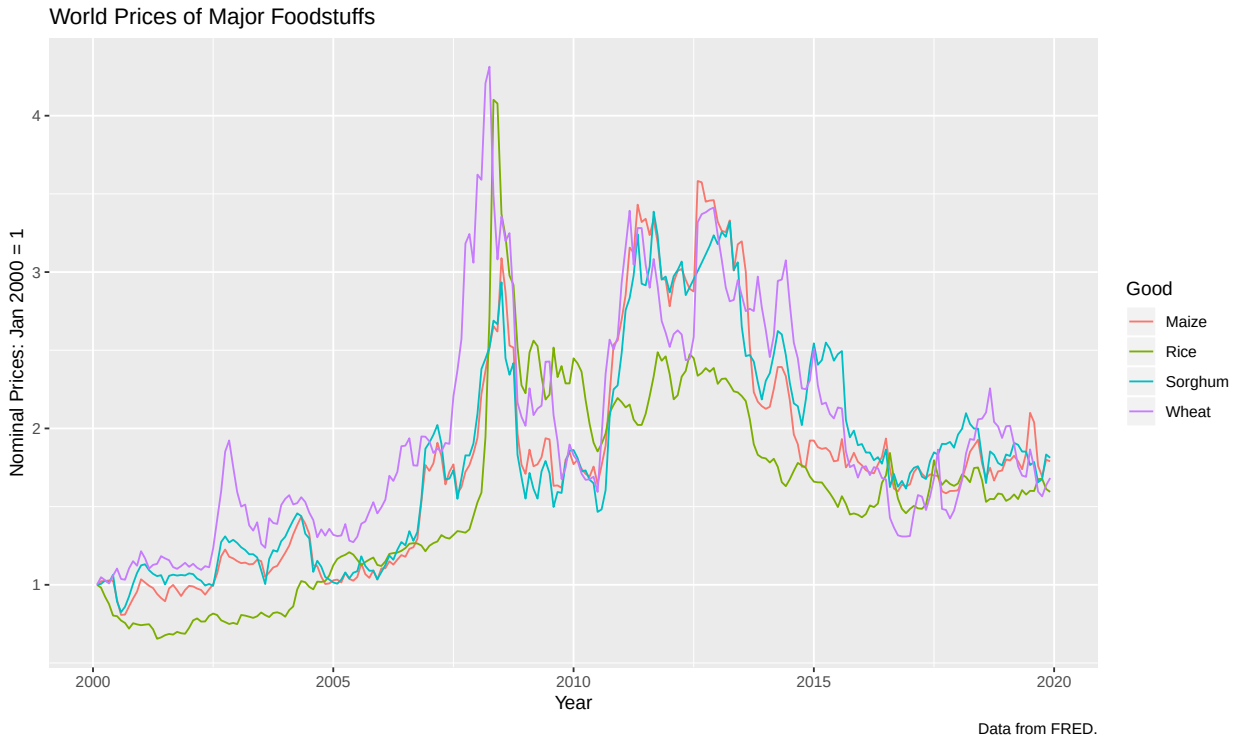

Figure 1: The 2008 Food Price Crisis

Despite the attention surrounding the crisis, there are few causal evaluations of its impact on nutrition and health– a limitation noted by the literature (Compton, Wiggins, and Keats 2010). Identifying the effect of the crisis is challenging because the shock affected the entire world at a specific time, leaving no obvious control group for comparison. In terms of existing research, Arndt et. al. (2016) approach this problem by using propensity-score matching to compare the height-for-weight of children observed during the crisis to those observed afterwards. Dimova et. al. (2013) take a different approach, examining the effect of the crisis on a synthetic measure of welfare, rather than looking at health.[2] While both papers contribute to our understanding of the quantitative effect of the 2008 crisis on health, they are limited by their datasets. The 2008 crisis affected both weight-for-height and household welfare for *everyone*, making it difficult to construct a compelling control group. Building off of these previous studies, my contribution is to find otherwise similar individuals who are differently affected by the 2008 crisis.

I exploit the biological mechanism of iron-transmission and hemoglobin formation in fetuses and infants. For reasons discussed below, fetuses are more sensitive than infants to nutritional iron-availability. Therefore, those who experienced the 2008 crisis as infants form a compelling control group for those who experienced the crisis *in-utero*. This strategy allows me to explore the effect of the 2008 crisis on maternal-fetal health. I examine the case of Senegal, which imports much of its food supply and has nationally-representative surveys which measure hemoglobin 2 and 4 years after the crisis.

Using a fetal origins approach provides a clear identification strategy and allows me to disentangle the effect of the crisis from seasonal fluctuations, addressing one of the limitations pf 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. The fetal origins approach allows me to bring empirical evidence to bear on previous work. Although lacking quantitative evidence in this regard, the consensus in existing literature is that the 2008 crisis had a strong negative effect on the urban population and a weak positive effect on the rural (Barrett 2010; Dimova and Gbakou 2013). My results support this consensus.

[2]My paper most closely resembles that of Block et. al. 2004, although the crisis of interest differs.

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* insults to iron *persist* as the infant ages has received little attention. Previous work has focused on small, controlled trials involving anemia and iron supplementation, while I use a large, nationally representative dataset.

1.1 Senegal in the 2008 Crisis

Senegal is a small, poor economy on the western coast of Africa. In 2008, the population was 12 million and the GDP per capita was $2,664 (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 outside of the river valleys. Population is concentrated around Dakar, with smaller concentrations in the river basins of the north and south. See Appendix 1 for more geographic information about 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. Foods which are mainly imported (rice and wheat) have increased in importance in Senegal over the past thirty years, while locally produced staples millet and sorghum have declined in per-capita terms.

In figure 2, I plot consumption, imports and import expenditure of the main staples of Senegal. 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. Still, total expenditure on imported rice increased, suggesting that consumers could not fully mitigate the effect of the price increase through substitution.

The world price spike is apparent in retail prices in Senegal. In figure 3, 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. 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.[3] I take the weighted average of the commodity prices in real USD, multiplied by the CFA-USD exchange rate in order to get prices in terms of local currency. In figure 4, I plot the index in log-terms below, so that proportional changes can be easily read. This index does not enter directly into my analysis except to illustrate the timing and severity of the 2008 crisis in Senegal.

[3]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.

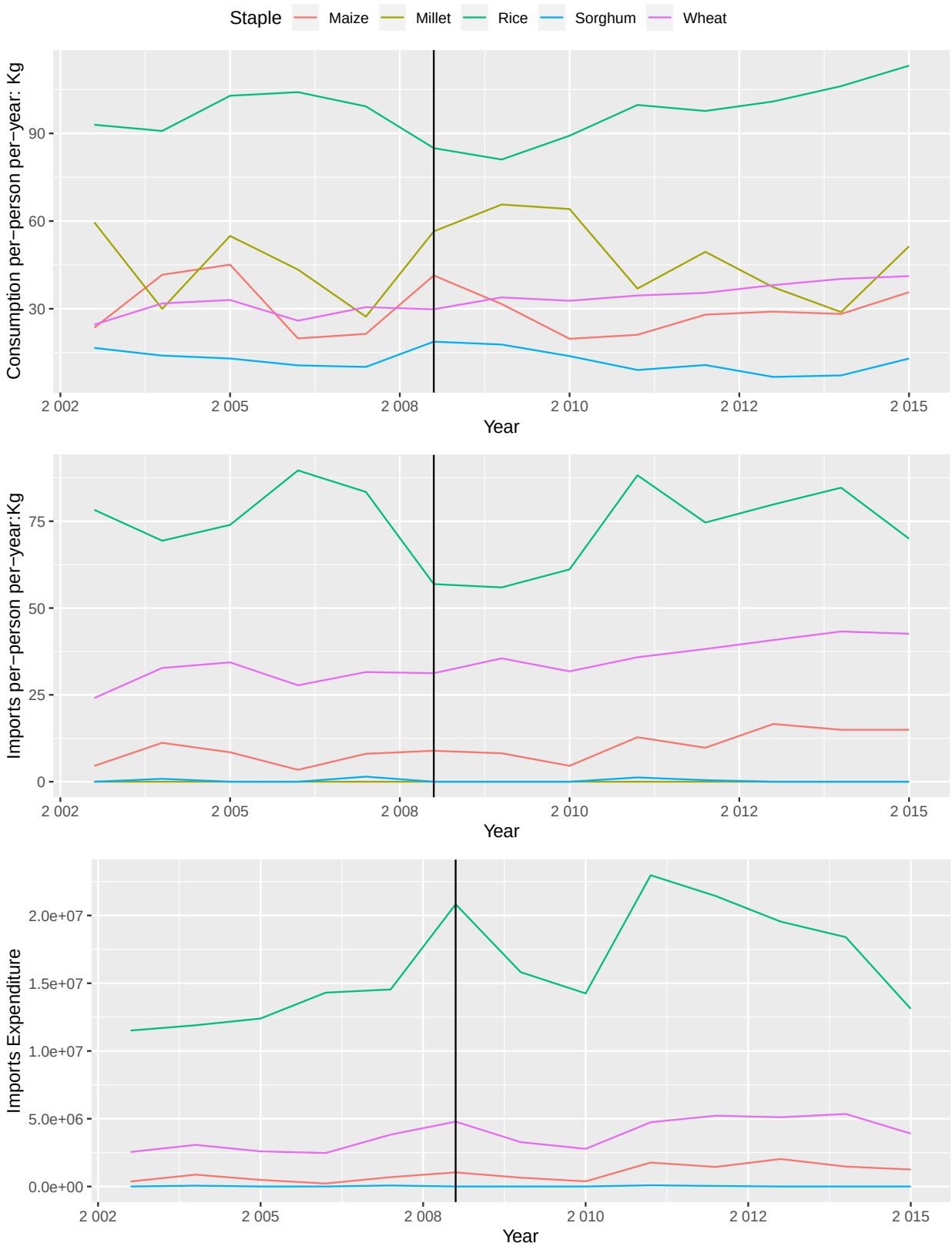

Figure 2: Food-stuffs in Senegal. Rice imports fell substantially during the year of the crisis (2008 indicated by a vertical black line) but expenditure increased.

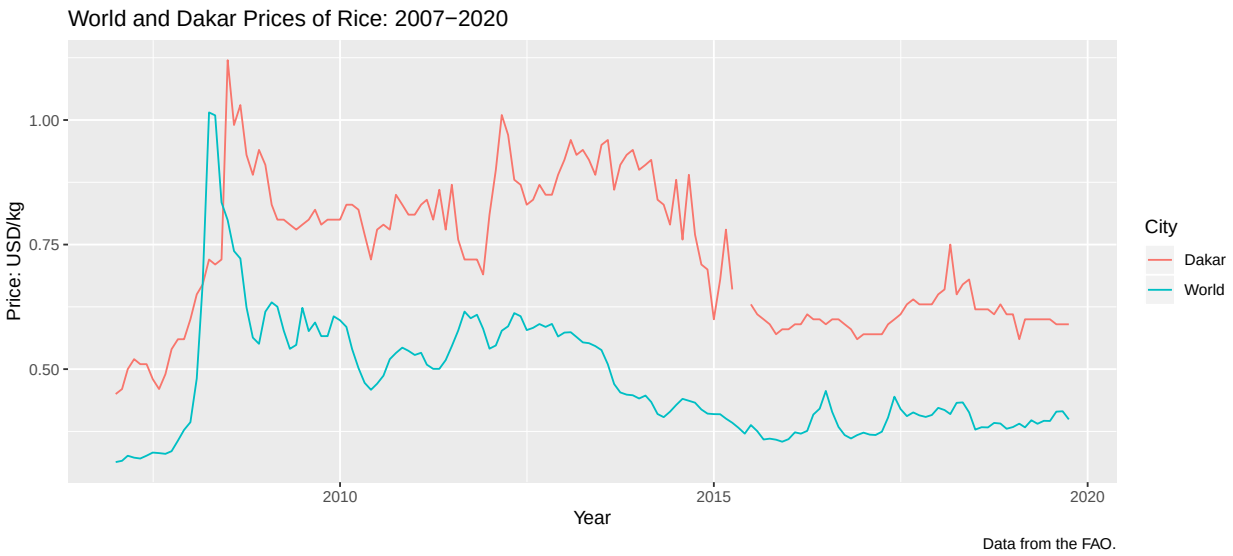

Figure 3: World Rice Price and Retail Price in Senegal

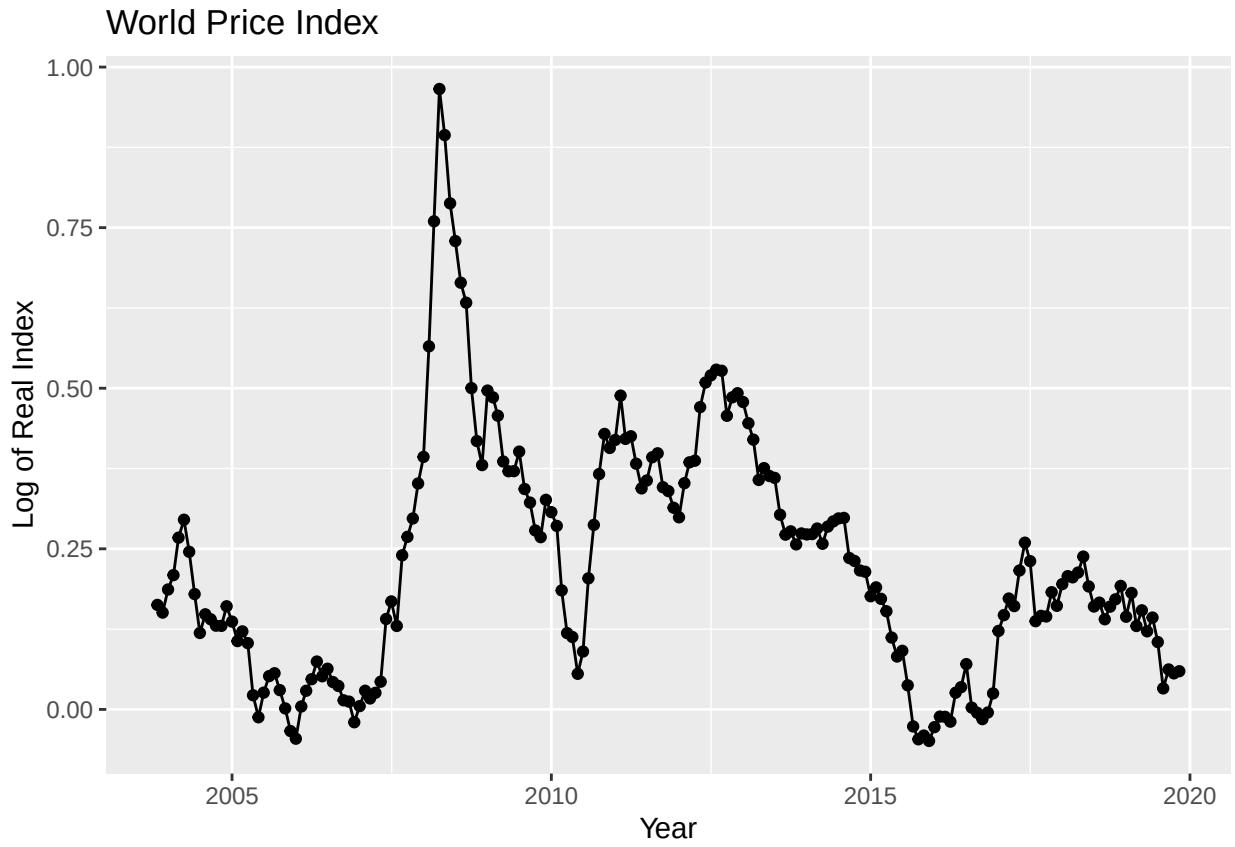

Figure 4: World Price Index, weighted by Senegal iron-consumption

2 Mechanisms

Given an increase in food prices while *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. From the first two steps, I argue that household wealth and distance to the country's port, Dakar, should mediate the effect of the 2008 crisis on health. The last 3 steps describe the biological mechanisms underlying my identification strategy.

2.1 World Price, Local Price and Transportation Costs

The effect of world price on local price depends 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, 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 imported after the increase. Price will be determined by local supply and demand, $\frac{\partial P_l}{\partial P_w} = 0$.
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 $0 < \frac{\partial P_l}{\partial P_w} < 1$.
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 $\frac{\partial P_l}{\partial P_w} = 1$.

Transportation costs also change the relative price of commodities faced by producers, making commercial, export-oriented production less profitable relative to local, subsistence production. Thus, we should also expect food-production 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 the price of a commodity increases there are substitution and income effects which lead to lower consumption of said commodity. Given that the 2008 price increase affected all of the major, imported staples, I would expect the substitution effect to be relatively small.[4] 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 income.

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). There are a host of issues surrounding intra-household allocation and gender-inequality which I will not

[4] 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.

treat in this paper. Instead, I assume that the price effect is transmitted, to some degree, to women of child-bearing age.

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.[5] Because the 2008 crisis wass a period of exceptionally high prices, my results should capture the upper end of the non-linear effect of price. My results would likely not apply to smaller increases in food 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. As urban-dwellers have less opportunity to grow their own food, we would expect the negative effects of a price increase to be greater in the city than in a rural setting. This difference is well-explored in the existing literature on the effect of food price shocks on households in poor countries.

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 fetuses and infants, iron is a critical nutrient, and anemia can impair cognitive and motor development.

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 intestine. 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).

The additional iron required for pregnancy is approximately 1,000 mg, which is nearly half of the total iron stores in a healthy, non-pregnant woman, making the risk of developing anemia particularly high for pregnant women (Lactation 1990). 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.

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.[6] Here I list the CDC's recommended daily intake of iron for mother and infant across different periods, which illustrates this point.

[5] 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.

[6] 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).

Table 1: CDC recommended daily iron intake for women and children.

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 is a much scarcer nutrient during the fetal period than 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 event, we can compare those who had already been born to those still *in-utero*.

3 Data

For my analysis, I use the Demographic and Health Survey (DHS), a nationally representative repeated cross-section of Senegal which measures the hemoglobin of infants and children age 6 months to 5 years. There are two rounds of the Senegal DHS which include children *in-utero* during the 2008 crisis: 2010 and 2012. The DHS data includes location and households characteristics. The variables which I use in my analysis are hemoglobin, child month of birth, household wealth, whether a household lives in an urban *commune* and driving-distance to Dakar.

3.1 Hemoglobin

At birth, a healthy infant has 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 curves are the conditional mean of hemoglobin over month of birth, and the gray banner is the 95% confidence interval of this mean. Each point is the mean hemoglobin for children of a certain age in a certain survey. 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.

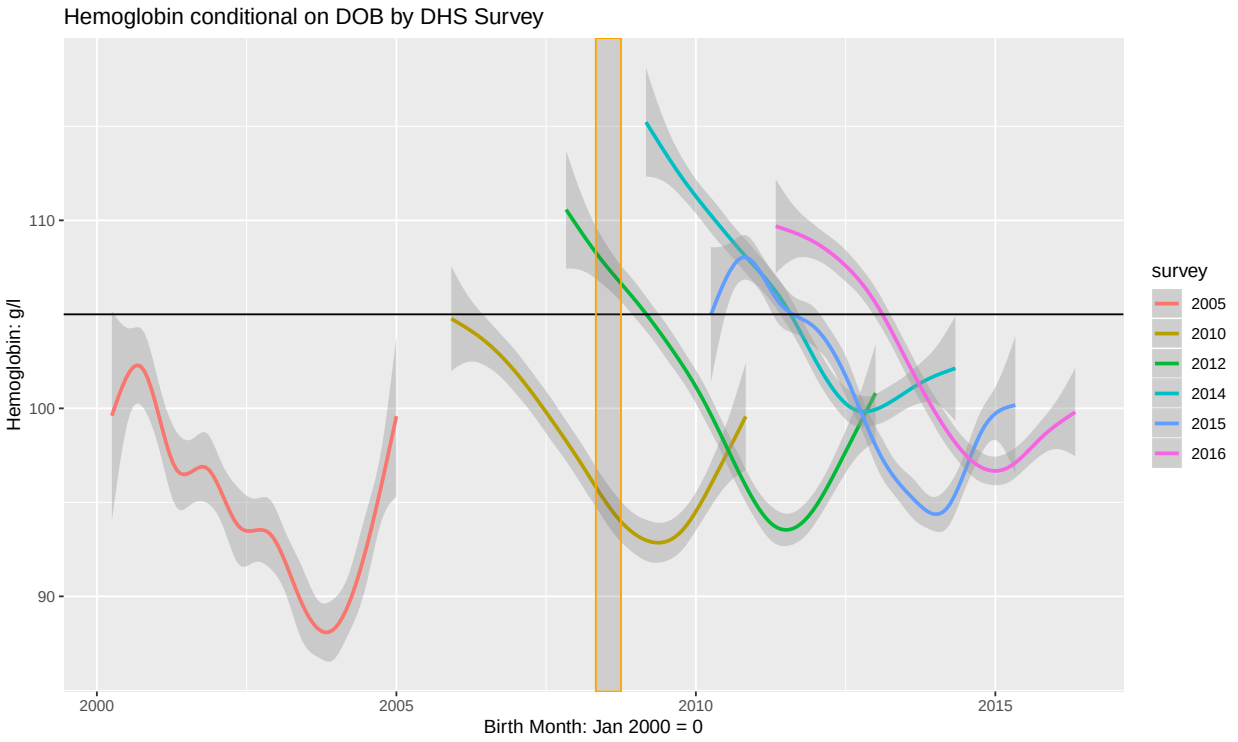

Figure 5: Senegal DHS Hemoglobin data, smoothed over time. The horizontal black line indicates the cutoff for anemia in children.

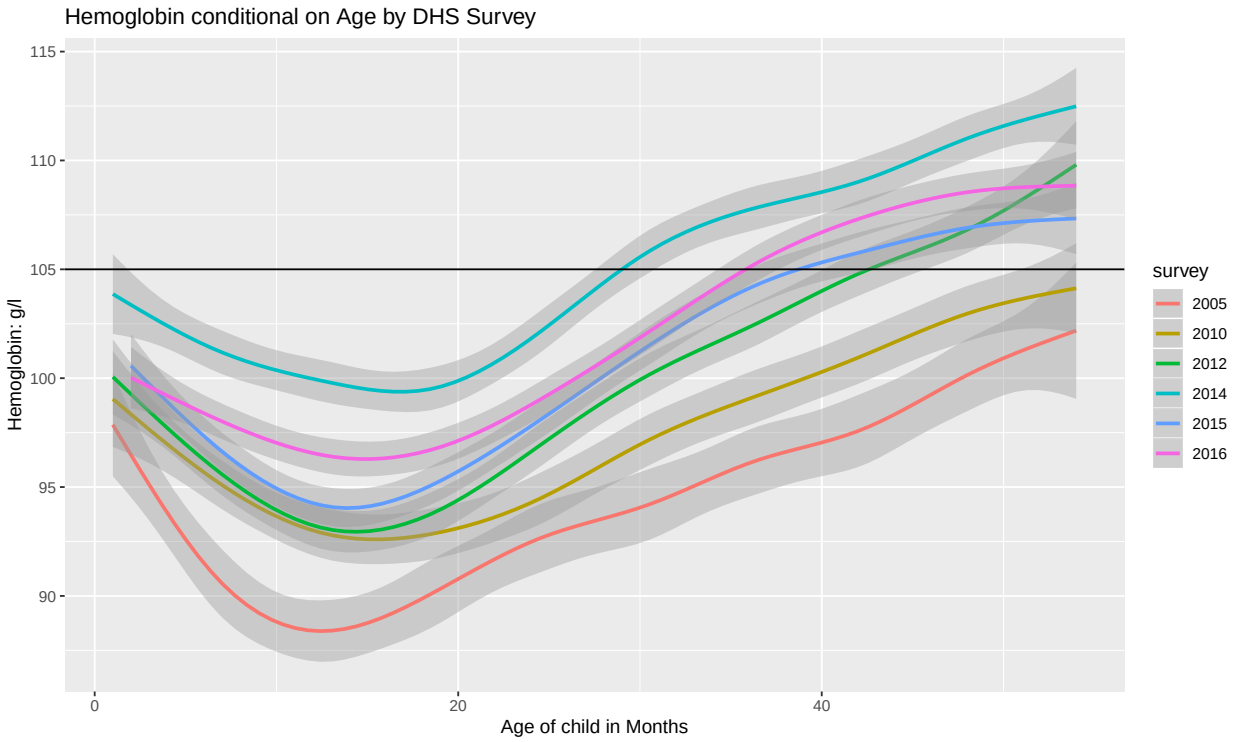

Figure 6: Senegal DHS Hemoglobin data, smoothed over time. The horizontal black line indicates the cutoff for anemia in children.

Figure 8 takes the curves from figure 7 and plots them by age-in-months, rather than by age-of-birth. The cutoff for anemia in infants and children, 105 grams per liter of blood is shown as a black, horizontal line. Below the age of 3, anemia is the norm in this sample. 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, symmetric 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 WHO. The only surveys which provide information on the 2008 crisis 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 across these 5 surveys, I normalize hemoglobin by age in the 2010 and 2012 surveys. The resulting variable, "hemoglobin-for-age," represents a child's hemoglobin, minus the mean hemoglobin for their age, divided by the standard deviation of hemoglobin for their age.

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.

Table 2: Summary Statistics for the Senegal DHS by survey and urban-rural.

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	Survey	Wealth	Distance	Land	N
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.

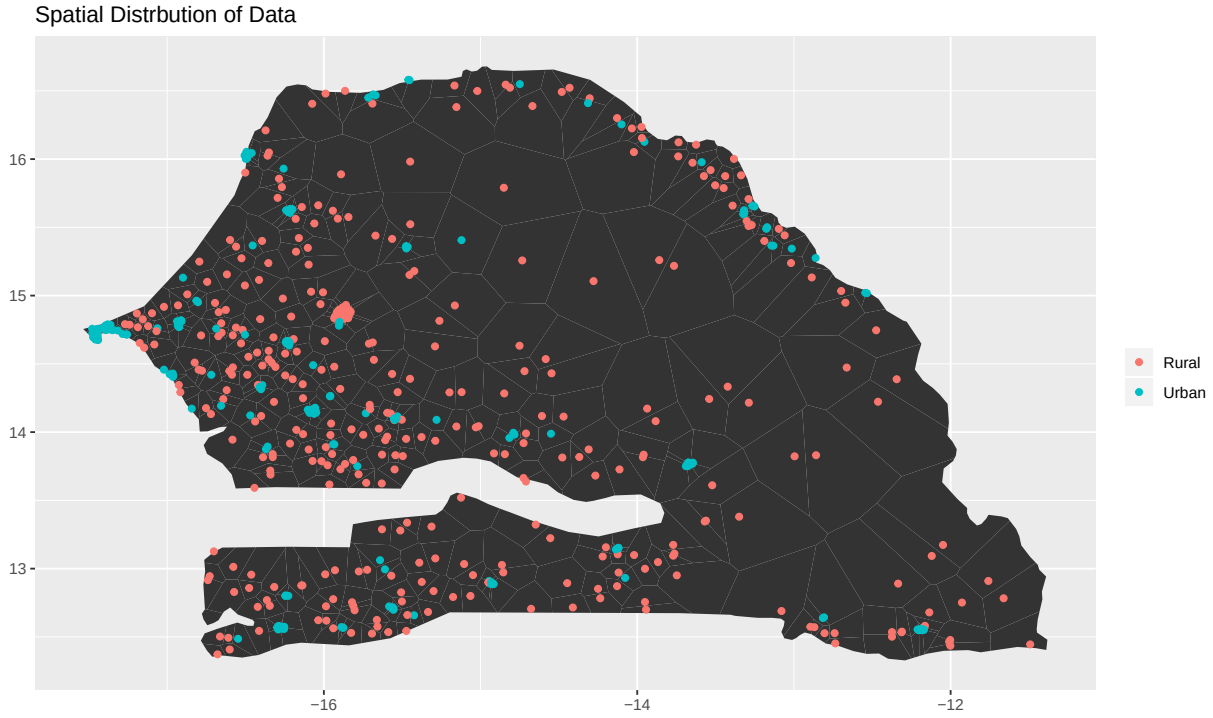

Figure 7: Distribution of DHS Clusters

We see below that the treatment variable, *in-utero* exposure to the 2008 crisis, is balanced across observables. However, I also interact exposure with other variables, which 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. Within 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 exposure is zero, and anyone conceived after September 2008 is excluded. 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.

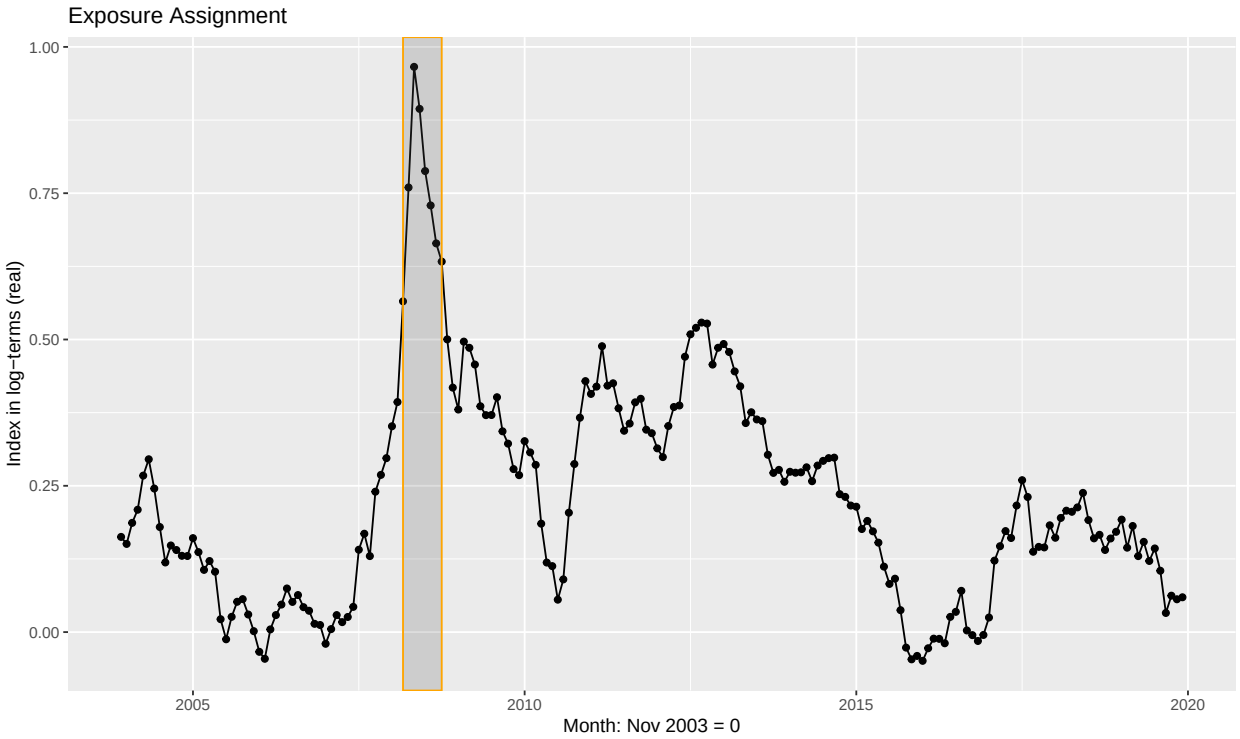

Figure 8: Definition of Exposure to the 2008 Crisis

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 contain the same, exposed cohort, 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: See Appendix 1.1 for summary statistics of variables

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 in 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.

There is no reverse causation from developments in Senegal to world prices, but my results are identified purely on the basis of time and therefore sensitive to temporal coincidence. Although the price shock is *external* to Senegal, it is not necessarily *exogenous* in the modern econometric usage. 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, an earthquake during the time-period would violate my identification strategy. I have found no evidence of developments from *within* Senegal which would coincide with the 2008 price spike.[7]

4.2 Specifications

I present two types of evidence:

1. The deviation from a quadratic time trend[8] of the exposed cohorts; and,
2. The median hemoglobin-for-age plotted by birth-quarter.

I use median-regression, rather than least-squares, as it is insensitive to the response of the extremes of the hemoglobin distribution.[9] I run the following regressions separately by survey and urban, in order to allow time trends to differ across the groups. The equivalent single regression would be fully saturated with respect to the survey and urban dummy variables. I exclude children born after the completion of the 2008 crisis because I expect there to be lagged effects of the crisis. The appropriate control group are those born before the crisis began.

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

[7] I would welcome any suggestions for potential coincidental factors. Falsification, after all, is the heart of science.

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

[9] 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 their families socio-economic position. At the low end, a certain level of hemoglobin is required by the body, and it cannot diminish beyond some point without causing death.

Where H_i is hemoglobin-for-age, Exp_i is the fraction of gestation during the crisis.

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

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

Where $Dist_i$ is the driving time to Dakar for the cluster of individual i, $Wealth_i$ is the DHS wealth index for the household of individual i.

My hypotheses are that $\delta > 0$ and $\rho > 0$. In the first regression, I expect that $\beta < 0$. In the second specification, the sign of β depends on the underlying distributions of wealth and distance-to-Dakar.[10]

5 Results

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

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.0088	−0.3547***	0.2995	0.0664
	(0.1303)	(0.0850)	(0.2140)	(0.2277)
Exposure	−0.0657	0.0877	−0.2669	−0.1306
	(0.3594)	(0.2050)	(0.4066)	(0.4442)
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 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. The black line represents the start of cohorts exposed to the 2008 crisis *in-utero*.

[10] 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.

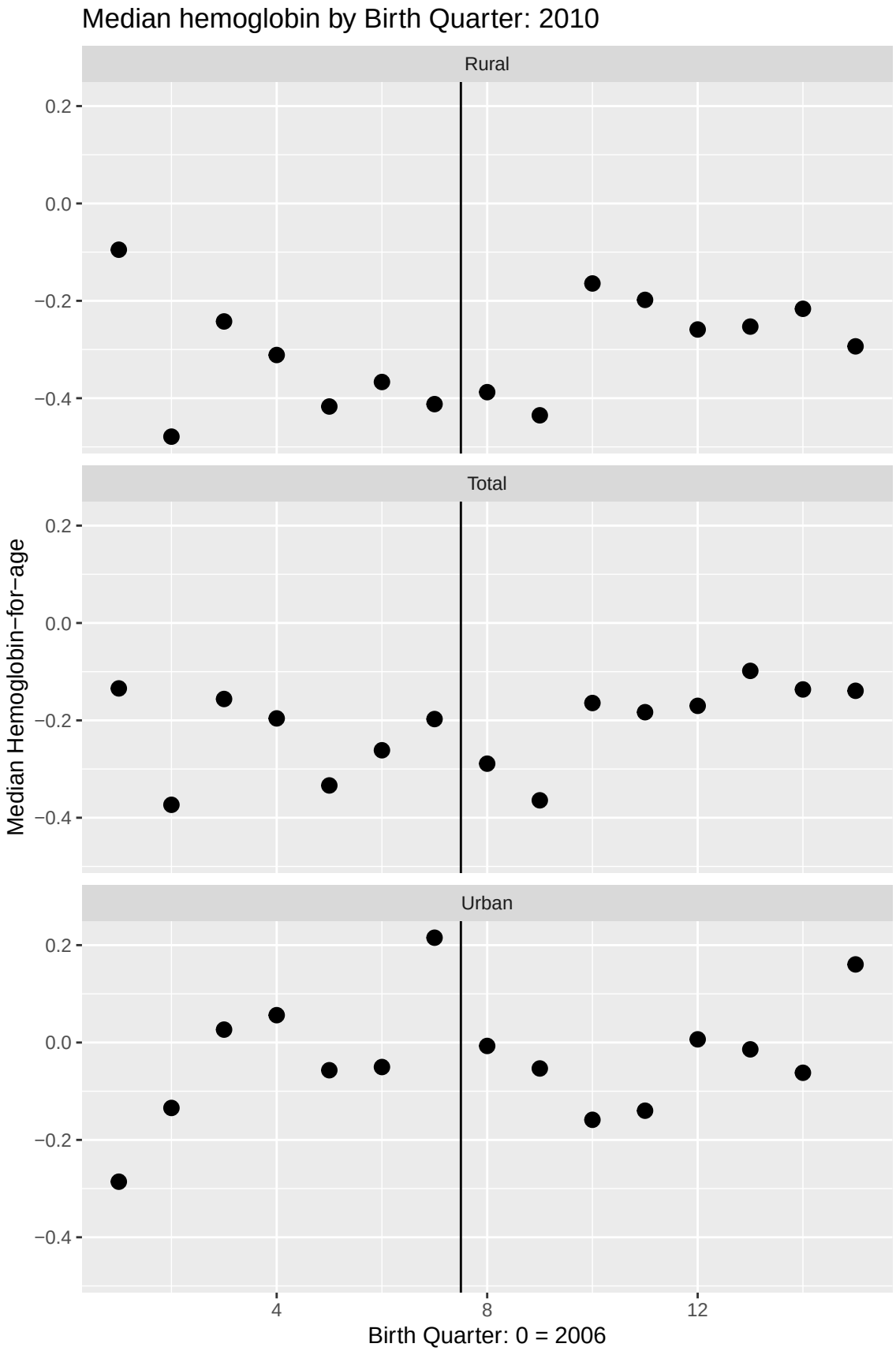

Figure 9: Hemoglobin-for-age in 2010 by Birth Quarter

Next I allow the effect of exposure to depend on these wealth and distance-to-Dakar, for reasons explained in sections 2.1-2.2. We see that there is now a strong, negative effect of exposure on the urban-poor. The small result above can be interpreted as the average of effects which are heterogeneous across wealth and location. 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 urban results are attenuated, while the 2012 rural cohort shows a significant, positive effect of 2008 crisis..

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	0.2215	-0.4824^{***}	0.0106	-0.3154
	(0.2200)	(0.1343)	(0.3404)	(0.2484)
Exposure	-1.5196^{**}	0.2737	-0.3105	0.8541^{*}
	(0.5523)	(0.2820)	(0.6073)	(0.4494)
Distance to Dakar	-0.0568	0.0522	0.0707	0.0944^{*}
	(0.0396)	(0.0375)	(0.0671)	(0.0479)
Wealth	1.0099	2.3263^{**}	0.5930	1.4001
	(1.3065)	(0.7347)	(2.0272)	(1.0284)
Distance to Dakar * Exposure	0.2574^{**}	-0.0299	-0.0020	-0.1905^{**}
	(0.0865)	(0.0726)	(0.1152)	(0.0786)
Wealth * Exposure	4.0122	0.0928	1.9335	2.1867
	(2.6916)	(2.0007)	(3.0848)	(1.8871)
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 5: Regression Results by Survey and Urban-Rural

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. I multiply this value by $\frac{8}{9}$, to capture the predicted effect of living through the entire crisis. I multiply the predicted effect size by the standard deviation of hemoglobin for the child's age, in order to depict effects in terms of hemoglobin levels.

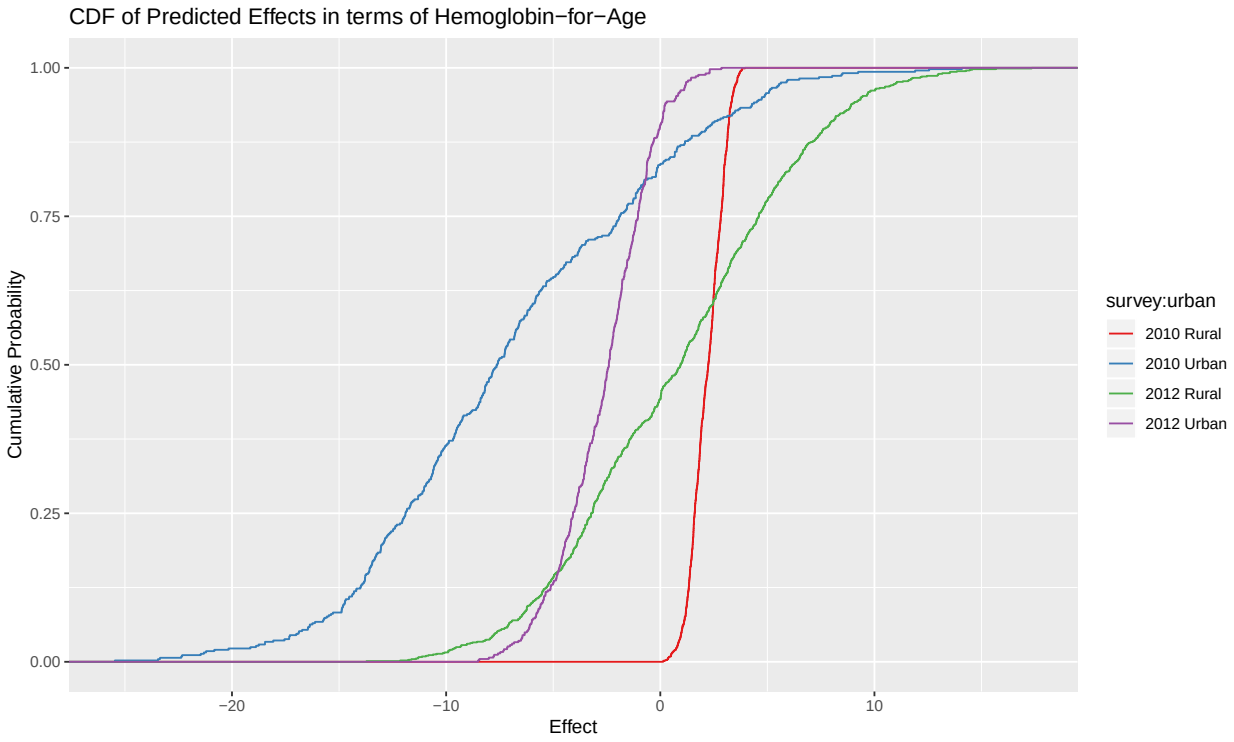

Figure 10: Distribution of Effects

From this figure, we can see that the median effect size for a child from an urban *commune* observed in 2010 is around 8 g/L of hemoglobin, which is more than an 8% change in percentage terms. The median effects for the 2010 and 2012 rural and the 2012 urban samples are close to zero, ranging from -2.5 to 2.5.

6 Robustness

In the analysis above, exposure is defined by age. Given that child health may also vary with age, we ought to be concerned that my results are generated simply by the exposed group's position in the age distribution. To check for such a result, I run a triple-difference in difference. I compare the exposed group in the 2010 survey with children of the same age in the excluded surveys of 2005, 2014, 2015 and 2017. The first difference is between the exposed group (age-defined for all surveys) and the control. The second difference is between the 2010 survey and the excluded surveys. The third difference is between urban and rural groups. Exposure should only matter for the 2010 survey urban-dwellers, thus the coefficient of interest is the triple interaction term. We see below that it is negative, suggesting that the position of the exposed cohort within the age distribution is not driving my results. Moreover, the magnitude of the coefficient is comparable to that of my results. As I have not included wealth or distance-to-Dakar, the -0.38 coefficient below represents the average of heterogeneous responses across these dimensions.

	Difference-in-Difference
Intercept	−0.0081
	(0.0228)
2010	−0.3333***
	(0.0611)
Exposure	−0.0676
	(0.0522)
Urban	0.2383***
	(0.0300)
2010*Exposure	0.0713
	(0.1313)
2010*Urban	0.1925
	(0.1022)
Exposure*Urban	0.0972
	(0.0732)
2010*Exposure*Urban	−0.3883
	(0.2202)
Num. obs.	15954
Percentile	0.5000

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

Table 6: Placebo Test. Median-regression comparing the psuedo-exposed cohorts in the excluded (2005,2014-17) surveys with the truly-exposed cohorts of the 2010 survey.

7 Discussion

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; and,
3. 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.

Among urban-dwellers, 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.

One possibility, however, is that the attenuation I observe is due to measurement error, rather than to catch-up. For the population as a whole, the effect of the 2008 crisis is larger for those observed in 2012 than in 2010. The difference is that the heterogeneity is more pronounced in the 2010 results. Observables like

wealth can change over time, making wealth in 2010 a better proxy than wealth in 2012 for wealth in 2008. This increased measurement error over time could explain the attenuated results for 2012.

Among rural-dwellers, we see the opposite result, as the observed effect of exposure in 2010 is smaller than in 2012. This is misleading, because the estimates on the interaction terms have also changed. From figure 12, we see that the median effect in 2010 is actually larger than in 2012. Simple decomposition of effect by region (figure 16 in Appendix 2) shows that the 2012 rural effect is being driven by Tambacoumba and Kedougou– the two most isolated parts of Senegal. One could construct a narrative based on this result, but I will refrain. Lacking further evidence or a compelling narrative, I do not place much weight on the 2012 rural results. Instead, I take this result as indicating the general noisiness of my results. Therefore, I call my results "suggestive."

With regards to interpretation of exposure, I do not wish to overstate my results. Although exposure was defined with respect to prices, prices *themselves* do not enter into any of the regressions. 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. The estimated effect of the crisis may have 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.

In terms of regional variation, Dakar stands out as driving the results above. 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. The rest of the regional birth-quarter plots can be found in Appendix 3.

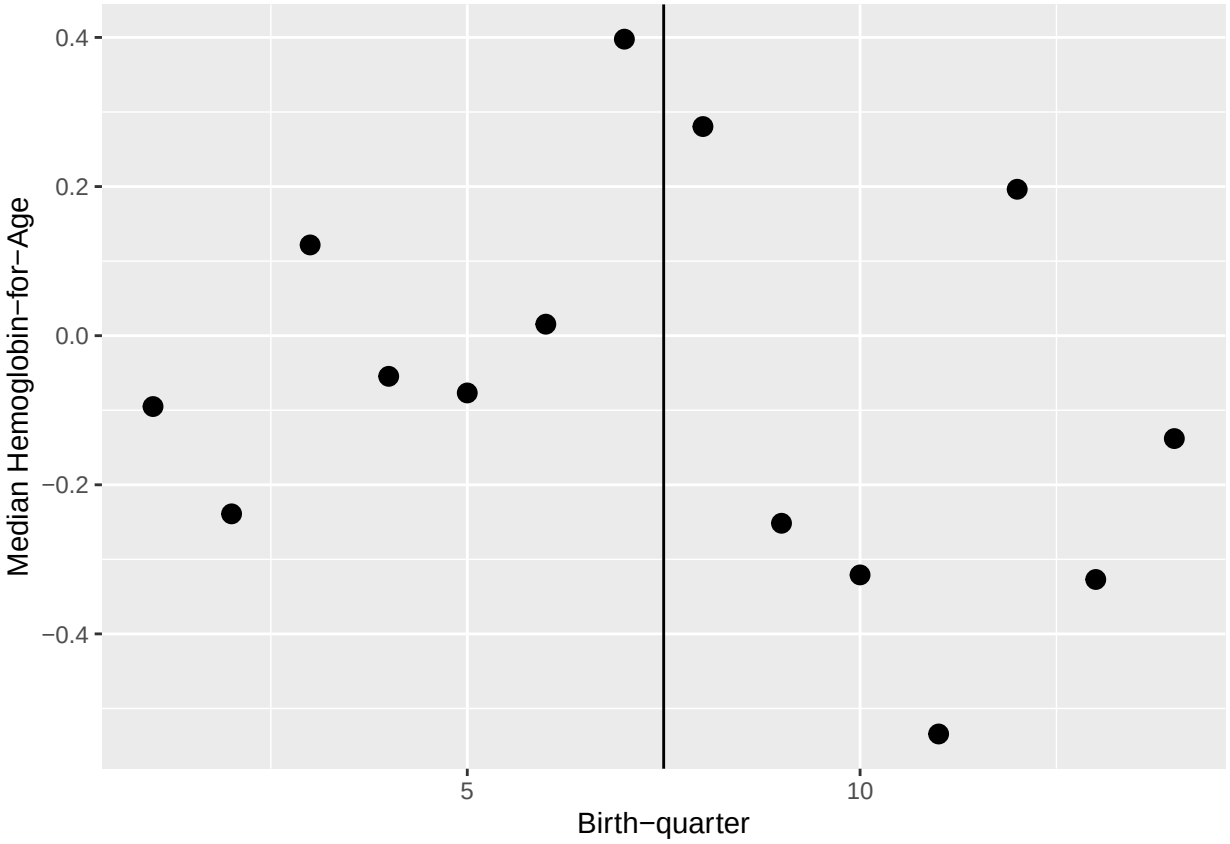

Figure 11: Median Hemoglobin-for-age in 2010 by birth-quarter in Dakar

This result suggests that distance to Dakar might be less important than the qualities of Dakar itself. Dakar is a large port with a unique relationship to the world market relative to other cities of Senegal. It is not surprising that the effect of an international crisis was felt most strongly in Dakar. The effect of the 2008 crisis also appears to be delayed. Several explanations of this lag come to mind. One, the first exposed cohort only experienced 1-3 months *in-utero* during the crisis. Later cohorts were exposed for 8 months. Two, as seen in figure 5, the transmission of world prices to local prices in Dakar takes several months. For both these reasons, it is not surprising that we observe above a lag in the effect of the crisis. Three, given that both credit constraints and hemoglobin are determined by stock variables (see section 2.2-2.3), we would expect lagged effects.

All of this discussion and analysis has been *specific to Senegal*. One should expect different effects of the 2008 crisis in a different setting. As shown above, millet production 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 shows the value of 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 relative to relying fully on imported food. 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 results provides some support for the "food-sovereignty" perspective (Rosset 2008).

8 Conclusion

I find suggestive evidence that the 2008 world food crisis negatively affected the health of women 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, while having a small, positive effect on rural households. 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.

My results also suggest that 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 at age 2, but has dissipated by age 4. 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. I hope other scholars will note the potential for child hemoglobin, widely measured in the DHS survey, as a potential indicator of maternal health with a clear identification strategy.

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 vulnerability and malnutrition of poor women in Senegal.

9 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. Both the distance from a particular community to Dakar and whether that community is urban are 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, in order to examine how each of these underlying variables mediated the effect of the 2008 crisis.

Another, complementary route would be to expand my analysis to include Senegal's neighbors 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 results to data on cognitive development, health and labor-market outcomes. Children *in-utero* during the 2008 crisis had significantly lower hemoglobin levels in 2010, indicating a general iron-deficiency early in life. Biomedical research has identified iron as a critical nutrient in development, suggesting the 2008 crisis may have had large, long-term effects on the urban-poor.

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11 Appendix 1: Context about Senegal

11.1 Summary Statistics

Survey	Urban	Wealth	Land	Hem-for-age	Dist-Dakar	Wealth	Land	Hem-for-age	Dist-Dakar
2010	0	-0.42	0.82	-0.36	3.54	0.61	0.15	1.14	3.04
2010	1	0.81	0.24	-0.06	2.73	0.61	0.18	0.96	4.31
2012	0	-0.51	0.85	-0.21	3.48	0.46	0.13	1.12	3.33
2012	1	0.62	0.29	0.09	2.92	0.66	0.20	0.90	3.95
		Mean	Mean	Mean	Mean	Variance	Variance	Variance	Variance

Note that I have scaled the wealth index by a factor of 10, in order to make it easier to read from the table.

11.2 Geographic Context

Here I present the FEW's livelihood map, which provides context on the various regions of Senegal and their agricultural production.

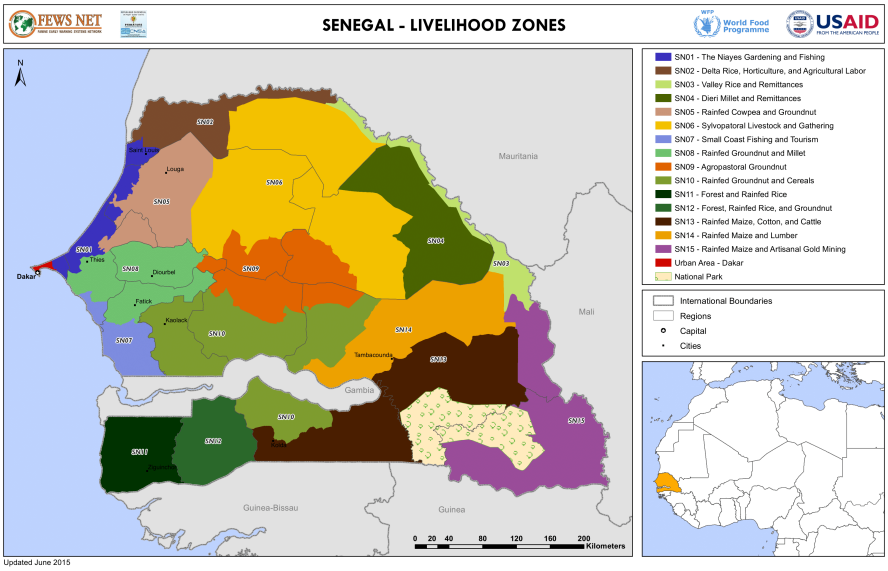

Figure 12: FEWs Livelihood Maps

11.3 Identification: Rainfall Anomaly in Senegal

Below I plot the rainfall anomaly for the Sahel region from 1950-2017. We see that 2007-08 was unremarkable, and I find no evidence for coincidental factors upsetting my identification strategy. We have already seen in the introduction that domestic food production was relatively high in 2008.

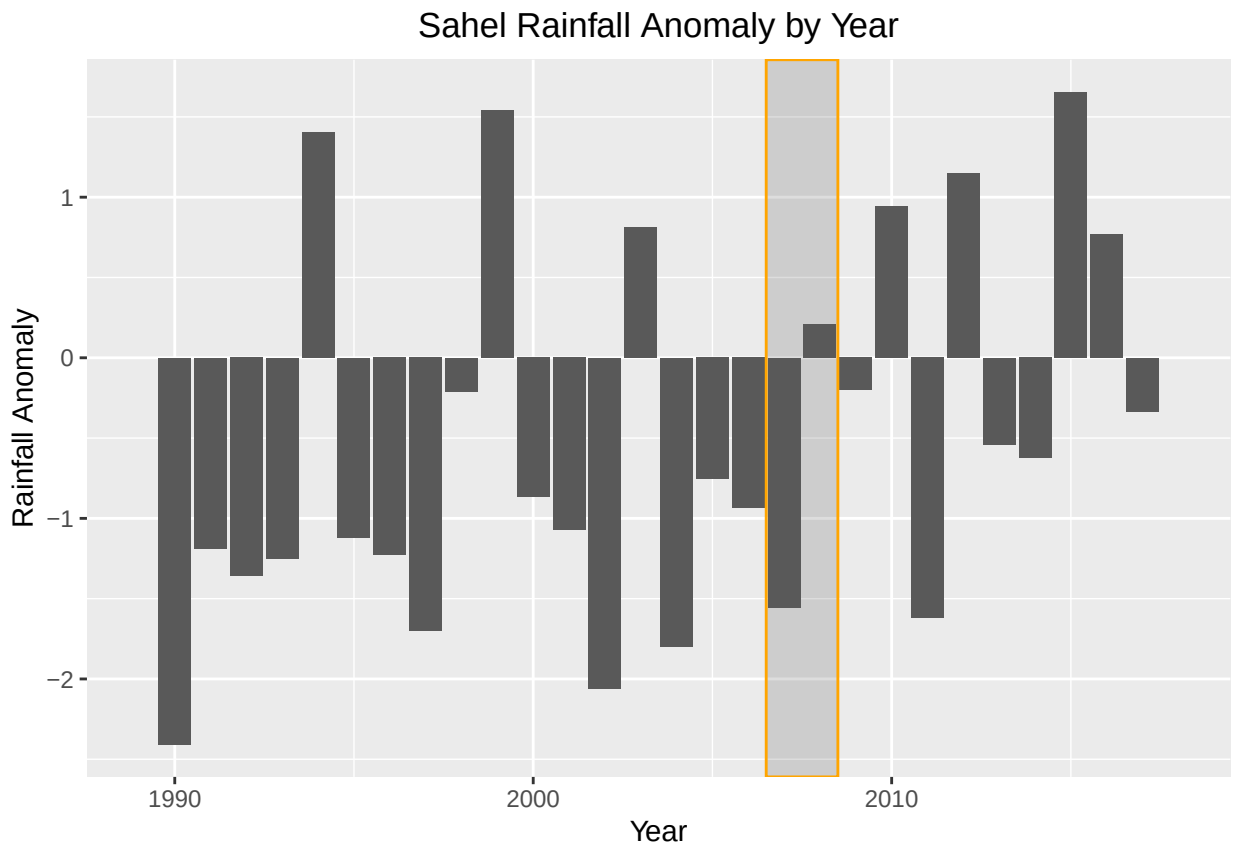

Figure 13: Searching for coincidence

Of course, this is just one test, and I can never *prove* that the identification assumption holds. I can only subject my assumption to as many falsification tests as possible.

12 Appendix 2: Alternate Specifications

Here I run several alternative specification to those reported above. They generally support my conclusions.

12.1 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.

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.

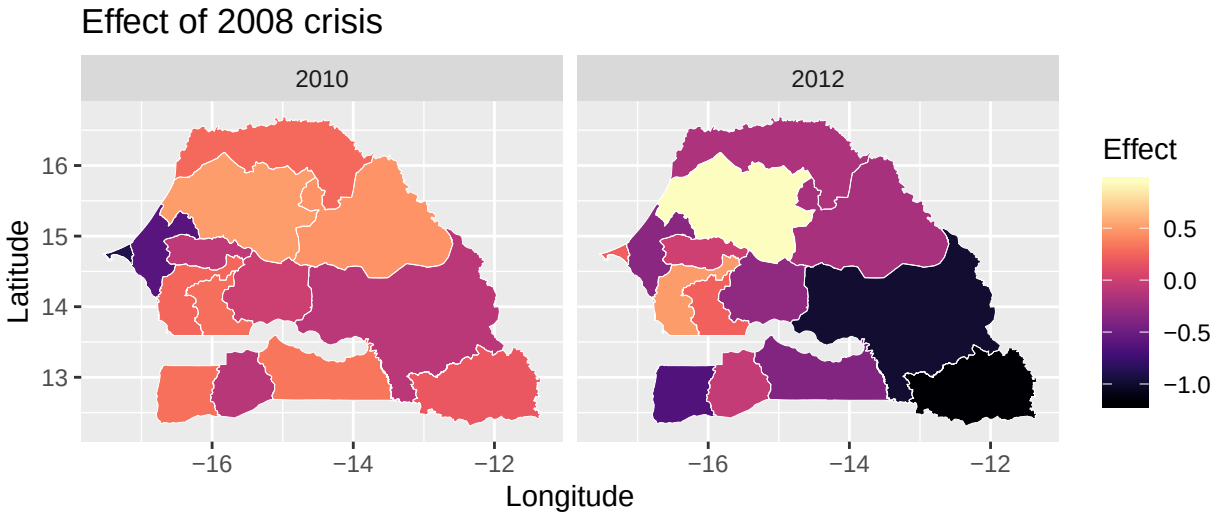

Figure 14: Effect of the 2008 crisis across Senegal by survey-year

12.2 A Test for Lagged Effects

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.

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	0.0210	−0.3794***	0.2987	−0.2626
	(0.1240)	(0.0916)	(0.1967)	(0.1721)
Exposure	−0.1926	0.1566	−0.2772	0.5054
	(0.3695)	(0.2285)	(0.3630)	(0.3154)
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: Test for Lagged Effects

	2010: Urban	2010: Rural	2012: Urban	2012: Rural
Intercept	−0.0862	−0.5352***	0.1400	−0.3880*
	(0.1928)	(0.1368)	(0.2706)	(0.2010)
Exposure	−0.5978	0.4047	−0.5678	0.9791**
	(0.5477)	(0.3089)	(0.4681)	(0.3443)
Distance to Dakar	−0.0247	0.0554	0.0454	0.0947*
	(0.0366)	(0.0326)	(0.0574)	(0.0432)
Wealth	2.6090*	2.4189**	0.6578	2.0140*
	(1.2047)	(0.7410)	(1.7776)	(1.0369)
Distance to Dakar * Exposure	0.1386	−0.0397	0.0432	−0.1990**
	(0.0960)	(0.0658)	(0.0945)	(0.0764)
Wealth * Exposure	−1.7403	−0.3079	1.6211	0.3323
	(2.6574)	(1.8236)	(2.8833)	(1.9677)
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 9: Test for Lagged Effects

12.3 Test for Selection

Next I test for fetal selection. Female fetuses are more robust than males, so if the 2008 crisis caused selective mortality, 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 10: Test for Selection

12.4 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	0.2462	-0.5187^{***}	0.1224	-0.3286
	(0.2411)	(0.1519)	(0.3605)	(0.2638)
Exposure	-1.5099^{*}	0.4504	-0.4412	0.9013
	(0.6216)	(0.3259)	(0.7657)	(0.5294)
Distance to Dakar	-0.0580	0.0545	0.0585	0.0913^{*}
	(0.0433)	(0.0393)	(0.0666)	(0.0478)
Wealth	1.2474	2.4422^{***}	0.0502	1.0803
	(1.3296)	(0.7250)	(1.7366)	(1.0345)
Distance to Dakar * Exposure	0.2705^{**}	-0.0438	0.0151	-0.1867^{*}
	(0.0915)	(0.0740)	(0.1121)	(0.0768)
Wealth * Exposure	3.9276	-0.3895	2.3134	1.4936
	(2.5898)	(1.8491)	(2.9350)	(1.8269)
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 11: Seasonal Fixed Effects

13 Appendix 3 Hemoglobin by Region by Birth-quarter

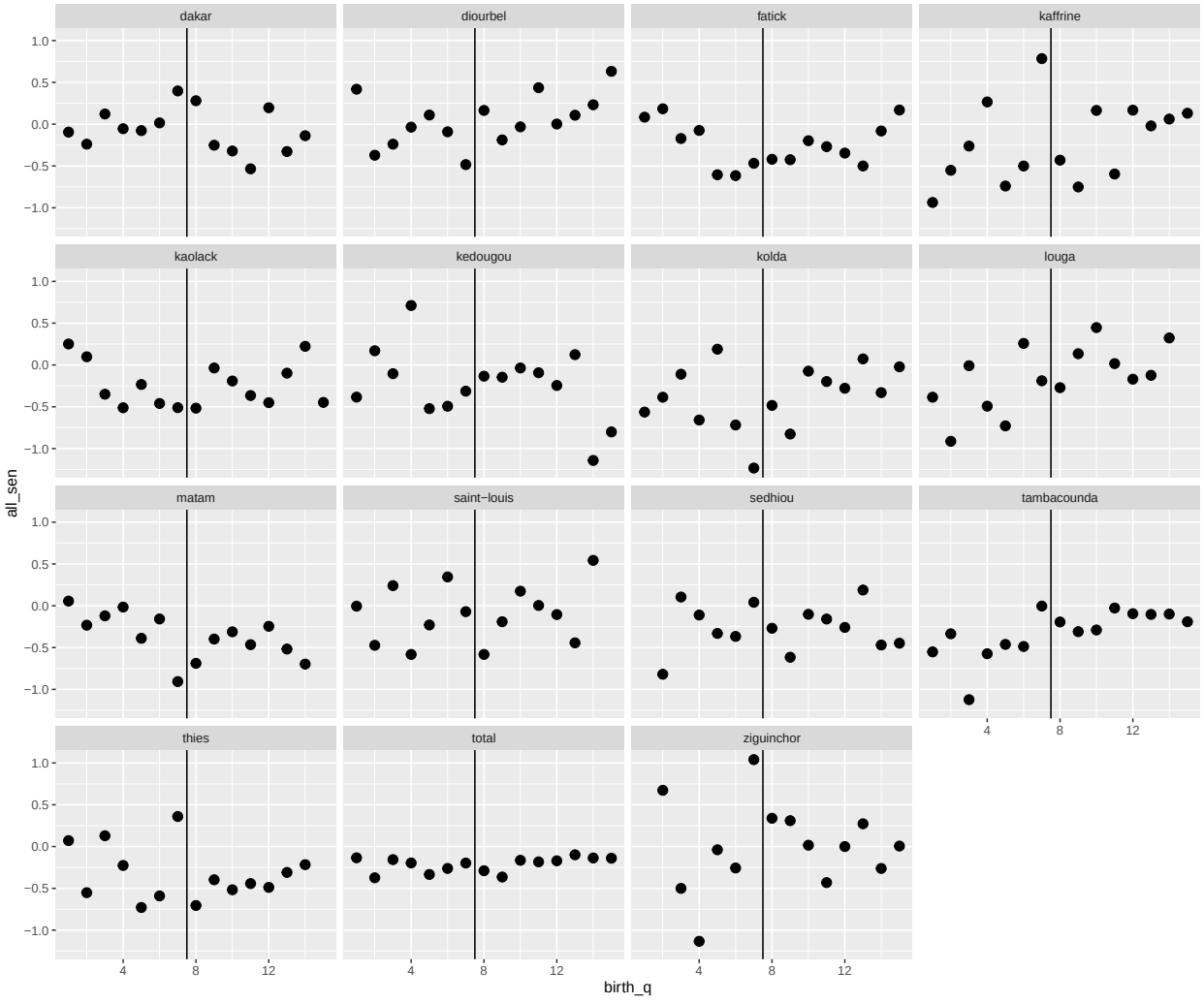

Figure 15:

Here I plot the same results as in figure 13, but for each region of Senegal. We see that Dakar is the only region where the effect of the 2008 crisis is evident to the eye.