Anemia in Senegal: The 2008 World Food Price Crisis

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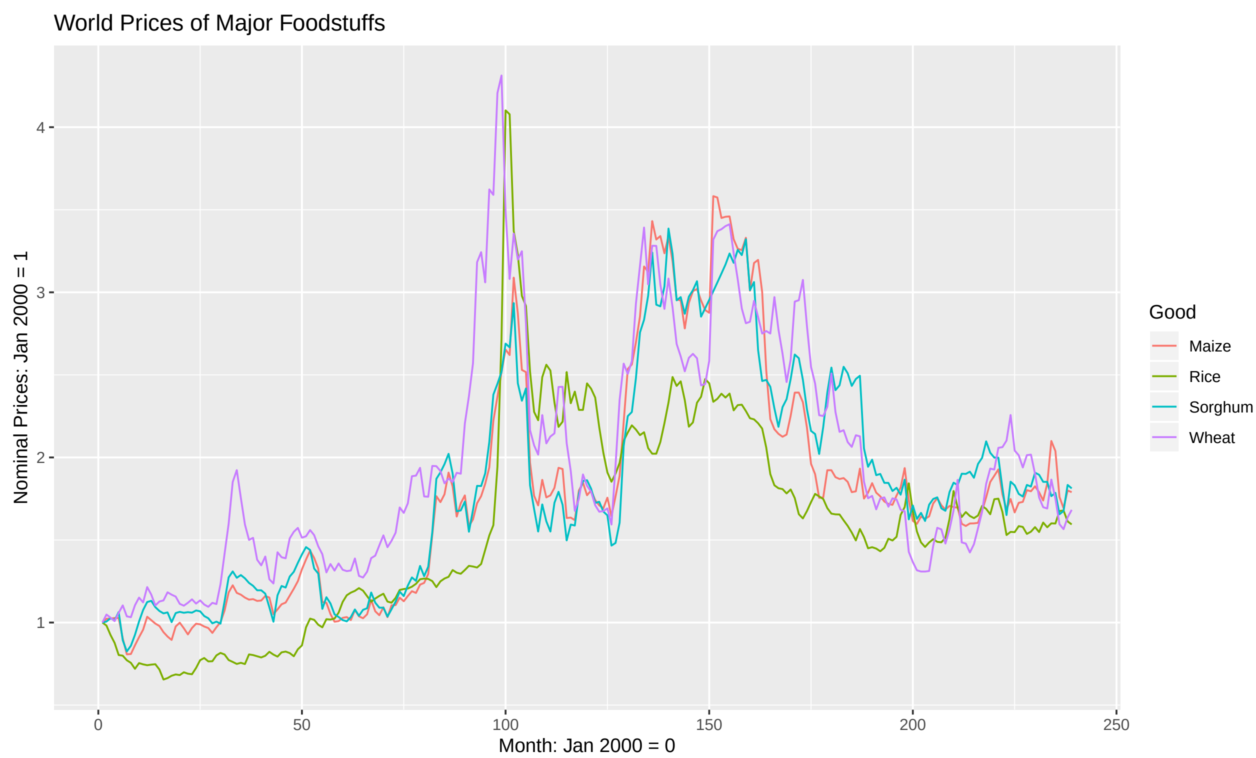
Summary

I will explore the effect of the 2008 world food price crisis on the health of Senegalese children who were in-utero at the time. I predict that the 2008 crisis had a negative impact on fetal health, and that its effect was diminishing with distance from Dakar, the port and economic hub of Senegal. Conceptually, I will compare the hemoglobin of children born just before or conceived just after the crisis, to those who were exposed in-utero.

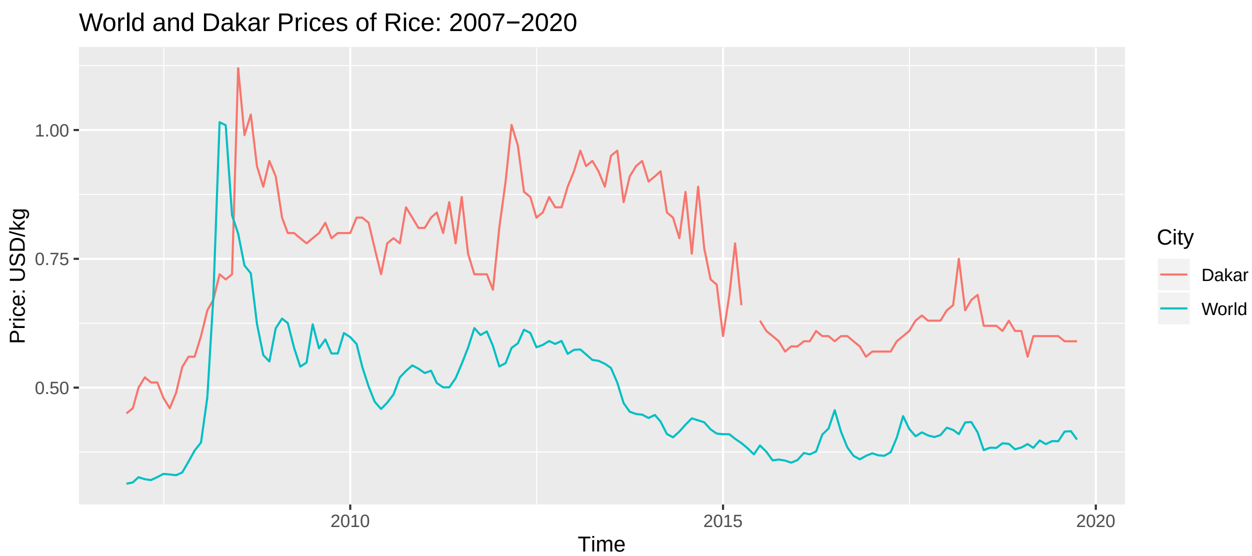
While previous work has explored the effect of the 2008 crisis on child health, mine will be the first to use a fetal origins approach, which allows data collected several years after the crisis to be used in analysis. With this increased variation, I will test the hypothesis that the crisis had a stronger effect in areas more closely connected to the world market (e.g. Dakar) than in more isolated areas.

Introduction

After an extended period of low and stable food prices, in early 2008 world food prices increased dramatically. The causes are debated, but include American agriculture policy as well as financial speculation (Rosset 2008). The prices of both rice and wheat doubled in fewer than 6 months, while the prices of other staples increased as well. This increase lead to riots and unrest in low to medium-income countries around the world, including Senegal.



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.



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

As emphasized by Sen (1980), food security depends on the combination of level and volatility of food entitlements. From the simplest model of comparative advantage, we know that trade will induce specialization. This specialization will increase average incomes, but the effect of specialization and trade on volatility is unresolved (Giovanni and Levchenko 2009; Burgess and Donaldson 2010).

Existing studies in economics explore the effect of the 2008 crisis on health and welfare (Arndt et al. 2016; Dimova and Gbakou 2013a). Dimova et. al. estimate welfare elasticity with respect to price in Cote d'Ivoire during the crisis and argue that the majority of rural dwellers actually benefited from the shock, while urban dwellers suffered. Arndt et. al. take a more similar approach to mine, looking at the effect of high food prices on child weight-for-height. The authors find that weight-for-height was negatively correlated with food price increases, but study is limited by its dataset, which relies on direct observation during and after the crisis.

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 disentangle the effect of the crisis 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, unlike weight-for-height. 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.

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 econometric evidence towards the discussion being held in adjacent fields about market integration and food sovereignty. Previous work has emphasized a rural-urban divide, with the urban poor suffering from increased food prices, and the rural poor minimally affected (Dimova and Gbakou 2013b; Barrett 2010; Ruel et al. 2009). While it is true that rural-dwellers are more likely to be net food-sellers, this hypothesis overlooks the broad class of rural poor who are net food-consumers. I will be able to test both this rural-urban hypothesis, and my own distance hypothesis.

[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).

The Economy of Senegal: Food and Trade

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. 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. See the FEWS Livelihood Zone Map below:

Although agriculture is the primary means of livelihood for 69% of the population, 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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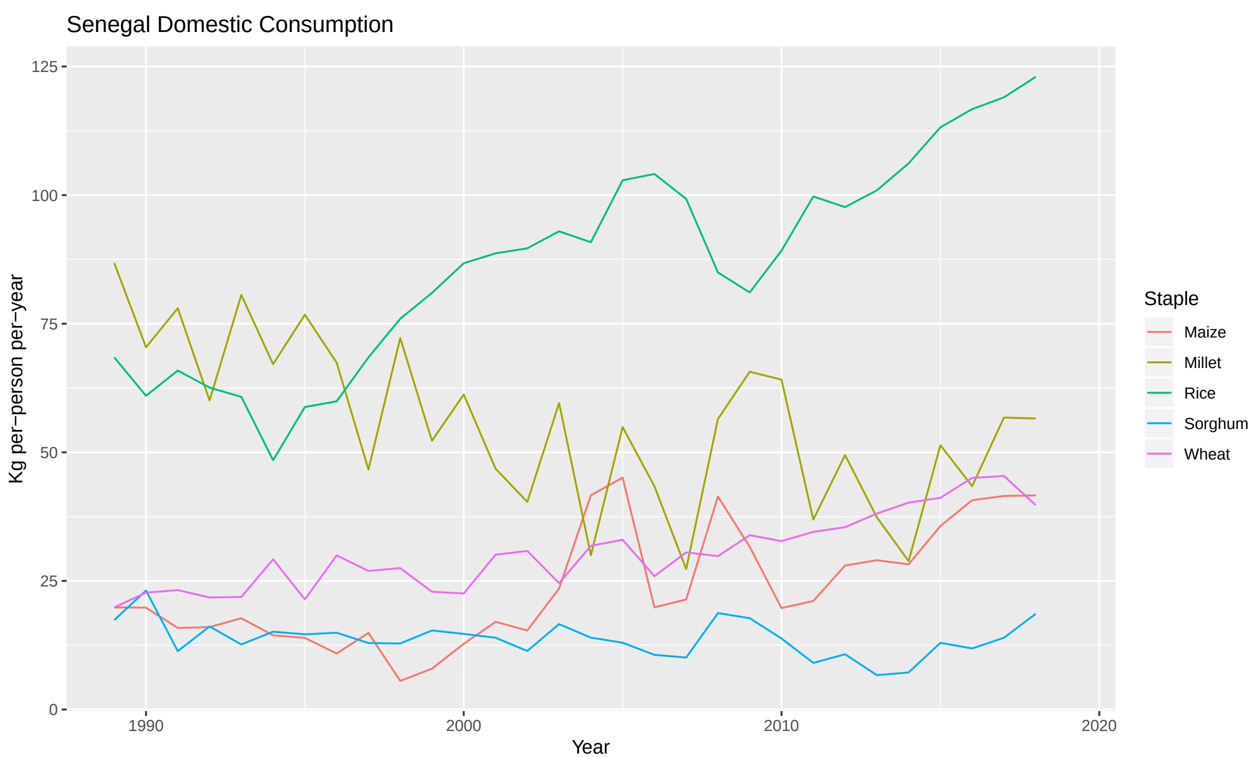


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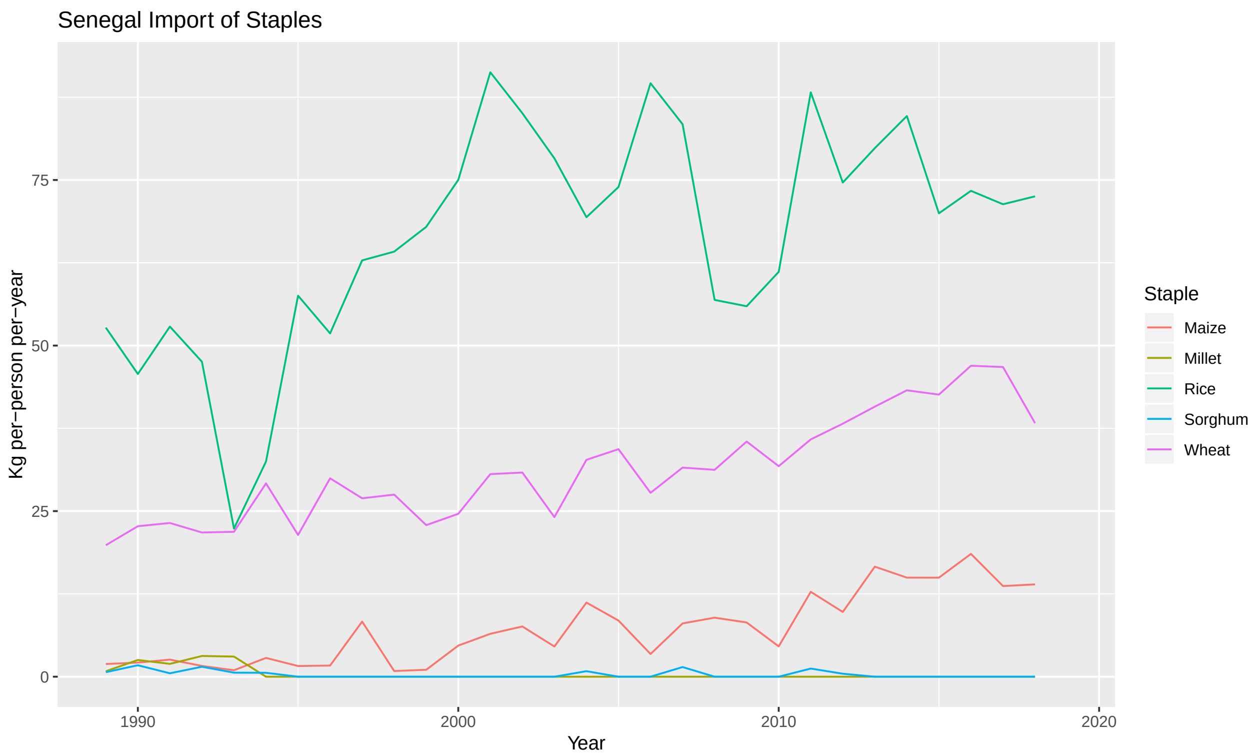


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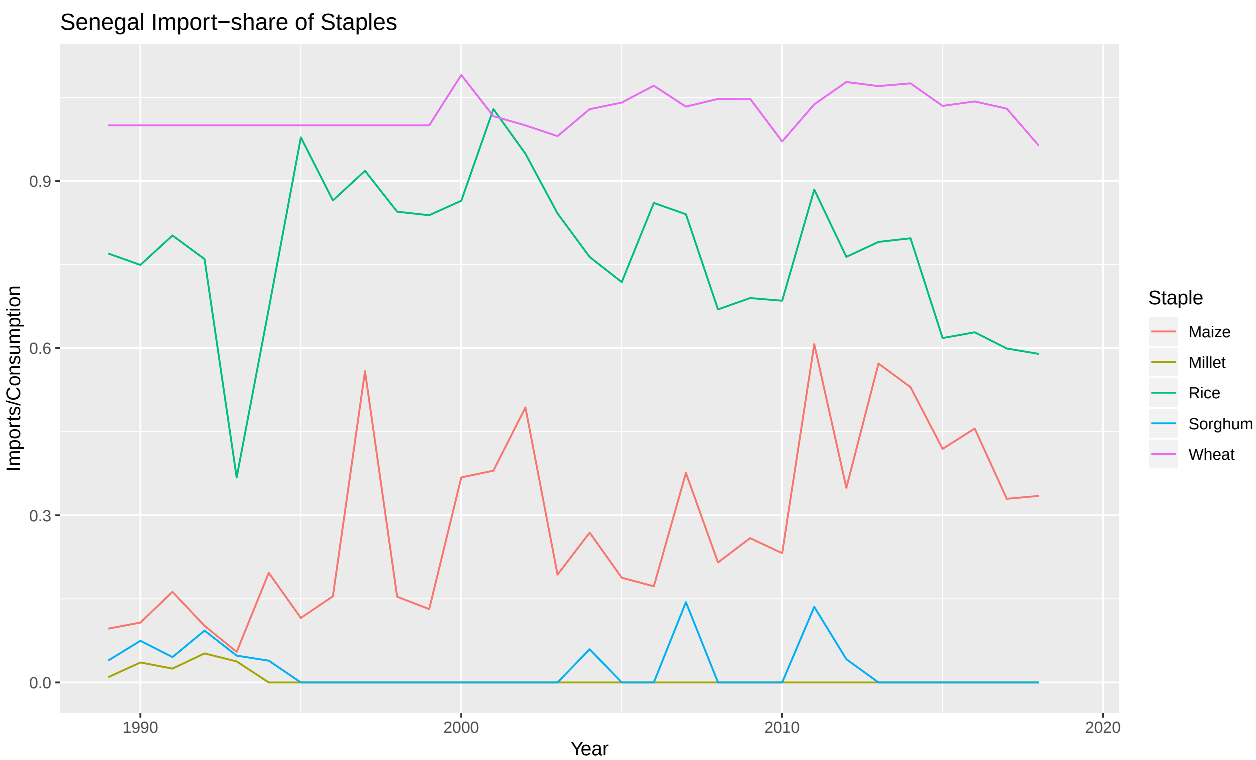
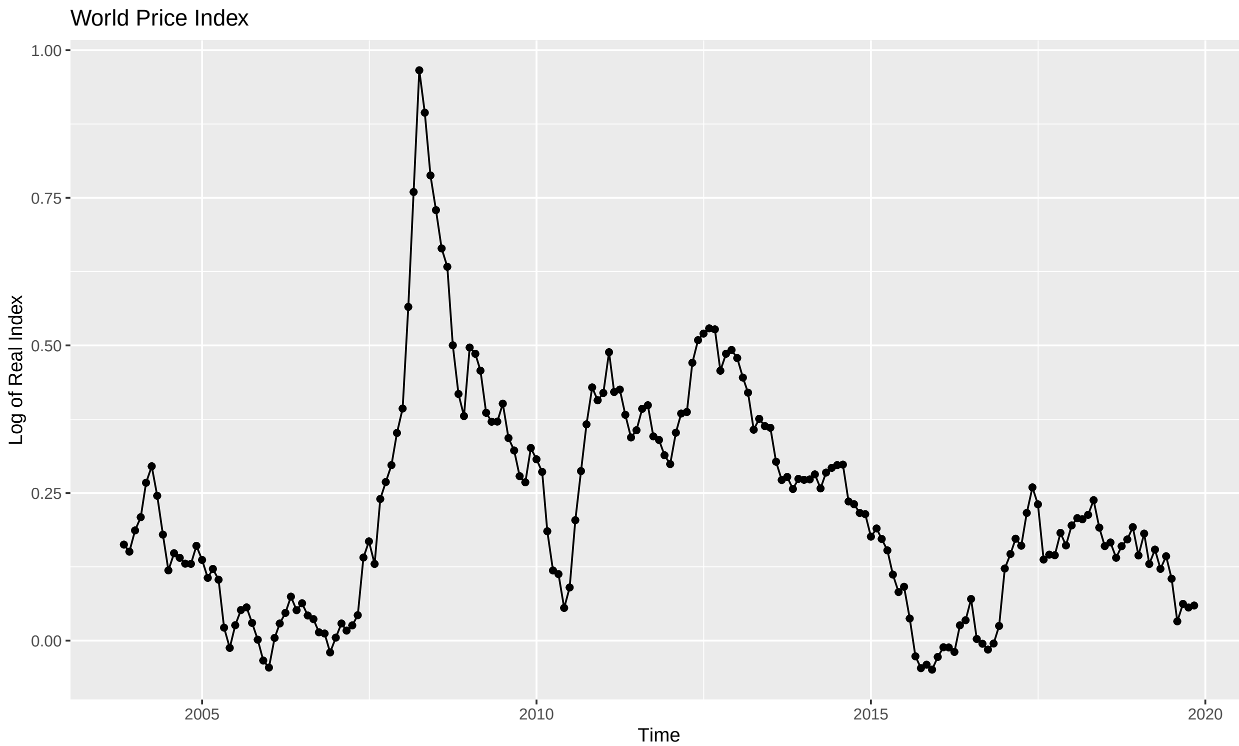


We see that in 2008, when prices increased dramatically, consumption of millet and sorghum increased while that of rice decreased. This might reflect some substitution away from high-priced, imported goods. It would indicate some reserve capacity for local food production.

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.



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 simple model. P_a is the price in autarky and is determined via local supply and demand. 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.

[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.

Greater food-producing capacity would shift the local supply curve outward, making the world price less likely to bind.

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 will 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.

Iron and Anemia in Mother and Child

Anemia is characterized by low levels of hemoglobin in the blood and can be caused by deficiencies in iron, folate and other micro-nutrients. By latest estimates, 1.2 billion people around the world suffer from iron deficiency anemia. Iron-deficiency anemia causes fatigue and impairs cognitive and motor development. Anemia is a health concern across the income spectrum, but is more prevalent among the poor. Iron availability depends on food intake, both quantity and quality. Absorption of heme-iron (animal-based) is greater than absorption of plant-based iron. In a healthy person, there are iron-stores about equal to the amount of iron in use in the person's blood. There is a small, constant loss of iron. The bioavailability of iron for a person is a stock, while iron intake is a flow, meaning that past iron consumption will affect current availability (Camaschella 2015).

Iron is important for both maternal and fetal health. For the mother, anemia makes complications (and death) from birth more likely. Iron is passed from the mother to fetus, allowing the development of fetal hemoglobin, which differs from adult hemoglobin. Over the first four months, the infant breaks down the fetal hemoglobin and synthesizes adult hemoglobin. Little iron is transmitted through breast-milk, so the fetus must develop sufficient stores of iron in order to make the transition from fetal to adult hemoglobin as they grow and their blood volume increases. The estimated iron requirement for pregnancy is 1,000 mg–nearly half of the total iron store in a healthy, non-pregnant woman (Lactation 1990).

Infants, on the other hand, need relatively little iron, given that they developed 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),

[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.

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, increasing her stores. Thus, iron-stores in infants should be relatively insensitive to maternal health during the first six months of life.[4]

Maternal anemia then has a direct effect on infant anemia, as the fetus cannot make adequate stores to last the breast-feeding period. Unless remedied through increased iron consumption, anemia will remain as the infant grows into a child. During menarche, iron requirements increase for girls, and many become anemic. Thus an anemic mother begets anemic child who becomes an anemic woman 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.

Data

The DHS measures the hemoglobin of infants and children from age 6 months to 5 years. There are two DHS surveys of Senegal, 2010 and 2012, which include children in-utero during the 2008 crisis. The DHS data includes location and households characteristics.

At birth, the infant has a large amount of fetal hemoglobin which must be broken down and replaced by adult hemoglobin. In general, hemoglobin levels decrease from birth until 6 months to 2 years, then they begin to increase again. We see such a pattern in the DHS data, which I plot below. The yellow window indicates the 2008 crisis.

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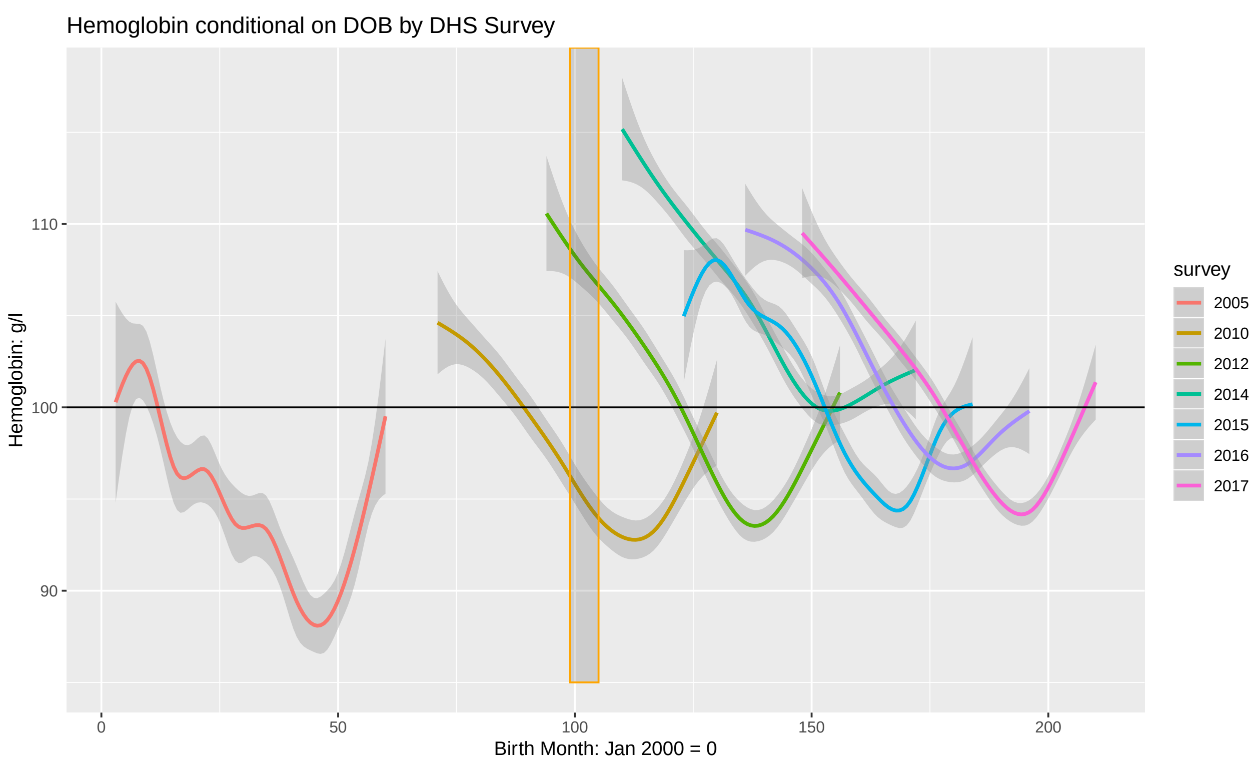


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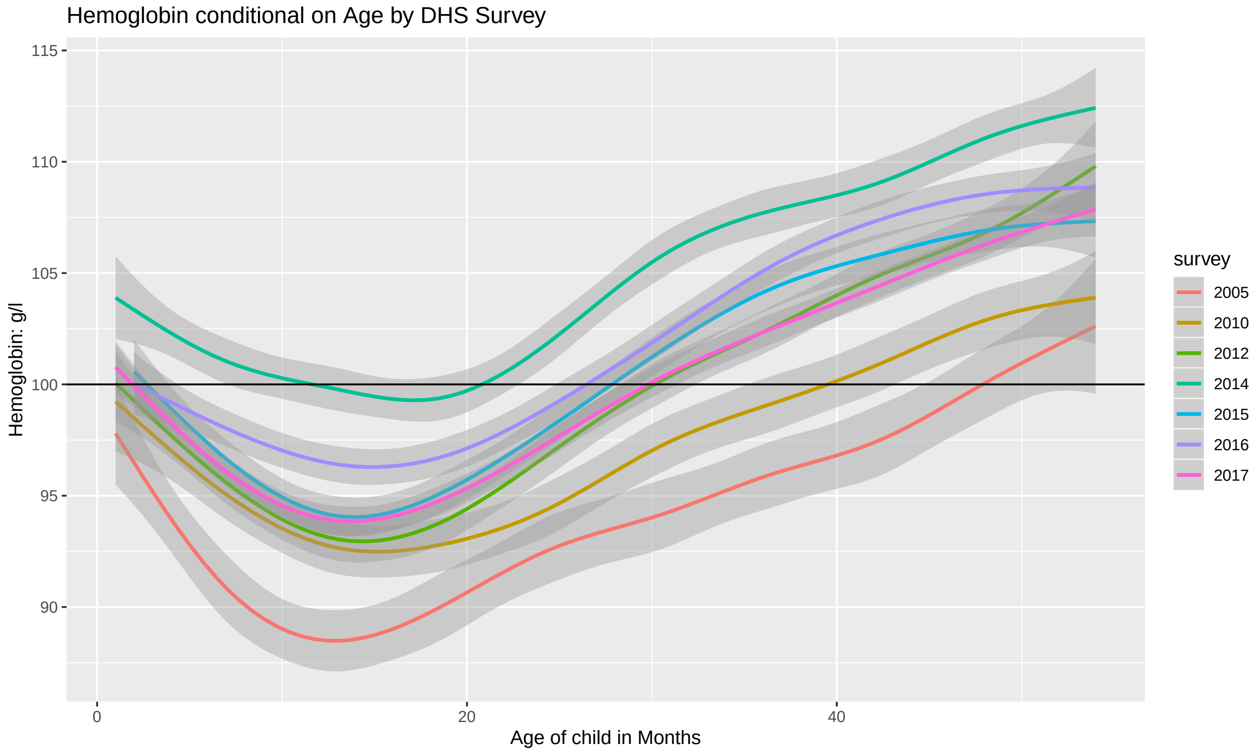
[4]Note that this all relies on women breastfeeding their children. In Senegal, breastfeeding is overwhelmingly common during the first six months, with very little cessation before the child reaches a year old (Mané et al. 2006).



Anemia is relatively rare in children, compared to in infants. Anemia resurges in girls upon menarche. The cutoff for anemia in adults, 100 grams per liter of blood is shown as a black, horizontal line.

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.

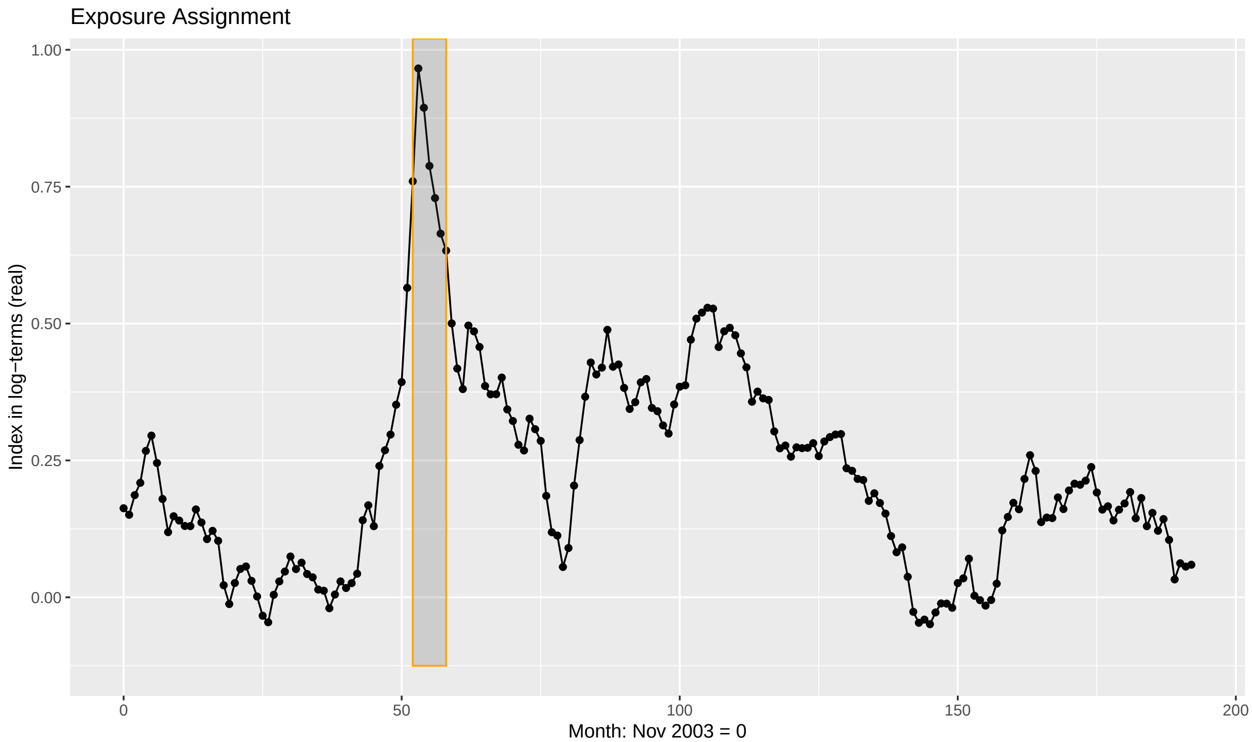
Methodology

One potential route would be to regress child hemoglobin on world prices during gestation. This approach is complicated by the fact that prices during gestation are a distribution, not a value. The relationship between monthly price and hemoglobin will likely be non-linear, as households are able to smooth small price changes, but not large increases. We could capture this non-linear relationship using a "bin" approach. First we would discretize the problem, breaking up price into n bins. Then we would count the number of months each child spent in each price-bin over the course of gestation.

However, this approach assumes that the timing of price is irrelevant. Given that different stages of pregnancy have very different iron requirements, the timing of price, not only the level each month, is important. Second, because of credit-constraints, a month of high prices will be harder on a household if it falls after another month of high prices than after a month of low prices. Third, because prices affect iron intake, a flow, but hemoglobin is determined by iron stocks, we would expect persistent effects over time.

Because of this complexity, and the fact that the 2008 crisis dominates variation over the period of study, I will instead concentrate on a single period of unusually high prices. I will construct the proper control group based on knowledge about the biological process of hemoglobin formation. Doing this will also allow me to speak directly to a substantial literature which is interested in effects of the 2008 crisis.

The 2008 crisis forms a contiguous 7-month period in which the price index is greater than at any other point in the 20 year span. I will take all those in-utero during this period, March and September 2008, to be exposed.[5] The degree of exposure will be the sum of months in-utero during the crisis period, weighted by the price index. For anyone born before March 2008, or conceived after September 2008, the in-utero exposure will be zero. For someone born in November 2008, the exposure measure would be 1, as they were in-utero for the entirety of the crisis period.



While world prices are exogenous to the economy of Senegal, they are a time series, meaning that they could be confounded with time trends. For example, if we simply ran a regression on the whole period, with a dummy for exposure to the 2008 crisis, the dummy would surely be negative, simply because anemia has decreased over the course of the period, as Senegal has gotten richer. I see two paths around the time problem:

1. The control approach. Add a polynomial (or some other control) for time, in order to account for the trend. In this case, I would use the full dataset, including children born from 2001 to 2016. Hypothetically, I could control for a whole host of factors, including weather, season and even cluster or household-fixed effects, though none of these should be correlated with shock exposure.
2. The zoom-in approach. Discard most of the data, and focus on those born just before the shock, conceived just after the shock, and exposed to the shock. By narrowing in on a particular window, the time trend should be minimized.[6]

Variables

Drawing on the sections above, I will use several variables for my analysis:

[5] February 2008 might also be considered part of the crisis, but I allow one month for world prices to transmit to Senegal.

[6] This would be similar to the approach of Arndt et. al. (2016). They compare different quarters of the 2008-2009 period with propensity matching. I see no reason to do propensity matching with my data, as the confounding factor of interest is time. Other confounders should be orthogonal to date of birth within a given survey.

- Hemoglobin-for-age: the hemoglobin of a child minus the mean and divided by the standard deviation hemoglobin for the age of the child;
- Wealth: an index, calculated by the DHS as a combination of goods which the household owns; and,
- Distance to Dakar: the driving distance from each DHS cluster to Dakar, calculated via the Open Street Maps API.

The effect of distance will be flexible for the Casamance region, which although physically close to Dakar, is separated by The Gambia.

Hypotheses

I propose that there will be a negative relationship between in-utero exposure to the 2008 food price spike and child hemoglobin in 2010 and 2012. The relationship of world price and hemoglobin will be mediated by:

- Distance to Dakar (protective); and,
- Wealth (protective)

Discussion

Because of the fetal origins approach, where we implicitly compare those in-utero and those already born, we can safely infer that any estimated effect reflects a change in in-utero health conditions. However, the crisis was more than just an increase in prices. 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*.

Similarly, distance-to-Dakar should be considered as a general proxy for integration to the world market. Distance will capture a broad set of socio-economic conditions. We would expect areas further from Dakar to be 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. In order to better understand the results, I will explore the relationship of distance-to-Dakar with other covariates.

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