Measuring the Effect of the 2008 World Food Price Crisis

Nutritional Outcomes from Senegal and Cambodia

Jesse McDevitt-Irwin

23 December, 2020

A note on the text

This essay is part of a larger project, the goal of which is to:

1. Identify and measure the impact of the 2008 crisis on nutritional outcomes;
2. Analyze the factors which contribute to the vulnerability of communities to said shocks; and,
3. Consider policy measures which could mitigate the vulnerability of communities to future market shocks.

I made a first attempt at goal (1) in my MA thesis. In this essay, I re-approach the problem and develop an empirical strategy to identify the impact of the 2008 crisis on maternal-infant nutrition, using data from Senegal and eventually Cambodia. Goals (2) and (3), for now, will be limited to brief discussion in my introduction, alongside a brief account of my motivation in studying this topic.

Introduction

Small, poor economies, such as Senegal or Cambodia, are increasingly squeezed between two sources of volatility. First is local weather shocks, which are increasing in frequency and severity in both the Sahel and South-East Asia due to climate change. Second is commodity price shocks on the world market, which cause drastic swings in food purchasing power for the poor. Relying on international trade is often cited as an promising way to mitigate the risks of climate change in developing countries, as stable international prices can smooth good and bad local years. However, with trade comes the volatility from world markets, and this volatility reduces the scope of useful adaptation to climate change.

The pair of Senegal and Cambodia are particularly enlightening because of their opposite positions with regard to macroeconomic impacts of the shock. Cambodia is a rice exporter and Senegal and importer. The rice price spike was a boon for the economy of Cambodia, but a damaging deterioration of the terms of trade for Senegal. I predict that both countries will exhibit similar, negative responses to the food crisis because the vast majority of people in both countries are net-food importers, despite the aggregate exporter position of Cambodia.

In this project, my first goal is to measure the harm that came from the 2008 crisis, harm which has long-run implications for health and well-being. Anthropologists and journalists have no hesitation in stating that market shocks cause great harm in poor countries. Economists are less sure, and recent evidence on the effect of producer prices on child health is mixed.[1]

[1] See (Cogneau and Jedwab 2012) and (Miller and Urdinola 2010) for conflicting views on the effect of commodity prices on producer households.

The 2008 world food price crisis, which saw the tripling of the price of rice in three months without any fundamental supply shifts, is a clear place to start such documentation. The crisis has received substantial attention across many disciplines since 2008,[2] but with a well-noted lack of direct evidence on health impacts from the crisis (Compton, Wiggins, and Keats 2010). Surveys found deterioration of diet-quality (Martin-Prevel et al. 2012), synthetic welfare analysis has suggested overall increases in poverty (Dimova and Gbakou 2013), but there has been no well-identified study of the health-effects of the 2008 shock.[3]

The reason for this lack of direct evidence is simple: everyone was affected by the 2008 shock, meaning there are no control or treatment groups. My contribution is leveraging the maternal-fetal nutritional process to develop a convincing identification strategy to measure the nutritional impacts of the 2008 crisis on child-bearing women. My outcome, infant hemoglobin, is both a bio-marker of maternal nutrition, and a key factor in cognitive development, meaning that the observed declines have permanent effects on well-being.

Infant Hemoglobin and Maternal Nutrition

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 (Baynes and Bothwell 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.

Breast-milk, on the other hand, contains very little iron, around 0.1 mg per gram (USDA). For reference, 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. Therefore, during the first six months of life hemoglobin in infants is 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.

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

This discontinuity in the response of infant hemoglobin to nutritional environment (very sensitive up until

[2] See (Minot, n.d.; Rosset 2008; Cohen and Garrett 2010; Von Braun et al. 2008), for example.

[3] The closest to a well-identified study would be Arndt et. al. (2016), who compare child weight-for-height before and after the crisis using propensity-score matching.

[4] This depends on whether the child is breastfed. 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). Similarly, 60-70% of Cambodian children are exclusively breast-fed during the first 6 months (Prak et al. 2014).

birth, and then insensitive) offers a clear identification strategy. Given a specific event, we can compare those who were breast-feeding at the time to those who were *in-utero*.

Data

My data for both Senegal and Cambodia comes from the Demographic and Health Surveys (DHS). I have repeated cross-sections of children aged 0-59 months, with their hemoglobin levels and household characteristics. For Senegal, I have cross-sections in 2005, 2010, and 2012, as well as a continuous sampling from 2014-2018. For Cambodia, I have cross-sections in 2000, 2005, 2010 and 2014. In both cases, the 2010 survey contains my group of interest, children who were exposed to the 2008 shock *in-utero*.

Empirical Strategy

Data Generating Process

Below I take a subsample of the 2014 DHS survey in Senegal, of children aged 5-59 months. I exclude the first five months of life because hemoglobin is very unstable during this period. In Figure 1, I plot hemoglobin by age, giving a basic idea of the age-time pattern: from five-months-old hemoblobin levels decline, until they increase again it the end of infancy, around age 2. This pattern is likely due to feeding patterns, as children in Senegal are weaned onto cereal-based diets which have low bio-availability of iron. As hemoglobin follows a clear age path, simply comparing two groups who are of different ages would confound the age effect with the treatment of interest.

In Figure 2, I construct a hemoglobin response function, based off of the biological process I described above. Given a shock at age t of magnitude γ, the effect on child hemoglobin is large for those *in-utero*, null for breast-feeding infants, and then slowly increasing with age as dietary iron becomes more important as a child is weaned and has depleted their fetal iron stores.

In Figure 3, I take simulate a shock, that lasts for 6 periods before gradually dissipating. Then I calculate the implied impact on hemoglobin for each cohort, by summing the response over the months of the shock.

From Figure 3, we can construct treatment and control groups. Those born just before the crisis are nearly completely insulated, while those conceived during the crisis are fully exposed. Thus, give a well-defined shock we can compare these two groups and estimate the effect of the shock.

There is also a wide range of children who are partially exposed to the shock: those exposed to the shock post-weaning, those conceived after the end of the shock, and those who were partially exposed to the shock *in-utero* and partly while being breastfed. The effect of their exposure depends on the assumptions I make about the linearity of the effect of the shock as well as the level of persistence in the shock. Both of these are very uncertain, so I plan to discard these groups in my main analysis.

In Figure 4, I apply the calculated shock to hemoglobin data from Senegal in 2014. First I take the median hemoglobin by age, then I subtract the effect of the shock, which is defined by month-of-birth, to each of these cohorts.

In Figure 4, we see that the effect of the shock cannot be determined simply by comparing the affected (red) and insulated (yellow) groups, as that would confound the effect of the shock with the month-of-birth and age patterns. However, other than time and age, the red and yellow groups should be comparable– balanced across observables. Therefore, the challenge is to properly control for age.[5]

[5]Seasonality of birth might also be a concern, as both disease environment and food-availability follow distinct seasonal patterns. I have found no evidence of season-of-birth being an important determinant in child hemoglobin, thus I do not discuss it in this essay.

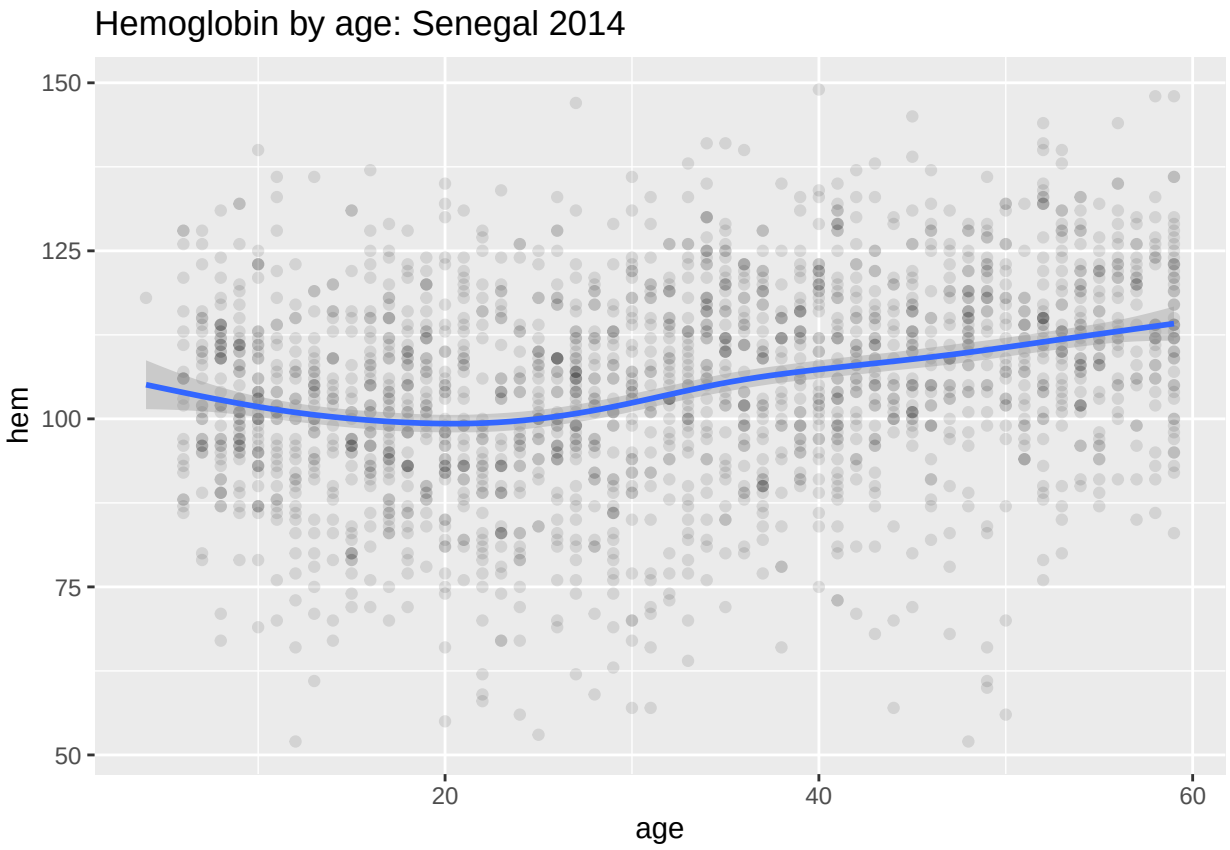

Figure 1: Hemoglobin by age in Senegal: DHS 2014. The blue curve is a LOESS smoothing curve.

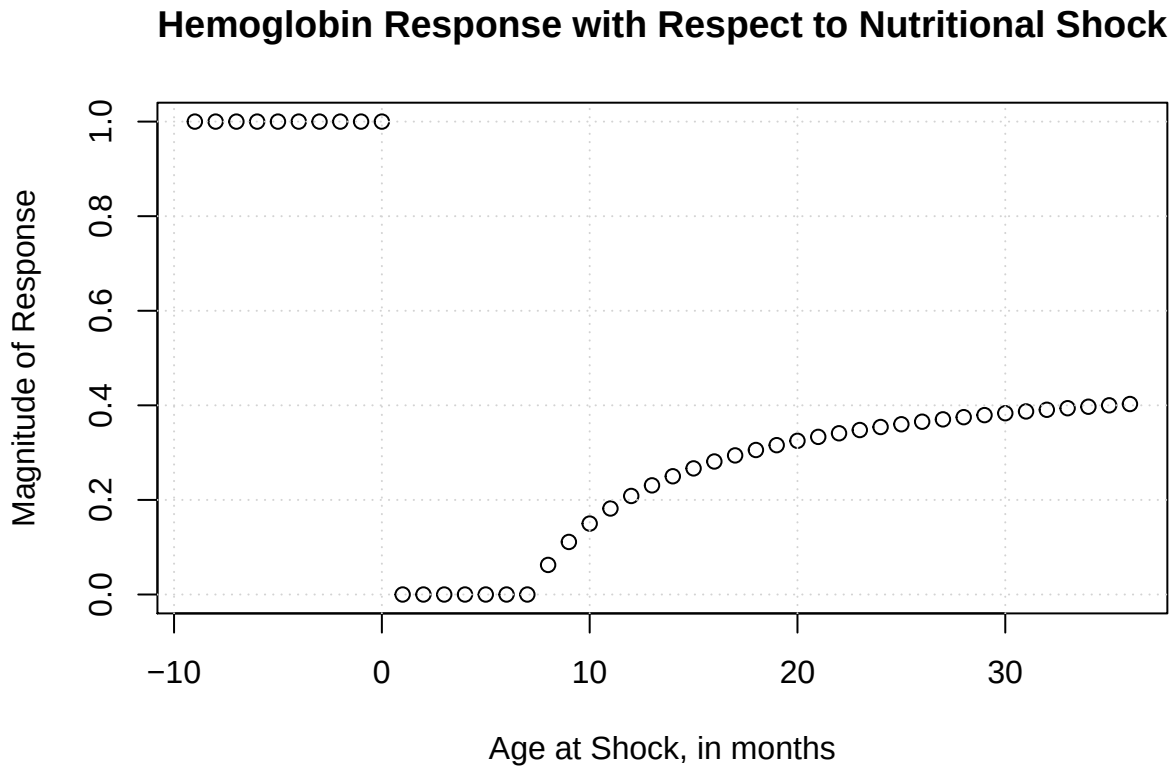

Figure 2: Hemoglobin response function with respect to a nutritional shock. Gestation is the most sensitive period, ages 0-6 months are relatively insensitive, and then sensitivity increases with age.

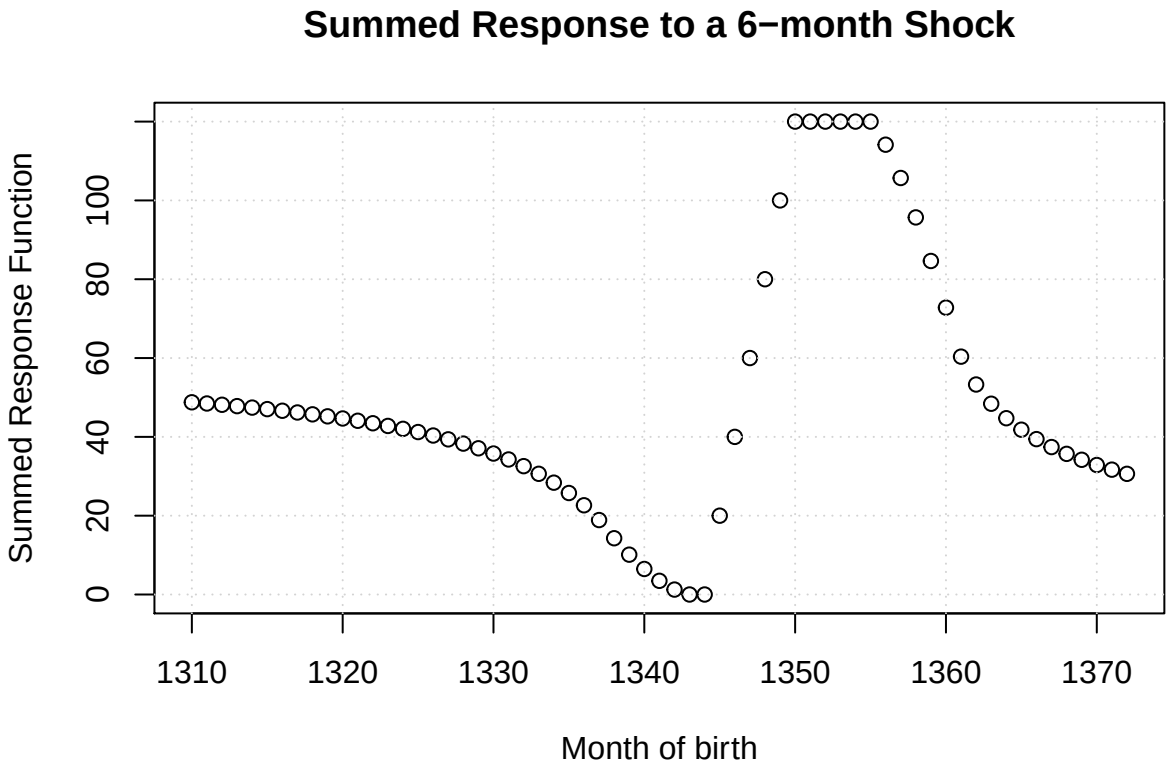

Figure 3: Effect of 6-month shock, calculated by summing the response function for each shock period. The shock has some degree of persistence past the six months, but declines with time.

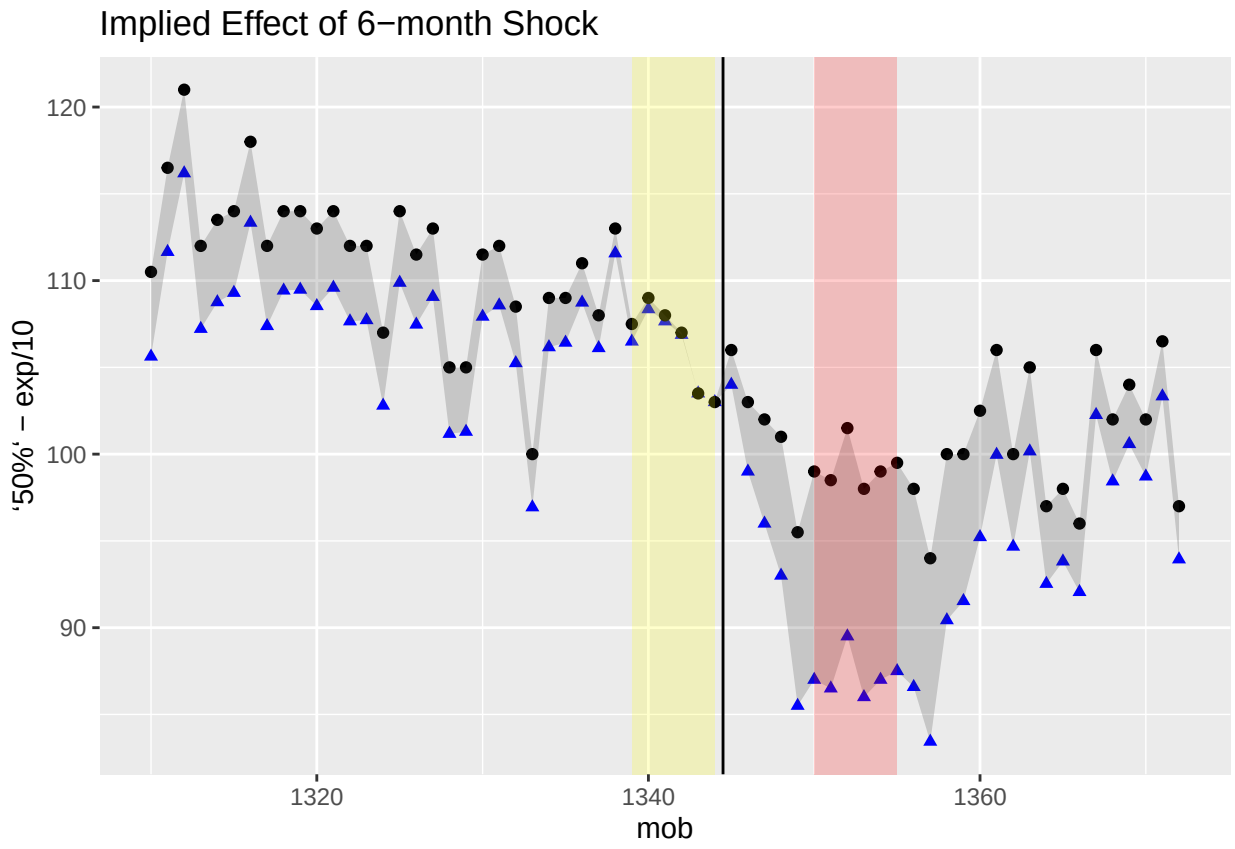

Figure 4: The Implied Effect of a 6-month shock, which starts at the black vertical line. The black dots are the original data, and the blue triangles are the original data minus the summed response to the shock. The gray ribbon show the effect side, the red rectangle shows the ideal treated group, and the yellow rectangle the ideal control group.

Regression Equation

Having given my thought-experiment, the issue becomes how to realize it. How best to "control" for age? That certainly depends on my goal, so I think that it becomes paramount to define this goal.

Goal: estimate the deviation between the treated and control group, relative to the expected deviation given differences in age-at-testing.

With this goal in mind, I will estimate:

$$H_{it} = \beta_1 Treated_t + \beta_2 Control_t + g(Age_{it}) + \epsilon_{it}$$

where Age_i is the age of the child in months, and t indexes the child's month-of-birth. The main question is how to convincingly control for age with as few parameters as possible.

Constructing the Control Functions

I am not interested in inference from functions g, only in the parameter $\delta = \beta_1 - \beta_2$. In order to choose the proper functional form for g and f, I use cross-validation of Chebyshev polynomials. My training dataset is the observations whose birth month is odd, and the validation set is those whose birth month is even. I use median-regression to obtain predicted values for each degree one through twenty. Then I calculate the mean absolute deviation from the true hemoglobin values in the validation set.[6] The mean absolute deviation is plotted in Figure 5. After declining sharply up until $N = 4$, the error stabilizes.

In Figure 6, I plot the predicted values from the fourth-degree Chebyshev polynomial as compared to a non-parametric median regression. The non-parametric regression stays centered around the polynomial for the entire support, but the Chebyshev polynomial of degree four only requires four parameters.

Going forward, I will run this regression separately for each subpopulations of interest, breaking up my data by administrative district and urban status. **One open question I have is whether there are other functional forms which I should compare with the Chebyshev approach.**

[6] MSE would be inappropriate in this setting, as extreme hemoglobin values are likely due to idiosyncratic biological factors, as well as errors in testing. Having this strong prior to down-weight outliers, I use the median.

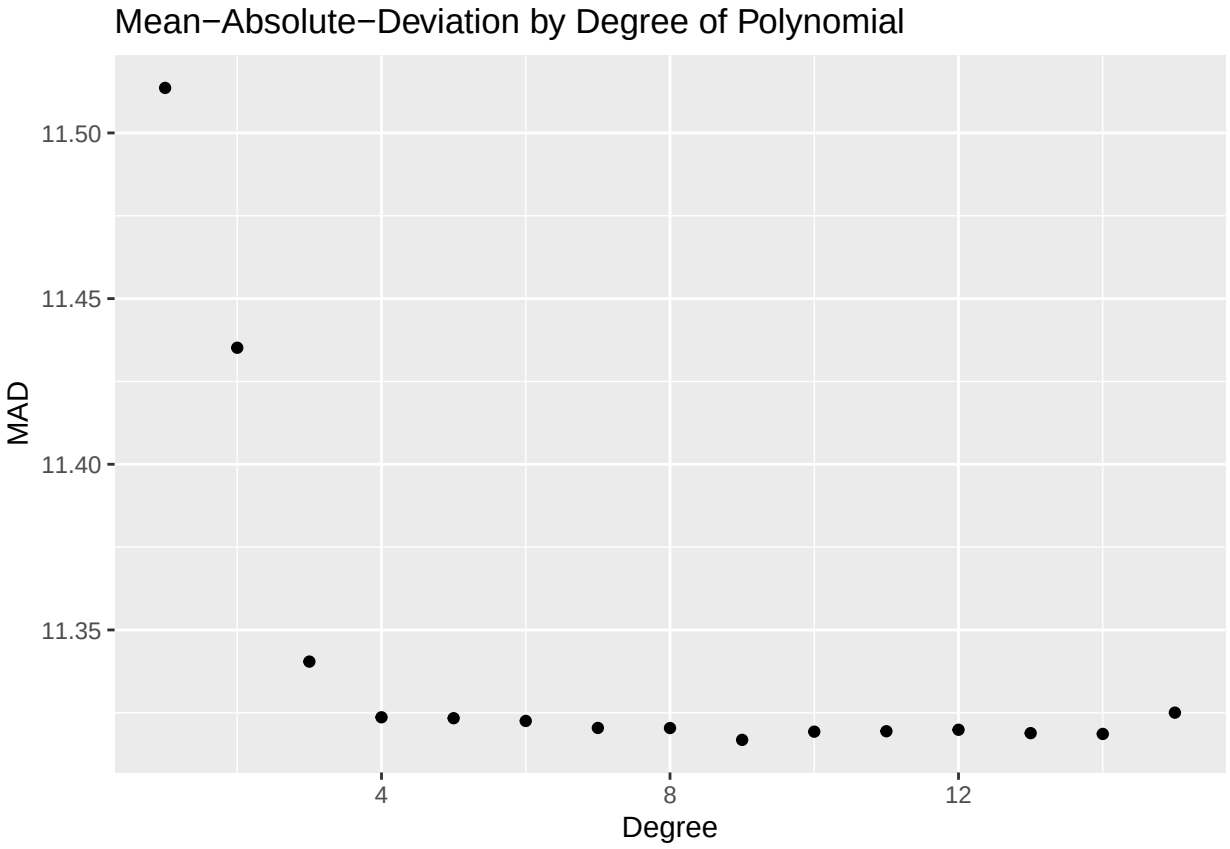

Figure 5: Mean absolute error from Chebyshev polynomial regression by the degree of the polynomial. The regressions are trained on observations whose birth month is odd and validated on the complement. The training regression in least-absolute deviation (aka quantile regression at the median).

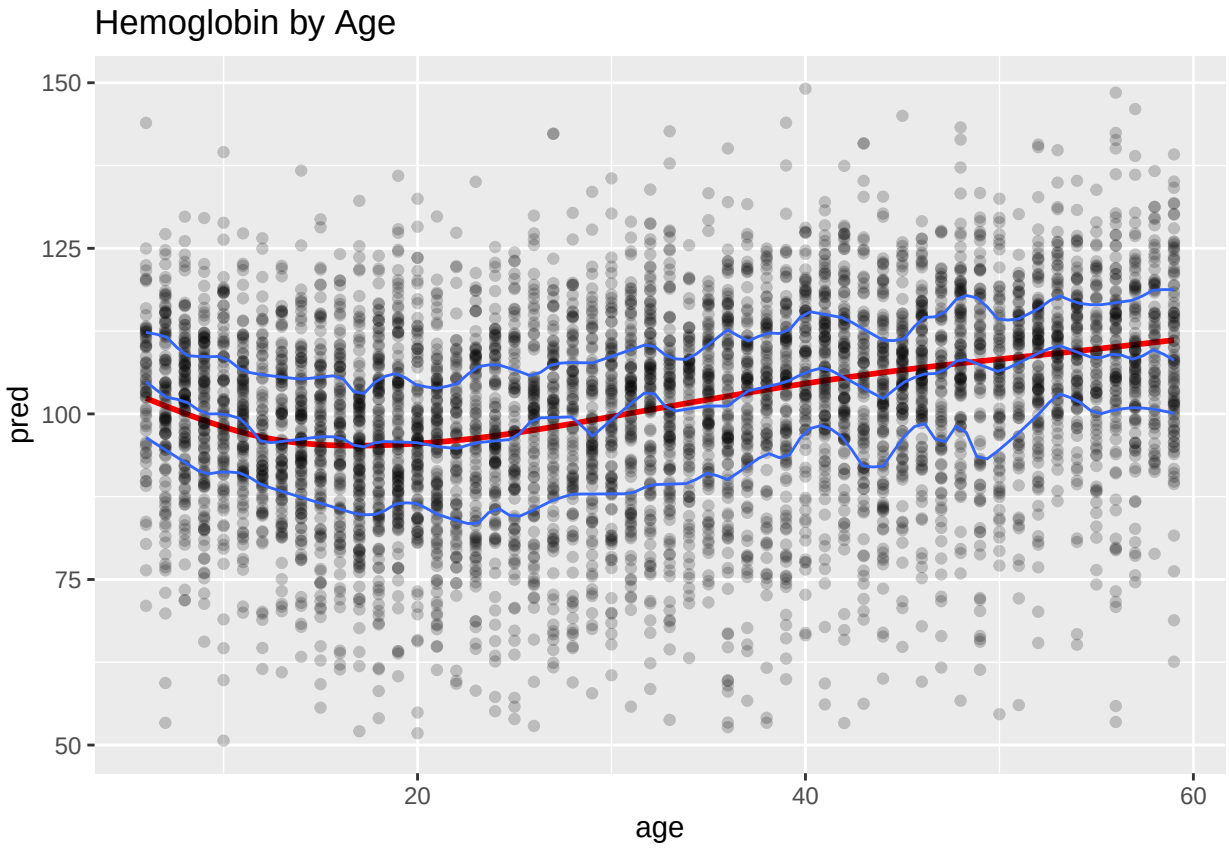

Figure 6: Hemoglobin by age. The fourth-degree Chebyshev polynomial, in red, is compared to the conditional median evaluated at every month of age, blue central line, as well as the conditional 25th and 75th percentiles..

References

Baynes, R. D., and T. H. Bothwell. 1990. "Iron Deficiency." *Annual Review of Nutrition* 10 (1): 133–48.

Camaschella, Clara. 2015. "Iron-Deficiency Anemia." *New England Journal of Medicine* 372 (19): 1832–43.

Cogneau, Denis, and Rémi Jedwab. 2012. "Commodity Price Shocks and Child Outcomes: The 1990 Cocoa Crisis in Côte d'Ivoire." *Economic Development and Cultural Change* 60 (3): 507–34.

Cohen, Marc J., and James L. Garrett. 2010. "The Food Price Crisis and Urban Food (in) Security." *Environment and Urbanization* 22 (2): 467–82.

Compton, Julia, Steve Wiggins, and Sharada Keats. 2010. "Impact of the Global Food Crisis on the Poor: What Is the Evidence." *London, ODI* 99: 99.

Dimova, Ralitza, and Monnet Gbakou. 2013. "The Global Food Crisis: Disaster, Opportunity or Non-Event? Household Level Evidence from Côte d'Ivoire." *World Development* 46: 185–96.

Mané, N. Binta, Kirsten B. Simondon, Aldiouma Diallo, Adama M. Marra, and François Simondon. 2006. "Early Breastfeeding Cessation in Rural Senegal: Causes, Modes, and Consequences." *American Journal of Public Health* 96 (1): 139–44.

Martin-Prevel, Yves, Elodie Becquey, Sylvestre Tapsoba, Florence Castan, Dramane Coulibaly, Sonia Fortin, Mahama Zoungrana, Matthias Lange, Francis Delpeuch, and Mathilde Savy. 2012. "The 2008 Food Price Crisis Negatively Affected Household Food Security and Dietary Diversity in Urban Burkina Faso." *The Journal of Nutrition* 142 (9): 1748–55.

Miller, Grant, and B. Piedad Urdinola. 2010. "Cyclicality, Mortality, and the Value of Time: The Case of Coffee Price Fluctuations and Child Survival in Colombia." *The Journal of Political Economy* 118 (1): 113–55.

Minot, Nick. n.d. "Transmission of World Food Price Changes to," 45.

Neville, Margaret C., Ronald Keller, Joy Seacat, Valerie Lutes, Marianne Neifert, Clare Casey, Jonathan Allen, and Philip Archer. 1988. "Studies in Human Lactation: Milk Volumes in Lactating Women During the Onset of Lactation and Full Lactation." *The American Journal of Clinical Nutrition* 48 (6): 1375–86.

Prak, Sophonneary, Miriam Iuell Dahl, Sam Oeurn, Joel Conkle, Aaron Wise, and Arnaud Laillou. 2014. "Breastfeeding Trends in Cambodia, and the Increased Use of Breast-Milk Substitute: Why Is It a Danger?" *Nutrients* 6 (7): 2920–30. https://doi.org/10.3390/nu6072920.

Rosset, Peter. 2008. "Food Sovereignty and the Contemporary Food Crisis." *Development* 51 (4): 460–63.

Von Braun, Joachim, Akhter U. Ahmed, Kwadwo Asenso-Okyere, Shenggen Fan, Ashok Gulati, John Hoddinott, Rajul Pandya-Lorch, Mark W. Rosegrant, Marie T. Ruel, and Maximo Torero. 2008. "High Food Prices: The What, Who, and How of Proposed Policy Actions."