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Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador

Ana Larrea[†] and Xavier Ramos[‡]

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Abstract:
We measure the effect of the 1999 Ecuadorian financial crisis on the z-score of height-for-age in 2012. A tax on all financial transactions was deployed on 1 Jan 1999 creating a liquidity, currency, and inflationary crisis. Individuals born after this shock were exposed to pre-natal maternal stress in-utero. We use a regression discontinuity model to estimate the average treatment effect by measuring the difference in outcomes between individuals born days before and those born days after the crisis. We find a significant deleterious effect of this shock on the z-scores of height-for-age in 2012 and propose the increase in stress affects the epigenome of the offspring affecting stunting in the long term.

JEl codes: I12, I14, C21
Keywords: Maternal stress, financial crisis, weight for age, regression discontinuity design.

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1. Introduction

After a decade of financial liberalization, risky lending operations and a general failure to effectively monitor bank operations, in 1998, the all-time lowest price of oil left Ecuador with a painful lack of foreign currency, and, the worst "El Niño" phenomenon in its history impaired banks assets and created a gaping hole in public finance. The last quarter of 1998 saw a drain in liquidity leading the Central Bank of Ecuador (CBE) to simultaneously provide lender of last resort assistance and perform open market operations in a futile attempt to control inflation. On 1 Jan 1999 an unusual tax on all financial transactions fueled a drastic fall in total deposits,[1] a swift and massive flight in liquidity as preferences shifted to the dollar and accelerated the collapse of various financial institutions in Ecuador. By March 1999 the run on deposits and the currency crisis led the government to declare a bank holiday and freezing financial assets. By October 1999 the government had suspended payments on Discount and PDI Brady Bonds and Brady and Eurobonds, and by March 2000, Ecuador had adopted the US dollar as legal tender (Jacome, 2004; Sturzenegger & Zettelmeyer, 2008).

We interpret the crash in liquidity in Jan 1999 as the point of infliction, as the sudden and precipitous collapse of the financial system is an objective stress shock for individual deposit holders. An unanticipated potentially measurable amount of hardship endured by a pregnant individual exposes the offspring to pre-natal maternal stress changing its fetal environment. This type of change can cause alterations in the series of "switches" which determine whether parts of a genome are expressed or not, such that, the health effects of an intra-uterine shock may remain latent though the life cycle (Almond & Currie, 2011).

In this paper we measure the effect of this 1999 intra-uterine shock on the 2012 z-score of height-for-age of the offspring. To estimate the average treatment effect (ATE) we use a regression discontinuity (RD) design. We compare children born just after the 1 Jan 1999 "cut-off" with those born just before. This creates a counter-factual (control group) which can be assumed to have very similar observable and unobservable characteristics to the treatment group, and thus, allows us to identify the causal effect of the exogenous in-utero stress shock. We find children born after the crash to have significantly lower z-scores (in 2012) than children born before. Although we cannot test this hypothesis directly, we propose that the financial crash created an intra-uterine shock through pre-natal maternal stress (Almond & Currie, 2011).

[1] See Figure 1.

RD models do not assume the treatment variation is "as good as random" but rather this variation is a consequence of the inability of agents to control the assignment variable near the cut-off. The choice of these bandwidths, polynomial forms, and kernel functions is fundamental in the analysis and interpretation of RD designs. Therefore, we make sure relevant observable characteristics are not significant determinants of selection into treatment. We use a data-driven method to select an appropriate bandwidth, we use the Akaike information criterion (AIC) as well as a dummy variable test in order to select the polynomial order, and finally, we test the sensitivity of the results to triangle, rectangle and Epanechnikov kernel functional forms (Cattaneo, et al., 2018; Lee & Lemieux, 2010). Additionally, we run 4 robustness checks: (1) placebo effects for the months and years predating the crisis and placebo effects for individuals without bank accounts; (2) we examine the density of the running variable, and (3) test for the sensitivity of the model to observations near the cut-off, and, finally, (4) we test to see if other observables have the same cut-off.

This study contributes to the literature in three ways: (i) we study a financial crisis which is not typically taken as a stressful life event in the literature; (ii) we measure long-term effects rather than short- or medium-term ones; (iii) we find a natural experiment where an exogenous cut-off allows for the measurement of a causal long-term effect on health. This paints a more comprehensive picture of the consequences of pre-natal maternal stress. (iv) Finally, we have not found studies which use regression discontinuity models or which analyze the long term health effects of pre-natal exposure to the 1999 Ecuadorian crisis which makes this an original contribution to the debate.

This paper is divided into six parts: Section 2 explains the origin, outbreak and aftermath of the 1999 Ecuadorian crisis; Section 3 outlines the fetal origins hypothesis, the empirical evidence, and how it applies to this case; Section 4 explains the empirical strategy; Section 5 presents the data and main results; Section 6 presents the robustness checks, which go through every case where our regression discontinuity model might fail; and finally Section 7 concludes with a discussion of our main findings.

2. Context: the financial crisis of 1999

1.1. Run-up to the crisis (1994-1998)

The run up to the crisis was marked by three important events: (1) The liberalization of financial markets leading to a first liquidity crisis in 1994, (2) a depleted oil price ($10/barrel) in 1997, coupled with, (3) the worst "El Niño" phenomenon in recorded history during the winter of 1997-8. Surrounding these events

was a period of political and social unrest. In this section we will briefly explain the details which are relevant to the 1999 financial crash.

In 1994 the Law of Financial System Institutions[2] which liberalized interest and exchange rates, was enacted. The law promoted the free entry and exit of institutions to the financial market and allowed for an expansion of bank operations particularly in foreign currency and in offshore branches. Central Bank of Ecuador (CBE) was named lender of last resort (LOLR) and was only allowed to provide liquidity assistance in the local currency (Sucres). Additionally, the amount of liquidity assistance allowed was unlimited and the deposit guarantees would rely on CBE funds. Finally, there was a rapid reduction in bank reserve requirements from 28% to 10% in domestic currency and from 35% to 10% in foreign currency. This was essentially part of a greater liberalization process which had begun in the early 1990's that coincided with a parallel increase of capital inflows and attracted to higher domestic returns. Between 1993 and 1994 the CBE international reserves doubled and the number of financial institutions increased by more than 30% (from 33 to 44) (Jacome, 2004; Martinez, 2006).

Financial intermediaries failed to gauge the risk in lending operations[3] and the Superintendence of Banks and Insurance Companies[4] failed to effectively monitor these operations, particularly in offshore branches. This allowed banks to circumvent regulations and controls and engage in transactions with currency and maturity mismatches in the denomination of assets and liabilities,[5] connected lending, large amounts of non-performing loans and, in some cases, even fraudulent operations (Jacome, 2004; Martinez, 2006).

In 1995, the border conflict with Peru[6] and "a number of other exogenous shocks" led to an unanticipated liquidity crunch. In order to control inflation, the CBE stabilized the exchange rate by contracting money through Open Market Operations (OMO). This pushed the nominal interest rate up to 50%[7] which created liquidity problems for banks with maturity mismatches. Banco Continental failed and was acquired by the

[2] Name in Spanish: Ley General de Instituciones del Sistema Financiero de 1994. Executive Order 1852 in Official Registry 475, 4 Jul 1994 (Decreto Ejecutivo 1852 Registro Oficial 475 4 de Julio de 1994).
[3] Credits increased 40% in 1993 and 50% in 1994.
[4] Superintendencia de Bancos.
[5] Currency or maturity of assets not equal to currency or maturity of liabilities.
[6] January 26 – February 28, 1995.
[7] And the real interest rate up to 30%.

State. The CBE isolated the crisis by providing liquidity support to other banks. An ominous equilibrium ensued in 1997 and, with the liquidity conditions restored, the interest rate decreased (Jacome, 2004).

Nevertheless, the banking system remained fragile due to poor quality of bank assets and a resulting equity shortage. In the winter of 1997-1998 Ecuador suffered the worst "El Niño" phenomenon in its history. This destroyed agricultural areas, particularly in the coastal regions, impairing banking assets. Additionally, in early 1997 both president and vice-president were removed from office and a very close general election was held in May 1998. Meanwhile, the price of oil was $10 a barrel making foreign currency scarce and hurting public finance (Jacome, 2004).

Solbanco was the first (small) bank to close in April 1998. This led to a wave of withdrawals in other banks. In August 1998, a medium sized bank (Banco de Préstamos) closed and returned depreciated deposits of only small savers after several weeks. Larger deposit holders did not receive savings back. In September 1998 a large bank (Filanbanco) along with 11 other financial institutions requested lender of last resort (LOLR) assistance from the CBE. Between September and November of 1998, the LOLR assistance provided by the CBE reached 30% of the money base. In order to hold down the depreciation of the currency, the CBE tried to mop up liquidity by simultaneously selling bonds[8] through OMOs (See Appendix 1 for Figures on financial assistance to banks, OMOs and net international reserves). This proved insufficient as the Sucre depreciated by 24%, inflation reached 15% and international reserves fell by 7.6%. Finally, in the last quarter of 1998 banks foreign credit lines experienced a US$300 million cut due to the Russian and Brazilian crisis (Jacome, 2004; Martinez, 2006).

1.2. The AGD and the 1% tax: first trimester of 1999

In December 1998, legislation[9] meant to deal with the absence of effective bank resolution instruments was approved by Congress. The law created the Deposit Guarantee Agency (AGD)[10] in order to provide a blanket guarantee of deposits and instituted a 1% tax on all financial transactions meant to increase government revenue while simultaneously eliminating all income tax (Cantos Bonilla, 2006; Jacome, 2004).

[8] *Bonos de estabilización monetaria, BEMs*

[9] Name in Spanish: Ley de Reordenamiento en Materia Económica en el Área Tributario - Financiera. Published in the Official Registry Supplement 78 1 Dic 1998. (Publicada en el Suplemento del Registro Oficial No. 78 del I de diciembre de 1998).

[10] Given its name in Spanish: Agencia de Garantía de Depositos.

The AGD began operating on 1 Dec 1998 and was entitled to "purchase and assume operations" of financial institutions. Notwithstanding, 6 banks were closed between December 1998 and January 1999 except Filanbanco which was considered "too big to fail." In order to materialize the blanket guarantee in a context of lacking fiscal funds long term securities (AGD bonds) were used. The AGD started honoring the blanket guarantee with resources from the CBE only in April 1999. This fueled withdrawals from other banks, eroded AGD credibility and stimulated contagion (Cantos Bonilla, 2006; Jacome, 2004).

On 1 Jan 1999 the financial tax was deployed. It proved devastating for the financial system as it was enacted in the context of waning confidence. This drove the largest liquidity flight since the first bank failure in April 1998 (Figure 1), a speculative run on the Sucre as preferences shifted to the Dollar, and indirectly, it increased pressure on the exchange rate, and, accelerated the collapse of various financial institutions as deposits plummeted [11]. By February 1999, CBE international reserves had shrunk to the point where sustaining the exchanged rate was no longer possible. During this month, the CBE floated the Sucre resulting in an almost immediate 50% devaluation (Jacome, 2004; Cantos Bonilla, 2006).

The ensuing months saw the predictable consequences. In the early days of March 1999, the largest bank (in terms of deposits, Banco del Progreso) experienced a massive run on deposits. This, coupled with the currency crisis and the systematic lack of confidence, led the government to declare a bank holiday on Monday March 8th, 1999.[12] This holiday lasted a week and finalized in the widespread freezing of all bank accounts with a balance over 500 USD to avoid further capital flight. Savings accounts would be frozen for a year and checking accounts for 6 months (Jacome, 2004).

1.3. Discussion on our crisis threshold

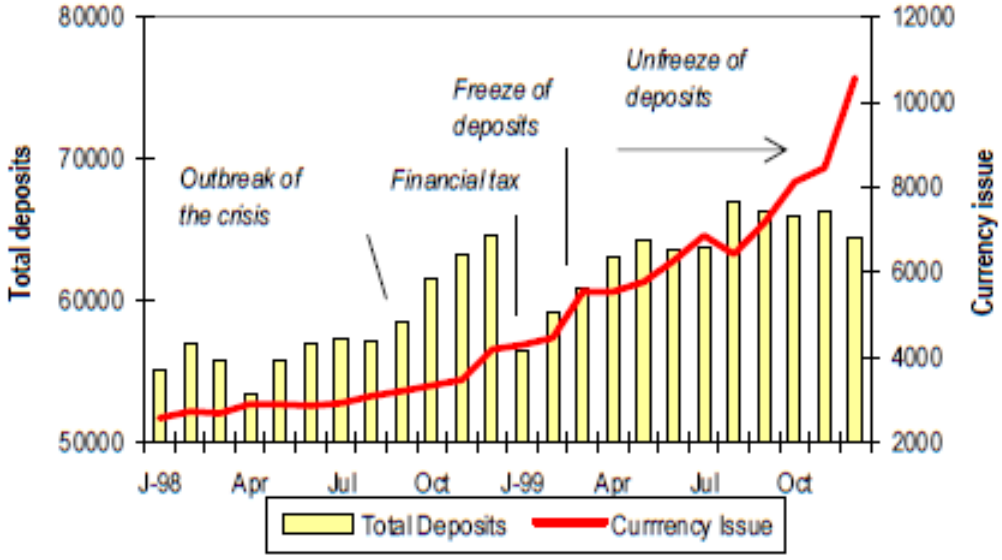
Waves of withdrawals occurred fairly regularly in Ecuador. However, as we can see in Figure 1, between the first bank failure (April 1998) and December 1998 total deposits continued to increase. Only in Jan 1999 did total deposits fall. Figure 2 shows how the largest liquidity crunch faced by the banks also occurred in January 1999 which was only stopped with the freezing of bank deposits in March 1999. Why would there be a bank run in January 1999 if banks were closing since April 1998 and inflation and devaluation was

[11] Notably the largest bank in terms of deposits (Banco del Progreso).
[12] Meaning that banks remained closed.

increasing since September 1998 (see Figure 3 & Figure 15) (Jacome, 2004)? Furthermore, wouldn't the creation of the AGD have been meant to prevent capital flight?

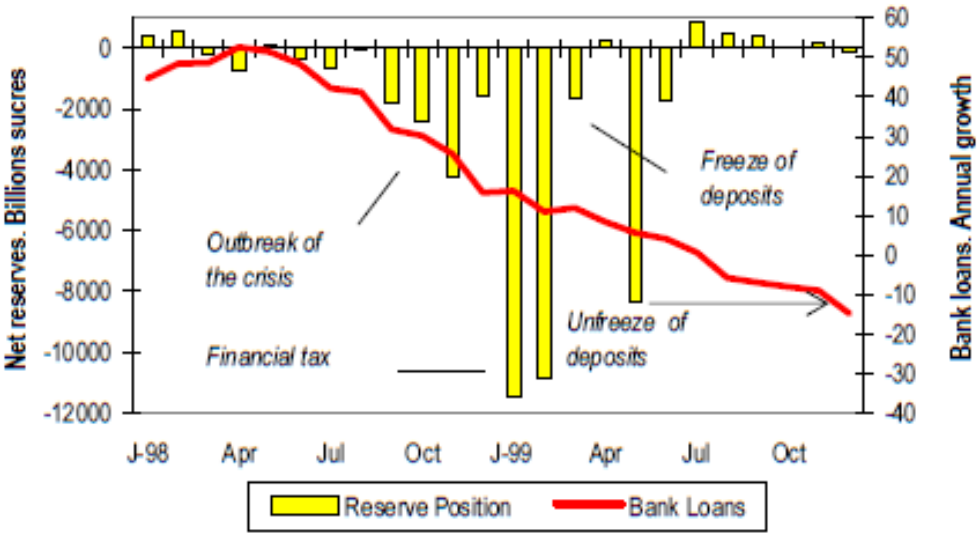
We believe the financial tax marks the beginning of the bank run, despite the approval of the tax occurring on 1 Dec 1998, leaving enough time for deposit holders to anticipate and adapt to it. Total deposits grew in December 1998 (Figure 1), therefore, the extent to which deposit holders adjusted expectations in anticipation of the tax did not take into account the collapse of the economy. If deposit holders could have anticipated the crisis with the announcement of the tax, deposits would have decreased in December 1998. We thus argue the contagion effect the tax had on deposits was unanticipated by policy makers and deposit holders. And it is this unanticipated nature which makes the 1 Jan 1999 a suitable cut-off for our regression discontinuity design.

Figure 1 Total Deposits and Currency Issue (Billions of Sucres)



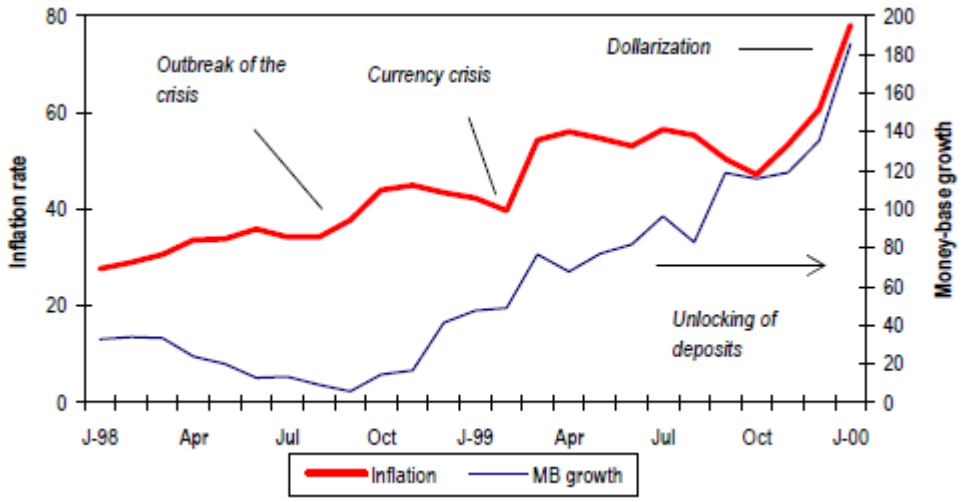
Source: Jacome, 2004; Source of data cites in Jacome, 2004: Central Bank of Ecuador

Figure 2 Liquidity and credit crunch



Source: Jacome, 2004; Source of data cites in Jacome, 2004: Central Bank of Ecuador

Figure 3 Inflation and monetary base growth (annual percentage rate)



Source: Jacome, 2004; Source of data in Jacome 2004: Central Bank of Ecuador.

3. Mechanism: intra-uterine shocks

3.1. Stress and the fetal environment

The fetal origins hypothesis, proposed by British physician and epidemiologist David J. Barker, suggests that exposure of the fetus to adverse environmental in-utero conditions affect the programming of certain

metabolic characteristics which may have effects later in life (Barker, 1990). Specifically, fetal conditions affect a series of "switches" referred to as the epigenome that determine whether parts of a genome are expressed or not (Almond & Currie, 2011). The genome of an individual, which can be described as the "hardware" of genetics is determined at conception and is fixed over time. However, the epigenome of an individual can be described as the "software" of genetics, i.e. the "switching" on or off of genes, and can change as a result of environmental shocks.

Gluckman et al. (2005) propose that this is basically a predictive adaptive response the fetus has to an early environmental "cue". In other words, an intra-uterine shock may be interpreted by the fetus as a signal of its post-natal environment, leading it to preemptively adopt a developmental trajectory which might better suit its expected future living conditions. This "coping" mechanism can be advantageous or disadvantageous depending on the degree of mismatch between the predicted and actual future environment. Therefore, the response can have long term effects on the individual's fitness for survival if it imposes costs that impact that individual at a later stage in life. For example, a response of the fetus to a reduction in maternal nutrition is to alter its fetal growth pattern in such a way that it matches the supply of nutrients. This allows the fetus to survive, however, it may have post-natal costs such as altered pancreatic development, insulin release and blood vessel (which supply nutrients) growth, leading, for example, to an abnormal level of insulin "resistance" meant to save energy consumption for survival. This may affect the individual's fitness later in life.

Rice and Thapar (2010) and Rice et al. (2010) effectively disentangle the effect of the fetal environment (on the epigenetics) from the effect of "hardware" genetics by studying parents who conceived by in vitro fertilization where some were genetically related to their offspring while others where not. This distinction allows them to identify the contribution of maternal intrauterine environment to offspring birth outcomes independently of the contribution of the genome. They find a correlation between maternal *height*, offspring *birthweight*, and *head circumference* among both genetically related and unrelated offspring. These results suggested a possible biological interaction between the intrauterine environment and birth outcomes beyond the genetic (Rice & Thapar, 2010). The same authors use the same in vitro fertilization design to study associations between prenatal stress and offspring birthweight, gestational age and antisocial behavior. They find significant correlations between pre-natal stress and birth outcomes among

genetically related and unrelated offspring. These results are consistent with the hypothesis that the pre-natal maternal stress has an important role in birth outcomes (Rice, et al., 2010; Rice & Thapar, 2010).[13]

Pre-natal maternal stress can increase levels of CRH (Corticotropin-releasing hormone) which regulates the duration of pregnancy and fetal maturation (Holzman, et al., 2001; Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008). Endocrinologist Jonathan Seckl[14] considers excess levels of stress hormones in the fetus "reset" an important arbitrator of stress in the body making it hypersensitive to even banal events (Couzin, 2002). There is an increasing amount of empirical evidence of the link between intra-uterine stress shocks and adverse health outcomes at birth and later in life.

3.2. Empirical evidence for intra-uterine shocks

Although Barker's initial work was essentially correlational (Barker & Osmond, 1986; Barker, 1995), we find increasing amounts of evidence which suggest an empirical link. We found five meta-analyses which describe the mixed evidence between pre-natal maternal stress and birth outcomes. Beydoun and Saftlas (2008) find that 9 out of 10 studies report significant effects of PNM stress on birth weight, low birth weight (LBW) or fetal growth restriction. Almond and Currie (2011) find numerous studies providing evidence of the long-term consequences of a wide variety of intrauterine shocks. Conversely, Zijlmans' et al. (2015) meta-analysis finds only a small number of significant associations between maternal prenatal *cortisol* and child outcomes. However, they find a large heterogeneity in study designs and cortisol assessment methods. They argue that maternal *cortisol* may not to be the only or main mechanism in the maternal prenatal stress - child outcomes relation (Zijlmans, et al., 2015).

Notwithstanding, Bussieres et al. (2015) find three factors are relevant on the magnitude of the effect: (1) Pregnancy-related stress (e.g. fear of childbirth) effects are greater in magnitude than non-pregnancy related stress (e.g. life event measures). (2) Studies involving high-risk samples (e.g. adolescents, mothers with hypertension, diabetes) tend to produce greater associations as compared to low-risk groups. Finally, (3) studies conducted outside of North America/Europe produce greater effect sizes (Bussières, et al., 2015)

[13] In contrast, the link between prenatal stress and offspring attention deficit hyperactivity disorder was only present in related offspring.

[14] Of Western General Hospital in Edinburgh, U.K.

Schetter & Tanner (2012) find that a majority of the more than a dozen published studies measuring objective stress events[15] have significant effects on pre-term birth and birth weight, while studies on perceived stress did not consistently predict pre-term birth or birthweight. On the other hand, Hobel et al. (2008) find mixed evidence of links between psychosocial stress and preterm birth. They argue there are two consistently relevant factors to preterm birth: (1) the timing of the stressor, and (2) the woman's perception of it. This seems to contradict Schetter & Tanner (2012), however, they are not referring to measures of perceived stress. Rather, they find that women become less responsive to stressful stimuli as pregnancy advances. Therefore, with some exception, objective life events stressors tend to affect birth outcomes most when they occur in the first trimester (Hobel, et al., 2008; Schetter & Tanner, 2012).

Various studies find significant associations between intra-uterine exposure to natural disasters, such as hurricanes, ice storms, floods and earthquakes, (Currie & Rossin-Slater, 2013; Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016) and the probability of abnormal conditions of the newborn,[16] birth lengths, low birthweight, and pre-term delivery in the US and Canada, and on low birthweight in Haiti. Such associations, however, do not always hold. For instance, no changes in birth outcomes were found after the Fukushima disaster (Leppold, et al., 2017). Family events such as the death of a loved one or a financial stress are found to be significant in shortened gestational age, pre-term birth, low birth weight, and small for gestational age in Sweden, particularly when the shock was in the 5th and/or 6th month while in China the effect on gestational weight gain was found to depend on pre-pregnancy BMI (Class, et al., 2011; Zhu, et al., 2013). Various authors study the events on September 11th 2001[17] and find significant associations with lower term birthweight and birth length (Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016). In Israel, exposure to rocket attacks during the second trimester, and, random landmine explosions in Colombia in the first trimester of pregnancy were associated with LBW (Wainstock, et al., 2013; Camacho, 2008). A study on immigration raids in the USA finds that infants born to Latina mothers had an increased risk of LBW while no such change was observed among infants born to non-Latina white mothers (Novak, et al., 2017).

[15] Acute stressors (e.g. "life events", catastrophic, community-wide disasters), chronic stressors (e.g. household strain or homelessness), and neighborhood stressors (e.g. poverty or crime).

[16] Such as being on a ventilator more than 30 min and meconium aspiration syndrome (MAS).

[17] Terrorist attack on the World Trade Center in New York City.

There is mixed evidence on the effect of a financial crisis in the literature. Studies in Iceland find increase in risk of LBW shortly after the financial collapse in 2008 (Eiríksdóttir, et al., 2013), however, other studies find that six years after the collapse, there is little notable impact of the crisis on key child health indicators (Gunnlaugsson, 2016). Additionally, in Sweden, a study finds no significant increase in the prevalence of gestational hypertension in the first year following the economic collapse (Eiríksdóttir, et al., 2015).

These financial crisis studies focus on the short to medium term effects. Furthermore, most of the studies reviewed in the meta-analysis and on stressful life events focus on short term effects of intra-uterine shocks such as birth outcomes or prevalence after the shock. The studies that focus on long term effect are mainly on pre-natal exposure to famine such as the Dutch famine of 1944[18] where obesity rates were twice as high among those who had first trimester exposure (Stein, et al., 1975) and there was an increase in schizophrenia among those affected (Hoek, et al., 1998). The findings have been replicated for the Chinese famine of 1959-1961 (St Clair, et al., 2005). However, no effect was found for individuals inflicted by the siege of Leningrad (Stanner, et al., 1997) nor for those who affected by the Finnish famine of 1866-1868 (Kannisto, et al., 1997; Almond & Currie, 2011).

This paper contributes to the literature in that it studies the long-term effects of a financial crisis. Our findings could be informative of the possible effect on the pre-natal exposure to the 2008 financial crisis, particularly in countries where the crisis affected individual's savings. Secondly, we measure long-term effects rather than immediate birth outcomes or medium-term effects on prevalence measures. This is relevant given it may provide an explanation for the lack of efficacy of certain public policies focused on improving health outcomes while ignoring the fetal origin hypothesis. It provides evidence that preventive public policy interventions during pregnancy could potentially be more effective in terms of health outcomes later in life. Finally, we find that most studies are correlational, few studies address issues of endogeneity, particularly when dealing with perceptions of stress or pregnancy-related stressors. In studies where there is an exogenous shock there are mostly simple regression methods which compare the before-after outcomes without providing an appropriate counter-factual. This is the first study to our knowledge which provides a causal effect by using regression discontinuity models.

[18] Known as the "Hunger Winter."

4. Methodology

We use a sharp RD model which we explain in this section. If we have an assignment variable $\mathrm{S_i}$ which determines whether the individual receives the "treatment" (the tax shock before birth) with an eligibility cut-off at $\mathrm{S^*}$ (1 Jan 1999) we are able to model the effect of the shock on the individual outcomes $\mathrm{y_i}$ (z-score of height-for-age) using the RD method. This allocation mechanism generates a non-linear relation between "treatment" and number of days born before/after the crisis (S_i). In general, the estimating equation is $y_i = \beta S_i + \varepsilon_i$, where individuals (children) with $s_i \geq s^*$ (born on or after 1 Jan 1999) receive the "treatment" and individuals with $s_i < s^*$ (born before 1 Jan 1999) do not. If we assume that limits exist on either side of the threshold s^*, the impact estimation for an arbitrarily small $\varepsilon > 0$ around that threshold would be as follows (Lee & Lemieux, 2010):

$$E[y_i|s^* - \varepsilon] - E[y_i|s^* + \varepsilon] = E[\beta S_i|s^* - \varepsilon] - E[\beta S_i|s^* + \varepsilon] \quad (1)$$

When taking the limit of both sides of equation (1) as $\varepsilon \to 0$ we identify β as the ratio of the difference in outcomes of individuals just above and below the threshold, weighted by the difference in their realizations of S_i as follows (Lee & Lemieux, 2010):

$$\lim_{\varepsilon \to 0} E[y_i|s^* - \varepsilon] - \lim_{\varepsilon \to 0} E[y_i|s^* + \varepsilon] \Rightarrow y^- - y^+ = \beta(S^- - S^+) \Rightarrow \beta = \frac{y^- - y^+}{S^- - S^+} \quad (2)$$

We assume, that individuals are assigned to "treatment" solely on the basis of the assignment variable (number of days born before/after crisis). Therefore, the assignment variable is deterministic in receiving the "treatment".

5. Data

The National Health and Nutrition Survey (ENSANUT)[19] is a cross-section database built by the National Institute for Statistics and Censes (INEC)[20] in Ecuador between 2011 and 2013. It covers various health topics including anthropometric measures for children, adolescents, and adults. It has a total sample of 92,502 individuals out of which we have a sample of 32,426 children between the ages of 5 and 19 with our outcome variable z-score of height-for-age (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013).

[19] Given its name in Spanish: Encuesta Nacional de Salud y Nutrición.

[20] Given its name in Spanish: Instituto Nacional de Estadísticas y Censos.

5.1. The dependent variable: z-score of height-for-age

The z-score of height-for-age (zhfa_i) was calculated by the INEC and the Ministry of Health using the method proposed by the World Health Organization (WHO). The zhfa_i establishes the growth standard of children by defining a normal growth curve (World Health Organization, 2013; World Health Organization, 1997).

$$\text{zhfa}_\text{i} = {}^{(x_i - x_{median})}/_{\sigma^x} \quad (3)$$

Where x_i is the height of child *i*, x_{median} is the median height from the reference population of the same age and gender and σ^x is the standard deviation of x of the same reference population (Imai, et al., 2014; World Health Organization, 1997). They use anthropometric data available in the LSMS (2006) to calculate the zhfa_i for each individual. In this case we are interested in children between the ages of 5 and 19.

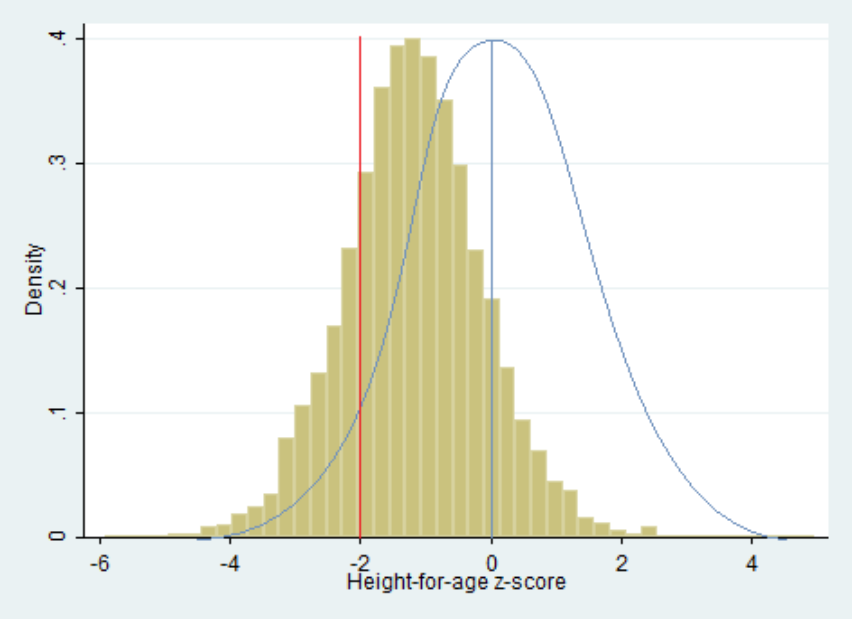
The zhfa_i ranges from $-\infty$ to ∞ as it is measured in standard deviations from the mean which is zero. If a child's zhfa_i is under two standard deviations below the mean, the child is chronically malnourished or "stunted" (World Health Organization, 1997). Figure 3.a and 3.b show the $zhfa_i$ distribution for the whole population and for our sub-sample of children born 30 days before/after the cut-off. As Table 1 shows, the average zhfa_i for children between 5 and 19 is -1.11, and approximately 19% of children in this age range are chronically malnourished, that is, have a zhfa_i under -2 (red line). In our sub-sample[21] the average is -1.14 and the prevalence is 21%.

Table 1 Descriptive statistics of dependent variable: $zhfa_i$

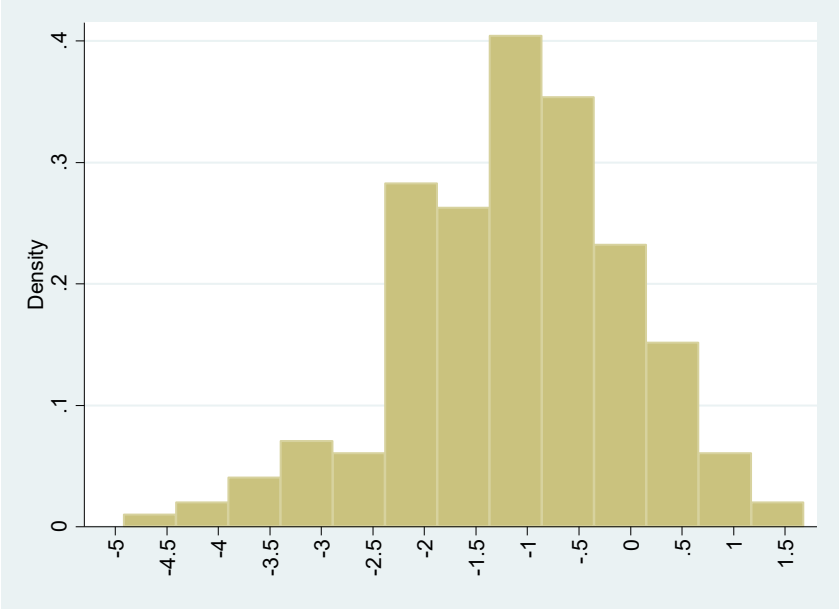
Variable	Obs.	Mean	Std. Dev.	Min	Max
zhfa (all)	18968	-1.11	1.07	-5.9	4.97
D. Malnutrition (all)	18968	0.19	0.39	0	1
zhfa (sub-sample)	195	-1.14	1.11	-4.92	1.68
D. Malnutrition (sub-sample)	195	0.21	0.41	0	1

[21] Of children born 60 days before/after the cut-off.

Figure 4 Distribution of z-score of height-for-age in 2012 among 5- to 19-year-olds in Ecuador



a. Distribution of full sample



b. Distribution of sub-sample of children close to the cut-off point (30 days before and after)

5.2. The assignment variable: days born before/after crisis

In this study we focus exclusively on the children born just before/after 1 Jan 1999 (12 to 13 years old). The assignment variable (S_i) is the number of days the child was born before or after the crisis. The children

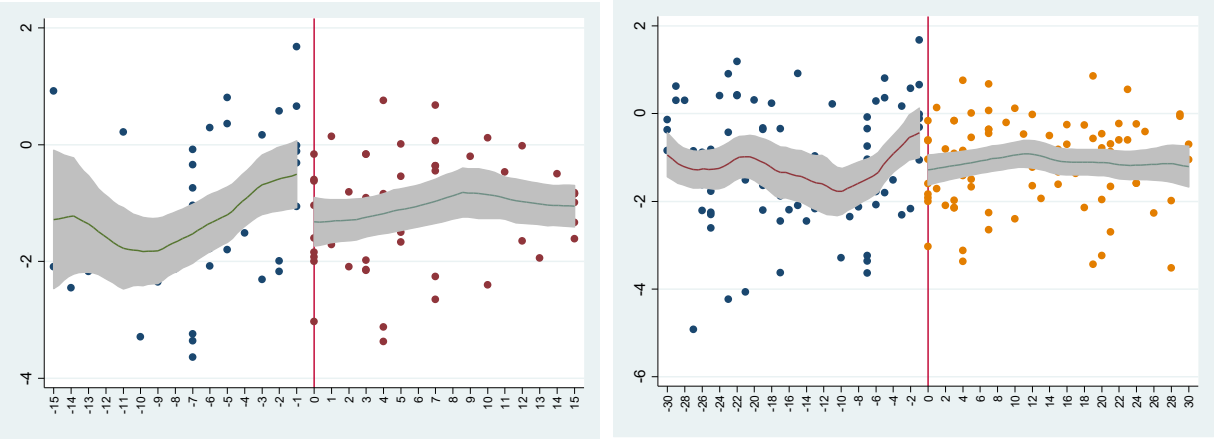
born on the day of the crisis will have an S_i value equal to zero while the children born before the crisis will have a negative S_i value and those born after the crisis, a positive S_i value. Table 2 provides descriptive statistics of S_i for the children born 30 days before/after the crisis.

Table 2 Descriptive statistics of the assignment variable S_i

Variable	Obs.	Mean	Std. Dev.	Min	Max
S_i	196	-1	17	-30	30

Figure 5 is a scatterplot with a local polynomial regression line of the z-score of height-for-age by the running variable S_i with a 15-day bandwidth (panel (a)), and with a 30-day bandwidth (panel (b)). We estimate a separate local polynomial regression on each side of the cut-off in order to visually represent the drop in the z-score of height-for-age which occurs on the day of the crisis, i.e. at $S_i = 0$ (see Appendix 4 for box plot representations of the z-score of height-for-age for different bandwidths around the cut-off).

Figure 5 Scatterplot and local polynomial of 2012 z-score for sample of children born 15/30 days before/after crisis



(a) 15-day bandwidth (b) 30-day bandwidth

6. Results

Before presenting the results, we justify our choice of bandwidth, polynomial order, and kernel function. After that, we also report on various robustness checks.

6.1. Choosing a bandwidth

Choosing a bandwidth within which we are comfortable assuming both observable and unobservable characteristics are randomly assigned is key to this method. A general rule is that the larger the window,

the higher the probability that co-variates might affect or be driving the outcome. The window must be sufficiently small so that 'as good as random' is a reasonable assumption and sufficiently large so that the sample size is large enough to assume the hypothesis test will have adequate power to reject the null hypothesis when it is false (Cattaneo, et al., 2018).

In order to select an appropriate window, we use what Cattaneo et al. (2018) refer to as the data-driven method where the information provided by relevant pre-determined co-variates is taken as an indicator for exogeneity. In this section we present two exogeneity tests. The first simply involves selecting observable characteristics that would be otherwise correlated with our running variable, S_i, everywhere except near the cut-off. The second is a probit model testing for observable differences between treatment and control groups.

In relation to the former, one variable which should be correlated with S_i might be weight. As S_i increases, the weight of the child also increases. We can see in Model 1 of Table 3 that weight has a strong correlation with S_i when the assignment variable has no bandwidth (see also Figure 6). However, when we use a 30- or 15-day bandwidth, there is no significant relationship between weight and age —see Models 2 and 3 in Table 3, and Figure 7.

In relation to the latter, we run 5 probit models using dummy treat as the dependent variable (Table 4). This way, we can test for the significance of various observable characteristics on selection into treatment. Age is significant when using the 30-day bandwidth, which is expected given it is one of the two variables which is used to construct the outcome variable height-for-age. However, with the 15-day bandwidth the results are optimal as none of the covariates are significant. Nevertheless, it is worth noting that there are 87 observations in the 15-day model (1), while in the 30-day model (4) there are 172 observations. We believe this may be a crucial factor given a small sample may not have sufficient power to reject a null hypothesis when it is not significant. Additionally, the 30-day model (4) also presents a highly exogenous probit model, given we expect the age variable to be significant. In any case, in our results we present both the 30- and the 15-day bandwidth models given both seem to be robust to observable characteristics influencing selection into treatment.

Table 3 Correlation between S_i and weight of child

	Model 1 No Bandwidth	Model 2 15-day Bandwidth	Model 3 30-day Bandwidth
Weight	-186.641***	0.031	-0.005

	(0.58)	(0.07)	(0.13)
Cons	6038.381***	-0.628	-1.061
	(31.44)	(3.63)	(6.26)
R^2	0.633	0.002	0.000
N	60058	**101**	**196**

Notes: Results of an OLS regression of z-scores on weight and a constant. Standard deviations below parameter estimates. * denotes statistical significance at 10%, ** at 5%, and *** at 1%.

Table 4 Probit model of relevant observables for selection into treatment for various bandwidths

1 Jan 1999	Model 1 15 days	Model 2 20 days	Model 3 25 days	Model 4 30 days	Model 5 35 days
Ln(income pc)	0.0490	-0.0351	-0.0946	-0.0634	-0.0756
	(0.195)	(0.171)	(0.151)	(0.136)	(0.127)
D health	0.485	0.0719	0.138	-0.335	-0.306
	(0.932)	(0.767)	(0.736)	(0.616)	(0.611)
Age in months	-0.00270	**-0.00534***	**-0.00843*****	**-0.00882*****	**-0.0101*****
	(0.00373)	**(0.00319)**	**(0.00264)**	**(0.00243)**	**(0.00236)**
Mother's schooling	-0.0157	-0.0191	-0.000929	-0.00967	0.00419
	(0.0448)	(0.0405)	(0.0345)	(0.0309)	(0.0289)
D female	-0.0258	-0.0240	-0.190	-0.306	**-0.364***
	(0.291)	(0.250)	(0.223)	(0.207)	**(0.196)**
D indigenous	-0.502	-0.313	0.0411	0.201	0.190
	(0.569)	(0.556)	(0.436)	(0.414)	(0.412)
D afro-ecuadorian	-0.0558	-0.383	-0.399	-0.237	-0.250
	(0.996)	(0.969)	(1.015)	(0.616)	(0.624)
D montubio	0.428	0.611	0.950	0.987	0.414
	(0.802)	(0.791)	(0.790)	(0.787)	(0.655)
D Quito	-0.192	-0.833	-0.618	-0.473	-0.616
	(0.901)	(0.638)	(0.514)	(0.503)	(0.454)
D Rural	0.140	-0.0704	-0.0519	0.0723	0.0834
	(0.373)	(0.305)	(0.265)	(0.238)	(0.225)
D food	0.489	0.285	0.272	0.527	0.520
	(0.955)	(0.732)	(0.699)	(0.698)	(0.695)
D malnutrition	**-1.102****	**-0.957****	**-1.178*****	**-1.119*****	**-0.979*****
	(0.545)	**(0.475)**	**(0.427)**	**(0.400)**	**(0.379)**
z-score	-0.278	-0.297	**-0.349****	**-0.313****	**-0.307****
	(0.200)	(0.186)	**(0.158)**	**(0.143)**	**(0.138)**
N	**87**	112	146	**172**	193

Notes: Standard deviations below parameter estimates. * denotes statistical significance at 10%, ** at 5%, and *** at 1%.

Figure 6 Scatter and local polynomial with confidence intervals relation between running variable and weight bandwidth of 5000 days

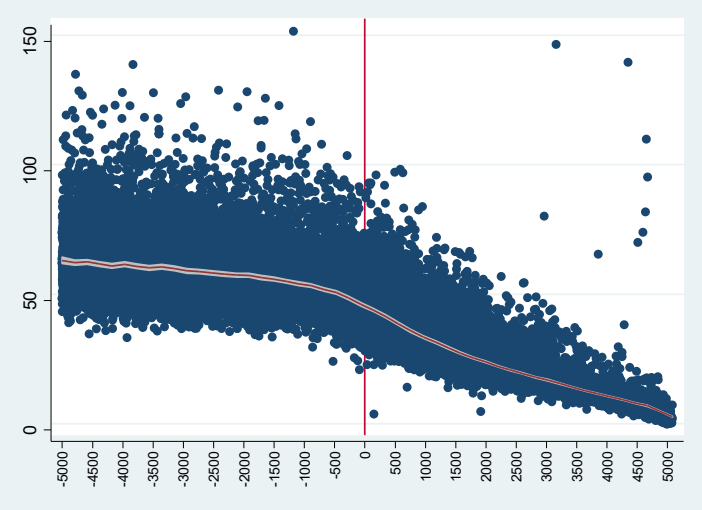
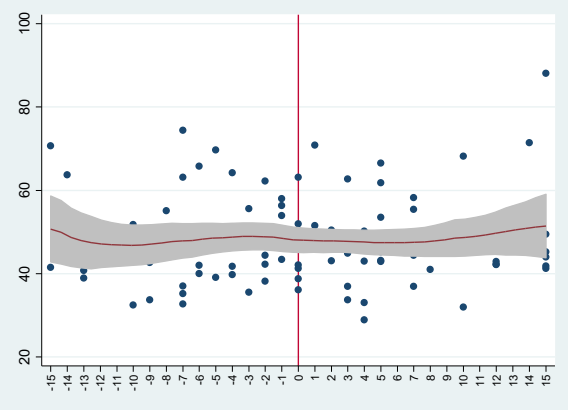
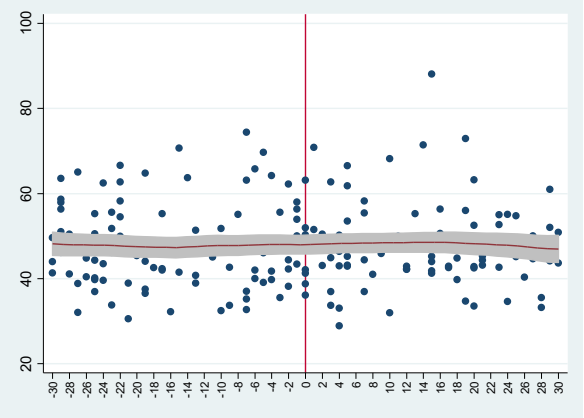


Figure 7 Relation between running variable and weight. Scatter and local polynomial with confidence intervals.



(a) 15-day bandwidth



(b) 30-day bandwidth

6.2. Choosing the correct functional form

A polynomial of order one, i.e. a linear functional form, may lead to an inaccurate jump at the cut-off given its lack of flexibility. A higher order polynomial can increase accuracy by increasing flexibility. However, it may also increase the variability of the treatment effect estimator. Cattaneo at al. (2018) recommend the linear estimation because it is the best tradeoff between simplicity, precision, and stability. We present

linear, quadratic and cubic models because, in finite samples, the ranking between different local polynomial estimators may differ from the asymptotic characteristics obtained in very large samples.

In this section, we apply two formal tests to guide us on the choice of polynomial, as recommended by Lee and Lemieux (2010). The first one is the Akaike information criterion (AIC),[22] see Table 5. We present the AIC for linear, quadratic, and cubic models and for our two selected bandwidths: 15 and 30 days. The results seem to indicate that the cubic model is the recommended functional form for the 30-day bandwidth while the quadratic form is recommended for the 15-day model (see Appendix 5 for the AIC test over various bandwidths).

Table 5 AIC for various bandwidths and polynomial orders

Bandwidth	Polynomial Order	Treatment effect	AIC
15	1	-0.82**	298.7
15	**2**	**-1.94*** **	**293.7**
15	3	-1.6**	297.1
30	1	-0.103	599.37
30	2	-0.895**	595.34
30	**3**	**-1.68*** **	**594.30**

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

The second test consists in including a series of bin dummies in the linear and non-linear models in order to see if there are significant jumps outside of the cut-off. Any significance in a bin dummy would signal a lack of flexibility of the polynomial order in terms of describing the behavior of the data. If there are significant dummies, we increase the polynomial order until all bin dummies are not significant (Lee & Lemieux, 2010).

We created bin dummies separately for each side of the bandwidth. We test various bin numbers (see discussion in Appendix 6) from 4 bin to 16 bins for each bandwidth. In all the specifications, be it linear, quadratic, or cubic, none of the bin dummies are significant. In other words, the number of bins, and therefore, the number of observations in the bins, do not seem to influence the results: the bin dummies are not significant; therefore, the models seem to be flexible enough to capture the behavior of the observations around the cut-off.

[22] $AIC = -2logL(\tilde{\Psi}) + 2n$ where $L(\tilde{\Psi})$ is the maximum value of the likelihood function and $2n$ is the number of parameters in the model.

We report on the 8-bin model for the 15-day bandwidth as it seems to be the intermediate level between number of bins and number of observations within each bin (see Appendix 6). We present the 16-bin model for the 30-day bandwidth as we have more observations and therefore can increase the number of bins. In Table 6, we present descriptive statistics of our bin dummies in relation to S_i. We can see that for the 15-day bandwidth we have approximately 10 observations (except in bin 5) in each bin, and for the 30 day bandwidth we have just over 10. Figures 8 and 9 provide and illustration of the bins.

Tables 8 and 9 show the RD models with the bin dummies for the 15 day and 30-day bandwidth respectively. As mentioned above, none of the bin dummies are significant (see Appendix 6 for additional models) in either the 15- or the 30-day bandwidth. Additionally, the treatment effect is not significant in any of the models. We suppose that the dummies are washing away the effect of the cut-off. In any case, this indicates that there are no jumps outside of the cut-off which may be affecting the ability of the polynomial specification to capture the behavior of the data. Therefore, taking both the AIC and the bin dummies into account we conclude that the quadratic model is probably the better fit for the 15-day model and the cubic model is probably the best fit for the 30-day model.

Table 6 Number of observations (15-day bandwidth, 8 bin)

Bins	Obs.	Min	Max
1	12	-15	-9
2	12	-8	-6
3	9	-5	-3
4	10	-2	-1
5	20	0	3
6	12	4	5
7	13	7	10
8	13	11	15

Table 7 Number of observations (30-day bandwidth, 16 bins)

Bins	Obs.	Min	Max
1	15	-30	-27
2	10	-26	-25
3	13	-24	-22
4	15	-21	-17
5	8	-16	-11
6	14	-10	-7
7	10	-6	-4
8	12	-3	-1
9	14	0	2
10	12	3	4
11	13	5	7
12	11	8	13
13	13	14	17
14	14	18	21
15	11	22	25
16	11	26	30

Figure 8. Eight bins before and after cut-off for 15-day bandwidth

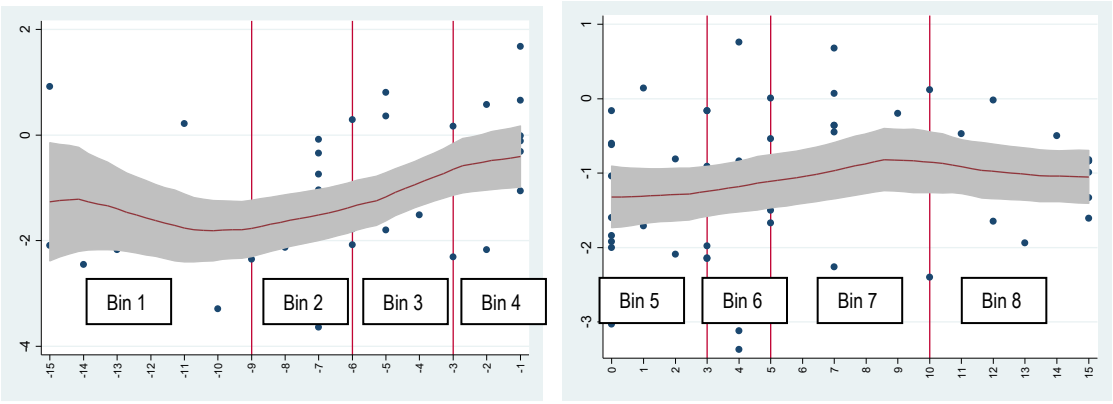


Figure 9. Sixteen bins before and after cut-off for 30-day bandwidth

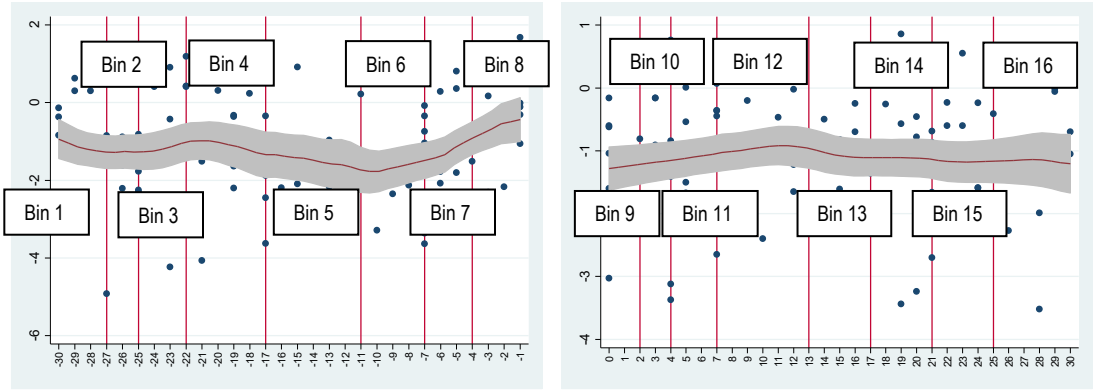


Table 8 Regression discontinuity (15-day bandwidth, various polynomial forms, OLS regression) 8 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
DTreat	2.155	-0.240	-0.293
	(2.17)	(3.15)	(3.40)
Running	-0.144	0.332	0.650
	(0.12)	(0.47)	(0.90)
DTreat*Running	0.153	-0.248	-0.547
	(0.17)	(0.51)	(0.94)
$Running^2$		0.022	0.074
		(0.02)	(0.13)
$DTreat*Running^2$		-0.028	-0.084
		(0.02)	(0.14)
$Running^3$			0.002
			(0.01)
$DTreat*Running^3$			-0.002
			(0.01)
1bn.cbin	.	.	.
	.	.	.
2.cbin	0.686	0.506	0.776

	(0.73)	(0.75)	(0.99)
3.cbin	1.876	1.081	1.490
	(1.03)	(1.28)	(1.62)
4.cbin	2.930*	1.129	1.299
	(1.35)	(2.19)	(2.25)
5.cbin	-0.153	-0.229	-0.340
	(1.47)	(1.49)	(1.93)
6.cbin	-0.144	-0.366	-0.467
	(1.14)	(1.23)	(1.65)
7.cbin	0.283	0.041	0.001
	(0.77)	(0.92)	(1.03)
8.cbin	.	.	.
	.	.	.
_cons	-3.307*	-0.916	-0.760
	(1.46)	(2.71)	(2.77)
R^2	0.160	0.173	0.174
N	100	100	100

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

Table 9 Regression discontinuity (30-day bandwidth, various polynomial forms, OLS regression) 16 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
DTreat	-2.154	-3.635	-3.070
	(4.26)	(4.28)	(4.29)
Running	0.084	0.572*	0.327
	(0.11)	(0.24)	(0.40)
DTreat Running	-0.103	-0.681*	-0.085
	(0.15)	(0.32)	(0.51)
$Running^2$		0.015*	-0.005
		(0.01)	(0.03)
DTreat $Running^2$		-0.012	-0.025
		(0.01)	(0.04)
$Running^3$			-0.000
			(0.00)
DTreat $Running^3$			0.001
			(0.00)
1bn.cbin	.	.	.
	.	.	.
2.cbin	-0.971	0.118	0.370
	(0.56)	(0.73)	(0.80)
3.cbin	-0.049	1.604	1.859
	(0.73)	(1.02)	(1.07)
4.cbin	-1.230	1.011	1.043
	(1.14)	(1.50)	(1.50)
5.cbin	-1.466	0.769	0.437
	(1.65)	(1.90)	(1.95)
6.cbin	-2.701	-1.369	-1.949
	(2.23)	(2.28)	(2.40)
7.cbin	-1.856	-1.388	-1.869
	(2.55)	(2.53)	(2.60)
8.cbin	-1.600	-2.436	-2.495
	(2.90)	(2.90)	(2.89)
9.cbin	-0.537	-0.592	-1.050
	(2.93)	(2.90)	(2.91)
10.cbin	-0.563	-0.387	-1.520
	(2.63)	(2.63)	(2.73)
11.cbin	0.019	0.351	-0.979
	(2.36)	(2.43)	(2.59)
12.cbin	0.027	0.530	-0.576
	(1.92)	(2.14)	(2.27)
13.cbin	0.092	0.656	0.252
	(1.41)	(1.79)	(1.81)
14.cbin	-0.238	0.251	0.470

	(0.99)	(1.38)	(1.38)
15.cbin	0.410	0.742	1.215
	(0.68)	(0.94)	(0.99)
16.cbin	.	.	.
	.	.	.
_cons	1.390	2.975	2.698
	(3.06)	(3.11)	(3.12)
R2	0.136	0.162	0.175
N	195	195	195

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

6.3. Choosing a Kernel Function

The kernel function assigns weights to each observation based on its distance to the cut-off point as expressed by S_i. The triangular kernel function assigns zero weights to all observations outside of the selected bandwidth, and positive weights to all observation inside it. This makes the weight reach its maximum at the cut-off point and decrease progressively as we move further away from it. The uniform kernel gives equal weights to all observations within the bandwidth and the Epanechnikov kernel gives quadratic decaying weights to observation within the bandwidth. As Table 10 shows, our estimations are not very sensitive to the choice of kernel weights as they are all significant in a similar way within bandwidth and functional forms and across kernel functions. For example, the quadratic models with a 15-day bandwidth are significant and have a magnitude of approximately 1.8 across all kernel functions, while the linear model with a 30-day bandwidth is not significant no matter what kernel specification we use. In the remainder of the paper, we present the regressions using the triangle kernel (see Appendix 6 for results with various bandwidths).

Table 10. Average treatment effect with 15- and 30-day bandwidth and various choices of kernel functions

Kernel function	Bandwidth	Linear	Quadratic	Cubic
Uniform	15	-0.82**	-1.9***	-1.60*
Triangle	15	-1.6*	-1.79***	-1.39***
Epanechnikov	15	-1.3***	-1.8***	-1.59*
Uniform	30	-0.10	-0.89**	-1.68***
Triangle	30	-0.40	-1.27**	-1.94***
Epanechnikov	30	-0.28	-1.15***	-1.95***

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

6.4. Average Treatment Effect

Table 11 shows the Average Treatment Effect (ATE) for different bandwidths (between 15 and 30 days) and functional forms (polynomial of order zero to two). The ATE is negative and significant for all bandwidths and functional forms except one: the 30-day linear model. However, our results are most accurate in the quadratic model. Notwithstanding, the significance of the effect across all but one specification is an indication of a robust effect. It is worth mentioning that the variability in the ATE seems to increase with the bandwidth and decreases with polynomial order (Figure 9). The 15-day bandwidth produces lowest variability in the ATE (-1.3 to -1.6) while the 30 day bandwidth produces the highest (-0.41 to -1.94). We can see that the linear model produces the highest variability (between -1.3 to -0.41) while the cubic model produces the lowest variability (-1.6 to -1.9).

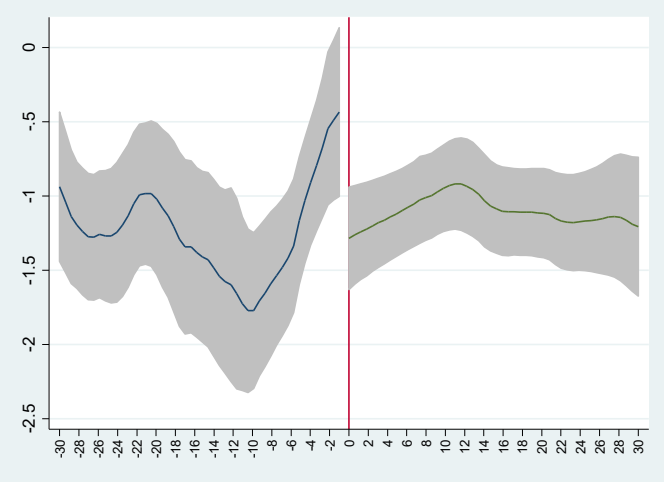
Importantly, the results indicate that the data seems to have an imbalance in the sample on the left as compared to the right of the cut-off. For a bandwidth of 15 days, for instance, we have 40 observations on the left of the cut-off and 52 observations on the right. We discuss this issue further in the next section.

Table 11 Average Treatment Effect and Frequency counts at either side of the cut-off, by Bandwidth and Functional Form

	Observations		ATE Estimate		
Bandwidth	left of cut-off	right of cut-off	Linear	Quadratic	Cubic
15	40	52	-1.3***	-1.79***	-1.65*
20	55	68	-0.94***	-1.76***	-1.83***
25	71	86	-0.57*	-1.62***	-1.89***
30	93	97	-0.41	-1.27***	-1.94***

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

Figure 9 Local polynomial on each side of jump: 1 Jan 1999
(bandwidth is 30 days, polynomial order is 0, and kernel is Epanechnikov)



7. Robustness checks

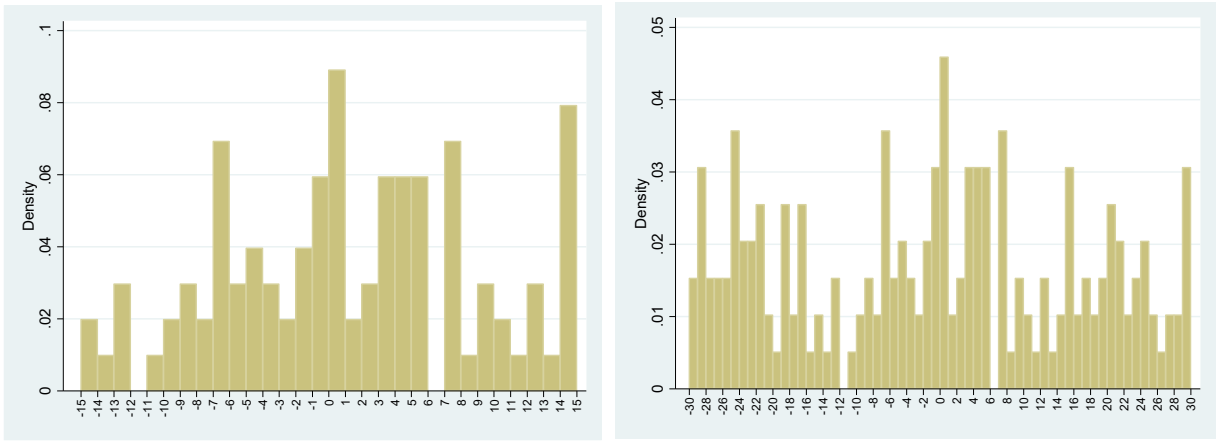
In the previous section we have discussed (i) predetermined covariates, (ii) the choice of bandwidth, (iii) the choice of functional form, and (iv) the choice of kernel function. This section reviews four robustness checks, as proposed by Cattaneo at al. (2018) and Lee and Lemieux (2010): (1) the density of the running variable (manipulation of the cut-off), (2) the sensitivity of observations near the cut-off, (3) placebo effects or anticipation bias, (4) the covariates with the same cut-off.

7.1. Density of the running variable (manipulation of the cut-off)

A basic principle of the RD model is that individuals are unable to determine which side of the cut-off they fall into, that is, they are unable to manipulate S_i which determines treatment. If this is true, the number of observations just above and below the cut-off should be similar. In our case, there are slightly more observations just above the cut-off (58) than just below (43). This is a concern because it reduces the credibility of a random assignment to treatment. The condition of equal sample size is not necessary or sufficient to an RD model (Cattaneo, et al., 2018), but it does lead to the question of whether it was possible for individuals to manipulate S_i. In Figure 10 we can see the distribution of the sample across S_i. There is a notable increase in observations of individuals born on the day of the crisis i.e. on 1 Jan 1999.

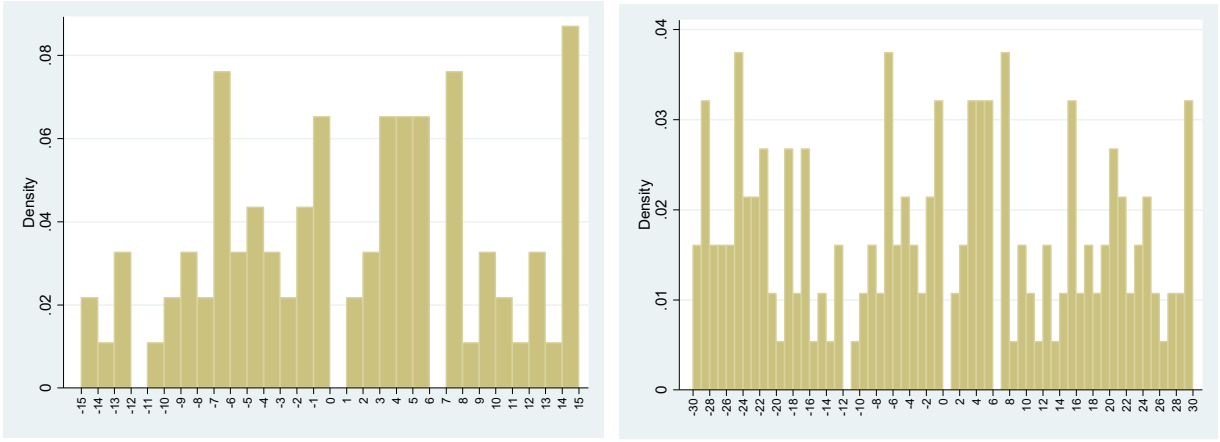
When we exclude the individuals who were born on 1 Jan 1999, Figure 11 shows that the distribution of S_i is much more uniform, with a more balanced assignment to treatment (treat:49, control: 43) (see Table 12 for totals across groups and dates).

Figure 10 Density of the running variable days born before/after 1 Jan 1999



(a)15-day bandwidth (b) 30-day bandwidth

Figure 11 Density of running variable days born before/after 1 Jan 1999 excluding observations at zero



(a) 15-day bandwidth (b) 30-day bandwidth

Table 12 Control and Treatment group frequencies with and without 1 Jan 1999, by bandwidth

Sample	Bandwidth	Control	Treatment	Total
Entire sample	15	43	58	101
Sample excluding 1 Jan 1999	15	43	49	92
Entire sample	30	97	99	196
Sample excluding 1 Jan 1999	30	97	90	187

In order to see if there is a discontinuity in the frequency or density of the assignment variable we use two related methods, as proposed by McCrary (2008). The first method involves a two-step process where, firstly, the assignment variable is partitioned into equally spaced bins, days in our case, and then, the frequency count by day is predicted with a local polynomial regression. Figures 12 and 13 show the McCrary smoothened histogram using a 15- and 30-day bandwidth, respectively. Panel (a) in both Figures include all observations within each bandwidth, while panel (b) exclude children born on 1 Jan 1999. In any of these four cases, the density of the running variable presents no discontinuity, as all jumps fall within confidence intervals. The clearest case of no discontinuity is when children born on 1 Jan are excluded and the bandwidth is 30 days.

A second way of checking for a discontinuity in the density of the assignment variable is using the frequency counts of the assignment variable as dependent variable in an RD model. Table 13 shows the estimates of the RD models for various bandwidths, functional forms, and samples —with or without 1 Jan 1999. At odds with the evidence from Figures 12 and 13, Table 13 shows that the discontinuity in the density of the assignment variable is significant for some models. Although the discontinuity is only significant in half of the models, this seems to present some partial evidence of a jump in the density. We further investigate this issue, in the next section, where we test the sensitivity of the outcome and results to the observations near the cut-off.

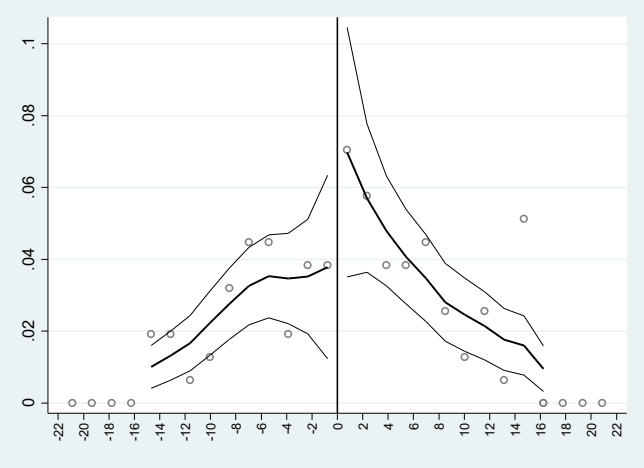
Table 13 Regression discontinuity model of frequency (density) of assignment variable using day of crisis as cut-off (Kernel=triangle)

Dependent variable: frequency count of assignment variable

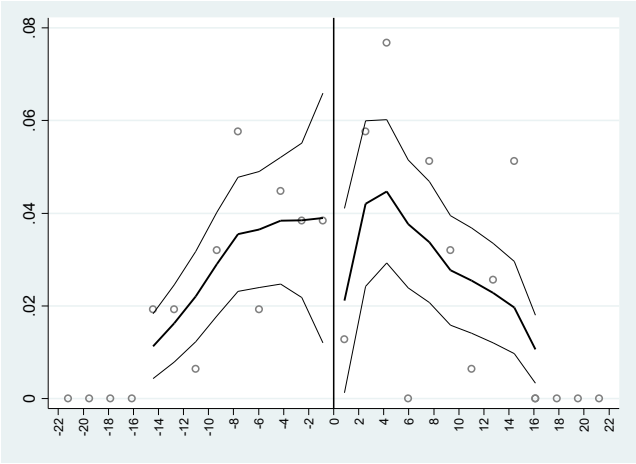
Sample	Bw	Left	Right	Linear	Quadratic	Cubic
Whole Sample	15	41	52	**2.34*** **	2.39	0.72
Sample excluding 1 Jan 1999	15	41	43	0.107	**-3.97***	**-9.31*** **
Whole Sample	30	94	97	**2.33*** **	**2.07***	2.27
Sample excluding 1 Jan 1999	30	94	88	**1.07***	-0.105	-1.73

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

*Figure 12 McCrary smoothened histogram of frequency counts in S_i with a **15-day bandwidth***

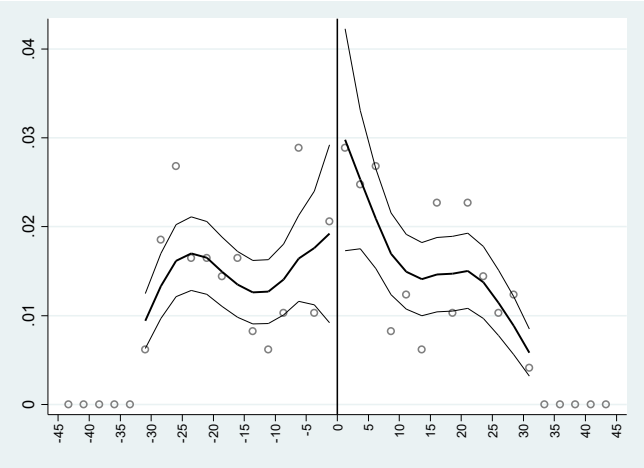


(a) Including all observations

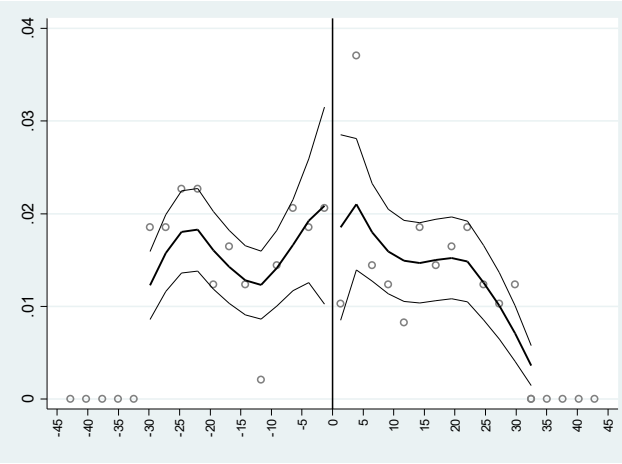


(b) Excluding 1 Jan 1999

*Figure 13 McCrary smoothened histogram of frequency counts in S_i with a **30-day bandwidth***



(a) Including all observations



(b) Excluding 1 Jan 1999

7.2. Sensitivity of observations near the cut-off

It is difficult to explain how or why an individual would want to wait until 1 Jan to give birth or why an individual would mislead the census taker regarding the date of birth of their children. Perhaps individuals who gave birth in difficult, isolated conditions round down the birthdate of their children in order to comply with the 30-day registration limit of newly born infants. This is unlikely because registration of infants is free for all children even after this date has passed and all the way up to the age of 18 (years of age).[23] We do not find any evidence of financial rewards given to the first-born children of the year or of any media attention provided to these children. In any case, we address this potential problem by measuring the sensitivity of the model to the observations around the cut-off.

The idea of this method is to exclude individuals near the cut-off and to repeat the estimation with the remaining sample (Cattaneo et al., 2018). Table 14 presents the estimates of interest of the RD models for the different bandwidths and polynomial order, including and excluding 1 Jan 1999. The estimates are robust to excluding children born on 1 Jan for our preferred models (i.e. quadratic specification with 15-day bandwidth and cubic specification with 30-day bandwidth). It is also worth noting that the ATE estimates are significant in similar ways across samples. For example, the linear 15-day model is negative and significant for both the whole sample and that which excludes children born on 1 Jan. Only in the cubic 15-day model the estimate is significant for the whole sample and not significant when children born 1 Jan are excluded. This may indicate that the effect of the observations near the cut-off is important when the sample size is small, i.e. in the 15-day model, which in turn may be an argument in favor of a larger sample size.

Table 14 Sensitivity of RD model to observations near the cut-off
(15- and 30-day bandwidths, and triangle kernel function)

Sample	Bandwidth	Left	Right	Linear	Quadratic	Cubic
Whole Sample	15	40	52	-1.39***	-1.79***	-1.65*
Sample excluding 1 Jan 1999	15	40	43	-1.33***	-1.71**	**-0.86**
Whole Sample	30	93	97	**-0.41**	-1.27***	-1.94***
Sample excluding 1 Jan 1999	30	93	88	**-0.29**	-1.2**	-1.96***

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

[23] https://www.registrocivil.gob.ec/nacimientos/

7.3. Placebo effects and anticipation bias

We present four placebo effect tests. (i) Anticipation bias, (ii) placebo effects after the crisis; (iii) New Year's Day effect; and (iv) a sub-sample of individuals with no access to financial services and who should not have been affected by the bank run. The first three are placebo effects based on alternative cut-offs of the assignment variable S_i. The last placebo effect is a group of children born into the treatment group who should, theoretically, not have been affected by the crisis. A sort of post-crisis control group.

7.3.1. Measuring placebo effects by changing S_i

The three placebo effects are described in Table 15. We can clearly see that there are no placebo effects except on two isolated models: 1 Feb 1999 cubic specification, and 1 Apr 1999 linear specification (note we present four different bandwidths for every model in order to establish robustness).

7.3.1.1. Anticipation bias & placebo effects after the crisis

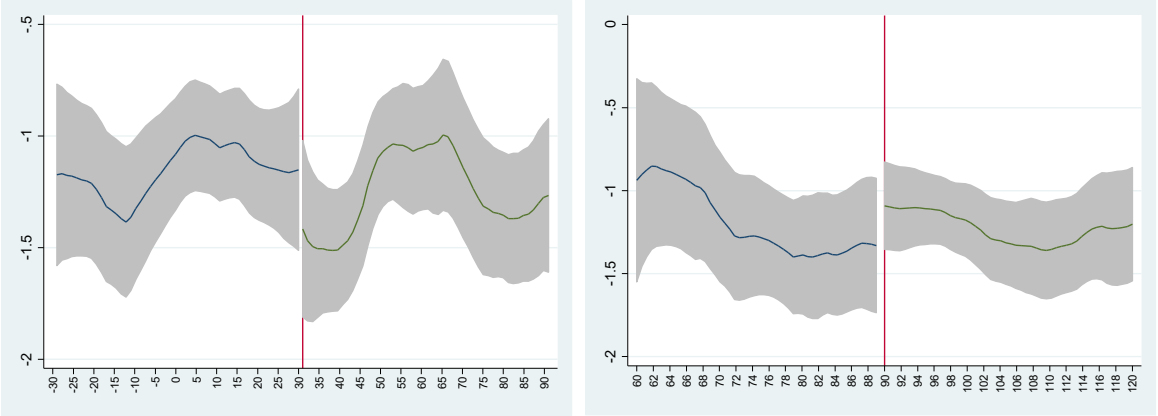
Firstly, we emphasize that we find no significant effects on the outcome variable in the months running up to the crisis. This is important because it excludes any anticipation bias of the crisis. Given the context described above it would have been difficult for individuals to anticipate the collapse of the financial system. These robustness checks are an important piece of empirical evidence in favor of our hypothesis. We do find two significant effects after the crisis, on 1 Feb and 1 April 1999, represented in Figure 14. We plot separate local polynomial regression on each side of the placebo cut-off and find that in neither case is the jump outside of the confidence interval. We argue that the effects are found only when using a specific bandwidth and polynomial order which is insufficient to prove an exogenous effect. This is why we do not see a graphic representation of an effect in Figure 14 (a) or (b).

Table 15 Placebo effects before and after the crisis

Year	Day/Month	Bandwidth	Linear	Quadratic	Cubic	Obs. left of cut-off	Obs. right of cut-off
1998	1-Dec	15	0.04	0.33	-0.72	37	56
1998	1-Dec	20	-0.12	0.28	0.33	49	63
1998	1-Dec	25	-0.13	0.008	0.46	61	78
1998	1-Dec	30	-0.15	-0.01	0.2	68	93
1998	1-Nov	15	0.92	1.26	0.28	43	29
1998	1-Nov	20	0.69	1.2	1.18	62	45
1998	1-Nov	25	0.46	1.08	1.3	80	60
1998	1-Nov	30	0.36	0.89	1.2	98	70
1998	1-Oct	15	-0.61	-0.83	-0.81	46	55
1998	1-Oct	20	-0.63	-0.61	-1.0	65	75
1998	1-Oct	25	-0.63	-0.68	-0.59	87	95
1998	1-Oct	30	-0.59	-0.7	-0.6	104	105
1998	1-Sep	15	-0.38	-0.24	0.8	57	55
1998	1-Sep	20	-0.36	-0.39	0.10	79	71
1998	1-Sep	25	-0.26	-0.26	-1.18	103	83
1998	1-Sep	30	-0.15	-0.51	-0.46	125	106
1998	1-Aug	15	-0.21	-0.22	0.02	53	65
1998	1-Aug	20	-0.12	-0.25	-0.12	75	99
1998	1-Aug	25	-0.12	-0.22	-0.27	98	112
1998	1-Aug	30	-0.13	-0.15	-0.22	117	131
1999	1-Feb	15	-0.26	-0.32	-1.19*	39	57
1999	1-Feb	20	-0.28	-0.27	-0.55	53	75
1999	1-Feb	25	-0.31	-0.26	-0.34	67	92
1999	1-Feb	30	-0.34	-0.23	-0.34	88	106
1999	1-Mar	15	0.21	0.80	0.53	49	42
1999	1-Mar	20	0.18	0.44	0.90	71	65
1999	1-Mar	25	0.12	0.34	0.77	87	80
1999	1-Mar	30	0.11	0.20	0.60	105	91
1999	1-Apr	15	0.28	0.34	1.5	39	77
1999	1-Apr	20	0.35	0.20	0.70	60	96
1999	1-Apr	25	0.37	0.25	0.28	76	112
1999	1-Apr	30	0.42*	0.26	0.26	89	139
1999	1-May	15	0.33	0.39	0.05	61	60
1999	1-May	20	0.32	0.34	0.34	85	78
1999	1-May	25	0.35	0.29	0.36	109	93
1999	1-May	30	0.41	0.25	0.32	134	113
1999	1-Jun	15	0.22	0.21	0.37	56	80
1999	1-Jun	20	0.23	0.29	0.12	75	99
1999	1-Jun	25	0.19	0.28	0.20	92	119
1999	1-Jun	30	0.15	0.28	0.28	112	139

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

Figure 14 Local kernel on each side of placebo jump (bw is 30 days, polynomial order is 0, Epanechnikov kernel)



(a) 1 Feb 1999 (b) 1 April 1999

We run a probit model to measure the observable differences between the placebo treatment and control groups in both the placebo effects for 1 Feb 1999 and 1 Apr 1999. As above, this will help us determine if the samples are similar in their observable characteristics. As we can see in Tables 16 and 17, neither 1 Feb 1999 nor 1 Apr 1999 effects are driven exclusively by the treatment. In the former, the schooling of the mother is significantly higher among the treated, and in the latter, the proportion of children living in Quito is significantly lower among the treated. Taking the lack of a consistent effect across polynomial forms into consideration, along with the lack of a visible jump in the local polynomial regressions, as well as the significant difference in observable characteristics in all bandwidths, we suggest this placebo effect does not hold up to robustness checks.

Table 16 Probit dummy treatment using 1 Feb 1999 as placebo cut-off, measuring effect of observables

1 Feb 1999	15 days	20 days	25 days	30 days
	P1	P2	P3	P4
Ln(income pc)	-0.00992	-0.0146	0.0223	-0.0167
	(0.213)	(0.194)	(0.177)	(0.152)
D health	.	0.620	0.683	0.850
	.	(0.933)	(0.920)	(0.914)
Age in months	-0.00668	**-0.00866****	**-0.0112*****	**-0.0142*****
	(0.00430)	**(0.00364)**	**(0.00336)**	**(0.00295)**
Mother's schooling	**0.0849****	**0.0875****	**0.0763****	**0.0850*****
	(0.0416)	**(0.0386)**	**(0.0337)**	**(0.0326)**
D female	0.225	0.202	0.0878	-0.0543
	(0.315)	(0.279)	(0.252)	(0.220)
D indigenous	0.0166	-0.0211	0.0307	0.0127
	(0.516)	(0.476)	(0.445)	(0.422)
D afro-ecuadorian	.	.	-1.006	-1.062
	.	.	(0.908)	(0.920)
D Montubio	.	.	**1.323****	**1.511****
	.	.	**(0.672)**	**(0.680)**
D Quito	-0.642	-0.782	-0.520	-0.451
	(0.652)	(0.630)	(0.558)	(0.467)
D Rural	-0.136	-0.128	-0.180	-0.103

	(0.343)	(0.305)	(0.283)	(0.255)
D food	0.0368	-0.366	-0.188	-0.0797
	(0.891)	(0.751)	(0.703)	(0.706)
D malnutrition	0.700	1.036*	0.455	0.0189
	(0.659)	(0.589)	(0.524)	(0.456)
z-score	-0.168	-0.0327	-0.271	**-0.393****
	(0.280)	(0.236)	(0.206)	**(0.184)**
N	80	103	133	167

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

Table 17 Probit dummy treatment using 1 Apr 1999 as placebo cut-off, measuring effect of observables

1 Apr 1999	15 days	20 days	25 days	30 days
	P1	P2	P3	P4
Ln(income pc)	0.313	0.121	0.198	**0.237***
	(0.194)	(0.167)	(0.157)	**(0.143)**
D health	-0.396	-0.170	-0.308	0.0745
	(0.660)	(0.648)	(0.536)	(0.425)
Age in months	**-0.0140*****	**-0.0135*****	**-0.0155*****	**-0.0159*****
	(0.00401)	**(0.00314)**	**(0.00289)**	**(0.00270)**
Mother's schooling	-0.0180	-0.000944	-0.0205	-0.0190
	(0.0411)	(0.0370)	(0.0346)	(0.0320)
D female	0.110	0.123	0.0441	0.142
	(0.295)	(0.246)	(0.222)	(0.203)
D indigenous	-0.299	0.0412	-0.0510	0.0974
	(0.437)	(0.390)	(0.384)	(0.360)
D afro-ecuadorian	-0.123	-0.149	-0.0276	0.212
	(0.658)	(0.636)	(0.621)	(0.575)
D montubio	0.657	0.456	0.115	-0.0179
	(1.010)	(0.648)	(0.550)	(0.519)
D Quito	**-1.706****	**-1.185***	**-1.439*****	**-1.514*****
	(0.758)	**(0.639)**	**(0.530)**	**(0.479)**
D Rural	0.373	0.0374	0.118	0.0965
	(0.325)	(0.269)	(0.248)	(0.224)
D food	.	.	.	.
	.	.	.	.
D malnutrition	0.448	0.376	-0.0616	-0.100
	(0.585)	(0.468)	(0.396)	(0.368)
z-score	0.188	0.0997	-0.0959	-0.146
	(0.268)	(0.215)	(0.169)	(0.155)
N	102	141	169	200

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

7.3.1.2. New Year's Day effect

We measure the effect on New Year's Day in the years preceding the crisis (1994-1998) in Table 18. We find no significant effect except for in the 25-day bandwidth cubic specification in 1995. Figure 15 represents this placebo effect graphically. There is no clear jump in the outcome variable outside of the confidence interval on 1 Jan 1995. This indicates there is no robust evidence of an unobservable "New Year Day" effect which might affect our outcomes.

Table 18 Placebo New Year's Day effect

Year	Day/Month	bandwidth	Linear	Quadratic	Cubic	Obs. left of cut-off	Obs. right of cut-off

1998	1-Jan	15	-0.26	-0.09	0.17	45	52
1998	1-Jan	20	-0.25	-0.22	0.01	57	65
1998	1-Jan	25	-0.30	-0.19	-0.12	72	78
1998	1-Jan	30	-0.11	-0.19	-0.56	56	61
1997	1-Jan	15	-0.37	-0.38	-0.92	36	36
1997	1-Jan	20	-0.37	-0.42	-0.39	46	49
1997	1-Jan	25	-0.28	-0.47	-0.40	54	63
1997	1-Jan	30	0.01	-0.14	-0.93	66	55
1996	1-Jan	15	0.08	0.55	0.65	43	22
1996	1-Jan	20	-0.02	0.32	0.58	60	22
1996	1-Jan	25	-0.07	0.14	0.51	70	43
1996	1-Jan	30	-0.11	0.10	0.33	79	56
1995	1-Jan	15	-0.37	-1.08	-0.56	35	24
1995	1-Jan	20	-0.08	-0.79	-0.97	44	35
1995	1-Jan	25	0.03	-0.48	-1.08*	56	45
1995	1-Jan	30	-0.22	-0.44	-0.46	69	72
1994	1-Jan	15	-0.30	-0.54	-0.34	27	34
1994	1-Jan	20	-0.14	-0.51	-0.47	39	48
1994	1-Jan	25	-0.11	-0.32	-0.69	47	53
1994	1-Jan	30	-0.26	-0.31	-0.07	83	96

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

Figure 15 Local polynomial on both sides of placebo cut off: 1 Jan 1995 (bw=30, polynomial=0, kernel=Epanechnikov)

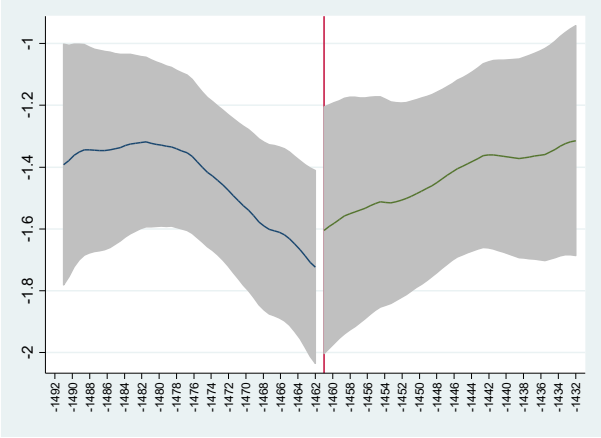


Table 19 presents various probit models of the dummy treatment using 1 Jan 1995 as a cut-off point. We can see that there are no observable characteristics that are significantly different between treatment and control groups. However, it is important to highlight the number of observations for a 15-day bandwidth is relatively small (40 for both treatment and control). This sample may be too small to have sufficient hypothesis testing power. Additionally, most of the control variables are dropped. Once the sample starts to approximate a similar size (79 for the 30-day bandwidth) to those used in our main model (87 for 15 day bandwidth and 172 for the 30 day bandwidth) we find a significantly higher amount of indigenous children in the treatment group. The relatively small sample size coupled with the lack of a consistent effect over other sample sizes and polynomial forms leads us to conclude that there is insufficient evidence of a

placebo effect on this date. Furthermore, the lack of an effect across various New Year's Days leads us to believe there is no unobservable driver on New Year's producing the effect of the crisis in 1999.

Table 19 Probit dummy treatment using 1 Jan 1995 as placebo cut-off, measuring effect of observables

1 Jan 1995	15 days	20 days	25 days	30 days
	P1	P2	P3	P4
Ln(income pc)	0.234	-0.00412	-0.0219	-0.0771
	(0.537)	(0.410)	(0.267)	(0.242)
D health	0.802	0.574	0.418	0.295
	(1.994)	(1.658)	(1.141)	(1.139)
Age in months	**-0.0289*** **	**-0.0248*** **	**-0.0177*** **	**-0.0187*** **
	(0.00915)	**(0.00709)**	**(0.00467)**	**(0.00433)**
Mother's schooling	-0.260	-0.158	-0.0608	-0.0377
	(0.182)	(0.115)	(0.0631)	(0.0558)
D female	-0.192	0.257	-0.0384	-0.117
	(0.796)	(0.569)	(0.420)	(0.401)
D indigenous	.	.	.	**1.776***
	.	.	.	**(0.959)**
D afro-ecuadorian	.	.	.	.
	.	.	.	.
D montubio	.	.	.	.
	.	.	.	.
D Quito	-1.472	-0.510	-0.711	-1.025
	(1.608)	(0.988)	(0.772)	(0.754)
D Rural	-0.211	0.167	0.469	0.315
	(0.765)	(0.650)	(0.445)	(0.401)
D food	.	.	.	.
	.	.	.	.
D malnutrition	1.552	0.606	0.435	0.485
	(1.056)	(0.758)	(0.580)	(0.542)
z-score	0.339	0.103	0.0830	0.0413
	(0.714)	(0.470)	(0.314)	(0.293)
N	40	54	70	79

* denotes statistical significance at 10%, ** at 5%, and *** at 1%

7.3.2. Individuals with no access to banking services as placebo

The effect of a bank run on individuals who have no access to financial services would be indicative of a non-observable driving the effect. We do not have information on whether the parents had access to banking services. However, we are able to identify the parents who belong to the lower end of the income distribution. We define this as the first decile, which corresponds to households with a per capita income between $5.5 and $30 a month. The mean income in the first decile is $21.4 per capita per month when using a 90-day bandwidth (see Appendix 10 for descriptive statistics on the income distribution). We contend that they are less likely to have access to banking and financial services and test the effect of the bank run on this subgroup. Table 20 presents the sample sizes for treatment and control groups for different bandwidths. As we can see, the 90-day bandwidth already has a relatively small sample (Treat: 37, Control: 29) which is why we are unable to reduce the bandwidth further. Table 21 presents the ATE for this decile and shows there is no significant effect of the crisis on the sample of children in the first

decile. This is probably not because they did not have access to financial services, as, when we decompose the distribution into deciles and estimate the ATE within each decile (see Table 22) we do not find effect within any decile. This implies that the effect is driven by the variation between the deciles rather than the variation within them.

Table 20 Sample size of first quintile of income distribution by bandwidth

Bandwidth	Control	Treatment	Total
365	153	170	323
180	67	73	140
90	29	37	66

Table 21 RD model for first decile of income distribution with various bandwidths and functional forms

bw	N Control	N Treat	Linear	Quadratic	Cubic
365	161	166	0.07	-0.17	0.12
180	77	77	-0.01	0.38	0.38
90	32	34	0.22	0.39	-0.10

Table 22 RD models for 1 Jan 99 by deciles and quintiles 180 days bandwidths

Decile	N left	n right	Linear	Quadratic	Cubic
1	77	77	-0.01	0.38	0.38
2	82	87	-0.48	-0.75	-0.69
3	57	54	-0.12	-0.46	-0.71
4	56	70	0.08	0.15	0.11
5	61	71	0.34	0.40	0.001
6	68	71	0.27	0.4	0.5
7	42	70	0.2	0.74	0.07
8	54	66	-0.36	-0.1	-0.4
9	61	64	0.04	0.1	-0.09
10	67	54	-0.5	-0.51	-0.8

** denotes statistical significance at 10%, ** at 5%, and *** at 1%*

Does this mean there are differentiated effects for poorest and richest? Not exactly. We divide the sample into "poor" and "non-poor" by using the 2012 poverty line defined by INEC of $77 per capita per month. Table 23 shows that there is a strongly negative and significant effect on the "poor" in all bandwidths and functional forms except one while Table 24 shows there are some models which are significant among the non-poor. Of course, this exercise does not prove differentiated effects firstly because the sample sizes are very small when dividing the group into poor and non-poor, and secondly, because there is no consistent effect among the non-poor which makes it difficult to state what is actually happening within that sub-group. Obviously, demonstrating differentiated effects is not the objective of the paper, however, this subsection is here to demonstrate that the lack of an effect in the first decile does not imply a lack of an effect among the "poor." Additionally, it is important to state, in closing this section, that without specific

information on the household's access to banking services during the crisis, it is not possible to estimate the effect of the crisis on this sub-group. It would be interesting to explore this option if this information ever is recoded in the future.

Table 23 RD models for individuals under poverty line ($77 per capita per month) using 1 Jan 99 cut-off and various bandwidths and functional forms

Poor=$77pc	N left	N right	Linear	Quadratic	Cubic
15 day	39	43	-2.04***	-2.12***	-1.6
30 day	54	54	-1.12**	-1.9***	-2.41***

** denotes statistical significance at 10%, ** at 5%, and *** at 1%*

Table 24 RD models for individuals over poverty line ($77 per capita per month) using 1 Jan 99 cut-off and various bandwidths and functional forms

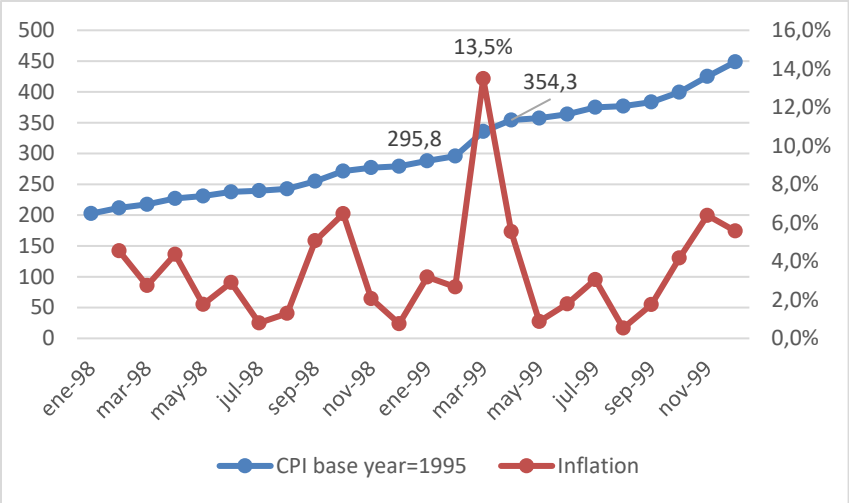
Non-poor	N left	N right	Linear	Quadratic	Cubic
15 day	39	43	-1.2***	-1.93*	-1.8
30 day	54	54	0.1	-0.91	-2.07**

** denotes statistical significance at 10%, ** at 5%, and *** at 1%*

7.4. Covariates with the same cut-off

It was also suggested that other baseline covariates might have experienced a jump on 1 Jan 1999. One particularly important variable would be the price level. During months running up to the crisis there was a non-negligible increase in prices which might also have created a shock through a reduction in the access to adequate nutrition. As we can see in Figure 16 there was an inflation shock in August 1998 (with a 5% hike in prices) and another in March 1999 (with an additional 14% hike in prices). None of these shocks happen simultaneously with the 1% tax or the bank run, and there does not seem to be a price shock which happened simultaneously, that is, which had the same cut-off as the bank run. This allows us to argue that, at least for the sample of children taken into consideration (those born 30 days before/after the crisis) a price shock was not driving the effect.

Figure 16. CPI and inflation 1998 - 1999



8. Conclusion and discussion

We find a significant deleterious effect of the outbreak of the 1999 financial crisis on the 2012 z-score of height-for-age of children born just before 1 Jan 1999 as compared to those born just after. This natural experiment finds an exogenous cut-off which allows us to measure the causal effects of the crisis on the health outcomes of children in the long run by using a sharp RD model.

The unanticipated financial crash is understood as an objective stress shock exposing unborn children to pre-natal maternal stress. The resulting change in the fetal environment can cause alterations in the series of "switches" which determine whether parts of a genome are expressed or not, such that, the health effects of an intra-uterine shock may remain latent though the life cycle (Almond & Currie, 2011).

Throughout this paper we provide evidence of a robust unanticipated effect. We justify the exogeneity of the sample by demonstrating that relevant observable characteristics are not significant determinants of selection into treatment. We use a data-driven method to select an appropriate bandwidth, we use the Akaike Information Criterion (AIC) as well as a dummy variable test in order to select the polynomial order, and, we test the sensitivity of the results to kernel functional forms (Cattaneo, et al., 2018; Lee & Lemieux, 2010). Additionally, we test for placebo effects in the months and years predating and following the crisis;

we examine how the density of the running variable and the observations near the cut-off affect the outcome, and, finally, we test to see if other observables have the same cut-off.

This study contributes to the literature in three ways: (1) we measure the effects of a financial crisis. The literature on the contextual variables affecting fetal development are usually limited to famine, natural disasters and terrorist attacks. (2) We measure effects in the long term which not only helps better mold public policy but paints a more comprehensive picture of the consequences of prenatal maternal stress. (3) We provide a method that attempts to identify causal effects while most studies are correlational. In studies where there is an exogenous shock there are mostly simple regression methods which compare the before-after without providing an appropriate counterfactuals. Additionally, we have not found studies which use regression discontinuity models or which analyze the long term health effects of pre-natal exposure to the 1999 Ecuadorian crisis.

Notwithstanding, there are various challenges that we addressed with the evidence presented in this paper. Firstly, despite testing and not finding any anticipation effects in the months before the crisis, we did find isolated significant placebo effects in the months after the crisis and on New Year's Day 1995, although they do not hold up to robustness checks. Secondly, there is a slight imbalance in the size of the samples, however, we find no evidence the density of the distribution or the observations near the cut-off have an effect on the outcome. Finally, despite our attempts, we are unable to test whether individuals with no access to financial services were effectively sheltered from the crisis, however, this is not the objective of the paper.

Another point of discussion is that we assume the cut-off is deterministic in increasing stress levels. There is an argument to be made that the relationship should be probabilistic, in that, stress can be caused by other unobservables which we cannot control for. We argue that there is always a certain percentage of mothers who suffer from prenatal maternal stress, and that this percentage would have otherwise been similar in the treatment and control group. The only change in the percentage would be that caused by the financial crisis.

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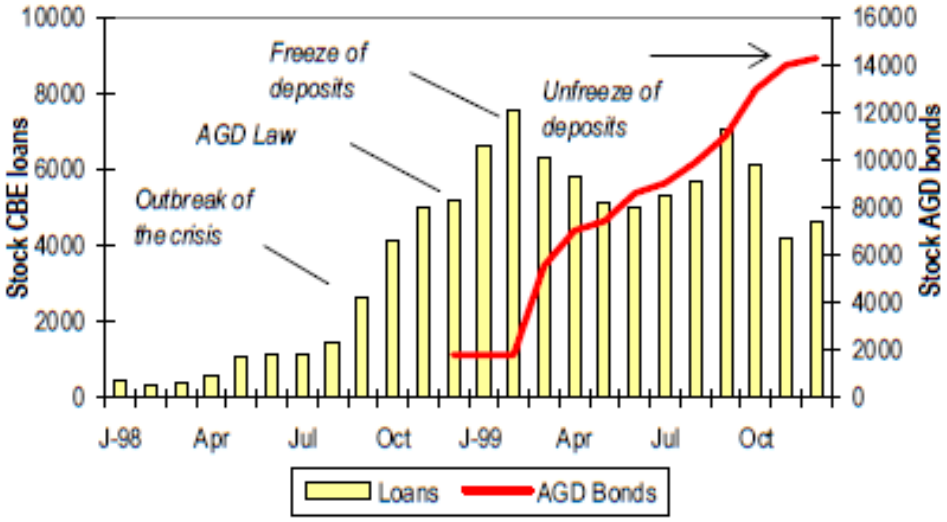
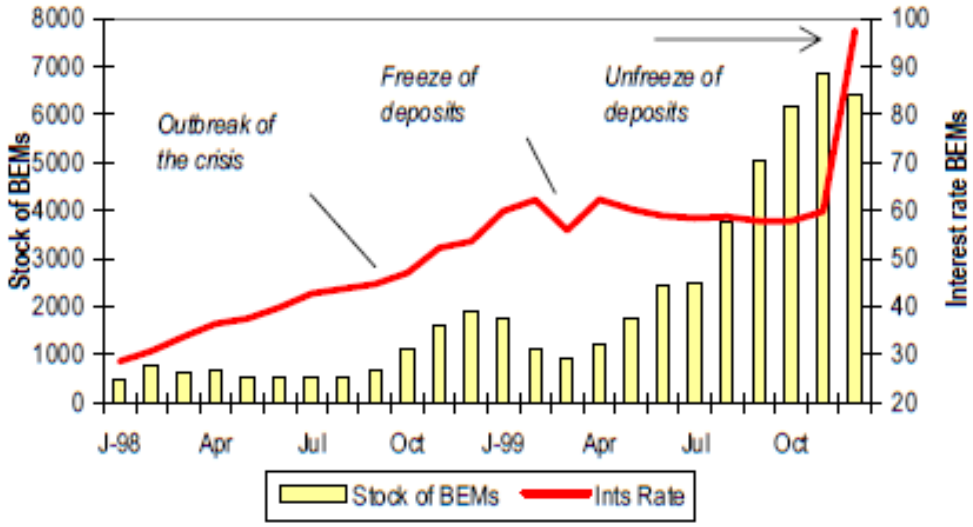
Appendix 1: Jacome 2004 Figures on Ecuadorian Crisis

Figure 17 Financial assistance to banks (Billions of Sucres)



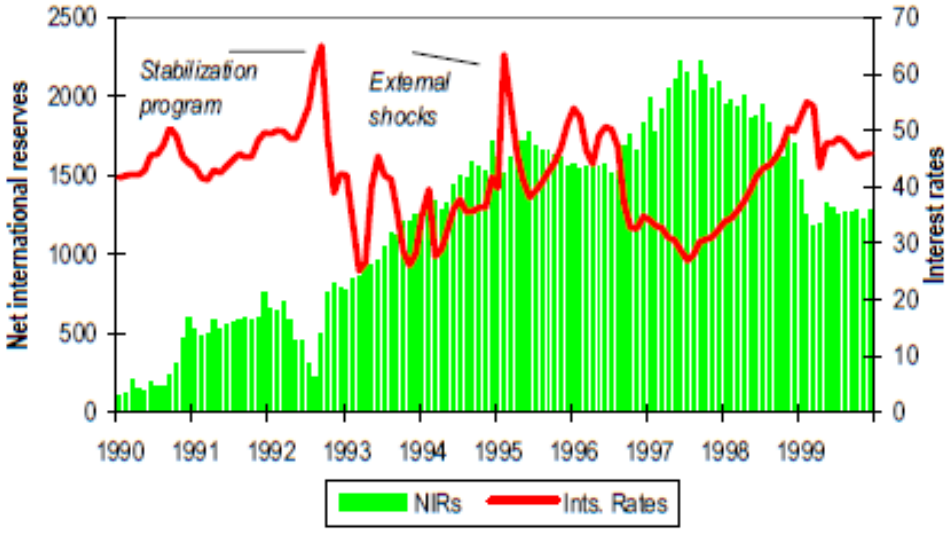
Source: Jacome, 2004; Source of data cites in Jacome, 2004: Central Bank of Ecuador

Figure 18 Open Market Operations (Billions of Sucres and annual rate)



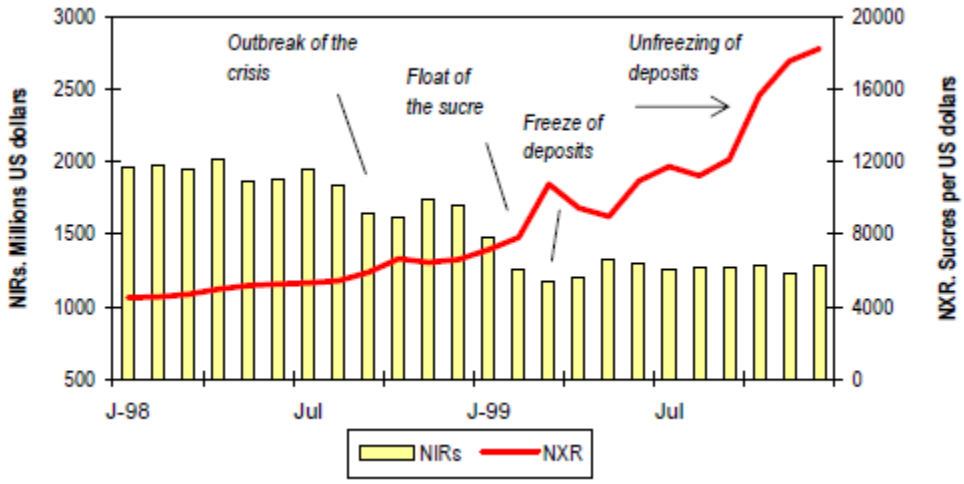
BEM: Government Bonds sold to mop up liquidity (Bonos de Estabilizacion Monetaria)
Source: Jacome, 2004; Source of data cites in Jacome, 2004: Central Bank of Ecuador

Figure 19 Net international reserves and interest rate (Millions of US dollars and annual rate)



Source: Jacome, 2004; Source of data cites in Jacome, 2004: Central Bank of Ecuador

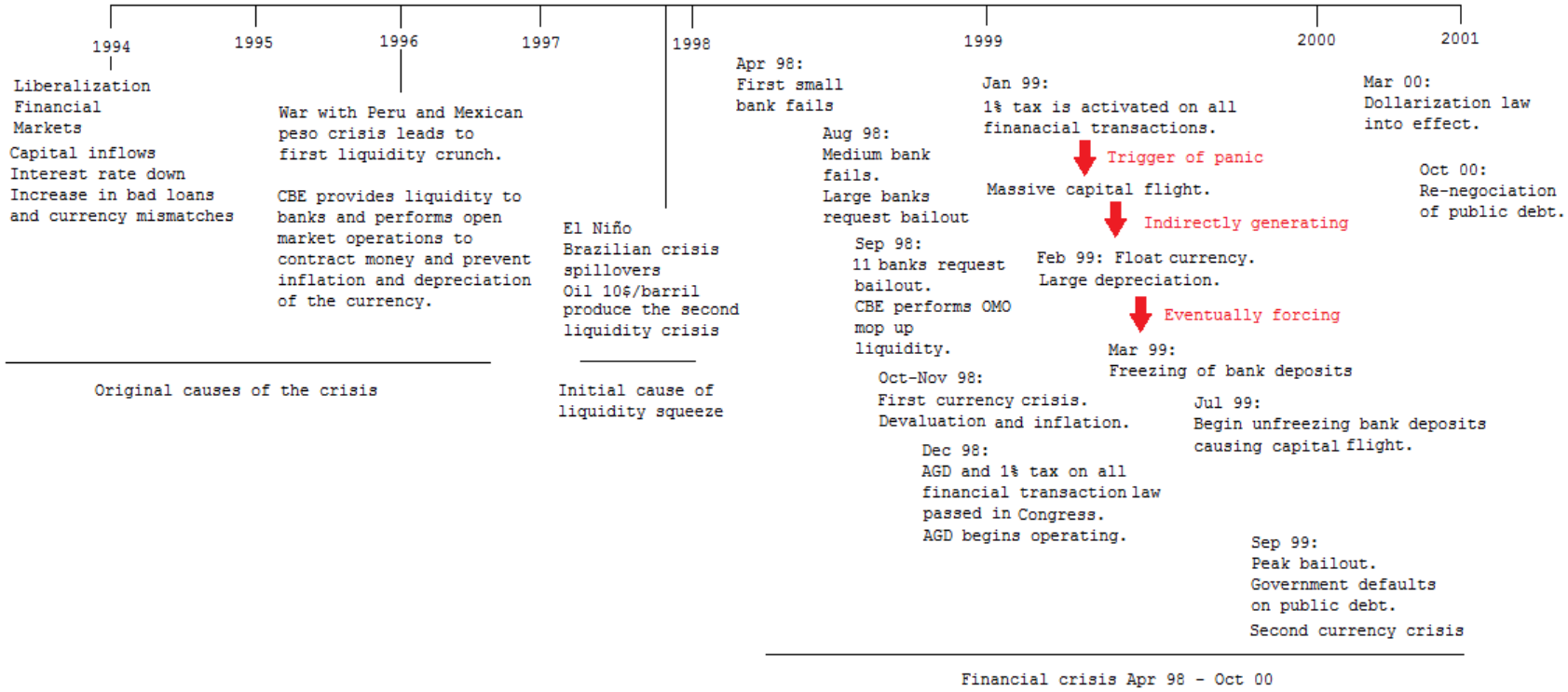
Figure 20 Net international reserve and nominal exchange rate (Millions of US dollars and Sucres per Dollar)



Source: Jacome, 2004; Source of data in Jacome 2004: Central Bank of Ecuador.

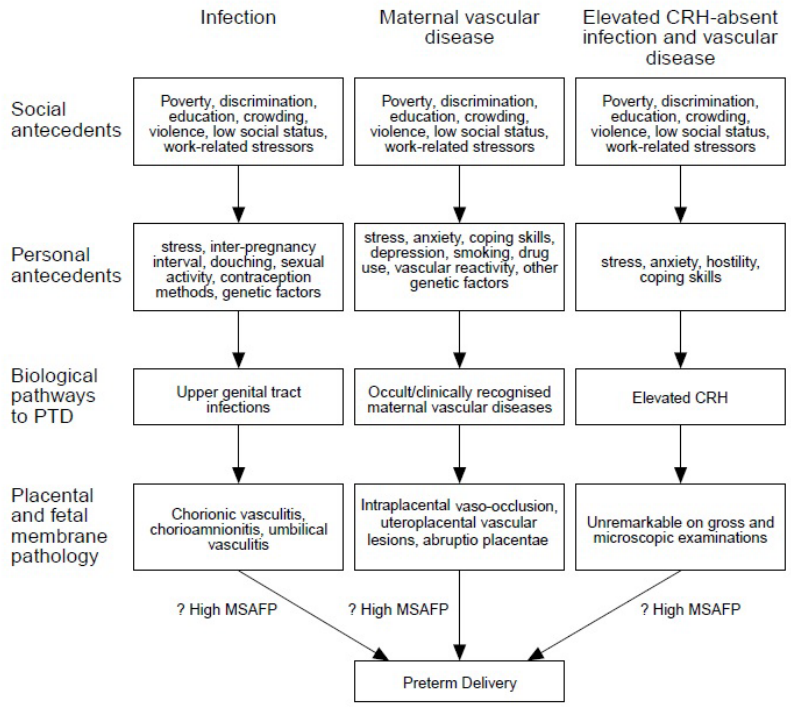
Appendix 2: Chronology of Crisis

Figure 21 Chronology of Ecuador's 1999 Financial Crisis



Appendix 3: Mechanism connecting pre-natal maternal stress to deleterious birth outcomes.

Figure 22 Prenatal maternal stress pathway

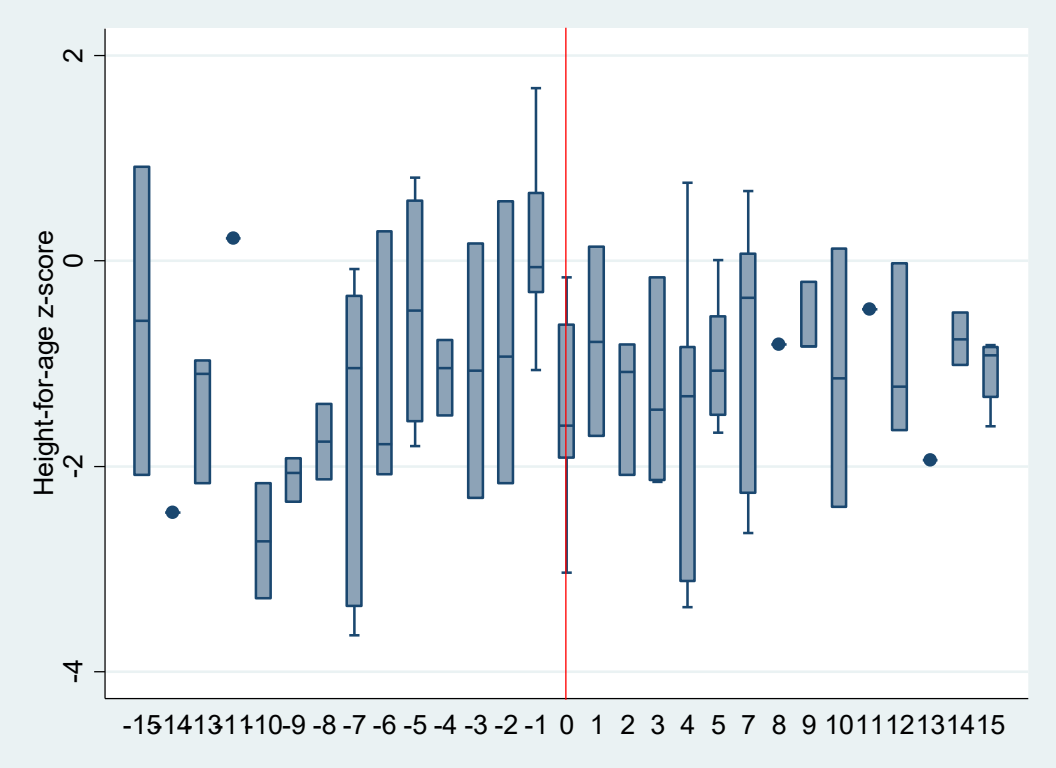


Source: C. Holzman, et al., 2001, Pregnancy outcomes and community health: the POUCH study of preterm delivery, Paediatric and perinatal Epidemiology, 15(2), pp. 138.

Appendix 4: Box plot of cut-off on 1 Jan 1999

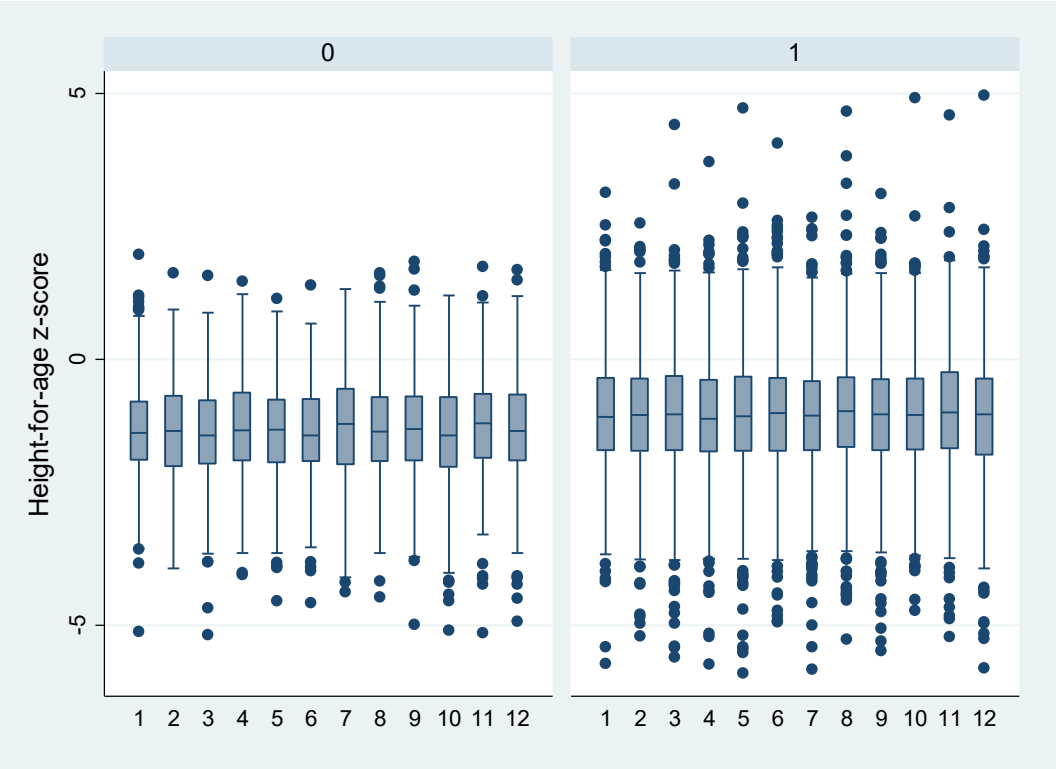
Figure 23 shows the average values (horizonal line within the box) and basic statistics (25th and 75th percentiles, in the lower and upper hinge, as well as the upper and lower adjacent values, in each end of the whiskers) of the distribution of z-scores per day for children born 15 days before/after the crisis. On the horizonal axis we have the running variable where zero is the cut-off day (1 Jan 1999), the negative numbers on the left of the cut-off are the number of days the individual was born before the crisis and the positive numbers represents the number of days born after the crisis.

Figure 23 Box-plot z-score height-for-age by day of birth for sample of children born just before/after crisis



In Figure 24 shows the average values and basic statistics of the distribution of z-scores per month a year before and after the crisis. That is, now the running variable is measured in months. Therefore, the negative values represent the number of months born before the crisis, while the positive numbers represent the number of months born after the crisis.

Figure 24 Box plot z-score height-for-age by month of birth for 12 months before/after cut-off



Appendix 5: Choosing a polynomial form: AIC for various bandwidths

Table 25 AIC for various bandwidths and polynomial orders

Bw	Order	Beta dtreat	AIC
30	1	-0.103	599.37
30	**2**	**-0.895****	**595.34**
30	3	-1.68***	594.30
25	1	-0.23	522.3
25	2	-1.05**	506.9
25	3	**-2.14*****	**501.7**
20	1	-0.35	392.07
20	**2**	**-1.7*****	**379.9**
20	3	-1.7**	383.8
15	1	-0.82**	298.7
15	**2**	**-1.94*****	**293.7**
15	3	-1.6**	297.1

* 0.1 ** 0.05 *** 0.01 *** 0.001

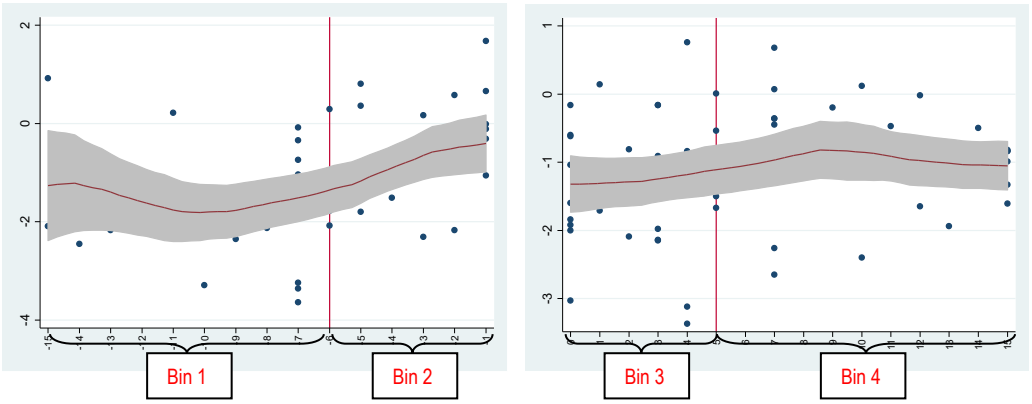
Appendix 6: Creating bin dummies for 15- and 30-day bandwidths.

Creating bin dummies for 15-day bandwidth

We created bin dummies separately for each side of the bandwidth. In Figure 25 we can see the frequency distribution of the running variable before and after the cut-off separately (we set the bins in the histogram to be the equivalent of a day each).

We use the egen xtile command which creates a variable which categorizes the running variable by its quantiles. The default value is 2 quantiles which effectively estimates the median. In the case of the observations before the cut-off, the median is -6. For those after the cut-off the median is 5. If the observations were equally distributed we would expect the median to be 7.5 on both sides. The fact that both medians are smaller demonstrates that there are more observations closer to the cut-off as on both sides, particularly after the cut-off.

Figure 25 Median of observations before and after cut-off for 15 day bandwidth



Before cut-off *After cut-off*

If we run the regression discontinuity model (using OLS) and integrating the bin dummies (total 4 by taking the two on each side), we can see that bin 1 is used as a reference bin, therefore, the coefficients of bin 3 to 4 are the difference between them and bin 1. For example, bin 2 has a positive significant coefficient which implies that the z-score is higher in bin 2 in relation to bin 1. It also implies that the linear model does not capture this behavior. Additionally, in every model bin 4 is dropped due to collinearity. This is probably due to the fact that the treatment variable is a dummy dividing the sample into two groups while the bin dummies are dividing the sample into 4 groups. Therefore, the bin dummies are almost identical to the treatment dummy when they are categorized into a small number of groups.

Table 26 Regression discontinuity model (15 day bandwidth, various polynomial forms, OLS regression) 4 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
dtreat99	1.003	-0.713	-0.633
	(0.90)	(1.22)	(1.31)
Z99	-0.010	0.362	0.272
	(0.07)	(0.20)	(0.40)
dtZ99	-0.008	-0.310	-0.196
	(0.08)	(0.24)	(0.46)
Z992		0.021	0.003
		(0.01)	(0.07)
dtZ992		-0.025	-0.013

		(0.01)	(0.08)
Z993			-0.001
			(0.00)
dtZ993			0.001
			(0.00)
1bn.cbin	.	.	.
	.	.	.
2.cbin	1.181*	0.508	0.428
	(0.54)	(0.64)	(0.71)
3.cbin	-0.523	-0.434	-0.499
	(0.53)	(0.54)	(0.75)
4.cbin	.	.	.
	.	.	.
_cons	-1.713**	-0.218	-0.245
	(0.65)	(1.00)	(1.02)
r2	0.129	0.167	0.168
N	100	100	100

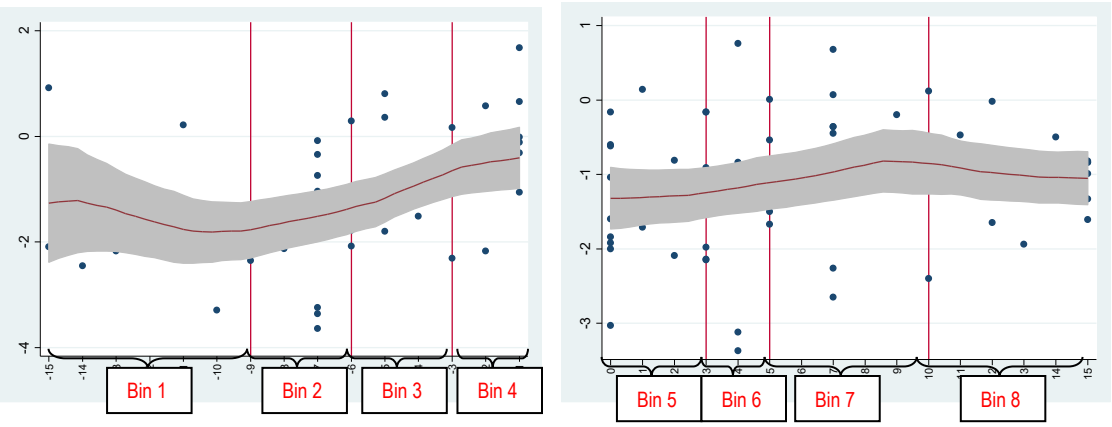
* 0.1 ** 0.05 *** 0.01 *** 0.001

What we can learn from this exercise is that, firstly, we need to think carefully about the reference bin. Do we want to compare the behavior of the z-score in relation to the first bin of observations which is always going to be those born the earliest before the crisis? Secondly, we need to think about the appropriate number of bins given a small number will resemble the treatment dummy and a large number will probably not have many observations within each category.

In relation to the former, our objective is to measure bumpiness in the running variable outside of the jump in the cut-off (which should be captured by the treatment dummy). Therefore, the reference bin should be irrelevant. Perhaps the only rule should be that it should not be at the cut-off point because we would expect to see a jump there.

In relation to the latter, we increase the number of bins to the point where no bin is dropped due to collinearity, then we measure how many observations are in each bin. We start with 4 bins on each side.

Figure 26 4 bins before and after cut-off for 15 day bandwidth



The regression discontinuity model using 8 bin dummies uses the first bin as the reference and also drops the 8th bin due to collinearity. None of the bin dummies are significant, which suggests that there are no bumps or jumps outside of the cut-off. Notwithstanding, the interaction between treatment dummy and the running variable (our treatment effect) is not significant.

Table 27 Regression discontinuity (15 day bandwidth, various polynomial forms, OLS regression) 8 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
dtreat99	2.155	-0.240	-0.293
	(2.17)	(3.15)	(3.40)
Z99	-0.144	0.332	0.650
	(0.12)	(0.47)	(0.90)
dtZ99	0.153	-0.248	-0.547
	(0.17)	(0.51)	(0.94)
Z992		0.022	0.074
		(0.02)	(0.13)
dtZ992		-0.028	-0.084
		(0.02)	(0.14)
Z993			0.002
			(0.01)
dtZ993			-0.002
			(0.01)
1bn.cbin	.	.	.
	.	.	.
2.cbin	0.686	0.506	0.776
	(0.73)	(0.75)	(0.99)
3.cbin	1.876	1.081	1.490
	(1.03)	(1.28)	(1.62)
4.cbin	2.930*	1.129	1.299
	(1.35)	(2.19)	(2.25)
5.cbin	-0.153	-0.229	-0.340
	(1.47)	(1.49)	(1.93)
6.cbin	-0.144	-0.366	-0.467
	(1.14)	(1.23)	(1.65)
7.cbin	0.283	0.041	0.001
	(0.77)	(0.92)	(1.03)
8.cbin	.	.	.
	.	.	.
_cons	-3.307*	-0.916	-0.760
	(1.46)	(2.71)	(2.77)
r2	0.160	0.173	0.174
N	100	100	100

* 0.1 ** 0.05 *** 0.01 *** 0.001

We repeat the exercise with 8 bins on each side for a total of 16 bin dummies in the model. Again, the first bin is used as a reference and the last (16th) bin is dropped due to collinearity. There are no bin dummies which are significant and the treatment has no effect. It would seem that the subdivision of the sample be it into 4 or be it into washes away the effect of the jump on the day of the crisis.

Figure 27 8 bins before and after cut-off for 15 day bandwidth

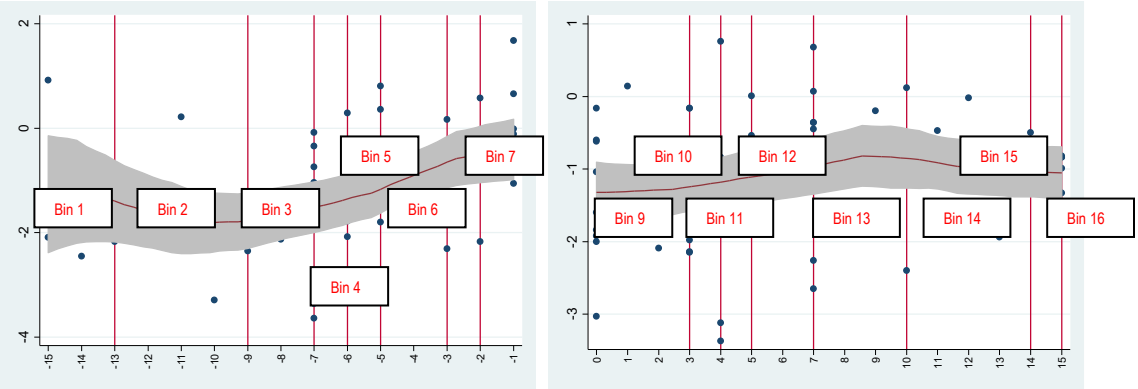


Table 28 Regression discontinuity (15 day bandwidth, various polynomial forms, OLS regression) 16 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
dtreat99	5.031	1.974	3.814
	(5.67)	(6.02)	(6.56)
Z99	-0.192	0.723	1.420
	(0.30)	(0.66)	(1.01)
dtZ99	0.049	-0.957	-1.426
	(0.39)	(0.83)	(1.35)
Z992		0.049	0.182
		(0.03)	(0.15)
dtZ992		-0.044	-0.234
		(0.04)	(0.24)
Z993			0.005
			(0.01)
dtZ993			-0.003
			(0.01)
1bn.cbin	.	.	.
	.	.	.
2.cbin	0.182	1.227	1.706
	(1.38)	(1.53)	(1.63)
3.cbin	0.803	1.675	3.026
	(2.04)	(2.11)	(2.58)
4.cbin	1.621	2.182	3.948
	(2.44)	(2.46)	(3.13)
5.cbin	2.513	2.704	4.733
	(2.71)	(2.70)	(3.49)
6.cbin	2.179	1.673	3.877
	(3.10)	(3.11)	(3.93)
7.cbin	3.520	1.494	3.353
	(3.75)	(3.96)	(4.46)
9.cbin	-2.494	-2.583	-3.246
	(3.90)	(3.92)	(4.50)
10.cbin	-1.919	-1.827	-2.720
	(3.30)	(3.32)	(4.44)
11.cbin	-2.032	-1.847	-2.671
	(2.90)	(3.02)	(4.07)
12.cbin	-1.328	-1.102	-1.797
	(2.65)	(2.84)	(3.66)
13.cbin	-0.832	-0.560	-0.903
	(2.14)	(2.48)	(2.74)
14.cbin	-0.588	-0.320	-0.270
	(1.62)	(2.04)	(2.06)
15.cbin	-0.249	-0.096	0.203
	(0.86)	(1.12)	(1.49)
16.cbin	.	.	.
	.	.	.
_cons	-3.961	-0.815	-1.992
	(4.12)	(4.59)	(4.79)
r2	0.188	0.212	0.221
N	100	100	100

* 0.1 ** 0.05 *** 0.01 *** 0.001

In order to get a better idea of what the bins contain, we present a series of tables with the number of observations in each bin. With 4 bins there are approximately 20 observations in each bin. With 8 bins there are around 10 in each bin (with the exception of bin 5 with 20). With 16 bins there are around 6 observations in each bin (with two exceptions near the cut-off).

Table 29 Number of observations (15 day bandwidth 4 bins)

bins	Obs	Min	Max
1	24	-15	-6
2	19	-5	-1
3	32	0	5
4	26	7	15

Table 30 Number of observations (15 day bandwidth, 8 bin)

bins	Obs	Min	Max
1	12	-15	-9
2	12	-8	-6
3	9	-5	-3
4	10	-2	-1
5	20	0	3
6	12	4	5
7	13	7	10
8	13	11	15

Table 31 Number of observations (15 day bandwidth, 16 bins)

bins	Obs	Min	Max
1	6	-15	-13
2	6	-11	-9
3	9	-8	-7
4	3	-6	-6
5	4	-5	-5
6	5	-4	-3
7	10	-2	-1
8			
9	9	0	0
10	11	1	3
11	6	4	4
12	6	5	5
13	7	7	7
14	6	8	10
15	7	11	14
16	6	15	15

Creating bin dummies for 30 day bandwidth

We use the same method to find the appropriate number of bins for the 30 day model. We find very similar results in that, for 4, 8 & 16 bins, the first bin is taken as a reference and last bin is dropped from the model due to collinearity. Additionally, the treatment effect is not significant in all models, much like when we use the 15 day bandwidth. We suppose that the dummies are also washing away the effect of the cut-off. In terms of number of observations, when we carve out 4 bins we have approximately 40 observations in each bin, with 8 we have about 20 and with 16 we have around 10 (with some exceptions).

Figure 28 4 bins before and after cut-off for 30 day bandwidth

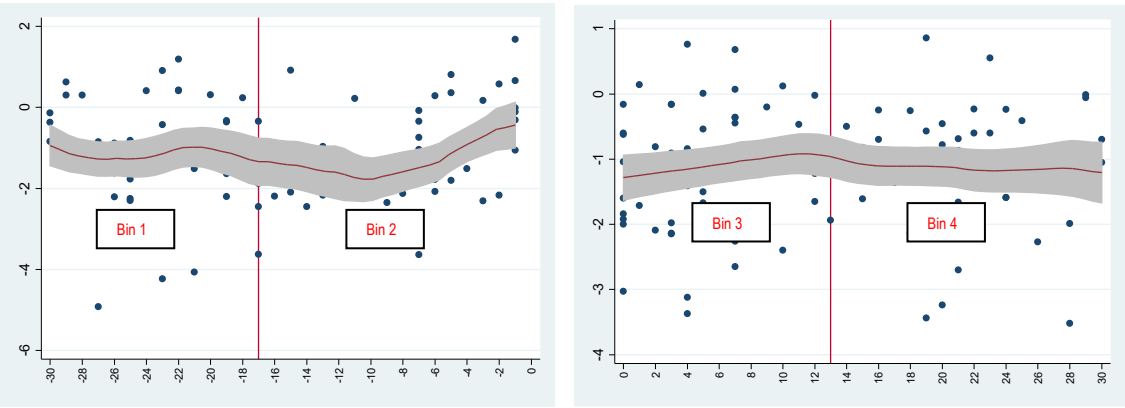


Table 32 Regression discontinuity (30 day bandwidth, various polynomial forms, OLS regression) 4 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
dtreat99	-1.088	-2.202*	-1.755
	(0.89)	(0.97)	(1.03)
Z99	0.038	0.188**	0.364*
	(0.03)	(0.06)	(0.15)
dtZ99	-0.029	-0.132	-0.232
	(0.04)	(0.08)	(0.18)
Z992		0.005**	0.021
		(0.00)	(0.01)
dtZ992		-0.006**	-0.031
		(0.00)	(0.02)
Z993			0.000
			(0.00)
dtZ993			-0.000
			(0.00)
1bn.cbin	.	.	.
	.	.	.
2.cbin	-0.678	-0.795	-0.204
	(0.52)	(0.51)	(0.69)
3.cbin	0.143	0.257	-0.101
	(0.49)	(0.49)	(0.66)
4.cbin	.	.	.
	.	.	.
_cons	-0.231	0.597	0.397
	(0.67)	(0.72)	(0.74)
r2	0.011	0.056	0.067
N	195	195	195

* 0.1 ** 0.05 *** 0.01 *** 0.001

Figure 29 8 bins before and after cut-off for 30 day bandwidth

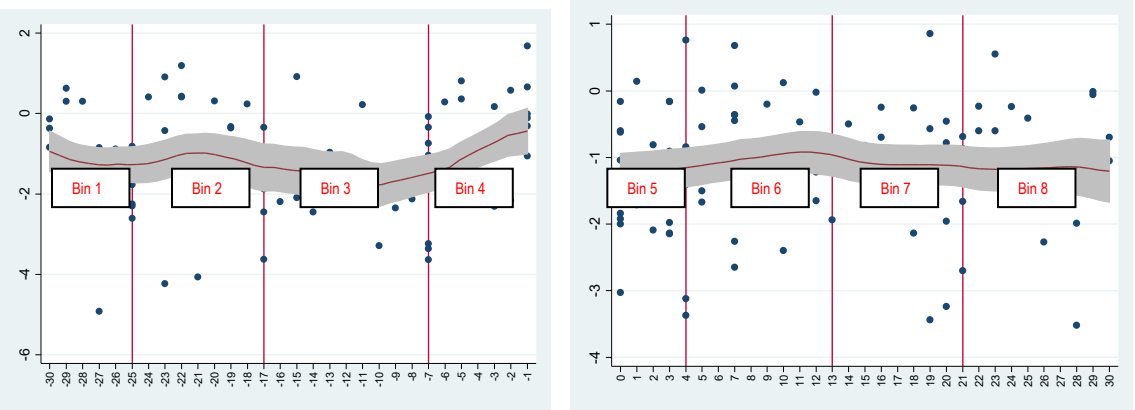


Table 33 Regression discontinuity (30 day bandwidth, various polynomial forms, OLS regression) 8 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
dtreat99	3.938*	3.016	3.114
	(1.79)	(1.82)	(1.85)
Z99	-0.080	0.165	0.254
	(0.05)	(0.10)	(0.19)
dtZ99	0.022	-0.189	-0.187
	(0.07)	(0.14)	(0.24)
Z992		0.008**	0.016
		(0.00)	(0.01)
dtZ992		-0.009*	-0.026
		(0.00)	(0.02)
Z993			0.000

			(0.00)
dtZ993			0.000
			(0.00)
1bn.cbin	.	.	.
	.	.	.
2.cbin	0.808	1.675**	1.629**
	(0.43)	(0.54)	(0.55)
3.cbin	0.960	1.767	1.952*
	(0.87)	(0.91)	(0.96)
4.cbin	2.616*	2.509*	2.622*
	(1.17)	(1.16)	(1.18)
5.cbin	-1.696	-1.622	-1.924
	(1.17)	(1.17)	(1.23)
6.cbin	-0.883	-0.944	-1.350
	(0.90)	(0.90)	(1.04)
7.cbin	-0.608	-0.723	-0.801
	(0.50)	(0.57)	(0.58)
8.cbin	.	.	.
	.	.	.
_cons	-3.479**	-2.688*	-2.608
	(1.30)	(1.32)	(1.33)
r2	0.093	0.127	0.131
N	195	195	195

* 0.1 ** 0.05 *** 0.01 *** 0.001

Figure 30 16 bins before and after cut-off for 30 day bandwidth

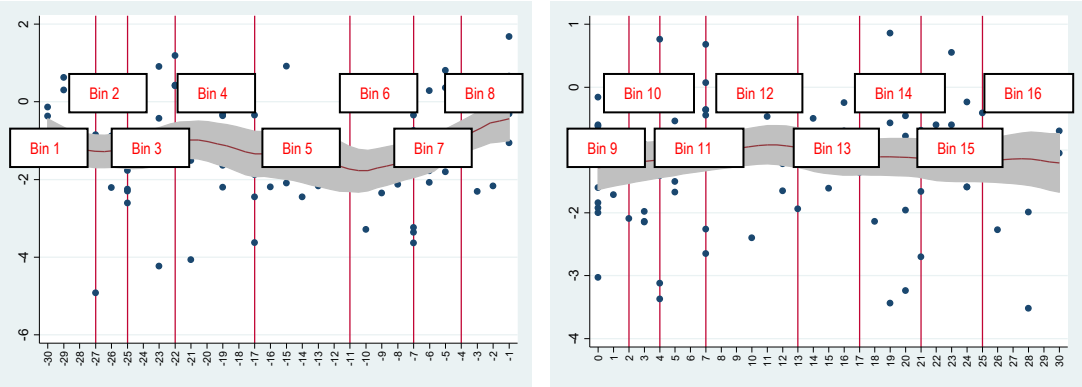


Table 34 Regression discontinuity (30 day bandwidth, various polynomial forms, OLS regression) 16 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
dtreat99	-2.154	-3.635	-3.070
	(4.26)	(4.28)	(4.29)
Z99	0.084	0.572*	0.327
	(0.11)	(0.24)	(0.40)
dtZ99	-0.103	-0.681*	-0.085
	(0.15)	(0.32)	(0.51)
Z992		0.015*	-0.005
		(0.01)	(0.03)
dtZ992		-0.012	-0.025
		(0.01)	(0.04)
Z993			-0.000
			(0.00)
dtZ993			0.001
			(0.00)
1bn.cbin	.	.	.
	.	.	.
2.cbin	-0.971	0.118	0.370
	(0.56)	(0.73)	(0.80)
3.cbin	-0.049	1.604	1.859
	(0.73)	(1.02)	(1.07)

4.cbin	-1.230	1.011	1.043
	(1.14)	(1.50)	(1.50)
5.cbin	-1.466	0.769	0.437
	(1.65)	(1.90)	(1.95)
6.cbin	-2.701	-1.369	-1.949
	(2.23)	(2.28)	(2.40)
7.cbin	-1.856	-1.388	-1.869
	(2.55)	(2.53)	(2.60)
8.cbin	-1.600	-2.436	-2.495
	(2.90)	(2.90)	(2.89)
9.cbin	-0.537	-0.592	-1.050
	(2.93)	(2.90)	(2.91)
10.cbin	-0.563	-0.387	-1.520
	(2.63)	(2.63)	(2.73)
11.cbin	0.019	0.351	-0.979
	(2.36)	(2.43)	(2.59)
12.cbin	0.027	0.530	-0.576
	(1.92)	(2.14)	(2.27)
13.cbin	0.092	0.656	0.252
	(1.41)	(1.79)	(1.81)
14.cbin	-0.238	0.251	0.470
	(0.99)	(1.38)	(1.38)
15.cbin	0.410	0.742	1.215
	(0.68)	(0.94)	(0.99)
16.cbin	.	.	.
	.	.	.
_cons	1.390	2.975	2.698
	(3.06)	(3.11)	(3.12)
r2	0.136	0.162	0.175
N	195	195	195

* 0.1 ** 0.05 *** 0.01 *** 0.001

Table 35 Number of observations (30 day bandwidth, 4 bins)

bins	Obs	Min	Max
1	53	-30	-17
2	44	-16	-1
3	50	0	13
4	49	14	30

Table 36 Number of observations (30 day bandwidth, 8 bins)

bins	Obs	Min	Max
1	25	-30	-25
2	28	-24	-17
3	22	-16	-7
4	22	-6	-1
5	26	0	4
6	24	5	13
7	27	14	21
8	22	22	30

Table 37 Number of observations (30 day bandwidth, 16 bins)

bins	Obs	Min	Max
1	15	-30	-27
2	10	-26	-25
3	13	-24	-22
4	15	-21	-17
5	8	-16	-11
6	14	-10	-7
7	10	-6	-4
8	12	-3	-1
9	14	0	2
10	12	3	4

11	13	5	7
12	11	8	13
13	13	14	17
14	14	18	21
15	11	22	25
16	11	26	30

Appendix 6: Descriptive statistics of the income distribution

Table 26, we can see the mean household income per capita. In Figure 24 we show the histogram for income per capita, and in Table 27 we have sample sizes for different bandwidths.

Table 38 Descriptive statistics of income per capita and the natural log of income per capita

Variable	Obs	Mean	Std. Dev.	Min	Max
Income per capita	60471	136.2	191.9	0	7500
Ln(income per capita)	57428	4.5	0.9	-0.35	8.9

Figure 31 Histogram of income per capita and the natural log of income per capita

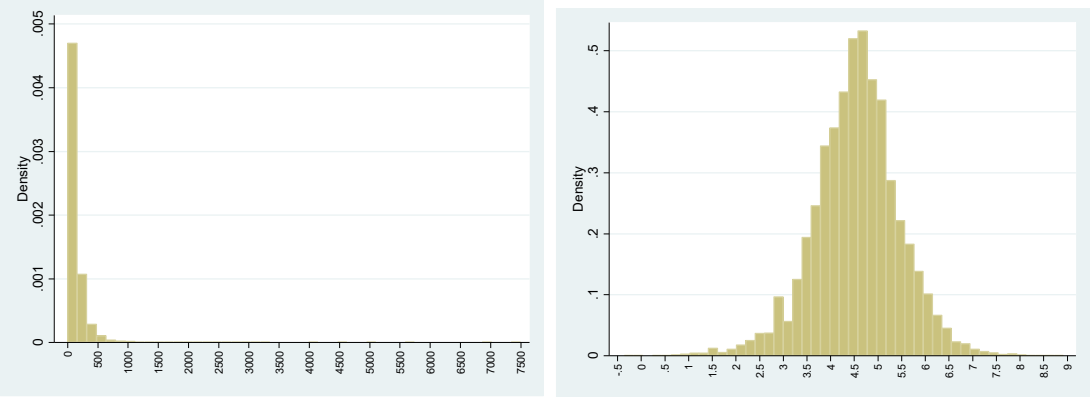


Table 39 Observations in deciles of the income distribution for various bandwidths

Deciles	No bandwidth		365 days		180 days		90 days	
	n	%	n	%	n	%	n	%
1	5639	10%	323	13%	140	11%	66	12%
2	4844	8%	274	11%	136	11%	55	10%
3	6454	11%	302	12%	152	12%	76	14%
4	4273	7%	164	6%	81	7%	35	7%
5	5948	10%	284	11%	143	12%	55	10%
6	5557	10%	230	9%	113	9%	39	7%
7	6008	10%	269	11%	118	10%	43	8%
8	5987	10%	230	9%	115	9%	52	10%
9	6136	11%	233	9%	116	9%	52	10%
10	6505	11%	238	9%	122	10%	56	11%
Total	57351	100%	2,547	100%	1,236	100%	529	100%

The distribution of income across deciles is fairly uniform in the sample with no bandwidth. This behavior is somewhat lost within the 90 day bandwidth and this tendency intensifies with the 30 day bandwidth. There is a higher percentage of observations in the first and second decile and a lower percentage in the middle of the distribution. Also, the number of observations decreases, as expected, as the bandwidth decreases.

In Table 28 we can see the mean income per capita is similar in 90 days bandwidth as compared to the sample with no bandwidth in all deciles except on the 10th. In 30 days bandwidth the maximum

income per capita in the tenth decile $867, in the 90 day bandwidth it is $1400 while in the whole sample it is $7500.

Table 40 mean income per capita in deciles for 90 day bandwidth as compared to no bandwidth

Deciles	No bandwidth			365 days			180 days			90 days		
	μ ypc	min ypc	max ypc	μ ypc	min ypc	max ypc	μ ypc	min ypc	max ypc	μ ypc	min ypc	max ypc
1	$19.8	$0	$30	$20.3	$1	$30	$20.5	$2.2	$30	$21.4	$5.5	$30
2	$38.8	$30.5	$46.6	$38.6	$31	$46.6	$38.7	$31	$46.6	$38.2	$31	$46.2
3	$54.1	$47	$60	$54.2	$41.6	$60	$54.4	$48	$60	$54.7	$48	$60
4	$68	$60.5	$73.5	$67.3	$60.5	$73.5	$67.5	$60.5	$73.5	$67.8	$60.5	$73.3
5	$81.6	$73.6	$90	$81.3	$73.6	$90	$81.4	$73.7	$90	$81.2	$73.7	$90
6	$99.2	$90.2	$107.1	$98.9	$91.4	$107.1	$98.7	$91.6	$106.6	$98.1	$91.6	$102.8
7	$121.9	$107.3	$136.6	$121.6	$107.5	$136	$121.7	$107.5	$136	$122.4	$108.3	$135.7
8	$155.4	$136.8	$176	$ 155.1	$137.1	$176	$154.3	$137.1	$176	$153.7	$137.1	$175
9	$216.5	$176.5	$266.6	$214.2	$176.5	$266.6	$215.2	$176.5	$266.6	$212.3	$176.5	$266.6
10	$502.4	$267	$7,500	$481.1	$270	$2375	$458.5	$270	$1400	$444.1	$272.6	$1400
Total	$136.2	$0	$7,500	$118.7	$0	$2375	$119.1	$0	$1400	$118.8	$0	$1400

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