Universitat Autònoma de Barcelona

Malnutrition and Inequality in Ecuador

Ana Larrea Peñaherrera

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Malnutrition and Inequality in Ecuador

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This work is dedicated to the children of Ecuador.

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List of Abbreviations

AIC	Akaike Information Criterion
BMI	Body Mass Index
CBE	Central Bank of Ecuador
CIBV	Wellbeing Childhood Center / Centros Integrales del Buenvivir
CNH	Growing with our Children / Creciendo con Nuestros Hijos
DANS	Diagnosis of the Nutritional and Health Situation of Ecuador survey / Encuesta para el Diagnóstico de la situación alimentaria, nutricional y de Salud
ENSANUT	National Health and Nutrition Survey (same as HNS) / Encuenta Nacional de Salud y Nutrition
Hb	Hemoglobin
HNS	Health and nutrition survey
IDA	Iron deficiency anemia
INEC	National Institute for Statistics and Censuses / Instituto Nacional de Estadísticas y Censos
ITT	Intention to treat
IV	Instrumental variable
LBW	Low birthweight
LOLR	Lender of Last Resort
LSMS	Living Standards Measurement Survey
MCDS	Ministry of Coordination of Social Development / Ministerio Coordinador del Desarrollo Social
MIES	Ministry of Economic and Social Inclusion / Ministerios de Inclusion Economica y Social
MSP	Ministry of Public Health / Ministerio de Salud Pública
OLS	Ordinary Least Squares
OMO	Open Market Operations
PNM	Pre-natal maternal
RD	Regression discontinuity
SAE	Small Area Estimates
WHO	World Health Organization
zhfa	z-score of height-for-age

Summary

Chronic malnutrition has been a persistent condition among Ecuadorian children. It has the potential to perpetuate the cycle of poverty by affecting cognitive development, schooling achievements and the potential lifetime income steam (Larrea, 2002; Larrea & Freire, 2002; Freire, et al., 1988) (Grantham-McGregor, et al., 2000; Granthan-MacGregor, et al., 2007; Walker, et al., 2000; Walker, et al., 2007; Martinez, et al., 2009).. The government-instituted nutritional-supplementation-program treats malnutrition by replenishing important micronutrient stocks through daily nutritional supplements (Ministerio de Inclusion Economica y Social, 2012; Ministerio de Inclusion Economica y Social, 2013). In Chapter 2, I apply a series of regression distribution (RD) and instrumental variable models and find no evidence that this treatment program has a significant average effect on hemoglobin levels[1] among children. Are there other social causes of chronic malnutrition?

The medical literature indicates that pre-natal maternal stress may increase the risk of adverse birth outcomes and can have effects later in life because fetal exposure to adverse in-utero conditions affects a series of "switches" in the genetic sequence of an individual called the epigenome (Almond & Currie, 2011; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997). In Chapter 3, I test this proposed mechanism directly by using the 1999 financial crisis as an unanticipated exogenous stress shock. I use the sharp RD method and find those exposed in-utero had significantly lower height-for-age z-scores than their non-exposed peers 12 years after the exposure. Consequently, the supplementation program's limited effect may be partially explained by exposure to pre-natal maternal stress. Additionally, a biological locus linking

[1] Iron-deficiency anemia (IDA) is a condition characterized by a depletion in iron reserves leading to a lower than normal level of Hb in the blood (U.S. Department of Health and Human Services, 2014). Hb is an iron-rich protein that carries oxygen from the lungs to the rest of the body.

pre-natal maternal stress to childhood growth trajectory provides a theoretical pathway linking social exclusion to individual health.

In order to assess the validity of the later, in Chapter 4, I test whether inequality has an effect on malnutrition at the individual level. I find a causal exogenous impact of the Gini coefficient on malnutrition independent of household income in 2006 but not in 2014. This concluding Chapter gives partial evidence that systematic social exclusion can both cause malnutrition and skew the effect of nutritional supplementation programs through its effect on pre-natal maternal stress (Larrea & Freire, 2002; Diez-Roux, 1998; Deaton, 2003; Preston, 1975; Lynch, et al., 2004; Wilkinson, 1996; Ellison, 2002; Macinko, et al., 2003; Wilkinson, 2000; Davey Smith & Egger, 1996) (Lynch, et al., 2000; Lynch, et al., 2000; Lynch, et al., 2001; Beydoun & Saftlas, 2008; Camacho, 2008; Mansour & Rees, 2011; Marins & Almeida, 2002; Willey, et al., 2009; Aerts, et al., 2004; El Taguri, et al., 2009) (Adair & David, 1997).

Chapter 1

Introduction

1.1 Outline of the problem

Childhood chronic malnutrition affects cognitive development, schooling achievements, potential lifetime income steam, and thus has the potential to perpetuate the cycle of poverty (Grantham-McGregor, et al., 2000; Granthan-MacGregor, et al., 2007; Walker, et al., 2000; Walker, et al., 2007; Martinez, et al., 2009). Currently, the Ecuadorian government distributes nutritional supplements to children under 5 (years of age) to improve their reserves of micro-nutrients, thus, treating the immediate biological cause of malnutrition. These efforts have had limited results (Chapter 2). So, why have these programs failed? I propose pre-natal maternal stress affects the post-birth growth trajectory of the child through the effect it has on their epigenetic make, which, unlike the genetic code, changes as a function of variations in, for example, the intra-uterine environment (Chapter 3). As a consequence, a social context which enables long periods of chronic stress during pregnancy can have an effect on the growth trajectory of a child (Chapter 4).

Chronic malnutrition has been a persistent condition among Ecuadorian children (Larrea, 2002; Larrea & Freire, 2002; Freire, et al., 1988). The government-instituted-nutritional-supplementation-program treats the direct biological causes of malnutrition by replenishing important micronutrient stocks in the body through daily nutritional supplements. The supplements are distributed free of charge to treat children under 5 and administered by the parents at home (Ministerio de Inclusion Economica y Social, 2012; Ministerio de Inclusion Economica y Social, 2013).

In Chapter 2 (An evaluation of Ecuador's policy to reduce childhood iron deficiency anemia), I find no evidence that this treatment program has a significant effect on hemoglobin (Hb) levels[2] among children. I apply a series of regression distribution (RD) and instrumental variable (IV) models. I find no significant effect of the change in the treatment policy in any of the RD or IV models. However, when I include heterogeneous effects by quantile of Hb in the IV model, I find a negative significant effect in the first

[2] Iron-deficiency anemia (IDA) is a condition characterized by a depletion in iron reserves leading to a lower than normal level of Hb in the blood (U.S. Department of Health and Human Services, 2014). Hb is an iron-rich protein that carries oxygen from the lungs to the rest of the body.

quantile and a positive significant effect in the second quantile. I suspect that these two opposing effects cancel each other out when the average effect is measured. The treatment can cause constipation or diarrhea, particularly among younger children who have never had "sprinkles" before (Ministerio de Salud Publica, World Food Program, 2011). I suspect this is the cause of the negative effect in the first quantile, which implies that the effect of the treatment policy might actually reduce Hb of the children when they become sick. In any case, the average treatment effect is zero, leading to the question: Are there other social causes of chronic malnutrition? And, what biological mechanism can explain the link between the social context in which a child lives and the growth trajectory they might follow? Can I test this mechanism directly?

The human genome is determined at conception and is fixed over time - it can be described as the "hardware" of genetics. The epigenome, on the other hand, is the program that "switches" genes on or off and can be described as the "software" of genetics. Consequently, the epigenome can change as a result of environmental shocks. The medical literature indicates that pre-natal-maternal (PNM) stress may increase the risk of adverse birth outcomes because fetal exposure to adverse in-utero conditions affects this series of "switches" in the genetic sequence of an individual. Consequently, pre-natal maternal stress can have effects later in life (Almond & Currie, 2011; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

In order to test this proposed mechanism directly, I find a natural experiment. In Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador) I use the 1999 financial crisis as an unanticipated exogenous stress shock, arguing it induced a potentially measurable amount of hardship on pregnant women at the time and, by extension, exposed the offspring to pre-natal maternal stress. I measure the effects on those who were exposed in-utero 12 years after the fact and use this natural experiment to identify an appropriate counter-factual (control group). I find a significant causal effect on chronic malnutrition. Those exposed in-utero had significantly lower height-for-age z-scores than their non-exposed peers 12 years after the exposure.

The results of this Chapter (3) have various important implications. Firstly, the micronutrient supplementation program's limited effect may be partially explained by exposure to PNM stress in-utero, a potential cause of malnutrition for which the program has no methodological instrument. Secondly, a biological locus linking PNM stress to the growth trajectory of a child provides a theoretical pathway linking stress inducing social exclusion to individual health. That is to say, income inequality, independently of income, can cause chronic child malnutrition, if inequality has an effect, independently of income, on chronic stress.

In order to assess the validity of the later, in Chapter 4 (Malnutrition and inequality in Ecuador), I test whether inequality has an effect on malnutrition at the individual level independent of income. I find a causal exogenous impact of the Gini coefficient on malnutrition independent of household income in 2006 but not in 2014. This concluding Chapter gives partial evidence that systematic social exclusion can both cause malnutrition and skew the effect of nutritional supplementation programs through its effect on PNM stress (Larrea & Freire, 2002; Diez-Roux, 1998; Deaton, 2003; Preston, 1975; Lynch, et al., 2004; Wilkinson, 1996; Ellison, 2002; Macinko, et al., 2003; Wilkinson, 2000; Davey Smith & Egger, 1996) (Lynch, et al., 2000; Lynch, et al., 2000; Lynch, et al., 2001; Beydoun & Saftlas, 2008; Camacho, 2008; Mansour & Rees, 2011; Marins & Almeida, 2002; Willey, et al., 2009; Aerts, et al., 2004; El Taguri, et al., 2009) (Adair & David, 1997).

1.2 State of the art review

As mentioned above, this thesis begins with an evaluation of Ecuador's policy to reduce childhood iron deficiency anemia. I find various studies on nutritional supplementation programs that have found mixed results, similar to the ones found in my first chapter. Nores and Bernet's (2010) review of 56 studies find substantial benefits to cognition, health and schooling in various countries. In Latin America, similar programs have varying results. Torrejon et al. (2004) find the Chilean fortified milk program favorably affects iron but not the zinc levels of women and children. Varea et al. (2012) find the Argentine food aid program significantly decreases the deficiency of Vitamin A and folate, however has no significant effect on anemia in lactating women one year after implementation. Silva et al. (2008) find the Brazilian fortified milk program contributed to improve nutritional status of children. However, Bortolini and Vitolo (2012) find the Brazilian dietary counseling program has no effect on the incidence of anemia, iron deficiency anemia or iron deficiency among children 12 to 16 months (Nores & Barnett, 2010; Torrejon, et al., 2004; Varea, et al., 2012; Silva, et al., 2008; Bortolini & Vitolo, 2012).

There is no evaluation of the nutritional supplementation program in Ecuador. The literature focuses on evaluating the effect of the conditional cash transfer program (Bono de Desarrollo Humano) on a range of health indicators. Nores and Bernet (2010) find cash-transfer programs have a similar mean effect on health to nutrition-based programs.[3] Manley et al.'s (2013) meta-analysis of 21 studies find the average effect of cash transfer programs impact on height-for-age is positive but not statistically significant. Leon and Younger (2007) find that the Ecuadorian cash transfer payment scheme has a significant effect on a child's nutritional status. Ponce and Bedi (2010) find no significant effect of the program on second grader's cognitive achievements. Fernald and Hidrobo (2011) found significant effects on language development however not for children living in urban areas and no significant effect on the z-score-of-height-for-age or on Hb concentration. Schady (2012) finds mixed results on anemia in women of reproductive age, and, finally, Carranza Carona and Mendez Sayago (2014) find no significant effect on exclusive breastfeeding practices (Nores & Barnett, 2010; Manley, et al., 2013; Leon & Younger, 2007; Ponce & Bedi, 2010; Fernald & Hidrobo, 2011; Schady, 2012; Carranza Baron & Mendez Sayago, 2014)

There is very little research on why any Ecuadorian nutritional supplementation or cash transfer program fail to obtain the desired results when they do indeed fail. There are no publications proposing other biological mechanisms that might dampen the effectiveness of these types of policies and therefore there is little debate on how they might be improved. As far as I can tell, there have been no previous published studies on the impact of PNM stress on the health outcomes of Ecuadorian children.

As mentioned above, I propose exposure to PNM stress has the potential to alter growth patterns post-birth, and, consequentially, may reduce the effectiveness of the nutritional supplementation program. Gluckman et al. (2005) propose that this is basically a predictive adaptive response the fetus has to an early environmental "cue," given the fetus may interpret an intra-uterine shock as a signal of its post-natal environment. This may result, in an effort to adapt to this new expected future living condition on the part of the fetus by preemptively changing its developmental trajectory. This "coping" mechanism can have long-term effects on the individual's fitness for survival as it imposes changes that may impact that individual at a later stage in life. For example, a reduction in maternal nutrition may trigger a change in the fetal growth pattern in an attempt to match the supply of nutrients. This allows to fetus to survive, however, it may have post-natal costs such as altered pancreatic development, insulin release and blood vessel

[3] Although the variance seems to be larger among the cash-transfer programs Cash transfer: 0.382 – SD 0.601, nutrition: 0.375 – SD 0.232 (Nores & Barnett, 2010).

growth. Consequently, the transport of nutrients later in life may be affected by the development of blood vessels during this period (Gluckman, et al., 2005).

Two publications by Rice & Thapar (2010) and Rice et al. (2010) essentially disentangle the effect of the fetal environment 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 intra-uterine environment to offspring birth outcomes independently of the contribution of the genome. They find significant correlations between PNM stress and birth outcomes among genetically related and unrelated offspring (Rice, et al., 2010; Rice & Thapar, 2010).[4] The same authors use the same in-vitro-fertilization scheme and also find a correlation between maternal height and offspring birth-weight and head circumference among both genetically related and unrelated offspring (Rice & Thapar, 2010). Conversely, Zijlmans' et al. meta-analysis finds only a small number of associations between maternal pre-natal cortisol and child outcomes are significant. 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).

I find a considerable amount of evidence suggesting an empirical link between stress inducing life-events and birth outcomes. Almond and Currie (2011) find numerous studies providing evidence of the long-term consequences of a wide variety of intra-uterine shocks. Schetter & Tanner (2012) find that a majority of the more than a dozen published studies measuring objective stress events[5] have significant effects on pre-term birth and birth-weight, while studies on perceived stress did not consistently predict pre-term birth or birth-weight. On the other hand, Hobel et al. (2008) find mixed evidence of links between psychosocial stress and preterm birth. Beydoun and Saftlas (2008), in their review of the literature on the effect of PNM stress on fetal growth, 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 & Currie, 2011; Schetter & Tanner, 2012; Hobel, et al., 2008; Beydoun & Saftlas, 2008).

Various studies find significant associations between intra-uterine exposure to natural disasters such as hurricanes, ice storms, floods and earthquakes and the probability of abnormal conditions of the newborn,[6]

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

[5] 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).

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

birth lengths, LBW, and preterm delivery (Currie & Rossin-Slater, 2013; Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016). In Sweden, family events such as a death or a financial stress are found to be significant in shortened gestational age, preterm birth, LBW, and small for gestational age, 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 Body Mass Index (BMI) (Class, et al., 2011; Zhu, et al., 2013). Various authors study the events on September 11th 2001[7] and find significant associations with lower term birth-weight 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).

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). Hidrobo (2014) studies the effect of the 1999 Ecuadorian economic crisis on health and receptive language data for children 0 to 5 years old. Results suggest that a single year of exposure to the crisis significantly decreases the z-score-of-height-for-age and vocabulary test scores (Hidrobo, 2014).

The recognition of the importance of psychosocial factors, specifically channeled through chronic stress is a crucial development in our understanding of the social determinants of health and has received greater attention as a source of chronic stress (Berkman & Glass, 2000; Marmot, 2004; Marmot & Wilkinson, 2006; Nguyen & Peschard, 2003; Agren, 2003; Bennett, 2003; Health Canada, 1999; Howden-Chapman & Tobias, 2000; Organization of Economic Cooperation and Development, 2001; Persson, et al., 2001) (Turrell, et al., 1999; Lynch, et al., 2004). Macinko et al. (2003) present a review of 45 studies and find 33 (73%) have a significant inequality health relation. Lynch et al. (2004) review 98 studies on inequality and health of which 65 (66%) found that all or some inequality-health associations were statistically significant. Wilkinson & Pickett (2006) reviewed 155 studies and found that 124 (80%) had supportive or partially supportive evidence. One of the studies was Larrea and Kawachi's (2005) paper on Ecuador, where the

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

authors find a significant relationship between inequality and chronic child malnutrition and is the publication on which Chapter 4 (Malnutrition and inequality in Ecuador) is based (Macinko, et al., 2003; Lynch, et al., 2004; Wilkinson & Pickett, 2006; Larrea & Kawachi, 2005).

1.3 Description of the central question

In Chapter 2 (An evaluation of Ecuador's policy to reduce childhood iron deficiency anemia) I evaluate the national micronutrient supplementation program in Ecuador. I measure the effect of iron supplements on the Hb[8] levels of 6 to 59-month-old children. I use a cross-section national health and nutrition survey (HNS[9]) which contains data on participation in the supplementation program and on the Hb levels of a sub-sample of children.[10] I apply two methods: firstly, a fuzzy RD model where the age eligibility rule is used as the cut-off, and secondly, an IV model where the age cut-off is the instrument. For the former, I present various bandwidths around the cut-off, as well as, various functional forms on either side of the cut-off in order to corroborate the treatment effect was not determined by either of these two factors. In the latter I include various individual, maternal, household and regional control variables as well as fixed effects for ethnicity and region. I also include heterogeneous effects for extreme poverty and children in public daycare centers, as well as, by Hb quantile. The age cut-off is a strong and exogenous instrument for two reasons. Firstly, because it is difficult to manipulate the age of a child in order to access the treatment after the cut-off. Secondly, I find evidence to support the hypothesis that the age cut-off is enforced and has not changed over time. There are two important limitations of this study, the first of which is the wording of the survey question, and the second of which is the lack of information on treatment completion i.e. number of doses taken.

With regards to the former, the survey asks if the child had participated over the previous 12 months rather than if they currently participate in the program. This affects our capacity to accurately measure the cut-off age.[11] In order to address this issue, I create a proxy cut-off 11-months after the age limit (71 months old) and run a Kernel-weighted local polynomial regression on either side of it. I find that the probability of receiving the treatment falls to zero at 71 months. Had there been no regard for the age cut-off, there would

[8] Hemoglobin
[9] The National Health and Nutrition Survey (ENSANUT which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC) in Ecuador between 2011 and 2013.
[10] Obtained through blood samples.
[11] Children 11 months older than the cut-off (71 months old) who respond "yes" to this question, could either have participated 12 months ago (at 59 months old) - while still under the age cut-off, or, could have participated 11 months ago (at 60 months old) - after exceeding the age cut-off. Similarly, children 1 month older than the cut-off (60 months old) who respond "yes" to this question, could either have participated 2 or more months ago (at most 59 months old) - while still under the age cut-off, or, could have participated within the last month (at 60 months old) - after exceeding the age cut-off.

be no visible jump in the probability of receiving treatment at any age, let alone exactly 11 months after the age limit. Therefore, I argue this jump is due to the formulation of the question. As a result, I assume 71 months accurately measures the cut-off point given the formulation of the survey question.

With regards to the latter, the HNS[12] does not provide information on treatment completion. The treatment requires at least 60 doses (once a day) over, at most, 4 months. Therefore, interrupting the treatment may have a deterministic effect on the outcome (Ministerio de Coordinacion de Desarrollo Social, 2011). In randomized trials, the issue of non-compliance is accounted for with an "intention to treat" (ITT) model where the effect of the treatment is estimated regardless of whether the patients completed the treatment or not (Armijo-Olivo, et al., 2009). This method is analogous to the ITT model in that it uses the initial randomization of the program in order to estimate the effect of a change in treatment policy rather than of the effect of treatment on compliers (Hollis & Campbell, 1999).

In Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador) I measure the effect of the 1999 Ecuadorian financial crisis on the 2012 height-for-age of children in-utero around the time of the crisis. On 1 Jan 1999 an unusual tax on all financial transactions fueled a drastic fall in total deposits, a swift and massive flight in liquidity, and accelerated the collapse of various financial institutions in Ecuador. The sudden and precipitous collapse of the financial system exposes those born after this date to an objective unanticipated pre-natal maternal stress shock. In order to estimate the effect of this shock I propose a sharp RD model. The method compares children born just after the 1 Jan 1999 "cut-off" with those born just before. This creates a counter-factual (control group) that can be assumed to have very similar observable and unobservable characteristics to the treatment group, and thus, allows us to identify a causal effect of the shock. The sharp RD models assume randomized variation is a consequence of the inability of agents to control the assignment variable near the cut-off. I make sure relevant observable characteristics are not significant determinants of selection into treatment, I use a data-driven method to select an appropriate bandwidth, I use the Akaike information criterion (AIC) as well as a dummy variable test in order to select the polynomial order, and finally, I test the sensitivity of the results to triangle, rectangle and Epanechnikov kernel functional forms (Cattaneo, et al., 2018; Lee & Lemieux, 2010). Additionally, I run 4 robustness checks as recommended by Lee and Lemieux (2010) and Cattaneo et al. (2018): (1) placebo effects for the months and years predating the crisis; (2) I examine the density of the running variable, (3) I test for the sensitivity of the model to observations near the cut-off, and, finally,

[12] The National Health and Nutrition Survey (ENSANUT which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC) in Ecuador between 2011 and 2013.

(4) I test to see if other observables have the same cut-off. I find children born after the crash have significantly lower z-scores-of-height-for-age in 2012 than children born before.

Notwithstanding, there are various weaknesses with the evidence presented in this paper. Firstly, despite testing and not finding any anticipation effects before the crisis, I did find an isolated significant placebo effect on New Year's Day 1995 and two significant effects after the crisis, on 1 Feb and 1 April 1999. Notwithstanding, none of these effects survive a simple local polynomial robustness check and are found only when using a specific bandwidth and polynomial order which is insufficient to prove an exogenous effect. Secondly, the density of the running variable is not uniform leading to a slight imbalance in the size of the samples, however, I find no evidence the density of the distribution of observations has an effect on the outcome and I find significant effects even after eliminating observations near the cut-off which concurrently also balances the sample sizes in treat and control groups. Finally, despite my attempts, I was 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 chapter. I believe I provide ample evidence there is no anticipation bias and no manipulation of the cut-off, making this a robust RD design.

In Chapter 4 (Malnutrition and inequality in Ecuador) I measure the effect of inequality on chronic child malnutrition (stunting) in Ecuador. I use two living standards measurement survey (LSMS): the first of 2006, and the second of 2014. I present two of models: a benchmark Ordinary Least Squares (OLS) model and an IV regression using the Gini coefficient measured at the provincial, county and parish level against the z-score-of-height-for-age. The instrument is slightly different for each year. In 2006 it is the proportion of households in the parish that report suffering a draught in the last year, and in 2014 it is the proportion of household that report suffering any natural disaster (including flood, draught, storms) in either the province, county or parish. I argue the proportion of households in the parish/county/province that report suffering a draught/natural disaster is an exogenous determinant of inequality because it is an unanticipated event. In both IV models, I control for household consumption per capita in order to account for the effect of individual income. Additionally, I include controls for the individual characteristics of the child, the mother, the household, and other contextual variables including but not limited to access to healthcare, education, employment conditions, prenatal and postnatal care, adequate housing, and diet and add fixed effects for ethnicity. I find the Gini coefficient has a deleterious significant effect on the z-score-of-height-for-age in every model in 2006, however, the relationship is no longer significant in 2014.

There are various limitations to this study: firstly, I cannot control for the anthropometric measure of the parents in 2006, however, I can and do in 2014. Secondly, the fact that the LSMS sample is not

representative at the county[13] and parish level, therefore, I cannot estimate the Gini coefficient directly from this survey (in either year). Unfortunately, the 2010 census data in Ecuador does not have information on income or consumption. Therefore, in order to estimate the Gini coefficient at the county and parish levels I use *small area estimates*, a methodology proposed by Elbers, et al. (2003). I build a consumption prediction model on the 2006 / 2014 LSMS and use the estimated parameters to simulate consumption on the Ecuadorian population census of 2010. This allows me to estimate the conditional distribution of consumption for every household in the census and thereafter, a point estimate of the Gini coefficient and its standard error. Tarozzi & Deaton (2009) argue that, in order to match survey and census data in the way which is proposed by Elbers et al. (2003), a degree of spatial homogeneity is required for which the method has no basis. They argue that estimates based on those assumptions may underestimate the variance of the error in predicting welfare estimated at the local level and therefore overstate the coverage of confidence intervals. In response, Elbers, et al. (2008) compare their small area estimate welfare results in Minas Gerais, Brasil, a notably heterogeneity area, with the true welfare values and find small mean squared errors and appropriate confidence interval estimations (Elbers, et al., 2003; Elbers, et al., 2008; Tarozzi & Deaton, 2009).

For the 2006 model Ecuador was divided into eight sub-regions, for the 2014 model I divide the data into 16 relatively homogeneous areas. Separate consumption models and simulations were run for each one of these 8 sub-regions (2006) and I did the same for the 16 sub-groups in 2014. However, the main limitation is that the *small area estimates* model depends heavily on a degree of heterogeneity which cannot be controlled for methodologically and cannot be guaranteed empirically. The efforts made to divide the country into homogeneous regions may abate this limitation, however, the simulated Gini coefficients are systematically under-estimated. This is in part due to a the LSMS sample which under-represents the rich (highest end of the income distribution) as its objective is to measure the living conditions of the middle and lower income earners (Unidad de Análisis de Información del Ministerio de Coordinación de Desarrollo Social, 2012). The resulting household consumption model is therefore "over-fitted" to the conditions of this section of the income distribution. Therefore, when it is simulated onto the (2010) census it reproduces this systematic bias, and under-estimated the household consumption of the rich. This results in under-estimated Gini coefficients (Tarozzi & Deaton, 2009).

Other simulations methods, particularly newer recurring neural networks based on artificial intelligence are more powerful in terms of prediction error, however, are black box methods with "hidden" layers where

[13] With the exceptions of highly populated counties such as Quito, and Guayquil.

the parameters (correlation coefficients) are not explicit (Montavon, et al., 2018). Apart from machine learning, there are limited alternatives to the *small area estimates* in terms of methods which address the issues raised by Deaton and Tarozzi (2009).

The results seem to indicate that the effect has lost its significance between these two surveys. I find the Gini coefficient drop in all cases between 2006 and 2014. Perhaps the reduction in the significance of the effect is due to the reduction in the severity of inequality. Further research would be needed regarding the relationship and the possible threshold beyond which inequality is too weak to be deleterious to health (Subramanian & Kawachi, 2004).

The main artery of the thesis is Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador) where I provide an explanation for the mixed effects found in Chapter 2 (An evaluation of Ecuador's policy to reduce childhood iron deficiency anemia) and the empirical evidence for the mechanism explaining the results in Chapter 4 (Malnutrition and Inequality in Ecuador). The analysis of the relationship between inequality and individual health is the main motivation behind this thesis, however, the exploration of the epigenetic mechanism and the construction of the empirical evidence to prove its workings is perhaps its most important contribution.

The thesis will firstly evaluate the current nutritional supplement public policy in Ecuador in Chapter 2. Secondly, test the effect of pre-natal maternal stress on growth outcomes later in life in Chapter 3. Thirdly, explore the effect of inequality on malnutrition in children in Chapter 4 and, finally, end with a discussion and concluding remarks in Chapter 5.

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Chapter 2

An evaluation of Ecuador's public policy to reduce iron-deficiency anemia in children

2.1 Introduction

Micronutrients, particularly iron, zinc and vitamin A, folic acid and iodine, play a vital role in childhood development. A micronutrient deficiency during infancy affects growth, the immune system, may increase the risk of mortality, and can have long term effects on cognitive development and schooling achievements (Martinez, et al., 2009). Iron-deficiency anemia (IDA) is a condition characterized by a depletion in iron reserves leading to a lower than normal level of Hb in the blood (U.S. Department of Health and Human Services, 2014).[14] It is the most widespread micronutrient deficiency in the world (WHO, 2015), approximately 40% of the infants in developing countries are iron deficient (Micronutrient Initiative, 2015), and it is the only nutrient deficiency which is also significantly prevalent in industrialized countries (WHO, 2015).

This paper attempts to evaluate the current Ecuadorian national public policy to reduce IDA in children 6 to 59 months of age. Ecuador is an important case study for the evaluation of public policy to reduce IDA as it has had a persistent problem with iron-deficiencies among children. In 1986 a diagnostic study on the nutritional health of children found that 69% of 6 to 12 month olds had anemia (Freire, et al., 1988). In 2012 the national Health and Nutrition Survey (HNS[15]) shows that the incidence in the same age group is 62% indicating a 7% reduction in 26 years. (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013).

The national nutritional program relies on supplements developed to prevent and treat micronutrient deficiencies among young children.[16][17] The program distributes nutritional supplements to the parents of children attending public daycare and public healthcare centers nationwide. The policy rule stipulates that children under the age of 59 months (5 years old) are eligible to receive the treatment, therefore, children

[14] Hb is an iron-rich protein that carries oxygen from the lungs to the rest of the body.
[15] The National Health and Nutrition Survey (ENSANUT which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC) in Ecuador between 2011 and 2013.
[16] "Sprinkles" were developed to prevent and treat micronutrient deficiencies among young children (Ministerio de Salud Publica, World Food Program, 2011).
[17] Among young children.

over this age are no longer eligible (Ministerio de Inclusion Economica y Social, 2012; Ministerio de Inclusion Economica y Social, 2013).

The 2012 HNS[18] is a cross-section survey which covers participation in public nutritional supplement programs and includes a sub-sample of 2047[19] children 6 to 59 months old who had blood samples taken and Hb measured.[20] In order to identify the causal effect of the treatment policy I apply two methods: (1) RD and (2) IV with and without heterogeneous effects.

The RD model uses the age eligibility to randomly divide the children around the cut-off into treatment and control groups. Those who are just under the age cut-off are in the treatment group and those who are just over the age cut-off are in the control group. The aim of the exercise is to define a sufficiently small bandwidth around this cut-off such that the control group is an appropriate counter-factual, in its (un)observable characteristics, to the treatment group. In this paper, I present various bandwidths and functional forms to examine the robustness of our results.

In the IV model I use the same cut-off as an exogenous instrument to treatment. This is an exogenous instrument because children cannot manipulate their age in order to receive the treatment. In both cases I am able to estimate the causal effect of the treatment. Additionally, using the IV model I present heterogeneous effects for each quantile of Hb. In an IV model with heterogeneous effects the model has two endogenous variables: the dummy treatment and the interaction between dummy treatment and the heterogeneous effect (dummy quantile). The first instrument is the cut-off (dummy age under 71 months) (same as for the regular IV model) and the second instrument is the interaction between the cut-off and the heterogeneous effect (dummy quantile). If the instrument is exogenous, as I argue it is, then the interaction of this instrument and the heterogeneous effect (dummy quantile) is also exogenous. In every IV model I control for the characteristics of the child, the mother, the household and have fixed effects for ethnicity and regions of Ecuador.

The HNS[21] does not provide information on treatment completion. This may be important given the treatment requires at least 60 doses (once a day) to be taken at most over the span of 4 months (Ministerio de Coordinacion de Desarrollo Social, 2011). Therefore, interrupting the treatment may have a deterministic

[18] The National Health and Nutrition Survey (ENSANUT which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC) in Ecuador between 2011 and 2013.
[19] Out of a total sample of 11506 children under the age of 5.
[20] Among other biomarkers
[21] The National Health and Nutrition Survey (ENSANUT which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC) in Ecuador between 2011 and 2013.

effect on the outcome. In randomized trials, the issue of non-compliance is accounted for with an "intention to treat" (ITT) model where the effect of the treatment is estimated regardless of whether the patients completed the treatment or not (Armijo-Olivo, et al., 2009). The ITT model gives an estimate of the effect of a change in treatment policy rather than an estimate of the effect of the treatment in patients who comply with it (Hollis & Campbell, 1999). The models applied in this study (RD and IV) identify the non-biased effect of the program by using the eligibility rule as an instrument. I believe this is analogous to the ITT model in that it uses the initial randomization of the program in order to estimate the effect of a change in treatment policy rather than of the effect of treatment on compliers.

I find no significant effect of the change in the treatment policy (or the intention to treat) in any of the RD models, and, I find no significant effect in the IV model with no heterogeneous effects. When heterogeneous effects by quantile of Hb are included, I find a negative significant effect in the first quantile and a positive significant effect in the second quantile. I suspect that these two opposing effects cancel each other out when measuring the average effect. However, this leaves us with the important question of how to explain the negative significant effect. The only explanation is the possible non-compliance with the number of required doses as it is a deterministic factor which is not taken into account in this model. Additionally, the supplement can cause constipation or diarrhea, particularly among younger children who have never had "sprinkles" before (Ministerio de Salud Publica, World Food Program, 2011). Therefore, the effect of the treatment policy might actually reduce Hb if the children get diarrhea and/or stop receiving doses.

Aside from the fact that identifying the completion of required dosage is impossible, there is one other important limitations of this study: the wording of the survey question. The survey question inquires not about "current" participation in the program[22] but rather asks if the child has participated in the program at any time over the past 12 months prior to the survey. This has important implications on our capacity to accurately measure the cut-off age. Children 11 months older than the cut-off (71 months old) who respond "yes" to this question, could either have participated 12 months ago (at 59 months old) - while still under the age cut-off, or, could have participated 11 months ago (at 60 months old) - after exceeding the age cut-off. Similarly, children 1 month older than the cut-off (60 months old) who respond "yes" to this question, could either have participated 2 or more months ago (at most 59 months old) - while still under the age cut-off, or, could have participated within the last month (at 60 months old) - after exceeding the age cut-off.

[22] i.e. participation in the program at the moment the survey was taken.

In order to address this issue, I run a Kernel-weighted local polynomial regression on either side of the 71-month cut-off (see Figure 2.5) and find that the probability of receiving the treatment falls to zero at 71 months. Had there been no regard for the age cut-off, there would be no visible jump in the probability of receiving treatment at any age, let alone exactly 11 months after the age limit. I believe this jump is due precisely to the formulation of the question which stipulates participation over the last 12 months. Had the question stipulated participation over the last 6 months, the jump in the probability of receiving treatment would have occurred exactly 5 months after the cut-off (64 months). Therefore, I assume 71 months accurately measures the cut-off point given the formulation of the survey question.[23]

Despite its limitations, this study is an important contribution to the literature on the nutritional health of children. Most studies present methods which allow for the identification of causal rather than correlational effects, as is done here, however, this study is the only study to evaluate a nutritional supplement transfer program in Ecuador. Most of the literature on the nutritional outcomes of children in Ecuador evaluate the "Bono de desarrollo humano," a national cash transfer program (Ponce & Bedi, 2010; Leon & Younger, 2007; Fernald & Hidrobo, 2011; Schady, 2012). Alternatively, there are studies on the effects of the Ecuadorian 1999 financial crisis or of exclusive breastfeeding practices (Hidrobo, 2014; Carranza Baron & Mendez Sayago, 2014). Additionally, most of the studies mentioned above focus on the effect of these programs, crisis or practices on the z-score of height for age or on cognitive development. There are no studies, to our knowledge, which focus on Hb.

The findings of this study essentially outline there is no average effect of the change in treatment policy on Hb levels in children. There is a positive significant effect among children in the highest Hb quantile, however, there is a negative significant effect among the children with the lowest Hb levels. Further research is needed on identifying the effect by dose and on the effect on children who get severe diarrhea as a consequence of the treatment, as I suspect attrition and diarrhea might be driving the negative effect.

This article will firstly provide a description of the Ecuadorian context and a revision of the literature on supplementation and transfer programs in developing countries. Secondly, I will explain the program and describe the data available to us in each case. Thirdly, I present the empirical strategies I have used and their results. Finally, I will discuss and conclude.

[23] 71 months old implies children who are up to 71 months and 31 days old i.e. children who are 72 months minus a day old. If children who are 72 months are included, children who are up to 13 months minus a day past the cut-off are included.

2.2 Context and review of the literature

Ecuador is a small upper middle income country (The World Bank, 2016) in Latin American which has seen important social improvements in the past decade. The incidence of poverty has gone down from 37% in 2007 to 23% in 2015; the incidence of extreme poverty has gone from 16% to 8% and the Gini coefficient from 0.55 to 0.47 in the same period (Instituto Nacional de Estadistica y Censos, 2015). Notwithstanding, Ecuador can be described as a country with pronounced social, regional and ethnic disparities with high levels of poverty and inequality (Larrea & Kawachi, 2005; Farrow, et al., 2005).

IDA refers to a condition in which a deficiency in iron leads to a lower than normal number of red blood cells and those which are produced have less than the normal levels of Hb. [24][25] (U.S. Department of Health and Human Services, 2014). Ecuador has had a persistent problem with infant micronutrient deficiency in general and IDA in particular (Larrea & Kawachi, 2005; Farrow, et al., 2005; Larrea & Freire, 2002). In 1986 a diagnostic study on the nutritional health of children found that 69% of children 6 to 12 months and 46% of those 6 to 24 months had anemia (Freire, et al., 1988). Recently, the HNS[26] of 2012 shows that anemia among children 6 to 12 months is still 62% and among those 12 to 23 months is 32%. Out of all the children between 6 and 59 months 26% are anemic in 2012 (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013). Additionally, Ecuadorian chronic childhood malnutrition[27] was recorded at 40% in 1986[28], 34% in 2004[29] and 25% in 2006[30], however, the incidence has remained around 25% since (25% in 2012[31], and 26% in 2014[32]) (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013).[33]

[24] Iron-deficiency anaemia may develop over time as an iron deficiency will force the body to use its iron reserves. The consequential depletion and eventual exhaustion of iron reserves pushes the body to produce less blood cells and those which are produced have less than the normal level of Hb.
[25] Hb is an iron-rich protein that carries oxygen from the lungs to the rest of the body.
[26] The National Health and Nutrition Survey (ENSANUT which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC) in Ecuador between 2011 and 2013.
[27] For detailed explanation of this variable see Appendix 1.
[28] As measured by the Encuesta Nacional de la Situacion Alimentaria, Nutricional y de Salud de la Poblacion Ecuatoriana Menor de Cinco Años also known as the 1986 DANS.
[29] As measured by the Encuesta Demografica y de Salud Materna e Infantil also known as the 2004 ENDEMAIN.
[30] As measured by the Encuesta de Condiciones de Vida also known as the Living Standards Measurement Survey or the 2006 LSMS. All estimation form HNS 2012 report except for the estimations of the LSMS of 2006 and 2014 which were made by the author.
[31] As measured by the Encuesta Nacional de Salud y Nutrition refered to it in this paper the national Health and Nutrition Survey or the 2012 HNS.
[32] As measured by the Encuesta de Condiciones de Vida also known as the Living Standards Measurement Survey or the 2014 LSMS. All estimation form HNS 2012 report except for the estimations of the LSMS of 2006 and 2014 which were made by the author.
[33] All estimation form HNS 2012 report except for the estimations of the LSMS of 2006 and 2014 which were made by the author.

Strategies to control IDA include daily and intermittent iron supplementation, fortification of food staples, fortification with micronutrient powders, broader activities to improve food security and dietary diversity (Pasricha, et al., 2013). Nores and Bernet (2010) in their meta-analysis of early childhood interventions review 56 studies, 38 contrasts of 30 interventions in 23 countries outside of the US and Canada. They find the benefits from various contexts and countries are substantial in terms of cognition, behavior, health and schooling and that these benefits are sustained over time. Additionally, they find the largest cognitive benefits when interventions have an educational or stimulation component. Specifically, they find that cash-transfer programs have similar mean effects on health than nutrition based programs although the variance seems to be larger among the cash-transfer programs.[34] Ecuador is included in this study, however, the only program that it takes into consideration for Ecuador is the conditional cash transfer program called Bono de Desarrollo Humano which I will describe below (Nores & Barnett, 2010).

Various studies have evaluated micronutrition supplementation programs in a variety of countries with mixed results. Mirmiran et al. (2012) in their review of 81 published articles on micronutrient deficiencies in the Middle East find that despite implementation of flour fortification the prevalence of iron deficiency is moderate to severe (Mirmiran, et al., 2012). Prado et al (2016) test the effect of lipid-based nutrient supplements on motor, language and personal-social development in children in Burkina Faso and find a positive significant effect (Prado, et al., 2016). Sadighi et al. (2008) evaluate the effect of Iran's 2001 flour iron fortification program which targeted women 15 to 49 and found no effects in terms of Hb levels or IDA, however, found a lower prevalence of low ferritin levels among the treated (Sadighi, et al., 2008).

There have also been various evaluations of supplementation programs in Latin America. Torrejon et al. (2004) evaluate the effect of iron and zinc fortified milk on the iron and zinc levels of children and women in Chile. They find that the fortified milk favorably affects the iron levels but not the zinc levels of children (Torrejon, et al., 2004). Silva et al. (2008) evaluate the effect of an iron fortified milk beverage with probiotic bacteria on both growth and iron levels of children who have a low iron diet intake in Brazil. They find that the fortified beverage contributed to improve nutritional status (Silva, et al., 2008). Varea et al. (2012) evaluate the effect of the Argentine food aid program on the micronutrient status of lactating women one year after the programs implementation and find a significant decrease in Vitamin A and folate deficiency, however no significant change in anemia levels (Varea, et al., 2012). Bortolini and Vitolo (2012) evaluate the effect a dietary counseling program covering breastfeeding and complementary feeding had on iron deficiency anemia among children 12 to 16 months of age in Brazil. They find no evidence that the

[34] Cash transfer: 0.382 – SD 0.601, nutrition: 0.375 – SD 0.232 (Nores & Barnett, 2010).

intervention had an effect on anemia incidence, iron deficiency anemia or iron deficiency (Bortolini & Vitolo, 2012). The effects of a program seems to depend on the implementation and the type of fortification or supplementation chosen in the policy.

Additional strategies to treat nutrition deficiencies include cash-transfer programs. Manley et al. (2013) perform a meta-analysis of the effect of cash transfer programs on nutritional status of children where they include 21 studies and 17 programs. They find the programs' average impact on height for age is positive but not statistically significant. Most of the literature regarding public policy evaluation in Ecuador test the effect of conditional cash transfer programs on a range of health indicators (Manley, et al., 2013). Ponce and Bedi (2010) test the effect of the cash transfer called Bono de Desarrollo Humano on children's cognitive achievements. They use a RD strategy to identify the impact of the program on second graders and find no significant effect (Ponce & Bedi, 2010). Leon and Younger (2007) evaluate the effect of the transfer payment scheme called Bono Solidario in Ecuador on a child's nutritional status. They find that this cash transfer payment scheme has a significant effect on a child's nutritional status (Leon & Younger, 2007). Fernald and Hidrobo (2011) evaluate the effect of the Bono de Desarrollo Humano on health and development outcomes such as language shills, the z-score of height for age and Hb concentration in children between 12 and 35 months. They found significant effects on language development however not for children living in urban areas and no significant effect on the z-score of height for age or on Hb concentration (Fernald & Hidrobo, 2011). Hidrobo (2014) studies the effect of the 1999 Ecuadorian economic crisis on health and receptive language data for children 0 to 5 years old. Results suggest that a single year of exposure to the crisis significantly decreases the z-score of height for age and vocabulary test scores (Hidrobo, 2014). Schady (2012) studies the effect of cash transfers (Bono de Desarrollo Humano) on anemia in women of reproductive age and find mixed results (Schady, 2012). Carranza Carona and Mendez Sayago (2014) study the effect of this cash transfer on exclusive breastfeeding practices in Ecuador and find no significant effect (Carranza Baron & Mendez Sayago, 2014). As far as I can tell, there have been no previous published studies on the impact of non-cash micronutrient supplement transfers on Hb levels in children in Ecuador.

2.3 Program description and data

The supplement known as "sprinkles" is a blend of micronutrients distributed as a powder in small envelopes. "Sprinkles" were developed to prevent and treat micronutrient deficiencies among young children. One envelope contains 12.5mg of iron, 5mg of zinc, 160µg of folic acid, 300µg RE of vitamin A, and 30mg of vitamin C. The administration of 60 doses in 60 days is sufficient to quickly improve the

concentration and deposits of Hb and iron in the blood. The sprinkles do not require a change of food consumption behaviours on the part of the family, they do not require any special measuring tools, they do not require the parents to be literate to be able to administer the dose, and they may be administered at any time during the day in any meal. They are encapsulated in lipids which prevents the interaction with food and masks the taste, consequently, there are minimal changes in taste, colour and texture of the food into which it is mixed. Notwithstanding, the supplement may cause constipation or diarrhea, particularly among younger children who have never had "sprinkles" before (Ministerio de Salud Publica, World Food Program, 2011).

2.3.1 National Iron Supplementation Program

There are two national parallel programs currently distributing "sprinkles." The first program distributes supplements through the Ministry of Economic and Social Inclusion's (MIES[35]) public daycare centers (CIBV[36]) for children between 6 and 59 months.[37] For children who are not in daycare centers,[38] the MIES distributes "sprinkles" through house visits & group session program[39] (CNH[40]) (MCDS MIES INFA MSP MINEDUC, 2011).[41] The second program is coordinated through the Ministry of Public Health (MSP[42]) which distributes "sprinkles" and vitamin A to children between 6 and 24 months during their routine checkups (Ministerio de Salud Publica, World Food Program, 2011).[43]

Both of these programs are coordinated by the Ministry of Coordination of Social Development (MCDS[44]) which establishes the umbrella public policy denominated Childhood Development Strategy[45] in which children under 5 years of age are reported as the target demographic (Ministerio Coordinador del Desarrollo Social, 2011). Therefore, I have chosen to analyze both of them together.

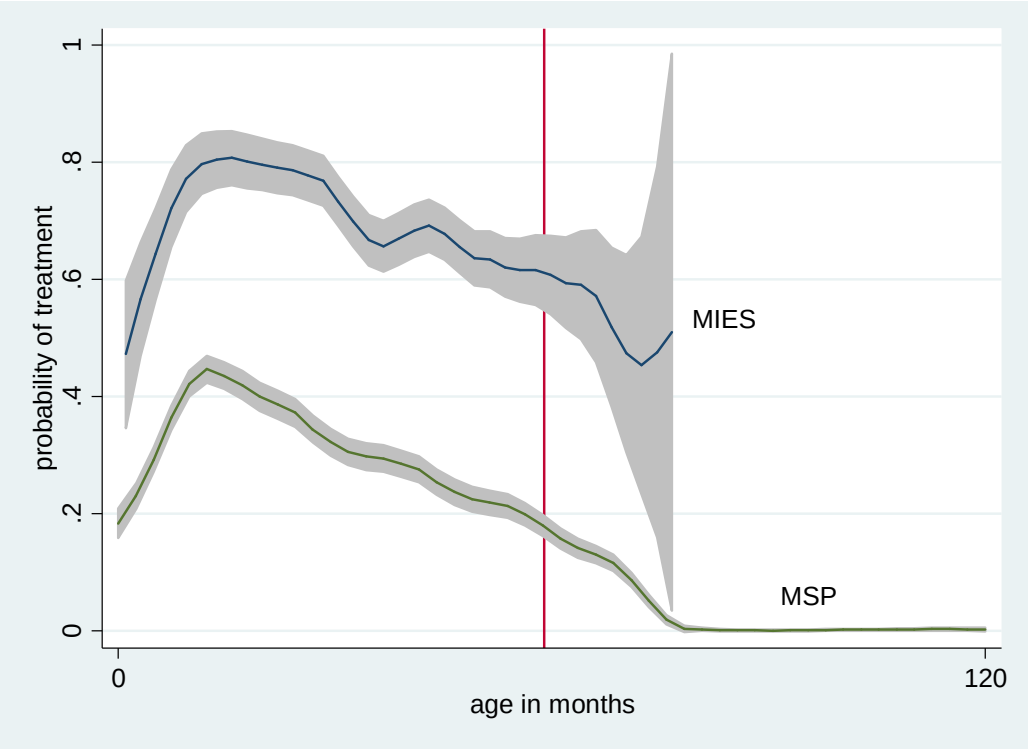
2.3.2 The sample

Hereafter, I will refer to the daycare program as the MIES program the health center program as the MSP program.[46] As can be seen in Figure 2.1, the children in the MIES program (blue) present a higher

[35] Given its name in Spanish: Ministerio de Inclusion Economica y Social.
[36] Well-being Childhood Centers given its name in Spanish: Centros Integrales del Buen Vivir.
[37] Administered by the caretakers at the daycares.
[38] Between 0 and 59 months.
[39] Administered in the context of the household by the parents.
[40] Growing with our Children given its name in Spanish: *Creciendo con Nuestros Hijos.*
[41] House visits are provided only for children between 0 and 12 months.
[42] Given its name in Spanish: *Ministerio de Salud Publica*.
[43] In this case, the doses are also given to the parents and administered by them in the context of the household
[44] Given its name in Spanish: *Ministerio Coordinador del Desarrollo Social.*
[45] In Spanish: *Estrategia de Desarrollo Infantil.*
[46] Even though the house visits program is technically coordinated through the same ministry as the daycare centers. This is done because it is not possible to identify the source of the treatment, however, it is not known whether the child is attending a public

probability of receiving the treatment than the children in the MSP program (green) at any age. The confidence interval among the children in the MIES program (blue) increases quite quickly after the 59 month cut-off point. This is due to the fact that the daycare centers only accept children up to 59 months old. The number of children still in daycare after 59 months is reduced to 49 children (details on sample size below).

Figure 2.1 Probability of treatment by program and age in months



Source: Author's computation using 2012 Nutrition & Health Survey

MIES : Ministry of Economic and Social Inclusion's (MIES) public daycare centers (CIBV) for children between 6 and 59 months. For children who are not in daycare centers, the MIES distributes "sprinkles" through house visits & group session program (CNH) (MCDS MIES INFA MSP MINEDUC, 2011).

MSP: Ministry of Public Health (MSP) which distributes "sprinkles" and vitamin A to children between 6 and 24 months during their routine checkups (Ministerio de Salud Publica, World Food Program, 2011).

daycare center or not. This allows us to differentiate the children who receive or might have received the treatment in daycare centers from the children who received it at home (through home visits or through their parents receiving the sachets at the healthcare center and administering it in the context of the household).

As mentioned above, in this study, I take into account children from both programs. I do this because children in the MIES program receive the treatment through public daycare centers and these centers also only accept children up to the age of 59 months. Therefore, to only take the children in daycare centers (MIES program) into account implies having an unacceptably small control group of 49 children (those above the cut-off point of 59 months) (See Table 2.1). Additionally, I would not want to exclude the children in public daycare centers from the analysis given they have the highest probability of receiving the treatment (see Figure 2.1).

Secondly, there is the question of the cut-off point for the MSP program. In this study I consider the umbrella MCDS program cut-off point of 59 months for the whole sample. This, despite some literature suggesting the cut-off point for the MSP program is 24 months (Ministerio de Salud Publica, World Food Program, 2011). I justify this decision with the data in Table 2.2 where the reader can see that most of the children not in public daycare centers who receive the treatment through the MSP program are over the age of 24 months. Additionally, Table 2.3 shows most of the children not in public daycare centers who receive the treatment through the MSP program are under the age of 59 months. Therefore, it seems that, in practice, the eligibility rule that is adhered to is the umbrella MCDS program cut-off point of 59 months.

Additionally, there is some evidence of a willingness to change the public policy eligibility rule from 59 to 36 months which began to appear in the literature in 2013 (Ministerio de Inclusion Economica y Social, 2013).[47] As Table 2.4 shows, a very large proportion of the pooled sample of children from both MIES and MSP programs who receive the treatment are over 36 months. In Table 2.5 it is shown that of the same pooled sample of all children the majority of children who receive the treatment are under the age of 59 months. Therefore, it would seem that this change in policy was not yet in place during the 2012 HNS.[48] Table 2.5 also indicates that this is clearly not a deterministic eligibility rule. Therefore, as will be explained below, I have chosen to use a fuzzy RD model and an IV model.

[47] As of October 2012 a statement (Ministerio de Inclusion Economica y Social, 2013) made by the head of State (President Rafael Correa) demonstrated a political will to change the public policy regarding eligibility by age from children under 59 to children under 36 months. The changes in the public policy literature began to appear in 2013 (Ministerio de Inclusion Economica y Social, 2013; Ministerio de Inclusion Economica y Social, 2013). Despite the survey being executed between 2011 and 2013 (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013), the data indicates that these changes seemed to not yet be underway while the survey took place.

[48] The National Health and Nutrition Survey (ENSANUT which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC) in Ecuador between 2011 and 2013.

Table 2.1 Treatment by 59 month cut-off among children who go to daycare

	Not treated	Treated	Total
Over 59 months	49	65	114
59 or under	509	1,211	1,720
Total	558	1,276	1,834

Source: Author's computation using 2012 Nutrition & Health Survey

Table 2.2 Treatment by 24 month cut-off point among children out of daycare

	Not treated	Treated	Total
Over 23 months	14,351	1,805	16,156
23 or under	2,552	1,495	4,047
Total	16,903	3,300	20,203

Source: Author's computation using 2012 Nutrition & Health Survey

Table 2.3 Treatment by 59 month cut-off point among children out of daycare centers

	Not treated	Treated	Total
Over 59 months	10,594	366	10,960
59 or under	6,309	2,934	9,243
Total	16,903	3,300	20,203

Source: Author's computation using 2012 Nutrition & Health Survey

Table 2.4 Treatment by 36 month cut-off point among all children

	Not treated	Treated	Total
Over 36 months	13,454	1,804	15,258
35 or under	4,007	2,772	6,779
Total	17,461	4,576	22,037

Source: Author's computation using 2012 Nutrition & Health Survey

Table 2.5 Treatment by 59 month cut-off point among all children

	Not treated	Treated	Total
Over 59 months	10,643	431	11,074
59 or under	6,818	4,145	10,963
Total	17,461	4,576	22,037

Source: Author's computation using 2012 Nutrition & Health Survey

Table 2.6 Treatment by Interval between 59 and 71 months among all children

	Not treated	Treated	Total
59 or under	6,945	4,184	11,129
Between 59 - 71	1,895	351	2,246
Over 71 months	8,621	41	8,662
Total	17,461	4,576	22,037

Source: Author's computation using 2012 Nutrition & Health Survey

2.3.3 The survey

The National Health and Nutrition Survey (ENSANUT[49] which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC[50]) in Ecuador between 2011 and 2013. It covers various health topics including participation in public nutritional supplementation programs. It has a total sample of 92,502 individuals[51] out of which 11,506 are children under the age of 5, and 31,293 are children under the age of 10. There was a sub-sample of 21,482 individuals, out of which 2,047 were children between 6 and 59 months and 5,372 were children 10 or under who had blood samples taken and on which various biomarkers were measured including Hb (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013).

2.2.3.1 The survey question about the cut-off

The survey question regarding receiving government issued nutritional supplements is formulated in such a way that leaves space for ambiguity which must be addressed before moving on. The text of the question is as follows (translation by author):

> "In the past 12 months from …[month beginning of period] to …[month of end of period] (…[Name of member of household]) is receiving or has received [during this period] benefits through the Nutritional Supplement Program (Sprinkles, iron, vitamin A, folic acid) FREE OF CHARGE FROM THE STATE [National government]?"[52] (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013)

The first issue pertains to the "In the past 12 months" (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013) section of the question: The fact that the question inquires on whether the child received treatment over the last twelve months directly affects the cut-off limit as it too is measured in age in months. For example, a child who is 71 months old (11 months older than the cut-off of 59 months) who received the treatment at the age of 59 months (11 months before the survey) will answer yes to this question despite being over the cut-off. This will happen without this representing a lack of enforcement of the cut-off. Similarly, a child who was 71 months old (11 months older than the cut-off of 59 months) and received

[49] Given its name in Spanish: Encuesta Nacional de Salud y Nutrición.
[50] Given its name in Spanish: Instituto Nacional de Estadísticas y Censos.
[51] 19949 households
[52] Text as it is written literally in survey in Spanish: "¿Durante los últimos 12 meses de ……. a …….. (…) recibe o recibió beneficios por el Programa Suplemento Nutricional (chispaz, hierro vitamina A, ácido fólico) GRATUITO, DEL ESTADO?"

the treatment at the age of 60 months (10 months before the survey) will also answer yes to this question and this will represent a lack of enforcement of the cut-off.

Therefore, it can be said that all the children who answer yes to this question up to the age of 71 months were potentially under the cut-off when they received the treatment. However, I cannot identify those who were and those who were not. In order to address this issue I run a Kernel-weighted local polynomial regression on either side of the 71 month cut-off and find that the probability of receiving the treatment falls to zero at 71 months (see Figure 2.5). Had there been no regard for the age cut-off, there would be no visible jump in the probability of receiving treatment at any age, let alone exactly 11 months after the age limit. I believe this jump is due precisely to the formulation of the question which stipulates participation over the last 12 months. Therefore, I assume 71 months accurately measures the cut-off point given the formulation of the survey question.

2.3.3.1 Type of supplement

The question asks if any member of the household received either "Sprinkles, iron, vitamin A or folic acid" (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013). As mentioned above, "sprinkles" contain iron, as well as other micronutrients,[53] therefore, I consider "iron" supplements to also refer to "sprinkles." By isolating the sample to children I can effectively eliminate the mothers who received folic acid.

However, the MSP does distribute vitamin A separately in the form of pills. Notwithstanding, the literature suggests that vitamin A deficiency is treated firstly with "sprinkles," as they contain 300µg RE of vitamin A, and, if necessary, to be used simultaneously with the pills which have "mega-doses" of vitamin A (15015,015µg RE) (Ministerio de Salud Publica, World Food Program, 2011).[54] Given there is no way to discern the exact source of the micronutrients and given the available information on treatment protocols I am assuming the children who answered yes to this question effectively received "sprinkles" (Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013).

2.3.3.2 Number of doses

Additionally, the 2012 HNS unfortunately does not provide information on the number of doses received by the children. Therefore, I am unable to discern if they completed the treatment or not. This lack of

[53] Including 12.5mg of Iron, 5mg of Zinc, 160µg of Folic Acid, 300µg RE of Vitamin A, and 30mg of Vitamin C.
[54] Given the MSP distributed iron and vitamin A supplements freely to all children under the age of 24 months, the daycare center sample may be composed of children who also received vitamin A on a routine checkup.

information might have a deterministic effect on our results. This implies I am testing the effect of the treatment on a sample of children who participated and received at least one dose in the past 12 months without knowing if they complied with the full treatment. The issue of non-compliance is dealt with in random control trials with an ITT model. ITT is a strategy used to analyze the results of a trial in which the individuals were initially randomized regardless of whether they completed the intervention (Armijo-Olivo, et al., 2009). Therefore, the ITT model gives an estimate of the effect of a change in treatment policy rather than an estimate of the effect of the treatment in patients who receive it exactly as planned (Hollis & Campbell, 1999). The models applied in this part of the study (RD and IV) use the eligibility rule as an instrument in order to identify the non-biased effect of the program. I believe this is analogous to using initial randomization of the program and, these models, like ITT models, estimate the effect of a change in treatment policy rather than of the effect of treatment on compliers.

2.3.3.3 Effect of altitude on Hb

Finally, the Hb measured in the survey is corrected for the altitude of the place of residence of the child. Altitude has been recognized to have an effect on Hb levels and on the red blood cell count. Specifically, a decline in oxygen partial pressure that occurs as altitude increases is normally associated with a decline in the oxygen saturation of arterial blood and an increased concentration of Hb. In the case of the children in the 2012 HNS the altitudes ranges from 0m to 3834m above sea level. Andean highlanders, such as those participating in these projects, who are exposed to high altitudes are generally characterized by high concentrations of Hb relative to individuals who live at sea level. This has been considered an adaptive response which allows the individual to maintain their oxygen supply under conditions of arterial hypoxia. This phenomenon can lead to an underestimation of the prevalence of IDA at high altitudes. In this study, I apply the correction proposed by Diren et al. (1994) who estimated corrections for Hb by altitude using Ecuador as an example (Dirren, et al., 1994).

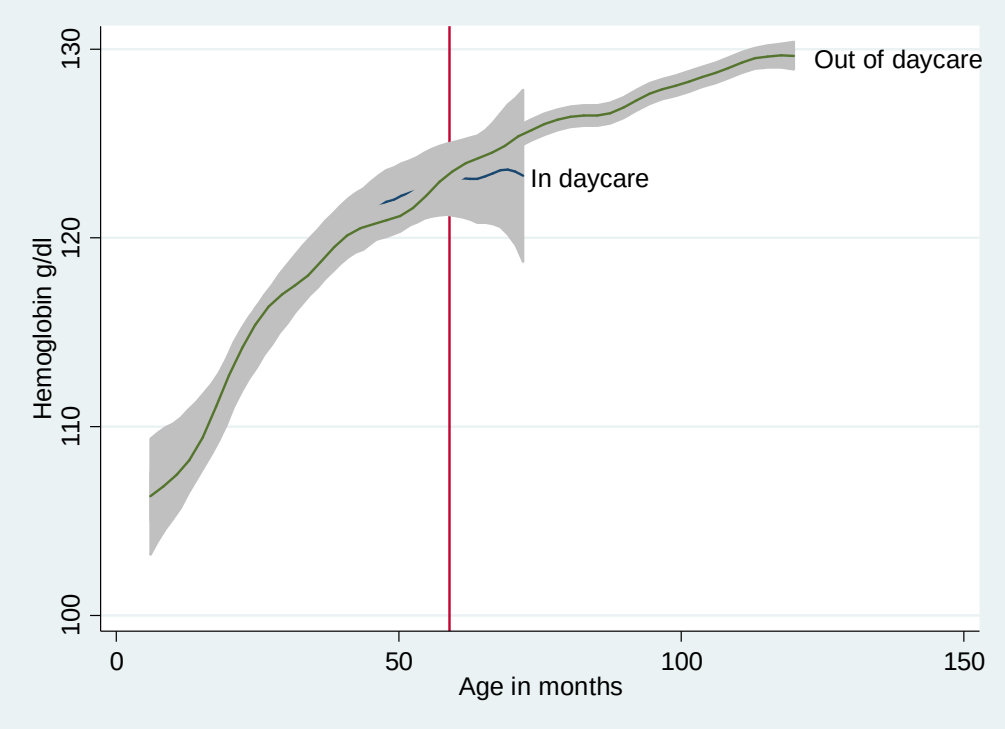
2.4 Empirical Strategy

2.4.1 Descriptive Statistics

In order to analyse the effect of the national iron supplement program on children under five I propose two separate methodologies. Firstly, a RD model that uses the age cut-off point (59 months) as the eligibility variable. In Figure 2.2 the outcome, Hb, is shown in blue for the MIES program and in green for the MSP program with a red line at the cut-off point. It is clear that the Hb levels increase with age and that they are

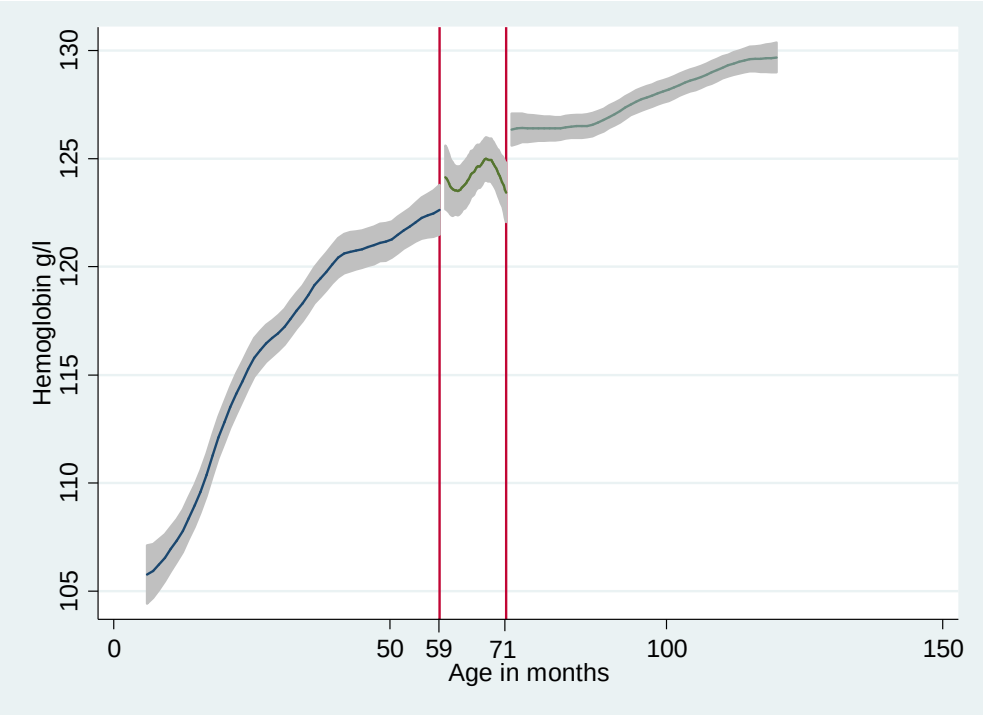
fairly similar across the two groups of children. In order to control for the effect of age I will attempt to make the bandwidth around the cut-off point as small as possible (e.g. as seen in Figure 2.3).

Figure 2.2 Hb levels by age in months



Source: Author's computation using 2012 Nutrition & Health Survey

Figure 2.3 Hb by treatment interval cut-off



Source: Author's computation using 2012 Nutrition & Health Survey

The descriptive statistics of the variables used in the RD model are presented in Table 2.7. Given a sample where the age eligibility cut-off point (71 months) can clearly be seen is needed, I include all children under the age of 120 months (10 years of age) in the sample. Secondly, I use an IV model with a long list of control variables in order to control for observables. Table 2.8 presents the descriptive statistics for the variables used in the IV model.

Table 2.7 descriptive statistics of variables used in RD models i.e. children under the age of 10

Variable	Obs	Mean	Std. Dev.	Min	Max
Hb g/l	5,286	122.8	11.18	64	208
D. Treatment	22,211	0.2	0.4	0	1
D. 59 months or under	22,519	0.5	0.5	0	1
D. 71 months or under	22,519	0.6	0.5	0	1
Age in months	22,519	59.42	35.17	0	120

Source: Author's computation using 2012 Nutrition & Health Survey

Table 2.8 descriptive statistics of variables used in IV model i.e. children under the age of 10

	Obs	Mean	Std.Dev.	Min	Max
Hb (g/l)	5230	122.68	11.19	64	208
D. Treat	22037	0.2	0.4	0	1
Age in months	22344	59.43	34.89	0	120
Age in months 2	22344	4749.82	4325.47	0	14400
D. Diarrhea	10473	0.16	0.36	0	1
D. Public daycare	22037	0.08	0.27	0	1
D. Female	22344	0.49	0.49	0	1
Hb mother	8579	127.43	10.99	51	160
Hb mother 2	8579	16360.87	2666.36	2601	25600
Schooling mother	21193	8.68	3.98	0	20
D. access maternity healthcare	22037	0.12	0.32	0	1
D. mother employed	21041	0.45	0.49	0	1
Ln(income per capita)	21189	4.35	0.89	0	8.111727
D. Extreme poverty	22344	0.28	0.45	0	1
D. Indigena	22037	0.14	0.35	0	1
D. Afro	22037	0.04	0.2	0	1
D. Montubio	22037	0.02	0.16	0	1
D. Urban Highlands	22344	0.2	0.4	0	1
D. Rural Highlands	22344	0.19	0.39	0	1
D. Urban Cost	22344	0.13	0.33	0	1
D. Rural Coast	22344	0.06	0.24	0	1
D. Urban Amazon	22344	0.11	0.32	0	1
D. Rural Amazon	22344	0.17	0.38	0	1
D. Galapagos	22344	0.02	0.15	0	1
D. Guayaquil	22344	0.03	0.18	0	1
D. Under 71 months old	22344	0.6	0.48	0	1

Source: Author's computation using 2012 Nutrition & Health Survey

2.4.2 Regression Discontinuity

RD is a policy evaluation method which uses eligibility rules as instruments to exogenously identify participants and non-participants. The individuals who fall within a certain neighborhood of the eligibility rule, that is, just above or just below the cut-off point, are used as a relevant sample for estimating the treatment impact. This method allows for observed as well unobserved heterogeneity (Shahidur, et al., 2010).

2.4.2.1 The model

If there is a variable S_i which determines program eligibility (such as age) with an eligibility cut-off at s^* (59 months) it is possible to model the effect of a program on individual outcomes y_i using the RD method. This allocation mechanism generates a non-linear relation between participation and age in months. In general, the estimating equation is $y_i = \beta S_i + \varepsilon_i$, where individuals with $s_i \leq s^*$ receive the program and individuals with $s_i > s^*$are not eligible to participate. If it can be assumed 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 (Shahidur, et al., 2010):

$$E[y_i|s^* - \varepsilon] - E[y_i|s^* + \varepsilon] = E[\beta S_i|s^* - \varepsilon] - E[\beta S_i|s^* + \varepsilon] \quad \textbf{(2.1)}$$

When taking the limit of both sides of (2.1) as $\varepsilon \to 0$, β is identified 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 (Shahidur, et al., 2010):

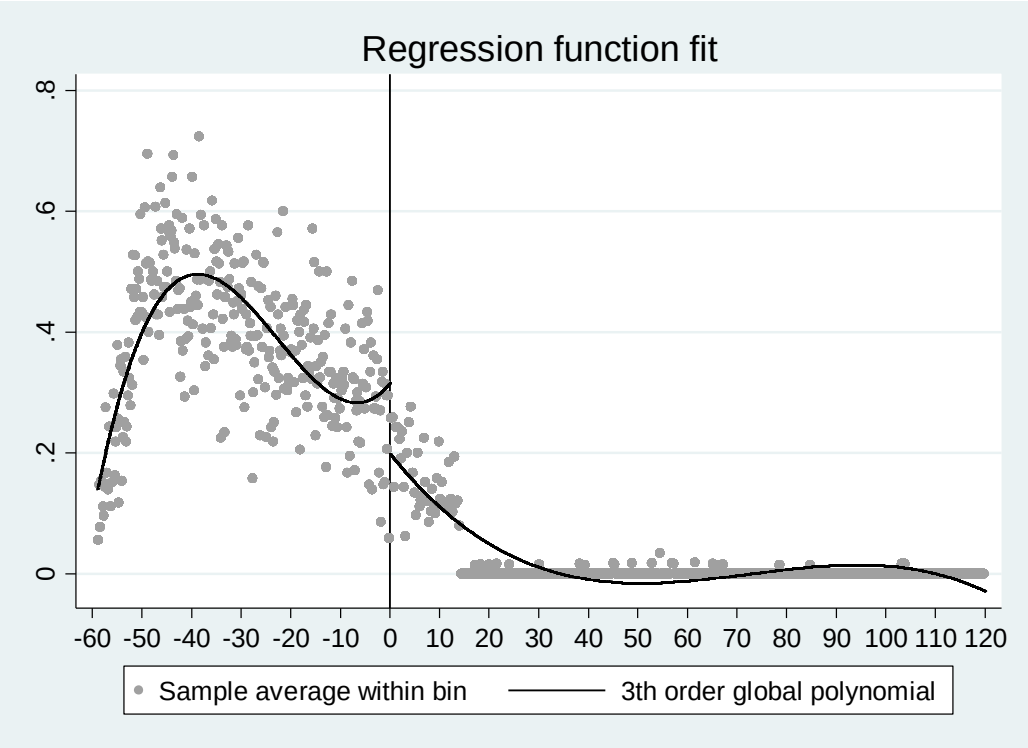
$$\lim_{\varepsilon \to 0} E[y_i|s^* - \varepsilon] - \lim_{\varepsilon \to 0} E[y_i|s^* + \varepsilon] = y^- - y^+ = \beta(S^- - S^+) \Rightarrow \beta = \frac{y^- - y^+}{S^- - S^+} \quad \textbf{(2.2)}$$

If individuals were assigned to treatment solely on the basis of the assignment variable, T would be deterministic which in this case, is unlikely. There is a large degree of "fuzzyness" in program assignment. Therefore, assignment to treatment status depends on age in a stochastic manner. To estimate the treatment effect in the presence of fuzzy discontinuity s is replaced with the probability of participating $\mathrm{P}(S) = E(T|S)$ where $T = 1$ if individual receives treatment and $T = 0$ if individual does not receive treatment. In this case, the discontinuity is stochastic; instead of measuring differences above and below s^*, the impact estimator will measure the difference in outcomes around a neighborhood of s^* (Shahidur, et al., 2010).

2.4.2.2 The cut-off

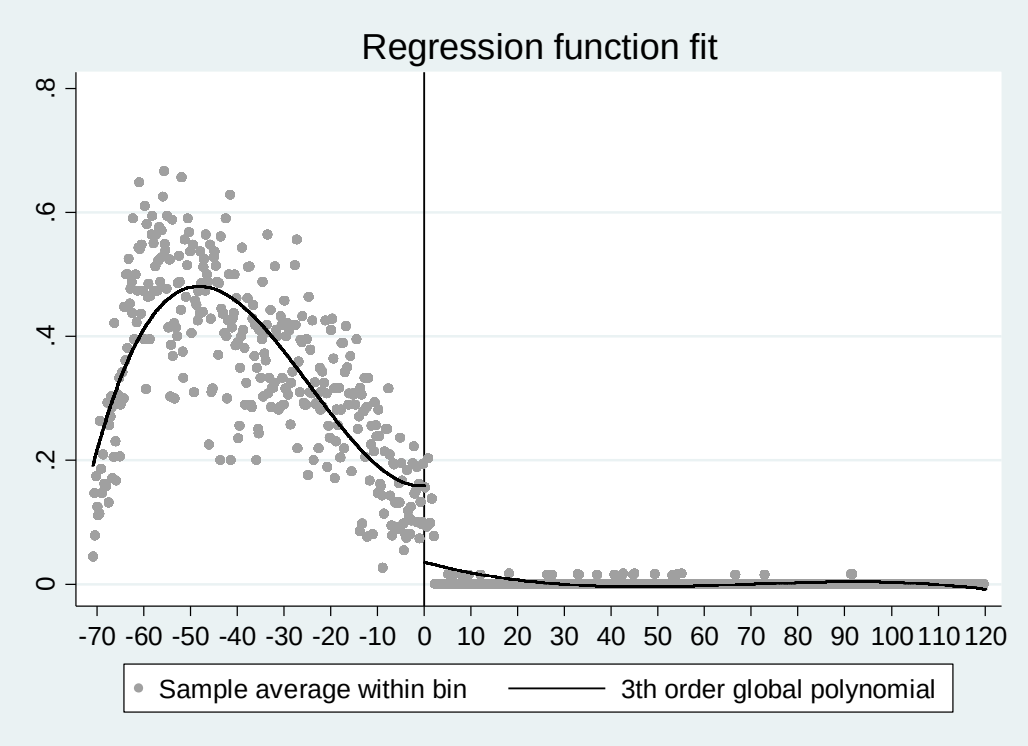
Figure 2.4 presents the discontinuity in the probability of treatment and outcome with two separate Kernel-weighted local polynomial regressions, one on either side of the cut-off point. As explained above, I include the children from all programs pooled together and include children up to 71 months old as those who potentially received treatment before the cut-off of 59 months. Figure 2.4 and Figure 2.5 present the discontinuity with both the 59 month and the 71 month cut-off respectively where a jump down in the probability of receiving treatment after 71 months can clearly be seen.

Figure 2.4 Probability of treatment by cut-off point, 59 months



Source: Author's computation using 2012 Nutrition & Health Survey

Figure 2.5 Probability of treatment by cut-off point, 71 months



Source: Author's computation using 2012 Nutrition & Health Survey

Concerns with RD include the possibility that eligibility rules will not be adhered to consistently, as well as the potential for them to change over time. In this paper fuzzy RD models will be applied in order to address the former concern. In relation to the latter, above I show how the standard may have recently changed, however, the data demonstrates that the 59 month cut-off point seems to have still been the eligibility rule while the survey was made.

2.4.3 Instrumental variables without heterogeneous effects

The second methodology, an IV model is used. The main difference between the fuzzy RD model and the IV model is the bandwidth around the cut-off applied in the former. It can be proven that the fuzzy RD is a specific case of the IV model (Calonico, et al., 2014). The IV models are presented in order to see the effect of the treatment on all the treated children, including the ones who are "far" from the cut-off (Shahidur, et al., 2010).

The age cut-off is an appropriate instrument given it complies with the two requirements of the exclusion restriction. Firstly, Z (our instrument) is correlated with T (treatment): $\text{cov}(Z, T) \neq 0$. The probability of

receiving treatment after 71 months is virtually zero (see Figure 2.3), however, and most importantly, our first stage probit regression shows the instrument is highly significant and it has a z-value of 4.22 (see Appendix 2). Secondly, I propose Z is uncorrelated with ε, $\text{cov}(Z, \varepsilon) = 0$ because the cut-off randomly assigns children to treatment and control groups as a function of a characteristic they cannot change.

This method allows for endogeneity in both individual participation and program placement. When treatment assignment is not random, endogeneity exists either due to program targeting or to unobserved individual heterogeneity arising from individuals self-selecting into or out of the program. The result will be a correlation between ε and T, $\text{cov}(T, \varepsilon) \neq 0$, and therefore a violation of one key assumption[55] of OLS.[56] This naturally results in a bias of the program effect β. (Shahidur, et al., 2010).

To isolate the treatment variable that is independent of other unobserved characteristics an estimation in two stages is set up. The first stage regression (2.3) will regress the treatment on the instrument Z, other covariates and a disturbance u_i. The resulting predicted $\hat{T}$ reflects the part of the treatment affected only by Z and therefore represents only the exogenous variation in the treatment. The second stage regression would regress the outcome Y_i on the predicted treatment $\hat{T}_i$ and other covariates X_i and can be found in (2.4). The reduced form regression which embodies both the first stage and second stage regressions is (2.5) (Shahidur, et al., 2010):

$$\hat{T}_i = \gamma Z_i + \phi X_i + u_i \quad \textbf{(2.3)}$$

$$Y_i = \alpha X_i + \beta \hat{T}_i + \varepsilon_i \quad \textbf{(2.4)}$$

$$Y_i = \alpha X_i + \beta\left(\hat{\gamma} Z_i + \hat{\phi} X_i + u_i\right) + \varepsilon_i \quad \textbf{(2.5)}$$

The program impact is then $\hat{\beta}_{\text{IV}}$. Taking into account the assumption $\text{cov}(Z, \varepsilon) = 0$, the treatment effect can also be written as follows. If assumptions $\text{cov}(Z, T) \neq 0$ and $\text{cov}(Z, \varepsilon) = 0$ hold then the IV model consistently identifies the mean impact of the program given it can be shown that $\hat{\beta}_{\text{IV}} = \beta + \frac{\text{cov}(Z,\varepsilon)}{\text{cov}(Z,T)}$ (Shahidur, et al., 2010):

$$\text{cov}(Y_i, Z_i) = \text{cov}[(\beta T_i + \varepsilon_i), Z_i] = \beta \text{cov}(T_i, Z_i) \quad \textbf{(2.6)}$$

[55] The assumption of independence of regressors and the disturbance term ε.
[56] in obtaining unbiased estimates

$$\Longrightarrow \frac{\text{cov}(Y_i, Z_i)}{\text{cov}(T_i, Z_i)} = \beta \quad \textbf{(2.7)}$$

In our case, the endogenous variable (T) is a binary (dummy treatment). The *ivtreatreg* command on **STATA** is used and the probit-2SLS option is specified. This model is constructed as follows:

(1) It runs a probit first stage regression of T on (z, x) predicting p_t (probability of treatment).

(2) It runs an OLS regression of T on (1, x, p_t,) obtaining fitted values of $\widehat{T_i}$

(3) It runs a second stage OLS regression of y on (1, x, $\widehat{T_i}$).

The coefficient of $\widehat{T_i}$is the most efficient estimator of average treatment effect for T (Cerulli, 2012).

2.4.4 Instrumental variables with heterogeneous effects

As mentioned above, the *ivtretreg* command on **STATA** is used to estimate the IV model. This command also allows for heterogeneous effects. In this section I explain how the model and the command estimate these effects. In this paper, the heterogeneous effects are quantiles. Therefore, the *ivqte* command in **STATA** could have been used. However, this command does not estimate heterogeneous effects, rather it estimates the effect of the treatment on one quantile. It also does not estimate the standard errors directly, making it difficult to interpret the results.

In order to see if the treatment has an idiosyncratic (heterogeneous) average treatment effect on children with (low)high levels of Hb I introduce a third class of models with heterogeneous effects for quantiles of the dependent variable (Hb). In a model with one heterogeneous effect there are two endogenous variables: (1) T, and, (2) $T * \text{Hb\_quantile}$.

In this section I will go over how our model produces a consistent estimator with two endogenous variables by summarizing Cerulli (2012).

The predicted outcome is:

$$y_i = y_{\text{control},\, i} + T\,(y_{\text{control},i} - y_{\text{treat},\, i}) \quad \textbf{(2.8)}$$

The ATE is:

$$ATE = E(y_{\text{treat}} - y_{\text{control}}) \quad \textbf{(2.9)}$$

The ATE with observable confounding conditioning factors:

$$ATE = E(y_{\text{treat}} - y_{\text{control}} \mid \mathbf{x}) \quad \textbf{(2.10)}$$

If there is only selection on observables then

$$E(y_{\text{control}} \mid \mathbf{x}, \mathbf{T}) = E(y_{\text{control}} \mid \mathbf{x}) \quad \textbf{(2.11)}$$

$$E(y_{\text{treat}} \mid \mathbf{x}, \mathbf{T}) = E(y_{\text{treat}} \mid \mathbf{x}) \quad \textbf{(2.12)}$$

$$y_{\text{treat}} = \mu_{\text{treat}} + v_{\text{treat}} \quad \textbf{(2.13)}$$

$$y_{\text{control}} = \mu_{\text{control}} + v_{\text{control}} \quad \textbf{(2.14)}$$

$$E(v_{\text{treat}}) = 0 \quad \textbf{(2.15)}$$

$$E(v_{\text{control}}) = 0 \quad \textbf{(2.16)}$$

The expected outcome is:

$$y = y_{\text{control}} + T\,(y_{\text{control}} - y_{\text{treat}}) \quad \textbf{(2.17)}$$

$$y = \mu_{\text{control}} + T\,(\mu_{\text{control}} - \mu_{\text{treat}}) + v_{\text{control}} + T\,(v_{\text{control}} - v_{\text{treat}}) \quad \textbf{(2.18)}$$

With non-random assignment and selection on unobservables to treatment it cannot be assumed that (2.15) or (2.16) is true, therefore:

$$E(v_{\text{treat}}) = E(v_{\text{treat}} \mid \mathbf{x}) = f_{\text{treat}}(\mathbf{x}) + e_{\text{treat}} \quad \textbf{(2.19)}$$

$$E(v_{\text{control}}) = E(v_{\text{control}} \mid \mathbf{x}) = f_{\text{control}}(\mathbf{x}) + e_{\text{control}} \quad \textbf{(2.20)}$$

$$y = \mu_{\text{control}} + \alpha T + v_{\text{control}} + T\,(v_{\text{control}} - v_{\text{treat}}) \quad \textbf{(2.21)}$$

Where $\alpha=(\mu_{\text{control}} - \mu_{\text{treat}})$

$$y = \mu_{\text{control}} + \alpha T + f_{\text{control}}(\mathbf{x}) + T\,[f_{\text{treat}}(\mathbf{x}) - f_{\text{control}}(\mathbf{x})] + e_{\text{control}} + T(e_{\text{treat}} - e_{\text{control}}) \quad \textbf{(2.22)}$$

Assuming $f_{\text{control}}(\mathbf{x}) = \mathbf{x}\boldsymbol{\beta}_{\mathbf{control}}$ and $f_{\text{treat}}(\mathbf{x}) = \mathbf{x}\boldsymbol{\beta}_{\mathbf{treat}}$ we have

$$y = \mu_{\text{control}} + \alpha T + \mathbf{x}\boldsymbol{\beta}_{\mathbf{control}} + T\,(\mathbf{x} - \boldsymbol{\mu}_{\boldsymbol{x}})\boldsymbol{\beta} + e_{\text{control}} + T(e_{\text{treat}} - e_{\text{control}}) \quad \textbf{(2.23)}$$

Adding and subtracting $E[T(e_{\text{treat}} - e_{\text{control}})]$

$$\begin{aligned} y = \mu_{\text{control}} + E[T(e_{\text{treat}} - e_{\text{control}})] + \alpha T + \mathbf{x}\boldsymbol{\beta}_{\mathbf{control}} + T\,(\mathbf{x} - \boldsymbol{\mu}_{\boldsymbol{x}})\boldsymbol{\beta} + e_{\text{control}} \\ + T(e_{\text{treat}} - e_{\text{control}}) - E[T(e_{\text{treat}} - e_{\text{control}})] \end{aligned} \quad \textbf{(2.24)}$$

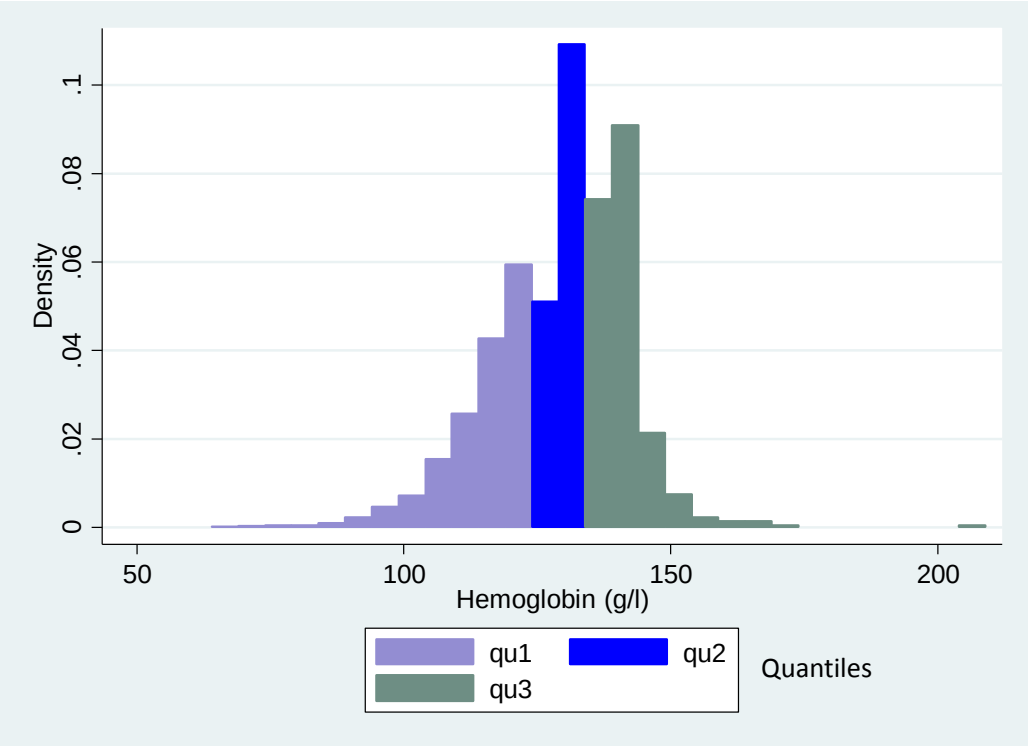
If the instrument is exogenous then it can be assumed $E[e_{\text{control}} + T(e_{\text{treat}} - e_{\text{control}}) - E[T(e_{\text{treat}} - e_{\text{control}})] \mid \mathbf{x}, \mathbf{z}] = 0$. This implies that any function of $(\mathbf{x}, \mathbf{z})$ can be used as an instrument in the y-equation.

$$y = \eta + \alpha T_i + \boldsymbol{x_i}\boldsymbol{\beta}_{\mathbf{control}} + T\,(\mathbf{x_i} - \boldsymbol{\mu}_{\boldsymbol{x}})\boldsymbol{\beta} + error \quad \textbf{(2.25)}$$

When heterogeneous effects are included there are have two endogenous variables T and $T\,(\mathbf{x} - \boldsymbol{\mu}_{\boldsymbol{x}})$. As explained above I use $(1, x, p_t,)$ as instruments of T in the model with no heterogeneous effects and I use $(1, x, p_t, p_t(\mathbf{x_i} - \boldsymbol{\mu}_{\boldsymbol{x}}))$ as instruments of T and $T\,(\mathbf{x} - \boldsymbol{\mu}_{\boldsymbol{x}})$ in the model with heterogeneous effects (Cerulli, 2012). In both IV models (with and without heterogeneous effects) additional first stage OLS regressions are presented. Here, the F-statistic on excluded instruments is shown to demonstrate they are not weak.

We estimate the heterogeneous effect of treatment on three quantiles of the distribution of Hb. Figure 2.6 shows the distribution of Hb and the quantiles. I chose three quantiles because I would like the measure the effect on the lowest and highest levels of Hb in relation to the "mean" effect.

Figure 2.6 Distribution of Hb (g/l) among children < 120 months with quantiles



Source: Author's computation using 2012 Nutrition & Health Survey

2.5 The results

2.5.1 Regression discontinuity

As mentioned above (Figure 2.2, 2.3), I consider the smallest bandwidths as the least biased given the effect of age on Hb levels. Table 2.9 presents linear, quadratic and cubic models with robust confidence intervals as proposed by Calonico et al. (2014). This estimation is presented for various bandwidths around the cut-off point which in our case is measured in age in months. For example, a bandwidth of 2 would include children 2 months younger and 2 months older than 71 months (Calonico, et al., 2014).

Table 2.9 shows none of the estimations are significant. Hb is measured in g/l, therefore, the magnitudes of the coefficients are within a reasonable range. Despite being insignificant, I will take a paragraph to explain the magnitude of the coefficients to show they are within a reasonable range. For children from 6 to 59 months of age the minimum non-anaemic levels of Hb is 110 g/l. When the bandwidth is one month, the linear coefficient is -8.295, which would imply a reduction in levels of Hb of around 8 g/l. For a child with a Hb level of 110g/l this would imply a drop to 102 g/l. This point is important to clarify as sometimes Hb

is reported in g/dl. The limit of anaemia would then be defined as 11.0 g/dl. In this case, the 8g/l coefficient would be equivalent to a 0.8 g/dl drop, which would imply a reduction form 11.0g/dl to 10.2g/dl which is a large drop but not an unreasonable quantity. In any case, as mentioned above, the drop is not significant and therefore the actual drop in Hb is zero.

Most of the linear estimations are positive. However, some of the coefficients are negative. Furthermore, there are more negative coefficients in the quadratic and cubic models. I cannot explain this phenomenon except to say that they are all not significant and therefore actually represent a zero change in Hb levels.

Table 2.9 Fuzzy regression discontinuity results

Bandwidth	Left	Right	Linear		Quadratic		Cubic	
			Coef.	Std. Err.	Coef.	Std. Err.	Coef.	Std. Err.
1 month	38	49	-8.2591	10.407	-6.766	13.568	-3.48	13.245
2 months	93	93	-3.0989	10.338	-4.85	9.55	-8.303	11.667
3 months	145	141	0.382	14.002	-3.317	9.07	-7.26	10.515
4 months	193	189	0.316	18.402	-0.872	10.679	-4.679	9.009
3 months	241	229	1.87	18.508	-1.434	14.361	-2.3727	8.96
6 months	297	286	1.34	17.59	0.720	17.418	-2.26	10.7
7 months	348	334	4.138	17.09	-1.803	18.577	-0.34	12.981
8 months	401	375	5.88	16.32	-2.167	19.08	-0.795	15.034
9 months	453	419	6.688	16.872	0.8132	17.887	-3.83	17.58
10 months	496	479	4.19	15.665	6.401	19.779	-6.0107	17.658
11 months	540	513	1.82	15.381	9.059	20.493	-4.726	18.581
12 months	602	541	0.053	14.649	9.0912	19.921	-0.632	18.18

Source: Author's computation using 2012 Nutrition & Health Survey

2.5.2 Instrumental variables

In previous section (Fuzzy RD model), I focus exclusively on individuals closely situated around s*. In the case of nutritional supplements, it may also be interesting to determine the effect of treatment on a sample which includes children who are "far" from the cut-off point. Particularly, children who are younger. In order to do this I introduce an IV model in this section (Shahidur, et al., 2010).

The results are presented as follows: firstly, Table 2.10 has the second stage regression which presents seven models. Model 1 is the simple probit-2 stage least squares (2SLS) where there are no homogeneous effects. The IV models with heterogeneous effects are shown in models 2 to 7. In models 2 to 4 I present models with 1 heterogeneous effect, one for each quantile, while, in models 5 to 7 each model has 2 heterogeneous effects i.e. 2 quantiles per model.

Given models 2 to 4 have one heterogeneous effect, they each have two endogenous variables (1) dummy treatment, and (2) the interaction between dummy treatment and dummy quantile. Therefore, there are two instruments in these models (2 to 4): the first instrument is the cut-off (dummy child under 71 months of

age) and the second instrument is the interaction between the cut-off and dummy quantile. Each instrument has its own first stage OLS regression. Table 2.11.A presents the first stage estimation of dummy treatment using the cut-off (dummy child under 71 months of age) as an instrument. Table 2.11.B presents the first stage estimation of the interaction between dummy treatment and dummy quantile, using the interaction between dummy cut-off and dummy quantile as an instrument.

Likewise, given models 5 to 7 have 2 heterogeneous effects, they each have three endogenous variables: (1) dummy treatment, (2) the interaction between dummy treatment and the first selected quantile, and (3) the interaction between dummy treatment and the second selected quantile. Similarly, there are three instruments in these models (1) dummy cut-off, (2) interaction between dummy cut-off and dummy first selected quantile, (3) interaction between dummy cut-off and dummy second selected quantile. Again, Table 2.11.A and 2.11.B presents the first stage regressions for the estimation of dummy treatment and the interaction between dummy treatment and dummy quantile respectively, and, finally, Table 2.11.C presents the first stage regression estimation of the interaction between dummy treatment and the second selected quantile using interaction between dummy cut-off and dummy second selected quantile as an instrument.

In every model controls for extreme poverty and access to public daycare centers are included, as well as for characteristics of the child, the mother, the household as control variables as well as fixed effects for ethnicity and region.

The average treatment effect, found in model 1, is negative and non-significant (-2.03 g/l). When heterogeneous effects are included, the dummy treatment coefficient becomes positive. The way the dummy treatment coefficient is interpreted depends on the model. Given quantiles of the dependent variable are included, it can no longer be said that the dummy treatment is the average treatment effect. It is rather, the effect on the group of children who do not fall into the category of the heterogeneous effect.

In model 2, a heterogeneous effect for quantile 1 of the Hb distribution is included. Here the dummy treatment coefficient is the effect on all children in quantile 2 and quantile 3, which is positive but not significant (3.6 g/l). The effect on the children in quantile 1 is negative and significant (-12.3 g/l). A similar effect should be found in model 6, where two heterogeneous effects are included (quantile 2 and quantile 3), leaving quantile 1 as the reference group. This means that the dummy treatment coefficient in model 6 should reflect the effect on quantile 1, however here the effect is positive and significant (5.7 g/l).

Something similar happens when trying to identify the effect on quantile 2. In model 2 one heterogeneous effect is included (quantile 2), and find the coefficient is positive and significant (24.8 g/l) while the dummy

treatment coefficient is positive but not significant (4.2 g/l). In model 7 two heterogeneous effects are included (quantile 1 and quantile 3), allowing quantile 2 to be the reference group. Here the dummy treatment coefficient represents the effect of the treatment on children in quantile 2, it is also positive and significant, however, the magnitude is virtually identical to that found in model 6 (5.7 g/l).

Finally, I find the same behaviour when measuring the effect on quantile 3. In model 3, where it is the one heterogeneous effect, its coefficient is positive but not significant (17.3 g/l) while the dummy treatment coefficient, which represent the effect on children in quantile 1 and 2, is positive but not significant (1.05 g/l). In model 5, where there are two heterogeneous effects (quantile 1 and 2) the dummy treatment dummy reflects the effect on quantile 3, however, it is positive and significant and has the same magnitude as model 6 and model 7 (5.7 g/l).

Table 2.10 Instrumental Variable results (with probit model in first stage)

	IV1	IV2	IV3	IV4	IV5	IV6	IV7
	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)
D. Treat	**-2.03**	**3.62**	**4.22**	**1.06**	**5.75****	**5.75****	**5.75****
	(2.81)	(2.45)	(2.83)	(2.73)	(2.43)	(2.43)	(2.43)
D. Treat * quantile 1		**-12.3*****			**-10.97**		**-16.57*****
		(2.85)			(11.26)		(3.24)
D. Quantile 1		-12.3***			-19.3***		-10.2***
		(0.37)			(0.65)		(0.32)
D. Treat * quantile 2			**24.8*****		**5.6**	**16.6*****	
			(4.07)		(11.5)	(3.24)	
D. Quantile 2			5.4***		-9.1***	10.2***	
			(0.49)		(0.6)	(0.32)	
D. Treat * quantile 3				17.3100		10.9650	-5.6076
				(14.55)		(11.26)	(11.5)
D. Quantile 3				13.6121***		19.3413***	9.1169***
				(0.68)		(0.65)	(0.6)
D. Extreme poverty	0.7937	0.1389	0.6526	-0.0202	-0.2121	-0.2121	-0.2121
	(0.88)	(0.66)	(0.82)	(0.8)	(0.64)	(0.64)	(0.64)
D. Daycare	-0.7847	-2.201	-1.9367	-1.2633	-2.6499	-2.6499	-2.6499
	(1.89)	(1.61)	(1.66)	(1.9)	(1.63)	(1.63)	(1.63)
Z-score height for age	0.1782	0.0432	0.1918	-0.0059	-0.0585	-0.0585	-0.0585
	(0.25)	(0.16)	(0.22)	(0.22)	(0.16)	(0.16)	(0.16)
Age in months	0.4560***	0.3545***	0.3904***	0.4462***	0.3633***	0.3633***	0.3633***
	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Age in months ^2	-0.0021***	-0.0018***	-0.0018***	-0.0021***	-0.0019***	-0.0019***	-0.0019***
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
D. Diarrhea	-0.1359	0.0089	-0.0626	-0.4785	-0.26	-0.2600	-0.26
	(0.7)	(0.55)	(0.68)	(0.62)	(0.53)	(0.53)	(0.53)
D. Female	0.8162*	0.5535	0.7364	0.4775	0.3553	0.3553	0.3553
	(0.48)	(0.38)	(0.46)	(0.43)	(0.36)	(0.36)	(0.36)
Hb. mother	-0.9055***	-0.1281	-0.4918*	-0.8948***	-0.214	-0.2140	-0.214
	(0.3)	(0.21)	(0.27)	(0.28)	(0.2)	(0.2)	(0.2)
Hb. mother ^2	0.0043***	0.001	0.0026**	0.0042***	0.0013	0.0013	0.0013
	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)	(0.00)
Schooling mother	0.2530***	0.1503**	0.2049***	0.2214***	0.1423**	0.1423**	0.1423**
	(0.07)	(0.06)	(0.07)	(0.06)	(0.05)	(0.05)	(0.05)
D. Free maternal healthcare	-1.6833	-1.3624	-1.6094	-1.6069	-1.419	-1.4190	-1.419
	(1.14)	(1.03)	(1.09)	(1.11)	(1.03)	(1.03)	(1.03)
D. Mother employed	-0.1149	0.3148	-0.0433	0.3121	0.5069	0.5069	0.5069
	(0.5)	(0.39)	(0.48)	(0.44)	(0.36)	(0.36)	(0.36)
Ln hh income pc	0.4509	0.0589	0.4270	-0.0402	-0.1735	-0.1735	-0.1735
	(0.43)	(0.32)	(0.42)	(0.39)	(0.31)	(0.31)	(0.31)
D. Indigenous	-3.4065***	-3.0079***	-3.1331***	-2.8189***	-2.5806***	-2.5806***	-2.5806***
	(0.97)	(0.78)	(0.89)	(0.92)	(0.76)	(0.76)	(0.76)
D. Afro-Ecuadorian	-1.7952	-1.2311	-1.5681	-1.2741	-0.9389	-0.9389	-0.9389
	(1.58)	(1.22)	(1.43)	(1.51)	(1.23)	(1.23)	(1.23)
D. Montubio	0.8262	-0.4821	-0.3379	1.1029	-0.3379	-0.3379	-0.3379
	(1.05)	(0.88)	(1.08)	(0.94)	(0.85)	(0.85)	(0.85)
D. Urban highlands	0.5757	-0.7783	-0.5611	1.2483	-0.6239	-0.6239	-0.6239
	(1.4)	(1.16)	(1.39)	(1.39)	(1.21)	(1.21)	(1.21)
D. Rural highlands	1.1777	0.115	0.3960	1.5155	0.0061	0.0061	0.0061
	(1.44)	(1.13)	(1.41)	(1.41)	(1.19)	(1.19)	(1.19)
D. Urban coast	1.3877	0.4624	0.6511	1.9845	0.6241	0.6241	0.6241
	(1.34)	(1.11)	(1.32)	(1.34)	(1.17)	(1.17)	(1.17)
D. Rural coast	0.1335	-0.7468	-0.4734	1.0322	-0.284	-0.2840	-0.284
	(1.53)	(1.26)	(1.5)	(1.5)	(1.3)	(1.3)	(1.3)
D. Urban amazon	1.2333	0.2439	0.6619	1.8567	0.4041	0.4041	0.4041
	(1.55)	(1.23)	(1.51)	(1.51)	(1.28)	(1.28)	(1.28)
D. Rural amazon	1.2445	0.8749	0.8468	1.422	0.5496	0.5496	0.5496
	(1.57)	(1.22)	(1.5)	(1.51)	(1.27)	(1.27)	(1.27)
D. Galapagos	3.5439**	1.6808	2.2022	4.3274***	2.0132	2.0132	2.0132
	(1.74)	(1.33)	(1.68)	(1.65)	(1.36)	(1.36)	(1.36)
D. Guayaquil	1.7943	-0.6025	-0.0390	3.0258*	0.1551	0.1551	0.1551
	(1.57)	(1.34)	(1.57)	(1.55)	(1.38)	(1.38)	(1.38)
_cons	142.9664***	115.2847***	120.3754***	145.4320***	128.5967***	109.2554***	119.4798***
	(19.53)	(13.2)	(17.46)	(18.36)	(13.1)	(13.13)	(13.1)
r2	0.5156	0.7019	0.5556	0.6088	0.7295	0.7295	0.7295
N	1344	1344	1344	1344	1344	1344	1344

Source: Author's computation using 2012 Nutrition & Health Survey

A partial explanation of the discrepancy may be found in the first stage regressions in Table 2.11. Here, a different first-stage regression than the one used to make the estimation of the IV models, is presented. The first stage here is a simple OLS regression for each instrument (in models 2 - 4 there are two instruments: one for the dummy treatment, one for the quantile heterogeneous effect, and in models 5 - 7 there are three instruments: one for the dummy treatment and one for each of the two quantile heterogeneous effects) where the F-statistic of the excluded instruments can be seen. In models 1 (no heterogeneous effects) and 2 - 4 (one heterogeneous effect) the F-statistic for the first-stage of the dummy treat is above 10. However, in the models (5 - 7) with more than three instruments, the F-statistic for the first-stage estimation of dummy treatment is under 10. This leads us to the conclusion that the dummy treatment coefficient in models 5 - 7 are based on a weak instrument. Therefore, it is best to rely on the estimations in models 2 - 4 in the identification of the effect in each quantile.

Therefore, the effect on average is negative and not significant (-2.03 g/l). This would imply a reduction from, say the minimum 110 g/l to 108 g/l or alternatively in g/dl a reduction from 11.0 g/dl to 10.8 g/dl if the effect was significant, which it is not. The effect on the children with the lowest levels of Hb is negative and significant (-12 g/l) which implies a reduction in Hb from, for example, 110 g/l to 98 g/l or from 11.0 g/dl to 9.8 g/dl. Again, I cannot explain the negative sign on the coefficient other than to state that this might be a consequence of diarrhea caused by the treatment and/or a failure to comply with the total needed doses. As explained above, this model is analogous to an ITT model, where the effect of a random assignment to the treatment group i.e. the effect of being selected, is measured rather than the effect of the treatment on those who comply with the entire treatment protocol. Notwithstanding, in this model (2) the dummy treatment coefficient becomes positive (albeit not significant) indicating the negative sign in model 1 might be driven by the negative effect among the children with the lowest Hb levels. Once a control for this effect is added, the sign becomes positive.

The effect of including quantile 2 in model 3 is similar on the dummy treatment coefficient, as it remains positive (not significant). The effect on quantile 2 is positive and significant (24 g/l), implying an increase in Hb from say, 110 g/l to 134 g/l or 11.0 g/dl to 13.4 g/dl. In model 4 a heterogeneous effect for quantile 3 is included, I find the dummy treatment coefficient is positive (not significant). Here the effect on quantile 3 is positive but not significant (17 g/l). Again, it seems reasonable to think that the negative sign in model 1 is driven mainly by the negative effect on children with low Hb levels. The rest of the children seem to enjoy either a positive and significant effect (those with mean Hb) or a positive non-significant effect (those with high Hb levels).

Table 2.11 First Stage results with OLS model in first stage

A. First instrument

First Instrument		OLS1: No hetero.		OLS2: Quant1		OLS3: Quant 2		OLS4: Qunat 3		OLS5: Qunat 1, Quant 2		OLS6: Qunat 2, Quant 3		OLS7: Qunat 1, Quant 3	
Endogenous		D treat		D. treat		D. treat		D. treat		D. treat		D. treat		D. treat	
Instrument		D71		D 71, D 71*D q1		D 71, D 71*D q2		D 71, D 71*D q3		D 71, D 71 * D q1, D 71 * D q2		D 71, D 71 * D q2, D 71 * D q3		D 71, D 71 * D q2, D 71 * D q3	
F value		28.74		26.58		26.61		26.74		24.87		24.87		24.87	
F value instruments		22.5		11.26		11.55		12.6		**8.51**		**8.51**		**8.51**	
	b	s.e.	b	s.e.	b	s.e.	b	s.e.	b	s.e.	b	s.e.	b	s.e.
Qunatile 1			-0.005	(0.02)					-0.005	(0.03)			-0.005	(0.02)
Qunatile 2					0.004	(0.02)			0	(0.03)	0.005	(0.02)		
Qunatile 3							0.002	(0.02)			0.005	(0.03)	0	(0.03)
D. Extreme poverty	0.008	(0.02)	0.008	(0.02)	0.009	(0.02)	0.006	(0.02)	0.007	(0.02)	0.007	(0.02)	0.007	(0.02)
D. Daycare	0.299	(0.04)	0.299	(0.04)	0.3	(0.04)	0.3	(0.04)	0.3	(0.04)	0.3	(0.04)	0.3	(0.04)
z-score	0.005	(0)	0.005	(0)	0.004	(0)	0.005	(0)	0.004	(0)	0.004	(0)	0.004	(0)
Age (months)	-0.005	(0)	-0.005	(0)	-0.004	(0)	-0.005	(0)	-0.005	(0)	-0.005	(0)	-0.005	(0)
Age^2 (months)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)
D. diarrhea	0.036	(0.02)	0.036	(0.02)	0.035	(0.02)	0.035	(0.02)	0.035	(0.02)	0.035	(0.02)	0.035	(0.02)
D. female	-0.007	(0.01)	-0.007	(0.01)	-0.006	(0.01)	-0.006	(0.01)	-0.006	(0.01)	-0.006	(0.01)	-0.006	(0.01)
Hb mother	0.009	(0.01)	0.01	(0.01)	0.009	(0.01)	0.009	(0.01)	0.009	(0.01)	0.009	(0.01)	0.009	(0.01)
Hb^2 mother	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)	0	(0)
Schooling mother	-0.001	(0)	-0.001	(0)	-0.001	(0)	-0.001	(0)	-0.001	(0)	-0.001	(0)	-0.001	(0)
D. free maternal healthcare	0.129	(0.03)	0.129	(0.03)	0.128	(0.03)	0.13	(0.03)	0.129	(0.03)	0.129	(0.03)	0.129	(0.03)
D. mother employed	-0.003	(0.01)	-0.003	(0.01)	-0.004	(0.01)	-0.002	(0.01)	-0.003	(0.01)	-0.003	(0.01)	-0.003	(0.01)
Ln(hh income pc)	0.02	(0.01)	0.019	(0.01)	0.02	(0.01)	0.02	(0.01)	0.02	(0.01)	0.02	(0.01)	0.02	(0.01)
D indigenous	-0.008	(0.03)	-0.008	(0.03)	-0.009	(0.03)	-0.007	(0.03)	-0.008	(0.03)	-0.008	(0.03)	-0.008	(0.03)
D. afro-ecuadorian	-0.047	(0.04)	-0.047	(0.04)	-0.047	(0.04)	-0.045	(0.04)	-0.045	(0.04)	-0.045	(0.04)	-0.045	(0.04)
D. montubio	0.034	(0.03)	0.034	(0.03)	0.033	(0.03)	0.033	(0.03)	0.033	(0.03)	0.033	(0.03)	0.033	(0.03)
D urban highlands	0.075	(0.04)	0.074	(0.04)	0.075	(0.04)	0.076	(0.04)	0.075	(0.04)	0.075	(0.04)	0.075	(0.04)
D. rural highlands	0.144	(0.04)	0.143	(0.04)	0.143	(0.04)	0.147	(0.04)	0.146	(0.04)	0.146	(0.04)	0.146	(0.04)
D. urban coast	0.003	(0.04)	0.002	(0.04)	0.003	(0.04)	0.005	(0.04)	0.004	(0.04)	0.004	(0.04)	0.004	(0.04)
D. rural coast	0.068	(0.04)	0.067	(0.04)	0.067	(0.04)	0.071	(0.04)	0.07	(0.04)	0.07	(0.04)	0.07	(0.04)
D. urban amazon	0.14	(0.04)	0.139	(0.04)	0.139	(0.04)	0.143	(0.04)	0.142	(0.04)	0.142	(0.04)	0.142	(0.04)
D. rural amazon	0.112	(0.04)	0.111	(0.04)	0.112	(0.04)	0.114	(0.04)	0.115	(0.04)	0.115	(0.04)	0.115	(0.04)
D. Galapagos	0.028	(0.05)	0.027	(0.05)	0.029	(0.05)	0.027	(0.05)	0.028	(0.05)	0.028	(0.05)	0.028	(0.05)
D. Guayaquil	-0.04	(0.05)	-0.04	(0.05)	-0.039	(0.05)	-0.036	(0.05)	-0.036	(0.05)	-0.036	(0.05)	-0.036	(0.05)
D. 71 months	0.156	(0.03)	0.15	(0.04)	0.169	(0.03)	0.146	(0.03)	0.31	(0.1)	0.157	(0.03)	0.129	(0.04)
D. 71 months * quantile 1			0.009	(0.04)					-0.153	(0.1)			0.027	(0.04)
D. 71 months * quantile 2					-0.034	(0.04)			-0.18	(0.1)	-0.027	(0.04)		
D. 71 months * quantile 3							0.164	(0.1)			0.153	(0.1)	0.18	(0.1)
_cons	-0.502	(0.63)	-0.513	(0.63)	-0.507	(0.63)	-0.491	(0.63)	-0.498	(0.63)	-0.504	(0.63)	-0.498	(0.63)
R2	0.3528		0.3529		0.3532		0.3542		0.3544		0.3544		0.3544	
N	1344		1344		1344		1344		1344		1344		1344	

Source: Author's computation using 2012 Nutrition & Health Survey

B. Second instrument

	OLS2:		OLS3:		OLS4:		OLS5:		OLS6:		OLS7:	
Second instrument	Quant1		Qunat 2		Qunat 3		Qunat 1, Quant 2		Qunat 2, Quant 3		Qunat 1, Quant 3	
Endogenous	D. treat*Dq1		D treat * D q2		D treat * D q3		D treat * D Q1		D treat * D Q2		D treat * D Q1	
Instrument	D 71, D 71*D q1		D 71, D 71*D q2		D 71, D 71*D q3		D 71, D 71 * D q1, D 71 * D q2		D 71, D 71 * D q2, D 71 * D q3		D 71, D 71 * D q1, D 71 * D q3	
F value	29.06		14.73		18.26		27.04		13.73		27.04	
F value instruments	23.81		133.18		194.18		15.89		87.39		15.89	
	b	s.e.	b	s.e.	b	s.e.	b	s.e.	b	s.e.	b	s.e.
Qunatile 1	0	(0.02)					0.003	(0.03)		0	0	(0.02)
Qunatile 2			0.003	(0)			0.003	(0.02)	0.003	(0)		
Qunatile 3					0.008	(0)			0	(0.01)	-0.003	(0.02)
D. Extreme poverty	0.001	(0.02)	-0.001	(0)	0.008	(0)	0	(0.02)	-0.001	(0)	0	(0.02)
D. Daycare	0.249	(0.03)	0.05	(0.01)	0	(0)	0.25	(0.03)	0.05	(0.01)	0.25	(0.03)
z-score	0.002	(0)	0.002	(0)	0	(0)	0.002	(0)	0.002	(0)	0.002	(0)
Age (months)	-0.004	(0)	0	(0)	0	(0)	-4.E-03	(0)	0	(0)	-0.004	(0)
Age^2 (months)	0	(0)	0.0E+0	(0)	0.0E+0	(0)	0	(0)	0.0E+00	(0)	0.0E+0	(0)
D. diarrhea	0.02	(0.02)	0.012	(0)	0.003	(0)	0.019	(0.02)	0.012	(0)	0.019	(0.02)
D. female	-0.011	(0.01)	0.003	(0)	0.001	(0)	-0.011	(0.01)	0.003	(0)	-0.011	(0.01)
Hb mother	0.007	(0)	0	(0)	0.001	(0)	0.007	(0)	0	(0)	0.007	(0)
Hb^2 mother	0	(0)	0.0E+0	(0)	0.0E+0	(0)	0	(0)	0.0E+0	(0)	0	(0)
Schooling mother	-0.002	(0)	0	(0)	0	(0)	-0.002	(0)	0	(0)	-0.002	(0)
D. free maternal healthcare	0.11	(0.03)	0.017	(0.01)	0.001	(0)	0.11	(0.03)	0.017	(0.01)	0.11	(0.03)
D. mother employed	-0.001	(0.01)	0	(0)	-0.001	(0)	-0.001	(0.01)	0	(0)	-0.001	(0.01)
Ln(hh income pc)	0.016	(0.01)	-0.001	(0)	0.004	(0)	0.016	(0.01)	-0.001	(0)	0.016	(0.01)
D indigenous	0.001	(0.02)	-0.01	(0.01)	0	(0)	0.001	(0.02)	-0.009	(0.01)	0.001	(0.02)
D. afro-ecuadorian	-0.032	(0.04)	-0.013	(0.01)	0	(0)	-0.032	(0.04)	-0.012	(0.01)	-0.032	(0.04)
D. montubio	0.014	(0.03)	0.029	(0.01)	-0.009	(0)	0.013	(0.03)	0.029	(0.01)	0.013	(0.03)
D urban highlands	0.093	(0.04)	0.016	(0.01)	-0.034	(0)	0.093	(0.04)	0.016	(0.01)	0.093	(0.04)
D. rural highlands	0.148	(0.04)	0.025	(0.01)	-0.028	(0)	0.149	(0.04)	0.026	(0.01)	0.149	(0.04)
D. urban coast	0.027	(0.04)	0.004	(0.01)	-0.028	(0)	0.028	(0.04)	0.004	(0.01)	0.028	(0.04)
D. rural coast	0.092	(0.04)	-0.001	(0.01)	-0.021	(0)	0.093	(0.04)	-0.001	(0.01)	0.093	(0.04)
D. urban amazon	0.166	(0.04)	0.004	(0.01)	-0.03	(0)	0.167	(0.04)	0.005	(0.01)	0.167	(0.04)
D. rural amazon	0.128	(0.04)	0.014	(0.01)	-0.03	(0)	0.13	(0.04)	0.015	(0.01)	0.13	(0.04)
D. Galapagos	0.062	(0.05)	0.003	(0.01)	-0.037	(0)	0.062	(0.05)	0.003	(0.01)	0.062	(0.05)
D. Guayaquil	-0.009	(0.04)	0	(0.01)	-0.028	(0)	-0.007	(0.04)	0	(0.01)	-0.007	(0.04)
D. 71 months	-0.069	(0.03)	-0.039	(0.01)	0.004	(0)	-0.007	(0.09)	-0.042	(0.01)	-0.078	(0.04)
D. 71 months * quantile 1	0.264	(0.04)					0.2	(0.09)			0.271	(0.04)
D. 71 months * quantile 2			0.245	(0.01)			-0.07	(0.1)	0.247	(0.01)		
D. 71 months * quantile 3					0.322	(0.01)			0.033	(0.03)	0.07	(0.1)
_cons	-0.453	(0.59)	0.047	(0.22)	-0.098	(0.1)	-0.453	(0.59)	0.05	(0.22)	-0.449	(0.59)
R2	0.3735		0.2321		0.2726		0.3737		0.2326		0.3737	
N	1344		1344		1344		1344		1344		1344	

Source: Author's computation using 2012 Nutrition & Health Survey

C. Third instrument

	OLS5:		OLS6:		OLS7:	
Third instrument	Qunat 1, Quant 2		Qunat 2, Quant 3		Qunat 1, Quant 3	
Endogenous	D treat * D Q2		D treat * D Q3		D treat * D Q3	
Instrument	D 71, D 71 * D q1, D 71 * D q2		D 71, D 71 * D q2, D 71 * D q3		D 71, D 71 * D q1, D 71 * D q3	
F value	13.73		17		17	
F value instruments	87.39		129.07		129.07	
	b	s.e.	b	s.e.	b	s.e.
Qunatile 1	0	(0.01)		0	-0.002	(0)
Qunatile 2	0.004	(0.01)	0.002	(0)		
Qunatile 3			0.01	(0)	0.007	(0)
D. Extreme poverty	-0.001	(0)	0.008	(0)	0.008	(0)
D. Daycare	0.05	(0.01)	0.00E+00	(0)	0.00E+00	(0)
z-score	0.002	(0)	0	(0)	0	(0)
Age (months)	0	(0)	0	(0)	0	(0)
Age^2 (months)	0.00E+00	(0)	0.00E+00	(0)	0.00E+00	(0)
D. diarrhea	0.012	(0)	0.003	(0)	0.003	(0)
D. female	0.003	(0)	0.001	(0)	0.001	(0)
Hb mother	0	(0)	1.00E-03	(0)	0.001	(0)
Hb^2 mother	0.00E+00	(0)	0.00E+00	(0)	0.00E+00	(0)
Schooling mother	0	(0)	0	(0)	0	(0)
D. free maternal healthcare	0.017	(0.01)	0.001	(0)	0.001	(0)
D. mother employed	0	(0)	-0.001	(0)	-0.001	(0)
Ln(hh income pc)	-0.001	(0)	0.004	(0)	0.004	(0)
D indigenous	-0.009	(0.01)	0	(0)	0	(0)
D. afro-ecuadorian	-0.012	(0.01)	0	(0)	0	(0)
D. montubio	0.029	(0.01)	-0.009	(0)	-0.009	(0)
D urban highlands	0.016	(0.01)	-0.034	(0)	-0.034	(0)
D. rural highlands	0.026	(0.01)	-0.029	(0)	-0.029	(0)
D. urban coast	0.004	(0.01)	-0.028	(0)	-0.028	(0)
D. rural coast	-0.001	(0.01)	-0.021	(0)	-0.021	(0)
D. urban amazon	0.005	(0.01)	-0.03	(0)	-0.03	(0)
D. rural amazon	0.015	(0.01)	-0.03	(0)	-0.03	(0)
D. Galapagos	0.003	(0.01)	-0.037	(0)	-0.037	(0)
D. Guayaquil	0	(0.01)	-0.028	(0)	-0.028	(0)
D. 71 months	-0.008	(0.03)	0.006	(0)	0.002	(0)
D. 71 months * quantile 1	-0.033	(0.03)			0.003	(0)
D. 71 months * quantile 2	0.214	(0.03)	-0.003	(0)		
D. 71 months * quantile 3			0.32	(0.01)	0.324	(0.01)
_cons	0.049		-0.104		-0.102	
R2	0.2326		0.2728		0.2728	
N	1344		1344		1344	

Source: Author's computation using 2012 Nutrition & Health Survey

The main concern with IV model is that the instrument might be weak which would increase the standard error of the IV estimate or that it might not be exogenous, that is, that it might be correlated with unobserved heterogeneity, which would make the IV estimate biased (Shahidur, et al., 2010). In relation to the former, I have a highly significant instrument which has a z-value of 4.22 (see appendix 2) in model 1 (where there are no heterogeneous effects). In models with heterogeneous effects the F-statistic is presented in order to differentiate those with strong (models 2 - 4) and weak (models 5 - 7) instruments. In relation to the latter, I cannot test for whether our specific instrument satisfied the restriction exclusion, I can only offer theoretical justification for how the instrument might identify participation exogenously.

In summary, the average effect is not significant and seems to have a positive coefficient in the RD model while having a negative one in the IV model. The negative sign in the IV model seems to be driven by the effect on the first quantile where children have the lowest Hb levels. Here, I find a negative significant effect. While in quantiles 2 and 3 I find a positive significant and a positive non-significant effect respectively. The negative significant effect of the first quantile may be driven by diarrhea caused by the treatment and/or by a lack of compliance in the number of doses as this is not measured in this model.

2.6 Discussion and Conclusion

IDA is a condition characterized by a depletion in iron reserves leading to a lower than normal level of Hb in the blood (U.S. Department of Health and Human Services, 2014).[57] It is a form of malnutrition which has the potential to affect development, learning abilities and schooling achievements in children (Martinez, et al., 2009; WHO, 2015; Micronutrient Initiative, 2015). Ecuador is an important case study for the evaluation of public policy to reduce IDA as it has had a persistent problem with the incidence of IDA among children[58] (Freire, et al., 1988; Ministerio de Salud Publica; Instituto National de Estadisticas y Censos, 2013).

This study is an evaluation of the public policy attempting to reduce the prevalence of IDA in Ecuador. I evaluate the national program that distributes micronutrient supplements through both public daycare and public healthcare centers. The policy stipulates that children up to the age of 59 months are eligible for the treatment. Children over this age are no longer eligible.

We use the 2012 cross-section HNS where there is information both on Hb levels of children and whether they received the treatment in the past 12 months. In order to identify the causal effect of the intention to

[57] Hb is an iron-rich protein that carries oxygen from the lungs to the rest of the body.
[58] 69% in 1988 and 62% in 2012 among children 6 to 12 months of age.

treat, I present two methods: Firstly, a fuzzy RD model in which I use the age eligibility rule as a cut-off point, and, secondly, an IV model in which I use the age eligibility rule as an instrument. Additionally, I present six IV models with heterogeneous effects by quantile of the dependent variable Hb.

Both methods allow us to exogenously identify participants from non-participants and account for observed as well unobserved heterogeneity. Both models allow for endogeneity in both individual participation and program placement by identifying a cut-off or instrument that is highly correlated with program placement and not correlated with the unobserved characteristics affecting outcome (Shahidur, et al., 2010). The difference between these two methods lies in the bandwidth around the cut-off used in the fuzzy RD model but not used in the IV model. In the fuzzy RD model, this bandwidth restricts the sample of both treatment and control groups to children who are 'close' to the cut-off age while the IV model measures the effect on all children from 6 to 120 months.

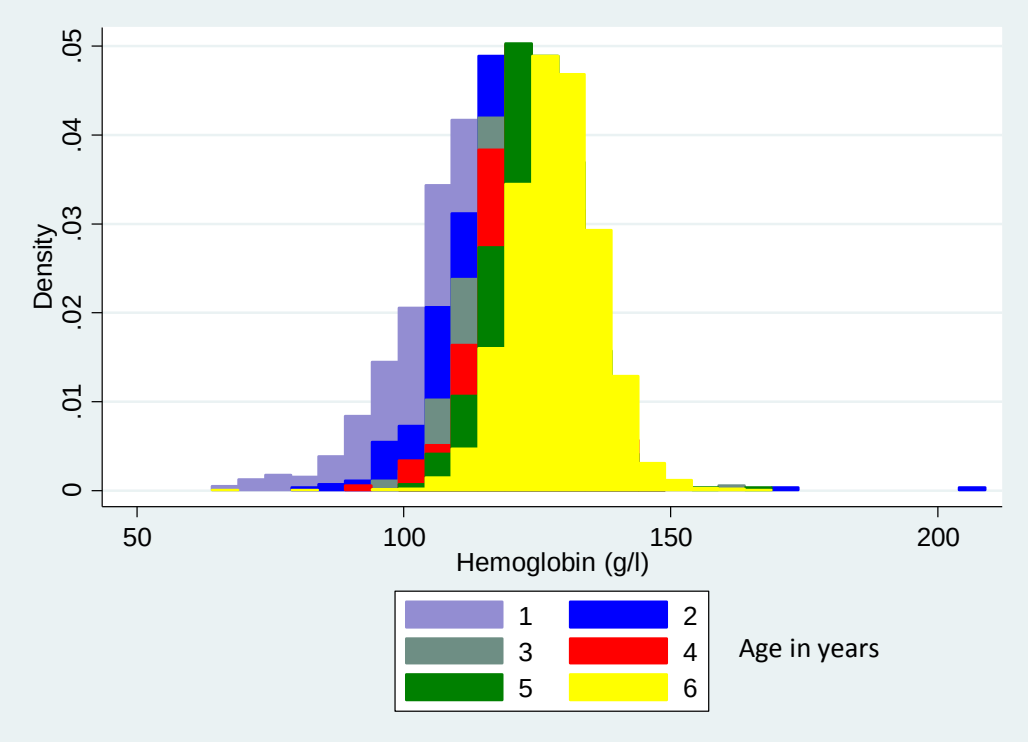
Despite specifying various bandwidths and functional forms, our results show no significant effects in the fuzzy RD models with some specification resulting in positive and others in negative coefficients. The average effect of the IV model is negative and non-significant, however, when a heterogeneous effect by quantile is included (models 2 - 4), the effect on the rest of the distribution is positive and non-significant. I include six models (models 2 - 7) with heterogeneous effects: three (models 2 - 4) with one heterogeneous effect (i.e. one quantile per model), and, three (models 5 - 7) where each model has two heterogeneous effects (i.e. two quantiles per model). I will only refer to models 2 - 4 in this discussion, as they, along with model 1 (no heterogeneous effects), have first stage regressions with F-statistics larger than 10, and are considered to have strong instruments. On the other hand, models 5 - 7 suffer from weak instruments.

In models 2 - 4 I build an instrument for every heterogeneous effect. A model with one heterogeneous effect (e.g. quantile 1) will have two endogenous variables: dummy treatment and the interaction between dummy treatment and dummy quantile 1. The first instrument is the cut-off (dummy child under 71 months of age) and the second instrument is the interaction between the cut-off and dummy quantile 1. If the first instrument is exogenous, then it is reasonable to assume the second instrument is as well (Cerulli, 2012). Each model has a separate OLS first stage regression to show the F-statistic.

As mentioned above, I find a negative non-significant effect in the IV model with no heterogeneous effects. However, I find two interesting outcomes when analysing the effect over the distribution of Hb. The effect is negative and significant for children in the first quantile (low Hb), a positive and significant for children in the second quantile (average Hb), and positive and non-significant in the third quantile (high Hb). I

suspect that the opposing effects in quantile 1 vs. quantile 2 and 3 cancel each other out when measuring the average effect in the IV and RD models rendering the coefficients non-significant. Additionally, by including heterogeneous effect by quantile I am indirectly identifying the heterogeneous effect by age, as children under the age of 1 tend to be in the lower end of the Hb distribution (Figure 2.7). Obviously I am controlling for the effect of age, and any other contemporaneous factor affecting IDA. However, younger children in the lower end of the distribution may help explain the negative significant effect in the first quantile, as it is more likely that younger children have never had the treatment before. If the treatment causes diarrhea, particularly among children who have never had "sprinkles" before, then it would show up as a negative effect of the intention to treat (Ministerio de Salud Publica, World Food Program, 2011).

Figure 2.7 Distribution of Hb by age in years



Source: Author's computation using 2012 Nutrition & Health Survey

We are not able to include heterogeneous effects in the RD model given the bandwidth around the cut-off needs to be very small and this reduces the sample size considerably. At the smallest bandwidth of 1 month the sample size is 38 for the treatment and 49 for the control, at the largest bandwidth of 12 months, the sample is 602 for treatment and 541 for control. In order to have heterogeneous effects, I would need to run

a model with a considerably larger bandwidth, which, if taken to the extreme of including all children, would be methodologically equivalent to running an IV model[59] (Calonico, et al., 2014).

The limitations of this study are twofold (1) I cannot determine if children in the 2012 HNS completed the treatment of 60 doses. This is determinant in the outcome. Notwithstanding, randomized trials with issues of non-compliance perform ITT models, which are generally widely accepted. In these models the results of a trial in which the individuals were initially randomly assigned into treatment or control groups are estimated regardless of whether they completed the intervention (Armijo-Olivo, et al., 2009). The results are an estimate of the effect of a change in treatment policy rather than an estimate of the effect of the treatment in patients who receive the treatment exactly as planned (Hollis & Campbell, 1999). I believe the models applied in this part of the study (fuzzy RD and IV) are analogous to the ITT models in that they use the initial randomization of the program in order to estimate the effect of a change in treatment policy rather than of the effect of treatment on compliers.

(2) The survey question inquiries about participation in the program over the past 12 months. This implies that children 11 months older than the cut-off (71 months old) could have participated while still within the age limit (59 months) or after going over it. The way in which the question is formulated restricts us from distinguishing them. However, beyond 71 months, the probability of receiving the treatment falls dramatically very close to zero, therefore, I assume this jump represents a cut-off in access due to lack of eligibility.

I believe this study is an important contribution to the literature as it identifies the causal effect of the intention to treat and it is the first to evaluate a non-cash transfer on the nutritional health of children in Ecuador. Most studies relating to the nutritional health of children in Ecuador use similar techniques to identify the causal effect of the treatment, however, they mainly study the effect of the national wide cash transfer program called “Bono de desarrollo humano,” others study the effect of the Ecuadorian 1999 financial crisis on nutritional outcomes and most use the z-score of height for age as the outcome variable. There are no studies, to our knowledge, which study non-cash transfer program such as nutritional supplements on the nutritional outcome of children. There are virtually no studies that use blood sample outcomes such as Hb as outcome variables. Rather, most focus on the anthropometric outcomes that are more widely available.

[59] given it is constructed in a twostep process: the first stage is the regression where the cut-off predicts the treatment and the second is the regression where predicted-treatment predicts the outcome (the estimand take the form of a ratio of the two).

In conclusion, I find no significant average effect of the intention to treat on children under 5, there is a negative significant effect on children with the lowest Hb levels and a positive significant effect on children in the middle quantile, I suspect these effects cancel each other out on average. These findings point firstly to the importance of further research on the effect of the treatment by dose, as it is deterministic in the outcome and may clarify why the intention to treat has no average effect. Additionally, the lack of a significant effect may indicate that the treatment of the direct cause of malnutrition might be insufficient. In the next chapter I attempt to construct the argument that there may be other causes, apart from those being treated (i.e. malnourishment), which may be driving the persistent levels of IDA and other forms of malnutrition in Ecuador. I explore the effect of intra-uterine shocks on chronic malnutrition i.e. stunting in an attempt to test the effect of environmental shocks on individual health.

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Appendix 1 Chronic child malnutrition and the z-score of height for age

The z-score of height for age is estimated using the methodology developed and distributed freely by the World Health Organization (2013). The normalized z-score (A.1) establishes the growth standard of children by defining a normal growth curve (World Health Organization, 2013; World Health Organization, 1997).

$$z\ score = {(x_i - x_{median})}/{\sigma^x} \quad \textbf{A.1}$$

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). This score is generally estimated using anthropometric data available in the diagnosis of the nutritional and health situation of Ecuador (DANS[60]) survey 1986, LSMS (2006, 2014) and HNS for each child below the age of five. The z-score ranges from $-\infty$ to ∞ as it is measured in standard deviations from the mean which is zero. If a child's z-score is under -2, that is to say, under two standard deviations below the mean, the child is chronically malnourished or "stunted" (World Health Organization, 1997).

[60] For its name in Spanish: *Diagnóstico de la situación alimentaria nutricional y de salud*

Appendix 2: IV models: First stage Probit models

Here the first stage prediction of dummy treatment is presented. The first stage prediction of other instruments is not presented the as they are not available in the output of *ivtreatreg*.

Table A2 First stage probit models

		IV1		IV2		IV3		IV4		IV5		IV6		IV7
Dependent variable: D. Treat	b	z-value	b	z-value	b	z-value	b	z-value	b	z-value	b	z-value	b	z-value
D. 71 months old (cut-off)	**1.48**	**(4.22)**	**1.47**	**(4.2)**	**1.48**	**(4.22)**	**1.51**	**(4.22)**	**1.51**	**(4.21)**	**1.51**	**(4.21)**	**1.51**	**(4.21)**
			-0.15	(-0.84)					-0.74	(-2.18)			-0.04	(-0.25)
					-0.04	(-0.24)			-0.69	(-2.01)	0.04	(0.25)		
							0.71	(2.19)			0.74	(2.18)	0.69	(2.01)
z-score of height for age	0.03	(0.75)	0.03	(0.77)	0.03	(0.74)	0.03	(0.71)	0.03	(0.72)	0.03	(0.72)	0.03	(0.72)
Age (months)	0	(-0.43)	0	(-0.48)	0	(-0.4)	0	(-0.18)	0	(-0.2)	0	(-0.2)	0	(-0.2)
Age^2 (months)	0	(-0.64)	0	(-0.67)	0	(-0.64)	0	(-0.93)	0	(-0.93)	0	(-0.93)	0	(-0.93)
D. diarrhea	0.17	(1.25)	0.18	(1.25)	0.17	(1.24)	0.17	(1.2)	0.17	(1.21)	0.17	(1.21)	0.17	(1.21)
D. daycare	0.75	(3.65)	0.75	(3.62)	0.75	(3.65)	0.75	(3.62)	0.75	(3.62)	0.75	(3.62)	0.75	(3.62)
D. female	0.01	(0.15)	0.01	(0.14)	0.01	(0.15)	0.01	(0.16)	0.01	(0.15)	0.01	(0.15)	0.01	(0.15)
Hb mother	0.1	(1.12)	0.11	(1.18)	0.1	(1.1)	0.1	(1.13)	0.11	(1.15)	0.11	(1.15)	0.11	(1.15)
Hb mother^2	0	(-1.08)	0	(-1.14)	0	(-1.06)	0	(-1.1)	0	(-1.11)	0	(-1.11)	0	(-1.11)
Schooling mother	-0.01	(-0.66)	-0.01	(-0.71)	-0.01	(-0.65)	-0.01	(-0.75)	-0.01	(-0.76)	-0.01	(-0.76)	-0.01	(-0.76)
D. free maternal healthcare	0.36	(2.08)	0.37	(2.11)	0.36	(2.07)	0.37	(2.12)	0.37	(2.13)	0.37	(2.13)	0.37	(2.13)
D. mother employed	-0.02	(-0.18)	-0.01	(-0.13)	-0.02	(-0.19)	-0.01	(-0.12)	-0.01	(-0.11)	-0.01	(-0.11)	-0.01	(-0.11)
Ln(hh income pc)	0.18	(1.87)	0.18	(1.86)	0.18	(1.87)	0.18	(1.82)	0.18	(1.82)	0.18	(1.82)	0.18	(1.82)
D.extreme poverty	0.16	(0.81)	0.15	(0.75)	0.16	(0.82)	0.13	(0.64)	0.12	(0.63)	0.12	(0.63)	0.12	(0.63)
D indigenous	-0.08	(-0.37)	-0.07	(-0.32)	-0.08	(-0.39)	-0.08	(-0.34)	-0.07	(-0.32)	-0.07	(-0.32)	-0.07	(-0.32)
D. afro-ecuadorian	-0.43	(-1.09)	-0.44	(-1.1)	-0.43	(-1.09)	-0.42	(-1.07)	-0.42	(-1.07)	-0.42	(-1.07)	-0.42	(-1.07)
D. montubio	0.24	(0.9)	0.24	(0.9)	0.24	(0.9)	0.25	(0.94)	0.25	(0.93)	0.25	(0.93)	0.25	(0.93)
D. urban highlands	0.36	(1.2)	0.36	(1.19)	0.37	(1.21)	0.42	(1.35)	0.41	(1.34)	0.41	(1.34)	0.41	(1.34)
D. rural highlands	0.91	(2.88)	0.92	(2.91)	0.92	(2.88)	0.99	(3.05)	0.99	(3.05)	0.99	(3.05)	0.99	(3.05)
D. urban coast	-0.18	(-0.6)	-0.18	(-0.59)	-0.18	(-0.59)	-0.13	(-0.42)	-0.13	(-0.43)	-0.13	(-0.43)	-0.13	(-0.43)
D. rural coast	0.33	(0.97)	0.34	(0.98)	0.33	(0.97)	0.39	(1.11)	0.39	(1.11)	0.39	(1.11)	0.39	(1.11)
D. urban amazon	0.79	(2.41)	0.79	(2.43)	0.79	(2.42)	0.85	(2.56)	0.85	(2.56)	0.85	(2.56)	0.85	(2.56)
D. rural amazon	0.67	(2.01)	0.68	(2.02)	0.68	(2.02)	0.74	(2.18)	0.74	(2.17)	0.74	(2.17)	0.74	(2.17)
D. Galapagos	-0.02	(-0.07)	-0.04	(-0.12)	-0.02	(-0.05)	0.01	(0.04)	0	(0.02)	0	(0.02)	0	(0.02)
D. Guayaquil	-0.43	(-1.08)	-0.43	(-1.1)	-0.42	(-1.06)	-0.36	(-0.9)	-0.36	(-0.91)	-0.36	(-0.91)	-0.36	(-0.91)
_cons	-9.79	(-1.61)	-10.02	(-1.63)	-9.68	(-1.59)	-9.97	(-1.63)	-9.34	(-1.52)	-10.08	(-1.64)	-10.04	(-1.63)
Pseudo R2		0.4464		0.447		0.4464		0.4504		0.4504		0.4504		0.4504
N		1344		1344		1344		1344		1344		1344		1344

Source: Author's computation using 2012 Nutrition & Health Survey

Chapter 3

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

3.1 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. In the same year, the worst El Nino 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 that lead 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 swift and massive flight in liquidity as preferences shifted to the dollar. The drastic fall in total deposits[61] 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 freeze financial assets. By October 1999 the government had suspended payments on Discount and PDI Brady Bonds and Brady and Eurobonds. In March 2000, Ecuador had adopted le US dollar as legal tender (Jacome, 2004; Sturzenegger & Zettelmeyer, 2008).

We interpret the crash in liquidity on 1 Jan 1999 as the point of infliction of the crisis. The sudden and precipitous collapse of the financial system is interpreted as 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; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013;

[61] See Figure 1.

Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

In this paper I measure the effect of this 1999 intra-uterine maternal stress shock on the 2012 z-score of height-for-age of the offspring. In order to estimate the average treatment effect (ATE) I propose a sharp RD model. The method compares 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 a causal effect of the exogenous in-utero stress shock. I find children born after the crash have significantly lower z-scores (in 2012) than children born before. Although I cannot test this hypothesis directly, I propose that the financial crash created an intra-uterine shock through pre-natal maternal stress (Almond & Currie, 2011).

RD models assume the randomized variation in treatment is a consequence of the inability of agents to control the assignment variable near the cut-off. The choice of bandwidths, polynomial forms and kernel functions is fundamental in the analysis and interpretation of RD designs. Therefore, two data-driven methods are used to select an appropriate bandwidth: firstly, the Akaike information criterion (AIC) and, secondly, a dummy variable test in order to select the polynomial order. Furthermore, the sensitivity of the results to triangle, rectangle and Epanechnikov kernel functional forms are tested, and finally, relevant observable characteristics are found not significant in determining selection into treatment (Cattaneo, et al., 2018; Lee & Lemieux, 2010). In addition, 4 robustness checks are run as recommended by Lee and Lemieux (2010) and Cattaneo et al. (2018): (1) placebo effects for the months and years predating the crisis; (2) the density of the running variable is examined, and (3) a test is run for the sensitivity of the model to observations near the cut-off. Finally, (4) other relevant observables are analyzed to make sure they do not have the same cut-off.

This chapter contributes to the literature in three ways: (1) it studies a financial crisis which is less commonly found as a stressful life event in the literature. (2) I measure long term effects rather than short or medium term effects. (3) I 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. Additionally, it may help explain the lack of significant results of the current public policy to reduce iron deficiency anemia (IDA) in Ecuador (see results chapter two) and may help shape preventative public policy interventions during pregnancy which could potentially be

effective at improving health outcomes later in life (Almond & Currie, 2011). Finally, I have not found studies which analyze the long term health effects of pre-natal exposure the 1999 Ecuadorian crisis, making this an original contribution to the debate.

Notwithstanding, there are various weaknesses with the evidence presented in this paper. Firstly, despite testing and not finding any anticipation effects before the crisis, I did find isolated significant placebo effects after the crisis and on New Year's Day 1995. Although, it is worth mentioning that these placebo effects lose their significance after a simple local polynomial robustness check. Secondly, the density of the running variable is not uniform leading to a small imbalance in the size of the (treatment v. control) samples. However, I find no evidence that the density of the distribution of observations has an effect on the outcome, and I find significant effects even after eliminating observations near the cut-off - which concurrently also balances the sample sizes. Finally, despite our attempts, I was 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 chapter. Taking all this into account, I believe I provide ample evidence there is no anticipation bias and no manipulation of the cut-off, making this a robust sharp RD design.

This paper is divided into six parts: (1) The context explains the origin, outbreak and aftermath of the 1999 Ecuadorian crisis; (2) the mechanisms explains the fetal origins hypothesis, the empirical evidence, and how it applies to this case; (3) the methodology gives the econometric account of the model; (4) the data and results outline parameters; (5) the robustness checks go through every case where a RD model might fail; and finally, (6) the conclusion and discussion.

3.2 Context: the financial crisis of 1999

3.2.1 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, I will briefly explain the details which are relevant to the 1999 financial crash (see Appendix 2 for graphic representation of chronology) (Jacome, 2004; Martinez, 2006).

In 1994 the Law of Financial System Institutions[62] 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[63] and the Superintendence of Banks and Insurance Companies[64] 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,[65] 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[66] and "a number of other exogenous shocks"[67] 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%[68] which created liquidity problems for banks with maturity mismatches. Banco Continental failed and was acquired by the State, however, 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

[62] 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).
[63] Credits increased 40% in 1993 and 50% in 1994.
[64] Superintendencia de Bancos.
[65] Currency or maturity of assets not equal to currency or maturity of liabilities.
[66] January 26 – February 28, 1995.
[67] The author (Jacome, 2004) does not specifies what he is refering to.
[68] And the real interest rate up to 30%.

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 Prestamos) closed and returned depreciated deposits of only small savers after several weeks. Larger deposit holders did not receive their 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[69] through OMOs (See Appendix 1 for Jacome (2004) 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).

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

In December 1998 legislation[70] meant to deal with the absence of effective bank resolution instruments was approved by Congress. The law created the Deposit Guarantee Agency (AGD[71]) 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).

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).

[69] Bonos de estabilizacion monetaria, BEMs
[70] 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).
[71] Given its name in Spanish: Agencia de Garantia de Depositos.

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 3.1), 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.[72] 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 (Cantos Bonilla, 2006; Jacome, 2004).

The ensuing months saw the predictable consequences. In the early days of March 1999 the largest bank (in terms of deposits, Banco del Progresso) 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.[73] This holiday lasted a week and finalized in the widespread freezing of all bank accounts with a balance over 500 USD in order to avoid further capital flight. Savings accounts would be frozen for a year and checking accounts for 6 months (Jacome, 2004).

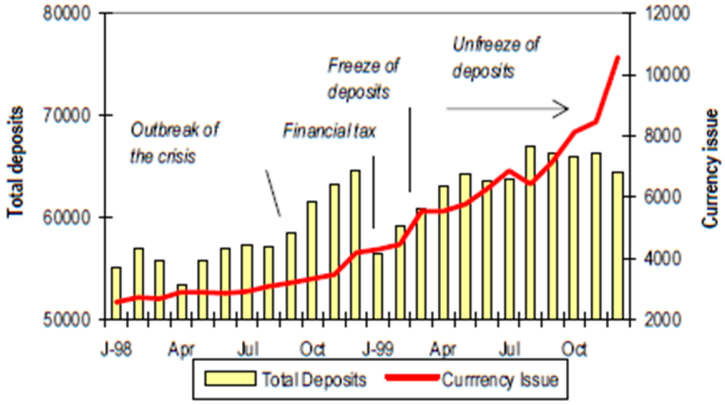
3.2.3 Discussion on our crisis threshold

Waves of withdrawals occurred fairly regularly in Ecuador. However, as is shown in Figure 3.1, between the first bank failure (April 1998) and December 1998 total deposits continued to increase. Only in Jan 1999 did total deposits fall. Figure 3.2 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 increasing since September 1998 (see Figure 3.3 & Figure 3.1) (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 sufficient time for deposit holders to anticipate and adapt to it. Total deposits grew in December 1998 (Figure 3.1), 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. I argue the contagion effect the tax had on deposits was unanticipated by policy makers and deposit holders.

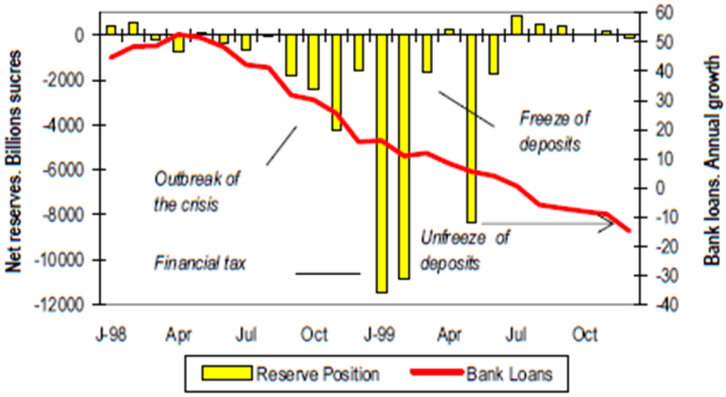
[72] Notably the largest bank in terms of deposits (Banco del Progreso).
[73] Meaning that banks remained closed.

Figure 3.1 Total Deposits and Currency Issue (Billions of Sucres)



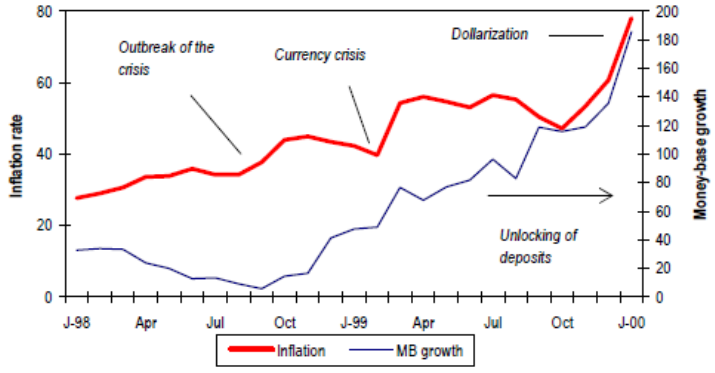
Source: Jacome, 2004; Source of data cites in Jacome, 2004: Central Bank of Ecuador

Figure 3.2 Liquidity and credit crunch



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

Figure 3.3 Inflation and monetary base growth (annual percentage rate)



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

3.3 Mechanism: intra-uterine shocks

3.3.1 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 to 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 (Gluckman, et al., 2005). This may affect the individual's fitness later in life. Pre-natal maternal (PNM) 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[74] 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).

Two publications by Rice & 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

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

not. This distinction allows them to identify the contribution of maternal intra-uterine 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 pre-natal 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).[75]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.

3.3.2 Empirical evidence for intra-uterine shocks

Although Backer's initial work was essentially correlational (Barker & Osmond, 1986; Barker, 1995), increasing amounts of evidence which suggest an empirical link are find in the literature. I 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, LBW or fetal growth restriction (Beydoun & Saftlas, 2008). Almond and Currie (2011) find numerous studies providing evidence of the long-term consequences of a wide variety of intra-uterine shocks (Almond & Currie, 2011). Conversely, Zijlmans' et al. meta-analysis finds only a small number of associations between maternal pre-natal cortisol and child outcomes are significant. 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 pre-natal 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).

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

Schetter & Tanner (2012) find that a majority of the more than a dozen published studies measuring objective stress events[76] 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, with some exception, therefore, objective life events stressors tend to affect birth outcomes most when they occur in the first trimester (Hobel, et al., 2008; Schetter & Tanner, 2012).

In our own review of the literature, various studies find significant associations between of 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,[77] birth lengths, LBW, and pre-term delivery in the US and Canada and on LBW in Haiti. On the other hand, 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, preterm birth, LBW, 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[78] 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 and were associated with LBW (Wainstock, et al., 2013; Camacho, 2008). A study on immigration raids in the USA finds that infants born to Latin American mothers had an increased risk of LBW while no such change was observed among infants born to non-Latin American mothers (Novak, et al., 2017).

There is mixed evidence on the effect of a financial crisis in the literature. Studies in Iceland find an increase 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

[76] 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).
[77] Such as being on a ventilator more than 30 min and meconium aspiration syndrome (MAS).
[78] Terrorist attack on the World Trade Center in New York City.

(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 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[79] 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. This is relevant because there is a potentially similar effect to be found on the pre-natal exposure to the 2008 financial crisis, particularly in countries where the crisis affected individual's savings. Secondly, I measure long term effects rather than immediate or medium term outcomes. This is relevant given it may provide an explanation for the lack of efficacy of Ecuador's public policy to reduce IDA (a form of malnutrition) which focuses on treating micronutrient depletion and does not take intra-uterine exposure to maternal stress shocks into account. It provides evidence that preventative public policy interventions during pregnancy could potentially be effective in terms of improving health outcomes later in life. Finally, most studies are correlational, few studies tackle issues of endogeneity, particularly when dealing with perceptions of stress or pregnancy related stressors. This study, provides a causal effect by using regression, discontinuity models and a theoretical mechanism explaining the pathway from PNM stress to outcome.

3.4 Methodology

We use a sharp RD model which I explain in this section. If an assignment variable S_i which determines whether the individual receives the "treatment" exists (the tax shock before birth) and, there is an eligibility cut-off at S^* (1 Jan 1999) it is possible to model the effect of the shock on the individual outcomes 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 limits exist on either side of

[79] Known as the "Hunger Winter."

the threshold s^*, the impact estimation for an arbitrarily small $\varepsilon > 0$ around that threshold would be as follows (Shahidur, et al., 2010; Lee & Lemieux, 2010):

$$E[y_i|s^* - \varepsilon] - E[y_i|s^* + \varepsilon] = E[\beta S_i|s^* - \varepsilon] - E[\beta S_i|s^* + \varepsilon] \quad \textbf{(3.1)}$$

When taking the limit of both sides of (3.1) as $\varepsilon \to 0$, β is identified 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 (Shahidur, et al., 2010; Lee & Lemieux, 2010):

$$\lim_{\varepsilon \to 0} E[y_i|s^* - \varepsilon] - \lim_{\varepsilon \to 0} E[y_i|s^* + \varepsilon] \Rightarrow y^- - y^+ = \beta(S^- - S^+) \Rightarrow \beta = \frac{y^- - y^+}{S^- - S^+} \quad \textbf{(3.2)}$$

We assume, that individuals are assigned to treatment (i.e. intra-uterine exposure to the crisis) 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.

3.5 Data

The National Health and Nutrition Survey (ENSANUT[80] which I refer to as HNS) is a cross-section database built by the National Institute for Statistics and Censes (INEC[81]) 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 there is 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).

3.5.1 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 (3.3) establishes the growth standard of children by defining a normal growth curve (World Health Organization, 2013; World Health Organization, 1997).

$$\text{zhfa}_i = {}^{(x_i - x_{median})}/_{\sigma^x} \quad \textbf{(3.3)}$$

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;

[80] Given its name in Spanish: Encuesta Nacional de Salud y Nutrición.
[81] Given its name in Spanish: Instituto Nacional de Estadísticas y Censos.

World Health Organization, 1997). They use anthropometric data available in the LSMS (2006) to calculate the $zhfa_i$ for each individual. In this case the group of interest is 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 -2, that is to say, under two standard deviations below the mean, the child is chronically malnourished or "stunted" (World Health Organization, 1997). Figure 3.4.A and 3.4.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. 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[82] the average is -1.14 and the prevalence is 21%.

Table 3.1 Descriptive statistics of dependent variable: $zhfa_i$

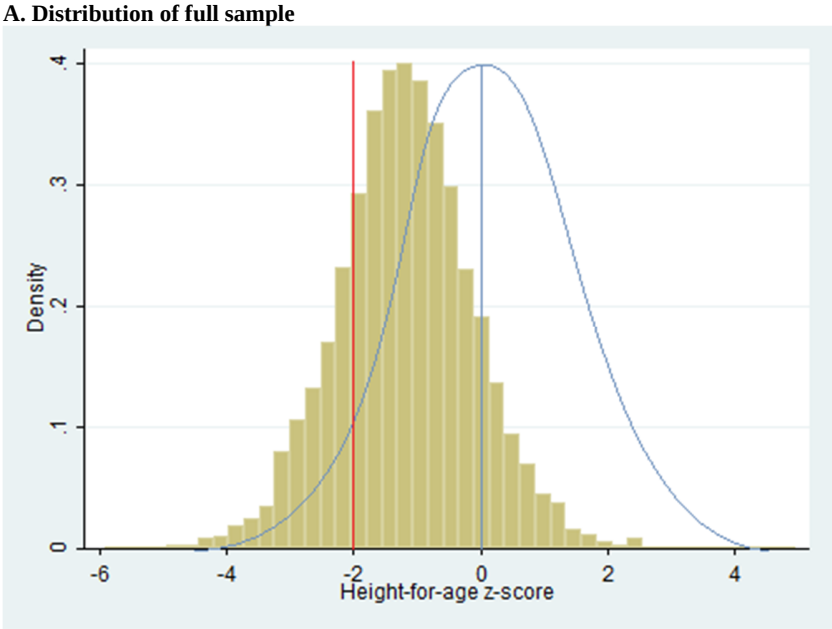
Variable	Obs.	Mean	Std. Dev.	Min	Max
zhfa (all)	18968	-1.11	1.07	-5.9	4.97
D. Malnutrition (all)	18968	0.19	0.39	0	1
zhfa (sub-sample)	195	-1.14	1.11	-4.92	1.68
D. Malnutrition (sub-sample)	195	0.21	0.41	0	1

Source: Author's computation using 2012 Nutrition & Health Survey

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

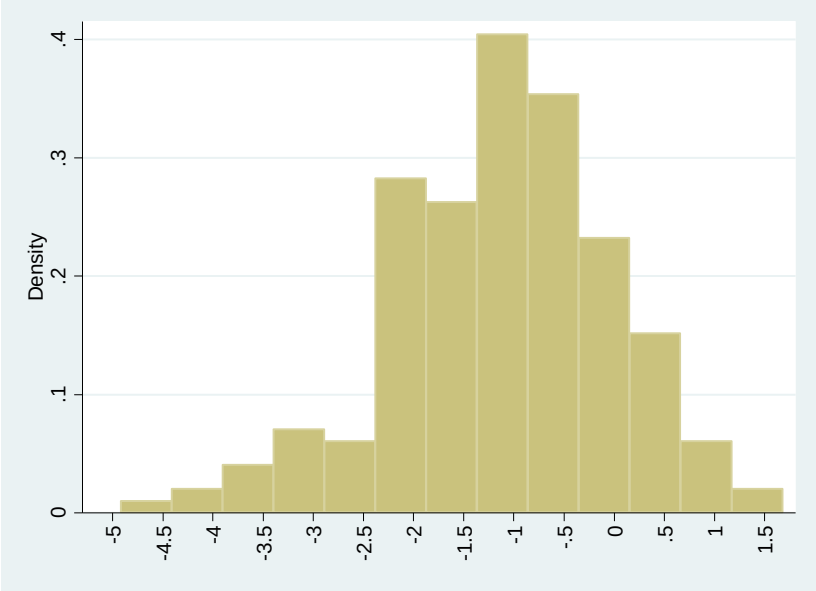
Figure 3.4 Distribution of z-score of height-for-age in 2012 among 5 to 19 year olds in Ecuador

A. Distribution of full sample



Source: Author's computation using 2012 Nutrition & Health Survey

B. Distribution of sub-sample of children close to the cut-off point



Source: Author's computation using 2012 Nutrition & Health Survey

3.5.2 The assignment variable: days born before/after crisis

In this study I 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, as is indicated in (3.4):

$$S_i = edob_i - edoc \tag{3.4}$$

Where $edob_i$ is the elapsed date of birth and $edoc$ is the elapsed date of the crisis. An elapsed date is the number of days transpired between 1 Jan 1960 (a reference date) and a given date, such as, the date of birth. This is the technique used by **STATA** to understand dates. Therefore $edob_i$ would vary as a function of the date of birth of the individual, while the $edoc$ is a fixed number equal to the difference between 1 Jan 1960 and 1 Jan 1999. 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 3.2 provides descriptive statistics of the three variables for the children born a 30 days before/after the crisis.

Table 3.2 Descriptive statistics of the assignment variable S_i

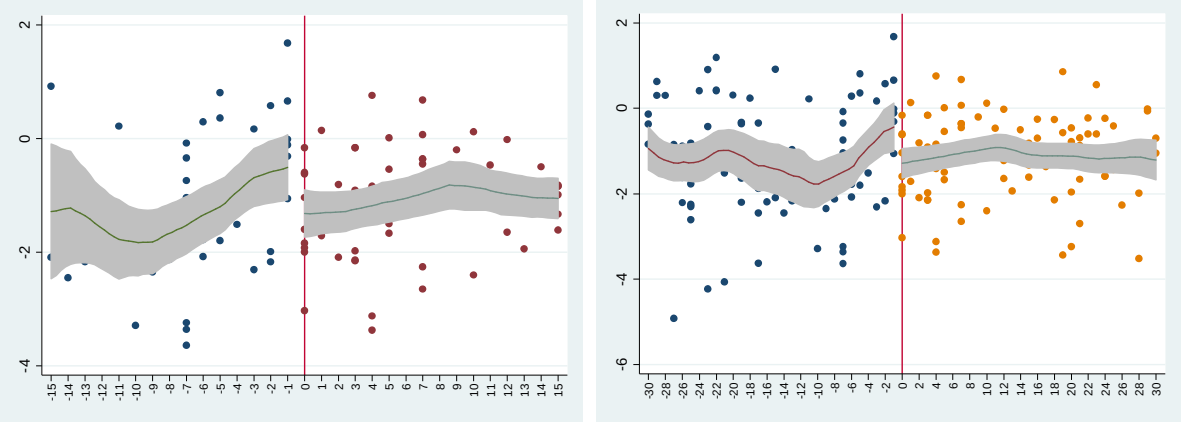
Variable	Obs	Mean	Std. Dev.	Min	Max
Elapsed date of crisis	196	14244	17	14215	14275
Elapsed date of birth	196	14311	0	14311	14311
S_i	196	-1	17	-30	30

Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.5.A is a scatterplot with a local polynomial regression line of the $zhfa_i$ by S_i with a 15 day bandwidth. Figure 3.5.B is a local polynomial regression with a fitted linear regression line of the $zhfa_i$ by S_i with a 30 day bandwidth. I estimate a separate local polynomial regression on each side of the cut-off in order to visually represent the drop in $zhfa_i$which occurs on the day of the crisis $S_i = 0$ (see Appendix 4 for box plot representations of the bandwidths around the cut-off).

Figure 3.5 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**



Source: Author's computation using 2012 Nutrition & Health Survey

3.6 Results

As suggested by Lee and Lemieux (2010) and Cattaneo at al. (2018) I justify the choice of bandwidth, polynomial order and kernel function before presenting the results. Additionally, in the following section, I will go over various robustness checks (Lee & Lemieux, 2010; Cattaneo, et al., 2018).

3.6.1 Choosing a bandwidth

Choosing a bandwidth within which one is 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 randomization is a reasonable assumption and a 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 I 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 two exogeneity tests are presented. The first simply involves selecting observable characteristics that would be otherwise correlated with 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. Model 1 of Table 3.3 has a strong correlation with S_i when the assignment variable has no bandwidth (also see Figure 3.6). In Model 2 in Table 3.3 there is no significant relationship between weight and age when a 30 or 15 day bandwidth is used around the assignment variable (see Figure 3.7).

In relation to the latter, 5 probit models using dummy treat as the dependent variable are used (Table 3.4). This way, the significance of various observable characteristics on selection into treatment can be tested for. In Table 3.4 it is shown that age is significant when using the 30 day bandwidth, which is expected given it is one of the two variable 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 noticing that there are 87 observations in the 15 day model (1), while in the 30 day model (4) there are 172 observations. I 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 exognenous probit model, given the age variable is expected to be significant. In any case, in our results both the 30 and the 15 day bandwidth models given both seem to be robust to observable characteristics influencing selection into treatment (Cattaneo, et al., 2018).

Table 3.3 Correlation between S_i and weight of child

Running	Model 1 No Bandwidth	Model 2 15 day Bandwidth	Model 3 30 day Bandwidth
	-186.641***	0.031	-0.005
weight	(0.58)	(0.07)	(0.13)
	6038.381***	-0.628	-1.061
_cons	(31.44)	(3.63)	(6.26)
r2	0.633	0.002	0.000
N	60058	**101**	**196**

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

Source: Author's computation using 2012 Nutrition & Health Survey

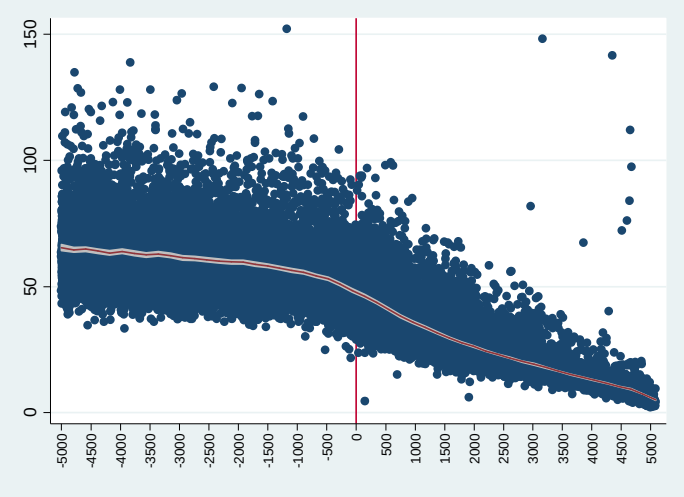
Table 3.4 Probit model of relevant observables for selection into treatment for various bandwidths

	Model 1	Model 2	Model 3	Model 4	Model 5
1 Jan 1999	15 days	20 days	25 days	30 days	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

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

Source: Author's computation using 2012 Nutrition & Health Survey

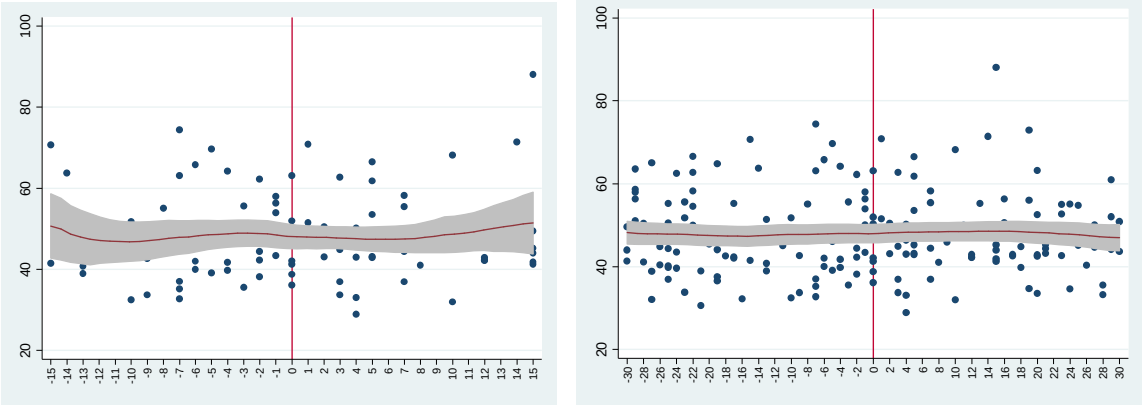
Figure 3.6 Scatter and local polynomial with confidence intervals relation between running variable and weight bandwidth of 5000 days



Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.7 Scatter and local polynomial with confidence intervals relation between running variable and weight

A. 15 day bandwidth **B. 30 day bandwidth**



Source: Author's computation using 2012 Nutrition & Health Survey

3.6.2 Choosing the correct functional form

A polynomial of order one, i.e. a linear functional form may, theoretically, 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 trade-off between simplicity, precision and stability. Linear, quadratic and cubic models are presented because, in finite samples, the ranking between different local polynomial estimators may differ from the asymptotic characteristics obtained in very large samples (Cattaneo, et al., 2018).

In this section, two formal tests are applied to guide the choice of polynomial order, as recommended by Lee and Lemieux (2010). The first is the Akaike information criterion (AIC)[83] seen in Table 3.5. The AIC for linear, quadratic and cubic models is presented 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 and the quadratic form is recommended for the 15 day model (see Appendix 5 for the AIC test over various bandwidths) (Lee & Lemieux, 2010).

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

Table 3.5 AIC for various bandwidths and polynomial orders

Bw	Order	Beta dtreat	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**

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

Source: Author's computation using 2012 Nutrition & Health Survey

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 the polynomial order is increased until all bin dummies are not significant (Lee & Lemieux, 2010).

Bin dummies are created separately for each side of the bandwidth. The *egen xtile* command is used to create a variable which categorizes the running variable by its quantiles. Various bin numbers are tested (see discussion in Appendix 6) from 4 to 16 bins for each bandwidth. In every model, be it one in which there are 4, 8 or 16 bins, be it one with a 15 or 30 day bandwidth, the first bin is taken as a reference and last bin is dropped from the model due to collinearity. I find that, in all the specifications (linear, quadratic or cubic) none of the bin dummies are significant. In other words, the number of bins, and therefore, the number of observation in the bins, do not seem to have an effect on 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.

We decided to present 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). I present the 16 bin model for the 30 day bandwidth as there are more observations and therefore can increase the number of bins. Table 3.6 presents descriptive statistics of our bin dummies in relation to S_i. For the 15 day bandwidth there are approximately 10 observations in each bin (except in bin 5), and for the 30 day bandwidth there are just over 10. Figure 3.8 and 3.9 shows the bins graphically.

Table 3.8 and 3.9 have 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. I 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 I 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 3.6 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

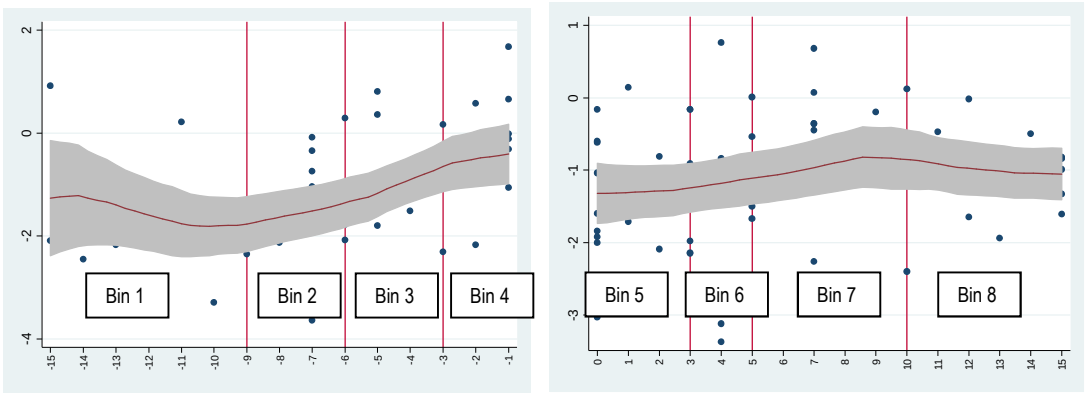
Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.7 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

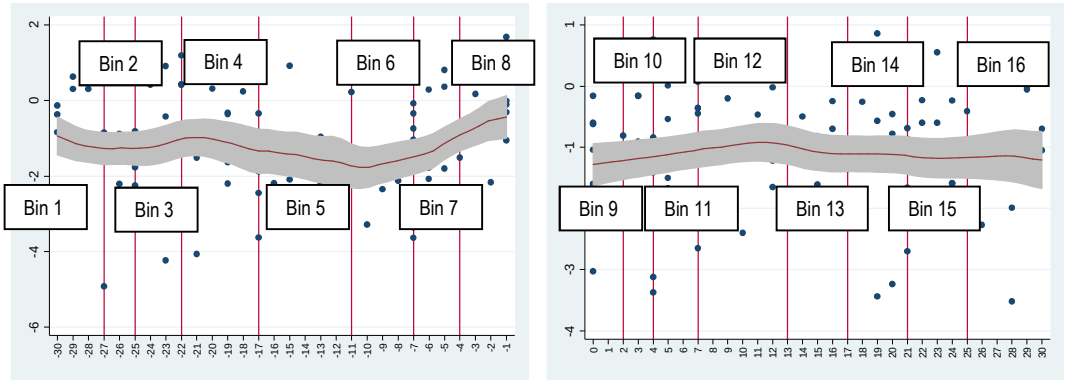
Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.8 4 bins before and after cut-off for 15 day bandwidth



Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.9 16 bins before and after cut-off for 30 day bandwidth



Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.8 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

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

Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.9 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

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

Source: Author's computation using 2012 Nutrition & Health Survey

3.6.3 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 it moves further away from it. The uniform kernel would give equal weights to all observations within the bandwidth and the Epanechnikov kernel would give quadratic decaying weights to observations within the bandwidth. In practice, (Table 3.10) our estimations are not sensitive to the choice of kernel weights as they are all significant in a similar way within bandwidth and function 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 is used. The regressions using the triangle kernel function are presented simply because it is the default (Cattaneo, et al., 2018).

Table 3.10 Average treatment effect with 15 day bandwidth & various choices of kernel functions

Kernel function	Bw	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***

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

Source: Author's computation using 2012 Nutrition & Health Survey

3.6.4 Average Treatment Effect

The Average Treatment Effect (ATE) is negative and significant for all bandwidths (between 15 and 30 days) and functional forms (polynomial of order zero to two) except one: the 30 day linear model (see Table 3.11). It is recommended (Cattaneo, et al., 2018; Lee & Lemieux, 2010) to present various functional forms and bandwidths, and in this case, 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 3.9). 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). The

linear model produce 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 (control) to the right (treated) of the cut-off (left: 40 obs., right: 52 obs.). I discuss this issue further in the next section.

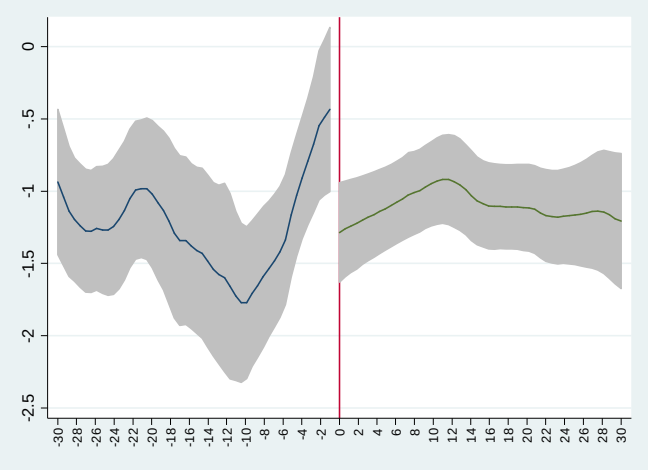
Table 3.11 Average Treatment Effect by Bandwidth and Functional Form

Characteristics of model			Estimation effect of treat		
Bandwidth	n left of c.o.	n right of c.o.	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***

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

Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.10 Local polynomial on each side of jump: 1 Jan 1999 (bw=30, polynomial order=0, kernel= Epanechnikov)



Source: Author's computation using 2012 Nutrition & Health Survey

3.7 Robustness checks

We have tackled (1) predetermined covariates, (2) the choice of bandwidth, (3) the choice of functional form, and (4) the choice of kernel function. Here I review 4 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, and (4) covariates with the same cut-off.

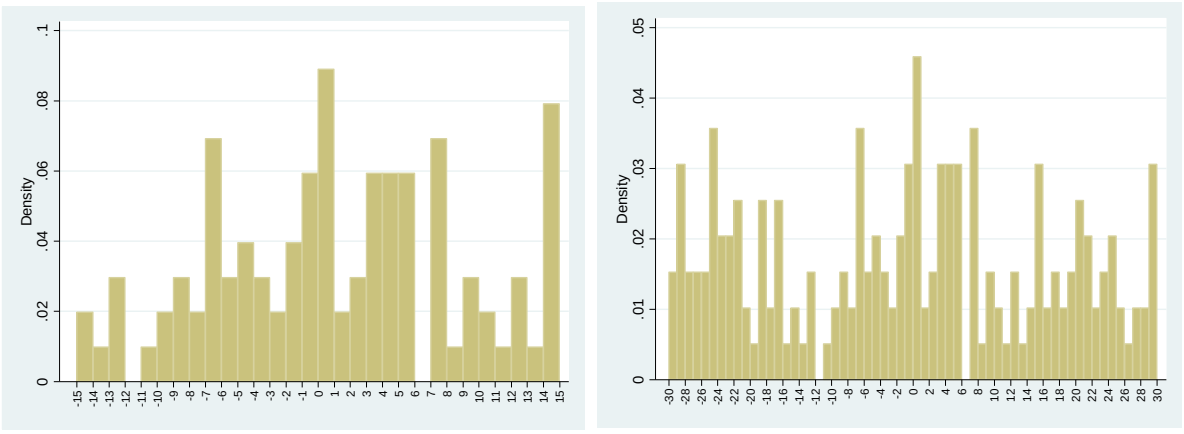
3.7.1 Density of the running variable (manipulation of the cut-off)

A basic principal 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/below the cut-off should be similar. In the case of the 30 day bandwidth the sample sizes are relatively equal (treat: 99, control: 97). However, in the case of the 15 day bandwidth, 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), however, it does lead to the question of whether it was possible for individuals to manipulate S_i. Figure 3.11 shows 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.

In order to tackle this issue the individuals who were born on 1 Jan 1999 are excluded and the distribution of S_i is re-graphed. In the case of the 15 days bandwidth, a much more uniform sample is found (Figure 3.12) with a more balanced assignment to treatment (treat:49, control: 43) while the sample seems to remain relatively equal in the case of the 30 day bandwidth (treat: 90, control: 97) (see Table 3.12 for totals across groups and dates).

Figure 3.11 Density of the running variable days born before/after 1 Jan 1999

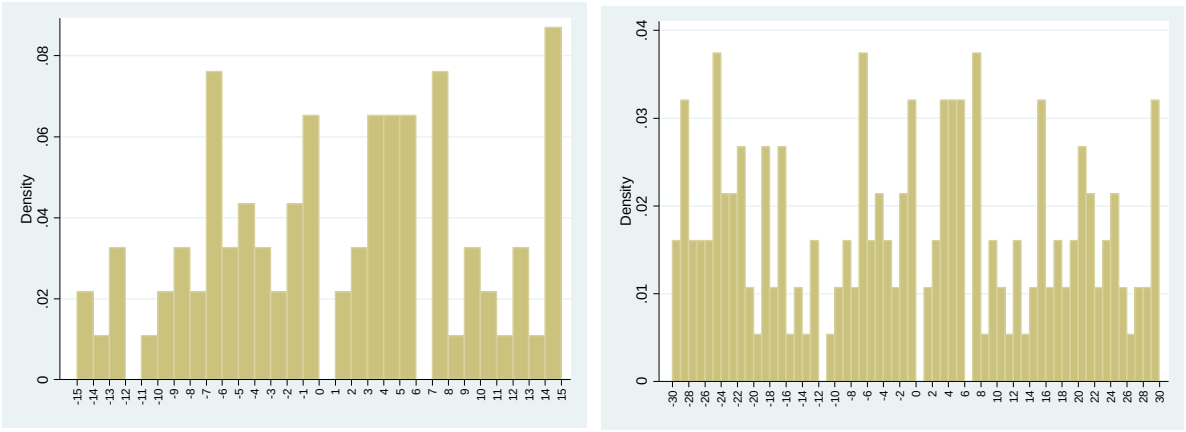
A. 15 day bandwidth **B. 30 day bandwidth**



Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.12 Density of running variable i.e. days born before/after 1 Jan 1999 excluding 0

A. 15 day bandwidth **B. 30 day bandwidth**



Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.12 Tabulation of dummy treatment with and without 1 Jan 1999

Sample	bw	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

Source: Author's computation using 2012 Nutrition & Health Survey

In order to see if there is a discontinuity in the frequency or density of the assignment variable I use two methods. Firstly, the method proposed by Lee & Lemieux (2010) and, originally, by McCrary (2008) is used, and, secondly, RD models where the dependent variable is the frequency of the assignment variable is run for various specifications of bandwidth and functional forms (Lee & Lemieux, 2010; McCrary, 2008).

The McCrary (2008) method involves a two-step process where, firstly, the assignment variable frequency count by day is estimated in a variable r, then r is imputed as a dependent variable in a local polynomial regression and represented graphically. By doing this I am looking for evidence of a jump in the frequency count of the observations around the cut-off point. Figure 3.13 shows the McCrary smoothened histogram using a 15 day bandwidth firstly (a) including all observations and, secondly (b) excluding observations born on 1 Jan 1999. In Figure 3.14 the process is repeated for the 30 day bandwidth. When every observation is included, the histogram (of 15 and 30 day bandwidth) are discontinuous at the cut-off point, however, not in a way which causes a jump outside of the confidence interval. When the 1 Jan 1999 observations are excluded, the histograms present no discontinuity (McCrary, 2008).

The results of the RD models are found in Table 3.13. The results indicate that, with or without the observations born on 1 Jan 1999, there are various specification which find a significant jump in the frequency of the assignment variable at the cut-off. These results seem contradictory to the McCrary (2008) method results presented above, given the RD models are significant for some specifications while the local polynomial graphs do not show a jump in any specification. The jumps illustrated in Table 3.13 are not consistent across the different specifications, however, they seem to presenting some partial evidence of a jump in frequency. In order to tackle this issue, in the next section I present a second robustness check where the sensitivity of the outcome and results to the observations near the cut-off is tested.

Table 3.13 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

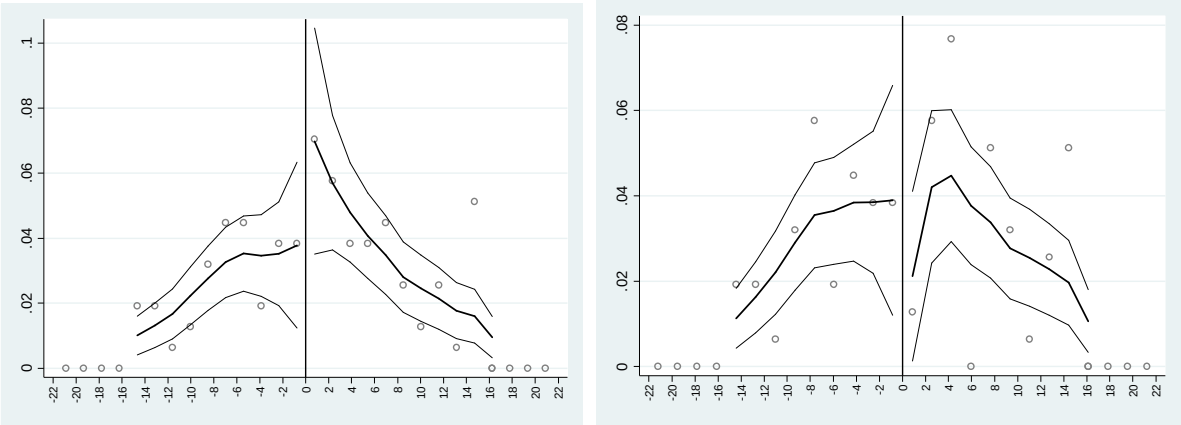
Sample	Bw	Left	Right	Linear	Quadratic	Cubic
Whole Sample	15	41	52	**2.34*** **	2.39	0.72
Sample excluding 1 Jan 1999	15	41	43	0.107	**-3.97***	**-9.31*** **
Whole Sample	30	94	97	**2.33*** **	**2.07***	2.27
Sample excluding 1 Jan 1999	30	94	88	**1.07***	-0.105	-1.73

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

Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.13 McCrary smoothened histogram of frequency counts in S_i with a 15 day bandwidth

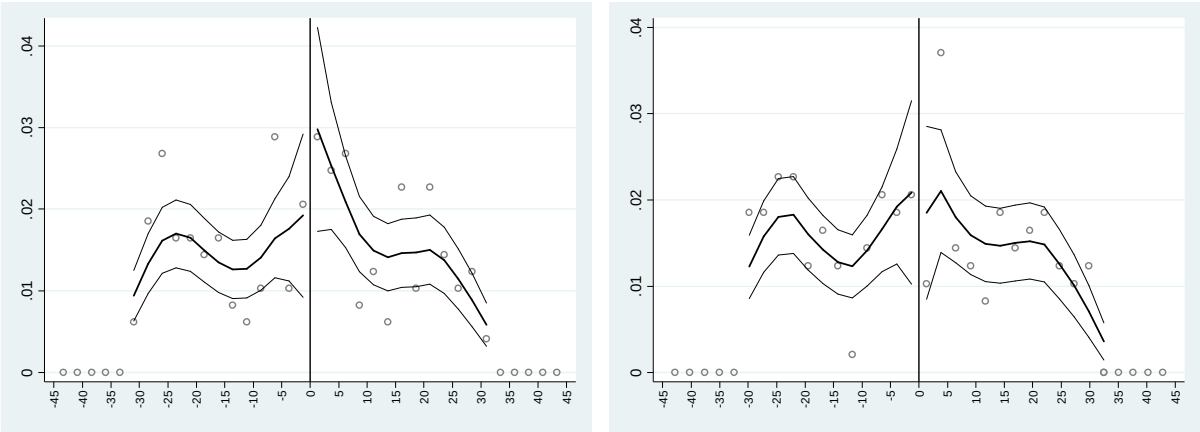
A. Including all observations **B. Excluding 1 Jan 1999**



Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.14 McCrary smoothened histogram of frequency counts in S_i with a 30 day bandwidth

A. Including all observations **B. Excluding 1 Jan 1999**



Source: Author's computation using 2012 Nutrition & Health Survey

3.7.2 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 survey 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).[84] I 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, I tackle this potential problem by measuring the sensitivity of the model to the observations around the cut-off.

The idea of this method, found in Cattaneo et al. (2018), is to exclude individuals near the cut-off and to repeat the estimation with the remaining sample. In Table 3.14 I present the two models for each bandwidth and polynomial order (1) without exclusion, and (2) excluding observations from 1 Jan 1999. Table 3.14 shows the ATEs are significant in similar ways across samples. For example, the quadratic 15 day model is negative and significant for both the whole sample and that which excludes children born on 1 Jan 1999. Additionally, the linear 30 day model is not significant for any of the two samples. The only difference is found in the cubic 15 day models where it is significant with the whole sample and not significant with that excluding children born on 1 Jan 1999. This leads us to believe the effect of the observations near the cut-off is important when the sample size is small, i.e. in the 15 day model. Therefore, this might be an argument

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

in favor of a larger sample size. As a final remark, I can also highlight that the "preferred" functional form was the quadratic for the 15 day bandwidth and the cubic for the 30 day bandwidth. As Table 3.14 indicates, the results for the quadratic model in the 15 day bandwidth are very similar for both samples, as are those of the cubic model in the 30 day bandwidth (Cattaneo, et al., 2018).

Table 3.14 Sensitivity of RD model to observations near the cut-off (15 and 30 day bandwidths, and triangle kernel function)

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

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

Source: Author's computation using 2012 Nutrition & Health Survey

3.7.3 Placebo effects and anticipation bias

We have four placebo effect tests. (1) Anticipation bias, (2) placebo effects after the crisis; (3) New Year's Day effect; and (4) 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.

3.7.3.1 Measuring placebo effects by changing S_i

The three placebo effects are described in Table 3.15. There are no placebo effects except on two isolated models: 1 Feb 1999 cubic model, and, 1 Apr 1999 linear model (note four different bandwidths are presented for every model in order to establish robustness).

3.7.3.2 Anticipation bias & placebo effects after the crisis

Firstly, no significant effects on the outcome variable in the months running up to the crisis are found. 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 this hypothesis. Two significant effects after the crisis are found on 1 Feb and 1 April 1999, represented in Figure 3.15. Separate local polynomial regressions are plot on each side of the placebo cut-off and find that in neither case is the jump outside of the confidence interval. I 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 there is no visible graphic representation of an effect in Figure 3.15.A or 3.15.B.

Table 3.15 Placebo effects before and after the crisis

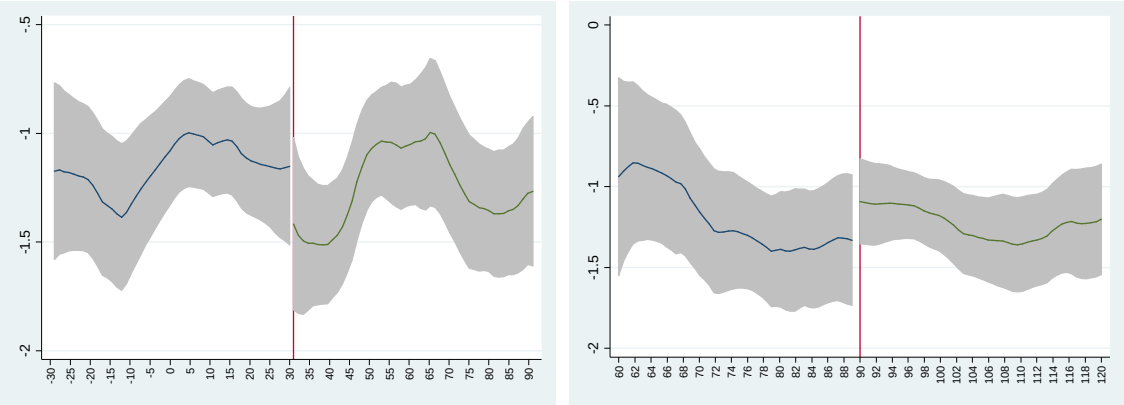
Year	Day/Month	bw	Linear	Quadratic	Cubic	n left of c.o.	n right of c.o.
1998	1-Dec	15	0.04	0.33	-0.72	37	56
1998	1-Dec	20	-0.12	0.28	0.33	49	63
1998	1-Dec	25	-0.13	0.008	0.46	61	78
1998	1-Dec	30	-0.15	-0.01	0.2	68	93
1998	1-Nov	15	0.92	1.26	0.28	43	29
1998	1-Nov	20	0.69	1.2	1.18	62	45
1998	1-Nov	25	0.46	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

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

Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.15 Local kernel on each side of placebo jump (bw=30, polynomial order=0, kernel= Epanechnikov)

A. 1 Feb 1999 **B. 1 April 1999**



Source: Author's computation using 2012 Nutrition & Health Survey

We run a probit model to measure the observable differences between the placebo treatment and control groups in both the placebo effect for 1 Feb 1999 and 1 Apr 1999. As above, this will help us determine if the samples are similar in their observable characteristics. Table 3.16 and 3.17 show, neither 1 Feb 1999 no 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, leads us to suggest that this placebo effect does not hold up to robustness checks.

Table 3.16 Probit dummy treatment using 1 Feb 1999 as placebo cut-off, measuring effect of observables

	15 days	20 days	25 days	30 days
1 Feb 1999	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

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

Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.17 Probit dummy treatment using 1 Apr 1999 as placebo cut-off, measuring effect of observables

	15 days	20 days	25 days	30 days
1 Apr 1999	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

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

Source: Author's computation using 2012 Nutrition & Health Survey

3.7.3.3 New Year's Day effect

We measure the effect on New Year's Day in the years preceding the crisis (1994-1998) in Table 3.18. No significant effect except for in the 25 day bandwidth cubic model in 1995 is found. Figure 3.16 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.

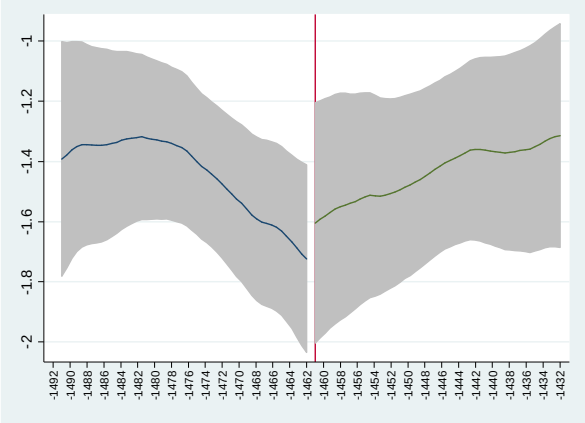
Table 3.18 Placebo New Year's Day effect

Year	Day/Month	bw	Linear	Quadratic	Cubic	n left of c.o.	n right of c.o.
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

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

Source: Author's computation using 2012 Nutrition & Health Survey

Figure 3.16 Local polynomial on both sides of placebo cut off: 1 Jan 1995 (bw=30, polynomial=0, kernel=Epanechnikov)



Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.19 presents various probit models of the dummy treatment using 1 Jan 1995 as a cut-off point. 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 groups). 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) there are 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 3.19 Probit dummy treatment using 1 Jan 1995 as placebo cut-off, measuring effect of observables

	15 days	20 days	25 days	30 days
1 Jan 1995	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

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

Source: Author's computation using 2012 Nutrition & Health Survey

3.7.3.4 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. The survey does not have information on whether the parents had access to banking services. However, it is possible to identify the parents who belong to the lower end of the income distribution. I define this as the first decile, that is, 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 7 for descriptive statistics on the income distribution). I argue 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 3.20 presents the sample sizes for treatment and control groups for

different bandwidths. The 90 day bandwidth already has a relatively small sample (Treat: 37, Control: 29) which is why it is not possible to reduce the bandwidth further. Table 3.21 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 the distribution is decomposed into deciles and the ATE is estimated within each decile (see Table 3.22) there are no effects within any decile. This implies that the effect is driven by the variation between the deciles rather than the variation within them.

Table 3.20 Sample size of first quantile of income distribution by bandwidth

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

Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.21 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

Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.22 RD models for 1 Jan 99 by deciles and quantiles 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

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

Source: Author's computation using 2012 Nutrition & Health Survey

Does this mean there are no differentiated effects for poorest and richest? Not exactly. The sample is divided into "poor" and "non-poor" by using the 2012 poverty line defined by INEC of $77 per capita per month. Table 3.23 shows that there is a strongly negative and significant effect on the "poor" in all bandwidths and functional forms except one while Table 3.24 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 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 3.23 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***

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

Source: Author's computation using 2012 Nutrition & Health Survey

Table 3.24 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**

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

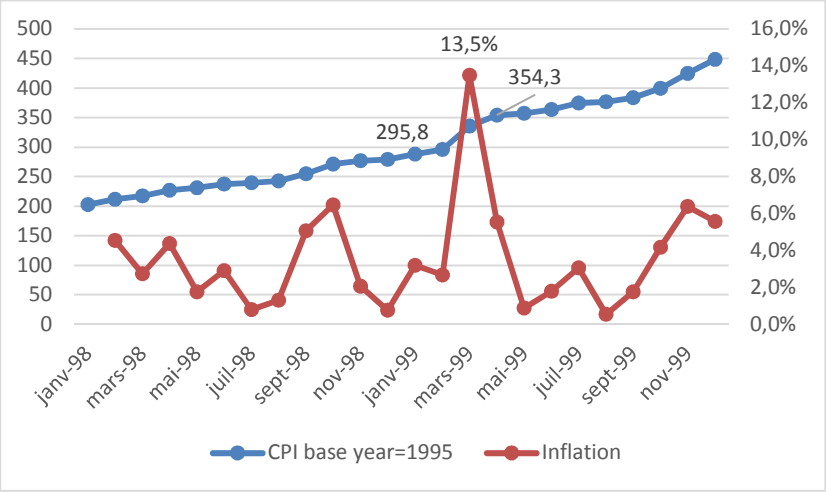
Source: Author's computation using 2012 Nutrition & Health Survey

3.7.4 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 or inflation. 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. Figure 13.7 shows 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 3.17 CPI and inflation 1998 - 1999



Data source: Institution Nacional de Encuestas y Censos (INEC)

Graphic representation: Author

3.8 Conclusion and discussion

I find a significant deleterious effect of the outbreak of the 1999 Ecuadorian financial crisis (Jacome, 2004; Cantos Bonilla, 2006; Martinez, 2006) on the 2012 z-scores 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 a 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; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017;

Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

Throughout this paper I provide evidence of a robust unanticipated effect. I justify the exogeneity of the effect by demonstrating that relevant observable characteristics are not significant determinants of selection into treatment. Data-driven methods are used to select an appropriate bandwidth: (1) the AIC, and, (2) a dummy variable test in order to select the polynomial order. Furthermore, the sensitivity of the results to kernel functional forms is tested (Cattaneo, et al., 2018; Lee & Lemieux, 2010). In addition, placebo effects are tested in the months and years predating and following the crisis; and the effect of the density of the running variable and the observations near the cut-off on the outcome is analyzed. Finally, other observables are tested to see if they have the same cut-off.

This study contributes to the literature in three ways: (1) I 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) I 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) I 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 logistical regression methods which compare the outcome variable before to after the treatment without providing an appropriate counter-factual (control group). Additionally, I have not found studies which use RD models or which analyze the long term health effects of pre-natal exposure the 1999 Ecuadorian crisis.

Notwithstanding, there are various challenges that are tackled with the evidence presented in this paper. Firstly, despite testing and not finding any anticipation effects in the months before the crisis, I 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, I 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, it is not possible to test whether individuals with no access to financial services were effectively sheltered from the crisis, however, this is not the objective of the chapter.

I 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 I cannot control for. I 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.

In this Chapter (3), I argue there are other mechanisms (aside from a deficiency in micornutrients) that may determine malnutrition at an individual level in the long run. I measure an acute maternal stress shock. In Chapter 4, I wish to assess how strict social hierarchies may affect individual health. I measure the effect of a chronic level of stress brought on by income inequality on stunting and I use the mechanism discerned in this Chapter (3) to construct the argument that a disadvantageous social context may also have a deleterious effect on children's growth patterns.

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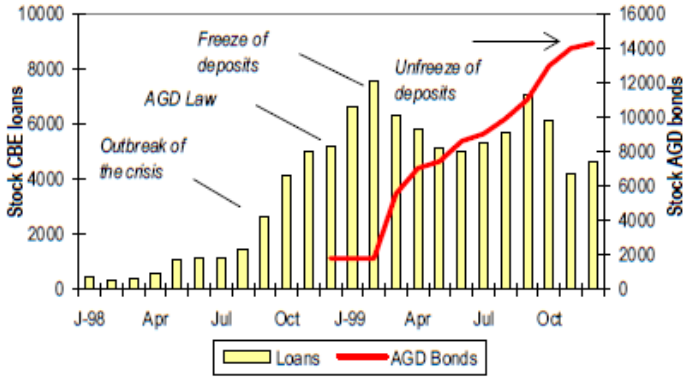
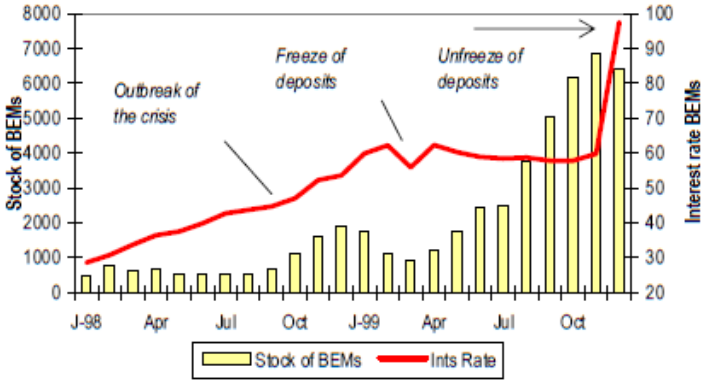
Appendix 1: Jacome 2004 Figures on Ecuadorian Crisis

Figure A1.1 Financial assistance to banks (Billions of Sucres)



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

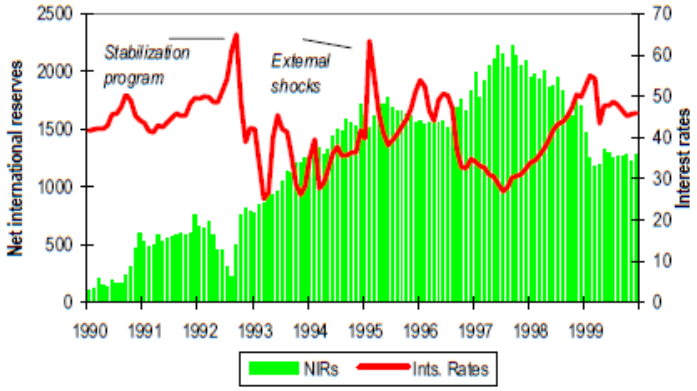
Figure A1.2 Open Market Operations (Billions of Sucres and annual rate)



BEM: Government Bonds sold to mop up liquidity (Bonos de Estabilizacion Monetaria)

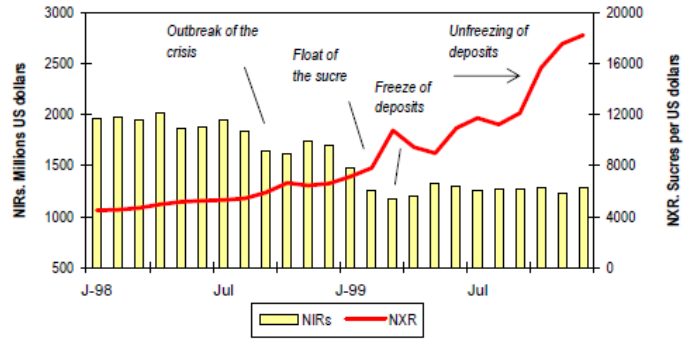
Source: Jacome, 2004; Source of data cites in Jacome, 2004: Central Bank of Ecuador

Figure A1.3 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

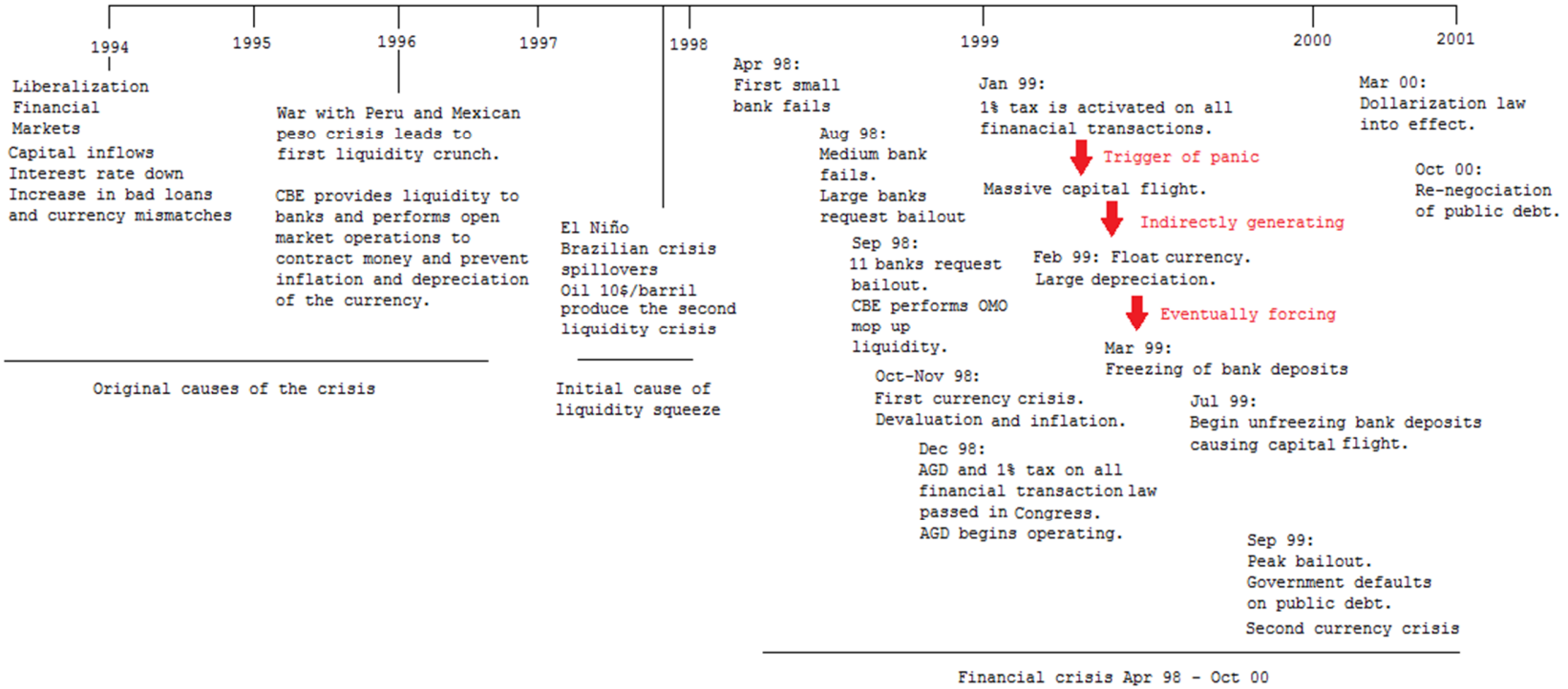
Figure A1.4 Net international reserve and nominal exchange rate (Millions of US dollars and Sucres per Dollar)



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

Appendix 2: Chronology of Crisis

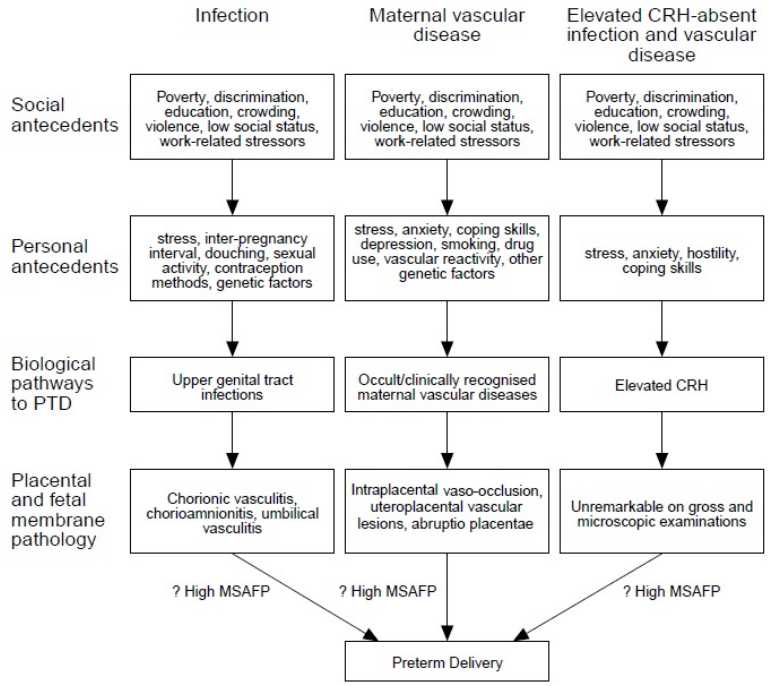
Figure A2.1 Chronology of Ecuador's 1999 Financial Crisis



Source: : Jacome, 2004 , Graphic representation: Author

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

Figure A3.1 Prenatal maternal stress pathway

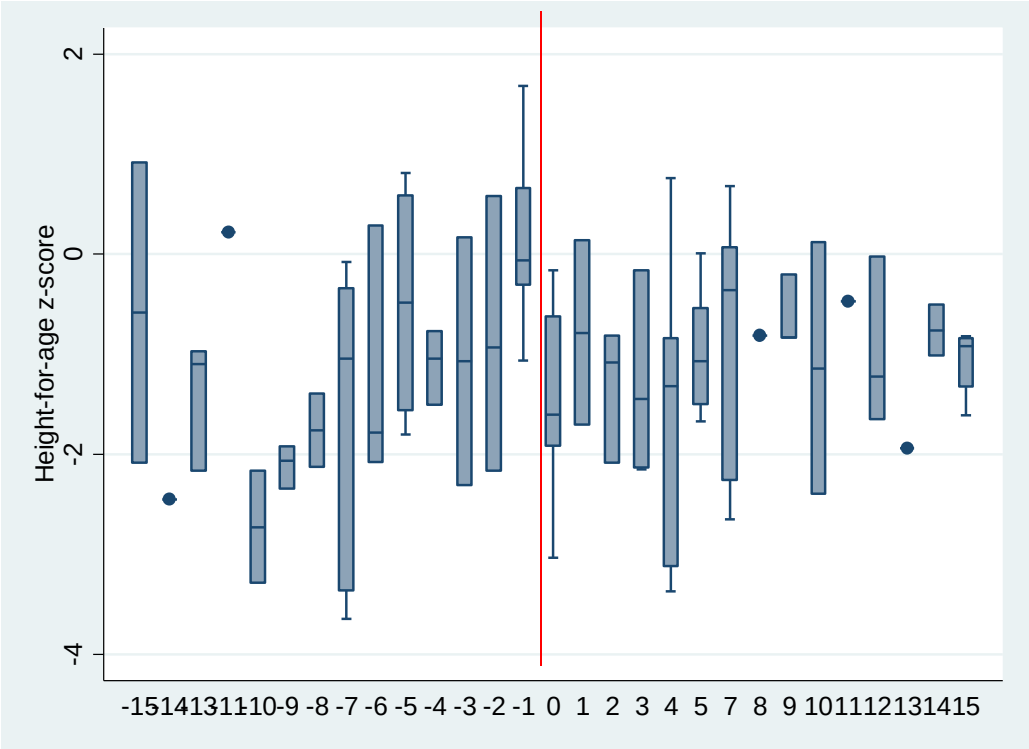


Source: C. Holzman, et al., 2001, Pregnancy outcomes and community health: the POUCH study of preterm delivery, Paediatric and perinatal Epidemiology, 15(2), pp. 138.

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

In Figure A4.1 the x-axis represents 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. In this case I am using the optimal bandwidth calculated in the article, i.e. 15 days. Therefore, the average values of z-scores per day as they are for children born 15 days before/after the crisis are seen.

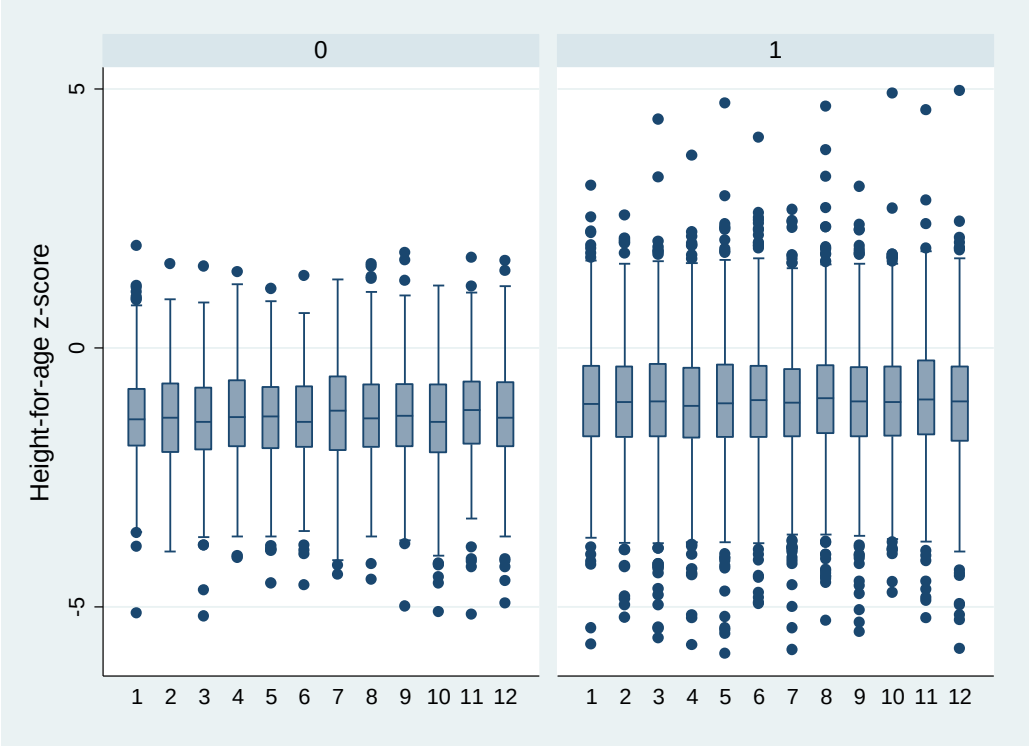
Figure A4.1 Box-plot z-score height for age by day of birth for sample of children born just before/after crisis



Source: Author's computation using 2012 Nutrition & Health Survey

In Figure A4.2 the x-axis is also the running variable where zero is equal to the cut-off point (1 Jan 1999). However, in this figure the variable is measured in months, therefore, the negative values represents the number of months born before the crisis, while the positive numbers represents the number of months born after the crisis. This implies that the bandwidth is one year wide, that is to say, the average values per month a year before and after the crisis are shown.

Figure A4.2 Box plot z-score height for age by month of birth for 12 months before/after cut-off



Source:

Source: Author's computation using 2012 Nutrition & Health Survey

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

Table A5.1 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

Source: Author's computation using 2012 Nutrition & Health Survey

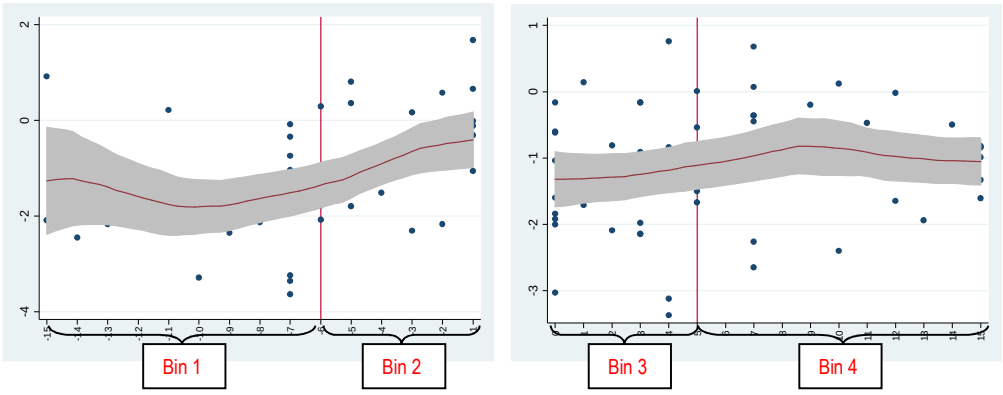
Appendix 6: Creating bin dummies for 15 and 30 day bandwidths.

Creating bin dummies for 15 day bandwidth

We created bin dummies separately for each side of the bandwidth. Figure A6.1 shows 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 the median would 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 A6.1 Median of observations before and after cut-off for 15 day bandwidth



Before cut-off **After cut-off**

Source: Author's computation using 2012 Nutrition & Health Survey

When the RD model (using OLS) using bin dummies (total 4 by taking the two on each side) is run, 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 A6.1 Regression discontinuity model (15 day bandwidth, various polynomial forms, OLS regression) 4 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
DTreat	1.003	-0.713	-0.633
	(0.90)	(1.22)	(1.31)
Running	-0.010	0.362	0.272
	(0.07)	(0.20)	(0.40)
DTreat*Running	-0.008	-0.310	-0.196
	(0.08)	(0.24)	(0.46)
$Running^2$		0.021	0.003
		(0.01)	(0.07)
DTreat*$Running^2$		-0.025	-0.013
		(0.01)	(0.08)
$Running^3$			-0.001
			(0.00)
DTreat*$Running^3$			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

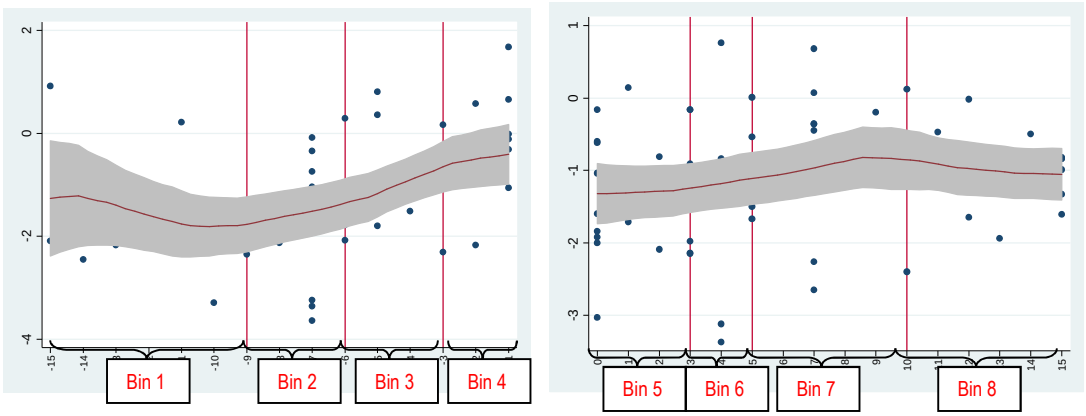
Source: Author's computation using 2012 Nutrition & Health Survey

In conclusion, firstly, the decision about the reference bin must be made carefully. Is it the objective to compare the behavior of the z-score in relation to the first bin of observations which is always going to be composed of those born the earliest before the crisis? Secondly, the appropriate number of bins also need to be decided carefully 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 given the jump is expected there.

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

Figure A6.2 4 bins before and after cut-off for 15 day bandwidth



Source: Author's computation using 2012 Nutrition & Health Survey

The RD 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 A6.2 Regression discontinuity (15 day bandwidth, various polynomial forms, OLS regression) 8 bin dummies

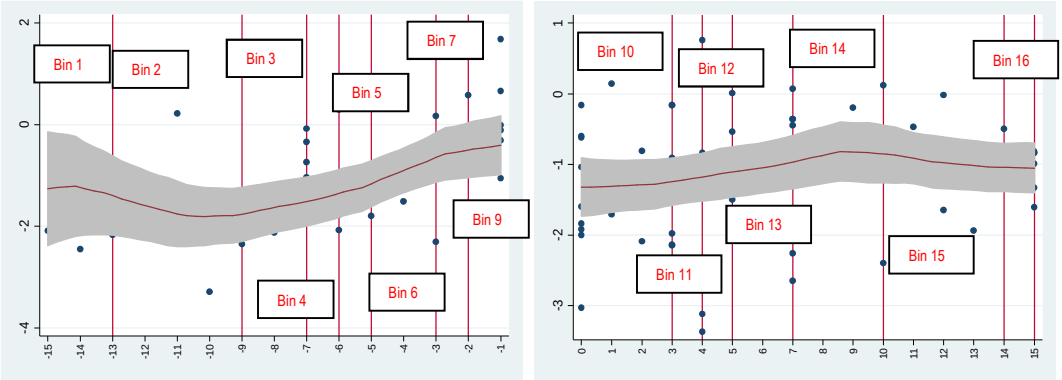
	OLS1	OLS2	OLS3
	b/se	b/se	b/se
DTreat	2.155	-0.240	-0.293
	(2.17)	(3.15)	(3.40)
Running	-0.144	0.332	0.650
	(0.12)	(0.47)	(0.90)
DTreat*Running	0.153	-0.248	-0.547
	(0.17)	(0.51)	(0.94)
$Running^2$		0.022	0.074
		(0.02)	(0.13)
DTreat*$Running^2$		-0.028	-0.084
		(0.02)	(0.14)
$Running^3$			0.002
			(0.01)
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)
r2	0.160	0.173	0.174
N	100	100	100

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

Source: Author's computation using 2012 Nutrition & Health Survey

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 (16^{th}) 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 A6.3 8 bins before and after cut-off for 15 day bandwidth



Source: Author's computation using 2012 Nutrition & Health Survey

Table A6.3 Regression discontinuity (15 day bandwidth, various polynomial forms, OLS regression) 16 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
DTreat	5.031	1.974	3.814
	(5.67)	(6.02)	(6.56)
Running	-0.192	0.723	1.420
	(0.30)	(0.66)	(1.01)
DTreat*Running	0.049	-0.957	-1.426
	(0.39)	(0.83)	(1.35)
$Running^2$		0.049	0.182
		(0.03)	(0.15)
DTreat*$Running^2$		-0.044	-0.234
		(0.04)	(0.24)
$Running^3$			0.005
			(0.01)
DTreat*$Running^3$			-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

Source: Author's computation using 2012 Nutrition & Health Survey

In order to get a better idea of what the bins contain, I 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 A6.4 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

Source: Author's computation using 2012 Nutrition & Health Survey

Table A6.5 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

Source: Author's computation using 2012 Nutrition & Health Survey

Table A6.7 Number of observations (15 day bandwidth, 16 bins)

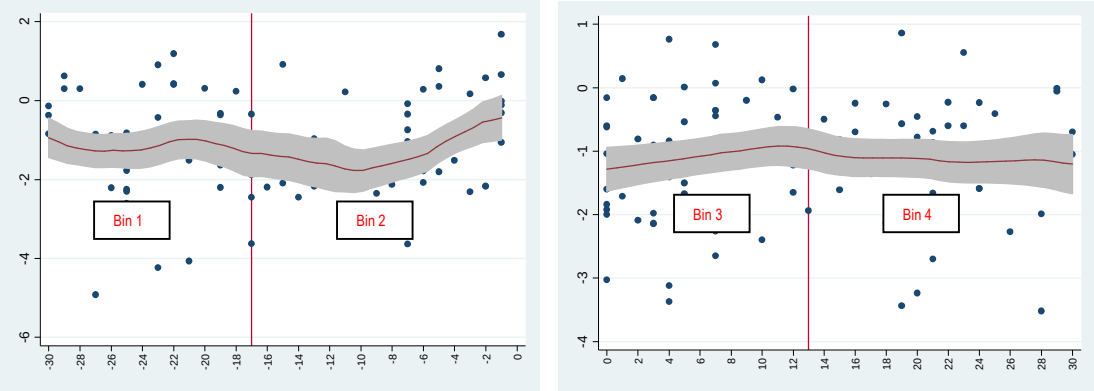
bins	Obs	Min	Max
1	6	-15	-13
2	6	-11	-9
3	9	-8	-7
4	3	-6	-6
5	4	-5	-5
6	5	-4	-3
7	10	-2	-1
8			
9	9	0	0
10	11	1	3
11	6	4	4
12	6	5	5
13	7	7	7
14	6	8	10
15	7	11	14
16	6	15	15

Source: Author's computation using 2012 Nutrition & Health Survey

Creating bin dummies for 30 day bandwidth

I use the same method to find the appropriate number of bins for the 30 day model. I 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 the 15 day bandwidth is used. I suppose that the dummies are also washing away the effect of the cut-off. In terms of number of observations, when 4 bins are carved out there are approximately 40 observations in each bin, with 8 there are about 20 and with 16 there are around 10 (with some exceptions).

Figure A6.4 4 bins before and after cut-off for 30 day bandwidth



Source: Author's computation using 2012 Nutrition & Health Survey

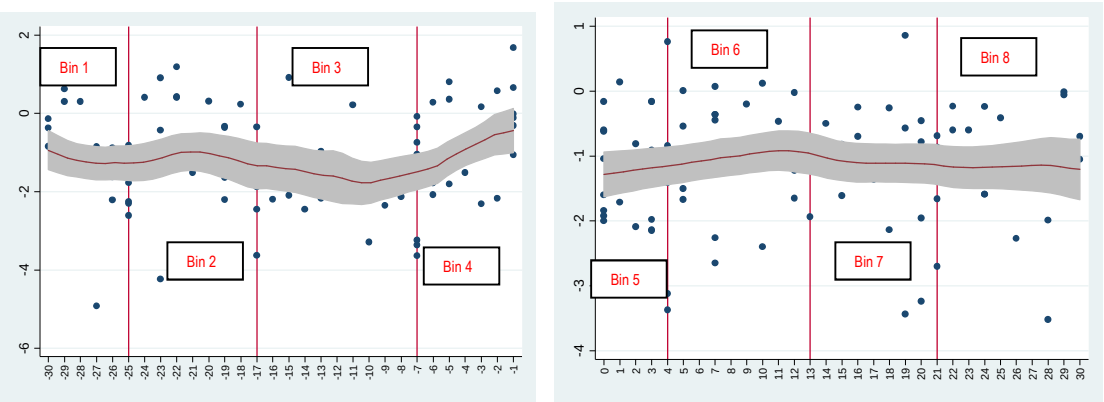
Table A6.8 Regression discontinuity (30 day bandwidth, various polynomial forms, OLS regression) 4 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
DTreat	-1.088	-2.202*	-1.755
	(0.89)	(0.97)	(1.03)
Running	0.038	0.188**	0.364*
	(0.03)	(0.06)	(0.15)
DTreat*Running	-0.029	-0.132	-0.232
	(0.04)	(0.08)	(0.18)
$Running^2$		0.005**	0.021
		(0.00)	(0.01)
DTreat*$Running^2$		-0.006**	-0.031
		(0.00)	(0.02)
$Running^3$			0.000
			(0.00)
DTreat*$Running^3$			-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

Source: Author's computation using 2012 Nutrition & Health Survey

Figure A6.5 8 bins before and after cut-off for 30 day bandwidth



Source: Author's computation using 2012 Nutrition & Health Survey

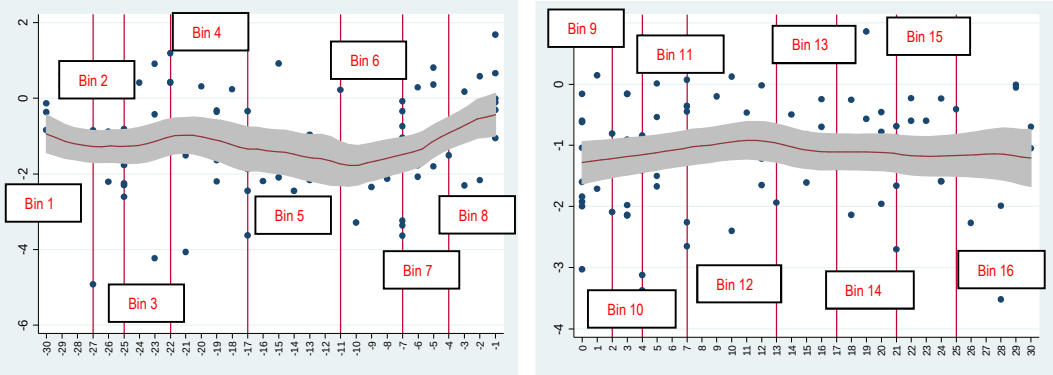
Table A6.9 Regression discontinuity (30 day bandwidth, various polynomial forms, OLS regression) 8 bin dummies

	OLS1	OLS2	OLS3
	b/se	b/se	b/se
DTreat	3.938*	3.016	3.114
	(1.79)	(1.82)	(1.85)
Running	-0.080	0.165	0.254
	(0.05)	(0.10)	(0.19)
DTreat*Running	0.022	-0.189	-0.187
	(0.07)	(0.14)	(0.24)
$Running^2$		0.008**	0.016
		(0.00)	(0.01)
DTreat*$Running^2$		-0.009*	-0.026
		(0.00)	(0.02)
$Running^3$			0.000
			(0.00)
DTreat*$Running^3$			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

Source: Author's computation using 2012 Nutrition & Health Survey

Figure A6.6 16 bins before and after cut-off for 30 day bandwidth



Source: Author's computation using 2012 Nutrition & Health Survey

Table A6.10 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

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

Source: Author's computation using 2012 Nutrition & Health Survey

Table A6.11 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

Source: Author's computation using 2012 Nutrition & Health Survey

Table A6.12 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

Source: Author's computation using 2012 Nutrition & Health Survey

Table A6.13 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

Source: Author's computation using 2012 Nutrition & Health Survey

Appendix 7: Descriptive statistics of the income distribution

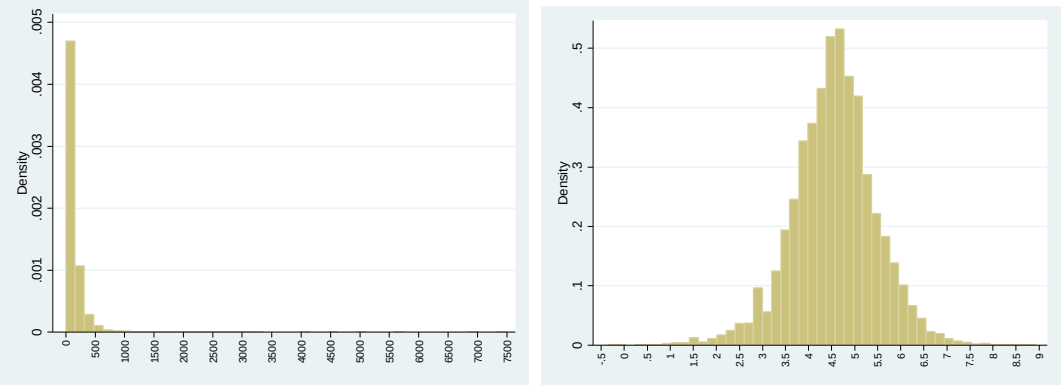
Table A7.1, shows the mean household income per capita. Figure A7.1 shows the histogram for income per capita, and Table 27 shows the sample sizes for different bandwidths.

Table A7.1 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

Source: Author's computation using 2012 Nutrition & Health Survey

Figure A7.1 Histogram of income per capita and the natural log of income per capita



Source: Author's computation using 2012 Nutrition & Health Survey

Table A7.2 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%

Source: Author's computation using 2012 Nutrition & Health Survey

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.

Table A7.3 shows 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 A7.3 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

Source: Author's computation using 2012 Nutrition & Health Survey

Chapter 4

Malnutrition and Inequality in Ecuador

4.1 Introduction

Chronic malnutrition affects 1 in 4 children in the world (De Onis, et al., 2012), and is the root cause of just under half (45%) of child (age<5 years) deaths (Horton & Lo, 2013). It is an important public health problem in many Latin American countries, particularly among indigenous populations in countries with strong socioeconomic disparities such as Bolivia, Peru, Ecuador, Guatemala and Honduras (Larrea & Freire, 2002; Farrow, et al., 2005).

There is considerable evidence that reduced protein-energy malnutrition is associated with deficits in cognition and school achievements (Grantham-McGregor, et al., 2000). Grantham-MacGregor, et al. (2007) find[85] that stunting[86] is related to literacy, numeracy, grade repetition, dropouts and intelligent quotient later in life. They argue that children who do not reach their developmental potential[87] have fewer years of education and learn less per year of schooling.[88] Their results support the conclusion that growth restriction has long-term functional consequences (Granthan-MacGregor, et al., 2007; Grantham-McGregor, et al., 2000; Walker, et al., 2000; Walker, et al., 2007) therefore potentially playing a key role in the intergenerational transmission of poverty.

There are two immediate causes of chronic malnutrition, firstly, insufficient access to nutrients and secondly, high disease exposure (Larrea & Freire, 2002). Biological mechanisms determine malnutrition at an individual level, however, the lifestyles and behaviours that lead to both a reduced nutritional intake and a high level of disease exposure may, in fact, be shaped and constrained by socioeconomic context and regional disparities at the aggregate level (Larrea & Freire, 2002; Diez-Roux, 1998).

[85] In their study on developmental potential in the developing world, that children who were currently stunted (chronically malnourished) were less likely to be enrolled in school, more likely to enroll late, attain lower achievement levels or grades and have poorer cognitive abilities.
[86] Predicted age of walking, later cognition and/or school progress
[87] Are less likely to become productive adults
[88] They estimate that the loss in adult yearly income from being stunted (chronically malnourished) is 22.2%, assuming an increase in yearly income of 9% per year of schooling (Granthan-MacGregor, et al., 2007). Other authors have also found that growth-restricted children have significantly poorer performance than non-growth-restricted children on a large range of cognitive tests.

Angus Deaton argues that inequality is relevant because income is relevant (Deaton, 2003) while others argue it is only relevant when income cannot explain the variance in health (Preston, 1975; Lynch, et al., 2004). I argue that inequality has inherent effects, independently of income, due to the intrinsic characteristics of unequal societies (Wilkinson, 2000), such that, the experience of having a low income, in societies with high levels of inequality is different than it would be in societies with low levels of inequality.

In this study, I measure the effect of inequality on chronic child malnutrition (stunting) in Ecuador using the 2006 and 2014 LSMS. The dependent variable is the z-score of height for age which is zero among healthy children and is under -2 (i.e. two standard deviations below the mean) for children with chronic malnutrition (World Health Organization, 2013; World Health Organization, 1997). The independent variable is the Gini coefficient of consumption measured over three geographic areas (the province, the county and the parish - a small administration equivalent to a village or a neighborhood).

A simple OLS model would produce biased betas as the model is not able to control for the unobservables which might affect the growth patterns of children. For example, in the 2006 model, the survey does not have information on the height or BMI of the parents. If genetics affect the growth patterns of children then the OLS model will produce biased betas. This also occurs in the 2014 model, as this model is lacking the indicator for geographic isolation.[89] If this indicator of isolation has an effect on the health outcomes of children then the betas in this OLS model will also be biased.

Therefore, in order to clean the betas of the Gini coefficients from the bias produced by omitted relevant variables the IV methodology is used. In the 2006 models, the instrument is the proportion of households which suffered from a draught in the last year. In the 2014 models, the instrument is the proportion of households who suffered a natural disaster in the last year. I believe this instrument is exogenous as it is an unanticipated meteorological event. In every model, there are controls for household consumption per capita, as well as, for the individual characteristics of the child, the parents, the household, and other contextual variables and include fixed effects for ethnicity. By including the household consumption variable, the effect of income on stunting is controlled for. The models on the 2006 LSMS and, on the 2014 LSMS are run separately it is not possible to pool the two survey data because our instrument is slightly different in each survey.

Our results show that the Gini coefficient has a significant deleterious effect on the z-score of height-for-age in 2006 but not in 2014. This may be due to a change in the severity of the Gini coefficients (province, county, parish) between 2006 and 2014. Other socio-economic indicators have also improved over this period: between 2007 and 2015 the incidence of poverty dropped by 14% from 37% to 23%, the incidence

[89] mean distance of properties in the parish to a main highway variable

of extreme poverty halved from 16% to 8% and the national Gini coefficient dropped from 0.55 to 0.47 (Instituto Nacional de Estadistica y Censos, 2015). Perhaps the improvements between these two periods are conducive to an erosion of the effect of inequality on individual health. Notwithstanding, the way the Gini coefficients change and how this affects the way stunting changes is not the topic of this paper, although it is related and would be interesting for further research.

The results are therefore not conclusive; however, they do give us partial evidence of an effect. It is argued that inequality erodes social cohesion, increasing psychosocial stress and ultimately affects individual health (Wilkinson, 1996; Ellison, 2002; Macinko, et al., 2003). A highly unequal context may lead to increased chronic stress due to feelings of anxiety, exclusion, shame and mistrust which may arise among those least advantaged (Lynch, et al., 2004; Wilkinson, 2000; Davey Smith & Egger, 1996; Lynch, et al., 2000; Lynch, et al., 2000; Lynch, et al., 2001) although I do not test this directly. In order to explain how this might affect children I propose a pathway which is based on the effects of chronic stress during pregnancy. Chronic stress increases the levels of Croticotrophin-Releasing Hormone (CRH), a hormone which regulates fetal maturation, increasing the risk of LBW which is an important determinant of chronic child malnutrition (Beydoun & Saftlas, 2008; Camacho, 2008; Mansour & Rees, 2011; Marins & Almeida, 2002; Willey, et al., 2009; Aerts, et al., 2004; El Taguri, et al., 2009; Adair & David, 1997). This may be due to a change in epigenetics brought on by a change in the fetal environment, as discussed in Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador) (Almond & Currie, 2011; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

The main limitation of this Chapter is the way I measure the Gini coefficient over small areas. The LSMS that includes measures of income and consumption is not representative at the local level and census data in Ecuador does not have information on income or consumption. Therefore, we estimate the Gini coefficients over small areas using the method proposed by Elbers et al. (2003). Using the LSMS they estimate the joint distribution of consumption and use the fitted model parameters to generate the distribution of consumption for any subpopulation of the census. They rely on this simulation of household

consumption to calculate the conditional distribution of the Gini coefficient, its point estimate and its prediction error (Elbers, et al., 2003).

The *small area estimates* (which I refer to as SAE) model depends heavily on a degree of heterogeneity which cannot be controlled for methodologically and cannot be guaranteed empirically using the method proposed by Elbers et al (2003). In this paper, I make a focused effort (in the 2010-2014 model) on dividing the country into homogeneous regions so as to reduce the effect of heterogeneity, however, I am unable to generate Gini coefficient which are not systematically under-estimated. I suspect the model over-fits to the conditions of the middle and lower income earners, resulting in a prediction model which is not therefore, generalizable to other samples outside of those found in the LSMS. When it is simulated onto the census it reproduces the systematic bias (Tarozzi & Deaton, 2009).

Notwithstanding, it is worth saying there are very limited options in terms of alternatives for Ecuador. Given there is no income or consumption data in the census, it is otherwise, not possible to measure the Gini coefficient over small areas. Alternative methods, particularly deep neural networks based on error backpropagation are more powerful in terms of prediction error. These models are validated by measuring the mean squared error on a separate (MSE) set of data. However, the MSE is only a proxy of the true error as the validation dataset may differ statistically from the true distribution. Additionally, these methods yield black box "hidden" layers where the parameters (correlation coefficients) are not explicit, it is difficult to render deep learning models interpretable. Some methods exist, however, the methods are fairly new (Montavon, et al., 2018). Despite the various limitations of the Elbers et al. (2003) model, the method is explicit and computationally accessible, which is way I chose it (Elbers, et al., 2003).

The contributions of this study are threefold. In the Lynch et al. (2004) review of 98 studies on the inequality health relation there is only a small percentage of studies which focus on children's health (23.7%). Most of these studies measure infant mortality, which unlike nutrition, does not play a role in the intergenerational transmission of poverty (Lynch, et al., 2001). Secondly, only 10.2% of the studies include Latin American countries which is a shortfall given it is one of the most unequal regions of the world (Inter-American Development Bank, 2000) and perhaps the ideal testing ground for the effect of inequality. Finally, only 9.2% of the studies measure the Gini coefficients at different levels. In this study, I find that the magnitude of the effect of the Gini coefficient is smaller as the areas over which it is measured decreases in size. Therefore, in order to fully assess the impact of inequality it is important to take different levels of aggregation as I have done. This Chapter expands on the evidence first put forward by Larrea and Kawachi (2005).

4.2 Literary review

In this section I will attempt to give the reader a comprehensive understanding of the health-inequality relation. Firstly, in order to provide a conceptual framework, the theorized functional relation between income, inequality and health is reviewed. Secondly, so as to provide the context into which this study will fall and how it contributes, the empirical evidence of the effect of inequality is reviewed. Thirdly, in order to present an understanding of the inherit effects of inequality, the pathways through which it is argued the nutrition-inequality relation runs is outlined.

4.2.1 The functional relation

I will focus on two of the hypotheses presented in Wagstaff and Van Doorslaer's (2000) review of the relation between income and health: the Absolute Income Hypothesis (AIH) and the Income Inequality Hypothesis (IIH) (Wafstaff & Van Doorslaer, 2000). The AIH is the notion that individual health is a positive function of individual income and the relationship between health and income is concave. Therefore, at the individual level, each additional dollar of income raises individual health by progressively smaller amounts. Theoretically, a concave association between income and health at the individual-level is sufficient to produce and aggregate-level association between income inequality and average health (Rodgers, 1979).[90] In other words, a one dollar income increment will improve the health of an individual on the top of the distribution by less than it will the health of an individual on the bottom of the distribution. Therefore, redistribution will improve average health.

The AIH argues that the only way income-inequality affects health is the health-purchasing-power of individual-level income, and therefore, the effect of inequality is actually an income driven effect. Notwithstanding, this hypothesis does not explain the significance of inequality on health outcomes once income is controlled for (Wilkinson, 2000). This leads us to the second hypothesis, the IIH, where individual health is a decreasing function of income inequality (Wilkinson, 2000). In this Chapter the dependent variable is measured at the individual level and the effect that income has on individual health is controlled for with the per-capita household consumption variable. This allows for the isolation of the effect inequality has on individual health independent of any income driven effect.

4.2.2 The empirical evidence

Wagstaff and Van Doorslaer (2000) find ample and strong empirical support for the AIH and some evidence for the IIH. They observe that the strength of the effect of inequality depends crucially on how well other

[90] Why does this happen? The idea is that, given the income-health relation is concave, if x income is taken from one person and give it to another, the former becomes poorer and the latter becomes richer. In this case, inequality will increase, however average income will stay the same. As a consequence, the health of the former will deteriorate more, in magnitude, than the improvement in the health of the latter. Therefore, average health will decrease even as average income is constant.

influences on health are controlled for, especially individual income and those which vary systematically over geographical areas and time (Wafstaff & Van Doorslaer, 2000).

Macinko et al. (2003) present a review of 45 studies where they find 33 (73%) have a significant inequality health relation, and 12 (27%) have a non-significant relation (Macinko, et al., 2003). Lynch et al. (2004) presented a review of 98 studies on inequality and health of which 40 (41%) found that all measures of association showed statistically significant relationships between smaller income difference and better health, another 25 (25%) were partially supportive and 33 (34%) provided no support (Lynch, et al., 2004). Wilkinson & Pickett (2006) reviewed 155 studies and found that 83 (53%) had supportive evidence, 41 (27%) had partially supportive evidence and 31 (20%) found no evidence of the association between income inequality and health (Wilkinson & Pickett, 2006).

Subramanian and Kawachi (2004) propose that the lack of association between income inequality and health in wealthy countries is due to a threshold effect. Studies conducted outside the United States have generally failed to find an inequality-health association. However, almost all the non-US countries listed in these studies are considerably more egalitarian in their distribution of income than the United States. Also, when there are cases of relatively more unequal countries there is some support for the relation. Therefore, there might be a threshold beyond which inequality is too low to have a significant effect on health (Subramanian & Kawachi, 2004).

This Chapter is based on Larrea and Kawachi (2005), however, it has various original contributions. Firstly, I use an IV model where our endogenous variable is the Gini coefficient and our instrument is arguably exogenous. Secondly, I run the model twice on two separate LSMS collected in 2006 and 2014. This shows how the effect evolved from one decade to another and allows us access to larger sample sizes (1998 had 2723 children while 2006 has 6003 and 2014 has 11473 children). Thirdly, I provide a theoretical framework which explains the pathways through which inequality may be affecting individual child nutrition based on the psychosocial effects of inequality (Wilkinson, 2000; Larrea & Kawachi, 2005).

4.2.3 The pathway

In Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador) I demonstrate empirically how an unanticipated intra-uterine stress shock affect a child's growth pattern. I argue it may be due to a change the epigenetic make of the fetus brought on by a change in the fetal environment (Almond & Currie, 2011; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al.,

2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

The implications of this finding are important to understanding the effects of inequality on individual health given, inequality may be conducive to psychological and psychosomatic stress. A social context where there are strict social hierarchies that generate a strong perception of place and station, common in relatively unequal societies, may alienate some people, increases an individual's perceptions of injustice and exclusion and produce feelings of shame or distrust. Additionally, inequality fosters inter-personal violence reducing social cohesion, and reflects a sociopolitical system with inadequate redistributive policies and depleted public health services (Lynch, 2000; Wilkinson, 1996; Kawachi, et al., 1997; Ellison, 2002; Macinko, et al., 2003).[91]

When feelings of exclusion are translated into an increase in stress, there is a risk of a change in the fetal environment. Not only can this cause a change in the epigenetic make of the fetus, as discussed in Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador), but additionally, the medical literature indicates that PNM stress can cause levels of CRH[92] - which regulates the duration of pregnancy and fetal maturation - to increase, augmenting the risk of adverse birth outcomes (Almond & Currie, 2011; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond,

[91] The unequal distribution of income seems to be a parallel phenomenon to the unequal distribution of social services relevant to individual health and nutrition (Ellison, 2002). Lynch et al. (2000) argue that income inequality is symptomatic of a lack of resources at the public level reflecting an under-investment in health and social infrastructure given it is the result of a historical, political and economic process which has influenced the nature and availability of health supportive infrastructure. This process shapes the structural matrix of contemporary life which likely influences individual health, particularly of those who have fewer resources (Davey Smith & Egger, 1996; Lynch, et al., 2000; Lynch, et al., 2001; Lynch, et al., 2004). However, this does not imply that income inequality is the cause of an inadequate redistribution system but rather another one of its outcomes. Therefore, there is not a pathway by which one (income inequality) causes the other (depleted public health services) as is found at the individual and community levels.

[92] Croticotrophin-Releasing Hormone

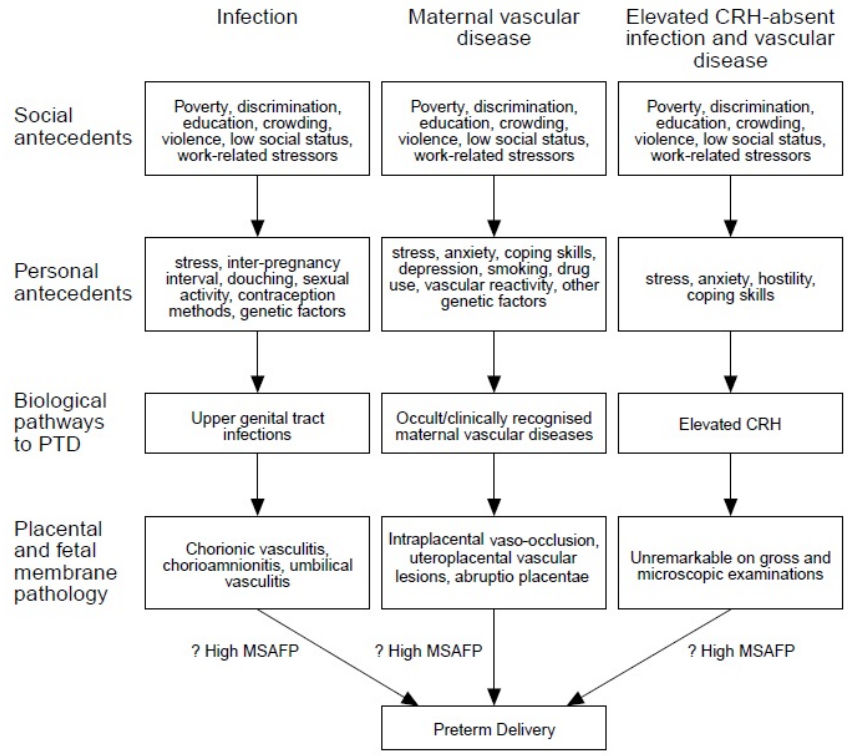
1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

Beydoun and Saftlas (2008), in their review of the literature on the effect of PNM stress on fetal growth, find that 9 out of 10 studies report significant effects of PNM stress on birth weight, LBW or fetal growth restriction (Beydoun & Saftlas, 2008).[93] Couzin (2002) summarizes how endocrinologist Hobathan Seckl, of Western General Hospital in Edinburgh, U.K., believes that 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).[94] Almond and Currie (2011) find numerous studies providing evidence of the long-term consequences of a wide variety of intra-uterine shocks (Almond & Currie, 2011; Camacho, 2008; Eskenazi, et al., 2007). Camacho (2008) finds that the intensity of random landmine explosions during a woman's first trimester of pregnancy has a significant negative impact on child birth weight[95] (Camacho, 2008).

So far I have argued inequality is associated with stress and pre-natal maternal stress with LBW. Kaplan et al. (1996) demonstrate the link is measurable and produce evidence that income inequality in the United States was significantly associated with rates of LBW. Figure (4.1) by Holzman et al. (2001) shows the various ways in which social context and stress may be conducive to a preterm delivery where babies are generally underweight. In this layout stress not only affects CRH levels but also may increase infection and vascular disease (Holzman, et al., 2001).

[93] However, they also find that the evidence was predominantly derived from animal studies. Nevertheless, and despite methodological study limitations, the overall evidence is revealing of an independent association between PNM stress and numerous physical and mental health outcomes (Beydoun & Saftlas, 2008).
[94] In other words, the body secretes glucose, cortisol, and other stress-related elements when they wouldn't be normally needed – a result now observed in LBW humans.
[95] This finding persists when mother fixed effects are included, suggesting that neither observable nor unobservable characteristics of the mother are driving the results.

Figure 4.1 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.

There are various studies which find that children with LBW are at higher risk of suffering chronic malnutrition. Marins and Almeida (2002) find[96] that LBW[97] could be characterized as important under-nutrition risk factors,[98] and that, birth weight deficits appear to have effects on a child's growth that extend for years after birth (Marins & Almeida, 2002). Willey et al. (2009) found[99] an increased likelihood of stunting (chronic malnutrition) was seen in LBW children (Willey, et al., 2009). Aerts, et al. (2004)[100] find that one of the main determinants of growth retardation was LBW (Aerts, et al., 2004). Taguri et al. (2009), who studied[101] predictors of stunting in children under five[102], found LBW to be of the main risk factors (El Taguri, et al., 2009). Adair and David (2007) studied[103] the likelihood of becoming stunted in each two

[96] in Niterói, Brazil
[97] and low family income
[98] both for the "0-12 months" and for the "above 13 months" age ranges
[99] in Johannesburg and Soweto
[100] perform a cross-sectional population-based study of determinants of growth retardation (chronic malnutrition) in children under five in Porto Alegre, Brazil
[101] used a multivariate analysis
[102] in Libya
[103] used a multivariate discrete time hazard model

month intervals and found a significant association with LBW which was strongest in the first year[104] (Adair & David, 1997).

The medical literature cited here allow us to formulate a chain of effects that connect high levels of inequality with PNM stress, which in turn has the effect of reducing birth weight, which is a significant determinant of malnutrition during infancy.

4.3 Data

There are two phases of data processing. The first phase consists of calculating the z-score of height for age using anthropometric data from the LSMS of 2006 and 2014 along with other control variables. In the second phase I estimate the Gini coefficient[105] using a SAE[106] methodology proposed by Elbers, et al. (2003). The Gini estimation I use in the 2006 models were estimated by the Universidad Andina Simon Bolivar with the participation of the author and published by the Ecuadorian government (Secretaria Nacional de Planificacion y Desarrollo, 2013) using the 2006 LSMS and the 2010 census. The Gini estimations used in the 2014 model were estimated by the author using the same Elbers et al. (2003) method, the 2014 LSMS and the 2010 census. In this section I will explain the data and in the following section I will explain the SAE methodology (Elbers, et al., 2003).

4.3.1 The Living Standards Measurement Survey

The 2006 LSMS has national coverage, 55 666 observations over 16 414 households. The 2014 LSMS has national coverage of 109 694 observations over 28 970 households. Ecuador is divided into 25 provinces, 224 counties and 1024 parishes; the 2006 LSMS covers 22 provinces, 186 counties and 443 parishes while the 2014 LSMS covers 24 provinces, 213 counties and 697 parishes. The questionnaire goes over various topics such as living conditions, education, health care, employment, food consumption and non-food consumption, income, access to credit, migration, and economic activities such as entrepreneurship and agriculture. This survey includes anthropometric measures for 6 003 children under five in 2006 and 11473 children under 5 in 2014 (Larrea & Kawachi, 2005). Table 4.1 presents descriptive statistics for the variables used in the 2006 model and Table 4.2 presents the same for the 2014 model. The Gini coefficients are obtained by simulation using the 2006 or 2014 LSTM and the 2010 census. The Gini coefficients from the 2006-2010 simulations were estimated by the Universidad Andina Simon Bolivar (Secretaria Nacional de Planificacion y Desarrollo, 2013), and the 2014-2010 simulation were estimated by the author. All the other determinants in both 2006 and 2014 were processed by the author. The list of variables between Table

[104] They argue that breast-feeding, preventive health care and taller maternal stature significantly decreased the likelihood of stunting.
[105] for every province, county and parish
[106] Small area estimates.

4.1 (2006 LSTM) and Table 4.2 (2014 LSTM) are different. The decisions of which variables to include in each model were done as a function of the relevance and significance of each determinant.

Table 4.1 Descriptive statistics LSTM 2006

Variable	Obs	Mean	Std. Dev.	Min	Max
z-score height_age	6003	-1.32	1.23	-5.53	4.05
Gini province	6003	0.40	0.05	0.31	0.56
Gini county	6003	0.39	0.05	0.29	0.56
Gini parish	6003	0.39	0.05	0.28	0.58
D. LBW	6003	0.03	0.16	0	1
D. Female	6003	0.49	0.50	0	1
Age in months	6003	30.13	17.15	0.07	59.96
Age in months^2	6003	1201.8	1066.2	0	3595.1
Age in months^3	6003	53941.5	60639.6	0	215556.8
Vaccines	6003	0.75	0.23	0	1
D. N. Supplement	5744	0.14	0.35	0	1
D. Diarrhea	6003	0.26	0.44	0	1
D. Daycare	5744	0.14	0.34	0	1
N. month breastfed	5638	4.57	2.57	0	24.00
Age mother	5940	28.29	7.38	12.00	64.00
Schooling mother	5909	8.09	4.17	0.00	22.00
Height mother					
BMI mother					
Height father					
BMI father					
Fertility mother	5847	0.16	0.08	0	0.67
D. C-section	6003	0.30	0.46	0	1
D. ObGyn	6003	0.74	0.44	0	1
D. Mother underemployed	6003	0.36	0.48	0	1
D. N. Supplement mother	6003	0.29	0.45	0	1
Ln hh consumption per capita	5986	3.97	0.75	0.75	6.48
D. welfare	6003	0.39	0.49	0	1
sqrt n children<12 in hh	6003	1.59	0.43	1	3.16
Index living conditions	6003	-0.29	1.02	-2.50	1.21
Work experience head hh	5984	24.74	14.27	0	55.00
Mean size agri. Land	5840	11.02	12.36	1.08	52.50
Mean rent/ha. ag. Land	5840	97.58	56.84	6.10	220.62
Rate attendence secondary school	6003	0.13	0.20	0	1
D. rural	6003	0.51	0.50	0	1
Ln(GDP prov)	5840	21.29	1.28	18.71	23.30
Poverty prov	6003	0.44	0.21	0.09	0.77
Mean consumption prov.	6003	105.17	44.55	50.34	197.81
Poverty county	6003	0.44	0.22	0.08	0.93
Mean consumption count.	6003	105.14	46.55	25.40	203.46
Poverty parish	6003	0.44	0.23	0.08	0.96
Mean consumption parish	6003	105.07	48.02	19.99	254.09
Index food consumption	5850	-0.18	1.64	-4.93	6.98
N. MD/10000 ppl.	5999	6.50	16.74	0	130.43
Mean distance highway	5792	1.66	0.57	1	4.00
Mestizo.ethnic	6003	0.75	0.43	0	1
Indigena.ethnic	6003	0.18	0.38	0	1
Afro.ethnic	6003	0.07	0.26	0	1
D. Rural Amazon	6003	0.07	0.26	0	1
Highlands.region	6003	0.44	0.50	0	1
Coast.region	6003	0.45	0.50	0	1
Amazon.region	6003	0.11	0.31	0	1
Galapagos.region					
Instrument	5840	0.18	0.15	0	0.53

Source: Author's computation using 2006 LSMS

Gini coefficient simulation was done by Unidad de Infromación Socio Ambiental, Universidad Andina Simón Bolívar (Secretaria Nacional de Planificacion y Desarrollo, 2013). All other determinants and table was processed by author.

*It is not possible to take the average of a Gini coefficient given it is not decomposable. This figure is not the Gini coefficient of the country it is here simply to provide the reader an idea of the range of the data.

Table 4.2 Descriptive statistics LSTM 2014

Variable	Obs	Mean	Std. Dev.	Min	Max
z-score height_age	11199	-1.14	1.38	-5.82	5.83
Gini province	11199	0.36	0.04	0.28	0.42
Gini county	11165	0.34	0.05	0.23	0.46
Gini parish	11165	0.32	0.05	0.18	0.48
D. LBW	11199	0.14	0.34	0	1
D. Female	11199	0.48	0.50	0	1
Age in months	11199	30.63	16.96	0	59.00
Age in months^2	11199	1226.0	1049.5	0	3481.0
Age in months^3	11199	54777.7	59677.7	0	205379.0
Vaccines	11199	0.94	0.15	0	1.89
D. N. Supplement	11199	0.52	0.50	0	1
D. Diarrhea	11199	0.19	0.39	0	1
D. Daycare					
N. month breastfed	11199	3.38	2.71	-1.03	12.00
Age mother	11199	5.17	7.07	0	80.00
Schooling mother	3698	4.58	3.51	0	17.00
Height mother	10867	151.97	6.35	83.65	204.00
BMI mother	10867	26.49	4.75	11.12	81.01
Height father	8157	163.80	6.88	132.95	191.50
BMI father	8157	26.09	3.93	13.81	60.40
Fertility mother					
D. C-section					
D. ObGyn					
D. Mother underemployed					
D. N. Supplement mother					
Ln hh consumption per capita	11165	4.47	0.68	1.96	7.07
D. welfare	11199	0.39	0.49	0	1
sqrt n children<12 in hh	11199	1.52	0.42	1	3.46
Index living conditions	8430	-0.48	1.65	-7.49	3.54
Work experience head hh	10786	23.39	13.95	0	90.00
Mean size agri. Land	11199	20.55	53.69	0.70	228.80
Mean rent/ha. ag. Land	11199	194.39	130.60	8.68	497.60
Rate attendence secondary school	11030	0.12	0.20	0	1
D. rural	11199	0.53	0.50	0	1
Ln(GDP prov)	11199	15.38	1.39	12.74	17.65
Poverty prov	11199	0.24	0.12	0.06	0.46
Mean consumption prov.	11199	182.33	43.52	128.48	288.17
Poverty county					
Mean consumption count.					
Poverty parish					
Mean consumption parish					
Index food consumption					
N. MD/10000 ppl.					
Mean distance highway					
Mestizo.ethnic	11199	0.74	0.44	0	1
Indigena.ethnic	11199	0.20	0.40	0	1
Afro.ethnic	11199	0.04	0.20	0	1
D. Rural Amazon					
Highlands.region	11199	0.42	0.49	0	1
Coast.region	11199	0.34	0.47	0	1
Amazon.region	11199	0.23	0.42	0	1
Galapagos.region	11199	0.01	0.12	0	1
Instrument	11199	0.03	0.03	0	0

Source: Author's computation using 2014 LSMS

Data processing: author. *It is not possible to take the average of a Gini coefficient given it is not decomposable. This figure is not the Gini coefficient of the country it is here simply to provide the reader an idea of the range of the data.

4.3.2 Dependent variable: Chronic child malnutrition

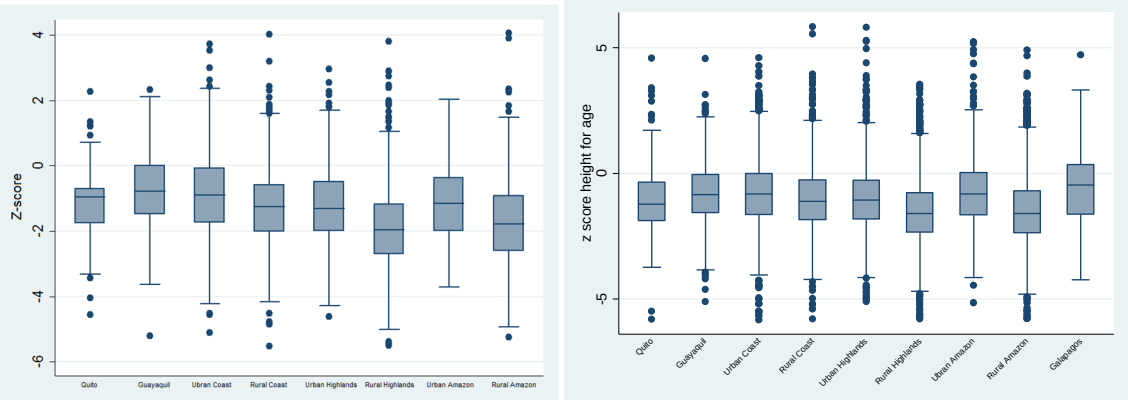
We estimate the z-score of height for age using the methodology developed and distributed freely by the World Health Organization (2013). The normalized z-score (4.1) establishes the growth standard of children by defining a normal growth curve (World Health Organization, 2013; World Health Organization, 1997).

$$z\ score = {(x_i - x_{median})}/{\sigma^x} \quad \textbf{(4.1)}$$

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). I use anthropometric data available in the LSMS (2006 and 2014) to calculate the normalized z-score for each child below the age of five. The z-score ranges from $-\infty$ to ∞ as it is measured in standard deviations from the mean which is zero. If a child's z-score is under -2, that is to say, under two standard deviations below the mean, the child is chronically malnourished or stunted (World Health Organization, 1997). Figure 4.2 and 4.3 show the z-score average in every sub-region and its distribution. The national average z-score was lower in 2006 (-1.22) (Figure 4.3.A) than in 2014 (-1.14) (Figure 4.3.B). However, the distribution is similar in that it is skewed to the left. The box-plot shows little change across regions between 2006 and 2014. Clearly, the rural highlands and rural amazon have the lowest averages in both years.

Figure 4.2 Box plot z-score of height for age over sub-regions

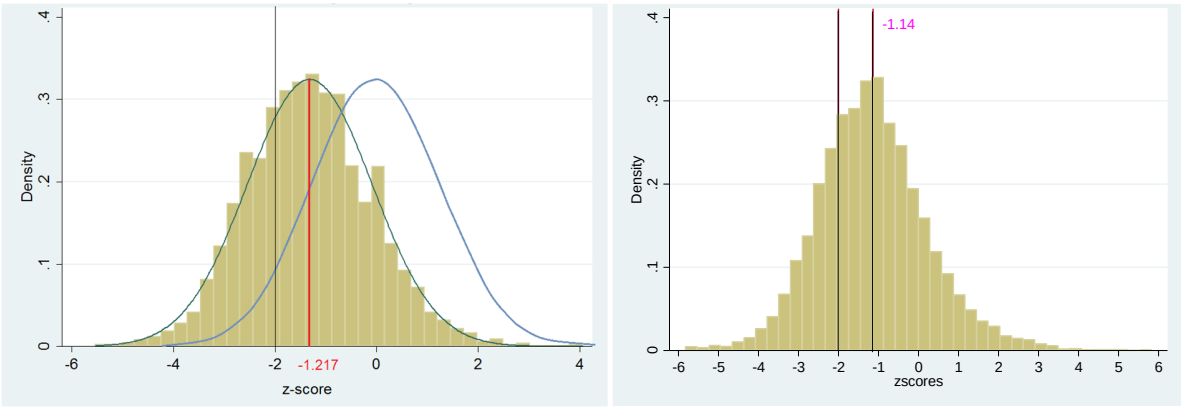
A. LSTM 2006 **B. LSTM 2014**



Source: Author's computation using 2006 /2014 LSMS

Figure 4.3 Distribution of z-score of height for age

A. LSTM 2006 **B. LSTM 2014**



Source: Author's computation using 2006 /2014 LSMS

4.3.3 Independent variable: The Gini Coefficient

The Gini coefficient is measured at the parish, county and provincial level using SAE. As mentioned above, the estimation on the 2006-2010 SAE was taken from data published by the government of Ecuador and measured by the Universidad Andina Simon Bolivar (Secretaria Nacional de Planificacion y Desarrollo, 2013). The author was part of the research project leading to this publication and contributed to the construction of the model along with many other team members. The estimation on the 2014-2010 SAE was done exclusively by the author by replicating the method used in 2006.

Household surveys that include measures of income and consumption are rarely representative at the local level because they are of insufficient size to yield statistically reliable estimates of poverty or inequality. Similarly, census data in Ecuador, which is generally of sufficient size to allow for reliability at small geographic scales, does not have information on income or consumption. Elbers, et al. (2003) estimate consumption, poverty and the Gini coefficient using this method on Ecuadorian data. They report that their results have levels of precision comparable to those of survey-based estimates. The combination of census and survey data allows for reliable estimations of the Gini coefficient in subpopulations one hundredth the size of the subpopulations in the survey data and yet obtain very similar prediction errors (Elbers, et al., 2003).

In order to explain the methodology, I will summarize the Elbers et al. (2003) article in the next few paragraphs. Let W be an indicator of welfare (the Gini coefficient) based on the distribution of household-

level consumption, y_h. Using the sample from the LSMS[107] the joint distribution of y_h and its covariates x_h can be estimated. The fitted model parameters can be used to generate the distribution of y_h for any subpopulation of the census[108] if the set of explanatory variables are restricted to those which can also be found in this census. Once a simulation of household consumption y_h is made, the conditional distribution of W (the Gini coefficient), its point estimate and its prediction error can be estimated (Elbers, et al., 2003).

Consider (2), a linear approximation of the conditional distribution of y_{ch}, where c is a sample cluster and h is household, where the vector of disturbances is $u \backsim \mathcal{F}(0, \Sigma)$. To allow for a within-cluster-correlation in disturbances the (4.3) specification where η and ε are independent of each other and uncorrelated with x_h is used (Elbers, et al., 2003).

$$\ln y_{ch} = E\left[\ln y_{ch} | x_{ch}^T\right] + u_{ch} = x_{ch}^T \beta + u_{ch} \quad \textbf{(4.2)}$$

$$u_{ch} = \eta_c + \varepsilon_{ch} \quad \textbf{(4.3)}$$

Basically, an initial estimation of β in (4.2) is obtained using OLS and the residuals of this regression are denoted as $\hat{u}_{ch}$. With consistent estimates of β the residuals e_{ch} can be used to estimate the variance of ε_{ch} (Elbers, et al., 2003).

$$\hat{u}_{ch} = \hat{u}_{c.} + (\hat{u}_{ch} - \hat{u}_{c.}) = \hat{\eta}_c + e_{ch} \quad \textbf{(4.4)}$$

Subsequently, this estimated distribution in (4.2) is used to generate the expected value of W in a subpopulation of the census which is denoted v for village. Thus it is written: $W(m_v, X_v, \beta, u_v)$ where m_v is the M_v-vector of household sizes in village v, X_v is the matrix of observable characteristics, and u_v is the vector of disturbances which is unknown and therefore estimated as explained above. The expected value of W is then $\mu_v = E[W|m_v, X_v, \zeta_v]$ where ζ is the vector of model parameters which includes the disturbances. In constructing an estimator of μ_v ζ_v is replaced with $\hat{\zeta}_v$. This gives us $\hat{\mu}_v = E\left[W|m_v, X_v, \hat{\zeta}_v\right]$ which is often analytically intractable so simulation is used to obtain the estimator $\tilde{\mu}_v$ (Elbers, et al., 2003).

The difference between $\tilde{\mu}_v$ and the actual level of W has three components and can be written as follows (Elbers, et al., 2003)

$$W - \tilde{\mu} = (W - \mu) + (\mu - \hat{\mu}) + (\hat{\mu} - \tilde{\mu}) \quad \textbf{(4.5)}$$

[107] In our case of 2006, in the case of Elbers et al (2003) of 1998.
[108] In our case of 2010, in the case of Elbers et al (2003) of 2001.

The idiosyncratic error – $(W - \mu)$: the difference between the actual value and the expected value of W arises from the unobserved component of consumption and increases as the size of the target population shrinks which limits the degree of desegregation possible (Elbers, et al., 2003).

The model error – $(\mu - \hat{\mu})$: given $\hat{\zeta}_v$ are consistent estimators of ζ_v, $\hat{\mu}$ is a consistent estimator of μ and $\sqrt{s}(\mu - \hat{\mu}) \xrightarrow{d} \mathcal{N}(0, \Sigma_M)$ as $s \longrightarrow \infty$. However, given that this component of the prediction error is determined in (4.2), it does not change systematically with changes in the size of the target population (Elbers, et al., 2003).

The computation error – $(\hat{\mu} - \tilde{\mu})$: when simulation is used as a method of computation, this error has an asymptotic distribution $\sqrt{R}(\hat{\mu} - \tilde{\mu}) \xrightarrow{d} \mathcal{N}(0, \Sigma_c)$ as $R \longrightarrow \infty$. Where R is the number of independent random draws used for the simulation and therefore this error can be as small as the computational resources allow (Elbers, et al., 2003). For a detailed explanation of standard errors and population size refer to Appendix 1.

Tarozzi & Deaton (2009) argue that, in order to match survey and census data in the way which is proposed by Elbers et al. (2003), a degree of spatial homogeneity is required for which the method has no basis. They propose that estimates based on those assumptions may underestimate the variance of the error in predicting W (estimated at the local level) and therefore overstate the coverage of confidence intervals (Tarozzi & Deaton, 2009). In response, Elbers, et al. (2008) compare their small area estimate welfare results in Minas Gerais, Brasil, a notably heterogeneity area, with the true welfare values and find that the methodology yielded welfare estimations which were close to these true values and had confidence interval estimations which were appropriate. This demonstrates that if the methodology is applied with careful control over the conditional distribution of income, the estimations can be reliable (Elbers, et al., 2003).

In the 2006-2010 simulation Ecuador is divided into eight sub-regions (see Appendix 1 for comparative results). Firstly, three general geographic regions: coast, highlands and Amazon basin which are further divided into rural and urban areas excluding the two largest cities (Quito and Guayaquil) which are considered their own sub-regions. A separate consumption model for each one of these eight sub-regions was built. Therefore, a household in the rural area of a given province will have a predicted consumption resulting from the model fitted using only observations in the rural part of that province. Likewise, a household in an urban area of the same province will have a predicted consumption resulting from the model fitted using only observations from the urban part of that province. Given there can be both rural and urban areas within the same province, county or parish, separating them increases the level of homogeneity within each sub-region and within each *small area estimate* model (see Appendix 2 for detailed on

consumption models per sub-region). Once each observation (household) has a predicted consumption on the census, it is possible to estimate Gini coefficients over every province, county and parish.

For the 2014-2010 simulation I divide the country into these eight sub-regions and then further divide the data into groups of provinces within the either the urban or rural, coast, highlands, and, amazon regions which are similar to each other. I define similar as those groups where the model yielded the smallest errors (through iteration) between true (2014 LSMS) and predicted values (2014-2010 SAE). I estimate 16 different consumption models for 16 different sub-sub-regions.

In Table 4.3 the 2006-2010 simulation results are presented and compared to the Gini coefficients estimated directly from the LSMS (2006) over the sub-regions. In Table 4.4 the 2014-2010 simulations results are presented and compared to the Gini coefficients from LSMS (2014) over the provinces. In both cases the SAE estimations consistently underestimate the Gini coefficients as measured by the LSMS's. This may be due to the fact that the SAE simulation models tend to underestimate the household consumption of high income homes given that the variables used in the equation (Appendix 2) estimations are generally measuring lack of resources.

Notwithstanding, the results have a certain geographic consistency. In the case of the 2006 LSTM, Quito and Guayaquil have the highest levels of inequality followed by the rural amazon and rural highlands both in the simulated and non-simulated estimations. Table 4.4 presents the Gini coefficients in ascending order to show how, in general, the highest simulated values coincide with the highest true values.

The most important limitation of the way this method is applied is that, in both cases, the 2010 census is used. Basically a consumption model on 2006 household behavior is built and simulate it onto the 2010 census. Similarly, a consumption model on 2014 household behavior is built and simulate that onto the same 2010 census. This might be methodologically interesting as - given there is no difference in the census population - the only difference in the Gini coefficient results would be the product of the change in household behaviors between 2006 and 2014. However, this is paradoxical because, in order to use the 2006 estimated parameters (correlation coefficients) to simulate consumption using the population characteristics of 2010, it must be assumed there is very little change in behaviours between 2006 and 2010. This same assumption must be made when the 2014 parameters are used to simulate consumption on the 2010 census. However, this cannot be true as the resulting Gini coefficients are fundamentally different. Obviously, it is very improbable to have a LSMS and a census on the same year, and in the case of Ecuador there is a census only once every 10 years. Therefore, I have chosen to use the 2010 census for both cases. However, once the Ecuadorian 2020 census is built and released it would be best to repeat the method using the 2014 LSMS and the 2020 census.

Table 4.3 Comparison of estimations of the Gini coefficient from the LSMS (2006) and using SAE (2006-2010)

	Regional Gini	
Region	LSMS (2006)	SAE (2006-2010)
Quito	0.463	0.403
Guayaquil	0.416	0.386
Urban Coast	0.409	0.358
Rural Coast	0.357	0.281
Urban Highlands	0.411	0.346
Rural Highlands	0.454	0.387
Urban Amazon	0.416	0.355
Rural Amazon	0.47	0.454
National Total	0.466	0.419

Source: Small Area Estimates using Living Standards Measurement Survey, Ecuadro 2006 (Encuesta de Condiciones de Vida, 2006) and Ecuadorian Census of 2010. Instituto Nacional de Encuestas y Censos, Ecuador. Data procesing: Unidad de Infromación Socio Ambiental, Universidad Andina Simón Bolívar.

Table 4.4 Comparing provincial Gini coefficients from LSTM (2014) to SAE (2014-2010)

	Gini province	
Province ID	LSMS (2014)	SAE (2014-2010)
20	0.319	0.308
7	0.357	0.291
12	0.363	0.28
9	0.365	0.36
3	0.368	0.327
8	0.376	0.376
23	0.376	0.303
13	0.377	0.301
2	0.379	0.365
21	0.382	0.385
24	0.382	0.305
4	0.384	0.339
5	0.385	0.344
11	0.399	0.363
1	0.401	0.388
19	0.402	0.362
18	0.404	0.355
6	0.411	0.404
17	0.429	0.418
10	0.43	0.363
14	0.446	0.397
22	0.446	0.425
16	0.473	0.368
15	0.495	0.381

Authros computation using 2014 LSMS and 2010 Ecuadorian Census.

4.3.4 Other regressors

The control variables include what the previous literature on the social determinants of malnutrition has shown influential (Larrea, et al., 2001; Larrea, 2002; Marins & Almeida, 2002; Willey, et al., 2009; Aerts, et al., 2004; El Taguri, et al., 2009; Adair & David, 1997). These variables are measured either at the individual level or are aggregated at a geographic level and can be grouped into 4 categories: the characteristics of child, the characteristics of the parents, the characteristics of the household, and, the characteristics of region. This last one is defined at various levels of aggregation depending on the nature of the information that is available.

There are various regressors which only exist in the 2006 model (daycare, maternal fertility, underemployment, and nutritional supplements, birth with c-section, access to ObGyn, index of food consumption, number of M.D.s/10000 ppl/, and mean distance to highway) because these variables either had no significant effect in 2014 or did not exist in the 2014 survey. There are many regressors which only exist in the 2014 model (parental BMI and height, a specification for Galapagos in geographic region) because they did not exist in the 2006 survey. In order to demonstrate that the inclusion or exclusion of certain regressors has no impact on the final results I present two specifications for each year in every type of model (OLS and IV): the first with the best fit for each year, and the second with the variables which exist in both surveys.

In the first category, various individual level characteristics which may affect the z-score of the child are controlled for: a dummy variable which discerns if the child was born with a LBW,[109] the gender and age of the child (in months), dummy nutritional supplements, dummy diarrhea, dummy access to public daycare, the number of months of breastfeeding, and the proportion of required vaccines by age.[110]

Various characteristics of the pre-natal, natal and post-natal health services the mother has access to. the age and years of education of the mother is included, as well as the fertility of the mother. [111] A dummy variable discerning whether she had a caesarian section and if there was a physician or obstetrician present while giving birth. A dummy of maternal under-employment is also included,[112] and, in the 2014 model the maternal and paternal BMI and height are included to control for genetic traits which might affect the growth patterns of children.

The characteristics of the household include variables which may affect the child during the pregnancy or after birth. The per capita household consumption in log form is included so as to capture the concave effect that income has on health. A dummy cash transfers, the square root of the number of children under the age of twelve living in the household, an index of household living conditions obtained using principal components analysis (see Appendix 4 for detailed results),[113] and, the years of work experience of the head of the household are included as well.

[109] LBW is typically defined as children weighing less than 2500g. In some cases, the mothers did not provide the exact weight of the child at birth; however, the survey does ask whenever the mother was told by her doctor or practitioner that the child was "underweight."
[110] BCG for tuberculosis, Pentavalente which is DTP for diphtheria, tetanus and pertussis, Hb for hepatitis B and HIB for type b Haemophilus influenzae, poliomyelitis, and finally, measles.
[111] The number of live births a woman has had is divided by her age since adolescence.
[112] Underemployed is defined as a person who falls into one of any three categories. Firstly, an employed person who works less than 40 hours a week and is willing to work more hours, secondly, an employed person who makes less than $156.21 per month and works more than 40 hours a week, and finally, an employed person who works less than 40 hours a week however makes less than $156.21/h of work.
[113] This methodology allows us to avoid using housing variables which may be highly correlated in our regression model in order to circumvent high levels of multicollinearity (see Appendix 4 for detailed results).

Finally, the contextual variables, apart from our variable of interest which has been explained above, firstly, the mean size and rent of agricultural land in the parish, the rate of attendance to secondary education in the census sector,[114] dummy rural, the natural log of provincial GDP,[115] and the head count for poverty in the geographic area of interest are included.[116] The last variable is included because of the effect that poverty may have on neighborhood environments or on the social fabric of subpopulations. This is an aggregated variable therefore it does not measure the same phenomenon as the household per capita consumption. Additionally, the mean consumption of the specified geographical area[117] is included given there is some evidence that having wealthier neighbors may create positive externalities in access to healthcare and individual health (Miller & Paxson, 2006), the number of M.D.'s per every 10 000 people and the mean distance of agricultural plots in the parish to a main highway - which is taken from the 2001 agricultural census.

Finally, a food consumption index which identifies the average carbohydrates, fat and protein consumed in each parish is built and added (Programa Alimentate Ecuador, Ministerio de Inclusión Económica y Social, Ecuador, 2009). The index gives high scores to parishes with high carbohydrate consumption, indicating a severe protein deficiency and a lack of micronutrient intake from fruits[118] (see Appendix 5 for details) (Larrea & Kawachi, 2005). This index gives us an idea of the social and institutional barriers present in the access to proper nourishment and food security. This index is rendered insignificant in the IV model and therefore, I decided to not include it in the 2014 models as the BMI of the parents is included and it indicates the health patterns of the household level. We consider the household level more accurate than the parish level.

4.3.5 Fixed effects

Fixed effects to control for the effect of ethnicity are used. Given this is a cross-section analysis, fixed effects amount to a dummy variable for each category of the following three categories: mestizo[119,] indigenous[120] and afro-Ecuadorian (Larrea & Kawachi, 2005). I also include fixed effect for larger regions (highlands, coast, amazon, Galapagos). In parallel I include a dummy variable for the amazon region as it is considerably different historically and economically to the rest of the country. It is the home of various

[114] It is assumed living in a more educated community may have positive externalities on the health and child care behaviours of individuals.
[115] In order to capture the effect of aggregate production as this may increase the access to specific health and welfare services.
[116] Be it province, county or parish.
[117] Province, county or parish, depending on the model.
[118] Essentially a potato based diet
[119] Defined as a mix between Caucasian and Indigenous.
[120] Indigenous people are usually fluent in one of various indigenous languages as well as Spanish, whereas it is rare for non-indigenous peoples to be fluent in such languages. Therefore, it may be suggested that the fluency in these indigenous languages be used as a proxy for ethnicity. Specifically, an individual is found to be indigenous if she or he either states it directly, or if she or he states that the spoken language in the household is an indigenous one. This is done in order to avoid an underestimation of the indigenous population.

relatively isolated first nations or indigenous groups such as the Waorani (Finer, et al., 2009). It is where extensive oil extraction activities and important environmental impacts of these activities take place (Finer, et al., 2009). The particular lifestyles of these groups along with the oil extraction activities may have an impact on human health which it is, otherwise, difficult to control for.

4.4 Methodology

OLS models (Table 4.5) and IV models (Table 4.6) are presented along with the first-stage results of the IV models (Table 4.6). There are two separate data sources, as mentioned above, LSMS 2006 and 2014. I have put both years in the same results Tables (4.5, 4.6, 4.7) in order to demonstrate that the same specifications in terms of regressors have been implemented. An additional specification for each year is appended at the beginning (2006 models) and end (2014 models) of each Table (4.5, 4.6, 4.7).

4.4.1 Ordinary least squares model

The OLS model is included as a baseline. The formal model is presented below. In (4.6) y_{ij} is the z-score for each individual child i in each province/county/parish j, $Gini_j$ is the Gini coefficient for each province/county/parish j, $X\beta$ is a vector of control variables, and, e_{ij} is a vector of residuals.

$$y_{ij} = \beta_0 + \beta_1 Gini_j + X\beta + e_{ij} \quad \textbf{(4.6)}$$

$$e_{ij} \sim N(0, \sigma^2) \quad \textbf{(4.7)}$$

4.4.2 Instrumental variable model

In the 2006 OLS model there is omitted variable bias given it is not possible to control for the height of the parents. That is to say, if the height of the parents has an effect on the growth patterns of the children, and the height of the parents is excluded from the model, then, the effect will be absorbed by the error term. This produces a bias in all the correlation coefficients of the model, including the beta of the effect of inequality on the z-score of height for age as it, as well as all other beta coefficients, are equal to the true beta coefficient plus the effect of the unobservable omitted variable in the following way:

$$y_i = \beta_0 + \beta_1 (gini_j) + X\beta + u_i \quad \textbf{(4.8)}$$

$$u_i = e_i + \gamma_i \quad \textbf{(4.9)}$$

Where e_i is the error and γ_i is the effect of the unobservable. If this is true then our beta will be as follows:

$$\hat{\beta}_{\text{OLS}} = \beta + (X'X)^{-1}X'u = \beta + (X'X)^{-1}X(e_i + \gamma_i) \quad \textbf{(4.10)}$$

If it is assumed that the effect of the unobservable variables are positive(negative), then $\hat{\beta}_{\text{OLS}}$ would over(under)-estimate the effect of the Gini coefficient. In the case of the 2006 models, it can be safely assumed that the height of the parents has a positive correlation with the z-score of height for age. This would mean taller parents have taller children and therefore children with a higher z-score on average. This is the case in the 2014 OLS model (Table 4.5) and in the 2014 IV model (Table 4.6) where the height and BMI of both parents are included. In every model both maternal BMI and paternal height and BMI have a positive significant effect on the outcome variable.

Therefore, omitting the height and BMI of the parents implies this positive effect is absorbed by the error term and increases the $\hat{\beta}_{\text{OLS}}$ of the Gini coefficient. Given the $\hat{\beta}_{\text{OLS}}$ of the Gini coefficient is negative, omitting these variables would augment it making it less negative. This means that the $\hat{\beta}_{\text{OLS}}$of the Gini coefficient should be larger in the 2006 OLS model (Table 4.5: province: -1.8 / -0.9, county: -1.3 / -0.7, parish: -0.8 / -0.2) than in the 2006 IV model (Table 4.6: province: -11.4 / -15.6, county: -9.9 / -19.6, parish: -21.9 / -30.6) which seems to be the case.

In the 2014 model I believe a similar effect is occurring. For example, in the 2006 model, the mean distance (of all plots of land in a parish) to the main highway was initially included - which was taken from the 2000 agricultural census. This variable is included despite there being 5 years between 2000 and 2006, because it was possible to assume the highway system remained relatively unchanged immediately after the financial crisis of 1999. However, it is not possible to include this variable in the 2014 model, as there had not been another agricultural census since 2000 and the variable does not exist in the other accessible datasets. Additionally, it is no longer possible to argue that the highway system has remained unchanged over a 14 year period, especially between 2007 and 2014 given, as mentioned above, there were considerable changes in the social investment policy (Instituto Nacional de Estadistica y Censos, 2015).[121] Notwithstanding, the effect of the distance to the highway was positive and significant in both the 2006 OLS and 2006 IV models. This indicates that the isolation of the parish in which the child lives has a deleterious effect on the z-score of the child. Therefore, when this measure of isolation is omitted from the 2006 and 2014 models the effect is absorbed by the error term and is added to all the beta coefficients in the model including the beta of the Gini coefficients. If this is true, then the 2014 $\hat{\beta}_{\text{OLS}}$ for the Gini (Table

[121] Between 2007 and 2015 the incidence of poverty decreased from 37% to 23%; the incidence of extreme poverty from 16% to 8% and the Gini coefficient from 0.55 to 0.47.

4.5: province: -1.9 / -1.28, county: -1.3 / -0.38, parish: -0.4 / 0.096) should and are larger than the 2014 $\hat{\beta}_{\mathrm{IV}}$ for the Gini (Table 4.6: province: -1.5 / -6.8, county: -1.2 / -4.6, parish: -1.6 / - 7.1).

Therefore, our models suffer from omitted variable bias. I attempt to tackle this problem using an IV model. The purpose of the model is to clean the endogeneity exclusively in the beta of the Gini coefficient. Therefore, a variable which is highly correlated specifically with the Gini coefficient must be found. I propose the proportion of households who have suffered from draught as an instrument. If this instrument is exogenous to the error term of the model i.e. has no influence on the outcome, then the endogeneity the omitted variables have on the beta of the Gini coefficient can be cleaned out. The same instrument in both years was attempted, however, the question is not formulated with the same wording. In 2014 the question inquires whether the household suffered from any natural disaster, including draught but also flooding, storms, etc., while in 2006 the question inquired specifically about draught. This is one of the important reasons why it is not possible to pool the data.

We argue both iterations of the 'natural disaster' instrument is a strong exogenous instrument given it complies with both parts of the exclusion restriction which I outline here:

(1) The instrument must be correlated with the endogenous explanatory variable. In 2006, in the first stage regressions of our models (Table 4.7) the t-values of the instrument are high except in the parish model (province: 11.4, county: 9.8, parish: 4.9 / province: 5.4, county: 4.06, parish: 2.5). The F-statistic of the instrument in the models are high except in the second specification of the parish model (province: 130, county: 97, parish: 24 / province: 27.4, county: 16.4, parish: 6.6). In 2014, the t-value of the instrument (Table 4.7) are also high (province: -12.7, county: -8.1, parish: -4.9 / province: -10.9, county: -7.7, parish: -4.15). The F-statistics of the instrument are also high (province: 161.4, county: 67, parish: 24 / province: 119, county: 60, parish: 17.4).

(2) The instrument cannot be correlated with the error term in the explanatory equation, conditional on other covariates. There is no way of measuring this directly; however, I argue that a draught and other natural disasters are unexpected exogenous meteorological phenomenon.

The formal model is presented in (4.11) and (4.12) where y_{ij} is the z-score for each individual child i in each province/county/parish j, Gini_j is the Gini coefficient of each province/county/parish j, and Daught_j is the proportion of household who had suffered a draught in province.

$$y_{ij} = \beta_0 + \beta_1 \text{Gini}_j + X\beta + + e_{ij} \quad \textbf{(4.11)}$$

$$\text{Gini}_j = \alpha_0 + \alpha_1 \text{Daught}_j + v_j \quad \textbf{(4.12)}$$

$$\text{cov}\left(\text{Gini}_j, \text{Daught}_j\right) \neq 0 \quad \textbf{(4.13)}$$

$$\text{cov}\left(\text{Daught}_j, e_{ij}\right) \neq 0 \quad \textbf{(4.14)}$$

4.5 Main Findings

We report the results in three tables: Table 4.5 has the OLS results for both years, Table 4.6 has the IV results for both years, and Table 4.7 presents the first stage IV model results for both years. I discuss the findings below.

Table 4.5 OLS models

	2006 full model			2006 model (same regressors as 2014)			2014 model (same regressors as 2006)			2014 full model		
	OLS1	OLS2	OLS3	OLS4	OLS5	OLS6	OLS7	OLS8	OLS9	OLS10	OLS11	OLS12
	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)
Gini province	**-1.808*** **			**-0.924**			**-1.989**			**-2.366**		
	(0.667)			**(0.639)**			**(1.656)**			**(1.797)**		
Gini county		**-1.308****			**-0.785**			**-1.335**			**-0.499**	
		(0.619)			**(0.655)**			**(0.864)**			**(0.938)**	
Gini parish			**-0.848**			**-0.219**			**-0.298**			**0.0640**
			(0.604)			**(0.632)**			**(0.729)**			**(0.769)**
D. LBW	-0.598***	-0.594***	-0.598***	-0.627***	-0.626***	-0.630***	-0.485***	-0.480***	-0.483***	-0.409***	-0.406***	-0.407***
	(0.102)	(0.102)	(0.101)	(0.108)	(0.108)	(0.108)	(0.097)	(0.098)	(0.097)	(0.110)	(0.110)	(0.110)
D. Female	0.148***	0.146***	0.145***	0.167***	0.165***	0.165***	0.176***	0.176***	0.178***	0.181***	0.182***	0.184***
	(0.033)	(0.033)	(0.033)	(0.036)	(0.036)	(0.036)	(0.059)	(0.059)	(0.059)	(0.063)	(0.063)	(0.063)
Age in months	-0.136***	-0.136***	-0.136***	-0.0683***	-0.0682***	-0.0679***	-0.0764***	-0.0767***	-0.0768***	-0.0769***	-0.0772***	-0.0772***
	(0.016)	(0.016)	(0.016)	(0.006)	(0.006)	(0.006)	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)	(0.010)
Age in months^2	0.003***	0.00351***	0.003***	0.00***	0.00***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***	0.00103***
	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Age in months^3	-0.00***	-0.00***	-0.00***									
	(0.000)	(0.000)	(0.000)									
Vaccines	0.145	0.141	0.140	0.0553	0.0537	0.0522	0.371	0.355	0.369	0.419	0.426	0.433
	(0.104)	(0.104)	(0.104)	(0.100)	(0.100)	(0.100)	(0.290)	(0.293)	(0.292)	(0.333)	(0.337)	(0.334)
D. N. Supplement	-0.0190	-0.0178	-0.0227	-0.0712	-0.0706	-0.0702	-0.0902	-0.0976	-0.0903	-0.0769	-0.0800	-0.0761
	(0.047)	(0.047)	(0.047)	(0.049)	(0.049)	(0.049)	(0.061)	(0.062)	(0.062)	(0.061)	(0.061)	(0.061)
D. Diarrhea	-0.0361	-0.0357	-0.0351	-0.101**	-0.101**	-0.101**	-0.117	-0.117	-0.117	-0.0831	-0.0873	-0.0864
	(0.040)	(0.040)	(0.040)	(0.042)	(0.042)	(0.042)	(0.086)	(0.086)	(0.086)	(0.082)	(0.082)	(0.082)
D. Daycare	-0.0670	-0.0557	-0.0598									
	(0.047)	(0.047)	(0.047)									
N. month breastfed	-0.00456	-0.00463	-0.00428	0.000951	0.000810	0.000469	-0.0132	-0.0137	-0.0143	-0.0251**	-0.0259**	-0.0262**
	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)	(0.012)
Age mother	0.00983***	0.00982***	0.00991***	0.008***	0.00847***	0.00845***	0.00145	0.00154	0.00156	-0.000890	-0.000925	-0.000995
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.007)	(0.007)	(0.007)
Schooling mother	0.0259***	0.0258***	0.0262***	0.03***	0.0348***	0.0345***	0.0168**	0.0166*	0.0167*	0.0269***	0.0270***	0.0272***
	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.006)	(0.009)	(0.009)	(0.009)	(0.009)	(0.010)	(0.009)
Fertility mother	-0.920***	-0.930***	-0.928***									
	(0.315)	(0.316)	(0.315)									
D. C-section	0.105***	0.113***	0.113***									
	(0.039)	(0.039)	(0.039)									
D. ObGyn	0.0872*	0.0738	0.0835									
	(0.053)	(0.053)	(0.054)									
D. Mother underemployed	-0.0642*	-0.0721**	-0.0746**									
	(0.035)	(0.035)	(0.035)									
D. N. Supplement mother	-0.0712*	-0.0730*	-0.0675									
	(0.042)	(0.042)	(0.042)									
Height mother										0.0488***	0.0486***	0.0487***
										(0.006)	(0.006)	(0.006)
BMI mother										0.00142	0.000905	0.000969
										(0.007)	(0.007)	(0.007)
Height father										0.0352***	0.0356***	0.0355***
										(0.004)	(0.004)	(0.004)
BMI father										0.0279***	0.0281***	0.0282***
										(0.008)	(0.008)	(0.008)
Ln hh consumption per capita	0.164***	0.164***	0.166***	0.187***	0.189***	0.193***	0.388***	0.390***	0.388***	0.174**	0.178**	0.177**
	(0.039)	(0.039)	(0.039)	(0.039)	(0.040)	(0.040)	(0.066)	(0.067)	(0.066)	(0.076)	(0.077)	(0.077)
D. welfare	-0.0606	-0.0568	-0.0604	-0.0805*	-0.0798*	-0.0797*	-0.00887	-0.00724	-0.0121	0.0346	0.0326	0.0309
	(0.041)	(0.041)	(0.041)	(0.043)	(0.043)	(0.043)	(0.076)	(0.077)	(0.077)	(0.070)	(0.070)	(0.070)
sqrt n children<12 in hh	-0.151***	-0.154***	-0.156***	-0.253***	-0.252***	-0.251***	-0.0761	-0.0794	-0.0852	-0.0157	-0.0167	-0.0199
	(0.056)	(0.056)	(0.056)	(0.049)	(0.049)	(0.049)	(0.095)	(0.096)	(0.095)	(0.109)	(0.110)	(0.109)
Index living conditions	0.0652**	0.0513	0.0494	0.100***	0.0985***	0.0961***	0.0705**	0.0755***	0.0713**	0.0657**	0.0658**	0.0637**
	(0.031)	(0.031)	(0.031)	(0.033)	(0.033)	(0.033)	(0.028)	(0.028)	(0.028)	(0.029)	(0.029)	(0.029)

Work experience head hh	0.00271**	0.00284**	0.00279**	0.00515***	0.00522***	0.00526***	0.00180	0.00160	0.00177	0.00388	0.00394	0.00400
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Mean size agri. Land	0.00484**	0.00454**	0.00343*	0.00345	0.00309	0.00231	0.000488	0.000566	0.000700	0.000323	0.000557	0.000633
	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Mean rent/ha. ag. Land	0.000727*	0.000790*	0.000829**	0.0000558	0.0000338	0.0000301	-0.000155	-0.000160	-0.000108	-0.000111	-0.0000692	-0.0000436
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Rate attendence secondary school	0.287***	0.319***	0.314***	0.277**	0.284***	0.278**	0.201	0.212	0.204	0.181	0.179	0.175
	(0.098)	(0.098)	(0.098)	(0.108)	(0.108)	(0.108)	(0.132)	(0.133)	(0.133)	(0.148)	(0.149)	(0.149)
D. rural	-0.309***	-0.126*	-0.113	-0.185*	-0.157*	-0.114	0.0745	0.0621	0.0713	0.122*	0.118	0.129
	(0.084)	(0.075)	(0.070)	(0.094)	(0.092)	(0.090)	(0.068)	(0.070)	(0.073)	(0.073)	(0.074)	(0.079)
Ln(GDP prov)	0.112***	0.0992***	0.0961***	0.0942***	0.0907***	0.0926***	0.213***	0.195***	0.182***	0.153***	0.124**	0.117**
	(0.016)	(0.016)	(0.016)	(0.020)	(0.020)	(0.020)	(0.052)	(0.046)	(0.046)	(0.055)	(0.049)	(0.048)
Poverty prov	-0.0990			1.431**	1.344**	1.287**	-0.154	-0.360	-0.538	-0.0897	-0.567	-0.653*
	(0.517)			(0.559)	(0.544)	(0.541)	(0.416)	(0.340)	(0.330)	(0.457)	(0.376)	(0.366)
Mean consumption prov.	-0.0055**			0.003	0.0031	0.003	-0.0058***	-0.006***	-0.0065***	-0.004**	-0.005***	-0.005***
	(0.002)			(0.002)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.001)
Poverty county		-0.941**										
		(0.390)										
Mean consumption count.		-0.00689***										
		(0.002)										
Poverty parish			-0.730**									
			(0.342)									
Mean consumption parish			-0.006***									
			(0.002)									
Index food consumption	-0.0577***	-0.0697***	-0.08***									
	(0.015)	(0.014)	(0.014)									
N. MD/10000 ppl.	0.000626	0.000797	0.000895									
	(0.001)	(0.001)	(0.001)									
Mean distance highway	0.203***	0.217***	0.219***									
	(0.038)	(0.038)	(0.038)									
Mestizo.ethnic	.	.	.	.	.	.	.	.	.	.	.	.
	.	.	.	.	.	.	.	.	.	.	.	.
Indigena.ethnic	-0.153**	-0.150**	-0.154**	-0.230***	-0.234***	-0.244***	-0.220**	-0.223**	-0.223**	-0.0126	-0.0154	-0.0161
	(0.060)	(0.061)	(0.062)	(0.061)	(0.061)	(0.061)	(0.110)	(0.111)	(0.110)	(0.120)	(0.120)	(0.120)
Afro.ethnic	0.217***	0.235***	0.230***	0.234***	0.239***	0.239***	0.0966	0.0980	0.0790	-0.139	-0.157	-0.171
	(0.077)	(0.077)	(0.078)	(0.079)	(0.079)	(0.079)	(0.133)	(0.132)	(0.132)	(0.130)	(0.132)	(0.132)
D. Rural Amazon	0.476***	0.456***	0.394***									
	(0.115)	(0.110)	(0.109)									
Highlands.region				.	.	.	.	.	.	.	.	.
				.	.	.	.	.	.	.	.	.
Coast.region				0.478***	0.490***	0.520***	0.183*	0.208**	0.251***	-0.0240	0.0311	0.0513
				(0.064)	(0.062)	(0.060)	(0.109)	(0.099)	(0.095)	(0.121)	(0.110)	(0.106)
Amazon.region				0.456***	0.453***	0.435***	0.446***	0.417***	0.366***	0.441***	0.342***	0.313**
				(0.082)	(0.083)	(0.086)	(0.119)	(0.110)	(0.109)	(0.132)	(0.122)	(0.122)
Galapagos.region							0.818***	0.839***	0.884***	0.577*	0.664**	0.684**
							(0.283)	(0.281)	(0.279)	(0.299)	(0.297)	(0.295)
_cons	-2.312***	-1.840***	-2.178***	-4.182***	-4.148***	-4.455***	-3.648***	-3.469***	-3.551***	-16.15***	-16.10***	-16.17***
	(0.603)	(0.552)	(0.551)	(0.745)	(0.777)	(0.776)	(0.738)	(0.758)	(0.767)	(1.286)	(1.308)	(1.302)
R-sq	0.2814	0.2804	0.2801	0.2819	0.2818	0.2815	0.2167	0.2178	0.2168	0.3132	0.3137	0.3136
N	5038	5038	5038	4475	4475	4475	2559	2548	2548	1917	1908	1908

Source: Author's computation using 2006 /2014 LSMS

Table 4.6 IV models

	2006 full model			2006 model (same regressors as 2014)			2014 model (same regressors as 2006)			2014 full model		
	IV1	IV2	IV3	IV4	IV5	IV6	IV7	IV8	IV9	IV10	IV11	IV12
	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)
Gini province	**-11.46*** **			**-15.60** **			**-1.451**			**-6.230**		
	(3.673)			**(7.305)**			**(5.791)**			**(6.143)**		
Gini county		**-9.903** **			**-19.67** **			**-1.105**			**-4.331**	
		(3.976)			**(9.680)**			**(4.661)**			**(4.618)**	
Gini parish			**-21.98** **			**-30.69***			**-1.507**			**-6.602**
			(8.707)			**(17.678)**			**(6.367)**			**(7.204)**
D. LBW	-0.555***	-0.546***	-0.503***	-0.480***	-0.433***	-0.407**	-0.385***	-0.381***	-0.380***	-0.336***	-0.318***	-0.306***
	(0.093)	(0.096)	(0.112)	(0.112)	(0.132)	(0.164)	(0.078)	(0.077)	(0.077)	(0.083)	(0.082)	(0.085)
D. Female	0.155***	0.148***	0.150***	0.150***	0.133***	0.141***	0.0905*	0.0918*	0.0941*	0.0859*	0.0840	0.0937*
	(0.029)	(0.029)	(0.033)	(0.032)	(0.036)	(0.041)	(0.048)	(0.048)	(0.048)	(0.052)	(0.052)	(0.053)
Age in months	-0.158***	-0.156***	-0.172***	-0.069***	-0.068***	-0.068***	-0.09***	-0.09***	-0.09***	-0.09***	-0.09***	-0.09***
	(0.013)	(0.013)	(0.017)	(0.006)	(0.006)	(0.007)	(0.006)	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)
Age in months^2	0.004***	0.004***	0.004***	0.00***	0.00***	0.00***	0.001***	0.001***	0.001***	0.001***	0.001***	0.001***
	(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Age in months^3	-0.00***	-0.00***	-0.00***									
	(0.000)	(0.000)	(0.000)									
Vaccines	0.228***	0.224**	0.273**	0.138	0.128	0.115	0.100	0.0860	0.0892	0.142	0.107	0.141
	(0.088)	(0.089)	(0.106)	(0.108)	(0.110)	(0.125)	(0.189)	(0.201)	(0.197)	(0.199)	(0.209)	(0.206)
D. N. Supplement	-0.0383	-0.0295	-0.0362	-0.0745*	-0.0701	-0.0725	-0.0627	-0.0670	-0.0669	-0.0263	-0.0425	-0.0439
	(0.042)	(0.042)	(0.047)	(0.043)	(0.045)	(0.053)	(0.052)	(0.056)	(0.056)	(0.056)	(0.061)	(0.063)
D. Diarrhea	-0.0310	-0.0329	0.00184	-0.0645*	-0.0618	-0.0352	-0.152**	-0.152**	-0.152**	-0.123*	-0.127*	-0.116*
	(0.035)	(0.035)	(0.043)	(0.038)	(0.041)	(0.056)	(0.065)	(0.065)	(0.065)	(0.069)	(0.069)	(0.070)
D. Daycare	-0.0432	-0.0179	-0.0646									
	(0.043)	(0.043)	(0.050)									
N. month breastfed	-0.00292	-0.00348	-0.00122	-0.00664	-0.00764	-0.00383	-0.00911	-0.00959	-0.00963	-0.0200*	-0.0208*	-0.0209*
	(0.006)	(0.006)	(0.007)	(0.006)	(0.007)	(0.008)	(0.010)	(0.010)	(0.010)	(0.011)	(0.011)	(0.011)
Age mother	0.009***	0.009***	0.01***	0.01***	0.01***	0.01***	0.001	0.001	0.001	-0.00	-0.00	-0.00
	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.005)	(0.003)	(0.003)	(0.003)	(0.005)	(0.005)	(0.006)
Schooling mother	0.0316***	0.0323***	0.0394***	0.0453***	0.0500***	0.0593***	0.00903	0.00903	0.00909	0.0200**	0.0192**	0.0191**
	(0.005)	(0.005)	(0.007)	(0.007)	(0.009)	(0.016)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.009)
Fertility mother	-0.933***	-0.907***	-1.037***									
	(0.284)	(0.285)	(0.334)									
D. C-section	0.0864**	0.101***	0.127***									
	(0.036)	(0.036)	(0.042)									
D. ObGyn	0.105**	0.0804*	0.0800									
	(0.045)	(0.047)	(0.053)									
D. Mother underemployed	-0.0437	-0.0703**	-0.0779**									
	(0.034)	(0.032)	(0.035)									
D. N. Supplement mother	-0.0635*	-0.0751**	-0.0500									
	(0.037)	(0.037)	(0.043)									
Height mother										0.0501***	0.0490***	0.0493***
										(0.005)	(0.005)	(0.005)
BMI mother										0.00136	0.000509	-0.00120
										(0.006)	(0.006)	(0.006)
Height father										0.0406***	0.0410***	0.0420***
										(0.004)	(0.004)	(0.004)
BMI father										0.0299***	0.0294***	0.0310***
										(0.007)	(0.007)	(0.007)
Ln hh consumption per capita	0.110***	0.112***	0.00651	0.0453	0.00812	-0.124	0.364***	0.365***	0.367***	0.113*	0.127**	0.127*
	(0.037)	(0.038)	(0.071)	(0.073)	(0.094)	(0.182)	(0.058)	(0.058)	(0.059)	(0.064)	(0.064)	(0.065)
D. welfare	-0.0465	-0.0359	-0.0666	-0.0664*	-0.0441	-0.114**	-0.116**	-0.116**	-0.122**	-0.0163	-0.0103	-0.0455
	(0.036)	(0.037)	(0.041)	(0.038)	(0.039)	(0.058)	(0.058)	(0.059)	(0.060)	(0.063)	(0.064)	(0.070)
sqrt n children<12 in hh	-0.152***	-0.152***	-0.166***	-0.224***	-0.207***	-0.228***	-0.104	-0.112	-0.107	-0.120	-0.124	-0.103

	(0.052)	(0.052)	(0.058)	(0.044)	(0.045)	(0.054)	(0.081)	(0.079)	(0.084)	(0.088)	(0.088)	(0.095)
Index living conditions	0.146***	0.134***	0.207***	0.219***	0.254***	0.343**	0.0498**	0.0520**	0.0529**	0.0528**	0.0603**	0.0673**
	(0.036)	(0.038)	(0.064)	(0.056)	(0.075)	(0.136)	(0.022)	(0.025)	(0.027)	(0.024)	(0.026)	(0.031)
Work experience head hh	0.00245**	0.00309***	0.00279**	0.00331**	0.00400***	0.00410***	0.00139	0.00125	0.00131	0.00414	0.00392	0.00474*
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)
Mean size agri. Land	0.00952***	0.00916***	0.0144***	0.0134*	0.0175*	0.0235	0.000676	0.000777	0.000697	-0.000413	0.0000352	-0.000409
	(0.003)	(0.003)	(0.005)	(0.007)	(0.010)	(0.015)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Mean rent/ha. ag. Land	0.000364	0.000646*	0.00100**	0.00132	0.00190*	0.00272	-0.000494	-0.000491	-0.000497	-0.000641	-0.000611	-0.000678
	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)	(0.002)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Rate attendence secondary school	0.177**	0.249***	0.148	0.408***	0.498***	0.619***	0.217*	0.228	0.237	0.153	0.189	0.235
	(0.088)	(0.087)	(0.105)	(0.103)	(0.136)	(0.215)	(0.122)	(0.142)	(0.164)	(0.130)	(0.148)	(0.181)
D. rural	-0.805***	-0.453***	-0.821***	-1.230**	-1.306**	-1.962*	0.0594	0.0520	0.0196	0.0385	-0.00392	-0.151
	(0.215)	(0.149)	(0.300)	(0.544)	(0.609)	(1.090)	(0.063)	(0.077)	(0.193)	(0.068)	(0.083)	(0.220)
Ln(GDP prov)	0.0957***	0.0791***	0.0659***	0.136***	0.141***	0.162***	0.164*	0.153***	0.147***	0.196*	0.149**	0.127***
	(0.016)	(0.014)	(0.016)	(0.038)	(0.043)	(0.061)	(0.095)	(0.055)	(0.040)	(0.103)	(0.062)	(0.047)
Poverty prov	1.764**			5.507**	6.084**	8.436*	-0.103	-0.206	-0.216	0.678	0.141	0.161
	(0.743)			(2.240)	(2.633)	(4.422)	(0.946)	(0.627)	(0.590)	(1.015)	(0.620)	(0.654)
Mean consumption prov.	-0.00142			0.0087**	0.011**	0.018*	-0.004**	-0.005***	-0.004***	-0.001	-0.003*	-0.002
	(0.002)			(0.004)	(0.005)	(0.010)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Poverty county		0.127										
		(0.491)										
Mean consumption count.		-0.00431**										
		(0.002)										
Poverty parish			0.965									
			(0.746)									
Mean consumption parish			0.000983									
			(0.003)									
Index food consumption	0.0220	-0.00250	0.0506									
	(0.035)	(0.033)	(0.054)									
N. MD/10000 ppl.	-0.000199	0.000273	0.000211									
	(0.001)	(0.001)	(0.001)									
Mean distance highway	0.194***	0.231***	0.360***									
	(0.035)	(0.036)	(0.067)									
Mestizo.ethnic												
Indigena.ethnic	-0.0254	-0.0658	0.115	0.0123	0.0658	0.270	-0.266***	-0.264***	-0.259***	-0.0177	-0.00997	0.00570
	(0.074)	(0.070)	(0.131)	(0.135)	(0.167)	(0.309)	(0.081)	(0.082)	(0.087)	(0.087)	(0.088)	(0.093)
Afro.ethnic	0.204***	0.276***	0.310***	0.160*	0.233***	0.261***	0.191	0.194	0.206	-0.0120	0.00725	0.0841
	(0.064)	(0.061)	(0.070)	(0.086)	(0.071)	(0.080)	(0.140)	(0.148)	(0.186)	(0.163)	(0.179)	(0.246)
D. Rural Amazon	1.482***	1.332***	2.703***									
	(0.421)	(0.453)	(0.986)									
Highlands.region				-1.255***	-1.464***	-2.362**	-0.537*	-0.550*	-0.539	-0.156	-0.236	-0.174
				(0.386)	(0.507)	(1.108)	(0.325)	(0.315)	(0.345)	(0.337)	(0.311)	(0.358)
Coast.region				-1.289**	-1.571*	-2.505*	-0.388	-0.394	-0.366	-0.375	-0.413	-0.304
				(0.648)	(0.819)	(1.495)	(0.281)	(0.283)	(0.313)	(0.292)	(0.295)	(0.322)
Amazon.region							-0.199	-0.229	-0.203	0.327	0.161	0.300
							(0.505)	(0.454)	(0.549)	(0.531)	(0.450)	(0.584)
Galapagos.region												
_cons	1.138	1.547	5.691*	0.444	1.492	4.742	-1.957***	-1.802**	-1.627	-15.86***	-15.26***	-14.63***
	(1.227)	(1.422)	(3.035)	(1.730)	(2.314)	(4.550)	(0.595)	(0.842)	(1.466)	(1.052)	(1.304)	(1.826)
R-sq	0.2540	0.2572	0.0589	0.1660	0.0790	-0.2623	0.2265	0.2285	0.2260	0.3259	0.3258	0.2938
N	5038	5038	5038	5281	5281	5281	2605	2594	2594	1947	1938	1938

Source: Author's computation using 2006 /2014 LSMS

Table 4.7 First stage IV models

	2006 full model			2006 regressors equal to 2014			2014 regressors equal to 2006			2014 full model		
	FSGiniProv	FSGiniCount	FSGiniParr	FSGiniProv	FSGiniCount	FSGiniParr	FSGiniProv	FSGiniCount	FSGiniParr	FSGiniProv	FSGiniCount	FSGiniParr
	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)	b/(se)
D. LBW	0.00664***	0.00833***	0.00553**	0.00859***	0.00923***	0.00675***	-0.00202*	0.00166	0.00141	-0.00184	0.00212	0.00312
	(3.10)	(3.65)	(2.35)	(3.54)	(3.71)	(2.69)	(-1.94)	(0.83)	(0.57)	(-1.53)	(0.92)	(1.11)
D. Female	-0.00	-0.00	-0.00	-0.001	-0.001**	-0.001	-0.00	-0.00	0.001	-0.001	-0.001	0.000485
	(-0.08)	(-0.99)	(-0.37)	(-1.35)	(-2.10)	(-1.04)	(-0.38)	(-0.34)	(0.79)	(-1.41)	(-1.04)	(0.28)
Age in months	-0.001***	-0.001***	-0.001***	-0.0004***	-0.0003***	-0.0001	-0.000	0.000	-0.000	0.000	0.000	-0.0000622
	(-3.46)	(-3.28)	(-3.63)	(-3.79)	(-2.62)	(-1.52)	(-0.02)	(1.03)	(-0.29)	(0.54)	(1.07)	(-0.27)
Age in months^2	0.000***	0.00***	0.00***	0.000***	0.000**	0.000	-1.84e-08	-0.000	0.000	-0.000	-0.000	0.00000132
	(3.12)	(2.87)	(3.58)	(3.68)	(2.38)	(1.40)	(-0.01)	(-1.22)	(0.32)	(-0.54)	(-1.17)	(0.37)
Age in months^3	-0.000***	-0.000**	-0.000***									
	(-2.72)	(-2.53)	(-3.40)									
Vaccines	0.00440**	0.00493**	0.00458**	0.00882***	0.00649***	0.00376*	-0.00137	-0.0143***	-0.00836	-0.00123	-0.0128**	-0.00335
	(2.14)	(2.24)	(2.02)	(3.99)	(2.87)	(1.65)	(-0.54)	(-2.89)	(-1.38)	(-0.42)	(-2.29)	(-0.49)
D. N. Supplement	-0.000101	0.000511	0.000125	-0.000557	-0.000218	-0.000216	-0.000823	-0.00459***	-0.00332**	-0.00113	-0.00473***	-0.00333*
	(-0.10)	(0.48)	(0.11)	(-0.49)	(-0.19)	(-0.19)	(-1.18)	(-3.38)	(-2.00)	(-1.37)	(-3.02)	(-1.73)
D. Diarrhea	0.00118	0.00112	0.00207**	0.00198**	0.00171*	0.00196**	-0.000803	0.000205	0.000408	-0.00108	-0.00144	0.000618
	(1.44)	(1.28)	(2.30)	(2.16)	(1.82)	(2.07)	(-0.92)	(0.12)	(0.20)	(-1.08)	(-0.75)	(0.26)
D. Daycare	0.00001	0.001	-0.00131									
	(0.02)	(1.60)	(-1.16)									
N. month breastfed	0.0002	0.0002	0.0001	0.0001	0.00008	0.0001	0.0005***	0.0007***	0.0005*	0.0006***	0.0007**	0.000463
	(1.60)	(1.29)	(1.18)	(1.07)	(0.52)	(1.07)	(4.34)	(2.90)	(1.66)	(3.92)	(2.48)	(1.28)
Age mother	0.00003	0.000009	0.00008	0.0001***	0.0001***	0.0002***	-0.00007*	-0.00008	-0.00004	-0.00002	-0.00002	0.0000236
	(0.62)	(0.17)	(1.46)	(3.05)	(2.95)	(3.43)	(-1.84)	(-1.13)	(-0.45)	(-0.36)	(-0.18)	(0.13)
Schooling mother	0.0003***	0.0004***	0.0005***	0.0006***	0.0007***	0.0008***	0.00007	-0.0001	-0.00004	-0.00001	-0.0002	-0.000191
	(2.62)	(3.34)	(3.75)	(5.27)	(5.84)	(5.92)	(0.77)	(-0.63)	(-0.21)	(-0.10)	(-1.08)	(-0.67)
Fertility mother	-0.0176***	-0.0170**	-0.0135*									
	(-2.68)	(-2.42)	(-1.86)									
D. C-section	-0.000929	-0.000121	0.00114									
	(-1.08)	(-0.13)	(1.21)									
D. ObGyn	-0.000849	-0.00288**	-0.00128									
	(-0.80)	(-2.52)	(-1.08)									
D. Mother underemployed	0.00355***	0.00183**	0.000521									
	(4.88)	(2.36)	(0.65)									
D. N. Supplement mother	0.00154*	0.000689	0.00130									
	(1.77)	(0.74)	(1.36)									
Height mother										0.000	-0.000166	-0.0000636
										(0.75)	(-1.31)	(-0.41)
BMI mother										-0.000	-0.000	-0.000311
										(-0.04)	(-0.49)	(-1.56)
Height father										-0.00**	-0.00	0.0000420
										(-2.03)	(-1.48)	(0.30)
BMI father										0.00	-0.00	0.000174
										(1.11)	(-0.51)	(0.75)
Ln hh consumption per capita	-0.005***	-0.005***	-0.007***	-0.009***	-0.009***	-0.01***	-0.001*	0.0007	0.001	-0.001	0.001	0.00103
	(-6.26)	(-6.20)	(-8.27)	(-10.74)	(-10.43)	(-11.39)	(-1.82)	(0.51)	(1.07)	(-1.45)	(0.89)	(0.47)
D. welfare	0.00131	0.00249***	-0.000300	-0.000901	0.000418	-0.00202**	0.000566	0.00251	-0.00193	0.0006	0.002	-0.00373*
	(1.53)	(2.72)	(-0.32)	(-0.93)	(0.42)	(-2.03)	(0.72)	(1.64)	(-1.04)	(0.67)	(1.37)	(-1.72)
sqrt n children<12 in hh	0.00116	0.00150	0.0000960	-0.00134	-0.000217	-0.000797	0.00336***	0.00232	0.00454*	0.00211	0.002	0.00451
	(0.95)	(1.16)	(0.07)	(-1.19)	(-0.19)	(-0.69)	(3.18)	(1.13)	(1.81)	(1.64)	(0.84)	(1.50)
Index living conditions	0.007***	0.007***	0.007***	0.007***	0.007***	0.007***	0.0007**	0.002***	0.002***	0.0004	0.002***	0.00269***
	(12.35)	(11.87)	(10.16)	(10.50)	(10.59)	(10.68)	(2.47)	(4.28)	(3.37)	(1.15)	(3.72)	(3.29)
Work experience head hh	-0.00***	-0.00	-0.00	-0.00***	-0.00**	-0.00	-0.00	-0.00	-0.00	-0.000	-0.00	0.0000750
	(-2.72)	(-0.87)	(-0.79)	(-4.04)	(-1.97)	(-1.14)	(-0.15)	(-1.49)	(-0.24)	(-0.13)	(-0.96)	(0.80)
Mean size agri. Land	0.0008***	0.0008**	0.0006***	0.001***	0.001***	0.0008***	-0.0001***	-0.0001***	-0.0001***	-0.0001***	-0.0001***	-0.000170***
	(17.44)	(16.33)	(12.40)	(20.74)	(20.07)	(16.45)	(-24.75)	(-10.52)	(-9.17)	(-19.56)	(-8.91)	(-7.88)
Mean rent/ha. ag. Land	-0.000***	-0.000	0.000	0.00009***	0.0001***	0.00009***	-0.00004***	-0.00005***	-0.00004***	-0.00004***	-0.00005***	-0.0000456***
	(-4.78)	(-0.92)	(1.14)	(9.34)	(10.04)	(8.91)	(-12.74)	(-7.88)	(-5.23)	(-9.74)	(-6.51)	(-4.49)

Rate attendence secondary school	-0.0064***	-0.002	-0.00**	0.00673***	0.009***	0.010***	0.00321*	0.017***	0.0189***	0.00499***	0.0179***	0.0186***
	(-3.10)	(-1.30)	(-2.39)	(2.87)	(4.13)	(4.26)	(1.94)	(5.51)	(4.80)	(2.62)	(4.95)	(4.19)
D. rural	-0.0554***	-0.0353***	-0.0342***	-0.0736***	-0.0622***	-0.0613***	0.0000177	-0.00795***	-0.0274***	0.000183	-0.00852***	-0.0278***
	(-35.14)	(-23.21)	(-23.24)	(-39.25)	(-32.44)	(-31.69)	(0.02)	(-4.78)	(-13.47)	(0.18)	(-4.45)	(-11.86)
Ln(GDP prov)	0.00388***	0.00247***	0.000753*	0.00589***	0.00493***	0.00385***	0.0155***	0.00927***	0.00244**	0.0160***	0.0109***	0.00372***
	(9.94)	(6.02)	(1.81)	(12.02)	(9.83)	(7.62)	(30.46)	(9.36)	(2.02)	(26.88)	(9.57)	(2.67)
Poverty prov	0.129***			0.291***	0.260***	0.244***	0.185***	0.156***	0.108***	0.188***	0.157***	0.106***
	(11.98)			(24.62)	(21.49)	(19.95)	(42.78)	(18.48)	(10.45)	(36.61)	(15.99)	(8.82)
Mean consumption prov.	0.00005			0.0004***	0.0004***	0.0005***	0.0002***	0.0001***	0.0001***	0.0002***	0.0001***	0.0001***
	(1.24)			(7.44)	(7.99)	(9.25)	(18.32)	(5.91)	(4.74)	(15.00)	(4.29)	(3.59)
Poverty county		0.0699***										
		(7.97)										
Mean consumption count.		0.0001**										
		(2.24)										
Poverty parish			0.0715***									
			(9.08)									
Mean consumption parish			0.000265***									
			(7.31)									
Index food consumption	0.00808***	0.00685***	0.00550***									
	(26.51)	(21.12)	(16.45)									
N. MD/10000 ppl.	-0.0000373*	-0.0000138	-0.0000112									
	(-1.65)	(-0.57)	(-0.45)									
Mean distance highway	-0.000459	0.00269***	0.00661***									
	(-0.55)	(2.96)	(7.16)									
Mestizo.ethnic												
Indigena.ethnic	0.0148***	0.0124***	0.0135***	0.0168***	0.0160***	0.0169***	0.000546	0.00163	0.00430*	-0.000143	0.00130	0.00322
	(12.61)	(9.79)	(10.15)	(13.28)	(12.38)	(12.97)	(0.50)	(0.76)	(1.65)	(-0.11)	(0.53)	(1.08)
Afro.ethnic	-0.00468***	0.000540	0.00181	-0.00740***	-0.00215	-0.000464	0.0115***	0.0174***	0.0211***	0.0121***	0.0221***	0.0261***
	(-3.19)	(0.35)	(1.12)	(-4.42)	(-1.25)	(-0.27)	(7.24)	(5.63)	(5.60)	(5.93)	(5.70)	(5.51)
D. Rural Amazon	0.122***	0.120***	0.117***									
	(57.82)	(55.20)	(52.60)									
Highlands.region				-0.0575***	-0.0562***	-0.0653***	0.0420***	0.0483***	0.0428***	0.0412***	0.0442***	0.0384***
				(-27.72)	(-26.48)	(-30.52)	(12.06)	(7.13)	(5.17)	(10.41)	(5.85)	(4.16)
Coast.region				-0.0916***	-0.0870***	-0.0862***	0.00653*	0.0110	0.0262***	0.00406	0.00516	0.0198*
				(-42.71)	(-39.63)	(-38.96)	(1.71)	(1.47)	(2.89)	(0.94)	(0.62)	(1.95)
Amazon.region							0.0764***	0.0812***	0.0773***	0.0755***	0.0777***	0.0720***
							(20.31)	(11.09)	(8.64)	(17.57)	(9.46)	(7.16)
Galapagos.region												
Instrument	**0.0447*** **	**0.0407*** **	**0.0204*** **	**0.0236*** **	**0.0187*** **	**0.0120*** **	**-0.220*** **	**-0.274*** **	**-0.201*** **	**-0.222*** **	**-0.297*** **	**-0.195*** **
	(11.43)	**(9.86)**	**(4.91)**	**(5.24)**	**(4.06)**	**(2.58)**	**(-12.78)**	**(-8.19)**	**(-4.91)**	**(-11.04)**	**(-7.74)**	**(-4.14)**
_cons	0.284***	0.311***	0.326***	0.210***	0.220***	0.247***	0.00766	0.120***	0.204***	0.0121	0.160***	0.200***
	(23.19)	(25.89)	(26.31)	(15.07)	(15.41)	(17.16)	(0.96)	(7.77)	(10.80)	(0.78)	(5.42)	(5.54)
R-sq	0.7008	0.6333	0.6092	0.6624	0.6193	0.6019	0.8461	0.5557	0.3638	0.8461	0.5748	0.3818
N	5038	5038	5038	5281	5281	5281	2605	2594	2594	1947	1938	1938
F(1, 5001)	130.54	97.27	24.15	.	.	.	.	.	.	.	.	.
F(1, 5253)	.	.	.	27.45	16.48	6.67	.	.	.	.	.	.
F(1, 2575)	.	.	.	.	.	.	161.41	.	.	.	.	.
F(1, 2564)	.	.	.	.	.	.	.	67.03	24.05	.	.	.
F(1, 1913)	.	.	.	.	.	.	.	.		119.64	.	.
F(1, 1904)	.	.	.	.	.	.	.	.	.	.	60.3	17.24

Source: Author's computation using 2006 /2014 LSMS

4.5.1 The effect of inequality

When discussing the results I will focus on the IV models of the best fit for each year, that is IV1-IV3 for 2006 and IV10-IV12 for 2014. If I refer to the IV models where I use the same variables for both years (IV4-IV9) or if I refer to the OLS results I say so explicitly.

The Gini coefficient is significant and has a deleterious effect in every model in 2006, however, it is negative but not significant in 2014. The effect is smaller in the OLS models than it is in the IV models which I believe is due to the omitted variables. It is difficult to define a "one unit" change in the Gini coefficient, however, the z-score of height for age is measured in standard errors. Therefore, in 2006, a one unit increase in the Gini coefficient decreases the z-score by -11.4 (province), -9.9 (county), -21.9 (parish) standard errors, and, that it had no effect in 2014.

In the first stage models, the F-statistics are all above 10 and all the t-values are above 4. The F-statistics are much lower for models IV4-IV6 (where there are the same variables as the 2014 model). The F-statistic for model IV6 is under 10 and the t-value is under indicating that the instrument is weak in this model.

In 2014 there is a similar tendency with relation to the magnitude and sign of the coefficients in the OLS with relation to the IV models, however, they are not significant. The model measures the effect of the variance in the Gini coefficient across provinces, counties and parishes on the variance of z-scores in children living in these provinces, counties and parishes. Therefore, it is likely that the variability of Gini's across provinces, counties and parishes changed from 2006 to 2014 in such a way that it no longer affects the individual health outcomes of children.

Additionally, there may be changes in the behaviour of households between 2010 and 2014 which are not captured in our small area estimations of the Gini coefficient. As mentioned above, in order to use the 2014 estimated parameters (correlation coefficients) to simulate consumption using the population characteristics of 2010, it must be assumed there is very little change in behaviours between 2010 and 2014. However, this cannot be true as the resulting 2006 Gini coefficients are fundamentally different from the resulting 2014 Gini coefficients. Therefore, the 2014 estimation might differ significantly if they are projected onto the (not yet constructed or released) 2020 census. Future research should focus on replicating the 2014 on the 2020 census and identifying which census is better suited to create the simulation. That is to say, identifying which time period (between LSMS and census) saw the least changes in living conditions.

Larrea and Kawachi (2006) do no find a significant relationship between consumption inequality and chronic child malnutrition at the county or at the parish level, in spite of using multilevel models in addition to multivariable regressions. This may be due to the data from the LSMS of 1998 had less variables as well as a smaller sample size. The LSMS of 1998 has a sample of 2723 children under the age of five which is

just under half of the sample (6003) which is available in the LSMS 2006 survey. They do, however, find a deleterious significant relationship at the provincial level providing partial evidence of the effect (Larrea & Kawachi, 2005). The 1998-2000 estimation model is not included in this paper as it would be a replication of the Larrea & Kawachi (2005) publication and our objective was to study the more current situation with alternative causal methods.

4.5.2 The effect of income

The natural log of per capita household consumption is positive and significant in 2006 and 2014 models (Table 4.5). The concave effect that income has on health is captured in this variable as it is logged. The magnitude of the effect is around 0.1 in 2006 and 0.3 in 2014. The effect of consumption has a larger magnitude than other income related variables such as the mean consumption per province (around 0.01 in 2006 and around 0.003 in 2014), the natural log of provincial GDP (around 0.09 in 2006 and 0.1 in 2014). The effect of the incidence of poverty in the province is the opposite: it is positive and significant in the 2006 (1.7) and not significant in the 2014 models.

We also incorporate the mean consumption of the subpopulation (province, county or parish) of the model so as to capture the possible externalities of having wealthier neighbors on access to healthcare or on individual behaviours. This variable is significant and has a positive effect in the second specification of the 2006 IV models and in some 2014 IV models.

4.5.3 Other effects

4.5.3.1 Individual characteristic of the child

Children who are born with LBW have significantly lower z-scores in every model. This implies that the pathway between psychosocial stress, prenatal maternal stress and LBW may be in action although it is not tested it directly here, providing evidence to indicate that there may be long-term effects of LBW. Dummy female is positive and significant in the 2006 and 2014 models. Age is also significant in every model (in 2006 a cubed model is presented and in 2014 it is squared). It would seem that the z-score decreases with age but by less and less as age increases. Children who are younger tend to still be breastfeeding which may help maintain their growth patterns within the normal range, however, as they get older they stop breastfeeding and perhaps at this moment they become more vulnerable to fall outside of the normal growth range. This is probably the reason the number of months of breastfeeding is not a significant variable in any model; this effect might be absorbed by the age variable. The proportion of vaccines is significant in the 2006 but not in the 2014 models, the effect of the nutritional supplement is negative and mainly not significant due to selection-bias, and the effect of access to daycare is not significant. Dummy diarrhea is negative and significant in 2014 but not in 2006.

4.5.3.2 Characteristics of the mother and father

The maternal age is only significant (positive) in 2006, however, the schooling of the mother has a positive significant effect on the outcome in every model. The fertility (number of children) of the mother has a negative significant effect, as does dummy maternal under-employment, while having access to a caesarian section and to a MD during birth is positive and significant in 2006. Maternal access to nutritional supplements has a negative significant effect due to selection bias. I did not find these variables relevant in 2014. On the other hand, in the 2014 model the BMI of the father and height of the mother and father are positive and significant both years demonstrating how important they are in the outcome.

4.5.3.3 Characteristics of the household, region and fixed effects

The index of living (housing) conditions is positive and significant in every model. The square root of the number of children under 12 is significant and negative in 2006 but not in 2014. Finally, the years of work experience of the head of household is significant in 2006 but not in 2014. It seems that most of the household conditions are absorbed by the household consumption per capita.

The regional characteristics (mean size and rent of agricultural land, dummy rural, the rate of attendance to secondary education) are significant in the 2006 OLS and IV models but lose all significance in 2014. Indicating a reduction in the importance of the local context on individual health outcomes. In 2006 the mean distance to the highway which is positive and significant, and the number of MD per every 10000 people is not significant.

In the ethnicity fixed effect, mestizo is the reference group. In 2006 afro-Ecuadorians have a positive significant effect while in the 2014 model dummy indigenous has a negative significant effect. In terms of regions, in 2006, the fixed effect for Galapagos cannot be measured, as it was not included in the survey, therefore, here the reference group is rural amazon. In 2014, Galapagos is the reference group. I find no significant differences between the regions in 2014. In the 2006 model rural amazon dummy is positive and significant. This dummy basically captures the same phenomenon as the fixed effects for regions. Obviously the purpose of the fixed effects is to measure the effect within regions and ethnicities rather than between them, not to see whether the effect is significantly different.

4.6 Conclusion

In this paper our objective is to measure the effect of inequality on stunting in children under the age of 5. I regress the z-score of height-for-age[122] using IV model regressions against the provincial, county and parish Gini coefficients while controlling for individual, maternal, household and contextual characteristics.

[122] The normalized *z*-score establishes the growth standard of children by defining a normal growth curve, see page 16 for formal definition.

I run two iterations of the model, one using the 2006 LSMS and a second using the 2014 LSMS. Our results show that, the Gini coefficient has a significant deleterious correlation on malnutrition in 2006 but not in 2014.

The effect is still negative, however, loses significance in 2014. This may be due to the recent reduction in inequality at a national and sub-national level, but most probably due to a shift in the distribution of Gini coefficient between 2006 and 2014. It is difficult to pin-point the exact cause of the erosion of the effect, however, the change in the distribution of the Gini coefficient and its effect on its relationship to stunting is not the topic of this paper. Notwithstanding, it is related and is a potential field of further research.

The main limitation is the way I measure the Gini coefficient over small areas. The *small area estimates* model depends heavily on a degree of heterogeneity which cannot be controlled for methodologically and cannot be guaranteed empirically. The efforts made to divide the country into homogeneous regions may abate this limitation, however, it was not possible to generate Gini coefficient which are not systematically under-estimated. This may in part be due to a consumption model which measures the conditions of the middle and lower income earners. Therefore, when it is simulated onto the census it under-estimates the consumption of the rich and under-estimates the Gini coefficient (Tarozzi & Deaton, 2009).

Other simulations methods, particularly newer recurring neural networks based on artificial intelligence are more powerful in terms of prediction error, however, these methods are black box methods with "hidden" layers where the parameters (correlation coefficients) are not explicit (Montavon, et al., 2018). Apart from machine/deep learning, there are limited alternatives to the *small area estimates* in terms of methods which address the issues raised by Deaton and Tarozzi (2009).

This study contributes to the larger literature regarding the health-inequality relation on three levels. Firstly, I focus on the nutritional health of children which has an effect on their educational achievements and income in adulthood, potentially playing a role in the intergenerational transmission of poverty. In Lynch at al. (2004) empirical review of the inequality health relation there is only a small percentage of studies[123] (23.7%) which focus on child health. Most of these studies measure infant mortality[124], which unlike nutrition, does not play a role in the intergenerational transition of poverty. Secondly, I focus on Latin America. In Lynch at al. (2004) empirical review only 10.2% of the studies 125 include Latin American countries despite the fact that it is the most unequal region in the world (Inter-American Development Bank, 2000) and perhaps therefore an ideal testing ground for the effect of inequality. Finally, I measure the Gini coefficient across different geographical scales. In Lynch at al. (2004) empirical review only 9.2% of the

[123] Studies where the information was specified.
[124] Infant mortality
[125] Studies where the information was specified.

studies[126] measure the Gini coefficients at different levels. Given the effects of the Gini may vary depending on the how it is measured[127], this exercise allows a more profound analysis of inequality on health (Lynch, et al., 2004).

[126] Studies where the information was specified.

[127] In the Wilkinson and Pickett review (2006) there are 45 international, 58 state levels, 25 county level and 40 small area level studies. There are relatively less studies performed at the county level while mostly studies focus on the state level. Additionally, the distribution of the supportive, mixed and unsupportive evidence favors the supportive at the state level (S: 51.7%, M: 25.8%, U: 22.4%) while it is fairly balanced at the small area level (S: 30%, M: 35%, U: 35%). In this study, I find that the magnitude of the Gini coefficient is smaller as the areas over which it is measured decreases in size. Therefore, in order to fully assess the impact of inequality it is important to take different levels of aggregation as I have done.

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Appendix 1 Detailed explanation of our small area estimates methodology based on Elbers et al (2003)

In 2003, Elbers et al. use the LSMS of 1994, and take household per-capita expenditure as their indicator of wellbeing. They construct populations of increasing sizes from a constant distribution $G_v(x, m)$ by randomly drawing households from the census households in one particular region of the country, the rural coast. For each population size the table shows the welfare estimations, the standard errors of the predictions and the share of total variance due to the idiosyncratic component of the error. The idiosyncratic error is important given that it is the component of the total error which increases with a reduction in the target population. Elbers, et al. (2003), demonstrate, in Table A1.1, that for a sample of 15000 households the idiosyncratic component, V_l, is small and there is little to gain from increasing the sample size or moving to higher levels of aggregation (Elbers, et al., 2003).

Table A1.1 Simulation Results

		Number of Household			
Measure	Estimated Values	100	1000	15000	100000
Headcount	μ	0.46	0.5	0.51	0.51
	Total Standard Error	0.067	0.039	0.024	0.024
	V_l/Total Variance	0.75	0.24	0.04	0.02
General Entropy (0.5)	μ	0.26	0.28	0.28	0.28
	Total Standard Error	0.048	0.029	0.022	0.022
	V_l/ Total Variance	0.79	0.28	0.03	0.01

Source: Redrawn from Elbers, et al., 2003

As they have shown, when combining census and survey data, de-aggregating to sub-regions and estimating poverty, in our case, the Gini coefficient, and the per-capita household consumption, for specific locations becomes possible. When they estimate their welfare indicators by parishes they demonstrate, in Appendix Table A1.2, that one can estimate the Gini coefficient using combined data for subpopulations one hundredth the size of those one can estimate in survey data and obtain very similar prediction errors (Elbers, et al., 2003).

Table A1.2 Improvement using combined data

	Sample Data Only (region)		Combined Data (suregions)	
Region	(2) S.E. of Estimate	(3) Population (1000s)	(4) S.E. of Estimate Median	(5) Population Median (1000s)
Rural Highlands	0.027	2509	0.038	3.3
Rural Cost	0.042	1985	0.046	4.6
Rural Amazon	0.054	298	0.043	1.2
Urban Highland	0.026	1139	0.026	10
Urban Cost	0.03	1895	0.031	11
Urban Amazon	0.05	55	0.027	8
Quito	0.033	1193	0.048	5.8
Guayaquil	0.027	1718	0.039	6.5

Source: Redrawn from Elbers, et al., 2003

Table A1.3 presents the median standard error, population and number of households in the parishes within each of these sub-regions. The number of households in every parish is well below the 15000 mark established in Elbers, et al. (2003) which indicates that the total variance due to the idiosyncratic component of the error might be somewhat above the ideal level (0.24). Nevertheless, the median standard errors in this study tend to be smaller than those presented by Elbers et al. (2003) and the population size is similar or larger than those found in Elbers et al. (2003).

Table A1.3 Population size and standard errors of the Gini coefficient

	Median #households parish (1000s)	Median Standard Error (parish)		Median Population (parish) (1000s)	
Region	This study	This study	Elbers et al. (2003)	This study	Elbers et al. (2003)
Quito	4.2	0.0075	0.048	14.4	5.8
Guayaquil	5.3	0.0081	0.039	15.1	6.5
Urban Coast	3.6	0.0102	0.031	9.2	11
Rural Coast	1.1	0.0127	0.046	4.3	4.6
Urban Highlands	2.6	0.0066	0.026	8.2	10
Rural Highlands	0.8	0.0144	0.038	2.8	3.3
Urban Amazon	3.2	0.011	0.027	9.3	8
Rural Amazon	0.4	0.022	0.043	1.4	1.2
National Total	1	0.014		3.6	

Source: Redrawn from Elbers, et al., 2003

Appendix 2 Consumption prediction models using Small Area Estimates

Table A2.1 Consumption prediction model for Small Area Estimates 2006-2010

Dependent Variable: LNCONPCM Weighted by: FEXP	Quito	Guayaquil	Urban Coast	Rural Coast	Urban Highlands	Rural Highlands	Urban Amazon	Rural Amazon
intercept	3.9244***	4.69609***	4.95897***	4.24822***	4.66441***	3.81323***	4.21719***	5.12909***
	(0.54644)	(0.32232)	(0.17108)	(0.16086)	(0.23849)	(0.17533)	(0.33065)	(0.2845)
Access to higher education	0.90309***							
	(0.09756)							
Average proportion of household with cement walls in statistical area				-0.14443***		0.13408***		
				(0.04016)		(0.03017)		
Average proportion of houses with connection to public water disposal service					-0.16132**	-0.12525**		
					(0.05754)	(0.03788)		
Average proportion of housing with exclusive toilets in statistical area	-0.11827		-0.11533		0.22179**			
	(0.14344)		(0.06497)		(0.07307)			
Average proportion of houses with garbage truck service in statistical area	1.231*		-0.12598*					
	(0.5303)		(0.05113)					
Average proportion of houses with publicly provided drinking water in statistical area			0.05851				-0.32875**	
			(0.03787)				(0.11025)	
Average proportion of persons per room in statistical area		-0.20641	-0.05895**			-0.06983***	0.04303	-0.20006***
		(0.15818)	(0.02121)			(0.01585)	(0.02425)	(0.02644)
Average years of schooling in parish		0.04799**						
		(0.0176)						
Dummy amplified nuclear family					-0.08971**	0.05536*		
					(0.02868)	(0.02572)		
Dummy Cuenca					0.16585***			
					(0.02294)			
Dummy bamboo flooring or similar		-0.21931**	-0.07458	-0.09438**		-0.06947**		
		(0.07354)	(0.0439)	(0.03403)		(0.02424)		
Dummy for cement or brick flooring		-0.1966***				-0.08137***		
		(0.04597)				(0.02228)		
Dummy for walls made of bamboo wood or similar		-0.17853**	-0.06676*					
		(0.0547)	(0.0309)					
Dummy head of household affiliated to social security		0.08396**	0.0631**	0.11415**		0.1265***		
		(0.03172)	(0.0237)	(0.03546)		(0.03291)		
Dummy head of household construction worker		-0.0573	0.32542***	-0.12684*	0.08557***	0.12598***	0.20716***	
		(0.04312)	(0.06257)	(0.04962)	(0.02146)	(0.02989)	(0.05118)	
Dummy head of household directive position	0.17944*	0.31372***		0.27233*			0.31744**	
	(0.07427)	(0.067)		(0.12508)			(0.10396)	
Dummy head of household employer	0.09348	0.15455***	0.23413***	0.19037***	0.22634***	0.16207***	0.21176**	
	(0.05634)	(0.04382)	(0.02869)	(0.02919)	(0.03255)	(0.03692)	(0.06899)	
Dummy head of household ethnic	0.07023	0.07685						-0.14128**
	(0.05121)	(0.04629)						(0.04509)
Dummy head of household female	-0.08383*				-0.09081**	0.04279		
	(0.03465)				(0.02807)	(0.02744)		
Dummy head of household in hotel industry					0.12719*	0.12641		
					(0.04931)	(0.07932)		
Dummy head of household in manufacturing			0.05821					
			(0.03067)					
Dummy head of household in retail sale			0.10744***	0.04692	0.0947**		-0.0909	-0.39423***
			(0.02441)	(0.03929)	(0.02895)		(0.06048)	(0.10429)
Dummy head of household inactive			0.09164*		0.04816		0.4985***	
			(0.04158)		(0.03294)		(0.1199)	
Dummy head of household marital status divorced/separated			-0.05765*	0.00655				
			(0.02748)	(0.03115)				
Dummy head of household marital status single			-0.13252***	-0.06892		-0.11749***		
			(0.03986)	(0.04049)		(0.03471)		
Dummy head of household non-qualified agricultural worker			0.0731*					-0.23459***
			(0.0363)					(0.053)
Dummy head of household non-qualified worker			-0.04806*					
			(0.02252)					

Dummy head of household other service position	-0.15888	0.12451*				-0.11051		
	(0.08409)	(0.05073)				(0.06869)		
Dummy head of household over 65 years of age				-0.15334**	-0.07733*	-0.158***		
				(0.04989)	(0.03123)	(0.02902)		
Dummy head of household public sector		0.38836	0.15076***			0.2044	-0.36949*	0.23886**
		(0.22173)	(0.03427)			(0.11238)	(0.16818)	(0.0731)
Dummy head of household salary worker	-0.08119*	-0.0754*			-0.08235**	-0.08073**		-0.14238**
	(0.0359)	(0.03323)			(0.02572)	(0.0247)		(0.05453)
Dummy head of household speaks native language						-0.03236		-0.10053*
						(0.0292)		(0.04922)
Dummy head of household speaks native language and Spanish							0.0881	
							(0.0559)	
Dummy head of household transportation		0.09764*	0.11166**		0.15424***	0.12366**	0.17751*	
		(0.04331)	(0.03431)		(0.03626)	(0.04404)		
Dummy head of household wholesale worker		0.10023*	0.09169*	0.24802***	0.12789***	0.19403***		
		(0.04689)	(0.0401)	(0.07271)	(0.03801)	(0.05837)		
Dummy head of household widow/widower		-0.05469		-0.01949				
		(0.05432)		(0.03492)				
Dummy head of household works in modern sector				0.05895**		0.09492***	0.12742*	
				(0.02019)		(0.01993)	(0.05373)	
Dummy household garbage is burnt or buried				0.06808***			0.13293	0.11474*
				(0.01873)			(0.08065)	(0.04524)
Dummy household garbage is thrown in empty lot							0.29669*	
							(0.11947)	
Dummy household that share or do not have toilet				0.04359				
				(0.02267)				
Dummy household water connection outside the building and the property	-0.27689*			-0.01256		-0.17965***	-0.38079**	
	(0.12968)			(0.01809)		(0.04664)	(0.14088)	
Dummy household with adobe walls				-0.15419*				
				(0.06985)				
Dummy household water connection outside the building but inside the property					-0.07658*	-0.0674*	0.1763**	
					(0.03296)	(0.02751)	(0.06746)	
Dummy household with asbestos roof or similar	-0.09016*			0.02289	0.00517	0.05291**		
	(0.03702)			(0.03795)	(0.02097)	(0.02006)		
Dummy household with electric stove	0.47731**					0.97216*		
	(0.15683)					(0.42308)		
Dummy household with palm/straw roof or similar				-0.08914*		-0.04628		
				(0.03572)		(0.08125)		
Dummy household with room for rent					0.0358			
					(0.03725)			
Dummy household with wood walls					-0.05554			
					(0.09368)			
Dummy household wood/coal stove				-0.10355***	-0.23951**	-0.17803***	-0.61124**	-0.28142***
				(0.02589)	(0.08161)	(0.02292)	(0.19751)	(0.05506)
Dummy housing with no electricity		-0.67314	0.0689		-0.38417*	-0.10891**		
		(0.38913)	(0.0757)		(0.17524)	(0.03945)		
Dummy housing with no telephone	-0.22134***	-0.13318***	-0.23438***	-0.26983***	-0.19948***	-0.17905***	-0.19044***	-0.28064***
	(0.03785)	(0.02912)	(0.02075)	(0.04135)	(0.02135)	(0.02539)	(0.04837)	(0.08092)
Dummy housing provided in exchange for services			-0.07415**	-0.07281**	-0.09788***	-0.04755*		-0.12341*
			(0.02395)	(0.02215)	(0.02516)	(0.02301)		(0.05236)
Dummy housing with exclusive room for cooking		0.18247**		0.1422*		0.36858**		0.40627***
		(0.06259)		(0.05986)		(0.11203)		(0.12162)
Dummy housing with latrine	-0.95173***	-0.18963*				-0.04757		
	(0.27272)	(0.08365)				(0.02945)		
Dummy housing with no shower	-0.20037***	-0.10793**	-0.14549***		-0.01579	-0.11995***	-0.13628*	
	(0.04564)	(0.03456)	(0.02258)		(0.03132)	(0.02232)	(0.05606)	
Dummy housing with other stove	-0.14827							
	(0.17555)							
Dummy housing with toilet and septic tank		0.04789	0.03785	0.12972***		0.0763***		
		(0.0315)	(0.02089)	(0.02104)		(0.02111)		
Dummy incomplete nuclear family			0.04721		-0.01417	0.09195**	0.08409	
			(0.02525)		(0.02866)	(0.02787)	(0.05071)	
Dummy indigenous head of household	0.08425							

	(0.09848)							
Dummy metal zinc roof		-0.07327*			0.0561		0.0607	
		(0.0302)			(0.03515)		(0.04587)	
Dummy tile flooring or similar					0.08308***			
					(0.02116)			
Dummy precarious housing		0.41124**				-0.12429		
		(0.15054)				(0.07863)		
Dummy rented housing	-0.12113***	-0.08006**	-0.07646**			-0.08938*	-0.12164*	
	(0.03102)	(0.0302)	(0.02409)			(0.04269)	(0.04872)	
Dummy semi-precarious housing		0.13243**	0.08558*	0.04575		-0.12763***		
		(0.04624)	(0.0334)	(0.03728)		(0.03208)		
Dummy tile flooring or similar		0.05854	0.19685***	0.23252***		0.11607*	0.14214**	
		(0.05148)	(0.02308)	(0.04915)		(0.04679)	(0.05326)	
Elementary school attendance net rate							0.23752	-0.54376**
							(0.16138)	(0.18509)
Head of household education * dummy head of household formal sector	0.0108**				0.01024***			
	(0.00329)				(0.00183)			
Head of household education * dummy head of household public sector		0.00409	0.00498*			-0.02533*		
		(0.00289)	(0.00227)			(0.01004)		
Head of household education * dummy head of household house worker		-0.02849**						-0.07134*
		(0.00974)						(0.03342)
Head of household education * dummy head of household public sector		-0.02311					0.01864	
		(0.0149)					(0.01284)	
Head of household education * head of household experience	0.00017	0.00078***	0.00027**	0.00065***	0.00046***			
	(0.00024)	(0.00022)	(0.0001)	(0.00012)	(0.000074955)			
Head of household experience	0.00323		-0.00072	0.01974*		0.0009		
	(0.00436)		(0.00214)	(0.00993)		(0.00104)		
Head of household experience2	0	-0.00036*		-0.00093*				
	(0)	(0.00014)		(0.00036)				
Head of household experience3		0*	0	0.000011198**				
		(0)		(0)				
Head of household schooling	0.01307	-0.03468*			0.03081***	0.00616		0.02007***
	(0.01875)	(0.01688)			(0.00305)	(0.00684)		(0.00584)
Head of household schooling2	0.00066	0.0012	0.00081***	0.0004		0.00168***	0.00078*	
	(0.00068)	(0.00064)	(0.0002)	(0.00027)		(0.00043)	(0.00033)	
High school attendance net rate in parish	0.15497*	-0.05731	0.05553	0.09346**		-0.07176*		
	(0.06223)	(0.0506)	(0.04261)	(0.03176)		(0.03481)		
Household water obtained from stream or similar			-0.13784***			0.09818**	0.17614	
			(0.03786)			(0.03553)	(0.10576)	
Household water obtained well			-0.13309***	0.03924*		0.18506**		
			(0.03445)	(0.01996)		(0.064)		
Household with room for family business			0.03804					0.2489***
			(0.03129)					(0.07349)
Household with toilet without septic tank, just dung up well			-0.02735		-0.1627*			0.09123
					(0.07835)			(0.05648)
Ln(Income per-capita)	0.04942***	0.17483***		0.14104***		0.14508***	0.11786***	0.17597***
	(0.01444)	(0.02635)		(0.01217)		(0.01589)	(0.0289)	(0.03269)
Percentages of houses in parish with parquet floors or similar		-0.39243		0.08402	-0.26581			
		(0.24413)		(0.07599)	(0.20628)			
Rate of literacy in statistical area			0.37477*		0.40517	0.21459**		
			(0.14607)		(0.2192)	(0.07405)		
Rooms per person	0.22059***	0.25524***	0.21138***	0.16251***	0.21087***	0.18977***	0.14312**	0.09979**
	(0.02652)	(0.03094)	(0.01879)	(0.01775)	(0.01513)	(0.01748)	(0.04331)	(0.03445)
Square root of number of basic needs met	-0.08573***	-0.06066	-0.05829**	-0.13819***	-0.11805***	-0.08635**	-0.21112***	-0.1249***
	(0.02511)	(0.03152)	(0.01799)	(0.0273)	(0.02079)	(0.02689)	(0.04291)	(0.03662)
Square root of number of hours of work of head of household		-0.02393**	0.02043***		-0.00328		0.03463**	0.02253
		(0.00832)	(0.0048)		(0.00457)		(0.013)	(0.01175)
Square root of number of people in household	-0.38042***	-0.28734***	-0.33855***	-0.33752***	-0.2499***	-0.30488***	-0.34108***	-0.43351***
	(0.05296)	(0.04401)	(0.03044)	(0.03138)	(0.03465)	(0.03308)	(0.0697)	(0.05541)
Square root of number of people under 12 in household	-0.08027**	-0.03039	-0.11979***	-0.11686***	-0.1102***	-0.04395*	-0.14246**	-0.10295*
	(0.03088)	(0.02715)	(0.01808)	(0.02152)	(0.01927)	(0.02126)	(0.04559)	(0.04293)
University attendance net rate in parish	-0.05373	0.20962**	-0.0629		0.18251***	0.17734**	0.47892**	
	(0.07623)	(0.06353)	(0.03785)		(0.03706)	(0.05998)	(0.18259)	

Water provision by water truck		0.17168***				0.20028***		
		(0.04711)				(0.05823)		
R2	0.77657	0.75135	0.69353	0.62719	0.69593	0.63059	0.8007	0.80229
N	878	1010	2566	2154	2314	3008	388	592

Source: Author's computation using 2006 LSMS

Table A2.2 Consumption prediction model for Small Area Estimates 2014-2010

	RA	UA (1)	UA (2)	RC (3)	RC (4)	UC	G	Q	RH(5)	RH (6)	RH (7)	RH (8)	RH (9)	UH (10)	UH (11)	UH (12)
	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)	b(s.e.)
Intercept	4.7	5.23	4.76	5.1	4.61	5.1938	6.77	6.29	5.05	4.16	4.93	5.2	8.19	5.52	5.25	5.06
	(0.24)	(0.2)	(0.4)	(0.2)	(0.32)	(0.18)	(0.34)	(0.64)	(0.22)	(0.39)	(0.24)	(0.44)	(1.57)	(0.63)	(1.04)	(0.35)
Access to higher education	0.42			0.44	1.25	0.2397	0.39	0.34				-0.65	0.3	-1.18	0.68	0.32
	(0.13)			(0.14)	(0.31)	(0.07)	(0.15)	(0.15)				(0.23)	(0.17)	(0.41)	(0.15)	(0.09)
Number of rooms per person	0.22	0.15	0.24	0.15		0.1203	0.23	0.24	0.27	0.16	0.23	0.19	0.13	0.13	0.23	0.18
	(0.03)	(0.04)	(0.04)	(0.02)		(0.01)	(0.03)	(0.03)	(0.05)	(0.03)	(0.04)	(0.04)	(0.04)	(0.06)	(0.03)	(0.02)
D. drinking water from distribution truck		0.66	-0.13		0.22	0.0447										
		(0.39)	(0.04)		(0.07)	(0.02)										
D. drinking water from well	0.04	0.66				-0.0395					-0.2	-0.44		0.32		
	(0.02)	(0.28)				(0.02)					(0.13)	(0.28)		(0.13)		
D. drinking water from public works			0.53		-0.51	-0.1023										
			(0.27)		(0.2)	(0.05)										
D. driking water from public works connected outside building		-0.1	-0.13		-0.08	-0.0713						-0.1	-0.13		-0.06	-0.06
		(0.04)	(0.04)		(0.05)	(0.01)						(0.03)	(0.03)		(0.04)	(0.02)
D. driking water from river or thelike		-0.19	-0.1			-0.1087	-0.41					-0.1	-0.11			
		(0.09)	(0.07)			(0.03)	(0.13)					(0.05)	(0.06)			
D. nuclear family plus extended	0.07		-0.07	0.04		0.0258			-0.09	0.08		0.07				0.03
	(0.02)		(0.04)	(0.02)		(0.01)			(0.05)	(0.03)		(0.03)				(0.02)
D. renting loggings or anthicresis						0.2011		-0.06								
						(0.2)		(0.03)								
D. renting loggings						-0.2456										
						(0.2)										
D. dispose garbage in own land	-0.11	-0.23							-0.13	-0.13						-0.12
	(0.02)	(0.09)							(0.05)	(0.06)						(0.06)
D. dispose garbage by burning				0.07	0.05											
				(0.02)	(0.04)											
D. homeowener		0.07				0.0624	0.03	0.09		0.03					0.16	0.11
		(0.03)				(0.01)	(0.02)	(0.03)		(0.02)					(0.02)	(0.01)
D. hut		0.79		0.39		-0.2434										0.81
		(0.28)		(0.16)		(0.16)										(0.38)
D. electric stove				-0.38			0.17	0.27								
				(0.26)			(0.13)	(0.16)								
D. wood stove	-0.11	-0.22		-0.05	-0.15	-0.115				-0.13	-0.12	-0.09	-0.13			-0.2
	(0.02)	(0.09)		(0.03)	(0.06)	(0.06)				(0.04)	(0.04)	(0.04)	(0.04)			(0.05)
D. house with exclusive kitchen area	0.03			0.07	0.07	0.0519		0.07		0.03	0.05	0.07	0.05	0.13	0.06	0.05
	(0.01)			(0.01)	(0.03)	(0.01)		(0.03)		(0.02)	(0.03)	(0.03)	(0.02)	(0.06)	(0.02)	(0.01)
D. Cuenca															0.17	
															(0.03)	
D. household with room for family business	0.09								0.22	0.09	-0.08	-0.05				
	(0.04)								(0.1)	(0.04)	(0.03)	(0.03)				
D. outhouse toilet	-0.06															-0.17
	(0.02)															(0.05)
D. toilet with septic tank	0.04		0.07		0.06	0.0283								-0.11		0.02
	(0.02)		(0.03)		(0.03)	(0.01)								(0.06)		(0.03)
D. head speaks Spanish and native language	-0.18	-0.11	0.19		-0.24					-0.18			-0.08			
	(0.02)	(0.05)	(0.12)		(0.11)					(0.05)			(0.04)			
D. head agricutural worker		0.11								-0.11	0.2					
		(0.09)								(0.04)	(0.08)					
D. head wage employee		-0.09			-0.07										-0.06	-0.15
		(0.03)			(0.03)										(0.02)	(0.03)
D. head of hh employed in retail industry		0.1					0.05					0.08	0.17			
		(0.05)					(0.03)					(0.06)	(0.06)			
D. head of hh employed in wholesale industry			-0.2	0.2		0.08	0.15					0.39	0.2			
			(0.13)	(0.07)		(0.03)	(0.06)					(0.16)	(0.13)			
D. head of hh works in construction industry					-0.13	-0.0298			0.17	-0.06		0.09				-0.08
					(0.09)	(0.02)			(0.07)	(0.03)		(0.03)				(0.03)
D. head of hh self-employed	0.13					0.0484	0.1		0.22	0.14	0.12	-0.07				-0.05
	(0.04)					(0.01)	(0.03)		(0.09)	(0.03)	(0.04)	(0.04)				(0.03)

D. head of hh employed in directing role	0.17		0.36	0.16		0.2834	0.18			0.31			0.19		0.17	0.15
	(0.09)		(0.16)	(0.09)		(0.04)	(0.1)			(0.13)			(0.09)		(0.07)	(0.05)
D. head employed in hotel-restaurant industry		-0.08	-0.13	0.07						-0.2				-0.39		
		(0.06)	(0.06)	(0.05)						(0.08)				(0.14)		
D. head access to social security	0.22		0.07	0.09		0.1082	0.12	0.04	0.15	0.14	0.09		0.04	0.1	0.06	0.09
	(0.02)		(0.03)	(0.02)		(0.01)	(0.02)	(0.02)	(0.08)	(0.03)	(0.06)		(0.03)	(0.07)	(0.02)	(0.02)
D. head of hh not in labour force		0.12	0.11			0.0596	-0.1	0.13	-0.14					0.15		0.07
		(0.07)	(0.08)			(0.02)	(0.03)	(0.05)	(0.06)					(0.11)		(0.04)
D. head in hh works in manufacturing industry	-0.1		-0.11			-0.0415				-0.14					-0.08	-0.07
	(0.05)		(0.05)			(0.02)				(0.04)					(0.03)	(0.02)
D. head of hh female				-0.07	-0.1	-0.0392							-0.12		-0.1	-0.07
				(0.02)	(0.05)	(0.01)							(0.03)		(0.03)	(0.02)
D. head of hh native american	-0.17							-0.68					-0.29			0.16
	(0.14)							(0.4)					(0.17)			(0.1)
D. head of hh unskilled		-0.08	-0.1	-0.06		-0.0606	-0.05	-0.06			-0.1	-0.14			-0.03	-0.05
		(0.04)	(0.04)	(0.02)		(0.01)	(0.03)	(0.04)			(0.05)	(0.03)			(0.03)	(0.02)
D. head of hh in service industry	0.14						-0.1			-0.35						-0.12
	(0.1)						(0.06)			(0.14)						(0.04)
D. head of hh in fishing industry	0.18			0.19	-0.12		0.15									
	(0.12)			(0.04)	(0.09)		(0.08)									
D. head of hh business owner and chief	0.45	0.18	0.28		0.46	0.2755	0.3	0.31		0.36		0.22	0.28	0.53	0.31	0.22
	(0.08)	(0.07)	(0.06)		(0.11)	(0.02)	(0.06)	(0.05)		(0.1)		(0.08)	(0.07)	(0.16)	(0.05)	(0.04)
D. head of hh domestic worker	0.13					-0.0019	-0.04	0.04	-0.57				0.26			
	(0.1)					(0.04)	(0.09)	(0.03)	(0.27)				(0.15)			
D. head in formal economic sector	0.08								0.19						0.07	0.08
	(0.03)								(0.1)						(0.03)	(0.02)
D. head of hh single	-0.14	-0.13	0.18		-0.1	-0.0872			-0.1	-0.1	-0.23	-0.1			-0.08	-0.09
	(0.04)	(0.05)	(0.06)		(0.07)	(0.02)			(0.07)	(0.04)	(0.07)	(0.05)			(0.04)	(0.03)
D. head of hh works in public sector	0.18							0.08		0.23			0.13			
	(0.1)							(0.04)		(0.14)			(0.06)			
D. head of hh separated/divorced	-0.08		0.1			-0.053	-0.04	-0.12	0.16	-0.1	-0.09					
	(0.04)		(0.05)			(0.02)	(0.03)	(0.05)	(0.09)	(0.04)	(0.07)					
D. head of hh in transportation industry										-0.09	-0.13				-0.13	-0.07
										(0.07)	(0.07)				(0.05)	(0.03)
D. head of hh widow(er)	-0.09					-0.0586				-0.06			0.11		0.07	0.07
	(0.04)					(0.02)				(0.04)			(0.05)		(0.05)	(0.04)
D. head o hh over 65 years of age	-0.09						0.18			-0.08		0.09	-0.05		0.11	
	(0.05)						(0.07)			(0.05)		(0.07)	(0.05)		(0.06)	
D. toilet is letrine			-0.29			-0.051	-0.13									
			(0.17)			(0.03)	(0.07)									
D. house basic prefabricated structure								-0.14			-0.1					
								(0.08)			(0.05)					
D. house has no shower	-0.1	-0.15		-0.1	-0.19	-0.0699			-0.12	-0.1	-0.06		-0.07		-0.09	-0.11
	(0.02)	(0.05)		(0.02)	(0.04)	(0.01)			(0.05)	(0.03)	(0.04)		(0.03)		(0.06)	(0.03)
D. house has no landline telephone	0.11	0.2	0.26	0.1	0.07	0.194	0.14	0.14		0.23		0.13	0.13	0.12	0.17	0.16
	(0.02)	(0.03)	(0.03)	(0.02)	(0.04)	(0.01)	(0.03)	(0.03)		(0.02)		(0.03)	(0.03)	(0.07)	(0.03)	(0.02)
D. house has no electricity connection				-0.13					-0.25			-0.19	-0.48	-0.54	-0.29	
				(0.05)					(0.12)			(0.12)	(0.19)	(0.18)	(0.17)	
D. nuclear family incomplete	0.06	-0.08			0.12	0.0546								-0.1		0.04
	(0.03)	(0.04)			(0.05)	(0.02)								(0.07)		(0.02)
D. house adobe walls				-0.19								-0.07			-0.11	-0.05
				(0.05)								(0.04)			(0.04)	(0.03)
D. house with precarious walls	0.09	-0.2													0.22	
	(0.05)	(0.1)													(0.19)	
D. house ceramic, vinyl or tile flooring	-0.12			0.15	0.23	-0.2177		-0.06			0.2				-0.05	-0.02
	(0.03)			(0.02)	(0.05)	(0.05)		(0.03)			(0.1)				(0.02)	(0.02)
D. househ with precarious floors				-0.06	-0.2	-0.0437						-0.07	-0.15		-0.14	
				(0.03)	(0.11)	(0.03)						(0.04)	(0.04)		(0.08)	
D. house with cement or brick flooring		-0.17	-0.11			-0.3522	-0.14	-0.17		-0.08	0	-0.2	-0.14	-0.14	-0.25	-0.16
		(0.03)	(0.03)			(0.05)	(0.02)	(0.04)		(0.03)	(0.09)	(0.03)	(0.03)	(0.08)	(0.03)	(0.02)
D. house with shared bathroom		-0.11				-0.0432			-0.26							-0.06
		(0.05)				(0.02)			(0.16)							(0.03)
D. house with asbestos roof	0.01				0.2			-0.07					-0.03	0.11	-0.08	

	(0.05)				(0.08)			(0.04)					(0.02)	(0.06)	(0.02)		
D. house with precarious roof	-0.14				-0.08							-0.67					
	(0.05)				(0.12)							(0.41)					
D. house with zinc roof	-0.05	-0.04	-0.11	-0.05	0.11	-0.0972		-0.11		-0.08	-0.08			-0.27	-0.09	-0.06	
	(0.03)	(0.03)	(0.04)	(0.02)	(0.06)	(0.01)		(0.04)		(0.02)	(0.03)			(0.07)	(0.05)	(0.03)	
D. house with wooden roof						-0.057							0.54		-0.11		
						(0.05)							(0.27)		(0.08)		
D. housing in exchange for service or other non conventional agreement															0.22		
															(0.15)		
Schooling of head * D. head of hh domestic worker							0										
							(0)										
Schooling of head * D. head of hh domestic worker_00																0.1	
																(0.07)	
Schooling of head * D. head of hh domestic worker_02				0.7													
				(0.35)													
Schooling of head * D. head of hh domestic worker_05										-0.62							
										(0.29)							
Schooling of head * D. head of hh domestic worker_08															-0.41		
															(0.26)		
Schooling of head * D. head of hh domestic worker_09											-0.37				-0.49		
											(0.36)				(0.22)		
Schooling of head * D. head of hh domestic worker_12			-0.55														
			(0.38)														
Schooling of head*Work experience	0			0	0	0.0001	0			0	0	0	0	0	0	0	0
	(0)			(0)	(0)	(0)	(0)			(0)	(0)	(0)	(0)	(0)	(0)	(0)	(0)
Schooling of head*D. formal sector		0	0			0.0074	0.01			0.01	0.01			0.01			
		(0)	(0)			(0)	(0)			(0)	(0)			(0)			
Schooling of head * D. head of hh in public sector	-0.01	0.01			0.02					-0.02	0.01						
	(0)	(0)			(0)					(0.01)	(0)						
Schooling of head of hh	-0.01		0.01	-0.04			-0.04	-0.03	-0.03	0		-0.04	-0.02	0.02		-0.03	
	(0.01)		(0)	(0.01)			(0.01)	(0.01)	(0.02)	(0.02)		(0.01)	(0.02)	(0)		(0.01)	
Schooling of head ^2	0	0	-0.15	0		0.0009	0	0	0	0	0	0	0		0	0	
	(0)	(0)	(0.07)	(0)		(0)	(0)	(0)	(0)	(0)	(0)	(0)	(0)		(0)	(0)	
Average schooling in census sector					0	0.0213	0.03	0.07				0.04		0	-0.05	0.01	
					(0.02)	(0)	(0.01)	(0.01)				(0.02)		(0.04)	(0.01)	(0.01)	
Years of work experience of head of hh	0	0.01			-0.01	0.0109				0			0				
	(0)	(0)			(0)	(0)				(0)			(0)				
Years of work experience of head of hh^2		0		0	0	-0.0003										0	
		(0)		(0)	(0)	(0)										(0)	
Years of work experience of head of hh^3				0	0	0	0	-0.19		0	0	0			0	0	
				(0)	(0)	(0)	(0)	(0.11)		(0)	(0)	(0)			(0)	(0)	
Proportion in census sector of houses with water connected to public works		-0.13			-0.48	-0.1048					0.19	0.1	-0.07	0.34	0.17	0.13	
		(0.05)			(0.13)	(0.02)					(0.11)	(0.06)	(0.05)	(0.2)	(0.07)	(0.03)	
Proportion in census sector of houses with water disposal connected to public works		0.18				-0.0295				-0.1			-0.11			-0.07	
		(0.06)				(0.02)				(0.04)			(0.05)			(0.04)	
Proportion in census sector of houses with garbage collection service		-0.15	0.16		0.3				0.21	0.07		0.21	0.11		0.26		
		(0.11)	(0.07)		(0.07)				(0.09)	(0.04)		(0.04)	(0.05)		(0.08)		
Mean number of people per rooms in census sector		0.05	-0.09	0.04	-0.05	-0.0289	-0.1					0.07		-0.24			
		(0.03)	(0.03)	(0.02)	(0.04)	(0.01)	(0.02)					(0.03)		(0.06)			
Proportion in census sector of houses with electricity			0.66			0.3621				0.4		-0.86		-0.74	1.53	0.46	
			(0.33)			(0.13)				(0.3)		(0.35)		(0.4)	(0.54)	(0.32)	
Proportion in census sector of houses with concrete, bloque, brick walls		0.2		-0.07	0.16	-0.1268	-0.09	-0.24		0.1						-0.16	
		(0.1)		(0.05)	(0.11)	(0.02)	(0.05)	(0.11)		(0.05)						(0.05)	
Proportion in census sector of houses with wooden, tile, vilyn flooring	0.05			0.1			-0.22	-0.28	0.17	0.1	0.04				-0.48		
	(0.04)			(0.04)			(0.09)	(0.14)	(0.07)	(0.06)	(0.07)				(0.1)		
Proportion in census sector of houses with exclusive bathroom				-0.19									-0.32				
				(0.06)									(0.11)				
Proportion of members of household in workforce	0.27	0.3	0.23	0.1	0.34	0.226	0.12	0.16		0.19		0.19	0.23	0.42	0.12	0.1	
	(0.04)	(0.07)	(0.08)	(0.04)	(0.08)	(0.03)	(0.06)	(0.06)		(0.06)		(0.06)	(0.06)	(0.14)	(0.06)	(0.04)	
Square-root of number of children<12 in hh			-0.05	-0.13		-0.0114			-0.09		-0.1		-0.01	-0.14	-0.04	-0.06	
			(0.04)	(0.02)		(0.01)			(0.04)		(0.03)		(0.03)	(0.06)	(0.02)	(0.02)	
Square-root of number of people in hh	-0.54	-0.62	-0.43	-0.42	-0.55	-0.5122	-0.46	-0.42	-0.31	-0.51	-0.44	-0.5	-0.54	-0.29	-0.46	-0.46	
	(0.02)	(0.04)	(0.05)	(0.03)	(0.05)	(0.02)	(0.03)	(0.04)	(0.06)	(0.03)	(0.05)	(0.04)	(0.04)	(0.09)	(0.04)	(0.03)	

Square-root of number of laking basic needs in hh	-0.06	0.01	-0.1	-0.08		-0.0684	-0.05	-0.07	-0.06	-0.03	-0.05	0.03	-0.07	-0.02		0
	(0.02)	(0.03)	(0.03)	(0.02)		(0.01)	(0.02)	(0.03)	(0.05)	(0.02)	(0.04)	(0.01)	(0.03)	(0.05)		(0.02)
Square-root of number of work hours of head of hh	0.02	0.04	0.02	0.03	0.04	0.0231			0.02	0.01	0.01		0.02	0.04	0.02	0.02
	(0)	(0.01)	(0.01)	(0)	(0.01)	(0)			(0.01)	(0)	(0.01)		(0.01)	(0.01)	(0.01)	(0)
Literacy rate in census sector	0.28			0.56	0.28		-0.92	-1.1			0.27	0.62				
	(0.17)			(0.15)	(0.26)		(0.3)	(0.57)			(0.17)	(0.22)				
Primary school attendance rate in census sector	0.37					0.1631		0.58		0.39			-2.41		-0.97	
	(0.14)					(0.07)		(0.29)		(0.24)			(1.52)		(0.85)	
Secondary school attendance rate in census sector			0.23	-0.2			0.15	-0.21	-0.1	0.1			0.1	0.33		
			(0.11)	(0.04)			(0.07)	(0.08)	(0.11)	(0.05)			(0.06)	(0.17)		
Univserity attendance rate in census sector			0.59	0.14		-0.0476	-0.06	0.05	-0.2	0.14		-0.14				
			(0.17)	(0.05)		(0.03)	(0.05)	(0.04)	(0.15)	(0.09)		(0.08)				
N	2482	825	663	2041	655	6196	1300	1032	357	1442	534	903	1135	248	1089	2775
R2	0.6779	0.7548	0.7123	0.5838	0.7356	0.65	0.7035	0.6729	0.6058	0.5938	0.6302	0.6016	0.6359	0.7764	0.7159	0.7066

Source: Author's computation using 2014 LSMS

Appendix 3 Point estimation and standard errors of Gini coefficients

Table A3.1 2006 Point estimation and standard errors of Gini coefficients estimations for provinces

Region	Provincial code	Gini coefficient	Standard error	Population	Number of HH
Quito (county)	1701	0.422	0.005	1933579	566115
Guayaquil (county)	901	0.401	0.007	1584401	589778
Urban Coast (excluding Guayaquil)	2	0.393	0.009	8766	3465
Urban Coast (excluding Guayaquil)	3	0.391	0.010	25560	8795
Urban Coast (excluding Guayaquil)	5	0.381	0.011	18015	6524
Urban Coast (excluding Guayaquil)	6	0.367	0.009	6220	2308
Urban Coast (excluding Guayaquil)	7	0.389	0.007	327899	119633
Urban Coast (excluding Guayaquil)	8	0.428	0.008	181985	69939
Urban Coast (excluding Guayaquil)	9	0.416	0.008	560022	214528
Urban Coast (excluding Guayaquil)	11	0.385	0.012	8329	3113
Urban Coast (excluding Guayaquil)	12	0.394	0.007	309347	114847
Urban Coast (excluding Guayaquil)	13	0.403	0.007	526870	193795
Urban Coast (excluding Guayaquil)	17	0.368	0.011	4552	1465
Urban Coast (excluding Guayaquil)	20	0.370	0.009	14601	5447
Urban Coast (excluding Guayaquil)	23	0.401	0.007	200708	69863
Urban Coast (excluding Guayaquil)	24	0.403	0.010	144331	49525
Rural Coast	2	0.353	0.009	31831	8387
Rural Coast	3	0.358	0.009	19543	4969
Rural Coast	4	0.370	0.015	6056	1351
Rural Coast	5	0.335	0.008	32110	8105
Rural Coast	6	0.339	0.017	4212	1118
Rural Coast	7	0.332	0.006	141682	39381
Rural Coast	8	0.342	0.006	240296	58969
Rural Coast	9	0.314	0.006	494855	136403
Rural Coast	10	0.345	0.012	8207	1928
Rural Coast	11	0.369	0.009	55279	14722
Rural Coast	12	0.313	0.005	318389	85089
Rural Coast	13	0.330	0.006	562389	144174
Rural Coast	17	0.344	0.007	47164	11460
Rural Coast	20	0.361	0.017	5720	1714
Rural Coast	23	0.337	0.007	85842	21646
Rural Coast	24	0.328	0.007	99719	24786
Rural Coast	90	0.319	0.006	31066	7834
Urban Highlands (excluding Quito)	1	0.371	0.004	273644	95965
Urban Highlands (excluding Quito)	2	0.375	0.005	21967	8391
Urban Highlands (excluding Quito)	3	0.370	0.004	38131	12784
Urban Highlands (excluding Quito)	4	0.362	0.004	58635	19304
Urban Highlands (excluding Quito)	5	0.366	0.004	74060	24665
Urban Highlands (excluding Quito)	6	0.361	0.004	116097	41975
Urban Highlands (excluding Quito)	10	0.380	0.004	187589	62345
Urban Highlands (excluding Quito)	11	0.377	0.004	157668	53480
Urban Highlands (excluding Quito)	17	0.381	0.004	133364	42489
Urban Highlands (excluding Quito)	18	0.356	0.003	163239	55994
Rural Highlands	1	0.400	0.007	306371	87950
Rural Highlands	2	0.444	0.010	96118	26867
Rural Highlands	3	0.401	0.007	100620	30827
Rural Highlands	4	0.386	0.009	76723	22245
Rural Highlands	5	0.426	0.009	235744	62505
Rural Highlands	6	0.421	0.008	256464	77644
Rural Highlands	10	0.420	0.008	136546	36813
Rural Highlands	11	0.403	0.009	141291	42390
Rural Highlands	17	0.435	0.008	358677	99396
Rural Highlands	18	0.385	0.006	278716	81438
Rural Highlands	23	0.382	0.009	8945	2514
Urban Amazon	14	0.397	0.009	26169	8657
Urban Amazon	15	0.397	0.007	22670	7428
Urban Amazon	16	0.391	0.008	30185	10249
Urban Amazon	19	0.378	0.007	16458	5275
Urban Amazon	21	0.379	0.007	49623	17991
Urban Amazon	22	0.394	0.009	38886	13992
Rural Amazon	14	0.560	0.010	81415	24128
Rural Amazon	15	0.524	0.010	59677	14910
Rural Amazon	16	0.540	0.010	33408	9212
Rural Amazon	19	0.491	0.012	53595	15710
Rural Amazon	21	0.485	0.013	83056	24791
Rural Amazon	22	0.511	0.012	64842	17385

Source: Author's computation using 2006 /LSMS

Appendix 4 2006 Housing conditions index

We use the first component as our housing conditions index. The index increases as the living conditions improve.

Table A4.1 Component Matrix Principal Components Analysis

	Component	
	1	2
Dummy houses with a sewage connection	.784	-.228
Dummy houses with public garbage collection services	.765	-.081
Dummy houses with exclusive washroom	.726	-.211
Dummy houses with electricity	.465	.536
Dummy houses with viable walls	.622	.368
Dummy houses with viable floors	.694	.207
Dummy houses with a water connection	.770	-.097
Dummy houses with viable roof	.232	.605
Dummy houses with phone connection	.657	-.287
Dummy houses with overcrowding	-.302	.349

Source: Author's computation using 2006 LSMS

Table A4.2 Total Variance Explained

	Initial Eigenvalues			Extraction Sums of Squared Loadings		
Component	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	3.980	39.803	39.803	3.980	39.803	39.803
2	1.149	11.489	51.293	1.149	11.489	51.293
3	.935	9.352	60.645			
4	.875	8.748	69.393			
5	.766	7.656	77.049			
6	.673	6.726	83.775			
7	.482	4.821	88.596			
8	.448	4.484	93.080			
9	.371	3.713	96.792			
10	.321	3.208	100.000			

Source: Author's computation using 2006 LSMS

Table A4.3 Results of principal component analysis of housing conditions

Sub-region	Mean	N	Std. Deviation
Quito	0.9154	496527	.38313230
Guayaquil	0.4493	541943	.70698084
Sierra Urbana sin Quito	0.7367	437262	.56331707
Sierra Rural	-0.4467	585807	.81424722
Costa Urbana sin Guayaquil	0.2085	632177	.75130373
Costa Rural	-1.0964	434422	.69612512
Amazonia Urbana	0.2571	44380	.80840630
Amazonia Rural	-1.0525	92347	1.05149895
Total	0.1005	3264866	.96987113

Source: Author's computation using 2006 LSMS

Appendix 5 2006 Food consumption index

We use the second factor component of this analysis as a control variable in our models. This factor assigns high values to households with high consumption of carbohydrates such as tubers.

Table A5.1 Component Matrix Principal Components Analysis

	Component					
	1	2	3	4	5	6
Total calories consumed on average (parish)	.658	-.256	.252	.363	-.132	.130
Carbohydrates from cereal: gr per day consumed on average in every parish	.403	-.350	.463	.422	-.149	.362
Carbohydrates from fruit: gr per day consumed on average in every parish	.628	.240	-.225	-.121	-.619	-.129
Carbohydrates from milk and derivatives: gr or ml per day consumed on average in every parish	.665	-.005	-.465	-.302	.152	.314
Carbohydrates from legumes: gr per day consumed on average in every parish	.462	.131	.701	-.502	.056	-.093
Total carbohydrates: gr or ml per day consumed on average in every parish	.714	-.071	.412	.342	-.230	.263
Carbohydrates from tubers: gr per day consumed on average in every parish	.124	.908	.054	.189	.107	.183
Carbohydrates from vegetables: gr per day consumed on average in every parish	.766	.025	-.032	.297	.341	-.369
Fat from meats and derivatives: gr per day consumed on average in every parish	.779	-.029	-.143	.005	.061	-.003
Fat from fruit: gr per day consumed on average in every parish	.746	.160	-.314	-.046	-.420	-.152
Fat from milk and derivatives: gr per day consumed on average in every parish	.691	-.109	-.390	-.406	.207	.333
Fat from fats and oils: gr per day consumed on average in every parish	.268	-.337	.531	.156	-.029	.101
Fat from legumes: gr per day consumed on average in every parish	.431	.225	.702	-.469	.015	-.117
Fat from tubers: gr per day consumed on average in every parish	.061	.937	.042	.223	.112	.188
Fat from vegetables: gr per day consumed on average in every parish	.808	.049	-.117	.247	.312	-.342
Protein from meats and derivatives: gr per day consumed on average in every parish	.779	.024	-.163	.030	.092	-.008
Protein from fruit: gr per day consumed on average in every parish	.725	.209	-.282	-.088	-.551	-.152
Protein from milk and derivatives: gr or ml per day consumed on average in every parish	.686	-.135	-.366	-.411	.209	.325
Protein from legumes: gr per day consumed on average in every parish	.453	.205	.715	-.459	.065	-.088
Protein from fish and seafood: gr per day consumed on average in every parish	.385	-.551	-.002	.100	.147	.060
Total protein: gr per day consumed on average in every parish	.915	-.112	.101	.183	.015	.135
Protein from tubers: gr per day consumed on average in every parish	.042	.928	.037	.226	.110	.184
Protein from vegetables: gr per day consumed on average in every parish	.832	.001	-.091	.215	.293	-.349

Source: Author's computation using 2006 LSMS

Table A5.2 Total Variance Explained

	Initial Eigenvalues			Extraction Sums of Squared Loadings		
Component	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	8.797	38.248	38.248	8.797	38.248	38.248
2	3.458	15.035	53.283	3.458	15.035	53.283
3	3.041	13.220	66.504	3.041	13.220	66.504
4	1.960	8.524	75.028	1.960	8.524	75.028
5	1.443	6.275	81.303	1.443	6.275	81.303
6	1.134	4.932	86.235	1.134	4.932	86.235

Source: Author's computation using 2006 LSMS

Table A5.3 Results of principal component analysis of food consumption

Sub-region	Mean	N	Std. Deviation
Quito	0.5279	424982	.0000000
Guayaquil	-1.0238	541943	.0000000
Urban highlands	0.4880	594103	1.0451720
Rural highlands	1.1804	489298	1.8296341
Urban coast	-1.4320	759663	.4421891
Rural coast	-1.8843	306937	.7794456
Urban Amazon	-0.3765	56187	.6162099
Rural Amazon	-0.3728	67627	.7688065
Total National	-0.3627	3240740	1.4087608

Source: Author's computation using 2006 LSMS

Table A5.4 List of food items and their food groups

Food staple	Category
Rice	Cereals
Barley rice	Cereals
Oatmeal	Cereals
Pasta	Cereals
Cookies	Cereals
Bean flower	Legumes
Corn flower	Cereals
Banana flower	Fruits
Wheat flower	Cereals
Machica	Cereals
Corn y morocho	Cereals
Mote	Cereals
Bread	Cereals
Quinua	Cereals
Lamb meat	Meats and derivatives
Pork	Meats and derivatives
Beef	Meats and derivatives
Cow entrails	Meats and derivatives
Chicken	Meats and derivatives
Chicken piece	Meats and derivatives
Chicken entrails	Meats and derivatives
Sausage	Meats and derivatives
Ham	Meats and derivatives
Mortadela	Meats and derivatives
Wiener	Meats and derivatives
Fresh fish	Fish and seafood
Tuna or sardines	Fish and seafood
Shrimp	Fish and seafood
Clam	Fish and seafood
Chicken egg	Eggs and derivatives
Powder milk	Milk and derivatives
Liquid milk	Milk and derivatives
Formula (baby milk)	Milk and derivatives
Cheese	Milk and derivatives
Yogurt	Milk and derivatives
Vegetable oil	Fats and oils
Pig fat	Fats and oils
Vegetable butter	Fats and oils
Margarine	Fats and oils
Butter	Fats and oils
Avocado	Fats and oils
Banana	Fruits
Lemon	Fruits
Mandarin	Fruits
Apple	Fruits
Passion fruit	Fruits
Melon	Fruits

Blackberry	Fruits
Orange	Fruits
Naranjilla	Fruits
Food staple	Category
Papaya	Fruits
Pineapple	Fruits
Sweet plantain	Fruits
Plantain	Fruits
Watermelon	Fruits
Tomate de árbol	Fruits
Grape	Fruits
Melloco/olluco	Tubers
Potato	Tubers
Beet	Vegetables
Yucca	Tubers
Carrot	Vegetables
Chard	Vegetables
Garlic	Vegetables
Fresh pea	Legumes
Celery	Vegetables
Broccoli	Vegetables
White onion	Vegetables
Red onion	Vegetables
Corn in grain	Cereals
Cabbage	Vegetables
Cauliflower	Vegetables
Cilantro and parsley	Vegetables
Red beans	Legumes
Brown beans	Legumes
Lettuce	Vegetables
Pickle	Vegetables
Pepper	Vegetables
Radish	Vegetables
Tomato	Vegetables
Pepper	Vegetables
Dry pea	Legumes
Corn on cob	Legumes
Dry red beans	Legumes
Dry chickpea	Legumes
Dry brown bean	Legumes
Lentil	Legumes
Sugar	Sugars
Cocoa	Sugars
Chocolate	Fats and oils
Brown sugar	Sugars
Breakfast cereal	Cereals
Condiments	Miscellaneous
Salt	Miscellaneous
Coffee	Miscellaneous
Water	Miscellaneous
Mineral water	Miscellaneous
Powder juice	Sugars
Juice from concentrate	Sugars
Soft drinks	Sugars

Source: Author's computation using 2006 LSMS

Chapter 5

Concluding remarks

Childhood chronic malnutrition affects cognitive development, schooling achievements, potential lifetime income steam, and it has been a persistent condition among Ecuadorian children (Grantham-McGregor, et al., 2000; Granthan-MacGregor, et al., 2007; Walker, et al., 2000; Walker, et al., 2007; Martinez, et al., 2009) (Larrea, 2002; Larrea & Freire, 2002; Freire, et al., 1988). Currently, the Ecuadorian government distributes nutritional supplements to children under 5 (yeas of age) treating the immediate biological cause of malnutrition but with limited success. I propose pre-natal maternal stress affects the post-birth growth trajectory of the child through the effect it has on the intra-uterine environment and, consequently, the epigenetic make of the child (Chapter 2). As a consequence, a social context which enables long periods of chronic stress during pregnancy can have an effect of the growth trajectory of the child (Chapter 4).

In Chapter 2 (An evaluation of Ecuador's public policy to reduce iron-deficiency anemia in children) I evaluate the effect of iron supplements on children's (6-59 months) Hb levels in Ecuador. The policy stipulates that children up to the age of 59 months are eligible for the treatment. Children over this age are no longer eligible. I use the 2012 cross-section national health and nutritional survey (HNS) and apply (1) a fuzzy RD model where age is the cut-off, and (2) an IV model where the age cut-off is the instrument. The survey lacks data on dose compliance, therefore, the initial randomization of assignment is effectively being used to estimate the effect of the intention to treat (ITT). In this way the causal effect of the policy is identified. I propose the long term effects of pre-natal stress shocks as a possible root cause of malnutrition.

Despite specifying various bandwidths and functional forms, I find no significant effect of the change in the treatment policy in any of the RD or IV models. However, when including heterogeneous effects by quantile of Hb in the IV model, a negative significant effect in the first quantile and a positive significant effect in the second quantile is found. I suspect that these two opposing effects cancel each other out when the average effect is measured. However, this leaves us with the important question of how to explain the negative significant effect. The only explanation I can give is that the treatment can cause constipation or diarrhea, particularly among younger children who have never had "sprinkles" before (Ministerio de Salud Publica, World Food Program, 2011), which can actually cause a deterioration of the outcome, if the illness is not addressed and continues or if the treatment is interrupted. Therefore, the effect of the treatment policy might actually reduce Hb if the children become sick and stop receiving doses.

Unfortunately, it is not possible to measure heterogeneous effect in the fuzzy RD model as this would reduce the sample size significantly. Additionally, increasing the bandwidth of the fuzzy RD model to the point where the sample size would be acceptable would imply not having a bandwidth which is methodologically equivalent to using an IV model (Calonico, et al., 2014).

Despite its limitations, this study is an important contribution to the literature on the nutritional health of children. Most of the literature on the nutritional outcomes of children in Ecuador evaluate cash transfer programs (Ponce & Bedi, 2010; Leon & Younger, 2007; Fernald & Hidrobo, 2011; Schady, 2012) or the effects of the Ecuadorian 1999 financial crisis or of exclusive breastfeeding practices (Hidrobo, 2014; Carranza Baron & Mendez Sayago, 2014). Additionally, most of the studies mentioned above use the z-score of height for age as the outcome variable. This is the only study, to our knowledge, to evaluate a nutritional supplement transfer program in Ecuador and to use Hb as the outcome variable.

In Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador) I 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. I use a sharp RD 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. This allows us to create an appropriate counter-factual in terms of (un)observable characteristics as the cut-off point is an exogenous unanticipated shock.

We find a significant deleterious effect of this shock on the z-scores of height-for-age in 2012. 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 through the life cycle (Almond & Currie, 2011; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

Throughout Chapter 3 I provide evidence of a robust unanticipated effect. I demonstrate that relevant observables do not determine selection into treatment, I use data-driven methods to select an appropriate bandwidth and polynomial order, and, I test results' sensitivity to kernel functional forms (Cattaneo, et al., 2018; Lee & Lemieux, 2010). Additionally, I test for placebo effects; examine how the density of the running variable affects the outcome, and, see if other observables have the same cut-off.

This Chapter (3) contributes to the literature in three ways: (1) I measure the effects of a financial crisis, while the literature on the contextual variables affecting fetal development are usually limited to famine, natural disasters and terrorist attacks. (2) I 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) I provide a method that attempts to identify causal effects and provide a theoretical biological pathway between the treatment and the outcome.

Notwithstanding, there are various challenges that were tackled with the evidence presented in this paper. Mainly, I assume the cut-off is deterministic in increasing stress levels while, if stress can be caused by other unobservables which are not possible to control for, the relationship should be probabilistic. I 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. Secondly, despite testing and not finding any anticipation effects, I do find isolated significant placebo effects although they do not hold up to robustness checks. Thirdly, there is an imbalance in the size of the samples, however, this does not seem to affect the outcome. Finally, despite our attempts, I am unable to test whether individuals with no access to financial services were effectively sheltered from the crisis.

This Chapter (3), in testing of the effect of pre-natal maternal stress, is the axis on which the rest of the thesis rotates both theoretically and empirically. The results from this chapter provide an explanation for the mixed effects of the treatment in Chapter 2 (An evaluation of Ecuador's public policy to reduce iron-deficiency anemia in children) and an empirically tested theoretical framework for the pathway connecting inequality to individual child health in Chapter 4 (Inequality and Malnutrition in Ecuador).

In Chapter 4 I measure the effect of inequality on stunting in children (age<5 years) by regressing the provincial, county and parish Gini coefficients against both the z-score of height-for-age using IV models where the proportion of households who suffered a draught or natural disaster in the last year is the instrument for the Gini coefficient. I present two iterations of the models: (1) using the 2006 LSMS, (2) using the 2014 LSMS. I find the Gini coefficient has a significant deleterious effect in 2006 but not in 2014. I argue inequality affects pre-natal maternal stress causing stunting later in life and I believe the effect might

be eroded in 2014 because the variance of the Gini coefficients in 2014 might have shifted in a way in which is it no longer having an effect. Therefore, the results are unconclusive.

Inequality can affect child growth patterns through the effect it might have on pre-natal maternal stress. As stated in Chapter 3 (Long term effects of pre-natal exposure to maternal stress: Evidence from the financial crisis in Ecuador) chronic amounts of stress such as those fostered by a relatively unequal societies have the potential to change the fetal environment and the epigenetic make of the child. However, the effect may be found, only beyond a threshold level of "high" inequality and may be eroded once inequality is reduced below this threshold (Subramanian & Kawachi, 2004) (Almond & Currie, 2011; Gluckman, et al., 2005; Couzin, 2002; Rice, et al., 2010; Rice & Thapar, 2010; Zijlmans, et al., 2015; Bussières, et al., 2015; Hobel, et al., 2008; Schetter & Tanner, 2012; Currie & Rossin-Slater, 2013) (Dancause, et al., 2011; Hilmert, et al., 2016; Tong, et al., 2011; Harville & Do, 2016; Leppold, et al., 2017; Lederman, et al., 2004; Eskenazi, et al., 2007; Maslow, et al., 2016; Wainstock, et al., 2013; Camacho, 2008) (Novak, et al., 2017; Eiríksdóttir, et al., 2013; Stein, et al., 1975; Hoek, et al., 1998; St Clair, et al., 2005; Kannisto, et al., 1997; Barker, 1990; Holzman, et al., 2001; Barker & Osmond, 1986; Barker, 1995) (Beydoun & Saftlas, 2008; Mansour & Rees, 2011; Camacho, 2008; Class, et al., 2011; Zhu, et al., 2013; Gunnlaugsson, 2016; Eiríksdóttir, et al., 2015; Stanner, et al., 1997).

The main limitation is the way I measure the Gini coefficient over small areas. The *small area estimates* model depends heavily on a degree of heterogeneity which cannot be controlled for methodologically and cannot be guaranteed empirically. The efforts made to divide the country into homogeneous regions may abate this limitation, however, it was not possible to generate Gini coefficients which are not systematically under-estimated (Tarozzi & Deaton, 2009).

Additionally, I build a consumption model on 2006 household behavior and simulate it onto the 2010 census, as well as, a consumption model on 2014 household behavior and simulate that onto the same 2010 census. This might be limiting as, in order to use the 2006 estimated parameters (correlation coefficients) to simulate consumption using the population characteristics of 2010, it must be assumed there is very little change in behaviours between 2006 and 2010. This same assumption must be made when the 2014 parameters are used to simulate consumption on the 2010 census. However, this cannot be true as the resulting Gini coefficients are fundamentally different. Therefore, there may be changes in household behaviours which are not reflected in either simulation. Obviously, it is impossible to use the 2020 census to simulate the 2014 data as it has not been constructed or released yet, however, it might be interesting the replicate the study once it does in order to observe the changes in the outcome and how they affect stunting.

This thesis is an original contribution to the field of development economics and to the understanding of the persistence of chronic malnutrition in Ecuador. I provide evidence of the link between consumption inequality and individual health, I test the proposed epigenetic mechanism directly identifying its causal effect and finally use it to explain the possible ways in which the public policy to reduce malnutrition is failing.

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