BLADIMIR CARRILLO BERMUDEZ

THREE ESSAYS ON HEALTH ECONOMICS

Tese apresentada à Universidade Federal de Viçosa, como parte das exigências do Programa de Pós-Graduação em Economia Aplicada, para obtenção do título de *Doctor Scientiae*.

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A Deus, e a toda a minha família.

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CONTENTS

ABSTRACT

CARRILLO BERMÚDEZ, Bladimir, D.Sc., Universidade Federal de Viçosa, April, 2017. **Three essays on health economics**. Advisor: José Gustavo Féres.

This thesis studies three topics on health economics using Brazilian data. In the first essay, we study the effects of low birth weight on infant mortality. Understanding such a relationship is important for the development of policies aimed at reducing the incidence of infant mortality. However, it is little known about this topic in developing countries and estimates from rich economies may have limited external validity to the developing world. Our empirical strategy to isolate the effect of low birth weight from other determinants of infant mortality exploits within-twin variation. The results indicate that lower birth weight babies exhibit higher rates of mortality within one year of birth. The effects are much larger than those derived from the US and Norwegian context. The second essay provides the first estimates of the local externalities of deforestation in infant health. The burning of forest releases a wide range of contaminants, some of which are known to be hazardous for health. Traditional estimates of the costs of deforestation rarely incorporate the health effects of pollution generated by deforestation. For identification, we use the large and heterogeneous reductions in deforestation across sites in the Brazilian Amazon generated by a conservation policy. The findings suggest that deforestation control policy led to reductions in the incidence of low birth weight and prematurity. This is especially true for boys. Collectively, these results provide additional justification for controlling deforestation. Finally, the third essay estimates the effect of increased supply of physicians on child hospitalizations. It is a widely held position that spending resources on increasing the availability of physicians is an effective way to improve child health. However, there is very little rigorous investigation documenting the quantitative importance of physicians for child health. Our empirical strategy exploits a dramatic increase in the number of physicians generated by the Mais Medico program to fill this gap. The results suggest that program implementation is associated with statistically significant reductions in avoidable, ambulatory sensitive hospitalizations only in poor areas. Our estimates indicate that policy lead to a fall of 4.8 percent in avoidable child hospitalizations. Together, the three essays contribute to a better understanding about

the causes of poor health in early life using data from Brazilian, a rapidly emerging economy.

RESUMO

CARRILLO BERMÚDEZ, Bladimir, D.Sc., Universidade Federal de Viçosa, abril de 2017. **Três ensaios sobre economia da saúde.** Orientador: José Gustavo Féres.

Esta tese estuda três tópicos em economia da saúde usando dados do Brasil. O primeiro ensaio investiga o efeito do baixo peso ao nascer sobre a mortalidade infantil. Entender esta relação é importante para o desenho da política que tem como objetivo reduzir a taxa de mortalidade infantil. Porém, é muito pouco conhecido sobre este tópico em países em desenvolvimento e as estimações de países ricos poderiam pouca validade externa para as economias em desenvolvimento. A estratégia empírica para isolar o efeito do baixo peso de outros determinantes da mortalidade infantil usa variação entre irmãos gêmeos. Os resultados indicam que os bebes que nascem com baixo peso têm maiores taxas de mortalidade no primeiro ano de vida. Estes efeitos são maiores que aqueles de estudos para Estados Unidos e a Noruega. O segundo ensaio fornece as primeiras estimações das externalidades locais do desmatamento na saúde infantil. A queima de floresta libera uma grande variedade de contaminantes, alguns dos quais são conhecidos como perigosos para a saúde. Estimações convencionais dos custos do desmatamento raramente incorporam os efeitos na saúde da poluição gerada pelo desmatamento. Para identificação econométrica, usam-se as grandes e heterogêneas reduções no desmatamento ao longo da Amazônia brasileira gerada por uma política de conservação. Os resultados principais indicam que esta política levou a reduções na incidência do baixo peso ao nascer e a prematuridade. Estes resultados são maiores para meninos. Conjuntamente, estes resultados fornecem justificativas adicionais para controlar o desmatamento. Finalmente, o terceiro ensaio estima o efeito de um aumento na oferta de médicos sobre as hospitalizações em crianças. É uma posição amplamente difundida que gastar recursos em aumentar a disponibilidade de médicos é uma maneira eficaz de melhorar a saúde infantil. Porém, há muito pouca investigação cuidadosa documentando a importância quantitativa dos médicos na saúde das crianças. Este estudo aproveita um aumento drástico no número de médicos induzido pelo programa Mais Médicos para preencher esta lacuna. Os resultados indicam que a introdução desse programa está associado a reduções estatisticamente significantes nas hospitalizações sensíveis à atenção primaria somente em áreas pobres. As estimações indicam que a política levou a uma queda de 4.8 por cento neste tipo de hospitalizações em crianças.

1. Introduction

A large literature on the long-term consequences of poor early health on human capital formation has prompted fervent debates regarding the causes of infant health and policies aimed at ameliorate inequalities originated in early life. The understanding of specific causes is necessary for prevention and cost-effective policy design. While this body of work has advanced our understanding about specific causes of poor early health, there is very little investigation on this topic in developing countries. As emphasized by Currie and Vogl (2013), research on the causes and consequences of early health insults has much policy relevance in poorer countries, but precisely measured infant health data are rare in large sample surveys from these countries. While public policy might rely on estimates that are derived from developed countries to poorer ones for cost-benefit assessments, there is a number of reasons to believe that these relationships may differ in most less-developed parts of the world. Indeed, the causes and consequences of poor early health may depend on non-linearities in the production function for child quality, financial constraints and parental education, all of which vary with economic development.

This thesis presents three essays aim to address these issues using data from Brazil. In the first essay, we will study the effects of low birth weight on infant mortality in Brazil. It is important for a number of policy reasons to know how birth weight affects infant health, and whether there are public policies that might act to mitigate the adverse consequences of low birth weight. In the absence of a well-functioning public health system and the presence financial constraints, the capacity to remediate health shocks may be simply more limited in poor countries, which would imply that birth weight might have a larger overall health impact in these economies. Moreover, one may observe different effects if there are non-linearities in the production function for child health or if there are interactions between birth weight and environmental factors (ALMOND; MAZUMDER, 2013; YI et al., 2015). The primary contribution of this essay is to use administrative data on the universe of births linked to death records and exploit a within-twin identification strategy to provide credible evidence on the causal effect of low birth weight on infant mortality in Brazil.

The second essay investigates the externalities of a deforestation control policy on infant health in the Brazilian Amazon region. The externalities of deforestation are an increasingly salient issue in the debate about the costs and benefits of conservation policies. The smoke from the burning of forest contains a wide range of contaminants, including fine particulate pollution (PM10), sulfur oxide (SO2) and carbon monoxide (CO), some of which are known to be hazardous for health. Many previous studies show that exposure during pregnancy to air pollution is associated with increased risk of poor health outcomes at birth (CURRIE; NEIDELL; SCHMIEDER, 2009; GLINIANAIA et al., 2004; GUYATT; SNOW, 2004). Despite this, traditional estimates of the costs of deforestation rarely address local externalities such as the health effects of deforestation. For identification, our research design exploits large reductions in deforestation generated by a conservation policy in Brazil. This essay overcomes several methodological difficulties in literature. In particular, our focus on infants mitigates the problem of unknown lifetime exposure by the low migration rates of pregnant women.

The final essay provide evidence of the effects of a large-scale intervention in Brazil, namely "Programa Mais Medico" (PMM), on child health measured per hospitalizations. This program aimed to place physicians in areas with shortage of doctors. To attract foreign and Brazilian newly graduated physicians, the program offered exceptional and flexible conditions for participation. For example, a foreign physician did not need to have a registered diploma in Brazil for enrollment. All doctors enrolled in the program were placed in Basic Health Units (UBS), where families have free access to primary health care services such as medical consultations, vaccines, dressings and health counseling to provide prevention and early detection of diseases.[1] This policy led to an unprecedented and immediate increase in the number of physicians willing to provide primary health care. This essay addresses the absence of direct empirical evidence on the role of policies of this sort outside the United States, especially in poor countries. Even within the United States, there is a limited number of studies concerned with the effects of increasing the number of physicians on child health. The only study examining direct measures of child health is that of Currie,

[1] The Basic Health Units (UBS) is a health facility where primary care teams provide health basic services. The UBS are usually within neighborhoods and cover a certain region, are close to where people live.

Griber and Fischer (1995), who examine the effect of increasing the availability of physicians serving to poor on infant mortality. While the focus on infant mortality is compelling, this is a relatively extreme outcome that reflects some of the most severe consequences of inadequate access to health care.

Together, the three essays contribute to a better understanding about the causes of poor health in early life using data from Brazilian, a rapidly emerging economy. The ordering of the essays was chosen to reflect a logical development. In the first essay, we study some of short-run consequences of poor early health measured by low birth weight on health, whereas the third essay investigate the impact of a policy that may act to mitigate the consequences of poor early health. The first essay suggests that lower babies have increased risk of death within one year, estimates are generally larger than those estimated with data from the US and Norway. This confirms that applying estimates that are derived from these rich countries to developing economies may be misleading for cost-benefit assessments of policy. In turn, the second essay indicates that a deforestation control policy implemented in Brazil had positive externalities in infant health through reductions local air pollution. In the third essay, we find evidence that increasing the number of physicians providing primary health care lead to reductions in child hospitalizations, but only in poor regions. Collectively, the three essays indicate that policies of this sort may have positive long-run impacts on human capital formation in Brazil.

2. Low Birth Weight and Infant Mortality: Lessons from Brazil

Abstract

Governments devote considerable resources on reducing the incidence of low birth weight with the reasoning that low birth weight is the cause of poor infant health. Much of what we know on the causal link between these variables comes from developed countries. However, these estimates may have limited external validity to the developing world if families with more resources are better able to remediate the effects of poor neonatal health or if there are non-linearities in the production function for child health. In this article, we estimate the relationship between birth weight and infant mortality using data from Brazil. Using a within-twin identification strategy, we document that lower birth weight babies exhibit higher rates of mortality within one year of birth. The effects are much larger than those derived from the US and Norwegian context. We also find that the effects are largely reduced when local sanitation coverage is high, suggesting that access to public health infrastructure may mitigate the consequences of low birth weight.

Keywords: Health human capital; health endowments at birth; Brazil; Twins.
JEL Classification: H51, I12, I18

2.1. Introduction

It has been widely believed that malnutrition *in utero*, commonly proxied by low birth weight, is an important contributor to poor infant health.[2] As a result, governments and international agencies have devoted considerable resources on preventing low birth weight. In India, for example, the World Bank allocated over US$100 million for a program aimed at cutting in half the incidence of low birth weight.[3] Preventing low birth weight has been also a major motivation for nutritional programs and maternal smoking campaigns worldwide.[4] The strong and well-documented association between low birth weight and infant health has led to the position that the social returns of these investments are large. Numerous studies indicate that low birth weight babies have

[2] Low birth weight is conventionally defined as a birth weight less than 2,500 grams.

[3]This is the Second Tamil Nadu Integrated Nutrition Project. The program also had other goals, such as improving nutrition and health status of children0-72 months (THE WORLD BANK, 1998).

[4] In the United States, for example, a motivation for the Medicaid expansion to pregnant women was the reduction of the incidence of low birth weight (CURRIE; GRUBER, 1996).

increased risk of death within one year of birth, and that who survive infancy are likely to suffer from a number of health and developmental difficulties, some of which are known to negatively affect acquisition of human capital.[5] Understanding whether low birth weight is in fact the cause of poor infant health and not simply a correlate of such problems is crucial for guiding the targeting of policies intended to reduce inequalities by improving early life health.

The challenges with uncovering the causal effect of birth weight are well known in the literature. A strong correlation between low birth weight and infant health may be the product of unobserved factors because the determinants of nutrition during pregnancy, including family background and parent's knowledge about health care, are also likely determinants of infant health. So any attempt to ascertain the importance of birth weight for infant health by simply looking at their correlation, or equivalently estimating a simple ordinary least square (OLS) regression, is unlikely to provide convincing evidence. Studies within economics have overcome these challenges using rich data from the United States and Norway, and within-twin identification strategies (ALMOND; CHAY; LEE, 2005; BLACK; DEVEREUX; SALVANES, 2007; OREOPOULOS et al., 2008). These studies suggest that low birth weight leads to increased risk of mortality, although the effects are much smaller than those derived from cross-sectional regressions. This body of research even suggests that low birth can have long-lasting effects on human capital accumulation, which in turn has been interpreted as evidence consistent with the literature emphasizing that early health conditions are a major determinant of individual capabilities.[6] For example, Figlio et al. (2014) illustrate that birth weight has negative effects on cognitive development, while that Black, Devereux, and Salvanes (2007) show that low birth weight babies exhibit reduced earnings, lower educational attainment, and worse health outcomes as adults.

While these studies have undoubtedly advanced our understanding of the effect of birth weight on infant welfare, we know fairly little about this relationship in

[5] Previous studies have shown, for example, that low birth weight is associated with health problems such as cerebral palsy, deafness, epilepsy, blindness, asthma, and lung disease (BROOKS et al., 2001; KAELBER; PUGH, 1969; LUCAS; MORLEY; COLE, 1998; MATTE et al., 2001).

[6] See Conti and Heckman (2010), Cunha, Heckman and Schennach (2010), Cunha, and Heckman (2007, 2008, 2009) for a theoretical discussion about the role of early health conditions in the accumulation of human capital.

developing countries. As emphasized by Currie and Vogl (2013), research on the consequences of early health insults has much policy relevance in poorer countries, but precisely measured birth weight data are rare in large sample surveys from these countries. Thus, it is very little known about whether the effects of birth weight vary at different economic development contexts. In the absence of a well-functioning public health system and the presence financial constraints, the capacity to remediate health shocks may be simply more limited in poor countries, which would imply that birth weight might have a larger overall health impact in these economies. Moreover, one may observe different effects if there are non-linearities in the production function for child health or if there are interactions between birth weight and environmental factors (ALMOND; MAZUMDER, 2013; YI et al., 2015). In consequence, estimates derived from rich countries may not be externally valid to the developing country context.

Many of the existing studies for developing countries are in the epidemiological literature. These studies has relied on cross-sectional estimates while controlling for parents' background characteristics. However, this empirical strategy might be subject to omitted variable bias from unobserved factors that can affect both birth weight and infant health. Furthermore, these studies are generally based on small and non-representative samples, making it the results difficult to generalize and limiting the development of clear stylized facts. Remarkably, research in the economic literature that aims to have a more causal and general interpretation of the relationship between birth weight and infant health in a developing country context is rare. To the best of our knowledge, only McGovern (2014) investigates the effects of birth weight on infant health in developing countries. He uses data from the Demographic and Health Surveys (DHS), which is conducted in more than 90 countries worldwide. However, the use of self-reported information on birth weight is likely to suffer from measurement error that may not be random. Most people in developing country rural areas, especially in sub-Saharan Africa, do not give births in hospitals, so birth weight is likely to be badly measured. Moreover, the use of these surveys does not allow excluding twin pairs with congenital defects and Almond, Chay and Lee (2005) show that it can lead to severe bias.

In this paper, we provide estimates of the effect of birth weight on infant mortality using administrative data on the universe of births linked to death records in

Brazil. As we describe in more detail in section 2.2, these matched data provide comprehensive information on birth weight, congenital defects, date and cause of death, and mother's background characteristics. With these rich data, we follow 19 million singletons and 300,000 pairs of twins from birth through the first year of life. The enormous sample size from this dataset gives us a strong statistical power to discern patterns. For identification, we take advantage of quasi-random variation in birth weight within twin pairs, as described in section 2.1. Using precisely measured birth weight data in a large nationally representative sample and a within-twin identification strategy, we provide what we believe is the most credible evidence on the causal effect of birth weight on infant health in a developing country context.

We document that lower birth weight babies exhibit higher rates of mortality within one year of birth. Our estimates imply that very low birth weight babies have 4 percentage points higher risk of death within one year. The mortality effects are concentrated on conditions originating in the perinatal period, which include respiratory and cardiovascular disorders specific to the perinatal period, and hematological disorders of fetus and newborn. In line with earlier studies for developed countries, the cross-sectional estimates tend be substantially larger in magnitude than the ones derived from the twin-fixed effects estimator. This confirms that policy designs based on cross-sectional estimates may exaggerate the benefits of reducing the incidence of low birth weight.

We then compare our estimates to those derived in the US and Norway. Specifically, we compare our estimates to Almond, Chay, and Lee (2005) and Black, Devereux and Salvanes (2007). In general, our estimates are larger in magnitude than those derived from these studies. The differences are substantial. For example, our estimates are about two times larger than those reported by Almond, Chay, and Lee (2005) for the United States. We argue that these results cannot be explained by specific features of our empirical setting, such as measurement error and the possibility of selection bias induced by miscarriage or stillbirth. A more plausible interpretation of these results is that developing and developed countries have a very different causal relationship between birth weight and infant mortality. Although it is difficult to make causal claims on the specific reasons behind these differences, we assess whether related explanations that are more common to developing countries, such as low

parental education, might be plausible candidates. Our results indicate that the effects of birth weight are stronger for infants born to mothers who have low educational attainment and are unmarried. The effects generally increase by 5 to 71 percent relative to infants born to more advantaged families. We also find that the effects of birth weight are smaller for families residing in municipalities with sanitation coverage over 85 percent. For these families, the impacts falls by 41 to 83 percent, which suggests that birth weight may be interacting with environmental factors. Taken together, we conclude tentatively that applying estimates that are derived from the US or Norway to developing countries may be misleading for cost-benefit assessments of policy.

The rest of the paper is organized as follows. In the next section, we describe our estimation strategy and the data used. Section 3 presents our main results, including robustness checks and a comparison of our estimates to those derived in the US and Norwegian setting. Section 4 explores different forms of heterogeneity in the impacts of birth weight on infant mortality. Finally, section 5 concludes.

2.2.Empirical approach and data

2.2.1. Identification strategy

The goal of the empirical analysis is to estimate the effect of birth weight on infant death. Following Almond and Lee (2005) and Black, Devereux, and Salvanes (2007), let:

$$Death_{ijt} = \alpha + \beta bw_{ijt} + x'_{jt}\delta + \mu_{jt} + \varepsilon_{ijt} \qquad (1)$$

The variable $Death$ is the probability of death within one year of life of the infant *i* born to mother *j* in year *t*. The variable *bw* is birth weight; x is a vector of mother's characteristics, including education, age at birth and marital status; μ_{jt} is a set of unobservable that are mother- and birth-specific, such as family background, the quality of prenatal, genetic factors, and mother's knowledge or awareness about health care; and ε_{ijt} is an idiosyncratic error term assumed orthogonal to other terms in the equation.

The parameter of interest for policy is β. If it is negative and large in magnitude, then targeting interventions during the in utero period to prevent low birth weight may

yield high returns. OLS estimates of the equation (1) that ignore μ_{jt} will be likely biased because many factors in μ_{jt} are also determinants of birth weight. For example, the quality of parent's education is likely to affect both prenatal and postnatal investments. Therefore, any OLS estimate of β would need to be a combination of omitted variable bias and the causal effect of birth weight. To isolate the effect of birth weight from unobservable factors, we use a twin-fixed effects estimator. This approach compares the probability of death of twin *i* to twin *k*, who were born to the same mother but had different levels of birth weight. Including twin-fixed effects is equivalent to estimating the following equation:

$$Death_{1jt} - Death_{2jt} = \beta\left(bw_{1jt} - bw_{2jt}\right) + (\varepsilon_{1jt} - \varepsilon_{2jt}) \qquad (2)$$

Where "1" refers to the first-born twin and "2" refers to the second-born twin. Note that the use of the equation (2) will produce consistent estimates since the mother- and birth specific component is differenced out and ε_{ijt} is assumed independent of birth weight. To see this, it is important to understand the mechanisms by which nearly all twin pairs differ in the birth weights. As discussed by Almond and Lee (2005) and Black, Devereux, and Salvanes (2007), the differences in birth weight could be the result of differences in nutritional intake induced by different umbilical cord insertion points within the placenta and different positions in the womb (PHILLIPS, 1993; ZHANG; BRENNER; KLEBANOFF, 2001). As parental control over these factors is limited, it becomes plausible the identifying assumption that within-twin differences in birth weight are exogenous.

Our main results are based on the equation (2). To account for gender differences in birth weight, we also include an indicator variable of infant's sex as a control in all regressions. There is no a priori justification for using a determined functional form. We compare the explanatory power of specifications that use either birth weight, log(birth weight), and a set of dummy variables for discrete birth weight categories (i.e, 1,500, 1,500-2,500 and 2,500-3,000 grams). This exercise reveals that the specification using dummy variables provides the best fit. Therefore, we use these birth weight categories as the independent variables of interest in our analysis.

2.2.2. Data and summary statistics

Our empirical analysis requires data on birth weight and infant mortality. We use microdata from the Brazilian National System of Information on Birth Records (SINASC) and the National System of Mortality Records (SIM). The SINASC provides information on all births in Brazil since 1994, although it did not cover most of the municipalities before 1998. The data includes the exact date of birth, weeks of gestation, sex, birth weight, and maternal characteristics such as marital status, age and education. The death certificates from the SIM provide comprehensive information on date and cause of death, birthdate, race, and gender, and mother's characteristics (education, marital status and age) are also provided for individuals who were under one year of life at death. Municipal governments are responsible for collecting all death certificates and sending them to the Health Ministry of the Government of Brazil, which consolidates finally the information in the SIM database. The laws governing the collection of the death certificates are national and no burial can be performed without a death certificate. The SIM covers over 96 percent of all annual deaths inferred from demographic census.[7]

We linked death certificate information for the infants who die in their first year of life to the SINASC database by using unique personal identifiers provided by the Health Ministry. The unique personal identifiers are available for births occurring from 2006 through 2012. During this period, there were 20,265,131 births, of which 1.9 percent were twins. The matching rates are nearly constant across time and States. About 70 percent of the infant death records are matched to one of the birth records. The matching rate is not 100 percent because the unique personal identifiers are missing for some infants in the death records. The matching rate is notably higher for twin births, approximately 80 percent. This is reassuring because our main analysis relies on the sample composed by twins. The fact that some infant deaths are not matched to the birth certificate records will introduce measurement error in our dependent variable. We discuss the implications of this issue in section 3.2.

Since the unique personal identifiers allow only identifying individual births, infants who were born to the same mother cannot be directly inferred. We exploit the

[7] Information on coverage of the deaths from SIM are available at http://tabnet.datasus.gov.br/cgi/sim/dados/cid10_indice.htm.

fact that multiple birth records are generally located next to each other in birth certificate files to construct twin-pairs codes. First, we identified all these adjacent twin pairs in our data. Second, we consider that a given adjacent twin pair is part of the same twin set if pregnancy characteristics are identical. Specifically, adjacent twin pairs are considered as born in the same mother if they have identical information for the following set of covariates: hospital of birth, exact date of birth, gestational age, mother's age, mother's education, mother's marital status, and municipality of residence. Approximately 90 percent of the 308,010 adjacent twin pair records have identical information for these variables. We restrict the sample to these twin births, although the results are extremely similar when used all adjacent twin pair records.

In total, there are 276,268 twin births in our sample. We exclude twin pairs where either twin was born with a congenital defect (about 7 percent), as differences in birth weight that are driven by this condition may introduce bias. Twin pairs where either twin had missing information about sex or birth weight are also excluded from the analysis. This restriction results in dropping about 0.1 percent of the sample. Our final sample consists of 255,362 twin births. While our main analysis focuses on the twin sample, we also present results for singletons. There are 18,929,949 singletons with non-missing information for birth weight and infant's sex.

Table 1 presents basic descriptive statistics splitting the sample between twins and singletons. It is apparent that there is differences between the two populations. Indeed, twins are more likely to be born with a weight less than 2,500 grams, have higher rates of prematurity, and are more likely to die within one year of birth than singletons. The differences are large. For example, the probability of dying in the first year is 4.5 times higher for twins than for singletons. These differences also suggest a negative relationship between birth weight and infant mortality. Since low birth weight is also the result of prematurity, it is difficult to establish in principle from these cross-sectional comparisons either whether birth weight or prematurity is the responsible for the increased rates of infant mortality among twins. As Table 1 shows, there are also substantive differences in mother's characteristics between the two groups. In general, twinning probabilities seem to be higher among advantaged families. Indeed, mother of twins are more likely to be older, more educated and more likely to be married. It is well known that the use of fertility treatments, such as in vitro fertilization pre-embryo

transfer, can increase the likelihood of multiple births.[8] Since these treatments are costly or provided by private health insurance, families with more resources may be more likely to use them and consequently parents' background characteristics could be systematically related to the incidence of twin births.[9] This fact calls into question the external validity of the analysis from twins. Despite these dissimilarities between the two populations, we provide suggestive evidence that the results from twins may be generalizable to the general population. In particular, we show that the pooled cross-sectional estimates for the twin population are remarkably similar with that for the singleton population.

Because our statistical approach relies on within-twin variation, we confirm that there is substantial within-twin variability in birth weight and mortality outcomes. Table 2 and Figure 2 show the distribution of the twin birth weight-difference. The mean birth weight difference is 276 grams, or 11 percent of the average twin's birth weight. The data also indicate that 60 percent of twin pairs exhibit a birth weight difference higher than 260 grams, and 10 percent have a birth weight difference higher than 600 grams. In Table 3, which reports mean squared errors from regressions with either birth weight or mortality outcomes as dependent variables, we explore in more detail the sources of the variation in both outcomes. Columns (2) reveals that gestational age explains over half of the overall variance in birth weight. This is consistent with prior literature indicating that gestation length plays a critical role in intrauterine growth (KRAMER, 1987). Despite the significant contribution of gestation length to variation in birth weight, there are great deal of variation that is due to within-twin differences. Indeed, column (3) shows that 20 percent of differences of the birth weight variation due to differential fetal growth rates is due to within-twin differences. This wide variation is the basis of our identification strategy.

[8] See http://www.ivf.comhttp://www.ivf.com.

[9] Ponczek and Souza (2012) provide a comprehensive discussion about the relationship between fertility treatments, twinning probabilities and parents' background characteristics in Brazil. They also show that mother of twins are more educated.

2.3. Results

2.3.1. Main results and robustness checks

Table 4 shows estimates of the effect of birth weight on four mortality outcomes: one-year mortality, neonatal mortality, seven-day mortality and one-day mortality. Each panel reports results for different sample and estimation techniques. In Panel A, we use the sample of singletons and estimate OLS regressions. Pooled OLS estimates for the sample of twins are presented in Panel B. Finally, our preferred results are presented in Panel C, where we use the twin-fixed effects strategy. All regressions include control for infant's sex. The sample sizes and R^2's of the regressions are shown at the bottom of each panel. All standard errors are robust against arbitrary heteroscedasticity, and allow for clustering at the twin-pair level when the sample of twins is used.

The pooled OLS estimates using the singleton sample suggest strong and negative effects of birth weight on infant mortality. The probability of dying within one year of birth of a baby weighting less than 1,500 grams is as much as 31 percentage points higher than that of babies weighting 3,000 grams or more. Relative to a sample average probability of one-year mortality of 34 percent for infants with birth weight less than 1,500, the effect is substantial at 91 percent. For the 1,500-2,500 grams birth weight category the effect falls to 2 percentage points, but remains significant. These estimated coefficients are quite similar in magnitude for neonatal mortality. This suggests that the cross-sectional relationship between birth weight and infant mortality is driven largely by deaths that occur within 28 days of birth. When we use pooled OLS in the sample of twins, we find a similar pattern. Importantly, the estimated coefficients are also similar in magnitude for twins than for singletons, which suggests that both populations are subject to the same relationship between birth weight and infant mortality. This provides reassuring evidence that the results from twins may be generalizable to the general population. Overall, these findings confirm the strong cross-sectional relationship between birth weight and infant mortality found in earlier studies for developed countries.

In direct contrast to the cross-sectional estimates, the twin-fixed effects estimator suggests much smaller impacts. For infants born with very low weight (less than 1,500 grams), there are about 4 percentage points higher risk of death within one year. This

estimated effect is only one sixth the size of the OLS coefficient. Similarly, when we look at the other mortality measures, we find much smaller impacts. The effect of very low birth weight falls from 23 to 3 percentage points for neonatal mortality, from 19 to 1.8 percentage points for seven mortality, and falls from 12 to 0.3 percentage points for one-day mortality. In sum, the estimated effect of very low birth weight falls by a factor of 6 to 40. Despite the estimates fall notably when family unobserved characteristics are accounted for, they remain statistically significant, with exception of one-day mortality. The fact that twin-fixed effects estimates are much smaller suggests that there is a prima facie case for a severe omitted variable bias in the cross-sectional regressions.

Since our sample includes both fraternal and monozygotic twins, one might be even worried if discordance in birth weight between twins are related to these genetic conditions. This type of caveat is recurrently mentioned in the literature that uses within-twin identification strategies. Still, these studies tend to find quite similar results when their samples are restricted to same-sex twin pairs, which clearly contain a larger fraction of identical twin births. In fact, Black, Devereux, and Salvanes (2007) are able to observe directly zigosity in a sub-set of twins and find identical results. The robustness of the findings in such sub-samples suggests that the bias generated by zygosity is, at best, small in practice. We perform the same robustness check by estimating the birth weight effects in a sub-sample that includes only same-sex twin pairs. As shown in Table 5, the estimates are similar to the baseline ones, indicating that zygosity is not affecting our estimates.

The enormous sample size we have at our disposal allows us to explore the relationship between birth weight and infant mortality by cause. We group our sample into four categories: conditions originating in the perinatal period, infectious and parasitic diseases, diseases of the respiratory system, and all other diagnoses. These results are presented in Table 6 using our preferred estimation technique, namely the twin-fixed effects estimator. The results show no robust evidence of a birth weight effect on mortality by infectious, parasitic, or respiratory causes. Furthermore, we find small estimates that are tightly bound around zero, indicating that birth weight does not have any discernible effects on these causes of death. In contrast, the results indicate that the birth weight effects are driven by deaths from conditions originating in the perinatal period, which include respiratory and cardiovascular disorders specific to the

perinatal period, hematological disorders of fetus and newborn, and disorders related to low birth weight. This is perhaps unsurprisingly because some of these disorders are directly diagnosed based on baby's birth weight.

While the functional form used allows for non-linear effects, it may not completely capture the relationship between birth weight and infant mortality if there is specific effects at other birth weight categories. For example, the effects of birth weight may be particularly higher among infants with a birth weight less than 1,000 grams. Next, we use a specification that allows the effects of birth weight to be more flexible. Specifically, we estimate models given by:

$$Death_{ijt} = \alpha + \sum_k D_{ijt}^k \beta^k + x_{jt}' \delta + \mu_{jt} + \varepsilon_{ijt} \qquad (3)$$

where D_{ijt}^k is a dummy variable that indicates if the birth weight of an infant is in the *k*th bin. We use 27 dummy variables corresponding to 100 gram-wide birth weight bins of the distribution of birth weight below 3,000 grams. The bins range from a low of 300-400 grams to a high of 2,900-3,000 grams. The omitted category is birth weight of 3,000 grams or more. We estimate these regressions using both OLS and twin-fixed effects. The results from this more flexible functional form are presented in Figure 3, which plots the coefficients from these weight-bins. In general, there appears to be a concave relationship between birth weight and infant mortality, indicating that reductions in birth weight are more detrimental at lower levels of birth weight. The effects tend to disappear when the birth weight is over 1,800 grams. The results also make clearer the severe omitted variable bias in the OLS regressions. Consider, for example, the cross-sectional results for one-day mortality. They indicate that infants who are born with a weight below 300 grams are 40 percentage points more likely to die within one day of birth. In contrast, the twin-fixed effects results suggest a statistically insignificant impact of about 1 percentage point. In general, the twin-fixed effects estimates are never significant for one-day mortality, with estimated coefficients tightly bound around zero.

Table 7 explores further alternative specifications of the relationship between birth weight and infant mortality. Panel A shows results using birth weight (in grams) as the primary variable of interest, while that Panel B uses the log of birth weight. In general, our results are qualitatively similar using these variables. Our estimates from

Panel A imply that a 50 grams increase in birth weight would reduce one-year mortality by one death per 1,000 births. Since infant mortality is a rare event, estimates may be sensitive to functional form. Panels C and D estimate logit models with twin-fixed effects. Using this functional form, we find results qualitatively similar, but the marginal effects tend to be higher. For example, the coefficient of -0.002 in Panel C implies that a 50 grams increase in birth weight would reduce one-year mortality by three deaths per 1,000 births. This is perhaps unsurprising given that logit models only includes cases in which one twin lives and one twin dies, which may change the composition of the sample.

Next, we replace our measure of infant health with other common measure of infant welfare, namely APGAR scores. This is a clinical test that is given to the newborn in which five parameters are assessed. These include muscle tone, respiratory effort, heart rate, reflexes and skin color. The test provides a total score between 0 and 10, where a higher score means "healthier". The results in Table 8 suggests one strong cross-sectional relationship between birth weight and APGAR scores. Very low birth weight babies are 33 percentage points more likely to have a low 5-minute APGAR score (less than 8). This estimated effect falls by a factor of 13 when twin-fixed effects are included, although it remains statistically significant.

2.3.2. Selective mortality and measurement error

As our analysis is based on live births, a bias could arise if a disproportioned number of the marginal fetus that survive are in the low end of the birth weight distribution. That is, if weak fetuses with potentially low birth weight are less likely to be born alive, then our results would be based on a select sample of surviving (and presumably stronger) births. However, note that the use of this select sample most likely will bias our estimates of the effect of birth weight on infant mortality towards zero. Therefore, we are less concerned about selection bias from selective miscarriage or stillbirth. As such, in the presence of this bias, our estimates should be viewed as lower bounds of the true effect of birth weight on infant mortality.

Another potential concern with our results is measurement error in health outcomes. As we mentioned earlier, some certificate death records were not matched to one of the birth records, which implies that infant mortality is measured with error for

some infants. If the measurement error is random then the consistency and unbiasedness of our estimates would be unaffected. Alternatively, if birth weight covaries with the measurement error, then our estimates would be biased. In Appendix A, we describe in full detail a simple test that measures the extent to which this measurement error may affect our estimates. In particular, the test takes advantage of the fact that the measurement error is observable in the certificate death records and we have information about birth weight for all these births. Thus, the within-twin correlation between the measurement error (or equivalently the likelihood of being matched to birth files) and the birth weight of the infants would be a simple test for this potential bias. Since we are unable to identify twin pairs who were born to the same mother in the death records, the within-twin correlation cannot estimated, but the overall correlation would provide useful information if it goes in the same direction and magnitude. The data indicate that a 200 grams increase in birth weight is associated with a decrease of 0.8 percentage point in the likelihood of being matched. While significant at the 5 percent level, this estimate is small in magnitude, with an implied elasticity of only -0.05. Assuming that the within-twin covariance is smaller than overall covariance between the probability of being matched to certificate birth records and birth weight, then the resulting bias is unlikely to be relevant in practice. [10]

2.3.3. Comparison to existing studies for developed countries

A natural question is whether estimates derived from developed countries are externally valid to the developing world. If the access to medical care is more limited in developing countries or if there are interactions between birth weight and infant mortality, the estimates derived from the US or Norway may not valid in conducting cost-benefit analysis of public health policies in developing countries. As we discussed in the Introduction, the presence of these potential factors likely imply an underestimating of the benefits of such policies.

We compare our estimates to those from Almond and Lee (2005) and Black, Devereux, and Salvanes (2007) in Table 9. Panel A presents our estimates for infant mortality. We present estimates that use either birth weight or log of birth weight as independent variables in order to make our results comparable to these previous studies.

[10] This seems a plausible assumption since it is difficult to think of reasons why, in a given twin pair, one twin has a non-missing unique personal identifier and not the other. In this case, the twin-specific component of the measurement error will tend to zero.

Panel B provides twin-fixed effects estimates for the papers in the US and Norway setting. The means of infant mortality and birth weight are also provided for ease of interpretation.

We find that a 50 grams increase in birth weight would reduce one-year mortality by one death per 1,000 births. Given the mean rate of 36.88, this implies that a 1 percent increases in birth weight leads to a 1.6 percent reduction in infant mortality. Thus, we find a much larger effect on the infant mortality rate than either Almond and Lee (2005) or Black, Devereux, and Salvanes (2007). The estimated elasticity for the US is -0.51, and for Norway is about -0.83. Given the discussion in section 3.2, it is clear that these results cannot be explained by bias from selective mortality or measurement error in mortality outcomes. Thus, a tentative conclusion is that estimates derived from the developed world are not generalizable to poor countries.

2.4. Heterogeneity

While it is beyond the scope of this study to understand why the causal effect of birth weight differ between developing and developed countries, we can assess whether the effects vary heterogeneously across different dimensions to provide tentative evidence of possible explanations. Furthermore, learning whether there are significant interactions also offers evidence about specific channels linking birth weight and mortality outcomes, as well as about possible policy prescriptions that may act to mitigate the consequences of low birth weight. Brazil provides a compelling setting for these purposes because it has a large, demographically heterogeneous, and socio-economically diverse population.

We look at two potential factors. First, the effects of low birth weight may depend on household behavior and it in turn might vary with family disadvantage. Parents with more resources may be simply better able to remediate the health consequences of low birth weight. As poorer families are more likely to be credit constraint, the use of important health services may be more limited. Moreover, neonatal health and parental inputs may be complements in the production function for child quality either because richer families are more likely to adopt compensating health investments or because the investments richer families make have higher returns. Second, one might expect the effects of birth weight to vary with economic

development due to differences in access to public health infrastructure. For instance, it is well-known that widespread open defecation that does not make use of a toilet is one leading cause of infant mortality in developing countries. Thus, low birth weight babies in poor regions are potentially at higher risk of death partly because they are more exposed to unhealthy environments.

We begin by exploring whether the impacts of birth weight vary with family disadvantage. As family disadvantage is unobservable, we proxy it by maternal education and marital status at the time of birth. In this case, family disadvantage should be view as differences in the quality and quantity of available household resources, including child-rearing inputs and parental attention (AUTOR et al., 2016). In Table 10, we estimate our preferred model separately for less- and more-educated mothers, and for married and unmarried mothers. The results for these separate regressions replicate qualitatively the pattern found before. The coefficients for infants born to married and more-educated mothers tend to be smaller. The decline in the estimates ranges from 5 percent to nearly 71 percent. The cross-equation tests of coefficients reject that the coefficients are the same. These results are consistent with the notion that the health effects of low birth weight vary proportionally with family disadvantage.

In Table 11, we assess whether the birth weights impacts vary with economic development. To that end, we first divide the sample according to the quintile of the municipal GDP and then we estimate regressions separately for each group. In general, the effects tend to be smaller for infants born in municipalities with higher economic development. The falls in the estimates are striking, ranging from 41 to 83 percent. The tests for equality of coefficients generally reject the null hypothesis that they are the same. Analogously, in panel B, we divide the sample by level of sanitation coverage and estimate regressions separately for each group. The results indicate that higher access to sanitation is associated with smaller health impacts of low birth weight. Again, the differences in the estimates tend to be large and statistically significant.

Overall, these results confirm that the effects of birth weight interact with family disadvantage and economic development. To disentangle the relative importance of both dimensions, we estimate our basic regression with twin fixed effects including interactions between the discrete birth weight categories and mother's education, marital status, sanitation coverage and GDP. We do so in Table 12. Column (1)

replicates our baseline estimates, while the remaining columns add progressively the interactions. The first thing to note is that there are significant interactions when considered each dimension individually, indicating that the effects of low birth weight vary inversely with family advantage and economic developing. This is consistent with the patterns found in Tables 10 and 11. The second thing is that the interactions tend to be larger and statistically significant for very low birth weight category. This is perhaps unsurprisingly because the effects of birth weight are detrimental at lower levels of birth weight. When all interactions are simultaneously added in column (6), the magnitude and significance of the interactions for education and GDP falls notably. In contrast, the interactions with sanitation and married continue to be large and statistically significant.

2.5.Conclusion

Despite the important reductions in infant mortality rate worldwide during the last 20 years, it continues to be high today in many developing countries. While a variety of factors are likely determinant of poor infant health, the understanding of specific causes is necessary for the most efficient design of policies. Previous studies suggest that low birth weight is a major cause of infant mortality, but much of what we know on the causal link between these variables is derived from developed countries and there is no a priori reason to believe that the results are generalizable to poorer countries. Previous studies for developing countries rely on self-reported survey data and do not have access to comprehensive birth record data, which makes it complicated to estimate the magnitude of the effect of birth weight on infant mortality.

In this article, we address these limitations by using rich administrative data on the universe of births in Brazil and shed light on the importance of birth weight for infant health in a developing country context. Using a within-twin identification strategy, we find that lower babies have increased risk of death within one year. Our estimates imply that very low birth babies have 4 percentage points higher risk of death within one year. Deaths from conditions originating in the perinatal period account for much of these effects. Our results are generally larger than those estimated with data from the US and Norway. This finding illustrate that there may be differences in estimates for developed and developing countries, which suggests that using estimates derived from rich countries may understate the benefits from interventions aimed at decreasing infant mortality by increasing birth weight in developing countries.

A natural question is why the effect of birth weight in developed and developing countries is different. Our findings suggest that financial constraints and parental attention may be an important explanation. If financial constraints hamper the use of important health services or if parental time is a powerful determinant of infant health, we should see even larger health impacts of birth weight in poorer regions. Indeed, we find the strongest birth weight effects for infants born to unmarried and less-educated mothers. In addition, we find that the effects are reduced when local sanitation coverage is high, suggesting that access to public health infrastructure may mitigate the consequences of low birth weight. Overall, these findings suggest that poverty is a likely driver behind the differences we observe in the effects of birth weight between developing and developed countries. Further research on the topic is needed to clarify these relationships.

Table 1. Summary Statistics

	Singletons	Twins	Same-sex male Twins	Same-sex female twins
Characteristics of birth				
Birth weight (in grams)	3,202.44	2,322.16	2,334.93	2,270.65
	(533.40)	(573.91)	(593.68)	(555.82)
Fraction low birth weight (<2,500 grams)	0.07	0.59	0.56	0.63
	(0.26)	(0.49)	(0.50)	(0.48)
Fraction preterm births (<37 weeks)	0.07	0.47	0.48	0.47
	(0.26)	(0.50)	(0.50)	(0.50)
1 minute APGAR score	8.25	7.77	7.71	7.78
	(1.25)	(1.61)	(1.68)	(1.59)
5 minute APGAR score	9.31	8.98	8.94	8.98
	(0.90)	(1.19)	(1.26)	(1.18)
Fraction C-section	0.50	0.79	0.78	0.78
	(0.50)	(0.41)	(0.41)	(0.41)
Fraction male	0.51	0.49	1.00	0.00
	(0.50)	(0.50)	(0.00)	(0.00)
Mother's characteristic				
Fraction high education (12 or more years of schooling)	0.16	0.21	0.20	0.20
	(0.37)	(0.41)	(0.40)	(0.40)
Fraction married	0.34	0.40	0.40	0.40
	(0.48)	(0.49)	(0.49)	(0.49)
age	25.47	27.61	27.21	27.20
	(6.47)	(6.41)	(6.42)	(6.44)
Mortality outcomes				
one-year mortality rate (per 1,000 births)	8.13	36.88	43.89	34.35
	(89.79)	(188.47)	(204.85)	(182.14)
Neonatal mortality rate (per 1,000 births)	6.13	31.29	37.61	29.10
	(78.04)	(174.09)	(190.26)	(168.10)
Seven-day mortality rate (per 1,000 births)	4.78	24.46	29.92	22.27
	(68.95)	(154.47)	(170.37)	(147.55)
one-day mortality rate (per 1,000 births)	2.86	14.11	17.51	12.92
	(53.39)	(117.94)	(131.16)	(112.92)
N	18,929,949	255,362	90,976	93,904

Source. Authors.
Note. Standard deviations are given in parentheses.

Table 2. Summary statistics: heavier versus lighter twins

	Heavier (1)	Lighter (2)
Birth weight:		
Mean	2456.5	2182.9
	(575.47)	(537.91)
Median	2,535	2,250
Twenty-fifth percentile	2,180	1,905
Tenth percentile	1,720	1,475
Fifth percentile	1,320	1,130
First percentile	665	575
Fraction low birth weight (<2,500 grams)	0.46	0.70
Mortality outcomes:		
one-year mortality rate (per 1,000 births)	34.19	39.66
	(181.72)	(195.17)
Neonatal mortality rate (per 1,000 births)	29.50	33.13
	(169.21)	(178.97)
Seven-day mortality rate (per 1,000 births)	23.58	25.35
	(151.76)	(157.21)
one-day mortality rate (per 1,000 births)	14.28	13.93
	(118.64)	(117.20)

Sources. Authors.
Note. Standard deviations are given in parentheses.

Table 3. Components of variance for birth weight and outcomes among twins

	Mean squared error in OLS regressions			Ratio
	(1)	(2)	(3)	(3)/(2)
Birth weight	32.93	16.8	3.52	0.20
One year mortality	0.035	0.024	0.008	0.35
Neonatal mortality	0.030	0.020	0.006	0.34
Seven-day mortality	0.023	0.016	0.005	0.31
one-day mortality	0.013	0.010	0.002	0.28
Controls for:				
Gestation length dummies	No	Yes	-	
Twin-fixed effects	No	No	Yes	

Source. Authors.

Notes. Columns (1)–(3) provide the means squared error from OLS regressions that include no controls, dummies for gestation length (less than 22 weeks, 22-27 weeks, 28-31 weeks, 32-36 weeks, and 37-41 weeks), and twin-fixed effects, respectively. The final column provides the ratio of column (3) to column (2). Birth weight is measured in 100s of grams. The sample size is 255,362.

Table 4. OLS and Twin-Fixed effects of the relationship between birth weight and infant mortality

	One-year mortality	Neonatal mortality	Seven-day mortality	One-day mortality
	(1)	(2)	(3)	(4)
		Panel A: OLS - singleton sample		
Birth weight < 1,500	0.319	0.286	0.228	0.142
	[0.001]***	[0.001]***	[0.001]***	[0.001]***
Birth weight 1,500-2,500	0.021	0.016	0.012	0.007
	[0.000]***	[0.000]***	[0.000]***	[0.000]***
Birth weight 2,500-3,000	0.002	0.001	0.001	0.001
	[0.000]***	[0.000]***	[0.000]***	[0.000]***
R^2	0.132	0.141	0.115	0.074
N	18,929,949	18,929,949	18,929,949	18,929,949
		Panel B: OLS - Twins sample		
Birth weight < 1,500	0.328	0.296	0.238	0.145
	[0.004]***	[0.004]***	[0.004]***	[0.003]***
Birth weight 1,500-2,500	0.013	0.01	0.007	0.003
	[0.001]***	[0.000]***	[0.000]***	[0.000]***
Birth weight 2,500-3,000	0.001	0.001	0.001	0.000
	[0.000]***	[0.000]***	[0.000]**	[0.000]
R^2	0.227	0.219	0.181	0.115
N	255,362	255,362	255,362	255,362
		Panel C: FE - Twins sample		
Birth weight < 1,500	0.057	0.04	0.024	0.004
	[0.007]***	[0.006]***	[0.005]***	[0.003]
Birth weight 1,500-2,500	0.007	0.004	0.003	0.002
	[0.001]***	[0.001]***	[0.001]***	[0.001]**
Birth weight 2,500-3,000	0.003	0.002	0.001	0.000
	[0.001]**	[0.001]*	[0.001]	[0.001]
R^2	0.755	0.77	0.777	0.779
N	255,362	255,362	255,362	255,362

Source. Authors.

Notes. The standard errors are in parentheses and are corrected for heteroskedasticity. In addition, Panels B and C use standard errors corrected for within-twin-pair correlation in the residuals. All regressions control for infant's sex. In addition, regressions in Panels C controls for twin-fixed effects. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

Table 5. Twin-Fixed effects of the relationship between birth weight and infant mortality (The role of Zigosity)

	One-year mortality (1)	Neonatal mortality (2)	Seven-day mortality (3)	One-day mortality (4)
		Panel A: Male same-sex twins		
Birth weight < 1,500	0.069	0.047	0.029	0.004
	[0.013]***	[0.012]***	[0.010]***	[0.006]
Birth weight 1,500-2,500	0.008	0.006	0.004	0.001
	[0.002]***	[0.002]***	[0.002]**	[0.001]
Birth weight 2,500-3,000	0.002	0.001	0.000	0.000
	[0.002]	[0.002]	[0.001]	[0.001]
R^2	0.761	0.774	0.782	0.788
N	90,976	90,976	90,976	90,976
		Panel B: Female same-sex twins		
Birth weight < 1,500	0.052	0.036	0.020	-0.000
	[0.011]***	[0.009]***	[0.008]***	[0.005]
Birth weight 1,500-2,500	0.006	0.003	0.002	0.000
	[0.002]**	[0.002]*	[0.002]	[0.001]
Birth weight 2,500-3,000	0.003	0.001	0.000	-0.000
	[0.002]	[0.001]	[0.001]	[0.001]
R^2	0.759	0.773	0.777	0.777
N	93,904	93,904	93,904	93,904

Source. Authors.

Notes. The standard errors are in parentheses and are corrected for heteroskedasticity and within-twin-pair correlation in the residuals. All regressions control for infant's sex and twin-fixed effects. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

Table 6. Twin-Fixed effects of the relationship between birth weight and one-year mortality (by cause of death)

	Conditions originating in the perinatal period	Infectious and parasitic diseases	Respiratory diseases	Other Diagnoses
	(1)	(2)	(3)	(4)
Birth weight < 1,500	0.037	0.004	0.002	0.013
	[0.006]***	[0.002]**	[0.001]*	[0.003]***
Birth weight 1,500-2,500	0.004	0.000	0.001	0.002
	[0.001]***	[0.000]	[0.000]	[0.001]**
Birth weight 2,500-3,000	0.002	0.000	0.000	0.000
	[0.001]***	[0.000]	[0.000]	[0.001]
R^2	0.775	0.536	0.513	0.523
N	255,362	255,362	255,362	255,362
Mean of dependent variable	0.031	0.001	0.0008	0.0035

Source. Authors.

Notes. The standard errors are in parentheses and are corrected for heteroskedasticity and within-twin-pair correlation in the residuals. All regressions control for infant's sex and twin-fixed effects. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

Table 7. Twin-Fixed effects of the relationship between birth weight and infant mortality (Alternative specifications)

	One-year Mortality	Neonatal Mortality	Seven-day mortality	One-day mortality
	(1)	(2)	(3)	(4)
	Panel A: Twin FE- linear specification			
Birth weight (in grams)	-0.026	-0.019	-0.012	-0.003
	[0.002]***	[0.002]***	[0.002]***	[0.001]***
	Panel B: Twin FE- linear-log specification			
Log(Birth weight)	-76.015	-59.351	-36.562	-10.969
	[6.069]***	[5.596]***	[4.877]***	[3.686]***
	Panel C: Logit model with Twin FE- linear-log specification			
Birth weight	-0.002	-0.002	-0.001	-0.001
	[0.000]***	[0.000]***	[0.000]***	[0.000]***
	Panel D: Logit model with Twin FE- linear-log specification			
Log(Birth weight)	-2.566	-2.317	-1.801	-0.878
	[0.153]***	[0.163]***	[0.176]***	[0.210]***
	Panel E: Logit model with Twin FE- discrete birth weight categories			
Birth weight < 1,500	2.067	1.942	1.545	1.04
	[0.228]***	[0.294]***	[0.332]***	[0.439]**
Birth weight 1,500-2,500	1.22	1.222	0.948	0.918
	[0.212]***	[0.281]***	[0.317]***	[0.421]**
Birth weight 2,500-3,000	0.549	0.491	0.217	0.195
	[0.205]***	[0.273]*	[0.318]	[0.422]

Source. Authors.

Notes. The standard errors are in parentheses and are corrected for heteroskedasticity and within-twin-pair correlation in the residuals. All regressions control for infant's sex and twin-fixed effects. Panels C, D, and E show estimated coefficients from logit models with twin-fixed effects. These logit models only includes cases in which one twin lives and one twin dies, implying a sample size of 8,904 for one-year mortality, 7,130 for neonatal mortality, 5,444 for seven-day mortality, and 3,142 for one-day mortality. In Panels A and B, we have multiplied the coefficients and standard errors by 100 to make them easier to read. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

Table 8. OLS and Twin-Fixed effects of the relationship between birth weight and APGAR scores

	1 minute APGAR score (1)	low 1 minute APGAR score (<8) (2)	5 minute APGAR score (3)	low 5 minute APGAR score (<8) (4)
		Panel A: OLS - Twins sample		
Birth weight < 1,500	-2.489	0.532	-1.843	0.332
	[0.023]***	[0.005]***	[0.021]***	[0.004]***
Birth weight 1,500-2,500	-0.407	0.117	-0.264	0.032
	[0.010]***	[0.003]***	[0.007]***	[0.001]***
Birth weight 2,500-3,000	-0.067	0.019	-0.044	0.004
	[0.009]***	[0.003]***	[0.007]***	[0.001]***
R^2	0.161	0.096	0.164	0.128
N	249,701	249,701	249,440	249,440
		Panel B: FE - Twins sample		
Birth weight < 1,500	-0.36	0.09	-0.138	0.024
	[0.045]***	[0.013]***	[0.030]***	[0.008]***
Birth weight 1,500-2,500	-0.11	0.032	-0.038	0.005
	[0.019]***	[0.006]***	[0.012]***	[0.003]**
Birth weight 2,500-3,000	-0.043	0.009	-0.013	0.003
	[0.016]***	[0.005]*	[0.010]	[0.002]
R^2	0.784	0.737	0.833	0.754
N	249,701	249,701	249,440	249,440

Source. Authors.

Notes. The standard errors are in parentheses and are corrected for heteroskedasticity. In addition, Panel B uses standard errors corrected for within-twin-pair correlation in the residuals. All regressions control for infant's sex. In addition, regressions in Panel B control for twin-fixed effects. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

Table 9. Comparison with Literature about developed countries

			Specification using birth weight (grams)		Specification using log of Birth weight	
	Infant mortality rate (per 1000 births) (1)	Mean of birth weight (2)	Effect size (3)	Elasticity (4)	Effect size (5)	Elasticity (6)
Panel A: Brazil						
Infant mortality	36.88	2,322	-0.026 [0.002]***	-1.63	-76.01 [6.06]***	-2.06
Panel B: Estimates from the US and Norway:						
Almond, Chay and Lee (2005)	38.71	2,417	-0.008 [0.001]***	-0.51	-	-
Black, Devereux and Salvanes (2007)	31.11	2,598	-0.010 [0.003]***	-0.83	-41.1 [7.74]***	-1.32

Source. Authors.

Notes. In Panel A, each column presents the results of a specification that use birth weight (in grams) and log of birth weight as the primary independent variable of interest. The dependent variable is mortality within one year of birth (per 1,000 births). All regressions control for infant's sex and twin-fixed effects. Panel B presents the corresponding estimates previous studies for developed countries. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.10.

Table 10. Twin-Fixed effects of the relationship between birth weight and one-year mortality (by mother's education and marital status)

	Less-educated mothers	More-educated mothers	Unmarried	Married
	(1)	(2)	(3)	(4)
Birth weight < 1,500	0.063	0.034	0.072	0.036
	[0.008]***	[0.012]***	[0.009]***	[0.010]***
Birth weight 1,500-2,500	0.008	0.003	0.007	0.007
	[0.002]***	[0.002]	[0.002]***	[0.002]***
Birth weight 2,500-3,000	0.003	0.001	0.003	0.003
	[0.001]**	[0.002]	[0.002]*	[0.002]
Test of equality of coefficients:				
$\chi 2$		61.998		76.098
p-value		0.000		0.000
R^2	0.753	0.767	0.758	0.749
N	200,870	53,542	151,856	103,506

Source. Authors.

Notes. The standard errors are in parentheses and are corrected for heteroskedasticity and within-twin-pair correlation in the residuals. All regressions control for infant's sex and twin-fixed effects. Less-educated mothers refer to mothers who have 11 years of schooling or less. More-educated mothers refer to mothers who have 12 years of schooling or more. The dependent variable is mortality within one year of birth. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

Table 11. Twin-Fixed effects of the relationship between birth weight and infant mortality (by GDP and sanitation coverage)

	Municipality GDP at the:					*% sanitation coverage at the municipality:*			
	1st quintile	2nd quintile	3rd quintile	4th quintile	5th quintile	<20	20-50	50-85	>85
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Birth weight < 1,500	0.067	0.056	0.084	0.068	0.045	0.063	0.062	0.077	0.037
	[0.030]**	[0.026]**	[0.020]***	[0.016]***	[0.009]***	[0.02]***	[0.02]***	[0.01]***	[0.01]***
Birth weight 1,500-2,500	0.022	0.004	0.008	0.006	0.005	0.012	0.010	0.005	0.005
	[0.006]***	[0.004]	[0.004]**	[0.003]*	[0.002]***	[0.004]***	[0.004]**	[0.003]**	[0.002]**
Birth weight 2,500-3,000	0.009	-0.002	0.003	0.003	0.002	0.004	0.004	0.002	0.002
	[0.005]**	[0.003]	[0.003]	[0.003]	[0.002]	[0.003]	[0.003]	[0.002]	[0.002]
Test of equality of coefficients									
$\chi 2$		11.342	9.414	23.470	43.428		3.574	35.429	81.371
p-value		0.010	0.024	0.000	0.000		0.311	0.000	0.000
R^2	0.734	0.754	0.755	0.759	0.759	0.739	0.736	0.766	0.761
N	21,004	27,602	33,788	47,418	125,536	40,738	34,878	81,878	97,868

Source. Authors.

Notes. The standard errors are in parentheses and are corrected for heteroskedasticity and within-twin-pair correlation in the residuals. All regressions control for infant's sex and twin-fixed effects. Per capita GDP is expressed in constant 2000 prices. The data on GDP and sanitation coverage are obtained from the Brazilian Institute of Geography and Statistics (IBGE). For the analysis by GDP quintile, The test of equality of coefficients compares the results from column (1) to those from columns (2)-(5). For the analysis by sanitation coverage, the test of equality of coefficients compares the results from column (6) to those from columns (7)-(9). The dependent variable is mortality within one year of birth. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

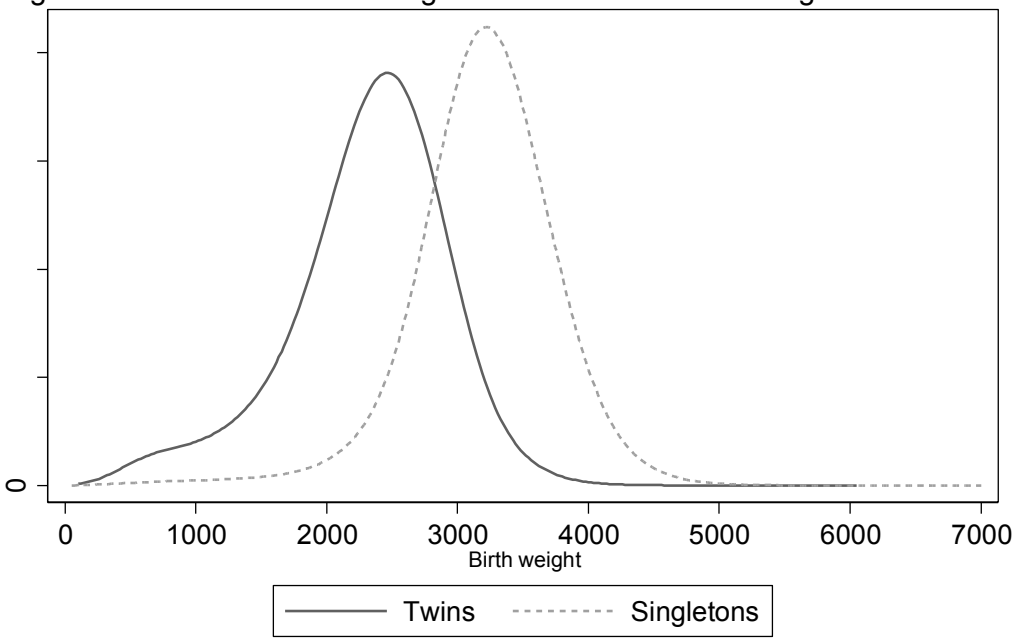
Table 12. Twin-Fixed effects of the relationship between birth weight and one-year mortality (Heterogeneous effects)

	(1)	(2)	(3)	(4)	(5)	(6)
Birth weight < 1,500	0.057	0.064	0.072	0.070	0.070	0.086
	[0.005]***	[0.006]***	[0.007]***	[0.007]***	[0.008]***	[0.009]***
Birth weight 1,500-2,500	0.007	0.008	0.007	0.008	0.009	0.009
	[0.001]***	[0.001]***	[0.001]***	[0.001]***	[0.001]***	[0.002]***
Birth weight 2,500-3,000	0.003	0.003	0.003	0.003	0.003	0.003
	[0.001]***	[0.001]***	[0.001]***	[0.001]***	[0.001]**	[0.001]**
(Birth weight < 1,500) interacted with:						
More-educated mothers		-0.030				-0.013
		[0.010]***				[0.011]
Married			-0.036			-0.030
			[0.010]***			[0.010]***
Sanitation coverage (>85 %)				-0.033		-0.024
				[0.010]***		[0.011]**
GDP at 5th quintile					-0.025	-0.008
					[0.010]**	[0.012]
(Birth weight 1,500-2,500) interacted with:						
More-educated mothers		-0.005				-0.005
		[0.002]***				[0.002]**
Married			-0.000			0.001
			[0.002]			[0.002]
Sanitation coverage (>85 %)				-0.002		-0.000
				[0.002]		[0.002]
GDP at 5th quintile					-0.003	-0.003
					[0.002]*	[0.002]
(Birth weight 1,500-2,500) interacted with:						
More-educated mothers		-0.002				-0.002
		[0.001]				[0.002]
Married			-0.000			0.000
			[0.002]			[0.002]
Sanitation coverage (>85 %)				-0.001		-0.001
				[0.002]		[0.002]
GDP at 5th quintile					-0.001	-0.000
					[0.002]	[0.002]
R^2	0.755	0.755	0.755	0.755	0.755	0.755
N	255,362	255,362	255,362	255,362	255,348	255,348

Source. Authors.

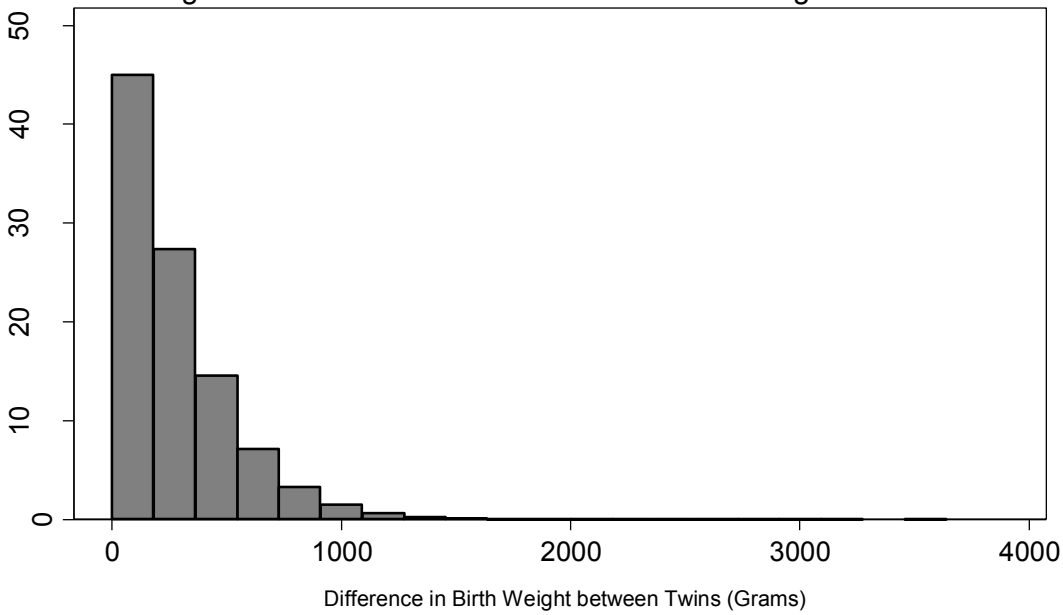
Notes. The standard errors are in parentheses and are corrected for heteroskedasticity and within-twin-pair correlation in the residuals. All regressions control for infant's sex and twin-fixed effects. More-educated mothers refer to mothers who have 12 years of schooling or more. The dependent variable is mortality within one year of birth. Statistical significance is denoted by: ***p < 0.01, **p < 0.05, *p < 0.1.

Figure 1. Difference in birth weight distributions between singletons and twins



Notes. Figure 1 plots kernel density distributions of infant birth weight for twins (solid line) and singletons (dashed line) in our sample.

Figure 2. Distribution of Differences in Birth Weight of Twins



Notes. Each bar represents the percentage of twins whose birth weight difference falls within the specified range. The mean birth weight difference among twins in our sample is 276 grams.

Figure 3. Relationship between infant mortality and birth weight

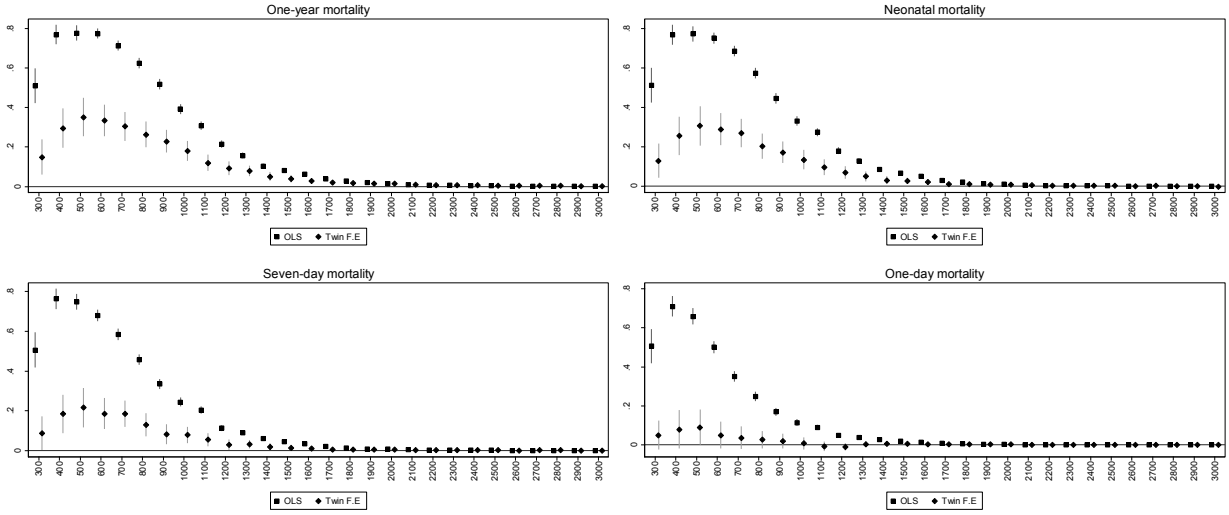


Figure 3 plots the coefficients from the equation (3), which is estimated using either OLS or Twin-fixed effects. We use 27 dummy variables corresponding to 100 gram-wide birth weight bins of the distribution of birth weight below 3,000 grams. The bins range from a low of 300-400 gr to a high of 2,900-3,000 grams.

Appendix A: *A simple test for non-random measurement error*

Consider the twin-fixed effects estimator:

$$Death^{*}_{1jt} - Death^{*}_{2jt} = \beta\left(bw_{1jt} - bw_{2jt}\right) + (\varepsilon_{1jt} - \varepsilon_{2jt}) \qquad (4)$$

where $Death^{*}_{ijt}$ is the true probability of death, but we only observe:

$$Death_{ijt} = Death^{*}_{ijt} + \eta_{ijt}$$

$$\eta_{ijt} = \zeta_{jt} + \phi_{ijt}$$

The measurement error is η_{ijt}, with family-and birth-specific (ζ_{jt}) and twin-specific (ϕ_{ijt}) components. Since the mortality outcomes can only take two values (0 or 1), the measurement error will be equal to 0 if the mortality outcome is measured without error and -1 otherwise. That is, the measurement error will be equal -1 if an infant in the death records is not matched in one of the births. Thus, the omitted variable formula implies that the within-twin estimator of β in the equation (4) is given by

$$\beta_{FE} = \beta + \frac{cov(bw_{1jt} - bw_{2jt}, \phi_{1jt} - \phi_{2jt})}{var(bw_{1jt} - bw_{2jt})}$$

The second term of the right-hand side is the resulting bias from the measurement error. Note that only a significant correlation between birth weight and the twin-specific component of the measurement error would lead to bias in our estimates. As the measurement error is a deterministic function of the probability of being matched, the following regression may be used to determine the importance of bias induced by measurement error:

$$Birthweight_{ijt} = \delta Matched_{ijt} + \varphi_{jt} + \xi_{ijt} \qquad (5)$$

where $Matched_{ijt}$ is a dummy variable indicating whether the infant death record *i* was matched to one of the birth records. The twin-fixed effects are represented by φ_{jt}, while that ξ_{ijt} is an idiosyncratic error term. The parameter δ measures the importance of the bias induced by measurement error. If we are unable to reject the hypothesis that $\delta = 0$, then we would conclude that the measurement error is unlikely to bias our estimates of the effect of birth weight on infant mortality.

3. The Externalities of a Deforestation Control Policy in Infant Health: Evidence from Brazil

Abstract

The burning of forest releases a wide range of contaminants, some of which are known to be hazardous for health. Traditional estimates of the costs of deforestation rarely incorporate the health effects of pollution generated by deforestation. This paper provides the first estimates of the local externalities of deforestation in infant health. Our approach exploits a conservation policy that generated a sharp drop in deforestation across municipalities in the Brazilian Amazon. The core findings are that deforestation control policy led to reductions in the incidence of very low birth weight and extreme prematurity, especially for boys. Collectively, these findings provide additional justification for controlling deforestation.

Keywords: Deforestation; Environmental Quality; Conservation Policy; Infant Health; Brazil
*JEL Classification***:** I12, K32, Q51

3.1.Introduction

The externalities of deforestation are an increasingly salient issue in the debate about the costs and benefits of conservation policies.[11] The smoke from the burning of forest contains a wide range of contaminants, including fine particulate pollution (PM_{10}), sulfur oxide (SO_2) and carbon monoxide (CO), some of which are known to be hazardous for health.[12] At the same time, penetration of sunlight and water accumulation in cleared forests brought by deforestation in rural areas may favor proliferation of malaria-carrying parasites (OLSON et al., 2010; VITTOR et al., 2006, 2009). Many previous studies show that exposure during pregnancy to malaria and air pollution is associated with increased risk of poor birth outcomes (CURRIE; NEIDELL; SCHMIEDER, 2009; GLINIANAIA et al., 2004; GUYATT; SNOW, 2004). Despite this, traditional estimates of the costs of deforestation rarely address local externalities such as the health effects of deforestation.

[11] In this study, deforestation is defined as the conversion of forested land to non-forested land.
[12] The effects of deforestation on air pollution might be particularly more pronounced if deforestation is done for agricultural purposes. One example is deforestation for sugarcane production purposes, which requires burning to remove plant debris, weeds and pests.

Learning about the consequences of deforestation for infant health may be particularly compelling to policy makers. Previous studies have documented that poor fetal conditions have adverse effects on later life outcomes. For instance, *in utero* exposure to air pollution or malaria is associated with reduced cognitive abilities, lower educational attainment and lower income (ALMOND; EDLUND; PALME, 2009; BARRECA, 2010; ISEN; ROSSIN-SLATER; WALKER, 2014; SANDERS, 2012; VENKATARAMANI, 2012). Therefore, evidence that deforestation impairs fetal health may influence policy debates over the costs and benefits of deforestation control.

This paper provides estimates of the effects of deforestation on infant health by examining a conservation policy that generated a sharp drop in deforestation within the Brazilian Amazon. Assunção, Gandour, and Rocha (2015) show that the Action Plan for the Prevention and Control of Deforestation in the Legal Amazon (PPCDAM) substantially reduced deforestation across municipalities. We investigate the effect of PPCDAM, and thus the sharp reductions in deforestation, on infant health. The Brazilian Amazon is an excellent place to study these questions because it is large (its forest of around 5 million km2 is equivalent to half the total area of Europe) and has detailed information on deforestation and infant health before and after intervention, providing an opportunity to answer these questions with extensive panel data.

The PPCDAM, launched in 2004, introduced new procedures to combat deforestation in the Amazon. In particular, it introduced the use of new satellites to detect and locate deforestation activities in real time. Once detected and localized, environmental police soon arrive to the area being deforested and conduct sanctions which include arrests, fines, seizure, confiscation and destruction of production materials. These satellites allowed closer monitoring of deforestation activities. The PPCDAM also promoted the creation of conservation units, leading to an unprecedented increase in the number of conservation areas in Amazon. A rapid and sharp reduction in deforestation characterized the post-intervention years, with deforestation declining from 27,000 km^2 in 2004 to 12,000 km^2 in 2008.

In the absence of a randomized trial, our research design uses the large reductions in deforestation across municipalities generated by the PPCDAM. Specifically, our identification strategy exploits the fact that areas with higher pre-intervention deforestation were more likely to benefit from the conservation policy in

terms of absolute reductions in deforestation. Our analysis then compares changes in birth outcomes before and after intervention in municipalities with different levels of pre-intervention deforestation. We define the timeframe of 2005-2008 as the post-intervention period and 1998-2004 as the pre-intervention period. Our core findings are that deforestation control policy led to lower incidence of very low birth weight and extreme prematurity. We also estimate the effects separately for boys and girls. Understanding whether there are gender differences is importantly in view that previous studies have found that male fetuses are more vulnerable to *in utero* shocks than female fetuses (ALMOND; MAZUMDER, 2011; ERIKSSON et al., 2010; KRAEMER, 2000). Consistent with this literature, we find that the effects of the deforestation control policy were larger in magnitude for boys.

To gain insight into the effects of deforestation control policy on infant health, we tested whether PPCDAM had significant effects on air pollution. The data indicate that conservation policy led to reductions in the levels of PM_{10}, CO, and SO_2. These findings suggest that improvements in air pollution were a plausible mechanism through which the PPCDAM led to better birth outcomes. However, we are unable to rule out the possibility that other non-environmental channels, such as changes in socioeconomic outcomes, play a role. Thus, our results should be interpreted as the reduced form effect of prenatal exposure to the deforestation control policy. Collectively, our findings suggest that deforestation control policies have positive externalities on infant health.

At this point, we should emphasize that there are various issues that may affect the validity of our findings. As in previous works in the birth outcome literature, we cannot fully address the issue of fetus selection.[13] The problem is that we only observe birth outcomes for surviving (and presumably stronger) fetuses. The use of this select sample most likely will bias our estimates of the effect of PPCDAM towards zero, so our effects can be taken to be lower bounds. To investigate this potential bias, we estimate the effect of PPCDAM on fetal deaths. We show that policy had, at best, small effects on fetal deaths. The shortcoming of this exercise is that fetal death records are likely to be subject to measurement error. So, we also investigate whether control policy affected sex ratios. If fetus selection is an important issue, then we should see

[13] See, for example, Currie (2009).

significant increases in sex ratios in favor of the boys, as predicted by literature on "fragile males". We find no significant effects on sex ratios, providing further evidence that bias associated with fetus selection in our sample is small.

More importantly, our estimates could be confounded with those of the *Bolsa Familia* (BF) program, a national antipoverty intervention that was also launched in 2004. We account for this possibility by directly including as a control in all specifications the interaction between a post-intervention dummy and the average percentage of beneficiary families. Since the BF program focused on the poorest municipalities, we also include as additional controls the interactions between a post-intervention dummy and 2004 GDP, child labor rate, illiteracy rate, and sanitation coverage. Reassuringly, our estimates are insensitive to using these controls.

Our research design relies on the assumption that the characteristics of mothers in municipalities with higher pre-intervention deforestation change over time in a way that is comparable to those of other mothers who live in municipalities with less pre-intervention deforestation. We test this assumption by examining whether observable characteristics of mothers change differentially before and after conservation policy adoption. We find that PPCDAM is not associated with changes in maternal characteristics, suggesting that the composition of women giving birth shows little change in the immediate aftermath of policy adoption.

Our paper relates to a set of studies that tried to estimate the effects of deforestation on health outcomes. Sastry (2002) showed that forest burning is associated with increased risk of mortality in Malaysia. Frankenberg, McKee, and Thomas (2005) found that forest burning is associated with worse self-reported health status in Indonesia. Recently, Garg (2014) found that deforestation is associated with increases in the likelihood of malaria infection in Indonesia. The major difference between these studies and ours is that they focused on adult population. In contrast, our study is the first attempt to show the systematic importance of controlling deforestation for infant health. The study of newborns overcomes several methodological difficulties. In particular, the focus on infants mitigates the problem of unknown lifetime exposure by the low migration rates of pregnant women.

The remainder of this paper is structured as follows. The following section presents a description of the PPCDAM and a review of the channels through which policy conservation may affect infant health. Section 3 describes our data set. Section 4 outlines our basic research strategy. Section 5 presents our main results. Finally, Section 6 presents our conclusions.

3.2.Background

3.2.1. Conservation policy efforts in Brazil

Prior to the commencement of policy conservation in 2004, deforestation was rapidly increasing in the Brazilian Amazon, with deforestation estimated at nearly 22,000 kilometers squared annually (2001-2004 years). Roughly 90% of deforestation was concentrated in four out of the nine Legal Amazon states: Amazonas, Mato Grosso, Pará and Rondônia. As a result, these states were a priority in efforts to control deforestation.

In 2004, the Brazilian federal government and the Ministry of the Environment launched the PPCDAM, which presented a set of strategies to combat deforestation in Brazilian Amazon as part of a mutual effort between federal, state and municipal governments. These strategies were implemented in coordination with specialized agencies such as the Brazilian Army, the Federal Police, and the National Institute for Space Research (INPE). This facilitated the implementation of innovative processes focused on command and control, territorial management and land use, and promotion of sustainable practices. By 2020, PPCDAM seeks to reduce deforestation 80% from the estimated 27,000 km^2 in 2004.

One of the most important strategies of the PPCDAM was the introduction of the Real-Time System for Detection of Deforestation (DETER), a satellite-based system that records geo-referenced images on forest cover. Managed by INPE, this system was introduced to closely monitor deforestation activities. From images generated at intervals of 15 days, areas being deforested are detected. Once deforestation activities are identified, INPE issues alert signals to the police responsible for enforcing environmental laws in Brazilian Amazon. Police arrive to the area being deforested and apply sanctions to infringers. Such sanctions include arrests, fines, seizures,

confiscation and destruction of goods, tools, and production materials. Before the adoption of DETER, monitoring activities were based on voluntary reports, which limited law enforcement.

The PPCDAM also promoted the creation of conservation units of integral protection in the Brazilian Amazon. As a result, the conservation areas increased by about 500,000 km^2 from 2004 through 2008. In 2009, nearly 40% of the territory in the Amazon was classified as protected. To control deforestation, the creation of conservation units focused on the municipalities that had recorded higher deforestation in the pre-intervention period.

Finally, the PPCDAM used two strategies to promote sustainable practices. First, the policy promoted large-scale training in forest management techniques and organic farming. Second, through credits and tax exemptions, the policy supported projects focused on organic farming, ecological agriculture, and recovery of degraded areas. Anyone with a sustainable project in the Amazon was eligible for funding. This strategy could be interpreted as a mechanism to compensate communities who adopt lower deforestation.

By all accounts, the deforestation control efforts were extremely successful in the short and medium terms. For example, in 2008, deforestation per year in Amazon fell to 12,000 km^2. The bulk of these declines occurred in areas with the higher deforestation prior to the intervention. Indeed, Assunção, Gandour, and Rocha (2015) illustrate a strong cross-municipality convergence in the annual deforested area (in km^2) after 2004. While deforestation was not completely controlled, deforestation levels declined and remained low throughout the 2000s relative to the pre-intervention era.

3.2.2. Conservation policy and infant health

We draw from existing literature insights to identify the main mechanisms underlying the effects of PPCDAM on infant health. There are at least three ways, or channels, through which policy conservation may have affected infant health. We describe these channels below.

3.2.2.1.Air pollution

Smoke from forest burning contains contaminants hazardous to health, including PM_{10}, SO_2 and CO (YOKELSON et al., 2007).[14] The contaminants believed to be more harmful to health are PM_{10} and CO. PM_{10} is thought to be harmful because it concentrates in the blood and weakens the immune system (SEATON et al., 1995). Although PM_{10} cannot cross the placenta, it may affect fetal health indirectly through deterioration in maternal health. On the other hand, CO directly reduces the availability of oxygen to the fetus. In addition, CO can easily cross the placenta and reach high levels of concentration in its blood. Previous studies show that exposure to PM_{10} and CO is associated with increased risk of low birth weight and prematurity, even at relatively low levels of pollution (CURRIE; NEIDELL, 2005; CURRIE; NEIDELL; SCHMIEDER, 2009; CURRIE; WALKER, 2011).[15] Previous studies also have found that SO_2 is associated with worse birth outcomes (ARCEO; HANNA; OLIVA, 2015). Taken together, these facts suggest that the PPCDAM may have positive externalities on infant health through reductions in air pollution.

3.2.2.2.Malaria risk

Lower malaria risk is also a potential mechanism through which the PPCDAM could contribute to better fetal health. Deforestation in rural areas may favor specific mosquitoes capable of carrying malaria. The development of such mosquitoes depends on sunlight and humidity. Cleared forests receive more sunlight and are more susceptible to the formation of puddles with a neutral pH, favoring the development of these mosquitoes (OLSON et al., 2010; VITTOR et al., 2006, 2009). There is evidence supporting this. For example, Vittor et al. (2006) found that the risk of being bitten by the primary vector of malaria in the Amazon, *Anopheles darlingi*, is 278 times higher in deforested areas than in those that were predominantly forested in Peru. However, not all mosquitoes carry parasites, and thus not all bites result in malaria. Therefore, this evidence is only suggestive of a link between deforestation and malaria. For the Brazilian Amazon, Hahn et al. (2014) also found evidence suggesting that higher deforestation leads to increased risk of malaria.

[14] One major cause of forest burning is human activity. Most human-induced forest burning clears land for agricultural or settlement purposes. Given its low cost and efficiency, it is not surprising that forest burning is a common practice. For the Brazilian Amazon, some estimates suggest that, on average, the fire-damaged area represents up to 55% of the annual area deforested (MORTON et al., 2011).

[15] For a complete review of the literature in general, see Glinianaia et al. 2004.

Malaria affects fetal health through direct transmission from the mother (MENENDEZ; MAYOR, 2007; POESPOPRODJO et al., 2010). In addition, the diversion of energy from the mother to fight against the disease can harm the fetus, restricting food intake and causing oxygen deprivation associated with anemia (CRIMMINS; FINCH, 2006). There is evidence showing that malaria *in utero* is associated with poorer birth outcomes (GUYATT; SNOW, 2004; HUYNH et al., 2011; SARR et al., 2006). In addition, there is indirect evidence documenting that exposure to malaria during pregnancy has negative effects on fetal health. For example, Chang et al. (2014) show that individuals exposed to malaria during pregnancy have higher risk of cardiovascular diseases.[16]

3.2.2.3.Agricultural productivity and income

As we mentioned before, PPCDAM offered credits for sustainable projects. At least in the short term, this could have resulted in higher income. In turn, higher income means more consumption and better nutrition during pregnancy. There is consistent evidence showing that improved nutrition during pregnancy leads to better birth outcomes, including reduced risk of low birth weight and prematurity (ALMOND; MAZUMDER, 2011). Through the income channel, then, conservation policy could have positive externalities on infant health.

Alternatively, if conservation policy was effective in controlling deforestation, then it could have adversely affected agricultural activities. For example, the production of sugarcane requires the use of large tracks of land around processing plants, which generates pressure on deforestation. Thus, a possibility is that the new conservation policy could have reduced production of such agricultural activities due to lower productivity and thereby led to a reduction in household income. Through this channel, the PPCDAM could contribute to higher risk of poor birth outcomes.

Overall, the net effect of deforestation policy on income is ambiguous. Indeed, previous studies have failed to show that conservation policy is associated with significant changes in local economic variables such as GDP (Assunção, Gandour, and

[16] In absolute terms, around 4 million people in the Brazilian Amazon region are at high risk of malaria, a figure that is similar to that of Sierra Leone, the country with the highest rate of deaths from malaria. In relative terms, around 800 cases per hundred thousand inhabitants per year are reported in the Brazilian Amazon; this is 34 times lower than that of Sierra Leone. While the risk of malaria in Brazil is not substantially high, we do not rule out the possibility that it plays a role.

Rocha 2015; Ferreira, Ribera, and Horridge 2015). Since both effects work in opposite directions, one possibility is that they happen to cancel each other out so that the reduced form estimates of the effects of policy on income are zero. We also present evidence consistent with previous studies that the PPCDAM had, at best, small effects on income.

3.3.Data and summary statistics

Our analysis is based on a municipality-by-year unbalanced panel data for the 1998-2008 period.[17] The analysis use all municipalities of Amazon states considered priorities, which comprise about 90% of the total deforestation. We do not include subsequent years to 2008 in the analysis as an effort to compare infants born immediately after the policy adoption with those born before. In total, there are 396 municipalities in the sample. These municipalities account for approximately 62% of total population in the Brazilian Amazon.

3.3.1. Birth outcomes

Our microdata for birth outcomes come from the Brazilian National System of Information on Birth Records, which records all births in Brazil. Its data do not, however, cover most municipalities in our sample before 1998. For this reason, our period of analysis is 1998-2008 – approximately 3 million birth records.[18] The data includes the exact date of birth, weeks of gestation, sex, birth weight, and maternal characteristics such as marital status, age and education. The database also provides the municipality of residence of the mother. Based on this information, we construct a municipality-by-year of birth panel over the 1998-2008 period. Since we use the municipality in which the mother lives as a reference for the panel, we are able to capture exposure *in utero* to changes in deforestation.

The consolidated dataset allows us to calculate rates of low birth weight (defined as birth weight less than 2,500 grams), very low birth weight (defined as birth weight less than 1,500 grams), prematurity (defined as gestation less than 38 weeks), and

[17] The panel is unbalanced due to the presence of missing values in some variables. However, the incidence of missing is relatively small (about 1% of all observations).
[18] This includes infants from mothers residing in rural and urban areas. Since data do not provide the type of municipal area, we are not able to identify births from mothers residing in rural areas.

extreme prematurity (defined as gestation less than 28 weeks) by municipality and year of birth. In the original dataset, the length of pregnancy is coded into categories. From 2000 onwards, this variable was collected using different categories, making it impossible to compare with previous years. Therefore, our analysis for prematurity corresponds to the period 2000-2008. We also build these variables separately for girls and boys.

Using this dataset, we also calculate three additional variables: *i*) rates of births from mothers with over 12 years of education[19]; *ii*) fraction of newborns from teenage mother (age <20 years); and *iii*) rates of births from married mothers (available from 2000).[20] Table 1 presents a statistical summary of all variables. On average, there are 715 births per municipality per year. 5.8% of these infants have low birth weight. Premature birth rate is 6.1%. Rates of births from teenage and married mothers are 31 and 30%, respectively.

3.3.2. Deforestation

Deforestation data are collected by INPE, which has used municipality-level satellite imagery since 2000. We define deforestation as km^2 of forest cleared annually (i.e., the increment in the total area deforested between years *t* and *t*-1). We used annual forest cleared in km^2 (not percentages) because that is INPE's official definition of deforestation. Thus, our definition of deforestation conforms to that monitored by PPCDAM. During 2001-2008, on average, 48 km^2 was deforested (with a standard deviation of 110 km^2) per municipality per year.

We use deforestation data for the period of 2001-2004 to construct a measure of pre-intervention municipality-level deforestation. Data for deforestation in 2004 likely do not capture pre-intervention deforestation adequately because it could be lower (or higher) in some municipalities due to year-specific measurement error. To mitigate this issue, we use the 2001-2004 period to compute a proxy of pre-intervention deforestation by estimating the following regression:

[19] We use 12 years of education as cut-off in Brazil because that is the number of school years required for a high school degree.

[20] Since the rates of newborns from married mothers are available from 2000, we do not include this variable in the birth weight regressions for more precise estimates. However, the inclusion of this variable does not alter our results.

$$D_{it} = \delta_0 + \delta_1 Time + \sum_i \lambda_i Time * M_i + \sum_i \rho_i M_i + \zeta_{it} \qquad (1)$$

where D_{it} is the measure of deforestation for municipality *i* in year *t*, *Time* is a linear time trend, *M* represents municipality dummies and ζ is an error term. We use the predicted value of deforestation in 2004 for each municipality as a measure of pre-intervention deforestation. Note that this strategy deals somewhat with year-specific measurement errors.

Figure 1 shows cross-sectional variation in pre-intervention deforestation across municipalities. This figure reveals that the levels of pre-intervention deforestation vary substantially, including within-state variation. For instances, the average pre-intervention deforestation of the municipalities in the top quartile of the pre-intervention deforestation distribution is 30 times higher than that of municipalities in the bottom quartile. The standard deviation in pre-intervention deforestation across municipalities in the sample is over 120 km^2 (relative to a mean of 67 km^2). As a result of this variation, as we shall see, different municipalities experienced differential changes in deforestation in the post-intervention period. This is the basis of our identification strategy.

3.3.3. Other data

We make use of information from other sources. In the appendix (section A1), we describe the construction of the municipality-level series for PM_{10}, CO y SO_2 for the period 1998-2008. In section A2 of the Appendix, we present the methodology for computing an annual index of crop prices at municipality level.

We also collect other municipality-level data on: *i*) fetal deaths for the period 1998-2008 from the Brazilian Mortality Information System; *ii)* BF beneficiary families for the period 2004-2008 from the Ministry of Social Development and Hunger Alleviation; *iii*) Gross Domestic Product (GDP) (available for 2000-2008 period); *iv)* agricultural production (available for 2000-2008 period) from the Brazilian Institute of Geography and Statistics (IBGE); *v)* child labor rates, illiteracy rates, and sanitation coverage from the 2000 Brazilian Demographic Census; *vi)* physicians per capita in 2000 from the Applied Economic Research Institute (IPEA); and finally *vii)* government spending share on education and health (available for 1999-2008 period) from the Finance Ministry.

3.3.4. Additional descriptive statistics

In this section, we present a graphical analysis of the main variables. To implement such an analysis, we classify municipalities into "Low and High" categories based on their pre-intervention deforestation. The Low (High) category consists of municipalities under (over) the 75 percentile of the pre-intervention deforestation.[21] Figure 2 illustrates striking differences across municipalities in deforestation reductions. The municipalities in the "High" category experienced the most remarkable reductions, with deforestation declining from 207 km^2 in 2004 to 87 km^2 in 2008. In contrast, the reductions were less pronounced in the municipalities in the "Low" category, with deforestation ranging from 13 km^2 in 2004 to 9 km^2 in 2008. This provides informal evidence that conservation policy was particularly more successful in municipalities with higher pre-intervention deforestation.

Figure 3 shows that there are little changes in birth outcomes over time. The most noticeable changes are in the incidence of very low birth weight. In fact, the rate of very low birth weight is getting worse over time, but the municipalities in the "High" group this birth outcome is getting less worse. There are no obvious trends in other birth outcomes across municipalities. Moreover, Figure 3 seems to show lack of parallel trends. For example, prematurity and very low birth weight show some sort of convergence before the program comes into place. As we will see in more detail below, such differential trends in maternal characteristics across municipalities are not statistically significant. Overall, it is difficult to infer any relationship between conservation policy and infant health from this informal analysis.[22]

3.4. Empirical strategy

To identify the impacts of the conservation policy, and thus the sharp reductions in deforestation, on birth outcomes, we estimate:[23]

[21] The results of this analysis are qualitatively similar to the use of 80, or 95 th percentiles as cut-off points.

[22] Figure A1 displays trends in maternal characteristics over time. There is a declining trend in births from teenage and married mothers. Meanwhile, the percentage of newborns from mothers with over 12 years of education increases over time. A look at national trends of these variables suggests a similar pattern.

[23] The use of an Ordinary Least Squares (OLS) regression of the relationship between a birth outcome and deforestation cannot provide causality because deforestation may be endogenous. Omitted variables bias

$$y_{it} = \alpha + \beta Post_t x BaseDeforestation_i + \theta \mathrm{Z_{it}} + \varphi Trend_{it} + \eta_i + \mu_t + \xi_{it} \qquad (2)$$

where y_{it} is the birth outcome of interest for the municipality *i* in year *t*; $Post = 1$ if year is 2005 or thereafter;$BaseDeforestation_i$ is the pre-intervention municipality-level deforestation (computed as described in section 3.2), which is time-invariant; Z is a vector including control variables (e.g., percentage of births from married mother); $Trend_{it}$ is a municipality-specific linear time trend; and η_i and μ_t are municipality and year fixed effects, respectively. In all specifications, we use robust standard errors clustered at the micro-region level.[24]In addition, the standard errors are corrected to take into account that baseline deforestation is the predicted value from Equation (1).

Model (2) is essentially a differences-in-differences setup that utilizes the fact that municipalities with higher pre-intervention deforestation were more likely to benefit from the intervention, to identify policy effects. Put differently, β compares the change in birth outcomes between infants born before and after the intervention in municipalities that benefited more from the policy against the same change for infants born in municipalities with less pre-intervention deforestation. If the conservation policy led to lower incidence of prematurity and low birth weight, then we would expect to see negative coefficients on β.

Our identifying assumption is that in the absence of the PPCDAM, municipalities with different levels of pre-intervention deforestation would have experienced the same proportional changes in birth outcomes. Naturally, municipalities with different levels of pre-intervention deforestation may differ in ways that could affect birth outcomes. Any such differences that are time invariant will be captured by the municipality fixed effects in Equation (2). Only differential trends in unobserved factors across these municipalities would be a threat to the validity of this approach. The focus on municipalities located in states classified as priorities by the PPCDAM is a basic step to increase comparability across municipalities and limit potential differential

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may lead to underestimates or overestimates of the true effect of changes in deforestation. Furthermore, deforestation indices are likely to be subject to measurement error, which would attenuate the OLS estimates, and the use of the fixed-effect estimator may exacerbate this problem. Not surprisingly, our estimates based on OLS regressions (available upon request from the authors) are inconclusive of a relationship between deforestation and infant health.

[24] In general, a micro-region is formed by a group of municipalities sharing the same border. On average, a micro-region is made of seven municipalities.

trends. While we cannot directly test the identifying assumption, we can use data prior to the policy to test if, for each outcome of interest, municipalities with different levels of pre-intervention deforestation were on parallel trends prior to the policy. For this, we estimate the following regression:

$$y_{it} = \alpha + \sum_{t=initial\ year}^{2004} \rho_t year\ dummies_t x BaseDeforestation_i + \omega_i + \lambda_t + \upsilon_{it} \qquad (3)$$

If trends across municipalities with different levels of pre-intervention deforestation were the same during the pre-treatment period, then they are likely to have been the same in the post-treatment period in the absence of the PPCDAM. Figure A2 plots coefficients and 95% confidence intervals from Equation (3) for deforestation and birth outcomes. All the coefficients on the pre-intervention years are insignificant, suggesting that the assumption of parallel trends might be valid. Note that this finding is not driven by large standard errors. For example, the coefficients for very low birth weight average -0.00017. Relative to the mean, this implies an estimated effect of only 0.50% for the municipality at the 75th percentile of the pre-intervention deforestation. To ensure that our model (2) identifies a policy effect separate from incidental municipality trends, we control for any differential preexisting trends by directly including municipality-specific linear time trends in all specifications.

We address several identification issues that emerge when following the specification (2). First, the pre-intervention deforestation is measured in km^2 and this measure does not take into account that municipalities vary in size. At some level, it is obvious that municipality size matters. For instance, if smaller municipalities have better healthcare, then likelihood of differential trends driven by other factors may increase. We mitigate this by including the interaction between $Post_t$ and municipality size (in Km^2) in all specifications. The inclusion of this variable controls for any differential trend across municipalities with different sizes. Furthermore, note that the inclusion of the municipality-specific linear time trends also deal, to a great extent, with this problem.

Second, PPCDAM's launch coincides with that of the BF, a conditional cash program that targets poor families in Brazil. Beneficiary families receive a monthly cash

transfer (equivalent to 40% of the monthly minimum wage), conditional on school attendance and health center visits. Approximately 22.3% of Brazilian families were beneficiaries of the BF in 2010. For the Amazon, that figure corresponds to 30%. Although all municipalities in our sample had beneficiary families in the period 2004-2008, there is great variation across municipalities.[25] If the BF implementation is correlated with that of the PPCDAM, then it may lead to biased estimates of β. To deal with this issue, we directly included as a control in all specifications the interaction between $Post_t$ and the average percentage of beneficiary families. Since the BF program focused on the poorest municipalities, we also included in all specifications the interaction between *Post* and 2004 GDP. As robustness checks, we include other controls, such as interactions between *Post* and sanitation coverage, and illiteracy rates.

Third, the years after 2004 witnessed a marked reduction in crop prices (Assunção, Gandour, and Rocha 2015), which may have discouraged deforestation and affected household income. This would threaten the validity of our empirical strategy if municipalities with different levels of pre-intervention deforestation experience different changes in income due to reductions in crop prices. To address this issue, we control for an annual index of crop prices at municipality levels in all regressions.

Fourth, as we only observe birth outcomes for surviving (and presumably stronger) fetuses, a biased selection could arise. Any selection bias that results from using this select group is likely to lead to bias our estimates of the effect of PPCDAM towards zero. We address this issue in the robustness section, specifically by examining the effect of PPCDAM on the rate of fetal death. Statistically insignificant effects indicate that such bias is unlikely to play a major role.

We cannot directly test the validity of the identifying assumption. However, the sample selection of municipalities localized in states that were classified as priorities, the conditioning on municipality-specific linear time trends, and the use of the additional controls in all regressions make the validity of the empirical strategy reliable.

[25]For example, in 2008 the percentage of beneficiary families ranging from 3 to 60% across municipalities (Mean: 29.6; Standard Deviation: 11.09).

3.5. Results

3.5.1. Main results

We begin by examining the effects of the PPCDAM on deforestation. We estimate Equation (2) using deforestation as dependent variable (Table 2). Column (1) is based on a specification that adjusts only for municipality-specific linear time trends, as well as municipality and year fixed effects. Results from this specification indicate that municipalities with higher pre-intervention deforestation experienced larger reductions in deforestation during the post-intervention period (coefficient estimated=-0.32 and statistically significant at 5%). This finding is robust to controls for the annual index of crop prices, and the interactions between *Post* and municipality size, the mean fraction of BF beneficiaries, and the 2004 GDP (Column (2)). This provides reassuring evidence on the credibility of the analysis.

Column (3) presents yearly interaction terms for each of the post-policy years to investigate potential non-linear effects. All coefficients on these interactions are negative and significant. These results also suggest that policy had immediate impacts on deforestation. Overall, for the municipality in the 75th percentile of the pre-intervention deforestation distribution, conservation policy led to a 35-37% reduction in deforestation. These effects resemble those of other deforestation policies. For example, Jayachandran et al. (2016) find that a randomized controlled trial of the Payments for Ecosystem Services (PES) program reduced deforestation 40% in Uganda.

Table 3 presents the results for birth outcomes. Each Panel presents results from different birth outcome. The number of observations and the effects of the PPCDAM at the 75th percentile of pre-intervention deforestation are presented at the bottom of each Panel. Column (1) uses a specification that controls only for municipality-specific linear time trends as well as municipality and year fixed effects, while the remaining columns correspond to specifications that include an additional set of controls.

The result from column (1) in Panel A reveals a statistically significant and negative estimate of the parameter of interest. Given that the rate of very low birth weight is increasing over time (as shown in Figure 3), the estimated coefficient indicates that municipalities with higher pre-intervention deforestation experienced lower increases in very low birth weight rates. This estimated coefficient is insensitive

to adding other controls. For the municipality in the 75th percentile of the pre-intervention deforestation distribution, deforestation control policy is associated with 18-20% reduction in the rate of very low birth weight. Results from Panel B show that the conservation policy is associated with reductions in low birth weight incidence, though the estimates are imprecise and statistically insignificant.

The results from Panel C show a statistically significant and negative coefficient, implying that conservation policy contribute to reduce the incidence of extreme prematurity. Although the effect is less precisely estimated with the inclusion of additional controls, it remains statistically significant at 5%. For prematurity, we also found a negative coefficient (Panel D) across specifications, but very imprecisely estimated and statistically insignificant.

In Table A1, we estimate yearly interaction terms for each of the post-policy years to examine possible dose-response effects. These regressions replicate the patterns found before, indicating that deforestation control policy is associated with reduced risk of poor birth outcomes. The coefficients on these interactions are similar relative to the baseline ones, although some are imprecisely estimated.

Table 4 investigates the gender specificity of the findings from Table 3. We estimate the complete specification for boys and girls separately. The results suggest that conservation policy had a much greater impact on male fetuses. For very low birth weight, boys from the municipality in the 75th percentile of the pre-intervention deforestation distribution experienced a 35.4% reduction. For girls in the same municipality such a reduction is estimated at 10%. For extreme prematurity, we find that, for the municipality in the 75th of pre-intervention deforestation, policy led to 53 and 25% reductions for boys and girls, respectively (column (3)). Although the estimates for premature births are imprecisely estimated (column (4)), they still suggest more pronounced effects on male fetuses.

That sex discrimination accounts for these differences seems implausible given that gender bias at early ages is not considered significant in Brazil. The sex ratio at birth, which has emerged as an indicator of sex-discrimination at early ages, is in the normal range 104-107 in our sample. Rather, our results are consistent with the literature on fragile males, which attributes gender differences to differences in ability

to produce nutrients in the placenta. That attribution is supported by studies documenting gender-specific effects of different shocks during pregnancy (ROSS; DESAI, 2005).

Taken together, the evidence suggests that the conservation policy had positive effects on infant health. Furthermore, such effects seem to be heterogeneous across the distribution of birth outcomes. In particular, newborns in the bottom of birth outcome distribution benefited more from this policy.

3.5.2. Channels

Our final effort is to uncover the channels governing the relationship between reduced deforestation and infant health. We begin by assessing whether the policy affected air pollution, given the well-documented evidence that pollutants such as CO and PM_{10} have harmful effects on fetal health (Currie, Neidell, and Schmieder 2009; Currie and Walker 2011; Glinianaia et al. 2004).

In Table 5, we estimate the specification of Equation (2) using CO, PM_{10}, and SO_2 as dependent variables. All specifications included controls for crop prices, and the interactions between the *Post* and municipality size, 2004 GDP, and the average percentage of BF beneficiaries. Overall, the results indicate that policy conservation is associated with significant reductions in air pollution. For the municipality in the 75th percentile of the pre-intervention deforestation, the reductions in the levels of CO, PM_{10} and SO_2 were 17.2, 17.6 and 8.8 percent, respectively. The finding that PPCDAM led to a 35% reduction in deforestation for the municipality in the 75th percentile of pre-intervention deforestation suggests that elasticities of CO, PM_{10}, and SO_2 with respect to deforestation are 0.54, 0.55, and 0.27, respectively. Assuming fire-damaged areas represent 50% of the area annually deforested (MORTON et al., 2011), crude estimates of the elasticities of CO, PM_{10}, and SO_2 with respect to biomass burning would be 1.08, 1.10, and 0.54, respectively. These estimates are in line with previous. For example, Sillapapiromsuk et al (2013) find an elasticity of PM_{10} with respect to biomass burning of 1.04.

To evaluate the quantitative significance of the reductions in air pollution for infant health, we combine the results from tables 2 and 5. These estimates suggest, for example, that a 1% reduction in CO lowers incidence of low birth weight 0.20%. While

imprecisely estimated, this point estimate is quite similar to that of other studies. For instance, Currie and Walker (2011) suggest an elasticity of low birth weight with respect to CO of 0.27. The caveat to our analysis is that our estimates may reflect also the importance of other potential mechanisms. Thus, this exercise should be viewed cautiously.

In Table 6, we empirically investigated whether the policy affected socioeconomic outcomes such as agricultural production and income. In the absence of a direct measure of household income, we use GDP at the municipality-level as a proxy. Column (1) reveals a positive estimate of the parameter of interest, suggesting that municipalities with higher pre-intervention deforestation experienced larger increases in GDP. However, such a coefficient is not statistically significant. We also find that conservation policy is no associated with changes in agricultural production or crop production (Columns (2)-(3)). Overall, results indicate little evidence that the conservation policy is associated with changes in agricultural activity or income. This finding is consistent with Jayachandran et al (2016), who show that the PES in Uganda had no socioeconomic impacts.

3.5.3. Robustness of findings

We performed a number of robustness checks designed to examine the validity of our identification strategy. Table A2 explores a variety of alternative specifications. Column 1 replicates our main estimates, while column 2 shows the results of a specification that includes government spending share on health and education as control variables. These additional controls could capture different dimensions of local policy correlated with both the implementation of the PPCDAM and the BF program. The inclusion of these variables slightly increases the estimated coefficients of interest (in absolute value). Columns (3)-(7) control for interactions between *Post* and child labor rate, illiteracy rate, sanitation coverage and physicians per capita. In some cases, the inclusion of these variables leads to slightly larger estimates. In particular, the estimated effect on low birth weight tends to be more negative and statistically significant. Overall, our estimates are robust to including these additional controls.

We also check for possible mean reversion by including a one-year lagged dependent variable (y_{it-1}) as a control (Table A3). The inclusion of this variable hardly

changes our results. However, the OLS estimator in column (2) is inconsistent because of the presence of the lagged dependent variable on the right-hand side. Column (3) estimates the same model using the Arellano-Bond GMM (Generalized Moment Method) dynamic panel estimator. In most cases, this leads to more negative coefficients than in our baseline specification.[26]

In the Table A4, we investigate the potential bias from fetus selection. In column (1), we estimate the specification of Equation (2) using the fetal death rate as the dependent variable. If the deforestation control policy reduced fetal death, then we should observe a negative and statistically significant estimate on the coefficient of interest. The estimate is indeed negative, but not statistically different from zero. However, it is exercise is imperfect because fetal death is registered only for pregnancies that were long enough. As a further check, we examine whether the policy had significant effects on sex ratio at birth. If policy reduced significantly fetal deaths, then we should see increases in the sex ratio in favor of boys. The results show a positive coefficient, but statistically insignificant (Column (2)). Put together, this evidence suggests that the bias from fetus selection is likely to be small.

We also test whether characteristics of mothers in municipalities with higher pre-intervention deforestation change over time in a way that is comparable to those municipalities with less pre-intervention deforestation. Columns (3)-(5) from Table A4 show the results of a natural falsification test: we estimate the baseline specification using maternal characteristics as dependent variables. To the extent that our estimates do not reflect the compositional changes in births, the PPCDAM should not predict maternal characteristic changes. This is exactly what we find. We also find a statistically insignificant relationship between PPCDAM and pregnancies per capita. This provides further evidence that compositional changes in births have not taken place.

We performed further robustness tests (not shown).[27] The use of 2004 deforestation as measure of pre-intervention deforestation reduces, in magnitude, the estimated coefficients of interest, although the results are qualitatively similar. Our results are also robust to using the average deforestation for the period 2001-2004 as

[26]These results are broadly similar to those derived from the Blundell-Bond's GMM estimator.
[27]These further robustness exercises are available upon author request.

measure of pre-intervention deforestation. Estimates are also unaffected when using the interaction between *Post* and the percentage of 2004 BF beneficiaries (rather that the average percentage of BF beneficiaries) as a control. Our results are also robust to excluding extremely high or low values of pre-intervention deforestation.[28] The results are qualitatively similar to using standard errors clustered at the municipal level (rather than micro-region level). The inclusion of quadratic and cubic terms of the interaction between *Post* and municipality size does not alter our estimates. Finally, we examine whether our results are affected when excluded the year 2000 of this analysis. In that year, it appears that there is some systematic deviance in birth outcomes across municipalities with different levels of pre-intervention deforestation (as can be observed in Figure 3). The estimates dropping this year indicate that our findings results are not driving by this deviance.

3.6. Final remarks

This study provides estimates of the externalities of a deforestation control policy in infant health. Indeed, this study finds that the Brazilian conservation policy launched in 2004 had positive effects on infant health. Specifically, we find that the conservation policy is associated with reductions in the incidence of very low birth weight and extreme prematurity. These findings are generally larger in magnitude for boys, which reinforces the idea that a gender-specific analysis can be useful when assessing effects of *in utero* shocks. The patterns in our data suggest that improvements in air pollution were an important pathway.

One way to assess the magnitude of the estimated impacts is to compare the effects of conservation policy with previous estimates of the effect of other environmental risk factors. Using exogenous changes in air pollution, Currie and Walker (2011) showed that the reductions in air pollution resulted in a 10% reduction in the incidence of low birth weight. In a recent study, Currie and Schwandt (2014) found that increases in the toxic dust and smoke due to the terrorist attack of September 11, 2001 on the World Trade Center increased the incidence of prematurity and low birth weight at 27% and 13%, respectively. Almond, Chay, and Lee (2005) showed that smoking during pregnancy increases the incidence of low birth weight by 4%.

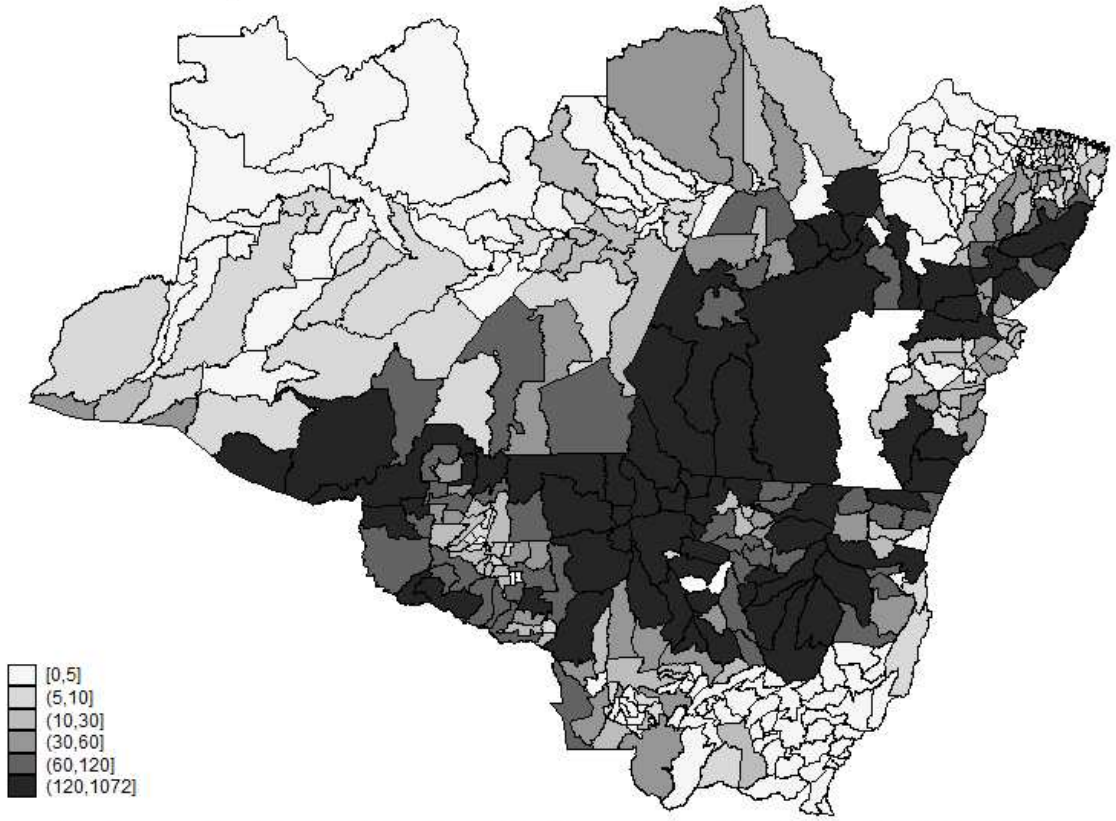
[28] We define extremely low (high) deforestation as those municipalities (over the 99th) under the 1th percentile of the distribution of pre-intervention deforestation.

Compared to our estimates, the conservation policy had smaller effects on birth weight and prematurity.

The findings from this study have important implications for policy and contribute to the growing literature on the importance of a healthy fetal environment. Our findings point to a group—infant boys— that is particularly vulnerable to changes in deforestation. The cost-benefit analyses of conservation policies should take into account this. As such, our findings provide additional justification for interventions that aim to prevent and control deforestation. Given previous studies documenting that poor fetal health has adverse consequences on education, life expectancy and productivity, our results suggest that deforestation control policies could reduce inequality and have lasting benefits in accumulation of human capital.

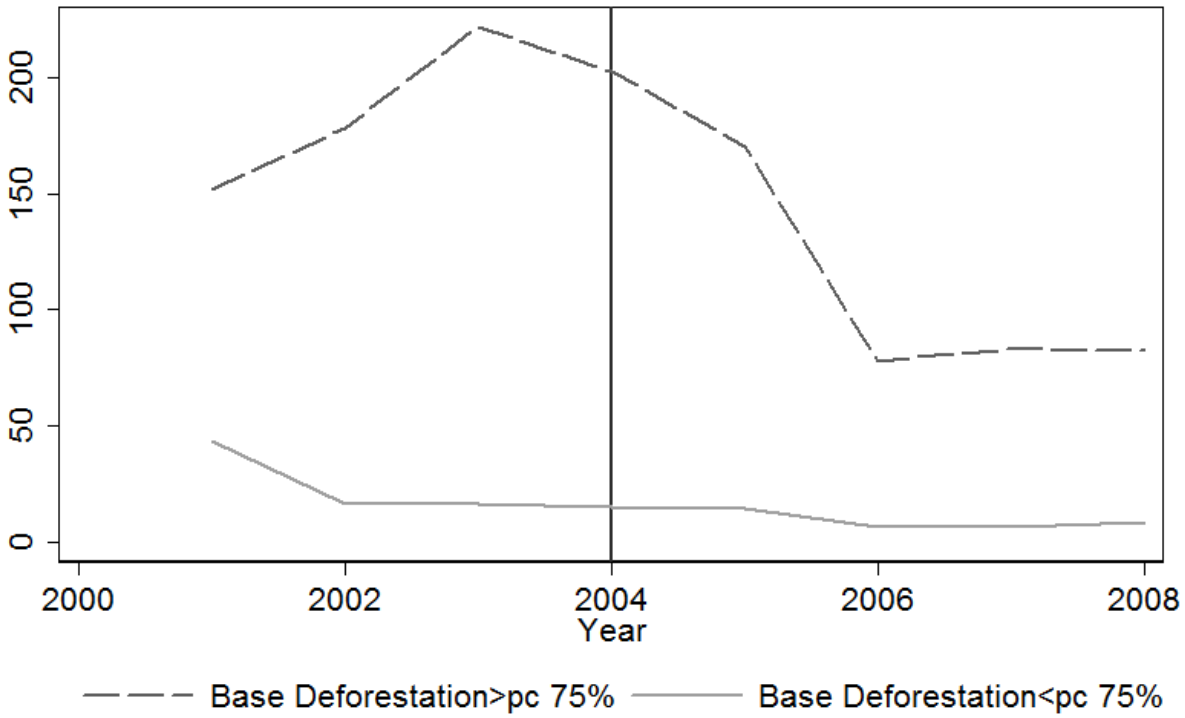
We should point out that there are various limitations to this study that should be addressed in future work. The most important one is that, given the data limitations, our estimates do not allow us to disentangle the channels that underpin the relationship between the conservation policy and infant health. Although, the evidence suggests that changes in air pollution are likely to be an important mechanism, we cannot rule out the possibility that other factors play a role. Our methodology provides only suggestive evidence of the combined effects of these channels. Future studies should empirically investigate the malaria risk channel. Data limitations do not allowed defining deforestation to exclude urbanization. This could be relevant given that previous studies document that urbanization leads to decreased risk of malaria. Moreover, it is not clear whether one should expect the estimated effects of reductions in deforestation to be the same across space and time as they may depend on the average health and nutritional status of pregnant women and specific environmental characteristics of each country or region. Setting aside the issue of generalizability, the fact that debates on conservation policies will become increasingly common implies that understanding the effects of controlling deforestation is an important question *per se*. We believe that future studies linking later socio-economic outcomes with birth records could yield even greater insights into the long-term impacts of Brazil's conservation policy.

Figure 1. Pre-intervention deforestation



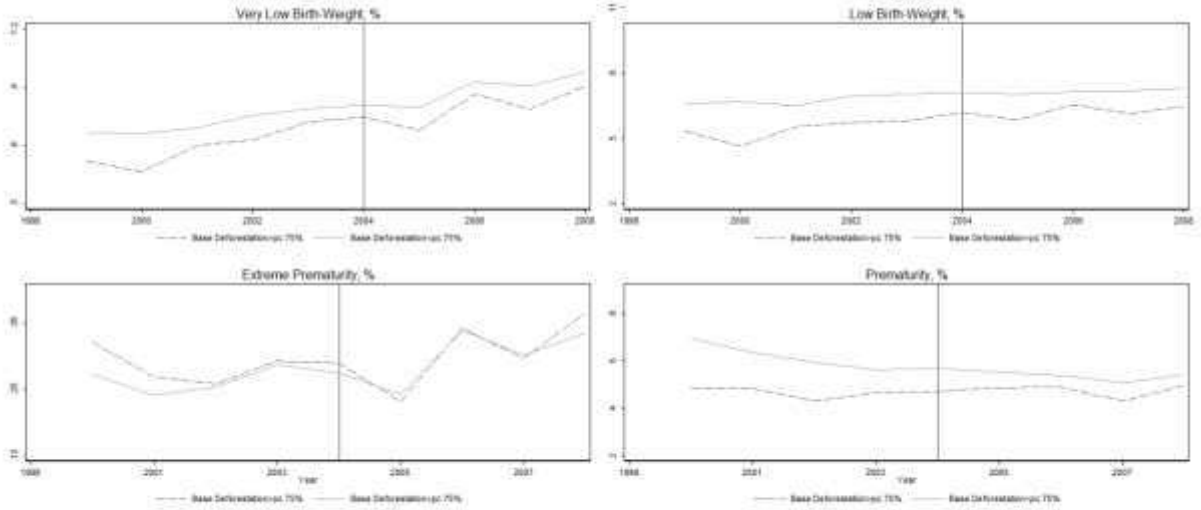
Note. This map displays the pre-intervention deforestation in km2 for each municipality in the sample

Figure 2. Deforestation Trends, 2001-2008



Note. Deforestation is defined as the annual forest cleared in km2. The municipalities were classified into Low and High categories based on their pre-intervention deforestation. The Low (High) category consists of municipalities under (over) the 75 percentile of the pre-intervention deforestation.

Figure 3. Trends in Birth Outcomes



Note. Deforestation is defined as the annual forest cleared in km2. The municipalities were classified into Low and High categories based on their pre-intervention deforestation. The Low (High) category consists of municipalities under (over) the 75 percentile of the pre-intervention deforestation.

TABLE 1

SUMMARY STATISTICS

	Number of Municipalities	Number of Observations	Mean	Standard Deviation
Number of births	396	4,356	715.86	2470.45
Very low birth-weight rate	396	4,310	0.60	0.69
Low birth-weight rate	396	4,310	5.89	2.43
Extreme prematurity rate	396	3,545	0.27	0.48
Prematurity rate	396	3,545	6.12	11.78
Maternal Characteristic:				
Teenage mother rate	396	4,307	31.45	6.08
Education years over 12 rate	396	4,310	5.55	5.24
Married mother rate	396	3,545	30.14	15.19

Note. Yearly observations by municipality from 1998 to 2008. The rates are expressed by 100 live births.

TABLE 2

EFFECTS OF PPCDAM ON DEFORESTATION

	(1)	(2)	(3)
Post *x* BaseDeforestation	-0.329	-0.309	
	[0.151]**	[0.153]**	
Year 2005 *x* BaseDeforestation			-0.314
			[0.188]*
Year 2006 *x* BaseDeforestation			-0.822
			[0.192]***
Year 2007 *x* BaseDeforestation			-0.887
			[0.27]***
Year 2008 *x* BaseDeforestation			-1.047
			[0.34]***
Effect at 75th Percentile of BaseDeforestation, (%)	-37.9	-35.73	
Observations	3,168	3,168	3,168
Additional controls	No	Yes	Yes
Municipality time trend	Yes	Yes	Yes

Note. The dependent variable is deforestation in km^2. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. All regressions include municipality and year fixed effects Additional controls include the annual index of crop prices, and the interactions between *Post* and municipality size, the average percentage of Bolsa Family beneficiaries, and the 2004 GDP. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ $p < 0.10$ $**$ $p < 0.05$, $***$ $p < 0.01$.

TABLE 3

EFFECTS OF PPCDAM ON INFANT HEALTH

	(1)	(2)	(3)	(4)	(5)
	Panel A: Dependent variable is Very Low Birth-Weight Rate				
Post *x* BaseDeforestation	-0.00049 [0.00022]**	-0.000601 [0.00025]**	-0.000505 [0.00025]**	-0.00052 [0.00025]**	-0.00054 [0.00026]**
Effect at 75th Percentile of BaseDeforestation, (%)	-18.54	-22.43	-18.87	-19.51	-20.33
Observations	4,310	4,310	4,310	4,310	4,307
	Panel B: Dependent variable is Low Birth-Weight Rate				
Post *x* BaseDeforestation	-0.00053 [0.00054]	-0.00084 [0.00063]	-0.00067 [0.00077]	-0.00069 [0.00078]	-0.00083 [0.00074]
Effect at 75th Percentile of BaseDeforestation, (%)	-2.12	-3.31	-2.65	-2.74	-3.29
Observations	4,310	4,310	4,310	4,310	4,307
	Panel C: Dependent variable is Extreme Prematurity Rate				
Post *x* BaseDeforestation	-0.00044 [0.00020]**	-0.00048 [0.00022]**	-0.00045 [0.00021]**	-0.00048 [0.00022]**	-0.00048 [0.00022]**
Effect at 75th Percentile of BaseDeforestation, (%)	-35.5	-38.64	-36.33	-38.80	-38.72
Observations	3,545	3,545	3,545	3,545	3,544
	Panel D: Dependent variable is Prematurity Rate				
Post *x* BaseDeforestation	-0.0012 [0.0016]	-0.0019 [0.0016]	-0.0027 [0.0018]	-0.0025 [0.0019]	-0.0027 [0.0020]
Effect at 75th Percentile of BaseDeforestation, (%)	-4.99	-8.06	-11.52	-10.73	-11.20
Observations	3,545	3,545	3,545	3,545	3,544
Post *x* Municipal size	No	Yes	Yes	Yes	Yes
Bolsa Familia	No	No	Yes	Yes	Yes
Crop price index	No	No	No	Yes	Yes
Maternal characteristics	No	No	No	No	Yes
Municipality time trend	Yes	Yes	Yes	Yes	Yes

Note. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. Bolsa Familia includes the interactions between Post and the average percentage of Bolsa Family beneficiaries, and 2004 GDP. All regressions include municipality and year fixed effects. Maternal characteristics include: % of teenage mother, % of married mother, and % of Mother with Education years> 12. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

TABLE 4

EFFECTS OF PPCDAM ON INFANT HEALTH BY GENDER

	(1) Very Low Birth-Weight Rate	(2) Low Birth-Weight Rate	(3) Extreme Prematurity Rate	(4) Prematurity Rate
Panel A: Boys				
Post *x* BaseDeforestation	-0.00090	-0.0015	-0.00070	-0.0026
	[0.00041]**	[0.00094]	[0.00032]**	[0.0021]
Effect at 75th Percentile of BaseDeforestation, (%)	-35.4	-6.64	-53.03	-11.20
Observations	4,307	4,307	3,544	3,544
Panel B: Girls				
Post *x* BaseDeforestation	-0.00027	-0.00048	-0.00028	-0.0024
	[0.000308]	[0.00098]	[0.00026]	[0.0018]
Effect at 75th Percentile of BaseDeforestation, (%)	-10.07	-1.73	-25.30	-10.09
Observations	4,306	4,306	3,543	3,543
Additional controls	Yes	Yes	Yes	Yes
Municipality time trend	Yes	Yes	Yes	Yes

Note. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. Aditional controls include: interactions between Post and the average percentage of Bolsa Family beneficiaries, and 2004 GDP; fixed effects for municipality and year; % of teenage mother, % of married mother, and % of Mother with Education years> 12. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

TABLE 5

EFFECTS OF PPCDAM ON AIR POLLUTION

	CO	CO	PM_{10}	PM_{10}	SO_2	SO_2
	(1)	(2)	(3)	(4)	(5)	(6)
Post *x* BaseDeforestation	-0.0008		-0.00082		-0.00041	
	[0.00040]**		[0.00041]**		[0.00023]*	
Year 2005 *x* BaseDeforestation		-0.0005		-0.0007		-0.0003
		[0.0003]*		[0.0003]*		[0.0002]
Year 2006 *x* BaseDeforestation		-0.00231		-0.00232		-0.00157
		[0.0009]***		[0.0009]***		[0.0006]***
Year 2007 *x* BaseDeforestation		0.00419		-0.00424		-0.00236
		[0.0011]***		[0.0012]***		[0.0008]***
Year 2008 *x* BaseDeforestation		-0.00489		-0.00495		-0.00289
		[0.0013]***		[0.0013]***		[0.0009]***
Effect at 75th Percentile of BaseDeforestation, (%)	-17.23		-17.68		-8.8	
Observations	4,356	4,356	4,356	4,356	4,356	4,356
Additional controls	Yes		Yes		Yes	
Municipality time trend	Yes		Yes		Yes	

Note. Dependent variables are in logs. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. All regressions include municipality and year fixed effects. Additional controls include the annual index of crop prices, and the interactions between *Post* and municipality size, the average percentage of Bolsa Family beneficiaries, and 2004 GDP. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ $p < 0.10$ $**$ $p < 0.05$, $***$ $p < 0.01$.

TABLE 6

EFFECTS OF PPCDAM ON GDP AND AGRICULTURAL ACTIVITY

	(1) GDP	(2) Agricultural Production	(3) Total Crop Production
Post *x* BaseDeforestation	-0.0001	0.00022	0.000109
	[0.00011]	[0.00021]	[0.000297]
Effect at 75th Percentile of BaseDeforestation, (%)	0.0930	1.035	2.373
Observations	3,551	3,551	4,271
Additional controls	Yes	Yes	Yes
Municipality time trend	Yes	Yes	Yes

Note. Dependent variables are in logs. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. All regressions include municipality and year fixed effects. Additional controls include the annual index of crop prices, and the interactions between *Post* and municipality size, the average percentage of Bolsa Family beneficiaries, and 2004 GDP. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ $p < 0.10$ $**$ $p < 0.05$, $***$ $p < 0.01$.

Appendix A: Additional data description, tables and figures

A1. Air pollution data

We build series for PM_{10}, CO y SO_2 using data from the Emissions Database for Global Atmospheric Research (EDGAR). This dataset provides worldwide annual estimates for various air pollutants (measures in tonnes) at the 0.1 *x* 0.1 degree latitude/longitude grid (0.1 degree correspond roughly to 11 kilometers). Using spatial interpolation methods, EDGAR computes values for each grid node from different emission sources (such as fuel combustion, industrial process, savanna burning, waste burning, forest fires, fossil fuel fires, etc.). We used geospatial software to aggregate the pollutant data to municipality-year level for the period 1998-2008. The mean per municipality per year of levels of PM_{10}, CO and SO_2 are 4,703 (Standard Deviation=12,560), 39,191 (Standard Deviation=103,200), and 678 (Standard Deviation=1,365), respectively.

A2. Crop price index

In this section, we described the construction of an annual index of crop prices at municipality level. Our procedure follows that of Assunção and Rocha (2015). As these authors, we use crop prices collected in the Agriculture and Supply Secretariat of the State of Paraná (SEAB-PR) for the period 1998-2008 since crop prices are likely to be endogenous to local productions. Paraná is a state that is not part of the Brazilian Amazon, but the average weighted prices across municipalities in the sample are highly correlated with price from SEAB-PR. The commodities include soybean, cassava, rice, corn and sugarcane. These crops are predominant in the Amazon, representing about 70% of the total 2004 harvested area.

The index of crop prices is calculated in three stages. First, the nominal annual price of each commodity is deflated and expressed as an index, taking 2000 as base. Second, we calculate the price of each crop for each municipality:

$$WIP_{itc} = IP_{tc} x \overline{sCrop}_{ic}$$

Where WIP_{itc} is the real price of the crop *c* for municipality *i* and year *t*. $IP_{tc}$is the price index of crop *c* in year *t* built in the first stage. $\overline{sCrop}_{ic}$ is the average (for the period

1995-1997) ratio between the sowed area of crop *c* and the total sowed area. This term captures the importance of crop *c* for municipality *i*.

Third, the principal components technique is used to build the annual index of crop prices. We normalize such an index by subtracting the minimum value and dividing the resulting value by the difference between the maximum and minimum values. Thus, the annual index ranges from 0 to 100.

A3. Additional tables and figures

TABLE A1

EFFECTS OF PPCDAM ON INFANT HEALTH (YEARLY INTERACTION EFFECTS)

	(1) Very low birth weight	(2) Low birth weight	(3) Extreme prematurity	(4) Prematurity
		Panel A: Baseline estimates		
Post *x* BaseDeforestation	-0.00054	-0.00083	-0.00048	-0.0027
	[0.00026]**	[0.00074]	[0.00022]**	[0.0020]
Observations	4,307	4,307	3,544	3,544
		Panel B: Yearly interaction effects		
Year 2005 *x* BaseDeforestation	-0.00069	-0.0009	-0.00057	-0.0022
	[0.00028]**	[0.00076]	[0.000280]**	[0.0022]
Year 2006 *x* BaseDeforestation	-0.00022	-0.00050	-0.00035	-0.0035
	[0.00041]	[0.00091]	[0.00027]	[0.0025]
Year 2007 *x* BaseDeforestation	-0.00061	-0.0011	-0.00060	-0.0054
	[0.00028]**	[0.0010]	[0.00032]*	[0.0036]
Year 2008 *x* BaseDeforestation	-0.00060*	-0.00025	-0.00067	-0.0048
	[0.00030]	[0.0014]	[0.00039]*	[0.0045]
Observations	4,307	4,307	3,544	3,544

Note. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. Additional controls include: interactions between Post and the average percentage of Bolsa Family beneficiaries, and 2004 GDP; fixed effects for municipality and year; % of teenage mother, % of married mother, and % of Mother with Education years> 12. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ $p < 0.10$ $**$ $p < 0.05$, $***$ $p < 0.01$.

TABLE A2
EFFECTS OF PPCDAM ON INFANT HEALTH
(ALTERNATIVE SPECIFICATIONS)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Panel A: Dependent variable is Very Low Birth-Weight Rate						
Post *x* BaseDeforestation	-0.00054 [0.00026]**	-0.00080 [0.00024]***	-0.00051 [0.00023]**	-0.00060 [0.00023]**	-0.00054 [0.00024]**	-0.00058 [0.00023]**	-0.00077 [0.00025]***
Observations	4,307	3,552	4,209	4,209	4,209	4,209	3,462
	Panel B: Dependent variable is Low Birth-Weight Rate						
Post *x* BaseDeforestation	-0.00083 [0.0007]	-0.0019 [0.0009]**	-0.0011 [0.00076]	-0.0010 [0.00073]	-0.00099 [0.00077]	-0.0010 [0.00077]	-0.0023 [0.00108]**
Observations	4,307	3,552	4,209	4,209	4,209	4,209	3,462
	Panel C: Dependent variable is Extreme Prematurity Rate						
Post *x* BaseDeforestation	-0.00048 [0.00022]**	-0.00048 [0.00021]**	-0.00043 [0.00021]**	-0.00051 [0.00022]**	-0.00048 [0.00023]**	-0.00049 [0.00022]**	-0.00041 [0.00022]*
Observations	3,544	3,170	3,446	3,446	3,446	3,446	3,080
	Panel D: Dependent variable is Prematurity Rate						
Post *x* BaseDeforestation	-0.0027 [0.0020]	-0.0028 [0.0020]*	-0.0023 [0.0021]	-0.0014 [0.0021]	-0.0013 [0.0021]	-0.0014 [0.0021]	-0.0026 [0.0021]
Observations	3,544	3,170	3,446	3,446	3,446	3,446	3,080
Education and health spending share	No	Yes	No	No	No	No	Yes
Post *x* Child labor rate	No	No	Yes	No	No	No	Yes
Post *x* Illiteracy rate	No	No	No	Yes	No	No	Yes
Post *x* Sanitation coverage	No	No	No	No	Yes	No	Yes
Post *x* Physicians per capita	No	No	No	No	No	Yes	Yes
Baseline controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Municipality time trend	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Note. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. Baseline controls include: interactions between Post and the average percentage of Bolsa Family beneficiaries, and 2004 GDP; fixed effects for municipality and year; % of teenage mother, % of married mother, and % of Mother with Education years> 12. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

TABLE A3

EFFECTS OF PPCDAM ON INFANT HEALTH (MEAN REVERSION)

	(1) Baseline specification	(2) Lagged dep. var	(3) Arellano-Bond
	Panel A: Dependent variable is Very Low Birth-Weight Rate		
Post *x* BaseDeforestation	-0.00054	-0.0007	-0.0011
	[0.00026]**	[0.00026]***	[0.00035]***
One-year lagged Very Low Birth-Weight Rate		-0.22	-0.029
		[0.0203]***	[0.026]
Observations	4,307	3,912	3,515
	Panel B: Dependent variable is Low Birth-Weight Rate		
Post *x* BaseDeforestation	-0.00083	-0.0014	-0.0019
	[0.00074]	[0.00091]	[0.00099]*
One-year lagged Low Birth-Weight Rate		-0.180	-0.0186
		[0.0327]***	[0.0409]
Observations	4,307	3,912	3,515
	Panel C: Dependent variable is Extreme Prematurity Rate		
Post *x* BaseDeforestation	-0.00048	-0.00041	-0.00052
	[0.00022]**	[0.00022]*	[0.00024]**
One-year lagged Extreme Prematurity Rate		-0.249	-0.0749
		[0.0627]***	[0.0648]
Observations	3,544	3,148	2,752
	Panel D: Dependent variable is Prematurity Rate		
Post *x* BaseDeforestation	-0.0027	-0.00156	-0.000553
	[0.0020]	[0.00175]	[0.00179]
One-year lagged Prematurity Rate		0.109	0.221
		[0.0477]**	[0.0398]***
Observations	3,544	3,148	2,752
Baseline controls	Yes	Yes	Yes
Municipality time trend	Yes	Yes	Yes

Note. Effect at 75th percentile of BaseDeforestation is computed by multiplying the pre-intervention deforestation for the municipality in the 75th percentile of the pre-intervention deforestation by the coefficient on Post *x* BaseDeforestation, and dividing the resulting value by the mean of dependent variable. Baseline controls include: interactions between Post and the average percentage of Bolsa Family beneficiaries, and 2004 GDP; fixed effects for municipality and year; % of teenage mother, % of married mother, and % of Mother with Education years> 12. Robust standard errors in brackets are clustered at the micro-region level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

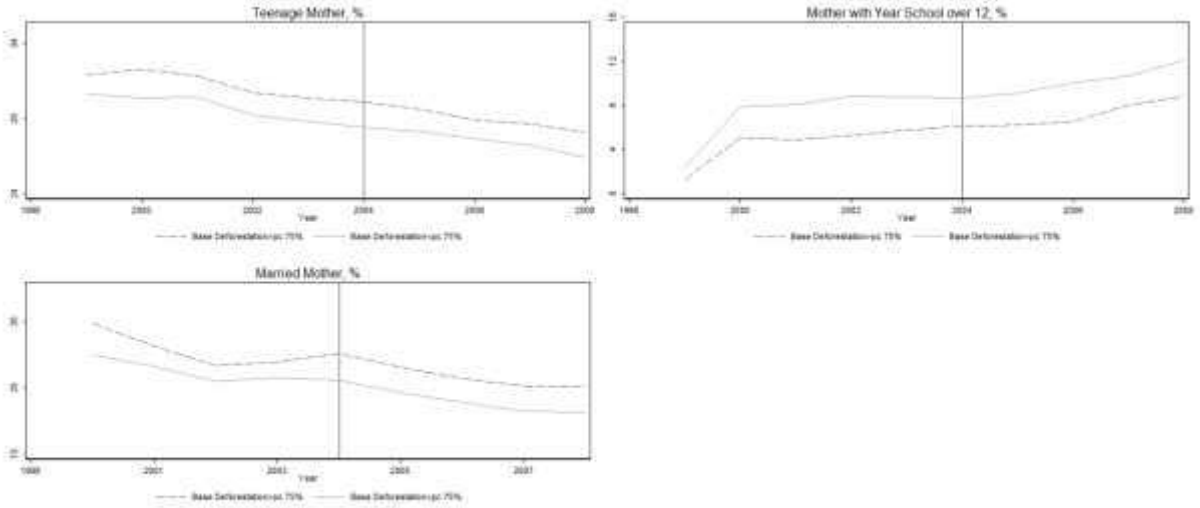
TABLE A4

FALSIFICATION TESTS AND EFFECTS OF PPCDAM ON FETAL DEATH AND SEX RATIO

	(1) Fetal Death Rate	(2) Sex Ratio	(3) % Teenage Mother	(4) % Mother's Education Years > 12.	(5) Pregnancy Rate
Post *x* BaseDeforestation	-0.000645	0.0000192	-0.0029	0.00274	-0.000862
	[0.000696]	[0.000068]	[0.00188]	[0.00348]	[0.00189]
Observations	4,313	4,308	4,307	4,310	4,317
Additional controls	Yes	Yes	Yes	Yes	Yes
Municipality time trend	Yes	Yes	Yes	Yes	Yes

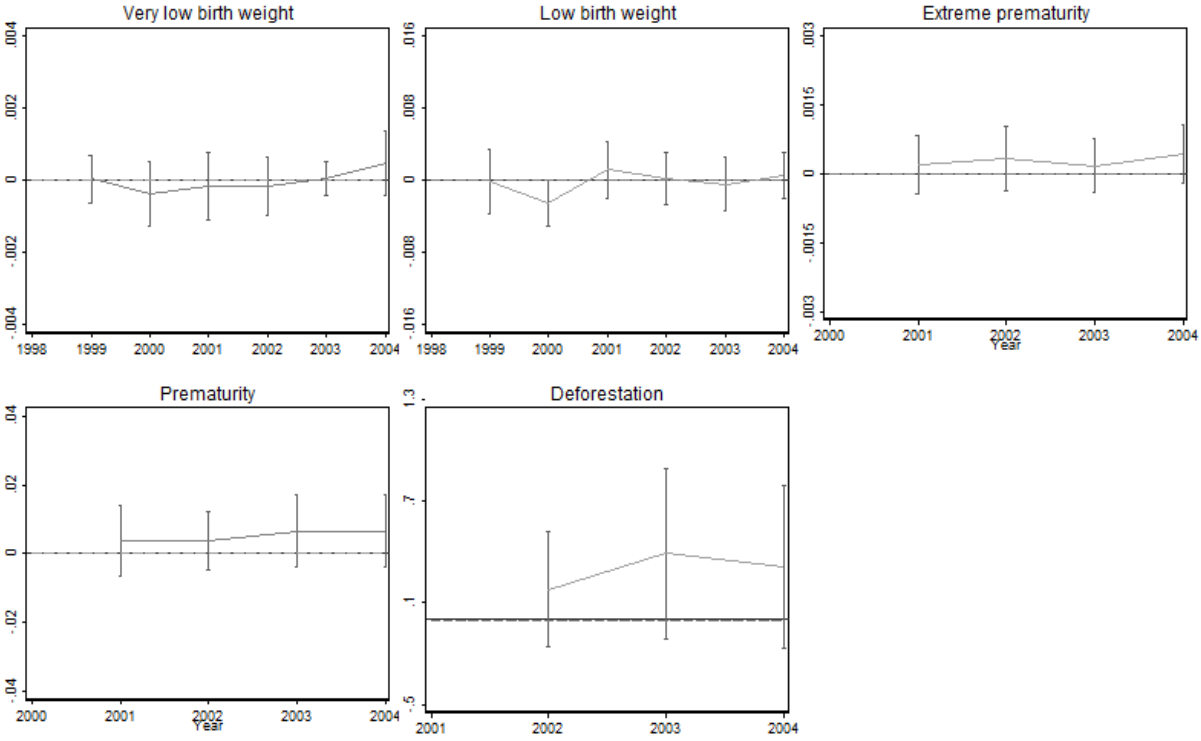
Note. All regressions include municipality and year fixed effects. Additional controls include the annual index of crop prices, and the interactions between *Post* and municipality size, the average percentage of Bolsa Family beneficiaries, and 2004 GDP. Robust standard errors in brackets are clustered at the micro-region level. In addition, columns (1)-(2) include controls regarding maternal characteristics. $*$ $p < 0.10$ $**$ $p < 0.05$, $***$ $p < 0.01$

Figure A1. Trends in Mother's Characteristics



Note. Deforestation is defined as the annual forest cleared in km2. The municipalities were classified into Low and High categories based on their pre-intervention deforestation. The Low (High) category consists of municipalities under (over) the 75 percentile of the pre-intervention deforestation.

Figure A2. Difference-in-Difference Coefficient in the Pre-intervention Period



Note. These figures plot coefficients and 95% confidence intervals from Equation (3).

4. The Effect of Physicians on Child Hospitalizations: Evidence from a Large-Scale Intervention in Brazil

Abstract

The question of whether increasing access to health care can improve child health is a basic concern for policy makers. While it is a widely held position that spending resources on increasing the availability of physicians is an effective way to improve child health, there is very little rigorous investigation documenting its quantitative importance. We exploit a dramatic change in policy to estimate the effect of physicians on child hospitalizations in Brazil. The results suggest that program implementation is associated with statistically significant reductions in avoidable hospitalizations only in poor areas, especially for children under 1 year old. Our estimates indicate that policy lead to a fall of 4.8 percent in avoidable child hospitalizations in treated municipalities. In addition, we find effects of the largest magnitude for gastroenteritis conditions as well as for emergencies related to asthma.

Keywords: Child hospitalizations; Physicians; Primary health care; Brazil.
*JEL Classification***:** I12, K32, Q51

4.1. Introduction

The question of whether increasing access to health care can improve child health is a basic concern for policy makers. A prominent body of work has documented that increasing health insurance coverage or providing financial incentives for health care is associated with better child health outcomes (AIZER, 2007; CURRIE; GRUBER, 1996; GRUBER; HENDREN; TOWNSEND, 2014). However, policies that affect primarily the supply side of the market have received much less attention in literature. A major indicator of health policy is the number of physicians serving in public health facilities. While it is a widely held position that spending resources on increasing the availability of physicians is an effective way to improve child health, there is very little rigorous investigation documenting its quantitative importance. We know very little whether policies of this sort are more or less important than other determinants of child health such as genetics, family background and exposure to poor environmental conditions. The absence of direct evidence on the role of physicians and other supply side factors in child health hampers the development of optimal health policies.

Policy efforts that seek increasing the number of physicians willing to provide primary health care presume that by doing so child health will unquestionably improve. In practice, this is not necessarily true. First, in many cases people expect a great deal of time to receive care they demand, so an increased number of physicians may reduce waiting time but this will not increase the number of ambulatory visits unless time is critical determinant of demand for health care. Second, as Currie, Gruber and Fischer (1995) noted, increasing the number of physicians may simply cause patients to substitute other sources of care for physician's office without affect the demand for health care.[29] Thus, whether physicians is in fact important for child health is an empirical question. But many of existing studies are based on correlations between the number of physicians and various indicators of child health that might be subject to important omitted variable bias, limiting the power of policy implications.

We exploit a dramatic change in policy to estimate the effect of physicians on child health in Brazil. In 2013, the Brazilian government launched a major public health program, the Programa Mais Medico (PMM), aimed to place physicians in areas with shortage of doctors. To attract foreign and Brazilian newly graduated physicians, the program offered exceptional and flexible conditions for participation. For example, a foreign physician did not need to have a registered diploma in Brazil for enrollment. Furthermore, physicians who participate in the program receive a monthly wage, tax-free, well above the average wage for newly graduated physicians. The program was implemented in municipalities that the government considered priority, approximately 70 percent of all municipalities. All doctors enrolled in the program were placed in Basic Health Units (UBS), where families have free access to primary health care services such as medical consultations, vaccines, dressings and health counseling to provide prevention and early detection of diseases.[30] This policy led to an unprecedented and immediate increase in the number of physicians willing to provide primary health care. The number of physicians per 1,000 residents increased by 50 percent in less than 8 months since implementation in treated areas. This was in direct

[29] Alternatives sources of health care include going to private providers, purchasing medicine without a prescription or using traditional medicine.

[30] The Basic Health Units (UBS) is a health facility where primary care teams provide health basic services. The UBS are usually within neighborhoods and cover a certain region, are close to where people live.

contrast to the less than 1 percent increase in the pretreatment years and the tiny increase in untreated areas during the post-treatment years.

Using comprehensive data on the universe of hospital discharge abstracts compiled monthly between January 2008 and December 2016, we linked child hospitalization outcomes with municipality-level data on policy. Since policy adoption might be endogenous, this study combines a matching procedure with a difference-in-difference estimator for identification. We first use an entropy balancing algorithm developed by Hainmueller (2012), which creates weights that makes control and treatment groups virtually identical in terms predetermined covariates. This technique has been shown to balance covariates in a more effective way than conventional propensity score methods. We apply this approach to a large set of pretreatment characteristics to increase the comparability of municipalities, in particular to minimize the likelihood of differential trends driven by other factors. We then employ a difference-in-difference empirical design that compares the evolution in child health outcomes in treated and reweighted untreated areas before and after policy adoption. This empirical strategy relies on the assumption that in the absence of the PMM treated and reweighted untreated areas would have experienced the same proportional changes in child health. We provide several pieces of evidence that are supportive of the validity of this identifying assumption. In particular, we show that in the pre-intervention period treatment and control groups presented identical trends in child health outcomes and in a large set of observable characteristics.

Our emphasis is on hospitalizations that have been denoted by medical literature as "avoidable". These include immunization preventable conditions, gastroenteritis, nutritional deficiencies, and asthma, among others. According to medical experts, hospitalizations due to these conditions may be avoided if timely and effective ambulatory care is received (EPSTEIN, 2001; GADOMSKI; JENKINS; NICHOLS, 1998; GILL; MAINOUS, 1998; PARCHMAN; CULLER, 1994; SHI et al., 1999). For instance, adequate practices in primary health care are considered essential for preventing emergencies related to asthma. Early access to preventive vaccinations is crucial during infectious disease outbreaks. Similarly, the vast majority of emergencies related to gastroenteritis may be avoided by improving parent's knowledge of inexpensive preventive health care such as practicing good hygiene. If families respond

to increased local supply of physicians by increasing the number of regular ambulatory care before their children become sick, then the number of avoidable hospitalizations should decline. On the other hand, an "access" effect may still operate through inpatient care if visits to doctor's office lead to referrals to specialists for additional evaluation and potential invasive procedures. Other things equal, this mechanism would imply that hospital admissions increase with access to primary health care.[31] If presents, our reduced form estimate of the effect of the PMM would provide a direct test of the relative importance of both mechanisms.

Public health policy is increasingly concerned with hospitalizations that result from inadequate access to primary health care because they account for a large portion of hospitalizations among children. The focus of our paper, Brazil, is one striking example. Between 2008 and 2016, avoidable, ambulatory care sensitive hospitalizations accounted for a portion of hospitalizations among children as large as 39 percent.[32] During the same period, Federal, State and local expenditures on these hospitalized children exceeded USD $50 million annually. Thus, reducing the incidence of these conditions is compelling on the grounds of economic efficiency.

We begin by investigating formally the impact of policy on physicians. The difference-in-difference results indicate that policy adoption lead to a rapid and robust increase in the number of physicians in UBSs, confirming the overall pattern in Figure 1. The number of physicians jump upward within the first 8 months since policy implementation for treated areas and stays elevated thereafter, relative to the pre-intervention period and control areas. The effects are large in magnitude. For example, we find that, relative to the control group, the number of physicians in UBSs per 1,000 residents in treated areas increased by 0.10 during the first months after policy adoption, relative to a pre-intervention mean of 0.20.

We then turn our focus to the hospital discharge data to assess how policy adoption affected avoidable, ambulatory care sensitive hospitalizations among children.

[31] There is evidence suggesting that this access effect may be important in practice. For example, Kaestner and Sasso (2015) document that greater outpatient spending is associated to increased inpatient events and to more hospital admissions in U.S.

[32] This is in direct contrast to U.S, where the share of hospitalizations that are avoidable is about 25 percent (AIZER, 2007).

The results suggest that program implementation is associated with statistically significant reductions in avoidable hospitalizations only in poor areas, especially for children under 1 year old. Our estimates indicate that policy lead to a fall of 4.8 percent in avoidable child hospitalizations in treated municipalities. When we group causes of hospitalizations into broad, mutually exclusive categories, we find effects of the largest magnitude for gastroenteritis conditions as well as for emergencies related to asthma. In particular, policy adoption is associated with a 7 percent reduction in hospitalizations due to gastroenteritis complications, within 15 months after implementation. Using the value of professional services for care of children, we also found a statistically significant reduction only in poor municipalities. Policy adoption is associated with a reduction of $150 Reais (USD $50) in the cost for care of hospitalized children due to avoidable conditions.

If we are willing to invoke the exclusion restriction that policy implementation only affects children's hospitalizations through physicians, we can combine our results to produce instrumental variable estimates of the impact of physicians on child health. By doing so, it shows that a 10 percent increase in the number of physicians implies a reduction of 1.6 percent in the incidence of avoidable hospitalizations among children. However, we should emphasize that these instrumental variable estimates should be only taken as an exploratory effort to understand how physicians affect hospitalization outcomes, since it cannot completely to rule out the possibility that other unobserved mechanism such as physician quality played a role.

Besides the evidence that treatment and control groups were on parallel trends prior to policy, a variety of falsification checks provide strong support for a causal interpretation of our results. First, we find that the effects of program on physicians, in either public or private sector, working outside of UBSs can be bounded to a tight interval around zero. For instance, we can rule out a policy effect on public physicians who do not work in UBSs one fourth as large as the effect of program on physicians in UBSs. This finding is important because these physicians were not target of policy. Second, we also assess whether program coincided with changes in other health resources as proxied by the number of UBSs, hospitals, rest beds, and a set of medical equipment. The analysis reveals virtually zero policy effects on each of these outcomes. Third, we also check the internal consistency of our results by using hospitalizations

due to “external” causes (e.g., accidents) as placebo. Since it is difficult to link morbidity from those causes to differences in medical inputs, statistically significant policy effects in such regressions would be of concern. Reassuringly, we find no statistically significant change in external hospitalizations associated to policy adoption. Finally, we continue to find no placebo effects of policy on each set of outcomes when we stratify the sample by municipality income as proxied by per capita GDP.

This study addresses the absence of direct empirical evidence on the role of policies of this sort outside the United States, especially in poor countries. Even within the United States, there is a limited number of studies concerned with the effects of increasing the number of physicians on child health. A comprehensive survey of literature by Leininger and Levy (2015) identified only three studies using quasi-experimental methods to estimate the effects of increased number of physicians on children. Two of these studies explore the impact of policy on dental care use among children, which is an input rather than a measured of child welfare. The only study examining direct measures of child health is that of Currie, Griber and Fischer (1995), who examine the effect of increasing the availability of physicians serving to poor on infant mortality. While the focus on infant mortality is compelling, this is a relatively extreme outcome that reflects some of the most severe consequences of inadequate access to health care. But PMM’s impacts are consistent with evidence from U.S. showing that an increased number of doctors lead to better child health among poor families.

The remainder of the paper proceeds as follows. Section 2 presents a background on policy and a brief literature review of related studies. Section 3 describes the data and Section 4 presents the methodology. Sections 5 present our results as well as a series of robustness checks and section 7 present an interpretation of our findings. Finally, Section 7 concludes.

4.2.Background

4.2.1. The Mais Medicos Program

Prior to the *Mais Medicos* program (PMM), Brazil had notable shortage of doctors, with an estimated of 1.8 physicians per 1,000 inhabitants. This figure is significantly low if compared to that of OECD countries, which have on average 3.7

doctors per person. This relatively low rate of doctors was accompanied by striking differences across regions. Only five states out of twenty-seven had more than 1.8 doctors per inhabitants. The number of doctors per capita was the lowest in the poorer regions, especially in small cities (see Figures 2 and 3). At the same time, the country had fewer newly graduated physicians than the annual job creation in the public and private sectors. Between 2002 and 2012, the labor market opened 143,000 new formal medical job openings, but medical schools only graduated 93,000 new physicians (UFMG, 2009).

Pioneering efforts to combat the shortage of doctors in Brazil began in 2011, when the Ministry of Health made a national call for newly graduated doctors to work in the poorest municipalities, offering salaries above the labor market average.[33] While this initiative guaranteed the participation of about 3,000 physicians in 2013, it was unable to meet the annual demand for doctors in the public sector.[34] Another initiative was the expansion of scholarships for medical residency.[35] However, such scholarships were offered only for medium and large cities. In general, these initial efforts were important but unable to eliminate the shortage of physicians in the country.

At the beginning of 2013, Brazil witnessed a strong movement of mayors, called *Cadê o médico?*, demanding the federal government to take actions to address the lack of physicians. As a result, in January 2013, the then President of Brazil Dilma Rousseff ordered the Minister of Health and his team to study and formulate a proposal to respond to the demands of the municipalities. Then, *Mais Medicos* program was created through the Provisional Measure 621, published on July 8, 2013, and regulated, in October of the same year, by the Federal law 12,871/2013.

The goal of the PMM is to increase the number of physicians per 1,000 inhabitants from 1.8 in 2013 to 2.7 in 2026. In order to deal with the shortage of doctors

[33] Municipality is a second-order administrative unit similar to U.S counties.

[34] A study by the Federal University of Minas Gerais (UFMG) estimates that the annual demand for physicians in the sector public is about 10,000.

[35] In Brazil, medical residency is a gradation-teaching modality for physicians, under a form of specialization course. Working in health institutions such as school-hospitals, graduate students engage in paid activities in a health facility. According to the Human Resources Observatory of São Paulo, 82 percent of doctors remain in the same place where they attended medical residency.

in the short-term, the PMM defined an emergency provision plan. Such a plan was intended to bring physicians, in the perspective of a teaching-service integration, to municipalities with greater need and difficulty in attracting these professionals. In a given municipality implementing the PMM, each doctor is placed in a Basic Health Units (UBS), where population has free access to primary health care services such as medical consultations, small urgencies, vaccines, dressings and promotion of healthy habits.[36] Furthermore, physicians who work in a UBS deliver home visits to families to prevent diseases and accompany health of people in the community who do not seek health care.

The Federal law 12,871/2013 allowed for municipalities to voluntarily set up the PMM by signing an agreement that states specific responsibilities. Consequently, PMM adoption may not be exogenous. We return to this issue below. Program take-up was high, with over 80 percent of those municipalities invited choosing to attend. In total, approximately 4,000 municipalities out of 5,588 joined the program.

Having signed the agreement, it establishes the maximum number of physicians that will be allowed to enroll in the program. While the program gave priority to professionals with registered diploma in Brazil, doctors with unregistered diploma were also given the opportunity to join the PMM. Foreign physicians who got their diploma from medical schools out of Brazil were also eligible for this program. All doctors enrolled in the program receive housing, food and displacement benefits and a monthly salary, tax free, of USD $3,200.[37] Each physician signs a three-year contract extendable for another three years, with a workload specifying that physicians must dedicate 32 hours per week providing health services at the UBS and 8 hours per week in education activities. In general, these working conditions are well above the average labor market conditions for newly graduated physicians in the country. In the case that the number of physicians interested in participating is less than the maximum number required, the Ministry of Health is allowed to make cooperation agreements with international

[36] The Basic Health Units (UBS) is a health facility where primary care teams provide health basic services. The UBS are usually within neighborhoods and cover a certain region, are close to where people live.

[37] For reference, the 2014 minimum wage in Brazil was USD $231

organizations to fill the remaining positions. This was rarely the case given the flexible requirements for participation and working conditions.

In general, the program was extremely successful in the short run. During the two first years the program recruited more than 18,000 Brazilian and foreign physicians, which represented an unprecedented annual increase in the number of doctors in Brazil. To take a closer look at these data, consider the Figure 1. It shows that the trends in the number of physicians working in a UBS per inhabitants in the municipalities implementing the PMM did not resemble the trends in the municipalities non-enrolled in the program after policy implementation. The treated municipalities' increase in the physician per capita ratio during the post policy period was the most rapidly increase on record in Brazil. From 2008-13 to 2014-16, physician rate was increased by about 0.097 physicians per 1,000 inhabitants in the treated group and about 0.021 physicians per 1,000 inhabitants in the control group, a difference of 0.076 physicians. This crude difference-in-difference is generally consistent with the PMM effects that we estimate more formally using a more sophisticated specification.

To meet the long-term goal, the PMM also implemented two further sets of actions. The first one aims to make investments in improving the infrastructure of the healthcare network. For that, the PMM seeks to modernize, expand and build new UBS, with an estimated total cost of USD $1.3 billion. The second set of actions aims to guarantee an adequate annual number of newly graduated physicians with a training appropriate to the needs of the Brazilian population. For that, the Federal Government seeks to create 11,500 new vacancies for undergraduate studies in medical schools and 12,400 new medical residency positions by 2017. Furthermore, the PMM is making a deep restructuration of what country's undergraduate medical schools should teach. Besides updating medical education, the objective of this measure is to integrate teaching-research-extension and make medical training at undergraduate level addressed to the needs of the population and the Brazilian health system.

As discussed above, municipalities were allowed to voluntarily implement the PMM. If variation in adoption is systematically related to municipality characteristics that are associated with differential trends in child health outcomes, then this could lead to spurious estimates of the program impact. To explore this issue, we compiled a set of geographic and pretreatment socioeconomic characteristics of municipalities. We then

use these predetermined characteristics to predict the probability that the municipality adopted the program by using logit and OLS regression models. The independent variables include UBSs per person, physicians per inhabitants, per capita GDP, percentage of indigenous population, Gini index, unemployment rate, illiterate rate, share of rural population, log of population, municipality area, altitude, distance to capital, temperature, rainfall and dummies for whether the municipality is part of legal Amazon and semiarid regions. Appendix Table A1 describes in more detail these time-invariant variables.

We present the results in Appendix Tables A2-A3 In all regressions, we include State fixed effects and use robust standard error clustered at the mesoregion-level.[38] We find that PMM adoption is significantly associated with pretreatment physician and UBS rates. In general, municipalities with lower physicians per person are more likely to implement the program. The patterns regarding UBS per capita are unclear, with positive and negative coefficients depending on the year in which this variable is measured. We suspect that this is due to multicollinearity problems, given the persistence of UBS over time. When we estimate the models separately for each year in which UBS is measured, we find a consistent pattern suggesting that municipalities with lower UBS per person were more likely to participate in the program. Looking at the other variables, we find that municipalities with lower per capita GDP, higher illiterate rates, larger population and larger size are more likely to implement the PMM. In addition, those municipalities that are part of legal Amazon region and municipalities with a larger share of the population that is rural are also more likely to participate in the program. The rest of variables are not statistically significant in any of the specifications.

Although these results suggest significant effects of predetermined characteristics on PMM adoption, the quantitative importance of each variable is small. For example, a one-standard-deviation increase in per capita GDP is associated to a decrease of 2.5 percentage points in the PMM adoption probability, with an estimated elasticity of only -0.007. Similar magnitudes are found for the other variables. More remarkably, these characteristics explain only 15 percent of the total variation in

[38] Mesoregion is a subdivision of the Brazilian states that congregates several municipalities of a given geographic area with similar economic and social characteristics. There are 108 mesoregions. They were created by the Brazilian Institute of Geography and Statistics and are used for statistical purposes.

program adoption, leaving a substantial portion of variation unexplained. Given the large set of variables considered, we interpret this as strong evidence that in practice much of the variation in PMM adoption appears to be idiosyncratic. While this is a strength for adequately identifying PMM impacts, we conservatively create balancing weights to control for possible differential trends across municipalities that may be spuriously correlated with PMM effects, and all of our main difference-in-difference regressions use these balancing weights.

The PMM improved the access to primary health care services. This is particularly true for poorer and remote sites, where prior to the PMM a family would have to travel to distant facilities in other municipalities for free preventive and primary health services. Consequently, a basic prediction is that this increased access to primary health services lead to higher use of preventive health care and better child health outcomes for children living in treated municipalities. The focus of our paper is to explore this hypothesis by examining the PMM impacts on avoidable child hospitalizations, an outcome that is presumably responsive to preventive and primary health care.

4.2.2. Related literature

A large literature has examined the effects of policies that promote access to medical care on children's health. Most of this literature has focused on policies that affect primarily the demand side of the market, namely children and their families. Examples of these studies include those investigating the effects of providing free or subsidized health insurance and financial incentives for use of preventive services. These studies find consistent evidence that increased access to health care services through these policies lead to better child health outcomes, measured by infant mortality and self-reported health status (AIZER, 2007; CURRIE; GRUBER; FISCHER, 1995).

While these studies have advanced our knowledge of the effects of these policies, we know very little on the effect of policies designed to increase the supply of medical care. The understanding of the effects of such policies is necessary for cost-effective program comparisons and guiding the targeting of future policies intended to improve child health. Many of existing studies concerned with the effects of directly expanding access to primary health care services are correlational. Furthermore, these

studies tend to focus on behavioral health outcomes and there is limited evidence on other child health outcomes such as infant mortality and hospitalizations.

One notable exception is Currie, Gruber and Fischer (1995), who study the effects of increased payments to health-care providers on infant mortality in US. Specifically, they analyzed the reimbursement from Medicaid to health-care providers, a policy that aimed to increase the number of physicians willing to serve publicly insured families in US. They exploited within-State variation in Medicaid fees and show that increases in the ratio of Medicaid to private fees for obstetricians and gynecologists are associated to significant reductions in infant mortality. Using a similar approach, other studies provide evidence for positive effects of Medicaid fees on dentists' participation in the program and the utilization of dental care services among children (BUCHMUELLER; ORZOL; SHORE-SHEPPARD, 2015; DECKER, 2011).

Other studies have been concerned with the effect of placing health-care providers in schools on students' health. Using variation in within academic years in a Florida school district's, Carrell and Carrell (2006) show that lower student to counselor ratios lead to better behavioral health outcomes, such as disciplinary problems and incidents. Reback (2010) finds similar results for other behavioral outcomes in Alabama, including suspension rates and weapons-related incidents. In a companion paper, Reback (2010b) shows that changes in state-level elementary school counselor policies lead to increases in counselor policies and decreases in behavioral measures reported by teacher. While this body of evidence is important, these studies did no considered other health outcomes.

In a recent study, Bailey, Goodman-Bacon (2015) use the rollout of the first Community Health Centers (CHCs) to investigate the long-term effects of increasing access to primary care in US. While the central focus of their paper is among adults 50 or older, they also provided some evidence of the effects of this intervention on children health. They find little evidence that CHCs affected infant and child mortality rates and argue that this is because the main causes of deaths among children during the study period were unlikely to be affected by the CHCs. Lovenheim, Reback and Wedenoja (2016) find that the expansion of school-based health centers (SBHCs) in the US since early 1990's lead to significant reductions in fertility and high school dropout rates. The authors suggest that these effects were primarily driven by the SBHCs that provided

birth control policies. There is also correlational evidence that increased funding to community health centers is associated to increases of behavioral health services offered by the clinics in US (LO SASSO; BYCK, 2010). Finally, some studies find that policies that offers free vaccines for children are associated with increases in immunization rates and decreases in fragmentation of care (LEININGER; LEVY, 2015).

4.3. Data and summary statistics

Our study period begins in January 2008, nearly 68 months before the PMM took effect, and continues through December 2016. The basic source is from the Brazilian National System of Hospital of Information (or SIHSUS for its Portuguese acronym), which has been collecting high-frequency data on hospitalizations in Brazil. Appropriate offices at the municipal level are responsible for collecting these data from the hospitals and sending them to the Ministry of Health, which finally consolidates the information in the SIHSUS microdata. This database includes information on hospitalization characteristics and patient characteristics. Clinical information includes diagnosis and procedures codes, charges incurred during hospitalization, discharge status, date of hospital admission and hospital location. Demographic information includes municipality in which patient lives, birthdate, sex, age, and race. This section describes the main data we used in this study. In Appendix, we describe in more detail other data sources used for supplementary analyses.

Our primary outcome of interest is avoidable hospitalization among children under five years old, which medical experts have defined it as hospitalizations that are likely to result from inadequate access to basic health services. Ordinance 221, April 2008, establishes a Brazilian list of hospitalizations considered as “avoidable”, which was elaborated by the Ministry of Health. In Appendix Table A4, we present this list in detail. Since this is a very general list, it may include conditions that are extremely rare among children. For example, in our study period, hypertension conditions represents less than 0.001 percent of hospitalizations among children, whereas that for adults 50 or older this rate is around 3 percent. The Ministry of Health created this list considering the set of avoidable hospitalizations in other countries and the need to create a list that reflects the diversity of health and disease conditions in Brazil. In creating this list, the Ministry of Health seek to provide an input for evaluation of the health system through the use of primary care services and the use of hospital care.

Of the 8.2 million child hospitalizations that occurred during the study period, 39 percent were classified as avoidable under this definition. In the last column of Appendix Table A4, we provide estimates of the share of each specific avoidable hospitalization condition in the total. The leading cause of avoidable hospitalization is complications from infectious gastroenteritis (33 percent), followed by immunization preventable conditions (13.50 percent), asthma (13.40 percent) and bacterial pneumonia (12.10 percent). Certainly, our study is not the first to use avoidable hospitalizations to examine impacts of policy. For example, Dafny and Gruber (2005) estimate the effect of Medicaid expansion on avoidable hospitalizations among children in U.S.

Using information about the municipality in which children live, we collapse the microdata to the municipality-by-month-by-year-by-sex level. Each cell contains the number of avoidable hospitalizations among children in a given municipality, month, year and gender. To explore potential heterogeneous effects by age, we compute separately the total number of avoidable hospitalizations for three age categories for each municipality, month and year. The age categories are children under 1 year old, 1-3 years old, and 3-5 years old. In addition, we also compute the number of total avoidable hospitalizations for disease conditions groups in order to examine potential heterogeneous treatment effects by hospitalization cause. We group avoidable hospitalizations into six categories: immunization preventable conditions, complications from infectious gastroenteritis, bacterial pneumonia, asthma, pulmonary diseases and "other diseases". While this grouping appears somewhat arbitrary, it is based on the representativeness of each condition group in the total of avoidable hospitalizations

The SIHSUS database includes detailed information about value of hospital services from the universe of discharge abstracts. This value refers to the total charges incurred by the hospital for the care of hospitalized children. This paper uses this variable as a proxy for hospital cost. For consistency with our hospitalization analysis, we focus on the value of hospital services for hospitalizations that result from avoidable conditions. The total value of services are deflated to 2010 year Brazilian Reais.

We also use municipality-level data on number of physicians to study more directly how the PMM affected this outcome. Although we focus on the number of physicians working in UBSs, results for physicians who do not work in UBSs are also presented. The source of these data is from the Brazilian Ministry of Health and are

measured monthly from January 2008 to December 2016. We then compute the number of physicians per capita for each municipality, month and year. Since one of the PMM's strategy is to invest in construction of new UBSs, one possibility is that the number of these facilities were affected by the policy during the study period. While this is a PMM's long-term strategy, we collected monthly data on the number of UBSs per capita and explore potential short-term treatment effects of the PMM on this outcome. Other variables used for supplementary analysis include hospitalizations from injuries, medical equipment, rest beds and hospitals. This set of information is also provided by the Brazilian Ministry of Health, from January 2008 through December 2016.

Additionally, we also use time-invariant covariates at the municipality level in our empirical analysis to construct balancing weights, which are described below. The basic source of socioeconomic characteristics is from the 2010 demographic census, which is conducted every ten years by the Brazilian Institute of Geography and Statistics (IBGE). Geographic characteristics such as Amazon legal status, semiarid region status, altitude, distance to capital and municipal area are obtained from the Brazilian Institute for Applied Economic Research (IPEA). Finally, we obtain annual mean temperature and rainfall data from Rocha and Soares (2015), who consolidated these weather data at the municipality-level for Brazil using information from Matsuura and Willmott (2012).

We restrict the sample to those municipalities with non-missing information for the subset of pretreatment characteristics used to construct balancing weights. The rate of missing information is low, approximately 2.5 percent. In total, we have 5,439 municipalities in our data. Of these municipalities, 3,945 and 1,494 are in the treatment and control group, respectively. Descriptive statistics for the main variables are presented in Table 1. The mean of physicians in UBSs per 1,000 residents per municipality, per month, per year is 0.25. There is a wide variability in hospital cost, with standard deviation about 18,000 Reais (relative to a mean of 4,000 Reais). The mean of avoidable hospitalizations in the sample is 5.43. The mean in the number of avoidable hospitalizations is the highest for children 1-3 years old. On average, the number of hospitalizations from injuries is 1.5.

4.4.Research design

4.4.1. Empirical strategy

To identify the causal impact of the PMM on child health, we combine matching and difference-in-difference (DiD). The idea of the matching procedure is to increase the comparability between municipalities by simulating randomization with regard to assignment of the treatment. We then create a control group comprised of municipalities no exposed to the program but that have pretreatment characteristics similar to municipalities exposed to the program. In the DiD part, we compare the evolution of child health outcomes before and after policy adoption of municipalities in the control and treatment groups. The DiD procedure eliminates time-invariant hospitalization outcome differences between municipalities in the treatment and control group that result from unobserved variables.

To increase similarity between the two groups, we implement a reweighting technique, entropy balancing developed by Hainmueller (2012). This technique reweights the control group observations in such a way that the control group satisfies pre-specified balanced constraints involving sample moments of pretreatment characteristics. Here, our balanced constraint is equal means across treatment and control groups. There are other matching methods that may be used instead of the entropy balancing technique, such as propensity score methods that reweight control group observations by using weights that depend on score values (CALIENDO; KOPEINIG, 2008). Unlike other matching methods, entropy balancing assures that balance improves on all covariates between treatment and control groups even when the sample size is small and the number of pretreatment characteristics is large. Using Monte Carlo simulations, Hainmuleller (2012) compares the performance of entropy balancing to commonly used matching procedures and shows that entropy balancing has better performance in terms of estimation bias.[39]

We then estimate the following model using weighted least squares, where the weights are the entropy balancing obtained above:

[39] Traditional matching and weighting procedures include difference in means, propensity score matching, Mahalanobis distance matching, genetic matching, combined propensity score matching and Mahalanobis distance matching, and propensity score reweighting

$$y_{imt} = \alpha + \beta Post_{mt} * PMM_i + \eta_{im} + \mu_{mt} + \xi_{it} \quad (1)$$

where y is our dependent variable of interest, namely avoidable hospitalization outcomes for municipality *i*, at month *m* and year *t*. Since there many municipality-by-month-by-year observations in which avoidable hospitalizations are equal to zero, we parameterized our dependent variable as $ln(1+z)$.[40] We use a binary indicator for treatment, *PMM*, equal to one if the municipality implemented the PMM and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016. η_{im} are municipality-by-month fixed effects and μ_{mt} are year-by-month fixed effects. In all of our regression models, we use robust standard errors clustered at the mesoregion-level to count for any temporal and spatial dependence in municipalities within the same State.

The parameter of interest in equation (1) is β, which compares the change in hospitalization outcomes before and after the intervention in municipalities that were exposed to the program against the same change for municipalities with similar pre-treatment characteristics that were no exposed to the intervention. As avoidable hospitalizations is in logarithmic form, coefficient estimates can be approximately interpreted as percentage changes.

Our key identifying assumption is that in the absence of the PMM, municipalities in the treatment and reweighted control group would have experienced the same proportional changes in the outcomes. The balancing entropy technique is a basic step to increase the comparability between the two groups and limit potential differential trends. Of course, a natural question that arises is that treatment and control groups might differ in characteristics beyond the set of covariates employed in the entropy balancing approach that may affect our outcomes. Any such characteristics that are time-invariant will be absorbed by the robust set of municipality-by-month fixed effects. Only differential trends in time-variant unobservable factors across municipalities would be a threat to our DiD identification strategy. We return to this issue below.

[40] Given the size of the dataset (about 1,000,000 observations) and the large number of right-hand observations (due to the inclusion of municipality-by-month-by-sex and year-by-month fixed effects), we are unable to estimate nonlinear count models.

4.4.2. Balancing tests

Table 2 shows the sample's summary statistics for outcomes determined before launching the PMM, which should not be affected by whether the municipality adopted or not the program. Columns (1) and (2) report the summary statistics (mean and standard deviations) for treated and untreated municipalities before the matching procedure. Column (3) reports *p*-values for a test of the null hypothesis that the means are identical, which is a simple OLS regression on a dummy for policy adoption. The robust standard errors are clustered at the mesoregion level.

Treated areas differ in several aspects significantly from untreated group counterparts. For instance, treated municipalities display lower per capita GDP, larger share of the population that is indigenous, greater municipal area and higher unemployment rate. In some cases, the unconditional comparison in means reveals larger differences in both groups compared to results shown in Appendix Tables A2-A3. Indeed, the number of UBSs and physicians per capita are about 1.5 times lager in untreated areas, and the difference in population size between control and treatment groups is almost 75. However, there are no significant differences with respect to the share of rural population, distance to capital and rainfall. The rate of rural population is only 1 percentage point (about 2.8 percent of the mean) lower for untreated municipalities and the difference is not statistically significant from zero.

In the rest of columns, we present the same statistics for treatment and control groups but now the observations of the control group are weighted using the entropy balancing weights. These comparisons represent a balancing test of how well the treated group compares to the synthetic control group prior to policy implementation. Of the 16 outcomes in this Table, none is statistically different at the conventional levels of significant. For example, physicians, UBSs and population size, characteristics with large differences before matching, are now virtually perfectly balanced and no statistically significant difference remains.

While the use of entropy balancing weights increases the comparability between areas, identification requires that, in the absence of policy, all relevant factors would have evolved in a similar way over time between treatment and synthetic control groups. The statistically insignificant difference in physicians and UBSs in each year

prior to policy shown in Table 2 already indicate that this assumption might be valid. To assess in more detail the plausibility of the identifying assumption, we test if both groups were on parallel trends prior to policy implementation for each outcome of interest. The model for testing this take the following form:

$$y_{imt} = \sum_{k=-66}^{-1} \alpha^k \left(\mathbf{1}[\mu_{mt} = k] * \mathbf{1}[PMM_i = 1]\right) + \eta_i + \mu_{mt} + \xi_{it} \qquad (2)$$

where α^k represents the difference in the outcome of interest between treatment and synthetic control groups in date k, relative to the difference in this variable between both groups in a base date. Note that in the estimation of Equation (2) we use only observations for the pretreatment policy period, so the base date in is January 2008 or *k* = -68. This specification also includes month-by-year fixed effects to allow for systematic general trends, and municipality fixed effects are also included to control for time-invariant unobserved factors. We estimate equation (2) for a number of outcomes. Estimates of α^k directly test whether trends in each outcome between both groups were similar prior to policy. If the identifying assumption is valid, significant estimates of α^k at the 5 percent level should be found roughly 5 percent of the time.

Figure 4 plots coefficients and 95 percent confidence intervals from equation (2) for avoidable hospitalizations and physicians in UBSs. For hospitalization outcomes, the results are also presented for each age category. Only 5 of the 335 coefficients in this Figure are statistically significant from zero. This is in fact a substantial lower rejection rate of the null hypothesis of no effect compared to that one would expect. Furthermore, note that we find extremely small estimates on these regressions, so it is unlikely that this finding is driven by large standard errors. For example, the coefficient for *k*=-67 for avoidable hospitalizations is 0.0005. Relative to the mean, this implies an estimated effect of only 0.04 percent. In Appendix Figure 1, we repeat the same exercise considering only municipalities in the lowest quintile of per capita GDP distribution. There is no systematic difference in trends prior to policy in treatment and synthetic control groups in this limited sample.

Appendix Figures A2 through A5 show analogous difference-in-difference plots for additional time-varying characteristics, including UBSs, physicians outside of UBSs, number of hospitals, number beds, and number of medical equipment. Medical

equipment is grouped into six categories: infrastructure (e.g., air conditioning), diagnostic imaging, dental equipment, life support, optical and graph diagnostics, and others.[41] In almost no case, there are statistically significant differences. We also estimate the difference-in-difference given by equation (2) for these outcomes limiting the sample to municipalities in the lowest quintile of per capita GDP distribution. Again, there is no evidence that treatment and synthetic control groups were on differential trends during the pretreatment period.

Taken together, the lack of statistically significant differential trends in the period pre-intervention yields support for the identifying assumption that the treatment and synthetic comparison municipalities would have had similar trends in our outcomes of interest in the absence of PMM introduction. This bolsters our confidence on the validity of our statistical approach to separate the effect of PMM from other influences on child health. We provide below further evidence to support the identifying assumption that there were no major differential trends in unobserved across municipalities correlated with PMM implementation.

4.5. Results

4.5.1. Effect of PMM on physicians

We begin by quantifying the effect of PMM on the number of physicians. This exercise is useful for interpreting the magnitudes of PMM impacts on avoidable hospitalizations. Exploring physicians also allows us test omitted variable bias by exploring whether PMM is correlated with changes in the number of physicians in the private sector or physicians placed outside of UBSs. The results are reported in Table 3. Panel A presents the results from a basic specification that control for month-by-year and month-by-municipality fixed effects. The effect of PMM on physicians in UBSs – the measure that PMM should affect – is positive and significant. The estimated coefficient implies that PMM implementation resulted in an increase of 0.08 in physicians in UBSs per 1,000 residents. The rate of physicians in UBSs in the treatment group increased by 0.09 over this period, so PMM is responsible for 90 percent of this increase. There seem to have been other factors causing increases in physicians placed

[41] See Appendix for a more detailed description of these variables.

in UBSs, but the bulk of the increases observed during the study period are those associated to the PMM. In Panel B, we add State-specific linear time trends, which may capture changes in unobserved correlated to PMM implementation varying at the State-level. Adding controls for State linear trends reduces slightly the sampling variance and leaves the coefficient estimates virtually identical.

The rest of columns present the results for other measures of physicians per 1,000 inhabitants. When considering the total number of physicians working in the sector public (in-or-outside of UBSs), it is found a coefficient of PMM *x* Post extremely similar to that of our main measure of physicians in UBSs. This suggests that the increases in physicians in public sector are completely driven by physicians that were placed in UBSs. Confirming this finding, we find that the PMM effect on public physicians working outside of UBSs can be bounded to a tight interval around zero. For instance, we can rule out an effect of PMM on physicians working outside of UBSs one fourth as large as the PMM impact on physicians placed in UBSs. We continue to find estimates that can be bound away from the PMM effects on physicians placed in UBSs when our dependent variable is the number of physicians in the private sector per 1,000 inhabitants. Both physicians in the public sector placed outside of UBSs and physicians in the private sector were not target of policy, so large and significant estimates would have suggested the presence of unobserved that were correlated with PMM implementation.

To uncover more detail about the relationship between physicians and PMM adoption, we also present event-study graphs that plot the effects of PMM on physicians per 1,000 inhabitants. These graphs are derived from a version of the equation (1) that allows the coefficients on $Post_{mt}xPMM_i$ to vary with event time, where date zero is the date that PMM took place. Given that the difference-in-difference estimates were generally not significantly different from zero for the pretreatment period, we present the event graph study using a parsimonious specification in where the entire pre-intervention period is the comparison time group. Figure 5 plots these coefficients and their 95 percent confidence intervals. The plotted coefficients represent the time path of physician rates in treated and synthetic control groups before and after of PMM adoption, conditional on municipality and year-by-month fixed effects. The dashed lines

present the coefficient estimates and the solid lines show 95 percent confidence intervals for these coefficients.

The picture in Figure 5 is very clear. It shows that, after policy implementation, the number of physicians in UBSs increased much more rapidly in treated municipalities than in the comparison group. Moreover, the estimated impacts show that the increases in physicians in the treatment municipalities occurred immediately after policy implementation and stays upward thereafter, relative to the pretreatment period. Overall, we find large, immediate and persistent effects of PMM on physicians placed in UBSs. We take this set of results as evidence of a strong "first stage" that PMM improved the supply of medical care by increasing the number of physicians willing to provide primary health care services.

4.5.2. Effect of PMM on child health

Having documented that the PMM positively affected the number of physicians in UBSs, we now turn to the analysis of avoidable child hospitalizations. Table 4 presents the results. Column (1) shows the difference-in-difference estimates of the PMM effect on avoidable hospitalizations among children under five years old. In Panel A, we present the basic weighted regression with no controls besides municipality-by-month-by-gender and year-by-month fixed effects. The R^2's of the regressions are shown at the bottom.

The results show no evidence of an effect of the program on avoidable hospitalizations. The coefficient on Post *x* PMM capture the effect of living post-intervention versus pre-intervention in a municipality that implemented PMM, relative to the effect of living in a municipality not exposed to the program. If PMM implementation lead to reductions in avoidable hospitalizations, we would expect these coefficients to be negative. In column (1), we see no significant differences in avoidable hospitalization changes for those living in treated areas relative to those living in non-exposed sites. The estimated coefficient on the interaction term is 0.02 with a standard error of 0.03, which makes it highly insignificant. Moreover, note that the point estimate has in fact the wrong (positive) sign. In the next columns, we examine the effects separately by age categories as defined above. These results also reveal little evidence that the PMM reduced the incidence of avoidable hospitalizations among children.

Again, all point estimates have the wrong positive sign and the hypothesis null of zero effect is never rejected at the conventional levels of significance. In Panel B, we report results from a specification that includes state linear time trends. Adding such trends has little effect on the estimates and does not alter the statistical significance.

One might be worried that the lack of relationship is because people did not notice the increase in the number of physicians and hence did not adjust their demand for primary and preventive health care. To allow for a delay, we redefine the *Post* dummy indicator by assuming that the PMM began six months after it really took effect. We estimate the specification that includes State-linear trends as in column (1), but interacting this new dummy indicator policy with the PMM treatment variable. The results are shown in Panel C of Table 4. We continue to find no evidence of reductions in avoidable hospitalizations among children.

In Table 5, we take a closer look at this exercise by fitting a version of equation (1) as follows:

$$y_{imt} = \sum_{k=1}^{K} \alpha^k \, PMM_{imt}^k + \eta_i + \mu_{mt} + \xi_{\ it} \qquad (3)$$

where PMM_{imt}^k is a dummy variable assuming value 1 if municipality *i*, in month *m* and year *t* has been in the program for *K* time. We divided the post-treatment period into three groups: from 4 to 15, 16 to 27 and 28 to 40 months since policy implementation. The comparison time group is the entire pretreatment period. By doing so, we find again no evidence for improves in child health measured per avoidable hospitalizations associated to the program. In fact, the difference-in-difference coefficient is positive and marginally significant for 16-27 months since implementation.

An alternative hypothesis is that this no evidence of significant hospitalization reductions is explained by spatial spillovers. Indeed, it could be argued that families in untreated areas may have travelled to neighbor treated areas to benefit from the increased supply of primary health care. To test whether spillovers of this sort may be driving our results, in Table 6 we run the difference-in-difference specification that includes controls for State-linear trends, but including as additional control the rate of neighbor municipalities implementing the PMM interacted with the post-policy

indicator. Columns (1) replicates the baseline specification for ease of comparison, while the next columns present results using different definitions of neighbor municipalities. For instance, column (2) considers that neighbor municipalities are those within a radius of 40 km^2, while that column (4) uses a radius as wide as 100 km^2. While the difference-in-difference coefficients for neighbor municipalities is negative, they are very small in magnitude and far from significant. Furthermore, the main difference-in-difference coefficient of interest is extremely similar to the baseline one. We take these results as evidence that our results are in fact not driven by spatial spillovers between untreated and treated areas.

On average, we find no significant gains in terms of avoidable hospitalizations associated to the PMM, despite the large increase in the number of physicians willing to serve in UBSs. We now investigate whether these average effects mask important form of heterogeneities. A hypothesis is that PMM would have larger effects in poorer areas where the access to primary health services was more limited prior to policy. This might especially true whether health care interacts with family resources or whether the health returns to medical care depending on income. To explore this possibility, we directly test whether policy affects disproportionally to poorer areas. We limit the sample to municipalities in the lowest quintile of GDP per capita distribution and estimate the difference-in-difference estimator.

The results of this exercise are presented in Table 7. Panel A presents the results of a basic specification that includes controls for month-by-municipality-by-gender and month-by-year fixed effects. The next columns reports analogues results for different age categories. Unlike the results above, we find significant negative difference-in-difference coefficients in this sample, suggesting that the PMM lead to lower avoidable hospitalizations among children living in poor municipalities. Specifically, we find a 7 percent reduction in avoidable hospitalizations among children under five living in such municipalities. The effect is the largest among children who are 0-1 years old. In Panel B, we add State-linear time trends and find very similar results. Now the point estimate for children 0-5 years old is somewhat smaller, estimated at -0.048 with a standard error equal to 0.024. To explore in more detail this heterogeneity, we run the difference-in-difference specification by stratifying the sample by different levels of per capita GDP in Figure 6. There is a consistent pattern indicating that the effect of policy on avoidable

hospitalizations is the largest in magnitude in the poorer areas, ranging from -0.07 to 0.01. The overall reductions in this outcome over study period was 20 percent in treated areas with low per capita GDP. Hence, the PMM account for 25 percent of this reduction.

These results reveal substantial heterogeneity in the effect of PMM on avoidable hospitalizations. Remember that in Section 5.4.2 we present evidence that treated and reweighed untreated municipalities with low GDP were on parallel trends prior to policy. This suggests that the reductions in avoidable hospitalizations we observe in poor municipalities are unlikely to be artifact of the data or the product of mere convergence effects between poorer and richer areas.

Motivated by the difference-in-difference results in Table 8, we present results based on equation (3) focusing on the subsample of poor municipalities. Panel A presents the results the basic specification that includes municipality-by-month-by-gender and month-by-year fixed effects. Column (1) reports the results for children 0-5 years old, while the next columns presents the results for age subcategories. The results suggests that after policy implementation avoidable hospitalizations diverged in the treatment and reweighted control poor areas, with treated municipalities experiencing larger reductions. This differential emerges within the first 15 months after policy adoption and persists thereafter. Adding State-linear time trends slightly in magnitude the estimated effects, but they remain qualitatively similar. Although in some cases the inclusion of State linear trends leads to insignificant estimates, the point estimates remain similar to the results from the basic specification and the test of coefficients do not reject the hypothesis of equality. These patterns are similar for each age category.

We now run our baseline specification for each disease groups in Table 9. Panel A estimates the effects for all municipalities, while that Panel B presents the results for poor areas. Again, we focus on the specification that includes State-linear time trends. On average, we find no evidence for reductions in avoidable hospitalizations by disease condition associated to the policy. However, we find negative and statistically significant effects for poor areas, indicating that policy is associated with reductions in hospitalizations in these areas. These results suggest that the PMM effects are largely driven by hospitalizations from gastroenteritis and asthma complications. In particular, PMM adoption is associated to a 7 percent reduction in hospitalizations due to

complications from infectious gastroenteritis and to a 5 percent decline in hospitalizations from asthma. Because hospitalizations from infectious gastroenteritis complications declined by 23 percent over time in poor treated areas, the program can explain a 21 percent of this reduction. This calculation is more striking for hospitalizations from asthma. It suggests that PMM adoption can account for 45 percent of the reductions in hospitalizations from this disease condition.

4.5.3. Additional robustness checks

In this section, we further test the robustness of our results in a number of ways. While results above already suggests that our findings are not driven by differential trends in unobservable factors, we performance a placebo test to assuage these concerns. Because physicians from the program were placed in UBSs, in which people receive only primary health care services, we presume that hospitalizations from external causes should not be affected by adoption of program. Thus, we examine PMM effects on external hospitalizations to check the internal consistency of our findings. The results are presented Appendix Table A5. These results are presented for all and the subset of poor municipalities to allow for possible heterogeneous effects between poor and non-poor areas. There is no evidence that policy affected this outcome. In addition, the estimated effects are small and tightly bound around zero. This result provides additional confidence that the observed reductions in avoidable hospitalizations in poor areas is due to policy rather than a random fluctuation.

Our second check examines the robustness of our results to different specifications. Column (1) replicates our baseline specification for poor areas. Columns (2) of Appendix Table A6 shows that the inclusion of State quadratic time trends slightly improves the precision of estimates with very little effect on the estimated coefficient of interest. In column (3), we use a much more demanding specification that incorporate State-by-month-year fixed effects, resulting in over 1,500 additional independent variables (14 States, 12 months, 9 years).[42] Including this robust set of fixed effects reduced leads to larger effects in magnitude and a decreased standard error. If there exists some unobserved factor varying at more local level that our matching difference-in-difference approach does not account for, our controls for State time

[42] The number of states in the subsample of poor municipalities is smaller, so municipalities from states are excluded of the analysis.

trends or State-by-month-by-year fixed effects will not capture such unobserved influences. To address this issue, columns (4) and (5) include mesoregion-specific linear and quadratic time trends instead of State-specific time trends, respectively. Adding these specific trends either linear or quadratic leads to difference-in-difference coefficients almost identical to that obtained in the specification that includes State-linear trends. Finally, columns (6)-(7) allow for differential trends at a more local level, namely microregion trends.[43] The inclusion of these trends lead to more negative and precisely estimated coefficients of the effect of policy on child health in poor areas, so the findings remained essentially the same. In sum, our results are robust to using these more demanding specifications and hence we are more confident about our empirical approach.

We also check for possible mean reversion in avoidable hospitalizations by including lags for dependent variable as explanatory variables. We consider 1, 6 and 12-month lags of dependent variable. Note that now the total PMM effects on the outcome of interest should be computed as $\beta/(1-\sum\theta_l)$, where θ_l is the coefficient on each lagged levels of dependent variables. Appendix Table A7 shows the results of this exercise. For ease of comparison, the total PMM effect is also presented for each outcome. Again, column (1) replicates our baseline estimates. While the lagged levels of the variables of interest are highly significant and the fit of regressions improves appreciably, the PMM (total) effects remain almost identical to our baselines. For example, the PMM effect on avoidable hospitalizations for poor areas goes from 4.8 percent in baseline to 4 percent in the specification that includes 12 lags for this outcome as independent variables.

In our baseline estimates, we clustered standard error at the State-level, which allows both for arbitrary correlation in residuals across municipalities within a mesoregion and for autocorrelation either at the mesoregion or municipality level. As a robustness check, Appendix Table A8 computes the standard errors for an arbitrary variance-covariance matrix at different geographic levels. Column (2) shows that clustering at the municipality-level decreases the standard errors. However, clustering at the municipality level ignores any potential correlation across municipalities. In column (3), we cluster at the microregion level and find standard errors slightly lower than those

[43] In general, a micro-region is formed by a group of municipalities sharing the same border. On average, a micro-region is made of seven municipalities.

in our baseline models. Finally, clustering at the State level also leads to almost identical standard errors compared to baselines. Irrespectively of how the standard errors are clustered, the statistically significance is very in line with our baseline findings.

Our main results parameterized hospitalization outcomes as $\ln(1+x)$ because there are many municipality-by-month-by-gender observations that are equal to zero. Appendix Table A9 presents estimates that use different parameterizations of avoidable hospitalizations. Column (2) uses a more flexible parameterization, namely the inverse hyperbolic sine parameterization, which is defined as $\ln(x+\sqrt{1+x^2})$. For small *x*, estimated coefficients can be interpreted as percentage impacts of policy. Parameterizing avoidable hospitalizations in this way yields to very similar results. In columns (3)-(4), we estimate Poisson and negative binomial count models. For computational feasibility, we use a limited number of explanatory variables in these models. Specifically, we control for State fixed effects, a linear trend, post-policy indicator, and the PMM dummy indicator. The results from these regressions are qualitatively similar to our baseline, but point estimates are considerably larger in magnitude, which is perhaps unsurprisingly given the crudeness of these specifications.

We also examine whether our results are driven by atypical areas-by-date-by-gender observations in Appendix Table A10. For this, we drop all municipality-by-month-by-year observations that are below the 5th or above the 95th percentile in the distribution of residuals in our baseline regressions. The point estimates change very little in these regressions. Making the same exercise for the 1th and the 99th percentiles yields to very similar results. As a further check, we run the regressions repeatedly, each time excluding all municipalities from one State, and find that the magnitude of the results does not change appreciably (see Appendix Figure A10). Overall, our results are not driven by outlier observations.

4.5.4. The effect of PMM on hospital costs

In this section, we provide additional inputs that may be used in a cost-effectiveness analysis. Specifically, one can estimate the potential benefits of the program in terms of reductions in hospital costs of caring children. For this, we computed the total hospital cost from avoidable hospitalizations at the municipality-by-

gender level for each month and year. We then estimate weighted difference-in-difference regressions using this outcome as a dependent variable. The results are presented in Table 10. Panel A we present the results for the full sample of municipalities. All of our weighted difference-in-difference regressions control for State-linear time trends and municipality-by-month-by-gender and month-by-year fixed effects. We find a small and insignificant effect of the effect of PMM on hospital costs in this regression. This is unsurprisingly given the null effects we find above on avoidable hospitalizations in the average treated municipality. Therefore, a significant and large impact on hospital costs would call into question our identification strategy.

Panel B present the results for the sub-sample of poor municipalities. We find negative and significant difference-in-difference coefficients on this sample. The PMM implementation leads to 160 Brazilian Reais (USD 50 dollars) decline in hospital cost from avoidable hospitalizations in poor areas. This effect is relatively large compared to a pre-intervention mean in treated areas of 1,700 Reais. Since the overall reduction in hospital cost during the entire period was 500 Reais, the data indicate that PMM accounted for 32 percent of this reduction. Overall, these results suggest that program contributed to reductions in avoidable hospital costs only in poor areas.

4.6. Interpretation

Our results represent reduced form of the effect of PMM on child health. Now, we seek to understand specific mechanisms underlying the relationship between PMM and child health. In section S, we provide strong evidence that the number of physicians serving in UBSs increased significantly after PMM implementation in treated areas. In addition, we find no evidence of similar break trends in physicians serving in private sector or outside of UBSs. In principle, it seems that the mechanism driving the changes in hospitalization outcomes is the increased number of physicians providing primary health care services. However, the program aims to expand the infrastructure network to the year 2027, through construction of new UBSs. Thus, a natural question is whether the number of UBSs increased differentially between treated and untreated areas after policy implementation during our period study. If so, this might also be a mechanism by which the program affected child health.

We directly test this hypothesis in column (1) of Appendix Table A11, where difference-in-difference estimates of the effect on the number of UBSs per 1,000 residents are shown for all and the sub-set of poor municipalities. We find highly insignificant estimates on these regressions. In addition, the estimates on *PMM x* Post are quantitatively small. For example, the coefficient implies that PMM adoption leads to a statistically insignificant increase of 0.002 (standard error =0.004) in the number of UBSs per 1,000 inhabitants, which is very small relative to the mean of 0.28. Visual inspection in the event-study shown in Appendix Figure A6 ratifies that there was not a statistically significant increase in UBSs rate. Overall, there is no evidence for a positive effect of PMM adoption on UBSs rate, at least in our study period. This is true also for municipalities in the lowest quintile of per capita GDP distribution.

The next columns of Appendix Table A11 investigates whether the implementation of PMM coincided with changes in other health resources as proxied by local hospital capacity. This could be the case if, for instance, PMM implementation encouraged municipality governments to increase local hospital size. Using the number of hospitals, the number of rest beds, and the number of medical equipment as dependent variables (all measured per 1,000 inhabitants), we find insignificant policy effects on these outcomes. In addition, the estimated coefficients are small in magnitude relative to the mean. The event-study graphs in Appendix Figures A6 through A9 also confirm that both outcomes evolved smoothly before and after PMM adoption. When the sample is limited to poor areas, the conclusion is the same. Taken together, the results indicate that program did not affect child health outcomes through changes in local infrastructure network.

The assumption that treated and synthetic control areas would have experienced the same proportional changes in hospitalization outcomes in the absence of the PMM is sufficient to produce unbiased estimates of the impact of program. Under an additional assumption, one can use the program to construct instrumental variable estimates of the impact of the number of physicians on children's hospitalizations. In this framework, the difference-in-difference in child hospitalizations is the reduced-form estimate and the difference-in-difference in physicians is the first stage. Thus, the interaction term *PMM**Post would be the instrument. In addition to the assumption aforementioned, the validity of this instrumental variable strategy rests crucially on the assumption that the

program had no effect on child hospitalizations other than by increasing the availability of doctors serving in UBSs. The absence of a significant effect on local infrastructure capacity suggests that such an assumption might be valid. However, we cannot rule completely out the possibility that another unobserved mechanism played a role. For example, the program may have affected physician quality, but data limitations do not allow us to examine this hypothesis. To the extent that physician quality and other potential unobserved mechanisms are important, a violation of the identifying assumption of the instrumental variable strategy may occur. Hence, we stress that the instrumental variables presented below should be only taken as an exploratory effort to understand how physicians affect hospitalization outcomes.

With this caveat in mind, we present instrumental variable estimates of the effect of physicians. We then estimate the effects of the program on physicians and child hospitalizations in the subsample of poor areas. Columns (1) of Appendix Table A12 shows that PMM adoption led to a highly significant increase of 0.071 (with standard error=0.009) in the number of physicians in UBSs per 1,000 residents, an estimated coefficient very similar to that observed in the regressions that use the full sample of municipalities. The reduced-form estimate on child hospitalization, shown in column (2), indicates that PMM adoption is associated with a reduction of 4.8 percent in this outcome. Thus, the instrumental variable estimate implies that a one 10 percent increase in the number of physicians in UBSs is associate to a decrease of 1.6 percent in the incidence of children's hospitalizations. Column (3) performances the same exercise for hospital cost. We find that a one 10 percent increase in physicians is associated with a statistically insignificant reduction of 55 Reais in hospital costs from hospitalizations that are the result of avoidable conditions, with an estimated elasticity at -0.32.

4.7.Conclusion

In this paper, we present novel evidence on the effect of a large scale program designed to increase the supply of physicians on child health in Brazil. Using a matching difference-in-difference estimator, we estimate that the policy reduced significantly avoidable hospitalizations among children but only in poor areas. In such areas, implementation of policy is associated with a 4.8 reduction in avoidable hospitalizations. The effects were the largest for children 0-1 years old and for gastroenteritis and asthma conditions. In particular, policy adoption is associated with a

7 percent reduction in hospitalizations due to gastroenteritis complications, within 15 months after implementation. We also find evidence that policy led to statistically significant reductions in hospital cost due to avoidable conditions, but again only in poor areas. A variety of evidence supports a causal interpretation of these results. For instance, we find no similar effects on hospitalizations from external causes, and in addition, it is documented that treatment and synthetic control groups were parallel trends prior to policy. Together, these findings suggest that increased supply of primary care physicians results in more efficient use of health care resources, fewer avoidable hospitalizations and better child health among disadvantaged families.

We can place our estimates in perspective by comparing them to differences in child health between poor and non-poor areas before and after policy implementation. Prior to PMM, poor areas had avoidable hospitalizations 12 percent higher than non-poor areas, but after PMM this difference was reduced to 5 percent. Our difference-in-difference estimates indicating that avoidable hospitalizations declined by 4.8 percent imply that PMM implementation was responsible for almost 70 percent of the reduction in the gap between poor and non-poor areas.

Another way to assess the size of the estimated effects of PMM found in this paper is to compare them with previous estimates of the effect of other policies. Dafny and Gruber (2005) find that Medicaid expansion is associated to small and statistically insignificant increase in avoidable hospitalizations among eligible children. In contrast, using an instrumental variable strategy, Aizer (2007) shows that increasing the number of children from poor families with health insurance by 10 percent is associated to a reduction of 2 to 3 percent in avoidable hospitalizations among children from poor families. This represents an estimated elasticity of avoidable hospitalizations with respect to health insurance of -0.20, which is slightly larger to the elasticity with respect to physicians we estimate here. Thus, our findings are comparable with that of Aizer (2007).

Figure 1. Physicians in UBSs per 1,000 residents

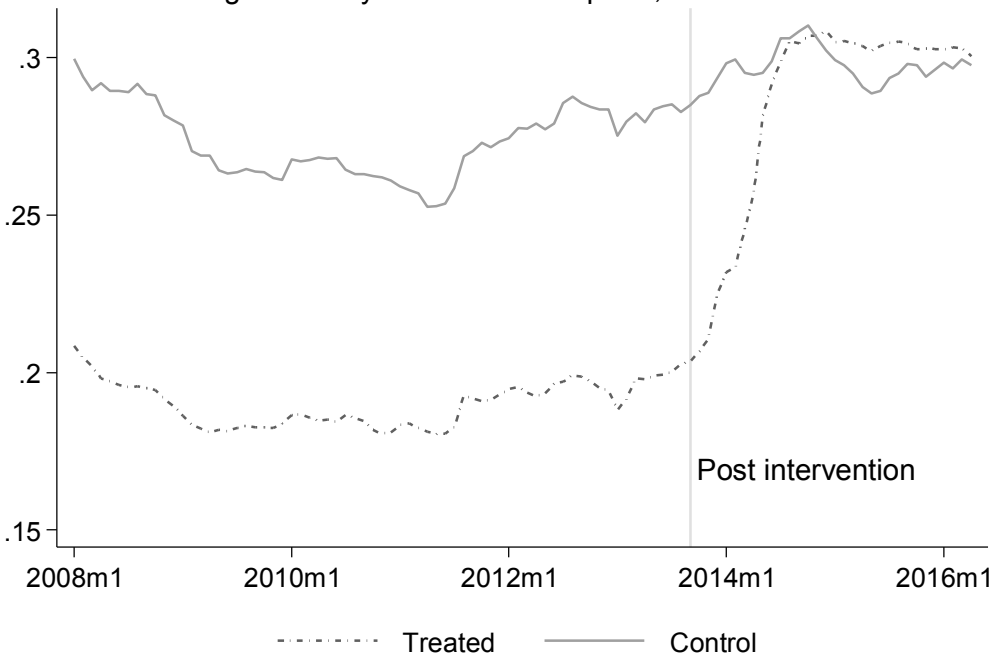


Figure 2. Physicians and income

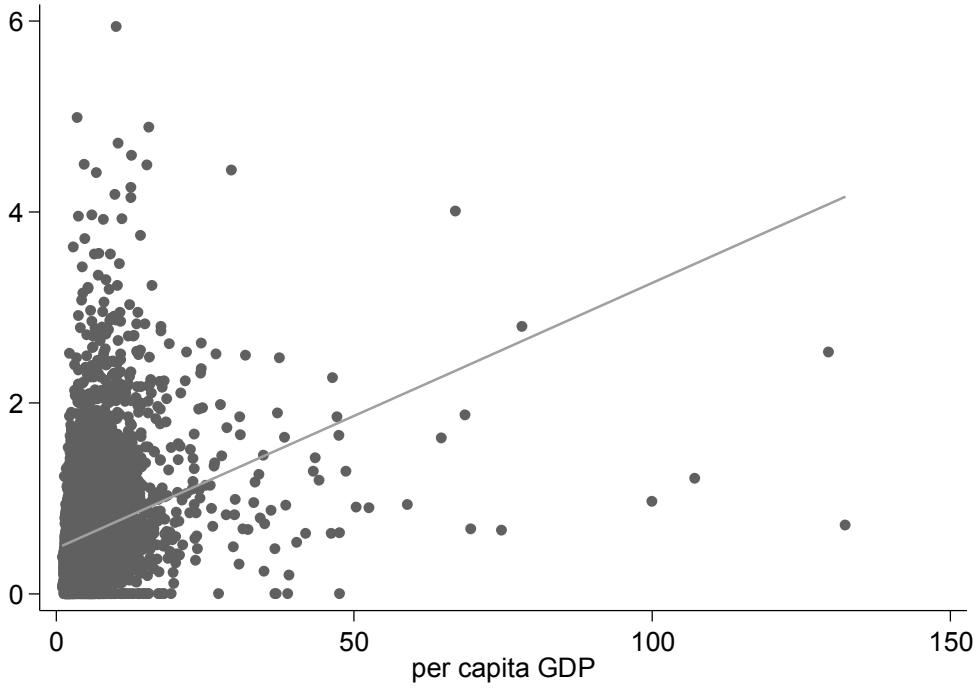


Figure 3. Physicians and population

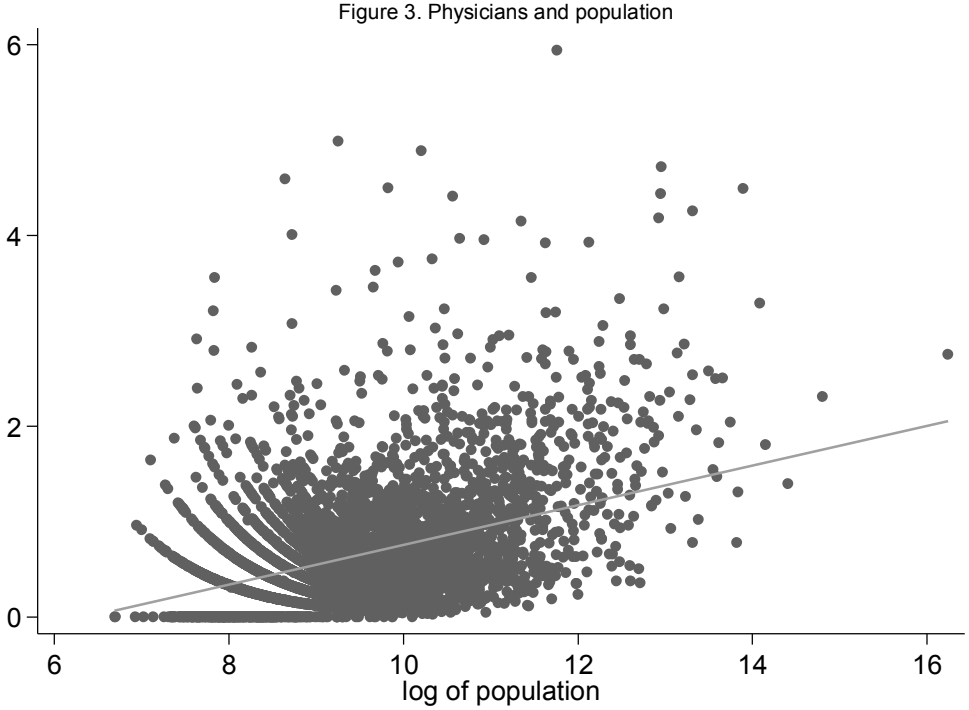
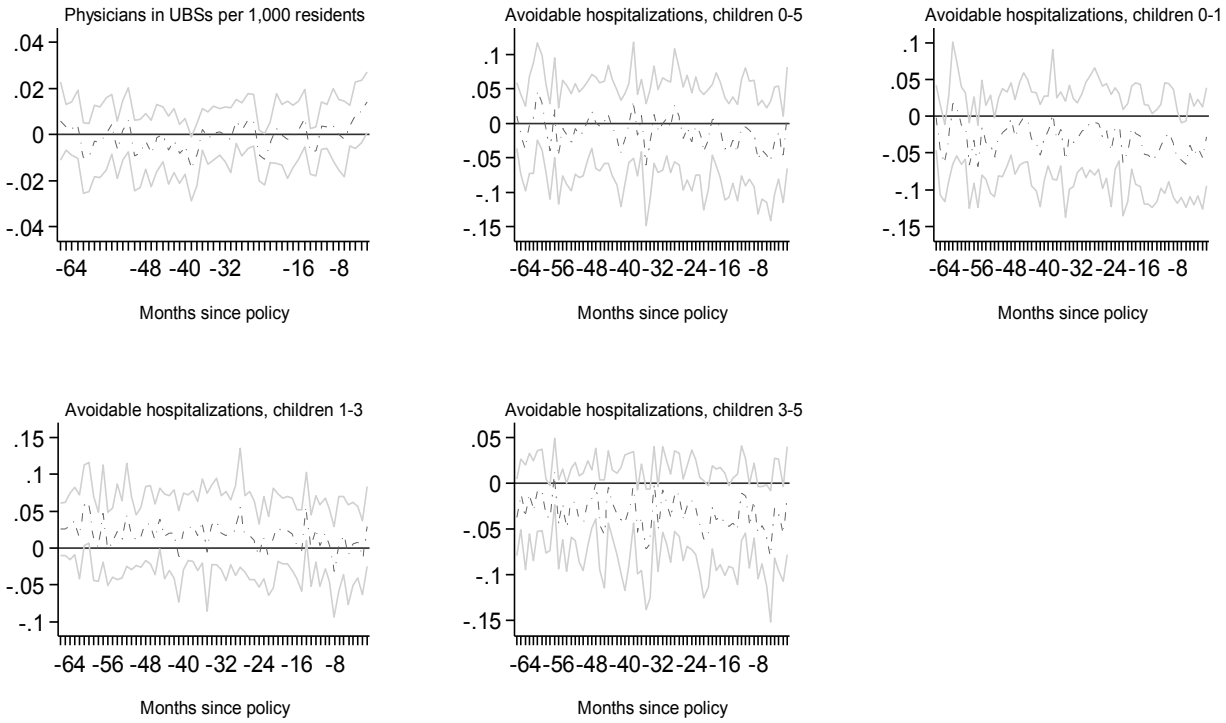
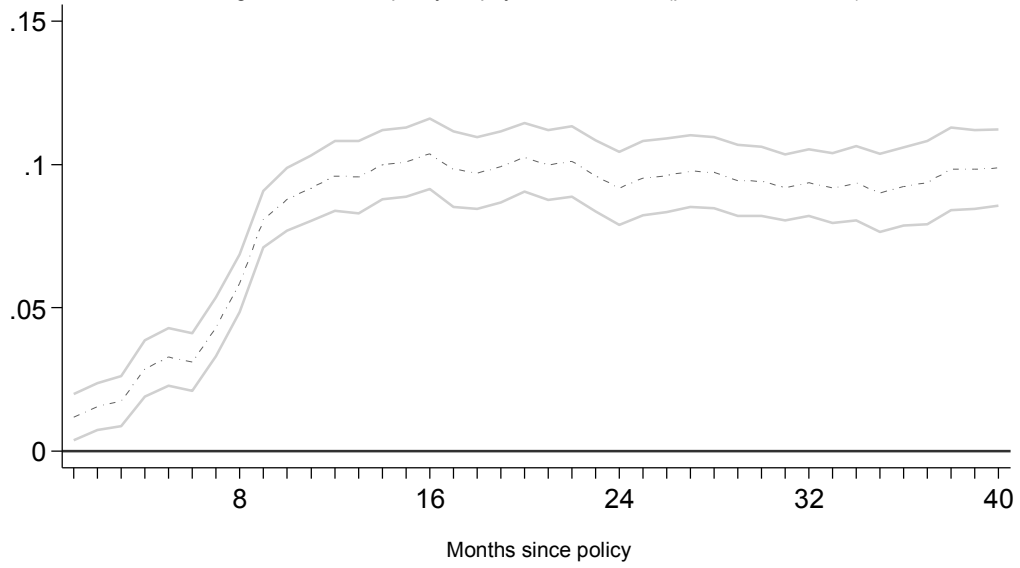


Figure 4. Testing pretreatment differential trends



Notes. This displays the difference-in-difference coefficients of each month prior to policy. The regressions include municipality-by-month and month-by-year fixed effects and a constant term. The omitted category is January 2008. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients.

Figure 5. Effect of policy on physicians in UBSs (per 1,000 residents)



Notes. This displays the difference-in-difference coefficients of each post-policy month. The regressions include municipality-by-month and month-by-year fixed effects, State-linear trends and a constant term. The omitted category is the entire pre-treatment peirod. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients.

Figure 6. Effect of policy on avoidable hospitalizations by income

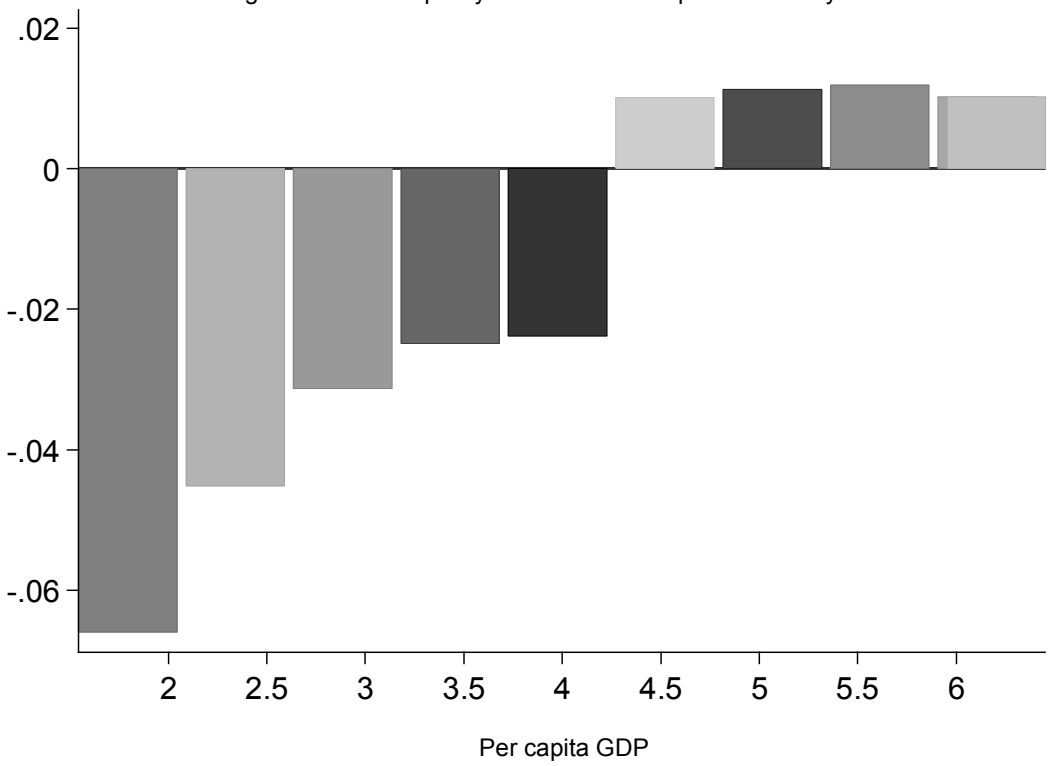


Table 1. Descriptive Statistics

	N	Municipalities	Mean	Standard Deviation	Min	Max
Treated municipalities	587,412	5,439	0.725	0.44	0	1
Physicians in UBSs (per 1,000 residents)	587,412	5,439	0.25	0.29	0	5.12
Hospital cost (2010 Reais)	384,922	5,439	4124.03	18757.77	20.86	1457547
Avoidable hospitalizations						
All children 0-5 years old	587,412	5,439	5.43	24.42	0	1749
Avoidable hospitalizations by age category:						
0-1 years old	587,412	5,439	1.94	9.10	0	715
1-3 years old	587,412	5,439	2.10	9.39	0	696
3-5 years old	587,412	5,439	1.39	6.34	0	439
Avoidable hospitalizations by condition:						
Immunization preventable	587,412	5,439	0.71	3.55	0	227
Gastroenteritis	587,412	5,439	1.97	7.37	0	626
Bacterial pneumonia	587,412	5,439	0.60	4.05	0	435
Asthma	587,412	5,439	0.71	4.52	0	384
Pulmonary diseases	587,412	5,439	0.51	5.24	0	701
Other diseases	587,412	5,439	0.93	5.76	0	488
Hospitalizations from injuries	587,412	5,439	1.50	3.06	1	268

Sources: SIHSUS microdata and Ministry of Health.

Table 2. Covariate Balancing

	Before Matching			After matching		
	Untreated	Treated	p-Value, mean difference	Untreated	Treated	p-Value, mean difference
	(1)	(2)	(3)	(4)	(5)	(6)
Per capita GDP	6.21	5.35	0.00	5.35	5.35	0.99
	[7.36]	[5.49]		[4.16]	[5.49]	
Indigenous population	0.37	0.85	0.00	0.60	0.85	0.19
	[3.20]	[4.70]		[3.04]	[4.70]	
Gini Index	0.48	0.51	0.00	0.51	0.51	0.34
	[0.06]	[0.06]		[0.06]	[0.06]	
Unemployment rate	5.95	6.49	0.00	6.53	6.49	0.85
	[3.56]	[3.69]		[3.43]	[3.69]	
Illiterate rate	15.22	16.29	0.07	16.29	16.29	0.99
	[9.44]	[9.82]		[9.97]	[9.82]	
% rural population	35.54	36.65	0.37	36.65	36.65	0.99
	[20.91]	[21.92]		[22.44]	[21.92]	
Log of population	8.85	9.60	0.00	9.60	9.60	0.99
	[0.91]	[1.10]		[1.00]	[1.10]	
Municipality area	662.83	1899.45	0.00	1899.38	1899.45	0.99
	[1227.61]	[6674.62]		[3060.45]	[6674.62]	
Altitude	4.65	3.92	0.00	4.27	3.92	0.18
	[2.72]	[2.96]		[2.85]	[2.96]	
Distance to capital	259.49	254.31	0.67	258.11	254.31	0.76
	[149.82]	[167.55]		[168.20]	[167.55]	
Temperature	22.26	22.63	0.08	22.93	22.63	0.14
	[2.90]	[3.01]		[2.80]	[3.01]	
Rainfall	114.70	116.27	0.52	112.95	116.27	0.14
	[30.78]	[38.24]		[35.92]	[38.24]	
Amazon	0.09	0.15	0.01	0.15	0.15	0.99
	[0.28]	[0.36]		[0.36]	[0.36]	
Semiarid	0.17	0.22	0.04	0.22	0.22	0.99
	[0.37]	[0.41]		[0.41]	[0.41]	

Table 2. Covariate Balancing (Continued)

	Before Matching			After matching		
	Untreated	Treated	p-Value, mean difference	Untreated	Treated	p-Value, mean difference
	(1)	(2)	(3)	(4)	(5)	(6)
UBSs (2008)	0.34	0.26	0.00	0.26	0.26	0.99
	[0.21]	[0.17]		[0.15]	[0.17]	
UBSs (2009)	0.34	0.25	0.00	0.25	0.25	0.99
	[0.21]	[0.17]		[0.15]	[0.17]	
UBSs (2010)	0.34	0.26	0.00	0.26	0.26	0.99
	[0.21]	[0.17]		[0.15]	[0.17]	
UBSs (2011)	0.35	0.27	0.00	0.27	0.27	0.99
	[0.20]	[0.17]		[0.15]	[0.17]	
UBSs (2012)	0.36	0.28	0.00	0.28	0.28	0.99
	[0.20]	[0.17]		[0.15]	[0.17]	
Physician (2008)	0.28	0.18	0.00	0.18	0.18	0.99
	[0.36]	[0.22]		[0.21]	[0.22]	
Physician (2009)	0.26	0.18	0.00	0.18	0.18	0.99
	[0.32]	[0.21]		[0.20]	[0.21]	
Physician (2010)	0.26	0.18	0.00	0.18	0.18	0.99
	[0.32]	[0.21]		[0.20]	[0.21]	
Physician (2011)	0.27	0.19	0.00	0.19	0.19	0.99
	[0.34]	[0.23]		[0.20]	[0.23]	
Physician (2012)	0.28	0.19	0.00	0.19	0.19	0.99
	[0.35]	[0.22]		[0.21]	[0.22]	

Notes. Columns (1)-(2) show the means and standard deviations of conditions for untreated and treated municipalities before matching algorithm. Column (4) show the means and standard deviations of conditions in treated municipalities and column (5) the means and standard deviations of conditions for the synthetic control group (control), which is created by entropy balancing. Columns (3) and (6) present the p-value of on the test of zero difference in means between the subsamples of treated and untreated municipalities. These tests adjust for clustering of errors. UBSs and physicians are measured per 1,000 residents. Physician refers to the number of doctors placed in UBSs. Sample size is 5,439 municipalities.

Table 3. The Effect of PMM on Physicians

	Physicians			
	Public Sector (1)	UBSs (2)	Outside of UBSs (3)	Private Sector (4)
		Panel A: Basic specification		
PMM x Post	0.087	0.083	0.002	-0.001
	[0.011]***	[0.006]***	[0.011]	[0.003]
R^2	0.859	0.672	0.879	0.857
		Panel B: Add State-linear time trends		
PMM x Post	0.089	0.082	0.006	-0.001
	[0.008]***	[0.006]***	[0.009]	[0.003]
R^2	0.86	0.674	0.88	0.859

Notes. Dependent variables are measured per 1,000 residents. All regressions use entropy balancing weights. Panel A controls for municipality-by-month and month-by-year fixed effects. In addition, Panel B includes State-linear time trends. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. Each cell reports the coefficient from a separate regression. N =587,412 (5,439 municipalities, 12 months, 9 years). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table 4. The Effect of PMM on Avoidable Hospitalizations

	Children			
	0-5 years old (1)	0-1 years old (2)	1-3 years old (3)	3-5 years old (4)
		Panel A: Basic specification		
PMM x Post	0.028	0.029	0.013	0.018
	[0.033]	[0.030]	[0.023]	[0.019]
R^2	0.734	0.675	0.629	0.58
		Panel B: Add State-linear time trends		
PMM x Post	0.036	0.031	0.019	0.023
	[0.025]	[0.021]	[0.017]	[0.014]
R^2	0.738	0.680	0.633	0.585
		Panel C: Add State-linear time trends and policy took place six months later		
PMM x Post	0.049	0.039	0.034	0.034
	[0.029]	[0.024]	[0.021]	[0.020]
R^2	0.738	0.680	0.633	0.585

Notes. Dependent variable in each cell is parameterized as $ln(1+z)$. All regressions use entropy balancing weights. Panel A controls for municipality-by-month-by-sex and month-by-year fixed effects. In addition, Panels B and C include State-linear time trends. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. Each cell reports the coefficient from a separate regression. N =1,174,824 (5,439 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table 5. The Effect of PMM on Avoidable Hospitalizations (Dose effects)

	Children			
	0-5 years old (1)	0-1 years old (2)	1-3 years old (3)	3-5 years old (4)
PMM x **1**(4-15)	0.015	0.016	0.007	0.009
	[0.024]	[0.022]	[0.016]	[0.011]
PMM x **1**(16-27)	0.047	0.040	0.021	0.031
	[0.025]*	[0.021]*	[0.017]	[0.017]*
PMM x **1**(28-40)	0.059	0.045	0.047	0.04
	[0.040]	[0.031]	[0.033]	[0.028]
R^2	0.738	0.680	0.633	0.585

Notes. Dependent variable in each cell is parameterized as $ln(1+z)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. **1**(.) is an event-time indicator equal to 1 for month groups since policy implementation. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. *N* =1,174,824 (5,439 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table 6. The Effect of PMM on Avoidable Hospitalizations (Spillovers)

		Neighbor municipalities within			
	Baseline	40 km^2	60 km^2	80 km^2	100 km^2
	(1)	(2)	(3)	(4)	(5)
PMM x Post	0.036	0.037	0.037	0.039	0.041
	[0.025]	[0.024]	[0.024]	[0.024]	[0.026]
Neighbor PMM rate x Post		-0.000	-0.000	-0.001	-0.001
		[0.001]	[0.001]	[0.001]	[0.001]
R^2	0.738	0.739	0.739	0.739	0.739

Notes. Dependent variable is $ln(1 + avoidable\ hospitazalitons)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. *Neighbor PMM rate* is the proportion of neighbor municipalities implementing the PMM expressed in %. Columns (2)-(5) use different definitions of neighbor municipalities. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. *N* =1,174,824 (5,439 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table 7. The Effect of PMM on Avoidable Hospitalizations (Poor areas)

	Children			
	0-5 years old (1)	0-1 years old (2)	1-3 years old (3)	3-5 years old (4)
	Panel A: Basic specification			
PMM x Post	-0.069	-0.051	-0.049	-0.036
	[0.027]**	[0.018]***	[0.018]**	[0.016]**
R^2	0.632	0.543	0.502	0.442
	Panel B: Add State-linear time trends			
PMM x Post	-0.048	-0.037	-0.034	-0.024
	[0.024]**	[0.016]**	[0.016]**	[0.013]*
R^2	0.638	0.547	0.507	0.446

Notes. Dependent variable is $ln(1 + avoidable\ hospitazalitons)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. Each cell reports the coefficient from a separate regression. *N* =290,304 (1,344 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table 8. The Effect of PMM on Avoidable Hospitalizations (Poor areas)

	Children			
	0-5 years old (1)	0-1 years old (2)	1-3 years old (3)	3-5 years old (4)
	Panel A: Basic specification			
PMM x **1**(4-15)	-0.071	-0.054	-0.043	-0.034
	[0.028]**	[0.017]***	[0.020]**	[0.019]*
PMM x **1**(16-27)	-0.057	-0.041	-0.044	-0.035
	[0.032]*	[0.021]*	[0.022]**	[0.018]*
PMM x **1**(28-40)	-0.087	-0.065	-0.059	-0.046
	[0.030]***	[0.021]***	[0.020]***	[0.016]***
R^2	0.632	0.543	0.502	0.442
	Panel B: Add State-linear time trends			
PMM x **1**(4-15)	-0.054	-0.043	-0.031	-0.024
	[0.024]**	[0.015]***	[0.017]*	[0.016]
PMM x **1**(16-27)	-0.036	-0.027	-0.029	-0.023
	[0.029]	[0.020]	[0.020]	[0.015]
PMM x **1**(28-40)	-0.062	-0.047	-0.041	-0.031
	[0.028]**	[0.020]**	[0.019]**	[0.014]**
R^2	0.638	0.547	0.507	0.447

Notes. Dependent variable in each column is parameterized as $ln(1+z)$. All regressions use entropy balancing weights. Panel A controls for municipality-by-month-by-sex and month-by-year fixed effects. In addition, Panels B includes State-linear time trends. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. **1**(.) is an event-time indicator equal to 1 for month groups since policy implementation. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. *N* =290,304 (1,344 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table 9. The Effect of PMM on Avoidable Hospitalizations (By disease condition)

	Condition					
	Immunization preventable	Infectious gastroenteritis	Bacterial pneumonia	Asthma	Pulmonary diseases	Others
	(1)	(2)	(3)	(4)	(5)	(6)
			Panel A: All municipalities			
PMM x **1**(4-15)	-0.001	-0.001	0.022	-0.006	0.005	0.021
	[0.010]	[0.019]	[0.033]	[0.014]	[0.008]	[0.008]***
PMM x **1**(16-27)	0.00	0.022	0.043	0.004	0.018	0.015
	[0.011]	[0.023]	[0.032]	[0.015]	[0.008]**	[0.012]
PMM x **1**(28-40)	0.018	0.05	0.042	0.009	0.007	0.013
	[0.013]	[0.042]	[0.027]	[0.018]	[0.011]	[0.018]
R^2	0.539	0.645	0.575	0.528	0.541	0.568
N	1,174,824	1,174,824	1,174,824	1,174,824	1,174,824	1,174,824
			Panel B: Poor municipalities			
PMM x **1**(4-15)	0.003	-0.068	0.003	-0.017	-0.003	0.007
	[0.010]	[0.025]***	[0.015]	[0.012]	[0.006]	[0.011]
PMM x **1**(16-27)	0.020	-0.061	0.014	-0.035	-0.001	0.006
	[0.013]	[0.026]**	[0.016]	[0.013]**	[0.007]	[0.013]
PMM x **1**(28-40)	0.009	-0.049	-0.003	-0.050	-0.004	-0.029
	[0.009]	[0.029]*	[0.016]	[0.017]***	[0.006]	[0.015]*
R^2	0.342	0.569	0.465	0.456	0.306	0.336
N	290,304	290,304	290,304	290,304	290,304	290,304

Notes. Dependent variable is $ln(1+z)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. **1**(.) is an event-time indicator equal to 1 for month groups since policy implementation. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

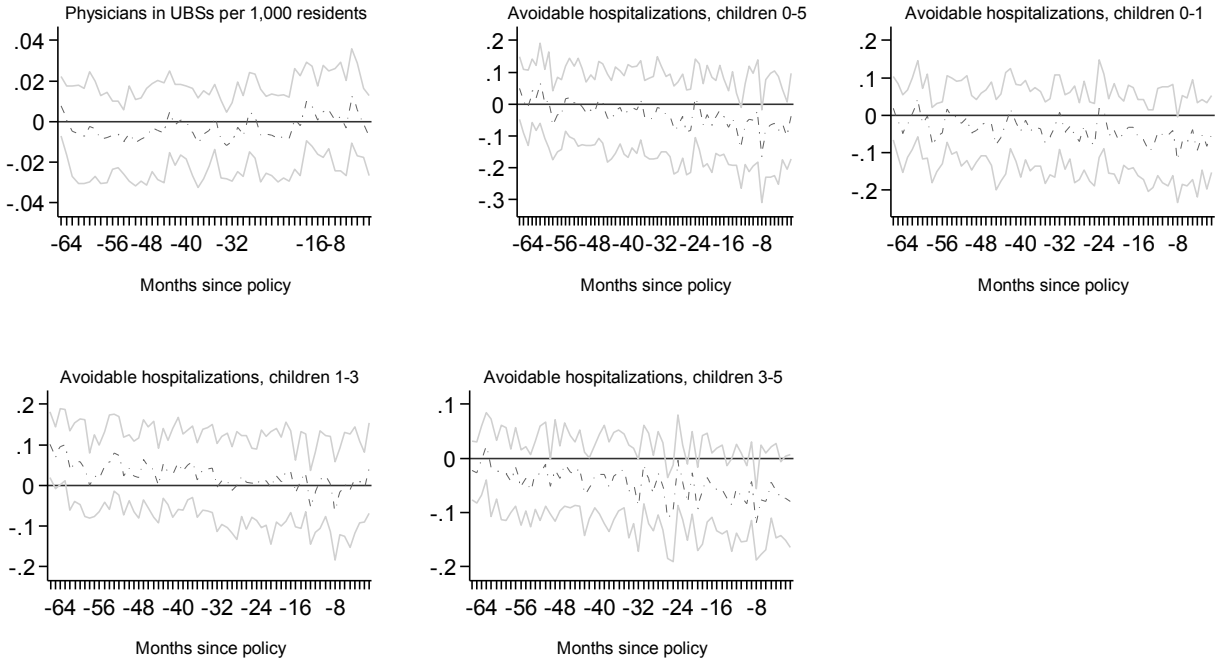
Table 10. The Effect of PMM on Hospital Costs from avoidable conditions

	Children			
	0-5 years old	0-1 years old	1-3 years old	3-5 years old
	(1)	(2)	(3)	(4)
		Panel A: All municipalities		
PMM *x* Post	3.455	15.056	-12.272	0.672
	[154.154]	[87.177]	[41.828]	[31.105]
R^2	0.868	0.791	0.744	0.705
N	1,174,824	1,174,824	1,174,824	1,174,824
		Panel B: Poor areas		
PMM *x* Post	-164.328	-53.651	-74.059	-36.618
	[56.456]***	[30.599]*	[20.648]***	[13.100]***
R^2	0.448	0.308	0.302	0.261
N	290,304	290,304	290,304	290,304

Notes. Dependent variable in each column is $ln(1+z)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

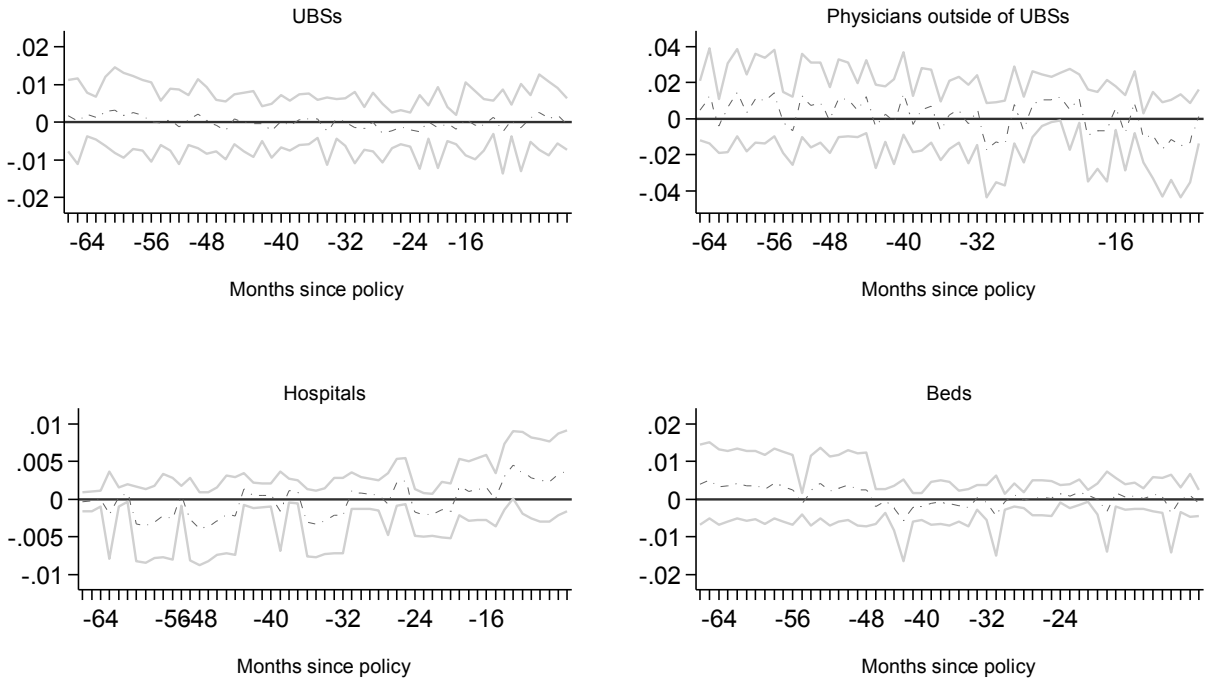
Appendix A: *Additional tables and figures*

Figure A1. Testing pretreatment differential trends in poor areas



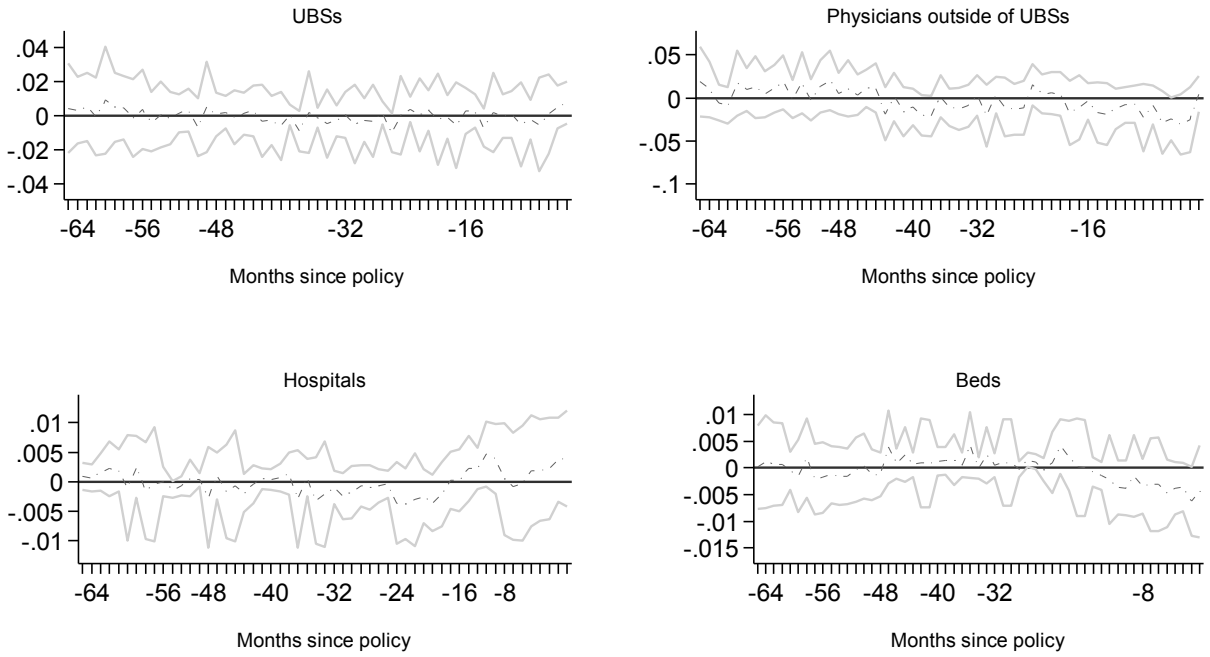
Notes. This displays the difference-in-difference coefficients of each month prior to policy. The regressions include municipality-by-month and month-by-year fixed effects and a constant term. The omitted category is January 2008. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution

Figure A2. Testing pretreatment differential trends - Other outcomes.



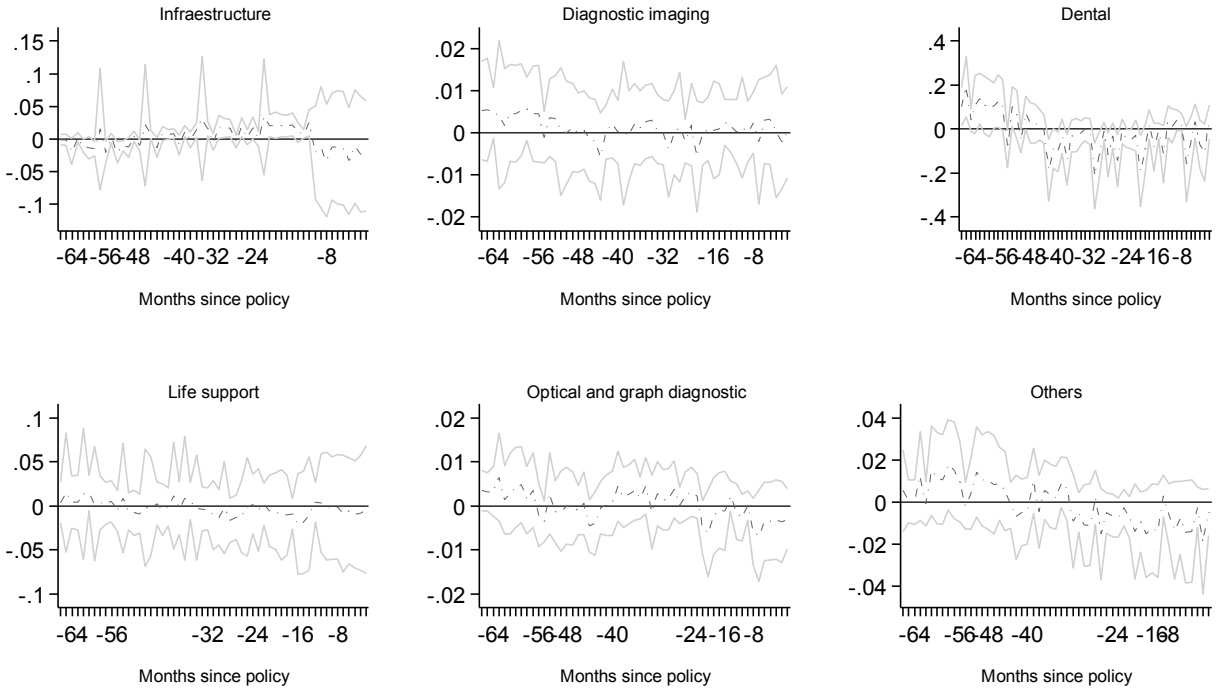
Notes. This displays the difference-in-difference coefficients of each month prior to policy. The regressions include municipality-by-month and month-by-year fixed effects and a constant term. The omitted category is January 2008. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. All dependent variables are measured per 1,000 residents

Figure A3. Testing pretreatment differential trends in poor areas- Other outcomes.



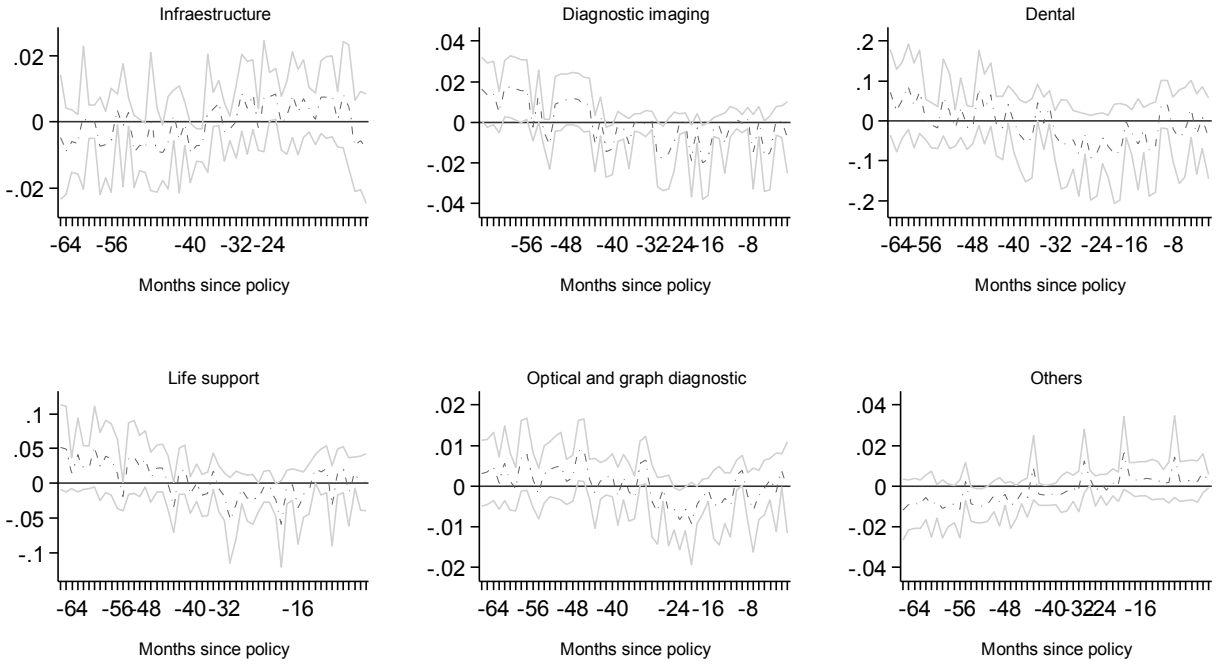
Notes. This displays the difference-in-difference coefficients of each month prior to policy. The regressions include municipality-by-month and month-by-year fixed effects and a constant term. The omitted category is January 2008. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. All dependent variables are measured per 1,000 residents

Figure A4. Testing pretreatment differential trends - Medical equipment.



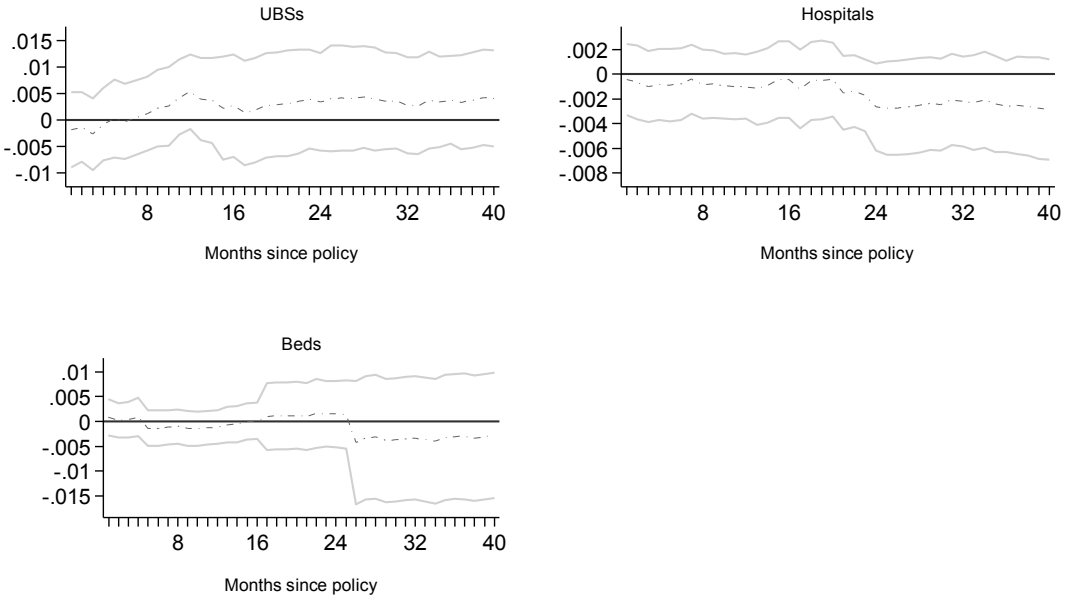
Notes. This displays the difference-in-difference coefficients of each month prior to policy. The regressions include municipality-by-month and month-by-year fixed effects and a constant term. The omitted category is January 2008. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. All dependent variables are measured per 1,000 residents

Figure A5. Testing pretreatment differential trends inn poor areas- Medical equipment.



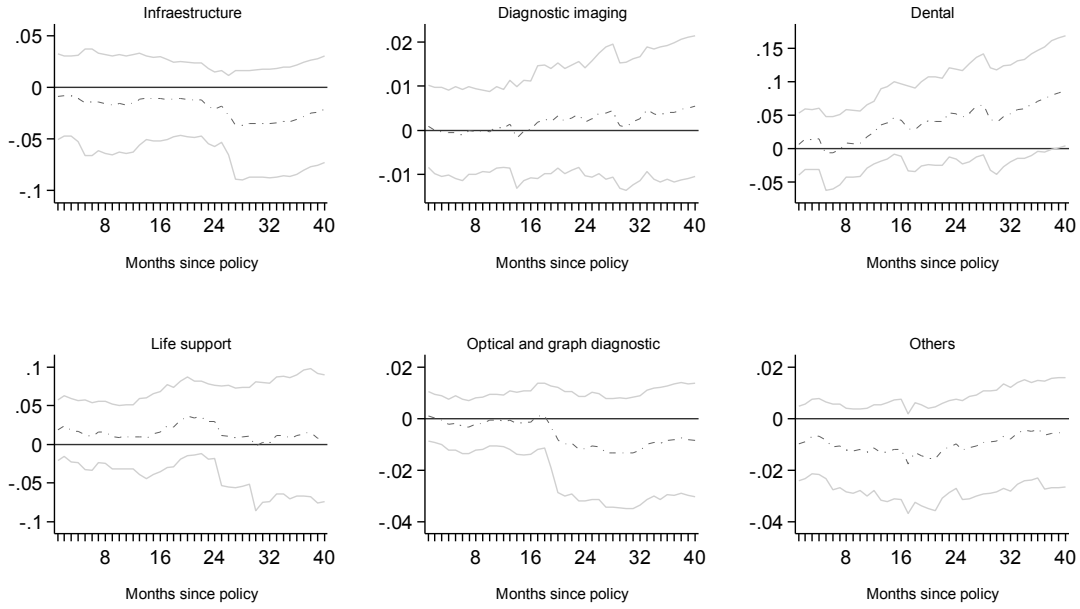
Notes. This displays the difference-in-difference coefficients of each month prior to policy. The regressions include municipality-by-month and month-by-year fixed effects and a constant term. The omitted category is January 2008. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. All dependent variables are measured per 1,000 residents

Figure A6. Effect of policy on local infraestructure



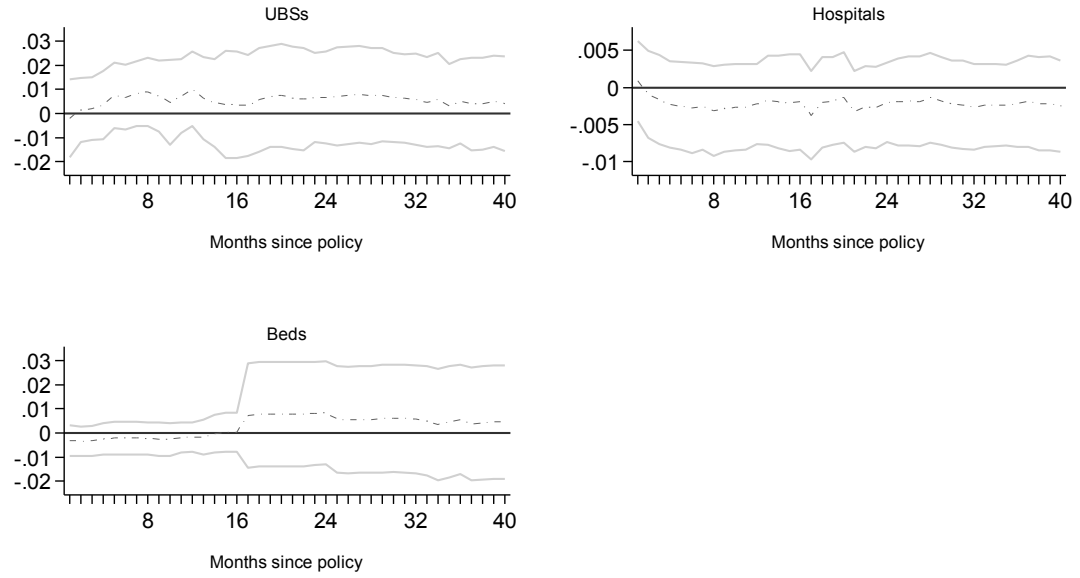
Notes. This displays the difference-in-difference coefficients of each post-policy month. The regressions include municipality-by-month and month-by-year fixed effects, State-linear trends and a constant term. The omitted category is the entire pre-treatment peirod. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. All dependent variables are measured per 1,000 residents

Figure A7. Effect of policy on medical equipment



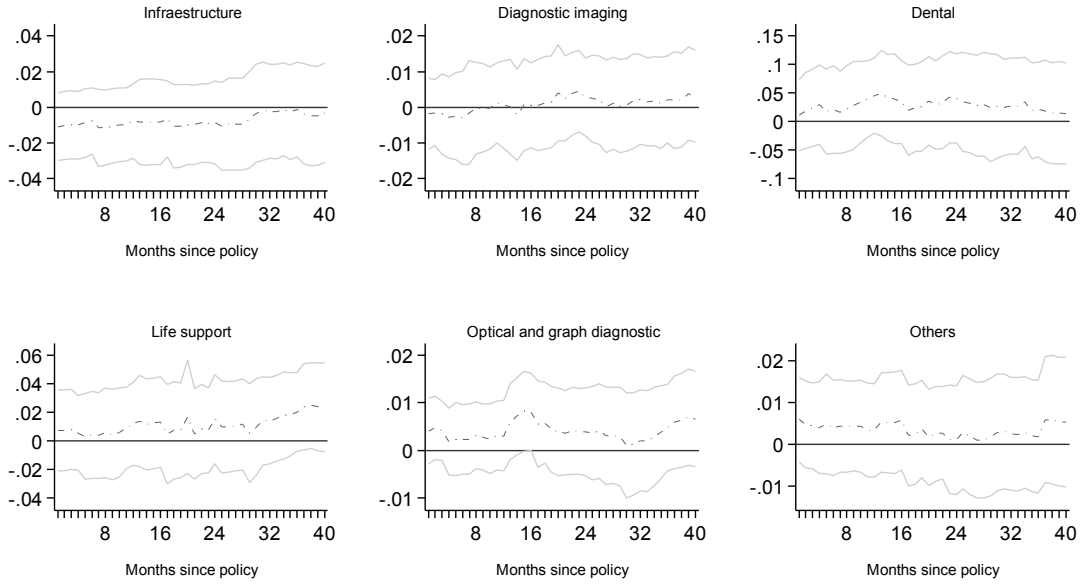
Notes. This displays the difference-in-difference coefficients of each post-policy month. The regressions include municipality-by-month and month-by-year fixed effects, State-linear trends and a constant term. The omitted category is the entire pre-treatment peirod. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. All dependent variables are measured per 1,000 residents

Figure A8. Effect of policy on local infraestructure in poor areas



Notes. This displays the difference-in-difference coefficients of each post-policy month. The regressions include municipality-by-month and month-by-year fixed effects, State-linear trends and a constant term. The omitted category is the entire pre-treatment peirod. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. All dependent variables are measured per 1,000 residents

Figure A9. Effect of policy on medical equipment in poor areas



Notes. This displays the difference-in-difference coefficients of each post-policy month. The regressions include municipality-by-month and month-by-year fixed effects, State-linear trends and a constant term. The omitted category is the entire pre-treatment peirod. The solid lines give the 95% confidence intervals. The dotted lines are the difference-in-indifference coefficients. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. All dependent variables are measured per 1,000 residents

Figure A10. Effect of policy on avoidable hospitalizations in poor areas

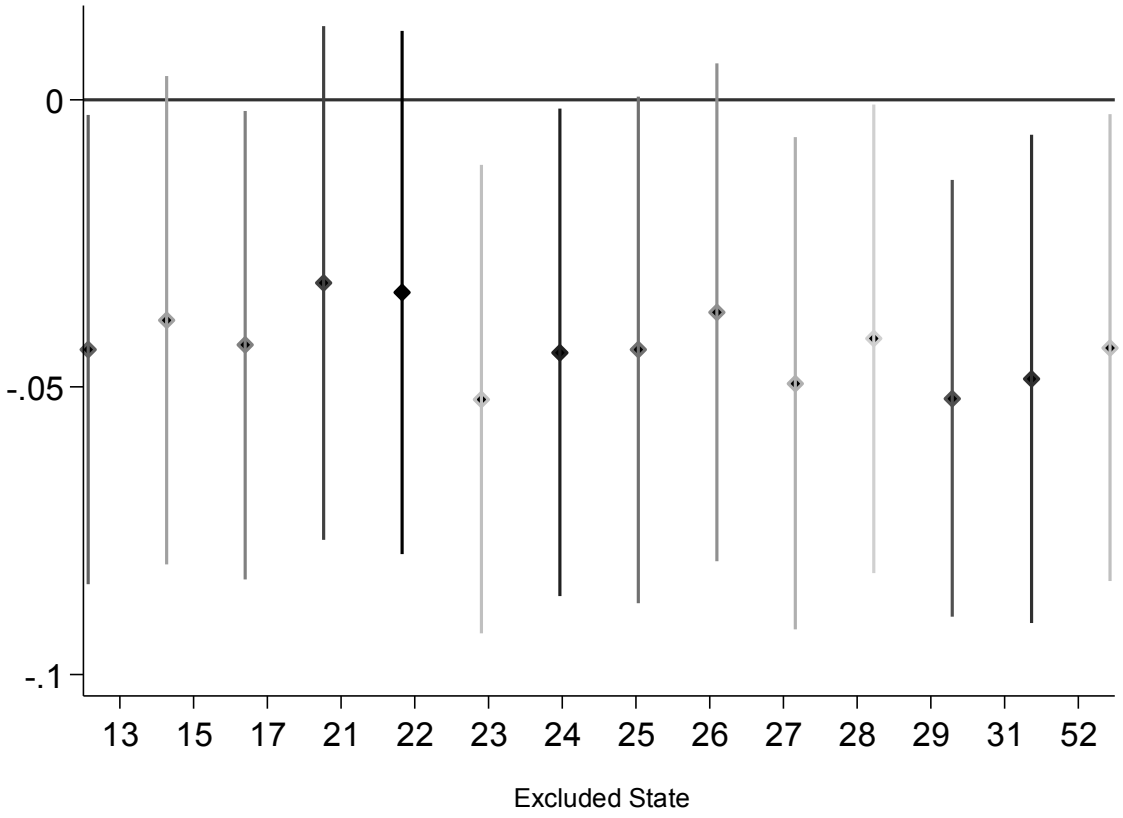


Table A1. Variables and Sources.

Variable	Description	Sources
	Time-variant characteristics	
Physicians in:		
Public sector	Number of physicians in public health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
UBSs	Number of physicians in UBSs. Measured monthly from January 2008 to December 2016.	Ministry of Health
Outside of UBSs	Number of physicians in public sector who are not working in UBSs. Measured monthly from January 2008 to December 2016.	Ministry of Health
Private sector	Number of physicians in private health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
UBSs	Number of Basic Health Units (UBS) in each municipality. Measured monthly from January 2008 to December 2016.	Ministry of Health
Beds	Number of observation beds in public clinics. Measured monthly from January 2008 to December 2016.	Ministry of Health
Hospitals	Number of hospitals. Measured monthly from January 2008 to December 2016.	Ministry of Health
Hospitalizations from injuries	Number of hospitalizations from injuries. Measured monthly from January 2008 to December 2016.	Ministry of Health
Medical equipment		
Infrastructure	Number of air conditioning, generator groups and oxygen plants in public health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
Diagnostic imaging	Number of camera range, mammography, x-ray machines, computerized tomography, magnetic resonance imaging and ultrasound machine in public health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
Dental	Number of complete dental equipment, dental compressor, photopolymerizer, amalgamated dental, low and high speed pen and prophylaxis apparatus in public health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
Life support	Number of intra-aortic balloon, infusion bomb, heated crib, bilirubin meter, debit meter, defibrillator, phototherapy equipment, incubator, temporary pacemaker, pressure monitor, lung resuscitator, and respirator/ventilator in public health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
Optical and graph diagnostic	Number of endoscopy of the Respiratory tract, endoscopy of the urinary tract, digestive endoscope, optometry equipment, laparoscopy, surgical microscope, electrocardiograph and electroencephalograph in public health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
Others	Number of ultrasonic diathermy device, electro stimulation device, hem derivative infusion pump, apheresis equipment, audiometry equipment, extracorporeal circulation equipment, hemodialysis equipment and bier oven in public health facilities. Measured monthly from January 2008 to December 2016.	Ministry of Health
	Time-invariant characteristics	
GDP	2010 Gross domestic product (GDP) per residents.	2010 Census
Indigneous population	Number of residents that are self-declared indigenous	2010 Census
Gini index	Giini index of income distribution.	2010 Census
Unemployment rate	Percentage of economically active population that is not employed.	2010 Census
Illiterate rate	Percentage of people aged 15 or over who cannot read and write.	2010 Census
Rural population	Share of population that lives in rural areas.	2010 Census
Population	Number of residents.	2010 Census
Municipality area	Total municipal area in hectares.	IPEA
Altitude	Altitude above sea level, in meters, of the urban center of each municipality.	IPEA
Distance to capital	Linear distance to the state's capital in thousands of kilometers.	IPEA
Temperature	Mean annual temperature in each municipality in degrees Celsius.	Rocha and Soares (2015)
Rainfall	Mean annual rainfall level in each municipality in millimeters	Rocha and Soares (2015)
Legal Amazon region	Dummy indicating whether the municipality is part of Legal Amazon region	IPEA
Semiarid region	Dummy indicating whether the municipality is part of semiarid region.	IPEA

Table A2. Determinants of PMM Adoption (Analysis using OLS models)

	OLS				
	(1)	(2)	(3)	(4)	(5)
UBS rate 2008	-0.149	-0.018	-0.015	-0.03	
	[0.079]*	[0.073]	[0.074]	[0.070]	
UBS rate 2009	-0.094	-0.141	-0.165	-0.139	
	[0.143]	[0.133]	[0.138]	[0.141]	
UBS rate 2010	0.034	0.043	0.037	0.016	
	[0.130]	[0.129]	[0.133]	[0.139]	
UBS rate 2011	0.219	0.256	0.27	0.275	
	[0.090]**	[0.092]**	[0.093]***	[0.095]***	
UBS rate 2012	-0.398	-0.251	-0.244	-0.254	
	[0.092]***	[0.074]***	[0.077]***	[0.078]***	
Physician rate 2008	-0.084	-0.054	-0.048		-0.05
	[0.040]**	[0.036]	[0.036]		[0.033]
Physician rate 2009	0.051	0.063	0.063		0.049
	[0.055]	[0.042]	[0.041]		[0.039]
Physician rate 2010	0	-0.001	-0.001		-0.001
	[0.000]	[0.000]	[0.000]*		[0.000]**
Physician rate 2011	0.02	0.019	0.022		0.033
	[0.044]	[0.041]	[0.042]		[0.042]
Physician rate 2012	-0.055	-0.02	-0.027		-0.037
	[0.044]	[0.039]	[0.039]		[0.041]
Per capita GDP		-0.004	-0.004	-0.004	-0.004
		[0.001]***	[0.001]***	[0.001]***	[0.001]***
% indigenous population		0.001	0.001	0.001	0.001
		[0.001]	[0.001]	[0.001]	[0.001]
Gini index		0.166	0.128	0.137	0.137
		[0.151]	[0.160]	[0.161]	[0.162]
Unemployment rate		0.002	0.002	0.002	0.002
		[0.003]	[0.002]	[0.002]	[0.002]
Illiterate rate		0.004	0.004	0.004	0.004
		[0.001]***	[0.002]**	[0.002]**	[0.002]**
% rural population		0.001	0.001	0.001	0.001
		[0.001]**	[0.000]**	[0.000]**	[0.000]**
Log of population		0.118	0.12	0.122	0.128
		[0.009]***	[0.009]***	[0.009]***	[0.008]***
Municipality area			0.000	0.000	0.00
			[0.000]	[0.000]	[0.000]
Altitude			-0.004	-0.004	-0.004
			[0.003]	[0.003]	[0.003]
Distance to capital			0.000	0.000	0.000
			[0.000]	[0.000]	[0.000]
Temperature			-0.004	-0.003	-0.005
			[0.008]	[0.009]	[0.008]
Rainfall			0.000	0.000	0.000
			[0.000]	[0.000]	[0.000]
Legal Amazon region			0.229	0.231	0.238
			[0.012]***	[0.012]***	[0.011]***
Semiarid region			0.018	0.019	0.018
			[0.038]	[0.038]	[0.039]

Notes. The data is at the municipality-level. All regressions include State fixed effects. Robust standard errors (reported in brackets) are clustered at the State level. Significance: $*$ $p < 0.10$ $**$ $p < 0.05$, $***$ $p < 0.01$. N=5,439.

Table A3. Determinants of PMM Adoption (Analysis using logit models)

	Logit				
	(6)	(7)	(8)	(9)	(10)
UBS rate 2008	-0.705	0.074	0.092	0.034	
	[0.420]*	[0.360]	[0.358]	[0.339]	
UBS rate 2009	-0.487	-0.765	-0.874	-0.747	
	[0.723]	[0.675]	[0.706]	[0.716]	
UBS rate 2010	0.196	0.249	0.186	0.089	
	[0.663]	[0.646]	[0.668]	[0.686]	
UBS rate 2011	1.132	1.443	1.463	1.476	
	[0.481]**	[0.523]***	[0.509]***	[0.507]***	
UBS rate 2012	-2.104	-1.426	-1.356	-1.372	
	[0.536]***	[0.421]***	[0.420]***	[0.418]***	
Physician rate 2008	-0.387	-0.208	-0.179		-0.168
	[0.189]**	[0.161]	[0.160]		[0.148]
Physician rate 2009	0.233	0.319	0.303		0.236
	[0.258]	[0.182]*	[0.182]*		[0.175]
Physician rate 2010	-0.002	-0.002	-0.003		-0.003
	[0.001]	[0.002]	[0.001]*		[0.001]**
Physician rate 2011	0.098	0.075	0.087		0.14
	[0.202]	[0.187]	[0.194]		[0.193]
Physician rate 2012	-0.248	-0.016	-0.051		-0.1
	[0.203]	[0.189]	[0.191]		[0.197]
Per capita GDP		-0.02	-0.02	-0.021	-0.021
		[0.005]***	[0.004]***	[0.004]***	[0.004]***
% indigenous population		0.013	0.009	0.009	0.009
		[0.013]	[0.012]	[0.012]	[0.012]
Gini index		0.807	0.465	0.502	0.527
		[0.879]	[0.941]	[0.943]	[0.953]
Unemployment rate		0.009	0.009	0.009	0.01
		[0.015]	[0.013]	[0.013]	[0.013]
Illiterate rate		0.023	0.02	0.02	0.02
		[0.010]**	[0.011]*	[0.010]*	[0.011]*
% rural population		0.006	0.006	0.006	0.006
		[0.003]**	[0.003]**	[0.002]**	[0.003]**
Log of population		0.752	0.711	0.716	0.746
		[0.052]***	[0.060]***	[0.056]***	[0.067]***
Municipality area			0.000	0.000	0.000
			[0.000]***	[0.000]***	[0.000]***
Altitude			-0.021	-0.02	-0.021
			[0.019]	[0.019]	[0.019]
Distance to capital			0.000	0.000	0.000
			[0.000]	[0.000]	[0.001]
Temperature			-0.016	-0.014	-0.021
			[0.045]	[0.045]	[0.046]
Rainfall			-0.003	-0.003	-0.003
			[0.003]	[0.003]	[0.003]
Legal Amazon region			1.415	1.419	1.462
			[0.076]***	[0.076]***	[0.078]***
Semiarid region			0.107	0.105	0.103
			[0.254]	[0.255]	[0.263]

Notes. The data is at the municipality-level. All regressions include State fixed effects. Physician rate refers to doctors who work in UBSs per 1,000 residents. Robust standard errors (reported in brackets) are clustered at the State level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01. N=5,439.

Table A4. Avoidable Hospitalization

Condition	ICD-10-CM Codes	(%)
Immunization preventable conditions	A37, A36, A33-A35, B26, B06, B05, A95, B16, G00.0, A17.0, A19, A15.0-A15.3, A16.0-A16.2, A15.4-A15.9, A16.3-A16.9, A17.1-A17.9, A18, I00- I02, A51-A53, B50-B54	13.50
Gastroenteritis	E86, A00-A09	33.23
Anemia	D50	0.18
Nutritional deficiencies	E40-E46, E50-E64	0.95
Ear, nose and throat infections	H66, J00, J01, J02, J03, J06, J31	2.91
Bacterial pneumonia	J13, J14, J15.3, J15.4, J15.8, J15.9, J18.1	12.10
Asthma	J45, J46	13.39
Pulmonary diseases	J20, J21, J40, J41, J42, J43, J47, J44	9.8
Hypertension	I10, I11	0.09
Angina	I20	0.01
Cardiac insufficiency	I50, J81	0.52
Vascular brain diseases	I63 a I67; I69, G45 a G46	0.12
Diabetes	E10.0, E10.1, E11.0, E11.1, E12.0, E12.1, E13.0, E13.1, E14.0, E14.1, E10.2-E10.8, E11.2-E11.8, E12.2-E12.8, E13.2-E13.8, E14.2-E14.8, E10.9, E11.9, E12.9, E13.9, E14.9	0.32
Epilepsies	G40, G41	2.63
Infection of the kidney and urinary tract	N10, N11, N12, N30, N34, N39.0	4.42
Infection of the skin and subcutaneous tissue	A46, L01, L02, L03, L04, L08	3.57
Inflammatory disease female pelvic organs	N70, N71, N72, N73, N75, N76	0.01
Gastrointestinal ulcer	K25-K28, K92.0, K92.1, K92.2	0.14
Diseases related to prenatal and delivery	O23, A50, P35	1.87

Source: Ordinance 221, April 2008.
Notes. The last column is the share of each specific avoidable hospitalization condition in the total in the 2008-2016 period.

Table A5. The Effect of PMM on Hospitalizations from external causes

	Basic	Add State-linear trends
	(1)	(2)
	Panel A: All municipalities	
PMM x Post	0.013	0.014
	[0.010]	[0.009]
R^2	0.584	0.586
N	1,174,824	1,174,824
	Panel B: Poor areas	
PMM x Post	0.002	0.007
	[0.008]	[0.008]
R^2	0.308	0.311
N	290,304	290,304

Notes. Dependent variable is $ln(1 + hospitazalitons\ from\ external\ causes)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. **1**(.) is an event-time indicator equal to 1 for each year. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table A6. The Effect of PMM on Avoidable Hospitalizations (Alternative specifications)

	Baseline	Add State quadratic trends	Add State-by-month-by-year fixed effects	Add mesoregion linear trends	Add mesoregion quadratic trends	Add microregion linear trends	Add microregion quadratic trends
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
PMM *x* Post	-0.048	-0.049	-0.052	-0.046	-0.045	-0.053	-0.056
	[0.024]**	[0.023]**	[0.022]**	[0.022]**	[0.022]**	[0.022]***	[0.020]***
R^2	0.638	0.642	0.668	0.648	0.658	0.664	0.688

Notes. Dependent variable is $ln(1 + avoidable\ hospitazalitons)$. All regressions use entropy balancing weights and municipality-by-month-by-year-sex and month-by-year fixed effects. In addition, column (1)-(2) include State linear trends. Columsn (3)-(7) drop State linear trends and instead include trends as indicated. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. The sample consists of municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the State level. *N* =290,304 (1,344 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table A7. The Effect of PMM on Avoidable Hospitalizations (Mean reversion)

		Add		
	Baseline	1 lags	6 lags	12 lags
	(1)	(2)	(3)	(4)
PMM *x* Post	-0.048	-0.032	-0.019	-0.018
	[0.024]**	[0.018]*	[0.011]*	[0.010]*
Total effect in low GDP areas	-0.048	-0.044	-0.041	-0.040
R^2	0.638	0.667	0.683	0.69
N	290,304	287616	274176	258048

Notes. Dependent variable is $ln(1 + avoidable\ hospitazalitons)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. The sample consists of municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the mesoregion level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table A8. The Effect of PMM on Avoidable Hospitalizations (Variance-covariance matrix)

	Standard errors clustered at the			
	Mesoregion-level (Baseline)	Municipality-level	Microregion-level	UF level
	(1)	(2)	(3)	(4)
PMM *x* Post	-0.048 [0.024]**	-0.048 [0.022] **	-0.048 [0.021] **	-0.048 [0.023] **
R^2	0.638	0.638	0.638	0.638

Notes. Dependent variable is $ln(1 + avoidable\ hospitazalitons)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. The sample consists of municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets). *N* =290,304 (1,344 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table A9. The Effect of PMM on Avoidable Hospitalizations (Specification checks)

	Baseline	Hyperbolic sine parameterization	Poisson count model	Negative binomial count model
	(1)	(2)	(3)	(4)
PMM *x* Post	-0.048	-0.051	-0.100	-0.083
	[0.024]**	[0.031]*	[0.55]*	[0.046]*

Notes. All regressions use entropy balancing weights. Column (1)-(2) control for state-linear trends and municipality-by-month-by-sex and month-by-year fixed effects. Columns (3)-(4) State fixed effects, quadratic time trends, a dummy indicating treated municipalities (*PMM*) and a dummy indicating the post-period policy (*Post*). The sample consists of municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the mesoregion level. Hyperbolic sine parameterization is given by $\ln(x + \sqrt{1 + x^2})$. *N* =290,304 (1,344 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table A10. The Effect of PMM on Avoidable Hospitalizations (Outliers)

		Excluding residuals observations below and above	
	Baseline	5 and 95th percentile	1 and 99th percentile
	(1)	(2)	(3)
PMM *x* Post	-0.048	-0.032	-0.039
	[0.024]**	[0.015]**	[0.021]*
R^2	0.638	0.787	0.689

Notes. Dependent variable is $ln(1 + avoidable\ hospitazalitons)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. The sample consists of municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the mesoregion level. Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

Table A11. The Effect of PMM on Health Infrastructure Network

				Medical equipment					
	UBSs	Beds	Hospitals	Infrastructure	Diagnostic imaging	Dental	Life support	Optical and graph diagnostics	others
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
				Panel A: all municipalities					
PMM x Post	0.002	-0.001	-0.001	-0.029	-0.001	0.017	0.015	-0.007	-0.011
	[0.004]	[0.003]	[0.001]	[0.022]	[0.006]	[0.037]	[0.027]	[0.008]	[0.009]
Mean of dep. var.	0.280	0.034	0.055	0.145	0.319	1.497	0.831	0.200	0.278
R^2	0.808	0.866	0.847	0.763	0.908	0.833	0.900	0.821	0.903
				Panel B: poor areas					
PMM x Post	0.005	0.002	-0.002	-0.009	-0.002	0.025	0.012	0.003	0.003
	[0.009]	[0.007]	[0.003]	[0.011]	[0.006]	[0.036]	[0.015]	[0.004]	[0.006]
Mean of dep. var.	0.300	0.029	0.040	0.058	0.137	0.938	0.244	0.071	0.061
R^2	0.777	0.892	0.740	0.716	0.789	0.859	0.856	0.781	0.846

Notes. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year and month-by-year fixed effects. All dependent variables ate measured per 1,000 residents. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. Sample sizes in all and poor areas are 587,412 and 145,152, respectively. Significance: $*$ $p < 0.10$ $**$ $p < 0.05$, $***$ $p < 0.01$.

Table A12. Reduced-form of PMM and IV Estimates of Physicians in Poor Municipalities

	Physician	Avoidable hospitalizations	Hospital cost
	(1)	(2)	(3)
		IV estimates	
Physician	-	-0.670	-2314.48
		[0.324]**	[783.071]***
	First stage	Reduced-form	
PMM *x* Post	0.071	-0.048	-164.328
	[0.009]***	[0.024]**	[56.456]***
F-stat excluded Instrument	107.155		

Notes. Dependent variable in column (1) is $ln(1 + avoidable\ hospitazalitons)$. All regressions use entropy balancing weights and include State-linear trends and municipality-by-month-by-year-sex and month-by-year fixed effects. *PMM* is a dummy variable equal to one if the municipality implemented the program and zero otherwise. *Post* is a dummy variable for observations from September 2013 to December 2016, the post-policy period. Poor areas refers to municipalities at the lowest quartile of per capita GDP distribution. Robust standard errors (reported in brackets) are clustered at the Mesoregion level. *N* =290,304 (1,344 municipalities, 12 months, 9 years, 2 sex). Significance: $*$ p < 0.10 $**$ p < 0.05, $***$ p < 0.01.

5. Concluding Remarks

The three essays presented here investigated three important issues in health economics using Brazilian data. Overall, the essays aim to examine the short-run consequences of poor early health as well as the impact of large-scale interventions on child health. The findings from these essays may contribute to the development of public policies aimed at ameliorate inequalities originated in early life in developing countries.

The first essay sheds light on the importance of birth weight for infant health in a developing country context. In doing so, we address several limitations of previous studies by using rich administrative data on the universe of births in Brazil. Our estimates imply that very low birth babies display higher risk of death within one year. These effects are generally larger than those estimated with data from the US and Norway, suggesting that there may be differences in estimates for developed and developing countries. In turn, this suggests that using estimates derived from rich countries may understate the benefits from interventions aimed at decreasing infant mortality by increasing birth weight in developing countries.

In the second essay, we provide estimates of the externalities of a deforestation control policy in infant health and find that the Brazilian conservation policy launched in 2004 had positive effects on infant health measured by low birth weight and prematurity, especially for boys. The evidence suggests that improvements in air pollution were an important pathway. Compared to the effects of other environmental risk factors in other countries, the conservation policy had smaller impacts on infant health. these results have important implications for policy and contribute to the growing literature on the importance of a healthy fetal environment. In particular, cost-benefit analyses of deforestation control policies should take into account the potential benefits in infant health by lower local air pollution levels due to reduced rates of deforestation.

Finally, the third essay presents novel evidence on the effect of a large-scale program designed to increase the supply of physicians on child health in Brazil. The literature on this topic is very scarce, even in developed economies. We fill this gap by exploiting a dramatic change policy that generated a sharp increase in the number of

doctors willing to provide primary health care in remote and poor areas in Brazil. Our results indicate that this policy change reduced significantly avoidable, ambulatory sensitive hospitalizations among children only in poor areas. We estimate that this policy reduced avoidable hospitalizations by 4.8 percent in these areas. An interesting finding is that the policy had the largest effects for children 0-1 years old and for gastroenteritis and asthma conditions. Together, these findings suggest that increased supply of primary care physicians results in more efficient use of health care resources, fewer avoidable hospitalizations and better child health among disadvantaged families. Our calculations suggest that policy implementation was responsible for almost 70 percent of the reduction in the gap between poor and non-poor areas in child hospitalizations.

As a whole, these essays provide inputs that may be used in cost-benefit analysis of policies that seek reducing inequalities originated in early life by improving child health. Previous studies have documented that poor health during the first years of life have adverse effects on later life outcomes measured by cognition, education and income. Thus, the evidence that deforestation control policies and increased access to primary health care have externalities positive on child health suggests that such policies may have long-lasting effects on human capital. Naturally, there are several questions that should be addressed in future research. In particular, future studies linking later socio-economic outcomes with birth records could yield even greater insights into the long-term impacts of these policies as well as of low birth weight.

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