Congenital Disability and Parents' Labor Supply: Evidence from the Zika Virus Outbreak

João Garcia* Rafael Latham-Proença† Marcela Mello‡

September 30, 2024

Please click here for the latest version

Abstract

Having a child with a severe disability ranks among the most consequential life shocks a parent can experience, but we know little about its economic effects. We study a shock to disability incidence caused by the Zika Virus epidemic in Brazil, which caused thousands of children to be born with microcephaly. Using data on the universe of births and formal employment links, we find that mothers of Zika-affected infants experience a 26% (Confidence Interval: $[-32.0, -20.8]$) decrease in formal employment and a 21.9% (CI: $[-31.54\%, -12.26\%]$) reduction in earnings, compared to control mothers, equivalent to a doubling of the motherhood penalty. Informal employment does not seem to increase to compensate. We show suggestive evidence of significant disemployment effects of social security benefits, but effects are still significant for non-recipients. In contrast, father's labor outcomes were unaffected. We also find lower fertility for affected families as well as local spillovers, but no effect on marriage dissolution.

*Universidad de Santiago de Chile. Email: joao.garcia@usach.cl.

†Open Philanthropy. Email: rafaelproenca@g.harvard.edu.

‡Universidad de los Andes. Email: mmello@uandes.cl.

Acknowledgement: We thank Anna Aizer, Michela Carlana, Anders Jensen, Eliana La Ferrara, Dan Björkegren, Andrew Foster, John Friedman, Emily Oster, Vítor Possebom, Flávio Riva, Jon Roth, Bryce Steinberg, and Neil Thakral for helpful comments and discussions.

1 Introduction

Parental labor market decisions are influenced by their children's characteristics, and severe, permanent disability may be one of the most profoundly impactful factors. Traditionally, women have taken on the additional demands, so the dip in labor market participation after childbirth may be larger for mothers of disabled children. This dip is especially problematic because disabled children also need more financial resources for medical treatment and adaptation in addition to time and attention. Therefore, estimating the effect of child disability on maternal employment is crucial for the design of policies that can support these families.

The existing literature on child disability and maternal employment faces challenges dealing with unobserved confounders. For instance, mothers who follow preventive recommendations such as folate supplementation or abstaining from smoking are less likely to have children with disabilities than those who do not, and also likely different on unobservable characteristics that affect labor market outcomes. This confounding effect invalidates standard event-study around birth approach – such as the one used by Kleven et al. (2019) in the context of the general child penalty – to examine the impact of births with disability. No work on congenital child disability has, to the best of our knowledge, dealt explicitly with this endogeneity concern (Chen et al., 2023; Cheung et al., 2023; Gunnsteinsson & Steingrimsdottir, 2019; Powers, 2001, 2003; Salkever, 1982; Wasi et al., 2012). While this endogeneity concern has been identified and addressed in recent literature on child health and developmental issues, the existing approaches are difficult to apply to cases of congenital disability.[1]

[1]The instrumental variables approach of Frijters et al. (2009) limits the effect to a relatively narrow LATE, and the panel data approach used by Breivik and Costa-Ramón (2022) leveraging differential timing of onset cannot be applied to congenital disability because there is no variation in timing, as most congenital disabilities are detected at birth or shortly after that.

In this paper, we exploit the 2015 Zika virus outbreak in Brazil to estimate the causal effects of child disability on parental labor force participation, household composition, fertility, and income. The outbreak caused several thousands of children to be born with microcephaly, a severe disability. We argue that the sudden onset of this event and the characteristics of the infection rule out the endogeneity of maternal health behaviors. Using detailed data on the universe of births and formal employment links in the country, we show that, before childbirth, affected mothers had similar labor market trajectories to other mothers matched on a simple set of characteristics. However, starting at the end of the typical maternity leave period, their labor force participation and earnings decline much faster. From six months after childbirth, mothers of children with microcephaly are on average 47.07% (CI: $[37.79\%, 56.37\%]$) less likely to have a job in the formal sector than mothers in the control group. This difference persists for as long as we can estimate (36 months). This means mothers of children with microcephaly face a 60.0% (CI: $[45.10\%, 75.05\%]$) larger motherhood penalty when compared with controls.

Using self-reported data, we find that informal employment does not seem to increase to compensate. Our analysis suggests that social security benefits may lead to large reductions in employment, though these effects are also observed among non-recipients. Interestingly, fathers' participation in the formal labor market appears unaffected. Additionally, we observed decreased fertility rates in families with children with microcephaly, but no effect on divorce. We also find negative local spillover effects on fertility, with women in areas of higher benefit incidence responding to the situation by having fewer children or delaying pregnancy.

We leverage three rich administrative datasets to conduct this study. The first is SINASC/SUS, which logs all births in the country and provides details on the municipality and date of the delivery, the mother's residence, the mother's date of birth, and whether the newborn has microcephaly. The second is Annual Account of Social Infor-

mation (Relação Anual de Informações Sociais, RAIS). This dataset allows us to follow an individual's employment history throughout the entire period and observe monthly earnings, hours, and maternity leave dates. We link these two datasets using the Single Registry[2], a federal registry of all recipients of social programs. Linking these datasets is not trivial due to the lack of common individual identifiers. To deal with this issue, we exploit the fact that both the births dataset and Single Registry include the mothers' date of birth, child's birth, and municipality of residence. These variables are enough information to uniquely identify individuals in all but 1% of cases of births with microcephaly in the sample. We drop all cases that cannot be uniquely linked between the datasets.

To isolate the causal effect of child disability, we compare the labor market trajectories of mothers of children with microcephaly to a matched comparison group. This group consists of mothers of the same age and educational level who gave birth in the same month and municipality as the mothers with a child with microcephaly. We compare the average labor force participation between these two groups each month following maternity leave.

We argue that this method yields causal estimates for a few main reasons. First and most important, the unexpected nature of the epidemic and the characteristics of the infection make selection bias unlikely. Since the Zika virus is transmitted by a common mosquito, anyone in affected areas could have been exposed. The sudden introduction of the virus, along with its undiscovered link with natal defects, means that differences in preventive behavior are unlikely: no one could have known to be concerned. Even after public health authorities identified the outbreak and raised awareness, prevention had only a long-delayed effect because infection is more likely to cause disability when it happens in the first trimester of pregnancy.

[2]Recipients undergo interviews with local government agents and answer a standardized questionnaire on the socioeconomic characteristics of all household members. Recipients must keep this information updated every couple of years to ensure eligibility for social programs.

Other potential threats to identification can be ruled out due to characteristics of the virus. Selective abortion is unlikely because Zika infection is asymptomatic in most cases, so women are unaware, and microcephaly is difficult to diagnose before birth. Zika has no lasting effects on adults, ruling out direct effects on labor supply not causally mediated by effects on the child. The fact that we find nearly identical prior labor market trajectories corroborates the argument that exposure is random conditional on observables.

The literature on the effects of child disability on parents' labor supply is still small. Powers (2001), Salkever (1982), and Wasi et al. (2012). More recently, Chen et al. (2023) and Cheung et al. (2023) study the impact of congenital disability in Taiwan, Gunnsteinsson and Steingrimsdottir (2019) in Denmark, and Martínez et al. (2023) in Chile. Our paper contributes to this literature by examining the case of an arguably exogenous increase in the chance of having a child with congenital disability. While this literature so far can only control for observable characteristics that are related to disability, the use of an exogenous shock provides a stronger argument for identification.

We also contribute to the literature on parental response to children's adverse health. Most previous research focuses on maternal labor supply only and relies mainly on survey data, which have limited capacity to examine parents' dynamic responses due to a lack of extended follow-up (Burton et al., 2017; Frijters et al., 2009; Lafférs & Schmidpeter, 2021; Wolfe & Hill, 1995). More recent work using longitudinal administrative data looked at parental labor supply response to various child health shocks (Adhvaryu et al., 2022; Breivik & Costa-Ramón, 2022; Chen et al., 2023; Cheung et al., 2023; Eriksen et al., 2021; Vaalavuo et al., 2023). Our study is restricted to a particular type of congenital disease caused by an exogenous shock. Therefore it is unlikely to be correlated to parents' behaviors, genetics, or age, mitigating bias in the estimated effects.

Our paper is also related to the literature on the motherhood penalty and gender inequality (Berniell et al., 2021; Budig & England, 2001; Cortés & Pan, 2023; De Quinto et al., 2020; Kleven et al., 2019; Musick et al., 2020; Sieppi & Pehkonen, 2019). While this literature on the motherhood penalty has extensively documented the adverse labor market effects on mothers post-childbirth, there is a paucity of research focusing specifically on how these penalties are exacerbated when a child is born with a severe disability. Our study addresses this gap by quantifying the additional motherhood penalty associated with microcephaly, thereby contributing to a deeper understanding of gender disparities in the labor market.

2 Background

The 2015 outbreak of Zika in Brazil provides an exogenous shock to the rate of child disability, with other characteristics that also help to isolate its effect on mothers' employment. Selection driven by differences in preventive behavior is addressed by the sudden and widespread nature of the outbreak across the affected regions. Selective abortion is unlikely because diagnosis is difficult *in utero*, adults have no symptoms, and it is illegal in Brazil. The lack of symptoms also rules out direct effects of the virus on labor outcomes.

2.1 The Zika Virus and Microcephaly

Zika is a flavivirus of the same genus as the viruses responsible for dengue, yellow fever, and West Nile fever. It was initially isolated in Uganda and was endemic to tropical areas of Africa, Asia and Oceania. Zika spreads through a common mosquito, the *Aedes aegypti*. Diseases carried by the *Aedes aegypti* also include dengue and chikungunya,

which together affect around 2 million Brazilians per year. Before 2014, the Zika virus had never been observed in Brazil or anywhere else in the Americas.

Exposure to the Zika virus in pregnant women, especially in the first trimester, can cause microcephaly in the child, a severe and lifelong disability. Microcephaly is characterized by underdevelopment of the brain, resulting in a smaller head circumference than normal. Children with microcephaly need frequent medical and parental attention. They often suffer from seizures, vision and hearing problems, intellectual disabilities, and difficulty with motor and speech development. Brazil's public health care system offers free treatment, including continuing therapy, but families may have trouble accessing it, particularly in remote areas.

In contrast with the dramatic effects on newborns, Zika infection has no lasting effects in adults, so it should not directly impact labor supply. About 80% of adult cases show no symptoms (Haby et al., 2018). In the other cases, typical symptoms are fever and rashes lasting up to a week. One exception is that there have been reports of an increased chance of developing Guillain-Barré syndrome, a severe, potentially lethal condition. However, even this increased risk is extremely rare and would not have any relevant impact on our results.

2.2 The 2015 Outbreak

The virus was introduced to Brazil around 2014, where it had never been observed before. The outbreak was first identified in late 2015 following a spike in cases of microcephaly. Brazilian researchers had been observing a new disease with symptoms similar to dengue for months before they identified the virus as Zika. At the time, it was not known that Zika infection could result in microcephaly of newborns. In areas where Zika is endemic, microcephaly is not often observed because women are typically exposed before pregnancy

and develop immunity.

The outbreak was focused on the Northeast, started suddenly, and ended fast. Figure 1 shows a map with the number of microcephaly cases per 1000 births in 2015 and 2016 in each of the five regions of Brazil. The Northeast region was hit the hardest by the epidemic, reaching an average rate of 1.55 births with microcephaly per 1,000, or 1,305 total cases. The South was relatively untouched, and the other regions had intermediate levels of incidence. Other than this regional variation, there are no apparent spatial patterns that could indicate, for instance, strong clustering around cities receiving tourists at the time.

Figure 2 shows the timeline of the epidemic, with cumulative cases in the top graph and monthly cases in the bottom. During the second half of 2015, the number of cases increased abruptly, from close to zero to the peak incidence in just about three months. The subsequent fall in cases was almost as fast, with a much more modest second wave in the latter half of 2016. This fast timeline is convenient for identification purposes, since the scope for preventive measures was very limited.

Differential exposure to the virus based on variations in mothers' preventive behavior is unlikely to cause bias for two primary reasons. First, Zika had never been observed in Brazil, and second, the link to microcephaly in newborns was unknown. The first signs of a new disease were observed in March 2015, and researchers first identified the increase in microcephaly in October. Researchers could only establish the causal link between these factors in 2016, so mothers would only know to take precautions afterward. Even then, preventive measures would likely only result in a reduction in cases of disabled children with a significant delay.[3] Since the virus is more likely to cause microcephaly

[3]One important caveat is that, since Zika has the same vector as dengue, it could be that health-sensitive mothers were protected from Zika due to efforts to prevent dengue. While we cannot totally rule out this concern, we believe it is minimized by the fact that dengue is difficult to prevent at the individual level. Most prevention efforts are based on controlling the vector through community-based

during the first trimester of pregnancy (Cauchemez et al., 2016; Johansson et al., 2016), its effects may remain undetected for several months.

2.3 An Informal Model of Infection

This section presents an informal model of the Zika outbreak. The goal is to articulate the main characteristics of the outbreak according to the medical literature, and link them to the modeling assumptions we will use in studying it.

The Zika virus spreads to humans through a mosquito vector. Therefore, the physical presence of the mosquito in the environment is a precondition for the outbreak. Specifically, the *Aedes aegypti*, which is also responsible for dengue fever, is the primary vector. This mosquito is endemic to most of the territory of Brazil, although there is variation in susceptibility, with some areas being unfavorable (such as drier parts of the cerrado) (Kraemer et al., 2015).

Similarly the *Aedes aegypti* is more active during the rainy season, because its life cycle depends on stagnant water (Lowe et al., 2011). Therefore, the concentration of vectors that make infection possible varies with the physical conditions of each area and with seasonal climate. Other municipality-level factors can affect the concentration of vectors, such as urbanization and public health measures. We denote the concentration of mosquito vectors in a given municipality m at a month t as c_{mt}.

Once the Zika virus is introduced, the presence of the vector can cause microcephaly. We can infer that the virus did not spread quickly enough to be present in the entire country at once because, if that were the case, the incidence of microcephaly would have been roughly proportional to the incidence of dengue. However, there are clear

programs, biological or chemical methods (Khan et al., 2023; Murray et al., 2013).

differences, such as the Northeast region having a much higher incidence of births with microcephaly than the Central-West region, which is not true for dengue. Let's denote the share of vectors that carry the virus as λ_{mt}.[4]

An individual's chance of infection will depend on the concentration of infected vectors in their municipality, $c_{mt}\lambda_{mt}$, but also on individual risk factors. Some risk factors identified in the literature include age (Siqueira-Junior et al., 2008), education level (ibid.), employment (Teurlai et al., 2015), urbanization and population density (Wu et al., 2009), as well as socioeconomic status (Delmelle et al., 2016). However, incidence of *Aedes aegypti*-borne disease is complex and not fully understood. For instance, there is mixed evidence on whether incidence is greater on areas of lower socioeconomic status, with almost half of studies finding a null or opposite relationship (Whiteman et al., 2020).[5] We denote individual characteristics relevant for infection risk X_i.

The main threat to identification in our context are that any one of c_{mt}, λ_{mt} and X_i can be correlated with higher labor force participation. To deal with this, we match mothers of affected children to unaffected mothers in the same municipality and month (thus, equalizing the values of c_{mt} and λ_{mt}) and with similar observable characteristics (X_i). Thus, our identification assumption is conditional independence.

Another potential threat to identification – differential rates of abortions – is unlikely for several reasons. First, microcephaly is difficult to identify *in utero*. Diagnosis is both expensive, requiring detailed ultrasound imaging, and inaccurate (Chervenak et al., 1984; Leibovitz & Lerman-Sagie, 2018). Mothers would have to decide to terminate the

[4]Pedrosa et al. (2020) presents evidence that the presence that a toxin produced by cyanobacteria may play a role in determining whether Zika infection during pregnancy results in microcephaly. For our purposes, such environmental factors would also be included in λ_{mt}.

[5]In the specific context of the zika virus epidemic in Brazil, there is some evidence suggesting lower-income individuals had higher exposure, at least within cities (Lobkowicz et al., 2021; Souza et al., 2018). Our sample is restricted to lower-income families for both treatment and controls, minimizing heterogeneity along this dimension.

pregnancy without confirmation that their child is affected. Second, Zika infection is often asymptomatic and otherwise can be similar to dengue, making it difficult for mothers to know if they have been infected. Third, even in infected mothers, the chance of the child developing microcephaly is relatively low.[6] Finally, abortion is illegal in Brazil except in cases of rape or serious risk to the mother's life.

A similar potential issue would be selection into the sample based on different rates of diagnosis. For instance, families with higher attachment to the labor force might have access to better hospitals where microcephaly is more likely to be diagnosed. They would, therefore, be selected into the sample at higher rates, potentially biasing our results. We argue this type of selection is unlikely. Diagnosis of microcephaly depends on the measured head circumference of the neonate being below a specific threshold, according to sex- and gestational-age-specific growth charts (Ashwal et al., 2009). The test is very simple, leaving little room for discretion or differences in skill. Virtually all infants born in the country are measured and registered. There is therefore, minimal scope for selection of this type based on family characteristics.[7]

Finally, one potential concern is that children with microcephaly have higher rates of mortality. In our main results, we do not adjust for this difference, meaning our results may be partially driven by the effects of child mortality as opposed to permanent disability (though the sign of the bias introduced is ambiguous). Infant mortality among children with Zika-induced microcephaly is 8 to 10 times higher than the average in Brazil at the time, about 12%–14% in the period 2015–2016. Although this could bias our estimates in theory, in practice, the absolute rate is small enough not to have a significant impact on our estimates.

[6] Ximenes et al., 2023 find a risk from 2.6% to 4% of microcephaly for children of mothers infected with Zika.

[7] Another reason why this type of selection is unlikely in our particular sample is that families are drawn from similarly low income population. See note 5.

3 Data

We use three administrative datasets that cover all births in the country and all formal employment links. The first is the SINASC (*Sistema de Informações de Nascidos Vivos*), a dataset collected by the Ministry of Health detailing every live birth in the country. Second, RAIS (*Relação Anual de Informação Social*), is an administrative dataset used and made available by the Ministry of Labor, containing detailed information on employment links. Finally, we use the Single Registry (*Cadastro Único)*, an administrative dataset used to manage and coordinate various social programs, covering essentially all of Brazil's poor population. We link these datasets using location, time of birth and mother's date of birth.

3.1 Data on Births

To identify the children affected by the Zika epidemic who were born with microcephaly, we rely on a publicly available administrative record of all births in Brazil, SINASC. We have access to data on the birth location, the mother's municipality of residence, date of birth, mother's age, and the presence of microcephaly or other birth anomalies.

The SINASC dataset provides comprehensive coverage of all live births in Brazil, capturing a range of variables including birth weight, APGAR score, and the ICD-10 codes for congenital malformations. We are able to identify whether a child is diagnosed with microcephaly at birth by the microcephaly ICD-10 code. These data are high quality and coverage is close to 100% (Oliveira et al., 2015).

3.2 Data on the Labor Market

To observe mothers' and fathers' labor market outcomes, we use administrative data covering all formal employment links in Brazil. We are able to follow an individual's employment history and observe monthly earnings, hours, and the dates of any maternity leave.

The RAIS is a longitudinal dataset of social security records for employees and employers. It is collected by the Ministry of Labor in a compulsory survey of all firms and their registered workers, covering around 230,000 formally registered firms and 3.5 million workers annually. RAIS provides information on workers' demographics (age, gender, schooling, race), job characteristics (occupation, wage, hours worked), hiring and termination dates, and personal tax ID (CPF). It also includes information on many firm-level characteristics, notably the number of employees, municipality, firm tax id (CNPJ), and industry code.

3.3 Single Registry

To link the household members, we use the Single Registry (*Cadastro Único*) to observe families' characteristics and link different family members to formal employment data. The Single Registry is a federal registry used for several social programs to verify eligibility and track recipients over time. It started exclusively as Bolsa Família's administrative database but became the primary federal dataset on poverty. More than 20 social programs use it, covering virtually all of Brazil's poor (Campello & Neri, 2013). Single Registry aims to include all households with income per capita below one-half of the minimum wage (R$255 in 2010), much higher than the official poverty threshold (R$140 in 2010).

To be eligible for any government benefit that uses the Single Registry, families must have a valid registration (complete and up-to-date), updated at least every two years. They must undergo interviews with local government agents, including a standardized questionnaire on their earnings, living conditions, demographic and occupational characteristics, and personal tax ID (CPF). They have to inform authorities of relevant changes to family size or income.

We use the Single Registry to obtain data on the family structure and self-declared employment and income. We also use to link the RAIS and SINASC data.

3.4 Linking the Datasets

The absence of personal identifiers in the public birth dataset precludes direct linkage to RAIS or the Single Registry. We deal with this challenge using the mothers' date of birth, municipality of residence, and date of childbirth, available on Single Registry. After identifying the control and treated mothers in the Single Registry, we use their tax ID to accurately match them with RAIS records.

If we find a woman at least once in RAIS, we can re-construct her formal employment history. If we do not see her any year, then we know she has never worked in the formal sector. Our measure of employment is a dummy indicating if the woman appears in the RAIS dataset in that year with at least one job reporting a non-zero amount of hours per week. We also obtain average monthly wages and hours worked from RAIS.

Since we rely on the Single Registry to be able to link the datasets, our population of study is restricted to families in the Single Registry. This restriction means that our results should be understood to apply to lower-income individuals, a crucial consideration when interpreting the results. It also implies that there is relatively little heterogeneity

along income and socioeconomic status within the sample.

4 Empirical Strategy

For our main results, we compare the outcomes for families of children born with microcephaly to matched control families with children without this anomaly. We match families in relatively few variables: year and month of birth of the child, municipality of birth, age of the mother, and an indicator of the mother completing high school. Our key identification assumption is that, conditional on these variables, child microcephaly is as good as random. We select this set of variables based on using LASSO to predict the incidence of microcephaly within each municipality.

Our main assumption is that microcephaly is random conditional on a set of observables. We have a priori reasons to include some variables in the set on which we need to condition (e.g. geography, since the epidemic was focused on particular regions) and exclude others (mother's knowledge of health practices, as explained in the Background section). However, we join these theoretical reasons with a data driven approach to select the relevant controls from a rich set of covariates by using a LASSO procedure.

We picked the set of controls as follows. We started by selecting all municipalities where we identified at least one case of microcephaly. We then drew a sample of nine other children born in the same municipality for each child with microcephaly to constitute our full sample. We then used a Logit-LASSO flexible specification to predict microcephaly using socioeconomic information in the Single Registry. Finally, we inspected the predictive power of the selected variables in the logit regression. Appendix A shows the full set of potential variables, as well as the chosen subset and the resulting logit regression.[8]

[8]While income is arguably an important determinant of microcephaly in newborns during this period (Barbeito-Andrés et al., 2020; Lobkowicz et al., 2021; Souza et al., 2018), we do not include it as a

Informed by this procedure, our main strategy is based on matching each birth with microcephaly to controls with identical month, municipality, age, and education of the mother (completed high school or not). Because we use exact matching with fairly coarse variables, it is possible for one treated unit to be matched to several possible controls, as well as for multiple treated units to have identical characteristics. In this case, we call the units with identical matching characteristics a match group. For our main estimates, we give all treated units a weight of 1, and all control units a weight of $\frac{n_t(g)}{n_c(g)}$, where $n_t(g)$ denotes the number of treated units in the match-group, and $n_c(g)$ denotes the number of control units. Therefore, the total weight of the controls is identical to the total weight of the treated within each group, ensuring a balanced sample.

We estimate the treatment effect through the fully saturated model:

$$y_{ft} = \sum_{k \in (-18,\ldots,36)} \left[\beta_k^{Control} \cdot \mathbb{1}(t - \tau(f) = k) + \beta_k^{Treated} \cdot T_f \cdot \mathbb{1}(t - \tau(f) = k)\right] + \mu + \varepsilon_{ft} \tag{1}$$

where y is the outcome of interest for family f at year-month t. T_f is a dummy indicating families with a child with microcephaly. $\tau(f)$ is the date of birth of the child of family f, such that k is the time relative to birth. Thus $\beta_k^{Treated}$, captures the weighted average (using the weights described above) of the outcome at period k for mothers of children with microcephaly and $\beta_k^{Control}$ captures the average of the outcome for control mothers. μ is a constant and ε_{ft} is the random error, which we cluster at the match-group level.

matching variable. The first reason is that our sample is already restricted to families in the Single Registry, and therefore lower-income. The second is that our LASSO procedure did not select the wage income or total income variables. This indicates income not predictive of microcephaly *within this sample*, conditional on other selected covariates. Third, although income was not explicitly matched on, we show that the controls are identical to treated units in mothers' and fathers' earnings before childbirth.

When we present results we plot the coefficients $\beta_k^{Treated}$ and $\beta_k^{Control}$ plus the constant μ. We could have simply computed the weighted means of control and treated mothers without using a regression model and would have obtained exactly the same point estimates, we only use a regression model to obtain standard errors that take into account the temporal correlation of errors within individuals, which allows us to do proper inference.

Our identification assumption is that, conditional on having a child around the same time, in the same municipality, and the mother's age and educational level, the incidence of microcephaly is uncorrelated with unobserved characteristics that affect the outcomes of interest. As discussed in Section 2, the characteristics of the outbreak rule out several threats to identification, making it plausible that unobserved characteristics, such as mothers' behaviors, are not correlated to the chance of having a child with microcephaly. The absence of pre-trends also lends credibility to our identification strategy. As robustness tests, we show how less restrictive empirical models, for example, including individual-level, match-pair, and/or year fixed effects, lead to virtually the same results.

Selective fertility as a response to the outbreak could have important implications for our estimates. However, the delay with which the Zika virus infection causes microcephaly means that, in practice, this channel is unlikely to affect our results. Because the infection is most dangerous in the first months of pregnancy and has mild symptoms otherwise, it went practically undetected until after the first babies were diagnosed with microcephaly. Furthermore, any selective fertility response that followed the widespread recognition of the seriousness of the outbreak would only impact births with 9 months of delay, resulting in births in a period when cases were already far past the peak.[9]

[9]One exception is late-stage abortion, which could have a faster effect on births. Abortion is illegal in Brazil, except in cases of risk to the mother's life, pregnancy resulting from rape, or fetal anencephaly.

5 Results

In this section, we present our estimates of the effects of child disability in the family. We find an additional decrease in mothers' formal employment corresponding to 60% (CI: $[45.10\%, 75.05\%]$) of the motherhood penalty for controls, or about 26.39% (CI: $[20.78\%, 32.01\%]$) relative to the average of the last six months before childbirth, and similar effects on earnings. We find no effects on fathers' labor outcomes. Regarding the fertility response, parents of disabled children are less likely to have another child in the future. Parents of healthy children in areas with a higher prevalence of microcephaly cases also reduce their fertility compared to those in areas with a lower prevalence.

5.1 Balance and Summary

Table 1 shows summary stats for affected mothers and for controls. Overall, our control group seems to be similar to the treatment group along observable characteristics. We do not reject the hypothesis of equality between the samples for all variables at the usual significance levels, and no difference is economically significant. In particular, the racial composition of controls is the same as that of mothers of children with microcephaly, even though they were not explicitly matched on this characteristic.[10]

In our sample, the mean mother's age at first birth is 26.36 for mothers of children with microcephaly and 25.64 for control mothers. This is very similar to estimates of age at first birth for the country in general, suggesting no strong selection along this margin. In terms of its racial composition, our sample is considerably less white than average Brazilians (roughly 45%), reflecting the regions most affected. The large majority of the

[10]Most official government data in Brazil typically recognizes five racial categories: White or Caucasian, Black, Indigenous (referring to Amerindians), Yellow (which refers to people of East-Asian ethnicity) and *Pardo* (usually referring to mixed ethnic ancestry).

sample self-declared as *pardo*. Around 60% of the sample has at least some high school, with most of the others having at least middle school. Overall, the differences between the control and treated characteristics are minimal and not statistically significant.

5.2 Formal Employment and Earnings

We find that after the birth of a child with microcephaly, mothers' formal employment falls by an additional 3.2 percentage points (a 15% decline), on top of the 5.1 percentage points associated with childbirth in controls (a 27% decline). The impact on earnings follows a similar path. When we restrict the sample to mothers with previous work experience in the formal sector, we find similar patterns relative to the share of employed mothers at baseline. We do not find any impact on fathers' employment or earnings.

Figure 4 shows the average labor force participation of mothers around the time of childbirth for the treated and control groups. Even though this variable is not used for matching and there the estimates are not covariate-adjusted, we see virtually identical rates of employment month-by-month before childbirth, with, if anything, a very slight difference in favor of the affected mothers. After month 6, corresponding to the end of typical maternity leave, we see that mothers of children with microcephaly see a a drop in employment that is approximately 50% larger than that of the controls, This difference persists over time. After 36 months, we see a difference of about 6 p.p., with only about 2% of mothers of children with microcephaly formally employed. The effects on formal earnings mirror closely those of employment. Note that the peak in earnings at about 4 months after childbirth likely corresponds to extra payments relative to job termination (e.g. vacations due).

Figure 5 shows the results focusing on the sample of mothers who had previous experience in the formal labor market. This difference in experience may make these

mothers more attached to the labor force and may indicate higher human capital, which could help deal with the health shock. We find that formal employment at the time of childbirth is more than double the sample average. However, we see a very similar pattern in both employment and earnings. By the end of our sample window, employment for control mothers is 23%, compared to only 5% for mothers of children with microcephaly.

Table 2 presents the results from the DID specification. The estimates for the Treated coefficient confirm the result that the pre-existing differences are small in magnitude and not statistically significant. Further, we can directly compare the average effect of microcephaly after childbirth with the raw motherhood penalty in each specification. We find that the additional penalty corresponds to about 60% of the motherhood penalty, both in employment and earnings for the full sample, and about 40% for the sample with previous work experience.

We repeat the same analysis for fathers, finding no effects on formal employment or earnings. Figure 6 shows the results. Note that we maintain the same match-groups as in the previous analysis, matched by mother characteristics. Therefore, fathers do not necessarily have the same level of education and age by construction as mothers do. Nevertheless, we find employment and earnings are remarkably similar, both in levels and in trends before childbirth. Table 3 shows the results of the DID specification. The estimates indicate a null effect on employment and a positive but not statistically significant effect on earnings. Notably, employment and earnings tend to increase after childbirth, although this effect is also not significant after accounting for match-group fixed effects. This suggests possible specialization in the household, with negative effects on labor market participation for women and positive for men. However, since we only observe the labor market outcomes of cohabiting fathers, a strict causal interpretation of the parameter requires strong assumptions.

We conduct a series of robustness tests. Figure A1 shows the results for employment and earnings controlling for different levels of fixed effects: year, match-group, individual, and individual plus year. Overall, the results are essentially unchanged, although the inclusion of year fixed effects increases the standard errors substantially.

5.3 Informal and Formal Employment and Earnings

In Section 5.2, we showed the effect of having a child with microcephaly on mothers' formal employment and earnings. Using data from the Single Registry, we complement the analysis by looking at those outcomes including the informal sector. While this data is less frequent and self-reported, it allows us to have a complete picture of the effects on labor market outcomes.

Figure 7 shows the effect of having a child with microcephaly on mothers' employment and earnings, including informal earnings. We find a reduction in employment after 1 year of about 5pp, which is a similar magnitude to the effect we had found when considering only formal employment. We also see a reduction in earnings of BRL48 three years after birth, which is smaller than the reduction we found in formal earnings after three years (BRL 103) but still significant. This indicates that the reduction in earnings and employment found in formal sectors is only partially offset by increases in informal earnings.

5.4 Fertility

One potential response to the demands of caring for a disabled child is that families may choose to avoid having more children, depressing subsequent fertility. Not only is this an important effect on its own right, it also informs the interpretation of the effects we

found on the labor market. Since fertility tends to depress labor market participation, this causal channel will tend to make differences in participation smaller. We show that child microcephaly seems to have only a very small and mostly not statistically significant impact on future fertility compared to paired controls.

Our measure of subsequent fertility comes from the Single Registry in 2019. Therefore, the affected child will be between 4 and 2 when the data is collected. We find the same family and the same mother and count the number of children born after the child with microcephaly or their matched control. In about half the cases, the reference child was the firstborn, and overall fertility over this interval is low over this time span, making detection of any possible effects challenging.

We estimate regressions of the form:

$$fertility_i = \beta \cdot T_i + \alpha_{p(f)} + u_i \tag{2}$$

where $fertility_i$ indicates the number of additional children by mother i. T_i is a dummy indicating whether mother i had a child microcephaly. We control for pair fixed-effect, $\alpha_{p(f)}$ to ensure we are comparing each treated family with the most comparable control families.

Table 5 shows that accounting for the fixed effects, mothers with a child with microcephaly had 0.005 fewer children until 2019 compared to controls. If we restrict the sample to families with only one child at the initial period, the effect on fertility is 0.022 (p-value: 9.3%), as shown in Column (3). There is no effect on fertility for families that already had more than one child, (Column (4)). This is to be expected, since fertility above 2 children is relatively uncommon, so there is not the possibility of further reducing it much more.

The modest impact on fertility may also be attributed to a decline in birth rates within the control group. Following the confirmation of the link between the Zika virus and microcephaly, many women postponed childbearing until the situation was under control (Junior & Rasul, 2019). We examine whether areas with a higher incidence of microcephaly cases experienced a more pronounced decrease in fertility rates. Given that some regions were more severely affected than others, women in these areas faced a higher likelihood of exposure to the virus and, consequently, an increased risk of having a child with microcephaly if they became pregnant. It is crucial to note that this primarily affected women who were not pregnant when the connection between the Zika virus and microcephaly was established. As a result, we anticipate observing changes in fertility rates approximately one year after the onset of the outbreak.

5.5 Spillover Effects on Fertility

Following the news establishing the link between the Zika virus outbreak and microcephaly, there were reports of women afraid of conceiving new children. This reaction may be reflected in local spillovers to fertility because people infer the risk is higher in an area where cases of microcephaly were reported, or because proximity raises the salience of the risk. In this section we investigate whether there were significant local negative spillovers to subsequent fertility in mothers not directly affected.

To estimate the spillover effects on fertility, we compare the fertility rate in municipalities with a higher incidence of microcephaly to places with a lower or no incidence. In this analysis, we include every municipality in the country (as opposed to only municipalities with at least one case). We identify the spillover effects by estimating the following equation:

$$fertility_{mt} = \sum_{t \in 2010,..,2020, t \neq 2014} \gamma_t \cdot Incidence_m \cdot \mathbf{1}_{t \geq 2015} + \delta_m + \delta_t + \epsilon_{it}$$

where $fertility_{mt}$ is the total number of babies born in a municipality m at year t per 1,000 inhabitants. $Incidence_m$ is a measure of the incidence of microcephaly cases during the Zika virus outbreak period in municipality m. We use two different measures: (A) a dummy indicating whether the municipality is above the 75th percentile of the distribution of cases per capita and (B) total number of cases per 1,000 inhabitants. $\mathbf{1}_{t \geq 2015}$ is an indicator function for years equal to or after 2015. We control for the municipality and year fixed effect, δ_m and δ_t, respectively. Standard errors are clustered at the municipality level.

Our parameters of interest are γ_t, which captures of effect on fertility in each year t. Here, we must rely on a parallel trends assumption instead of conditional independence. This is because municipality-level incidence of the virus was determined by several factors that are correlated with the level of fertility, such as local climate. Therefore, our identification assumption is that fertility would have followed parallel paths in municipalities relatively highly affected relative to municipalities relatively unaffected.

Our analysis reveals a decline in overall fertility rates in municipalities with a higher incidence of microcephaly cases following the confirmation of the link between the Zika virus and this condition. Figure 10 illustrates the results using two different measures of incidence. Panel A compares areas with high versus low/no incidence of microcephaly cases per birth. Prior to the outbreak (2010-2014), fertility rates were similar across all areas. In 2015, the first year of the outbreak and when the link between Zika and microcephaly was confirmed, we observe no significant changes in fertility rates, as expected. However, in 2016, we note a decrease of 0.3 births per thousand inhabitants

in highly affected areas. This represents a 1.92% reduction from the average fertility rate (15.61) or 9.35% of one standard deviation in the fertility rate (3.2). This effect persists through 2019.[11]

Panel B presents an alternative analysis using the number of microcephaly cases per thousand births as the incidence measure. Again, we find no evidence of differential fertility in the years preceding the outbreak or in its first year. In 2016, we observe that each additional case of microcephaly per 1,000 births corresponds to a reduction of 2 births per 1,000 inhabitants. In other words, an increase of one standard deviation in the share of microcephaly cases per 1,000 births (0.38) leads to a 24% decrease of one standard deviation in the fertility rate. Unlike the results in Panel A, this effect does not persist over time for this measure. Table 6 summarizes the results aggregating the coefficients for before (2010-2014) and after (2015-2019) the outbreak.

5.6 Family Structure

Child disability creates severe stress in the household, and one of the possible medium-term effects is divorce or separation of the parents. Following the Zika epidemic, there were several news stories about divorce in households where in families with a child with microcephaly, providing anecdotal evidence that this may be an important dimension.

To test this hypothesis, we try to identify the child's father in the Single Registry with the same family in 2017 and 2019. We estimate the Equation 2, with the outcome variable being an indicator of the presence of the father in the household. Overall rates of cohabitation in the population in the Single Registry are extremely low, on the order of 15% to 20%.

[11] Our analysis is limited to 2019 due to the onset of the COVID-19 pandemic.

Table 7 presents the results. We find that, if anything, there is a slightly higher chance of the father being present in families with a child with a disability, although the difference is small in magnitude and not significant. The estimates are similar for 2017 and 2019. In column (5), we attempt to see the effect in 2019 conditional on presence in 2017. We find that the father being present in a year is a strong predictor of being present afterward, and adding this control renders the estimate of the effect of microcephaly negative and not significant.

5.7 Social Assistance Benefits

The interpretation of our results depends crucially on the provision of social security payments for families with disabled children. We show suggestive evidence that the negative effects on female labor force participation are partially driven by income effects from receiving social assistance payments, but effects are still strong and significant for non-recipients.

The role of social assistance is a first-order concern, since Brazil has a relatively well developed social safety net. In particular, low-income households with a disabled member can be entitled to the *Benefício de Prestação Continuada* (BPC), which pays the equivalent of one minimum wage.[12] This fact raises the question of how much our estimates are driven by these payments. If results are strongly reflecting income effects, the external validity of the findings may be limited.

To analyze this question, we identify Social Assistance recipients in publicly available data from the Brazilian social security institution (INSS). It is not possible to identify

[12] To be eligible, the household income must be less than one quarter of the minimum wage per household member. The recipient must also prove their physical or mental disability precludes normal independent participation in society, including labor. This benefit is also available for people over 65 in low-income households.

precisely when recipients started receiving the benefit, so we divide our sample into recipients and non-recipients. One quarter of affected families in our sample are recipients of the BPC. Interpreting the results causally would require strong assumptions. Since we cannot infer eligibility ex-ante, the estimates may suffer from collider bias.

Table 8 shows the results for the full sample. Among non-recipients, we find no significant difference between affected mothers and controls in formal employment before childbirth (p-value: 0.575) although the affected group is slightly below the control (average difference from $t = -6$ to $t = -1$: -1.11 p.p.; CI: $[-3.00, 0.78]$). The groups start to diverge six months after childbirth. We find microcephaly increases the size of the motherhood penalty by an average of 2.60 percentage points (CI: $[1.52, 3.69]$).

Meanwhile, the recipient sample shows apparent imbalance before childbirth. Although the joint test of no difference during the pre-birth period is not significant (p-value: 9.4%), the absolute difference is sizeable and the average of the coefficients is significantly different from zero (4.49 p.p., CI: $[1.52, 7.47]$). This potential imbalance may reflect positive selection into receiving the BPC. However, six months after childbirth the employment of affected mothers falls to very close to zero. The average effect is -5.15 p.p. (CI: $[-6.27, -4.03]$), increasing over time to reach -8.64 (CI: $[-10.32, -6.96]$).

Figure 9 shows the results when conditioning on having some formal employment experience before child birth. The patterns for non-recipients look very similar, although starting at a much higher level of participation. The average effect is -9.83 p.p. (CI: $[-14.68, -.4.98]$). For BPC recipients, conditioning makes the differences before childbirth relatively smaller and non-significant. The affected "mothers similarly exit almost entirely the formal labor force (average effect: -12.61 p.p., CI: $[15.37, -9.86]$). Similarly to the full sample, the estimated effects are substantially larger for recipients than it is for non-recipients.

Overall, these results suggest that recipients overwhelmingly leave the formal labor force, but there are significant reductions even for non-recipients. However, it is important to stress that these results are suggestive due to the potential for collider bias.

6 Conclusion

In this paper, we analyze how congenital microcephaly in a child affects the labor outcomes of the parents, subsequent fertility, and family structure. We show that mothers' labor market participation drops by nearly 50%, an effect that does not seem to fade over time. On the other hand, fathers' labor outcomes are not affected. We also find suggestive evidence that affected families have lower subsequent fertility and fathers are not more likely to divorce or leave the family.

We conduct our analysis in the context of the Zika virus epidemic. Unique features of the outbreak allow us to rule out or minimize several concerns, such as endogeneity of maternal care and health behaviors and selective abortion or mortality. Our paper contributes to the literature on the Zika virus by highlighting its impact on families' labor market outcomes.

Overall, our results help quantify the enormous human costs associated with disease and disability, and highlight the disproportionate effect on women. A better understanding of the ways individuals and families deal with persistent health shocks and disabilities can be an important input in the design of public policy to address these issues.

References

Adhvaryu, A., Daysal, N. M., Gunnsteinsson, S., Molina, T., & Steingrimsdottir, H. (2022). Impact of child health on families: Evidence from childhood cancers. *5th IZA Workshop on Gender and Family Economics: Families as an Insurance Mechanism*.

Ashwal, S., Michelson, D., Plawner, L., & Dobyns, W. B. (2009). Practice parameter: Evaluation of the child with microcephaly (an evidence-based review): Report of the quality standards subcommittee of the american academy of neurology and the practice committee of the child neurology society. *Neurology*, *73*(11), 887.

Barbeito-Andrés, J., Pezzuto, P., Higa, L., Dias, A. A., Vasconcelos, J., Santos, T., Ferreira, J., Ferreira, R., Dutra, F., Rossi, A., et al. (2020). Congenital zika syndrome is associated with maternal protein malnutrition. *Science Advances*, *6*(2), eaaw6284.

Berniell, I., Berniell, L., De la Mata, D., Edo, M., & Marchionni, M. (2021). Gender gaps in labor informality: The motherhood effect. *Journal of Development Economics*, *150*, 102599.

Breivik, A.-L., & Costa-Ramón, A. (2022). The career costs of children's health shocks. *University of Zurich, Department of Economics, Working Paper*, (399).

Budig, M. J., & England, P. (2001). The wage penalty for motherhood. *American Sociological Review*, *66*(2), 204–225.

Burton, P., Chen, K., Lethbridge, L., & Phipps, S. (2017). Child health and parental paid work. *Review of Economics of the Household*, *15*, 597–620.

Campello, T., & Neri, M. C. (2013). Programa bolsa família: Uma década de inclusão e cidadania.

Cauchemez, S., Besnard, M., Bompard, P., Dub, T., Guillemette-Artur, P., Eyrolle-Guignot, D., Salje, H., Van Kerkhove, M. D., Abadie, V., Garel, C., et al. (2016). Association

between zika virus and microcephaly in french polynesia, 2013–15: A retrospective study. *The Lancet*, *387*(10033), 2125–2132.

Chen, K.-M., Lin, M.-J., & Lo, W.-L. (2023). Impacts of childhood disability on family: Labor, marriage, fertility, and depression. *Age*, *1*, 90.

Chervenak, F. A., Jeanty, P., Cantraine, F., Chitkara, U., Venus, I., Berkowitz, R. L., & Hobbins, J. C. (1984). The diagnosis of fetal microcephaly. *American Journal of Obstetrics and Gynecology*, *149*(5), 512–517.

Cheung, T. T., Kan, K., & Yang, T. (2023). Gender difference in parental responses to child disability: Empirical evidence and mechanisms.

Cortés, P., & Pan, J. (2023). Children and the remaining gender gaps in the labor market. *Journal of Economic Literature*, *61*(4), 1359–1409.

De Quinto, A., Hospido, L., & Sanz, C. (2020). The child penalty in spain. *Banco de España Occasional Paper*, (2017).

Delmelle, E., Hagenlocher, M., Kienberger, S., & Casas, I. (2016). A spatial model of socioeconomic and environmental determinants of dengue fever in Cali, Colombia. *Acta Tropica*, *164*, 169–176.

Eriksen, T. L. M., Gaulke, A., Skipper, N., & Svensson, J. (2021). The impact of childhood health shocks on parental labor supply. *Journal of Health Economics*, *78*, 102486.

Frijters, P., Johnston, D. W., Shah, M., & Shields, M. A. (2009). To work or not to work? Child development and maternal labor supply. *American Economic Journal: Applied Economics*, *1*(3), 97–110.

Gunnsteinsson, S., & Steingrimsdottir, H. (2019). *The long-term impact of children's disabilities on families* (tech. rep.). Working paper.

Haby, M. M., Pinart, M., Elias, V., & Reveiz, L. (2018). Prevalence of asymptomatic zika virus infection: A systematic review. *Bulletin of the World Health Organization*, *96*(6), 402.

Johansson, M. A., Mier-y-Teran-Romero, L., Reefhuis, J., Gilboa, S. M., & Hills, S. L. (2016). Zika and the risk of microcephaly. *New England Journal of Medicine*, *375*(1), 1–4.

Junior, I. L., & Rasul, I. (2019). *The anatomy of a public health crisis: Household and health sector responses to the zika epidemic in Brazil*.

Khan, M. B., Yang, Z.-S., Lin, C.-Y., Hsu, M.-C., Urbina, A. N., Assavalapsakul, W., Wang, W.-H., Chen, Y.-H., & Wang, S.-F. (2023). Dengue overview: An updated systemic review. *Journal of infection and public health*.

Kleven, H., Landais, C., & Søgaard, J. E. (2019). Children and gender inequality: Evidence from denmark. *American Economic Journal: Applied Economics*, *11*(4), 181–209.

Kraemer, M. U., Sinka, M. E., Duda, K. A., Mylne, A. Q., Shearer, F. M., Barker, C. M., Moore, C. G., Carvalho, R. G., Coelho, G. E., Van Bortel, W., et al. (2015). The global distribution of the arbovirus vectors Aedes aegypti and Ae. albopictus. *elife*, *4*, e08347.

Lafférs, L., & Schmidpeter, B. (2021). Early child development and parents' labor supply. *Journal of Applied Econometrics*, *36*(2), 190–208.

Leibovitz, Z., & Lerman-Sagie, T. (2018). Diagnostic approach to fetal microcephaly. *European Journal of Paediatric Neurology*, *22*(6), 935–943.

Lobkowicz, L., Power, G. M., De Souza, W. V., Montarroyos, U. R., Martelli, C. M. T., de Araùjo, T. V. B., Bezerra, L. C. A., Dhalia, R., Marques, E. T., de Barros Miranda-Filho, D., et al. (2021). Neighbourhood-level income and Zika virus infection during pregnancy in Recife, Pernambuco, Brazil: An ecological perspective, 2015–2017. *BMJ Global Health*, *6*(12), e006811.

Lowe, R., Bailey, T. C., Stephenson, D. B., Graham, R. J., Coelho, C. A., Carvalho, M. S., & Barcellos, C. (2011). Spatio-temporal modelling of climate-sensitive disease risk: Towards an early warning system for dengue in Brazil. *Computers & Geosciences*, *37*(3), 371–381.

Martínez, C., Perticará, M., & Smith, R. (2023). *Maternal child penalties and children with disabilities: Preliminary findings* (tech. rep.). Inter-American Development Bank Discussion Paper.

Murray, N. E. A., Quam, M. B., & Wilder-Smith, A. (2013). Epidemiology of dengue: Past, present and future prospects. *Clinical epidemiology*, 299–309.

Musick, K., Bea, M. D., & Gonalons-Pons, P. (2020). His and her earnings following parenthood in the United States, Germany, and the United Kingdom. *American Sociological Review*, *85*(4), 639–674.

Oliveira, M. M. d., Andrade, S. S. C. d. A., Dimech, G. S., Oliveira, J. C. G. d., Malta, D. C., Rabello Neto, D. d. L., & Moura, L. d. (2015). Evaluation of the national information system on live births in Brazil, 2006-2010. *Epidemiologia e Servicos de Saúde*, *24*, 629–640.

Pedrosa, C. d. S., Souza, L. R., Gomes, T. A., de Lima, C. V., Ledur, P. F., Karmirian, K., Barbeito-Andres, J., Costa, M. d. N., Higa, L. M., Rossi, Á. D., et al. (2020). The cyanobacterial saxitoxin exacerbates neural cell death and brain malformations induced by zika virus. *PLoS Neglected Tropical Diseases*, *14*(3), e0008060.

Powers, E. T. (2001). New estimates of the impact of child disability on maternal employment. *American Economic Review*, *91*(2), 135–139.

Powers, E. T. (2003). Children's health and maternal work activity estimates under alternative disability definitions. *Journal of Human Resources*, *38*(3), 522–556.

Salkever, D. S. (1982). Children's health problems: Implications for parental labor supply and earnings. *Economic Aspects of Health* (pp. 221–252). University of Chicago Press.

Sieppi, A., & Pehkonen, J. (2019). Parenthood and gender inequality: Population-based evidence on the child penalty in Finland. *Economics Letters*, *182*, 5–9.

Siqueira-Junior, J. B., Maciel, I. J., Barcellos, C., Souza, W. V., Carvalho, M. S., Nascimento, N. E., Oliveira, R. M., Morais-Neto, O., & Martelli, C. M. (2008). Spatial

point analysis based on dengue surveys at household level in central Brazil. *BMC Public Health*, *8*, 1–9.

Souza, W. V. d., Albuquerque, M. d. F. P. M. d., Vazquez, E., Bezerra, L. C. A., Mendes, A. d. C. G., Lyra, T. M., Araujo, T. V. B. d., Oliveira, A. L. S. d., Braga, M. C., Ximenes, R. A. d. A., et al. (2018). Microcephaly epidemic related to the Zika virus and living conditions in Recife, Northeast Brazil. *BMC public health*, *18*, 1–7.

Teurlai, M., Menkès, C. E., Cavarero, V., Degallier, N., Descloux, E., Grangeon, J.-P., Guillaumot, L., Libourel, T., Lucio, P. S., Mathieu-Daudé, F., et al. (2015). Socio-economic and climate factors associated with dengue fever spatial heterogeneity: A worked example in New Caledonia. *PLoS neglected tropical diseases*, *9*(12), e0004211.

Vaalavuo, M., Salokangas, H., & Tahvonen, O. (2023). Gender inequality reinforced: The impact of a child's health shock on parents' labor market trajectories. *Demography*, *60*(4), 1005–1029.

Wasi, N., van den Berg, B., & Buchmueller, T. C. (2012). Heterogeneous effects of child disability on maternal labor supply: Evidence from the 2000 US Census. *Labour Economics*, *19*(1), 139–154.

Whiteman, A., Loaiza, J. R., Yee, D. A., Poh, K. C., Watkins, A. S., Lucas, K. J., Rapp, T. J., Kline, L., Ahmed, A., Chen, S., et al. (2020). Do socioeconomic factors drive Aedes mosquito vectors and their arboviral diseases? A systematic review of dengue, chikungunya, yellow fever, and Zika Virus. *One Health*, *11*, 100188.

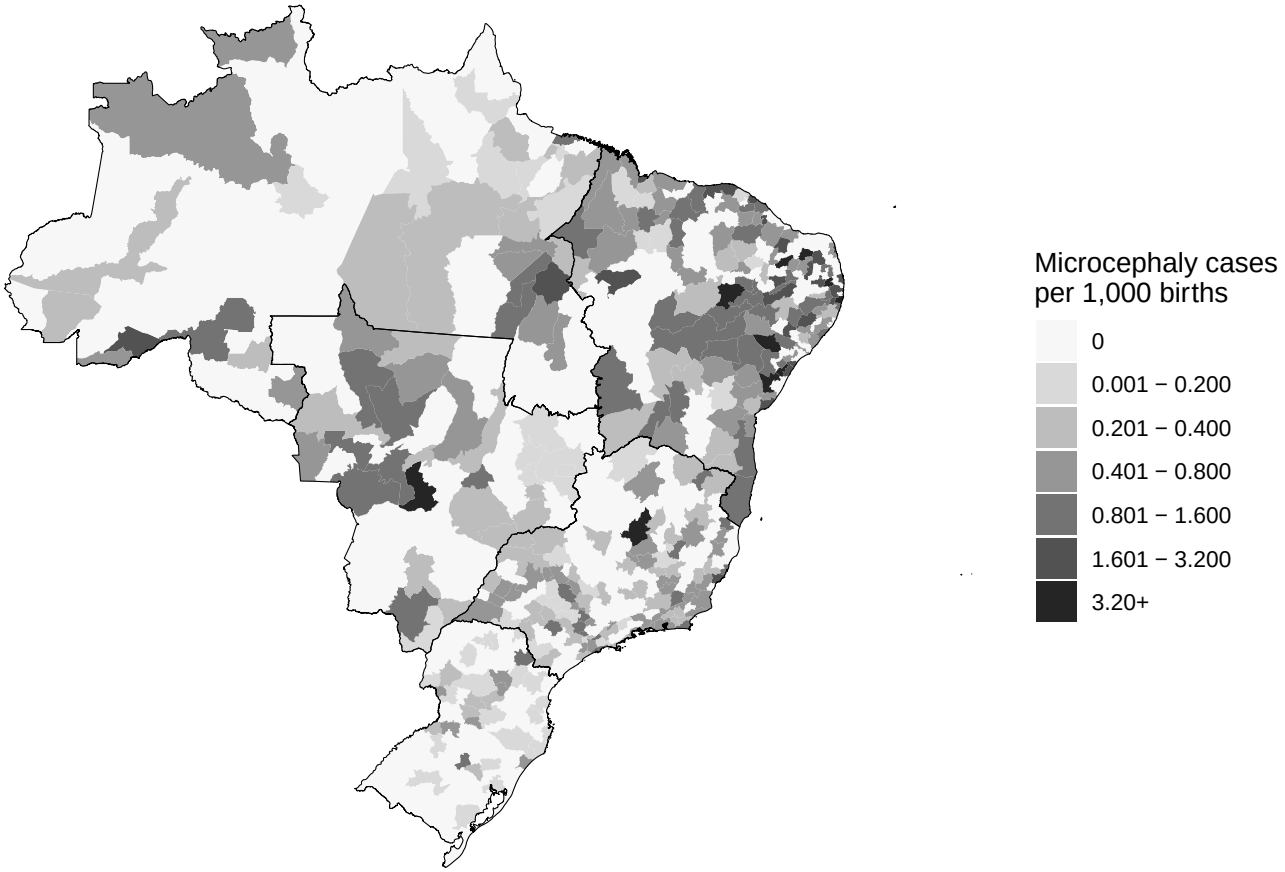
Wolfe, B. L., & Hill, S. C. (1995). The effect of health on the work effort of single mothers. *Journal of Human Resources*, 42–62.

Wu, P.-C., Lay, J.-G., Guo, H.-R., Lin, C.-Y., Lung, S.-C., & Su, H.-J. (2009). Higher temperature and urbanization affect the spatial patterns of dengue fever transmission in subtropical Taiwan. *Science of the total Environment*, *407*(7), 2224–2233.

Ximenes, R. A. d. A., de Barros Miranda-Filho, D., Brickley, E. B., de Araújo, T. V. B., Montarroyos, U. R., Abtibol-Bernardino, M. R., Mussi-Pinhata, M. M., Duarte, G., Coutinho, C. M., de Moura Negrini, S. F. B., et al. (2023). Risk of adverse outcomes in offspring with RT-PCR confirmed prenatal zika virus exposure: An individual participant data meta-analysis of 13 cohorts in the zika brazilian cohorts consortium. *The Lancet Regional Health–Americas*, *17*.

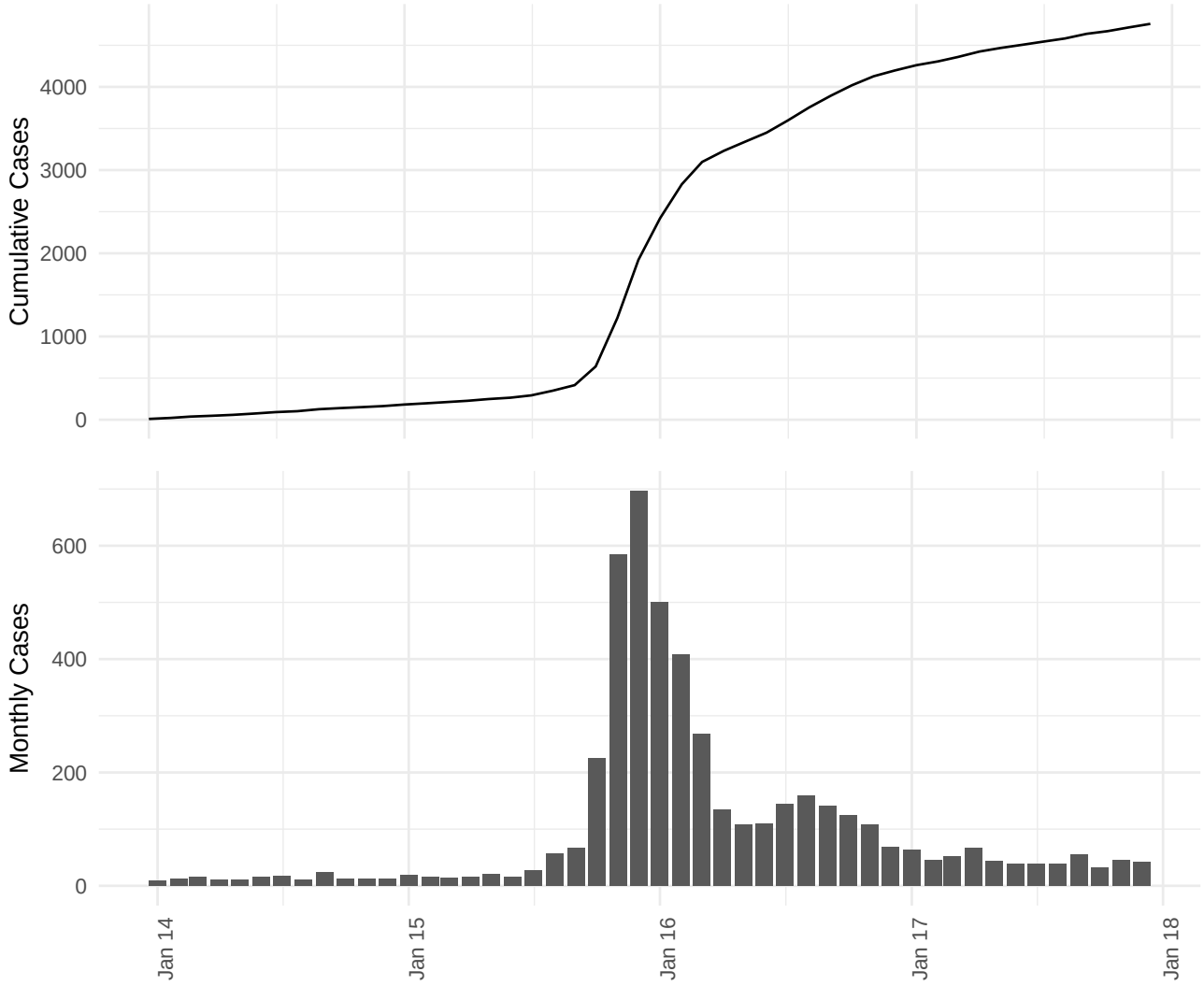
Figures

Figure 1: Geographic Variation on the Number of Microcephaly cases per 1000 Births



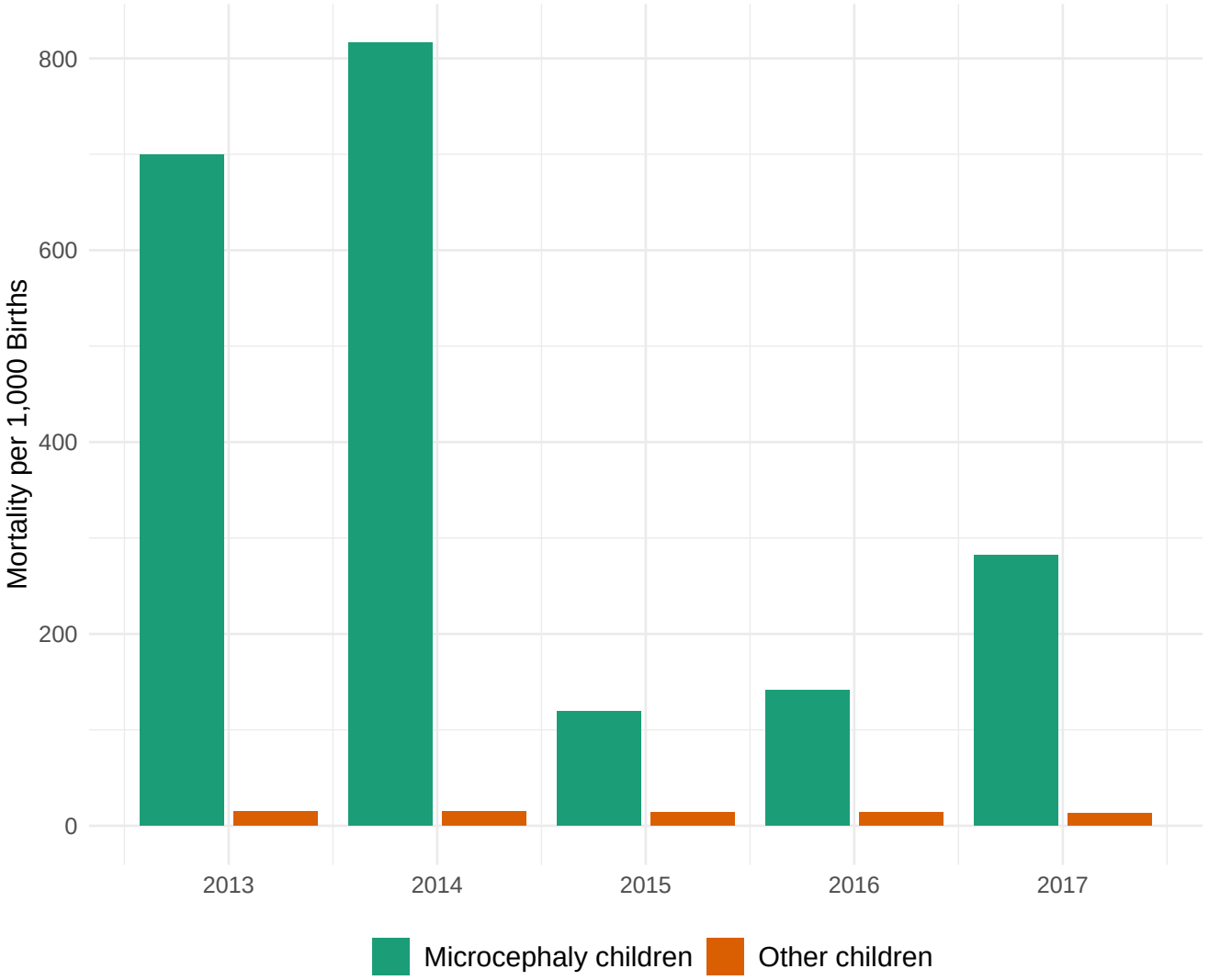
Notes: This figure illustrates the geographic variation on the number of microcephaly cases per thousand births in 2015 and 2016. Each polygon is a micro-region, comprising on average about 10 municipalities. Micro-regions with zero births in the period are assigned to the zero cases per 1,000 births category. The total number of births and cases of microcephaly is available from SINASC/SUS.

Figure 2: Microcephaly Cases by Month



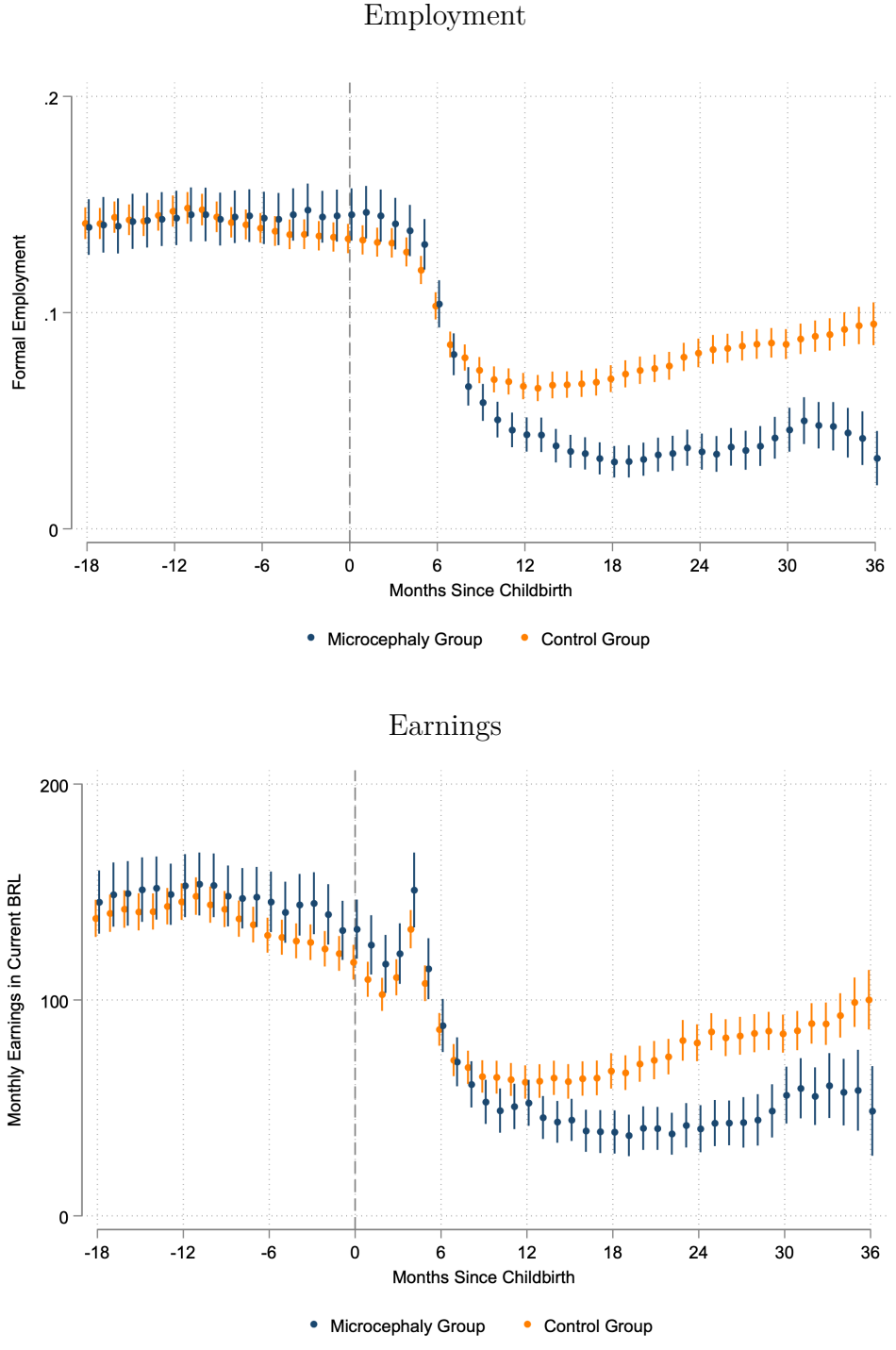
Notes: These figures show the evolution in the total number of cases of microcephaly, over the Northeast and Southwest regions. The top graph shows cumulative cases, while the bottom shows monthly incidence. The data is from SINASC/SUS.

Figure 3: Mortality Rates of Children with Microcephaly



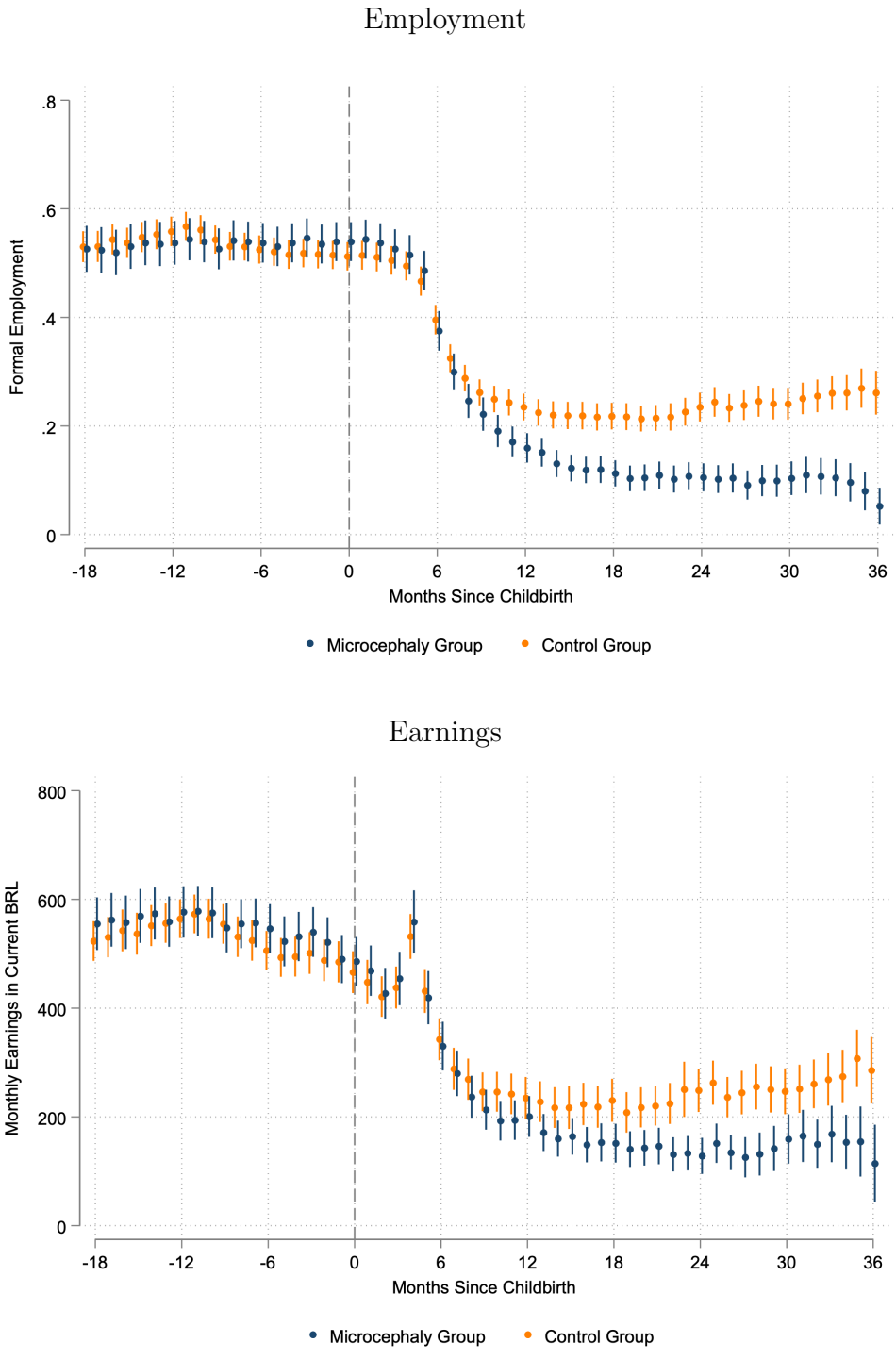
Notes: This figure shows mortality by age 5 per thousand births, separately for children born with microcephaly compared to others. The year indicates year of birth, not death. The total number of births and cases of microcephaly are made available by SINASC/SUS. Microcephaly is identified by the ICD-10 code Q02. Infant mortality is made available by SIM/SUS.

Figure 4: Mothers of Children Affected by Microcephaly and Matched Controls



Notes: This figure shows the employment rate (above) and earnings (below) of mothers in the formal sector. The Microcephaly Group consists mothers of children diagnosed with microcephaly, while the Control Group consists of mothers of children without this condition, matched on location, age and time of childbirth. Earnings are in BRL, and the error bars represent 95% confidence intervals, with errors clustered at the match-group level.

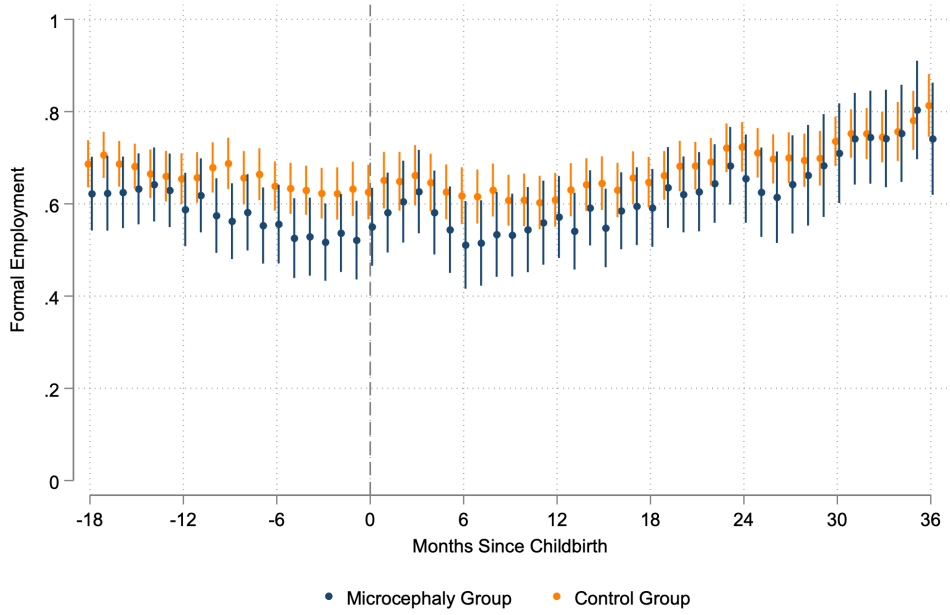
Figure 5: Subsample with Previous Formal Employment



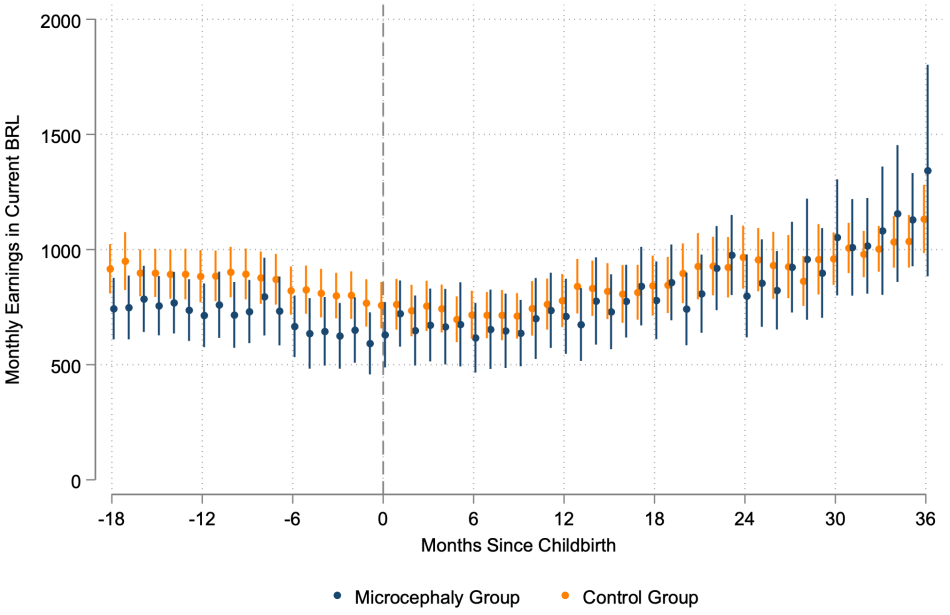
Notes: This figure shows the employment rate (above) and earnings (below) of mothers in the formal sector. This subsample is selected such that every mother had at worked for at least one month in the private sector in the two years before childbirth. The Microcephaly Group consists mothers of children diagnosed with microcephaly, while the Control Group consists of mothers of children without this condition, matched on location, age and time of childbirth. Earnings are in BRL, and the error bars represent 95% confidence intervals, with errors clustered at the match-group level.

Figure 6: Effects on Fathers

Employment

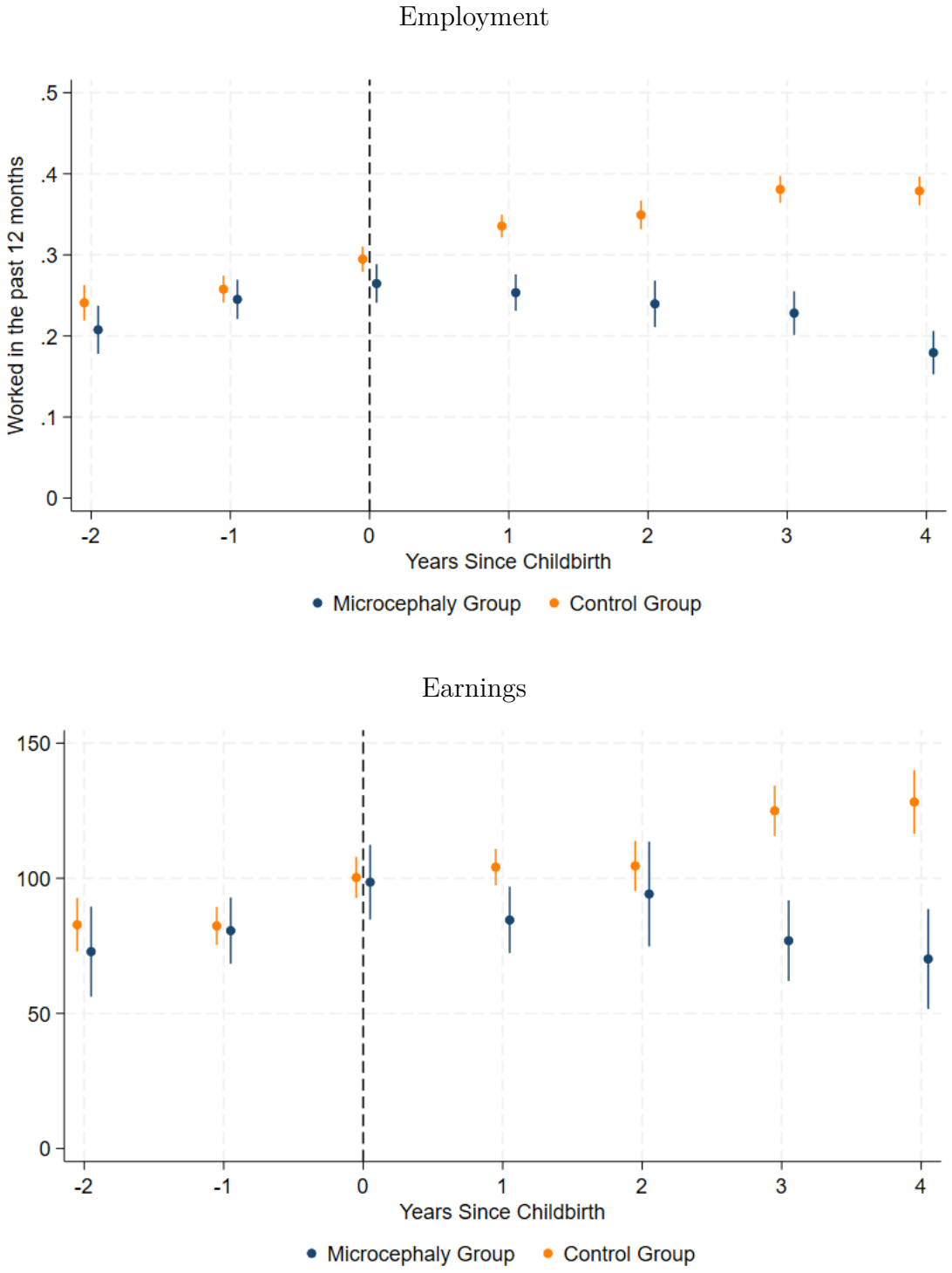


Earnings



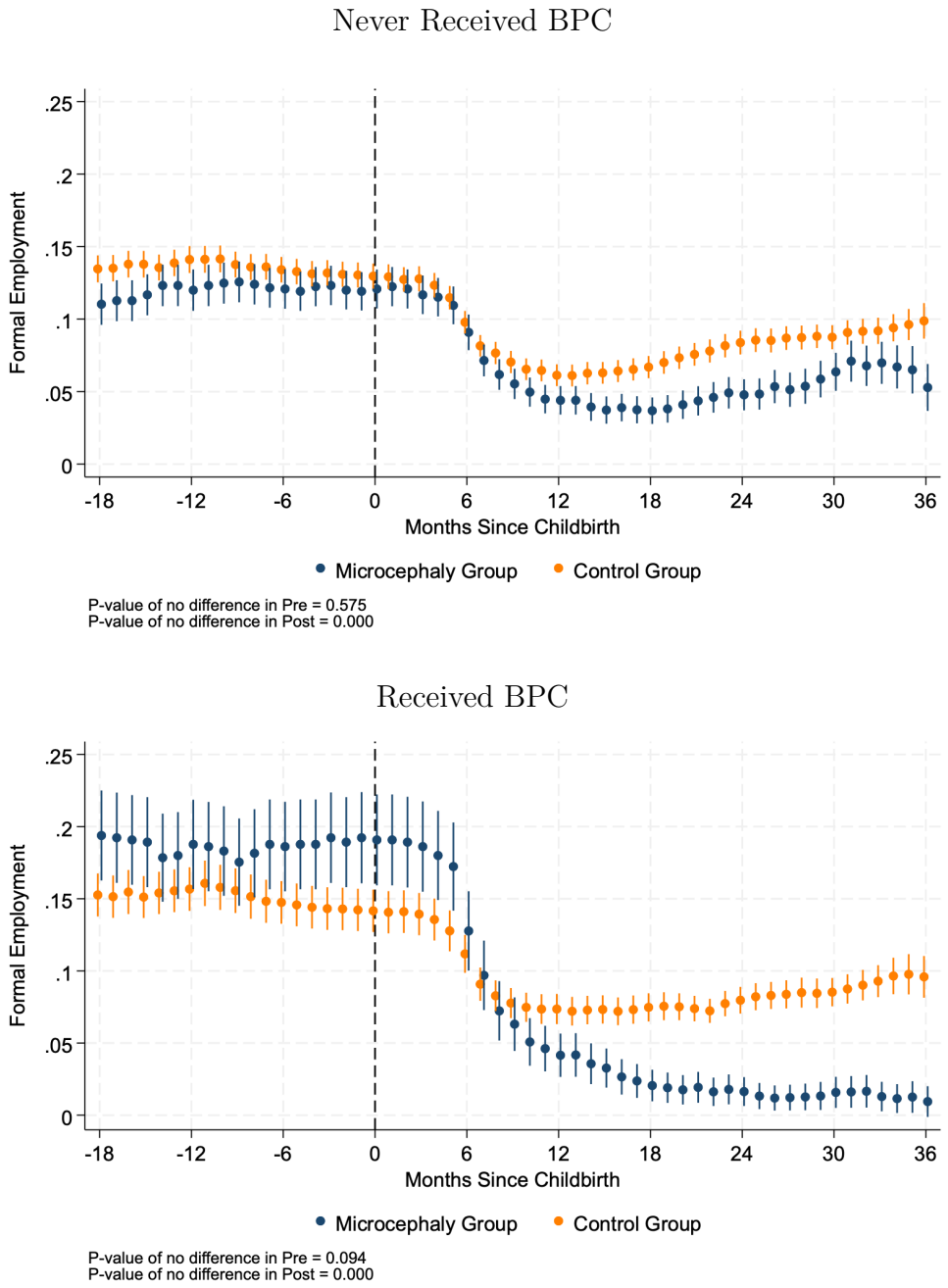
Notes: This figure shows the employment rate (above) and earnings (below) of fathers in the formal sector. The Microcephaly Group consists fathers of children diagnosed with microcephaly. The Control Group consists of fathers of children without this condition, matched on location, age, time of childbirth, and mother's age. Earnings are in BRL, and the error bars represent 95% confidence intervals, with errors clustered at the match-group level.

Figure 7: Effects on Mothers' Labor Market Outcomes – Formal and Informal



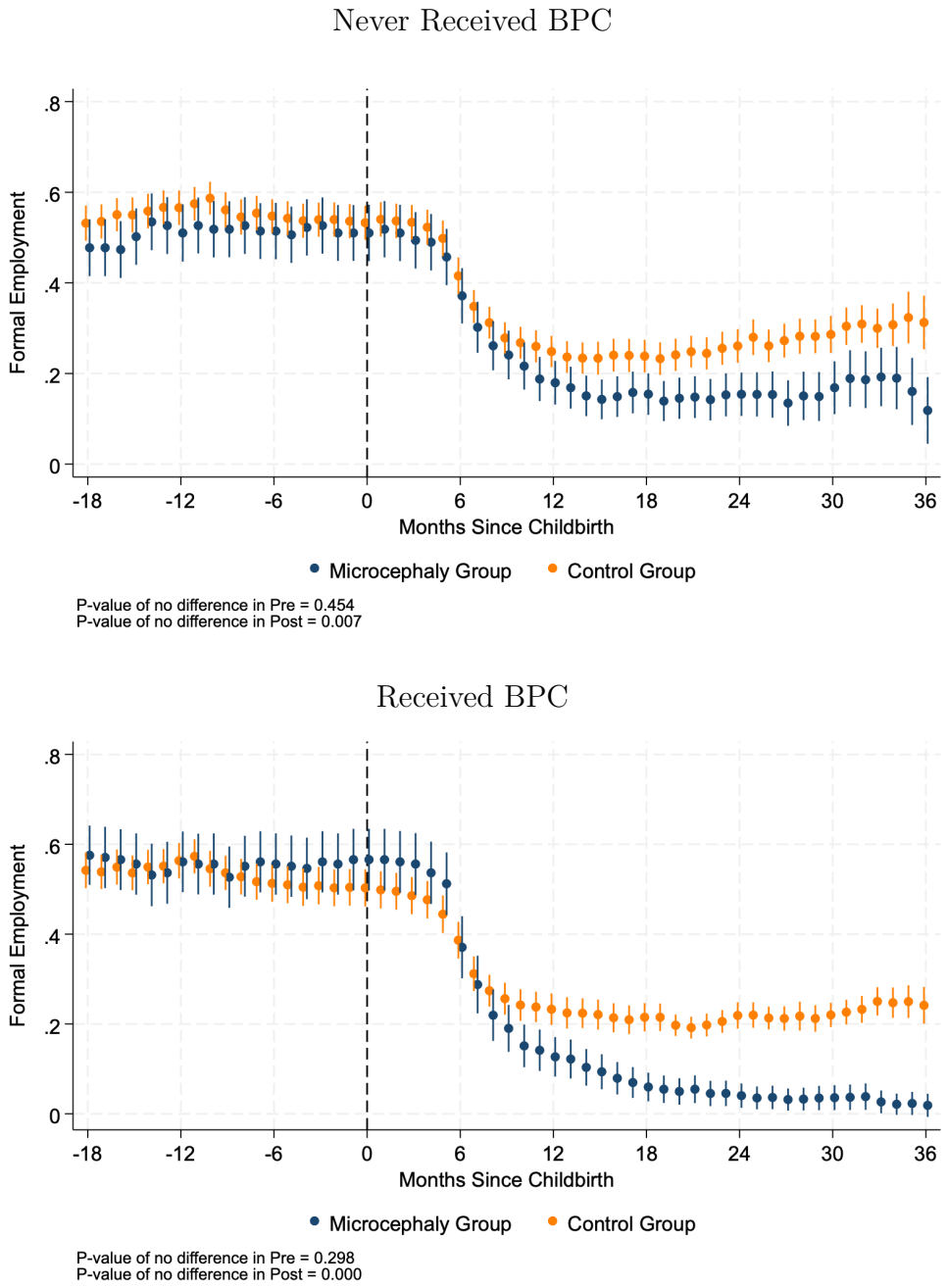
Notes: This figure shows the estimated effect on employment rate (above) and earnings (below) of mothers, accounting for both formal and informal work. The Microcephaly Group consists mothers of children diagnosed with microcephaly, while the Control Group consists of mothers of children without this condition, matched on location, age and time of childbirth. Data is self-reported. Earnings are in BRL, and the error bars represent 95% confidence intervals, with errors clustered at the match-group level.

Figure 8: Formal Employment by Social Assistance Benefits (BPC)



Notes: This figure shows the employment rate of mothers in the formal sector, for those that were recipients of Social Assistance Benefits (BPC) at some point (below) and those that never were (above). The Microcephaly Group consists mothers of children diagnosed with microcephaly, while the Control Group consists of mothers of children without this condition, matched on location, age and time of childbirth. Error bars represent 95% confidence intervals, with errors clustered at the match-group level.

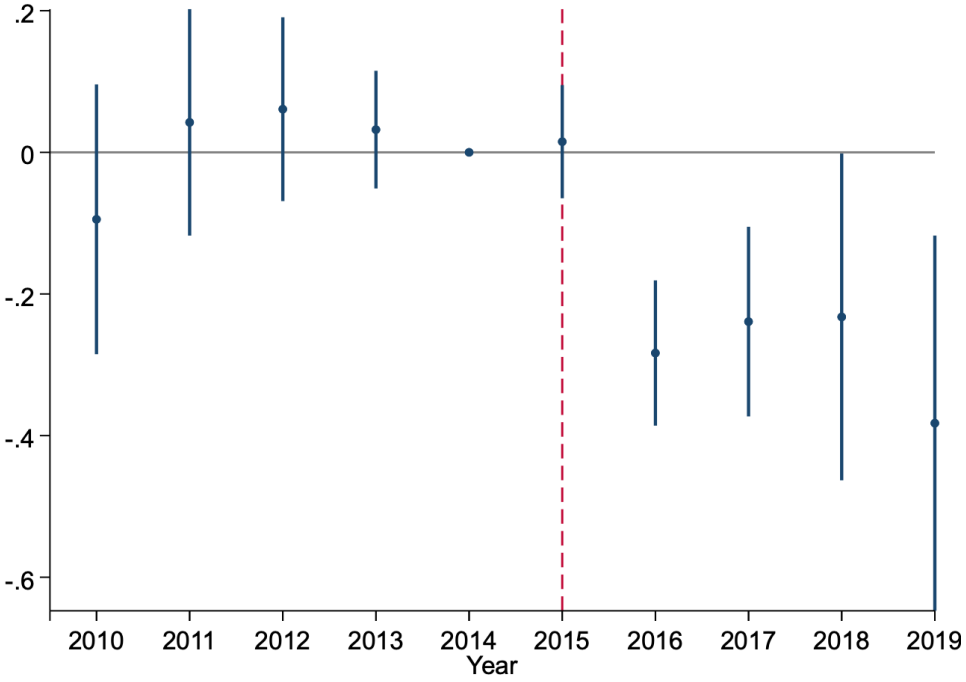
Figure 9: Formal Employment by Social Assistance Benefits (BPC) – Subsample with Previous Formal Employment



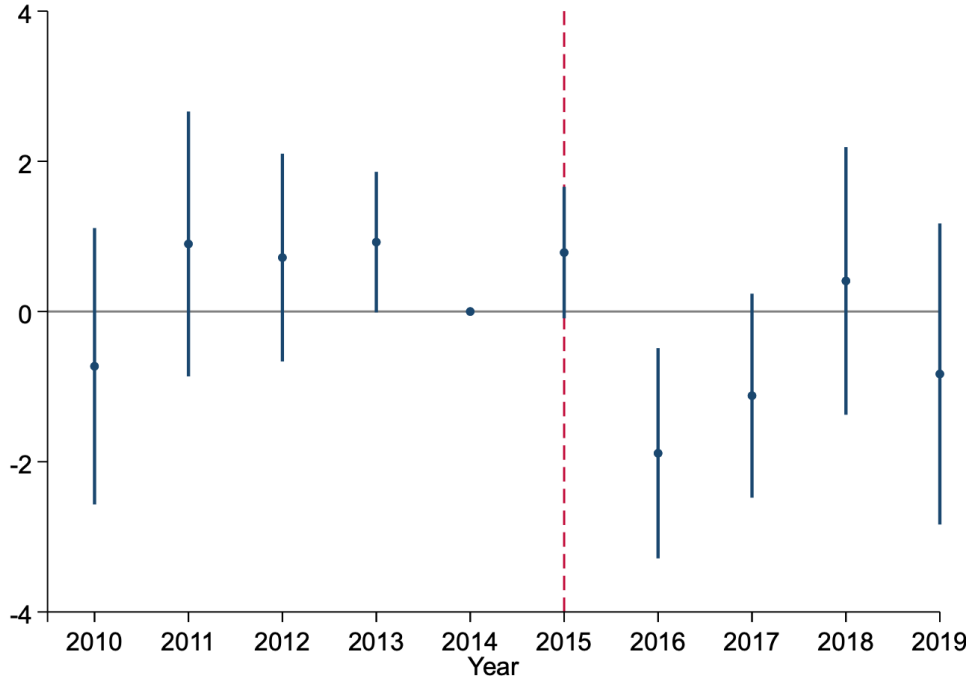
Notes: This figure shows the employment rate of mothers in the formal sector, for those that were recipients of Social Assistance Benefits (BPC) at some point (below) and those that never were (above). This subsample is selected such that every mother had at worked for at least one month in the private sector in the two years before childbirth. The Microcephaly Group consists mothers of children diagnosed with microcephaly, while the Control Group consists of mothers of children without this condition, matched on location, age and time of childbirth. Error bars represent 95% confidence intervals, with errors clustered at the match-group level.

Figure 10: Spillover Effects on Fertility

A: Indicator for High vs Low/No of Cases of Microcephaly



B: Share of Microcephaly on All Births



Notes: This figure shows the estimated spillover effects on fertility. The dependent variable is the fertility rate (per thousand). The independent variables are leads and lags of a dummy for being above the 75th percentile of the distribution of share of microcephaly cases during 2015-2017 (Figure A) and the share of microcephaly cases among all births in the municipality over the same period (Figure B).

Tables

Table 1: Summary Statistics

	Treated	Control	p-value
Age	26.36	25.64	76.7%
Standard Deviation	(6.20)	(5.42)	
Race			
Indigenous	0.5%	0.2%	93.3%
White	19.6%	22.2%	74.3%
Black	10.0%	10.5%	78.8%
Asian	1.3%	1.0%	65.7%
Pardo	69.3%	66.3%	83.1%
Education			
Less than High School	40.1%	29.6%	66.9%
High School or more	53.3%	64.4%	40.5%
N	1,887	35,202	

Notes: This table shows means and standard deviations for the treated and control samples along demographic variables. The treated sample consists of mothers of children with microcephaly, and the control sample consists of matched mothers. The p-value is calculated based on a regression including match-group fixed effects.

Table 2: Effect of Microcephaly on Mothers' Labor Supply

Full Sample				
	Employment		Earnings	
	(1)	(2)	(3)	(4)
Treated	.0077	.0077	12	12
	(.0078)	(.0078)	(8.705)	(8.705)
Post	-.058***	-.056***	-50***	-47***
	(.0031)	(.0032)	(3.341)	(3.433)
Treated × Post	-.042***	-.042***	-39***	-39***
	(.0071)	(.0071)	(7.805)	(7.805)
Number of Obs	1,563,559	1,563,559	1,563,559	1,563,559
Number of Clusters	1,728	1,728	1,728	1,728
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.14	0.14	146.62	146.62

Work Experience Sample				
	Employment		Earnings	
	(1)	(2)	(3)	(4)
Treated	.0048	.016	-1.7	17
	(.0234)	(.0233)	(29.77)	(30.02)
Post	-.27***	-.27***	-241***	-234***
	(.0113)	(.0116)	(13.39)	(14.33)
Treated × Post	-.12***	-.12***	-97***	-98***
	(.023)	(.0229)	(27.83)	(27.88)
Number of Obs	277,116	277,116	277,116	277,116
Number of Clusters	418	418	418	418
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.53	0.53	552.23	552.23

Notes: This table the effect of having a child with microcephaly on mothers' employment an monthly earnings. In the top panel, we show results for the full sample, while the bottom panel shows results for the sample of mothers that worked at least one month of the two years before birth. Post is a dummy that equals one starting 6 months after childbirth, to account for maternity leave. In Columns (1) and (3), there are no additional controls. In Columns (2) and (4), we add match-group fixed-effects. Control observations are weighted by the inverse of the number of controls in the match-group. Standard errors are clustered at the level of the match-group.

Table 3: Effect of Microcephaly on Fathers' Labor Supply

	Employment		Earnings	
	(1)	(2)	(3)	(4)
Treated	.014	-.082*	98	-152
	(.0322)	(.0491)	(67.72)	(100.7)
Post	.043**	.025	81**	74**
	(.0184)	(.0188)	(34.9)	(35.46)
Treated × Post	.025	.0058	58	58
	(.0343)	(.0352)	(71.75)	(66.62)
Number of Obs	88,621	88,618	88,621	88,618
Number of Clusters	833	830	833	830
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.62	0.62	806.77	806.77

Work Experience Sample

	Employment		Earnings	
	(1)	(2)	(3)	(4)
Treated	-.041	-.063	-27	-59
	(.0537)	(.0602)	(81.95)	(99.7)
Post	.052	.04	298***	260**
	(.0353)	(.0331)	(108.4)	(105.2)
Treated × Post	.054	-.0076	-50	-115
	(.0506)	(.0494)	(138.3)	(124.2)
Number of Obs	15,259	15,259	15,259	15,259
Number of Clusters	110	110	110	110
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.59	0.59	741.54	741.54

Notes: This table the effect of having a child with microcephaly on mothers' employment and monthly earnings. In the top panel, we show results for the full sample, while the bottom panel shows results for the sample of mothers that worked at least one month in the two years before birth. In Columns (1) and (3), there are no additional controls. In Columns (2) and (4), we add match-group fixed-effects. Control observations are weighted by the inverse of the number of controls in the match-group. Standard errors are clustered at the level of the match-group.

Table 4: Effect of Microcephaly on Mothers' Self-Declared Labor Supply

	Employment		Earnings	
	(1)	(2)	(3)	(4)
Treated	-.02*	-.019	-4.7	-3
	(.012)	(.012)	(6.763)	(6.78)
Post	.091***	.087***	28***	22***
	(.0074)	(.0069)	(3.554)	(3.471)
Treated × Post	-.083***	-.084***	-20***	-21***
	(.0123)	(.0123)	(7.297)	(7.258)
Number of Obs	115,327	115,327	112,889	112,889
Number of Clusters	1,729	1,729	1,729	1,729
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.25	0.25	81.49	81.49

Notes: This table the effect of having a child with microcephaly on mothers' employment, including both formal and informal, self-declared in the Single Registry, and monthly earnings. Standard errors are clustered at the level of the match-group.

Table 5: Effect on Subsequent Fertility

	Total Children After Treated/Control Child			
	(1)	(2)	(3)	(4)
Microcephaly	.000087	-.005	-.022*	.0076
	(.0081)	(.0087)	(.013)	(.014)
Constant	.13***	.13***	.15***	.13***
	(.003)	(.0044)	(.0069)	(.0065)
Number of Obs	36856	36457	17093	18970
Number of Clusters	1729	1717	1289	1457
Match FE	No	Yes	Yes	Yes
Sample	Full	Full	Firstborn	Not firstborn

Notes: This table shows the total fertility up to three years after the birth of the child with microcephaly. Columns (1) and (2) include all families. We split the sample among families where the child with microcephaly or matched control was the first child (column (3)) and those where it was not (column (4)).

Table 6: Spillover on Fertility

	Cases per thousand births	High incidence
	(1)	(2)
After × Exposure	-.89	-.23**
	(.79)	(.096)
Number of Obs	55,650	55,650

Notes: This table shows the results of the incidence of microcephaly in the municipality on fertility. We use two different measure of exposure. The first measure, shown in column (1), is the total number of cases between 2015 and 2017, divided by total births over the same period. The second measure, shown column (2), is a dummy indicating whether the number of cases per capita is in the 75th percentile of the distribution. Standard errors clustered at the municipality level.

Table 7: Family Structure

	Father Present in 2017	Father Present in 2019	
	(1)	(2)	(3)
Microcephaly	.013	.0085	-.0028
	(.0098)	(.0093)	(.0039)
Father present 2017			.86***
			(.0095)
Constant	.19***	.16***	.0023
	(.0049)	(.0046)	(.0033)
Number of Obs	37,089	37,089	37,089
Number of Clusters	1,728	1,728	1,728
Match FE	Yes	Yes	Yes

Notes: This table shows the effect of having a child with microcephaly on the likelihood of cohabiting fathers. Column 1 shows effects in 2017 and columns 2 and 3 show effects in 2019.

A Appendix

Table A1: Variables Included in the LASSO Regression

Category	**Variable**	**Type/Levels**
Birth Characteristics	Municipality	Dummy for each in sample
	Month	12 Levels
	Year	2015, 2016 or 2017
Mother's Education	Year of birth	Fourth Power
	Completed high school	Binary
	Highest level of education attended	14 Levels
	Was it completed?	Binary
	Education level currently enrolled	14 Levels
Socioeconomic Factors	Mother's race	5 levels
	Mother's type of job (e.g. informal, temporary)	11 Levels
	Mother's wage income	Fourth power
	Mother's total income	Fourth power
	Family income	Fourth power
Housing Characteristics	Number of rooms	Cube
	Number of bedrooms	Linear
	Type of floor	7 Levels
	Dwelling material	8 Levels
	Formal dwelling	3 levels
	Is rural	Binary
	Access to water	4 Levels
	Piped water	Binary
	Has a bathroom	Binary
	Type of sewer	6 Levels
	Type of trash disposal	6 Levels
	Type of lighting	6 Levels
	Type of sidewalk	3 Levels
	Number of inhabitants	Cube

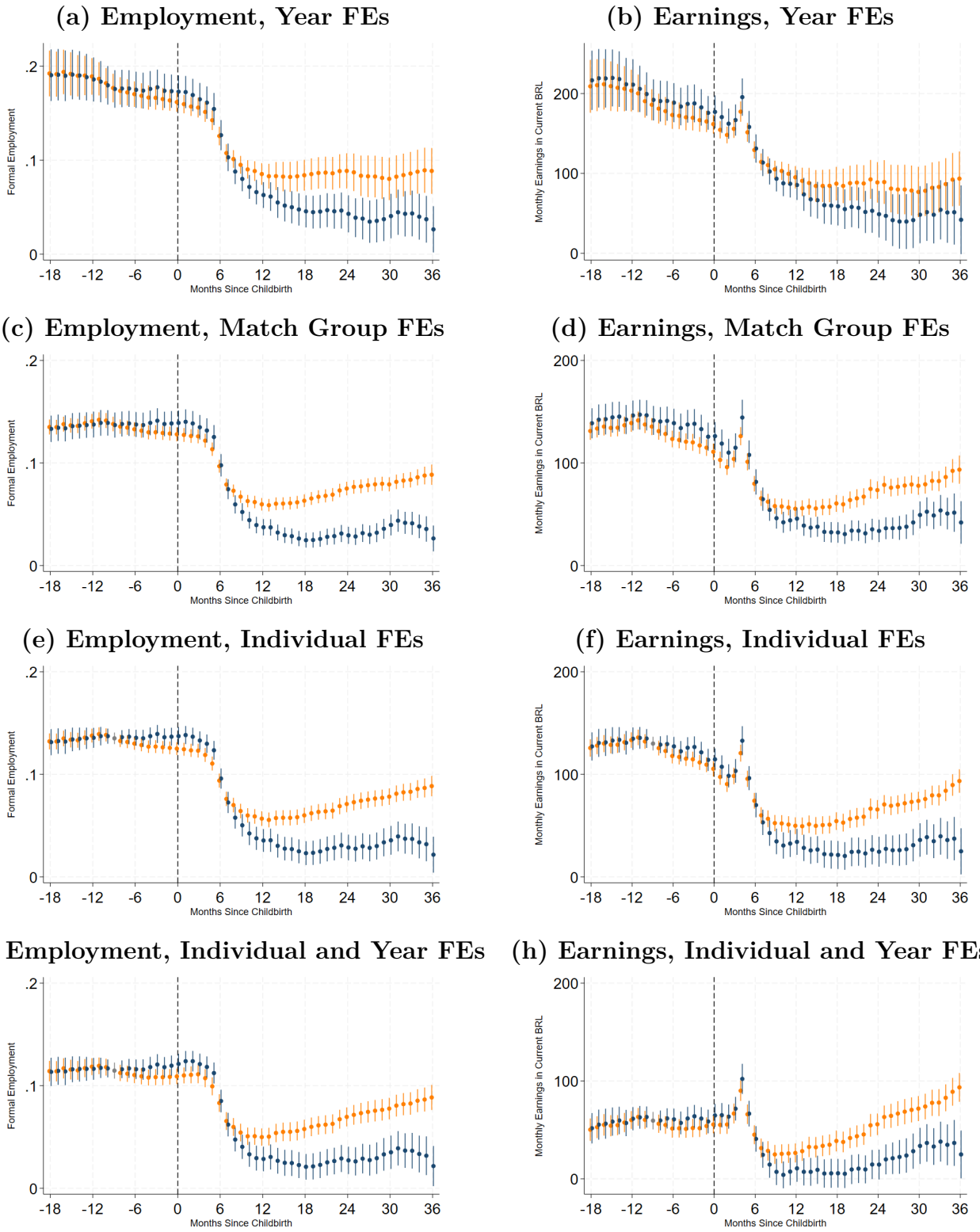
Notes: This table shows the variables included in the Logit LASSO to predict microcephaly in a child. Birth characteristics are from the SUS/NASC, all other variables are from the Single Registry.

Table A2: Logistic Regression Results

	Coefficient	p-value
Mother's Characteristics		
Race: "Black"	0.065	0.545
Age (years)	0.013	0.021
Type of job: Formal	-0.148	0.524
Type of job: No answer	0.168	0.049
Joint F-test		*0.0075*
Mother's Education		
Attending: Adult Education	0.538	0.193
Attending: Fifth grade	-0.575	0.002
Attending: Sixth grade	-0.182	0.239
Attending: Ninth grade	0.100	0.622
Did not finish highest grade attended	-0.226	0.001
Joint F-test		*0.0005*
Dwelling Characteristics		
Dwelling Type: "Informal"	-0.404	0.073
Number of Bedrooms: "Zero"	0.733	0.478
Type of Floor: "Wooden"	0.098	0.793
Type of Walls: "Thatch"	1.146	0.075
Trash Disposal: "Other"	0.132	0.410
Lighting: "Electric, community owned"	0.168	0.269
Street Paving: "Partial"	-0.032	0.818
Joint F-test		*0.2416*
Year-month Fixed Effects	Yes	0.0000
Observations		8,735

Notes: This table shows the results of a logit regression of an indicator of microcephaly in the child on characteristics of the mother and of the dwelling. The regressors were chosen based on LASSO using a large set of variables in the Single Registry and health data. The joint F-tests presented correspond to the Wald test with the null hypothesis the each coefficient in the corresponding group is equal to zero. The sample includes all cases of microcephaly we identified plus a random sample of nine other births in the same municipality for each microcephaly case. Standard errors clustered at the municipality level.

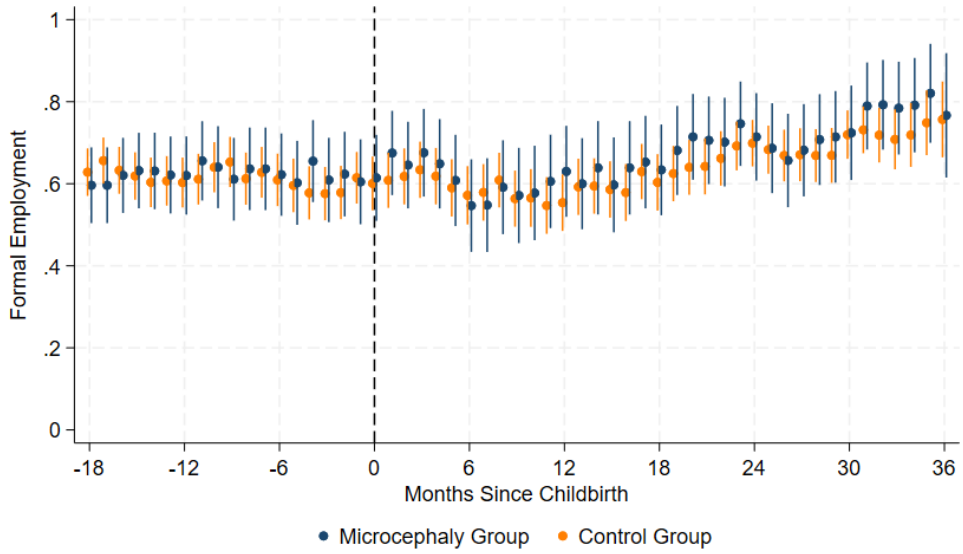
Figure A1: Robustness to Fixed Effects



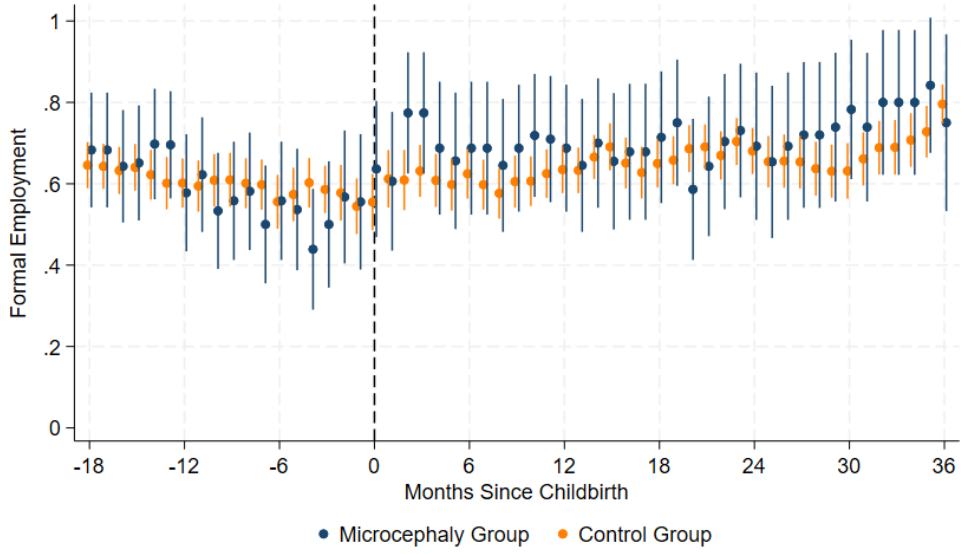
Notes: Robustness checks of employment and wage outcomes using different fixed effects specifications. Orange represents controls, and blue represents mothers of children with microcephaly. Panels (a) and (b) include year fixed effects. Panels (c) and (d) incorporate match group fixed effects. Panels (e) and (f) account for individual fixed effects, while panels (g) and (h) combine both individual and year fixed effects.

Figure A2: Fathers' Formal Employment by Social Assistance Benefits (BPC)

Never Received BPC

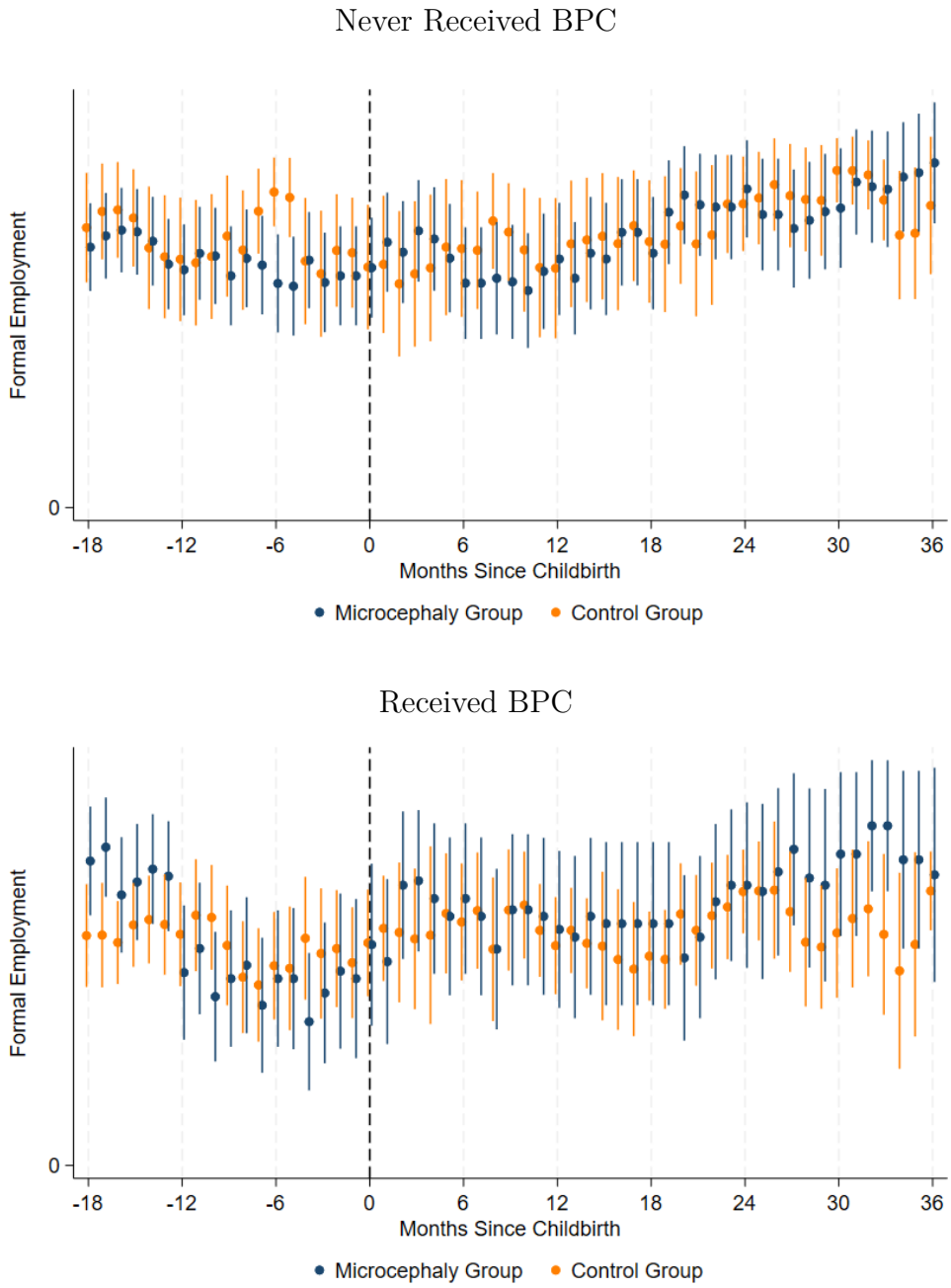


Received BPC



Notes: This figure shows the employment rate of fathers in the formal sector, for those that were recipients of Social Assistance Benefits (BPC) at some point (below) and those that never were (above). The Microcephaly Group consists fathers of children diagnosed with microcephaly. The Control Group consists of fathers of children without this condition, matched on location, age, time of childbirth, and mother's age. Error bars represent 95% confidence intervals, with errors clustered at the match-group level.

Figure A3: Fathers' Formal Employment by Social Assistance Benefits (BPC) – Subsample with Previous Formal Employment



Notes: This figure shows the employment rate of fathers in the formal sector, for those that were recipients of Social Assistance Benefits (BPC) at some point (below) and those that never were (above). This subsample is selected such that every father had at worked for at least one month in the private sector in the two years before childbirth. The Microcephaly Group consists fathers of children diagnosed with microcephaly. The Control Group consists of fathers of children without this condition, matched on location, age, time of childbirth, and mother's age. Error bars represent 95% confidence intervals, with errors clustered at the match-group level.