Congenital Disability Effects on Parents' Labor Supply and Family Composition: Evidence from the Zika Virus Outbreak

João Garcia* Rafael Latham-Proença† Marcela Mello‡

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Abstract

Severe child disability is among the most consequential events to parents' labor market outcomes, but there is still a small literature studying its effects. We study this question in the context of the Zika Virus epidemic in Brazil, which caused thousands of children to be born with microcephaly. We argue that several characteristics of the epidemic make it suitable as a natural experiment. Infection was sudden, and the link between Zika and microcephaly was unknown at the time. Using data on the universe of births and formal employment links in the country, we show that affected mothers' formal employment falls by 3.2 percentage points (15%), in addition to the fall of 5.1 percentage points (27%) associated with childbirth in the controls, despite identical labor market trajectories before childbirth. We do not observe any effects on fathers' labor market outcomes. We do not find significant differences in subsequent fertility or marriage dissolution.

*Department of Economics, Brown University. Email: joao_garcia@brown.edu.

†Department of Public Policy, Harvard University. Email: rafaelproenca@g.harvard.edu.

‡Department of Economics, Brown University. Email: marcela_mello_silva@brown.edu.

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1 Introduction

Mothers' labor market decisions are influenced by their children's characteristics, and severe, permanent disability may be one of the most profoundly impactful. Traditionally, women meet 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 help these families.

The small existing literature on child disability and maternal employment faces challenges dealing with unobserved co-founders. For instance, mothers who follow preventive recommendations such as folate supplementation or abstaining from smoking are likely different in other relevant dimensions than those who do not. This concern is identified and dealt with in various ways in the broader literature on child health and mother's work (e.g., Frijters et al. (2009) use instrumental variables, Breivik and Costa-Ramón (2022) use panel data to obtain a valid comparison group). Existing work on child disability, however, has not dealt explicitly with it (Chen et al., 2023; Cheung et al., 2023; Gunnsteinsson & Steingrimsdottir, 2019; Powers, 2001, 2003; Salkever, 1982; Wasi et al., 2012).

In this paper, we provide evidence on the causal effects of child disability on parental labor force participation, household composition, fertility, and income by exploiting a large shock to the incidence of child disability: the 2015 Zika virus outbreak in Brazil. The outbreak caused several thousands of children to be born with a severe disability, microcephaly. We argue that the sudden onset of this event and the characteristics of the infection rule out 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 in a simple set of characteristics. However, starting the typical maternity leave (6 months), their labor force participation and earnings fall much faster. These mothers see a 60% larger motherhood penalty, corresponding to a fall from 15% to about 5%. For fathers, we do not find any effect in the formal labor market participation, nor see lower cohabitation rates. We also document that, for households where the first child was born during the Zika outbreak, families with a child with microcephaly are less likely to have other children.

The Zika virus outbreak in Brazil in 2015 provides a valuable case study because its particular characteristics rule out several threats to identification. Since it is transmitted by a common mosquito, anyone in affected areas could be 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 know 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 microcephaly when it happens in the first trimester. Another potential threat to identification, selective abortion, is unlikely for two reasons: infection is asymptomatic in most cases, so women are unaware, and diagnosis of microcephaly is difficult before birth. Finally, Zika has no lasting effects on adults, ruling out direct effects on labor supply.

Children affected by the Zika outbreak developed microcephaly, a severe, life-long disability that puts significant strain on parental resources. The condition is characterized by underdevelopment of the brain, resulting in cognitive and developmental disabilities. Children often suffer from seizures and must have access to therapy to develop speech and movement. Brazil's public health care system offers free treatment, but families may have difficulty accessing it, particularly in remote areas. Furthermore, even with free medical

treatment, families must spend significant time caring for affected children at home.

To study the impact of the outbreak on maternal labor outcomes, we use three administrative datasets. The first is SINASC/SUS, which logs all births in the country and details the municipality and date of the delivery, the mother's residence, the mother's date of birth, and whether the newborn has microcephaly. Microcephaly occurs very rarely due to causes unrelated to Zika, so we can confidently link cases during 2015-2016 to the outbreak. The second is the Annual Account of Social Information (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, a federal registry of all recipients of social programs. 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.

To isolate the causal effect of child disability, we compare the labor market trajectory of mothers of children with microcephaly to a matched comparison group. This group is composed of all mothers in the same municipalities who gave birth during the same months as mothers of children with microcephaly. We compare the average labor force participation between these two groups each month following maternity leave. We argue this method yields causal estimates for two main reasons. First, the unexpected nature of the epidemic and the characteristics of the infection make selection bias unlikely. Second, the groups are similar in observable characteristics, including previous trajectories in the labor force.

We find that mothers of children with microcephaly are about 50% (3.2 percentage points) less likely to have a job in the formal sector than matched mothers. This difference

starts about six months after the start of maternity leave and persists for as long as we can estimate, i.e., 36 months. We find no effect on father's labor market outcomes using the same method.

The literature on the effects of child disability on parents' labor supply is still small. Powers (2001), Salkever (1982), and Wasi et al. (2012).Chen et al. (2023) and Cheung et al. (2023) study the impact of congenital disability in Taiwan, and Gunnsteinsson and Steingrimsdottir (2019) study this question in Denmark. 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). We focus on the additional penalty associated with a disabled child, which can compound

the adverse labor market effects on mothers and increase gender inequality, as childcare responsibilities tend to fall on mothers.

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 mother's employment. Selection driven by differences in preventive behavior is addressed by the sudden and widespread nature of the outbreak in the affected regions. Selective abortion is unlikely because diagnosis is difficult in the uterus, and adults have no symptoms. The lack of symptoms also rules out direct effects of the virus on labor outcomes.

The Zika virus was introduced to Brazil around 2014, where it had never been observed before. The virus spreads through a common mosquito, the Aedes Aegypti, which also transmits dengue, yellow fever, and Chikungunya. It affects around 2 million Brazilians per year. The outbreak was first identified in late 2015 following a spike in cases of microcephaly. The Northeast of Brazil was particularly affected, but infection was widespread within the region and anyone could be exposed.

Exposure to the Zika virus in pregnant mothers, especially in the first trimester, can cause microcephaly in the newborn, a severe, lifelong disability. Microcephaly is characterized by underdevelopment of the brain, resulting in 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 Guillan-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.

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 microcephaly births 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.

Differential exposure to the virus based on differences in mothers' preventive behavior is unlikely cause bias for two main 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 identify the causal link between these

facts in 2016, so mothers would only know to take precautions afterward. Even then, preventive measures would probably only cause a reduction in cases of disabled children with a significant delay. Since the virus is more likely to cause microcephaly during the first trimester of pregnancy, its effects can be undetected for several months.

Another potential threat to identification, differential rates of abortions, is unlikely for several reasons. First, microcephaly is difficult to identify in the uterus, and mothers would have to decide to terminate pregnancy without confirmation that their baby 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 baby developing microcephaly is relatively low. Finally, abortion is illegal in Brazil except in cases of rape, or serious risk to the mothers' life.

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.

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*, or

Information System on Live Births), a dataset collected by the Ministry of Health detailing every live birth within a health facility. Second, RAIS (*Relação Anual de Informação Social*, Annual Report of Social Information), 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 age.

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 observe the municipality where the birth occurred, the municipality of the mother's residence, the date, the mother's age, and whether the newborn has microcephaly or any other birth anomaly.

This dataset contains detailed information on all live births in Brazil. It provides the location of the birth, the mother's municipality of residence, date of birth, and several variables, such as 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 over 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.

3.4 Linking the Datasets

Because the public dataset on births does not include personal identifiers, we cannot directly link it to RAIS or Single Registry. We deal with this challenge using the mothers' date of birth, municipality of residence, and date of childbirth, available on Single Registry. Once we select the control and treated mothers in the Single Registry, we use their tax ID to find them in RAIS.

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.

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 test this hypothesis by comparing observable variables and find no pre-existing differences, and we argue that the characteristics of the epidemic made selection on non-observables unlikely.

Because we use exact matching with fairly coarse variables, it is possible for one treated unit is matched to several possible controls, as well as for multiple treated units to have identical characteristics. In this case, we call we cell of 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.

While our main estimates are simple comparisons of (weighted) means, we also present differences-in-differences estimates, corresponding to the following model:

$$y_{ft} = \sum_{k \in (-18,\ldots,36), k \neq -9} \beta_k \cdot T_f \times \mathbb{1}(t - \tau(f) = k) + \alpha_{p(f)} + \delta_t + \varepsilon_{ft} \tag{1}$$

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 β_k, captures the difference between the outcomes of families with microcephaly and the other families. We control for pair fixed effects, $\alpha_p(f)$, to ensure we are comparing each treated family with the most similar control families. We also add for year-month fixed effects, δ_t, to capture to any time-trend common to all families. We normalize the coefficients relatively to nine months before the childbirth. ε_{ft} is the random error, clustered at the match-group level.

Our identification assumption is that, conditional on having a child around the same time, in the same municipality, and mothers's age and educational level, the incidence of microcephaly is uncorrelated with unobserved characteristics that affect the

outcomes of interest. As discussed in details in Section 2, the characteristics of the outbreak rules 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.

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.[1]

5 Results

In this session we present our estimates of the effects of child disability in the family. We find a decrease on mothers' labor supply and earnings corresponding to half the motherhood penalty, or about 15% relative to 9 months before childbirth and no effects for fathers. In terms of 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 lower prevalence.

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

5.1 Balance and Summary

Table 1 shows summary stats for affected mothers and for controls. Overall, our control group seem 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 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 sample self-declare 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 control and treated in characteristics are minimal and not statistically significant.

5.2 Employment and Earnings

We find that after the birth of child with microcephaly, mothers' formal employment falls by an additional 3.2 percentage points (15%), in addition to the fall of 5.1 percentage points (27%) associated with childbirth in general. 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 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 the affected mothers. After month 6, corresponding to the end of typical maternity leave, we see that mothers of children with microcephaly see a fall in employment roughly 50% larger than that of the controls, and the difference is entirely persistent. 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 shows the results with a 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 for formal employment or earnings. Figure 6 shows the results. Note that we maintain the same match-groups as

the ones 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 results of the DID specification. The estimates indicate a null effect on employment and a positive but not statistically significant effect on earnings. Notably, employments and earnings tend to increase after childbirth, although this effect is also not significant after accounting for match-group fixed effects. This may suggest 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, so a strict causal interpretation of the parameter requires strong assumptions.

5.3 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 impact in future fertility compared to paired controls, and mostly not statistically significant.

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 4 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 of 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.

5.4 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 for 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%.

Table 5 shows the results. We find that, if anything, there is a slightly higher chance of the father being present in families with a child with disability, although the difference is small in magnitude and not significant once we adjust for match-group fixed effects and re-weight. The estimates are very 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 afterwards, and adding this control renders the estimate of the effect of microcephaly equal to zero.

6 Conclusion

In this paper, we analyse 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 falls by close to one half, 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 substantially reduce several concerns, such as endogeneity of maternal care and health behaviors and selective abortion or mortality. Our paper contributes to the literature studying the effects of this outbreak by highlighting the effects 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.

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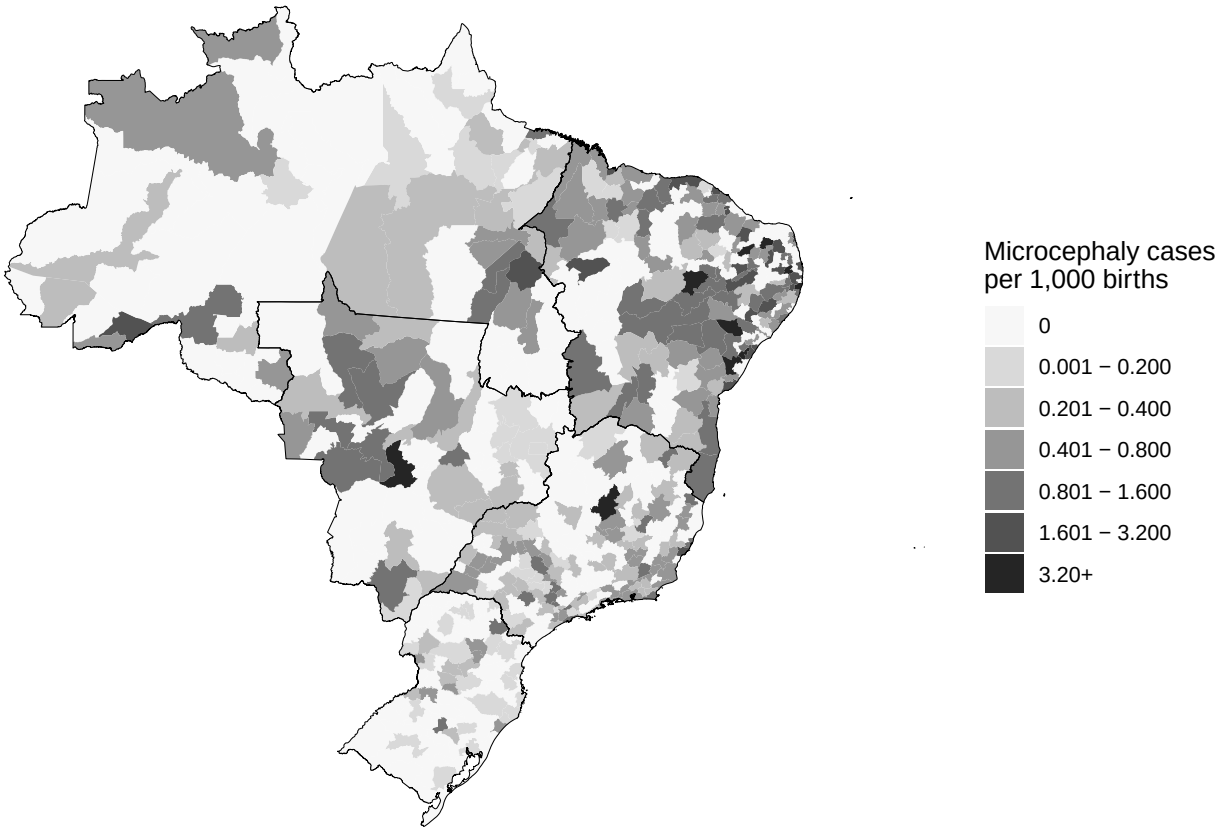
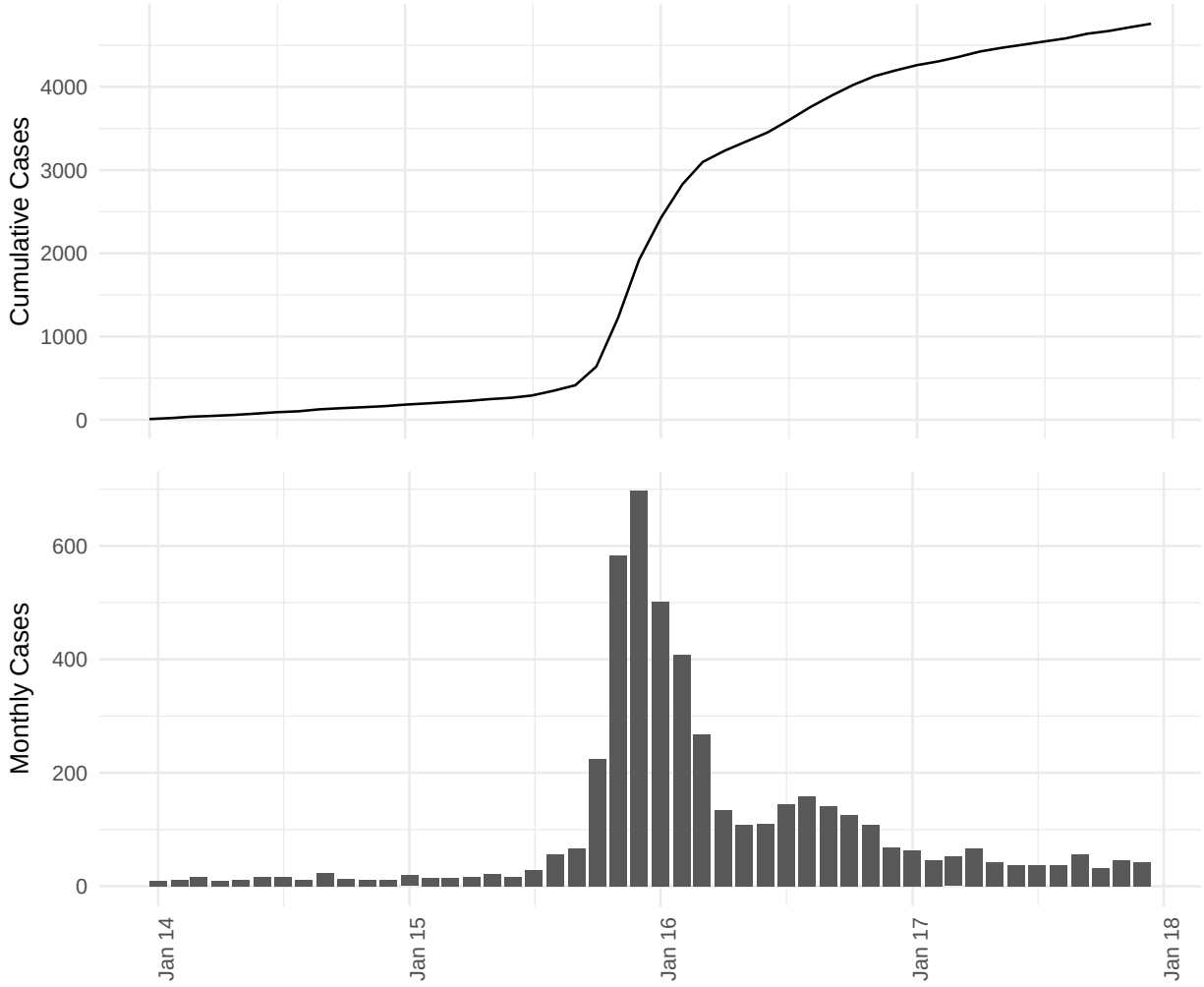
Figures

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



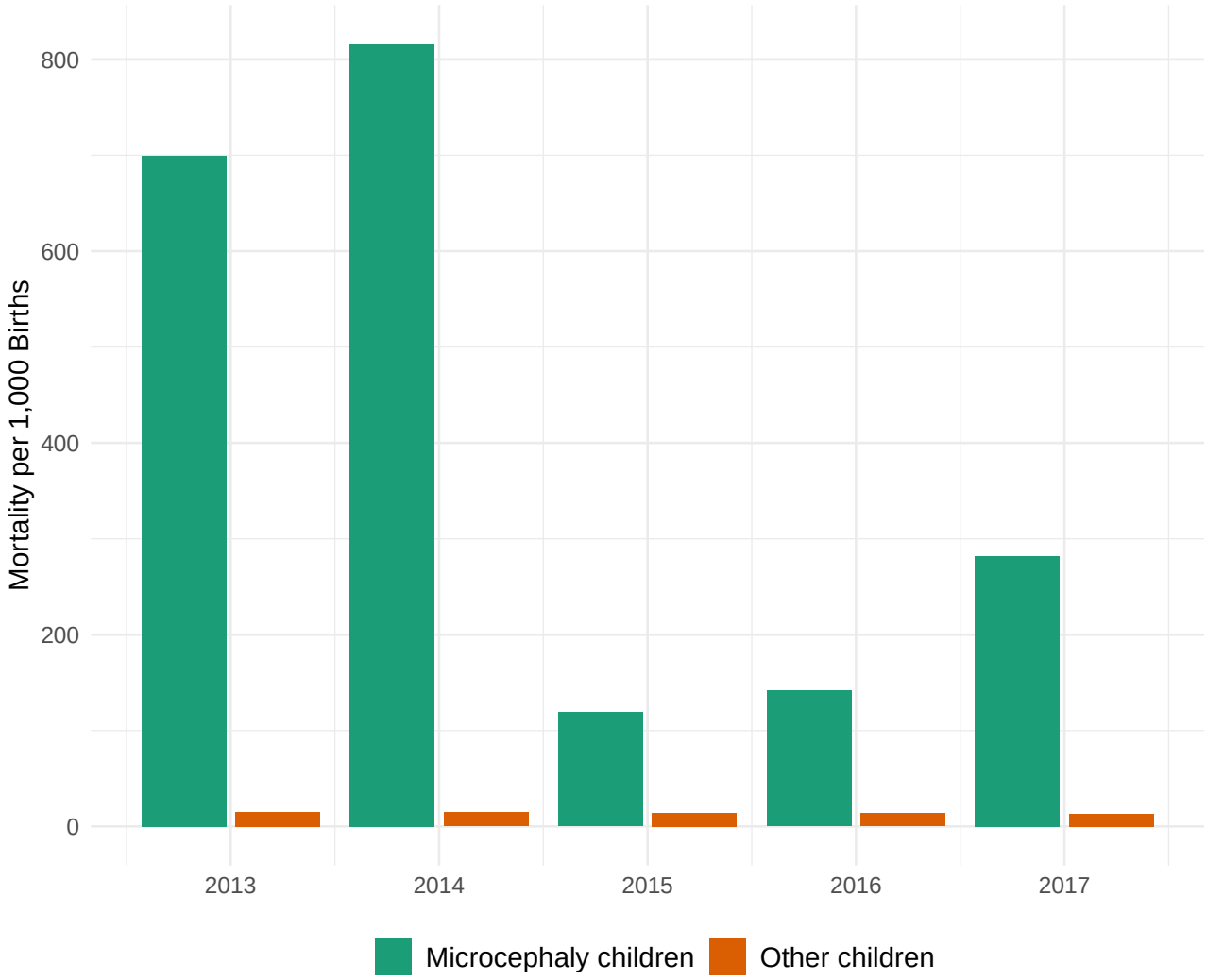
Notes: This figure illustrates the geographic variation on the number of microcephaly cases per thousand births in 2015 and 2016. Each polygon is a micro-region, comprising on average about 10 municipalities. Micro-regions with zero births in the period are assigned to the zero cases per 1,000 births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

Figure 2: Microcephaly Cases by Month



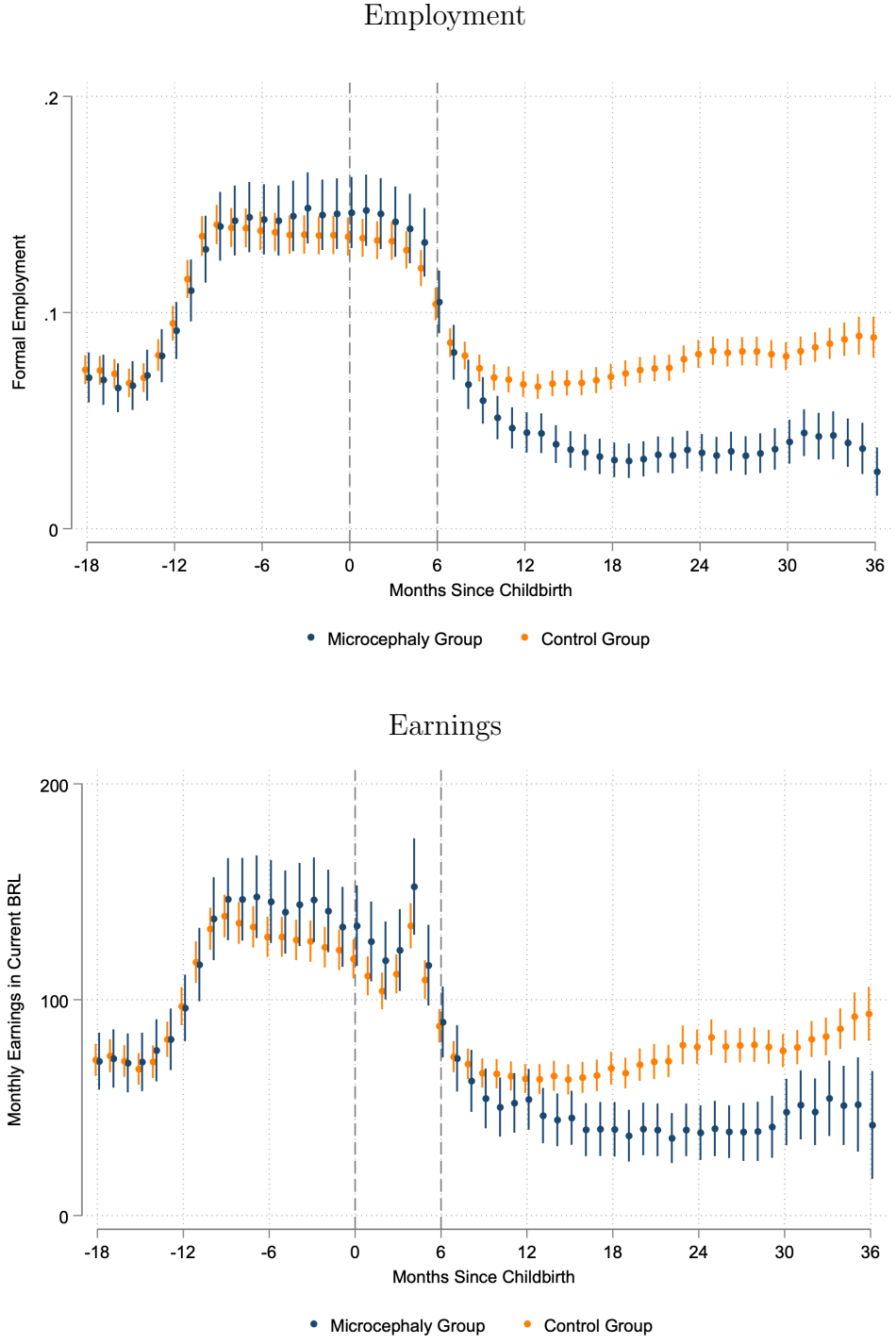
Notes: These figures show the evolution in the total number of cases of microcephaly, over the Northeast and Southwest regions. The top graph shows cumulative cases, while the bottom shows monthly incidence. The data is from SINASC/SUS.

Figure 3: Mortality Rates of Children with Microcephaly



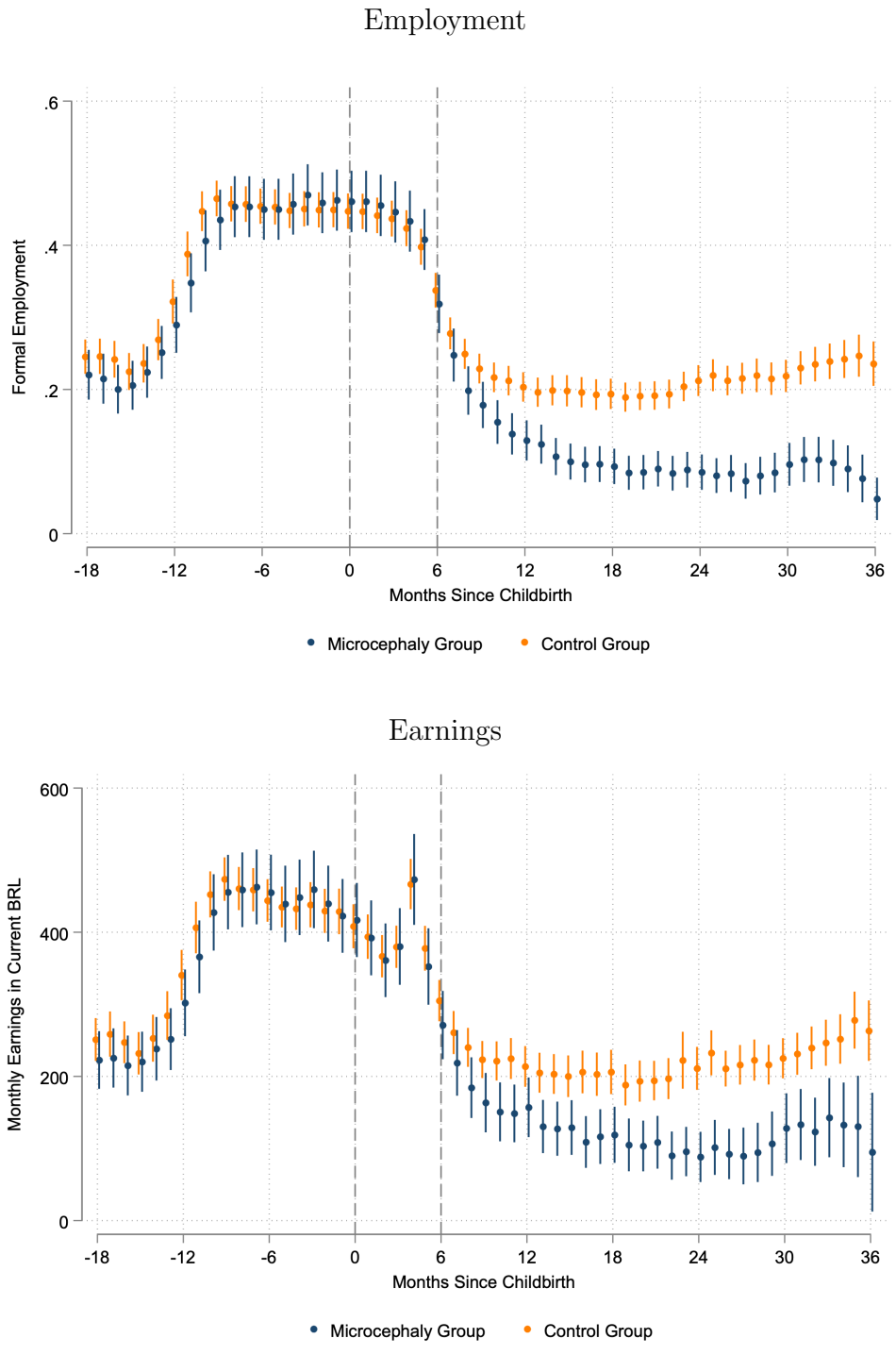
Notes: This figure shows mortality by age 5 per thousand births, separately for children born with microcephaly compared to others. The year indicates year of birth, not death. The total number of births and cases of microcephaly are made available by SINASC/SUS. Microcephaly is identified by the ICD-10 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 in location, age and time of childbirth. Vertical dashed lines at 0 and 6 months indicate the month of childbirth and the typical end of maternity leave, respectively. Earnings are in BRL, and the error bars represent 95% confidence intervals.

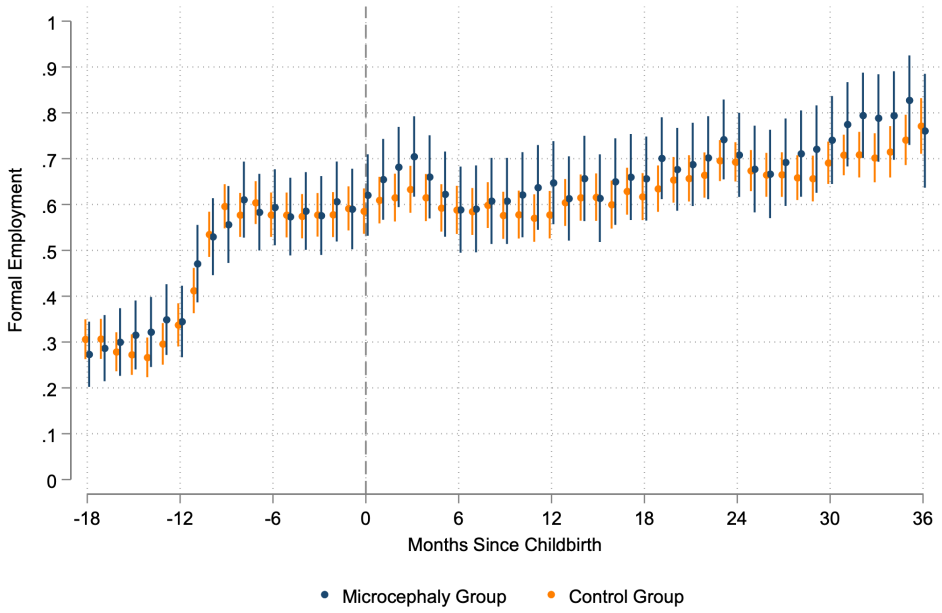
Figure 5: Subsample with Previous Formal Employment



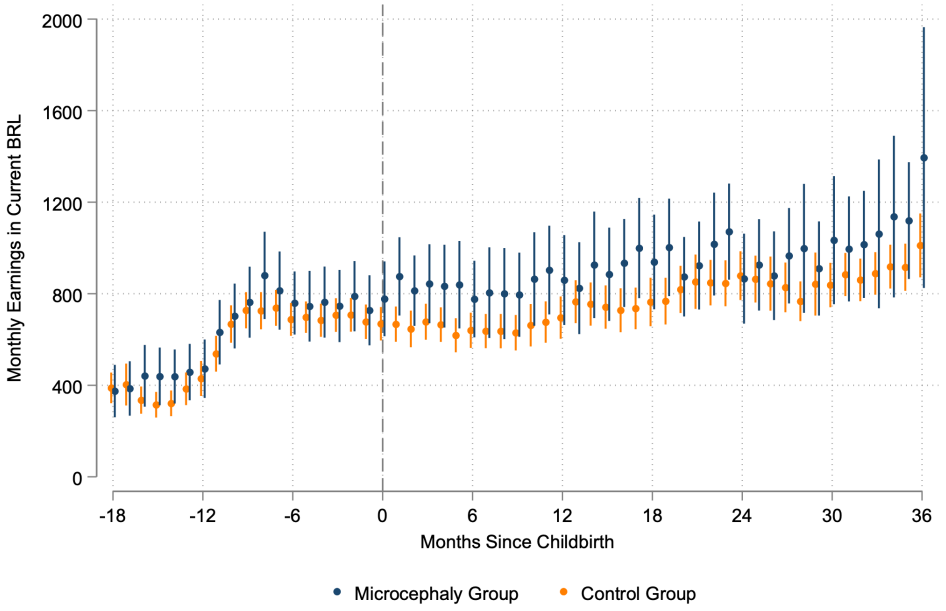
Notes: This figure shows the employment rate (above) and earnings (below) of mothers in the formal sector. This subsample is selected such that every mother had at worked for at least one month in the private sector in the two years before childbirth. The Microcephaly Group consists mothers of children diagnosed with microcephaly, while the Control Group consists of mothers of children without this condition, matched in location, age and time of childbirth. Vertical dashed lines at 0 and 6 months indicate the month of childbirth and the typical end of maternity leave, respectively. Earnings are in BRL, and the error bars represent 95% confidence intervals.

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 mothers of children diagnosed with microcephaly, while the Control Group consists of mothers of children without this condition, matched in location, age and time of childbirth. Vertical dashed lines at 0 and 6 months indicate the month of childbirth and the typical end of maternity leave, respectively. Earnings are in BRL, and the error bars represent 95% confidence intervals.

Tables

Table 1: Summary Statistics

	Treated	Control	p-value
Age	26.36	25.64	.767
sd	(6.20)	(5.42)	
Race			
Indigenous	.005	.002	.933
White	.196	.222	.743
Black	.100	.105	.788
Asian	.013	.01	.657
Pardo	.693	.663	.831
Education			
Less than High School	.401	.296	.669
High School or more	.533	.644	.405
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				
	Works		Earnings	
	(1)	(2)	(3)	(4)
Treated	.0065	.0065	14	14
	(.008)	(.008)	(9.1)	(9.1)
Post	-.051***	-.049***	-49***	-46***
	(.0028)	(.0028)	(3.1)	(3.1)
Treated × Post	-.032***	-.032***	-32***	-32***
	(.0061)	(.0061)	(7.1)	(7.1)
Number of Obs	1563559	1563559	1563559	1563559
Number of Clusters	1728	1728	1728	1728
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.14	0.14	142.69	142.69

Work Experience Sample				
	Works		Earnings	
	(1)	(2)	(3)	(4)
Treated	.00071	.014	4.5	29
	(.022)	(.023)	(27)	(28)
Post	-.2***	-.2***	-190***	-186***
	(.0083)	(.0085)	(9.9)	(11)
Treated × Post	-.081***	-.081***	-80***	-80***
	(.018)	(.018)	(22)	(22)
Number of Obs	356108	356108	356108	356108
Number of Clusters	507	507	507	507
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.45	0.45	462.32	462.32

Notes: This table the effect of having a child with microcephaly on mothers' employment. 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 36 months 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 3: Effect of Microcephaly on Fathers' Labor Supply

	Works		Earnings	
	(1)	(2)	(3)	(4)
Treated	.0023	-.08	71	-154
	(.038)	(.052)	(71)	(103)
Post	.052***	.027	51	31
	(.019)	(.018)	(34)	(34)
Treated × Post	.037	.0076	87	52
	(.04)	(.041)	(69)	(65)
Number of Obs	88621	88618	88621	88618
Number of Clusters	833	830	833	830
Match FE	No	Yes	No	Yes
Mean Dep. Var. Baseline	0.58	0.58	745.66	745.66

Notes: This table the effect of having a child with microcephaly on mothers' employment. 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 36 months 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 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 chance of having another child 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 5: Family Structure

	Father Present in 2017		Father Present in 2019		
	(1)	(2)	(3)	(4)	(5)
Microcephaly	.049***	.013	.043***	.0085	-.0028
	(.0094)	(.0098)	(.0088)	(.0093)	(.0039)
Father present 2017					.86***
					(.0095)
Constant	.15***	.19***	.13***	.16***	.0023
	(.004)	(.0049)	(.0035)	(.0046)	(.0033)
Number of Obs	37089	37089	37089	37089	37089
Number of Clusters	1728	1728	1728	1728	1728
Match FE	No	Yes	No	Yes	Yes
Weights	Uniform	Inv. Control #	Uniform	Inv. Control #	Inv. Control #

Notes: This table shows the effect of having a child with microcephaly on the likelihood of cohabiting fathers in 2019. Column (1) shows the simple comparison of means between families of children with microcephaly and matched controls. Column (2) introduces match-group fixed effects. Column (3) introduces weighting equal to the inverse of the number of controls in each match group. Column (4) restricts the sample to children born in 2015 or 2016. Standard errors are clustered at the level of the match-group.