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Evidence from Mexico

Kerstin Perlik
Nicolás Corona Juárez
Jan Priebe

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Kerstin Perlik*

Nicolás Corona Juárez**

Jan Priebe***

* Bernhard-Nocht Institute for Tropical Medicine (BNITM)

** Universidad de las Américas Puebla

*** BNITM and Hamburg Center for Health Economics

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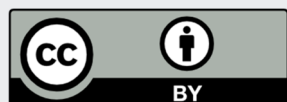
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Abstract^{*}

Congenital disorders are a principal cause of early mortality, long-term disabilities, impaired cognitive development and constitute a major challenge to families, communities, and health care systems. The origins of congenital disorders are, however, not yet well understood. Using a high-dimensional fixed-effects model that includes municipality specific time and locality-by-month fixed effects, this study provides the first causal evidence on the role of high ambient temperature during pregnancy in affecting the onset of congenital disorders. We compiled a large dataset comprising about 19 million births from about 63,000 Mexican localities during 2008–2021 and connect it with local temperature data. We estimate that a 1°C increase in the average monthly maximum temperature during gestation is associated with a rise in the incidence of congenital disorders by 2.4 percent (0.022 percentage points). Furthermore, we provide suggestive evidence that newborns from indigenous mothers are more likely to develop congenital birth disorders compared to children from non-indigenous parents when exposed to high ambient temperatures.

JEL classifications: I14, I31, Q54

Keywords: Birth outcomes, Climate shocks, Indigenous, Mexico

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1. Introduction

Each year more than 9 million babies are born with a congenital disorder, representing 3 percent of all newborns in the world. Congenital disorders are functional anomalies that exist at or before birth and can be identified perinatally, at birth, or later in infancy. The most common congenital disorders are heart defects, neural tube defects, and Down syndrome (WHO, 2023).

The consequences of congenital birth disorders are massive. Annually, over 400,000 newborns with congenital disorders die within the first five years. Additionally, congenital disorders can cause long-term disabilities as well delayed cognitive and physical development, representing a significant burden on individuals, families, communities, and health care systems (WHO, 2023).

Despite their significance, the causes of most congenital disorders remain largely unknown. Some are associated with genetic abnormalities (e.g., Down syndrome) or nutrient deficiencies (e.g., neural tube defects). In addition to genetic and socioeconomic factors, environmental conditions during the perinatal phase—particularly heat exposure—have been linked to increased risk. Correlative evidence suggests that extreme weather events like heat waves may contribute to higher rates of congenital disorders (Dalugoda et al., 2022; Haghighi et al., 2021; Harville et al., 2011; Lakhoo et al., 2025; Li et al., 2018; van Zutphen et al., 2012). Animal studies have shown a causal relationship between elevated temperatures and congenital disorders, while epidemiological and public health research in humans supports this association (see Haghighi et al., 2021; Lakhoo et al., 2025). However, robust quantitative and causal evidence for humans remains limited.

Climate change is expected to increase global ambient temperature and the frequency and intensity of heat waves. Given this background, it is important to understand the link between exposure to high ambient temperature in utero and the risk of congenital disorders to be able to derive adequate counter policies and preventive measures, reducing the burden of congenital disorders.

Mexico provides an ideal case to study because it is considerably affected by climate change and has experienced several strong heat waves in recent years. Likewise, the region still falls short on global targets regarding neonatal health. Within this context, indigenous populations appear particularly vulnerable. Infant mortality rates are well documented to be higher among indigenous groups compared to non-indigenous populations, indicating increased susceptibility among indigenous newborns. Jones (2019) and Sahu et al. (2022) highlight that indigenous peoples

are disproportionately threatened by climate change due to factors including strong environmental ties, socioeconomic deprivation, higher burdens of pre-existing diseases, and limited access to quality healthcare.

In this paper, we study the effects of high ambient temperature in utero on the likelihood of being diagnosed with congenital disorders at birth in Mexico. Our research questions are as follows:

1. Does high ambient temperature in utero increase congenital disorders?
2. If so, what are the mechanisms behind this impact?
3. Are there heterogeneous effects by indigenous status?

Our empirical analysis utilizes a comprehensive dataset of approximately 19 million births registered in Mexico between 2008 and 2021. We link these birth records with high-resolution temperature data measured at the locality level (the third administrative division), covering around 63,000 localities nationwide. Temperature exposure is assigned based on the mother's place of residence, allowing us to precisely measure ambient temperature during the in utero period for each newborn. To isolate the effect of heat exposure on congenital birth disorders, we exploit temporal and spatial variation in temperature across localities and gestational windows. Our regression models include municipality-specific time and locality-specific month-of-the-year fixed effects to control for unobserved, seasonal local characteristics and common temporal shocks.

Our results show that high ambient temperature in utero increases the likelihood of congenital disorders in Mexican newborns. A 1°C higher maximum temperature in utero is estimated to increase the likelihood of any congenital disorder by 0.022 percentage points, a 2.4 percent increase. The effect size is meaningful. While some behavioral risk factors (e.g., maternal age over 40, daily smoking, and alcohol consumption during pregnancy) might have stronger effects on the onset of congenital disorders (Pethő et al., 2024; Hackshaw et al., 2011; Qi et al., 2020), the magnitude of the impact of high ambient temperatures seems to be comparable to the effect of other known environmental risk factors such as air pollution (Vrijheid et al., 2010).

Focusing on subcategories of congenital disorders, we find that elevated temperature is linked to congenital disorders of the respiratory and musculoskeletal system as well as to those associated with the mouth.

Our analysis of mechanisms suggests that the effect is driven by elevated temperatures in the first trimester through both direct thermal effects and indirect pathways. Heat exposure causes placental dysfunction and increases placental permeability via inflammation and oxidative stress, impairing nutrient transfer and allowing harmful agents to reach the fetus. These biomedical vulnerabilities are further exacerbated by heat-related droughts that cause maternal undernutrition and by heightened air pollution, which—under conditions of compromised placental integrity—can more readily impact the fetal environment.

After establishing the general impact of high temperatures on congenital birth disorders, this paper compares newborns from indigenous and non-indigenous groups. Among others, this line of research is motivated by the circumstance that indigenous people in Mexico tend to live in areas that exhibit higher average temperatures. Our hypothesis is that, within the same geographic area, indigenous newborns are more vulnerable to temperature-related congenital disorders. This is based on two factors. First, heat-protection measures in Mexico are wealth-dependent (Randazzo et al., 2023), and indigenous populations are disproportionately poorer (Nun, 2020), making them less able to afford such protections. Second, indigenous communities tend to rely more on subsistence farming, resulting in greater outdoor exposure to heat and increased risk from nutritional deficiencies caused by droughts and climate disruptions. Supporting our hypothesis, and based mainly on self-identified indigenous status, our results indicate that indigenous newborns are relatively more likely to experience congenital birth disorders following exposure to high ambient temperatures. This increased vulnerability likely reflects both limited access to heat protection and greater heat exposure due to subsistence farming.

Our study contributes to a rich economic literature that investigates conditions in utero on birth outcomes and health conditions in adulthood. Several empirical studies identified climatic conditions such as severe storms, high ambient temperature, or droughts in utero as one driver behind abnormal birth weight and height, child mortality, and worse socio-economic and health conditions in adulthood (e.g., Almond and Currie, 2011; Currie and Rossin-Slater, 2013). We contribute to this literature by being able to abstain from those general health indicators (e.g., child mortality or birth weight), focusing on congenital disorders, a birth condition with particularly severe long-term consequences. Closely related to our analysis is the study by Currie and Rossin-Slater (2013) that establishes a causal link between exposure to hurricanes in utero and abnormal conditions at birth using birth records from Texas. Our study considers temperature during

pregnancy and focuses on congenital disorders. Additionally, our detailed dataset allows us to elaborate on selection effects caused by stillbirth, miscarriages, and abortion. Eventually, our study provides the first causal evidence on the effects of ambient temperature on congenital disorders.

Lastly, our study speaks to the medical and epidemiological literature investigating the determinants of congenital disorders. Heat exposure, particularly during the organogenesis (first trimester of pregnancy) has been associated with congenital disorders (Miao et al., 2021). Animal studies show that hyperthermia induces cell death that translates to malformations in numerous animal models (Benett, 2010). Maternal fever, internal heat caused by hot tubs, saunas, electrical blankets and intensive exercising have been linked with birth defects in a number of human studies, though the overall evidence is mixed (Haghighi et al., 2021). Additionally, elevated ambient temperature goes along with behavioural and environmental changes such as a higher consumption of alcohol, a higher risk of infections (vector-borne or food-borne zoonosis), and increased air pollutants that cause oxidative stress during gestation and might contribute to congenital disorders (Haghighi et al., 2021; Mio et al., 2021; Ravindra et al., 2020). We add to this literature by providing estimates that i) that rest on substantially larger sample sizes and ii) that use more stringent fixed-effect specifications, which come closer to a causal identification of the relation between ambient temperature in utero and congenital birth disorders.

2. Background

2.1 Climate Vulnerabilities

The climate of Mexico is very diverse and mostly includes areas with temperate or tropical zones. Locations north of 24° latitude exhibit lower temperatures during winter months, while areas south of this demarcation tend to experience fairly consistent temperatures all year round. The country has distinct wet and dry seasons, and most of Mexico faces a rainy season from June to mid-October. On average, the northern parts of the country receive less rain, resulting in some regions being classified as arid, such as the Northwest and the Northeast. According to the United Nations Framework Convention on Climate Change (UNFCCC), Mexico is considered particularly vulnerable to the impacts of global climate change. Identified impacts are an increasing number of heat events, droughts, flooding, and storms. Although heat events regularly occur throughout all of Mexico, some regions such as the northwestern and northeastern parts of the country are particularly likely to experience heat episodes (Schwarz et al., 2025).

The vulnerability of Mexico to global warming has been established for a long period, resulting in various policy outcomes initiatives. The country adopted the United Nations Framework Convention on Climate Change (UNFCCC) in 1992, has been a party to the Kyoto Protocol since 1998, and is a regular member of the conference of parties (COP) initiatives. Domestically, Mexico held its first Inter-Ministerial Commission on Climate Change in 2005, leading to the formulation of climate change strategies in the National Development Plan from 2007-2012. Furthermore, as Mexico is a federalist state, it should be noted that subnational governments can tailor national climate policies to their own needs, resulting in substantial variations in definitions, goals, and implementation practices across the country.

2.2 Congenital Disorders

Congenital disorders are the second leading cause of infant mortality in Mexico and a major contributor to the onset and exacerbation of disability-related reductions in physical functioning. Despite its severe health and economic impact, it has been observed that in many Latin American countries, including Mexico there is scope for more concerted policy efforts to improve surveillance, prevention measures and care in key policy areas (Zarante et al., 2021). Official statistics on the overall extent of congenital birth disorders in Mexico are regularly collected but not always published. The most recent evidence from the Mexican Registry and Epidemiological Surveillance of External Congenital Malformations (RYVEMCE) for 2016 lists that 19,767 newborns were recorded with congenital birth disorders, which is about 0.9 percent of all live births in that year (GoM, 2018).

Some types of birth disorders qualify for legal abortion practices if detected during pregnancy. Detection of congenital birth disorders in Mexico is performed as part of prenatal maternal checks and is based on different prenatal tests and ultrasound. The most common of these tests relate to NIPT (non-invasive prenatal test) and cfDNA (cell-free DNA analysis) to detect chromosomal abnormalities, amniocentesis and chorionic villus sampling to detect genetic disorders, and 12-week (20-week) ultrasound to detect serious anatomical abnormalities. Although all federal hospitals are capable of performing these checks, access to federal hospitals is more difficult for rural populations as these hospitals are exclusively located in urban centers across the country. We provide detailed information on the legal framework concerning abortion in Mexico in Appendix B.

3. Data

3.1 Sample

Our analytical sample is an individual-by-month pseudo-panel that covers any registered newborn in Mexico between the years 2008 to 2021. Combining detailed birth-related information from publicly-available administrative sources (birth registry from the Mexican Ministry of Health) with geographical information on local temperature, our sample covers 18,856,373 individual-level observations from 63,139 localities in Mexico. Climate-related data are linked to the birth registry at the local level (administrative level 3, “locality”) using geographic administrative maps from the Mexican statistical office (INEGI).”

3.2 Data Sources and Construction of Variables

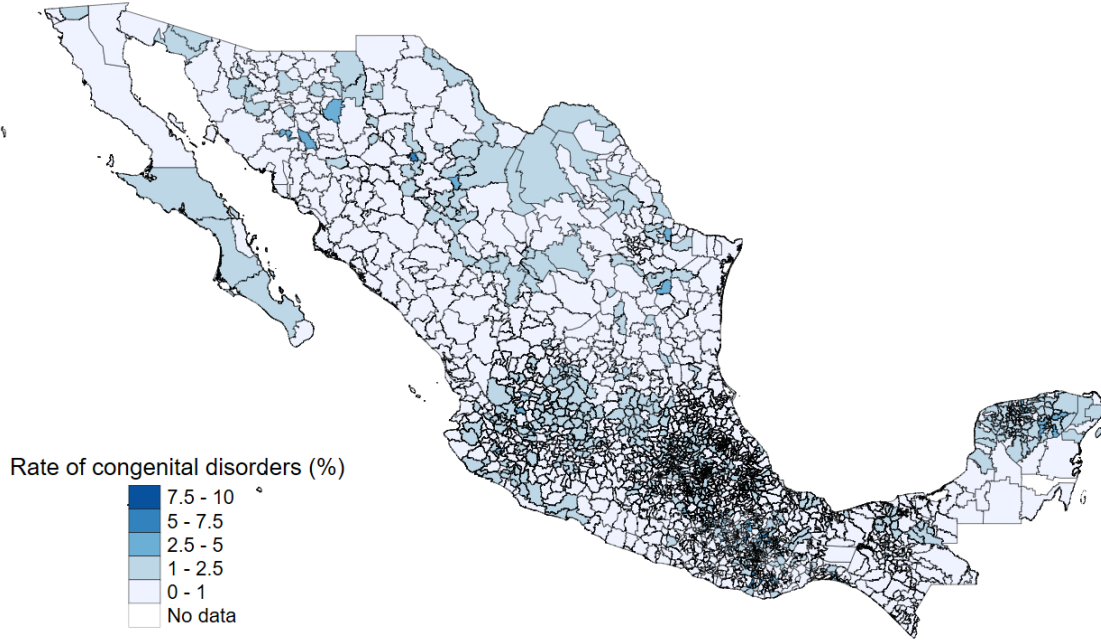
Our main dependent variable is a binary indicator (0, 100) that identifies whether a newborn had a congenital disorder at the time of birth.¹ Information on newborn health, specifically congenital disorders, is taken from the birth registers of Mexico provided by the Ministry of Health. The registers document the timing, location, and several characteristics of newborns and mothers for any registered birth in Mexico.

Several considerations are the following for our analysis. The first is the availability of information on the precise location and timing of birth, gestation period, and the place of residence of the mother (admin-3 level), which allows us to construct an exact measure of exposure to climatic conditions in utero. Second, the registers systematically report on congenital disorders using the ICD-10 classification system. We consider any diagnosis that falls into the category Q “Malformations, deformations and chromosomal abnormalities” of ICD-10 as an indication of a congenital disorder. This category includes microcephaly, spina bifidia, congenital heart defects, cleft palate, Down syndrome, among others. Furthermore, we consider category P “Conditions of the newborn originating in the perinatal phase” of ICD-10 to investigate mechanisms such as in utero infections or malnutrition and illness of the mother during pregnancy. In the data cleaning process, we dropped observations with a gestational age less than 22 weeks, as there is hardly any chance of survival for the fetus. In our sample and consistent with official government publications

¹ Please note that the binary variable deviates from the standard notation (0, 1) due to a better readability of the regression result tables.

for 2016 (GoM, 2018), around 1 percent of newborns are diagnosed with a congenital disorder. Figure 1 plots the rate of congenital disorders per municipality in our sample.

Figure 1. Rate of Congenital Disorders per Municipality



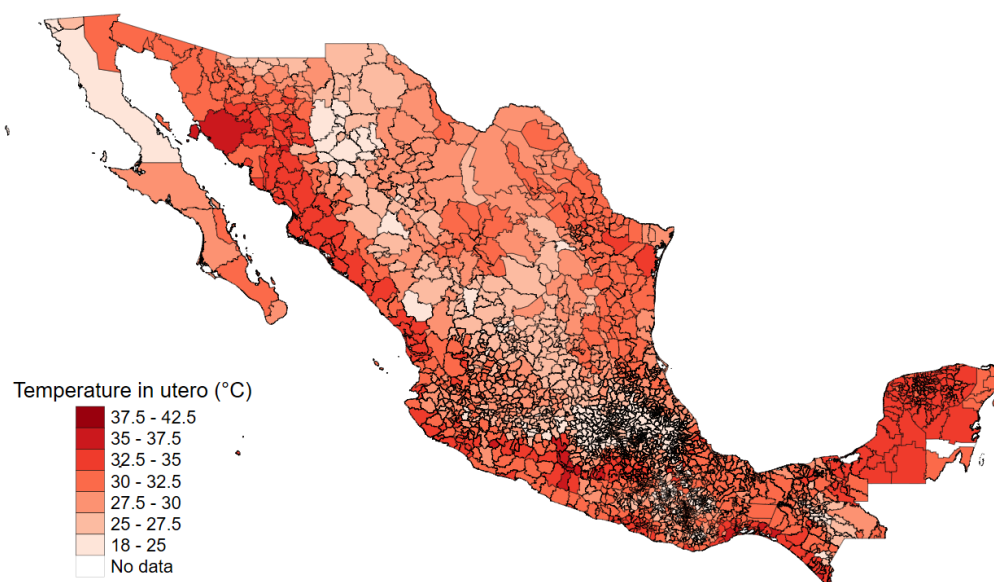
Notes: The graph reports the average rate of congenital disorder per municipality (admin-2 level) over the sample period 2008 – 2021. Source: Mexican birth registers.

Our main explanatory variable, *temperature in utero*, gives the average monthly maximum temperature to which the newborn is exposed to in utero. To construct this measure, we use monthly maximum temperature data at the 2.5 arc minute resolution level (approximately 0.04 degrees)² from the WorldCLim database (Fick and Hijmans, 2017). These data are linked to the locality of mother's residence, defined at the admin-3 level. Since multiple grid cells may fall within a single locality, we compute an area-weighted average of the temperature values. Grid cells that partially overlap with a locality are weighted according to the share of their area that falls within that locality. To capture individual heat exposure, we calculate the average of these temperature values over the period the newborn was in utero. The period is determined by subtracting the reported pregnancy months from the birth date. Figure 2 presents the distribution of the main explanatory variable: the average monthly maximum temperature during the perinatal phase. In our sample, this measure ranges from 10.41°C to 43°C with a mean of 28.27 °C.

² A grid cell of 2.5 arc minutes refers to a cell of around 5km x 5km at the equator and around 6 km in North America.

We use the average monthly maximum temperature during gestation as our primary measure of exposure to high ambient temperature because it captures both the duration of and intensity to heat exposure. As robustness checks, we consider alternative measures, namely anomalies in maximum temperature³ and the average of the monthly mean temperature during gestation. Information on average temperature comes from the GHCN CAMS Gridded 2m Temperature dataset, provided by NOAA (Fan and van den Dool, 2008), which reports monthly averages at the 0.5-degree resolution (approximately 50 km). The average temperature during gestation is 19.93°C.

Figure 2. Distribution of Temperature in Utero



Notes: The graph reports the average maximum temperature in utero in degree Celsius. The variable is aggregated to the Mexican municipality (admin-2) level and over the sample period.
Source: WorldClim database.

In addition to temperature, we also consider other climatic conditions in our robustness checks, including average monthly precipitation during gestation and the exposure to tropical cyclones in utero. Information on precipitation is taken from the WorldClim database and constructed in the same way as the temperature measure. Data on the location, timing, and influence of tropical cyclones come from Pérez-Alarcón et al. (2022). We define a binary indicator

³ Anomalies of the maximum temperature are deviations of the monthly maximum temperature from the long-term mean of that month (1960-2007) divided by its standard deviation.

that equals one if any tropical cyclone reached the mother's locality of residence during gestation. A locality is considered affected if it falls within the outer radius of a cyclone. Over the study period, 70 localities were impacted by at least one cyclone.

Besides measures of congenital disorders and exposure to climatic shocks, we construct further measures for the analysis, which we summarize in the following. A structured description of those measures can be found in Table A11 in the Appendix and descriptive statistics are reported in Table A1 in the Appendix.

First, we construct a binary indicator of each mother's indigenous status using data from the birth registers. Since information on a person's indigenous status is only available from 2015 onwards, our related analyses are restricted to a subsample (observations from the period 2015–2021). In our main specification, a mother is classified as indigenous if she self-identified as such when registering at the healthcare facility. Alternatively, we use broader definitions that classify a mother as indigenous if she either speaks an indigenous language or fulfils at least one of the two criteria. The construction of our primary indigenous status indicator (self-identification) follows the approach of Villareal (2014), who emphasizes that self-identification is the preferred method for ethnic classification in sociology and ethnology, as it allows individuals autonomy in defining their own identity.⁴

Second, we control for annual locality-level population density estimates in our regressions. These estimates are obtained from the Gridded Population of the World (GPW) dataset, version 4, provided by SEDAC. For years without direct observations, we linearly interpolate the values. In addition, we incorporate a range of environmental and socioeconomic locality characteristics including information on air pollution, measured by monthly estimates of PM2.5 concentrations derived from satellite data (Washington University in St. Louis, van Donkelaar et al., 2021), and drought exposure, captured by the Standard Precipitation and Evapotranspiration Index (SPEI) values below -1, based on the SPEIbase v2.9 dataset (SPEIbase v2.9, Vicente-Serrano et al., 2010). To approximate the risk of vector-borne infections, we use the zika suitability index from Messina

⁴ In Mexico, the self-identification question was introduced in the 2000 population census in response to national indigenous rights movements and international commitments, such as Mexico's ratification of the ILO's Indigenous and Tribal Peoples Convention in 1990. While self-identification is not without its critiques, it was intended to address limitations of the language-based approach, which can be influenced by factors such as stigma (e.g., parents avoiding the transmission of indigenous languages to protect their children from discrimination), socioeconomic status (e.g., higher-educated parents maintaining indigenous identity without language retention), and general measurement error (Villareal, 2014).

et al. (2016), which is a time-invariant measure. We also construct an indicator of the risk of domestic violence based on monthly reported domestic violence cases at the municipality level from the Mexican National Security Secretary, which are publicly available for the years 2015 to 2022. All these variables are constructed following the same approach used for our primary exposure variable—namely, averaging over the gestation period at the appropriate geographic level—except for the Zika suitability index, which does not vary over time. Finally, to address potential mortality selection, we include data on fetal deaths that come from administrative death registries maintained by the Mexican Ministry of Health and cover the years 2012-2021.

4. Empirical methodology

In order to estimate the average effect of exposure to high ambient temperature in utero on the likelihood of a congenital disorder, we exploit variations in temperature over space and time. Our econometric model regresses an indicator variable that is 100 if child i from mother m residing in locality l that is part of municipality r and is born on date d (month-year) has been diagnosed with a congenital disorder, Y_{imlrd} ,⁵ on the exposure to ambient temperature in the perinatal phase $Temp_{lrmr}$ and further controls:

$$Y_{imlrd} = \varphi Temp_{lrmr} + X'_{imlrd}\beta + \theta_{ls} + \delta_{rd} + \varepsilon_{imlrd}, \quad (1)$$

where X_{imlrd} refers to a vector of control variables including child, mother, and locality characteristics. Our baseline set of controls includes population density, maternal age, maternal educational attainment, and the gender of the newborn. We include locality-by-month fixed effects θ_{ls} , which absorb the average tendency of a given locality to experience specific temperature conditions in a particular month (e.g., January typically being cooler in locality l). In addition, we control for municipality-specific time fixed effects δ_{rd} , which capture broader temporal shocks or trends that can relate to changes in temperature and newborn health—such as climate change, policy reforms, or climate phenomena like El Niño/La Niña—at the second administrative level. Our coefficient of interest is φ , which gives the effect of average maximum temperature in utero on the likelihood of the newborn being diagnosed with a congenital disorder. ε_{imlrd} represents

⁵ Please note that our outcome variable is a binary variable that takes the two values of 0 and 100. We deviate from the standard notation (0 and 1) due to a better readability of the coefficients in the tables.

standard errors that are clustered at the locality level to account for within-locality correlation over time.

The identifying variation in our empirical approach stems from deviations in temperature during gestation from the average in a given month and locality within municipalities over time.⁶ We assume that, conditional on the included fixed effects and control variables, local temperature conditions are effectively as good as randomly assigned. This identification strategy follows previous literature (e.g., Rocha and Soares, 2015). In robustness checks, we confirm that our results remain stable when varying the measurement of ambient temperature, adjusting the correction of standard errors, and accounting for other potential confounders such as birth facility characteristics or other climatic conditions.

Our estimation results could be biased if individuals were to time their fertility to avoid hotter months or migrate to cooler locations during pregnancy. However, our use of locality-by-month fixed effects accounts for any seasonal patterns in fertility timing. Moreover, since medium-term local temperature fluctuations are largely unpredictable and moving is costly, it is unlikely that individuals systematically migrate based on anticipated temperature conditions during pregnancy.

In addition to our main empirical analysis, we investigate potential mechanisms underlying the observed relationship and examine heterogeneous effects by indigenous status. The mechanisms are analyzed in two ways: i) by employing alternative outcome variables within the main regression framework, and ii) by estimating heterogeneous treatment effects based on locality characteristics. To assess whether there are differences by indigenous status, we estimate the following regression model:

$$Y_{imlrd} = \varphi Temp_{lmd} + \pi Ind_{imlrd} + \eta(Ind_{imlrd} \times Temp_{lmd}) + X'_{imlrd} \beta + \theta_{ls} + \delta_{rd} + \varepsilon_{imlrd}. \quad (2)$$

Ind_{imlrd} is a binary indicator that equals one if the mother self-identifies as indigenous. The coefficient π captures the average difference in the likelihood of congenital disorders between indigenous and non-indigenous newborns, while η gives the differential effect of temperature

⁶ The variation that we exploit is shown Figures A1 and A2 in the Appendix. Figure A1 presents the distribution of residuals and panel A in Figure A2 illustrates the relation of residuals relative to the fitted values of our main regression model.

during gestation on the likelihood of a congenital disorders for indigenous newborns compared to their non-indigenous counterparts, conditional on exposure.

5. Results

5.1 Main Results

Table 1 presents our main results. Column 1 controls for year, location, and month fixed effects, in column 2 we add basic characteristics of the child, mother, and the locality to the regression. Location fixed effects control for time-invariant local factors that may be correlated with both temperature and congenital disorders, while year fixed effects account for temporal changes—such as improvements in the diagnosis of congenital disorders—that could coincide with rising temperatures due to climate change. Columns 3 to 5 step-wise adjust the fixed effects specification to be more restrictive. In column 3 we utilize year, location, and municipality-month fixed effects to capture regional seasonal patterns in temperature and newborn health. Column 4 replaces the separate year and month fixed effects with year-month fixed effects to control more precisely for time trends. Column 5, our preferred specification, incorporates municipality-date fixed effects in combination with locality-month fixed effects. This specification accounts for both local seasonality and broader regional shocks, such as monthly variation in the healthcare system or in the political environment.

Starting with results from column 1, we find no statistically significant relation between the average maximum monthly temperature in utero and the likelihood of a congenital disorder among newborns. The inclusion of basic socio-economic characteristics (column 2) does not alter our main finding. The coefficients on the control variables align with existing literature: female newborns are, on average, less likely to have a congenital disorder, and the incidence of congenital disorders increases with maternal age. When we include municipality-month fixed effects in the regression model (column 3), our main coefficient of interest becomes statistically significant and rises to 0.021 ppt. We argue that municipality-month fixed effects are essential for identifying a causal relationship, as they address endogeneity concerns related to seasonality. For example, pesticides, which are known to be associated with higher rates of congenital disorders, are typically applied during the growing season, which might coincide with periods of elevated temperature. In such cases, the observed increase in congenital disorders could mistakenly be attributed to temperature rather than pesticide exposure. Similarly, seasonal variation in the reporting or

detection of congenital disorders could bias the results. By incorporating municipality-month fixed effects, we mitigate these potential confounders.

In our preferred specification (column 5), our coefficient of interest is 0.022 percentage points, indicating that a 1°C (or 1 standard deviation (3.74°C)) increase in the average maximum temperature during gestation raises the rate of congenital disorder by 0.022 (0.08) percentage points. This corresponds to an approximate increase of 2.4 (8.8) percent relative to the mean incidence rate (0.904 percent), or roughly 474 (1749) additional cases per year.

Table 1. Temperature in Utero and Congenital Disorders

	Congenital disorder				
	(1)	(2)	(3)	(4)	(5)
Temperature in utero	0.006 (0.005)	0.006 (0.005)	0.021*** (0.007)	0.021*** (0.008)	0.022*** (0.005)
Population density		-0.046 (0.100)	-0.046 (0.100)	-0.046 (0.099)	-0.590* (0.341)
Female		-0.242*** (0.010)	-0.243*** (0.010)	-0.243*** (0.010)	-0.244*** (0.010)
Age of mother		0.011*** (0.001)	0.011*** (0.001)	0.011*** (0.001)	0.011*** (0.001)
No education		0.034* (0.018)	0.035** (0.018)	0.035** (0.018)	0.034** (0.018)
Primary education		-0.012 (0.014)	-0.012 (0.014)	-0.012 (0.014)	-0.019 (0.015)
Tertiary education		-0.029** (0.012)	-0.029** (0.012)	-0.029** (0.012)	-0.033*** (0.013)
Year FE	Yes	Yes	Yes		
Locality FE	Yes	Yes	Yes	Yes	
Month FE	Yes	Yes			
Municipality-month FE			Yes	Yes	
Locality-month FE					Yes
Date FE				Yes	
Municipality-date FE					Yes
Mean of dependent	0.904	0.904	0.904	0.904	0.904
Observations	18,856,373	18,856,373	18,856,373	18,856,373	18,856,373

Notes: The table reports OLS coefficient estimates and standard errors of the regression of any congenital disorder on the average maximum monthly temperature in utero and further controls. Fixed effects are included in the regression as indicated in the table. Standard errors are clustered at the locality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

To better understand whether specific types of congenital disorders are driving our results, we re-estimate our preferred specification (column 5) using ICD-10 sub-category indicators as dependent variables. The results, shown in Table A2 in the Appendix, indicate that exposure to high temperature in utero is particularly associated with an increased incidence of congenital

disorders affecting the mouth (column 5), as well as the respiratory (column 4) and musculoskeletal systems (column 9).

5.2 Robustness Checks

We further examine the sensitivity of our main regression results to i) alternative birth outcomes, ii) alternative temperature measures, iii) variations in the correction of standard errors, iv) potential confounders, v) placebo checks and vi) mortality-related selection effects. We summarize the findings in the following. The regression results tables are available in Appendix A.

First, we explore alternative birth outcomes. Table A3 shows that higher average maximum temperature in utero is not only associated with higher likelihood of congenital disorders but also with adverse perinatal outcomes. Specifically, we observe lower birth weights, lower APGAR scores, higher Silverman index scores, and an increased incidence of preterm births.

Second, we assess the sensitivity of our results to alternative temperature concepts (Table A4). In particular, we consider temperature anomalies—defined as deviations from long-term (1960-2007) locality-level means—and average monthly temperature instead of maximum temperature. Both alternative specifications yield positive and statistically significant effects, supporting a robust link between in utero heat exposure and congenital health risks, though magnitudes vary with the measure employed.

Third, we test whether our inference is sensitive to the level at which standard errors are clustered. While our baseline approach is standard errors clustered at the locality level, we additionally cluster at the year-locality, municipality, and state levels (Table A5). Across all specifications, our main result remains statistically significant, demonstrating robustness to alternative clustering strategies.

Fourth, we consider potential confounding factors. In Table A6, we examine whether other climatic events—specifically precipitation and cyclone in utero exposure—confound the relationship between temperature and congenital disorders. Neither variable shows a statistically significant effect. Table A7 further tests robustness to maternal characteristics (e.g., marital status, prenatal care, parity, indigenous status), gestational age, facility-specific factors, and additional weather conditions (humidity, wind). The inclusion of these variables does not meaningfully affect the magnitude or significance of our main coefficient, with the exception of gestational age, suggesting a mediating role of earlier birth in the temperature-congenital disorder relationship.

Fifth, we implement several placebo tests to validate our identifying assumption. As shown in Table A8, temperature exposure during non-relevant periods (11–13 months before birth or 1–3 months after birth) shows no significant association with congenital disorders. Moreover, average minimum temperatures, which may correlate with maximum temperatures, do not predict congenital disorders, ruling out measurement bias from this channel.

Sixth, we address potential endogeneity concerns arising from the definition of our exposure variable, which is based on the actual gestational period. If shorter gestations are associated with higher temperature and risk of congenital disorders, this could bias our results. To examine this, we instrument for in utero temperature using the average maximum temperature over the nine months prior to birth (Table A9). The IV results are statistically insignificant. In addition, we compare the relation between fitted values and residuals of the main regression model to the one using locality-date fixed effects that only captures variation coming from differences in the duration of gestation. The comparison shows that the variation is considerably reduced in the latter case. Taken together, while we cannot rule out endogeneity concerns from gestational age completely, the limited variation that is left after taking locality-date fixed effects into account suggests that it is unlikely driving the full effect.

Lastly, we explore the possibility of selective mortality. If fetuses with congenital disorders are more likely to result in miscarriage or abortion under high-temperature conditions, our baseline estimates would be downward biased. Using fetal death registry data (2012–2021), we find that higher in utero temperatures significantly increase the risk of fetal death, particularly among cases diagnosed with congenital disorders (Table A10). This suggests that our main results likely represent conservative estimates of the true impact of heat exposure.

5.3 Mechanisms

Heat exposure may affect newborn health through both direct and indirect pathways. The direct channel is supported by evidence from animal models and medical studies showing that elevated maternal body temperature during the first trimester can disrupt gene expression and induce cell death during organogenesis, leading to congenital disorders (Benett, 2010; Haghighi et al., 2021). In addition, thermal stress can impair placental function by triggering inflammation, oxidative stress, and impaired vascularization, which reduce nutrient and oxygen transfer to the fetus and increase the risk of developmental anomalies. Placental barrier integrity may also be compromised,

increasing permeability and allowing harmful substances to reach the fetal environment (Ramirez et al. 2024).

The indirect channel involves four interrelated factors that emerge later in pregnancy or act through environmental and social stressors, often amplifying the underlying biomedical vulnerabilities. First, heat-related droughts can impair agricultural yields and deplete water resources, leading to economic hardship, maternal stress, and undernutrition—all of which disrupt placental development and efficiency, limiting fetal access to nutrients and oxygen. Second, elevated temperatures increase the risk of infections such as Zika, rubella, and toxoplasmosis, which can cross a heat-compromised placental barrier more readily, or trigger damaging maternal immune responses (Rostami et al., 2021; Tesla et al., 2018). Third, heat intensifies air pollution by boosting ozone formation and trapping fine particulate matter (PM_{2.5}) near the ground (Zhou et al., 2023; Stingone et al., 2019; Liu et al., 2016); when placental permeability is elevated due to heat-induced stress, these pollutants are more likely to enter the fetal environment. Fourth, extreme heat can increase psychosocial stress, raise maternal cortisol levels, and heighten the incidence of intimate partner violence and child maltreatment—all of which are associated with impaired placental function and poor pregnancy outcomes (Currie and Rossin-Slater, 2013; Persson and Rossin-Slater, 2018; Gu and Guan, 2021; Sanz-Barbero et al., 2018; Evans et al., 2025).

First, we focus on the direct channel, by examining temperature conditions in each pregnancy trimester separately and regressing our primary outcome of interest on these variables. Table 2 presents the results, where column 1 reports the findings for the average monthly maximum temperature in the first trimester, column 2 for the second trimester, column 3 for the third trimester, and column 4 includes the temperature measures jointly. Our analysis shows that elevated temperatures during the first trimester increase the likelihood of congenital disorders. We find no statistically significant relation between temperature and congenital disorders at later stages of pregnancy, which is consistent with the medical and biological literature. We interpret this finding as providing evidence in support of the direct channel of heat on congenital disorders. In terms of magnitude, a 1°C increase in the average monthly maximum temperature during the first trimester is associated with a 0.01 percentage point increase in the rate of congenital disorders.

Table 2. Congenital Disorders and Temperature during Pregnancy Trimesters

	Congenital disorder			
	(1)	(2)	(3)	(4)
Temperature during 1 st trimester	0.013*** (0.003)			0.010*** (0.003)
Temperature during 2 nd trimester		0.002 (0.003)		0.003 (0.003)
Temperature during 3 ^d trimester			-0.000 (0.004)	0.000 (0.004)
Municipality-date FE	Yes	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes
Observations	18,856,373	18,856,373	18,607,694	18,607,694

Notes: The table reports OLS coefficient estimates and standard errors of regressing the indicator variable identifying congenital disorders on the average maximum monthly temperature during the pregnancy trimesters. Fixed effects as indicated in the table. Standard errors are clustered at the locality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

To examine the proposed indirect mechanisms (droughts, infections, air pollution, domestic violence), we analyze each of them separately (Table 3). Column 1 shows that a higher number of drought months in utero is associated with a lower risk of congenital disorders, however, an increased number of drought months enhances the effect of temperature on congenital disorders. Column 2 presents no evidence of a differential effect between localities suitable for Zika transmission and other areas. In column 3, we find that higher temperatures in utero coupled with increased air pollution have a stronger effect on the onset of congenital birth disorders. Given the influence of rainfall on air pollution levels, we control for the average monthly precipitation in utero.

Table 3. Heterogeneity Analysis by Locality Characteristics

	Congenital disorder			
	(1)	(2)	(3)	(4)
Temperature in utero	0.023*** (0.006)	0.024*** (0.006)	0.027*** (0.006)	0.034** (0.013)
Drought in utero	-0.732*** (0.074)			
Temperature in utero x drought in utero	0.006** (0.002)			
Temperature in utero x zika		-0.005 (0.005)		
PM2.5 in utero			-0.302*** (0.117)	
Temperature in utero x PM2.5 in utero			0.011*** (0.004)	
Precipitation in utero			0.005 (0.004)	
Violence in utero				0.240 (0.165)
Temperature in utero x violence in utero				-0.009 (0.006)
Municipality-date FE	Yes	Yes	Yes	
Locality-month FE	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes
Municipality FE				Yes
Date FE				Yes
Observations	18,856,373	16,799,087	18,851,481	7,142,773

Notes: The table reports OLS coefficient estimates and standard errors of congenital disorders on temperature in utero and interactions with locality characteristics. *Drought in utero* gives the number of drought months in utero. A drought month is defined by a SPEI index value below -1. *PM2.5 in utero* gives the average particulate matter density of size 2.5 μm during utero, and *zika* is a time-invariant indicator ranging from 0 to 1 that captures the climatic suitability of a locality for *Aedes* mosquitos, which transmit the Zika virus. *Violence in utero* gives the number of reported cases of domestic violence in the municipality during the period the newborn is in utero. For the latter analysis, the sample is reduced to the years 2015-2022. All explanatory variables are standardized. Fixed effects as indicated in the table. Standard errors are clustered at the locality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Finally, column 4 shows no statistically significant evidence for domestic violence as an important mechanism. However, it is important to note that information on domestic violence is only available for the years 2015-2021 and at the municipality level. Hence, this analysis is conducted with a smaller sample and less restrictive fixed effects.

To complement the analysis of indirect mechanisms, we conduct additional analyses at the individual level. The birth registers enable us to identify specific child health conditions that originate in the perinatal phase and provide information on maternal health during pregnancy. Our analysis proceeds in two steps. First, we estimate the effect of average maximum temperature on these child and maternal health conditions. Second, we derive correlations between each respective

health condition and our child-specific indicator of congenital disorders. In principle, the first step sheds light on the question whether high ambient temperatures lead to certain health outcomes, while the second step generates suggestive evidence whether the health condition might matter for the onset of a congenital disorder. The respective results are shown in Table 4. We find that higher temperatures in utero increase the incidence of malnutrition and respiratory and circulatory diseases among newborns (panel A). Furthermore, these factors are correlated with a higher likelihood of congenital disorders (panel B). We speculate that these results are largely in line with the drought and the air pollution channel. Regarding the former, maternal and fetal malnutrition can plausibly be attributed to droughts while maternal and fetal respiratory and cardiovascular conditions in the perinatal phase can be consequences of air pollution. In contrast, the additional results (Table 4) provide no support in favor of the other three channels (infections, maternal stress as proxied by maternal hypertensive disorders, and domestic violence as proxied by physical injuries).

Table 4. Congenital Disorders, Temperature, and Birth Conditions of Mother and Child

Panel A: Temperature in utero and maternal and fetal health								
	Mother Malnutri- tion	Mother infect	Mother resp. circ.	Mother Hyperten- sion	Mother injury	Child Malnutri- tion	Child infect	Child resp. circ.
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Temperature in utero	-0.000 (0.000)	-0.000 (0.000)	0.000** (0.000)	0.004 (0.003)	0.001 (0.001)	0.002** (0.001)	0.002 (0.002)	0.034*** (0.007)
Panel B: Maternal and fetal health and congenital disorders								
	Congenital disorder							
Explanatory factor	0.000 (0.006)	-0.004 (0.003)	-0.003 (0.007)	0.004*** (0.001)	0.001 (0.002)	0.005* (0.003)	0.003** (0.001)	0.002** (0.001)
Municipality-date FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Observations	18,856,373	18,856,373	18,856,373	18,856,373	18,856,373	18,856,373	18,856,373	18,856,373

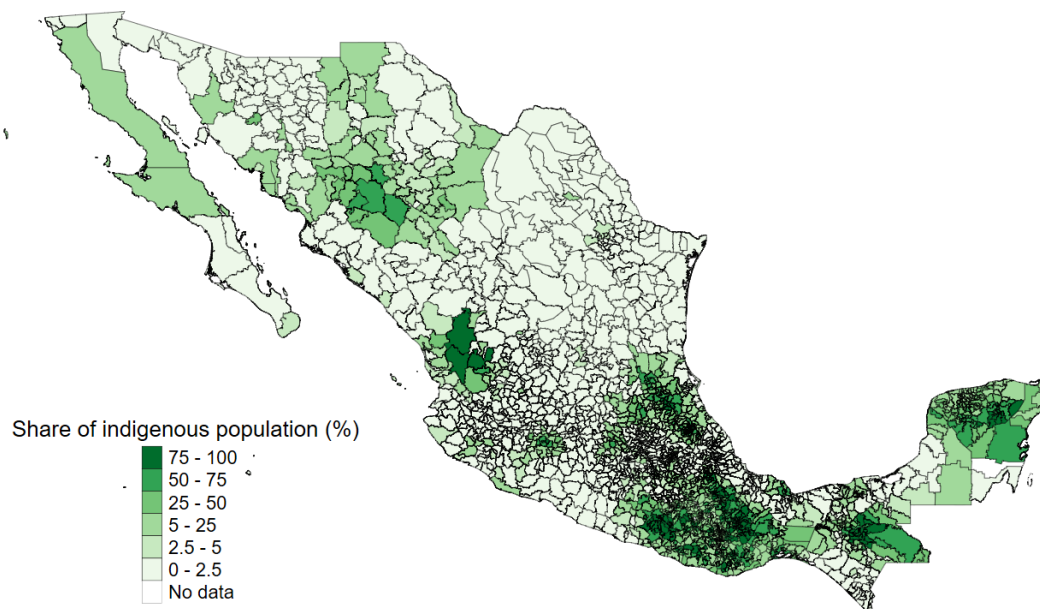
Notes: The table reports OLS coefficient estimates and standard errors of temperature in utero on several birth conditions in the perinatal phase in Panel A and of the regression of those conditions on congenital disorders at birth in Panel B. Birth conditions are classified using the sub-categories of the ICD-10 category “P.” “Mother infection” refers to infectious diseases of the mother during pregnancy (P00.2), “Mother resp. circ.” captures respiratory and cardiovascular diseases of the mother (P00.3); “Mother malnutrition” refers to malnutrition of the mother that harms the fetus (P00.4). “Mother hypertension” refers to hypertensive disorders (P00.0) and “mother injury” to physical injuries of the mother (P00.5). Additionally, we consider diagnosis of the fetus related to the perinatal phase: “Child infection” captures diagnosis of fetus infections (P35-P39); “Child resp. circ.” refers to respiratory and cardiovascular conditions originating in the perinatal phase (P20-P29); “Child malnutrition” refers to diagnosis that fall into category “P052.” Standard errors clustered at the locality level. Fixed effects and standard controls are included in all regressions. * p < 0.1, ** p < 0.05, *** p < 0.01.

5.4 Heterogenous Effects by Indigenous Status

In this section, we focus on the indigenous population. We first explore descriptive differences between indigenous and non-indigenous population groups, before estimating heterogenous treatment effects as outlined in equation (2).

Turning to descriptive patterns, Mexico is home to one of the largest and most diverse indigenous populations in the world. Approximately 20 percent of the population self-identify as indigenous. As illustrated in Figure 3, the majority of the indigenous population is concentrated in the southern and south-central regions of the country.

Figure 3. Share of Indigenous Population per Municipality



Notes: The figure reports the share of indigenous persons per municipality.

Source: Mexican birth registries.

Table 5 compares indigenous and non-indigenous individuals in our analytical sample (2015-2021) across basic socio-economic characteristics, as well as our key dependent variable (congenital disorders) and independent variable (average maximum temperature in utero). The table reports mean values and the standard errors in brackets by sub-sample and the corresponding p-values from the Kruskal-Wallis test. The results indicate several notable differences between the two subgroups:

First, while differences in newborn health are present, they are not particularly pronounced. Notably, newborns of indigenous mothers are somewhat less likely to be diagnosed with a

congenital birth defect, which, however, could be due to underreporting, selection bias or differences in the access to healthcare. Second, indigenous mothers are more likely to reside in rural and less developed localities, as proxied by night-time light intensity and average educational attainment. Third, access to healthcare seems to be more limited for indigenous mothers, as evidenced by fewer prenatal care visits. Finally, indigenous mothers are more frequently exposed to extreme climatic conditions, particularly higher ambient temperatures during pregnancy.

Table 5. Descriptive Statistics of Indigenous and Non-indigenous Newborns

	Non-indigenous	Indigenous	Kruskal-Wallis test (p-value)
	(1)	(2)	(3)
Congenital disorder	0.990 (9.900)	0.917 (9.530)	0.002
Any birth defect	4.218 (20.100)	3.781 (19.073)	0.00
Birth condition (perinatal)	3.253 (0.18)	2.887 (0.17)	0.00
APGAR Sore	8.895 (0.774)	8.809 (1.058)	0.00
Birth weight	3134.873 (468.500)	3119.296 (432.845)	0.00
Gestational age (weeks)	38.640 (1.744)	38.976 (1.576)	0.00
Urban	0.854 (0.353)	0.545 (0.498)	0.00
Population density (ihs)	8.007 (1.711)	6.270 (1.738)	0.00
Night-time light (ihs)	4.340 (0.811)	3.339 (1.040)	0.00
Age of mother	25.759 (6.311)	25.502 (6.462)	0.00
No formal education	0.041 (0.197)	0.144 (0.351)	0.00
Primary education	0.041 (0.199)	0.053 (0.223)	0.00
Secondary education	0.390 (0.488)	0.362 (0.481)	0.00
Tertiary education	0.433 (0.495)	0.229 (0.284)	0.00
Mother employed	0.238 (0.426)	0.089 (0.284)	0.00
Temperature in utero	28.550 (3.609)	28.570 (4.132)	0.00
Temperature during 1st trimester	28.378 (4.348)	28.570 (4.505)	0.00
Precipitation in utero	7.297 (4.913)	11.041 (6.657)	0.00
Cyclone in utero	0.001 (0.022)	0.000 (0.009)	1.00
Mother is health insured	0.792 (0.406)	0.806 (0.396)	0.00

Turning to the heterogenous treatment effects analysis, Table 6 presents the differential effect of temperature in utero on congenital disorders for indigenous, considering that the definition of “being indigenous” commonly includes self-identification and/or language proficiency. Focusing on our preferred specifications—which consistently define indigeneity based on self-identification (Columns 1 and 3)—we find robust evidence that newborns of indigenous mothers are more vulnerable to congenital birth disorders when exposed to elevated ambient temperatures during pregnancy. In terms of magnitude, a 1°C increase in temperature in utero raises the likelihood of a congenital disorder by 0.025 percentage points for non-indigenous newborns and by 0.034 percentage points for indigenous newborns. When using language-based definitions of indigeneity (Columns 2 and 4), we observe no statistically significant differential effect. Our regression models control for maternal education as a proxy for socioeconomic status, as well as locality-specific effects. Therefore, the results likely capture unobserved lifestyle differences among Indigenous populations, such as increased heat exposure from subsistence farming or limited access to heat protection measures.

Table 6. Heterogeneity Analysis by Indigenous Status

Definition of indigenous	Congenital disorder			
	Self-identified	Language or self-identified	Language and self-identified	Language
	(1)	(2)	(3)	(4)
Temperature in utero	0.025*** (0.008)	0.026*** (0.008)	0.024*** (0.008)	0.026*** (0.008)
Indigenous	-0.195 (0.155)	0.230 (0.187)	-0.187 (0.158)	0.226 (0.183)
Temperature in utero x indigenous	0.009* (0.005)	-0.005 (0.006)	0.009* (0.005)	-0.005 (0.006)
Municipality-date FE	Yes	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes
Observations	10,049,980	10,035,571	10,061,558	10,061,558

Notes: Table reports OLS coefficient estimates and standard errors of the main regression specification and heterogeneous effects with an indicator variable identifying the indigenous status of the mother. A newborn is defined as indigenous if the mother reports to consider herself as indigenous or if she can speak an indigenous language or combinations thereof. Standard errors are clustered at the locality level. Fixed effects as indicated in the table. Sample is restricted to the years 2015 - 2021, for which information on indigenous status is available.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

6. Discussion and Conclusion

This study examines the causal impact of high ambient temperatures during pregnancy on the incidence of congenital disorders in Mexico, using data on over 18 million births from 2008 to 2021. By linking detailed birth register information with localized temperature data based on maternal residence, and applying a high-dimensional fixed effects regression model, we isolate the effect of in utero heat exposure from confounding time and location-specific factors. We find that a 1°C increase in average monthly maximum temperature during gestation leads to a 2.4 percent increase (0.022 percentage points) in the likelihood of congenital disorders diagnosed at birth. Our estimates likely represent a lower bound, given two important considerations: i) some congenital disorders are diagnosed postnatally and therefore not captured in our dataset, and ii) selective mortality may bias results downward. To ensure the robustness of our findings, we examine alternative model specifications and address potential sources of bias.

Furthermore, we provide empirical evidence supporting both direct and indirect mechanisms through which elevated temperatures affect fetal health. First, in line with the direct channel, we find that temperature increases during the first trimester—when organogenesis occurs—have the strongest impact on the incidence of congenital disorders, consistent with medical and biological research. Second, regarding the indirect channel, our results indicate that episodes of drought and elevated air pollution significantly exacerbate the effects of high ambient temperatures. These interactions are plausibly explained by heat-induced placental dysfunction and increased placental permeability, which heighten fetal vulnerability to environmental and physiological stressors.

Lastly, our analysis reveals that indigenous mothers in Mexico are more frequently exposed to high in utero temperatures but report slightly lower diagnosis rates of congenital disorders, likely due to limited healthcare access. Moreover, indigenous newborns are significantly more vulnerable to temperature-related congenital disorders.

Finally, we acknowledge two empirical identification challenges of our study that relate to potential endogeneity concerns with respect to migration and gestational age. Due to data limitations, we cannot adjust our estimates for migration behavior. However, in contrast to acute climate events like hurricanes, high temperatures are less likely to prompt relocation, making substantial bias from temperature-induced migration unlikely. Furthermore, any migration would need to be systematically related to the fetus's health status to bias our results, which seems

implausible. Regarding gestational age, we show that most of the temperature variation we exploit arises from cross-locality differences rather than timing differences, suggesting minimal bias from this source.

We believe that the findings of this study have important implications for public policy, particularly in the areas of maternal health and climate adaptation. Given the clear evidence that high ambient temperatures during pregnancy—especially during the first trimester—significantly increase the likelihood for the onset of congenital disorders, maternal health programs should be revised to explicitly incorporate climate-related risks. Prenatal care protocols can be updated to include guidance on avoiding heat exposure, and heat advisories can be tailored to address the specific vulnerabilities of pregnant women. In regions prone to extreme heat, particularly rural and low-income areas, expanding access to cooling infrastructure—such as shaded public spaces, fans, and air conditioning—would serve as a preventive health measure.

Our results also highlight deep-seated structural inequalities, particularly affecting indigenous populations. Indigenous mothers are more exposed to extreme heat, yet their children are less likely to be diagnosed with congenital conditions at birth, likely due to limited access to healthcare and diagnostic services. This underlines the urgent need to invest in healthcare infrastructure in indigenous and underserved communities to ensure equitable access to diagnostic tools and early interventions. In addition, the indirect pathways we identify—such as heat-related air pollution and drought-induced malnutrition or maternal stress—point to the importance of strengthening social safety nets. Policies that provide nutritional assistance or financial support to pregnant women during climate shocks can mitigate these risk factors.

More broadly, our findings suggest that environmental health must become a central pillar of climate adaptation strategies. As the climate crisis intensifies, understanding and addressing its impact on vulnerable populations, including unborn children, is not only a health imperative but also a matter of social equity. Public awareness campaigns targeting at-risk communities can further empower individuals to adopt protective behaviors during periods of extreme heat.

In sum, this study contributes to the growing body of evidence on the health consequences of climate change, highlighting a previously underexplored pathway through which heat exposure affects human development. Addressing these challenges will require coordinated action across health, environmental, and social policy domains to ensure that vulnerable populations—particularly pregnant women and indigenous communities—are protected in a warming world.

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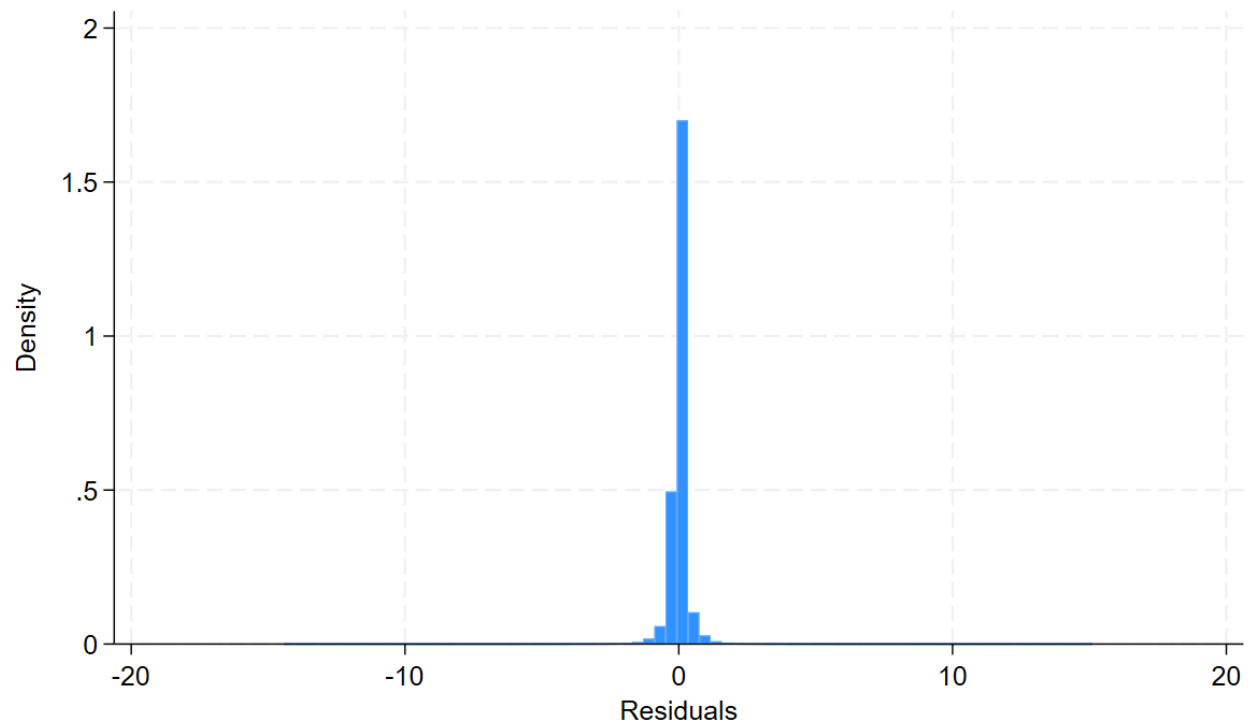
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Appendix

Appendix A. Robustness Checks and Further Analyses

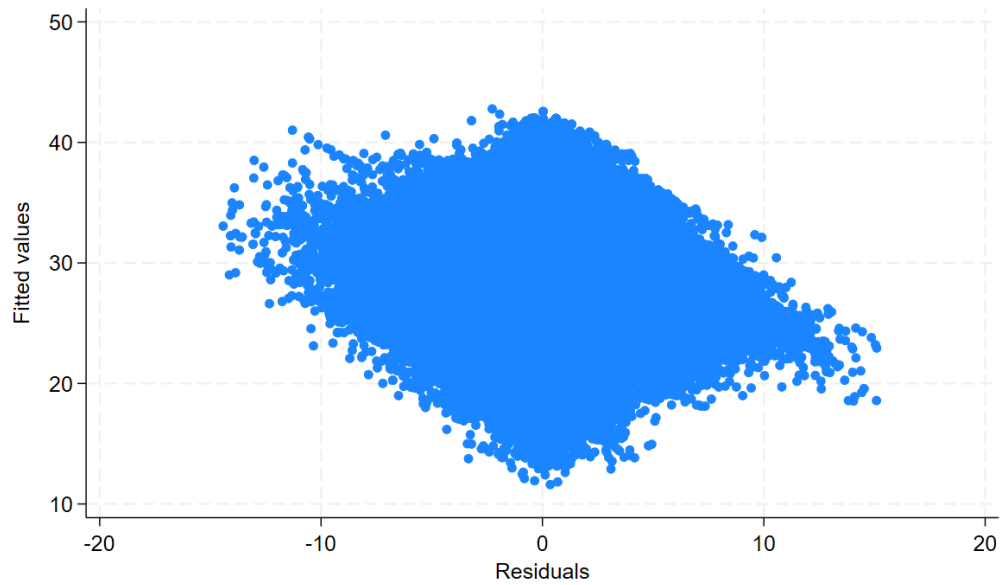
Figure A1. Histogram of Residuals



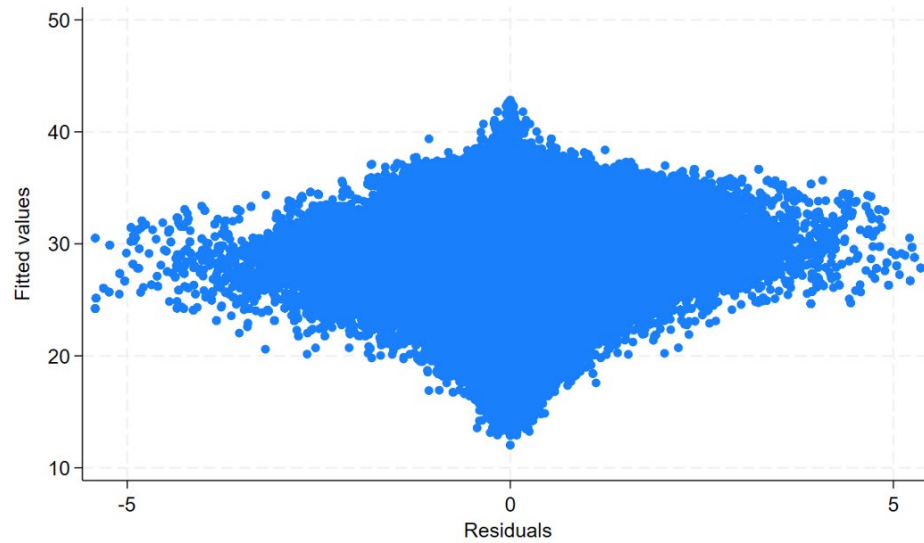
Notes: The graph plots the density of the residuals from the regression of average maximum temperature in utero on the fixed effects and standard controls of the main regression specification as shown in equation (1).

Figure A2. Residual Plots

Panel A: Residual plot from regression with municipality-date and locality-month fixed effects



Panel B: Residual plot from regression with locality-date fixed effects



Notes: The figure reports the residuals from the regression of average maximum temperature in utero on the fixed effects and standard controls relative to the fitted values. In graph a, municipality-date and locality-month fixed effects are incorporated into the regression, whereas in graph b locality-date fixed effects are included in the model.

Table A1. Descriptive Statistics

	Mean	SD	Min.	Max.	Obs.
Congenital disorder	0.90	9.47	0.00	100	18856373
Any birth condition	3.62	18.68	0.00	100	18856373
Birth condition originating in perinatal phase	2.72	16.27	0.00	100	18856373
Apgar index	8.88	0.83	0.00	10	18708354
Silverman sore	0.23	0.99	0.00	10	18634152
Height at birth (cm)	49.88	2.70	7.00	84	18467993
Birth weight (g)	3146.46	468.00	22.00	8700	17882765
Female	0.49	0.50	0.00	1	18856373
Week of pregnancy	38.73	1.72	22.00	49	18856373
Age of mother	25.50	6.31	13.00	62	18856373
No education	0.07	0.25	0.00	1	18856373
Primary education	0.05	0.23	0.00	1	18856373
Secondary education	0.39	0.49	0.00	1	18856373
Tertiary education	0.37	0.48	0.00	1	18856373
Indigenous (self-identification)	0.07	0.25	0.00	1	10126622
Indigenous (language-based)	0.05	0.22	0.00	1	10112440
No. of dead children born	0.17	0.48	0.00	25	18733929
No. of children born alive	2.05	1.21	0.00	25	18839081
Mother health insured	0.89	0.32	0.00	1	18856373
Received prenatal care	0.98	0.15	0.00	1	18752719
Urban	0.82	0.39	0.00	1	18856373
IHS(population)	7.78	1.82	0.00	12	18856373
IHS(nightlight)	4.21	0.95	0.00	5	18856373
Suitability of zika infection (index)	0.12	0.24	0.00	1	16800233
Temperature in utero (°C)	28.27	3.74	10.41	43	18856373
Avg. temperature in utero	19.93	5.46	5.73	41	18856373
Temperature anomaly in utero	0.13	0.18	-4.48	5	18856373
Precipitation in utero (cm)	7.56	5.34	0.00	119	18854321
Cyclone in utero	0.00	0.02	0.00	1	18856373
PM2.5 in utero	17.69	5.60	1.53	79	18853762
Drought in utero	1.75	1.89	0.00	10	18856373
Temperature during 1 st trimester	28.13	4.45	9.46	43	18856373
Temperature during 2 nd trimester	28.35	4.37	10.01	43	18856373
Temperature during 3 ^d trimester	28.32	4.61	9.00	44	18610044
Temperature 11-13 months before birth	28.22	4.47	9.98	43	18856368
Temperature 1-3 months after birth	28.15	4.41	9.98	43	18723917

Table A2. Temperature in Utero and Types of Congenital Disorder

	nerv e (1)	sense (2)	circ (3)	resp (4)	mout h (5)	digesti on (6)	genit al (7)	urina ry (8)	muscle (9)	chromos on (10)	other (11)	alcoh ol (12)
Temp. in utero	0.000 (0.00 2)	0.001 (0.00 2)	0.000 (0.00 1)	0.001 * (0.00 0)	0.004 ** (0.002)	0.002 (0.001)	0.001 (0.00 2)	0.001 (0.001)	0.009* ** (0.003)	0.000 (0.000)	0.001 (0.00 1)	0.000 (0.00 1)

Notes: The table reports OLS coefficient estimates and standard errors of temperature in utero on several types of congenital disorders. Types are classified using the sub-categories of the ICD-10 category “Q” and refer to congenital disorders. “Nerve” reports congenital disorders of the nervous system (Q00-Q09); “Sense” refers to congenital disorders of eye, ear, face, and neck (Q10-Q19); “Circ” refers to congenital malformations of the circulatory system (Q20-Q29); “resp” captures congenital disorders of the respiratory system (Q30-34); “mouth” refers to congenital disorders associated with the mouth (Q35-37); “digestion” refers to congenital disorder of the digestive system (Q38-Q45); “genital” captures congenital malformations of the genital organs (Q50-Q56); “urinary” of the urinary system (Q60-Q63); “muscle” of the musculoskeletal system (Q65-Q79); “chromoson” refers to chromosomal abnormalities not elsewhere classified (Q90-Q99); “other” refers to other congenital disorders (Q80-Q88); and “alcohol” refers to the diagnosis of fetal alcohol syndrome (Q86). Regressions include municipality-date and locality-month fixed effects as well as the standard control variables. Number of observations is 18,856,373. Standard errors clustered at the locality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A3. Temperature in Utero and Other Birth Outcomes

	Any birth condition (1)	Birth cond. perinatal (2)	APGAR (3)	Silverman (4)	Birth weight (5)	Gestational age (6)
Temperature in utero	0.147*** (0.017)	0.125*** (0.016)	-0.007*** (0.001)	0.007*** (0.001)	-15.228*** (1.066)	-0.099*** (0.007)
Municipality-date FE	Yes	Yes	Yes	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes	Yes	Yes
Observations	18,856,373	18,856,373	18,705,455	18,628,838	17,868,937	18,856,373

Notes: Table reports coefficient estimates and standard errors of the main regression model using alternative indicators of newborn health. Any birth condition refers to any diagnosis that falls into ICD-10 category “Q” or “P,” “Birth condition perinatal” indicates that the newborn has been diagnosed with a birth condition that originates in the perinatal phase. Standard errors clustered at the locality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A4. Variation of Temperature Measures

	Congenital disorder	
	(1)	(2)
Temperature anomaly in utero	0.073*** (0.025)	
Avg. temperature in utero		0.423*** (0.026)
Min. monthly temp. in utero		
Municipality-date FE	Yes	Yes
Locality-month FE	Yes	Yes
Controls	Yes	Yes
Observations	18,856,257	18,856,257

Notes: The table reports OLS coefficient estimates and standard errors of alternative temperature measures on congenital disorders. The main regression specification is used. Standard errors are clustered at the locality level. Avg. max. temp. anomaly are standardized anomalies of the average maximum monthly temperature in utero; average temperature refers to the average temperature over the period in utero and min. monthly temperature in utero gives the minimum value of temperature in utero. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A5. Variation of Clustered Standard Errors

Dependent: Level of se cluster:	Congenital disorder		
	Locality and year SE	Municipality SE	State SE
	(1)	(2)	(3)
Temperature in utero	0.022*** (0.006)	0.022*** (0.006)	0.022*** (0.006)
Municipality-date FE	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes
Controls	Yes	Yes	Yes
Observations	18,856,373	18,856,373	18,856,373

Notes: The table reports OLS coefficient estimates and standard errors of the main regression specification with alternative specifications of clustered standard errors. Column 1 uses standard errors that are clustered at the locality and year level, column 2 at the municipality level and column 3 at the state level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A6. Other Climatic Conditions and Congenital Disorders

	Congenital disorder		
	(1)	(2)	(3)
Precipitation in utero	0.006 (0.004)		0.005 (0.004)
Cyclone in utero		0.023 (0.200)	-0.004 (0.204)
Temperature in utero			0.021*** (0.005)
Municipality-date FE	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes
Controls	Yes	Yes	Yes
Observations	18,854,144	18,856,373	18,854,144

Notes: Fixed effects as indicated in the table. Standard errors are clustered at the locality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A7. Controlling for Additional Confounders

		Congenital disorder		
	(1)	(2)	(3)	(4)
Temperature in utero	0.024*** (0.008)	0.022*** (0.005)	0.021*** (0.005)	0.018*** (0.006)
Municipality-date FE	Yes	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes	Yes
Basic controls	Yes	Yes	Yes	Yes
Mother controls	Yes			
Locality-specific time trend		Yes		
Birth facility FE			Yes	
Weather controls				Yes
Observations	9,760,248	18,856,373	17,482,381	18,856,373

Notes: The table reports OLS coefficient estimates and standard errors of the main regression specification including additional controls and fixed effects as indicated in the table. Mother controls include marital status, no. of children alive and dead, no. of pregnancies, type of residence and residence area. Weather controls include average wind direction in utero and its speed as well as humidity in utero. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A8: Placebo checks

		Congenital disorder			
	(1)	(2)	(3)	(4)	(5)
Temp. in 11-13 months before birth	-0.004 (0.007)		-0.001 (0.007)	-0.001 (0.007)	
Temperature in utero	0.023*** (0.006)	0.031*** (0.007)	0.032*** (0.008)		0.022*** (0.005)
Temp. in months 1-3 after birth		-0.011 (0.007)	-0.011 (0.007)	-0.004 (0.007)	
Temperature during 1 st trimester				0.012*** (0.003)	
Temperature during 2 nd trimester				0.005 (0.003)	
Temperature during 3 ^d trimester				0.002 (0.005)	
Min. temperature in utero					-0.075 (0.082)
Municipality-date FE	Yes	Yes	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes	Yes
Observations	18,856,368	18,721,871	18,721,866	18,475,261	18,856,373

Notes: Table reports coefficient estimates and standard errors of temperature in utero and two placebo checks, namely 11-13 months before birth and 1-3 months after birth. A further check is shown in column 5 with the average of the monthly minimum temperature in utero. Standard errors clustered at the locality level. Fixed effects as indicated in the table. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A9. Instrumental Variable (IV) Approach

	Congenital disorder (1)
Temperature in utero	0.010 (0.006)
Municipality-date FE	Yes
Locality-month FE	Yes
Controls	Yes
First-stage KP F-statistics	14,098,593
Observations	18,856,373

Notes: The table reports coefficient estimates and standard errors of a 2SLS regression. Temperature in utero is instrumented by the average maximum temperature during the 9 months before birth.

Table A10. Selective Mortality

	Fetal death (1)	Stillbirth (2)	Miscarriage (3)	Abortion (4)	Fetal death (5)
Temperature in utero	0.477*** (0.155)	0.145*** (0.048)	0.168*** (0.054)	0.003*** (0.001)	0.226*** (0.072)
Congenital disorder					-45.055*** (11.089)
Temperature in utero x congenital disorder					1.827*** (0.419)
Municipality-date FE	Yes	Yes	Yes	Yes	Yes
Locality-month FE	Yes	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes	Yes
Observations	14,731,742	14,731,742	14,726,131	14,726,131	14,658,631

Notes: The table reports OLS coefficient estimates and standard errors of average maximum temperature on fetal death and sub-categories. Fetal death is an indicator variable identifying any registered death before or at birth. Stillbirth refers to deaths after gestational age of 28th week, whereas miscarriage captures fetal death beforehand. Abortion refers to a provoked fetal death. The sample includes all registered birth and fetal deaths. Sample is restricted to the years 2012 - 2021 for which information on fetal death is available. Standard errors are clustered at the locality level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A11. Variables Description

Variable	Description and Source
Temperature in utero	Average value of monthly max temperature at locality of mother residence during the in utero period. Source: WorldClim.
Congenital disorder	Takes 100 if newborn has congenital disorder and 0 otherwise. Congenital disorder diagnosed with ICD-10 code Q. Source: Birth register.
Types of congenital disorders	Indicator variables for types of congenital disorders (ICD-10 Q subcategories). Source: Birth register.
Female	Dummy = 1 if newborn is female. Source: Birth register.
Age of mother	Age of the mother in years. Source: Birth register.
No education	Indicator = 1 if mother has no educational degree. Source: Birth register.
Primary education	Indicator = 1 if mother completed primary education. Source: Birth register.
Tertiary education	Indicator = 1 if mother completed tertiary education. Source: Birth register.
Population density	Inverse hyperbolic sine of population density. Source: SEDAC (GPW v4).
Temperature during 1st,2nd, 3d trimester	Average value of monthly maximum temperature at locality of mother residence during the respective trimester. Source: WorldClim, birth register.
Mother malnutrition	Mother malnourished during pregnancy/birth (ICD-10 P00.4). Source: Birth register.
Mother infection	Mother diagnosed with infection (ICD-10 P00.2). Source: Birth register.
Mother resp. circ.	Mother diagnosed with respiratory/circulatory disease (ICD-10 P00.3). Source: Birth register.
Child malnutrition	Child malnourished (ICD-10 P052). Source: Birth register.
Child infection	Child infection (ICD-10 P35-P39). Source: Birth register.
Child resp. circ.	Child respiratory/circulatory disease (ICD-10 P20-P29). Source: Birth register.
Drought in utero	Months with drought during in utero. Drought is defined by SPEI < -1. Source: SPEIbase.
Zika	Zika transmission suitability index. Source: Messina et al. (2016).
PM25 in utero	Average PM2.5 density during in utero. Source: van Donkelaar et al. (2021).
Precipitation in utero	Average monthly precipitation during in utero. Source: WorldClim.
Any birth condition	Indicator = 100 if any diagnosis in Q or P (ICD-10). Source: Birth register.
Birth cond. perinatal	Indicator = 100 if any condition in P (ICD-10). Source: Birth register.
APGAR	Health condition index at birth (1–10). Source: Birth register.
Silverman	Respiratory distress index (0–10). Source: Birth register.
Birth weight	Weight at birth in grams. Source: Birth register.
Gestational age	Pregnancy weeks at birth. Source: Birth register.
Avg. temperature in utero	Average of the average monthly temperature during gestation (°C). Source: GHCN CAMS.
Min. temperature in utero	Average of the minimum monthly temperature during in utero. Source: WorldClim.
Foetal death	Indicator = 100 if foetus born dead. Source: Foetal death register.
Stillbirth	Indicator = 100 if death after 20 weeks. Source: Foetal death register.
Abortion	Indicator = 100 if foetus was aborted. Source: Foetal death register.
Cyclone in utero	Indicator = 1 if cyclone exposure during in utero. Source: Pérez et al. (2022).
Indigenous	Indicator = 1 if mother is indigenous (self-identification or language-based). Source: Birth register.

Appendix B. Background Information

Abortion: Birth itself might be endogenous to both ambient temperature and the likelihood of a congenital disorder of the newborn, e.g., via stillbirths or abortions. In this context we briefly review the legal abortion context in Mexico.

The legal aspects of abortion in Mexico are centered on a federal penal code from 1931, which made abortion punishable with one to six years of imprisonment for both the pregnant woman and the abortion practitioner (e.g., a midwife). According to this law penalties were waived only if the pregnancy resulted from rape or if the abortion resulted from negligent behavior on the part of the pregnant woman. Due to Mexico's federal structure, the 1931 federal penal code had no direct implications for state-level legislation and abortion practices but served as a model for state penal codes. Bearing in mind that state-level penal codes were adjusted multiple times, all 31 states waived punishments for abortion in the case of rape. Additionally, and focusing on the 2000s and 2010s punishments were waived in some other cases, too, which strongly depended on the specific state legislation. For instance, punishment for abortion as a result of negligent behavior on the part of a pregnant woman was waived in 29 states, while punishment was waived in the case of the abortion saving the life of the pregnant woman in 27 states. Likewise, punishment was waived if the abortion helped protect the health of the pregnant woman in 10 states and if the fetus had serious malformations in 13 states.

Traditionally, elective abortions were prohibited according to both the national and state-level legislation. In the year 2007 the federal district of Mexico City decriminalized and effectively allowed for elective abortions in the first twelve weeks of pregnancy. In 2021 the Mexican Supreme Court ruled that generally penalizing abortion is unconstitutional across the country. Consequently, states were asked to revise their legislation. Until February 2025, about 70 percent of all states (22 out of 31) had adjusted their legislation to allow for elective abortions in the first 12 weeks of pregnancy, the exceptions being the following nine states: Chihuahua, Durango, Guanajuato, Morelos, Sonora, Tabasco, Tamaulipas, Tlaxcala, and Queretaro.

Though certain types of abortion practices have been decriminalized at the state and federal level, access to professional abortion services is often still limited to institutions run by the federal Ministry of Health. Though federal hospitals are spread out through the different Mexican states, they are placed in urban areas, therefore making access to abortion services more expensive and more difficult to access for rural residents. Furthermore, and bearing in mind that elective abortions

were still criminalized over the last 20 years throughout all 31 Mexican states, official statistics on abortions are known to substantially underreport abortion practices. For the year 2006 a study from El Colegio de México and the Guttmacher Institute estimated that about 875,000 abortions were carried out annually in Mexico (Guttmacher, 2008), while a more recent study puts this number to 1,050,000 abortions for the year 2019 (Bearak et al., 2022). Unfortunately, abortion statistics in Mexico do not yet allow for a more disaggregated analysis by type of abortion.