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ORIGINAL PAPER

The effects of an epidemic on prenatal investments, childhood mortality and health of surviving children

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Abstract

The potential death toll from an epidemic is larger than the number of deaths directly associated with the infection. In this study, we find that prenatal exposure to a cholera epidemic in Peru increased childhood mortality and that surviving children were more likely to be underweight and to suffer from diarrhea. We further find that a significant part of this mortality happened during the first day of life, and that prenatal exposure to cholera decreased prenatal care and institutional deliveries, suggesting that the mortality and possibly other longer-term effects were partially driven by a reduction in prenatal investments.

Keywords Childhood mortality · Parental investments · Epidemics · In utero · Prenatal care · Institutional deliveries · Infectious diseases · Diarrheal diseases · Cholera

JEL Classification I10 · J10 · O10

1 Introduction

Epidemics have been on the rise for the past several decades and will likely continue to do so in the near future. The potential death toll attributable to an epidemic is considerably larger than the number of deaths directly associated with the infection. Children exposed to an epidemic while in utero are particularly vulnerable; infected

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pregnant women can transmit the disease through the placenta, and even if the virus or bacteria does not affect the fetus directly, symptoms of the disease, such as dehydration, high fever or stress can affect the fetus in ways that are not visible until after the child is born or even later in life. Additionally, the economic hardship that an epidemic imposes to pregnant women and their households, and the potential saturation of the healthcare capacity can affect parental investments, including maternal nutrition, vaccines and skilled care during pregnancy and during birth. We know from a number of studies in epidemiology and economics that in utero is a critical developmental period and that shocks during this period, including epidemics, have long-term consequences (Almond and Currie 2011). We know less about their effect on postnatal childhood mortality and the potential mediating role of prenatal investments. In this study, we aim to contribute to this literature by exploring the effects of prenatal exposure to the Peruvian cholera epidemic in the early 1990s.

There have been seven cholera pandemics in the last 200 years. The seventh began in the early 1960s in Asia and spread through Africa and Europe in the early 1970s. Cholera rates remained relatively low during the 1980s and were confined to Asia and Africa, but in the early 1990s an outbreak began in Peru that became the first cholera epidemic in Latin America since 1895 (Lam et al. 2010). Peru suffered the most severe outbreak of cholera of the region and the health system collapsed in many regions of the country (Cueto 2017). With a large majority of the population working informally, without access to medical paid-leave, the epidemic also generated important economic consequences for households (Suárez and Bradford 1993).

From an empirical perspective, studying the cholera epidemic in Peru is convenient because it began unexpectedly. "Disease control experts are largely at a loss to explain why cholera has returned to South America in the first epidemic since 1895" (Brookes, 1991, p. 3). The bacteria seemed to originate from the Pacific Ocean; the first cases spread from the coastal cities of Peru to other regions of the country, and later to other countries as well. The abruptness and severity of the epidemic, as well as the speed of contagion, provide us with a nearly ideal natural experiment. In April 1991, a professor from the University of Maryland School of Medicine commented about the cholera epidemic in Peru, "It's pure guesswork as to where it is going" (Brookes 1991, p. 4). In retrospect, it seems that the spread of the disease over different regions of the country over time, in particular during the first year of the epidemic, was largely determined by each region's geographical proximity to the initial cases. Within a region contagion was more likely under unsanitary conditions, but not even households with access to piped water at home were safe from contagion because water was not sufficiently chlorinated (Cueto 2017).

We apply a difference-in-difference methodology exploiting the spread of the cholera epidemic in Peru by month and region. Our data comes mainly from two sources: the Population Census of 1993, in which mothers were asked the birth date of their youngest child born alive and whether or not that child was alive at the time of the interview; and the Health and Demographic Survey of 1992, which includes information of childhood mortality and parental investments, and allows us also to control for mother fixed effects.

We find that an increase in 1 standard deviation in the incidence of cholera during the last trimester in utero increases average childhood mortality rate by 0.05 percentage points, or 4%, according to our most conservative estimate. The event study shows that this effect was not driven by a pre-existing trend. We also find that later mortality is concentrated in girls and that surviving children are more likely to suffer from severe diarrhea, and have lower weight for age, and are more likely to be underweight. Finally, we find that the epidemic increased first-day mortality and reduced prenatal care and institutional deliveries, suggesting the mortality, and possibly, other longer-term effects were partially driven by a reduction in prenatal investments.

There is a well-established literature that shows that in utero is a particular sensitive stage in life for the formation of human capital. Most of this literature focuses on long-term effects of prenatal exposure to a variety of shocks (Almond and Currie 2011). The seminal study of Almond (2006) finds that in utero exposure to the 1918 influenza pandemic had long-term negative effects in terms of education attainment, physical disability, income and socioeconomic status. Closely related to our study, Ogasawara and Inoue (2018) and Ritter (2020) find negative long-term effects of prenatal exposure to the cholera epidemic among adults in Japan and Peru, respectively. A relatively smaller number of studies have shown that in utero shocks may have an effect on postnatal childhood mortality. In particular, it has been shown that exposure to pollution (Jayachandran 2009), extreme rainfall (Rocha and Soares 2015), extreme heat (Banerjee and Maharaj 2020), and violent conflict (Dagnelie et al. 2018) increases infant mortality[1]. This paper aims to contribute to this literature by analyzing the effect of prenatal exposure to an epidemic on childhood mortality. Our results are particularly relevant in a context where official reports of deaths due to epidemics in many countries differ considerably from estimations of the number of excess deaths (FT 2020).[2] These estimations, however, do not disentangle what proportion of the excess deaths is due exclusively to underreporting of direct deaths from that attributable to indirect deaths. According to our estimations, the total number of children who died due to exposure to the cholera epidemic while in their last trimester in utero was approximately 900, while the official number of total cholera deaths for 1991 is 2,909. This means that the human cost of the epidemic was approximately 30% larger if we consider the effect of prenatal exposure on postnatal childhood mortality. Moreover, the official mortality rate from the cholera epidemic in Peru, during 1991, is 9 in every 100,000 inhabitants, while our estimated mortality rate due to prenatal exposure is 150 in every 100,000 children that were born alive in 1991.

With this paper we also aim to contribute to the literature by providing evidence of the potential role of prenatal investments, in particular prenatal care and institutional deliveries, as mediators between the in utero shock and the effect on childhood mortality and possibly longer-term human capital outcomes. Prenatal care is important to prevent and treat conditions that might affect the mother and/or the fetus, but it is also

[1] Other important studies that investigate *postnatal* early childhood shocks on mortality rate are included in the review of Almond et al. (2018).

[2] As of July 2020, Peru has more than 1,000 excess deaths per million inhabitants, the highest rate in the world, which largely exceed reported numbers of Covid-19 deaths (FT 2020).

important because it can affect maternal behavior such as smoking, alcohol consumption, nutrition and weight gain, folic acid intake, among others (see, for example, Evans and Lien, 2005) and these behaviors have been shown to affect birth outcomes and have long-term human capital outcomes (Aizer and Currie 2014; Nilsson 2017; Simon 2016; Bharadwaj et al. 2014; Barreca 2010). Institutional deliveries typically involve skilled professionals with adequate equipments during and immediately after birth, which are important because they can perform C-sections, when needed, resuscitate newborns at birth and treat maternal infections, among other things. According to the World Health Organization, approximately 40% of all under-five child deaths are neonatal deaths, and approximately half of all neonatal deaths occur after a home birth and without any healthcare.

There is also more rigorous evidence of the impact of institutional births and access to healthcare on birth outcomes and infant lives (Currie and Gruber 1996; Maitra 2004; Daysal et al. 2015; Godlonton and Okeke 2016; Goodman-Bacon 2018; Adam et al. 2018; Okeke and Chari 2018; Friedman and Keats 2019b, a). Moreover, there is evidence that skilled care during and immediately after birth can treat prematurity, underweight, and other conditions affecting newborns with proper care such as artificial lung surfactant and specialized nutritional supplements that has long-term consequences on human capital (Bharadwaj et al. 2013). Epidemics have the potential to affect access to prenatal care and institutional deliveries; this has been seen, for example, in the case of the Ebola epidemic in West Africa (Jones et al. 2016; Strong and Schwartz 2019), although this evidence comes from correlational studies.[3] The Ebola epidemic is probably an extreme case, given the high probability of getting infected in hospitals in addition to the fear of getting tested and isolated (Strong and Schwartz 2019; WHO 2014). In the case of COVID-19, there is evidence that the lockdown in India increased mortality rate by 64% among dialysis patients. The main channels in this case appear to be barriers to transportation and disruption in hospital services (Jain and Dupas 2020).

In this study, we show that even epidemics involving less contagious diseases, and that do not impose a need for social isolation, such as the cholera epidemic, can generate a reduction in prenatal care and institutional deliveries. Moreover, many other shocks that have been analyzed in the fetal origins literature (droughts, wars, famines and hurricanes among other things) have the potential to affect access to skilled care during pregnancy and birth; hence, the results of this paper can apply to a wide range of shocks.

Finally, this paper speaks to the literature on diarrheal diseases, childhood mortality and malnutrition. Although we have found that the cholera epidemic reduced prenatal investment, we cannot discard the potential direct role of the disease in our mortality estimates and on the health status of the surviving children. The World Health Organization estimates that each year diarrheal diseases such as cholera kill around half a million children under five and are the leading cause of malnutrition.

[3] On a positive note, Christensen et al. (2020) show that certain interventions focused on enhancing health worker performance led to improvements in maternal utilization of clinics during the Ebola crisis in rural Sierra Leone.

Typically, these estimates come from contemporaneous effects of these diseases, and, hence, most programs target children after they are born. This study suggests, however, that protecting children from these diseases after they are born might already be too late.

2 Background: cholera and the cholera epidemic in Peru

Cholera is one of the most common diarrheal diseases, along with *Rotavirus*, *E. coli*, *Shigella*, *Campylobacter* and *Salmonella* (WHO 2009). It is generated by a bacterial infection. The main effect is acute diarrhea, and all the other consequences are believed to derive from this main effect (Seas and Gotuzzo 2005). Profuse diarrhea causes dehydration; pathogens damage the intestines, causing an excessive amount of water and other nutrient-rich fluids to be secreted rather than being absorbed. Among pregnant women, particularly during the third trimester, severe dehydration is associated with a reduction of amniotic fluid, reduced blood flood to the placenta, placental hemorrhage, hypotension, preterm births and miscarriages (Grados and Battilana 1994; Grout et al. 2015; Hirschhorn et al. 1969; Ciglenecki et al. 2013).

Over the last 200 years, there have been 7 cholera pandemics. The last pandemic still lingers in many countries. As of 2012, approximately 1.4 billion people were at risk for cholera and approximately 2.8 million cholera cases occur annually in countries where it is endemic, while approximately 87,000 cholera cases occur in non-endemic countries (Ali et al. 2012). In terms of deaths, approximately 91,000 and 2,500 people are reported to die annually of cholera in endemic countries and non-endemic countries, respectively; these numbers do not include estimations of the impact on mortality derived by in utero exposure to the disease. Today, approximately 126 million of individuals live in hotspots with a prevalence of cholera higher than 1/1000 (WHO 2020). Most of these hotspots are located in African countries and the rest are located in the Middle East and Central Asia, where the healthcare capacity and quality is very poor. Moreover, the spread of cholera around the world is likely to increase due to global warming (Chowdhury et al. 2017; Shope 1991; Emch et al. 2008).

The cholera epidemic in Peru started in the early 1990s, in the middle of an economic crisis. Real GDP per capita fell circa 30% between 1987 and 1990 (Paxson and Schady 2005). The situation of the health sector was also precarious. Individuals were supposed to have access to free healthcare, including prenatal services, labor and delivery. In practice however, the capacity of the health sector was very limited. The public expenditure in health in 1991 was 0.4% of the GDP, one of the lowest from Latin America at the time. Many health centers did not function normally because of a lack of personnel (Pollarolo 2000). The travel costs for many individuals were significantly high, given the long distances to the closest health center, some times as far as 6 or more hours away (INEI 1991). In addition, there were important opportunity costs, in particular for informal workers (the large majority of workers) for whom a day without working was a day without earnings.

Naturally, a health system like this was not prepared to deal with an epidemic. Many hospitals simply collapsed, and the probability of a patient being rejected due

to lack of capacity or personnel increased significantly. Here are some quotes that reflect this situation:

> "... hospitals received a growing number of patients that was sometimes four times greater than the number of bed and staff available. In several cities, staff had to be taken from other hospital services to look after cholera, some hospitals focused only on patients stricken with the disease, and the physicians requested the help of one relative per patient. According to one physician from Cajamarca hospital, during the epidemic "all doctors had to forget their original specialty"." Cueto (2017, p. 117)
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> "The emergency room of the hospital ... looked like a battlefield. Sick patients barely fit in the suffocating room. Some of them were seated ted on the floor, other lying in beds and others, many, were left to lie on the floor, [...]. The few nurses and auxiliary personnel extraordinarily called were insufficient to assist the patients. Several nuns, with their white robes, joined the effort to attend patients. In some cases, even police officers became spontaneously improvised nurses" (our translation from LaRepública (1991, p. 14) IN: Valdivia Rey (2014)).

The lack of capacity to attend patients during the cholera epidemic was very generalized; according to a nationally representative survey applied in October and November 1991: only 50% of people who were sick and wanted to get attention got access to health services (Pollarolo 2000). This evidence illustrates how the cholera epidemic increased the costs of getting access to healthcare in that it eroded the already limited capacity of the health sector, increasing the patients' waiting hours and the probability of being rejected. It is not surprising, therefore, that we find that the cholera epidemic reduced prenatal care and institutional births.

The epidemic reached 322,562 suspected cases and a total of 2,909 deaths (a mortality rate of 9 per 100 000 inhabitants and 0.9 per 100 cases) in the first year (1991) and a total of 625,259 accumulated suspected cases and 9,642 accumulated deaths by September 1994. By the end of 1991, the disease had spread to fourteen countries in Latin America and the Caribbean, totaling 366,017 cases, with Peru responsible for 83% of all cases presented in the Americas (Mújica et al. 2013). The epidemic was caused by the bacterium Vibrium Colérico zero group 01 (Lanata 1989). The means of contagion is through food handling and consumption, with drinking water being the most common vehicle of dissemination (Gotuzzo 1991; Maguiña Vargas et al. 2010). Working age adults were disproportionally affected by the disease, and becoming infected with cholera typically resulted in medical expenses as well as 1 to 4 days without working (Suárez and Bradford 1993).

Figure 1 shows that the largest number of cases was reported in 1991, the number decreasing over time, with a peak of approximately 0.16 per 100 inhabitants per week in the regions of the jungle. The mortality rate was also much higher in 1991, as we can see in Fig. 2.

As explained in the Introduction, disease control experts could not explain why cholera returned to South America in 1991, nor could they predict where it was going (Brookes 1991). In retrospect, however, several factors seemed to influence the intensity of the disease by region and time. The first of these is the geographical location

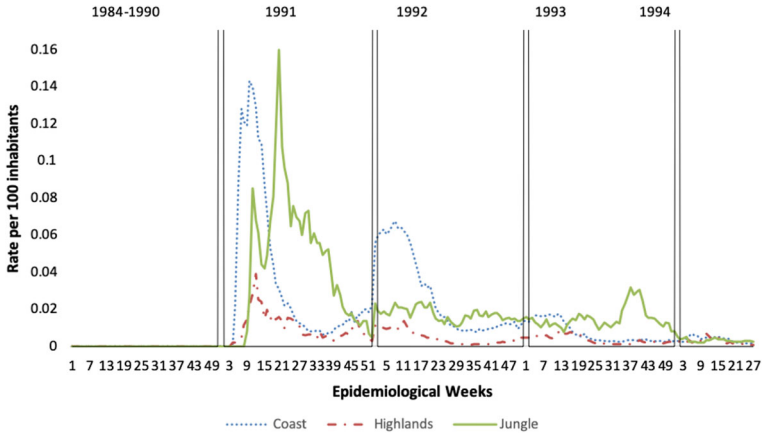

Fig. 1 Weekly incidence of cholera epidemic. Data source: General Directorate of Epidemiology, of the Ministry of Health of Peru

and time of the first case. The disease was first reported in the areas around the coastal cities of Chancay, Chimbote, and Piura at the end of January 1991. Epidemiological studies suggest that the massive contamination of phytoplankton and seafood in the warm part of the sea in the North of Lima explains the explosive spread of the diseases (Gotuzzo 1991; Maguiña Vargas et al. 2010). In general, cholera is more likely to develop in coastal ecologies (Collins 2003). Importantly, this is believed to be the first case, not just the first diagnosis since adults and children had been tested for some time before 1991 without any sign of the bacterium *Vibrium Cholerae* (Gotuzzo 1991). From these zones, the epidemic advanced to other urban and rural areas of Peru, spreading from coast to the highlands, finally penetrating the Peruvian jungle, as we can see in Fig. 3.

The second factor that seemed to influence the spread of the disease is warm temperatures and humidity (Lama et al. 2004). As we can see in Fig. 1 the disease peaked

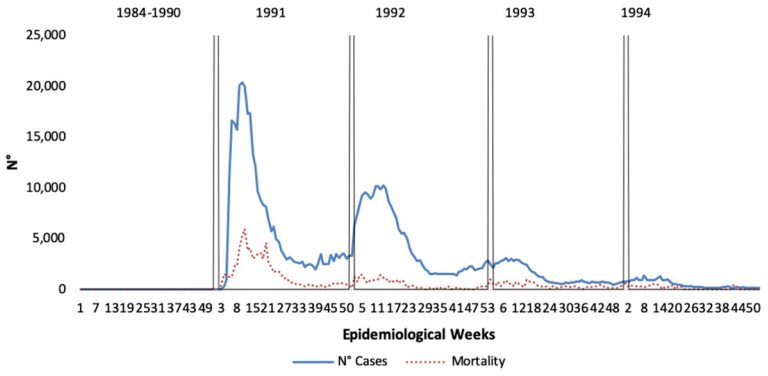

Fig. 2 Incidence and mortality of cholera epidemic. Data source: General Directorate of Epidemiology, of the Ministry of Health of Peru

in the first months of the year, when it is summer on the coast; it spread more homogeneously over the year in the jungle, where it is warm and humid all year around, and the incidence of cholera in the highlands is lower, which is colder and drier all year around.

Naturally, poor levels of basic sanitation, with inadequate processing of drinking water also seems to have influenced the expansion of the epidemic (Gotuzzo 1991; Maguiña Vargas et al. 2010). Water, even piped water, was commonly not (or not sufficiently) chlorinated and low water pressure and continuous breakdowns in the service forced people to store water in containers where contaminations was even more likely. Untreated sewages emptied into the sea, rivers and lakes or used to irrigate agricultural land (Cueto 2017).

Finally, belonging to the human blood group O has been associated with increased severity of the disease, and approximately 90% of the indigenous population in Peru is blood type O (Matson et al. 1966). Individuals with blood type O are not more likely to be infected by the bacterium, but the cholera toxin exerts a more potent effect on cells expressing the blood type O-associated glycan (Harris et al. 2005). The relationship between blood type O and the severity of the disease has been confirmed in very different settings, and in the particular case of Peru in 1991 it was found that

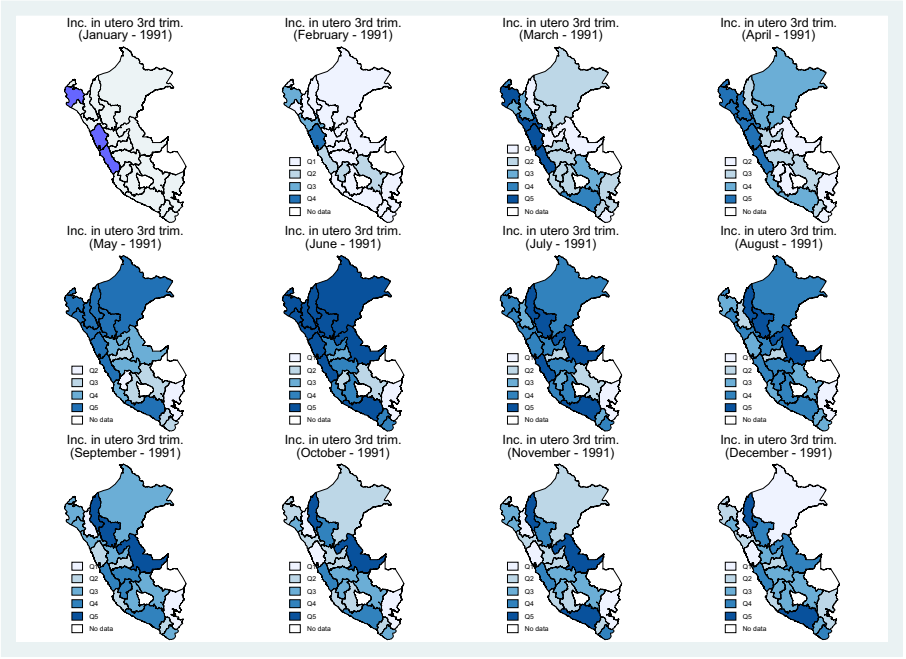

Fig. 3 Spread of the Cholera Epidemic in Peru by Month and Region Note: The graph from January shades the departments where the first cases where detected. The graphs from the rest of the months show the raw variation we exploit in our regressions, that is, the three-month cholera incidence by region and month. Different shades represent different quintiles of incidence

individuals with blood type O were eight times more likely to be hospitalized with severe cholera (Swerdlow et al. 1994).

3 Data and summary statistics

Our data make use of four sources of information. The first source of data is the Population Census of Peru of 1993 (INEI 1993), which asks women age 12 or older whether their youngest child born alive is still alive. It also asks the birth date (month and year) of that child. The mortality rate corresponds to the proportion of those children by cohort and region who have died. We restrict our sample initially to 10 years of cohorts (1984 to 1993) but in our main specification we narrow the sample from 1984 to 1991, for reasons we will explain in Section 5.

The second source of data is the Health and Demographic Survey (DHS) of 1992. This dataset collects information on all children born alive in the previous 5 years of the survey, and records whether or not they are still alive at the time of the interview, meaning we do not have to rely only on the youngest child in this case. Moreover, this data set allows us analyze within-siblings effects. It also includes information of parental investment and other nutritional and health question about the children. We also limit this data set to children born until 1991.

The third source of data provides information about the incidence of cholera registered in Peru during the years 1991 to 1993, which was obtained from a report produced by the General Directorate of Epidemiology, of the Ministry of Health of Peru (Suárez-Ognio 2011). This information is disaggregated by week, year, and region (with the exception of Apurimac and Madre de Dios, for which information is lacking).

Finally, the fourth source of data is from Willmott and Matsuura (2001), which provides terrestrial air temperature and precipitation information per month and meteorological station, which we match to regions.

Table 1 shows summary statistics of our sample of children corresponding to the youngest child born between 1984 and 1991 to women age 12 or older in 1993. Peru has 25 regions, but as we mentioned above, we only have data on cholera incidence in 23 of them. There are a total of 2,208 observations that result from collapsing the data at the region-month level. We weight the means by the number of children born alive per region and month.

Children are on average 5 years old. The mothers are on average 34 years old, 21% are indigenous, have an average of 4 children in total, 9 years of educational attainment and 15% of them are illiterate. Forty-nine percent of the children have piped water at home, 64% have access to a toilet or cesspit. The average mortality rate is 1.8%. The average temperature and rainfall by trimester was 14 °C and 65 mm, respectively.

The average cholera incidence during the third trimester in utero for those children born in 1991 is 0.1% (for children born before that year the incidence is zero), with a maximum incidence of 3.4%. The cholera incidence, is calculated by dividing the number of cases by the population of the region as of 1993. Cholera cases are collected from diagnosis made on the basis of symptoms, not blood tests. This type

Table 1 Summary statistics census 1993—last child born alive

	Mean	sd	min	max
Child's age (alive or dead)	4.65	2.25	1.58	9.50
Birth order/number of children	3.67	0.83	2.27	6.96
Mother's age	33.58	3.48	27.29	45.27
Indigenous mother	21.46	23.78	0.00	88.71
Mother's years of education	8.82	2.05	2.72	11.85
Illiterate mother	15.40	12.27	0.00	67.57
Piped water	48.53	18.91	9.88	85.42
Toilet or cesspit	64.27	19.39	8.33	91.76
Mortality rate	1.79	1.02	0.00	7.87
Temperature—1st Trim.	14.24	6.07	5.06	27.92
Temperature—2nd Trim.	14.28	6.06	5.06	27.92
Temperature—3rd Trim.	14.21	6.06	5.06	27.87
Precipitation—1st Trim. (per 100)	64.05	62.97	0.00	321.79
Precipitation—2nd Trim. (per 100)	65.86	64.37	0.00	321.79
Precipitation—3rd Trim. (per 100)	64.26	63.20	0.00	321.79
Cholera—1st Trim.(per 100)	0.05	0.23	0.00	3.27
Cholera—2nd Trim.(per 100)	0.08	0.30	0.00	3.41
Cholera—3rd Trim.(per 100)	0.09	0.31	0.00	3.43
Observations	2208			

Note: Table show summary statistics for the main period of analysis (1984–1991), except for cholera incidence that only include data from 1991, since we know the incidence before that year was zero

of diagnosis has the advantage of not relying on testing capacity, test approval, etc. Naturally, this type of diagnosis is not perfect either. For example, other acute diarrheal infections might be misclassified as cholera infections. Nevertheless, it seems that the diagnoses were very accurate; in the most important hospital in Peru, of all the cases of cholera between February and May 1991 (diagnosed by symptoms only), circa 8% were tested and 100% of these cases reported positive results from the test (Saona et al. 1991).

Table 2 shows summary statistics of our sample of children corresponding to the DHS data of children born between 1986 and 1991. There is a total of 9,155 observations (children) but only 802 combinations of regions and month-year of birth. Back in 1991, and for a brief period of time only, Peru was divided into 13 regions. So, the survey captures that division with the exception of 4 regions in November 1986 for which there we have no observations. In 1993, the country went to back to have 25 regions and that is captured in the census. We do not collapse this data in order to exploit the within-sibling variation. We weight the means using the sampling weights of the survey.

Children in the survey sample are on average 3 years old. The mothers are on average 29 years old, have an average of 3 children age 0 to 5, and only 6 years of educational attainment. Fifty-five percent of the children have piped water at home.

Table 2 Summary statistics DHS 1992—children under five born alive

	Count	Mean	sd	min	max
Birth order	9155	3.43	2.59	1.00	19.00
Child's age	9155	2.51	1.43	0.00	4.92
Child's sex—female	9155	48.68	49.99	0.00	100.00
Mother's age	9155	29.15	6.74	15.00	49.00
Indigenous mother	9155	14.77	35.48	0.00	100.00
Mother's years of education	9150	6.34	4.23	0.00	16.00
Piped water at home	9155	55.23	49.73	0.00	100.00
Access to a toilet or cesspit	9155	0.55	0.50	0.00	1.00
Mother received tetanus vaccine	9105	35.57	47.88	0.00	100.00
Number of antenatal visits	9059	4.02	4.19	0.00	40.00
Mother has antenatal card	9064	32.01	46.65	0.00	100.00
Institutional births	9155	51.21	49.99	0.00	100.00
Low birth weight (< 2.5 kg.)	5490	7.78	26.79	0.00	100.00
Small size of child at birth	9099	20.01	40.01	0.00	100.00
Very small size of child at birth	9099	3.70	18.89	0.00	100.00
Premature child	9141	3.86	19.26	0.00	100.00
Mortality rate—children age 0-5	9155	6.18	24.07	0.00	100.00
Mortality rate—3 months or younger	9155	3.51	18.41	0.00	100.00
Neonatal mortality	9155	2.47	15.53	0.00	100.00
First-day mortality	9155	1.07	10.31	0.00	100.00
Child had diarrhea in past 2 weeks	8456	18.54	38.87	0.00	100.00
Child had diarrhea severe in past 2 weeks	8456	11.97	32.46	0.00	100.00
Child ill in past 2 weeks	8465	39.48	48.88	0.00	100.00
Stunting (height for age < 2 std.)	7548	31.90	46.61	0.00	100.00
Underweight (weight for age < 2 std.)	7548	10.70	30.91	0.00	100.00
Height for age (std.)	7548	−1.36	1.38	−5.97	5.58
Weight for age (std.)	7548	−0.57	1.23	−5.81	5.29

The total mortality rate of children is 6%. The prevalence of severe diarrhea (diarrhea episodes that last three days or more) is 12%, and the prevalence of stunting and underweight children is 32% and 11%, respectively.

Approximately half of the births are "institutional births", that is in public hospitals or health centers, or in a private location but with the assistance of a doctor. More precisely, 50% of birth are at home, 49% are in hospitals, health centers and private clinics or offices and 1% is others. Approximately, 90% of births that occur in medical institutions or health centers are assisted by a doctor or obstetrician. On the contrary, less than 6% of the births at home are assisted by a doctor. Most of them are assisted by a traditional midwife (typically without license), a relative, or no body.

Women have on average 4 prenatal visits, and 32% of women have prenatal cards ("tarjetas de control prenatal"). These are cards filled by the doctor/nurse

with the date of each prenatal control as well as with special comments of the doctor, for example, whether the woman is experiencing problems with the pregnancy. This information represents a complement and a more objective measure than the mothers' report of whether they had prenatal controls. Similarly, 36% reported having had a tetanus vaccine. The tetanus vaccine was, during those years, the only vaccine required to be given to pregnant women. It has a duration of 5 years and it is typically administered to the pregnant woman in her second or third trimester.

4 Empirical strategy

We estimate three models. We first apply a difference-in-difference (DD) approach, or more accurately, a Fuzzy difference-in-difference (De Chaisemartin and d'Haultfoeuille 2018), exploiting differences in the intensity of the cholera epidemic by month and region. Under certain conditions that we explain below, this model estimate a Local Treatment Effect on the "switchers" or compliers. We collapse the data at the region-month level and weight the regressions by the number of children born alive per region and month. In Appendices A and B, we show the estimation using total population as weights instead. Our main specification is the following:

$$Y_{r,m,t} = \beta_0 + \beta_1 Ch^1_{r,m,t} + \beta_2 Ch^2_{r,m,t} + \beta_3 Ch^3_{r,m,t} + \beta_4 X_{r,m,t} + \rho_{m,t} + \Omega_{m,r} + \phi_r trend + \varepsilon_{r,m,t}$$

where $Y_{r,m,t}$ stands for the mortality rate as of July of 1993 (Census' interview date) of children born alive in month m, and year t to women age 12 or more in 1993, who at the time of the census lived in region r. Unfortunately, we do not know the region of residence when a given woman was pregnant with her treated child but in Appendices A and B we estimate our results using only children born to women who did not relocate during the 5 years preceding the census (between 1988 and 1993), and our results are very similar.

$Ch^j_{r,m,t}$ stands for the cholera incidence during the jth trimester in utero of the child. For example, $Ch^3_{Ica,4,1991}$ represents the 14-week incidence before April 15th, 1991 in the region of Ica. Our data does not provide day of birth, so we assume all children were born on the 15th day of the month. Our data also does not provide information on gestational age at birth, hence we assume 38 weeks of gestation and we follow the medical literature regarding the definition of the trimesters: the first trimester comprises weeks 1 to 10, the second trimester comprises weeks 11 to 24 and the third trimester comprises weeks 25 to 38. Exposure to cholera, however, could have reduced gestational age at birth. So, in Appendices A and B, we estimate our regressions assuming 36 and 34 weeks of gestation.

The medical literature affirms that third trimester is the most sensitive for cholera cases (Hirschhorn et al. 1969), but we also estimate the effect of exposure to cholera during the other trimesters to confirm this previous result. In addition, we estimate the effect of exposure to cholera in the first trimester after birth, because prenatal and postnatal incidence of cholera might be correlated and postnatal exposure may also have an effect on childhood mortality. Cholera incidence of further away periods is

less likely to be correlated with the incidence while in utero and less likely to affect childhood mortality, since typically around half of childhood mortality happens soon after birth. Finally, we additionally estimate the effect of exposure to cholera in the trimester before conception, as a placebo test.

$\rho_{m,t}$ stands for month-year of birth fixed effects, $\Omega_{m,r}$ stands for month-region fixed effect, $\phi_r trend$ stands region-specific trends, and $X_{r,m,t}$ stands for control variables: ethnicity, number of children (which in this case is also the birth order), age, and education of the mother, whether or not they have access to piped water, to a toilet or cesspit, and average temperature and rainfall by trimester in utero. Standard errors are clustered at the regional level. Since we only have data from 23 regions in the census and 13 regions in the survey, we present the p-values of wild bootstrap inferences, to correct for the small number of regions, following Cameron et al. (2008).

This empirical strategy addresses several potential sources of bias. First of all, the regions fixed effects control for time-invariable regional variables that might be correlated with cholera infection and childhood mortality. Second, the region-specific trends control for the underlying trends in mortality, which were already increasing during those years in most regions. The region-month of birth fixed effects control for regional-specific seasonal variables that can be correlated with the intensity of cholera and childhood mortality. Finally, the month and year birth effects control for any potential nationwide shock that could affect mortality rates during those months. Month and year birth effects also control for differences in mortality by children' age.

As with any other DD application, we assume that our results are not biased by changes in unobservable variables that are correlated both with the cholera epidemic and with childhood mortality. As a way to increase the credibility of this assumption, in Appendix A we show with placebo regressions that our observable variables do not vary meaningfully with the variation in cholera intensity that we exploit. We also restrict the sample to those born by the first semester of 1991, with the objective of including only children who were conceived before the outbreak of the epidemic and in this way rule out the possibility that our results are generated by selective fertility.

In addition to the traditional assumptions, a Fuzzy-DD approach has two additional assumptions: a stable treatment effect over time and homogeneous treatment effects for switchers of both treatment and comparison groups. Our comparison group are region-months combinations that were affected by the cholera epidemic in a smaller degree than the region-months combinations from treatment group. In Appendix A, we relax these assumptions and estimate Intertemporal Treatment Effects, following de Chaisemartin and D'Haultfœuille (2020).

Our second model is an event study similar to that of Cutler et al. (2010) and Alpert et al. (2018), among others. This non-parametric model allow us to test whether the intensity of the cholera epidemic was greater in regions and months in which childhood mortality and our other outcomes were already growing. The following is our event study specification:

$$Y_{r,m,t} = \alpha_o + \sum_{t=1984}^{1991} \beta_t Ch_{r,m}^{3,1991} t + \gamma_1 X_{r,m,y} + \rho_{m,y} + \Omega_{m,r} + \phi_r trend + \varepsilon_{r,t}$$

$Ch_{r,m}^{3,1991}$ stands for cholera incidence corresponding to the last trimester in utero of the child of region r born in month m of year 1991. Thus, β_{1991} gives us the effect on mortality of a 1 percentage point increase in cholera incidence for those children who effectively were in utero in 1991, while β_{1990} gives us the effect on mortality of a 1 percentage point increase in cholera incidence for those children who were in utero in 1990, a year before the epidemic started, β_{1989} gives us the effect on mortality of a 1 percentage point increase in cholera incidence for those children who were in utero in 1989, 2 years before the epidemic started, and so forth. It is important to note that even in the absence of pre-existing trends, β_t for $t < 1991$ are not necessarily equal to 0 , because they give us the effect on mortality of postnatal exposure to cholera. However, the largest part of childhood mortality happens in the first year of life, and so most of the children that die in our sample, has already died before their first year of life, and so β_t for $t < 1991$ should be noisier and probably smaller than the effect of the exposure while in utero. More importantly, if the event study shows no long-term trend in increasing mortality rates leading up to the cholera epidemic, then that is evidence that our effects are not endogenously driven by omitted variables correlated with the spread of the epidemic.

Finally, we estimate a third model with the survey data applying the same DiD strategy but including a mother's fixed effects. Working with this data and empirical strategy has advantages and disadvantages. The main disadvantage is that we have considerably less statistical power for our estimations; first, we only exploit within-sibling variation, second, the data contains a representative but small sample of the population, and third, the data is divided in 13 larger regions, instead of the 23. Hence, we lose much of the cross-sectional variation.

The main advantage of this augmented empirical strategy and additional data set is that it removes several of the concerns that we have with our first model and data. First of all, this model controls for selection at the mother's level. As we have seen in the Introduction, while not even experts seemed to have anticipated the cholera epidemic at that precise time, with our DD model we cannot completely discard the possibility that the effects are driven by differences in the sample of women who gave birth during the cholera epidemic. With this third model, however, we control for such potential differences. Second, with the census data we estimate only the effect on last-born child, hence should we not want to assume the same average treatment effect between last-born and early-born children, our first model estimates a Local Treatment Effect (LATE) on last-born children. With this third model and data, however, we include all children age 0 to 5, irrespective of whether or not they were last born. Hence the external validity of this model is greater. Third, since the data we use to estimate this model was collected at the end of 1991 and at the beginning of 1992, it reduces the likelihood that the region of residence is other than the native region of children exposed to the epidemic. Finally, with this data we observe the age of death, and hence we can estimate the effects on mortality by age of death. For more robustness and sensitivity tests on this model, please see our Appendix B.

Before concluding this empirical section, we would like to make a final note about our main regressor. Other studies use maternal infection rate as the main regressor, for example Almond (2006). Unfortunately, we do not have mother's infection rate, or infection by gender or age. However, we believe, our regressor, cholera incidence

Table 3 Effects on childhood mortality (census data)

	(1) 1984–1993	(2) 1984–1991	(3) 1984–1991	(4) 1984–1991	(5) 1984–1991	(6) 1984–1991	(7) 1984–1991
In utero—3rd trim. (Std)	0.05	0.05	0.04			0.05	0.04
	(0.01)	(0.00)	(0.00)			(0.01)	(0.01)
	[0.01, 0.11]	[0.02, 0.12]	[0.02, 0.12]			[0.02, 0.12]	[0.02, 0.12]
In utero—2nd trim. (Std)	−0.02	−0.01				−0.01	−0.01
	(0.03)	(0.35)				(0.38)	(0.45)
	[−0.06, -0.00]	[−0.04, 0.01]				[−0.05, 0.01]	[−0.04, 0.01]
In utero—1st trim. (Std)	0.02	0.03		0.02		0.03	0.03
	(0.09)	(0.06)		(0.16)		(0.07)	(0.11)
	[−0.01, 0.04]	[−0.00, 0.07]		[−0.01, 0.05]		[−0.00, 0.07]	[−0.01, 0.08]
In utero—9 months (Std)					0.04		
					(0.03)		
					[0.01, 0.09]		
Postnatal—1st trim. (Std)						−0.00	−0.00
						(0.80)	(0.76)
						[−0.05, 0.03]	[−0.05, 0.03]
Preconception 1 trim. (Std)							0.02
							(0.72)
							[−0.18, 0.16]
R^2	0.805	0.782	0.782	0.781	0.781	0.782	0.797
Observations	2760	2208	2208	2208	2208	2208	2001

Note: All regressions include month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order/ number of children, ethnicity, age, and education of the mother, whether they have access to piped water at home, a toilet, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

at the regional level, is a more appropriate way to address our research question. With the Covid-19 epidemic, we now know more than ever that an epidemic can affect individuals not just through the disease itself but through other mechanisms. We believe this is part of our contribution to the literature. By exploiting the variation of the cholera incidence at the regional level we incorporate the potential effects of, for example, the saturation of the health infrastructure of a region. This can be particularly harmful for pregnant women that need attention several times during their pregnancies and, of course, during the delivery. This is especially true in poor countries, and was especially true during the cholera epidemic in Peru, as we have seen above.

5 Results from census data

Table 3 presents the estimated effects of exposure to cholera on childhood mortality according to different specifications. The first column shows the effects of estimating the difference-in-difference specification for the entire period between January 1984 and July 1993. We estimate the effect of the three trimesters in utero. All regressions include birth month-year, and month-region fixed effects, region-specific trends and control for ethnicity, birth order (or number of children born alive), age, and education of the mother, whether they have access to piped water, to a toilet and temperature and rainfall by trimester. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets. According to the first column, exposure during the 3rd trimester has the greatest effect on childhood mortality; an increase of 1 standard deviation in the incidence of cholera during the last trimester in utero increases average childhood mortality rate by 0.05 percentage points, which represents and increase of 3% or 0.04 standard deviations. The medical literature also affirms that the third trimester is the most sensitive for cholera cases (Hirschhorn et al. 1969). We also find that an increase of 1 standard deviation in the incidence of cholera during the first trimester in utero increases average childhood mortality rate by 0.02 percentage points, which represents and increase of 1.2% or 0.02 standard deviations. Finally, we find that an increase of 1 standard deviation in the incidence of cholera during the second trimester in utero *decreases* average childhood mortality rate by 0.02 percentage points. This result might seem odd at first, however this is actually possible; it could be the result of miscarriage and of the surviving children being a healthier sub-set of the total cohort. "Selective mortality/miscarriage" has been noticed by many researchers (Bozzoli et al. 2007; Currie and Vogl 2013). In fact, according to Saona et al. (1991), most miscarriages due to cholera infection in Peru occurred during the first and second trimester of pregnancy.

In the rest of the columns, we estimate the same regression, but restricting our data to the period between January 1984 and December 1991. By including only the first year of the epidemic, we reduce the possibility of selective fertility. Moreover, since the epidemic was quite unexpected when it appeared, but over time individuals, in particular educated individuals, became aware of the illness and learned ways to prevent it, we would expect that the effect is less likely to be endogenous in the first year of the epidemic. In Appendices A and B, we restrict the sample even further

to those born by the first semester of 1991, with the aim of including only children conceived before the outbreak of the epidemic. In column 2, we can see that the effect of exposure to cholera during the third trimester remains statistically significant in all the specifications. In the case of exposure to cholera during the second trimester, the negative effect is no longer statistically significant, and in the case of exposure to cholera during the first trimester, the coefficient is statistically significant only in some of the specifications. Columns 3 and 4 show the estimates of exposure to cholera only during the third and first trimester, respectively. In particular, we can see that the estimate for the exposure during the third trimester is very insensitive to controlling for the incidence during other trimesters. This result is not surprising given that the intensity of cholera varied significantly within regions over time, even within months, and given that we are working with census data and, therefore, have good statistical power.

In column 5, we show the effect of exposure during the 38 weeks in utero, and we can see a significant effect on mortality; an increase of 1 standard deviation in the incidence of cholera during the 38 weeks in utero increases average childhood mortality rate by 0.04 percentage points or 2%.

Column 6 includes the estimation of the effect of cholera during the first trimester after birth. It is somewhat surprising that postnatal exposure during the first trimester after birth does not affect childhood mortality rate. However, we need to keep in mind that typically 40% of childhood deaths happen in the first month after birth, and a third of neonatal deaths occur in the first day of life. This means that some of the children in our sample were not actually exposed to postnatal incidence of cholera, because they were already dead after the first month. In fact, we will see in the next section that there is a significant effect on first-day mortality. Another explanation could be that breastfeeding reduced babies' exposure to the epidemic. There is evidence that mothers respond in this way to protect babies from contaminated water (Keskin et al. 2017). This does not have to be a premeditated response, since breastfeeding is very generalized during the first trimester of life.

Finally, in column 7 we include as a placebo, the estimation of the effect on mortality of cholera during one trimester before conception. It is reassuring to find no significant effect, but this result is not sufficient to prove causality. During the 1980s and early 1990s, Peru was going through a very bad economic situation, including an increase in infant mortality (Baird et al. 2011; Paxson and Schady 2005). Hence our results could be driven by a previous trend. Figure 4 shows event study for exposure during the third trimester and we can see, however, that there is no previous trend in those regions and in those months with higher incidence of cholera in 1991 in comparison with the rest of regions and months. This result provides good evidence that our estimation is causal. Moreover, Fig. 5 presents an event study of maternal educational attainment as a placebo test that provides evidence that there was no sample selection in the mothers of children affected in greater magnitude by the epidemic. In Appendix A, we show more placebo tests and robustness checks that increase even further the reliability of our results. The event study for exposure during the first trimester in Fig. 6 shows what we were observing in the regressions: a much noisier and mostly non-significant effect on mortality.

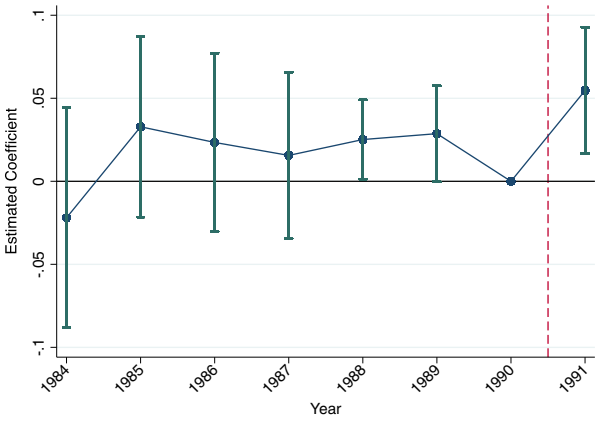

Fig. 4 Event study—mortality rate—cholera incidence 3rd trimester in utero. Note: Graph includes point estimates from the event study (normalized to 0 the coefficient of 1990) and 95% confidence intervals of wild bootstrap inferences to correct for the small number of regions

These results are most likely a lower bound estimate for several reasons: first, the epidemic likely generated a sizable number of miscarriages, which tend to be concentrated among those who are physically weakest, meaning the survivors will tend to be a healthier subpopulation (Bozzoli et al. 2007; Currie and Vogl 2013). Second, the epidemic killed a large number of adults, meaning pregnant women who survived a cholera infection were probably the healthiest, and our sample does not contain information on motherless children. Third, we do not have the age of death for children and we observe only whether children have died before mid 1993, and the epidemic started in 1991. Hence, we can only see whether the children affected by the epidemic died at age 2.5 or before, and some children might have died after reaching that age.

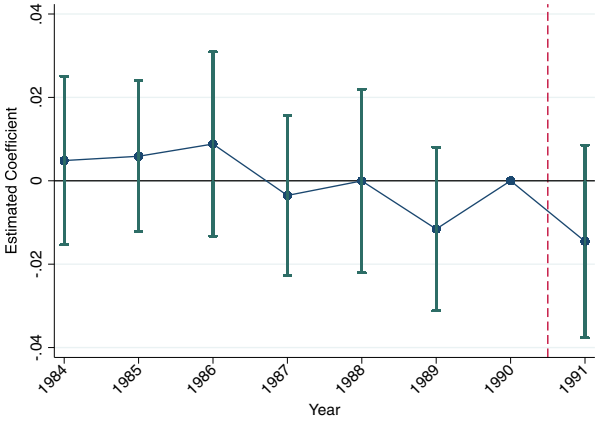

Fig. 5 Placebo event study—maternal education—cholera incidence 3rd trimester in utero. Note: Graph includes point estimates from the event study (normalized to 0 the coefficient of 1990) and 95% confidence intervals of wild bootstrap inferences to correct for the small number of regions

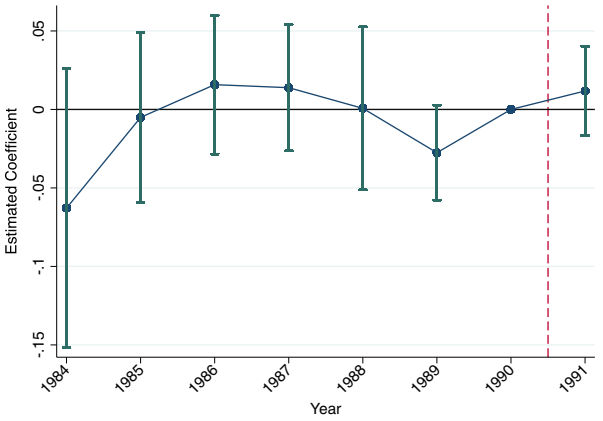

Fig. 6 Event study—mortality rate—cholera incidence first trimester in utero. Note: Graph includes point estimates from the event study (normalized to 0 the coefficient of 1990) and 95% confidence intervals of wild bootstrap inferences to correct for the small number of regions

Table 4 shows heterogeneous effects by poverty. The level of poverty by district is defined by the proportion of households with at least one unsatisfied basic need, which was calculated by the INEI from the Census 1993. We define "poor districts" as districts with a larger proportion of "poor" households than the median district. As expected, the effect of exposure to cholera during the third trimester on mortality is greater among poorer districts. Interestingly though, the effect of exposure to cholera during the first trimester on mortality is greater among the less poor districts. This last result could be derived from a smaller number of miscarriages among the less poor districts, which in turn reduces the "selection" effect.

Table 4 also shows heterogeneous effects by maternal education. We define women with "high educational attainment" as those women with educational attainment higher than the median and women with "low educational attainment" as those women with educational attainment lower than the median. Again, as expected, the effect of exposure to cholera during the third trimester on mortality is greater among children of less well-educated mothers.

6 Results from survey data

In this section, we present the results from the estimations using the survey Data (DHS 1992). One of the shortcomings of this data, aside from the obvious fact that it is a survey and, therefore, has significantly fewer observations than a census, is that the configuration of regions in this data is different than in our cholera data and in the census data; the country is divided in 13 larger regions, instead of the 25. We thus, lose much of the cross-sectional variation. With this data, however, we have the advantage of knowing the age of death, and we can estimate within-siblings effects.

Table 5 presents the estimated effects of exposure to cholera on mortality using this data. In the first column we show the result of running the difference in difference,

Table 4 Heterogeneous effects by district poverty and maternal education

	(1) Poor districts 1984–1991	(2) Non-poor districts 1984–1991	(3) Low educational attainment 1984–1991	(4) High educational attainment 1984–1991
In utero—3rd trim. (Std)	0.05	0.00	0.08	−0.01
	(0.01)	(0.81)	(0.00)	(0.35)
	[0.02, 0.13]	[−0.07, 0.11]	[0.04, 0.19]	[−0.07, 0.03]
In utero—2nd trim. (Std)	−0.02	−0.01	−0.03	0.02
	(0.08)	(0.52)	(0.07)	(0.16)
	[−0.05, 0.00]	[−0.08, 0.03]	[−0.09, 0.01]	[−0.02, 0.06]
In utero—1st trim. (Std)	0.01	0.03	0.03	0.02
	(0.67)	(0.04)	(0.20)	(0.23)
	[−0.07, 0.06]	[0.00, 0.10]	[−0.03, 0.10]	[−0.02, 0.06]
R^2	0.554	0.393	0.618	0.446
Observations	2208	1824	2208	2208

Note: All regressions include month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order/ number of children, ethnicity, age, and education of the mother, whether they have access to piped water at home, a toilet, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

Table 5 Effects on mortality by age of death (survey data)

	(1) Total mortality rate OLS	(2) Total mortality rate FE	(3) Mortality rate 3 month FE	(4) Neonatal mortality FE	(5) First-day mortality FE	(6) Mortality rate 3 month FE	(7) First-day mortality FE
In utero—3rd tr. (Std)	0.22	0.66	1.09	0.70	0.70	1.22	0.79
	(0.70)	(0.37)	(0.06)	(0.16)	(0.06)	(0.06)	(0.04)
	[−1.86, 1.21]	[−1.52, 2.74]	[−0.06, 3.02]	[−0.57, 2.27]	[−0.04, 1.89]	[−0.16, 3.52]	[0.05, 1.85]
In utero—2nd tr. (Std)	−0.05	0.70	−0.18	−0.01	−0.23		
	(0.91)	(0.72)	(0.84)	(0.97)	(0.34)		
	[−1.44, 1.15]	[−3.34, 3.70]	[−3.44, 1.98]	[−1.72, 1.44]	[−1.29, 0.32]		
In utero—1st tr. (Std)	−0.66	−1.35	−0.77	−0.86	−0.56		
	(0.07)	(0.32)	(0.41)	(0.26)	(0.25)		
	[−1.31, 0.04]	[−5.49, 2.55]	[−4.05, 1.88]	[−3.72, 1.44]	[−2.60, 0.90]		
R^2	0.052	0.080	0.070	0.061	0.071	0.070	0.070
Observations	9145	9155	9155	9155	9155	9155	9155

Note: All regressions include mother fixed effect (except for the first column), month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order, sex, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

just as we did with the census data. As expected, our estimate is significantly less precise; the point estimate of exposure to cholera during the third trimester is approximately 5 times larger, but it is not statistically significant. Columns 2 to 6 show the effects of estimating the within-siblings effect, on total mortality, on mortality in the first 3 months of life, on mortality in the first month of life and on first-day mortality respectively. We concentrate on early life mortality only, because at the time of the survey approximately half of the children born in 1991 were less than 6 months old. We can see that including the mother fixed effect approximately triplicates the estimate and limiting the age of death to 3 months or less increases the significance. We find that an increase of 1 standard deviation in the incidence of cholera during the last trimester in utero increases three-month mortality by 1.1 percentage points or 31% and first-day mortality rate by 0.7 percentage points or 70%. This last result is very interesting and suggests that a significant part of the deaths might have occurred due to complications at birth. The magnitude of the effects are large, but so are the confidence intervals; the effects represents only 0.06 and 0.07 of a standard deviation of three-month mortality and first-day mortality, respectively. Also this is a sample of mothers less well educated, as we saw in the summary statistics.

Table 6 shows the effect of the epidemic on some fetal health indicators, following Goodman-Bacon (2018): sex composition, low, and very low birth weight/size and gestational age. Column 1 shows the effect on the sex composition of children born alive. We do not observe a statistically significant effect on the probability of a child being born female, but we cannot dismiss this possibility out of hand. Column 2 shows the effect on low birth weight. Unfortunately, only about half of households answered this question. We do not find any significant effect on low birth weight (in our data there are no children with a very low birth weight), nor any effect on the perceived size of the baby at birth, as shown in columns 3 and 4 or in the likelihood of being premature, as shown in column 5. In column 6, we follow Kling, Liebman, and Katz (2007), Anderson (2008) and Hoynes et al. (2016) and construct a summary indicator for "fetal health" with the objective of increasing the statistical power of our estimation. This indicator is the simple average across standardized measures of each component: low, and very low birth weight/size and whether the child was a preterm baby. The standardization of each variable is made by subtracting the mean and dividing by the standard deviation. Since all the indicators of fetal health are negative, we multiply the indicator by -1 to get a positive indicator. We can see that there is no significant effect on this indicator either.

Table 7 presents the estimated effects on prenatal investments. We find no significant effect on the number of prenatal visits but exposure to cholera during the third trimester in utero reduces the probability of having an prenatal card, and of a mother having had a tetanus shot in the last 5 years. Column 4 shows that exposure to cholera during the third and first trimester in utero reduces the likelihood of an institutional birth by 1.6 percentage points or 3%. In column 5, we construct another summary indicator, this time for "prenatal investment" with the objective of increasing the statistical power of our estimation. This indicator is the simple average across standardized measures of each component: number of prenatal visits, having an prenatal card, having had a tetanus shot and institutional birth. The standardization of each variable is made by subtracting the mean and dividing by the standard deviation. We

Table 6 Fetal health

	(1) Child's sex female	(2) Low birth weight	(3) Small size at birth	(4) Very small size at birth	(5) Premature child	(6) Fetal health
In utero—3rd tr. (Std)	0.02	−1.21	0.47	0.49	−0.41	0.01
	(0.35)	(0.17)	(0.75)	(0.11)	(0.55)	(0.83)
	[−0.04, 0.11]	[−7.59, 1.09]	[−4.60, 4.99]	[−0.33, 1.32]	[−1.16, 1.48]	[−0.09, 0.15]
In utero—2nd tr. (Std)	0.01	1.17	−0.41	−0.08	−0.08	−0.01
	(0.69)	(0.43)	(0.63)	(0.88)	(0.87)	(0.73)
	[−0.05, 0.05]	[−1.96, 3.11]	[−3.87, 2.22]	[−2.63, 1.38]	[−0.85, 1.61]	[−0.04, 0.06]
In utero—1st tr. (Std)	−0.00	−0.92	2.01	0.42	0.32	0.00
	(0.58)	(0.53)	(0.14)	(0.42)	(0.37)	(0.93)
	[−0.03, 0.02]	[−3.88, 3.13]	[−1.48, 6.54]	[−0.64, 2.95]	[−0.33, 1.90]	[−0.09, 0.08]
R^2	0.073	0.184	0.075	0.092	0.078	0.174
Observations	9155	5490	9099	9099	9141	5472

Note: the summary index "Fetal health" is a simple average across standardized measures of each component: low, and very low birth weight/size and whether the child was a preterm baby. All regressions include mother fixed effect, month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order, sex, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

Table 7 Prenatal investments

	(1) Prenatal visits	(2) Prenatal card	(3) Tetanus vaccine	(4) Institutional births	(5) Prenatal investment
In utero—3rd tr. (Std)	0.00	−1.72	−2.68	−1.61	−0.03
	(0.95)	(0.06)	(0.07)	(0.03)	(0.03)
	[−0.24, 0.20]	[−3.77, 0.17]	[−8.09, 1.19]	[−3.83, -0.58]	[−0.07, -0.01]
In utero—2nd tr. (Std)	0.01	0.53	1.92	−0.11	0.01
	(0.78)	(0.34)	(0.40)	(0.92)	(0.20)
	[−0.07, 0.28]	[−1.68, 2.84]	[−2.40, 3.16]	[−2.13, 3.27]	[−0.01, 0.05]
In utero—1st tr. (Std)	−0.03	0.10	0.49	−1.92	−0.01
	(0.41)	(0.89)	(0.42)	(0.01)	(0.41)
	[−0.25, 0.05]	[−1.93, 3.48]	[−1.27, 3.18]	[−4.40, -0.46]	[−0.03, 0.03]
R^2	0.107	0.094	0.111	0.083	0.095
Observations	9059	9064	9105	9155	8978

Note: the summary index "Prenatal investments" is a simple average across standardized measures of each component: number of prenatal visits, having an prenatal card, having had a tetanus shot and institutional birth. All regressions include mother fixed effect, month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order, sex, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

find that 1 standard deviation in the incidence of cholera during the last trimester in utero decreases prenatal investment by 0.03 standard deviations. These results suggest that childhood mortality, in particular first-day mortality could have been driven at least in part by the lack of skilled care during pregnancy and birth.

With respect to the potential magnitude of the effect of prenatal investments on mortality, the effects, for example, of institutional births on mortality rates estimated by previous studies are significantly large: between 50% and 100% (Daysal et al. 2015; Godlonton and Okeke 2016; Okeke and Chari 2018; Friedman and Keats 2019a, b). We find that 1 standard deviation in the incidence of cholera during the last trimester in utero reduces institutional births by 1.6 percentage points. Based on previous studies, we would expect an increase in mortality between 0.8 and 1.6 percentage points, which corresponds very closely to our estimates. Hence, a large proportion of the first-day mortality (and to a lesser extent 3-month mortality) could be explained by the effect of the cholera epidemic on institutional births, and more generally prenatal investments.

Table 8 shows the effects on some health indicators of surviving children at the time of the interview. Columns 1 and 2 show no significant effect on being ill or having at least one episode of diarrhea in the two weeks prior to the interview, respectively. Column 3, however, shows that an increase of one standard deviation in the incidence of cholera during the third trimester increases the likelihood of severe diarrhea (defined as diarrhea for at least 3 days) by 2.2 percentage points or 18%. Column 4 shows that an increase of one standard deviation in the incidence of cholera during the first trimester decreases height for age by 0.05 standard deviations but column 5 shows no effect on the probability of the child being stunted. Columns 5 and 6 show that an increase of one standard deviation in the incidence of cholera during the first trimester decreases weight for age by 0.15 standard deviations and increases the likelihood of being underweight by 2.87 percentage points or 26%, respectively.

Tables 9 and 10 show heterogeneous effects. In these tables, we do not show the coefficients of the exposure to cholera during the second trimester in order to save space and focus the attention on our main results. We cannot identify districts in the survey data, which is why we cannot construct the heterogeneous effect by poverty with this data, but in this data we observe maternal education and sex of the child. Table 9 shows heterogeneous effects by education. The effects on total mortality estimated with the census data, 3-month mortality and severe diarrhea estimated with the survey data are concentrated among children of less well-educated mothers. The effect on prenatal investments, however, is larger for children of better educated women, and the effect on 1st-day mortality does not show much difference by education. One *potential* explanation for these results is that, on the one hand, the effects on total mortality, 3-month mortality and severe diarrhea are mostly driven by the impact of the disease while in utero, which presumably affected less well-educated women to a greater degree. On the other hand, the effect on first-day mortality is driven by a combination of the impact of the disease and the impact on prenatal investments, and the latter presumably depends more on the saturation of the health system in populated areas where women tend to be more educated.

A similar pattern can be seen in Table 10; the effects on 3-month mortality and severe diarrhea are concentrated among female children. The effect on prenatal

Table 8 Health surviving children

	(1) Ill in past 2 weeks	(2) Diarrhea in past 2 weeks	(3) Severe diarrhea	(4) Height for age (std.)	(5) Stunting (height for age < 2)	(6) Weight for age (std.)	(7) Under weight (weight for age < 2)
In utero—3rd tr. (Std)	−1.04	2.07	2.22	−0.05	1.76	−0.05	0.73
	(0.57)	(0.18)	(0.00)	(0.11)	(0.46)	(0.21)	(0.47)
	[−2.91, 2.78]	[−0.65, 8.10]	[0.54, 7.21]	[−0.16, 0.02]	[−6.80, 5.07]	[−0.23, 0.02]	[−1.19, 5.22]
In utero—2nd tr. (Std)	1.00	0.17	−0.28	−0.06	1.31	−0.05	2.43
	(0.77)	(0.85)	(0.71)	(0.20)	(0.45)	(0.21)	(0.09)
	[−4.39, 3.26]	[−3.20, 2.48]	[−3.87, 2.03]	[−0.22, 0.06]	[−3.87, 7.37]	[−0.16, 0.05]	[−0.85, 6.76]
In utero—1st tr. (Std)	0.90	2.29	2.54	−0.03	−0.58	−0.15	2.88
	(0.78)	(0.21)	(0.17)	(0.64)	(0.72)	(0.00)	(0.06)
	[−4.31, 11.09]	[−3.21, 7.89]	[−1.71, 9.82]	[−0.26, 0.07]	[−5.29, 6.40]	[−0.33, -0.08]	[−0.14, 7.02]
R^2	0.174	0.214	0.173	0.363	0.259	0.324	0.147
Observations	8465	8456	8456	7548	7548	7548	7548

Note: All regressions include mother fixed effect, month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order, sex, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

Table 9 Heterogeneous effects by education

	(1) Mortality rate 3 months	(2) First-day mortality	(3) Prenatal investment	(4) Severe diarrhea	(5) Weight for age (std.)	(6) Under weight
In utero 3rd tr. x low educ. att. (std)	1.33	0.71	−0.02	4.22	−0.05	0.43
	(0.05)	(0.12)	(0.21)	(0.00)	(0.19)	(0.79)
	[0.00, 4.04]	[−0.46, 2.94]	[−0.06, 0.03]	[1.74, 11.85]	[−0.25, 0.02]	[−2.58, 7.15]
In utero 3rd tr. x high educ. att. (std)	0.60	0.64	−0.05	−0.60	−0.05	1.26
	(0.25)	(0.10)	(0.05)	(0.59)	(0.36)	(0.32)
	[−0.85, 1.97]	[−0.20, 1.86]	[−0.10, 0.00]	[−2.02, 1.49]	[−0.30, 0.06]	[−2.50, 5.40]
In utero 1st tr. x low educ. att. (std)	−0.58	−0.55	−0.02	2.51	−0.14	2.70
	(0.53)	(0.29)	(0.14)	(0.21)	(0.01)	(0.07)
	[−3.81, 2.11]	[−2.58, 0.97]	[−0.05, 0.01]	[−2.26, 11.22]	[−0.38, -0.08]	[−0.69, 7.12]
In utero 1st tr. x high educ. att. (std)	−1.18	−0.58	0.01	3.05	−0.19	2.91
	(0.26)	(0.32)	(0.22)	(0.15)	(0.01)	(0.13)
	[−5.90, 1.27]	[−2.48, 0.90]	[−0.02, 0.03]	[−0.89, 10.67]	[−0.30, -0.07]	[−1.61, 7.11]
R^2	0.070	0.070	0.098	0.178	0.325	0.149
Observations	9150	9150	8973	8451	7543	7543

Note: All regressions include month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order/ number of children, ethnicity, age, and education of the mother, whether they have access to piped water at home, a toilet, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

Table 10 Heterogeneous effects by sex

	(1) Mortality rate 3 months	(2) First-day mortality	(3) Prenatal investment	(4) Severe diarrhea	(5) Weight for age (std.)	(6) Under weight
In utero 3rd tr. x female (std)	1.91	0.77	−0.02	2.90	−0.05	0.21
	(0.03)	(0.05)	(0.12)	(0.02)	(0.33)	(0.84)
	[0.25, 5.02]	[−0.04, 1.91]	[−0.06, 0.01]	[1.13, 7.26]	[−0.23, 0.05]	[−2.37, 3.59]
In utero 3rd tr. x male (std)	0.03	0.61	−0.04	1.52	−0.06	1.11
	(0.96)	(0.11)	(0.02)	(0.29)	(0.07)	(0.34)
	[−1.87, 1.88]	[−0.19, 2.01]	[−0.11, -0.01]	[−0.76, 6.81]	[−0.24, 0.01]	[−1.58, 6.49]
In utero 1st tr. x female (std)	−1.03	−0.69	−0.00	1.83	−0.10	3.03
	(0.21)	(0.24)	(0.71)	(0.35)	(0.02)	(0.06)
	[−4.50, 1.09]	[−2.79, 1.08]	[−0.03, 0.04]	[−3.93, 10.71]	[−0.23, -0.04]	[−0.33, 7.31]
In utero 1st tr. x male (std)	−0.49	−0.42	−0.01	3.36	−0.21	2.77
	(0.62)	(0.35)	(0.14)	(0.10)	(0.00)	(0.07)
	[−4.12, 1.85]	[−1.85, 0.77]	[−0.04, 0.01]	[−0.95, 9.81]	[−0.38, -0.12]	[−0.38, 7.44]
R^2	0.071	0.070	0.096	0.174	0.327	0.147
Observations	9155	9155	8978	8456	7548	7548

Note: All regressions include month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order/ number of children, ethnicity, age, and education of the mother, whether they have access to piped water at home, a toilet, and temperature and rainfall by trimester in utero. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

investments, however, is larger for males, and the effect on 1st-day mortality does not show much difference by sex. Larger results for females are not uncommon in the fetal origins literature. This result is typically attributed to stronger fetal survival for women, and a positive selection effect among surviving boys (Dagnelie et al. 2018). There is not obvious explanation of why the effect of on prenatal investments seems larger for males, but again the lack of differential effect by sex on first-day mortality might be explained by the fact that this mortality is driven by a combination of the impact of the disease while in utero and the impact on prenatal investments.

7 Magnitude of the effects and estimation of the additional deaths from the epidemic

With the census data we find that a 1 percentage point incidence of cholera during the last trimester in utero increases average childhood mortality rate by 0.04 percentage points. With the survey data, results are much larger in magnitude but also significantly less precise. Applying the same methodology, the point estimate is approximately five times larger and it is not statistically significant. Applying the fixed effect methodology, we find that 1 standard deviation in the incidence of cholera during the last trimester in utero increases 3-month mortality rate by 1.1 percentage points. If we look at the effects in terms of standard deviations, the magnitudes are not so different from each other: with the census data we find an increase on total childhood mortality of 0.04 standard deviations, while with the survey data we find an effect on three-month mortality of 0.06 standard deviations. Assuming the difference between the census estimate and the survey estimate (using the same methodology) give us the magnitude of the imprecision of the survey estimates, we would expect that the true effect is five times smaller, that is approximately 0.2 percentage points or 6%. As a comparison, Jayachandran (2009) finds that the wildfires in Indonesia during the 5 months in late 1997 and early 1998 caused an increase in the under-three mortality rate of 10% (assuming half of the deaths were miscarriages), Rocha and Soares (2015) find that 1 standard deviation increase in rainfall generates a decrease of 5% in infant mortality rate, and Banerjee and Maharaj (2020) find that excessive heat during pregnancy (20 days on average) increases infant mortality by 4%.

According to our estimation, the total number of children who died due to exposure to the cholera epidemic while in their last trimester in utero was in the range of 207 to 1036 of all youngest children born in 1991 that died by the age of 2.5[4]. Extrapolating our result to all children born in 1991, we have a total of 305 to 1,526 extra deaths not estimated in the official number of cholera deaths of 1991, which is 2,909. This means that the human cost of the epidemic was approximately 30% larger, if also we take into account the effect of prenatal exposure on childhood mortality. Moreover, the official total mortality rate due to postnatal exposure to the

[4]Given that our the effect on mortality ranges from 0.04 to 0.2 percentage points

cholera epidemic in Peru is 9 in every 100,000 inhabitants, while our estimated mortality rate due to prenatal exposure is 150 in every 100,000 children who were born alive in 1991. The striking difference in these numbers shows that in utero exposure to an epidemic can be more deadly than postnatal exposure. Naturally, this will vary from epidemic to epidemic; cholera might affect children in utero much more than other diseases. Nevertheless, we have seen that lack of prenatal investment is also an important driver of the effect on mortality. This means that even if the disease has no direct effect on the fetus, the economic consequences of the epidemic as well as the saturation of the health system can impose severe consequences in terms of childhood mortality.

8 Concluding remarks

Epidemics have been on the rise for the past several decades and will likely continue to do so in the near future. Much of the economic literature has focused on the consequences of viral outbreaks (Influenza, HIV, Ebola, Covid19), in spite of the fact that the number of bacterial outbreaks have increased as much as have the number of viral outbreaks, and the two most common outbreaks in the last two decades have been bacterial outbreaks: Salmonellosis and cholera (Smith et al. 2014). Poor countries, in particular, are highly vulnerable to bacterial infections given the high prevalence of antibiotic resistance (WHO 2018) and more generally, given their lack of access to basic health services, clean water and sanitation (WB 2017). In this study we find that an increase in 1 standard deviation in the incidence of cholera during the last trimester in utero increases average childhood mortality rate by approximately 5%, according to our most conservative estimate. This means that the human cost of the epidemic was approximately 30% larger, if we consider the effect of prenatal exposure on postnatal childhood mortality. Moreover, the official mortality rate from the cholera epidemic in Peru, during 1991, is 9 in every 100,000 inhabitants, while our estimated mortality rate due to prenatal exposure is 150 in every 100,000 children that were born alive in 1991.

We further find that the epidemic increased first-day mortality and reduced prenatal care and institutional deliveries and that later mortality is concentrated among girls. Additionally, we find that surviving children are more likely to suffer from severe diarrhea, have lower weight for age, and are more likely to be underweight. These results provide evidence that prenatal investments, in particular prenatal care and institutional deliveries, can act as mediators between the in utero shock and the effect on childhood mortality, and possibly also on surviving children's health status, and even longer-term outcomes. Epidemics in particular, but also many other shocks that have been analyzed in the fetal origins literature (droughts, wars, famines and hurricanes among other things) have the potential to affect access to skilled care during pregnancy and birth, hence, the results of this paper can apply to a wide range of shocks.

Appendix A. Robustness—difference-in-difference model—census data

This section presents placebo estimates and sensitivity analysis to further increase the reliability of the results estimated with our first model. In these tables, we do not show the coefficients of the exposure to cholera during the second trimester in order to save space and focus the attention on our main results. Table 11 tests whether mothers who gave birth in regions and times affected by the cholera epidemic are different from the rest of mothers in several observable variables. Columns 1 to 4 of show that there is no significant correlation between the variation of the epidemic that we exploit and the maternal educational attainment in years and levels, with the probability of a mother to be illiterate and with mother's age. In column 5, we observe a negative relationship with respect to the probability of the mother of being indigenous, however, this correlation attenuates the effect of the epidemic on childhood mortality, since childhood mortality was larger among indigenous mothers exposed to the epidemic.

Columns 1 and 2 of Table 12 test whether access to piped water and a toilet or cesspit (as opposed to open defecation) are correlated with the spread of the disease. We find no significant correlation between these variables and the variation of the epidemic that we exploit. Unfortunately we do not have access to variables regarding sanitation at the time of the epidemic, only at the time of the census (2 years after the epidemic broke up), but it is still reassuring to see no significant correlation. These results are maybe unsurprising, since at the regional level and over time, and at least in the first year, the abruptness and severity of the epidemic, as well as the speed of contagion, make the geographical proximity of the regions to the initial cases the main drivers of the disease.

Columns 3 and 4 of Table 12 present other placebos for the correlation with the number of children born alive per mother and on the size of the cohort of "last children" born alive. Pregnancy rates could have decreased as a response to the cholera epidemic. We can see, however, that there is a statistically significant correlation with the total number children born alive per women, but the correlation is positive and very small. This result is not surprising given that most children in our sample were conceived before the outbreak of the epidemic. Moreover, we will see below that our results remain very similar even when we limit the sample to those children born in the first semester of 1991, that is conceived before the outbreak of the epidemic. Finally, the exposure to the cholera outbreak while in utero may have reduced the probability that a family replace a lost child and this, in turn, would bias our results on mortality. If that were the case, exposure to cholera would reduce the number of all the children born alive and would increase size of the cohort of "last children" born alive. As we have seen above, there is no negative correlation between exposure to cholera during the third trimester and the number of all children born alive, nor is there any positive correlation with the size of the cohort of "last children" born alive..

Finally, Table 13 tests whether temperature are rainfall were correlated with the spread of the disease. Column 1 reveals that there is no significant correlation

Table 11 Placebos

	(1) Mother years of education	(2) Mother level of education	(3) Illiterate mother	(4) Mother age	(5) Indigenous mother
In utero—3rd trim. (Std)	−0.014	−0.003	0.109	0.002	−0.179
	(0.31)	(0.72)	(0.24)	(0.96)	(0.01)
	[−0.028, 0.007]	[−0.009, 0.004]	[−0.058, 0.199]	[−0.027, 0.048]	[−0.293, −0.043]
In utero—1st trim. (Std)	−0.009	−0.001	0.122	0.006	−0.220
	(0.45)	(0.77)	(0.40)	(0.37)	(0.02)
	[−0.017, 0.013]	[−0.004, 0.006]	[−0.145, 0.230]	[−0.009, 0.043]	[−0.425, −0.043]
R^2	0.996	0.994	0.992	0.996	0.998
Observations	2208	2208	2208	2208	2208

All regressions include month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order/ number of children, ethnicity, age, and education of the mother, whether they have access to piped water at home, a toilet, and temperature and rainfall by trimester in-utero, except when used as the placebo variable. *p*-values of wild bootstrap inferences shown in parentheses and 95-percent confidence intervals shown in brackets

Table 12 Placebos 2

	(1) Piped water	(2) Toilet or cesspit	(3) Birth order/number of children	(4) Cohort size last child born alive
In utero—3rd trim. (Std)	−0.038	0.001	0.006	−0.003
	(0.41)	(0.98)	(0.04)	(0.59)
	[−0.142, 0.137]	[−0.141, 0.139]	[0.001, 0.011]	[−0.020, 0.012]
In utero—1st trim. (Std)	−0.128	−0.100	0.003	−0.001
	(0.53)	(0.23)	(0.22)	(0.59)
	[−0.251, 0.167]	[−0.327, 0.114]	[−0.005, 0.014]	[−0.014, 0.005]
R^2	0.994	0.994	0.992	0.998
Observations	2208	2208	2208	2208

All regressions include month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order/ number of children, ethnicity, age, and education of the mother, whether they have access to piped water at home, a toilet, and temperature and rainfall by trimester in-utero, except when used as the placebo variable. *p*-values of wild bootstrap inferences shown in parentheses and 95-percent confidence intervals shown in brackets

Table 13 Placebos 3

	(1) Temperature 3rd trimester	(2) Temperature 1st trimester	(3) Rainfall 3rd trimester	(4) Rainfall 1st trimester
In utero—3rd trim. (Std)	−0.022	0.044	1.223	0.121
	(0.18)	(0.01)	(0.07)	(0.85)
	[−0.067, 0.030]	[0.012, 0.099]	[−0.129, 3.356]	[−1.621, 1.968]
In utero—1st trim. (Std)	0.004	−0.006	0.460	1.361
	(0.71)	(0.58)	(0.30)	(0.09)
	[−0.059, 0.023]	[−0.027, 0.036]	[−0.918, 2.245]	[−0.651, 5.784]
R^2	0.998	0.998	0.980	0.977
Observations	2208	2208	2208	2208

All regressions include month-year of birth fixed effects, month-region fixed effect, and region-specific trends. Control variables include: birth order/ number of children, ethnicity, age, and education of the mother, whether they have access to piped water at home, a toilet, and temperature and rainfall by trimester in-utero, except when used as the placebo variable. *p*-values of wild bootstrap inferences shown in parentheses and 95-percent confidence intervals shown in brackets

between temperature in the third trimester in utero and the variation of the epidemic that we exploit in that same trimester. Likewise, column 2 reveals that there is no significant correlation between temperature in the first trimester in utero and the variation of the epidemic that we exploit in that same trimester. Columns 3 and 4 show rainfall is indeed positively correlated with cholera incidence. Nevertheless, including or not rainfall as a control in our regressions has virtually no effect in our results except for additional statistical significance in some of our estimates. The lack of sensitivity of our results to the inclusion on temperature and rainfall as control variables is in part due to the inclusion of month-region fixed effects in our regressions, which diminish the role of seasonal factors on childhood mortality.

We test the sensitivity of our results to other specifications and samples, including children born to women who did not relocate to a different district during the 5 years preceding the census (between 1988 and 1993), and our results are very similar. We also show that the estimated effect on mortality is concentrated on those children born in the first half of 1991, that is, those who were already in utero when the epidemic broke out. Please, refer to Tables A.4 and A.5 of the working paper (Ritter and Sanchez 2020) to see these results.

As we mentioned in Section 4, a Fuzzy-DD approach has two important and somewhat strong assumptions: a stable treatment effect over time and homogeneous treatment effects for switchers of both treatment and comparison groups. We relax these assumptions and estimate Intertemporal Treatment Effects following de Chaisemartin and D'Haultfœuille (2020) and exploiting the Stata command "Did-Multiplegt" (de Chaisemartin et al. 2019). Please, refer to Figure A.1 to Figure A.3 and Table A.6 of the working paper (Ritter and Sanchez 2020) to see these results. This exercise shows that the Fuzzy-DiD assumptions are too strong for our setting but the bias seems to go against our results. Since, our Fuzzy-DiD model is more conservative and it has more statistical power we keep it as the main specification of the study.

Appendix B. Robustness survey data

Table 14 presents placebo regressions for the correlation with birth order, temperature and rainfall. We do not observe any significant correlation. Additionally, we test the sensitivity of our results to other specifications. Please, refer to Tables B.2 to B.7 of the working paper (Ritter and Sanchez 2020) to see these results. Our main results are very robust to the different specifications. Finally, we do not find any significant effect of exposure to cholera during the first trimester after birth or during one trimester before conception.

Table 14 Placebos

	(1) Birth order	(2) Temperature 3rd tr.	(3) Temperature 1st tr.	(4) Rainfall 3rd tr.	(5) Rainfall 1st tr.
In utero—3rd tr. (Std)	−0.01	−0.01	0.09	1.67	−1.05
	(0.44)	(0.86)	(0.26)	(0.21)	(0.39)
	[−0.05, 0.04]	[−0.12, 0.10]	[−0.05, 0.23]	[−1.69, 7.46]	[−5.82, 2.56]
In utero—1st tr. (Std)	0.01	0.08	0.05	0.19	1.49
	(0.25)	(0.22)	(0.12)	(0.66)	(0.36)
	[−0.01, 0.03]	[−0.07, 0.16]	[−0.03, 0.15]	[−1.12, 2.00]	[−3.54, 9.39]
R^2	0.904	0.956	0.959	0.945	0.938
Observations	9155	9155	9155	9155	9155

Note: All regressions include mother fixed effect, month-year of birth fixed effects, month-region fixed effect, and region-specific trends. *p*-values of wild bootstrap inferences shown in parentheses and 95% confidence intervals shown in brackets

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