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Prenatal Exposure to Acute Diarrheal Diseases and Childhood Mortality

Patricia I. Ritter*and Ricardo Sanchez‡

August, 2019

Abstract

There is a large body of evidence that shows the effect of diarrheal diseases on childhood mortality. Nevertheless, most of this literature focuses on post-natal exposure to diarrheal diseases. This study exploits the Cholera Epidemic in Peru, finding that a 1% point increase in cholera incidence in the third trimester in-utero increases average childhood mortality rate by 0.2% points or 14%. This study suggests that public programs that aim to reduce diarrheal diseases should target not just children but also pregnant women and raises the question of whether pregnant women should take vaccines to prevent diarrheal diseases in poor countries.

Keywords: acute diarrheal diseases; cholera; clean water; in-utero.

JEL Codes: I15, I18, O10

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1 Introduction

Diarrheal disease is the second leading cause of death of children under five years old. The World Health Organization (WHO) estimates that each year diarrhea kills around 525 000 children under five. This number, however, under-estimates the total effect of diarrheal diseases on childhood mortality, if *prenatal* exposure to diarrheal diseases has an effect on childhood mortality rate.

This study exploits the cholera epidemic in Peru in the early 1990s to explore whether in-utero exposure to an acute diarrheal disease affects childhood mortality. Cholera is an acute, diarrheal disease generated by a bacterial infection. When the infection is severe, the main symptom is profuse diarrhea that causes dehydration. Among pregnant women, the severe dehydration Cholera produces is associated with a reduction of the amniotic fluid, reduced blood flood to the placenta, placental hemorrhage, hypotension, pre-term births and miscarriages (Grados and Battilana, 1994; Grout et al., 2015). Given the severity of the potential effects of an acute diarrheal disease on maternal-child health and the vast evidence that in-utero is such a sensitive period in the formation of health capital (Almond and Currie, 2011), including childhood mortality (Jayachandran, 2009)[1], it is possible that the causal effect of acute diarrheal diseases on childhood mortality begins even before children are born.

We apply a Difference-in-Difference methodology exploiting differences in the intensity of the cholera outbreak by month and geographical location. Event studies show that previous trends do not explain our results. We find that prenatal exposure

[1] Other important studies that investigate *post-natal* early childhood shocks on mortality rate are included in the review of Almond, Currie and Duque (2018).

to a high incidence of cholera has an important effect on childhood mortality rate. Additionally, we exploit heterogeneous effects by the year of the epidemic. Rapid treatment to correct dehydration can effectively reduce mortality from cholera infection. Since the shock was unexpected when it appeared, we would expect a higher effect on mortality in the first year of the epidemic, 1991, and at the same time, the effect in this year is less likely to be endogenous. As expected, we find that the effect on mortality is concentrated among those children who were in utero in 1991: a 1% point incidence of cholera during the 3rd trimester in-utero increases average childhood mortality rate by 0.2% points or 10%. Finally, we explore heterogeneous effects by ethnicity exploiting the fact that individuals blood type O are more likely to suffer from severe cases of cholera (Harris et al., 2005) and that indigenous population in Peru are by large blood type O (Matson et al. 1966). We find that the effect on mortality is significantly larger on indigenous children: 1% point incidence of cholera during the 3rd trimester in-utero increases indigenous childhood mortality rate by 0.7% points or 22%. It is important to note that our results are a lower bound estimate for a couple of reasons: first, the epidemic likely generated an sizable number of miscarriages, which tend to be concentrated among those who are the weakest, meaning the survivors will tend to be a healthier subpopulation (Bozzoli, S. Deaton and Quintana-Domeque, 2007). Second, we observe only whether children of cohort 1991 have died until 1993, that is, when they are 3 years old or less, and these children might have died after 1993.

This study makes several contributions. First, it builds on the literature about in-utero shocks with evidence of early-life impacts. There is significant literature on the long term consequences of in-utero shocks, but much less do we know about

the early-life impacts (Almond and Currie, 2011), in particular about the effect on childhood mortality. Second, this study contributes to a better understanding of the relationship between diarrheal diseases and childhood mortality. To the best of our knowledge, this is the first study to show that prenatal exposure to a diarrheal disease increases childhood mortality. Cholera is not a rare disease; approximately 1.4 billion people are 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. Annually, approximately 91,000 and 2,500 people die of cholera in endemic countries and non-endemic countries, respectively (Ali et al., 2012). And these numbers do not include estimations about the impact on mortality derived by in-utero exposure to the disease. Furthermore, because acute diarrhea is the main effect of a Cholera infection, and all the other consequences are derived from this main effect, we believe our results are informative not only with respect to the effects of cholera epidemics but also about acute diarrheal diseases, in general. Hence public programs that aim to reduce childhood mortality due to diarrheal diseases should target not just children but also pregnant women. Moreover, evaluation of these programs might be underestimating the benefits if they do not measure the impact on children who were in-utero at the time of the program. Finally, although universal access to improved water and sanitation is desirable, this is unattainable for many countries (Zwane and Kremer, 2007). This study, then, raises the question of whether cholera vaccine, in particular, and vaccines to prevent other diarrheal diseases, in general, should be prescribed for pregnant women in such countries.

2 The Cholera Epidemic in Peru

In the early 1990s, Peru suffered a cholera epidemic, reaching 322,562 suspected cases of cholera with a total of 2,909 deaths (mortality rate 9 per 100 000 inhabitants) 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 (Mujica, Gomez Mujica and Gomez, 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*a*).

Figure 1 shows 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 figure 2. By 1992, it seems most people had already been warned about the epidemic and had more knowledge about how to prevent it, including awareness of the symptoms and the treatment: i) rapid treatment to correct severe dehydration, using intravenous saline solution ii) adequate use of oral hydration salts and iii) immediate availability of medications, especially intravenous sera (Gotuzzo, 1991; Maguiña Vargas et al., 2010*a*). Additionally, the Ministry of Health made efforts to strengthen the clinical management of patients suffering from dehydration (Lanata, 1989) and the dissemination of personal hygiene, food hygiene and fast treatment of patients. In addition, environmental measures were adopted, such as the chlo-

rination and monitoring of existing water supply systems in urban areas and the distribution of chemical products for water purification in homes and comparators for the monitoring of residual chlorine (Maguiña Vargas et al., 2010*a*).

Several factors seemed to influence the intensity of the disease by region and time. The first of these is the geographical location (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 (Gotuzzo, 1991; Maguiña Vargas et al., 2010*b*). Importantly, this is believed to be the first case, not just the first time it was diagnosed because for some time before 1991 groups of adults and children were tested and the bacterium *Vibrium Cholerae* was never identified (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 (Maguiña Vargas et al., 2010*a*). High temperatures (Lama et al., 2004) and poor levels of basic sanitation, with inadequate processing of drinking water (Gotuzzo, 1991; Maguiña Vargas et al., 2010*b*) also seem to influence the expansion of the epidemic. Finally, belonging to the human blood group O has been associated with the severity of the disease. Individuals with blood type O are not more likely to be infected by the bacterium but 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 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

The data used in this paper uses two sources of information. The first source 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. This information is disaggregated by week, year, and region (with the exception of Apurimac and Madre de Dios). The second source of data is the Population Census of Peru of 1993, which asks women age 12 or older whether their youngest child born alive is still alive. They also ask the month and year of birth of that child. The mortality rate correspond to the proportion of those children by cohort and region who have died. We work with 10-year cohort of children; those children born between 1984 and 1993.

Table 1 shows summary statistics of our sample of children that corresponds to the youngest child born between 1984 and 1993 to women age 12 or older in 1993. As we would expect, children born between 1984 and 1993 are in 1993 on average 4 years old. The mortality rate is 1.4%, the average age of the mother, 31. She has an average of 3 children in total. 16% of the mothers are illiterate, with an average educational attainment is 11 years. 55% of the children has piped water at home, 48% has indoor toilets, 67% has electricity, and 42% has adequate roofing materials. The table also shows that indigenous children are poorer, their mothers less educated, and their mortality rate is higher. Finally, the table show the average cholera incidence per year. In 1991, the average cholera incidence per trimester was 0.11 per 100 inhabitants.

4 Empirical Strategy

We begin by estimating the following

Difference-in-Difference regression to estimate the effect of cholera incidence on mortality rate:

$$Y_{r,m,t} = \beta_o + \beta_1 Ch^1_{r,m,t} + \beta_2 Ch^2_{r,m,t} + \beta_3 Ch^3_{r,m,t} + \beta_4 X_{r,m,t} + \alpha_r + \gamma_r t + \rho_{m,t} + \varepsilon_{r,t}$$

where $Y_{i,r,t}$ stands for the mortality rate of children born in month m, and year t to women age 12 or more in 1993, who currently lives in region r. $Ch^i_{r,m,t}$ stands for the cholera incidence that correspond to ith trimester in-utero of the child. For example, $Ch^3_{Ica,4,1992}$ represents the 12-week accumulated incidence before April 15th[2], 1992 in the region of Ica. This estimation allows us to identify which trimester(s) is (are) the critical in terms of the exposure to Cholera and its effect on mortality. The incidence for all cohorts born before 1991 is 0, since the first case appeared in 1991. α_r stands for region fixed effects, $\gamma_r t$ stands region-specific trends, $\rho_{m,t}$ stands for month-year of birth fixed effects, and $X_{r,m,t}$ stands for control variables: ethnicity, age, literacy, education of the mother, whether or not they have access to piped water, indoor toilet, electricity, number of assets at home, and whether the roofing material of the house is adequate. Standard errors are clustered at the regional level. Since we only have data from 22 regions, we present the p-values of Wild Bootstrap inferences, to correct for the small number of regions, following Cameron, Gelbach

[2]The Census does not provide day of birth, so we assume all children were born on the 15th day of the month. The Census does also not provide information on preterm births, hence we assume nine months of gestation.

and Miller (2008).

We then explore differences in the impact on mortality by the year of the epidemic. Rapid treatment to correct dehydration can effectively reduce mortality from cholera infection, but the shock was unexpected when it appeared. Thus, in the first year of the epidemic, 1991, we would expect a higher effect on mortality and at the same time we would expect that the effect is less likely to be endogenous. In order to analyze differences in the impact on mortality by year, we estimate the following regression:

$$Y_{r,m,t} = \beta_o + \beta_1 Ch^3_{r,m,t} t_{1991} + \beta_2 Ch^3_{r,m,t} t_{1992} + \beta_3 Ch^3_{r,m,t} t_{1993} + \beta_4 X_{r,m,y} + \alpha_r + \gamma_r t + \rho_{m,y} + \varepsilon_{r,t}$$

where $Ch^3_{r,m}$ stands for cholera incidence corresponding to the third trimester in-utero. We chose the cholera incidence during the third trimester in utero, because, as we will see later, this is when the effect on mortality is concentrated. t_i stands for a dummy equal to 1 for year i.

Finally, we estimate the following event-study specification:

$$Y_{r,m,t} = \alpha_o + \sum_{t=1984}^{1993} \beta_t Ch^{3,1991}_{r,m} t + \alpha_1 X_{r,m,y} + \alpha_2 Ch^{3,1992}_{r,m} t_{1992} + \alpha_3 Ch^{3,1993}_{r,m} t_{1993} + \alpha_r + \rho_{m,y} + \varepsilon_{r,t}$$

$Ch^{1991}_{r,m}$ stands for cholera incidence corresponding to the third trimester in-utero of the child of region r born in month m of year 1991. We chose the cholera incidence for year 1991, because, as we will see later, this is when the effect on mortality is concentrated. Thus, β_{1991} would give us the effect of the exposure to 1 percentage point increase in cholera incidence during the third trimester in-utero of the child in

region r that was born in month m of 1991. β_t of other cohorts of children give us the placebo effect on mortality of rate among children that were born in the same region an in the same month but in years previous to or after 1991. For example, β_{1990} would give us the effect of the exposure to 1 percentage point increase in cholera incidence of 1991 one year after the child was in his third trimester in-utero and β_{1992} would give us the effect of the exposure to 1 percentage point increase in cholera incidence of 1991 one year before the child was in his third trimester in-utero. Since in 1992 and 1993 the cholera epidemic was not completely over and the cholera incidence of 1992 and 1993 is very likely correlated with the cholera incidence of 1991, we control for the actual cholera incidence on those years ($Ch_{r,m}^{1992}t_{1992}$ and $Ch_{r,m}^{1992}t_{1992}$). The purpose of this estimation is to test whether mortality rate on those regions and months that had a higher incidence of cholera was already increasing before the epidemic.

5 Results

5.1 Average Effects

Table 2 presents the effects of prenatal exposure to cholera. It shows the average effect on all cohorts born between 1984 and 1993, that is, on all children younger than 10 in 1993. The first column shows the effects of estimating the difference-in-difference specification without adding any control variable or trend. We can see that only exposure during the 3rd trimester has effects on childhood mortality. According to this estimate, exposure to 1% point incidence of cholera during the

3rd trimester in-utero increases average childhood mortality rate by 0.3% points or 20%. Including the age of the mother as a control variable reduces our estimates somewhat to 0.2% points. None of the other controls have an effect on our estimates, as we can see in column 3. Finally, the inclusion of region-specific trends also has no effect on our estimates.

Table 3 shows the effect of exposure to Cholera during the third trimester by the year of the epidemic. We can see that the effect of the cholera incidence in 1992 rapidly disappears as we control for age of the mother, suggesting as we suspected that the effects after the first year could be endogenous because the epidemic was no longer unexpected. According to our final and preferred estimate in column 4, a 1 percentage point incidence of cholera during the 3rd trimester in-utero in 1991 increases average childhood mortality rate by 0.2% points, or 14%, and cholera in the following years had no significant effect on mortality.

Figure 3 shows the estimations of the event study. We can see no previous trend in childhood mortality in those regions and months that had a higher incidence of cholera in 1991. This result provides good evidence that our estimations are causal.

5.2 Heterogeneous Effects by Ethnicity

We then estimate heterogeneous effects by ethnicity. As explained above, for biological reasons, blood type O individuals are more likely to suffer from severe cases of cholera (Harris et al., 2005), and more than 90% indigenous population in Peru blood type O (Matson et al., 1966), thus we would expect a larger effect on indigenous children.

Tables 4 and 5 present the effects of prenatal exposure to cholera among non-indigenous and indigenous children, respectively. We can see that the effects are larger in absolute terms for indigenous children. The inclusion of control variables, in this case, increases the significance of our estimate.

Tables 6 and 7 show the effect by year among non-indigenous and indigenous children, respectively. Again, we can see that the effect of the cholera incidence is concentrated in 1991. The estimate for indigenous children is larger and more significant, and it is not particularly sensitive to the inclusion of control variables and region-specific trends. According to our final and preferred estimate in column 4, 1 percentage point incidence of cholera during the 3rd trimester in-utero in 1991 increases indigenous childhood mortality rate by 0.8% points or 32%, and cholera in the following years had no significant effect on mortality.

Figure 4 shows the estimations of the event study for indigenous children. We can see no previous trend in childhood mortality in those regions and months that had a higher incidence of cholera in 1991. Again these results provide good evidence that we are estimating causal effects.

6 Robustness Analysis

The Difference-in-Difference strategy of this paper, in combination with the heterogeneous effects and the event studies provide credible evidence that exposure to cholera while in-utero increases childhood mortality rate. Nevertheless, in order to increase the reliability of our results, in this section, we test whether observable variables change significantly with the exposure to Cholera. Tables 8 to 15 show the placebo regressions using the control variables as dependent variables. Almost

all relevant coefficients are statistically insignificant, except for birth order and access to a toilet at home in our first specification, but the significances disappear when we estimate the effect of exposure during the third trimester in the first year of the epidemic (1991). Additionally, the coefficient on access to a toilet is positive so if anything, it would attenuate our results. Finally, we find a significant effect on having electricity at home when we estimate the effect of exposure during the third trimester in the first year of the epidemic (1991), however the estimate is not significant for indigenous children.

7 Conclusions

This paper exploits the Cholera Epidemic in Peru to analyze effects on childhood mortality of acute diarrheal diseases. We find that 1% point of cholera incidence in the third trimester in-utero increases average childhood mortality rate by 0.2% points or 14% and by 0.8% points or 32% on indigenous children. This study makes several contributions. First, it builds on the literature about in-utero shocks with evidence of effects on childhood mortality. Second, this study contributes to a better understanding of the relationship between diarrheal diseases and childhood mortality. To the best of our knowledge, this is the first study to show that prenatal exposure to a diarrheal disease increases childhood mortality. Since acute diarrhea is the main effect of a Cholera infection, and all the other consequences seem to derive from this main effect, we believe our results are informative not only regarding the effects of cholera epidemics but also acute diarrheal diseases more generally. Hence public programs that aim to reduce childhood mortality due to diarrheal dis-

eases should target pregnant women and not just children. Moreover, evaluation of these programs might be underestimating the benefits, if they do not measure the impact on children who were in-utero at the time of the program. Finally, this study raises the question of whether cholera vaccine, in particular, and vaccines to prevent other diarrheal diseases, in general, should be prescribed for pregnant women in poor countries.

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Figure 1: Weekly Incidence of Cholera Epidemic

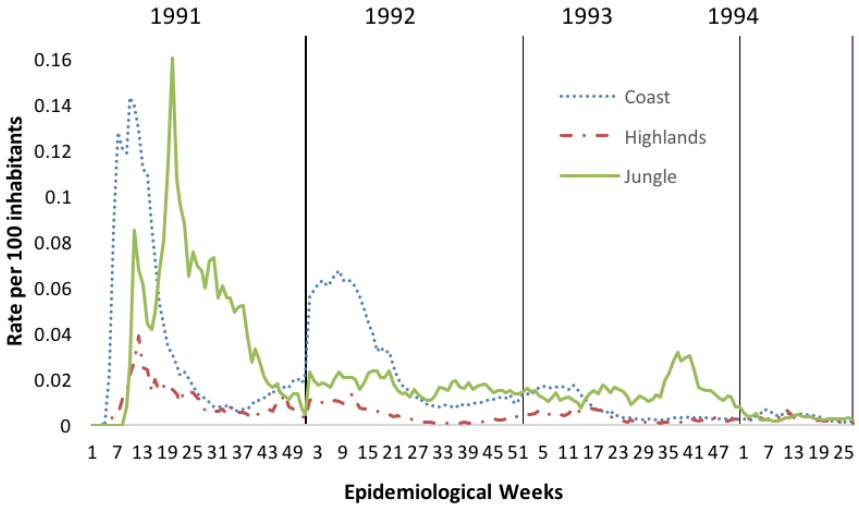


Figure 2: Incidence and Mortality of Cholera Epidemic

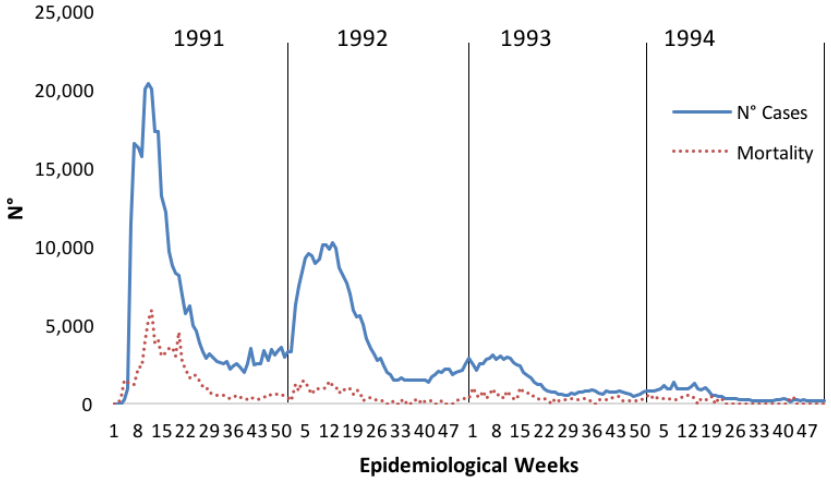
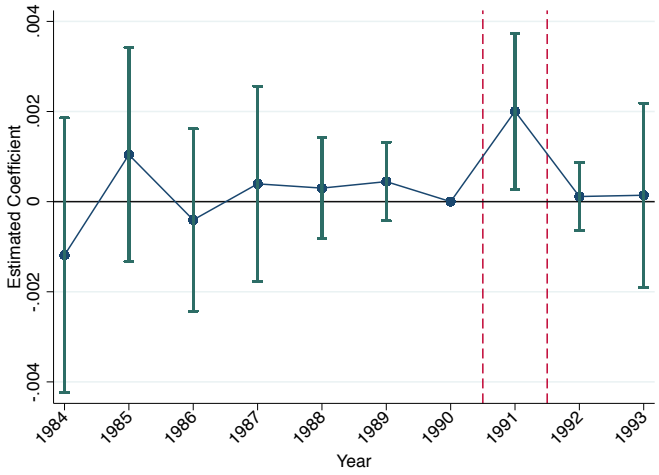
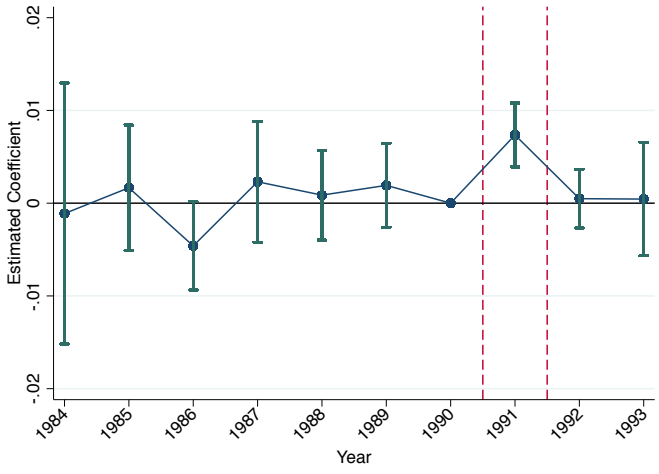


Figure 3: Event Study- Mortality Rate - Cholera Incidence 1991



Note: Graph includes point estimates from the event study (normalized to 0 in the the third trimester of 1990) and 95 percent confidence intervals of Wild Bootstrap inferences to correct for the small number of regions.

Figure 4: Event Study- Mortality Rate - Indigenous Cholera Incidence 1991



Note: Graph includes point estimates from the event study (normalized to 0 in the the third trimester of 1990) and 95 percent confidence intervals of Wild Bootstrap inferences to correct for the small number of regions.

Table 1: Summary Statistics

	Non-Indigenous	Indigenous	Total
Youngest child's age (alive or dead)	3.536	3.337	3.504
	(2.641)	(2.613)	(2.637)
Mortality rate	0.0123	0.0253	0.0144
	(0.110)	(0.157)	(0.119)
Mother's number of children	3.033	3.818	3.158
	(2.245)	(2.545)	(2.314)
Mother's age	31.29	32.06	31.42
	(7.923)	(8.256)	(7.982)
Illiterate	0.0161	0.0655	0.0240
	(0.126)	(0.247)	(0.153)
Mother's years of Education	10.74	7.722	10.26
	(4.204)	(3.153)	(4.203)
Piped water at home	0.596	0.330	0.554
	(0.491)	(0.470)	(0.497)
Toilet at home	0.521	0.252	0.478
	(0.500)	(0.434)	(0.500)
Electricity at home	0.715	0.448	0.673
	(0.451)	(0.497)	(0.469)
Number of assets	2.821	1.879	2.671
	(1.879)	(1.194)	(1.820)
House has a roof of adequate material	0.440	0.340	0.424
	(0.496)	(0.474)	(0.494)
Cholera - 1st Trim.(per 100 inhbts.)	0.116	0.0808	0.110
	(0.284)	(0.212)	(0.274)
Cholera - 2nd Trim.(per 100 inhbts.)	0.120	0.0855	0.114
	(0.278)	(0.211)	(0.269)
Cholera - 3rd Trim.(per 100 inhbts.)	0.119	0.0850	0.114
	(0.271)	(0.202)	(0.261)
Observations	1702048		

Table 2: Average Effects of Cholera Incidence on Childhood Mortality by Trimester In-Utero

	(1) No Controls	(2) Controls for Age	(3) All Controls	(4) All Controls and Trends
Cholera - 3rd Trim.	0.003***	0.002**	0.002*	0.002*
	(0.00)	(0.03)	(0.04)	(0.02)
	[0.00, 0.01]	[0.00, 0.00]	[0.00, 0.00]	[0.00, 0.00]
Cholera - 2nd Trim.	-0.000	-0.001	-0.001	-0.000
	(0.64)	(0.10)	(0.17)	(0.29)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
Cholera - 1st Trim.	0.001	0.000	0.000	0.001
	(0.23)	(0.80)	(0.45)	(0.36)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
R^2	0.759	0.765	0.765	0.776
Observations	2760	2760	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Control variables include: age, literacy, education, ethnicity of the mother, whether they have access to piped water, toilets, electricity, number of assets at home, and adequate roofing material. Trends are region-specific.

Table 3: Average Effects of Cholera Incidence during the 3rd. Trimester In-Utero on Childhood Mortality by the Year of the Epidemic

	(1) No Controls	(2) Controls for Age	(3) All Controls	(4) All Controls and Trends
Cholera x 1991	0.003*	0.002*	0.002*	0.002*
	(0.03)	(0.04)	(0.05)	(0.05)
	[0.00, 0.01]	[0.00, 0.01]	[-0.00, 0.01]	[-0.00, 0.01]
Cholera x 1992	0.003**	0.001	0.001	0.001
	(0.05)	(0.60)	(0.58)	(0.35)
	[0.00, 0.01]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
Cholera x 1993	-0.003	-0.008	-0.006	-0.004
	(0.70)	(0.27)	(0.37)	(0.48)
	[-0.02, 0.01]	[-0.03, 0.01]	[-0.02, 0.01]	[-0.02, 0.01]
R^2	0.759	0.766	0.766	0.776
Observations	2760	2760	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Control variables include: age, literacy, education, ethnicity of the mother, whether they have access to piped water, toilets, electricity, number of assets at home, and adequate roofing material. Trends are region-specific.

Table 4: Effects of Cholera Incidence on Non-Indigenous Childhood Mortality by Trimester In-Utero

	(1) No Controls	(2) Controls for Age	(3) All Controls	(4) All Controls and Trends
Cholera - 3rd Trim.	0.001	0.000	0.000	0.001
	(0.12)	(0.50)	(0.72)	(0.36)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
Cholera - 2nd Trim.	0.000	-0.000	-0.000	-0.000
	(0.74)	(0.48)	(0.50)	(0.82)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
Cholera - 1st Trim.	0.001	0.000	0.000	0.000
	(0.29)	(0.84)	(0.88)	(0.56)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
R^2	0.628	0.649	0.659	0.675
Observations	2760	2760	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Control variables include: age, literacy, education, ethnicity of the mother, whether they have access to piped water, toilets, electricity, number of assets at home, and adequate roofing material. Trends are region-specific.

Table 5: Effects of Cholera Incidence on Indigenous Childhood Mortality by Trimester In-Utero

	(1) No Controls	(2) Controls for Age	(3) All Controls	(4) All Controls and Trends
Cholera - 3rd Trim.	0.003***	0.002**	0.002*	0.002*
	(0.00)	(0.03)	(0.04)	(0.02)
	[0.00, 0.01]	[0.00, 0.00]	[0.00, 0.00]	[0.00, 0.00]
Cholera - 2nd Trim.	-0.000	-0.001	-0.001	-0.000
	(0.64)	(0.10)	(0.17)	(0.29)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
Cholera - 1st Trim.	0.001	0.000	0.000	0.001
	(0.23)	(0.80)	(0.45)	(0.36)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
R^2	0.759	0.765	0.765	0.776
Observations	2760	2760	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Control variables include: age, literacy, education, ethnicity of the mother, whether they have access to piped water, toilets, electricity, number of assets at home, and adequate roofing material. Trends are region-specific.

Table 6: Effects of Cholera Incidence during the 3rd. Trimester In-Utero on Non-Indigenous Childhood Mortality by the Year of the Epidemic

	(1)	(2)	(3)	(4)
	No Controls	Controls for Age	All Controls	All Controls and Trends
Cholera x 1991	0.001	0.001	0.001	0.001
	(0.26)	(0.36)	(0.38)	(0.20)
	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
Cholera x 1992	0.003	-0.000	-0.001	-0.000
	(0.11)	(0.77)	(0.45)	(0.88)
	[-0.00, 0.01]	[-0.00, 0.00]	[-0.00, 0.00]	[-0.00, 0.00]
Cholera x 1993	0.006	0.000	-0.000	0.000
	(0.26)	(0.91)	(0.91)	(0.92)
	[-0.00, 0.02]	[-0.01, 0.01]	[-0.01, 0.01]	[-0.01, 0.01]
R^2	0.628	0.649	0.659	0.675
Observations	2760	2760	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Control variables include: age, literacy, education, ethnicity of the mother, whether they have access to piped water, toilets, electricity, number of assets at home, and adequate roofing material. Trends are region-specific.

Table 7: Effects of Cholera Incidence during the 3rd. Trimester In-Utero on Indigenous Childhood Mortality by the Year of the Epidemic

	(1)	(2)	(3)	(4)
	No Controls	Controls for Age	All Controls	All Controls and Trends
Cholera x 1991	0.008***	0.007***	0.007***	0.008***
	(0.01)	(0.00)	(0.00)	(0.01)
	[0.00, 0.02]	[0.00, 0.01]	[0.00, 0.01]	[0.00, 0.01]
Cholera x 1992	0.003	0.001	0.002	0.003
	(0.35)	(0.86)	(0.47)	(0.29)
	[-0.02, 0.02]	[-0.01, 0.01]	[-0.01, 0.02]	[-0.00, 0.01]
Cholera x 1993	-0.023*	-0.029**	-0.024	-0.017
	(0.13)	(0.08)	(0.27)	(0.35)
	[-0.05, 0.01]	[-0.06, 0.01]	[-0.06, 0.02]	[-0.05, 0.03]
R^2	0.458	0.462	0.463	0.474
Observations	2698	2698	2534	2534

* p<0.1, ** p<0.05, *** p<0.01

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Control variables include: age, literacy, education, ethnicity of the mother, whether they have access to piped water, toilets, electricity, number of assets at home, and adequate roofing material. Trends are region-specific.

Table 8: Placebo Effects of Cholera Incidence on Childhood Mortality by Trimester In-Utero

	(1) Birth Order	(2) Indigenous	(3) Mother's Age	(4) Mother Illiterate	(5) Mother's Education
Cholera - 3rd Trim.	0.021*	-0.001	0.056	0.000	0.017
	(0.09)	(0.72)	(0.20)	(0.83)	(0.39)
	[-0.00, 0.05]	[-0.01, 0.01]	[-0.02, 0.19]	[-0.00, 0.01]	[-0.03, 0.09]
Cholera - 2nd Trim.	0.010	-0.000	0.012	-0.001	0.009
	(0.10)	(0.98)	(0.57)	(0.58)	(0.57)
	[-0.00, 0.03]	[-0.00, 0.01]	[-0.03, 0.08]	[-0.01, 0.00]	[-0.04, 0.06]
Cholera - 1st Trim.	-0.000	0.002	-0.006	0.004*	-0.016
	(0.98)	(0.33)	(0.75)	(0.09)	(0.34)
	[-0.02, 0.02]	[-0.00, 0.01]	[-0.06, 0.04]	[-0.00, 0.01]	[-0.08, 0.03]
R^2	0.992	0.997	0.997	0.989	0.990
Observations	2640	2640	2640	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.

Table 9: Placebo Effects of Cholera Incidence during the 3rd. Trimester In-Utero on Childhood Mortality by the Year of the Epidemic

	(1) Birth Order	(2) Indigenous	(3) Mother's Age	(4) Mother Illiterate	(5) Mother's Education
Cholera x 1991	0.012	-0.002	0.058	0.000	0.024
	(0.43)	(0.15)	(0.33)	(0.98)	(0.31)
	[-0.03, 0.04]	[-0.00, 0.00]	[-0.04, 0.28]	[-0.00, 0.01]	[-0.03, 0.15]
Cholera x 1992	0.059**	0.001	0.065	-0.002	0.016
	(0.01)	(0.89)	(0.34)	(0.65)	(0.75)
	[0.02, 0.11]	[-0.02, 0.02]	[-0.08, 0.19]	[-0.01, 0.01]	[-0.08, 0.12]
Cholera x 1993	0.063	0.007	-0.038	-0.035**	-0.191
	(0.45)	(0.55)	(0.87)	(0.01)	(0.49)
	[-0.13, 0.26]	[-0.02, 0.03]	[-0.49, 0.69]	[-0.07, -0.01]	[-0.70, 0.32]
R^2	0.992	0.997	0.997	0.989	0.990
Observations	2640	2640	2640	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.

Table 10: Placebo Effects of Cholera Incidence on Childhood Mortality by Trimester In-Utero

	(1) Piped Water	(2) Toilet	(3) Electricity	(4) Num. Assets	(5) Adequate Roof
Cholera - 3rd Trim.	-0.000	0.004	-0.001	-0.007	0.002
	(0.88)	(0.07)	(0.25)	(0.14)	(0.71)
	[-0.00, 0.00]	[-0.00, 0.01]	[-0.00, 0.00]	[-0.02, 0.00]	[-0.01, 0.01]
Cholera - 2nd Trim.	0.002*	0.001	-0.002*	-0.011	0.000
	(0.07)	(0.40)	(0.06)	(0.17)	(0.93)
	[-0.00, 0.00]	[-0.00, 0.01]	[-0.00, 0.00]	[-0.03, 0.01]	[-0.01, 0.01]
Cholera - 1st Trim.	-0.002	0.004*	0.002*	-0.015	-0.003
	(0.24)	(0.06)	(0.02)	(0.03)	(0.43)
	[-0.00, 0.00]	[-0.00, 0.01]	[0.00, 0.01]	[-0.04, -0.00]	[-0.01, 0.00]
R^2	0.997	0.997	0.997	0.996	0.992
Observations	2640	2640	2640	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.

Table 11: Placebo Effects of Cholera Incidence during the 3rd. Trimester In-Utero on Childhood Mortality by the Year of the Epidemic

	(1) Piped Water	(2) Toilet	(3) Electricity	(4) Num. Assets	(5) Adequate Roof
Cholera x 1991	-0.001	0.002	-0.002	-0.001	0.004
	(0.36)	(0.24)	(0.16)	(0.85)	(0.37)
	[-0.00, 0.00]	[-0.00, 0.01]	[-0.00, 0.00]	[-0.01, 0.01]	[-0.00, 0.01]
Cholera x 1992	0.005	0.008	-0.002	-0.028	-0.001
	(0.09)	(0.23)	(0.40)	(0.07)	(0.86)
	[-0.00, 0.01]	[-0.01, 0.02]	[-0.01, 0.00]	[-0.07, 0.00]	[-0.01, 0.01]
Cholera x 1993	0.009	0.010	0.004	0.044	0.021*
	(0.10)	(0.26)	(0.64)	(0.52)	(0.04)
	[-0.00, 0.02]	[-0.02, 0.03]	[-0.02, 0.02]	[-0.08, 0.18]	[0.00, 0.04]
R^2	0.997	0.997	0.997	0.996	0.992
Observations	2640	2640	2640	2640	2640

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.

Table 12: Placebo Effects of Cholera Incidence on Indigenous Childhood Mortality by Trimester In-Utero

	(1) Birth Order	(2) Mother's Age	(3) Mother Illiterate	(4) Mother's Education
Cholera - 3rd Trim.	0.023	0.061	0.012	0.045
	(0.45)	(0.40)	(0.14)	(0.30)
	[-0.04, 0.11]	[-0.10, 0.20]	[-0.01, 0.03]	[-0.04, 0.16]
Cholera - 2nd Trim.	0.007	0.039	-0.004	0.085*
	(0.78)	(0.59)	(0.37)	(0.03)
	[-0.04, 0.08]	[-0.17, 0.21]	[-0.02, 0.01]	[0.01, 0.21]
Cholera - 1st Trim.	-0.017	0.005	0.014**	0.049
	(0.62)	(0.97)	(0.07)	(0.17)
	[-0.13, 0.04]	[-0.22, 0.32]	[-0.00, 0.03]	[-0.03, 0.14]
R^2	0.959	0.989	0.970	0.817
Observations	2534	2534	2534	2534

* p<0.1, ** p<0.05, *** p<0.01

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.

Table 13: Placebo Effects of Cholera Incidence during the 3rd. Trimester In-Utero on Indigenous Childhood Mortality by the Year of the Epidemic

	(1) Birth Order	(2) Mother's Age	(3) Mother Illiterate	(4) Mother's Education
Cholera x 1991	0.012	0.097	0.006	0.052
	(0.75)	(0.29)	(0.51)	(0.24)
	[-0.07, 0.16]	[-0.07, 0.25]	[-0.01, 0.04]	[-0.02, 0.21]
Cholera x 1992	0.064*	0.010	0.016	0.087**
	(0.12)	(0.96)	(0.51)	(0.08)
	[-0.03, 0.18]	[-0.23, 0.28]	[-0.01, 0.04]	[-0.01, 0.28]
Cholera x 1993	0.221**	-0.264	-0.027	-0.174
	(0.11)	(0.34)	(0.32)	(0.37)
	[-0.07, 0.40]	[-1.32, 0.39]	[-0.08, 0.03]	[-0.54, 0.18]
R^2	0.959	0.989	0.970	0.817
Observations	2534	2534	2534	2534

* p<0.1, ** p<0.05, *** p<0.01

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.

Table 14: Placebo Effects of Cholera Incidence on Indigenous Childhood Mortality by Trimester In-Utero

	(1) Piped Water	(2) Toilet	(3) Electricity	(4) Num. Assets	(5) Adequate Roof
Cholera - 3rd Trim.	0.000	0.007*	-0.000	-0.009	-0.001
	(0.77)	(0.08)	(0.79)	(0.25)	(0.47)
	[-0.00, 0.01]	[-0.00, 0.02]	[-0.01, 0.00]	[-0.03, 0.01]	[-0.00, 0.00]
Cholera - 2nd Trim.	0.001	-0.003	-0.001	-0.005	0.008
	(0.73)	(0.59)	(0.88)	(0.44)	(0.45)
	[-0.00, 0.01]	[-0.02, 0.01]	[-0.01, 0.01]	[-0.02, 0.02]	[-0.01, 0.02]
Cholera - 1st Trim.	-0.002	0.003	0.002	-0.019**	-0.005
	(0.56)	(0.32)	(0.26)	(0.04)	(0.46)
	[-0.01, 0.01]	[-0.00, 0.01]	[-0.00, 0.01]	[-0.04, -0.00]	[-0.02, 0.01]
R^2	0.985	0.988	0.987	0.977	0.975
Observations	2534	2534	2534	2534	2534

* p<0.1, ** p<0.05, *** p<0.01

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.

Table 15: Placebo Effects of Cholera Incidence during the 3rd. Trimester In-Utero on Indigenous Childhood Mortality by the Year of the Epidemic

	(1) Piped Water	(2) Toilet	(3) Electricity	(4) Num. Assets	(5) Adequate Roof
Cholera x 1991	-0.001	0.005	-0.001	-0.004	-0.001
	(0.32)	(0.25)	(0.78)	(0.58)	(0.63)
	[-0.00, 0.00]	[-0.00, 0.02]	[-0.01, 0.00]	[-0.03, 0.01]	[-0.01, 0.00]
Cholera x 1992	0.006*	0.008	-0.001	-0.016	0.010**
	(0.05)	(0.46)	(0.79)	(0.41)	(0.10)
	[-0.00, 0.01]	[-0.01, 0.03]	[-0.01, 0.01]	[-0.08, 0.02]	[-0.01, 0.03]
Cholera x 1993	-0.001	0.018	0.012	0.080**	0.047*
	(0.89)	(0.19)	(0.56)	(0.07)	(0.22)
	[-0.03, 0.03]	[-0.01, 0.06]	[-0.02, 0.04]	[-0.01, 0.13]	[-0.05, 0.13]
R^2	0.985	0.988	0.987	0.977	0.976
Observations	2534	2534	2534	2534	2534

* p<0.1, ** p<0.05, *** p<0.01

Note: P-values of Wild Bootstrap inferences shown in round parentheses and 95-percent confidence intervals shown in square parentheses. Regressions include all control variable, except from the dependent variable, and region-specific trends.