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Short Report

Long-run effects of early childhood exposure to cholera on final height: Evidence from industrializing Japan☆

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ABSTRACT

Pandemic cholera is one of the most topical and urgent issues in many developing countries. However, although a growing body of research has shown the negative long-run effects of infectious disease exposure on human health, the long-run influences of early childhood exposure to cholera have thus far been understudied. To bridge this gap in the body of knowledge, we draw both on new data describing adult height from 1899 to 1910 from comprehensive official Japanese army records and on data recording the regional variation in the intensity of cholera pandemics. By using a difference-in-differences estimation strategy, we find that exposure to pandemic cholera had stunting effects on the final height of men at that time. Our estimates also suggest that early-infancy exposure to cholera seems to have a stronger long-run effect on adult height than late-infancy exposure.

1. Introduction

One of the most topical and urgent issues in many developing countries is cholera pandemics due to the spread of the *Vibrio cholerae* bacillus (Harris et al., 2012). The current (seventh) cholera pandemic that started in 1961 has not yet converged and continues to cause a heavy burden worldwide (Hu, Liu, & Feng, 2016). Indeed, recent estimates show 2.9 million that cases of infections and 95,000 deaths occur every year, predominantly in Asia and Africa (Ali, Nelson, Lopez, & Sack, 2015; Didelot, Pang, & Shou, 2015). Although the mechanisms of cholera epidemics have been widely investigated, the persistent effects of pandemic cholera on human health have been understudied (Mutreja, Kim, & Thomson, 2011).

The long-run adverse effects of early-life exposure to infectious diseases on health and socioeconomic outcomes have attracted wide attention. A growing body of the literature has examined the impacts of malaria (Barofsky, Anekwe, & Chase, 2015), typhoid fever (Beach, Ferrie, Saavedra, & Troesken, 2016), yellow fever (Saavedra, 2017), and influenza (Ogasawara, 2017). Almond and Currie (2011a) and Currie and Vogl (2013) provide comprehensive reviews of previous studies. As for historical cholera infections, Acemoglu and Johnson (2007) investigate the relationship between predicted mortality from various diseases including cholera and life expectancy. More recently, Ambrus, Field, and Gonzalez (2015) study the persistent effects of the cholera epidemic in 19th-century London on real estate prices. However, despite the frequent outbreak of cholera in the world, little is known about the potential long-run influences of early-life exposure to cholera on human health.

To bridge this gap in the body of knowledge, we estimate the effects of early childhood exposure to cholera on the final height of Japanese men. The strengths of the present study are as follows.

First, we draw on new data describing adult height from 1899 to 1910 by compiling comprehensive official Japanese army records. While previous studies have investigated the association between fetal flu exposure and adult height by using interview survey data (Mazumder et al., 2010), our dataset covers virtually the entire male population at age 20 at that time, which enables us to measure the overall impacts of the cholera epidemic on population health. The panel structure of the data allows us to compare the average height of affected and non-affected cohorts by using the difference-in-differences approach, controlling for a set of unobservable factors, which is difficult for the cross-sectional individual-level datasets often used in previous studies to deal with.

Second, we take advantage of the random variation in cholera epidemics throughout the Japanese archipelago to identify the effects of early-life cholera exposure on height. Since cholera is an acute watery diarrheal disease that leads to severe watery stools and diarrhea, maternal nutritional deprivation can be stronger than in other infectious

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diseases (Clemens et al., 2017). This fact implies that infection by the cholera bacterium could impose a heavy burden on the fetus and infants owing to the inadequate nutrition of mothers and infants and could thus be an appropriate exogenous treatment for testing the long-run exposure effects. Another advantage of our analysis is that we use the incidence rate rather than the mortality rate adopted in previous studies (Currie & Vogl, 2013), thereby minimizing the potential measurement error in our key exposure variable.

The present study offers evidence on the long-run adverse effects of early-life cholera exposure as a case study of industrializing Japan, a past-developing Asian country in which the public health environment was similar to those in current developing countries (Lin & Liu, 2014; Ogasawara, 2017). A growing body of research investigating the impacts of fetal exposure to infectious diseases has focused on developed countries for which detailed individual-level datasets are available (Almond & Currie, 2011a; Currie & Vogl, 2013). However, as for cholera epidemics, both South and East Asia are important pathogenic reservoirs and sources of international transmissions (Didelot et al., 2015). Therefore, the findings of this study could explain the potential long-run health effects of pandemic cholera on people in developing countries today, especially in Asia. Moreover, given the associations between wealth and human physique found in recent works (Bozzoli & Quintana-Domeque, 2014), understanding the impacts of exposure to cholera on physical development is an important research topic, especially for developing economies.

The structure of the remainder of the paper is as follows. Section 2 examines the empirical setting. Section 3 presents the main results. Section 4 discusses the results and concludes.

2. Empirical setting

2.1. Pandemic cholera and possible channels

Cholera was prevalent during the late 19th century in Japan. Having been brought to Japan in September 1877 on a British trading vessel, cholera spread throughout the country with devastating consequences. There were 162,637 infected people and 105,786 deaths in 1879, and 155,923 infected people and 108,405 deaths from cholera in 1886. As illustrated in Fig. 1, which shows the trend in the ratio of cholera infections and deaths, cholera continued to be prevalent until around 1890. The government quickly recognized polluted drinking water as the cause of infections by waterborne diseases, including cholera. Nevertheless, temporary measures were unable to lower the risk of cholera infection, as Fig. 1 shows (see Appendix A.1 for more details).

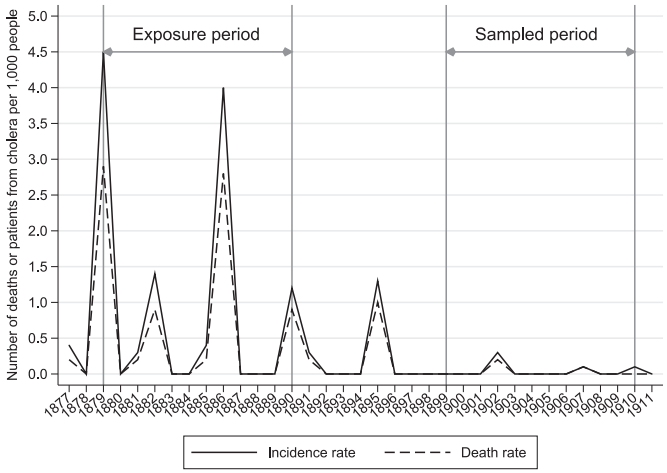

Fig. 1. Cholera pandemics in Japan, 1877–1911. Notes: The incidence rate is the number of cholera patients per 1,000 people. The death rate is the number of deaths from cholera per 1,000 people. Sources: See Appendix B.2.

Vibrio cholerae of the O1 or O139 serogroups cause cholera infection. Since cholera is an acute watery diarrheal disease, it leads to severe dehydration within hours and causes death if not treated adequately. The incubation period is approximately 18 hours to 5 days and symptoms start with the passage of watery stools and vomiting without fever. In the case of severe dehydration with a fluid deficit in excess of 10% of body weight, rehydration therapy with more than 100 mL/kg and the replacement of ongoing losses are necessary. Before the establishment of modern approaches to rehydration therapy, case fatality rates of cholera exceeded 50% (Clemens et al., 2017, p. 5).

In the empirical analysis, we estimate the effects of exposure to cholera in the womb and during early childhood (ages −1 to 4). The fetal-origins hypothesis argues that the inadequate nutrition of the fetus *in utero* can lead to diseases in adulthood (Barker, 1992, 1998). For instance, Almond and Mazumder (2005) and Almond (2006) show a causal link between fetal influenza exposure and later-life health and socioeconomic outcomes. The other possible factor is maternal stress because the severe symptoms of cholera may cause mental disorders in pregnant women, which would then be associated with adverse pregnancy outcomes (Hibino, Takaki, & Kambayashi, 2009; Stein, Pearson, & Goodman, 2014; Torche, 2011; Yonkers, Smith, & Forray, 2014). In addition to fetal exposure, infants are directly affected by severe and acute dehydration due to cholera infection (Katja, Mølbak, & Sandström, 2006). In fact, a growing body of the literature has found evidence that postnatal exposure to undernutrition could also have adverse long-run effects (Neelsen & Stratmann, 2011; Ampaabeng & Tan, 2013; Nandi, Ashok, & Kinra, 2016). Thus, both in utero and postnatally, exposure to infectious disease could be associated with lower socioeconomic outcomes in later life (Barreca, 2010).

Given the presence of severe and acute dehydration via cholera exposure, maternal as well as infants' nutritional deprivation owing to diarrhea and vomiting is the main cause of adverse health shocks to the fetus and infants (Ciglenecki, Bichet, & Tena, 2013). Although we cannot distinguish either channel because of the nature of our dataset, we speculate that nutritional deprivation owing to dramatic dehydration that often leads to death could be a plausible factor behind the health shock to fetuses and infants.

2.2. Estimation strategy

The basic identification strategy widely used in related studies compares affected cohorts with non-affected surrounding cohorts to capture the effects of fetal health shocks on the outcome variables (Almond, 2006). One potential issue of this strategy is that it may not use the differences in treatment intensity for identification purposes. If the incidence rates of cholera varied across units, this may attenuate the estimates of the parameters of interest.

Therefore, we employ a simple difference-in-differences approach as our estimation strategy in the spirit of Kelly (2011), Neelsen and Stratmann (2012), and Ogasawara (2017). This approach asks whether adults born in the study areas in those years in which the infection rate of cholera is higher because of the pandemic are shorter than their counterparts. Our baseline regression specification is given as follows:

$$\text{Height}_{ijt} = \alpha + \beta \text{Cholera}_{jt} + \mathbf{x}'_{jt}\boldsymbol{\gamma} + \nu_{ij} + \lambda_t + t\theta_{ij} + e_{ijt} \quad (1)$$

where Height_{ijt} is the mean final height of men in regiment i of prefecture j and year t, and Cholera_{jt} is the cholera incidence rate. We use the cholera incidence rate in the two years before birth to the four years after birth to fully capture the potential negative effects of cholera exposure. $\mathbf{x}_{jt}$ is a vector of the prefecture year-level control variables and λ_t is a year fixed effect. Note that several regiments are classified into two prefectures. Thus, we use the regiment prefecture-specific fixed effect (ν_{ij}) rather than the simple regiment fixed effect (ν_i). $t\theta_{ij}$ is the regiment prefecture-specific time trend and e_{ijt} is a random error term.