Fertility and Early-Life Mortality: Evidence from Smallpox Vaccination in Sweden*

Philipp Ager Casper Worm Hansen Peter Sandholt Jensen†

[This version: December 2016]

Abstract

The smallpox vaccination method was the paramount medical innovation of the late 18th and early 19th centuries. We exploit the introduction of the smallpox vaccine in Sweden to identify the causal effect of early-life mortality on fertility. Our analysis shows that parishes in counties with higher levels of smallpox mortality prior to the introduction of vaccination experienced greater declines in infant mortality afterwards. Exploiting this finding in an instrumental-variable approach reveals that the decline in infant mortality had a negative effect on the number of children born, whereas we find a small insignificant effect on the number of surviving children and natural population growth.

Key Words: Fertility transition; infant mortality; smallpox vaccine.

JEL: J10; J13; I15; N33

*Acknowledgements: We would like to thank the editor of the Journal (Dirk Krueger), four anonymous referees, Marcella Alsan, Marcus Asplund, Davide Cantoni, Tommy Bengtsson, Anette Boom, Gregory Clark, Carl-Johan Dalgaard, Matthias Doepke, Meltem Daysal, Oded Galor, Erich Gundlach, Fane Groes, David Jinkins, Marc Klemp, Andreas Kotsadam, Nils-Petter Lagerlöf, Lars Lønstrup, Christopher Meissner, Nathan Nunn, Per Petterson-Lidbom, Morten Saaby, Battista Severgnini, Carol Shiue, Peter Sköld, Holger Strulik, Uwe Sunde, Michele Tertilt, Mircea Trandafir, David N. Weil, Joachim Voth, and seminar participants at Copenhagen Business School, University of Copenhagen, Ludwig-Maximilians Universität, University of Hamburg, Oslo University, Deakin University, Monash University, the 2015 EEA congress in Mannheim, and the 2016 AEA Meeting in San Francisco for useful comments and suggestions.

†Contact: Philipp Ager, University of Southern Denmark, phag@sam.sdu.dk. Casper Worm Hansen, Univeristy of Copenhagen, Casper.Worm.Hansen@econ.ku.dk. Peter Sandholt Jensen, University of Southern Denmark, psj@sam.sdu.dk.

1 Introduction

An important question in long-run growth and development is how fertility and population growth respond to changes in mortality rates (Acemoglu and Johnson, 2007; Bleakley, 2010; Weil, 2014). We shed new light on this question by exploiting the invention of the vaccination method as a unique historical experiment to provide estimates of the causal effect of early-life mortality on fertility and population growth.

Historical demographic data for Sweden on fertility as well as infant and child mortality at the parish level provide a rare opportunity to examine the causal effect between early-life mortality and fertility.[1] The main challenge in identifying the causal effect of early-life mortality on fertility is omitted variables as well as reverse causality. For example, fertility and mortality are most likely determined by the same factors, of which some are unobservable (e.g., Schultz, 1997). We develop a strategy that circumvents the endogeneity issue by exploiting pre-vaccination variation in smallpox mortality across Swedish counties along with time variation arising from the introduction of the smallpox vaccine to construct an instrument for early-life mortality.

The smallpox vaccine was the first successful vaccine developed and the paramount medical innovation of the late 18th and early 19th centuries (Cutler et al., 2006). Besides data availability, the Swedish case offers several advantages compared to other countries: First, smallpox was a severe disease in Sweden, which killed approximately 10 percent of the population—most of which were infants and young children—in the second half of the 18th century (Fenner et al., 1988). This means that the introduction of the smallpox vaccine was a large shock to early-life mortality and constitutes an attractive source of exogenous variation for this reason. Second, vaccination started at the end of 1801 in Sweden and was widely distributed at zero or low cost to citizens, making the uptake of the smallpox vaccine unlikely to be correlated with regional income levels.[2] Third, Sweden subsequently introduced a compulsory vaccination law in 1816, allowing us to construct an additional instrumental variable for early-life mortality and test for over-identification.

We find that the introduction of the vaccination method had a profound negative effect on the infant mortality rate: a one-standard-deviation higher level of pre-treatment smallpox

[1] Sweden has been used as a typical example of the fertility transition (Weil, 2009).

[2] Moreover, Guinnane (2011) lists smallpox vaccination among the health influences that can be regarded as exogenous at the household level. Our fertility data are measured at the parish level, which is highly disaggregated, and it seems plausible to assume that smallpox vaccination is also exogenous at this level.

mortality is associated with a decrease in infant mortality of about 30 deaths per 1,000 live births. The effect of the compulsory vaccination law in 1816 is also negative but substantially smaller in numerical magnitude. Next, we use the interventions as instrumental variables to obtain the causal effect of early-life mortality on fertility behavior. The instrumental-variable estimates reveal that while the decline in infant mortality has a negative effect on the number of children born (gross fertility), there is no statistically significant effect on the number of surviving children (net fertility). As adjustments in gross fertility offset the extensive effect from the increasing infant survival rate, we also find that the decline in early-life mortality has no impact on natural population development as measured by the rate of natural increase.[3] These findings are in line with the theory described in Galor (2011, chapter 4) showing that parents reduce gross fertility as infant mortality declines, but that net fertility remains unchanged.[4]

Our empirical approach follows the literature on health, education, fertility, and economic growth that uses disease eradication in a differences-in-differences (DiD) framework to obtain identification. The identification strategy builds on Bleakley (2007), who combines the timing of hookworm eradication in the American South with the pre-eradication distribution of hookworm to obtain a causal effect on education. Bleakley and Lange (2009) also find that fertility decreased after the eradication of hookworm in the American South, which they attribute to an underlying quantity-quality trade-off as caused by a change in child morbidity.[5] In the same vein, we use the pre-vaccination distribution of smallpox mortality to capture which areas would experience the greatest decreases in infant mortality—and fertility in the reduced form—after the intervention. We demonstrate that pre-vaccination trends in infant mortality and fertility are the same between control and treatment counties, supporting the main identifying assumption in the DiD framework. Next, we go one step further and exploit this variation in an instrumental-variables approach similar to that of Acemoglu and Johnson (2007) and Hansen (2014) to obtain a causal effect of infant mortality on fertility. This means that we use the variation in infant mortality due to vaccination to obtain the causal effect on fertility.

[3] This finding supports the assumption in Ashraf et al. (2008), who simulate the economic consequences of a health shock, that in the long run fertility adjusts to mortality, such that population growth is unaffected.

[4] Doepke (2005) adds cost of pregnancies to the model, which means that the number of surviving children increases. Section 4 of the online appendix contains a simple model, which for the convenience of the reader, produces the main predictions of Doepke (2005) and Galor (2011). Kalemli-Ozcan (2002, 2003) adds an uncertainy effect, which would produce a decrease in surviving children.

[5] See also Aaronson et al. (2014) on the quantity-quality trade-off for the American South. Our investigation is also related to Alsan and Goldin (2015), who show that public health interventions (clean water supply) were important in explaining infant mortality declines at the end of the 19th century in the United States.

Exploiting the introduction of the smallpox vaccine to identify the impact of early-life mortality on fertility has some appealing features compared to the work of Acemoglu and Johnson (2007) and Hansen (2014). Since smallpox vaccination was the first vaccination and the major medical innovation at the time, it is difficult to think of other medical interventions that occurred at the same time and also correlate with pre-treatment smallpox mortality rates. While Acemoglu and Johnson (2007) and Hansen (2014) use the timing of the elimination of a host of infectious diseases combined with prevalence rates prior to those interventions as identification strategy, this paper provides a much clearer mechanism through which smallpox eradication worked, as the vaccination mainly reduced early-life mortality. In particular, we are able to ascertain whether there is an effect of vaccination on early-life mortality as well as whether early-life mortality affects the number of surviving children.

This paper contributes to the literature on the fertility transition, which has swept the world since the 19th century. Both Galor (2011) and Doepke (2005) argue that the fertility decline during the demographic transition was likely not a result of falling mortality rates, while the existing empirical literature on the infant mortality-fertility link provides conflicting evidence. In the literature review, we argue that the existing literature cannot credibly separate the effects from infant and adult mortality. These studies typically also encounter problems in identifying the exact effect on mortality of the shock considered. Our study exploits the fact that smallpox mainly affected infants and young children and tests whether infant mortality is affected by the shock. Given that the shock did not impact fertility directly, we identify the effect of infant mortality on fertility. We add credibility to the exclusion restriction by controlling for initial urbanization, population density, economic activity, human capital, among others, so as to rule out as many alternative explanations as possible. We interpret the evidence as strongly suggestive of a causal effect on fertility.

Our study also relates to the debate on whether health improvements make a population richer (Weil, 2014). The research by Acemoglu and Johnson (2007) suggests that this is not the case.[6] Central to their argument is that health improvements translate into population increases, as people do not die at the same rate as before the improvements. However, the long-run population effect depends on how gross fertility adjusts, and the evidence on this mechanism is scant (Bleakley, 2010). Our findings show that gross fertility adjusted to changes

[6] For other recent contributions to the literature on health and development, see, e.g., Cervellati and Sunde (2011a; 2011b; 2015) and Voigtländer and Voth (2013).

in mortality, suggesting that any effect of mortality on net fertility is offset. This suggests that health improvements for infants and children do not have adverse effects on income through population size, which could have important policy implications for developing countries.

We further contribute to the debate on the effectiveness of the smallpox vaccine in relation to reducing early-life mortality. Various authors have taken different points of view on this matter by inspecting either the aggregate or regional time series of smallpox mortality. Sköld (1996), along with Razzell (1974) and Mercer (1990), argues that smallpox mortality declined after vaccination because of the combined effect of most adults having obtained immunity and young children getting vaccination. By contrast, Fridlizius (1984), along with Bengtsson and Ohlsson (1994), suggests that the decline of smallpox mortality was caused by changes in the epidemic climate prior to vaccination. Moreover, vaccination itself could only have had an effect after it was made compulsory. Compared to the existing literature, our empirical strategy builds on a relative comparison of Swedish counties in a differences-in-differences setup. Hence, our findings do not rule out other explanations of the overall decline in mortality.

Finally, our paper belongs to a relatively small literature on the causes of the Swedish fertility transition using disaggregated data.[7] Schultz (1985), Dribe (2009), and Lagerlöf (2015) explore county-level data to study the determinants of fertility across Swedish counties. These studies find that changes in the price of women's time (Schultz, 1985), industrialization and the expansion of education (Dribe, 2009), or variation in harvests and grain prices (Lagerlöf, 2015) were important determinants of the fertility transition in Sweden.[8] Unlike these studies, we focus on early-life mortality based on a different identification strategy to obtain a causal effect of early-life mortality on fertility at the parish level.

2 Existing literature on mortality and fertility

In this section, we highlight our contribution to the existing empirical literature on mortality and fertility. Table 1 of the online appendix provides a detailed summary of the data, methods, instruments, and findings of the studies mentioned. The table classifies the studies according to three different approaches.

[7] Reher (2004) dates the onset of the fertility decline in Sweden to 1865 using aggregated data.

[8] Using model simulations, Eckstein et al. (1999) find that a decline in infant and child mortality can explain 30 percent of the decline in children per woman between 1856 and 1946 in Sweden. They also find that the number of surviving children would have increased if only infant and child mortality had declined.

First, a number of studies apply dynamic panel model methods. As recently pointed out by Acemoglu et al. (2015) and Hansen et al. (forthcoming), the main advantage of using these methods is that they remove the mechanical bias resulting from the presence of fixed effects and lagged dependent variables. Thus, the results from these studies are best interpreted as robust correlations rather than causal effects. As appears from Appendix Table 1, these studies produce results that suggest strong positive correlations (Angeles, 2010; Bhalotra and Soest 2008) and non-robust results (Murtin, 2013; Hansen et al., forthcoming).[9]

Second, some studies employ instrumental variables to estimate the fertility response to changes in infant mortality. Several papers use malaria prevalence or other malaria related variables as instruments for infant or child mortality. These include Benefo and Schultz (1994), Lorentzen et al. (2008), and McCord et al. (2017). Yet, cross-sectional measures of malaria prevalence may be correlated with unobservables and are, therefore, unlikely to be exogenous. Further, these measures affect both infant and adult mortality and may have biological effects on fertility as also pointed out by McCord et al. (2017). Other studies employ measures of the mortality environment as instruments. In general, these measures would also reflect adult mortality and would violate the exclusion restriction for this reason.[10] For example, Murphy (2015) uses temperature as an instrument, which has been argued to have direct biological effects.[11] Schultz (1997) uses calories per capita as an instrument for child mortality, which again would also affect adult mortality. Again, the results from these studies are mixed.

Finally, a group of studies use a DiD approach. Lucas (2013) exploits the eradication of malaria in Sri Lanka and finds that this increased fertility. Yet, it is not clear through which channels malaria would impact fertility as it both affects infant and adult mortality and may have direct biological effects.[12] Fortson (2009) and Juhn et al. (2013) use the differential increase in HIV in African countries and find that higher HIV is associated with less fertility.[13] The caveats regarding Lucas's study also apply to these studies. Wilson (2016) shows that

[9] Herzer et al. (2012), who apply panel co-integration methods, also find a positive correlation.

[10] This is true for Galloway et al. (1998), who use adult male mortality as an instrument, and Haines (1998), who uses the proportion of dead children of a woman as an instrument for the number of dead children.

[11] The exclusion restriction may be questioned, as it has been argued that temperature directly affects fertility as a result of changes in coital frequency or from direct physiological effects (Lam and Miron, 1996).

[12] Murray et al. (2012) and Desai et al. (2014) both document that in the recent past, adult malaria mortality is non-negligible. Murray et al. (2012) report that annual adult deaths caused by malaria was 40-50% of all deaths caused by malaria in Guyana, El Salvador, Sri Lanka, and Thailand during most of the 20th century. Desai et al. (2014) find that adult mortality was 1.5 per 1,000 population in 2003 in Kenya and only recently declined to 0.4 in 2010.

[13] Fortson (2011) also looked at the impact of HIV on human capital and found this effect to be negative.

the roll-out of medicine that prevents mother-to-child transmission of HIV lowered fertility in Zambia. Yet, Wilson's study cannot test the impact on infant mortality due to data availability. Further, the roll-out of the intervention is highly correlated with other HIV targeted interventions, making it difficult to disentangle effects from infant and adult mortality.[14]

In contrast to the existing literature, the present paper exploits an interesting episode in history using unique data to investigate the impact of reducing infant mortality on fertility. Moreover, our study establishes the effect of vaccination on infant and child mortality beyond reasonable doubt. We also investigate the effect of the intervention on fertility from the reduced form as in the other DiD studies. Given that vaccination worked through reducing infant and child mortality only, our instrumental-variable estimates can be given a causal interpretation.[15]

3 Historical background

In this section, we discuss the history of smallpox vaccination in Sweden and provide evidence that smallpox (also known as the variola virus) mainly impacted infants and young children. We further demonstrate that the Swedish age distribution of smallpox mortality rates during the late 18th and early 19th centuries is not qualitatively different from those of other countries for which early data are also available.

3.1 The history of smallpox vaccination in Sweden

Smallpox vaccination came into use in Sweden at the end of 1801 (Pettersson, 1912; Sköld, 1996), and was made compulsory in 1816 (Sköld, 1996). Prior to the invention of vaccination, the practice of inoculation was used as a preventive measure against smallpox. Inoculation is a deliberate infection with smallpox via the skin in the hope that a mild but immunizing effect would be the outcome (Baxby, 1996). Sköld (1996, p.247) concludes that: "Inoculation against smallpox was introduced in Britain in 1721, but was not practised in Sweden until 1756, and even then the method encountered difficulties in gaining acceptance." The likely reasons for public skepticism against inoculation as stated by Sköld (1996, pp.294-296) were 1) a high

[14] The other interventions are "voluntary counseling and testing" and "antiretroviral therapy" which arguably also affect adult mortality.

[15] It is important to note that most studies use gross fertility measures with the two exceptions being Bhalotra and van Soest (2008) and Angeles (2010). They find that both measures decrease as mortality is reduced, though Angeles (2010) finds small effects on net fertility. We note, however, that their methods do not allow for a causal interpretation as they rely on dynamic panel data methods.

risk of dying from the procedure, 2) that it could serve as a source of infection for those not inoculated, 3) the cost of inoculation, and 4) general conservatism in the public. Sköld (1996) concludes that inoculation did little to lower mortality in Sweden in the 18th century and that this was also largely true for the rest of Europe.

In 1798, Edward Jenner published *An Inquiry into the causes of Variolae Vaccinae, Discovered in some of the Western Counties of England, particularly Gloucestershire, and known by the name of Cow Pox*, which described the method of vaccination against smallpox. Jenner carried out his first vaccination on eight-year-old James Phipps in 1796. He inoculated the boy with cowpox, and eight weeks later Jenner inoculated the boy with smallpox, and as there was no reaction, Jenner concluded that the vaccine was effective.

A few years after Jenner's discovery, vaccination reached Sweden and was first mentioned on December 7th, 1801 by the Medical Board of Sweden. From 1803, it was official policy that the Inoculation House of Stockholm should keep fresh vaccine matter, though inoculation was not banned at this stage (Sköld, 1996). After 1803, there was no official discussion of inoculation, which was still used in some areas, but only when vaccination was not possible. The first vaccinations in Sweden have been credited to Eberhard Zacharias Munch of Rosenschöld, who carried these out at the end of 1801 (Sköld, 1996). At first there was skepticism among physicians, but by the summer of 1803, most physicians and surgeons had taken up the practice of vaccination (Sköld, 1996, p.380).

From June 1805, all church assistants had to learn to vaccinate (Sköld, 1996). This implied that there was no monopoly on vaccination. Dribe and Nystedt (2003) document that church assistants were, in fact, the most common vaccinators. Moreover, fees for vaccination were either very low or not charged at all, and vaccination was free for the poor (Sköld 1996, p.415; Dribe and Nystedt 2003, p.27). This suggests that there are good reasons to believe that there were no differences by social class in the practice of vaccination in Sweden as argued by Sköld (1996, p.466). He also notes that the authorities quickly adopted a strategy aiming at promoting vaccination. As early as 1804 every parish was instructed to appoint a vaccinator and statistics on vaccination and mortality were gathered. This served as convincing proof of the accuracy of the method to the general public.

On March 1816, the Swedish king enacted the compulsory law that all children below the age of two should be vaccinated. If parents did not have their children vaccinated they would have to pay a fine. Also, in the advent of epidemics, parents were instructed to vaccinate their

children and isolate them until the police could take care of them. Parents would be imprisoned on a diet of water and bread if they could not pay the fine (Sköld, 1996).[16] Sköld (1996, p.255) concludes that "the effect was immediate, and between 1816 and 1820 more than 73 percent of all children were vaccinated." An appealing feature of the compulsory vaccination law is that it was targeted at the group from 0 to 2 years, which suggests that it would mainly affect infants and young children (see also Section 3.2 for more details).

To gauge whether there was a substantial change in smallpox mortality when vaccination was introduced, we plot the smallpox mortality rate (i.e., total smallpox deaths per 100,000) and the smallpox share of total mortality from 1774 to 1860 in Figures 1 and 2. Both graphs indicate a negative trend in both variables consistent with the work of Fridlizius (1984), but yet there is a substantial break in the level and trend of smallpox mortality from 1802 after vaccination became available. The levels of both variables drop markedly, and while a negative trend still appears after 1801, the slope is flatter after this point. As smallpox mortality data prior to 1774 also include deaths from measles (Fridlizius and Ohlsson, 1984), we do not present these data. Yet, we note that the results are similar if we include data before 1774. We have also tested formally whether there are breaks in the levels and the trends after 1801 using a Chow test, which confirms the presence of a structural break.[17] This result is also confirmed when we test the impact of vaccination on smallpox mortality in the DiD setup below.

While we use five-year averages in Figures 1 and 2, we still find a fall in smallpox mortality after 1801 when we plot the yearly data. The yearly data also reveal a negative trend in smallpox mortality before 1802 (see Appendix Figure 1), yet they also reveal a significant break in 1802 after smallpox vaccination was introduced as was the case for the five year data.[18] Importantly, when we analyze the pattern of typhoid mortality in Sweden we do not observe any drop in typhoid mortality around 1802 when smallpox vaccination was introduced in Sweden, which confirms that the shock happened to smallpox and not to other diseases; see Appendix Figure 2 in the online appendix.

Figures 1 and 2 about here

[16] Sköld (1996, p.449) observes that "anyone disobeying this instruction was fined from 3 riksdaler 16 skilling to 50 riksdaler, an enormous sum of money for common people."

[17] This result is available upon request.

[18] Although Appendix Figure 1 also reveals a decline in the volatility of smallpox mortality, and hence a reduction in the uncertainty of child survival after 1801, our empirical results shown below do not indicate that vaccination changed the precautionary demand for children.

3.2 Descriptive evidence on early-life smallpox mortality

This subsection presents evidence that smallpox mainly affected infants and young children as argued in the literature (e.g.,Sköld, 1996; Baxby, 1996). We first consider the Swedish case, and then provide suggestive evidence from England and Wales.

The aggregate Swedish data for smallpox mortality per 100,000 by age and time (1788–1854) clearly show that mortality rates were much higher for infants and young children; see Table 1. Before the intervention, mortality appears monotonically decreasing with age, but mortality rates for infants and young children drop by more than 60 percent after the introduction of vaccination in 1801. Further, while mortality for these groups decreased monotonically over time, this is not the case for the groups above 5 years, which all experienced small increases at the end of the period. Overall, the the aggregate Swedish evidence indicates that smallpox mainly affected infants and young children.

Table 1 about here

We next investigate whether the Swedish evidence is similar to that of England and Wales by considering suggestive evidence from the latter countries after 1853 (the year in which vaccination was made compulsory there). Table 2 shows that in 1851–1860 —the decade in which compulsory vaccination was introduced—the mortality of children between 0 and 5 years was systematically higher than in the following decade 1861–1870. On average, mortality rates across registration divisions fell from 99.3 to 59.8 per 100,000 population. For everyone else, there was a modest fall from 9.09 to 7.91 per 100,000. Overall, this case confirms that mainly infants and young children were affected by smallpox vaccination and the associated compulsory vaccination laws.[19]

Table 2 about here

4 Data

This study links aggregate (county) data on smallpox mortality from Sköld (1996) with parish level data on, e.g., measures of fertility and infant and child mortality from Swedish Historical

[19] Davenport et al. (2011) use datse on burials from St. Martin-in-the-Fields in London, England, to estimate data for mortality rates and smallpox mortality rates for infants and children. Both series show a marked fall after 1798. A further strand of literature investigates whether there is a direct impact of smallpox survival on height in England; see Voth and Leunig (1996), Oxley (2003), and Sharpe (2013), for example.

Population Statistics (SHiPS). SHiPS contains digitized information about the population in Swedish parishes during the period 1749 to 1859 as reported by the clergymen to *Tabellkommissionen*, which was the predecessor to Statistics Sweden. These data provide parish-level information on infant mortality, child mortality, crude death rate, and crude birth rate, for example.[20] We have aggregated some of these parish level data up to the national level in Figure 3, showing that while the national mortality data series indicates a structural break after 1800, there is no clear trend in the development of the crude birth rate at the national level. We also note that crude birth rates are higher in 1830 than in 1800. However, this does not necessarily imply that infant mortality has no effect on the crude birth rate as the aggregate data series are likely to mask up regional heterogeneity and also contain other possible shocks to mortality and fertility. Our empirical analysis is set up to deal with these matters. In the analysis, we will estimate a partial effect suggesting that lower infant mortality reduced gross fertility, which implies that the 1830 value would have been higher without vaccination. In fact, the counterfactual, based on our estimates, suggests that fertility would have been 12.4 percent higher in the absence of vaccination.

Data on smallpox mortality were also compiled by the clergymen, and we use the data reported in Sköld (1996) for the periods 1749–1773 and 1796–1859 for 25 Swedish counties. As argued by Fridlizius and Ohlsson (1984) and Sköld (1996), smallpox was easy to diagnose, so data are likely to be accurate by historical standards. Our baseline analysis starts in 1795, which allows us to consider two periods prior to the vaccine introduction (1795 and 1800). One advantage of starting the main analysis in 1795 is that we circumvent the fact that smallpox deaths were reported together with measles before 1774 as noted above (Fridlizius and Ohlsson, 1984). Since data on infant mortality for the parishes in Norrbotten in the early periods are not available, we can only use 24 counties for the empirical analysis. The dataset we end up using is a five-year balanced panel from 1795–1860 with 24 counties and 777 parishes. However, when testing the common pre-treatment trend assumption for the introduction of the vaccination method, we widen the sample period to include additional pre-treatment years with the cost of losing some observations due to data availability (see Section 5.1).

The geographic distribution of the smallpox mortality rate (1796-1801) and the share of smallpox mortality out of total mortality (1796-1801), which we later exploit as treatment intensity in a differences-in-differences (DiD) framework, are illustrated in Figure 4. A darker shade

[20] The digitized data are made available by Umeå University at http://ships.ddb.umu.se/.

indicates a higher smallpox mortality rate (share), and the maps reveal a pattern where counties located in the northern parts of Sweden were plagued more by the disease. Consequently, our robustness analysis investigates how counties located in the northern and southern part of Sweden influence the results. These maps also suggest that the 1796-1801 smallpox-mortality rate is unrelated to county population density, which is confirmed with a raw correlation of 0.08.

Figures 3 and 4 about here

In our analysis, we also use vaccination rates from Sköld (1996), which are calculated as the number of children vaccinated as a proportion of children born in the previous five-year period. For example, for 1811-1815 the variable is calculated as the number of children vaccinated divided by children born in the period 1810-1814. Further control variables are introduced as the analysis progresses. Appendix Tables 2 and 3 provide a detailed description and summary statistics of the variables used in the empirical analysis.

5 Identification strategy

This section describes the strategy we apply to identify the effect of infant mortality on measures of gross and net fertility and natural population growth. The baseline estimation equation is:

$$y_{ijt} = \alpha \ \textit{infant mortality}_{ijt} + \delta_j + \tau_t + \rho_j \times \tau + \mathbf{X}'_{ijt}\beta + \varepsilon_{ijt}, \tag{1}$$

where y_{ijt} denotes the outcome of interest: The crude birth rate, the general fertility rate, surviving children to the ages one and five, the child-woman ratio, and natural population growth in parish i of county j at time t. We broadly refer to these outcomes as measures of gross and net fertility and natural population growth. The variable of interest, $\textit{infant mortality}_{ijt}$, is the infant mortality rate as measured by the number of infant deaths per 1,000 live births in parish i of county j in year t.[21] Our specification controls for county fixed effects (δ_j), time fixed effects (τ_t), and county-specific linear time trends ($\rho_j \times \tau$). We further include a set of parish-specific control variables, $\mathbf{X}_{ijt}$, such as initial infant mortality and other initial outcomes, which are interacted with a time indicator variable (see below), and "initial" refers to the year 1800. We cluster the error term ε_{ijt} at the county level to ensure that the standard errors

[21] The child-woman ratio is the number of children of the ages 0-5 divided by the number of women of childbearing ages (15-45).

are robust to arbitrary correlation across parishes in each county. Clustering at the county level also allows for serial correlation in the error term across time within counties. We obtain similar results when taking the relatively small number of clusters into account by collapsing our data into two time periods (i.e., pre and post-treatment periods), as proposed by Bertrand et al. (2004), or by utilizing the wild cluster bootstrap suggested by Cameron et al. (2008). As will be discussed below, we have also tried to correct our standard errors for spatial correlation using Conley standard errors. The regressions are weighted by the initial parish-population size, so that the estimates reflect an average population effect.[22] Finally, we restrict the sample to parishes that are observed all the years.

While the panel structure of the dataset allows us to non-parametrically control for time invariant county-specific (and linear time-varying) characteristics affecting both mortality and fertility, the WLS estimate of α does not necessarily measure the causal effect because of omitted factors and reverse causation. For this reason, our empirical strategy exploits a sharp decline in smallpox mortality in Sweden caused by the introduction of the vaccination method after 1801. The time variation from this episode combined with cross-county differences in the pre-treatment smallpox mortality rates represents the first-stage DiD model:

$$\begin{aligned} \textit{infant mortality}_{ijt} = \pi_1 \left(SMR_j^I \times 1[\tau > 1801] \right) + \pi_2 \left(SMR_j^{II} \times 1[\tau > 1815] \right) \\ \delta_j + \tau_t + \rho_j \times \tau + \mathbf{X}'_{ijt}\beta + \ + \bar{\varepsilon}_{ijt}, \end{aligned} \tag{2}$$

where SMR_j^I is the average smallpox mortality rate over the five-year period 1796–1801 in county j, which is just prior to the introduction of the vaccination method. In the DiD framework, this variation should be interpreted as a continuos measure of treatment intensity. The indicator variable $1[\tau > 1801]$ equals zero during the pre-treatment years (i.e., 1795 and 1800) and one in the years after treatment (i.e., 1805, 1810,...., 1860). The second interaction variable, $SMR_j^{II} \times 1[\tau > 1815]$, attempts to capture the effect of what we consider as a follow-up intervention: the enactment of the compulsory vaccination law in 1816. Its logic and construction follows the principal intervention variable.[23] The remaining variables are as defined above. We will refer to the first intervention, $SMR_j^I \times 1[\tau > 1801]$, as *vaccination* and the follow-up intervention,

[22] The online appendix shows that we obtain similar unweighted results.

[23] In particular, SMR_j^{II} is the five-year average smallpox mortality rate, measured just before the enactment of the vaccination law, and $1[\tau > 1815]$ is a time indicator equal to zero in the pre-treatment period (i.e., 1795, 1801, ..., 1815) and one hereafter.

$SMR_j^{II} \times 1[\tau > 1815]$, as *law 1816*. If $\hat{\pi}_1 < 0$ and $\hat{\pi}_2 < 0$, the introduction of the vaccination method and the compulsory vaccination law decreased infant mortality.

In the DiD framework, the main identifying assumptions include common pre-treatment trends in mortality and fertility and no time-varying omitted variables that i) systematically correlate with the continuous treatment measure and ii) start to matter for the development of mortality and fertility *only* after 1801. Besides the fact that the baseline DiD specification controls for county-specific linear time trends and initial outcomes interacted with the time indicator, we start our empirical analysis by showing that pre-treatment trends in infant mortality and fertility are the same between control and treatment counties (as measured by SMR_j^I). This finding implies that it is *not* enough to bias the DiD estimates if treatment is correlated with, say, economic activity. Economic activity should at the same time begin to matter for infant mortality and fertility only after 1801. We argue that the parallel pre-treatment trends shown in the data are supportive evidence that the introduction of the vaccination method as an instrument may satisfy the exclusion restriction required for a consistent estimate of α, and we exploit this to test the validity of the follow-up intervention as an instrument in overidentification tests. Nevertheless, the exclusion restriction may not be perfectly satisfied, as one might, for example, argue that the interventions also influence fertility through morbidity. While we think that this is not highly plausible in the case of smallpox, we also report reduced-form estimates that are not subject to such issues, as we interpret them as measuring the total effect of vaccination on fertility. Moreover, we add additional control variables and apply the plausibly exogenous technique by Conley et al. (2012) so as to add credibility to the identification strategy.

In addition, as the adoption of vaccination is endogenous, the identification strategy relies on an intention-to-treat design, where counties with a higher level of smallpox mortality potentially were given a more advantageous shock (in terms of reducing mortality) when the vaccination technology diffused. However, in contrast to many previous studies that follow a similar approach (e.g., Bleakley, 2007; Acemoglu and Johnson, 2007; Hansen, 2014), we can study whether counties with a higher burden of smallpox mortality actually had higher adoption rates of the new technology. This is possible because we have data on vaccination rates at the county level.

6 Empirical results

6.1 Pre-trends and falsification

We begin the empirical analysis by providing two types of evidence supporting the main identifying assumption (in the DiD framework) of common pre-treatment trends for the introduction of the vaccination method in 1801.

First, Figure 5 plots the estimated coefficients from event-study regressions (or distributed lag models), which modify our baseline (reduced-form) specification by interacting SMR_j^I, as well as the initial outcomes interacted with a full set of year fixed effects. The sample period has been widened to also include the pre-treatment years 1780, 1785, and 1790.[24] Panel A plots the estimated coefficients for the infant mortality rate, while panel B plots them for the crude birth rate. For both measures, we see that in the 15 years preceding the introduction of the vaccination method, trends between parishes in counties with higher and lower levels of smallpox mortality evolve relatively similarly. By 1805, however, panel A reveals a negative relationship, which is statistically significant at the one percent level, between the treatment-intensity measure (SMR_j^I) and the infant mortality rate. This effect seems relatively constant until 1825, after which it turns even stronger in numerical magnitude (with larger standard errors though). This pattern suggests that areas with a higher level of smallpox mortality rate prior to the intervention experienced larger decreases in infant mortality afterwards. The estimated coefficient pattern for the crude birth rate (in panel B) is roughly similar to that of the infant mortality rate, but with some distinct differences: We see a lagged response of five years, so that the point estimate becomes negative and statistically significant by 1810, indicating that after a period of time people adjust to changes in the mortality environment. By 1855, the negative relationship becomes less strong and is some distance from being statistically significant.[25]

Second, Table 3 reports the results from estimating our baseline specification in the pre-

[24] This implies that the period under consideration in the event-study analysis is 1780-1860. The main reason for starting the pre-treatment period in 1780 is that from this year onwards, there are data available from all the 24 counties used in the main DiD analysis. Note that the omitted year of comparison is 1800, and 1780 is also omitted since we control for county-specific linear time trends.

[25] One explanation for this could be that later years (e.g., 1850-1860) might not be particularly comparable to years close to the intervention. We thank an anonymous referee for pointing this out. In addition, Appendix Figure 3 depicts the corresponding event-study for the net fertility rate (i.e., surviving children to the age of 1). Importantly, we find no differences in pre-treatment trends and only the post-treatment estimate in 1810 is negative and significant, whereas the remaining estimates are insignificant.

treatment period 1750–1800, using "false" assumptions about the intervention year. Since the true intervention is after 1800, the results from this exercise can be interpreted as placebo experiments.[26] In particular, column (1) falsely assumes that the time indicator is equal to one after 1755, while column (2) assumes an intervention date five years later, and so on. Panel A reports the results for the infant mortality, and panel B reports the corresponding results for the crude birth rate. For both outcome measures, we see that the point estimates are (mostly) positive, close to zero, and always statistically insignificant. This finding resonates with the event-study analysis and strongly indicates that *vaccination* is not capturing some secular pre-trends in infant mortality and fertility.

In sum, the results in Figure 5 and Table 3 provide suggestive evidence that mortality and fertility changes in areas with low levels of smallpox mortality prior to 1801 serve as a reasonable counterfactual for what would have happened to areas with high levels of smallpox mortality in the absence of the introduction of the vaccination method in 1801 in Sweden.

Figure 5 and Table 3 about here

6.2 The impact of vaccination on mortality

Baseline results. Table 4 reports estimates of the effect of vaccination on the development of infant mortality, which later constitutes our first-stage framework in estimating the impact of infant mortality on measures of fertility. The estimation equation is (2), and the method of estimation is weighted least squares (WLS).

Column (1), which *only* controls for county and time fixed effects, shows that the introduction of the vaccination method is associated with a significant decrease in the infant mortality rate. While the event-study results already indicated that this is not an issue when all the baseline controls are included, one might worry that this estimate picks up some sort of convergence/divergence pattern in infant mortality. However, the estimates are very similar when we control for the infant mortality rate measured in 1800 (*initial mortality*) interacted with the time indicator (column 2) and the crude birth rate measured in 1800 (*initial fertility*) interacted with the time indicator (column 3).

[26] Due to data availability in the placebo-treatment period, the sample in Table 3 is a balanced sub-sample of the baseline sample, which only includes data from 21 out of the 24 counties. However, it should be noted that we obtain similar baseline results (i.e., the impact of vaccination on infant mortality, fertility, etc.) for this sub-sample.

Adding county-specific linear time trends in column (4), which takes out possible (linear) trend differences across counties, increases the numerical magnitude of the coefficient such that a one-standard-deviation increase in the treatment-intensity measure (SMR_j^I) is associated with a decrease in the infant mortality rate of around 30 deaths per 1,000 live births. As an alternative way of constructing the treatment measure, we have also tried to group counties with a treatment value above median into a treatment group. Each group is assigned the value one after the intervention, and counties below the median then constitute the control group. If *vaccination* is replaced with this alternative coded intervention variable, which is closer to a "classical" DiD approach, we find $\hat{\pi}_1 = -50.03$ with a standard error of 11.45. This suggests that the treatment areas experienced a decrease in infant mortality of 50 deaths 1,000 live birth relative to the controls areas (not reported in the table).

Finally, as a follow-up shock to our principal intervention, we now consider the mortality implications of the compulsory vaccination law in column (5). We see that the impact of *vaccination* remains unchanged and that the compulsory vaccination law also seems to have decreased infant mortality. Still, this effect is much smaller in magnitude compared to the effect of the introduction of the vaccination method in 1801, which is evident from the standardized beta coefficients reported in the bottom of the table. We regard the specification in column (5) as our baseline first-stage result.[27]

Table 4 about here

Additional outcomes. We next consider additional mortality related outcomes that support our first-stage framework. As our approach is an intention-to-treat design, we assume that our treatment measures capture increases in vaccination and decreases in smallpox mortality. The first two columns of Table 5 validate this approach. Column (1) exploits the fact that we have a measure for the adoption of the vaccination method; that is, the outcome is now county i's vaccination rate for children aged 0-5 years at time t. The coefficients on the interventions are both positive but only borderline significant for *vaccination* and significant at the five percent level for *law 1816*. This suggests that counties with a higher level of smallpox mortality before the interventions had higher adoption rates of the new medical technology afterwards.

One feature of our empirical strategy is that the estimation equation (2) is the reduced form

[27] It is important to stress that any of our subsequent results do not hinge upon the inclusion of the follow-up intervention (*law 1816*).

of the following first stage:[28]

$$\textit{smallpox mortality}_{it} = \gamma_1 \left(SMR_j^I \times 1[\tau > 1801]\right) + \gamma_2 \ \left(SMR_j^{II} \times 1[\tau > 1815]\right) + \mathbf{X}'_{jt}\overline{\beta} + \bar{\delta}_j + \bar{\tau}_t + \bar{\rho}_j \times \bar{\tau} + \bar{\varepsilon}_{ijt}, \tag{3}$$

where $\textit{smallpox mortality}_{it}$ is the smallpox mortality rate in county i at time t. Column (2) reports the coefficients of estimating this equation. The coefficients on the two interaction terms are negative and highly statistically significant. These results would imply that one could use a three-stage least squares (3SLS) approach: $SMR_j^I \times 1[\tau > 1801]$ and $SMR_j^{II} \times 1[\tau > 1815] \Rightarrow \textit{smallpox mortality}_{it} \Rightarrow$ *infant mortality*$\Rightarrow y_{ijt}$. However, having now established the first chain in this line of argumentation, we follow the literature and regress infant mortality directly on the two interactions, implying that we end up with the suggested 2SLS model.

Column (3) reports the estimates for child mortality (aged 1-5 years), while column (4) reports the estimates for the crude death rate. We see that both measures are reduced as a consequence of the introduction of the vaccination method in 1801, but also notice that the vaccination law did not seem to decrease any of these two measures, which is arguably related to the fact that compulsory vaccination was for 0–2-year-olds. Finally, columns (5) and (6) demonstrate that there are no significant gender differences operating through the impact of vaccination on infant mortality as measured by infant mortality for boys and girls, respectively.

Table 5 about here

6.3 Reduced-form evidence

This section presents evidence of the reduced-form effects of the two interventions on the crude birth rate, general fertility rate, surviving children, the child-woman ratio, and natural population growth. We interpret the reduced-form estimates as measuring the *overall* effects of the introduction of the vaccination method and the vaccination law. The method of estimation is weighted least squares, and all the specifications include the baseline controls: county and year fixed effects, county-specific linear time trends, initial infant mortality interacted with the time indicator, and the initial outcome of interest interacted with the time indicator.

The reduced-form estimates, reported in Table 6, show that the introduction of the vaccination method had negative effects on our two measures of gross fertility, namely, the crude

[28] In our 2SLS notation this equation should be referred to as a zeroth stage.

birth rate and the general fertility rate (columns 1 and 2), whereas there are no noticeable effects on the measures of net fertility (columns 3–5) and natural population growth (column 6). For example, the estimate for the number of surviving children to the age of one, which is constructed as the infant *survival* rate times the crude birth rate, is around 10 times smaller than the corresponding estimates for the crude birth rate and statistically highly insignificant. The magnitude of the estimates for gross fertility implies that a one-standard deviation increase in treatment intensity is associated with a decrease of 1.16 births per 1,000 population and 5.57 births per 1,000 women of childbearing ages (15-45).

Together with the results on infant mortality, these findings provide evidence suggesting that the vaccination method lowered infant mortality and people responded by lowering the number of live-born children, so that the number of surviving children (and, thus, natural population growth) remains unaffected. Finally, we also observe that the vaccination law did not influence any of the measures shown in Table 6.

One concern particularly related to the 2SLS estimates (reported in the next section) is the morbidity effects associated with vaccination. Sköld (2003) discusses the possibility that male fecundity was affected by smallpox because infected men would be disadvantaged in the marriage markets due to facial pockmarks. This effect would tend to work over a prolonged period of time, and we note that our results still hold if we stop in 1820. Sköld (1996) further demonstrates that infected and vaccinated men had similar fecundity levels, and Rutten (1993) presents similar evidence for the Netherlands. We have also run regressions with marriage rates as independent variable and find little effect of vaccination. Thus, the existing empirical evidence tends to reject this effect. Second, blindness is a rare complication associated with surviving smallpox. Existing evidence shows that blindness occurs in less than 0.5 percent of cases (Fenner et al., 1988). This suggests that smallpox vaccination is unlikely to drive a large reduction in the incidence of blindness and fertility choices. Finally, Sköld (1996, p.123) mentions that after 30 years of vaccination, the smallpox mortality distribution began to change in the direction of adults. This might affect the 2SLS results, though we obtain similar results when stopping in 1820. Nevertheless, while it cannot be completely ruled out that morbidity could bias the 2SLS estimates, the reduced-form evidence in this section measures the overall effects of the interventions.

Table 6 about here

6.4 Infant mortality, fertility, and population growth

Baseline results. Table 7 presents the coefficient on *infant mortality* for the gross- and net-fertility outcomes and natural population growth estimated by WLS (panel A) and 2SLS (panel B), using equation (2) as the first stage. All specifications include the baseline controls.

Panel A reports significant negative correlations between the infant mortality rate and the six outcomes. For example, the magnitude of the estimate in column (1) implies that a one-standard deviation decrease in the infant mortality rate is associated with a 0.25 increase in the crude birth rate, which corresponds to three percent of a standard deviation. Thus, although the coefficient is statistically significant, it is also quantitatively very small in magnitude. Taken at face value, this negative correlation then implies that as the infant mortality rate decreases, the number of surviving children increases: In column (3), we find $\hat{\alpha} = -0.03$ (significant at the one percent level), indicating that a one-standard deviation decrease in the infant mortality rate is related to an increase in the number of surviving children to the age of one of 2.77. This number is close to 40 percent of a standard deviation in the number of surviving children and the effect seems substantial. Consistently, we also find significant negative correlations between the infant mortality rate and the child-woman ratio (column 5), which we interpret as another measure of net fertility, and natural population growth (column 6).

Hence, taking this type of evidence at face value, we would tend to reject the theoretical predictions of Doepke (2005) and Galor (2011) that a decrease in infant mortality would have a negative effect on gross fertility and a small (or no) effect on net fertility, measured as the number of surviving children and, thus, natural population growth. However, the interpretation of the WLS estimates is problematic due to omitted variable bias and reverse causality. One example of omitted variable bias is income, which could be negatively correlated with infant mortality. In the case that Malthusian effects are present, income would affect fertility positively and, thus, downward bias the coefficient on infant mortality.

To address this concern, panel B presents 2SLS estimates of the impact that the vaccination interventions had on these six outcomes through its impact on infant mortality. Exploiting *vaccination* and *law 1816* as the excluded instruments, column (1) reveals that the 2SLS estimate on infant mortality is 0.04 and statistically significant at the one percent level. This magnitude suggests that a fall in the infant mortality rate by 30 deaths per 1,000 live births leads to a decrease in the number of births by circa 1.1 per 1,000 population. Column (2) shows an almost identical finding for the general fertility rate: the standardized beta coefficients are 0.46 and

0.47, respectively. Therefore, once we take potential endogeneity concerns into account, we obtain a different conclusion where, in line with the theory of Doepke (2005) and Galor (2011), the results show that a decline in infant mortality has a negative impact on measures of gross fertility.

On the other hand, columns (3)-(6) demonstrate that the effect of infant mortality on measures of net fertility and natural population growth is close to zero.[29] One interpretation of this discrepancy is that the 2SLS estimates for net fertility take into account that the increase in the infant survival rate of children is fully offset by a decrease in the birth rate. The standardized beta coefficient for the crude birth rate was 0.46 (column 1), whereas for the number of surviving children to the age of one it is equal to 0.009 (column 3). Thus, although there is a substantial difference in point estimates and beta coefficients, a simple comparison of the 95-percent confidence bands suggests no differences. We have therefore conducted two formal tests to check whether this is really the case. First, we estimate a 3SLS regression, which allows us to test directly for the statistical equality between the two point coefficients. Second, we calculate the difference between the crude birth rate and the number of surviving children to the age of one and regress this variation on infant mortality and the baseline controls, using (2) as the first stage. In both cases, we strongly reject the null hypothesis of equality. In sum, the empirical evidence, reported in Table 7, indicates that fertility adjusts to changes in infant mortality, so that surviving children and, therefore, natural population development are unaffected.

In terms of instrument quality, the 2SLS estimation strategy yields a reasonable first-stage fit. The Kleibergen-Paap F-statistic reported in all 2SLS specifications is always larger than 10, thus mitigating the concern that our statistical inference would yield misleading results due to the presence of weak instruments (Stock et al., 2002). Since all the reported 2SLS regressions are overidentified, we can compute the Hansen J-test on the joint hypothesis that the instruments (*vaccination* and *law 1816*) are uncorrelated with the second-stage error term. The Hansen-J test on the overidentifying restriction produces p-values of 0.23 and above, such that we cannot reject the joint hypothesis that the two instruments are uncorrelated with the second-stage error terms. Hence, given the assumption of exogeneity of *vaccination*, which is

[29] We note that the estimate for the number of surviving children to the age of five is negative and actually borderline significant (column 4). As shown in Section 3 of the online appendix, this finding is consistent with models in the style of Doepke (2005) and Galor (2011), where fixed costs of having children has been added. We also note that the result is not very robust.

supported by the evidence presented in Section 5.1, we cannot reject the null that *law 1816* is a valid instrument.

In Appendix Figure 4, we apply the "plausibly exogenous" technique of Conley et al. (2012) to gauge how large a potential direct effect of the instruments needs to be to render the 2SLS estimate on infant mortality insignificant.[30] The estimates for the crude birth rate (panel A) and the general fertility rate (panel B) suggest that some omitted variable that is also captured by *vaccination* needs to explain more than half of the overall reduced form effect to render the 2SLS estimate on infant mortality insignificant.[31]

Table 7 about here

Robustness. We have carried out a number of robustness checks based on the baseline 2SLS estimates. Table 8 starts this analysis by investigating the robustness to initial variation in measures of economic development, income, human capital, population structure, and marriage rates. We follow a strategy that controls for variation in these variables prior to the first treatment (i.e., measured in 1800 and referred to as "initial") in order not to include any potential endogenous controls that could bias the estimate of interest. The table is structured so that each panel reports 2SLS estimates, controlling for the aforementioned variables interacted with the time indicator.[32]

Panel A considers population density and the urbanization rate as proxies for economic development.[33] These measures of economic activity are often used in the long-run economic development literature (e.g., Acemoglu et al., 2002; Nunn and Qian, 2011). We find that 2SLS coefficients are very similar to the baseline in terms of magnitude and statistical significance. Panel B adds the price of rye and the log of rye production per capita as our second indicators of county-level development or income (Dribe et al., 2011). We see that the 2SLS estimates are robust to the inclusion of these controls. This is perhaps not altogether surprising since there are not many preventive measures that richer people can take against contracting smallpox—for

[30] The results presented in Appendix Figure 4 are based on using only *vaccination* as the excluded instrument.

[31] We refer to the online appendix for further details.

[32] To save space, the robustness tables do not report the first-stage results, but they are qualitatively similar and available upon request.

[33] The city population data are census data and can be downloaded at www.balticctowns.com/cybcity/befolkning/index.htm, whereas the total county population size is derived from our main data source (SHiPS). According to this variable, the mean urbanization rate was about 10 percent in 1800.

example, in terms of personal hygiene, as the disease is airborne (Sundin, 1995). We also note that this is corroborated by the fact that SMR_j^I and SMR_j^{II} are uncorrelated with the above mentioned measures of (initial) economic activity.[34] Panel C considers whether initial variation in human capital is confounding our main findings. In particular, we have obtained data on i) whether there was a "high school" present in the county or not, ii) the number of so-called trivial schools, which taught at lower levels than proper high schools, and iii) university enrollment as measured by population per university student by county.[35] These are then interacted with the time indicator and added to the baseline specification. However, the 2SLS relationships between infant mortality and the outcomes are virtually unchanged compared to the baseline.

While the general fertility rate already takes in the number of women of childbearing ages into account variation, this variation is, of course, *not* accounted for in the other outcomes. Therefore, panel D controls for the initial share of women of childbearing ages (15-45) out of the population interacted with the time indicator, but with almost no changes in the estimated 2SLS coefficient as a result. Panel E of Table 8 shows that our findings are robust to initial variation in the marriage rate. Finally, we note that the first-stage results are very similar to the baseline: The Kleibergen-Paap F-statistic never goes below 9.25 and the Hansen-J p-value is always larger than 0.22 (not reported).[36]

Next, panels A and B of Table 9, which is structured as Table 8, provide further evidence suggesting that our 2SLS estimates are, in fact, not capturing any secular pre-trends in either infant mortality or the second-stage outcome. First, while linear time trends are good approximations even if the actual trends tend to be quadratic, panel A instead controls for county-specific quadratic time trends. We see that including them does not change much compared to using county linear trends in the baseline. Second, panel B adds infant mortality in 1795 and the outcome of interest in 1795, both interacted with the time indicator to take out possible mean reverting behavior in the data. In any case, we end up with almost identical 2SLS estimates.

[34] Moreover, we find that the interventions had no effects on the urbanization rate, population density, the price of rye, and the production of rye. These reduced-form estimates are available upon request.

[35] Besides the university enrollment variable, which is measured in 1815, these are measured around the time of the first intervention (i.e., 1800).

[36] Below we report the coefficients along with t-ratios in Panels A to E for the control variables (all interacted with a dummy which equals one from 1801) for the crude birth rate: Panel A: urbanization rate in 1800: -10.22 (2.97), population density in 1800: -0.019 (0.05); Panel B: log rye per capita in 1800: 0.43 (0.81), price rye in 1800: 0.56 (1.00); Panel C:, trivial schools -0.60748 (1.97) high school 1.2348 (1.32), university enrollment 0.0004 (0.54); Panel D: female population share (ages 15 to 45) in 1800: -26.94 (-4.40); Panel E: marriage rate in 1800: 0.017 (0.40).

Panel C of Table 9 shows the results when controlling for parish fixed effects instead of county fixed effects. As the two interventions only vary at the county-by-year level, the results would be similar if we did not include the parish-level interactions. Nevertheless, we see increases in magnitude of the estimated coefficients on the infant mortality rate in the models for the crude birth rate and the general fertility rate (columns 1 and 2), whereas the corresponding estimates in the models for the number of surviving children (ages one and five) and natural population growth remain statistically insignificant (columns 3, 4, and 6).

Finally, as Figures 3 and 4 revealed a north-south gradient in the smallpox mortality rate in 1796–1801, using the introduction of the vaccination method to estimate the impact of infant mortality on the outcomes could lead to biased estimates. This would be the case if other (geographical) characteristics which systematically varied along this gradient, became important for infant mortality and fertility in the beginning of the 19th century. As a check of this concern, panel D (E) takes out counties in the southern (northern) part of Sweden.[37] Taking out the southern counties does not change any of our conclusions, as the estimates remain both stable in size and significance. When taking out the northern counties, we see a reduction in the estimated coefficient for the crude birth rate, but it remains positive and statistically significant at the 10-percent level (column 1). This is also the case for the general fertility rate, though the estimated coefficient is borderline significant. As the fertility response appears weaker in this subsample, we see that the estimates for net fertility and natural population growth turn negative (columns 3–5). We also note that the first-stage fit becomes weaker when taking out northern counties with Kleibergen-Paap F-statistics ranging from around 5 to 8 (not reported). However, this is surprisingly *not* due to a decrease in the estimate on *vaccination*, but rather due to a reduced impact of *law 1816* on infant mortality. Consequently, we tried dropping this follow-up intervention, which, in fact, increases the first-stage fit substantially. Yet, we find similar 2SLS estimates as in panel E, suggesting that the estimates are not biased by weak first stages. This is also confirmed by the Anderson-Rubin P-values. Though, the effects are less precisely estimated in the subsample without the northern counties, we note that the results remain largely in line with our overall findings.

Tables 8 and 9 about here

[37] In particular, the counties in the southern (northern) part of Sweden refer to the five most southerly (northerly) of the 24 counties included. We remind the reader that the northernmost county (Norrbotten) has been excluded throughout our empirical analysis due to lack of pre-vaccination outcome data.

Additional robustness checks. We now briefly describe additional robustness tests, which are reported in the online appendix. First, our baseline specifications use parish population size in 1800 as weights, so that the estimates reflect an average population effect. Appendix Table 4 shows similar estimates if we do not use any weights.

Second, Appendix Tables 5 and 6 demonstrate the robustness of our findings to changes in the panel and time structure. In particular, we first collapse the five-years observations into *one* pre–treatment period and *one* post–treatment period based on the cutoff date of the main intervention and cluster the standard errors at the parish level instead of the county level. According to Bertrand et al. (2004), this is one way of dealing with the problem of too few clusters and allow us to cluster at the lower (parish) level. We find that both the first- and second-stage results are robust to these changes. Next, we find similar results to the baseline by collapsing the data into 10-year periods or collapsing the parish level observations to the county level. In addition, following the approach of Acemoglu and Johnson (2007), Appendix Table 6 exploits only *one* pre-treatment period (i.e., the average of the years 1795 and 1800) and various post-treatment periods (i.e., 1805/1810, 1815/1820, 1825/1830, etc.). In line with the reduced-form event-study analysis (shown in Figure 5), we find that the effects of infant mortality on the crude birth rate and the general fertility rate are strongest when using either the post-treatment period 1825/1830 or the post-treatment period 1835/1840.

Third, Appendix Table 7 shows that our findings are robust in the reduced form to estimating the standard errors differently. Specifically, we compute standard errors based on the wild cluster bootstrap method, two-way clustered by county and year, and Conley standard errors with a cut-off of 150 kilometers.

Fourth, we report the 2SLS estimate when directly controlling for log population and urbanization rate instead of their initial values interacted with the time indicator. However, Appendix Table 8 reveals that this matters little for our results.

Fifth and finally, Appendix Table 9 uses different functional-form specifications (log-log, log-level, and level-log), Appendix Table 10 uses the infant *survival* rates (instead of infant mortality), and Appendix Table 11 includes lagged dependent variables (both in the first and second stages). These changes, however, make little difference for our overall conclusions.[38]

[38] We further obtain very similar results, both in the first and second stage if, instead of using the pre-treatment smallpox mortality rates, we use the pre-treatment smallpox mortality share out of total mortality, which has the advantage that it holds the mortality environment (not related to smallpox) constant. These results are available upon request.

7 Conclusion

This paper has demonstrated that infant mortality in Sweden strongly responded to the introduction of smallpox vaccination. We find that a one-standard-deviation higher level of pre-vaccination smallpox mortality was associated with a decrease in infant mortality of about 20 deaths per 1,000 live births, while compulsory vaccination reduced infant mortality with about five deaths per 1,000 live births. We then used the vaccination-shock variables as excluded instruments to estimate the causal response of fertility and natural population growth to changes in infant mortality. The two-stage least squares estimates reveal that a decline in infant mortality had a significant negative impact on gross fertility as measured by the crude birth rates and the general fertility rate. We find smaller effects for net fertility, most of the estimated coefficients are statistically insignificant being in line with zero effects. We obtain significant results in some specifications using surviving children to age 5, but this is not a very robust result. For this reason, we conclude that infant mortality was unlikely to be a main driver of net fertility and natural population growth in Sweden, which is in line with the theoretical predictions of Doepke (2005) and Galor (2011).

Our findings could have important policy implications for developing countries. They suggest that infant mortality could be reduced without increasing population growth. Yet, we believe that one should exert caution, as the results suggest that the effects on net fertility and natural population growth could be either positive or negative, even if they are small.

The current study is based on data for Sweden, and while we have shown descriptive evidence consistent with the same mechanism being at play elsewhere, one may naturally question the external validity. Nonetheless, Sweden and the rest of Scandinavia served as role models for the rest of the world in combating smallpox (Fenner et al., 1988). This suggests that the Swedish case is of interest on its own, as it provided the blueprints for combating smallpox elsewhere. Yet, we believe it is important for future research to obtain evidence on the mortality-fertility nexus from other countries and time periods.

References

[1] Aaronson, D., Lange, F., Mazumder, B. (2014). Fertility Transitions Along the Extensive and Intensive Margin. American Economic Review 104(11), 3701–3724.

[2] Acemoglu, D., Johnson, S., Robinson, J. (2002). Reversal of Fortune: Geography and Institutions in the Making of the Modern World Income Distribution, Quarterly Journal of Economics, 117, 1231–1294.

[3] Acemoglu, D., Johnson, S. (2007). Disease and Development: the Effect of Life Expectancy on Economic Growth. Journal of Political Economy, 115(6), 925–985.

[4] Alsan, M., Goldin, C (2015). Watersheds in Infant Mortality: The Role of Effective Water and Sewerage Infrastructure, 1880 to 1915. NBER Working Paper 21263.

[5] Angeles, L. (2010). Demographic transitions: Analyzing the effects of mortality on fertility. Journal of Population Economics, 23(1), 99–120.

[6] Ashraf, Q., Lester, A., Weil, D.N. (2008). When Does Improving Health Raise GDP? In Acemoglu, Rogoff, and Woodford (eds.): NBER Macro Annual.

[7] Baxby, D. (1996). The Jenner bicentenary: the introduction and early distribution of smallpox vaccine. FEMS Immunology and Medical Microbiology, 16, l–10.

[8] Benefo, K., Schultz, T.P. (1996). Fertility and child mortality in Cote d'Ivoire and Ghana. World Bank Economic Review, 10(1), 123–158.

[9] Bengtsson, T., Ohlsson, R. (1994). The Demographic Transition Revised. In Bengtsson, T. (ed). Population, Economy, and Welfare in Sweden. Springer Verlag, Heidelberg, Germany.

[10] Bhalotra, S., van Soest, A. (2008). Birth-spacing, fertility and neonatal mortality in India: Dynamics, frailty, and fecundity. Journal of Econometrics, 143, 274–290

[11] Bertrand, M., Duflo, E., and Mullainathan, S. (2004). How Much Should We Trust Differences-in-Differences Estimates? Quarterly Journal of Economics, 119(1), 249–75.

[12] Bleakley, H., (2007). Disease and development: evidence from hookworm eradication in the American South. Quarterly Journal of Economics, 122 (1), 73–117.

[13] Bleakley, H., Lange, F. (2009). Chronic disease burden and the interaction of eduation, fertility, and growth. Review of Economics and Statistics, 91(1), 52–65.

[14] Bleakley, H. (2010). Health, human capital, and development. Annual Review of Economics, 2, 283–310.

[15] Cameron, A.C., Gelbach, J.B. and Miller, D.L. (2008). Bootstrap-Based Improvements for Inference with Clustered Errors. Review of Economics and Statistics, 90(3), 414–427.

[16] Cervellati, M., Sunde, U. (2011a). Life expectancy and economic growth: The role of the demographic transition. Journal of Economic Growth, 16(2), 99-133.

[17] Cervellati, M., Sunde, U. (2011b). Disease and development: the role of life expectancy reconsidered. Economics Letters, 113(3), 269–272.

[18] Cervellati, M., Sunde, U. (2015). The effect of life expectancy on education and population dynamics. Empircal Economics, (2015) 48, 1445–1478.

[19] Conley, T. G., Hansen C.B., Rossi, P.E. (2012). Plausibly Exogenous. Review of Economics and Statistics 94 (1), 260–272.

[20] Cutler D., Deaton, A., Lleras-Muney, A. (2006). The Determinants of Mortality. Journal of Economic Perspectives, 20(3), 97–120.

[21] Davenport, R., Schwarz, L., Boulton, J. (2011). The decline of adult smallpox in eighteenth-century London. Economic History Review, 64(4), 1289–1314.

[22] Desai, M., Buff, A.M., Khagayi, S., Byass, P., Amek, N., van Eijk, A., Slutsker, L., Vulule, J., Odhiambo, F.O., Phillips-Howard, P.A., Lindblade, K.A., Laserson, K.F., Hame, M.J. (2014). Age-Specific Malaria Mortality Rates in the KEMRI/CDC Health and Demographic Surveillance System in Western Kenya, 2003–2010. Plos One, 9(9), 1-6.

[23] Doepke, M. (2005). Child mortality and fertility decline: Does the Barro-Becker model fit the facts? Journal of Population Economics, 18, 337–366.

[24] Dribe, M., Nystedt, P. (2003). Information, trust and the diffusion of smallpox vaccination: The Case of Scania in Sweden, 1802–1835. Scandinavian Economic History Review, 51(1), 9–28.

[25] Dribe, M. (2009). Demand and supply factors in the fertility transition: a county-level analysis of age-specific marital fertility in Sweden, 1880–1930. European Review of Economic History, 13, 65–94.

[26] Dribe, M., Olsson, M., Svensson, P. (2011). Production, prices and mortality: Demographic response to economic hardship in rural Sweden, 1750–1860. Mimeo.

[27] Eckstein, Z., Mira, P., Wolpin, K. (1999). A quantitative analysis of Swedish fertility dynamics: 1751-1990. Review of Economic Dynamics, 2, 137-165.

[28] Fenner, F., Henderson, D. A., Arita, I., JeZek, Z., Ladnyi, I. D. (1988). Smallpox and its eradication. WHO, Geneva, Switzerland.

[29] Fortson, J. (2009). HIV/AIDS and Fertility. American Economic Journal: Applied Economics, 1(3), 170–194.

[30] Fortson, J. (2011). Mortality risk and human capital investment: the impact of HIV/AIDS in Sub-Saharan Africa. Review of Economics and Statistics, 93(1), 1–15.

[31] First report of the Royal Commission Appointed to Inquire into the Subject of Vaccination.

[32] Fridlizius, G. (1984). The mortality decline in the first phase of the demographic transition: Swedish experiences. In Bengtsson, T., Fridlizius, G., Ohlsson, R., (eds.): Pre-industrial population change. The mortality decline and short-term population movements. Almquist and Wiksell, Stockholm, Sweden.

[33] Fridlizius, G., Ohlsson, R. (1984). Mortality patterns in Sweden 1751-1802 - a regional analysis. In Bengtsson, T., Fridlizius, G., Ohlsson, R., (eds.): Pre-industrial population change. The mortality decline and short-term population movements. Almquist and Wiksell, Stockholm, Sweden.

[34] Galloway, P.E., Lee, R.D., Hammel, E.A. (1998). Infant mortality and the fertility transition: macro evidence from Europea and New Findings from Prussia. In Montgomery, M. and Cohen, B. (eds): From death to birth: mortality decline and reproductive change. National Academy Press, Washington D.C.

[35] Galor, O. (2011). Unified growth theory. Princeton University Press.

[36] Guinnane, T.W. (2011). The historical fertility transition: A guide for Economists. Journal of Economic Literature, 49(3), 589–614.

[37] Haines, M. (1998). The relationship between infant and child mortality and fertility: Some historical and contemporary evidence from the United States. In Montgomery, M. and Cohen, B. (eds): From death to birth: mortality decline and reproductive change. National Academy Press, Washington D.C.

[38] Hansen, C.W. (2014). Cause of death and development in the US. Journal of Development Economics, 109, 143–153.

[39] Hansen, C.W., Jensen, P.S., Lønstrup, L. (Forthcoming). The Fertility Transition in the US: Schooling and Income. Macroeconomic Dynamics.

[40] Herzer, D., Strulik, H., Vollmer, S. (2012). The Long-run Determinants of Fertility: One Century of Demographic Change 1900–1999. Journal of Economic Growth, 17(4), 357-385.

[41] Juhn, C., Kalemli-Ozcan, S., Turan, B. (2013). HIV and fertility in Africa: first evidence from population-based surveys. Journal of Population Economics, 26, 835–853.

[42] Kalemli-Ozcan, S. (2002). Does the mortality decline promote economic growth?, Journal of Economic Growth, 7(4), 411-439.

[43] Kalemli-Ozcan, S. (2003). A stochastic model of mortality, fertility, and human capital investment. Journal of Development Economics, 70(1), 103–118.

[44] Lagerlöf, N. (2015). Malthus in Sweden. Scandinavian Journal of Economics, 117(4), 1091–1133.

[45] Lam, D.A., Miron, J.A. (1996). The effects of temperature on human fertility. Demography, 33(3), 291-305.

[46] Lorentzen, P., McMillan, J.,Wacziarg, R., 2008. Death and development. Journal of Economic. Growth, 13(2), 81–124.

[47] Lucas, A.M. (2013). The Impact of Malaria Eradication on Fertility. Economic Development and Cultural Change, 61(3), 607–631.

[48] McCord, G.C., Conley, D., Sachs, J.D. (2007). Malaria ecology, child mortality & fertility. Economics and Human Biology 24, 1–17.

[49] Mercer, A.J. (1990). Smallpox and Epidemiological-Demographic Change in Europe: The Role of Vaccination. Population Studies: A Journal of Demography, 39(2), 287-307.

[50] Murray, C.J.L., Rosenfeld, L.C., Lim, S.S., Andrews, K.G., Foreman, K.J., Haring, D., Fullman, N., Naghavi,M., Lozano, R., Lopez, A.D. (2012). Global malaria mortality between 1980 and 2010: a systematic analysis. Lancet, 379, 413–431.

[51] Murphy, T. (2015). Old habits die hard (Sometimes). Journal of Economic Growth, 20(2), 177–222.

[52] Murtin, F. (2013). Long-term Determinants of the Demographic Transition: 1870-2000. Review of Economics and Statistics, 95(2), 617–631.

[53] Nunn, N., Qian, N. (2011). The potato's contribution to population and urbanization: Evidence from a historical experiment. Quarterly Journal of Economics, 126, 593–650.

[54] Oxley, D. (2003). The seat of death and terror: urbanization, stunting, and smallpox. Economic History Review, 56(4), 623–656.

[55] Pettersson, A. (1912). Smittkopdödeligheten i Sverige under åren 1776-1875. Hygiensk Tidskrift, 1–17.

[56] Razzell, P. (1974). An Interpretation of the Modern Rise of Population in Europe–A Critique. Population Studies, 28(1), 5-17.

[57] Reher, D.S. (2004). The Demographic Transition Revisited as a Global Process. Population Space Place, 10, 19–41.

[58] Rutten, W. (1993). Smallpox, subfecundity, and sterility: a case study from a nineteenth-century Dutch municipality. Social History of Medicine, 61(1), 85–99.

[59] Second report of the Royal Commission Appointed to Inquire into the Subject of Vaccination.

[60] Schultz, T.P. (1985). Changing World Prices, Women's Wages, and the Fertility Transition: Sweden, 1860-1910. Journal of Political Economy, 93(6), 1126–1154.

[61] Schultz, T.P. (1997). Demand for Children in Low Income Countries. In M.Rosenzweig and O. Stark (eds.): Handbook of Population and Family Economics, North-Holland, Amsterdam, pp. 349-430.

[62] Sharpe, P. (2012). Explaining the short stature of the poor: chronic childhood disease and growth in nineteenth-century England. Economic History Review 65(4), 1475–1494.

[63] Sköld, P. (1996). The two faces of smallpox - a disease and its prevention in eighteenth- and nineteenth century Sweden. UmU Tryckeri Umeå University.

[64] Sköld, P. (2003). The Beauty and the Beast – Smallpox and Marriage in Eighteenth and Nineteenth-Century Sweden. Historical Social Research, 28(3), 141-161.

[65] Stock, J.H., Wright, J.H., Yogo, M. (2002). A Survey of Weak Instruments and Weak Identification in Generalized Method of Moments. Journal of Business and Economic Statistics, 20(4), 518–529.

[66] Sundin, J. (1995). Culture, Class, and Infant Mortality during the Swedish Mortality Transition, 1750-1850, Social Science History, 19(1), 117-145.

[67] Voigtländer, N., Voth, H-J. (2013). The three horsemen of riches: plague,war, and urbanization in early modern Europe. Review of Economic Studies, 80(2), 774-811.

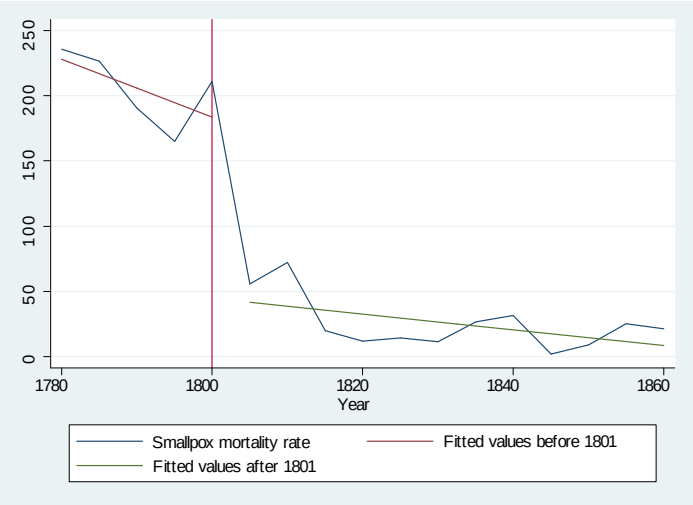
[68] Voth, H-J., Leunig, T. (1996). Did smallpox reduce height? Stature and the standard of living in London, 1770-1873. Economic History Review, 49(3), 541-560.

[69] Weil, D.N. (2009). Economic Growth. Pearsson, USA.

[70] Weil, D.N. (2014). Health and Economic Growth. Durlauf, S.N., Aghion, P. (eds). Handbook of Economic Growth, North-Holland, Netherlands.

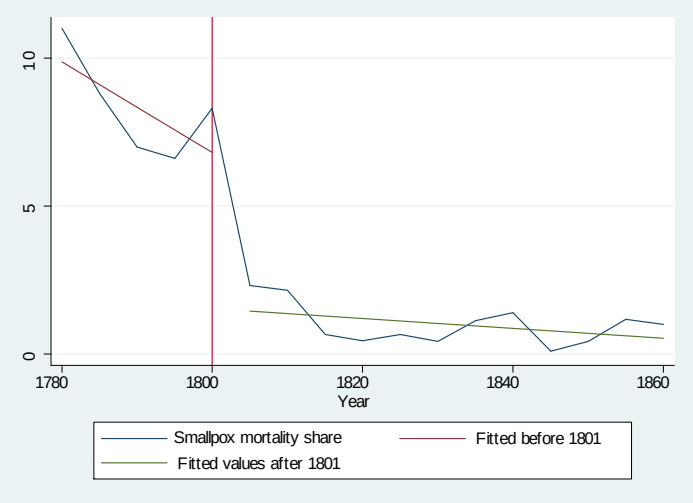
[71] Wilson, N. (2016). Child mortality risk and fertility: Evidence from prevention of mother-to-child transmission of HIV. Journal of Development Economics, 116, 74-88.

Figure 1: Smallpox mortality rates in Sweden, 1774-1860



Notes: The data are five-year averages of smallpox mortality rates for all ages starting in 1774. The "year" 1780 on the first axis includes the period 1774-1779 corresponding to the first period that only includes smallpox. The solid vertical line indicates the years 1796-1801, which is the period just prior to vaccination. Source: Sköld (1996).

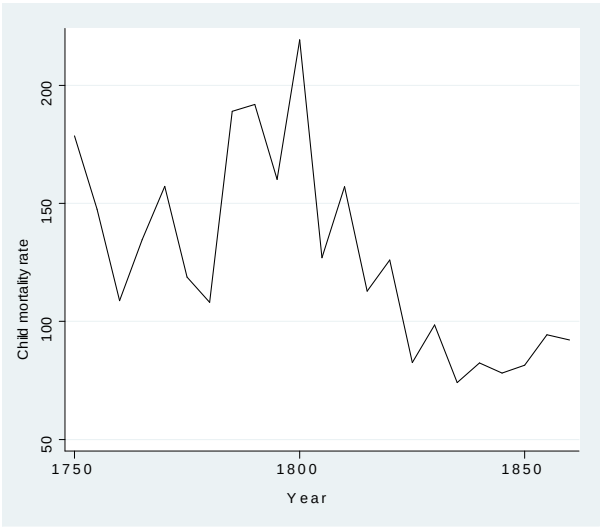
Figure 2: Smallpox mortality share in Sweden, 1774-1860



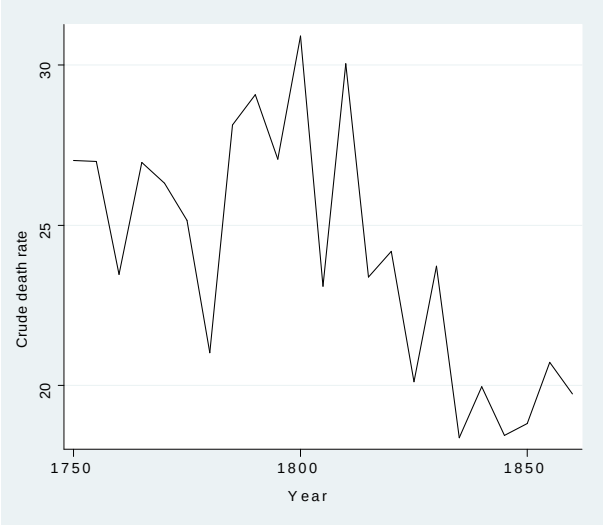
Notes: The data are five-year averages for smallpox mortality for all ages as a share of total mortality starting in 1774. The "year" 1780 includes the period 1774-1779 corresponding to the first period that only includes smallpox. The solid vertical indicates the years 1796-1801, which is the period just prior to vaccination. Source: Sköld (1996).

Figure 3: Main outcome variables aggregated to the national level, 1750-1860

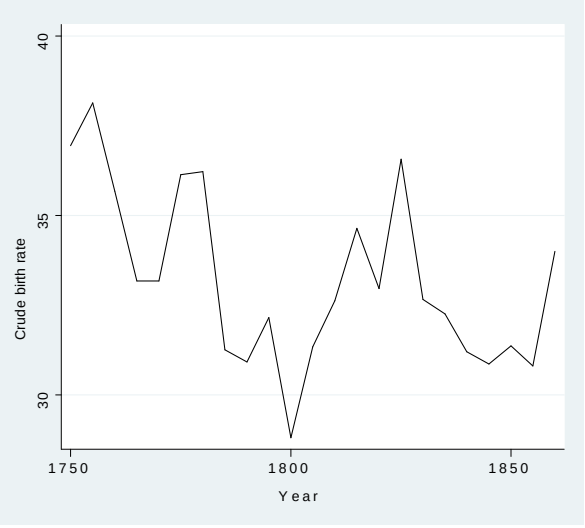
Panel A: Infant mortality rate



Panel B: Child mortality rate



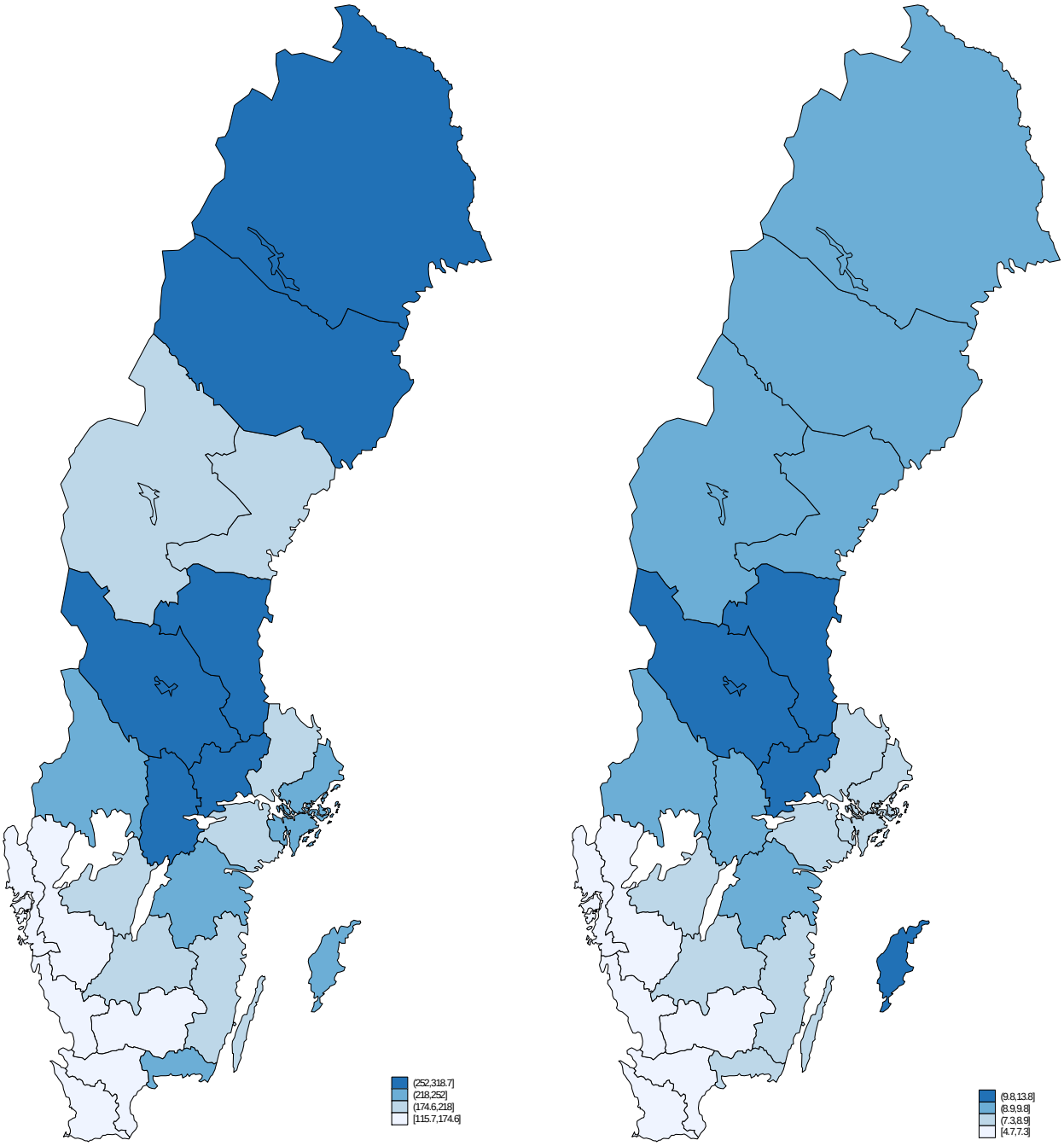
Panel C: Crude death rate



Panel D: Crude birth rate

Notes: These data show the development of infant mortality, child mortality, crude death rate, and crude birth rate at the national level. Source: SHiPS and the authors' own calclulations.

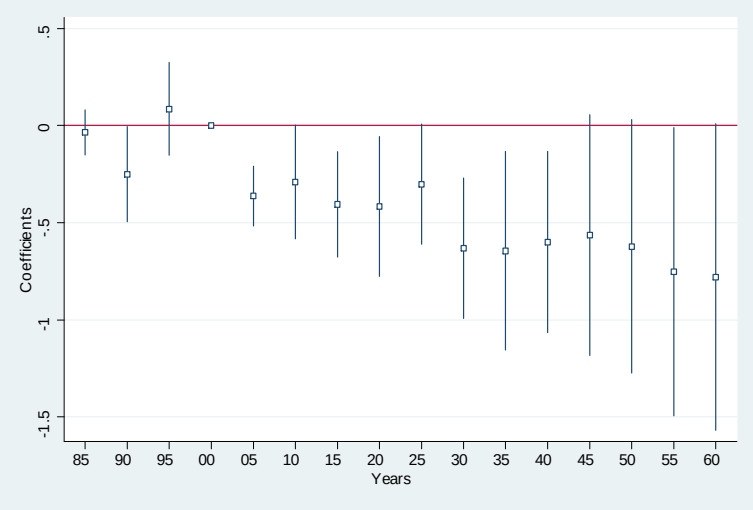
Figure 4: Smallpox mortality rate and share in Sweden, 1796-1801



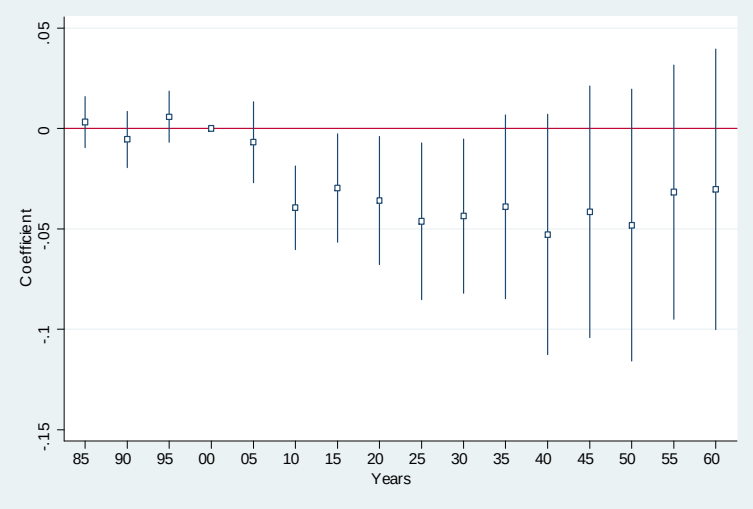
Panel A Panel B

Notes: Panel A shows the average smallpox mortality rate over the period 1796-1801. Panel B shows the average smallpox mortality share out of total mortality over the period 1796-1801. Source: Sköld (1996).

Figure 5: Event-study analysis, 1780-1860



Panel A



Panel B

Notes: This graph reports estimated differences in infant mortality (panel A) and the crude birth rate (panel B) in the treatment-intensity measure for the period 1780-1860 relative to the omitted year 1800, by estimating the outcomes on SMR_j^I, county and time fixed effects, county-specific linear time trends, and initial outcomes, for each year. The estimate for 1780 is also omitted due to the county-specific linear time trends. The vertical lines indicate 95 percent confidence intervals, based on robust standard errors clustered at the county level.

Table 1: Smallpox mortality per 100,000 by age group

	0 years	1-2 years	3-4 years	5-9 years	10-24 years	25-49 years	50 years+
1788-92	2471	1339	820	293	40	2	1
1806-10	765	486	289	119	15	1	1
1831-35	410	81	39	15	10	15	1
1850-54	404	68	n/a	19	20	23	6

Notes: The table reports age-specific smallpox mortality rates for different time periods in Sweden. Source: Sköld (1996).

Table 2: Smallpox mortality before and after compulsory vaccination

		Smallpox mortality rate:	
Division:	Period:	age 0–5	age 5 +
London	1851-60	130	13
	1861-70	116	14
South-Eastern	1851-60	56	8
	1861-70	35	7
South Midland	1851-60	62	9
	1861-70	39	7
Eastern	1851-60	47	5
	1861-70	27	6
South-Western	1851-60	95	9
	1861-70	37	4
West Midland	1851-60	123	10
	1861-70	64	7
North Midland	1851-60	69	6
	1861-70	39	4
North-Western	1851-60	113	5
	1861-70	62	8
York	1851-60	116	8
	1861-70	107	10
Northern	1851-60	117	10
	1861-70	78	11
Welsh	1851-60	164	17
	1861-70	54	9
Average	1851-60	99.3	9.09
	1861-70	59.8	7.91

Notes: The table reports the smallpox mortality rates before and after compulsory vaccination for different geographical areas and age groups. Source: First report of the Royal Commission Appointed to Inquire into the Subject of Vaccination.

Table 3: The vaccination method with alternative cutoffs

	Placebo treatment period is 1750–1800.						
	False cutoff assumption are $\tau >$						
	1755	1760	1765	1770	1775	1780	1785
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Panel A: Infant mortality						
$SMR_j^I \times 1$[see column]	0.169	0.0626	-0.0364	-0.0157	0.00238	0.0313	0.0643
	(0.103)	(0.0696)	(0.0771)	(0.102)	(0.0937)	(0.0701)	(0.0669)
	Panel B: Crude birth rate						
$SMR_j^I \times 1$[see column]	0.0118	0.00900	0.00808	0.00500	0.00759	0.00790	0.00637
	(0.00741)	(0.00786)	(0.00544)	(0.00637)	(0.00671)	(0.00837)	(0.0110)
Controls $\times 1$[see column]:							
initial mortality	yes	yes	yes	yes	yes	yes	yes
initial fertility	yes	yes	yes	yes	yes	yes	yes
Observations	4,565	4,565	4,565	4,565	4,565	4,565	4,565

The table reports least squares estimates weighted by log population size in 1800. The outcome variables are the infant mortality rate (Panel A) and the crude birth rate (Panel B). The placebo pre-treatment period is 1750-1800 (every five years), and the false cutoff assumption is indicated in the top row of each column. $SMR^I$is the smallpox mortality rate in 1796–1801, which is interacted with the various "false" indicators. Initial mortality is the infant mortality rate in 1800, and initial fertility is the crude birth rate in 1800, both interacted with the false indicators. All regressions include county and year fixed effects. Due to data availability in the placebo-treatment period, this is a balanced sub-sample of the baseline sample. Constants are not reported. Robust standard errors are clustered at the county level.

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

Table 4: Vaccination and infant mortality

	Dependent variable: infant mortality				
	(1)	(2)	(3)	(4)	(5)
vaccination	-0.327**	-0.352**	-0.345**	-0.524***	-0.531***
	(0.124)	(0.128)	(0.131)	(0.117)	(0.117)
law 1816					-0.451***
					(0.130)
Controls $\times 1[\tau > 1801]$:					
initial mortality	no	yes	yes	yes	yes
intial fertility	no	no	yes	yes	yes
County linear trends	no	no	no	yes	yes
Std. beta:					
vaccination	-0.361	-0.390	-0.381	-0.579	-0.586
law 1816					-0.095
Observations	10,878	10,878	10,878	10,878	10,878

Notes: The table reports least squares estimates weighted by log population size in 1800. The outcome variable is the infant mortality rate, measured as the number of deaths per 1,000 live-born children at the parish level between 1795 and 1860 (every five years). *Vaccination* is the smallpox mortality rate in 1796–1801 interacted with a time indicator that equals one after 1801. *Law 1816* is constructed as the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811–1815) interacted with a time indicator that equals one after 1815. Initial mortality is the infant mortality rate in 1800 and initial fertility is the crude birth rate in 1800. All regressions include county and year fixed effects. Constants are not reported. Robust standard errors are clustered at the county level.

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

Table 5: Additional mortality-related outcomes

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	vac. rate	smallpox mortality	child mortality	death rate	infant boys	infant girls
vaccination	0.0944	-1.205***	-0.528***	-0.0303***	-0.538***	-0.486***
	(0.0620)	(0.0442)	(0.107)	(0.00763)	(0.107)	(0.147)
law 1816	0.257**	-0.910***	0.756*	0.0330	-0.383**	-0.449**
	(0.113)	(0.123)	(0.405)	(0.0378)	(0.158)	(0.172)
Controls $\times 1[\tau > 1801]$**:**						
initial mortality	yes	yes	yes	yes	yes	yes
initial fertility	yes	yes	yes	yes	yes	yes
Std. beta:						
vaccination	0.301	-1.617	-0.535	-0.320	-0.427	-0.409
law 1816	0.155	-0.271	0.145	0.067	-0.058	-0.072
Observations	10,314	10,101	7,727	10,877	10,878	10,878

Notes: The table reports least squares estimates weighted by log population size in 1800. The outcome variables are the vaccination rate in column (1), the smallpox mortality rate in column (2), the child mortality rate in column (3), the death rate in column (4), and the infant mortality rate by sex in columns (5) and (6) at the parish level between 1795 and 1860 (every five years). *Vaccination* is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801. *Law 1816* is constructed as the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815. In columns (1), (2), (5), and (6) initial mortality refers to the infant mortality rate in 1800, while in column (3) initial mortality refers to child mortality in 1800 and in column (4) to the death rate in 1800. Initial fertility is the crude birth rate in 1800. All regressions include county and year fixed effects, and county-specific linear time trends. Constants are not reported. Robust standard errors are clustered at the county level.

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

Table 6: Vaccination, fertility and population growth

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
vaccination	-0.0210***	-0.101***	-0.00207	0.00877	-0.0736	0.00944
	(0.00566)	(0.0269)	(0.00555)	(0.00667)	(0.154)	(0.00861)
law 1816	0.0171	0.0554	0.0300	0.00820	0.791	-0.0158
	(0.0249)	(0.168)	(0.0222)	(0.0273)	(0.584)	(0.0411)
Controls $\times 1[\tau > 1801]$**:**						
initial mortality	yes	yes	yes	yes	yes	yes
intial outcome	yes	yes	yes	yes	yes	yes
Std beta:						
vaccination	-0.300	-0.300	-0.031	0.134	-0.055	0.085
law 1816	0.046	0.030	0.087	0.022	0.107	-0.027
Observations	10,878	8,845	10,878	7,727	8,845	10,877

Notes: The table reports least squares estimates weighted by log population size in 1800. The name of the specific outcome variable is indicated in the top row. *Vaccination* is the smallpox mortality rate in 1796–1801 interacted with a time indicator that equals one after 1801. *Law 1816* is constructed as smallpox mortality prior to the vaccination law of 1816 (i.e., 1811–1815) interacted with a time indicator that equals one after 1815. Initial mortality is the infant mortality rate in 1800 and the initial outcome is the specific outcomes (indicated in the top tow) measured in 1800. All regressions include county and year fixed effects, and county-specific linear time trends. Constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Table 7: The impact of infant mortality on fertility and population growth

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
	Panel A: OLS estimates					
infant mortality	-0.00267**	-0.0244***	-0.0322***	-0.0332***	-0.172***	-0.0494***
	(0.00114)	(0.00500)	(0.000939)	(0.000744)	(0.0175)	(0.00197)
	Panel B: 2SLS estimates					
infant mortality	0.0359***	0.175***	0.000658	-0.0165*	0.0916	-0.0151
	(0.0109)	(0.0546)	(0.00933)	(0.00990)	(0.265)	(0.0129)
Std. beta	0.461	0.466	0.009	-0.218	0.061	-0.124
Kleibergen-Paap F-stats.	12.41	10.73	12.32	13.54	10.95	13.45
Anderson-Rubin [p-value]	[0.001]	[0.000]	[0.382]	[0.336]	[0.380]	[0.509]
Hansen-J [p-value]	[0.251]	[0.605]	[0.231]	[0.974]	[0.230]	[0.576]
Controls $\times 1[\tau > 1801]$**:**						
initial mortality	yes	yes	yes	yes	yes	yes
intial outcome	yes	yes	yes	yes	yes	yes
Observations	10,878	8,845	10,878	7,727	8,845	10,877

Notes: Panel A reports OLS estimates and panel B reports 2SLS estimates, using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 and *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments. The name of the specific outcome variable is indicated in the top row. The explanatory variable is the infant mortality rate, which is the number of infant deaths per 1,000 live-born children. Initial mortality is the infant mortality rate in 1800 and the initial outcome is the specific outcome (indicated in the top tow) measured in 1800. All the regressions are weighted by log population size in 1800 and include county and year fixed effects, and county-specific linear time trends. Constants are not reported. Robust standard errors are clustered at the county level.

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

Table 8: Second-stage robustness analysis I

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
	Panel A: controlling for initial population density and urbanization					
infant mortality	0.0373***	0.184***	0.00147	-0.0171	0.0518	-0.00773
	(0.0133)	(0.0646)	(0.0112)	(0.0118)	(0.269)	(0.0170)
observations	10,878	8,845	10,878	7,727	8,845	10,877
	Panel B: controlling for initial "income"					
infant mortality	0.0340***	0.165***	-0.000504	-0.0149*	0.0933	-0.0161
	(0.0101)	(0.0492)	(0.00859)	(0.00854)	(0.127)	(0.0122)
observations	10,313	8,369	10,314	7,422	8,369	10,313
	Panel C: controlling for initial human capital					
infant mortality	0.0351***	0.173***	-0.000167	-0.0175**	0.165	-0.0148
	(0.0102)	(0.0528)	(0.00860)	(0.00887)	(0.181)	(0.00971)
observations	10,878	8,845	10,878	7,727	8,845	10,877
	Panel D: controlling for initial female population share					
infant mortality	0.0369***	0.186***	0.00136	-0.0161	0.153	-0.0138
	(0.0119)	(0.0637)	(0.0103)	(0.0111)	(0.295)	(0.0149)
observations	10,062	8,845	10,062	7,216	8,845	10,062
	Panel E: controlling for initial marriage rate					
infant mortality	0.0360***	0.175***	0.000666	-0.0166*	0.0892	-0.0150
	(0.0109)	(0.0547)	(0.00934)	(0.00995)	(0.266)	(0.0129)
observations	10,808	8,795	10,808	7,692	8,795	10,807

Notes: All panels report 2SLS estimates using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801, and *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments. The name of the specific outcome variable is indicated in the top row. The explanatory variable is the infant mortality rate, which is the number of infant deaths per 1,000 live-born children. Initial mortality is the infant mortality rate in 1800 and the initial outcome is the specific outcome (indicated in the top tow) measured in 1800. All the regressions are weighted by log population size in 1800 and include county and year fixed effects, county specific linear time trends, and initial outcomes (measured in 1800) interacted with the time indicator. To this baseline specification, panel A controls for the initial urbanization rate and population size interacted with the time indicator, panel B controls for initial income (i.e., price rye and log rye production) interacted with the time indicator, panel C controls for initial measures of human capital interacted with the time indicator, panel D controls for the initial female reproductive share of the population interacted with the time indicator, and panel E controls for the initial marriage rate interacted with the time indicator. First-stage results and constants are not reported. Robust standard errors are clustered at the county level.

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

Table 9: Second-stage robustness analysis II

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
	Panel A: controlling for county-specific quadratic trends					
infant mortality	0.0360***	0.176***	0.000742	-0.0164*	0.0942	-0.0150
	(0.0109)	(0.0548)	(0.00934)	(0.00990)	(0.266)	(0.0129)
observations	10,878	8,845	10,878	7,727	8,845	10,877
	Panel B: controlling for initial outcomes in 1795					
infant mortality	0.0365***	0.195***	0.00108	-0.0181*	0.0791	-0.0140
	(0.0110)	(0.0594)	(0.00949)	(0.0102)	(0.295)	(0.0144)
observations	10,878	8,005	10,878	6,609	8,005	10,877
	Panel C: controlling for parish fixed effects					
infant mortality	0.0794***	0.413***	0.0344	0.0230	0.924*	0.0273
	(0.0252)	(0.117)	(0.0217)	(0.0253)	(0.487)	(0.0345)
observations	10,878	8,784	10,878	7,631	8,784	10,877
	Panel D: without counties in the South					
infant mortality	0.0369***	0.186***	0.00136	-0.0161	0.153	-0.0138
	(0.0119)	(0.0637)	(0.0103)	(0.0111)	(0.295)	(0.0149)
observations	7,966	6,503	7,966	5,864	6,503	7,965
	Panel E: without counties in the North					
infant mortality	0.0177*	0.0652	-0.0155*	-0.0363***	-0.291	-0.0278
	(0.0103)	(0.0417)	(0.00791)	(0.00937)	(0.279)	(0.0183)
observations	9,870	8,003	9,870	6,910	8,003	9,869

Notes: All panels report 2SLS estimates using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 and *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments. The name of the specific outcome variable is indicated in the top row. The explanatory variable is the infant mortality rate, which is the number of infant deaths per 1,000 live-born children. Initial mortality is the infant mortality rate in 1800 and the initial outcome is the specific outcome (indicated in the top tow) measured in 1800. All the regressions are weighted by log population size in 1800 and include county and year fixed effects, county specific linear time trends, and initial outcomes (measured in 1800) interacted with the time indicator.To this baseline specification, panel A controls for county-specific quadratic trends, panel B controls for the outcomes in 1795 interacted with the time indicator, panel C replaces the county fixed effects with parish fixed effects, and panels D and E take out the 5 most northern and southern counties (in the data) respectively. All estimations exclude the most northerly county (Norrbotten), First-stage results and constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.