Fertility and early-life mortality: Evidence from smallpox vaccination in Sweden

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Abstract

We examine how the introduction of smallpox vaccination affected early-life mortality and fertility in Sweden during the first half of the 19th century. We demonstrate that parishes in counties with higher levels of smallpox mortality prior to the introduction of vaccination experienced a greater decline in infant mortality afterwards. Exploiting this finding in an instrumental-variable approach reveals that this decline had a negative effect on the birth rate, while the number of surviving children and population growth remained unaffected. These results suggest that the decline in early-life mortality cannot account for the onset of the fertility decline in Sweden.

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

JEL: J10; J13; I15

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1 Introduction

A crucial question in the field of economic growth and development is whether health improvements make a population richer (Weil, 2014). The research by Acemoglu and Johnson (2007) suggests that this is not the case. 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 fertility adjusts, and the evidence on this mechanism is scant (Bleakley, 2010). In this paper, we aim to fill this gap in the literature by investigating the causal effect of early-life mortality on fertility.[1]

To carry out this investigation, we face the challenge that early-life mortality and fertility are most likely determined by the same factors of which some are unobservable (see e.g., Schultz, 1997). We address this issue by using pre-vaccination variation in smallpox mortality at the county level 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 vaccine successfully developed and the major medical innovation of the late 18th and early 19th century (Cutler et al., 2006). We focus on the case of Sweden for which historical data on fertility, infant and child mortality exist at the parish level. Vaccination in Sweden started at the end of 1801 and was widely distributed at zero or low cost to citizens which makes the uptake of vaccine unlikely to be correlated with regional income levels.[2]

Exploiting the introduction of the smallpox vaccine to identify the impact of early-life mortality on fertility has a number of appealing features compared to earlier work. First, since smallpox affected mainly infants, it is much clearer through which mechanism the elimination of the disease works. Earlier work by Acemoglu and Johnson (2007) and Hansen (2014) use the timing of elimination of a host of infectious diseases combined with prevalence rates prior to those interventions to identify the impact of health on wealth.[3] In terms of mortality across

[1] In the literature on the fertility transition, historical evidence indicates that the decline in early-life mortality cannot account for the fertility decline since it started beforehand (Galor, 2011). By contrast, Kalemli-Ozcan (2003, 2008) argues that the decline in the uncertainty of the survival rates of children, brought on by lower child mortality, leads parents to decrease their precautionary demand for children, and this reduces fertility.

[2] Moreover, Guinnane (2011) lists smallpox vaccination among a list of 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.

[3] Using the same type of estimation strategy, Hansen (2013) demonstrates that the decline in infectious-disease mortality in the second half of the 20th century is positively related to human capital accumulation

the life cycle, their instrument could work through a much broader set of mechanisms than the one used in the present paper. Second, since smallpox vaccination was the first vaccination and the major medical innovation at the time, it is difficult to think of medical interventions that occurred at the same time and also correlate with pre-intervention smallpox mortality rates. Third, Sweden also introduced a compulsory vaccination law in 1816 which adds an additional plausibly exogenous source of time variation in our empirical framework. This allows us to construct an additional instrument for early-life mortality.

Our empirical analysis documents that the advent of vaccination in 1801 together with the introduction of compulsory vaccination in 1816 had profound negative effects on the infant mortality rate in Sweden. In particular, a one-standard-deviation higher level of pre-vaccination smallpox mortality is associated with a decrease in infant mortality of about 20 deaths per 1000 live births, while compulsory vaccination yields to infant mortality a reduction of about 5 deaths per 1000 live births. Using these two intervention episodes to obtain the causal effect of early-life mortality on fertility behavior, our results show, in line with Galor (2011), that while the decline in infant mortality has a negative effect on fertility, there is no statistically significant effect on the number of surviving children. Because of the fertility adjustment, we find that the decline in infant mortality has no effect on natural population growth.[4]

The Swedish case is interesting for a number of additional reasons. First, smallpox was a severe disease in Sweden which killed approximately 10 percent of the population in the second half of the 18th century (Fenner et al., 1988). Second, Sweden and the other Scandinavian countries provided an example for the rest of the world. "Subject to severe endemic and epidemic smallpox before vaccination became available, they eliminated smallpox by the end of the 19th century." (Fenner et al., 1988). Third, Sweden has been used as a typical example of the fertility transition (Weil, 2009, p.104–105) and health transition (Weil, 2014, p.634).

Our paper contributes to the recent literature on the historical fertility decline based on panel data. Angeles (2010), Murtin (2013) and Hansen et al. (2014) all estimate dynamic panel models using lagged values as instruments for endogenous variables. Angeles (2010) concludes that child mortality plays a large role for fertility decline, while Murtin (2013) and Hansen et al.

across countries.

[4]This finding also 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, so that population growth is unaffected.

(2014) suggest that infant and child mortality is not robustly correlated with fertility.[5] As these studies use lagged variables as instruments for infant or child mortality, this naturally brings into question whether their findings can be given a causal interpretation. A notable exception is Murphy (2010) who finds little effects of infant mortality by instrumenting infant mortality by deviations from mean temperature in a panel study of fertility in French departements from 1876-1896. While an improvement over other studies, the exclusion restriction may be questioned as it has been argued that temperature directly effects fertility. These effects may "result from changes in coital frequency or from direct physiological effects" (Lam and Miron, 1996, p.292). Conley et al. (2007) use the percentage of population at risk of malaria as an instrument for infant mortality and find a strong, positive impact on fertility, but they also mention that malaria risk may affect fertility directly. Compared to the previous studies we provide an identification strategy for which it is less plausible that the introduction of vaccine and the pre-vaccination distribution of smallpox has a direct impact on fertility.[6]

Our paper also builds on the literature on health, education, and economic growth which uses disease eradication in a differences-in-differences framework to obtain identification. Our identification strategy builds on Bleakley (2007) who combines the timing of hookworm eradication in the US South with the pre-eradication distribution of hookworm to obtain its effects on education.[7] In a similar vein, we use the pre-vaccination distribution of smallpox mortality to capture what areas would experience the greatest decreases in infant mortality after the intervention. We also 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.

Moreover, our paper belongs to a relatively small literature on the causes of the Swedish fertility transition using disaggregated data. Schultz (1985), Dribe (2009) and Lagerlöf (2014)

[5] All these studies consider relatively long panels. Angeles (2010) covers 1955–2000 for a world sample, Murtin (2013) covers 1870–2000 for a world sample, and Hansen et al. (2014) cover 1840–1980 for US states.

[6] An older literature also proposed instrumental variables estimation. Benefo and Schultz (1996) use presence of malaria eradication and child immunization programmes as an instrument for child mortality in Ghana and Cote d'Ivoire. These programmes tend "to be fielded in poorer, more remote regions of Ghana, where women are relatively less educated" (Benefo and Schultz, 1996, p. 133), raising doubts about the validity of these instruments. Our strategy avoids their issue by the fact that vaccination reached the Swedish regions at the same time, and the fact that vaccination was not conditional on poverty.

[7] Bleakley and Lange (2009) also find that fertility decreased upon the eradication of hookworm in the US south, whereas Lucas (2013) finds that fertility increased after the eradication of malaria in Sri Lanka.

apply 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, 2014) were important determinants of the fertility transition in Sweden. Compared to these studies we use an identification strategy that exploits the pre-vaccination distribution of smallpox mortality together with the introduction of the smallpox vaccine to obtain a causal effect of early-life mortality on fertility at the parish level.

The rest of the paper is structured as follows. Section 2 presents a simple theoretical model and discusses the theoretical predictions regarding the relation between early-life mortality and fertility. Section 3 provides background on the history of smallpox vaccination in Sweden and descriptive evidence on early-life smallpox mortality. Section 4 presents the data. Section 5 explains our estimation framework. Sections 6 presents the empirical results. Section 7 concludes.

2 Theory

This section outlines a simple one-period static model that provides us with some straightforward testable predictions on the effects of early-life mortality on fertility (children ever born) and net fertility (i.e., surviving children).

Consider a household that derives utility from normal consumption, c, and the number of born children, n^b:

$$V = \ln c + \phi \ln n^b, \tag{1}$$

where ϕ is the infant (or child) survival rate (i.e., $1 - \phi =$ the infant mortality rate). The construction of the proposed utility function in equation (1) implies that the household receives utility from surviving children, and that the marginal utility from born children is increasing in the survival rate.[8] The household is confronted with the following budget constraint:

[8] This way of theoretically modelling the relationship between (infant) mortality and fertility has recently been applied by Strulik (2014), for example, who also find that the decline in infant mortality plays no role in explaining the fertility transition.

$$(\rho + \phi)\, n^b + c = w, \tag{2}$$

where $\rho + \phi$ is the cost of raising a born child. This cost involves a fixed term, ρ, which is independent of the survival rate,[9] such that the cost of raising a non-surviving child is always larger than zero; otherwise the cost per child is increasing in the survival rate, ϕ. The total household income is denoted by w.

The problem for the household consists of maximizing equation (1) subject to equation (2). The explicit solution for the number of children born is:

$$n^b = \frac{\phi}{(1+\phi)\,(\phi+\rho)} w. \tag{3}$$

The number of surviving children is given by:

$$n^n = \phi n^b = \frac{\phi^2}{(1+\phi)\,(\phi+\rho)} w. \tag{4}$$

It is evident from equations (3) and (4) that if the fixed costs of children are not too large (i.e., $\phi > \rho^{\frac{1}{2}}$), fertility, n^b, is decreasing in the survival rate, whereas the number of surviving children, n^n, is (unambiguously) increasing concave in the survival rate. Intuitively, this happens because the positive extensive effect—which comes from the fact that the number of surviving children is per definition increasing in the survival rate (holding the number of born children constant)—always dominates the negative intensive effect (i.e., $\frac{\partial n^b}{\partial \phi} < 0$).

In sum, this theory predicts that an increase in the survival rate (i.e., a decline in infant mortality) has the following implications:

1. a negative effect on the number of born children if $\phi > \rho^{\frac{1}{2}}$,

2. a small but positive effect on the number of surviving children.

It is worthwhile to note that these predictions are relatively robust. First, they are independent of the curvature of the utility function, that is, similar results are obtained assuming $u'(x) > 0$ and $u''(x) < 0$, $x = c, n^b$. Second, if we assume that the cost of children is

[9] One can think ρ as a cost which is related to reduced productivity in the labor market during pregnancy.

related to the household unit-time endowment, so that the budget constraint takes on the form $\left(\frac{\rho}{w}+\phi\right) n^b w + c = w$, the testable predictions also remain unchanged. On the other hand, suppose that the household's preferences instead are represented by the utility function $W = \ln c + \ln \phi n^b$ as in Galor (2011), then while our first prediction remains unaffected, the effect on surviving children is now predicted to be zero.[10]

3 Historical Background

In this section, we discuss the historical background for the introduction of vaccination and the subsequent compulsory vaccination law which we use in our empirical analysis. Further, we substantiate that smallpox, which is also known as 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 century is not qualitatively different from those of other countries for which early data are also available.

3.1 Introduction of vaccination and compulsory vaccination law in Sweden

Smallpox vaccination came into use in Sweden at the end of 1801 (Peterson, 1912; Sköld, 1996), and was made compulsory in 1816 (Sköld, 1996). In this subsection, we discuss how smallpox vaccination was introduced and provide further historical background information that is relevant to understand our empirical setting.

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 a high risk of dying from the procedure, it could serve as a source

[10] See Galor (2011) for the theoretical conditions under which prediction (2) is reversed.

of infection for those not inoculated, the cost of inoculation, and general conservatism in the public. Sköld (1996, p. 355) 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 he inoculated him with smallpox, and as there was no reaction, he 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, p. 359). After 1803, there was no official discussion of inoculation. It was still used in some areas, but only when vaccination was not possible. The first vaccinations in Sweden have been credited to Eberhard Zacharia Munch of Rosenschöld, who carried these out at the end of 1801 (Sköld, 1996, p.375). 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 should learn to vaccinate (Sköld, 1996, p.403). This implied that there was no monopoly on vaccination. Dribe and Nystedt (2003, p.11) note 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. This suggests that there are good reasons to believe that there were no differences by social class in the practise 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 of 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. If they did not, they would have to pay a fine and in the case that they could not pay, they would be imprisoned on a diet of water and bread (Sköld, 1996, p.449). Sköld (1996, p.255) concludes that "the effect was immediate, and between 1816 and 1820 more than 73 per cent of all children were vaccinated." An appealing feature of the compulsory vaccination law is that it was targeted the group from 0-2 years, which suggests that it would mainly affect infants and young children (see also Section 3.2 for more details).

To gauge whether there is a substantial change in smallpox mortality when the vaccination was introduced we plot the smallpox mortality rate and the smallpox share of total mortality from 1750-1859 in Figures 1 and 2.[11] Both graphs indicate a negative trend in both variables, but yet there is a break in this trend in 1802 after vaccination became available. The levels of both variables drop markedly, and while a negative trend still appears after 1802, the slope is flatter after this point.

Figures 1 and 2 about here

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 evidence, and then consider suggestive evidence from other countries.

3.2.1 Evidence from Sweden

The aggregate Swedish data for smallpox mortality per 100,000 by age and time (1788–1854) compiled by Sköld (1996) 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

[11] The graphs represent averages across counties. Since there are no data on smallpox mortality available at county level between 1774 and 1795, mortality in this period is interpolated.

decreased monotonically over time, this is not the case for the groups above 5 years who all experienced small increases at the end of the period. Overall, the descriptive evidence clearly indicates that smallpox affected much more infants and young children and that the effect for this age group was long-lasting.

Table 1 about here

3.2.2 Evidence from other contexts

The aggregate Swedish evidence indicates that smallpox mainly affected infants and young children. In this subsection we investigate whether the Swedish evidence is similar to other countries. In the late 1880s, a British Royal Commission was appointed to investigate the effects of vaccination. and collected various data on smallpox deaths which are of our interest here. Two cases are of particular interest. The first case is presented in Table 2, which contains data for the 1795-96 epidemic in Posen. As in Sweden, mortality rates were much higher for infants and young children. Table 2 indicates that infants who are under 1 year had a mortality rate of 35.9 per 100 which was three times the one for 5-10 year olds.

A second source of suggestive evidence comes from England and Wales after 1853 (the year in which vaccination was made compulsory there). Table 3 shows that in 1851–1860 —the decade in which compulsory vaccination was introduced— mortality of children between 0-5 years was systematically higher than in the following decade 1861–70. On average mortality rates across registration divisions fell from 99.3 to 59.8 per 100,000. For children above 5 years, there was a modest fall was from 9.09 to 7.91 per 100,000. This again corroborates that mainly infants and young children were affected by smallpox vaccination and associated compulsory vaccination laws.[12]

Table 2 about here

[12]Davenport et al. (2011) use date 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.

Table 3 about here

4 Data

This study links aggregate (county) data on smallpox mortality from Sköld (1996) with parish level data on birth rates and infant and child mortality from Swedish Historical Population Statistics (SHiPS). The later statistics contain digitized information from *Bastatabellen* which is a compilation of *Tabellverket*. *Tabellverket* contains information about the population in Swedish parishes during the period 1749 to 1859, as reported by the clergymen in large forms of tables to the Tabellkommissionen in Stockholm. These data provide parish-level information on birth rates, infant mortality, child mortality, total mortality rate, and population size, for example.[13]

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. However, because of lack of data on infant mortality for the parishes in Norbotten in the early periods, we end up using 24 counties in the analysis. Our analysis starts in 1795, which allows us to consider two periods prior to the vaccine introduction. One additional advantage of starting the main analysis in 1795 is that we avoid having to deal with the fact that smallpox deaths were reported together with measles before 1774 (Fridlizius and Ohlsson, 1984). Moreover, 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.

Sköld (1996) also provides vaccination rates which are calculated as the number of children vaccinated as a proportion of children born in the previous 5 year period. For example, for 1810-1815 the variable is calculated as the number of children vaccinated divided by children born in the period 1809-1814 (Sköld, 1996, p. 571). We also add some control variables, which generally are introduced as the analysis progresses. The dataset we end up using is a 5-year balanced panel from 1795–1860 with 24 counties and 777 parishes. Further details about the data and summary statistics are provided in the supplementary online appendix.

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

5 Identification strategy

This section describes how we propose to identify the effect of early-life mortality on fertility and surviving children. Our baseline estimation equation takes the following functional form:

$$y_{ijt} = \alpha \; \textit{Infant mortality}_{ijt} + \mathbf{X}_{ijt}\beta + \delta_j + \tau_t + \varepsilon_{ijt}, \tag{5}$$

where y_{ijt} denotes the outcome of interest — the birth rate, surviving children, or natural population growth — in a Swedish parish i of county j at time $t \in (1795; 1860)$. The main variable of interest, $\textit{Infant mortality}_{ijt}$, is the infant mortality rate as measured by the number of infant deaths per 1000 live births. We further include a set of parish-specific control variables, $\mathbf{X}_{ijt}$, such as, initial infant mortality and population size (interacted with the time indicator variable; see below) and county (δ_j) and time (τ_t) fixed effects.[14] We cluster the error term ε_{ijt} at the county level to ensure that the standard errors of our estimates are robust to arbitrary correlation across parishes in each Swedish county. We restrict the sample to parishes that are observed for all years. In the online appendix we also report the estimates for the unbalanced panel of parishes for which we obtain qualitatively similar results.

While the panel structure of the dataset allows us to perfectly control for time invariant county-specific (or parish-specific) characteristics affecting both mortality and fertility, the OLS estimate of α does not necessarily measure the causal effect because of reverse causation, that is fertility is likely to also influence mortality, and omitted variable bias due to time varying unobserved factors, for example. For these reasons, our empirical strategy exploits two important episodes in the relation to the advancement of vaccination against smallpox, which induced a sharp decline in smallpox mortality. The first episode is the introduction of the vaccination method after 1801, and the second is the enactment of the compulsory vaccination law in 1816.

The time variation from these episodes combined with cross-county differences in pre-treatment smallpox mortality rates represent our differences-in-differences approach, which we use as the first stage of our two-stage least squares (2SLS) estimation approach to estimate the effect of mortality on fertility.

[14] We also also report estimates on infant mortality that control for parish fixed effects instead of county fixed effects.

We consider the following first-stage relationship:

$$\begin{aligned} \textit{Infant mortality}_{ijt} = \pi_1 \; S_j^{pre\_I} \times I_t^{t>1801} + \pi_2 \; S_j^{pre\_II} \times I_t^{t>1816} \\ \mathbf{X}_{jt}\bar{\beta} + \bar{\delta}_j + \bar{\tau}_t + \bar{\varepsilon}_{ijt}, \end{aligned} \tag{6}$$

where $S_j^{pre\_I}$ is the smallpox mortality rate measured prior to the introduction of vaccination in 1796-1801 in county j and $I_t^{t>1801}$ is an indicator variable that equals one for the period after 1801 (i.e., 1805, 1810, .., 1860). In a similar way, $S_j^{pre\_II}$ is the smallpox mortality rate measured just before the enactment of the compulsory vaccination law of 1816 and $I_t^{t>1815}$ is an indicator that equals one afterwards. The remaining variables are defined above. Notice, the two 'shock variables', $S_j^{pre\_I} \times I_t^{t>1801}$ and $S_j^{pre\_II} \times I_t^{t>1816}$, which we shall refer to as *Vaccination* and *Law 1816*$_{jt}$ in the regression tables, vary only at the county-by-year level (we only have data on smallpox mortality at the county level). The regressions are weighted by initial parish-population size, so that the estimates reflect an average population effect.[15] If we find that $\hat{\pi}_1 < 0$ and $\hat{\pi}_2 < 0$, then the introduction of the vaccination method and the compulsory vaccination law decreased infant mortality.

Finally, because 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 was given a more advantageous shock (in terms of reducing mortality) when the vaccination technology diffused. However, in contrast to many previous studies, which follow a similar approach (e.g., Bleakley, 2007; Acemoglu and Johnson, 2007; Hansen, 2014), we can study whether the counties with a higher burden of smallpox mortality actually had a higher level of adoption of the new technology. This is possible because we have data on vaccination rates at the county level.

[15] The unweighted least squares regressions yield similar results and are available from the authors upon request.

6 Empirical Results

6.1 The effect of vaccination on infant mortality

6.1.1 Main results

Table 4 presents our main results for the first-stage relationship. The estimation equation is (6) and the method of estimation weighted least squares. Column (1) demonstrates that infant mortality rates are strongly and negatively affected by the introduction of vaccination, as we observe a negative and statistically significant coefficient on the shock variable $S_j^{pre\_I} \times I_t^{t>1801}$, which we refer to as *Vaccination* in the following tables. The point estimate is statistically significant at the 1 percent level.

One concern might be that this coefficient might pick up some sort of convergence or divergence process in outcome at the parish level. Hence, we control in column (2) for the infant mortality rate in 1800 (*Initial mortality*) and the log initial population size (*Initial population*) interacted with the indicator, $I_t^{t>1801}$. The coefficient increases in numerical magnitude, such that a one-standard-deviation increase in smallpox mortality prior to the breakthrough of vaccination is associated with a decline in infant mortality of 16.4 deaths per 1000 live births afterwards, which corresponds to 0.11 of a standard deviation in the pretreatment infant-mortality rate.

Columns (3) and (4) consider the compulsory vaccination law measured by $S_j^{pre\_II} \times I_t^{t>1816}$ (*Law 1816*) as alternative shock variable. While the estimated coefficient is negative and statistically significant in both specifications, we see that controlling for *Vaccination* in column (5), increases the numerical magnitude substantially. In the specifications that only include the shock from the vaccination law but disregard the shock from the introduction of vaccination, the estimate of $\hat{\pi}_2$ is biased towards zero if the two intensity measures (i.e., $S_j^{pre\_I}$ and $S_j^{pre\_II}$) are negative correlated and *Vaccination* has a negative effect on infant mortality (in our case both conditions are satisfied).[16] Thus, in column (5), where *Vaccination* and *Law 1816* are included together the effect increases in magnitude. The magnitude of the estimated coefficient

[16] The negative correlation between $S_j^{pre\_I}$ and $S_j^{pre\_II}$ can be explained by the fact that counties with higher level of smallpox mortality in 1795-1801 received a more favorable shock due to the introduction of vaccination. Accordingly, the level of smallpox mortality could be lower in these places 15 years later.

on *Law 1816* implies that a one-standard-deviation higher level of smallpox mortality before the enactment of the compulsory vaccination law is associated with a decrease in infant mortality of 4.7 deaths per 1000 live births afterwards, which is the same as 0.05 of a standard deviation in the pretreatment infant mortality rate. In this specification, the effect of *Vaccination* is 19.5 deaths per 1000 live births. As the "intention-to-treat" is higher around the first shock, we find that the introduction of vaccination had a large effect on the development of infant mortality compared to the vaccination law.

Table 4 about here

Before presenting the robustness analysis for the baseline first-stage estimates, it is worthwhile to note that we reach the same conclusion estimating a flexible model with the effects for each time period from 1795 to 1860. Table 2a of the supplementary online appendix shows a discontinuity in the coefficients for both interventions around the adoption dates, and the p-values of the F-tests reveal that the coefficients in the post-treatment years are jointly statistically different from the estimated coefficient(s) in the pretreatment year(s) at conventional levels. The results from the flexible specification also suggest that our (pre-treatment) smallpox mortality rates, $S_j^{pre\_I}$ and $S_j^{pre\_II}$, are not correlated with pre-existing trends in infant mortality.

6.1.2 Robustness

In this subsection, we have carried out a number of robustness checks which are based on estimation equation (6). The method of estimation is weighted least squares. First, if counties had different trends in mortality prior to the interventions, the decrease in infant mortality could have happened irrespectively of the interventions. While the presence of initial mortality rates as a baseline control variable should soak up mean reversion in the outcome, Table 5 considers whether pre-existing trends in infant mortality could account for our baseline results. As a first check, columns (1) and (2) show the results from a falsification test where the outcome variable is the infant mortality rate in the 50-year period preceding the introduction of the vaccination

method in Sweden (i.e., 1750–1800). We observe that the coefficients are now positive on both interactions suggesting that our baseline estimates are not capturing a pre-existing downward trend in infant mortality. In the next two columns, we add a placebo intensity measure (i.e., smallpox mortality in 1749–1753), which is the period prior to the introduction of inoculation in Sweden. While the coefficients of interest remain reassuringly stable in both magnitude and statistical significance, the estimated effect of the placebo shock variable is basically zero. Moreover, in that respect, estimating a model (for the period 1750–1795) where we, in the same way, attempt to capture the introduction of the inoculation method, we find that the coefficient on the "inoculation-shock variable" is -0.09 (standard error $= 0.08$).[17] Thus, in line with the view of Sköld (1996), there is no evidence of the inoculation method reducing infant mortality, which also indicates that our interactions do not capture pre-existing trends set in motion from the introduction of inoculation into Sweden. Finally, we control for trend differences across areas by including county-specific time trends. Nevertheless, estimates of this model, reported in columns (5) and (6) show little changes in the estimated $\pi's$.

Table 5 about here

Table 6 reports additional sensitivity tests. The first three columns replace the intensity measure of our interactions (i.e., the smallpox mortality rate) with the share of smallpox mortality out of total mortality. The two new intervention variables, which are indicated *alternative* in the Table 6, take into account the possibility that smallpox mortality is correlated with other diseases. While the literature stresses that smallpox mortality was the only disease which exhibited a significant decline around this period of time, our baseline interactions might capture declines in other diseases as well. However, as observed in columns (1)–(3), the estimates on the "alternative" interactions are also negative and statistically significant at the 1 percent level. This means that parishes in counties with a high share of smallpox mortality before the advent of vaccination and before the vaccination law experienced greater decreases in infant mortality afterwards. Column (4) demonstrates that our baseline estimates are robust to controlling for the number of still births. In some unreported specification, we demonstrate that *Vaccination*

[17] This result is available upon request.

has a small positive and statistically significant effect on still births.[18] This finding is not surprising as parents giving birth in, for example, 1810, which is in the post-treatment period, were not exposed to an environment with less smallpox mortality in general since the method of vaccination was unavailable in their childhood. In the words of the 2SLS strategy followed in Section 7, the coding of the time indicator implies that our interaction, $S_j^{pre\_I} \times I_t^{t>1801}$, is not likely to capture a direct biological effect from the parents on fertility.[19]

Columns (5)–(7) add indicators for the economic environment. Specifically, we add the price of rye and the log of rye production per capita as indicators of county level income (see Dribe et al., 2011). We add these income indicator to our estimation equation, as compliance to the compulsory vaccination law was more costly for poorer families. Yet, we observe that the estimated effect of (compulsory) vaccination remains unaffected by including these controls.

Finally, it is worthwhile to note that we generally obtain similar results controlling for parish fixed effect instead of county fixed effect, which indicates that our results are not driven by unobserved time-invariant factors at the parish level. This also becomes clear when we present our 2SLS estimates in Section 6.2.

Table 6 about here

6.1.3 Alternative outcomes

We next consider alternative outcome variables. Our approach is an intention-to-treat design, and we posit that our measures capture increases in vaccination and decreases in smallpox mortality. The first four columns of Table 7 validate our approach. Columns (1) and (2) exploit the fact that we have a measure for the adoption of vaccination, that is, the outcome variable is now county i's vaccination rate for the children in the age group 0–5 at time t. The estimated coefficients on both interventions are positive and statistically significant at the 5

[18] These results are available from the authors upon request.

[19] Rutten (1993) and Sköld (1996) note that some authors have proposed that male fecundity was affected by smallpox e.g. because infected men would be disadvantaged in the marriage markets due to pockmarks. Sköld (1996, p. 195) demonstrates that infected and vaccinated had similar fecundity levels. Rutten (1993) presents similar evidence for the Netherlands. Thus, the empirical evidence tends to reject this effect of smallpox.

percent level. This implies that counties with a higher level of "intention-to-treat" before the interventions also had higher adoption rate of the new technology afterwards.

The idea in the intention-to-treat design is that equation (6) is the reduced form of the following first stage:[20]

$$\begin{aligned} \textit{Smallpox mortality}_{it} = \pi_1 \; S_j^{pre\_I} \times I_t^{t>1801} + \pi_2 \; S_j^{pre\_II} \times I_t^{t>1816} + \\ \mathbf{X}_{jt}\bar{\beta} + \bar{\delta}_j + \bar{\tau}_t + \bar{\varepsilon}_{ijt}, \end{aligned} \tag{7}$$

where $\textit{Smallpox mortality}_{it}$ is the smallpox mortality rate in county i at time t. Columns (3) and (4) report the coefficients of estimating this equation. The estimated coefficients of the two interaction terms are positive and highly statistically significant. These results would imply that one could use a three-stage least squares (3SLS) approach, i.e. $S_j^{pre\_I} \times I_t^{t>1801}$ and $S_j^{pre\_II} \times I_t^{t>1816} \Rightarrow \textit{Smallpox mortality}_{it} \Rightarrow \textit{Infant mortality} \Rightarrow y_{ijt}$. However, having established the first chain in this line of argumentation in columns (3) and (4), we follow the literature and regress directly infant mortality on the two interactions, implying that we end up with the suggested 2SLS model.

Columns (5) and (6) report the estimates for child mortality for the age group 1–5, while columns (7) and (8) report the estimates for the total mortality rate. We observe that both are reduced as a consequence of vaccination, but also notice that the observed effects are significantly smaller on the total mortality rate as compared to child and infant mortality. In particular, the estimated coefficient for the total mortality rate (in column 8) implies that a one-standard-deviation increase in the pre-intervention smallpox-mortality rate is associated with a decrease in the death rate of 1.1 deaths per 1000 population afterwards. This number corresponds to 0.09 of a standard deviation in the total death at time $t = 1800$. Thus, consistent with the arguments in the literature, the method of vaccination had most profound effects on infant and child mortality. Finally, the vaccination law does not have the same quantitative effect on these measures, which is arguably related to the fact that compulsory vaccination was for 0–2 year old.

[20] In our 2SLS notation this equation should be referred to as the zero-stage.

Table 7 about here

6.2 The effect of infant mortality on fertility

This section reports the main results of the paper, which are the 2SLS estimates of the effect of infant mortality on four different outcomes: 1) the birth rate (fertility), which is the number of live births per 1000 population, 2) surviving children to the age of one, defined as the birth rate times the infant survival probability, 3) surviving children to the age of five, and 4) natural population growth. The results for these outcomes are shown in Tables 8–11.[21] The estimating equation is (5) and the method of estimation is 2SLS weighted by initial parish population size.

Table 8 shows six different specifications. Columns (1)–(3) show the results for the baseline setup with county and time fixed effects. In addition to fixed effects for periods and counties, column (1) includes only infant mortality instrumented by $S_j^{pre\_I} \times I_t^{t>1801}$. In line with theoretical predictions, the result is a positive coefficient on infant mortality which is significant at the 5 percent level. In terms of magnitude, the coefficient suggests that decreasing the infant mortality rate by 20 deaths per 1000 live births decreases the number of birth by about 1 per 1000 population. Column (2) shows that this estimate is robust to our baseline controls. In column (3), we add the instrument based on the timing of the compulsory vaccination law, and see that this leads to a slightly smaller estimate on infant mortality, but the estimated coefficient remains statistically significant at the 1 percent level.

In terms of instrument quality, the instrumental variables estimation strategy yields a reasonable first-stage fit. The Kleibergen-Paap F-statistic reported in column (3) of Table 8 is 16. A Kleibergen-Paap F-statistic above 10 mitigates the concern that our statistical inference yields misleading results due to the presences of weak instruments (Stock et al., 2002). Moreover, since our 2SLS regressions are overidentified, we can compute the Hansen J-test on the joint hypothesis that our instruments (*Vaccination* and *Law 1816*) are uncorrelated with the second-stage error term. With a p-value of 0.376, the Hansen J-test does not reject the joint hypothesis that the two instrumental variables are uncorrelated with the second-stage error

[21] Note that the p-values in square brackets refer to the Anderson–Rubin test of statistical significance.

term. Given we can assume exogeneity of $S_j^{pre\_I} \times I_t^{t>1801}$, then we cannot reject the null that $S_j^{pre\_II} \times I_t^{t>1816}$ is a valid instrument. Columns (4)–(6) show results for similar, but more demanding specifications where we replaced county fixed effects by parish fixed effects. The coefficients on infant mortality in columns (4) and (6) are similar in magnitude to the ones reported in columns (1) and (3), respectively.[22]

Table 8 about here

Tables 9 and 10 replace the birthrate by measures of surviving children as the outcome variable. Table 9 reports the estimates for the number of children surviving to the age of one, which is constructed as the birth rates times the survival probability. We observe that across specifications, the coefficient on infant mortality is mostly statistically insignificant, which is a result of the fact that the coefficient reduces in magnitude and *not* because it is imprecisely estimated. Thus, a decrease in the number of births, caused by a decrease in the infant mortality rate, does not translate into less surviving children as more children survive. That is, in accordance with the theory of Galor (2011), the extensive effect outweight the intensive effect from Table 6. Table 10 reveals similar conclusions for the number of children surviving to the age of five, which is constructed as the birth rates times the probability of survival to the age of five.

Tables 9 and 10 about here

Finally, Table 11 reports the effects on natural population growth as measured by the birth rate minus the death rate. Consistent with the previous results infant mortality has no effect on population growth. These findings are not due to weak first stages as we see the first-stage Kleibergen-Paap F-statistics is around 7 or above in all the specifications. Moreover, the

[22] In some unreported specifications, we show that our 2SLS estimates are robust to controlling for the number still births, indicating the baseline 2SLS estimates do not capture a direct biological/cultural link from the parents around the infant mortality rate. These estimates are available from the authors upon request.

p-values for the Hansen J-test suggest that we cannot reject the validity of the instruments. Thus, this evidence indicates that fertility adjusts to infant mortality, so that natural population growth is unaffected.

Table 11 about here

To summarize, this section demonstrates, in line with prediction (1) of Section 2 and the theory in Galor (2011), that infant mortality has a positive effect on fertility. In line with Galor's (2011) theory we show that there is no empirical evidence of an effect on surviving children.

7 Conclusion

This paper has demonstrated that infant mortality in Sweden was strongly driven by smallpox vaccination and the associated compulsory vaccination law. Our empirical tests suggest that this cannot be attributed to the initial mortality level, initial population, crude measures of regional income as well as time and cross-sectional (county or parish) fixed effects. We then used the vaccination variables to obtain causal estimates of the effects on fertility and population growth, and demonstrated that infant mortality was unlikely to be a driver of natural population growth in Sweden, which is in line with the theoretical predictions of Galor (2011).

The current study used 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 of the current study. 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 this was one of the cases that provided the blueprints for combating smallpox elsewhere.

Since our study suggests that infant mortality did not impact net fertility, this naturally raises the question of what then drives net fertility? Recent research of Bleakley (2007) and Bleakley and Lange (2009) provide evidence that is consistent with the quantity-quality trade-off theories of fertility transition. They focus on a disease—the hookworm—which relates to morbidity rather than mortality suggesting that the type of disease matters for the fertility

response.[23] These studies also point to the importance of schooling, as also suggested by the dynamic panel analyses by Angeles (2010), Murtin (2013) and Hansen et al. (2014). Yet, none of them provide an estimate of the causal effect of schooling on fertility based on a credible, exogenous source of variation and fixed effects for cross-sectional units. We believe that this is an important task for future research.

[23] See also Andersen, Dalgaard and Selaya (2014) who focus on eye disease.

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Figure 1: Smallpox mortality rate for all of Sweden, 1750–1860

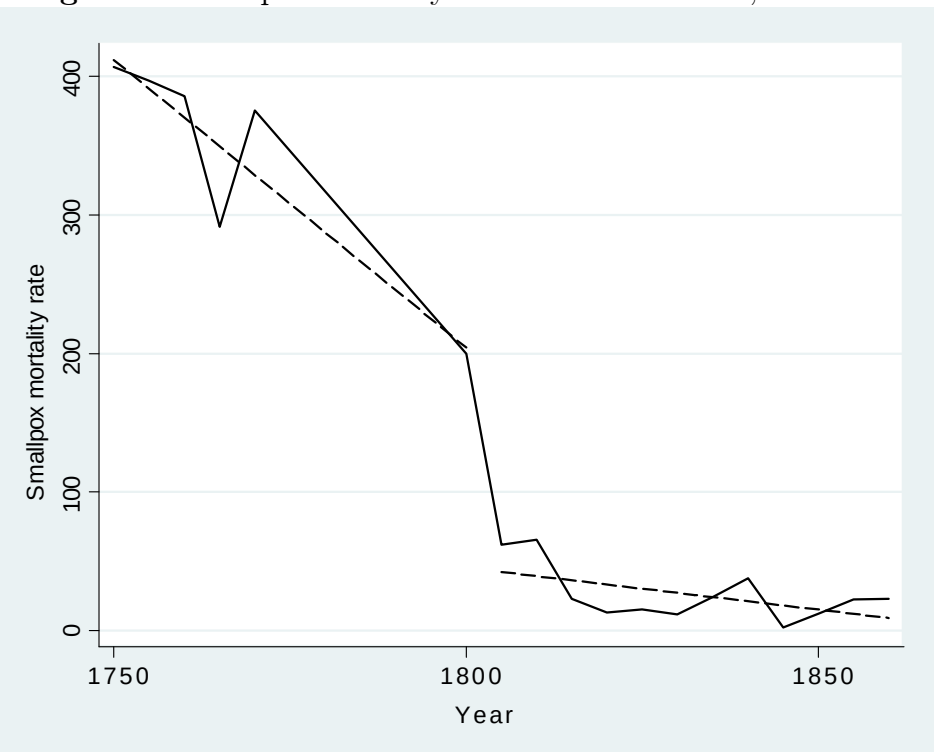

Figure 2: Smallpox share out of total mortality, 1750–1860.

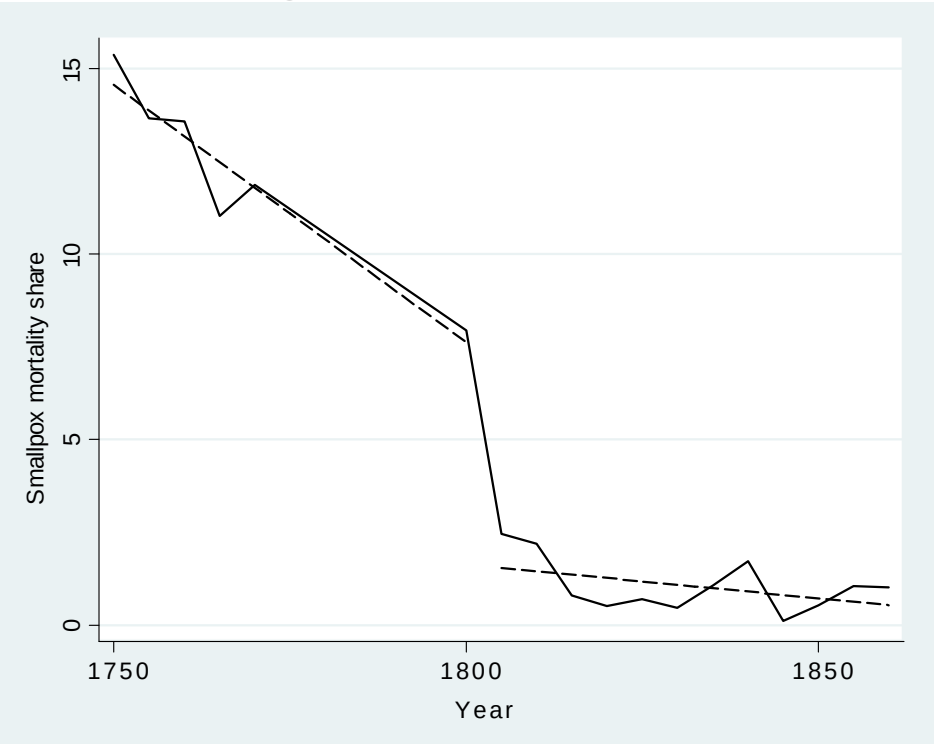

Table 1: Smallpox Mortality per 100,000 by age group

	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: Incidence and mortality

	Cases	Deaths	Deaths per 100
under 1 year	39	14	35.9
1-2 years	145	42	29.0
2-3 years	168	33	19.6
3-4 years	205	34	16.6
4-5 years	186	25	13.4
0-5 years	743	148	19.9
5-10 years	241	48	10.9
10-15 years	58	2	3.4
15-20 years	10	1	10.0

Notes: The table reports the number of incidence and mortality from smallpox during the 1795-1796 epidemic in three towns in Posen. Source: Second report of the Royal Commission Appointed to Inquire into the Subject of Vaccination.

Table 3: 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
	1861-79	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 4: Main results

	Dependent Variable: Infant Mortality Rate				
	(1)	(2)	(3)	(4)	(5)
Vaccination	-0.327***	-0.370***			-0.439***
	(0.121)	(0.117)			(0.118)
Law 1816			-0.248**	-0.228**	-0.543***
			(0.110)	(0.114)	(0.104)
Controls ($\times I^{t>1801}$)**:**					
Initial mortality		0.0538***		0.0471***	0.0540***
		(0.0145)		(0.0174)	(0.0146)
Intial population		3.826		0.940	3.474
		(3.365)		(2.852)	(3.380)
Observations	10,878	10,878	10,878	10,878	10,878

Notes: The left-hand-side variable is infant mortality measured as the number of death per 1000 born at the parish level between 1795–1860. The table reports least squares estimates, weighted by log population size in 1800. All regressions include county and year fixed effects. *Vaacination* is the smallpox mortality rate in 1796–1801 interacted with an 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 an indicator that equals one after 1816. Initial mortality is the infant mortality rate in 1800 and initial population is log population size in 1800. Constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Table 5: Falsification and pre-existing trends

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent Variable:					
	Infant Mortality Rate 1750–1800		Infant Mortality Rate 1795–1860			
Vaccination	0.215	0.219*	-0.350***	-0.418***	-0.546***	-0.553***
	(0.130)	(0.121)	(0.115)	(0.120)	(0.109)	(0.109)
Law 1816		0.0376		-0.508***		-0.448***
		(0.426)		(0.104)		(0.130)
Placebo intensity			0.0901	0.0744		
			(0.0698)	(0.0712)		
Controls ($\times I^{t>1801}$)**:**						
Initial mortality	Yes	Yes	Yes	Yes	Yes	Yes
Intial population	Yes	Yes	Yes	Yes	Yes	Yes
County linear trends	No	No	No	No	Yes	Yes
Observations	7,204	7,204	10,842	10,842	10,842	10,842

Notes: The left-hand-side variable is infant mortality measured as the number of death per 1000 born at the parish level between 1750–1800 in columns (1)-(2) and 1795–1860 in columns (3)-(6). The table reports least squares estimates, weighted by log population size in 1800. All regressions include county and year fixed effects. *Vaacination* is the smallpox mortality rate in 1796-1801 interacted with an 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 an indicator that equals.one after 1816. *Placebo intensity* is smallpox mortality in 1749-1753 interacted with an indicator that equals one after 1801. Initial mortality is the infant mortality rate in 1800 and initial population is log population size in 1800. Constants are not reported. Robust standard errors are clustered at the county level.

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

Table 6: Sensitivity tests

	Dependent Variable: Infant Mortality Rate						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
		Mortality Environment			Economic Environment		
Vaccination		-0.443***		-0.357***	-0.454***	-0.627***	-0.642***
		(0.119)		(0.107)	(0.114)	(0.147)	(0.141)
Vaccination	-9.859***		-10.11***				
(alternative)	(2.282)		(1.940)				
Law 1816	-0.357***			-0.579***	-0.510***	-0.501***	-0.471***
	(0.0961)			(0.146)	(0.101)	(0.126)	(0.124)
Law 1816		-16.48***	-14.92***				
(alternative)		(2.865)	(2.844)				
Still births				0.201***			
				(0.0536)			
Price rye					3.600*		3.156
					(1.928)		(2.003)
Log rye/capita						-2.973	-2.880
						(8.331)	(7.957)
County fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Parish fixed effects	No	No	No	No	No	No	No
County linear trends	No	No	No	No	No	No	No
Observations	10,878	10,878	10,878	6,697	10,878	9,490	9,490

Notes: The left-hand-side variable is infant mortality measured as the number of death per 1000 born at the parish level between 1795–1860. The table reports least squares estimates, weighted by log population size in 1800. All regressions include year fixed effects, initial mortality, and initial population size. *Vaccination* is the smallpox mortality rate in 1796-1801 interacted with an indicator that equals one after 1801. *Vaccination (alternative)* uses the share of smallpox mortality out of total mortality as the intensity measure. *Law 1816* is constructed as smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with an indicator that equals one after 1816. *Law 1816 (alternative)* uses the share of smallpox mortality out of total mortality at the intensity measure. Initial mortality is the infant mortality rate in 1800, initial population is log population size in 1800, *still births* is the number of still births per 1000 births, *price rye* is the price on rye at county level and *log rye/capita* is the natural logarithm of the production of rye per capita at the county level. Constants are not reported. Robust standard errors are clustered at the county level.

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

Table 7: Alternative outcomes

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Intention-to-treat design				Mortality			
	Vaccination Rate		*Smallpox Mortality*		*Child Mortality*		*Death Rate*	
Vaccination	0.0648**	0.0651**	-1.165***	-1.164***	-0.366***	-0.314***	-0.0164*	-0.0200**
	(0.0259)	(0.0254)	(0.0298)	(0.0297)	(0.0941)	(0.101)	(0.00856)	(0.00892)
Law 1816	0.220**	0.219**	-0.699***	-0.699***	0.702*	0.671*	-0.00766	-0.00646
	(0.104)	(0.104)	(0.150)	(0.149)	(0.402)	(0.405)	(0.0287)	(0.0299)
Controls ($\times I^{t>1801}$)**:**								
Initial mortality	No	Yes	No	Yes	No	Yes	No	Yes
Intial population	No	Yes	No	Yes	No	Yes	No	Yes
Observations	10,314	10,314	10,101	10,101	7,627	7,627	10,877	10,877

Notes: The left-hand-side variable is the vaccination rate in columns (1)-(2), smallpox mortality in columns (3)-(4), child mortality in columns (5)-(6) and the death rate in columns (7)-(8) at the parish level between 1795–1860 (in columns (3)-(4) the observation period is 1800–1860). The table reports least squares estimates, weighted by log population size in 1800. All regressions include county and year fixed effects. *Vaccination* is the smallpox mortality rate in 1796-1801 interacted with an indicator that equals one after 1801. *Law 1816* is constructed as smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with an indicator that equals one after 1816. In columns (1)-(4), initial mortality refers to the infant mortality rate in 1800, while in columns (5)-(6) initial mortality refers to child mortality in 1800 and in columns (7)-(8) to the death rate in 1800. Initial population is log population size in 1800. Constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Table 8: The effect on fertility

	Dependent Variable: Birth rate					
	(1)	(2)	(3)	(4)	(5)	(6)
Infant mortality	0.0542***	0.0533***	0.0421***	0.0542***	0.166***	0.0527*
	(0.0205)	(0.0176)	(0.0141)	(0.0205)	(0.0529)	(0.0308)
Anderson-Rubin [p-value]	[0.011]	[0.004]	[0.008]	[0.011]	[0.002]	[0.006]
Controls ($\times I^{t>1801}$)**:**						
Initial mortality	No	Yes	Yes	No	Yes	Yes
Intial population	No	Yes	Yes	No	Yes	Yes
Instruments:						
Vaccination	Yes	Yes	Yes	Yes	Yes	Yes
Law 1816	No	No	Yes	No	No	Yes
Kleibergen-Paap						
F-statistic	6.95	9.47	16.00	6.97	7.65	12.64
Hansen-J [p-value]	-	-	[0.376]	-	-	[0.066]
County fixed effects	Yes	Yes	Yes	No	No	No
Parish fixed effects	No	No	No	Yes	Yes	Yes
Obsservations	10,878	10,878	10,878	10,878	10,878	10,878

Notes: The left-hand-side variable is the birth rate measured as the number of live births per 1000 populations at the parish level between 1795–1860. The table reports two-stage least squares estimates for *infant mortality*, weighted by log population size in 1800. All regressions include year fixed effects. *Infant mortality* is the number of death per 1000 born. *Vaccination* is the smallpox mortality rate in 1796-1801 interacted with an 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 an indicator that equals one after 1816. Initial mortality is the infant mortality rate in 1800 and initial population is log population size in 1800. Constants are not reported. Robust standard errors are clustered at the county level.

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

Table 9: The effect on surviving children to the age of one

	Dependent Variable: Surviving Children (age 1)					
	(1)	(2)	(3)	(4)	(5)	(6)
Infant mortality	0.0187	0.0172	0.00646	0.0187	0.108**	0.0117
	(0.0176)	(0.0150)	(0.0117)	(0.0176)	(0.0460)	(0.0258)
Anderson-Rubin [p-value]	[0.229]	[0.202]	[0.394]	[0.229]	[0.006]	[0.016]
Controls ($\times I^{t>1801}$)**:**						
Initial mortality	No	Yes	Yes	No	Yes	Yes
Intial population	No	Yes	Yes	No	Yes	Yes
Instruments:						
Vaccination	Yes	Yes	Yes	Yes	Yes	Yes
Law 1816	No	No	Yes	No	No	Yes
Kleibergen-Paap						
F-statistic	6.95	9.47	16.00	6.97	7.65	12.64
Hansen-J [p-value]	-	-	[0.330]	-	-	[0.069]
County fixed effects	Yes	Yes	Yes	No	No	No
Parish fixed effects	No	No	No	Yes	Yes	Yes
Obsservations	10,878	10,878	10,878	10,878	10,878	10,878

Notes: The left-hand-side variable is surviving children age 1, which is constructed as the birth rate time the probability of surviving to the age of one at the parish level between 1795–1860. The table reports two-stage least squares estimates for *Infant mortality*, weighted by log population size in 1800. All regressions include year fixed effects. *Infant mortality* is the number of death per 1000 born. *Vaccination* is the smallpox mortality rate in 1796-1801 interacted with an 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 an indicator that equals one after 1816. Initial mortality is the infant mortality rate in 1800 and initial population is log population size in 1800. Constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Table 10: The effect on surviving children to the age of five

	Dependent Variable: Surviving Children (age 5)					
	(1)	(2)	(3)	(4)	(5)	(6)
Infant mortality	-0.00775	-0.00295	0.000354	-0.00798	0.0467	0.0224
	(0.0144)	(0.0132)	(0.0140)	(0.0157)	(0.0370)	(0.0375)
Anderson-Rubin [p-value]	[0.601]	[0.826]	[0.960]	[0.617]	[0.220]	[0.452]
Controls ($\times I^{t>1801}$)**:**						
Initial mortality	No	Yes	Yes	No	Yes	Yes
Intial population	No	Yes	Yes	No	Yes	Yes
Instruments:						
Vaccination	Yes	Yes	Yes	Yes	Yes	Yes
Law 1816	No	No	Yes	No	No	Yes
Kleibergen-Paap						
F-statistic	7.86	10.07	13.44	7.92	14.07	14.23
Hansen J [p-value]	-	-	[0.795]	-	-	[0.618]
County fixed effects	Yes	Yes	Yes	No	No	No
Parish fixed effects	No	No	No	Yes	Yes	Yes
Obsservations	9,015	9,015	9,015	9,015	9,015	9,015

Notes: The left-hand-side variable is surviving children age 5, which is constructed as the birth rate time the probability of surviving to the age of five at the parish level between 1795–1860. The table reports two-stage least squares estimates for *Infant mortality*, weighted by log population size in 1800. All regressions include year fixed effects. *Infant mortality* is the number of death per 1000 born. *Vaccination* is the smallpox mortality rate in 1796-1801 interacted with an 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 an indicator that equals one after 1816. Initial mortality is the infant mortality rate in 1800 and initial population is log population size in 1800. Constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Table 11: The effect on natural population growth

	Dependent Variable: Natural Population Growth					
	(1)	(2)	(3)	(4)	(5)	(6)
Infant mortality	0.00717	0.000587	-0.000308	0.00717	0.0807	0.0229
	(0.0252)	(0.0194)	(0.0209)	(0.0252)	(0.0582)	(0.0572)
Anderson-Rubin [p-value]	[0.764]	[0.976]	[0.999]	[0.764]	[0.097]	[0.244]
Controls ($\times I^{t>1801}$)**:**						
Initial mortality	No	Yes	Yes	No	Yes	Yes
Intial population	No	Yes	Yes	No	Yes	Yes
Instruments:						
Vaccination	Yes	Yes	Yes	Yes	Yes	Yes
Law 1816	No	No	Yes	No	No	Yes
Kleibergen-Paap						
F-statistic	6.95	9.47	15.97	6.97	7.65	12.62
Hansen-J [p-value]	-	-	0.966	-	-	0.442
County fixed effects	Yes	Yes	Yes	No	No	No
Parish fixed effects	No	No	No	Yes	Yes	Yes
Obsservations	10,877	10,877	10,877	10,877	10,877	10,877

Notes: The left-hand-side variable is natural population growth, constructed as the birth rate minus the death rate at the parish level between 1795–1860. The table reports two-stage least squares estimates for *Infant mortality*, weighted by log population size in 1800. All regressions include year fixed effects. *Infant mortality* is the number of death per 1000 born. *Vaccination* is the smallpox mortality rate in 1796-1801 interacted with an 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 an indicator that equals one after 1816. Initial mortality is the infant mortality rate in 1800 and initial population is log population size in 1800. Constants are not reported. Robust standard errors are clustered at the county level.

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