Economic History Working Papers No: 340

Did Smallpox Cause Stillbirths? Maternal Smallpox Infection, Vaccination and Stillbirths in Sweden, 1780-1839

Eric B. Schneider, LSE & CEPR
Sören Edvinsson, Umeå University
Kota Ogasawara, Tokyo Institute of Technology

May 2022

Economic History Department, London School of Economics and Political Science,
Houghton Street, London, WC2A 2AE, London, UK. T: +44 (0) 20 7955 7084.

DID SMALLPOX CAUSE STILLBIRTHS? MATERNAL SMALLPOX INFECTION, VACCINATION AND STILLBIRTHS IN SWEDEN, 1780-1839

Eric B. Schneider (LSE and CEPR),
Sören Edvinsson (Umeå University),
and Kota Ogasawara (Tokyo Institute of Technology)[†]

3 May 2022

Abstract

Woods (2009) argued that smallpox was an important cause of stillbirths in the past. While there is strong evidence that maternal smallpox infection could lead to fetal loss, it is not clear whether smallpox infections were a demographically important source of stillbirths. In this paper, we use parish-level data from the Swedish Tabellverket dataset from 1780 to 1839 to test the effect of smallpox on stillbirths quantitatively. We use two empirical strategies: dynamic panel regressions that test the instantaneous effect of smallpox epidemics on stillbirths; and a continuous treatment difference-in-difference strategy to test whether the reduction in smallpox prevalence following vaccination led to a larger decrease in the stillbirth rate in parishes where smallpox was more prevalent before vaccination. We find very little evidence that smallpox infection was a major cause of stillbirths in history. Our coefficients are largely insignificant and close to zero. This is because the vast majority of women contracted smallpox as children and therefore were no longer susceptible during pregnancy. We do find a small, statistically significant effect of smallpox on stillbirths from 1820-39 when waning immunity from vaccination put a greater share of pregnant women at risk of contracting smallpox. However, the reduced prevalence of smallpox limited the demographic impact. Thus, smallpox was not an important driver in historical stillbirth trends and did not contribute to *in utero* scarring effects for cohorts born when smallpox prevalence was high.

JEL Codes: J13, N34

Keywords: stillbirth, fetal death, smallpox, vaccination, historical demography

[†] Corresponding author: Eric Schneider (e.b.schneider@lse.ac.uk). The authors wish to thank the team at the Umeå Demographic Data Base for retrieving specialised data for us. We also thank Romola Davenport, Ingrid van Dyke, Luciana Quaranta and conference participants at the Fourth European Society for Historical Demography Conference in Madrid for helpful discussion and comments on the paper. We thank Anne Løkke and Frans van Poppel for sharing their long-run stillbirth rate time series for Denmark and Zeeland respectively and Juliana Jaramillo Echeverri for excellent research assistance. All remaining errors are our own.

Exposure to infectious diseases during pregnancy can lead to a number of adverse birth outcomes. The recent outbreak of Zika virus which caused substantial cases of microcephaly raised this issue once again, but a number of infectious diseases including rubella, chickenpox and smallpox are known to lead to congenital malformations and fetal loss (Silasi et al. 2015; Racicot and Mor 2017; Nishiura 2006). In addition, a growing literature shows that exposure to infectious disease *in utero* leads to poorer health in later life and human capital outcomes. For instance, cohorts exposed to the 1918 flu pandemic *in utero* had lower educational attainment and income and also experienced slower growth (Almond 2006; Mazumder et al. 2010; Ogasawara 2017), though some of these results have been disputed recently (Beach et al. 2022; Helgertz and Bengtsson 2019). Given the importance of infectious disease for health across the life course, the eradication of infectious disease in the nineteenth and twentieth centuries may have caused significant improvements in fetal health and cohort health more generally.

This paper explores the consequences of smallpox, a particularly virulent and prevalent disease in history, for stillbirths using parish-level data from Sweden in the eighteenth and nineteenth centuries. Woods first argued that maternal smallpox infections were an important source of stillbirths in the eighteenth and nineteenth centuries using a mixture of evidence from medical case books and back-of-the-envelope estimations (Woods 2009, p. 231-32). Given the decline in smallpox mortality across the nineteenth century due to vaccination, if smallpox were a major driver of stillbirth rates, then the changing epidemiology of smallpox may have had an important effect on trends in stillbirth rates over time. There is strong historical evidence from the nineteenth and twentieth centuries that smallpox could lead to fetal deaths (Nishiura 2006; Woods 2009, pp. 219-21), but these studies tended to be drawn from smallpox epidemics long after vaccination was introduced. To date, no one has been able to quantify how important

smallpox infections in pregnancy were as a cause of stillbirths before, during and after vaccination.

The importance of smallpox for stillbirths is dependent on two factors: the share of women of childbearing age who were susceptible to smallpox and the overall prevalence of smallpox in the population. We can see these factors at play in the eighteenth and nineteenth centuries. In the North of England and Sweden before vaccination, the vast majority of smallpox deaths occurred among children, indicating that most adults would have been immune (Sköld 1996b). Thus, although smallpox prevalence was high, smallpox infections in pregnancy may have been rare. However, Woods argued based on a small case study from eighteenth-century England that adult smallpox mortality was more common (Woods 2009, p. 224), and a more recent and extensive study of smallpox in England found that the majority of smallpox deaths in southern England occurred among adults (Davenport et al. 2018). This age pattern was different because inoculation and isolation of cases in pesthouses helped to reduce smallpox prevalence. Similarly, after vaccination, smallpox prevalence fell dramatically, but because immunity from vaccination diminished with time, the prevalence of smallpox among adults likely increased as suggested by the tenfold increase in the smallpox age-specific mortality rate for adults aged 25-49 between 1790 and 1850 in Sweden (Sköld 1996b, pp. 579, 588). Thus, after vaccination, there were women of childbearing age who were susceptible to smallpox and who could have been at risk of smallpox-related stillbirths. However, after vaccination, the overall prevalence of smallpox was far lower, perhaps counteracting the increased susceptibility of pregnant women.

This paper tests the net impact of these factors using parish-level data from Sweden between 1780 and 1839 where smallpox deaths and stillbirths were registered from the eighteenth century onward. We analyse a panel dataset of 622 parishes with consistent boundaries from the Tabellverket Database constructed by the Umeå Demographic Data Base

team. We use several empirical strategies to test whether the effect of smallpox on stillbirths changed before and after vaccination and to isolate exogenous variation in smallpox prevalence. Section I presents historical background on the causes of and trends in stillbirths, the changes in smallpox epidemiology over time and existing theories of how smallpox might have caused stillbirths. Section II presents the Tabellverket dataset, section III presents our two empirical strategies for estimating the causal effect of smallpox on stillbirths. Section IV presents the results and section V extends these Swedish results to other time periods and places. Finally section VI concludes.

I. Background

A. Historical trends in and causes of stillbirths

Compiling long-run data on stillbirth rates is a particular challenge because stillbirths were often not registered the same way as other vital events and because definitions of stillbirths changed over time and between countries. Scandinavian countries and Zeeland, a province of the Netherlands, were the first to register stillbirths. Sweden and Norway seem to have had good registration practices from the early days (Woods 2009, pp. 56-7; Sommerseth 2021), but in Denmark, neonatal deaths in the first 24 hours were considered stillbirths until 1861, after which they were meant to be excluded (Løkke 2018b). In Zeeland, stillbirths were denoted as children who died before registration, which was required to occur within three days of birth. Thus, it is likely that very early neonatal deaths were also included in the Zeeland series (van Poppel 2018). Catholic countries tended to have relatively few stillbirths registered as families sought to baptise stillborn children, making stillbirth registration for countries like France, Spain and Italy unreliable until the twentieth century (Woods 2009, pp. 77-82). More recently, changes in the age threshold between miscarriages and stillbirths have affected data series, although these age differences were not an important bias in stillbirths historically.

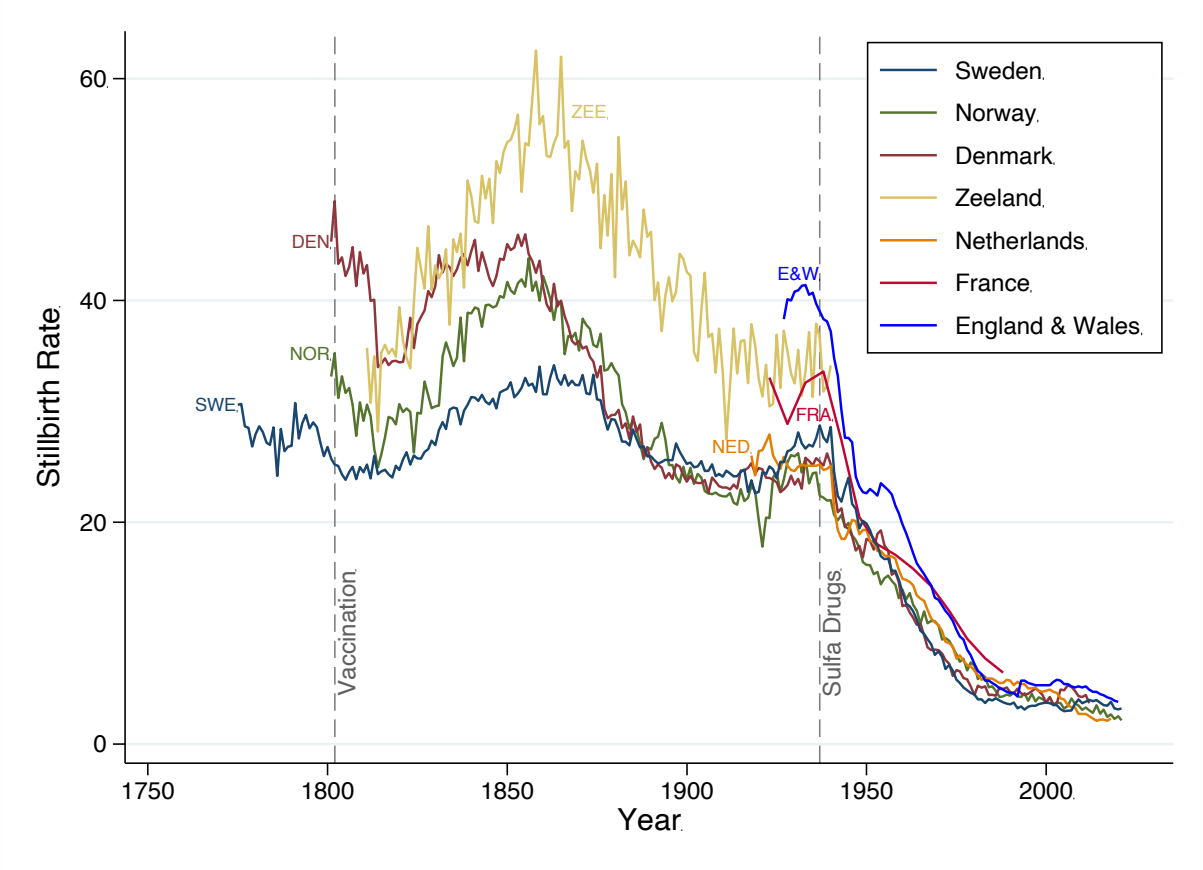
Woods et al. (2006) presented intriguing trends in late-fetal mortality for four Northern European countries/province (Sweden, Denmark, Norway and Zeeland) across the nineteenth and twentieth centuries (reproduced and updated in Figure 1). Series for England and Wales, France and the Netherlands are included from when stillbirth registration became reasonably comprehensive and accurate. Woods (2009, p. 59) argued that the rise in stillbirths from the early nineteenth to mid-nineteenth century across all countries may have been partly due to improving registration practices, and the subsequent decline was in part driven by the removal of neonatal deaths in the first 24 hours from the series.[1] However, the uniformity of the patterns across all contexts and its presence in Sweden and Norway, which did not record early neonatal deaths as stillbirths, suggests that registration practices alone were not driving the pattern.

Explanations for the nineteenth century trends are somewhat limited, especially when considering the increasing stillbirth rate in the first half of the nineteenth century. This could be related to smallpox, which will be discussed below, but it also might be the consequence of improving obstetric care. Løkke (2018a) argues that the successful implementation of invasive intrapartum procedures that prevented children from being stuck in the uterus led to fewer maternal deaths where the child was undelivered. The dissemination of these techniques in the early nineteenth century may explain some of the increase in stillbirth rates across countries as children who had previously not been registered at all were added to the ranks of stillbirths. The decline in stillbirth rates in the second half of the nineteenth century has been attributed to further dissemination of best practice maternal care and especially the use of antiseptics from the 1870s onward (Løkke 2018a; Högberg and Wall 1986; Högberg 2004; Woods *et al.* 2006).

Stillbirth rates were relatively stable from the 1880s until the late 1930s when all countries experienced sharp declines. Woods ascribed this to sulfa drugs, antibiotics and better

[1] Løkke (2018b, p. 91) shows that the removal of neonatal deaths in the first 24 hours of life does not explain away the decline in stillbirths in Denmark.

Figure 1: Stillbirth rates in the long run in selected countries



Sources: European Stillbirth Rate Time Series Dataset (2022). Adapted from Woods et al. (2006). Sweden: Statistics Sweden (2022); Norway: Statistics Norway (2022); Denmark: Anne Løkke, personal communication (2022); Zeeland: Frans van Poppel, personal communication (2022); Netherlands: Statistics Netherlands (2022); France: Macfarlane et al. (2000), pp. 664-65; England and Wales: ONS (2022).

quality of maternal care but did not understand the precise mechanism through which these medical innovations would have affected stillbirths (Woods 2009, pp. 82-85). Løkke (2012) provided a potential explanation for the effects of sulfa drugs and antibiotics on stillbirths by studying childbirth cases in the National Hospital in Copenhagen: once doctors could treat puerperal fever with these drugs, they were much more likely to perform invasive surgeries to save the child when there were intrapartum complications. These interventions along with a decline in incidence of maternal syphilis explained a large share of the decline in the stillbirth rate after 1940 (Schneider 2017).

The existing explanations for changing stillbirth rates over time tend to emphasise the importance of obstetric care foremost with underlying maternal and fetal health being

secondary causes of changes in stillbirths. Thus, more research is needed on other factors such as disease that might influence stillbirths in history.

B. Smallpox in Sweden

Smallpox was a leading cause of death in eighteenth-century Sweden and was responsible for 8-14 per cent of total deaths. Mortality in the eighteenth century was concentrated among children (Table 1), and there were severe epidemics at least once per decade killing thousands of people (Figure 3). There was spatial variation in these epidemics, which recurred at the local level every 3 to 25 years except in Stockholm where smallpox was endemic (Sköld 1996a, pp. 248-49). Although inoculation was growing in popularity in eighteenth-century England and seemingly affecting the epidemiology of smallpox there (Davenport et al. 2018), Sköld (1996a) argues that inoculation was largely ineffective in Sweden because it was costly, there was concern about the health risks and a lack of confidence in the procedure, and the Department of Health monopolized inoculation preventing its diffusion. Thus, smallpox mortality rates remained high in Sweden throughout the eighteenth century.

The situation changed with the introduction of the smallpox vaccine, published by Jenner in 1798 (Crosby 1993). It took a few years for vaccination to be practiced in Sweden with the first vaccination being administered in late November 1801 (Sköld 1996b, p. 371). Vaccination uptake increased very rapidly from 0 to 60 per cent between 1800 and 1820 though with substantial regional variation. Vaccination was targeted at young children, and it became compulsory in 1816 (Sköld 1996a). It led to a sharp reduction in smallpox mortality (see Figure 3 below) with smallpox never again reaching its eighteenth century zenith despite a resurgence in the mid-nineteenth century (Sköld 1996a, Ager et al. 2018).

Table 1: Period age-specific smallpox mortality rates per 100,000

	Age-specific smallpox mortality rate per 100,000						
Period	0-1	1-2	3-4	5-9	10-24	25-49	over 50
1788-1792	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

Source: Sköld (1996b, pp. 579-88) and Ager et al. (2018).

In the early days of vaccination, there was little understanding of waning immunity over time. Swedish doctors expected vaccination to provide the same life-long immunity as a previous infection (Sköld 1996b, pp. 480-82). However, with time it became clear that the vaccine provided only limited immunity, and there was a resurgence of smallpox beginning in the 1820s. The Swedish Medical Board eventually recognised the need for revaccination and allowed physicians to revaccinate individuals beginning in 1839. However, aside from Swedish military recruits, revaccination was never a requirement nor widespread in Sweden. Instead, revaccination was practiced during smallpox epidemics to contain the spread of the disease (Sköld 1996b, pp. 482-84). Vaccination and waning immunity from vaccination changed the age pattern of smallpox mortality (Table 1). Age-specific mortality rates at young ages dropped dramatically after vaccination as fewer young children contracted the disease. However, the age-specific mortality rates of individuals above the age of 25 increased because having escaped smallpox in childhood through vaccination, adults in that age group were now susceptible to smallpox at higher rates than they had been in the eighteenth century. The changes in smallpox epidemiology in eighteenth- and nineteenth-century Sweden present fertile ground for testing the effect of smallpox on stillbirths.

C. Smallpox and stillbirths

There are three mechanisms through which smallpox infection could have affected stillbirths in the eighteenth and nineteenth centuries in Sweden. The first mechanism is perhaps the most straightforward. If smallpox were present in a parish, and a susceptible pregnant woman contracted smallpox, then she would be at risk of stillbirth from the smallpox infection. There is ample historical evidence that smallpox could be passed from the mother to the fetus and that this could lead to miscarriage and stillbirth (Nishiura 2006; Woods 2009, pp. 218-23). A meta-analysis estimate of fetal death rates among women who contracted smallpox in pregnancy was 39.9 per cent (Nishiura 2006), suggesting that this direct mechanism could lead to substantial fetal losses among pregnant women infected.

Smallpox could have also caused stillbirths directly if subclinical smallpox infections were possible among women who had already contracted smallpox. To be clear, there is no medical evidence that this was possible. The modern literature on smallpox is quite clear that smallpox infection granted life-long immunity and that smallpox virus did not persist in the body after infection (Breman and Henderson 2002; Fenner et al. 1988, pp. 144-47; Petersen et al. 2014). However, we do not rule out this possibility because the smallpox virus has evolved: a more lethal strain of smallpox emerged globally in the sixteenth century (Harper 2021, pp. 362-3; Carmichael and Silverstein 1987) and a less lethal form, variola minor, appeared in the late nineteenth century (Fenner et al. 1988, pp. 242-43).

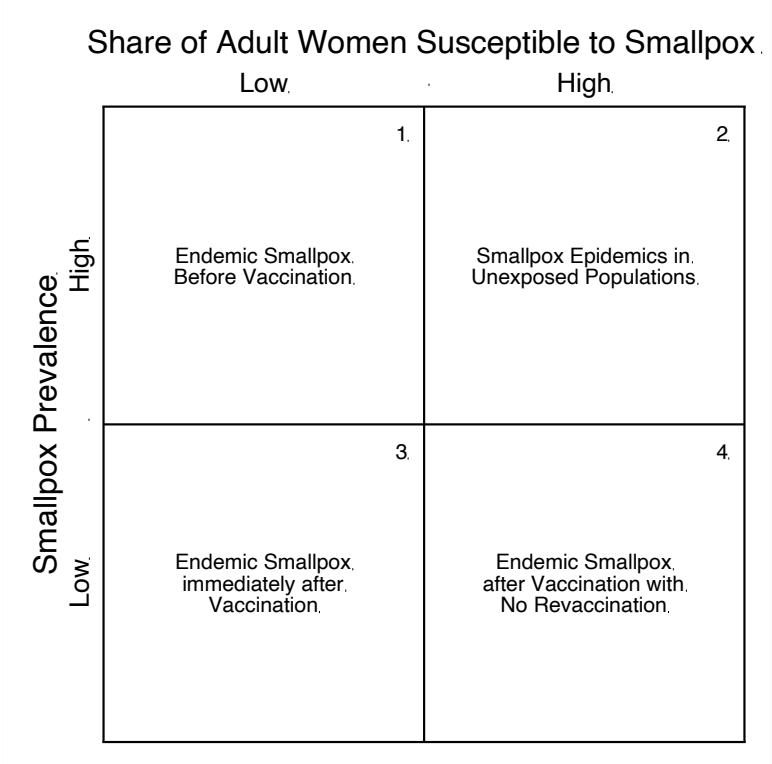
The final mechanism through which smallpox epidemics could affect stillbirth rates is an indirect mechanism: smallpox epidemics could disrupt economic and social systems and increase stress for pregnant women, increasing the likelihood of stillbirths. Discussing the smallpox epidemics in the Americas following the Colombian Exchange, Jones (2003) argues the large share of populations sick during epidemics would have prevented normal economic activities like harvesting, planting and trade from being conducted and could have had large consequences for mortality from causes unrelated to the epidemics. However, there is reason

to doubt that this disruption would have been similar in Sweden given that smallpox epidemics were relatively frequent and familiar occurrences and they mainly led to morbidity and mortality among children who were not as critical to economic activity. Still, a smallpox epidemic may have increased stress levels for pregnant women, and stress has been shown to increase the risk of stillbirths (Wisborg et al. 2008).

The importance of these mechanisms depends on the two factors highlighted in the introduction: the share of pregnant women who were susceptible to smallpox and smallpox prevalence. We can combine these two factors in a simple two-by-two matrix to understand the different equilibria that would have been present at different points in Swedish history (see Figure 2). In equilibrium one, smallpox prevalence is high because there is little to no control of smallpox and therefore most people contract smallpox as children leaving very few pregnant women susceptible. This equilibrium reflects the situation in Sweden in the eighteenth century before vaccination. As shown above, smallpox mortality in Sweden was highly concentrated among children before vaccination, so it is unclear whether enough pregnant women were still susceptible to smallpox infections during pregnancy to matter. However, given the high mortality rate of fetuses exposed to smallpox, if even a small proportion of women were still susceptible to smallpox during pregnancy, there could be a substantial impact on stillbirths. Likewise, if subclinical infections were possible, then we would expect to see an effect in this period. Finally, if smallpox disruption contributed to stillbirths through a stress channel, then we would expect that this indirect path might matter most when smallpox prevalence was high.

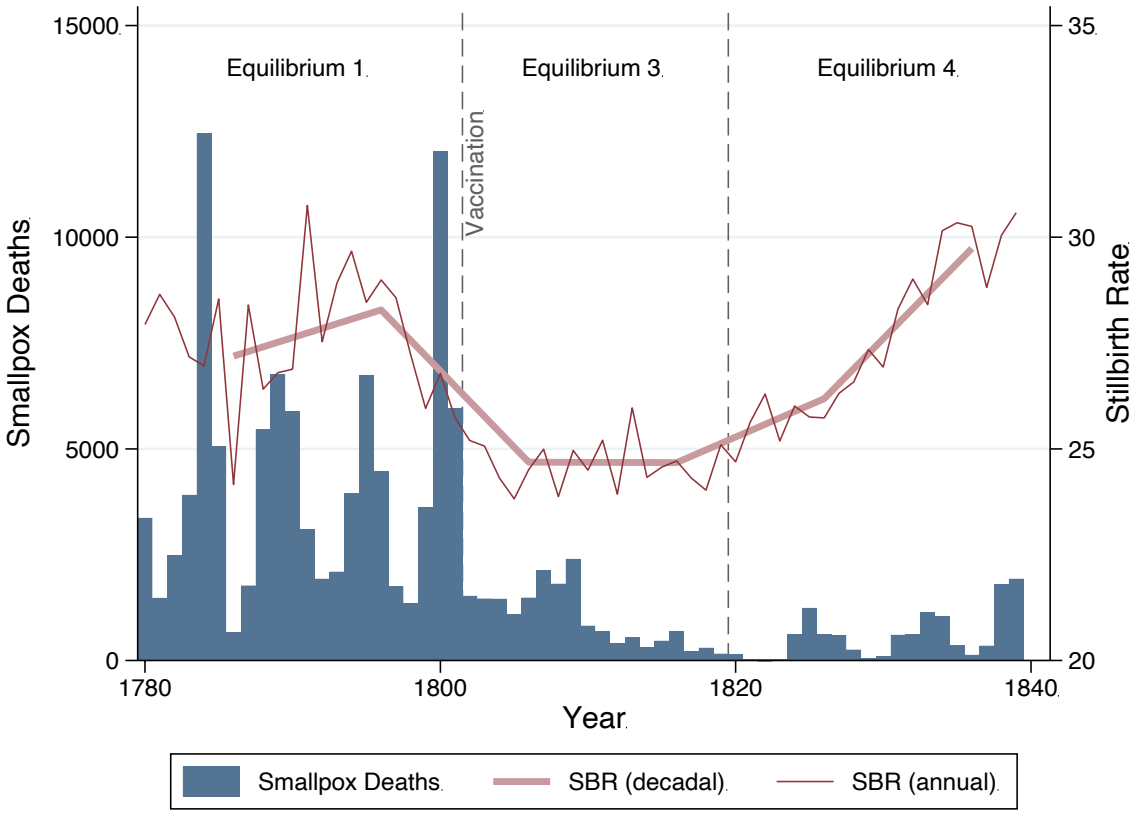
Immediately after vaccination, Sweden would have moved to equilibrium three. Smallpox prevalence would have fallen substantially but most adult women would still have had immunity from smallpox based on infections in childhood. Given that the share of women susceptible to smallpox was low, we might again expect to find a small effect in this period, but the first two mechanisms should still mattered even if smallpox prevalence was lower.

Figure 2: Equilibria of smallpox and its effect on stillbirths



Equilibrium four arises from the changing nature of smallpox epidemiology following vaccination. Because of the waning immunity from vaccination, it is possible that a greater share of pregnant women were susceptible to smallpox in the mid-nineteenth century than had been in the eighteenth century as reflected through age-specific mortality rates (see Table 1). Evidence from the 1878 smallpox epidemic in Philadelphia shows that both vaccinated and unvaccinated pregnant women contracted smallpox and experienced fetal deaths (Nishiura 2006; Welch 1878). Thus, it is possible that smallpox mortality might have been more important in driving stillbirth rates after vaccination than when smallpox was far more prevalent and deadly in the eighteenth century. Revaccination would have blunted this effect somewhat, but the focus of revaccination on areas already experiencing smallpox outbreaks may have limited the effectiveness of revaccination for preventing smallpox among pregnant women.

Figure 3: Smallpox deaths and stillbirth rates in Sweden from 1780 to 1839



Sources: Smallpox deaths from Sköld (1996b, pp. 52); SBR (decadal) from Historisk Statistik för Sverige (1967, pp. 108-109); SBR (annual) from Statistics Sweden (2022).

We do not observe equilibrium two, high smallpox prevalence and high susceptibility among pregnant women, since smallpox was endemic in Sweden during this time period, but we will discuss how our findings can be extended to this equilibrium in the discussion.

Keeping the mechanisms and equilibria in mind, we can reinterpret the national time series evidence for stillbirths in Figure 3 in relation to the prevalence of smallpox and the equilibria presented above. The sharp decline in stillbirth rates at the beginning of the nineteenth century seems to suggest that the introduction of vaccination and the decline in smallpox prevalence may have reduced stillbirths accordingly.[2] Thus, perhaps smallpox was

[2] This is especially clear when looking at the decadal stillbirth rates, which were the only data available before this study: the annual data shows that that the stillbirth rate was declining before vaccination.

virulent enough to fetuses that even a few cases among pregnant women were contributing to the stillbirth rate. These lower stillbirth rates persisted for twenty years, but beginning in the 1820s, stillbirths began to increase again. This increase in stillbirths could have been caused by a greater share of women becoming susceptible to smallpox in pregnancy even though smallpox prevalence was far lower in the mid nineteenth century. Testing whether these trends in the national time series were driven by the epidemiology of smallpox is the key objective of this paper.

II. Data

We use a panel of Swedish parishes from 1780 to 1839 to test the effect of smallpox on stillbirths. The data is drawn from the SHIPs database, which itself is based on the Tabellverk records kept by Swedish clergy and reported to the state during the eighteenth and nineteenth centuries (Tabellverket Database 2015). Swedish clergy kept meticulous records of births, marriages, deaths and migration in their parish and reported their figures to the Swedish state at regular intervals (Sköld 2004; Jeub 1993). They also recorded stillbirths and deaths from smallpox in each parish and year. As noted above, changes in stillbirth registration practices were not as pronounced in Sweden as they were in other parts of Scandinavia. Still, there are likely to be inconsistencies in registration over time even if these are not as clearly spelled out for Sweden as for other countries.[3] The most important registration issue we faced was a temporary change between 1802 and 1821 from reporting the number of stillbirths to the number of women experiencing stillbirths. This is discussed at length in Online Appendix A, and does not seem to produce major error in our data.

[3] Note that all regressions include year fixed effects to capture unobserved changes in registration patterns over time.

In analysing smallpox and stillbirths, we use 622 parishes, which form a balanced panel between 1780 and 1839. These are parishes that are linked explicitly in the SHIPs database, suggesting that there were no or only very minor border changes over time. However, this is a subset of all parishes: there are around 1,900 in total in our period. To judge whether our balanced panel is representative of all parishes, Figure A.7 in the web appendix compares parishes included and excluded from our balanced panel on the stillbirth rate, smallpox mortality rate, maternal mortality rate, infant mortality rate, population and births. The balanced and unbalanced parishes are remarkably similar in both level and trend for stillbirth, smallpox mortality, maternal mortality and infant mortality rates, but the balanced panel parishes were larger on average by about two or three hundred people, and consequently there were more births in the balanced panel parishes each year as well. While we prefer the balanced panel for econometric rigor, we have also reproduced our empirical results using the whole unbalanced Tabellverket data and the interpretation of the results is the same (reported in Appendix B). Table 2 presents descriptive statistics of our key variables of interest for the three periods/equilibria in our data.

Table 2: Descriptive statistics of key variables by time period

	1780-1801		1802-1819		1820-1839	
	Mean	SD	Mean	SD	Mean	SD
Stillbirth Rate per 1000 Total Births	31.2	41.0	27.3	42.0	30.7	35.3
Smallpox Mortality Rate per 1000	1.81	3.88	0.42	1.54	0.17	0.75
Parish-years with Smallpox Deaths (binary)	0.35		0.15		0.10	
Number of parishes	622		622		622	
Number of years	22		18		20	
Number of Observations	13684		11196		12440	

Sources: Tabellverket dataset.

III. Methods

A. Dynamic panel regressions

We employ two empirical strategies to test the mechanisms presented above. First, we use dynamic panel regressions to test whether the presence or severity of smallpox in a parish leads to an instantaneous increase in stillbirth rates. We estimate the following model using OLS regression:

$$SBR_{i,t} = \alpha + \beta SBR_{i,t-1} + \gamma Pox_{i,t} + \rho_i + \phi_t + \varepsilon_{i,t} \quad (1)$$

where the dependent variable ($SBR_{i,t}$) is the stillbirth rate in parish i in year t. The main independent variable of interest ($Pox_{i,t}$) takes two forms: either the smallpox mortality rate in parish i in year t or a binary indicator variable equal to one in years in which any smallpox deaths are recorded in the parish.[4] We would expect smallpox to have an instantaneous effect on stillbirths since smallpox infections develop within a couple of weeks of exposure and any fetal deaths would occur during the worst of the infections within four weeks of exposure (Woods 2009, pp. 218-22).

We include parish fixed effects (ρ_i) to control for time invariant parish characteristics that could confound the relationship between smallpox and stillbirths such as geographical features, population density, settlement types and the placement of a parish in the transport network. We include time fixed effects (ϕ_t) to capture common shocks to all parishes by year. These could include national smallpox epidemics, changes in national registration policies regarding stillbirths, greater integration of the transport network over time (Bergenfeldt et al. 2013) or general improvements in medical knowledge and care. Finally, we include the lagged dependent variable ($SBR_{i,t-1}$) to capture dynamic spill overs in the quality of medical care from year to year.[5] These effects would be greater for stillbirths since a skilled midwife may

[4] There may have been spatial spill overs in the smallpox variable, so we ran specifications that clustered the standard errors at the county level and found nearly identical results (not reported).
[5] The results are nearly identical if we exclude the lagged dependent variable from the regressions (not reported).

have helped to reduce intrapartum deaths but are less clear for smallpox since smallpox occurred as epidemics in most parishes.

We estimate the models for our entire period, 1780-1839, and for three sub-periods. The first period, 1780-1801, captures the relationship during equilibrium one when smallpox mortality was high before vaccination began in earnest in Sweden. The second period, 1802-19, captures equilibrium three when vaccination reached high levels causing smallpox prevalence to fall dramatically. The final period, 1820-39, measures the relationship in equilibrium four when waning immunity from vaccination would have first increased the susceptibility of child-bearing age women of contracting smallpox: the first cohorts vaccinated in the 1800s and 1810s would have been giving birth in the 1820s and 1830s. We end the third period in 1839 when revaccination was officially endorsed. Thus, these sub-periods allow us to understand the relationship between smallpox and stillbirths in three of the equilibria possible. We would expect the effects to differ across the equilibria, so being able to test this directly is important.

Although this empirical strategy does not rely on any quasi-experimental variation in smallpox mortality, we argue that once conditioning on our controls, annual smallpox mortality was as if randomly assigned. The only potential source of endogeneity in our setting is omitted variable bias because reverse causality and attenuation bias are not major problems in this historical context. There is no mechanism to explain why stillbirths would lead to smallpox mortality and substantial measurement error in the smallpox mortality variable is unlikely because the symptoms of smallpox were so distinctive that misdiagnosis in the cause of death is unlikely (Sköld 1997). Although it is impossible to rule out all confounders, the causes of stillbirths and smallpox mortality are disparate enough that it is difficult to think of omitted confounders. For instance, although stillbirths may be sensitive to income shocks, smallpox mortality is not related to nutritional status (Riley 2010), which means that localised famines

would not be confounders, at least through that pathway. Given our controls, omitted confounders would have to be time-variant, localised effects that could influence both the stillbirth rate and smallpox mortality: for instance, a localised famine that increased stillbirths and also increased labour mobility leading to smallpox epidemics. We are not aware of any historical cases that match this description. One might also consider parish-specific time trends in the variables, but these would be largely captured by the lag dependent variable. Breaking the analysis into sub-periods also reduces the possibility that long-run trends might bias our results.

B. Difference-in-differences strategy

Although we believe that the scope for endogeneity in our panel regressions is minimal, we also exploit exogenous variation in smallpox mortality in a continuous treatment difference-in-differences framework.[6] We use the introduction of vaccination in 1802 as an exogenous shock to smallpox mortality (treatment) that varied in intensity in relation to the pre-vaccination level of smallpox in each parish. This tests whether a reduction in smallpox prevalence affected stillbirths holding the share of pregnant women susceptible to smallpox constant since women of childbearing age immediately following vaccination would have acquired immunity from smallpox infections in childhood. Essentially, this tests the impact on

[6] Recent econometric work on continuous treatment difference-in-differences highlights two key assumptions for identification: the treatment cannot be anticipated and the average causal response has to be homogenous across all dosage levels of the treatment (Callaway et al. 2021). On anticipation, given that vaccination was only practiced in Sweden from the end of 1801, it is very difficult to see how the treatment could have been anticipated, especially since there were very few things that women in this period could do to cause or prevent stillbirths. Inoculation could have served as a partial anticipation, but it was never widespread in Sweden (Sköld 1996a). On the second assumption, there is no reason to believe that a one-unit decrease in smallpox prevalence (from vaccination) would have yielded different changes in stillbirths for parishes with high or low pre-vaccination smallpox prevalence. The concern is that there is selection on unobservables based on the treatment dosage that would affect the average causal response between dosage groups. The treatment effect of smallpox on stillbirths is largely a biological causal path that should not vary whether women are living in places with high or low pre-vaccination smallpox prevalence. Given that medical doctors could not treat smallpox at the time and could not prevent stillbirths caused by smallpox, it is difficult to see how the causal response would vary based on the treatment dosage.

stillbirths of moving from equilibrium one to equilibrium three (see Figure 2). The empirical specification takes the following form, estimated by OLS:

$$SBR_{i,t} = \alpha + \beta SBR_{i,t-1} + \theta Pox_{1780-1801} \times post_t + \rho_i + \phi_t + \varepsilon_{i,t} \quad (2)$$

where the dependent variable ($SBR_{i,t}$) is again the stillbirth rate in parish i and year t. The main coefficient of interest is θ, which is the effect of the interaction of smallpox prevalence before vaccination was introduced in the period 1780 to 1801 ($Pox_{1780-1801}$) and a binary indicator variable ($post_t$) equal to one in years following vaccination in 1802. We measure pre-vaccination smallpox prevalence in two ways: as the mean smallpox mortality rate in a parish between 1780 and 1801 and as the number of years with smallpox deaths in a parish in the same period. These measures capture the intensity and frequency of smallpox deaths, both of which would affect prevalence of smallpox. We use an indicator variable ($post_t$) as the vaccination shock variable because vaccination rates may have been endogenous to local, time-varying confounders that would bias the relationship of interest, and vaccination rates were only recorded at the county level anyway, preventing us from exploring parish-level variation. Again, we include the lagged dependent variable and parish and year fixed effects as controls.[7] Thus, this empirical strategy tests whether parishes with higher levels of smallpox mortality before vaccination experienced lower stillbirth rates following vaccination in 1802. Most immediately, this can be seen as a test of the extent to which pregnant women were contracting smallpox in the pre-vaccination era.

We analyse the period 1780 to 1819 for a number of reasons. First, we begin our analysis in 1780 because smallpox and measles mortality were reported together until 1774 (Ager et al. 2018). Starting from 1780 ensures that this change was fully implemented before our analysis begins. We also wanted to capture the long run average level of smallpox in a parish before vaccination in order to extrapolate away from shorter run shocks or periods

[7] Excluding the lagged dependent variable again does not change the key results.

without smallpox epidemics. Finally, we include a large number of years after vaccination in order to explore dynamic treatment effects across that period.

IV. Results

The results of the dynamic panel regressions are presented in Table 3. Again, these show the instantaneous effect of a smallpox epidemic on the stillbirth rate controlling for parish and year fixed effects and the lagged stillbirth rate. We first test the effect for the entire period from 1780 to 1839 in specifications 1 and 2. Whether we measure smallpox through the smallpox mortality rate or through a binary indicator variable equal to one when there was a smallpox death in a parish, the coefficients are statistically insignificant and close to zero.

We next break the analysis into the three subperiods reflecting the three equilibria. Before vaccination, again the effect is statistically insignificant and close to zero, which suggests that prior to vaccination when smallpox prevalence was high, very few women of childbearing age contracted smallpox. This also suggests that subclinical smallpox cases were not a source of fetal deaths and likely did not exist. In the period immediately after vaccination (1802-19), the coefficients are also statistically insignificant, but the coefficient for the smallpox mortality rate increased somewhat in specification 5 This suggests that a growing share of pregnant women were becoming susceptible, most likely because smallpox prevalence had fallen and they had escaped childhood infection rather than because of waning immunity from vaccination.

In the final period, 1820-39, when women who had been vaccinated as children were reaching childbearing age, we see positive and statistically significant coefficients on the smallpox mortality variables. Smallpox did contribute to the stillbirth rate in this period (equilibrium four). However, the size of the effect was relatively small. A one standard

Table 3: Dynamic panel regressions results for the instantaneous effect of smallpox on stillbirths

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	1780-1839	1780-1839	Equil. 1 1780-1801	Equil. 1 1780-1801	Equil. 3 1802-1819	Equil. 3 1802-1819	Equil. 4 1820-1839	Equil. 4 1820-1839
Smallpox Mortality Rate	0.003		-0.026		0.186		0.807**	
	[-0.1411,0.1472]		[-0.1734,0.1212]		[-0.1584,0.5307]		[0.1305,1.4835]	
Binary Smallpox Dummy		0.295		0.121		0.181		1.338*
		[-0.5052,1.0951]		[-1.0537,1.2959]		[-1.2355,1.5976]		[-0.2395,2.9158]
Lagged Dependent Variable	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Parish FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
N	36,698	36,698	13,062	13,062	11,196	11,196	12,440	12,440

Notes: OLS regressions estimates of Equation 1. *, ** and *** denote significance at the 10, 5 and 1 per cent level respectively. 95% confidence intervals are given in square brackets: standard errors are based on the CRV estimator. Binary smallpox dummy is equal to one in years where a smallpox death is recorded in a parish and zero otherwise. The analysis is conducted on a balanced panel of 622 parishes.

Sources: Tabellverket dataset.

deviation increase in smallpox mortality in specification 7 increased the stillbirth rate by 0.6 and the presence of a smallpox death in a parish in specification 8 increased the stillbirth rate by 1.3. These magnitudes are small relative to the variation in the stillbirth rate across parishes and years in the 1820-39 period (sd = 35.3) and can only explain a fraction of the increase in the national stillbirth rate in the first half of the nineteenth century. Thus, although more pregnant women were susceptible to smallpox in the final period and smallpox did influence stillbirths, the prevalence of smallpox was likely low enough that the impact of smallpox on stillbirths was relatively small.

We also test whether vaccination affected the stillbirth rate by drastically reducing the prevalence of smallpox in the population. Table 4 presents the results from the differences-in-differences empirical strategy. We interact the pre-vaccination smallpox measures with two periods after the introduction of vaccination to determine whether there were dynamic treatment effects. The key period of interest is the period immediately after vaccination, 1802-10, when smallpox prevalence fell sharply but women of childbearing age would still have had immunity from prior smallpox infections. We also include the period 1811-19 to understand whether the stillbirth effects were clearer once vaccination was widespread.

When looking at mean smallpox mortality before vaccination, the coefficients are actually positive suggesting that stillbirth rates were higher after vaccination in parishes with higher mean smallpox mortality rates. This is the opposite sign to what we would expect if smallpox were behaving as outlined above and is puzzling. Some of the coefficients were also statistically significant at the 10 per cent level, but overall the coefficients were small in magnitude. A one standard deviation increase in pre-vaccination smallpox mortality rates (0.66 smallpox deaths per 1000) led to an increase in the stillbirth rate of 0.90 when considering the entire post-vaccination period 1802-19 (specification 1). When interacting the two post-

Table 4: The effect of reduction in smallpox prevalence due to vaccination on stillbirth rates (difference-in-differences results)

	(1)	(2)	(3)
Smallpox Rate x Post	1.352*		
	(0.816)		
Smallpox Rate x 1802-10		0.910	
		(0.899)	
Smallpox Rate x 1811-19		1.793*	
		(0.971)	
Smallpox Count x 1802-10			-0.343
			(0.328)
Smallpox Count x 1811-19			-0.501
			(0.504)
Lag Dependent Variable	Yes	Yes	Yes
Parish FE	Yes	Yes	Yes
Time FE	Yes	Yes	Yes
N	24,258	24,258	24,258

Notes: OLS regressions estimates of Equation 2. Smallpox rate is the mean smallpox mortality rate per 1000 before vaccination (1780-1801) and smallpox count is the number of years with smallpox deaths in the same period. Post is a binary variable equal to one after vaccination began in 1802 and zero otherwise. *, ** and *** denote significance at the 10, 5 and 1 per cent level respectively. Standard errors based on the CRV estimator are in parentheses. The analysis is conducted on a balanced panel of 622 parishes.

Sources: Tabellverket dataset.

vaccination periods with the pre-vaccination smallpox rate, a one standard deviation increase in smallpox rates led to an increase of 0.60 and 1.19 in the stillbirth rate respectively (specification 2). These figures should be compared against a standard deviation of the stillbirth rate of 42.03 in the post-vaccination period.

The results are similar when we use the number of years with smallpox deaths before vaccination as our treatment variable (specification 3). Here the coefficients are negative: high frequency of smallpox epidemics before vaccination led to lower stillbirth rates after vaccination as we would expect, but again the magnitude of the coefficient is very small. These results confirm that the exogenous drop in smallpox prevalence driven by vaccination did not influence stillbirth rates. This suggests that there were very few women who contracted

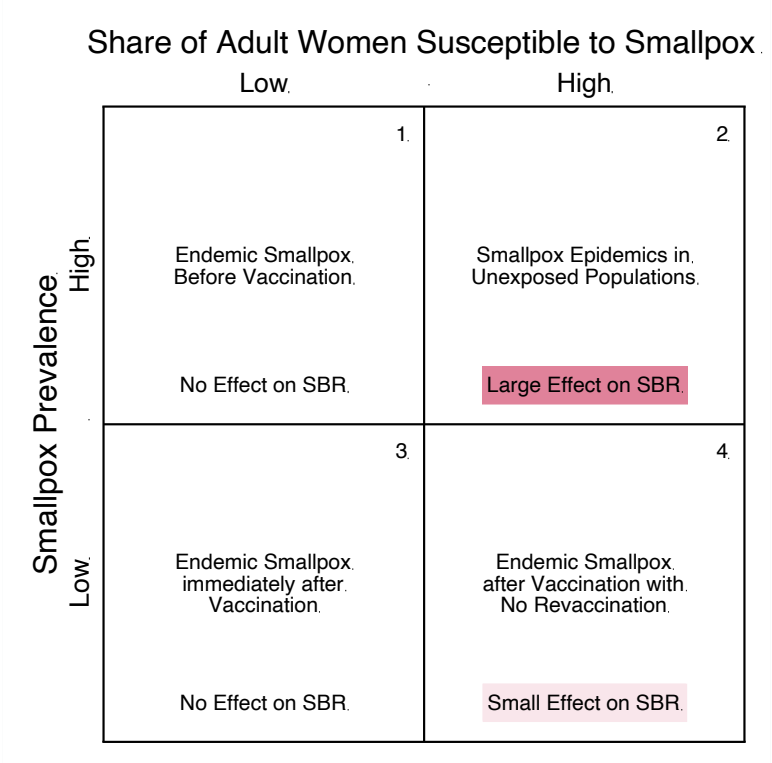
smallpox while pregnant in the pre-vaccination era. It also suggests that the decline in the national series of stillbirth rates at the beginning of the nineteenth century in Sweden, Norway and Denmark was not related to a decline in smallpox prevalence as one might have guessed.

V. Implications for other populations and contexts

Overall, in this paper we have found that smallpox was not an important cause of stillbirths in the past. Figure 4 restates our findings by placing them in the framework of the four equilibria. We find that before vaccination (equilibrium one) and immediately following it (equilibrium three), smallpox epidemics did not affect stillbirth rates. The most likely explanation for this null result is that very few women were still susceptible to smallpox by the time they reached childbearing ages because they had contracted smallpox as children. This explanation is confirmed by our difference-in-differences analysis. If pregnancies were affected by smallpox before vaccination, then an exogenous and large reduction in smallpox prevalence should have led to a decrease in stillbirth rates. However, this was not the case. We do find an instantaneous effect of smallpox on stillbirths from the 1820s onwards as women of childbearing ages who had been vaccinated as children became susceptible because of waning immunity from vaccination (equilibrium four). This effect was very small though, and cannot account for the substantial increase in stillbirth rates from the early- to mid-nineteenth century.

Although this paper has focussed explicitly on Sweden between 1780 and 1839, the results are generalisable to other times periods and places. To make inferences about other contexts, it is helpful to consider where a particular population would sit in the two-by-two matrix of equilibria (Figure 4). For countries starting at equilibrium one, the pattern of vaccination seems clear. Vaccination, if it proceeded quickly enough, would shift the population from equilibrium one to equilibrium three in the short run, but as soon as waning

Figure 4: Equilibria of smallpox and its effect on stillbirths



immunity from vaccination became an important feature of smallpox epidemiology, the population would shift to equilibrium four. The rise of revaccination in Sweden and elsewhere may have shifted populations back to equilibrium three as fewer women were susceptible to smallpox in pregnancy. Alternatively, measures such as efficient notification and isolation of cases, alongside universal child vaccination, could have also worked to keep smallpox prevalence low enough that few pregnant women would have been infected despite their continued susceptibility to smallpox in equilibrium three (Hardy 1993, p. 147-50). Thus, in the typical trajectory of the disease, it seems that smallpox was likely not an important driver of stillbirth rates.

It is also worth considering the case of southern England where inoculation and isolation of smallpox cases in pesthouses reduced the prevalence of smallpox in the eighteenth century before vaccination (Davenport et al. 2018). While it is reasonable to assume that

southern England started in equilibrium one, inoculation was not widespread enough to shift southern England to equilibrium three: the high share of smallpox deaths occurring in people over age 15 suggests pregnant women would have been at risk of contracting smallpox. Thus, it is possible that southern England was in equilibrium four in the eighteenth century in the absence of vaccination. What this means for stillbirths is difficult to establish precisely. Waning immunity should not have been a problem with inoculation since it involved infecting people with actual smallpox virus. Thus, inoculated women would not have been susceptible to smallpox infections in the same way vaccinated women were in the mid-nineteenth century. However, inoculation was never as widespread as vaccination, so there would have still been a large number of women at risk. The effect of smallpox on stillbirths was largely dependent on smallpox prevalence. In epidemics, smallpox could have caused some stillbirths, but it seems likely that the average effect was small.

Of course some sub-populations may have been at greater risk. Rural migrants to London where smallpox remained endemic in the pre-vaccination era would have been at higher risk of smallpox-induced stillbirths than women born in London. However, it is not clear whether these migrants would have had a demographically meaningful effect on the population stillbirth rate. Migrants tended to be unmarried and young. In addition, there is growing evidence that London-bound migrants were inoculated before leaving the countryside (Davenport et al. 2016). Thus, it is not clear how many births would have occurred among this subgroup before they contracted smallpox in London.

There are, however, contexts where smallpox would have been an important driver of stillbirths, reflected by equilibrium two in Figure 4. If smallpox occurred in an epidemic form and attacked a population without prior acquired immunity to the disease, then very high rates of stillbirths would be possible. The most obvious example of this would have been the smallpox epidemics that occurred among indigenous Americans as part of the Colombian

Exchange (Riley 2010, p. 274). These epidemics led to mortality on a very large scale, but as several authors have suggested, a decrease in the birth rate may have also been important in explaining depopulation (Jones 2003, p. 721; Livi-Bacci 2006). If 40 per cent of pregnant women infected with smallpox experienced stillbirths,[8] then the smallpox epidemics would have affected both births and deaths directly with important implications for the population growth rate. Of course, populations would have only suffered these massive consequences when large shares of adults had no acquired immunity to smallpox. This could occur when smallpox was first introduced or in repeated epidemics where population size and density was low enough to prevent smallpox from becoming endemic. As smallpox became endemic, populations would shift to equilibrium one.

VI. Conclusion

In conclusion, our paper has shown that smallpox is unlikely to have been an important cause of stillbirths in the past outside of very specific and short-run instances such as the smallpox epidemics during the Colombian Exchange. Our findings contradict Woods (2009)'s earlier arguments mainly because there were simply too few women who were still susceptible to smallpox in pregnancy for smallpox to matter. Thus, other factors such as obstetric practice and maternal health must have been more important in driving trends in stillbirth rates in the eighteenth and nineteenth centuries (Woods et al. 2006), despite the intriguing trends that made smallpox appear like a potentially important factor.

This also means that changes in smallpox exposure *in utero* did not have a strong influence on cohort health in the eighteenth and nineteenth centuries. One could have imagined that the decline in smallpox prevalence after vaccination would have been associated with a

[8] Pregnant women are also at greater risk for death from smallpox, so many of the women would have died as well.

reduction of fetal scarring which could have also influenced adult mortality (c.f. Quaranta 2013). However, before vaccination, smallpox affected very few births because the vast majority of women had contracted smallpox in childhood. It is possible that smallpox could have produced scarring effects among children in equilibrium four in the mid-nineteenth century, but this group of children would likely have been small enough that it would not have had a strong influence on population health.

Although this paper has focused on stillbirths, our findings can also be extended to maternal mortality. Pregnant women are also at much greater risk of dying from smallpox (Nishiura 2006), but given that very few pregnant women appear to have been infected with smallpox, it seems very unlikely that declines in smallpox prevalence could explain declining maternal mortality rates in England and Sweden in the eighteenth and early nineteenth centuries (Högberg and Wall 1986; Wrigley et al. 1997, p. 313). Large epidemics of smallpox among a vaccinated or susceptible population could lead to cases, providing the evidence Nishiura (2006) uses to estimate the effects of smallpox, but these were rare, at least in Sweden, and likely did not influence population rates.

Finally, the paper highlights how vaccination drastically changes the epidemiology of a disease and its potential to cause *in utero* shocks to health. While vaccination reduces the prevalence of a disease, it may also make pregnant women more vulnerable if immunity from vaccination wanes over time. While this paper has focused on smallpox, the same mechanism could be at play for rubella or chickenpox. The extent of fetal exposure depends on whether the prevalence of the disease is great enough to infect pregnant women. Again, when prevalence was low, few pregnant women were likely to be infected, but during epidemics the risk of infection could increase substantially. Thus, our results highlight yet again the importance of repeated vaccination to keep the prevalence of disease at low levels and protect pregnant women from infection.

References

Ager, P., Hansen, C. W., & Jensen, P. S. (2018). Fertility and early-life mortality: Evidence from smallpox vaccination in Sweden. *Journal of the European Economic Association*, *16*(2), 487–521. doi: 10.1093/jeea/jvx014

Almond, D. (2006). Is the 1918 Influenza Pandemic Over? Long-Term Effects of In Utero Influenza Exposure in the Post-1940 U.S. Population. *Journal of Political Economy*, *114*(4), 672–712. doi: 10.1086/507154

Beach, B., Brown, R., Ferrie, J., Saavedra, M., & Thomas, D. (2022). Re-evaluating the Long-Term Impact of In Utero Exposure to the 1918 Influenza Pandemic. *Journal of Political Economy*, forthcoming.

Bergenfeldt, F., Olsson, M., & Svensson, P. (2013). Wagons at work, or a transport revolution from below: the case of southern Sweden, 1750-1850. *Agricultural History Review*, *61*(1), 63–82.

Breman, J. G., & Henderson, D. A. (2002). Diagnosis and Management of Smallpox. *The New England Journal of Medicine*, *346*(17), 1300–1308. doi: 10.1056/nejmra020025

Callaway, B., Goodman-Bacon, A., & Sant'Anna, P. H. C. (2021). Difference-in-Differences with a Continuous Treatment. *arXiv*.

Carmichael, A., & Silverstein, A. M. (1987). Smallpox in Europe before the Seventeenth Century: Virulent Killer or Benign Disease? *Journal of the History of Medicine and Allied Sciences*, *42*(2), 147-68.

Crosby, A. (1993). *Smallpox* (K. F. Kiple, Ed.). In *The Cambridge World History of Human Disease* (pp. 1008–1013). Cambridge University Press.

Davenport, R. J., Boulton, J., & Schwarz, L. (2016). Urban inoculation and the decline of smallpox mortality in eighteenth-century cities-a reply to Razzell. *Economic History Review*, *69*(1), 188–214. doi: 10.1111/ehr.12112

Davenport, R. J., Satchell, M., & Shaw-Taylor, L. M. W. (2018). The geography of smallpox in England before vaccination_ A conundrum resolved. *Social Science & Medicine*, *206*, 75–85. doi: 10.1016/j.socscimed.2018.04.019

Fenner, F., Henderson, D. A., Arita, I., Jezek, Z., & Ladnyi, I. D. (1988). *Smallpox and its Eradication*. Geneva: World Health Organization.

Hardy, A. (1993). *The Epidemic Streets: Infectious Disease and the Rise of Preventive Medicine, 1856-1900.* Oxford: Oxford University Press.

Harper, K. (2021). *Plagues upon the Earth: Disease and the Course of Human History*. Princeton: Princeton University Press.

Helgertz, J., & Bengtsson, T. (2019). The Long-Lasting Influenza: The Impact of Fetal Stress During the 1918 Influenza Pandemic on Socioeconomic Attainment and Health in Sweden, 1968–2012. *Demography*, *56*(4), 1389–1425. doi: 10.1007/s13524-019-00799-x

Högberg, U., & Wall, S. (1986). Secular trends in maternal mortality in Sweden from 1750 to 1980. *Bulletin of the World Health Organization*, *64*(1), 79–84.

Högberg, U. (2004). The Decline in Maternal Mortality in Sweden: The Role of Community Midwifery. *American Journal of Public Health*, *94*(8), 1312-1320.

Jeub, U. N. (1993). *Parish Records: 19th Century Ecclesiastical Records*. Umeå University Press.

Jones, D. S. (2003). Virgin Soils Revisited. *The William and Mary Quarterly*, *60*(4), 703–742. doi: 10.2307/3491697

Livi-Bacci, M. (2006). The Depopulation of Hispanic America after the Conquest. *Population and Development Review*, *32*(2), 199-232.

Løkke, A. (2012). The antibiotic transformation of Danish obstetrics. The hidden links between the decline in perinatal mortality and maternal mortality in the mid-twentieth century. *Annales de démographie historique*, *1*(123), 205–224. doi: 10.3917/adh.123.0205

Løkke, A. (2018a). Mrs Stone and Dr Smellie: British eighteenth-century birth attendance and long-run levels and trends in maternal mortality discussed in a north European context. *Population Studies*, *72*(1), 1–14. doi: 10.1080/00324728.2017.1415036

Løkke, A. (2018b). Statistical, legal, religious and medical definitions of stillborn infants in Denmark 1683-2012. In P. Charrier, G. Clavandier, V. Gourdon, C. Rollet, & N. Sage-Pranchere (Eds.), *Morts avant de naître. La mort périnatale* (pp. 83–97). Tours: Presses universitaires François Rabelais.

Macfarlane, A., Mugford, M., Henderson, J., Furtado, A., Stevens, J., and Dunn, A. (2000). *Birth Counts: Statistics of pregnancy and childbirth. Volume 2, tables*. London: The Stationery Office.

Mazumder, B., Almond, D., Park, K., Crimmins, E. M., and Finch, C. E. (2010). Lingering prenatal effects of the 1918 influenza pandemic on cardiovascular disease. *Journal of Developmental Origins of Health and Disease*, 1(1), 26–34. doi: 10.1017/S2040174409990031.

Nishiura, H. (2006). Smallpox during pregnancy and maternal outcomes. *Emerging Infectious Diseases*, *12*(7), 1119–1121. doi: 10.3201/eid1207.051531

Ogasawara, K. (2017). Persistence of pandemic influenza on the development of children: Evidence from industrializing Japan. *Social Science & Medicine*, *181*, 43–53. doi: 10.1016/j.socscimed.2017.03.023

Office for National Statistics, 'Birth Summary Tables, England and Wales 2020', retrieved from https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/livebirths/datasets/birthsummarytables on 17 March 2022.

Petersen, B. W., Karem, K. L., & Damon, I. K. (2014). Orthopoxviruses: Variola, Vaccinia, Cowpox, and Monkeypox. In *Viral Infections of Humans, Epidemiology and Control*. 501–517. doi: 10.1007/978-1-4899-7448-8_21

van Poppel, F. (2018). Legal rules and practices of registration and burial of stillbirths in 19th and 20th century Netherlands. In P. Charrier, G. Clavandier, V. Gourdon, C. Rollet, & N. Sage-Pranchere (Eds.), *Morts avant de naître. La mort périnatale* (pp. 141–69). Tours: Presses universitaires François Rabelais.

Quaranta, L. (2013). *Scarred for life: How conditions in early life affect socioeconomic status, reproduction and mortality in Southern Sweden, 1813-1968*.

Racicot, K., & Mor, G. (2017). Risks associated with viral infections during pregnancy. *Journal of Clinical Investigation*, *127*(5), 1591–1599. doi: 10.1172/jci87490

Riley, J. C. (2010). Smallpox and American Indians Revisited. *Journal of the History of Medicine and Allied Sciences*, *65*(4), 445–477. doi: 10.1093/jhmas/jrq005

Schneider, E. B. (2017). Fetal health stagnation: Have health conditions in utero improved in the United States and Western and Northern Europe over the past 150 years? *Social Science & Medicine*, *179*, 18–26. doi: 10.1016/j.socscimed.2017.02.018

Schneider, E. B. (2022). European Stillbirth Rate Time Series Dataset (1.0) [Data set]. Zenodo. https://doi.org/10.5281/zenodo.6505519

Silasi, M., Cardenas, I., Kwon, J., Racicot, K., Aldo, P., & Mor, G. (2015). Viral Infections During Pregnancy. *American Journal of Reproductive Immunology*, *73*(3), 199–213. doi: 10.1111/aji.12355

Sköld, P. (1996a). From Inoculation to Vaccination: Smallpox in Sweden in the Eighteenth and Nineteenth Centuries. *Population Studies: A Journal of Demography*, *50*(2), 247–262. doi: 10.1080/0032472031000149336

Sköld, P. (1996b). *The Two Faces of Smallpox: A Disease and its Prevention in Eighteenth- and Nineteenth-Century Sweden*. Umeå University Press.

Sköld, P. (1997). Escape from Catastrophe: The Saami's Experience with Smallpox in Eighteenth- and Early-Nineteenth-Century Sweden. *Social Science History*, *21*(1), 1–25. doi: 10.1017/s0145553200017624

Sommerseth, H. L. (2021). Born Dead or Alive? Revisiting the Definition of Stillbirths in Norway. *Historical Life Course Studies*, *10*, 96–100. doi: 10.51964/hlcs9575

Statistics Netherlands, StatLine, 'Population, households and population dynamics; from 1899', retrieved from https://opendata.cbs.nl/statline/#/CBS/en/dataset/37556eng/table?ts=1649688016225 on 17 March 2022.

Statistics Norway, 'Table 05803: Population 1 January and population changes during the calendar year, by year and contents', retrieved from https://www.ssb.no/en/statbank/table/05803/tableViewLayout1/ on 11 April 2022.

Statistics Sweden, 'Live births, stillbirths and infant mortality by year, sex and observations', retrieved from https://www.statistikdatabasen.scb.se/pxweb/en/ssd/START__BE__BE0101__BE0101I/LevandeFodda/table/tableViewLayout1/ on 11 April 2022.

Tabellverket Database, version 1.0, Umeå University Demographic Data Base, retrieved 2015-01-09.

Welch, W. M. (1878). Smallpox in the pregnant woman and in the foetus. *Philadelphia Medical Times*, *8*, 390–398.

Wisborg, K., Barklin, A., Hedegaard, M., & Henriksen, T. (2008). Psychological stress during pregnancy and stillbirth: prospective study. *BJOG: An International Journal of Obstetrics & Gynaecology*, *115*(7), 882–885. doi: 10.1111/j.1471-0528.2008.01734.x

Woods, R. (2009). *Death before birth fetal health and mortality in historical perspective*. Oxford University Press.

Woods, R. I., Lokke, A., & van Poppel, F. (2006). Two hundred years of evidence-based perinatal care: late-fetal mortality in the past. *Archives of Disease in Childhood - Fetal and Neonatal Edition*, *91*(6), F445–F447. doi: 10.1136/adc.2006.098178

Wrigley, E. A., Davies, Oeppen, J., & Schofield, R. (1997). *English Population History from Family Reconstitution*. Cambridge: Cambridge University Press.

Web Appendix:

Did Smallpox Cause Stillbirths?
Maternal Smallpox Infection, Vaccination and Stillbirths in Sweden, 1780-1839

Eric B. Schneider (LSE and CEPR),
Sören Edvinsson (Umeå University)
and Kota Ogasawara (Tokyo Institute of Technology)

3 May 2022

Appendix A Data Appendix

A.1 Systematic Missings in the Original Stillbirths Statistics

The statistics suggest that the data on the number of stillbirths after 1802 suffer systematic missing observations. Table A.1 summarizes this issue (we show the summary statistics between 1798 and 1808 for brevity). As shown, the number of parishes that reported the number of births listed in Table A.1 is relatively stable over the period. Before 1802, the number of parishes that reported the stillbirths is very close to that reported the births: for instance, in 1801, the number of parishes reporting the births was 1,692, whereas the number of parishes reporting the stillbirths was 1711. However, the number of parishes that reported the number of stillbirths drops dramatically in 1802 by 562 parishes (from $1,711$ in 1801 to $1,149$ in 1802), accounting for 33% of the number of parishes reported in 1801. This substantial declines in the number of parishes causes an underreporting issue in the number of stillbirths. In fact, the solid line illustrated in Figure A.1 shows the time-series plots of the number of stillbirths in our sampled periods, which indicates that there was a clear valley between 1802 and 1821.

Table A.1: Number of Births, Stillbirths, and Women giving Stillbirth in Original *Tabellverk* Statistics between 1798 and 1808

								Number of Women giving Stillbirth					
		Number of Births			Number of Stillbirths [A]			Full sample			Missing cells in [A]		
Year	Unit	Observations	Mean	Std. Err.	Observations	Mean	Std. Err.	Observations	Mean	Std. Err.	Observations	Mean	Std. Err.
1798	Parish	1764	34.74	32.24	1778	0.97	1.45	–	–	–	–	–	–
1799	Parish	1742	33.44	31.30	1761	0.90	1.29	–	–	–	–	–	–
1800	Parish	1722	29.77	28.54	1751	0.81	1.29	–	–	–	–	–	–
1801	Parish	1692	31.30	29.58	1711	0.82	1.27	6	1.50	2.74	2	4.00	4.24
1802	Parish	2025	31.34	30.51	1149	0.19	0.75	1869	0.85	1.37	728	1.89	1.49
1803	Parish	2057	30.58	28.81	1149	0.21	0.80	1893	0.83	1.28	746	1.78	1.30
1804	Parish	2081	31.86	32.45	1204	0.19	0.74	1916	0.83	2.09	713	1.92	2.98
1805	Parish	2108	31.44	29.74	1159	0.20	1.11	1913	0.84	2.23	757	1.88	3.18
1806	Parish	2115	30.69	29.77	1182	0.18	0.74	1924	0.82	1.94	744	1.85	2.67
1807	Parish	2127	31.42	30.70	1141	0.16	0.68	1935	0.83	1.29	795	1.78	1.35
1808	Parish	2141	30.64	29.28	1207	0.15	0.65	1932	0.77	1.18	730	1.78	1.17

Notes: This table presents the summary statistics of the number of births, stillbirths, and women giving stillbirth between 1798 and 1808 obtained from original *Tabellverk* dataset. Sources: *Tabellverk* dataset.

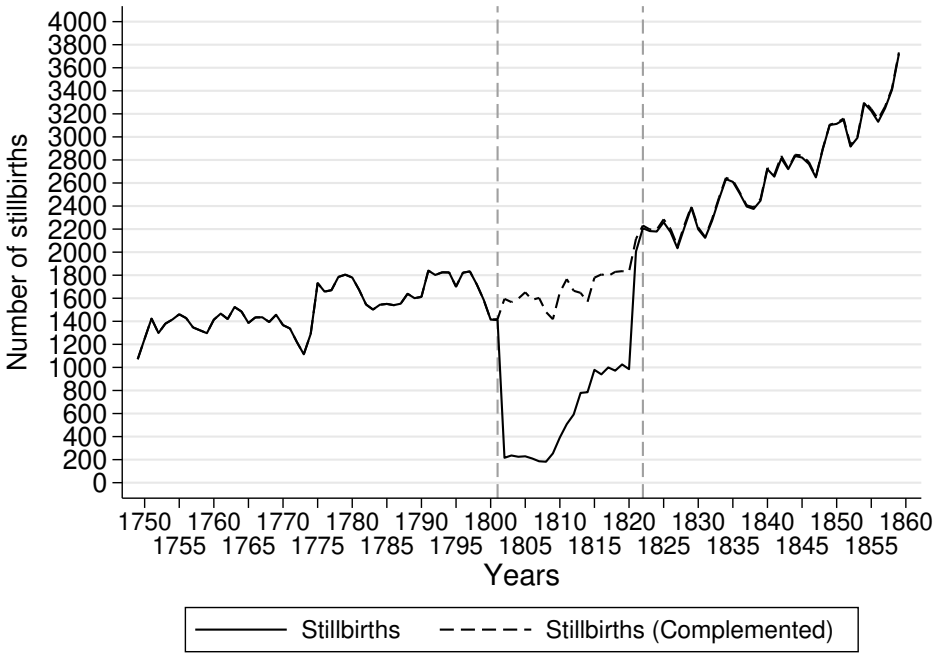


Figure A.1: Number of Stillbirths: Original vs Complemented Statistics

Notes: The solid line indicates the number of stillbirths originally reported in the *Tabellverk* dataset. The dotted line indicates the number of stillbirths complemented using the number of women giving stillbirths reported in the *Tabellverk* dataset. In the complemented series, the missing observations in the data on the number of stillbirths are replaced with the number of women giving stillbirth. The gray dashed lines show 1801 and 1822, respectively. Sources: *Tabellverk* dataset.

A.2 Complementation of the Stillbirths Statistics

Fortunately, we can use the data on the number of women giving stillbirths from 1801, which are considered to be a quantitatively similar variable to the number of stillbirths. In order to deal with this sort of systematic underreporting issue, therefore, we complement the number of stillbirths using the data on the number of women giving stillbirths. We simply replace the missing observations in the stillbirths with the women giving stillbirths. Table A.1 illustrates this mechanism. The number of parishes that reported the number of women giving stillbirths was $1,869$ in 1802, and 728 parishes of those did not report the number of stillbirths. Then, we replaced the 728 missing observations in the stillbirth data with the data on the number of women giving stillbirth. Note that if we add the number of parishes reporting the stillbirths (i.e., $1,149$) to the number of parishes reporting the number of women giving stillbirth alone (i.e., 728), the total number of parishes (i.e., $1,877$) is reasonably much closer to the number of parishes reporting the births $(2,025)$. Considering this consistency, without loss of generality, we have done the same replications for all measured years.

Figure A.1 compares the original stillbirth data (containing substantial missing observations) and complemented stillbirth data. Clearly, the complemented stillbirth data

reasonably bridge the gaps in the stillbirth statistics in the early 19th century. Table A.2 presents the details of this complementation around 1802, suggesting our complementation would correct roughly 0.6–0.7 underreported stillbirths after 1801 (see the final columns of Table A.2). One must be careful about the fact that a set of women might had gave twin stillbirths rather than the single stillbirths. However, an important fact is that both original and complemented series become mostly identical after 1822 (see Figure A.1). This strongly supports the evidence that our method employed herein is less likely to be influenced by such a small proportion of twin stillbirths. Finally, A.2 shows the differences between the number of stillbirths and the number of women giving stillbirths. There is a clear spike in the class of "zero", in which the women data are identical to the stillbirth data. Considering these facts, our complemented data must provide a much better statistics on the stillbirths than the originally reported ones.

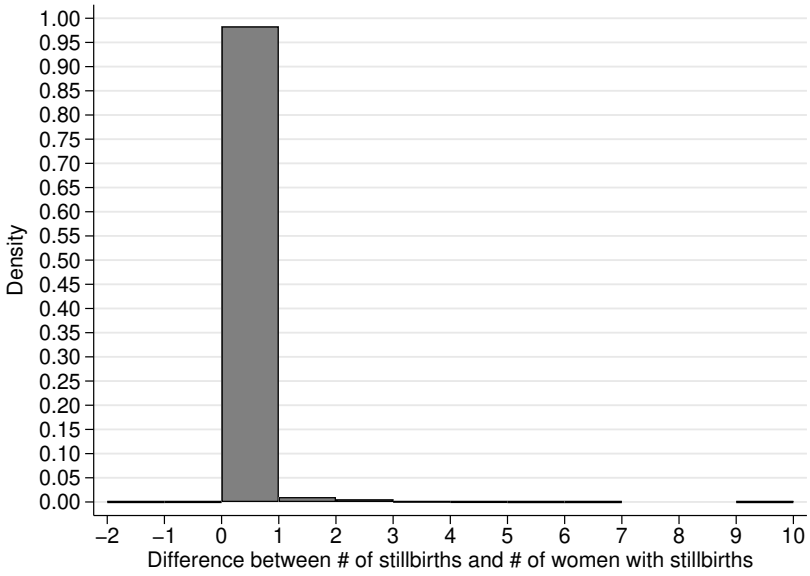


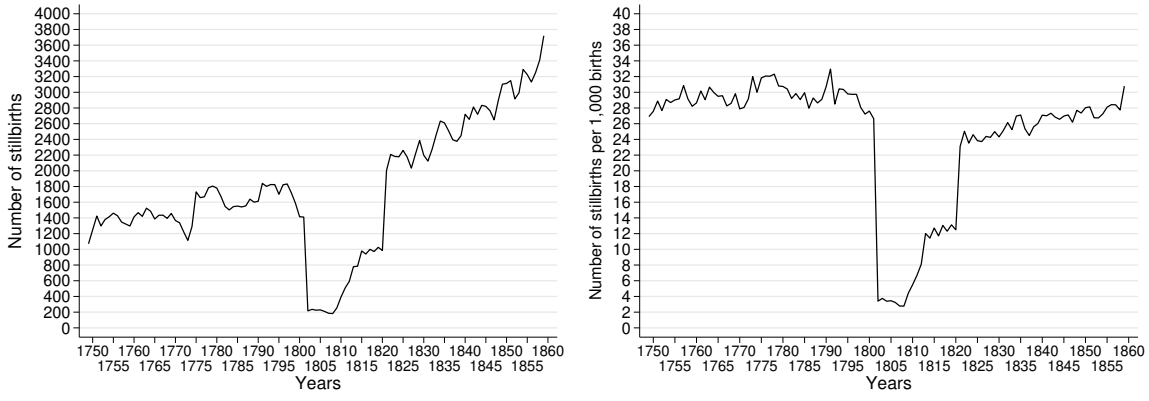
Figure A.2: Density of the differences between the number of stillbirths and the number of women giving stillbirths

Notes: This histogram shows the distribution of the differences between the number of stillbirths originally reported and the number of women giving stillbirths. Among 161,470 total observations, 80,713 observations have both the stillbirths and women statistics at the same time, which are used in this figure. Note that, in the complementation, we replaced the stillbirths with the number of women giving stillbirths if the stillbirths statistics are missing. Sources: *Tabellverk* dataset.

Table A.2: Examples of the Systematic Bias in the Original Stillbirth Statistics: Comparison between Original Stillbirths and Complemented Stillbirths Statistics (1798–1808)

		Number of Stillbirths			Number of Stillbirths [Complemented]			Mean Difference
Year	Unit	Observations	[A] Mean	Std. Err.	Observations	[B] Mean	Std. Err.	([A]-[B])
1798	Parish	1778	0.97	1.45	1778	0.97	1.45	0
1799	Parish	1761	0.90	1.29	1761	0.90	1.29	0
1800	Parish	1751	0.81	1.29	1751	0.81	1.29	0
1801	Parish	1711	0.82	1.27	1713	0.81	1.29	0.01
1802	Parish	1149	0.19	0.75	1877	0.85	1.38	−0.66
1803	Parish	1149	0.21	0.80	1895	0.83	1.28	−0.62
1804	Parish	1204	0.19	0.74	1917	0.83	2.09	−0.64
1805	Parish	1159	0.20	1.11	1916	0.86	2.33	−0.66
1806	Parish	1182	0.18	0.74	1926	0.82	1.94	−0.64
1807	Parish	1141	0.16	0.68	1936	0.83	1.29	−0.67
1808	Parish	1207	0.15	0.65	1937	0.76	1.18	−0.61

Notes: This table presents the summary statistics of the number of stillbirths and the number of stillbirths complemented using the number of women giving stillbirth. Sources: *Tabellverk* dataset.

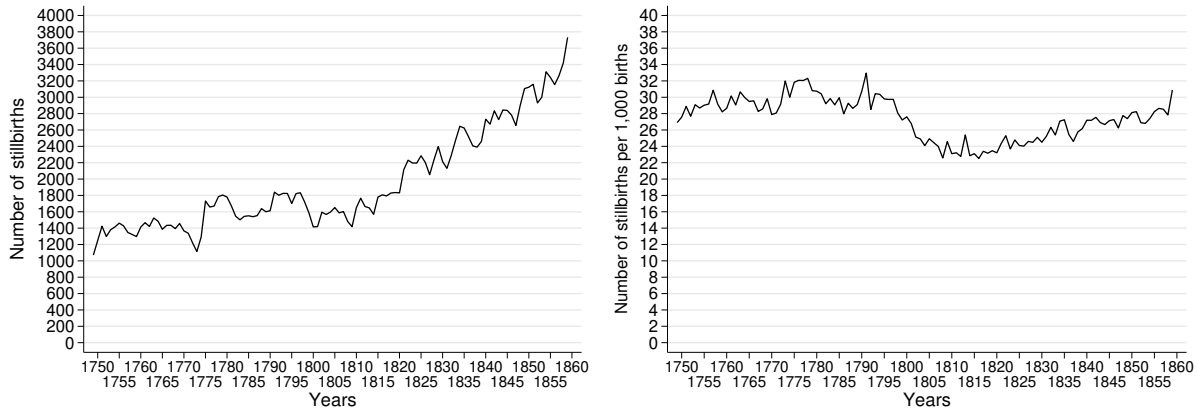


(a) Number of stillbirths (counts)

(b) Stillbirth rate (permille)

Figure A.3: Five-year Total Stillbirths and Average SBR: Original Stillbirth Statistics

Notes: Figure A.3a illustrates the number of stillbirths from original stillbirth statistics. Figure A.3b illustrates the stillbirth rate calculated using the number of stillbirths from original stillbirth statistics (Figure A.3a). Source: *Tabellverk* dataset.

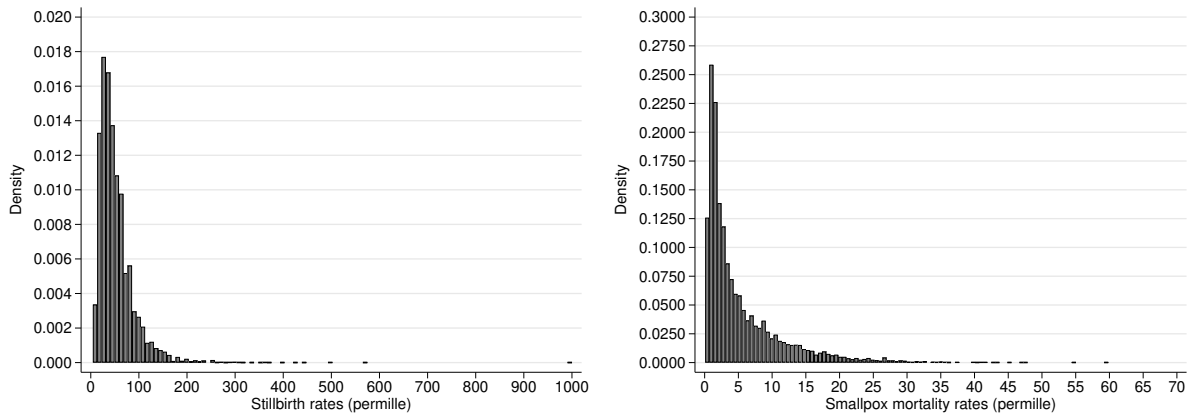


(a) Number of stillbirths (counts)

(b) Stillbirth rate (*SBR*, permille)

Figure A.4: Five-year Total Stillbirths and Average SBR: Complemented Stillbirth Statistics

Notes: Figure A.4a illustrates the number of stillbirths from the complemented stillbirth statistics. Figure **??** illustrates the stillbirth rate (*SBR* in equation 1) calculated using the complemented stillbirth statistics (Online Appendix A.2). Source: *Tabellverk* dataset.



(a) Stillbirth rate (*SBR*, permille)

(b) Smallpox mortality rate (*SMR*, permille)

Figure A.5: Density of the Stillbirth and Smallpox Mortality Rates

Notes: Notes: SBR and SMR are defined as equation 1. Censored observations are not shown in both figures. Source: *Tabellverk* dataset.

A.3 Smallpox Mortality and Stillbirth Rates

A.3.1 Definitions

For parish p in year t, the stillbirth rate (SBR) and smallpox rate (SMR) are defined as as follows:

$$\begin{aligned} SBR_{pt} &= 1,000 \times \frac{Stillbirths_{pt}}{Births_{pt}} \\ SMR_{pt} &= 1,000 \times \frac{Smallpox_{pt}}{Pred\ Pop_{pt}}, \end{aligned} \tag{1}$$

where *Stillbirths*, *Births*, *Smallpox*, and *Pred Pop* are the number of stillbirths, births, smallpox deaths, and predicted population, respectively. Since there is a systematic underreporting in the number of stillbirths in the 1800s and 1810s, we complemented stillbirth data using the women giving stillbirth. We provide a finer discussion on this method and its validity in Online Appendices A.1 and A.2. Specifically, we show that the original stillbirth statistics are underreported from 1801 to 1801 in Online Appendix A.1. In Online Appendix A.2, we provide evidence that our complemented stillbirth statistics would be able to efficiently trace the actual stillbirth statistics.

A.3.2 Trimming

We explain how we trimmed the missing observations.

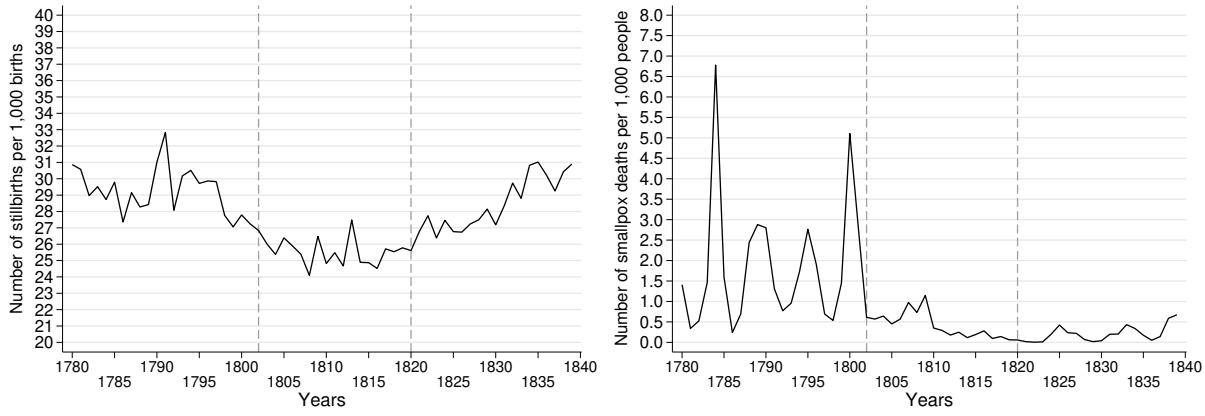
First of all, we trimmed the missing observations in the following ways. There are originally 228, 187 observations in total. (1) we exclude the missing observations in the stillbirths in counts (*Stillbirths*). This cut 28, 072 observations, and 200, 115 observations are left. (2) we exclude the missing observations in the births in counts (*Births*). This cut 1, 740 observations, and 198, 375 observations are left. (3) we exclude the missing observations in the smallpox deaths in counts (*Smallpox*). There are no missing observations. (4) we exclude the missing observations in the predicted population in counts (*Pred Pop*). This cut 6, 752 observations and 191, 623 observations are left.

Second, as explained in the main text, we keep the sample periods between 1780 and 1839. This excludes 88, 598 observations, and 106, 154 observations are left.

Third, we exclude the parishes with missing observations, i.e., the unbalanced panels, to make balanced panel dataset between 1780 and 1839. This excludes 68, 834 observations, leaving 622 parishes with 37, 320 (622 parishes $\times$ 60 years).

A.3.3 Trends

Figure A.6a Figure A.6b



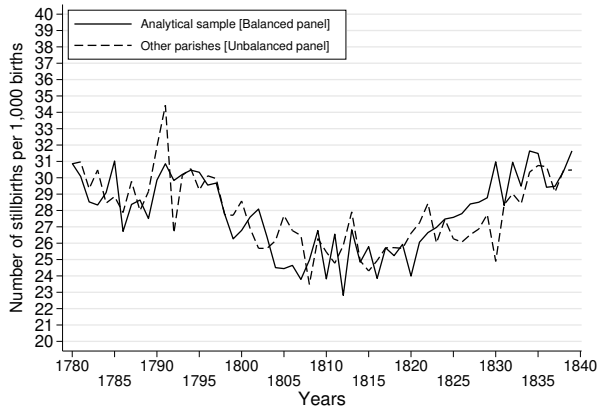
(a) Stillbirth rate (*SBR*) (b) Smallpox rate (*SMR*)

Figure A.6: Stillbirth and smallpox rates in Sweden: 1749–1859

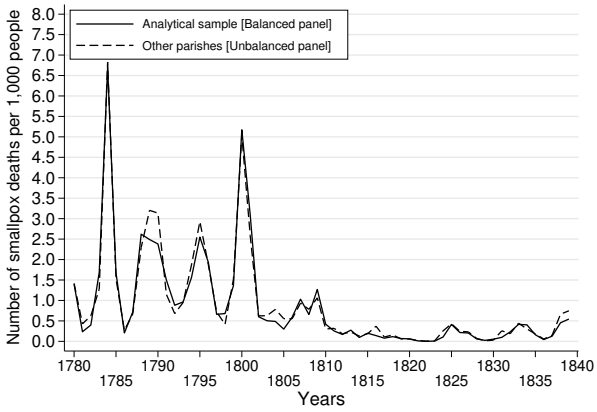
Notes: Stillbirth rate (*SBR*) is the number of stillbirths per 1,000 births. Smallpox rate (*SMR*) is the number of smallpox deaths per 1,000 people. *SMR* uses the complemented stillbirth statistics (Online Appendix A). Source: *Tabellverk* dataset.

A.3.4 Balanced-Analytical Sample vs Other Parishes

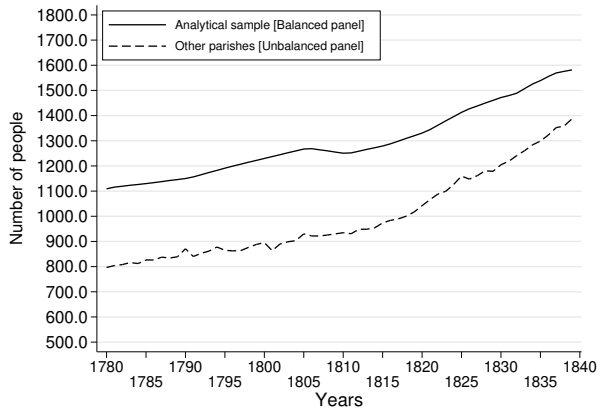
Figure A.7a compares the time-series plots of the stillbirth rates between our analytical balanced panel data and the other parishes, i.e., unbalanced panels. Two series show similar transitions and indeed the mean difference between both series is close to zero (-0.6). Figure A.7b comapres the time-series plots of the smallpox mortality rates by the panels. As shown, both series show very similar transitions and the mean difference is again very close to zero (-0.02). Although both figures provide evidence that our analytical sample can be used as the representative parishes in the sense that it shows very similar mortality patterns to the other parishes not included in the sample. A potentially important difference is that the size of parishes used in the analyses are slightly greater than those of the other parishes. Figure A.7c shows the average number of people in both groups, suggesting that the number of people in the parishes in our analytical sample is larger than the other parishes by roughly 200-300 people. This means that the number of births in the analytical sample is also larger than the other parishes as shown in Figure A.7d. Therefore, despite the similarities in the mortality rates, one must be careful about the fact that this paper focuses on the parishes that have relatively larger scale of people.



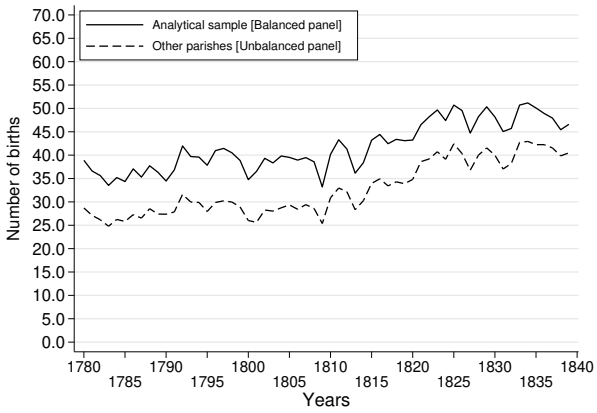
(a) Stillbirth rate (permille)



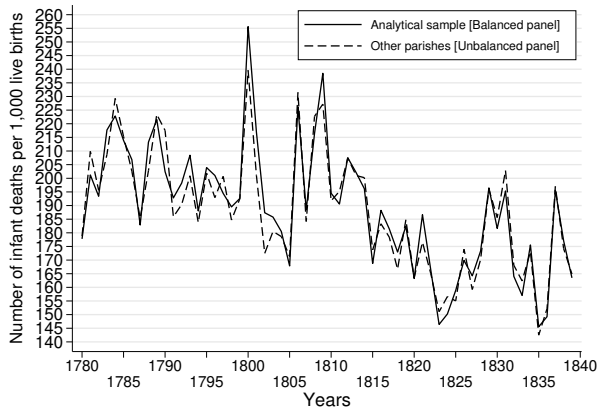
(b) Smallpox mortality rate (permille)



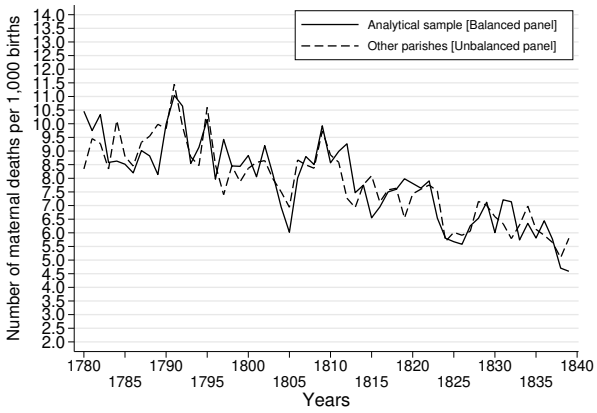
(c) Number of people



(d) Number of births



(e) Infant mortality rate



(f) Maternal mortality rate

Figure A.7: Analytical Sample vs Other Parishes Comparisons

Notes: Notes: Figure A.7a and A.7b illustrate the stillbirth and smallpox rates for the balanced and unbalanced parishes (panels), respectively. The mean difference in the stillbirth rates between two groups is -0.61 (p-value $= 0.039$). The mean difference in the smallpox mortality rates between two groups is -0.02 (p-value $= 0.299$). Figure A.7c and A.7d show the number of people and births for the balanced and unbalanced parishes (panels), respectively. Figure A.7e and A.7f show the infant mortality and maternal mortality rates for the balanced and unbalanced parishes (panels), respectively. The number of balanced (unbalanced) parishes is 622 (1,977). Source: *Tabellverk* dataset.

Appendix B Additional Results

B.1 Results for Unbalanced Panel Dataset

Our main analytical sample used in the main text is the balanced panel dataset. We show that the main results are largely unchanged if we use the unbalanced panel dataset including almost all of the parishes. Table B.1 presents the results for all the parishes with more than or equal to 5 measured years.[1] The results are largely unchanged to those reported in Table 3. Table B.2 shows the results for the difference-in-difference styled regressions using the same unbalanced panel. As shown, the results are similar to those listed in Table 4.

[1] I have removed the parishes with very short panels (i.e., less than 5 years) because we use the lagged dependent variable in the specifications.

Table B.1: Dynamic panel regressions results for the instantaneous effect of smallpox on stillbirths: Results using unbalanced panels

	1780–1839		1780–1801		1802–1819		1820–1839	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Smallpox Mortality Rate	0.033		0.031		0.083		0.356**	
	[-0.0574,0.1243]		[-0.0694,0.1315]		[-0.1545,0.3212]		[0.0031,0.7093]	
Binary Smallpox Dummy		0.503*		0.625		0.198		1.125**
		[-0.0522,1.0579]		[-0.2248,1.4744]		[-0.8189,1.2155]		[0.0592,2.1910]
Lagged Dependent Variable	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Parish FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Time FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
N	100,728	100,728	33,400	33,400	31,193	31,193	36,135	36,135

Notes: OLS regressions estimates of Equation 1. *, ** and *** denote significance at the 10, 5 and 1 per cent level respectively. 95% confidence intervals are given in square brackets: standard errors are based on the CRV estimator. Binary smallpox dummy is equal to one in years where a smallpox death is recorded in a parish and zero otherwise. Sources: Tabellverk dataset.

Table B.2: The effect of reduction in smallpox prevalence due to vaccination on stillbirth rates (difference-in-differences results): Results using unbalanced panels

	(1)	(2)	(3)
Smallpox Rate × Post	1.011**		
	(0.468)		
Smallpox Rate × 1802-10		0.742	
		(0.543)	
Smallpox Rate × 1811-19		1.270***	
		(0.460)	
Smallpox Count × 1802-10			-0.203
			(0.193)
Smallpox Count × 1811-19			0.075
			(0.150)
Lagged Dependent Variable	Yes	Yes	Yes
Parish FE	Yes	Yes	Yes
Time FE	Yes	Yes	Yes
N	24,258	24,258	24,258
R-square	0.0074	0.0074	0.0572

Notes: OLS regressions estimates of Equation 2. Smallpox rate is the mean smallpox mortality rate per 1000 before vaccination (1780-1801) and smallpox count is the number of years with smallpox deaths in the same period. Post is a binary variable equal to one after vaccination began in 1802 and zero otherwise. *, ** and *** denote significance at the 10, 5 and 1 per cent level respectively. 95% confidence intervals are given in square brackets. Standard errors based on the CRV estimator are in parentheses.
Sources: Tabellverk dataset.