Online Apppendix for

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

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1 Introduction

This online appendix reports supplementary material to the paper "Fertility response to early-life mortality: Evidence from smallpox vaccination in Sweden" by Philipp Ager, Casper Worm Hansen, and Peter Sandholt Jensen.

2 Literature overview

Appendix Table 1 provides an overview of the literature on mortality and fertility.

Appendix Table 1

3 Theory

This section outlines a simple one-period static model, which follows Doepke (2005) and Galor (2011) closely, to fix ideas on the possible effects of early-life mortality on (gross) fertility or liveborn, and net fertility, which is broadly defined as the number of surviving children.[1]

Consider a household deriving utility from consumption, c, and the number of surviving children, ϕn:

$$V = (1-\alpha)\ln c + \alpha \ln(\phi n), \tag{1}$$

where $0 < \alpha < 1$ is the utility weight on surviving children, n is the number of children born, and $0 < \phi \leq 1$ is the early childhood survival rate. We think of this survival rate as being determined by the economic environment, w, and the extrinsic mortality environment, x:

$$\phi = \phi[w, x], \tag{2}$$

where $\phi_w > 0$ and $\phi_x < 0$. Therefore, if the probability of getting an infectious disease decreases (e.g., by smallpox vaccination), x decreases, and the survival rate increases.

The household is confronted with the following budget constraint:

$$(\rho + \phi)\, n + c = w, \tag{3}$$

[1] For another recent application of this type of model; see Strulik (2014).

where $\rho + \phi$ is the cost of raising a child, which involves a fixed term, ρ, independent of the survival rate (i.e., the "cost" of pregnancy), such that the cost of raising a non-surviving child is always larger than zero;[2] 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 (3), taking the survival function in equation (2) as given. The explicit solution for the number of liveborn children is:

$$n = \frac{\alpha}{(\rho + \phi[w, x])} w. \tag{4}$$

The number of surviving children is given by:

$$\phi n = \frac{\alpha \phi[w, x]}{(\rho + \phi[w, x])} w. \tag{5}$$

It is evident from inspecting equations (4) and (5) that fertility is decreasing in the survival rate, whereas net fertility is increasing and concave, implying that the numerical magnitude of an increase in the survival rate is always smaller for net fertility. 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 liveborn children, n, constant) —always dominates the negative intensive effect (i.e., $\frac{\partial n}{\partial \phi} < 0$). Furthermore, if the fixed cost of raising children is zero ($\rho = 0$), the survival rate has no effect on net fertility, as also argued in Galor (2011).[3]

Finally, had there been a wage cost of having children, while holding the survival rate constant, the demand for children would be independent of the income w in the log-utility case (i.e., $\frac{\partial n}{\partial w}|_{\phi} = 0$). We also note that the optimal level of consumption, c, is unrelated to the survival rate, implying that a fall in the extrinsic mortality environment, x, would not give rise to any changes in the normal consumption level in this simple setup.

In sum, this theory predicts that a decrease in the extrinsic mortality environment increases the early childhood survival rate which has the following implications for fertility:

1. *A negative effect on the number of liveborn children,*

2. *A (small but) positive effect on the number of surviving children.*[4]

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

[3] Galor (2011) uses a similar setup, though he allows time spent on child rearing to impact the income earned by the household. This setup leads to similar predictions as discussed above for the case of $\rho = 0$.

[4] It is worthwhile to mention that using Barro-Becker type of models, Doepke (2005) shows that a fall in

4 Figures with yearly data

Appendix Figure 1 shows yearly smallpox mortality rates, whereas Appendix Figure 2 shows similar data for typhoid mortality rates. The yearly data show appreciably more fluctuations, though we note that the large epidemics die out after 1801 for smallpox. As noted in the text, mean smallpox mortality is also reduced remarkably. This is not the case for typhoid fever. The graphs show smallpox and typhoid fever mortality per 1,000,000. Using a Chow test, we find that the breaks in the smallpox series is significant, whereas there is no significant break for typhoid fever.

Appendix Figures 1 and 2

5 Data sources and explanation

Infant and child mortality: The digitized data are available at http://ships.ddb.umu.se/ and come from official statistics as described in Section 4 (of the paper). *Smallpox mortality:* These data were provided by Sköld (1996) from the Umeå database. *Rye Harvest:* The data are summarized in *Statistiska Meddelanden Serie A. Band VI:4* published in 1949 for the years 1802-1820 by county on a yeary basis. From 1828, data are available for each county in *Kongl. Maj:ts befallningshafvandes femårsberättelse.* In 1828, the data are available in separate reports for each county. From 1833 to 37 the data are available in summary publications. The digitized publications are available at: http://www.scb.se/sv_/Hitta-statistik/Historisk-statistik/Statistik-efter-serie/Officiell-statistik-1811-1860/Femarsberattelser-1817-1855/. *Price of rye*: These data are reported in Jörberg, L. (1972) Prices in Sweden, 1732-1914, volume II in kronor per hectolitre. Appendix Table 2 describes the main variables used in the empirical analysis and Appendix Table 3 reports the summary statistics.

child mortality has a negative effect on fertility but a positive effect on the number of surviving children, with the theoretical implication that decreasing early childhood mortality cannot play any part in explaining the fertility transitions observed historically in present-day developed countries. Note, that our second prediction may be reversed, when the number of surviving children is uncertain and certain conditions, such as having a precautionary demand for children, are satisfied, see Galor (2011, p.121) for details. For example, Kalemli-Ozcan (2003, 2008) shows that declining mortality rates reduce the uncertainty of child survival, and thus fertility, as parents decrease their precautionary demand for children.

Appendix Tables 2 and 3

6 Event study for net fertility

Appendix Figure 3 depicts the event study for net fertility (i.e., surviving children to the age of 1). The setup in the same as in Figure 5 of the paper, but now the outcome is net fertility. Importantly, we find that trends between parishes in counties with higher and lower levels of smallpox mortality evolve relatively similarly prior to the introduction of the vaccination method. In the post-treatment period only the coefficient for 1810 is negative and significant, while the remaining post-treatment estimates are statistically insignificant. Overall, the evidence suggests that there was no effect on net fertility of introducing the vaccination method.

Appendix Figure 3

7 Instrument exogeneity

Appendix Figure 4 presents 2SLS estimates of infant mortality using the modified Instrumental-Variable approach of Conley et al. (2012). In both graphs we report the 90% confidence intervals for the second-stage estimates of infant mortality for the crude birth rate (panel A) and the general fertility rate (panel B) as dependent variable. The specifications are as in columns 1 and 2 of Table 7, except using only *vaccination* as the excluded instrument (for the sake of simplicity we excluded *law 1816* from these regressions). The union of confidence interval approach (UCI) is used to evaluate the (potential) direct effect of smallpox vaccination, which we denote as γ, on gross fertility (i.e. the crude birth rate in panel A and the general fertility rate in panel B) imposing that the support of the direct effect γ is $[-\delta, 0]$, with $\delta > 0$, (i.e., we impose that the direct effect of smallpox vaccination on gross fertility is negative ranging between $-\delta$ and 0).[5]

Both panels show the threshold at which the estimated 2SLS coefficient of infant mortality (the endogenous variable) becomes statistically insignificant at the 10-percent level (the crossing

[5] We refer to Conley, Hansen and Rossi (2012) for further details.

of the zero line (red) with the dashed lower-bound of the confidence interval). This threshold has a straightforward interpretation: for panel A it shows that at $\delta \approx -0.012$ the direct effect on smallpox vaccination would turn the second-stage effect insignificant; for panel B it is at $\delta \approx -0.055$. In other words, to invalidate the overall reduced form effect (displayed by the vertical green line), the omitted variable that is also captured by *vaccination* needs to be about 57% $(-0.012/-0.021)$ of the overall reduced form effect for panel A and 54% $(-0.055/-0.101)$ for panel B to render our 2SLS estimate on infant mortality insignificant. Given the presented evidence this magnitude seems implausible.

Appendix Figure 4

8 Unweighted results

Appendix Table 4 shows that the 2SLS estimates without population weights are very similar to the baseline weighted 2SLS estimates reported in the paper.

Appendix Table 4

9 Panel-time structure and standard errors

Firstly, this section reports the main 2SLS estimates, using different assumptions about the time and panel structure. Secondly, while some of these changes—to some extent—already deal with the problem of too few clusters, this section also reports standard errors estimated in ways that deal with this issue, as well as the potential problem of spatial correlation.

Appendix Table 5 reports 2SLS estimates with different assumptions about the time and panel structure. In particular, panel A collapses the 5-years observations into *one* pre–treatment period and *one* post–treatment period based on the cutoff date of the vaccination-shock variable (i.e., $\tau > 1801$) and cluster the standard errors at the parish level instead of the county level. This approach implies that we cannot include the second instrumental variable (*law 1816*) nor control for county-specific linear time trends as there is only one pre-treatment period observation. Nevertheless, according to Bertrand et al. (2008), this is one way of dealing with the problem of too few clusters since this would reduce the problem of serial correlation and thus allow us to cluster at the lower (parish) level, which increases the number of clusters to 777. As seen from panel A, the results are similar to our baseline 2SLS estimates in the paper. Panel B collapses the 5-years observations into 10-years observations. Thus, 1795/1800 now constitutes the pre–treatment period and 1805/1810, 1815/1820, .., 1855/1860 are the 10-year post-treatment periods. As in panel A, we cannot control for county-specific linear time trends, although we are now able to include *law 1816*. Again, the results are the same as in the baseline. Finally, collapsing the parish level observations to the county level also give rise to similar results; see panel C.

Following the approach as in the published paper by Acemoglu and Johnson (2007),[6] Appendix Table 6 uses a two-period approach, that is, exploiting only *one* pre-treatment period (i.e., the average of the years 1795 and 1800) and various post-treatment periods (i.e., 1805/1810, 1815/1820, 1825/1830, etc.). In line with the reduced-form event-study analysis, reported in Figure 5, we find that the effects of infant mortality on the crude birth rate and the general fertility rate are strongest using either the post-treatment period 1825/1830 or the post-treatment period 1835/1840.[7]

Appendix Table 7 reports reduced-form estimates using the wild cluster bootstrap method proposed by Cameron et al., (panel A), using two-way clustering by county and year (panel B), and Conley standard errors allowing for a spatial correlation cutoff of 150 kilometers, and a lag of 20 years to account for autocorrelation (panel C). Making these corrections to the standard errors does not alter our basic conclusions.

Appendix Tables 5, 6 and 7

10 Controlling for population and urbanization

Panel A of Table 8 in the paper already shows that our findings are robust to controlling for initial (log) population size and initial urbanization rate (both interacted with the time indicator). While we follow this approach in order not to include any potential endogenous control variables, Appendix Table 8 reports the 2SLS estimate when directly controlling for log

[6] In the associated working paper (Acemoglu and Johnson, 2006), they also consider panel models with more than two years (e.g., 10-year panel model from 1940 to 2000).

[7] As in panels A and B of Appendix Table 4, this type of specification does not allow us to control for county-specific linear time trends.

population and urbanization rate, respectively. In particular, the specifications in panel A are similar to the baseline exempt for the fact that we now also control log population size, however, this does not really change the estimated 2SLS coefficients. Panel B considers the implications of controlling for the urbanization rate. Because of data availability on urbanization rates, these specifications only include observations every decade starting in 1800, which also means that we cannot control for county-specific linear time trends (as we only have one pre-treatment period). Nevertheless, we generally reach the same conclusion, namely that infant mortality has a positive impact on measures of gross fertility, whereas the effect on net fertility is limited.

Appendix Table 8

11 Functional forms and survival rates

The theory in Appendix Section 3 suggests the following prediction for live-born children:

$$\ln n = \ln \alpha + \ln w - \ln(\rho + \phi), \tag{6}$$

where we have used the using the natural logarithmic form of equation (4). Since $\phi = 1 - \lambda$, where λ is the infant mortality rate, this implies that:

$$\ln n = \ln \alpha + \ln w - \ln(1 + \rho - \lambda) \approx \ln \alpha + \ln w + \lambda, \tag{7}$$

where the last approximation assumes that $\rho - \lambda$ is small (i.e., close to zero). Adding an error term, ε_{it}, and indices, we end up with the following estimation equation:

$$\ln n_{it} = \ln \alpha + \pi_1 \lambda_{it} + \beta \ln w_{it} + \varepsilon_{it}. \tag{8}$$

Thus, assuming $\rho - \lambda$ is close to zero then, according to the theory, we should use a log-level specification. Moreover, the theory suggests that $\pi_1 = 1$.

Appendix Table 9 reports 2SLS estimates using different functional-form specifications. Panel A reports the 2SLS estimates from a log-log specification, panel B uses a log-level specification, whereas panel C reports the findings from a level-log specification. First, we see that, regardless of the functional-form assumption applied, infant mortality has a positive effect on the crude birth rate and general fertility rate, while the effect on the various measures of net fertility (columns 3–6) is generally statistically insignificant from zero, although the effect on the number of surviving children to the age of 5 actually comes in negative and significant, which is consistent with theory if $\rho > 0$. Second, the 2SLS estimate in columns 1 and 2 of panel B (the log-level model) is *very* close to the magnitude predicted by theory, remembering that infant mortality is defined as the number of death per 1,000 liveborn. In particular, we find that $\hat{\pi}_1 = 1.02$ (column 1) and $\hat{\pi}_2 = 1.15$ (column 2), and the theory predicts that $\pi_1 = 1$.

Assuming that $\rho = 0$, the theoretical relationship between the number of live-born children and the infant survival rate is given by:

$$\ln n = \ln \alpha + \ln w - \ln \phi, \tag{9}$$

which means that if we specify the empirical model in terms of the infant *survival* rate instead of the infant mortality rate, the theory suggests that we should use a log-log model. Therefore, the estimation equation now reads:

$$\ln n_{it} = \ln \alpha - \pi_1 \ln \phi_{it} + \ln w_{it} + \varepsilon_{it}, \tag{10}$$

where theory predicts that $\pi = 1$, that is, a one percent increase in the survival rate should *reduce* the number of live-born children with one percent.

Appendix Table 10 reports the 2SLS estimates, using the log infant survival rate as the explanatory variable and the usual measures of fertility (in logs) as outcomes. First, we observe that the first stage remains unaffected by this change, as the Kleibergen-Paap F-statistics is around 10 in all the specifications. Second, we find negative relationships between the infant survival rate and the gross-fertility measures, which, in terms of magnitude, are close to the what the theory predicts (column 1: $\hat{\pi}_1 = -0.80$ and column 2: $\pi_1 = -0.90$). Third, the relationships between the infant survival rate and the net fertility measures are in most cases statistically indistinguishable from zero.

Appendix Tables 9 and 10

12 Lagged-dependent-variable models

Appendix Table 11 shows that our main 2SLS estimates are robust to the inclusion of a lagged dependent variable, as well as the lagged infant mortality rate.

Appendix Table 11

References

[1] Acemoglu, D, and S. Johnson. 2006. Disease and Development: The Effect of Life Expectancy on Economic Growth. Working paper no. 12269 (June), NBER, Cambridge, MA.

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

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

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

[5] Forsell (1833). Utkast til en svensk statistisk, andra afdelingen, 1820.

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

[7] Kalemli-Ozcan, S. (2002). Does the mortality decline promote economic growth?, Journal of Economic Growth 7(4), 411.439.

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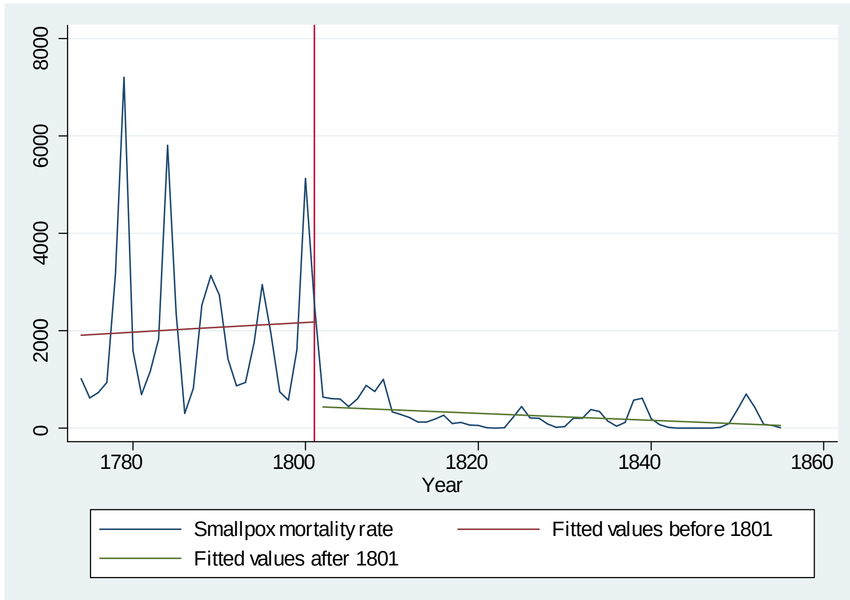
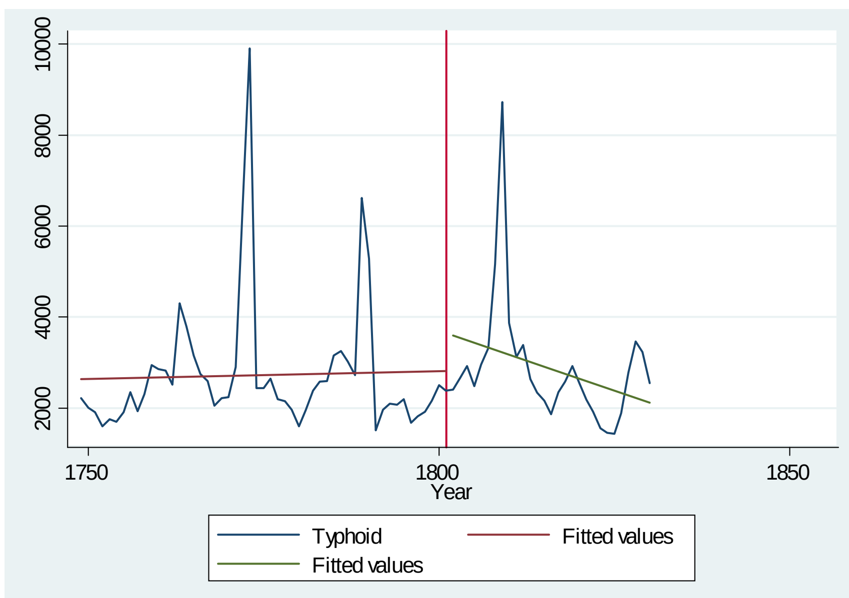
[9] Schulz, D. (1801). Tal om den offentliga värden, i hänseende til folkets seder og helsa, samt de fattigas lifbergning. Jon P. Lindh, Stockholm, Sweden.

[10] Strulik, H. (2014). Contraception and Development: A Unified Growth Theory. Discussion Papers on Business and Economics.

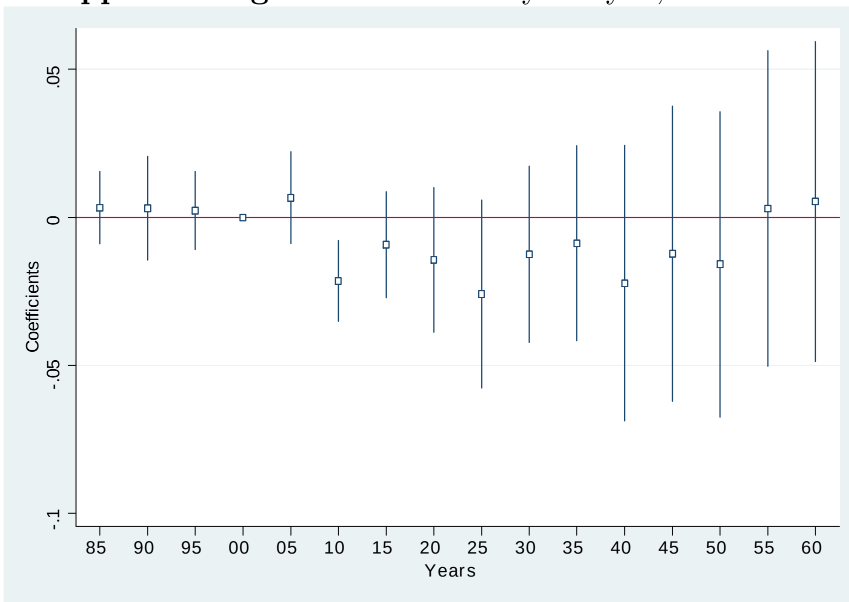
Appendix Figure 1: Yearly smallpox mortality rates for Sweden.



Appendix Figure 2: Yearly typhoid fever mortality rates for Sweden.

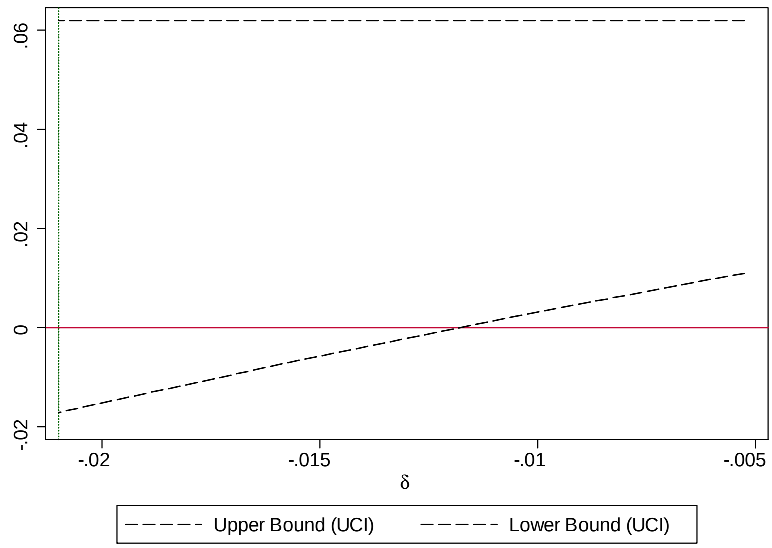


Appendix Figure 3: Event-study analysis, 1780-1860

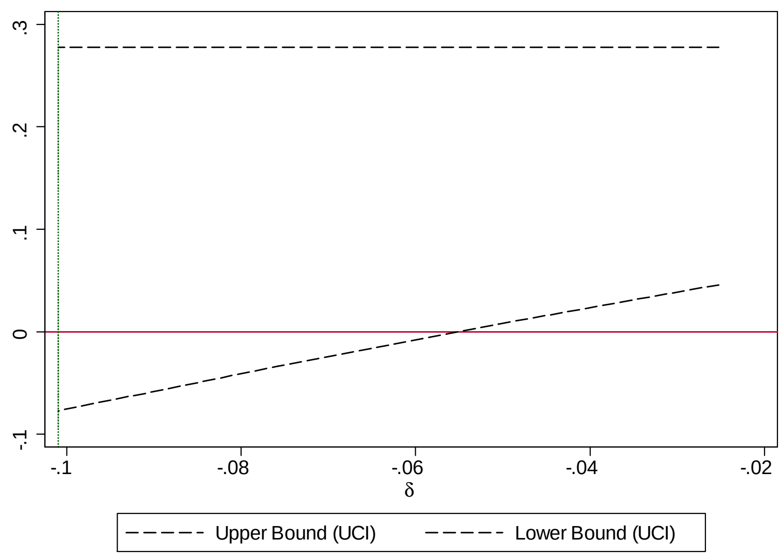


Notes: This graph reports estimated differences in net fertility (i.e., surviving children age 1) in the treatment-intensity measure for the period 1780-1860 relative to the omitted year 1800, by estimating the net fertility on SMR_j^I, county and time fixed effects, county-specific linear time trends, and initial infant mortality and crude birth rate for each year. The estimate for 1780 is also omitted due to the county-specific linear time trends. The vertical lines indicate 95 percent confidence intervals, based on robust standard errors clustered at the county level.

Appendix Figure 4: Plausibly exogenous technique



Panel A



Panel B

These figures show the upper and lower bound of the 90% confidence interval of the second-stage coefficient of infant mortality on the crude birth rate (panel A) and the general fertility rate (panel B) using the union of confidence interval (UCI) approach suggested by Conley et al. (2012). Estimates are based on specifications from column 1 and 2 in Table 7 in the paper using smallpox vaccination as excluded instrument. These figures were produced using the 'plausexog' Stata code produced by Damian Clark.

Appendix Table 1: Literature overview

Study	Data	Instruments	Findings
Dynamic panel estimation			
Angeles 2010	Cross-country panel	lagged variables	Positive and significant relationship
Bhalotra and van Soest 2008	Micro level data for India	n/a	Positive and significant relationship
Hansen et al. forthcoming	US states panel	lagged variables	Nonrobust relationship
Herzer et al. 2012	Cross-Country panel	n/a	Positive and significant relationship
Murtin 2013	Cross-country panel	lagged variables	Nonrobust relationship
Instrumental variables estimations			
Benefo and Schultz 1996	Survey data for Ghana and Cote d'Ivoire	malaria eradication	Positive and significant relationship
Haines 1997	US census data for 1900 and 1910	proportion of dead children	Positive and significant relationship
Lorentzen et al. 2008	Cross-country data	malaria ecology	Positive and significant relationship
Lee et al. 1997	Prussian city panel	male adult mortality	Positive and significant relationship
McCord et al. 2017	Cross-Country panel	malaria risk	Positive and significant relationship
Murphy 2015	French counties	temperature	Negative and insignificant
Schultz 1997	Cross-country data	calories consumption per capita	Positive and significant relationship
Differences-in-differences			
Fortson 2009	Survey data for African countries	n/a	Small, negative effects of HIV
Lucas 2013	Survey date for Sri Lanka	n/a	Positive effects of Malaria eradiaction
Juhn, Kalemli-Ozcan, and Turan 2013	Survey data for African countries	n/a	Negative effects of HIV
Wilson 2016	Survey data for Zambia	n/a	Negative effect of HIV prevention intervention on fertility

Appendix Table 2: Data explanations and sources

Variable:	Explanation and source:
birth rate:	Number of birth per 1000 population (SHiPS).
survings children (age 1):	Number of surviving children to the age of 1. This variable is constructed as (1000-infant mortality) × birth rate (SHiPS).
survings children (age 5):	Number of surviving children to the age of 1. This variable is constructed as (1000-child mortality) × birth rate (SHiPS).
general fertility rate	Number of births per 1000 women of child bearing ages, 15-45 (SHiPS).
child-women ratio	Number of children below the age of 5 per 1000 women of child bearing ages, 15-45 (SHiPS).
infant mortality:	Number of infant deaths (age 0-1) per 1000 live births (SHiPS).
child mortality:	Number of child deaths (age 1-5) per 1000 live births (SHiPS).
death rate:	Number of deaths per 1000 population (SHiPS).
natural population growth:	Birth rate minus death rate (SHiPS).
vaccination (instrument):	The smallpox mortality rate measured in 1796-1801 interacted with a time indactor which takes in the value 1 after 1801 (Sköld, 1996).
law 1816 (instrument):	The smallpox mortality rate measured in 1811-1816 interacted with a time indactor which takes in the value 1 after 1815 (Sköld, 1996).
smallpox mortality:	The smallpox mortality rate measured at the number of deaths from small-pox per 100,000 population (Sköld, 1996).
vaccination rate:	The proportion of children born in the previous 5 year period which was vaccinated (Sköld, 1996).
price rye:	Price of rye (Dribe et al., 2011).
log rye/capita:	Log of rye production per capita (Dribe et al., 2011).
initial human capital:	
high school	A dummy which is equal to 1 if the county had a high school (in Swedish Gymnasium) and zero otherwise (Schulz, 1801)
..trivial schools	The number per county of so-called trivial schools which taught at lower levels than proper high school (Schulz, 1801)
..university enrollment	Population per university student in 1815 by county (Forsell, 1833)

Notes: This table describes the main variables used in the analysis.

Appendix Table 3: Summary Statistics

	(1)	(2)	(3)	(4)	(5)
	N	mean	sd	min	max
birth rate	10,878	31.96	6.618	6	151
surving children (age 1)	10,878	26.77	6.177	2.331	121.7
surving children (age 5)	9,011	24.35	6.377	0.925	115.5
infant mortality	10,878	163.4	84.89	0	857
general fertility rate	9,549	138.4	32.28	13.03	808.7
child-women ration	9,549	543.2	130.1	152.9	2,444
child mortality	9,011	99.55	89.72	6	923
death rate	10,877	22.74	8.690	2	237
natural population growth	10,877	9.216	10.39	-125	50
vaccination (instrument)	10,878	187.0	93.72	0	318.7
law 1816 (instrument)	10,878	14.38	17.80	0	55.80
smallpox mortality	10,101	40.14	60.35	0	318.7
vaccination rate	10,314	57.90	29.97	0	119.9
price rye	10,878	7.039	2.476	2.121	13.63
log rye/capita	9,490	-2.451	0.755	-5.931	2.911

Notes: This table reports summary statistics for the main variables used in the regression analysis.

Appendix Table 4: Unweighted (baseline) 2SLS estimates

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
infant mortality	0.0359***	0.186***	-0.000530	-0.0205***	0.174	-0.0203*
	(0.0102)	(0.0436)	(0.00811)	(0.00766)	(0.186)	(0.0121)
Kleibergen-Paap F-stats.	12.70	10.48	12.64	11.94	10.37	13.07
Anderson-Rubin [p-value]	[0.001]	[0.000]	[0.259]	[0.073]	[0.092]	[0.255]
Hansen-J [p-value]	[0.171]	[0.541]	[0.169]	[0.957]	[0.146]	[0.731]
Controls $\times 1[\tau > 1801]$:						
initial mortality	yes	yes	yes	yes	yes	yes
intial outcome	yes	yes	yes	yes	yes	yes
Observations	10,878	8,845	10,878	7,727	8,845	10,877

Notes: The table reports 2SLS estimates using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 and *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments. The name of the specific outcome variable is indicated in the top row. The main explanatory variable is the infant mortality rate, which is the number of infant deaths per 1000 live born. All the regressions are unweighted and include county and year fixed effects, and county-specific linear time trends. Initial mortality is the infant mortality rate in 1800 and the initial outcome is the specific outcome (indicated in the top tow) measured in 1800. Constants are not reported. Robust standard errors are clustered at the county level.

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

Appendix Table 5: Time and panel structure

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
	Panel A: collapsing into two periods: pre/post 1801					
infant mortality	0.0495***	0.290***	0.0143	-0.00957	0.493	0.000776
	(0.0129)	(0.0750)	(0.0107)	(0.0103)	(0.300)	(0.0147)
observations	1,554	1,479	1,554	1,374	1,479	1,554
	Panel B: collapsing into 10-yr periods					
infant mortality	0.0403**	0.227***	0.00508	-0.00530	0.154	0.00285
	(0.0164)	(0.0772)	(0.0136)	(0.0177)	(0.331)	(0.0257)
observations	5,439	4,971	5,439	4,416	4,971	5,439
	Panel C: collapse to the county level					
infant mortality	0.0578**	0.272**	0.0165	0.0165	-0.310	-0.00644
	(0.0231)	(0.112)	(0.0193)	(0.0358)	(0.511)	(0.0213)
observations	336	312	336	332	312	336

Notes: The table reports 2SLS estimates. Panel A uses *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 as one excluded instrument. Panels B and C *also* use *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as an excluded instrument; so that panels B and C exploit two instruments in total. The name of the specific outcome variable is indicated in the top row. The main explanatory variable is the infant mortality rate, which is the number of infant deaths per 1000 live born. All regressions are weighted by log population size in 1800 and include the controls: county and year fixed effects and initial outcomes (measured in 1800) interacted with the time indicator. Panel C also controls for county-specific linear time trends. Panel A collapses the pre-treatment periods 1795 and 1800 into one pre-treatment period and the post-treatment period 1805, 1810,.., 1860 into one post-treatment period. Panel B collapses the model into 10 yr. periods instead of 5-yr periods. Panel C collapses the parish level observations to the county level. First-stage results and constants are not reported. Robust standard errors are clustered at the parish level in Panel A and at the county level in Panels B and C.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 6: A two-period approach

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	age 5	child-women ratio	natural pop. growth
	Panel A: pre-period: 1795/1800 and post-period: 1805/1810					
infant mortality	0.0260**	0.103	-0.00692	-0.0257**	-0.154	-0.0152
	(0.0129)	(0.0648)	(0.0110)	(0.0128)	(0.318)	(0.0175)
observations	1,554	1,471	1,554	1,356	1,471	1,554
	Panel B: pre-period: 1795/1800 and post-period: 1815/1820					
infant mortality	0.0461**	0.258***	0.00942	-0.0178	0.157	-0.0153
	(0.0182)	(0.0889)	(0.0150)	(0.0139)	(0.346)	(0.0213)
observations	1,554	1,477	1,554	1,346	1,477	1,554
	Panel C: pre-period: 1795/1800 and post-period: 1825/1830					
infant mortality	0.0787**	0.482**	0.0374	0.0119	1.026	-0.00763
	(0.0323)	(0.205)	(0.0280)	(0.0227)	(0.672)	(0.0353)
observations	1,554	1,475	1,554	1,359	1,475	1,554
	Panel D: pre-period: 1795/1800 and post-period: 1835/1840					
infant mortality	0.0558**	0.335***	0.0203	0.0177	0.809	0.0324
	(0.0223)	(0.119)	(0.0190)	(0.0195)	(0.661)	(0.0396)
observations	1,554	1,472	1,554	1,338	1,472	1,554
	Panel E: pre-period: 1795/1800 and post-period: 1845/1850					
infant mortality	0.0391	0.195	0.00441	0.00246	-0.141	0.0176
	(0.0360)	(0.166)	(0.0295)	(0.0346)	(0.504)	(0.0561)
observations	1,554	1,469	1,554	1,342	1,469	1,554
	Panel F: pre-period: 1795/1800 and post-period: 1855/1860					
infant mortality	0.00746	0.0205	-0.0196	-0.0570	-0.523	-0.0378
	(0.0351)	(0.152)	(0.0280)	(0.0398)	(0.479)	(0.0460)
observations	1,554	1,457	1,554	1,350	1,457	1,554

Notes: The table reports 2SLS estimates. Panel A uses *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 as one excluded instrument. Panels B-E *also* exploits *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as an excluded instrument; so that Panels B-E exploit two instruments in total. The name of the specific outcome variable is indicated in the top row. The main explanatory variable is the infant mortality rate, which is the number of infant deaths per 1000 live born. All the regressions are weighted by log population size in 1800 and include the controls: county and year fixed effects and initial outcomes (measured in 1800) interacted with the time indicator. In all the specifications, the pre-treatment periods 1795 and 1800 are collapsed into only one pre-treatment period, and the specific post-treatments periods are indicated in the panel headlines. First-stage results and constants are not reported. Robust standard errors are clustered at the county level (note, even smaller standard errors are obtained if we instead cluster at the parish level).
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 7: Standard errors

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Dependent variable:						
	infant mortality	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
Panel A: wild cluster bootstrap							
vaccination	-0.531***	-0.0210**	-0.101**	-0.00207	0.00877	-0.0736	0.00944
bootstrap p-value	0.002	0.014	0.012	0.742	0.254	0.802	0.344
observations	10,878	10,878	8,845	10,878	7,727	8,845	10,877
Panel B: clustering by the county-year level							
vaccination	-0.531***	-0.0210**	-0.101*	-0.00207	0.00877	-0.0736	0.00944
	(0.155)	(0.0104)	(0.0556)	(0.0114)	(0.0152)	(0.201)	(0.0174)
observations	10,878	10,878	8,845	10,878	7,727	8,845	10,877
Panel C: conley standard errors							
vaccination	-0.531***	-0.0210***	-0.101***	-0.00207	0.00877	-0.0736	0.00944
	(0.081)	(0.007)	(0.036)	(0.007)	(0.008)	(0.110)	(0.012)
observations	10,878	10,878	8,845	10,878	7,727	8,845	10,877

Notes: The table reports least squares estimates weighted by log population size in 1800. The main explanatory variable is *vaccination*, which is the smallpox mortality rate in 1796–1801, interacted with a time indicator that equals one after 1801. *Law 1816*, which is constructed as smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811–1815) interacted with a time indicator that equals one after 1815, is also included, but not reported. All regressions include the baseline controls: county and year fixed effects, county specific linear time trends, and initial outcomes (measured in 1800) interacted with the time indicator. In panel A, the reported bootstrap two tailed p-values are calculated using the wild cluster bootstrap method of Cameron et al., 2008. Panel B reports robust standard errors are clustered at the county-by-year level. Panel C reports Conley standard errors in parentheses, allowing for spatial correlation between counties that are within 150 kms of each other and for autocorrelation of 20 years. Constants are not reported.
*** $p<0.01$, ** $p<0.05$, * $p<0.1$.

Appendix Table 8: Robustness to population and urbanization

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
	Panel A: controlling for population size directly					
infant mortality	0.0353***	0.175***	0.000120	-0.0172*	0.0915	-0.0149
	(0.0109)	(0.0543)	(0.00933)	(0.00983)	(0.265)	(0.0128)
observations	10,814	8,845	10,814	7,684	8,845	10,813
	Panel B: controlling for the urbanization rate directly					
infant mortality	0.0263*	0.155***	-0.00442	-0.0216*	0.0494	-0.000218
	(0.0139)	(0.0593)	(0.0114)	(0.0128)	(0.234)	(0.0237)
observations	5,439	4,101	5,439	3,942	4,101	5,438

Notes: The table reports 2SLS estimates using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 and Law 1816, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments.The name of the specific outcome variable is indicated in the top row. The explanatory variable is the infant mortality rate, which is the number of infant deaths per 1000 live born. All the regressions are weighted by log population size in 1800 and include the following controls: county and year fixed effects, and initial outcomes (measured in 1800) interacted with the time indicator. Panel A also controls for county-specifc linear time trends and log population size. Panel B controls for the urbanization rate and, due to data availability, only includes observations every 10th year starting in 1800. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 9: Robustness to functional-form assumptions

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
	Panel A: log-log					
infant mortality	0.180***	0.208***	-0.0528	-0.254***	0.00162	-0.307
	(0.0550)	(0.0657)	(0.0686)	(0.0891)	(0.0949)	(0.577)
observations	10,283	8,358	10,283	7,366	8,358	4,368
	Panel B: log-level					
infant mortality	0.00102***	0.00115***	-0.000263	-0.00139***	4.08e-05	-0.00168
	(0.000329)	(0.000387)	(0.000349)	(0.000424)	(0.000495)	(0.00322)
observations	10,878	8,845	10,878	7,703	8,845	4,656
	Panel C: level-log					
infant mortality	6.380***	32.05***	-0.0236	-3.176*	13.20	-2.814
	(1.750)	(8.945)	(1.737)	(1.905)	(50.84)	(2.592)
observations	10,283	8,358	10,283	7,389	8,358	10,282

Notes: The table reports 2SLS estimates using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 and *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments. The name of the specific outcome variable is indicated in the top row; its functional form (level or log) is indicated in panel headline. The explanatory variable is the infant mortality rate, which is the number of infant deaths per 1000 live born; its functional form (level or log) is indicated in panel headline. All the regressions are weighted by log population size in 1800 and include the baseline controls: county and year fixed effects, county specific linear time trends, and initial outcomes (measured in 1800) interacted with the time indicator. First-stage results and constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 10: The impact of the infant survival rate on fertility and population growth

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable (in logs):					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
log infant survival rate	-0.788***	-0.882***	0.212	1.102***	-0.0327	1.367
	(0.266)	(0.314)	(0.266)	(0.315)	(0.380)	(2.639)
Kleibergen-Paap F-stats.	10.93	8.45	10.93	12.09	8.56	10.22
Anderson-Rubin [p-value]	[0.003]	[0.002]	[0.297]	[0.035]	[0.380]	[0.655]
Hansen-J [p-value]	[0.290]	[0.643]	[0.290]	[0.421]	[0.247]	[0.400]
Controls $\times 1[\tau > 1801]$**:**						
initial survival rate	yes	yes	yes	yes	yes	yes
intial outcome	yes	yes	yes	yes	yes	yes
Observations	10,878	8,845	10,878	7,703	8,845	4,656

Notes: The table reports 2SLS estimates using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 and *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments. The name of the specific outcome variable (in logs) is indicated in the top row. The explanatory variable is the log infant survival rate, which is the number of infant survivers per 1000 live born. All the regressions are weighted by log population size in 1800 and include the baseline controls: county and year fixed effects, county specific linear time trends, and initial outcomes (measured in 1800) interacted with the time indicator. Constants are not reported. Robust standard errors are clustered at the county level.
*** p<0.01, ** p<0.05, * p<0.1.

Appendix Table 11: Including lagged dependent variables

	(1)	(2)	(3)	(4)	(5)	(6)
	Dependent variable:					
	crude birth rate	general fertility rate	surviving children age 1	surviving children age 5	child-women ratio	natural pop. growth
infant mortality	0.0269***	0.136***	-0.00633	-0.0245**	-0.0838	-0.0203**
	(0.00800)	(0.0424)	(0.00689)	(0.00965)	(0.138)	(0.00959)
lagged dependent variable:						
infant mortality	yes	yes	yes	yes	yes	yes
2nd stage outcome	yes	yes	yes	yes	yes	yes
observations	10,814	8,342	10,814	6,615	8,342	10,814

Notes: The table reports 2SLS estimates using *vaccination*, which is the smallpox mortality rate in 1796-1801 interacted with a time indicator that equals one after 1801 and *law 1816*, which is the smallpox mortality rate prior to the vaccination law of 1816 (i.e., 1811-1815) interacted with a time indicator that equals one after 1815, as the two excluded instruments. The name of the specific outcome variable is indicated in the top row. The explanatory variable is the infant mortality rate, which is the number of infant deaths per 1000 live born. All the regressions are weighted by log population size in 1800 and include the baseline controls: county and year fixed effects, county-specific linear time trends, initial outcomes (measured in 1800) interacted with the time indicator, and lagged dependent variables (e.g., column 1 includes lagged infant mortality and lagged crude birth rate.). First-stage results and constants are not reported. Robust standard errors are clustered at the county level.

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