The contribution of infrastructure investment to Britain's urban mortality decline 1861-1900*

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January 5, 2018

Abstract

It is well-recognized that both improved nutrition and sanitation infrastructure are important contributors to mortality decline. However the relative importance of the two factors is difficult to quantify, since most studies are limited to testing the effects of specific sanitary improvements. This paper uses new historical data regarding total investment in urban infrastructure, measured using the outstanding loan stock, to estimate the extent to which the mortality decline in England and Wales between 1861 and 1900 can be attributed to government expenditure. Fixed effects regressions indicate that infrastructure investment explains approximately 30 per cent of the decline in mortality between 1861 and 1900. Since these specifications may not fully account for the endogeneity between investment and mortality, I estimate additional specifications using lagged investment as an instrument for current investment. These estimates suggest that government investment was the major contributor to mortality decline, explaining up to 60 per cent of the reduction in total urban mortality between 1861 and 1900. Additional results indicate that investment in urban infrastructure led to declines in mortality from both waterborne and airborne diseases.

Keywords: sanitation, mortality, public investment, Britain, urban infrastructure.

* I thank Philip Hoffman, Jean- Laurent Rosenthal and Erik Snowberg for their helpful advice. I am also grateful for comments from three anonymous referees, Matthew Chao, Tim Hatton, Samantha Myers, Eric Schneider, Matthew Shum, Frank Trentmann, Werner Troesken, and audiences at Caltech, the University of Southern Denmark, All-UC Economic History workshops, the Economic History Society and the World Cliometrics Conference. I am grateful for financial support from the History Project of the Institute of New Economic Thinking, and from NSF grant 1357995. All errors remain my own.

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Between 1851 and 1900 mortality rates in Britain declined by almost 20 per cent. Over the same period, local government expenditure on urban infrastructure increased rapidly so that by 1890 spending by local authorities accounted for over 41 per cent of total public expenditure, with much of the money used for water supply and sewers.[1] This simple pattern leads to the natural conclusion that government sanitation expenditure was the driving force behind the improvement in life expectancy. This belief is also supported by evidence from other countries showing that investment in sanitary infrastructure, such as clean water supply, can have positive effects on mortality both in the present day[2] and historically[3].

Yet the role of public health in explaining British mortality decline in the nineteenth century remains disputed. The classic explanation of the dramatic fall in mortality rates after 1850 – due to McKeown – has emphasized the importance of better nutrition rather than improvements in the sanitary environment.[4] This conclusion followed from estimates showing that the greatest contribution to the decrease in mortality rates during this period came from reductions in airborne, rather than waterborne or foodborne, diseases. More recent studies, however, have questioned his conclusion without ending the debate or pinning down the precise quantitative impact that sanitary investment had on mortality.[5] In particular, this later work has argued that McKeown's thesis overlooks the potential contribution of sanitary reform in reducing overcrowding (and hence deaths from airborne diseases) and does not account for differences in the death rates from different

[1] Lizzeri and Persico 'Why did the elites', p. 711.
[2] e.g. Günther and Fink, 'Water and sanitation'; Zwane and Kremer 'What works'; Deaton, 'The great escape'.
[3] e.g. Cain and Rotella , 'Epidemics'; Troesken, 'Typhoid rates'.
[4] McKeown *Modern Rise.*
[5] e.g., see Williamson, *Coping with city growth;* Szreter, *Health and Wealth.*

airborne diseases.[6] After accounting for the latter factor Szreter argues that 'the classic sanitation diseases come to the fore' in explaining the mortality decline after 1850.[7]

The importance of government public health interventions in the early twentieth century is supported by evidence from other countries. Cain and Rotella, for example, estimate that a 1 per cent increase in sanitation expenditures would have led to close to a 3 per cent decline in the annual death rate in 48 American cities between 1899 and 1929.[8] Clean water technologies had a social rate of return that was 23 to 1 in major US cities in the early twentieth century.[9] Improvements to Chicago's water supply led to reduced mortality not only from waterborne disease, but also from several other causes of death including tuberculosis, pneumonia and kidney failure.[10] Several studies within the development literature also show significant effects of water improvements and sanitation access on health outcomes, particularly amongst infants.[11] However, the relative importance of infrastructure and better nutrition in increasing life expectancy remains unresolved.[12] Few studies assess several types of infrastructure spending together, and as a result cannot measure the overall importance of government's ability and willingness to invest in public infrastructure to achieving mortality decline.[13]

In this paper I analyze Britain's mortality decline through constructing and putting to use a new panel dataset identifying town-level infrastructure investment across England and Wales between

[6] e.g. Woods, 'Mortality patterns'; Szreter, *Health and Wealth.*
[7] Szreter *Health and Wealth*. p. 115.
[8] Cain and Rotella, 'Death and spending'.
[9] Cutler and Miller 'The role of public health'. See also Troesken, 'The limits of Jim Crow'; Kesztenbaum and Rosenthal, 'Sewers' diffusion'.
[10] Ferrie and Troesken, 'Water and Chicago'.
[11] e.g. Zwane and Kremer, 'What works'; Ahuja et al., 'Providing safe water'; Fink et al., 'Effect of water'; Zhang, 'Impact of water quality'.
[12] Fogel, *Escape from hunger*.
[13] Although see Alsan and Goldin 'Watersheds'.

1861 and 1900. During this period decisions over investment in public goods were made by local town councils, leading to great variation in the extent of investment across the country – variation which can be exploited for empirical analysis. Most of the investment that town councils undertook was focused on goods that improved the sanitary environment, including items such as street paving, public parks and sewer systems alongside clean water. In contrast to previous studies, I use data from a large number of districts, rather than relying on particular case studies[14] or using small samples of towns[15]. By combining this expenditure data with mortality information drawn from registration reports I am able to estimate the relative importance of spending by town councils in reducing mortality, accounting for changes in town wealth.

Several features of the particular historical setting facilitate identifying the overall impact of infrastructure investment. First, this period marked the very beginning of the public health movement, meaning that the counterfactual – of essentially no public investment – is very clear. Second, at this time responsibility for infrastructure investment fell almost exclusively on local governments, removing concerns that the data excludes spending by other authorities (such as different levels of government, or non-governmental organizations). Third, I can capture the combined impact of a broad range of infrastructure, rather than focusing on one particular type of investment (e.g. sewage systems).

The analysis proceeds in two steps. First, I use ordinary least squares specifications to establish the fact that infrastructure expenditure had a negative impact on overall mortality. Once demographic control variables or town fixed effects are included in the specifications, there is clear evidence that infrastructure investment led to significant declines in mortality rates. In particular,

[14] e.g. Woods, 'Mortality patterns'.
[15] Millward and Sheard, 'Urban fiscal problem'; Millward and Bell, 'Economic factors'.

infrastructure investment is estimated to explain approximately 30 per cent of the decline in mortality between 1861 and 1900.

While these results establish the effectiveness of infrastructure investment they may underestimate the magnitude of the effect. Expenditure on public goods was not random: towns would be more likely to invest where health problems were greatest. While the inclusion of fixed effects reduces this problem, it fails to account for reverse causality or expenditure undertaken to avoid anticipated increases in mortality.

To address this issue, I estimate two stage least squares regressions instrumenting for per capita expenditure on infrastructure using per capita expenditure in the previous decade. The results of the instrumental variables regressions show that infrastructure investment was the major contributor to urban mortality decline in the second half of the nineteenth century, with between 54 and 60 per cent of the decline in total mortality explained by infrastructure investment.

The use of a lagged independent variable as an instrument is problematic, with several reasons to question the validity of the exclusion restriction. As such I use a second weak instrument, expenditure on outrelief per pauper, to test the overidentification restrictrictions using the approach suggested by Hahn, Ham and Moon.[16] The results provide reassurance that the lagged level of infrastructure is a valid instrument. Second, as a placebo test I estimate the same specifications utilizing mortality from childbirth and (separately) violence as dependent variables. Change in childbirth mortality during this period was driven largely by improved medical understanding and this specification serves as a test of whether the results are capturing medical behavioral change (e.g. hand-washing) rather than infrastructure investment. Similarly, mortality from violence is a

[16] Hahn, Ham and Moon 'Hausman test'.

useful placebo since it includes deaths from industrial accidents and hence can capture potential confounding effects from industrial areas investing more in infrastructure. The results show no evidence of any statistically significant relationship between infrastructure investment and either variable.

Building on these results, I analyze the contribution of infrastructure investment to the decline in mortality from different types of disease. The largest effects are on waterborne diseases (cholera, diarrhea and typhoid), with infrastructure investment accounting for approximately 100 per cent of the decline in mortality from these diseases between 1871 and 1900. However, I also find significant evidence that infrastructure investment accounted for up to 30 per cent of the decline in mortality from airborne diseases. This suggests that public health investment had effects beyond the diseases most directly affected by sanitation, either through reducing transmission of disease (for instance through reducing overcrowding) or through strengthening immune systems.

Finally, using separate data for the 1871-1890 period I control for mortality trends in rural areas surrounding towns. By so doing, I treat these rural areas as a counterfactual for urban areas, and account for any district-specific time-varying factors such as weather patterns or improved medical understanding. The estimated effect of sanitation infrastructure is robust to this test and remains large and statistically significant. Further, as expected, there is no evidence of any relationship between infrastructure investment and mortality in the rural parts of districts.

Together, these estimates indicate that government spending on urban infrastructure was the major contributor to the mortality decline in England and Wales between 1861 and 1900. Government engagement in public health was crucial to overcoming the mortality penalty associated with urbanization. These findings are particularly striking if we consider that the

benefits to public health investments were by no means exhausted at this point in time. Even in 1914 not all urban households had access to piped water and it was not until the very end of the century that the benefits of water chlorination were recognized. Similarly programs of social housing and slum clearance were by no means fully developed until after 1900. Once these investments are properly accounted for, the longer run contribution of public works to urban mortality decline may have been even greater.

I

Britain became a much healthier place in the second half of the nineteenth century, with crude total mortality rates falling from 22 to 18 per 1,000 living between 1851 and 1900. Deaths from waterborne diseases such as cholera and diarrhea fell at an even faster rate, as shown in Figure 1. At the same time – as shown on the right hand axis of the figure – the level of spending on urban infrastructure increased dramatically, with the level of loans outstanding used to finance that investment increasing more than eight fold over the same period.

[Figure 1 here]

However, this overall picture of mortality decline and urban investment masks significant variation in the experience across different localities. While life expectancy increased across all major cities during the second half of the century, the extent of the increase differed considerably across different towns. This is illustrated by the two towns, Hull and Sunderland, highlighted in Figure 2. While both towns had similar life expectancy at birth in the decade 1861-1870 – if

anything slightly lower in Hull – by 1891-1900, life expectancy in Hull was three years higher than Sunderland.

[Figure 2 here]

The question for this paper is whether, and to what extent, these differences in mortality between towns were caused by different levels of sanitation investment. As suggestive evidence, in 1891-1900 Hull – where life expectancy rose sharply – spent an average of £6.6 per capita each year on sanitation public goods, while Sunderland – where life expectancy stagnated – spent only £3.2 per capita.[17]

[17] These figures relate to the Sunderland and Hull Registration Districts respectively, and are based on the dataset discussed in detail in the following subsection.

II

To answer this question comprehensively I construct a dataset that measures mortality and infrastructure investment across England and Wales in the second half of the nineteenth century. [18] Financial data are drawn from the *Local Taxation Returns* reported to Parliament and collected in the *Parliamentary Papers* collection. These reports detail the annual accounts of every town council – the bodies responsible for the vast majority of infrastructure investment. Data was collected for all (approximately 900) "urban sanitary authorities" for each year from 1867 to 1900. The accounts report the value of loans outstanding in each year, with the values disaggregated by type of expenditure from 1884 onwards. They also report the value of the rateable value of property in each town, which formed the tax base available to councils. I translate these nominal values into real values using the Rousseaux Price Index[19].

Data on cause of death in different districts are drawn from official statistics reported by the Registrar General for the period 1861-1900. The geographic unit of analysis is the registration district, of which there were approximately 630 across England and Wales. The primary source is a series of decennial reports digitized by Woods.[20] These reports are well known to both economic historians and demographers since they provide a wealth of data on both cause and age of death (in five or ten year intervals) in each district averaged by decade.[21]

I supplement the decennial data with information from the Quarterly Returns of the Registrar General regarding annual third quarter mortality data for the period 1871-1890. This source reports

[18] See Appendix 1 for additional details of the data sources used.
[19] Mitchell, *Abstract of British Historical Statistics*, pp. 723-4 following Millward and Sheard, 'Urban fiscal problem'.
[20] Woods, *Causes of death*.
[21] Examples of works using these sources include Szreter, *Health and Wealth*; Woods and Shelton, *Atlas of Victorian mortality;* and Beach and Hanlon 'Coal smoke'.

mortality statistics at a more disaggregated geographical level than the decennial data, and allows me to distinguish between mortality in urban and rural parts of registration districts (although at a lesser level of detail than in the decennial reports).[22] Unfortunately, collecting this data is complicated by the fact that sub-district information was only reported in quarterly, rather than annual, reports. To create a consistent time series, the third quarter was chosen for transcription since waterborne diseases, such as diarrhea, were particularly likely to strike during the summer months. As such, this period provides the best test of whether infrastructure had an effect – if it had no impact in the third quarter, it seems unlikely it would have made a substantial contribution in the remainder of the year.[23]

Unfortunately, town boundaries during this period did not match the boundaries of the registration districts (or sub-districts) for which mortality data was reported. Large towns comprised whole (and sometimes multiple) registration districts, while some registration districts included multiple smaller sanitary authorities. Given this issue, I link the financial and mortality data by first linking each town to the registration sub-district(s) in which it was situated using information reported in the 1881 census.[24] Where town boundaries crossed multiple registration districts (a relatively rare occurrence), town spending was allocated to each registration district according to the population residing in each district at the time of the census. Where multiple

[22] Specifically this information was reported at the level of registration sub-district which were the smaller administrative units underlying registration districts. There were approximately 2000 sub-districts in England and Wales.

[23] Across London districts the mean infant mortality rate consistently exceeded the level of the next highest quarter by 20 per cent and as a result levels of summer infant mortality were used by contemporaries as a measure of sanitary progress (Mooney 'Did London', p. 61).

[24] See Appendix 3 for detailed discussion of this procedure.

registration districts were combined in a single town (such as Liverpool and Manchester), I combine them into a single district for the purposes of the analysis.

This approach has the advantage of matching directly to the mortality information reported in the Registrar General's Decennial reports – and it is those reports that provide the most detailed disaggregation of mortality. Further, the boundaries of these districts were, in general, relatively stable between 1860 and 1900, allowing me to construct a panel dataset. Major boundary changes were largely limited to mergers or splits of sanitary districts; where this occurred I construct "synthetic" districts consisting of the larger, merged, district.

More difficulties arise when using the data on registration sub-districts to construct urban and rural mortality series, since there were frequent reallocations of boundaries within registration districts. To address this issue, when analyzing rural and urban mortality patterns, I adjust the mortality data for each year to consistent 1881 district boundaries. To do this, I first identified all sub-district boundary changes between 1871 and 1891 and then re-weighted the data to the 1881 district boundaries based on population weight.[25] As a result, analysis utilizing the sub-district data only covers the two decades between 1871 and 1890.

In addition, to construct the measure of outrelief per pauper used in the instrumental variables analysis, I use information on annual expenditure on outdoor relief in Poor Law Unions contained in the Local Taxation Returns, combined with a series of Parliamentary Papers reporting the number of paupers on January and July 1 each year. Finally, additional demographic data was collected from census reports.

[25] See Appendix 3 for further details.

Crude mortality rates The key dependent variables in the analysis are mortality rates disaggregated, in some specifications, by cause of death. Because each cross-section in our panel covers a decade – following the information reported in the decennial reports – the appropriate measure is the average death rate over the decade:

$$death\_rate\_causeI = \frac{number\ of\ deaths\ from\ cause\ I}{average\ population \times\ 10}$$

Infrastructure investment The key measure of urban infrastructure investment is the average level of loans outstanding per capita in each district over a decade. This variable is an accurate measure of the level of investment since nearly all town investments in infrastructure needed to be funded by borrowing. In 1902 on average over 95 per cent of the capital invested in trading entities (such as water and gas supply bodies) had been borrowed.[26] Furthermore, the stock of loans outstanding was seen as the single best measure of urban progress by contemporaries.[27]

Importantly for this paper, most of the loans that were taken out were dedicated to infrastructure that had a clear sanitary component. Most loans sanctioned by central government departments or obtained via local Acts of Parliament were associated with water, sanitation or street improvements[28] – all of which would improve the quality of the urban environment.[29] Turning to the actual stock of loans outstanding, after 1884 around one-quarter of towns' loans were devoted to each of water and sewer systems, with approximately a further 12 per cent used for spending on

[26] Author's calculation based on figures in *Report from the Joint Select Committee of the House of Lords and the House of Commons on Municipal Trading*, 1903 (270) VII.1.
[27] Wohl, *Endangered Lives,* p.112.
[28] Harris and Hinde, 'Local government and sanitary reform', Figures 3, 7 and 8.
[29] Millward and Sheard 'Urban fiscal problem'.

streets on average.[30] Others items of infrastructure which were not disaggregated included public parks, public baths and public housing, which could also have had an impact on reducing mortality.[31] There were also some spending items which would not have contributed to mortality declines including gas supply and, in larger towns after 1890, tram systems and electricity supply. Any concern that the measure partially captures infrastructure which does not have a clear sanitary impact should be balanced against the fact that not all urban spending on sanitation would be included in a measure of infrastructure. For instance, neither 'scavenging' (the process by which privy middens were emptied) nor cleaning of streets are included.

One issue with the loans data is that it is significantly right skewed, since a few towns spent an extremely high amount.[32] As a result, some observations have very high leverage in some specifications. These high leverage points are a particular concern since understanding the size of the effect (rather than just its direction) is an important goal of this paper. As such, I transform the loan stock per capita data using a square root transformation, and use the resulting variable as the main independent variable in the remainder of the paper. I also estimate a number of robustness tests to check that the results are not driven by outlying observations.

Other variables The second major independent variable captures the size of the urban tax base in each district since the taxes raised by local authorities – and used to repay loans – were property taxes (rather than, for instance, income taxes). As such, I use this variable as a proxy for urban

[30] Detailed disaggregated information is not available before 1884, and so I use the total stock of loans outstanding throughout the analysis. More detailed information on the percentage of loans devoted to different purposes is presented in Appendix 4.
[31] Public housing in particular would be likely to have a significant impact but was only a very small part of spending until the end of the nineteenth century. Public baths were seen by sanitary reformers as a relatively cheap way to improve public health (Sheard, 'Profit is a dirty word').
[32] See Figure A.III in Appendix 4.

wealth. As with the level of the loan stock per capita, I apply a square root transformation to this variable. As detailed below, I also control for several demographic characteristics: fuller details of the construction of these variables are presented in Appendix 2.

III

The data is used to construct a four-period panel dataset, where each cross-section relates to a decade reported in the decennial reports of the Registrar General: 1861-1870, 1871-1880, 1881-1890 and 1891-1900.[33] I then estimate the effect of infrastructure investment on deaths using the specifications of the following form:

$$death\_rate_{i,t} = \alpha + \beta InfrastructureInvestmentPC_{i,t} + \gamma X_{i,t} + \delta_0 Z_i + \delta_1 T + \varepsilon_{i.t} \quad (1)$$

where *i* indexes registration districts and *t* indexes each decade. The variable *death_rate* measures the number of deaths per capita, and *InfrastructureInvestmentPC* is the per capita level of urban infrastructure investment in each district – measured by the square-rooted per capita stock of loans outstanding. *X* is a vector of control variables, Z_i includes district fixed effects, *T* is a vector of decade fixed effects, and ε is an error term. The basic set of control variables includes the decadal average population in the districts, the percentage of population female, the percentage aged under 15 and the percentage aged 15-45.[34] As a proxy for district wealth, I control for the urban tax base per capita in the district (also square rooted). I run an additional set of tests using as a dependent variable third quarter mortality in the urban portion of districts for the period 1871-1890. Specifically, I include rural mortality as an additional control variable in each district and, by so doing check that the results are not driven by time varying factors that are common across a whole district – for instance, weather, or improved hygiene.

[33] Descriptive statistics of the variables used in the regressions are presented in Appendix 4.

[34] In additional specifications presented in the Online Appendix I also include controls for population density (excluded from the main specification due to concerns of multicollinearity), the population in the largest town in the district, the district population squared, the percentage urban, and mortality from childbirth and violence (separately).

Identifying the effects of mortality change is complicated by the endogeneity in the location of infrastructure. Towns did not spend their resources at random, and were likely to increase infrastructure investment in response to the disease environment. The effect of this reverse causality could be to mask any beneficial results of infrastructure expenditure on mortality – a hypothesis supported by results below showing a positive correlation between higher spending and higher mortality.

I take two approaches to isolate the causal effect of infrastructure investment on mortality. First, I estimate specifications including district fixed effects. By doing so I account for time-invariant factors, such as location, that affect both the level of mortality and the level of spending. While this approach accounts for many potential sources of endogeneity, it does not address any endogeneity resulting from reverse causality within a decade – for instance, if towns responded to high mortality by building more infrastructure. In fact, official statistics highlighting towns with high death rates were used to push for sanitary reform, through both the "league tables" published by the General Registration Office[35] and through local press reports[36]. Even more problematic, it cannot account for the fact that towns may have acted to forestall *expected* increases in mortality through building additional infrastructure.

To provide a better indication of the likely upper bound of this effect we need a variable correlated with infrastructure investment but otherwise uncorrelated with mortality rates. Such a variable is, unfortunately, hard to find. However we can identify two possible instruments: the lagged level of loans outstanding per capita (square rooted) and the level of expenditure on poor relief on outdoor paupers ("outrelief") in the registration district. For reasons outlined below,

[35] Szreter, *Health and Wealth*, p. 259.
[36] Lewes, 'The GRO', p. 485.

neither of these is a perfect instrument, however together they provide a way to estimate the extent to which the fixed effects regressions may underestimate the impact of infrastructure investment.

The lagged level of loans outstanding is clearly related to the current level of infrastructure. However, lagged independent variables are problematic instruments since there are several plausible threats to the exclusion restriction—i.e., the requirement that the level of infrastructure in the previous period is not related to the level of mortality in the current period, except through its effect on the infrastructure in the current period.[37] This assumption is plausible in that the main effect of improved infrastructure would have been to prevent individuals catching the diseases that would eventually kill them. However, if endogoneity is an issue—so that the fixed effects estimates are biased downward—then historic spending may well be correlated with mortality shocks in the previous period. If the error term is serially correlated—and implementing a simple test[38] suggests that it is— then historic investment will be correlated with spending in the current period.[39]

A second potential concern is that the benefits of infrastructure investment may directly affect mortality in the following decade. Since surviving a disease could lead to weaker immune systems, if infrastructure investment reduced the chances of individuals catching a disease at all (i.e. lowering morbidity as well as mortality) then it could increase the ability of a population to fight disease in the future. Is this a major concern? Ferrie and Troesken find that in Chicago survivors from typhoid were more likely to die from other diseases in the following years, suggesting there

[37] See Bellemare et al. 'Lagged explanatory variables' for a discussion of the issues associated with using lagged independent variables as instruments.
[38] Specifically, I use the test suggested by Wooldridge, *Econometric Analysis (1st edition)*, pp. 274-276.
[39] Historic investment could also affect current mortality through changing the age distribution of the town population and hence reducing the proportion. To address this concern in the instrumental variables specifications I control for the lagged age structure.

were some follow on effects. However, they find that other diseases did not have the same effects, and no 'evidence that lagged typhoid rates of greater than 1 or 2 years had any systematic effect'. As such, there is little reason to think that these effects will lead to considerable bias.[40]

With these concerns in mind we turn to the second potential instrument: the level of out relief expenditure per pauper. This variable relates to the level of support for the destitute (the paupers) and may be related to spending on public goods through capturing support for public spending within the district.[41] However, it is plausibly exogenous since spending on the poor law was not controlled by the town councils but by a separate authority (the poor law guardians) that governed individual poor law unions—the boundaries of which were the same as registration districts. While there is a potential channel to mortality through improved nutrition, this is likely to be very small since relatively few citizens received out relief at all: the stock of outdoor paupers per capita was, on average, only around 3 per cent of the population in this period.

Unfortunately the instrument relating to poor law expenditure suffers from the weak instruments problem: the Kleibergen-Papp statistic (a robust version of the F-statistic) is less than five in all specifications. As such, standard errors on the two stage least squares estimates may be inflated. Further, even a small violation of the exclusion restriction may lead to severe bias in the two stage least squares estimate. We are thus faced with the problem of a single strong instrument with threats to exogoneity and a second instrument that is more plausibly exogenous but also weak. This econometric issue is examined by Hahn, Ham and Moon: following their guidance I estimate the instrumental variables estimates including utilizing only the lagged level of infrastructure

[40] Ferrie and Troesken, 'Water and Chicago' pp. 9-11.
[41] I exclude expenditure on paupers in workhouses to avoid issues associated with the large fixed costs of building and maintaining those institutions.

investment (the strong instrument) as an instrument, but use the outrelief per pauper (the weak instrument) to carry out overidentification tests using a version of the Hausman test that is robust to weak instruments.[42]

To measure the *relative* importance of infrastructure, I compare the estimated effects to the overall mortality decline across the period of study. Specifically, I use the regression results to estimate the reduction in the mortality rate in each district explained by town spending on infrastructure in 1891-1900. I then take an average of this effect, weighted by district population, and compare it to the (weighted) average actual decline that occurred in these districts.

Denoting the measure of infrastructure investment in district *i* in the decade ending in year *t* as $I_{i,t}$ (i.e., in this paper the square root of loans per capita outstanding), the estimated regression coefficient $\hat{\beta}$, the mortality rate as $M_{i,t}$, and the district population as $P_{i,t}$ the magnitude of the effect is then estimated as:

$$Magnitude = \frac{\sum_i P_{i,t}\hat{\beta}(I_{i,t})}{\sum_i P_{i,t}(M_{i,t} - M_{i,1870})} \quad (2)$$

where *t* refers to the decade at the end point of the period under consideration (either 1881-1890 or 1891-1900), depending on the specification), and 1870 refers to the decade 1861-70.

42 Hahn, Ham and Moon 'Hausman test'. The use of the Hausman statistic developed by Hahn, Ham and Moon is recommended in Bazzi and Clemens 'Blunt instruments', and I use their implementation of the statistic.

IV

Table 1 displays the results of six specifications analyzing the relationship between infrastructure investment (measured by the square rooted per capita stock of loans outstanding) and the total mortality rate. The mortality rate, the level of investment, the tax base and district population are standardized and so the associated coefficients should be interpreted as the effect of a one standard deviation increase in the relevant variable in terms of standard deviations of the mortality rate.

The first specification includes only infrastructure investment per capita as an independent variable. There is evidence of a statistically significant *positive* relationship between infrastructure investment per capita and the mortality rate. The likely explanation for this is that towns with higher mortality invested more as a reaction to the disease environment.

[Table 1 here]

However this relationship changes sign once other town characteristics are accounted for. Once time and year fixed effects (specification (2)) are included there is statistically significant evidence that higher investment led to lower mortality. The negative relationship is robust to the inclusion of control variables (specification (3)) and excluding the final decade, when towns began major investments in infrastructure less associated with sanitary improvements (specification (4)). The fact that the results are similar suggests that that any difference in the composition of infrastructure in this decade is not affecting the results significantly. Interestingly, there is no evidence that mortality was affected by changes in the town tax base per capita (a proxy for wealth): both specification 3 and 4 show statistically insignificant and close to zero effects of this variable.

Together these results provide clear evidence that greater spending on urban infrastructure led to lower mortality. They also show that the effect was large: approximately 30 per cent of the mortality decline is estimated to have resulted from investment in infrastructure. Further, as discussed in the next sub-section, this is likely to be an underestimation of the effect.

The remaining two specifications explore the robustness of the finding to the inclusion of allowing time trends to differ across groups of towns. Specification (5) allows for geographically-focused trends by including linear time trends for each county. By doing so we capture differences driven by reactions to local climactic conditions or trends driven by similarities in districts near each other—for instance due to similar industrial structures which could be correlated with both the level of expenditure and mortality decline. In specification (6) I allow for differing time trends according to population density in 1871. These trends allows for, example, the fact that dense districts may have invested in infrastructure but also have reacted to high mortality rates in other ways—in which case we may falsely attribute the decline in mortality to the investment in infrastructure.

The finding of a negative effect of infrastructure remains strongly negative after allowing for both these different trends. Results in the Online Appendix show similar effects after allowing for differing trends according to town population and rateable value per capita. The estimated magnitude of the effect is slightly smaller, but this is not surprising since the inclusion of these trends reduces the variation available to estimate the effect of infrastructure investment, which can in turn cause attenuation bias. As such, both of these specifications provide a large degree of reassurance in the fixed effects estimates.

Some readers may be concerned that the results are driven by either the selection of control variables or by the effects of a few influential observations. To address these concerns I present the results of a number of additional robustness tests in the Online Appendix. I examine the effects of varying the group of control variables, including replacing the district population measure with a measure of population density or the largest town population. I also include a specification including mortality from other, specific causes (namely childbirth and violent deaths) as control variables in order to capture the effects of any factor causing a general downward trend in mortality.

In additional robustness tests, I estimate several specifications to check the results are not driven by the presence of outliers. First I split the sample according to the level of loans outstanding in the first decade and then, separately, the final decade. I also re-estimate specification (3) removing the top 5 per cent and bottom 5 per cent percent of towns according to 1871 population density, 1861-70 population, and 1861-70 total mortality. The estimated coefficient ranges from -0.14 to -0.20 in these specifications and is strongly statistically significant in all cases. Finally I estimate a median estimator[43]: the estimated coefficient in this case is -0.17 and is again, strongly statistically significant.

Together these results provide strong evidence that infrastructure investment led to significant declines in mortality. The inclusion of fixed effects removes a large degree of the potential endogeneity in investment decisions, by accounting for time-invariant factors that could affect both mortality and the decision to invest. However, as argued above, there may be other forms of

[43] Specifically, I use the estimator suggested by Wooldridge *Econometric Analysis (2nd ed.)* p. 461.

bias – such as pre-emptive investment in infrastructure – that mean that these specifications do not capture the full contribution of infrastructure to mortality decline.

To address these issues I estimate two stage least squares specifications using the lagged level of infrastructure as the exogenous instrument. As discussed above, there are a number of threats to the exclusion restriction when using this instrument; as such I report two overidentification tests using the second, weak, instrument of outrelief per pauper: the usual Hansen J test and the Hahn, Ham, and Moon weak-instrument-robust Hausman test discussed in Section 3.1 (Hausman WIV in the table below).

Table 2 presents the results of the two stage least squares estimations, with Panels A and B displaying the second and first stage results, respectively. Since the instrument is the lagged value of the loans outstanding, these specifications consist of three periods only (in contrast to four in Table 1); thus for comparison I also include fixed effects regressions for the same periods. The estimated effects from these specifications indicate infrastructure investment can explain between 18 per cent and 29 per cent of the mortality decline – similar in magnitude to the corresponding specifications estimated over the entire period in Table 1.

[Table 2 here]

As expected, there is a strongly significant positive relationship between the instrument and the current level of infrastructure investment. Additional tests, presented at the bottom of Table 2 confirm the validity of the instrumental variables approach. The C-statistic, which tests for endogeneity of infrastructure investment, is strongly statistically significant in all specifications.

Both overidentification tests show very little evidence that the overidentifying restriction is violated except, possibly, when county time trends are included.[44]

Columns (1) and (2) present the estimates from the fixed effects and two stage least squares regressions with no control variables, while (3) and (4) present the results including the control variables. Mirroring the penultimate specification in Table 1, in columns (5) and (6) I include county time trends. In all specifications, the estimated coefficient on the measure of infrastructure investment is negative and strongly statistically significant. Further the effect sizes are large with the instrumental variables estimates indicating that infrastructure investment accounted for between 45 per cent and 60 per cent of the decline in urban mortality between 1861 and 1900. After accounting for endogeneity, therefore, spending on infrastructure appears to be the major force behind Britain's urban mortality decline.

V

Having established the importance of sanitation investment in reducing total mortality, we can examine in more detail the specific causes of deaths that expenditure affected. Since the infrastructure investments in question were largely associated with sanitary improvements, the primary causes of death affected are likely to be waterborne diseases such as cholera (although this accounted for only a few deaths during this period), diarrhea and typhoid. However, there could also have been effects on other forms of mortality. Poor living conditions—particularly poorly ventilated housing—are today considered a major risk factor for the spread of tuberculosis[45]

[44] The relatively low p-value in this instance is likely to reflect the low correlation between the weak instrument and the errors in the second stage specification once county time trends are accounted for. This explanation is supported by the high coefficient on the infrastructure investment when only the weak instrument is included: see the Online Appendix.

[45] Lönnroth et al., 'Drivers of tuberculosis'.

and Szreter argues that infrastructure investment may have reduced nineteenth-century mortality from airborne disease through reducing overcrowding and hence the spread of disease[46]. Empirical evidence for this type of spillover is limited, but Watson finds that sanitation investment led to reductions in infectious respiratory disease amongst infants in US Indian reservations, a result she explains through improved cleanliness and behavioral change associated with sanitation investment.[47] Alternatively, Ferrie and Troesken argue that sanitation can reduce mortality from non-waterborne disease since typhoid survivors are more likely to die from other causes—a phenomenon much discussed by contemporaries, quoting one contemporary expert: 'it is said … two-thirds of the deaths [from typhoid] are due to the numerous complications, among which tuberculosis and pneumonia are prominent'.[48]

Table 3 explores these hypotheses through re-estimating the fixed effects and instrumental variables specifications, but using mortality by various different causes of death as the dependent variable. The first two specifications examine mortality from three major waterborne diseases, cholera, diarrhea and typhoid. These diseases would be directly affected by sanitation investment and so we might affect particularly sizable effects in these specifications. Unfortunately, however, typhoid was not distinguished in the Registrar General's reports until the decade 1871-1880, and so the estimated decline in mortality is measured over the period after 1871.[49]

Specifications (3) and (4) analyze a group of airborne diseases including 'diseases of the respiratory system' (such as bronchitis), pulmonary tuberculosis, smallpox, scarlet fever,

[46] Szreter, *Health and Wealth.*
[47] Watson, 'Public health investments'.
[48] Whipple, *Typhoid Fever*, p. 6 quoted in Ferrie and Troesken 'Water and Chicago', p7.
[49] Appendix A1 discusses the changes in nosology of disease during the period. Additional specifications using alternative measures for waterborne mortality as the dependent variable are included in the Online Appendix.

whooping cough, measles and diphtheria. Specifications (5) – (8) then carry out placebo tests using as the dependent variable mortality from two causes, childbirth and violence, that would not be expected to be affected by investments in urban infrastructure.

[Table 3 here]

The results show strong and statistically significant effects of infrastructure spending on mortality from both waterborne and airborne diseases. The instrumental variables specifications indicate that mortality from waterborne diseases was reduced by approximately 100 per cent by infrastructure investment – that is, it would have increased in the absence of public health expenditure. There is also evidence that urban infrastructure contributed significantly to the reduction in airborne disease, accounting for between 16 and 30 per cent of the decline between 1861 and 1900. It is interesting – and reassuring – that, in comparison to the waterborne disease estimates, the instrumental variables estimate is closer to the OLS specification. Such a finding is consistent with the argument that the fixed effects estimates are biased downwards because a reaction to those (waterborne) diseases directly affected by sanitation infrastructure; hence it is intuitive that the effect would be smaller in the case of airborne diseases.

Disaggregating mortality by cause of death also provides a valuable placebo test to check that the instrumental variables estimates are not incorrectly capturing other factors associated with both lower mortality and higher infrastructure investment. Specifications (5) and (6) use mortality in childbirth (including puerperal fever) as the dependent variable.[50] This provides a good test of whether the effects we find are causal, since the major contributor to decline in maternal mortality

[50] Mortality from childbirth is measured as deaths from childbirth divided by the population aged 15-44 mortality rate, adjusted by the estimated female population at all ages (sex-specific population rates are not available for all decades).

was most likely improved medical knowledge, rather than an improved sanitary environment.[51] As such, finding a negative coefficient in these specifications would cause concern that the measure of infrastructure investment is still capturing the effects of broader improvements in medical understanding. Similarly, in specifications (7) and (8) the dependent variable is mortality from 'violence'. The major component of deaths in this category relates to accidents, including industrial accidents and also a large number of deaths in early childhood from, for instance, suffocation in bedclothes. As such it serves as a useful placebo since it could capture improved healthcare and changes in industrial structure leading to a reduction in industrial accidents. It may also capture the effect of increasing wealth, since the category also includes mortality from homicide and suicide, which tend to be positively correlated with poverty[52]. Further, in contrast to childbirth mortality, mortality from violence decreased significantly over the period, making it more likely that it was affected by the time-varying factors that may also have affected mortality from other causes. There is no evidence of such an effect in either the fixed effects or the instrumental variables regressions.

Together these results provide evidence that urban investment had important effects in reducing mortality from both airborne and waterborne disease. However, the precise estimates should be treated with some caution since additional specifications (reported in the Online Appendix) indicate that the results are more sensitive to the exclusion of outliers than those in Tables 1 and 2. In particular, there is no evidence of an effect on waterborne diseases when a median estimator is used, while the two stage least squares estimate for airborne diseases is no longer statistically significant once county time trends are allowed for. Further, the bottom panel of Table 3 shows

[51] Loudon, 'Maternal mortality'.
[52] Pratt and Cullen, 'Assessing macro-level predictors'.

that both overidentification tests reject the validity of the exclusion restriction for both airborne and waterborne disease: a result of the fact that when only the weak instrument is used the coefficient on the infrastructure investment variable becomes very large and, in the case of the waterborne mortality variable, changes sign. Together, these robustness tests indicate a degree of noisiness in the cause-specific estimates that creates some instability in the results.

VI

One remaining concern could be that the measure of infrastructure investment is picking up something more general about 'urban' versus 'rural' areas. The registration districts we have analyzed were, in many cases, comprised of both rural and urban portions. An increase in spending per capita could therefore result from the spread of urban areas across registration districts. Thus the estimated effects of infrastructure investment could be capturing other factors associated with urbanization such as better education or understanding of disease transmission.

To address this concern, in this subsection I use data at the registration sub-district level to distinguish the urban and rural parts of registration districts for the period 1871-1890.[53] The urban parts of sub-districts are those that contained an urban area in 1881, while rural parts are those sub-districts that contained no urban area at all in 1881. I then use data regarding mortality in the third quarter from cholera, diarrhea and fever (which would include both typhoid and other forms of fever).[54]

53 As discussed previously, difficulties in creating consistent district boundaries at the sub-district level precluded carrying out this analysis over a longer time period.

[54] The Online Appendix presents similar specifications using total mortality as the dependent variable. The results are similar, except that the relationship with infrastructure investment is statistically insignificant for districts that were partly rural when control variables are included. This is likely to reflect two factors. First, the third quarter data is noisier than the annual data since it relates to a smaller sample and because of the need to adjust for boundary changes.

I carry out three tests of the effects of urban infrastructure using this data. First, I check that there is evidence that infrastructure led to a decline in mortality when focusing only on mortality in urban sub-districts. Second, I include rural mortality as a control variable in these specifications, as a check that the results are not spuriously capturing other time-varying factors such as local weather patterns that affect mortality. Third, I carry out a placebo test with mortality in rural areas as the dependent variable, checking whether greater infrastructure spending is capturing any effects that affected the broader area of a district. This latter test rules out a situation where everyone in a district becomes better informed about disease, leading to lower mortality and higher spending in urban areas.

The results, displayed in Table 4, again show consistent evidence of infrastructure investment on waterborne mortality in urban areas. Specification (1) displays the estimates for all registration districts, while specification (2) includes only districts with both urban and rural areas. Specification (3) shows that the estimated effect is essentially unchanged when controlling for mortality in rural areas. Finally, specifications (4) and (5) use rural mortality as the dependent variable, and show that there is no evidence that infrastructure spending affected mortality in those areas whether control variables are included.

[Table 4 here]

These results provide strong evidence that the effects we are capturing for urban infrastructure investment relate directly to mortality in urban portions of the registration districts. There is no

Second, many of the forms of mortality that had the largest decline, such as tuberculosis, had a lower "base" mortality in the summer months (Fares et al., 'Seasonality').

evidence that we are capturing any effect that would affect mortality in both towns and their rural environs.

VII

This paper has tested the effects of government spending on sanitation infrastructure on mortality rates from waterborne disease in England between 1861 and 1900. During this period local government took responsibility for improving urban environments, leading to rapid growth in expenditure on public goods such as clean water supply, sewer systems and street paving and cleaning. Using a new panel dataset, I estimate that this investment was responsible for between 18 per cent and 35 per cent of the mortality decline during this period. Given the endogoneity in the location of expenditure these estimates can be seen as a lower bound on the effect, and instrumental variables estimates identify a much large effect of up to 60 per cent of mortality decline. These estimates are based on using the lagged independent variable as an instrument, meaning that there are a number of potential threats to the exclusion restriction. As such, we should be cautious in placing significant emphasis on this precise figure. However the validity of the estimates is supported by both tests of the overidentifying restrictions and placebo tests and, moreover, there is no strong reason to think that any bias means that the effects of investment are overestimated: the true effect may have been even larger. Government involvement in public health thus emerges as the single most important factor in reducing urban mortality in this period. Nor were the benefits limited to classic "sanitation diseases", with investment in infrastructure also associated with a decline in mortality from airborne diseases.

Together, these results support an explanation of Britain's mortality decline from around 1870 onwards based predominantly around the provision of public infrastructure, rather than nutrition. Further, they suggest that in estimating the potential benefits of public investment, we should be careful to properly account for the wide range of investments that can improve health outcomes.

Some of these investments may offer less clear cut causal mechanisms than, for instance, a new water filtration plant. However they may nevertheless offer important cumulative benefits that significantly improve urban environments and hence life expectancy – for instance through reducing overcrowding and hence the spread of airborne disease. Future research will look at disaggregating the role of these different types of infrastructure in great detail.

Appendix

Appendix 1: Data sources

1. Mortality data

Mortality data reported at registration district level are drawn from the decennial reports of the Registrar General for 1861-1900, which report the annual average number of deaths by cause and by age group split by registration district.[55] Mortality data at registration sub-district level were collected from the Quarterly Returns of the Registrar General for the third quarter of each year between 1871 and 1890, with the exception of 1880 and 1882 (information for the years 1871 and 1881 was drawn from existing datasets[56]).

The Quarterly Reports during this period are less detailed than the decennial reports, but do contain information on the total number of deaths, and deaths from nine causes: smallpox, measles, scarlet fever, cholera, diarrhea, violence, whooping cough, diphtheria and fever (including a range of causes such as typhoid, simple continued fever and puerperal fever). The reports, however, do not detail a set of important causes of death such as tuberculosis and so it is not possible to construct a measure of mortality from airborne diseases.

The Registrar General attempted to enforce a consistent nosology on registration officials around the country, and in general we can consider the categories of individual diseases as reasonably accurate. However, there are some exceptions to this general rule. The most major relates to typhoid which was not distinguished at all from typhus – a disease with similar symptoms but that is not waterborne – until 1869, and not in the decennial reports until 1871-1880. A second

[55] These data were obtained from Woods, *Causes of death*.
[56] Southall and Gilbert, *Vital Statistics.*

potential issue is that relatively substantial revisions were made to the nosology used in 1881. Fortunately for the purposes of this paper, most of the changes were relatively minor categories or were later reported as separate categories allowing the original classification to be reconstructed (See Registrar General, 1895, Table H).

2. Financial data

Information regarding expenditures on infrastructure are drawn from the *Local Taxation Returns* reported to Parliament and collected in the *Parliamentary Papers* collection. Data is collected for all "urban sanitary authorities" for each year from 1867 to 1900. Prior to 1872 the accounts are reported under the titles of Local Boards of Health and Improvement Commissions – the bodies which were renamed Urban Sanitary Authorities in the 1872 Public Health Act. This includes approximately 900 towns, granted standardized expenditure powers under the terms of the 1872 Public Health Act.

Information regarding expenditures on poor relief was reported in a separate set of accounts for 1861-1870 in the *Local Taxation Returns.*

3. Population, paupers and area data

Information regarding town and district population was drawn from decennial census reports. Town-level data was collected from the original census reports, while registration district

data was drawn from existing datasets.[57] Data on area and population for 1871 and 1891 were taken from parish-level census statistics[58].

The number of paupers in each poor law union was reported biannually (on January 1 and July 1) in a series of *Parliamentary Papers* between 1861 and 1900.

[57] The 1871 and 1891 information was aggregated from the parish-level information reported in Southall et al. *Census Data*. 1881 population was obtained from the 100 per cent census sample hosted by the North Atlantic Population Project (www.nappdata.org). 1901 population was downloaded from the Integrated Census Microdata project at the UK data archive; see Schürer and Higgs *Integrated Census Microdata.*

[58] Data from Southall et al. *Census Data.*

Appendix 2: Variable Definition

1. Mortality rates

Mortality rates are calculated as the average number of deaths in a decade divided by the average town population, multiplied by 10,000. The decennial registration district reports also detail average decadal district population. Average populations are not available for the registration sub-districts, so I estimate an average population using geometric interpolation between censuses. The average of this interpolated population then serves as the denominator of the measure.

Mortality data for deaths in registration sub-districts was reported by quarter: I use the third quarter in each year. The numerator of the measure is the average number of deaths in the sub-districts for years for which data is available.

2. Financial variables

Financial variables, including loans outstanding and rateable value, are calculated as decennial averages using the town-level dataset. The average annual total over the decade for each town was first estimated by averaging over years for which data is available. Amounts were then allocated to each registration district (as explained in Appendix 3) and per capita variables were calculated using the average district population in the decade. District-level estimates were calculated by aggregating the town-level spending allocated to the district.

In most cases towns had data for loans outstanding available in all years after 1867. Rateable value on the other hand was missing for some years between 1866 and 1870, and for 1871. In some cases, no data was available for the 1860s; in this case I use the first year in which data was available as the data point for 1861-70.

The number of outdoor paupers in each year is estimated by adding the January figure to one half of the figure for the previous July and one half the figure for the following July, to match the financial year variable. Outrelief per pauper is then calculated by dividing the expenditure on outrelief in a district by the number of outdoor paupers in each year and a decadal average is then calculated by averaging across the relevant decade.

3. Demographic variables

Demographic variables were defined as follows:

- Average district population: reported in the Registrar General's decennial reports.
- Average urban and rural population: calculated using geometric interpolation between census years.
- Percentage of population by age group: calculated using information from the Registrar General's decennial reports. Age groups were reported by half-decade or decade: the three aggregated categories used in the paper were constructed based on the correlations between these groups.
- Percentage of population female: for 1861-1870, 1881-1890 and 1891-1900 calculated as average female population/average total population using the Registrar General's decennial reports. For 1871-1880 a breakdown by sex was not provided; instead the average of the census estimates for 1871 and 1881 is used.
- Population density: Total district population divided by district area. Estimated using population data for the census year at the end of each decade. For the 1871 and 1881 estimates, the area is taken from the 1871 census; for the 1881, 1891 and 1901 estimates it is taken from the 1891 census.

Appendix 3: Boundary changes and linking towns to registration districts

Each town is linked to a registration sub-district using information reported in the 1881 census, Vol II. This report splits the population of each town according to sub-district. For example, of a total town population of 10,000 it identifies that 4,000 lived in sub-district A, 3,500 in sub-district B, and 2,500 in sub-district C. To aggregate expenditure data at the level of registration district, expenditure is allocated to each district proportionally to the portion of the town population that falls in each district. That is, if 85 per cent of the town live in district X in 1881, and 15 per cent in district Y, then 85 per cent of town expenditure is assigned to district X and 15 per cent to district Y (for all decades). Registration district level expenditure is then calculated through summing the spending amounts for parts of towns within the district.

A further complication is that the registration district boundaries changed over time. To account for this, in analyses focusing on registration sub-districts I adjust all sub-district mortality data to the 1881 boundaries by first identifying all sub-district boundary changes (using the reports of the Registrar General) and then creating a synthetic district based on population weight. That is, deaths in each year were reassigned to the 1881 district based on the population of the actual district reporting that lived in the 1881 district boundary in 1881. For instance, if two equally sized districts merged in 1885, mortality data from the new district after this point would be split evenly between the two synthetic districts.

Appendix 4: Descriptive statistics

Table A1 presents summary statistics of the main variables. Figure A1 displays the density of the loans outstanding per capita variable before and after the square root transformation. Figure A2 displays the trends in mortality from the causes of death analyzed in the specification in Table 3.

[Table A1 here]

[Figure A1 here]

[Figure A2 here]

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Table 1: Infrastructure investment is associated with large declines in total mortality between 1861 and 1900 after accounting for time invariant town characteristics.

	DV = Total mortality rate (all ages, standardized coefficients)					
	(1)	(2)	(3)	(4)	(5)	(6)
Infrastructure investment p.c.	0.07**	-0.21***	-0.17***	-0.16***	-0.12***	-0.12***
	(0.036)	(0.027)	(0.031)	(0.036)	(0.028)	(0.029)
Tax base p.c.			-0.00	-0.01	-0.01	0.03
			(0.038)	(0.040)	(0.037)	(0.039)
District population			-0.16**	-0.23**	-0.12*	-0.06
			(0.076)	(0.090)	(0.068)	(0.071)
% population female			0.06***	0.07***	0.04**	0.05**
			(0.019)	(0.023)	(0.020)	(0.020)
% population age under 15			-0.06***	-0.06***	-0.06***	-0.05***
			(0.017)	(0.020)	(0.017)	(0.017)
% population age 15-44			-0.01	0.01	-0.01	-0.02
			(0.016)	(0.019)	(0.015)	(0.016)
Reg Dist FE	N	Y	Y	Y	Y	Y
Decade FE	N	Y	Y	Y	Y	Y
Time trends	N	N	N	N	County	Pop density
% decline explained	0	35	29	28	21	20
Observations	1520	1520	1520	1140	1520	1520
No. Districts	380	380	380	380	380	380

All coefficients are standardized. Observations are 'registration district decades', between 1861-1870 and 1891-1900. *Infrastructure investment p.c.* is the square root of average stock of loans outstanding over the decade divided by average district population. *Tax base p.c.* is the square root of average per capita rateable value of property. '% decline explained' is the estimated reduction in mortality explained by the level of infrastructure investment as a percentage of the total decline in mortality from 1861-1900. Specification (4) excludes the decade 1891-1900.
Standard errors are clustered by registration district, and are displayed in parentheses.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 2: Instrumental variable regressions show that infrastructure investment explained more than half of the urban mortality decline between 1861 and 1900.

	DV = Total mortality rate (all ages, standardized coefficients)					
	(1)	(2)	(3)	(4)	(5)	(6)
	FE	IV	FE	IV	FE	IV
Panel A: Fixed effects and second stage results from 2SLS specifications						
Infrastructure investment p.c.	-0.17***	-0.32***	-0.15***	-0.36***	-0.11***	-0.27***
	(0.030)	(0.048)	(0.038)	(0.070)	(0.031)	(0.085)
Tax base p.c.			0.11*	0.23***	0.03	0.14*
			(0.057)	(0.068)	(0.051)	(0.074)
Panel B: Abbreviated first stage regressions for infrastructure investment per capita						
Lag Infrastructure investment		0.56***		0.42***		0.31***
p.c.		(0.033)		(0.038)		(0.037)
Tax base p.c.				0.44***		0.51***
				(0.052)		(0.054)
Controls	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y
Time trends	N	N	N	N	County	County
% decline explained	29	54	26	60	18	45
Kleibergen-Papp Stat		289		119		73
Hansen C p-value		0.000		0.002		0.047
Hansen J p-value		0.791		0.901		0.118
Hausman WIV p-value		0.775		0.893		0.110
Observations	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380

Panel A includes fixed effects (specifications 1, 3, and 5) and second stage estimates from the two stage least squares regressions using the lagged level of infrastructure investment as an instrument for current infrastructure investment. All coefficients are standardized. Observations are "registration district decades", between 1871-1880 and 1891-1900. *Infrastructure investment p.c.* is the square root of the average stock of loans outstanding over the decade divided by average district population. *Tax base p.c.* is the square root of the average per capita rateable value of property. Control variables include district population, percentage of population female, the lagged percentage aged under 15, and the lagged percentage aged 15-44; see the Online Appendix for full results. '% decline explained' is the estimated reduction in mortality explained by the level of infrastructure investment as a percentage of the total decline in mortality from 1861-1900.

The Hansen C-value is a test of endogoneity, estimated by comparing the Sargan-Hansen statistics from the specification in which infrastructure investment variable is treated as exogenous and where it is treated as endogenous. The Hansen J and Hausman WIV p-values are tests of overidentification when including outrelief expenditure per pauper as a second instrument. The Hansen J test is the usual test for overidentification, while the Hausman WIV test is robust to the inclusion of weak instruments discussed in Section III. In both cases, the null hypothesis is that the overidentifying restrictions are valid.

Standard errors are clustered by registration district, and are displayed in parentheses.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 3: Infrastructure spending caused significant decline in mortality from both waterborne and airborne diseases, but does not have any effect in placebo tests with mortality from childbirth or violence as a dependent variable.

	DV = Mortality rate at all ages by cause							
	Waterborne		Airborne		Childbirth		Violence	
	FE	IV	FE	IV	FE	IV	FE	IV
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Infrastructure investment p.c.	-0.17***	-0.36***	-0.10***	-0.19**	-0.08	-0.02	-0.04	-0.14
	(0.045)	(0.090)	(0.037)	(0.076)	(0.068)	(0.123)	(0.072)	(0.120)
Tax base p.c.	0.15**	0.28***	-0.04	0.01	0.04	-0.00	0.09	0.15
	(0.075)	(0.092)	(0.053)	(0.068)	(0.110)	(0.126)	(0.085)	(0.100)
Controls	Y	Y	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y
% decline explained	50	108	21	40	57	10	23	71
Hansen C p-value		0.010		0.163		0.525		0.370
Hansen J p-value		0.000		0.076		0.349		0.273
Hausman WIV p-value		0.000		0.066		0.286		0.343
Observations	1140	1140	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380	380	380

The table displays second stage results from two stage least squares estimates instrumenting for infrastructure investment using the lagged level of infrastructure investment. First stage results are the same as Table 2. Observations are 'registration district decades', between 1861-1870 and 1891-1900. Infrastructure investment p.c. is the square root of the average stock of loans outstanding over the decade divided by average district population. Tax base p.c. is the square root of the average per capita rateable value of property. Control variables include district population, percentage of population female, the lagged percentage aged under 15, and the lagged percentage aged 15-44; see the Online Appendix for full results. '% decline explained' is the estimated reduction in mortality explained by the level of infrastructure investment as a percentage of the total decline in mortality from 1871-1900 (specifications (1)-(2)) or 1861-1900 (specification (2)-(8)). The shorter period is used in the first two specifications since typhoid was not distinguished in the Registrar General's reports before 1871.
The Hansen C-value is a test of endogoneity, estimated by comparing the Sargan-Hansen statistics from the specification in which infrastructure investment variable is treated as exogenous and where it is treated as endogenous. The Hansen J and Hausman WIV p-values are tests of overidentification when including outrelief expenditure per pauper as a second instrument. The Hansen J test is the usual test for overidentification, while the Hausman WIV test is robust to the inclusion of weak instruments discussed in Section 3.1). In both cases, the null hypothesis is that the overidentifying restrictions are valid.
Standard errors are clustered by registration district, and are displayed in parentheses.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 4: Estimated effect on waterborne mortality in urban areas is similar after controlling for neighboring rural mortality.

	All Districts	Districts with rural portions			
	DV = urban mortality	DV = urban mortality		DV = rural mortality	
	(1)	(2)	(3)	(4)	(5)
Infrastructure investment p.c.	-0.30***	-0.19**	-0.19**	-0.03	-0.00
	(0.076)	(0.081)	(0.081)	(0.108)	(0.102)
Tax base p.c.	0.06	0.04	-0.00		0.37**
	(0.093)	(0.108)	(0.108)		(0.162)
Rural waterborne morality			0.12***		
			(0.041)		
Controls	Y	Y	Y	N	Y
Reg Dist FE	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y
Period	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890
Obs.	757	550	550	550	550
No. Districts	380	275	275	275	275

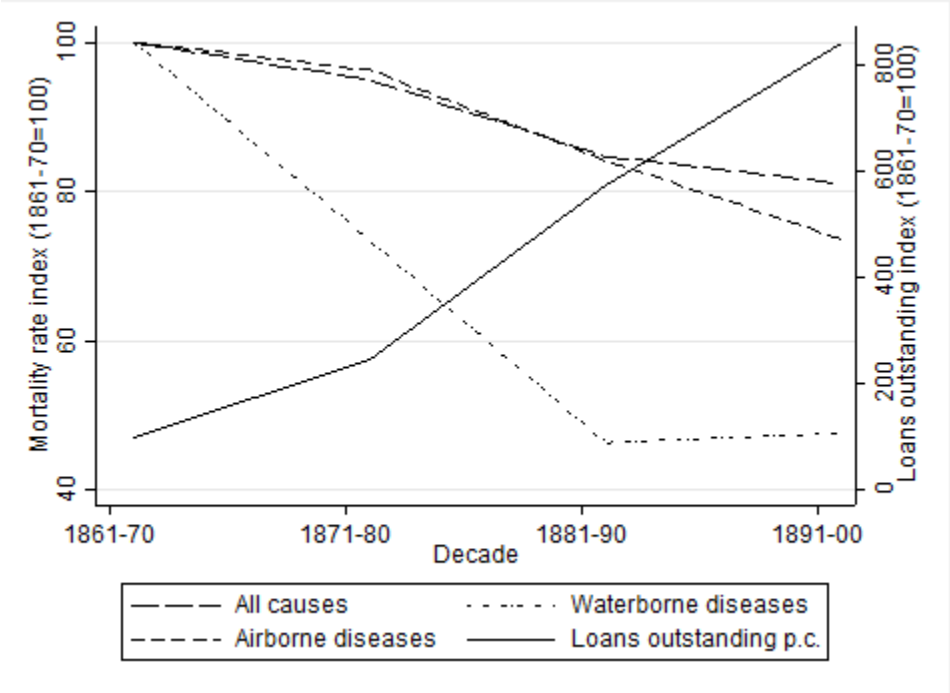
All variables are standardized. Observations are 'registration district decades' for the two decades 1871-1880 and 1881-1890, and using synthetic district boundaries to account for sub-district boundary changes over this period. *Infrastructure investment p.c.* is the square root of the average stock of loans outstanding over the decade divided by average district population. *Tax base p.c.* is the square root of the average per capita rateable value of property. Control variables include district population, percentage of population female, the lagged percentage aged under 15, and the lagged percentage aged 15-44; see the Online Appendix for full results.

Only registration districts with both rural and urban sub-districts are included in specifications (2)-(5). The dependent variable in specifications (1)-(3) is the waterborne mortality rate in the urban sub-districts of each registration district those sub-districts of each registration district containing part of a town in 1881. In specification (5) the dependent variable is mortality in rural sub-districts. Waterborne mortality in these specifications refers to mortality from cholera, diarrhea and fever.

Standard errors are clustered by registration district, and are displayed in parentheses.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Figure 1: Rapid growth in infrastructure loans between 1861 and 1900 coincided with decline in overall mortality of 20% and decline in mortality from waterborne diseases of over 50%.



Each variable is displayed as an index with 1861-70 as the base period. Source: Author's calculations using database described in section II.

Figure 2: General increase in life expectancy across English cities, but with significant variation in experiences between towns.

Source: Data from Szreter and Mooney 'Urbanization'.

Online Appendix to *The contribution of infrastructure investment to Britain's urban mortality decline 1861-1900*

This additional appendix includes full regression results for the abbreviated specifications presented in the paper and presents additional robustness tests of the main results.

Table S1 presents additional fixed effects estimates varying the set of control variables. These specification include both varying subsets of the control variables in Table 1 as well as additional variables, such as population density, which are excluded from the paper due to concerns of multicollinearity. I also allow for, in the last two specifications, time trends based on the wealth and population of the district respectively. Neither the statistical significance nor the size of the effect varies considerably across these specifications.

Table S2 presents results of a number of specifications that test the robustness of the results in Table 1 to controlling for outliers. Specifications (1) and (2) split the sample into two based on the level of debt in 1861-70, while (3) and (4) do the same but based on the level of debt in the final decade (1891-1900). Specifications (5), (6), and (7) then test the robustness to removing the top and bottom 5% of towns based on 1871 population density, 1861-70 district population and 1861-70 mortality respectively. Specification (8) removes observations defined as outliers based on a Cook's distance>4/the number of observations (in this case, 1520). Specification (9) presents the results of using the median estimator suggested by Wooldridge (2010, p461). Again, both the size and statistical significance of the results is similar across specifications.

Table S3 presents the full results of the main instrumental variable regressions in Table 2. Table S4 then presents the two stage least squares estimates when using both instruments (specifications (1)-(3)) and only the weak instrument (specifications (4)-(6)). In specifications (4) and (5) the results are similar when using only the weak instrument; although no longer statistically significant reflecting the imprecision of the estimates with the weak instrument.

However, in specification (6) the estimated effect with the weak instrument is much larger. This finding could reflect the fact that there is limited variation in the level of outrelief per pauper once county time trends are controlled for.

Table S5 presents robustness tests of the two stage least squares estimates to removing outliers, using the same groups as in Table S1 (see earlier discussion). Again, the results are largely unchanged in these specifications, with the exception of the low debt 1891-1900 group, where the estimated effect becomes extremely large. In this case, also, the Kleibergen-Papp statistic is much lower (at 14) than other specifications, indicating that in this group the instrument is relatively weak.

Table S6 presents the full results from Table 3, while Table S7 repeats the analysis in Table 3 in the paper, but adding controls for county time trends. As we can see the results for waterborne mortality are relatively stable. However, the two stage least squares estimate for airborne mortality is no longer statistically significant and the coefficient is closer to the fixed effects estimate. The fixed effects estimate for childbirth mortality is now also statistically significant at a 10% level of significance. This is likely to reflect the fact that childbirth mortality is relatively noisy after partialling out the time trends due to the relatively low level of decline in the rates from this cause of death (as depicted in Figure A.4).

Table S8 presents specifications estimating mortality from different definitions of waterborne disease which are available for the entire period 1861-1900 (unlike the category used in the paper). First, specifications are estimated using only mortality from cholera and diarrhea as the dependent variable. A second set of specifications then estimate a “broad” mortality category – this includes the three causes of death listed in the main paper (cholera, diarrhea and typhoid) but also typhus and continued fever – which were combined with typhoid until 1869 and 1873 respectively – and other causes of death which Szreter (2005) suggests may have been affected by waterborne diseases: diseases of the nervous system and non-

pulmonary tuberculosis. The results show that the estimated effects are still high (albeit lower) when the broad category is analyzed. The effect sizes when analyzing mortality from just cholera and diarrhea, on the other hand, are higher than when typhoid is also included. This differences may reflect the ongoing uncertainty in diagnosing typhoid.[1]

Table S9 and S10 include specifications testing the robustness of the fixed effects specifications in Table 3 to removing outliers for waterborne and airborne diseases, respectively (see the text discussing Table S1 above for the definition of the groups in this table). Tables S11 and S12 then present the same robustness tests for the two stage least squares specifications. The fixed effects regressions are largely unchanged when removing outliers—the main exception being that there is no evidence of any effect on waterborne disease when using the median estimator. The two stage least squares estimates also continue to show consistent evidence of a negative effect on both causes of mortality although the size of the coefficient is more variable across specifications and the effect on airborne diseases is not statistically significant when restricting the sample to towns with low levels of debt in 1891—1900 or those with very high or very low mortality in 1861—1870.

Table S13 then reports the results for the cause-specific death rates including the alternate instrument sets. Here the results for waterborne and airborne mortality are very different when including only the weak instrument—both coefficients become large and statistically insignificant, with the waterborne mortality estimate changing sign.

Finally, Table S14 presents analysis of third quarter total mortality and Table S15 does the same for waterborne mortality (including full results for the specifications in Table 4 in the main paper).

[1] There were considerable reporting inaccuracies even after deaths from typhoid were distinguished from typhus in the annual reports of the Registrar General after 1869, with typhoid often incorrectly diagnosed as either typhus or continued fever. For discussion of these problems, see *The Lancet*, September 21 1878 and *Supplement to the Fifty-Fifth Report of the Annual Report of the Registrar-General*, p.xxvii.

Table S1: Estimated effect on total mortality in fixed effects regressions robust to inclusion of different sets of control variables

	DV = Total mortality rate (all ages, standardized coefficients)										
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
Infrastructure investment p.c.	-0.16***	-0.19***	-0.21***	-0.20***	-0.19***	-0.17***	-0.16***	-0.17***	-0.15***	-0.15***	-0.15***
	(0.031)	(0.026)	(0.028)	(0.028)	(0.028)	(0.029)	(0.028)	(0.031)	(0.030)	(0.030)	(0.031)
Tax base p.c.			-0.00		-0.01	0.00		-0.01	-0.02	0.01	-0.01
			(0.041)		(0.039)	(0.038)		(0.040)	(0.037)	(0.039)	(0.039)
District population	-0.20**							-0.16**	-0.12*	-0.08	-0.15*
	(0.080)							(0.075)	(0.066)	(0.074)	(0.077)
% population female					0.06***	0.06***	0.06***	0.06***	0.07***	0.06***	0.06***
					(0.019)	(0.019)	(0.019)	(0.020)	(0.019)	(0.020)	(0.020)
District popn sq											
Population density		-0.12**			-0.07						
		(0.061)			(0.062)						
Largest town popn						-0.16***	-0.15***				
						(0.056)	(0.055)				
% population age under 15				-0.08***	-0.06***	-0.06***	-0.06***	-0.06***	-0.06***	-0.06***	-0.05***
				(0.017)	(0.017)	(0.017)	(0.017)	(0.017)	(0.016)	(0.017)	(0.017)
% population age 15-44				-0.02	-0.01	-0.01	-0.01	-0.01	-0.02	-0.02	-0.01
				(0.016)	(0.016)	(0.016)	(0.016)	(0.016)	(0.015)	(0.016)	(0.016)
R.D. tax base p.c.							-0.07				
							(0.114)				
% urban population								0.15			
								(0.292)			
Mortality from violence									0.14***		
									(0.022)		
Mortality from childbirth									0.10***		
									(0.014)		
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Time Trends	N	N	N	N	N	N	N	N	N	Population	Wealth
Observations	1520	1520	1520	1520	1520	1520	1515	1520	1520	1520	1520
No. Districts	380	380	380	380	380	380	380	380	380	380	380

Table illustrates the robustness of the results of the fixed effects specifications presented in Table 1 to inclusion of different sets of control variables. “District popn sq” is the square of the population variable. Largest town population is the population of the largest town in the registration district, identified according to 1881 population. Mortality from violence is at all ages and for childbirth is for age 15-44 adjusted by estimated female population. See note to Table 1 for further details. * p< 0.10, ** p < 0.05, *** p < 0.01.

Table S2: Estimated effect on total mortality in fixed effects regressions robust to exclusion of outliers

	DV = Total mortality rate (all ages, standardized coefficients)								
	Low Debt 1861-70	High Debt 1861-70	Low Debt 1891-00	High Debt 1891-00	No extreme density	No extreme population	No extreme mortality	No Cook's outliers	Median estimator
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Infrastructure	-0.20***	-0.15***	-0.20***	-0.14***	-0.18***	-0.17***	-0.14***	-0.17***	-0.17***
investment p.c.	(0.046)	(0.041)	(0.070)	(0.039)	(0.031)	(0.030)	(0.031)	(0.031)	(0.052)
Tax base p.c.	0.04	-0.01	0.02	0.01	0.03	0.03	0.03	-0.00	-0.07
	(0.046)	(0.059)	(0.052)	(0.058)	(0.037)	(0.036)	(0.037)	(0.038)	(0.067)
District population	-0.23	-0.13	-0.39	-0.15*	-0.15*	-0.10	-0.11*	-0.16**	-0.32***
	(0.141)	(0.083)	(0.280)	(0.083)	(0.080)	(0.068)	(0.067)	(0.076)	(0.101)
% population female	0.05*	0.08***	0.08**	0.06**	0.06***	0.06***	0.05**	0.06***	0.06
	(0.028)	(0.027)	(0.032)	(0.025)	(0.020)	(0.020)	(0.024)	(0.019)	(0.037)
% population age under 15	-0.04**	-0.06**	-0.03	-0.06**	-0.04**	-0.05***	-0.06***	-0.06***	-0.04
	(0.022)	(0.028)	(0.023)	(0.027)	(0.018)	(0.018)	(0.018)	(0.017)	(0.025)
% population age 15-44	0.01	-0.02	0.02	-0.02	-0.00	-0.01	-0.01	-0.01	0.00
	(0.024)	(0.021)	(0.025)	(0.021)	(0.017)	(0.016)	(0.018)	(0.016)	(0.027)
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Time trends	N	N	N	N	N	N	N	N	N
Observations	760	760	760	760	1368	1364	1368	1520	1520
No. Districts	190	190	190	190	342	341	342	380	380

Table tests the robustness of the results of the fixed effects specifications presented in Table 1 to various methods of removing outliers. See text at beginning of Appendix B for details of groups. For further details see note to Table 1. * p< 0.10, ** p < 0.05, *** p < 0.01.

Table S3: Full results of instrumental variables regressions (extension of Table 2)

	DV = Total mortality rate (all ages, standardized coefficients)					
	(1)	(2)	(3)	(4)	(5)	(6)
	FE	IV	FE	IV	FE	IV
Panel A: Fixed effects and 2SLS specifications for mortality at all ages						
Infrastructure investment p.c.	-0.17***	-0.32***	-0.15***	-0.36***	-0.11***	-0.27***
	(0.030)	(0.048)	(0.038)	(0.070)	(0.031)	(0.085)
Tax base p.c.			0.11*	0.23***	0.03	0.14*
			(0.057)	(0.068)	(0.051)	(0.074)
District population			-0.11	0.01	-0.03	0.03
			(0.100)	(0.071)	(0.057)	(0.046)
% population female			0.05**	0.06**	0.02	0.03
			(0.026)	(0.027)	(0.025)	(0.025)
Lag % age under 15			-0.10***	-0.09***	-0.11***	-0.11***
			(0.022)	(0.022)	(0.020)	(0.019)
Lag % age 15-44			-0.05***	-0.04**	-0.03**	-0.03*
			(0.017)	(0.018)	(0.015)	(0.015)
Panel B: Abbreviated first stage regressions for infrastructure investment per capita						
Lag Infrastructure investment		0.56***		0.42***		0.31***
p.c.		(0.033)		(0.038)		(0.037)
Tax base p.c.				0.44***		0.51***
				(0.052)		(0.054)
District population				0.29***		0.24**
				(0.112)		(0.109)
% population female				-0.02		-0.00
				(0.025)		(0.028)
Lag % age under 15				0.03		-0.00
				(0.019)		(0.024)
Lag % age 15-44				0.04**		0.01
				(0.019)		(0.020)
Controls	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y
Observations	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380

See note to Table 2 for details of specifications.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S4: Two stage least squares estimates for total mortality including both instruments and only weak instrument

	DV = Total mortality rate (all ages, standardized coefficients)					
	Both instruments			Weak instrument only		
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Two stage least squares for mortality at all ages						
Infrastructure investment p.c.	-0.32***	-0.36***	-0.29***	-0.21	-0.31	-0.78
	(0.047)	(0.069)	(0.084)	(0.556)	(0.458)	(0.513)
Tax base p.c.		0.23***	0.15**		0.21	0.46
		(0.068)	(0.074)		(0.287)	(0.327)
District population		0.01	0.04		-0.02	0.22
		(0.071)	(0.045)		(0.262)	(0.218)
% population female		0.06**	0.03		0.06**	0.04
		(0.027)	(0.025)		(0.027)	(0.037)
Lag % age under 15		-0.09***	-0.11***		-0.09**	-0.10***
		(0.022)	(0.020)		(0.039)	(0.027)
Lag % age 15-44		-0.04**	-0.03*		-0.04	-0.02
		(0.018)	(0.016)		(0.039)	(0.023)
Panel B: Abbreviated first stage regressions for infrastructure investment per capita						
Lag Infrastructure investment	0.56***	0.42***	0.31***			
p.c.	(0.033)	(0.039)	(0.037)			
Expend per pauper	0.03	0.04	0.06*	0.05	0.06**	0.07**
	(0.029)	(0.028)	(0.032)	(0.037)	(0.030)	(0.033)
Tax base p.c.		0.45***	0.52***		0.62***	0.64***
		(0.052)	(0.054)		(0.068)	(0.065)
District population		0.29***	0.24**		0.55**	0.37**
		(0.113)	(0.109)		(0.225)	(0.175)
% population female		-0.02	-0.00		0.01	0.03
		(0.025)	(0.028)		(0.029)	(0.032)
Lag % age under 15		0.03	-0.00		0.07***	0.01
		(0.019)	(0.024)		(0.026)	(0.028)
Lag % age 15-44		0.04**	0.01		0.07***	0.01
		(0.018)	(0.020)		(0.022)	(0.023)
Controls	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y
% decline explained	54	60	48	35	53	130
Kleibergen-Papp Stat	151	63	38	2	4	4
Hansen C p-value	0.000	0.002	0.025	0.951	0.719	0.092
Observations	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380

Table displays results for two stage least squares regressions in Table 2 for alternative instrument sets. In specifications (1)-(3) the instruments are both lagged loans outstanding per capita (per capita) and outrelief per pauper. In specifications (4)-(6) only the outrelief per pauper is used. See note to Table 2 for further details.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S5: Two stage least squares regressions for total mortality: robustness to removing outliers

	DV = Total mortality rate (all ages, standardized coefficients)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Low Debt 1861-70	High Debt 1861-70	Low Debt 1891-00	High Debt 1891-00	No extreme density	No extreme population	No extreme mortality
Infrastructure investment p.c.	-0.518***	-0.312***	-0.818**	-0.380***	-0.342***	-0.322***	-0.268***
	(0.114)	(0.092)	(0.367)	(0.105)	(0.077)	(0.076)	(0.079)
Tax base p.c.	0.279***	0.289***	0.184**	0.324***	0.204***	0.197***	0.192***
	(0.084)	(0.107)	(0.085)	(0.103)	(0.071)	(0.071)	(0.072)
District population	0.214	0.010	-0.443	0.018	0.010	0.058	0.017
	(0.185)	(0.085)	(0.420)	(0.071)	(0.076)	(0.061)	(0.073)
% population female	0.055	0.060	0.014	0.075**	0.057**	0.053*	0.050
	(0.039)	(0.038)	(0.042)	(0.037)	(0.028)	(0.028)	(0.031)
Lag % age under 15	-0.038	-0.134***	-0.037	-0.121***	-0.080***	-0.089***	-0.097***
	(0.028)	(0.036)	(0.035)	(0.036)	(0.023)	(0.023)	(0.024)
Lag % age 15-44	-0.003	-0.067***	-0.014	-0.053**	-0.034*	-0.040**	-0.052**
	(0.026)	(0.025)	(0.028)	(0.026)	(0.019)	(0.018)	(0.020)
Controls	Y	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y
Kleibergen-Papp Stat	57	94	14	62	113	108	111
Hansen C p-value	0.001	0.051	0.041	0.007	0.007	0.010	0.037
Hansen J p-value	0.215	0.226	0.866	0.815	0.134	0.291	0.859
Hausman WIV p-value	0.161	0.240	0.842	0.804	0.109	0.256	0.847
Observations	570	570	570	570	1026	1023	1026
No. Districts	190	190	190	190	342	341	342

Table presents the second stage results of tests for the robustness of the results of the instrumental variable specifications presented in Table 2 to various methods of removing outliers. See text at start of Appendix B for details of the observations included in specification. See note to Table 2 for further details of the specifications.

* p< 0.10, ** p < 0.05, *** p < 0.01.

Table S6: Extended results for effects on mortality by cause 1871-1900 (Following from Table 3)

	DV = Mortality rate at all ages by cause							
	Waterborne		Airborne		Childbirth		Violence	
	FE	IV	FE	IV	FE	IV	FE	IV
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Infrastructure investment p.c.	-0.17***	-0.36***	-0.10***	-0.19**	-0.08	-0.02	-0.04	-0.14
	(0.045)	(0.090)	(0.037)	(0.076)	(0.068)	(0.123)	(0.072)	(0.120)
Tax base p.c.	0.15**	0.28***	-0.04	0.01	0.04	-0.00	0.09	0.15
	(0.075)	(0.092)	(0.053)	(0.068)	(0.110)	(0.126)	(0.085)	(0.100)
District population	0.06	0.17**	-0.15*	-0.10	0.14	0.10	-0.19	-0.14
	(0.083)	(0.074)	(0.092)	(0.087)	(0.100)	(0.105)	(0.167)	(0.170)
% population female	-0.02	-0.02	0.05*	0.05*	-0.13**	-0.13**	0.04	0.04
	(0.042)	(0.043)	(0.026)	(0.026)	(0.053)	(0.053)	(0.044)	(0.043)
Lag % age under 15	-0.01	0.00	-0.01	-0.01	-0.04	-0.04	-0.15***	-0.14***
	(0.029)	(0.030)	(0.021)	(0.021)	(0.042)	(0.043)	(0.031)	(0.032)
Lag % age 15-44	0.05*	0.06**	-0.00	0.00	0.05	0.04	-0.08***	-0.08***
	(0.027)	(0.027)	(0.017)	(0.017)	(0.038)	(0.037)	(0.027)	(0.027)
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y
% decline explained	50	108	21	40	57	10	23	71
Hansen C p-value		0.010		0.163		0.525		0.370
Hansen J p-value		0.000		0.076		0.349		0.273
Hausman WIV p-value		0.000		0.066		0.286		0.343
Observations	1140	1140	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380	380	380

See notes to Table 3 for further details of specifications.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S7: Results for effects on mortality by cause 1871-1900, including county time trends

	DV = Mortality rate at all ages by cause							
	Waterborne		Airborne		Childbirth		Violence	
	FE	IV	FE	IV	FE	IV	FE	IV
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Infrastructure investment p.c.	-0.13***	-0.34***	-0.07**	-0.10	-0.13*	-0.03	-0.01	-0.07
	(0.042)	(0.113)	(0.035)	(0.095)	(0.072)	(0.170)	(0.062)	(0.154)
Tax base p.c.	0.08	0.21**	-0.11**	-0.09	0.05	-0.02	0.02	0.06
	(0.064)	(0.096)	(0.056)	(0.081)	(0.114)	(0.146)	(0.087)	(0.118)
District population	0.05	0.13**	-0.07	-0.06	0.12	0.08	-0.09	-0.07
	(0.064)	(0.062)	(0.052)	(0.057)	(0.098)	(0.101)	(0.131)	(0.132)
% population female	-0.05	-0.05	0.04	0.04*	-0.12**	-0.13**	0.03	0.04
	(0.040)	(0.040)	(0.025)	(0.024)	(0.052)	(0.051)	(0.044)	(0.043)
Lag % age under 15	0.01	0.02	-0.03	-0.03	-0.03	-0.03	-0.15***	-0.15***
	(0.030)	(0.030)	(0.022)	(0.022)	(0.045)	(0.044)	(0.032)	(0.032)
Lag % age 15-44	0.07**	0.07***	0.02	0.02	0.05	0.05	-0.06**	-0.06**
	(0.027)	(0.026)	(0.017)	(0.016)	(0.040)	(0.039)	(0.029)	(0.028)
Controls	Y	Y	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y
% decline explained	40	101	15	21	90	17	4	36
Hansen C p-value		0.043		0.733		0.501		0.668
Hansen J p-value		0.108		0.096		0.136		0.082
Hausman WIV p-value		0.126		0.092		0.102		0.111
Observations	1140	1140	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380	380	380

See notes to Table 3 for further details of specifications.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S8: Estimated results for effects on different measures of waterborne mortality

	DV = Mortality from cholera and diarrhea			DV = Broad waterborne category		
	FE	FE	IV	FE	FE	IV
	(1)	(2)	(3)	(4)	(5)	(6)
Infrastructure investment p.c.	-0.15***	-0.15***	-0.29***	-0.16***	-0.16***	-0.35***
	(0.040)	(0.040)	(0.084)	(0.037)	(0.037)	(0.078)
Tax base p.c.	-0.00	-0.00	0.08	0.11*	0.11*	0.23***
	(0.067)	(0.067)	(0.082)	(0.059)	(0.059)	(0.072)
District population	0.07	0.07	0.14*	-0.08	-0.08	0.02
	(0.075)	(0.075)	(0.076)	(0.077)	(0.077)	(0.064)
% population female	-0.03	-0.03	-0.03	0.00	0.00	0.01
	(0.038)	(0.038)	(0.039)	(0.029)	(0.029)	(0.030)
Lag % age under 15	-0.03	-0.03	-0.02	-0.09***	-0.09***	-0.07***
	(0.027)	(0.027)	(0.027)	(0.023)	(0.023)	(0.023)
Lag % age 15-44	0.07***	0.07***	0.08***	-0.01	-0.01	0.01
	(0.026)	(0.026)	(0.026)	(0.020)	(0.020)	(0.021)
Controls	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y
Period	1861-1900	1871-1900	1871-1900	1861-1900	1871-1900	1871-1900
% 1871-1900 decline explained	67	67	128	38	38	83
% 1861-1900 decline explained	40	40	77	25	25	55
Kleibergen-Papp Stat			119			119
Hansen C p-value			0.053			0.006
Hansen J p-value			0.000			0.026
Hausman WIV p-value			0.000			0.012
Observations	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380

Table displays results for specifications (1) and (2) in Table 3, using alternative definition of waterborne mortality. See note to Table 3 for further details of specifications.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S9: Fixed effects regressions for waterborne diseases: robustness to removing outliers

	DV = Mortality rate from waterborne diseases at all ages 1861-1900								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Low Debt 1861-70	High Debt 1861-70	Low Debt 1891-00	High Debt 1891-00	No extreme density	No extreme population	No extreme mortality	No Cook's outliers	Median estimator
Infrastructure	-0.17**	-0.21***	-0.10	-0.16***	-0.17***	-0.16***	-0.13***	-0.18***	0.03
investment p.c.	(0.067)	(0.066)	(0.078)	(0.060)	(0.045)	(0.045)	(0.046)	(0.045)	(0.060)
Tax base p.c.	0.14	0.32***	0.12	0.38***	0.21***	0.17**	0.14*	0.18***	-0.14
	(0.091)	(0.113)	(0.088)	(0.111)	(0.075)	(0.075)	(0.076)	(0.071)	(0.102)
District population	0.37*	0.04	0.40	0.07	0.09	0.14**	0.13**	0.06	-0.18**
	(0.215)	(0.100)	(0.465)	(0.089)	(0.079)	(0.065)	(0.065)	(0.084)	(0.083)
% population female	0.01	0.03	0.01	0.03	0.02	0.02	0.01	0.02	-0.17***
	(0.054)	(0.054)	(0.050)	(0.053)	(0.041)	(0.040)	(0.046)	(0.038)	(0.035)
% population age under 15	0.06	0.01	0.06	0.02	0.05	0.04	0.03	0.03	0.02
	(0.044)	(0.050)	(0.042)	(0.049)	(0.035)	(0.036)	(0.036)	(0.034)	(0.035)
% population age 15-44	0.07	0.14***	0.11**	0.12***	0.13***	0.13***	0.11***	0.12***	0.02
	(0.045)	(0.039)	(0.046)	(0.039)	(0.033)	(0.033)	(0.033)	(0.031)	(0.035)
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Observations	570	570	570	570	1026	1023	1026	1140	1140
No. Districts	190	190	190	190	342	341	342	380	380

Table displays the robustness of specification (1) in Table 3 to excluding groups of outliers. See text at start of Appendix B for details of the observations included in each specification. See note to Table 3 for further details of the specifications.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S10: Fixed effects regressions for airborne diseases: robustness to removing outliers

	DV = Mortality rate from airborne diseases at all ages 1861-1900								
	(1) Low Debt 1861-70	(2) High Debt 1861-70	(3) Low Debt 1891-00	(4) High Debt 1891-00	(5) No extreme density	(6) No extreme population	(7) No extreme mortality	(8) No Cook's outliers	(9) Median estimator
Infrastructure.	-0.13***	-0.11***	-0.09	-0.08**	-0.11***	-0.10***	-0.10***	-0.12***	-0.11***
investment p.c	(0.044)	(0.038)	(0.076)	(0.038)	(0.032)	(0.030)	(0.031)	(0.030)	(0.035)
Tax base p.c.	0.02	-0.01	-0.00	-0.01	0.01	-0.00	0.00	-0.02	-0.05
	(0.050)	(0.056)	(0.063)	(0.050)	(0.039)	(0.039)	(0.040)	(0.037)	(0.056)
District population	-0.17	-0.07	0.12	-0.11	-0.11	-0.04	-0.05	-0.11*	-0.32***
	(0.125)	(0.065)	(0.248)	(0.070)	(0.068)	(0.049)	(0.050)	(0.061)	(0.059)
% population female	0.01	0.08***	0.03	0.07***	0.04**	0.04*	0.03	0.05**	0.02
	(0.026)	(0.023)	(0.030)	(0.023)	(0.019)	(0.019)	(0.023)	(0.018)	(0.037)
% population age	0.03	-0.00	0.01	0.02	0.02	0.02	0.00	0.01	0.04*
under 15	(0.023)	(0.023)	(0.024)	(0.025)	(0.017)	(0.017)	(0.018)	(0.016)	(0.026)
% population age 15-	0.08***	0.02	0.06**	0.04*	0.05***	0.05***	0.04**	0.05***	0.07**
44	(0.025)	(0.021)	(0.025)	(0.023)	(0.017)	(0.017)	(0.017)	(0.016)	(0.028)
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Period	1861-1900	1861-1900	1861-1900	1861-1900	1861-1900	1861-1900	1861-1900	1861-1900	1861-1900
Obs.	760	760	760	760	1368	1364	1368	1520	1520
No. Districts	190	190	190	190	342	341	342	380	380

Table displays the robustness of specification (3) in Table 3 to excluding groups of outliers. See text at start of Appendix B for details of the observations included in each specification. See notes to Table 3 for further details of the specifications.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S11: Two stage least squares regressions for waterborne diseases: robustness to removing outliers

	DV = Mortality rate at all ages 1861-1900						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Low Debt 1861-70	High Debt 1861-70	Low Debt 1891-00	High Debt 1891-00	No extreme density	No extreme population	No extreme mortality
Infrastructure investment p.c.	-0.579***	-0.312***	-0.346	-0.488***	-0.349***	-0.331***	-0.268***
	(0.138)	(0.119)	(0.409)	(0.148)	(0.096)	(0.095)	(0.099)
Tax base p.c.	0.322***	0.336**	0.128	0.462***	0.284***	0.254***	0.205**
	(0.122)	(0.139)	(0.119)	(0.127)	(0.096)	(0.097)	(0.100)
District population	0.836***	0.091	0.579	0.174**	0.192**	0.225***	0.204***
	(0.249)	(0.087)	(0.470)	(0.072)	(0.075)	(0.079)	(0.076)
% population female	-0.005	-0.015	-0.030	-0.022	-0.025	-0.023	-0.027
	(0.067)	(0.054)	(0.059)	(0.056)	(0.048)	(0.045)	(0.051)
Lag % age under 15	0.040	-0.034	0.027	-0.056	0.013	0.014	0.001
	(0.039)	(0.045)	(0.039)	(0.044)	(0.032)	(0.032)	(0.031)
Lag % age 15-44	0.068*	0.056	0.068**	0.042	0.068**	0.072**	0.066**
	(0.038)	(0.038)	(0.034)	(0.040)	(0.030)	(0.029)	(0.029)
Controls	Y	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y
Hansen C p-value	0.004	0.181	0.515	0.007	0.021	0.024	0.073
Hansen J p-value	0.000	0.088	0.000	0.164	0.003	0.002	0.000
Hausman WIV p-value	0.000	0.088	0.000	0.165	0.005	0.002	0.000
Observations	570	570	570	570	1026	1023	1026
No. Districts	190	190	190	190	342	341	342

Table displays the robustness of specification (2) in Table 3 to excluding groups of outliers. See text at start of Appendix B for details of the observations included in each specification. See notes to Table 3 for further details of the specifications. *$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S12: Two stage least squares regressions for airborne diseases: robustness to removing outliers

	DV = Mortality rate from airborne diseases at all ages 1861-1900						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Low Debt 1861-70	High Debt 1861-70	Low Debt 1891-00	High Debt 1891-00	No extreme density	No extreme population	No extreme mortality
Infrastructure investment p.c.	-0.213*	-0.200**	-0.085	-0.202**	-0.173**	-0.126	-0.081
	(0.117)	(0.091)	(0.337)	(0.103)	(0.083)	(0.084)	(0.080)
Tax base p.c.	0.090	0.050	0.013	0.060	-0.003	-0.037	-0.032
	(0.088)	(0.100)	(0.080)	(0.099)	(0.071)	(0.072)	(0.071)
District population	-0.171	-0.053	0.047	-0.082	-0.089	-0.040	-0.070
	(0.188)	(0.085)	(0.298)	(0.082)	(0.087)	(0.067)	(0.079)
% population female	-0.023	0.112***	-0.024	0.111***	0.055*	0.042	0.018
	(0.038)	(0.034)	(0.040)	(0.032)	(0.028)	(0.027)	(0.029)
Lag % age under 15	0.019	-0.022	-0.026	0.021	-0.007	-0.016	-0.021
	(0.028)	(0.030)	(0.032)	(0.034)	(0.021)	(0.021)	(0.021)
Lag % age 15-44	0.033	-0.018	-0.015	0.022	0.000	-0.003	-0.016
	(0.024)	(0.022)	(0.025)	(0.024)	(0.018)	(0.017)	(0.018)
Controls	Y	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y
Hansen C p-value	0.280	0.170	0.801	0.139	0.162	0.446	0.794
Hansen J p-value	0.306	0.272	0.140	0.820	0.031	0.001	0.008
Hausman WIV p-value	0.298	0.264	0.121	0.824	0.026	0.001	0.005
Observations	570	570	570	570	1026	1023	1026
No. Districts	190	190	190	190	342	341	342

Table displays the robustness of specification (4) in Table 3 to excluding groups of outliers. See text at start of Appendix B for details of the observations included in each specification. See notes to Table 3 for further details of the specifications.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S13: Two stage least squares estimates for mortality from specific causes including both instruments and only weak instrument

	Both instruments				Only weak instrument			
	Waterborne	Airborne	Childbirth	Violence	Waterborne	Airborne	Childbirth	Violence
Infrastructure investment p.c.	-0.34***	-0.20***	-0.03	-0.15	1.15	-0.79	-0.78	-0.66
	(0.089)	(0.074)	(0.124)	(0.119)	(0.785)	(0.520)	(1.044)	(0.665)
Tax base p.c.	0.26***	0.02	0.01	0.15	-0.66	0.38	0.47	0.47
	(0.091)	(0.067)	(0.128)	(0.100)	(0.487)	(0.320)	(0.665)	(0.416)
District population	0.15**	-0.10	0.11	-0.13	-0.68	0.23	0.52	0.15
	(0.074)	(0.085)	(0.108)	(0.167)	(0.562)	(0.306)	(0.619)	(0.377)
% population female	-0.02	0.05*	-0.13**	0.04	-0.04	0.06*	-0.12**	0.05
	(0.043)	(0.026)	(0.053)	(0.043)	(0.059)	(0.034)	(0.058)	(0.045)
Lag % age under 15	0.00	-0.01	-0.04	-0.14***	-0.10	0.03	0.01	-0.11**
	(0.030)	(0.021)	(0.043)	(0.032)	(0.068)	(0.044)	(0.084)	(0.054)
Lag % age 15-44	0.06**	0.01	0.05	-0.07***	-0.05	0.05	0.10	-0.04
	(0.027)	(0.017)	(0.037)	(0.027)	(0.067)	(0.044)	(0.083)	(0.056)
Controls	Y	Y	Y	Y	Y	Y	Y	Y
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y
% decline explained	100	42	20	76	0	164	526	346
Hansen C p-value	0.043	0.109	0.546	0.284	0.007	0.092	0.502	0.296
Observations	1140	1140	1140	1140	1140	1140	1140	1140
No. Districts	380	380	380	380	380	380	380	380

Table displays results for two stage least squares regressions in Table 3 for alternative instrument sets. In specifications (1)-(4) the instruments are both lagged loans outstanding per capita (per capita) and outrelief per pauper. In specifications (5)-(8) only the outrelief per pauper is used. See notes to Table 3 for further details of specifications. *$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S.14: Extended results from regressions for comparison of total urban and rural mortality in the third quarter

	All districts		Districts with no rural portions		Districts with rural portions				
	DV = urban mortality		DV = urban mortality		DV = urban mortality			DV = rural mortality	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Infrastructure investment p.c.	-0.23***	-0.26***	-0.24*	-0.39**	-0.22***	-0.13	-0.14	-0.16	0.06
	(0.074)	(0.091)	(0.146)	(0.164)	(0.084)	(0.108)	(0.108)	(0.139)	(0.149)
Tax base p.c.		0.29***		0.49***		0.19	0.18		0.17
		(0.101)		(0.141)		(0.146)	(0.146)		(0.167)
District population		-0.24		-0.03		-0.77*	-0.67		-1.63***
		(0.278)		(0.264)		(0.459)	(0.443)		(0.557)
% population female		-0.03		0.02		-0.06	-0.06		-0.06
		(0.059)		(0.076)		(0.085)	(0.083)		(0.116)
% population age under 15		-0.01		0.03		-0.00	-0.00		-0.07
		(0.052)		(0.089)		(0.063)	(0.063)		(0.082)
% population age 15-44		0.01		0.03		0.02	0.01		0.02
		(0.054)		(0.084)		(0.070)	(0.071)		(0.092)
Rural total morality							0.07		
							(0.051)		
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Period	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890
Observations	757	757	206	206	550	550	550	550	550
No. Districts	380	380	104	104	275	275	275	275	275

Extended results from specifications presented in Table 4, but with dependent variable of total (rather than waterborne) mortality. See notes to Table 4 for further details of specification.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table S15: Extended results from regressions for comparison of urban and rural waterborne mortality in the third quarter 1871-1890

	All districts		Districts with no rural portions		Districts with rural portions				
	DV = urban mortality		DV = urban mortality		DV = urban mortality			DV = rural mortality	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Infrastructure investment p.c.	-0.23***	-0.30***	-0.30***	-0.41***	-0.31***	-0.19**	-0.19**	-0.03	-0.00
	(0.074)	(0.076)	(0.110)	(0.123)	(0.081)	(0.081)	(0.081)	(0.108)	(0.102)
Tax base p.c.		0.06		0.12		0.04	-0.00		0.37**
		(0.093)		(0.157)		(0.108)	(0.108)		(0.162)
District population		-0.26		-0.03		-0.76*	-0.69*		-0.55
		(0.273)		(0.263)		(0.401)	(0.362)		(0.700)
% population female		0.00		0.15*		-0.10	-0.09		-0.07
		(0.059)		(0.078)		(0.077)	(0.077)		(0.120)
% population age under 15		-0.04		0.07		-0.05	-0.05		-0.01
		(0.040)		(0.093)		(0.046)	(0.046)		(0.076)
% population age 15-44		0.12**		0.27**		0.06	0.04		0.18**
		(0.049)		(0.112)		(0.049)	(0.049)		(0.079)
Rural Waterborne morality							0.12***		
							(0.041)		
Reg Dist FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Decade FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Period	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890	1871-1890
Observations	757	757	206	206	550	550	550	550	550
No. Districts	380	380	104	104	275	275	275	275	275

Extended results from specifications presented in Table 4: see notes to that table for further details.
*$p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A1: Descriptive statistics of main variables

Variable	Obs	Mean	Std. Dev.	Min	Max
Total average loans outstanding per capita (£ p.c.)	1,520	1.68	2.92	0.00	34.81
Square root of average loans outstanding per capita (£ p.c.)	1,520	0.99	0.84	0.00	5.90
Square root of tax base per capita (£ p.c.)	1,520	1.22	0.50	0.03	2.89
Deaths from all causes per 1,000 popn (all ages)	1,520	18.88	2.67	12.32	32.13
Deaths from waterborne diseases per 1,000 popn (all ages)	1,140	0.77	0.43	0.08	3.01
Deaths from airborne diseases per 1,000 popn (all ages)	1,520	6.14	1.45	2.97	12.87
Deaths from violence per 1,000 popn (all ages)	1,520	0.64	0.18	0.320	1.82
Deaths from childbirth per 100,000 females, 15-44	1,515	0.68	0.21	0.078	1.46
Population (10,000s)	1,520	4.86	6.32	0.58	76.78
Population density (population per acre)	1,520	2.50	6.96	0.06	69.18
% population age under 15	1,520	36	2	28	42
% population age 15-44	1,520	43	3	37	59
% population female	1,520	51	2	38	59
% of loans outstanding in Water 1884-1890	369	24	28	0	100
% of loans outstanding in Sewers 1884-1890	369	25	27	0	100
% of loans outstanding in Street 1884-1890	369	14	19	0	100
% of loans outstanding in Gas 1884-1890	369	8	17	0	85
% of loans outstanding in other category 1884-1890	369	28	28	0	100
% of loans outstanding in Water 1891-1900	374	28	27	0	100
% of loans outstanding in Sewers 1891-1900	374	25	24	0	100
% of loans outstanding in Street 1891-1900	374	12	14	0	100
% of loans outstanding in Gas 1891-1900	374	9	17	0	85
% of loans outstanding in other category 1891-1900	374	27	24	0	100
% of loans outstanding in Water 1884-1890	369	24	28	0	100

Figure A1: Square root transformation reduces positive skewness in average outstanding loans per capita

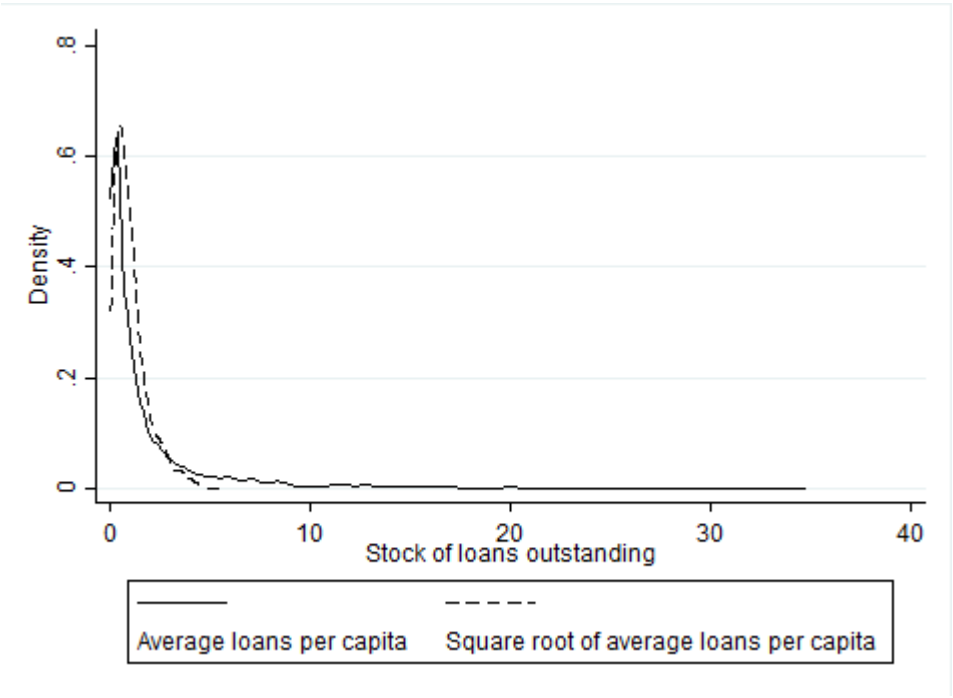
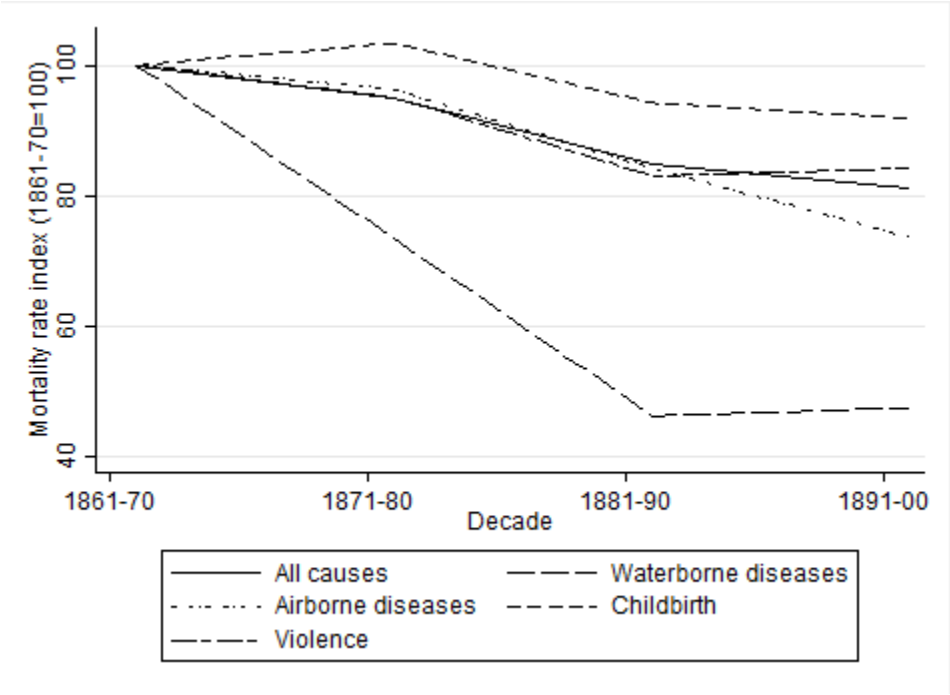


Figure displays the kernel density of the decadal average outstanding loans per capita for each registration district and decade included in the main regressions presented in the text.

Figure A2: Trends in mortality from different causes 1861 to 1900



Source: Author's calculations using database described in the text.