Urban shitscapes and the late decline of infant diarrhoeal mortality in England and Wales: evidence from Medical Officer of Health reports, 1895-1911.

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Note:

This paper reports the results of a pilot study to investigate the suitability of Medical Officer of Health reports to study the relationships between changes in sanitary provision and faecal-oral disease outcomes in British cities. This working paper has been produced to demonstrate the method in support of a proposal to the ESRC for funding to, among other things, expand this work both chronologically (to 1930) and geographically (to include more towns). All results reported here are preliminary, and the panel study is based on a small sample (of ten towns).

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This paper addresses a persistent puzzle in British historical demography: the late decline of infant diarrhoeal mortality during the early stages of the demographic transition. While survival improved for children and young adults in Britain from the late 1860s, infant mortality did not improve, either nationally or in cities, until after 1900 (Figure 1). Part of the reason for this late decline was high rates of infant diarrhoeal mortality in towns and cities, which peaked in the 1890s due to a series of hot summers that caused very large diarrhoeal epidemics. After 1900 a series of cooler summers reduced diarrhoeal mortality, and infant mortality embarked on a decisive secular decline. However diarrhoeal mortality did not show the same improvement. Rates in the period 1901-10 were similar to those of the 1880s, and diarrhoeal mortality remained a very substantial component of infant mortality, as was evident in 1911 when a very hot summer caused a major epidemic of diarrhoea amongst infants across much of Europe (Figure 1).

The lack of improvement in diarrhoeal mortality over the 19th and early 20th centuries is particularly puzzling because other faecal-oral diseases declined very decisively over the same period. Water supplies improved in British cities after the mid-nineteenth century, and cholera and dysentery had virtually disappeared by 1870 (Davenport et al., 2019; Aidt et al., 2022). Typhoid on the other hand underwent a two-stage decline (Figure 1a). Typhoid was only reported separately from typhus in 1869, from which point it declined dramatically until the mid-1880s, and then showed little further improvement until after 1900.

The diverse timing of declines in different faecal-oral diseases seems likely to reflect the chronology of improvements in water supplies and sanitation in British cities. Diarrhoeal diseases, cholera, typhoid and dysentery are transmitted by ingestion of faeces that are infected with pathogenic bacteria (as well as viruses, fungi and various parasites in some cases). However the routes by which these pathogens enter the human host are diverse. Ingestion of infected faeces can occur via faecally-contaminated drinking water; by inadequate hand-washing leading to direct ingestion (by touching hands to mouth) or infection via contamination of food; by direct exposure to faeces in the environment (for example if children play with faeces or handle contaminated objects); or via flies that feed on faeces and then alight on food or drink or on skin. In a study of historical disease outbreaks in which the causative pathogen was identified together with the source of the outbreak, Ewald (1991) demonstrated a strong relationship between the most virulent faecal-oral pathogens and water-borne transmission (Table 1). Most outbreaks of 'Asiatic' cholera (the strain associated with the nineteenth century pandemics of cholera) were traced to faecal contamination of drinking water. Similarly, dysentery, the scourge of armies, was generally associated with polluted drinking water. Both diseases killed 20 – 30 per cent of those infected. In contrast, diarrhoeal diseases kill mainly very young children and elderly people, and display age-specific mortality patterns very similar to all-cause mortality (Preston, 1976). In Ewald's study, diarrhoeal outbreaks were usually traced to pathogens with low relative virulence (including *Campylobacter jejuni, Escherichia coli* and non-typhoid salmonella) and were traced mainly to contaminated food.[1] Typhoid presented an intermediate case, because the proportion of outbreaks traced to contaminated water supplies declined steeply with time (as water supplies improved) (Ewald, 1991). Typhoid is unusual because in addition to transmission by contaminated water, food and possibly flies, it can also be transmitted by asymptomatic human carriers. After infection, human hosts continue to excrete typhoid bacteria (*Salmonella typhi*) in their urine and faeces for several months after symptoms have cleared, and 2-5 % of those infected remain infected and infectious for at least a year (Gunn et al. 2014). These carriers can then infect others if they handle food without washing their hands.

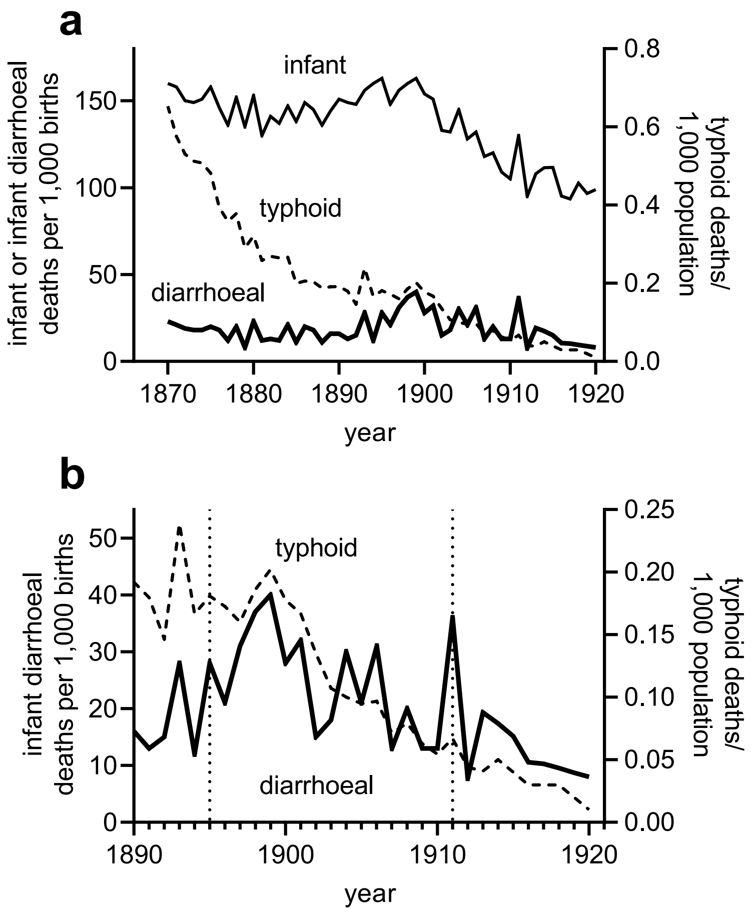

Figure 1. Infant, infant diarrhoeal and crude typhoid mortality, England and Wales 1870-1920. Typhoid rates include deaths attributed to 'typhoid' and to 'continued fever'. Dashed vertical lines in the lower panel indicate the period covered by the study (1895-1911).

[1] The pathogens associated with infant diarrhoea in nineteenth-century British cities remain unknown (Hardy, 2014, pp. 71-3).

The precocious decline of the most waterborne diseases is consistent with the chronology of improvements in urban water supplies and sanitation. Most towns prioritised clean water supplies over efficient disposal of faecal waste. By the early nineteenth century many towns already had conduits built in earlier centuries to bring clean spring water from a distance into the town, and many acquired public or private waterworks in the first half of the nineteenth century (Hassan, 1985). These initially supplied piped water to more affluent houses and to industries, but also supplied street standpipes serving multiple houses. Dissatisfaction with the quantity and quality of these water supplies (for industrial and fire-fighting purposes as well as domestic demands) led in the majority of cases to municipalisation of waterworks (Hassan, 1985). Ironically, the supply of piped water led in some cases at least initially to higher exposure to faecal-oral diseases. This is because the greater abundance of domestic water made it possible for wealthier inhabitants to install flush toilets, and this led to the release of untreated sewage into local waterways that also often served as water sources. The advent of piped water supplies could also operate to amplify the effects of faecal-oral diseases, because any such contaminated water was then pumped efficiently to hundreds of households. This contrasts with the contamination of local public or private wells, because these usually served a much smaller proportion of the population and therefore caused more localised outbreaks (Davenport et al., 2019).

Table 1. Pathogens with faecal-oral transmission routes

Pathogen	*Mortality (% of cases)*	*Waterborne outbreaks (% of all outbreaks)*
Vibrio cholerae, classical biotype [Asiatic cholera]	15.7	83.3
Shigella dysenteriae type 1 [dysentery]	7.5	80
Salmonella typhi [typhoid]	5.8	74
Vibrio cholera, el tor biotype	1.44	50
Shigella flexneri	1.32	48.3
Shigella sonnei	0.65	27.8
Enterotoxic *E. coli*	<0.1	20
Campylobacter jejuni	<0.1	10.7
Non-typhoid salmonella	<0.1	1.6

Source: Ewald, 1991, pp. 83–119.

Although a direct role for faecally contaminated water in disease was not widely recognised before the 1860s, associations between dirty water and ill health were of longstanding (Rawcliffe, 2013; Geltner, 2019; Gentilcore, 2020). By the 1850s many of the largest towns had therefore adopted two main strategies to obtain clean water: the sourcing of remote upland or deep aquifer waters (as in Glasgow, Liverpool, Manchester and Birmingham); and the filtration of more polluted water from rivers and streams (as in London). Waterworks often relied on a mix of water sources that could include both cleaner spring or upland water and dirtier river water. Water shortages during periods of drought could also force towns to resort temporarily to less salubrious sources. These circumstances generally occurred in the summer, when poor quality water sources were made more dangerous by high temperatures. Nonetheless, purer sources gradually replaced the more polluted, and piped in-house supplies were gradually extended to all households. By the time of the first comprehensive survey of water supplies in 1911, almost all households in larger towns (population 20,000 or more) had access to piped water (Table 2). This near-ubiquity of piped water in larger urban centres stood in stark contrast to rural areas, where piped water was a rarity and most communities still relied on traditional sources (*Water undertakings*, 1914).

Table 2: Piped water supplies and flush toilets in English and Welsh towns with populations of 20,000 or more, 1911. Values indicated the percentage of towns where under 50 %, 50-94% or 95-100% of all households had access to piped water or flush toilets (N = 209).

	Percentage of towns in each category	
Percentage of households served	Piped water supply	Flush toilets
<50	0	18.2
50-94	2.4	25.4
95-100	97.6	56.4

Sources: *Water undertakings*, 1914; Local Government Board (1913)

The early development of communal water supplies was not matched by an equal attention to the efficient removal of faecal waste from urban environments. Before the widespread availability of piped water, all towns depended on so-called ‘dry’ faecal disposal methods. These involved storage of faeces and other household waste in large pits, called midden privies or cesspits, which were only emptied when they were full. From the mid-nineteenth century, as piped water supplies increased, British towns generally pursued one of two strategies. Some towns, including Bristol and London, opted for flush toilets (called water closets) and sewerage, which removed faeces rapidly from households, but also led to sewage pollution of rivers and coastal areas. However many especially inland towns preferred to install improved ‘dry’ storage methods, which usually involved toilets with movable pails (see Figure 2).[2] These pails were emptied and replaced at frequent intervals, often once or twice a week. These methods were a notable improvement on midden privies, but still meant that large volumes of faeces were stored in or near houses. Corporations that opted for these improved dry storage methods generally justified their strategy on one or more of four grounds; that dry disposal held the possibility of recouping the costs of collection via sale of 'night soil' to farmers; that town water supplies were insufficient to supply flush toilets in all houses; that poorer households would misuse flush toilets; and that water-based disposal of sewerage would exacerbate the pollution of rivers.

Figure 2. Rochdale pail closet system. Urine and faeces were collected in the pail under the toilet seat, and household refuse in the larger barrel behind, and these were emptied by the local council or private contractors at regular intervals.
Source: Gray, S.M. (1884) https://archive.org/stream/proposedplanfor01graygoog#page/n45/mode/1up
[Gray, S.M., (1994) Proposed plan for a sewerage system and for the disposal of the sewage of the city of Providence (Providence, Providence Press)]

Towns that relied on dry or so-called 'conservancy' faecal disposal methods were widely considered to be associated with higher rates of diarrhoeal and typhoid mortality. Medical Officers of Health (MOH), the local officials responsible for public heath, frequently published evidence of higher disease and mortality rates in 'conservancy' compared with 'water closet' towns in their annual reports. Many also documented the spatial association of typhoid cases and deaths with the prevalence of middens

[2] See Crook (2016) chapter 5 for a concise history of the development of dry and water-based faecal disposal technologies.

or privies within their own towns. Typhoid was considered endemic in most larger towns in the late nineteenth century, however by the 1890s significant surges in cases and deaths were investigated by Local Government Board inspectors. Inspectors always checked the water supply first, and some of these outbreaks were traced to contamination of local water supplies (including in some cases direct use of river water and private wells, as opposed to piped water from waterworks). However most outbreaks were not traced to drinking water (Boobbyer, 1897). Therefore by the turn of the century many commentators recognised that the quality of piped drinking water was not, or was no longer, the major factor in diarrhoeal or typhoid mortality. Instead, many MOHs associated both typhoid and diarrhoeal mortality with poor sanitation, in particular with the methods that towns used to dispose of faeces (Hardy, 2014: 32; Local Government Board, 1910a: xi; Boobbyer, 1894; Niven, 1910).

By the mid-1890s a widespread consensus had emerged that flush toilets were both preferable for public health and practicable (Boobbyer, 1894). Most of the larger towns that still relied on dry faecal disposal methods therefore embarked on programmes of conversion to flush toilets, and these programmes were often impressively rapid and aggressive. This paper addresses the question of whether these programmes were effective in reducing diarrhoeal mortality or typhoid incidence. I first present cross-sectional evidence of associations between sanitation and diarrhoeal and early childhood mortality in a large sample of English and Welsh towns in the years 1907-12. I then present longitudinal evidence for a panel of ten towns for which annual information on faecal disposal methods and faecal-oral disease outcomes was available.

Sources and methods

Faecal disposal methods in towns 1907-12

It is difficult to track precisely the development of sanitary provision in British towns, because the sources are patchy and idiosyncratic, and because the changes were incremental. The first systematic survey of sanitation in English and Welsh towns was only published in 1913, as a supplement to the *Annual report of the Local Government Board* (hereafter the 'LGB report') for 1912/13 (Local Government Board, 1913). This report published information on types of toilet in all towns with populations of 20,000 or more (comprising 240 Urban Districts, Boroughs, County and Metropolitan Boroughs), together with more patchy information on rubbish disposal and infant welfare programmes. The information on waste disposal arrangements and on mortality was compiled for the LGB report from the annual reports of Medical Officers of Health for all 240 towns. While the mortality data were fairly complete, the information available regarding faecal and especially rubbish disposal were more varied in format and less complete. Metropolitan Boroughs were excluded from the analysis presented here, because water and sanitary provision in London were administered by a variety of organisations with varied administrative geographies, and their analysis requires separate study.[3] This left 213 urban units in the sample. These units were then linked to data on water supplies for urban districts in 1911 (see Table 2). Four towns in the LGB report could not be linked to water data (see below), reducing the sample to 209 towns.

The 1913 LGB report reported average infant mortality (deaths under one year per thousand births) and early childhood mortality (deaths at ages 1-4 years per population aged 1-4 years) in towns for the period 1907-10, when summer temperatures were fairly average, and also infant mortality in the years 1911, when summer temperatures were well above average and diarrhoeal mortality was high, and 1912. These rates were used as outcome variables in separate models.

Faecal exposure was measured as the proportion of all toilets reported in each town that were water closets (flush toilets). Faecal disposal methods were reported for most towns in terms of counts of flush toilets, privies, pan privies, cesspools and middens, rather than counts of the numbers of households served by each type of toilet. The distinction is an important one, because in poorer areas multiple households often shared toilet facilities, and this type of arrangement was associated by commentators with poorer hygiene. Conversely, affluent households could have more than one water closet (although this was rarer). My explanatory variable did not capture these dimensions of toilet access. In addition, some reports distinguished between fresh-water and waste-water water closets. Fresh-water water closets were connected to constant running water and could be flushed immediately after use so that faeces were not retained in or near the house. Waste-water water closets on the other hand were not connected to a constant water supply and used waste water from other domestic activities. This meant that faeces were often retained until water was available, and these types of toilet (hand-flushed and trough water closets) were associated by MOHs with lower standards of hygiene. Again, my explanatory measure does not distinguish between these types of water closet.

Table 3 presents descriptive statistics for the cross-sectional sample. The percentage of all toilets that were flush toilets was modelled as a categorical variable because the distribution of values was highly skewed towards high values. Models also included a variable for waterworks ownership (municipal or private), derived from the survey of water supplies undertaken in 1911 (*Water undertakings*, 1914). As indicated in the introduction, most towns in the sample were supplied completely with piped water

[3] London is also much better studied than the rest of the urban hierarchy in England and Wales. See Rafferty, 2021 for recent work on London.

(Table 2), and so I excluded this variable from analyses. ‘Control’ variables included town populations in 1911 and crude birth rates in 1911. Mortality was not linearly related to population size and so population was modelled as a categorical variable (Table 3). Data on rubbish disposal were too patchy to be included. Data were modelled using ordinary least squares multivariate regression models. The models were described by the equation

$$M_i = \alpha + \beta_1 WC_i + \beta_2 Pop_i + \beta_3 CBR_i + \beta_4 WW_i + \varepsilon_{it}, \quad (1)$$

where M_i is the mortality rate for town *i*, β_1- β_4 are coefficients, WC_i, Pop_i and WW_i are categorical variables describing respectively the percentage of toilets that were flush toilets, the population in 1911, and the type of waterworks ownership in town, CBR_i is the crude birth rate (births per 1,000 total population) in town *i* in 1911, and and ε_{it} is the residual.

Table 3. Descriptive statistics for a cross-sectional sample of English and Welsh towns with populations 20,000 or more (excluding London), 1907 – 1912 (N = 209).

Variable		*Mean*	*Std. Dev.*	*Min value*	*Max value*
Infant mortality 1907-10		114.9	24.2	66.8	189.0
Infant mortality 1911		131.9	29.2	66.0	218.0
Infant mortality 1912		96.7	22.9	44.0	166.0
Early childhood mortality 1907-10		63.2	20.9	24.8	122.8
Infant diarrhoeal mortality 1907-10		14.1	6.9	1.8	47.1
Crude birth rate 1911		24.0	5.0	13.6	35.6
% piped water		99.1	3.5	61.3	100
		no of towns in category		*% of towns in category*	
% flush toilets	*0-49*	38		18.2	
	50-89	44		21.1	
	90-99	45		21.5	
	100	82		39.2	
Population (1,000s)	*20 – 49*	106		54.4	
	50 – 99	49		25.1	
	100-199	28		14.4	
	200+	12		6.2	
Waterworks	*Private*	42		21.5	
	Public	130		66.7	
	Mixed	23		11.8	

Sources: Water undertakings, 1914*;* Local Government Board (1913).

Panel study of faecal disposal, infant mortality and typhoid in 10 towns, 1895-1911

To study the relationship between *changes* in faecal disposal methods and health improvements I constructed a panel of ten towns for which I could identify fairly complete annual data on faecal disposal methods between 1895 and 1911 in MOH reports. These towns were among the largest non-metropolitan towns in England, and were all included in the Registrar-General's *Weekly Returns* for 'Great Towns' from 1870. The MOH reports and *Weekly Returns* referred throughout to the Urban Sanitary District area (usually coterminous with the municipal borough boundaries). This study therefore sidesteps the problem that has dogged most demographic studies of Victorian England, which is the mismatch between the registration district units used by the Registrar-General to report demographic measures, and the geographical units in which reforms were undertaken (in the case of public health, the urban or rural sanitary district). The study covers the period 1895-1911, and therefore also avoids the period of extensive boundary changes in the early 1890s. The towns included, and the populations of the boroughs in 1911, were Bristol (357,059), Leicester (227,242), Manchester (714,427), Newcastle (266,671), Norwich (121,493), Nottingham (259,942), Portsmouth (231,165), Salford (231,380), Sheffield (454,653) and Sunderland (151,162).

Outcome variables were diarrhoeal mortality (all diarrhoea deaths per 1,000 births), infant mortality and crude typhoid case and death rates (per 1,000 population). Diarrhoeal deaths (at all ages) were reported in the *Weekly Returns*. These data were converted to annual totals and to rates using annual totals of births reported in the same source. By the 1890s infants accounted for around 80 % of diarrhoeal deaths nationally, and therefore I used births rather than total population as the denominator for the diarrhoeal mortality rate. Use of total population as the denominator would have produced an artifactual decline in diarrhoeal mortality over time due to strong fertility declines over the period of the study, that reduced the size of the infant population (the main source of diarrhoeal deaths) relative to the total population. All towns in the sample voluntarily adopted compulsory notification of infectious diseases before 1895[4], and their MOH reports listed counts of typhoid cases as well as typhoid deaths (the diagnosis of diarrhoea and typhoid is discussed below). Annual

[4] The Infectious Disease (Notification) Act of 1889 made notification of certain diseases compulsory in London but not elsewhere: compulsory notification was extended to the country as a whole under an extension Act of 1899.

population totals were estimated by geometric interpolation between the censuses of 1891, 1901 and 1911, and were used to convert typhoid cases and deaths into crude rates.[5]

The main explanatory variable of interest was the percentage of all toilets that were water closets (fresh or waste water flushed). I used fixed effects models to address the problem of omitted variables, most notably breastfeeding practices (this is discussed further in the Results section). Models were also adjusted for time-varying factors that may have affected mortality. These included population, crude birth rate, and average wealth, which was proxied by the assessable value of all real property in each town per capita. Descriptive statistics are presented in Table 4. The models are described by the equation:

$$M_{it} = \alpha + \beta_1 WC_{it-1} + \gamma X_{it} + \eta_i + \lambda_t + \varepsilon_{it}, \tag{1}$$

where M_{it} is the mortality rate for town *i* in year *t*, WC_{it-1} is % flush toilets in period *t-1*, X_{it} is a vector of controls, η_i is a town fixed effect, λ_t is a time effect, and ε_{it} is the residual.

Table 4. Descriptive statistics for ten panel towns, 1895-1911.

Variable	*Mean*	*Std. Dev.*	*Min value*	*Max value*
Infant mortality	159.5	28.9	90.7	230.3
Infant diarrhoeal mortality	35.3	16.0	5.7	69.7
Typhoid cases/1,000	1.03	0.85	0.13	6.13
Typhoid deaths/1,000	0.16	0.13	0.02	0.94
% flush toilets	63.5	30.4	9.1	100
Crude birth rate	29.8	3.4	21.7	36.5
Population	270,737	141,758	105,143	714,427
Assessable value per capita (£)	5.3	0.9	3.5	7.7

Notes: Towns are Bristol, Leicester, Manchester, Newcastle, Norwich, Nottingham, Portsmouth, Salford, Sheffield and Sunderland.
Sources: Medical Officer of Heath reports for Bristol, Leicester, Manchester, Newcastle, Norwich, Nottingham, Portsmouth, Salford, Sheffield and Sunderland; *Local Taxation Returns*; *Weekly Returns; Censuses of Great Britain, 1891-1911*.

[5] In cases where a boundary change occurred between census dates, I used the ratio of the number of (weekly) births in the 52 weeks before the boundary change to the number in the 52 weeks following the change to estimate the change in population. I then used the intercensal growth rate in the decade closest to the change to either forward-project or back-project the nearest census population to the date of the boundary change to estimate the population at the date of change. I estimated the population before or after that date from the ratio of births before and after the boundary change. See Aidt et al., 2022, fn. 44.

Figure 3 shows trends in diarrhoeal mortality, typhoid case rates and flush toilet provision in the panel sample. While levels of diarrhoeal mortality varied markedly between towns, trends were broadly similar, with a peak in the last years of the 1890s, when summers were unusually hot, a second peak c. 1905, and an abrupt rise in 1911, when most of Europe experienced a very hot summer and widespread diarrhoeal epidemics. Typhoid case rates also varied substantially in absolute terms, with very large epidemics in Portsmouth (1900) and Sunderland (1895). However by 1911 typhoid rates had converged to very low levels in all ten towns.

The red lines in Figure 3 indicate the percentage of toilets that were flush toilets (fresh or waste-water water closets). Two towns, Bristol and Portsmouth, were early to adopt flush toilets, and MOH reports indicate that all toilets were flush toilets throughout the study period. It is quite likely that toilet provision still improved across the period in these towns as older water closets, some hand-flushed, were replaced with more sanitary freshwater ones, but the MOH reports did not document this. Four towns, Manchester, Norwich, Salford and Sunderland, relied very heavily on privy pails and middens in the 1890s, but undertook very large-scale and intrusive campaigns of conversion to flush toilets in the early 1900s. Nottingham on the other hand was notoriously slow to adopt flush toilets, despite the very active campaigning of its MOH, Philip Boobbyer.

Diagnoses of diarrhoea and typhoid

Historical cause of death statistics are problematic because causes were often poorly defined or misattributed, and because diagnostic fashions changed over time. As a cause of death, 'diarrhoea' is a description of symptoms rather than an aetiological category. Until 1911 the Registrar-General's diarrhoeal category included mainly deaths attributed to 'epidemic diarrhoea', and the reported deaths were highly concentrated in very sharp late summer epidemics. From 1911 onwards however the International Classification of Diseases (ICD) system was adopted. The new ICD scheme distinguished between 'epidemic diarrhoea' and 'enteritis', however these were subsequently grouped together in a new 'diarrhoeal' category in the Registrar-General's annual reports. Fortunately however, the older definition, which corresponded largely to 'epidemic diarrhoea', was used in the 1912 report which is the basis of the cross-sectional analysis presented here, and in the *Weekly Returns* from which the panel data derive. While the pathogens that caused these summer diarrhoeal outbreaks were never satisfactorily identified (Hardy, 2014: 71-3), the diagnostic term seems to have described a consistent and discrete phenomenon between at least the 1840s and 1911.

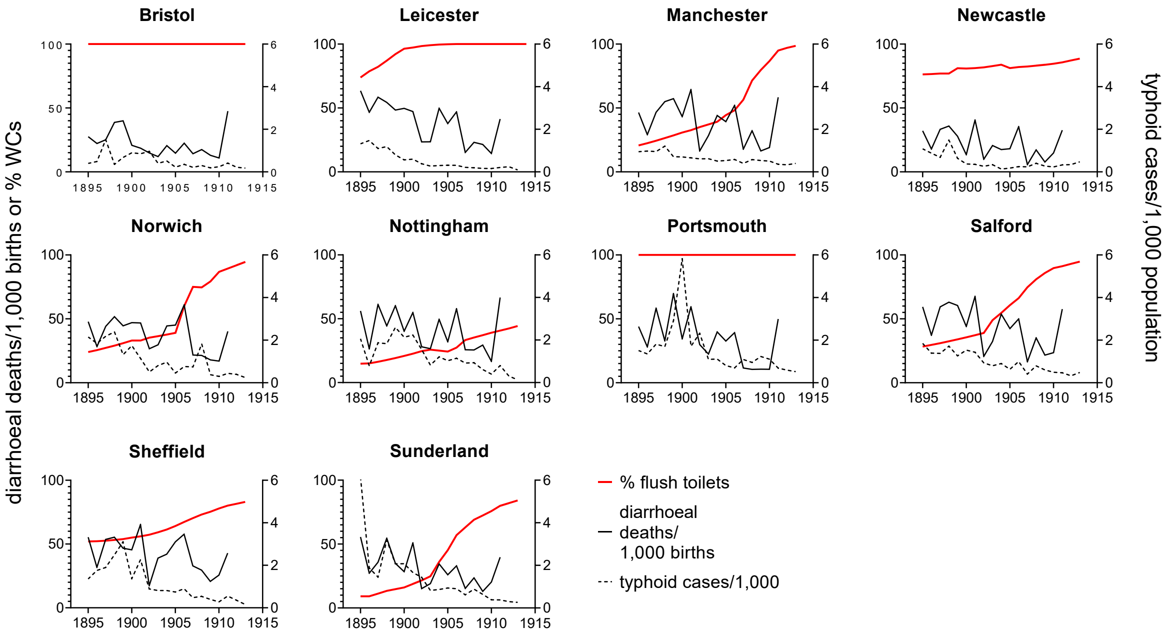

Figure 3. Diarrhoeal mortality, typhoid case rates and water closet provision in ten English towns, 1895 – 1913. Notes: Diarrhoeal mortality includes diarrhoeal deaths at all ages per 1,000 births for the years 1895-1911. Typhoid rates refer to all cases per 1,000 population, 1895-1911. Flush toilets are expressed as a percentage of all types of toilet, 1895-1913.
Sources: *MOH reports* 1895-1913; *Weekly Returns of the Registrar-General*, 1895-1911.

In the case of typhoid there was more diagnostic confusion. Typhoid and typhus were not distinguished diagnostically before the 1830s (Wilson, 1978). Deaths attributed to each disease were only reported separately by the Registrar-General from 1869. Moreover 'typhoid' was used fairly interchangeably with 'enteric fever', although the latter category was also used to refer to both typhoid and (generally milder) paratyphoid infections. An ingenious diagnostic test for enteric fever, the antibody coagulation-based Widal test, was developed in 1896 and became very widely used. When the test was used by MOHs they generally reported that most but not all notified cases of typhoid were confirmed as typhoid (or enteric fever). Interestingly, the use of the test does not seem to have altered notification practices – suspected cases were still notified, and reported in annual MOH reports, even where subsequent administration of the test did not indicate typhoid or paratyphoid. Where I could find data, the ratio of confirmed to negative tests remained fairly constant across the period (data not shown). Similarly, case-fatality rates remained remarkably consistent over the period 1895-1911, even as rates dropped substantially. Figure 4 shows moving five year averages of case-fatality rates (typhoid deaths per 100 cases) in the ten panel towns. While there were clear persistent differences in average case-fatality rates between towns, with averages ranging from around 10 to 25 deaths per 100 cases, there was no obvious trend over the period 1895 – 1913.

Perhaps more surprisingly, given the marked declines in typhoid incidence over the period of the study, the seasonal pattern of typhoid deaths also seems to have been stable over the period. Typhoid deaths tended to peak in autumn, in contrast to diarrhoeal deaths. These seasonal patterns are based on weekly reports of deaths attributed to 'fever', because typhoid deaths were not distinguished in the Registrar-General's weekly returns until 1911. 'Fever' was broadly used to describe deaths due to typhoid, typhus and 'continued fever' in this period. However comparison with quarterly notifications of these diseases, reported separately under the heading of 'fever' in Local Government Board (LGB) reports, indicates that the vast majority of cases attributed to fever were described as typhoid. Therefore it is plausible to assume that seasonal patterns of 'fever' deaths in the largest towns reflected patterns of typhoid mortality in this period. Moreover, weekly deaths from typhoid were reported for London districts in the LGB reports, and these display the same autumnal seasonality as deaths from fever in other towns.

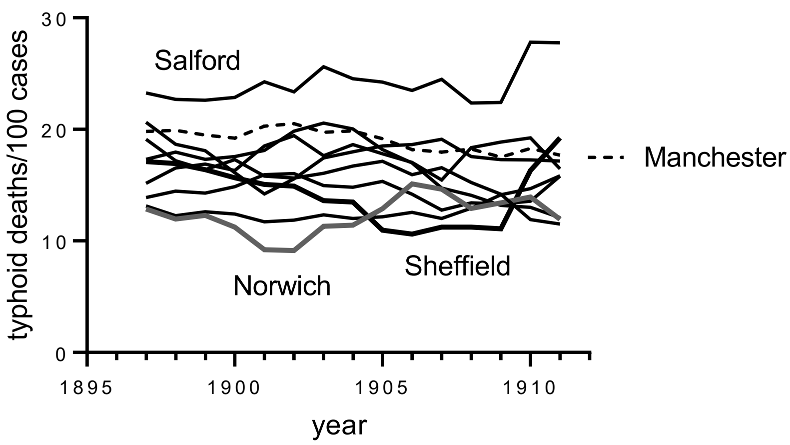

Figure 4. Case-fatality rates in ten panel towns, five year moving means.
Source: Annual MOH reports.

The stability of typhoid case-fatality rates, seasonal mortality patterns and notification practices suggest that diagnostic practices did not change markedly during this period, at least in the panel towns. Thus while diagnoses of typhoid often proved wrong, in using reported typhoid cases or deaths we are probably measuring the same phenomenon over time. That is, the typhoid case rates reported by MOHs are probably fairly representative of trends in typhoid incidence, even if they over- or underestimate the true incidence.

Results

Cross-sectional models of faecal disposal methods and early life mortality

Table 5 reports the results of multivariate OLS modelling of the relationship between flush toilets and mortality outcomes, adjusted for crude birth rate, population and waterworks ownership. The variable representing the percentage of houses supplied by piped water was excluded because it showed no relationship with mortality in any of the models. In cross-sectional analyses, a high prevalence of flush toilets in 1911 (above 90% of all toilets) was associated with markedly lower infant mortality in all periods (1907-10, 1911 and 1912). This effect was graduated to some extent, in that towns with complete flush toilet provision had lower infant mortality than towns with 90-99 % provision. However dividing the categories more finely (for example by deciles) did not produce a more linear relationship between flush toilet prevalence and infant mortality (data not shown), perhaps because the numbers of towns in some deciles were very small (reflecting the skewed distribution of values). In towns with completely waterborne faecal disposal, after adjustment for population, birth rate and waterworks ownership, infant mortality was predicted to be 18 – 26 deaths/1,000 lower than in towns where less than half the toilets were water closets (a difference equivalent to almost a fifth of the average infant mortality rate: see Table 3). The predicted reduction in infant mortality was largest in 1911 (at 26 deaths/1,000), the year when diarrhoeal rates were highest. Flush toilet provision also showed a strong negative relationship with mortality in early childhood (ages 1 -4 years). Surprisingly however, infant diarrhoeal mortality was not strongly associated with flush toilets. Coefficients were negative, for values of percentage flush toilets that were 90 % or above, however the relationship was statistically weak.

Waterworks ownership was also closely associated with infant, early childhood and diarrhoeal mortality, in adjusted models. Compared with public waterworks, private waterworks, or supply from both public and private waterworks, were generally associated with lower rates of mortality. This was at first sight surprising, because municipalised waterworks have generally been associated by historians with improvements in the purity, volume and coverage of water supplies. Larger towns were more likely to have public waterworks (32 of the 40 towns with populations 100,000 or more had public waterworks, compared with 98/155 smaller towns), and larger towns also experienced higher child mortality rates on average (as indicated by the significant positive coefficients for population of 100,000 or more in most models). Therefore public ownership of waterworks may have been acting as a proxy for larger, denser urban populations in the models (despite the inclusion of population as a variable). However when the larger towns were excluded then the advantage of private and mixed supplies persisted (data not shown). If this result does reflect an advantage to private waterworks ownership (as opposed to proxying some other favourable characteristics of these towns) then this suggests that the long process of municipalisation of waterworks over the course of the nineteenth century may have exerted a strong selective pressure on private waterworks, such that only the most high-performing private waterworks avoided municipalisation by 1911. Alternatively, lower infant and diarrhoeal mortality in towns with private waterworks could be a product of endogeneity, if towns with lower mortality (for whatever reason) were less motivated to change their water provider. Brian Beach and colleagues found that in nineteenth century England and Wales municipalisation was associated with rising typhoid death rates in the years leading up to municipalisation. Municipalisation caused substantial reductions in typhoid mortality, although not enough to confer an absolute advantage on towns with municipalised waterworks compared with those that remained private (Beach et al., 2016). Their results strongly suggested that typhoid mortality acted as a driver of municipalisation in Britain. It is not clear that infant and diarrhoeal mortality rates were similarly used to motivate arguments for municipalisation of waterworks, however where typhoid and diarrhoeal mortality were correlated (for whatever reason) then this could produce a potentially spurious association between high diarrhoeal mortality and municipalised waterworks.

Taken together the results suggest that both water supply (waterworks ownership) and faecal disposal methods were associated with mortality variations between towns. However while these associations were clear in the case of infant and early childhood mortality, faecal disposal methods were only weakly associated with diarrhoeal mortality. This is a puzzling result, because we would normally assume that the main effects of sanitary provision on infant and childhood mortality would operate via reductions in diarrhoeal mortality. However this is a cross-sectional study, and we lack some key variables that we might expect to influence diarrhoeal mortality, and that might distort the relationship between a town's sanitation and mortality.

The most obvious missing variable is information on breastfeeding. The large majority of infants who died of diarrhoea were artificially fed. Surveys by MOHs of local breastfeeding practices and infant diarrhoeal deaths found that breastfeeding was generally very common at least in the first six months of life, and that diarrhoeal mortality was highly concentrated amongst weaned or never-breastfed babies. Therefore variations in the prevalence of breastfeeding between towns could have had large effects on diarrhoeal mortality. Bob Woods' survey of such data from English towns in the late nineteenth and early twentieth centuries indicated no obvious geographical patterns (Woods, 2000: 284-8). However such reports were infrequent and unstandardised, and we lack consistent and comparable measures of breastfeeding prevalence for the towns in our sample.

Table 5. Regression modelling of faecal disposal methods and early life mortality in a cross-sectional sample of 195 towns, 1907-12.

	Dependent variables				
	IMR 1907-10	*IMR 1911*	*IMR 1912*	*ECMR 1907-10*	*Infant diarrhoea 1907-10*
% flush toilets					
0-49	1.0	1.0	1.0	1.0	1.0
50-89	4.1	5.8	0.0	-2.3	1.6
90-99	-8.6*	-13.8*	-8.8*	-16.2***	-1.4
100	-20.0***	-25.9***	-18.0***	-23.1***	-2.3
Crude birth rate	2.1***	1.7***	2.1***	1.5***	0.48***
Population (1,000s)					
20-49	1.0	1.0	1.0	1.0	1.0
50-99	-1.6	3.1	-4.8	2.0	0.2
100-199	2.3	8.2	-2.3	2.9	1.0
200+	14.3*	18.0*	9.7	17.9***	3.6
Waterworks					
Public	1.0	1.0	1.0	1.0	1.0
Mixed	-12.5**	-8.9	-9.3*	-5.1	-3.0*
Private	-12.1***	-10.4*	-5.6	-10.2***	-3.9***
Adjusted R^2	0.46	0.34	0.42	0.46	0.23

Notes: Models are Ordinary Least Squares regression models. IMR = infant deaths per 1,000 births in the same period; ECMR = deaths at ages 1 – 4 years per 1,000 children aged 1 – 4; infant diarrhoea = diarrhoeal deaths aged under one year per 1,000 births. * P < 0.05, ** P < 0.01, *** P < 0.001.
Sources: see Table 3.

Fixed effects modelling of faecal disposal methods and mortality in ten towns

To address the problem of omitted variables we can turn to the panel dataset, where we can employ fixed effects models. Fixed effects models adjust for time-invariant fixed characteristics of the towns in the sample, by centring all variable values on the mean values for each town. Thus fixed effects models can adjust for differences between towns in breastfeeding patterns on the assumption that these/ patterns were fairly stable within each town over the period 1895-1911. This is a strong assumption, but on the other hand we have no *a priori* reasons to think that breastfeeding patterns changed in this period (Woods, 2000: 284-8).[6] In some models I also include time fixed effects to control for interannual variations that affected all towns, such as hot summers or new health

[6] Valerie Fildes made the argument, based on similar sources of evidence, that there was a major change in methods of feeding artificially-fed babies in the first two decades of the twentieth century, from the use of long-necked feeding vessels that were difficult to clean, to more hygienic boat-shaped vessels, a change she associated with falls in infant mortality (Fildes, 1998). However this type of change may have been common to all towns in the sample.

technologies. All models were adjusted for population growth, tax base and crude birth rates. Unfortunately we did not have evidence of changes in water supply over the period. All towns in the sample were characterised by piped supply to all households by 1911, but we don't know when this was achieved. The towns varied in the ownership of waterworks (public or private), but no municipalisation occurred in the panel towns within the period of the study, and so waterworks ownership type was incorporated as a fixed effect. The main explanatory variable was the percentage of toilets that were flush toilets, and this was measured at the end of each year prior to the year in which the deaths or cases occurred.

Table 6 shows the results of modelling the relationship between the percentage of all toilets that were flush toilets, and diarrhoeal mortality. Model 1 is adjusted for control variables (population, tax base and crude birth rate) but excludes fixed effects, and shows a strong negative relationship between the percentage of flush toilets and diarrhoeal mortality. However the (total) R^2 value is relatively low ($R^2 = 0.12$), indicating that the model did not account for much of the variation in rates. Model 2 included town fixed effects, to adjust for differences between the towns that were time-invariant. The relationship between flush toilets and diarrhoeal mortality remained strongly negative, but the model fit was still relatively poor (within towns $R^2 = 0.14$).

Table 6. Fixed effects regression modelling of faecal disposal methods and diarrhoeal and infant mortality in a panel of ten towns, 1895-1911.

	Dependent variables					
	Diarrhoeal deaths/1,000 births			*Infant deaths/1,000 births*		
	(1)	(2)	(3)	(4)	(5)	(6)
% flush toilets	-0.19**	-0.23*	-0.08	-0.47***	-0.53*	-0.16
Tax base	-2.10	-1.36	1.39	0.15	-7.10	3.33
Crude birth rate	0.66	0.20	-0.42	4.85***	3.80**	0.42
Population (1,000s)	0.00	-0.08	0.01	-0.04	-0.19	0.00
Town FE	NO	YES	YES	NO	YES	YES
Year FE	NO	NO	YES	NO	NO	YES
R^2	0.12	0.14	0.79	0.40	0.59	0.85

Notes: R^2 is for within towns (for fixed effects models) or total variation (models 1 and 4). * P < 0.05, ** P < 0.01, *** P < 0.001.
Sources: see Table 4.

Model 3 includes two-way fixed effects for town and year, to adjust for trends or variations between years that affected all towns. Inclusion of year fixed effects dramatically improved model fit (R^2 = 0.79), and the effect of flush toilets became much smaller and no longer statistically strong (P = 0.37). This implies that the apparent relationship in models 1 and 2 between increasing provision of flush toilets and improvements in diarrhoeal mortality was probably driven in the main by annual variations that affected all towns very similarly. The importance of yearly variations was unsurprising, because most of the inter-annual variation in diarrhoeal mortality was caused by variations in summer temperatures, and average summer temperatures tended to vary fairly synchronously across England. The importance of summer temperature is illustrated in Figure 5. The solid black line plots the year coefficients from Table 5 model 3, and the grey line plots the number of weeks per year when the mean central England weekly temperature exceeded 15.5 degrees Celsius (60 degrees Fahrenheit). This was roughly comparable to the weekly temperatures at which Walker Hanlon and colleagues reported marked rises in infant mortality in London in the same period (Hanlon et al., 2021). The two series are fairly well-synchronised, indicating that most of the variation captured by the year fixed effects reflected variations in summer temperatures. The dashed line in Figure 5 shows annual infant diarrhoeal mortality for England and Wales. Although the model coefficients are derived from modelling of all diarrhoeal deaths in only ten towns, they clearly capture most of the variation in infant diarrhoeal mortality in the country as a whole. The implication is that there was very little improvement in diarrhoeal mortality over the period 1895-1911, once variations in summer temperatures were accounted for. That is, even very marked improvements in faecal disposal methods, as witnessed in some of the towns in our sample, did little to improve diarrhoeal mortality, in our sample.

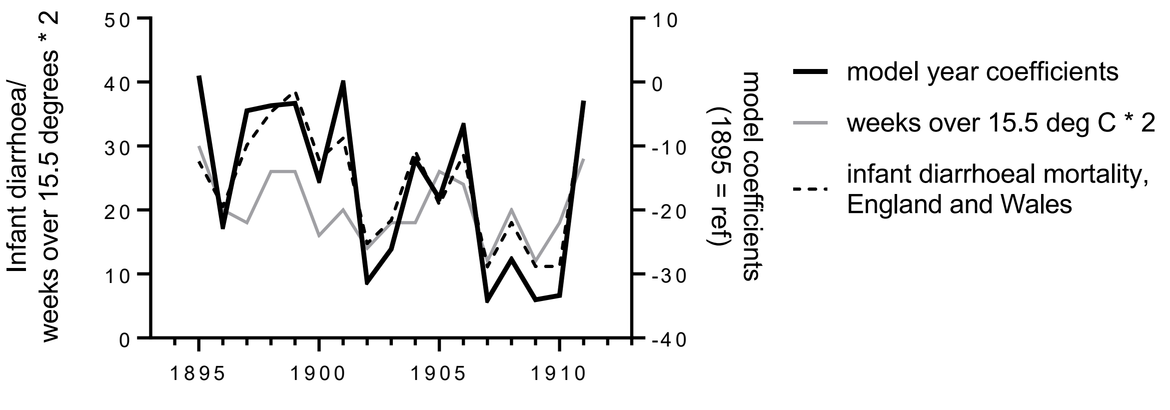

Figure 5. Year fixed effects, summer temperatures and infant diarrhoeal mortality, 1895-1911.
Notes: Temperatures refer to Central England mean weekly temperatures (CET series) (multiplied by 2).
Sources: see Table 6 (for model coefficients), Parker et al. (1992) (CET series), Davenport, 2007 (national diarrhoeal rates).

Table 7 shows the results of modelling the relationship between freshwater flush toilets and (logged) typhoid case and death rates. Rates were logged to normalise the distribution of values. In model 1, which excludes fixed-effects (but includes controls for population growth and annual fluctuations in the crude birth rate), there was a strong negative relationship between the provision of flush toilets and typhoid case rates (P= 0.000). This relationship persisted when town fixed effects were included (model 2) (P = 0.008). However once year fixed effects were included (model 3), the relationship between flush toilet provision and typhoid case rates weakened and was no longer statistically significant (P = 0.0223). Models of typhoid death rates exhibited very similar patterns (Table 7, models 4-6). As was the case for typhoid case rates, once fixed effects for town and year were included then the relationship between flush toilet provision and typhoid death rates disappeared (model 6). In both two-way fixed effects models (models 3 and 6) the only variable that remained strongly (and negatively) associated with typhoid case or death rates was the average wealth (or tax base) of the towns. The proportion of typhoid cases that were isolated in the same or previous year was not associated with case or death rates (data not shown).

Table 7. Fixed effects regression modelling of faecal disposal methods and typhoid case and death rates in a panel of ten towns, 1895-1911.

	Dependent variables					
	Typhoid cases/1,000 population			*Typhoid deaths/1,000 population*		
Model	(1)	(2)	(3)	(4)	(5)	(6)
% flush toilets	-0.01***	-0.01*	-0.00	-0.01**	-0.01*	-0.00
Tax base	-0.37***	-0.49**	-0.44**	-0.34***	-0.48**	-0.46*
Crude birth rate	0.12***	0.11**	0.01	0.12***	0.13***	0.01
Population (1,000s)	-0.00	-0.00	-0.00	0.12**	0.08*	0.00
Town FE	NO	YES	YES	NO	YES	YES
Year FE	NO	NO	YES	NO	NO	YES
R^2	0.38	0.56	0.74	0.44	0.53	0.74

Notes: Dependent variables are logged values. * P < 0.05, ** P < 0.01, *** P < 0.001.
Sources: see Table 4.

Figure 6 plots the year coefficients from Table 7 model 6 (for typhoid death rates), together with the number of weeks per year when the mean central England weekly temperature exceeded 15.5 degrees Celsius, and national typhoid death rates. In contrast to diarrhoeal rates, the year coefficients from the typhoid model do not follow the patterns of seasonal variations in summer temperatures. However they are very similar to the national trend in typhoid death rates. The implication is that typhoid rates (deaths and cases) were falling everywhere, and the decline was not notably faster in towns that undertook aggressive programmes of conversion to flush toilets. This is also the impression given by the trends in individual towns shown in Figure 3. Typhoid case rates fell everywhere, and declines were very marked not only in towns such as Manchester that pursued rapid improvements in faecal disposal methods, but also in towns such as Newcastle, Nottingham and Sheffield, where changes were relatively slow, and in towns where flush toilets were already universal (Portsmouth and Bristol).

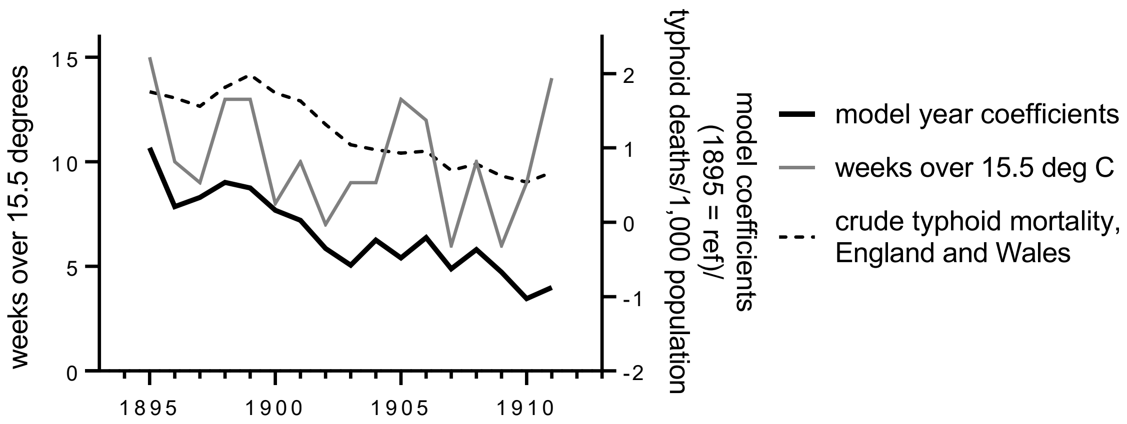

Figure 6. Year fixed effects, summer temperatures and typhoid case and death rates, 1895-1911.
Notes: Temperatures refer to Central England mean weekly temperatures (CET series).
Sources: see Figure 5.

Discussion

This paper addressed the question of whether variations in faecal disposal methods in English towns in the late nineteenth and early twentieth centuries were associated with differences in faecal-oral disease rates (measured as diarrhoeal mortality, or typhoid case and death rates). A cross-sectional analysis of a large sample of towns with populations of 20,000 or more indicated that complete or near-complete provision of flush toilets was associated with lower infant and early childhood mortality in the period 1907-10 and in 1911, however this pattern was not explained by the influence of flush toilet provision on variations in diarrhoeal mortality, because diarrhoeal mortality was not apparently related to the prevalence of flush toilets (or privies), in adjusted models.

Analysis of a much smaller panel of towns that included some of the largest towns in England (excluding London) also indicated that provision of flush toilets was not closely associated with improvements in diarrhoeal mortality or typhoid incidence or mortality in the period 1895-1911, once annual variations common to all towns were taken into account. In the case of diarrhoeal mortality, annual fluctuations in rates were closely associated with variations in summer temperatures, and the impact of summer conditions was apparently very similar across the towns in the sample and in the country as a whole (Figure 5). The impact of summer temperatures on diarrhoeal mortality was very widely appreciated by contemporaries, but the causal relationship between unusually high or low summer temperatures and diarrhoeal diseases remained hotly contested. High temperatures increase the rate at which pathogens multiply on food and drink, and temperature is still strongly correlated with food poisoning incidence. However temperature also influences the growth rates of insect vectors, and many MOHs argued that house flies were the main cause of faecal contamination of food and drink during hot summers (Hardy, 2014, chapt. 3).

An important clue to the drivers of infant diarrhoeal mortality must lie in the timing of declines in diarrhoeal mortality. While infant mortality from non-diarrhoeal causes fell from the late nineteenth century (Woods, 2000: 275; Hanlon et al., 2021), diarrhoeal mortality does not appear to have declined markedly before the second decade of the twentieth century (although annual variations in summer temperatures meant that diarrhoeal rates were highest in the late 1890s and fell subsequently). Recent work by Hanlon and colleagues demonstrated a persistent effect of high summer temperatures on mortality in London until World War I (Hanlon et al., 2021). This relationship had almost disappeared by 1919, and infant diarrhoea had become a very minor cause of death by 1930 (Graham-Smith, 1929, Hanlon et al., 2021). The present study stopped at 1911 and therefore could not shed light on the changes that occurred in the apparently crucial second decade of the twentieth century. This decade is difficult to study because the war was associated with very marked reductions in the detail provided by MOHs in their annual reports. However future work will use those MOH reports that provide some detail on infant diarrhoeal and sanitary trends to extend the series to 1930.

In the case of typhoid there was a marked decline in typhoid case and death rates across the period 1895-1911. However this trend was common to all towns in the panel sample, and closely matched the national trend in typhoid mortality (Figure 6). Superficially at least, this trend would suggest that broad factors were operating to reduce typhoid infection rates everywhere, regardless of sanitary efforts. This conclusion is supported by the evidence that falls in typhoid mortality between the 1890s and 1900s were of similar magnitude in rural and urban registration districts, despite much more intense sanitary interventions in towns compared with rural areas (Hinde and Harris, 2019). If typhoid rates did indeed fall fairly uniformly regardless of heterogeneous improvements in water quality and sanitation, then this implicates driving forces of broad effect. Candidates include a change in the virulence of the circulating strains, typhoid vaccination, and spillover effects.

Any change in pathogen virulence would normally be expected to affect case-fatality rates. However as Figure 4 indicates, the case-fatality rates reported by MOHs remained fairly unchanged across the period of our study, making a change in virulence unlikely. It remains possible that a change in virulence could have acted to reduce both death rates and cases rates, if a higher proportion of cases then went undetected or misdiagnosed as symptoms became milder. However the fairly constant rate of false positives reported by MOHs across the period also suggests that there was little change in the underlying properties of the pathogens involved in enteric fever. Similar arguments apply to the effects of vaccination. A vaccine against typhoid was developed in 1896 and was used in some civilian as well as military contexts before WWI, although Hardy implies that adoption was low and patchy (Hardy, 2000). The most obvious effect of vaccination would have been to reduce case-fatality rates, and there is no evidence that this occurred in our panel study.

Spillover effects describe the phenomenon of changes in some areas producing wider effects on neighbouring or connected areas. Such effects could have occurred through several mechanisms. In the period 1895-1911 a significant proportion of typhoid outbreaks were traced to contaminated shellfish, including around 30% of all cases in Manchester (Local Government Board, 1910b; Niven, 1923: 39-59; Hardy, 2014: chapt. 2). Oysters and other shellfish were harvested in estuarine areas, and were often contaminated by untreated sewage released from towns upriver. These shellfish were then transported often considerable distances to other towns. For example, mussels harvested in Morecambe Bay were distributed very widely to Manchester and other towns in Lancashire. In this case improvements in sewage treatment in the town of Morecambe could have reduced exposure to typhoid-infected shellfish in a wide area (Local Government Board, 1910b: 225-30). An alternative explanation is that the growing awareness of the association between shellfish and typhoid in the 1890s and especially after 1900, led both to greater inspection of shellfish beds, and to reduced consumption (Hardy, 2003; Hardy, 2014: chapt. 2), both factors that could have produced widespread reductions in typhoid infections without any necessary improvements in sanitary conditions.

Another type of spillover effect could in theory have arisen from the role of human carriers in sustaining typhoid transmission. It remains unclear to what extent human carriers constitute a reservoir of endemic typhoid that contributes to ongoing transmission, and carriers may become more important in sustaining transmission as other sources of infection fall. Local Government Board investigations of typhoid outbreaks in rural communities sometimes traced infection either to a visitor from an urban area or to a local inhabitant who appeared to have contracted typhoid on a visit to an urban area (Local Government Board, 1910a: 116-8). If carriers were important in sustaining endemic infection in towns and in distributing typhoid to other towns and rural areas, then falls in local infection rates due to sanitary improvements in some towns could have reduced the prevalence of carriers and led to wider reductions in the circulation of the disease.

Limitations

The study had a number of important limitations that constrain the conclusions that can be drawn. First, I used flush toilets as my measure of sanitary conditions. As discussed in the Methods section, this measure was an imperfect proxy for faecal disposal facilities for several reasons. First, it was measured as a percentage of all toilets and so did not measure how many households had exclusive access to a flush toilet. Secondly, it did not distinguish between the more sanitary fresh water-flushed toilets and waste-water toilets. This meant that the measure may not have captured some of the differences in sanitary provision between towns, and may have underestimated improvements over time. Almost all the new flush toilets installed over the period 1895-1911 were fresh water-flushed, and these replaced both dry faecal disposal methods and waste water-flushed toilets.

More broadly, changes in toilet type were not the only sanitary improvements undertaken in this period. Many towns undertook reforms of rubbish disposal, including replacement of middens and ash-pits with so-called 'sanitary bins' (lidded movable bins), more regular rubbish collection, and the replacement of rubbish tips with incinerators ('destructors'). These changes were often accompanied by more efficient removal of manure and other rubbish from streets. These types of improvements in rubbish disposal may have helped to reduce fly populations, which have been implicated in the transmission of faecal-oral diseases. Unfortunately, these measures were reported less scrupulously than toilet provision by MOHs, but it may be possible to reconstruct time series for some towns.

Other factors that influenced faecal-oral disease transmission, and that were excluded from our study, were of a more domestic nature. Most notable of these were breastfeeding patterns, which are particularly important to infant and diarrhoeal mortality. While fixed differences in breastfeeding patterns between towns could be accounted for in our fixed-effects models, it was possible that breastfeeding or artificial infant feeding patterns changed over the period of our panel study, and this could have distorted the relationship between diarrhoeal mortality and sanitary improvements. For example, declining duration or prevalence of breastfeeding could have caused rises in the number of infants at high risk of diarrhoea and counteracted any beneficial effects of improved sanitation. This seems unlikely given the striking similarities in time trends in diarrhoeal mortality between towns, but it cannot be ruled out. There may also have been changes over the period of the study in domestic hygiene, such as increased use of soap, or changes in food preparation and consumption patterns (as suggested with respect to shellfish). Such changes may be very difficult to quantify, at least below the national level. Infant welfare programmes were also introduced very widely in the period studied in this paper. These are documented to some extent in MOH reports but are more idiosyncratic to extract and to standardise, something that future work will undertake. It is important to note however that the problem to be addressed with respect to infant diarrhoeal mortality before 1912 is why it improved so little, and also why patterns were apparently so uniform between towns. Therefore the task is likely to involve unpicking the balancing effects of positive and negative influences on infant health, at least before the marked downturn in diarrhoeal rates after 1911.

Conclusion

This paper started with the puzzle of the late decline in infant diarrhoeal mortality, compared with death rates from other types of faecal-oral diseases (including cholera, typhoid and dysentery). I suggested that these diseases improved before diarrhoeal mortality in infants because they depended to a greater extent on the ingestion of contaminated water, and the problem of contaminated drinking water was addressed much earlier in British towns than other sources of faecal contamination. Infants on the other hand probably contracted diarrhoea mainly from milk and other food or household objects that were faecally contaminated because of high exposure to faeces in domestic or neighbourhood environments. Poor toilet facilities could contribute to faecal contamination of domestic environments if for example privy pan had to be carried through the house to be emptied (resulting in some spillage), or if young children or flies had access to faeces in privies. This study therefore sought to test whether variations or improvements in toilet facilities were associated with lower diarrhoeal mortality rates. Typhoid was included in the study because it was the other main type of faecal-oral disease of concern in this period, and because it was expected that trends in typhoid incidence or mortality might help to shed light on patterns of diarrhoeal mortality.

The results so far suggest that the replacement of privy toilets with flush toilets during the period 1895-1911 had little effect on diarrhoeal or typhoid mortality. Diarrhoeal mortality remained high despite large scale replacement of privies, and typhoid incidence and mortality declined regardless of local changes in toilet provision. To some extent these findings simply echo with greater statistical rigour the observations of some contemporaries. For example Charles Niven, reflecting on his experiences as MOH for Manchester, noted that falls in typhoid had preceded the conversion of middens to pails, and later pails to water closets, despite an apparently close coincidences between these factors in cross-section (Niven, 1923: 42-4).

A key step in furthering our understanding of declines in diarrhoeal mortality is to extend the study to the decades after 1911. The secular decline of infant diarrhoeal mortality began during WWI in Britain, and the pattern of summer diarrhoeal epidemics and excess summer mortality amongst infants had largely disappeared by 1930 (Graham-Smith, 1929; Hanlon et al., 2021). A similar pattern occurred with markedly similar timing, in U.S. cities. A recent study by Mark Anderson and colleagues sought to link the disappearance of summer diarrhoeal epidemics in the U.S. to water purification, sewage treatment, or improvements in milk supplies (Anderson et al., 2020). However they found that only water filtration was associated with any reduction in diarrhoeal mortality (of c. 11 %), and then only in the non-summer months. The lack of any effect of improvements in water supplies reported by Anderson and colleagues is consistent with the trajectory of infant diarrhoeal mortality in Britain, which showed no response to major improvements in urban water supplies in the decades after 1850. Davenport and colleagues (2019) noted that infants were relatively unaffected by cholera epidemics compared with other age groups, and speculated, as did Woods, that infants may have been given only boiled water to drink, or were otherwise protected from untreated water (Woods, 2000: 331).

If improvements in water and faecal disposal methods were insufficient to improve diarrhoeal mortality, then it is all the more puzzling that rates declined so steadily and rapidly from the second decade of the twentieth century. This decline pre-dated the development of reliable infant formula milk, as well as chlorination of British water supplies. Nor is there any evidence for increases in breastfeeding in this period. Some MOHs writing in the early twentieth century proposed an important role for flies in faecal-oral disease transmission, most notably Charles Niven, the MOH for Manchester (Niven, 1910; 1923). Niven (1910) demonstrated a strong correlation between weekly numbers of flies and diarrhoeal cases in Manchester. Writing at the tail-end of the decline of infant diarrhoeal mortality, Graham-Smith (1929) proposed that the decline was driven by reductions in urban fly populations as a consequence of the motorisation of transport. Because horse manure constituted an important breeding ground for flies, he argued that the substantial and progressive reductions in urban horse populations after 1906, as cars, trams and buses replaced horses, had produced a parallel reduction in fly populations and in the transmission of fly-borne diseases (see also Morgan, 2002). Alternative explanations include improvements in infant feeding practices and domestic hygiene. While there were no obvious breakthroughs in infant feeding formulas, it is possible, as Fildes argued, that improvements in the design of feeding bottles made artificial feeding substantially safer (Fildes, 1998). A third alternative is that infant health improved more generally, and that this was associated with greater resistance to diarrhoeal infections. This is plausible given that infant mortality fell fairly steadily from around 1901. However most of the improvement in survival was confined to older infants, and neonatal mortality rates did not fall until the 1930s. That is, there is no evidence for improvements in foetal and neonatal health before the 1930s, and this implies that any improvements in infant health and resistance to diarrhoeal diseases were probably related to improvements in nutrition or to declines in co-morbidities at older ages. Finally, there may have been autonomous changes in the pathogens involved in infant diarrhoeal infections. Key to disentangling these potential factors is the apparent simultaneity of improvements in diarrhoeal mortality not only across British towns, but also in the United States, which implicates factors that were fairly ubiquitous and synchronised in effect.

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