The first stage of the epidemiological transition in British cities: a comparison of Manchester and London, 1750-1820

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Abstract

The most striking feature of the first stage of the mortality transition in England in the late eighteenth century was the precipitous fall in mortality levels in urban populations. This period marked a transition between an early modern pattern characterised by very high urban mortality with apparently little differentiation by social status to a nineteenth century pattern of relatively moderate urban mortality penalty and the emergence of marked socioeconomic differentials in mortality characteristic of modern mortality regimes. Landers characterised pre-transitional cities in terms of a 'high potential' mortality model that described successfully many of the features of seventeenth and eighteenth century London. Here we review the applicability of Landers' model in the light of new evidence from London and from Manchester and identify several key features, notably smallpox and infant feeding practices, that distinguished seventeenth and eighteenth English cities from their successors and which may account to some extent for the excessive nature of the urban penalty in this period.

Introduction

Pre-transitional mortality patterns in the English population

Thanks to the work of Tony Wrigley and colleagues at the Cambridge Group for the History of Population and Social Structure we now have a fairly clear picture of mortality trends in England over the course of the last half millenium. Historically favourable levels of life expectancy prevailed in sixteenth and early seventeenth centuries, punctuated by subsistence and epidemic crises and especially by soaring mortality rates during plague outbreaks. From the mid-sixteenth century the amplitude of these crises diminished, and

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plague disappeared after the 1660s. However as mortality fluctuations dampened, the average level of mortality actually rose. Life expectancy was lowest in the early eighteenth century before recovering to sixteenth century levels by the turn of the nineteenth century. Life expectancy then stagnated in the middle quarters of the nineteenth century before embarking on a secular decline that has continued almost uninterrupted to the present.[2]

Wrigley & Schofield initially interpreted the recovery of life expectancy in the eighteenth century as marking simply a return to earlier conditions, rather than marking the onset of an epidemiological transition.[3] However these modest changes in life expectancy concealed three profound changes in the structure of mortality in this period. First, the rise in mortality in the seventeenth century was associated with a reduction in the volatility of mortality that contradicted earlier models that assumed a dominant role for crises in overall mortality.[4] Secondly there was a decisive shift in the age structure of mortality. Adult survival improved from the early eighteenth century, in parallel with a worsening of mortality in children. From the mid-eighteenth century survival rates improved for older children and infants, but mortality in early childhood remained higher at the end of the century than it had been in the sixteenth century. The age structure of mortality in the English population before 1750 was such that it could not be approximated by existing model life tables, all of which derived from late nineteenth or twentieth century populations.[5]

In addition to changes in the age structure and volatility of mortality, a third major change in mortality took place in the eighteenth century that was of much greater magnitude than changes at the national level, and which could not be mistaken for a return to earlier conditions. This was a dramatic reduction in mortality in urban areas.

Urban mortality patterns

Figure 1 presents estimates of infant mortality rates (IMR) for other cities and towns in England, together with rates for London Quakers, and for a relatively remote rural parish. Three aspects are immediately apparent. First, trends in urban mortality followed the national pattern in highly exaggerated form. Both small and major urban centres in England

[2] Wrigley & Schofield, 1989; Wrigley, Davies, Oeppen & Schofield, 1997
[3] Wrigley & Schofield, 1989
[4] Flinn, 1981
[5] Wrigley, Davies, Oeppen & Schofield, 1997

seem to have experienced a rise in infant and child mortality in the late seventeenth century, with IMR in London reaching perhaps as much as 400-450/1000 in the 1740s. After 1750 the modest improvements at the national level were accompanied by spectacular declines in urban mortality, so that by the 1820s London's IMR was comparable to the national average, at around 160/1000. Although infant mortality varied widely by settlement type and location in the nineteenth century, there was nonetheless a substantial convergence in rates between the eighteenth and the nineteenth centuries. Second, as Galley and Shelton have argued, infant mortality was apparently very high in even quite small urban settlements in the seventeenth and eighteenth centuries.[6] York had a population of approximately 12,000 through the seventeenth century, and barely grew over the period, yet its infant mortality rates appear to have exceeded those of even Liverpool in the mid-nineteenth century, when the Liverpool population exceeded a quarter of a million, and was the most notorious mortality blackspot of the period.[7] Market towns of several thousand inhabitants in the eighteenth century, such as Banbury, similarly rivalled the infant mortality rates of the great industrial cities of the nineteenth century. A third point is that infant mortality seems to have changed relatively little (either absolutely or proportionately) in those settlements that remained rural. This raises the possibility that the changes in mortality rates observed in the national aggregate may reflect mainly the vicissitudes of mortality in urban and urbanising areas, with relatively little change in rural populations.[8] The rural Devon parish of Hartland was relatively remote parish and the trajectory of mortality in rural communities in closer contact with urban centres remains unclear.

The exceptional mortality levels of even relatively small urban settlements and the unusually high levels of adult to child mortality in the national population before c.1750 distinguish the the early modern epidemiological regime from the mortality patterns of the English population and urban centres revealed by civil registration in the mid-nineteenth century. Despite rapid urbanisation and population growth the English population by the Victorian period was characterised by relatively small urban-rural differences in mortality and by historically favourable mortality levels but also by socioeconomic differentials in mortality that appear to have been absent before the nineteenth century.[9] These nineteenth century patterns are more easily interpreted in terms of differences in living standards, medical and

[6] Galley & Shelton, 2001
[7] Galley, 1998
[8] Unfortunately trends in mortality for individual parishes within the twenty-six parish sample used by Wrigley and colleagues in their national analysis have been published for only a few of the parishes (Banbury, Colyton and Hartland in Figure 1).
[9] Landers, 1986; Smith & Oeppen, 2006; Kelly & O'Grada, 2014

public health interventions than early modern mortality patterns. Indeed the apparent independence of mortality from living standards narrowly construed before c.1800 has led a number of researchers to posit genuinely autonomous factors such as climate or pathogen evolution as the main drivers of mortality trends before the mid-nineteenth century.[10] However these hypotheses largely fail to account for the changes in the age structure of mortality and for the exceptional falls in urban mortality compared with national rates.

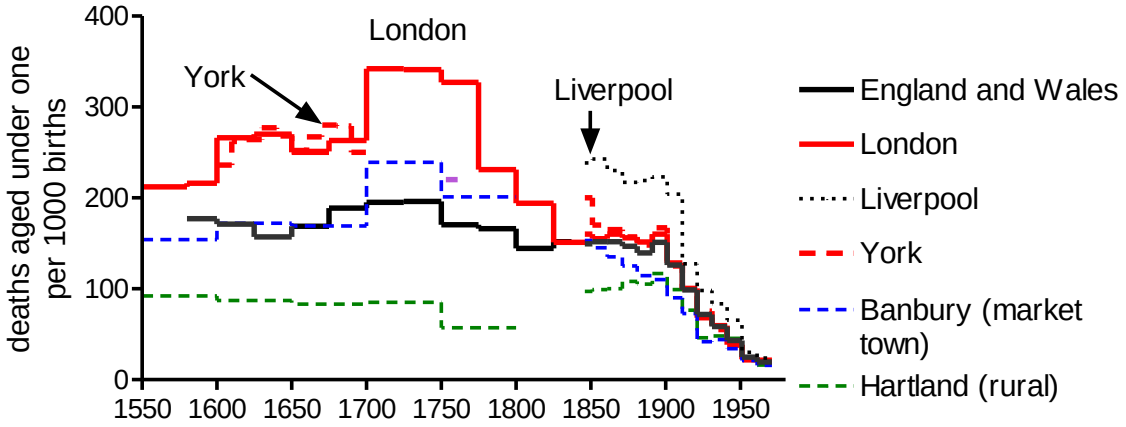

Figure 1. *Infant mortality rates by settlement type, England 1550-1970.*
Sources: England and Wales: Wrigley et al., 1997; Human Mortality Database. London: Newton, 2011, table 2; Landers, 1993, 136; Annual reports of the Registrar-General and decennial supplements. Liverpool: Annual reports of the Registrar-General and decennial supplements; York: Galley, 1998, table 4.9; Annual reports of the Registrar-General and decennial supplements. Banbury and Hartland: Wrigley & Schofield, 1983; Annual reports of the Registrar-General and decennial supplements.

The most successful analysis of early modern urban mortality patterns remains that provided by John Landers.[11] Landers proposed a structural model that described the relationship of urban mortality to enduring (structural) economic, social and political patterns. Taking infectious diseases as the dominant cause of mortality in urban populations, Landers described mortality as the outcome of the potential for exposure to infectious disease, the degree of resistance to infection, and the levels of infectious diseases circulating in the population (the pathogenic load). He considered cities before the demographic transition to be characterised by a high potential for infection as a consequence of high population densities, poor housing, sanitation and water supplies that facilitated disease transmission. Critically, large dense urban populations could maintain many diseases in endemic form

[10] Fridlizius, 1984; Perrenoud , 1997
[11] Landers, 1992,1993

(that is circulating constantly within the population without requiring re-introduction). The nodal roles of cities in trading networks, and their dependence on migration for growth also exposed urban populations to the frequent introduction of epidemic diseases.[12]

Landers also considered the potential for *resistance* to infectious diseases to be high in pre-transitional cities, at least amongst long-term residents. In the case of diseases that conferred long-lasting resistance on survivors, the high levels of exposure to infectious diseases in large urban centres meant that although mortality was severe in childhood those urban-born residents who survived to adulthood had acquired immunity to a wide range of urban pathogens. However the lower population densities of migrant hinterlands and the incomplete economic and epidemiological integration of the national population also meant that recent migrants lacked immunity to many urban diseases and were at particular risk. In addition to immunological experience nutritional status is a key determinant of resistance to many infectious diseases but Landers considered this to be less relevant in the case of pre-transitional cities given the relative insensitivity of urban mortality to variations in real wages and the lack of evidence for significant socioeconomic differentials in mortality. He predicted therefore that mortality should be high but relatively invariant, as many diseases were endemic and the high resistance of most of the population reduced the scope for epidemic outbreaks. Moreover the relative security of food provision should limit the impact of food shortages and dearth-related mortality.

Landers thus characterised his as a 'high potential' model in which both exposure and resistance displayed strong spatial structure. The extent to which this high potential for disease exposure and mortality was realised depended on a third factor, the pathogenic load. This variable was a measure of the types of pathogens to which the population is exposed, and depended on the extent to which pathogens were introduced and/or maintained in the population, as well as on the characteristics of those pathogens (for example their lethality, whether they confer long-lasting immunity or cause recurrent infections, and their modes of transmission).

Changes in mortality levels were a function according to the model of changes in the factors affecting the potentials for exposure and resistance and changes in the pathogenic load.[13]

[12] This and the following paragraph paraphrase Landers (1992: 52)

While changes in the potentials for exposure and resistance depended on structural changes in the urban and wider society the degree of pathogenic load could also vary both in response to structural changes or as a result of autonomous genetic changes in pathogens or changes in the array of pathogens liable to introduction from other populations.

Landers tested his model against London for the period c.1670 – 1830 using the Bills of Mortality, parish register material and a family reconstitution of London Quakers.[14] London was the largest city in Europe in this period and a reservoir of endemic diseases. As expected mortality was very high but relatively stable. Excess mortality was concentrated amongst children and there was evidence for extra vulnerability of adult migrants especially with respect to smallpox. Married adult Quakers on the other hand experienced mortality rates comparable with the national population. There was evidence for mortality hikes associated with periods of high food prices, but Landers speculated that these rises in mortality resulted from influxes of vulnerable adults into the population, especially demobilised soldiers in the economic aftermath of eighteenth century wars, rather than direct effects of dearth on the health of the metropolitan population. Indirect evidence against the importance of food availability or nutritional levels for mortality was the comparability of mortality, at least in childhood, between London Quakers, a relatively affluent group, and the population of the London Bills area.

Landers was more hesitant regarding the power of his model in explaining mortality change. He speculated that deterioration in the quantity and quality of housing stock increased crowding and exposure to disease in the early eighteenth century and conversely that improvements towards the end of the century reduced exposure and lightened infectious disease mortality. He also invoked other possible improvements in hygiene and living standards such as increased availability of cotton clothing, soap and piped water that could have reduced disease transmission, as well as changes in national migration patterns. He placed rather less emphasis on the two pieces of evidence that emerged most clearly from

[13] Landers described the potential for exposure and resistance in terms of four proximate variables: diet, conduction (the ease with which infections could be transmitted between individuals in the population), bounding (the ease with which pathogens could be introduced into the population from outside), and retention (the extent to which pathogens could be maintained in an endemic state in the population).
[14] Landers, 1993

his study of London Quakers, the rise and fall of smallpox mortality that affected particularly early childhood mortality, and an apparent rise in the prevalence of maternal breastfeeding.[15]

Smallpox rose as a proportion of causes in the London Bills in the second half of the seventeenth century and accounted almost entirely for the rise in childhood mortality amongst London Quakers in this period. Smallpox declined as a cause of death amongst Quaker children in the last half of the eighteenth century before the advent of vaccination c.1796 and the timing of this decline suggests that the forerunner of vaccination, inoculation may have been practiced at least amongst London Quakers in this period.

Infant mortality also fell dramatically in the period 1750-1825 both amongst London Quakers and in the Bills of Mortality population. Age-specific evidence from the London Quaker sample indicated that the fall was greatest amongst neonates, and was accompanied by a lengthening of birth intervals suggestive of either deliberate contraceptive practice or a rise in maternal breastfeeding.[16] In the English population more generally relatively long maternal breastfeeding of perhaps 12-18 months was the norm in this period and neonatal mortality varied little by season.[17] In contrast birth intervals amongst London Quakers were short in the early eighteenth century and were accompanied by a summer peak in neonatal mortality strongly suggestive of artificial feeding of newborns (high summer mortality is suggestive of high levels of diarrhoeal diseases that are most common in summer when high temperatures and the presence of insect vectors promote contamination of food and drink). Birth intervals lengthened considerably in parallel with a fall in neonatal mortality from 112 deaths in the first month of life per 1000 live births to 40/1000 between 1725-49 and 1800-24.[18] However the potential impact of any increases in maternal breastfeeding was difficult to judge. Any substitution of maternal breastfeeding for artificial feeding should have acted to reduce disproportionately the summer peak in neonatal mortality. However the summer peak in neonatal mortality persisted throughout the period 1750-1849, forcing Landers to speculate that neonatal mortality had become concentrated in a shrinking minority of families that continued to hand-feed newborns, or that a new disease appeared that affected neonates specifically and had greatest effect in summer months.

[15] Landers, 1992; Landers, 1993, chap. 9
[16] Landers, 1990:106. The term 'neonate' refers to infants in the first month of life.
[17] Wilson, 1986
[18] Landers, 1990: 106; Landers, 1993: 136.

Landers' work on London argued persuasively that structural factors within the capital and its relationship to the wider population created a situation of high but relatively stable mortality that fell particularly heavily on young children and new immigrants. Although he speculated on changes in the urban environment and living standards that reduced transmission of disease and mortality levels after 1750 his research pinpointed two main possible causes of improvements in mortality that were relatively independent of economic conditions: the medical innovation of inoculation against smallpox, and changes in breastfeeding habits. In the next section we evaluate these conclusions against new evidence for London.

New evidence for mortality patterns in London 1600-1812

Our understanding of the historical demography of urban populations remains limited by the difficulties of applying robust demographic techniques to large and highly mobile urban populations in the absence of population registers or even periodic censuses.[19] These problems are exacerbated in the period 1750-1837 by the deterioration of quality and coverage of parish registers as a consequence of lengthening birth-baptism intervals and the rise of non-conformism. These difficulties were avoided in Landers' case by the use of Quaker records that recorded birth and death rather than baptisms and burial, and which were kept with exceptional punctiliousness.[20] Nevertheless two recent studies have attempted partial family reconstitutions of several London parishes. A long-running project on the demography of early modern London included reconstitution of five very small wealthy intramural parishes in Cheapside (1600-1723) and the large and poorer suburban parish of Clerkenwell (1600-1753).[21] Davenport & Boulton also conducted a partial family reconstitution of the large Westminster parish of St. Martin in the Fields in the period 1750-1812.

In the early seventeenth century infant mortality was higher in the poorer suburban parish of Clerkenwell than the wealthy Cheapside parishes, but there was substantial convergence over the seventeenth century to the higher level of the suburban parish (with IMRs of 250 – 300/1000) (Figure 2). Analysis of birth intervals and the Marriage Duty Act returns, that recorded household members, suggested that the initially lower infant mortality of the

[19] Landers, 1993; Newton, 2013

[20] These factors did however undermine the reliability of Finlay's estimates of mortality for several London parishes before 1700, and these estimates are not discussed in detail here (Finlay, 1981).

[21] Newton, 2011; see also http://www.geog.cam.ac.uk/research/projects/earlymodernlondon/ and http://www.history.ac.uk/cmh/pip/resources.html

wealthier Cheapside parishes was probably mainly a consequence of the prevalence of the practice of extra-parochial wet-nursing of infants of wealthier families.[22] In the seventeenth century large numbers of London infants were sent out to rural parishes near London to be breastfed and reared by rural nurses often for a period of several years.[23] Infants that died at nurse were usually buried in the nurse's parish rather than the London parish of their parents. Young children, but not their older siblings, were notably absent from households in Cheapside, and birth intervals were shorter in the Cheapside parishes than in suburban Clerkenwell, suggesting that early curtailment of maternal breastfeeding increased conception rates in these parishes. Newton attributed the rise in infant mortality in the Cheapside parishes to an increasing preference for wet-nursing nearer to home that resulted in the burials of infants in their home parish and their inclusion in the reconstitution.[24] If correct this shift in the location of burial would have contributed to the apparent rise in mortality in London over the seventeenth century, independent of other changes in survival rates.

Newton's evidence covered the period of highest metropolitan mortality c.1600-1750. The period of falling mortality after 1750 is even more difficult to study, however in the case of St Martin in the Fields the exceptional richness of the surviving records compensated for many of the defects of parochial registration in this period and provided additional information on burial and baptism fees that permitted the calculation of mortality rates by social status. Although St. Martin's was a relatively wealthy parish it was also very large both physically and in population terms and was broadly representative of the metropolis as a whole with respect to male occupational structure.[25] After detection of and adjustments for missing burials, infant and child mortality rates were comparable to those of London Quakers in the mid-eighteenth century (Figure 2, Table 1).[26]

[22] In accordance with Finlay's evidence for wealthier London parishes in the seventeenth century: Finlay, 1981.
[23] Finlay, 1981; Fildes, 1986, 1988; Clark, 1988
[24] Newton, 2011
[25] Davenport,Boulton & Black, 2013
[26] Birth interval analysis (including multiple birth rates) indicated that capture of births amongst reconstitution families of fixed address was probably relatively complete but that a number of infants escaped observation at some point in the first year of life especially amongst wealthy families. It could not be determined whether these missing infants were sent to nurse or died and their corpses were exported without being recorded in the parish. The mortality rates given in Figure 2 and Tables 1,2 represent an average of rates adjusted on two assumptions: (1) that missing infants were sent to nurse outside the parish and (2) missing infants died and were exported for burial. See Davenport, Boulton & Black (2013) & Davenport, Boulton & Schwarz (2014) for details of the methodology.

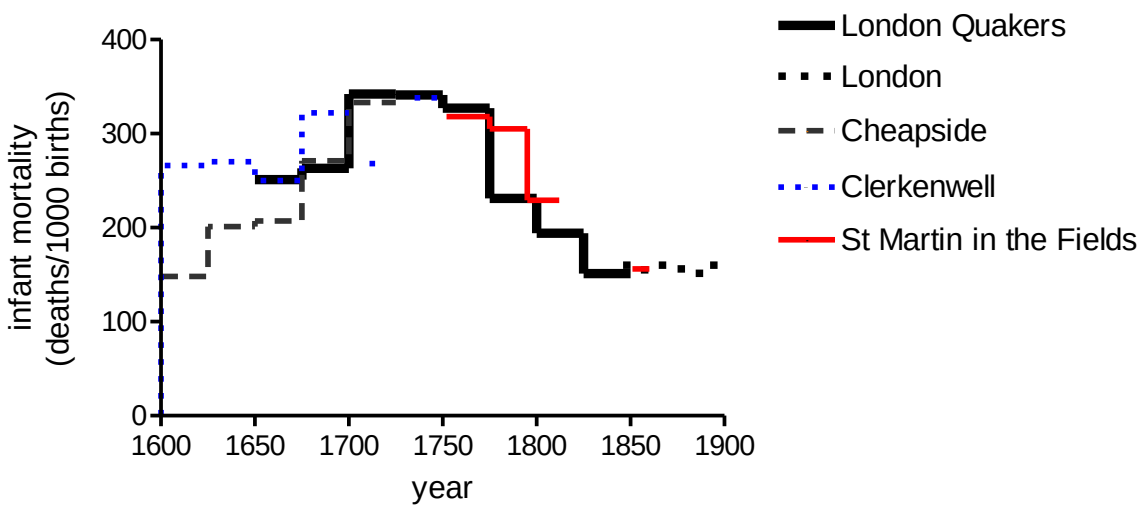

Figure 2. *Infant mortality in London*
Sources: Newton, 2011, table 2; Landers, 1993, 136; Annual reports of the Registrar-General and decennial supplements; family reconstitution of St. Martin in the Fields (unpubl.).

Neonatal mortality was very high in St. Martin's and was associated with a pronounced summer peak in mortality very similar to that observed by Landers amongst London Quakers (Table 1, Figure 3). Improvements in infant mortality were confined to neonates before c.1800 and were associated with a lengthening of birth intervals suggestive of increases in the incidence and duration of maternal breastfeeding (Figure 4). Strikingly however the summer peak in neonatal mortality persisted as the level of mortality fell. These patterns are remarkably similar to those for London Quakers. However the ability to analyse mortality by social status in St. Martin's made it possible to delve slightly deeper into the causes of these patterns.

Table 1. *Probability of dying in age interval *1000, St. Martin in the Fields reconstitution families and London Quakers*.

	St. Martin in the Fields		
Age (months)	***1752-74***	***1775-99***	***1800-12***
0	102	65	61
1-11	237	276	224
12-23	159	149	115
	London Quakers		
	1750-74	***1775-99***	***1800-24***
0	96	81	40
1-11	255	163	160
12-23	150	101	93

Notes: see fn. 26
Sources: Family reconstitution of St. Martin in the Fields (unpubl.); Landers, 1997.

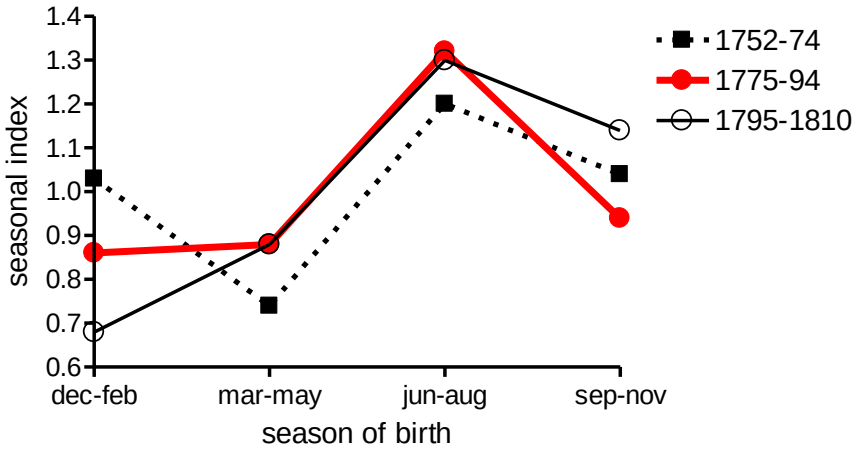

Figure 3. *Seasonality of neonatal mortality in St. Martin in the Fields (unity corresponds to the rate over twelve months).*
Source: family reconstitution of St. Martin in the Fields 1752-1812 (unpubl.)

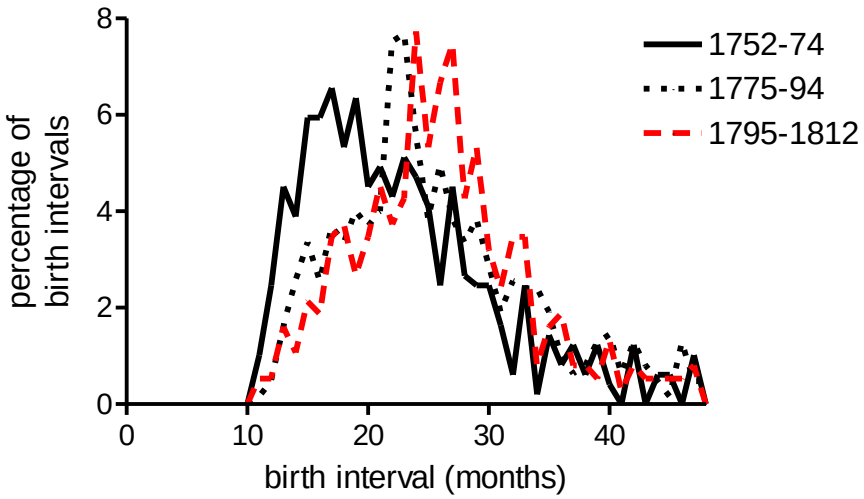

Figure 4. *Distribution of birth intervals in reconstitution families in St. Martin in the Fields where the infant whose birth opened the interval was known to have survived one year or more.*

Source: family reconstitution of St. Martin in the Fields 1752-1812 (unpubl.)

Mortality rates in the first year life showed no obvious social gradient at least before 1800 (Table 2). Infants of lower status but non-pauper families fared better than infants from either pauper or wealthy families. Wealthier families (social status groups 2 and 3 in Table 2) had shorter birth intervals in the mid-eighteenth century and this was associated with a pronounced summer peak of neonatal mortality in this group. Amongst the bottom half of the wealth distribution average birth intervals were longer (although still short by national standards) and there was no summer peak in neonatal mortality in the period 1752-74. Neonatal mortality improved in all status groups and was associated with a lengthening of birth intervals in all groups. However the summer peak in neonatal mortality persisted amongst wealthy families and remained absent amongst the non-pauper poor (although a summer peak emerged amongst paupers in the last quarter of the eighteenth century, perhaps as a function of early weaning of pauper infants to accommodate an apparently growing local market for wet-nurses).[27] The absence of a summer peak amongst the group with longest birth intervals and highest infant survival suggests that the summer peak was associated with hand-feeding of infants rather than the emergence of some new summer disease. Fildes has argued that a rising aversion to wet-nursing in the second half of the eighteenth century was associated with parallel rises in both maternal breastfeeding and

[27] Fildes, 1986

substitution of the more lethal practice of hand-feeding in preference to wet-nursing.[28] The evidence from London Quakers and St Martin's of a lengthening of birth intervals and the persistence of a summer peak of neonatal mortality is consistent with a rise in maternal breastfeeding together with a rise in hand-feeding of those infants not fed, or fed for very short periods only, by their mother. A rise in maternal breastfeeding incidence and/or duration would have reduced the exposure of newborns to infectious diseases (both gastrointestinal and respiratory) and improved their resistance to diseases later in infancy and childhood (through improved nutrition and reduced morbidity during early development). A lengthening of birth intervals should also have contributed to improvements in maternal health, with advantages for both mothers and infants.

Table 2. *Infant mortality by social status of family in St. Martin in the Fields.*

		Social status group				
period	***age (months)***	***0 (paupers)***	***1***	***2***	***3 (wealthiest)***	***all***
1752-74	*0 (neonates)*	125	87	107	96	102
	1-11	207	212	261	213	237
	0-11 (IMR)	306	281	339	288	314
1775-94	*0 (neonates)*	81	54	85	82	70
	1-11	315	211	294	260	254
	0-11 (IMR)	369	259	352	319	305

Notes: Social status was assigned on the basis of fees paid for baptism, a measure that corresponded well with both rates levied on fathers and with fees paid for burial (see Davenport, Boulton & Black, 2013). Paupers were families designated as poor in the baptismal fee books and comprised c.10% of reconstitution families, status group 1 paid the standard fee for public baptism (1s 6d) and comprised 25-40% of families depending on period, status group 2 paid the standard private baptism fee (4s or later 5s) and comprised 25-40% of families depending on period, and status group 3 paid more than standard fees and comprised c.10% of families. Baptismal fees were highly graduated only before 1795 and social status could not be assigned beyond this date. Rates were adjusted as described in fn. 26.

Amongst older infants and one year olds mortality fell in St. Martin's only after 1800, in contrast to London Quakers where mortality fell after 1750 in these age groups (Table 1). Amongst Quakers this fall was driven, at least for children aged one year and over, largely by a specific fall in smallpox mortality. This fall pre-dated the advent of vaccination in the

[28] Fildes, 1986, 1988

very last years of the eighteenth century, and may reflect the precocious use of inoculation amongst London Quakers. In St. Martin's gains in child survival were similarly associated with a specific fall in smallpox mortality, but this fall occurred only after the introduction of vaccination.[29]

Evidence of the age structure of smallpox burials from St. Martin in the Fields, St. Dunstan Stepney and St. Mary Whitechapel also indicated a substantial vulnerability amongst adult migrants to London.[30] In St. Martin's a male and non-pauper bias amongst adult smallpox victims in the mid-eighteenth century suggested that these were mainly migrants from relatively remote rural areas and were atypical of the majority of London's immigrants.[31] The proportion of smallpox burials that was adult fell dramatically in the 1770s in all three parishes, suggesting some sudden change in exposure to smallpox, natural or artificially acquired, in the English population. A reduction in adult vulnerability to smallpox would have reduced adult mortality in London but could also have contributed to reductions in infant mortality via improvements in maternal health if a significant proportion of migrant mothers experienced smallpox infection in pregnancy (although the latter is an unlikely scenario).[32]

One challenge to Landers' model has come from Kelly and O'Grada, who analysed disaggregated burial series from the 404 parish series originally used by Wrigley and Schofield. The authors argued that although the positive check, in the form of mortality hikes in response to rises in food prices, had virtually disappeared amongst most of the English population by 1650 it persisted in London until c.1750.[33] Kelly and O'Grada argued that the volatility of London burials in response to food prices reflected the inadequacy of welfare provision in the capital compared with rural parishes and smaller towns. This issue requires further investigation. Landers argued that rises in mortality in response to food prices in London were a product of in-migration in these periods of adults with low immunity to urban diseases especially smallpox. However he was puzzled by the age patterns of mortality during these crises because the greatest rises in mortality were concentrated amongst children rather than young single adults who are usually considered to have constituted the majority of rural-urban migrants in the English population.

[29] Davenport, Boulton & Schwarz, 2011; 2014.
[30] Davenport, Boulton & Schwarz, 2011; Razzell, 2011.
[31] Davenport, Boulton & Schwarz, 2011
[32] Woods, 2009; Davenport, Boulton & Schwarz, 2011
[33] Kelly and O'Grada, 2014.

Taken together these recent studies of London reinforce Landers' original findings regarding the high mortality potential of London. The similarity of trends amongst London Quakers, the London Bills population, social status groups in St. Martin in the Fields and wealthy and poor parishes in the seventeenth century indicate that the disease environment of London was sufficient to overwhelm the advantages of wealth, at least in childhood. Critically, these studies also confirmed Landers' findings of the importance of infant feeding practices in heightening exposure potential, and of the contribution of smallpox to the pathogenic load in the metropolis. Patterns of smallpox mortality amongst adult migrants also confirmed the key role of spatial structure in sustaining differences in exposure to disease as predicted by Landers' model.

Mortality drivers and trends in non-metropolitan urban centres.

While this brief survey of recent work on London is consistent with Landers' model the wider applicability of the model remains largely untested. In the seventeenth and early eighteenth centuries burial surpluses and high infant and child mortality were also typical of relatively small urban settlements such as Banbury and York where the scope for endemicisation of infectious diseases was more limited. However the trajectory of mortality improvements in urban centres outside London after 1750 remains highly speculative. The main source underpinning the assumption of widespread improvements is baptism:burial ratios. Where the evidence survives these ratios were generally below unity (that is burials exceeded baptisms) for urban centres in England in the 'urban graveyard' period c,1650-1750, but in most cases showed a progressive improvement after 1750 with baptisms generally exceeding burials by the last quarter of the eighteenth century. Although subject to major problems regarding various sources of under-recording these crude indicators of mortality trends are borne out by the relatively modest levels of mortality evident in urban settlements by the mid-nineteenth century that suggest significant improvements (Figure 1). Nevertheless it remains unclear whether mortality decline was ubiquitous and whether the drivers of this decline were the same across the urban heirarchy. The dramatic decline in metropolitan mortality coincided with a remarkable restructuring of the urban hierarchy in England, as older provincial towns were eclipsed by the rapid growth of northern industrial and manufacturing towns, port cities and to some extent resort towns. London also grew rapidly in this period and its share of the national population rose from 10 to 14% between 1750 – 1850. However although London remained by far the largest city in England (and

Western Europe) its mortality levels were anomalously modest throughout the nineteenth century.

In an attempt to compare the mortality experiences of different settlement types Wrigley et al. compared mortality in reconstitution parishes grouped into four types (agricultural, industrial, market towns and mixed) on the basis of their characteristics in 1831. The industrial parishes were probably largely agricultural in the seventeenth century, acquiring an urban and industrial complexion only in the course of the eighteenth century.[34] To compare change over time Wrigley et al. compared mortality in each reconstitution settlement with mortality rates in the registration district that included the reconstitution settlement in 1838-44. This comparison was problematic because registration districts were deliberately contrived to include both rural and urban parishes where possible and therefore any comparison would dilute the mortality of urban centres in the later period and inflate the mortality of rural components. Nevertheless Wrigley et al. found that mortality levels improved for market towns, changed little for agricultural and 'mixed' settlements and worsened only in the case of those parishes that developed an industrial function over the course of the eighteenth and early nineteenth centuries. While the transition from a small agricultural to a large industrial population would perhaps be expected to result in a rise in mortality regardless of falls in urban mortality, these findings serve as a reminder that mortality trends may have been less obviously benign in the case of the rapidly growing northern towns whose growth drove most of the urbanisation that occurred during the Industrial Revolution.

Indeed the malign contribution of northern industrial cities to the stagnation of national life expectancy over the period 1820-1870 forms part of one of the most long-running debates in English economic history, regarding the impact of early industrialisation on living standards. The pessimistic view argues that industrial cities experienced a worsening of mortality especially in the second quarter of the nineteenth century, and that this was due to the peculiar conditions of industrialisation, including administrative breakdown and rising social inequalities.[35] An alternative view is that any mortality rise that did occur was largely a function of population density and exogenous epidemiological change.[36] The debate has centred on life expectancy and mortality rates during the period 1820-1850, when the paucity

[34] Wrigley et al. 1997: 268-277
[35] Szreter, 1997; Szreter & Mooney, 1998
[36] Woods, 2000: chap.9.

of data is most extreme. Less attention has been paid to developments during the early stages of the growth of industrial and manufacturing towns, 1750-1820, although most of the urban disamenities of the later period were manifest well before 1820, including rapid population growth in conditions of inadequate housing and infrastructure, overcrowding and the growth of factory labour. Here we compare developments in Manchester with Landers' model and evidence for London in the period 1750-1820, when mortality improvements were most remarkable in the capital.

Manchester

In 1750 Manchester was a town of around 17,000 people; by 1850 it had grown to become Britain's third largest city, with a population of c. 250,000, its growth predicated on its role as the commercial and manufacturing centre of the British cotton industry (Figure 5). Manchester exemplified in extreme form the new type of city that developed during the Industrial Revolution, lacking the administrative infrastructure of older towns (Manchester was governed by a manorial court leet until 1838), and characterised by rapid growth, very high population densities, and an unusually pronounced segregation of housing by social class.[37] By contrast, London was a mature metropolis of perhaps 675,000 by 1750, with a complex system of parochial and urban institutions. While its population continued to expand rapidly after 1750 this was probably not accompanied by a net rise in population density, and average population density was well below that of Manchester or Liverpool in the mid-nineteenth century. Moreover London had long surpassed the theoretical population thresholds at which the major infectious diseases of the period could establish themselves in endemic form (for example, the so-called 'Bartlett threshold' of c.250,000 in the case of measles in twentieth century populations).[38] By contrast Manchester's population density was exceptionally high even in the mid-eighteenth century (Figure 6), and the population probably crossed various epidemiological thresholds in the process of expansion.

Manchester presents substantial difficulties for historical demographic analysis. On the plus size the town of Manchester was contained wholly within the very large parish of Manchester and at least before c.1800 most baptisms, marriages and burials of town residents were registered at Anglican churches within the town itself. In addition Manchester published bills of mortality and although these have in most cases not survived the sextons' burial books of the parish (collegiate) church recorded age and cause of death for burials for much of the

[37] Pooley & Pooley, 1984
[38] Bartlett, 1960

period 1753-1820. The single greatest difficulty in the case of Manchester is the decline of Anglican registration of events especially after 1800. Non-conformist sects proliferated in the early decades of the nineteenth century and by 1821 c.70 percent of burials were estimated to be non-Anglican. Irish immigration also created a substantial Catholic population, and a Catholic chapel (and baptism register) was established in 1772 although a specifically Catholic burial ground only opened in 1816.[39] To complicate matters non-Anglicans often used Anglican burial sites due to the paucity of non-Anglican facilities (at least before the opening of the so-called 'Dissenters' cemetery on Rusholme Road in 1821). The same probably applied to baptisms in the absence of adequate chapels and churches for non-Anglicans and it is impossible to tell what proportion of baptisms and burials recorded in Anglican churches were attributable to non-Anglicans.

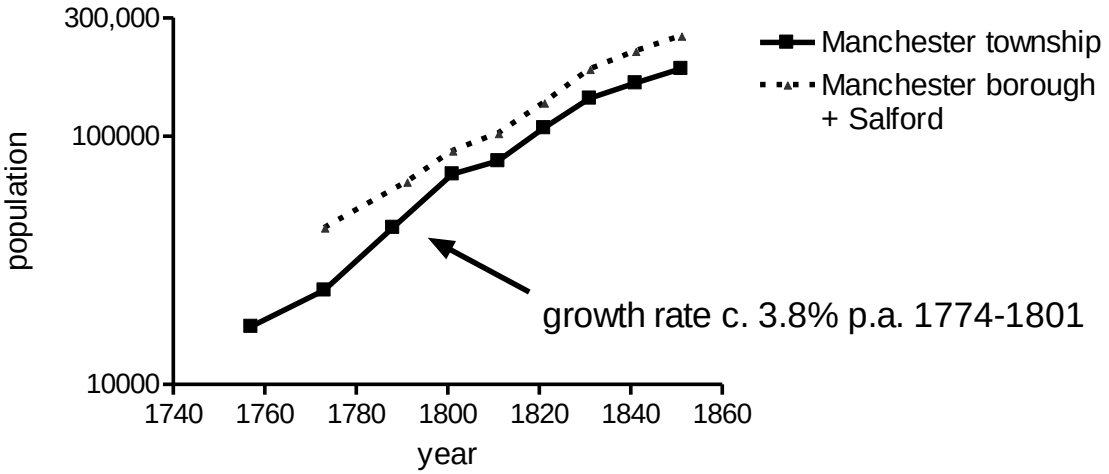

Figure 5. *Population of Manchester township and Manchester borough with Salford town. Note the log y-axis.*

Sources: Manchester township: Chaloner, 1959, 41; *Census of the town and parish of Manchester 1773-74. Enumeration of the houses and inhabitants made by Thomas Percival and John Whittaker*, Chetham Library Mun.A.4.54-56); *Census Enumeration Abstracts 1801, 1811, 1821, 1831, 1841.* Manchester borough with Salford population from Wrigley, 2011.

[39] Edge, n.d.

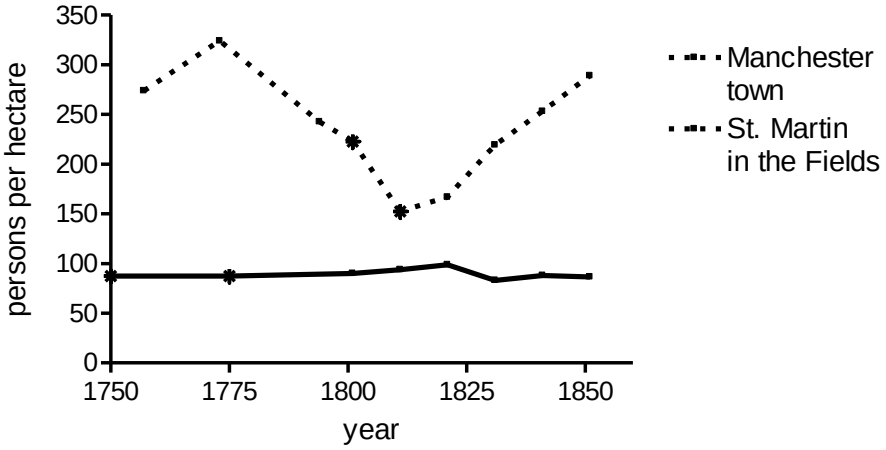

Figure 6. *Population densities, St. Martin in the Fields and Manchester town*
Sources: Manchester population as for Figure 5, Manchester urban area estimated from historical maps by Dr Max Satchell to 1794 and area of Manchester town*ship* from 1871 census; St. Martin in the Fields population *Census Enumeration Abstracts 1801, 1811, 1821, 1831, 1841;* area of parish from 1871 census. Asterixes denote figures that are based on estimates of population for St. Martin's, and of area for Manchester.

Figure 7 shows annual baptisms and burials recorded in the Manchester Bills of Mortality, together with annual series for London. In both cases there was a slight baptismal excess in non-plague years before c.1650. The disappearance of mortality crises associated with plague was accompanied by the development of a persistent burial surplus in the period 1650-1750, and the resumption of a baptismal excess in the late eighteenth century. However in the case of Manchester the excessive rise in baptisms relative to burials after c.1770 is a spurious consequence of the inability of the collegiate church to provide sufficient burial facilities for the town's expanding population. However the collegiate church claimed a longstanding monopoly on fees for baptismal and marriage registration and so a high proportion of baptisms continued to take place at the church to avoid the incursion of double fees for baptising elsewhere.

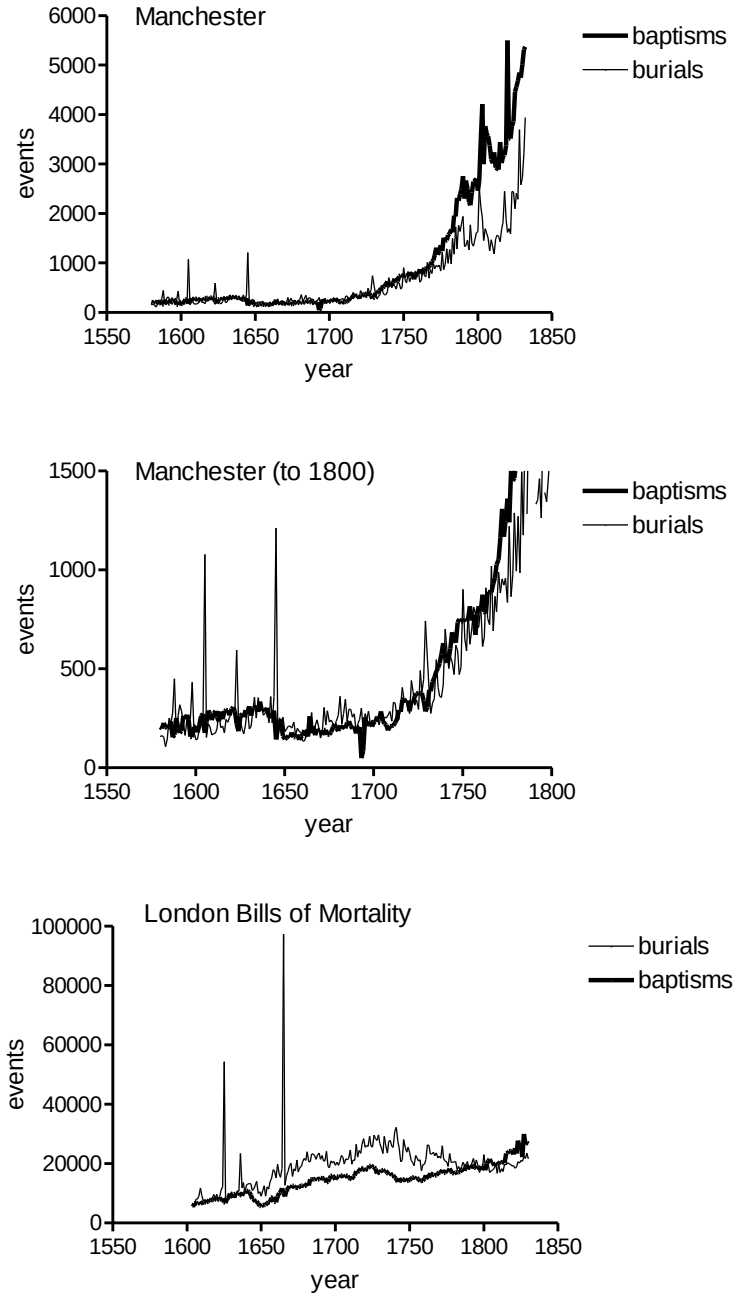

Figure 7. *Annual baptisms and burials recorded in the Bills of Mortality for Manchester and London.*
Sources: Baines and Harland 1864; Marshall, 1832.

In the face of the complexity of registration practices in Manchester we adopted two approaches. The first was to use evidence of the age structure and disease spectrum provided by the cause of death and age information contained in the sextons' books of the collegiate church (and from 1769-1812 the very complete records of the church of St.

Deansgate Manchester) to estimate changes in the mortality patterns of particular diseases and the contribution of different diseases to mortality at each age.[40] The collegiate church records comprised almost all burials in the town in 1750 and the two churches together recorded more than 75% of burials in the town in 1800. This method therefore assumed that trends in burials in these two churches reflected processes occurring throughout the population of Manchester town.[41]

The second approach was to estimate mortality *rates.* For this we needed to determine the number of events occurring within the population of Manchester town, and to establish the population at risk for the town. This was simplest in the case of infant mortality and here we limit our analysis to this rate. To estimate infant mortality we required annual counts of baptisms (the nominal population at risk) and of burials of infants. Since age was not recorded in most sources of burial information before 1813 we used the proportion of infant burials in our two main sources (the collegiate church and the St. John Deansgate records) to estimate the proportion of burials that were infant in sources lacking age information. As with our first approach this method assumed that burials at the collegiate and St. John Deansgate churches were representative of the whole population of Manchester town. To determine the numbers of events we have counted *all* extant burial and baptism records from Anglican, non-conformist and catholic chapels and churches in the parish of Manchester and the town of Salford by month and year. These records usually gave the abode of the deceased or of the parents of the baptised infant (in all cases from 1813 and in most cases where the abode was outside the township associated with each church before 1813). This abode information allowed us to extract burials and baptisms of residents of Manchester town from registers from outside Manchester town, and to exclude events of non-Manchester residents recorded in registers from Manchester town.

Infant mortality in Manchester 1753-1812

Baptisms and burials provide an imperfect record of births and burials, and under-registration of births and deaths is assumed to have increased over the eighteenth century as the increasing tendency to delay registration of baptism resulted in greater numbers of infants dying before baptism and their burials being excluded from burial registers. We have argued elsewhere that at least in the case of Anglicans the trend in delay of baptismal

[40] Davenport, Boulton & Schwarz, 2014

[41] This assumption was given some validity by the very similar patterns of burials by age and cause at the colleigate church and St John Deansgate (see for instance Figure 11 below).

registration was accompanied by a rise in private baptism.[42] Therefore while baptismal registration may have become increasingly inadequate the registration of infant burials was probably unaffected, since most infants were still baptised rapidly. To the extent that the shortfall in baptisms was a function of deaths before baptismal registration then this can be corrected by adding early infant burials to the baptismal totals. Nonetheless some infant deaths will always have gone unrecorded and this was particularly the case for very early neonatal deaths. A well-established test for the under-registration of neonatal deaths is the biometric method of Bourgeois-Pichat. Bourgeois-Pichat argued that mortality in early infancy was dominated by 'endogenous' causes arising from genetic factors or incidents during gestation or birth, but subsequent mortality was largely a function of infectious diseases ('exogenous' causes) that produced a linear rise in cumulative mortality when plotted on a semi-log plot. In this model under-registration of early infant deaths should be evident in unrealistically low mortality in the first month of life, and/or non-linearity of subsequent mortality patterns. Figure 8 shows biometric plots of infant mortality estimates for Manchester derived from the age patterns of infant burials of Manchester residents at the collegiate and St John Deansgate churches, inflated for burials of Manchester residents at other churches and expressed per thousand baptisms of Manchester residents. Also included are data for 1839 reported by the Registrar-General.

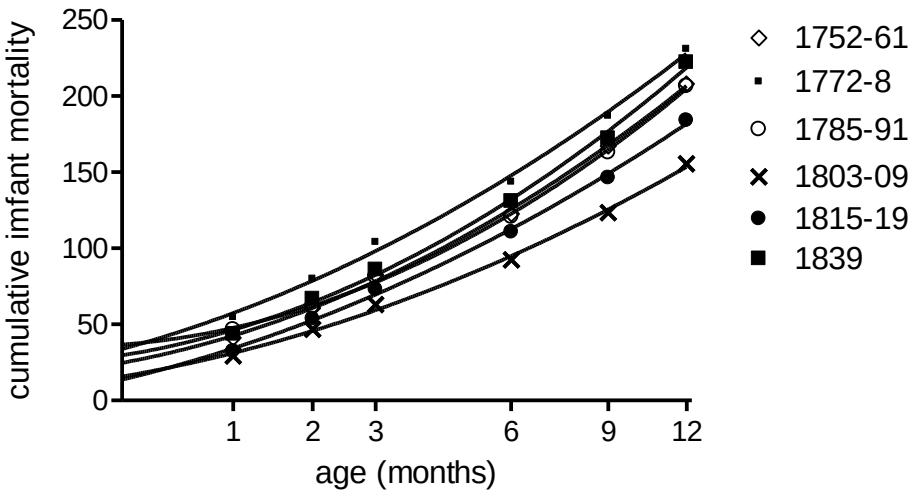

Figure 8. *Cumulative mortality over the first year of life in Manchester town, fitted with second-order polynomials (only years where >95% of burial entries included age information were included)*

[42] Boulton & Davenport, 2014

In all cases the plots of cumulative mortality over the first year of life showed a pronounced curvature and were best fitted by a second-order polynomial rather than a linear equation. While such curvature can be interpreted as indicative of under-registration of mortality in early infancy it is also typical of populations with high infectious disease mortality particularly in cases where early weaning resulted in higher susceptibility to disease later in infancy.[43] In the case of Manchester it appeared that smallpox was the main cause of this upward curvature at least before 1810, and fits were linear when smallpox burials were subtracted (Figure 9). The upward curvature had diminished by the second decade of the nineteenth century together with declining smallpox mortality, but appears to have been re-established by 1838. The estimates of endogenous mortality derived from polynomial fits were at the low end of the range of reliable estimates for the English population in this period (26 – 36/1000) except for the first two decades of the nineteenth century where there was some evidence for under-registration of early infant deaths.[44] This does not imply that registration of early infant burials was substantially complete in Manchester before 1800, but indicates at least that under-registration was not extreme.

If we take these measures of infant mortality in Manchester at face value then infant mortality was apparently relatively modest in Manchester compared with London in the mid-eighteenth century (comparable to that in the market town of Banbury at around 210/1000, rising to 230/1000 in the 1770s) (Figure 10). Infant mortality did not improve notably in the last half of the eighteenth century in Manchester and was apparently at a similar level in 1838-44 as it was in the 1770s. However the two periods for which we have estimates for the early nineteenth century, 1803-09 and 1815-19, were associated with lower infant mortality rates (156/1000 and 184/1000 respectively). These estimates of infant mortality in Manchester require further validation, but raise the interesting possibility that infant mortality was comparatively modest in Manchester in the eighteenth century and even fell in the early nineteenth century despite the explosive growth of the city in this period, before regaining its eighteenth century level sometime after 1820. Below we investigate this scenario further using evidence from the age structure and causes of burials in Manchester and seasonality of infant burials. We focus on the factors identified as key in London: smallpox, infant feeding practices, and the geography of migration, as well as population size and density.

[43] Knodel & Kintner, 1977
[44] Wrigley et al. 1997: 232

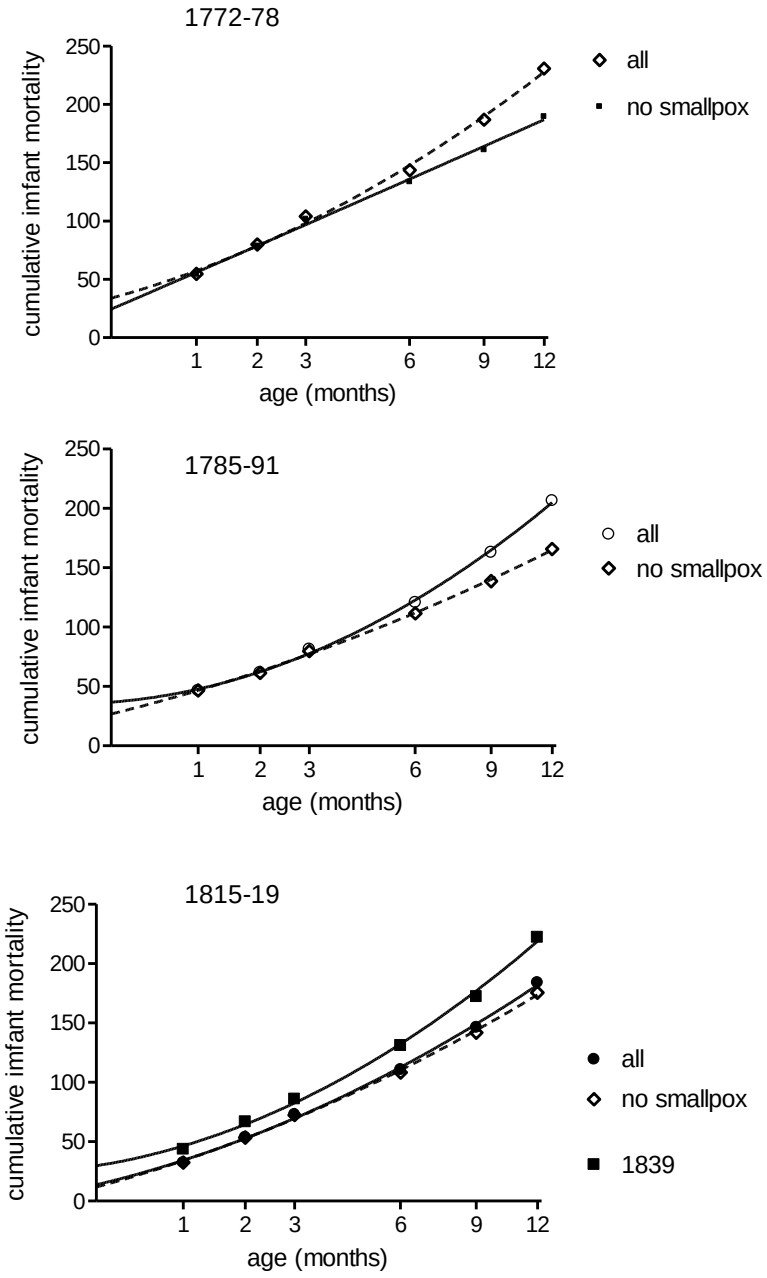

Figure 9. *Cumulative mortality over the first year of life in Manchester town, all burials included or only non-smallpox burials (only years where >95% of burial entries included age and cause of death information were included). All curves fitted with second-order polynomial equations.*

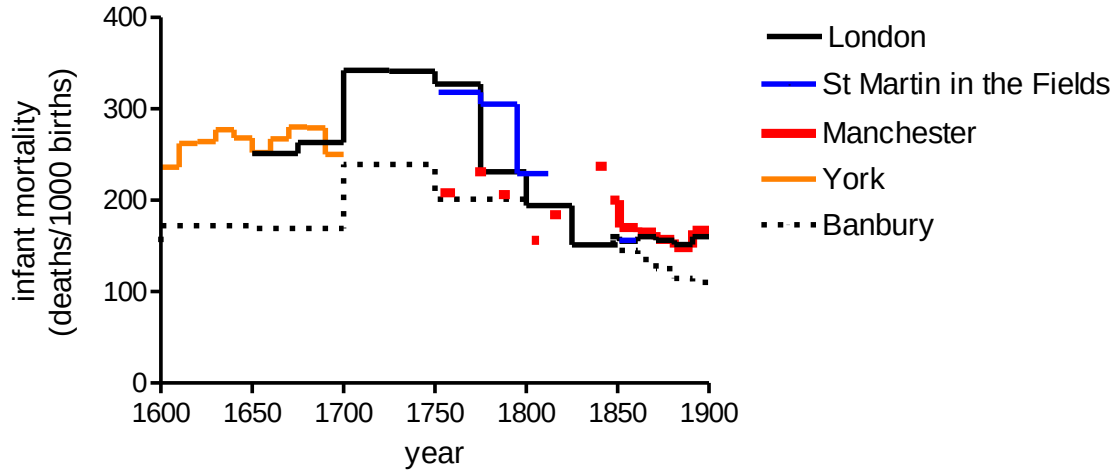

Figure 10. *Infant mortality rates for urban populations.*

Sources: as for Figure 1; unpubl. estimates for Manchester.

Population size

The smaller size of Manchester appears to have played some role in reducing exposure to infectious diseases compared with London. The potential impact of population size is best illustrated by the case of measles. Measles was endemic in London and a biennial epidemic cycle was superimposed in a weekly toll of measles burials. By contrast measles was a minor cause of death in Manchester before c.1810 and appeared at roughly four yearly intervals in epidemic form. By the late 1830s however measles had become endemic in Manchester, reflecting the growth of its population by this date and its attainment of a critical threshold for measles transmission attained by London two centuries earlier.

Smallpox and spatial inequalities in potentials for exposure and resistance and pathogenic load

Smallpox was a much more significant cause of death in eighteenth century Manchester than it was in London, accounting for up to 40% of burials in some years (Figure 11).[45] This predominance of smallpox was not simply a function of the relatively low levels of mortality from other causes. Instead there is reason to think that smallpox was more lethal in Manchester than in London. Smallpox was a major cause of death in infancy, accounting for 30% of infant burials compared with less than 20% in St. Martin in the Fields, and crude

[45] Davenport, Boulton & Schwarz, 2014.

rates of smallpox mortality in infancy were higher in Manchester than St. Martin's.[46] Moreover the average age of child smallpox burials was lower in Manchester than in London.[47] The average at burial is an indicator of the probability of infection with (or frequency of circulation of) an infectious disease, and suggests that smallpox circulated at least as rapidly within Manchester as within the metropolis despite the much greater size of London's population. Smallpox was clearly endemic in both cities, with smallpox burials recorded in most weeks. Superimposed on this background were epidemic cycles of around two years in both cities in the late eighteenth century.

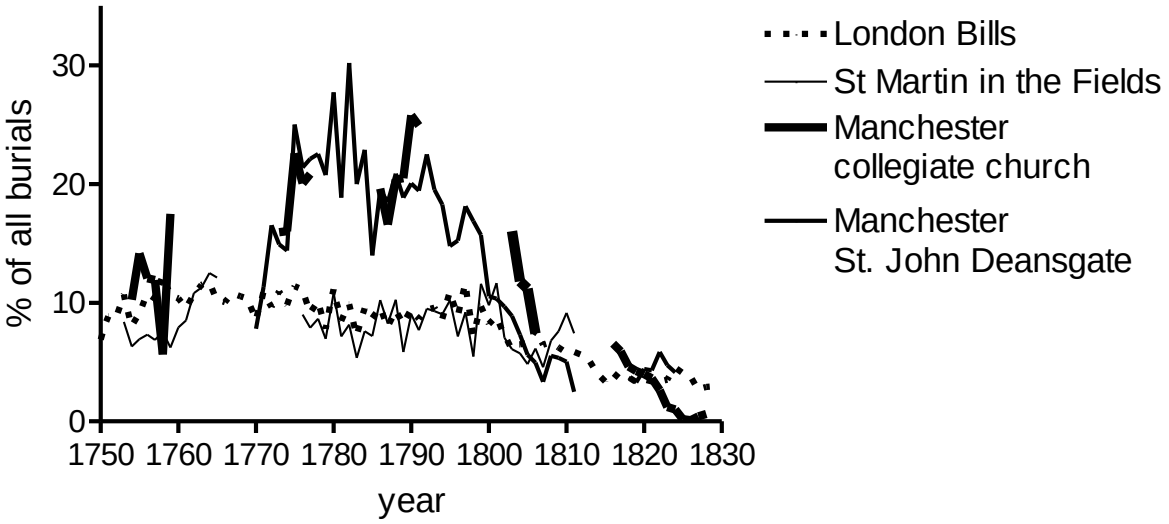

Figure 11. *Percentage of all burials described as smallpox, three year moving means. Sources:* Marshall, 1832, unpag. tabs.

The apparently greater intensity of smallpox circulation in Manchester fits the paradoxical evidence of a north-south divide in smallpox patterns uncovered by Razzell. In London the age distribution of smallpox burials indicates that adult migrants remained at risk of smallpox infection until at least the last quarter of the eighteenth century. Creighton argued that this was a situation peculiar to London which received "a constant recruit direct from the country... from parishes where as Lettsom says, "the smallpox seldom appears"".[48] However Razzell has produced credible evidence of substantial vulnerability of immigrants in other southern towns in the late eighteenth century, for example Southampton.[49] By contrast scattered evidence from northern communities indicates that at least by the eighteenth

[46] Davenport, Boulton & Schwarz, 2014
[47] Davenport, Boulton & Schwarz, 2014
[48] Creighton, 1894: 533
[49] Razzell, 2011

century almost no adults died of smallpox (Figure 12). This peculiar geography of smallpox vulnerability is borne out by comparison of Manchester and St Martin in the Fields. Whereas 20% of smallpox victims were aged 10 years or more in mid-eighteenth century Westminster, the corresponding figure for Manchester was less than five percent. This difference was not a function of differences in the age structure of the two populations but probably reflects differences in the migration fields of the two cities.[50] Manchester not only drew migrants from a smaller area than London, but as Figure 12 suggests most of these migrants had probably been exposed to smallpox in childhood. Of the few adult victims of smallpox recorded in the collegiate church burial books 1753-61, 12 of the 32 were soldiers, who were usually drawn from a much wider migration field than other types of migrant.

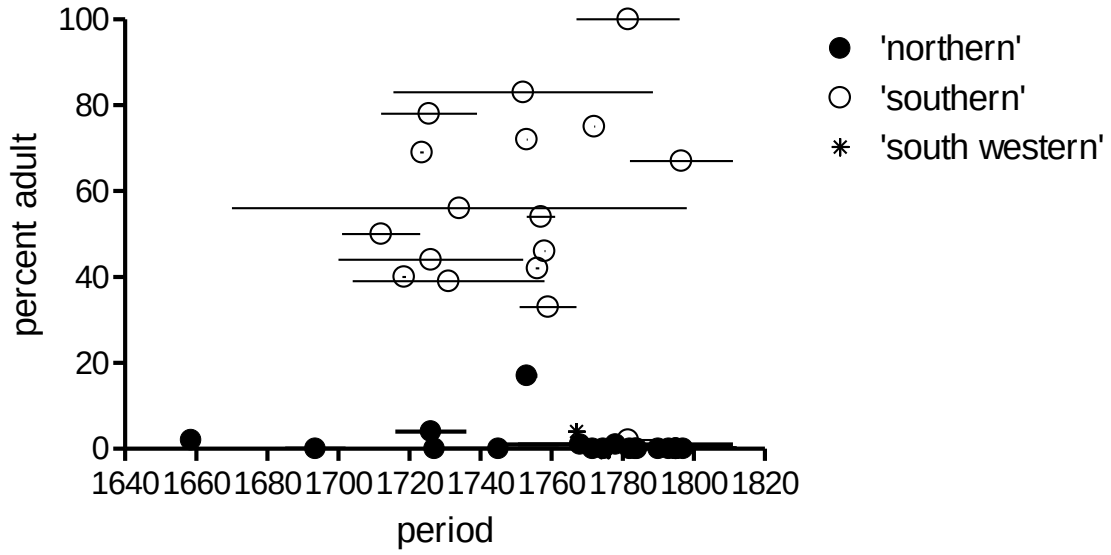

Figure 12. *Percent of smallpox burials aged 10+ or adult by period and region of Britain.*
Notes: Where burial registers did not give age child status was assigned to burial records described as 'infant', 'son of', 'daughter of'. In rural parishes these descriptors were applied to teenagers resident with a parent as well as younger children.
Source: Razzell, 2003: xi-xiii.

The apparent ability of smallpox to endemicise within relatively small urban populations may help to explain the peculiar lethality of even market towns in the century and a half before c.1800. For other diseases where the frequency of disease exposure was a function of

[50] Davenport, Boulton & Schwarz, 2014

population size, as seems to have been the case with measles, then mortality from these types of diseases would be highest in the largest cities. However for diseases that did not confer immunity, or that could be sustained for long periods outside a human host (as was the case with smallpox) then population size probably played a smaller role in influencing mortality levels. It is tempting to speculate that the higher population densities of Manchester compared with London also played a role in facilitating smallpox transmission and/or raising case-fatality rates (Figure 6).[51] In this sense then the particular nature of the pathogenic load in early modern populations may have been crucial in determining some of the key features of urban mortality patterns not readily captured otherwise by Landers' structural model.

The importance of smallpox even in infancy makes it likely that vaccination had a significant impact on mortality in Manchester as suggested by the modest infant mortality rates estimated for the period 1803-09 and 1815-19 as smallpox diminished dramatically as a cause of death (Figures 10,11). It also appears likely however that mortality rose again in the middle decades of the nineteenth century and it remains to be determined whether this rise reflected the endemicisation of diseases such as measles and scarlet fever as Manchester's population grew large enough to sustain transmission, or was more a function of growing urban disamenities and changes in the composition of the population.

The superior immunological experience of Manchester's migrants would have helped to moderate mortality in the town in the eighteenth century, although it is possible that this difference between Manchester and London was limited to smallpox. There is certainly anectdotal evidence for the greater vulnerability of recent immigrants to various types of fever and typhoid in Manchester.[52]

Infant feeding practices

In London there was good evidence for relatively low rates of maternal breastfeeding before c.1775 and this was accompanied by short birth intervals and a strong summer peak in neonatal mortality. We lack birth interval data for Manchester, but analyses of seasonality by infant age at burial indicate that there was no summer peak evident in neonatal or older infant burials in the period 1753-78 (Figure 13). However a summer peak in burials at all

[51] The course of changes in IMR also appeared to follow changes in population density in Manchester (Figures 6 and 10).
[52] Aiken, 1795: 193

infant ages 0-5 months emerged in the period after 1780 and persisted until at least 1850. These data are not conclusive evidence of infant feeding practices but do suggest that relatively lengthy maternal breastfeeding may have been the norm in Manchester before the last quarter of the eighteenth century. It is tempting to speculate that the emergence of a summer peak in mortality in early infancy coincided with reductions in the incidence and/or duration of breastfeeding associated with the increase in opportunities for female work outside the home that accompanied the increase in manufacturing and the development of factories in Manchester in the last two decades of the eighteenth century.[53] If maternal breastfeeding was common in Manchester in the mid-eighteenth century then this is likely to have resulted in much lower levels of neonatal and post-neonatal mortality than was the case in London, and may account for the relatively modest levels of infant mortality recorded in this period. Conversely any reduction in the prevalence of maternal breastfeeding would, all else being equal, have raised infant mortality in Manchester in a period when infant mortality was falling in London in part as a consequence, we have argued, of rising rates of maternal breastfeeding.[54]

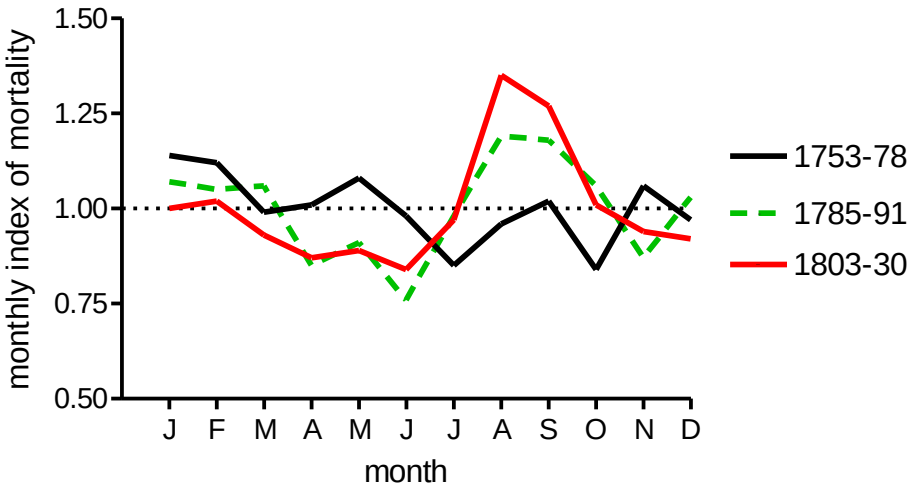

Figure 13. *Seasonality of mortality in the first six months of life, Manchester*

Sources: collegiate church sextons' books, St John Deansgate burial registers

[53] Vigier, 1970

[54] A fall in the prevalence of maternal breastfeeding would also explains Huck's evidence of a (relative) rise in the summer component of mortality amongst neonates in industrial communities in the early nineteenth century: Huck, 1994.

Conclusions

A consideration of new evidence for Manchester and London provides strong support for Landers' 'high potential' model of mortality in early modern cities. Exposure to infectious diseases appears to have been high enough to overwhelm any survival advantages of wealth at least in early childhood. Resistance appears to have varied as Landers argued according to prior immune experience, itself a function of age and of spatial differences in disease frequency that rendered young children and recent adult migrants relatively susceptible to infection.

Critically, the evidence presented here both supports Landers' argument that the early modern urban mortality regime differed profoundly from that of the nineteenth century, and helps to account for the transition between the two regimes. Three factors appear to be of key importance: smallpox, infant feeding practices, and the geography of migration and disease transmission.

Landers identified smallpox as the major reason for the rise and fall of mortality in early childhood (ages 1-4) amongst London Quakers.[55] Given the importance of smallpox in Manchester where it accounted for twenty percent of annual burials, and as a cause of death in young adult migrants to London, it seems clear that smallpox must be given some credit for the rise of mortality in urban centres in the late seventeenth century. Before c.1650 despite lethal outbreaks of plague it appears that at least some urban centres including London and Manchester were able to maintain positive baptism:burial ratios in most non-plague years. That is, in the century before c.1650 the urban graveyard effect (the baptismal deficit evident in decadal averages of baptisms and burials) was due mainly to periodic outbreaks of plague that occasioned devastating mortality surges. In intervening years towns were probably mostly capable of modest natural increases. The disappearance of plague coincided with a rise in 'background' mortality so that in the case of London and Manchester at least burials came to outnumber baptisms in almost every year despite the reduction in severe mortality events. Carmichael and Silverstein have argued that smallpox developed from a relatively benign disease into a more lethal form in the course of the seventeenth century in Europe, a claim consistent with the progressive rise in smallpox burials recorded in the London Bills.[56] As argued above, the apparent ability of smallpox to persist in

[55] Landers, 1993: 154
[56] Carmichael & Silverstein, 1987

circulation even within dispersed populations may help to account for the peculiar lethality particularly to children of even relatively small market towns before 1800. Within Landers' high potential model smallpox represented a major component of the pathogenic load that translated high potential for infection into realised mortality.

Conversely, vaccination campaigns that reduced smallpox to a minor cause of death by the mid-nineteenth century must have made a major contribution to improving life expectancy in cities and towns. While the impact of vaccination was muted at the national level this may reflect partially the rural bias of the sample used to calculate national mortality rates, as well as the apparent infrequency of smallpox epidemics in smaller settlements in southern England.[57] It is likely that the impact of vaccination on urban populations was very significant.[58] Moreover the benefit may have been greatest in small northern towns where smallpox appears to have constituted a high proportion of burials.[59] The extension of vaccination to rural populations would also have made towns substantially safer for adult migrants.

Infant feeding practices also seem to have been key in creating a very high potential for infection amongst the most vulnerable age groups in London. By contrast Manchester in the mid-eighteenth century apparently enjoyed fairly modest infant mortality on a par with Banbury, a town a tenth its size, and displayed no evidence of early weaning. However the evidence of infant feeding practices is indirect. In London (and seventeenth century York) birth intervals were very short and neonatal mortality displayed a marked summer peak characteristic of weanling diarrhoea and absent from the national population where relatively long breastfeeding was the norm. A substantial decline in neonatal mortality in London in the second half of the eighteenth century coincided with a lengthening of birth intervals consistent with longer maternal breastfeeding and with documentary evidence of both profound shifts in maternal preferences for breastfeeding and the virtual disappearance of evidence for rural wet-nursing of London infants (except parish and Foundling hospital

[57] It is notable that although infant mortality rates calculated from family reconstitution dove-tailed well with the national series derived from civil registration from 1838, early childhood mortality rates (ages 1-4 years) were substantially lower in the reconstitution sample than the national series derived from civil registration: Woods, 2000. Since immunising childhood diseases have their greatest impact ages 1-4 years and are most lethal in large and dense populations, the omission of large urban settlements from the reconstitution sample may have led to under-estimation of the impact of smallpox to mortality in this age range.

[58] Mercer, 1990, chap. 3

[59] See for example Fleishman, 1985: 283 for estimates of the impact of vaccination in several northern rural and urban communities.

children).[60] The evidence is ambiguous however because the summer peak in neonatal mortality persisted despite evidence of increased maternal breastfeeding. Nonetheless it seems very plausible that part of the extravagant mortality of infants in eighteenth century London was due to an urban culture of limited or no maternal breastfeeding amongst certain sectors of the population, a culture which shifted over the course of the late eighteenth century. There is little doubt that maternal breastfeeding must have been very widespread in nineteenth century London. It is very unlikely that a city the size of London could have achieved infant mortality rates (and fertility rates) comparable to the national average and well below those of many much smaller continental towns without a high incidence of maternal breastfeeding.[61] It also seems very likely that this represented a profound change from practices amongst large sections of the metropolitan population before the late eighteenth century. The extent to which low levels of maternal breastfeeding was the norm in other urban centres in England before 1800 remains to be investigated. The evidence for Manchester suggests that early weaning was not a feature there before the late eighteenth century, and that the appearance of summer peaks in early infant mortality coincided with the development of factory labour and perhaps more regular employment of women away from home.

A third feature of Landers' model, spatial inequalities, appears to have been of pervasive if complex importance. Landers argued for the importance of the spatial structure of the national population and its degree of integration in determining the immune status of migrants from rural to urban areas. However the evidence presented here regarding smallpox complicates the elegant patterns of migration and endemicisation proposed by McNeill and incorporated into Landers' model.[62] Smallpox does not appear to have behaved as a classic immunising person-to-person infection endemic only in large dense populations. Rather it appears to have circulated efficiently in the more sparsely populated areas of northern England and less readily within the denser and apparently better connected settlements of southern England. The geography of smallpox epidemics is very puzzling and calls for closer analysis of regional patterns of disease transmission more generally. Similarly, the apparent heterogeneity of urban cultures with respect to wet-nursing and maternal breastfeeding suggested by the differences in seasonal patterns of neonatal mortality between London and Manchester also underscores the importance of regional variations to an understanding of the drivers of mortality trends.

[60] Fildes, 1986, 1998; Clark, 1988

[61] See Vögele, 2010 table 2 for a comparison of summer excess in infant mortality in the late nineteenth century in European cities.

[62] McNeill, 1984

At the finer spatial level Landers suggested that variations in housing conditions and the distribution of recent immigrants promoted spatial differences in disease patterns within the metropolis.[63] The evidence presented in this study could not address this question, except to reiterate for London that the rich may have fared no better than the rest of the population in terms of survival in the seventeenth and eighteenth centuries. In St. Martin in the Fields rich and poor were segregated only at the level of the street, with the wealthy more likely to live on broad thoroughfares and the poor in courts behind the main roads, a level of segregation apparently inadequate to shield the wealthy from the common infectious disease environment.[64] With increasing suburbanisation residential segregation became more pronounced. As Reid and colleagues have demonstrated, even at the end of the nineteenth century the strong gradient in infant mortality by social class was driven mainly by differences in the environments in which different social classes lived. Within a given environment differences by social class were minimal.[65] The limited evidence we have suggests therefore that increasing residential segregation and suburbanisation of urban populations was probably necessary to the emergence of the large spatial differences in mortality between different parts of cities evident in Victorian cities, and contributed to the shift from the promiscuous lethality of early modern cities to the more modest and differentiated mortality of Victorian cities.

A final issue raised by the evidence presented here is the extent to which the early modern urban mortality regime described by Landers' model was specific to north-western Europe in the early modern period. At least in English and Dutch towns regular annual burial surpluses may have been specific to the period c.1650-1770.[66] We have suggested here infant feeding practices and smallpox were significant causes of the high mortality associated with towns in this period. It remains an open question to what extent the urban graveyard phenomenon was a function of more ubiquitous structural characteristics of pre-transitional cities.

[63] Landers, 1992; Landers, 1993, chap. 8
[64] Dr Max Satchell, mapping of addresses at baptism by baptism fee and rateable value of property, unpubl. analyses.
[65] Reid, 1997; Garrett, Reid, Schűrer & Szreter, 2001
[66] Van der Woude, 1982

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