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

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Introduction

The limited evidence we have of mortality levels in historical urban populations indicates that mortality was very severe in English cities and towns before the nineteenth century. The seventeenth and eighteenth centuries in particular have been characterised as an 'urban graveyard' period, with mortality regimes so extreme that the population could only be maintained by sustained inflows of rural migrants to compensate for the excess of deaths over births of urban residents.[2] At the national level this period was characterised by a fall in life expectancy, although the extent to which this was due to trends in urban populations (and the rising proportion of the population that lived in urban settlements) remains unclear.[3] However from around 1750 urban mortality rates began to fall and by the mid-nineteenth century even the largest British cities were capable of natural growth, albeit with a persistent 'urban penalty' relative to rural areas. 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.[4] However the extent to which the excessive urban mortality of the period 1600 – 1750 can be regarded as typical of 'pre-transitional' urban mortality patterns requires further investigation, as does the assertion that nineteenth century patterns were fundamentally different.

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[2] Wrigley, 1967; Landers, 1993.

[3] Wrigley & Schofield, 1989; Wrigley et al., 1997.

[4] Landers, 1986; Reid, 1997; Smith & Oeppen, 2006; Kelly & O'Grada, 2014.

Figure 1 presents historical infant mortality rates (IMR - deaths in the first year of life per thousand births) for several cities and towns in England, together with rates for London Quakers and the national population. Two 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 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 in the national aggregate 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 still 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.[5] 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.[6] Even market towns of several thousand inhabitants in the eighteenth century, such as Banbury, rivalled the infant mortality rates of the great industrial cities of the nineteenth century. Thus the period 1750 – 1820 marked a profound transition at least with respect to infant mortality.

[Figure 1 about here]

The most successful analysis of early modern urban mortality patterns remains that provided by John Landers.[7] 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*).[8] He predicted that mortality would be very high especially in large cities, as a consequence of their density, size, and nodal position in trading and

[5] Galley & Shelton, 2001
[6] Galley, 1998
[7] Landers, 1992,1993
[8] See Landers, this volume.

migration networks. However mortality should also be relatively stable because urban food supplies were relatively reliable and immunity high amongst adults. Many of the prevailing infectious diseases in urban areas conferred lifelong resistance on survivors and so mortality should be most severe in young children and amongst newly arrived immigrants from rural areas where these diseases circulated only infrequently. Socioeconomic differentials should be muted by the intensity of the disease environment and the relative unimportance of nutritional status.

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.[9] London was the largest city in Europe in this period and a reservoir of endemic diseases. As expected mortality was very high (before the early nineteenth century) 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 to explain 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 factors that emerged most clearly from his study of London Quakers, and that were relatively independent of economic conditions: the medical

[9] Landers, 1993

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.[10] 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.[11] 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).[12] 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 wealthier Cheapside parishes was probably mainly a consequence of the prevalence of the practice of extra-parochial wet-nursing of infants of wealthier families.[13] 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.[14] Infants that died at nurse were usually buried in the nurse's parish rather than the London parish of their

[10] Landers, 1993; Newton, 2013
[11] 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).
[12] Newton, 2011; see also http://www.geog.cam.ac.uk/research/projects/earlymodernlondon/ and http://www.history.ac.uk/cmh/pip/resources.html
[13] In accordance with Finlay's evidence for wealthier London parishes in the seventeenth century: Finlay, 1981.
[14] Finlay, 1981; Fildes, 1986, 1988; Clark, 1988

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.[15] 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.[16] 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).[17]

[Figure 2 about here]

Patterns of neonatal mortality (mortality in the first month of life) were remarkably similar in Landers' Quaker population and St. Martin in the Fields. Neonatal mortality was very high in

[15] Newton, 2011.
[16] Davenport,Boulton & Black, 2013.
[17] 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.

both populations, and was associated with a pronounced summer peak (Table 1, Figure 3).[18] 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, in both St. Martin's and London Quaker samples. 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. Infants of wealthier families (especially the top ten percent of the wealth distribution) experienced higher mortality rates and shorter birth intervals than infants of poorer families, and displayed a summer peak of neonatal risk that was absent in poorer families. The absence of a summer peak amongst poorer neonates suggests that this seasonal 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 substitution of the more lethal practice of hand-feeding in preference to wet-nursing.[19] 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.

[Table 1 about here]

Figures 3,4 about here]

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 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.[20]

[18] Landers, 1993: 144
[19] Fildes, 1986, 1988
[20] Davenport, Boulton & Schwarz, 2011; 2015.

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.[21] 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.[22] 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).[23]

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.[24] 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.

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

[21] Davenport, Boulton & Schwarz, 2011; Razzell, 2011.
[22] Davenport, Boulton & Schwarz, 2011
[23] Woods, 2009; Davenport, Boulton & Schwarz, 2011
[24] Kelly and O'Grada, 2014.

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. However they also suggest, together with Landers' own findings, that much of the fall in urban mortality after 1750 was associated with specific changes in breastfeeding patterns and artificial immunisation against smallpox, rather than wider structural changes in the urban environment, economy or population.

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. The explosive growth of northern industrial and manufacturing centres has been argued to have resulted in worsening mortality conditions in the period 1820-60 as a consequence of administrative breakdown and rising social inequalities.[25] An alternative view is that any mortality rise that

[25] Szreter, 1997; Szreter & Mooney, 1998

did occur was largely a function of population density and exogenous epidemiological change.[26] The debate has centred on life expectancy and mortality rates during the period 1820-1850, when the paucity 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.

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, 'retail and handicraft' (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. Comparing infant mortality in each reconstitution settlement 1675-1749 with rates in the registration district that included the reconstitution settlement in 1838-44, 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 industrialising parishes.[27]

Figure 5 shows recent estimates of infant mortality in those reconstitution parishes where data were available for the period after 1750 and which Wrigley et al. classified as market town or industrialising.[28] Clearly the industrialising parishes did not enjoy the improvements evident in market towns. However given that the transition from a small agricultural to a large industrial population would generally be expected to result in a rise in mortality, the stability of infant mortality in these rapidly growing settlements suggests either that mortality improvements were sufficient to counterbalance the negative effects of urbanisation, or that infant mortality was relatively insensitive to urbanisation per se. This finding is in contrast to the higher estimates of infant mortality for these parishes in the second quarter of the nineteenth century and with the worsening of infant mortality in the industrialising midlands parish of Sedgley in the same period, and suggests that there may have been a deterioration in mortality in these settlements that coincided with the apparently widespread worsening of

[26] Woods, 2000: chap.9.
[27] Wrigley et al. 1997: 268-277
[28] Dawlish in Devon was also a market town but considered by Wrigley et al. to be atypical with respect to mortality patterns, as were all low-lying reconstitution settlements.

urban mortality after c.1820.[29] The stability of infant mortality trends in industrialising parishes in the eighteenth and early nineteenth centuries, while in contrast to our limited evidence for market towns and for London, is consistent with trends in infant mortality in the largest of the northern manufacturing towns, Manchester.

[Figure 5 about here]

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 6). 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.[30] 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).[31] By contrast Manchester's population density was exceptionally high even in the mid-eighteenth century (Figure 7), 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

[29] Wrigley et al., 1997: XXX; Levine, 1977; Kitson, 2014.
[30] Pooley & Pooley, 1984
[31] Bartlett, 1960

the parish (collegiate) church recorded age and cause of death for burials for much of the 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.[32] 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.

[Figures 6,7 about here]

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.[33] 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.[34]

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. To estimate infant mortality we required annual counts of baptisms (the population at risk) and of burials of infants. Since age was not recorded in

[32] Edge, n.d.
[33] Davenport, Boulton & Schwarz, 2014
[34] This assumption was given some validity by the very similar patterns of burials by age and cause at the collegiate church and St John Deansgate (see for instance Figure 11 below).

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 the age structure of burials at the collegiate and St. John Deansgate churches was representative of the whole population of Manchester town. To determine the total 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 registration was accompanied by a rise in private baptism.[35] 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

[35] Boulton & Davenport, 2014

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.

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.[36] 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. 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.[37] 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. The same cannot be said of baptisms. Although it appears that non-Anglicans used mainly Anglican facilities for burials before the nineteenth century in Manchester (giving relatively complete capture of burials in our sample), non-Anglican baptismal registration was far more extensive in the eighteenth century and it is probable that our capture of these events is incomplete. On balance the greater under-registration of baptisms than burials would serve to inflate our measures of infant mortality above their true levels.

[Figure 8 about here]

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 9). 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 and the 1770s. However the two periods for which we have estimates for the early

[36] Knodel & Kintner, 1977
[37] Wrigley et al. 1997: 232

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.

[Figure 9 about here]

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. However smallpox mortality appears to have been more severe in Manchester than London (see next section), suggesting that population size, at least above a certain threshold, may have been a relatively unimportant variable with respect to this most serious of eighteenth century diseases.

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 10).[38] 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

[38] Davenport, Boulton & Schwarz, 2014.

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 rates of smallpox mortality in infancy were higher in Manchester than St. Martin's.[39] Moreover the average age of child smallpox burials was lower in Manchester than in London.[40] 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 10 about here]

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"".[41] However Razzell has produced credible evidence of substantial vulnerability of immigrants in other southern towns in the late eighteenth century, for example Southampton.[42] By contrast scattered evidence from northern communities indicates that at least by the eighteenth century almost no adults died of smallpox (Figure 11). 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.[43] 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

[39] Davenport, Boulton & Schwarz, 2014
[40] Davenport, Boulton & Schwarz, 2014
[41] Creighton, 1894: 533
[42] Razzell, 2011
[43] Davenport, Boulton & Schwarz, 2015

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 11 about here]

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 9,10). 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.[44]

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. However a summer peak in burials at all infant ages 0-5 months emerged in the period after 1780 and persisted until at least 1850 (Figure 12). 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

[44] Aiken, 1795: 193

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.[45] 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.[46]

[Figure 12 about here]

Conclusions

Consideration of recent studies of mortality in 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.

However new evidence for Manchester, combined with the striking patterns of infant mortality observed in London, suggest that while Landers' model captures many important features of mortality in early modern English towns and cities, some of the most important aspects of mortality in this period are better explained by specific factors that fit only weakly within a structural model. Three factors appear to be of key importance: smallpox, infant feeding practices, and the geography of migration and disease transmission. Of these only the latter is a fundamental feature of Landers' model, and the patterns suggested here are more complex than expected.

[45] Vigier, 1970

[46] 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.

Landers identified smallpox as the major reason for the rise and fall of mortality in early childhood (ages 1-4) amongst London Quakers.[47] 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.[48]

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 population size, as seems to have been the case with measles and scarlet fever, 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[49]) 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 7).[50] 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.

[47] Landers, 1993: 154
[48] Carmichael & Silverstein, 1987.
[49] Fenner et al., 1988; chap.4; Walther & Ewald, 2004.
[50] The course of changes in IMR also appeared to follow changes in population density in Manchester (Figures 6 and 10).

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.[51] It is likely that the impact of vaccination on urban populations was very significant.[52] Moreover the benefit may have been greatest in small northern towns where smallpox appears to have constituted a high proportion of burials (Figure 13).[53] The extension of vaccination to rural populations would also have made towns substantially safer for adult migrants.

[Figure 13 about here]

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 children).[54] The evidence is ambiguous however because the summer peak in neonatal mortality persisted despite evidence of increased maternal breastfeeding. Nonetheless it

[51] 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.
[52] Mercer, 1990, chap. 3
[53] See for example Fleishman, 1985: 283 for estimates of the impact of vaccination in several northern rural and urban communities.
[54] Fildes, 1986, 1998; Clark, 1988

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.[55] 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. Infant feeding practices were included as a determinant of exposure potential in Landers' model, but the evidence of geographical, temporal and social heterogeneity in feeding practices suggests that this variable shouldn't be regarded as a structural feature of early modern urban populations.

A key 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.[56] 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, mainland Scotland and Sweden, 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

[55] See Vögele, 2010 table 2 for a comparison of summer excess in infant mortality in the late nineteenth century in European cities.
[56] McNeill, 1984

patterns of neonatal mortality between London and Manchester also underscores the importance of regional variations to an understanding of the drivers of mortality trends.

This chapter has been limited to a discussion of infant mortality, and this provides an incomplete test of Landers' model. Infants are buffered by (or exposed to) maternal immunity and care practices to a greater extent than older children, and infant mortality is less affected by environmental conditions than mortality at other ages. In the second half of the nineteenth century childhood diseases showed the strongest relationship of all causes of death to population size and density and early childhood mortality (mortality at ages 1-4: ECMR) exceeded infant mortality rates in large cities including London.[57] These patterns suggest that at least in Victorian England ECMR was a much more sensitive indicator than IMR of urban conditions, and an examination of early childhood mortality rates may provide a clearer test of the importance of structural determinants of exposure and resistance potentials and pathogenic load. Unfortunately these rates are also harder to determine for populations such as Manchester. However the enormous importance of smallpox at these ages must mean that this single disease, and specific prophylactic measures against it, largely determined urban mortality patterns in this age range in the eighteenth and early nineteenth centuries.

A final issue raised by the evidence presented here is the extent to which the 'pre-transitional' urban mortality regime Landers sought to 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.[58] We have suggested that 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.

[57] Woods, 1993; 2000
[58] Van der Woude, 1982

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