The decline of adult smallpox in eighteenth-century London. 1,2

Romola Davenport Leonard Schwarz Jeremy Boulton

The late eighteenth century was a crucial period in English population history, marking the beginning of the demographic transition. On the mortality side, the period saw a rise in life expectancy that was moderate in rural areas but resulted in the transformation of the urban mortality regime. Despite the huge significance of this period for our understanding of population growth and mortality decline, we still know little more than the bare outline of events. While the work of the Cambridge Group has provided very detailed information on the age patterns of mortality decline through the technique of family reconstitution³, we know very little about the changes in disease patterns that were the proximate cause of these changes, because parish data rarely included information on cause of death. Moreover the reconstituted populations did not include any large towns, and it is clear that early modern cities, and especially London, had very different mortality regimes from rural areas, and experienced more profound changes in the late eighteenth and early nineteenth centuries. However although we know relatively little about changes in death rates in urban areas, almost all we know of causes of death in this period comes from urban populations, because the main source for London and several other large towns, the Bills of Mortality, include information on cause of death. Used with caution, urban cause of death data offer a rare insight into epidemiological changes in the national as well as urban populations, in part because urban populations often contained large numbers of rural migrants. In this paper we discuss evidence from a novel source of mortality data, the sextons' books of the large London parish of St. Martin's in the Fields, which allows us to follow age-specific changes in smallpox burials, and provides a new insight into smallpox mortality in both London and its migrant hinterland. We use these data to test the hypotheses that mortality changes in the eighteenth century were the consequence primarily of a process of endemicisation of diseases such as smallpox, and that a decline in smallpox mortality occurred in the late eighteenth century.

At the national level, the frequency and amplitude of mortality crises declined over the seventeenth and early eighteenth centuries, as 'background' mortality levels rose⁴. A plausible explanation for this is the McNeill thesis⁵, that increasing economic integration was accompanied by epidemiological integration of the population, resulting in more rapid and frequent transmission of especially diseases such as measles and smallpox⁶. This would have increased the frequency of epidemics but reduced the impact of each

¹ This is a draft of a paper to be submitted to *The Social History of Medicine*.

² We would like to thank the Wellcome Trust, Award no. 081508 for funding the research on St. Martin's Bills of Mortality, and Drs. John Black and Peter Jones for inputting the data.

³ Wrigley et al., 1997

⁴ Wrigley & Schofield, 1989

⁵ McNeill, 1976

⁶ Wrigley & Schofield, 1989; Kunitz, 1983; Walter & Schofield, 1989, 66-68; Landers, 1993, 28-39

epidemic as an increasing proportion of the population acquired immunity, until epidemic oscillations became progressively damped and these diseases were relegated to childhood⁷. This account is consistent with the age patterns of death in the Cambridge Group reconstitution sample. While mortality rose at all ages in the seventeenth century (consistent with a rising risk of exposure to epidemic diseases), the first half of the eighteenth century was characterised by a striking divergence in life chances between children and young adults. A serious deterioration in infant and child survival was accompanied by a marked improvement in young adult life chances. Again, this pattern is consistent with a further stage in the process of endemicisation, as diseases such as smallpox and measles became endemic, infecting all children but leaving the survivors immune to attacks in adulthood⁸.

Of the diseases that displayed this pattern of endemicisation, smallpox was by far the most deadly in the eighteenth century. Smallpox accounted for 6-10% of all burials in the yearly London Bills throughout the century, with epidemics every two to three years (Figure 1)⁹. Famously, a third of all missing persons described in London's newspapers in the early eighteenth century exhibited smallpox scarring. Outside London the pattern of epidemic frequency seems to have varied, and we have no clear picture of the pattern and sequence of endemicisation¹¹. However wherever epidemics were frequent then smallpox would have contributed a significant proportion of all deaths.

While the process of endemicisation should be accompanied by an initial rise in mortality, as epidemics become more frequent, it has also been suggested that this period of heightened mortality should then give way to a more benign mortality regime, as lethal diseases are transformed into childhood illnesses. McNeill and Kunitz claimed that many diseases are less lethal in childhood than in adulthood, resulting in lower case-fatality rates as epidemic diseases become confined to childhood 12. They also suggested that prolonged exposure to endemic diseases would have selected for greater resistance in the population, contributing to lower mortality rates despite universal exposure. These points may well be true in the case of measles, but not with respect to smallpox. What evidence there is indicates that smallpox case-fatality rates were highest in infancy and early childhood, with a minimum in adolescence 13. There is also scattered evidence that case-fatality rates were rising in the course of the eighteenth and nineteenth centuries, with no indication of evolution of greater host resistance 14.

⁷ McNeill confined his hypothesis regarding the process of endemicisation to diseases with particular attributes, including human to human transmission without animal hosts, and the conferral of long-lasting immunity on survivors of infection.

⁸ The term 'endemic' can be used in a strict epidemiological sense to mean the state in which an infectious disease persists at a stable level, each infected individual infecting exactly one other. This situation is rare, and even very infectious diseases in large populations usually show epidemic behaviour. A looser definition, employed here, describes the more usual situation, where a disease persists in the population, producing cases every year but also fluctuating in its incidence.

Duncan et al. 1996

¹⁰ Thomas 1971, .8.

¹¹ Duncan et al. 1994. This issue is discussed further below.

¹² McNeill, 1976; Kunitz, 1983

¹³ Razzell, 2003, xvii-xviii, 166-168

¹⁴ Razzell 2003, 169-180; Hardy, 1983, 113. This issue is discussed further below.

Thus it seems unlikely that endemicisation of smallpox could have contributed to the improvement in life expectancy that occurred between the mid-eighteenth century and the second quarter of the nineteenth century. However it is possible that it may explain some some curious features of the age pattern of mortality decline. Mortality improvements in the second half of the eighteenth century and early nineteenth century were greatest in early infancy, where infectious diseases account for a relatively small proportion of deaths, and were negligible between ages 6-24 months, when infectious disease mortality is highest. Most deaths in the first weeks of life are considered to be 'endogenous', arising from hereditary and *in utero* conditions and the circumstances of birth, in contrast to deaths arising from exogenous causes in later infancy (mainly infectious diseases in this period). It is possible that much of the improvement in endogenous mortality derived from improvements in maternal health, against a background of persistent high infectious disease mortality of older infants¹⁵. In addition, older children and young adults enjoyed rapid gains in survival, consistent with a reduction in the average age at infection, brought about by endemicisation of major diseases.

Cities occupy a special place in McNeill's theory. As cities grew they became increasingly capable of sustaining infectious diseases in endemic form, by virtue of their large populations of susceptibles and high population densities. Moreover cities were necessarily more open to the introduction and reintroduction of infectious diseases, through their functions as commercial and political hubs. McNeill also drew attention to the peculiar age structure of the 'susceptible' population in cities¹⁶. In the case of diseases where infection conferred longstanding resistance upon the survivors, and where epidemics were frequent, almost all city-born adults would have been infected in childhood, leaving only children susceptible. However many immigrants to cities would have come from areas where such epidemics were still infrequent and a high proportion of adults were still susceptible. Thus in the period when such diseases were endemic in urban but not rural areas the age structure of mortality would be expected to show a bimodal pattern with peaks of mortality in young children and in the age groups where new migrants were concentrated (usually early adulthood). As these diseases became endemic throughout the population then the second peak, of immigrants, should have disappeared as smallpox became a childhood disease throughout the national population. London was a population sink until the late eighteenth century, consuming the population surplus of the countryside to maintain its rapid growth in the face of excessive death rates; as such, it drew in vast numbers of migrants, and most of these were young adults. Therefore the age pattern of a disease such as smallpox can serve as an indicator of the progress of the endemicisation of smallpox in the areas from which migrants to London were mostly drawn.

John Landers explicitly tested the McNeill hypothesis with respect to London in the period 1670-1830. Although he provided an exhaustive analysis of the London Bills of Mortality, his findings remained inconclusive due to several limitations of the Bills as

¹⁶ McNeill, 1980

¹⁵ Wrigley, 1998; Smith & Oeppen, 2006, 77

sources of evidence. The London Bills report burials by age and cause separately, so it is not possible to follow changes in the age pattern of burials by cause. Moreover the Bills do not allow calculation of any rates except the infant and maternal mortality rates, because only the number of burials and baptisms are known, but not the size nor age structure of the population at risk. To remedy this problem Landers also performed a family reconstitution exercise using records from two London Quaker Meetings, which generated age-specific mortality rates, and even mortality rates by age and cause for some causes. Apart from issues of representativeness of the Quaker sample, the study was limited by the small size of the sample (perhaps ca. 1000 people in the mid-eighteenth century¹⁷), which precluded the calculation of cause-specific rates for periods of less than fifty years.

As expected, Landers' data indicate rather different trends in metropolitan mortality from those of the wider population. As with the national population, London death rates appear to have risen in the seventeenth century, however metropolitan mortality worsened further in the first half of the eighteenth century, peaking in the 1740s. Infant mortality appears to have declined from the 1750s, with a hiatus c.1760-1775. Overall, the infant mortality rate declined from levels possibly exceeding 350/1000 births in the 1740s to approach the national average of c.160/1000 by the 1840s, an extraordinary achievement, and a much more profound change than occurred in the national population. Although the Quaker sample indicated that the greatest improvements were in the endogenous component of infant mortality, there was also a significant decline in infectious disease mortality, in both older infants and young children, in contrast to the Cambridge Group sample.

Landers concluded that while the high death rates in London, especially of children and young adults, fitted McNeill's 'high potential' model of urban mortality, the rise in mortality in the early eighteenth century, during a period when national rates were stable, did not. He suggested instead that sanitary conditions and crowding in the metropolis worsened in this period, and then improved after mid-century, contributing to the mortality reductions from 1750. However he also concluded that there was an autonomous role for smallpox. Smallpox was clearly endemic in London by the early eighteenth century, and smallpox mortality was concentrated amongst children and young adults, the latter presumably recent migrants. In Landers' Quaker sample smallpox accounted for nearly 30% of deaths in younger children (aged 1-4) and 18% of deaths in young adulthood (ages 20-29), in the period 1700-49¹⁸. Smallpox seems to have worsened in the 1720s and peaked in the 1760s, before declining at first slowly, and then more rapidly after 1800 (Figure 1). Landers attributed a large part of the reduction in infectious disease mortality of infants and young adults to a decline in smallpox in the second half of the eighteenth century, with an acceleration after 1800, when vaccination was introduced. While a reduction in crowding could have reduced transmission rates, he suggested that the early phase of the decline of smallpox may have been due in the main to the spread of the practice of variolation. Variolation involved deliberate inoculation with a small dose of (probably attenuated) smallpox virus, and became popular in

¹⁷ Landers, 1993, 133

¹⁸ Landers, 1993, 154

England from the 1760s¹⁹. Razzell has produced much evidence of the practice before it was superceded by vaccination with cowpox in the early nineteenth century, but the impact of variolation on smallpox mortality rates remains unknown. Any effect of variolation on smallpox rates in London probably occurred through variolation of migrants before migration, because variolation seems to have been relatively unpopular in the capital²⁰. Londoners did however take up vaccination with more enthusiasm, and this may account for the rapid decline in smallpox mortality after 1800. Smallpox was reduced from a major killer, accounting for 10% of all burials in the London Bills in the mid-eighteenth century, to a relatively minor cause of death by the 1840s.

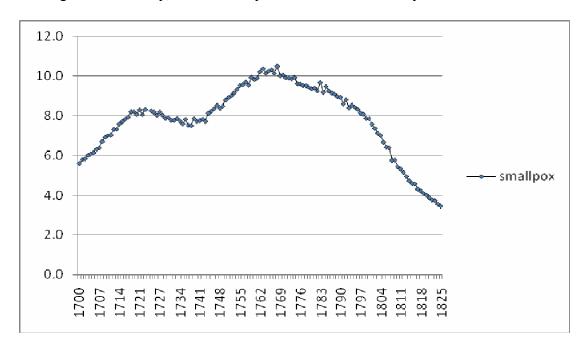


Figure 1. Smallpox as a percentage of all burials in London Bills of Mortality, 1700-1825. Source: Marshall (1832)

Other authors, notably Razzell and Mercer, have also emphasised the roles of variolation and later vaccination in reducing mortality in the eighteenth and early nineteenth centuries²¹. However the role of smallpox and of medical measures to curb its destructiveness have been rather neglected in the heated debate regarding the drivers of secular mortality decline. In particular, McKeown attempted to explain the late eighteenth century decline in mortality by extrapolation from his analysis of the Registrar-General's cause of death data from 1838 onwards, and was apparently unaware of the large decline in smallpox mortality in the intervening period, and so dismissed both variolation and vaccination as making an insignificant contribution to mortality decline²². This neglect of the eighteenth century may be justified to some extent. The mortality

¹⁹ Razzell, 2003

²⁰ Landers, 1993, 356; Razzell, 2003, 94-98

²¹ Razzell, 2003; Mercer, 1990, chapter 3

²² McKeown, 1976

decline in the late eighteenth and early nineteenth centuries accomplished no more than a return to sixteenth century life expectancies, and was followed by a period of stagnation and probably even a worsening of mortality levels²³. However this impression of the persistence of an *ancien regime* of mortality in the period 1750-1840 is swept away if one considers the extraordinary changes in urban mortality in this period. London was transformed from a demographic sink to a population capable of self-sustaining growth by the early nineteenth century. Even in Liverpool, the unhealthiest city for infants in Victorian Britain and an exemplar of the evils of rapid urbanisation, infant mortality rates in the second half on the nineteenth century were moderate by eighteenth century London standards (c.220-250 deaths/1000 births)²⁴. Clearly there were profound epidemiological changes in London and other English cities in the late eighteenth and early nineteenth centuries, but the nature of these changes, and their relationship to changes in the wider population, remain very poorly understood.

In this paper we present new evidence regarding smallpox in London in the period 1750-1805. We use this evidence to assess the extent of endemicisation in London's migration sphere, and to examine the causes of the apparent fall in smallpox mortality in the late eighteenth century, before vaccination. This study is pioneering in the sense that, unlike those who rely on the Bills of Mortality, we can look at the age-specific incidence of diseases in a large area of London from 1750 to 1805. It uses the sextons' records of the Westminster parish of St Martin-in-the Fields, giving the cause of death, age, name, address and burial fee of almost everyone who was buried in the parish.²⁵ These thus make it possible to quantify the incidence and location of smallpox at the heart of England and Europe's largest city. St. Martins was a large Westminster parish, with probably some 25-30,000 inhabitants throughout the course of the eighteenth century²⁶. Westminster had, even by London standards, a high proportion of recent migrants.²⁷ These migrants were predominantly young adults, with a high proportion of women for domestic service. The sexton's reports for the parish are remarkably complete. There is some omission of age and cause of death, but such omissions appear to be random. Exact age at death is given, and although there is considerable age heaping at older adult ages, this does not affect the ages where most smallpox burials occurred. Importantly, age at death was recorded in days, weeks and months for infants, allowing fine analysis of the age structure of mortality in infants and young children. Although most causes of death are problematic to identify in this period, smallpox was easy to recognise, and was probably reported fairly accurately. The only systemic bias is with fulminating smallpox, that killed before the pock-marks appear, and was most common in infants. Smallpox could be confused with severe chickenpox, but the latter was very rarely lethal. Where possible, totals of smallpox burials and burials by broad age groups have been corrected for missing ages and causes, but such corrections had little effect on the conclusions,

_

²⁷ Schwarz, 2002

²³ Wrigley et al. 1997

²⁴ Laxton & Williams, 1989, 113

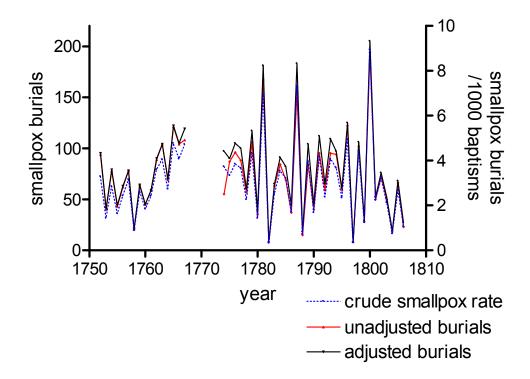
²⁵ About 1,000 burials per year.

²⁶ Or rather from 1725, when St. George Hanover Square formed a parish of its own. The population can be estimated roughly from the baptism rate over the period, which remained fairly constant at c. 800 per year over the whole period, by assuming a constant birth rate, such as 30 births/1000 population, or by assuming a basic continuity of age structure with the census population (25,752 in 1801).

given the lack of bias in these omissions. Although the dataset covers the period to 1825, data for years after 1805 have been omitted, because the workhouse burials were moved elsewhere from 1806, and although these records have been retrieved, they lack information on cause of death.

Smallpox in St. Martin in the Fields.

Figure 2 shows the total burials attributed to smallpox in St Martin's between 1752 and 1805. Where the 'unknown' ages are more than five percent of the total, the years have been omitted. This was only the case for 1747-51 and 1767-73. Figure 2 also shows the crude smallpox death rate, estimated using a population size calculated from annual baptisms on the assumption of a crude birth rate of 30 births/1000 population. While these rates are almost certainly wrong in detail, they are probably roughly correct. Importantly, the population of St. Martin's does not appear to have grown much over the period 1750-1805, at least if baptisms are an adequate guide to population size²⁸. The evidence suggests that smallpox mortality peaked in the 1760s, and only declined decisively after 1801.



²⁸ There are two reasons why they might not: first, it is likely that the recording of births declined over the late eighteenth century, so that baptisms progressively under-estimated births; and secondly, the rate of adult migration was not necessarily reflected in changes in the birth rate, because a high proportion of migrants to St. Martin's were unmarried females, and the skewed sex ratio amongst the adult population probably meant that many of these women remained unmarried or migrated to other parishes upon marriage. Therefore changes in migration rates might go unmarked in the baptism series.

4:06:21 PM 12/25/2009

Figure 2. Annual smallpox burials in St. Martin in the Fields, and crude smallpox burial rate²⁹.

Table 1 gives the ages of smallpox deaths in St Martin's. It must be stressed that this information is virtually unique for this period, being based on large sample sizes with little bias in omission by age and cause.

Age	1752-66	1775-99
0	13.7	23.3
1-4	54.5	61.5
5-9	10.9	9.4
10-19	4.6	1.8
20-49	15.6	3.5
50+	0.7	0.6
Mean age at death (years)	7.8	3.9

Table 1. Percentage age distribution of burials from smallpox in St. Martin's, 1753-66, 1775-99. 30

The contrast between the two periods is obvious. The effect of the influx of young people to London is clear *only* in the earlier period. There is a small bulge of those aged fifteen and over, with another bulge for those aged twenty and over. What is remarkable is that

²⁹Burials included only named smallpox burials ('unadjusted'), or adjusted for missing causes. The crude smallpox burial rate was calculated using adjusted burials, and assuming a population size calculated using a crude birth (baptism) rate of 30 births/1000 inhabitants. Years where missing ages exceeded 5% of the total burials were excluded.

³⁰ Data derive from the St. Martin's sextons' books. The periods include only years where burials without exact ages formed less than 5% of the total. Smallpox burials were adjusted for burials of unknown age and cause. Most burials without exact age were designated as child ('C') or adult ('M' or 'F') in the sextons' books, and almost all child burials were aged under ten, where exact age was given. Where cause was given but not exact age, burials were distributed to exact ages using the cause-specific distribution of burials by age for age groups under 10 or ten and over. Burials with no cause of death given were first distributed to exact ages, and then distributed according to the age-specific ratio of smallpox burials to other causes. Almost all smallpox burials included exact age, and there was little age bias amongst burials with no given cause. Therefore the patterns produced by the redistribution of burials of unknown age and cause did not differ very significantly from those of unadjusted smallpox burials. This is in contrast to Landers' analysis of London Quakers, where the redistribution of deaths of unknown cause caused large changes in the age patterns of smallpox burials especially at younger ages (Landers, Death in the Metropolis, pp. 153-154). Both Landers' and the current analysis assumed that the risk of omission of cause of death was independent of the cause. This assumption is not critical in the case of St Martin's, because the adjusted series is not very different from the unadjusted (using only burials explicitly described as smallpox victims). However if smallpox were more likely to be recorded than other causes, this may invalidate some of Landers' conclusions.

this is almost absent in the last quarter of the century. This represents a key finding of this study. The decline in smallpox was associated with a dramatic decline in smallpox deaths amongst adults, and a concentration amongst children under five.

The large size of the St. Martin's population makes it possible to analyse the smallpox burials by single years, and thus to pinpoint the period when this dramatic age shift occurred. Figure 3 shows the proportion of smallpox burials attributed to adults (corrected for missing causes and ages). A feature of the decline is its rapidity. Adults halved as a proportion of smallpox burials (from 20% of all smallpox deaths to 10%) in a six year period 1769-1774. Although this decline coincided with a period of poor recording of age and cause in the sextons' books, there was no corresponding change in the proportion of adults dying from all causes, so the decline in adult smallpox deaths cannot be attributed to some artifactual change in age recording or a precipitous decline in adult immigration³¹. Rather, Figure 3b indicates that smallpox ceased to constitute a major risk to adults, as indicated by the steep reduction in the proportion of adult burials attributed to smallpox. The reduction in adult smallpox burials was preceded by a rise in smallpox mortality in the 1760s, indicated by an absolute rise in smallpox burials in both London (Fig. 1) and St. Martin's (Fig. 2) and a rise in the proportion of burials attributed to smallpox (Figure 3b). Also notable is that the decline in adult smallpox mortality was accompanied by an increase in the importance of smallpox as a cause of death in children. This is discussed further below.

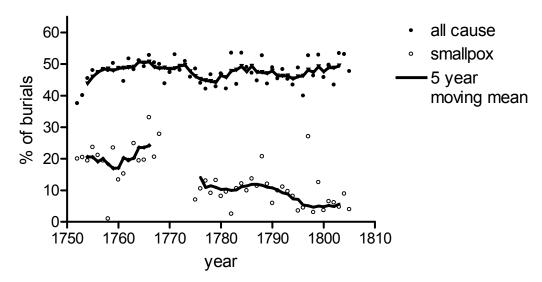
Since this phenomenon, of precipitous changes in the age structure of smallpox mortality, appears in a local study, we need to establish that it is not a product of some local peculiarity in the care of smallpox sufferers. Were adult burials being sent outside the parish to institutions? One institution purposely designed to do just this was the London Smallpox Hospital, established in 1746. This laid down that no person under the age of seven was to be inoculated. It admitted only persons over the age of seven and recommended by one of the subscribers. Between 1746 and 1763 the Hospital admitted 6,456 persons, or an average of 380 a year; from 1776-1800 it took 7017, or an average of 281 a year, a miniscule number in a city the size of London. Moreover, some of these were for inoculation only. Since the case fatality rate in the Hospital was only about a quarter, the Smallpox Hospital cannot have removed more than a hundred or so victims per year from the entire metropolis, and thus cannot explain the disappearance of adult smallpox victims.

_

³² Razzell, 2003, 96; Woodville, 1796, ii.231

³¹ Adults aged 10-39 accounted for 17.8% of burials in years 1752-66, and 16.6% in 1775-99., indicating that this age group probably remained fairly stable as a proportion of the population, and that there was no abrupt change in the level of migration into St. Martin's.

a Adult percentage of burials (ages 10+)



b Smallpox percentage of all-cause burials

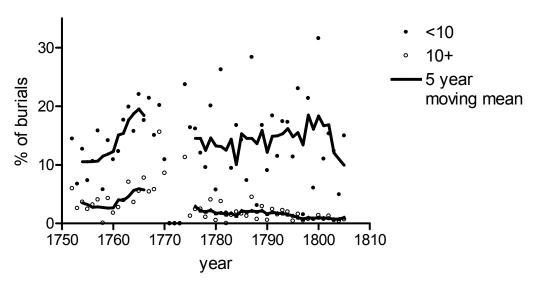


Figure 3. St. Martin's, percentage of adult smallpox victims, and percentage of all burials attributable to $smallpox^{33}$

However, to be sure that St. Martins was indeed typical, it was necessary to study another parish. Fortunately, it was possible to do this for the large East London of St. Dunstan Stepney. The parish had a larger population as St. Martin's – about 40,000 in 1801 – and covered a very different, and poorer part of the capital.³⁴ A reasonably complete set of its

³³ Smallpox and all-cause burials were adjusted for burials with missing ages and missing causes, as described in Table 1.

³⁴ Schwarz, 1982.

sextons' records have survived. Figure 4 shows that the same trend was at work. The two parishes are remarkably similar.

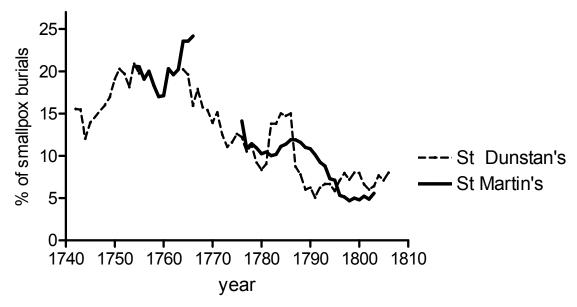


Figure 4. Proportion of smallpox deaths aged ten and over, five year moving average, St. Dunstan's, Stepney, and St. Martin in the Fields.³⁵

We can therefore conclude that by the last quarter of the eighteenth-century, smallpox in London was confined largely to childhood. Such a change in the nature of smallpox mortality is also suggested for London as a whole by various aspects of Landers' analysis of the London Bills. He found smallpox burials to be associated with child and young adult burials before 1775, but more closely with child burials subsequently³⁶. Moreover before the 1770s smallpox mortality was positively associated with conditions that favoured migration of especially young adult males into the capital, specifically demobilisation of armed forces after conflicts³⁷. From the 1770s onwards these associations ceased to be significant (although they remained significantly associated with fever mortality, indicating that the migratory patterns supposedly underlying these phenomena probably had not changed)³⁸.

.

³⁵ St. Martin's smallpox burials are adjusted for burials with missing ages and missing causes, as described in Table 1. In the case of St. Dunstan's only smallpox burials were transcribed from the sextons' books, so these have been corrected for missing ages but not unknown causes. Cause of death data were missing for the years 1756-63 in the St Dunstan's sextons' books.

³⁶ Landers, 1993, 115-119.

³⁷ Landers, 1993, 288, 297-8

³⁸ Contrary to these indirect forms of evidence from the London Bills, Landers' analysis of the London Quakers did not show any evidence of a shift in age structure of smallpox mortality after the 1760s. However any such shift would have been difficult to detect because the small numbers of burials involved precluded further division of the data beyond the 50 year periods 1700-49 and 1750-99. There was some evidence of a reduction in adult burials in the second period amongst deaths explicitly labeled as smallpox,

The fact of this decline in adult smallpox is of key importance in understanding the decline in smallpox in the eighteenth century. If this change in age incidence proves to be a general phenomenon, this has important implications. Since the disease seems to have remained common amongst the capital's children during the eighteenth century, and assuming that adults were as likely to encounter such children as before, then a decline in the incidence of the disease amongst adults suggests very strongly that London migrants were increasingly immune. Such immunity amongst London's migrants could have been due to either:

- a) A spread of endemic childhood smallpox throughout London's migrant catchment area
- b) Inoculation and later vaccination of virtually all London migrants.

What evidence is there to support our empirical findings? In the later seventeenth century Sydenham regarded smallpox as liable to occur to adults, smallpox 'seizing whole families and sparing none of what age soever they be, unless such as have already had it'³⁹A century later doctors were claiming that smallpox was predominantly a disease of children in London. Table I shows that it was largely so in the mid-eighteenth century, and overwhelmingly so during the last quarter of the century

Outside London, the extent to which smallpox was a childhood disease by the mideighteenth century remains unclear. Razzell has collated evidence of very low levels of adult smallpox mortality (defined as ages 21+) in a small number of northern towns and smaller settlements in the first half of the eighteenth century. In Kilmarnock, Scotland, no adults were recorded as dying of smallpox between 1728 and 1763, and in Manchester only one adult (aged over 20) was recorded as dying, of 589 smallpox victims in the period 1769-74⁴¹. If correct, these figures imply that even most migrants to these northern towns were immune to smallpox. However at least in parts of southern England smallpox seems to have remained an episodic disease, affecting adults as well as children, until at least the last quarter of the eighteenth century⁴². For example, a smallpox outbreak in the market town of Burford in 1758 caused very high mortality amongst adults as well as children, with perhaps less than 40% of deaths occurring amongst children under ten⁴³. A smallpox 'census' of Stratford upon Avon in 1765, taken to ascertain the numbers of inhabitants vulnerable to smallpox, indicated that many adults lacked immunity⁴⁴. Razzell concluded that smallpox mortality exhibited a strong north-south divide, with adults comprising a substantial proportion of smallpox burials only in southern settlements⁴⁵.

but this disappeared when the deaths were adjusted for burials without recorded cause – see footnote to Table 1.

³⁹ Sydenham, 1742, 96-125

⁴⁰ Razzell, 1977, 28.

⁴¹ Razzell, 2003, xi-xiii

⁴² ibid.

⁴³ Ibid.; Gani & Leach, 2001

⁴⁴ R Davenport, unpublished analysis of data from the Shakespeare Trust. In addition, the source lists the settlement status of inhabitants, and analysis indicated no difference in risk between those adults with settlement and those migrants without settlement in the town.

⁴⁵ Razzell, 2003, xii-xiii.

From these fragmentary sources of evidence it seems possible that at least in the mainly southern communities from which most migrants to London were drawn, smallpox was not endemic by the mid-eighteenth century. This contrasts with contemporary claims that smallpox was endemic in the dispersed populations of mainland Scotland and Ireland, and vital registration evidence indicating that smallpox was endemic in rural Sweden at least from the mid-eighteenth century⁴⁶. While the reasons for the persistence of adult susceptibility in the south of England are unclear, it seems likely that migrants from these southern settlements accounted for the bulge of adolescent and young adult smallpox burials in London. Where smallpox was endemic adults (those aged 10+) comprised less than 10% of all smallpox burials⁴⁷. Although London had an unusually high proportion of adults in the population, the constant influx of young adult immigrants is not sufficient in itself to account for the bulge of smallpox burials at these ages, without the further assumption that young adult immigrants were at higher risk of smallpox⁴⁸.

Who died of smallpox?

Smallpox was clearly endemic in London, and children under five were the main victims. While most of these were probably London-born, the identity of young adult victims is less clear. Theory suggests that they were mainly recent migrants from areas where smallpox was still an infrequent epidemic disease. Landers identified demobilised young males, and more generally subsistence migrants, as the main candidates. Both Landers and Galloway, using different statistical techniques, found strong associations between smallpox mortality and wheat prices, and poor weather, suggesting a connection between migration into London during periods of dearth and elevated smallpox mortality⁴⁹. The information on sex and burial fees in the sextons' books allow us to test these propositions with respect to St. Martin's. These provide equivocal support for Landers' thesis that smallpox mortality tended to peak with the influx of immunologically naïve subsistence migrants in periods of dearth or demobilisation. Although the St Martin's population was heavily female-biassed, due to the predominance of women in domestic service, young adult smallpox burials were significantly more likely to be male than other causes of death at these ages, before 1767 (Table 2). After 1774 the pattern changed markedly, to one resembling the low sex ratio of other causes of death. At the same time the adult proportion of smallpox burials dropped after the 1760s to levels typical in populations where smallpox was endemic (ca. 5%)⁵⁰. Such a change in the sex ratio of smallpox burials supports the idea that military recruits from areas where smallpox was not endemic could have swelled the numbers of adult smallpox victims before more

46 Razzell, 2003, xiv; Skold, 1996, 261.

⁴⁷ In Sweden adults aged 10+ comprised 5-6 % of smallpox deaths in the period 1776-1805; Skold, 1996, 261.

⁴⁸ This is because in an endemic situation the number of smallpox burials should decline exponentially with age (assuming similar age-specific case-fatality rates), so the age structure of the population would need to be distorted to an improbable degree by migration to account for the excess of young adult burials (calculations not shown).

⁴⁹ Landers, 1986: Galloway, 1985

⁵⁰ Note that this implies a very high level of endemicity in London, because the age structure of the population was much older than that of national populations, due to high rates of adult immigration.

widespread endemicisation. However there was no evidence that adult smallpox victims were more likely to be poor (that is, subsistence migrants). In fact both child and adult smallpox burials were less likely to be pauper than were burials from other causes, and this relationship was especially strong before 1775 (Table 2). Meier reported a similar distribution of smallpox burials by cost for St Martin's in the seventeenth century⁵¹. Therefore if migrants comprised the majority of adult smallpox victims before 1775, then these migrants were typically neither destitute, nor predominantly female domestic servants.

These findings are intriguing. The evidence that females were at lower risk than males before the 1770s is consistent with the evidence that female servants in this period tended to move to London via other urban centres⁵², where they would probably have encountered smallpox if they hadn't before. Moreover they may have been reluctant to enter the capital until they had been inoculated, naturally or artificially, against smallpox (see below). Unfortunately the sextons' books do not contain occupational data, so it is not possible to determine whether smallpox victims were more likely to belong to certain occupational groups, such as apprentices or artisans, who might have been drawn from a wider geographical sphere than domestic servants, that included more isolated areas. St. Martin's was also a point of entry for elite families, residing in London for the Season and drawn from all over the country. However very few smallpox burials were in the highest quintile of burial fees, so this particular group of migrants cannot account for the curious patterns of smallpox burials by sex and status before the 1770s.

After 1775, the changes in the proportion and sex ratio of adult smallpox burials suggest that there was no longer any distinction in smallpox risk between the London-born and immigrant populations. These dramatic changes in the pattern of adult smallpox deaths might point to widespread inoculation, or to a change in the nature of the disease.

-

⁵¹ Meier, 2009, 107

⁵² Sharpe, 2000

Period	other causes	Smallpox	cause not given	
sex ratio (male:female)				
1752-66	65.03	122.99**	61.11	
1775-99	77.91	83.67	108.03	
	% paupers			
1752-66	46.30	25.77**	31.82	
1775-99	54.67	38.46	21.28	
	geometric mean cost of non-pauper burials			
1752-66	353.60	325.11*	340.68	
1775-99	158.05	173.24	76.43	
	sample size			
1752-66	1812	194	29	
1775-99	3423	90	285	

Table 2. Sex ratios and cost of burial for adults aged 10-39, by cause and period⁵³.

Inoculation

Peter Razzell is the main exponent of the importance of variolation in the eighteenth century, and dates its rise in popularity, especially outside London, to the 1760s. While the coincidence in timing is striking, it seems unlikely that such a novel and apparently risky practice could have spread fast enough to explain the sudden drop in adult smallpox burials in London. However it is possible that variolation was adopted with particular avidity by would-be migrants, as suggested by this treatise of 1767:

'No sooner are the lower sort recovered [from inoculation], but they aim (the women especially) to get a servitude in London, or to use their own words *to better themselves*; this is the only objection that can be made to inoculation, and indeed it is one, for before they did not dare to quit to quit the place of their birth for fear of that distemper, so remained honest and useful in the country'⁵⁴

The author of this treatise was so splenetic towards the lower orders that his objectivity must be in considerable doubt; however the passage might suggest that, like modern day travellers from the developed world to Third World Countries today, inoculation became part of the preparation of the sensible London migrant.

5

⁵³ Geometric mean cost was calculated for non-pauper burials to normalise the distribution of costs. Asterixes indicate statistical significance of differences between smallpox and other named causes, *P<0.05, ** P<.01. Statistical tests used were Fisher's exact test for differences in proportions (sex ratios and proportions pauper), and one-way ANOVA tests for differences in geometric mean cost. Sample sizes refer to the samples used for calculations of sex ratios and % pauper.

⁵⁴ Anon., *Considerations on the dearness of corn and provisions*, p. 7, also quoted (with wrong title) by Razzell, 1977, 60.

Once in London, the evidence is inconclusive. Dealing with specialised institutions that provided inoculations to adults is straightforward. Within London there was only the London Smallpox Hospital, founded in 1746 but, as noted above, this only provided some 632 inoculations in an average year between 1746 and 1832.⁵⁵ In its early years it was anything but popular; when it moved to new buildings in Clerkenwell in 1752, patients leaving the hospital had to leave by night, to avoid abuse and insult, at any rate according to Woodville writing almost half a century later. 56

Lettsom sought to establish a society for inoculating the London poor in their homes in 1775, presumably adults as well as children. It failed, partly owing to the opposition of Dimsdale, an eminent and opinionated doctor, who feared the consequences of introducing infection in London - as if smallpox in London were not already endemicbut perhaps also because of popular indifference to inoculation. Lettsom tried and failed again in 1779.⁵⁷

There was, however, one set of institutions in London that did inoculate poor children whatever their parents thought, and these were parish workhouses and the Foundling Hospital.⁵⁸

The Foundling was doing this already in 1743 and in 1749 they advertised that all the children who had not had smallpox when at nurse would be inoculated before their return, when they were still safely outside the metropolis.⁵⁹ The parish of St James, Westminster entered it in their standing orders in 1756:

All the children are inoculated for the smallpox when deemed proper by the surgeon, and is paid ten shillings and sixpence for every child that survives that disorder.

The nurse is likewise paid the shillings and sixpence for every child that has it in the natural way, or is inoculated and survives, but not else. 60

It is likely that a number of other London parishes did this, and the documentation may come to light. However, the London workhouses, large though they were, served only a small proportion of a parish's poor, and many of these never reached adulthood. In the nineteenth century, when vaccination was safer than inoculation ever had been, Anne Hardy has reported many cases of its absence. For instance, a survey of the nearby parish of St. Margaret and St. John, Westminster in 1840 found that 40 per cent of 6163 children had not been vaccinated.⁶¹

⁵⁸ There were also the armed forces, where inoculation seems to have been rather an ad hoc affair, sometimes given to soldiers and sailors, sometimes not. In 1756 and 1775 sections of the Army were inoculated: Kopperman, 2007; and for the Navy a rather uncertain reference in Lincoln, 2007.

⁵⁵ Razzell, 1977, 72. The Hospital would not inoculate anyone under the age of seven.

⁵⁶ Woodville, 1796, i. 237-238

⁵⁷ Razzell, 1977, 71.

⁵⁹ We owe this information to Dr. Alysa Levene.

⁶⁰ Sketch of the state of the children of the poor in the year 1756.... in the parish of St James, Westminster

^{61 &#}x27;Report of a Committee of the Statistical Society of London, on the State of the Working Classes in the Parishes of St. Margaret and St. John Westminster'., Journal of the Statistical Society of London, Vol. 3,

'It seems reasonably clear that vaccination (and inoculation) per se could not have been the sole instrument in the disappearance of the disease, because of prejudice, apathy, ignorance, and absence of enforced re-vaccination.'62

A lesser degree of opposition to inoculation is unlikely in the eighteenth century.

Although inoculation remained unpopular in London in the eighteenth century, it is possible that the rural surge in popularity of inoculation in the 1760s induced many young adults to seek inoculation before migration to London. However there is also evidence in the St. Martin's burials that, perhaps in addition to changes in individual behaviour, there were changes in the behaviour of the smallpox virus itself.

Smallpox: a biological shift?

Close consideration of the age pattern of smallpox mortality amongst children in St. Martin's, most of whom would have been London-born, suggests that the virus itself changed, to become more infectious. The evidence for this is that in the same period that adult smallpox burials halved, the age distribution of smallpox burials amongst children under ten also shifted to younger ages. In particular, the infant smallpox mortality rate doubled, while rates in older children probably declined. Figure 5a shows the total and smallpox infant mortality rates per 1000 baptisms. While there was some increase in the total infant mortality rate, the smallpox rate doubled, from around 15 to 30 deaths/1000 baptisms from the mid-1770s. Smallpox also rose from 4% of burials to account for almost 7% of burials in the first year of life (Figure 5b). At the same time, smallpox declined as a proportion of burials in older children, suggesting increased levels of immunity at these ages. While the most obvious explanation for such an effect is an increase in population density, caused for instance by an increase in the birth rate, there is no evidence in the baptism series for any increase in births⁶³. Obviously, care must be exercised in interpreting these data. The total infant mortality rate in Figure 5a is very high, suggesting an under-recording of baptisms particularly in the last quarter of the century. However there was no evidence of any bias in recording of smallpox burials that would have caused such a great increase specifically in smallpox burials compared to other causes. However the ratio of burials in early infancy to burials in later infancy declined over the period (see burial totals in Table 3), and it was possible that this reflected in part a deterioration in the recording of neonatal deaths. Since smallpox mortality was higher in the later months of the first year of life, deficiencies in recording of deaths in early infancy could have exaggerated the importance of smallpox as a cause of infant death (although these could not account for the doubling of the smallpox infant mortality rate). Table 3 presents smallpox burials as a proportion of all burials in early

4:06:21 PM 12/25/2009

No. 1 (Apr., 1840), p. 18. 'Children' were not defined – presumably they were living with their parents, and exactly this number -6163 – were given as liable to attend school: Ibid., p. 20.

⁶² Hardy, 1983, 138

⁶³ Such an increase should also have reflected in a decrease in the average age of measles burials, which did not change – see below.

and late infancy (the first and second six months of life). In both age groups smallpox burials increased by nearly 50% as a proportion of all burials, indicating that the rise in importance of smallpox as a cause of death in infancy was not simply a consequence of a shift in the pattern of burials between early and late infancy. Notably, even very young infants were at risk of smallpox, because although maternal smallpox antibodies transferred *in utero* conferred immunity to infants of immune mothers, this immunity waned quickly, regardless of breastfeeding⁶⁴. Thus at high levels of infectiousness and epidemic frequency smallpox could become concentrated in infancy (as indicated in Table 1, where infants accounted for almost a quarter of smallpox burials in the period 1775-99).

This rise in importance of smallpox in infants and decline at older ages is consistent with a reduction in the average age of infection. Such a reduction, in the London-born population, indicates an intensification of the endemicisation process⁶⁵. In the absence of any abrupt increase in population density, the most plausible explanation is an increase in infectiousness of the smallpox virus. Obviously smallpox was already endemic in London by the mid-eighteenth century, as indicated by the concentration of smallpox deaths in early childhood, and the biannual cycle of epidemics⁶⁶. The high population densities and ease of mixing would have resulted in relatively efficient transmission of the virus, and the main effect of any increase in infectiousness would have been a further concentration of infection in infancy. The effect we have detected here is therefore rather small, given the already low age of childhood infection in London. However the impact on small or isolated populations would have been much more profound. Smallpox transmission was relatively weak compared for instance to measles, and smallpox victims were usually only infectious once the symptoms were apparent, and were often too sick to move about⁶⁷. This probably made it possible for communities in southern England to avoid smallpox for years at a time (by luck and quarantining) and ultimately made it possible to eradicate smallpox globally with relatively low levels of vaccine coverage. An increase in infectiousness would have raised the chances of infection in infancy and early childhood in large urban populations, and at the same time favoured the endemicisation of smallpox in rural communities (by increasing the frequency of successful introduction of the virus, and reducing the effectiveness of quarantine). A higher rate of introduction, and more efficient transmission within a community (resulting in higher rates of export to other

-

born population directly, since these are not identified in the source.

⁶⁴ Breastfeeding does not provide specific immunity to viral diseases such as smallpox, although it may reduce the ability of viruses and other pathogens to gain entry via the digestive and respiratory tracts. See for instance Hoshower, 1994. In the twentieth century neonates were frequent victims of smallpox, and so vaccination of newborns was the preferred immunization strategy. Of course, neonates of non-immune mothers were particularly vulnerable, and some neonatal smallpox deaths presumably belong in this category, although the proportion of non-immune mothers presumably declined steeply after the 1770s. ⁶⁵ The average age of smallpox victims halved over between the third and fourth quarters of the eighteenth century (Table 1), however it is not possible to estimate the change in average age at death for the *London-*

⁶⁶ Duncan et al., 1996

 $^{^{67}}$ Foege et al., 1975; Arita & Wickett, 1986. Estimates of ' R_o ' values (a measure of the number of susceptibles infected by a single smallpox case, at the beginning of an outbreak) for smallpox are low compared to most epidemic diseases (ca. 4-5): Anderson & May, 1991, 70; Gani & Leach, 2001. However Gain & Leach also suggest that R_o values could have been as high as 10-12 in the crowded conditions of eighteenth century London.

populations) would have promoted unification of small populations into a single epidemiological population. The period of sustained smallpox mortality in the 1760s may thus represent a period of adjustment, in which smallpox circulated more rapidly within the London population and enjoyed a large pool of child and young adult immigrant susceptibles. As smallpox became endemic in areas previously subject to only sporadic visitations, then the vulnerability of adult migrants would have declined, and smallpox would have become a disease of childhood in both London and its hinterland.

Other evidence for such a scenario, of increasing infectiousness, comes from case-fatality rates. In smallpox, infectiousness is usually associated with virulence (lethality), since both depend on the number of viral particles produced. Razzell has argued for an increase in virulence over the eighteenth century, and produced some evidence for an increase specifically in the 1760s.⁶⁸ Anne Hardy found an increase in the smallpox case fatality rate at the London smallpox hospital from 255 per thousand between 1746 and 1763, to 320 per thousand between 1776 and 1800.⁶⁹ This apparent increase in lethality in the last quarter of the eighteenth century would be expected to be accompanied by an increase in transmission of smallpox.

The evidence for a shift in the infectiousness of the smallpox virus is tenuous, and depends on relatively small changes in the age pattern of mortality, raising the possibility that these changes are artifactual. The most serious problem in using burial records as evidence of mortality patterns is the lack of information on the 'population at risk', that is, how many people in each age group comprised the population from which the burials were derived. Changes in the absolute numbers of people at risk, or the relative sizes of different age groups, can cause significant changes in the numbers and age distribution of burials, without any real change in the age-specific rates (burials per thousand persons of a given age). In the case of St. Martin's there were several factors that probably operated to cause fluctuations in the numbers at risk, including relatively high levels of migration, and a traffic in corpses for burial between parishes⁷⁰. Therefore we used several methods to test whether the observed patterns of smallpox burials reflected 'real' changes in smallpox mortality rates and age patterns. In addition to basic checks comparing the numbers of all-cause burials to smallpox burials (see previous figures), we also compared St. Martin's with the parish of Stepney, where possible⁷¹. As another type of internal check, we compared burials attributed to smallpox with those of measles. Measles and smallpox are both viral infections spread by droplets and conferring long-lasting immunity on survivors, and are very similar with respect to 'generation time' (the average period between contact with an infectious person and onset of symptoms in the newly infected person)⁷². However measles is more infectious, and therefore appears to have been more fully endemicised by the mid-eighteenth century in London. In St.

60

⁷² Anderson & May, 1991, 129

⁶⁸ Razzell, 2003, xxii

⁶⁹ Hardy, 1983, 113. Hardy gives a fatality rate 'between 20 and 40 percent' for the nineteenth century: Hardy, 1993, 113.

⁷⁰ Boulton, in prep.

⁷¹ It was not possible to compare changes in smallpox burials at single years of age in Stepney, because until 1756 ages under 2 were recorded only as being children, and could not be separated from other child burials at older ages. From 1757-1773 age was poorly recorded.

Martin's 99% of all measles burials were aged under ten by the mid-eighteenth century. and most deaths occurred at ages under five. By contrast, only 79% of smallpox burials were aged under ten in this period (1752-66), and burials of children were less concentrated at the earliest ages. To examine changes in age at death amongst the London-born population, we restricted the analysis to children under ten years old, most of whom would have been born locally (Fig. 6a,b). In the last quarter of the eighteenth century measles showed little change in age pattern, indicating that there were no large changes in the age structure of the child population (the age distributions between the two periods did not differ significantly in a two-sample Kolmogorov-Smirnov test). However smallpox mortality shifted to resemble that of measles, with burials concentrated in the first two years of life. Indeed, smallpox burials became even more concentrated in the first year of life than was the case for measles, probably because maternal-derived antibodies to measles persist for longer in the infant than antibodies to other viruses⁷³ including smallpox. A similar pattern held in Stepney after 1773 (Figure 6b)⁷⁴, and in Sweden, where infants accounted for around 30% of smallpox deaths in the last quarter of the eighteenth century⁷⁵. The shift in the age distribution of smallpox burials in childhood was substantial (P=0.000 by two-sample Kolmogorov-Smirnov test), and when contrasted with the lack of change in the age pattern of measles burials, indicates a genuine reduction in the age of death (and therefore age at infection) from smallpox after 1775.

If smallpox did become more transmissible, resulting in endemicisation throughout the migrant hinterlands of London, then there should be other evidence of this in the hinterlands themselves. As described earlier, there seems to have been a north-south divide in the age distribution of smallpox deaths before the 1770s, with adults significantly at risk only in southern settlements, but the evidence gathered by Razzell is too patchy to detect changes over time. Few parishes recorded cause of death over long periods in the eighteenth century, but one example is Ackworth, Yorkshire, where there was a dramatic increase in the number of smallpox burials relative to all burials in the 1760s⁷⁶. A change in the nature of the smallpox virus should also be detectable in other populations in contact with London. The evidence from Sweden and Copenhagen is suggestive. Swedish smallpox mortality data indicate an abrupt decline in smallpox deaths from the 1770s⁷⁷, and by the 1780s smallpox was clearly highly concentrated in infancy, with adults (ages 10+) accounting for less than 6% of deaths 78. The decline in smallpox from the 1770s is apparently inconsistent with a rise in smallpox infectiousness and probably virulence, but the timing is conspicuous. It is probable that any shift in the average age of infection to younger ages would have resulted in a greater level of underrecording of smallpox mortality, because the disease seems to have gone unrecognised in many infants.⁷⁹ In Copenhagen the amplitude of smallpox epidemics declined abruptly

⁷³ Nicoara et al. 1999

⁷⁴ It was not possible to estimate age pattern by single years in Stepney before this date – see above.

⁷⁵ Skold, 1996, 261

⁷⁶ Razzell, 2003,xxii

⁷⁷ Skold, 1996, 248; Fridlizius, 1984, 81

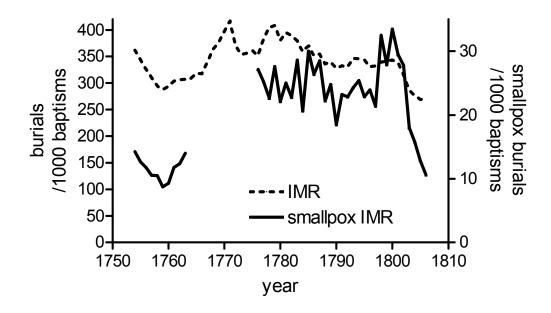
⁷⁸ Skold, 1996, 261

⁷⁹ An explanation proposed by Fridlizius (1984) to account for the apparent decline in smallpox in Sweden in this period.

around 1770, although the frequency seems to have increased (consistent with endemicisation)⁸⁰. This change coincided with the opening of a variolation establishment outside Copenhagen, but this would not explain the increased frequency of epidemics. In Sweden variolation seems to have been unpopular, and unlikely to have contributed to the change in smallpox death patterns⁸¹.

⁸⁰ Mercer, 1990, 49-50 81 Skold, 1996

a. Total and smallpox infant mortality rates



b. Smallpox percentage of all burials

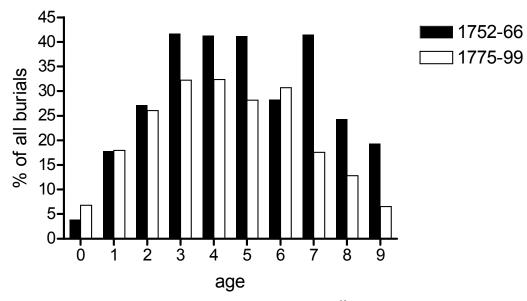


Figure 5. Smallpox in infants and children, St. Martin in the Fields. 82

⁸² Smallpox and all-cause burials were adjusted for missing age (and cause in the case of smallpox). 'IMR' refers to 'Infant Mortality Rate' or infant deaths per thousand births, calculated here as infant burials per thousand baptisms.

Period	0-5 months	6-11 months
1752-66	2.06 (2979)	11.90 (724)
1775-99	2.99 (4960)	16.68 (1983)
% change	145	140

Table 3. Percentage of all burials attributed to smallpox, by months of life, St Martin's (total burials in age group in brackets)⁸³

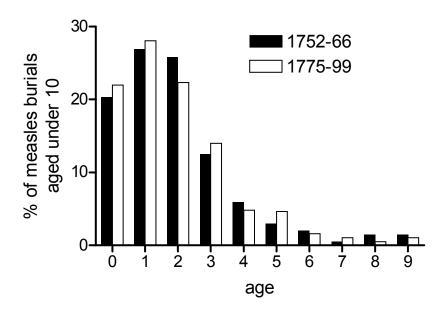
The evidence presented here, of a dramatic shift in the age distribution of smallpox burials in London from the 1770s, raises two further questions. First, what caused smallpox to begin to decline in the late eighteenth century, both in absolute terms and as a proportion of burials in the London Bills? This question highlights the problems involved in interpreting trends in mortality in the Bills, where burial totals by cause obscure changes in the age structure of burials. Since adult migrants comprised a large proportion of the London population, a decrease in their susceptibility, caused by higher rates of infection in childhood, and perhaps inoculation, would have reduced the smallpox burial totals, without any change in the rate of infant and child smallpox mortality, because susceptible adults were a distinct subpopulation, derived from immigrants to London. After the abrupt halving of adult smallpox burials in the 1770s, the adult proportion of smallpox burials seems to have declined more gradually over the last quarter of the C18th, from 10-5% in St. Martins, possibly reflecting the progress of endemicisation in London's hinterlands, and contributing to a progressive decline in the smallpox burial totals in the London Bills. In addition, smallpox was probably increasingly prone to under-recording in the later eighteenth century, as it became more concentrated at very young ages. Infants were particularly susceptible to fulminating smallpox, where the victim died before the characteristic pocks appeared. Such cases would generally have been attributed to some other cause, particularly 'convulsions'⁸⁴. Fridlizius offered a similar explanation for the apparent decline in smallpox mortality in Sweden from the 1770s⁸⁵.

⁸³ Smallpox burials were adjusted for missing cause, but not for missing age burials. The relatively small numbers of burials precluded further breakdown by age.

⁸⁴ Mercer, 1990, 63-4

⁸⁵ Fridlizius, 1984, 104-5

a. Measles



b. Smallpox

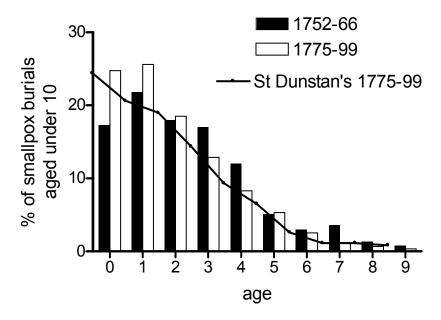


Figure 6. Age distribution of measles and smallpox at ages under ten (as a percentage of burials from each cause aged under 10). 86

⁸⁶ Measles and smallpox burials for St. Martin's were adjusted for unknown ages and causes. The St. Dunstan's burials were adjusted for unknown ages.

The second question concerns the impact of smallpox on especially infant mortality in London. Infant mortality appears to have declined in the capital from at least the 1770s 87, yet this is exactly the period in the St Martin's data when smallpox mortality amongst infants appears to have doubled. 88 While smallpox appears to have accounted for only 4-7% of infant mortality (Figure 5b), it is likely that its effects were far greater, given both the potential for under-recording (mentioned above), and synergy between smallpox and other diseases. Smallpox caused debility in many survivors and may have contributed significantly to susceptibility to other diseases⁸⁹; its importance as a contributory cause of death is suggested by the large decline in infant mortality in the early 1800s that coincided with introduction of vaccination. Assuming that the St. Martin's data are not aberrant or artifactual, the explanation for the apparent paradox, of rising smallpox mortality and declining overall mortality, probably lies partly in the different trends of the various components of infant mortality. The decline of infant mortality nationally in the second half of the eighteenth century was driven almost totally by a decline in mortality in the first few months of life, ages when smallpox was not a major contributory cause. For London, where the decline in infant mortality was dramatic, the only evidence for the relative contributions of endogenous and infectious factors derives from Landers' Quaker family reconstitutions. The Quaker data indicate a large decline in endogenous infant mortality, and a smaller decline in mortality of older infants and children (the latter in contrast to the stubborn persistence of rates at these ages in the Cambridge Group sample). It would be surprising if mortality declined at the ages of greatest smallpox risk, as smallpox mortality rose. One possibility is that the London Quakers showed greater zeal for variolation than the majority of Londoners. Infant mortality of Quakers showed some unusual characteristics, being notable for unusually low levels of endogenous mortality even at the start of the period, and it is possible that Quakers differed from the majority of the population in various practices that affected infant survival. Therefore it seems possible that infant mortality in London declined in the late C18th despite an increase in smallpox mortality, and mainly as a consequence of improvements in neonatal mortality.

(Brief) Conclusions

Our finding that adults comprised a high proportion of London smallpox burials before the 1770s is consistent with McNeill's thesis and with previous findings, and indicates

⁸⁷ Landers, 1993, 192, esp. fn 12

Notably, infant mortality in St. Martin's did not exhibit secular decline until around 1800. However Fig. 5b is misleading in this regard, because infant burials were probably undercounted in the earlier period, 1752-66, due to the export of some corpses for extraparochial burial, a practice which was not recorded before 1767 (Boulton, in prep.). This phenomenon does not affect our analysis of smallpox mortality, because the pattern of exported burials by age and cause was very similar to that of burials in the parish, when exported burials could be distinguished from 1767. Further tests for potential artifacts arising from this problem were detailed earlier in the text. Furthermore, pauper burials, which were not subject to export or import, show the same ratios of smallpox to all-cause burials, and changes in these ratios, as non-pauper burials.

⁸⁹ Razzell, 2003; Mercer, 1990, 73. This was widely thought at the time, however the standard medical work on smallpox plays this down (Dixon, 1962, 104).

that endemicisation of smallpox was not complete within London's migrant catchment areas by the mid-eighteenth century. Since this catchment area extended beyond the English population, and since adult smallpox burials did not appear to be typical of the female domestic servants that comprised the bulk of migrants in St. Martin's, it remains an open question as to where these vulnerable adults originated. The pattern of endemicisation of smallpox in England appears mysterious. Many northern settlements seem to have been almost free of adult smallpox by the mid-eighteenth century, yet a southern market town such as Burford could experience severe adult smallpox mortality in this period. However we also discovered a very abrupt shift in the age pattern of smallpox burials to younger ages in the 1770s, in both St. Martin's, Westminster, and St. Dunstan's, Stepney. The existence of this phenomenon in two large and non-contiguous parishes, and the stability of other causes and age distributions of burials, indicate that this result is not an artefact. Rather it points to a sudden change in the pattern of infection in London's migration sphere. This change coincided with the spread of variolation documented by Razzell, however variolation alone could not account for the simultaneous shift in the age pattern of smallpox in children, most of whom would have been London-born. Smallpox became concentrated at the youngest ages, and appears to have become more destructive, the infant smallpox death rate almost doubling between 1766 and 1775. These changes point to a biological explanation, that the dominant smallpox viral strain circulating in England in the late eighteenth century became more infectious. An increase in infectiousness (probably accompanied by an increase in virulence) would have accelerated the spread of smallpox into rural populations, and also amongst London's children.

If correct, these findings have substantial implications for our understanding of mortality changes in the eighteenth century. They suggest, together with Razzell's evidence of a north-south divide, a geographically uneven pattern of smallpox epidemics and susceptibility in the period before 1775. After 1775 the disappearance of young adult smallpox victims suggests that the average age of smallpox infection probably fell everywhere, contributing to further declines in mortality of young adults and older children, but increasing the smallpox risk at the youngest ages. This may account for the apparently anomalous stagnation of mortality rates at ages 6-24 months in this period, despite gains at older and younger ages in the Cambridge Group sample, because these ages experienced the greatest impact of highly endemic smallpox.

With respect to London, these data bring into question some of Lander's conclusions drawn from his London Quaker reconstitutions. While the reconstitutions have the strong advantage that they enable calculation of rates based on the population at risk (at least for infants and children), they suffer from a number of shortcomings⁹⁰, including the possibility that Quakers differed from the rest of the population in behaviours affecting smallpox risk (such as early adoption of variolation). Biometric analysis of infant mortality in St. Martin's, which allows the separation of infant deaths into endogenous

⁹⁰ Briefly, the composition of the sample seems to have changed quite dramatically over the period, with a steep decline especially in the proportion of servants; the sample size was very small, for analysis of specific causes of death; and there seem to have been biases in the recording of age and possibly cause (see footnote to Table 1).

and exogenous components, suggests that, as in the English population more generally but in contrast to the Quakers, the only source of improvement in the infant mortality rate in St. Martin's before 1800 was in the endogenous component, with no improvement in infectious disease mortality. Note however that if smallpox incidence did indeed rise in infancy and early childhood, as suggested here, then the absence of a *rise* in exogenous mortality at these ages would imply declines in other exogenous causes, sufficient to offset the impact of smallpox. Moreover, the simultaneous decline in smallpox risk of young adults and endogenous infant mortality raises the fascinating question of whether the two were related, through some effect on maternal health. Endogenous infant mortality rates in urban and rural areas converged in the late eighteenth century, despite persistent differences in environmental conditions, suggesting the common influence of some factor unrelated to living conditions.

The decline in smallpox mortality evident in the London Bills in the late eighteenth century is unlikely to represent a genuine decline in smallpox incidence or lethality. Rather it was probably the result of a rapid decline in the number of burials of adult migrants, with no improvement in smallpox mortality at younger ages. While the endemicisation of smallpox at the national level probably increased mortality (since casefatality rates were highest at youngest ages, and the more frequent occurrence of epidemics increased the risk of dying of smallpox before something else), this would not have applied in the case of London. The heterogeneous age structure of the city, with migrants concentrated in the young adult ages, meant that declines in adult migrant smallpox rates could occur without any necessary impact on the rates in the London-born population. This phenomenon indicates the danger of relying on the aggregate cause of death data in the London Bills to infer changes in disease rates, when the age structure, and the composition of the population at each age, is unknown and very different from that of the national population. The evidence from sextons' books presented here provide a critical corrective to the Bills, because they allow us to track changes in cause-specific burials at particular ages. After 1800 a further rapid drop in smallpox burials coincided with the introduction of vaccination, and it was probably at this point, but not earlier, that London-born infants and children would have begun to escape the inevitability of smallpox infection.

References

Anderson RM & May RM (1991) *Infectious Diseases of Humans: Dynamics and Control.* Oxford: Oxford University Press.

Arita JW & Wickett FF (1986) 'Impact of population density on immunisation programmes', *The Journal of Hygiene*, 96(3), 459-66.

Dixon CW (1962) Smallpox, London: Churchill

Duncan CJ, Duncan SR & Scott S (1996) 'Oscillatory dynamics of smallpox and the impact of vaccination', *Journal of Theoretical Biology*, 183, 447-54

Duncan SR, Scott S & Duncan CJ (1994) 'Smallpox epidemics in cities in Britain', *Journal of Interdisciplinary History*, 25(2), 255-71

Foege WH, Millar JD & Henderson DA (1975) 'Smallpox eradication in West and Central Africa', *Bulletin of the WHO* 52, 209-22

Fridlizius G (1984) 'The mortality decline in the first phase of the demographic transition; Swedish experiences' in Begtsson T, Fridlizius G & Olsson R (eds.) *Preindustrial Population Change: the Mortality Decline and Short-term Population Movements*, Stockholm: Almquist & Wiksell, 71-103.

Galloway PR (1985) 'Annual variations in deaths by age, deaths by cause, prices, and weather in London 1670-1830', *Population Studies* 93(3), 487-505

Gani R & Leach S (2001) Transmission potential of smallpox in contemporary populations. *Nature*, 114, 748-751

Hardy A (1983) 'Smallpox in London: factors in the decline of the disease in the nineteenth century'. Medical History, 27, 111-138

Hardy A (1993) The Epidemic Streets, Oxford: Clarendon

Hoshower LM (1994) 'Immunologic aspects of human colostrum and milk - A misinterpretation', *American Journal of Physical Anthropology*, 94(3), 421-5 Kopperman PE (2007) 'The British army in North America and the West Indies 1755-

83: a medical perspective' in Hudson GL (ed.) *British Military and Naval Medicine 1600-1830*, Amsterdam/New York.

Kunitz SJ (1983) 'Speculations on the European mortality decline' *Economic History Review* 36(3), 349-64

Landers J (1986) 'Mortality, weather and prices in London 1675-1825: a study of short-term fluctuations', *Journal of Historical Geography*, 12(4), 347-64

Landers J (1993) *Death and the Metropolis*. Cambridge: Cambridge University Press. Laxton P & Williams N (1989) 'Urbanisation and infant mortality in England: a long term perspective and review', in Nelson MC & Rogers J (eds.) *Urbanisation and the Epidemiologic Transition*, Uppsala.

Lincoln M (2007) 'The medical profession and representations of the Navy, 1750-1815', in Hudson GL (ed.) *British Military and Naval Medicine 1600-1830*, Amsterdam/New York, 225-69.

Marshall J (1832) *The Mortality of the Metropolis*, London.

McKeown T (1976) *The modern rise of population*, London: Edward Arnold McNeill WH (1980) 'Migration patterns and infection in traditional societies'. In Stanley NF & Joske RA (eds) *Changing Disease Patterns and Human Behaviour*, London: Academic Press, 28-36

Meier H (2009) Smallpox in Stuart London: Causes and Effects of an Emerging Disease, unpublished D.Phil. thesis, Oxford.

Mercer A (1990) Disease, Mortality, and Population in Transition, Leicester: Leicester University Press.

Nicoara C, Zäch K, Trachsel D, Germann D, Matter L (1999) 'Decay of passively acquired maternal antibodies against measles, mumps, and rubella viruses', *Clinical and Diagnostic Laboratory Immunology*, 6(6), 868-71

Razzell P (1977) The Conquest of Smallpox: the impact of inoculation on smallpox mortality in eighteenth century Britain, Firle: Caliban.

Razzell P (2003) *The Conquest of Smallpox: the impact of inoculation on smallpox mortality in eighteenth century Britain.* 2nd edn: Caliban Books

Schwarz LD (1982) 'Social class and social geography: the middle classes in London at the end of the eighteenth century'. *Social History*, 7, 167-85.

Schwarz L (2002) 'Hanoverian London: the making of a service town' in P. Clark and R. Gillespie (eds.) *Two Capitals: Dublin and London 1500-1840*, Proceedings of the British Academy, 93-110

Sharpe P (2000) 'Population and society 1700-1840', in Clark P (ed) *The Cambridge History of Urban Britain vol. II 1540-1840*. Cambridge: Cambridge University Press, 491-528.

Skold P (1996) 'From inoculation to vaccination: smallpox in Sweden in the eighteenth and nineteenth centuries', *Population Studies*, 50(2), 247-62

Smith R & Oeppen J (2006) 'Place and status as determinants of infant mortality in England c.1550-1837', in Garrett E, Galley C, Shelton N & Woods R (eds.) *Infant Mortality: a Continuing Social Problem*, Aldershot & Burlington: Ashgate.

Sydenham T (1742) *The Entire Works of Dr Thomas Sydenham, Newly Made English from the Originals,* London.

Woods RI & Shelton N (1997) *An Atlas of Victorian Mortality*. University of Liverpool Press, Liverpool.

Woodville W (1796) *The history of the Inoculation of the Small-pox, in Great Britain.* Walter J & Schofield R (1989) 'Famine, disease and crisis mortality in early modern society', in Walter J & Schofield R (eds.) *Famine, Disease and the Social Order in Early Modern Society*, Cambridge: Cambridge University Press, 1-74.

Thomas K (1971) *Religion and the Decline of Magic*, London: Weidenfeld-Nichols. Wrigley EA (1998) 'Explaining the long rise in marital fertility in England in the 'long' eighteenth century' *Economic History Review*, 51(3),435-64

Wrigley EA & Schofield RS (1989) *The Population History of England 1541-1871*. Cambridge: Cambridge University Press.

Wrigley EA, Davies RS, Oeppen JE & Schofield RS (1997) *English Population History from Family Reconstitution 1580-1837*. Cambridge: Cambridge University Press.