

Historical epidemiology and the structural analysis of mortality



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Abstract

Attempts to explain long-term variations in pre-transitional Western European mortality in terms of changing living standards have met with little success, and this has led to the view that such variations were biologically, or climatically determined. This conclusion can, however, be avoided by a fuller specification of the determinants of exposure to infection that incorporates the dimensions of spatial structure. This paper advances a model of the proximate determinants of exposure and resistance to infection, and derives predictions for the mortality patterns of pre-transitional metropolitan centres that are tested against data from London c1670–1830. The latter generally bear out the predictions of the model whilst also demonstrating the importance of certain features of England's political economy over this period.

Demographers' understanding of long-term and much cross-sectional mortality variation has long been dominated by the concept of 'transition' with its implication that the present and future states of high-mortality populations can be fruitfully understood in terms of the past experience of populations currently experiencing much lower levels. Much of the theorizing previously carried out in this mould may now strike us as unacceptably simplistic, unilinear, or Eurocentric, but it remains true that the range of possible relationships between demographic variables and their determinants is not unlimited and that it can be usefully elucidated by historical studies.

Thus the past can further our understanding of the present and future by defining the limits of the possible, and the contours of the probable, rather than by defining necessary evolutionary sequences, and it may indeed be the very particularity of contemporary Third World experience which emerges most clearly in the light of European demographic history. This result in itself, however, would be a healthy corrective to the view that the latter is recapitulated necessarily in the former and can thus serve as a source of policies and prescriptions: a view owing more to ahistoric social-scientific preoccupations with developmental 'stages' than to truly historical concerns with the specificity of time and place.

The secular decline in mortality, the checks and interruptions that this has suffered, and the persistence of large-scale differentials are all bound up with movements in infectious disease mortality¹ (Arriaga 1989; Horiuchi 1989). Moreover, earlier expectations that mortality transition would yield regular monotonic declines in the latter have proved unfounded, and developed-world populations may even face an increase in infectious-disease deaths in parallel with further reductions in those from non-infectious causes (Coleman and Salt 1992:246–253). If these processes are to be elucidated it will first be necessary to understand the long-term dynamics of infectious-disease mortality itself.

In this context, data from pre-transitional Europe have an essential contribution to make, offering as they do a much greater time depth than that afforded by material from contemporary high-mortality

¹ This has, of course, ceased to be the case largely where differentials within the developed world are concerned, but it remains substantially true for those within Third World countries and between high- and low-mortality populations.

populations. Nonetheless, until recently historical demography's contribution has been hampered by conceptual inadequacies in the frame of reference adopted for the analysis of pre-transitional mortality. New developments in the field, particularly the rise of the approach termed 'historical epidemiology', promise, however, to resolve many of these. In this paper I shall review briefly the theoretical problems attending earlier analyses of pre-transitional mortality change and the conceptual framework which has emerged from recent work. I shall then outline one particular model generated by the latter and put it to an empirical test.

European historical demography and epidemiology

The dynamics of infectious-disease mortality² in pre-transitional Europe provide the historical demographic context of secular mortality decline. Only in such a context can we hope to isolate mortality changes stemming in fact from 'modernization' from those which reflect the intrinsic variability and instability of 'traditional' demographic regimes, and thus to distinguish historical contingency from the necessary interrelationships governing long-term social and economic change. In spite of this, however, most studies of mortality change in the relatively recent past have placed them in a greatly foreshortened historical context. Whilst it may be acknowledged that twentieth-century patterns are the outcome of historical processes, recognition of the latter is generally confined to the changes which have occurred since the eve of the mortality transition.

In the case of England, whose historical experience has been particularly influential in this respect, mortality patterns prevailing in the nineteenth century's central decades have been taken as a benchmark for the pre-transitional mortality regime and the point of origin for the transition itself, despite their historically specific character. In fact, the early Victorian pattern differed in important respects from that characterizing earlier periods—for instance in the structure of geographical and socioeconomic mortality differentials (Kunitz 1983, 1987; Landers 1986)—and appears to have been of relatively recent origin.³

This truncation of perspective should come as no great surprise, nor as any reproach to the demographers concerned. Studies of earlier periods have been pursued generally in a separate compartment from those of the immediate pre-transitional decades, let alone the transition itself; and the two have pursued quite distinct intellectual agendas with a large measure of incommensurability. This partly reflects varying methodologies, imposed by differences in the sources available before and after 1837, but it has been underpinned by fundamental interpretative and theoretical divergences.

The last century-and-a-half of mortality change in the developed world has been the subject of many competing accounts, but there is little disagreement that such change must be explained in terms of structural characteristics, social, economic or political,⁴ of the populations in question and of the

² Strictly speaking, we cannot separate the effects of infectious from those of non-infectious causes of death in historical populations. The former, however, were certainly responsible for the great majority of deaths before the present century, and it seems safe to assume that differences in overall mortality substantially reflected variations in the infectious component.

³ The influence of McKeown has, of course, been fundamental in this respect. By rejecting the evidential status of material dating from before the establishment of Civil Registration (in 1837), McKeown was constrained to take the mid-nineteenth century as the 'before' point for his series of 'before and after' comparisons (McKeown and Record 1962; McKeown 1976). Among other things this led him, as Szreter (1988) has pointed out, to understate greatly the contribution of smallpox to long-term mortality decline.

⁴ In this context, and throughout, we use the term 'structure' to refer to enduring sets of relationships characterizing populations, their economic, social and political arrangements, as well as their cultural life, as opposed to contingent or adventitious elements that may be present at any given time. This usage is familiar to social historians, and is

global system within which they are embedded. When we move back to the 'parish register' period of European historical demography, however, the situation is very different. Attempts to explain mortality change in terms of living standards, or real wages, have been generally unsuccessful, and interpretations based on the incidence and severity of mortality crises, themselves determined partly by economic and political variables, have also proved unconvincing (Schofield 1985; Lee 1986).

Under these circumstances the dominant 'neo-classical' theory of pre-industrial population dynamics looks to fertility, and in particular to nuptiality, as the variable articulating demographic and economic structures in Western Europe.⁵ Mortality change is now seen as an 'exogenous' variable, determined not by the level of real wages, but by some other, and more elusive, factor or factors. In practice, these have often been sought outside the realm of economy and society altogether, in microbiological or climatic changes (Perrenoud 1984, 1991), and the notion of exogenous mortality has thus been assimilated to the older concept of the 'autonomous death rate'; a variable whose determinants lie altogether outside the world of human agency (Chambers 1972).

Neo-classical theory has provided a remarkably successful account of social, economic and demographic interactions in pre-industrial populations, and it has fostered closer links between historical demography and cognate areas of economic and social history, but it has had problematic consequences for the study of mortality (Landers 1986). The failure of attempts to explain mortality variations in terms of real wages, and the widespread acceptance of the autonomous death rate, have left the determinants of mortality undefined and effectively removed from empirical enquiry.

This is unfortunate in itself but doubly so if we wish to place the transition in its deeper historical context. For as we progress through the early to the central decades of the nineteenth century we move abruptly from an interpretative framework in which mortality is biologically or climatically determined to one in which it is inextricably interwoven with the structures of economy and society. These and other difficulties with the concept of autonomous death rates have led a number of recent studies to examine a broader range of factors capable of influencing levels of infectious-disease mortality. These studies, which take as their goal the development of an historical epidemiology, have analysed possible structural determinants of mortality 'beyond the real wage', and particularly the effects of spatial structure on levels of exposure to infectious agents (Dobson 1989a, b; Landers 1991).

The structural model

The relationships postulated by studies of this kind can be elucidated usefully through a set of intermediate variables, or proximate determinants, intended to depict the pathways through which the ultimate determinants of mortality variation, be they economic, social, or biological, exercise their effects.⁶ We shall start by looking at the determinants of mortality at a given time t and then consider the question of change over time.

associated particularly with the work of Braudel (1980). It is however, broader than that usually employed by demographers and social scientists since it incorporates phenomena such as systems of belief as well as social and economic structures narrowly defined.

⁵ For a definition of neo-classical population theory, in the sense in which the term is used here, and an extended discussion of the theoretical issues raised, see Landers (1993) Chapter 1.

⁶ The concept of intermediate variables, or proximate determinants, has been employed extensively in fertility analysis where it has proved possible to develop a quantifiable frame of reference (e.g., Bongaarts 1978), unlike mortality analysis where the framework remains conceptual. Our system differs from that developed for present-day mortality analysis (Mosley and Chen 1984) in being pitched at the level of population variations rather than that of individuals or households.

Mortality at time t

At any given time the level of mortality prevailing in the population will reflect the balance between exposure to infectious agents and resistance to infection on the part of members of the population in question.⁷ These two variables are influenced by a variety of structural features of the population, but the latter interact with the character of the pathogens actually present in the population at any time: the population's pathogenic load.⁸ The quality of water supply for instance is of little relevance to mortality from bubonic plague. Nutritional status will have an important effect on the mortality of populations subjected to a high level of exposure to respiratory tuberculosis, but be of much less significance if plague or smallpox are major mortality factors (Lunn 1991). Hence we must distinguish between the population's structurally-determined resistance and exposure potentials and the realized levels which arise from the interactions of these with the pathogenic load.

The exposure variables

To begin with, it is useful to think of a human population and its environment as a network in which the points are individuals and the links are constituted by pathways allowing pathogens to move between individuals, and between them and any animal, or other, reservoirs of infection that may be present. It is important at this point to note that such pathways form a matrix with an existence analytically independent of the pathogenic load. The density of this matrix determines the population's potential level of exposure to infection which is realized to a varying degree through its interaction with the pathogenic load.⁹

It is ultimately determined by a wide range of factors including personal hygiene, population density, and many of the traditional 'public health' variables such as housing conditions, and the quality and quantity of water supplies. Where the youngest age-group is concerned methods of infant feeding are also of major importance. In the absence of a suitable term to denote this proximate determinant of the level of exposure to infection I shall refer to it as the degree of conduction present.

The resistance variables

A population's potential resistance to infection can be considered as a joint function of nutritional and immunological status, whilst realized resistance again depends on the interaction of these with the pathogenic load. Nutritional status itself also reflects the interaction of diet and morbidity.¹⁰

In this context the term **diet** refers to nutrients actually consumed and thus reflects methods of food preparation and storage as well as the quantity and quality of food available to the population.

⁷ For reasons explained above we are restricting attention to the determinants of infectious-disease mortality. In principle the framework developed here could be generalized to include non-infectious causes by a widening of the concept of 'exposure' and allowing for 'negative resistance', that is, propensity to develop disease in the absence of exposure.

⁸ The experience of subgroups of the population, defined geographically, socioeconomically, or in some other way, may have differed substantially in these respects. Under these circumstances the population's epidemiological regime is said to be 'segmented' (see Landers 1993, Chapter 1).

⁹ The most striking example of this is provided by pre-Columbian America. Here the high population densities attained in some regions created a high level of conduction, and thus exposure potential, but realized exposure apparently remained very low until the coming of Europeans transformed the pathogenic load.

¹⁰ A further determinant of individual nutritional status is the energy demand imposed by the work load, but it seems unlikely that this was sufficiently variable over time, or between populations (though it may well have varied socioeconomically within populations) to constitute a significant determinant of mortality. It would, however, be easy to include this effect, as well as those of other sources of stress such as temperature, by incorporating the intermediate variable stress as a further proximate determinant of resistance potential.

Morbidity affects nutritional status, and thus resistance to infection, through the energy demands it places on the organism and the longer-term effect that certain types of infection, particularly gastrointestinal, may have on the ability of the organism to absorb nutrients from the diets. Current resistance potential is thus affected by both past and present levels of morbidity.

Immunological status depends on a combination of nutritional status and the past history of exposure to infectious agents, whether by 'natural' infection or by artificial immunization. For the youngest age-group it is also affected by the methods of infant feeding employed.

Change over time

The proximate determination of mortality at a given time t is evidently highly complex. A number of the proximate determinants interact with each other, or appear more than once, and the role of morbidity means that the state of the system at t is not independent of its state at earlier points. Fortunately, these complexities are simplified when we consider change over time, for a number of the variables are endogenously determined and so drop out of the analysis.

Resistance variables

Of the resistance variables, the level of morbidity is, as we have seen, set internally to the system. This is also true of changes in immunological status which, in the absence of migration,¹¹ are determined by variations in pathogenic load and in nutritional status. Nutritional status reflects the interaction of diet and morbidity, and since morbidity is also endogenously determined only diet remains as the principal proximate determinant of changes on the resistance side of the balance.

Exposure variables

Realized exposure at a given moment depends on conduction and pathogenic load. Changes in conduction will thus affect levels of exposure and can occur through variations in any of the social, economic or ecological variables to which this variable refers.

Pathogenic load

Pathogenic load, as we have seen, affects the realized levels of both exposure and resistance to infection. The determinants of changes in this variable are more complex than those considered so far and reflect the interaction of two distinct sets of factors. It is evident that the pathogenic load will vary according to the ability of pathogens to remain in the population and the introduction of new pathogens. The first of these is determined by a variety of factors including population size and density, and the existence of animal or other reservoirs of infection, and it can be defined as a further proximate determinant of mortality which I shall term the degree of retention.

The introduction of new pathogens can occur either by their physical entry from the outside world—by means of trade, migration, warfare, or other forms of movement—or biologically, through genetic or chromosomal alterations to the existing stock. The latter represent truly 'autonomous' phenomena, but the former depend in part on the degree of physical movement between the population and the outside world. This depends on a variety of geographical, economic, and political variables and can be referred to as the population's degree of bounding.

¹¹ Migration will affect immunological status, at the population level, if the population from which immigrants are drawn differs in this respect from the host population in question. This appears to have been the case where early modern European cities are concerned.

Epidemiological regimes

Change over time thus reflects the effects of variations in resistance and exposure potentials, governed by the four proximate determinants diet, conduction, bounding, and retention. Changes in realized resistance and exposure depend on these but also on two factors outside the system which cause truly 'exogenous' variations in pathogenic load. The first of these includes biological events such as gene mutations, whilst the second refers to the character of the pathogenic load in the 'outside world' populations with which the regime is in contact.

Epidemiological regimes¹² can thus be characterized in terms of varying levels of the proximate determinants of mortality. Of the former, particular interest attaches to those characterizing the populations of large metropolitan centres in the pre-transitional era.¹³ Here high population densities combined with poor housing, sanitation, and water supplies to produce high levels of conduction and retention, whilst at the same time the nodal position of such centres in networks of trade and migration reduced the degree of bounding to a low level. The exposure potential of such regimes was thus high, and they appear to have functioned as endemic reservoirs of infection.

Mortality was correspondingly severe, but individuals who survived for any length of time acquired a degree of immunological resistance to a range of pathogens. Hence at any given time the level of immunological resistance characterizing such populations was likely to be substantially higher than that found in more thinly settled 'hinterland' regions where the degree of retention was much lower. The high exposure potential of such regimes was thus partly offset by a high resistance potential, and in many instances the latter was further enhanced by the economic and political centrality enjoyed by metropolitan centres which provided a food base that was more secure, and possibly also more abundant, than those of smaller settlements.

This configuration of proximate determinants can thus be described as a 'high potential' epidemiological regime. According to our model such a regime should manifest itself in a characteristic mortality pattern. Mortality would be severe, concentrated among children and recent immigrants who lacked previous exposure to urban infections, but levels should be relatively stable from year to year since the scope for epidemic outbreaks was limited by the high level of endemic infection, and major subsistence crises would be rare. In the remainder of this paper we shall see how far these predictions are borne out in a particular critical instance.

The case of London

The experience of London over the 'long eighteenth century' provides an important test case for the predictions of the high potential model of metropolitan epidemiological regimes. In 1700 London numbered around half a million inhabitants, some ten per cent of England's population at large and more than half of its urban population (Wrigley 1967). More than ten times the size of the largest provincial centre, London was an indisputable 'primate city'. The demands of the London market had played a crucial role in integrating the national economy over the previous century or more. It was

¹² The concept of the epidemiological regime is defined by analogy with that of the vital, or demographic, regime. It refers to the persisting structural relationships which govern levels of both morbidity and mortality in a population over time, although in practice the former are generally hidden from observation.

¹³ This is primarily because of the substantial burial surpluses characterizing such centres, which have long been a focus of attention among economic and demographic historians (Sharlin 1978; DeVries 1984; Landers 1987). The particular model outlined here is based on suggestions advanced by McNeill (1980) and is considered in greater detail in Landers (1990).

England's judicial and administrative centre and the hub of a nationwide system of long-range migration.¹⁴

In the course of the eighteenth century, England's urban hierarchy became more sophisticated with the development of a tier of large provincial cities and a range of new functional urban types (De Vries 1984). London's population in 1800 was, relative to that of England, no larger than it had been a hundred years earlier, but in absolute terms it had nearly doubled. What makes this growth the more remarkable is that it was accomplished in the face of an excess of recorded burials over baptisms every year throughout the century until the 1790s. Under these circumstances the city's continued existence, let alone its expansion, depended on a continuing stream of immigrants from the countryside and smaller urban settlements.

The study of London's demographic history over this period is greatly assisted by the existence of a remarkable source of aggregated vital data. These are the London *Bills of Mortality*, sets of burial totals broken down by cause of death, the parish in which they occurred and, from 1728, by age. This information was published every week, together with a global baptism total, and a consolidated annual Bill was issued at the end of each year.¹⁵ The Bills suffer from problems of under-registration, particularly where baptisms are concerned, and caution is required in their use, but for all their shortcomings they represent a source of demographic data on a scale unparalleled elsewhere before the beginning of the statistical era.

Tables 1 and 2 give the breakdown of the global burial totals by cause of death and by age for a series of subperiods. The cause-of-death terminology presents obvious difficulties, and only in the case of smallpox can we make an unequivocal identification with a category known to twentieth-century medicine. The category of 'fevers' was retained in the early decades of civil registration, after 1837, before being split into typhus and typhoid. It is likely that these two causes, particularly the former, predominated among fever deaths in the eighteenth century although many other infections were doubtless represented in addition. 'Diseases incidental to infancy' (henceforth simply 'infancy') groups together a number of causes of death and the total is roughly equivalent to that for burials under the age of two years.

Table 1
Contribution(%) of causes of death to total burials

Cause of death	1675-99	1700-24	1725-49	1750-74	1775-99	1800-24
Consumption	16.7	12.6	15.7	19.5	24.5	23.2
Fevers	15.2	14.9	15.3	13.9	11.3	6.9
Infancy	29.9	39.1	37.6	35.0	32.2	29.2
Smallpox	6.4	7.3	7.8	10.0	9.3	5.4
Other causes	31.8	26.1	23.6	21.5	22.7	35.3

Table 2
Age distribution of total burials

¹⁴ See Landers (1993), Chapter 2, for a general review of the economic and social character of London over this period and its relationship to that of England as a whole.

¹⁵ Marshall (1831) provides data from the annual Bills in a conveniently tabulated form and was used as the source for the analyses which follow.

Age group	1728–49	1750–74	1775–99	1800–24
0–1	36.3	33.6	33.2	28.4
2–4	8.6	9.1	10.1	10.6
5–9	3.6	3.6	3.8	4.2
10–19	3.1	3.4	3.5	3.3
20–29	7.8	8.0	7.2	6.9
30–39	9.5	9.3	9.0	9.4
40–49	9.8	9.7	9.9	10.3
50–59	7.9	7.9	8.5	9.2
60–69	6.2	8.1	7.2	8.1
70–	7.2	7.3	7.6	9.6

It is probable that most deaths from respiratory tuberculosis appear under the heading of ‘consumption’, but the latter term seems to have been used as a ‘catch-all’ for a variety of emaciating conditions and cannot be equated with this disease alone (Hardy 1988a). Table 1 shows the importance of infancy deaths and the two most ill-defined categories, consumption and ‘all others’ in the overall burial totals, but where change over time is concerned it is fever and smallpox, together with infancy, which contribute most to the decline in numbers at the end of the period.

Mortality levels and their stability

The age-specific burial totals underline the scale of mortality in the youngest age-groups and the eventual reduction in infant mortality. The distribution of burial totals by age is sufficient to give us an impression of the corresponding mortality risks, but for a more rigorous investigation we require a set of age-specific mortality probabilities (the life-table function q_x). These cannot be obtained directly from the material in the London Bills, since the latter contain no information on the underlying population at risk, but they can be calculated from nominal vital registers using the technique of family reconstitution. The rates in Table 3 were obtained from the registers of two of London’s six Quaker Monthly Meetings,¹⁶ and a comparison between these and similar figures obtained from reconstitution studies of parish registers elsewhere in England reveals the extent of London’s excess mortality in infancy and childhood.¹⁷

Table 3
Age-specific mortality rates (q_x) per thousand in 13 English parishes and two London Quaker Meetings

Cohort	Age-group	English parishes	London Quakers
1650–99	0	170	260
	1–4	101	244
	5–9	40	67

¹⁶ See Vann and Eversley (1992) for a general discussion of the registration system together with the Quakers’ social, economic and demographic characteristics. Data on the composition and location of the two meetings used in this analysis are provided in Landers (1991).

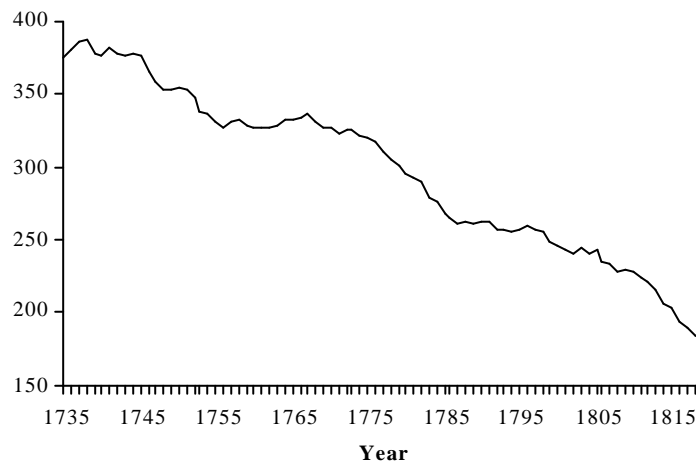
¹⁷ It is unfortunate that the methodology of family reconstitution does not lend itself at all well to the measurement of adult mortality (Wrigley 1968; Wrigley and Schofield 1983), and relatively little is known about its level in pre-industrial England. The limited information available, however, suggests that the differential between London and other English communities was much smaller than was the case with infant and child mortality (Landers 1990).

	1-9	137	295
	0-9	284	478
1700-49	0	195	342
	1-4	107	298
	5-9	41	95
	1-9	143	365
1750-99	0-9	310	582
	0	165	276
	1-4	103	253
	5-9	33	57
	1-9	133	296
	0-9	277	490

Source: Wrigley and Schofield (1983) and Reconstitution Tabulations.

As we have seen, the Bills of Mortality do not lend themselves easily to the analysis of age-specific mortality levels, but from 1728 at least, it is possible to construct a series of infant-mortality estimates on the basis of assumption about the under-registration of baptisms relative to burials and the distribution of ages at burial in the youngest of the Bills' age-groups (0-1 year).¹⁸ The results in Figure 1 are reassuringly close to those obtained from the Quaker records and suggest that, before the late eighteenth-century decline in mortality, over a third of London-born infants died before their first birthday.

Figure 1
Infant mortality rate in London Bills (11 point moving mean)



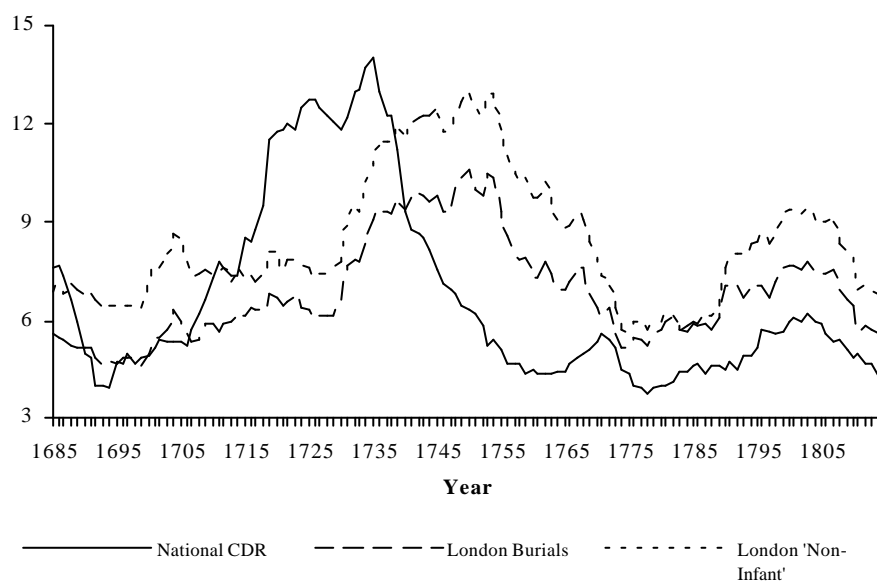
Metropolitan mortality levels, according to the high potential model, not only should be higher than those of hinterland populations, they should also display a greater short-run stability. This expectation can be tested quite easily using the Bills' burial series. The first step in the procedure is to remove the effects of medium- and long-term trends from the series. This 'de-trending' can be done in a number of ways, and I have employed the relatively simple method of dividing each annual burial

¹⁸ The major problem involved in this process is to estimate the shortfall in the registration of baptisms relative to that of burials. In order to do this a set of correction factors was constructed based on those used by Wrigley and Schofield (1981). For details see Landers (1993) Chapter 5.

total by the mean figure for the adjacent quinquennia and multiplying the result by a hundred. The resulting 'crisis mortality ratio' (CMR) thus measures the departure of each year's crude death rate from its short-run average, and by taking the mean absolute deviation (MAD) of the CMR series from its mean we have a convenient measure of the short-run stability of mortality.

Figure 2 plots 21-year moving averages for MADs calculated from the London Bills series (both including and excluding infancy deaths) together with a comparable series constructed from Wrigley and Schofield's estimates of the national crude death rate over this period (Wrigley and Schofield 1981). Other things being equal, the short-run stability of a mortality series should rise with the size of the population concerned, whilst for technical reasons CMRs constructed from crude death rates (CDR) should be less volatile than those derived from the corresponding burial totals being unaffected by short-run movements in population size. It is thus noteworthy that the MADs for the London burial series are actually lower than the national CDR for much of the high-mortality period and at comparable levels throughout.

Figure 2
Short-run instability of mortality



An examination of extreme CMR values leads to similar conclusions. In Figure 3a I have plotted values calculated from the national and London series, together with a further set based on the Bills of Mortality but excluding infancy deaths, for the years in which the national series registers its 15 highest values. In Figure 3b I have done the same for the years which fall into the 'top 15' in either of our London series without doing so nationally. The designation of 'mortality crises' has an inevitably arbitrary character in situations of this kind, but Flinn (1974) has suggested a CMR of 130 as the appropriate criterion when working on a regional scale. If we adopt this, for purposes of demonstration, we find that there were no 'crises' in London at any time during our period, using either of our two

series, although the threshold is passed once in the national series. The level of 120 is reached on only six occasions in the 'non-infant' series, and three times in the total burials. The national series also contains six values above this level but passes the 40 per cent mark in the crisis year of 1729.

Figure 3a
Crisis mortality ratios extreme values: national criterion

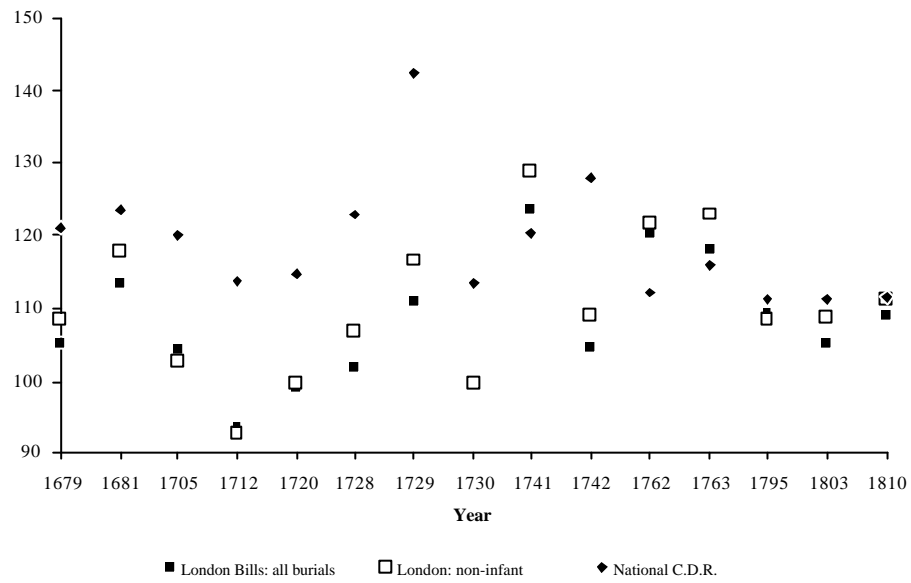
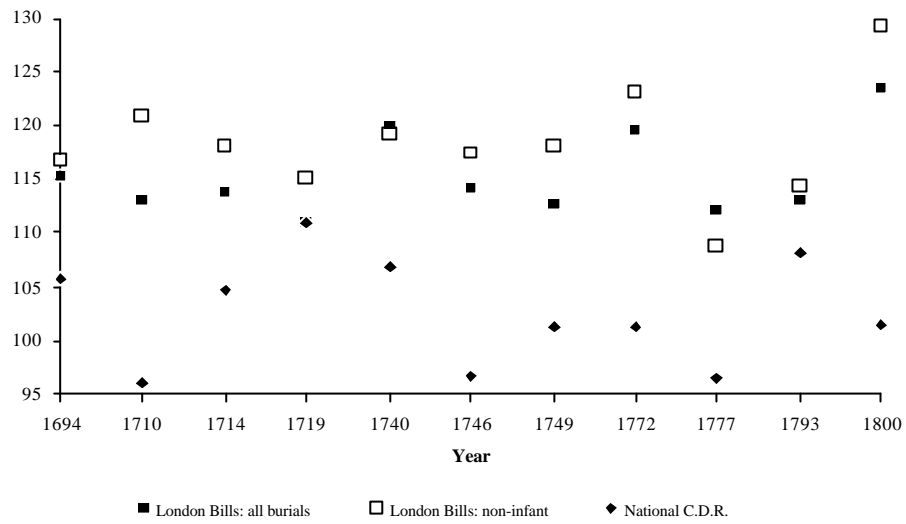


Figure 3b
Extreme values: London criterion

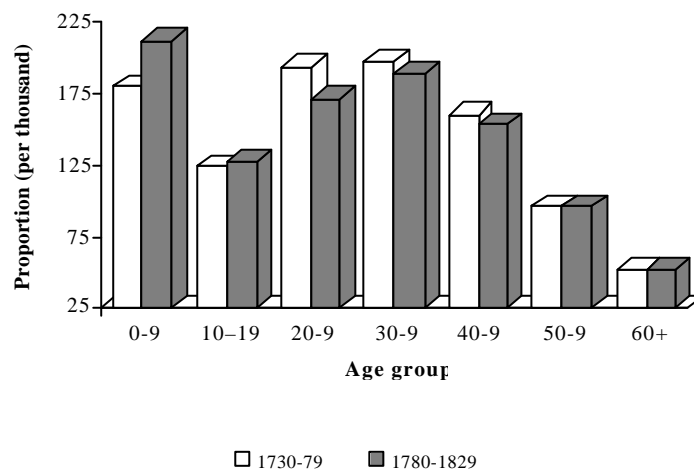


It is interesting to note that London's 'advantage' appears to have been most marked in the earlier part of the period when absolute levels of mortality were most severe. Of the 15 worst years in the national series, eight occur before 1740, but only in 1681 and 1729 does the London CMR pass the 110 mark. Similarly, only four of the years excluded on the national criterion, for this part of our period, feature among the 15 worst years in London. Furthermore, in two of these cases, 1710 and 1719, the divergence is probably less marked than appears at first sight, since both prefigure major upswings in the national series and the 1719 CMR for the latter is, in any case, of comparable magnitude to the London non-infant figure. The position after the mortality wave of the early 1740s is, however, very different. The London CMRs are now close to, or above, the national figure in each of the remaining five national crises, and six major upswings which are included on the national criterion.

Migration

The scale of London's burial surplus combined with the city's secular growth implies a substantial net inflow of migrants. Although the data do not allow us to estimate the absolute numbers involved to any useful degree of precision, it is possible to obtain a rough approximation of the age-structure of net immigration using the age-specific burial series. In order to do this we must first obtain a set of age-specific death rates (life table m_x) which are then divided into the burial totals so as to obtain estimates of the underlying population size. Death rates of this kind cannot be obtained directly from the Bills, but they can be estimated indirectly using model life tables together with the infant mortality estimates obtained earlier.¹⁹

Figure 4
Estimated age-structure for London



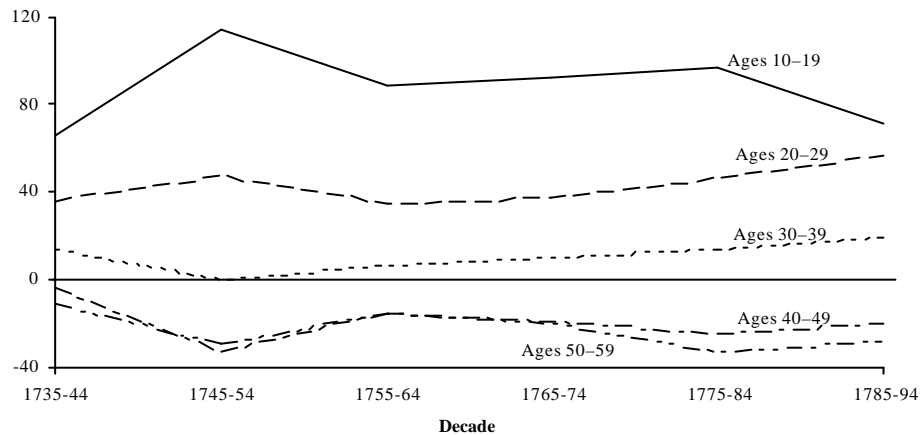
Applying these estimates to the burial totals, decade by decade, yields the results in Figure 4 in which the data for the decades of high mortality and mortality decline have each been pooled. The relative predominance of young adults in London's population emerges clearly from Figure 4 as does

¹⁹ The method used was based on the Brass two-parameter model life-table system (Brass 1971) and is discussed in detail in Landers (1993), Chapter 5.

the increase in the proportion of children as mortality declined. The uncertainties and approximations of the method are such that the detail of the results will not bear too much scrutiny, but these two general observations are both fairly robust and follow from the very distinctive age profile of the burial totals.

The age-structure of net migration can now be estimated by comparing the population total for each ten-year age-group with that for the next youngest age-group in the preceding decade.²⁰ The results in Figure 5 demonstrate the importance of adolescents and young adults in net immigration whilst suggesting an overall outflow of older adults. Paradoxically these figures are probably more reliable than are the decadal age-structure estimates from which they were derived because any systematic errors in the latter will be, at least in part, self-cancelling when the results for successive decades are compared.²¹

Figure 5
Net immigration by age (as percentage of total net immigration)



The high potential model predicts that, as a group, recent immigrants should show different immunological and epidemiological characteristics from native-borne adults, reflecting their limited exposure to urban infections. The Bills themselves do not provide a double-classification of deaths by age and cause, and so we cannot approach the problem directly using this material. The Quaker registers, however, use the same cause of death and age labels as the Bills and, since they identify individuals, allow us to construct a classification of this kind. The results in Figure 6 are based on an analysis of this kind carried out on smallpox burials. They reveal that a substantial minority of all

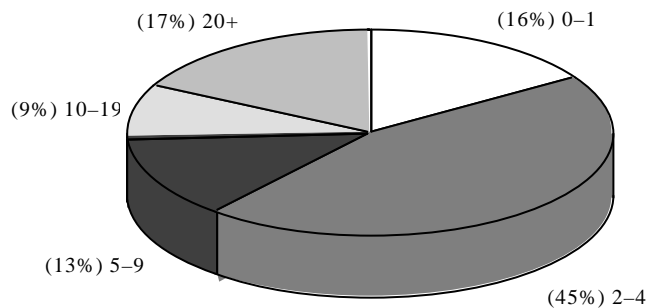
²⁰ Population estimates by age were calculated for each decade and were assumed to be valid for the midpoint of that decade. Net migration totals were then calculated by comparing the inter-decadal changes with the corrected age-specific burial totals for the intervening period.

²¹ The most likely source of error in the age-structure estimates is an underestimate of mortality in early adulthood relative to childhood. This is because the model system, being based on more recent experience, does not take account of what are believed to have been the peculiar risks experienced by immigrants in this age-group to early modern cities. If this is so, the corresponding population estimates should be somewhat too high before the onset of secular mortality decline, but the discrepancy should be consistent from decade to decade.

deaths occurred among young adults and adolescents, despite the fact that smallpox was apparently a universal disease of childhood in London at this time.

The implication of this finding is that a significant proportion of the immigrants to London at this time had not been previously exposed to smallpox and were thus immunologically unprotected, as were the London-born children. We can pursue this question indirectly, with the aid of the material from the London Bills, by examining the relationship between movements in the age-specific CMR series. Figure 7 displays the principal components of the inter-correlation matrix for the latter.²²

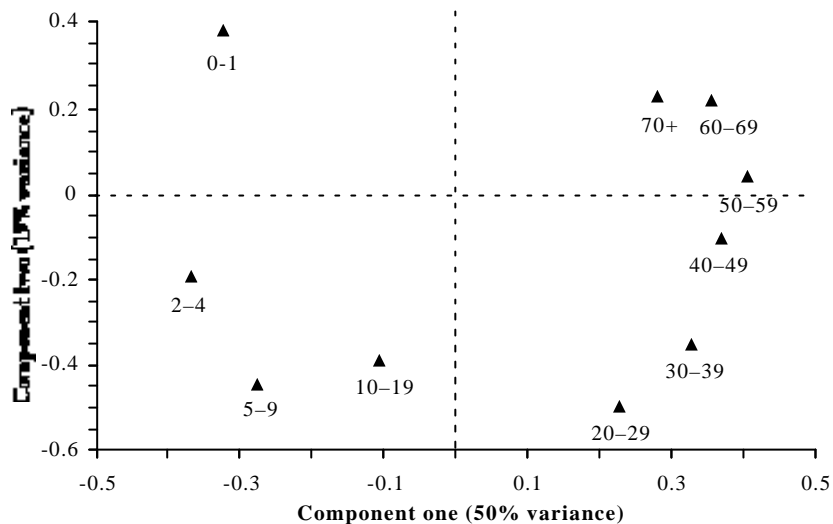
Figure 6
Age distribution of smallpox burials in two London Quaker Meetings 1700–49



The first of these accounts for half of the total variance and divides the age-groups fairly clearly into two clusters corresponding to the adult and childhood-teenage years respectively. The second component, however, produces a different grouping, distinguishing the age-groups 5-39 years from those aged 0-1 and 60+, with the 2-4 and 40-59 year olds having intermediate values. One interpretation of this finding is that the second component is picking out a pattern of disease susceptibilities common to children and recent immigrants, but distinct from those imposed by the physical frailties characterizing the oldest and youngest age-groups.

²² In order to remove the effects of shifts in the overall level of mortality from this analysis the raw age-specific annual burial figures were divided by the global burial total for that year prior to the calculation of CMRs.

Figure 7
Short-run movements in burials by age 1733–1824: principal components analysis



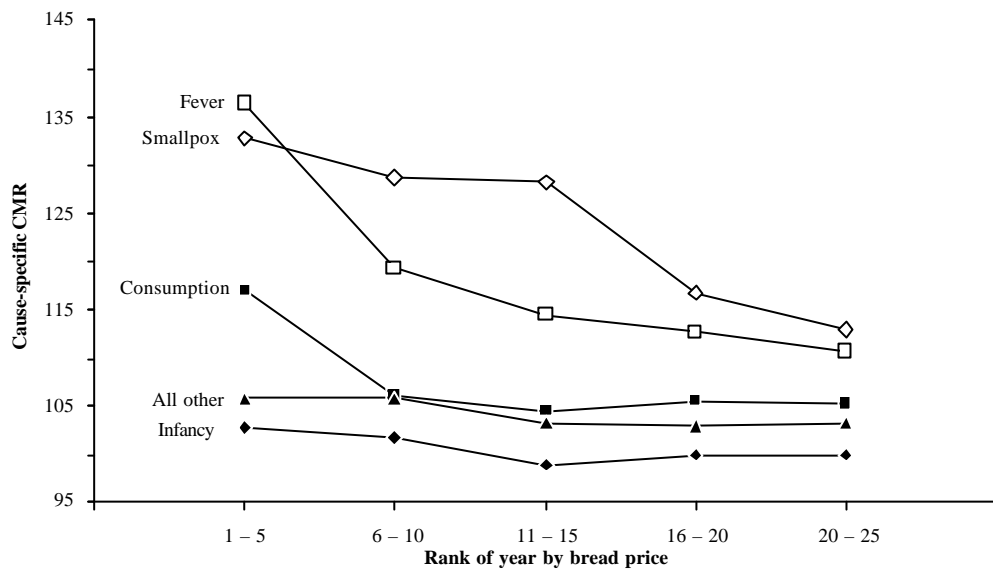
Mortality fluctuations

We have already seen that short-term mortality fluctuations in London were of relatively modest amplitude and that there appear to have been no real ‘mortality crises’ at any time during our period. There were, however, a number of marked upswings in mortality, and it is of some interest to examine the circumstances surrounding these. Orthodox ‘crisis theory’ distinguishes three factors capable of producing disturbances of this kind: price rises linked to crises of subsistence, outbreaks of epidemic disease, and war or civil unrest. We shall begin by examining the effects of movements in the price of bread on those in the burial series.

There are a number of ways in which this can be done, including some sophisticated econometric techniques, but I have adopted a relatively simple method. First the detrended annual burial totals, and the average annual price for a quartern loaf, were ranked in order of magnitude. The years having the top 1–5, 6–10, 11–15 etc. bread prices were then identified and the mean detrended burial total calculated in each case. Figure 8 displays the results of this analysis for the cause-specific burial series. These suggest that mortality in general was little affected by high bread prices but that these had a major impact on mortality from smallpox and, to a lesser extent, from fever.

One shortcoming of this analysis is that it can only detect effects which occur in the same year as the price rise itself, whereas it is likely that much of the impact will be ‘lagged’ by one or more years. An elementary means of examining this possibility is to repeat the analysis taking the detrended burial total for the year following the price rise should this exceed the value for the year in question. The results in Figure 9 reveal that this exercise strengthens the relationship substantially in the case of the fever and smallpox burial series, whilst having little effect on the others.

Figure 8
The effect of high bread prices on mortality in the London Bills



This finding is at first sight surprising, since the most straightforward interpretation of such associations is nutritional. On this basis higher prices would lead to a reduction in the quantity and quality of food intake, and in resistance to infection, on the part of the members of the population most affected. Smallpox, however, was an infection whose outcome was very little affected by the nutritional status of the victim, and nutritional status also seems to have been of secondary importance at most in the case of typhus, the disease likely to have been responsible for the bulk of the excess fever deaths.

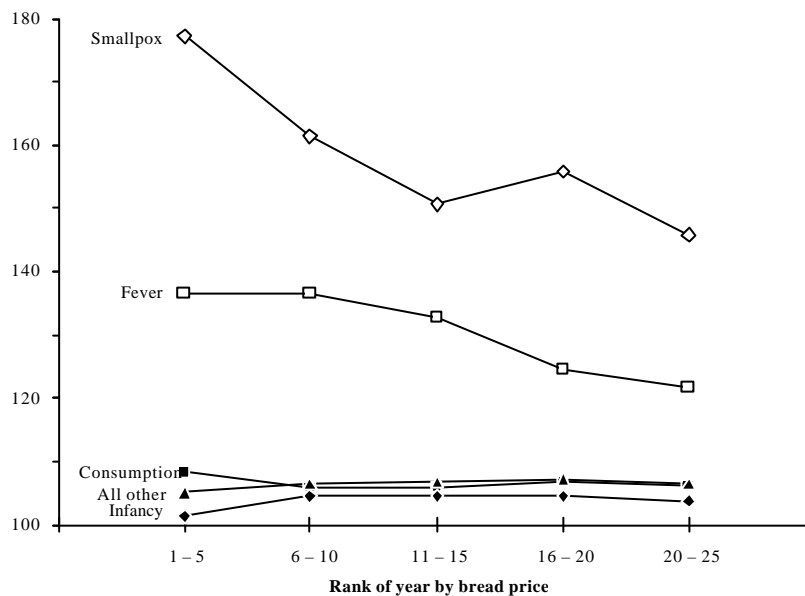
In this case an explanation must be sought in the variables governing exposure to infection; in particular, bounding and conduction. It is likely that high food prices in London generally coincided with periods of economic stress at least as severe in the country at large and that migration to London was one response to such circumstances. Many such migrants would, as we have seen, lack resistance to smallpox and in all probability to other urban diseases as well. Once in the capital they congregated in the suburban districts with their filthy and overcrowded lodging houses, further exacerbating an already chronic shortage of accommodation.

Under these circumstances the overall immunological resistance of the population was reduced by compositional change. Many new migrants were wretchedly housed, and their swelling numbers meant that the existing population experienced increased competition for the inadequate supply of accommodation, a factor of particular importance where economic disruption reduced employment opportunities in the capital for migrant and native alike.²³ It is this combination of increased movement

²³ For an analysis of the relationship between price shocks, social dislocation and increased mortality in pre-industrial Europe see Post (1985). Hardy (1988b) has demonstrated an analogous relationship between economic dislocation and increased housing densities, giving rise to epidemic disease, in Victorian London.

and effective population density under conditions of indifferent hygiene which can best explain the rise in smallpox and fever mortality.

Figure 9
The effect of bread prices on mortality (including one year lags)



The effect of this broader social and economic disruption can be seen in the mortality waves accompanying the transition from war to peace over this period. England's seventeenth century experience, under republic and Stuart monarchy alike, had endowed its political class with a hostility both to standing armies, and to the fiscal apparatus required to maintain them; so the cessation of hostilities was followed by the demobilization of tens, and later hundreds, of thousands of servicemen with no effective measures taken to reintegrate them into peacetime society. Many ended up on the roads of England, most of which led to London.

Postwar London was, however, ill-prepared to receive such an influx. Its labour market usually suffered badly from the transition between war- and peace-time economies, whilst the housing stock was adversely affected by wartime depression in the construction sector. The effects can be seen in Table 4 which sets out the detrended burial totals for fever, smallpox, and all causes, together with a comparable figure for bread prices and absolute numbers for the size of the armed forces. Only at the very beginning and end of the period, in 1698 and again in 1816, does the coming of peace coincide with a major price rise, but a mortality wave is apparent on each occasion, the only exception being the close of the American war. In the early part of the period it is apparently smallpox deaths which are the most sensitive to these conditions. This ceases to be the case from the last quarter of the eighteenth century, but fever deaths remain as responsive as ever down to the close of the Napoleonic wars. Fewer deaths, however, now constitute such a small proportion of the overall burial totals that these are largely unaffected.

Table 4
The consequences of peace

Year	Bread price	Burials (as % of trend)			Armed forces (thousands)
		Fever	Smallpox	Total	
1697	116	94	60	103	87
1698	124	102	209	99	36
1699	110	113	90	101	13
1712	97	90	108	93	153
1713	98	87	85	91	46
1714	100	135	145	114	29
1748	102	116	96	103	105
1749	102	135	145	113	75
1750	99	128	62	103	39
1762	87	122	115	120	151
1763	90	106	157	118	136
1764	103	121	97	102	48
1783	112	94	89	97	234
1784	110	80	99	90	57
1785	94	95	119	97	48
1802	87	120	115	104	274
1803	76	130	82	105	194
1815	76	126	85	102	326
1816	135	125	83	108	257
1817	108	123	143	105	203

Sources: For armed forces figures: Ashton (1959); Floud et al. (1990).
For bread prices: Mitchell and Deane (1962).

The seasonality of mortality

According to the high potential model, metropolitan centres function as endemic reservoirs of infection giving rise to intermittent epidemics in their more thinly settled hinterlands. Underlying this is the assumption that much of the excess mortality characterizing such centres reflected the action of airborne, or other, infections transmitted directly from person to person.²⁴ Such an assumption appears plausible given the high population densities they displayed, but it runs counter to the widespread attribution of such excesses to water- and food-borne pathogens which might not be so readily transmissible beyond the city itself.

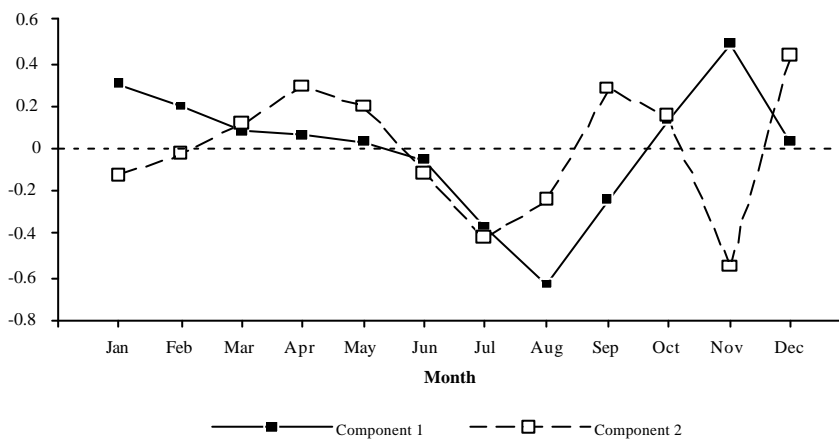
The resolution of this question is hampered by the weakness of the cause-of-death classification contained in the Bills, but it can be approached by moving from the annual to the monthly level and analysing the seasonal incidence of mortality in London. The rationale of this procedure is that the seasonality of mortality tends to differ according to the mode of transmission of the pathogen concerned. Thus data from Victorian London show a strong summer peak for dysentery, diarrhoea, and

²⁴ For this purpose typhus, which is actually arthropod-borne, can be treated as a person-to-person infection since close physical proximity is usually required in order to transmit the disease.

non-respiratory tuberculosis mortality, with a corresponding winter peak for typhus, respiratory tuberculosis, and bronchitis. A third group of conditions, including diphtheria and scarlet fever as well as typhoid, were distinguished by a marked autumn seasonality.²⁵

A reconstruction of burial seasonality in London over our period, however, yielded unexpected results, for the overall pattern proves to have altered substantially in the course of the eighteenth century (see Table 3).²⁶ There appear to be two main changes, of which the first, accomplished between the late seventeenth and mid-eighteenth centuries, is the more far-reaching. This transforms an initial summer peak, strongly centred on August, into a much broader 'cold-weather plateau' stretching from November to April; the months of June and July now representing a trough in mortality, with only a moderate excess remaining in September. In the second phase, from 1775 to the end of the period, a markedly bimodal pattern emerges. The cold-weather plateau is much reduced, becoming clearly focused on the months of January and February, whilst a new burial peak emerges in November.

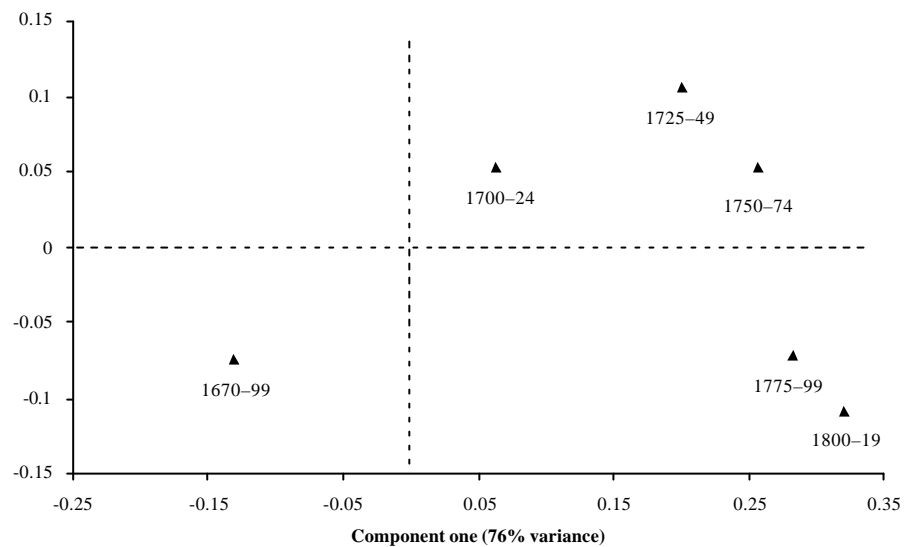
Figure 10a
Bills of mortality: burial seasonality principal component coefficients



²⁵ The Victorian data are taken from Buchan and Mitchell (1875); see Landers and Mouzas (1988) for details of the methodology employed in the analysis of the Bills' seasonality, and the technical and conceptual problems encountered.

²⁶ The monthly burial indices given in the table express the observed burial total for each month as a percentage of what would be expected if the risk of mortality were to be distributed evenly across the year.

Table 10b
Principal component scores



These movements in seasonality were summarized in the form of a principal components analysis (Everitt and Dunn 1983), as in Figures 10a and 10b in which 97 per cent of the variation between the indices for the six periods was accounted for by the first two components. The plot of scores on the latter suggests that the first should be thought of as an axis of secular change: a change continuing throughout the period, though at a substantially reduced rate after 1750. Inspection of the coefficients reveals, as we might expect, a pattern of strong negative associations with the indices for the months in the third quarter of the year, especially August.

The second axis, by contrast, separates the 1700-74 periods from the remainder. The coefficients here are harder to interpret, but the combination of relatively strong positive values in December and April, with weak or negative ones for the intervening months, is consistent with the prevalence of the cold-weather plateau of mortality at this time. The first three-quarters of the eighteenth century thus stand out with a distinctive seasonal pattern of burials, over and above the longer-term movement from a summer to a winter peak of mortality.

The decline in the initial summer peak of mortality is apparently associated with a decline in deaths from food-borne gastro-intestinal conditions (particularly that known to contemporaries as 'gripping in the guts'), and it is most likely that this was due either to changes in the relevant pathogen or, conceivably, to the climatic change which occurred at this time.²⁷ The development of the 'cold-weather plateau', however, seems to have reflected an increase in the relative weight of deaths from diseases such as typhus and the bronchitis-influenza-pneumonia group in the global burial total.

²⁷ This assertion is based partly on negative evidence: the apparent absence of any significant improvement in sanitary conditions at this time; and partly on the occurrence of a similar decline in summer deaths in neighbouring rural districts (Greatorex n.d.)

The trend in overall mortality in London between the later seventeenth and early eighteenth century is difficult to establish with any certainty, but it seems very unlikely that it was declining at this time, implying that there was an absolute as well a relative increase in mortality from these causes of death. The pattern is further complicated by a rise in mortality from a further cause of death, or group of causes, with a marked autumn seasonality in the central decades of the eighteenth century, a phenomenon most marked in the case of the fever burial series. Outside London, however, there appears to have been a definite amelioration of mortality in south-eastern England in the early eighteenth century.

We can pursue this further with the aid of an alternative source of vital data: the parish registers. The advantage of these is that, in many cases, it is possible to distinguish the burials of adults from those of children and thus to perform an elementary analysis of age-specific patterns of burial seasonality. Against this, however, must be set the much more time-consuming nature of the preliminary data collection involved. Hence the analysis was limited to two decades, 1695-1704 and 1750-59, for which the available parish registers were examined and those distinguishing child burials included in the main sample.

If the assumptions of the high potential model are correct we should expect to find that most variation in burial seasonality occurs in the childhood age-groups, since it is here that individuals first encounter the pathogens present in the environment and either succumb or develop a degree of immunity. The results in Figures 11 and 12 generally bear out this expectation. Some change occurs in both adult and child burial seasonality, but the latter is far more pronounced suggesting that it is indeed in childhood that the major changes in cause-of-death structure are apparent.

Figure 11
Burial seasonality indices 1695-1704

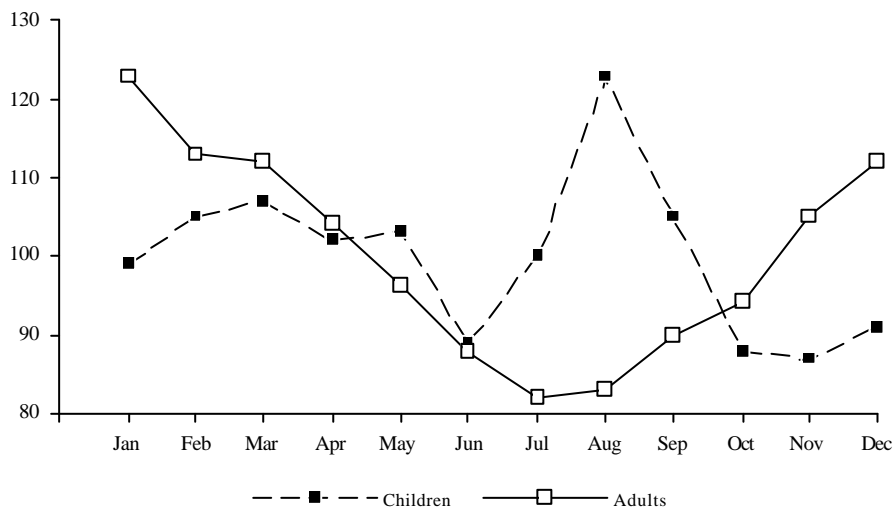
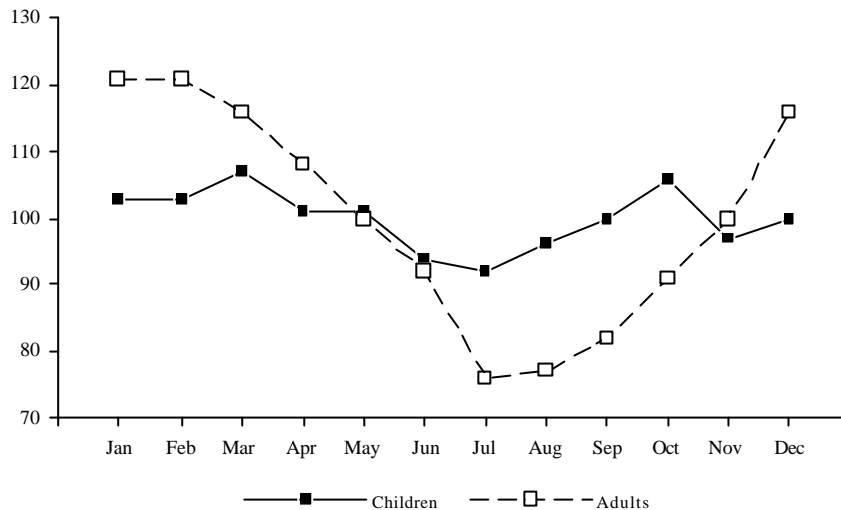


Figure 12
Burial seasonality indices 1750-59



Mortality change and its context

We can also use the parish register sample to investigate spatial variations in burial seasonality. Figures 13 and 14 depict the seasonal pattern of child burials for four groups of parishes in each of the two decades. There are some differences between the groups for the decade 1695-1704, but this is primarily a matter of quantitative variation in a common underlying pattern. In 1750-59, however, the position is transformed. The summer peak has evidently disappeared throughout London, but the new autumn excess is very unevenly distributed, being wholly absent from the wealthier western districts.²⁸

This emerging spatial differentiation in London's epidemiological regime seems to point to a factor underlying the latter's evolution over the course of our period. This was the problem of housing conditions. The peculiar forms of housing tenure and finance in the capital at this time gave rise to a pattern of house building which was highly speculative and characterized by a dramatic alternation of 'booms' and 'busts'.²⁹ Under favourable conditions the system was capable of turning out large numbers of houses in a short time, but the quality was often very poor and any deterioration in the economic climate, due to the outbreak of war or one of the period's recurrent financial crises, stopped activity dead in its tracks with a wave of bankruptcies and a crop of abandoned half-built houses (Ashton 1959; Hoppitt 1987).

Figure 13
Child burial seasonality indices, 1695-1704

²⁸ The evidence of baptism burial ratios suggests that child mortality was also substantially higher in districts with the autumn burial peak, and that spatial differences in mortality levels had increased markedly since the decade 1695-1704.

²⁹ See Landers (1993) for a survey of the literature on this subject. The standard accounts are those of Ashton (1959); George (1966), Summerson (1978) and Sheppard, Belcher and Cottrell (1979), whilst Clarke (1992) provides a detailed analysis from the standpoint of Marxist mode-of-production theory.

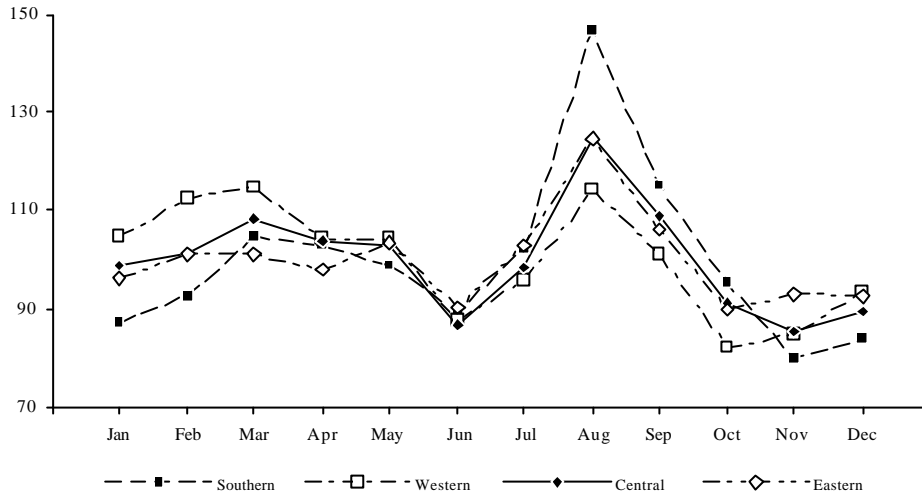
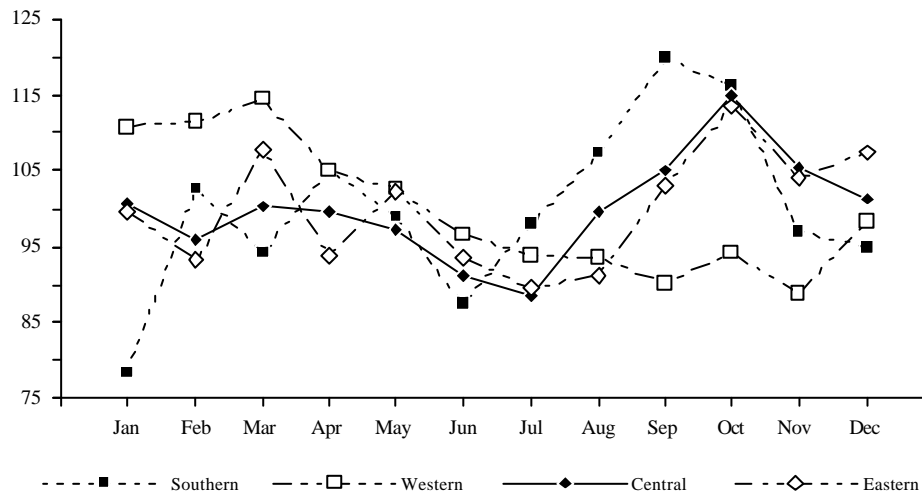


Figure 14
Child burial seasonality indices 1750-59



Equally significant from our point of view is the fact that the great bulk of new housing was built for the Žlite and the better off among those whom contemporaries termed ‘the middling sort’. The result of this was that the housing stock grew, in effect, ‘from the top down’ and increments to the accommodation available to the mass of the population came about through an involutory process akin to ‘internal colonization’, properties being divided and subdivided as they were left behind by the westward movement of the better off.

Thus, Francis Sheppard, the historian of nineteenth-century London, describing the process at work shortly after 1800, noted

the absence, except in parts of East London, of new building specifically for the working class, for most early nineteenth century landlords, being anxious for the maintenance of the value of their property, intended that their estates should be occupied by the upper or the middle classes. But... many houses intended for the 'respectable' classes often degenerated into slums within a decade or two of their erection. Once this process had begun it was almost impossible to reverse it. Decaying houses intended for occupation by a single family, with cellars, large rooms and an inadequate water supply, were invaded by half-a-dozen or more families, for whom they provided utterly unsuitable accommodation (Sheppard 1971:94).

Such a system might have provided tolerable results as long as the built-up area was expanding, but this ceased to be the case, for any sustained period, for a generation after the financial crisis of the late 1720s. Under these circumstances it is likely that popular housing conditions deteriorated considerably, especially in the older inner-suburban districts and parts of the East End, with an accompanying increase in the two proximate determinants of exposure to infection which we have termed conduction and retention.

It is the latter which was probably responsible for the emergence of spatial variations in the seasonality of burials. The autumn peak in childhood mortality seems to be a reflection of a 'new' disease which had arrived in an epidemic form in the late 1730s and was probably a form of streptococcal infection (Landers 1993, Appendix 2). On its first appearance this had taken a substantial toll across the capital and among all social classes, but it apparently became endemic only in those districts worst affected by the economic stagnation of the period.

If this argument is correct, then the resumption of growth in the last quarter of the eighteenth century would have produced a corresponding amelioration of conditions capable of reducing mortality despite the contemporary decline in real wages (Schwarz 1985, Floud et al. 1990). It does not seem plausible, however, to attribute the whole of such a dramatic and sustained decline in mortality to this factor alone, and it is likely that some of the traditional explanations, based on contemporary economic changes, are of relevance. In particular, the wider availability of cotton cloth, and the increased output of soap, probably improved personal hygiene in a manner particularly important for typhus mortality (Chambers 1972; Razzell 1974).

At the same time, water supplies increased in volume, even if their quality was as bad as, or worse than, ever with important implications for the 'water washed' diseases such as dysentery (Bradley 1974; Rudden 1985; Hardy 1991). In addition there is evidence to suggest that improvements in street paving and cleaning, in some parts of London, may have reduced the volumes of filth and excrement in public spaces that had earlier fostered the breeding of flies and thus the transmission of disease (George 1966; Porter 1991). It is likely that changes in infant feeding regimes made a substantial contribution to the decline in infant mortality (Landers 1993, Chapter 4), and inoculation against smallpox may have been important for child mortality although the direct evidence on this point is equivocal at best (Vann and Eversley 1992:222–223).

Conclusion

The secular decline in mortality, the 'mortality transition', primarily reflects a reduction in mortality from infectious diseases, and it is this which explains much of the cross-sectional variation in contemporary death rates. Recent experience has shown that the relationship between infectious disease

mortality and socioeconomic change is more complex than was once thought, and if the problems thus uncovered are to be resolved we will have to gain a much deeper understanding of the factors responsible for the former's long-term dynamic. European historical demography provides an important basis for pursuing such an understanding, but in practice its study has been pursued with little reference to the question of mortality transition and its *sequelae*.

This in turn reflects theoretical difficulties which have arisen in historical mortality studies following the failure of explanations based on a narrow standard-of-living determinism. Recent work in historical epidemiology, however, has suggested that a broader range of structural characteristics of past populations, in particular their spatial structure, may be invoked to explain mortality change which need not be regarded as an 'autonomous' variable determined by factors outside the human world. The implications of this work can be drawn out with the aid of a proximate determinants framework, giving rise to the concept of structurally-determined 'mortality potentials'.

One such configuration of proximate determinants characterized large urban centres in pre-transitional Europe and defined a 'high-potential' epidemiological regime. According to this model such centres functioned as endemic reservoirs of infection with mortality levels which were correspondingly high, especially early in life, but relatively constant from year to year. Eighteenth-century London, a critical test case, displayed these characteristics in a highly developed form. Nonetheless, its mortality patterns were strongly affected by the political events of the period, in particular the alternation of war and peace, and by the social and economic organization of the construction sector. These factors were highly specific to the time and place in question, and they serve to underline the inadequacy of ahistoric conceptualizations of mortality change.

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