Medical and Demographic History: Inseparable?

Robert Woods*

Summary. The case is made for forms of medical history that focus explicitly on sickness, health and life chances; ones that explore the effects of health interventions by examining their impact on mortality risks. Using a series of examples drawn from environmental health, midwifery and obstetric care, the paper illustrates various ways in which long-term trends in health and mortality may be read together. But it also demonstrates how fraught with problems of description and interpretation this process is likely to be. Finally, a plea is made for evidence-based medical history where ‘progress’, ‘outcomes’ and ‘results’ are given privileged positions.

Keywords: health impact assessment; mortality differentials; sickness; public health; midwifery; fetal mortality; maternal mortality; historical demography; evidence-based medical history

That part of medical history concerned with illness and health is obliged to consider consequences; the impact of medical interventions on life chances. Health impact assessment (HIA) needs to occupy a central position in medical history, just as it does in contemporary medical practice. This paper makes the case for such a repositioned medical history and for its alliance with demographic history.¹ It demonstrates that close readings of ‘health’ and ‘mortality’ are essential, need to be conducted in conjunction with one another, but are rarely straightforward. Four sets of examples will be employed to support the case. The first uses a recent review of medical history as a positioning device. The second focuses on how one might use mortality to assess health or sickness, while the third looks at work on the environment and public health. The fourth set of examples uses research on long-term changes in maternal, infant and fetal mortality to ask how, in these special cases, one might begin to establish the role of medicine.

Bad Medicine

David Wootton’s Bad Medicine: Doctors Doing Harm Since Hippocrates (2006) provides a convenient starting point for a backward look at developments in medical history during

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¹It is always interesting to look back on how disciplines develop and how a particular journal, like Social History of Medicine, has made its contribution. A comparison of Porter 1995, and Pickstone 2005, suggests that while for the former (p. 356) historical demography had ‘come of age as a relevant discipline for the social history of medicine’; the latter thought demographers might have something interesting to say about epidemics.

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Advance Access published 12 October 2007
the last 20 years. Wootton’s intention is to operate ‘against the grain of contemporary historical writing’. He argues the need for medical history to be concerned with ‘progress’, with success and failure.

In recent years the medical profession has discovered what it calls ‘evidence-based medicine’—that is, medicine that can be shown to work. This [Bad Medicine] is the first history of medicine properly to acknowledge that most medicine, even into the present day, has not been evidence-based, and indeed that it did not work. If the story I tell in this book is very often one of failure not success that is because we have begun to redefine success, which means we are now in a position to rethink the history of medicine.

His account has a turning point, 1865, which is associated with Joseph Lister’s discoveries and the recognition of good antiseptic practice in hospitals and among health professionals in general. Before 1865, the story of medicine is largely one of failure, whereas after 1865 some forms of success were increasingly possible. There was a transition from ‘bad’ to potentially ‘good’. ‘Before 1865 all medicine was bad medicine, that is to say, it did far more harm than good.’ Wootton sees medicine as a technology.

I have deliberately introduced the term ‘technology’ because I want to stress that medicine, at least since Hippocrates, has always been a technology, a set of techniques used to act on the material world, in this case the physical condition of the patient’s body. With technology it is perfectly legitimate, and not at all anachronistic, to talk about progress.

In the case of medicine, progress means that pain is alleviated, periods of sickness are shortened, and/or death is postponed. He believes that history, medical included, has favoured the ‘worm’s eye view’, ‘in which small things loomed large, and it was impossible to get one’s bearings’ over the ‘bird’s eye view’, ‘which surveyed the past from the point of view of the present, and was necessarily biased and anachronistic’. He also notes that progress in medical knowledge and therapy are not necessarily linked, since, for example, ‘if you look at therapy, not theory, then ancient medicine survived more or less intact into the middle of the nineteenth century and beyond’.

So, if all medicine before 1865 was ineffective or dangerous, why was this not recognised by doctors and why did patients keep on paying to be harmed? Wootton has several answers. One relates to the ‘placebo effect’. Patients could believe that they were being helped; their purchase of medical advice, recipes, and physical treatment was in hope or desperation of an alternative.

2Wootton 2006. Bad Medicine is a highly polemical work with a serious and important argument. References and other scholarly apparatus have been consigned to a website.
3Wootton 2006, p. 3.
5Wootton 2006, p. 8.
6Wootton 2006, p. 15. This passage refers to Herbert Butterfield’s The Whig Interpretation of History (1932).
7Wootton 2006, p. 17. The point is made in relation to bloodletting (see also p. 141). ‘A doctor in ancient Rome would have done you just about as much good as a doctor in early nineteenth-century London, Paris, or New York’ (p. 70).
8Wootton 2006, pp. 67–70.
A second possibility brings us closer to the central point of this paper. Doctors relied on anecdotal evidence, on apparently successful case histories and on the assumption that ailments should be treated in ways that were specific to each patient. Until the early decades of the nineteenth century and Pierre Louis’s work in Paris, they did not apply statistical reasoning in their accounts. ‘We might go so far as to say that the statistical table was the first direct threat that Hippocratic medicine had faced in over two thousand years. By 1860 the revolution represented by the table was complete.’ Wootton displays considerable enthusiasm for the statistical approach and observes at one point that ‘In order to get the achievements of modern medicine in perspective we have to start thinking about life expectancies.’ But he also warns that although between 1865 and 1942 ‘doctors began for the first time to defer deaths in significant numbers’ this cannot account for the ‘astonishing increase in life expectancy that took place during the same period. Medicine has been taking the credit for something that would have happened anyway’. Wootton supports Thomas McKeown’s argument in The Modern Rise of Population (1976) that ‘medicine has had almost nothing to do with modern gains in life expectancy’; is critical of the neglect of fertility; and concludes, ‘McKeown’s thesis that increased life expectancy is due to improved nutrition is thus, if one accepts [Robert] Fogel’s argument, broadly correct, but it requires one simple modification: what is crucial is nutrition in infancy and childhood, and here what matters is not only the number of calories consumed, but also the consumption of protein and vitamins’.

Bad Medicine raises important issues, therefore, and it has answers. It champions the bird against the worm; demands the reinstatement of ‘progress’; returns ‘success’ and ‘failure’, ‘good’ and ‘bad’ to the lexicon; draws a clear timeline in the sand, before and after 1865; it applauds statistical tables (and obstetric forceps); tests medical efficacy using life expectancy; and, failure demonstrated, it turns to psychological and cultural blockages in the medical profession (‘doctors’ sense of themselves’) to explain the slow pace of change. Wootton is moving along the right lines in reacting to the inconsequential, but his attack lacks rigour because it fails to deal in sufficient detail with the question ‘what is medicine for, what is its purpose?’ and it avoids a sophisticated discussion of method, such as health impact assessment in history, for example. In terms of purpose and progress, medicine must, surely, be about more than pain alleviated, sickness shortened, death postponed. Life expectancy is given a touchstone role, but then doubts are raised over its power since its level is revealed to be due to multiple causes, not just medicine.

Reading Health from Mortality

Over the past 20 years, several contributors to Social History of Medicine have observed that while health is a socially constructed concept, death is not; and that the extent of ill-health (sickness) in a population cannot be inferred from its level of mortality. Although social constructionist approaches are regularly misunderstood,
often deliberately, they offer vital insights on health, particularly from the patient's perspective and in terms of the relationship between patient and practitioner. Patients are not passive, they self-report their physical and mental condition, and they do so in ways that are affected by social conventions. How health and disease are conceptualised is of fundamental importance, it is fluid and contested. Further, the division of labour among practitioners (physicians, surgeons, apothecaries, midwives, nurses, etc.), between specialists and generalists, qualified and unqualified, and between institutions has contributed to the creation of a 'market for medicine', which has been of particular importance in framing responses to health demands from patients. David Harley has argued that health and healing should lie at the centre of medical history, and that they are 'rhetorical constructs created in particular social locations'. So that 'healing anywhere is a social construction that requires a plausible practitioner who can deploy a credible system in a successful negotiation that brings order to the patient's experience', because 'culturally specific beliefs and social structures create local realities which define and determine health and illness'. Harley is also clear that 'The rationality of diagnosis and therapy can only be understood in terms of the theory employed at the time, since it is always actors' categories that shape attitudes and actions', and that 'Retrospective rediagnosis is deeply misleading not only because it relies on rather naive acts of translation but also because it privileges supposedly stable modern categories'.

Social constructivist approaches are, therefore, strangely liberating and restrictive, realistic and idealistic in what they propose. The definition of health (thus ill-health and sickness) is made mutable, specific to individual, culture, circumstance and period; beyond retrospective analysis with today's 'understanding'. What may we say, in consequence, of sickness in ancient Rome or early-modern England? Only what contemporaries said of it themselves?

Death is another matter entirely. Although the conventions surrounding death, including grief and mourning, are socially constructed, the biological fact of death (absence of vital signs) has until recently been a constant. Now life, independent life, and the specific vital signs required must all be defined in medico-legal terms so that induced abortion and the removal of life support systems do not need to be counted as unlawful killings. The relative ease with which death may be defined, even when compared with live birth, has facilitated its registration and encouraged its analysis in the form of mortality statistics. We know a great deal about the relative risks of dying in the past in terms of age, place of residence, gender, occupation, even cause; and although this cannot be treated as some form of pure fact, as 'objective knowledge', much has been accomplished in the application of modern categories. This success has further encouraged attempts to draw wider implications from mortality concerning sickness and health; to read health as an inverse function of mortality.

13 Jordanova 1995, outlines these issues.
14 Harley 1999, pp. 432 and 434.
15 Harley 1999, pp. 417 and 419.
16 Woods 2006a.
The work of James C. Riley may come to mind at this point, but especially his *Sick, Not Dead* (1997) and associated papers. In simple terms, Riley argued that there was a positive association between rising life expectancy and morbidity during the mortality transition of the late nineteenth and early twentieth centuries. His case was based largely on British friendly society records drawn from this period. Riley fuelled a lively debate in which his several detractors took turns to launch attacks, all of which were staunchly defended. Three principal forms of criticism were made. First, following a social constructivist line, Sheila Ryan Johansson focused on the ‘cultural inflation of morbidity’. Morbidity, sickness, ill-health, feeling unwell are, as we noted above, socially and culturally defined notions that demand close contextual scrutiny. There is little that is objective, rational or consistent about them. Second, while it may be claimed that friendly society records do consistently record the duration of ‘work preventing illness’ or ‘sickness absence’ they do so in ways that are financially limited, restricted to certain groups of employees and often geographically circumscribed. Third, Riley’s point that mortality and morbidity were not positively related because, following a fundamental equation from epidemiology \( P = I \times D \), ‘mortality cannot run parallel to all three gauges of sickness [prevalence, incidence, duration], since they are unable to move parallel to one another’. However, Bernard Harris has pointed out that whilst this may be true, there is also no reason to maintain, as Riley does, that morbidity and mortality moved in opposite directions.

We have, then, a dilemma: while mortality can be defined and measured, morbidity is problematic to define and difficult to measure in a way that is comprehensive, as well as being potentially misleading even in its more specific forms, such as ‘sickness absence from work’. One possibility would involve an even more simple argument: social health, and thus social well-being, is a positive function of life expectancy. This would be to use mortality as the key indicator of health, to make the history of health also the history of life chances. A second possibility might focus more closely on relative differences rather than changes in absolute levels; to think of social groups or national populations as being more or less healthy depending upon their experience compared with, say, the elite or Sweden, for example. On the former, we have already noted how Wootton rediscovered the fact that life expectancy at birth is a compound measure of mortality in which age-groups and causes of death are combined. Riley’s own global history of life expectancy makes this all too clear.
In the high-income countries of the West, life expectancy at birth (e(0)) has doubled during the last 150 years from around 40 to nearly 80 years. It may well rise to 90 or more years by 2050, at least for females.\textsuperscript{24} In several African countries, life expectancy at birth is now less than 40 years according to World Health Organization estimates. It has declined during the last couple of decades from a level in the mid-1950s. How should these figures be read in terms of the health status of the populations concerned? In broad terms, life expectancy does reflect dramatic and sustained improvements in health (reduction in the prevalence of lethal sickness among those under 60), on the one hand, and recent deterioration after moderate gains, on the other. The deterioration is due especially to the combined effects of HIV/AIDS, tuberculosis and malaria on adult and child mortality, as well as worsening economic and security situations in a number of the countries concerned, which makes the new disease burden even more difficult to cope with. In these circumstances it would be reasonable to infer from life expectancy that health has also deteriorated and that the health transition has stalled because life chances in Malawi, for instance, are now equivalent to those experienced in Victorian England. As a foundation for broad generalisation at the society/country/region levels, life expectancy has much to recommend it.

The second possibility mentioned above concentrates on relative positions; how mortality differentials may also reflect health/sickness differences. Let us consider an example to illustrate the point further. Figure 1 uses partial life expectancy between ages 25 and 65 (e(25–65)) to compare the experience of members of the medical profession with that of all males in the same age-group in England and Wales during the last 150 years.\textsuperscript{25} It also shows evidence for Fellows of the Royal College of Surgeons (FRCSs) and of Physicians (FRCPs), as well as members of the Royal Medical and Chirurgical Society in the early nineteenth century. In the 1850s, ordinary members of the medical profession could expect to live for only 60 per cent of the maximum 40 years between ages 25 and 65. It would seem they were disadvantaged when compared with elite members of their profession and, to a lesser extent, all males of an equivalent age. Being in general practice was to engage in a dangerous trade, one in which exposure to the unwell was a constant threat. But the most interesting aspect of Figure 1 concerns the way in which these differentials crossed over around 1900. During the twentieth century, and with the possible exception of the 1930s, the life chances of male doctors were superior to all males and the gap began to widen from the 1950s onwards as their e(25–65) edged towards 40 years.

The evidence of Figure 1 is both enticing and at the same time potentially deceptive. It encourages further reflections on the state of medical knowledge in the nineteenth century, and before. If doctors were unable to protect themselves, then how could they possibly provide effective health care for their patients? Wootton would say ‘Quite so, but 1900 rather than 1865?’. On the sceptical side, it has been noted on many occasions that vital statistics and population censuses are neither free from bias within their own terms of reference, nor are they socially or culturally neutral. The registration system devised by Dr William Farr and his successors at the General Register

\textsuperscript{24}Bongaarts 2006.
\textsuperscript{25}Woods 1996; Crowther and Dupree 2007.
Office, London, mirrored a particular medical view of what was wrong with the world. It emphasised certain age-groups, places, occupations, causes of death, and it constructed categories for analysis such as ‘healthy districts’, ‘zymotic diseases’ and ‘social classes’.\(^{26}\) In this specific case, the subject group is easily defined (registered male members of the medical profession), but it is heterogeneous in terms of residence, income, skills and the social standing of patients. The experience of the Fellows of the Royal Colleges indicates this. Despite these reservations, Figure 1 allows a close reading and tells an interesting story, one that may be highly significant.\(^{27}\)

**Environment and Public Health**

Like many before him, Wootton is most taken by the story of Dr John Snow and the Broad Street pump. He reproduces the famous map of 1854, a cartoon of 1866 entitled ‘Death’s dispensary’ showing people, mainly children, clustered round a water pump with Death working the handle, and he devotes a chapter to ‘John Snow

\(^{26}\)See the contributions to Szreter 1991; as well as Hardy 1994, Bryder 1996, Higgs 1996.

\(^{27}\)Ewbank and Preston in Caldwell et al. (eds) 1990, Preston and Haines 1991, explore the possibility that well-educated professionals were the first to take advantage of new knowledge about health care practices and that this benefited the survival chances of their infants and children during the first years of the twentieth century. It also led to sharper social class mortality differentials. Their findings for the USA are consistent with the evidence presented here in Figure 1.
and Cholera’. Snow was able to demonstrate the mode of communication of cholera without understanding its scientific cause. Making empirical observations, drawing reasonable inferences and implementing the associated health intervention policies could improve the public’s health by thwarting Death. This was the simple, yet most effective lesson. Snow was not the first, and certainly not the last, to use geographical analysis in an epidemiological study, but his is still one of the most telling accounts.

This section draws examples from the history of public health to illustrate the sorts of connections that Snow made between unhealthy environments, indicated by Death’s repeated visits, the recognition of such regularities and the responses of governments. Places, epidemics and politics are inter-linked.

In the early modern period there was little that individuals and authorities could do to limit the spread of disease except by quarantine, the isolation of infected cases, early warning and keeping out of harm’s way. The retreat of bubonic plague in seventeenth-century England illustrates the effects of each of these to some degree. In recent years work on plague, as well as cholera, has responded to the realisation that demographic crises, including epidemics and famines, may have had dramatic short-term impacts that captured the attention of contemporaries, but their longer-term consequences for morbidity and mortality were far more muted. While demographers have become disillusioned, historians have turned to using the drama and challenge of epidemics as social litmus paper. How did individuals, society and the authorities cope with the crisis? Were they prepared? Were the victims poor? Were foreigners blamed? Who was held responsible? Was there a lasting legacy? Late outbreaks of plague or cholera were treated like Chernobyl or Three Mile Island, administrative failings, the symptoms of a deeper malaise.

Mary Dobson has not followed this line. Her perspective is both local and global. It is concerned with mortality gradients, ‘contours of death’, with specific disease environments and transatlantic transfers. Although she does deal with disease in its epidemic state, she is more concerned with background causes of mortality, with the everyday and commonplace rather than the dramatic. Her work emphasises the importance of place and the sorts of risks people had to cope with in making a living in low-lying marshland parishes or in small towns. All of which helps to create a picture of light and shade, of interconnectedness rather than isolation, where environments are differentiated and some can lead a relatively charmed life. John Landers has also taken the epidemiological road, but his environment is the most dangerous. Eighteenth-century London was a ‘demographic sink’ an ‘urban graveyard’ which drew off the surplus rural population from surrounding counties. But, according to the Bills of Mortality, its position was neither static nor undifferentiated. These studies by Dobson, Landers and others are of

29 Cliff et al. 1998, has many more examples.
30 Epidemics, often the isolated epidemic, became very popular among ‘worm’s eye view’ specialists who often lost sight of ‘killing power’ and the relevance for health. London 1665, Marseille 1720, Hamburg 1892, have all received this treatment.
vital importance in charting the history and geography of disease before and after Snow. They also describe the environmental hazards to health and premature death that set the challenge for more directly interventionist public health measures in the nineteenth century.33

The very first paper published in Social History of Medicine, Simon Szreter’s ‘The Importance of Social Intervention in Britain’s Mortality Decline c. 1850–1914: A Re-interpretation of the Role of Public Health’, proclaimed the need for a new perspective in work on the ‘sanitary revolution’.34 He argued for a perspective that emphasised the contribution of the state via its application of engineering technology and its use of social regulation by the legal system. Szreter’s critique of the McKeown thesis, which played down the roles of both medical and public health interventions during the early decades of the mortality transition whilst emphasising nutrition and autonomous epidemiological changes, led to a lively debate with Sumit Guha.35 This debate still rumbles on, and it does so for several interesting reasons.36 First, McKeown asked an important question about the causes of mortality decline. He constructed a list of the factors involved and he gave them numerical weights to reflect their relative importance. His method of enquiry was from the social sciences rather than history and it purported to give a bird’s eye picture. Second, although McKeown was wrong about the causes of population growth in England since increased fertility turned out to be more important than mortality decline in the eighteenth and early nineteenth centuries, his list of possible influences on that mortality decline (medicine, nutrition, public health, epidemiology) was comprehensive.37 Third, McKeown’s ranking and weighting of his factors for the nineteenth century was probably wrong. It was certainly premature and far too precise. McKeown’s knowledge of the causes of long-term demographic change was, in short, insufficient to sustain his thesis, but the very existence of that thesis helped to provoke the development of a richer, more securely founded evidence base.38

The evidence now available supports Szreter’s insistence that we give more weight to the positive benefits of public health interventions, although exactly how much still remains uncertain. It also warns of the care that needs to be taken over periodisation. To be sure, the benefits of public health interventions were to be seen in the twentieth century, but they had been laid in the nineteenth. It repeatedly demonstrates how complex the patterns of change were, how hard won such evidence is, and how open to contested interpretations it remains. Policing, institutions and monitoring mortality trends offer some examples. Christopher Hamlin has illustrated the importance of the ‘local state’ in the organisation of environmental policing, but especially the variability of practices among English and Welsh towns in the 1870s.39 Deaths in institutions are now routinely reassigned to ‘usual place of residence’ so that local mortality measures can more closely reflect normal conditions. In the nineteenth century the location of

33Hardy 1993, applies historical epidemiology to Victorian London.
36Harris 2004.
37Wrigley and Schofield 1981.
hospitals, workhouses, barracks and prisons had a dramatic effect on the apparent quality of local environments. The removal of these institutional effects has proved a challenging, yet particularly important task. Finally, Graham Mooney has discussed the use of mortality measures as health performance indicators in the creation of ranked lists of local authorities, and how such monitoring procedures led to conflicts between Medical Officers of Health and officials from the General Register Office, London.

Given these and other advances it might now be thought possible to evaluate the impact of public health interventions at least on mortality, if not health outcomes. Figures 2 and 3 illustrate why this may still be just beyond our reach. They use American and French data to show the cumulative nature of sanitary progress especially in urban environments. Figure 2 traces the expansion of piped water supply and the sewerage system in the USA and France during the late nineteenth century. The graphs display the typical form of S-shaped innovation adoption curves with a slow initial phase followed by rapid mass adoption and finally a phase in which the take-up rate slows down and only the laggard towns are left. They indicate that the USA and France moved through this sequence at roughly the same time, as did the industrialised countries in general. Figure 2 also shows the increase in the number of US cities with populations over 2,500. This reflects the challenge of urban growth and urbanisation to public health. Figure 3 uses the case of Chicago to illustrate the inputs and outputs of the sanitary revolution. The benefits of a secure water supply and an effective sewerage system included an increase in the per capita consumption of water and a dramatic decline in the death rate from typhoid.

Figures 2 and 3 make the nineteenth-century water supply and sanitation initiatives look very neat and tidy, ‘dose-response’ almost. But there are still several points for debate, particularly concerning the urban environment as a threat to health (the urban penalty) and the way it was tamed. One of these relates to the consequences of urbanisation in populations where there was a strong urban–rural mortality gradient. Sanitary measures must not only improve a poor health environment, they must also cope with rapid expansion in the scale of that physical environment as the country dwellers move to town. A second problem is concerned with the confounding effects of environment and wealth. What will be the health and mortality experience of wealthy people who live in dangerous environments and, conversely, how will the poor fare in safer, less epidemiologically demanding rural environments? The challenge of the urban environment was tackled first in the nineteenth century; motherhood and class came later.

Fetus, Infant, Mother

It has been remarked that ‘The period immediately before birth, birth itself and the period immediately after birth became radically less dangerous to both mother and child in the

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40 Mooney et al. 1999.
41 Mooney 1997.
42 Melosi 2000, has many examples of how sanitary engineering transformed American cities. Woods in Schofield et al. (eds) 1991, links the public health movement to changing mortality conditions.
45 Schofield et al. 1991; Garrett et al. 2006.
long eighteenth century. The same observation could be made with even more assurance of the twentieth century but not, it would seem, of the nineteenth. Wrigley’s statement, and its extension, provides a test of the contention that medical and demographic history are indeed inseparable. Beginning with the eighteenth century, we would need to demonstrate that fetal, infant and maternal mortality did indeed decline substantially, that little change occurred in the nineteenth, and that even sharper downward trends re-emerged in the last century. This in itself would be a long first step after which could come an informed discussion of developments in medical practice, with the presumption that progress came at different rates in different centuries.

This is a difficult area for historical demography. There are problems of definition and data. Infant mortality has received the most attention because it is relatively straightforward to define (infant deaths related to live births) and because it can be derived by linking entries from burial with baptism registers as well as directly from infant deaths and births where civil registration data are available. Ideally, maternal mortality should involve only those deaths directly associated with childbirth (maternal deaths

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Wrigley 2004, p. 83.
Garrett et al. 2006.
related to births or deliveries), but in practice all maternal deaths within an arbitrarily fixed period of time after childbirth are included. Late-fetal deaths or stillbirths are defined as those fetuses that have reached a viable stage of development in terms of gestational age, but are born without vital signs. Both ‘viability’ and ‘vital signs’ are subject to a variety of interpretations that affect practical definitions. In England and Wales, maternal deaths were not registered until 1847 and stillbirths not until 1927. In Sweden, by contrast, both entered the state registration system in the 1750s. It is a considerable challenge to derive late-fetal, infant and maternal mortality rates so that long-term trends can be described even for what would normally be regarded as statistically advanced countries.

Table 1, which focuses on mortality among females of reproductive age as well as maternal mortality, illustrates what has been achieved in the case of England and Wales. It uses partial life expectancy between ages 20 and 45 ($e_{20-45}$) alongside the maternal mortality rate (MMR), and it compares England as a whole and the wives of members of the British peerage. Several of the series span four centuries by combining parish register and vital registration based evidence. None is without its own distinctive complications. For example, series (4) is based on the ratio of deaths in childbed to baptisms reported in the London Bills of Mortality. Neither the numerator nor the

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49Strictly speaking, national estimates based on the analysis of parish register data from 1538 to 1837 usually relate to England while those using civil register material from 1837 relate to England and Wales.
denominator has been adjusted to allow for under-reporting although it is now usual to inflate the number of baptisms to bring them closer to the probable number of births. If the denominator is adjusted upwards, then the rate is brought down to a level very close to the MMR for England (3).

Tables 4 and 5 illustrate the series from Table 1. Figure 4 compares the best available estimates of maternal mortality for England (column (3)) with London (4) and the peers’ wives (5). The shaded area gives the range within which the last-mentioned series can

Table 1. Estimates of partial life expectancy between ages 20 and 45 (e(20–45)) and maternal mortality (MMR) for all females in England and London, and the wives of British peers

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<tr>
<td>1875–99</td>
<td>18.4</td>
<td>20.9</td>
<td>48</td>
<td></td>
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<tr>
<td>1900–24</td>
<td>21.0</td>
<td>21.3</td>
<td>40</td>
<td></td>
<td></td>
<td>40</td>
</tr>
<tr>
<td>1925–49</td>
<td>21.5</td>
<td>21.8</td>
<td>31</td>
<td></td>
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<tr>
<td>1950–74</td>
<td>22.6</td>
<td></td>
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<tr>
<td>1975–99</td>
<td>24.2</td>
<td></td>
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</tbody>
</table>

Notes: Partial life expectancy gives the number of years between ages 20 and 45 that an average female may expect to live. The maternal mortality rate (MMR) gives the number of childbirth related maternal deaths per 10,000 births or deliveries. Columns (5) and (6) are for 50-year periods: 1550–99, 1600–49, etc. The MMR of the principal London lying-in hospital, 1750–99, was 150. Woods 2007, Table 2.

Sources: (1) estimated from e(25) in Wrigley et al. 1997, Figure 6.20, p. 305, using Princeton Model North, and for 1841 onwards interpolated from national life tables for England and Wales; (2) derived from Hollingsworth 1964, Table 4.3, p. 57; (3) Wrigley et al. 1997, Table 6.29, column (3), p. 313, and for 1850 onwards, Loudon 1992, Appendix 6, Table 1, p. 542, based on vital registration data for England and Wales; (4) derived from Loudon 1992, Figure 10.1, p. 159, based on the London Bills of Mortality; (5) derived from Smith and Oeppen 2006, Figure 4.7, p. 73, based on Hollingsworth’s British peerage data; (6) Schofield in Bonfield et al. (eds) 1986, Table 9.5, p. 248.
reasonably be expected to fall. Figure 5 shows the partial life expectancies ($e^{(20–45)}$) for all females and the peers’ wives (columns (1) and (2)).

There are some surprises here. The first point to emphasise is that maternal mortality appears to have risen in the seventeenth century, declined in the eighteenth, stabilised in the nineteenth and declined again from the 1930s. The estimates for England, London and peers’ wives support one another in this. London was worse than England, but not dramatically so, and the peers’ wives seem to have experienced even higher average risks of dying in childbirth. Indeed, Table 1 indicates that the peers’ wives had, in general, even higher maternal mortality than the ‘maximal’ series originally constructed by Roger Schofield. It was not until the first half of the twentieth century that peers’ wives became indistinguishable from all females. For most of the four centuries we have a case of similar trends, but at different levels. However, the partial life expectancies give a different account. Among all females, decline in the seventeenth century

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51 The MMR series for peers’ wives is based on relatively small numbers, although they do increase over time and the shaded area narrows (± 2 standard deviations round the best estimate should contain the true rates with a 95% probability). This series was kindly made available by Richard Smith and Jim Oeppen of the Cambridge Group for the History of Population and Social Structure. It was constructed from the original database compiled by T. H. Hollingsworth. Hollingsworth 1964, 1977.

52 Table 1 makes clear that the $e^{(20–45)}$ for all females has been estimated from the $e^{(25)}$ series for females reported in Wrigley et al. 1997. Their Figure 6.20 also shows $e^{(25–45)}$ for females, but these partial life expectancies are remarkably high (17.4 out of a maximum of 20 years for the mid-eighteenth century). Using a model life table system, Princeton Model North, with life expectancy at age 25 in years ($e^{(25)}$) provides a way of generating more realistic $e^{(20–45)}$ estimates. It has also been necessary to derive period $e^{(20–45)}$s for the peers’ wives from life expectancies reported for birth cohorts in Hollingsworth 1964.

53 Schofield 1986, Table 9.5, p. 248. Schofield’s original ‘best estimate’ of the MMRs among English females (using family reconstitution studies for 13 parishes) was somewhat lower than the series in Table 1, column (3).
The eighteenth century was followed by recovery in the eighteenth, and continuous improvement in the nineteenth and twentieth so that $e_{20-45}$ is now 24.4 years. The life expectancy of peers’ wives did not recover so quickly in the eighteenth century, yet by 1800 there was little difference with all females and the two series moved upwards together.

Irvine Loudon’s account of maternal mortality stresses the role of puerperal fever. It compares the general female population with the minority who gave birth in the lying-in hospitals or Poor Law institutions, and it looks at the contributions of midwifery, obstetric and antiseptic practices as they influenced maternal mortality rates in a number of countries. However, Loudon is mainly interested in the late nineteenth and twentieth centuries, and especially the reasons for the marked and very dramatic downturn in maternal mortality that occurred virtually simultaneously in his case study countries during the 1930s. The introduction of the sulphonamide drugs in hospitals and general practice was the critical cause of the decline in puerperal fever deaths. Before the 1930s, differences in level and trend are more difficult to explain. Loudon has suggested the following.

High maternal risk could be associated with cheap untrained midwives or expensive over-zealous and unskilled doctors. Sound obstetric practice by well-trained midwives could produce low levels of maternal mortality even in populations which were socially and economically deprived.

The availability of good-quality, well-trained and state-registered midwives in Norway, Sweden, Denmark and the Netherlands has been used to explain the decline in maternal mortality during the late nineteenth century. Conversely, the failure of the state to provide

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55 Loudon 2000, Figure 11.2, p. 186. The turning point was 1936 in England and Wales; the same year as the Midwives Act, which established a national service of salaried midwives.
such support in Britain may help to account for the continuing high level of maternal deaths up to the point of common decline in the 1930s.\textsuperscript{57}

What of maternal mortality in the eighteenth century? All the demographic evidence summarised in Table 1 points to the conclusion that deaths in childbirth did decline in England. Was this the result of improvement in the quality of birth attendants and obstetric care in general? There is some evidence to support this argument, just as there is for the speculation that a small group of ‘expensive over-zealous and unskilled doctors’ were responsible for higher maternal mortality among the peers’ wives.\textsuperscript{58} However, it is also important to take account of the evidence provided by the partial life expectancies. The peers’ wives were only disadvantaged during the first three-quarters of the eighteenth century, before and after their mortality between ages 20 and 45 was the same as or lower than the female population in general. Other causes of death were also responsible for mortality in this age-group. Tuberculosis, for one, was an especially important contributor alongside, and often associated with, pregnancy-related deaths. It seems likely that Figures 4 and 5 illustrate different aspects of the same story. Mortality among females aged 20 to 45 did decline during the eighteenth century and this was partly due to some improvements in obstetric care having beneficial effects on maternal mortality. But members of the small, socially exclusive, endogamous aristocracy exposed themselves to greater risk by employing specialist male doctors who, whilst technically more skilled than female midwives, were also more effective communicators of puerperal fever. Members of the aristocracy may also have experienced higher levels of tuberculosis mortality than the commoner population.\textsuperscript{59}

What happened to the offspring before and after birth? Loudon has demonstrated that in Britain maternal and infant mortality did not share the same course during the nineteenth and twentieth centuries.\textsuperscript{60} The former went into secular decline from about 1900, while the latter, as we have seen, declined from the mid-1930s. The causes of these changes were very different, and rather specific. It might be supposed that late-fetal mortality would be related closely with both maternal and infant mortality but, again, the matter is a complicated one. Late-fetal and neonatal mortality (first month or 29 days after live birth) were associated, and, in some circumstances, it would also appear that late-fetal and maternal mortality were linked. Figure 6 compares the relationships between late-fetal and maternal mortality in England and Sweden. All four series share a common turning-point in the 1930s, but in Sweden the stillbirth rate fluctuated

\textsuperscript{57}Högberg 2004; Loudon in Marland and Rafferty (eds) 1997.

\textsuperscript{58}Wilson 1995; Woods 2007. Lewis 1998, argues that the aristocracy and the commoner populations experienced similar levels of maternal mortality in the eighteenth century. Her finding has been taken up in Razzell and Spence 2006, p. 402, in their discussion of wealth-related mortality gradients. Table 1 would make their point even more effectively. Wealth did not secure health before 1900. Peers’ wives were as badly served as those poor married women who gave birth in lying-in hospitals (MMRs of 170 compared with 150).

\textsuperscript{59}This is no more than a ‘working hypothesis’, which it would be very difficult to test. Quite why during the nineteenth century peers’ wives remained at a disadvantage in terms of maternal mortality, but not in terms of mortality in general in the age-group 20–45, remains a mystery. It is also important to consider the potential effects of smallpox in pregnancy.

\textsuperscript{60}Loudon 1991.
around 30 per 1,000 total births while maternal mortality declined during the nineteenth century. In England, as far as we can tell, the two rates ran parallel to one another before the twentieth-century downturn with decline in the eighteenth and stagnation in the nineteenth.\footnote{The stillbirth rate for England must be estimated. Woods 2005 describes the various procedures possible.} Since the stillbirth rate is responsive to the quality of obstetric care (affecting intrapartum deaths during labour), the health of the mother (especially her nutritional status affecting birthweight and prematurity) and the prevailing disease environment (relating to fetal infections), accounts that emphasise improvements in maternal health and the quality of birth attendance will find additional support during periods when late-fetal mortality is in decline.

This section does not have a simple conclusion. Its purpose has been to reflect on Wrigley’s observations concerning the long eighteenth century and to illustrate how demographic evidence may be used to make a case for health. If, as appears likely, the risks associated with childbirth diminished, while fetal and infant survival improved, then there must be at least a prima facie case for certain specific sections of the population to have enjoyed healthier lives, for short periods at least. Even to get this far requires many assumptions and approximations. Several readings are possible; one would involve a positive role for medicine in the eighteenth century.

**Evidence-Based Medical History**

The first meeting of the Society for the Social History of Medicine (8 May 1970) heard a lecture by Thomas McKeown entitled ‘A sociological approach to the history of medicine.’\footnote{McKeown 1970.} He made a case similar to the one being made here: health, treatment and

![Fig. 6. Maternal mortality (MMR) and late-fetal mortality (SBR), England and Sweden. Note: MMR is per 10,000 births or deliveries and SBR is per 1,000 total births (live births plus stillbirths). Source: See text for explanation and sources.](https://academic.oup.com/shm/article-abstract/20/3/483/1641460)
effectiveness should lie at the heart of medical history and, if they do, they will need to be
linked with demographic enquiries so that impact may, perhaps crudely, be assessed. The
demise of the McKeown thesis appears, ironically, to have tarnished the entire argument.
The polarisation of research methodologies (modern or post-modern, bird or worm,
biology or culture, science or humanities, etc.), disciplinary defensiveness and the reaction
to ‘death by numbers’ will also have played their parts. McKeown and Wootton have
come to similar conclusions in terms of approach because both wish to concentrate on
outcomes: death postponed, sickness cured, healthy life expectancy extended. The
examples illustrated above demonstrate how important this goal is, but also how difficult
it is to achieve. The evidence is often of indifferent quality, it is always socially constructed,
it is rarely unambiguous, and it never speaks for itself. Quantitative evidence in the form
of statistical tables, maps and time-series conveys a precision that is often spurious. It
requires critical and sceptical interpretation, just like the patient’s first-hand account,
the prescription, the medical textbook or the physician’s autobiography, for example.63
We have concentrated here on such quantitative evidence, particularly that from demo-
graphy, but the broader argument would assert the need for an evidence-based medical
history, one committed to evaluation (which may involve ‘bad medicine’ or ‘progress’),
where the sources are widely drawn and their forms are varied.64

Acknowledgements
I should like to thank the Wellcome Trust for its financial support of a project entitled
‘Fetal Health and Mortality in the Past’.

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63It is interesting to compare Thomas and Williams 1998 with Howard 2003. The former reviews a poorly-
designed social survey from the 1930s which attempted to derive the proportion of spontaneous and
induced abortions, while the latter applies ‘discourse analysis’ to the writings of Alice Thornton concern-
ing her experience of childbirth and is, in passing, rather disparaging of Schofield’s (1986 in Bonfield
et al.) reductionist attempt to derive statistics to show the general risks. The failed survey becomes a
mine for material on individuals’ stories and Mistress Thornton is a victim of ‘reception theory’.
64This is a mixed methods approach now commonplace in some areas of the social sciences. Quantitative
and qualitative evidence are given equal standing.
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