Shifting Sex Differentials in Mortality During Urban Epidemiological Transition: The Case of Victorian London

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ABSTRACT

Unlike some forms of mortality inequality, sex differentials in England and Wales increased unequivocally during the Victorian era. A gradual reversal of this trend was observed only recently. The origin of the widened mortality gap between males and females lies in the transformation of the epidemiological landscape during the second half of the nineteenth century, from one where epidemic and endemic infectious diseases dominated, to one where chronic, degenerative conditions began to prevail. This so-called 'epidemiological transition' had differential impacts on males and females through time and in space, partly because mortality change was mediated by socially constructed gender roles. Research on rural and urban contrasts has shown consistent excess female mortality in youth and early adulthood. But because London did not exhibit this particular sex bias in the Victorian period, examination of internal variations in the capital offers an opportunity to explore the influence of factors that varied *within* the urban environment. Using life table decomposition, the article reveals that contrasts in cause-specific mortality between boys and girls and elderly men and women contributed most forcefully to overall male/female differences in life expectation at birth. Copyright © 2002 John Wiley & Sons, Ltd.

INTRODUCTION

Sex differentials have formed a minor but important component of studies of mortality in Victorian England and Wales. That females enjoyed a higher life expectancy at birth than males at the beginning of Queen Victoria's reign in 1837, and that the gap widened as the nineteenth century progressed, are not in dispute. But the deceptive simplicity of this narrative has come under scrutiny. Debate has tended to revolve around three main themes: the ages at which female mortality exceeded that of males; how these discrepancies varied from place to place, or between types of place; and which social, cultural and economic influences might explain the observed differentials. Despite the overall advantage of females over males, closer examination of age-specific mortalities in urban and rural England and Wales demonstrates that girls and young adult women tended to have higher rates of mortality than boys and young adult men. Hitherto, an urban/rural anomaly in female mortality rates has been at the heart of debate. Explanations for sex differential mortality have thus been

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sought which explore the impact of capitalist industrial organisation on the social status of women and girls in a society that concentrated the means of production in towns and cities. Primary consideration has been given to sex differentials in the earliest age groups (Ryan Johansson, 1977, 1984; Wall, 1981) and the middle stages of adulthood as the periods of life most clearly responsible for the mortality differential between Victorian males and females. There has been a focus on explanatory factors concerning the economic value and cultural worth of girls and, particularly, women (Humphries, 1991; Kearns, 1991; Ryan Johansson, 1977). Since the lion's share of attention has been devoted to explaining sex differentials in adult mortality, heavy emphasis has been placed on respiratory tuberculosis as the signal disease of differentiation (Ryan Johansson, 1984: 465).

This paper argues that greater understanding of the determinants of sex differential mortality in the past can only be reached by considering a wide range of causes of death. By adopting a life-course perspective, it also demonstrates that insufficient attention has been devoted to those age groups which were in fact most implicated in the increasing sex differential in life expectancy at birth–infants, the elderly and, to a lesser extent, children.[1] The suggestion is made that, as indications of the relative social and cultural value of the sexes, transformations in health between girls and boys and men and women that took place *within* the urban environment were as significant as urban/rural variation, particularly as suburbanisation and the residential segregation of classes assigned a spatial dimension to class divisions. More specifically, it is proposed that at least within a metropolitan context, urban socio-cultural and socio-economic environments mediated the impact of 'fertility' – broadly defined as reproductive function, childbearing, parity, and completed family size – on sex differentials in mortality.

Before describing the data sources used for the present study, the article outlines a framework of determinants of sex differences in mortality in biological, cultural and economic terms. It then addresses the broad question of widening inequality during a period of mortality improvement in England and Wales. The increasing national sex differential in life expectancy at birth is described, and the importance of the Victorian period is established. Because of its size – never accounting for less than 13% of the national population in the second half of the nineteenth century – Victorian London represents the one city in Britain for which a detailed intra-urban investigation may be undertaken (Williams and Mooney, 1994; Mooney, 1994).[2] Changes in age-specific death rates and life expectancy at birth are then documented to assess the scale of sex differential mortality in the British capital between 1851 and 1910. An approach is developed which emphasises that the sex differential in life expectancy at birth was variable not only through time but also in metropolitan space. Each of the districts in London is placed into one of four large subdivisions, termed sectors, which are classified according to prevalent socio-environmental conditions. A range of epidemiological and demographic methods are employed to assess the magnitude and significance of differentials between these sectors, and life table decomposition is used to ascertain cause of death and age-group contributions to the sex differential in life expectancy at birth. This procedure reveals that at mid-century, metropolitan sex differentials were determined primarily by variations in early-age mortality, underpinned by discrepancies in the rate at which girls and boys succumbed to the ravages of airborne infectious diseases and a range of indeterminate conditions. The middle stages of the epidemiological transition shifted the focus of the metropolitan differential more towards these indeterminate causes in the oldest age groups.

DETERMINANTS OF SEX DIFFERENCES IN MORTALITY IN HISTORICAL PERSPECTIVE

The potential list of determinants of sex differentials in mortality is as long as the list of determinants of mortality itself. It is beyond the scope of this paper to scrutinise each of these determinants in any great detail, but it is at least possible to categorise many of them into groups that embrace biological, socio-cultural and socio-economic factors (Nathanson, 1984; Hemström, 1999: 1762). It should
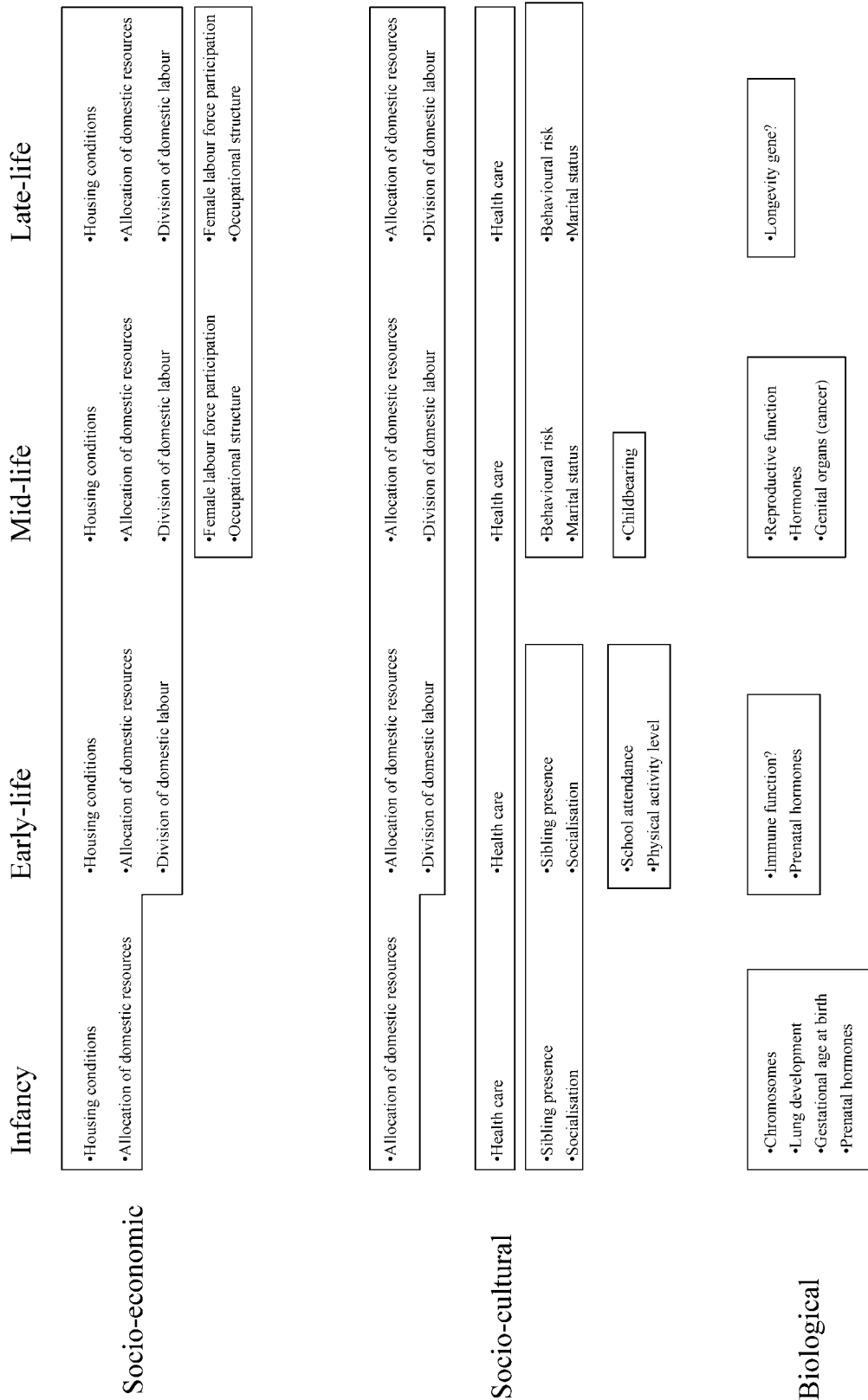
Figure 1. Determinants of sex differentials in mortality in historical perspective.

also be remembered that their impact varies through time, in space, and throughout the life of individuals. Figure 1 attempts to capture each of these rudiments by dividing the life course into four stages: infancy, early-life, mid-life and late-life (other divisions and terms have been adopted by various authors; see Laslett, 1989; Wadsworth, 1997: 861–3; Arber and Cooper, 2000). Each stage is partitioned into three segments to reflect the potential contribution of grouped determinants to the sex differential in mortality. The purpose of the diagram is to make the point that the total sex differential in mortality or life expectancy in one place at a specific time can be related to the life course/determinant matrix.

Biological factors have an important role to play throughout life, but the current state of medical knowledge about their relative contributions to sex differential mortality is somewhat uncertain and controversial (Waldron, 1984, 1998; Wizeman and Pardue, 2001). Chromosomal differences between boys and girls may affect susceptibility to a small number of diseases. Foetal lung development is slower in males than females and this may have some bearing on resistance to respiratory disease in infancy and early childhood. Males are also more often born at a lower gestational age (although they generally have higher birth weights than girls), perhaps resulting in a higher risk of premature death. It is also possible that higher prenatal levels of testosterone may mean that boys engage more fully in physical and aggressive activity, although, as the diagram also indicates, gender differences in activity levels and socialisation are culturally constructed (Carpenter, 2000: 53–4). Waldron's review (1998) found little or no evidence that biological factors cause the immune function to differ greatly between boys and girls, clearly an important consideration in societies prone to intermittent and endemic bouts of infectious disease mortality such as nineteenth-century urban Britain. In adulthood, the biologically-based differences in the reproductive functions and organs of women and men have been socially, culturally and economically fashioned into gender roles that lead to uneven health risks. Notice should also be given to the potentially protective role of endogenous hormones for ischaemic heart

disease in women and vice versa in men (Waldron, 1983: 326–7). A matter of contemporary debate is the extent to which genetic factors might unevenly influence longevity, but any potential differences between males and females await clear elucidation (Johnson and Shook, 1997; Wizeman and Pardue, 2001).

Given that this paper examines the sex gap in mortality from a spatial, intra-urban perspective, Waldron's conclusion that 'the effects of genetic factors on sex differences in mortality are variable depending on environmental conditions' (1983: 329) is particularly apposite. It should be emphasised here that by 'environmental' Waldron means all non-biological factors, from occupational hazards, through smoking, to reduced discrimination against females. These 'environmental' influences can be allocated to practically any number of subcategories – demographic, behavioural, occupational, and so on – but a simple bipartite formulation into socio-cultural and socio-economic determinants will suffice here. These two groupings are by no means mutually exclusive, as shown by the presence of siblings and allocation of domestic resources in infancy and early life in Fig. 1: both are culturally and economically constructed. In contemporary Asian societies the impact that neglect has on sex differential health and mortality, and the 'masculinity' of the population, is now well-recognised (Koenig and D'Souza, 1986; Das Gupta, 1987; Harriss, 1989; Coale, 1991), but the extent to which it was a common familial experience in historical urban Britain is far from clear (Wall, 1981). Ryan Johansson argued that because the modernisation of agriculture 'gave specific cash values to various forms of farm production' (Ryan Johansson, 1984: 464), the involvement of men and boys in production for the market reduced the importance of the domestic labour contribution made by women and girls, resulting in the greater concentration of resources on sons as opposed to daughters. The role of sibling presence is also ambiguous, but because infectious diseases tend to have different exposure and fatality rates in boys and girls, the decline of family size – which has been linked to the decline in childhood mortality as a result of the reduced opportunities for the transmission of infectious disease in the

domestic setting – takes on a gendered significance (Reves, 1985). Together with declining fertility, provision by the state of 'fever' hospitals has also been implicated in the decline of epidemic diseases, particularly in London (Ayers, 1971; Hardy, 1993). The ailments catered for in these institutions – smallpox, scarlet fever, typhoid and typhus fever – struck down infants and children in disproportionate numbers. Furthermore, the pattern of boys' and girls' attendance at school varied greatly in later nineteenth century urban Britain (Davin, 1996), and many contemporaries were of the opinion that schools acted as a focus for disease transmission (Hardy, 1993; Mooney, 1996). On the one hand, the role of education connects to the presence of siblings in that diseases contracted in school (index cases) were subsequently passed on in the domestic environment (secondary cases). On the other hand, schooling also connects to the division of labour within the home, because girls were more likely than boys to be kept back from school to perform household chores and childcare duties (Davin, 1996: 98). Not only did this expose a girl to the risk of domestic accidents, it also meant that she was more likely to become a secondary case of disease, which, research into measles has indicated, may have been more prone to fatality than index cases (Aaby, 1998).

Marriage is generally considered to have a positive effect on health, but more so for men than women. The division of domestic labour is obviously a prime cultural–economic determinant of mid-life sex differentials in mortality. This, together with the uneven distribution of income, food and other resources within the home, produces a situation in which the very presence of children in the family disproportionately impairs the health potential of mothers (Shorter, 1991: 240–41). Recent individual-level research in the UK suggests that this may no longer in fact be the case (Arber and Cooper, 2000), but in late eighteenth- and early nineteenth-century rural Germany, the birth of a child to a typical father and mother increased the risk of mortality of the father by 4.8%, but of the mother by 12.4%. This result was independent of deaths associated directly with childbirth (Klasen, 1998: 456). Of course, part of the reason for this startling difference is that country-dwelling women were also required to assist men in rural labour, but there is no reason to suppose that a high level of female participation in the urban labour market did not exact a similar price. The occupational structure of local labour markets was crucial in this respect: female employment was dominated by domestic service in some parts of London, by sweated trades in others, and by teaching and professional occupations in yet more (see also Alexander, 1983). Occupational structures were also significant for exposing males and females to specific types of disease environment; women workers in the sweated boot and shoe trade in the East End and men employed as clerks in cramped City offices were both probably exposed to a heightened risk of contracting respiratory tuberculosis (Woods and Williams, 1995).

In the absence of financial and state-supported provision for the elderly, there was no single retirement age and many determinants of the gender bias in health and mortality in mid-life remained pertinent in the later stages. For example, behavioural risks, such as tobacco and alcohol consumption, and the social acceptability of them, are clearly structured along gender lines (on tobacco, see Graham, 1993; Hilton, 2000; on alcohol, particularly in relation to Swedish mortality, see Fridlizius, 1988; Willner, 2001). As we have already observed in relation to metropolitan fever hospitals, the importance of access to health care transcends the life course. The term 'health care' is used rather loosely in connection with Fig. 1, since many of the medical therapies on offer, even in the early twentieth century, were ineffectual. Rather, it is intended to indicate that private health insurance schemes run by friendly societies were accessible only to men in work. Often, these schemes would offer medical consultations and security of income in times of illness, but whether they actually deferred death and improved the health of the male population is still a matter of debate (Riley, 1997).

Social, cultural, economic and biological factors act in a gender-specific way to shape the relative resistance and exposure of boys and girls, men and women, to uneven levels of health risk. It is now acknowledged that these risks 'accumulate' throughout the life course

and impinge upon the health status of the individual at a later stage (Riley, 1989). Mid- and late-life mortality rates partly reflect the health experiences of individuals many years earlier. It should therefore not be forgotten that cohort influences are also gender-specific. Cohort effects can only be investigated indirectly with the population-based data published for districts in England and Wales in the nineteenth century (Mooney, 1994; for the Netherlands, see van Poppel, 2000), and no attempt will be made to do that here. Rather, this paper underlines the importance of investigating sex differential mortality with specific reference to age groups and causes of death in order to help unravel the nature and form of sex variation in health. But before these differentials can be explored in depth, a brief review of the data sources used in the remainder of this paper is required.

DATA SOURCES

A little more than a decade ago, Michael Anderson observed that sex differentials in mortality, and particularly excess female mortality, had failed to attract sustained attention (Anderson, 1990: 18). Perhaps one of the reasons for the partial neglect in the Victorian period is that data are not always available at the level of detail required for a satisfactorily exhaustive evaluation. In the case of England and Wales, comprehensive mortality tables – that is, comprehensive in terms of cause of death, age at death and sex – were published in *Annual Reports* by the General Register Office (GRO) for the country as a whole, for London, and, on occasion, for counties (Humphries, 1991). The national tables have already been employed to provide summaries of sex differential mortality in England and Wales (Lopez, 1983; United Nations Secretariat, 1988; Tabutin and Willems, 1998). Less commonly used are the *Decennial Supplements*, which offer a geographical perspective on mortality analysis with data for 600 or so registration districts (Ryan Johansson, 1977; Kearns, 1991). Cause of, and age at, death are less detailed in the *Supplements* than the *Annual Reports*. Furthermore, many of the cause-of-death groupings used in the *Decennial Supplement* for 1851–60 had been amended or abandoned by the

twentieth century, as medical knowledge accumulated, diagnostic fashions came and went, and old nosological classifications were superseded. For these reasons, a proportion of cause-of-death categorisations in 1901–10 are not directly comparable to those of 1851–60, and caution must be exercised in interpretation (Hardy, 1994; Woods and Shelton, 1997). Although many diseases were readily distinguishable throughout the period (smallpox and whooping cough might be cited as examples), reliance here is placed on broad groupings of conditions rather than individual causes of death (Caselli, 1991). Analysis of sex differentials is further hampered by the fact that for 1871–80, 1881–90 and 1891–1900, information is not provided separately for males and females. However, the demographer of Victorian England and Wales is able to scrutinise district-level sex differentials in mortality by cause in 1851–60, 1861–70 and 1901–10 and the changes that took place between any of these three decades.

IMPROVING MORTALITY AND WIDENING SEX INEQUALITY

The broad course of mortality transition in Victorian England and Wales is now well known. During the first three-quarters of the nineteenth century, national life expectancy at birth (e_0) hovered around 40 years. It began to increase in the final quarter, and on the eve of the First World War it exceeded 50 years, a precipitous downward trend in mortality that has continued to the present (Woods, 2000: Fig. 1.1 and Table 9.3). The subject of intense contemporary scrutiny from epidemiologists, social scientists, doctors and civil servants, the components of mortality decline in the Victorian era have proved no less fascinating for researchers in the recent past (McKeown, 1976; Woods and Woodward, 1984; Szreter, 1988). It is important to note, however, that all sections of society did not benefit in the same way from these positive developments, and it is now accepted that inequalities in death can worsen during the early and middle stages of rapid overall mortality improvement. The interpretation favoured by Woods is that 'as rapid change occurs – decline in mortality, for instance – differentials are bound to increase
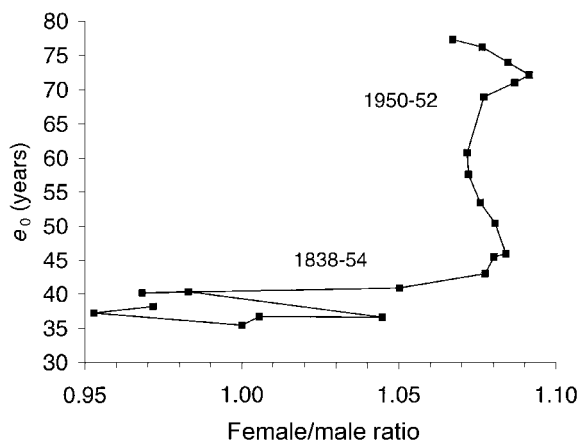
Figure 2. The relationship between total life expectation at birth (e_0, years) and the ratio of female/male total life expectation at birth, England and Wales, 1625–49 to 1996–98.
Sources: Wrigley *et al.* (1997) Table 6.27, p. 308 (1625–1799); Office for National Statistics (2000) Table 23, p. 114 (1800–1998).
Note: Time periods are not equal. Dots represent the following: 1625–49 to 1775–99; 1838–54; 1871–80...1901–10; 1910–12...1990–92 (excluding 1940–42); and 1996–98.

as leading and lagging occupations or areas move into the phase of transition at different speeds' (Woods, 2000: 221).

Woods's evidence on the middle stages of mortality transition does not provide conclusive proof that socio-economic and/or spatial health inequalities widened during the initial period of downward mortality (on occupations, see Woods, 2000: 234, Table 6; on urban/rural contrasts, see 365, Table 9.3; and 367, Table 9.4). On the other hand, the rapid improvement in the national level of life expectancy in England and Wales during the second half of the nineteenth century conceals a marked aggravation of the sex differential. The third English Life Table showed that a girl born in 1838–54 could expect to live two years longer than an infant boy.[3] This relatively small discrepancy had increased to three and a half years by the 1880s and had doubled to four years by 1901–10. This pattern has been explained by the declining significance of some epidemic infectious diseases – which tended to be more fatal for girls and women than boys and men – and the concentration of

mortality in older age groups, where improvement was much greater for females than males (United Nations Secretariat, 1988: 90–91; Pinnelli and Mancini, 1999: 350–51). These shifts characterise the middle stages of the epidemiological transition (Omran, 1971). Differences between males and females in the premodern era were variable and inconsistent (Wrigley *et al.*, 1997: 306–8), but as Fig. 2 demonstrates, regular female advantage in e_0 was largely a product of the Victorian era (Martin, 1951: 288).[4] The discrepancy between the sexes gradually narrowed during the first half of the twentieth century, widened between the 1950s and 1970s, and has lessened over the last 30 years or so. A similar pattern has been observed for other industrialised Western nations and is closely linked to sex differentials in tobacco-related mortality. Also, larger improvements in other cause-specific mortality among men than women have been responsible, particularly heart diseases, accidents and violence (excluding suicide), notably in the 55–75 age range (Trovato and Lalu, 1998: 18). It remains to be seen whether the most recent trend will continue, assuming that life expectancy continues to rise; for the moment, however, Fig. 2 confirms that a key characteristic of the steep mortality improvement that took place between 1850 and 1900 in England and Wales was a relative and significant deterioration in the health position of males, and that further deterioration took place in the 1950s and 1960s.

AGE-SPECIFIC MORTALITY AND LIFE EXPECTANCY AT BIRTH IN LONDON

The second half of the nineteenth century was an important period in the history of health in the British capital. Life expectation at birth increased from 36.1 years in 1851–60 to 49.0 years in 1901–10. Notwithstanding a phase of stagnation, if not regression, between the 1830s and 1860s, this represented the continuation of a downward trend that had its origins in the mid-eighteenth century and which culminated in London attaining a very creditable position in the urban mortality tables published by the GRO from the mid-nineteenth century onwards (Table 1).

Some basic interactions between age at

Table 1. Overall life expectancy at birth (years),
London, 1730 to 1910.

1730s	18.2	1820s	34.4
1740s	17.6	1830s	36.9
1750s	20.1	1840s	36.7
1760s	20.5	1850s	36.1
1770s	21.6	1860s	36.4
1780s	25.5	1870s	39.1
1790s	27.5	1880s	41.4
1800s	28.0	1890s	42.6
1810s	32.4	1900s	49.0

Sources: Landers, 1993: 171, Table 5.4 (1730s to 1820s),
reproduced by permission of Cambridge University Press);
Woods, 2000: 369, Table 9.4 (1830s and 1840s, reproduced
by permission of Cambridge University Press); remaining
values calculated from the relevant Registrar-General's
Decennial Supplements.

death and the sex differential in mortality can
be elucidated for London in the Victorian
period through an analysis of age-specific
mortality rates and the summary indicator,
life expectation at birth. Over the second half
of the nineteenth century, death rates declined
in all age groups for both sexes, but the
generally higher rates of mid-nineteenth cen-
tury male mortality persisted (Fig. 3). For all
but two age groups, 5–9 and 10–14 year olds,
the discrepancy between males and females
was exacerbated. This point is underlined by
the ratio of male/female mortality in the two
decades, where a ratio above one indicates

excess male mortality (Fig. 4). One instance of
excess female mortality in 1851–60 at ages
above 75 was reversed by 1901–10. We might
also observe that a discrepancy between boy
and girl toddlers (ages 1–4 years) appeared;
and that by 1901–10, excess male mortality was
far less pronounced in adolescence than in
most other age ranges.

The overwhelming message communicated
by Figs 3 and 4 is that while the mortality
transition spelt good news for metropolitan
males and females of all ages, the benefits were
biased heavily towards adult women.
Although this reflected national trends, not to
mention experience in other parts of Europe
(Hart, 1989; Vallin, 1989; Caselli, 1996; van
Poppel, 2000), it is probable that working-class
women were fortunate in this respect (for
Sweden more recently, see Hemström, 1999).
Corroborating evidence comes from the Uni-
ted States, where life expectancy in early
adulthood (e_{20}) for the nineteenth century
shows that excess male mortality was common
in urban areas (Haines, 2001). Urban females
frequently enjoyed lower mortality rates than
their country-dwelling sisters (Vögele, 1998:
49). Comparing age- and sex-specific mortality
rates in contrasting environments of northeast
Cornwall (rural Stratton and industrial Re-
druth), Ryan Johansson made the point that in
1851–60 ' … for girls and women it seemed to
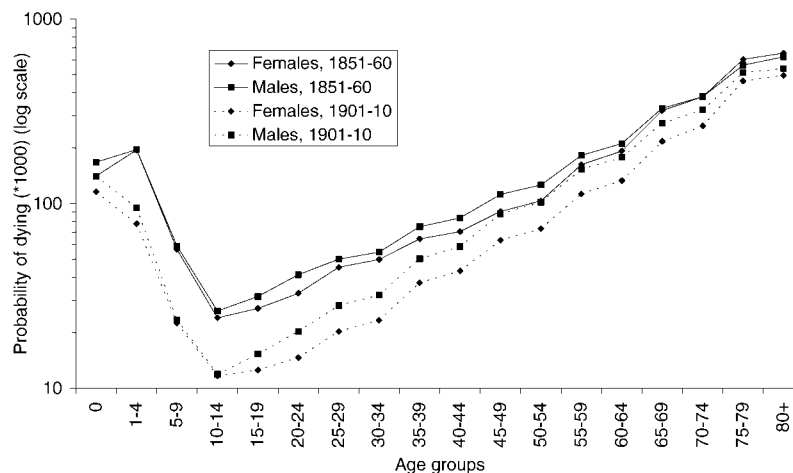make no difference whether they lived in a

Figure 3. Age-specific mortality rates ($1000_{n}q_{x}$), males and females, London, 1851–60 and 1901–10.
Sources: Calculated from the relevant Registrar-General's *Decennial Supplements*.
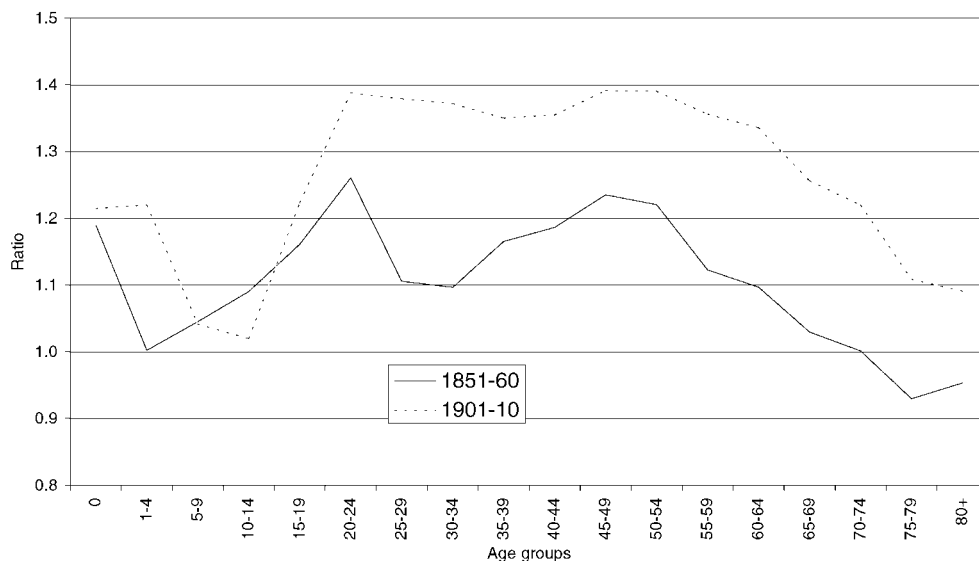
Figure 4. The ratio of male/female mortality (1000_nq_x), London, 1851–60 and 1901–10.
Sources: Calculated from the relevant Registrar-General's *Decennial Supplements*.

healthy rural district or a polluted and unsanitary urban one' (Ryan Johansson, 1977: 177). Of course, the validity of this observation depends on which urban and rural districts are chosen for comparison, but it would appear unquestionable that metropolitan women were particularly favoured. Kearns has shown that, in the second half of the nineteenth century, the metropolitan/rural female mortality ratio was less severe at all age groups than was the ratio for other urban-living females when compared with their rural counterparts (Kearns, 1991: Fig. 1.3). On the evidence of higher young adult female mortality rates in predominantly rural counties surrounding the metropolis, it was the belief of some contemporaries that London's apparent advantage was an artefact. The suggestion was that many young women had been attracted by the availability of wage-labour in London, only to contract a fatal illness in the capital and subsequently return to their rural birthplaces prior to death (Welton, 1875). It was argued that metropolitan early adult female death rates were artificially reduced while corresponding rates in the surrounding countryside were inflated. As Ryan Johansson pointed out, there was in fact little empirical evidence to support this theory. Indeed, a converse theory

has been suggested that the propensity of the capital to attract large numbers of relatively healthy young adult women from the surrounding countryside was reflected in the comparatively high male to female mortality ratio (Martin, 1951: 289). In addition, recent historical research suggests that the rigours of rural life did in fact place women at a health disadvantage, at least compared with their rural male counterparts (although cf. Guha, 1994; Szreter, 1994; see also Martin, 1956; Shorter, 1991: 237–40). Expected to assist with men's work outside the home, women were also predominantly responsible for the domestic sphere, including the rearing of children.

With this focus on the urban/rural dichotomy, it has gone largely unnoticed that because of variations in occupational structure, class division and physical environment, intra-urban comparisons of male and female mortality differentials might yield insights into gendered social roles of equal importance. One demographic starting point is to summarise transformations in the age-specific mortality rate by calculating life expectancy at birth, which increased for males in London from 34.8 to 46.2 years over the period 1851–60 to 1901–10. The corresponding figures for females were 37.4 and 51.7 years. For the whole metropolitan

Table 2. Male and female life expectancy at birth, London and metropolitan sectors, 1851 to 1910.

	London	Contracting core	Stable core	Inner suburbs	Outer suburbs
Males, 1851–60	34.82	33.55	33.98	33.96	37.21
Females, 1851–60	37.37	35.87	35.68	36.64	40.48
Males, 1861–70	34.33	32.98	32.22	34.00	35.17
Females, 1861–70	38.34	35.89	35.30	38.37	39.86
All, 1871–80	39.13	36.87	37.25	39.12	42.59
All, 1881–90	41.42	39.57	38.80	42.20	45.79
All, 1891–1900	42.61	40.02	40.42	43.40	46.40
Males, 1901–10	46.15	45.72	43.16	46.16	50.38
Females, 1901–10	51.70	51.75	47.80	52.11	55.17

Source: Calculated from the relevant Registrar-General's *Decennial Supplements*.

area, the rather small gap (2.6 years) between the sexes in 1851–60 reached 4.0 years in the next decade and was more than 5.5 years by 1901–10 (Table 2). In other words, metropolitan females gained an average of one half year in life expectation at birth over metropolitan males for every passing decade.

There were, of course, important spatial variations within London. While it is possible to produce measures of life expectancy for each metropolitan district, the results are difficult to summarise in a meaningful way. This is only partly due to the internal complexity of mortality change within London. Underwritten by spatially demarcated and gendered occupational structures, and extensive suburbanisation, class segregation became a deeply ingrained aspect of metropolitan society. As the spatial polarisation of social classes within the metropolis intensified, so too did experiences of ill-health and death, reflecting nutritional levels, working conditions, and access to affordable, good-quality housing and health care. However, problems of even greater magnitude occur because the mortality rate of many individual districts in the capital was distorted by deaths occurring to patients in hospitals and other institutions such as workhouse infirmaries (Mooney *et al.*, 1999; Mooney, 2000). Districts containing such institutions had overstated mortality rates, and not until 1911 were such deaths 'returned' to the mortality schedules of the district in which the patient last resided. In order to simplify these intricate processes, districts in the capital have been classified into

one of four sectors on a decade-by-decade basis. Districts are divided into groups according to the level of population density at the end of a decade (for example, 1861 for 1851–60) and the annual compound rate of housing growth (measured as the number of inhabited buildings per acre) over that decade (Table 3). A district was deemed to belong to the 'contracting core' if its population density was falling compared with the previous decade and/or if it had a negative or nominally positive rate of housing growth. The 'stable core' comprises metropolitan districts characterised by population densities that remained relatively high – in the region of 100 to 200 persons per acre – yet possessed either a contracting housing stock or one which failed to increase by an average of more than 1% a year. Districts belonging to the 'inner suburbs' usually displayed an intermediate population density of approximately 50 to 100 persons per acre and a rate of housing growth which was always positive during the second half of the nineteenth century and the first decade of the twentieth century. Finally, 'outer suburban' districts consistently sustained a positive rate of building growth that in some decades exceeded 5% per annum. By 1901–10, none of the districts in this sector had population densities in excess of 45 persons per acre. One advantage of this classification is that districts can 'move' to a different sector between decades. As a result, and unlike the Registrar-General's favoured subdivision of the capital into north, west, east, south and central zones, the procedure groups districts experiencing

Table 3. Selected characteristics of metropolitan sectors, 1851–60 and 1901–10.

Decade	Sector and composition	Average population density[a]	Average annual housing change[c]	Average annual population change[b]	Persons per house[a]	Fertility[d]	Children per house[a]
Contracting core							
1851–60	Holborn, London City, Shoreditch	193	0.03	0.14	8.81	215	2.59
1901–10	Bermondsey, Islington, Lambeth, London City, Marylebone, Pancras, Westminster	79	−0.63	−0.60	8.69	87	2.11
Stable core							
1851–60	Bethnal Green, Southwark, Stepney	140	0.82	1.09	7.43	251	2.41
1901–10	Bethnal Green, Holborn, Shoreditch, Southwark, Stepney	160	−1.01	−0.74	10.10	131	3.12
Inner suburbs							
1851–60	Bermondsey, Lambeth, Marylebone, Pancras, Westminster	69	0.99	1.06	8.67	198	2.45
1901–10	Camberwell, Hackney, Kensington, Poplar	70	−0.05	0.08	7.56	84	1.96
Outer suburbs							
1851–60	Camberwell, Greenwich, Hackney, Hampstead, Islington, Kensington, Poplar, Wandsworth	16	3.45	3.89	6.94	215	2.12
1901–10	Greenwich, Hampstead, Wandsworth	31	1.44	1.20	6.36	86	1.72

[a] 1851–60 calculated from 1861 census; 1901–10 calculated from 1911 census. Population density in persons per acre; fertility measured as the average number of births per 1000 females aged 15–49.
[b] Average annual compound rate of change, all persons (%).
[c] Average annual compound rate of change in the number of inhabited buildings (%).
[d] 1851–60 calculated from 1861 census; 1901–10 calculated from 1911 census.

Table 4. Occupational location quotients, metropolitan sectors, 1901.

	Males				Females			
	Contracting core	Stable core	Inner suburbs	Outer suburbs	Contracting core	Stable core	Inner suburbs	Outer suburbs
Agriculture (incl. fishing)	0.81	0.27	1.00	2.08	1.05	0.94	0.90	1.15
Administration	1.14	0.64	0.97	1.22	0.98	0.57	1.15	1.24
Defence	1.13	0.30	0.55	2.18	N/A	N/A	N/A	N/A
Professional and teaching	1.06	0.46	1.17	1.26	0.94	0.60	1.06	1.43
Entertainment and sport	1.21	0.60	1.06	1.01	1.11	0.56	1.04	1.21
Commerce, finance	0.92	0.33	1.18	1.57	1.01	0.46	1.01	1.56
Clerical	1.03	0.54	1.19	1.19	1.12	0.53	1.06	1.19
Retail and distribution	1.00	0.54	1.18	1.23	1.12	0.78	1.07	0.89
Personal service	1.51	0.53	1.01	0.70	1.00	0.55	1.14	1.26
Transport and storage	1.06	1.16	0.99	0.76	1.14	1.22	0.80	0.85
Building industry	0.92	0.68	1.19	1.21	1.36	0.85	0.72	1.00
Wood and furniture	0.82	1.86	0.90	0.50	0.96	2.05	0.70	0.46
Metal and engineering	0.83	0.89	0.94	1.46	1.39	1.37	0.50	0.76
Shipbuilding	0.48	0.66	1.58	1.36	0.48	0.80	1.98	0.44
Precision industry	1.30	0.82	0.85	0.94	1.35	1.31	0.65	0.64
Printing and paper	1.06	1.43	0.89	0.60	0.91	2.55	0.53	0.31
Leather and hides	1.14	1.44	0.84	0.53	0.87	2.65	0.59	0.16
Food and drink manufacture	1.06	1.05	0.99	0.87	1.06	1.44	0.84	0.69
Textile manufacture	1.12	0.94	1.03	0.84	0.99	1.22	0.92	0.92
Clothing trade	0.90	1.87	0.75	0.60	1.02	1.34	0.93	0.71
Boot and shoe trade	0.62	2.09	0.96	0.47	0.32	2.96	1.01	0.09
Chemicals, allied trades	0.88	0.94	0.98	1.28	0.92	1.30	1.08	0.66
Utilities	0.82	0.68	1.17	1.38	1.33	0.92	1.22	0.10
Misc. other	0.81	1.29	0.83	1.22	0.93	1.77	0.76	0.73
Misc. labour	0.79	1.10	0.98	1.25	0.94	2.09	0.57	0.70
Total working population	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00

Source: 1901 Census for England and Wales.
N/A: Not applicable.
The location quotient demonstrates the degree of concentration of a particular occupation in a particular sector. First, the distribution of the working population in each sector of London is calculated. Next, the distribution of a certain occupation or socio-economic group in each sector is calculated. The location quotient is arrived at by dividing the second percentage by the first. A location quotient equal to 1 indicates that the distribution of an occupation is co-extensive with the distribution of the working population as a whole. Values of more or less than 1 demonstrate the level of concentration or otherwise of an occupation in a particular sector (adapted from Stedman-Jones, 1984: 370–71, Table 7A).
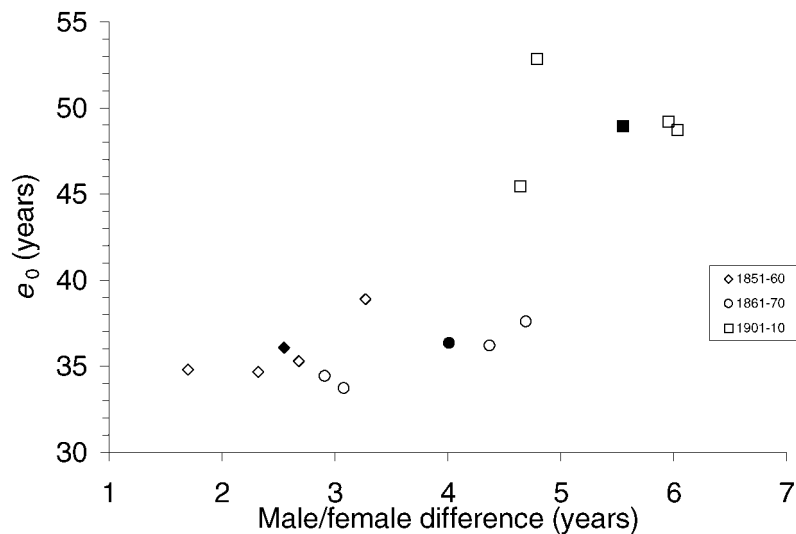
Figure 5. Total life expectation at birth (e_0, years) and the male/female difference, London and London sectors, for 1851–60, 1861–70 and 1901–10.
Sources: Calculated from the relevant Registrar-General's *Decennial Supplements*.

comparable, period-specific socio-environmental conditions, irres-pective of location on the metropolitan map. Furthermore, the problem caused by the 'transfer' of deaths between districts due to the presence of institutions is reduced by grouping individual districts into sectors.

A summary of the occupational structure in 1901 provides an indication of the types of socio-economic environment represented by each sector (Table 4). Precision industries and manufacturing or 'sweated' trades – wood and furniture, printing and paper, leather and hides, food and drink, textiles, clothes and boots and shoes – tended to be strongly represented for either males or females in the two core sectors, but particularly for females in the stable core. And while many professional and commercial occupations predominated in the outer suburbs, the ascendancy of central London as the hub of commerce and entertainment is reflected by above-average location quotients for men in the contracting core in professional occupations related to administration, defence and teaching, not to mention entertainment, sport, and personal service (a high proportion of whom were employed in hotels, lodging and eating-houses). All of these forms of employment were heavily under-represented for both

males and females in the stable core. Professional and teaching occupations and personal service dominated the female occupational structure in the outer suburbs.

Variations in sectoral e_0 can be found in Table 2.[5] As might be expected, the two suburban sectors generally have the better life expectancies for both males and females (with the exception of inner suburban males in 1851–60). By the end of the Victorian era, the stable core was established as the least favoured metropolitan sector in terms of male and female health status. The close connection between the extent of sex inequality and the level of overall mortality improvement is confirmed by data for the metropolitan sectors, captured by Fig. 5 (in Fig. 5 the symbols for London Division have been filled). In the first two decades, when metropolitan life expectation at birth in the sectors spanned 33 to 39 years, the registered difference between males and females ranged from 1.7 years in the stable core in 1861–70 to 3.3 years in the outer suburbs in 1851–60. In 1901–10, the discrepancy between the sexes had increased in accordance with the rise in life expectation at birth, although the outer suburbs had a much lower sex differential than would be expected, with an e_0 of almost 53 years. The next section

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Table 5. Male and female age-specific death rates per 100,000 population by cause, London, 1851–60 and 1901–10.

Disease group	All ages	0-1	1-4	5-9	10-14	15-19	20-24	25-34	35-44	45-54	55-64	65-74	75+
Gastro-intestinal													
Males 1851-60	179	1948	305	58	27	26	34	50	69	97	164	333	729
Males 1901-10	81	1927	123	6	6	11	14	15	14	12	17	36	126
Females 1851-60	161	1715	319	52	22	22	31	52	76	97	179	369	847
Females 1901-10	61	1638	103	6	6	7	7	8	9	8	13	33	108
Infectious													
Males 1851-60	685	3911	2671	580	174	152	150	141	172	236	344	526	846
Males 1901-10	170	1522	1026	151	39	26	21	19	18	22	20	20	15
Females 1851-60	582	3497	2926	555	173	133	107	104	121	147	237	422	701
Females 1901-10	146	1425	898	163	41	23	16	14	14	12	13	18	23
Respiratory													
Males 1851-60	490	3174	983	70	21	37	70	94	201	462	1065	2196	3767
Males 1901-10	324	2511	567	39	15	28	38	71	162	315	695	1638	3881
Females 1851-60	417	2505	1047	75	21	29	36	63	139	341	947	2145	3996
Females 1901-10	275	2069	465	39	16	18	21	37	91	200	529	1412	3588
Respiratory tuberculosis													
Males 1851-60	360	185	131	60	61	209	369	457	603	618	519	360	198
Males 1901-10	195	59	46	16	16	79	161	234	368	442	389	276	114
Females 1851-60	284	170	149	70	88	212	285	410	448	363	278	187	118
Females 1901-10	105	53	36	20	32	79	96	133	189	180	142	104	60

Sources: Calculated from the relevant Registrar-General's *Decennial Supplements*.
Note: Composition of disease groups can be read from Table 6.

Table 5. Continued.

Disease group	All ages	0–1	1–4	5–9	10–14	15–19	20–24	25–34	35–44	45–54	55–64	65–74	75+
Neoplasms													
Males 1851–60	26	4	3	2	1	3	4	9	24	62	132	204	216
Males 1901–10	98	5	7	4	4	6	7	16	59	226	526	834	951
Females 1851–60	66	3	5	1	1	2	5	23	93	194	293	363	351
Females 1901–10	113	3	4	3	3	4	5	22	103	270	500	720	863
Violent deaths													
Males 1851–60	119	366	108	58	60	77	66	80	118	151	194	226	380
Males 1901–10	89	501	87	39	25	31	44	57	86	131	169	196	381
Females 1851–60	51	336	89	35	14	18	15	20	27	40	65	117	390
Females 1901–10	43	467	65	23	8	9	12	16	24	36	51	95	303
Childbirth													
Females 1851–60	35	0	0	0	0	13	51	89	79	5	0	0	0
Females 1901–10	16	0	0	0	0	4	25	44	36	2	0	0	0
Other causes													
Males 1851–60	950	7203	807	158	114	131	148	228	463	877	1884	4763	12,787
Males 1901–10	777	7537	387	107	109	128	121	185	409	830	1753	3854	10,110
Females 1851–60	927	5889	812	147	103	134	177	303	490	806	1834	4767	15,089
Females 1901–10	686	5922	290	107	108	113	134	201	392	690	1335	2950	8685
All causes													
Males 1851–60	2808	16,792	5008	986	459	636	840	1058	1651	2502	4301	8608	18,924
Males 1901–10	1735	14,061	2244	361	213	309	407	597	1116	1978	3570	6854	15,578
Females 1851–60	2488	14,115	5347	936	421	551	655	974	1394	1989	3833	8370	21,492
Females 1901–10	1429	11,576	1861	361	214	253	291	432	822	1397	2584	5333	13,631

Sources: Calculated from the relevant Registrar-General's *Decennial Supplements*.
Note: Composition of disease groups can be read from Table 6.

investigates the causes of death that were responsible for these metropolitan variations.

CAUSES OF DEATH AND SEX DIFFERENTIAL MORTALITY IN VICTORIAN LONDON

London's rise to prominence in the British urban mortality league tables was probably underpinned by a dramatic reduction of infant mortality and the attenuation of endemic smallpox and fever (probably typhus rather than typhoid) towards the end of the 'long' eighteenth century (Landers, 1993; Scott and Duncan, 1998). This was augmented in the later nineteenth and early twentieth centuries by substantial reductions in infectious childhood diseases, such as scarlet fever and whooping cough (Hardy, 1993). In common with many other towns and cities in England and Wales, London also experienced substantial reductions in its rate of respiratory tuberculosis mortality and other diseases of the respiratory system (Kearns, 1993). As infectious diseases gradually relinquished their grip on the Victorian population, their domination of the mortality schedules was challenged by ailments such as neoplasms and a range of heart conditions (Omran, 1971). This well-known narrative concerning the epidemiological transition in mid- and late-nineteenth century England and Wales conceals more fine detail than it reveals broad trends. Firstly, the timing of the decline for some diseases was earlier than for others. Respiratory tuberculosis began to decline from the 1850s, scarlet fever from the 1860s, whooping cough in the 1880s and measles only in the early twentieth century (McKeown, 1976). Secondly, the transformation of the epidemiological panorama was draped over the landscape of England and Wales like a patchwork quilt. Modifications in the fatality of one disease – either because of declining virulence, changed human behaviour or environmental factors – had varied implications for age- and sex-differential mortality, and were frequently place-specific; modifications to another disease might produce an opposing set of impacts. Declines in respiratory tuberculosis, for example, were beneficial for young adults both in urban areas and the countryside. In contrast, a

lessening in the incidence of water- and food-borne gastro-intestinal diseases such as diarrhoea and dysentery had the greatest impact on the survival chances of babies born into an urban environment (Woods and Shelton, 1997).

Encapsulating some key features of this transition for metropolitan males and females, Table 5 puts causes of death into broad categories in order to facilitate interpretation and to ensure comparability across decades. Declining rates of mortality across the age range are common in both sexes for all causes of death, with the noteworthy exception of neoplasms.[6] Most significant improvements for gastro-intestinal diseases occurred after the first year of life. Infectious disease mortality declined by at least 60% across the age range. With the exception of the oldest men, all groups enjoyed rates of respiratory diseases that were at least 20% lower in 1901–10 than they had been in 1851–60. More substantial gains than this were made across all age groups for respiratory tuberculosis. As a final example of improvement, deaths attributable to violence (a category including accidents, murder and suicide) were greatly reduced for both sexes at almost all ages, the notable exception here being in the most vulnerable stage of life, infancy. Indeed, it was in this age group that the other exception from this general process of advancement is found, with a metropolitan reversal of fortune experienced in infancy for 'other causes'.

More light can be shed on the nature of sex differential mortality in the capital by calculating a sex ratio for these groups of causes (see United Nations Secretariat, 1988: 79–80). Disparities that already existed in 1851–60 between men and women were most forcibly exacerbated by widening gaps in mortality from respiratory tuberculosis (Fig. 6d) and, to a much lesser extent, respiratory diseases (Fig. 6c). Gastro-intestinal diseases also reinforced this trend, but at much lower rates of absolute mortality (Fig. 6a). Cancer provides an interesting case in that the rise in mortality erased the female advantage in the over-45s: male death rates in the age groups 45–54, 55–64, 65–74 and over-75 increased by 265, 299, 309 and 340% respectively (Fig. 6e). Figure 6 also shows that the frightening increase in deaths inflicted
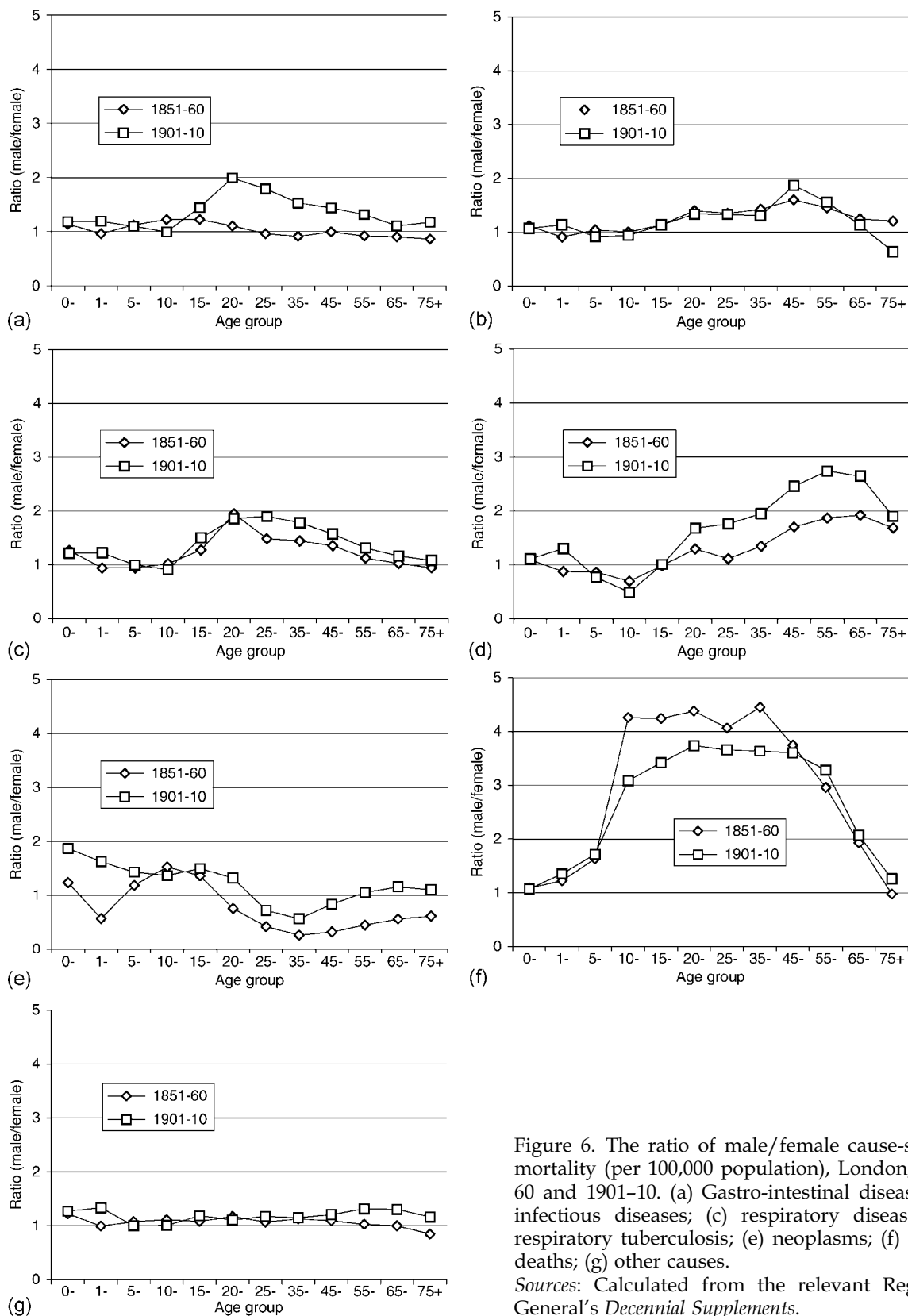
Figure 6. The ratio of male/female cause-specific mortality (per 100,000 population), London, 1851–60 and 1901–10. (a) Gastro-intestinal diseases; (b) infectious diseases; (c) respiratory diseases; (d) respiratory tuberculosis; (e) neoplasms; (f) violent deaths; (g) other causes.

Sources: Calculated from the relevant Registrar-General's *Decennial Supplements*.

on infants as a result of violence affected baby boys and baby girls almost equally. But after adolescence there was, by and large, a reduced level of risk from these causes. In correspondence with the somewhat unchanged ratio in infancy from violent deaths, Fig. 6 also shows that only minor alterations occurred in the ratio in the first year of life for most groups of causes. With the notable exception of neoplasms, any small change that did take place tended to work to the advantage of baby boys, whereas the reverse was true in early childhood.

DECOMPOSING THE SEX DIFFERENTIAL IN LIFE EXPECTATION AT BIRTH

The three forms of demographic measurement used so far in this paper – life expectation at birth, age-specific death rates and mortality ratios – do not complement one another particularly well. As we have already seen, an approach that incorporates comparisons of age-specific death rates can provide a wealth of detail and insight into the nature and form of sex differential mortality in Victorian London and elsewhere (Ryan Johansson, 1977; Humphries, 1991). Calculated from such rates, mortality ratios are also valuable epidemiological tools for highlighting anomalous age-specific mortalities between the sexes (Kearns, 1991). But mortality ratios are limited in that they obscure the importance of the *level* of mortality and absolute differences between the sexes, which may vary in time and among places (Preston, 1976; United Nations Secretariat, 1988: 66; United Nations Secretariat, 1998: 92–93). The use by demographers and epidemiologists of life expectation at birth has also been criticised by a variety of commentators. Problems arise with life expectation at birth because it fails to reveal the complexities of the age-specific mortalities from which it is calculated: as Ryan Johansson ruefully observes, 'life expectancy at birth … compresses too much valuable information' (Ryan Johansson, 1991: 139; Humphries, 1991: 458). This is a major shortcoming for mortality studies when the focus of attention – cause of death or sex differential – varies so widely between age groups. More positively, however, life expectation at birth has the advantage of being unaffected by the age distribution of the population, thus making it eminently appropriate for the kinds of spatial and temporal comparison that lie at the heart of this paper (United Nations Secretariat, 1988: 66). Furthermore, it is possible to decompose the sex differential in life expectation at birth in a way that enumerates the contribution made to that differential either by each cause of death, or by each age group, or both cause and age group together (Pollard, 1982, 1988; Arriaga, 1984).[7] As such, the decomposition of the expectation of life is an increasingly favoured method by which to represent shifting mortality regimes in both historical and contemporary populations (Caselli, 1991; Goldman and Takahashi, 1996; Myers, 1996; van Poppel *et al.*, 1996; Trovato and Lalu, 1998).

Infant mortality comprised almost half (44%) of the observed metropolitan sex differential in e_0 in London in 1851–60 (Table 6, upper panel). This impressive proportion is in keeping with what is now known about England and Wales and the Netherlands in the second half of the nineteenth century. Yet it is a low percentage contribution when compared with Italy at the same time (on the Netherlands, see van Poppel *et al.*, 1996; on England and Wales and Italy, see United Nations Secretariat, 1988: 92–6). Discrepancy in London between the sexes in this age group was primarily driven by the ambiguous 'other causes' category (incorporating the important, if indistinct, medical diagnoses of premature birth, atrophy, debility and wasting, and diseases of the brain, which, in turn, included convulsions), and to a much reduced extent by respiratory diseases, infectious diseases and gastro-intestinal ailments. The mortality disadvantage for almost all causes of death in infancy was heavily skewed towards baby boys. The exception was whooping cough, but the female disadvantage associated with this disease was even more marked in early childhood (1–4 years). Overall, this age group contributed less than 1% to the sex differential in e_0. While sex differentials in mortality in early childhood were not necessarily strong, excess female mortality in both England and Wales and France was detectable at three years of age towards the end of the nineteenth century (Anderson, 1990: 18; Tabutin and Willems, 1998). The evidence for mid-

Table 6. Disease- and age-group percentage contributions to the sex differential in life expectancy at birth in London, 1851–60 and 1901–10.

	0	1–4	5–14	15–29	30–49	50–69	70+	All ages
1851–60								
Cholera, diarrhoea and dysentery	4.0	0.4	0.7	0.3	−0.4	−0.3	−0.1	4.5
Gastro-intestinal diseases	**4.0**	**0.4**	**0.7**	**0.3**	**−0.4**	**−0.3**	**−0.1**	**4.5**
Smallpox	0.4	−0.2	0.5	1.0	0.4	0.1	0.0	2.2
Measles	0.6	−0.5	−0.1	−0.0	−0.0	0.0	0.0	−0.1
Scarlatina	0.8	1.9	1.1	−0.1	−0.1	−0.1	0.0	3.7
Diphtheria	0.2	−0.2	−0.4	−0.1	0.0	0.0	0.0	−0.4
Whooping cough	−1.0	−9.5	−1.6	−0.0	0.0	0.0	0.0	−12.1
Typhus, typhoid	0.1	−0.9	−0.2	1.3	1.0	0.7	0.1	2.1
Other zymotic	1.7	0.4	0.1	1.1	3.4	2.0	0.2	8.8
Scrofula and Tabes Mesenterica	1.4	1.1	1.0	0.7	0.1	0.0	0.0	4.4
Hydrocephalus	3.0	4.2	1.0	0.1	0.0	0.0	0.0	8.2
Infectious diseases	**7.2**	**−3.7**	**1.4**	**4.0**	**4.8**	**2.7**	**0.3**	**16.8**
Diseases of the lungs	10.8	0.3	−0.0	2.9	5.5	3.4	−0.1	22.7
Respiratory diseases	**10.8**	**0.3**	**−0.0**	**2.9**	**5.5**	**3.4**	**−0.1**	**22.7**
Phthisis	0.3	−0.5	−2.1	5.0	12.5	7.0	0.4	22.4
Respiratory tuberculosis	**0.3**	**−0.5**	**−2.1**	**5.0**	**12.5**	**7.0**	**0.4**	**22.4**
Cancer	0.0	−0.1	0.0	−0.5	−5.5	−4.4	−0.4	−10.8
Neoplasms	**0.0**	**−0.1**	**0.0**	**−0.5**	**−5.5**	**−4.4**	**−0.4**	**−10.8**
Violent deaths	0.5	1.4	4.2	7.0	7.7	3.5	0.2	24.6
Violent deaths	**0.5**	**1.4**	**4.2**	**7.0**	**7.7**	**3.5**	**0.2**	**24.6**
Childbirth	0.0	0.0	−0.1	−5.7	−6.5	−0.1	0.0	−12.3
Childbirth	**0.0**	**0.0**	**−0.1**	**−5.7**	**−6.5**	**−0.1**	**0.0**	**−12.3**
Diseases of brain	9.1	1.8	0.7	1.1	3.3	1.8	0.2	18.0
Diseases of heart and dropsy	0.1	0.3	−0.3	0.3	1.1	−0.5	−0.1	0.9
Diseases of stomach	2.2	0.2	0.4	−0.3	−1.4	−1.3	−0.3	−0.5
Diseases of kidneys	0.0	0.2	0.4	0.9	2.0	2.5	0.9	7.0
Diseases of generative organs	−0.0	−0.0	−0.0	−0.7	−2.2	−1.3	−0.2	−4.4
Diseases of joints	−0.0	0.0	0.4	0.4	0.3	0.2	0.0	1.3
Diseases of skin	0.1	−0.1	−0.0	0.1	0.0	0.1	0.0	0.2
Other causes	10.0	0.4	−0.0	0.3	0.6	0.3	−1.9	9.6
Other causes	**21.5**	**2.8**	**1.6**	**2.1**	**3.7**	**1.8**	**−1.4**	**32.1**
All causes	**44**	**1**	**6**	**15**	**22**	**14**	**−1**	**100**
1901–10								
Enteric fever	0.0	−0.0	0.0	0.4	0.3	0.0	−0.0	0.8
Diarrhoea and dysentery	2.9	0.8	0.0	0.0	0.1	0.1	0.0	4.0
Gastro-intestinal diseases	**2.9**	**0.8**	**0.0**	**0.4**	**0.4**	**0.1**	**0.0**	**4.8**
Smallpox	−0.0	−0.0	−0.0	−0.0	0.0	0.0	0.0	−0.0
Measles	0.6	3.0	0.0	0.0	−0.0	0.0	0.0	3.6
Scarlet fever	0.0	0.7	0.1	0.0	−0.0	0.0	0.0	0.8
Typhus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Whooping cough	−0.7	−1.1	−0.3	−0.0	0.0	0.0	0.0	−2.1
Diphtheria	0.1	0.7	−0.3	0.0	−0.0	−0.0	0.0	0.6
Tuberculous meningitis	0.2	1.1	0.1	0.1	0.1	0.0	0.0	1.5
Tuberculous peritonitis	0.3	0.3	−0.0	−0.0	0.0	0.0	0.0	0.6
Tabes Mesenterica	0.3	0.2	−0.0	−0.0	0.0	0.0	0.0	0.5
Other tuberculosis	0.3	0.5	0.1	0.3	0.3	0.2	−0.0	1.7
Infectious diseases	**1.1**	**5.4**	**−0.3**	**0.4**	**0.4**	**0.2**	**0.0**	**7.2**

Table 6. Continued.

	0	1–4	5–14	15–29	30–49	50–69	70+	All ages
Influenza	0.1	0.1	−0.0	0.2	0.3	0.1	−0.1	0.7
Pneumonia	2.6	3.4	0.1	1.3	3.6	2.8	0.7	14.5
Bronchitis	1.8	0.7	0.0	0.1	0.4	1.0	0.4	4.5
Respiratory diseases	**4.5**	**4.2**	**0.1**	**1.6**	**4.3**	**3.9**	**1.0**	**19.7**
Pulmonary tuberculosis	0.0	0.3	−0.3	2.7	7.3	3.9	0.3	14.3
Phthisis	0.0	0.1	−0.4	1.4	3.9	2.4	0.2	7.6
Respiratory tuberculosis	**0.0**	**0.4**	**−0.7**	**4.1**	**11.2**	**6.3**	**0.5**	**21.9**
Cancer	0.0	0.1	0.1	−0.0	−2.1	0.2	0.4	−1.4
Neoplasms	**0.0**	**0.1**	**0.1**	**−0.0**	**−2.1**	**0.2**	**0.4**	**−1.4**
Violence	0.4	0.9	1.3	2.6	4.1	2.7	0.3	12.3
Violent deaths	**0.4**	**0.9**	**1.3**	**2.6**	**4.1**	**2.7**	**0.3**	**12.3**
Childbirth and puerperal fever	0.0	0.0	−0.0	−1.9	−2.2	−0.0	0.0	−4.1
Childbirth	**0.0**	**0.0**	**−0.0**	**−1.9**	**−2.2**	**−0.0**	**0.0**	**−4.1**
Pyrexia	−0.0	−0.0	−0.0	0.0	0.0	0.0	0.0	−0.0
Septic diseases	−0.0	0.1	0.0	0.1	0.4	0.4	0.1	1.1
Rheumatic fever and rheumatism of the heart	−0.0	0.0	−0.1	0.0	0.0	−0.0	−0.0	−0.0
Other causes	16.1	3.8	0.5	1.5	3.5	9.0	4.4	38.6
Other causes	**16.1**	**3.9**	**0.4**	**1.6**	**3.9**	**9.4**	**4.5**	**39.7**
All causes	**25**	**16**	**1**	**9**	**20**	**23**	**7**	**100**

Source: Calculated from the relevant Registrar-General's *Decennial Supplements*.

nineteenth-century London confirms this point. Infectious diseases in early childhood were responsible for narrowing the total male/female e_0 difference by 3.7%. This was primarily linked to the greater susceptibility of girls to whooping cough, but also to their relative vulnerability in the face of smallpox, diphtheria, typhus/typhoid and measles. It has been supposed that because girls were nutritionally deprived and less well tended to than boys, they were more prone to infectious and parasitic diseases (Preston, 1976; Ryan Johansson, 1984: 467). Certainly the relative neglect of girls played a part, but the picture is probably a lot more complicated than this. In the case of measles, Aaby downplays the roles of the relative strength of the sexes and differential treatment, in favour of explanations that emphasise intensity of exposure, dose of infection and cross-sex transmission. In short, he writes that it is possible 'girls … have more and closer contacts, and transmit infections more easily than boys' (Aaby, 1998: 242).

Respiratory tuberculosis between the ages of

1 and 14 also served to narrow the e_0 sex differential, but this was not the case in adulthood, particularly in the 30–49 age group. Violent deaths in adulthood – including accidental death, homicide, murder, manslaughter and suicide – emerge as a crucial determinant of the difference. Maternal mortality (childbirth) between ages 15–49 was the single most important cause in bringing female e_0 closer to that of males. Finally, neoplasms also reduced the gap between the sexes, probably as a result of the impact of breast and ovarian cancer mortality in women (United Nations Secretariat, 1988: 97). There being no readily available information as to the site of cancer leading to death, this can be no more than speculative. On the one hand, the figure of −10.8% might be an underestimate, since it is likely that ovarian cancer was frequently classified as a disease of the generative organs or as old age, which is subsumed under 'other causes' in the *Decennial Supplements*. On the other hand, it is also likely that deaths from the leading sites of cancer for males in modern societies – trachea, bronchus

and lung – were in fact classified under diseases of the lungs (United Nations Secretariat, 1988: 83–5).

The lower panel of Table 6 shows how the metropolitan epidemiological transition had a variable impact on these patterns. By 1901–10, the impressive reduction in mortality rates from infectious diseases and respiratory diseases had served to increase the contribution made by early childhood to sex differentials in e_0, but reduce it in later childhood (5–14 years) and infancy. Nevertheless, infancy remained the most important age group in terms of the overall gap between the sexes in e_0. Attention should be drawn to the fact that the decline in mortality from respiratory tuberculosis in adulthood, and the resultant increased male/female mortality ratio, had no discernible effect on that disease's contribution to the sex differential in e_0. It was in this age range that increasing mortality from neoplasms had a major influence by practically eliminating the male advantage. It is notable that deaths due to childbirth declined in importance over the period. Because the positive impacts of medical and technical advances in obstetric care did not begin to make their presence fully felt until the 1930s at the earliest, one possible explanation for this transformation lies with the decline of fertility.

Finally, mortality in the elderly (over 50 years) was far more important for the observed sex differential in e_0 in 1901–10 than it had been half a century earlier (29% compared with 12%). As overall e_0 rose, mortality was compressed at older ages (Buettner, 1995). Consequently, differential old age mortality came to account for increasing proportions of the gap between male and female expectation of life at birth, a trend that continued right up until the end of the twentieth century, at least at the national level (Lopez, 1983: 113–14; United Nations Secretariat, 1988: 93). In Victorian London, however, it is impossible to isolate any single cause of death, or even group of causes, responsible for this transition towards the importance of old age: certainly neoplasms were implicated, but, more importantly, so too was the opaque 'other causes' category. One victim of vague diagnoses in old age has been the elucidation of mortality from cardiovascu-

lar disease, which was frequently higher in females than males, particularly in childhood and early adulthood (Shorter, 1991: 232). It has been estimated that up to 40% of deaths caused by heart disease were certified as being due to old age (Preston *et al.*, 1972; Nikiforov and Mamaev, 1998). Given that cardiovascular mortality has been shown to vary ecologically between the sexes in modern societies on account of socio-economic status and other environmental variables (Park and Clifford, 1989), more epidemiological detail on the deterioration of cardiovascular mortality in the second half of the nineteenth century would doubtless be informative on this point (on male and female decline in the US in the twentieth century, see Patrick *et al.*, 1982). Unfortunately, it is not available by sex for individual districts for the two decades under consideration.

Sectoral variation is summarised in Fig. 7 by showing for both decades the contribution of each disease group without the age disaggregation. The increased predominance of the 'other causes' category is the most notable trend that registered across the metropolis. As a result, in percentage terms most broad categories of disease became less important as determinants of variation, even though their absolute contribution rose: infectious diseases, respiratory diseases, neoplasms, childbirth and violent deaths belonged to this group. The percentage contribution made by respiratory diseases to the sex differential reduced markedly in the contracting core and the stable core, and the contribution of violent deaths fell dramatically in all sectors apart from the contracting core. Sectoral change was equivocal for gastro-intestinal ailments, remaining practically unchanged percentage-wise in the outer suburbs, halving in importance in the contracting core, increasing two-fold in the stable core and by almost the same amount in the inner suburbs. A reduced percentage contribution from childbirth occurred in all sectors. Finally, transition was imperceptible for respiratory tuberculosis; the increasing disparity between the sexes in terms of age-specific death rates – attested by Fig. 6 and alluded to above in Table 5 – in fact made a minor impact on the e_0 sex differential in percentage terms.
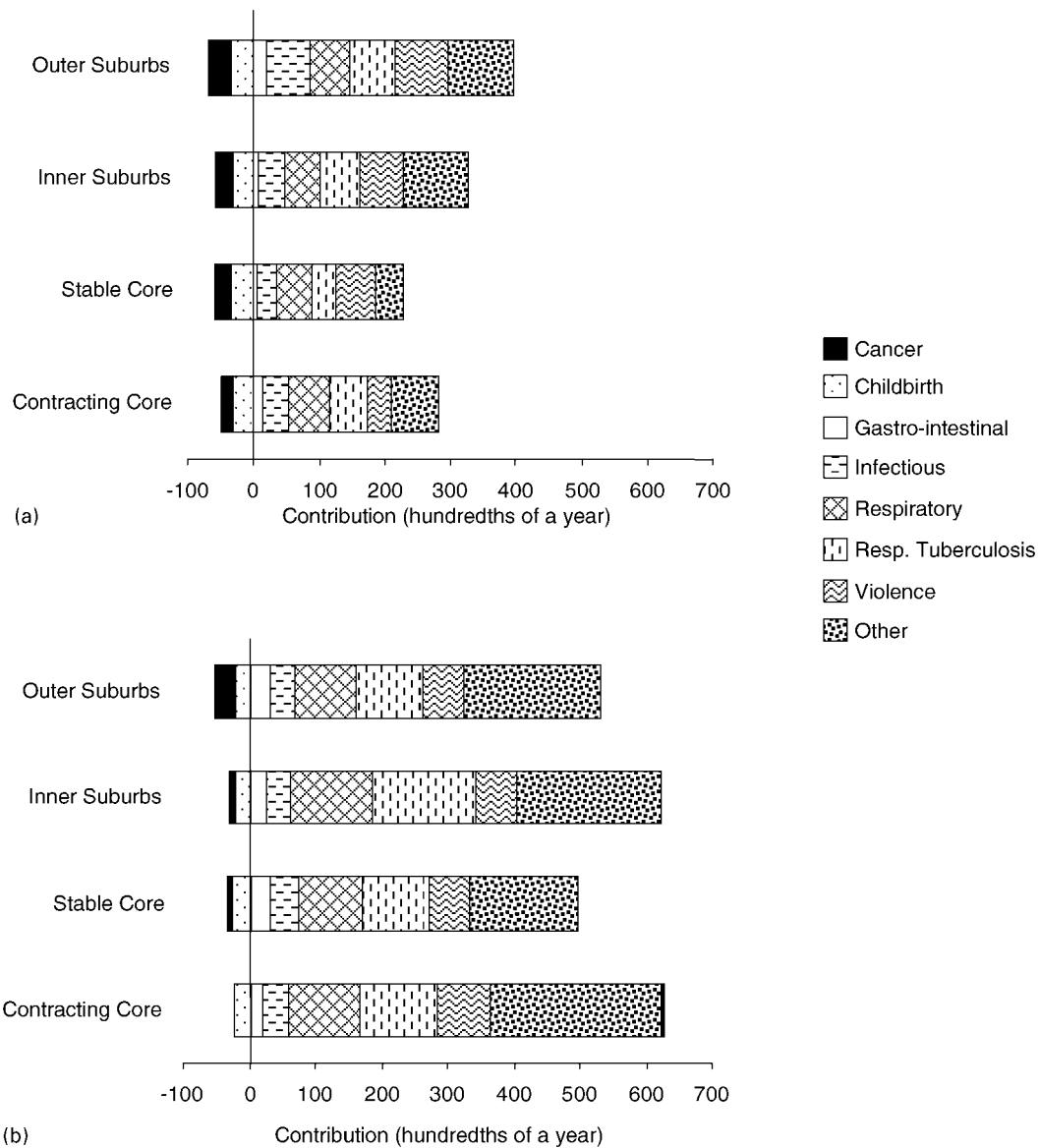
Figure 7. Contribution of disease groups to the sex differential in life expectation at birth, metropolitan sectors: (a) 1851–60; (b) 1901–10.
Sources: Calculated from the relevant Registrar-General's *Decennial Supplements*.

These cross-sectional comparisons can be augmented by introducing a temporal dimension that calculates for the sectors an age- and cause-specific contribution to the changing sex differential in e_0 between the two decades (Pollard, 1988). This approach emphasises that the determinants outlined earlier in Fig. 1 are not constant over time and can be related in broad terms to the particular characteristics of

London sectors. The gap in e_0 between the sexes in London increased from 2.55 years in 1851–60 to 5.55 years in 1901–10 (Table 2). Table 7 shows that change in infectious diseases over this period accounted for 14.7% of this three-year rise. More than a third of the increased differential (35.9%) was a result of decline in early childhood mortality. Taking cause of death and age together, 19.4% of the

Table 7. Percentage contributions to the change in life expectation at birth by age and cause, London, 1851–60 to 1901–10.

	0	1–4	5–14	15–29	30–49	50–69	70+	All ages
London								
Gastro-intestinal	0.8	1.7	−0.1	0.7	1.7	1.6	1.3	7.6
Infectious	−1.9	19.4	1.7	−1.6	−2.4	−1.0	0.4	14.7
Respiratory	−2.5	7.9	0.6	−0.3	1.2	3.7	2.7	13.3
Resp. tuberculosis	−0.0	1.5	1.6	3.6	6.0	1.4	0.1	14.1
Neoplasms	−0.0	0.2	0.1	0.4	2.1	3.1	0.4	6.3
Violent deaths	−0.1	0.1	−1.8	−2.7	−1.9	−0.2	0.3	−6.3
Childbirth	0.0	0.0	0.0	2.9	3.5	0.1	0.0	6.5
Other causes	3.5	5.1	−0.5	0.6	2.4	12.2	20.4	43.8
All causes	−0.2	35.9	1.5	3.6	12.7	20.8	25.7	100.0
Stable core								
Gastro-intestinal	0.9	1.6	0.0	0.7	1.3	0.7	3.1	8.3
Infectious	−0.3	9.2	0.6	1.1	−0.9	−0.7	1.4	10.5
Respiratory	−0.5	4.3	0.1	0.3	−0.2	0.1	3.2	7.3
Resp. tuberculosis	−0.1	0.3	0.1	4.1	3.5	0.4	0.1	8.4
Neoplasms	0.0	0.2	0.0	0.3	1.2	1.6	−0.0	3.3
Violent deaths	0.1	0.6	−0.9	−1.8	−1.4	−0.4	0.7	−3.1
Childbirth	0.0	0.0	0.0	1.5	1.5	0.0	0.0	3.0
Other causes	2.7	3.4	−0.6	1.4	1.1	4.9	49.3	62.2
All causes	2.8	19.6	−0.6	7.6	6.1	6.7	57.8	100.0
Outer suburbs								
Gastro-intestinal	2.3	3.1	−1.0	−0.5	2.5	2.8	1.6	10.8
Infectious	−7.6	39.1	1.0	−15.2	−6.7	−2.6	0.2	8.1
Respiratory	−4.8	13.1	0.2	−3.8	−0.1	4.4	3.0	12.1
Resp. tuberculosis	−0.8	2.6	2.4	−0.8	8.4	0.3	−0.9	11.2
Neoplasms	−0.1	0.6	−0.0	0.5	5.5	4.5	−0.5	10.5
Violent causes	−0.7	−0.8	−7.8	−12.3	−6.0	−1.0	−0.5	−29.2
Childbirth	0.0	0.0	0.1	7.4	9.7	0.2	0.0	17.3
Other causes	2.4	12.2	−0.4	−5.4	1.7	23.7	25.2	59.3
All causes	−9.2	69.9	−5.6	−30.2	14.9	32.3	28.0	100.0

Source: Calculated from the relevant Registrar-General's *Decennial Supplements*.

shift towards greater female mortality advantage in London can be pinpointed to the decline of infectious diseases in early childhood (included in this percentage is whooping cough, which accounted for 8.8% on its own). Also shown in Table 7 are two metropolitan sectors, the stable core and the outer suburbs, selected to represent the 'worst' and 'best' sectors in 1901–10 in terms of overall e_0. Some intriguing and interpretatively challenging results emerge. Firstly, a key component of spatial variation lies in transformations in mortality relating to early childhood and particularly the overall contributions made by causes of death that share a common mode

of airborne transmission.[8] Taken together, infectious diseases, respiratory diseases and respiratory tuberculosis in early childhood accounted for 55% of the increased differential in e_0 in favour of outer suburban females, but only 14% in the stable core. Secondly, the contribution made by 'other causes' is approximately equal in both sectors, but in the stable core it is concentrated in the very old (over 70 years) which served to make changes in that age group far and away the most significant driver of change. Thirdly, death in childbirth was far more influential in the outer suburbs than in the stable core. Fourthly, transformations in neoplasms exacerbated the sex differ-

ential by 11% in the outer suburbs, and only 3% in the stable core. Finally, violent deaths narrowed the sex differential in both sectors, but to a much greater degree in the outer suburbs.

DISCUSSION

The foregoing analysis suggests that a revised interpretation of sex differentials in mortality and life expectation at birth in Victorian Britain is required. A 'metropolitan' version emphasises those connections between 'fertility' broadly defined – that is, reproductive function, childbearing, parity, completed family size–and mortality that are mediated by the socio-cultural and socio-economic environment (cf. Hart, 1989: 118–22). We can begin with discussion of the direct risk of childbearing. The great majority of deaths in childbirth were due to puerperal fever, but unlike other infectious diseases, it has been consistently shown that historically death in childbirth varied remarkably little according to social class (Loudon, 1992: 111). Indeed, wealthier areas tended to have higher rates of maternal mortality than poorer districts (Marks, 1996: 119; Cullingworth, 1898 and Dudfield 1924, both cited in Loudon, 1992: 244–45). No direct evidence for London in this period is available for the observation regarding social class. If, however, the sectoral categorisation described in this paper is accepted as broadly depicting areas of contrasting wealth, then the measured contribution of death in childbirth to the increasing gap in e_0 between the sexes in the outer suburbs is initially surprising.[9] In his magisterial study of maternal mortality, Irvine Loudon concluded that:

'The evidence leaves little doubt that until the final ten to fifteen years of the nineteenth century, the safest way for a woman in London to be delivered, *regardless of social class*, was at home by a trained member of the staff of the Royal Maternity Charity or a trained midwife. The next safest was to be delivered at home by a private doctor and the next in the wards of a workhouse infirmary. The most dangerous by a long measure was in the wards of a prestigious

hospital such as Queen Charlotte's where the mortality was ten or more times as high as the Royal Maternity Charity.' (Loudon, 1992: 201, emphasis in original)

Whatever the means of delivery (that is, the combination of medical/lay personnel and location), the outcome did not vary by social class; but the utilisation of maternal services certainly did. In England and Wales in the 1880s, mothers giving birth as an institutional in-patient constituted only 3.5% of all deliveries, and charity out-patient deliveries amounted to a further 4.0% (Loudon, 1992: 195–6). More than 90% of babies, then, were born at home. Parents in poorer families more frequently relied on midwives to deliver their offspring, whereas wealthier clients would call on the services of a general practitioner (Marks, 1996: 197–200). The reverse social gradient in maternal mortality, and particularly that of puerperal fever, is thus explained, since 'midwives interfered with labour to a lesser extent than doctors, and were less likely to come into contact with cases of sepsis such as erysipelas and septic wounds' (Loudon, 2000: 189).

There was a marked decline in puerperal fever mortality rates in London between 1890 and 1912, but neither socio-economic factors nor the adoption of antiseptic methods in lying-in hospitals can explain the downturn, which was in any case reversed after the First World War. A more convincing argument is that the strain of Group A streptococcus, which caused the disease, declined in virulence (Loudon, 2000: 152–5). The impact of this would probably have been felt in all situations, even where the adoption of antiseptic techniques was by no means universal, such as general practitioner deliveries (Loudon, 2000: 164–5).

Assuming autonomous decline of the disease, it is unlikely that puerperal fever mortality had anything to do with the spatial patterning shown in Table 7 above. Alternatively, parity is an important risk factor in maternal death. Although the parity risk varies for individual causes of death in childbirth, Loudon's survey confirmed that:

'Maternal risk in first births (primiparity) is high, in second and third births it is low, and

then from fourth births onwards (referred to as 'grand multiparity') the risk begins to rise steeply, soon reaching levels in excess of first births.' (Loudon, 1992: 242)

It is now suspected that the average parity of women marrying husbands in upper and middle-class occupations in the late 1880s was possibly below four children by 1911; in most other classes it remained above four children, often more than five, and in some cases, more than six (Woods, 2000: 118–20). One need not lend support to the 'social diffusion hypothesis' of fertility control to argue that women married to wealthier husbands lowered their exposure to death in childbirth by having fewer children sooner than did women in other socio-economic groups, for whom grand multiparity declined but nevertheless remained; and by extension, that smaller families were more likely to become the social norm in places where upper and middle-class occupations were spatially concentrated within London, namely the outer suburbs.

Completed family size is also intrinsically related to parity. The shift towards smaller families acted to reduce the number of potential routes of disease transmission within a household, particularly between siblings. If, as a result of there being higher proportions of upper and middle classes in the outer suburbs, the average number of children per family in that sector declined before it did in the stable core – and the historical evidence discussed above certainly points to this chronology – then it is not fanciful to propose that opportunities for the domestic transmission of disease became increasingly unequal between the sectors. And since the fatality of infectious childhood diseases was unevenly divided between boys and girls for the social, cultural and economic reasons discussed in relation to Fig. 1 above, then the large contribution made to the sex differential in e_0 by infectious diseases in early childhood in the outer suburbs becomes more understandable. Contrasting the outer suburbs with the situation in the stable core provides guarded support for what might be termed the 'modernisation' hypothesis, whereby industrialisation and urbanisation help to eliminate excess female

mortality 'by first providing more employment opportunities for girls and ultimately by raising living standards to such a degree that differential investment [between boys and girls] ceases or continues without involving marked material deprivation' (Ryan Johansson, 1984: 474). If, as has been argued, girls suffer from infectious and parasitic disease mortality 'at higher rates than boys because they are relatively less well fed and cared for than their male counterparts' (Ryan Johansson, 1984: 467), it is tempting to conclude that the relatively muted contribution to the changing sex differential in life expectation at birth in the stable core made by the infectious diseases, respiratory diseases and respiratory tuberculosis might be caused by relative underprivilege in this sector and thus continued differential investment. The speculative nature of these hypotheses is underlined by the fact that domestic transmission did not operate as a single causal factor, but interacted with the quality of housing, nutritional levels, school attendance and the utilisation of isolation hospitals. All of these variables have place-specific influences and impacts on disease prevalence, but, with the exception of Aaby's work on measles (1998), little or no research has been carried out in an historical urban context to determine whether they worked to the advantage or disadvantage of boys or girls.

Alongside these influences, it must be remembered that the decline in mortality from some diseases was biologically determined, largely independent of human activity. The reduced virulence of puerperal fever in the late-nineteenth and early-twentieth centuries was referred to above. Scarlet fever is another disease that became far less fatal in the latter part of the nineteenth century, and an early-twentieth-century hypothesis that the respiratory tuberculosis bacillus underwent a similar transformation has recently been revived (Woods, 2000: 332–40). A portion of the changing sex differential in life expectancy can therefore be attributed to biological factors. Scarlet fever itself was one of those airborne infections whose decline ultimately served to disadvantage metropolitan boys. The reverse was true for men, particularly those who lived in the outer suburbs. Excess adult male mortality from infectious diseases in the

1850s was greatly attenuated by the 1900s, and it would seem that men in the outer suburbs were particularly well placed to reap the benefits. Violent death was another category of mortality where changes in adulthood contributed to a reduction in the female advantage. The mortality rate of all violent deaths for males in the outer suburbs declined by 44% between 1851–60 and 1901–10, but only by 15% in the stable core. This degree of improvement greatly exceeded that of females; violent death rates for females actually worsened in the stable core. Without more detail on the composition of violent deaths at district level, it is difficult to provide a plausible explanation as to why the increase in male advantage was so large in the outer suburbs compared with the stable core, other than to say that in relative terms, and despite the encroachment of the hazards and risks related to urban living, the outer suburbs became an increasingly safe and secure environment in which to live. Partial verification of this viewpoint is that infant violent death mortality rates – overwhelmingly dominated by suffocation and other murders – increased by more than 100% in the stable core, but remained virtually unchanged in the outer suburbs.

The displacement of dominant epidemic infections also served to benefit females because more children survived into adulthood and old age. The overall metropolitan risk of mortality was greater for adult men during the whole of the period (Figs 3 and 4). For the elderly, mortality risks initially favoured men but came to favour women. Neoplasms were implicated in this pattern, more so in the outer suburbs than the stable core. In the twentieth century, cancer has made the second largest contribution, after heart disease, to the increasing inferiority of male life expectancy at birth (United Nations Secretariat, 1988: 97). Information is not available from the *Decennial Supplements* on the sites of neoplasms, but it is probable that an increase in cancers of the respiratory system in males accounted for the change. Historians remain unsure as to whether this was a real rise, associated with greater exposure to risks such as chemicals and irritants, alcohol and tobacco (few if any women smoked before the twentieth century, when mass cigarette manufac-

ture began) (Graham, 1993; Hilton, 2000), or whether it was an artificial rise connected to the triumph of antisepsis. Antisepsis led to invasive abdominal and thoracic surgery in the 1880s and the subsequent revelation of many previously inaccessible internal cancers of the stomach, intestines and lungs, all of which had a greater incidence in males than females (King and Newsholme, 1893; Smith, 1990: 328). Whether these cancers can be connected to lifestyle behaviours and occupational risks across the sectors is yet another matter of historical uncertainty. What little evidence there is for the twentieth century suggests that the tobacco habit began as an upper- and middle-class pursuit (Doll *et al.*, 1997).

Finally, mention should be made of the compression of mortality in older age groups. By 1901–10, 12% of male and 16% of female deaths were in the over-65s. The corresponding figures for 1851–60 had been 8% and 10%. This key transformation was accompanied by a substantial decline of mortality rates in the elderly, particularly for females (Table 5). It is interesting to note that gains in life expectancy in many developed countries over the last half-century or so have been strongly influenced by improvements in mortality of the elderly, and these changes have also tended to favour females (Myers, 1996: 99; Hemström, 1999: 1772). In the case of Japan, declining mortality in childhood from gastro-intestinal and respiratory infections in the late 1950s and early 1960s accounted for much of the improvement in e_0. But in the 1970s and 1980s, longevity was increased by mortality reductions from chronic diseases, particularly stroke, in middle and older-aged people (Goldman and Takahashi, 1996: 158–62). Quite why these reductions should impact on women more than men is not clear, but the contrast provided by Victorian London is a revealing one, in that many adult chronic diseases of old age such as heart disease and stroke, not discussed in detail here but subsumed under 'other causes' of death, were in fact on the rise (probably both in real terms and because of improved diagnosis), and affected men more than women. Whether they did so irrespective of where people lived is difficult to say, because so many other residual causes of death were grouped into the same class.

CONCLUSION

Investigation of sex differentials in mortality in the past is valuable because such differentials may indicate deep-seated divisions in the structure and values of society that are no longer with us. However, prior to the task of interpreting the potential links between sex-specific mortality and social change, many problems are encountered in measuring these health differences in a simple and convincing way. Focusing on inequality via the level of mortality (as opposed to inequalities alone through the use of standardised mortality ratios), writers variously adopt either age-specific mortality rates or life expectancy as indicators; some, wittingly or unwittingly, attempt to explain transformations in the latter by reference to the former. This paper elected to examine the epidemiological dimensions of urban sex mortality differentials in Victorian Britain using both sets of measures, plus life table decomposition, which can evaluate the contribution of age groups and diseases to the change in overall life expectation at birth.

It must be admitted that such an analysis does not readily lend itself to simple descriptive or explanatory summaries, particularly when it incorporates a spatial dimension. Nevertheless, it can be said that in London in the mid-nineteenth century, the female advantage in life expectation at birth was largely determined by lower mortality from a range of ailments in infancy, in addition to respiratory tuberculosis and violent deaths in adulthood. The amassed female disadvantage of higher mortality from infectious diseases in early childhood, neoplasms in adulthood, and maternal mortality, was insufficient to tip the scale of life expectancy at birth in the favour of males. The widening sex differential witnessed over the next half century or so was heavily weighted in early childhood and old age, driven by the decline in infectious diseases and the survival of more children into adulthood, where females reaped the benefits of improved longevity.

One recent review of sex differentials in mortality suggested that urban areas were particularly interesting historically because of the interactions between cultural and economic factors that took place in such environments:

'The city, centre of new ideas regarding the family, fertility and the value of children, contributed to reduce the culturally-based inequalities between the sexes, but industrialization created other links with the conditions of work.' (Tabutin and Willems, 1998: 49)

Certainly, the significance of this observation has been underlined in this paper by the emphasis placed on the role of fertility in shaping spatial variations in mortality between the sexes. Women's reproductive function clearly exposed them to risks men did not have to contend with, related not only to the direct danger of childbirth but also to the possibility of puerperal infection introduced by doctors in attendance and, to a lesser extent, midwives. However, these risks varied in space because of cultural and socio-economic influences that impacted on the number of children that women had during their reproductive lives. Since completed family sizes differed between places, so too did the potential for the domestic transmission of infection.

In this article, a conscious attempt has been made to move the centre of attention away from those features of the urban/rural dichotomy that have tended to dominate historical inquiry into sex differences in mortality. It remains to be seen whether the metropolitan version of shifting sex differentials in mortality was replicated in other urban areas of Britain and elsewhere. The intra-urban comparisons that are possible for London are not only enlightening in themselves, but they also serve as a caution against assuming that there was a common 'urban' – or, for that matter, rural – experience of sex-differential mortality in nineteenth century Britain. The classification of metropolitan districts into sectors has representative meaning not only in terms of social and physical environments but also in terms of gendered occupational structures. The probable links between men's and women's health and the conditions of gendered urban work patterns have therefore been underlined. But the primary emphasis has been on infectious diseases in early childhood and mortality from a range of conditions in elderly people. This may lure researchers

away from explanations that focus predominantly on social, economic and cultural constructions of the adult working world, and address factors that shape experiences of the sexes when they are very young and very old.

NOTES

(1) In defence of historians, it should be noted that this observation also applies to research into gender inequalities in health in contemporary society. See Arber and Cooper (2000).
(2) Although for a discussion of mortality differentials within the largest provincial cities, see Szreter and Mooney (1998).
(3) This difference of two years was probably not as precise as English Life Table 3 would have us believe. The table was constructed by William Farr using the age structure of the 1841 and 1851 censuses and information on age at deaths occurring between 1838 and 1854. As such, Woods (2000: 180) describes it as 'the most reliable cornerstone we can have for a discussion of Victorian mortality', acknowledging, however, that age misreporting over age 50 in these two censuses makes the reliability of its e_0 estimates uncertain (Wrigley and Schofield, 1981: Appendix 14, 708–14).
(4) Of course, variation in the pre-civil vital registration era may be explained in part by the constraints of family reconstitution, which captures a relatively small number of events and cannot adequately address mortality patterns in old age.
(5) These values are based on the decennial total of deaths in each district, and the population at risk was estimated using a calculation that assumed each district had a constant annual rate of population growth between decennial censuses (Mooney, 1994: 91).
(6) Initial capital letters are used to signify grouped causes of death that are drawn from the various classifications of disease that appear in the Registrar-General's *Decennial Supplements* during the period.
(7) The software package 'Lifetime, Version 1.23' was used to perform these calculations. In order to render useable with this computer application the nineteenth-century *Decennial Supplement* material, cause of death mortality totals, published in ten-year age bands 25–34, 35–44… 75 and over, were subdivided into five-year age bands in proportion to the population at risk in each of these age bands (Pollard, 1992).
(8) Exceptions here are the non-respiratory forms of tuberculosis such as scrofula, tabes mesenterica and tubercular peritonitis in the infectious diseases group. These causes of death could be readily contracted by feeding babies with milk containing bovine tubercular bacteria or which had become infected by humans during milk processing procedures.
(9) It should be remembered here that life tables relate deaths in childbirth to the age range of the female population in which the deaths occur, whereas maternal mortality rates relate to the number of births.

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