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Historical and Life Course Timing of the Male Mortality Disadvantage in Europe

Epidemiologic Transitions,
Evolution, and Behavior

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LABOR AND POPULATION

Historical and life course timing of the male mortality disadvantage in Europe:
Epidemiologic transitions, evolution, and behavior

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ABSTRACT

This study employs vital statistics data from Sweden, England, Wales, France, and Spain to examine male:female mortality differentials from 1750 through 2000 and their inter-relationship with epidemiological transitions. Across all ages and all time periods, the largest relative mortality disadvantages are to young adult men. When crisis mortality from the two world wars is removed, we show that the mortality in this young male age group is about two to three times the level of female mortality cross-nationally. In addition, we show that the timing of this stabilization in male mortality disadvantages occurs during the last half of the twentieth century, when our measure of epidemiological change also stabilizes at a new low level. The findings are consistent with an interdisciplinary theoretical model that links social, technological and epidemiological changes that occurred through the first half of the 20th century with the unmasking of mortality disadvantages among young adult men.

INTRODUCTION

Overview

An extensive literature in demography, population biology, and social epidemiology documents male disadvantages in longevity, across populations and even species (e.g. see: Carey 1997; Kalben 2002; Lopez et al. 1995; Preston 1976; Waldron 1990). This research highlights two age periods during which male disadvantages are most notable: older adulthood, when the *number of deaths* have been greater for males; and young adulthood, when the *relative rate of dying* has been greater for males (Nathanson 1984). Observable excesses in male mortality in the industrialized world are largely recognized to have appeared during the 19th and 20th centuries, depending on the country of interest (Lopez et al. 1995; Preston 1976). Interestingly, the emergence of male mortality disadvantages (MMD) appear to be concurrent with demographic and epidemiological changes that shifted the age and cause structure of mortality in industrialized countries. Previous research has described the role of social, economic, public health and medical transformations in shaping these shifts in mortality patterns (e.g. see: Fogel 2004; McKeown and Record 1962; Szreter 2004). In addition, a few studies have linked urbanization and industrialization to sex differentials in mortality during epidemiological transitions (Mooney 2002; Preston 1976). In this study, we integrate an historical and evolutionary approach with biodemographic theory and methods to consider questions about the historical timing and age-specificity of the male

mortality disadvantage within several countries in western Europe, and with several centuries of data.

We are specifically interested in the following questions: 1) whether male mortality disadvantages (MMD) are in fact highest in young adulthood; 2) whether there are changes in the trends of MMD when we consider historical time periods that cover multiple centuries; 3) whether there are differences in MMD across different industrialized countries; and finally 4) whether there is in fact a temporal relationship between the historical trends in MMD and the timing of demographic and epidemiological transitions.

In the following sections, we place the developmental and historical timing of the emergence of MMD in an evolutionary-life history context, deepening the explanatory frame for mechanisms underlying such trends.

Biodemographic and social structural explanations

Historically contemporaneous trends in MMD and the age-specific and cause-specific patterns of mortality in industrialized countries suggest that there is a theoretical and empirical relationship between the patterns of demographic and epidemiological change and patterns of sex-differences in mortality over time. The most popular framework for describing epidemiological change, Omran's Epidemiological Transition Theory (ETT), involves several phases of mortality that track improvements in living conditions experienced by Western European countries undergoing industrialization. From an initial period of high and variable mortality associated with plague, famine and war, life expectancy starts to increase. During this increase there is first a transition to period of high infectious disease mortality, with highest mortality rates in infancy and

early childhood, and then a second period of chronic disease mortality, with the highest mortality rates in later life (Omran 1971). In more recent extensions of ETT, increasing improvements in survival at the oldest ages have been incorporated into a final phase of the model (Olshansky and Ault 1986)¹.

The shifts described in ETT from pandemic to infectious disease mortality are associated with technological advancements in agricultural production, food storage, and irrigation helped to gradually bring basic material sustenance within human control (Fogel 2004; McKeown and Record 1962). These advancements allowed populations to buffer environmental threats to survival increasing life expectancy and reducing the variability of the age of death (Wilmoth and Horiuchi 1999). Further improvements in living conditions, economic development, and public health initiatives reduced infant and child mortality, such that, the turn of the century saw an epidemiological transition from infectious to chronic disease mortality (Szreter 2004). Moreover, through the end of the twentieth century, continued innovations in knowledge and technology for improving health and longevity have been observed through both the increases in life expectancy and the greater concentration of deaths at the oldest ages (Edwards and Tuljapurkar 2005; Robine 2001; Wilmoth and Horiuchi 1999).

We argue that both the technological, social and political changes associated with ETT and the changes in the age distribution of mortality involved in ETT are relevant to understanding the historical patterns of MMD. Beginning with pre-industrial trends in sex differentials before the first epidemiological transition, historical accounts suggest that

¹ Researchers have also discussed the possibility of reverse transitions (particularly with the growing impact of infectious diseases associated with HIV/AIDS) and have critiqued the association between demographic or epidemiological transition and a uniform, linear process of industrialization in cultures outside of Western Europe (Greenhalgh 1990; Wilmoth and Horiuchi 1999).

mortality was as high, if not higher among women than men (Henry 1989). These sex differences are explained by an unequal distribution of food, harsh working conditions for women, and high maternal mortality (Henry 1989). Technological innovations that increased agricultural output and stability not only helped to increase total life expectancy, but also eliminated the issue of food scarcity as a driver of gender inequities in the distribution of food. We expect that sex differences in mortality through the first epidemiological transition might reflect these relative improvements for women.

In addition, previous research has suggested that biological differences between men and women in their immunological responses may account for higher male mortality during historical periods when infectious disease has predominated (Gage 1994; Lopez et al. 1995). Thus, an emergence of might be expected between the first epidemiological transition to infectious disease mortality and the second epidemiological transition to chronic disease mortality. Furthermore, we hypothesize that the improvements in medical technologies during the first several decades of the twentieth century that reduced infection and improved maternal outcomes (CDC 1999) may also have served to shift the sex differential to the disadvantage of *young adult* men.

This last hypothesis is consistent with literature which links twentieth century sex differentials in chronic disease mortality to the sex differences in behavior and environmental exposures (Lopez et al. 1995; Nathanson 1984; Waldron 1990). Differences in gender norms about behavior and risk-taking, as well as differences in sex roles that determine the types of activities and exposures men and women experience in their daily lives, have been described as structural explanations for MMD in young adulthood and midlife (Nathanson 1984). Gender differences in norms about behavior

and risk taking have been particularly useful in explaining the MMD in adolescence and young adulthood associated with accidental and violent death, as well as the MMD in midlife associated with tobacco-related cancers and heart disease (Chapman Walsh et al. 1995; Pampel 1998; Pampel 2002). These behavioral motivations for MMD are given further relevance in the context of literature on evolutionary theory and psychobiology.

Linking history and demography with evolutionary and biological mechanism

An evolutionary life history approach lends additional explanatory insight to historical and social structural theories regarding the emergence of MMD during industrialization, and helps link basic, shared properties of human behavior to the historical and technological changes occurring during this time. Evolutionary life history theory examines the way that neural and endocrine systems of organisms produce behavioral tendencies and physiological investment patterns that represent “best guess” predictions regarding current and future environmental conditions (Stearns 2000). Theoretical and empirical work has shown that males and females face different social and physiological constraints and opportunities for reproductive success, resulting in sex-specific physiological, psychological and behavioral vulnerabilities (McNamara and Houston 1996; Svensson and Sheldon 1998). Specifically, the necessity of high physiologic investment during pregnancy and the advantage of maternity certainty centers female somatic and behavioral investment on the production and nurturance of offspring. Males face a much different competitive field, with the dual constraints of paternity uncertainty and status-linked access to mates yielding a highly skewed distribution of reproductive success. As a result, male somatic and behavioral investment

is often heavily channeled towards status acquisition and male-male competition throughout the lifespan (Bjorklund and Pellegrini 2000; Lindstrom 1999).

It has been suggested, thus, that the evolutionary process has yielded specific status-linked behavioral and psychological propensities and sensitivities in human males, many of which are potentially destructive to the self and others (Chisholm 1999; Daly and Wilson 1985). The endocrine architecture of such propensities seems to be the critical intersection of gonadal and adrenal hormonal response to social threat or instability (Flinn et al. 1996; Mazur 1995; Sapolsky 2004). Critically, such strong psychobiological linkages to environmental cues of status position among men yield diverse behavioral risks, from propensity to homicide (Dabbs, Jr. et al. 2001) to propensity to smoke (Booth et al. 1999). Generally, empirical research has shown that status-linked male mortality risk is driven by behaviors that emerge in the mid-teens, and peak in the mid-twenties (Daly and Wilson 1985). We suggest that this research on male psychobiology are relevant to understanding when and why researcher have observed male mortality disadvantages in young adulthood.

Turn of the century technological and institutional change has helped usher in a new generation of “risk societies” (Beck 1998) across the western world, whereby the onus of inequality has become increasingly individualized.. Social isolation and anomie (Durkheim 1897:1979) and male role-frustration have been invoked to explain trends in suicide and homicide that track modernization and industrialization (Pampel 1998; Pampel and Gartner 1995). Meanwhile, work in evolutionary psychology and social epidemiology has linked wealth inequality with homicide and a host of other destructive

and self-destructive behaviors dubbed the “young male syndrome” (Daly et al. 2001; Daly and Wilson 1985).

Contradictions between the opening of opportunity via rapid social and economic change post-WWII and the limitations of economic opportunities via class and ethnic discrimination through the later part of the twentieth century (Massey 1996), theoretically present young men with everyday psychological conditions that have become increasingly conducive to status competition and status-frustration. Social epidemiologists have suggested that this is analogous to experimental or natural inductions of social instability induced by Saplosky and colleagues (1983) in primate societies, which often produce intense and violent struggles for rank that result in both short and long-term detrimental health consequences (Wilkinson 1997). Studies on social inequalities in health argue that social and political changes attendant to de-industrialization through the later half of the twentieth century, have had a direct psychological, biological, and behavioral impact on individuals living in the western European countries and the U.S. (Marmot 2004; Wilkinson and Pickett 2006). This theoretical background leads us to hypothesize that the historical changes in social, political and economic conditions that have transformed the primary sources of mortality risk may not just have unmasked male-biased risk processes, as described earlier, but may have in fact *accentuated* such risk pathways for young adult men.

Specific Aims

We employ vital statistics data from several industrialized countries (Sweden, England, Wales, France, and Spain) to examine male-female mortality differentials from 1750 through 2000 and their inter-relationship with trends in epidemiological transitions

that are indicated by changes in life expectancy and the age distribution of death (variability in the age of death). The demographic indicators of life expectancy and mortality inequality allow us to historically locate epidemiological transitions in each country, and consider their temporal relationship with the unmasking of male mortality differentials. Furthermore, age-specific mortality data allow us to locate the life course “peak” of mortality differentials across countries. The combination of these data in a comparative framework allows us to locate and theorize the specific intersections of historical processes, social context, and bio-behavioral life course processes causing the emergence of MMD in the past century.

DATA & METHODS

Mortality Data

Mortality statistics for Sweden, England and Wales, France, and Spain are obtained from the Human Mortality Database (HMB) and are compiled for demographic manipulation. The HMB provides life table functions for men and for women in a standardized format for various countries (<http://www.mortality.org/>). In this study, we use period life tables in the one by five format, wherein mortality rates and other life table functions are provided in single-year age groups averaged over every five years. We have selected four different countries with at least a century of data available through the HMB and which represent some of the cultural and regional differences among industrialized countries. The life tables from Sweden cover the longest historical series, nearly three centuries of data from 1751 through 2000. The life tables from England &

Wales cover nearly two centuries of data from 1841-2003.² And for France and Spain, the data cover the most recent century (1900-2000 and 1908-2000, respectfully).

Indicators of Age-Specific Sex Differentials and Overall Population Health Trends

Two types of indicators are used in this study. The first captures the sex differentials in mortality. We calculate age-specific male/female mortality ratios (i.e. sex ratios, SR) using the age-specific mortality rates for every five years.

$$SR = m(\text{age } 'x', \text{ males}) / m(\text{age } 'x', \text{ females})$$

A male mortality disadvantage (MMD) is indicated by $SR > 1$. We also calculate the ratio of the log of the mortality rates for use in figures where the mortality rates are displayed in log-scale. It is important to note that we display the inverse of the ratio of log-mortality so that MMD is indicated by a ratio greater than one.

The second set of indicators we use captures the population trends in mortality consistent with demographic and epidemiological transitions. As noted earlier, life expectancy at age zero (e_0) and the interquartile range of the life table distribution of deaths from age zero (IQR_0) have been used in previous studies to indicate the timing of demographic and epidemiological transitions (Robine 2001; Wilmoth and Horiuchi 1999). In addition, at an individual level, IQR_0 may serve as an indicator of the degree of uncertainty about the timing of death and thus have behavioral implications for the likelihood of risk taking (Wilmoth and Horiuchi 1999). We are interested in the distribution of the ages of deaths across all ages (IQR_0) as well as the distribution of the ages of deaths across all ages including and after age 10 (IQR_{10}). This is because research on indicators similar to IQR_0 and IQR_{10} advocate excluding deaths prior to age

² Please note that we use the population representative life tables that include the death of men and women in the military services, in addition to the deaths in the civilian population.

10 so that the survivorship distribution is less skewed by the high spike in mortality at birth and early childhood (Edwards and Tuljapurkar 2005).

We calculate the ratio of life expectancy at birth using the values of e_0 from the HMD for each five-year period for men and for women. The calculation of the distribution of the age of death via interquartile range is more intensive. The IQR_0 is the difference in years between the 25th percentile (i.e. the upper quartile) and the 75th percentile (i.e. the lower quartile) of the survivorship function (l_x) which is available from the HMD. The IQR_{10} is the difference in years between the 25th percentile (i.e. the upper quartile) and the 75th percentile (i.e. the lower quartile) of the survivorship function starting at age 10. In order to calculate the survivorship starting at age 10, we rescale the l_x function available from the HMD so that the survivorship function at age 10 is equal to 1 (i.e. we calculate l_x/l_{10} for ever age at or above age 10).

Analytical Plan

Our analysis of the historical trends in the age-varying pattern of male-to-female mortality differences involves several steps. First, we construct contour maps of the mortality ratios by age and year for each country (Figures 1-4). This allows us to locate the life course “peak” of mortality differentials across countries. We then examine the trends in the mortality ratio in young adulthood in conjunction with the indicators of population health differences for men and women over all ages (Figures 5-7). This allows us to locate the historical emergence of the third epidemiological transition in each country and to test its relationship with the unmasking of male mortality differentials.

FINDINGS

Historical Trends in Sex Ratios of Mortality

Contour maps of the ratio of male:female mortality rates for Sweden, England and Wales, France and Spain are depicted in Figures 1-4. Ratios of period life table mortality rates by age and year are depicted from age zero to age 100 along the x-axis (left to right) and from the earliest year that data are available for a given country to year 2000 along the y-axis (back to front). The height of the peaks depicted in the figures corresponds with the level of male mortality disadvantage, and the depth of the valleys corresponds, respectively, with the level of female mortality disadvantage (light blue areas). For the mortality peaks, ratios range along the z-axis from one to over four, and for the mortality valleys, ratios range from one to zero. The burgundy regions on figures indicate about equal mortality among men and women (i.e. a ratio of 1-2). Ratios below one, or female disadvantage. Ratios above three covering the top of the peaks are colored light green. And finally the area at the base of the peaks between the highest ratios in light green and the equal ratios in burgundy, the contour plot is yellow, indicating that male mortality is 2 to 3 times female mortality.

[Figure 1 about here]

Sweden, 1751-2000

The first figure depicts mortality ratios for the country with the longest series of historical data, i.e. Sweden. The 250 years of age-specific mortality differences depicted in Figure 1 show that it is not until mid-way through the twentieth century that mortality among males reaches levels greater than twice that among females. From 1751 through 1930, mortality is no more than 1.5 times greater among men than among women in any

age group. The highest ratios during the pre-industrial period (prior to 1900) are in adulthood, at a level about 20-30% higher than female mortality (i.e. ratio of about 1.2-1.3). At other ages, mortality is no more than 20% higher.

In fact, before the turn of the century, there are age groups where mortality is lower among men than women. For the later part of the eighteenth century and throughout the nineteenth century, mortality in adolescence (age 15) is consistently *lower* among men than women. In addition, male mortality is also lower among young adolescents and children (age 5 and 10) for a clustering of years in the nineteenth century. In the late nineteenth century and the early twentieth century, there are also years when mortality in adulthood (30-34, 35-39, 40-44) is lower among men than women.

Beginning in 1915-1919 the pattern of relative similarity between male and female mortality (and in fact more pronounced female disadvantage in selected age groups) begins to change. This is specifically the case for young adult men. In 1900-1904 male mortality in the young adulthood (age 20) is only 20% greater than (1.2 times) female mortality. This difference levels off during the depression years of 1930 to near equality (i.e. a ratio of 1.1). Then by 1945 the MMD age at age 20 has increased such that male mortality is 1.6 times female mortality, and by 1960 mortality of young adult men has nearly doubled to 3 times the mortality of women.

Similar patterns occur in other age groups; however, the relative differences between men and women are most striking for young adults age 20 and 25. The highest peaks occur in these age groups during the years 1980-1984 and 2000-2004. A slight increase in MMD is also observed in midlife in the ages 55, 60, 65 and 70. The mortality ratios in these older age ages never reach the levels observed in young adulthood, but at

about 1.9-2.1 times the mortality of females in these ages, they are higher than the mortality differences in any of the surrounding ages.

England & Wales, 1841-2000

Similar to Sweden, men in England and Wales are not at a large mortality disadvantage until after 1900, when these disadvantages are concentrated in young adulthood. The data in Figure 2 show that mortality rates for men and women during the late nineteenth century in England and Wales were fairly similar and that in adolescence and young adulthood the rates were actually *lower* among men than among women.

[Figure 2 about here]

Between 1900 and 1950 large changes in the male-to-female mortality differential occur in conjunction with the two world wars³. During the years of WWI (1915-1919) male mortality in the ages 20 and 25 is, respectively, about 8.5 and about 6.5 times higher than female mortality in these ages. The next large peak corresponds with WWII, beginning in 1940-1944 when mortality is about 3.7 and 3.3 times higher in the ages 20 and 25 respectively. The peak continues into the period 1945-1949, with mortality about 3.3 and 2.4 times higher among men in the ages 20 and 25 respectively.⁴

The next change in the pattern of mortality occurs around 1950, when mortality among men again begins climbing relative to women. Over ten years the ratio increases from 1.8 to nearly 2.7 among 20 year-olds, and by 1980-1984 mortality is 2.8 times

³ Here it is important to point-out the benefits of the HMD data we are using, in that they are population representative and thus include both the casualties to civilians and the casualties to enlisted men and women.

⁴ Note that the data from 1945 are averaged with the years 1946-1949 after the war, so that the influence of the war in 1945 appears less dramatic than the three years of war that are included in the period 1915-1919. We use life tables in which the age-specific rates are calculated for five-year periods of time in order to obtain more reliable estimates. When the trends are examined using data in single years, the ratios for 1945, in which there were the greatest WWII casualties, are higher than the ratios for any of the years between 1915-1919 corresponding with WWI.

higher among men than women age 20. During this period there is a slight increase in the mortality of men in midlife relative to women (i.e. age 55-70). This is not observed during the last two decades of the century, but the disadvantage among young men persists, and grows larger. In the last year 2000-2004, male mortality is nearly three times female mortality at age 20 (i.e. a ratio of 2.9).

France, 1900-2000

The century of data available from France is used to depict historical trends in mortality differences between men and women. We find trends similar to those described above for England and Wales, with the greatest differences between male and female mortality in young adulthood. However, in France we also find a slightly more widespread MMD after 1950 such that the ages with mortality ratios above two extend from age 15 through 75 (Figure 3). Like England and Wales, we also find that the two world wars factor dramatically into mortality patterns. There are particularly high ratios during the years of WWI (1915-1919) that exceed the limits of the figure⁵. For France, these large mortality ratios occur at three ages—age 20, 25, and 30—in which mortality is respectively 8.0, 7.2, 5.3 and 4.3 times as great among men as among women. The mortality peak during the years of WWII is earlier in France (1940-1944) than it is in England in Wales, and it does not involve as large of differences between men and women; the ratio is 2.6 for age 20 and 2.9 for age 25.

[Figure 3 about here]

In France, like England and Wales and Sweden, there are increases in mortality in young adulthood that emerge about 1950 in the ages 20 and 25. By 1960, the two peaks

⁵ Note that for France, in contrast with England and Wales, even when using single-year data the highest mortality differentials occur during the years of WWI not WWII.

of male mortality disadvantage observed previously in these two countries are also found in France for young adults and adults in midlife. In addition by 1970 in France, even in the “trough” between these two peaks for the ages 30-50 now involves growing MMD become apparent. By 1990, the mortality rates among men between the ages 20-65 years are all at least 2.5 times those among women. The rates for young men (20-30 years) reach as high as 3.4 times those of young women.

Spain, 1908-2000

The historical mortality patterns discussed in relationship to the past four countries are also observed in Spain. First, the mortality ratios depicted in Figure 4 reveal that the mortality disadvantage among men does not reach two times the mortality level of women until after 1950. Secondly, there are actually *female* mortality disadvantages among children, adolescents and young adults during the first several decades of the twentieth century. In the case of Spain, female mortality disadvantage also appears among older adults. Thirdly, there are wartime mortality differentials associated with the two world wars during the first half of the twentieth century. In the case of Spain, only during WWI is the mortality of men more than two times the mortality of women. Finally, like the previous countries, the late twentieth century mortality disadvantage among men is highest among 20 year-olds and has a second peak in later adulthood; however like France, in Spain, there is also a mortality disadvantage among men in the ages between young adulthood and later adulthood.

[Figure 4 about here]

Summary

The contour maps of the historical trends in the mortality ratios indicated that, in all four countries, the highest mortality differentials were to men in young adulthood (age 20). Using Sweden, the country with the longest history of recorded mortality from the HMD, we show that this increase in mortality disadvantage for young men arose in light of faster mortality decline among women than men between 1940-1950 (See Figure 5; trends for the other countries are available upon request from the authors). This trend was not unique to Sweden. Despite international variability in the exact historical timing and magnitude, there is a notable increase in the male:female mortality differentials for young adult men across all four countries during the second half of the twentieth century.

[Figure 5 about here]

Historical Trends in Population Health Trends and Young Adult Male Mortality

Disadvantage

In the preceding sections, we identified young adulthood (age 20) as the age-period when we observed the most dramatic sex differences in survival across all three industrialized countries. In the following section we examine whether the increase in young adult male mortality disadvantage occurred in relationship to changes in overall population health in the various countries. The trends in expected survival from age zero (e_0) help us to ascertain when large-scale declines in mortality have occurred, while the trends in the population variability in expected survival (IQR_0 and IQR_{10}) help us to ascertain when the timing of the epidemiological transitions have occurred.

Sweden

We begin with Sweden in light of its long historical record of mortality data. Figure 5 depicts the dramatic changes in e_0 and IQR_0 for Sweden that Wilmoth and Horiuchi (1999) used to demonstrate the utility of IQR as an indicator of epidemiological transitions. There are very large gains in e_0 from a life expectancy in the mid eighteenth century of about 30-40 years to a life expectancy of nearly 80 years at the end of the twentieth century. The timing of the changes in e_0 is similar to the pattern in IQR_0 with large declines in IQR_0 from a wide spread in the survivorship distribution of about 60 to 70 years to a fairly narrow spread in the survivorship distribution of about 10-20 years. Wilmoth and Horiuchi (1999) explain that the decline in IQR_0 over the later decades of the nineteenth century and the first half of the twentieth century is determined by, first a decline in infant and child mortality, and secondly a decline in midlife mortality (age 45-65) and a shift to mortality in later adulthood (at or above age 65) (Wilmoth & Horiuchi 1999).

[Figure 6 about here]

In this study, we superimpose the changes in the male mortality disadvantage in young adulthood in relationship to these trends in the overall level of mortality and the age- and projected cause-structure of mortality⁶. The timing of the increases in the mortality differentials is most clear. For the two centuries prior to 1950, the mortality ratio hovers just slightly above one (indicating that mortality of men at age 20 is almost equal to mortality of women at this same age). Then, between 1930 and 1955, the relative difference in male and female mortality at age 20 increases sharply. The increase in the

⁶ As noted earlier, the age-composition is used here and by previous authors (Wilmoth & Horiuchi 1999; Robine 2001) to provide information about the cause-structure upon which discussions of epidemiological transitions are based.

ratio of the log-mortality observed on the graph from about 1.01 to 1.15 is equal to nearly a three-fold increase in the ratio of “unlogged” mortality represented in the earlier contour plots.

Contemporaneous with the timing of the increases in the mortality ratio in young adulthood are changes in $IQR0$ and $IQR10$. As noted earlier, $IQR0$ makes a dramatic decline between 1890 and 1950 from a variability of about 60 years to only 15 years. Because $IQR10$ does not include the changes in infant and child mortality, the variability is about 30 years at its maximum prior to 1900. By 1910, declines in $IQR10$ become evident and by 1950 it stabilizes at about the same level as $IQR0$. The asymptotic declines in variability quantified by $IQR0$ and $IQR10$ appear to match the asymptotic increases in the mortality ratio more closely than the linear pattern of increase in $e0$ described earlier. In addition, of all the three indicators, $IQR10$ has a temporal pattern of initial change, as well as stabilization, that is closest to the temporal pattern of growth in the young adult mortality differential. In the next section we examine whether the historical trends in $IQR10$ and the historical trends in the young male mortality disadvantage co-occur in other industrialized countries.

International Comparisons

Contemporaneous trends in the variability of survival and in the male:female mortality ratio are depicted in Figure 7. In order to improve the visual clarity, the ratios are capped at 1.5 and the values for years in which the ratios are above 1.5 are noted at the bottom of the figure. There is variability between countries in the magnitude and the historical timing of overall population health and young adult male mortality disadvantage. Despite this variability, however, there is a similar asymptotic pattern of

increase in the mortality ratios and decrease in the variability of survival that is observable across the countries. As we observed in Sweden, the asymptotic trends in *IQR10* and MMD stabilize at new low and high values (respectively) by about 1950. In addition, if we set aside the dramatic peaks of mortality disadvantage that have occurred during the two world wars, Figure 7 suggests that the beginning of the increase in MMD occurred in the first three decades of the twentieth century.

[Figure 7 about here]

DISCUSSION AND CONCLUSION

In this study we have identified four central findings about sex differentials in mortality. First, by using international data that cover long historical periods prior to industrialization, we have confirmed that the largest mortality differentials have involved disadvantages to men in the young adult age-group. Secondly by setting aside crisis mortality from the two world wars, we have clarified that it is not until last half of the twentieth century that MMD reached a level where mortality for men was two to three times that for women. Lastly, we showed that there are strong relationships between historical change in young adult mortality disadvantages and historical change in overall population health. We have found that the male mortality disadvantage in young adulthood stabilized at a new high level in the 1950s at the same time as our measure of epidemiological change (the *IQR*) stabilized at a new low level.

Our findings are consistent with previous literature. We have used a rich historical database to show that mortality rates were similar among men and women prior to the twentieth century and that in the young adult age groups mortality rates were often lower

for males than females. This is consistent with earlier research on historical trends in mortality (Henry 1989). In addition, during the twentieth century, we observe high male:female mortality differentials in the two age groups identified in earlier literature: young adulthood and later midlife (Nathanson 1984; Waldron 1990). Furthermore, our results show that a particularly large differential has only been observed among young men in their late teens and early twenties, and that it has been a result of faster declining female than male mortality. Finally, we identified the historical timing of the large increase in the sex differential as occurring in the first three decades of the twentieth century. This is a historical period when previous literature has noted greater benefits to women due to the twentieth century reductions in infectious disease (Gage 1994), as well as the improvements in the effectiveness of surgical procedures that reduced obstetric complications during pregnancy and childbirth (CDC 1999).

With minor differences, our results were replicated across Sweden, England and Wales, France, and Spain. The similarity of these trends suggests that a broad set of historical, social structural, and technological changes are likely to be responsible for the timing and stabilization of male mortality disadvantages in the 20th century. While previous authors have described how turn of the century improvements in public infrastructure, living conditions, nutrition, and medical technology led to mortality reductions (Fogel 2004; McKeown and Record 1962; Szreter 2004), we highlight how the new trend of a persistently high mortality differential in young adulthood occurred at a time when male:female differences in suicide and violent mortality predominated in this age group as well (Hueveline 2002; Nathanson 1984). Such behaviors are more widely distributed in time and space, and much less subject to biomedical intervention than

pregnancy. As a result, public health is currently struggling with effective theories and interventions for such diverse behaviors in diverse settings.

The increase in the sex ratio for suicide during the later half of the twentieth century has been related to shifting institutional structures after WWII that changed women's legal rights and economic opportunities (particularly with respect to labor force participation and divorce) (Pampel 1998). In this context, sex differences in mortality are associated with differences in social norms, policies and institutions that determine different constraints and opportunities for men and women—particularly with respect to their differential exposure to “risky or stressful situations” (Nathanson 1984). In addition, they are recognized to be responsive to other environmental forces—such as modernization -- which reshape the constraints and opportunities of men and women in unequal ways (Nathanson, 1984).

We suggest that evolutionary life history theory helps bring sex-specific behavioral motivations into better relief. In addition, we suggest that life history theories may also help to explain why the social and environmental changes associated with modernization and industrialization present not only extrinsic sources of risk, but also have had pathogenic influences on psychological and behavioral functioning.

Dismantling older and more stable class structures and ways of making a life, modernity has placed behavioral decision-making (and the psychological burden of relative status) more heavily on the individual than in past historical periods (Beck 1998; Bourgois 2003). Evolutionary and animal behavior studies suggest particularly pathogenic results for male behavior. With the disappearance of more stable ways to define and delimit social groups, Sapolsky has suggested that the individual salience of social comparison

across class lines may have actually *gained* purchase over the 20th century, with effects most concentrated on psychobehavioral functioning (Sapolsky 1999). For males, behavioral responses to status inequity and social instability involve significant risk-taking and violent behavior (Bjorklund and Pellegrini 2000). Meanwhile, the increasing technological sophistication and widespread availability of mass-produced alcohol and other addictive substances, firearms and other weapons, and automobiles has provided particularly lethal tools for risk-taking and violent behavior.

Clearly, more work is needed to hone the relationship between historical analyses of social change and social hierarchy to demographic trends within and across countries during the third epidemiologic transition. This paper represents the beginnings of a theoretical and empirical stance with which to conduct such analyses. The use of demographic indicators for the third epidemiologic transition has allowed us to locate the concurrent emergence of male mortality differentials with the potential unmasking effects of social, technological, and medical change beginning during the turn of the century. Harder to locate with strictly demographic data is the proposal that social and technological change during this period actually *accentuated* and enabled male-biased risk processes leading to destructive and self-destructive behaviors among men. However, such ideas are testable. For example, future analyses could track the marketing and widespread availability of tools for mutual male destruction and self-destruction (particularly the acute tools of suicide and homicide), match such availability with indices of social inequality and economic disruption likely to motivate such behaviors, and compare such trends with demographic markers for the emergence of male mortality disadvantages within and across countries.

This study represents the integration of evolutionary life history, historical, sociological, and biological approaches to help understand the emergence of male mortality differentials in the western world. In doing so, it uses a new indicator of epidemiologic transition states (changed in IQR) in a rigorous international comparison to extract the historical timing and age-range of the emergence of excess male mortality in the industrialized world. With the effects of acute wartime shocks removed, we are faced with a robust pattern indicating the peak and stabilization of excess male mortality in the 1950s, at the same time as the reduction of IQR begins to plateau. Historical, biodemographic, and evolutionary analytic frames allow the beginnings of leverage with which to understand such trends. We hope such blends of theory and empirical analysis will inspire future cross-disciplinary collaborations and international comparisons, particularly with respect to cause-specific mortality and country-specific historical data.

REFERENCES

- Beck,U. 1998. *World Risk Society*. Cambridge, UK: Polity Press.
- Bjorklund,D.F. and A.D.Pellegrini. 2000. "Child development and evolutionary psychology." *Child Development* 71(6):1687-708.
- Booth,A., D.R.Johnson, and D.A.Granger. 1999. "Testosterone and men's health." *Journal of Behavioral Medicine* 22(1):1-19.
- Bourgois,P. 2003. "Crack and the political economy of social suffering." *Addiction Research and Theory* Vol 11(1) Feb 2003, 31-37).
- Carey,J.R. 1997. "What Demographers Can Learn from Fruit Fly Actuarial Models and Biology." *Demography* 34(1):17-30.
- CDC. 1999. "Infant and Mathernal Mortality in the United States: 1900-1999." *Population & Development Review* 25(4):821-6.
- Chapman Walsh,D., G.Sorensen, and L.Leonard. 1995. "Gender, Health, and Cigarette Smoking." Pp. 131-71 in *Society and Health*, edited by Benjamin C.Amick III., Sol Levine, Alvin R.Tarlov, and Diana Chapman Walsh. New York, NY: Oxford University Press.

Chisholm, J.S. 1999. *Death, hope and sex: Steps to an evolutionary ecology of mind and morality*. New York, NY, US: Cambridge University Press.

Dabbs, J.M., Jr., J.K. Riad, and S.E. Chance. 2001. "Testosterone and ruthless homicide." *Personality & Individual Differences* 31(4) Sep 2001):England, www.

Daly, M. and M. Wilson. 1985. "Competitiveness, risk taking, and violence: The young male syndrome." *Ethology & Sociobiology* 6(1):59-73.

Daly, M., M. Wilson, and S. Vasdev. 2001. "Income inequality and homicide rates in Canada and the United States." *Canadian Journal of Criminology* 43(2):219-36.

Durkheim, E. 1897. *Suicide*. New York, NY: The Free Press.

Edwards, R.D. and S. Tuljapurkar. 2005. "Inequality in Life Spans and a New Perspective on Mortality Convergence Across Industrialized Countries." *Population and Development Review* 31(4):645-74.

Flinn, M.V., R.J. Quinlan, S.A. Decker, M.T. Turner, and B.G. England. 1996. "Male-female differences in effects of parental absence on glucocorticoid stress response." *Human Nature* 7(2):125-62.

Fogel, R.W. 2004. *The Escape from hunger and Premature Death, 1700-2100*. Cambridge: Cambridge University Press.

- Gage,T.B. 1994. "Population Variation in Cause of Death - Level, Gender, and Period Effects." *Demography* 31(2):271-96.
- Greenhalgh,S. 1990. "Toward A Political-Economy of Fertility - Anthropological Contributions." *Population and Development Review* 16(1):85-106.
- Henry,L. 1989. "Men's and Women's Mortality in the Past." *Population: An English Selection* 44(1):177-201.
- Hueveline,P. 2002. "An international comparison of adolescent and young adult mortality." *Annals of the American Academy of Political and Social Science* 580):172-200.
- Kalben,B.B. 2002. "Why Men Die Younger: Causes of Mortality Differences by Sex." *Society of Actuaries Monograph* M-LI01-1).
- Lindstrom,J. 1999. "Early development and fitness in birds and mammals." *Trends in Ecology and Evolution* 14(9):343-8.
- Lopez,A.D., G.Caselli, and T.Valkonen (eds.). 1995. *Adult Mortality in Developed Countries: From Description to Explanation*. Oxford: Clarendon Press.
- Marmot,M. 2004. *The Status Syndrome: How Social Standing Affects our Health and Longevity*. New York, NY: Times Books.

- Massey,D.S. 1996. "The age of extremes: Concentrated Affluence and poverty in the twenty first century." *Demography* 33(4):395-412.
- Mazur,A. 1995. "Biosocial models of deviant behavior among male army veterans." *Biological Psychology* 41(3):271-93.
- McKeown,T. and R.G.Record. 1962. "Reasons for the Decline of Mortality in England and Wales during the Nineteenth Century." *Population Studies* 16(2):94-122.
- McNamara,J.M. and A.I.Houston. 1996. "State-dependent life histories." *Nature* 380(6571):215-21.
- Mooney,G. 2002. "Shifting sex differentials in mortality during urban epidemiological transition: the case of Victorian London." *International Journal of Population Geography* 8(1):17.
- Nathanson,C.A. 1984. "Sex Differences in Mortality." *Annual Review of Sociology* 10):191-213.
- Olshansky,S.J. and A.B.Ault. 1986. "The fourth stage of the epidemiologic transition: the age of delayed degenerative diseases." *Milbank Memorial Fund Quarterly* 64(3):355-91.

- Omran,A. 1971. "Epidemiologic Transition - Theory of Epidemiology of Population Change." *Milbank Memorial Fund Quarterly* 49(4):509-&.
- Pampel,F.C. 1998. "National Context, Social Change, and Sex Differences in Suicide Rates." *American Sociological Review* 63(5):744-58.
- , 2002. "Cigarette Use and the Narrowing Sex Differential in Mortality." *Population and Development Review* 28(1):77-104.
- Pampel,F.C. and R.Gartner. 1995. "Age Structure, Socio-Political Institutions, and National Homicide Rates." *European Sociological Review* 11(3):243-60.
- Preston,S. 1976. *Mortality Patterns in National Populations*. New York, NY: Academic Press.
- Robine,J.M. 2001. "Redefining the stags of the epidemiological transition by a study of the dispersion of life spans: The case of France." *Population: An English Selection* 13(1):173-93.
- Sapolsky,R.M. 1983. "Endocrine aspects of social instability in the olive baboon (*Papio anubis*)." *American Journal of Primatology* 5(4):365-79.

-----, 1999. "The physiology and pathophysiology of unhappiness." P. 453 in *Well-being: the foundations of hedonic psychology*, edited by D.Kahneman, E.Diener, and N.Schwarz. New York: Russell Sage Foundation.

-----, 2004. "Social Status and Health in Humans and Other Animals." *Annual Review of Anthropology* 33):394-418.

Stearns,S.C. 2000. "Life history evolution: successes, limitations, and prospects." *Naturwissenschaften* 87(11):476-86.

Svensson,E. and B.C.Sheldon. 1998. "The social context of life history evolution." *Oikos* 83):466-77.

Szreter,S. 2004. "Industrialization and health." *British Medical Bulletin* 69(75):86.

Waldron,I. 1990. "What do we know about the causes of sex differences in mortality? A review of the literature." Pp. 45-63 in *The Sociology of Health and Illness, 3rd edn.*, edited by P.Conrad and R.e.Kern. New York, NY: St. Martin's Press.

Wilkinson,R. 1997. *Unhealthy Societies: The Afflictions of Inequality*. New York, NY: Routledge.

Wilkinson,R.G. and K.E.Pickett. 2006. "Income inequality and population health: A review and explanation of the evidence." *Social Science & Medicine* 62(7):1768-84.

Wilmoth,J.R. and S.Horiuchi. 1999. "Rectangularization revisited: Variability of age at death within human populations." *Demography* 36(4):475-95.

Figure 1. Contour Plot of Sex Ratio of Mortality Rates by Year and Age, Sex Ratio: $m(\text{age, male}) / m(\text{age, female})$, Sweden 1750-2000

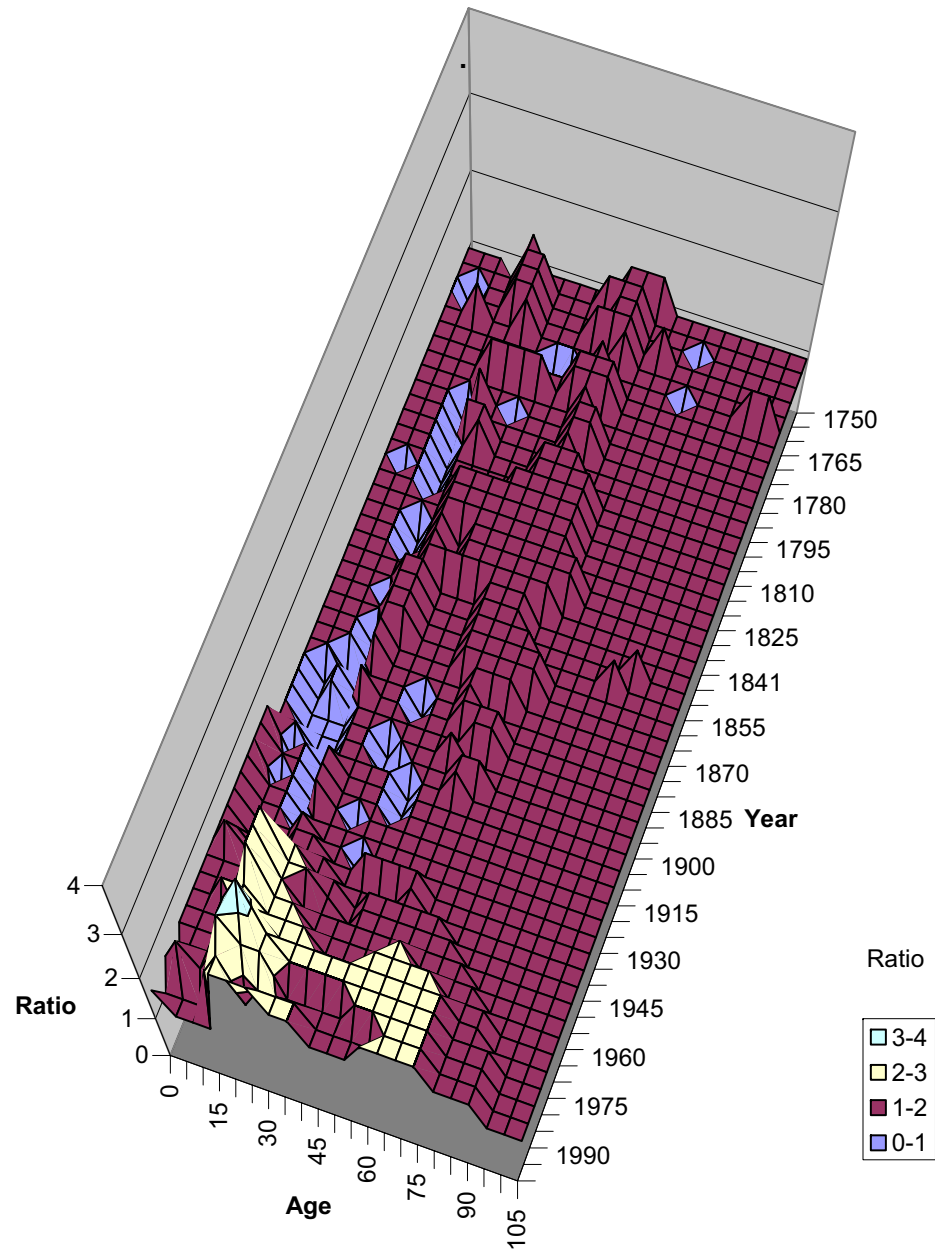
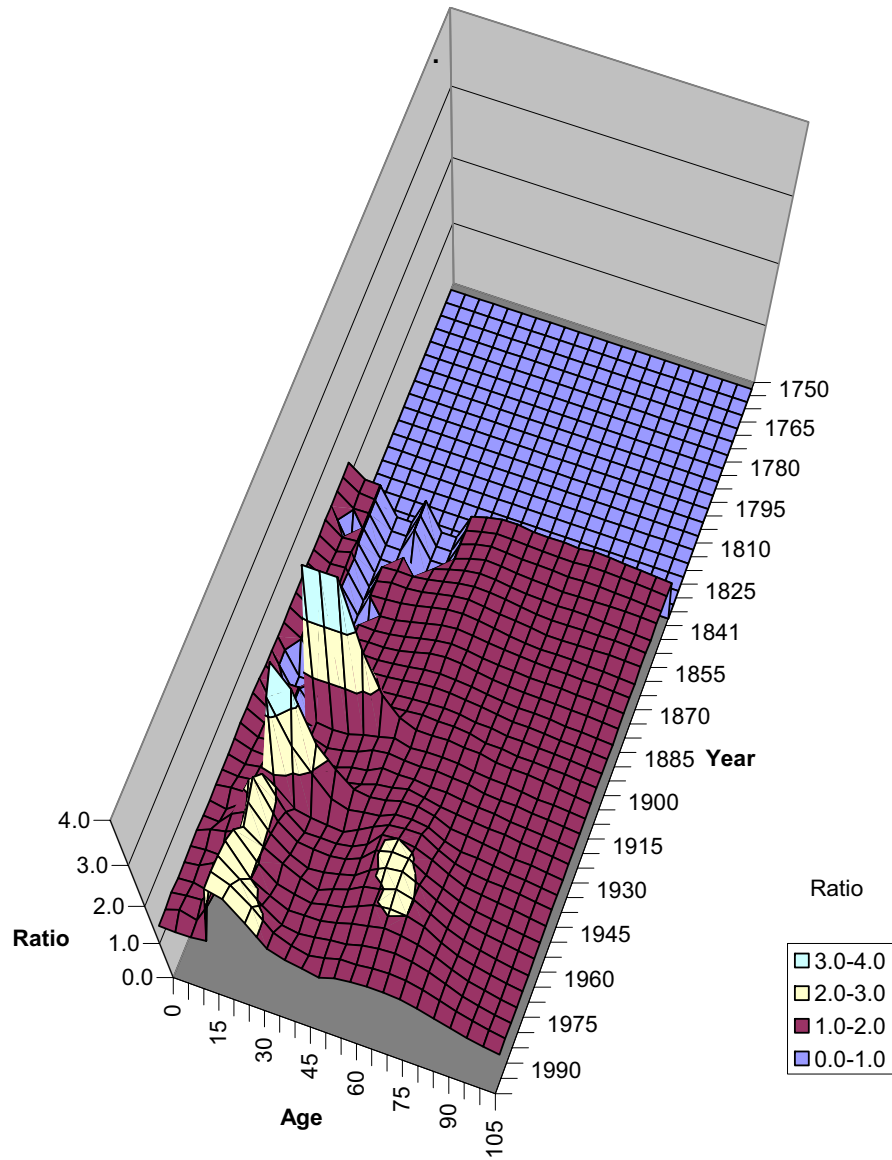
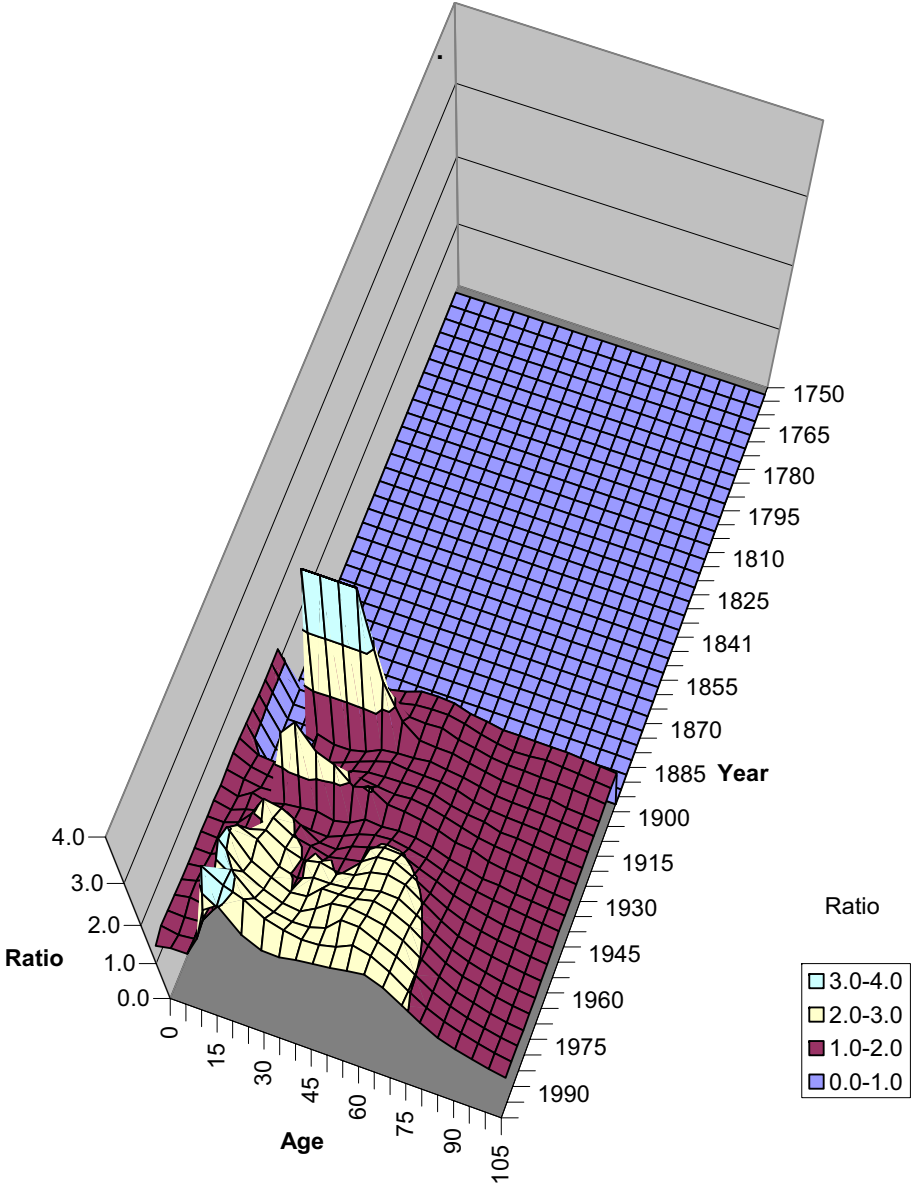


Figure 2. Contour Plot of Sex Ratio of Mortality Rates by Year and Age, Sex Ratio: $m(\text{age, male}) / m(\text{age, female})$, England & Wales 1841-2000*



*Note: The sex ratios of mortality in 1915-1919 for the ages 20 and 25 are not fully represented here because they exceed the limits of the graphic; the ratio at age 20 is 8.5 and at age 25 is 6.5.

Figure 3. Contour Plot of Sex Ratio of Mortality Rates by Year and Age, Sex Ratio: $m(\text{age, male}) / m(\text{age, female})$, France 1900-2000*



*Note: The sex ratios of mortality in 1915-1919 for the ages 20-35 are not fully represented here because they exceed the limits of the graphic; the ratio at age 20 is 8.0, at age 25 is 7.2, at age 30 is 5.3, and at age 35 is 4.3.

Figure 4. Contour Plot of Sex Ratio of Mortality Rates by Year and Age, Sex Ratio: $m(\text{age, male}) / m(\text{age, female})$, Spain 1908-2000*

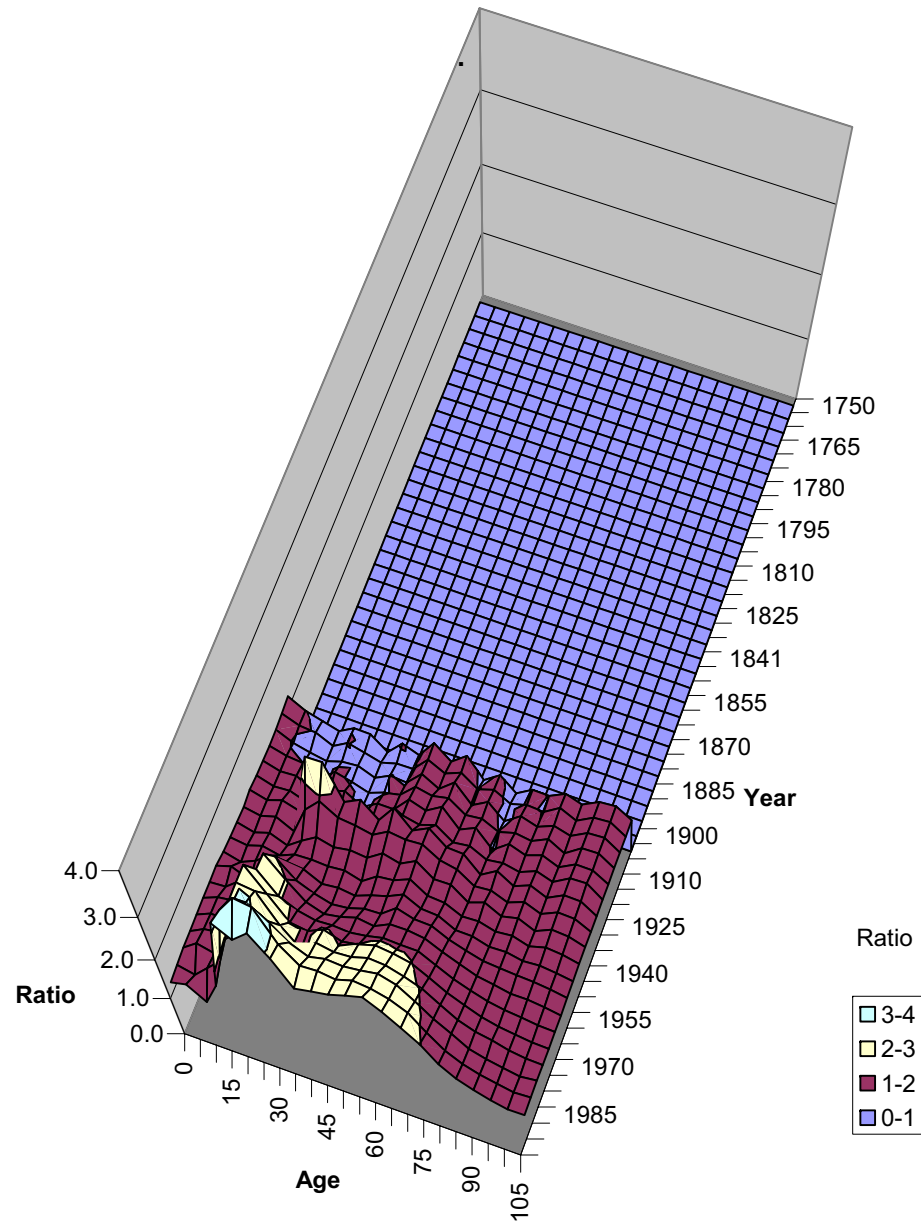


Figure 5: Historical Trends in Male and Female Mortality during Young Adulthood ($m(20, \text{male})$ and $m(20, \text{female})$) and Trends in the Indicator of Male Mortality Disadvantage during Young Adulthood (i.e. inverse-ratio of log mortality for males versus females), Sweden 1751-2000

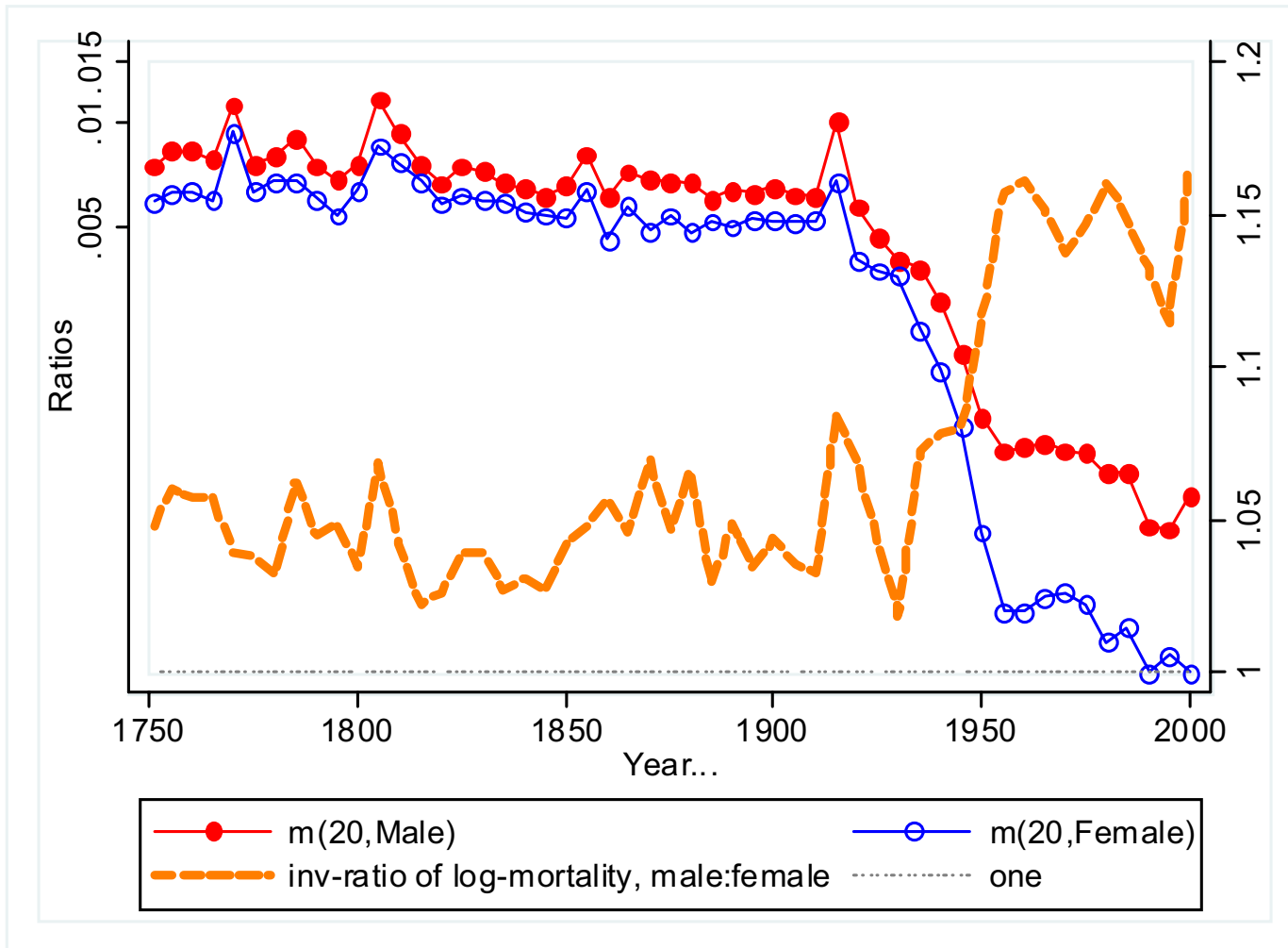


Figure 6: Historical Trends in Population Health Indicators (e_0 , IQR_0 , and IQR_{10}) and the Indicator of Male Mortality Disadvantage in Young Adulthood (i.e. inverse-ratio of log mortality for males versus females), Sweden 1751-2000

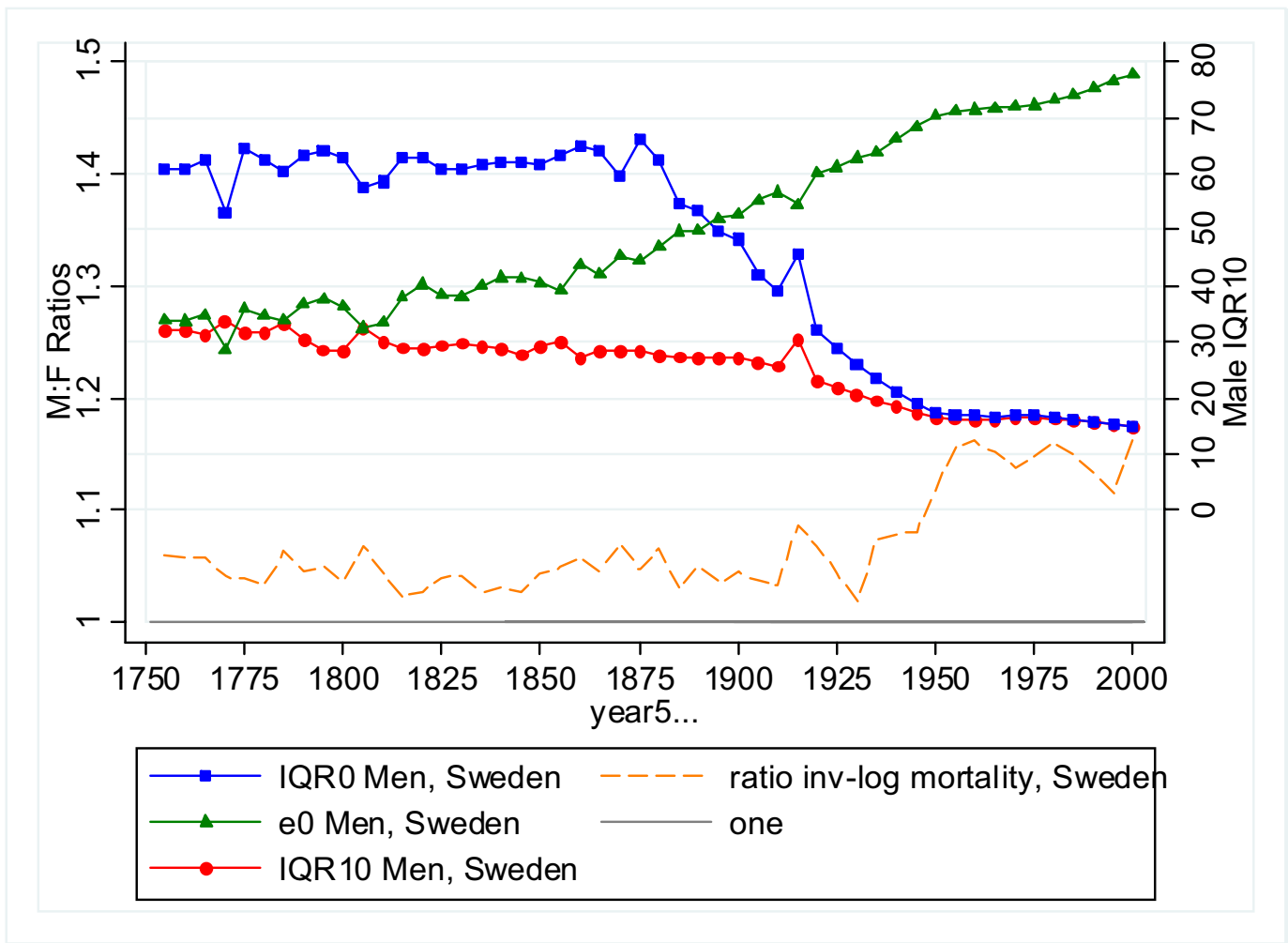
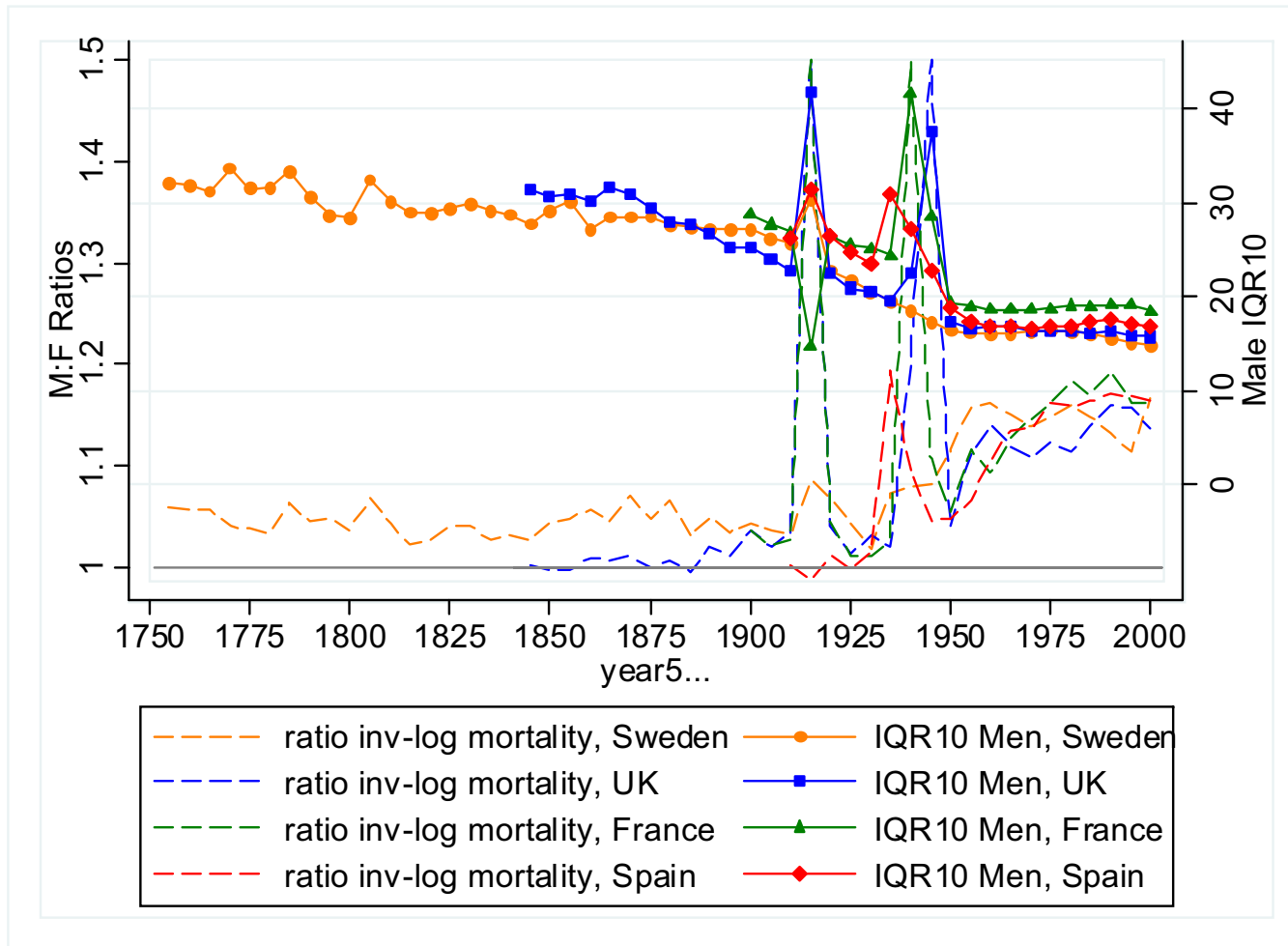


Figure 7: Historical Trends in Population Health and the Young Adult Male Mortality Disadvantage, Sweden 1751-2000, England & Wales (UK) 1841-2000, France 1900-2000, and Spain 1908-2000*



*Population health is measured using *IQR10* and the mortality disadvantage in young adulthood is measured using the inverse of the male:female ratio of log mortality at age 20. Please note that for clarity the ratios over 1.5 for the years 1915, 1940 and 1945 in France and the UK are not fully represented on the graph.