Life expectancy:

Past and future variations by gender in England & Wales

LSAP paper 2
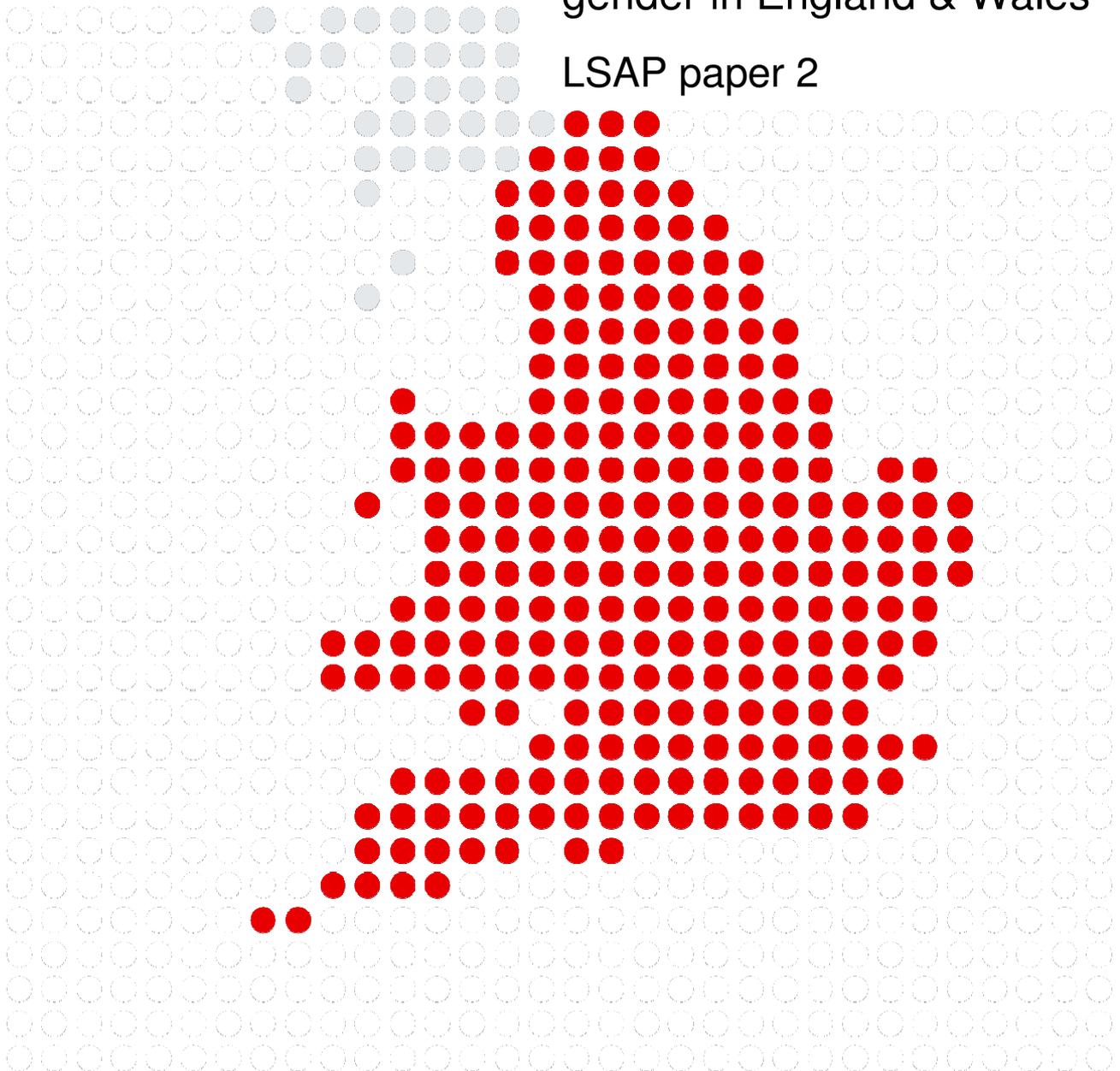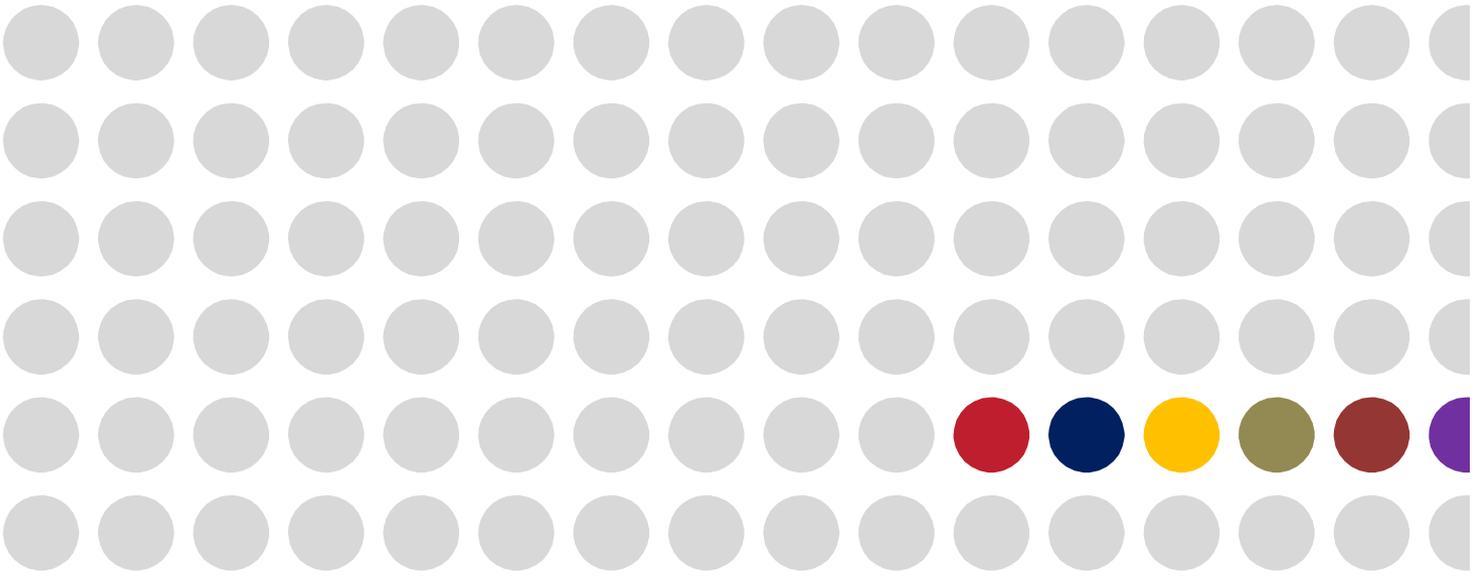
Foreword by Sir John Pattison
Acting Chair, Longevity Science Advisory Panel

Sir Derek Wanless
1947-2012

The Longevity Science Advisory Panel (LSAP) was established to bring actuarial science and epidemiology closer together to develop our understanding of past and current improvements in longevity and the factors which might drive future change.

The panel was established by Sir Derek Wanless, its first chairman. Sir Derek brought together a team of people with a spread of expertise, and professionals in Legal & General supported its work. The overall aim was to develop a coherent view of the impact on longevity of advances in biological and clinical sciences and changing social habits and attitudes.

Sir Derek set the agenda for LSAP's early work and established publication of position papers as a key component. Happily he was able to oversee the publication of the first of those papers which considered variations in life expectancy by socio-economic group and to initiate a second paper on gender differences.

Current LSAP members are in no doubt that they responded as much to Derek personally as to the intriguing challenges of the panel's work. His high standing in both the financial and public health sectors was unique and working with him was a delight. It is proving very difficult to find a replacement as chairman.

His death in 2012 shocked us all and we dedicate this second paper to his memory. We hope he would have approved the style, one rich in data and analysis. Hopefully it is a modest contribution to maintaining the pressure, as Derek wished to do, to confront the social and economic impacts of increasing longevity.

Longevity Science Advisory Panel Membership

Sir John Pattison, formerly Director of Research and Development at the Department of Health in England, who is well placed to assess the health services' ability to deliver the changes emerging from health research findings.

Klim McPherson, a public health academic at Oxford who is recognised for his work on obesity and the wide ranging detrimental effect this has on the health of the population and particular sub-groups.

Steven Haberman, Professor of Actuarial Science and Dean of Cass Business School is experienced in statistical modelling and mortality research, with the expertise to consider how to convert the findings of scientific research into a format for actuarial analysis.

Colin Blakemore, Professor of Neuroscience & Philosophy at the University of London, Emeritus Professor at Oxford and former Chief Executive of the Medical Research Council, brings knowledge of the biological basis of ageing and of trends in medical research and practice that are likely to influence life expectancy.

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Life expectancy: Past and future variations by gender in England and Wales

Section 1. Background

Preparing for a future where more people live longer is one of society's greatest challenges. The long-term social and economic impact on health and care services as well as on the provision of pensions, annuities and insurance needs a great deal of thought. It will require the best possible understanding of what has been happening and why, and the use of that knowledge to try to narrow the range of uncertainty about future trends.

For much of the recent past, forecasts about improvements in life expectancy have been wrong. They have incorporated an assumption that we would begin to see falls in the rate of improvement in life expectancy but the opposite has occurred. Over recent decades rates of improvement have risen to historically high levels. This has implications for many aspects of public policy and for the nature of society. There are variations in the figures, and in the improvements, by socio-economic group and by gender.

The Longevity Science Advisory Panel (LSAP) was set up by Legal & General to explore the impact that a range of factors may have on future life expectancy in the UK. This includes the drivers that are enhancing life expectancy, for example, medical advances and social change, as well as the inhibitors such as aspects of lifestyle and delays in development and use of treatments.

The purpose of LSAP's first paper[1] was to provide a synopsis of the past and possible future differentials in life expectancy by socio-economic group. Based upon a review of population information and recent literature, the purpose of this, the second paper, is to examine differentials in life expectancy by gender, where the increasing differential in favour of women has begun to reverse in recent decades.

In the next section we set out the data/resources used and examine the historical trends in life expectancy and mortality improvement by gender for England and Wales and other industrialised countries. In Section 3, some of the evidence examining the possible effects of lifestyle, pre-existing conditions, physiology and genetics on gender differences in life expectancy are considered. Section 4 then asks the open question: are gender differentials in life expectancy likely to persist into the future?

The paper is being published because the Panel is keen to share its conclusions with others and to support the continuing debate about the many implications of changing demographics. In addition the Panel is very keen to hear from others working on any related research which can aid understanding and be recognised in our future work. Comments on this paper should be emailed to longevity@landg.com. It is intended that periodic updates will be produced, drawing on any new evidence available to LSAP.

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[1] *Longevity Science Advisory Panel. (2012). Life expectancy: Past and future variations by socio-economic group in England & Wales.*
(http://www.longevitypanel.co.uk/docs/life-expectancy-by-socio-economic-group.pdf)

Section 2. Evidence of the gender gap: Available data

Section 2 highlights data (primarily 1840-2009) showing, by gender, past trends in: i) life expectancy (at birth and age 65) and ii) annual rates of improvement in mortality for England and Wales and other industrialised countries. The gender differences and the trends thereof are evident and in the final section we outline some international comparisons.

2.1. Historical trends in life expectancy for males & females (E&W, Human Mortality Database)

For England and Wales, Figures 1 and 2 illustrate data from the Human Mortality Database or HMD (www.mortality.org) showing estimates of period life expectancy at birth (e_0) and at age 65 (e_{65}) from 1841 to 2009. The figures show e_0 and e_{65} have increased for both males and females from 1841 to 2009. For males and females, e_0 had increased by 37.8 and 39.9 years to 78.4 and 82.5 years (2009) respectively. The life expectancy for males and females at age 65 had increased by 7.0 and 9.1 years to 18.0 and 21.0 years (2009) respectively.

The overall improvements for males and females between 1841 and 2009 in life expectancy at birth were very similar at 93% for men and 94% for women. Life expectancy at birth for women in 2009 was 5.3% higher than for men; slightly higher than the 5.1% recorded in 1841.

Figures 3-4 and 5-6 illustrate data (1841-2009) from the HMD showing the differences in period life expectancy between men and women at birth and at age 65.

The calculation of life expectancy at birth in a particular year is a function of death rates during that year. The figures for males in the 1910s and 1940s are therefore affected by deaths during the World Wars as the troughs in Figure 1 shows. Ignoring those two decades, as Figures 3-4 show, the difference between female and male life expectancy at birth peaked at 6.25 years in 1965-69 (8.3% of the female life expectancy at birth). In each subsequent five year period the difference has fallen in absolute and percentage terms. In 2005-09 the difference was down to 4.15 years (5.1%). At that percentage level it has returned close to the averages seen in the mid-1800s, raising an interesting question: Will it stabilise there or will it continue to fall to levels lower that any seen since the 1840s?

Figures 5-6 shows the data for differences in female and male life expectancy at age 65. The absolute difference in years peaked at 4.01 in 1980-84 whilst the % difference (expressed as a % of female life expectancy at age 65) peaked at 24.3% in 1970-1974. Since then both peaks, the absolute and percentage figures have consistently fallen to 2.69 years and 13.3% in 2005-09.

These changes in the gender gap in life expectancy will be discussed alongside comparisons with other developed nations in subsequent sections.

Figure 1. Male and female life expectancy at birth (E&W, 1841-2009)
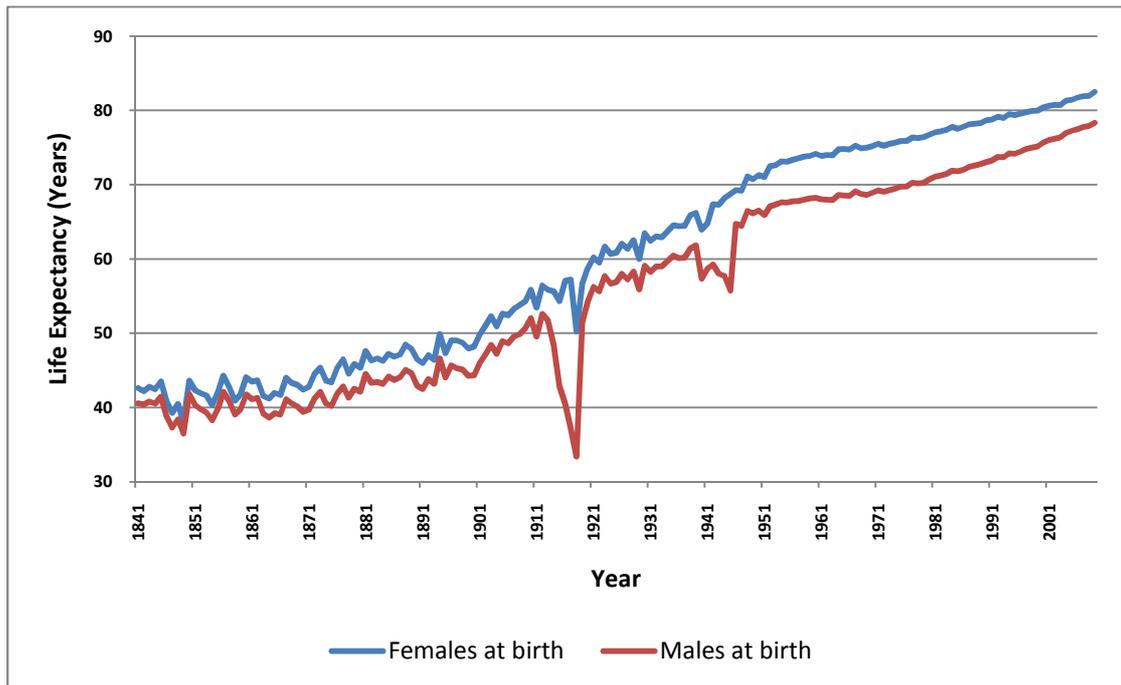
Figure 2. Male and female life expectancy at age 65 (E&W, 1841-2009)
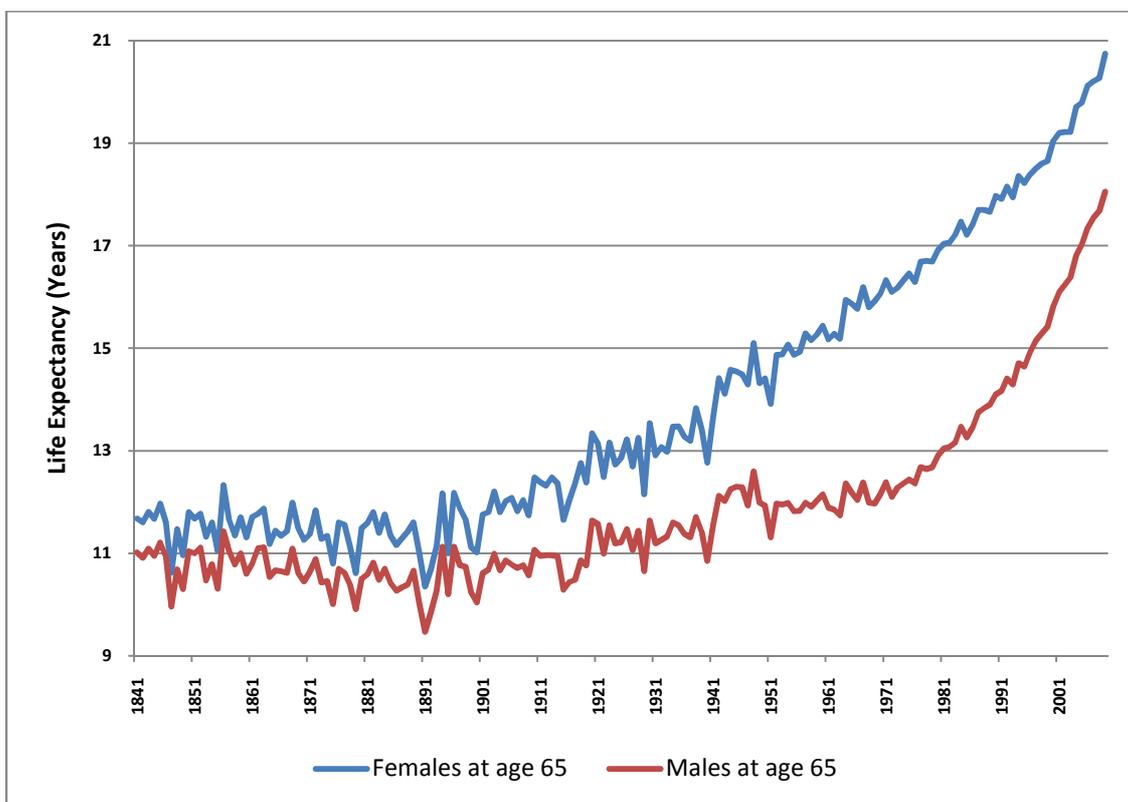
Figure 3. Difference between male and female period life expectancy at birth (England & Wales, 1841/44 – 2005/09)
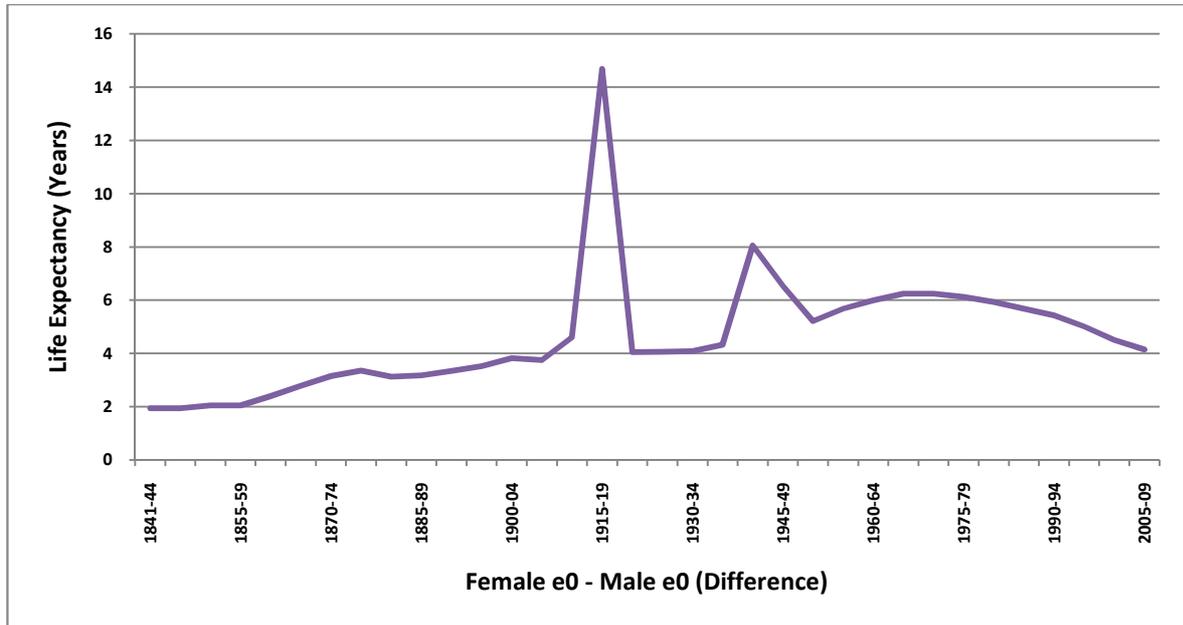
Source: England and Wales, Total Population, Life tables (period 1x5), Males, Females and Total Population. Last modified: 03-Nov-2010, MPv5 (May07).
© Human Mortality Database. University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany). (www.mortality.org)

Figure 4. Difference between male and female period life expectancy at birth: Difference as percent of female life expectancy at birth (England & Wales, 1841/44 – 2005/09)
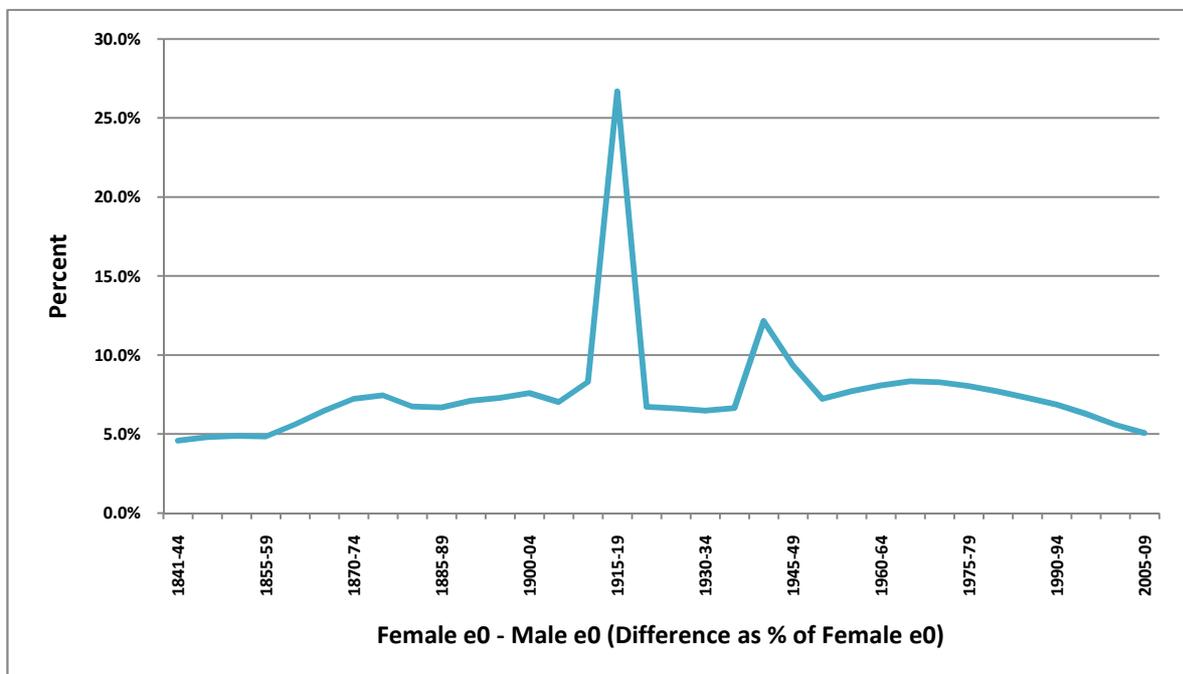
Source: England and Wales, Total Population, Life tables (period 1x5), Males, Females and Total Population.   Last modified: 03-Nov-2010, MPv5 (May07). © Human Mortality Database. University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany). (www.mortality.org)

Figure 5. Difference between male and female period life expectancy at age 65 (England & Wales, 1841/44 – 2005/09)
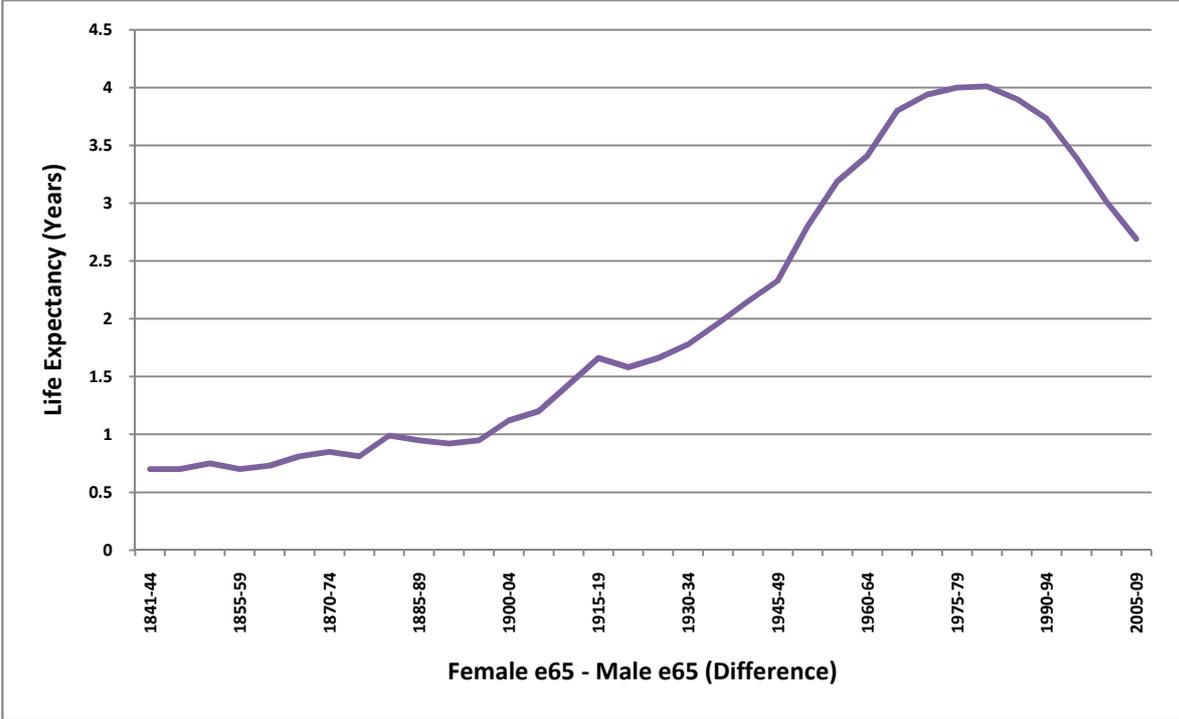
Figure 6. Difference between male and female period life expectancy at age 65: Difference as percent of female life expectancy at age 65 (England & Wales, 1841/44 – 2005/09)
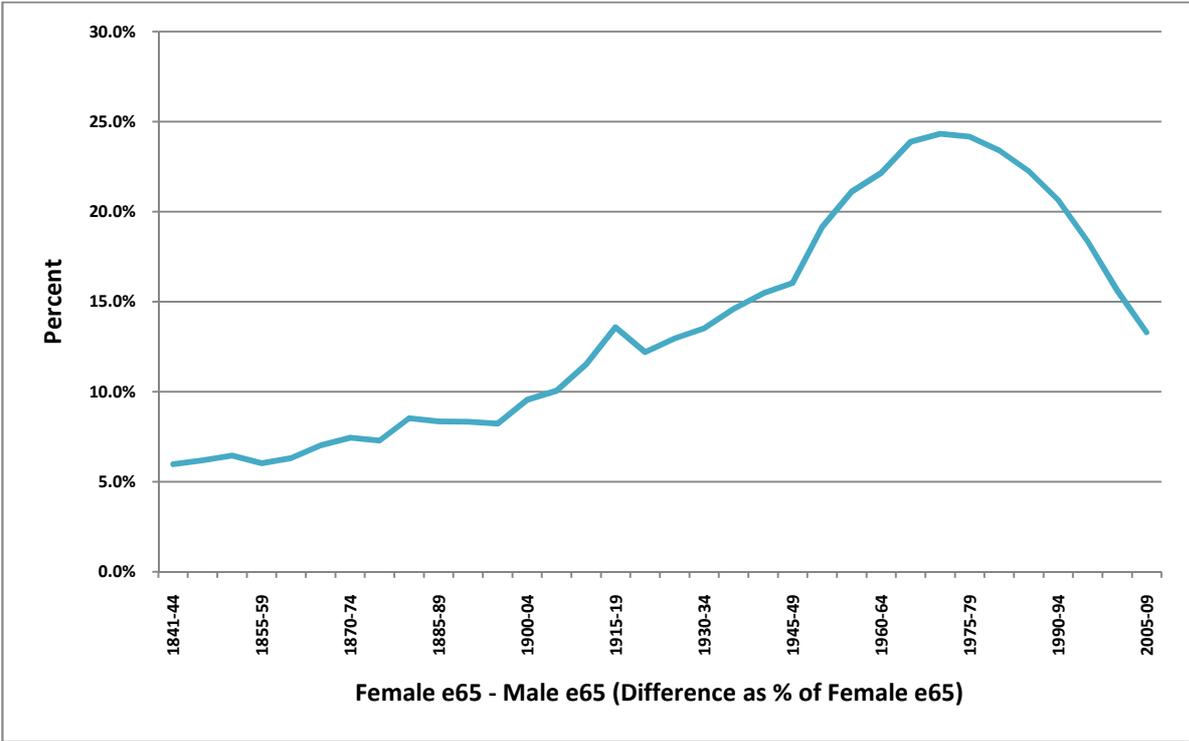
2.2. Historical trends in annual rates of mortality improvement for males and females (England & Wales, Continuous Mortality Investigation)

In 2009, the Working Party for the Continuous Mortality Investigation (CMI) published Working Papers 38 and 39 and featured a prototype mortality improvement projection model. Using data from the HMD the Working Party conducted an analysis of past trends in mortality improvement for 10 year age bands over successive 25 year periods.

The annual rate of improvement in mortality, in percentage terms, can be described as the "pace of change in mortality rates" and defined as $100(1 - Q_{x,t}/Q_{x,t-1})$ where $Q_{x,t}$ is the probability of death for a person age x over 1 year at time t. To derive the rate of improvement for an age group, the Working Party first calculated the average mortality rate for the 3 year period centred on the beginning and end of each period (e.g. mortality improvement for 1979-2004 is the average annual rate of change based on the mortality rates calculated for the period 2003-05 and the period 1978-80).

Figure 7 shows that for most age groups, the rates of improvement in mortality for males have been much higher in the previous 25 year period (1979-2004) than in any other period. The rapid improvements experienced within the last 25 years followed a quarter century over which there was only little to moderate change in mortality rates for ages 40 to 89. The Working Party attributes the changes to the initial "drag down" effect of smoking in the earlier period, with the corresponding increase in mortality improvement due to decreases in smoking prevalence alongside a general decrease in cardiovascular mortality.

Rates of change for males aged 40-89 over the last 25 year period was 2.1% p.a.. The figure for the preceding 125 years was 0.5% p.a.. For females, Figure 8 also shows rapid increases in rates of mortality improvement in the last 25 year period as compared with the previous 25 year. Rates of change for females aged 40-89 over the last 25 year period was 1.7% p.a., 0.4% p.a. lower than for males. The figure for the preceding 125 years was 0.8% p.a., 0.3% p.a. higher than for males.

The Working Party reiterated previous evidence for England & Wales showing persistent year of birth cohort features (particularly the 1931 cohort for both men and women) along with a more general increase in mortality improvement across a wide age range over the past 25 years.[2]

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[2] *Continuous Mortality Investigation . CMI Working Paper 39. A prototype mortality projections model: Part two – Detailed analysis, 2009. Institute of Actuaries and Faculty of Actuaries, p.5.*

Figure 7. Average annual rate of improvement in mortality for males (England & Wales) by 25 year period and age grouping
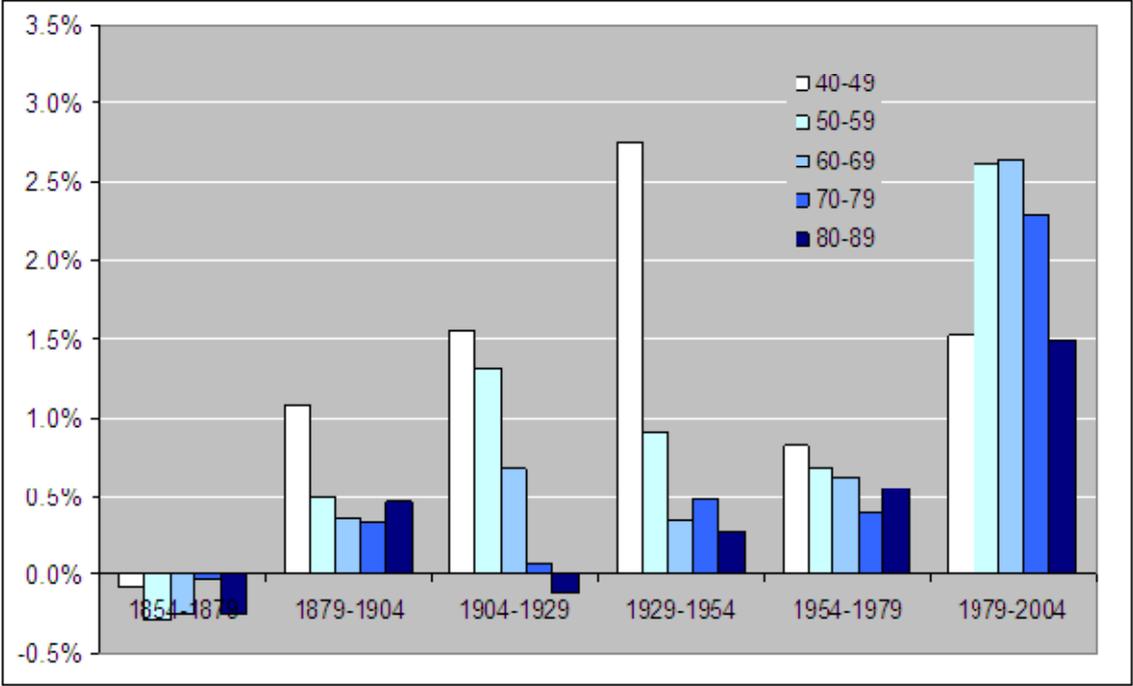
Source: Reproduced from Figure 4.7. Average annual rate of improvement for males in England and Wales, successive periods of 25 years, by age group. Continuous Mortality Investigation Working Paper 39. A Prototype Mortality Projections Model: Part Two – Detailed Analysis. Institute of Actuaries and Faculty of Actuaries, 2009, p.46. © 2009 Institute of Actuaries and Faculty of Actuaries

Figure 8. Average annual rate of improvement in mortality for females (England & Wales) by 25 year period and age grouping
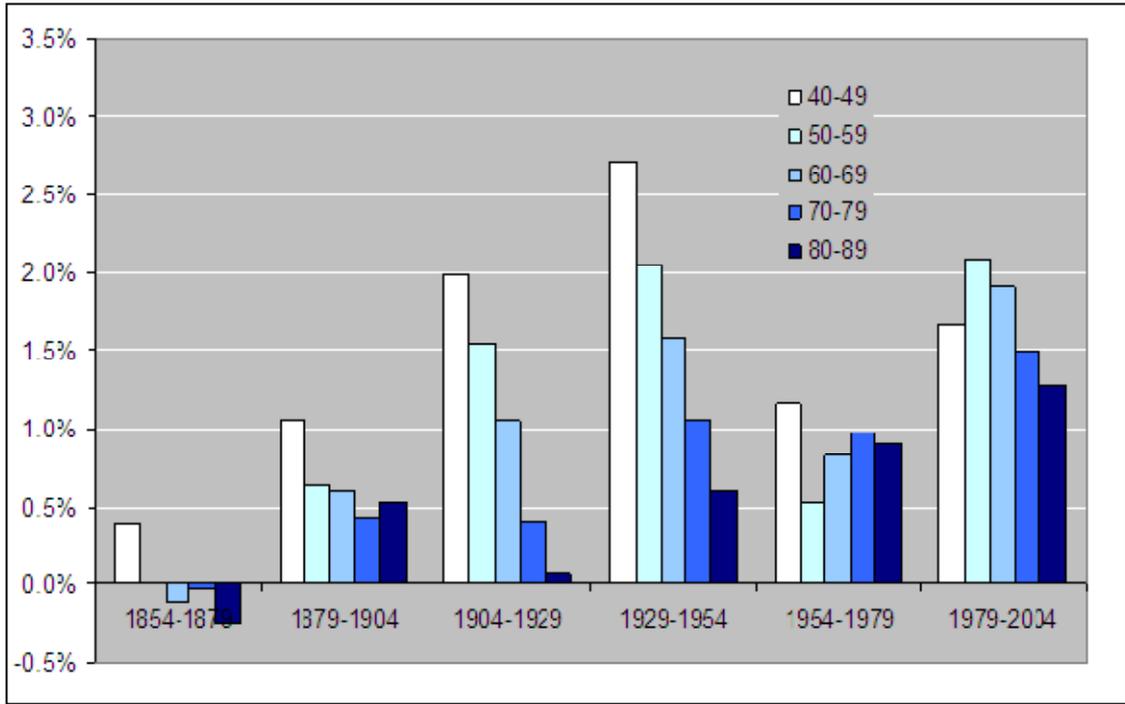
Source; Reproduced from Figure 4.8. Average annual rate of improvement for females in England & Wales, successive periods of 25 years, by age group. Continuous Mortality Investigation Working Paper 39. A Prototype Mortality Projections Model: Part Two – Detailed Analysis. 2009, p.47. © 2009, Institute of Actuaries and Faculty of Actuaries

2.3. Historical trends in gender differences in life expectancy (International comparisons)

In Working Paper 39, the Working Party for the Continuous Mortality Investigation (CMI) compared past trends in mortality improvement for England & Wales with 6 other developed nations. The 6 countries used as benchmarks for comparison were Belgium, Denmark, France, Netherlands, Norway and Sweden. They were selected due to the availability of data for the 150 year period.

Tables 1 (males) & 2 (females) show the average annual rate of mortality improvement by age group for both England and Wales and the comparative group. They show that, for both genders, the England & Wales pattern of long-term improvement by age is very similar to this international average.

Table 1. Comparison from 1854-2004 of the average annual rate of improvement in mortality for males (England & Wales vs. 7 countries) by age grouping

Average Annual Rate of Improvement in Mortality (Males)		
Age Group	England & Wales	International Average *(7 Countries)*
40-49	1.3%	1.2%
50-59	1.0%	0.9%
60-69	0.7%	0.8%
70-79	0.6%	0.6%
80-89	0.4%	0.4%
All Ages (40-89)	0.8%	0.8%

Source: Reproduced from Table 4.1. Average annual rate of improvement for males in England & Wales and an average of seven countries, 1854-2004, by age group. Continuous Mortality Investigation Working Paper 39. A Prototype Mortality Projections Model: Part Two – Detailed Analysis. Institute of Actuaries and Faculty of Actuaries., 2009, p.46. © 2009 Institute of Actuaries and Faculty of Actuaries

Table 2. Comparison from 1854-2004 of the average annual rate of improvement in mortality for females (England & Wales vs. 7 countries) by age grouping

Average Annual Rate of Improvement in Mortality (Females)		
Age Group	England & Wales	International Average *(7 Countries)*
40-49	1.5%	1.5%
50-59	1.1%	1.1%
60-69	1.0%	1.1%
70-79	0.7%	0.9%
80-89	0.5%	0.6%
All Ages (40-89)	1.0%	1.0%

Source: Reproduced from Table 4.2. Average annual rate of improvement for females in England & Wales and an average of seven countries, 1854-2004, by age group. Continuous Mortality Investigation Working Paper 39. A Prototype Mortality Projections Model: Part Two – Detailed Analysis. Institute of Actuaries and Faculty of Actuaries, 2009, p 47. © 2009 Institute of Actuaries and Faculty of Actuaries

International comparisons of gender differences in period e_0 and e_{65} are shown in both Figure 9/Table 3 and Figure 10/Table 4 respectively. For e_0, Figure 9/Table 3 show that until 1970/80, general declines in mortality (especially since 1850) within many industrialised countries had been accompanied by a widening differential in gender life expectancy (Female e_x – Male e_x). In Russia the gap is particularly high. However (and with the exception of Japan), since 1970/80 this widening gap between women and men had peaked and begun to narrow. Within England and Wales, the gender differences in life expectancy had begun to narrow from 1970. The differences between women and men in life expectancy at birth had declined from 6.3 years (1970) to 4.1 years (2009).

Similarly, for e_{65}, Figure 10/Table 4 show that until 1980/90, general declines in mortality (particularly since 1850) had also been accompanied by an increasing differential in gender life expectancy. With the exception of Japan and Russia, this increasing gap had peaked in the 1980/90 period and begun to narrow. Within England and Wales, the gender differences in life expectancy at age 65 had begun to narrow since 1980. The differences between women and men in life expectancy at age 65 had declined from 4.01 years (1980) to 2.69 years (2009).

Table 4 shows that, in 1930, the gender difference in period life expectancy at age 65 in a number of European countries was low. For Sweden, the figure was 0.58 years, for Denmark 0.44 years, for the Netherlands 0.47 years and for Norway 0.72 years.

Analysis of lifestyle and other factors by country and by gender are useful in adding to our understanding about, the impact, for example, of smoking or alcohol effects.

Second order gender differentials in life expectancy were also calculated. Defined as the difference between 2 time points in Female e_x – Male e_x, the right most column in Tables 3 and 4 show, for each country, the 2nd order differentials between the most current year and the year with the widest gender differential. The figures illustrate both the direction and magnitude of the change in gender differentials within the period of investigation (1850 to 2009).

Figure 9. International comparison of gender differences in period life expectancy (years) at birth (HMD)
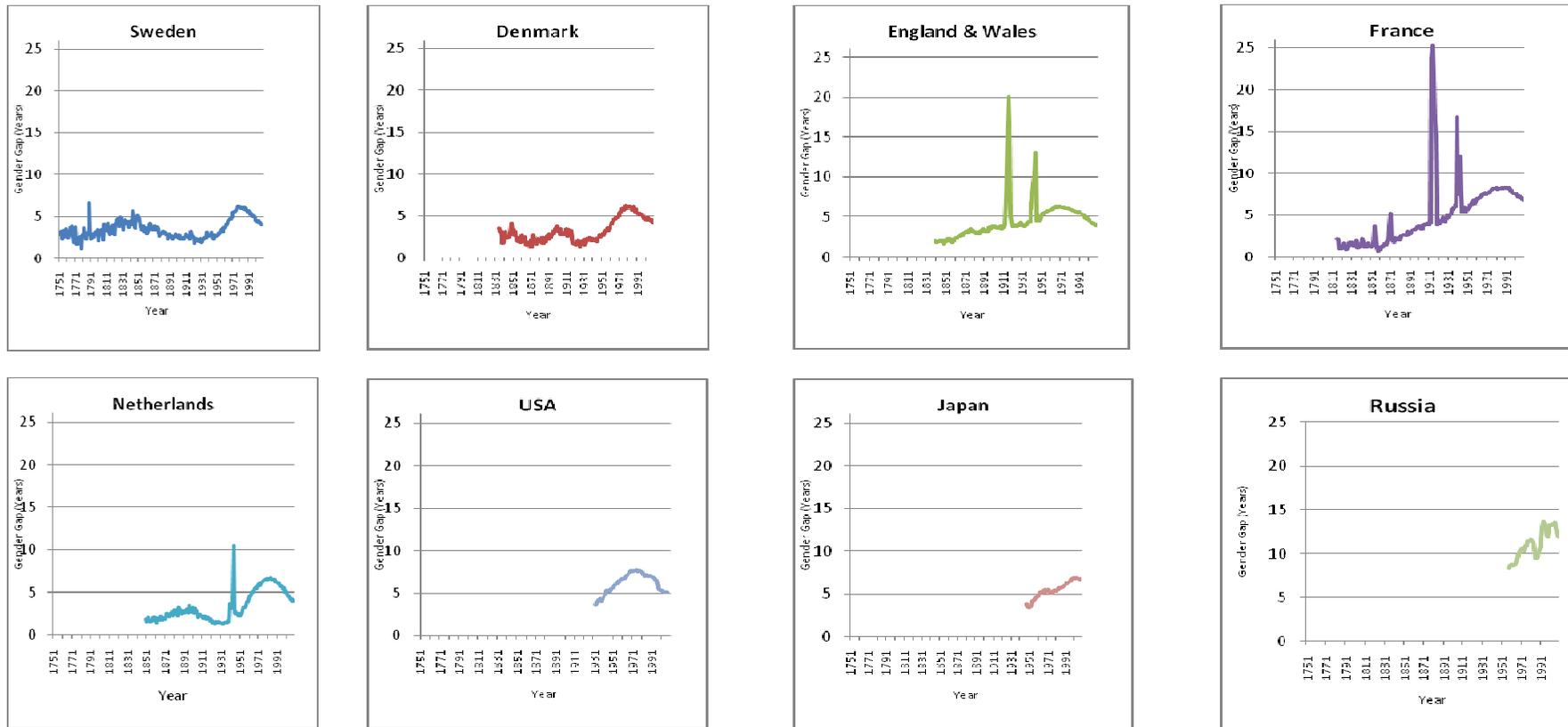
Source: Sweden, Denmark, E&W, France, Netherlands, Norway, USA, Japan. Total Population, Life tables (period 1x1), Males and Females. Last modified: 12-Dec-2008, MPv5 (May07). © Human Mortality Database. University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany). (www.mortality.org)

Table 3. International comparison at 10 year intervals of gender differences in period life expectancy at birth: (HMD, 1850 to 2009)

Country/ Year	1850	1860	1870	1880	1890	1900	1910	1920	1930	1940	1950	1960	1970	1971	1980	1990	2000	2009	2nd Order Difference (1971 to Current)	2nd Order Difference (Highest Gender Gap to Current)
Sweden	5.12	3.67	3.66	3.01	2.69	2.83	2.62	2.69	2.07	2.75	2.61	3.64	4.98	5.42	6.07	5.58	4.64		-0.78	-1.43
Denmark	3.28	1.75	1.93	1.9	2.33	3.32	3.22	1.56	1.92	1.91	2.42	3.55	5.05	5.35	6.01	5.71	4.68	4.20	-1.15	-1.81
England & Wales	1.80	2.29	3.00	3.25	3.58	3.86	3.82	4.44	4.36	6.60	4.8	5.9	6.3	6.28	6.02	5.65	4.73	4.13	-2.15	-2.47
France	1.37	1.02	3.71	1.99	2.75	3.63	3.86	3.83	5.01	16.67	5.76	6.59	7.44	7.56	8.24	8.24	7.56	6.71	-0.85	-9.96
Netherlands	1.91	2.09	2.01	2.86	2.64	2.81	2.19	1.90	1.45	3.7	2.27	3.86	5.69	5.78	6.7	6.26	5.04	4.11	-1.67	-2.59
Norway	3.53	2.64	3.38	2.79	2.91	3.38	3.11	2.65	3.03	5.40	3.34	4.53	6.31	6.2	6.77	6.38	5.42		-0.78	-1.35
USA										4.43	5.62	6.68	7.63	7.59	7.49	7.01	5.33		-2.26	-2.30
Japan											3.32	4.83	5.35	5.34	5.37	5.92	6.88	6.81	1.47	-0.07
Russia												8.64	10.37	10.53	11.58	10.56	13.26	11.96	1.43	-1.30

Figure 10. International comparison of gender differences in period life expectancy (years) at age 65 (HMD)
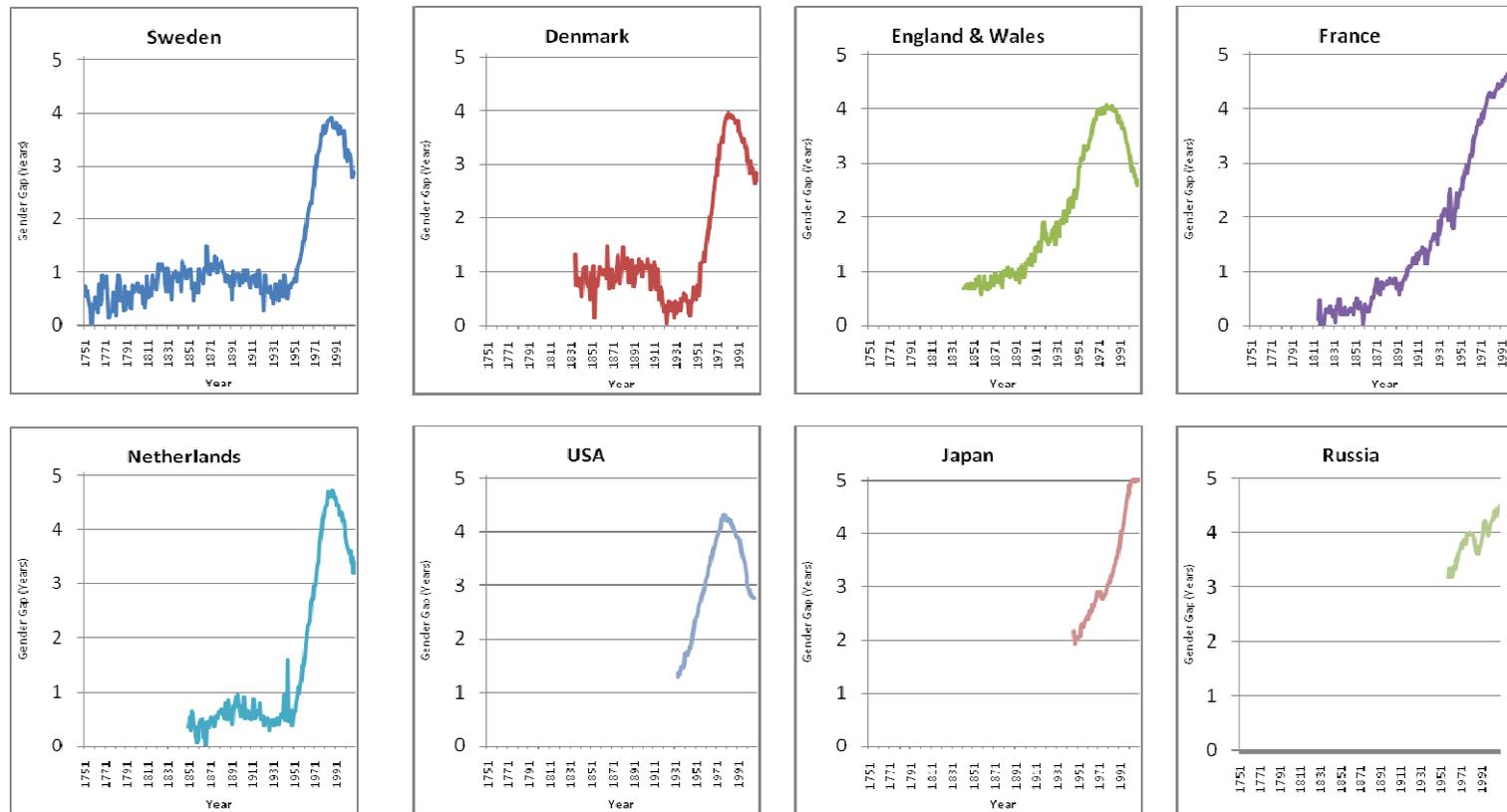
Source: Sweden, Denmark, E&W, France, Netherlands, Norway, USA, Japan. Total Population, Life tables (period 1x1), Males and Females. Last modified: 12-Dec-2008, MPv5 (May07). © Human Mortality Database. University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany). (www.mortality.org)

Table 4. International comparison at 10 year intervals of gender differences in period life expectancy at age 65: (HMD, 1850 to 2009)

Country/Year	1850	1860	1870	1880	1890	1900	1910	1920	1930	1940	1950	1960	1970	1971	1980	1990	2000	2009	2nd Order Difference (1971 to Current)	2nd Order Difference (Highest Gender Gap to Current)
Sweden	1.03	1.04	1.01	1.15	0.73	0.89	0.86	0.69	0.58	0.63	0.78	1.58	2.65	2.99	3.63	3.72	3.38		0.39	-0.34
Denmark	0.97	0.98	1.04	1.12	1.01	1.13	1.18	0.37	0.44	0.55	0.52	1.47	2.81	3.09	3.92	3.83	3.06	2.71	-0.38	-1.21
England & Wales	0.76	0.71	0.81	0.99	0.98	0.98	1.41	1.70	1.9	1.92	2.48	3.29	3.91	3.94	4.01	3.87	3.22	2.69	-1.25	-1.32
France	0.40	0.39	0.67	0.75	0.77	0.92	1.33	1.31	1.91	1.94	2.41	3.03	3.72	3.76	4.29	4.41	4.57	4.21	0.45	-0.36
Netherlands	0.33	0.43	0.34	0.64	0.55	0.56	0.5	0.52	0.47	0.58	0.52	1.50	2.94	2.96	4.4	4.58	3.89	3.36	0.4	-1.22
Norway	1.07	1.06	1.02	0.64	0.95	0.84	0.87	0.85	0.72	0.72	0.98	1.61	2.92	2.91	3.71	3.9	3.64		0.73	-0.26
USA										1.56	2.34	3.09	3.87	3.92	4.21	3.91	3.03		-0.89	-1.18
Japan											2.05	2.45	2.83	2.86	3.08	3.74	4.89	5.04	2.18	0.00
Russia												3.35	3.73	3.81	3.98	3.77	4.15	4.50	0.69	0.00

Section 3. Explanations for differences in gender life expectancy

We examine possible causes of gender differences in life expectancy by grouping them into three broad categories:

i) environmental conditions or lifestyle behaviours;
ii) mortality due to major categories of disease; and
iii) physiology and/or genetic makeup.

3.1. Differences in lifestyle behaviour (tobacco and alcohol consumption) and obesity between genders

3.1.1. Tobacco and alcohol

Several conditions (e.g., cancers of oesophagus, throat or liver) due to tobacco and/or alcohol consumption are inextricably linked. The attribution of mortality due to either of the risk factors can be difficult as any division into alcohol or smoking related causes can underestimate the impact of the other on mortality. This is further complicated by the large gender related differences in tobacco or alcohol consumption in the countries under review.

Nevertheless, in a study of 30 European nations (2003-2005), McCartney et al. (2011) calculated that smoking related deaths have accounted for 40-60% of the gender gap in the majority of the countries reported[3]. Within the same period, alcohol related deaths have contributed to approximately 20% of the gender gap in all cause mortality (typically higher in the Eastern European nations). The authors conclude the contribution of smoking related mortality to the gender gap in all cause mortality was greater than that of alcohol related mortality in all the countries examined.

In analysing the relationship between tobacco/cigarette consumption and the increase in gender differences in mortality, several studies have examined the time delay in smoking related mortality (primarily men) and the gender gap. A cohort pattern had been demonstrated showing that changes in cigarette consumption among men with the 20th century can help explain the gap in mortality during the period.

Examining USA data, Preston and Wang (2006) argued that the increase in smoking behaviour after World War II had partially obscured the reductions in mortality that would otherwise have occurred during the period. Similarly, the decrease in smoking behaviour over the previous two decades had exaggerated its improvements. The authors contend that, when smoking history was taken into account, mortality levels had actually declined by 56% during this post-war period.[4] Period specific estimates which did not take into account smoking history would show a decline of only 48%.

The authors argue that just as mortality improvements for older ages (during the past 50 years) had been inhibited by an increase in smoking behaviour, so mortality improvements (and the narrowing of the gender gap in life expectancy) in future would be accelerated by a reduction in smoking behaviour. In a subsequent paper, Wang and Preston (2009) provided survival probabilities (2004-2034) for males and females by taking into account their smoking history. They found that by 2034, the probability of a man surviving from age 50 to 85 is 22.5% greater when smoking is accounted for. The difference for women is 7.4% (Wang and Preston, p.397).

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[3] *Exceptions include Denmark, Portugal and France with smaller proportions and Malta with a higher proportion.*
[4] *Preston SH and Wang H.(2006). Sex mortality differences in the United States: The role of cohort smoking patterns. Demography; 43(4), p.641.*

Smoking is widely recognised as the main avoidable risk factor for coronary heart disease and select cancers.[5] In England and Wales, differing rates of lung cancer and ischaemic heart disease (conditions heavily influenced by cigarette consumption) between men and women have reflected the large gender differences in smoking patterns from 1960-1970 (Gjonca et al. 2005).[6] For example, work by Waldron (1995) has suggested that 50% of the gender gap in ischaemic heart disease (IHD) mortality could be attributed to differences in smoking behaviour[7]. Since the 1970s, however, the prevalence of cigarette smoking has generally declined; with substantial falls until the early 1980s. From 1974 to 2008, smoking prevalence for men was greater than for women. However, the gender gap in the proportion of smokers has narrowed with 2008 figures for the UK showing no statistical difference in male and female smoking rates (Table 5).

To further illustrate, we have used age-standardised death rates (direct method, European standard population) provided by the European Health for All Database (HFA-DB, http://data.euro.who.int/hfadb/), to derive the male/female ratio of death rates per 100,000 for alcohol and smoking related causes of death within the United Kingdom (1979-2009, refer Figure 11). The figures provide only a rough indicator of deaths which are related (but not directly attributable) to smoking or alcohol consumption.[8] Further information is provided in Appendix B.

Since 1979 the ratio has risen for alcohol related deaths (from approximately 2 to 2.4) and decreased for smoking related deaths (from approximately 2.14 to 1.7). Smoking and alcohol related death rates have declined (men and women) from 1979 to 2009. For men, figures indicate falls of 63.6% in smoking-related deaths and 29.0% for alcohol-related deaths. For women, figures show declines of 53.8% for smoking-related deaths and 40.0% for alcohol-related deaths.

Although smoking-related death rates have outpaced alcohol-related death rates during this period, the changing trends in smoking behaviour are reflected in an overall decline of its impact on mortality relative to alcohol consumption. For men, smoking-related death rates have declined from approximately 6 (1980-89) to 3.5 times (2000-09) that of alcohol-related deaths. For women, 2000-09 smoking related death rates were 4.5 times that of alcohol-related death rates. This suggests the strong downward trend in male smoking-related deaths could be a major contributor to the recent convergence in gender mortality.

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[5] Department of Health (1998). Smoking Kills – A white paper on tobacco. TSO: London.
[6] Gjonca A., Tomassini C., Toson B., Smallwood S. (2005). Sex differences in mortality, a comparison of the United Kingdom and other developed countries. Health Statistics Quarterly 26 Summer 2005.
[7] Waldron I. (1995). Contributions of biological and behavioural factors to changing sex differences in ischaemic heart disease mortality. (Eds.) Lopez A.D., Caselli G., and Volkonen T. Adult mortality in developed countries: From description to explanation. Pp. 161-178. Clarendon Press Oxford, England.
[8] Figures indicate selected causes of death known from the literature to be related to smoking or alcohol consumption.

Table 5. Changes in smoking rates by gender, 1995-2008 (International Comparison)

	Females			Males		
	1995	**2008**	**% Diff.**	**1995**	**2008**	**% Diff.**
UK	27.0	21.0	-22.2	29.0	22.0	-24.1
EU	22.0	19.2	-12.7	38.6	31.3	-18.9
Denmark	33.0	22.0	-33.3	38.0	24.0	-36.8
Norway	32.0	22.0	-31.3	33.0	21.0	-36.4
Sweden	23.6	16.7	-29.2	22.0	12.3	-44.1
Netherlands	31.0	25.0	-19.4	41.0	32.0	-22.0
France	21.0	22.3	6.2	36.0	30.6	-15.0

Source: Based on data from figure 2.6.2 Change in smoking rates by gender, 1995-2008 (or nearest year available) from OECD (2010), Health at a Glance: Europe 2010, OECD Publishing .http://dx.doi.org/10.1787/health_glance-2010-en. OECD (2010), Health at a Glance: Europe 2010, OECD Publishing. http://dx.doi.org/10.1787/health_glance-2010-en © OECD 2010, All rights reserved.

Figure 11. Male/Female ratio of death rates for smoking and alcohol related causes (United Kingdom, 1979-2009)
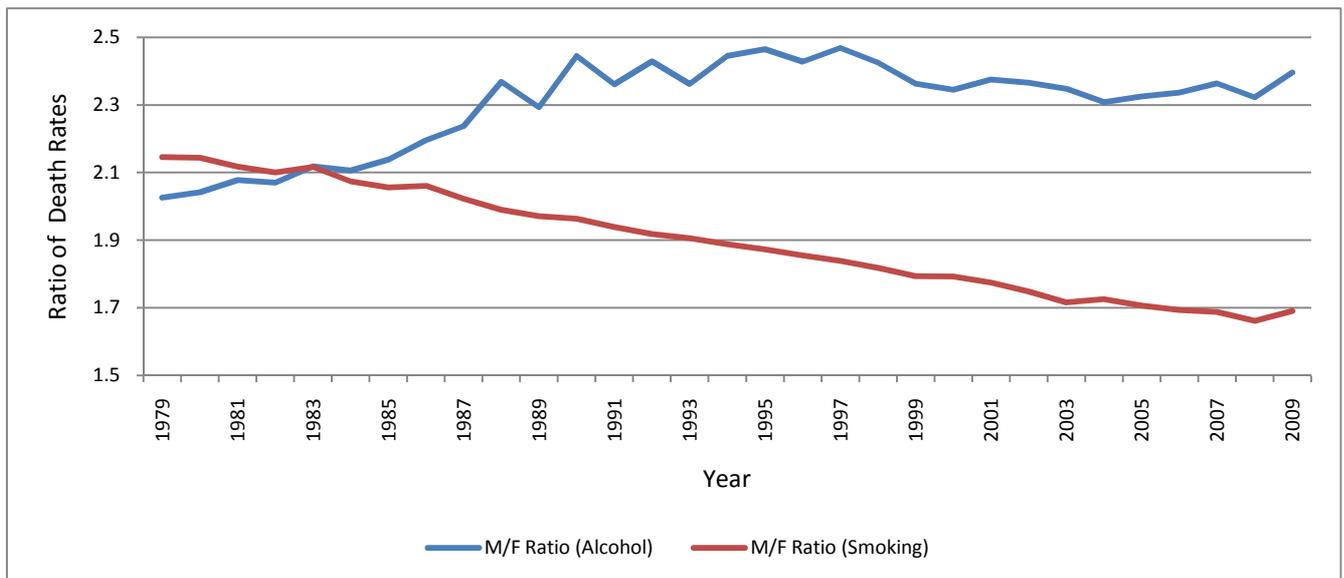
Source: Data from European Health for All Databases (HFA-DB). World Health Organization Regional Office for Europe. Accessed 11 November 2011. (http://data.euro.who.int/hfadb/). © Copyright World Health Organization (WHO) 2011. All Rights Reserved

3.1.2. Obesity

Obesity is a risk factor for a number of conditions including hypertension, cardiovascular disease, diabetes and respiratory diseases such as asthma. Recent analyses from the Organisation for Economic Co-operation and Development (OECD, 2010) indicate that more than half (50.1%) of adults within the European Union are either overweight (Body Mass Index or BMI 25-30 kg/m^2) or obese (BMI >30 kg/m^2).[9] Of the 27 EU countries (2010), obesity rates have more than doubled over the past 20 years for which data was available (Figure 12). In addition, the prevalence of overweight or obesity among adults is greater than 20% in at least 5 of the countries including the United Kingdom (Table 6). Outside of the EU, the prevalence of obesity among many industrialised countries has also increased.

Within the UK, current prevalence rates (2010) show small absolute differences between men and women. However, the relative risks of developing a number of chronic conditions can vary by gender. Table 7 shows the extent to which obesity increases the risk of developing a number of conditions relative to the non-obese population. For example, an obese woman is 12.7 times more likely to develop Type 2 Diabetes and 4.2 times more likely to have hypertension than a woman who is not obese. In contrast, an obese man is 5.2 times more likely to develop Type 2 Diabetes and 2.6 times more likely to have hypertension than a man who is not obese.

Figure 12. Increasing obesity rates among adults within the EU-27 nations

1 Luxembourg, Slovak Republic (2008) and the United Kingdom figures are based on health examination surveys, rather than health interview surveys.

Source: Figure 2.8.2. Increasing obesity rates among adults in EU countries from OECD (2010), Health at a Glance: Europe 2010, OECD Publishing, p.73. http://dx.doi.org/10.1787/health_glance-2010-en. OECD (2010), Health at a Glance: Europe 2010, OECD Publishing. http://dx.doi.org/10.1787/health_glance-2010-en © OECD 2010, All rights reserved. Original Data Sources: OECD Health Data 2010; Eurostat Statistics Database; WHO Global Infobase

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[9] *OECD (2010). Health at a Glance: Europe 2010. OECD Publishing (http://www.oecd-ilibrary.org/social-issues-migration-health/health-at-a-glance-europe-2010_health_glance-2010-en)*

Table 6. Obesity rates among adults within the EU-27 nations, 2008 or nearest year available

EU 27	Total (%)	Females (%)	Males (%)
Romania	7.9	8.0	7.7
Switzerland	8.1	7.7	8.6
Italy	9.9	9.1	10.8
Norway	10.0	8.0	11.0
Sweden	10.2	10.1	10.3
Netherlands	11.1	12.2	10.1
France	11.2	11.5	10.9
Denmark	11.4	11.8	11.0
Bulgaria	11.5	11.3	11.6
Austria	12.4	12.7	12.0
Poland	12.5	12.5	12.6
Germany	13.6	12.8	14.4
Belgium	13.8	14.4	13.1
Spain	14.9	14.7	15.1
Turkey	15.2	18.5	12.3
Portugal	15.4	16.1	14.6
EU	**15.5**	**15.6**	**15.4**
Cyprus	15.6	14.5	16.7
Finland	15.7	16.0	15.4
Slovenia	16.4	15.8	17.0
Slovak Republic[1]	16.9	16.7	17.1
Latvia	16.9	20.9	12.0
Czech Republic	17.1	17.1	17.1
Estonia	18.0	18.3	17.5
Greece	18.1	18.5	17.7
Hungary	18.8	18.0	19.6
Lithuania	19.7	19.2	20.6
Luxembourg[1]	20.0	18.8	20.9
Iceland	20.1	21.3	18.9
Malta	22.3	20.6	24.3
Ireland[1]	23.0	24.0	22.0
United Kingdom[1]	24.5	24.9	24.1

1 Data for Ireland, Luxembourg, the Slovak Republic and the United Kingdom are based on health examination surveys, rather than health interview surveys.

Table 7. Relative risk factors for obese people of developing selected diseases, by gender

England	Males	Females
Type 2 Diabetes*	5.2	12.7
Hypertension	2.6	4.2
Myocardial Infarction	1.5	3.2
Cancer of the Colon	3.0	2.7
Angina	1.8	1.8
Gall Bladder Diseases	1.8	1.8
Ovarian Cancer	-	1.7
Osteoarthritis	1.9	1.4
Stroke	1.3	1.3

Non-insulin dependent diabetes mellitus (NIDDM)

3.2. Access to health care

One of the factors that might contribute to the differentials in life expectancy by gender is variability between men and women in their access to health services and the management of their conditions. In the UK research on differentials in access to health care has focussed particularly on socio-economic circumstances and we summarised the findings in our first paper. It is clear that there are important interactions between gender inequalities and socio-economic circumstances, as there are with age and ethnicity, but in this paper we try to examine the effect of gender alone.

In 2008 the Department of Health of England published the final report of the Gender and Access to Health Services Study (Wilkins et al 2008). Although the list of references is extensive the report notes that the research base focussing specifically on the link between gender and use of health services is poor. Many of the studies that exist analyse data gathered before the various NHS service frameworks will have had their full effect and few use mortality as an outcome measure. Nevertheless some trends emerge.

With respect to cardiovascular disease the report concludes that women are more likely to delay seeking help, partly because IHD is regarded as a man's problem and partly because the symptoms of IHD in women can be atypical. Women are also less likely to be referred to specialists and receive less invasive treatment. These conclusions accord with other earlier studies (Raine, 2000; Sproston and Primatesta, 2003). Similar findings have been reported from other countries. In Finland men receive more active treatment than women (Kattainen et al., 2006) and in Sweden the majority of women with coronary heart disease were judged to receive sub-optimal treatment in the past (Hjart Lungfonden, 2009).

In a more recent study of referral of patients with stable angina Sekhri and colleagues (2008) found that women, along with older people, and south Asians were less likely to be referred for coronary angiography. Moreover deaths from coronary heart disease and admissions for unstable angina and myocardial infarction were more common in those in whom coronary angiography was deemed appropriate but not done. Buckley and colleagues (2009) in a cohort study of patients with an index episode of angina in primary care also found that women were less likely to receive surgical intervention. However in this study neither coronary bypass grafting nor percutaneous transluminal coronary angioplasty was associated with significantly improved survival.

Although the research conclusions with regard to IHD appear fairly consistent it should be noted that in the case of stroke no gender differential was found in the receipt of secondary prevention for stroke in primary care (Raine.et al., 2009).

Cancer accounts for about 30% of annual deaths. However assessing the impact of gender differentials in access to cancer care as a factor in influencing longevity is complicated by the fact that some common cancers are wholly or largely confined to one sex. Even so it seems likely that the current situation favours women. There are national programmes of prevention (cervical cancer), early detection (breast, cervical and, in the not too distant future, ovarian cancer screening programmes) and continuing developments in treatment. The current situation with regard to prostate cancer is not so advanced.

The final report of the Gender and Access to Health Services Study summarises the findings of the 2004 review by Macdonald and colleagues on the effect of gender on the delay between onset of symptoms and seeking help in primary care. For 10 different groups of cancers combined greater delay for men was reported in 13 studies, greater delay for women in 11 and no effect of gender in 23. In the same review the authors presented the data for delay in appropriate referral to secondary care. In 4 studies there was a greater delay if the patient was male, in 6 a greater delay for women and no gender difference in 4.

More recently Raine and colleagues (2010) have reported on social variations in access to hospital care for patients with breast, colorectal and lung cancer. For the last two of these they also looked at the effect of gender. Using emergency admission as a marker for sub-optimal referral they found that emergency admission was more common in women for both cancers (odds ratio following mutivariable analyses 1.15 and 1.12 respectively with 95%confidence intervals of 1.12-1.17 and 1.09-1.14). Interestingly women were slightly more likely to receive the optimal surgical treatment (anterior resection for colon cancer and surgical resection for lung cancer).

In 2010 the Expert Group on Gender Equality and Social Inclusion (EGGSI) of the EU published its final synthesis report "Access to Healthcare and Long-Term Care: Equal for Women and Men?" The report uses information from a network of national experts and statistical data from Eurostat and OECD sources but for some analyses data from the UK was not presented.

In all European Member States women live longer than men but women report higher levels of ill health than men at all ages. Interestingly for 2006 the figures from the UK are among the highest but the gender differential is the lowest among the 27 countries illustrated. Eurostat data for 2004 shows that the UK hospitalisation rate is the fourth lowest of the 19 countries illustrated but the rate for women is higher in every country bar Latvia, Estonia and Greece. UK figures for total consultations with a doctor are not included but in 20 other countries the rate is higher for women except in Austria and Malta.

The EGGSI report recognises that many countries (Austria, Bulgaria, Germany, Iceland, Ireland, Italy, Norway Slovenia, Spain, the Netherlands and the UK) acknowledge gender differences in healthcare and most have plans and/or national programmes to promote equality. Nevertheless the report states "It must be acknowledged that the gender-mainstreaming approach to healthcare is generally still underdeveloped and aside from reproductive care, little taken into account when offering service provisions."

In the UK the three most common causes of death are ischaemic heart disease (IHD), cancer and respiratory diseases. Such evidence as there is suggests sub-optimal access and treatment for women with IHD and some cancers compared to men. There is no data on respiratory diseases. However there is inconsistency of results from study to study and condition to condition some of which is likely to be due to methodological differences. There are many steps (e.g. symptom recognition, initial help seeking, specialist referral, appropriate treatment, secondary prevention) along the pathway to the resolution of serious illness, with each step involving appropriate decision making by patients and professionals. At every step there are potential differences in the way men and women react or are treated. Some of these are explored by Katz (2001), in the final report by Wilkins and colleagues (2008) and by McBride and colleagues (2010). It is clear that a better understanding of gender differences in this complex area will be required before much further progress can be made.

If the gender differentials could be eliminated then it is likely that there would be a further increase in female longevity as a consequence. Note however that there is a strong age-related differential in access to healthcare especially at 80+ years so this would have to be addressed too in order to achieve maximum gain.

3.3. Differences between the genders in mortality due to major categories of disease

Trovato and Heyen (2006) examined the gender differentials in e_0 within the G7 nations (Canada, France, Germany, Italy, England and Wales, USA, Japan) from early 1970 to late 1990. Confirming similar findings which indicate a reversal (since early 1970) of the long term trend in gender differences in life expectancy, the authors provided a decomposition of mortality by four major causes of death:

i) circulatory diseases,
ii) cancer;
iii) accidents, violence and suicide;
iv) other causes.

Within the six countries where gender differentials (e_0) have narrowed (with Japan being the exception), the authors have indicated that the reduction in the gender gap has been due largely to reduced gender differentials in mortality by: i) circulatory diseases; and ii) accidents, violence and suicide.

For example, the authors contend that diseases of the circulatory system have accounted for 22-50% of the gender gap in survival. Its net impact has tended however, to weaken over time, indicating a declining gender difference in the risk of death. Depending on the period and country examined, circulatory diseases have been responsible for approximately 1.6 to 3.4 years in the gender differential for e_0. But its effect has varied among the G7 nations and has tended to be larger in England and Wales, Canada, and the USA; and less so for France and Italy.

Also the authors argue that gender variability in mortality by accident, violence or suicide have contributed 10-25% of the observed gender gap in survival. Like circulatory diseases, however, the net impact has declined over time.

The authors have found that the contribution of cancer, although substantial, has tended to have a mixed effect on the gender gap in survival. Since the early 1990s, there has appeared to be a convergence of gender differences in cancer mortality rates in England and Wales, France, Canada and the USA. Cancer death rates for males, however, have continued to outpace those of females in Germany, Italy and Japan.

It can be noted that in Japan, although gender differences in mortality by circulatory diseases have been narrowing, its impact has been small. The combined effects of cancer, accidents, violence and suicide and 'other' causes have resulted in the widening gap (in favour of women) in Japan in gender differences in life expectancy at birth.

Progressing further, the authors calculated the second order differences[10] between the peak point in the gender gap and the most current year by; i) all cause mortality and ii) cause of death for each nation. They show considerable second order differences between the selected periods, indicating a narrowing of the gender differential in life expectancy in recent years. The figures quoted for the G7 countries include the following: i) USA (-2.3 years); ii) Canada (-1.7 years); iii) England and Wales (-1.6 years); iv) France (-0.8 years); v) Germany (-0.6 years); vi) Italy (-0.4 years); vii) Japan (0.5 years).

Their findings by cause of death support the contention that the reduction in gender differentials in mortality from circulatory diseases and accidents, violence and suicide have accounted for the bulk in the narrowing of the gender gap in LE from the 1980s to 1990s - with men having experienced greater gains in survival from these causes of death than women. In the next section, we will examine some of the major causes of death within the UK responsible for the decline in gender differences in life expectancy.

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[10] *Trovato and Heyen (2006) define second order differences as the difference in gender differentials (e_0) between 2 time points.*

3.4. Causes of death and gender differences (Trends over time)

Reflecting the decline in the gender gap in life expectancy, a recent study by Ashton et. al. (2010) highlights the continual fall in all-cause mortality rates for men and women aged 50 years and above. For men, age-standardised mortality had declined by 30% from 3,216 per 100,000 (1991) to 2,267 per 100,000 (2005). In comparison, age-standardised death rates for women had declined by 20% from 2,032 per 100,000 (1991) to 1,626 per 100,000 (2005).

The study listed the top 5 causes of mortality for men and women within England (1991 to 2005). Table 8 shows the relative decline (% change) in age-standardised death rates (per 100,000) for 3 conditions common to both men and women. For men, they indicate large relative declines (as compared to women) in age-standardised death rates for ischaemic heart disease, cerebrovascular disease and malignant neoplasm for the trachea, bronchus and lung.

Table 8. Top causes of mortality in males and females (England, 1991 to 2005)

Males (Ages 50 and Over)	Age-Standardised Rates (per 100,000)		Relative Change (%)	Absolute Change
	1991	**2005**		
Ischaemic Heart Disease	980.9	475.6	-51.5%	45,244
Cerebrovascular Disease	315.9	182.7	-42.2%	12,815
Malignant Neoplasm: Trachea, bronchus & lung	281.6	171.0	-39.3%	9,620

Females (Ages 50 and Over)	Age-Standardised Rates (per 100,000)		Relative Change (%)	Absolute Change
	1991	**2005**		
Ischaemic Heart Disease	479.4	228.6	-52.3%	33,601
Cerebrovascular Disease	275.3	172.4	-37.4%	15,349
Malignant Neoplasm: Trachea, bronchus & lung	98.4	95.4	-3.1%	557

Source: Adapted from Table 1. Top 5 causes of mortality in men and women, 1991 to 2005. Ashton C., Bajekal M., Raine R. Quantifying the contribution of leading causes of death to mortality decline among older people in England, 1991-2005. Office for National Statistics. Health Statistics Quarterly 45, Spring 2010, p.109. Crown Copyright 2009. This information is licensed under the terms of the Open Government License v1.0 (http://www.nationalarchives.gov.uk/doc/open-government-licence/open-government-licence.htm)

Using age-standardised death rates for the same conditions within the UK, we investigate further the male/female ratio of deaths rates for all ages and by specific age groups. For ischaemic heart disease (IHD), figures from the European Health for All Database (http://hfadb.who.dk/hfa/) show age-standardised death rates (all ages) for men have declined by roughly 70% from 388 per 100,000 (1970) to 116 per 100,000 (2009). Similarly, age-standardised death rates for women have fallen from 174 per 100,000 to 52 per 100,000 within the same period (an approximate 70% reduction) (Figure 13).

For cerebrovascular disease, figures show age-standardised death rates (all ages) for men have declined by roughly 72% from 161 per 100,00 (1970) to 44 per 100,000 (2009). Age-standardised death rates for women have fallen similarly from 143 per 100,000 to 42 per 100,000 within the same period (an approximate reduction of 71%). Figures show the contribution of older ages (age 65 and over) to the decline in gender differences in life expectancy for cerebrovascular disease (Figure 14).

In examining mortality rates for lung cancer, figures show age-standardised death rates (all ages) for men have declined by roughly 54% from 108 per 100,000 (1970) to 50 per 100,000 (2009). Age-standardised death rates for women, however, have risen from 18 to 31 per 100,000 within the same period (an increase of roughly 72%). The relative difference in mortality improvement for men is reflected in Figure 15 which shows the male/female ratio of death rates (all ages) for lung cancer falling significantly from a ratio of 6.05 (1970) to 1.6 (2009).

Figure 13. Male/Female ratio of death rates for ischaemic heart disease by age (United Kingdom, 1970-2009)

Figure 14. Male/Female ratio of death rates for cerebrovascular disease by age (United Kingdom, 1970-2009)

Figure 15. Male/Female ratio of death rates for lung cancer by age (United Kingdom, 1970-2009)

As both lung cancer and cardiovascular disease can be attributed to smoking consumption, a number of studies (e.g., Gjonca 2005, Pampel 2003) have highlighted the large gender differences in smoking patterns by examining the cohort born at the beginning of the 20th century for England and Wales. Large differences in smoking patterns within the cohort have been indicated, which has been reflected in the different rates of both lung cancer (with a death rate of 566 and 73 per 100,000 respectively for men and women in the UK aged 65 and above) and IHD (with a death rate of 2,363 and 1,309 per 100,000 respectively for men and women in the UK aged 65 and above) in the 1970s.

Since then, however, trends in smoking patterns have reversed. Using a Lexis or contour map to show cohort effects (as indicated by the male/female ratio of death rates for England and Wales), Gjonca et. al. (2005) indicated the gender difference (by age) in mortality from 1990 to 2002 (Figure 16).

Compared with females, male/female ratios of mortality rates greater than one would indicate a higher male death rate within the specified age groups and periods. In Figure 16, areas in grades of pink and red show a mortality disadvantage for males. The deepest (red) colour highlights the widest gap between male and female mortality rates for ages 18 to 25 between 1960 and 2002. With a male death rate 2.5 times greater than or equal to the female mortality rate, Gjonca et al. explain that: i) higher male than female mortality resulting from accidents and; ii) a decline in maternal mortality have contributed to the mortality disadvantage for males within the specified age groups and period.[11]

The authors highlighted figures showing differences in mortality were higher at older age groups among the cohort (where smoking was dominant among men) born at the beginning of the 20th century. For the cohorts born after 1920 (which showed a lower gender gap in mortality at older ages), however, the uptake in smoking among women had begun to increase. For example, recent figures (2008) show smoking rates for men and women within the UK as largely comparable. Allowing for a "diffusion" in smoking patterns (whereby there is period of increase in smoking behaviour, followed by a peak and eventual period of abatement), lung cancer mortality rates for women would then likely increase 10-20 years after their general adoption of smoking (resulting in a decline in the gender gap in lung cancer mortality).

Figure 16. Lexis map for the male/female ratios of deaths rates for England and Wales, 1900-2002
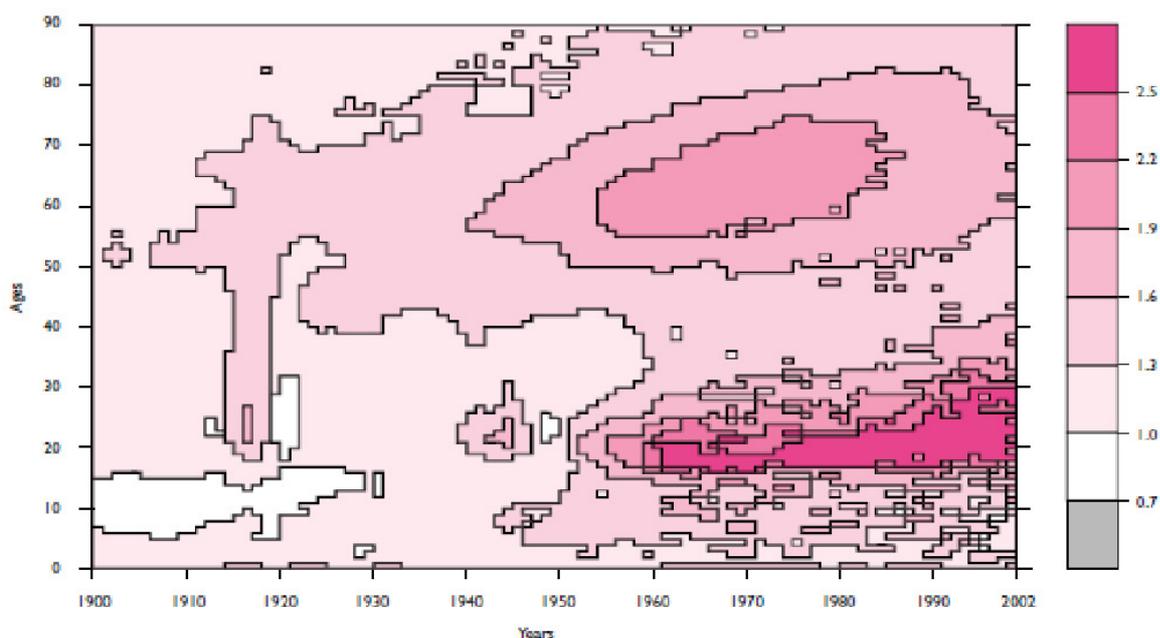
Source: Figure 2b. Lexis map for the sex ratios of death rates for England and Wales, 1900-2002. Gjonca A., Tomassini C., Toson B., and Smallwood S. Sex differences in mortality, a comparison of the United Kingdom and other developed countries. (2005). Office for National Statistics. Health Statistics Quarterly 26, Summer 2005,, p.12. © Crown Copyright 2005. This information is licensed under the terms of the Open Government License v1.0 (http://www.nationalarchives.gov.uk/doc/open-government-licence/open-government-licence.htm)

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[11] *Gjonca A., Tomassini C., Toson B., and Smallwodd S. Sex differences in mortality, a comparison of the United Kingdom and other developed countries. (2005). Health Statistics Quarterly 25, Summer 2005. Office for National Statistics, p.11.*

3.4.1. Analysis of 2010 data

The conclusions to be drawn from the work described in the previous section seem clear but we have sought to confirm them by analysing the 2010 data for England & Wales. Figure 17 illustrates the number of deaths by age and sex in that year. The excess of deaths among elderly women is simply a consequence of there being many more women still alive.

Most of the factors discussed in this paper can be expected to have complex and subtle relationships with disease aetiology and they will be very different for different diseases. Figures 18 (male) and 19 (female) illustrate the relative impact of the different categories of disease. They show the cumulative totals for each cause plotting the most common cause of death as the bottom line and successively adding the values for each of the other categories so that the highest line is a replica of the graph of total deaths. When classified into these broad categories the difference in the distribution between men and women is characterised by more male deaths from circulatory disease and cancer at all ages up to 80-84. Thereafter the number of female deaths is greater as expected from the gender specific age profile with the most marked increase in female deaths being from "All Remaining Causes".

Plotting the ratio of Male/Female deaths by age on a log scale is more illuminating, but ignores the age effect on mortality (Figure 20). It can be seen the ratio is almost always higher for men at all ages. The excess for cancer among middle aged women is due largely to hormonally associated breast cancer and for all other categories the differences diminish after the menopause – except for cancer where previous exposure, to tobacco for example, is likely to have a long delayed effect. The increasing excess among older men will be from lung and prostate cancer, both diseases of the elderly, thereby having only a modest effect on the gender differences in lifespan. By contrast the nearly five-fold excess for death from external causes among men occurs at a relatively young age. Fortunately the overall number of deaths involved is relatively small (Figures 18 and 19) but they do contribute importantly to years of life lost, accounting for a gender differential of over 1 year which is very similar to that contributed by cancer (Figure 22).

The contrast between the 2010 picture and that of 1979 (Figure 21) is interesting. The gender difference in external deaths is somewhat higher more recently and considerably so in the older ages. Differences attributable to respiratory illness are now much less marked among the elderly presumably reflecting the convergence of long-term smoking habits with time. The progression to unity after the menopause is similar except for cancer, an agglomerate of disease at many sites.

It is instructive to investigate the relative contribution to years of life lost by these diverse patterns of death. We have analysed this by multiplying the proportion of deaths by age and cause by the years lost before age 100 separately by sex (Figures 22 and 23).

Figure 22 shows that circulatory disease makes the largest contribution, at almost 3 years (out of a total of 5-6 years), to the gender difference in years of life lost. Injury has an effect of a little over one year, as does cancer though it is important to further analyse cancer by specific site and the results of this are shown in Figure 23.

Breast cancer is a common disease of middle-aged women and thereby has a dominant effect on the gender differences in life expectancy, but one which is opposite to the general trend of excess death in men. Consequently, while breast cancer mortality is as high as it is, the true comparable gender difference in life expectancy is greater than that which we currently observe. That aside the dominant cause of the gender difference is the conglomerate of digestive organ cancers including alcohol and dietary related ones. Respiratory cancer differences will be determined largely by life-course smoking patterns of the two sexes.

The difference in exposure to alcohol, tobacco and unhealthy food by men through their lives will explain some of these differences but that requires further study. The residual will be largely due to hormonal differences with complex effects of testosterone and oestrogen on some cancers, on the immune system and on the cardiovascular system.

Whatever the complex array of explanations our own analysis of ONS data confirms the trend in the gender differential in life expectancy over the past 60 years (Figure 24). The steep rise between the end of WWII and the mid-1960s and the steep fall from the late 1970s to the present are evident.

Figure 17. Total deaths recorded in England and Wales (2010)
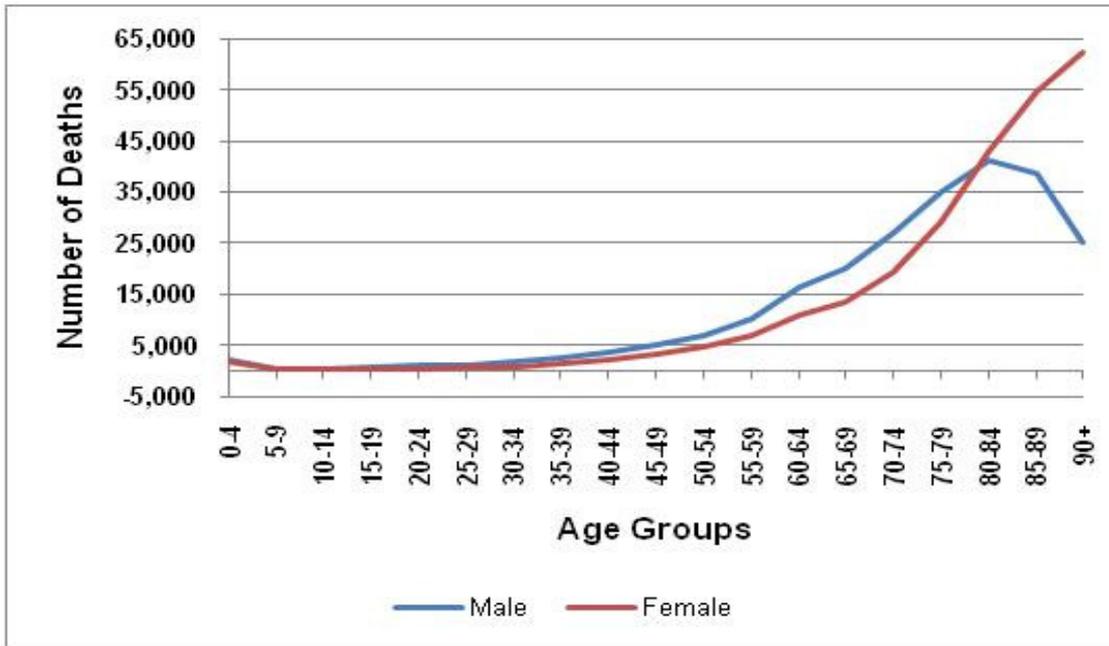
Source: Table 5. Mortality Statistics: Deaths registered in England and Wales (Series DR, Table 5 2010). Office for National Statistics. http://www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm%3A77-230730. © Crown Copyright 2011. This information is licensed under the terms of the Open Government License v1.0 (http://www.nationalarchives.gov.uk/doc/open-government-licence/open-government-licence.htm)

Figure 18. The cumulative contribution of major disease categories to the number of total deaths (Male, England & Wales 2010)
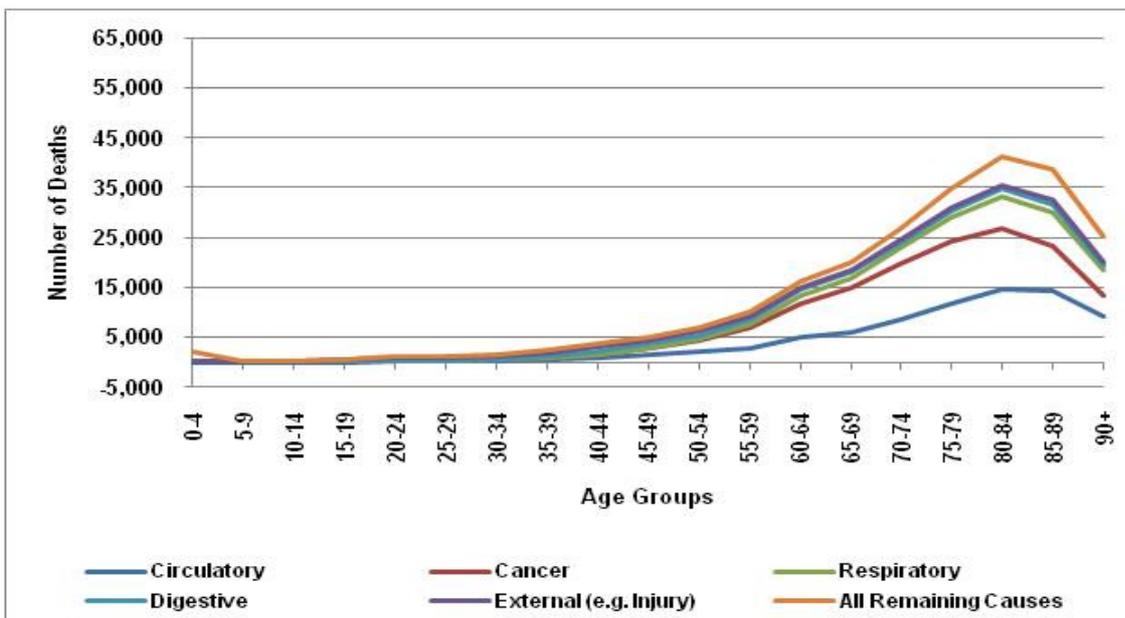
Source: table5causeof death_tcm77-23986.xls. Mortality statistics: Deaths registered in 2010 (Series DR) Table 5. Office for National Statistics. http://www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm%3A77-230730. © Crown Copyright 2011. This information is licensed under the terms of the Open Government License v1.0 (http://www.nationalarchives.gov.uk/doc/open-government-licence/open-government-licence.htm)

Figure 19. The cumulative contribution of major disease categories to the number of total deaths (Female, England & Wales 2010)

Figure 20. Male/Female ratio of death rates from selected causes (England & Wales 2010)

Figure 21. Male/Female ratio of death rates from selected causes (England & Wales 1979)
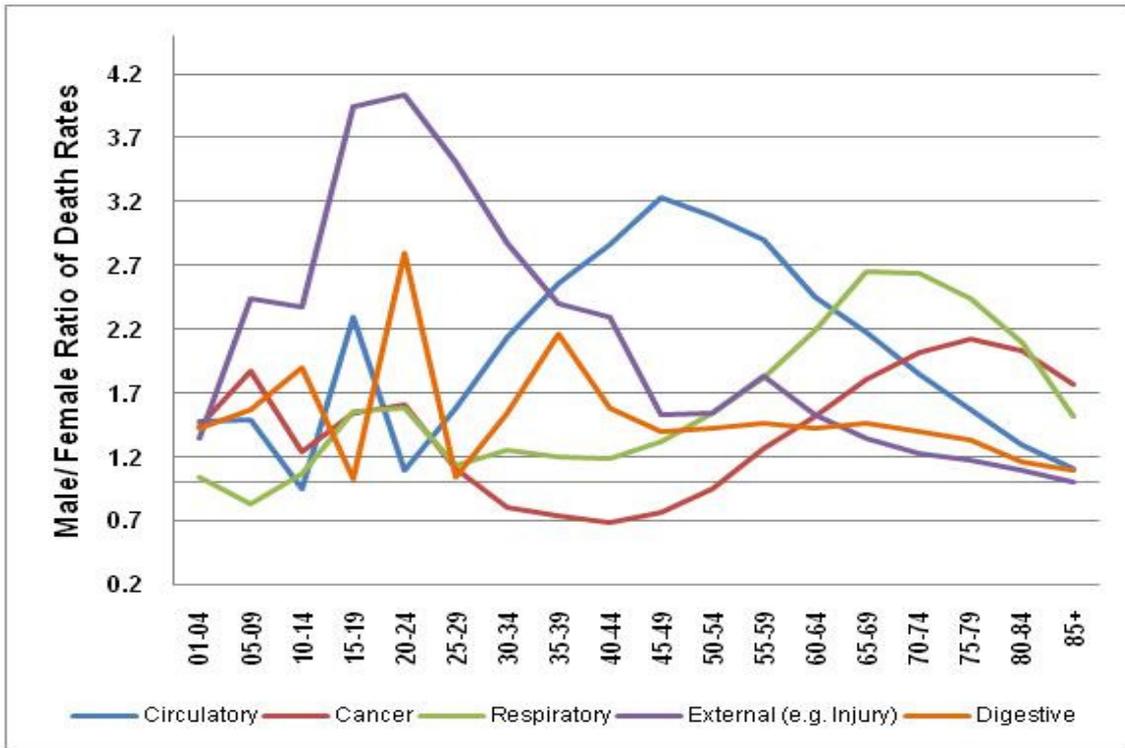
Figure 22. Average years of life lost before age 100, by cause separately by gender

Figure 23. Average years of life lost before age 100, by cancer sub-type separately by gender

Figure 24. Difference in life expectancy at birth between males and females (England & Wales, 1949-2009): Reference to 1979 and 2009 (proxy for 2010)
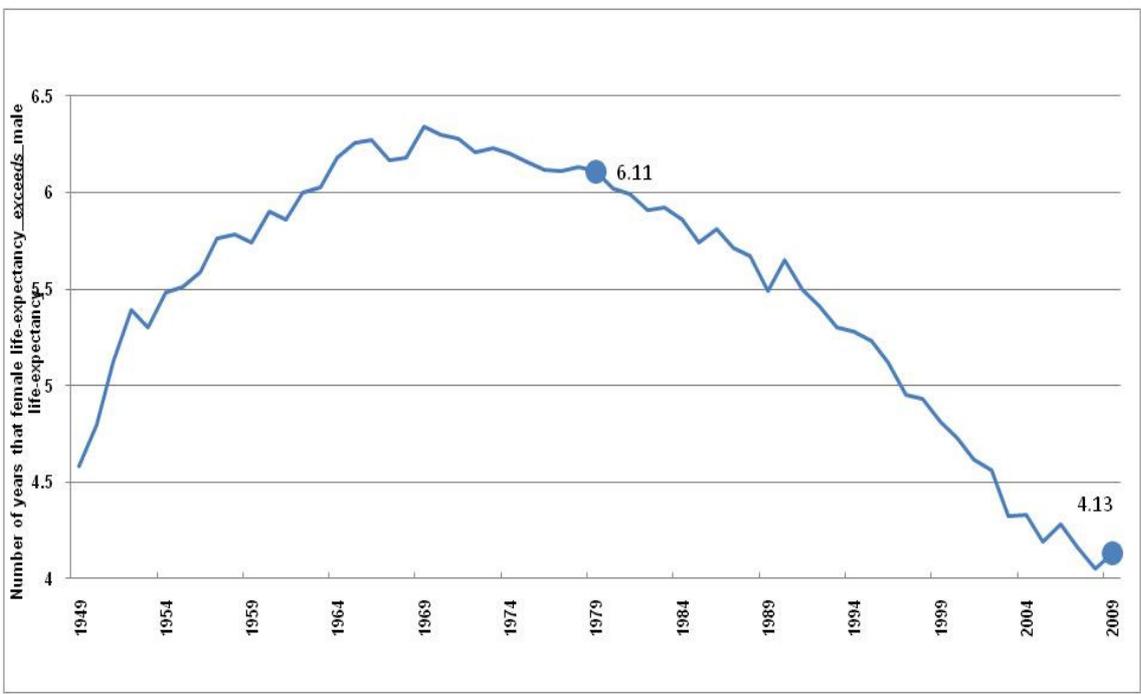
3.5. Gender differences in life expectancy by National-Statistics Socio-economic Classification (NS-SEC)

Since 2001, the ONS has regarded the NS-SEC as the official (occupation based) measure of socio-economic class (refer Appendix C, Table B.1 for a breakdown of the classification). First estimates of life expectancy at birth and at age 65 (by NS-SEC) were published in February 2011 by the ONS in *Statistical Bulletin: Trends in life expectancy by the National Statistics Socio-economic Classification 1982-2006*. Some of its findings are provided in Appendix C (Tables C.2-C.6) in this document.

Figures 25 and 26 show the gender gap in life expectancy at birth and at age 65 respectively by (Condensed) NS-SEC. Although gender gaps in life expectancy (e0 and e65) have continued to decline since 1982, differences in the magnitude of the gaps continue to exist, highlighting the inequalities in life expectancy by socio-economic class.

However, trends in the decline of the gender gap by NS-SEC have not been consistent. For example, when examining life expectancy at birth (Figure 25) by NS-SEC over the full study period, we find that those classified within the "Managerial and professional" class have experienced the greatest fall in gender differences; declining from 5.3 years in the 1982-86 period to 3.5 years in 2002-06 (a reduction of 34%). In comparison, persons classified within the "Routine and manual" class have shown the smallest decline, falling by 24% from 6.3 years (1982-86) to 4.8 years (2002-06). More recent data (1997-2001 to 2002-06) show the largest decline in those classified within the "Routine and manual" class; with a fall in the gender gap from 5.4 to 4.8 years (a reduction of 11%). In comparison, the fall in the gender gap for those classified within the "Managerial and professional" class represented a slightly smaller decline of about 10% from 3.9 to 3.5 years. The smallest decline of 4.7 to 4.6 years (2%) was experienced by those classified within the "Intermediate" class.

Socio-economic differences in the decline in gender gap in life expectancy at age 65 (Figure 26) have been less variable. Persons classified in either the "Managerial and professional" or "Routine and manual" categories have experienced similar declines of approximately 28% over the study period. Those classified within the "Intermediate" category have displayed the largest fall in the gender gap; declining by 33% from 4.5 years to 3 years over the period. By 2002-06, the gender gap in life expectancy at age 65 for all 3 classes were broadly similar, ranging from 2.9 (Managerial and Routine) to 3.1 (Routine and manual) years. For those categorised within the "Managerial and professional" class, the most recent investigation (1997-2001 to 2002-06) show no change in the gender gap in life expectancy.

The convergence in the gender gap in life expectancy of the 3 groups at age 65 in 2002 to 2006 is striking particularly when compared with the differences in life expectancy at birth. By age 65 the factors which contribute most to gender differences in life expectancy, notably external causes in young men, death in middle aged men from heart disease and breast cancer in women (Figure 20) will have worked through and may help explain the convergence seen in Figure 26.

Figure 25. Gender gap in life expectancy at birth by NS-SEC (England & Wales)
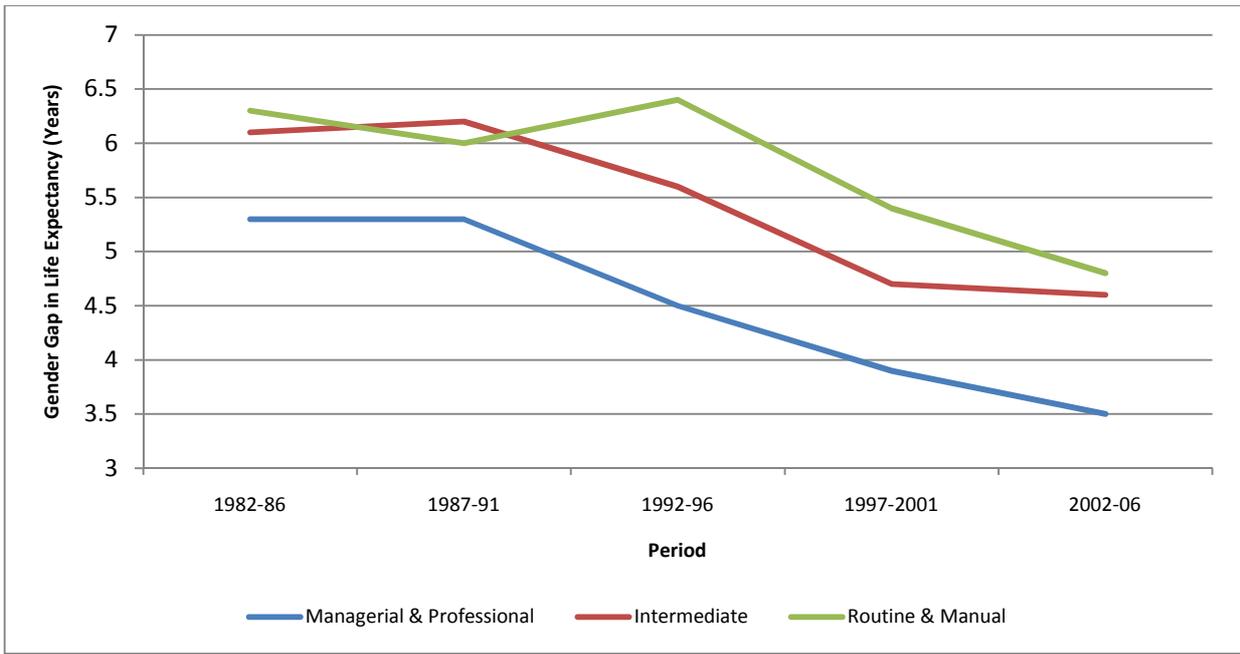
Figure 26. Gender gap in life expectancy at age 65 by NS-SEC (England & Wales)
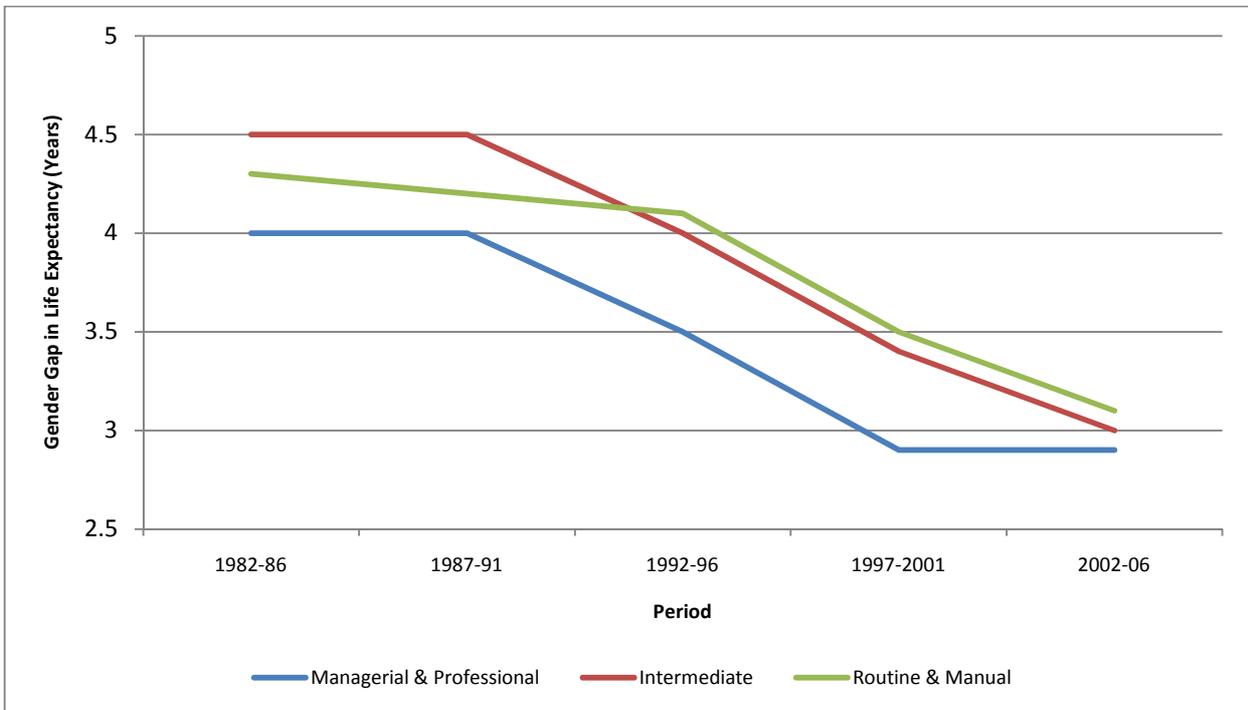
3.6. Differences in the physiology or genetic makeup between genders

3.6.1. Gender differences in mortality

Survival and longevity are distinct entities. Survival to, and through, adulthood in any species depends on avoiding death during birth and later – from infections, fighting, predation, accidents and other catastrophic events. Longevity, on the other hand, implies approaching the maximum potential lifespan of a species, which is presumed to be determined by progressive degenerative changes associated with ageing. Both of these factors – extrinsic and intrinsic – contribute to average life expectancy. But for human beings in the developed world, with good sanitation, access to vaccination and antibiotics, and low risk of violent death, the intrinsic effects of ageing contribute much more to overall life expectancy (May 2007) . Studies of other species in the wild tell us more about survival than ageing, because weakened individuals are quickly removed from wild populations through predation and disease (Smith 1989). On the other hand, observations in outbred laboratory animals (Partridge et al. 2011) and data from zoos and domestic animals (e.g. Hamilton 1965) are more informative about inherent factors that contribute to ageing and life expectancy.

3.6.2. The conservation of gender differences in ageing

Sex differences in survival and lifespan are observed across a huge range of species, from mammals (Clutton-Brock and Isvaran 2007), to birds (Barrett and Richardson 2011; Liker and Szekely 2005) to invertebrates (e.g. Tu et al. 2002). With the notable exception of many bird species, females are usually the longer-lived sex. Most mammals show a higher male death rate at all ages, including differences in maximum lifespan, with few males in the oldest age groups. This is even the case before birth: the ratio of male to female conceptions is 120:100, but the gender ratio falls to 105: 100 by term. This pattern continues in childhood, where the death rate from all causes is 41% greater in boys than in girls in England and Wales (Kraemer 2000). Throughout life, men have a higher death rate from most principal diseases (Smith and Warner 1989); however, the gender gap is most apparent in longevity: life expectancy for women is significantly higher than for men. 75% of those over 100 are women (Blagosklonny 2010).

One of the features co-incidental with sex differences in lifespan is sex-specific behaviour. In animals behavior such as male-male aggression (Clutton-Brock and Isvaran 2007), territorial ranging and childcare (Liker and Szekely 2005) can certainly contribute to male mortality. Procedures that abolish male-specific behaviour, like castration, can have a profound affect on annual survival rates in mammals (see *hormones*). Risky behavior in human males affects survival, where men are more likely to die from accidents or violence at all ages. For example, the suicide rate in young men is several times higher than young women: in 15-24 year-olds in Ireland, the ratio of male to female suicides is over 7:1. In addition, addiction, particularly substance abuse, is more common in men. To compound effects further, men often do not notice genuine symptoms of illness and are less likely to seek help from doctors if they do. However, studies indicate that behavioural differences are insufficient to explain higher male mortality in many species, and the gender gap remains in humans when death rates are normalized for risky behaviour (May 2007; Moore and Wilson 2002).

What then of the relationship between behaviour and longevity in man? Behavioural differences, even early in life, can contribute to long-term survival, as well as to the chances of dying prematurely. Smoking, alcohol consumption, exercise and diet can obviously have long-term consequences on health and survival. Historically, women have tended to adopt healthier diets than men (Wardle et al. 2004), while men have smoked and drunk more. The recent trend towards narrowing of the gender gap, especially in Britain, has largely been attributed to convergence in behaviours such as smoking, drinking and lifestyle choices, such as involvement in childcare (Mansdotter 2010; Bobak 2003). The crucial question is whether there are residual sex differences in the pattern of ageing, underpinned by basic biological differences. Obvious differences in karyotype, steroid hormones and their consequences need to be explored.

3.6.3. Karyotype

The karyotype of an organism describes the number and appearance of its chromosomes. Male/female biological differences are ultimately underscored by the genetic process of sex determination. In mammals, females are 'homogametic'; they have two copies of the sex chromosome X, while males have one X and one Y chromosome. The X chromosome contains thousands of genes, most of which are not female-specific. In contrast, the Y chromosome is small and largely heterochromatic, containing only a few genes important for male development and fertility. In order to achieve the same dosage of X linked genes in males and females, one X chromosome is inactivated early in development in female embryos. This occurs at random in each cell; therefore, females are mosaics of functionally hemizygous cells, each containing only one functional X chromosome.

An obvious benefit in having two X chromosomes (as for all other matched pairs of chromosomes) is that a mutated gene on the X chromosome inherited from one parent will not affect cells in which the other X chromosome is active. Hence homogametic individuals would be expected to have a survival advantage over heterogametic. Examples of this phenomenon are X-linked diseases such as haemophilia and Duchenne muscular dystrophy, which are devastating in males but difficult to even detect in the female heterozygote carrier. However, as X-linked diseases are relatively rare, this may not significantly affect average lifespan, unless their occurrence leads to protective changes in the genome of the species that result in subtle defects that have an impact on late life health. Support for this theory is so-called 'hemizygous selection' (Abkowitz et al. 1998), where ratios of cell mosaicism in older women become skewed to favour one active X chromosome, presumably the one that contains variant genes that give a selective advantage (Christensen 2000).

In birds, females are heterogametic (having an unmatched pair of sex chromosomes) and males (which are homogametic) tend to have lower mortality (Barrett and Richardson 2011; Liker and Szekely 2005). However, equal lifespan and even higher male mortality are also observed , and diverse mating systems, testosterone and varied levels of aggressive competition confound direct comparison of karyotype and longevity (Clutton-Brock and Isvaran 2007; Liker and Szekely 2005).

3.6.4. Telomeres and telomerase

Vertebrate telomeres consist of non-coding DNA repeats that protect the ends of chromosomes from fusion, degradation and recognition by the DNA damage response (Blackburn 2000). Telomeres are degraded by agents such as reactive oxygen species (ROS) and, during cell division, by incomplete DNA replication. However, telomere length can be maintained by the enzyme telomerase. Critically short telomeres induce replicative senescence and cell death (Blasco 2007). Although both short telomeres and increased degradation rate have been linked to reduced lifespan, whether telomere dynamics are a causative factor for life expectancy is still under debate. However, it is clear that there are pronounced sex differences in telomeres in humans, with men having shorter telomeres and a higher rate of degradation than women (Stindl 2004). The relationship between telomere length and lifespan is not consistent across species although higher male mortality is usually linked to shorter telomeres, no studies have yet identified shorter female telomeres, even when female mortality is higher. Nevertheless, information is emerging on the possible roles for sex in telomere biology, including; the effect of unpaired sex chromosome, differential oxidation by mitochondrial and immune-generated ROS and oestrogen regulation of telomere maintenance.

3.6.5. Hormones and ageing

3.6.5.1. Testosterone

Testosterone has a profound affect on male lifespan; for example, castration of male Soay sheep shortly after birth stops them taking an active part in the rut and leads to spectacular increases in adult survival relative both to intact males and to females (Jewell 1997). Data on human castration are limited, but in a classic 30-year study of eunuchs from a mentally retarded population, castrated men were found to have a median lifespan 13.5 years older than intact males from a comparable group. In line with the well-known immunosuppressive function of testosterone, intact males are at a greater risk of death by infection. (Hamilton and Mestler 1969). Among domesticated cats (which in general have not been selectively bred, are well cared for and routinely castrated), castrated males lived as long as intact females, and also had significantly lower death by infection than intact males. Most studies, such as those described, have reported a negative effect of testosterone on survival, correlated with its effects on aggressive (and therefore risky) behaviour and/or on immunity; however, it is important to note that low testosterone levels in ageing men are associated with decreased muscle mass and bone density and increased central body fat, contributing to a range of pathologies (Horstman et al. 2012).

3.6.5.2. Oestrogen

The positive effects of oestrogen are wide-ranging and include influences on muscle strength and skeletal muscle repair (Horstman et al. 2012), and on glucose and lipid metabolism (lowering the risk of metabolic syndrome and type 2 diabetes (Faulds et al. 2012) as well as lowered risk of cardiovascular disease (CVD), particularly atherosclerosis (Horstman et al. 2012; Novella et al. 2012). The protective effects on vascular physiology underlying lower incidence of CVD is via Oestrogen Receptor-mediated effects on nitric oxide bioavailability, endothelial constrictive factors, calcium signalling, collagen, elastin and ROS (Novella et al. 2012). The beneficial effects of oestrogen are illustrated by the rapid physiological decline following menopause, (Horstman et al. 2012; Novella et al. 2012) including a risk for CVD that is equal to men of the same age (Novella et al. 2012). However, menopause does not equalise mortality, which is still consistently lower in post-menopausal women than in men of the same age.

Although invertebrates do not have sex steroids, they do have steroid hormones such as Ecdysone in insects and Daf-12 in worms which are known regulators of life history traits including ageing (Galikova 2011). Differential steroid hormone regulation between the sexes has been described and this might play a part in sex differences in lifespan observed in these animals (Schwedes and Carney 2012).

3.6.5.3. Insulin signalling

Recent studies show that particular individual genes ('gerontogenes') in conserved signalling pathways can have a profound affect on lifespan in laboratory animals such as worms, flies and mice (Clancy et al. 2001; Tatar et al. 2001; Kenyon et al. 1993). These gerontogenes are largely linked to sensing nutritional state via insulin signalling and its control of the transcription factor FOXO, which modulates the expression of hundreds of genes including those involved in immunity and oxidative stress (Partridge et al. 2011). Experimental mutation of gerontogenes has different outcomes for the sexes, suggesting that the baseline state of these pathways is different in males and females. Indeed, data from cohort studies suggests an interaction between gender and insulin signalling in human longevity (van Heemst et al. 2005).

3.6.6. Infection and immunity

Evolutionary arguments suggest that female mammals gain in reproductive success by investing more in longevity than males (Clutton-Brock et al. 1985); hence female investment in immune mechanisms ought to be higher (Folstad and Karter 1992). Indeed, in a wide range of species, males have a higher disease burden (Moore and Wilson 2002), potentially contributing to higher mortality. It is suggested that this is a consequence of testosterone 'handicapping' males by immunosuppression, providing a way for females to select fitter males (Hamilton and Zuk 1982). Other theories do not rest on suppression by testosterone but suggest that immunity is fundamentally different between males and females (Rolff 2002; Zuk 2009). The costs of evolving (Kraaijeveld and Godfray 1997; Webster and Woolhouse 1999) and activating (Moret and Schmid-Hempel 2000) immunity and the related trade-offs for competitiveness and fertility have been demonstrated, supporting the idea that high immune investment will have a negative impact on reproductive success for males. It is known that in humans, women have a more active immune system than males, underlying a lower incidence of infection and a higher rate of autoimmunity (Gilliver 2010).

Studies on mammals indicate that ageing is associated with a decline in adaptive immune function, but an increase in innate immunity leading to constitutive low-grade inflammation (Goto 2008). Inflammation is a strong risk factor for age-related pathologies in humans including cancer, CVD, type-2 diabetes, and Alzheimer's disease (Couzin-Frankel 2010). The role of sex steroids as inflammatory regulators (Gilliver 2010) results in gender differences in some inflammation-related pathologies, whose risk factors change with ageing as sex steroid levels decline (Horstman et al. 2012; Gilliver 2010).

3.6.7. The mitochondrial free radical theory of ageing

An influential theory proposed to explain senescence is the 'free radical theory' (Harman 1956). Briefly, this states that oxygen-derived free radicals are responsible for age-related cellular damage. A large body of empirical evidence supports this theory, for example, there is an inverse correlation between the rate of ROS production and the maximum lifespan across mammalian species (Barja and Herrero 2000). In addition, over-expression of antioxidants in mitochondria in mice (Schriner et al. 2005), or in whole flies (Orr and Sohal 1994), extends lifespan significantly. Mitochondria are a major source of free radicals within cells, forming the superoxide anion O_2^- during respiration, which is converted to hydrogen peroxide (H_2O_2). Mitochondria are themselves damaged as a function of age, involving oxidative damage to mitochondrial DNA, proteins and lipids (Vina et al. 2005).

3.6.7.1. Gender differences in oxidative damage and ageing

Mitochondria in female mammals produce significantly lower levels of oxidants such as H_2O_2 than mitochondria in males (Borras et al. 2003), and higher levels of antioxidants such as glutathione (Sastre et al. 2000). It is well-established that oestrogen modulates ROS concentration via its nuclear receptor to increase antioxidant enzymes and decrease oxidative proteins (Vina et al. 2005). Conversely, testosterone has no antioxidant properties and is linked to increased susceptibility to oxidative stress (Alonso-Alvarez et al. 2007). This differential effect on oxidative damage by sex steroids has been put forward as an underlying molecular mechanism for gender differences in human lifespan (Vina et al. 2005).

3.6.7.2. Alternative functions for ROS in ageing

Recent studies have shed doubt on the validity of the free radical theory of ageing, suggesting that the relationship between oxidative stress and lifespan may be correlative, not causative. For example, comparison of exceptionally long-lived with short-lived bird species indicated that longevity is independent of mitochondrial ROS (Montgomery, Hulbert and Buttemer 2012), antioxidant mechanisms, and accumulation of oxidative damage (Montgomery, Buttemer and Hulbert 2012). In addition, in organisms in which mitochondrial function and levels of free radicals or antioxidants can be experimentally manipulated, such as flies and worms, oxidative damage and lifespan can be uncoupled (Van Raamsdonk et al., 2012). However, given that ROS have other important functions, namely inter-cellular signalling (Ray et al. 2012), immunity (Ha et al., 2005) and damage to telomeres (Blackburn 2000) it is certainly possible that ROS can directly influence the ageing process. These functions are likely to show sex differences, given the major role played by sex hormones in modulating cellular ROS (Vina et al. 2005).

Section 4. Summary and conclusions

Women live longer than men, a gender differential found in most mammalian species and documented in the UK since 1841. In this paper we explore this phenomenon by reviewing data and analyses from a number of sources.

Ignoring the effects of the two World Wars, the difference in male and female life expectancy at birth reached a peak of 6.25 years in the period 1965-1969 and the difference at age 65 peaked at 4.01 years in 1980-84. Since then the gaps have narrowed to 4.15 years and 2.69 years respectively in the period 2005-09. Analysis of HMD data by the CMI reveals a contrast between the marked improvement in the 25 years to 2004 compared to previously but again a differential favouring males is apparent. The rate of mortality improvement for men aged 40-89 over the last 25 year period was 2.1% per annum compared to 0.5% for the previous 125 years. The corresponding figures for women were 1.7% and 0.8%. Similar trends in both mortality improvement and a narrowing of the gender gap have been observed in many industrialised countries, though with respect to the gender gap there are notable exceptions (Japan and Russia).

The interesting question is, will the gap continue to close and ultimately disappear? Arriving at an answer will depend upon a clear understanding of the determinants of the difference. Both nature and nurture play a part with the powerful effect of social factors being underpinned by basic biological differences. Traditionally (and in practice) men are the greater risk takers but in some important respects this is changing. The mortality due to external causes (injury/accidents/self-harm/violence) is significantly greater in young men and it is probable that this gender difference will persist. But the absolute number of deaths from these causes is small and even if changes were to occur the impact on overall longevity would be small.

However other risky behaviours (smoking, alcohol consumption, poor diet) are tending to equalise between the genders and these have a profound impact on longevity acting via the major causes of mortality, cancer and cardiovascular disease. There is general agreement that, to date, the rise and fall of tobacco consumption has been a major determinant of the gender gap in longevity. McCartney et al (2011) calculate that smoking related deaths account for 40-60% of the gap in many countries. For the US Wang and Preston (2009) estimate that the gain from smoking cessation in the next 20 years in men will be three times that in women. In the UK the 2008 data show no statistical difference in male and female smoking rates. We derived the male/female ratio of death rates for smoking related causes in the UK and found they have fallen from 2.1 to 1.7 between 1979 and 2009. As a consequence we are likely to see a further narrowing of the gender gap in longevity as the effects of smoking cessation take sometime to work through. Interestingly the ratio for alcohol related deaths rose from 2 to 2.4 over the same period.

The incidence of diabetes has increased dramatically in many countries during the last 20 years but it is not clear that the consequences for the two genders will be the same. There are small differences in the current prevalence rates between men and women in the UK but more dramatic gender differences in the extent to which obesity increases the risk of a number of potentially serious conditions.

In 2011 ONS published data on life expectancy for three socio-economic (SEC) groups, managerial and professional, intermediate and routine and manual. Importantly the gender gaps at birth and at age 65 have declined between 1982-86 and 2002-2006 for all three SECs and the figures at age 65 for the three groups are converging rapidly at the end of the period of analysis. We concluded our first paper on variations in life expectancy between SECs by saying that the inequality between SECs will not narrow. It is pleasing to note therefore that the inequalities in the gender gaps by SEC are narrowing, at least at age 65.

Cardiovascular disease and cancer each account for approximately 30% of deaths in the UK and these diseases manifest the biggest gender differentials in years of life lost before 100. In the case of cardiovascular disease a significant impact on the gender gap in longevity would be seen if there was a further improvement in the prevention or management of coronary heart disease. This is because the age profile of deaths from CHD in men is skewed towards a younger age group than in women. It is quite likely that there will be further reductions in male mortality from this cause as the full effect of smoking

cessation becomes evident. However the data show that the male/female ratio of death rates for ischaemic heart disease (IHD) has changed little from 1970 to 2009 and remains stubbornly high for the age range 0-64 years (Figure 13). Research to date suggests that the management of women with CHD is sub-optimal. If this was corrected an increase in female longevity might result, thus helping to maintain or widen the gender difference though the effect would be relatively small. For cerebrovascular disease the difference is not as dramatic as it is in IHD and indeed approaches parity for the 65+ age group.

Cancer contributes the second biggest difference between the genders of lives lost before age 100 (Figure 21) and consideration of organ specific cancers yields further insights (Figure 22). Breast cancer makes a major contribution to female mortality at a relatively young age and any significant improvement in survival will tend to increase the gender gap in longevity. The male/female death ratio for lung cancer has been falling since 1970 (Figure 15) but appears to be reaching a plateau of about 1.5 in 2006. Nevertheless the effects of equalising the smoking habits of men and women are not yet fully apparent and there may be a further reduction in the approximately half year difference between the genders in life lost by age 100 from this cause. Apart from breast cancer the dominant cause of the gender difference in years of life lost due to cancer is digestive organ cancer including those related to alcohol and diet. It is not possible to predict how this might change as a result of the national screening programme for colorectal cancer and any advances in treatment. Prostate cancer is common in elderly men so the impact of any improvements in detection and treatment on the gender gap in longevity is likely to be small.

There is progress in understanding the basic biology of ageing. Evidence supports a possible role for a number of individual factors in affecting lifespan and for most of these there are gender differences. Women have two X chromosomes and men only one. This provides a survival advantage and there is some evidence for an advantage for health in late life. The relationship between telomere length and lifespan is still undecided but the telomeres of women are longer and degrade at a lower rate than those of men. Genes known as 'gerontogenes' have been shown to have a marked effect on lifespan in laboratory animals acting via nutrient sensing pathways and there is some evidence of an interaction between gender and insulin signalling pathways in human longevity. Other evidence supports relatively deleterious effects of testosterone on male lifespan (e.g. via immunosuppression) and positive effects of oestrogen in women (e.g. via a protective effect on vascular physiology). There remains doubt about a causative role for reactive oxygen species in ageing. If there is one then it is notable that females are at an advantage compared to males because of their mitochondrial physiology and the modulating effects of oestrogen.

Ageing is a multi-factorial process and theories attempting to explain gender differences in ageing based on single causes tend to be oversimplified and controversial. The gender gap in human lifespan is profoundly affected by societal and behavioural factors and movement towards greater parity in lifestyle between men and women is a major factor in the recent reduction in gender gap in life expectancy. Nevertheless there is such a significant range of genetic, endocrine, cell and molecular biology differences between men and women with impacts on longevity that we are led to the conclusion that a gender difference in longevity will persist. At age 65 this is probably of the order of 1-2 years.

Finally we believe that raw data exists which could be analysed to eliminate social and environmental factors and provide a more accurate estimate of the underlying gender gap in longevity. We plan to explore this possibility in the near future.

Appendix A

Male and female cohort life expectancy at birth and age 65 (International comparisons)

A.1. Male cohort life expectancy at birth (1751-1918)
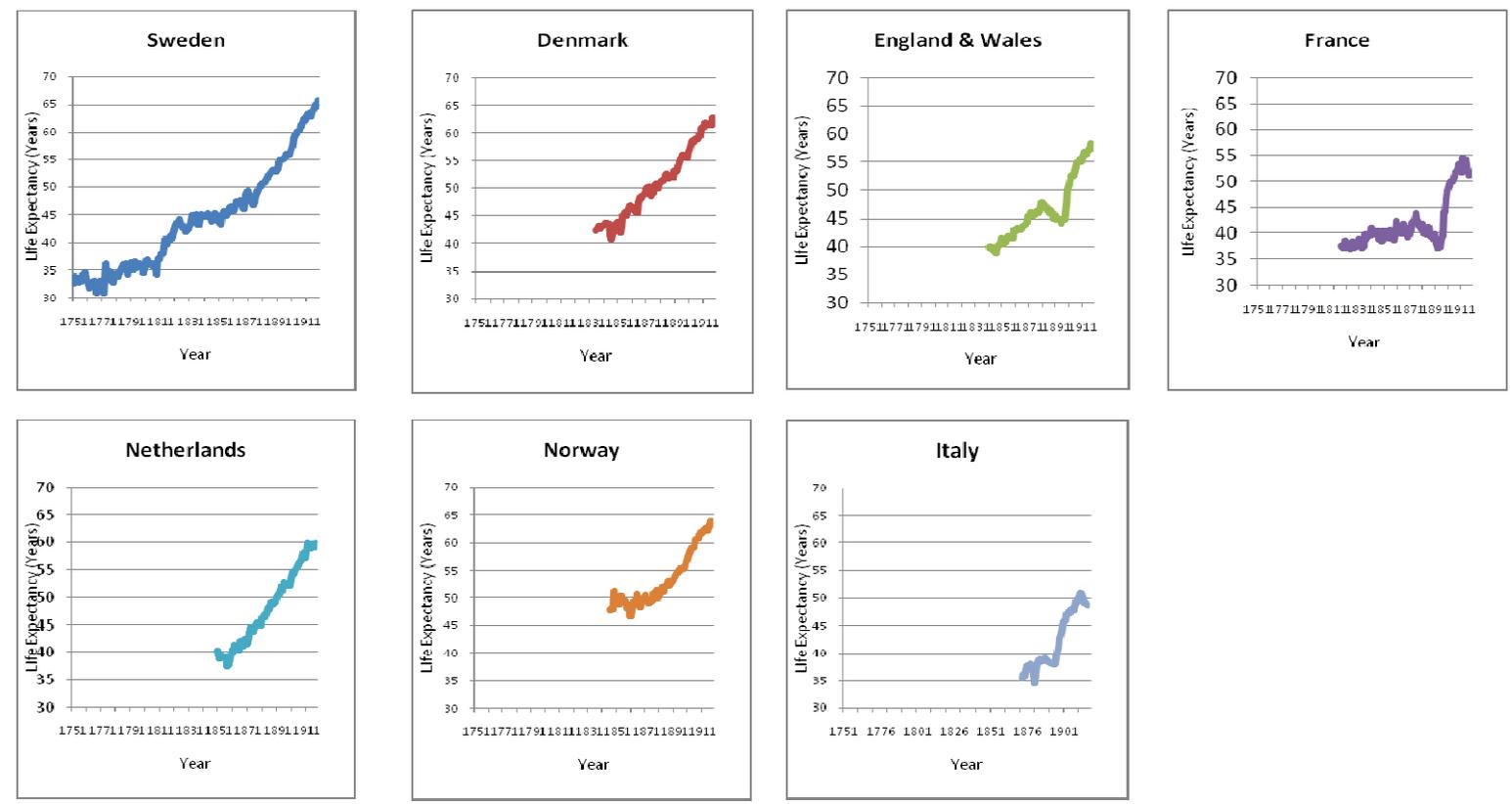
Source: Sweden, Denmark, E&W, France, Netherlands, Norway. Total Population, Life tables (cohort 1x1), Males and Females. © Human Mortality Database. University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany). (www.mortality.org)

A.2. Female cohort life expectancy at birth (1751-1918)

A.3. Male cohort life expectancy at age 65 (1751-1918)

A.4. Female cohort life expectancy at age 65 (1751-1918)

Appendix B

Description of age-standardised deaths rates provided by Data for European Health for All Database (HFA-DB)

Classification of smoking or alcohol related deaths includes mortality from combined, selected causes of death known from literature to be related to smoking or alcohol consumption. Figures are rough indicators (simple pooling of related deaths) and are not an estimate of smoking or alcohol attributable mortality.

B.1. Selected alcohol related causes, per 100000:

Some known alcohol related causes are not included, as they are not available separately in the mortality data files reported to WHO (mainly when causes were coded using ICD-9 Basic Tabulation List or the list of 175 causes used in countries of former USSR. Includes: Cancer of oesophagus and larynx (Cancer of liver is not available in 175 list); Alcohol dependence syndrom (alcoholic psychoses not available in BTL); Chronic liver disease and cirrhosis; All external causes.

B.1.1. Coding:

ICD9: 150, 161, 303, 571, E800-E999.
BTL: 090, 100, 215, 347, E47-E56
List 175: 46, 52, 75, 122,123, 160-175
ICD-10: C15, C32, F10, K70, K73, K74, K76, V00-V99, W00-W99, X00-X99, Y00-Y99.

B.2. Selected smoking related causes, per 100000:

Figures represent a simple pooling of smoking related deaths (regardless of actual proportion of deaths due to tobacco within each cause of death). Includes: Cancers of mouth and pharynx, larynx, traxea, bronchus, lung and oesophagus;Ischaemic heart disease; Cerebrovascular diseases; Chronic obstructive pulmonary disease.

B.2.1. Coding:

ICD-9: 140-149, 161, 162, 150, 410-414, 430-438,490-496.
BTL: 08, 100, 101, 090, 27, 29, 323-325.
List 175: 45, 52, 53, 46, 90-95, 98- 99 (or 196-205), 108-110.
ICD-10: C00-C14, C32-C34, C15, I20-I25, I60-I69, J40-J47.

Appendix C

National Statistics–Socio-Economic Classification (NS-SEC)

C.1. Description of National Statistics – Socio-Economic Classification (NS-SEC) Categories

Eight Classes	Three Classes

1. Higher managerial, administrative and professional occupations
 	
   1.1 Large employers and higher managerial and administrative occupations
 	
   1.2 Higher professional occupations
 	
2. Lower managerial, administrative and professional occupations
	1. Higher managerial, administrative and professional occupations

3. Intermediate occupations 4. Small employers & own account workers	2. Intermediate occupations
5. Lower supervisory & technical occupations 6. Semi-routine occupations 7. Routine occupations	3. Routine & Manual occupations
8. Never worked and long-term unemployed	4. Never worked and long-term unemployed

Source: Adapted from Table 3. Eight-, five- and three- class versions. Standard Occupational Classification. Volume 3. The National Statistics Socio-economic Classification: (Rebased on the SOC2010) User Manual. Office for National Statistics, p.13. (http://www.ons.gov.uk/about-statistics/classifications/current/soc2010/ soc2010-volume-3-ns-sec--rebased-on-soc2010--user-manual/section-12--choosing-a-derivation-method.pdf)

C.2. Male life expectancy at birth by NS-SEC

Male Life Expectancy at Birth	1982-86	95% CI (+/-)	1987-91	95% CI (+/-)	1992-96	95% CI (+/-)	1997-2001	95% CI (+/-)	2002-06	95% CI (+/-)
Higher managerial & professional	75.6	0.7	76.6	0.6	77.5	0.6	78.8	0.7	80.4	0.6
Lower managerial & professional	74.3	0.7	75.4	0.6	76.5	0.6	78.2	0.5	79.6	0.5
Intermediate	73.3	0.8	74.5	0.8	75.3	0.8	76.8	0.8	78.5	0.8
Small employers & own a/c workers	73.6	0.8	74.4	0.8	75.6	0.7	76.6	0.7	77.8	0.7
Lower supervisory & technical	72.3	0.6	73.2	0.6	73.8	0.6	75.3	0.6	76.8	0.6
Semi-Routine	71.3	0.6	71.7	0.6	72.4	0.6	74	0.6	75.1	0.6
Routine	70.7	0.5	71.5	0.5	71.6	0.5	72.6	0.5	74.6	0.5
Condensed NS-SEC										
Managerial & professional	74.8	0.5	75.9	0.4	77	0.4	78.4	0.4	80	0.4
Intermediate	73.5	0.5	74.5	0.5	75.5	0.5	76.7	0.5	78.1	0.5
Routine & manual	71.4	0.3	72	0.3	72.5	0.3	73.8	0.3	75.4	0.3
Unclassified	60.2	1.4	60.3	1.2	65.8	1.3	67.2	1.2	71.5	1.1
All Men	71.7	0.2	72.6	0.2	73.8	0.2	75.2	0.2	77	0.2

C.3. Female life expectancy at birth by NS-SEC

Female Life Expectancy at Birth	1982-86	95% CI (+/-)	1987-91	95% CI (+/-)	1992-96	95% CI (+/-)	1997-2001	95% CI (+/-)	2002-06	95% CI (+/-)
Higher managerial & professional	80.9	1.1	81.7	1	82.3	0.9	82.6	0.8	83.9	0.7
Lower managerial & professional	79.7	0.7	81	0.6	81.2	0.5	82.2	0.5	83.4	0.5
Intermediate	79.6	0.7	81.1	0.7	81.4	0.6	81.5	0.6	82.7	0.6
Small employers & own a/c workers	79.1	1	79.9	0.9	80.7	0.9	80.8	0.8	82.6	0.8
Lower supervisory & technical	78.5	0.9	78.1	0.8	79.4	0.7	79.5	0.7	80.4	0.7
Semi-Routine	78.1	0.6	78.5	0.6	79.2	0.6	79.6	0.5	80.6	0.6
Routine	77.1	0.6	77.5	0.6	78.3	0.5	78.6	0.5	79.7	0.5
Condensed NS-SEC										
Managerial & professional	80.1	0.6	81.2	0.5	81.5	0.5	82.3	0.4	83.5	0.4
Intermediate	79.6	0.6	80.7	0.5	81.1	0.5	81.4	0.4	82.7	0.4
Routine & manual	77.7	0.4	78	0.4	78.9	0.3	79.2	0.3	80.2	0.3
Unclassified	71.5	1.1	73.1	1	74.2	1.2	75.8	0.9	76.9	0.9
All Women	77.4	0.2	78.3	0.2	79.2	0.2	79.9	0.2	81.1	0.2

C.4. Male life expectancy at age 65 by NS-SEC

Male Life Expectancy at Age 65	1982-86	95% CI (+/-)	1987-91	95% CI (+/-)	1992-96	95% CI (+/-)	1997-2001	95% CI (+/-)	2002-06	95% CI (+/-)
Higher managerial & professional	15.2	0.6	15.9	0.5	16.6	0.5	18.1	0.5	18.8	0.4
Lower managerial & professional	15.1	0.5	15.4	0.4	16	0.4	17.2	0.4	18.2	0.4
Intermediate	13.9	0.5	15	0.5	15.7	0.5	16.4	0.5	17.5	0.5
Small employers & own a/c workers	14	0.5	14.7	0.5	15.5	0.5	16.1	0.5	17.5	0.5
Lower supervisory & technical	13.4	0.4	13.7	0.3	14.5	0.3	15.3	0.3	16.4	0.4
Semi-Routine	12.9	0.4	13.3	0.3	13.8	0.3	14.7	0.4	15.6	0.4
Routine	12.9	0.3	13.1	0.3	13.4	0.3	14	0.3	15.3	0.3
Condensed NS-SEC										
Managerial & professional	15.1	0.4	15.6	0.3	16.2	0.3	17.6	0.3	18.4	0.3
Intermediate	13.9	0.4	14.8	0.4	15.6	0.4	16.2	0.3	17.5	0.3
Routine & manual	13	0.2	13.3	0.2	13.9	0.2	14.6	0.2	15.8	0.2
Unclassified	11.1	0.3	10.2	0.4	11.2	0.6	12	0.7	14.2	0.7
All Men	13.1	0.1	13.7	0.1	14.5	0.1	15.5	0.1	16.7	0.1

C.5. Female life expectancy at age 65 by NS-SEC

Female Life Expectancy at Age 65	1982-86	95% CI (+/-)	1987-91	95% CI (+/-)	1992-96	95% CI (+/-)	1997-2001	95% CI (+/-)	2002-06	95% CI (+/-)
Higher managerial & professional	19.7	1	20.1	0.8	20.8	0.7	20.9	0.6	21.7	0.6
Lower managerial & professional	18.9	0.6	19.4	0.5	19.3	0.4	20.3	0.4	21.1	0.4
Intermediate	18.3	0.6	19.5	0.5	19.6	0.4	19.8	0.4	20.5	0.4
Small employers & own a/c workers	18.6	0.8	18.9	0.7	19.6	0.6	19.1	0.6	20.5	0.6
Lower supervisory & technical	18.7	0.7	17.6	0.5	18.3	0.5	18.3	0.5	18.8	0.5
Semi-Routine	17.4	0.5	17.6	0.4	18.3	0.4	18.4	0.3	19.4	0.3
Routine	16.7	0.4	17.4	0.4	17.7	0.3	17.8	0.3	18.5	0.3
Condensed NS-SEC										
Managerial & professional	19.1	0.5	19.6	0.4	19.7	0.4	20.5	0.3	21.3	0.3
Intermediate	18.4	0.5	19.3	0.4	19.6	0.4	19.6	0.3	20.5	0.3
Routine & manual	17.3	0.3	17.5	0.2	18	0.2	18.1	0.2	18.9	0.2
Unclassified	16.2	0.2	16.2	0.3	16.3	0.3	16.6	0.4	17.4	0.5
All Women	17	0.1	17.5	0.1	18	0.1	18.5	0.1	19.5	0.1

C.6. Change in life expectancy at birth and age 65 by NS-SEC (1982-86 to 2002-06 and 1997-01 to 2002-06)

NS-SEC	Men at Birth		Women at Birth		Men at Age 65		Women at Age 65	
	Change b/t 1982-86 & 2002-06	Change b/t 1997-01 & 2002-06	Change b/t 1982-86 & 2002-06	Change b/t 1997-01 & 2002-06	Change b/t 1982-86 & 2002-06	Change b/t 1997-01 & 2002-06	Change b/t 1982-86 & 2002-06	Change b/t 1997-01 & 2002-06
Higher managerial & professional	4.8	1.6	3	1.3	3.6	0.7	2	0.8
Lower managerial & professional	5.3	1.4	3.7	1.2	3.1	1	2.2	0.8
Intermediate	5.2	1.7	3.1	1.2	3.6	1.1	2.2	0.7
Small employers & own a/c workers	4.2	1.2	3.5	1.8	3.5	1.4	1.9	1.4
Lower supervisory & technical	4.5	1.5	1.9	0.9	3	1.1	0.1	0.4
Semi-Routine	3.8	1.1	2.5	1	2.7	0.9	2	1
Routine	3.9	2	2.6	1.1	2.5	1.3	1.8	0.7
Condensed NS-SEC								
Managerial & professional	5.2	1.6	3.4	1.2	3.3	0.9	2.2	0.8
Intermediate	4.6	1.4	3.1	1.3	3.6	1.3	2.1	0.8
Routine & manual	4	1.7	2.5	1	2.8	1.2	1.6	0.8
Unclassified	11.3	4.3	5.4	1.1	3.1	2.2	1.2	0.8
All	5.3	1.8	3.7	1.2	3.6	1.3	2.5	0.9

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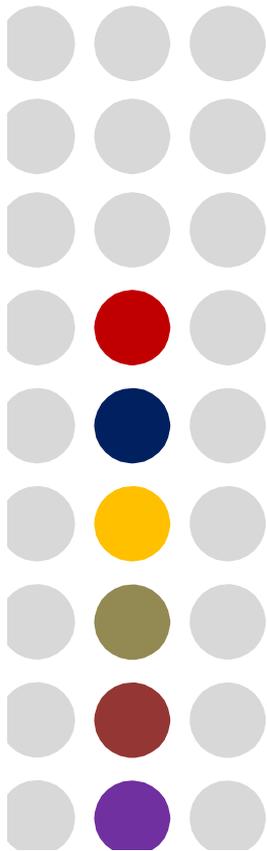
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