

Gender-Specific Life Expectancy in Europe 1850–2010

Edited by Martin Dinges and Andreas Weigl

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Gruppenbild rauchende Soldaten in Esnes, Frankreich, 1916.

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The drifting apart of gender-specific life expectancies in Europe 1850–2010

Some introductory remarks

Andreas Weigl

At least as far as the last two or three decades are concerned there can be no doubt that the gender gap in life expectancy has become a topical research issue in a number of separate scientific disciplines, both in the natural and the social sciences. There is an obviously increasing demand for gender studies on health behaviour and its consequences on health policies on a national, European and international level. The conclusive common ground is nevertheless limited, because these studies are based on two quite contradictory approaches: the biological on the one hand and the behavioural and environmental on the other hand. Due to the fact that methods and “scientific cultures” of these approaches differ widely it is not surprising that general conclusions are rare and often more or less simple enumerations of factors from both sides. But there are some exceptions. Some authors have attempted to calculate crude weightings of biological and “cultural” influences¹, while others argued more cautiously by stressing the need for more “intersectional” approaches in studies of both health related behaviour and mortality.² In 2005 a convincing model of gender health inequality was elaborated by Birgit Babitsch, who teaches New Public Health at the University of Osnabrück. The three level model (macro/meso/micro) integrated both behavioural and biological factors, though the focus is predominantly on the former.³ Unfortunately for the question of the gender gap in life expectancy, the results of Babitsch’s study based on German data is only to some extent helpful, because understanding morbidity differentials by either sex or gender does not necessarily lead to a full understanding of differentials in mortality. In general one receives the impression that there is still a wide methodological gap to overcome. It can be shown easily that even scientists who gained degrees in the humanities and in the natural sciences tend to stress or neglect either the biological or the behavioural factors.⁴

Therefore there are still a lot of open questions to be answered and multidisciplinary remains “wishful thinking”. This does not only apply to research in the natural versus social sciences, but also to the problem of social and historical disciplines. The social sciences in particular have given little attention to the historical roots of gender-specific lifestyles and the long-term transformation of gender roles, while – since the “cultural turn” in the study of history

1 Johansson, 1991; Luy 2002a.

2 Waldron, 2000, p. 177.

3 Babitsch, 2005, pp. 138–141.

4 Austad, 2006.

– many historians have largely ignored quantitative research. Fortunately, there is one discipline that is rooted in both fields. Demography has – to a certain extent – managed to consider the historical view as well as include the findings of other social sciences and the natural sciences in its analyses.

The late nineteenth and twentieth centuries are of particular interest for historical research into the gender gap in life expectancy in the industrialized world. Because this period is very close to the present it allows the use of historical findings as a means of enhancing recent research into women's and men's mortality. In addition, the encouraging availability of data – compared with the early modern period – and, not least, the remarkable development of the gender gap during that period offers the chance to gain a deeper understanding from international comparisons. The comparison of Northern, Western and Central Europe in this volume was chosen for pragmatic reasons because these countries offer a sufficiently wide range of gender gap variations and the actual state of research is sufficient for a relatively differentiated comparison.⁵ Nevertheless, it would certainly make sense in further research to include eastern and southern Europe as well as non-European industrialized countries in the picture.

As mentioned before, specific research in the natural and cultural sciences proves that the gender gap can best be explained against the background of a combination of biological, genetic and behavioural factors. The significance of these factors is, however, disputed. Research in communities with very similar living conditions for men and women provides evidence that the gender gap has only a minor biological component. Following almost identical results from investigation of the general population and that of monasteries, it was rated for adults at one year or one and a half years at the most.⁶ This figure is higher, however, when we include the influence of biological factors on behavioural roles.⁷ Genetic-biological factors that influence the gender gap are best proved with babies, since in babies, and to a limited extent also in young children, acquired gender-specific behaviours hardly influence the excess mortality of boys. Although infant mortality decreased substantially in late nineteenth and twentieth century Europe, an advantage persisted for girls in their first year of life. Based on an analysis of infant mortality by major causes of death Ingrid Waldron has shown that there are multiple sex differences in biology and not all of them are a disadvantage for male infants. "The available evidence suggests that males have inherently greater vulnerability for mortality due to perinatal conditions and for total mortality in the neonatal period, but the assumption that males have a pervasive inherent disadvantage is incorrect for some types of congenital anomalies, and is of uncertain validity for

5 Unfortunately it was not possible to find colleagues to present the French case; but cf. Vallin / Meslé, 1988, chap. 12, pp. 467–505, for a differentiated analysis of the causes of death and their contribution to the gender gap until 1978.

6 Luy, 2002a, p. 424.

7 Ritzmann, 2001, p. 70.

infectious diseases and total mortality in early childhood.”⁸ This indicates that, although the weight of infant mortality for the overall gender gap in mortality is almost fading away, further medical and biological studies in this field should not be ignored.

Although the attempts to estimate the components of the gender gap are quite valuable to adjust downwards popular estimates in the medical sciences⁹ I would suggest that we should consider the combination of biological and behavioural factors not only as a simple equation of “ $a + b = c$ ”. If newborn girls come with a basic gender-specific advantage – and there is much to support this assumption – this advantage can be tapped to a greater or lesser extent during their lives. Furthermore, recent medical research also shows that a person’s basic genetic disposition can be improved or made worse during their lifetime.¹⁰ Splitting the causes of the gender gap into behavioural-“social” and “biological” factors is therefore an auxiliary structure that supports a statistically more complex situation.

While biological explanations of the gender gap due to the recent rise of genetics seem to me to gain greater importance again in medical research – which would mean that older medical explanatory models are staging a comeback – the social sciences, social medicine included, underscore the part played by gender-specific behaviour patterns and lifestyles. Their findings are corroborated by those of historians, but the interpretations of these findings differ widely. While some see the gender gap in life expectancy as proof of the provocative thesis, formulated by Martin van Creveld, that women are a “privileged sex”¹¹ or that men are “health idiots”¹², others, such as Maria Danielson and Gudrun Lindberg, speak of a “new gender paradox”: This new gender paradox implies that women live longer than men despite the fact that they continue to be discriminated against in society.¹³ The old paradox – dismissed by Danielson and Lindberg – states the alleged greater health impairment of women compared to men.¹⁴ From this old paradox it was no big step to DFLE (disability-free life expectancy) and HALE (health-adjusted life expectancy), concepts that basically made the gender gap in industrialized countries in the disability-free part of the life span disappear because, on average, women suffer from chronic disease for longer periods of time. This is a fact documented by a huge number of studies. But the definition of such severe health conditions remains vague and assessment of such values is biased as a result of national statistical traditions. In addition one gets the impression that the interpretation of the results of studies based on these concepts is strongly

8 Waldron, 1998, p. 79.

9 See for instance Klotz, 1998, p. 101, who prorates 60% of the gender gap to genetic-biological factors.

10 Tammen / Friso / Choi, 2013.

11 Van Creveld, 2003.

12 Dinges, 2005, p. 512.

13 Danielson / Lindberg, 2001, p. 61.

14 Babitsch, 2005, p. 65.

influenced by the current political gender agenda in the European Union, which is hardly focused on male disadvantages in public health.¹⁵ Introducing such concepts as the DFLE and the HALE may be justified with a view to gender-specific health policies, but it does not really help to explain the gender gap in life expectancy. On the contrary: it raises a new question: Why is it that women survive chronic illness for longer than men? If this question is not explained by gender-specific biological differences alone, it continues to be a field of research in social medicine.

But what insights can be expected from a comparative historical view? From the late nineteenth century the widening of the gender gap in life expectancy became a European phenomenon that continued into the 1980s, in eastern European transition countries even beyond the fall of the Iron Curtain in 1989. This widening varied, however, in the various countries and groups of countries, a fact that points to the influence of “exogenous” factors such as the two world wars, but not least also to different economic and social developments with regard to the working world and gender roles.

Richard Wilkinson and Kate Pickett have shown in a recent comparative study that within the group of countries with high GDPs per head a more even income distribution and an advanced health care system contribute to a general high level of life expectancy.¹⁶ This strengthens the argument that economic well-being is a key factor for a narrowing of the gap, but it reveals that the relationship is more complex. Even if the relatively small gender gap in life expectancy of some Scandinavian countries and the Netherlands needs to be seen in the context of economic vitality and modern societal structures, economic inequality, which was and is low in some North European welfare states, can clearly not adequately explain the development of the gender gap in industrialized countries during the twentieth century, because the last three decades saw a narrowing of the gender gap in life expectancy and a widening of economic inequality, not to mention the fact that beside undisputable discrimination of women in many developing countries the gender gap in favour of the female population was a global phenomenon in the early twenty-first century. I refer in particular to the headline of an editorial in a 2006 issue of the *British Medical Journal*: Life expectancy: Women are now on top everywhere!¹⁷

While many pertinent studies, such as the British *Black Report* of 1982¹⁸, show a considerable gradient for life expectancy depending on income and education (as a proxy), the range is much smaller with women than it is with men. Studies from other countries, including Austria, show that the life expectancy of a female labourer was and still is as high or even higher as that of an

15 For a critical discussion see Dinges / Weigl, 2011, p. 198, fn. 20.

16 Wilkinson / Pickett 2009.

17 Barford et al., 2006.

18 Black, 1992.

academic.¹⁹ According to Pierre Bourdieu's capital theory²⁰ this is a strong hint that the uneven distribution of "social capital" must be included in the analysis too. There is much to support the view that women have the better "social capital" due to their traditional gender roles and possibly also due to their biological make-up.

As mentioned before, some demographic studies have successfully refuted explanations of the gender gap that are based on purely biological premises. For instance in the long-term comparison of monastic populations (monks and nuns) with the general population, Marc Luy was able to present compelling evidence that – at least for adults – it was mostly the male gender roles and the strains of working life (stress at work, consumption of addictive substances) that enhanced the widening of the gender gap during the twentieth century.²¹ Luy's study results therefore refer to a specific field of inequality research, the research into people's lifestyles. Unfortunately, gender-specific lifestyles have so far not been the preferred subject of sociological research.²² The few existing studies certainly present a wide range of differing gender "health behaviours", in particular on the male side.²³ On the basis of a cluster analysis a Swiss study showed a statistically significant connection, for men, between "somatic culture" and social milieu but not social class.²⁴ In addition, this study stressed the fact that men in general still often need to justify body-sensitive health behaviours.²⁵ This need for justification is clearly fed by the explanation pattern that became popular in the 1950s, which claims that the excess male mortality was the price to be paid for modern (Fordist) work situations and a male-dominated success-oriented working world – the rise of the male manager.²⁶

Making the changes in the working world during the twentieth century alone responsible for the growing gap in life expectancy up to the 1980s would be a questionable step, however. The service societies that emerged in Europe in the last decades of the twentieth century and the parallel exodus of (heavy) industries to developing countries obviously reduced the physical hazards of the working world and this has affected men in particular. Accident statistics and the drop in what is termed "occupational diseases" also support this observation although the male/female-ratio in this respect is quite stable.²⁷ But this says nothing about mental stress which seems to be an influential factor if one considers the prominent mortality due to cardiovascular disease and can-

19 Klotz, 2008, p. 150.

20 Bourdieu, 1992, pp. 49–79.

21 Luy, 2003, pp. 647–676.

22 Abel, 1999, pp. 43–61.

23 Robertson, 2007, p. 156; Nettleton / Watson, 1998; Ervø / Johansson, 2003; Schneider 2002.

24 Nideröst, 2007, p. 97, 106.

25 Robertson, 2007, p. 63.

26 Forth, 2008, p. 205; Ehrenreich, 1983, p. 70.

27 For instance in Austria in the time period 1970–2012 the ratio is about 3 to 1. See Statistik Austria, 2014, pp. 84–89.

cer. Given the established high shares of high-ranking male managers and full time jobs in general this could explain the still existing higher mortality of men, at least in cardiovascular disease.²⁸ The uneven distribution of time spent on childrearing and housework between female and male employees is still present and diminishing rather slowly.²⁹ The question that remains unanswered is whether there are gender-specific differences in respect of stress-related mortality. For the last three decades the narrowing of the gender gap at least coincides with an interpretation of stress as an equalizer of male and female mortality, seeing that the incidence of “burnout-related diseases” has risen among the highly-qualified female workforce as well. The same is true for the shrinking of the gap in gender-specific consumption of nicotine and alcohol that has been demonstrated by studies for many industrialized countries.³⁰

This brings us to another question that has, in my opinion, still not been sufficiently answered and that relates to the long-term effect of the two world wars. Although some crude estimates are available,³¹ the effect of chronic illness, war injuries and other morbidities caused by the war is not easy to measure and certainly goes beyond these “direct” consequences. As a recent study based on an evaluation of (West-)German, Austrian and Swiss “popular autobiographies” has shown, even the long term mental consequences of wars are a very complex issue. In her thesis on gender specific discourse and health-related lifestyles Susanne Hoffmann stressed the point that there is evidence of a positive judgement of severe war-injuries, because wounding could bring soldiers out of danger zones, at least as far as surviving soldiers are concerned.³² On the contrary, it is possible that the two wars played a part in retaining and reinforcing images of a “military masculinity” – after World War II transformed in male economic competition in the “battlefield” of the labour market³³, at least among the men of age groups that were actively involved in the war or educated during war times, and therefore in further widening the gender gap. New insights will emerge in this volume from the comparison of countries and populations that were, to various degrees, involved in the wars.

To summarize: for a long term perspective on the gender gap in life expectancy in the “long” twentieth century the following questions are particularly relevant:

1. Why did the gender gap in life expectancy widen in a time period when sectoral change and automation in manufacturing reduced the disadvantages of the male working population, at least as far as physical strains (in factories, in the construction business) were concerned.

28 Siegrist, 2010, p. 79.

29 See i. e. McGinnity / Russell, 2008.

30 Johansson 1991, p. 157.

31 Haudidier, 1996; Höhn, 1996

32 Hoffmann, 2010, pp. 288–299.

33 Hanisch, 2005, pp. 118–121.

2. What are the long-term effects of the two World Wars, since the effect of war and the consumption of addictive substances overlap? A comparison between nations involved in the war and neutral states would allow for a more accurate evaluation of this factor.
3. Considering the partly diametrically opposed developments of female labour force participation, a comparison of the gender gap in West and East Germany from 1949 to 1989 or between countries with a long tradition of high female labour force participation (Sweden) and countries with the opposite tendency (FRG, Switzerland, Austria) would be particularly interesting regarding the influence factor “world of work”.

Comparing the state of research in the various countries into sub-disciplines relevant to this question – such as (historical) demography, economic and social history, the history of medicine, social medicine and gender research – might also yield synergies and new insights, especially as far as the reduction of the gender gap in life expectancy in highly developed countries in the last three decades is concerned.

What links the demographic case studies collected in this volume is a long term perspective with a focus on the twentieth century. The course of the gender gap over 150 years in the advanced Atlantic economies of North-West Europe has been elaborated by Alice Reid and Chris Dibben in their study on gender specific mortality trends over the epidemiological transition on the British mainland including the “national patterns” of England/Wales and Scotland, and by Frans van Poppel and Fanny Janssen on the Netherlands. Given the differing involvement of these countries this also opens insights into the deteriorating effects of World War I. Based on a smaller set of data these effects were discussed by Andreas Weigl in his article on Austria in the first half of the twentieth century as well, though his main focus is on the changing working environment. The Austrian experience of a reduction of the gender gap in life expectancy since the 1980s is discussed by Johannes Klotz. Klotz stresses the importance of high education for the particular convergence of male and female life expectancy, reinforcing academics as a “vanguard group”. A different methodological approach is presented by Marc Luy. Luy’s comparison of Catholic order members and the overall German (East and West) population in the second half of the twentieth century is embedded in a broad overview of the biological/non biological factors-debate that traces, as he demonstrates, back to the eighteenth century. Furthermore, like several other contributions of this volume, Luy’s study also includes an analysis of non-biological factors like smoking. A comparison of the Benelux experience is another interesting issue. It is no big surprise that Patric Deboosere’s case study on the gender gap in Belgium in the nineteenth and twentieth century shows important similarities as well as differences compared to the Netherlands. The same could be said of the neutral countries Switzerland and Sweden. An overview of the much more fluctuating Swedish trends in the gender gap is given by Sam Willner, while Örjan Hemström (like Johannes Klotz in the case of Austria) discusses the reasons for the narrowing of the gap in the

last decades. Although Switzerland, like Sweden, was not involved in both World Wars, the Swiss case is much more “Central European” than “Scandinavian” as the papers of Raymond Kohli in general and Christoph Junker by analysis of cause specific mortality show

The questions I have outlined and those raised in the case studies and the editors’ résumé in this volume do, of course, not constitute all the questions that are still open with regard to the gender gap in life expectancy and not all of them should be seen in a long term perspective. But even if one focuses on the most recent changes in the gender gap, social history cannot be ignored. As an analysis based on meta-data of 72 recent studies on the subject reveals, specific male subpopulations are in all probability the core factor of the gender gap in life expectancy in present industrialized countries.³⁴ If this conclusion is correct, public health programs focusing on these specific subpopulations certainly need a historical perspective for a deeper understanding of these risk groups including socialization, representations of masculinity and many more factors. Almost all these factors are in a wider sense embedded in social, economic, cultural history. To conclude: history matters in the long run.

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34 Luy / Gast, 2013.

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The impact of biological factors on sex differences in life expectancy: insights gained from a natural experiment

Marc Luy

1. Introduction

That women live longer than men has been known at least since the middle of the eighteenth century when Kersseboom mentioned his observation that the mortality experiences of males and females differ sufficiently to make it worthwhile using separate tables for calculating annuities.¹ A few years later, the first sex-differentiating life tables by Struyck and Deparcieux added the corresponding empirical evidence.² The finding of male excess mortality was confirmed with the introduction of official population statistics in all western societies and has been documented in Sweden from 1751 onwards.³ Until recently, a higher life expectancy at birth for men was known only for some countries in Africa and Asia, mainly due to an excessive female mortality among infants and in early childhood.⁴ A few years ago, Barford et al. announced in a British Medical Journal editorial entitled “Life expectancy: women now on top everywhere” that females outlive males now even in the poorest countries of the world.⁵

Men have higher mortality than women not only in terms of overall measures like life expectancy at birth but also – at least in industrialized societies – in all ages and leading causes of death. The mortality differences between women and men remained more or less constant until the end of the nineteenth century and started to increase during the twentieth century. This increase of the sex gap coincided with a rise among men in cardiovascular disease, cancer, and accidents, and a fall in maternal mortality and in causes of death related to pregnancy.⁶ However, since the beginning of the 1980s the gap between women and men in overall mortality has been slowly narrowing in the western world. In Eastern Europe, the trend reversal set in during the 1990s and only recently reached Japan, the sole laggard in the western world.⁷

The hypotheses advanced to explain male excess mortality can be divided into two basic categories, those concentrating on the biological factors (factors largely beyond human control which are also called “inherited risks”) and those concentrating on non-biological factors (behavioural, cultural and environmental factors, i. e. factors directly or indirectly influenced by human ac-

1 Kersseboom (1737), Kersseboom (1740).

2 Deparcieux (1746), Struyck (1740).

3 Tabutin (1978).

4 See e.g. Aden et al. (1997), Langford (1984).

5 Barford et al. (2006).

6 Lopez (1983).

7 Liu et al. (2013).

tion which are also called “acquired risks”). The three following sections summarize the main biological (section 2) and non-biological factors (Section 3) discussed in the literature, as well as the various interactions between them (Section 4). Section 5 includes an overview of the existing attempts to quantify the impact of biological factors and section 6 provides a refined estimation of the biologically caused sex difference in life expectancy based on our data for Catholic order members from Germany and Austria. Section 7 demonstrates the empirical application of this estimate to data from Germany by isolating the impact of biological factors in order to estimate the impact of smoking on sex differences in life expectancy in comparison to other non-biological factors. The article ends with a discussion in which our estimates of the impact of biological factors are compared to those of other studies on sex differences in mortality among Catholic order members.

2. Biological factors

Ever since the differences in mortality between the sexes have been known, one has assumed that women outlive men because humans are subject to a general “law of nature” according to which females enjoy a longevity advantage.⁸ There are several biological differences between the sexes which might be responsible for this natural female survival advantage.⁹ It is the established view that sex differences in mortality are built upon a “fundamental genetic basis”¹⁰ which is supposed to act along three basic axes:

- The first relates to the implications of homogametic (XX) and heterogametic sex (XY) in the viability of the human organism.¹¹ In each female cell, one X chromosome is randomly inactivated, protecting women against a double dose of X chromosome expressions as well as against disadvantageous genes on one X chromosome.¹²
- Another relevant genetic sex difference is seen in relation with the positive correlation between telomere length and length of life, as men exhibit shorter telomeres than women because of faster telomere attrition.¹³ This line of reasoning is directly related to the “costly growth hypothesis” stating that the costs of growing and the sexual size dimorphism lead to higher mortality in the sex with the larger body size.¹⁴
- Most recently, it has been proposed that the mitochondrial genome is optimized for function with the female genome through natural selection as

8 Trivers (1972).

9 For an extensive overview see Seifarth et al. (2012). The summary of hypotheses and findings presented in this Section is based primarily on this reference.

10 Hayflick (1982), p. 248.

11 See e.g. Christensen et al. (2001), Puck / Willard (1998), Smith / Warner (1989).

12 Seifarth et al. (2012).

13 Barrett / Richardson (2011), Stindl (2004).

14 Kalmbach et al. (2005), Owens (2002), Promislow (1992), Samaras et al. (2002).

humans inherit mitochondria from mothers only.¹⁵ As explicated by Seifarth et al.,¹⁶ this optimization of mitochondrial functionality in females might result in a survival advantage because mitochondrial dysfunction has been demonstrated to be related to ageing¹⁷ as well as to a number of mortality relevant diseases such as cancer¹⁸ and cardiovascular disease.¹⁹

Upon this genetic basis, the endogenous sex hormones testosterone and oestrogen comprise the second group of biological factors which are likely to affect the mortality of women and men. Again, there are several routes on which hormones are expected to contribute to sex differences in life expectancy:

- One important sex-specific hormonal effect concerns the handling of lipids. Men tend to store more fat in the abdominal region, whereas women tend to store more fat in hips, thighs and buttocks. Moreover, women tend to have greater amounts of subcutaneous fat, whereas men are more likely to accumulate visceral adipose tissue which has been implicated in a number of diseases including metabolic syndrome,²⁰ coronary artery disease²¹ and ischemic heart disease,²² among others.²³ These sex differences in body fat distribution are complemented by sex differences in the lipoprotein metabolism. Most importantly, oestrogens have been shown to increase HDL (“good cholesterol”) and lower LDL (“bad cholesterol”) levels, whereas androgens lower HDL concentrations but raise those of LDL.²⁴ Cholesterol is a well-known risk factor for atherosclerosis. Differences between women and men in sex hormone levels therefore lead to a sex differential in lipoprotein metabolism which is supposed to cause sex differences in cardiovascular disease and these, in turn, lead to sex differences in mortality.²⁵
- Other studies indicate that hormonally caused sex differences in “immuno-competence”—i. e. an organism’s all-around ability to avoid the harmful effects of infections – may underlie male excess mortality, supposing that oestrogens are immunity enhancers, whereas androgens and progesterone are natural immunosuppressants.²⁶ However, a study by Fairweather and Cihakova suggests that the female advantage in infection resistance may turn into a disadvantage when an immune response is initiated against host

15 Tower (2006).

16 Seifarth et al. (2012).

17 Trifunovic / Larsson (2008).

18 Brandon et al. (2006).

19 DiMauro / Andreu (2000).

20 Albu et al. (1997).

21 Nakamura et al. (1994).

22 Matsuzawa et al. (1994).

23 For more details see Seifarth et al. (2012), pp. 393–394.

24 Hazzard (1989), Hazzard / Applebaum-Bowden (1990).

25 See Hazzard (1986), p. 464.

26 Caruso et al. (2013), Hamilton (1948), Hazzard (1989), Moore / Wilson (2002), Owens (2002).

- cells.²⁷ Consequently, whereas males are more susceptible to infection, females may be more susceptible to autoimmune disease. Nonetheless, this contrasting susceptibility still favours women because infectious diseases, cancer and cardiovascular disease – all with significant male excess – produce much higher mortality than autoimmune diseases.²⁸
- Another hypothesis based on sex hormones relates sex differences in mortality to sexually dimorphic mechanisms of combatting oxidative stress. The thesis is built on the observation that the combination of the antioxidant properties of oestrogen and associated antioxidant genes in females leads to more favourable handling of oxidative stress and its accumulation over the lifespan.²⁹
 - A further hormone based sex dimorphism has been suggested by physiological studies of the hypothalamic-pituitary-adrenal (HPA) axis stress response to psychosocial stress. For instance, Kirschbaum et al. found that the mere prospect of an upcoming psychological stress task produced a cortisol response in men, but not in women.³⁰ This finding has been supported by Dahl et al., who found that male children exhibit a significantly higher cortisol response to corticotropin-releasing hormone ingestion.³¹ At the organ and cellular levels, several differences in the ability of female cells to deal with cellular perturbations have been documented.³² Although these findings were drawn from studies with rats they point to sex differences in the hormonal and cellular response to stress, which could contribute to the female advantage in life expectancy.³³ Nielsen suggests that androgens affect foetal lung development via a mechanism dependent on the presence of androgen receptors within the HPA axis, causing male infants to be at greater risk of respiratory distress syndrome than female infants.³⁴

Male excess mortality has been observed in very different animal species, including house flies,³⁵ rats,³⁶ chimpanzees,³⁷ and many others.³⁸ Among humans higher male mortality rates hold among children³⁹ and even among infants and in the prenatal period, when higher rates cannot be caused by ac-

27 Fairweather / Cihakova (2009).

28 For more details see Seifarth et al. (2012), pp. 395–396.

29 Behl et al. (1997), Borrás et al. (2003), Proteggente et al. (2002).

30 Kirschbaum et al. (1992).

31 Dahl et al. (1992).

32 Brown et al. (2005), Thorp et al. (2007).

33 For details see Seifarth et al. (2012), pp. 396–397.

34 Nielsen (1985).

35 Rockstein / Lieberman (1959).

36 Asdell et al. (1967).

37 Hill et al. (2001).

38 Ciocco (1940), Clutton-Brock / Isvaran (2007), Comfort (1979), Hamilton (1948), Judge / Carey (2000), Smith (1989).

39 Théré / Rohrbasser (2006).

quired risks.⁴⁰ The existence of at least a biological basis for the female survival advantage is therefore undoubted.

3. Non-biological factors

The biological sex differences in mortality are complemented by a number of non-biological impacts on the mortality of women and men. Corresponding research argues that society and culture influence men to lead lifestyles that are increasingly detrimental to health and life (in terms of smoking habits, alcohol consumption, reckless driving, diet, exercise, involvement in religious activities, etc.), that men are subjected to greater health risks at work, that environmental factors lead to survival disadvantages for men, and that men are generally more exposed and susceptible to different kinds of social and psychological stress than their female counterparts. Several causation lines can be found in the literature:

- Many studies suggest that nicotine consumption is the health behaviour contributing most to increasing male excess mortality.⁴¹ In general, men have higher proportions of smokers than women, they start smoking earlier and therefore smoke longer, and they smoke more and stronger cigarettes than women. Smoking also appears to play a considerable role in the currently observable narrowing of the male-female differentials in mortality since the proportion of female smokers has increased greatly in recent decades,⁴² complemented by declining differences between women and men in alcohol consumption.⁴³
- Estimates for occupational hazards show that risks caused at the workplace are related to a number of severe and fatal diseases.⁴⁴ Men are exposed more to occupational hazards because more men are employed, and among those who are employed more men than women work in hazardous occupations. Reviewing several studies, Waldron concluded that approximately 95% of fatal work accidents involve men and that these higher rates for work accidents account for roughly one-fifth of the sex differences in accident fatalities.⁴⁵ In a multivariate analysis of Swedish labour force participants, Hemström arrived at the same estimate that sex differences in occupational fields account for approximately 20% of men's excess mortality from external causes.⁴⁶ This contribution originated mainly from high job hazard levels in traditional male jobs including, for example,

40 Hazzard (1986), Hazzard / Applebaum-Bowden (1990), Kalben (2000), Lopez (1983), Waldron (1985), Wingard (1982).

41 E. g. McCartney et al. (2011), Payne (2001), Waldron (1986).

42 Luy / Wegner-Siegmundt (2015), Nathanson (1995), Pampel (2002).

43 Martelin et al. (2004), Simons-Morton et al. (2009).

44 E. g. Concha-Barrientos et al. (2004), Leigh (1988), Nurminen / Karjalainen (2001).

45 Waldron (1991).

46 Hemström (1999).

- heavy lifting, heavy shaking or vibration, contact with dirt, inadequate ventilation and exposure to gas, vapour or smoke and exposure to chemicals and carcinogens.⁴⁷
- Social stress is seen as another basic causal factor for male excess mortality.⁴⁸ In this context, Jenkins introduced the term “type A behaviour”, which is characterized by intensive striving for achievement, competitiveness, easily provoked impatience, time urgency, abruptness of gesture and speech, over-commitment to vocation or profession, and excess of drive and hostility.⁴⁹ In western societies, type A behaviour is found more frequently among men since it is strongly linked to professional life and social status.⁵⁰
 - Because lifestyles generally differ with the level of social status, male-female differences in mortality could also be affected by the fact that men and women are not equally distributed within various social classes.⁵¹ Nathanson and Lopez hypothesized that the extent of male excess mortality is mainly determined by the harmful lifestyles of men of low socioeconomic status.⁵² Wingard et al.⁵³ and Luy and Gast⁵⁴ supported this hypothesis with different empirical approaches.
 - Finally, a survival advantage among women may also be inferred from the tendency in women to consult a doctor more often than men, both on noticing symptoms of illness and because of their health care needs related to childbearing.⁵⁵ This could lead to an early detection of serious diseases with increased chances to treat them successfully.⁵⁶ The contribution of this factor to the sex difference in mortality is discussed controversially, however.⁵⁷

All these acquired risks develop in the context of the prevailing economic, social, cultural and political system that immediately affect the opportunity structures of individuals and groups and the external forces that affect their behaviour and life chances.⁵⁸ Some research suggests that the population’s living environment itself is the central driver of the non-biologically caused difference in mortality between women and men. For instance, Preston argues that increasing sex differences in life expectancy during the twentieth century were an effect of the economic modernization of society improving the status of women more than that of men, and this leading to a greater reduction in

47 See also Waldron (1991).

48 Jarvik (1963).

49 Jenkins (1976).

50 Luy / Di Giulio (2005), Luy / Di Giulio (2006), Nathanson (1984), Waldron (1978).

51 E. g. Johansson (1991), McDonough et al. (1999), Vallin (1995).

52 Nathanson / Lopez (1987).

53 Wingard et al. (1983).

54 Luy / Gast (2014).

55 Galdas et al. (2005), Hazzard (1986), Verbrugge / Wingard (1987), Wallen et al. (1979).

56 Lang et al. (1994).

57 Johansson (1991), Verbrugge (1985).

58 Angel (2011), Anson (2003).