The Impact of Smoking and Other Non-biological Factors on Sex Differences in Life Expectancy in Europe

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Abstract

Tobacco consumption is seen as the predominant driver of both the trend and the extent of sex differences in life expectancy. We compare the impact of smoking to the effect of other non-biological factors to assess its significance among the causes that can be influenced by direct or indirect interference. Sex differences in life expectancy are decomposed into fractions caused by biological factors, smoking, and other non-biological factors for 44 European countries and the period 1950-2009. We find that the trend of the sex gap in life expectancy can indeed be attributed to smoking in most populations of Western Europe. In most Eastern European populations, however, other non-biological factors have a stronger impact than smoking. With regard to the overall extent of male excess mortality, smoking is the main driver only in 21 of the studied populations. Thus, over-generalised statements which might suggest that smoking is the main force behind the sex gap in all populations are misleading. The public health sector rather needs population-specific estimates to introduce the most appropriate measures to further reduce the inequalities in life years between women and men. While the impact of smoking to the sex gap in life expectancy declines in all studied populations, the contribution of other non-biological factors is in most cases higher at the end than at the beginning of the observation period. This demonstrates that, regardless of the prevailing effect of smoking, many European populations have still remarkable potentials to further narrow their sex gaps in life expectancy.
1. INTRODUCTION

Throughout the developed world, women live several years longer than men. Nonetheless, the sex gap shows huge variations and underwent significant changes during the 20th century (Büttner 1995, Trovato and Lalu 1996, Newman and Brach 2001, Gjonça et al. 2005, Trovato and Heyen 2006, Liu et al. 2013). The differences in life expectancy between women and men remained more or less constant until the 1940s and started to increase thereafter. This trend came to a halt in basically all western populations at the beginning of the 1980s, when the gap decreased immediately or with a time lag of some years. In Eastern Europe, the trend reversal set in during the 1990s and only recently reached Japan, the sole laggard of the western world. Nevertheless, the sex gap in life expectancy is still striking in all developed populations. It currently ranges from around four years in Iceland and Israel to more than twelve years in Russia.

As all industrialised societies continue to age and the shares of older people grow, women’s higher longevity has become an ever more important factor in public health and social policy (Lemaire 2002). Hence, understanding the causes underlying these differences is an essential prerequisite for designing adequate coping strategies. Ever since the 1970s, many researchers have identified cigarette consumption as the most important reason for the sex differential in mortality (Preston 1970, Burbank 1972, Retherford 1972, Retherford 1975, Waldron 1985, Nathanson and Lopez 1987, United Nations Secretariat 1988, Nathanson 1995, Zhang et al. 1995, Pampel 2001, Pampel 2002, DesMeules et al. 2004), usually in direct relation to the so-called ‘smoking epidemic model’ (Lopez et al. 1994). One of the most famous studies in this context was conducted by Miller and Gerstein (1983) who found that sex differences in life expectancy were practically non-existent in a specific sample of non-smokers. Although this finding was criticized (Holden 1983, Feinleib and Luoto 1984) and challenged by other empirical investigations (Rogers and Powell-Griner 1991, Bronnum-Hansen and Juel 2001), its message helped to create the
common view that smoking is the predominant driver of both the trend and
the extent of differences in life expectancy between women and men—a
perspective that prevails until today (e.g. Lindahl-Jacobsen et al. 2013).

However, several aspects raise doubts about the general validity of
this viewpoint, which, in most cases, implicitly refers to women’s and men’s
life expectancy at birth. It is well known that sex differences in mortality are
caused by a complex combination of biological (genetic, hormonal) and non-
biological (behavioural, economic, social, environmental and cultural)
Within this network of causes, tobacco consumption is just one of many non-
biological risk factors and, in addition, a cause that only has an impact from
the mid-adult ages onwards. Moreover, populations not only differ in the
smoking prevalence of women and men but also regarding the time and
speed they pass through the smoking epidemic (Waldron 1991, Nathanson
2006, Peto et al. 2006). Although it appears plausible that trends in cigarette
consumption have played a decisive role in the observed changes in sex
differences in life expectancy since the middle of the 20th century (besides
the above mentioned studies see Waldron 1986, Waldron 1993, Hummer et
seems rather unlikely that smoking is also responsible for most of the overall
male excess in mortality in all developed populations.

In fact, studies that quantify the impact of smoking on sex
differences in mortality state a wide range of results, which depend on the
investigated population, the included age groups and the respective
observation period. For instance, Valkonen and van Poppel (1997) estimate
in their study of five northern European countries that the contribution of
smoking to sex differences in life expectancy at age 35 ranged from 14% in
Sweden in the period 1985/89 to 72% in the Netherlands in 1970/74.
Contingent upon the specific roles of other behavioural and socio-economic
risk factors, Rogers et al. (2005) identify a similarly broad impact range of
30-83%. In a more recent study, McCartney et al. (2011) estimate that
smoking accounted for 40-60% of the sex gap in 30 European countries in the first years of the 21st century. Estimates for the US population, to which most existing studies refer, range from 50% to 62% for the 1960s and 1970s (Retherford 1972, Waldron 1986) and from 22% to 25% for the 1990s (Rogers et al. 2000, Rogers et al. 2010).

These examples demonstrate that the role of smoking in determining the extent and the trend in sex mortality differences is not as evident as often stated. Moreover, a general assessment is impeded by the fact that available studies not only differ regarding the investigated populations, age groups and calendar years, but also regarding the methods used for quantifying overall mortality and mortality attributable to smoking (including the basic concepts of relative versus absolute measurements as well as period versus cohort analysis). To overcome these shortcomings, we use the same data and identical well-established methods to estimate the impact of smoking on sex differences in life expectancy from the middle of the 20th century until today for a large number of European populations and for the same periods of calendar years. We compare the impact of smoking to the combined effect of all other non-biological factors. This approach is helpful in assessing the significance of tobacco consumption among the causes leading to sex differences in life expectancy that can be influenced by human action as well as direct or indirect political interference.

2. DATA AND METHODS

The basic idea underlying our analysis is simple. We decompose the sex difference in life expectancy (SEXDIF) into the three components biological factors (BIO), smoking (SMOKE) and other non-biological factors (ONBF), thus

\[
\text{SEXDIF} = \text{BIO} + \text{SMOKE} + \text{ONBF}.
\]
Our analysis covers 44 populations from Europe and 55 years of observation from 1955 to 2009. The following sections summarise our strategies to estimate the single components and to assess the impacts of smoking and other non-biological factors on the trends and overall extents of the sex gap in life expectancy.

2.1 Sex difference in life expectancy

We used age-specific data on mortality and the population at risk for ages 0, 1-4, 5-9, 10-14, …, 80-84, 85+ from the WHO Mortality Database (WHO 2012) to calculate life tables separately for women and men. We combined the data for single calendar years to five-year periods (1955/59-2005/09) and calculated the sex gap by subtracting the life expectancy (at birth) of men from that of women. Truncated time series were completed by extrapolation based on sex differences in life expectancy derived from data of the 2010 revision of the UN World Population Prospects (United Nations Population Division 2011). Across all 44 European countries and observation periods, the sex difference in life expectancy varies between 1.7 years in TYFR Macedonia in 1955/59 and 13.2 years in Russia in 2000/04.¹

2.2 Impact of biological factors

Genetic and hormonal differences between the sexes constitute the natural basis for higher female life expectancy (Waldron 1983a, Waldron 1985, Hazzard 1986, Kalben 2002). We quantified the pertinent part of the sex difference in life expectancy in the data of a study on female and male Catholic order members (Luy 2003). In order to account for the interaction between biological and non-biological factors (e.g. Retherford 1975, Wingard 1982, Krieger 2003, Rieker and Bird 2005) we estimated the biologically caused difference as a function of the overall sex gap in life

¹ In fact, the highest sex difference in life expectancy was estimated for Bosnia and Herzegovina in the period 1990/94 with 17.6 years caused by the high number of male victims during the Balkan war.
expectancy (see Appendix 1 for more details). The estimates for the impact of biological factors vary between 0.5 and 1.6 years and thus lie within the range of all existing quantifications of the impact of biological factors (Bourgeois-Pichat 1952, Pressat 1973, Waldron 1976, Waldron and Johnston 1976, Trovato and Lalu 1996, Luy 2003).

2.3 Impact of smoking

We used the method proposed by Peto et al. (1992) to estimate smoking-attributable mortality. The required data on the causes of death were taken from the WHO Mortality Database (WHO 2012) for 5-year age groups and single calendar years. The contribution of smoking to the sex difference in life expectancy was estimated by applying the demographic standard method for age- and cause-specific decomposition (Arriaga 1984). Truncated time series were completed with a log-square function in keeping with the smoking epidemic model (Lopez et al. 1994). The lowest estimated impact of smoking on sex differences in life expectancy is 0.02 years for Tajikistan in 1955/59 and the highest is 5.0 years for Russia in 1990/94.

2.4 Impact of non-biological factors

The impact of other non-biological factors was derived from the difference between the estimates for the other two components and the overall sex difference in life expectancy, thus

\[\text{ONBF} = \text{SEXDIFF} - (\text{BIO} + \text{SMOKE}).\]

Note that because the estimated biological impact includes interactions with non-biological factors other than smoking (see Appendix 1), we implicitly assume that the estimates regarding the impact of smoking include the respective interaction effects between smoking and biological factors. The estimated contribution of other non-biological factors varies between 0.3 years in the Netherlands in 1955/59 and 7.1 years in Russia in
2000/04 (excluding the war-related 12.6 years in Bosnia and Herzegovina in 1990/94; see Footnote 1).

2.5 Assessment of impacts

We estimated the impacts of smoking and other non-biological factors for each country and 5-year period. In order to summarise the results for the entire observation time, we assessed these impacts in a simple illustrative manner. We deduced the impacts of the two cause categories on the trend from the ranges of the contributions of smoking and other non-biological factors over the whole period and used the averages of the two components as measure for the impacts on the overall extent of the sex gap. Thus, each population can be assigned to one of four groups of causes of the trend and the extent of the sex differences in life expectancy:

1. The trend and extent of the sex gap were predominantly driven by smoking.
2. The trend was predominantly driven by smoking while the extent was mainly driven by other non-biological factors.
3. The trend was predominantly driven by other non-biological factors while the extent was mainly driven by smoking.
4. The trend and extent of the sex gap were predominantly driven by other non-biological factors.

3. Results

Figure 1 shows the estimated contributions of biological factors, smoking and other non-biological factors to sex differences in life expectancy for the example Belarus. In 1955/59, women outlived men by 9.3 years. The sex gap increased almost continuously until the end of the observation period. This trend was only temporarily interrupted in the second half of the 1980s when sex differences in life expectancy decreased to 8.9 years. In 2005/09, women’s life expectancy exceeded those of men by 11.8 years. The light grey area shows that biological factors played only a
minor role in the causation of the sex gap. The estimated contribution lies between 1.4 and 1.5 years. The estimated impact of smoking increased from 0.8 years in 1955/59 to 4.7 years in 1995/99 and then decreased to 4.0 years in 2005/09. The irregular trend of the sex gap in Belarus is mainly due to the other non-biological factors. Their estimated impact ranges between 3.3 years in 1985/89 and 7.1 years in 1955/59. Table 1 summarizes the estimates for all countries over the entire observation period. In Belarus, women outlived men by 10.1 years on average. This difference was caused to 14.1% by biological factors, 31.6% by smoking and 54.3% by other non-biological factors. The contribution of smoking ranged by 3.9 years of life expectancy, and those of other non-biological factors by 3.8 years. Thus, according to our classification, Belarus belongs to Group 2 with the trend of sex differences in life expectancy being predominantly driven by smoking, while the extent of the sex gap was mainly caused by other non-biological factors.

**Figure 1** Decomposition of sex differences in life expectancy at birth into years caused by biological factors, smoking, and other non-biological factors, Belarus, 1955/59-2005/09.

Source: own calculations with data of WHO (2012) and UN Population Division (2011)
Table 1  Average and range of the contributions of biological factors (BIO), smoking (SMOKE) and other non-biological factors (ONBF) to the sex gap in life expectancy throughout all periods, 44 European countries.

<table>
<thead>
<tr>
<th>Country</th>
<th>Average sex gap in Years</th>
<th>Average contribution in %</th>
<th>Range in years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average</td>
<td>BIO</td>
<td>SMOKE</td>
<td>ONBF</td>
</tr>
<tr>
<td>Group 1: Trend and extent of the sex gap predominantly driven by smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PL Poland</td>
<td>7.7</td>
<td>16.7</td>
<td>43.0</td>
</tr>
<tr>
<td>FI Finland</td>
<td>7.7</td>
<td>16.8</td>
<td>42.7</td>
</tr>
<tr>
<td>SK Slovakia</td>
<td>7.5</td>
<td>17.0</td>
<td>47.9</td>
</tr>
<tr>
<td>HU Hungary</td>
<td>7.0</td>
<td>17.5</td>
<td>44.7</td>
</tr>
<tr>
<td>LU Luxembourg</td>
<td>6.6</td>
<td>18.3</td>
<td>49.9</td>
</tr>
<tr>
<td>CZ Czech Republic</td>
<td>6.6</td>
<td>18.4</td>
<td>52.7</td>
</tr>
<tr>
<td>HR Croatia</td>
<td>6.5</td>
<td>18.3</td>
<td>43.9</td>
</tr>
<tr>
<td>BE Belgium</td>
<td>6.3</td>
<td>18.9</td>
<td>57.1</td>
</tr>
<tr>
<td>DE Germany</td>
<td>6.2</td>
<td>19.0</td>
<td>41.7</td>
</tr>
<tr>
<td>IS Spain</td>
<td>6.1</td>
<td>19.2</td>
<td>41.2</td>
</tr>
<tr>
<td>IT Italy</td>
<td>6.0</td>
<td>19.4</td>
<td>45.1</td>
</tr>
<tr>
<td>SC Scotland</td>
<td>5.8</td>
<td>19.8</td>
<td>50.9</td>
</tr>
<tr>
<td>EW England &amp; Wales</td>
<td>5.6</td>
<td>20.2</td>
<td>51.5</td>
</tr>
<tr>
<td>NI Northern Ireland</td>
<td>5.5</td>
<td>20.3</td>
<td>43.1</td>
</tr>
<tr>
<td>NL The Netherlands</td>
<td>5.5</td>
<td>20.2</td>
<td>62.5</td>
</tr>
<tr>
<td>GR Greece</td>
<td>4.5</td>
<td>22.6</td>
<td>50.4</td>
</tr>
<tr>
<td>MK TFYR Macedonia</td>
<td>4.0</td>
<td>23.6</td>
<td>46.0</td>
</tr>
</tbody>
</table>

Group 2: Trend predominantly driven by smoking, extent driven by other non-biological factors

<table>
<thead>
<tr>
<th>Country</th>
<th>Average sex gap in Years</th>
<th>Average contribution in %</th>
<th>Range in years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average</td>
<td>BIO</td>
<td>SMOKE</td>
<td>ONBF</td>
</tr>
<tr>
<td>BY Belarus</td>
<td>10.1</td>
<td>14.1</td>
<td>31.6</td>
</tr>
<tr>
<td>BE Estonia</td>
<td>9.7</td>
<td>14.4</td>
<td>35.0</td>
</tr>
<tr>
<td>LV Latvia</td>
<td>9.7</td>
<td>14.5</td>
<td>35.0</td>
</tr>
<tr>
<td>LT Lithuania</td>
<td>9.5</td>
<td>14.6</td>
<td>36.2</td>
</tr>
<tr>
<td>UA Ukraine</td>
<td>9.1</td>
<td>15.0</td>
<td>34.9</td>
</tr>
<tr>
<td>FR France</td>
<td>7.6</td>
<td>16.8</td>
<td>36.3</td>
</tr>
<tr>
<td>SI Slovenia</td>
<td>6.9</td>
<td>17.8</td>
<td>38.1</td>
</tr>
<tr>
<td>PT Portugal</td>
<td>6.6</td>
<td>18.4</td>
<td>20.8</td>
</tr>
<tr>
<td>AT Austria</td>
<td>6.5</td>
<td>18.5</td>
<td>38.0</td>
</tr>
<tr>
<td>CH Switzerland</td>
<td>6.1</td>
<td>19.2</td>
<td>39.5</td>
</tr>
<tr>
<td>NO Norway</td>
<td>5.7</td>
<td>19.8</td>
<td>22.1</td>
</tr>
<tr>
<td>DK Denmark</td>
<td>5.0</td>
<td>21.3</td>
<td>36.4</td>
</tr>
</tbody>
</table>
Table 1 cont.

<table>
<thead>
<tr>
<th>Country</th>
<th>Average sex gap in Years</th>
<th>Average contribution in %</th>
<th>Range in years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BIO</td>
<td>SMOKE</td>
<td>ONBF</td>
</tr>
<tr>
<td>Group 3: Trend predominantly driven by other non-biological factors, extent driven by smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BA Bosnia and Herzegovina</td>
<td>5.8</td>
<td>18.7</td>
<td>43.1</td>
</tr>
<tr>
<td>RO Romania</td>
<td>5.6</td>
<td>19.8</td>
<td>44.9</td>
</tr>
<tr>
<td>BG Bulgaria</td>
<td>5.6</td>
<td>20.0</td>
<td>43.9</td>
</tr>
<tr>
<td>MT Malta</td>
<td>4.4</td>
<td>22.9</td>
<td>54.5</td>
</tr>
<tr>
<td>Group 4: Trend and extent of the sex gap predominantly driven by other non-biological factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RU Russia</td>
<td>10.8</td>
<td>13.5</td>
<td>37.8</td>
</tr>
<tr>
<td>KZ Kazakhstan</td>
<td>10.4</td>
<td>13.8</td>
<td>35.6</td>
</tr>
<tr>
<td>KG Kyrgyzstan</td>
<td>8.6</td>
<td>15.7</td>
<td>33.4</td>
</tr>
<tr>
<td>GE Georgia</td>
<td>7.9</td>
<td>16.6</td>
<td>23.6</td>
</tr>
<tr>
<td>MD Republic of Moldova</td>
<td>7.4</td>
<td>17.1</td>
<td>29.9</td>
</tr>
<tr>
<td>TM Turkmenistan</td>
<td>6.8</td>
<td>18.0</td>
<td>18.4</td>
</tr>
<tr>
<td>UZ Uzbekistan</td>
<td>6.3</td>
<td>18.8</td>
<td>21.5</td>
</tr>
<tr>
<td>SE Sweden</td>
<td>5.0</td>
<td>21.2</td>
<td>18.4</td>
</tr>
<tr>
<td>IS Iceland</td>
<td>5.0</td>
<td>21.4</td>
<td>8.9</td>
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<tr>
<td>IE Ireland</td>
<td>4.9</td>
<td>21.5</td>
<td>37.2</td>
</tr>
<tr>
<td>TJ Tajikistan</td>
<td>4.7</td>
<td>22.1</td>
<td>12.5</td>
</tr>
</tbody>
</table>

Notes:

Sex differences in life expectancy are extrapolated on the basis of UN data and smoking-attributable mortality is estimated by log-square function for the following countries and periods:  


Data for 2, 4 based on former Czechoslovakia before 1990; 5, 12, 19, 23 based on former Yugoslavia before 1990; 7 based on West Germany before 1990;


Source: own calculations with data of WHO (2012) and UN Population Division (2011)
Our analysis revealed that all four combinations of the causes underlying the trends and extents of the sex gap in life expectancy (Groups 1-4) can be found in the 44 studied populations. Figures 2 and 3 illustrate these four basic patterns represented by the Netherlands, France, Sweden and Bulgaria. The x-axes of the graphs show the estimated contribution of smoking measured in years of life expectancy, while the y-axes show the estimated contribution of other non-biological factors. Each circle represents the estimate for a specific 5-year period. The arrows between the circles illustrate the trend over time. The estimates for the earliest (1955/59) and latest (2005/09) periods are highlighted in black and white, respectively. The dotted diagonal lines (from top left to bottom right) show the total sex difference in life expectancy (with values given at one end of these lines). The solid diagonal line (from bottom left to top right) divides the estimates into those indicating a higher impact of smoking as compared to other non-biological factors (area below the diagonal line) and vice versa (area above the diagonal line).

For instance, the black circle in the time series for the Netherlands (Figure 2) shows that the sex difference in life expectancy was 3.4 years in the period 1955/59 (see position between the dotted lines for 3 and 4 years). Smoking caused 2.2 years of this difference (position of the black circle on the x-axis), while the other non-biological factors contributed 0.3 years of life expectancy surplus for women. The estimated impact of biological factors is not illustrated in this graph. It is, however, included as background information as it is the difference between the total sex difference in life expectancy and the estimates for the contributions of smoking and other non-biological factors. Thus, in the case of the Netherlands, biological factors are estimated to contribute 0.9 years of life expectancy difference between the sexes in the period 1955/59. The time series for the Netherlands exemplifies the trend of changing sex differences in life expectancy, which rose from 3.4 years in 1955/59 to a maximum of 6.9 years in 1980/84 and then continuously declined to 4.3 years in 2005/09. The fact that all circles are located in the bottom right triangle of the graph indicates that the impact of
smoking was higher than the impact of other non-biological factors throughout the entire observation period. Moreover, as the increase of the sex gap in life expectancy until the early 1980s and the subsequent decrease develop predominantly along the x-axis we can conclude that the changes in the trend were, as well, primarily caused by smoking and only to a minor extent by other non-biological factors. However, from the 1980s onwards, the contribution of smoking decreased to a lower level in 2005/09 as compared to the initial period (1955/59), while the contribution of other non-biological factors increased slowly but continuously from 0.3 to 1.6 years.

**Figure 2** Trends of the impacts of smoking and other non-biological factors on sex differences in life expectancy at birth, France and the Netherlands, 1955/59-2005/09.

Source: own calculations with data of WHO (2012) and UN Population Division (2011)
Figure 2 shows the patterns for trends in the sex gap that are predominantly driven by smoking. The Netherlands is an example for populations in which smoking contributed also strongest to the overall sex difference in life expectancy (Group 1). Over the entire observation period, 62.5% of the difference in life expectancy between Dutch women and men were caused by smoking and only 17.2% by other non-biological factors. The ranges of smoking and other non-biological factors were 3.0 years and 1.3 years, respectively (see Table 1). France is an example for populations in which the trend was also predominantly caused by smoking, while the overall difference between the sexes was mainly due to other non-biological factors (Group 2). Throughout the entire observation period, 36.3% of the sex gap were caused by smoking and 46.9% by other non-biological factors; the ranges were 2.0 years for smoking and 0.7 years for other non-biological factors.

Figure 3 shows the patterns for trends in the sex gap that are predominantly driven by other non-biological factors. Bulgaria is typical for populations in which the trend was mainly caused by other non-biological factors, while smoking was largely responsible for the overall sex difference (Group 3). Throughout the entire observation period, 43.9% of the sex gap were caused by smoking and 36.1% by other non-biological factors; the ranges were 1.2 years for smoking and 2.1 years for other non-biological factors (see Table 1). Sweden is an example for populations in which other non-biological factors were predominantly responsible for both the trend and the extent of the sex gap (Group 4). Only 18.4% of the difference in the life expectancy of Swedish women and men were attributed to smoking, while 60.4% were due to other non-biological factors. The ranges of smoking and other non-biological factors were 1.2 and 1.8 years, respectively.

The figures for all studied countries are contained in Appendix 2 and demonstrate the very different trends in the sex gap as well as the differences in the impacts of smoking and other non-biological factors. Figure 4 depicts this great heterogeneity by showing the average contributions of smoking and other non-biological factors across all 44 countries for the entire
Figure 3  Trends of the impacts of smoking and other non-biological factors on sex differences in life expectancy at birth, Sweden and Bulgaria, 1955/59-2005/09.

source: own calculations with data of WHO (2012) and UN Population Division (2011)

observation period. Populations, in which the average impact of smoking was larger than the impact of other non-biological factors are located in the bottom right triangle, while countries characterised by a reversed importance of impacts are located in the upper left triangle of the figure. In addition, the graph shows whether the ranges of smoking (black circles) or other non-biological factors (grey triangles) were higher during the observation period of 55 years. The distribution of circles and triangles in the areas between the diagonal lines indicate the variety of causes that yield similar average sex gaps in life expectancy. For instance, Uzbekistan (UZ) and Belgium (BE) have almost the same average sex difference in life expectancy during the 55
Figure 4  Average impacts of smoking and other non-biological factors on sex differences in life expectancy at birth, 44 European countries, 1955/59-2005/09.

Source: own calculations with data of WHO (2012) and UN Population Division (2011)

years of the observation period (i.e. around 6.3 years). However, in Belgium 3.6 and 1.5 years of this difference can be attributed to smoking and other non-biological factors, respectively, while the corresponding figures for Uzbekistan are 1.4 and 3.7 years.

All in all, most of the European populations belong to Group 1, i.e. both the trend and the extent of the sex gap were predominantly driven by smoking (17 countries). In 11 populations the opposite held true, i.e. both the trend and the extent were predominantly driven by other non-biological factors (Group 4). In another 12 populations, the trend of the sex gap was
driven predominantly by smoking, while the extent was mainly due to other non-biological factors (Group 2). In as few as four populations from Central and Eastern Europe (Bosnia and Herzegovina, Bulgaria, Malta and Romania), the development of the sex gap was predominantly driven by other non-biological factors, while smoking was responsible for a larger part of the extent (Group 3).

4. DISCUSSION

This study proposes a novel approach to the investigation of the impact of smoking on sex differences in life expectancy. While most of the earlier studies compare smoking-attributable mortality to the overall rest category, we isolated those causes that can be influenced by direct or indirect human interference from biological factors. We performed a systematic literature review in order to find similar approaches to estimate the impact of smoking on sex differences in mortality in comparison to other non-biological factors. The search was carried out in PUBMED without any time limitations by browsing titles and abstracts for the broad terms “sex” OR “gender” AND “life expectancy” OR “mortality” OR “survival” AND “difference” OR “differential” OR “gap” AND “smoking” OR “tobacco”. The PUBMED search yielded 616 studies, which were independently screened by both authors. All 60 articles identified as potentially relevant by at least one author were analysed in detail. We found 27 studies containing numerical estimates for the impact of smoking on sex differences in mortality. All of them were within the range outlined in the introduction. We also did an additional search of our own literature database, which includes more than 8,000 mainly demographic and epidemiologic publications with a special focus on health and mortality. In total, we found only four papers which directly compare the impact of smoking on sex differences in mortality to another specific cause, namely alcohol-related mortality (Nizard and Muñoz-Pérez 1994, Martelin et al. 2004, Wong et al. 2006, McCartney et al. 2011). However, we found no study which controlled for biological sex
differences when estimating the impact of smoking on overall mortality differences between women and men or comparing the effects of tobacco consumption to other specific risk factors.

It is important to note, however, that our study has two major limitations. First, the very simple and broadly defined groups of causes responsible for the sex gap comprise populations which are characterised by significantly different patterns of impacts and trends of smoking and other non-biological factors. On the one hand, for instance, both Italy and Scotland belong to Group 1 although the trends in these countries developed very differently, with Scotland ending up close to the point at which Italy started, reflecting the different times in which these populations went through the stages of the smoking epidemic (see Appendix 2). Sweden and Russia are another example. Both belong to Group 4 because non-biological factors other than smoking have a stronger impact on the trend and overall extent of the sex gap in life expectancy. However, all elements of our analysis for these two populations differ strongly in their extents. On the other hand, populations with apparently similar trends (such as Sweden and Norway) are assigned to different groups because of our classification criteria.

The second limitation is that our estimates of the impacts of biological factors and smoking are based on specific assumptions which include several uncertainties. The impact of biological factors is, however, low and the estimates obtained with different approaches yield very similar results (see Appendix 1). The Peto-Lopez method we used for estimating smoking-attributable mortality is the standard technique for this purpose. Comparisons with alternative approaches show that the inherent insecurities and obtained results do not differ significantly (Brønnum-Hansen and Juel 2000, Pérez-Ríos and Montes 2008, Preston et al. 2009, Rostron 2010, Oza et al. 2011, Murphy and Di Cesare 2012). Note, however, that we assumed that both estimation procedures—i.e. those for the impacts of biological factors and smoking—can be applied to all studied populations. While this seems to be a logical assumption with regard to the quantification of biological factors, it might not hold true for the Peto-Lopez method for the
assessment of the impact of smoking (see also Valkonen and van Poppel 1997). Completing missing information on trends in the sex gap in life expectancy and the impact of smoking by way of extrapolation based on available data adds further uncertainties, which should, however, be minor as these trends tend to be regular in populations with complete time series.

With these limitations in mind, the central research questions addressed in this paper can be answered by two basic conclusions. First of all, the trend of sex differences in life expectancy can indeed be attributed to smoking in most populations of Western Europe. The only exceptions are Sweden, Iceland and Ireland whose populations are characterised by a low prevalence of smoking (Forey et al. 2002, Peto et al. 2006). Populations in which the trend of the sex gap is predominantly driven by other non-biological factors are those from Eastern Europe, where alcohol-related and external causes of death in mid-adult ages are known to lead to an extraordinarily high mortality among men (Bobadilla et al. 1997, Vallin and Meslé 2001, Meslé 2004, Luy et al. 2011). Secondly, with regard to the overall extent of male excess mortality, smoking is the main driver only in 21 of the studied populations, whereas other non-biological factors are the dominating cause in the other 23 countries.

A deeper analysis of the country-specific trends (Appendix 2) reveals some additional, interesting details. The contribution of smoking to the sex gap declines in all studied populations, although the beginning of the decline varies in accordance with the model of the smoking epidemic. However, in most populations, the contribution of other non-biological factors—including alcohol consumption, nutrition habits, specific infectious diseases, external mortality (accidents, poisoning, homicides) and health risks related to occupation—is higher at the end than at the beginning of the observation period with France being one of the few exceptions (see Fig. 3). Nevertheless, the patterns vary widely. In some populations (e.g. Norway and Portugal), the impact of non-biological factors other than smoking first rose and then dropped. In other countries (e.g. Luxembourg, Poland or the Republic of Moldova), the opposite holds true (initially low, then higher) or
the impact continuously increases (e.g. in the UK, the Netherlands, Finland and many Central and Eastern European countries). The successor states of the former Soviet Union follow a rollercoaster pattern in accordance with the well-documented trends in alcohol-related mortality (Leon et al. 1997, Shkolnikov and Cornia 2000, Murphy 2011, Shkolnikov et al. 2013). In yet another group of populations (e.g. Italy and Greece), the impact of these factors remains almost unchanged throughout the observation time. Further research is needed to better understand the role of specific non-biological factors other than smoking that cause these different trends, albeit it is likely that their impact is as heterogeneous and context-dependent as that of smoking.

Summing up, this study highlights the enormous heterogeneity in impacts of smoking and other non-biological causes on sex differences in life expectancy among the populations of Europe. Hence, over-generalised statements which might suggest that smoking is the main force behind the sex gap in all developed populations are misleading. Our study shows that the public health sector rather needs population-specific estimates to introduce the most appropriate measures in order to further reduce the inequalities in life expectancy between women and men in the most effective way. In this context it is important to note that the overall decrease of the impact of smoking causes on the one hand a decrease of the sex gap but on the other comes along with an increase or stall of the impact of other non-biological factors. Thus, the results of this study demonstrate that, regardless of the prevailing effect of smoking, many populations have still remarkable potentials to further narrow their sex gaps in life expectancy.

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References


APPENDIX 1: Estimation of the impact of biological factors on sex differences in life expectancy

There is consensus that biological factors—defined as those components of mortality that are neither due to individual behaviours nor to social or environmental influences—have been responsible for only a minor fraction of the sex gap in life expectancy in the post-war populations of developed countries (Enterline 1961, Kitagawa 1977, Lopez 1984, Hart 1989, Büttner 1995, Kraemer 2000, Manton 2000, Lemaire 2002, Planchard et al. 2009). However, it is difficult to quantify the exact degree of their influence because it is impossible to carry out pertinent experiments in human beings and research is therefore limited to what can be observed (Kalben 2002, Austad 2006).

Bourgeois-Pichat (1952) was the first who tried to assess the biologically caused difference in life expectancy between the sexes in the early 1950s. Based on an analysis of deaths due to causes that were considered unavoidable at that time, he found a natural difference of 1.9 years of life expectancy at birth. Around two decades later, Pressat (1973) presented an almost identical estimate by stating that biological factors are responsible for approximately two years higher life expectancy of women. This assessment was based on observations of pre-industrial populations and males’ 25-30% higher mortality in the first year of life in the post-industrial era; both identified by Pressat as being primarily biologically driven. Studying similar records of mortality conditions during the early stages of the epidemiologic transition and mortality levels reflected in model life tables, Trovato and Lalu (1996) surmised that biological factors cause women’s life expectancy to be in general 1-2 years, but under no conditions more than 2-3 years, higher than that of men. Waldron (1976) and Waldron and Johnston (1976) did an in-depth analysis of causes-of-death data for the US population in 1967 to identify the main causes of the mortality differences between women and men. They attribute about 70% of the overall sex differential to specific social and behavioural causes and estimate
that genetic factors are responsible for 5% of the difference. The remaining 25% were not assigned to specific risk factors, but the authors note the contribution of the possibly protective role of female hormones. By analysing the mortality of around 12,000 members of female and male Catholic orders in Germany between 1890 and 1995, Luy (2003) estimated that biological factors cause a maximum sex difference of 2 years in life expectancy at young adult ages. DesMeules and colleagues used a cause-of-death analysis to show that the difference in life expectancy at birth between women and men in Canada in the years 1997-1999 dropped from 5.5 to 2.2 years when controlling for smoking-related and other external causes, which made them conclude that “women do not appear to have as large a biological survival advantage” (DesMeules et al. 2004: 5).

All these estimates are definitely imprecise because environmental effects can never be completely isolated (Waldron 1983b, Nobile 2007) and each estimate is basically only valid for the specific conditions studied (Waldron 1995, Rogers et al. 2000). Nonetheless, they all agree on the modest size of the naturally caused sex gap with only minor deviations in the estimated extent. Interestingly, all these estimates are similar to results obtained in twin studies, which attribute approximately 25% of the general (not sex-specific) variation in both health (Christensen et al. 1999) and mortality (Christensen and Herskind 2007) to genetic factors.

We assessed the impact of biological factors on sex differences in life expectancy on the basis of the data obtained in our study on female and male Catholic order members in Germany. Although it is known that cloistered life entails specific risk factors which influence women’s and men’s mortality differently (among them the high tuberculosis mortality in nuns in the first half of the 20th century and the impact of smoking on the mortality of monks since the 1970s), it seems that nuns and monks are an almost ideal experimental setting for this research question (see also Madigan and Vance 1957, Hansson et al. 1997). Above all, this holds true for the period 1960-1970 for the following three reasons: (1) the tuberculosis threat among nuns—mainly caused by their nursing activity—ceased to exist
as bias for female order members’ mortality, (2) smoking could not yet have caused high mortality in male religious communities where cigarette smoking only became common after World War II (note that even nowadays smoking is not tolerated in female communities), and (3) during the first half of the 20th century, female and male religious communities were affected by specific circumstances—high tuberculosis prevalence for nuns and two world wars for monks—which increased the mortality among their young members and led to similar selected populations in the post-war periods. In addition, our concept of decomposing sex differences in life expectancy into biological factors, smoking and other non-biological factors requires an estimate of the biological impact that includes interactions between biological and non-biological factors other than smoking. An analysis of external cause mortality among order members suggests that such interactions of biological and non-biological factors are, in fact, inherent when using the differences between nuns and monks in this context (Luy 2009). Moreover, the data permit estimates of the naturally caused differences as a function of the overall sex gap in life expectancy. As biological factors interact with non-biological drivers of mortality—including developments in medical technology and treatments—it seems plausible to assume that their contribution is not stable but rather varies with the respective social and disease environments (Johansson 1991).

Figure 5 shows the estimate based on the order members’ life tables for the periods around the years 1960, 1965 and 1970 (Luy 2003). We compared the difference in life expectancy between nuns and monks for all available age groups with the respective differences prevailing in the total West German population. The data suggest a logarithmic relationship between the impact of biological factors (estimated on the basis the difference in life expectancy between nuns and monks) and the extent of the sex differences in life expectancy (indicated by the respective values for the total population). Note that this function is based on mortality experiences at adult ages only. Nevertheless, using it to quantify the impact of biological factors on sex differences in life expectancy at birth seems justified as it was
recently reported that sex differences in infant mortality are also not primarily caused by biological factors (Pongou 2013).

The dashed line in Figure 5 indicates one quarter of the sex difference in life expectancy of the total population. This corresponds to the estimated 25% for the biological impact identified in some other studies. It becomes apparent that our estimated logarithmic function and the constant 25% line are very close for sex differences in life expectancy between 1.0 and 6.0 years, i.e. the range prevailing in most industrialised populations since World War II. However, the functions yield different estimates for the biological impact in populations with higher sex differences as they are found in Russia and other Eastern European countries (see Table 1).

**Figure 5** Estimated contribution of biological factors to the sex gap in life expectancy.

Note: Estimates refer to life expectancy between ages 25 and 90 from three calendar year periods around the years 1960, 1965 and 1970.

Source: Own calculations with data of Luy 2003 (extended by one male community).
APPENDIX 2: Trends in the sex gap in life expectancy at birth decomposed into smoking, other non-biological factors and biological factors between 1955/59-2005/09, 44 European countries, sorted by cause groups and extent of the sex gap

Group 1: Trend and extent of the sex gap predominantly driven by smoking
Group 2: Trend predominantly driven by smoking, extent driven by other non-biological factors
Latvia (LV)  Lithuania (LT)  Other non-biological factors

Ukraine (UA)  France (FR)
Group 3: Trend predominantly driven by other non-biological factors, extent driven by smoking
Group 4: Trend and extent of the sex gap predominantly driven by other non-biological factors

Russian Federation (RU) 
Kazakhstan (KZ)

Kyrgyzstan (KG) 
Georgia (GE)
Republic of Moldova (MD) Turkmenistan (TM) Uzbekistan (UZ) Sweden (SE)

Smoking

Other non-biological factors

Uzbekistan (UZ) Sweden (SE)

Smoking

Other non-biological factors