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The impact of smoking on gender differences in life expectancy: more heterogeneous than often stated

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Background: Throughout industrialized countries, tobacco consumption is seen as the predominant driver of both the trend and the extent of gender differences in life expectancy. However, several factors raise doubts to this generalization. We hypothesize that the impact of smoking on the gender gap is context-specific and differs between populations. **Methods:** We decompose the gender differences in life expectancy into fractions caused by smoking and other non-biological factors for 53 industrialized countries and the period 1955–2009 to assess the significance of smoking among the causes that can be influenced by direct or indirect interference. **Results:** The trend of the gender gap can indeed be attributed to smoking in most populations of the western world. However, with regard to the overall extent of male excess mortality, smoking is the main driver only in the minority of the studied populations. While the impact of smoking to gender differences in life expectancy declines in all populations, the contribution of other non-biological factors is in most cases higher at the end than at the beginning of the observation period. **Conclusions:** Over-generalized statements suggesting that smoking is the main driver of the gender gap in all populations can be misleading. The results of this study demonstrate that—regardless of the prevailing effect of smoking—many populations have still remarkable potentials to further narrow their gender gaps in life expectancy. Although measures to further reduce the prevalence of tobacco consumption must be continued, more attention should be directed to the growing importance of other non-biological factors.

Introduction

Throughout the developed world, women live several years longer than men. Yet, the gender gap shows huge variations and underwent significant changes during the 20th century.¹ Gender differences (GD) in life expectancy (LE) remained more or less constant until the 1940s and started to increase thereafter. This trend came to a halt in most western populations at the beginning of the 1980s, when the gap started to decrease immediately or after a stalling 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. Nonetheless, GD in LE are still striking in all developed populations. They currently range from around 4 years in Iceland and Israel to more than 12 years in Russia.

As all industrialized societies continue to age and the shares of older people grow, women's higher longevity and its causes have become an ever more important topic in public health and social policy.² Ever since the 1970s, researchers have identified cigarette consumption as the most important reason for GD in mortality (e.g. Refs. 3–5), usually in direct relation to the so-called 'smoking epidemic model'⁶—a perspective that prevails until today (e.g. Ref. 7).

However, several aspects raise doubts about the general validity of this viewpoint. It is well known that GD in LE are caused by a complex combination of biological (genetic, hormonal) and non-biological (behavioural, economic, social, environmental and cultural) factors.^{8,9} Within this network of causes, tobacco consumption is just one of many non-biological risk factors, which has an impact only from the mid-adult ages onwards. Moreover, populations differ in the smoking prevalence of women and men

as well as the time and speed they pass through the smoking epidemic.¹⁰ Although it appears plausible that trends in cigarette consumption have played a decisive role in the observed changes in GD in LE since the middle of the 20th century, it seems rather unlikely that smoking is also responsible for most of the overall male excess in mortality in each population of the developed world. Finally, a general assessment is impeded by the fact that available studies not only differ with regard to the investigated populations, age groups and calendar years, but also the methods used for quantifying overall mortality and mortality attributable to smoking (e.g. Refs. 4, 5, 9, 11–13). All in all, the contribution of smoking to the extent and the trend in mortality differences between women and men is not as clear as often stated.

We hypothesize that the impact of smoking on GD in LE is highly context-specific and differs between populations. To test this hypothesis, we estimate the impact of smoking on GD in LE from the middle of the 20th century until today for a large number of populations using the same data and identical well-established methods. Moreover, we compare the impact of smoking to the combined effect of all other non-biological factors. This approach is important for public health policies because it enables us to assess the significance of tobacco consumption among the causes leading to GD in LE that can be influenced by human action as well as direct or indirect political interference.

Methods

We decompose GD in LE at birth into three components: biological factors, smoking, and other non-biological factors. The analysis

covers 53 populations from developed countries and 55 years of observation from 1955 to 2009. GD in LE are derived from period life tables using age- and gender-specific data on mortality and the population at risk for ages 0, 1–4, 5–9, 10–14, . . . , 80–84, 85+ from the Mortality Database of the World Health Organization.¹⁴ We summarize the data for single calendar years into 5-year periods and calculate the gender gap by subtracting the LE of men from that of women. Truncated time series are completed by extrapolation based on GD in LE derived from data of the 2010 revision of the United Nations World Population Prospects.¹⁵ Among the populations included to this study, GD in LE range between 1.7 years in TYFR Macedonia in 1955/59 and 17.6 years in Bosnia and Herzegovina in 1990/94.

Biological factors—defined as those components of mortality that are neither due to individual behaviours nor to social or environmental influences—constitute the natural basis for higher female LE.^{16,17} However, corresponding quantifications are rare and we found only five such attempts which are based on very different approaches.^{18–22} They all agree on the modest size of the naturally caused GD in LE with only minor deviations in the estimated extent (up to a maximum of 30% or 2 years of LE). We follow the approach suggested by Luy¹⁹ and quantify the pertinent part of the gender gap from data of his study on female and male Catholic order members as a function of the overall GD in LE (see Ref. 23 for more details). The corresponding estimates range between 0.5 and 1.6 years and thus lie within the range of the above mentioned quantifications of the impact of biological factors.

Smoking-attributable mortality is estimated with the Peto–Lopez method.²⁴ The required data on the causes of death are taken from the WHO Mortality Database.¹⁴ The contribution of smoking to GD in LE is estimated with the demographic standard tool for age- and cause-specific decomposition.²⁵ Truncated time series are completed with a log-square function in keeping with the smoking epidemic model. Across the analysed populations the estimated impact of smoking on GD in LE ranges between 0.02 years in Tajikistan in 1955/59 and 5.0 years in Russia in 1990/94.

Finally, the impact of other non-biological factors is derived from the difference between the overall GD in LE and the sum of the estimates for smoking and biological factors. The estimated contribution of other non-biological factors to the gender gap ranges from 0.3 years in the Netherlands in 1955/59 to 12.6 years in Bosnia and Herzegovina in 1990/94. The numerical estimates for each country and period can be found in Online Supplementary material S1.

Results

The decomposition of GD in LE into the effects of smoking and other non-biological factors leads to four possible basic patterns during the observation period:

- Trend and the extent of GD in LE were predominantly driven by smoking.
- Trend of GD in LE was predominantly driven by smoking, whereas the extent was mainly driven by other non-biological factors.
- Trend of GD in LE was predominantly driven by other non-biological factors, whereas the extent was mainly driven by smoking.
- Trend and the extent of GD in LE were predominantly driven by other non-biological factors.

Our analysis reveals that all four combinations of the causes underlying the trends and extents of GD in LE can be found in the 53 studied populations. Figure 1 illustrates 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 LE, whereas 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 show the total GD in LE (with values given at one end of these lines). The solid diagonal line divides the estimates into those indicating a higher impact of smoking as compared with other non-biological factors (area below the diagonal line) and vice versa.

For instance, the black circle in the time series for the Netherlands (figure 1a) shows that the GD in LE was 3.4 years in 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 LE 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 GD in LE 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 difference in LE between women and men in 1955/59.

The time series for the Netherlands exemplifies the trend of changing GD in LE, which rose from 3.4 years in 1955/59 to a maximum of 6.9 years in 1980/84 and then continuously declined

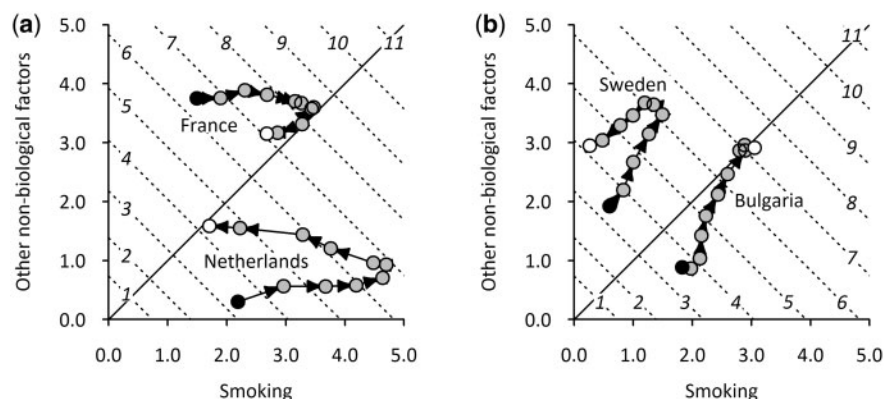


Figure 1 Trends of the impacts of smoking and other non-biological factors on gender differences (GD) in life expectancy at birth, selected countries, 1955/59–2005/09. (a) Trends in GD predominantly driven by smoking. (b) Trends in GD predominantly driven by other non-biological factors

Source: Own calculations with data of WHO¹⁴ and UN Population Division.¹⁵

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 that of other non-biological factors throughout the entire observation period. Moreover, as the increase of the gender gap in LE 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 with the initial period (1955/59), whereas the contribution of other non-biological factors increased slowly but steadily from 0.3 to 1.6 years.

Figure 1a shows the patterns for trends in the gender gap that are predominantly driven by smoking. The Netherlands is an example for populations in which smoking contributed also stronger to the overall GD in LE (pattern A). France is an example for populations in which the trend was also predominantly caused by smoking, while the overall GD was mainly due to other non-biological factors (pattern B).

Figure 1b shows the patterns for trends in the gender 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 GD (pattern C). Finally, Sweden is an example for populations in which other non-biological factors were predominantly responsible for both the trend and the extent of GD in LE (pattern D).

The figures for all countries are contained in Online Supplementary material S1 and demonstrate the very different trends in the gender gap as well as the differences in the impacts of smoking and other non-biological factors. Figure 2 illustrates this heterogeneity by arranging the 53 populations according to the four basic patterns of causes responsible for GD in LE. To summarize the impact of smoking and other non-biological factors on the trend of the gender gap, we use the range of their contributions

over the whole period. The impact of the two components on the overall extent of GD in LE is measured by their average contributions (the corresponding data and a graphical illustration can be found in Online Supplementary material S2). All in all, we find similar numbers of populations in which both the trend and the extent of GD in LE were predominantly driven by smoking and populations in which the opposite held true, i.e. where both the trend and the extent were predominantly driven by other non-biological factors (17 and 15 populations, respectively). In another 17 populations, the trend of the gender gap was driven predominantly by smoking, while the extent was mainly due to other non-biological factors. Finally, in four populations from Central and Eastern Europe, the development of GD in LE was predominantly driven by other non-biological factors, while smoking was responsible for a larger part of the extent.

Discussion

The innovative approach presented in this article permits a direct comparison of the roles played by smoking and other non-biological factors in trends of GD in LE since the mid-20th century. For this reason, the results of our study are not only interesting from the demographic and epidemiologic viewpoints but are also highly relevant for public health policies. Yet, it is important to note that this study has a number of limitations. First of all, the decomposition of GD in LE into the effects of biological factors, smoking and other non-biological factors can only be approximate because it neglects the manifold interactions between biological and non-biological factors. For instance, smoking is raising mortality from coronary heart disease against which female hormones provide some protective effect.²⁶ In addition, sex hormones as well as sex differences in central cholesterol, serotonin levels and reproductive function are shaping gendered health behaviours including tobacco consumption.²⁷ According to our approach to estimate the impact of biological factors, these interaction effects appear to be included in the estimated smoking-attributable mortality, but to

		Extent of the gender gap predominantly driven by			
		Smoking		Other non-biological factors	
Trend of the gender gap predominantly driven by	Smoking	Belgium Croatia Czech Republic Slovakia Finland Germany Greece Hungary Italy	Luxembourg TFYR Macedonia Netherlands Poland Spain England & Wales Northern Ireland Scotland	Austria Belarus Denmark Estonia France Latvia Lithuania Norway Portugal	Slovenia Switzerland Ukraine Canada USA Japan Australia New Zealand
	Other non-biological factors	Bosnia & Herzegovina Bulgaria Malta Romania		Georgia Iceland Ireland Kazakhstan Kyrgyzstan Republic of Moldova Russia Sweden	Tajikistan Turkmenistan Uzbekistan Chile Mexico Israel South Korea

Figure 2 Countries by impact of smoking and other non-biological factors on overall gender differences in life expectancy at birth and on the trend of the gender gap during the period 1955-2009

Note: The impacts of smoking and other non-biological factors on the trend of the gender gap are assessed by the range of the contributions over the whole observation period and the impacts of smoking and other non-biological factors on the overall extent of the gender gap are assessed by the average contributions; the corresponding data can be found in Online Supplementary material S2

be excluded from the mortality due to other non-biological factors (see Ref. 23 for more details).

The estimates of the impacts of smoking and biological factors are based on specific assumptions which include further uncertainties. Nonetheless, comparisons of alternative approaches for estimating smoking-attributable mortality show that the inherent insecurities and obtained results do not differ significantly.^{28–30} Also the impact of the applied estimation strategy for biological factors is low as the estimates obtained with different approaches yield very similar results. However, we assumed that both estimation procedures—i.e. those for the impacts of smoking and biological factors—can be applied to all studied populations. Although this seems to be a valid 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 Ref. 11).

Another limitation concerns the numerical indicators used in figure 2 to assign the populations to specific causation patterns of GD in LE. The averages and ranges of smoking and other non-biological factors reflect solely the conditions and trends within the observed time span from 1955 to 2009. The boundaries of this period do not correspond the beginning and the end of the overall trends in GD in LE and their causes, and they might include even different stages of the analysed trends. For instance, the range of smoking observed in the Netherlands describes the decline in the impact of tobacco consumption since 1980, but not the increase which started well before 1955. Hence, it is imperative to note that the differentiation between the four basic patterns serves exclusively the purpose to summarize the heterogeneity of impacts and trends of smoking and other non-biological factors in a simple and illustrative manner. The populations characterized by one of these patterns cannot be seen as a homogeneous group for which specific conclusions about the nature of GD in LE can be derived. For instance, the gender gaps prevailing in Italy and Scotland have been identified to be characterized by pattern A, although the trends developed very differently, with Scotland ending up close to the point at which Italy started (see Online Supplementary material S1). Likewise, populations with apparently similar trends are assigned to different patterns because of our classification criteria, such as Sweden and Norway.

With these limitations in mind, we can conclude that our hypothesis about the role of smoking in the causation of GD in LE is confirmed regarding to the extent of the gender gap. But we have to reject it with regard to the trend during the observation period. The latter can indeed be attributed to smoking in most populations of the western world, including Western Europe, North America, Japan, Australia and New Zealand. The only exceptions are Sweden, Iceland, Ireland and Israel whose populations are characterized by a low prevalence of smoking.³¹ Populations in which the trend of the gender 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,³² and from Latin America and South Korea, which are still in the earliest stages of the tobacco epidemic with a low level of smoking-attributable mortality.³³

With regard to the overall extent of the gender gap we found that smoking is the main driver only in a minority of the studied populations. Interestingly, these are exclusively located in Europe. This is mainly due to the fact that the contribution of non-biological factors other than smoking was considerably lower in European populations during the observation period. A second reason is that most European populations are already in the late stages of the smoking epidemic. Consequently, the analysed time span includes the years of maximum GD in smoking-attributable mortality for these populations, what is not the case for several non-European countries, such as Chile, Mexico and South Korea.

A deeper analysis of the country-specific trends reveals some additional, interesting details. The contribution of smoking to the gender 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, physical exercise, 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. Nevertheless, the patterns vary widely. In some populations (e.g. Norway, Portugal, Mexico and South Korea), the impact of non-biological factors other than smoking first rose and then dropped. In other countries (e.g. Luxembourg, Poland and the Republic of Moldova), the opposite holds true (initially decreasing, then rising) or the impact continuously increased (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.^{34–36} In yet another group of populations (e.g. North America, Australia, Italy and Greece), the impact of these factors remained almost unchanged throughout the observation time.

An important question that exceeds the scope of this article is whether and to what extent the differences between populations in terms of impact of smoking and other non-biological factors reflect the effectiveness of specific public health strategies (see Ref. 37 for an overview of different measures to reduce smoking prevalence). Most importantly, it has been shown that women and men not only exhibit different patterns of tobacco use,⁶ but they also respond differently to specific tobacco control policies what should be analysed in more detail.³⁸ As well, further research is needed to better understand the role of specific non-biological factors other than smoking that cause the different trends of the gender gap across populations, albeit it is likely that their impact is as heterogeneous and context-dependent as that of smoking. For instance, it has been shown that in Eastern Europe GD in LE are strongly influenced by alcohol-related mortality, whereas in North America they are caused predominantly by mortality related to accidents and violence.^{39,40} The data presented in Online Supplementary material S1 can serve as basis for addressing these research questions and translating them into effective and gendered public health programmes to prevent or reduce tobacco consumption and other non-biological risk factors.

Summing up, this study highlights the enormous heterogeneity in impacts of smoking and other non-biological causes on GD in LE. Hence, over-generalized statements which suggest that smoking is the main force behind the gender gap in all developed populations can be 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 LE 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 gender gap but on the other comes along with an increase or stall of the impact of other non-biological factors. The simplistic view that smoking drives the whole non-biological component of GD in LE entails the risk that the increasing importance of specific other factors is overlooked. Thus, our results demonstrate that—regardless of the prevailing effect of smoking—many populations have still remarkable potentials to further narrow their gender gaps in LE.

Supplementary data

Supplementary data are available at *EURPUB* online.

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M.L. developed the research idea and wrote the article. C.W.S. carried out all the data works, developed the extrapolation method for truncated smoking data and contributed to the text. Earlier drafts of the article were presented at the 2013 Annual Meeting of the Population Association of America in New Orleans, USA (12 April 2013), the XVII IUSSP International Population Conference in Busan, South Korea (28 August 2013), the SSRI/DuPRI seminar series at Duke University, Durham, USA (20 March 2014), and the seminar series of the Dondeña Centre for Research on Social Dynamics at Bocconi University, Milan, Italy (31 March 2014). The authors are grateful to the participants of these events and three anonymous reviewers of the *European Journal of Public Health* for their helpful comments and suggestions.

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Conflicts of interest: None declared.

Key points

- This study presents the most extensive analysis of the impact of smoking on the gender gap in LE ever done.
- The role of smoking is by far less clear and homogeneous than commonly stated.
- Although the impact of smoking declines in all studied populations, the impact of the other non-biological factors increases in most countries.
- Populations in which not only the trend but also the extent of the gender gap can be attributed predominantly to smoking can be found exclusively in Europe.
- Many populations have still remarkable potentials to further narrow the gender gap in LE.

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