

## Author Query Form

**Journal:** *European Journal of Public Health*  
**Article Doi:** 10.1093/eurpub/cky273  
**Article Title:** *The role of demographic and behavioural change for the long-term decline in daily smoking in Norway*  
**First Author:** Tord F. Vedøy  
**Corr. Author:** Tord F. Vedøy

### AUTHOR QUERIES – TO BE ANSWERED BY THE CORRESPONDING AUTHOR

The following queries have arisen during the typesetting of your manuscript. Please click on each query number and respond by indicating the change required within the text of the article. If no change is needed please add a note saying “No change.”

- AQ1:** Please check whether the short title is OK as set.
- AQ2:** Please check that name has been spelled correctly and appear in the correct order. Please also check that initial is present. Please check that the author surname (family name) has been correctly identified by a pink background. If this is incorrect, please identify the full surname of the author. Occasionally, the distinction between surname and forename can be ambiguous, and this is to ensure that the authors’ full surname and forename are tagged correctly, for accurate indexing online. Please also check author affiliation.
- AQ3:** Please provide university name for the affiliation.
- AQ4:** Please provide fax number for the corresponding author.
- AQ5:** The citation of the figures is not in the sequential order. Please check and correct.
- AQ6:** Figures have been placed as close as possible to their first citation. Please check that they have no missing sections and that the correct figure legend is present.
- AQ7:** These figures are currently intended to appear online in colour and black and white in print. Please reword the legend/text to avoid using reference to colour. Alternatively, please let us know if you wish to pay for print colour reproduction or to have both versions in black and white. The standard charge for colour reproduction in print is £350/€525/\$600 per figure. Please check the black and white versions at the end of the paper and contact us if you have any concerns.
- AQ8:** Two different expansions (Fraction of daily smokers and Fraction of smokers) have been used for the abbreviation (FrS). Please check.
- AQ9:** Please provide a Funding statement, detailing any funding received. Remember that any funding used while completing this work should be highlighted in a separate Funding section. Please ensure that you use the full official name of the funding body, and if your paper has received funding from any institution, such as NIH, please inform us of the grant number to go into the funding section. We use the institution names to tag NIH-funded articles so they are deposited at PMC. If we already have this information, we will have tagged it and it will appear as coloured text in the funding paragraph. Please check the information is correct.
- AQ10:** In order to validate your funding information prior to publication, please check and confirm whether the name of the funding body given in your manuscript is complete and correct. If any edits are required please mark them on the text.
- AQ11:** Please provide publisher’s name for references [2, 8].
- AQ12:** Please check that all names have been spelled correctly and appear in the correct order. Please also check that all initials are present. Please check that the author surnames (family name) have been correctly identified by a pink background. If this is incorrect, please identify the full surname of the relevant authors. Occasionally, the distinction between surnames and forenames can be ambiguous, and this is to ensure that the authors’ full surnames and forenames are tagged correctly, for accurate indexing online. Please also check all author affiliations.

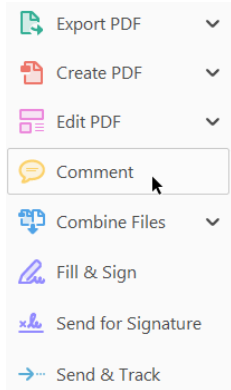
## MAKING CORRECTIONS TO YOUR PROOF

These instructions show you how to mark changes or add notes to your proofs using Adobe Acrobat Professional versions 7 and onwards, or Adobe Reader DC. To check what version you are using go to **Help** then **About**. The latest version of Adobe Reader is available for free from [get.adobe.com/reader](http://get.adobe.com/reader).

### DISPLAYING THE TOOLBARS

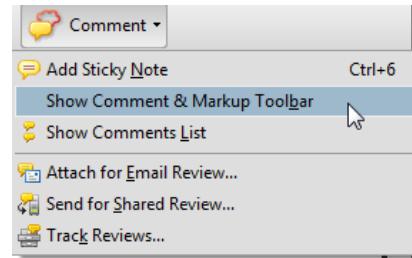
#### Adobe Reader DC

In Adobe Reader DC, the Comment toolbar can be found by clicking 'Comment' in the menu on the right-hand side of the page (shown below).



#### Acrobat Professional 7, 8, and 9

In Adobe Professional, the Comment toolbar can be found by clicking 'Comment(s)' in the top toolbar, and then clicking 'Show Comment & Markup Toolbar' (shown below).



The toolbar shown below will then be displayed along the top.

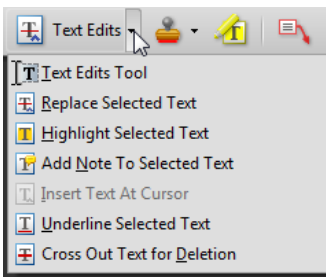


The toolbar shown below will then display along the top.



### USING TEXT EDITS AND COMMENTS IN ADOBE ACROBAT

This is the quickest, simplest and easiest method both to make corrections, and for your corrections to be transferred and checked.



1. Click **Text Edits**
2. Select the text to be annotated or place your cursor at the insertion point and start typing.
3. Click the **Text Edits** drop down arrow and select the required action.

You can also right click on selected text for a range of commenting options, or add sticky notes.

### SAVING COMMENTS

In order to save your comments and notes, you need to save the file (**File, Save**) when you close the document.

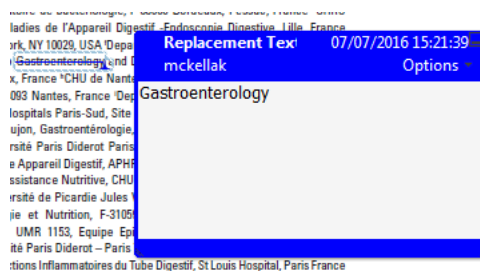
### USING COMMENTING TOOLS IN ADOBE READER

All commenting tools are displayed in the toolbar. You cannot use text edits, however you can still use highlighter, sticky notes, and a variety of insert/replace text options.

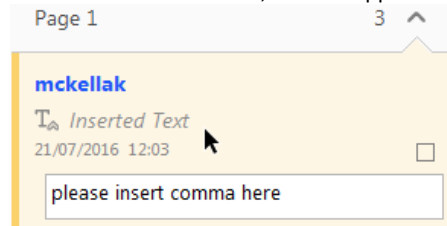


### POP-UP NOTES

In both Reader and Acrobat, when you insert or edit text a pop-up box will appear. In **Acrobat** it looks like this:



In **Reader** it looks like this, and will appear in the right-hand pane:



**DO NOT MAKE ANY EDITS DIRECTLY INTO THE TEXT, USE COMMENTING TOOLS ONLY.**

5

# The role of demographic and behavioural change for the long-term decline in daily smoking in Norway

Tord F. Vedøy

Department of Alcohol, Tobacco and Drugs, Norwegian Institute of Public Health, Oslo, Norway

**Correspondence:** Tord F. Vedøy, Department of Alcohol, Tobacco and Drugs, Norwegian Institute of Public Health, PO Box 4404, Nydalen, 0403 Oslo, Norway, Tel: +47 21077000, Mob: +47 975 86 435, e-mail: tord.vedoy@fhi.no

**Background:** The aim of this paper was to compare the effects of demography (population aging and the increasing fraction of tertiary educated) and behaviour (intra-cohort ageing and inter-cohort change) on long-term change in the fraction of daily smokers (FrS), using a counterfactual framework. **Methods:** Using aggregated data on smoking prevalence, education and population size from Norway 1978 to 2017, the probabilities of smoking for men and women were calculated using a pseudo-panel approach. From these estimates, four counterfactual scenarios of FrS were constructed by holding the age effect, the cohort effect and the distribution of age and education constant over-time. **Results:** FrS decreased from 45 to 14% among men, and from 33 to 14% among women over the study period. Holding the age distribution constant did not have any substantial effect on FrS. Holding the distribution of education constant led to a five percentage points increase in FrS among women, but not among men. In the case of no intra-cohort ageing, FrS would have been 11/12 percentage points higher among women/men. The corresponding figures for no inter-cohort change were 13 points for women and 27 points for men. **Conclusions:** If the age distribution had remained stable over-time, FrS would have been almost identical to the current level. In contrast, if smoking behaviour had remained stable over the life course or between birth cohorts, FrS would have been substantially higher than it is today. These results highlight the large cumulative effect of reducing smoking uptake in successive cohorts.

AQ12



AQ3



AQ4



15

AQ2



20

25

## 30 Introduction

In most developed countries, cigarette smoking has declined substantially over the last decades. In Norway, daily smoking prevalence fell from 44 to 12% among men and 31 to 10% among women in the period 1978–2017.<sup>1</sup> These figures closely resemble findings from other countries in the same phase of the cigarette epidemic.<sup>2,3</sup>

The decline in smoking has been regarded as a public health success,<sup>4</sup> and there is much support for the idea that tobacco control initiatives have reduced smoking, both at the individual and population level.<sup>2,5–7</sup>

Yet smoking persists and forecasting models show that the current smoking prevalence in most European countries is not likely to drop significantly below ten percent over the next decade, unless initiation and cessation rates change substantially.<sup>8,9</sup> In addition, given that many developed countries already have implemented a wide range tobacco control initiatives, and that smokers today may be qualitatively different than previous generations of smokers, the potential for further decline in smoking may be limited.<sup>10,11</sup>

From a macrosocial point of view, long-term variations in smoking can be a result of people changing their behaviour because they grow older (age effects) and/or because new birth cohorts, with different smoking behaviours, enter the social system (cohort effects).<sup>12,13</sup> In addition, events at certain points in time may affect smoking among all groups, for example changes in taxation or new restrictions on where to smoke (period effects).<sup>14,15</sup>

However, long-term variation in smoking prevalence may also result from changes in the demographic composition of a population (demographic effects). If the relative size of a social group with a specific pattern of consumption increases, smoking may vary over-time even in the absence of age, cohort and period effects.<sup>14</sup>

To gauge the potential for further decline of cigarette smoking and potential effects of public health initiatives, we need to assess the relative importance of both demographic change and changes in people's behaviour for long-term changes in smoking.

To this end, the aim of this paper was to examine how the fraction of daily (FrS) smoking has been affected by two demographic processes (population ageing and an increasing fraction of higher educated), and two processes related to changes in behaviour (intra-cohort ageing and inter-cohort change) using Norwegian data from 1978 to 2017.

## Demographic effects on long term changes in smoking

Because of lower birth rates and increased longevity, the populations in most developed countries are ageing.<sup>16,17</sup> A direct consequence is that smoking behaviour among older age groups are increasingly important for the mean smoking prevalence.

A study from the United States showed that demographic change in the period 1980–2010 had a weak negative effect on smoking prevalence.<sup>18</sup> The authors argued that since younger age groups have traditionally had a higher smoking prevalence compared with older age groups, increased population ageing will likely decrease over-all smoking prevalence further.

However, this scenario assumes that smoking behaviours among different age groups are stable. Given that smoking prevalence has decreased among each consecutive birth cohort over the last decades in most developed countries,<sup>12,13</sup> and that smoking has become more prevalent among older compared with younger birth cohorts in some countries,<sup>19</sup> it is possible that population ageing could compensate for decreasing smoking prevalence among younger age groups.

Regarding the distribution of education, the fraction of the population with college or university education has increased substantially over the last decades. In Norway, the percentage who had completed a Bachelor's degree or higher among people aged 25–66 increased from 14 to 40% from 1980 to 2016.<sup>20</sup> This rapid increase in tertiary education can be observed in most other developed countries.<sup>21</sup>

65

70

75

80

85

90

95

The effect of an increased population fraction with tertiary education on smoking prevalence will depend on the mechanisms linking education and smoking. Given that education has a direct effect on people's health related choices,<sup>22</sup> it seems likely that a larger fraction of higher educated people in the population will lead to a lower fraction of smokers, *ceteri paribus*.

To address the issue of demographic and behavioural change for long-term variations in smoking prevalence, this author used data on daily smoking prevalence, population size and the distribution of higher education in the Norwegian population in the period 1978–2017 to examine four counterfactual scenarios of smoking. More specifically, what would the fraction of smokers among men and women be if there were: (i) no changes in the population's age structure, (ii) no changes in the fraction of the population with tertiary education, (iii) no changes in smoking prevalence within birth cohorts (no intra-cohort ageing) or (iv) no changes in smoking prevalence between birth cohorts (no inter-cohort change)?

## Methods

### Data

This study used aggregate data from three different sources, one for the prevalence of smoking, one for the fraction with tertiary education and one for population size. First, information about smoking status came from Statistics Norway's nationally representative survey of smoking habits (SHS) among adults (16–74 years) conducted each year from 1978 to 2017.

In the period 1978–91, the survey was conducted annually and the mean annual number of respondents was 2507. Response rates were above 85% for all these years.<sup>23</sup> From 1992 to 2017, 2000 respondents were invited to participate in each quarter, except for Q2 and Q4 in 2015–17 when 3000 were invited. Response rates varied around 65% in the period 1992–2007, and around 60% from 2008 to 2017. The total number of respondents in the period 1978–2017 aged 16–74 was 154 796.

Second, to minimize selection bias related to education, information about education was retrieved from Statistic Norway's Labour Force Survey (LFS), conducted in each quarter from 1972. The number of respondents in the LFS has varied between 48 000 and 96 000 annually and response rates have been consistently high (85–90%).<sup>23</sup> The total number of respondents aged 16–74 years in the period 1978–2017 was 2 827 075.

Third, information about the population's age structure was retrieved from Statistics Norway's population register and included the number of men and women of all ages (16–74) in each year from 1978 to 2017.

### Measures

Respondents in the SHS were asked: *Do you ever smoke?* Respondents who answered *yes* were then asked if they smoked *daily* or *occasionally*. Our target group were those who answered *daily*.

Both the SHS and the LFS distinguish between having completed nine years of compulsory education (primary), additional three years of college education (secondary) or at least three years of university education (tertiary). However, Statistics Norway redefined secondary education in 2005 so that those who had not completed high school, previously defined as having secondary education, were classified as having primary education. For this reason, respondents with primary or secondary education were combined.

Age and year were grouped in five-year intervals. Respondents below 25 years of age were excluded to ensure that respondents were old enough to have been able to complete at least three years of tertiary education. At the other end of the age span, respondents older than 69 years were excluded to ensure a sufficient number of respondents in each cell.

Information from the SHS and LFS was aggregated according to gender, age, education and survey year using the 'collapse' command in Stata 15 and combined with information about population size. In the case of the LFS, the procedure was weighted by geographic region, age, sex and labour market participation.<sup>24</sup> The resulting data set contained 288 cells [nine age groups (25–29...65–69) × eight time periods (1978–82...2013–17) × two genders × two education groups] with information on the FrS, population size and the fraction with tertiary education.

## Statistical analysis

### The pseudo-panel approach

This study employed a 'pseudo-panel' approach. As shown by Deaton,<sup>25</sup> in the absence of panel data, it is still possible to follow groups, for example birth cohorts, across surveys using aggregate cross-sectional data. It has been shown that when the size of each cohort is large enough, the sample mean provides a consistent estimator of the time-invariant population mean.<sup>26</sup>

The four counterfactual scenarios were constructed based on estimates from two gender specific age-period-cohort regression models. The dependent variable was the fraction of smokers within each year/age/gender/education cell. Because the dependent variable was a fraction, the model was estimated using a robust generalized linear model with a logit link function and binomial distribution of the dependent variable.<sup>27</sup>

Independent variables were education (*primary/secondary* or *tertiary*), dummy variables for each 5-year age group and birth cohort (omitting the youngest age group and the first cohort), and a set of transformed period dummies (see below). Interactions between education and age and education and cohort were included to model the sequential variation in smoking over-time.<sup>28</sup>



### APC-models and the 'identification problem'

As for most social phenomena, long-term variation in smoking is a result of age, period and cohort effects. However, since each temporal dimension is a perfect linear function of the two others, simultaneous estimation of all three effects is not possible without breaking the linear relationship, most often by imposing one or more restrictions.<sup>29</sup>

Several workarounds to this 'identification problem' have been proposed.<sup>30</sup> This study employed a method developed by Deaton in which *age* and *cohort* were entered as dummy variables and *period* was transformed to model short-term variations that average to zero in the long run. For the model to run, the two first year dummy variables were dropped ( $P_{1978-82}$  and  $P_{1983-87}$ ).<sup>31</sup> Because of how the period dummy variables were calculated, the coefficients for the two first dummy variables could be calculated post-regression (the complete models are presented in [supplementary appendix 1](#)).<sup>31</sup>

### Four counterfactual scenarios

From the two gender specific models, the predicted *probability* and variance of daily smoking for each cell was calculated using the 'predictnl' command in Stata 15. From this, the weighted mean and variance for each five-year period was calculated by multiplying the predicted probability and variance for each cell with the relative cell size from the SHS.

The weighted population *fraction* and variance for each cell was calculated by multiplying the predicted probability and variance for each cell with the relative population fraction instead of the relative cell size. 95% confidence intervals for means and fractions were calculated manually from the weighted variances. Note that the transformation of the period dummy variables made it impossible to calculate confidence intervals for the two first periods.

65

70

75

80

85

90

95

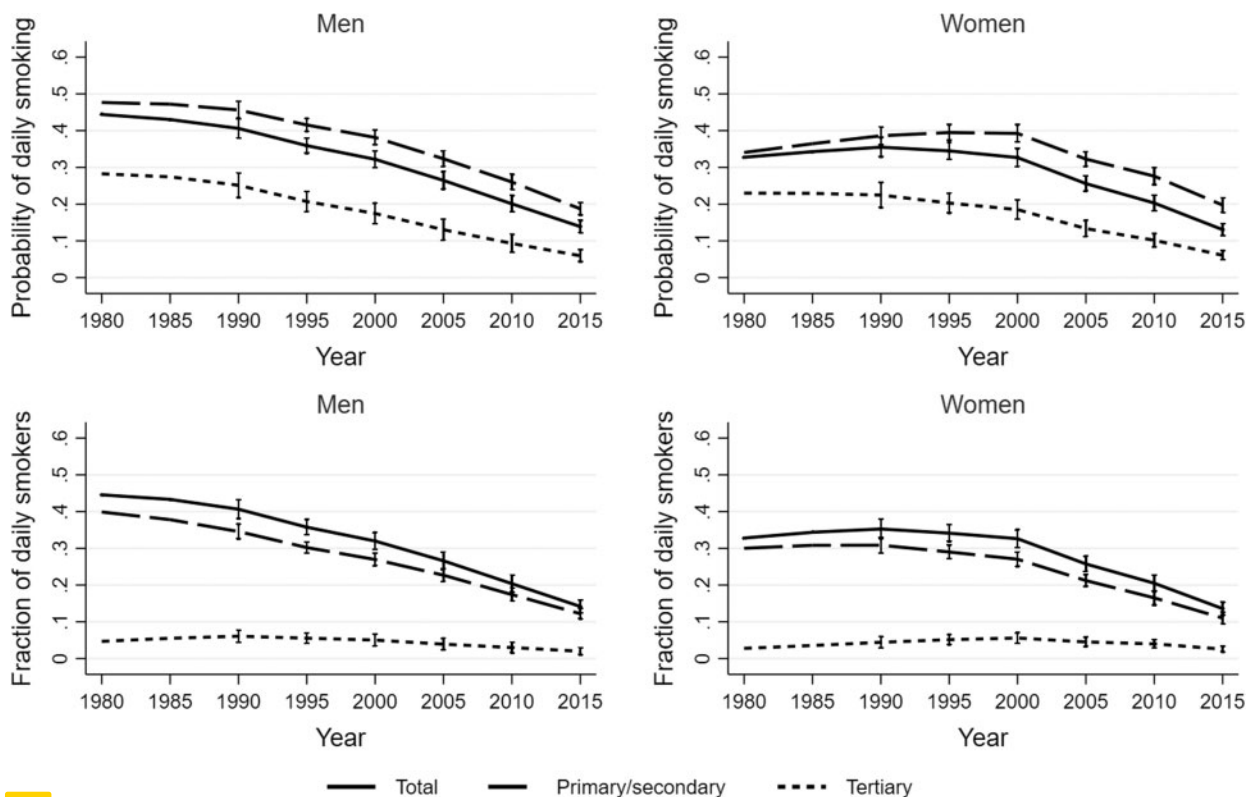
100

105

110

115

120



**Figure 1** The predicted probability (above) and population fraction (below) of daily smoking/smokers, with 95% confidence intervals. Norwegian men and women 25–69 years with primary/secondary or tertiary education, 1978–2017

The two counterfactual scenarios of *no demographic change* were constructed by multiplying the predicted probabilities and variances with:

- (i) a manipulated population variable in which the age distribution in each year was equal to the age distribution in the first period (1978–82).
  - (ii) a manipulated education variable in which the distribution of education was equal to the distribution in 1978–82.
- The two counterfactual scenarios of *no change in behaviour over-time* were created by multiplying the unaltered population fractions with the predicted probabilities of smoking, as they would have been if all groups had:
- (iii) the same age coefficient as the youngest age group within each birth cohort (no intra-cohort ageing).
  - (iv) the same cohort coefficient as the oldest cohort in the corresponding survey year (no inter-cohort change).

Removing the age and cohort effects was accomplished using the ‘predictnl’ command in Stata 15. Since the linear prediction (logit index) of the dependent variable is the sum of the model parameters, the counterfactual scenario of, for example, no intra-cohort ageing can be constructed by replacing the age coefficients for each cell with the age coefficient for youngest age group (25–29), for those cohorts in which 25–29 year olds were the youngest age group.

## Results

Among men, the probability of smoking declined steadily from 0.44 to 0.14 in the period 1978–2017 (figure 1). Among women, the probability was around 0.34 from 1978 to 1997 before declining to 0.13 in 2013–17. Compared with men with tertiary education, the probability of smoking among men with primary or secondary education was 19 points higher in the period 1978–2007 and around 14 points higher in the latest period. Among women, this

gap increased from 11 points in 1978–82 to 21 points in 1998–2002, then decreased to 14 points. The distribution of daily smoking over-time is very similar to the distribution in other countries with a similar smoking history.<sup>32,33</sup>

Given that a relatively small fraction of the population had completed tertiary education, the population fraction of smokers (FrS) with tertiary education varied between two and six percent over the years, both among men and women.

Figure 2 displays the predicted fractions of daily smokers among men and women from the four counterfactual scenarios. The results indicated no difference between the counterfactual scenario of no population ageing (red line) and the predicted values from the base model (black line).

In the case of no change in the population fraction with tertiary education (beige line), FrS among women would have been around five points higher in the period 2003–17 compared with the predicted values from the base model. Among men, the difference between the predicted values and the counterfactual scenario of no change in education was small (around three points) and insignificant.

The effects related to behaviour were stronger than the demographic effects. Compared with the predicted values from the base model, FrS among men in the period 2013–17 would have been 12 points higher if smoking did not vary with age (green line) and 27 points higher if there was no change in smoking across birth cohorts (blue line). Among women in the latest period, FrS would have been 11 and 13 points higher compared with the predicted values, in the case of no intra-cohort ageing and no inter-cohort change respectively.

## Discussion

The main finding from this study was that demographic change has not affected long-term changes in smoking to any large degree, while the effects of changed behaviour were substantial. If the age



35



40

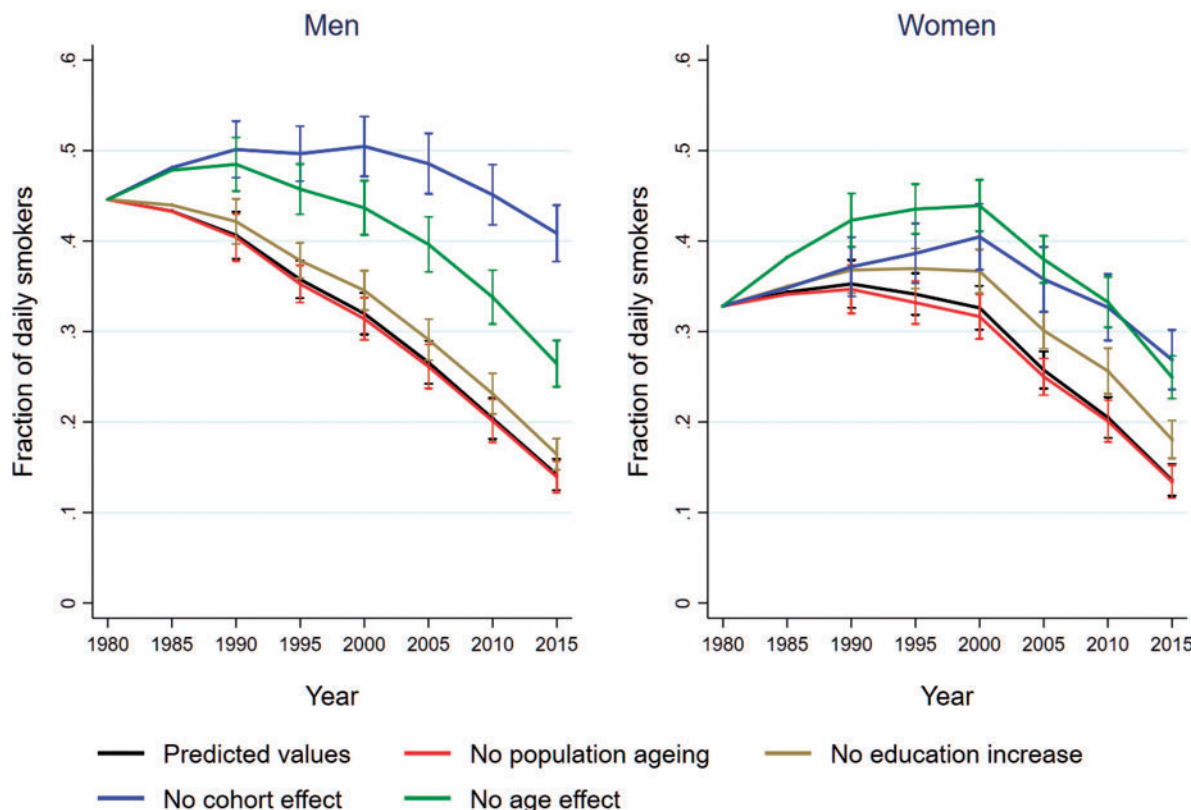
45

50

55

60

65



**Figure 2** Predicted population fractions and 95% confidence intervals of smokers according to four counterfactual scenarios of (i) no population ageing, (ii) no change in the distribution of education, (iii) no inter-cohort change and (iv) no intra-cohort ageing. Norwegian men and women 25–69 years, 1978–2017

distribution had remained stable over the course of time, the fraction of smokers among men and women would have been almost identical to the current level. One exception was the fraction of female smokers, which would have been around five percentage points higher, had the distribution of education remained stable over-time.

The lack of a discernible effect of population ageing on the fraction of smokers is in line with findings from Tam et al.,<sup>18</sup> and does not support the idea that population ageing has been an important factor for the decline in smoking over the last decades.

An explanation of the weak effect of population ageing needs to take into account the underlying population dynamics. When comparing the age structures in 1978 and 2017 we find that middle-aged people (around 45 years) have become relatively more numerous and that young people (around 10 years) have become less numerous. Since smoking is negligible in the latter group, the reduced size of this group has not had any impact on the distribution of smoking.

However, according to population projections, the share of adolescents and young adults will continue to decline while the share of older adults will increase.<sup>34</sup> From a public health perspective, both these processes may help sustain the number of smokers in the future, even if anti-smoking initiatives were to be effective. Moreover, given that fertility rates have been relatively high in Norway over the last decades,<sup>16</sup> the effect of population ageing will likely be stronger in other European countries.

In contrast to population ageing, the increased fraction of tertiary educated had a negative effect on the fraction of smokers, but this effect was limited to women. The small, and in the case of men, indiscernible effect of education was unexpected.

One explanation could be that because of the strong increase in the percentage who have completed higher education in recent decades, the additional number of higher educated must hail from

social groups in which higher education has been less common and smoking more common. Given that smoking patterns to some degree are inherited,<sup>35,36</sup> it seems likely that smoking prevalence would be higher among those men who increasingly constitute the segment of higher educated. This would weaken the negative effect of an increased fraction of higher educated on the over-all fraction of male smokers.

Compared with the two forms of demographic change, the effects of behavioural change (intra-cohort ageing and inter-cohort change) were more important for the over-all decline in smoking over the study period. More specifically, smoking would be substantially higher if smoking remained constant within or across birth cohorts. This is in line with Warner et al.<sup>5</sup> who argued that ‘tobacco control appears to be a much more important factor than demographics in determining California’s low smoking rates’.

While the effect of inter-cohort change was stronger among men compared with women, the effects of intra-cohort ageing, a measure of smoking cessation (given that almost no one initiates smoking after the age of 25), was similar among men and women. This indicates that reduced smoking initiation among men has been the main driver for gender convergence in smoking.

These results show the relative strength of demographic and behavioural effects in a society that has implemented many of the tobacco control measures that most other European countries have implemented, or will implement in the future.<sup>37</sup> From this perspective, the Norwegian experience can shed light on the future of cigarette smoking in countries that follow a similar tobacco control strategy.<sup>38</sup>

### Strengths and limitations

This study was based on a 40-year long series of cross-sectional data on smoking behaviour. By aggregating cross sectional data, it is

AQ1

Fraction of daily smokers 5 of 6

possible to combine different data sources, and by using data on education from the LFS, measurement error related to education was minimized.

However, as with all APC models, the results depend to some degree on the type of model used. The strategy used in this paper, developed by Deaton,<sup>25</sup> posits that long-term social change primarily is a product of inter-cohort change and intra-cohort ageing, while the period-effect represent short-term variations. To gauge the consistency of the model, this author created two gender specific age-cohort-period-characteristic models,<sup>39</sup> where the period-variables were replaced by the real price of cigarettes. These models produced very similar estimates.

## Supplementary data

Supplementary data are available at *EURPUB* online.

## Acknowledgements

I would like to thank Ståle Østhus for comments. Data from the Norwegian Labour Force Survey were collected by Statistics Norway (SSB) and distributed by the Norwegian Centre for Research Data (NSD). Neither SSB nor NSD is responsible for the analyses or interpretations in this study.

## Funding

AQ9 This study received no particular funding and was based on data collected by the Norwegian Institute of Public Health and Statistics

AQ10 Norway.

25 Conflict of interest: None declared.

## Key points

- Changes in the age distribution had no substantial effect on the fraction of smokers in Norway in the period 1978–2017.
- The fraction of female smokers would have been around five percentage points higher, had the distribution of higher education remained stable in the period 1978–2017.
- If smoking patterns had been constant across birth cohorts, the fraction of smokers today would be over three times higher among men and over two times higher among women, compared with the current level.
- These results highlight the large cumulative effect of reducing smoking uptake in successive cohorts.

## References

- 1 Statistics Norway (SSB). Available at: <https://www.ssb.no/en/helse/statistikker/royk> (21 January 2017, date last accessed).
- 2 U.S. Department of Health and Human Services (USDHHS). The Health Consequences of Smoking – 50 Years of Progress. A Report of the Surgeon General. Rockville, MD, 2014.
- 3 Office for National Statistics (ONS), United Kingdom. Available at: <https://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/druguse/cohohandsmoking/datasets/adultsmokinghabitsingreatbritain> (21 April 2016, date last accessed).
- 4 Fiore MC, Baker TB. Stealing a march in the 21(st) century: accelerating progress in the 100-year war against tobacco addiction in the United States. *Am J Public Health* 2009;99:1170–5.
- 5 Warner KE, Mendez D, Alshaqeety O. Tobacco control success versus demographic destiny: examining the causes of the low smoking prevalence in California. *Am J Public Health* 2008;98:268–9.
- 6 Gilpin EA, Messer K, White MM, Pierce JP. What contributed to the major decline in per capita cigarette consumption during California's comprehensive tobacco control programme? *Tob Control* 2006;15:308–16.
- 7 Warner KE. The effects of the anti-smoking campaign on cigarette consumption. *Am J Public Health* 1977;67:645–50.
- 8 World Health Organization (WHO). WHO Global Report on Trends in Prevalence of Tobacco Smoking 2015. Geneva, Switzerland, 2015.
- 9 Pérez-Ferrer C, Jaccard A, Knuchel-Takano A, et al. Inequalities in smoking and obesity in Europe predicted to 2050: findings from the EConDA project. *Scand J Public Health* 2018;46:530–40.
- 10 Talati A, Wickramaratne PJ, Keyes KM, et al. Smoking and psychopathology increasingly associated in recent birth cohorts. *Drug Alcohol Depend* 2013;133:724–32.
- 11 Khlal M, Pampel F, Bricard D, Legleye S. Disadvantaged social groups and the cigarette epidemic: limits of the diffusion of innovations vision. *Int J Environ Res Public Health* 2016;13:1230–5.
- 12 Vedøy TF. Tracing the cigarette epidemic: an age-period-cohort study of education, gender and smoking using a pseudo-panel approach. *Soc Sci Res* 2014;48:35–47.
- 13 Chen X, Lin F, Stanton B, Zhang X APC. modeling of smoking prevalence among US adolescents and young adults. *Am J Health Behav* 2011;35:416–27.
- 14 Ryder NB. The cohort as a concept in the study of social change. *Am Sociol Rev* 1965;30:843–61.
- 15 Yang Y, Land KC. Age-period-cohort analysis of repeated cross-section surveys: fixed or random effects? *Sociol Methods Res* 2008;36:297–326.
- 16 Eurostat. Available at: [http://ec.europa.eu/eurostat/statistics-explained/index.php/Fertility\\_statistics](http://ec.europa.eu/eurostat/statistics-explained/index.php/Fertility_statistics) (11 November 2016, date last accessed).
- 17 Eurostat. Available at: [http://ec.europa.eu/eurostat/statistics-explained/index.php/Mortality\\_and\\_life\\_expectancy\\_statistics](http://ec.europa.eu/eurostat/statistics-explained/index.php/Mortality_and_life_expectancy_statistics) (11 November 2016, date last accessed).
- 18 Tam J, Warner KE, Gillespie BW, Gillespie JA. The impact of changing U.S. demographics on the decline in smoking prevalence, 1980–2010. *Nicotine Tob Res* 2014;16:864–6.
- 19 Anderson CM, Burns DM, Dodd KW, Feuer EJ. Chapter 2: birth cohort specific estimates of smoking behaviors for the U.S. population. *Risk Anal* 2012;32(Suppl. 1):14–24.
- 20 Statistics Norway (SSB). Available at: <https://www.ssb.no/statistikkbanken/selecttable/hovedtabellHjem.asp?KortNavnWeb=utniv&CMSSubjectArea=utdanning&PLanguage=1&checked=true> (07 February 2017, date last accessed).
- 21 Organisation for European Economic Co-operation (OECD). Available at: <https://data.oecd.org/eduatt/adult-education-level.htm#indicator-chart> (07 March 2017, date last accessed).
- 22 Cutler DM, Lleras-Muney A. Understanding differences in health behaviors by education. *J Health Econ* 2010;29:1–28.
- 23 Thomsen I, Villund O. Nonresponse in the Norwegian Labour Force Survey (LFS): Using Administrative Information to Describe Trends. Oslo: Statistics Norway, 2012.
- 24 Bø TP, Håland I. Dokumentasjon av Arbeidskraftundersøkelsen (AKU) etter Omleggingen i 2006 (Documentation for the Labour Force Study (LFS) after Changes Implemented in 2006). Oslo: Statistics Norway, 2015.
- 25 Deaton A. Panel data from time series of cross-sections. *J Econom* 1985;30:109–26.
- 26 Verbeek M, Nijman T. Can cohort data be treated as genuine panel data? *Empir Econ* 1992;17:9–23.
- 27 Papke LE, Wooldridge JM. Econometric methods for fractional response variables with an application to 401 (K) plan participation rates. *J Appl Econ* 1996;11:619–32.
- 28 Thun M, Peto R, Boreham J, Lopez AD. Stages of the cigarette epidemic on entering its second century. *Tob Control* 2012;21:96–101.
- 29 Glenn ND. *Cohort Analysis*. Thousand Oaks, California: Sage Publications, 2005.
- 30 Harding DJ. Recent advances in age-period-cohort analysis. A commentary on Dregan and Armstrong, and on Reither, Hauser and Yang. *Soc Sci Med* 2009;69:1449–51.
- 31 Deaton A. *The Analysis of Household Surveys: A Microeconomic Approach to Development Policy*. Baltimore, Maryland: John Hopkins University Press, 1997.
- 32 Kemm JR. A birth cohort analysis of smoking by adults in Great Britain 1974–1998. *J Public Health Med* 2001;23:306–11.
- 33 de Walque D. Education, information, and smoking decisions: evidence from smoking histories in the United States, 1940–2000. *J Hum Resour* 2010;45:682–717.

AQ11

- 34 The World Bank. Available at: <https://data.worldbank.org/data-catalog/population-projection-tables> (06 October 2017, date last accessed).
- 35 Mays D, Gilman SE, Rende R, et al. Parental smoking exposure and adolescent smoking trajectories. *Pediatrics* 2014;133:983–91.
- 5 36 Breitling LP. Current genetics and epigenetics of smoking/tobacco-related cardiovascular disease. *Arterioscler Thromb Vasc Biol* 2013;33:1468–72.
- 37 Joossens L, Raw M. *The Tobacco Control Scale 2016 in Europe*. Brussels: Association of European Cancer Leagues, 2017.
- 38 Bilano V, Gilmour S, Moffiet T, et al. Global trends and projections for tobacco use, 1990–2025: an analysis of smoking indicators from the WHO Comprehensive Information Systems for Tobacco Control. *Lancet* 2015;385:966–76. 10
- 39 O'Brien R. *Age-Period-Cohort Models: Approaches and Analyses with Aggregate Data*. New York: Chapman and Hall/CRC, 2014.