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Assumptions on mortality

Projecting future mortality in the Netherlands taking into account mortality delay and smoking

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Summary

Estimates of future mortality often prove inaccurate as conventional extrapolative mortality projection methods do not capture the impact of smoking nor the mortality delay: the shift in the age-at-death distribution towards older ages.

The added value of incorporating information on smoking into mortality projections has been demonstrated recently. In addition, recent analyses of mortality trends reveal a transition from mortality compression (a changing shape of the age-at death distribution with more deaths occurring around the modal age at death) towards mortality delay.

In this paper we will estimate future life expectancy for the Netherlands by simultaneously taking into account the effect of smoking and developments in delay and compression of mortality.

Based on lung-cancer and all-cause mortality data from 1950 to 2012, we (1) assess the relationship between smoking and the extent of mortality delay versus mortality compression using our CoDe mortality model, (2) project non-smoking-related mortality by extrapolating the obtained delay (and compression) parameter values up until 2050, (3) project all-cause mortality by combining this projection of non-smoking-related mortality with an earlier projection of smoking-related mortality.

Changes over time in delay and compression for the total population in the Netherlands result from changes in the age at death distribution of smokers and non-smokers, and the prevalence of smoking. Compared to the total population, for non-smokers increases in the modal age at death – indicating mortality delay – are much more linear, and more similar for men and women. Projections based on mortality delay for the Netherlands should therefore take into account smoking.

Our mortality projection – simultaneously taking into account mortality delay and smoking – results in higher life expectancy values in 2050 than a conventional (=Lee-Carter) projection, more delay, and more deaths at advanced ages.

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I. Introduction

1. Mortality forecasts and projections are essential for predicting the extent of population ageing, and for determining the sustainability of pension schemes and social security systems. They are also useful in setting life insurance premiums, and in helping governments to better plan for the changing needs of their societies for health care and other services (European Commission 2009).
2. However, current forecasts of future mortality often prove inaccurate. That is, the forecasts are not robust over time. As past mortality forecasts have repeatedly proven too pessimistic (Oeppen & Vaupel 2002), forecasts of future life expectancy by national statistical offices are frequently being adjusted upwards.
3. Mortality forecasts are inaccurate largely because conventional extrapolative methods do not capture (1) the impact of lifestyle ‘epidemics’ on mortality, which result in non-gradual trends over time and in large differences between men and women, birth cohorts, and countries; and (2) mortality delay: the shift in the age-at-death distribution towards older ages.
4. With the general increase in life expectancy at birth (e_0), changes in the age distribution of dying occurred. The theory distinguishes two scenarios which actually operate in tandem: a decline in premature mortality with no increase in maximum lifespan, which results in more people dying at the same ages (‘mortality compression’; also referred to as ‘rectangularization of the survival curve’) (Fries 1980); and a mortality delay, which manifests in a shift in the age-at-death distribution to the right, and thus in increases in lifespan and in the number of centenarians (‘shifting mortality regime’) (Vaupel 2010).
5. The relative roles of the two processes in the mortality trend are important for the future development of e_0 . If only ‘rectangularization’ occurs, we would be approaching a limit to life expectancy. If, however, ‘delay in aging’ occurs, a limit to life expectancy is unlikely for the near future.
6. Whereas, historically, in low-mortality countries a general decline in the variability in the age of dying has been observed (e.g. Wilmoth & Horiuchi, 1999; Kannisto 2000,2001; Robine 2001; Engelman et al. 2010) the evidence for the post-war period is much more mixed. Evidence exists for a continuing compression albeit slowing down (Wilmoth & Horiuchi, 1999; Kannisto 2000,2001; Robine 2001; Edwards and Tuljapurkar 2005; Canudas-Romo 2008; Cheung et al. 2008; Thatcher et al., 2010), for increases in the variability of mortality (Nusselder & Mackenbach 1996; Wilmoth & Horiuchi, 1999; Yashin et al. 2001; Shkolnikov et al 2003; Shkolnikov et al. 2011) and for a shift of the survival curve to the right (Kannisto 1996; Bongaarts 2005; Cheung et al. 2008, 2009; Canudas-Romo 2008; Ouellette & Bourbeau 2011). Recent formal analyses of mortality trends reveal a transition from mortality compression towards mortality delay (Bergeron-Boucher et al. 2015; Janssen et al. in preparation).
7. Mortality projections including the changing patterns in the age-at-death distribution have been applied before, albeit very rarely, only for single populations/countries, and mostly including only the mortality delay and not mortality compression (Bongaarts, 2005). Also these mortality projections did not account for the effect of smoking.
8. The added value of incorporating information on smoking into mortality projections has been demonstrated recently (Pampel 2005, Bongaarts 2006, Wang & Preston 2009, King & Soneji 2011, Preston et al. 2012, Janssen, van Wissen & Kunst, 2013). Within Europe, smoking is known to be the single most important determinant of mortality levels, mortality trends and differences therein between countries and sexes (Lopez et al. 1994, McCartney et al. 2011). Smoking has been taken up first among men in north-western Europe, at varying rates by

different birth cohorts, and has resulted in non-linear mortality trends (Janssen & Kunst 2005; Preston & Wang 2006; Janssen et al. 2007). Smoking-attributable mortality increased and subsequently declined about 30 years after the increase and subsequent decline in smoking prevalence (Lopez et al. 1994).

9. Because of the high prevalence of smoking in the past, the smoking epidemic will leave an imprint on population health for many years to come (Bongaarts 2006; Wang & Preston 2009), although differentially for males and females. More importantly, however, because of the non-linear and differential patterns imposed by the smoking epidemic, linear extrapolations of the past trends will very likely result in non-robust outcomes. Distinguishing between the gradual long-term mortality decline, due to socio-economic factors and associated medical improvements (Mackenbach 2013), and the smoking epidemic which causes deviations from and variations in the general mortality decline is essential to come to more robust projection outcomes.
10. Recent analyses for men in 10 European countries showed already that smoking also greatly affects the age-at-death distribution (Janssen, Rousson & Paccaud 2015). Also this study showed there was a greater delay in ageing in the trends for non-smoking-related mortality than in all-cause mortality (Janssen, Rousson & Paccaud 2015).
11. In the current analysis we will estimate future life expectancy for the Netherlands by simultaneously taking into account the effect of smoking and developments in delay and compression of mortality.

II. Data and methods

12. For the purpose of our analysis we've obtained data on population and mortality by sex and separate ages (ranging from 0 to 110+) for the period of 1950 to 2013 from Statistics Netherlands. Statistics on mortality attributable to lung cancer for the years 1950 to 2012 by five-year age categories are obtained by the WHOSIS, and are transformed into age-specific mortality rates $m(x)$ by using data on the population (average population) from Statistics Netherlands. These age-specific mortality rates $m(x)$ are, in turn, converted to age-specific mortality probabilities $q(x)$.
13. To outline the role of compression and the role of the delay in age of dying over time within the age distribution of dying we use the CoDe model (see De Beer & Janssen, submitted). In this paper we use a simplified version of the model as we apply the model only to the ages of 40 and above. This model divides the age-specific probability of dying into four components: background mortality, adult-age mortality, middle-age mortality and old-age mortality.

$$q(x) = a + I(x \leq x_1) \left[\frac{b_1 e^{b_1(x-M)}}{1 + \frac{b_1}{g} e^{b_1(x-M)}} \right] + I(x_1 < x \leq x_2) \left[\frac{b_2 e^{b_2(x-M)}}{1 + \frac{b_2}{g} e^{b_2(x-M)}} + c_1 \right] \\ + I(x > x_2) \left[\frac{b_3 e^{b_3(x-M)}}{1 + \frac{b_3}{g} e^{b_3(x-M)}} + c_2 \right]$$

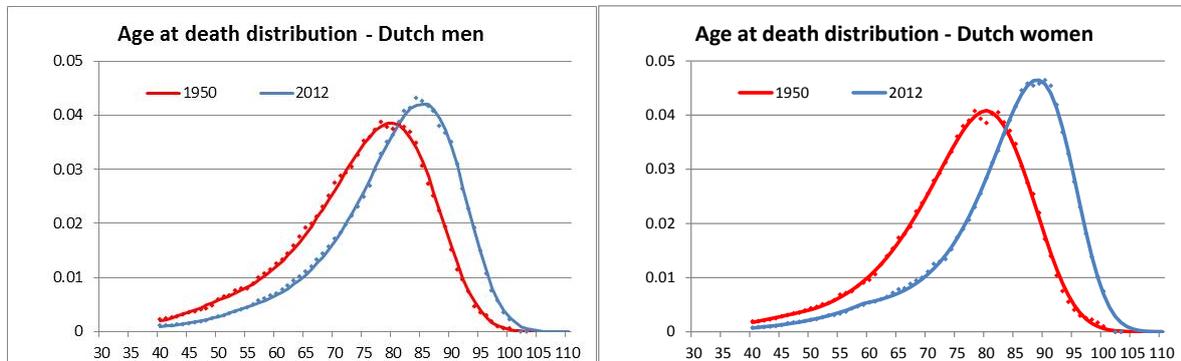
where $x_2 = M$ and $x_1 = M - h$.

and where $c_1 = \frac{b_1 e^{b_1(x_1-M)}}{1 + \frac{b_1}{g} e^{b_1(x_1-M)}} - \frac{b_2 e^{b_2(x_1-M)}}{1 + \frac{b_2}{g} e^{b_2(x_1-M)}}$ and $c_2 = \frac{b_2 e^{b_2(x_2-M)}}{1 + \frac{b_2}{g} e^{b_2(x_2-M)}} + c_1 - \frac{b_3 e^{b_3(x_2-M)}}{1 + \frac{b_3}{g} e^{b_3(x_2-M)}}$

14. Here, both g – the value of $q(x)$ at the maximum age – and h are set at fixed values of 0,7 and 30 respectively. This results in five interpretable time-dependent variables: a , b_1 , b_2 , b_3 and M . M represents the modal age of dying. An increase in M would thus indicate a delay in mortality. The b -parameters display the shape of the age distribution. An increase in b_1 and b_2 lead to a steeper curve *before* the modal age, and thus a compression of mortality, which will, in turn, lead to an increase in life expectancy. An increase in b_3 , on the other hand, leads to a steeper curve *past* the modal age of dying, and thus also a compression of mortality. An increase in b_3 will thus lead to a decrease in life expectancy at these ages (see Appendix I).
15. In those instances where the values of b_1 and b_2 were very dissimilar, which would imply an irregular age at death distribution, we applied the restriction $b_1=b_2$, so we effectively used a simpler model.
16. The role of smoking is outlined by estimating the share of mortality attributable to smoking, for which we use an adjusted version of the indirect Peto-Lopez-technique (Peto et al. 1992; Janssen et al. 2013). Subsequently, we studied the trends of total mortality and non-smoking-related mortality (when the effect of smoking is removed) over time. The non-smoking-related mortality is obtained by multiplying the sex- and age-specific mortality $m(x)$ by 1 minus the share of the total mortality explained by smoking.
17. Furthermore, we studied changes in the distribution of the age of dying for the total population, smokers and non-smokers by using the CoDe model (which only applies to ages of 40 and above). The probability of dying for non-smokers is calculated by multiplying the sex- and age-specific probability of dying $q(x)$ for the of the total mortality by 1 minus the share of the total mortality explained by smoking. The probability of dying for smokers is obtained by multiplying the sex- and age-specific probability of dying $q(x)$ for non-smokers by the relative risks of dying due to smoking.
18. Lastly, we calculated future mortality for non-smoking-related mortality by extrapolating the various time-varying parameters of the CoDe model, which were then applied to non-smoking related mortality. The modal age at death (M) is projected by means of a random walk with drift, whereas we fitted linear regression to the other parameters. We compared one estimation where we only extrapolated M and another estimation where we extrapolated all parameters. This projection is compared to a standard Lee-Carter projection (Lee & Carter 1992). Furthermore, a projection for total mortality is carried out, which is based on the projection of non-smoking-related mortality and a previous projection of the share of total mortality which is attributable to smoking (Stoeldraijer et al. 2015). We used the periods of 1950-2012 as well as 1980-2012 as the base period for these various projections by which we obtained the remaining life expectancy at the age of 40 for 2050.

III. Results

Figure 1 Lifetable age at death distribution (radix = 1), observed (dots) and fitted (lines), the Netherlands, 1950-2012, 40+, by sex



19. Figure 1 clearly shows that the fit of the CoDe model is excellent. In addition it can be observed, when focusing on the trend over time, that delay is much more important than compression in describing the trends over time (which also shows when examining the trends in the different parameters of the CoDe model)(see Appendix II). For men in addition we observe some compression before the modal age at dying leading to additional gain in life expectancy; and some compression at higher ages leading to a small decline in life expectancy. For women, delay is much stronger, and coupled with expansion before the mode (decline in e_{40}) and clearly compression after the mode (also resulting in decline in e_{40}).
20. Figure 2 shows different shapes of the age at death distribution for nonsmokers and smokers among men and women in 1950, 1980 and 2012. For men in 1950, we can clearly see that the age at death distribution is more compressed for nonsmokers than for the total population. Especially before the mode mortality among nonsmokers exhibits a steeper curve than mortality for the total population. For smokers however we see that the modal age is lower than for the total population. A similar pattern can be observed for men in 2012, however for men in 1980 we see a different pattern when comparing smokers with the total population. That is, in 1980 the age-at-death distribution for the total male population almost resembles the age-at-death distribution for male smokers. This has everything to do with the past smoking intensities being extremely high at that point in time. Almost 70 % among Dutch men aged 40 and over were exposed to smoking (see Appendix III). Similarly for women in 1950 and 1980, when lifetime smoking is virtually absent, the age at death distribution for the total population fully resembles the age at death distribution for female non-smokers. For smokers again the mode is lower than for the total and the non-smoking population. For women in 2012 we can again observe that the age at death distribution is more compressed for non-smokers in comparison to the total population and that the mode for smokers occurs at a younger age as compared to the total population, although differences are less than for men, because of much lower past smoking prevalence. The age at death distribution for all-cause mortality thus clearly depends on the age at death distribution for smokers and non-smokers and the prevalence of smoking, whereas smokers tend to have a modal age at death which is lower than nonsmokers, and the age at death distribution for nonsmokers is generally more compressed than the age at death distribution for the smokers.

Figure 2 Fitted lifetable age at death distribution (radix = 1) for the total population, non-smokers and smokers

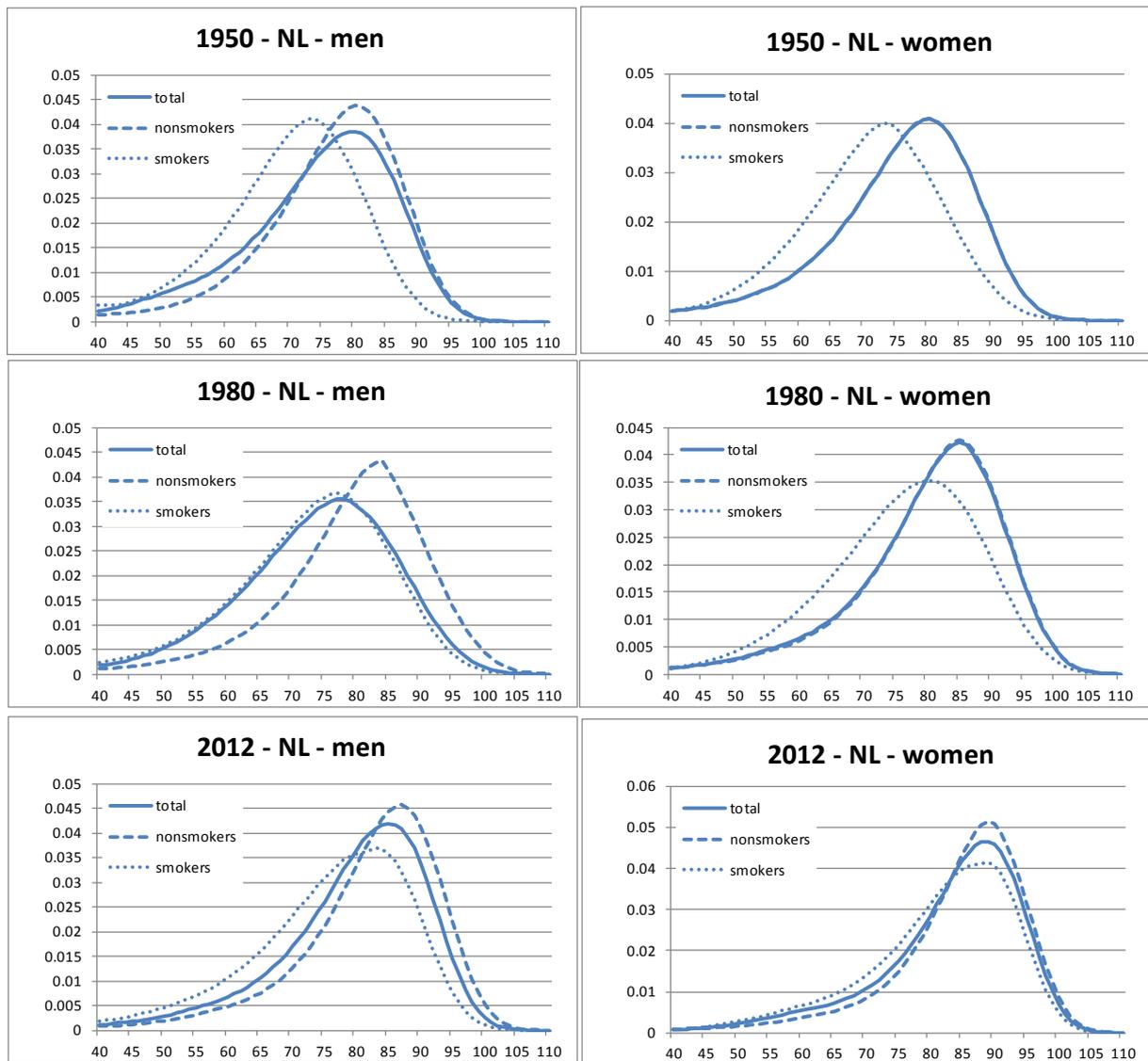
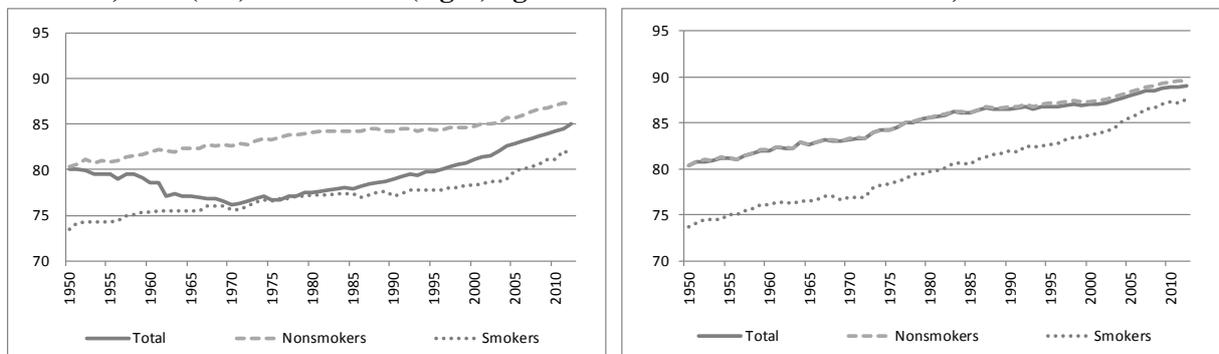


Figure 3 Trends over time in the modal age at death, for the total population, nonsmokers and smokers, men (left) and women (right) aged 40 and over in the Netherlands, 1950-2012



21. Figure 3 shows that for the total male population the modal age at death declined till 1970, after which it has increased. This can be linked to our earlier observation that the age-at-death distribution is determined both by the age at death distribution of smokers and nonsmokers, and by a change in the prevalence of smoking. For nonsmokers and smokers the trends in the modal age at dying are clearly more linear as compared to the total population and more parallel between men and women. For both men and women a slight stagnation of the increase in M occurred in the 1990s as well as an acceleration of the increase from 2003 onwards. What can be observed as well is that the modal age converges between smokers and nonsmokers.
22. Examining the trends for the compression parameters b_1 , b_2 and b_3 (see Figure 4) it shows that for nonsmokers trends are more similar between the sexes and more linear as compared to the total population, except for b_3 among men. Especially compression before the mode (b_1 and b_2) seems to be influenced by smoking.

Figure 4 Trends in the compression parameters, for the total, nonsmoking and smoking 40+ population in the Netherlands, by sex, 1950-2012

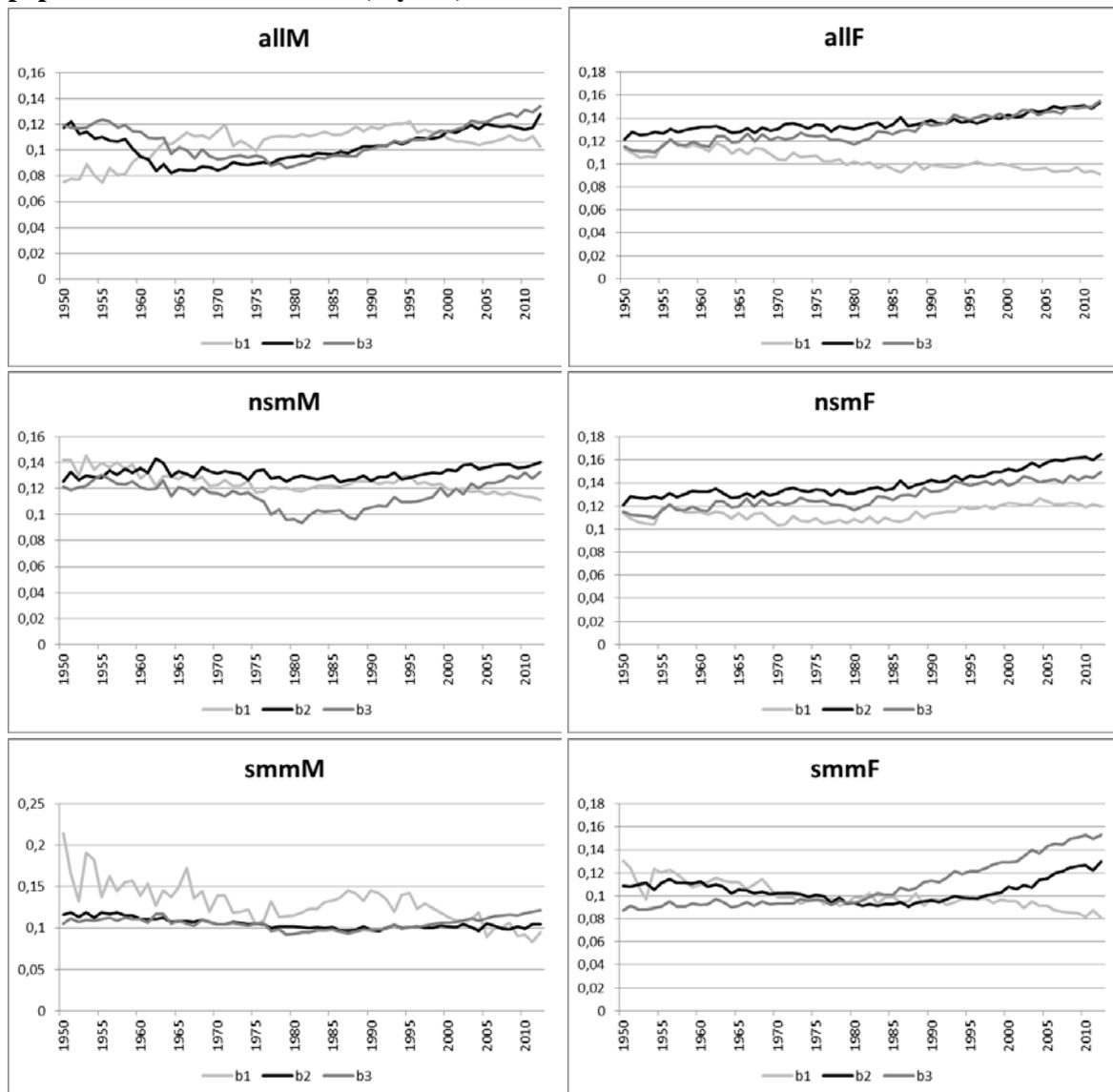
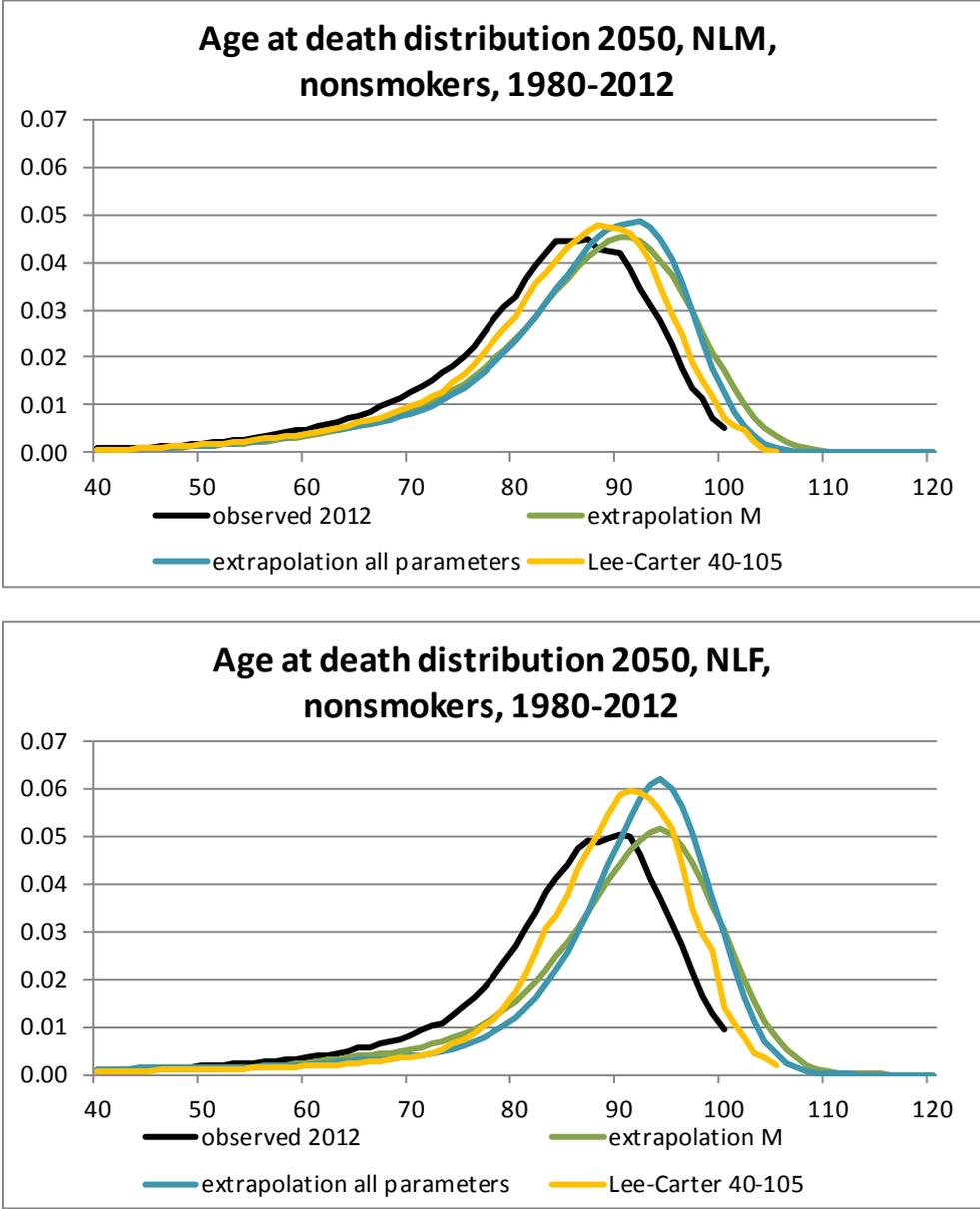


Table 1 Projected life expectancy at age 40 in 2050 for nonsmokers and the total population, for different projections and different historical periods, the Netherlands, by sex

| | 1950-2012 | | 1980-2012 | |
|---|-----------|-------|-----------|-------|
| | men | women | men | women |
| nonsmokers (e40 2012 = 42.76 (M), 45.15 (F)) | | | | |
| extrapolation M; rest similar to 2012 ns | 46.80 | 50.51 | 46.37 | 49.62 |
| extrapolation a, b1, b2, b3, M | 46.47 | 50.58 | 46.00 | 50.02 |
| LC 40-100 | 45.94 | 49.23 | 45.07 | 48.68 |
| total (e40 2012 = 40.22 (M), 43.57 (F)) | | | | |
| extrapolation nonsmokers (M) + SAM (APC) | 45.63 | 48.79 | 45.20 | 47.92 |
| extrapolation nonsmokers (all parameters) + SAM (APC) | 45.21 | 48.94 | 44.87 | 48.51 |
| LC 40-100 | 42.69 | 47.54 | 44.89 | 46.39 |
| SAM = smoking-attributable mortality | | | | |
| APC = age-period-cohort analyses | | | | |

23. Simple extrapolation of the parameters of the CoDe model for nonsmokers, either solely the modal age at death or all parameters, results in a higher e40 in 2050 compared to a Lee-Carter projection.
24. The effect of taking into account the additional – mainly compression - parameters results in slightly lower e40 for men, and a slightly higher e40 for women. This can be linked to the prediction of less compression before the modal age at death for men as compared to before, and the prediction of an increase in compression before the modal age at death for women. The impact of projecting delay of mortality on the projection of life expectancy is clearly bigger than the effect of compression.
25. Adding to the CoDe extrapolations for nonsmokers the extrapolation of mortality for smokers (based on our age-period-cohort methodology) results as well in higher e40 than the direct LC extrapolation for all-cause mortality, except when for men trends in only the modal age at death from 1980-2012 are extrapolated. Then the outcomes are virtually the same as the direct LC extrapolation, at least in terms of life expectancy. Note that the projections of the Lee-Carter are more sensitive to the choice of the base period than the projections taking into account the impact of smoking and delay of mortality.
26. Looking at the full age at death distribution (Figure 5), some interesting differences between both projections can be observed. From Figure 5, four observations can be made. Firstly, we can clearly see that the Lee-Carter projection shows much less delay than our two extrapolations. Secondly, more people die above ages 95 or 100 in our projections (especially for women). Thirdly, the projection in which we extrapolate all parameters seems to reflect the LC forecast + delay. Fourthly, the Lee-Carter projection seems to show slightly more fluctuations /irregularities in the age at death distribution.

Figure 5 Projected lifetable age at death distribution for Dutch male and female nonsmokers aged 40 and over, in 2050. Comparison of different projections



IV. Conclusion

- 27. Changes over time in delay and compression for the total population in the Netherlands result from changes in the age at death distribution of smokers and non-smokers, and the prevalence of smoking. Compared to the total population, for non-smokers increases in the modal age at death – indicating mortality delay - are much more linear, and more similar for men and women. Projections based on mortality delay for the Netherlands should therefore take into account smoking.
- 28. Our mortality projection – simultaneously taking into account mortality delay and smoking - results in higher life expectancy values in 2050 than a conventional (=Lee-Carter) projection, more delay and more deaths at advanced ages.

V. Discussion

- Comparison to the Statistics Netherlands forecast

29. The current mortality forecast of Statistics Netherlands (Stoeldraijer et al. 2013) already includes the effect of smoking and also includes the mortality experience of the opposite sex and in other countries, thereby using the methodology developed by Janssen et al (Janssen et al. 2013). However it does not take into account the effect of mortality delay.
30. Their estimate for e_{40} in 2050 is 46.6 for men and 49.2 for women - based on the trends since 1970 - whereas our estimate is slightly lower for men (44.8-45.2) and about equal to the estimate for women (49.4-49.0). It is as yet unclear which part of the difference is due to not taking into account mortality delay, and which part is due to the use of a coherent mortality projection by Statistics Netherlands.

- Further work

31. A logical expansion of the discussed mortality projection technique would be to include the mortality experience of the opposite sex and trends in other countries when projecting non-smoking-relating mortality, and thus making it a coherent mortality projection including smoking and the mortality delay.

VI. Acknowledgements

32. This work is partly financed by the Netherlands Organisation for Scientific Research (NWO) in relation to the research programme “Smoking, alcohol, and obesity, ingredients for improved and robust mortality projections.” (grant no. 452-13-001).

See as well: <http://www.rug.nl/research/future-mortality>.

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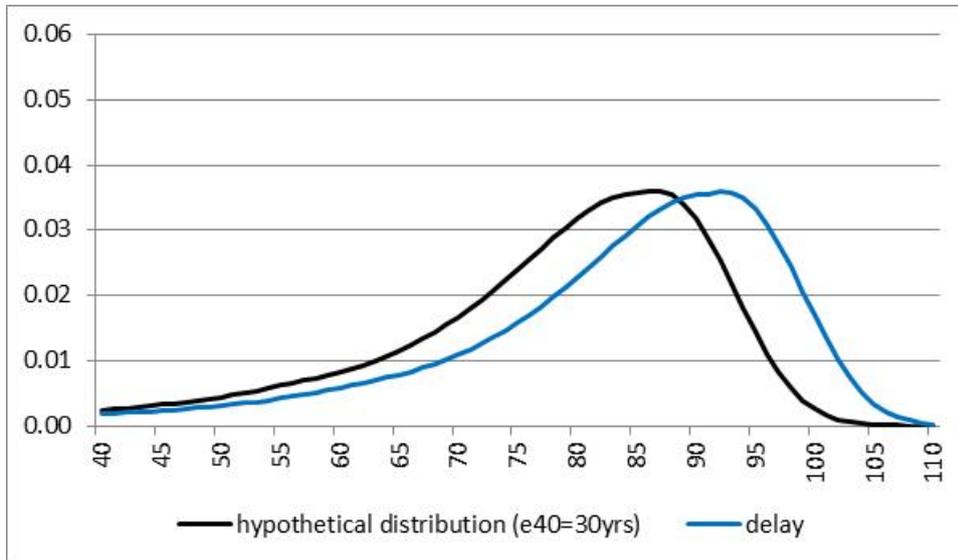
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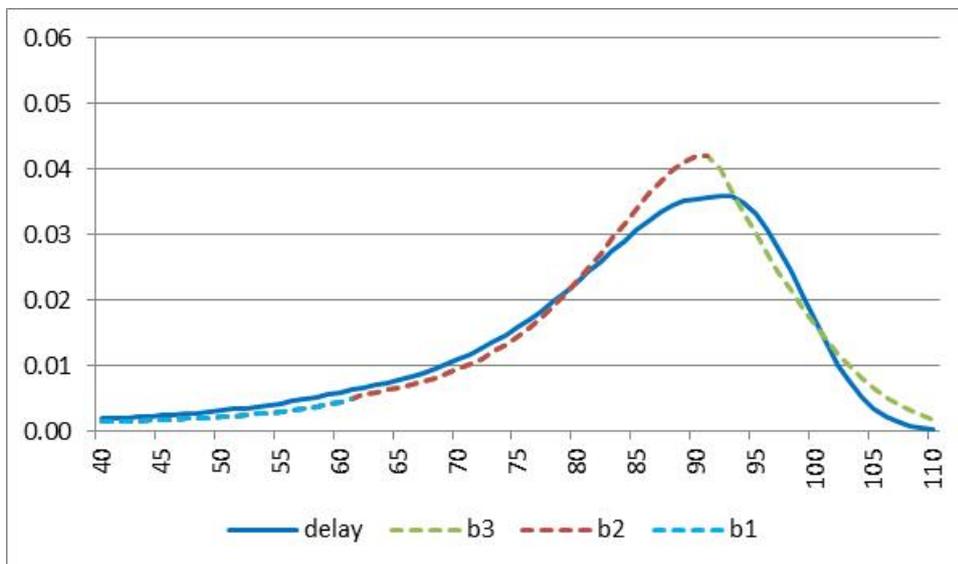
Appendix I

Effect of the parameters of the applied model based on a hypothetical age at death distribution which resembles a remaining life expectancy at age 40 (e_{40}) of 30 years

Effect of an increase in the modal age at death corresponding with a 5 years increase in e_{40}



Effect of an increase in b_1 combined with the effect of an increase in b_2 combined with a decline in b_3 , all reflecting a 0.5 years increase in e_{40} (thus 1.5 years increase in total)



Appendix II

The different parameters of the CoDe model for those aged 40 and over in the Netherlands, by sex, 1950, 1980, 2012

| | total population | | | nonsmokers | | | smokers | | |
|--------------|------------------|---------|---------|------------|---------|---------|---------|---------|---------|
| | 1950 | 1980 | 2012 | 1950 | 1980 | 2012 | 1950 | 1980 | 2012 |
| Men | | | | | | | | | |
| a | -0.0017 | 0.0000 | 0.0000 | 0.0009 | 0.0005 | 0.0002 | 0.0031 | 0.0009 | 0.0000 |
| b1 | 0.0755 | 0.1103 | 0.1029 | 0.1419 | 0.1188 | 0.1115 | 0.2137 | 0.1152 | 0.0948 |
| b2 | 0.1177 | 0.0948 | 0.1277 | 0.1258 | 0.1285 | 0.1402 | 0.1157 | 0.1017 | 0.1044 |
| b3 | 0.1193 | 0.0880 | 0.1341 | 0.1215 | 0.0963 | 0.1329 | 0.1057 | 0.0933 | 0.1215 |
| M | 79.9984 | 77.5729 | 85.0469 | 80.4220 | 84.0962 | 87.2444 | 73.5540 | 77.2369 | 81.4735 |
| Women | | | | | | | | | |
| a | 0.0007 | 0.0002 | -0.0003 | 0.0007 | 0.0004 | 0.0005 | 0.0002 | -0.0010 | -0.0011 |
| b1 | 0.1133 | 0.1020 | 0.0916 | 0.1136 | 0.1084 | 0.1193 | 0.1303 | 0.0983 | 0.0809 |
| b2 | 0.1211 | 0.1305 | 0.1535 | 0.1211 | 0.1313 | 0.1649 | 0.1087 | 0.0939 | 0.1301 |
| b3 | 0.1150 | 0.1168 | 0.1548 | 0.1150 | 0.1166 | 0.1496 | 0.0873 | 0.0938 | 0.1531 |
| M | 80.3754 | 85.5382 | 89.0655 | 80.3748 | 85.6003 | 89.5696 | 73.7691 | 79.7570 | 87.5596 |

Appendix III

Past smoking intensities (40+), the Netherlands, 1950-2012

