Changes in patterns of smoking related causes of death and the impact on the "Golden generations" in the UK

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Introduction

It is widely recognized that tobacco use is a risk factor for many diseases such as lung cancer, oral cancer, coronary heart disease and emphysema among others. The analysis of mortality trends for these causes of deaths suggests a strong cohort effect (especially for lung cancer) and differences between trends for men and women indicate differences in gender cause-specific cohort patterns and implicitly in smoking patterns as well.

The cohort approach is particularly important in UK since changes in smoking patterns are often considered to be the main driver of the higher-than-average rates of mortality improvement of the 1925-1945 UK-born generations (often referred to as the "Golden generations"), and that as they are replaced by less-favoured generations, mortality improvements observed in the last decades would slacken. This assumption underpins all official mortality forecasts since 1992 and since mortality is the main determinant of future numbers of older people in decades to come, it is important for policy and planning purposes. A plausible hypothesis for nearly 20 years for the existence of these "Golden generations" is a "one-off" benefit from reductions in smoking, although evidence to support this most commonly cited reason is lacking (Office of Population Censuses and Surveys, 1995, see also Willets, 2004). In the authoritative consensus view of this area, the British National Statistician (Dunnell, 2008, p. 19) accepted the existence of the "Golden generations", but concluded that no clear-cut causal mechanisms have been established and there are only a series of hypotheses that include:

1. Changing smoking patterns between generations
2. Better diet and environmental conditions during and after the Second World War
3. Those born in periods of low fertility facing less competition for resources as they age
4. Benefits from the introduction in the late 1940s of the Welfare State
5. Benefits from medical advances.

Singer and Manton (1998) suggested an additional specific explanation for these cohorts, that improvement in food preparation and packaging in the 1920s and 1930s may also have had an influence on later mortality. Alternative cohort influences have been suggested: Finch and Crimmins (2004) identified cohort effects in four northern European countries by examining the relationship between infant and child mortality with mortality of the same cohorts at older ages of 75 to 79, underpinned by a theoretical framework on the role of childhood morbidity, especially inflammation, and later extended to include height (Crimmins and Finch, 2006). However, they confine analysis to cohorts born before around 1900 since they argue that for later cohorts, period factors relating to the environment or medical advances means that such childhood effects would not be expected to be observed, i.e., that the suggested mechanism is relevant only to cohorts born in the Nineteenth and earlier centuries, a similar conclusion was reached by Su (2009). It is therefore surprising how little attention has been given to establishing the validity or otherwise for the primacy of the smoking explanation in Britain, although work has been undertaken to examine how far smoking can account for observed patterns of sex differentials in mortality in United States by Preston and Wang (2006). In particular, any explanation based on the role of smoking on mortality should be able to explain observed differences in the patterns of mortality for men and women separately.

This paper presents cohort estimates of some of the main relevant causes of death that are used to elucidate the role of selected smoking related causes of death in England and Wales, and we analyze the relation between the annual change in the cohort effect (as estimated by the cohort function in an Age-Period-Cohort model) for lung cancer, chronic obstructive pulmonary diseases (COPD) and overall mortality in order to attribute the proportion of overall cohort mortality change due to smoking patterns.

Smoking and its effect on mortality

Smoking is the single most important cause of premature death in developed countries, with the risk of death from smoking far exceeding that of any other addiction, exposure or injury (Lopez, Collishaw, and Piha, 1994; Peto et al., 1992).

Much of the analysis of tobacco-related mortality is based on the Peto-Lopez method (1992), which attributes a fraction of certain causes of death to tobacco using period data. Peto et al (2006) estimated that almost one hundred thousand people died from smoking at 2000 in England and Wales. Smoking-related causes are responsible for five times the number of deaths as external causes (such as murder, assault, suicide, and accidents). About 27,000 of these smoking-related deaths were among those aged 35-69, or one in

five deaths in this age-group. For these decedents, smoking represents an average loss of about 21 years of life.

Smoking increases the risk of death for many causes such as lung cancer, upper autodigestive cancer, cardiovascular diseases, and respiratory diseases (Rogers et al. 2005; Ravenholt, 1990). International comparisons suggest that the impact of smoking on the pace and timing of mortality decline varies considerably from country to country and between the two sexes, in part due to differences between birth cohorts in lifetime exposure to smoking (Janssen, Kunst, and Mackenbach, 2007; Preston and Yang, 2006; Preston et al. 2009). Smoking deaths contribute to the international mortality gender gap; for example after controlling for smoking, the gender gap in mortality is identical in Eastern and Western Europe (Bobak, 2003). The United Kingdom can be considered a good example for investigation because of the different levels and uptake of smoking by men and women.

Data and methods

A range of methods are used to assess the magnitude of smoking attributable mortality (Pérez-Ríos and Montes 2008), the majority of which break down into methods that use information on smoking prevalence and those that use only cause-specific mortality. We use the latter approach, based on registered deaths by sex, age and cause of death in England and Wales in the period 1950-2007 (Twentieth and Twenty-first century mortality database, ONS).

However, while smoking deaths are known to be strongly associated with cohort experiences, cohort approaches have only recently been reconsidered and refined (Preston and Wang, 2006). Since our particular interest is in cohort patterns, and, in particular, the future implications of recent trends, we use Age-Period-Cohort (APC) models to separate out age, period, and cohort effects for people aged 35 and over (we consider smoking as a cause of death to be negligible for people aged less than 35) for lung cancer and chronic obstructive pulmonary diseases (COPD). This means that it is possible to reconstruct at least part of the mortality experience of cohorts as far back as those born in 1865 up to the latest cohort born in 1972. The general APC model for the log-death rates m_{apc}, at age *a*, in period *p* for persons in birth cohort (*c=p-a)*, is:

$$\ln(m_{apc}) = f(a) + g(p) + h(c)$$

where the age function $f(a)$ represents the log age-specific rates in the reference cohort c_0 (in our case 1900), the cohort function $h(c)$ is the log rate ratio of cohort c compared to cohort c_0, and $g(p)$ is the log rate ratio relative to the age-cohort prediction and can be interpreted as the residual rate ratio. The value of *h(c)* is set zero for the reference cohort, and the average values of *h(c)* and *g(p)* over the fitted period are approximately zero (i.e. the average value of the relative risks are approximately one). The common linear "drift" component (on the log scale) has been included, as conventionally done, in the cohort component. The models were fitted using the Epi package in R (Carstensen, 2006). The previous function may be written as:

$$m_{apc} = \exp(f(a))\exp(g(p))\exp(h(c)).$$

We also fit separate models to lung cancer mortality rates, m^{lc}_{apc}, as follows:

$$\ln(m^{lc}_{apc}) = f_{lc}(a) + g_{lc}(p) + h_{lc}(c)$$

and similarly for COPD mortality rates m^{copd}_{apc}.

The cohort function for lung cancer, $h_{lc}(c)$, may be used as an index of the time trend of total smoking attributable mortality relative the value for the 1900 cohort, although cancer accounts for only about one third of tobacco-related deaths, with approximately one third due to respiratory causes and one third to cardiovascular diseases (Vineis 2008). Lung cancer is a particularly useful cause of death for analysis because there is a particularly close relationship between smoking and lung cancer (Lopez, Collishaw, and Piha, 1994; Knoke et al., 2004) and it is essentially unaffected by other risk factors in highly developed societies (in countries such as China, other factors such as use of cooking fires may also be important). Therefore lung cancer mortality may be used to index change in total smoking related mortality of successive cohorts. Rates of change of smoking-related mortality are assumed to be proportional to rates of change in the cohort function as long as the proportion of smoking-attributable mortality due to lung cancer remains largely constant over time. We use the first derivative for the identification of the most- and least -favoured cohorts.

The difference between tobacco-related mortality estimated from annual changes in lung cancer and overall mortality by cohort will be used to assess the contribution of changing patterns of smoking on overall mortality with special emphasis of experiences of the men and women of the "Golden generations".

Results

Figure 1 shows the proportion of deaths caused by smoking for selected causes of death in developed countries in 2000. Smoking is responsible for almost 87% of lung cancer deaths (91% for men), followed by 73% for COPD, and upper aerodigestive cancer (55%). The estimates for the other four groups – other cancers, other respiratory, vascular diseases, and other medical – are around 10%.

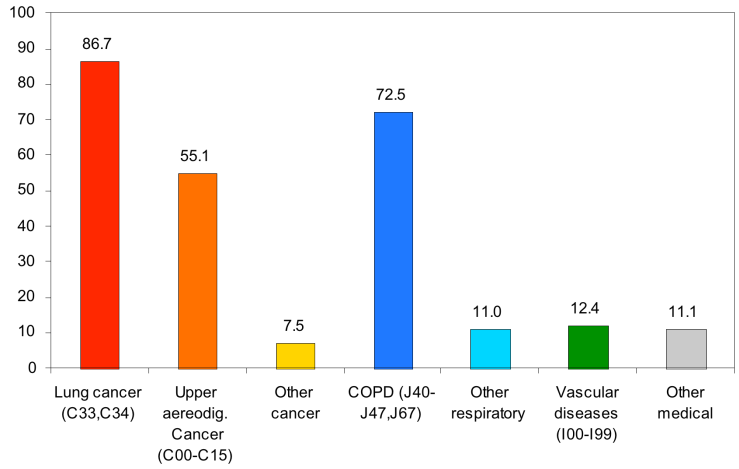

Figure 1 – Proportion of deaths in different causes due to smoking, 2000

Source: Peto, R., A.D. Lopez, J. Boreham, M. Thun, C. Heath (1992). Mortality from tobacco in Developed countries: indirect estimation from national vital statistics. Lancet 339:1268-78

Although lung cancer is so strongly linked to smoking, the number of smoking-related deaths is similar to that for vascular diseases (for which smoking is responsible for just 12% of assigned deaths), followed by COPD (18%) (Figure 2). A lower proportion of smoking-related deaths among women are due to lung cancer, but higher proportions for vascular, COPD and respiratory diseases. Moreover, additional gender-age differences the distribution of causes exist between those aged 35-69 years and 70 years and over: in the first group, lung cancer accounts for 33% for both sexes, while among older people there is a gap of 10% between men and women (27.4% for men and 17.8% for women) and a reversed 7% absolute gap in vascular diseases (22.7% for men and 29.6% for women).

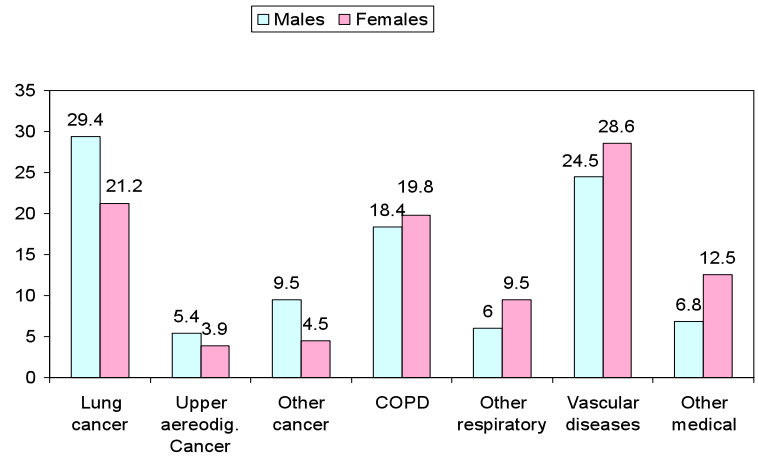

Figure 2 – Distribution of smoking related deaths by cause, 2000

Source: Peto, R., A.D. Lopez, J. Boreham, M. Thun, C. Heath (1992). Mortality from tobacco in Developed countries: indirect estimation from national vital statistics. Lancet 339:1268-78

The results of the APC model for lung cancer and COPD are shown in Figures 3a and 3b for cohorts born in years 1865 to 1972 and for time periods 1950 to 2007. The functions are fitted as smooth cubic splines to identify the main trends.

The age specific rates for the reference cohort born in 1900, $\exp(f(a))$, (the first pair of curves on the left side of both figures) shows how the death rate for lung cancer and COPD increases with age. Note that men have death rates that are almost ten times those of women for lung cancer, and five times for COPD, which has implications for the magnitude of potential impact of smoking-related mortality for men and women.

The rate ratio for the period, $\exp(g(p))$ (last pair of curves on the right side of the figures) suggests a relatively small time period effect (note that the average slope of the curve is close to zero since drift is attributed to the cohort component, what is important is that the slope of the period rate ratio is close to zero everywhere); for lung cancer, this period rate ratio is essentially constant over the whole period (1950-2007). For COPD, a slight non-linear period effect is visible but which is irrelevant when compared with non-linear changes in the cohort function (shown as the pair of curves in the middle of the figures). (Note that in this formulation of the APC model, the common linear drift component is included with the cohort function, so the relevant comparison is with the non-linear part of the cohort function.)

For both causes of death analysed, the cohort component represented by the cohort rate ratio $\exp(h(c))$ shows very similar patterns: for men (red line) the rate ratio (fixed at 1 for the cohort born in 1900) shows a peak corresponding to the generations born in period 1900-1910, while the peak for women is observed among the cohorts born around 1920-1930, reflecting the later uptake of smoking by women than by men (IMASS, n.d.).

From the beginning of the 20th century the consumption of manufactured cigarettes increased rapidly (replacing to some extent use of pipes or cigars). Men born around the end of the Nineteenth century were aged around 20 during the First World War when provision of low cost cigarettes for conscripts was widespread and the smoking habit of these generations was adopted early in life and consequently most of these men and the immediately following generations became heavy and persistent smokers.

(a) Lung (b) COPD

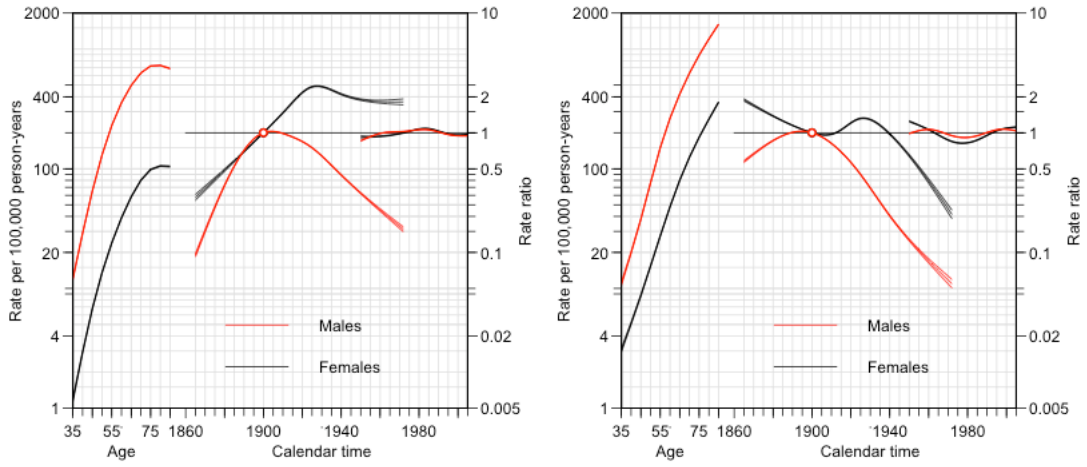

Figure 3 – APC model for (a) lung cancer and (b) COPD diseases, mortality rate per 100,000 person-years (age component) and rate ratio (cohort and period component)

Women's up-take of smoking followed men's, but with a 20 year delay and at a lower level of intensity, with differences in duration, number of cigarettes per day, and degree of inhalation (Burger and Gochfeld, 1989; Prescott, Scharling, and Osler, 2002). The peak in the 1900-1910 (men) and 1920-1930 (women) generations is visible by simply calculating the death rate for both causes by cohort instead of period (See Appendix

Figures 1 and 2), confirming that smoking patterns are very likely to have been the main driver of change in lung cancer and COPD mortality for both sexes.

The APC model is operationalised using spline models and therefore the first derivatives of the cohort functions in Figure 3, which are obtained analytically, are shown in Figure 4. To analyze the rise and fall in lung cancer and COPD mortality, we concentrate on the values in Figure 4. The highest rate of mortality improvement occurs for the generations corresponding to the inflection point of the curves shown in Figure 3 when the second derivative changes from positive to negative, although this is easier to locate in Figure 4 as it at the minimum value of the curve. The cohorts where higher rates of improvement in lung cancer and COPD mortality are visible when compared with the previous and the subsequent cohorts for both men and women are found for those where the values shown in Figure 4 are negative, i.e. for women born around 1925-1945, the "Golden generations" and about 5 years earlier of men. For lung cancer, the minimum for men in Figure 4 is just before 1930 and for women, just after 1930 when lung cancer rates were declining at about 2% p.a. for men and 4% p.a. for women; for COPD among men, the minimum is around 1920, and in the early 1940s for women when similar maximum rates of improvement were observed. Secondly the improvement in the rate ratios for these causes of mortality is in general bigger for men than for women[1], which suggests that the effects on overall mortality by sex would differ.

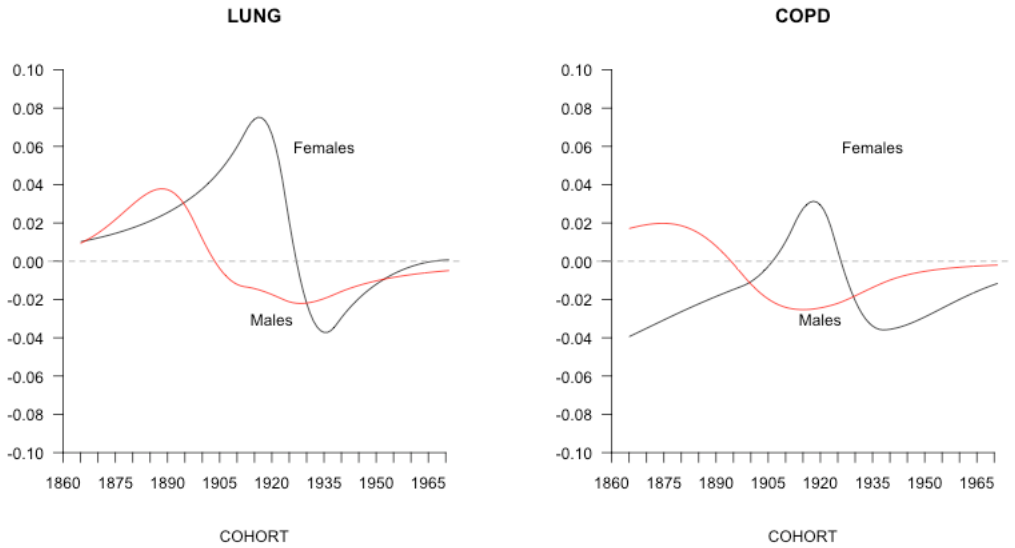

Figure 4 – First derivative of exp(*h(c)*), lung cancer and COPD models

[1] The absolute magnitude of cohort change for a particular cause depends both on the value of the mortality rate in question and how proportionately quickly it is changing. We show later that the first term is given by the first derivative of the cohort function. In the interpretation of the results of APC models based on rectangular data classified by age and time period, it is important to keep in mind that the estimates for both the oldest and youngest generations are based on very few data values so they could be strongly biased if the model is not correct.

The maximum increase in lung cancer mortality, about 8% p.a., was experienced by men born just before 1890, about 30 years before the peak for women. Thus while the timing and level of uptake of smoking was very different for men and women, the patterns of smoking reductions (both stopping and reductions in adoption of smoking) were much more similar, especially for lung cancer; the reasons presumably being due to the recognition of the health hazards of smoking by both sexes simultaneously, whereas the drivers of smoking adoption were much more sex-differentiated.

Can the mortality improvement of the "Golden generations" be explained by changing smoking patterns?

Causes of death largely driven by smoking habits, such as lung cancer and chronic obstructive pulmonary diseases, show peak levels for the generations of men born in 1900-1910 and for the generations of women born in 1920-1930 (Figure 3), but both sexes show the maximum rates of improvement from such causes for the cohorts born in the 1930s (Figure 4, see also Table 1 in Appendix).

Smoking differentials have played a central role in determining time trends and international differences in overall mortality, and remain the leading candidate for explaining the UK "Golden generations" mortality levels (Dunnell, 2009). We therefore apply a simple decomposition of overall mortality levels based on the approach above to estimate the proportion of change in overall adult mortality among cohorts, particularly the "Golden generations" that can be accounted for by changing patterns of smoking.

According to the APC decomposition the mortality rate for the cause $C1$ can be expressed as the product of the age function m_a for the rate ratio of the cohort RR_c and the rate ratio of the period RR_p:

$$m_{apc}^{C1} = m_a^{C1} RR_c^{C1} RR_p^{C1} \qquad (1)$$

The expression (1) can be simplified by assuming RR_p is approximately equal to 1, which is observed empirically for the causes analysed here when the common linear "drift" component is included in the cohort component (Figure 3), consequently

$$m_{apc}^{C1} = m_{apc}^{C1} RR_c^{C1} \qquad (2)$$

Taking logarithms and then differentiating equation (2) with respect to the cohort variable

$$\frac{1}{m_{apc}}\frac{\partial (m_{apc}^{C1})}{\partial c} = \frac{\partial \ln RR_c^{C1}}{\partial c} \tag{3}$$

$$\frac{\partial (m_{apc}^{C1})}{\partial c} = m_{apc}^{C1}\frac{\partial}{\partial c}\ln RR_c^{C1} \tag{4}$$

(Note that the above expression is independent of the time period, *p*.) The overall mortality rate m_{apc}^{T} can be decomposed as the sum of the lung cancer mortality rate m_{apc}^{LC} and non-lung cancer mortality rate $m_{apc}^{\overline{LC}}$

$$m_{apc}^{T} = m_{apc}^{LC} + m_{apc}^{\overline{LC}} \tag{5}$$

Differentiating with respect to the cohort variable, as shown in (4), equation (5) can be written as:

$$m_{apc}^{T}\frac{\partial}{\partial c}\ln RR_c^{T} = m_{apc}^{LC}\frac{\partial}{\partial c}\ln RR_c^{LC} + m_{apc}^{\overline{LC}}\frac{\partial}{\partial c}\ln RR_c^{\overline{LC}} \tag{6}$$

The assumption of RR_p constantly equal to one is valid for the overall mortality as well (see Appendix Figure 3). However, even though well over 80% of lung cancer mortality is smoking related, the majority of deaths caused by smoking result from among other recorded causes (Figures 1 and 2). Therefore lung cancer deaths are insufficient to explain completely the annual variation in overall mortality level. Denoting all smoking related mortality by SR, (6) can be re-written as:

$$m_{apc}^{T}\frac{\partial}{\partial c}\ln RR_c^{T} = m_{apc}^{SR}\frac{\partial}{\partial c}\ln RR_c^{SR} + m_{apc}^{\overline{SR}}\frac{\partial}{\partial c}\ln RR_c^{\overline{SR}} \tag{7}$$

We assume that

$$m_{apc}^{SR} = k_c m_{apc}^{LC} \text{ where } k_c \text{ is approximately constant i.e. } \frac{d}{dc}k_c \approx 0 \tag{8}$$

Thus (7) may be written as

$$m_{apc}^{T}\frac{\partial}{\partial c}\ln RR_c^{T} = m_{apc}^{SR}\frac{\partial}{\partial c}\ln RR_c^{LC} + m_{apc}^{\overline{SR}}\frac{\partial}{\partial c}\ln RR_c^{\overline{SR}} \tag{9}$$

Equation (9) suggests that the closer the patterns of the first derivatives of the overall mortality and smoking related rates, the greater the potential for explaining change in the overall mortality level by the change in smoking related mortality (and therefore by the change in smoking patterns). In fact, the final component of equation (9) should be approximately linear or constant if non-linear mortality changes in overall mortality were due to changes in smoking behaviours (note that the final component of equation (6) would contain a substantial smoking related component and would therefore not be linear/constant).

To check the validity of our assumptions (i.e. the decomposition in equation (9)), the overall mortality rate has been decomposed using different subgroups of causes of deaths summing up the total number of deaths. Results (see Appendix Figure 4) suggest that the first derivatives for both, the total deaths and the sum of subgroups, are almost the same (differences may be due to the fact that the assumption of constant period effects that for some cause of deaths may not be valid).

Figure 5 shows the first derivatives of the overall mortality and lung cancer mortality rates per 100,000. As expected, for both men and women, the annual change in lung cancer mortality rates do not explain the substantial change in overall mortality rates for the cohorts born around 1930s.

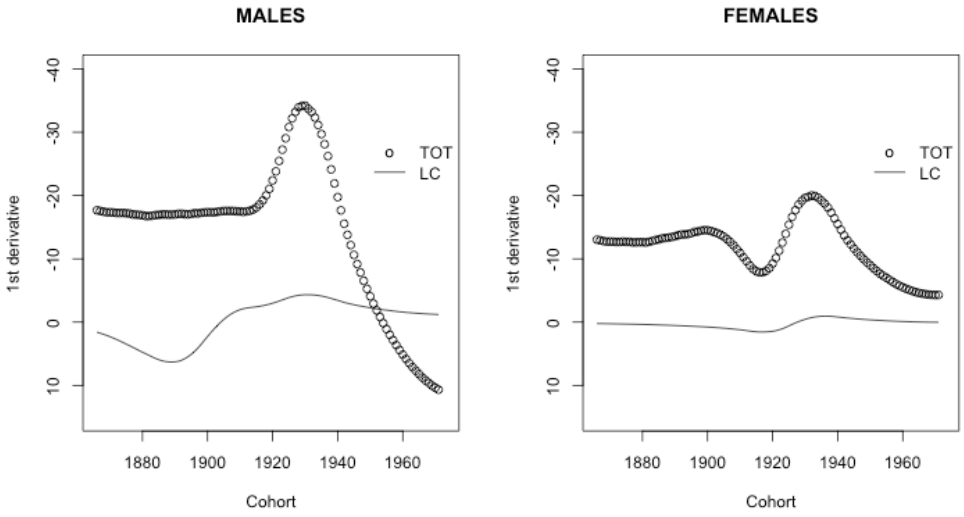

Figure 5 – Annual change in cohort overall (TOT) and lung cancer (LC) mortality rate, Males and Females.

Note: y-axis shows negative changes

For this reason, three other estimates of the potential burden of smoking related mortality functions (see Appendix Table 2) have been plotted in Figure 6 (for simplicity, we assume the same form for men and women), together with the first derivative of the cohort overall mortality rate (dotted points) and lung cancer (dashed line) as follows:

1. The solid black points (0.8LC+0.2C+0.7COPD) represents the annual change (i.e. first derivative) in the cohort smoking mortality rate calculated as proportion of lung cancer (80% of deaths), cardiovascular diseases (20% of deaths), and chronic obstructive pulmonary diseases (70% of deaths);
2. The solid line (LC+COPD+O+IHD+A) represents the annual change in the cohort smoking mortality rate calculated as the sum of lung cancer, chronic obstructive pulmonary diseases, oral cancer, ischemic heart diseases, and arteriosclerosis;
3. The dotted line (C) is the annual change in cohort cardiovascular mortality rate.

For both men and women, the change in smoking related mortality using formula 1. above (solid dotted points in figure 6) does not substantially help to explain overall annual cohort mortality change. The solid line using formula 2. above represents the most "generous" option, in that the total annual change in lung cancer, chronic obstructive pulmonary diseases, oral cancer, ischemic heart diseases, and arteriosclerosis are considered to be smoking related, i.e. the annual change due to causes of death other than smoking related (the difference between the first derivative of the overall mortality rate and the combination of the five chosen causes of deaths) is smaller. Moreover, this formulation more closely follows the trend observed for the overall mortality variation, with a distinctive improvement in mortality among the cohorts born around 1930s. However the gender comparison shows that for females this option fits better. The gap between the solid black points and the solid line is small for women.

Finally the annual change in cardiovascular diseases (dotted line) is the one that best approximates the annual change in the overall mortality. For men the function is almost identical to the function shaped by the "generous" option (lung cancer, chronic obstructive pulmonary diseases, oral cancer, ischemic heart diseases, and arteriosclerosis). For women the change in overall mortality may be mostly accounted for by changing in cardiovascular mortality. This suggests that for both sexes the annual change in cardiovascular mortality itself accounts for most of the overall annual change.

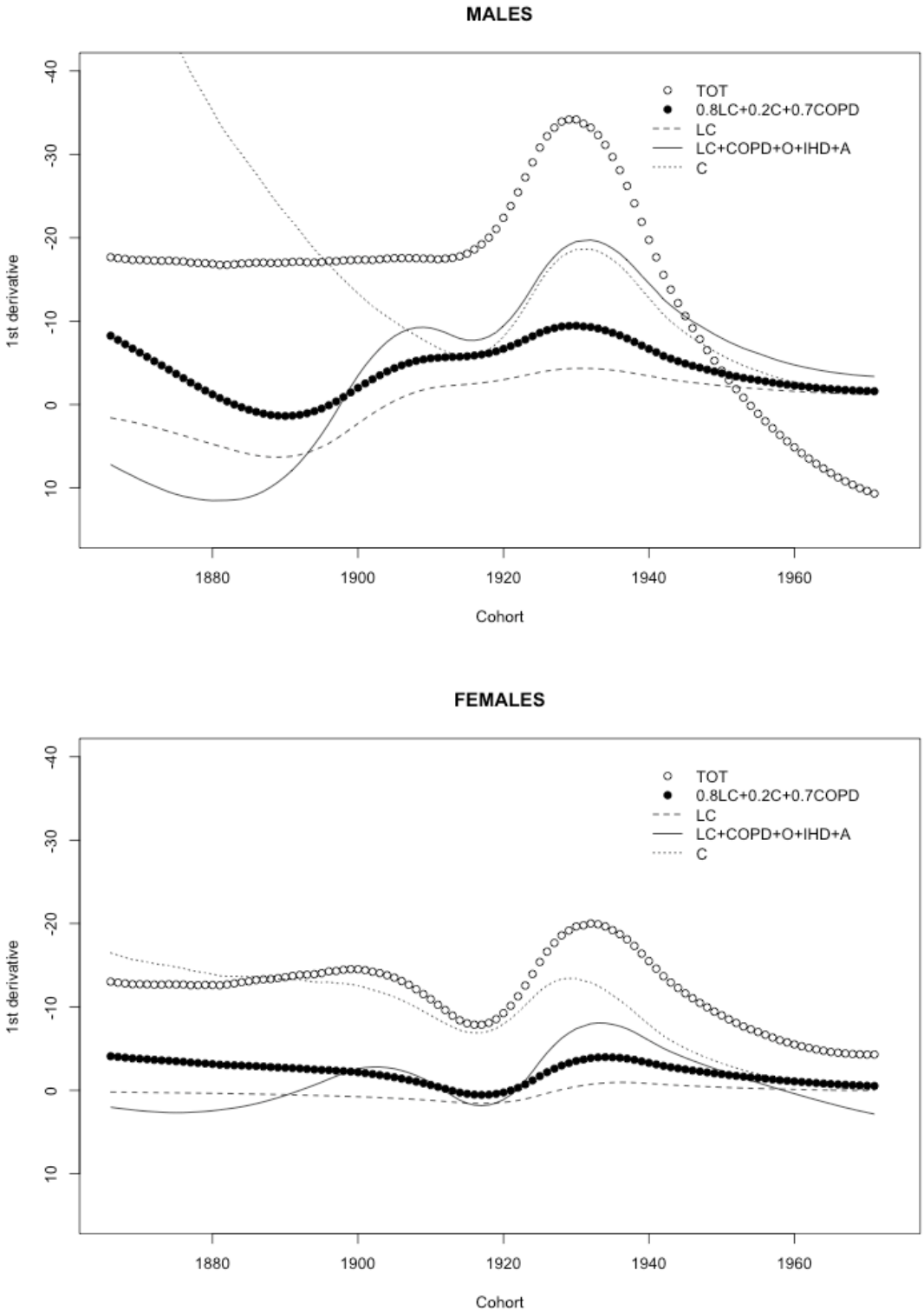

Figure 6 – Annual change in cohort rates for different sets of causes, Males and Females.

Note: y-axis shows negative changes.

Conclusions and Summary

The methods available for the estimation of the proportion of deaths attributable to smoking are largely based on period approaches (Peto and Lopez, 1992; Preston et al. 2009), even though it is widely recognized that smoking related mortality is strongly influenced by cohort smoking behaviours. In this paper the analysis of cohort patterns of mortality for lung cancer and COPD (two causes of deaths highly dependent on individuals' smoking history) shows the impact of smoking patterns more clearly by separating out the role of period and cohort factors. In particular, we use lung cancer to index the magnitude of cohort smoking related mortality. The APC model applied to lung cancer and COPD deaths from 1950 to 2007 among people aged 35 years and over shows a clear cohort pattern. Males born during the first decade of the past century and females born in the 1920s present the highest level of lung cancer mortality, reflecting these generations smoking habits – massive introduction of cigarettes on the market during the first decades of the past century, cheap cigarettes provision for conscripts, and adoption of smoking habits early in life. Despite this gender gap in the peak of cohort lung cancer mortality, the highest rates of mortality improvement were experienced by the 1930s cohorts for both men and women, the so-called "Golden generations". However, this association is not sufficient to conclude that the above-average mortality improvement for the 1930s generations was due to smoking patterns. The second part of the paper focuses on disentangling how much of the overall mortality improvement could be explained by changes in smoking patterns by considering a wider set of mortality causes that are plausibly related to smoking. The results show that annual changes in cardiovascular mortality rates better explain changes in the overall mortality than the other options considered. However, since we are unable, at this stage, to distinguish between smoking and non-smoking related cardiovascular deaths, the good fit may arise in part from the fact that any cause or set of causes, such as cardiovascular diseases (Appendix Table 2), that account for a large fraction of overall deaths are likely to show a positive relationship with overall change rather than necessarily reflecting change in smoking patterns.

A cohort perspective overcomes some of the problems that lung cancer deaths tend to lag use of tobacco, since for cohorts, it should provide a simultaneous indicator of smoking burden. Cohorts are particularly important since unlike period approaches, they provide insight into possible future changes in mortality and hence may be used to elucidate the validity of population projections.

We show that variation in smoking related mortality in Britain in recent decades can be well-summarised by an age-cohort model rather than a full age-period-cohort model which leads to considerable simplification of analysis of trends. We argue that interest is frequently in changes in mortality rather than absolute levels and that, for example, this is more determined by changes in behaviours rather than by levels of, for example, smoking. We show that analysis of the timing of turning points in changes provides additional insight into drivers of change.

The limitations of this preliminary study should be noted. We have used recorded underlying cause of death data which are subject to changing conventions of classification; lung cancer, in particular, may be less accurately classified for the "old old". We have assumed the relationship between lung cancer and total smoking related mortality has been constant over tine – although this is probably more reasonable for cohort data than for period data approaches, this is an area that would benefit from further investigation, as would use of multiple cause of death coded data. We have also assumed for simplicity that the relationship of lung cancer and smoking related mortality is the same for both men and women, although the patterns are somewhat different. While we have decomposed overall mortality into smoking related and non-smoking related mortality, it should also be noted that we have not computed estimates of cause-deleted non-smoking mortality.

While bearing these caveats in mind, nevertheless, the analysis of cohort patterns of mortality for lung cancer and COPD, show more clearly the impact of smoking patterns by separating out the role of period and cohort factors, suggesting that the best way to analyze smoking related cause of death is to incorporate a cohort perspective.

Future analysis will include the estimation of a new set of decompositions based on Peto and Lopez estimation of smoking related proportion of deaths among all possible causes of deaths, to assess the relationship between annual change in lung cancer rates and a wider set of smoking related causes of deaths.

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Appendix

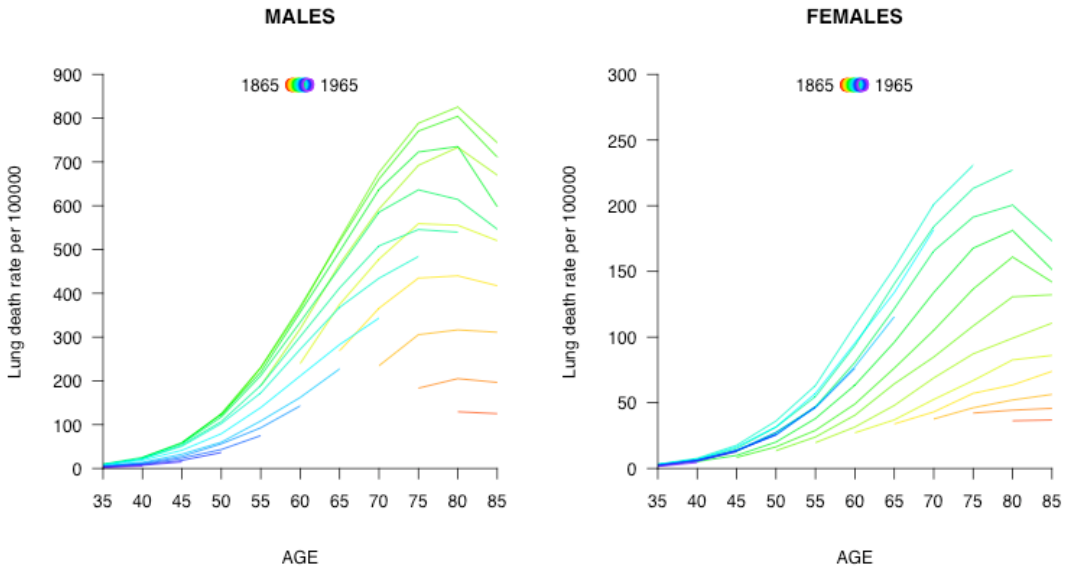
	Max in RR_C[(1)]	
	Males	Females
Lung Cancer (LC)	1904	1928
Chronic Obstructive Pulmonary Diseases COPD)	1900	1926

(1): after 1900

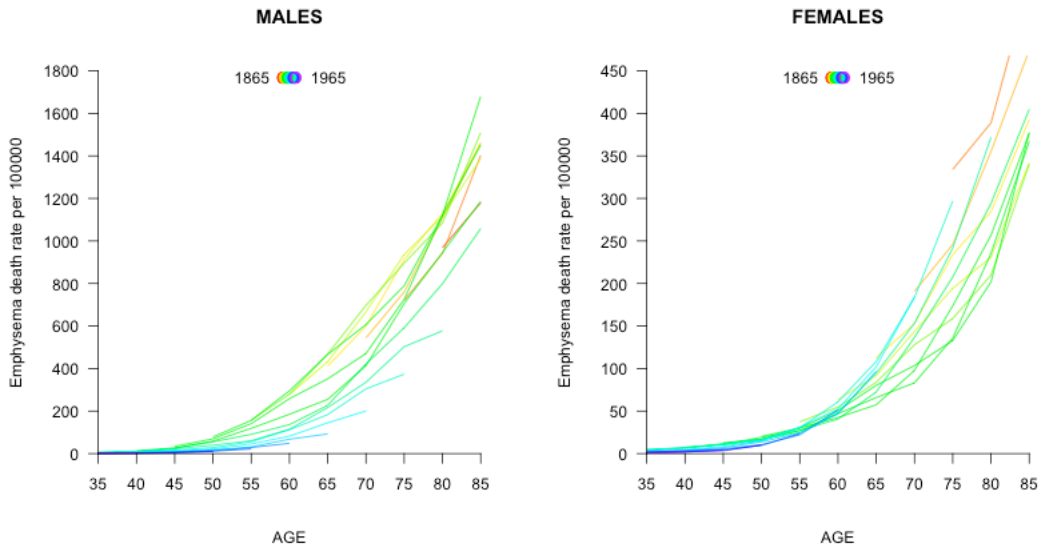
Table 1 – Year maximum value of RRc function achieved, by sex and cause

	ICD10	Proportion (%) of total deaths (aged 35+ yrs)			
		1950		2007	
		M	F	M	F
Lung Cancer (LC)	C33-C34	4.3	0.9	7.3	4.9
Cardiovascular diseases (C)	I00-I99	50	57	35	34
Chronic Obstructive Pulmonary Diseases COPD)	J40-J47, J67	7.2	4.1	5.4	5.0
Oral Cancer (O)	C00-C14	0.7	0.3	0.5	0.2
Ischemic Heart Diseases (IHD)	I20-I25	14.7	8.7	19.2	13.5
Arteriosclerosis (ART)	I70	2.4	2.6	0.1	0.2
0.8LC+0.2C+0.7COPD		18.5	15.0	16.6	14.2
LC+COPD+O+IHD+A		29.3	16.6	32.5	23.8

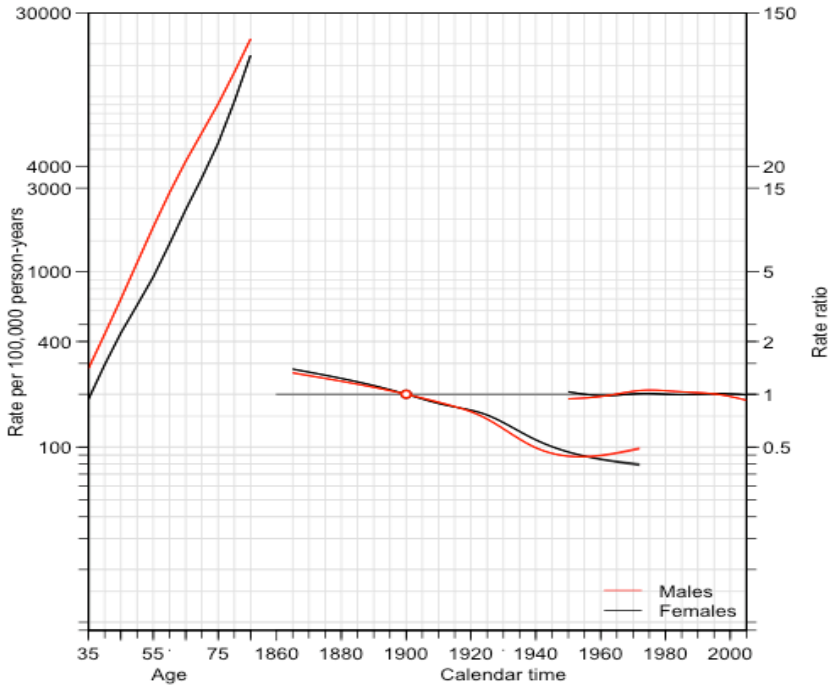
Table 2 – ICD10 codes, and proportion of death in specific years



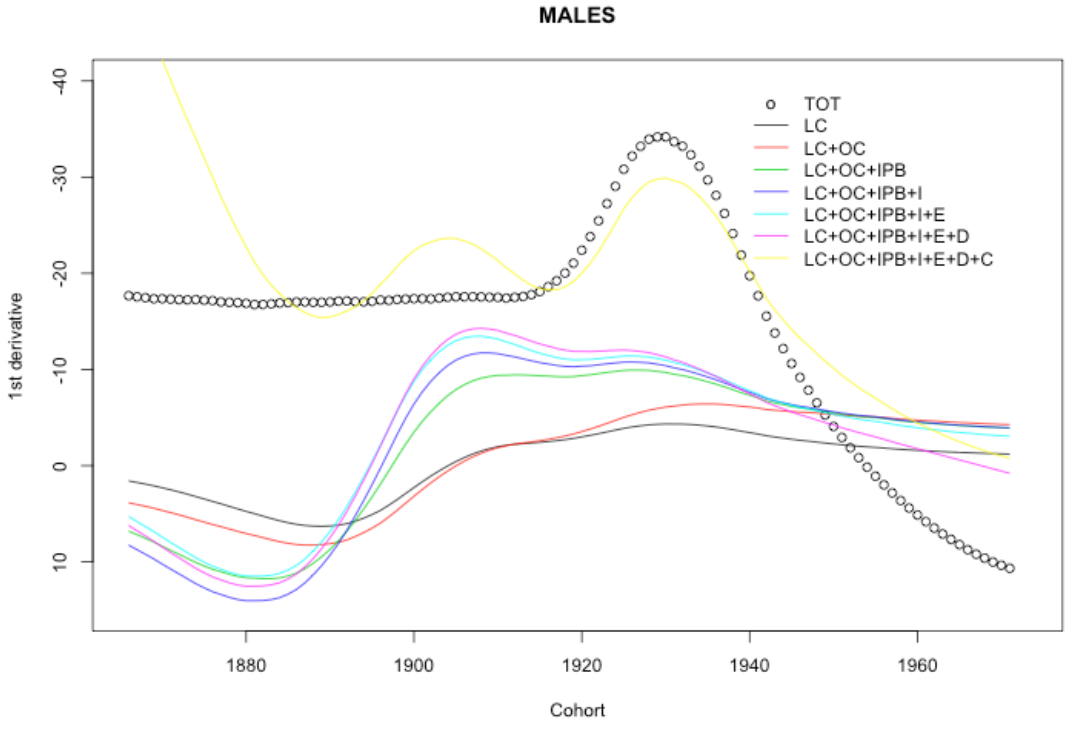
Appendix Figure 1 – Death rates by age and cohort, Lung cancer

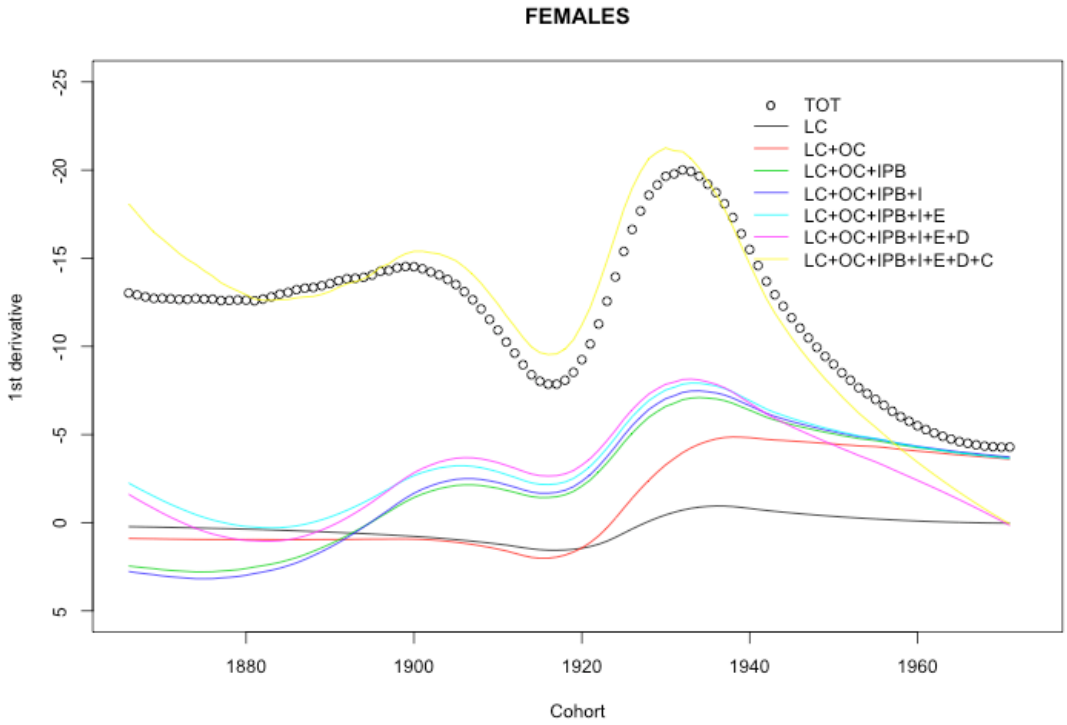


Appendix Figure 2 – Death rates by age and cohort, COPD



Appendix Figure 3 - APC model for overall mortality: mortality rate (age component) and rate ratio (cohort and period component)





Appendix Figure 4 – Annual change in cohort overall (TOT) and subgroup mortality rates, Males and Females.

Notes: LC: lung cancer; OC: oral cancer; IPB: Influenza, Pneumonia, Bronchitis; I: Infectious diseases; E: External causes; D: Degenerative diseases; C: Cardiovascular diseases.

y-axis shows negative changes.