MEMORANDUM

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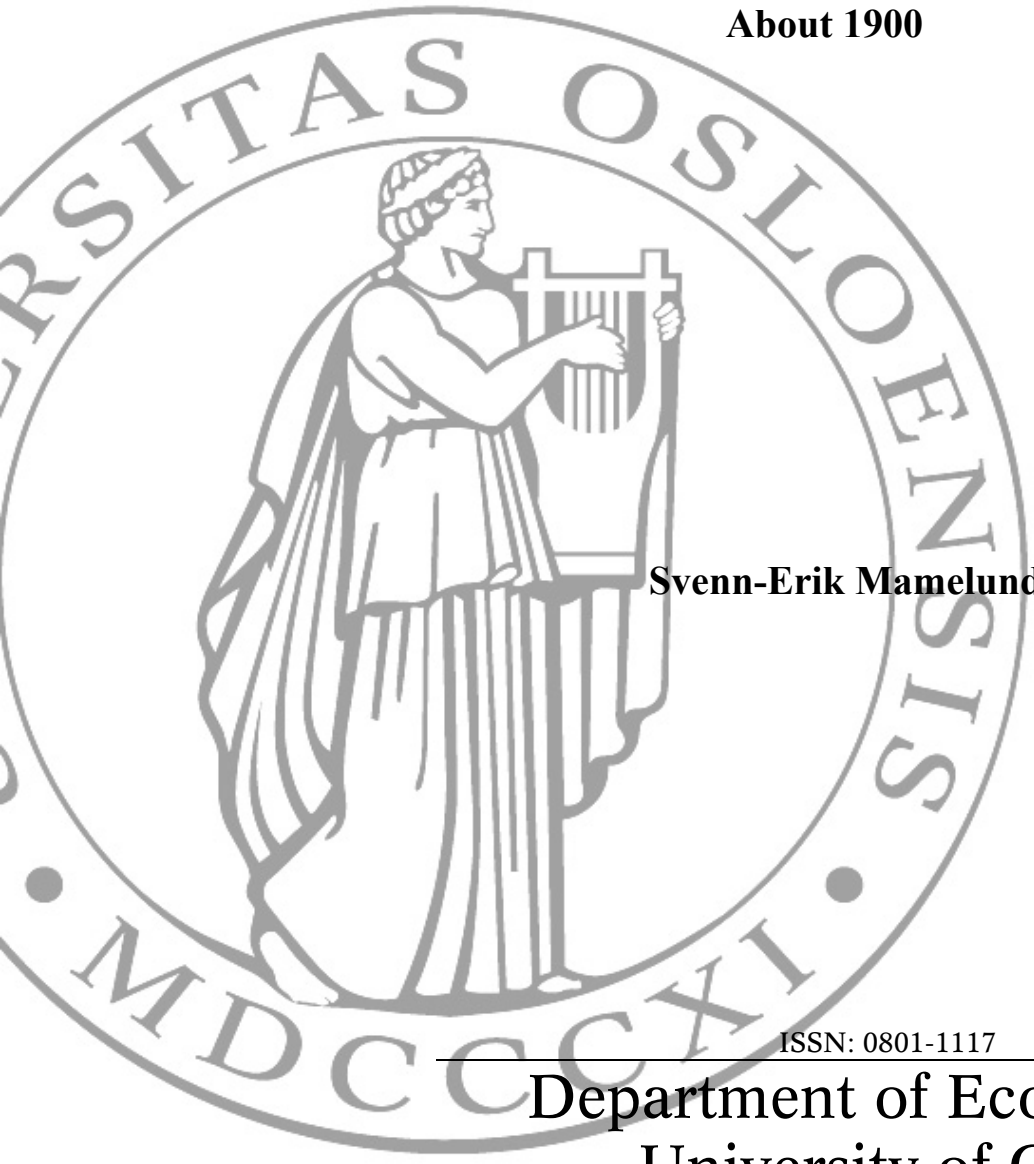
Effects of the Spanish Influenza Pandemic of 1918-19 on Later Life Mortality of Norwegian Cohorts Born About 1900



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Effects of the Spanish Influenza Pandemic of 1918-19 on Later Life Mortality of Norwegian Cohorts Born About 1900

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Abstract

By using Age-Period-Cohort analysis the paper shows that Norwegian male and female cohorts born about 1900 have experienced significantly higher all-cause mortality in middle and old ages relative to "neighbor" cohorts. In a widely cited study, Horiuchi suggests that only males from belligerent countries who were adolescents during WW I exhibit this cohort effect. The finding in this paper demonstrates that Horiuchi's explanation may be incomplete. The search for explanations for neutral Norway must clearly go beyond the direct (soldiers wounded physically and mentally) and indirect effects (rationing of food) of WW I on later life mortality. This paper suggests that Spanish Influenza 1918-19 is the most important of several possible factors priming the Norwegian cohorts. A large proportion of the cohorts considered contracted Spanish Influenza, but only a small proportion died of it immediately. The net effect on later life mortality is thus assumed to be that of debilitation.

Keywords: Spanish Influenza 1918-19; morbidity; mortality; debilitation; selection; APC-analysis; cohort effects; Norway

1. Introduction

There are studies that have found that German, Austrian, Italian, French, and Polish cohorts that went through adolescence 1914-18 and young adult cohorts directly engaged in WW I (primarily men) experienced elevated all-cause mortality in middle and old ages compared to older and younger cohorts (Horiuchi 1983; Boleslawski 1985; Caselli et al. 1987; Caselli and Capocaccia 1989; Caselli 1990; Wilmoth et al. 1990). The authors basically attributed their findings to indirect (shortage of food, malnutrition, and poor hygiene) and direct (physical and mental scars of combat) long-term effects of WW I. Horiuchi (1983) analyzed the mortality in belligerent Germany, Austria and, France as well as neutral Sweden and Japan. The analysis showed that only men born about 1900 from Germany, Austria and France exhibited relative high mortality in middle and old life. Horiuchi therefore concluded that the cohort variation in mortality most likely was a long-term effect of the WW I, specifically a result of malnutrition. Gómez de León (1991), however, found that in neutral Norway the same male and female cohorts were debilitated relative to younger and older cohorts. What could be the explanations for neutral Norway? The most obvious is neither direct nor indirect effects of WW I. Gómez de León (1991) was tempted to explain the cohort differentiation by Spanish Influenza 1918-19 "but without a clear understanding of the nature of the mechanisms involved, [he found it] adventurous to attribute them directly to any particular form of determinacy" (p. 81). One other explanation suggested by Gómez de León, surprisingly not mentioned by other authors, is that a late wave of predominantly male overseas emigration, which peaked in the mid 1920s, might have introduced additional selection if the most selective of the survivors left. Gómez de León showed, although he did not state it, that Horiuchi's (1983) hypothesis that only men from belligerent countries exhibit this cohort effect was wrong. A fourth explanation, in addition to the war 1914-1918, Spanish Influenza 1918-19, and a large wave of emigration in the mid 1920s, of the differences in cohort mortality in belligerent and neutral Europe, is cigarette smoking which became increasingly popular during and after WW I, especially for men (Horiuchi 1983). The four events occurred when the cohorts born about 1900 was adolescents and young adults, and they may all be important predictors for the mortality patterns they experienced in middle and old life in the latter half of the twentieth century.

The aim of the present paper is to have a second look at the Norwegian case, and to challenge Horiuchi's WW I hypothesis by following up Gómez de León's Spanish Influenza hypothesis. To do this, information on the immediate and long-term effects of Spanish Influenza on health and mortality is needed. A review of the literature regarding this issue is presented in section 2. The results of the analysis are presented in section 3. To compare the results for neutral Norway with Horiuchi's for belligerent Germany, Austria, and France, a

replication of exactly what Horiuchi did to separate the cohort from the age and period effects is pursued; First, estimation of period rate of mortality change with age, and second estimation of an Age-Period-Cohort (APC) regression model with dummy variables. Results are also presented estimating refined and extended versions of the descriptive and statistical tools applied by Horiuchi. Although it is believed that neutral Norway fits well as a test case to study effects of Spanish Influenza on later life mortality as it may counterbalance effects of WW I, in section 4 it is discussed whether shortage (1914-19) and rationing (1918-19) of food during and shortly after WW I (section 4.1) and possible post-traumatic stress disorders among the Norwegian neutrality-keeping soldiers and seamen in the merchant fleet, who survived WW I (section 4.2), may confound the analysis. Also, effects of mass-emigration on mortality (section 4.3) and cigarette smoking (sections 2.2, 3.2, and 3.3) are discussed as a possible confounding factors.

2. Spanish Influenza 1918-19 and post recovery-problems

2.1. The incidence and mortality of Spanish Influenza 1918-19

Spanish Influenza affected at least 500 million globally or over one fourth of the population (Laidlaw 1935). It killed 50-100 million people in less than a year (Johnson and Mueller 2002) ─ five to ten times the death toll of more than four years of war. Unlike other influenza epidemics or pandemics, the bulk of excess mortality occurred among persons 20-40 years old. The Spanish Influenza affected 1.2 million Norwegians or little less than half of the population and it took the life of 15,000 people or 5.7 lives per 1 000 (Mamelund 1998). The pandemic appeared in three bouts; the first during the summer of 1918, the second during the fall of 1918, and the third during the first months of 1919. The excess all-cause death rate during the highly virulent second bout from October to December 1918 (8.6 deaths per 1 000) was twelve times as high as the corresponding death rate during the first bout of influenza from July to September 1918 (0.7 deaths per 1 000). Mortality during the third bout of influenza was relatively low. Spanish Influenza was so serious because of the bacterial complications, mainly pneumonia, but also meningitis, bronchitis and acute diarrhea (Mamelund 1998). A little more than one per cent of those who were infected by the disease in Norway died. Although this lethality may seem relatively low, it is the highest registered lethality of the four influenza pandemics that came to Norway in the twentieth century (Mamelund and Iversen 2000).

[Figure 1 approximately here]

The influenza-censuses carried out in 1918-19 for a number of cities in the United States and England, and for the city of Bergen, Norway, give a fairly reliable picture of the age-sex pattern of influenza incidence (Great Britain Ministry of Health 1920; Vaughan 1921; Hanssen 1923; Collins 1931; Sydenstricker 1931; Britten 1932). These studies clearly show an unusually high influenza incidence during the summer wave of 1918 for those between 10-39 years of age, especially men, and a rapidly falling incidence by age for those above 40 years (see Figure 1 for Bergen, Norway). The age-specific incidence curve during the fall wave was similar to that seen during the summer wave, but those hardest hit during the first wave seem to have been less hit during the second wave in 1918, probably due to relative immunity acquired. The cross over in sex-difference in incidence at ages 10-39 is therefore also apparent. The W-shaped age pattern of influenza and pneumonia death rates for Norway 1918-19 presented in Figure 2 is representative for the global picture. However, the average influenza and pneumonia death rates for the two years 1918-19 stand in great contrast to the U-shaped age distribution of influenza and pneumonia death rates for the average of the two non-pandemic years of 1917 and 1921 (Figure 2). The relatively high lethality and the peculiar excess age-specific influenza and pneumonia mortality of Spanish Influenza have received substantial research attention. Despite recent and extensive molecular and paleomicrobiological research efforts, however, these issues are still a mystery (Basler et al. 2001; Davis et al. 1999; Gibbs et al. 2001; Reid et al. 1999, 2000, 2001, 2002; Tauenberger et al. 1997, 2001; Tumpey et al. 2002). Those born about 1900 as was identified as high mortality cohorts by Gómez de León (1991) are represented among those with the highest influenza incidence (Figure 1). The Norwegian cohorts born 1904-09 (10-14 years 1918-19), 1899-1904 (15-19 years 1918-19), however, experienced relatively low mortality in 1918-19 compared to the cohorts born 1880-1899 (20-40 years 1918-19) (Figure 2). The disease thus marked a large proportion of the cohorts born 1899-09, but only a small proportion died of it immediately (see Figure 3). In other words, the cohorts that were adolescent in 1918 and 1919 may have experienced large debilitation effects of morbidity and small selection effects of mortality due to Spanish Influenza. The interplay of the two mechanisms is assumed to lead to a net or average cohort effect caused by high relative mortality in later life.

[Figures 2 and 3 approximately here]

The young adults, those born 1890-99 (20-29 years 1918-19), however, experienced high incidences and death rates in 1918-19. In this situation there are two effects that work in opposite directions. On the one hand it is likely that those with the poorest health died while those with the best health survived. This is assumed to result in low future mortality for the cohorts considered. On the other hand it is possible that Spanish Influenza weakened the

average survivors compared to the original cohort. This is assumed to give higher future cohort mortality. The net cohort effect for the 1890-99 cohort may therefore not necessarily deviate from that of neighbor cohorts as the effects of selection may have cancelled out the effects of debilitation.

2.2. Later-life health and mortality of Spanish Influenza survivors after 1918

Spanish Influenza survivors were reported to have problems with sleeping, depressions, mental distractions, low blood pressure, dizziness and to cope at work and with everyday life for weeks, months or even years after 1918-19 (Mamelund 1998). According to data from the Norwegian asylum hospitals (calculated from Medisinaldirektøren 1916, 1917, 1920a-b, 1921, 1923a-b, 1924,1925a-c, 1927, 1928, 1929, 1930, and 1931), there was an excess in the number of first time hospitalized patients with mental diseases caused by influenza and pneumonia each year from 1918 to 1923 when compared to the average of the years 1915-17 and 1924-26 (80 men and 78 women). The number of persons, who suffered mental distractions after Spanish Influenza, is probably much higher than this calculation show as it is likely that people affected by milder or temporary post influenza melancholia did not see a psychiatrist. Hepatitis, ear illnesses, deafness, blindness, and baldness (esp. young girls) are other after-effects that have been linked to Spanish Influenza. It has also been reported that one-third of the influenza survivors have experienced heart problems, lung tuberculosis and kidney disease in later life (Collier 1974).

Those who struggled with one or several post-recovery problems and diseases associated with Spanish Influenza may have experienced higher mortality immediately in 1918-19 or later in life. There are at least three examples from the literature that gives support to this view: First, Wasserman (1992) has found that excess in influenza death rates 1918-20 was significantly and positively related to suicide in the United States independent of factors like alcohol consumption and the number of casualties during WW I. The suggested explanations were a drop in social integration (closing of schools, churches, theatres, banning of large public meetings and so on) and fear induced by the pandemic (the infected could die within three days). Several cases of suicide may also have occurred after 1920 either due to the above-mentioned psychological health problems of some survivors (direct effect) or to the unbearable loss of a spouse, children or close relatives (indirect effect) (Rice 1988; Crosby 1989; Phillips 1990).

The second example of higher later-life mortality of Spanish Influenza survivors that may be an after-effect of Spanish influenza is mortality associated with the Sleeping Sickness Pandemic (encephalitis lethargica or VON ECONOMOS's disease) of 1919-28. The Sleeping Sickness Pandemic caused hundreds of survivors to slip into a bizarre rigid paralysis with similarities to advanced Parkinson's disease. These patients, only occasionally able to

communicate or move, were nearly all institutionalized for life (Sacks 1999). It was largely the work by Ravenholt and Foege (1982) that established the link between the Spanish Influenza, the Sleeping Sickness Pandemic and increased risk of developing and dying from Parkinson's disease in later life. The hypothesis of a causal link was built up on the basis of two observations. The first is that the pandemics seemed to share etiology. The fact that the incidence of Spanish Influenza (see Figure 1) and the Sleeping Sickness (see Lund 1997) was highest among adolescents and young adults (10-30 years), and that the incidence was higher in men than in women in both pandemics, support this view. The second of the observations are that the Sleeping Sickness Pandemic followed the Spanish Influenza Pandemic in time and space. The time sequence given by Ravenholt and Foege (1982), however, has recently been questioned. In a review of the literature, Casals et al. (1998) found reports of Sleeping Sickness in several European countries three years before Spanish Influenza broke out in pandemic dimensions in 1918. Recent genetic as well as archival research, however, now also place the origin of the Spanish Influenza virus and the first influenza cases around 1915 (Reid et al. 1999; Oxford et al. 1999; Shortridge 1999). This gives additional support to the view that the Sleeping Sickness Pandemic was causally linked to Spanish Influenza.

Globally it is estimated that more than one million were infected and that half a million died from the Sleeping Sickness Pandemic in the period 1919-1928 (Ravenholt and Foege 1982). In Norway 268 cases and 52 deaths were reported (Mamelund 1999). When compared to the reported cases per 1 000 in the two neutral countries of Sweden (3.0) and Denmark (5.9) the figure for Norway (1.0) seems curiously small (Swedish and Danish figures are from Matheson Commission 1929). The likely explanation is diagnostic difficulties in Norway with a widespread population and shortage of doctors (Lund 1997). Poskanzer and Schwab (1963) analyzed 1 000 patients at Massachusetts General, the United States, 1920-1959, and found that those experiencing a severe case of the Sleeping Sickness developed Parkinsonism immediately, whereas those experiencing a mild or undiagnosed attack developed Parkinsonism later in life. For England and Wales, Martyn (1997) has found that the generation born 1889-1908 was two to three times as likely to die of Parkinson's disease in the period 1950-92 than those born before 1888 or after 1924. Ben-Shlomo et al. (1993) have found similar cohort-effects for the mortality of Parkinson's disease in the Republic of Ireland. There are no similar studies on mortality of Parkinson's disease in Norway.

The third example of relatively high later-life mortality of Spanish Influenza survivors that may be linked to Spanish Influenza is from coronary heart disease (CHD). Azambuja and Duncan (2002) using cross sectional data have found that Spanish Influenza mortality is a good predictor for the rise (1920-67) and the fall (1968-85) in CHD mortality in the United States. The analysis revealed that cohorts born about 1900, who had the highest

incidence and mortality of Spanish Influenza, also had the highest CHD mortality in later life. Preceding and succeeding cohorts had subsequently lower CHD mortality. The higher incidence of Spanish Influenza 1918-19 among men than among women was also used to explain why men always have had higher CHD mortality than women after 1920. The same temporal rise and fall in CHD mortality has also been observed in Norway. However, traditional risk factors like smoking, unhealthy diet, and little physical activity have hitherto been used to explain the cyclical mortality pattern (see for example Huserbråten 1993 or Aase and Storm-Furru 1996). Since traditional risk factors in most studies of CHD mortality do not explain more than half of the variance in CHD mortality, Azambuja and Duncan (2002) believes that including whether or not a person was exposed to Spanish Influenza in 1918-19 in the analysis will add more understanding to the pattern of CHD mortality.

3. Results

3.1. An empirical tool to trace cohort differentiation in mortality

Horiuchi (1983) formulated the hypothesis that only male members of cohorts who were adolescents and young adults in belligerent countries during WW I have experienced elevated mortality or debilitation relative to other cohorts as they passed through middle and old ages. The purpose of the analysis in sections 3.1 and 3.2 is to show that Horiuchi's hypothesis was wrong and that his explanation was incomplete. Hourichi explained the relatively high mortality of men born about 1900 in Germany by high mortality of cardiovascular diseases in middle and old age caused by malnutrition experienced in adolescence during WW I. The hypothesis proposed here is that Spanish Influenza debilitated the same cohorts of males and females in neutral Norway and that the Spanish Influenza hypothesis is more plausible than other possible explanations of the differences in cohort mortality like WW I, emigration, and cigarette smoking.

The first thing Horiuchi (1983) did to separate the effects of period from effects of cohort and age was to calculate the log of the period rate of mortality change K(x) from one five-year age group to the next (starting at age 40) for the single calendar years of 1959, 1964, 1969, and 1974. K(x) for a given calendar year is defined as

$$\mathrm{K(x)} = \frac{d\log(\mu(\mathrm{x}))}{dx}, \qquad (1)$$

where $\mu(\mathrm{x})$ is the force of mortality at exact age x. K(x) is estimated as

$$\hat{\mathrm{K}}(\mathrm{x}) = \log \mathrm{M(x+5)} - \log \mathrm{M(x)}, \qquad (2)$$

where M(x) is a five-year age specific death rate. After age 40, mortality increases relatively smoothly with age. Therefore, when a high-mortality cohort follows a low-mortality cohort, K(x) tends to be large. In the opposite case, K(x) tends to be small. Thus, right-way moving peaks and hollows in the K(x) curve for consecutive periods indicate that there are high mortality cohorts between the peak and the hollow. K(x) is calculated for all-cause mortality for males and females aged 40 and above for calendar years of 1959, 1964, 1969, and 1974 (data from Mamelund and Borgan 1996).

[Figures 4 and 5 approximately here]

For males, those born between 1895 and 1910 are identified as high mortality cohorts (using model (2)), seen by the downward turning and upward turning arrows in the period 1959-74 in Figure 4. K(x) curves estimated for smoothed one-year age groups confirm that that men born between 1895 and 1910 had higher mortality than neighbor cohorts, not only in the period 1959-1974, but also in the 1950s (results not shown). No striking cohort effect was found for other male cohorts or for the 1895-1910 cohorts before 1950 or after the mid 1970s. This is at odds with Horiuchi (1983) who suggested that this cohort effect is exclusive to men from belligerent countries. The K(x) curves for females presented in Figure 5, however, do not display visible cohort differentiation in mortality. This seems to be in accordance with what Horiuchi found for females in belligerent countries.

A weakness of K(x) is that it only can be used to detect age and cohort variation in the death rates. Thus, the effect of age and cohort on the variation in the death rates independent of *period* is not accounted for. This lead Horiuchi to apply a statistical method, (on data for men only), which controls for age, cohort, and period simultaneously. This is important, as cohort effects derived from a K(x) analysis may be different for different time periods.

3.2. A statistical tool to trace cohort differentiation in mortality

In this section, the assumptions and structure of the APC-model applied by Horiuchi (1983) are first described. Secondly, a refined and extended version of Horiuchi's model is presented. And thirdly, the results of both models are displayed.

Horiuchi (see pp. 81-84) wrote the log-transformed death rate M_{ij}, for each sex as

$$G_{ij} = \log M_{ij}, \qquad (3)$$

where i = 1,2,…,10 are five-year age groups 35-39 to 80-84 and j = 1,…,4 are the single calendar years 1959, 1964, 1969, and 1974. This implies that k = 1,…,13 are five-year cohorts born between 1870-74 and 1935-39, with k = j - i. The suggested model for G_{ij} was

$$G_{ij} = \alpha + \beta_i + \gamma_j + \delta_k + \varepsilon_{ij}, \qquad (4)$$

where α is a constant, and β, γ and δ are age, period and cohort effects respectively. It is the simplest APC model as the effects of age, period and cohort on G_{ij} are assumed to be additive, while there is no interaction effects. Horiuchi selected k = 13 as reference category for the cohort effects, i = 10 as reference category for the age effects, and j = 4 as the reference category for the period effects. In addition, he assumed that the effect for the next youngest cohort (1930-34, k = 12) was equal to that of the reference cohort (1935-39, k = 13). This way he broke the perfect linear relationship between age, period, and cohort, so that the model could be identified. The model was estimated using ordinary least squares (OLS).

By including only ages up to 84 years and thereby assuming that no debilitation occurred after this age, and by including only four calendar years in the analysis, each estimated cohort effect in model (4) might result in too large a standard deviation due to few observations included. For this reason, model (4) needs to be extended. Model (5), which will be presented below, is an extension of Horiuchi's model (4) and differs in several ways. The major difference is the number and selection of age-groups, periods, and cohorts. I use i = 1,2,…,14 for the five-year age groups 35-39,…,100-104 and j = 1,…,17, for the single calendar years 1917, 1922, 1927, …, 1992, and 1997. I also use k = 1,…,15 for the five-year cohorts born between 1865-69 and 1935-39. I use death rates for single calendar years 1917-97 and one-year age groups 35-104 to estimate the model. The total number of observations in the data material is 238. The all-cause death rates from 1917 to 1994 are from Mamelund and Borgan 1996, while the all-cause death rates from 1995 to 1997 are from Statistics Norway's (SSB) online database (see http://www3.ssb.no/statistikkbanken/). The average number of observations for each five-year cohort is 11.2, with a maximum of 14 observations (followed from 35-39 years to 100-104 years) for the four five-year cohorts born between 1880 and 1899, and with a minimum of observations for the cohorts born 1925-29 (8), 1930-34 (7), and 1935-39 (6).

The suggested model is

$$G_{ij} = \alpha + \beta_1 i + \beta_2 i^2 + \gamma_j + \delta_k + \varepsilon_{ij}, \qquad (5)$$

where α is a constant, and γ and δ are period and cohort effects respectively. When estimating Horiuchi's model (4) on the Norwegian data it was found that the age effects increased linearly with age for males and females. Thus the age effects could be parameterized as a linear function of age. In other words, it was assumed that mortality increases exponentially with age (Gompertz curve). To take into account the possibility of slower increase, stabilization or even a decline in mortality among the oldest old due to selection effects, a quadratic effect of age was added. Hence the term $\beta_2 i^2$ in model (5). Given this parameterization the perfect relationship between the three variables age, period, and cohort was broken, and the model could be identified without identification constraints. Thus, model (5) estimates only two coefficients for the age-effects as compared to 10 in Horiuchi's model (4), thereby sharpening all parameter estimates in model (5). In model (5), j = 1 (1917) is selected as reference category for the period effects, and k = 1 (1865-69) as the reference category for the cohort effects. Because the G_{ij}'s are not observed, but estimated, weighted least squares (WLS) estimation was used to estimate model (5), using the inverse value of the estimated variance for each G_{ij} as weight (Horiuchi used OLS).[1] The parameter estimates for the effect of age, period or cohort on G_{ij} in model (5) did not change significantly whether the analysis started with one of the calendar years 1917, 1918, 1919, 1920, or 1921 (and ended with one of the calendar years 1997, 1998, 1999, 2000, or 2001).

The estimated 5-year cohort effects (δ's) of model (4) follow a straight line from one cohort to the next for males and females, and do not confirm the hypothesis of high relative mortality of cohorts born about 1900 (Figure 6). Not a single of the cohort effects, however, differed significantly from zero as model (4) estimated relatively many parameters (23) compared to the number of observations (40). The age effects were all negative and statistically different from that of the reference category (i = 80-84 years), and increased as expected with age (estimates not shown here). The period-effects (γ's) are in accordance with a previous study on Norwegian mortality (Mamelund 1996); mortality declined for women, but stagnated for men in the 1960s (estimates not shown here).

In Figure 7 the estimated 5-year cohort effects (δ's) from model (5) are presented. This time, the cohort effects do not follow a straight line from one cohort to the next. Instead,

[1] An approximate expression for the variance of the logarithm of the death rate is found as follows. Assume that the survival probability l(t)= 1+bt is a linear function of time t through a given calendar year, where 0<t<1 and b<0. The Maximum Likelihood estimator for b and the death rate m are $\hat{b} = -D/N$ and $\hat{m} = D/(N - \frac{1}{2}D)$ respectively, where *D* is the observed number of deaths in a given calendar year, and *N* is the number of persons alive at t=0. The variance of $\hat{b}$ equals

there is clear cohort differentiation with maximum cohort effects or highest relative mortality occurring for women born 1890-99 and for men born 1900-09, and with gradually decreasing cohort effects for older and younger male and female cohorts. The cohort effect for men born 1905-1909 is statistically different from the cohort effects of those born during the 1870s (at 0.01 level), the 1880s (at 0.05 level), the five-year period of 1890-94 (at 0.1 level), as well as those born in the 1930s (at 0.05 level). The cohort effects for those born 1895-1904 and 1910-1929 do not differ significantly from the maximum cohort effect at 0.1 level (calculations not shown here). Nevertheless, the results show that it is highly unlikely that the concave curve for the cohort effects with maximum occurring for men born about 1900 are due to mere chance. Of the oldest female cohorts, only those born 1870-74 (0.05 level) differ relatively strongly from that of the maximum cohort effect estimated for those born 1890-94 (the 1875-79 cohort is significantly different at 0.2 level). All cohort effects for women born after 1904, however, differ strongly from that of the maximum cohort effect for women born 1890-94 (at 0.01 level or lower).

The reason why the K(x) analysis for women did not show a similar cohort differentiation as that identified in the APC-analysis might be that it only controls for age and cohort, and not for the three dimensions of age, period, and cohort simultaneously. Cohort effects may for instance be counterbalanced by the general decline in period mortality throughout the period studied.

[Figures 6, 7, 8, and 9 approximately here]

The estimates of the parameters β_1 and β_2 that parameterize the age-effects for males and females were all statistically significant at 0.01 level. Figure 8 gives the estimated effects $\beta_1 i + \beta_2 i^2$; The death rates increases from one five-year age group to the next by an average of 20 and 23 per cent for men and women respectively at ages below 70, while the average increase in the death rates at ages 70 and above is 23 and 35 per cent for men and women respectively. When removing the constant (α) when estimating model (5), the crossover in the age-effects by sex seen in Figure 8 disappears. The period-effects (γ's) are in accordance with what we already know (Figure 9). Men on the one hand, experienced significant mortality decline from 1917 until the late 1950s. The decline stagnated in the 1960s (the differences between the 1950s and the 1960s not significant at 0.1 level), but then it continued into the late 1970s,

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$(N-D)D/(N^3)$ (Keilman and Gill 1986). By the Delta-method we find an approximate expression for the variance of the log of the estimated death rate $\hat{m}$ as $Var(\log \hat{m}) \approx \frac{N(N-D)}{D(N-D/2)^2}$

1980s, and 1990s. Females on the other hand experienced a significant decline in mortality from 1917 until 1977, while mortality increased significantly from 1977 and into the 1980s and 1990s (at 0.01 level). Because of this, the difference between male and female life expectancy decreased in the late 1980s and the 1990s (Mamelund 1996). One of the reasons why this happened may be that women have adapted the smoking behavior of men, while fewer young men started to smoke and long-time smokers was able to quit, with the result that CHD mortality declined faster for men than for women (Pampel 2002). As discussed in section 2.2, however, Azambuja and Duncan (2002) have suggested that the degree in which Spanish Influenza 1918-19 infected the different cohorts might be a competing or additional factor explaining the changing risks of CHD mortality.

3.3. Discussion of results

The analysis showed that Norwegian males and females born about 1900 experienced significantly higher mortality in middle and old ages than neighbor cohorts. The results confirm the finding of another study on the Norwegian data (Gómez de León 1991). The same male and females cohorts were also identified as high mortality cohorts in Germany (only men), Austria, Hungary, Italy, France, and Poland, all belligerent countries during WW I (Horiuchi 1983; Boleslawski 1985; Caselli et al. 1987; Caselli and Capocaccia 1989; Caselli 1990; Wilmoth et al. 1990). The results in this paper show that Horiuchi (1983) was wrong insisting that only male cohorts born around 1900 in belligerent countries have experienced high relative mortality in middle and old life. Clearly, the search for causes that can explain the cohort differentiation in neutral countries, and probably also belligerent countries, must go beyond the indirect and direct effects of WW I. Hourichi explained the relatively high average mortality of men born about 1900 by high mortality of cardiovascular diseases in old age caused by malnutrition experienced in adolescence. In a country such as Germany, which suffered severe malnutrition (see last part of section 4.2), it is also reasonable to believe that malnutrition played a relatively important role in shaping the identified pattern of male cohort differentiation. However, there are also other candidates, for instance CHD mortality related to cigarette smoking, mentioned by Horiuchi, but considered to be of less importance compared to effects of WW I. Anyway, there seems to be no reason to believe that only teenagers and young adults at the end of WW I should become the heaviest smokers, and that the death rates of cardiovascular diseases would be boosted for a particular group of cohorts relative to that of preceding and succeeding cohorts. Spanish Influenza is a third candidate, relevant for both neutral and belligerent countries.

In his K(x) analysis, Horiuchi did not find cohort differentiation for German females. It seems as if this was the reason why Horiuchi did not further investigate female mortality by controlling for age, period, and cohort effects simultaneously. Instead, Horiuchi suggested

that women, who on average have more body-fat than men, were better able to bear a period of malnutrition without experiencing long-term effects on health and mortality. Wilmoth et al. (1990) were not convinced by Horiuchi's explanation; The authors believed that German women also were debilitated by indirect effects of WW I, including malnutrition, but that the weakening was obscured by female mortality decline in the post WW II period. For men, they further suggested that the cohort differentiation in mortality was maintained or even made more marked in Germany as mortality for men stopped to decline and slightly increased in the 1960s and 1970s (as was the case in most industrialized countries). This development support an argument for not only using K(x) analysis, but that further analysis should be carried out using APC-analysis. As seen in section 3.2 the period effects estimated for females in Norway from 1917 until the late 1970s was not strong enough to counterbalance the cohort differentiation found for the women born about 1900. Horiuchi (1983) suggests that the increase in CHD mortality of German men in the 1960s and 1970s may have been caused by malnutrition experienced in the years 1914-18. In this paper it is suggested that Spanish Influenza 1918-19 may be the most important cause why CHD mortality among men also stopped to decline and slightly increased in Norway in the 1960s. According to the latter assumption, the increase in CHD mortality is thus not a factor, which "maintain" the weakening of the cohorts born about 1900, but instead a direct result of living through a period with Spanish Influenza and malnutrition in adolescence.

Horiuchi (1983) did not find a clear pattern of cohort differentiation among men in neutral Sweden and Japan using the K(x) tool (women were not analyzed). The reason why Horiuchi did not find cohort differentiation for men in Sweden and Japan nor cohort differentiation for women in Germany might be that he assumed that the same male cohorts that were debilitated in belligerent Germany, especially that of the five-year cohort of 1899-1904, also were debilitated in all other countries, and that German males and females were equally affected. There are several problems with this assumption. First, the center of high mortality generations may vary from country to country and between the sexes. As seen in section 3.2, the center of the high mortality generations of Norwegian females was ten years later than that of their male counterpart. Wilmoth et al. (1990) identified the same sex difference as was seen in Norway for France. The reason why the debilitated female cohorts (1890-1899) were older than their male counterpart (1900-1909) may be that women were extremely vulnerable to complications in connection with pregnancy and birth when infected by Spanish influenza (Mamelund 2003). Also, the reason why the cohort differentiation in all-cause mortality identified for Germany, France and for Norway is more pronounced for men than for women might be that the incidence of Spanish influenza 1918-19 was greater among men than among women, which is believed to be the reason why CHD mortality in the latter part of the twentieth century was greater for men than for women. Second, although Horiuchi

found no visible cohort effects in the data before 1959 for German males, that is 40-45 years after the cohorts of interest experienced deteriorating living conditions and hardship due to WW I (and Spanish Influenza), it is a dubious assumption that the expected negative effects on health and mortality do not show up in the data before 1959 for German females or for males in neutral countries (i.e. not including data in the analysis before 1959). This is confirmed by the smoothed one-year K(x) analysis in the present paper (results not shown), which shows that Norwegian men born between 1895 and 1910 had higher mortality than neighbor cohorts, not only in the 1960s, but also the 1950s. Third, by including only ages up to 84 years and thereby assuming that no debilitation occurred after this age, and by including only four calendar years in the analysis, each estimated cohort effect might have too large a standard deviation due to few observations included. (The author of the present paper is aware that 20 years have passed since Horiuchi published his paper, and that observations in period 1983-2003 were not available to him (see section 3.2)). As mentioned in section 3.1, however, no striking cohort effect was found for male cohorts born about 1900 after 1970 when they where 70 years (results not shown). A closer look at different causes of death, for example suicide, CHD and Parkinson's disease may have given better understanding of the phenomenon studied, but was not carried out here.

The estimates from the refined and extended APC-model applied in this paper (see expression (5)) are clearly sharper and trustworthier than the estimates from Horiuchi's APC-model (se expression (4)). The estimates are sharper because the number of observations is six times higher (238 compared to 40), while the number of parameters estimated does not differ much (33 compared to 23). This gives many more degrees of freedom (205 compared to 17). The use of weights in the estimation of model (5) probably made the parameter estimates trustworthier.

4. Discussion of some confounding factors

As discussed in section 3.3, one possible reason other than Spanish Influenza why the cohorts born about 1900 have experienced relatively high later life mortality may be cigarette smoking. In the following, three more hypotheses explaining the relatively high mortality found for Norwegian cohorts born about 1900 are presented. It is discussed why each of the three causes may have had an independent debilitating effects on the health of the cohorts considered net of the effect of Spanish Influenza, whether they affected males and females differently, and whether a specific cause, for example food shortage and rationing, during WW I was severe or widespread enough to have deteriorating effects on later life health and thereby the risk of premature death.

4.1. War accidents and post-traumatic stress disorders among WW I survivors in later life

When considering that enormous number of soldiers from the belligerent nations in Europe was wounded, exposed to toxic gas warfare, and experienced poor sanitary conditions and nutritional deprivation, it is not surprising that it has been found the male cohorts directly involved in combats experienced relatively high mortality in later life (Horiuchi 1983; Boleslawski 1985; Caselli et al. 1987; Caselli and Capocaccia 1989; Caselli 1990; Wilmoth et al. 1990). Of British cohorts born 1862-96 and of French cohorts born 1869-99, 60 per cent were finally mobilized (Winter 1977; Wilmoth et al. 1990). Of all men in military age 80 per cent were mobilized in Germany (Gregory 1997). Of the British soldiers, one in eight was killed and more than one in four was wounded, and of the German soldiers 15 per cent were killed. French cohorts born 1892-95 were reduced by more than a fourth due to battlefield deaths.

In this section, evidence is presented that relatively few Norwegian men born about 1900 was injured or died in military accidents during and shortly after WW I. It is therefore believed that the direct effects of WW I on male Norwegian cohort mortality must have been small. The following discussion is not relevant for women as even fewer women were victims of military accidents.

The Norwegian navy did not have to take part in combats to secure the Norwegian neutrality or to protect the Norwegian merchant fleet. The only casualties registered in the years 1914-20 are due to accidents. Four navy soldiers and six civilians, for instance, lost their lives when navy soldiers were detonating mines (Marinens admiralstab 1940). There are no reports of people wounded working on this dangerous task. The number of Norwegians that volunteered for combats and were wounded or killed at the front is unknown to the author, but the number is believed to be too small to have significant effect on the phenomenon studied.

A substantial number of Norwegian seamen in the Norwegian merchant fleet and Norwegian seamen on foreign ships were injured and died in military accidents, especially after the onset of the all-out submarine war in April 1916. It is estimated that 300 or 0.1 per cent of the seamen was *physically* disabled and that 1,892 died due to military accidents in the years 1914-20 (Sjømennenes Minnehall, Stavern; Riksforsikringsanstalten 1923). Fear of being dragged into the war, being recruited for frontier guard duty, and of being physically injured or killed if torpedoed or mined may also have *psychologically* disabled sailors as well as the navy conscripts in later life. However, the number of first-time hospitalized patients at Norwegian asylum hospitals with mental disease linked to these reasons was only 17 over the period 1914-20 (Medisinaldirektøren 1916, 1917, 1920a-b, 1921, 1923a-b). This figure is small, but there are reasons to believe that many persons with equally strong or milder stress

disorders compared to those hospitalized did not see a psychiatrist. Askevold (1976), for example, found that one third of the Norwegian sailors in the merchant navy, which survived WW II, were psychologically disabled and on invalid pension in the late 1970s. Most suffered from the "War Sailor Syndrome", which consisted of two parts, the first being a non-neurotic anxiety repeating the terrors of war, and the second being a brain damage caused by the constant fear of death. Of the two-thirds that were not disabled, many were not able to take up their work at sea and had to go ashore to low-status, low-wage jobs. If it is assumed that also a third of the WW I veteran seamen developed the "War Sailor Syndrome" in later life, 10,000 Norwegian seamen were psychologically disabled after WW I. Only 3.4 and 2.5 per cent of the male high mortality cohorts of 1901-05 and 1891-1900, however, were seamen by occupation respectively in 1920 (Calculated from SSB 1924). This means that only about one per cent of the cohort born 1891-1905 can have been debilitated due to psychological after-effects due to service in the merchant fleet during WW I.

The recruitment to the Norwegian navy was basically based on compulsory military service of men 20-22 years old. The conscripts on duty in the navy during the years 1914-20 were thus born between 1893 and 1900. In the period 1914-20, 11,474 conscripts or less than one per cent of the cohorts considered were recruited for active service in the navy (SSB 1920ab, 1921c, 1922a-c; Forsvarsdepartementet 1919). If one assumes that also one third of the navy veterans experienced the "War Sailor Syndrome" in later life, only a little more than one per cent of the cohorts born about 1900 could have been psychologically debilitated in later life due to active service in the navy or in the merchant fleet during the war. This share is so small that it cannot have caused the cohort effect identified in the analysis for men born about 1900.

There are also studies that have reported that WW II debilitated (primarily males, but also to a certain degree females) adolescent and young adults survivors in Japan, Germany, the former Soviet Union, France, and Italy (Okubo 1981; Horiuchi 1983; Anderson and Silver 1989; Caselli et al. 1987; Caselli and Capocaccia 1989; Caselli 1990; Wilmoth et al. 1990). Could WW II partly explain the debilitation of the male cohorts born about 1900 in Norway? More than 10,000 war-deaths were reported in Norway 1940-45, of which nine out of ten were men (Backer 1948). If one supposes that it were the healthiest and the fittest men that were selected for duty and were injured or died in the war, and that men with the poorest health were found unfit for service and therefore survived the war, this would on average lead to a debilitation of the male cohorts considered. The soldiers that survived military combats, and the soldiers and seamen that survived military accidents, probably also suffered poor mental and physical health and thus higher mortality in later life (Askevold 1976). Those born about 1900, however, were on average 40-45 years in the period 1940-45, far from the average age of excess deaths due to the war (here assumed to be the average age of a mentally

or physically wounded soldier or seaman), which was 25 years (Gómez de León 1991). This factor might therefore contribute to explain the relatively high cohort effects found for males born 1910-14 and 1915-19 found in the APC-analysis (Figure 7). As it was mostly men that were affected by military casualties, either by being a soldier or a sailor during WW II, it is not surprising to find that the cohort effects for the corresponding female cohorts were not similarly high. As suggested by Wilmoth et al. (1990) and Caselli and Capocaccia (1989) in the case of the Italian and French population, an equally important explanation behind the high later-life mortality might be that large proportion of the cohorts born 1918-1919 were exposed to the Spanish Influenza virus in utero or as infants.

4.2. Shortage and rationing 1914-19

In this section it is discussed whether the shortage and rationing of food during WW I might have affected immediate or later life mortality of Norwegians born about 1900 positively. The evidence that is presented, however, gives little support to this hypothesis.

The daily calorie intake did not decline in Norway during the years 1914-17 as food that was less expensive, more abundant and equally nutritious substituted consumption of expensive food of short supply: Fish, whale oil margarine, whole milk, and whole meal bread replaced meat, butter, eggs, skimmed milk and white bread (SSB 1917; SSB 1918). In September 1917 (butter) and January 1918, however, milk, sugar, coffee, tea, bread, macaroni, peas, beans, barley, and oat were rationed because of increasing shortage and inflation. How did the rationing of food affect the weight and health of cohorts born about 1900? Pupils between 10 and 15 years (born 1903-1908) from two elementary schools in the city of Bergen were found to suffer from malnutrition and weight loss during the spring of 1918, but this did not lead to an increase in absence from school due to sickness (Looft 1919). In a study of height and weight among 12-18 years pupils (born 1900-06) in one secondary school in Kristiania, Schreiner and Schreiner (1922) found that the body-mass index fell during the spring of 1918, and that this might be explained by the rationing of food. The emaciation, however, was fully compensated for most pupils during stays at summer camps or visits to relatives in the countryside, where the supply of milk and other foodstuff was better than in the cities.

Several socialist newspapers claimed that the Norwegian neutrality-keeping soldiers were underfed due to the poor rations served in the army during the war, and for this reason the exercises should be stopped and the recruits sent home (Sjefen for hærens sanitet 1919). Because of this critique, the 20-22 year old recruits (born 1897-99) were weighted at the beginning of the recruit-school and every second week in 1918. The analysis of the weights showed that in every battalion, average weight increased by 0.5 to 3.0 kilogram per recruit per year. The average daily calorie-intake was 3,700 gram a recruit compared to 3,000 gram for

the average worker and low paid public clerical officer in the two cities of Kristiania and Bergen. The rationing of food was gradually lifted in the five month period from March to July 1919 and during the summer of 1919 most food articles were in normal pre-war supply (SSB 1920c; SSB 1921). The average calorie consumption and consumption of earlier rationed food articles increased markedly after this, but as people learned that demand was fulfilled, as normal, consumption went down (SSB 1921).

From the above presentation, it seems reasonable to conclude that although the period of heavy rationing in 1918-19 affected food intake of cohorts born about 1900, it is not likely that the rationing was serious and long lived enough to leave lasting marks which can give a significant rise in later-life mortality. Also, it has been reported elsewhere that the mortality during the first half of 1918, which was a period of relatively heavy rationing of food, was lower than the average of the first halves of the three years 1915-17 which was characterized by increasing shortage of food, but no rationing (Mamelund 2003). A comparison with Germany also shows how relatively well-off Norway was when it comes to nutritional problems during and after WW I. Grebler and Winkler (1940) have estimated that the German population in 1920 only had 50 per cent of the supply of the most need food items compared to 1913. The average Frankfurt resident subsisted on roughly 1,500 calories a day in the summer of 1918, with 2,500 calories daily considered normal, and the average weight loss a person approached 23 per cent of pre-war pounds (Fritz 1992).

4.3. Mass emigration and selection

Gómez de León (1991) was tempted to explain the high relative mortality in later life of the Norwegians that were adolescents in 1918 by debilitation effects of the Spanish Influenza Pandemic. But he also assumed that a late wave of predominantly male overseas emigration, which peaked in the mid 1920s, might have introduced additional selection if the most selective of the survivors left. The question of selection effects imposed by emigration is surprisingly not discussed in the previous papers on this topic (Horiuchi 1983; Boleslawski 1985; Caselli et al. 1987; Caselli and Capocaccia 1989; Caselli 1990; Wilmoth et al. 1990). Gómez de León's hypothesis is appealing. Bævre (2001), for example, has found that the massive overseas emigration 1846-1939 raised mortality of Norwegian cohorts, male mortality more than female mortality because men migrated more than women, suggesting a strong selection mechanism. Moreover, Bævre et al. (2001) have documented that by age 50 male and female cohorts born 1865-85 were reduced by 30 and 20 per cent due to emigration respectively, while male and female cohorts born 1890-1900 were reduced by only 13-19 and 7-13 per cent due to emigration respectively. If those with the best health were selected for emigration, this would on average leave more persons with poor health among those born 1865-85 as compared to the average health of those born 1890-1900. The 1865-85 cohort is

thus assumed to have higher mortality in later life than the cohort born 1890-1900 due to emigration.

5. Conclusion

The analysis showed that male and female cohorts born about 1900 in Norway, which was neutral during WW I, experienced significantly higher all-cause mortality in middle and old ages than preceding and succeeding cohorts. This finding demonstrates that Horiuchi (1983) who has insisted that this cohort effect is exclusive for males in the countries that were belligerent during WW I, for instance Germany, Poland, Austria, Hungary, Italy, and France, seems to be wrong. Clearly at least, the search for a possible explanation in neutral countries must go beyond the direct (soldiers wounded physically and mentally) and indirect effects (shortage and rationing of food) of WW I on later life mortality by sex. Hourichi (1983) explained the high relative mortality of German men born about 1900 by high mortality of cardiovascular diseases in middle and old age caused by malnutrition experienced in adolescence during WWI. The finding in this paper demonstrates that Horiuchi's explanation may be incomplete. This paper has argued that Spanish Influenza may be the most important candidate explaining the debilitation of the male and female cohorts considered in neutral Norway. The cohorts born 1890-1910 experienced the highest incidence, but the lowest immediate death rates of Spanish Influenza 1918-19. The disease did thus kill relatively few but marked relatively many of the persons belonging to these cohorts. Mental distractions, hepatitis, ear illnesses, deafness, blindness, and baldness are examples of the peculiar after-effects that the survivors faced in later life. The survivors seem to have had high risks of committing suicide immediately or shortly after 1918-19. The influenza survivors may also have had higher risk of dying from the Sleeping Sickness, Parkinson's disease, and coronary heart disease in later life.

It has been shown that neutral Norway fits well as a test case to study effects of Spanish Influenza on later life mortality as it is partly counterbalancing effects of WWI. One of the indirect effects of the war that may have caused an increase in later life mortality is shortage and rationing of food. Although some of the Norwegian cohorts considered experienced a drop in weight during the relatively heavy rationing of food spring of 1918, there was little or no immediate increase in morbidity or mortality. In the years 1915-17 the daily calorie intake was maintained as food that was less expensive, more abundant and equally nutritious substituted consumption of expensive food of short supply. It is therefore believed that the shortage and rationing of food is confounding Spanish Influenza relatively little as the major factor debilitating the cohorts considered. Although the Norwegian navy did not have to take part in combats to secure the Norwegian neutrality or to protect the Norwegian merchant fleet, the paper has documented that post-traumatic stress disorders was present among the

neutrality-keeping soldiers and the seamen in the merchant fleet, who survived WW I. Thus, direct effects of WW I are a possible confounder of Spanish Influenza. However, as it is estimated that only a little more than one per cent of the men born about 1900 can have experienced poorer health and thereby higher mortality due to this event, it is believed to be of relatively little importance compared to Spanish Influenza.

Another possible confounding factor, surprisingly not dealt with in previous studies on this topic, is selection effects imposed by periods of mass emigration. Did the late wave of predominantly male overseas mass emigration, which peaked in 1923 and 1927, select those with the best health among those born 1900, leaving more persons with poor health? The answer might be yes. The reason why the cohort effects in neutral Norway were found to be more pronounced for men than for women may be that more men than women emigrated. The sex-difference in the cohort effects, however, may also be explained by the sex difference in mortality of coronary heart disease; Men have always had higher CHD mortality than women, but what was the reason for this? Two possible answers stands out; higher incidence of Spanish Influenza among men than women in 1918-19 and/or that more men than women are or have been cigarette smokers.

The APC-analysis applied in the paper, which utilizes no other covariates than age, period and cohort to explain the variance in the death rates, cannot contribute to the *explanation* of why cohorts born about 1900 were debilitated. It is possible though, to create and to discuss plausible hypotheses that caused the cohort differentiation identified in the analysis. The answer whether or why the cohorts considered were debilitated in later life due to Spanish Influenza 1918-19 can most likely only be found by linking the data on incidence by occupation, housing standard, and socioeconomic status from the individual "influenza-censuses" carried out in the years 1918-19 in Bergen, Norway, and in a number of cities in the United States and England with modern individual death registers.

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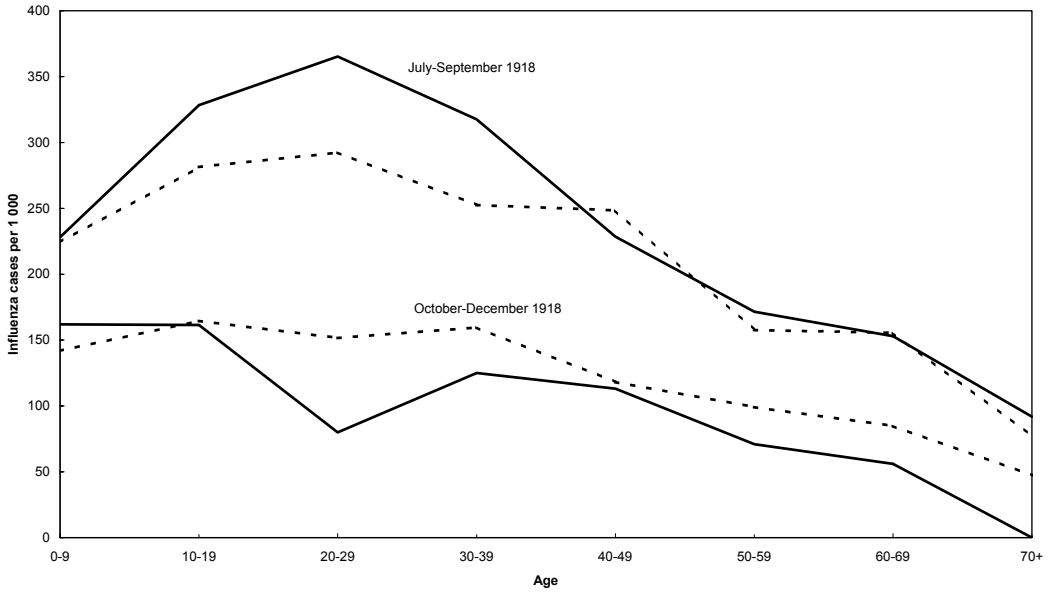

Figure 1. Age-sex specific incidence rates of influenza in the city of Bergen, July-September 1918 and October-December 1918 (male solid and female dotted)

Source: Hanssen 1923.

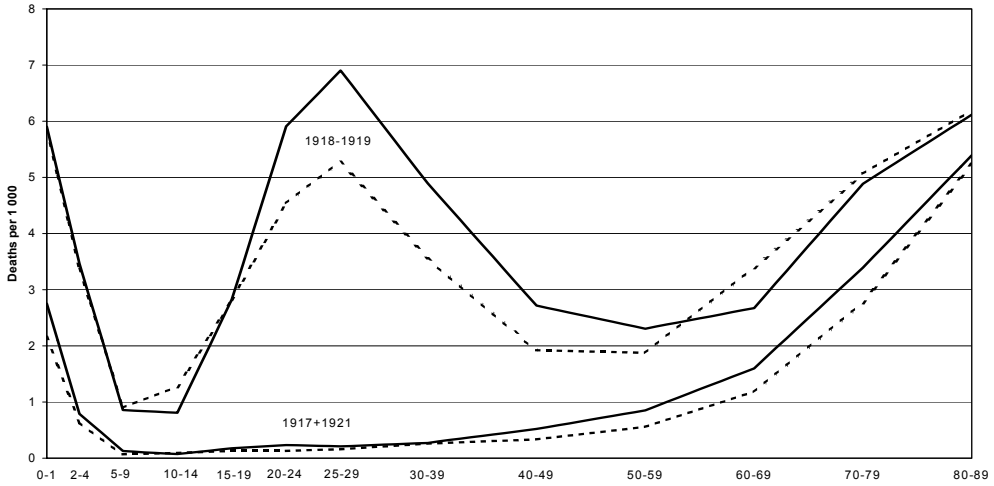

Figure 2. Age-sex specific death rates of influenza and pneumonia, Norway 1918-19 and 1917 and 1921 (male solid and female dotted).

Source: DCM (1921, 1922, 1923, and 1925).

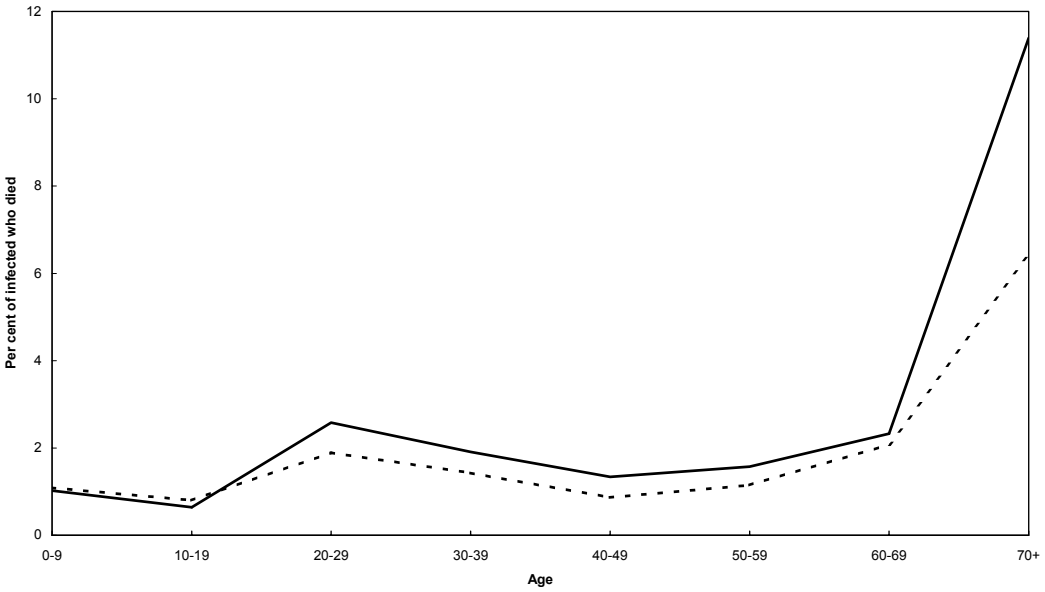

Figure 3. Lethality of influenza and pneumonia in Norway 1918-19 (males solid and females dotted).[1]

Source: Calculated from DCM (1921, 1922) and Hanssen (1923).
[1] The number of influenza and pneumonia cases by age and sex are estimated by multiplying age-sex specific incidence rates from Bergen July 1918-March 1919 by the population in Norway 31.12 1918 by age and sex.

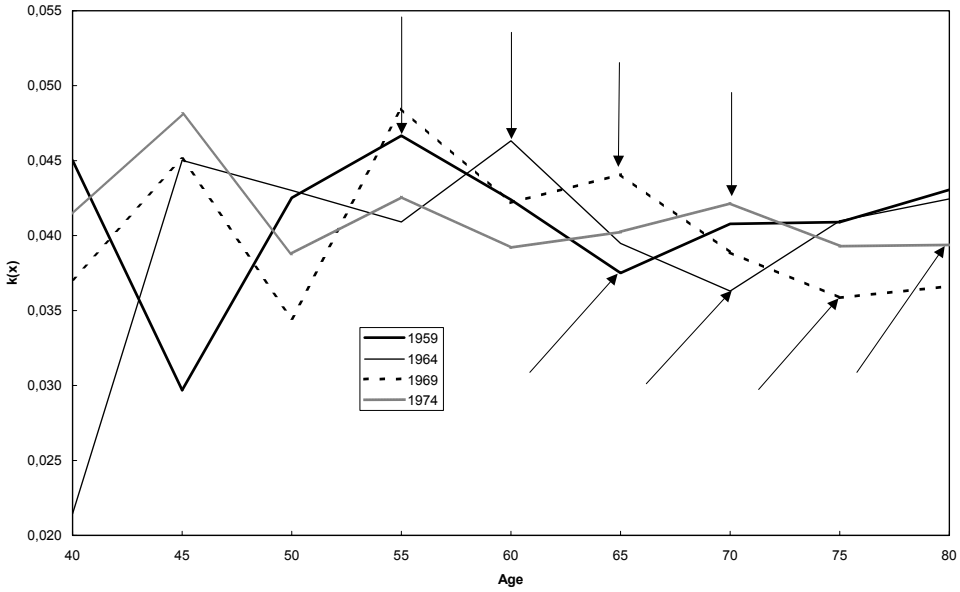

Figure 4. Rate of mortality change with age, K (x), for Norwegian males 1959, 1964, 1969 and 1974 using five-year age groups

Note: The downward-pointing and upward-pointing arrows mark the last and the first of high mortality cohorts respectively.

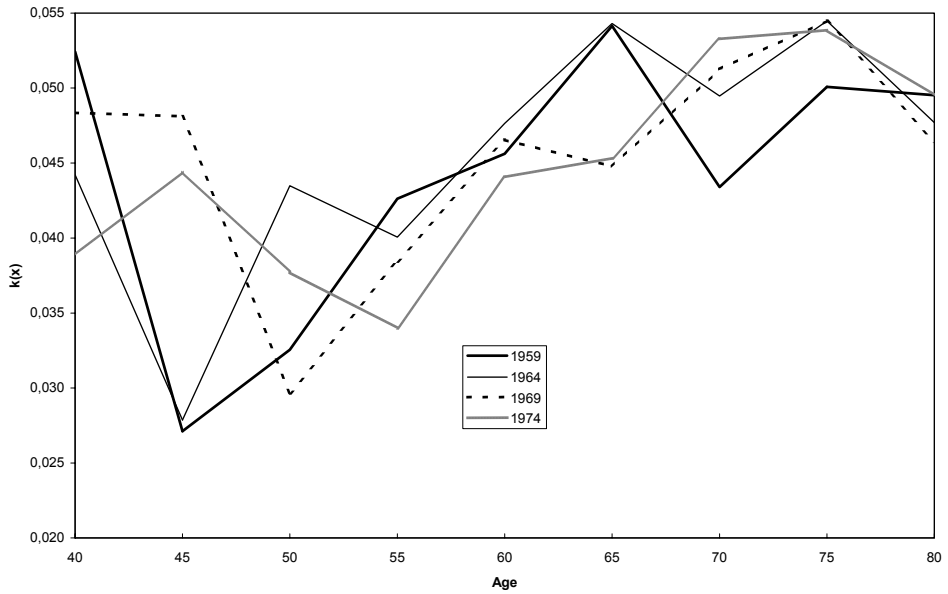

Figure 5. Rate of mortality change with age, K (x), for Norwegian females 1959, 1964, 1969 and 1974 using five-year age groups.

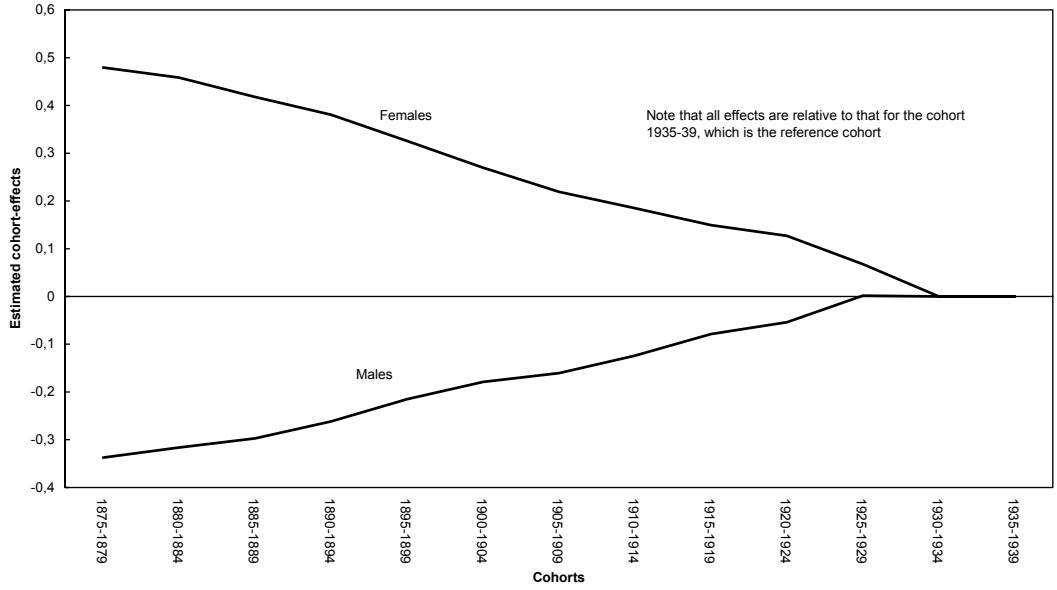

Figure 6. Estimates of cohort effects (δ's) for Norwegian males and females in model (4) proposed by Horiuchi 1983, pp. 81-84.

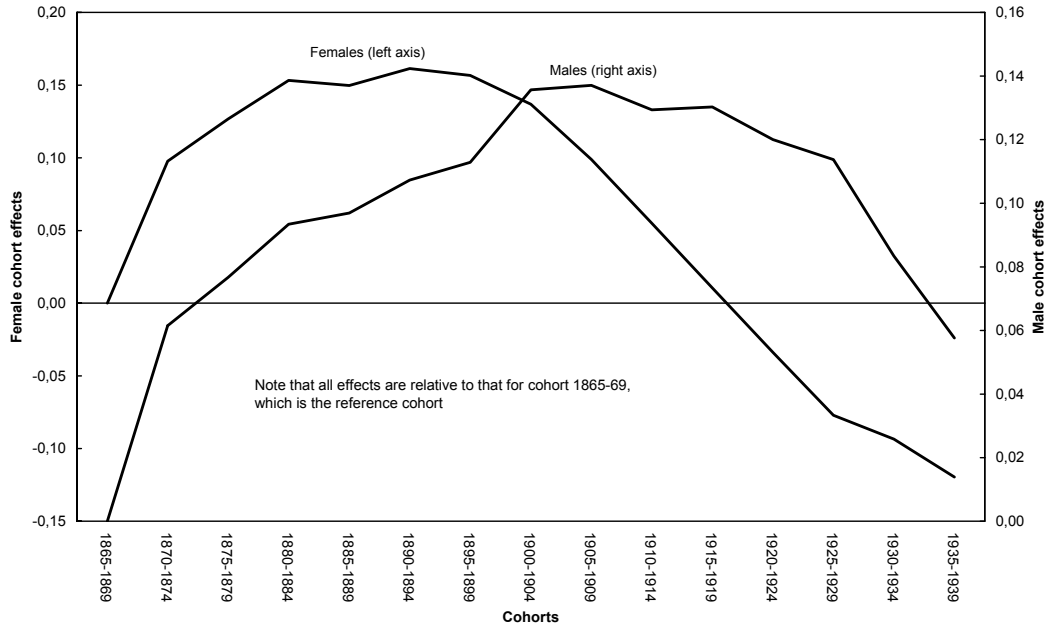

Figure 7. Estimates of the cohort effects (δ's) for Norwegian males and females in model (5)

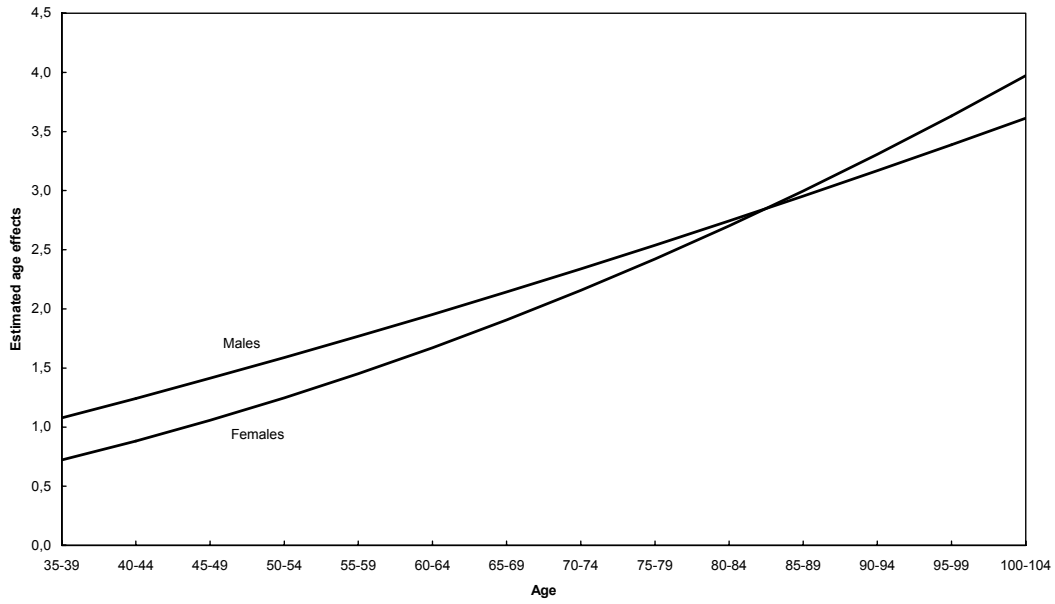

Figure 8. Estimates of the age effects ($\beta_1 i + \beta_2 i^2$) for Norwegian males and females in model (5)

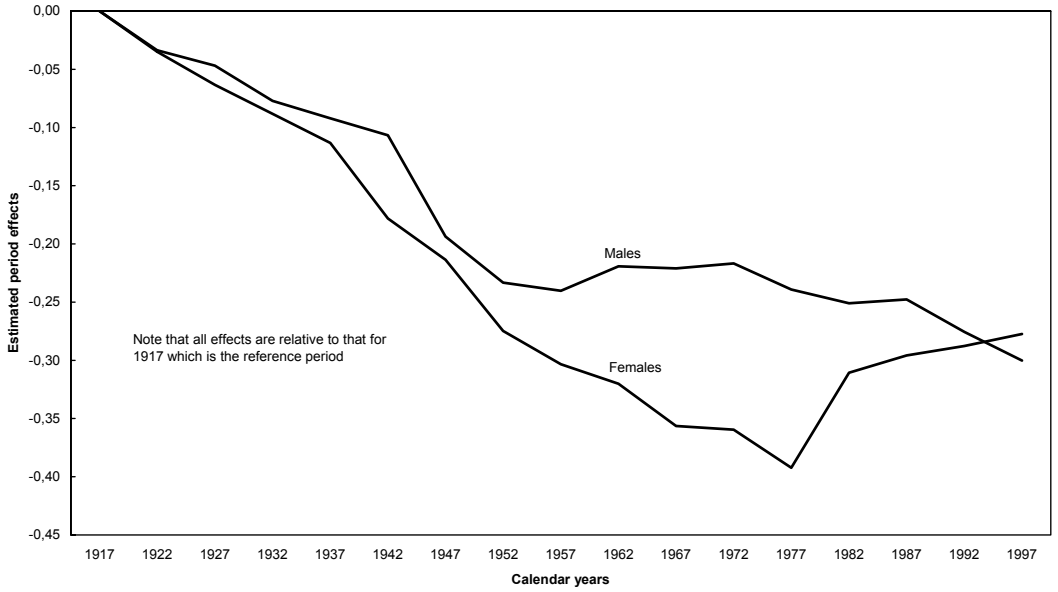

Figure 9. Estimates of the period effects (γ's) for Norwegian males and females in model (5)