Period and cohort changes in mortality risk over the twentieth century in the UK: an exploratory analysis

Jones, P.M., Minton, J. and Bell, A.

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Authors

1. Jones, P.M., University of Sheffield (ORCID 0000-0001-5173-3245)
2. Minton, J., University of Glasgow (ORCID 0000-0003-1207-6259)
3. Bell, A., University of Sheffield (ORCID 0000-0002-8268-5853)

Corresponding author

Phil Mike Jones
School of Law
University of Sheffield
Bartolomé House
Winter Street
Sheffield
S3 7ND

phil.jones@sheffield.ac.uk

philmikejones@gmail.com

Abstract

This paper explores discrete changes in age-specific mortality risk across periods and cohorts, using data from 40 countries during the twentieth century with a particular focus on the UK. Our interest is in the discrete changes in mortality seen as a result of global events, rather than the overall changes in mortality throughout the twentieth century. We use innovative exploratory visual and statistical methods to explore

these changes. First, Lexis surface plots are used to visualise period and cohort ‘scars’ in age-specific period and cohort mortality in the population across the twentieth century. Second, we use a version of the hierarchical age-period-cohort (APC) model to statistically separate discrete period and cohort effects. We avoid the APC identification problem by analysing only discrete deviations net of any linear APC effects. We find period and cohort effects in mortality associated with the First World War of 1914–18, the influenza pandemic of 1918, the Second World War of 1939–45, and, intriguingly, a decline in mortality in many western countries associated with the 1948 birth cohort. We believe a combination of factors influenced this improvement in mortality, including the developments of post-war social welfare, the economic investment in Europe by the United States, the accessibility of antibiotics such as penicillin, and, in the UK, the NHS. Our findings suggest universal or accessible health care make significant and detectable improvements to population mortality for those born under it. These methods could productively be used by epidemiologists and others to explore deviations from APC trends in other longitudinal research contexts.

Keywords

UK; Age-period-cohort; Lexis surface; mortality; NHS

Introduction

Mortality risk decreased during the twentieth century for most groups of people, in most places in the world (The World Bank, 2017). Separate from this overall downward trend there are annual deviations in mortality. Some of this deviation will occur naturally without any outside influence or stimuli: everything varies; some of this deviation will be due to important influences or events whose effects are worthy of study.

In this paper we explore deviation in mortality risk during the twentieth century in the UK. Our interest is not in the long-run downward trend in mortality or in natural variation, but in the discrete changes in mortality seen as a result of global events. The twentieth century was notable for remarkable social and technological progress, as well as catastrophic global conflict, and we explore how these affected mortality.

Deviations over time in mortality can occur in two ways. First, period effects affect everyone at the time who is exposed to the event. Second, cohort effects occur when events affect specific generations of people. Throughout their lives individuals in the affected birth cohort benefit from or are hindered by these events that occurred in their formative years.

To explore these effects on changes to mortality risk we use a combination of visual and statistical exploratory techniques. First, we use Lexis diagrams to show the patterns in mortality change in the UK and beyond. Second, we use a modified version of the hierarchical age–period–cohort model (HAPC, Yang and Land (2006)) to find similar patterns. These are not age–period–cohort (APC) models in the sense that we do not consider long–running linear APC trends; instead we focus only on deviations from those trends, avoiding the APC identification problem (Glenn, 2005).

Literature

Age, period, and cohort effects on mortality

Suzuki (2012 p. 452) outlines the following fictional dialogue to illustrate the difference between age, period, and cohort effects:

A: I can't seem to shake off this tired feeling. Guess I'm just getting old. [Age effect]

B: Do you think it's stress? Business is down this year, and you've let your fatigue build up.
[Period effect]

A: Maybe. What about you?

B: Actually, I'm exhausted too! My body feels really heavy.

A: You're kidding. You're still young. I could work all day long when I was your age.

B: Oh, really?

A: Yeah, young people these days are quick to whine. We were not like that. [Cohort effect]

Age is the measurement of time passed since birth. Period is 'historical time' when the measurement was taken, so represents a snapshot of all people, of all ages, in the study at that instance (Goldstein, 1979 p. 19; Suzuki, 2012 p. 452). A cohort refers to:

... those individuals (human or otherwise) who experienced a particular event during a specified period of time. The kind of cohort most often studied by social scientists is the human *birth cohort*, that is, those persons born during a given year, decade, or other period of time (Glenn, 2005 p. 2, original emphasis).

Ryder argues that "[e]ach cohort has a distinctive composition and character reflecting the circumstances of its unique origination and history" (1965 p. 845).

Each of age, period, and cohort can have effects on individuals. Using mortality as an illustrative example

outcome, an age effect might suggest the risk of death increases or decreases as a person gets older. A period effect could be caused by an event that affected multiple groups at once, for example a war, disease, or economic recession causing increased likelihood of death across individuals of all ages at that point in time. A cohort effect might manifest as subsequent cohorts having incrementally lower mortality risk than earlier cohorts, perhaps because of improvements in living standards in their formative years (Bell and Jones, 2013 p. 163). For this paper we are primarily interested in period and cohort effects, since the effect of age on mortality is relatively well established (as you get older, the mortality rate increases after the first few years of life), and there are fewer reasons to expect discrete effects that apply to specific age groups (as opposed to long-run gradual changes over the life course).

We anticipate being able to detect period effects for significant events such as war, famine, or epidemic because more deaths are observed at the time of the event. Literature on developmental plasticity (Gluckman et al., 2010) suggest cohort effects on mortality over the life course are also plausible. Developmental plasticity is primarily adopted and advanced by two key literatures, the Developmental Origin of Health and Disease (DOHaD) hypothesis and life course epidemiology (Hanson and Gluckman, 2016). These hypothesise that developmental environment affect the structure, physiology, and function of organs and systems throughout the individual's life (Fall et al., 1995; Wadsworth and Kuh, 1997; Ben-Shlomo and Kuh, 2002; Ben-Shlomo et al., 2016; Hardy and Tilling, 2016; Newman, 2016). 'Better' *in utero* and early-life environment leads to longer, healthier lives, while lower quality early-life environments lead to shorter, less healthy lives (Hertzman, 1999 p. 85). Links between prenatal malnutrition and low birth weight, neonatal mortality, cardio-vascular disease, coronary heart disease, ischaemic heart disease, and hypertension have been demonstrated (Hales and Barker, 1992).

Under this paradigm a stimulus, such as economic circumstances, sudden improvement in healthcare, and so on, can have biological and physiological effects on the individual that last throughout their life course, which has been shown to affect their morbidity and mortality. If the same stimulus affects a large number of individuals from the same or similar cohorts in the same way, patterns of mortality will be seen throughout the lives of the cohort members. This has been demonstrated in response to severe, discrete events such as the 1918–19 influenza pandemic. Even populations that diverged following the influenza pandemic, such as those of East and West Germany, show remarkably similar mortality 'scars' in cohorts born in 1918–19 (Minton et al., 2013).

... those born in early 1919 who were exposed prenatally to the most virulent phase in the Fall of 1918, had lifetime deficits in economic productivity and in education, as well as excess work disability, which suggests developmental impairments or lifetime health issues (Mazumder et al.,

2010 p. 26).

Events that affected mortality in the twentieth century

A number of significant events occurred in the twentieth century, both globally and nationally, that are likely to have affected population mortality in the UK, both as period effects and as cohort effects. Here we briefly discuss four that we see as particularly important: World War I, the 1918 influenza pandemic, World War II, and the enormous social progression following the end of the second world war, including the formation of the National Health Service (NHS) in 1948.

We would expect a period effect increase in mortality associated with the First World War of 1914–18. For the most part we would expect this to be limited to military personnel, but we might expect to see a period effect in the civilian population in other countries with higher civilian casualties, such as those in continental Europe. In the UK, civilians were not directly affected by the conflict but effects of deteriorating environmental conditions may be detectable. A detectable cohort effect among those born during and immediately after the conflict is also possible, for example because of poor maternal nutrition, exposure to disease, or inadequate health care as a result of the conflict.

The 1918 influenza pandemic is likely to result in detectable period effects as recent estimates have put the number of deaths from this disease at 50 million worldwide, or approximately five per cent of the global population (Patterson and Pyle, 1991; Johnson and Mueller, 2002). Approximately 250,000 died in the UK. Cohort effects for those born during the outbreak (1918 to early 1919) are also possible. Increased incidence of cardiovascular disease (Mazumder et al., 2010), decreases in life expectancy at birth (Noymer and Garenne, 2000), and increases in socio-economic deprivation (Almond, 2006) have been demonstrated in cohorts in the United States born with prenatal exposure to the disease. Of course, it is difficult to tell apart cohort effects of the war and the influenza pandemic given their temporal proximity. In the case of period effects the different age and gender of those theorised to be affected by each give a clue as to what caused each (with young men most likely to be affected by the war, whilst the effects of the influenza pandemic are likely to be more universal).

We anticipate a detectable increase in period mortality during World War II for both military and civilian populations. Civilian populations are likely to be more affected than in World War I, due to the changing nature of warfare, specifically the increase in bombings of civilians made possible by advances in technology. As well as the period effects there could also be cohort effects among individuals born during World War II. Although these are not theorised to occur in the UK, elsewhere many have found such cohort effects. Specific

events such as the Siege of Leningrad and the Dutch *Hongerwinter*, where significant numbers of individuals perished, have been shown to be associated with period and cohort mortality increases. Survivors of the Siege of Leningrad had a significantly higher risk of dying from breast cancer (Koupil et al., 2009), ischaemic heart disease, or stroke (Sparén et al., 2004) than those born during the same period who were not exposed to the seige. Similarly survivors of the Dutch *Hongerwinter* who took part in the Dutch Famine Birth Cohort Study were more likely to have blunted cardiovascular and cortisol stress responses, which are in turn associated with a range of adverse health outcomes (Carroll et al., 2017).

There may also be cohort effects which do not become apparent at birth, but later in life. A clear example not related to World War II is smoking uptake, where the age of exposure is much older than birth (Schöley and Willekens, 2017 p. 633). As a result, there is also a higher risk of psychological and physiological trauma among older cohorts which may manifest as differences in age-specific mortality later in their life course.

Following the Second World War, many Western nations implemented a greater number of progressive policies aimed at improving population health and wellbeing. Perhaps the most prominent example of this in the UK is the formation of the NHS in 1948 (Rivett, 1998). This involved a comprehensive reorganisation and rationalisation of medical provision, and treatment became free at the point of access for all. This included previously marginalised groups, such as working-class women, for whom treatment had previously been limited due to the prohibitive cost (Webster, 2002). A detectable period effect of reduced mortality is plausible at this time; although no new treatments were available with the founding of the NHS, treatment was suddenly available to everyone regardless of ability to pay.

A cohort effect is also anticipated in cohorts born in and after 1948. Being limited to the quality of care accessible during the antenatal and perinatal period—critical periods for the child—is likely to have adversely affected the development trajectory of many children. With the NHS, pregnant women could now access antenatal care, for the first time often provided by general practitioners, and give birth in hospital. Increasing the opportunities for intervention at critical periods *in utero* could result in improved health and reduced mortality over the life course for the infant.

Moreover, exposing pregnant mothers to the health care system through antenatal care and a hospital birth may have the cultural effect of ‘normalising’ the use of medical care. If this contributed to earlier detection of disease or illness this cultural effect could have benefits to the mortality of children born under the NHS throughout their lives, for whom seeing a doctor became part of their early socialisation.

Methods

Mortality data for 40 countries for the twentieth century were obtained from *The Human Mortality Database* [dataset] (University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany), 2017). Data were entered and prepared using the R programming and statistical environment, version 3.4.2 (R Core Team, 2017).

Lexis surface diagrams were plotted of yearly change in age-specific mortality rates in the UK, for both males and females, and these were additionally broken down into civilian and military populations. In order to compare the trends in the UK to other countries, plots were additionally made for other countries in Europe and North America (these can be found in the online appendix). Lexis surface diagrams are commonly used in demography to depict cohort information as well as period and the event of interest (Carstensen, 2006). Lexis diagrams were produced using the `Lattice` package for R, version 0.20-35 (Sarkar, 2008).

Hierarchical age-period-cohort (HAPC) models were constructed for countries or sub-regions of interest. The original version of Yang and Land's HAPC model (Yang and Land, 2006) treats the age effect as a fixed effect polynomial, with the period and cohort effects as cross-classified random effects. The model is thus:

$$y_{i(j_1 j_2)} = \beta_{0j_1 j_2} + \beta_1 \text{Age}_{i(j_1 j_2)} + \beta_2 \text{Age}_{i(j_1 j_2)}^2 + \epsilon_i(j_1 j_2) \quad (1)$$

$$\beta_{0j_1 j_2} = \beta_0 + u_{1j_1} + u_{2j_2} \quad (2)$$

$$e_{i(j_1 j_2)} \sim N(0, \sigma_e^2), u_{1j_1} \sim N(0, \sigma_{u1}^2), u_{2j_2} \sim N(0, \sigma_{u2}^2) \quad (3)$$

where $y_{i(j_1 j_2)}$ is the dependent variable (in our case change in age-cohort specific mortality from the previous year) for individual (or in our case age-period measurement) i in cohort group j_1 and year of measurement j_2 . u_{1j_1} represents the cohort random effects and u_{2j_2} the period random effects, both of which are assumed to be normally distributed.

When considering age, period, and cohort there is a problem that by knowing two variables we can perfectly predict the other: age equals period minus cohort, so the three variables have only two degrees of freedom. This is referred to as the 'identification problem' (Glenn, 2005; Bell and Jones, 2013). The HAPC model (Reither et al., 2009), as well as the 'intrinsic estimator' (Yang and Land, 2006; Yang et al., 2008), are

attempts to statistically separate the three components. Unfortunately the HAPC model, like the intrinsic estimator, has been shown to apportion linear trends in ways that often do not fit with the true data generating processes (DGPs) (A. Bell and Jones, 2014a; 2014b; 2017; Luo, 2013; Luo and Hodges, 2015).

In our case, however, we are interested only in non-linear period and cohort stochastic fluctuations, once the age, period and cohort long-run trends are controlled. As such we can control for these trends in the fixed part of the model, leaving only discrete deviations in the random part of the model (see Chauvel et al. (2016)). Whilst we cannot control for all three of APC in the fixed part of the model because of the identification problem, controlling for two of APC will control out the linear component of the third by default. Thus, we can extend Equations (1) – (3) to include an additional cohort term within $\beta_{0j_1j_2}$, producing:

$$y_{i(j_1j_2)} = \beta_{0j_1j_2} + \beta_1 Age_{i(j_1j_2)} + \beta_2 Age_{i(j_1j_2)}^2 + \epsilon_i(j_1j_2) \quad (4)$$

$$\beta_{0j_1j_2} = \beta_0 + \beta_3 Cohort_{j_1} + u_{1j_1} + u_{2j_2} \quad (5)$$

$$e_{i(j_1j_2)} N(0, \sigma_e^2), u_{1j_1}, N(0, \sigma_{u1}^2), u_{2j_2} N(0, \sigma_{u2}^2) \quad (6)$$

We cannot trust the estimates of β_1 or β_3 (because they will incorporate any period linear effects if they exist in the DGP), but we are not particularly interested in their estimates. We can say that u_{1j_1} and u_{2j_2} will be accurate estimates of deviations from the long-run trends in periods and cohorts (whatever they are), and we can be confident (linear) APC trends will not be included in those estimates. The models were fitted in R with the `lme4` package version 1.1-13 (Bates et al., 2015) and random effects were extracted using the `nlme` package version 3.1-131 (Pinheiro et al., 2017).

Full HAPC results tables can be found in an online appendix. It should be noted, however, that the APC fixed terms should not be interpreted because of the APC identification problem.

Results

Figure 1 shows a Lexis surface for mortality change in England and Wales, with blue and green representing a decline in mortality, and red and orange representing an increase in mortality on the previous year for a given age of person. Cohort effects appear as diagonal ‘scars’ (Minton et al., 2013), emanating through

Figure 1: England and Wales total population Lexis surface plot for annual change in age-specific mortality

age-time upwards and rightwards from the affected birth cohorts.

A period effect shows as a vertical red line immediately followed by a vertical blue line, or vice-versa. A cohort effect will show as a diagonal red line immediately followed by a diagonal blue line, or vice-versa. A red line followed by a blue line might represent temporary excess mortality caused by an event such as the influenza pandemic. A blue line followed by a red line would represent a temporary decrease in mortality, that later returned to its previous level. A mild winter might exhibit such an effect if excess winter deaths are lower than neighbouring years. Figure 1 shows evidence of both period and cohort effects in England and Wales.

In addition to these effects, it is possible to see fundamental changes in age-specific mortality change, where a decrease in mortality change is not followed by an increase, and vice-versa. A lone red line represents a long-term increase in mortality rate, for example caused by an enduring economic crash and recession. A blue line without a corresponding red line would represent a long-term decrease in mortality rate. Figures 2 and 3 show the period and cohort residuals from the adapted HAPC model respectively, demonstrating the non-linear period and cohort fluctuations based on the same data as Figure 1.

Most noticeably a cohort effect with increased mortality is detectable in both males and females born in 1918. Whilst this could be in part due to World War One, given the lack of effect for those born earlier in the war, it seems likely that this is primarily the result of the 1918 influenza pandemic. A period effect is also clearly visible around the year 1918 in females and males under the age of about 55. A sharp increase in mortality is followed by a commensurately sharp decrease, indicating a sudden increase in deaths which then returned to the previous level. For males the period mortality is accompanied by increases in mortality in the preceding years for those between ages 15 and 35. This high increase in mortality is concentrated in young men during

Figure 2: Plot of year (period) residuals for males from the adapted HAPC model. The residuals can be interpreted as a difference in mortality compared between the previous year to the current year, over and above any linear APC trends

Figure 3: Plot of birth year residuals for males from the adapted HAPC model. The residuals can be interpreted as a difference in mortality compared between the previous birth year to the current birth year, over and above any linear APC trends.

the entirety of the First World War, reflecting the increasing deadliness of this conflict.

Another period effect appears around the Second World War. Both males and females from birth to old age exhibit higher period mortality in the year around 1939. This suggests either civilians suffered greater exposure to the conflict or environmental conditions worsened during this time, or both. Both males and females show improved mortality rates in the years immediately following the end of the war, reflecting the rise of welfare policies around this time. There does not appear to be a cohort effect for those born during the Second World War.

A less obvious, but nonetheless present, change in mortality rate is a cohort effect observed among males and females born in the year 1948 in the UK. This is visible in Figures 1 and 3. A small drop in mortality is evident in 1948, and this is not followed by a comparable increase in mortality in the following years. The effect is small, but does suggest people born in 1948 and later experienced a lower mortality rate throughout their lifecourse than individuals born even just one year previously.

The obvious change that occurred in the UK at this time was the formation of the NHS. If the NHS is indeed the cause of this improvement in cohort mortality, the implication is that being *born* under the NHS institution gave an advantage in terms of mortality. Whilst those born just prior to 1948 lived the majority of their lives under the NHS, the mortality rate for this cohort is higher than those born after the formation of the NHS.

It is possible, therefore, that the NHS improved population-level mortality, in particular for those who were born under the system, and not for those who were born just one year prior even if those individuals still lived with the NHS for the vast majority of their lives. This could be for a number of reasons. First, pre-natal and early life care are particularly important in improving mortality for individuals throughout their lives. Second, the NHS may have had a cultural effect on those born under it—and their parents—making them more likely to seek treatment through it.

The formation of the NHS was situated within a context of high employment, the implementation of welfare policies such as the National Assistance Act (1948)—which was itself an addition to the National Insurance Act 1946 which introduced social protections, nationalisation of energy and rail transport, and substantial financial aid from the United States in 1946 and again in 1947 under the European Recovery Program (often called the ‘Marshall Plan’) (Hill, 1970 p. 291; Medlicott, 1967).

Whilst the localisation of this effect to 1948 implies the NHS is important in this, the other policies that occurred at similar times in the UK and elsewhere could also help explain some of the effect. For instance, the provision of social welfare may have improved the nutrition of the most at-risk mothers sufficiently to

mitigate the most extreme malnutrition in future pregnancies. The winter of 1946–47 was especially harsh with fuel and food shortages reported from late January 1947. If the severity of this winter affected the nutrition available to pregnant mothers it may have also affected the later morbidity and mortality of their children born up to late 1947. It is possible the lower mortality in 1948 is partially explained by the returning to background levels of mortality after the small increase in 1946–47.

It should be noted that the UK was not alone in seeing similar effects occurring in 1948. Both Canada and the USA shared this pattern (Figures 6 and 7, respectively). Given the lack of the NHS in these countries, this requires some explanation; we propose a number of reasons. First, all of these countries experienced a revival in health and welfare policy after the war, and the finding could be a result of a more general improvement in health and welfare provision. However, this does not provide a clear reason why this would happen specifically in 1948, and not before or after.

Second, there may be another factor causing the effect in all three countries, independent of the NHS's formation. For example, penicillin was first produced in bulk during the early 1940s, but became more accessible to patients as costs were driven down during the mid- to late-1940s. Coupled with the formation of the NHS, it is possible that penicillin became more *accessible* in 1948 in the UK, as well as in the US, and other countries leading to reductions in cohort and period mortality. Penicillin could have been used both to treat young children, and also to treat new mothers (particularly for postpartum infections), improving survival rates of mothers in labour and thus, plausibly, the life outcomes of their children. However, penicillin also became more accessible in countries such as Portugal in 1947–48 (Bell et al., 2017), but Portugal shows an *increase* in cohort mortality in 1948 (Figure 4) suggesting, if penicillin availability were partly responsible for decline in cohort and period mortality, the picture is complicated by other factors.

Third, the formation of the NHS could have had a cultural effect beyond the UK's border, improving the way healthcare provision was used even without a sudden change in policy. Fourth, the effect could be a statistical artefact if, for instance, the UK's data were used to impute missing mortality data in Canada and the USA, or vice-versa. This work is very much descriptive and more work would be needed to confirm these hypotheses.

The Lexis surface and HAPC plots above have been repeated for a number of western countries, and these can be found in the online appendix. Whilst there are too many interesting patterns in these plots to cover in detail, we pick out a few notable patterns here.

Based on the plot for The Netherlands a decline in mortality around World War II is detectable in the Dutch population (figure 5). The Lexis surface plot shows increased mortality for all ages and both sexes during the

Figure 4: Portugal female cohort mortality

Figure 5: Lexis surface plot of mortality for The Netherlands

Figure 6: Canada female cohort mortality

Second World War, but for a greater time period beginning in 1940. This suggests a greater exposure to the conflict for the Dutch civilian population than the UK population. As The Netherlands was occupied from May 1940 until 1944–45 this is to be expected.

Of particular interest is the mortality rate in the year 1945, where the increase in mortality spans a much greater age range. Much of The Netherlands was liberated in 1944, but several areas to the north east remained occupied during the winter of 1944–45. It is these areas that suffered the *Hongerwinter* (Warmbrunn, 1963 pp. 14–17). Therefore, it is possible that much of the increase in mortality rate observed in 1945 could be because of the famine in occupied areas of The Netherlands, before the mortality rate recovered following the end of the Second World War.

Figure 7: USA female cohort mortality

Conclusions

This paper has explored period and cohort effects on UK mortality during the twentieth century, using Lexis surface plots and hierarchical age–period–cohort models. The paper makes both a substantive and methodological contribution. Substantively, we have shown where key events in the UK appear to have affected national mortality rates, both as period effects and cohort effects. In particular, World Wars I and II both appear to have had period effects on male mortality, whilst the influenza pandemic of 1918–19 appears to have had both a period and cohort effect on mortality. The formation of the NHS also appears to have had a cohort effect on mortality, although this conclusion can only be tentative given the appearance of similar effects in other countries.

Methodologically this paper has shown the value of APC analysis of non–linear stochastic variation, both using statistical methods (such as the adapted HAPC model) and graphical techniques (such as the use of Lexis diagrams). These techniques can be used to assess a range of outcomes across the health and social sciences, wherever age, period, and cohort stochastic effects are of interest.

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