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The Boomer Penalty:

Excess Mortality among Baby Boomers in Canada and the United States[*]

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Abstract

Studies suggest that, relative to adjacent cohorts, baby boomers in Canada and the United States have experienced a slowdown, or even a deterioration, in mortality improvements. These findings are counterintuitive and surprising since the unprecedented improvements in early life conditions experienced by baby boomers should have led to declines in morbidity and mortality in later life, as was the case for earlier generations.

The present study explores the mechanisms that could have produced this "excess" mortality among the baby boom cohorts in Canada and in three racial/ethnic groups in the United States. Using micro-level mortality data from vital statistics systems, we analyzed the contributions of the causes of death that are likely driving this cohort's excess mortality, and their dynamics over time. The analyses were done using demographic decomposition, visual, and statistical methods.

We found evidence of a higher susceptibility of the trailing edge boomers (those born around 1960) to behavioral causes of death: namely, mortality from drugs, alcohol, HIV/AIDS, hepatitis C, COPD, and suicide. Most of these causes contributed to the all-cause mortality disadvantage of baby boomers through sustained cohort effects that followed the cohorts over time. This finding calls into question the assumption that secular improvements in early life conditions lead to a monotonic decline in cohort mortality rates. Instead, there may be important disruptions in the continuous progress in health and mortality, and it is possible that the baby boom generation represents one such disruption. This insight calls for a rethinking of the mechanisms that drive current age-period-cohort mortality patterns. The mechanisms that can generate the observed cohort disadvantage of baby boomers – such as the higher levels of distress and frustration as well as the riskier attitudes toward drug use and sexual practices that are constituent of the boomer generation identity – are addressed and discussed.

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1. Introduction

Previous research has established that all-cause mortality improvements slowed down or even reversed among baby boomers in Canada (Bourbeau and Ouellette 2016) and the United States (Canudas-Romo and Guillot 2015; Rau et al. 2013). These findings were unexpected, and they were especially surprising given the outcomes proposed by the technophysio evolution theory. According to this theory, the unprecedented gradual improvements in early life conditions experienced in recent history – such as better nutrition, reduction of infectious diseases, enhanced medical measures, and higher levels of education – have led to massive declines in mortality across birth cohorts (Floud et al. 2011; Fogel and Costa 1997). Other studies focusing on specific causes of death also identified mortality disadvantages for U.S. baby boomers with respect to overdoses (Chauvel et al. 2016) and other external causes (Remund et al. 2018; Zang et al. 2019). Moreover, it has been shown that U.S. boomers report being less satisfied with their health than adjacent birth cohorts, and that they tend to have a high prevalence of obesity, diabetes, hypertension, hypercholesterolemia, substance and alcohol abuse, as well as functional limitations (Duncan et al. 2010; King et al. 2013; Leveille et al. 2005; Martin et al. 2009). There are at least four alternative mechanisms that may drive this "boomer penalty" in mortality. First, the relatively low levels of mortality at early ages among baby boomers could have increased the health heterogeneity within the cohort, resulting in a decrease of the average physiological capital of its members (Canudas-Romo and Guillot 2015). Second, the large sizes of the boomer cohorts, compounded by the wide range of socioeconomic contexts they experienced in childhood and adulthood, may have increased the prevalence of stress and frustration among the members of this generation (Easterlin 1987). Third, the distinctive attitudes toward risk and the risk-taking behaviors associated with the boomers' generational identity could have increased their mortality risks (Johnston 1991). Finally, the slowing or absence of mortality improvements observed among the boomers may have little to do with the usual cohort effects, and might instead result from the many unrelated period crises that affected these cohorts at different life stages.

Although this phenomenon has contributed substantially to the mortality patterns in several western societies, the boomer disadvantage in mortality is still poorly understood. The existing studies on this topic have tended to emphasize long-term cohort changes, while overlooking short-term relative differences across adjacent cohorts. The research to date on the boomers' excess mortality has not

determined its causes or described its temporal dynamics. Furthermore, there is still uncertainty about whether the composition and the temporal patterns of excess mortality in the boomer generation are similar across sexes, race, ethnicities, and countries.

This paper attempts to compare the cause-specific contributions to the excess mortality among baby boomers, and the temporal patterns of these contributions. This study has two primary objectives: 1) to examine the causes of death underlying the excess mortality among baby boomers in Canada and the United States; and 2) to determine whether this disadvantage is the result of a sequence of unrelated period crises that disproportionately affected the boomer cohorts at different ages, or of a sustained, cause-specific disadvantage that has followed the boomers throughout their life course. In other words, we aim to determine whether the boomers' excess mortality resulted from a series of temporary "bruises" aligned diagonally in the Lexis configuration, or from lasting "scars" with lingering effects (Chauvel 2013; Ellwood 1982). The methodological approach taken in this study is based on decomposition techniques, age-period-cohort (APC) statistical models, and visual tools for analyzing the temporal dynamics of nonlinear effects. These methods were applied to mortality data retrieved from the Canadian and the U.S. vital statistics systems.

To our knowledge, this is the first comprehensive analysis of the composition and the temporal dynamics of the disadvantage in mortality among baby boomers in Canada and across races and ethnicities in the United States.

2. Data and Analytical Strategy

2.1. Data sources

Death counts for all-cause mortality for both countries between 1959 and 2016 were obtained from the Human Mortality Database (2019). Mortality data by cause were retrieved from available vital statistics. For Canada, death counts by sex, calendar year, single year of age (0-100), and cause of death between 1974 and 2014 were aggregated from the Vital Statistics - Death Database (CVSD) (Statistics Canada 2018). For the United States, death counts by cause, sex, race, ethnicity, calendar year, and single year of age between 1974 and 2016 were retrieved using mortality microdata from

the National Center for Health Statistics (2018). Information about race and ethnicity has been included in U.S. death certificates since 1990, but is unavailable for Canada.

The period under analysis spans three International Classification of Diseases (ICD) revisions (8th, 9th, and 10th). Table S1 (in the supplement material) shows the codes used to identify mortality by major causes of death in each ICD revision. For these broad categories, we found no important disruptions in the trend of mortality from these causes during the observed period of time.

Annual counts of the Canadian and the U.S. population at risk by sex and single year of age (0-100) between 1959 and 2016 were taken from the Human Mortality Database (2019). Estimates of the racial/ethnic proportions within the U.S. population between 1990 and 2016 were obtained from the Bridged-Race Population Estimates (NVSS 2019).

2.2. Analytical Strategy

We analyzed the excess mortality among baby boomers in four steps. We first located the cohorts with the smallest and the largest mortality deviations from the linear trend, and called them, respectively, the *advantaged* and the *disadvantaged* cohorts. Second, we identified the leading causes that contributed to these mortality deviations. Third, for each of these leading causes, we estimated the cohort effects on mortality when age and period variations are accounted for. Fourth, we analyzed the temporal dynamics of these cohort effects by cause. Because Canadian and U.S. populations are highly heterogeneous in terms of race and ethnicity, and mortality differs considerably between these groups (Masters 2012; Woolf et al. 2018; Zang et al. 2019), we also conducted the above analyses separately for three racial/ethnic groups in the U.S. population: namely, Non-Hispanic blacks (NHB), Hispanics, and Non-Hispanic whites (NHW). For the sake of clarity, we present for each step of the analysis a detailed description of the methods used, immediately followed by a summary of the results obtained from the application of these methods. All of the data and code for reproducing analyses and results are openly available in (Acosta 2020).

# 3. Analysis of the Boomers' Excess Mortality

3.1. Cause-specific contributions to the boomers' excess mortality

To decompose the excess mortality among boomers by leading causes of death, we first identified those cohorts located at the beginning (advantaged) and the end (disadvantaged) of the relative deterioration in mortality. Second, we identified the leading causes of death responsible for this deterioration.

Identification of the advantaged and disadvantaged cohorts

We first plotted Lexis surfaces of smoothed mortality changes from one period to the next within the same age (see Figure 1), and then pinpointed the cohorts located at the onset and at the end of the deterioration in mortality – i.e., the *advantaged* and the *disadvantaged* cohorts, respectively. The smoothing of mortality rates was performed using two-dimensional P-splines. Additional information about the smoothing process, the estimation of the relative changes in mortality, and the construction of the Lexis surfaces is presented in the supplementary materials.

Figure 1. Lexis surfaces of mortality changes over periods/cohorts.
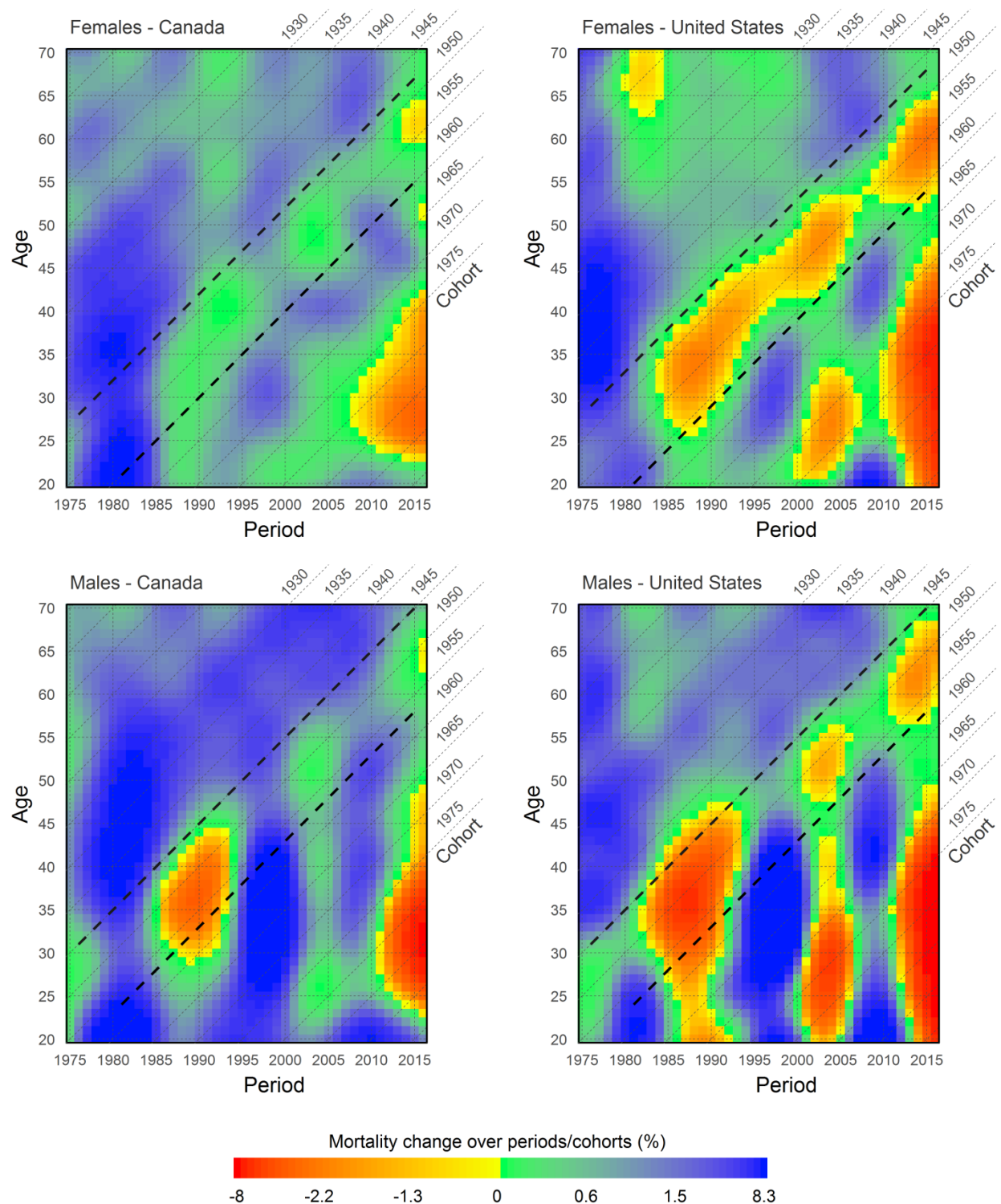
Notes: Read horizontally from earlier to more recent calendar years/cohorts. The green-to-blue scale indicates the mortality rate decline for year t compared to year t-1 (or cohort c compared to cohort c-1) at the same age, and the yellow-to-red scale indicates a relative mortality increase between consecutive calendar

years/cohorts. For example, if we examine age 50 for U.S. females, we see that the death rate decreased over time, reaching its minimum value in 2000 (i.e., cohort 1950). We can also see that from 2000 to 2006, the death rate increased over time, reaching its maximum value in 2007 (i.e., cohort 1956). The diagonal black dashed lines indicate the proximate location of the advantaged and the disadvantaged cohorts for each subpopulation.

The diagonal patterns shown in Figure 1 indicate that the *advantaged* and the *disadvantaged* birth cohorts were centered in 1940 and 1960, respectively (black dashed lines). To identify the precise locations of these cohorts in the Lexis configuration, we needed to compare the mortality rates across cohorts. For this purpose, we proposed an index of the cohort's partial mortality rate ($CPMR^{c(k,l)}$). This index was the sum of the age-specific death rates along the cohort c, between ages k and l. See the supplementary materials for more details about the formulation and attributes of the $CPMR^{c(k,l)}$.

Since our goal was to identify relative and not absolute mortality changes, we based our analysis on the deviation from the linear trend in mortality. We obtained the linear trend by applying a linear regression over the $CPMR^{c(k,l)}$ estimates between the cohorts 1940 and 1960. The cohorts with the largest negative and positive differences relative to the linear trend were labeled, respectively, as the *advantaged* and the *disadvantaged* cohorts. To compare mortality rates across cohorts over the longest possible lifespan, we estimated the $CPMR^{c(k,l)}$ for the age interval 35-54 between the cohorts 1940 and 1960. These estimates cover the period 1975-2014, as 2014 was the last year for which information about causes of death was available for Canada.

Figure 2 displays the estimates of $CPMR^{1940(35,54)}$ to $CPMR^{1960(35,54)}$ (solid lines), as well as the respective linear trends (dashed lines). As expected, the analysis showed that males had higher levels of mortality than females within each country. Mortality was considerably higher in the United States than in Canada (~40% higher for females and ~70% higher for males). Indeed, for the cohorts born at the end of the 1950s, the mortality levels of U.S. females and Canadian males were quite similar. In absolute terms, the male and female $CPMR^{c(35,54)}$ deteriorated in the United States, whereas in Canada, mortality improvement stagnated, but mortality did not deteriorate.

Figure 2 identifies the cohorts with the largest negative and positive deviations from the $CPMR^{c(35,54)}$ linear trend. These cohorts are labeled, respectively, the *advantaged* (circles) and the

disadvantaged (triangles) cohorts. Whereas the advantaged cohorts are located in proximate birth cohorts for all subpopulations (between 1947 and 1949), the disadvantaged cohorts of U.S. males were born considerably earlier (1952) than the disadvantaged cohorts of the other groups (between 1957 and 1960). The selection of the disadvantaged cohorts for Canadian males and U.S. females was not as straightforward as it was in the other cases. To test the consistency of our estimates, we performed sensitivity tests in which we changed the location of the disadvantaged cohorts and the cohort intervals under observation. These estimates are presented in the supplemental materials (Figures S5.3 to S5.5).

Figure 2. Cohort's partial mortality rate within the age interval 35-54
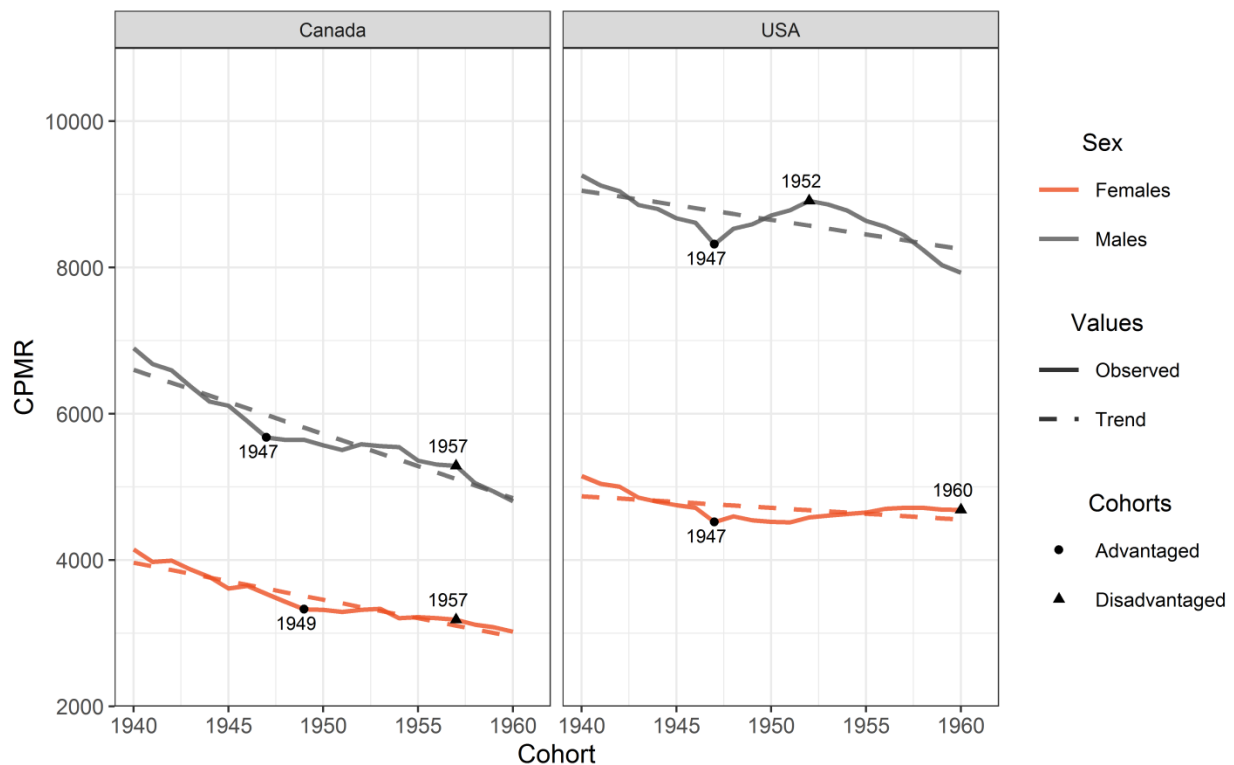
Notes: $CPMR^{c(35,54)}$ by country and sex (solid lines), and their respective linear trends (in dashed lines). The points and labels indicate the year of birth of the *advantaged* (circles) and the *disadvantaged* (triangles) cohorts.

Decomposition of the cohorts' excess mortality by cause of death

After we established the precise locations of the *advantaged* and the *disadvantaged* cohorts using the $CPMR^{c(35,54)}$, we proceeded to decompose the mortality changes between the two cohorts ($\Delta CPMR^{d-a(35,54)}$) by causes of death. See the supplementary materials for additional information about this decomposition.

The decomposition of $\Delta CPMR^{d-a(35,54)}$ into broad categories of causes of death showed that the largest contributions to the deterioration in mortality across cohorts, regardless of country and sex, were from external causes, infectious and parasitic diseases, diseases of the digestive system, mental and behavioral disorders, and diseases of the respiratory system. In all subpopulations, the contributions of external causes and infectious diseases were especially large (see Figure S5.1 in the supplemental materials).

Next, we disaggregated these broad causes into more detailed causes of death and re-estimated their contributions to mortality changes. The ICD codes used to identify deaths from these causes are detailed in Table S2 in the supplemental materials. The six causes with the largest positive contributions to mortality changes (i.e., mortality deterioration) across cohorts were those related to alcohol, drugs, HIV/AIDS, hepatitis C, chronic obstructive pulmonary disease (COPD), and suicide (see Figure S5.2 in the supplementary materials). These six leading causes of together contributed ~75% - 80% of the total positive changes in mortality from the advantaged to the disadvantaged cohorts in all groups (Figure 3). The relative cause-specific contributions were consistent across subpopulations, with two exceptions. First, for U.S. males, HIV/AIDS was the cause that made the largest positive contribution, whereas for the other groups, the cause that made the largest positive contribution was drugs. Second, for Canadian males, the relative contribution of suicide (~25%) to the total positive changes in mortality was considerably larger than in any other group (~5-10%). In the next steps of the analysis, we focus on these six leading causes of death as we attempt to determine the magnitude of the cause-specific cohort disadvantages and their temporal dynamics.

Figure 3. Cumulative contribution to positive $\Delta CPMR^{d-a(35,54)}$ (i.e., mortality deterioration) by cause between the *advantaged* and the *disadvantaged* cohorts
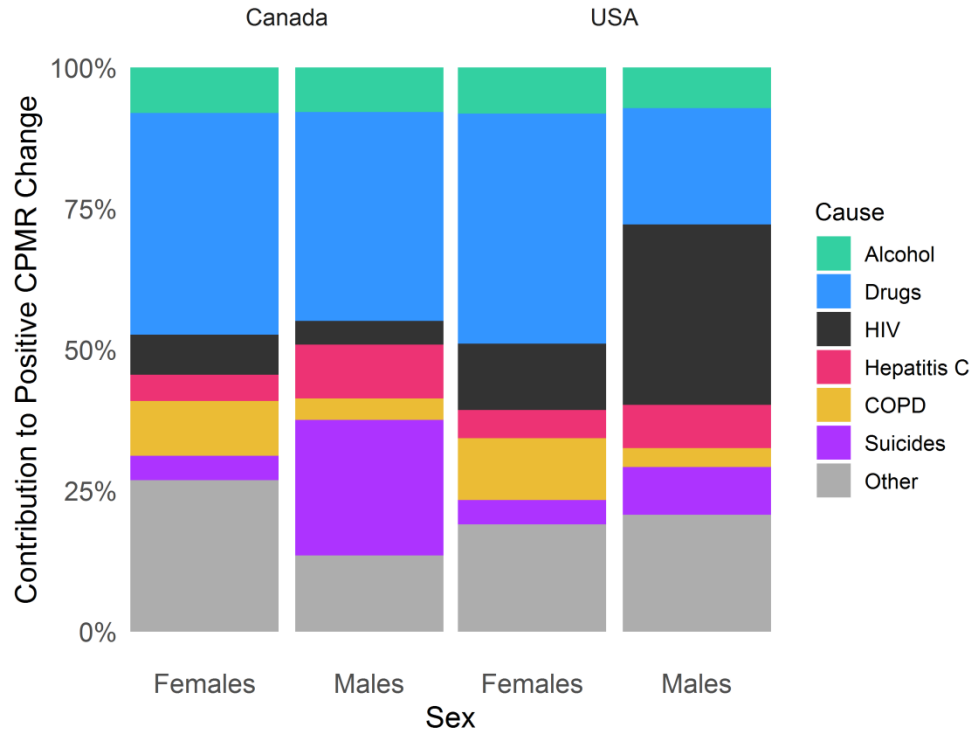
3.2. The magnitude and the temporal dynamics of the boomer cohorts' disadvantage

Detrended cohort effects on mortality by cause of death

The estimates from the $\Delta CPMR_i^{d-a(35,54)}$ reported in Figure 3 were useful for identifying the causes of death that made the largest contributions to the relative deterioration in mortality from the *advantaged* to the *disadvantaged* cohorts. However, to properly assess the cohort penalty by cause of death, we need to account simultaneously for variations over the three age-period-cohort dimensions (A further discussion on this topic is presented in the supplementary materials). We should, however, note that the use of APC models has been challenged because of the well-known *identification problem*, in which the perfect multicollinearity between the variables (*cohort = period − age*)

results in an infinite number of solutions with an identical fit. While several attempts have been made to solve this problem by imposing arbitrary restrictions on the APC models (e.g., constrained generalized linear (Fienberg and Mason 1985) and intrinsic estimator (Yang et al. 2004) models), these approaches have been strongly criticized (Bell and Jones 2013; Fienberg 2013; Fosse and Winship 2018, 2019; Luo 2013).

Nevertheless, this limitation of the APC models is exclusively related to the partition of the linear trend (i.e., linear effects) into age, period, and cohort components (Rodgers 1982). In the present study, we were not interested in decomposing the linear effects, but were instead concerned with analyzing the deviation of the mortality of the boomer cohorts from the linear trend. As these divergences from the linear trend – which are also referred in the APC literature as nonlinear effects, curvatures (Holford 1983), or humps (Chauvel et al. 2016; Remund et al. 2018) – were unaffected by the constraints chosen for the model identification, they were unambiguously identifiable (Clayton and Schifflers 1987; Holford 1983; Rodgers 1982).

For the analysis of the cohort effects on mortality by cause, we estimated the relative mortality risks across cohorts (i.e., the nonlinear cohort effects) using a cohort-detrended APC model (APCd) (Carstensen 2007; Chauvel 2013). In this approach, the linear trend is attributed entirely to variations over age and period dimensions, which results in a series of cohort components with zero slope. Under this parameterization, the logarithm of the cohort effects can be interpreted as relative risks with respect to the overall linear trend (Carstensen 2007; Holford 1991). To estimate the APCd model, we grouped ages, periods, and cohorts into two-year categories, and fitted splines to a Poisson model, using the R package Epi (Carstensen et al. 2019).

Comparisons between Canada and the United States

Figure 4 and Table S3 show the relative risks and confidence intervals obtained from the APCd model by country, sex, and cause of death (see Figure S5.6 in the supplemental materials for a facetted version of Figure 4). In all boomer groups, the largest cohort disadvantages were observed for hepatitis C, HIV/AIDS, and drug abuse mortality (with relative risks between 1.7 and 3.0), while the smallest cohort disadvantages were found for alcohol abuse, suicide, and COPD (with relative risks between 1.2 and 1.5). The main difference detected between the two countries was in the pattern of mortality due to COPD, for which only the U.S. boomers had a risk that was higher than the overall cohort average.

The mortality patterns of Canadian females differed considerably from those of their male counterparts and from those of U.S. males and females. For all causes of death, the Canadian female boomers had little to no disadvantage. For alcohol, suicide, and COPD, the mortality risks of this group were not significantly higher than the overall cohort average. In contrast, boomer males in Canada and boomer males and females in the United States had similarly high relative risks of dying from hepatitis C, HIV/AIDS, drugs, and suicide. More precisely, these three subpopulations of boomers had the largest disadvantages in mortality for causes related to drugs, HIV/AIDS, and hepatitis C; moderate disadvantages for causes related alcohol; and considerably smaller disadvantages for death from suicide.

Figure 4. Cohort relative risks by country, sex, and cause of death
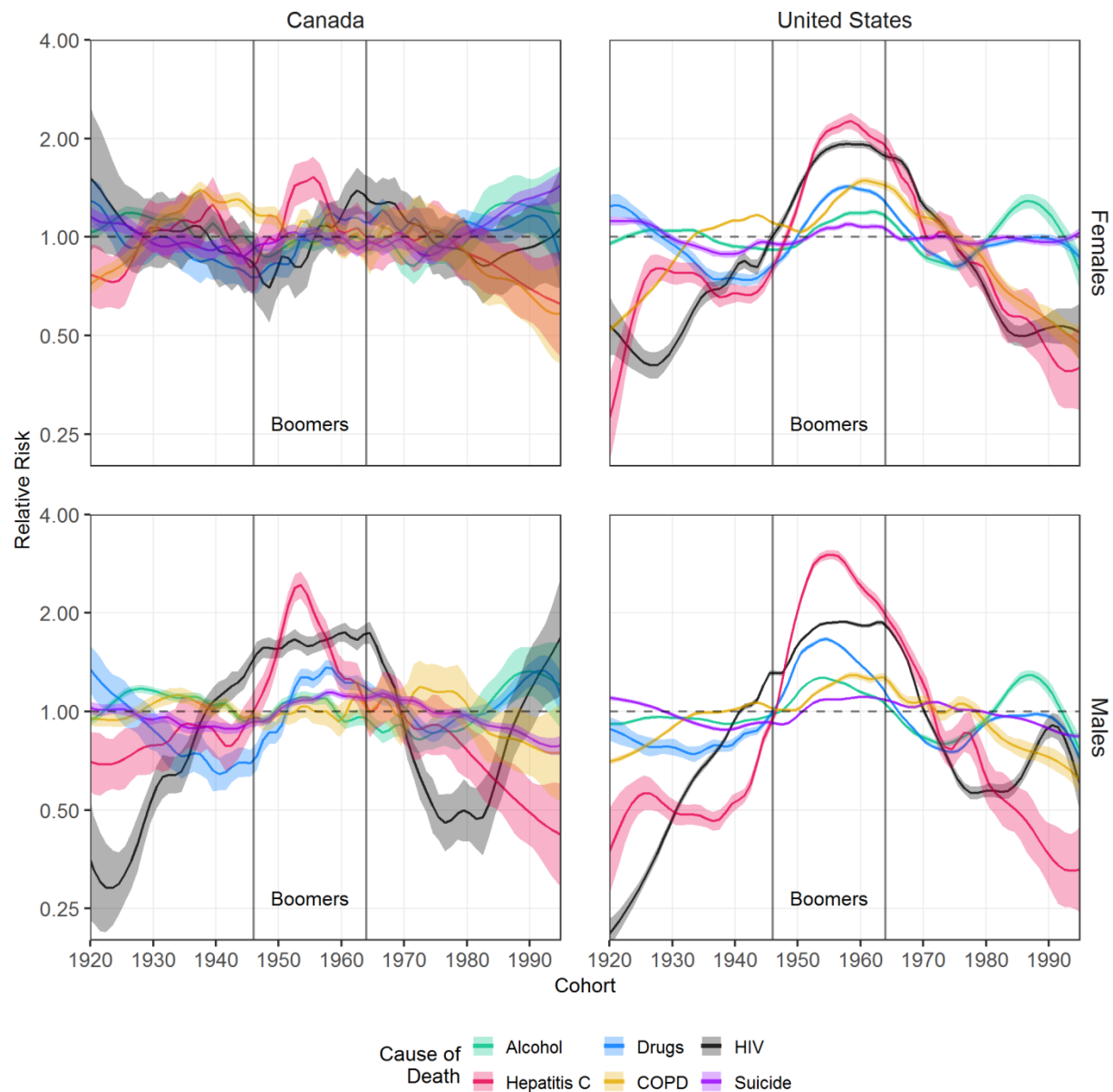
Notes: The width of the ribbon indicates the confidence interval at the 95% level. Estimates were obtained from a cohort-detrended model (APCd). The reference category is the overall cohort average, depicted in the plot with a horizontal dashed line. The beginning and end of the baby boom (i.e., 1946 and 1964, respectively) are marked with vertical gray bars.

Comparisons across races and ethnicities in the United States

In order to explore the disparities across racial and ethnic groups in the United States, we estimated detrended cohort effects as cohort relative risks for the NHB, Hispanic, and NHW populations (Figure 5; see also separate plots in Figure S5.7 in the supplemental materials). Except for a few cases, the female and male boomers in the United States had similar cause-specific cohort disadvantages, regardless of their race or ethnicity. The causes that contributed the most to the boomers' disadvantages in all subpopulations were hepatitis C and HIV/AIDS, and the cause that contributed the least was suicide.

The main difference between the sexes in the causes that contributed to the boomers' mortality disadvantage was that hepatitis C posed a much greater risk for males (reaching a maximum of a 2.5-fold risk on average) than for females (2.1-fold risk on average), regardless of race/ethnicity.

When analyzing the cause-specific contributions to the boomers' mortality disadvantage by race and ethnicity, two results stand out. First, the NHW boomers of both sexes were the only groups with a significantly higher relative risk of suicide mortality. Second, although boomers of all groups had a large drug-related mortality disadvantage, the relative risks for NHB (1.9 for females and 2.0 for males) were substantially higher than those for Hispanics (1.32 and 1.45) and NHW (1.3 and 1.5).

Figure 5: **Cohort relative risks by race/ethnicity within the U.S., sex, and cause of death**
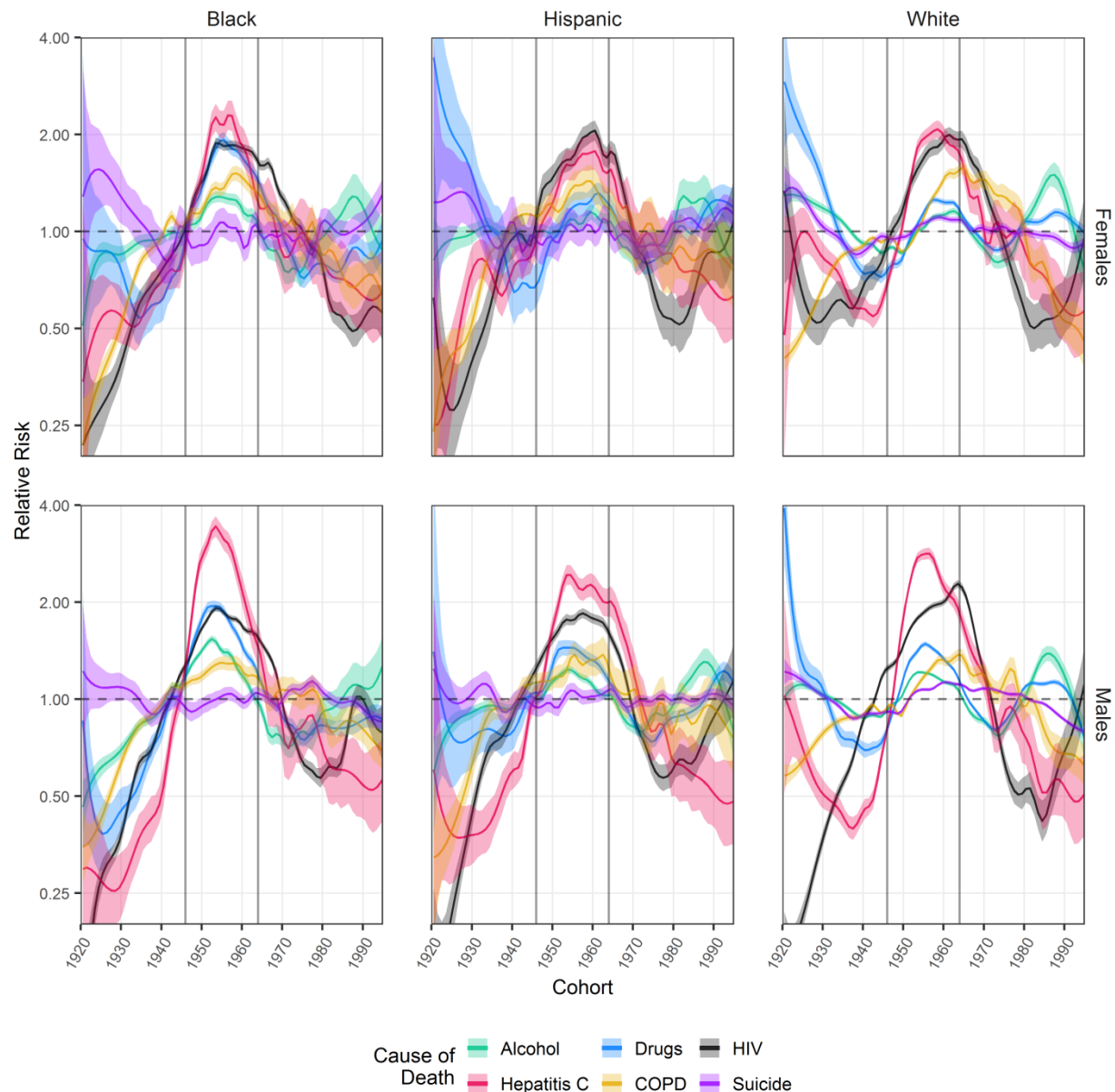
Notes: The width of the ribbon indicates the confidence interval at the 95% level. Estimates were obtained from a cohort-detrended model (APCd). The reference category is the overall cohort average, depicted in the plot with a horizontal dashed line. The beginning and the end of the baby boom (i.e., 1946 and 1964, respectively) are marked with vertical gray bars.

In summary, these results show that, with few exceptions, boomer cohorts had higher relative risks of dying from most of the causes that were making the largest contributions to the deterioration in

mortality from the advantaged to the disadvantaged cohorts, regardless of national context, sex, and race/ethnicity. Three exceptions are, however, noteworthy. First, among Canadian female boomers, the relative risks of mortality were significantly higher only for causes related to drugs, HIV, and hepatitis C. Second, only boomers in the U.S. had a significant disadvantage in COPD mortality. Third, only Canadian males and U.S. NHW males and females had a significantly higher relative risk of mortality from suicide. In the next section, we analyze the temporal dynamics of these cause-specific cohort disadvantages.

Dynamics of the cohorts' excess mortality over time

The APCd estimates presented above were useful for the estimation of the average cohort disadvantage. However, these estimates were of limited use for the analysis of the temporal dynamics of nonlinear effects because, as averages, they did not allow us to observe variations in relative risks over time (Chauvel 2013). The changes in the magnitude or the location of the curvatures by cause over time could help us determine whether the cohort disadvantage resulted from a sequence of temporary age-period interactions, or, conversely, from a process that operated continuously throughout the life course of the boomers. To analyze these temporal dynamics, we constructed *APC curvature plots* (Acosta and van Raalte 2019). This graphical tool allowed us to display the changes in the nonlinear APC components over time on a Lexis diagram by focusing on the ridges; i.e., the series of Lexis coordinates in which the relative risk reaches a maximum. These plots helped provide synthetic information on nonlinear APC effects simultaneously across several populations or for several causes of death.

To construct the *APC curvature plots*, we needed to extract the boomer curvatures – in this case, the excess mortality by cause of death. To do this, we first estimated a mortality baseline by excluding the cohorts that diverged from the secular trend in mortality, and interpolating the mortality rates. The excess mortality was defined as the difference between the observed and the interpolated surface. The interpolation of the mortality rates was estimated with two-dimensional P-splines, and performed with the R package *MortalitySmooth* (Camarda 2012). The measured excess mortality was then translated into visual attributes: the locations of the ridge over time (i.e., the age/cohort for which the positive divergence in mortality reached its maximum level in each period) were indicated through the coordinates position in the Lexis diagram; and the magnitude (i.e., the relative risk in the

ridge compared to in the base of the hump) was indicated by the point size. We constructed *APC curvature plots* to compare the cohorts' excess mortality across causes of death, countries, and races/ethnicities simultaneously in the same figure.

### Comparisons between Canada and the United States

The APC curvature plots presented in Figure 6 show the temporal dynamics of excess mortality (i.e., the age/cohort with the greatest excess mortality in each period) by cause of death, country, and sex (see also Figure S8 in the supplementary materials). We first describe the results for HIV/AIDS-related mortality. The patterns for HIV/AIDS were similar across subpopulations, but differed considerably from the patterns for the other causes. Between the mid-1980s and the mid-1990s, the ridge for HIV/AIDS-related excess mortality largely surfaced in the diagram as an age effect for both sexes in Canada and the United States, with young adults (ages 25-35) being disproportionately targeted. However, after the turn of the 21st century, the ridges for HIV/AIDS shifted and began to align diagonally toward a cohort pattern for all groups, with some noticeable differences. Whereas Canadians of both sexes and U.S. males born around 1965 were most susceptible, the largest disadvantage for U.S. females was observed for those born around 1955. For Canadian females, this cohort effect was interrupted in the second part of the 2000s, when it shifted once again to being an age effect, but this time targeted those aged ~40.

The locations of the ridges of excess mortality related to hepatitis C, drugs, and alcohol were similarly centered on the cohorts born around 1955-1960, with the relative risks of dying from drugs and alcohol being considerably lower. The main exception was for Canadian females, who did not have a sustained cohort mortality disadvantage related to alcohol, suicide, or COPD. Whereas the COPD disadvantage did not follow any clear pattern, the horizontal traces of the alcohol and suicide ridges were more indicative of short-term age-period interaction effects.

Turning now to the variation in the magnitude of the cohort disadvantage over time (see also Figure S5.8 in the supplemental materials), we can see that the relative risks of HIV/AIDS mortality were considerably higher at the turn of the 1990s for all groups (up to 10.7), and for U.S. males in particular. Nevertheless, at the turn of the century, the relative risks of HIV/AIDS mortality decreased sharply, and remained below 1.8 during the rest of the period. Apart from HIV/AIDS, the largest boomer disadvantages were observed for mortality from hepatitis C and drug abuse,

which, respectively, hit their maximum relative risk levels of 4.9 and three at the turn of the century. Compared to the other causes, the relative risks of alcohol, COPD, and suicide mortality among boomers were substantially lower, never exceeding 1.7, 1.7, and 1.5, respectively.

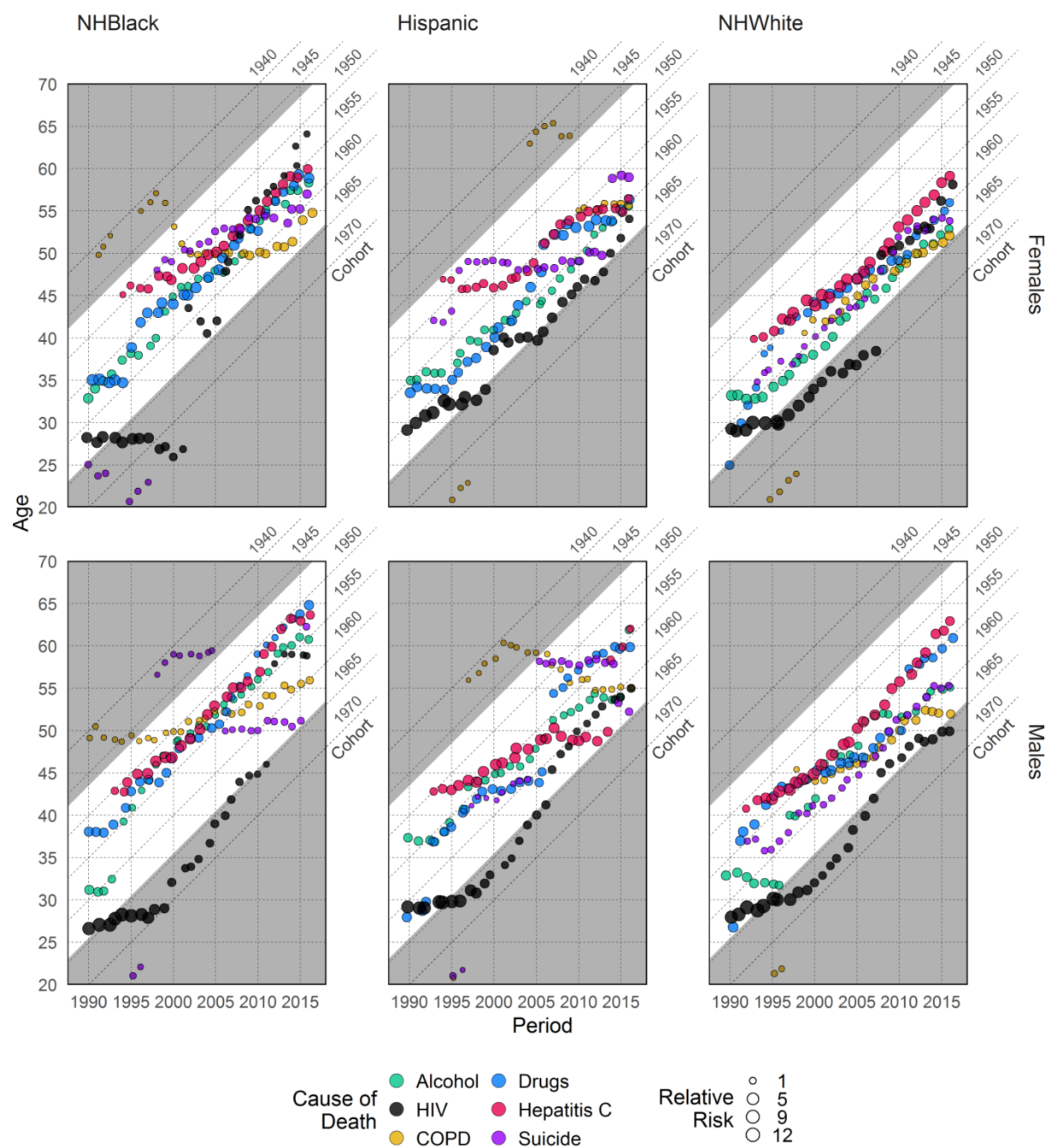**Figure 6. APC curvature plots by country, sex, and cause of death**
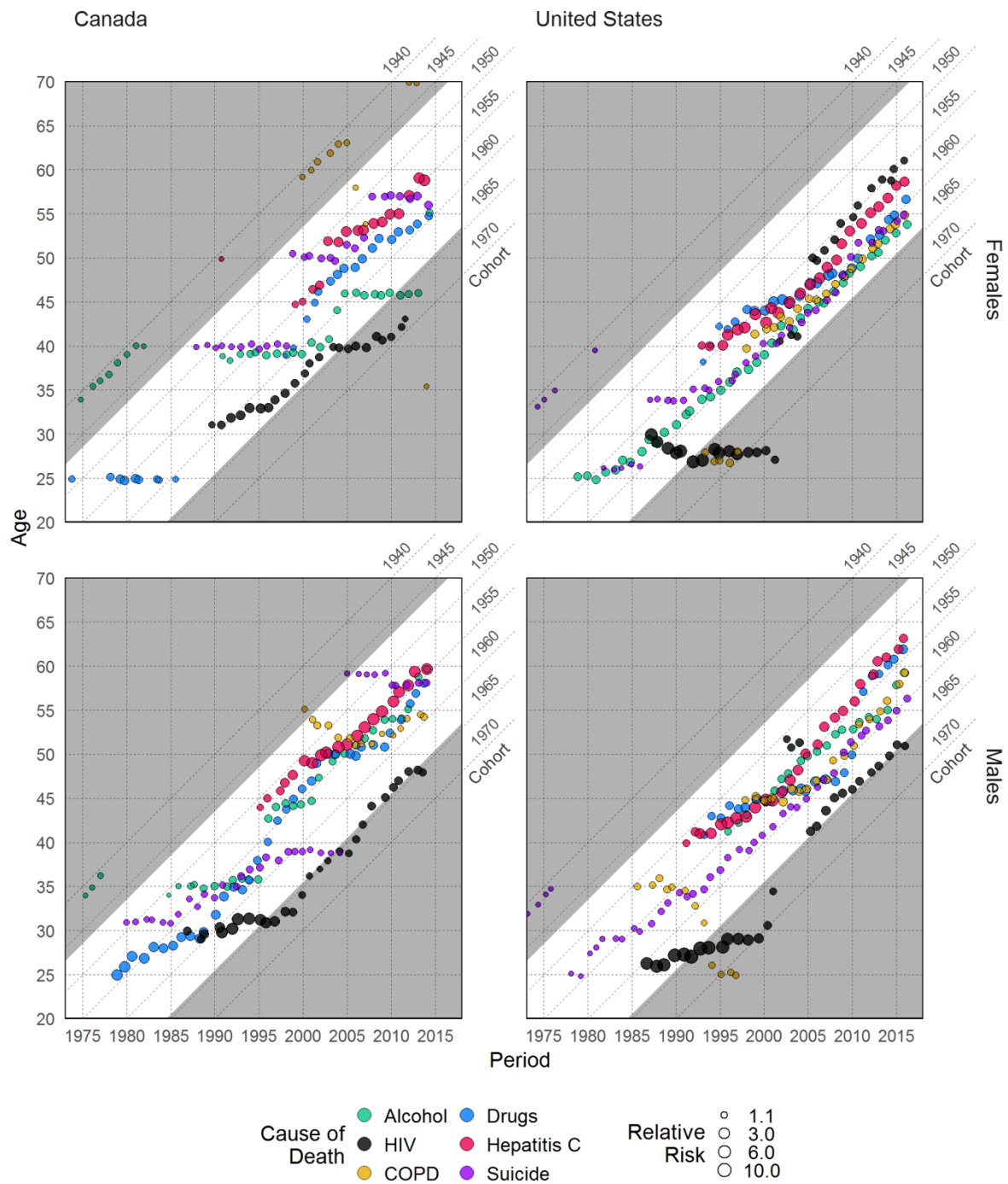
Notes: Location (age/cohort) and magnitude (in relative risks) of the largest excess mortality in each period. The color of the points indicates the cause of death and the size indicates the relative risk at the ridge, compared with the mortality baseline. The white diagonal band indicates the location of the baby boomer cohorts (i.e., 1946-1964).

Comparison across races and ethnicities within the United States

Figure 7 presents APC curvature plots of the cause-specific disadvantage in mortality by sex and race/ethnicity in the United States (see also Figure S5.9 in the supplementary materials). The trends displayed in Figure 7 show that boomers of both sexes and of all race/ethnicities had clear and sustained cohort disadvantages for all causes of death except COPD and suicide. Systematic cohort disadvantages in COPD and suicide were only identified for NHW. For the other ethnic/racial groups, the ridges for these causes were more indicative of an age effect. Consistent with the estimates at the national level (presented in Figure 6), the ridges of HIV/AIDS-related mortality for all racial/ethnic groups showed an age pattern of disadvantage until the end of the 1990s, with the largest relative risks among all the causes (12.2). At the turn of the century, the relative risk of HIV/AIDS mortality among boomers decreased considerably, and shifted into a sustained cohort effect. Moreover, consistent with the patterns at the national level, U.S. boomers of all race/ethnicities had the highest relative risks of hepatitis C and drug abuse mortality, reaching maximum levels of 4.7 and 3.8, respectively. The smallest relative disadvantages among boomers were for mortality from alcohol, COPD, and suicide (2.8, 2, and 1.9, respectively).

Turning now to the variations in relative risks over time, we can see that for most racial/ethnic groups, the relative risks of hepatitis C mortality peaked at the turn of the century, and decreased thereafter. However, for Hispanic and NHB females, the relative risks of hepatitis C mortality did not started to decrease until 2010. The ridge of the drug-related mortality disadvantage peaked three times: first at the turn of the 1990s, then at the turn of the 2000s, and, finally, during in the last year of observation (i.e., 2016). The relative risks of alcohol-related mortality were especially high in 1990 (2.1- to 2.8), and decreased progressively over time for Hispanics and NHW. However, for NHB female and male boomers, this declining trend was reversed in 2010, as the relative risks of alcohol mortality increased markedly. For COPD and suicide, the relative risks among boomers increased monotonically during the observation period, reaching an average of 1.5.

Figure 7. **APC curvature plots by race/ethnicity within the U.S., sex, and cause of death**

Notes: Location (age/cohort) and magnitude (in relative risk) of the largest excess mortality in each period, across causes of death, sexes, and races/ethnicities within the United States. The color of the points indicates the cause of death and the size indicates the relative risk at the excess ridge, compared with the mortality baseline. The white diagonal band indicates the location of the baby boomer cohorts (i.e., 1946-1964).

Taken together, these results suggest that what could be called "the boomer penalty" is the result of several cause-specific disadvantages that accompanied boomers throughout their life course, regardless of their country, sex, or race/ethnicity. However, the absence of a cohort disadvantage for several causes among Canadian female boomers should be noted.

4. Discussion

This study was designed to identify the leading causes of death that have contributed to the baby boomers experiencing a mortality disadvantage relative to their adjacent cohorts, and to explore the temporal dynamics of these contributions in Canada and the United States. Relative to previous cohorts, Canadian boomers experienced a deceleration in mortality improvements, while American boomers experienced an increase, in absolute terms, in all-cause mortality.

In summary, six behavioral causes of death – namely, drug and alcohol abuse, HIV/AIDS, hepatitis C, suicide, and COPD – contributed at least 75% of the positive changes in mortality – i.e., the deterioration in mortality – across cohorts. The disadvantages observed among the boomers were substantially larger among males than among females for most causes, regardless of national context or race/ethnicity. The highest relative risks of mortality were from hepatitis C and drug-related causes, and the lowest relative risks of mortality were from COPD and suicide. These results should, however, be interpreted with caution, given that higher relative risks do not necessarily indicate greater excess mortality in absolute terms. For instance, even though boomers had considerably higher relative risks of mortality from hepatitis C than from alcohol, because the baseline for mortality from alcohol was much higher, alcohol contributed more than hepatitis C to the mortality disadvantage among boomers.

On the question of the temporal dynamics of the boomers' excess mortality by cause, our findings revealed that most of these dynamics operated as long-term, sustained cohort disadvantages throughout the boomers' young and adult lives. The patterns over time of these cohort disadvantages in mortality were similar for boomer males in Canada and both sexes in the United States, regardless of race and ethnicity. The mortality disadvantages of Canadian female boomers were, however, much smaller and less indicative of sustained cohort effects.

In the rest of this section, we discuss the temporal dynamics of the cause-specific contributions to the boomer penalty, as well as the mechanisms that may have played a role in modulating such patterns. In particular, we focus on the factors that could have contributed to an increased boomer susceptibility to mortality from behavioral causes, and on the differences across population groups. We close this section by pointing out the advantages and limitations of our analysis, and recommend further investigations on the current topic for future research.

4.1. Temporal dynamics of the cause-specific excess mortality among boomers

The distinction between age-period interactions and cohort effects is relevant, since several of the causes that contributed the most to the cohort disadvantage in mortality among boomers were also responsible for important period crises during the observed period. Such was the case for the HIV/AIDS, the crack, and the opioid epidemics. It could be argued that the boomers' excess mortality resulted not from sustained cohort effects, but from successive period crises that targeted the boomers at different life stages – that is, a sequence of age-period interaction effects that disproportionately affected these cohorts.

The temporal patterns of mortality from HIV/AIDS are indicative of age-period interaction effects during the most critical stage of the epidemic (i.e., between the late 1980s and the early 1990s), which disproportionately affected those in their thirties. Although HIV/AIDS mortality decreased substantially after the introduction of the antiretroviral therapy in 1996 (Murphy et al. 2001; Palella et al. 2006; Vittinghoff et al. 1999), we found evidence that the effects of the epidemic lingered throughout the later adult lives of the late boomers (Figures 6, 7, S5, and S6). This higher susceptibility among boomers to HIV/AIDS may be the reason for the recent increases in HIV prevalence among people aged 50 and older, who account for nearly the half of those living with diagnosed HIV in the United States (CDC 2018a).

In the case of drug overdoses, our findings indicated that although the absolute changes in severity were largely driven by period-based factors that affected most ages, the boomer cohorts faced higher risks of drug-related mortality than the adjacent cohorts during the whole observation period. The magnitude of this cohort susceptibility was not, however, constant over time (Figures 6, 7, S8, and

S9). The disadvantage in drug-related mortality among boomers peaked twice: at the turn of the 1990s, which marked the beginning of the crack epidemic; and at the turn of the 2000s, which corresponded to the start of the opioid epidemic. These increases in relative risks at the beginning of each crisis and their posterior attenuation may be attributable to the vanguard role of boomers in abusing these drugs before the epidemics spread to other cohorts. This inter-cohort "contagion" of substance abuse is particularly notable for the case of the millennial cohorts. While the boomer cohorts had the largest relative risks of alcohol- and drug-related mortality during the 1990s and 2000s, the millennial cohorts have started experiencing relative increases in mortality from these causes, to the point that the millennials' risks of alcohol- and drug-related mortality have exceeded those of boomers in recent years (Huang et al. 2017; Miech et al. 2013; Sauer et al. 2018; Zang et al. 2019). For both HIV/AIDS and drug mortality, punctual and strong period crises affecting most age groups (i.e., period effects) have been detected. However, lagged effects from these disturbances along the cohorts have been observed among the boomers (i.e., cohort effects).

The similar locations of the most disadvantaged cohorts across causes of death (see Figures 6 and 7) suggest that the same mechanisms underlie multiple outcomes. Our results are consistent with previous findings indicating that behavioral risks related to opioid abuse, HIV/AIDS, and hepatitis C interacted with each other in these cohorts. In most groups, the cohorts with the largest disadvantages for these causes of death had similar locations and synchronic variations in magnitude since the mid-2000s. Some of these similarities may result from the ongoing opioid epidemic, which might have contributed to the spread of HIV/AIDS and hepatitis C infections among chronic intravenous drug users (IVDUs) (Strader 2005; Zibbell et al. 2017). In addition, while IVDUs who share contaminated needles face a higher risk of HIV infection, individuals who have been diagnosed with HIV have both higher rates of prescription opioid use to treat chronic pain symptoms and higher risks of developing drug use disorders (Becker et al. 2016). Similarly, our observation of the synchronicity between COPD and drug abuse mortality among NHB females and NHW of both sexes (Figure 7) was consistent with previous findings suggesting that the risk of COPD mortality is higher among opioid users (Levine 2017; Vozoris et al. 2016). However, the results for the other groups did not display this kind of synchronicity.

Taken together, our findings suggest that the mortality penalty endured by the boomers is not the result of a series of age-period interactions that coincidentally increased mortality at different life stages. Instead, the boomer disadvantage resulted from multiple and parallel long-term disadvantages

that have accompanied these cohorts over time. In short, the baby boomers have been suffering from concurrent long-term scars, not from a succession of temporary bruises. These disadvantages are apparent for several distinct, but interrelated causes of death that point to this generation having a unique set of attributes that stem from riskier behaviors and attitudes, which we discuss next.

4.2. Factors contributing to the boomer penalty

Although our research design is exploratory, our findings are consistent with numerous mechanisms already proposed in the literature. Below, we present and discuss in more detail the mechanisms – mentioned in the introduction – that may be driving the survival disadvantage among boomer cohorts. These mechanisms pertain to selection processes, birth cohort effects, and generational identity effects.

According to the *frailty hypothesis* (Vaupel et al. 1979; Zheng 2014), low selection pressure during infancy and childhood would have resulted in a heterogeneous cohort, with a large proportion of frail individuals surviving to adult ages and being susceptible to mortality from *intrinsic* causes of death. It has been argued that the higher mortality experienced by the boomers during their young and adult ages could be the consequence of increased survival rates early in life due to a reduced infection burden and improved nutrition intakes (Canudas-Romo and Guillot 2015). However, our results do not corroborate this hypothesis. The leading causes of death contributing to the boomer penalty are not intrinsic, but are, rather, behavioral or extrinsic. The likelihood that frail individuals who were "saved" in early life through better nutrition and reduced infection loads would go on to develop risky behaviors later in life seems rather low. Moreover, the *frailty hypothesis* does not explain the substantial mortality improvements among young adults in the cohorts born after the boomers, who experienced lower initial mortality rates than the boomers.

Based on our results, we propose that the higher susceptibility of the boomers is the result of two complementary mechanisms: namely, Ryder (1965)'s *birth cohort* influence and Mannheim (1952)'s historical *generational*[†] membership influence.

[†] Here, we use *generation* in a historical sense, so it should not be confused with the genealogical sense of kinship. See Alwin and McCammon (2007) for an extensive discussion about the three uses of the concept *generation* in the social sciences.

In his seminal work, Ryder (1965) stated that some the characteristics of some birth cohorts have permanent effects through the life course. According to Ryder, cohort size was the most evident manifestation of inter-cohort differences, since it is a persisting feature of the cohort's lifetime, with cascading effects on education, family formation, and labor force participation. Following Ryder's approach, Easterlin extensively studied the implications of cohort size and other cohort-specific characteristics for the historical locations of the boomers during their life course (Easterlin 1976, 1987; Easterlin et al. 1993). Easterlin's central argument was focused on the mismatch between the early life and the later life conditions that the boomers were known to have experienced. Born amid a post-war economic boom, the development of the welfare state, and unprecedented enrollment rates in higher education, the boomers became adults in a social context characterized by competition for resources – because of the large size of the cohort – a progressive erosion of the welfare state, and a weaker and increasingly precarious labor market. According to Easterlin, this imbalance between expectations and reality had serious implications for the perceived well-being of the boomers, increasing the prevalence of mental distress and frustration among these cohorts. Easterlin (1987) expected baby boomers to experience higher mortality from suicide, substance abuse, vehicle accidents, and homicide.

In contrast, *generational* membership, in the Mannheimian sense, implies a more complex social process in which individuals participate in the social movements of their time, and develop a shared identity with a unique worldview (Alwin and McCammon 2007; Eyerman and Turner 1998; Mannheim 1952). The enormous and unprecedented generational rift experienced by the boomers at sensitive life stages helped to consolidate their distinctive generational identity. The social importance and the inclusion of social movements in the popular culture and mass consumption – especially in rock music and literature – boosted and magnified the influence of these minorities on their contemporaries (Alwin et al. 2014; Alwin and McCammon 2007; Bristow 2015; Eyerman and Turner 1998; Stewart and Torges 2014). For the first time in history, a generational identity was simultaneously diffused to several western societies, including the United States, Canada, the United Kingdom, France, and Australia (Edmunds and Turner 2005).

The signatures of solidarity within this generation were the defiance of social norms and the rebellion against the older generations, which were expressed in drug use and a more explicit sexuality (Cross and Kleinhesselink 1985; Johnston 1991). An increasing number of studies have found that dispositions and attitudes toward riskier behaviors regarding drug use and sexuality tend

to be drawn from formative experiences and peer influences during the early stages of life, rather than from successive period-based influences throughout the life course (Johnson and Gerstein 2000; Keyes et al. 2011; Rhodes 1997). Thus, the differential constructions of risk perception and habituation during the early stages of the boomers' life course have been linked to higher risks of mortality from HIV/AIDS and other infections (McBride 1990), from substance abuse (Colliver et al. 2006; Crome and Rao 2018; Duncan et al. 2010; Miech et al. 2011; Patterson and Jeste 1999; Rao and Roche 2017), and from road traffic accidents (Puac-Polanco et al. 2016; T. Rao 2019).

Although they refer to different phenomena, *birth cohort* and *generational identity* are not completely independent. Their increased exposure to peers might have made the boomers less apt to identify with the values and beliefs of previous generations (Easterlin 1987; Phillips 2014; Stewart and Torges 2014), and encouraged the rise and spread of the youth-based social movements of the 1960s and 1970s (Abrams 1970; Bristow 2015, 2016; Cross and Kleinhesselink 1985; Goertzel 1972).

It is, therefore, possible that the cohort attributes of boomers not only reinforced their rebellious and risky generational identity, but that these two mechanisms may have acted in parallel to increase their mortality risk along the life course. In light of the mechanisms presented above, we now discuss some of the similarities and differences in the magnitude and the temporal dynamics of the boomer penalty across countries, sexes, and races/ethnicities.

4.3. Comparison of findings across the population groups under study

The unprecedented transnational diffusion of the boomer identity that we discussed above, as well as some similarities in the socioeconomic contexts in Canada and the United States during the sensitive life stages of the boomers, could explain our findings to some extent. We found that the causes that made the largest contributions to the excess mortality among the boomers were the same in both countries, and that the temporal patterns of the cohort effects were quite similar in the United States and Canada.

However, we also uncovered substantial differences in the magnitude of the boomer penalty between the two countries. As Figures 1 and 2 show, the Canadian boomers mainly experienced a slowdown in mortality improvements (indicated by the green diagonal trace in Figure 1), while the

U.S. boomers experienced a noticeable deterioration in mortality (indicated by the change of the scale sign from a negative to a positive change in mortality in Figure 1).

A possible explanation for this difference in the magnitude of the penalty may be related to the dissimilar experiences of the Canadian and the U.S. boomers during critical stages of their life course, which could have led to sizable differences in how the birth cohort and the generational identity mechanisms were embodied in these groups, and unfolded in their later lives. First, the levels of stress and frustration may have been substantially lower among Canadian boomers than among their counterparts in the United States. Although Canadian boomers are also part of a large cohort and grew up amid a post-war economic boom, the mismatch between the expectations they developed in childhood and the reality they encountered in young adulthood was not as extreme as it was in the United States. Compared to the United States, Canada has a stronger welfare system (Banting and Hoberg 1997; Myles 1998), much lower levels of inequality (Lemieux 1993; Ross et al. 2000; Rycroft 2013), and a less pronounced cultural orientation toward individualistic values (Adams 2004; Clark 1991; Lipset 2013; Steger et al. 1989).

Second, the generational rift experienced by Canadian boomers may have been less striking than that experienced by their counterparts in the United States. The dominant values in Canadian society – such as traditional views on the rights of women, racial/ethnic minorities, and non-heterosexual communities, as well as attitudes regarding religion, sexuality, and drug use – were also challenged by the counterculture movements (Palmer 2008). However, the scale of the political and social conflicts during the 1960s and 1970s was smaller in Canada than it was in the United States (Campbell et al. 2012). At that time, Canada had no Jim Crow laws and was not involved in the Vietnam War. Indeed, Canada has historically been perceived and has served as a refuge for U.S. citizens fleeing racism and the draft. Such was the case for the NHB who were escaping from slavery through the Underground Railroad to Canada during the 19th century, and for the draft-dodgers – mostly young boomers – who were seeking to avoid military service in the Vietnam War during the 1960s. Previous research has shown that the civil rights and the antiwar movements were the most important sources of the generational rift. Thus, the most influential and cohesive events for the U.S. boomers occurred around the counterculture movements during the 1960s and 1970s (Alwin et al. 2014; Alwin and McCammon 2007; Bristow 2015; Stewart and Torges 2014). Meanwhile, the Canadian boomers may have experienced milder birth cohort effects that translated into lower levels of anxiety, which led them to adopt a less rebellious generational identity associated with less risky

attitudes and behaviors. These social and political differences between the two countries might have contributed to the boomer penalty being smaller among the Canadians.

With respect to differences by sex, there is a large amount of evidence pointing to a higher propensity among males than among females to engage in risk-taking behaviors that could lead to death (Ferrence 1988; Fingerhut and Cox 1998; Harris and Jenkins 2006; Pampel 2001; Veevers and Gee 1986; Waldron et al. 2005). Since most of the excess mortality among the boomers stemmed from behavioral causes, it is not surprising that the boomer penalty was larger among males in all groups under observation.

Regarding the temporal patterns, two male-female differences stand out. First, the ridges of excess mortality by cause of death follow horizontal, rather than diagonal lines for Canadian females. The differences in the welfare state policies of Canada and the United States discussed above could explain this smaller boomer penalty among Canadian females. There is evidence that welfare state and social policies buffer social gender inequalities to some extent (Karamessini and Rubery 2013; Kushi and McManus 2018; Rubery 2012). It is possible that because they benefited from public policies that supported single and married mothers, and that incentivized women to enter and remain in the labor market, Canadian female boomers experienced less work-family conflict than their U.S. counterparts. Nevertheless, there is still evidence of a boomer penalty in mortality among Canadian female boomers (see Figures 1, 2, and 4) involving the same causes of death as those that affected mortality among the other boomer groups (see Figure 3). Hence, these results need to be interpreted with caution. The analytical strategy adopted here may not able to capture the actual temporal pattern of the boomer penalty among Canadian females because of its small magnitude.

A second male-female difference was in the location of the most disadvantaged cohorts. As Figures 1, 6, and 7 indicate, the cohorts with the largest excess mortality among females were the more recent boomer cohorts. No similar pattern was observed among male boomers. This difference in location could reflect the age differences within couples, which was 2.4 years on average for married couples between 1960 and 1985 (USCB 2018). Since males are more prone to risky behaviors and may have more success in influencing couple choices (de Palma et al. 2011), the negative outcomes in boomer couples may have disproportionately affected females of the more recent cohorts.

Interestingly, when we looked at the differences across races and ethnicities within the United States, we found that despite the significant differences in the socio-historical contexts experienced by the

different racial and ethnic groups, the patterns of sustained disadvantages among boomers were similar across these groups. This finding is somewhat surprising given the birth cohort effect proposed by Easterlin. When describing the large mismatch between the high expectations in childhood and the harsher reality encountered in adulthood, Easterlin was referring to the life courses of NHW boomers in the United States. These experiences were in stark contrast with those lived by minorities. NHW were the main beneficiaries of the economic boom and social policies implemented during the post-war period, which led boomer children to develop outsized expectations. The unprecedented numbers of university admissions and the large numbers of mortgage loans offered during the post-war period were selectively addressed to the young NHW population – the parents of the NHW boomers. For racial/ethnic minorities, however, residential segregation (Luders-Manuel 2017; Massey and Denton 1993; Rothstein 2017; Sharp and Hall 2014; Steil et al. 2018) and educational segregation (Herbold 1994; Humes 2006; Turner and Bound 2002), among other discriminatory policies, hindered their access to the social and economic benefits of that period.

By contrast, the generational rift the boomer cohorts experienced and embraced, albeit in very different ways, had a substantial impact on most ethnic-racial groups within the United States. Young boomers from different social, racial, and ethnic backgrounds became involved in the diverse social movements of the time, such as the student, anti-war, feminist, gay liberation, civil rights, Black Power, Red Power, and Chicano movements (Reed 2019; Rollins 1986; Stewart et al. 1998). Hence, it may be expected that the generational identities the boomers developed influenced wider segments of the U.S. population, regardless of their socioeconomic status and race/ethnicity. Thus, the birth cohort effects proposed by Easterlin may have been less relevant to the boomer generation.

These differences across race/ethnicities in the birth cohort effects and the generational identity mechanisms could help to explain the disparities in the temporal dynamics of suicides ridges. NHW boomers of both sexes were the only groups within the United States that showed sustained cohort disadvantages in suicide mortality. Whereas suicide mortality may have been more associated with the higher prevalence of stress and frustration resulting from the birth cohort effects proposed by Easterlin (Chauvel et al. 2016; Easterlin 1987; Phillips 2014), the higher susceptibility to mortality from substance abuse, HIV/AIDS, and hepatitis C may have been more responsive to the risky

attitudes and behaviors embodied in the boomer generational identities (M. W. Boeri et al. 2006; Jalal et al. 2018; Johnston 1991; Keyes et al. 2011; Miech et al. 2011).

Regarding the drug epidemics, the crack and the opioid crises have been respectively associated with NHB and NHW populations in the scientific literature and media coverage. It is possible that the stigma and the lower price of crack made the poor black neighborhoods in the inner city more vulnerable to the crack epidemic at the turn of the 1990s (Agar 2003; Johnston 1991; Palamar et al. 2015; Palamar and Ompad 2014). Likewise, the medical-legal origin of the opioid crisis, as well as racial discrimination in prescription practices (Barnett et al. 2017; Hwang et al. 2015; Jones et al. 2018; King et al. 2014; Manchikanti et al. 2017; Quinones 2015; Zang et al. 2019), appear to have put the NHW blue-collar population at greater risk during the early stages of the opioid crisis at the turn of the 21st century. However, there is evidence that these drug epidemics have ravaged all social groups, even though they disproportionately affected specific social status or racial/ethnic groups in certain periods (Agar 2003; Ho 2017; Jalal et al. 2018; Woolf et al. 2018).

We have attempted here to provide a summary of the mechanisms that could contribute to the mortality penalty among baby boomers in Canada and the United States, as well as to point out some similarities and disparities in these mechanisms by country, sex, and race/ethnicity. To do so, we adopted a more comprehensive approach to analyzing the excess mortality among boomers as a whole than was used by most previous research on the topic. Instead of exploring a single cause of death, as most of the previous studies on mortality among boomers did, our strategy began by exploring the penalty in all-cause mortality common to boomers in Canada and the United States, and to subsequently decompose this disadvantage by cause of death. The decomposition of the mortality changes using the Cohort Partial Mortality Rate measure ($CPMR^{c(k,l)}$) – proposed for the first time in this research – allowed us to identify a common set of causes underlying the excess mortality among boomers in Canada and the United States.

The analysis of the temporal dynamics of nonlinear cohort effects using *APC curvature plots* permitted us to show that the disadvantage in mortality among boomers resulted from multiple and simultaneous cause-specific cohort effects, instead of from a sequence of age-period interaction effects. As we noted above, we would have been unable to generate such findings if we had limited our analytical strategy to the more conventional APC statistical models. Using this approach, we were able to show that multiple disadvantages were experienced concurrently by the boomer cohorts, and that these disadvantages may be interrelated and modulated by common mechanisms,

independent of national context, sex, and race/ethnicity. These findings offer additional clues that can be used to analyze causal mechanisms that were not exclusively related to one output, but that instead generated simultaneous and sustained disadvantages throughout the life course of the boomers.

To our knowledge, this is the first analysis that has reported concurrent and sustained cohort effects for Canadian and U.S. boomers. Such findings provide valuable information for designing health policies. Currently, the populations of these countries are dealing with health crises involving the causes that made the largest contributions the boomers' excess mortality. Specifically, these populations are experiencing dramatic increases in mortality from drug overdoses (Helmerhorst et al. 2017; Ho 2017; Huang et al. 2017; Jalal et al. 2018), alcohol abuse (CIHI 2018; Tapper and Parikh 2018), and suicide (Curtin et al. 2016); increases in binge drinking (Bulloch et al. 2016; Dwyer-Lindgren et al. 2015; Manthey et al. 2019) and in the incidence of hepatitis C infections (CDC 2018b; PHAC 2019; Zibbell et al. 2017); as well as the stalling of the long-term decrease in deaths from of HIV/AIDS (CDC 2019). Our research suggests that policy-makers should encourage prevention and diagnosis based not only on an individual's risk factors and age, as is usually done, but on the person's birth cohort. Canadian and U.S. health authorities have already proposed adopting such an approach for dealing with hepatitis C, and have recommended systematic testing for baby boomers (CATIE 2018; CDC - Division of Viral Hepatitis 2019; CDC 2012; Shah et al. 2018). Our findings also suggest that such an approach should be extended to other behavioral causes. In the rest of this section, we consider the limitations of this study, and offer some recommendations for further research.

4.4. Limits of the analytical strategy and suggestions for future work

We are aware that our research has several limitations. The first is related to the information recorded on the death certificates. On the one hand, deaths from some of the causes under analysis may be misreported on the death certificates. HIV/AIDS and hepatitis C mortality began to be recognized within the ICD codes in 1987, and thus long after actual deaths from these causes started to occur. It may have taken some time for medical authorities to accurately recognize and record such causes on death certificates. Hence, underreporting is expected for these two causes. In addition, the attribution of just one cause to each death can hide multiple interactions, which can be

especially problematic when the causes are related to behavioral factors. Because risk behaviors tend to cluster (Ho 2017), several deaths classified in one specific cause may have multiple contributing factors. For example, a death may be caused by a combination of drug and alcohol abuse, or of HIV and hepatitis C infections. For suicide mortality, suicidal intent is difficult to assess, and the approaches used in assigning such an intent vary by geographical location. Moreover, some causes cannot be discerned from the classification when the intentions and the means are mutually exclusive, as is the case for suicides by drug overdose. In Table S2 in the supplemental materials, we classified such deaths as suicides, which could mean that drug-related death rates have been underestimated, while the number of suicides has been overestimated. This bias in drug-related mortality could also be magnified by other underreporting issues for this cause of death on death certificates (Ho 2017; Paulozzi et al. 2006).

Another limitation of the identification of causes of death is the discontinuity resulting from the ICD revisions implemented during the observation period (see Tables S1 and S2). Although we did not find evidence of significant disruptions in the classification at an aggregated level, several specific codes that did not exist in previous ICD revisions were introduced during the period, including codes for several alcohol- and drug-related causes of death. For instance, death from drug abuse was introduced in the 9th ICD version, and poisoning from both drugs and alcohol with undetermined intent was introduced in the 10th revision.

An additional potential source of error in the data is related to the racial/ethnic classification within the United States. Discrepancies between the numerator and the denominator in the Hispanic ethnic classification have been detected. Whereas on the death certificates (i.e., the numerator) the information on ethnicity is provided by funeral directors – who may be asked to use a certain classification by relatives of the deceased, or who simply impute the deceased's ethnicity using their own judgment – in the census (i.e., the denominator) this information is self-reported (Arias et al. 2008; Zang et al. 2019). This divergence in data collection procedures may have resulted in an underestimation of mortality in the Hispanic population. In addition, the lack of racial and ethnic information in the Canadian death register limited our analyses of the heterogeneity of boomers within Canada, and made it impossible to compare similar groups in the two countries.

With regard to the methods used to analyze the temporal pattern of the disadvantage in mortality, there is more than one approach to detecting excess mortality (Acosta and van Raalte 2019). Because

each approach is conceptually different, their application can lead to different estimations. Here, we used an interpolation approach because our aim was to analyze the excess mortality among the boomers relative to the mortality levels of the more advantaged cohorts surrounding the boomers. If the question is about the deviation from the overall cohort average, the estimation of the residual from an age-period model would be more appropriate. It is noteworthy that the main difference between the two approaches lies in the magnitude of the excess they report, but that their findings regarding the temporal pattern over time are consistent.

On the question of the explanatory power of this work, neither the analytical strategy nor the data for the analysis presented here allowed us to test or disentangle the role played by the birth cohort and the generational identity effects on the boomer penalty in mortality. Here, we proposed such mechanisms as underlying determinants of the boomer disadvantage in mortality in a speculative manner. Longitudinal data that include measures of drug use, alcohol consumption, sexual behavior, mental health, mental stress, and/or expectations, among other factors, would allow for an inquiry that takes an explanatory approach.

We believe that our research will serve as a basis for future studies on the excess mortality among boomers. We propose that further research should be undertaken in the following areas. First, future studies should test the causal mechanisms identified here as being potentially responsible for the excess mortality among boomers, and measure to what extent each of these mechanisms has contributed to the boomer penalty. Second, an important question that future studies should address is why the pattern of the cohort disadvantage among Canadian female boomers, for which no sustained cohort effects were identified, differed from that among Canadian male boomers and U.S. boomers for all races/ethnicities under analysis. Third, more analysis of the impact of the boomer disadvantage on changes in life expectancy and lifespan inequality, and on years of life lost, would help to clarify the implications of this excess mortality among the boomers at the population level for future mortality trends. Fourth, it is not yet known whether strong generational differences are a common feature of all socioeconomic groups, or whether they are disproportionately concentrated among the socially disadvantaged. It is vital to assess the contributions of different dimensions of social position to the inter- and intra-cohort inequalities in mortality related to substance abuse and infections. Finally, the similarities in the mortality penalty among boomers found in Canada and the United States suggest that similar mechanisms could be involved in other national contexts with similar mortality patterns, such as in France, Australia, and England (Acosta et al. 2017; HMD 2019).

5. Conclusions

We found evidence that most of the excess mortality among baby boomers in Canada and the United States was driven by behavioral causes of death: namely, drug abuse, alcohol abuse, HIV/AIDS, hepatitis C, COPD, and suicide. The main exception to this general finding was that among Canadian female boomers, these causes of death did not translate into sustained cohort effects over time. We also found that the contributions of these behavioral causes of death to the excess mortality among boomers in Canada and the United States were the consequence of multiple and simultaneous long-term sustained disadvantages that have followed the members of this cohort since their twenties.

The behavioral nature of the excess mortality among the boomers, and the sustained cause-specific effects of this excess mortality throughout their young and adult lives, highlight the pertinence of a more comprehensive and structural analysis of the boomer mortality disadvantage. The observation that the causes that made the largest contributions to the excess mortality among boomers were linked to behavioral risks suggests that a common set of mechanisms underlie the boomer penalty in Canada and the United States. We propose that the relatively high levels of distress and frustration among boomers – the birth cohort effect proposed by Easterlin – and the riskier attitudes toward drug use and sexual behavior that are constituent of the boomer generation identity have together played a substantial role in their mortality disadvantage. Further analyses are needed to test how these mechanisms have affected the mortality penalty among boomers, and how the impact of these mechanisms has differed across socioeconomic groups.

If the cohort differences in mortality continue along the same trend, it is possible that people who are currently aged 65 or older will experience substantial increases in mortality in the upcoming years. Such increases might be even greater than those previously experienced by the boomer cohorts, because the mortality risks related to suicide and mental health disorders are considerably higher at older than at younger ages. Moreover, as the baby boomers age, they will become increasingly likely to experience chronic pain, which has been a fundamental factor in the ongoing opioid abuse epidemic (Jones et al. 2018).

6. Contributions

E.A. conceived the research design, developed all the program codes in R, and prepared the electronic materials and guidelines for reproducibility. E.A. and R.B. conceived the analysis strategy. E.A., A.G., N.O., and A.v.R., did the analyses of results. E.A., A.G., and N.O. wrote the manuscript. R.B., M.N., and A.v.R. Revised and validated the manuscript.

7. Acknowledgements

8. References

Abrams, P. (1970). Rites de Passage: The Conflict of Generations in Industrial Society. *Journal of Contemporary History*, 5(Generations in Conflict), 175–190.

Acosta, E. (2020). Reproducible Materials for 'The Boomers' Excess Mortality in Canada and the United States'. Open Science Framework (OSF). [electronic resource]. https://osf.io/c3efa/. Accessed 2 October 2019

Acosta, E., Gagnon, Alain, Ouellette, Nadine, & Bourbeau, Robert. (2017). Baby Boomers' Excess Mortality: An International Comparative Analysis. In IPC 2017. Presented at the IUSSP - 2017 International Population Conference, Cape Town. https://iussp.confex.com/iussp/ipc2017/meetingapp.cgi/Paper/7386. Accessed 26 June 2018

Acosta, E., & van Raalte, A. (2019). APC Curvature Plots: Displaying Nonlinear Age-Period-Cohort Patterns on Lexis Plots. *Demographic Research*, 41(42), 1205-1234. https://doi.org/10.4054/DemRes.2019.41.42

Adams, M. (2004). *Fire and Ice: The United States, Canada and the Myth of Converging Values* (First edition.). Toronto: Penguin Canada.

Agar, M. (2003). The Story of Crack: Towards a Theory of Illicit Drug Trends. *Addiction Research & Theory*, 11(1), 3–29. https://doi.org/10.1080/1606635021000059042

Alwin, D. F., & McCammon, R. J. (2007). Rethinking Generations. *Research in Human Development*, 4(3–4), 219–237. https://doi.org/10.1080/15427600701663072

Alwin, D. F., McCammon, R. J., & Hofer, S. M. (2014). Studying Baby Boom Cohorts Within a Demographic and Developmental Context: Conceptual and Methodological Issues. In *The Baby Boomers Grow Up: Contemporary Perspectives on Midlife* (pp. 45–71). New York: Psychology Press.

Arias, E., Schauman, W. S., Eschbach, K., Sorlie, P. D., & Backlund, E. (2008). The validity of race and Hispanic origin reporting on death certificates in the United States. Vital and Health Statistics. Series 2, Data Evaluation and Methods Research, (148), 1–23.

Banting, K. G., & Hoberg, G. (1997). *Degrees of Freedom: Canada and the United States in a Changing World* (First edition.). Montreal: McGill-Queen's University Press.

Barbi, E., & Camarda, C. G. (2011). Period and cohort effects on elderly mortality: a new relational model for smoothing mortality surfaces. *Statistica*, 71(1), 51–69. https://doi.org/10.6092/issn.1973-2201/3604

Barnett, M. L., Olenski, A. R., & Jena, A. B. (2017). Opioid-Prescribing Patterns of Emergency Physicians and Risk of Long-Term Use. *New England Journal of Medicine*, 376(7), 663–673. https://doi.org/10.1056/NEJMsa1610524

Becker, W. C., Gordon, K., Jennifer Edelman, E., Kerns, R. D., Crystal, S., Dziura, J. D., et al. (2016). Trends in Any and High-Dose Opioid Analgesic Receipt Among Aging Patients With and Without HIV. *AIDS and Behavior*, 20(3), 679–686. https://doi.org/10.1007/s10461-015-1197-5

Bell, A., & Jones, K. (2013). The impossibility of separating age, period and cohort effects. *Social Science & Medicine*, 93(Supplement C), 163–165. https://doi.org/10.1016/j.socscimed.2013.04.029

Boeri, M. W., Sterk, C. E., & Elifson, K. W. (2006). Baby Boomer Drug Users: Career Phases, Social Control, and Social Learning Theory*. *Sociological Inquiry*, 76(2), 264–291. https://doi.org/10.1111/j.1475-682X.2006.00154.x

Bourbeau, R., & Ouellette, N. (2016). Trends, patterns, and differentials in Canadian mortality over nearly a century, 1921-2011. *Canadian Studies in Population*, 43(1–2), 48–77.

Bristow, J. (2015). *Baby Boomers and Generational Conflict*. Palgrave Macmillan UK.

Bristow, J. (2016). *The Sociology of Generations: New Directions and Challenges*. Springer.

Bulloch, A. G. M., Williams, J. V. A., Lavorato, D. H., & Patten, S. B. (2016). Trends in binge drinking in Canada from 1996 to 2013: a repeated cross-sectional analysis. *CMAJ Open*, 4(4), E599–E604. https://doi.org/10.9778/cmajo.20150124

Burnham, K. P., & Anderson, D. R. (2002). *Model Selection and Multimodel Inference: A Practical Information-Theoretic Approach* (2nd ed.). New York: Springer-Verlag. https://www.springer.com/de/book/9780387953649. Accessed 8 February 2019

Camarda, C. G. (2012). MortalitySmooth: An R Package for Smoothing Poisson Counts with P-Splines. *Journal of Statistical Software*. https://doi.org/10.18637/jss.v050.i01

Campbell, L. A., Clement, D., & Kealey, G. S. (2012). *Debating Dissent: Canada and the 1960s* (1 edition.). Toronto: University of Toronto Press, Scholarly Publishing Division.

Canudas-Romo, V., & Guillot, M. (2015). Truncated cross-sectional average length of life: A measure for comparing the mortality history of cohorts. *Population Studies*, 69(2), 147–159. https://doi.org/10.1080/00324728.2015.1019955

Carstensen, B. (2007). Age-period-cohort models for the Lexis diagram. *Statistics in Medicine*, 26(15), 3018–3045. https://doi.org/10.1002/sim.2764

Carstensen, B., Plummer, M., Laara, E., & Hills, M. (2019). Epi: A Package for Statistical Analysis in Epidemiology. R. https://cran.r-project.org/web/packages/Epi/index.html

CATIE. (2018). Latest Canadian hepatitis C guidelines encourage offer of a test to baby boomers (CATIE News). Canada: CATIE - Canada's source for HIV and hepatitis C information. https://www.catie.ca/en/catienews/2018-06-12/latest-canadian-hepatitis-c-guidelines-encourage-offer-test-baby-boomers. Accessed 20 August 2019

CDC. (2012). Recommendations for the Identification of Chronic Hepatitis C Virus Infection Among Persons Born During 1945–1965 (Recommendations and Reports No. 61). CDC. https://www.cdc.gov/mmwr/preview/mmwrhtml/rr6104a1.htm. Accessed 20 August 2019

CDC. (2018a). HIV Surveillance Report, 2017 (The HIV Surveillance Report No. 29) (p. 125). CDC. https://www.cdc.gov/hiv/pdf/library/reports/surveillance/cdc-hiv-surveillance-report-2017-vol-29.pdf

CDC. (2018b). Surveillance for Viral Hepatitis – United States, 2016. CDC, Division of Viral Hepatitis. https://www.cdc.gov/hepatitis/statistics/2016surveillance/commentary.htm. Accessed 20 August 2019

CDC. (2019). Estimated HIV incidence and prevalence in the United States, 2010–2016 (HIV Surveillance Supplemental Report No. 24 (1)). https://www.cdc.gov/hiv/pdf/library/reports/surveillance/cdc-hiv-surveillance-supplemental-report-vol-24-1.pdf. Accessed 19 August 2019

CDC - Division of Viral Hepatitis. (2019). CDC Recommendation: Adults Born from 1945-1965 (Baby Boomers) get Tested for Hepatitis C. https://www.cdc.gov/hepatitis/populations/1945-1965.htm. Accessed 20 August 2019

Chauvel, L. (2013). Spécificité et permanence des effets de cohorte : le modèle APCD appliqué aux inégalités de générations, France/États-Unis, 1985-2010. *Revue française de sociologie*, 54(4), 665–705. https://doi.org/10.3917/rfs.544.0665

Chauvel, L., Leist, A. K., & Ponomarenko, V. (2016). Testing Persistence of Cohort Effects in the Epidemiology of Suicide: an Age-Period-Cohort Hysteresis Model. *PLOS ONE*, 11(7), e0158538. https://doi.org/10.1371/journal.pone.0158538

CIHI. (2018). Alcohol Harm in Canada. Examining Hospitalizations Entirely Caused by Alcohol and Strategies to Reduce Alcohol Harm. Canadian Institute for Health Information.

Clark, P. G. (1991). Ethical Dimensions of Quality of Life in Aging: Autonomy vs. Collectivism in the United States and Canada. *The Gerontologist*, 31(5), 631–639. https://doi.org/10.1093/geront/31.5.631

Clayton, D., & Schifflers, E. (1987). Models for temporal variation in cancer rates. II: Age–period–cohort models. *Statistics in Medicine*, 6(4), 469–481. https://doi.org/10.1002/sim.4780060406

Colliver, J. D., Compton, W. M., Gfroerer, J. C., & Condon, T. (2006). Projecting drug use among aging baby boomers in 2020. *Annals of Epidemiology*, 16(4), 257–265. https://doi.org/10.1016/j.annepidem.2005.08.003

Crome, I., & Rao, R. (2018). Our Invisible Addicts (No. CR211). *Royal College of Psychiatrists*. https://www.rcpsych.ac.uk/usefulresources/publications/collegereports/cr/cr211.aspx. Accessed 11 September 2018

Cross, H. J., & Kleinhesselink, R. R. (1985). The Impact of the 1960s on Adolescence. *The Journal of Early Adolescence*, 5(4), 517–531. https://doi.org/10.1177/0272431685054009

Curtin, S. C., Warner, M., & Hedegaard, H. (2016). Increase in Suicide in the United States, 1999-2014. NCHS data brief, (241), 1–8.

de Palma, A., Picard, N., & Ziegelmeyer, A. (2011). Individual and couple decision behavior under risk: evidence on the dynamics of power balance. *Theory and Decision*, 70(1), 45–64. https://doi.org/10.1007/s11238-009-9179-6

Duncan, D. F., Nicholson, T., White, J. B., Bradley, D. B., & Bonaguro, J. (2010). The baby boomer effect: changing patterns of substance abuse among adults ages 55 and older. *Journal of Aging & Social Policy*, 22(3), 237–248. https://doi.org/10.1080/08959420.2010.485511

Dwyer-Lindgren, L., Flaxman, A. D., Ng, M., Hansen, G. M., Murray, C. J. L., & Mokdad, A. H. (2015). Drinking Patterns in US Counties From 2002 to 2012. *American Journal of Public Health*, 105(6), 1120–1127. https://doi.org/10.2105/AJPH.2014.302313

Easterlin, R. A. (1976). The Conflict between Aspirations and Resources. *Population and Development Review*, 2(3/4), 417–425. https://doi.org/10.2307/1971619

Easterlin, R. A. (1987). *Birth and Fortune: The Impact of Numbers on Personal Welfare* (2 edition.). Chicago: University Of Chicago Press.

Easterlin, R. A., Schaeffer, C. M., & Macunovich, D. J. (1993). Will the Baby Boomers be Less Well off Than Their Parents? Income, Wealth, and Family Circumstances over the Life Cycle in the United States. *Population and Development Review*, 19(3), 497–522. https://doi.org/10.2307/2938464

Edmunds, J., & Turner, B. S. (2005). Global generations: social change in the twentieth century. *The British Journal of Sociology*, 56(4), 559–577. https://doi.org/10.1111/j.1468-4446.2005.00083.x

Eilers, P. H. C., & Marx, B. D. (1996). Flexible Smoothing with B-splines and Penalties. *Statistical Science*, 11(2), 89–102.

Eilers, P. H. C., Marx, B. D., & Durbán, M. (2015). Twenty years of P-splines. *SORT-Statistics and Operations Research Transaction*s, 39(2), 149-186–186.

Ellwood, D. T. (1982). *Teenage Unemployment: Permanent Scars or Temporary Blemishes? In The Youth Labor Market Problem: Its Nature, Causes, and Consequences*. University of Chicago Press. https://www.nber.org/books/free82-1. Accessed 29 March 2019

Eyerman, R., & Turner, B. S. (1998). Outline of a Theory of Generations. *European Journal of Social Theory*, 1(1), 91–106. https://doi.org/10.1177/136843198001001007

Ferrence, R. G. (1988). Sex differences in cigarette smoking in Canada, 1900-1978: a reconstructed cohort study. *Canadian Journal of Public Health = Revue Canadienne De Sante Publique*, 79(3), 160–165.

Fienberg, S. E. (2013). Cohort Analysis' Unholy Quest: A Discussion. *Demography*, 50(6), 1981–1984. https://doi.org/10.1007/s13524-013-0251-z

Fienberg, S. E., & Mason, W. M. (1985). Specification and Implementation of Age, Period and Cohort Models. In *Cohort Analysis in Social Research* (pp. 45–88). Springer, New York, NY. https://doi.org/10.1007/978-1-4613-8536-3_3

Finch, C. E., & Crimmins, E. M. (2004). Inflammatory exposure and historical changes in human life-spans. *Science*, 305(5691), 1736–1739. https://doi.org/10.1126/science.1092556

Fingerhut, L. A., & Cox, C. S. (1998). Poisoning mortality, 1985-1995. *Public Health Reports*, 113(3), 218–233.

Floud, R., Fogel, R. W., Harris, B., & Hong, S. C. (2011). *The Changing Body: Health, Nutrition, and Human Development in the Western World since 1700*. Cambridge ; New York: Cambridge University Press.

Fogel, R. W., & Costa, D. L. (1997). A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs. *Demography*, 34(1), 49–66.

Fosse, E., & Winship, C. (2018). Moore–Penrose Estimators of Age–Period–Cohort Effects: Their Interrelationship and Properties. *Sociological Science*, 5(14), 304--334. https://doi.org/10.15195/v5.a14

Fosse, E., & Winship, C. (2019). Analyzing Age-Period-Cohort Data: A Review and Critique. *Annual Review of Sociology*, 45(1), 467–492. https://doi.org/10.1146/annurev-soc-073018-022616

Goertzel, T. (1972). Generational Conflict and Social Change. *Youth and Society*, 3(3), 327–352.

Harris, C. R., & Jenkins, M. (2006). Gender Differences in Risk Assessment: Why do Women Take Fewer Risks than Men? *Judgment and Decision Making*, 1(1), 16.

Helmerhorst, G. T. T., Teunis, T., Janssen, S. J., & Ring, D. (2017). An epidemic of the use, misuse and overdose of opioids and deaths due to overdose, in the United States and Canada. *The Bone & Joint Journal*, 99-B(7), 856–864. https://doi.org/10.1302/0301-620X.99B7.BJJ-2016-1350.R1

Herbold, H. (1994). Never a Level Playing Field: Blacks and the GI Bill. *The Journal of Blacks in Higher Education*, (6), 104–108. https://doi.org/10.2307/2962479

HMD. (2019). Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). http://www.mortality.org/. Accessed 15 March 2019

Ho, J. Y. (2017). The Contribution of Drug Overdose to Educational Gradients in Life Expectancy in the United States, 1992–2011. *Demography*, 54(3), 1175–1202. https://doi.org/10.1007/s13524-017-0565-3

Holford, T. R. (1983). The Estimation of Age, Period and Cohort Effects for Vital Rates. *Biometrics*, 39(2), 311–324. https://doi.org/10.2307/2531004

Holford, T. R. (1991). Understanding the effects of age, period, and cohort on incidence and mortality rates. *Annual Review of Public Health*, 12, 425–457. https://doi.org/10.1146/annurev.pu.12.050191.002233

Huang, X., Keyes, K. M., & Li, G. (2017). Increasing Prescription Opioid and Heroin Overdose Mortality in the United States, 1999–2014: An Age–Period–Cohort Analysis. *American Journal of Public Health*, 108(1), 131–136. https://doi.org/10.2105/AJPH.2017.304142

Humes, E. (2006). How the GI Bill Shunted Blacks into Vocational Training. *The Journal of Blacks in Higher Education*, (53), 92–104.

Hwang, C. S., Chang, H.-Y., & Alexander, G. C. (2015). Impact of abuse-deterrent OxyContin on prescription opioid utilization. *Pharmacoepidemiology and Drug Safety*, 24(2), 197–204. https://doi.org/10.1002/pds.3723

Jalal, H., Buchanich, J. M., Roberts, M. S., Balmert, L. C., Zhang, K., & Burke, D. S. (2018). Changing dynamics of the drug overdose epidemic in the United States from 1979 through 2016. *Science*, 361(6408), eaau1184. https://doi.org/10.1126/science.aau1184

Johnson, R. A., & Gerstein, D. R. (2000). Age, period, and cohort effects in marijuana and alcohol incidence: United States females and males, 1961-1990. *Substance Use & Misuse*, 35(6–8), 925–948.

Johnston, L. (1991). Toward a theory of drug epidemics. In Communication. *Persuasive communication and drug abuse prevention* (pp. 93–131).

Jones, M. R., Viswanath, O., Peck, J., Kaye, A. D., Gill, J. S., & Simopoulos, T. T. (2018). A Brief History of the Opioid Epidemic and Strategies for Pain Medicine. Pain and Therapy, 7(1), 13–21. https://doi.org/10.1007/s40122-018-0097-6

Karamessini, M., & Rubery, J. (2013). *Women and Austerity: The Economic Crisis and the Future for Gender Equality*. Routledge.

Keyes, K. M., Schulenberg, J. E., O'Malley, P. M., Johnston, L. D., Bachman, J. G., Li, G., & Hasin, D. (2011). The social norms of birth cohorts and adolescent marijuana use in the United States, 1976-2007. *Addiction*, 106(10), 1790–1800. https://doi.org/10.1111/j.1360-0443.2011.03485.x

King, D. E., Matheson, E., Chirina, S., Shankar, A., & Broman-Fulks, J. (2013). The status of baby boomers' health in the United States: the healthiest generation? *JAMA internal medicine*, 173(5), 385–386. https://doi.org/10.1001/jamainternmed.2013.2006

King, N. B., Fraser, V., Boikos, C., Richardson, R., & Harper, S. (2014). Determinants of Increased Opioid-Related Mortality in the United States and Canada, 1990–2013: A Systematic Review. *American Journal of Public Health*, 104(8), e32–e42. https://doi.org/10.2105/AJPH.2014.301966

Kushi, S., & McManus, I. P. (2018). Gender, crisis and the welfare state: Female labor market outcomes across OECD countries. *Comparative European Politics*, 16(3), 434–463. https://doi.org/10.1057/cep.2016.21

Lemieux, T. (1993). Unions and Wage Inequality in Canada and the United States. Small Differences That Matter: Labor Markets and Income Maintenance in Canada and the United States, 69–108.

Leveille, S. G., Wee, C. C., & Iezzoni, L. I. (2005). Trends in obesity and arthritis among baby boomers and their predecessors, 1971-2002. *American Journal of Public Health*, 95(9), 1607–1613. https://doi.org/10.2105/AJPH.2004.060418

Levine, M. (2017). In older adults with COPD, new opioid use was linked to increased risk for respiratory and all-cause mortality. *Annals of Internal Medicine*, 166(2), JC11. https://doi.org/10.7326/ACPJC-2017-166-2-011

Lipset, S. M. (2013). *Continental Divide : The Values and Institutions of the United States and Canada*. Routledge. https://doi.org/10.4324/9781315021201

Luders-Manuel, S. (2017, September 18). The Inequality Hidden Within the Race-Neutral G.I. Bill. *JSTOR Daily*. https://daily.jstor.org/the-inequality-hidden-within-the-race-neutral-g-i-bill/. Accessed 3 September 2019

Luo, L. (2013). Assessing Validity and Application Scope of the Intrinsic Estimator Approach to the Age-Period-Cohort Problem. *Demography*, 50(6), 1945–1967. https://doi.org/10.1007/s13524-013-0243-z

Manchikanti, L., Kaye, A. M., Knezevic, N. N., McAnally, H., Slavin, K., Trescot, A. M., et al. (2017). Responsible, Safe, and Effective Prescription of Opioids for Chronic Non-Cancer Pain: American Society of Interventional Pain Physicians (ASIPP) Guidelines. *Pain Physician*, 20(2S), S3–S92.

Mannheim, K. (1952). The problem of Generations. In *Essays on the Sociology of Knowledge* (Vol. 5, pp. 276–322). New York: Routledge.

Manthey, J., Shield, K. D., Rylett, M., Hasan, O. S. M., Probst, C., & Rehm, J. (2019). Global alcohol exposure between 1990 and 2017 and forecasts until 2030: a modelling study. *The Lancet*, 393(10190), 2493–2502. https://doi.org/10.1016/S0140-6736(18)32744-2

Martin, L. G., Freedman, V. A., Schoeni, R. F., & Andreski, P. M. (2009). Health and Functioning Among Baby Boomers Approaching 60. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 64B(3), 369–377. https://doi.org/10.1093/geronb/gbn040

Massey, D. S., & Denton, N. A. (1993). *American Apartheid: Segregation and the Making of the Underclass*. Harvard University Press.

Masters, R. K. (2012). Uncrossing the U.S. Black-White Mortality Crossover: The Role of Cohort Forces in Life Course Mortality Risk. *Demography*, 49(3), 773–796. https://doi.org/10.1007/s13524-012-0107-y

McBride, D. C. (1990). Generational Differences in HIV Risk and AIDS. *American Behavioral Scientist*, 33(4), 491–502. https://doi.org/10.1177/0002764290033004009

Meslé, F., & Vallin, J. (2000). Transition sanitaire : tendances et perspectives. *Médecine/sciences*, 16(11), 1161. https://doi.org/10.4267/10608/1549

Miech, R., Bohnert, A., Heard, K., & Boardman, J. (2013). Increasing Use of Nonmedical Analgesics Among Younger Cohorts in the United States: A Birth Cohort Effect. *Journal of Adolescent Health*, 52(1), 35–41. https://doi.org/10.1016/j.jadohealth.2012.07.016

Miech, R., Koester, S., & Dorsey-Holliman, B. (2011). Increasing US mortality due to accidental poisoning: the role of the baby boom cohort. *Addiction*, 106(4), 806–815. https://doi.org/10.1111/j.1360-0443.2010.03332.x

Murphy, E. L., Collier, A. C., Kalish, L. A., Assmann, S. F., Para, M. F., Flanigan, T. P., et al. (2001). Highly active antiretroviral therapy decreases mortality and morbidity in patients with advanced HIV disease. *Annals of Internal Medicine*, 135(1), 17–26.

Myles, J. (1998). How to Design a "Liberal" Welfare State: A Comparison of Canada and the United States. *Social Policy & Administration*, 32(4), 341–364. https://doi.org/10.1111/1467-9515.00120

NCHS. (2018). Data Access - Vital Statistics Online. http://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm. Accessed 5 December 2018

NVSS. (2019). Bridged-Race Population Estimates - Data Files and Documentation. Centers for Disease Control and Prevention. https://www.cdc.gov/nchs/nvss/bridged_race/data_documentation.htm. Accessed 25 May 2018

Palamar, J. J., Davies, S., Ompad, D. C., Cleland, C. M., & Weitzman, M. (2015). Powder Cocaine and Crack Use in the United States: An Examination of Risk for Arrest and Socioeconomic Disparities in Use. *Drug and alcohol dependence*, 149, 108–116. https://doi.org/10.1016/j.drugalcdep.2015.01.029

Palamar, J. J., & Ompad, D. C. (2014). Demographic and socioeconomic correlates of powder cocaine and crack use among high school seniors in the United States. *The American Journal of Drug and Alcohol Abuse*, 40(1), 37–43. https://doi.org/10.3109/00952990.2013.838961

Palella, F. J. J., Baker, R. K., Moorman, A. C., Chmiel, J. S., Wood, K. C., Brooks, J. T., et al. (2006). Mortality in the Highly Active Antiretroviral Therapy Era: Changing Causes of Death

and Disease in the HIV Outpatient Study. *JAIDS Journal of Acquired Immune Deficiency Syndromes*, 43(1), 27. https://doi.org/10.1097/01.qai.0000233310.90484.16

Palmer, B. (2008). Canada's 1960s: The Ironies of Identity in a Rebellious Era (First Edition edition.). Toronto ; Buffalo: University of Toronto Press, Scholarly Publishing Division.

Pampel, F. C. (2001). Gender equality and the sex differential in mortality from accidents in high income nations. *Population Research and Policy Review*, 20(5), 397–421. https://doi.org/10.1023/A:1013307620643

Patterson, T. L., & Jeste, D. V. (1999). The potential impact of the baby-boom generation on substance abuse among elderly persons. *Psychiatric Services*, 50(9), 1184–1188. https://doi.org/10.1176/ps.50.9.1184

Paulozzi, L. J., Budnitz, D. S., & Xi, Y. (2006). Increasing deaths from opioid analgesics in the United States. *Pharmacoepidemiology and Drug Safety*, 15(9), 618–627. https://doi.org/10.1002/pds.1276

PHAC. (2019). Reported cases from 1991 to 2016 in Canada - Notifiable diseases on-line. Public Health Agency of Canada. https://diseases.canada.ca/notifiable/charts?c=yl. Accessed 20 August 2019

Phillips, J. A. (2014). A changing epidemiology of suicide? The influence of birth cohorts on suicide rates in the United States. *Social Science & Medicine*, 114(Supplement C), 151–160. https://doi.org/10.1016/j.socscimed.2014.05.038

Preston, S. H., Heuveline, P., & Guillot, M. (2000). *Demography: Measuring and Modeling Population Processes* (1 edition.). Malden, MA: Wiley-Blackwell.

Puac-Polanco, V., Keyes, K. M., & Li, G. (2016). Mortality from motorcycle crashes: the baby-boomer cohort effect. *Injury Epidemiology*, 3, 19. https://doi.org/10.1186/s40621-016-0083-6

Quinones, S. (2015). *Dreamland: The True Tale of America's Opiate Epidemic*. New York: Bloomsbury Press.

Rao, R., & Roche, A. (2017). Substance misuse in older people. *BMJ*, 358, j3885. https://doi.org/10.1136/bmj.j3885

Rao, T. (2019). Baby boomers are increasingly more likely to risk drink-driving than millennials. *The Conversation*. http://theconversation.com/baby-boomers-are-increasingly-more-likely-to-risk-drink-driving-than-millennials-113130. Accessed 7 September 2019

Rau, R., Bohk, C., Muszynska, M. M., & Vaupel, J. W. (2013). Rates of Mortality Improvement on the Lexis Surface: Visualizing Age-, Period-, and Cohort-Effects. Presented at the PAA 2013, Washington, D.C. http://paa2013.princeton.edu/papers/130485

Reed, T. V. (2019). *The Art of Protest: Culture and Activism from the Civil Rights Movement to the Present*. U of Minnesota Press.

Remund, A., Camarda, C. G., & Riffe, T. (2018). A Cause-of-Death Decomposition of Young Adult Excess Mortality. *Demography*, 55(3), 957–978. https://doi.org/10.1007/s13524-018-0680-9

Rhodes, T. (1997). Risk theory in epidemic times: sex, drugs and the social organisation of 'risk behaviour.' *Sociology of Health & Illness*, 19(2), 208–227. https://doi.org/10.1111/1467-9566.ep10934410

Rodgers, W. L. (1982). Estimable Functions of Age, Period, and Cohort Effects. *American Sociological Review*, 47(6), 774–787. https://doi.org/10.2307/2095213

Rollins, J. (1986). Part of a Whole: The Interdependence of the Civil Rights Movement and Other Social Movements. *Phylon* (1960-), 47(1), 61–70. https://doi.org/10.2307/274695

Ross, N. A., Wolfson, M. C., Dunn, J. R., Berthelot, J.-M., Kaplan, G. A., & Lynch, J. W. (2000). Relation between income inequality and mortality in Canada and in the United States: cross sectional assessment using census data and vital statistics. *BMJ*, 320(7239), 898–902. https://doi.org/10.1136/bmj.320.7239.898

Rothstein, R. (2017). *The Color of Law: A Forgotten History of How Our Government Segregated America* (1 edition.). New York ; London: Liveright.

Rubery, J. (Ed.). (2012). *Women and Recession* (1 edition.). London: Routledge.

Rycroft, R. S. (2013). *The Economics of Inequality, Poverty, and Discrimination in the 21st Century* [2 volumes]. ABC-CLIO.

Ryder, N. B. (1965). The Cohort as a Concept in the Study of Social Change. *American Sociological Review*, 30(6), 843–861. https://doi.org/10.2307/2090964

Sauer, T., Ebeling, M., & Rau, R. (2018). The Fateful Cohorts. *American Journal of Public Health*, 108(10), e1–e2. https://doi.org/10.2105/AJPH.2018.304671

Schöley, J., & Willekens, F. (2017). Visualizing compositional data on the Lexis surface. *Demographic Research*, 36(21), 627–658. https://doi.org/10.4054/DemRes.2017.36.21

Shah, H., Bilodeau, M., Burak, K. W., Cooper, C., Klein, M., Ramji, A., et al. (2018). The management of chronic hepatitis C: 2018 guideline update from the Canadian Association for the Study of the Liver. *CMAJ*, 190(22), E677–E687. https://doi.org/10.1503/cmaj.170453

Sharp, G., & Hall, M. (2014). Emerging Forms of Racial Inequality in Homeownership Exit, 1968–2009. *Social Problems*, 61(3), 427–447. https://doi.org/10.1525/sp.2014.12161

Statistics Canada. (2018, November 8). Vital Statistics - Death Database (CVSD). http://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&SDDS=3233. Accessed 5 February 2019

Steger, M. A. E., Pierce, J. C., Steel, B. S., & Lovrich, N. P. (1989). Political culture, postmaterial values, and the new environmental paradigm: A comparative analysis of Canada and the United States. *Political Behavior*, 11(3), 233–254. https://doi.org/10.1007/BF00992298

Steil, J. P., Albright, L., Rugh, J. S., & Massey, D. S. (2018). The Social Structure of Mortgage Discrimination. *Housing studies*, 33(5), 759–776. https://doi.org/10.1080/02673037.2017.1390076

Stewart, A. J., Settles, I. H., & Winter, N. J. G. (1998). Women and the Social Movements of the 1960s: Activists, Engaged Observers, and Nonparticipants. *Political Psychology*, 19(1), 63–94. https://doi.org/10.1111/0162-895X.00093

Stewart, A. J., & Torges, C. M. (2014). Social, Historical, and Developmental Influences on the Psychology of the Baby Boom at Midlife. In *The Baby Boomers Grow Up: Contemporary Perspectives on Midlife* (pp. 23–43). New York: Psychology Press.

Strader, D. B. (2005). Coinfection with HIV and Hepatitis C Virus in Injection Drug Users and Minority Populations. *Clinical Infectious Diseases*, 41(Supplement_1), S7–S13. https://doi.org/10.1086/429489

Tapper, E. B., & Parikh, N. D. (2018). Mortality due to cirrhosis and liver cancer in the United States, 1999-2016: observational study. *BMJ*, k2817. https://doi.org/10.1136/bmj.k2817

Termote, M. (1998). L'indice synthetique de mortalite. Un indicateur meconnu. In Morbidité, Mortalité : problèmes de mesure, facteurs d'évolution, essai de prospective (pp. 183–190). Presented at the Colloque international de Sinaia - AIDELF, Sinaia: Association Internationale des Demographes de Langue Francaise. https://www.ncbi.nlm.nih.gov/nlmcatalog/101079058. Accessed 17 October 2017

Turner, S. E., & Bound, J. (2002). Closing the Gap or Widening the Divide: The Effects of the G.I. Bill and World War II on the Educational Outcomes of Black Americans (Working Paper No. 9044). National Bureau of Economic Research. https://doi.org/10.3386/w9044

USCB. (2018). Historical Marital Status Tables. US Census Bureau. https://www.census.gov/data/tables/time-series/demo/families/marital.html. Accessed 27 August 2019

Vaupel, J. W., Gambill, B. A., & Yashin, A. I. (1987, July). Thousands of Data at a Glance: Shaded Contour Maps of Demographic Surfaces. Monograph. http://pure.iiasa.ac.at/2905/. Accessed 28 March 2016

Vaupel, James W., Manton, K. G., & Stallard, E. (1979). The Impact of Heterogeneity in Individual Frailty on the Dynamics of Mortality. *Demography*, 16(3), 439–454. https://doi.org/10.2307/2061224

Veevers, J. E., & Gee, E. M. (1986). Playing It Safe: Accident Mortality and Gender Roles. *Sociological Focus*, 19(4), 349–360.

Vittinghoff, E., Scheer, S., O'Malley, P., Colfax, G., Holmberg, S. D., & Buchbinder, S. P. (1999). Combination Antiretroviral Therapy and Recent Declines in AIDS Incidence and Mortality. *The Journal of Infectious Diseases*, 179(3), 717–720. https://doi.org/10.1086/314623

Vozoris, N. T., Wang, X., Fischer, H. D., Bell, C. M., O'Donnell, D. E., Austin, P. C., et al. (2016). Incident opioid drug use and adverse respiratory outcomes among older adults with COPD. *European Respiratory Journal*, 48(3), 683–693. https://doi.org/10.1183/13993003.01967-2015

Waldron, I., McCloskey, C., & Earle, I. (2005). Trends in gender differences in accidents mortality: Relationships to changing gender roles and other societal trends. *Demographic Research*, S4(17), 415–454. https://doi.org/10.4054/DemRes.2005.13.17

Woolf, S. H., Chapman, D. A., Buchanich, J. M., Bobby, K. J., Zimmerman, E. B., & Blackburn, S. M. (2018). Changes in midlife death rates across racial and ethnic groups in the United States: systematic analysis of vital statistics. *BMJ*, 362, k3096. https://doi.org/10.1136/bmj.k3096

Yang, Y., Fu, W. J., & Land, K. C. (2004). A Methodological Comparison of Age-Period-Cohort Models: The Intrinsic Estimator and Conventional Generalized Linear Models. *Sociological Methodology*, 34(1), 75–110. https://doi.org/10.1111/j.0081-1750.2004.00148.x

Zang, E., Zheng, H., Yang, Y. C., & Land, K. C. (2019). Recent trends in US mortality in early and middle adulthood: racial/ethnic disparities in inter-cohort patterns. *International Journal of Epidemiology*. https://doi.org/10.1093/ije/dyy255

Zheng, H. (2014). Aging in the Context of Cohort Evolution and Mortality Selection. *Demography*, 51(4), 1295–1317. https://doi.org/10.1007/s13524-014-0306-9

Zibbell, J. E., Asher, A. K., Patel, R. C., Kupronis, B., Iqbal, K., Ward, J. W., & Holtzman, D. (2017). Increases in Acute Hepatitis C Virus Infection Related to a Growing Opioid Epidemic and Associated Injection Drug Use, United States, 2004 to 2014. *American Journal of Public Health*, 108(2), 175–181. https://doi.org/10.2105/AJPH.2017.304132

9. Supplementary Materials

9.1. Construction of Lexis surfaces of mortality change

Lexis surfaces of changes in mortality rates are widely recognized in the demographic literature as powerful, yet simple tools for identifying APC effects (Barbi and Camarda 2011; Rau et al. 2013; Schöley and Willekens 2017; J. W. Vaupel et al. 1987). Over periods (and, thus, cohorts) these changes reflect a combination of period and cohort effects, because age is controlled by estimating mortality changes within the same age group (i.e., horizontal mortality changes in the Lexis surfaces, from earlier to more recent calendar years/cohorts). To construct Lexis surfaces reflecting these changes, we first estimated two-dimensional smoothed mortality rates to eliminate random variations that are not part of the mortality trend. We applied the P-splines method (Eilers et al. 2015; Eilers and Marx 1996) for the two-dimensional smoothing, using the R package *MortalitySmooth* (Camarda 2012), which allowed us to select the best fitting parameters based on the Akaike Information Criteria (AIC) (Burnham and Anderson 2002). From the smoothed death rates, we estimated the rates of mortality change ($\Delta pc_{x,t}$), and then plotted them in a Lexis surface. Additional information about the estimation of the relative changes in mortality and the construction of the Lexis surfaces can be found in the supplementary materials. According to the diagonal patterns shown in Figure 1, the advantaged and the disadvantaged birth cohorts were born during the mid-1940s and around 1960, respectively (black dashed lines).

From the smoothed death rates, we estimated, for each age *x,* the relative change in mortality from year *t-1* to year *t* (or from cohort *c-1* to cohort *c*) as:

$$\Delta pc_{x,t} = \log(m_{x,t}^{s}) - \log(m_{x,t-1}^{s}), \tag{1}$$

where $m_{x,t}^{s}$ is the smoothed death rate for age *x* in period *t*.

We then plotted $\Delta pc_{x,t}$ values in a Lexis surface in two color scales to depict the yearly changes in mortality over periods/cohorts (Figure 1). The relative mortality decrease for year *t* compared to that for year *t-1* (or cohort *c* compared to cohort *c-1*) in the same age *x* is indicated with a green-to-blue scale, while the relative mortality increase is indicated with a yellow-to-red scale. Vertical traces on the Lexis surface are indicative of nonlinear period effects on mortality, and 45° diagonal traces are indicative of nonlinear cohort effects.

9.2. Cohort partial mortality rate measure

For the estimation and comparison of cohort mortality levels, we propose an index of the *cohort's partial mortality rate*, defined as:

$$CPMR^{c(k,l)} = \sum_{x=k}^{l} m_x^c ,\qquad (2)$$

where m_x is the age-specific mortality rate for the age interval $k - l$ for cohort *c*. Additional information on this method and the attributes that make it convenient for our purposes are detailed in the supplementary materials.

A similar index (*indice synthétique de mortalité*) was suggested by Termote (1998) as a complementary measure for analyzing mortality changes on a period basis. Being the sum of the age-specific mortality rates between two ages, this measure is the mortality analogous of the cohort's total fertility rate (TFR^C) (Preston et al. 2000), but framed within a specific age interval. This index is appropriate for our objective for at least three reasons. First, it is not influenced by variations in size across ages or cohorts. Second, contrary to other measures of mortality, such as life expectancy or life years lost, the $CPMR^{c(k,l)}$ is not weighted by age – that is, it does not overestimate the importance of the causes of death that are more prevalent in the younger age groups. Third, the index is fairly easy to decompose by causes of death.

The *change in the cohort's partial mortality rate* between the advantaged (a) and disadvantaged (d) cohorts for the age interval $k - l$ is defined as

$$\Delta CPMR^{d-a(k,l)} = CPMR^{d(k,l)} - CPMR^{a(k,l)}.\qquad (3)$$

The decomposition of the $\Delta CPMR^{Cd-a(k,l)}$ by cause of death is straightforward, since this index satisfies a simple balance equation in which the sum of all *changes in the cohort's partial mortality rate by cause of death i* $(\Delta CPMR_i^{d-a(k,l)})$ equals the total *change in the cohort's partial mortality rate*:

$$\Delta CPMR^{d-a(k,l)} = \sum_i \Delta CPMR_i^{d-a(k,l)}.\qquad (4)$$

9.3. Classification of causes of death and measurement of mortality change

The period under analysis spans three ICD revisions (8th through 10th). To facilitate an initial decomposition by cause of death of the mortality deterioration, we first constructed broad causes of death based on the ICD chapters (see Table S1). This broad categorization allowed us to analyze mortality changes across a few groups of causes, and guaranteed a low degree of variation across the three ICD revisions covered during the period of observation.

Table S1. ICD Chapters revisions 8th to 10th

ICD	8	9	10
Period	1968-1978	1979-1998	1999-2016
Certain infectious and parasitic diseases	001-139	001-139	A00–B99
Neoplasms	140-239	140-239	C00–D48
Endocrine, nutritional, and metabolic diseases	240-279	240-279	E00–E90
Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	280-289	280-289	D50–D89
Mental and behavioral disorders	290-319	290-319	F00–F99
Diseases of the nervous system	320-359	320-359	G00–G99
Diseases of the eye and adnexa	360-379	360-379	H00–H59
Diseases of the ear and mastoid process	380-389	380-389	H60–H95
Diseases of the circulatory system	390-459	390-459	I00–I99
Diseases of the respiratory system	460-519	460-519	J00–J99
Diseases of the digestive system	520-579	520-579	K00–K93
Diseases of the genitourinary system	580-629	580-629	N00–N99
Pregnancy, childbirth, and the puerperium	630-679	630-679	O00–O99
Diseases of the skin and subcutaneous tissue	680-709	680-709	L00–L99
Diseases of the musculoskeletal system and connective tissue	710-739	710-739	M00–M99
Congenital malformations, deformations, and chromosomal abnormalities	740-759	740-759	Q00–Q99
Certain conditions originating in the perinatal period	760-779	760-779	P00–P96
External causes of morbidity and mortality	800-999	800-999	V01–Y98
Other causes	780-799	780-799	R-U

Figure S1 shows the estimates of the cause-specific decomposition of the mortality deterioration from the advantaged to the disadvantaged cohorts. According to these results, most of the excess

mortality among boomers is composed of increases in deaths from causes within the ICD Chapters covering external, infectious, digestive, mental/behavioral, and respiratory diseases.

Figure S1: Percentage contributions of broad causes to the mortality deterioration from the *advantaged* to the *disadvantaged* cohorts
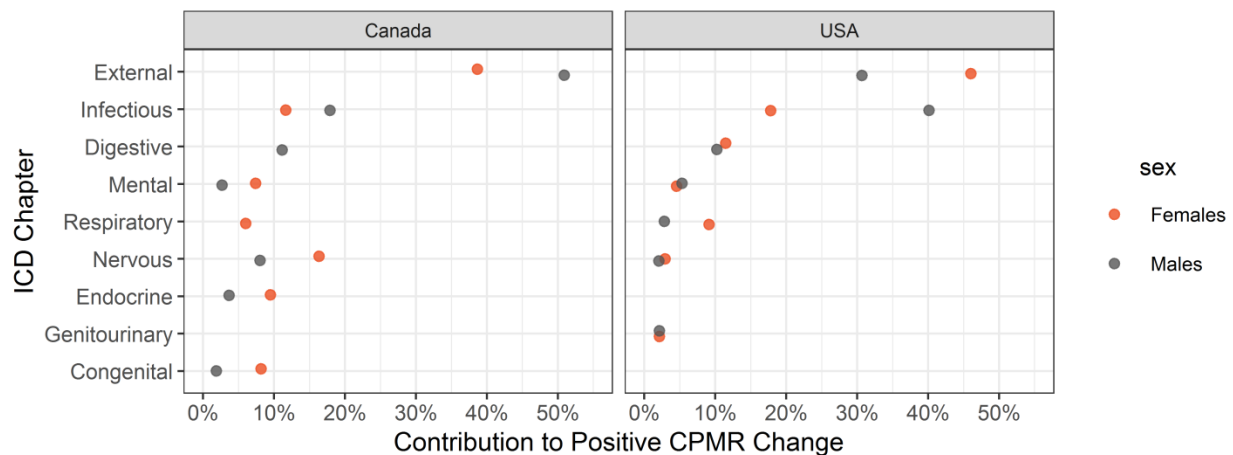
Based on the leading broad causes of mortality deterioration identified in Figure S1, we constructed more detailed causes of death, and decomposed the mortality deterioration again. Table S2 presents the ICD codes used to classify deaths from HIV/AIDS, hepatitis C, COPD, suicide, alcohol, and drugs. Note that none of these causes of death was confined to the same broad category – that is, to the same ICD chapter. For instance, alcohol-related mortality included deaths from mental and behavioral disorders due to alcohol (which are covered by the mental and behavioral disorders chapter), from alcoholic liver disease (which are covered by the digestive system chapter), and from alcohol poisoning (which are covered by the external causes chapter).

Table S2: ICD codes included in each category of causes of death, revisions 8th to 10th

	ICD	8	9	10
	Period	1968-1978	1979-1998	1999-2016
HIV/AIDS		NA	0420-0449	B20-B24
Hepatitis C		NA	0704-0705	B171, B182
Chronic lower respiratory diseases		4900-4939	4900-4939	J40-J47
Suicides		9500-9599	9500-9599	X60-84

ICD	8	9	10
Period	1968-1978	1979-1998	1999-2016
Drug-related causes (accidental overdoses + drug dependence)	2943, 3040-3049, 3091, 8500-8599, 9800-9803	2920-2929, 3040-3049, 3052-3059, 8490-8589, 9800-9805	F11-19, F55, X40-44, Y10-14
Alcohol-related causes (accidental alcohol intoxication + long-term harm from liver cirrhosis +)	2910-2919, 3030-3039, 5353, 5710, 8600-8609	2910-2919, 3030-3039, 3050, 3575, 4255, 5353, 5710-5713, 7903, 8600-8609	E244, F10, G312, G621, G721, I426, K292, K700-K709, K860, X45, Y15, Y90, Y91

Figure S2 depicts the contributions to the mortality deterioration of each cause of death. Increases in mortality from HIV/AIDS, hepatitis C, COPD, suicide, alcohol, and drugs contributed between 75% and 80% of the deterioration in mortality from the advantaged to the disadvantaged cohorts for both sexes in Canada and the United States.

Figure S2. Percentage contributions to the increase in $\Delta CPMR^{d-a(35,54)}$
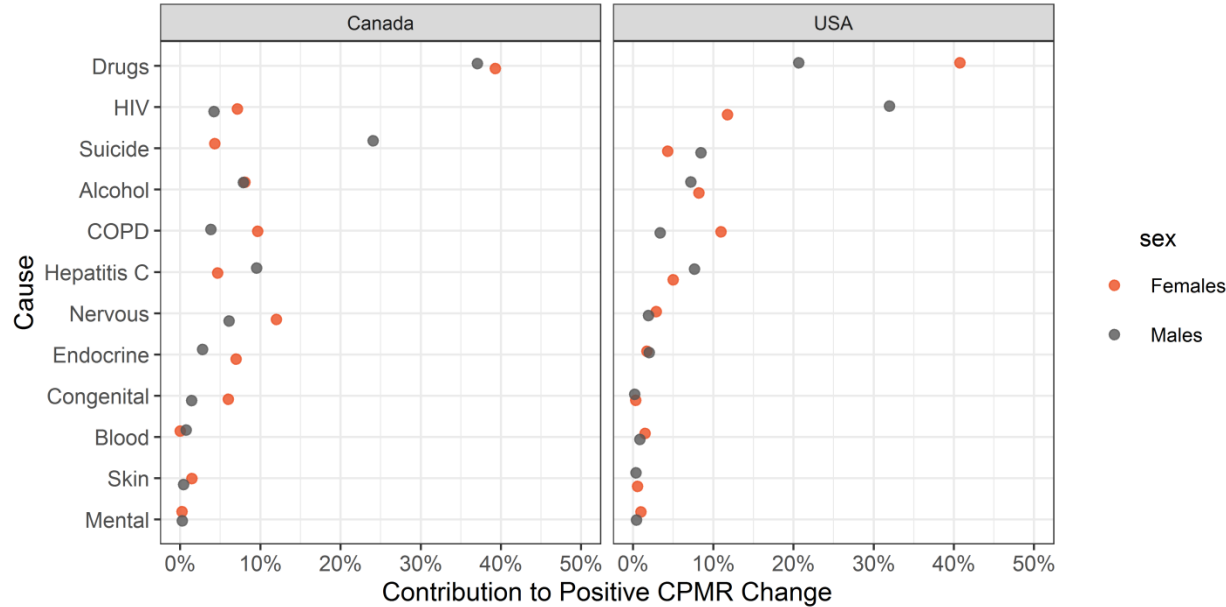
Notes: Only categories with positive contributions to $\Delta CPMR^{d-a(35,54)}$ of least 2% in the four subpopulations were included.

Estimates from the $\Delta CPMR_i^{d-a(35,54)}$ depicted in Figure 3 in the main text were useful for identifying the causes of death that made the largest contributions to the relative mortality deterioration between the *advantaged* and the *disadvantaged* cohorts within the age interval 35-54y.

However, this measure has two limitations. First, whereas the variable age is controlled when the mortality levels are compared within the same age interval, the variable period is not; consequently, changes in mortality over cohorts are confounded with changes over periods. For instance, the estimates of cohort differences in drug-related mortality levels could be the result of period variations. During the observed age interval, i.e., 35-54, the earliest cohorts were exposed for a shorter period of time and at incipient stages of the opioid epidemic (e.g., because the cohort 1945 was observed during the period 1980-1999, they had only been exposed to the initial years of the opioid crisis, which started in the late 1990s). The more recent cohorts, by contrast, had been exposed to the crisis for a more extended period, when it was in its more advanced stages (e.g., the cohort 1955 was observed during the period 1990-2009, when the opioid crisis was fully underway).

Second, while the decomposition of $\Delta CPMR^{d-a(35,54)}$ allows us to identify the causes of the relative mortality deterioration between the disadvantaged and the advantaged cohorts, because of how our window of observation was configured, we were not able to identify whether the causes of death that were responsible for the deterioration were also responsible for the subsequent improvements in mortality for the cohorts born after the boomers. If that was not the case (i.e., if the causes of the mortality deterioration were different from the causes of the subsequent improvements), the excess in all-cause mortality among the boomers would not be strictly related to the sum of multiple cause-specific excesses, but would instead be an artifact of more intricate processes involving increases in some causes and decreases in others.

To overcome these two limitations, and to properly assess the cohort's excess mortality by cause of death, we need to account simultaneously for variations over the three age-period-cohort (APC) dimensions.

9.4. Alternative selection of disadvantaged cohorts

The selection criteria of the disadvantaged cohorts depicted in Figure 2 could be problematic for two reasons. First, for Canadian males, the cohorts 1954 and 1957 have similar degrees of deviance from the linear trend of mortality (172 and 179, respectively), and it could be argued that $CPMR^{c(35,54)}$ is in absolute terms larger in 1954 than in 1957 (5,545 and 5,288, respectively). Second, since the highest degree of positive deviance from the mortality trend for U.S. females was reached by the cohort 1960 – i.e., the last cohort observed – we were not able to determine whether the deterioration in mortality continued among more recent cohorts. To address these points, we tested the consistency of our estimates by selecting 1954 as the disadvantaged cohort for Canadian males, and by extending the estimation of $CPMR^{c(35,54)}$ to more recent cohorts for U.S. females. These estimations are presented in the supplemental materials (see Figures S3 to S5).

The cause-specific contributions to the deterioration in mortality from the advantaged to the disadvantaged cohorts were sensitive to the locations imposed on these cohorts. In order to test the consistency of the estimates presented in Figure 3, we chose alternative locations for the disadvantaged cohorts in the cases in which the greatest divergence from the linear trend was not obvious. For Canadian males, the degree of deviation was roughly similar for the cohorts 1954 and 1957. Hence, we use 1954 as an alternative disadvantaged cohort. For U.S. females, the level of divergence did not stop increasing over the observed cohorts. We extended the estimation up to the cohort 1963, which was only possible by reducing the age interval to 35-53. These $CPMR^{c(35,53)}$ estimates between the cohorts 1940 and 1963 for U.S. females are depicted in Figure S3. According to these findings, the most disadvantaged cohort for this age interval is located in 1958.

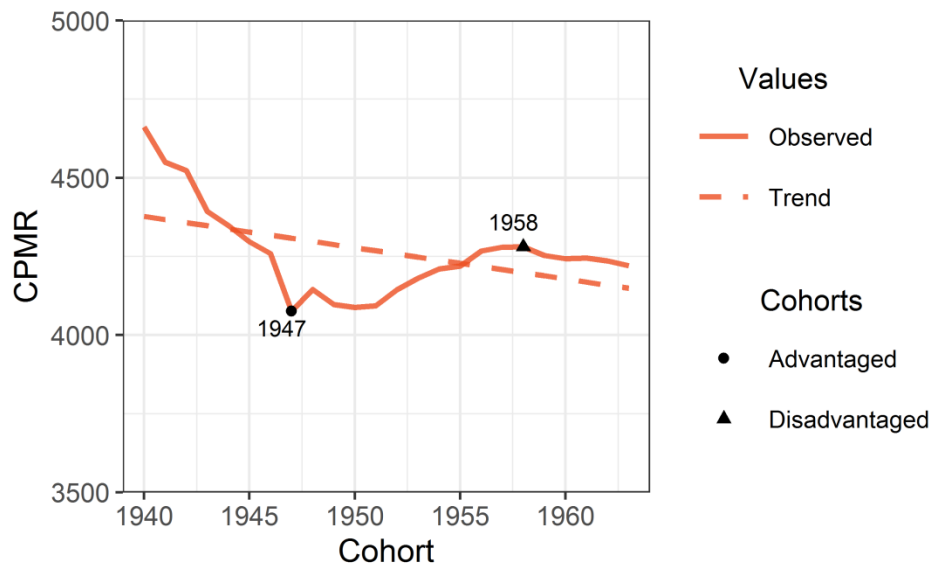**Figure S3. Alternative estimates of the cohorts' partial mortality rates for U.S. females**

Figure S3. Cohorts' partial mortality rates (solid line) and the linear trend (dashed line) for U.S. females within the age interval 35-53 ($\text{CPMR}^{c(35,53)}$), between the cohorts 1940 and 1963. The labels indicate the year of birth of the *advantaged* (circular shape) and the *disadvantaged* (triangular shape) cohorts.

In Figure S4, we compare the contributions by cause to the mortality deterioration of the *advantaged* and the *alternative disadvantaged* cohorts for Canadian males and U.S. females (in purple). To facilitate this comparison, the plot also presents the estimations of the cause-specific contributions obtained with the original disadvantaged cohorts (in green). Similarly, Figure S5 shows the cumulative contributions of the six leading causes of death to the mortality deterioration. As Figures S4 and S5 show, the contributions by cause of death to the mortality deterioration were highly similar between the estimates using the original location and those using the alternative location of the disadvantaged cohorts.

According to the estimates presented in Figures S3 to S5, the contributions of the leading causes to the mortality deterioration from the advantaged to the disadvantaged cohorts did not differ substantially when different disadvantaged cohorts were selected for Canadian males and U.S. females.

Figure S4. **Alternative estimates of percentage contributions to the increase in** $\Delta CPMR^{d-a(k,l)}$ **for Canadian males and U.S. females**
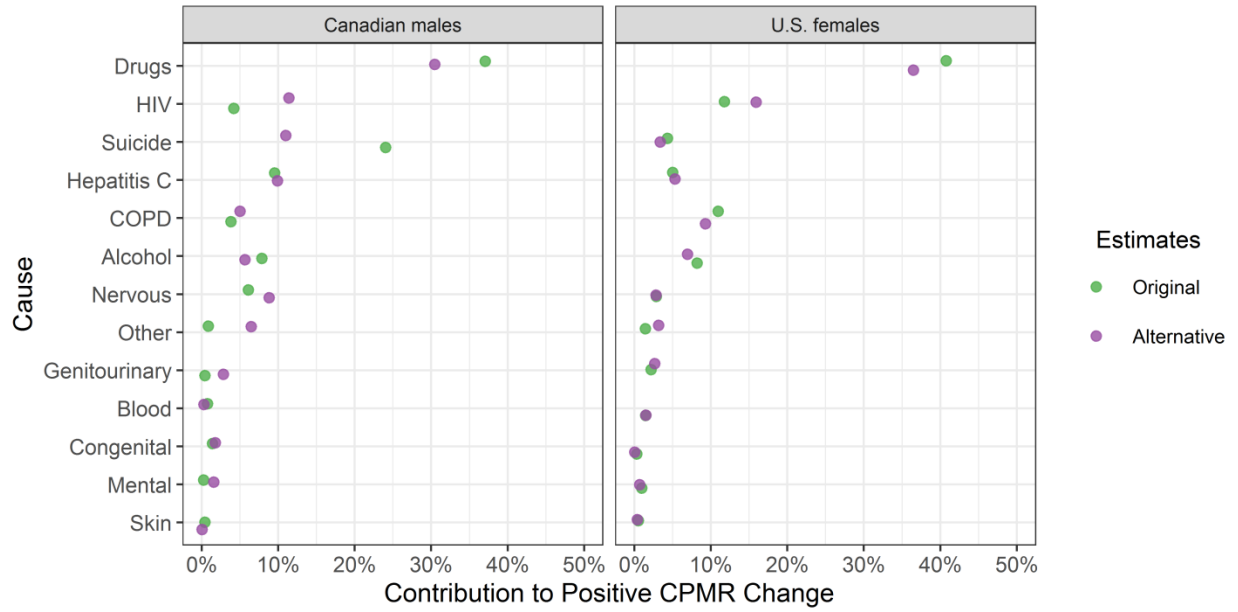
Notes: Percentage contributions to the increase in $\Delta CPMR^{d-a(k,l)}$ by cause of death for Canadian males ($\Delta CPMR^{d-a(35,54)}$) and U.S. females ($\Delta CPMR^{d-a(35,53)}$), according to the original (respectively, 1957 and 1960, in green) and the alternative (respectively, 1954 and 1958, in purple) disadvantaged cohorts. Only causes that contributed to mortality deterioration in all cases are shown.

Figure S5. **Alternative estimates of cumulative contributions by leading causes to the deterioration in mortality from the advantaged to the disadvantaged cohorts**
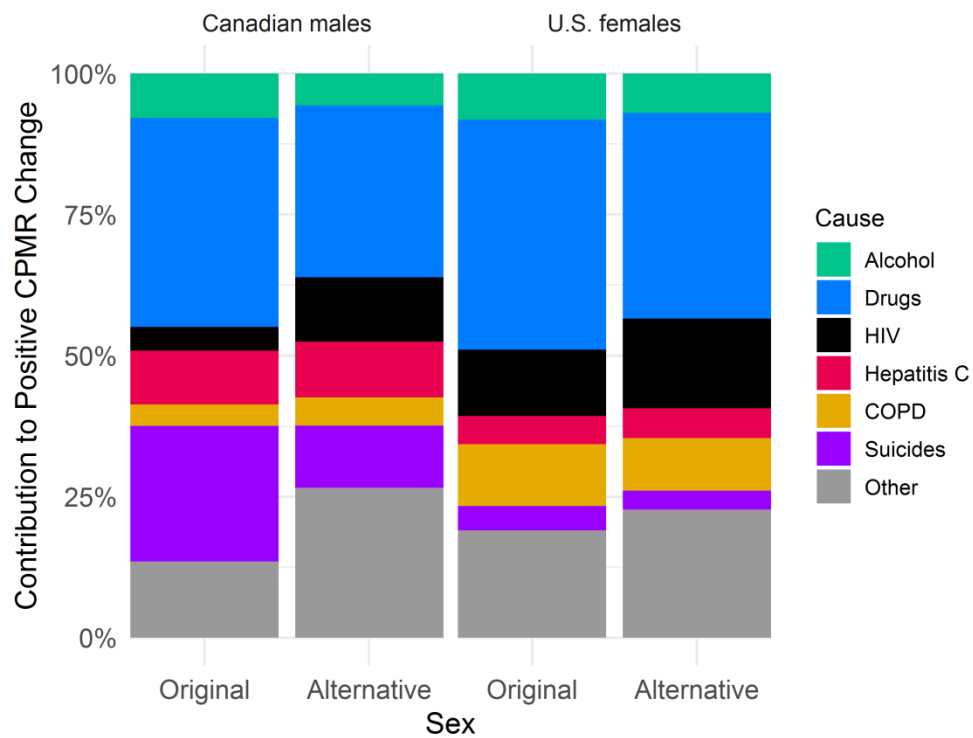
Notes: Estimates according to the location of the disadvantaged cohort. For Canadian males, the disadvantaged cohort was originally placed in 1957, and alternatively in 1954. For U.S. females, the disadvantaged cohort was originally placed in 1960, and alternatively in 1958.

9.5. Detrended cohort effects from the APC model

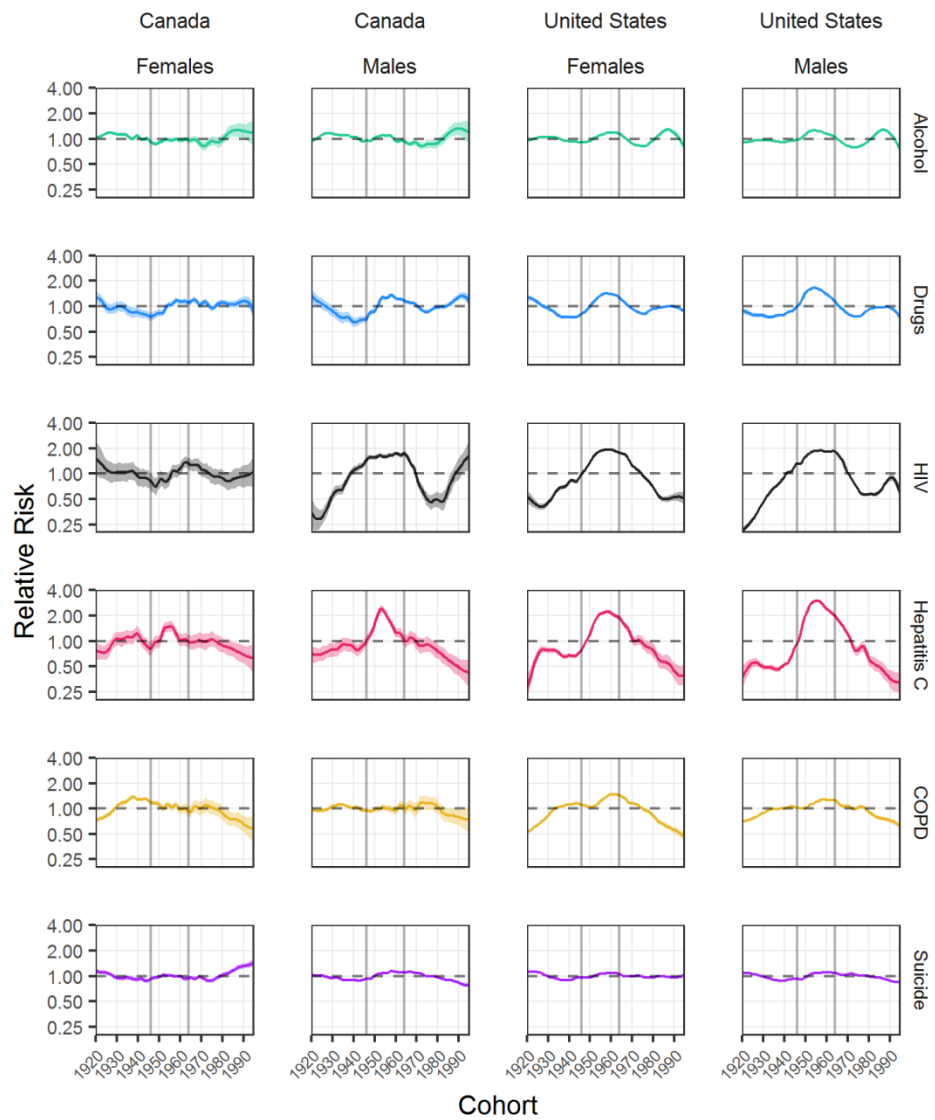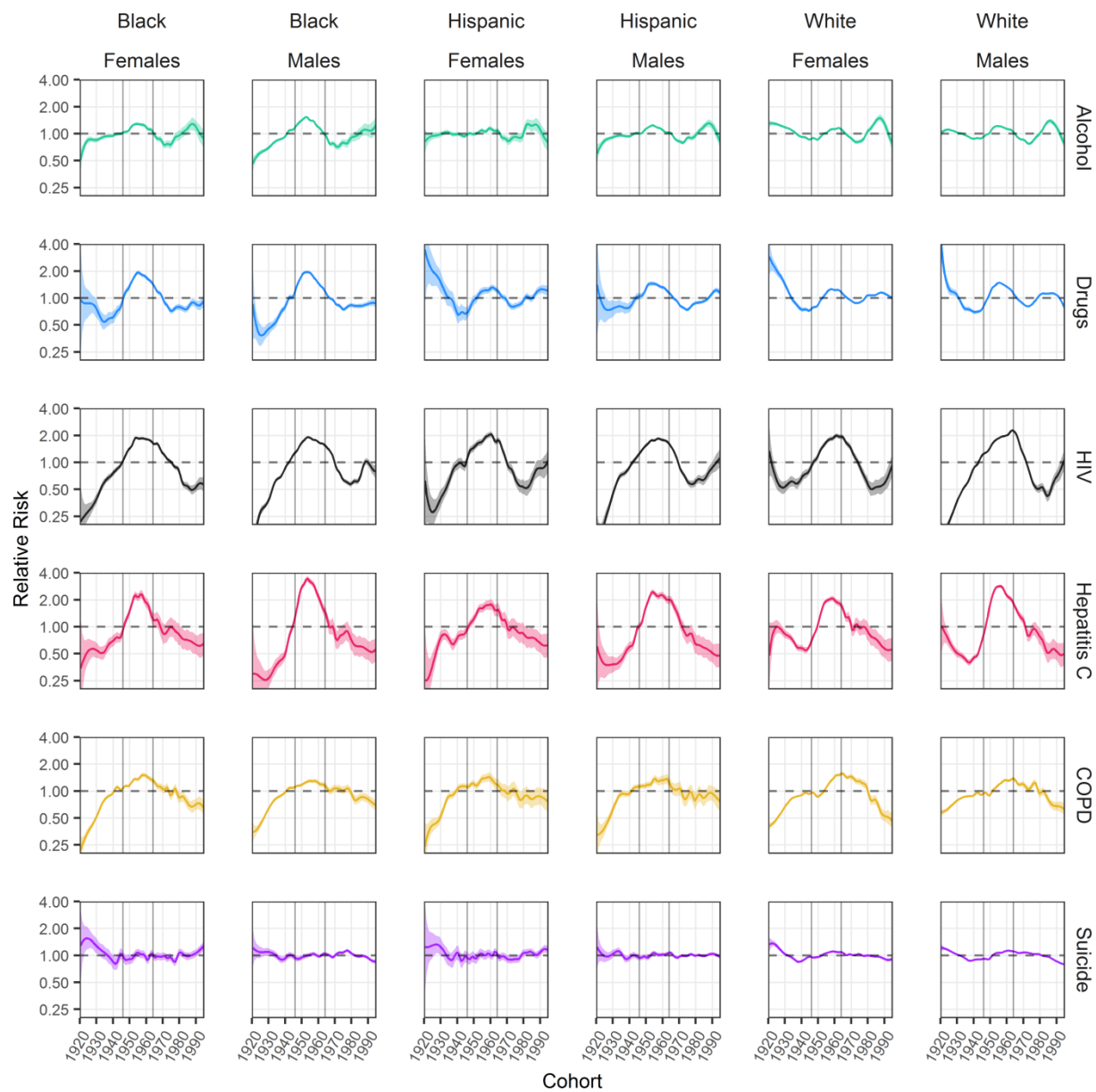**Figure S6**: APCd estimates by cause of death, sex, and country.

Figure S7: **APCd estimates for each sex and racial-ethnic group, comparing the leading causes of the boomers' excess mortality**

9.6. Temporal dynamic of the excess mortality among boomers by cause of death

Figure S8: Variation in relative risk at the ridge compared to the baseline over time by country
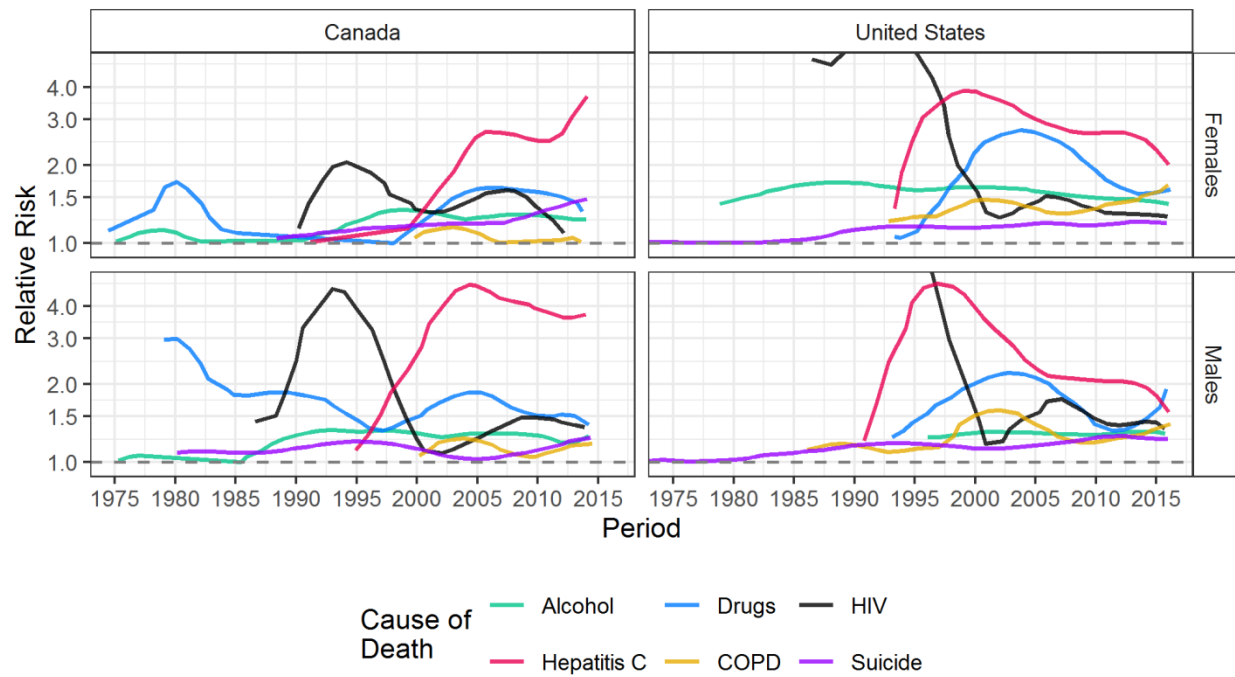
Figure S9: Variation in relative risk at the ridge compared to the baseline over time by race/ethnicity