The Boomer Penalty: Excess Mortality among Baby Boomers in Canada and the United States

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Abstract

Studies suggest that baby boomers in Canada and the United States have experienced a slowdown, or even deterioration, in the all-cause mortality improvements relative to neighboring cohorts. These findings are counterintuitive and surprising. According to the technophysio evolution theory, 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 generations born earlier.

The present study explores mechanisms that could have produced the excess mortality for 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 contribution of causes of death that are likely driving this cohort's excess mortality and their dynamic over time. The analyses are done using demographic decomposition, visual, and statistical methods.

We found evidence of a higher susceptibility of the trailing edge boomers to behavioral causes of death, namely mortality from drugs, alcohol, HIV/AIDS, hepatitis C, COPD, and suicides. Most of these causes contributed to the all-cause mortality disadvantage of boomers by sustained cohort effects that escorted the cohorts over time. This finding gives little support to 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 march of an alleged continuous progress in health, and perhaps the baby boom is one of them. This invites a rethinking of the mechanisms driving current age-period-cohort mortality patterns. Mechanisms that can generate the observed cohort disadvantage, such as more prevalent levels of distress and frustration among boomers –the birth cohort effect proposed by Easterlin–, and the riskier attitudes toward drug use and sexual practices that are constituent of the boomer generation identity are addressed and discussed.

Introduction

Previous research has established that all-cause mortality improvements slowed down or even reversed for baby boomers in Canada (Bourbeau and Ouellette 2016) and the United States (Canudas-Romo and Guillot 2015; Rau et al. 2013), respectively. These findings were unexpected, and they are 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. boomers regarding overdoses (Chauvel et al. 2016) and other external causes (Remund et al. 2018; Zang et al. 2019). U.S. boomers also reported having a more unsatisfactory health performance when compared to neighboring birth cohorts, and 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; D. E. King et al. 2013; Leveille et al. 2005; Martin et al. 2009). There are at least four alternative mechanisms that may drive such boomer penalty. First, the relatively low level of mortality at early ages among boomers could have increased the health heterogeneity within the cohorts, resulting in a decrease of the physiological capital average (Canudas-Romo and Guillot 2015). Second, the large size of the boomer cohorts compounded by the contrasted socioeconomic contexts in childhood and adulthood likely increased the prevalence of stress and frustration among boomers (Easterlin 1987). A third postulate states that the distinctive riskier attitudes and behaviors of the boomer generational identity has increased their mortality risks (Johnston 1991). Finally, the slow or lack of mortality improvements among the boomers could have little to do with usual cohort effects and result instead from multiple and unrelated period crises that targeted these cohorts at different life stages.

Despite the importance of this phenomenon for understanding the mortality pattern in several western societies, there is still little understanding of the boomer disadvantage. Much of the emphasis has mostly been placed on long-term cohort changes, overlooking the relative, short-term differences across adjacent cohorts. Research to date has not determined the boomers' excess mortality by cause or its temporal dynamic. Furthermore, there is still uncertainty as to whether the composition and temporal pattern of such excess are similar across sexes, race, ethnicities, and countries.

This paper attempts to compare the cause-specific contribution to the boomer excess mortality and the temporal patterns of these contributions. There are two primary objectives of this study. 1) To examine the causes of death underlying the boomer excess mortality in Canada and the United States. 2) To check whether the disadvantage operates as a sequence of unrelated period crises that disproportionately targeted the boomer cohorts at different ages, or as sustained cause-specific disadvantages that continually followed the boomers throughout their life course. In other words, we aim to identify 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 dynamic 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 dynamic of the boomer's disadvantage in mortality in Canada and across races and ethnicities in the United States. This study aims to offer hints for evaluating the proposed mechanisms underlying the excess in mortality among boomer cohorts. On the one hand, the identification of the leading causes underlying the boomers' excess mortality will provide some clues about the contribution of extrinsic and intrinsic factors. On the other hand, the temporal dynamic of these contributions will serve to determine whether this death penalty resulted from a sequence of age-period interaction shocks (i.e., temporary bruises) or from sustained cohort disadvantages (i.e., permanent scars).

Data and Analytical Strategy

Data

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 years 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 years of age between 1974 and 2016 were retrieved using mortality microdata from

the National Center for Health Statistics (2018). Information about race and ethnicity was included in U.S. death certificates since 1990.

The period under analysis spans three International Classification of Diseases (ICD) revisions (8th, 9th, and 10th). Table S1 (in the supplement material) shows the employed codes used to identify mortality by major causes of death in each ICD revision. For these broad categories we did not find important disruptions during the observed period of time.
Annual counts of the Canadian and U.S. population at risk by sex and single years of age (0-100) between 1959 and 2016 were taken from the Human Mortality Database (2019). The racial/ethnic proportions within the U.S. population estimates between 1990 and 2016 were obtained from the Bridged-Race Population Estimates (NVSS 2019).

Strategy of data analysis

We analyzed boomers' excess mortality for each country and each sex in four steps. We first located the cohorts with the lowest and the highest mortality deviation from the linear trend, respectively called the *advantaged* and *disadvantaged* cohorts. Then we identified the leading causes that contributed to such mortality deviation as a second step. Third, for each of these leading causes, we estimated the cohort effects on mortality when age and period variations are accounted for. Then, we analyzed the temporal dynamic of these cohort effects by cause. Because Canadian and U.S. populations are highly heterogeneous in terms of race and ethnicity –with important differences in mortality (Masters 2012; Woolf et al. 2018; Zang et al. 2019)–, we also made 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 clearness, we present, for each step of the analysis, a detailed description of the methods, immediately followed by the results obtained from their application.

Analysis of the Boomers' Excess Mortality

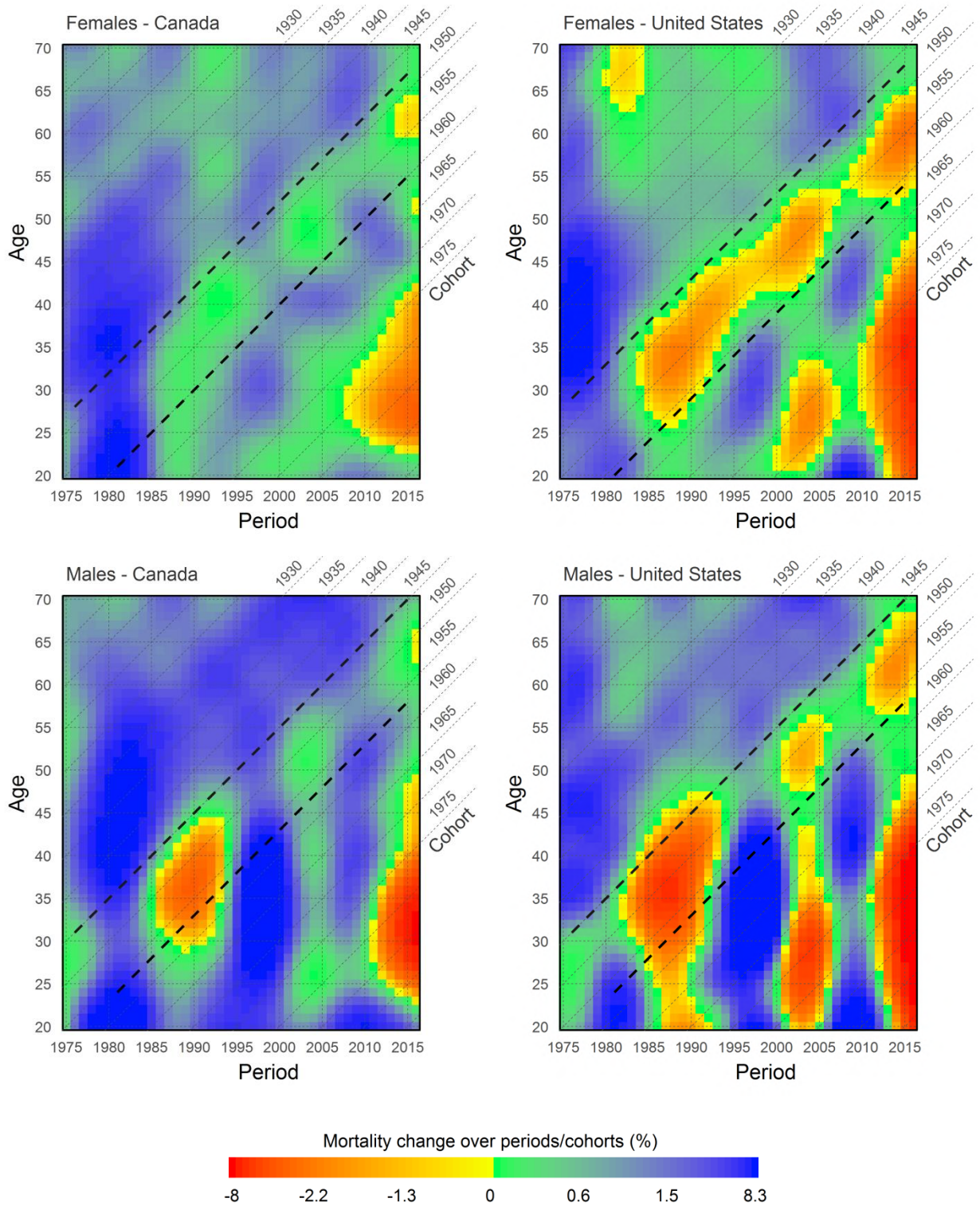
Cause-specific contribution to the boomers' excess mortality

To decompose the boomer excess mortality by leading causes of death, we first detected those cohorts located at beginning (advantaged) and ending (disadvantaged) of the relative mortality deterioration. Second, we identified the leading causes of death responsible for it.

Identification of the advantaged and disadvantaged cohorts

We first plotted Lexis surfaces of smoothed mortality change from one period to the next within the same age (see Figure 1) and then we pinpointed the cohorts located at the onset and at the end of the mortality deterioration –i.e., the *advantaged* and *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 change in mortality, and the construction of the Lexis surfaces is presented in the Supplementary Material.

Figure 1. Lexis surfaces of mortality change over period/cohort.



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 a yellow-to-red scale indicating 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 rates decreased with time reaching their minimum value in 2000 (i.e., cohort 1950). From 2000 to 2006, the death rates increased over time until hitting a 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.

According to the diagonal patterns shown in Figure 1, the *advantaged* and the *disadvantaged* birth cohorts centered in 1940 and 1960, respectively (black dashed lines). To identify their precise location in the lexis configuration we need to compare the mortality across cohorts. For this, we propose an index of the cohort's partial mortality rate ($\mathrm{CPMR}^{\mathrm{c(k,l)}}$). This index is the sum of the age-specific death rates along the cohort c, between ages *k* and *l*. See the Supplementary Material for more details about the formulation and attributes of the $\mathrm{CPMR}^{\mathrm{c(k,l)}}$.

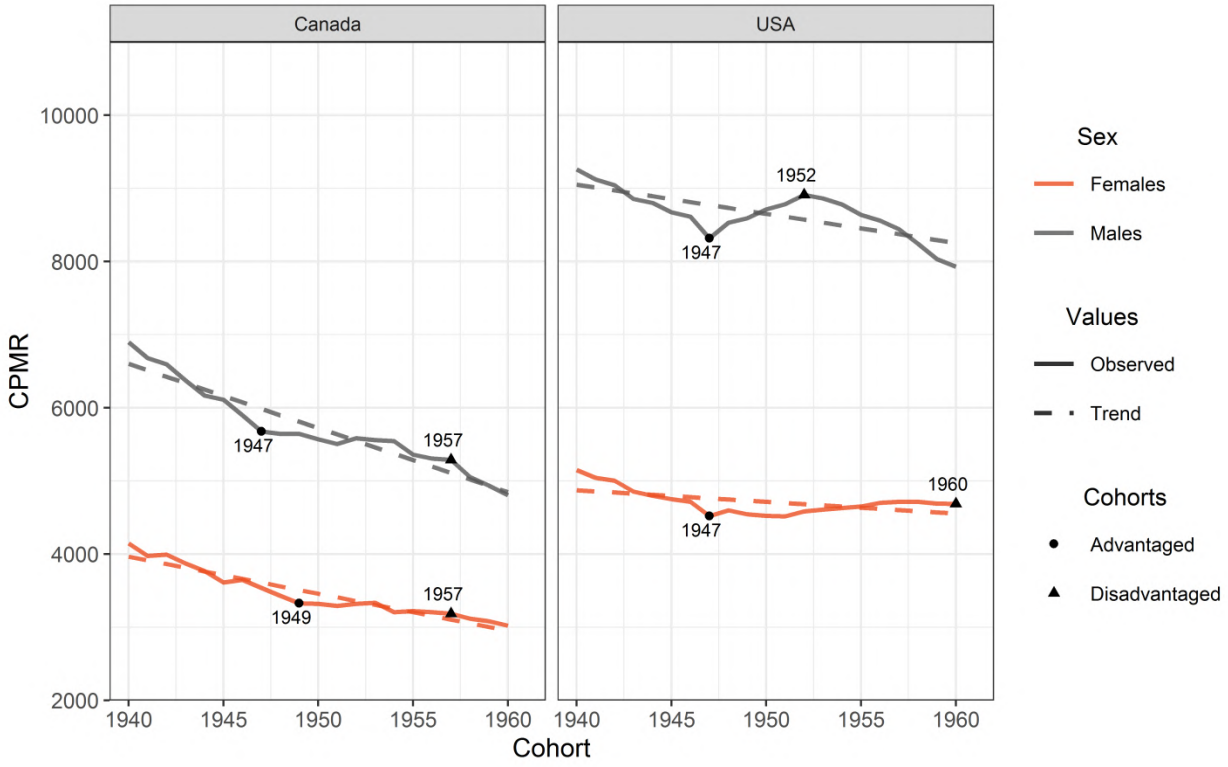
Since our goal is to identify relative and not absolute mortality changes, we base our analysis on the deviance from the linear trend in mortality. We obtained the linear trend by applying a linear regression over the $CPMR^{c(k,l)}$ estimates between cohorts 1940 and 1960. The cohorts with the largest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

Figure 2 shows 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, males had higher levels of mortality within each country. Mortality was considerably higher in the United States than in Canada (~40% and ~70% higher for females and males, respectively), to the point that the mortality levels of U.S. females and Canadian males were quite similar for the cohorts born at the end of the 1950s. Concerning mortality changes in absolute terms, whereas U.S. male and female $CPMR^{C(35,54)}$ levels deteriorated, for Canada the mortality improvement stagnated without deterioration.

Figure 2 indicates the cohorts that reached the largest negative and positive deviances from the $CPMR^{c(35,54)}$ linear trend, defining the *advantaged* (circles) and *disadvantaged* (triangles) cohorts,

respectively. Whereas the advantaged cohorts are located in proximate birth cohorts for all subpopulations (between 1947 and 1949), the disadvantaged cohort of U.S. males was born considerably earlier (1952) than those of the other groups (between 1957 and 1960). The selection of the disadvantaged cohorts for Canadian males and U.S. females is not as evident as in the other cases. To test the consistency of our estimates we made sensitivity tests changing the location of disadvantaged cohorts and the cohort interval under observation. These estimates are presented in the Supplemental Material (Figures S3 to S5).

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 trend (in dashed lines). The points and labels indicate the year of birth of the *advantaged* (circles) and *disadvantaged* (triangles) cohorts.

Decomposition of cohort's excess mortality by cause of death

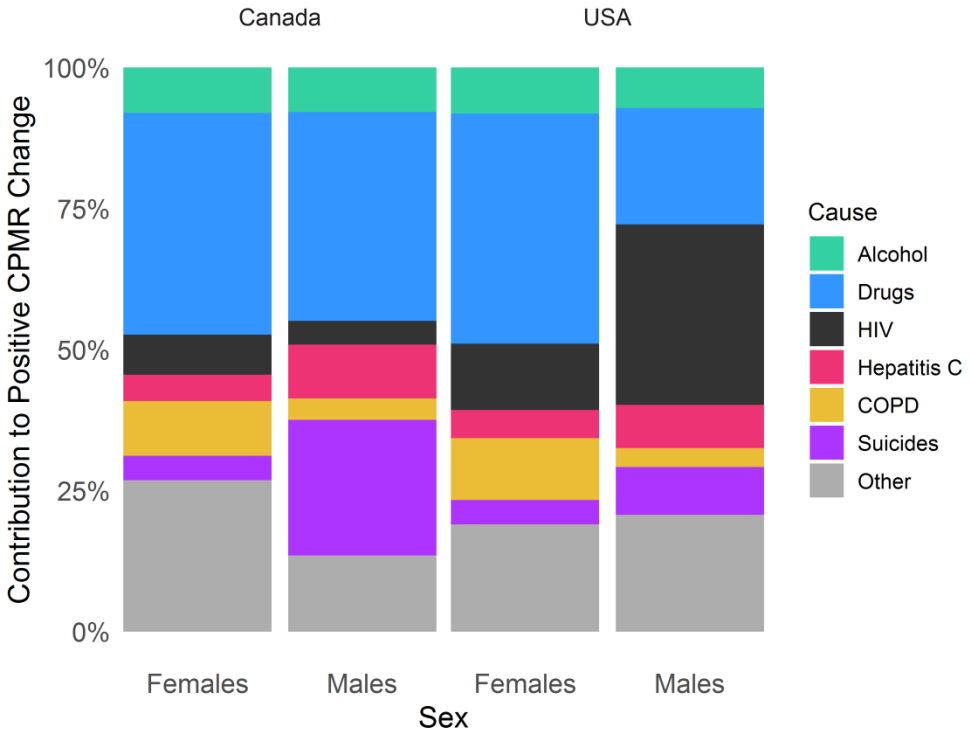
Once more precise locations of the *advantaged* and the *disadvantaged* cohorts are identified using the $CPMR^{c(35,54)}$, we proceeded to decompose the mortality change between the two cohorts

($\Delta CPMR^{d-a(35,54)}$) by causes of death. See the Supplementary Material for additional information about this decomposition.

According to the decomposition of $\Delta CPMR^{d-a(35,54)}$ in broad categories of cause of death, the largest contributions to the mortality deterioration 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. Among these causes, external and infectious diseases had considerably larger contributions in all subpopulations (see Figure S1 in the supplemental material).

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

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



Magnitude and temporal dynamic of the boomer cohorts' disadvantage

Detrended cohort effects on mortality by cause of death

Estimates from the $\Delta CPMR_i^{d-a(35,54)}$ depicted in Figure 3 were useful to identify the leading causes of death contributing to the relative mortality deterioration between the *advantaged* and the *disadvantaged* cohorts. However, to properly assess the cohort penalty by cause of death, we need to account simultaneously for variations over the three APC dimensions (A further discussion on this topic is presented in the Supplementary Material). Yet, the use of APC models is contentious given the well-known *identification problem*, in which the perfect multicollinearity between the variables (cohort = period – age) results in an infinite number of solutions with identical fit. Several attempts have been proposed to solve the model by imposing arbitrary restrictions (e.g., constrained

generalized linear (Fienberg and Mason 1985) and intrinsic estimator (Yang et al. 2004) models), not without strong criticism (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 into the age, period, and cohort components (Rodgers 1982). In the present study we are not interested in decomposing the linear effects, but instead in analyzing the deviance of the boomer cohorts' mortality from the linear trend. These divergences from the linear trend –also referred in the APC literature as nonlinear effects, curvatures (Holford 1983), or humps (Chauvel et al. 2016; Remund et al. 2018), are unaffected by the constraints chosen for the model identification and, as a result, are 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 risks of mortality over 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, resulting in a series of cohort components with zero-slope. Under this parameterization, the logarithm of the cohort effects are interpretable 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 in two-year categories, and fitted splines to a Poisson model, using the R package Epi (Carstensen et al. 2018).

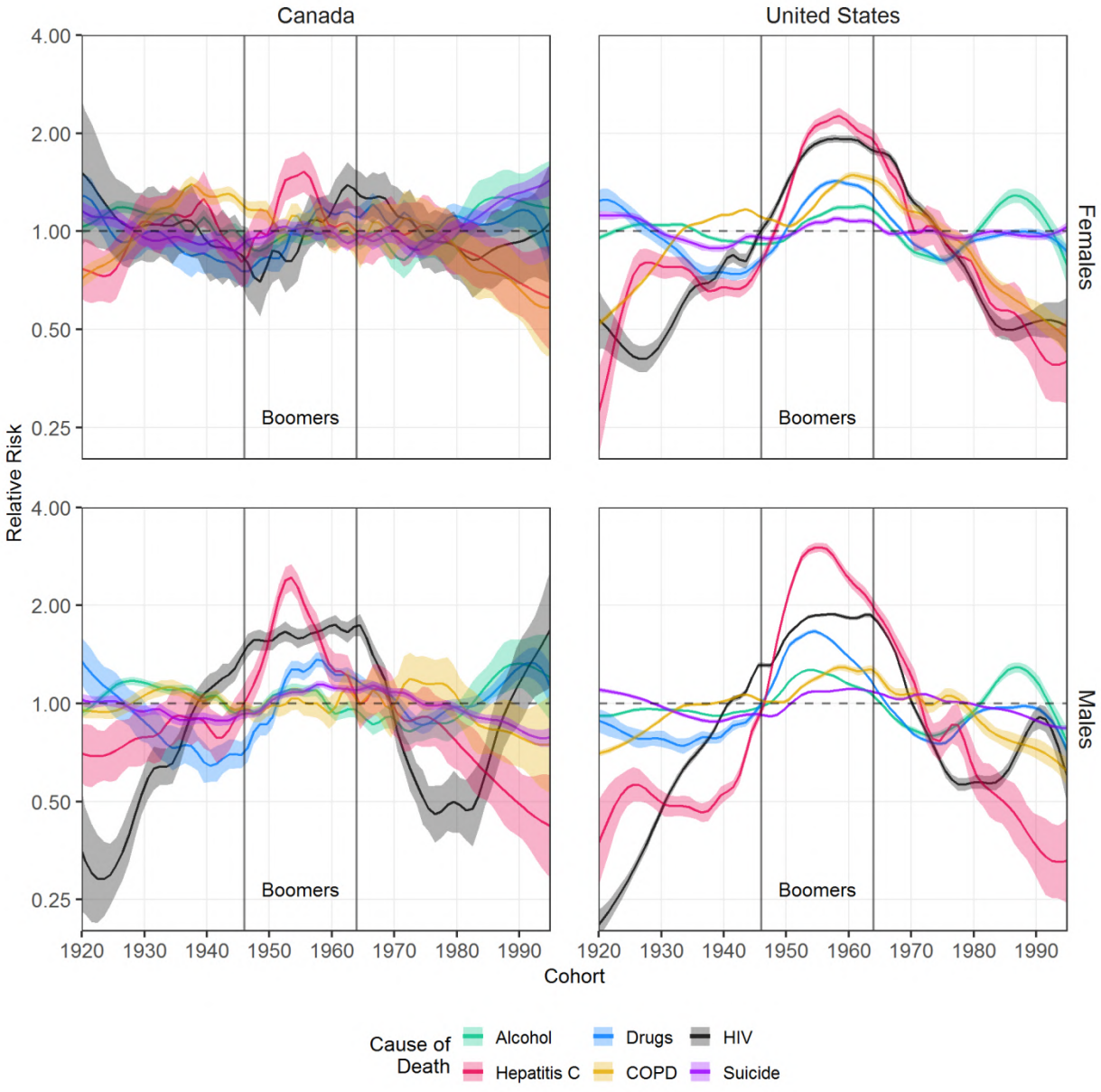
Comparisons between Canada and the United States

Figure 4 shows the relative risks and confidence intervals obtained from the APCd model by country, sex, and cause of death (see Figure S6 in the supplemental material for a facetted version of Figure 4). In all boomers 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 lowest were for alcohol abuse, suicide, and COPD (with relative risks between 1.2 and 1.5). The main difference between the two countries is the pattern of mortality due to COPD, for which only the U.S. boomers have a larger risk, compared to the overall cohort average.

Canadian females' mortality is considerably different from their male counterparts and from U.S. males and females. For all causes of death, the Canadian female boomers had a negligible disadvantage, if any at all. Regarding alcohol, suicide, and COPD, they did not have significantly larger risks compared to the overall cohort average. In contrast, boomer males in Canada and of

both sexes in the United States have similar relative risks and cohorts for which this relative risk is maximal for hepatitis C, HIV/AIDS, drugs, and suicide. More precisely, these three subpopulations of boomers had the largest disadvantage in mortality from drugs, HIV/AIDS, and hepatitis C, a moderate for alcohol, and considerable lower for suicides.

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 ending of the baby boom (i.e., 1946 and 1964, respectively) are marked with vertical grey bars.

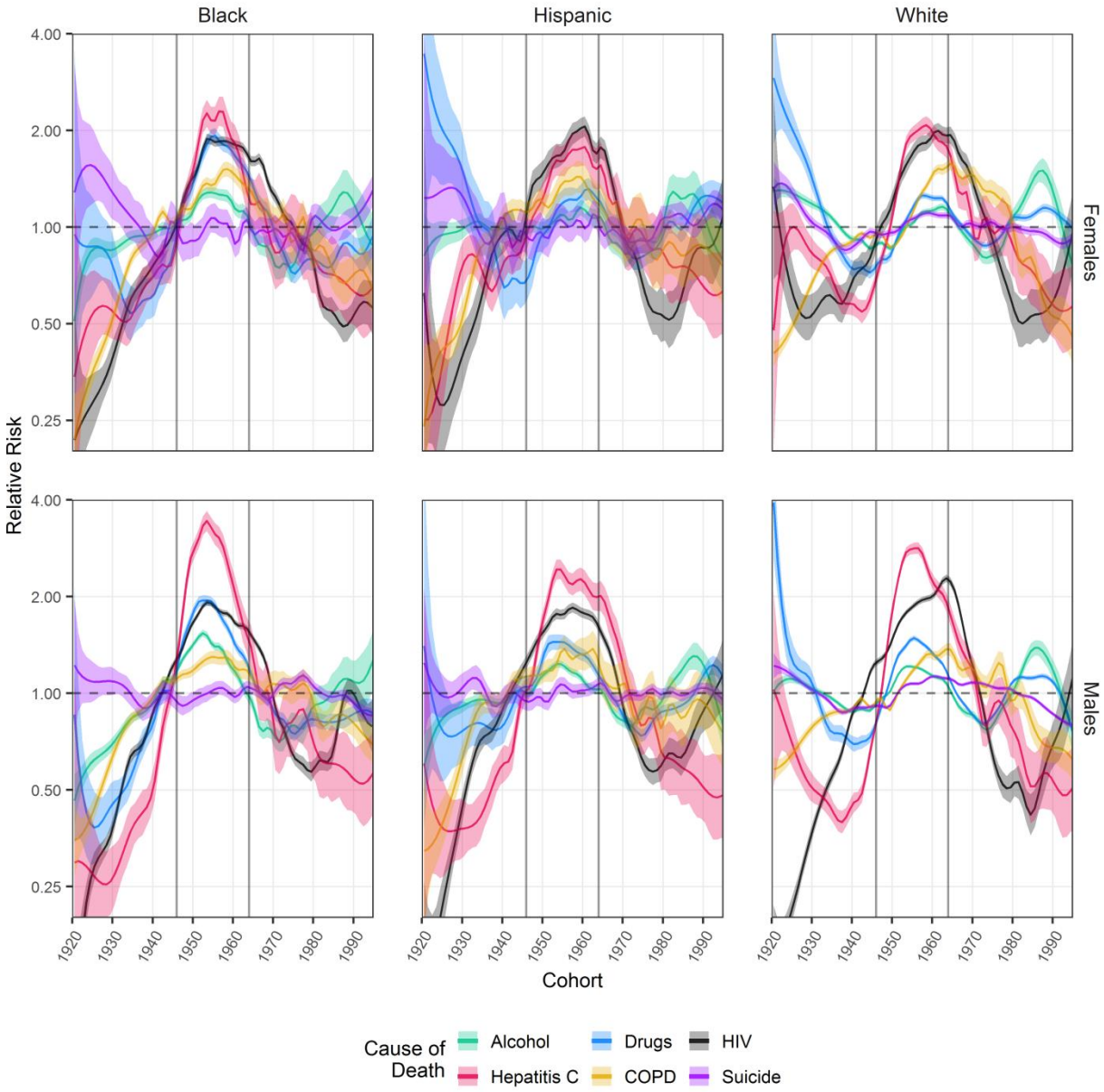
Comparison across race and ethnicity within the United States

In order to explore the disparities across racial and ethnic groups within the United States, we estimated detrended cohort effects as cohort relative risks for NHB, Hispanic, and NHW (Figure 5; see also separate plots in Figure S7 in the supplemental material). Except for a few cases, female and male boomers in the United States have similar cause-specific cohort disadvantages, regardless of race and ethnicity. The causes that contributed the most to the boomers' disadvantage in all subpopulations were hepatitis C and HIV/AIDS, and the lowest contribution was made by suicide.

The main difference between sexes concerns the magnitude of the boomer disadvantage for hepatitis C, which was considerably larger for males (reaching a maximum of 2.5-fold risk on average) than for females (2.1-fold risk on average), regardless of race/ethnicity.

When analyzing the cause-specific contribution to the boomers' disadvantage by race and ethnicity, two results stand out. First, NHW boomers from both sexes were the only ones with significantly higher relative risks of suicide mortality. Second, although boomers from all groups had a large drug-related mortality disadvantage, the relative risks for NHB (1.9 and 2.0 for females and males, respectively) were substantially larger compared to those of 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 ending of the baby boom (i.e., 1946 and 1964, respectively) are marked with vertical grey bars.

In summary, these results show that, with few exceptions, boomer cohorts had higher relative risks of mortality in most of the leading causes contributing to mortality deterioration between the advantaged and disadvantaged cohorts, regardless of the national context, sex, and race/ethnicity.

Yet, three exceptions are noteworthy. First, among Canadian females, relative risks of mortality were significantly higher for boomers only for drugs, HIV, and hepatitis C. Second, boomers' disadvantage in COPD mortality was only found significant for the U.S. Third, only Canadian males and U.S. NHW from both sexes showed a significant disadvantage for suicides. In the next section we turn to analyze the temporal dynamic of these cause-specific cohort disadvantages.

Dynamic of the cohort excess mortality over time

The APCd estimates presented above are useful for the estimation of the average of cohort disadvantage. However, they are limited for the analysis of the temporal dynamic of nonlinear effects because, as averages, they do not allow us to observe variations in the relative risk over time (Chauvel 2013). The changes in magnitude or location of the curvatures by cause over time, help us to identify whether the cohort disadvantage results from a sequence of temporary age-period interactions or, to the contrary, from a process that operate 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 enables 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 where the relative risk reaches a maximum. These plots help providing synthetic information on nonlinear APC effects simultaneously across several populations or for several causes of death.

To construct the *APC-curvature plots*, we need 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 is estimated 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). Then, the measured excess mortality is translated into visual attributes: the locations of the ridge over time (i.e., the age/cohort for which the positive divergence in mortality reaches its maximum level in each period) are indicated through the coordinates position in the Lexis diagram; and the magnitude (i.e., the relative risk in the ridge compared to the base of the hump) is 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

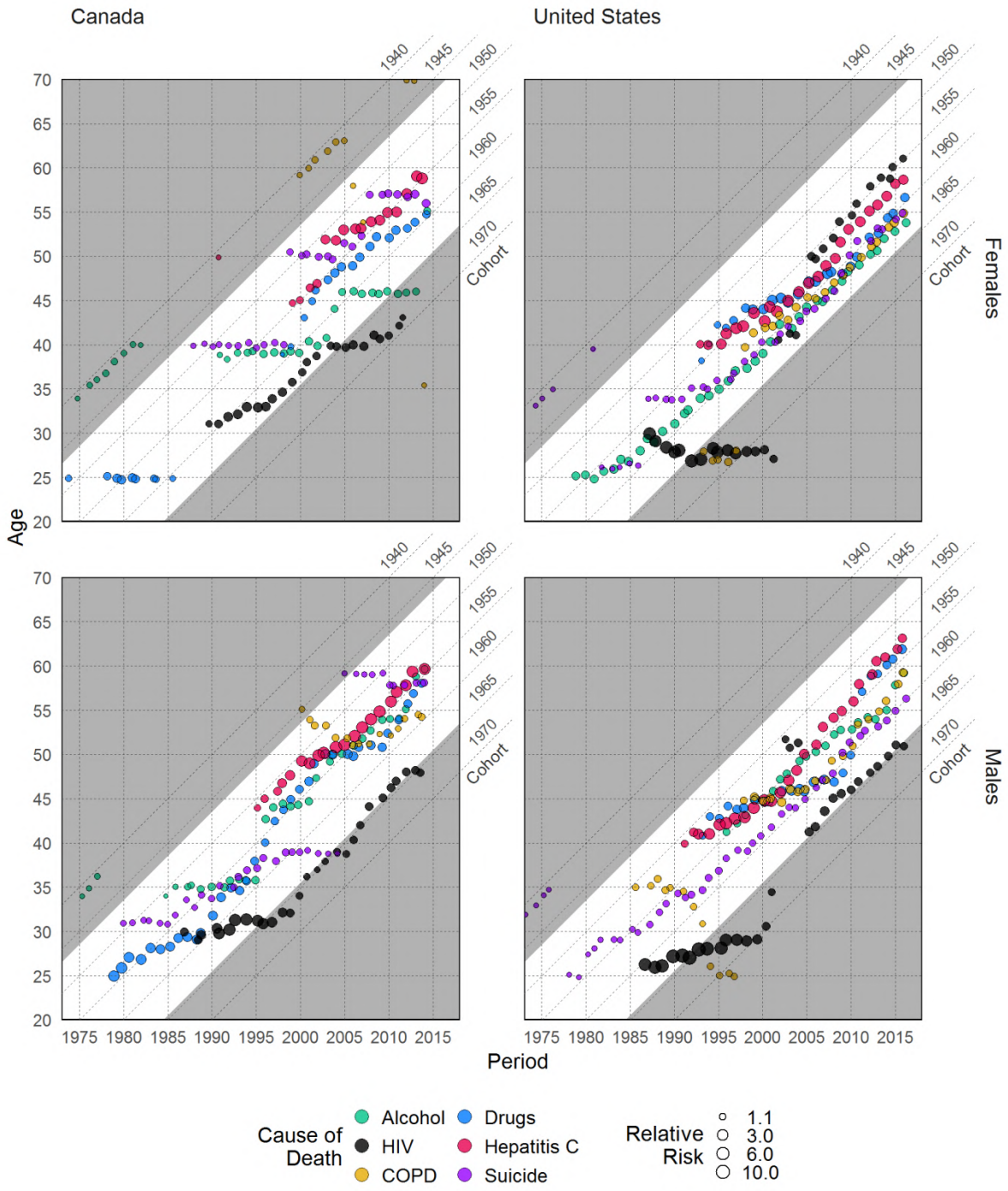
APC curvature plots presented in Figure 6 show the temporal dynamic of excess mortality (i.e., the age/cohort with the greatest excess mortality in each period) by cause of death, country, and sex (see also Figure S8 in the supplementary material). We first describe the results for HIV/AIDS-related mortality. The patterns for HIV/AIDS are similar across subpopulations, but considerably different from the other causes. Between the mid-1980s and the mid-1990s the ridge of the HIV/AIDS excess mortality largely surface in the diagram as an age effect, for both sexes in Canada and the United States, disproportionally targeting young-adults (25-35y). However, since 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 had the largest susceptibility, for the U.S. females, the largest disadvantage was observed for those born around 1955. For Canadian females this cohort effect was interrupted in the second part of the 2000s, shifting once again to an age effect, but this time targeting those aged ~40.

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

Turning now to the variations in magnitude of the cohort disadvantage over time (see also Figure S8 in the supplemental material), the relative risks of HIV/AIDS mortality were considerably larger at the turn of the 1990s for all groups (up to 10.7), and considerably larger for U.S. males. Nevertheless, at the turn of the century, the HIV/AIDS disadvantage decreased sharply, with a relative risk below 1.8 during the rest of the period. Apart from HIV/AIDS, the largest boomer disadvantages were observed in mortality from hepatitis C and drug abuse, which respectively hit the maximum relative risks of 4.9 and 3 at the turn of the century. The boomer relative risks for alcohol,

COPD, and suicide mortality were substantially lower, compared to the other causes, without exceeding 1.7, 1.7, and 1.5, respectively.

Figure 6. APC curvature plots by country, sex, and cause of death



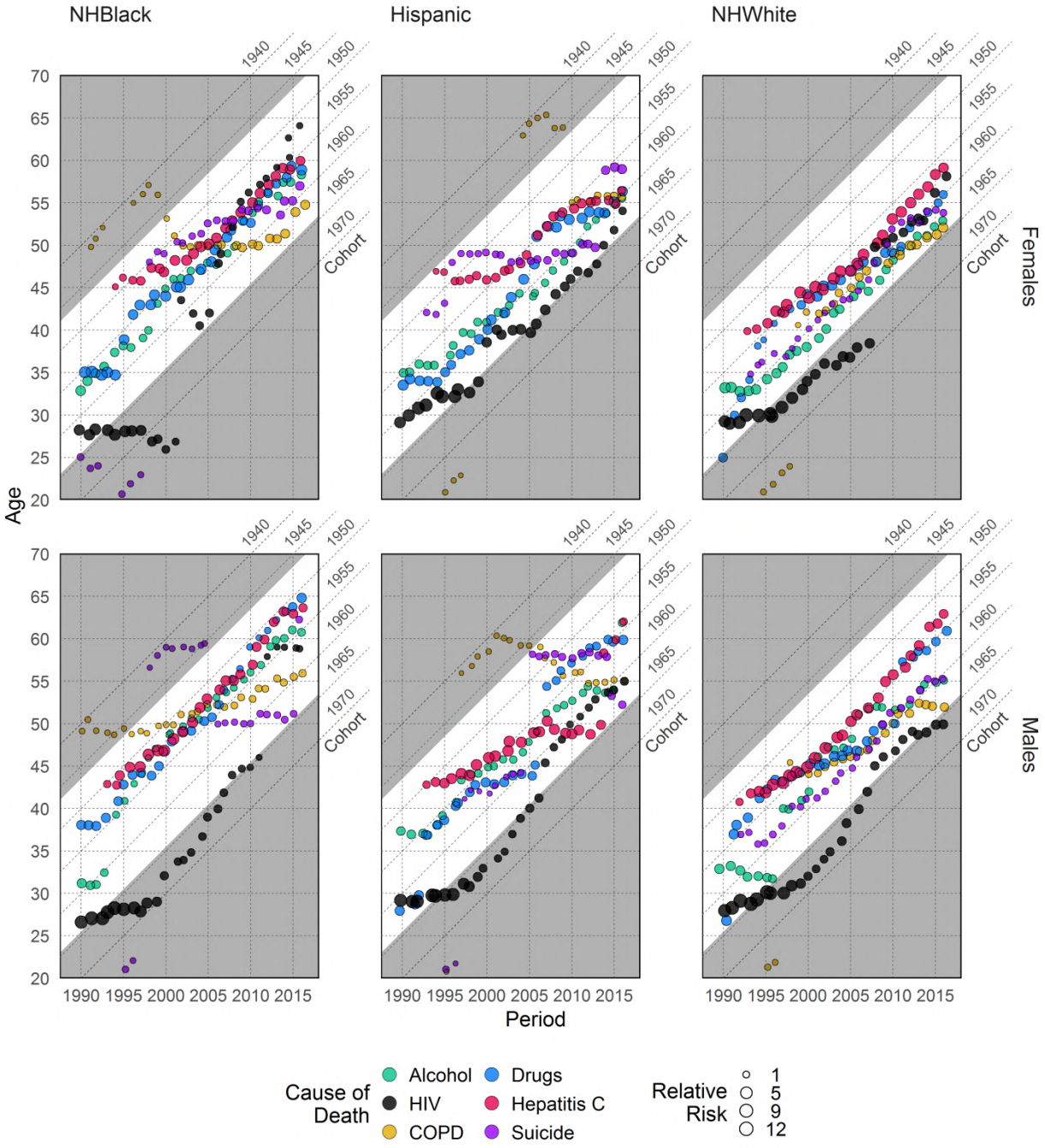
Notes: Location (age/cohort) and magnitude (in relative risks) of the largest excess mortality in each period. The color of the points indicates the cause of death and the size indicates the relative risk at the ridge, compared with the mortality baseline. The white diagonal band indicates the location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

Comparison across race and ethnicity 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 S9 in the supplementary material). According to the trends depicted in Figure 7, except from COPD and Suicide mortality, boomers from both sexes and all race/ethnicities had clear and sustained cohort disadvantages in all causes. Systematic cohort disadvantages in COPD and suicide were only identified for NHW. For the other ethnic/racial groups, the ridges for these causes were rather 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 boomer relative risk of HIV/AIDS mortality decreased considerably and shifted into a sustained cohort effect. Also consistent to the national level, U.S. boomers from all race/ethnicities showed the largest relative risks for hepatitis C and drug abuse mortality, reaching a maximum level of 4.7 and 3.8, respectively. The lowest relative disadvantage among boomers was observed for mortality from alcohol, COPD, and suicides (2.8, 2, and 1.9, respectively).

Turning now to the variations of relative risks over time, for most race/ethnicities the relative risks of hepatitis C peaked at the turn of the century, and then decreased afterwards. For Hispanic and NHB females, however, the relative risk of hepatitis C did not started to decrease until 2010. The ridge of drug-related mortality disadvantage peaked three times: first at the turn of the 1990s, then at the turn of the 2000s, and lastly during in the last year of observation (i.e., 2016). The relative risk of alcohol mortality was large in 1990 (2.1- to 2.8) and progressively decreased over time for Hispanics and NHW. For NHB female and male boomers, however, this declining trend was reversed in 2010, as the relative risk of alcohol mortality increased markedly. For COPD and suicide, the boomer relative risks 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 race/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 "escorted" boomers throughout their life course, regardless of country, sex and race/ethnicity. Of notice, however, is the absence of a cohort disadvantage for the Canadian female boomers in several causes.

Discussion

This study was designed to identify the causes of death that contributed the most to the boomer disadvantage in mortality, and to explore the temporal dynamics of these contributions, taking into account differences by country, sex, and race/ethnicity. Whereas Canadian boomers experienced a deceleration in mortality improvements with respect to previous cohorts, 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, suicides, and COPD, contributed to at least 75% of positive changes in mortality –i.e., mortality deterioration– across cohorts. The observed boomer disadvantages were substantially larger in males than in females for most causes, regardless of national context or race/ethnicity. The largest relative risks were identified in mortality from hepatitis C and drug-related causes and the lowest in COPD and suicide mortality. These results should be interpreted with caution, considering that larger relative risks do not necessarily indicate greater excess mortality in absolute terms. For instance, despite boomers had considerably larger relative risks of mortality from hepatitis C than from alcohol, the mortality baseline of the latter is substantially larger, contributing with greater mortality rates to the boomer disadvantage.

On the question of the temporal dynamic of the boomers' excess mortality by cause, our findings revealed that most operated as long-term and sustained cohort disadvantages throughout the boomers' young and adult life. 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 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 dynamic of the cause-specific contribution to the boomer penalty, as well as the potential 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 pointing out the advantages and limitations of the analysis and further investigations that may follow on the current topic.

Temporal dynamic of the cause-specific excess mortality among boomers

The distinction between age-period interaction and cohort effects is relevant, since several of the leading causes contributing to the cohort disadvantage in mortality were also responsible for important period crises during the observed period. Such was the case of 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 rather from successive period crises that targeted the boomers at different life stages –that is, a sequence of age-period interaction effects that disproportionally 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 early-1990s), which disproportionally affected those in their 30s. 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 that the late boomers carried out lingering effects throughout their later adult life (Figures 6, 7, S5, and S6). This higher susceptibility among boomers toward HIV/AIDS may be the reason for the recent increases in HIV prevalence among people aged 50 and older, accounting for nearly the half of those living with diagnosed HIV in the United States (CDC 2018a).

In the case of drug overdoses, despite the fact that absolute changes in severity were largely driven by period-based factors that affect most ages, our findings demonstrated that the boomer cohorts had larger risks of drug-related mortality during the whole observation period, compared to neighboring cohorts. The magnitude of this cohort susceptibility, however, was not constant over time (Figures 6, 7, S8, and S9). The boomer disadvantage for drug-related mortality peaked twice: at the turn of the 1990s and at the turn of the 2000s, corresponding to the beginning of the crack and

opioid epidemics, respectively. These increases in relative risks at the beginning of each crisis and their posterior attenuation may result from the vanguard role of boomers in drug abuse, before the epidemics spread to other cohorts. This inter-cohort "contagion" of substance abuse is particularly notable for the case of the millennial cohorts. After the boomer cohorts had the largest relative risk of alcohol and drug related mortality during the 1990s and 2000s, the millennial cohorts began to experience relative increases in mortality from these causes, to the point of surpassing the risk of boomers in recent years (Huang et al. 2017; Miech et al. 2013; Sauer et al. 2018; Zang et al. 2019). In both HIV/AIDS and drug mortality, despite punctual and strong period crises affecting most age groups (i.e., period effects), lagged effects from these disturbances along the cohorts were identified for the boomers (i.e., cohort effects).

The similar location in the most disadvantaged cohorts across causes of death (see Figures 6 and 7) suggests that common mechanisms underlie behind multiple outcomes. Our results are consistent with previous findings of the interrelation between several behavioral risks. This is the case of the opioid abuse, HIV/AIDS, and hepatitis C. In most groups, the cohorts with the largest disadvantages in these causes had similar locations and synchronic variations in magnitude since the mid-2000s. part of these similarities may result from the ongoing opioid epidemic, which might have contributed to spread HIV/AIDS and hepatitis C infections among the chronic intravenous drug users (IVDU) (Strader 2005; Zibbell et al. 2017). In addition, not only IVDUs have higher risks of HIV infection by sharing contaminated needles, but diagnosed HIV patients have both higher rates of prescription of opioids to treat chronic pain symptoms and higher risks of developing drug use disorders (Becker et al. 2016). Similarly, the synchronicity between COPD and drug abuse mortality among NHB females and NHW of both sexes (Figure 7) was consistent with previous findings suggesting a higher risk of COPD mortality among opioid users (Levine 2017; Vozoris et al. 2016). However, such interrelation was not supported by the results of the other groups, which did not display such synchronicity.

Taken together, our findings suggest that the mortality penalty endured by the boomers is not the result of 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 escorted the cohorts over time. In a nutshell: concurrent long-term scars, not a succession of temporary bruises. These disadvantages are manifest for several distinct, but

interrelated causes of death that point toward a unique set of attributes related to riskier behaviors and attitudes, as discussed next.

Factors contributing to the boomer penalty

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

According to the *frailty hypothesis* (James W. 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 susceptible to mortality from *intrinsic* causes of death. It has been stated that the higher mortality experienced by the boomers during young and adult ages would be the consequence of increased survival rates early in life from 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 rather behavioral or extrinsic and it is unclear how frail individuals that were "saved" in early life through better nutrition and reduced infection loads would develop risky behaviors later in life. 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; Ryder (1965)'s *birth cohort* influence and the Mannheim (1952)'s historical *generational*[1] membership influence.

In his seminal work, Ryder (1965) stated that some birth cohort's characteristics have permanent effects through the life course. Cohort size was for Ryder the most evident manifestation of inter-

[1] 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 social sciences.

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 implication of cohort size and other cohort-specific characteristics with the historical location of the boomers during their life course (Easterlin 1976, 1987; Easterlin et al. 1993). Easterlin's central argument was focused on the mismatch between early life and adult life conditions that the boomers notoriously 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 contrasted 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. Under the Easterlin perspective, this imbalance between expectations and reality had serious implications for the perceived wellbeing of the boomers, increasing the prevalence of mental distress and frustration among these cohorts. Easterlin (1987) expected baby boomers to experience a higher mortality from suicide, substance abuse, vehicle accidents, and homicides.

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 at sensitive life stages of the boomers consolidated a distinctive generational identity. The social importance and the inclusion of the 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, 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 have more influence from the formative experiences and peer influences during the early stages of life than from successive period-based influences throughout the life course (Johnson and Gerstein 2000;

Keyes et al. 2011; Rhodes 1997). In this sense, the differential construction of risk perception and habituation during the early stages of boomers life 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; R. Rao and Roche 2017), and from road traffic accidents (Puac-Polanco et al. 2016; T. Rao 2019).

Although referring to different phenomena, *birth cohort* and *generational identity* are not completely independent. The increased exposure among peers might 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 social youth-based movements of the 1960s and 1970s (Abrams 1970; Bristow 2015, 2016; Cross and Kleinhesselink 1985; Goertzel 1972).

It may be the case, therefore, that the cohort attributes of boomers not only reinforced their rebellious and risky generational identity, but both mechanisms may have acted in parallel increasing the mortality risk along the life course. Let us now turn to discuss, in light of the mechanisms presented above, some of the similarities and differences in magnitude and temporal dynamic of the boomer penalty across countries, sexes, and race/ethnicities.

Comparison of findings across population groups under study

The unprecedented transnational diffusion of the boomer identity discussed earlier, as well as some similarities in the socioeconomic context in Canada and the United States during the sensitive life stages of boomers, could explain to some extent our findings. We identified that the leading causes behind the boomer excess mortality were the same in both countries, and the temporal patterns of the cohort effects were quite similar.

Yet, there are remarkable differences in the magnitude of the boomer penalty between the two countries. As shown in Figures 1 and 2, while Canadian boomers mainly experienced a slow-down in mortality improvements (indicated by the green diagonal trace in Figure 1), the U.S. boomers had a noticeable deterioration of mortality (indicated by the change of scale sign, from negative to positive change of mortality in Figure 1).

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

Second, the generational rift experienced by Canadian boomers may have been less striking than for their counterparts in the United States. The dominant values in the Canadian society –such as the rights of women, racial/ethnic minorities, and no-heterosexual communities, as well as the attitudes regarding religion, sexuality, and drug use– were also challenged by the counterculture movements (Palmer 2008). However, the magnitude of the political and social conflicts in Canada during the 1960s and 1970s did not have the same scale as those in the United States (Campbell et al. 2012). At that time, Canada neither had Jim Crow laws nor did participate in the Vietnam War. On the contrary, historically, Canada has been perceived and served as an alternative for the U.S. population to flee from racism and draft. Such was the case of the NHB escaping from slavery through the Underground Railroad during the 19th century, and of the draft-dodgers –mostly young boomers– avoiding the Vietnam War during the 1960s. Previous research has established that the civil rights and the antiwar movements were the most important causes for the generational rift, and thus, the most influential and cohesive events around the counterculture movements during the 1960s and 1970s in the United States (Alwin et al. 2014; Alwin and McCammon 2007; Bristow 2015; Stewart and Torges 2014). Therefore, Canadian boomers may have experienced milder birth cohort effects translated into lower levels of anxiety, and adopted a less rebellious generational identity that enacted less risky attitudes and behaviors. Such differences between both countries might have contributed to dampening the boomer penalty among Canadians.

With respect to differences by sex, there is a large amount of evidence pointing to a higher propensity among males to engage in risk taking behaviors that could lead to death, compared to

females (Ferrence 1988; Fingerhut and Cox 1998; Harris and Jenkins 2006; Pampel 2001; Veevers and Gee 1986; Waldron et al. 2005). Since most of the boomer excess mortality stem from behavioral causes, it is not surprising that the boomer penalty is larger among males in all groups under observation.

Regarding the temporal patterns, two male-female differences stand out. First, ridges of the excess by cause of death show horizontal, rather than diagonal traces for Canadian females. The contrasts regarding the welfare state policies between Canada and the United States discussed earlier could explain this lower boomer penalty among Canadian females. There is evidence that the welfare state and social policies buffer to some extent the social gender inequalities (Karamessini and Rubery 2013; Kushi and McManus 2018; Rubery 2012). Public policies supporting single and married mothers, as well as incentivizing the inclusion, the stability, and the reentrance of females into the labor market could have diminished the burden among Canadian female boomers, relative to the experienced by their U.S. counterparts. Nevertheless, there is still evidence of a boomer penalty in mortality for Canadians female boomers (see Figures 1, 2, and 4) involving the same causes of death than for 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 low magnitude.

A second male-female difference is the location of the most disadvantaged cohorts. As seen in Figures 1, 6, and 7, the cohorts with the largest excess mortality for female boomers are located in more recent cohorts, compared to those of males. This difference in location could reflect the age differences within couples, which was 2.4 years in average for married couples between 1960 and 1985 (USCB 2018). Since males are more prone to riskier behaviors and may had more success in influencing couple choices (de Palma et al. 2011), the negative outcomes in boomer couples may affect females from more recent cohorts, compared to males.

Let us now turn to discuss the differences across races and ethnicities within the United States. Interestingly, despite the significant differences of the sociohistorical contexts lived across racial and ethnic groups in the United States, we found similar patterns of sustained boomer disadvantages among them. This finding is somewhat surprising under the birth cohort effect proposed by Easterlin. When describing the large mismatch between high expectations in childhood and the harsher reality encountered in adulthood, Easterlin described 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 from the economic boom and social policies implemented during the post-war, which were embodied as oversized expectations among boomer children. The unprecedented numbers of admissions to the universities and the massive mortgage loans offered during the postwar period were selectively addressed to the young NHW population –the parents of the NHW boomers. For racial/ethnic minorities, however, the residential (Luders-Manuel 2017; Massey and Denton 1993; Rothstein 2017; Sharp and Hall 2014; Steil et al. 2018) and educational (Herbold 1994; Humes 2006; Turner and Bound 2002) segregations, among other discriminatory policies, hindered their access to the social and economic benefits of that period.

By contrast, the generational rift lived by boomer cohorts, although experienced and embraced 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 resulted involved in some of 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 is expected that the effects from the boomer generational identities had impacts on wider segments of the U.S. population, regardless of socioeconomic status, and race/ethnicity, compared to the birth cohort effects proposed by Easterlin.

This difference across race/ethnicities in the influences of the birth cohort and the generational identity mechanisms could be the reason behind the disparity in the temporal dynamic of suicides ridges. NHW boomers from both sexes are the only groups within the United States that show sustained cohort disadvantages in suicide mortality. Whereas suicide mortality could be more associated with the large prevalence of stress and frustration, resulting from the birth cohort effect proposed by Easterlin (Chauvel et al. 2016; Easterlin 1987; Phillips 2014), higher susceptibility to mortality from substance abuse, HIV/AIDS, and hepatitis C may be more responsive to the risky attitudes and behaviors embodied in the boomer generational identities (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 opioid crises have been respectively associated with NHB and NHW populations in scientific literature and media coverage. It is possible that the stigma, and the lower price 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 the racial discrimination in the prescription practice (Barnett et al. 2017; Hwang et al. 2015; Jones et al. 2018; N. B. King et al. 2014; Manchikanti et al. 2017; Quinones and Hellegers 2016; Zang et al. 2019), made the NHW blue-collar population at higher risk during the beginning 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 when at certain periods affected disproportionally specific social status or racial/ethnic groups (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 boomer penalty in Canada and the United States, as well as to point out some similarities and disparities by country, sex, and race/ethnicity. For this, we adopted a more comprehensive approach to analyze the boomers' excess mortality as a whole, compared to most research on the topic. Instead of exploring a single cause of death, as most of the previous studies about boomers' mortality, our strategy began by the common boomer penalty in all-cause mortality in Canada and the United States, to subsequently decompose it by cause of death. The decomposition of the mortality change 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 boomers' excess mortality in Canada and the United States.

The analysis of the temporal dynamic of nonlinear cohort effects using *APC curvature plots* permitted us to identify that the boomer disadvantage in mortality resulted from multiple and simultaneous cause-specific cohort effects, instead of a sequence of age-period interaction effects. As discussed earlier, this finding would not have been possible if we had limited our analytical strategy to the more conventional APC statistical models. In this way, we were able to identify the concurrence of multiple disadvantages for the boomer cohorts that may be interrelated and modulated by common mechanisms, independent of national context, sex, and race/ethnicity. These findings offer additional clues to analyze causal mechanisms that are not exclusively related to one output, but instead enacted simultaneous and sustained disadvantages throughout the life course of the boomers.

To our knowledge, this is the first analysis reporting concurrent and sustained cohort effects on Canadian and U.S. boomers. Such findings provide valuable information to the design of health policies. Currently, the populations from these countries are amid health crises involving the leading causes behind the boomers' excess mortality. That is the case of the dramatic increases in mortality

from drug overdoses (Helmerhorst et al. 2017; Ho 2017; Huang et al. 2017; Jalal et al. 2018), alcohol (CIHI 2018; Tapper and Parikh 2018), and suicides (Curtin et al. 2016), increases in binge drinking (Bulloch et al. 2016; Dwyer-Lindgren et al. 2015; Manthey et al. 2019), and hepatitis C infection incidence (CDC 2018b; PHAC 2019; Zibbell et al. 2017), as well as the stall in the long-term decrease in deaths from of HIV/AIDS (CDC 2019). Our research suggests that the policy-makers should encourage prevention and diagnosis not exclusively based on risk factors and age, as usually done, but also on birth cohort. Canadian and U.S. health authorities have already proposed such approach for the case of 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 suggest that such approach should be extended to other behavioral causes. The rest of this section moves on to consider the limitations of this study, and to propose some suggestions for further research.

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 in the death certificates. On the one hand, deaths from some of the causes under analysis may be misreported in the death certificates. HIV/AIDS and hepatitis C mortality began to be recognized within the ICD codes in 1987, long after actual deaths from these causes started to occur. It may have took some time for medical authorities to accurately recognize and record such causes on death certificates. Hence, underreport is expected in these two causes. In addition, the attribution of just one cause to each death hides multiple interactions, gaining even more importance when the causes are related to behavioral factors. Because risk behaviors tend to cluster (Ho 2017), several deaths classified in one specific cause had multiple contributing factors. Such is the case of mortality combining drug and alcohol abuse, or HIV and hepatitis C infections. For suicide mortality, not only the identification of the suicide intent is difficult to assess and may vary by geographical location, but also some causes are indistinguishable in classification when intentions and means are mutually exclusive, as it is the case of suicides by drug overdose. As seen in Table S2 in the supplemental material, we classified such deaths as suicides, which implied an underestimation of the drug-related death rates, and a potential source for the overestimation of suicides counts. This

bias in drug-related mortality could be magnified by other underreporting issues of 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 were introduced and did not exist in previous ICD revisions. Such is the case of several alcohol- and drug-related deaths. For instance, deaths from drug abuse were introduced in the 9th ICD version, and the poisoning from both drug and alcohol with undetermined intent were introduced in the 10th revision.

An additional potential source of error in data is related to the racial/ethnic classification within the United States. There have been identified discrepancies between the numerator and the denominator in the Hispanic ethnic classification. Whereas in the death certificates (i.e., the numerator) the ethnicity information is provided by funerary directors –which may have asked to deceased relatives or just imputed it 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 procedures of data collection may have underestimated the mortality of the Hispanic population. Besides, the lack of race and ethnic information in the Canadian death register limited our analyses about the heterogeneity of boomers within Canada and made it impossible to compare similar groups between both countries.

With regard to the method to analyze the temporal pattern of the disadvantage in mortality, there is more than one approach to extract the excess mortality (Acosta and van Raalte 2019). Because each approach is conceptually different, their application led to different estimations. Here, we used an interpolation approach because our interest was to analyze the excess mortality relative to the advantaged cohorts surrounding the boomers. If the question is about the deviance from the overall cohort average, the estimation of the residual from an Age-Period model would be more appropriate. It is noteworthy that the difference between both approaches concerns the magnitude of the excess but its temporal pattern over time is 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 generational identity effects on the boomer penalty in mortality. We proposed here such mechanisms as underlying determinants for the boomer disadvantage in mortality in a speculative

manner. Longitudinal data including measures about drug use, alcohol consumption, sexual behavior, mental health, mental stress, and/or expectations, among others, would enable such inquiry for an explanatory approach.

We believe that our research will serve as a base for future studies on the boomer excess mortality. We propose that further research should be undertaken in the following areas: First, future studies should target the test of the causal mechanisms proposed here as responsible for the boomer excess mortality, and measure to which extent each of these mechanisms has contributed to the boomer penalty. Second, an important question to resolve for future studies is the pattern of the cohort disadvantage among Canadian female boomers, for which sustained cohort effects were not identified, in contrast to Canadian male boomers and U.S. boomers for all race/ethnicities under analysis. Third, more work on analyzing the impact of the boomer disadvantage on changes in life expectancy and lifespan inequality, and on years of life lost would help identifying the implications of this boomer excess mortality at the population level and to for future trends of mortality. Fourth, it is not yet known whether strong generational differences are a common feature to all socioeconomic groups or they are disproportionately concentrated among the socially disadvantaged. It is vital to assess the contribution of different dimensions of the social position to the inter- and intra-cohort inequalities in mortality related to substance abuse and infections. And finally, the similarities between Canada and the United States regarding the boomer penalty 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).

Conclusions

We identified six causes of death related to risky behavior contributed approximately to 75% of the boomer excess mortality in Canada and the United States. We found evidence that most of the boomer excess mortality in Canada and the United States is driven by behavioral causes of death, namely mortality from drugs, alcohol, HIV/AIDS, hepatitis C, COPD, and suicides. Canadian female boomers were the only group in which the contributions of the identified leading causes did not operate as sustained cohort effects over time. Apart from this slight non-alignment, our findings are confirmation that the contribution of these behavioral causes to most of the boomer excess mortality in Canada and the United States is the consequence of multiple and simultaneous long-term sustained disadvantages that have escorted the boomers since their 20s.

The behavioral nature of the boomer excess mortality, and the sustained cause-specific effects throughout the young and adult life of boomers, highlight the pertinence of a more comprehensive and structural analysis of the boomer disadvantage in mortality. The fact that all the leading causes contributing to the boomer excess mortality are linked to behavioral risks suggests a set of common underlying mechanisms behind the boomer penalty in Canada and the United States. We propose that the more prevalent 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 this cohort disadvantage in mortality. Further analyses are needed to test the role played by these mechanisms in the boomer penalty on mortality, and the differences of these influences according to socioeconomic position.

If the cohort differences in mortality continue along the same trend, we predict the age group 65-75 to experience substantial increases in mortality in the upcoming years. This increase might be even greater than the previously experienced by the boomer cohorts because at these ages the mortality risks related to suicides and mental disorders are considerable larger. Moreover, as boomers age, they will be increasingly exposed to a higher prevalence of chronic pain, which has been a fundamental factor at the origin of the ongoing opioid abuse epidemic (Jones et al. 2018).

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., 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*, *Forthcomming*.

Adams, M. (2004). *Fire and Ice: The United States, Canada and the Myth of Converging Values* (First Printing 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 Soft-Cover Edition 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. (2018). *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. I: Age–period and age–cohort models. *Statistics in Medicine*, *6*(4), 449–467. https://doi.org/10.1002/sim.4780060405

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 Transactions*, *39*(2), 149-186–186.

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 (New York, N.Y.)*, *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: Critiques and New Directions. *Annual Review of Sociology*, *45*.

Goertzel, T. (1972). Generational Conflict and Social Change. *Youth and Society; Beverley Hills, Calif.*, *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 (Abingdon, England)*, *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 (Abingdon, England)*, *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 (Washington, D.C.)*, *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., & Hellegers, N. (2016). *Dreamland: The True Tale of America's Opiate Epidemic* (Unabridged edition.). Audible Studios on Brilliance Audio.

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

Supplementary Material

The Boomer Penalty: Excess Mortality among Baby Boomers in Canada and the United States

Measurement of mortality change by causes of death

Construction of Lexis Surfaces of mortality Change

Lexis surfaces of changes in mortality rates are widely recognized in the demographic literature as a powerful, yet simple method for the recognition of 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 rate of mortality change ($\Delta pc_{x,t}$) and then we plotted them in a Lexis surface. Additional information about the estimation of the relative change in mortality and the construction of the Lexis surfaces can be found in the Supplementary Material. 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:

$$\Delta pc_{x,t} = \log\left(m^s_{x,t}\right) - \log\left(m^s_{x,t-1}\right), \quad (1)$$

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

Then we plotted $\Delta pc_{x,t}$ values in a Lexis surface in two color scales to depict the yearly changes of mortality over period/cohort (Figure 1). The relative mortality decrease for year *t-1* compared to year *t* (or cohort *c-1* compared to cohort *c*) in the same age *x* is indicated in 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 of nonlinear cohort effects.

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

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

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

A similar index (*indice synthétique de mortalité*) was suggested by Termote (1998) as a complementary measure to analyze 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 appropriated 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

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

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*:

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

Contribution by causes of death

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 few groups of causes and guarantee a low 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 behavioural 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

ICD	8	9	10
Period	1968-1978	1979-1998	1999-2016
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 between the advantaged and disadvantaged cohorts. According to these results, most of the boomer excess mortality is composed by increases in deaths from causes within the ICD Chapters external, infectious, digestive, mental/behavioral, and respiratory diseases.

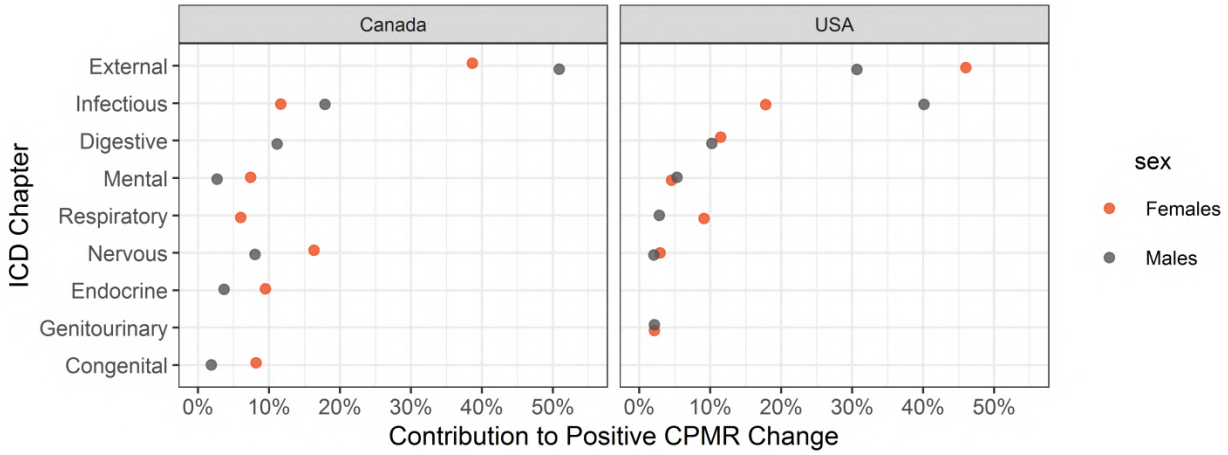


Figure S1 percentage of contribution of broad causes to the mortality deterioration between the *advantaged* and *disadvantaged* cohorts for both sexes in Canada and the United States

Based on the leading broad causes of mortality deterioration identified in Figure S1, we constructed more detailed causes of death and re-decomposed the mortality deterioration. Table S2 presents the ICD codes used to classify deaths from HIV/AIDS, hepatitis C, COPD, suicides, alcohol, and drugs. Note that each cause of death was not constrained within the same broad category –that is, within the same ICD chapter. For instance, alcohol-related mortality included deaths from mental and behavioral disorders due to alcohol (contained within the mental and behavioral disorders chapter), from alcoholic liver disease (within the digestive system chapter), and from poisoning with alcohol (within the external causes chapter).

Table S2: ICD codes included in each category of cause 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
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 contribution to the mortality deterioration by each cause of death. Increases in mortality from HIV/AIDS, hepatitis C, COPD, suicides, alcohol, and drugs contributed between 75% and 80% of the mortality deterioration between the advantaged and disadvantaged cohorts for both sexes in Canada and the United States.

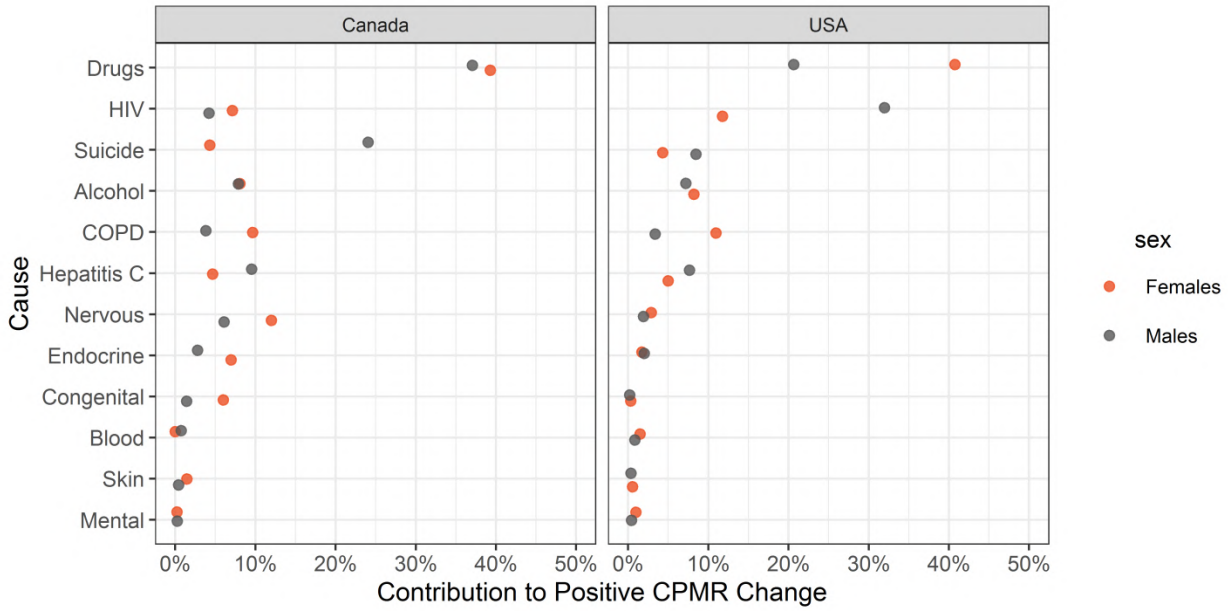


Figure S2. Percentage of contribution to the increase in $\Delta CPMR^{d-a(35,54)}$ by cause of death, for each sex and country. We only included the categories having at least 2% of positive contributions to $\Delta CPMR^{d-a(35,54)}$ in the four subpopulations

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 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 greatest positive deviance of mortality for U.S. females was reached by the cohort 1960 –which was the last cohort to be observed–, we were not able to verify whether the deterioration of mortality continued for more recent cohorts. To address these points, we test 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 newer cohorts for U.S. females. These estimations are presented in the supplemental material (see Figures S3 to S5).

The cause-specific contribution to the mortality deterioration between advantaged and disadvantaged cohorts is sensitive to the locations imposed to these cohorts. In order to test the consistency of the estimates presented in Figure 3, we chose alternative locations to the disadvantaged cohorts in the cases in which the largest divergence from the linear trend was not

obvious. For Canadian males, the deviation was pretty similar for cohorts 1954 and 1957. Hence, we use 1954 as alternative disadvantaged cohort. For U.S. females, the divergence did not stop to increase during 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 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.

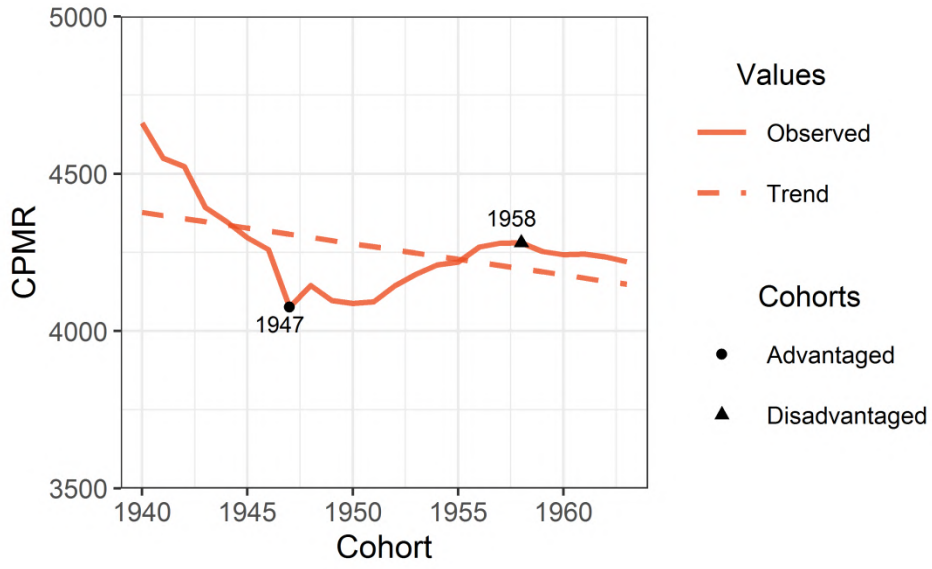


Figure S3. Cohorts' partial mortality rates (solid line) and the linear trend (dashed line) for U.S. females within the age interval 35-53 ($\mathrm{CPMR}^{c(35,53)}$), between cohorts 1940 and 1963. The labels indicate the year of birth of the *advantaged* (circular shape) and *disadvantaged* (triangular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

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

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

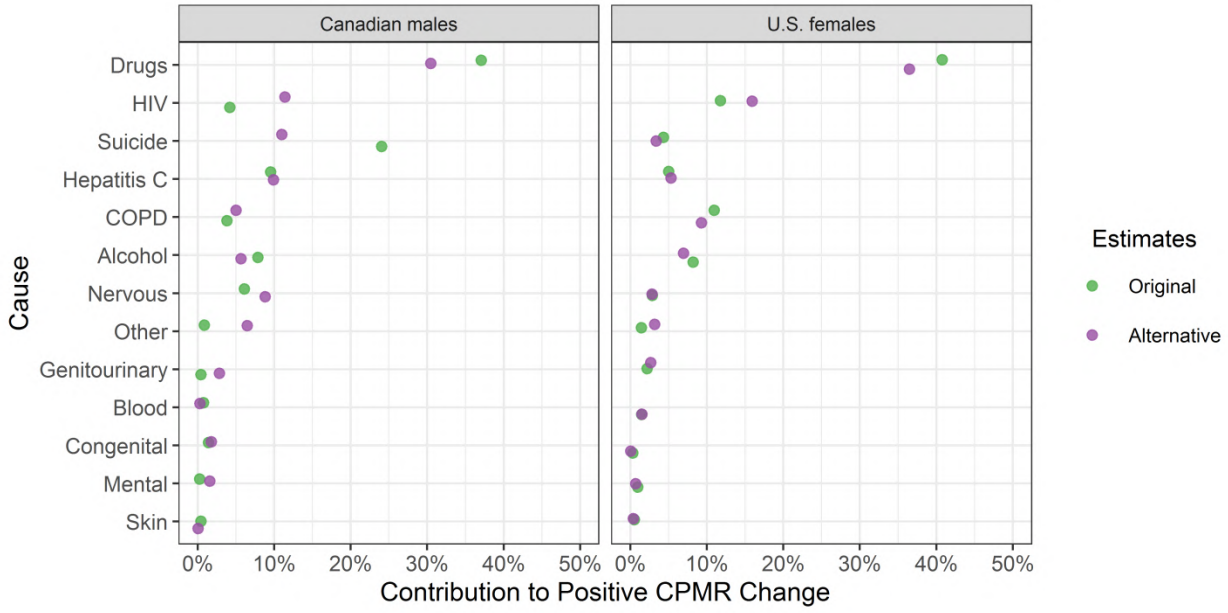


Figure S4. Percentage of contribution to the increase in $\Delta CPMR^{d-a(k,l)}$ by cause of death for Canadian males ($\Delta CPMR^{d-a(35,54)}$) and U.S. females ($\Delta CPMR^{d-a(35,53)}$), according to the original (respectively 1957 and 1960, in green) and the alternative (respectively 1954 and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

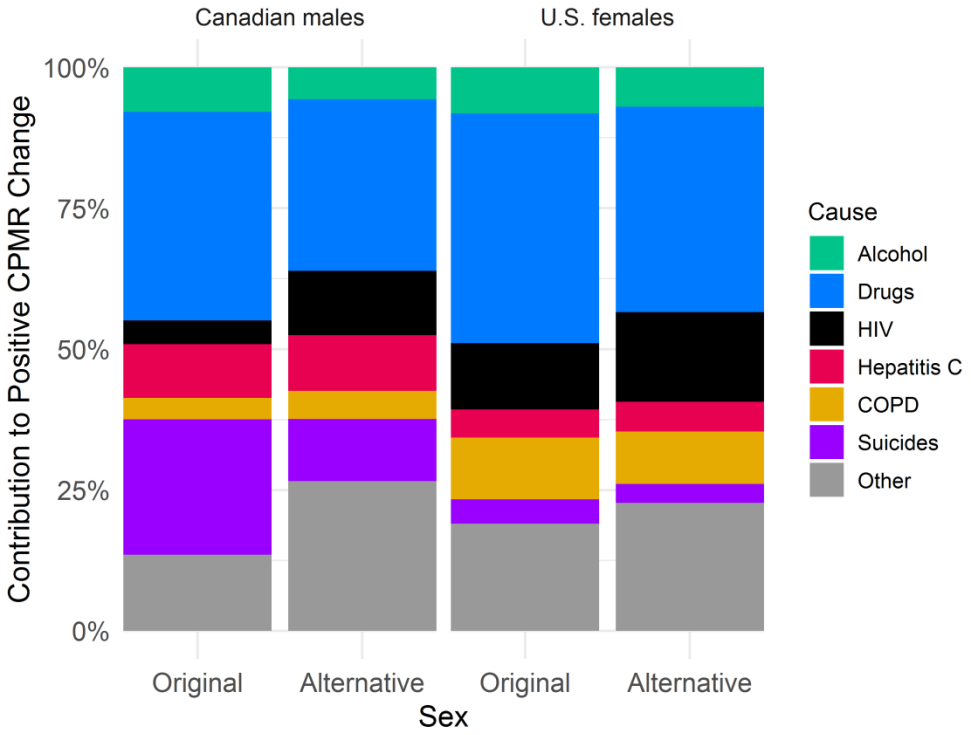


Figure S5. Cumulative contribution by leading causes to the deterioration in mortality between the advantaged and disadvantaged cohorts, according to the location of the disadvantaged cohort. For Canadian males, disadvantaged cohort originally placed in 1957, and alternatively in 1957, and U.S. females, originally placed in 1960, and alternatively in 1958.

Cohort partial mortality rate measure and APC variables

Estimates from the $\Delta CPMR_i^{d-a(35,54)}$ depicted in Figure 3 were useful to identify the leading causes of death contributing to the relative mortality deterioration between the *advantaged* and the *disadvantaged* cohorts within the age interval 35-54y.

However, the interpretation of these findings should be done cautiously for two reasons. First, whereas the variable age is controlled when comparing the mortality 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 could be result from period variations. During the age-interval of observation, 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., the cohort 1945 was observed during the period 1980-1999, being exposed only to the first years of the opioid crisis, which started in the late-1990s). The more recent cohorts, by contrast,

were exposed during a more extended period and at more advanced stages of the epidemic (e.g., the cohort 1955 was observed during the period 1990-2009, most of it amid the opioid crisis).

Second, the decomposition of $\Delta CPMR^{d-a(35,54)}$ allows us to identify the causes behind the relative mortality deterioration between the disadvantaged and the advantaged cohorts, but because of how our window of observation is configured, we were not able to identify whether the same causes of death that were responsible for the deterioration were also behind the subsequent improvements in mortality for the cohorts born after the boomers. If that is not the case (i.e., the causes behind the mortality deterioration were different from those behind subsequent improvements), the boomer excess in all-cause mortality would not be strictly related to the sum of multiple cause-specific excesses, but rather an artifact from 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.

Detrended cohort effects from the APC model

Figure S6 shows the detrended cohort effects of the leading causes to the boomer excess mortality in the Canada and the United States, and Figure S7 those in the United States by race/ethnicity. These plots are the facetted version of Figures 4 and 5.

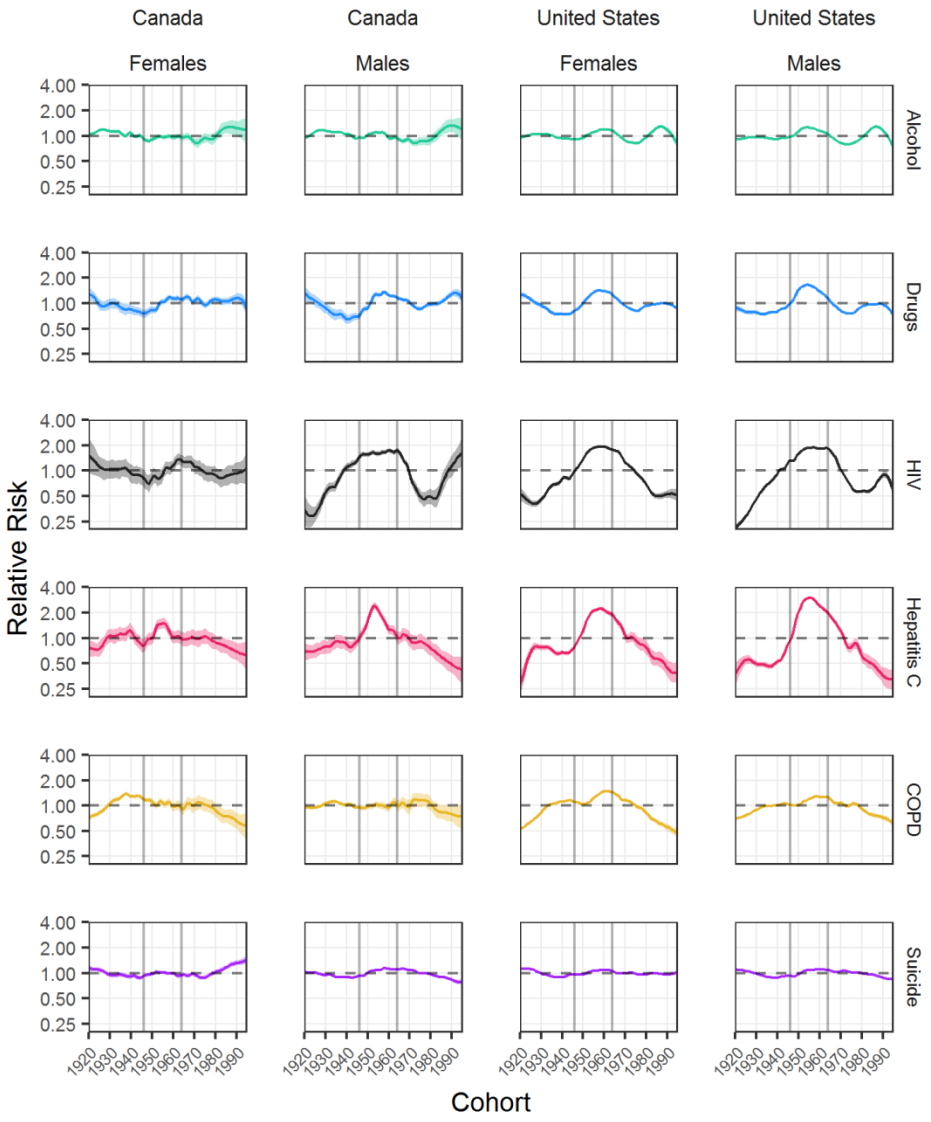


Figure S6. APCd estimates by cause of death, sex, and country.

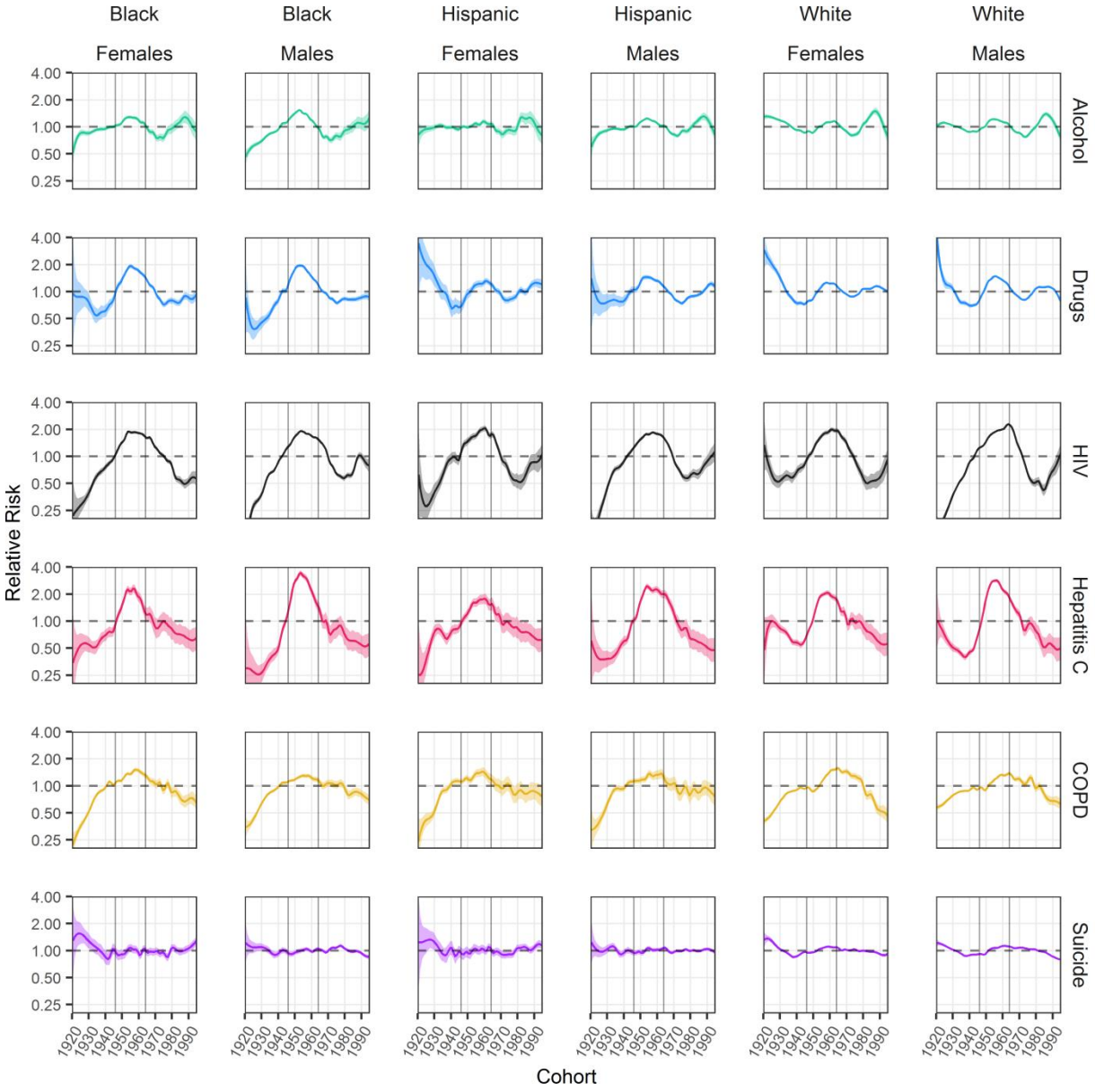


Figure S7. APCd estimates for each sex and racial-ethnic group, comparing the leading causes of boomers' excess mortality

Temporal dynamic of the boomer excess mortality by cause of death

Figures S8 and S9 are complimentary to Figures 6 and 7.

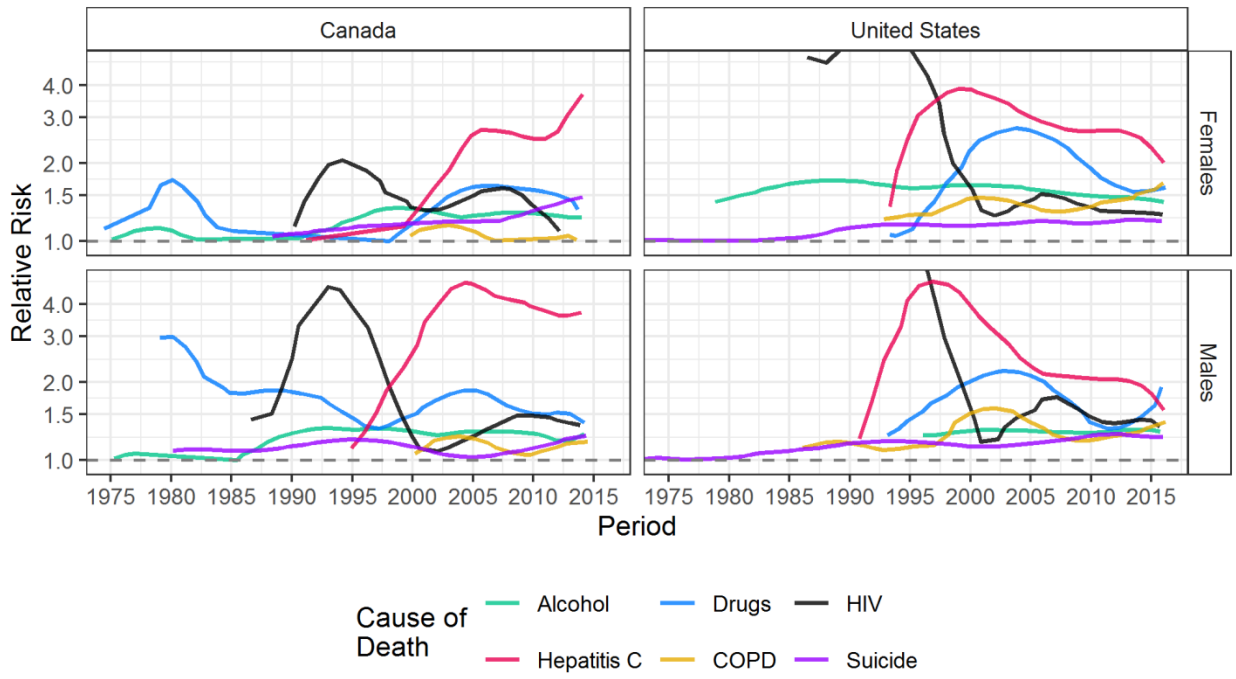


Figure S8. Variation of the magnitude of the relative risk of boomers compared to the baseline (upper), and Variation in magnitude of relative risk and cohort location of the boomers' hump ridge over age by cause of death (bottom panel)

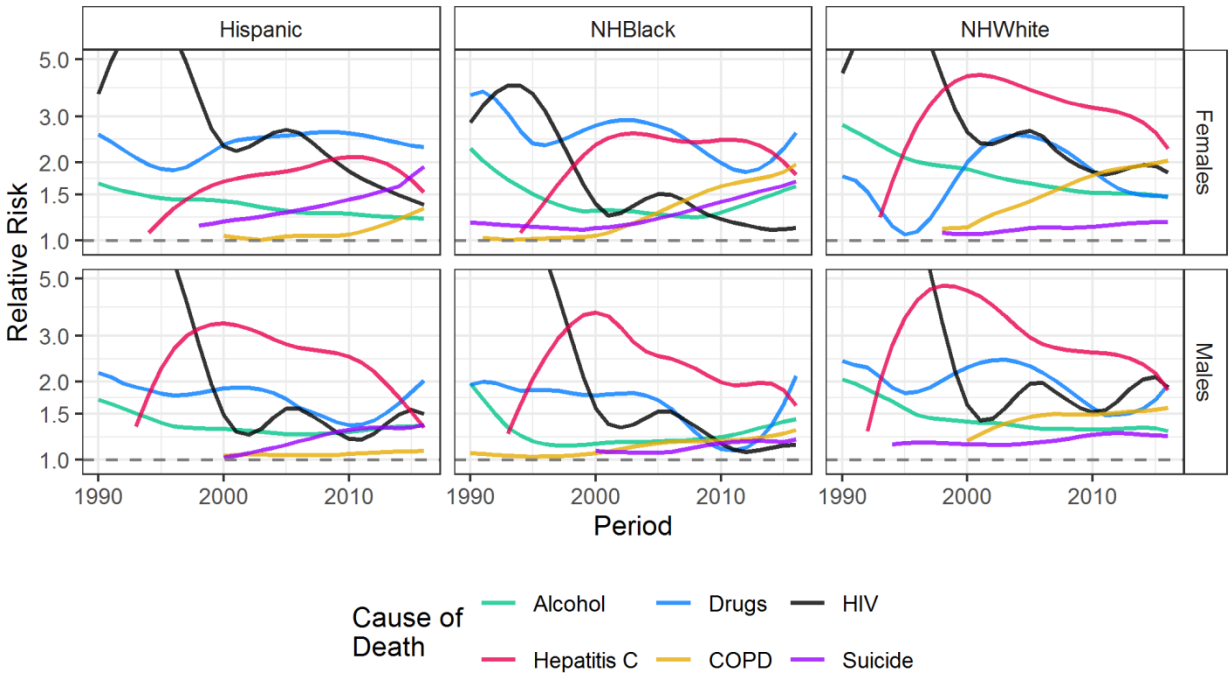


Figure S9. Variation of the magnitude of the relative risk of boomers compared to the baseline (upper panel) and Variation in intensity (relative risk) and cohort location of the boomers' hump ridge over age by cause of death (bottom panel)