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Analysis of Historical U.S. Population Mortality Improvement Drivers 1959-2016

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Author

Andrés M. Villegas, Ph.D.
University of New South Wales

Madhavi Bajekal, Ph.D.
University College London

Steven Haberman, Ph.D., FIA
City, University of London

Luke Zhou
University of New South Wales

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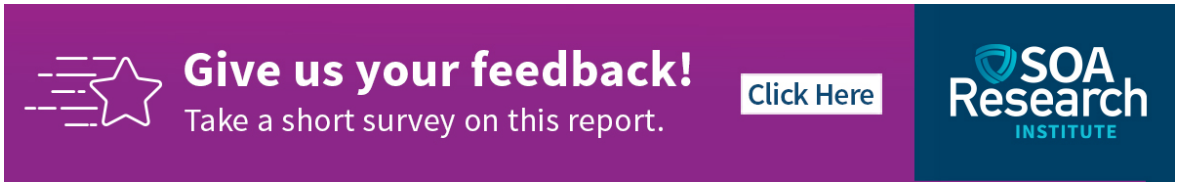


CONTENTS

Analysis of Historical U.S. Population Mortality Improvement Drivers 1959-2016

Executive Summary

This report has been prepared in response to the SOA call for research proposals for the analysis of the drivers of historical U.S. population mortality improvement since 1950 to build on an earlier SOA-sponsored project "Components of Historical Mortality Improvements" (Li et al., 2017a,b). The research objectives of the current project are:

i. Identifying significant mortality drivers in the U.S. population that have a high likelihood of being linked to the improvement or deterioration of mortality in each of the age-period-cohort (APC) components quantified in the companion study, and

ii. Quantifying possible correlations using cause of death and other relevant data sources and quantifying the likely degree of causality between each APC mortality improvement component and the relevant extrinsic drivers.

To achieve these objectives, this report uses cause of death mortality data for the period 1959-2016, stemming from work sponsored by the SOA to develop a cause of death extension of the HMD and which contains data for 92 subcategories of death (see Barbieri (2017)). These data have been aggregated into two levels. The first level comprises 6 broad groups including circulatory diseases, neoplasms, respiratory diseases, digestive system diseases, external causes and other causes. The second level is a more detailed grouping with 26 of the main subcauses within each cause in the first level.

We also identify and analyze some key causes that serve as markers of trends in behavioral risk factors that could drive mortality change. These risk factors are: AIDS and tuberculosis, alcohol abuse, dementia and Alzheimer's disease, diabetes and obesity, drug dependency, homicide, hypertensive disease, self-harm, and smoking.

These different groupings of causes of death have been analyzed using several analytical strategies, including a graphical descriptive analysis of trends in age-standardized-death-rates, improvement rate heatmaps, life expectancy decomposition methods borrowed from

demography, and formal age-period-cohort decomposition of mortality rates. In particular, to disentangle the period and cohort components of mortality change, this report relies on the Period-Cohort improvement rate (PCi) model:

$$-\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha + \tilde{\kappa}_t + \tilde{\gamma}_{t-x},$$

where $m_{x,t}$ is the mortality rate at age x in year t and $-\log \frac{m_{x,t}}{m_{x,t-1}}$ are the corresponding mortality improvement rates. This model decomposes the mortality improvements into: the average mortality improvements for all ages during the 1959-2016 study period captured by α; the period (calendar year) deviations from this average improvements captured by $\tilde{\kappa_t}$; and the cohort (year of birth) deviations from this average improvements captured by $\tilde{\gamma}_{t-x}$.

Figures 2 and 3 show for women and men aged 20 to 89, respectively, the results of applying the PCi model for causes of death at the two levels of disaggregation. Figure 4 shows the corresponding decomposition for the risk factors.[1]

The main findings of this report are:

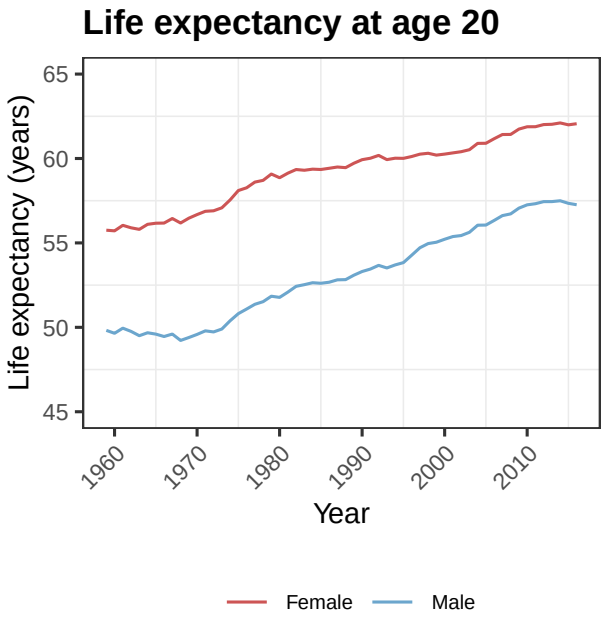
- **The main story of mortality improvements in the period 1959-2016**

Between 1959 and 2016, the U.S. saw a remarkable reduction in mortality rates, with rates of mortality for both women and men aged 20-89 improving at an average pace of around 1% p.a. This resulted in period life expectancy at age 20 increasing from 55.75 years to 62.06 for females and from 49.82 years to 57.26 years for males between 1959 and 2016 (see Figure 1).

The story of mortality evolution in the U.S. in the second half of 20th century which continued into the first decade of the 21st century has been mainly a story of mortality improvements from circulatory diseases, with this group of diseases accounting for 6.21 years of the 6.31 years of increase in life expectancy at age 20 for females and for 6.23 years of the 7.44 years of increase in life expectancy at age 20 for males. The improvement of mortality from circulatory diseases can be linked to both a delayed onset of disease due to improvements in the prevalence of cardiovascular risk factors such as hypertension, high cholesterol and smoking and reductions in case fatality with the introduction of novel surgical and pharmacological treatments. However, increases in body mass index and diabetes have partially offset some of the gains.

[1] In these *mortality improvement stripes* plots, the left panel labeled *"Avg."* represent the parameter values of α, the middle panel labeled *"Calendar year"* the period effects as captured by $\tilde{\kappa}_t$, and the right panels labeled *"Birth cohort"* the cohort effects, $\tilde{\gamma}_c$. In the plots, the values of the PCi model are mapped to a color so that blue hues signify mortality improvements and red hues mortality deterioration. We note that in the improvement rate stripes we have capped very large improvements and deteriorations at ±5% p.a. so that very intense reds represent a deterioration of more than 5% p.a. and very intense blues represent improvements of more than 5% p.a.

Figure 1: Period life expectancy at age 20, 1959–2016



Nevertheless, despite the clear dominance of circulatory diseases as a leading cause of death and as the main contributor to mortality improvements over the study period, the decline of mortality has been far from linear. The trajectories of other major causes have ebbed and flowed; and the patterns of behavioral risk factors saw changes between generations.

- **Period trends**

In this study we have identified five broad periods of mortality change:

1959-1970: This period saw a general leveling off in mortality rates after the unprecedented decline in mortality experienced by Americans from 1940 to the mid 1955s. Noticeably, the 1960s were the first decade in which a decline in mortality rates from cardiovascular diseases as a whole started to become evident after reaching a peak in the 1950s. However, despite the overall fall in cardiovascular mortality rates, their rates of mortality improvements in the 1960s were sluggish, mainly due to the increase in mortality rates from ischaemic heart disease (the dominant group of cardiovascular deaths) which only reached peak mortality rates in the mid 1960s, partially offsetting the mortality improvements from stroke and other circulatory diseases. In addition, lung cancer and traffic accidents stand out as the other causes with the most noticeable adverse mortality changes during the 1960s.

1970-1980: Mortality trends experienced a turning point around 1968 with mortality decline accelerating thereafter with the 1970s being the decade of the fastest all-cause mortality improvements over the study period. The 1970s marked the beginning of rapid declines in mortality from cardiovascular diseases, resulting from sustained reductions in lifestyle risk

Figure 2: Smooth improvement stripes for causes of death, Females, 1959–2016, 20–89

Figure 3: Smooth improvement stripes for causes of death, Males, 1959–2016, 20–89

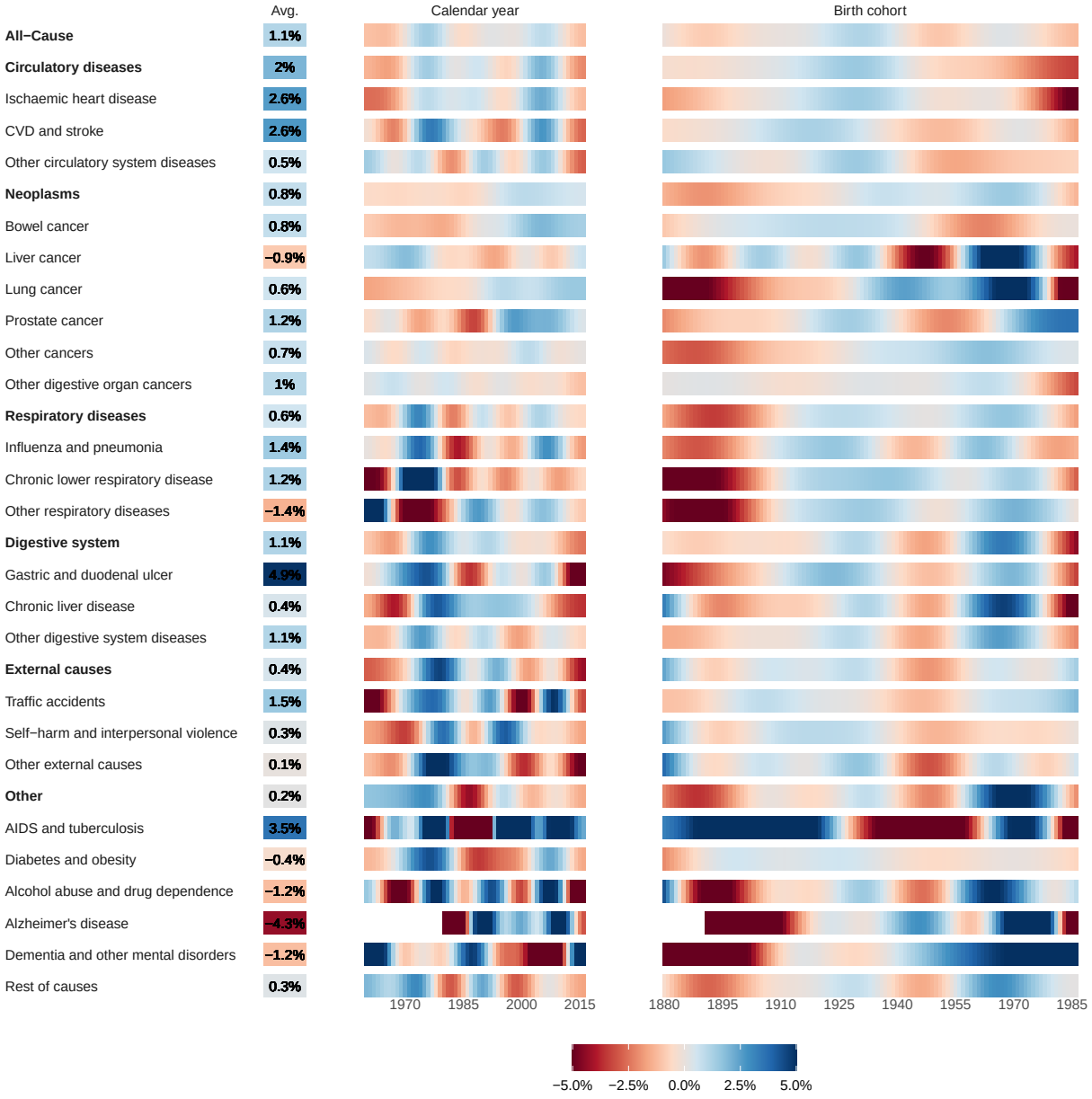
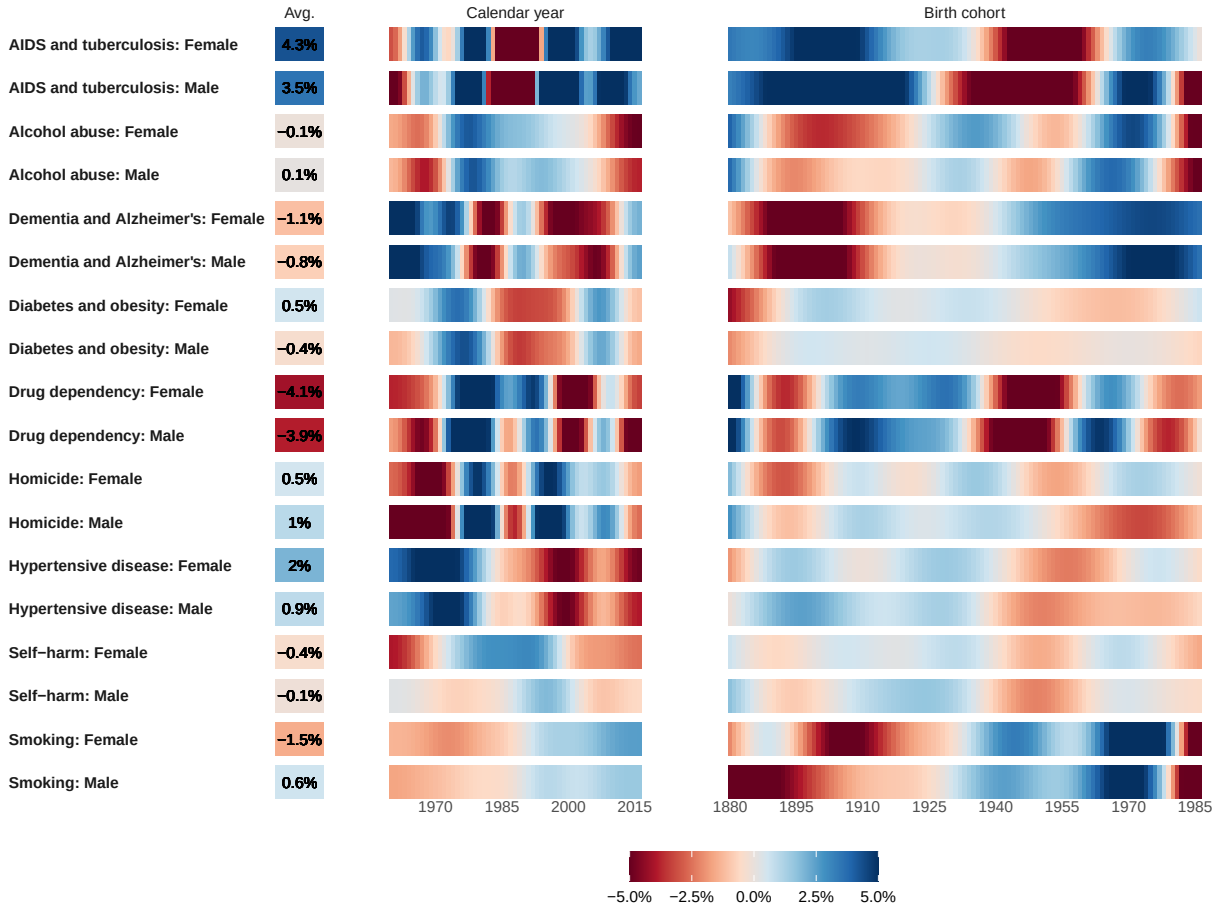


Figure 4: Smooth improvement stripes for risk factors, 1959–2016, 20–89



factors such as cholesterol levels and smoking, as well as major advances in medical interventions such as prehospital resuscitation, coronary artery bypass surgery and the treatment of hypertension and cholesterol. Importantly, 1970s was the decade in which cigarette consumption in the U.S. started to decrease steadily, following the influential first report of the Surgeon General's Committee on Smoking and Health in 1964 establishing a causal link between smoking and lung cancer. However, despite the general decline in smoking prevalence in the 1970s, lung cancer continued exerting a large negative contribution to mortality change as trends in lung cancer are mainly determined by cigarette consumption patterns with a 20-30 year lag. Finally, death rates from traffic accidents decreased significantly during this period following the passage in 1966 of the Highway Safety Act and the National Traffic and Motor Vehicle Safety Act.

1980-1995: Over this period mortality improvements continued although at a much slower pace than in the 1970s. This deceleration was mainly driven by the emergence of the AIDS/HIV epidemic in the early 1980s. In addition, cardiovascular diseases continued to show important mortality improvements albeit at a slower pace than in the 1970s, partially due to the rising trends in the prevalence of obesity and diabetes, resulting from secular

changes in agricultural policies, diet, food environment, physical inactivity, and sleep deprivation.

1995-2010: Mortality improvements accelerated from 1995 through to 2010, especially for men. This accelerated improvements resulted from improvements in several causes. First, the improvement in cardiovascular disease mortality continued at a pace faster than the period preceding it, but with a clear age-divide with older Americans experiencing steep mortality declines from the early 2000s that were not matched among younger adults. Second, neoplasms as a whole also started to experience sustained mortality improvements after having peaked in the early 1990s. While cohort effects, especially related to smoking patterns, are behind much of the decrease in cancer mortality during this period, there are some noteworthy period patterns for lung, prostate and breast cancer, linked, respectively, to the lagged effects of the steady decrease in tobacco consumption following the 1964 Surgeon General's reports on smoking and health; the widespread introduction of prostate-specific antigen (PSA) screening; and the increase in the use of adjuvant chemotherapy and in the detection of small palpable breast tumors. Third, the incidence and mortality from AIDS showed a sharp decline after the introduction of anti-retroviral therapy in 1996. In contrast to these positive trends, this period saw the emergence of Alzheimer's, dementia and other mental disorders as a very important cause of old-age mortality, especially among women. Moreover, mortality rates from unintentional drug poisoning linked to prescribed opioid use experienced a significant increase, foreshadowing the narrative of "deaths of despair" which would come to dominate the discussion of U.S. mortality change in the 2010s.

2010-2016: Starting from around 2010, overall mortality improvements in the U.S. show a considerable slowdown leading to a stagnation in life expectancy. The main narrative behind the slowdown of mortality improvements after 2010 has been that of "deaths of despair", which argues that the slowdown of mortality improvement is mainly a cohort effect driven by an increase in accidental drug and alcohol poisoning, chronic liver disease, cirrhosis and suicide, especially among middle-aged white Americans. However, this narrative has been challenged and it is argued that period effects play an important role and that, beside opioids, there are other drivers of the slowdown of improvements including increases in older age mortality from Alzheimer's disease, increases in homicide and suicide, and a slowdown in mortality improvements from cardiovascular diseases.

- **Cohort effects**

We can identify five broad groups of birth cohorts, with the cohort boundaries for women differing to those for men as a result of the delayed onset of common risk factors among women.

Female 1881–1925, Male 1881-1920: This cohort group shows average mortality improvements for women and slightly below average improvement for men. These below average improvements are mainly explained by smoking patterns, with cigarette consumption

(in terms of average numbers of years spent as cigarette smoker before age 40) increasing steadily from the 1885-1889 generation to reach a plateau for the 1910-1925 generations of men and the 1925-1940 generation of women.

Female 1925-1945, Male 1920-1940: This cohort group shows above average mortality improvement, stemming from positive cohort effects for the majority of causes of death and most risk factors. In particular, this is the first generational group which does not show an increase in smoking prevalence relative to the preceding generations. In contrast to the positive cohort effects from other risk factors, this generation – which was in their 40s to 60s in the 1980s – shows negative cohort effects from AIDS and tuberculosis, albeit milder than those seen in the following generation.

Female 1945-1960, Male 1940-1955: The generation of early baby boomers is noticeably one of the generations with the slowest mortality improvements among the generations included in the study. The reasons for the below average improvement are multi-factorial and reflect adverse patterns in a diverse number of causes and risk factors. Early baby boomers continued to experience positive cohort effects from smoking-related causes of death as a consequence of the continued decline in tobacco consumption. However, the positive gains from smoking were counterbalanced by negative trends in other factors and causes. In particular, this generation marked the start of a turning point in generational patterns of cardiovascular mortality with cohorts born from around 1945-1950 onwards showing negative cohort effects for this group of causes. This pattern coincides with similar turning points in the mortality rates from markers of cardiovascular risk factors including hypertension and obesity and diabetes. Early baby boomers also show distinctive negative patterns in mortality rates associated with AIDS, alcohol abuse, drug dependency and suicide.

Female 1960-1975, Male 1955-1970: This generation shows average mortality improvements which reflect offsetting patterns for different causes and risk factors. Similarly to the preceding generation, this generation continued the negative cohort effects from cardiovascular diseases, obesity and diabetes, and hypertension, consistent with the increasing trend in obesity prevalence which started with the 1955 birth cohort. Likewise, this generation continues to benefit from the positive cohort effects stemming from reductions in smoking prevalence. However, in sharp contrast with the previous and subsequent generations, this cohort group shows positive cohort effects for alcohol abuse and drug dependency.

Female 1975-1986, Male 1970-1986: Together with the baby boomer generation, this youngest generation shows the worst mortality improvements among the cohorts in the study. This cohort groups shows important negative cohort effects for alcohol and drug abuse mortality. Noticeably, the positive cohort effects associated with smoking and lung cancer seem to be winding down, with women and men of this generation showing negative cohort effects in lung cancer mortality.

Section 1: Introduction

This report has been prepared in response to the SOA call for research proposals for the analysis of the drivers of historical U.S. population mortality improvement since 1950.

This research project is designed to build on an earlier SOA-sponsored project "Components of Historical Mortality Improvements" (Li et al., 2017a,b). This project evaluated alternative age-period-cohort (APC) modeling approaches for mortality rates and mortality improvement rates with the objective of decomposing mortality improvements in the U.S. into age, period, cohort and residual components.

1.1 RESEARCH OBJECTIVES

The research objectives of the current project are:

i. Identifying significant mortality drivers in the U.S. population that have a high likelihood of being linked to the improvement or deterioration of mortality in each of the APC components quantified in the companion study, and

ii. Quantifying possible correlations using cause of death and other relevant data sources and quantifying the likely degree of causality between each APC mortality improvement component and the relevant extrinsic drivers.

1.2 BACKGROUND: DRIVERS OF MORTALITY CHANGE IN THE U.S.

The understanding of the drivers behind mortality change is a topic that has attracted significant attention in the epidemiological, medical and economic literature. The SOA has recognized this, producing the 2013 report "Literature Review and Assessment of Mortality Improvement Rates in the U.S. Population: Past Experience and Future Long-Term Trends" (Rosner et al., 2013) which reviewed the literature investigating the interaction of age, period and cohort effects in U.S. mortality improvement and the external factors that might be correlated with variations in mortality improvement.

In the period since the production of this literature review, there have been important academic contributions to the understanding of mortality change in the U.S.. We present here a short review of the main recent contributions in the literature and provide a fuller review in Section 2.

Chief among recent contributions was the widely-publicized work of Case and Deaton (2015) reporting an increase in mortality rates among middle-aged U.S. white men and women with low levels of education. They argued that this was a cohort-effect and that the reversal in the long-term decline in the all-cause mortality rate was largely driven by increases in deaths from suicides, alcohol and drug poisonings, and chronic liver diseases and cirrhosis

(pooled for analysis as "deaths of despair"). Subsequently, Masters et al. (2018) applied APC methods to analyze trends for each of these causes separately for men and women and confirmed that deaths for middle-aged whites had increased in the 21st century, but primarily due to rapid increases in drug-related mortality from the late 1990s rather than in suicides or alcohol use; and crucially, that this increase was a period rather than a cohort effect linked to prescribed opioid use. In an earlier paper, Masters et al. (2014) employed an APC model to study the long-term trend in all-cause and cause-specific mortality for the U.S. black and white population over the 1959 to 2009 period and found clear cohort-based trends in heart disease, stroke, lung cancer, female breast cancer and other cancer mortality, accompanied by especially pronounced period-based reductions in mortality from heart disease, stroke, infectious diseases and homicide.

Despite the long-term trend in health and mortality improvements in the U.S., recent research has highlighted the persistence and widening of trends in inequalities in life expectancy by socioeconomic circumstances measured at either the individual level (e.g. educational attainment, relative income, race/ethnicity) or area-level (unemployment, poverty, urban-rural); or analyzed for all-causes or partitioned into major causes of death (Chetty et al., 2016; Masters et al., 2018; Singh et al., 2017). The pace of change has varied with the unit of analysis, the causes of death and specific age groups; but essentially the overall pattern has been one of heterogeneity and unequal gains in mortality improvements.

Some of the newly emerging insights point to possible causal mechanisms driving the gradient in life expectancies. Chetty et al. (2016) found that at county-level, life expectancy in middle age for low income people was highly correlated with health behaviors (e.g. smoking, obesity) but not significantly associated with area differences in levels of medical care. Like in England (Marmot, 2010), they also found that life expectancy of low income men varied more between areas than it did for more advantaged men implying that unmeasured contextual factors play a role in addition to compositional factors in the spatial patterns of disadvantage at area level (Macintyre et al., 2002). They also found little association between life expectancy variation and health care access variables.

To estimate the independent effects of known drivers of mortality change, Dwyer-Lindgren et al. (2017) specifically modeled the contribution of three sources of influences to explain the variation in life expectancy at birth at county level:

i. broader socioeconomic factors,

ii. behavioral and metabolic risk factors,

iii. access to health care and health care quality.

Each group was calculated as a composite index of a range of estimates available cross-sectionally for all counties in one year, 2009. They concluded that the association of life

expectancy with race/ethnicity and socioeconomic factors at the county level is largely mediated through behavioral and metabolic risk factors, with a much smaller but significant contribution of health care access/quality.

1.3 RESEARCH METHODOLOGY

The choices of the three drivers identified above from the literature review are constrained by data availability: namely that the lowest level of at which mortality data are available for the U.S. is at county-level; that survey-based estimates of health behaviors are not available for all years included in this study and that there are problems with obtaining stable measures of broader socioeconomic factors.

On this last point, we had planned to use a composite index of relative deprivation at county level (Singh, 2003; Barbieri, 2020). The index has been updated at each successive census from 1970 onwards (GK Singh, personal communication) and the relative ranking of areas seemed to have remained fairly stable over time. The index includes multiple (17) indicators of social disadvantage of which measures of poverty, income, education have the largest weights. The composite deprivation index captures both the compositional and contextual effects and satisfies our first two criteria. Because the number of deaths per year for a county is fairly small, the plan was to stratify counties into population weighted quintiles or deciles of increasing deprivation. This is the approach that we have successfully applied to model long-run trends in inequalities in mortality in the UK (see Villegas and Haberman (2014) and Alai et al. (2018)). However, access to this data source was not possible.

Because of problems with the availability of data consistently at an appropriate level of disaggregation, we decided to adopt an indirect approach to measuring the effect of these known drivers of mortality change.

In order to proxy the impact of behavioral risk factors and access to health care on mortality change, we propose to use mortality partitioning by cause of death into biologically relevant subcomponents. For example, Carnes et al. (2006) partitioned deaths into 'intrinsic' and 'extrinsic' components. Of those classed as extrinsic (i.e., related to exogenous environmental factors) others have further partitioned deaths into specific markers of risky behaviors – e.g. smoking related deaths, deaths related to alcohol and drug misuse, deaths related to obesity (Dwyer-Lindgren et al., 2016; Masters et al., 2018) and deaths related to causes amenable to healthcare treatment (Nolte and McKee, 2012). To provide a broad landscape of mortality change, we complement this analysis with an analysis of mortality trends for the leading causes of death: neoplasms, circulatory diseases, respiratory diseases, digestive diseases, external causes, and 'all other' causes.

After deriving a suitable classification of mortality according to causes of death (COD), we use three main approaches for understanding trends in mortality change. First, to identify main features of the data that would need to be accounted for in further modeling, we perform a descriptive analysis of mortality trends by COD. This includes, among others,

a graphical descriptive analysis of trends in age-standardized-death-rates and improvement rate heatmaps.

In order to obtain an assessment of the age groups and causes of death driving the temporal improvement in life expectancy, we apply life expectancy decomposition methods borrowed from demography such as the Arriaga method (Arriaga, 1984). Such methods, have the advantage of enabling a simple and easy to communicate graphical representation of the contribution of each age group and cause of death to gains in life expectancy over a certain period of time.

Second, we analyze national mortality trends by cause of death to assess their contribution to change in all-cause mortality and their possible decomposition into age, period and cohort components.

Cause-specific analysis of mortality trends encompasses an analysis starting at the broadest grouping of cause-partitioning, i.e. leading causes of death at level 1 (or ICD Chapters such as neoplasms, circulatory diseases, respiratory diseases, digestive diseases external causes), to provide a landscape and overview of the relative contribution of leading causes. We then drill down to finer levels such as partitioning mortality into extrinsic causes associated with behavioral risk factors.

Third, similarly to the preceding SOA project, we apply different APC modeling approaches to the cause-specific mortality rates and mortality improvement rates to derive a possible decomposition of cause-specific rates into APC components. Among the existing APC modeling specifications, we consider the simplified Plat model:

$$\log m_{x,t} = \alpha_x + \kappa_t^{(1)} + (x - \bar{x})\kappa_t^{(2)} + \gamma_{t-x},$$

the standard APC model,

$$\log m_{x,t} = \alpha_x + \kappa_t + \gamma_{t-x},$$

and the Period-Cohort improvement rate model

$$-\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha + \tilde{\kappa}_t + \tilde{\gamma}_{t-x},$$

where $m_{x,t}$ is the mortality rate at age x in year t and $-\log \frac{m_{x,t}}{m_{x,t-1}}$ are the corresponding mortality improvement rates.

The simplified Plat model was suggested by the previous SOA project as being appropriate to model all-cause mortality trends for the U.S. populations for ages 20 to 95, while the standard APC model was found to be appropriate for ages 55 to 95. The standard APC model has been previously considered in the decomposition of cause-specific U.S. mortality trends into

age-period-cohort components (Masters et al., 2014, 2018; Yang, 2008). Further, the Period-Cohort improvement rate (PCi) is the improvement rate equivalent of the standard APC mortality rate model.

1.4 DISCUSSION OF LIMITATIONS AND RELATED ISSUES

The analysis of mortality trends by cause of death and their association with extrinsic mortality drivers comes with several important limitations and challenges that need to be acknowledged.

1.4.1 CHANGES IN CAUSE OF DEATH CODING

First, trend analysis of cause-specific mortality can be affected by changes in the diagnosis and in the classification of causes of death. The World Health Organization (WHO) has reviewed the International Classification of Diseases (ICD) regularly in the 20th century to reflect new diseases, changes in medical terminology and the development of medical knowledge (Moriyama et al., 2011). These data production changes can cause substantial discontinuities in the mortality trends of some causes of death which would need to be accounted for in the analysis. Alternatives for handling these discontinuities include the use of bridge coding (Anderson et al., 2001) and statistical correction methods (Rey et al., 2011). Fortunately, the SOA has sponsored the expansion of the Human Mortality Database to include consistent cause of death information for eight countries including the United States for periods going back as close as possible to 1950 (Barbieri, 2017). We use these data, coupled with statistical correction methods, to circumvent the limitations imposed by cause of death data coding changes.

1.4.2 IDENTIFICATION AND AGE-PERIOD-COHORT DECOMPOSITION

It is well known that the decomposition of health trends into age/period/cohort components can be problematic, as the relationship $cohort = period - age$ poses an inherent identification problem that makes it difficult to clearly separate the mutually dependent changes across the three dimensions. This implies that one needs to be extremely cautious when interpreting results from APC models as any such split is model dependent and is only correct if the underlying (unknown) data generating process happens to be the same as the statistical model chosen (Bell and Jones, 2013, 2014; Harper, 2015).

While, in the preceding SOA-sponsored project, Li et al. (2017a,b) tested the robustness of the decomposition of mortality to the use of alternative identification constraints, they did not acknowledge explicitly the limitations in the interpretation of their APC decomposition results. Further, the identification constraints used by Li et al. (2017a,b) in their robustness tests were all of a similar nature, which involved relegating the cohort dimension to a secondary role with the dimensions of age and period being assumed to be dominant.

In this report, we aim to be cautious in interpreting any age-period-cohort decomposition that we derive. Thus, we expand the test set of identifiability constraints used by Li et al. (2017a,b) also to include identification schemes where cohort effects are assumed to be dominant over period effects. Further, we use external information (e.g. on smoking patterns) to help confirm the existence of the age, period or cohort patterns that we might identify.

1.4.3 CORRELATION VS. CAUSATION

Finally, it is worth noting that our methodological approach only allows us to establish the association between mortality trends and relevant extrinsic drivers, and not to establish the degree of causality between them. The determination of causality between mortality and the extrinsic drivers is a difficult task as the causal relationship could be in either or both directions, and identifying the direction of this association would require the use of prospective longitudinal health and mortality data which is out of the scope of this research project.

1.5 STRUCTURE OF THE REPORT

The report is structured as follows. In Section 2, we provide an updated literature review of U.S. mortality trends, with a focus on three areas: the approaches typically used to group causes of death; the key risk factors driving recent mortality change; and the use of APC modeling for understanding trends in mortality.

Section 3 considers possible sources of U.S. mortality data. We provide an overview of several widely used public data sources. Then, we consider the issues surrounding the construction of a consistent time series of mortality rates by cause of death and how this affects our choice of data source. Finally, we set out the rationale for the choice of cause of death groupings for use in our analysis.

Section 4 provides a descriptive analysis of U.S. mortality trends by cause of death. First we focus on general time trends for various causes of death and risk factors using age-standardized death rates. Then, we move to the examination of trends in life expectancy with a focus on the contribution of different age groups and causes of death to changes in life expectancy over the 1970–2015 period. Finally, we present mortality improvement rate heatmaps which allow us to make an initial discussion of mortality improvement rate patterns across the dimensions of age, period and cohort by cause of death.

In Section 5, we focus on a systematic decomposition of cause-specific mortality trends across the dimensions of age, period and cohort using well-known actuarial and demographic modeling techniques. We provide a description of generalized APC mortality models, then consider model selection and the need to balance goodness of fit and parsimony and consider robustness to changes in parameter constraints. We describe the reasons for our choosing the PCi model for the detailed analysis. We then consider the effects on the models of changes

in the coding of cause of death. The section concludes with a detailed presentation of results for the principal model under consideration, the PCi model.

Section 6 provides an interpretation of the decomposition of mortality improvements into the APC components that arise from the modeling in Section 5 and considers the links to possible mortality drivers and the features that characterize the identified time periods and generations.

Section 7 provides some concluding comments and identifies areas for further research.

Section 2: Literature Review

The understanding of the drivers behind mortality change is a topic that has attracted significant attention in the epidemiological, medical and economic literature. The SOA has recognized this, producing the 2013 report "Literature Review and Assessment of Mortality Improvement Rates in the U.S. Population: Past Experience and Future Long-Term Trends" (Rosner et al., 2013). This report reviewed the literature investigating the interaction of age, period and cohort effects in U.S. mortality improvement and the external factors that might be correlated with variations in mortality improvement.

Since the production of this literature review, there have been important academic contributions to the understanding of mortality change in the U.S. As such, this section provides a brief literature review of recent literature to update and complement the 2013 SOA report. This review focuses on three areas of particular relevance for the rest of the report, namely: the approaches typically used to group causes of death, the key risk factors driving recent mortality change, and the use of APC modeling for understanding trends in mortality.

2.1 CAUSE OF DEATH GROUPING

In the analysis of death by cause, it is important to establish a grouping of causes which can be linked to possible drivers of mortality change over the last four decades. One comprehensive, 4-level, hierarchical grouping of causes is the Global Burden of Disease (GBD) (Wang et al., 2016). Level 1 of the GBD has three broad groupings of causes of death, namely: communicable, maternal, neonatal and nutritional diseases; noncommunicable diseases; and injuries. Level 2 subdivides the causes into 21 major groupings, while levels 3 and 4 consist of 166 and 261 causes, respectively. It is then possible to rank causes of death at varying levels of detail.

For example, Dwyer-Lindgren et al. (2016) study deaths in the United States from 1980 to 2014, at Level 2 of the GBD groupings (with 21 groups). In 2014, the leading three causes of death were cardiovascular diseases, neoplasms, and neurological disorders. In addition, Dwyer-Lindgren et al. (2016) find that while some major causes of death such as cardiovascular disease and neoplasms recorded a reduction in mortality rates between 1980 and 2014, other causes such as neurological diseases and mental and substance abuse disorders showed the opposite trend with a significant increase in mortality rates.

In another study, Mokdad et al. (2018) apply the GBD methodology to analyze health trends in the U.S. between 1990 and 2016. At the more detailed Level 3 grouping of the GBD, they report that ischaemic heart disease (IHD) remained the leading cause of death in 2016 despite a reduction of 50.7% in its age-standardized death rate (ASDR) between 1990 and 2016. The next four leading causes of death in 2016 were cancer of the trachea, bronchus, and lung; chronic obstructive pulmonary disease; Alzheimer's disease and other dementias; and cancer of the colon and rectum. Notably, Alzheimer's disease and other dementias were only the

seventh leading cause of death in 1990, but climbed to the fourth position in 2016 due to an increase of 11.3% in their ASDR. By contrast, deaths due to motor vehicle road injuries passed from being ranked the third cause of death in 1990 to the sixth cause in 2016 as result of a decline of 35.4% in its ASDR.

2.2 RISK FACTORS

A step beyond the analysis of mortality trends by causes of death group is the identification of the main risks factors associated with mortality.

For example, McGinnis and Foege (1993) introduce the concept of *actual causes of death* to identify and quantify the major external factors that contribute to death. Applying this concept, Mokdad et al. (2004) report that in 2000 the top three leading causes of death in the U.S. were tobacco; poor diet and physical inactivity; and alcohol consumption. Other factors identified by Mokdad et al. (2004) include microbial agents, toxic agents, motor vehicle crashes, incidents involving firearms, sexual behaviors, and illicit used of drugs.

The GBD has also developed a comprehensive approach for the evaluation of risk factors, their association with causes of death, and their contribution to total deaths (GBD 2017 Risk Factor Collaborators, 2018). The GBD approach uses medical studies to quantify the relationship between 84 risk factors and multiple causes of death. By amalgamating the causes of death across the different risk factors, it is then possible to map each risk factor to the number of deaths attributable to it. These risk factors are grouped into three broad categories to aid interpretation, namely: behavioral; environmental and occupational; and metabolic risks. Using the GBD framework, Mokdad et al. (2018) has identified that in 2016 the top 7 risk factors associated with U.S. mortality belonged to the behavioral and metabolic categories. The leading behavioral risk factors were dietary risks, tobacco, and alcohol and drug use; while the top metabolic factors were hypertension, obesity, diabetes and high cholesterol.

In what follows we discuss some risk factors which may be particularly relevant in explaining recent trends in U.S. mortality.

2.2.1 SMOKING

Smoking remains the main contributor to preventable mortality in the U.S. (Mokdad et al., 2018). The estimation of smoking-attributable mortality can be done using either a direct or an indirect approach. The direct approach uses surveys to elicit the smoking status of participants who are then followed up to measure the differential mortality outcomes between smokers and non-smokers. Using this approach, Lariscy et al. (2018) estimate that in 2010 18% of female deaths and 26% of male deaths in the U.S. were attributable to smoking. The indirect approach, by contrast, exploits the close association between lung cancer mortality and smoking: it is estimated that 70% to 92% of lung cancer deaths are

attributable to smoking (Ezzati and Lopez, 2003) while lung cancer incidence among never-smokers is relatively low (Wakelee et al., 2007). Using an indirect approach, Lariscy (2019) estimates that between 1980 to 2004 15% of female deaths and 22% of male deaths among Americans aged 50 or older were attributed to smoking, in close agreement with the estimates obtained using a direct approach. Interestingly, both the direct and the indirect methods, estimate that, after lung cancer, ischaemic heart disease is the cause of death with the largest contribution to excess deaths associated with smoking (Lariscy et al., 2018, Lariscy (2019)).

One key feature of smoking-attributable mortality is that men and females show different trajectories as a consequence of smoking prevalence differentials. We refer the reader to Gutterman (2015) for a comprehensive review of gender differentials in smoking prevalence and the associated implications on mortality.

2.2.2 OBESITY

The prevalence of obesity in the United States has increased significantly in the past 40 years with the percentage of American adults considered obese increasing from 12% in 1975 to 36% in 2016 (WHO Global Health Observatory, 2020). Gutterman (2016) provides a detailed discussion on obesity prevalence in the U.S., its effect on mortality and implications for mortality projections. Some suggest that these obesity trends could lead to a potential decline in life expectancy in the U.S. in the 21st century (Olshansky et al., 2005). However, the relationship between obesity and mortality often shows counter-intuitive patterns. For example, there is the so-called obesity paradox whereby people who are moderately overweight have lower mortality than normal weight individuals (Gutterman, 2016). Moreover, while the U.S. has significantly higher prevalence of obesity compared to other developed countries, this is not matched by accompanying higher death rates from obesity related mortality (Barbieri et al., 2017).

Part of this complex picture is explained by the fact that obesity is a multi-system condition associated with a variety of health risks (Mitchell et al., 2011). Obesity is itself a cause of death, and is also associated with several other causes of death, most notably diseases of the circulatory system, diabetes, kidney diseases, and respiratory infections, as well as with cancers of several sites (Barbieri et al., 2017). However, Barbieri et al. (2017) estimate that in 2010 deaths from obesity as the underlying cause in the United States account for only 16% of all deaths from obesity. Thus, it is essential to identify other risk markers for obesity-related deaths other than obesity itself. Barbieri et al. (2017) find that obesity was most strongly associated with cardiovascular diseases, with diabetes and other endocrine diseases playing a major role. Hypertension and other heart diseases were also associated with obesity. Given the complex metabolic process underlying obesity as a cause of death, multiple causes of death should ideally be considered as markers of obesity-related deaths.

2.2.3 ACCIDENTAL DRUG AND ALCOHOL POISONING

There has been a reported increase in deaths due to accidental drug and alcohol poisoning, chronic liver disease, cirrhosis and suicide among middle-aged white Americans (Case and Deaton, 2015). This increase was significant enough to increase the all-cause mortality for white Americans aged 45-54 between 1999-2013. These so-called "deaths of despair" reflected in alcohol abuse and the opioid epidemic, arise from economic insecurity since the early 1970s. This finding has been challenged by Masters et al. (2018), who claim that this increase has been primarily due to deaths from drugs, in particular, opioids, and not alcohol use or suicide. By showing white male and female deaths between 45-54 separately, from 1980 to 2013, they find that the increasing mortality is confined to deaths caused by drug use, being particularly profound for females.

While much of the attention has been to the impact of opioid use on the mortality of white Americans in the 21st century, the longer term trends of the opioid epidemics show complex racial and geographical patterns. Alexander et al. (2018) suggest that the opioid epidemic can be divided into three waves. In the first wave between 1979 and the mid 1990s, the epidemic was mainly driven by heroin with opioid mortality being higher among black Americans. During the second wave from the mid 1990s to 2010, the epidemic expanded rapidly to white Americans and was mainly associated with painkillers. The third and current wave from 2010 affects both black and white Americans and, similarly to the first wave, is associated with heroin although synthetic opioids have gained prominence in the more recent years. Geographically, the opioid epidemic has traditionally been associated with Appalachian and Mid-western states. However, Kiang et al. (2019) find that in recent years opioid-mortality has increased in the Eastern United States.

2.2.4 HYPERTENSION

Hypertension is also widely recognized as an important factor in premature death. In fact, Mokdad et al. (2018) estimate that in 2016 high systolic blood pressure was the third most important risk factor (and the highest metabolic risk factor) associated with deaths in the U.S. Hypertension is associated with heart disease, stroke and cancer, as well as being a cause of death itself (Kung and Xu, 2015). Noticeably, while reductions in population-level systolic blood pressure have had an important contribution to the decline in cardiovascular mortality (Ford et al., 2007), hypertension-related mortality as a whole has in contrast seen a significant increase from 1980 through 2018 (Ayala et al., 2004, Rethy et al. (2020)).

2.2.5 DEMENTIA AND ALZHEIMER'S DISEASE

The number of deaths due to dementia including Alzheimer's disease has rapidly increased in recent years, in contrast to other major causes. The CDC reports that deaths attributed to dementia increased from 30.5 deaths per l00,000 in 2000 to 66.7 in 2017 (Kramarow

and Tejada-Vera, 2019). According to Weuve et al. (2014), this has been due in part to an increased propensity for physicians to list dementia as the underlying cause of death, reflecting improved awareness and diagnosis of dementia. However, the authors also suggest that there is still wide underreporting of dementia. This is confirmed in a clinical study by James et al. (2014), who estimate that there were 503,000 deaths attributable to Alzheimer's disease in 2010, in contrast to official death statistics reporting only 84,000 deaths.

2.2.6 HIV/AIDS

The HIV/AIDS epidemic is also a unique event, where death rates from this infectious, communicable disease rose dramatically in the early 1990s. In the United States, this particularly affected men who have sex with other men and intravenous drug users. Other form of transmission included heterosexual contact and mother-to-child transmission (Centers for Disease Control, 2006). For women, the main paths to infection were through intravenous drug use and heterosexual contact (Ellerbrock et al., 1991). The introduction of anti-retroviral treatments in the early l990s, combined with education and awareness campaigns, has dramatically reduced the incidence and mortality rates of AIDS (Armstrong et al., 1999).

2.2.7 SUICIDE AND HOMICIDE

Homicide and suicide are an important contributor to mortality among younger Americans and, combined, have overtaken traffic accidents as the main external cause of death (Dwyer-Lindgren et al., 2016).

After a number of upswings, current homicide rates in the U.S. are at similar levels to where they were in the 1950s (Rosenfeld and Fox, 2019). During the 1960 and the 1970 there was a sharp rise in homicide rates which was followed by a period of stable homicide rates between 1970 and the early 1990s. However, between 1985 and 1990 there was an up turn in homicide rates which has been attributed to the emerging crack markets and related gang violence (Blumstein et al., 2000) and to changing cohort characteristics (O'Brien and Stockard, 2002). From about 1992 to 2015 homicide rates saw a rapid decrease as a result of several factors including: reactive measures to the homicide increase of the 1980s, changes in the drug market, economic expansion, a decrease in the access to guns, and a decline in domestic violence (Blumstein et al., 2000). Recently, however, there has been an increase in homicide rates in 2015 and 2016. Some preliminary potential explanations for this increase include: strained police-community relations leading to controversial cases of police violence against young black Americans; an increase in drug-related killings among white Americans; and an increase in the availability and lethality of fire arms (Rosenfeld and Fox, 2019).

Suicide rates have also shown important fluctuations over the past 70 years with increases between 1950 through 1980 followed by a drop until the turn of the millennium (Phillips, 2014). More recently, from 1999 through 2018, suicide rates have seen a sharp increase from 10.5 per 100,000 in 1999 to 14.2 in 2018 (Hedegaard et al., 2020). This increase has

been partially linked to an increase in suicide rates among baby boomers and subsequent generations (Phillips, 2014).

2.3 APC MODELING OF MORTALITY TRENDS IN THE U.S.

Age-Period-Cohort (APC) modeling is one technique to identify and understand the driving forces behind mortality change. On the one hand, age is perhaps the clearest marker of mortality patterns with certain causes of death being more prominent at different ages. On the other hand, trends in mortality are the result of changes in environmental and social factors which may manifest themselves as period or cohort effects. However, there are technical complications in disentangling these three effects due to the inherent identification issues stemming from the relation *cohort = period - age* (Bell and Jones, 2013, 2014; Harper, 2015). In spite of these technical challenges, there have been a few studies using the APC approach to understand and explain the evolution of mortality trends for specific causes of death in the U.S. population.

One of the most prominent contributions in this space is the work by Yang (2008) who looks at the APC components for (ischaemic) heart disease, stroke, lung cancer, and breast cancer in the USA during the period from 1960 to 1999. For these four causes of death, Yang (2008) finds a dominance of cohort over period effects in explaining recent trends in mortality reductions, albeit with some differentials in intensity and timings between the causes. For heart disease and stroke, Yang (2008) reports moderate period effects which contrast with strong declines in mortality across the cohort dimension. Notably, while stroke shows consistent decline in mortality for all cohorts born between 1885 and 1975, heart disease shows a slowing decline starting from the late baby boomers. A potential explanation for the difference in trends between these two cardiovascular diseases are cohort trends in cholesterol levels, a risk factor strongly linked to heart disease but not to stroke, and which may be higher among late baby boomer and recent generations who tend to have diets high in animal fat; and at the same time a population wide reduction in mean systolic blood pressure the main risk factor for stroke (Feinleib et al., 1993). For lung cancer, Yang (2008) reports strong cohort patterns resembling the gender specific patterns in smoking prevalence, whereby the smoking take up for women lagged behind the take up among men. However, in contrast to heart disease and stroke, lung cancer does show a consistent period pattern of increasing mortality between 1960 and 1999. Finally, for breast cancer Yang (2008) documents a strong monotonic pattern of mortality decline along the cohort dimension, which is hypothesized to be partially linked to the reduction in fertility rates and the increase in the age of childbearing among younger generations.

In a related follow-up study, Reither et al. (2011) discuss the role of APC modeling in producing mortality projections. Using coronary heart disease as an example and based on the predominant role of cohort effects documented by Yang (2008) and others, Reither et al. (2011) argue that APC-based projections are to be preferred over age period projections as they will better reflect the current health behaviors of those still alive rather than of the

recently deceased. They illustrate this argument with obesity, which is an important risk factor for heart disease (Barbieri et al., 2017) and for which cohort trends have been found to be important, with Americans born after 1955 showing higher chances of being obese (Reither et al., 2009).

Other studies have looked at the role of age-period-cohort components on other more specific and less prevalent causes of death. Phillips (2014) uses an APC model to analyze the epidemiology of suicide rates in the U.S., finding that age, period and cohort effects are all important. Specifically, she finds fluctuating period effects in accordance with fluctuations in economic and social factors such as unemployment and alcohol consumption. More noticeably, Phillips (2014) reports an increase in suicide rates along the cohort dimension starting with the baby boomer generation.

Acosta et al. (2019) use an APC approach to analyze the determinants of influenza mortality trends between 1959 and 2016, finding that both period and cohort factors are important and that they are associated with the virulence of the influenza subtype (e.g. H1N1, H3N2) more prevalent at a given point in time. Specifically, they find important period mortality peaks for the 1968 H3N2 "Hong Kong Flu"-pandemic and the 2003-2004 and 2014-2015 H3N2 seasons, with contrasting period dips during the 1981-1982, 1993-1994 and 2005-2006 flu season where the less virulent H1N1 subtype was predominant. More interestingly, Acosta et al. (2019) report significant "imprinted" cohort effects for the generations of 1947, 1957 and 1968, where a cohort's susceptibility to influenza in later life depends on the strain of the virus prevalent around their time of birth. This is known as the antigenic imprinting hypothesis. For example, the generation born in 1968-1969 during the H3N2 pandemic shows lower influenza mortality relative to the neighboring cohorts due to having a more robust immune response to future H3N2 flu seasons.

Another strand of the literature uses APC modeling to explain differential mortality trends among socioeconomic subgroups. For example, Masters et al. (2012) use APC modeling to study mortality differentials among educational groups finding that much of the widening gap in mortality between lower and higher educated Americans between 1986 and 2006 was driven by cohort factors. In particular, they find that causes with clear behavioral risk factors, such as heart disease and lung cancer, show significant cohort differentials among educational groups while causes less amenable to modifiable health behaviors such as unpreventable cancers have a less steep educational gradient. Similarly, Masters et al. (2014) employ an APC model to study the long-term trend in all-cause and cause-specific mortality for the U.S. black and white population over the 1959 to 2009 period and find clear cohort-based trends in heart disease, stroke, lung cancer, female breast cancer and other cancer mortality, accompanied by especially pronounced period-based reductions in mortality from heart disease, stroke, infectious diseases and homicide. They find that most of the narrowing in the black-white mortality gap is explained by faster cohort reduction in chronic disease mortality among blacks.

More recently, Masters et al. (2018) have applied the APC approach to understand the

drivers of the so-called “deaths of despair”. Case and Deaton (2015) have argued that the recent increase in mortality rates among middle-aged U.S. white men and women with low levels of education was mainly a cohort-effect and that the reversal in the long-term decline in the all-cause mortality rate was largely driven by increases in deaths from suicides, alcohol and drug poisonings, and chronic liver diseases and cirrhosis. By applying APC methods to analyze trends for each of these causes separately for men and women, Masters et al. (2018) have confirmed that deaths for middle-aged whites had increased in the 21st century, but primarily due to rapid increases in drug-related mortality from the late 1990s rather than in suicides or alcohol use; and crucially, that this increase was a period rather than a cohort effect linked to prescribed opioid use.

Section 3: Data

For the purposes of our analysis, we require mortality data which satisfies several criteria, namely; a long time series, underlying cause of death information, and single year of age. Such elements would permit the measurement of historical period effects and the calculation of cohort specific mortality rates. In order to identify a dataset that matches the required criteria, we first provide an overview of the suitability of several well-known public data sources for this task. Following this, we describe the issues surrounding the construction of a consistent time series of mortality rates by cause of death, and how we address this issue in our choice of dataset. Finally we describe the choice of cause groups that will underpin the rest of our analysis.

3.1 OVERVIEW OF AVAILABLE DATA

The Human Mortality Database (Human Mortality Database, 2020) is a widely-used database of mortality and population data. For the United States, it has a long time series of mortality rates by single year of age; however, it does not incorporate cause of death information. The Center for Disease Control (CDC) provides detailed data on mortality rates by cause of death, for a long time series from 1968–2016 through an interactive web service, CDC Wonder (Centers for Disease Control, 2019), but this dataset only provides mortality rates for broad age groupings (between 5 and 10 year bands), which is insufficiently detailed for our purposes. The Human Cause of Death Database (HCDD)[1] also provides mortality data by cause of death at 5-year age bands for the years 1999–2016. Mortality microdata is available from the National Center for Health Statistics (NCHS) which tabulates individual death records by age, year of death, and cause of death for the years 1959–2016. The Human Mortality Database Cause of Death Data (HMD COD) (as distinct from the sources mentioned previously) is a compilation of cause of death mortality data, adapted from the NCHS microdata from 1959–2016. This data is described in Barbieri (2017) and provides mortality rates by cause of death and single year of age.

3.2 PARTICULAR COMPLICATIONS WITH CAUSE OF DEATH DATA

One complication with cause of death data for long time series is with changes in the the classification system and rules for coding underlying cause of death. These are revised at regular intervals in the International Classification of Diseases (ICD) maintained by the World Health Organization (WHO). Table 3.1 shows the years of each ICD regime.

In each regime, the ICD uses a certain 3 digit or 4 digit code to identify a particular underlying cause of death. These codes vary across regimes, which causes inconsistencies across the ICD regime boundaries. Reasons for this variation include simple relabeling of codes;

[1] https://www.causesofdeath.org/

Table 3.1: Years of ICD regimes

Regime	Year
ICD-7	1959–1967
ICD-8	1968–1978
ICD-9	1979–1998
ICD-10	1999–2016

identification of new causes of death, and hence assigning new codes; merging or splitting of codes; and changes in the rules used to select a single underlying cause of death from several causes mentioned in the death certificate. This variation can be corrected in a number of ways.

First, the process of bridge coding can be used to link causes across ICD regimes. This is where medical experts code a sample of deaths in the transition year under both the old and new ICD regimes. The ratio of deaths under the old regime compared to the new regime can be calculated, called a comparability ratio. Since a sample of deaths is coded, it cannot consider every possible cause of death, and there may be certain rare causes which cannot be adequately compared between the two ICD regimes. The CDC has published bridge coding reports since the transition to ICD-8 (Rey et al., 2011). A practical issue with using bridge coding is that the comparability ratios need to be manually extracted from the relevant reports, and matched to the cause of death groupings used in this study.

Second, Rey et al. (2011) outline the method of concordance table and cause recombination. This is a more comprehensive process whereby every possible cause of death code in each regime is mapped to a consistent set of causes. Although this method can take into account the merging and splitting of causes, it still cannot account for changes in the selection of the underlying cause of death.

In light of these limitations, statistical methods can be used, either independently or in conjunction with bridge coding. For example, Rey et al. (2011) develop a statistical methodology to automatically detect jumps, due to data issues, coding changes, or other types of discontinuities. This results in the detection of a wide range of anomalous cases, including coding changes due to a change from manual to automatic coding and secular shifts such as the reunification of Germany. This method is applicable to long time series of mortality data with multiple jumps, for which it might be unfeasible to do manual adjustment using comparability ratios. Changes in the categorization of causes of death are not limited to changes across ICD boundaries. For instance, there were minor changes to the way dementia was coded as an underlying cause of death between 2005 and 2006 and again in 2010 with the release of the revised ICD-10 2010v coding rules (Kramarow and Tejada-Vera, 2019).

In addition, the data we use only considers the underlying cause of death. In reality, individuals often have multiple health conditions interacting with each other, with one cause starting a train of events which leads to the individual's death. This is referred to as the

underlying cause, which may be different to the immediate cause of death (or any contributing causes of death). For instance, the underlying cause may be cancer while the immediate cause may be organ failure. Despite WHO guidance on selection rules when multiple causes are recorded on the death certificate, consistent implementation of these might be difficult, especially when the individual has multiple chronic conditions. This, for example, has been noted as a problem in determining the number of deaths attributed to dementia (Weuve et al., 2014).

3.3 DATASET USED FOR THE ANALYSIS

In light of these complications, we create a bespoke dataset with the assistance of Magali Barbieri from University of Berkeley and the French Institute for Demographic Studies. This dataset stems from work sponsored by the SOA to develop a cause of death extension of the HMD (see Barbieri (2017)). Our dataset consists of cause of death mortality data by single year of age from the HMD COD dataset for ICD regimes 7 and 8 (1959–1978), which is not adjusted for ICD coding changes between ICD-7 and 8. This is joined with a preliminary version of the Human Cause of Death database for ICD regimes 9 and 10 (1979–2016), which is adjusted for ICD coding changes between these two regimes. There are no adjustments made for the discontinuity between ICD regimes 8 and 9.

For the purposes of our analysis, we use a mapping from the ICD 3 or 4 digit code to the 92 subcategories of death, proposed by the authors of the HMD COD. The approach they took was to identify groups of codes that were broadly equivalent in terms of medical content across the ICD coding regimes. This somewhat ameliorates the problem of major discontinuities due to ICD regime changes. We initially group all deaths by these 92 subcategories, which we will refer to as the '92 causes'. The details of this grouping with the 3 digit codes composing each of the 92 subcategories are described in Barbieri (2017, Appendix A).

3.4 CAUSES OF DEATH GROUPING FOR ANALYSIS

We have carefully considered the groupings of causes that we wish to analyze. If the number of groupings chosen is too large, any broad trends or patterns in mortality will be difficult to determine. By contrast, if the number of groupings is too small, the information is not as useful as any heterogeneity in trends within groupings of causes is masked.

Accordingly, for our analysis we consider further aggregation of the 92 causes of death under two levels. A first Level 1 grouping comprising 6 broad groups and a Level 2 more detailed grouping with some of the main subcauses within each Level 1 cause. This two-level grouping of causes is summarized in Table 3.2 where we indicate the mapping of each of our grouping of causes to the 92 causes used by the HMD COD.

The Level 1 grouping allows us to get a first overview of cause-specific trends. The Level 2 grouping decomposes the Level 1 causes into leading causes of death, allowing us to drill down

Table 3.2: Cause of deaths groupings

Level 1	Level 2	HMD Cause groups
Circulatory diseases		
	Ischaemic heart disease	47
	CVD and stroke	51
	Other circulatory system diseases	45, 46, 48, 49, 50, 52, 53, 54, 55
Neoplasms		
	Bowel cancer	13, 14
	Liver cancer	15
	Lung cancer	18
	Breast cancer	20
	Prostate cancer	24
	Other cancers	17, 19, 21, 22, 23, 25, 26, 27, 28, 29, 30, 31, 32
	Other digestive organ cancers	10, 11, 12, 16
Respiratory diseases		
	Influenza and pneumonia	56, 58
	Chronic lower respiratory disease	59, 60, 61
	Other respiratory diseases	57, 62
Digestive system		
	Gastric and duodenal ulcer	63
	Chronic liver disease	65
	Other digestive system diseases	64, 66
External causes		
	Traffic accidents	84
	Self-harm and interpersonal violence	89, 90
	Other external causes	85, 86, 87, 88, 91, 92
Other		
	AIDS and tuberculosis	1, 8
	Diabetes and obesity	34, 35
	Alcohol abuse and drug dependence	37, 38
	Alzheimer's disease	42
	Dementia and other mental disorders	39
	Rest of causes	2, 3, 4, 5, 6, 7, 9, 33, 36, 40, 41, 43, 44, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83

on the trends observed at Level 1 by looking at trends for individual causes which merit a separate analysis and interpretation. For instance, separate trends in ischaemic heart disease and CVD/stroke are easier to interpret than trends from the broader circulatory disease group.

Table 3.3: Risk factors and drivers of mortality change groupings

Risk Factor	**Associated cause of death**	**HMD Cause groups**
AIDS and tuberculosis	AIDS and tuberculosis	1, 8
Alcohol abuse	Alcohol abuse and poisoning, chronic liver disease	37, 65, 86
Dementia and Alzheimer's disease	Dementia and Alzheimer's disease	39, 42, 80
Diabetes and obesity	Diabetes and obesity	34, 35
Drug dependency	Drug dependency	38, 87
Homicide	Homicide	90
Hypertensive disease	Hypertensive disease	48
Self-harm	Self-harm	89
Smoking	Lung cancer	18

3.5 RISK FACTORS AND DRIVERS OF MORTALITY CHANGE

We also identify some key causes that can serve as markers of trends in behavioral risk factors that could drive mortality change. Based on our literature review, we have identified nine factors which we have mapped to causes according to Table 3.3.

Some of the risk factor groupings in Table 3.3 have a direct mapping with the causes defined in our Level 2 grouping in Table 3.2. However, others combine or split some of the Level 2 groupings to align better with risk factors known to cause a large proportion of deaths for that cause. For example, whilst smoking contributes to deaths from heart disease, respiratory diseases and many cancers, about 80% or more of lung cancer deaths are attributable to smoking. Hence lung cancer is a more sensitive marker for tracking smoking behavior. We combine Alzheimer's disease and dementia because they are related causes and often coded interchangeably by the certifying doctor. We split up drug and alcohol abuse into separate categories, in line with the analysis of (Masters et al., 2018). For alcohol abuse, besides the deaths under mental and behavioral disorders (HMD cause group 37), we also include deaths from chronic liver diseases and cirrhosis (HMD cause group 65) and from accidental poisoning from alcohol (HMD cause groups 86). Similarly, for drug dependency besides the deaths under mental and behavioral disorders (HMD cause group 38) we also include deaths from other accidental poisoning (HMD cause group 87). We note that HMD group 87 also includes ICD-10 codes X46-X49 which are not related to drug abuse, but these are small relative to codes X40-44 which account for drug related poisoning. Finally, we consider self-harm and homicide separately as in our preliminary analysis they tend to exhibit different trends across the period and cohort dimensions.

Section 4: Descriptive Analysis

We start our examination of the possible drivers of mortality change in the USA by performing a descriptive analysis of mortality trends using the cause of death grouping identified in Sections 3.4 and 3.5. On the one hand, this helps us identify the main features of the data that would need to be accounted for in the further modeling we do later in Section 5. On the other hand, it gives us an initial understanding of the patterns observed in the different causes of death and their associated risk factors.

We focus first on general time trends for various causes of death and risk factors using age-standardized death rates. Then, we move to the examination of trends in life expectancy with a focus on the contribution of different age groups and causes of death to gains in life expectancy over the 1970–2015 period. Finally, we end this section by presenting mortality improvement rate heatmaps which allow us to make an initial discussion of improvement rate patterns across the dimensions of age, period and cohort.

4.1 AGE-STANDARDIZED DEATH RATE

4.1.1 METHODOLOGY

The age-standardized death rate (ASDR) is a weighted average of age-specific mortality rates at a particular time, which provides an overall view of the mortality of a given cause of death. This weighted average is computed with reference to a given *standard population*, so that differences in the population age structure across time are eliminated. This allows the comparison of overall mortality trends over arbitrary time periods. For our purposes, we use the U.S. gender-specific 2010 census population as our standard (NBER, 2016), grouped by 5 year age bands. We present the details of this standard population in Table A.1 in Appendix A. We note that since we are using gender-specific standard populations, the ASDR trends for men and women are not fully comparable.

The ASDR is calculated as follows: the mortality rate for each age band is weighted by the corresponding population for that age band in the reference population, then summed across age bands. It is then divided by the total reference population to arrive at the ASDR. More concisely:

$$ASDR = \frac{\sum_x m_x E_x}{\sum_x E_x} \times 100,000,$$

where x is each age band, e.g. 0, 1-4, 5-9, ... 100+, m_x is the mortality rate for that age band, and E_x is the reference population for that age band. For ease of reading, we report all our ASDR as rates per 100,000 of standard population.

Table 4.1: Age-standardized death rate for selected years, females, by cause of death

Level 1	Level 2	1959	1990	2016
All-Cause		**1353.03**	**928.31**	**757.73**
Circulatory diseases		**867.84**	**435.07**	**230.53**
	Ischaemic heart disease	377.57	249.59	85.48
	CVD and stroke	220.58	83.97	46.59
	Other circulatory system diseases	269.69	101.51	98.46
Neoplasms		**199.40**	**214.65**	**163.95**
	Bowel cancer	34.33	24.91	14.30
	Liver cancer	9.56	4.91	6.23
	Lung cancer	7.72	43.28	38.03
	Breast cancer	35.98	38.51	23.60
	Prostate cancer	0.00	0.61	0.00
	Other cancers	84.40	81.61	64.02
	Other digestive organ cancers	27.42	20.81	17.78
Respiratory diseases		**61.13**	**75.08**	**76.58**
	Influenza and pneumonia	48.00	28.93	14.86
	Chronic lower respiratory disease	7.52	33.99	45.81
	Other respiratory diseases	5.61	12.16	15.91
Digestive system		**40.17**	**33.34**	**28.27**
	Gastric and duodenal ulcer	4.97	2.86	0.88
	Chronic liver disease	9.42	8.13	8.54
	Other digestive system diseases	25.78	22.34	18.84
External causes		**56.33**	**34.91**	**44.75**
	Traffic accidents	12.05	7.61	3.93
	Self-harm and interpersonal violence	7.98	8.94	8.58
	Other external causes	36.30	18.36	32.24
Other		**128.17**	**135.27**	**213.65**
	AIDS and tuberculosis	4.65	3.29	1.10
	Diabetes and obesity	28.58	25.24	22.63
	Alcohol abuse and drug dependence	0.67	1.53	2.04
	Alzheimer's disease	0.00	11.03	45.08
	Dementia and other mental disorders	1.63	10.54	44.12
	Rest of causes	92.64	83.63	98.68

4.1.2 ASDR TRENDS FOR LEVEL 1 AND LEVEL 2 CAUSES

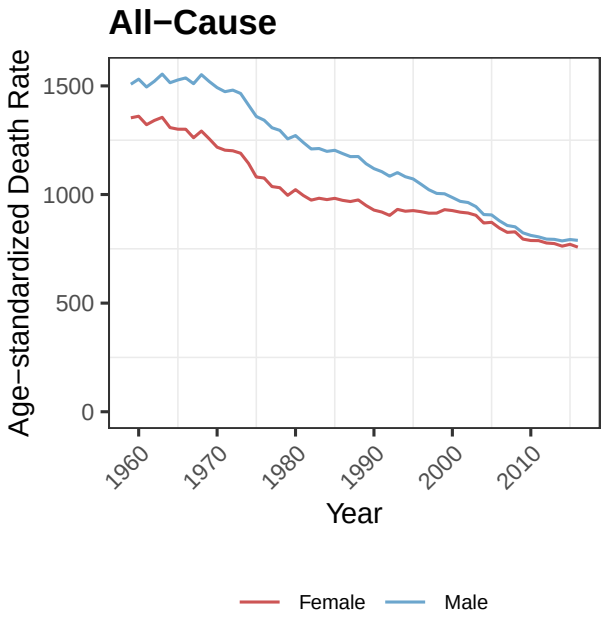
Tables 4.1 and 4.2 show the decomposition of the ASDR by Level 1 and Level 2 causes for selected years for women and men, respectively.

We plot the ASDR by time from 1959–2016 for all-causes in Figure 4.1 and for each grouping of causes in Figures 4.2 to 4.8. These plots are useful for two reasons. First, since the ASDR is a weighted average, these plots allow the concise representation of the overall mortality magnitude and trend for a particular cause of death. Second, we can examine material disruptions caused by changes in the classification used by the International Classification of Diseases (ICD).

Table 4.2: Age-standardized death rate for selected years, males, by cause of death

Level 1	Level 2	1959	1990	2016
All-Cause		**1507.75**	**1119.11**	**788.94**
Circulatory diseases		**882.93**	**465.64**	**237.47**
	Ischaemic heart disease	499.56	300.73	116.42
	CVD and stroke	169.38	64.17	32.86
	Other circulatory system diseases	213.99	100.74	88.19
Neoplasms		**222.60**	**277.86**	**180.04**
	Bowel cancer	30.64	28.68	15.76
	Liver cancer	7.45	6.38	10.90
	Lung cancer	44.02	89.30	44.80
	Breast cancer	0.35	0.83	0.26
	Prostate cancer	25.48	33.56	16.55
	Other cancers	67.11	85.66	64.77
	Other digestive organ cancers	47.57	33.45	27.00
Respiratory diseases		**83.52**	**96.56**	**71.30**
	Influenza and pneumonia	51.21	28.75	13.75
	Chronic lower respiratory disease	22.34	52.32	40.13
	Other respiratory diseases	9.96	15.48	17.43
Digestive system		**55.13**	**39.61**	**32.27**
	Gastric and duodenal ulcer	12.99	3.04	0.93
	Chronic liver disease	18.50	16.78	15.23
	Other digestive system diseases	23.64	19.79	16.12
External causes		**114.11**	**91.17**	**98.00**
	Traffic accidents	36.34	17.72	10.75
	Self-harm and interpersonal violence	28.32	36.01	31.04
	Other external causes	49.46	37.43	56.21
Other		**149.46**	**148.29**	**169.85**
	AIDS and tuberculosis	12.39	19.91	3.00
	Diabetes and obesity	18.57	21.96	27.03
	Alcohol abuse and drug dependence	3.08	5.24	5.82
	Alzheimer's disease	0.00	7.54	18.99
	Dementia and other mental disorders	1.12	6.88	21.29
	Rest of causes	114.30	86.75	93.72

Figure 4.1: Age-standardized death rate for all-causes, 1959–2016

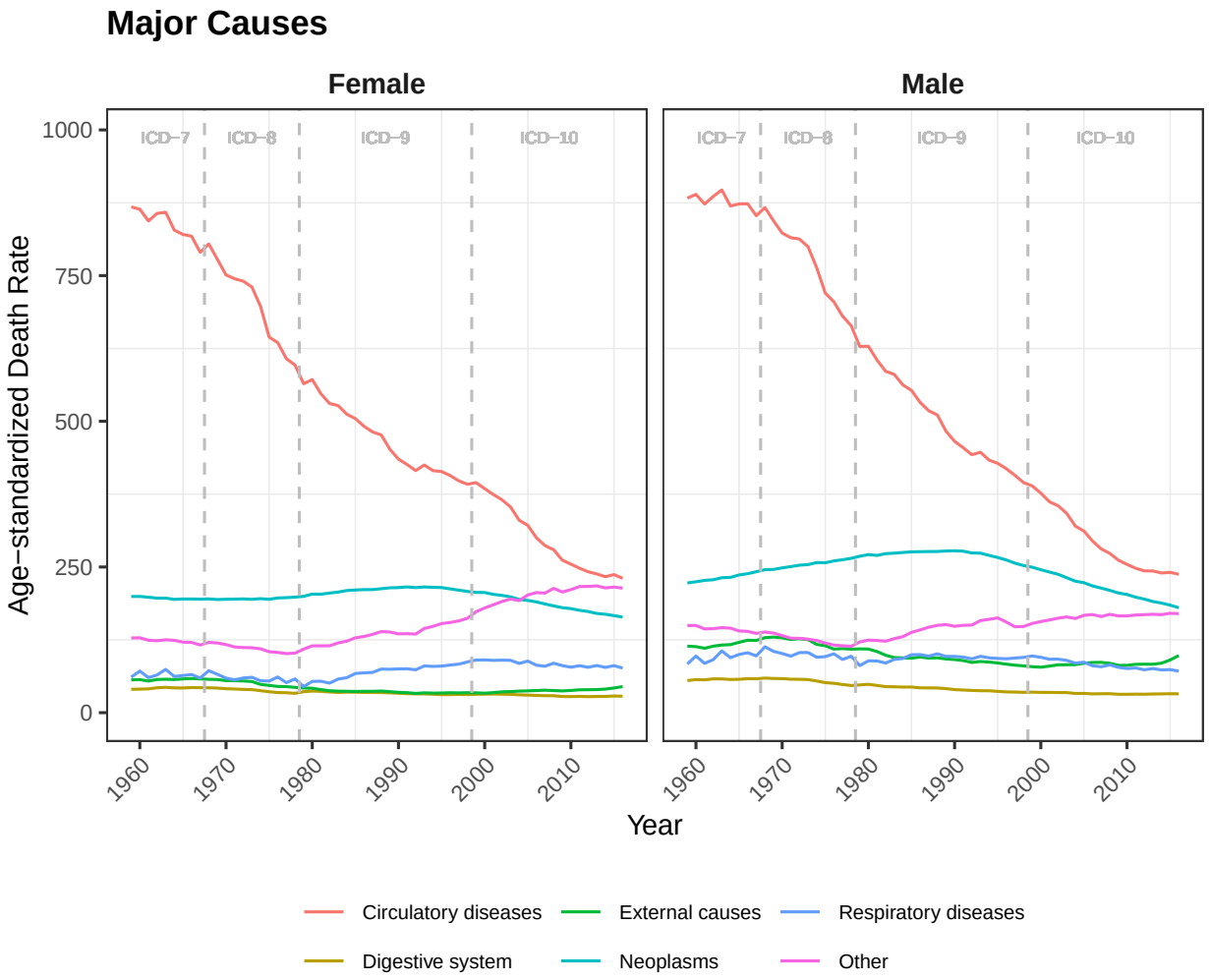


In Figure 4.1 we see that for both genders the ASDR has decreased by almost a half passing from 1508 and 1353 (per 100,000) in year 1959 for males and females, respectively, to 789 and 758 in year 2016. This decrease has been accompanied by an important change in the cause of death composition. While in 1959 circulatory diseases accounted for 59% and 64% of the all-cause ASDR for male and female, respectively, in 2016 circulatory diseases accounted for only 30% for both genders. As a result, neoplasms are starting to replace circulatory disease as the leading cause of death. Neoplasms accounted for just 15% of the all-cause ASDR in 1959, increasing to about 23% in 2016. The "Other" group of Level 1 causes has also increased its importance. This group accounted for just 10% of the all-cause ASDR in 1959, doubling to about 20% in 2016.

Moreover, Figure 4.2 shows that the decline in mortality rates has mainly been driven by large overall decreases in mortality from circulatory diseases and neoplasms (cancer). Overall, the magnitude for various causes of death for females and males are similar, although generally males have higher mortality rates than females. This is particularly true for neoplasms and external causes. For neoplasms, there has been a characteristic "hump-shape" where mortality peaked around 1990 for males and 2000 for females. For "Other" causes of death, there has been a sharp increase for females over the past two decades, with a less pronounced increase for males.

In Figure 4.2, we can also notice some patterns in the data related to ICD coding changes. In particular, respiratory diseases appear more volatile in ICD-7 and 8 compared to ICD-9 and 10 for both genders. In addition, the trend for other causes for both genders changes around the boundary of ICD-8 and 9 reflecting the impact of coding changes.

Figure 4.2: Age-standardized death rate for major causes, 1959–2016



We now turn our attention to the trends in the more detailed Level 2 grouping of causes. In Figure 4.3, we can see that the majority of the decline in mortality in circulatory diseases for both males and females is due to the consistent decline in ischaemic heart disease (IHD). This resulted in IHD comprising a smaller proportion of the ASDR for all-circulatory diseases: in 1959 IHD represented 57% and 44% of the ASDR of circulatory diseases for males and females, respectively, this proportion reduced to 49% and 37% in 2016. Finally, the jump around the year 1967 for IHD and other circulatory system diseases is due to the transition between ICD-7 and ICD-8 (Pechholdová et al., 2017).

For cancers, we see in Figure 4.4 that the magnitude and trends of mortality rates differs markedly for males and females. Nevertheless, there has been a steady decline in mortality rates for most cancers, despite an increase overall in the 1980s for females, and from the 1960s to the 1980s for males. For females, this has been due to the offsetting effect of a decrease in deaths due to other cancers, combined with an increase in deaths due to lung cancer. For

Figure 4.3: Age-standardized death rate for circulatory diseases, 1959–2016

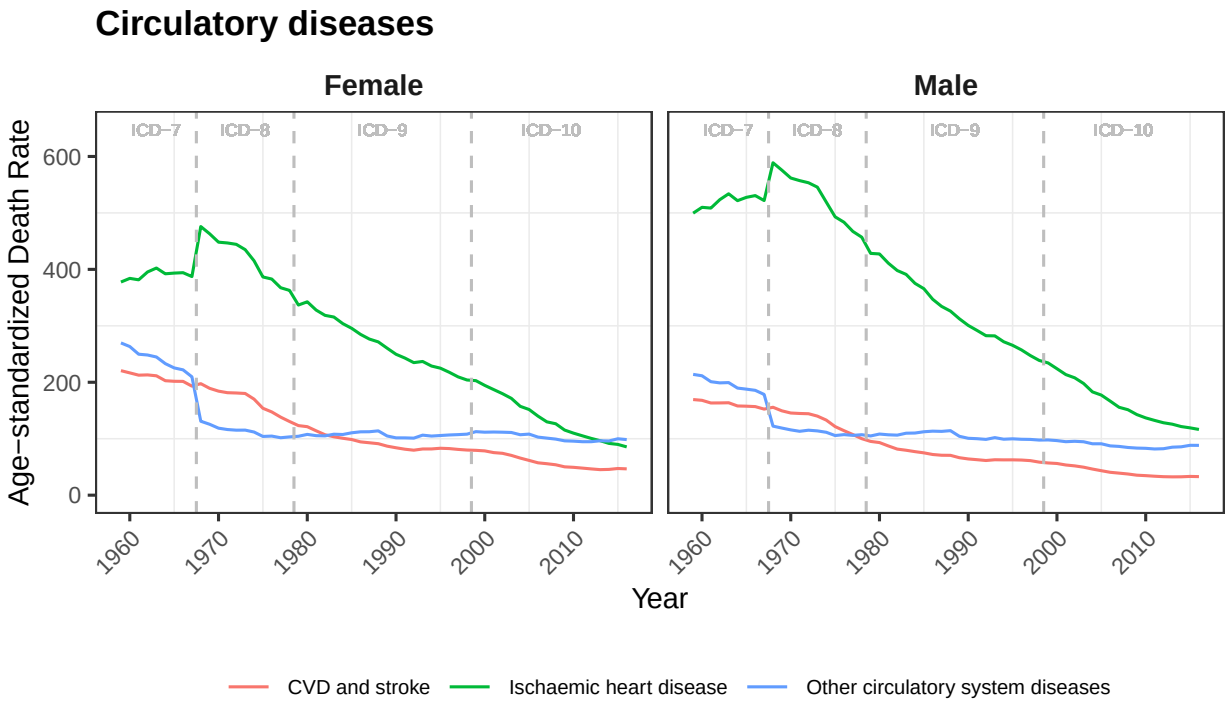


Figure 4.4: Age-standardized death rate for neoplasms, 1959–2016

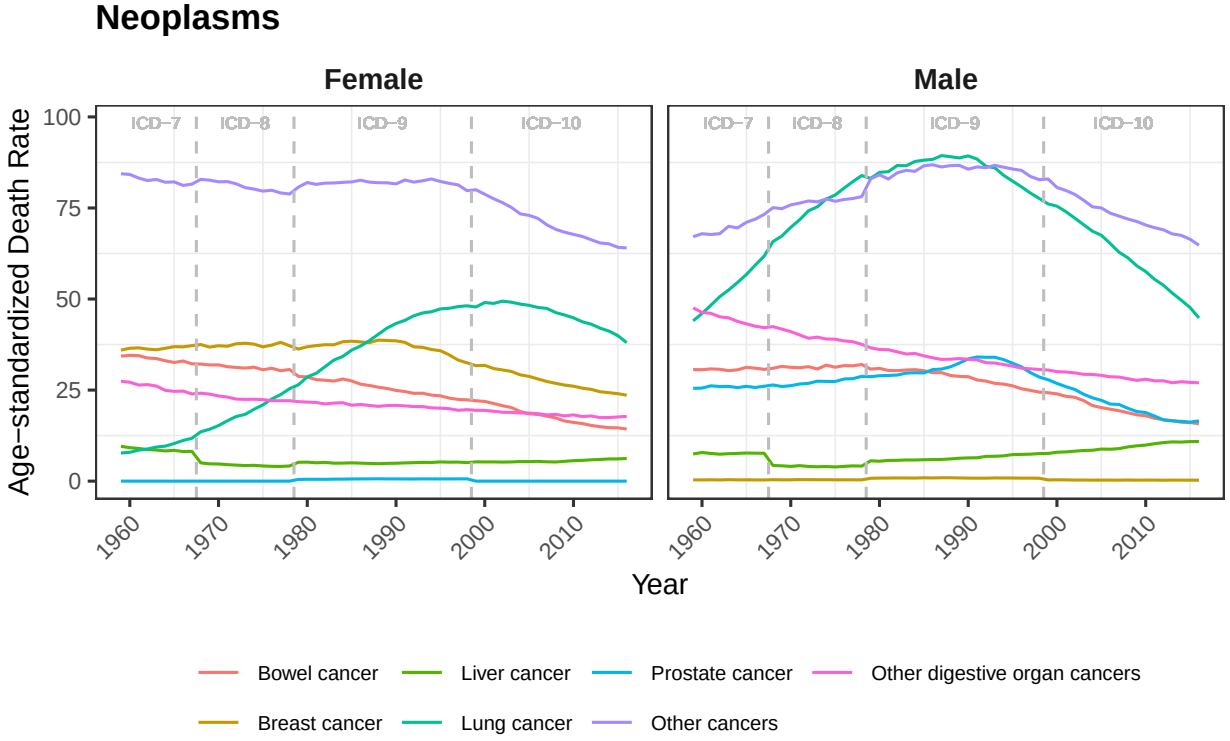
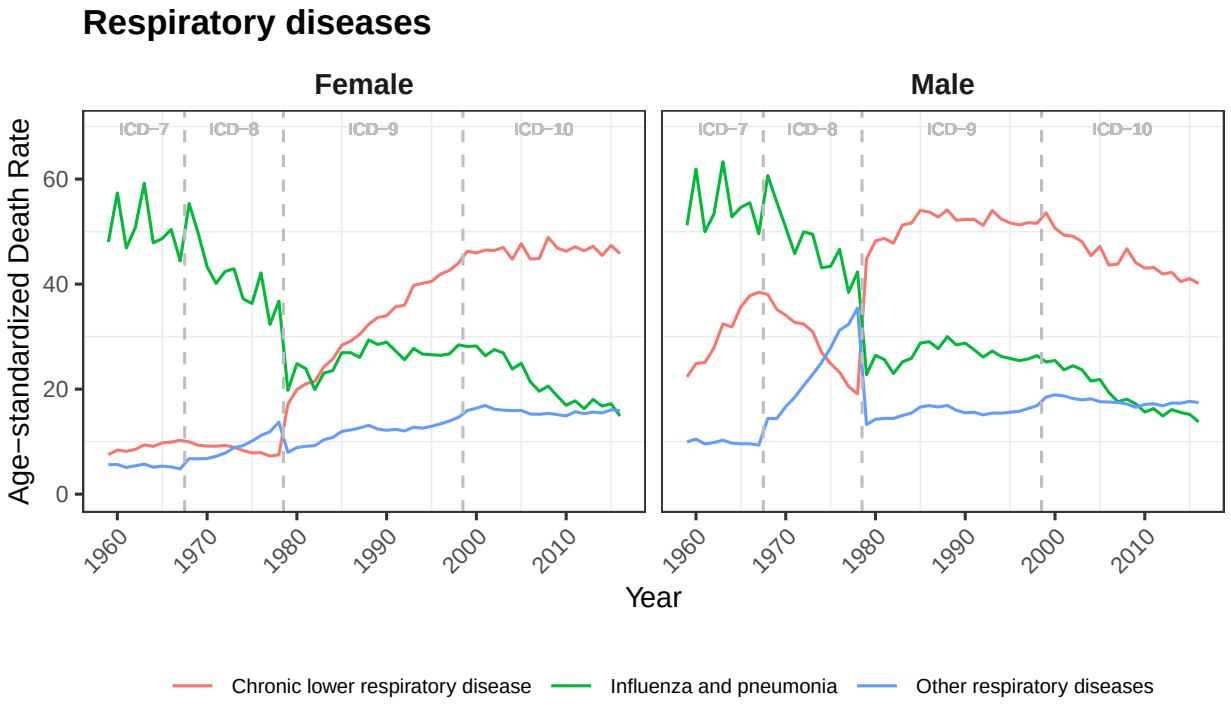


Figure 4.5: Age-standardized death rate for respiratory diseases, 1959–2016



males, deaths due to lung cancer and other cancers peaked in the 1980s, declining thereafter. A similar, smaller effect is noted for prostate cancer in the 1990s.[1] The discontinuity in liver cancer is due to an ICD regime change affecting mainly the ICD-8 period.

For respiratory disease, which are depicted in Figure 4.5, we see broadly similar trends for males and females. The ICD coding change from ICD-8 to ICD-9 has redistributed the deaths among all three subcauses. We also see that the volatility in the ASDR in the periods of ICD-7 and 8 are driven by influenza and pneumonia deaths.

For digestive system diseases Figure 4.6 indicates an overall pattern of gradual decline in mortality for all subcauses. An exception to this downward trend is chronic liver disease which has seen a mortality increase from 2000 for both genders. For other digestive system diseases we see a clear break in the trend occurring at the transition from ICD-8 to ICD-9 in 1979. The change in the trend for gastric and duodenal ulcer is also likely to indicate an ICD regime change between ICD-8 and ICD-9.

For external causes, mortality rates have increased in the past decade. Figure 4.7 indicates that this increase has been mainly due to the increase in mortality due to other external causes, which has increased at almost twice the rate for males compared to females. The mortality rate due to self-harm and interpersonal violence has also increased in the last

[1]The small but non-zero rates for "female prostate cancer" and "male breast cancer" in Tables 4.2 and 4.1 are a data error in the database available to us.

Figure 4.6: Age-standardized death rate for diseases of the digestive system, 1959–2016

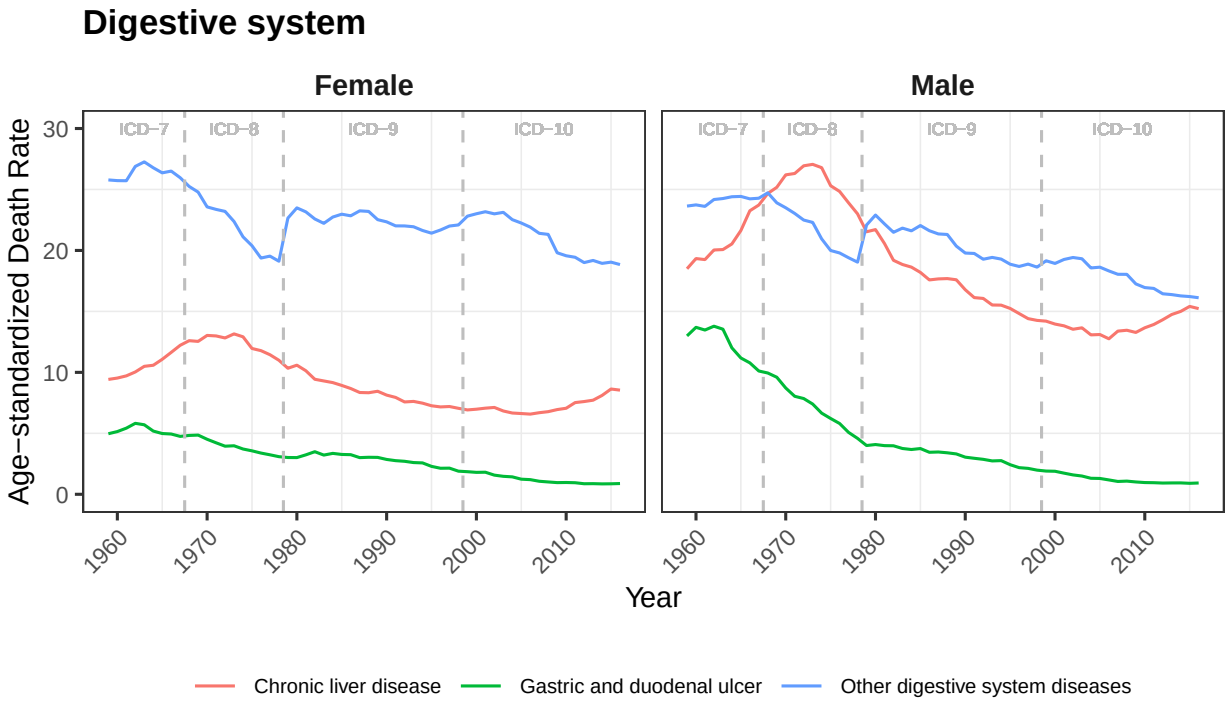


Figure 4.7: Age-standardized death rate for external causes, 1959–2016

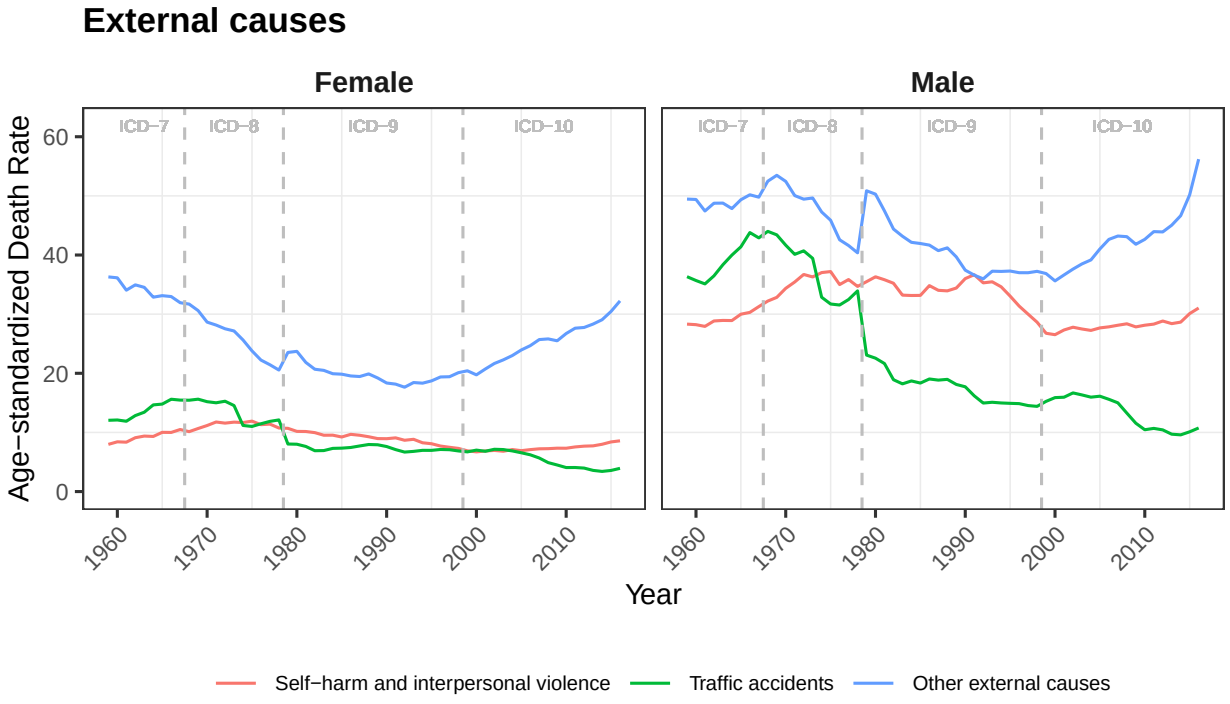
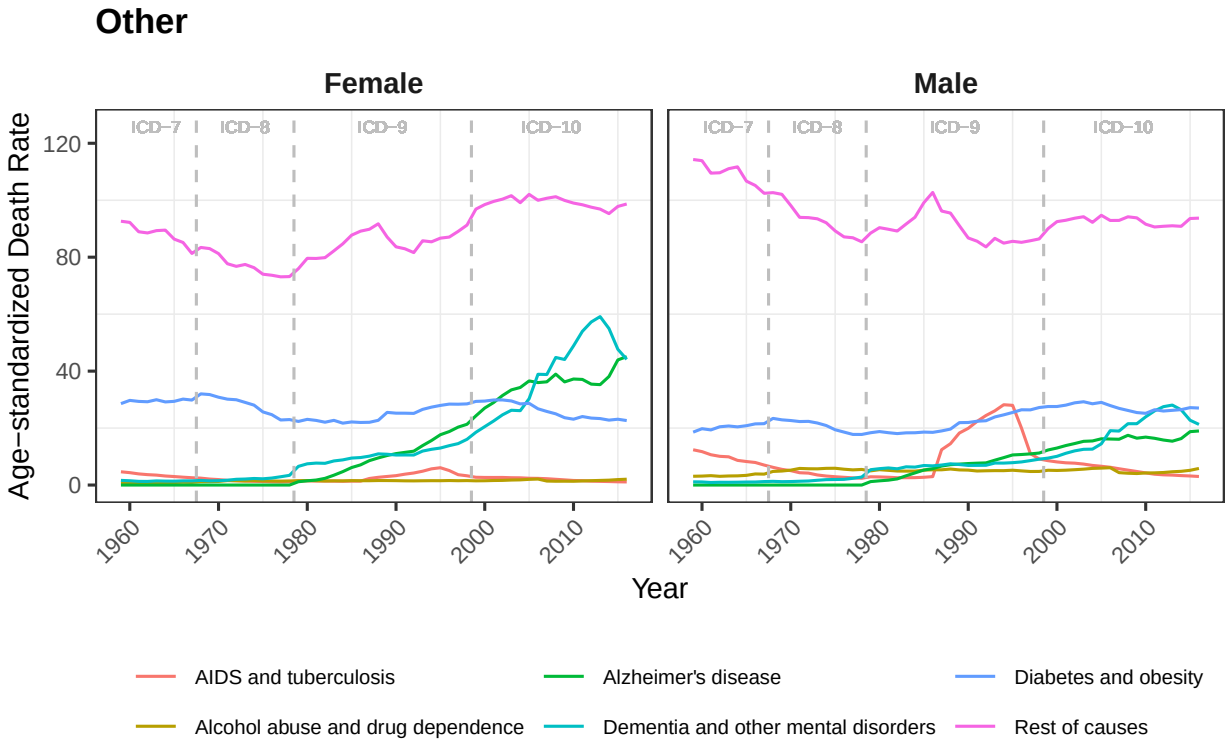


Figure 4.8: Age-standardized death rate for other causes, 1959–2016



decade for both males and females.

In the "Other" category, we outline only key causes of interest seen in Figure 4.8. The HIV/AIDS epidemic has affected males more heavily than females, and the introduction of anti-retroviral treatments has led to the sharp decrease in mortality rates. We also see two causes of death with sharp increases – Alzheimer's disease and dementia and other mental disorders. Diabetes and obesity has remained relatively flat over the entire period, except for a sharp increase in mortality rates in the 1980s and 1990s.

4.1.3 ASDR TRENDS FOR SELECTED RISK FACTORS

We now focus on ASDR trends for selected drivers of mortality change as depicted in Figure 4.9. The corresponding ASDR values for selected years are shown in Tables 4.3 and 4.4. These drivers of mortality change only act as proxies for selected risk factors, and are likely to underestimate the mortality attributable, or linked to, a particular risk factor. Thus, it is important to focus on the general trends rather than on the absolute values of the ASDRs.

In brief, the main patterns we find are as follows:

- As discussed before, AIDS and tuberculosis mortality rates show an important spike in the late 1980s and early 1990s with a significant and steady decrease from 2000

Figure 4.9: Age-standardized death rate for selected risk factors, 1959–2016

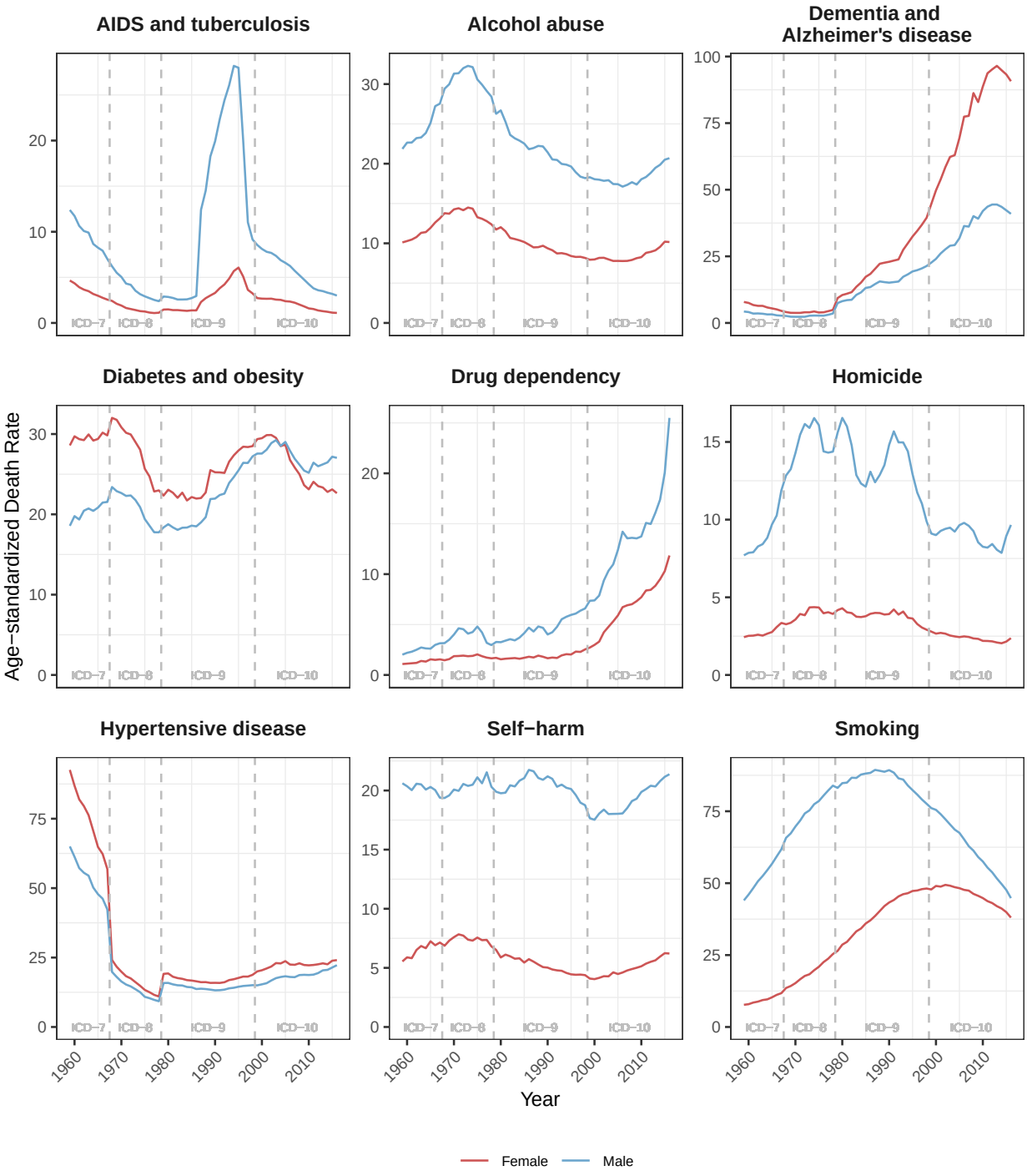


Table 4.3: Age-standardized death rate for selected years, females, for selected risk factors

Risk Factor	1959	1990	2016
AIDS and tuberculosis	4.65	3.29	1.10
Alcohol abuse	10.11	9.37	10.16
Dementia and Alzheimer's disease	7.87	22.95	90.73
Diabetes and obesity	28.58	25.24	22.63
Drug dependency	1.08	1.66	11.85
Homicide	2.44	3.92	2.37
Hypertensive disease	92.57	15.90	24.09
Self-harm	5.55	5.02	6.21
Smoking	7.72	43.28	38.03

Table 4.4: Age-standardized death rate for selected years, males, for selected risk factors

Risk Factor	1959	1990	2016
AIDS and tuberculosis	12.39	19.91	3.00
Alcohol abuse	21.86	21.43	20.69
Dementia and Alzheimer's disease	4.29	15.12	40.96
Diabetes and obesity	18.57	21.96	27.03
Drug dependency	2.01	4.02	25.50
Homicide	7.70	14.81	9.66
Hypertensive disease	65.02	13.20	22.25
Self-harm	20.61	21.20	21.38
Smoking	44.02	89.30	44.80

onwards.

- There has been an increase in mortality due to alcohol abuse, drug dependency and self-harm since around early 2000s for both males and females. The trend for drug dependency is particularly noteworthy, as it can be seen from Tables 4.4 and 4.3 that it has increased substantially from a very low base before 1990.
- Mortality rates due to Dementia and Alzheimer's disease have seen a significant increase since the 1980s albeit with a slight reverse in trend since around 2013.
- Mortality rates from diabetes and obesity are driven largely by diabetes. This has increased in the 1980s for both male and females, and continuing in the 1990s for males, though recently post-2010 it has declined or remained flat.
- Homicide rates are quite volatile and do not seem to exhibit a clear pattern for males; for females the pattern is broadly flat.
- After an important decrease between 1960 and 1980, there has been an increase in hypertensive disease mortality rates since the 1990s, for both males and females. It is noticeable that hypertensive disease together with diabetes and obesity are the only

two risk factors where women have a higher ASDR than men. It also worth noting that there is some impact of coding changes for hypertensive disease affecting mainly the ICD-8 period.

- Mortality rates due to smoking have declined since 1990s for males and since 2000 for females.

4.2 LIFE EXPECTANCY AT AGE 20 AND DECOMPOSITION OF CHANGES

We next perform an analysis of trends in period life expectancy at age 20 and decompose changes across three dimensions: by age group, by time period, and cause of death. This has been achieved using life expectancy decomposition methods borrowed from demography. Such methods have the advantage of enabling a simple and easy to communicate graphical representation of the contribution of each age group and cause of death to gains in life expectancy over a certain period of time.

We note that, as opposed to our previous analysis of ASDR trend which encompassed the entire age range, our life expectancy calculations start at age 20. This is to avoid the possible complications that infant and child mortality may bring to our analysis.

4.2.1 METHODOLOGY

Life expectancy decomposition analysis was pioneered by Arriaga (1984), among others, who decomposed changes in life expectancy by age band across several points in time. He was thus able to link improvements in life expectancy in the general population to particular improvements occurring in certain ages and decades. This has been extended by Shkolnikov et al. (2003) who further segment such improvements by causes of death.

As Andreev et al. (2002) note, one disadvantage of Arriaga's approach lies in the order of decomposition. When decomposing by time period, Arriaga measured improvements in life expectancy with respect to the initial time period. However, the resulting decomposition changes if the measurement is done with respect to the final time period. In order to avoid such inconsistency, we apply the averaging formula found in Andreev et al. (2002) to provide a so-called "symmetrical" decomposition. This approach is in line with much of the demographic literature (see, e.g. Shkolnikov et al. (2013)).

Another issue that Andreev et al. (2002) highlight is the problem of "path dependence". That is, the decomposition from, say, year x to $x+n$ could be computed directly, or as the sum of decompositions from x to $x+1$, $x+1$ to $x+2$, $\ldots$, $x+n-1$ to $x+n$. The difference between these two approaches is usually small, but is not guaranteed to be so in all cases. In our case, we use the formulae given in Andreev et al. (2002) and Shkolnikov et al. (2003) to decompose the life expectancy by 5-year age band, by 92 causes of death, and single calendar

Figure 4.10: Period life expectancy at age 20, 1959–2016

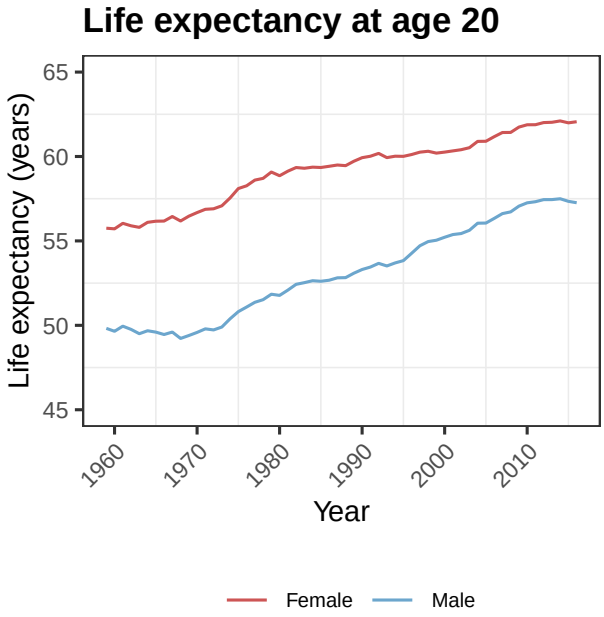


Table 4.5: Period life expectancy at age 20 for selected years

Gender	1970	1985	2000	2015
Female	56.68	59.35	60.26	61.99
Male	49.58	52.61	55.22	57.34

year, and then aggregate to appropriate time periods, in order to minimize the effect of path dependency. We describe the details of our decomposition methodology in Appendix B.

4.2.2 RESULTS

In Figure 4.10 we see that period life expectancy at age 20 in the United States increased significantly for both genders during the period 1959–2016 with the majority of this increase occurring post 1970. As indicated in Table 4.5, life expectancy at age 20 moved from 56.68 years for females and 49.58 years for males in 1970 to 61.99 and and 57.34 years in 2015, for an increase of 5.31 and 7.76 years for females and males, respectively. In what follows, we consider the contribution of different ages and causes to these changes in life expectancy.

We first consider the life expectancy decomposition for ages 20 and above over varying 15-year intervals, by Level 1 and Level 2 causes of death. This is shown in Figure 4.11 as well as in Tables 4.6 and 4.7.

Positive numbers represent contributions (in years) to gains in life expectancy relative to the base year; and conversely, negative numbers represent relative losses in life expectancy. We

Figure 4.11: Life expectancy decomposition for major causes

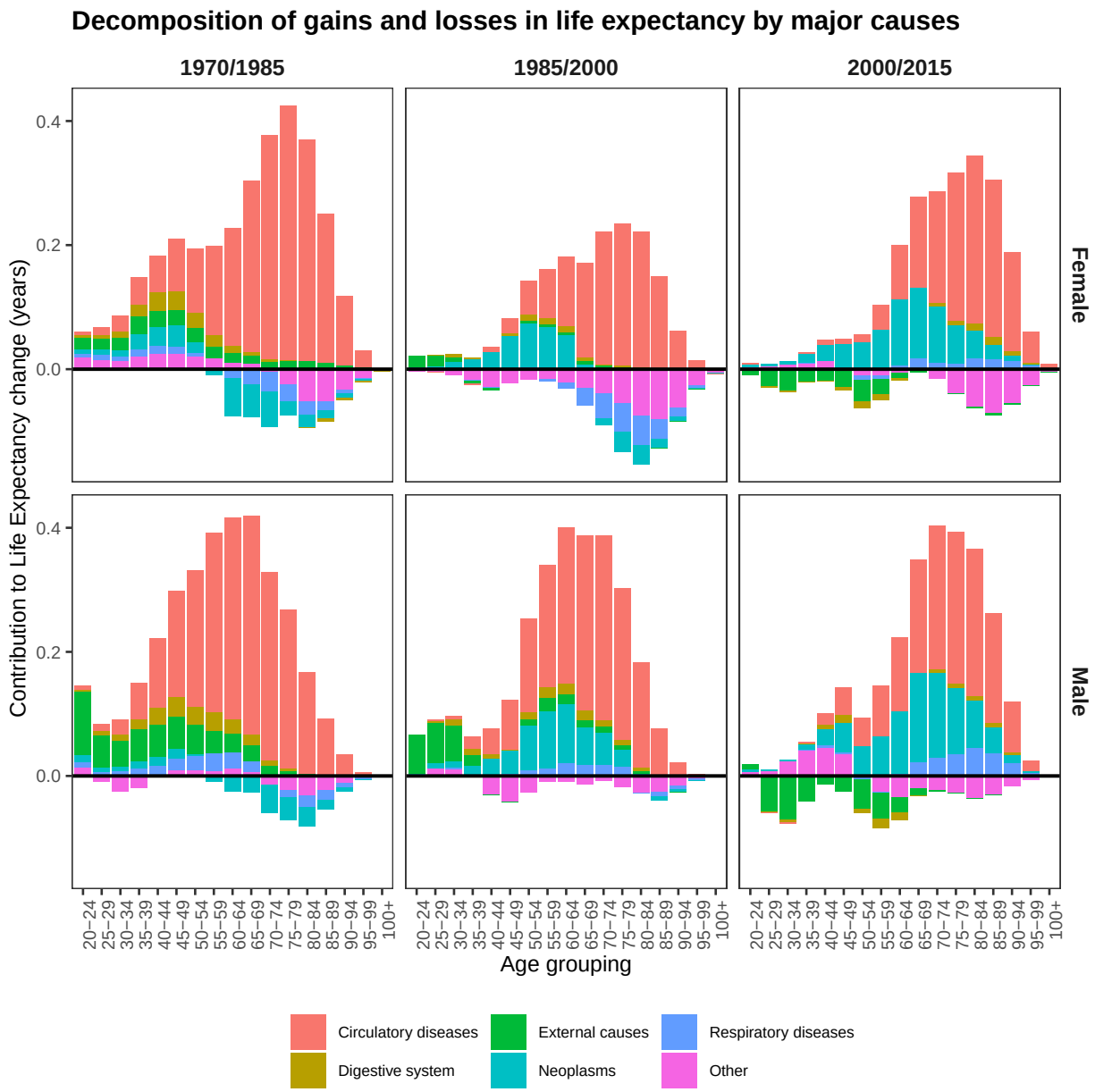


Table 4.6: Decomposition of gains and losses in life expectancy at age 20 by causes of death, over selected time intervals. Females

Level 1	Level 2	1970-1985	1985-2000	2000-2015	Total
All-Cause		**2.67**	**0.91**	**1.73**	**5.31**
Circulatory diseases		**2.46**	**1.31**	**1.47**	**5.25**
	Ischaemic heart disease	1.53	1.07	1.05	3.65
	CVD and stroke	0.81	0.21	0.31	1.33
	Other circulatory system diseases	0.12	0.04	0.12	0.27
Neoplasms		**-0.12**	**0.21**	**0.65**	**0.74**
	Bowel cancer	0.07	0.08	0.09	0.24
	Liver cancer	0.00	0.00	-0.01	-0.02
	Lung cancer	-0.30	-0.14	0.18	-0.25
	Breast cancer	0.00	0.14	0.13	0.27
	Prostate cancer	-0.01	0.01	0.00	0.00
	Other cancers	0.08	0.10	0.24	0.42
	Other digestive organ cancers	0.03	0.03	0.02	0.09
Respiratory diseases		**-0.07**	**-0.23**	**0.08**	**-0.22**
	Influenza and pneumonia	0.18	-0.01	0.08	0.24
	Chronic lower respiratory disease	-0.22	-0.18	0.00	-0.39
	Other respiratory diseases	-0.03	-0.04	0.00	-0.07
Digestive system		**0.15**	**0.06**	**0.00**	**0.22**
	Gastric and duodenal ulcer	0.02	0.02	0.01	0.05
	Chronic liver disease	0.10	0.04	-0.04	0.10
	Other digestive system diseases	0.04	0.01	0.03	0.07
External causes		**0.26**	**0.06**	**-0.22**	**0.10**
	Traffic accidents	0.14	0.01	0.06	0.21
	Self-harm and interpersonal violence	0.05	0.06	-0.04	0.08
	Other external causes	0.06	-0.01	-0.25	-0.19
Other		**-0.01**	**-0.50**	**-0.25**	**-0.77**
	AIDS and tuberculosis	0.00	-0.04	0.05	0.01
	Diabetes and obesity	0.12	-0.09	0.08	0.10
	Alcohol abuse and drug dependence	-0.01	0.00	-0.01	-0.01
	Alzheimer's disease	-0.05	-0.14	-0.13	-0.33
	Dementia and other mental disorders	-0.05	-0.07	-0.20	-0.32
	Rest of causes	-0.02	-0.16	-0.03	-0.21

Table 4.7: Decomposition of gains and losses in life expectancy at age 20 by causes of death, over selected time intervals. Males

Level 1	Level 2	1970-1985	1985-2000	2000-2015	Total
All-Cause		**3.03**	**2.61**	**2.13**	**7.76**
Circulatory diseases		**2.42**	**1.84**	**1.48**	**5.75**
	Ischaemic heart disease	1.85	1.49	1.20	4.54
	CVD and stroke	0.53	0.17	0.23	0.93
	Other circulatory system diseases	0.05	0.18	0.05	0.29
Neoplasms		**-0.13**	**0.49**	**0.82**	**1.18**
	Bowel cancer	0.02	0.07	0.09	0.18
	Liver cancer	-0.02	-0.03	-0.04	-0.09
	Lung cancer	-0.12	0.21	0.41	0.50
	Breast cancer	0.00	0.01	0.00	0.00
	Prostate cancer	-0.02	0.05	0.11	0.14
	Other cancers	-0.05	0.12	0.21	0.28
	Other digestive organ cancers	0.06	0.06	0.04	0.16
Respiratory diseases		**0.11**	**0.08**	**0.20**	**0.39**
	Influenza and pneumonia	0.20	0.03	0.09	0.32
	Chronic lower respiratory disease	-0.13	0.06	0.10	0.03
	Other respiratory diseases	0.04	-0.01	0.01	0.04
Digestive system		**0.21**	**0.12**	**0.01**	**0.34**
	Gastric and duodenal ulcer	0.05	0.02	0.01	0.08
	Chronic liver disease	0.13	0.07	-0.02	0.17
	Other digestive system diseases	0.03	0.04	0.03	0.09
External causes		**0.52**	**0.28**	**-0.33**	**0.47**
	Traffic accidents	0.39	0.05	0.11	0.54
	Self-harm and interpersonal violence	0.03	0.13	-0.09	0.08
	Other external causes	0.10	0.09	-0.35	-0.15
Other		**-0.10**	**-0.21**	**-0.06**	**-0.37**
	AIDS and tuberculosis	0.01	-0.10	0.12	0.03
	Diabetes and obesity	0.04	-0.10	-0.01	-0.07
	Alcohol abuse and drug dependence	0.00	0.01	0.00	0.01
	Alzheimer's disease	-0.03	-0.04	-0.05	-0.12
	Dementia and other mental disorders	-0.03	-0.02	-0.10	-0.15
	Rest of causes	-0.09	0.04	-0.03	-0.08

Table 4.8: Decomposition of gains and losses in life expectancy at age 20 between 1970 and 2015 by causes of death, for selected age bands. Females

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**0.58**	**1.46**	**2.65**	**0.62**	**5.31**
Circulatory diseases		**0.17**	**0.94**	**3.06**	**1.08**	**5.25**
	Ischaemic heart disease	0.06	0.64	2.16	0.78	3.65
	CVD and stroke	0.07	0.22	0.75	0.29	1.33
	Other circulatory system diseases	0.04	0.08	0.15	0.00	0.27
Neoplasms		**0.19**	**0.48**	**0.09**	**-0.02**	**0.74**
	Bowel cancer	0.01	0.07	0.14	0.02	0.24
	Liver cancer	0.00	-0.01	-0.01	0.00	-0.02
	Lung cancer	0.02	-0.03	-0.21	-0.03	-0.25
	Breast cancer	0.06	0.16	0.05	0.00	0.27
	Prostate cancer	0.00	0.00	0.00	0.00	0.00
	Other cancers	0.11	0.24	0.08	-0.01	0.42
	Other digestive organ cancers	0.01	0.04	0.03	0.00	0.09
Respiratory diseases		**0.05**	**-0.01**	**-0.22**	**-0.04**	**-0.22**
	Influenza and pneumonia	0.04	0.05	0.10	0.06	0.24
	Chronic lower respiratory disease	0.01	-0.06	-0.26	-0.08	-0.39
	Other respiratory diseases	0.01	-0.01	-0.05	-0.02	-0.07
Digestive system		**0.07**	**0.09**	**0.05**	**0.01**	**0.22**
	Gastric and duodenal ulcer	0.00	0.01	0.02	0.01	0.05
	Chronic liver disease	0.05	0.05	0.00	0.00	0.10
	Other digestive system diseases	0.02	0.02	0.03	0.00	0.07
External causes		**0.04**	**0.00**	**0.05**	**0.01**	**0.10**
	Traffic accidents	0.12	0.06	0.03	0.00	0.21
	Self-harm and interpersonal violence	0.05	0.01	0.01	0.00	0.08
	Other external causes	-0.14	-0.07	0.02	0.00	-0.19
Other		**0.06**	**-0.03**	**-0.38**	**-0.42**	**-0.77**
	AIDS and tuberculosis	0.00	0.00	0.01	0.00	0.01
	Diabetes and obesity	0.00	0.02	0.08	0.01	0.10
	Alcohol abuse and drug dependence	0.00	-0.01	0.00	0.00	-0.01
	Alzheimer's disease	0.00	-0.01	-0.15	-0.17	-0.33
	Dementia and other mental disorders	0.00	-0.01	-0.14	-0.18	-0.32
	Rest of causes	0.07	-0.02	-0.17	-0.08	-0.21

Table 4.9: Decomposition of gains and losses in life expectancy at age 20 between 1970 and 2015 by causes of death, for selected age bands. Males

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**1.01**	**2.80**	**3.53**	**0.43**	**7.76**
Circulatory diseases		**0.30**	**1.98**	**2.97**	**0.50**	**5.75**
	Ischaemic heart disease	0.24	1.70	2.23	0.37	4.54
	CVD and stroke	0.05	0.21	0.54	0.12	0.93
	Other circulatory system diseases	0.01	0.07	0.20	0.01	0.29
Neoplasms		**0.15**	**0.54**	**0.46**	**0.02**	**1.18**
	Bowel cancer	0.00	0.05	0.11	0.01	0.18
	Liver cancer	0.00	-0.05	-0.03	0.00	-0.09
	Lung cancer	0.05	0.29	0.16	0.00	0.50
	Breast cancer	0.00	0.00	0.00	0.00	0.00
	Prostate cancer	0.00	0.02	0.11	0.02	0.14
	Other cancers	0.09	0.15	0.05	-0.01	0.28
	Other digestive organ cancers	0.01	0.08	0.06	0.01	0.16
Respiratory diseases		**0.06**	**0.14**	**0.17**	**0.02**	**0.39**
	Influenza and pneumonia	0.05	0.09	0.14	0.05	0.32
	Chronic lower respiratory disease	0.00	0.03	0.01	-0.01	0.03
	Other respiratory diseases	0.01	0.02	0.01	-0.01	0.04
Digestive system		**0.10**	**0.14**	**0.09**	**0.01**	**0.34**
	Gastric and duodenal ulcer	0.01	0.03	0.03	0.00	0.08
	Chronic liver disease	0.07	0.09	0.02	0.00	0.17
	Other digestive system diseases	0.03	0.02	0.04	0.01	0.09
External causes		**0.33**	**0.07**	**0.07**	**0.00**	**0.47**
	Traffic accidents	0.41	0.10	0.04	0.00	0.54
	Self-harm and interpersonal violence	0.03	0.03	0.02	0.00	0.08
	Other external causes	-0.11	-0.05	0.01	0.00	-0.15
Other		**0.07**	**-0.08**	**-0.23**	**-0.13**	**-0.37**
	AIDS and tuberculosis	0.01	0.01	0.01	0.00	0.03
	Diabetes and obesity	-0.01	-0.04	-0.01	0.00	-0.07
	Alcohol abuse and drug dependence	0.02	0.00	0.00	0.00	0.01
	Alzheimer's disease	0.00	0.00	-0.07	-0.05	-0.12
	Dementia and other mental disorders	0.00	-0.01	-0.08	-0.06	-0.15
	Rest of causes	0.06	-0.03	-0.08	-0.03	-0.08

can immediately see that the large increase in life expectancy through the whole period of analysis is due to the reduction in deaths due to cardiovascular diseases in general and in ischaemic heart diseases in particular. For males, for example, 5.75 of the 7.76 years of the increase in life expectancy between 1970 and 2015 was due to circulatory diseases.

Medical improvements in treating cancers have also contributed to a significant improvement in life expectancy, especially in recent years. By contrast, "Other" causes of death have contributed to a decline in life expectancy for both genders. The negative contribution of Alzheimer's disease and Dementia and other mental disorders for females is also particularly noteworthy.

We now consider the decomposition of life expectancy gains across different age groups from 1970 to 2015, shown in Tables 4.8 and 4.9. More detailed decompositions across age groups for the subperiods 1970-1985, 1985-2000 and 2000-2015 are shown in Appendix C.

For 20-44 year olds, the improvements are fairly evenly distributed among Level 1 causes. For males, it is noteworthy that out of the 1.01 years attributed to the 20-44 age range over the 1970-2015 period, 0.21 and 0.27 years correspond, respectively, to gains in circulatory diseases and traffic accidents in the 1970-1985 period (see Table C.2). It is also noteworthy that other external causes have a negative contribution at ages 20-44 for both sexes with most of this loss attributed to changes in the 2000-2015 period (see Tables C.5 and C.6 and right panels in Figure 4.11).

For 45-64 year olds, the magnitude of improvement is greater than for the youngest age band. The improvement is mostly driven by cardiovascular disease for both males and females in the period 1970-1985 which contribute 0.52 and 1 years for females and males (see Tables C.1 and C.2), respectively, out of the 1.46 and 2.8 years attributed to the 45-64 age group in the 1970-2015 period. There is a moderate positive contribution due to neoplasms after 1985 for both genders. Again, there is a noticeable negative contribution for external causes for males in the most recent period.

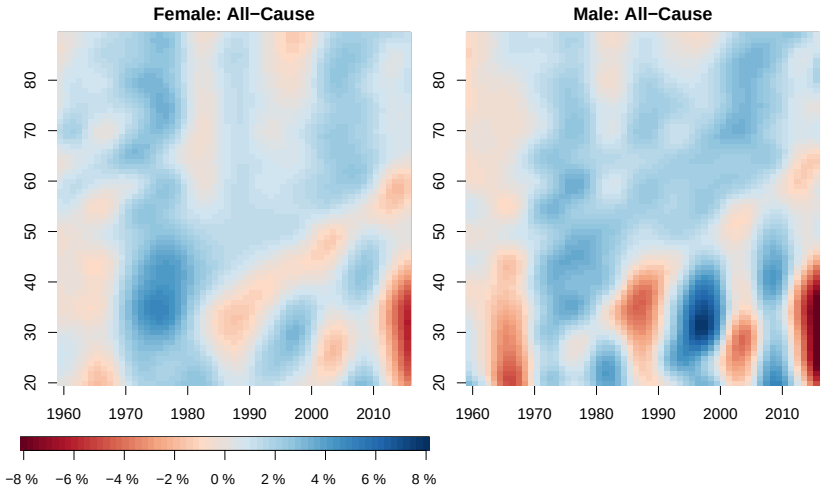
For 65-84 year olds, similarly, the highest positive contribution is from cardiovascular disease, followed by neoplasms, particularly in the most recent periods. Here, the significant negative contributions occur for females between 1970 and 2000, driven by a wide variety of causes. For males, the negative contributions are limited to the period of 1970-1985, for neoplasms.

For the highest age band, 85+, the magnitude of changes is comparatively smaller. The dominating cause is cardiovascular disease, with negative contributions particularly for females in the most recent period due to "Other" causes of death including Alzheimer's and dementia.

4.3 HEATMAPS

We next turn to an initial separation of age, period and cohort effects in the mortality data. We can show this through the use of color-coded rates of mortality improvement

Figure 4.12: Heatmaps for all-causes of death combined, ages 20–89, years 1959–2016



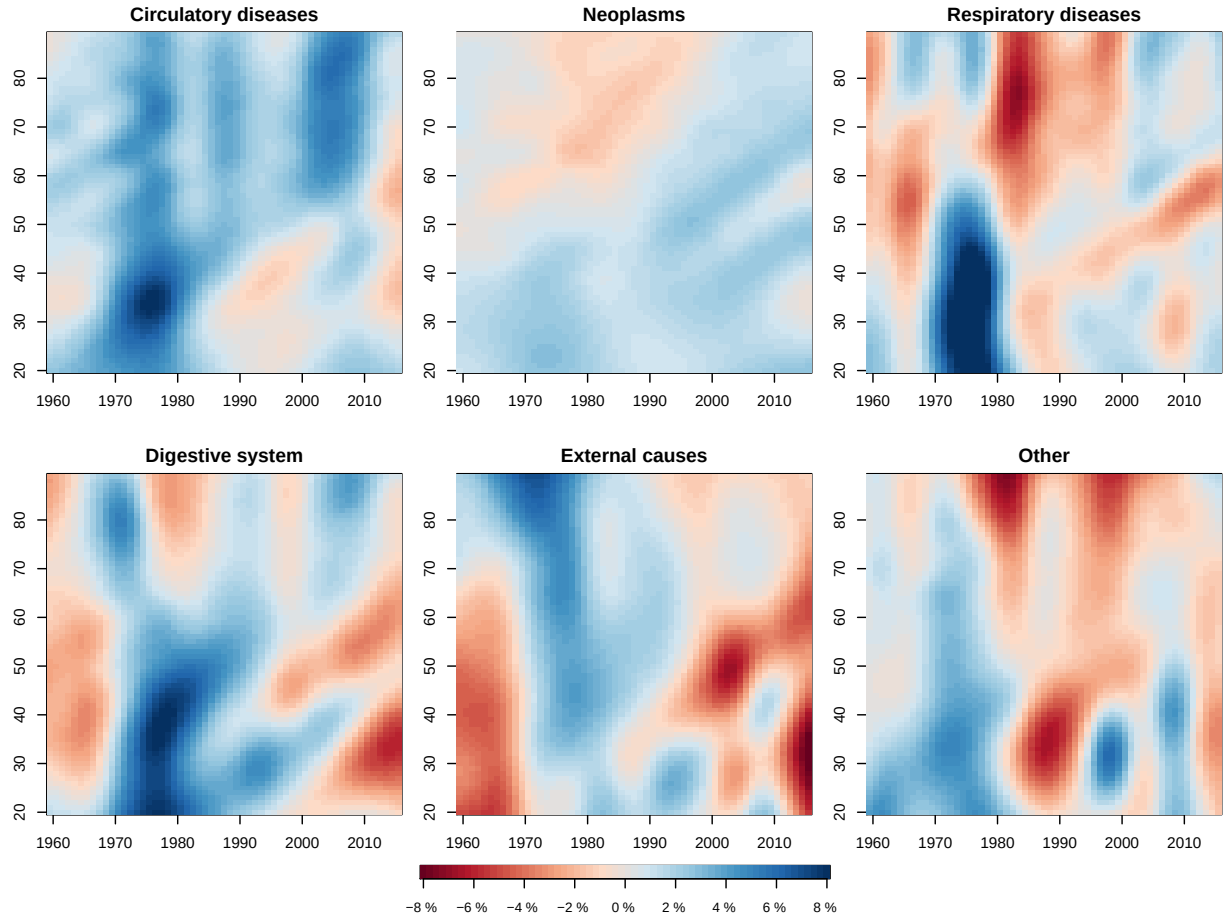
surface plots as developed by Rau et al. (2018). We simply refer to these plots as *heatmaps*. As an example, Figure 4.12 shows the heatmaps of mortality improvement for all-causes of death. The horizontal axis shows the progression of mortality improvement through time, from 1959 to 2016. The vertical axis shows the progression by age, from 20 to 89. The colors represent smoothed annual percentage rates of improvement, with blue hues signifying mortality improvement, and red hues mortality deterioration. We note that in the heatmaps we have capped very large improvements and deteriorations at ±8% p.a. so that very intense reds represent a deterioration of more than 8% p.a. and very intense blues represent improvements of more than 8% p.a.

As in Rau et al. (2018), the smoothing is performed using the P-splines methodology implemented in the `R` package **MortalitySmooth** (Camarda, 2012). The P-spline methodology allows the simultaneous smoothing of mortality rates along the age and period dimensions (Currie et al., 2004). This smoothing thus addresses random fluctuations due to scanty data, while not being prone to overfitting.

In an improvement rate heatmap, period effects are shown through vertical alignment, as mortality changes occur for the whole population at a particular point in time. Age patterns are shown through horizontal patterns, as mortality changes occur for similar ages across time. Finally, cohort patterns are represented through diagonal alignment, as the group of individuals sharing the same birth year experience similar changes.

The predominance of blue hues in Figure 4.12 shows that for all-causes improvement has been steady for the majority of ages and time periods, with the exception of younger ages in the 1960s, 2000s and from about 2012 onwards for both genders. For young men below age 40 we also see a deterioration in mortality during the late 1980s coinciding with the HIV epidemic. For older men and women we see a slight deterioration of mortality in the early 1980s and 1990s. The birth cohorts around 1950 for both males and females exhibit a

Figure 4.13: Heatmaps for broad causes of death, ages 20–89, years 1959–2016, females



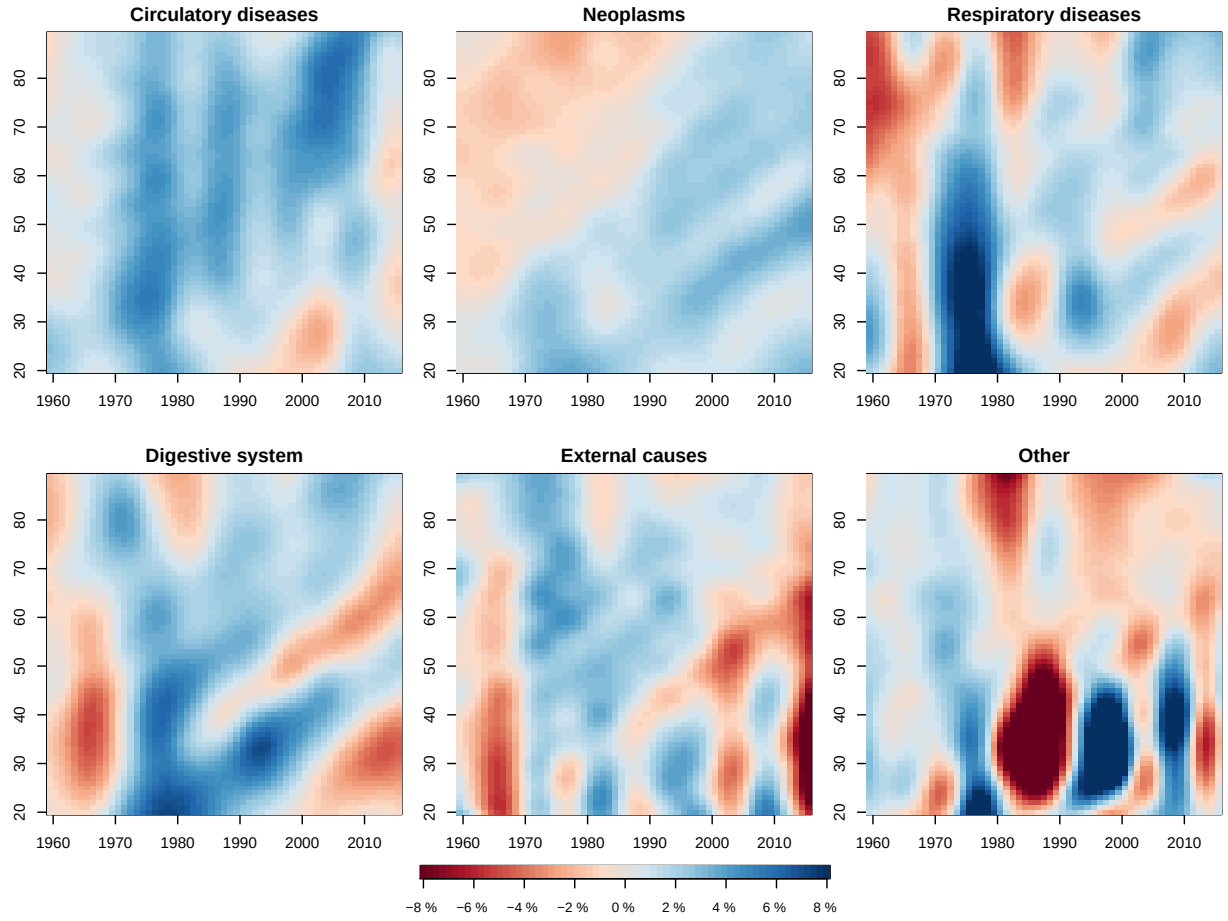
deterioration in mortality overall as indicated by the diagonal patterns of light red hues.

4.3.1 HEATMAPS FOR BROAD CAUSES OF DEATH (LEVEL 1)

We now examine improvement rate patterns for major causes of death. In Figures 4.13 and 4.14 we see that period effects are present for most causes. Such effects are most clearly seen in circulatory diseases for both males and females, where the vertical striations show differing rates of mortality improvements. For circulatory diseases, blue hues dominate indicating mortality improvements throughout the period with the noticeable exception of the years post 2012 where some patterns of mortality deterioration (red hues) have started to emerge. Similar deterioration patterns post 2012 have also emerged for external causes and "Other" causes of death. We also see a deterioration of mortality in the period between to 1960 and 1970 for respiratory diseases, digestive diseases and external causes.

Horizontal pattern indicating age effects are less pronounced. However, for some causes there are some noticeable differences in mortality improvements among older and younger Americans. For younger men, the most prominent of these patterns is the clear deterioration

Figure 4.14: Heatmaps for broad causes of death, ages 20–89, years 1959–2016, males



of mortality from "Other" causes for men aged 20-50 in the late 1980s due to the AIDS epidemic which is followed by a period of significant mortality improvement in the 1990s. For men and women older than 70, we see clear mortality deterioration for respiratory, digestive and "Other" causes in the period between 1980 and 1985. It is noticeable that for women age 65 and older the deterioration of mortality from respiratory diseases extended until the year 2000.

Cohort effects, while less pronounced than period effects, are present for digestive and respiratory diseases as indicated by the diagonal patterns in the corresponding panels of Figures 4.13 and 4.14. For respiratory diseases, the cohorts centered around 1950 and 1980 for males and the cohorts centered around 1950 for females show distinctive patterns of mortality deterioration. Deaths due to diseases of the digestive system also exhibit cohort effects for both males and females for the 1950s birth cohorts.

Figure 4.15: Heatmaps for subcategories of death related to circulatory disease, ages 20–89, years 1959–2016, females

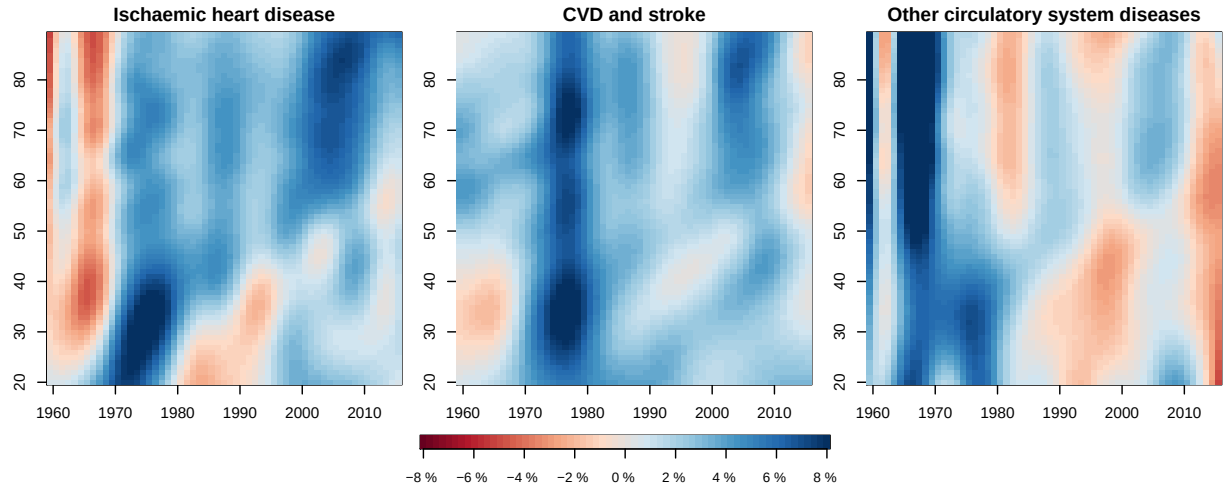
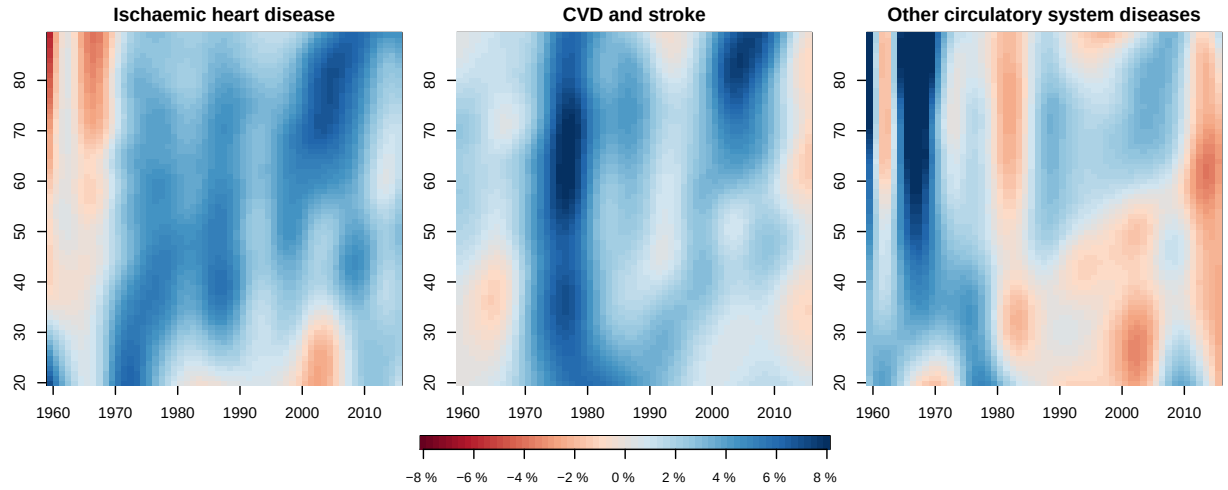


Figure 4.16: Heatmaps for subcategories of death related to circulatory disease, ages 20–89, years 1959–2016, males

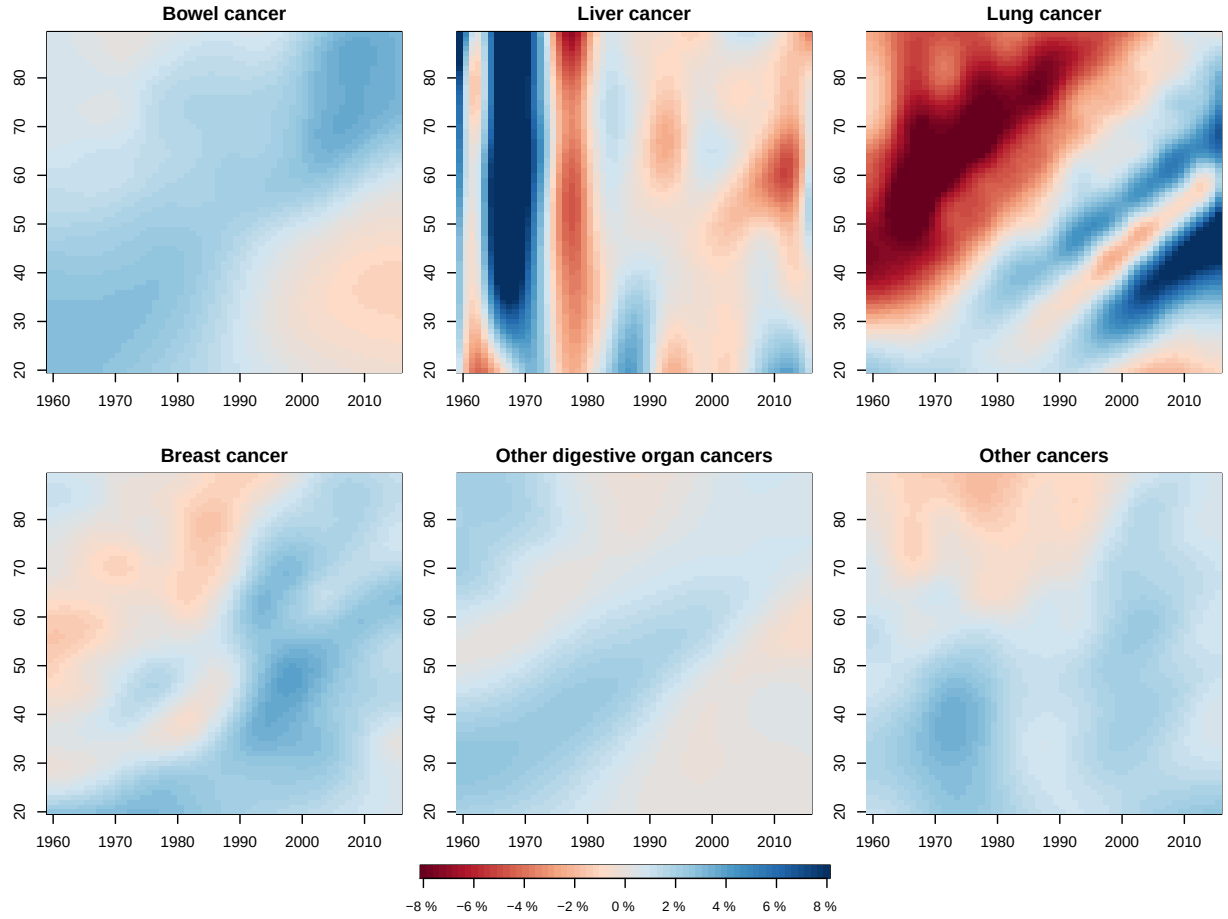


4.3.2 HEATMAPS FOR LEVEL 2 CAUSES

Figures 4.15 to 4.26 show more detailed heatmaps for subcategories of death at the Level 2 grouping of causes. These plots should shed more light on the complex patterns present within some of the broad groups of causes such us cancers, external and "Other" causes of death.

Figures 4.15 and 4.16 decompose deaths due to circulatory disease for males and females, respectively. We see in these two figures that the improvement patterns for male and female are very similar. Like the total of circulatory diseases, the subcategories are dominated by clear period effects. We note, however, that some of the period patterns observed in

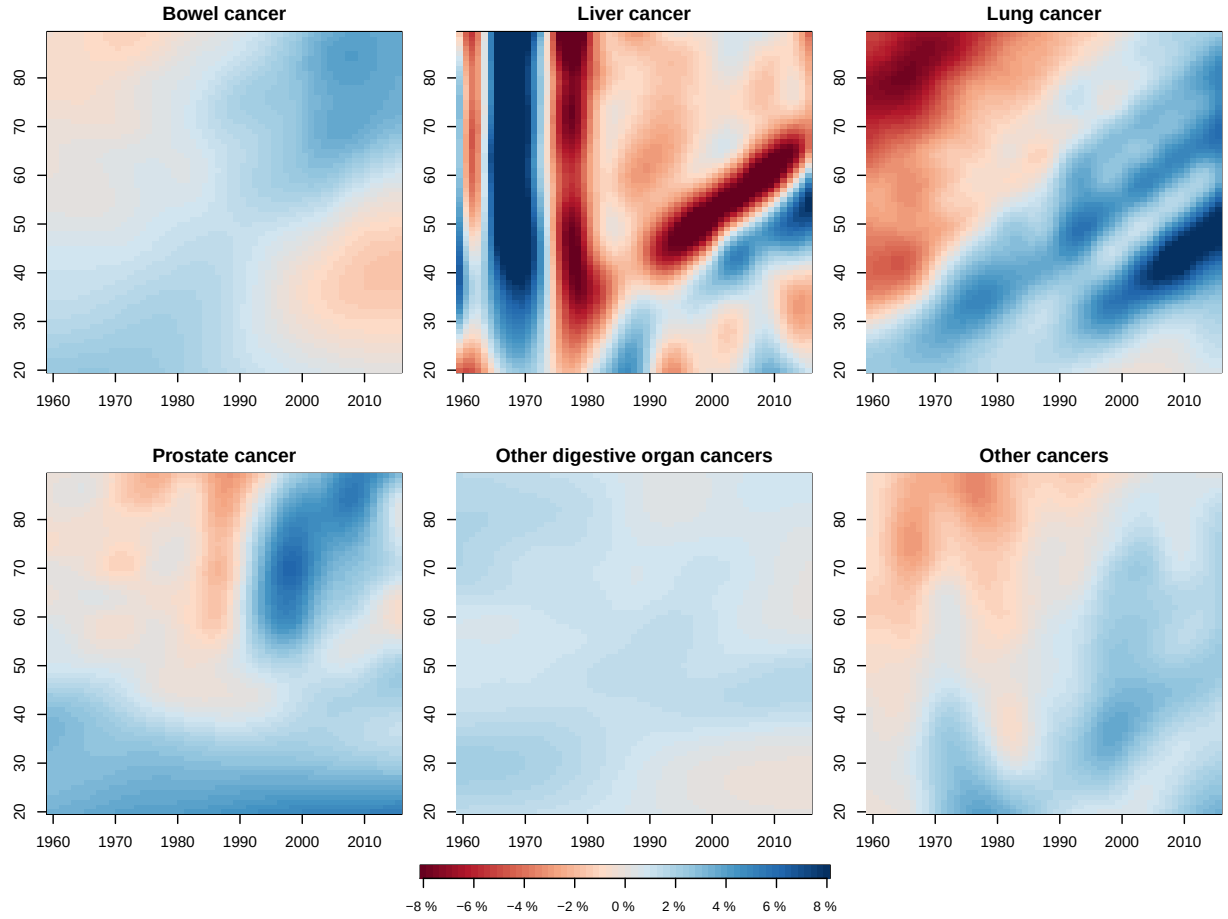
Figure 4.17: Heatmaps for subcategories of cancer deaths, ages 20–89, years 1959–2016, females



the heatmaps are confounded with ICD coding changes. An example of this is the vertical patterns between 1960s and 1970s in ischaemic heart disease (IHD) and other circulatory diseases which in principle would suggest significant period effects. Nevertheless, part of these vertical patterns is the result of the clear disruption in the coding of deaths from these two causes induced by the move from ICD-7 to ICD-8 (see Figure 4.3). Other noteworthy periods effects for the subcategories of circulatory diseases are the large improvement in CVD and stroke between 1970 and 1980 and the mortality deterioration in CVD and stroke and other circulatory diseases post 2012.

Subcategories of cancer deaths are shown in Figures 4.17 and 4.18, where there are contrasting patterns of improvement for different cancers. For bowel cancer, recent improvements are mainly confined to older ages, with younger ages experiencing deterioration. However, based on the heatmaps it is difficult to attribute the improvement in bowel cancer to period or cohort effects. For liver cancer, we see strong period patterns during the 1960s and 1970s which can mostly be attributed to coding changes as indicated by the discontinuity affecting ASDR trends for this cause observed in Figure 4.4. However, there is a strong cohort

Figure 4.18: Heatmaps for subcategories of cancer deaths, ages 20–89, years 1959–2016, males



effect in liver cancer mortality for the 1945–55 birth cohort. For lung cancer, the diagonal regions of alternating blue and red striations indicate predominantly cohort patterns. This is consistent with the widely documented variation of smoking prevalence across birth cohorts (Jemal et al., 2001; Gutterman, 2015). We see a period effect in the late 1980s for prostate cancer, but only for older ages. We also see particularly strong period effects for breast cancer with a general improvement in mortality from this cause from 1980 onwards. Finally, it is difficult to identify clear age, period or cohort patterns for other digestive organ cancers or other cancers for both genders.

Figures 4.19 and 4.20 present deaths due to respiratory diseases, where there are distinct period patterns in the earlier periods for all three subcauses. However, these period patterns can be traced back to the discontinuities in mortality induced by coding changes between ICD-8 and ICD-9 (see Figure 4.5). Period effects in influenza and pneumonia as well as other respiratory disease are persistent throughout the period of analysis. Furthermore, a distinct cohort effect emerges for deaths due to chronic lower respiratory disease at ages over 50, for males and females born in the 1950s.

Figure 4.19: Heatmaps for subcategories of deaths due to respiratory disease, ages 20–89, years 1959–2016, females

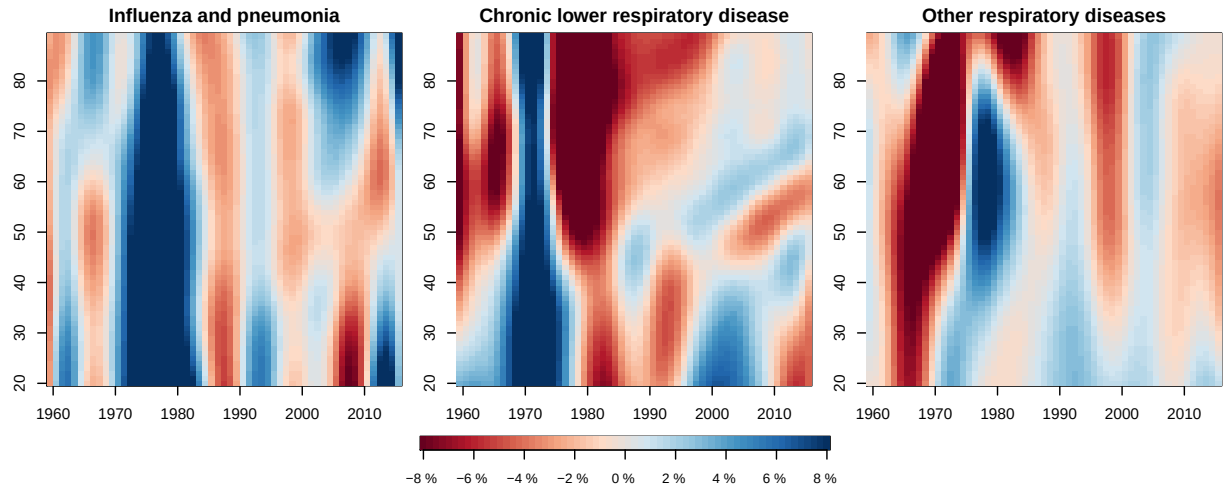


Figure 4.20: Heatmaps for subcategories of deaths due to respiratory disease, ages 20–89, years 1959–2016, males

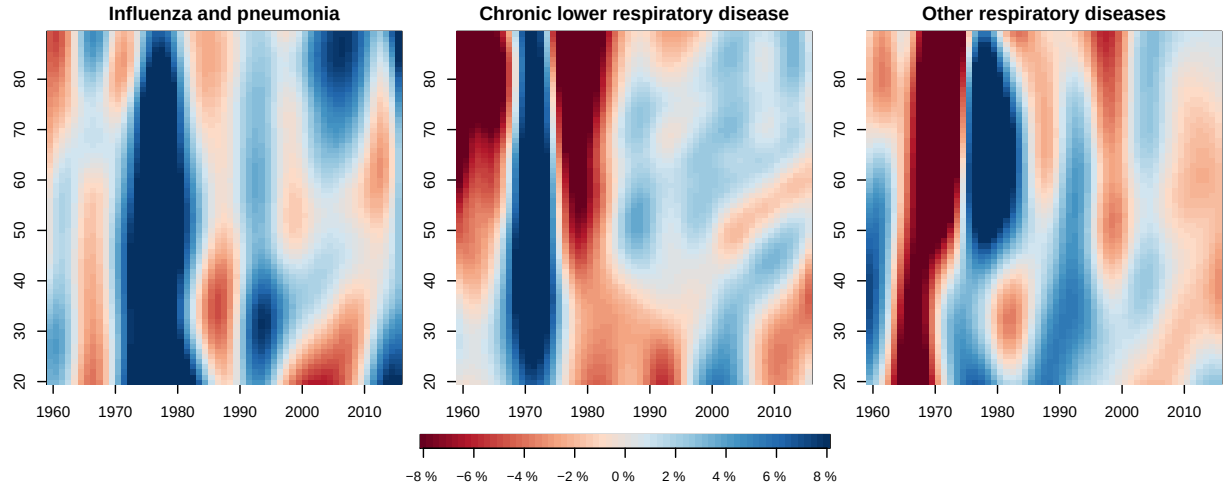


Figure 4.21: Heatmaps for subcategories of deaths due to digestive diseases, ages 20–89, years 1959–2016, females

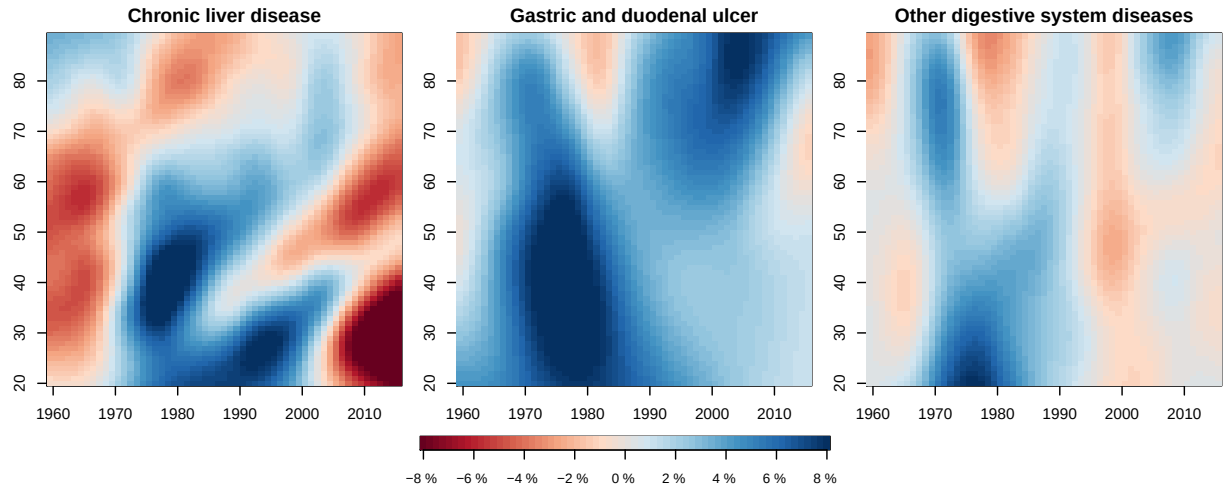


Figure 4.22: Heatmaps for subcategories of deaths due to digestive diseases, ages 20–89, years 1959–2016, males

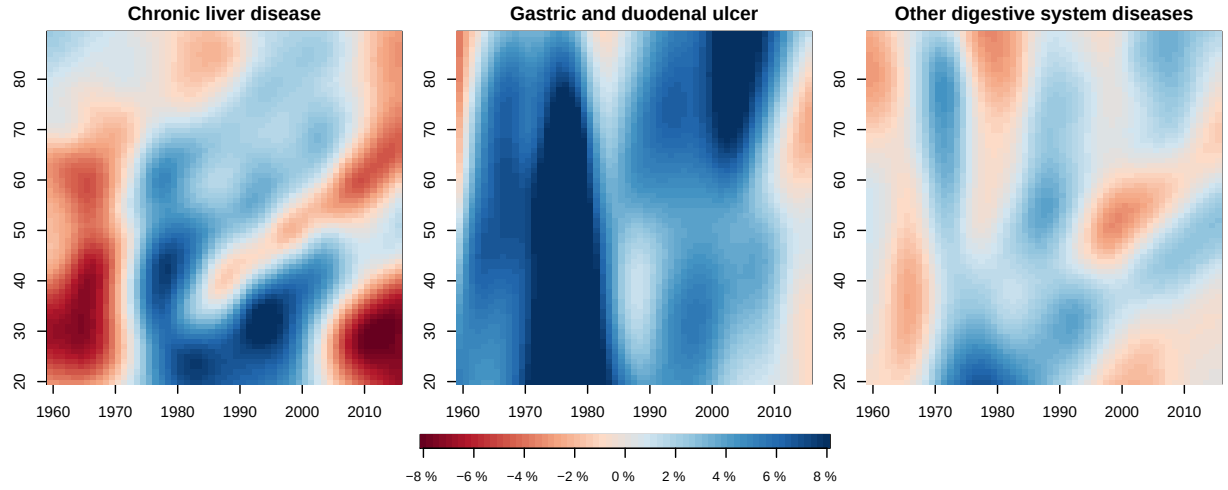
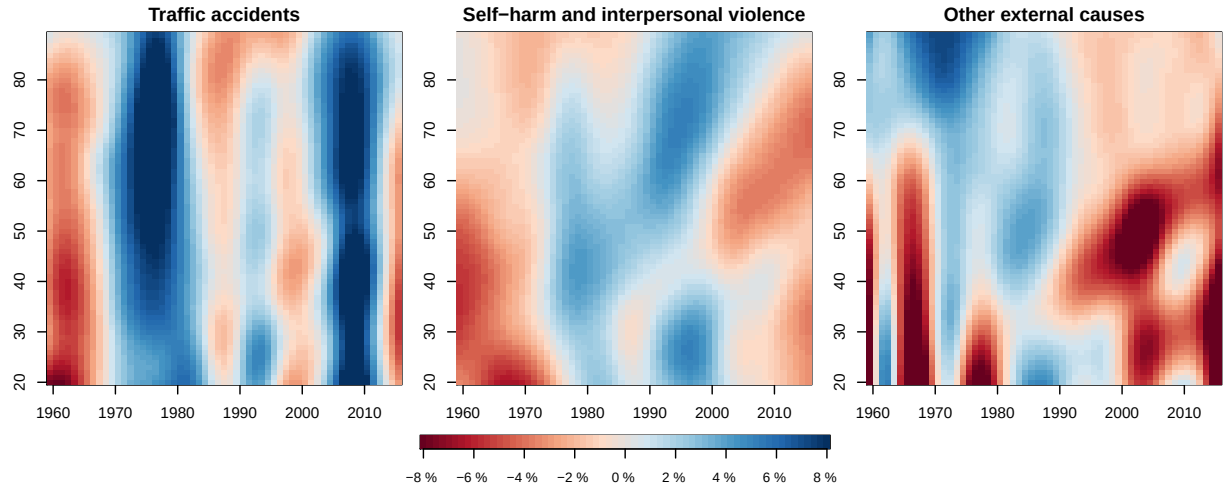


Figure 4.23: Heatmaps for subcategories of deaths due to external causes, ages 20–89, years 1959–2016, females



Subcategories of deaths due to digestive diseases are shown in Figures 4.21 and 4.22, where a variety of age, period and cohort effects can be seen. For chronic liver disease, younger ages have experienced volatile changes in mortality rates, particularly in the most recent period with rapid deterioration. Also, we can see a cohort effect for the 1945–55 birth cohort, which is more pronounced for males compared to females. This matches the effect seen in Figures 4.17 and 4.18 for liver cancer. For men in particular, we also see a similar, but less pronounced cohort effect for other digestive system diseases. For gastric and duodenal ulcer, we see mostly a consistent improvement in mortality through the whole period for all ages. However, there are the noticeable exceptions of the 1983-1985 period which shows a deterioration for men and women aged 80+ and of the post 2012 period which shows signs of deterioration in gastric and duodenal ulcer mortality for men of all ages.

Figures 4.23 and 4.24 decompose deaths due to external causes, where there are contrasting patterns of improvement. Period effects are most clearly seen for traffic accidents and for other external causes. However, the patterns observed around 1980 need to be interpreted with care as the change between ICD-8 and ICD-9 saw a shift in deaths between these two causes (see Figure 4.7). For self-harm and interpersonal violence there is a sharp deterioration in mortality post-2000 for both genders which seems to have accelerated in the most recent years. There are some faint diagonal patterns in the heatmaps for self-harm and interpersonal violence and for other external causes which may suggest the presence of cohort effects.

Finally, various other categories of death are presented in Figures 4.25 and 4.26. The profound deterioration in mortality due to AIDS and tuberculosis primarily affected younger ages between the period of 1970 and 1990, with significant improvement thereafter. After a brief period of deterioration in the early 2000s, particularly for middle-aged Americans, we see that there has been subsequent overall improvements. Period effects are also present for diabetes and obesity, where periods of improvement occurred from 1970–1980 and 2000–

Figure 4.24: Heatmaps for subcategories of deaths due to external causes, ages 20–89, years 1959–2016, males

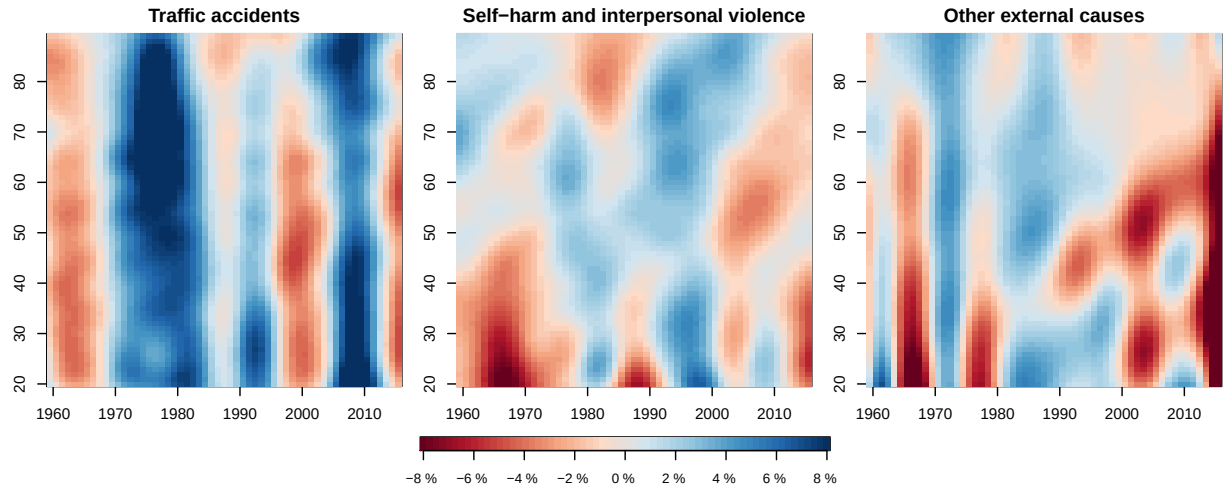


Figure 4.25: Heatmaps for subcategories of deaths due to other causes, ages 20–89, years 1959–2016, females (except for Alzheimer's disease: ages 20–89, years 1979–2016)

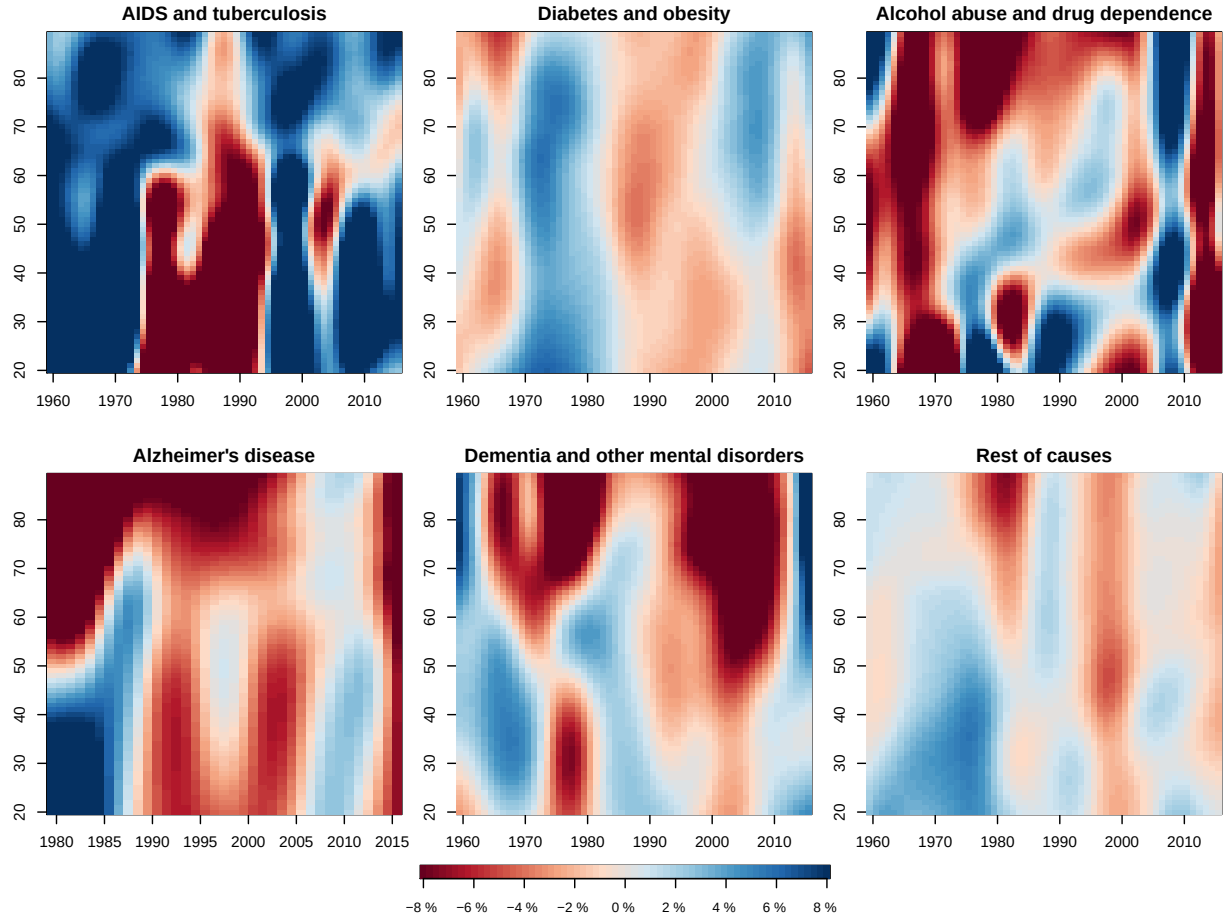
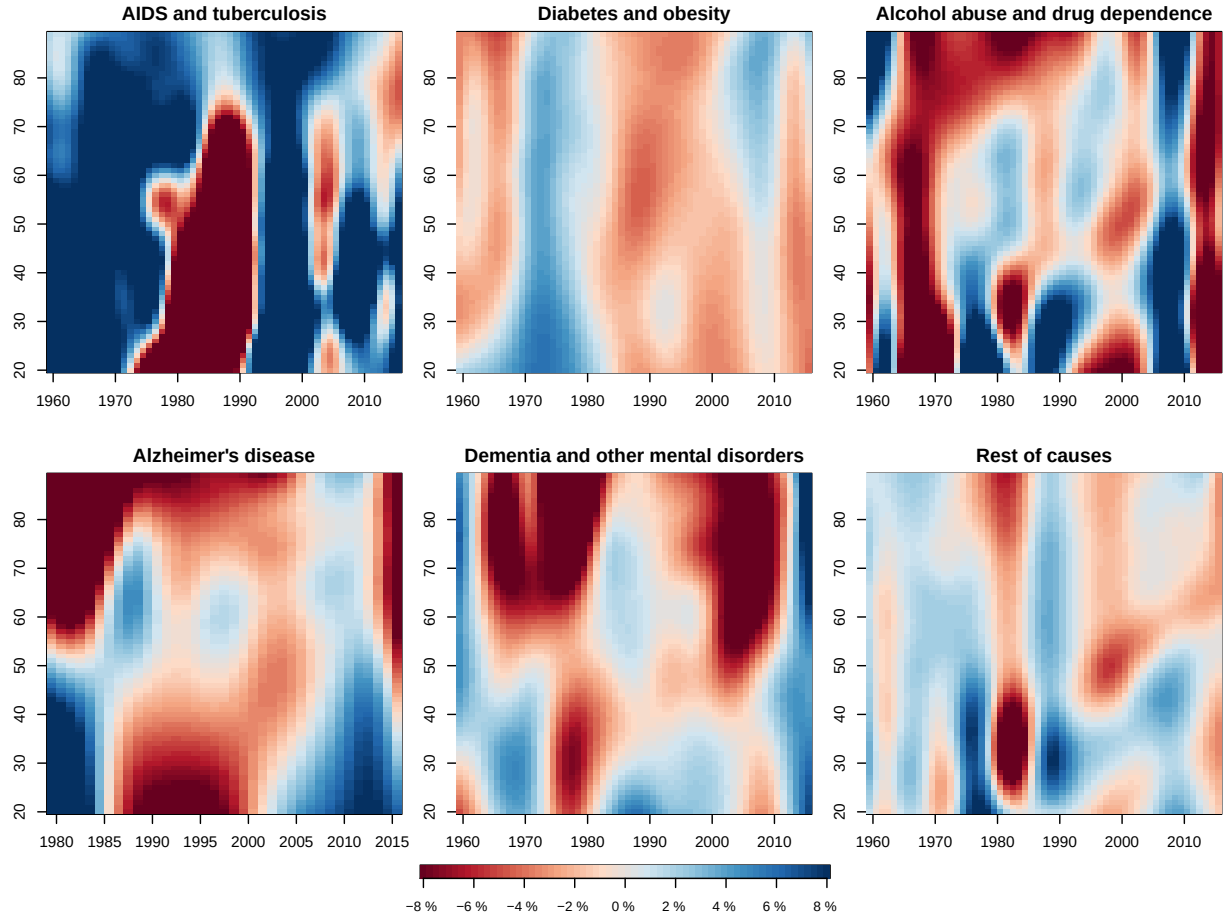


Figure 4.26: Heatmaps for subcategories of deaths due to other causes, ages 20–89, years 1959–2016, males (except for Alzheimer's disease: ages 20–89, years 1979–2016)



2010, which were more pronounced for females than for males. Deaths due to alcohol abuse and drug dependence are harder to interpret, with the only clear patterns being the deterioration in mortality in the 1970s and post-2010. In addition improvement patterns for younger ages appear more volatile than for older ages. Mortality due to Alzheimer's disease has deteriorated overall from 1979–2016, especially for older females. However there was a short period of mortality improvement from 2010–2013, followed with a dramatic deterioration thereafter. For dementia and other mental disorders, age-related effects are most prominent. This is to be expected, as dementia primarily affects older ages. An exception to the overall deterioration in mortality due to this cause can be seen in the last few years, from about 2012. Interestingly, this improvement in Dementia in the 2012–2016 period matches the deterioration seen in the same period for Alzheimer's disease which might suggest a shift in the coding of deaths between the two causes. Mortality due to the rest of causes is quite heterogeneous, with contrasting patterns for males and females. There is a significant period effect for younger males in the 1980s, and a slight 1950s cohort effect. For females, we see a moderate deterioration of mortality from 1995–2005.

4.3.3 HEATMAPS FOR SELECTED RISK FACTORS

We now turn our attention to the mortality improvement patterns of selected risk factors. This is shown in Figures 4.27 and 4.28 where we see contrasting period and cohort patterns among the risk factors: AIDS and tuberculosis, dementia and Alzheimer's disease, diabetes and obesity, hypertensive disease, and homicide are dominated by period effects, while alcohol abuse and smoking by cohort effects.

As discussed before, AIDS and tuberculosis show significant period effects with a deterioration between 1970 and 1990 and significant improvement thereafter.

For dementia and Alzheimer's disease we see contrasting patterns for younger and older Americans, with persons aged 60 and over experiencing a significant deterioration in mortality from this risk factor between 1970 and 2012. However, in the more recent 2012-2016 period dementia and Alzheimer's disease combined show an improvement pattern in spite of the possible offsetting effect between the two individual causes.

Hypertensive disease and diabetes and obesity show very similar period patterns with important mortality deterioration post 1985. However, we note that for hypertensive disease the pattern before 1980 needs to be carefully interpreted as deaths from this factor were significantly impacted by ICD coding changes during the ICD-7 and ICD-8 periods (see Figure 4.9).

Drug dependency shows a very sharp deterioration in mortality from 1995 onwards for both gender and most ages. However, it is difficult to attribute this deterioration to period or cohort effects as both diagonal and vertical pattern are observable.

For homicide and self-harm we see interesting contrasting patterns. While homicide clearly shows a prevalence of period effects, self-harm shows some diagonal patterns.

Consistent with the pattern discussed previously for liver cancer and chronic liver disease, alcohol abuse shows very strong cohort patterns affecting the 1945–55 birth cohorts. Similarly, smoking, for which we are using lung cancer deaths as the sole marker, shows very clear cohort effects for both males and females as discussed before.

Figure 4.27: Heatmaps for for selected risk factors, ages 20–89, years 1959–2016, females

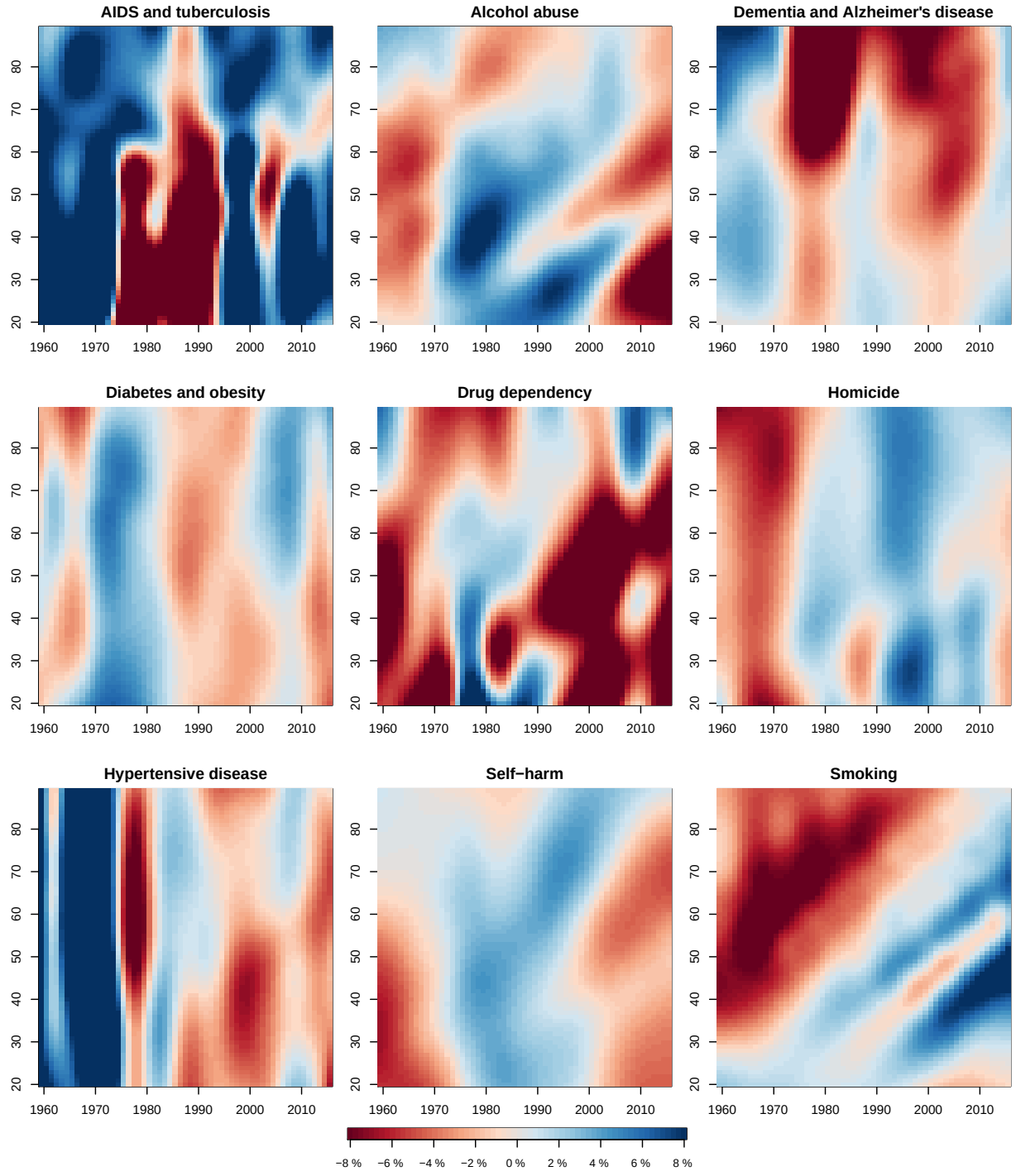
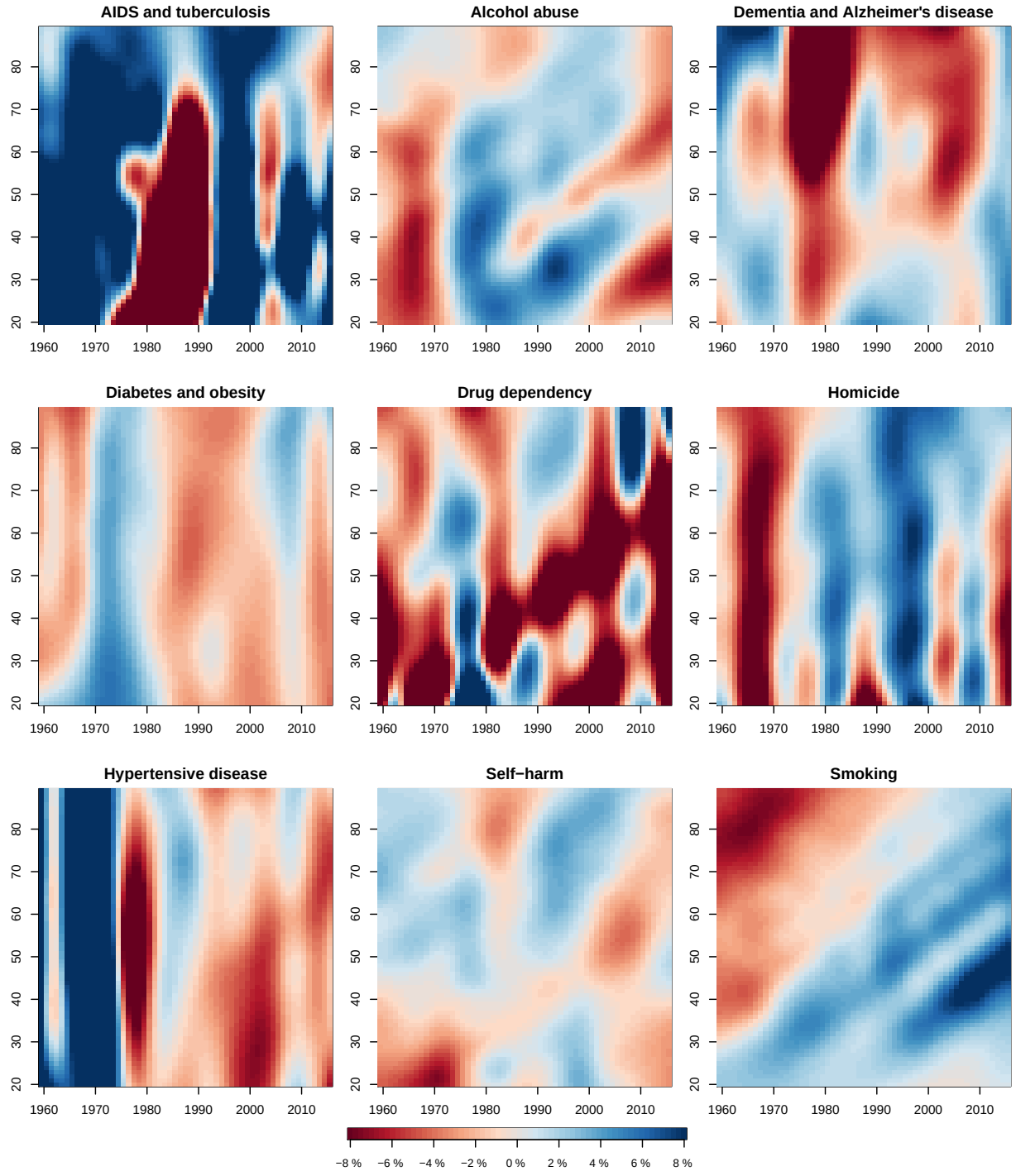


Figure 4.28: Heatmaps for for selected risk factors, ages 20–89, years 1959–2016, males



Section 5: Age-Period-Cohort decomposition of mortality

After the descriptive analysis of the previous section, in this section we focus on the more systematic separation and quantification of mortality trends across the dimensions of age, period and cohort using common actuarial and demographic modeling techniques.

5.1 GENERALIZED AGE-PERIOD-COHORT MODELS

In order to systematically approach the decomposition of mortality improvement rates into age, period and cohort components, we consider the framework of Generalized Age-Period-Cohort (GAPC) models. Originally, GAPC models were developed as a general structure for mortality rate models (Hunt and Blake, 2021; Villegas et al., 2018). However, here we use GAPC models as a general structure of mortality improvement rate models as discussed in Hunt and Villegas (2020).

For a given cause of death, let $m_{x,t}$ denote the mortality rate at age x in year t. Under the GAPC improvement rate framework, we define the annual improvement rate at age x in year t as $\eta_{x,t} = -\log \frac{m_{x,t}}{m_{x,t-1}}$, and assume that it follows the following specification:

$$\eta_{x,t} = -\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha_x + \sum_{i=1}^{N} \beta_x^{(i)} \kappa_t^{(i)} + \gamma_{t-x}, \tag{5.1}$$

where

- α_x is a static function of age, which gives the average (constant) rate of improvement in mortality at each age x;
- $\kappa_t^{(i)}$ are period functions governing the change in improvement rate in year t;
- $\beta_x^{(i)}$ are age functions which modulate the corresponding period functions; and
- γ_c is a cohort function describing systematic differences in the rate of improvement which depend upon a cohort's year of birth, $c = t - x$.

Hunt and Villegas (2020) note that there is a close link between mortality improvement rate GAPC models and traditional mortality rate models. Specifically, the improvement rate structure in Equation (5.1) has the following parallel mortality rate GAPC modeling structure:

$$\log m_{x,t} = A_x - \alpha_x t + \sum_{i=1}^{N} \beta_x^{(i)} K_t^{(i)} + \Gamma_{t-x}, \tag{5.2}$$

where A_x is the general age level of mortality, $K_t^{(i)}$, $i = 1, \ldots, N$, are period effects and Γ_c are cohort effects. The period effects and cohort effects in Equation (5.2) are linked to the period and cohort improvements in Equation (5.1) by the relationships

$$\kappa_t^{(i)} = -\Delta K_t^{(i)} = -(K_t^{(i)} - K_{t-1}^{(i)}) \quad \text{and} \quad \gamma_c = -\Delta \Gamma_c = -(\Gamma_c - \Gamma_{c-1}). \tag{5.3}$$

Accordingly, to estimate the GAPC improvement rate models, we follow an indirect estimation approach whereby we estimate the mortality rate model in Equation (5.2) under a Poisson maximum likelihood approach and recover the improvement rate parameters of Equation (5.1) using the relationships in Equation (5.3). The details of this estimation method can be seen in Hunt and Villegas (2020). Moreover, we estimate mortality improvement rate models using the `R` package **iMoMo** available in Github at https://github.com/amvillegas/iMoMo and which extends the `R` package **StMoMo** (Villegas et al., 2018).

5.1.1 PREDICTOR STRUCTURES

The GAPC structures in Equations (5.1) and (5.2) encompass several common mortality modeling structures including, among others, the popular model of Lee and Carter (1992), the standard age-period-cohort model and the Cairns-Blake-Dowd family of models (Cairns et al., 2009).

Table 5.1 summarizes the predictor structures which we consider as potential candidate for the age-period-cohort decomposition of mortality improvements among the different causes of death in the U.S.

Table 5.1: Candidate GAPC predictor structures for age-period-cohort decomposition of improvement rates

Model	**Improvement rate structure**	**Equivalent mortality rate structure**
APCi	$-\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha_x + \kappa_t + \gamma_{t-x}$	$\log m_{x,t} = A_x - \alpha_x t + K_t + \Gamma_{t-x}$
PCi	$-\log \frac{m_{x,t}}{m_{x,t-1}} = \kappa_t + \gamma_{t-x}$	$\log m_{x,t} = A_x + K_t + \Gamma_{t-x}$
ACi	$-\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha_x + \gamma_{t-x}$	$\log m_{x,t} = A_x - \alpha_x t + \Gamma_{t-x}$
APi	$-\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha_x + \kappa_t$	$\log m_{x,t} = A_x - \alpha_x t + K_t$
PLATi	$-\log \frac{m_{x,t}}{m_{x,t-1}} = \kappa_t^{(1)} + (x-\bar{x})\kappa_t^{(2)} + \gamma_{t-x}$	$\log m_{x,t} = A_x + K_t^{(1)} + (x-\bar{x})K_t^{(2)} + \Gamma_{t-x}$

The APCi model given by

$$-\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha_x + \kappa_t + \gamma_{t-x}, \tag{5.4}$$

is our reference model. This model is the improvement rate version of the classical age-period-cohort model and broadly corresponds to the model used by the Continuous Mortality Investigation (2016b; 2016a) in the United Kingdom in their latest mortality projection model.

The PCi, ACi, APi structures are submodels of the APCi model in which we assume, respectively, that $\alpha_x \equiv 0$, $\kappa_t \equiv 0$ and $\gamma_c \equiv 0$ so that age, period and cohort effects are absent from

the mortality improvement rate specifications. Being submodels of the full APCi model, these predictor structures allow us to gauge the relative importance of each of the three components. We note that in its mortality rate representation, the PCi model corresponds to the standard APC model for mortality rates commonly used in demography, sociology and epidemiology. In particular, the standard APC model has been previously considered in the decomposition of cause-specific U.S. mortality trends into age-period-cohort components (see. e.g., Masters et al. (2014) and Yang (2008)).

Finally, the PLATi model is an extension of the PCi which allows for variations in the pace of mortality improvement by age through the incorporation of a second age-period term, $(x - \bar{x})\kappa_t^{(2)}$, where $\bar{x}$ is the mean of the ages in the data. In its mortality rate form this model corresponds to the simplified Plat model proposed by Plat (2009) and which has been suggested in the SOA project preceding this work as being appropriate to model All-cause mortality trends for the U.S. populations for ages 20 to 95 (Li et al., 2017a,b, 2020). Moreover, this model has recently been used by Lourés and Cairns (2021) to investigate cause-specific cohort effects in the USA among education groups. The consideration of the PLATi model is further motivated by the fact that in the preliminary descriptive analysis in Section 4.3, we noted that although for most causes age effects were generally less prominent than period and cohort effects, there were causes such as AIDS and dementia which exhibited distinct improvement patterns for young and older Americans.

A key aspect of GAPC modeling is that the parameters of the models are only identifiable up to a set of transformations and thus require constraints to ensure unique parameters estimates. Therefore, to estimate the predictors structures in 5.1 we impose the constraints in Table 5.2.

Table 5.2: Estimation constraints for GAPC mortality improvement predictors

Model	**Period constraints**	**Cohort constraints**
APCi	$\sum_t \kappa_t = 0$	$\sum_c \gamma_c = 0$, $\sum_c c\gamma_c = 0$
PCi	-	$\sum_c \gamma_c = 0$
ACi	-	$\sum_c \gamma_c = 0$
APi	$\sum_t \kappa_t = 0$	-
PLATi	-	$\sum_c \gamma_c = 0$, $\sum_c c\gamma_c = 0$

For the APCi and the PLATi models, the constraints in Table 5.2 remove linear trends from the cohort component and thus relegate the cohort dimension to a secondary role with the dimensions of age and period being assumed to be dominant. This is an important assumption which needs to be taken into account when interpreting age-period-cohort decompositions. We elaborate on this issue in Section 5.2.2.

Table 5.3: Effective number of parameters for models fitted to the age range 20-89 and the period 1959–2016

APCi	PCi	APi	ACi	PLATi
320	252	196	265	308

5.2 MODEL SELECTION

To select a model from the predictor structures in Table 5.1, we take into account two important criteria: the parsimony of the model and the robustness of the parameters to the identification strategy.

5.2.1 GOODNESS-OF-FIT AND PARSIMONY

In order to assess the trade-off between the goodness-of-fit and parsimony of the predictors structures, we use the Bayesian Information Criterion (BIC), defined as:

$$BIC = \nu \log K - 2\mathcal{L},$$

where ν is the effective number of parameters of the model, K is the number observations in the data and $\mathcal{L}$ is the Poisson log-likelihood of the model.

We fit all the models to data for the age range 20-89 and the period 1959-2016, hence the number of observations is $K = \text{ages} \times \text{periods} = 70 \times 58 = 4060$. For this data range, the effective number of parameters ν for each of the predictors structure is presented in Table 5.3.

Since we are taking an indirect estimation approach, the effective number of parameters ν is the numbers of parameters of the equivalent mortality rate model minus the numbers of constraints required to uniquely identify the mortality rate model. For example, for the APCi models we have 70 parameters for A_x, 70 parameters for α_x, 58 parameters for K_t, $78+58-1 = 127$ parameters for Γ_c, and 5 identifications constraints[1] for an effective number of parameters $\nu = 2 \times 70 + 58 + 127 - 5 = 320$.

Figure 5.1 presents the BIC values of the different models fitted to each of the causes of death. In this figure, model ranks in terms of BIC are indicated by the cell colors, with lower BIC value being preferable.[2] We see some noticeable regularities in the model rankings. Specifically, for the majority of causes the APCi and PLATi models are the best two models,

[1]We note that while for identifying the improvement rate structure we require three constraints as indicated in Table 5.2, for identifying the mortality rate structure we require two more constraints to properly identify the A_x term. See Hunt and Villegas (2020, Appendix A) for a discussion on this.

[2]Due to the low number of deaths at some ages for prostate cancer and gastric and duodenal ulcer in males and Alzheimer's disease in both genders, the PLATi model did not converge. Hence, the absence of BIC values for these causes in Figure 5.1.

Figure 5.1: BIC values by causes of death for different models fitted to data for ages 20–89 and years 1959–2016 (except for Alzheimer's disease: ages 20–89, years 1979–2016)

	Female					Male				
	APC	PC	AP	AC	PLAT	APC	PC	AP	AC	PLAT
All−Cause	70487	84873	107148	111577	75732	93886	127756	170320	136156	78064
Circulatory diseases	52318	71255	78434	87637	68670	56844	68339	108474	95092	63197
Ischaemic heart disease	45255	52548	62019	80400	49832	51306	56136	85471	93203	53630
CVD and stroke	40553	42165	43384	63859	40512	40331	42104	46644	60856	40179
Other circulatory system diseases	45572	49867	56109	110955	45300	45126	51128	60709	81530	47735
Neoplasms	46578	50398	60114	53526	47395	48196	49912	57232	51571	48180
Bowel cancer	34212	34632	37454	34375	34792	34702	36215	38758	34912	35742
Liver cancer	28194	27744	27773	32278	28039	29334	29149	35377	36778	29321
Lung cancer	34721	35722	50176	34816	34433	38563	38329	42971	38740	38016
Breast cancer	37066	36952	38429	38239	37108					
Prostate cancer						28124	28835	29440	33332	
Other cancers	41557	42676	45706	44775	42114	41561	41563	42961	43089	41638
Other digestive organ cancers	33917	33683	34637	33567	33827	35560	35126	35299	35281	35460
Respiratory diseases	41798	66533	55382	63377	40749	42827	49110	70155	57724	41360
Influenza and pneumonia	39306	44543	47631	66616	34262	40711	42344	54144	68161	35861
Chronic lower respiratory disease	34758	40567	47405	60728	34199	35792	36312	56219	85871	35732
Other respiratory diseases	33602	33958	37152	44011	33109	36318	36460	41820	94631	34574
Digestive system	48357	54030	57052	50786	38049	48898	50899	56036	55061	40000
Gastric and duodenal ulcer	24438	24376	24824	24968	23962	26900	26600	28186	28077	
Chronic liver disease	34917	35266	43136	42279	32410	37429	38211	42741	53000	34926
Other digestive system diseases	37295	39211	39469	41417	34485	37285	38291	39315	38611	35703
External causes	44509	47790	59195	64370	42827	54050	57741	74668	91810	50358
Traffic accidents	33368	34008	33283	44337	32601	38836	38740	41392	66982	37424
Self−harm and interpersonal violence	34430	34614	36392	42390	34451	45829	51787	55337	62265	44513
Other external causes	40546	43121	49827	48208	38915	44990	47250	60260	64493	42380
Other	53006	66344	59085	85378	50682	108827	170342	205510	141773	65517
AIDS and tuberculosis	33615	33434	44152	55921	29030	50343	56305	130440	192927	34235
Diabetes and obesity	36169	37964	38750	55608	37543	35958	38450	37239	49413	36999
Alcohol abuse and drug dependence	25406	25159	27077	26406	24853	32552	32355	35619	36798	30854
Alzheimer's disease	14705	16041	17658	19145		14291	14575	16135	17021	
Dementia and other mental disorders	26408	28074	29740	46457	27699	26171	26760	28082	38272	26803
Rest of causes	44926	52802	61496	59522	42297	47240	47676	63100	57015	44529

BIC rank 1 2 3 4 5

the PCi model ranks third, and the APi and ACi models rank in the last two places. This suggests that for most causes of death, period and cohort effects are more prominent than age effects, which is in agreement with some of the preliminary findings discussed in Section 4.3 when examining visually the improvement rates heatmaps for the different causes of death. There is, however, a small number of causes where these regularities do not hold. These include:

- Bowel cancers for both females and males where we observe that the ACi model ranks second while the APi model ranks fifth, suggesting that age and cohort effects dominate period effects.
- Liver cancer for females where the APi model ranks second and the ACi model ranks fifth suggesting that period effects dominate over cohort effects. This result agrees with the top center panel of Figure 4.17 in which vertical patterns are far more pronounced that diagonal patterns.

- Lung cancer for females where the ACi model ranks third indicating that cohort effects are important and which agrees with the very clear diagonal patterns seen in the top right panel of Figure 4.17.
- Other digestive organ cancers where the ACi model ranks first for females and second for males. However, we note that the BIC values for this cause are very similar across all models which indicates that it might be difficult to single out one of the three effects as the most prominent one.
- Traffic accidents for females where the APi model ranks second and the ACi model ranks fifth suggesting that period effects dominate cohort effects. This is consistent with the very clear vertical patterns seen in the left panel of Figure 4.23.

Based on the above discussion, we opt to discard the ACi and APi models and focus on the APCi, PCi and PLATi models as our potential candidates for the decomposition. These three models coincide in the fact that they all allow for period and cohort effects but differ in the degree in which they allow for age effects. The APCi permits a full variation of improvements by age, the PLATi permits variations in improvement rates between older and younger ages, and the PCi model assumes that age effects are absent.

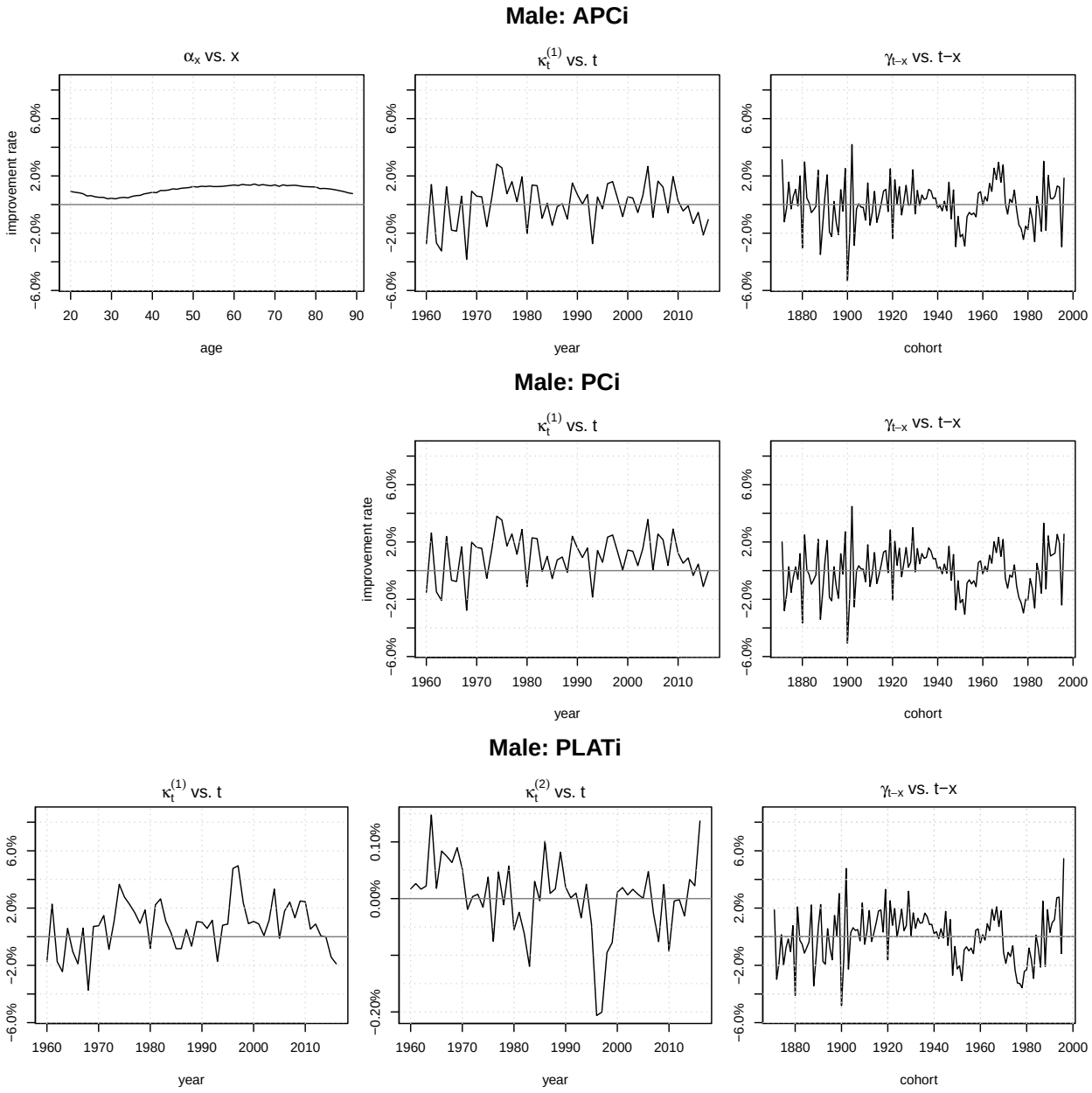
To illustrate the parameters of the three models and their interpretation, we present in Figure 5.2 the fitted parameters for mortality rates from all-causes of death for males. In the APCi model, the α_x parameters indicate that over the 1959-2016 period, mortality for males improved at a pace of about 1% p.a., with much faster improvements for males aged 50-80.

In the APCi model, the $\kappa_t^{(1)}$ parameters represent period deviations from the average improvements. For example, in 1975 all-cause mortality for males improved 2.6% p.a. more than the average of the whole period. Thus, the total average model improvement rate for 1975 is 3.6% p.a. By contrast, in 2015 all-cause mortality for males improved 2.1% p.a. less than the average of the 1959-2016 period, for a total average mortality deterioration of 1.1% p.a. in 2015.

The interpretation of the $\kappa_t^{(1)}$ parameters in the PCi and PLATi models differs to that in the APCi model, since these two models do not have an age effect. For example, in the PCi we have that $\kappa_{1975}^{(1)} = 3.5\%$ p.a, indicating that in 1975 for males mortality improved on average 3.5% for all ages, which is close to the 3.6% from the APCi model. It is worth noting that, in spite of the differences in the level, $\kappa_t^{(1)}$ for the APCi and the PCi models follows essentially the same pattern being a good indicator of period effects.

In the PLATi model, the $\kappa_t^{(1)}$ captures the improvement rates at the average age in the data; in this case at age $\bar{x} = 54.5$. For ages below and above $\bar{x}$ the improvement rates are modulated by the values of $\kappa_t^{(2)}$ with positive (negative) values indicating slower (faster) improvement at younger ages as compared to older ages. For example, the noticeable negative values of $\kappa_t^{(2)}$ around 1995 indicate very fast improvements for younger men relative to older men,

Figure 5.2: All-cause mortality improvement rate parameters for models APCi, PCi, PLATi, males, years 1959–2016, ages 20–89



which is consistent with the fast improvements in mortality experienced by younger men as a result of new AIDS treatments during this period (recall Figures 4.12 and 4.26).

In the three models, the γ_c parameters, which account for cohort specific deviations from the average improvements, follow essentially the same pattern. For example, in the APCi model we have that γ_{1950} =-2.3% indicating that the generation born in 1950 has experienced annual mortality improvements which are 2.3% worse than the average.

5.2.2 ROBUSTNESS TO MODEL CONSTRAINTS

The previous discussion shows that for most causes the APCi, PCi and PLATi models result in very similar interpretations of period and cohort effects. Thus, to decide among the three alternative models, we now examine how robust these interpretations are to the constraints used in the model identification. This is an issue that affects primarily the APCi and PLATi models.

It is well known that the decomposition of health trends into age-period-cohort components can be problematic, as the relationship $cohort = period - age$ poses an inherent identification problem that makes it difficult to clearly separate the mutually dependent changes across the three dimensions. This implies that one needs to be extremely cautious when interpreting results from the APCi and PLATi models as any such split depends on the identification scheme used and is only correct if the underlying (unknown) data generating process happens to be the same as the statistical model chosen (Bell and Jones, 2013, 2014; Harper, 2015).

The identification constraints for the APCi and PLATi models in Table 5.2 relegate the cohort dimension to a secondary role and assume that period effects are dominant. In order to test the robustness of these two models, we consider alternative identification schemes where cohort effects are assumed to be dominant over period effects. If a cause of death has clearly identifiable APC effects, we would expect that the model parameters obtained under the two different identification schemes would be similar.

For the APCi model, the alternative identification scheme we consider is as follows:

$$\sum_t \kappa_t = 0, \quad \sum_t t\kappa_t = 0 \quad \sum_c \gamma_c = 0. \tag{5.5}$$

Similarly, for the PLATi model we consider the following alternative identification scheme:

$$\sum_t \kappa_t^{(1)} = 0, \quad \sum_t t\kappa_t^{(1)} = 0. \tag{5.6}$$

Figures 5.3 and 5.4 plot the parameters estimates for the APCi and PLATi model, respectively, for selected causes of death under the two alternative identification schemes. In these figures, we see very noticeable differences in the parameter estimates depending on whether we choose period effects to dominate (black lines) or cohort effects to dominate (red lines).

Figure 5.3: APCi improvement rate parameters for selected causes of death with alternative identification constraints, years 1959–2016, ages 20–89

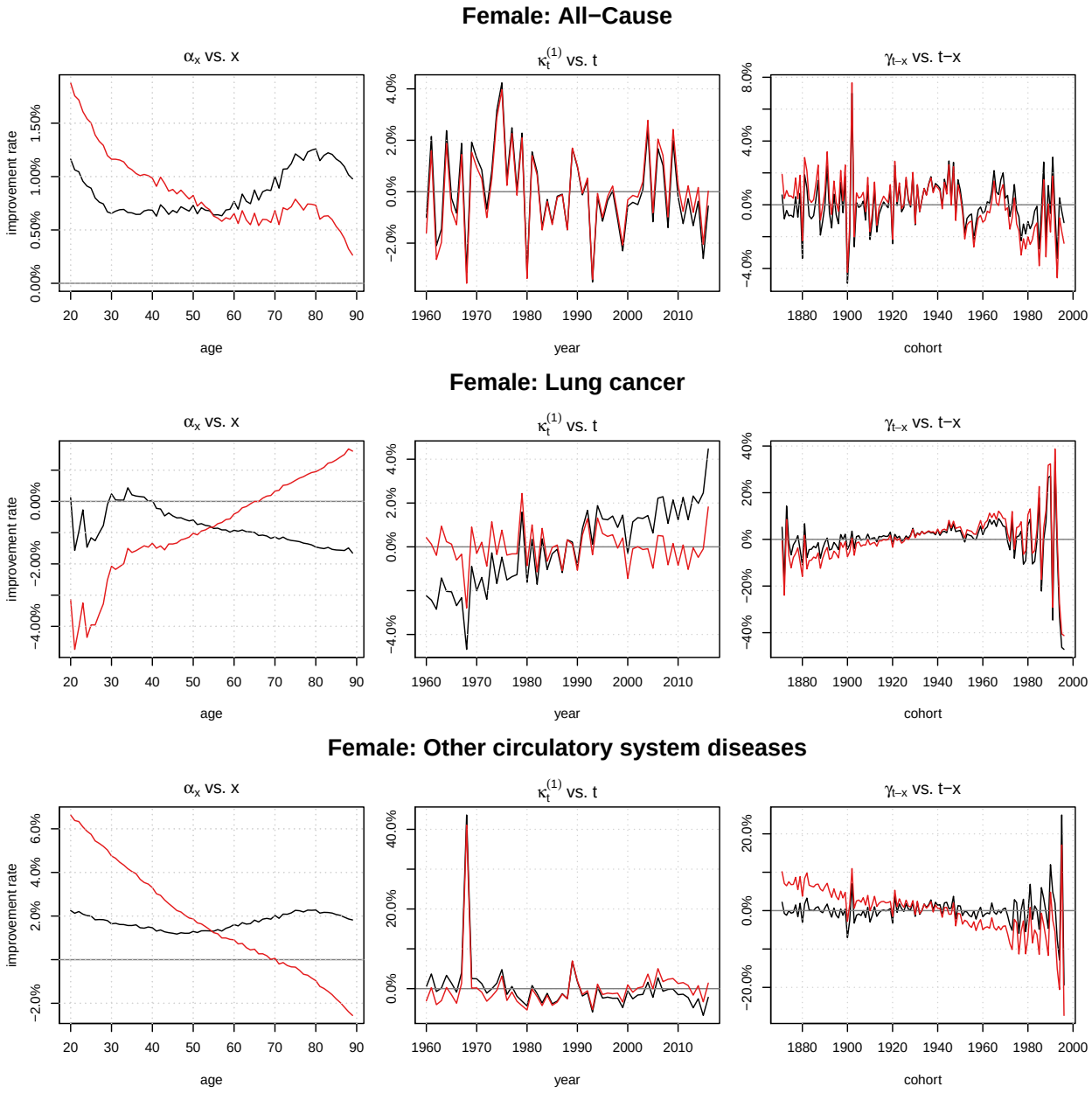


Figure 5.4: PLATi improvement rate parameters for selected causes of death with alternative identification constraints, years 1959–2016, ages 20–89

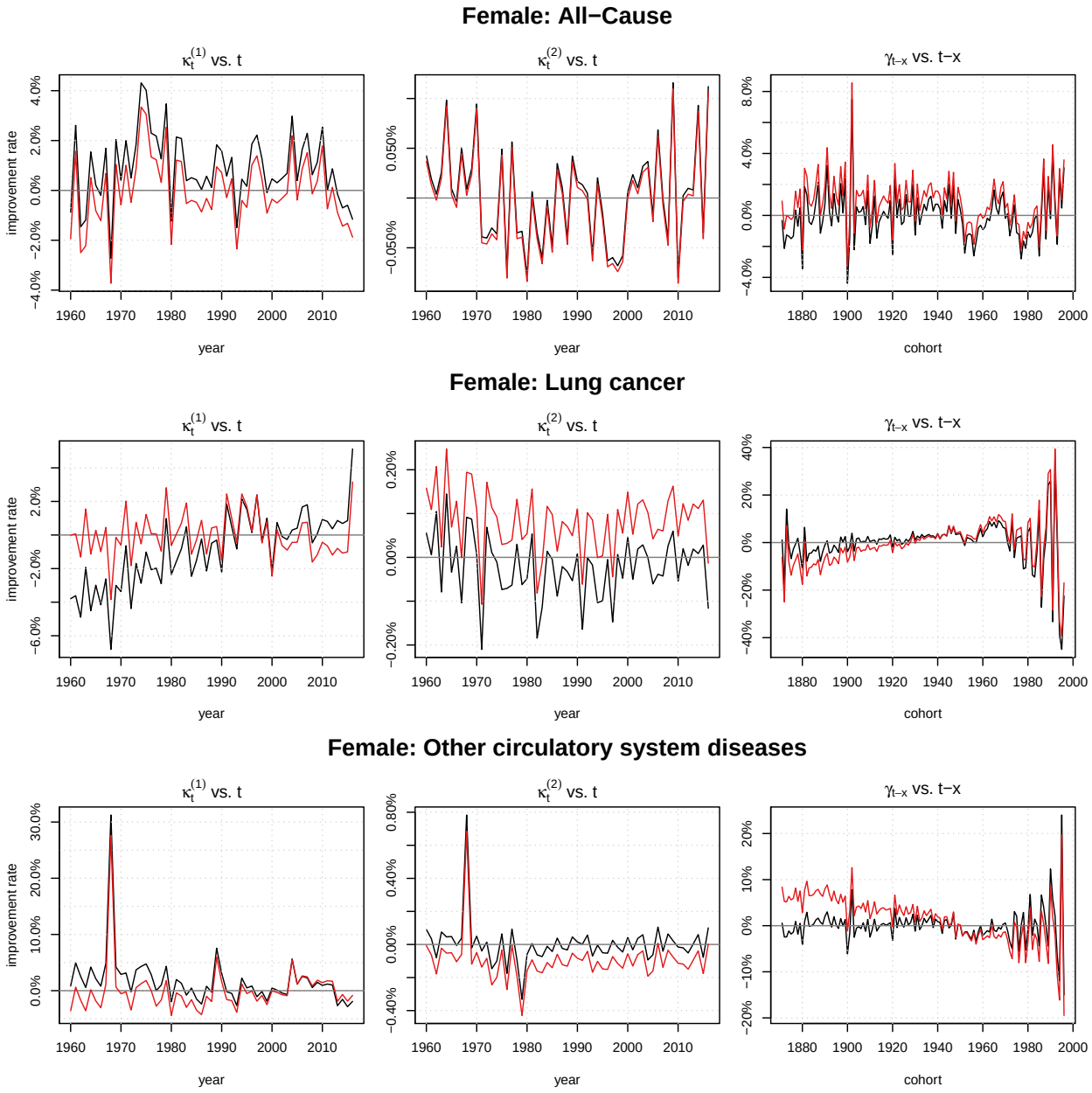
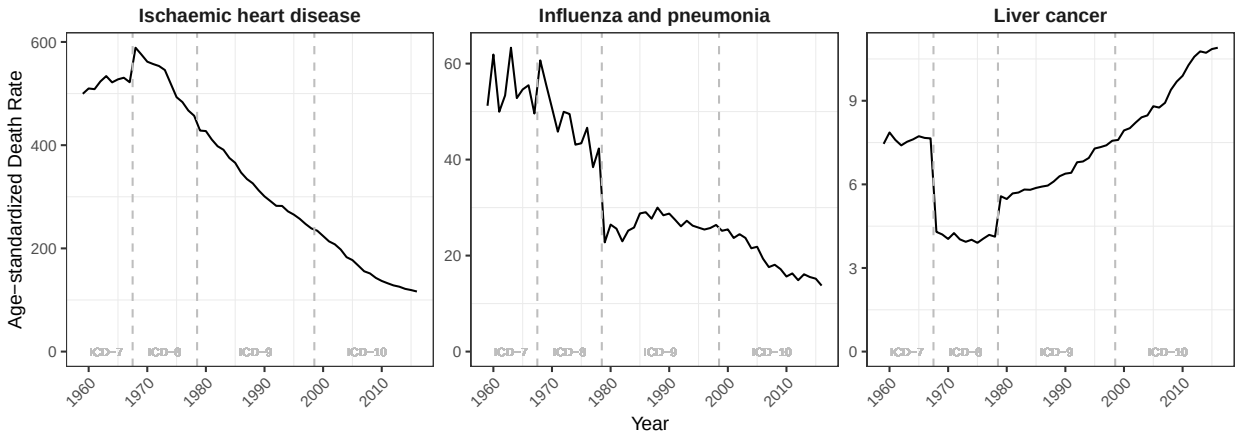


Figure 5.5: Age-standardized death rate for samples causes of death, 1959–2016, males



These differences may lead to conflicting interpretations of the parameters estimates and hence we refrain from using the APCi and PLATi models for our subsequent analysis. Accordingly, we choose to focus our discussion on the PCi model which does not suffer from such identification issues facilitating the interpretation of the parameter estimates.

5.3 SMOOTHNESS AND CODING CHANGES

Before proceeding to apply the PCi model to decompose mortality improvements for the different causes of death there are two further issues that need attention. First, as discussed before in the descriptive analysis, changes in the cause of death classification systems can induce disruptions in mortality trends for certain causes. Second, in Figures 5.2, 5.3 and 5.4 we can see that period and cohort improvements show significant year on year variability. Therefore, to facilitate the recognition and interpretation of general trends, it would be useful to impose some smoothness on the parameters estimates of the models.

5.3.1 CODING CHANGES

In Section 4.1 we saw that the changes in the ICD regimes may induce important disruptions in the mortality trends of some causes of death which would need to be controlled for in our modeling. As an example, Figure 5.5 shows the ASDR for males from ischaemic heart disease, influenza and pneumonia, and liver cancer. These causes are representative of the types of trends disruptions produced by the changes in cause of death coding.

We see that the change from ICD-7 to ICD-8 induced a small but clear disruption in mortality trends for ischaemic heart disease. For influenza and pneumonia, it is the change from ICD-8 to ICD-9 which induced a disruption, while for liver cancer both the changes from ICD-7 to 8 and from ICD-8 to 9 induced a discontinuity in the mortality trends. In order to account for these possible discontinuities, we first need to identify for each cause of death which ICD

coding changes induced a disruption, and, second, we need to allow for these disruptions in our mortality improvement rate models.

5.3.1.1 DETECTION OF ICD CODING DISRUPTIONS

To automatically detect which coding changes matter for each cause of death and risk factors, we follow an adapted version of the statistical methods proposed by Rey et al. (2011) as described in Appendix D. The results of this analysis are shown in Tables D.1 and D.2 in the appendix. For example, in a consistent manner with the clear disruption seen in Figure 5.5, for ischaemic heart diseases the statistical approach detected a significant disruption in mortality trends from this cause as a result of the change from ICD-7 to ICD-8 with the comparability ratio for male mortality from this cause estimated to be around 1.12. That is, one death from ischaemic heart disease reported during ICD-7 is equivalent to 1.12 deaths during ICD-8.

From Tables D.1 and D.2 we conclude that the majority of disruptions in the trend occurred for the change from ICD-8 to ICD-9. By contrast, there are far fewer coding disruptions in the change from ICD-7 to ICD-8. Despite the fact that our data has used bridge coding to correct for disruptions between ICD-9 and ICD-10, there are some causes for which a further adjustment, albeit small, is required.

5.3.1.2 ICD CODING DISRUPTIONS IN THE PCI MODEL

Having identified for each cause of death the important coding disruptions, the next step is to allow for these disruptions in our improvement rate modeling. To do so, recall from Table 5.1 that the PCi improvement rate model is equivalent to a standard age-period-cohort model for mortality rates:

$$\log m_{x,t} = A_x + K_t + \Gamma_{t-x}. \tag{5.7}$$

Assume that the procedure outlined in Appendix D identified that for a given cause there are significant disruptions at years $s_1, s_2, \ldots, s_h$. To control for these disruptions, we extend the APC model in equation (5.7) so that:

$$\log m_{x,t} = A_x + K_t + \Gamma_{t-x} + \sum_{i=1}^{h} \delta_i f^{(i)}(t), \tag{5.8}$$

where $f^{(i)}(t) = \mathcal{I}_{\{s_{i-1} \leq t < s_i\}}$, $i = 1, \ldots, h$, is the indicator function taking value 1 if $t \in [s_{i-1}, s_i)$ with the convention that s_0 is the first year where data is available. In this extended model, the δ_i parameters captures the magnitude of the possible jumps in the mortality trend arising from changes in coding regimes. The details of the estimation of this model are described in Appendix D.

5.3.2 SMOOTHNESS

Most stochastic mortality models including the APC model in Equations (5.7) and (5.8) fall within the category of generalized linear models (GLM) (Currie, 2016). Generalized additive models (GAMs) offer an alternative to GLMs to obtain smooth estimates of the age, period and cohort effects (Dodd et al., 2020). Thus, we also consider the following alternative GAM formulation of the APC model with coding changes:

$$\log m_{x,t} = S_A(x) + S_K(t) + S_\Gamma(t-x) + \sum_{i=1}^{h} \delta_i f^{(i)}(t), \tag{5.9}$$

where $S_A(x)$, $S_K(t)$ and $S_\Gamma(t-x)$ are smooth functions representing the age, period and cohort effects respectively. This model can easily be estimated in `R` using the **MGCV** `R` package (Wood, 2011, 2020). In our implementation, we assume that $S_A(x)$, $S_K(t)$ and $S_\Gamma(t-x)$ are thin plate penalized regression splines with their degree of smoothness determined automatically using generalized cross-validation.

5.4 ILLUSTRATION OF RESULTS

Before presenting the results of the age-period-cohort decomposition of the different causes of death and risk factors, in this section we illustrate for selected causes of death the application of the methods discussed in Section 5.3.

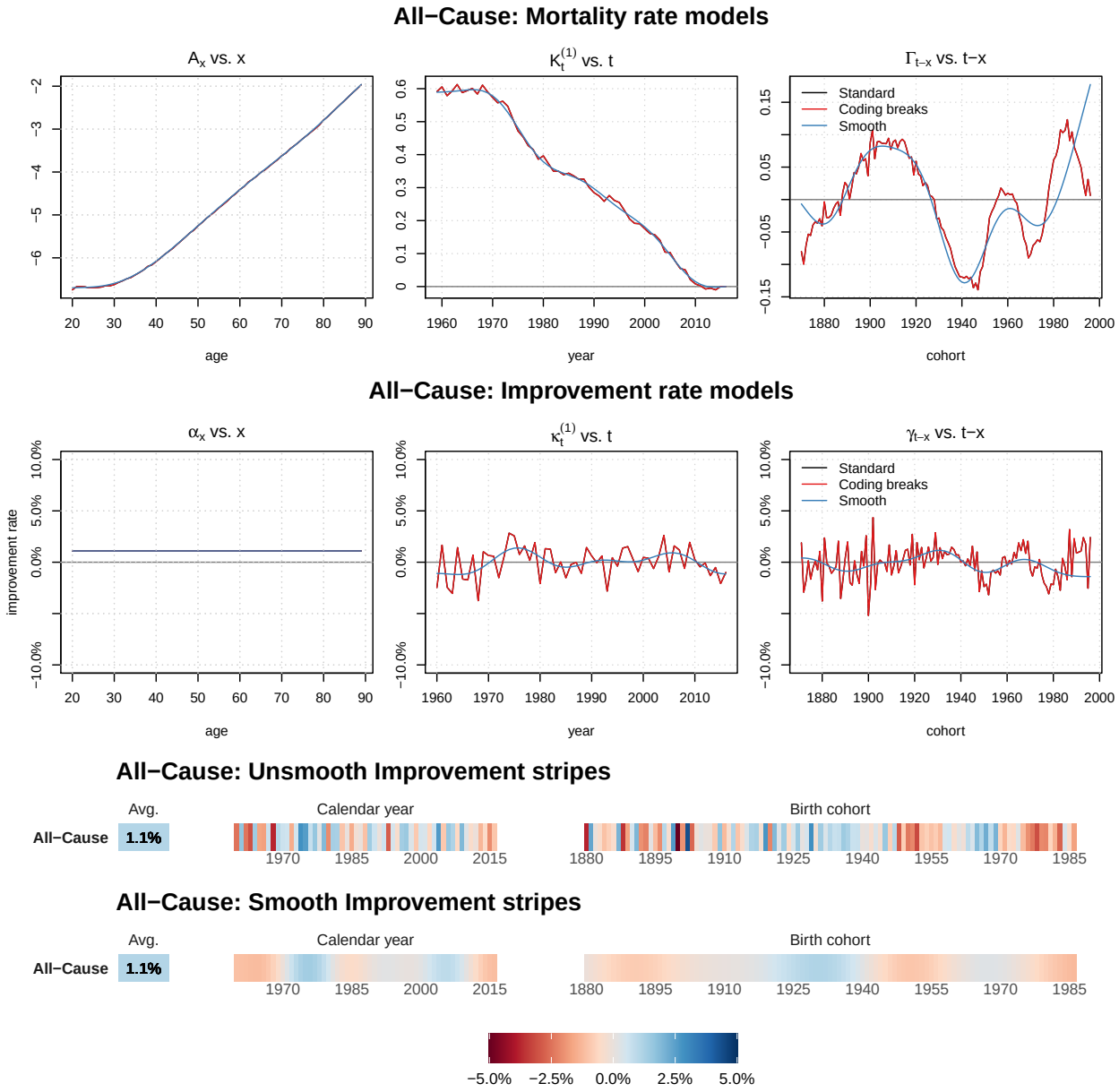
As illustration, Figure 5.6 presents the results of applying the different versions of the PCi model to male data mortality for all-causes of death. Similarly, Figures 5.7, 5.8 and 5.9 show matching results for ischaemic heart disease, influenza and pneumonia, and liver cancer, respectively. These three causes of death, which are the same presented before in Figure 5.5, help to highlight how the models deal with the effects of coding changes and with smoothing out the year on year variability of the period and cohort effects.

For each cause we show the parameter estimates associated with the "standard" PCi model defined by Equation (5.7), the PCi model allowing for "coding breaks" defined by Equation (5.8), and the "smooth" PCi model defined by Equation (5.9). For each of the models we present the fitted parameters in the mortality rate scale (top plots) and in the improvement rate scale (middle and bottom plots). Moreover, for the parameters in the improvement rate scales, we present two type of plots: traditional line plots and *mortality improvement stripes*. The latter type of plot are inspired by the "warming stripes" designed by Ed Hawkings to communicate temperatures change across the globe over the past century.[3]

When presenting improvement rate parameters and in order to avoid possible identification issues associated with assigning main mortality improvements to period or cohort, we have reparameterized the PCi models in the following form:

[3]See http://www.climate-lab-book.ac.uk/2018/warming-stripes/ and https://showyourstripes.info/

Figure 5.6: Fitted parameters for the PCi model for all-causes of death, males , 1959–2016, 20–89



$$\log \frac{m_{x,t}}{m_{x,t-1}} = \alpha + \tilde{\kappa}_t + \tilde{\gamma}_{t-x}, \tag{5.10}$$

with $\alpha = \frac{\sum_x \sum_t (\kappa_t + \gamma_{t-x})}{\sum_x \sum_t 1}$, $\tilde{\kappa}_t = \kappa_t - \frac{\sum_t \kappa_t}{\sum_t 1}$ and $\tilde{\gamma}_{t-x} = \gamma_{t-x} - \frac{\sum_x \sum_t \gamma_{t-x}}{\sum_x \sum_t 1}$. In this parameterization, α is interpreted as the average mortality improvement across all the data cells, and $\tilde{\kappa}_t$ and $\tilde{\gamma}_{t-x}$ as the period and cohort deviations from this average improvement.

Figure 5.6 shows the results for all-cause mortality for males. These illustrative results are

indicative of the type of discussion we can derive from the results of the different versions of the PCi model.

In the top row of Figure 5.6, we see that the parameters for the "standard" PCi model and the model with coding breaks coincide. This is because ICD coding changes do not affect mortality rates from All-causes of death. We also see that the "smooth" version of the model follows closely the patterns of the standard model while smoothing out the the year on year fluctuations in the period effect, K_t, and in the cohort effect, Γ_{t-x}.

The middle row of Figure 5.6 presents the parameters in the improvement scale which are obtained by taking the first difference of the parameters in mortality rate scale (recall Equation (5.3)). The advantages of the smoothing for the recognition of trends become more apparent in the improvement rate scale where year on year fluctuations are more evident.

The bottom row of Figure 5.6 depicts the improvement rate stripes which are a more compact away of presenting the improvement rate parameters α, $\tilde{\kappa}_t$ and $\tilde{\gamma}_c$. In the improvement stripes, the left panel labeled *"Avg."* represent the parameter values of α, the middle panel labeled *"Calendar year"* the period effects as captured by $\tilde{\kappa}_t$, and the right panels labeled *"Birth cohort"* the cohort effects, $\tilde{\gamma}_c$. The unsmooth improvement stripes corresponds to the red "coding break" lines in the middle row line plots, while the smooth stripes correspond to the blue "smooth" lines in the middle row plots. Similarly to the heatmaps of Section 4.3, the values of the PCi model are mapped to a color so that blue hues signify mortality improvements and red hues mortality deterioration. We note that in the improvement rate stripes we have capped very large improvements and deteriorations at $\pm 5\%$ p.a. so that very intense reds represent a deterioration of more than 5% p.a. and very intense blues represent improvements of more than 5% p.a. We also note that in the improvement stripe plot we have not included the parameters values of the cohort improvement deviations, $\tilde{\gamma}_{t-x}$, for the ten older and younger cohorts. These corner cohorts have only a limited number of observations which can lead to erratic parameter estimates.

From Figure 5.6 we can note the following interesting points. All-cause mortality from males improved between 1959 and 2016 at an average pace of 1.1% p.a. There are also clear period effects with mortality improving faster than average in the 1970s and late 2000s and slower than average in the 1960s, early 1980s and after 2010. Moreover, there are some clear cohort effects with the interwar cohorts born between 1920 and 1940 having above average improvements and the cohorts born between 1945 and 1955 having worse than average improvements.

Figure 5.7 plots the results of fitting the PCi model to mortality rates from ischaemic heart disease (IHD). In the top row we see that allowing for coding breaks – either through model (5.8) or model (5.9) – effectively removes the discontinuity in the mortality trends induced by the change from ICD-7 to ICD-8. In the middle and bottom plots, we see clearly that period effects are an important feature of mortality rates from IHD: mortality improved significantly slower than average before 1970 and after 2010 and faster than average in the 2000s. Although, cohort effects are less prominent, there are clear above average improve-

Figure 5.7: Fitted parameters for the PCi model for mortality from ischaemic heart disease, males , 1959–2016, 20–89

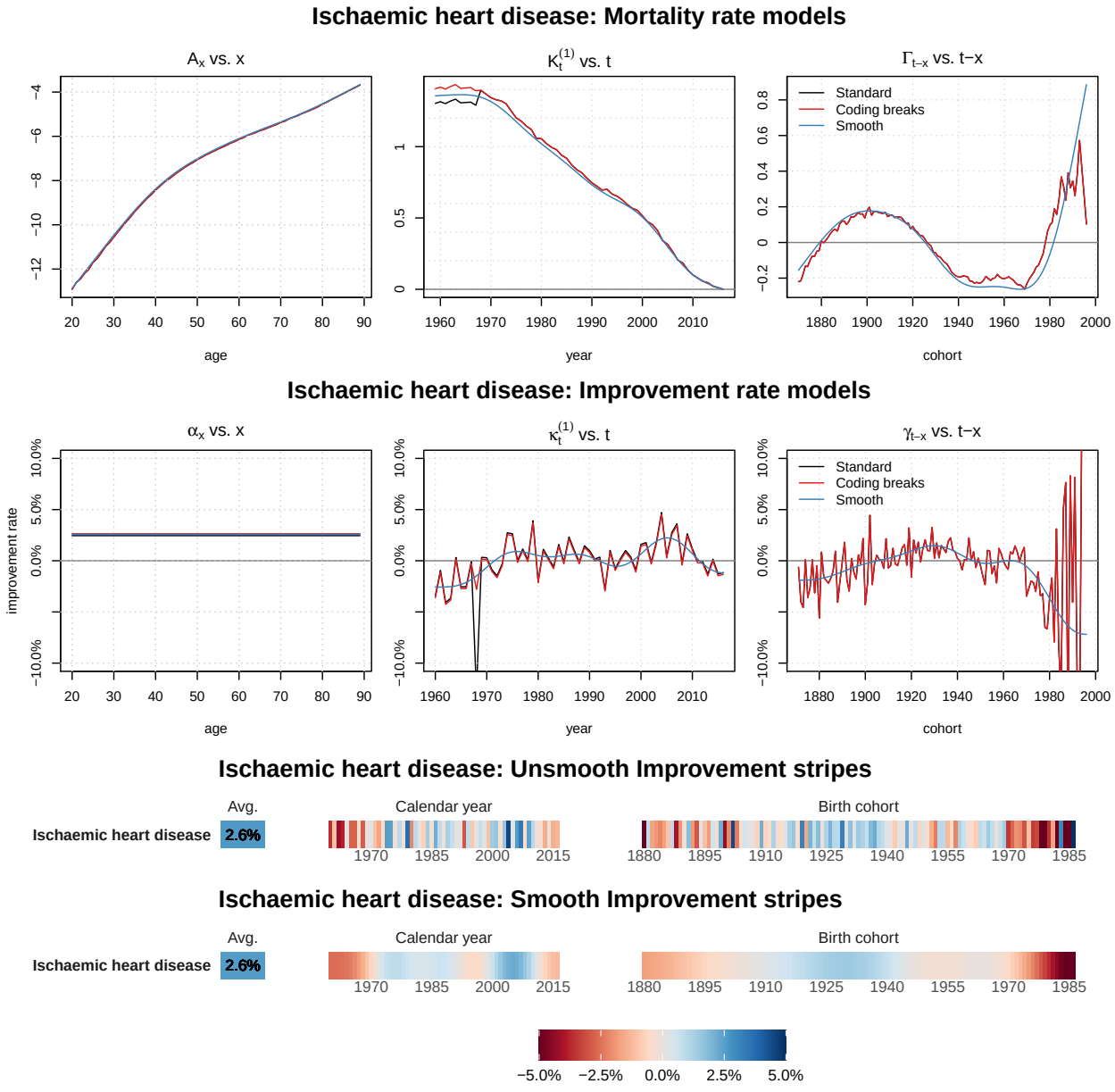
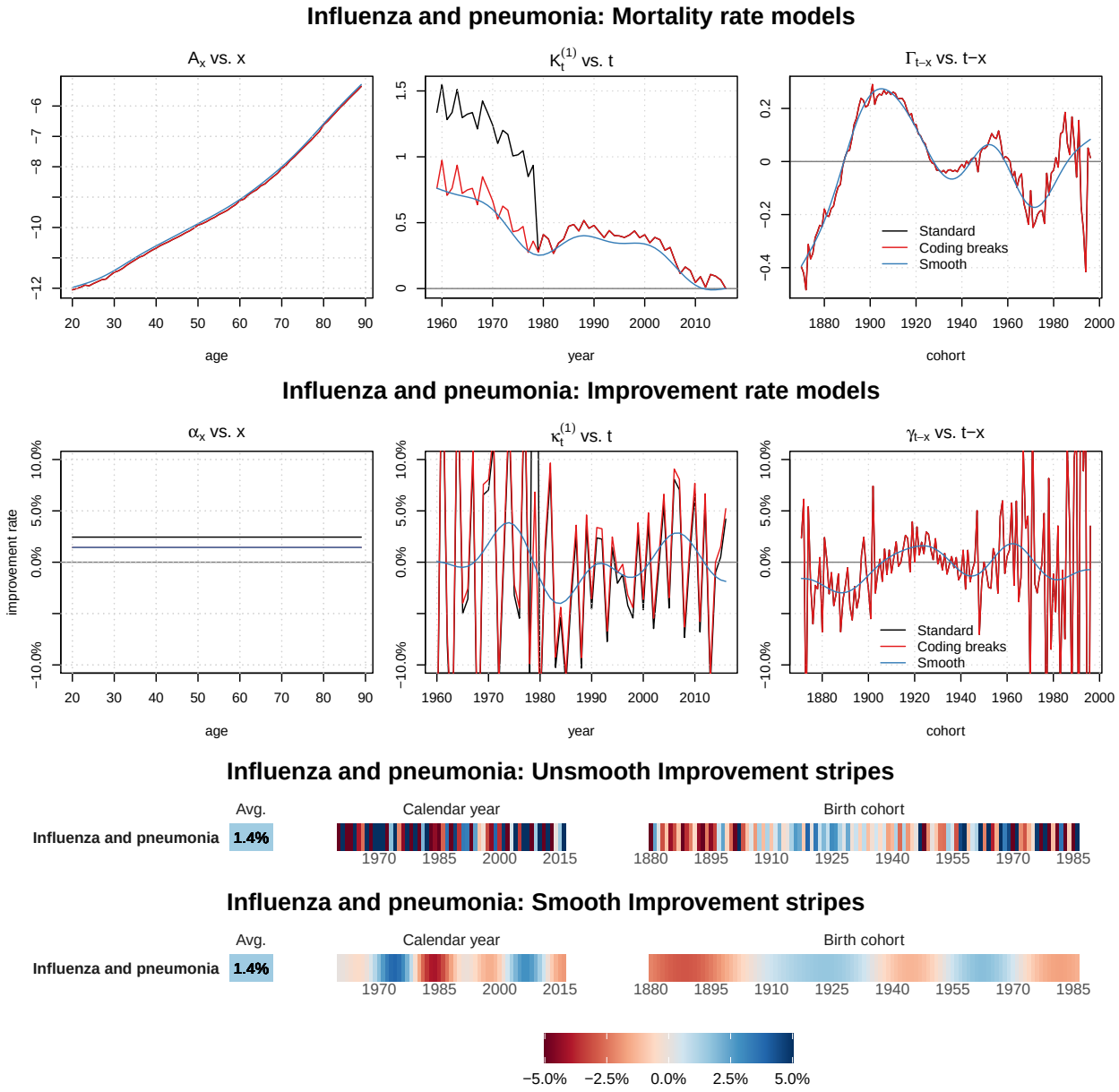


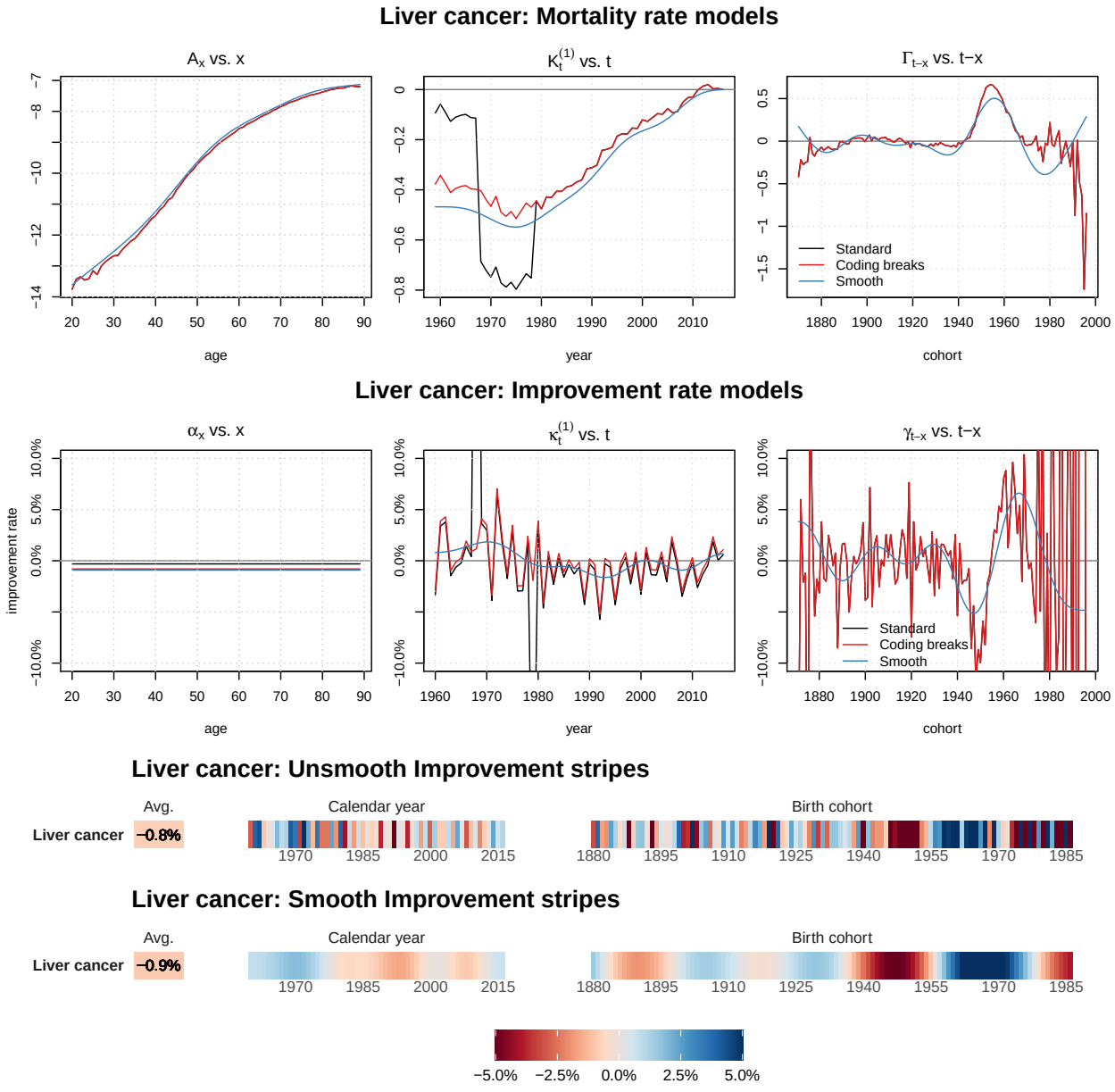
Figure 5.8: Fitted parameters for the PCi model for mortality from influenza and pneumonia, males , 1959–2016, 20–89



ments for the 1920-1940 cohorts as indicated by the blue hues in the birth cohort panes of the improvement stripe plots.

Figure 5.8 depicts the parameter estimates of the PCi model for male mortality from influenza and pneumonia. Again, we see that the models with coding breaks correct the very clear jump in mortality rates rates during the transition from ICD-8 to ICD-9. Mortality rates from influenza and pneumonia tend to exhibit very extreme year on year fluctuation in their mortality rates, which makes it difficult to identify secular trends in the unsmoothed

Figure 5.9: Fitted parameters for the PCi model for mortality from liver cancer, males , 1959–2016, 20–89



parameters. By contrast, the smoothed parameters unveil very clear period effects with mortality improving faster than average in the 1970s and 2000s and slower than average between 1980 and 2000 and more recently after 2010. Albeit less pronounced, there are clear negative cohort effects for the 1940-1950 cohorts and above average mortality improvements for the 1950-1970 cohorts.

Finally, Figure 5.9 shows the result of fitting the different versions of the PCi model to male mortality rates from liver cancer. After the correction of coding discontinuities and during the 1959-2016 period, mortality from this cause of death deteriorated at a pace of around 0.8% p.a. In addition, the clearest feature of liver cancer mortality is the very negative cohort effects for the 1940-1955 birth cohorts which contrast with the above average improvement for younger cohorts born between 1955 and 1970.

For completeness, figures corresponding to Figures 5.6-5.9 for each causes of death and risk factor are presented in Appendix F for females and in Appendix G for males.

5.5 RESULTS

We now show the results of applying the smooth PCi models to causes of death at the different level of disaggregation as well as the results of applying this methodology to causes of death grouped according to risk factors. These results are shown in Figures 5.10 and 5.11 for causes of death and in Figure 5.12 for risk factors. Matching pictures without smoothing are presented in Appendix E.

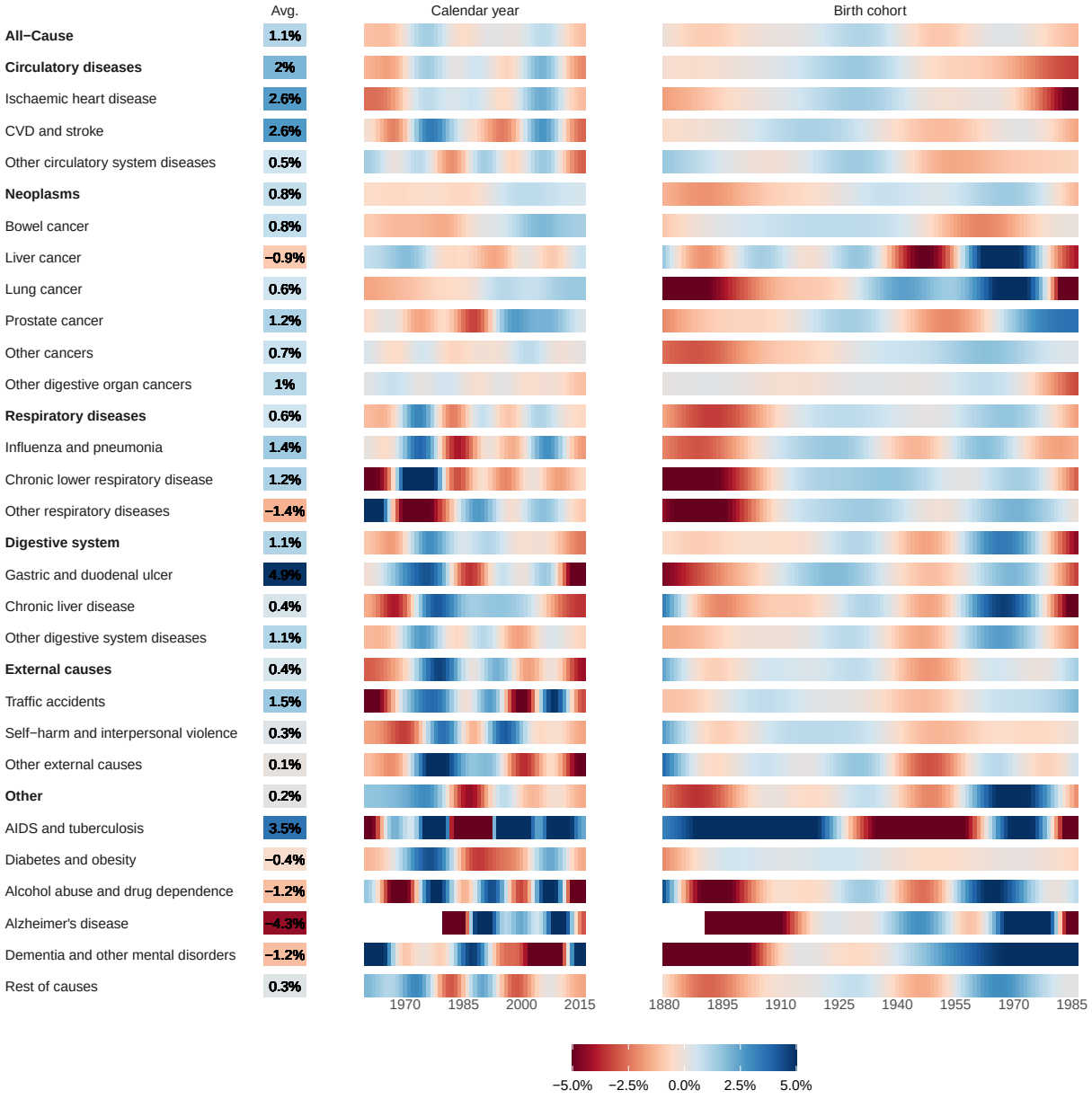
In the left panels of Figures 5.10 and 5.11, we see that in the period between 1959 and 2016 most causes of death experienced on average an improvement in mortality. The faster improvements occurred for circulatory diseases as a whole, IHD, CVD and stroke, gastric and duodenal ulcer, and AIDS and tuberculosis, where mortality for both genders improved at an average pace of more than 2% p.a. There are, however, some causes of death which are the exception and which experienced an overall deterioration of mortality. Particularly noteworthy are the sharp deteriorations in both male and female mortality from alcohol abuse and drug dependence, Alzheimer's disease, and dementia and other mental disorders. Other causes where mortality deteriorated include liver cancer and diabetes for males, lung cancer for females, and other respiratory diseases for both genders.

We see in the middle panes of Figures 5.10 and 5.11 that most causes of death exhibit a clear period effect with similar patterns observed in females and males. We can identify the following five regimes in the period evolution of mortality:

- **Period 1959-1970:** During this period all-cause mortality improved very slowly for women and stagnated for men as a result of the below average improvements (or deterioration) in mortality for the majority of the causes. IHD, lung cancer, traffic accidents and chronic liver disease stand out as the causes with the most noticeable negative period effects.

Figure 5.10: Smooth improvement stripes for causes of death, Females, 1959–2016, 20–89

Figure 5.11: Smooth improvement stripes for causes of death, Males, 1959–2016, 20–89



- **Period 1970-1980:** During this decade all-cause mortality rates experienced a clear acceleration in mortality improvement derived from an above average mortality improvement for all broad (Level 1) causes of death with the exception of neoplasms. Among the more detailed (Level 2) causes the fastest rate of mortality improvement occurred for CVD and stroke, influenza and pneumonia, chronic lower respiratory diseases, and traffic accidents.
- **Period 1980-1995:** This period was characterized by a deceleration in the pace of improvement for all-cause mortality. This deceleration was mainly driven by the dramatic worsening of mortality from AIDS and Tuberculosis resulting from the HIV epidemic. During this period, mortality from influenza and pneumonia, chronic lower respiratory diseases, and gastric and duodenal ulcer also exhibited clear negative period effects.
- **Period 1995-2010:** This period saw a return to faster than average all-cause mortality improvements which were mainly driven by faster improvements from circulatory diseases and from AIDS and tuberculosis.
- **Period 2010-2016:** Finally, the most recent period has seen a clear deceleration of mortality improvements in all-cause mortality for both genders. This slowdown is the result of below average improvement (or even deterioration) in mortality from several causes among which circulatory diseases, diseases of the digestive system, and alcohol abuse and drug dependence stand out.

In contrast to period effects where both genders have similar patterns, we see in the right panes of Figures 5.10 and 5.11 that there are contrasting patterns in the mortality improvements of females and males along the cohort dimension. In particular, the brighter intensity of the reds and blue stripes for males indicate more marked cohort effects for men than women. In addition, the boundaries of the cohort groups with similar behavior differ slightly among the genders. While for men we can identify cohort groups encompassing the generations born in 1920-1940, 1940-1955 and 1955-1970, for women these cohort groups are 1925-1945, 1945-1960, 1960-1975. Since cohort patterns are more prominent for men than women, we deviate from the order we have followed in the rest of this report and discuss first the features of the male birth cohort groups seen in Figure 5.11:

- **Male generation of 1920-1940:** The interwar generation of American men has experienced above average mortality improvements for most causes of death. This is particularly noticeable for mortality from circulatory, respiratory, and digestive diseases. In addition, mortality improvements for some neoplasms including liver and prostate cancer show important positive cohort effects.
- **Male generation of 1940-1955:** This generation includes the early baby boomers who are characterized by below average mortality improvements. The very clear negative cohort effects for liver cancer, digestive diseases, alcohol abuse and drug dependence, and external causes suggest that alcohol and drug consumption may be a driving

factor of the below average improvements for this generation. Effectively, in Figure 5.12 we see a very clear negative effect in mortality associated with alcohol abuse and drug dependency. In addition, this generation, which was in their young adulthood during the HIV epidemic, shows very significant negative cohort effects in mortality from AIDS and tuberculosis.

- **Male generation of 1955-1970:** The generation of late baby boomers and early Gen X men shows average mortality with no distinctive all-cause positive or negative cohort effect. However, in sharp contrast to the 1940-1955 generation, in Figure 5.12 we see that this generation has above average improvements for causes linked to alcohol abuse and drug dependency as well as above average improvements for AIDS and tuberculosis.

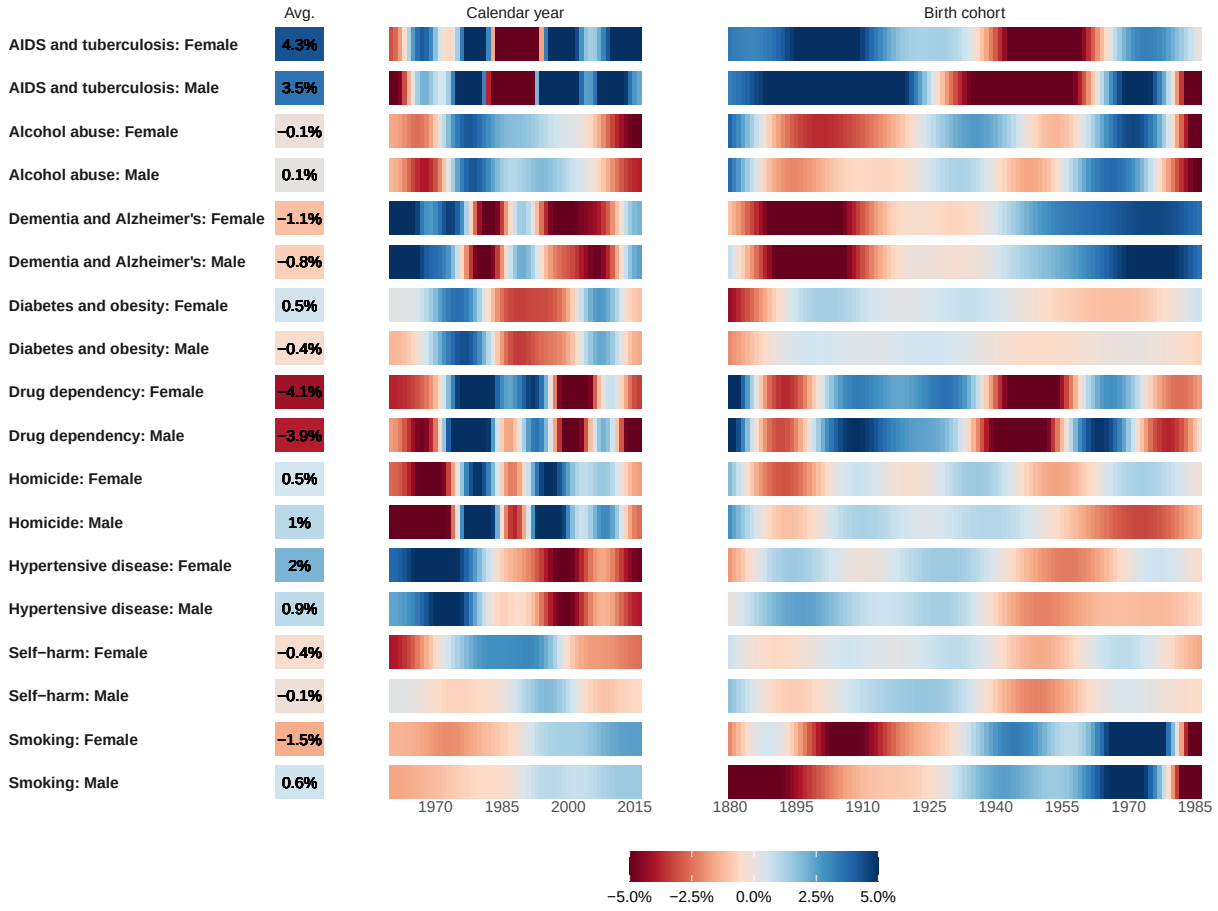
For women we see in Figure 5.10 the following features among the birth cohort groups:

- **Female generation of 1925-1945:** In comparison to the matching generation of men, the generation of interwar women starts slightly later and has less marked above average improvements. Nevertheless, the drivers of this above average improvements are very similar to those of men with noticeable positive cohort effects for digestive system diseases and external causes.
- **Female generation of 1945-1960:** For women from the early baby boomer generation we see, as with men, that they have experienced below average mortality. However, in contrast to men, women from this generation do not show a clear cohort pattern in mortality improvements from digestive diseases and liver cancer. They do show, however, a rather marked negative cohort effect in mortality from drug and alcohol abuse that spans the generation of women born between 1940 and 1955. Similarly to men, women from this generation also show a negative cohort effect in mortality from AIDS and tuberculosis. However, when compared to men, this effect is slightly less pronounced and spans a shorter generational group.

- **Female generation of 1960-1975:** Similar to men, women from this younger generation do not show clear positive or negative cohort effects in all-cause mortality. This is explained by the contrasting patterns seen for different causes. This generation shows noticeable positive cohort effects associated with lung cancer, chronic lower respiratory disease, chronic liver disease and most subcauses within the Level 1 group of other causes of death. These positive cohort effects have nonetheless being counteracted by large negative effects in mortality from IHD.

Figure 5.12 presents the improvement stripes for risk factors for both females and males. For most risk factor we see consistent period patterns among women and men, with the noticeable exceptions of drug dependency and self-harm which show some dissimilar pattern among the

Figure 5.12: Smooth improvement stripes for risk factors, 1959–2016, 20–89



genders between 1970 and 1990. Consistent with the patterns seen for the different causes of death along the cohort dimension, most risk factors for women show cohort patterns which are shifted relative to the patterns for men. In Figure 5.12, we can identify the following more specific features for each of the risk factors:

- **AIDS and tuberculosis:** In the *Calendar year* panel we see a fast deterioration in mortality in the 1980s followed by rapid improvements from 1990 coinciding, respectively, with the onset of the HIV epidemic and the introduction of anti-retroviral treatment. The very intense red and blue hues in the improvement stripes for both men and women would suggest period effects of very similar magnitude in the two genders. However, this is the result of our decision to cap extreme improvements and deteriorations at 5% in the graphs. By contrast, the line plots at the top of Figures F.32 and G.32 in the Appendix, where the period effects are uncapped, show that the impact of the HIV epidemic is far more prominent for men than for women. There are very marked negative cohort effects for the generation of men and women who were in their early and middle adulthood in the 1980s during the peak of the HIV epidemic.

For men, this negative cohort spans those born between 1925 and 1960 covering men who were in their late 50s in the 1980s. By contrast, this negative cohort effect is confined to younger women born between 1935 and 1960. Finally, there is a noticeable negative cohort effect for young men born between 1980 and 1985 which is not matched in women.

- **Alcohol abuse:** For both genders mortality linked to alcohol abuse experienced nearly no overall improvement nor deterioration between 1959 and 2016. Nevertheless, there are clear period patterns with mortality for both genders deteriorating before 1970 and improving between 1970 and 2000. More recently, from the turn of the millennium in 2000, mortality for the causes associated with this risk factor started to deteriorate with women being more affected than men. There are also clear cohort patterns in alcohol abuse in both genders, albeit the patterns for females are a delayed version of those of men. There is a marked negative cohort effect affecting the generation of men born in 1940-1955 which is matched by a negative cohort effect for the slightly younger women born in 1945-1960. In contrast, the generations of men born in 1955-1975 and of women born in 1960-1980 show very clear positive cohort effects.

- **Drug dependency:** Between 1959 and 2016, mortality for causes related to drug dependency saw a very marked deterioration with women and men experiencing an average deterioration of 4.1% and 3.9% p.a., respectively. This deterioration slowed down between 1970 and 1995, where mortality rates for causes linked to drug dependency remained stable. However, there was a brief period in the mid 1980s, coinciding with the so called "crack epidemic"(Hartman and Golub, 1999), during which this slowdown was interrupted. Similarly to alcohol abuse, drug dependency is characterized by strong cohort effects. The generations of men born in 1930-1950 and women born in 1935-1955 show very significant negative cohort effects which are in sharp contrast with the positive cohort effects of younger men and women born in 1950-1970 and 1955-1975, respectively. Noticeably, the youngest generations of men born after 1970 and of women born after 1975 show marked negative cohort effects.

- **Dementia and Alzheimer's disease:** Females and males show very similar mortality improvement patterns for dementia and Alzheimer's disease, with both genders characterized by an average deterioration of about 1% p.a between 1959 and 2016. Nevertheless, from 1959 to 1975 mortality from this risk factor experienced a clear improvement. From 1975 to 2012, there are clear negative period effects with a very short period of interruption around 1990. Noticebly, after 2012 there is a period of positive period effects consistent with the slight decline in mortality rates from this risk factor seen previously in the corresponding age-standardized death rates (see Figure 4.9). Along the cohort dimension, there are contrasting patterns for Americans born before and after 1940, with the older generation showing a negative cohort effect and the younger generations a positive cohort effect. However, the heatmaps in Figures 4.27 and 4.28 showed that this risk factor had very clear age effects, and, hence, these

cohort patterns need to be interpreted with care as the may be acting as a substitute for the lack of age effects in our PCi models.

- **Diabetes and obesity:** Between 1959 and 2016 women and men showed contrasting general trends in mortality rates from diabetes and obesity: while women experienced an average mortality improvement of 0.5% p.a., men experienced an average mortality deterioration of 0.4% p.a. In spite of this dissimilar overall trend, mortality improvements for both genders are dominated by very marked period effects with cohort effects being almost non-existent. This pattern is consistent with the predominant vertical striation seen before in the improvement rate heatmaps for this risk factor (see Figures 4.27 and 4.28). Both genders exhibit clear positive period effects between 1965 and 1980 which are followed by clear negative period effects between 1980 and 2000. During the new millennium positive effects have predominated, although there is a noticeable reversal in 2010 when negative period effect have started to arise.

- **Hypertensive disease:** Mortality rates from this risk factor for women and men improved between 1959 and 2016 at an average pace of 2% and 0.9% p.a., respectively. This average improvement was accompanied by period and cohort effects which show consistent patterns in both genders. Period effects show a noticeable change in trend around 1980 when mortality rates linked to this risk factors transitioned from positive period effects to negative period effects. Similarly, cohort effects for both females and males exhibit a change from positive cohort effects for the generation born around 1940 to a negative cohort effect for the generations born thereafter. A noticeable deviation from this alignment between the genders, are the faint, but clear, positive cohort effects observed in the generation of women born in the 1970s which is not matched by a similar cohort effect in men.

- **Homicide:** Mortality trends from homicide for both genders are dominated by period effects. From 1959 through 1975 there is a clear negative period effect indicating a significant deterioration of homicide mortality rates during this period. After 1975 and until 2010 this trend has been mostly reversed as indicated by the predominantly blue hues seen in the *Calendar year* panel of Figure 5.12 during this period. Nevertheless, there was a brief but clear period of negative period effects between 1985 and 1990 consistent with the dramatic rise in homicide rates during the later half of the 1980s to reach a peak in 1991 (Blumstein et al., 2000; Cooper and Smith, 2011). Interestingly, the negative period effect for homicide rates between 1985 and 1990 matches a similar negative period effect for drug dependence which is consistent with the fact that the rise of homicide rates in the late 1980s was mainly driven by gang violence linked to the emerging crack market (Blumstein, 1995). More recently, after 2010, homicide rates show a clear deterioration, but have still stayed far below the rates seen in the 1980s (Rosenfeld and Fox, 2019).

- **Self-harm:** Contrary to homicide which is dominated by period factors, self-harm

shows both period and cohort effects. However, men and women have contrasting patterns along the calendar year dimension with women showing far stronger period effects than men. In particular, we see that between 1959 and 1970 women show a strong negative period effect which is not present in men. After this period of deterioration, there is for women a period of stark positive period effects ending in 2000. For men, by contrast, the positive period effects have a much delayed onset starting only in 1985 but lasting also until 2000. For both genders the turn of the century is characterized by noticeable negative period effects linked to the dramatic increase in suicide rate from 1999 through 2018 (Hedegaard et al., 2020). Finally, both genders exhibit a clear negative cohort effect for the 1940-1960 generation which is consistent with the shift in cohort patterns in suicide rates which began to rise sharply starting from the baby boomer generation and continued for subsequent generations (Phillips, 2014).

- **Smoking:** For smoking we see that both period and cohort effects are present, but with cohort effects being far more marked. Along the period dimension, we see for both genders a clear change around 1990 shifting from a negative period effect to a positive period effect. As expected, cohort effects for this risk factor are very noticeable for both genders with the oldest cohorts showing a negative cohort effects and the younger cohorts a positive one. Interestingly, the change from negative to positive cohort effects for males occurs around the 1920 birth cohort while for women this takes place at around the sightlier younger cohort born in 1930. These sex differentials are consistent with the fact that cigarette consumption for men peaked for the 1910-1925 birth cohorts while for women this occurred for the 1925-1940 birth cohorts (Preston and Wang, 2006; Wang and Preston, 2009).

Section 6: A summary of mortality change in the U.S., 1959–2016

In this section we aim to bring together the results from the previous sections to construct a narrative of mortality change in the U.S. between 1959 and 2016, emphasizing the key drivers of this change and the features that characterize different time periods and generations.

6.1 THE MAIN STORY OF MORTALITY IMPROVEMENTS OVER SIXTY YEARS

By way of summary, Table 6.1 shows the contribution of different causes of deaths to the change in life expectancy at age 20 between 1959 and 2016 obtained using the decomposition approach introduced in Section 4.2; alongside the cause-specific average improvement rates over the same period derived from the smooth PCi models discussed in Sections 5.3 and 5.5.

Over the study period, the U.S. saw a remarkable reduction in mortality rates, with rates of mortality for both women and men aged 20-89 improving at an average pace of around 1% p.a. This resulted in period life expectancy at age 20 increasing from 55.75 years to 62.06 for females and 49.82 years to 57.26 years for males between 1959 and 2016.

While the first half of the 20th century was dominated by improvements in mortality from infectious diseases, the story of mortality evolution in the U.S. in the second half of 20th century which continued into the first decade of the 21st century has been mainly a story of mortality improvements from circulatory diseases. In fact, in Table 6.1 we see that 6.29 years of the 6.31 years of increase in life expectancy at age 20 for females over the study period can be attributed to reductions in mortality rates from circulatory causes of death. Overall, the increase in life expectancy for men was larger than for women, but the magnitude of the gain attributable to a decline in circulatory diseases (6.23 years) was of a similar scale.

The improvement of mortality from circulatory diseases can be linked to both a delayed onset of disease due to improvements in the prevalence of cardiovascular risk factors such as hypertension, high cholesterol and smoking and reductions in case fatality with the introduction of novel surgical and pharmacological treatments (Mensah et al., 2017). For ischaemic heart disease in particular, Ford et al. (2007) estimate that 47% of the decrease in mortality rates between 1980 and 2000 can be attributed to improved uptake of evidence-based medical and surgical treatments while the net reduction in the prevalence of major risk factors accounted for 44% of the decrease.[1] Notably, Ford et al. (2007) reports that increases in body mass index and diabetes partially have offset some of the gains.

[1] Among treatments, Ford et al. (2007) report that 11% is attributed to "secondary therapies after myocardial infarction of revascularization", 10% to "initial treatments for acute myocardial infarction or unstable angina, 9% to treatment for heart failure and 5% to revascularization for chronic angina. For risk factors, Ford et al. (2007) report that 24% is attributed to reductions in total cholesterol, 20% to reductions in systolic blood pressure, 12% to reductions in smoking prevalence and 5% to reductions physical inactivity

Table 6.1: Summary of contribution to life expectancy change at age 20 and average improvement rates for the period 1959-2016

	Life Expectancy Change		Improvement rate	
Cause	**Female**	**Male**	**Female**	**Male**
All-Cause	**6.31**	**7.44**	**1.04%**	**1.1%**
Circulatory diseases	**6.29**	**6.23**	**2.21%**	**2.01%**
Ischaemic heart disease	3.34	4.31	2.63%	2.58%
CVD and stroke	1.65	1.08	2.97%	2.65%
Other circulatory system diseases	1.3	0.84	1.05%	0.55%
Neoplasms	**0.85**	**1.04**	**0.82%**	**0.78%**
Bowel cancer	0.28	0.18	1.25%	0.8%
Liver cancer	0.03	-0.06	0.45%	-0.91%
Lung cancer	-0.32	0.32	-1.51%	0.6%
Breast cancer	0.25	0	1.03%	-
Prostate cancer	0	0.13	-	1.17%
Other cancers	0.5	0.25	1.13%	0.68%
Other digestive organ cancers	0.12	0.21	0.85%	0.98%
Respiratory diseases	**-0.23**	**0.27**	**-0.38%**	**0.56%**
Influenza and pneumonia	0.27	0.31	1.16%	1.44%
Chronic lower respiratory disease	-0.4	-0.03	-0.11%	1.2%
Other respiratory diseases	-0.11	-0.01	-2.01%	-1.4%
Digestive system	**0.18**	**0.27**	**1.15%**	**1.14%**
Gastric and duodenal ulcer	0.05	0.12	3.93%	4.93%
Chronic liver disease	0.03	0.06	0.24%	0.41%
Other digestive system diseases	0.1	0.09	1.36%	1.12%
External causes	**-0.13**	**-0.08**	**0.05%**	**0.44%**
Traffic accidents	0.14	0.44	1.36%	1.51%
Self-harm and interpersonal violence	-0.01	-0.11	0.01%	0.3%
Other external causes	-0.26	-0.42	-0.43%	0.05%
Other	**-0.66**	**-0.29**	**0.18%**	**0.17%**
AIDS and tuberculosis	0.06	0.12	4.28%	3.55%
Diabetes and obesity	0.1	-0.09	0.53%	-0.35%
Alcohol abuse and drug dependence	-0.03	-0.04	-2.9%	-1.18%
Alzheimer's disease	-0.34	-0.12	-6.47%	-4.31%
Dementia and other mental disorders	-0.29	-0.13	-0.72%	-1.16%
Rest of causes	-0.16	-0.02	0.16%	0.27%
Risk factors				
AIDS and tuberculosis			4.28%	3.55%
Smoking			-1.51%	0.6%
Diabetes and obesity			0.53%	-0.35%
Alcohol abuse			-0.06%	0.08%
Drug dependency			-4.13%	-3.85%
Dementia and Alzheimer's disease			-1.12%	-0.78%
Hypertensive disease			2.03%	0.92%
Self-harm			-0.38%	-0.12%
Homicide			0.52%	0.99%

Nevertheless, despite the clear dominance of circulatory diseases diseases as a leading cause of death and as the main contributor to mortality improvements over the 1959-2016 period, the decline of mortality has been far from linear with different causes taking a primary role during different periods and different generations being characterized by different risk factors and behaviors.

6.2 PERIOD TRENDS

Based on our analysis in previous sections, we can identify five broad periods of mortality change which are summarized in Tables 6.2 and 6.3.

6.2.1 1959-1970

The first period comprises the years between 1959 and 1970 in which life expectancy at age 20 for women showed a small increase from 55.75 to 56.68 years and for men decreased from 49.82 to 49.58 years. This period saw a general leveling off in mortality rates after the unprecedented decline in mortality experienced by Americans from 1940 to the mid 1955s (Crimmins, 1981). Some key features of this period are:

- The 1960s were the first decade in which a decline in mortality rates from cardiovascular diseases as a whole started to become evident after mortality rates from this cause had reached a peak in the 1950s (Centers for Disease Control and Prevention, 1999a).
- However, despite the overall fall in cardiovascular mortality rates, the rate of mortality improvements in the 1960s were sluggish, mainly due to the increase in mortality rates from ischaemic heart disease (the dominant group of cardiovascular deaths) which only achieved peak mortality rates in the mid 1960s (Centers for Disease Control and Prevention, 1999a). In fact, ischaemic heart disease accounted for 0.35 years and 0.26 years of life expectancy deterioration between 1959 and 1970 for females and males, respectively.
- Lung cancer was another major contributor to mortality deterioration during this period accounting for a fall 0.11 years and 0.22 years of life expectancy deterioration for females and males, respectively. This is in line with the noticeable negative period effects we identified for this period in our APC analysis in Section 5.5 and which coincide with those reported by Yang (2008).
- External causes of death also played a central role, contributing 0.15 and 0.35 years to the deterioration of life expectancy for females and males, respectively, during this period. Among the external causes the negative contribution of self-harm and interpersonal violence is noteworthy.

Table 6.2: Summary of contribution to life expectancy change at age 20 for different subperiods

	1959-1970		1970-1980		1980-1995		1995-2010		2010-2016	
Cause	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**
All-Cause	**0.93**	**-0.24**	**2.18**	**2.19**	**1.15**	**2.06**	**1.87**	**3.42**	**0.18**	**0.01**
Circulatory diseases	**0.99**	**0.45**	**1.8**	**1.74**	**1.63**	**1.94**	**1.65**	**1.92**	**0.22**	**0.18**
Ischaemic heart disease	-0.35	-0.26	1.07	1.26	1.2	1.6	1.18	1.46	0.24	0.25
CVD and stroke	0.31	0.15	0.6	0.4	0.37	0.24	0.33	0.27	0.03	0.02
Other circulatory system diseases	1.03	0.56	0.13	0.08	0.06	0.1	0.14	0.19	-0.05	-0.09
Neoplasms	**0.07**	**-0.21**	**-0.04**	**-0.11**	**-0.04**	**0.19**	**0.63**	**0.83**	**0.24**	**0.34**
Bowel cancer	0.04	0	0.05	0.01	0.08	0.05	0.09	0.09	0.02	0.03
Liver cancer	0.05	0.03	-0.01	-0.01	0	-0.02	-0.01	-0.04	-0.01	-0.01
Lung cancer	-0.11	-0.22	-0.19	-0.11	-0.24	0.1	0.09	0.36	0.12	0.19
Breast cancer	-0.03	0	0.02	0	0.04	0	0.18	0.01	0.04	0
Prostate cancer	0	0	0	-0.01	0	-0.01	0.01	0.13	0	0.03
Other cancers	0.08	-0.06	0.07	-0.04	0.05	0.02	0.25	0.23	0.06	0.09
Other digestive organ cancers	0.04	0.05	0.02	0.05	0.03	0.06	0.03	0.05	0	0.01
Respiratory diseases	**-0.04**	**-0.15**	**0.06**	**0.16**	**-0.27**	**0.03**	**0.02**	**0.18**	**0**	**0.04**
Influenza and pneumonia	0.01	-0.02	0.19	0.21	-0.01	0.02	0.07	0.09	0.01	0.02
Chronic lower respiratory disease	-0.02	-0.07	-0.12	-0.1	-0.23	0.01	-0.03	0.1	0.01	0.03
Other respiratory diseases	-0.03	-0.05	-0.01	0.05	-0.03	0	-0.02	-0.01	-0.02	-0.01
Digestive system	**-0.04**	**-0.07**	**0.1**	**0.13**	**0.12**	**0.17**	**0.03**	**0.06**	**-0.03**	**-0.02**
Gastric and duodenal ulcer	0.01	0.04	0.02	0.05	0.01	0.02	0.01	0.02	0	0
Chronic liver disease	-0.07	-0.11	0.06	0.07	0.07	0.1	0	0.03	-0.03	-0.03
Other digestive system diseases	0.02	-0.01	0.02	0.01	0.04	0.06	0.01	0.01	0	0.01
External causes	**-0.15**	**-0.35**	**0.17**	**0.21**	**0.14**	**0.45**	**-0.11**	**0.04**	**-0.17**	**-0.44**
Traffic accidents	-0.06	-0.09	0.13	0.31	0.03	0.15	0.05	0.08	0	-0.01
Self-harm and interpersonal violence	-0.08	-0.16	0.03	-0.06	0.06	0.1	0.02	0.09	-0.03	-0.08
Other external causes	-0.02	-0.1	0.02	-0.03	0.06	0.2	-0.18	-0.13	-0.14	-0.35
Other	**0.11**	**0.09**	**0.09**	**0.06**	**-0.44**	**-0.73**	**-0.35**	**0.38**	**-0.08**	**-0.09**
AIDS and tuberculosis	0.05	0.08	0	0.02	-0.14	-0.59	0.13	0.58	0.01	0.03
Diabetes and obesity	0	-0.03	0.1	0.04	-0.06	-0.07	0.07	0.01	-0.01	-0.04
Alcohol abuse and drug dependence	-0.01	-0.03	-0.01	0	0	0.01	0	0.02	-0.02	-0.03
Alzheimer's disease	0	0	-0.01	-0.01	-0.12	-0.05	-0.13	-0.04	-0.08	-0.02
Dementia and other mental disorders	0	0	-0.04	-0.02	-0.03	-0.01	-0.27	-0.13	0.05	0.03
Rest of causes	0.07	0.06	0.05	0.04	-0.09	-0.01	-0.16	-0.06	-0.04	-0.06

Table 6.3: Improvement rate above average for different subperiods

	1959-1970		1970-1980		1980-1995		1995-2010		2010-2016	
Cause	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**
All-Cause	**-0.19%**	**-0.93%**	**1.17%**	**0.96%**	**-0.35%**	**-0.09%**	**0.02%**	**0.45%**	**-0.78%**	**-0.78%**
Circulatory diseases	**-0.63%**	**-1.39%**	**1.1%**	**0.92%**	**-0.3%**	**0.18%**	**0.56%**	**0.82%**	**-1.33%**	**-1.47%**
Ischaemic heart disease	-2%	-2.01%	0.44%	0.58%	-0.39%	0.26%	1.39%	1.1%	0.46%	-0.68%
CVD and stroke	-0.37%	-1.33%	2.26%	2.26%	-0.52%	-0.53%	0.18%	0.74%	-2.21%	-1.83%
Other circulatory system diseases	1.93%	0.59%	0.58%	0.11%	-0.56%	-0.1%	-0.3%	0.42%	-2.35%	-2.05%
Neoplasms	**0.5%**	**-0.56%**	**-0.12%**	**-0.48%**	**-0.54%**	**-0.29%**	**0.29%**	**0.82%**	**-0.09%**	**0.51%**
Bowel cancer	-0.15%	-1.1%	-0.7%	-1.39%	-0.22%	-0.33%	0.6%	1.51%	0.49%	1.38%
Liver cancer	2%	1.27%	1.5%	0.73%	-0.62%	-1%	-0.96%	-0.51%	-2.22%	0.25%
Lung cancer	-1.54%	-1.46%	-1.83%	-0.82%	-0.23%	0.06%	1.58%	0.95%	2.49%	1.52%
Breast cancer	-0.46%	-	-0.34%	-	-0.28%	-	0.85%	-	-0.02%	-
Prostate cancer	-	-0.19%	-	-1.2%	-	-1.55%	-	2.19%	-	0.75%
Other cancers	1.07%	-0.17%	0.56%	0.17%	-0.68%	-0.39%	-0.11%	0.42%	-0.92%	-0.03%
Other digestive organ cancers	0.7%	0.52%	0.26%	0.28%	0.02%	0.03%	-0.34%	-0.23%	-0.91%	-0.9%
Respiratory diseases	**0.07%**	**-0.68%**	**1.04%**	**1.59%**	**-1.74%**	**-0.62%**	**0.86%**	**0.24%**	**0.33%**	**-0.45%**
Influenza and pneumonia	0.69%	0.13%	2.46%	2.11%	-2.5%	-1.92%	0.67%	0.78%	-0.78%	-0.9%
Chronic lower respiratory disease	0.96%	-1.88%	4.56%	6.83%	-2.81%	-1.64%	-0.69%	-1.12%	-0.61%	-1.05%
Other respiratory diseases	1.79%	2.53%	-5.02%	-5.33%	0.6%	1.08%	1.41%	0.81%	0.06%	-0.48%
Digestive system	**-0.74%**	**-1.33%**	**2.66%**	**2.01%**	**0.5%**	**0.81%**	**-0.68%**	**-0.34%**	**-2.62%**	**-2.07%**
Gastric and duodenal ulcer	0.14%	1.04%	1.75%	3.61%	-1.06%	-1.43%	1.21%	0.46%	-3.57%	-5.48%
Chronic liver disease	-1.85%	-2.86%	3.15%	2.19%	1.91%	1.88%	-0.75%	0.08%	-4.76%	-3.3%
Other digestive system diseases	0.39%	-0.72%	2.59%	1.83%	-0.53%	0.15%	-1%	-0.68%	-1.2%	-0.41%
External causes	**-2.31%**	**-2.06%**	**2.97%**	**2.76%**	**1.68%**	**1.52%**	**-1.01%**	**-0.59%**	**-2.36%**	**-3.15%**
Traffic accidents	-3.96%	-3.05%	2.9%	3.09%	-0.83%	1.15%	1.38%	-0.64%	1.06%	-0.83%
Self-harm and interpersonal violence	-3.21%	-2.43%	1.05%	0.44%	2.19%	1.11%	0.37%	0.88%	-2.26%	-1.24%
Other external causes	-1.84%	-1.53%	3.4%	3.92%	1.94%	2.18%	-2.01%	-1.99%	-2.09%	-4.18%
Other	**2.82%**	**1.86%**	**3.05%**	**2.35%**	**-1.33%**	**-2.03%**	**-2.23%**	**-0.45%**	**-1.35%**	**-1.12%**
AIDS and tuberculosis	1.12%	-1.14%	3.39%	6.08%	-10.65%	-15.13%	5.16%	10.16%	6.01%	4.39%
Diabetes and obesity	0.63%	-0.3%	2.9%	3.55%	-2.53%	-1.98%	0.22%	0.08%	-0.23%	-0.62%
Alcohol abuse and drug dependence	-2.29%	-2.75%	1.67%	1.9%	2.23%	1.59%	0.43%	1.3%	-5.24%	-5.35%
Alzheimer's disease	-	-	-18.53%	-17.52%	-2.57%	-1.73%	2.94%	2.53%	2.17%	0.93%
Dementia and other mental disorders	7.84%	4.76%	1.24%	-0.51%	0.53%	1.72%	-8.22%	-6.1%	2.78%	3.06%
Rest of causes	2.21%	1.56%	1.42%	1.12%	-0.59%	-0.09%	-1.59%	-1.3%	-0.96%	-1.28%
Risk factors										
AIDS and tuberculosis	1.12%	-1.14%	3.39%	6.08%	-10.65%	-15.13%	5.16%	10.16%	6.01%	4.39%
Alcohol abuse	-1.96%	-2.78%	2.87%	2.03%	1.91%	1.8%	-0.57%	0.2%	-4.56%	-3.29%
Dementia and Alzheimer's disease	6.75%	5.89%	1.75%	-0.41%	-1.99%	-0.88%	-4.47%	-3.53%	0.86%	0.89%
Diabetes and obesity	0.63%	-0.3%	2.9%	3.55%	-2.53%	-1.98%	0.22%	0.08%	-0.23%	-0.62%
Drug dependency	-3.02%	-3.35%	4.61%	7.19%	3.95%	1.73%	-4.07%	-2.04%	-1.84%	-5.07%
Homicide	-4.4%	-7.67%	0.38%	1.23%	1.12%	2%	2.19%	3.16%	-0.84%	-0.89%
Hypertensive disease	4.93%	3.38%	4.43%	4.39%	-1.43%	-0.89%	-3.52%	-3.23%	-4.05%	-3.22%
Self-harm	-2.59%	-0.01%	1.36%	-0.67%	2.84%	0.6%	-0.91%	0.13%	-2.34%	-0.69%
Smoking	-1.54%	-1.46%	-1.83%	-0.82%	-0.23%	0.06%	1.58%	0.95%	2.49%	1.52%

6.2.2 1970-1980

After the slow mortality improvements of the 1960s, mortality trends in the U.S. experienced a turning point around 1968 with mortality decline accelerating thereafter (Crimmins, 1981; Ouellette et al., 2014) with the 1970s being the decade of the fastest all-cause mortality improvements over the study period. This very fast decline in mortality rates translated in life expectancy at age 20 increasing from 55.68 to 58.86 years for women and from 49.58 to 51.77 years for men over the decade 1970 to 1980. The key features of this period are:

- The turning point in all-cause mortality trends coincides with the turning point in mortality trends from circulatory causes of death seen in 1971 which marked the beginning of rapid declines in mortality from cardiovascular diseases (Ouellette et al., 2014). This revolution in mortality improvements from circulatory diseases in the 1970s was the result of reductions in lifestyle risk factors such as cholesterol levels and smoking, as well as major advances in medical treatments such as prehospital resuscitation, coronary artery bypass surgery and the treatment of hypertension (Goldman and Cook, 1984).

- Importantly, the 1970s was the decade in which cigarette consumption in the U.S. started to decrease steadily. After increasing rapidly for much of the first part to the 20th century, cigarette consumption reached a plateau in the 1960s and early 1970s following the influential first report of the Surgeon General's Committee on Smoking and Health in 1964 establishing a causal link between smoking and lung cancer, resulting in the introduction of anti-smoking ads in 1969, and a ban on cigarette commercials on TV and Radio in 1971. From 1973 onwards cigarette consumption decreased steadily as a result of the so-called "non-smoker movement" which led to legislations banning smoking in several public places (Garfinkel, 1997; Centers for Disease Control and Prevention, 1999c).

- However, despite the general decline in smoking prevalence in the 1970s, lung cancer continued its important negative contribution to mortality change. This is because period trends in lung cancer are mainly determined by the changes in cigarette consumption with a 20-30 year lag (Crimmins, 1981).

- After circulatory diseases, traffic accidents and influenza and pneumonia were the two other major contributors to the decline in mortality in the 1970s. On the one hand, death rates from traffic accidents decreased significantly during this period after having peaked in the late 1960s. This decline in traffic accident fatalities followed the passage in 1966 of the Highway Safety Act and the National Traffic and Motor Vehicle Safety Act which authorized the federal government to set and regulate standards for motor vehicles and highways (Centers for Disease Control and Prevention, 1999b). On the other hand, after having leveled off between 1950 and 1960, mortality rates from influenza and pneumonia fell sharply in the 1970s until reaching a turning point in 1980 when they started to rise again (Armstrong et al., 1999).

6.2.3 1980-1995

Over this period mortality improvements continued although at a much slower pace than in the 1970s. Between 1980 and 1995 life expectancy at age 20 for women increased from 58.86 years to 60.01 years, while for men it increased from 51.77 years to 53.83 years. The key drivers of the slower improvements of this period are:

- After having decreased steadily during the 20th century, mortality rates from infectious diseases started to rise unexpectedly in the 1980s driven by an increase in mortality rates from influenza and pneumonia and, more importantly, by the emergence of the AIDS/HIV epidemic in the early 1980s (Armstrong et al., 1999). AIDS/HIV would become one of the main drivers of the slowdown in mortality improvements, with AIDS and tuberculosis accounting for 0.14 years and 0.59 years of deterioration in life expectancy between 1980 and 1995 for females and males, respectively.
- For women, the other major contributors to slower mortality improvement were smoking-related lung disease with lung cancer and chronic lower respiratory diseases (CLRD) accounting, respectively, for 0.24 and 0.23 years of life expectancy deterioration in the 1980-1995 period. Noticeably, this negative effect is seen for women but not for men, reflecting the historic differences in tobacco consumption patterns among men and women and the 20-30 year latency period for lung cancer and CLRD to manifest themselves (Kazerouni et al., 2004). This is consistent with the fact that smoking prevalence for men peaked in the 1950s while for women it peaked a decade later (Garfinkel, 1997; Centers for Disease Control and Prevention, 1999c).
- Cardiovascular diseases continued to show important mortality improvements albeit at a slower pace than in the 1970s, partially due to the rising trends in the prevalence of obesity and diabetes (Ford and Capewell, 2007). In particular, the prevalence of obesity among Americans aged 20-74 increased dramatically since the late 1970s doubling from 15% in 1976-1980 to 30.9% in 1999-2000 (Fryar et al., 2012). Similarly, the prevalence of diagnosed diabetes among Americans aged 20-79 started to increase in the late 1980s rising from 3.5% in 1990 to 8.3% in 2012 (Geiss et al., 2014). The key drivers of the so-called "diabesity epidemic" are multi-factorial and include secular changes in agricultural policies, diet, food environment, physical inactivity, and sleep deprivation (Bhupathiraju and Hu, 2016).

6.2.4 1995-2010

After the slight slump in the pace of mortality improvements over the preceding 15 years, improvements accelerated from 1995 through to 2010, especially for men. As a result, period life expectancy rose in this period by a remarkable 3.42 years for men (from 53.83 years to 57.25 years) and by 1.87 years for women (from 60.01 years to 61.88 years). Some of the key features of mortality change in this period include:

- A continued improvement in cardiovascular disease mortality at a pace faster than the preceding period. However, between 1995 and 2010, a clear age-divide in the pace of mortality improvements for cardiovascular diseases emerged with the mortality rates among young adults – and especially women – showing signs of stagnation in sharp contrast with older Americans who experienced steep mortality declines which accelerated from the early 2000s (Ford and Capewell, 2007; Wilmot et al., 2015). This stagnation in cardiovascular disease mortality among younger adults coincides with a substantial deterioration in the 1980s and 1990s in the prevalence of major traditional risk factors such diabetes, obesity and hypertension (Flegal et al., 1998; Fox et al., 2007).

- Noticeably, in this period mortality rates from neoplasms as a whole also started to experience sustained mortality improvements after having peaked in the early 1990s (Edwards et al., 2010; Ouellette et al., 2014). Thus, neoplasms contributed 0.63 and 0.83 years to the increase in life expectancy at age 20 between 1995 and 2010 for females and males, respectively, with much of this increase stemming from improvements in mortality from major cancers – lung, breast and prostate cancer. While cohort effects, especially related to smoking patterns, are behind much of the decrease in cancer mortality during this period, there are still some noteworthy period patterns. For lung cancer both women and men show matching positive period effects starting from 1990 which, as discussed before, reflect with a 20-30 year lag the onset in 1970s of a steady decrease in tobacco consumption following the 1964 Surgeon General's reports on smoking and health. In the case of prostate cancer, mortality rates peaked in the early 1990s following the widespread introduction of prostate-specific antigen (PSA) screening with its subsequent benefits in terms of earlier detection. This, together with improved treatments, contributed to a continuing decline in prostate cancer mortality rates from the early 1990s to the early 2010s, which has nonetheless leveled-off in recent years due a decrease in the uptake of PSA (Negoita et al., 2018). Similarly, after a period of stable mortality rates in the 1980s, breast cancer mortality started to decrease steadily in the early 1990s. While this decline coincides with the introduction of mammography screening in the late 1980s and early 1990s, the impact of mammographies on the reduction of mortality remains contentious: it is argued that the decline is more likely attributable to an increase in the detection of smaller palpable tumors and to the increase in the use of adjuvant chemotherapy (Jatoi and Miller, 2003; Narod et al., 2015).

- After the dramatic negative impact of the HIV epidemic in the 1980s, this period showed a sharp decline in the incidence and mortality from AIDS coinciding with the introduction of anti-retroviral therapy in 1996 (Murphy et al., 2001).

- In contrast to these positive trends, this period saw the emergence of Alzheimer's disease, dementia and other mental disorders as a very important cause of mortality, especially among women. In fact, between 1995 and 2010, Alzheimer's and dementia

combined, contributed to a fall in life expectancy at age 20 by 0.40 years and 0.27 years for women and men, respectively. One explanation for the mortality increase is the improved clinical awareness and diagnosis of Alzheimer's and dementia (Weuve et al., 2014). However, with population aging and mortality from major causes in decline, more people are surviving to ages where the risk of Alzheimer's and dementia is highest, explaining in part the observed increase (Kramarow and Tejada-Vera, 2019).

- Mortality rates from other external causes of death also experienced a significant increase during this period, with a significant portion of this mortality deterioration coming from an increase in mortality rates from unintentional drug poisoning linked to prescribed opioid use (Alexander et al., 2018; Masters et al., 2018). This increase foreshadowed the narrative of "death of despairs" which would come to dominate in the discussion of U.S. mortality change in the 2010s.

6.2.5 2010-2016

Starting from around 2010, overall mortality improvements in the U.S. show a considerable slowdown leading to a stagnation in life expectancy. For women, between 2010 and 2016 life expectancy at age 20 increase by only 0.18 years rising from 61.88 years in 2010 to 62.06 years in 2016. For men, the slowdown in mortality improvements resulted in life expectancy increasing minimally from 57.25 years in 2010 to 57.26 years in 2016. Some of the key drivers behind this slowdown in improvements are discussed below:

- The main narrative behind the slowdown of mortality improvements after 2010 has been that of "deaths of despair" popularized by the widely cited paper of Case and Deaton (2015). They argue that the slowdown of mortality improvement is mainly due to an increase in accidental drug and alcohol poisoning, chronic liver disease, cirrhosis and suicide, especially among middle-aged white Americans. In a follow-up paper, Case and Deaton (2017) argue that these deaths of despair are a cohort effect resulting form the cumulative decline in living standards across generations. However, this "death of despair" narrative has been challenged. Masters et al. (2018) suggest that much of the change is due to period rather than cohort effects linked to an increase in drug-use related mortality associated with opioids. Harper et al. (2020) argue that while opioids are one of the key drivers of the slowdown of improvements, there are other causes of death that have contributed, including increases in mortality from Alzheimer's disease, homicide and suicide, together with a slowdown in mortality improvements from cardiovascular diseases.

- Our results from the previous sections support the complementary findings of Case and Deaton (2015) and Harper et al. (2020) in relation to the diversity of causes driving the stagnation of mortality improvements. Moreover, in agreement with Masters et al. (2018), the results of our APC decomposition suggest that period effects play a central

role. In particular, in the last two columns of Table 6.3, we report large negative period effects for circulatory diseases, chronic liver disease, self-harm and interpersonal violence, and notably, from deaths linked to alcohol abuse and drug dependency.

- Circulatory diseases deserve a special discussion as they remain the leading cause of death in the 21st century. In Table 6.3, we see that during this period mortality from circulatory diseases for women and men, respectively, experienced mortality improvements that were 1.33% p.a and 1.47% p.a slower than the average 2.21% p.a and 2.01% p.a reported in Table 6.1 over the whole study period. Abstracting from cohort improvements, this resulted in net improvements of just 0.88% p.a. and 0.54% p.a. between 2010 and 2016 for women and men, respectively. Mehta et al. (2020) argue that this slowdown in the pace of mortality improvements for circulatory diseases could even be the main driver for the stagnation in life expectancy as opposed to the counter narrative of deaths of despair. Some of the preliminary potential explanations for the stagnation of improvements include the increase in the prevalence of diabetes and obesity, the plateauing of the benefits of the decrease in smoking prevalence, and the fact that advancements in the treatment and prevention of cardiovascular diseases have become more incremental in recent years (Sidney et al., 2016; Mensah et al., 2017; Mehta et al., 2020).

6.3 COHORT EFFECTS

Based on our analysis in previous sections, we can identify five broad groups of birth cohorts, whose mortality improvements above or below the average improvement in the study period are summarized in Table 6.4. In this table we note that the cohort boundaries for women differ to those for men, reflecting the delayed onset of common risk factors among women.

6.3.1 FEMALE 1881–1925, MALE 1881-1920

This cohort group shows average mortality improvements for women and slightly below average improvements for men corresponding, respectively, to all-cause cohort effects of 0.01% p.a. and -0.29% p.a. These below average improvements are mainly explained by patterns of cigarette consumption among this generation, as reflected by the large negative cohort effects that both genders show for lung cancer and chronic lower respiratory diseases. In particular, Preston and Wang (2006) report that cigarette consumption (in terms of average numbers of years spent as cigarette smoker before age 40) increased steadily from the 1885-1889 generation to reach a plateau for the 1910-1925 generations of men and the 1925-1940 generation of women.

6.3.2 FEMALE 1925-1945, MALE 1920-1940

This cohort group shows above average mortality improvement with women having a positive cohort effect of 0.48% p.a. and men a positive cohort effect of 0.84% p.a., stemming from

Table 6.4: Improvement rate above average for different cohorts. Note that cohort groups for women and men have different boundaries

Female cohort:	**1881-1925**		**1925-1945**		**1945-1960**		**1960-1975**		**1975-1986**	
Male cohort:	**1881-1920**		**1920-1940**		**1940-1955**		**1955-1970**		**1970-1986**	
Cause	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**
All-Cause	**0.01%**	**-0.29%**	**0.48%**	**0.84%**	**-0.44%**	**-0.69%**	**-0.07%**	**-0.05%**	**-0.44%**	**-0.59%**
Circulatory diseases	**0.33%**	**0.06%**	**0.74%**	**1.24%**	**-1.23%**	**-0.3%**	**-1.21%**	**-1.03%**	**-0.68%**	**-2.65%**
Ischaemic heart disease	0.51%	-0.36%	0.85%	1.25%	-1.38%	0.02%	-1.83%	-0.14%	-1.39%	-3.19%
CVD and stroke	0.03%	0.38%	0.15%	0.47%	-0.81%	-1.04%	0.4%	-0.43%	0.09%	-0.51%
Other circulatory system diseases	0.03%	0.41%	0.68%	1.11%	-0.88%	-1.02%	-0.59%	-1.3%	0.68%	-0.82%
Neoplasms	**-1.11%**	**-1.26%**	**0.52%**	**0.35%**	**1.04%**	**0.49%**	**0.94%**	**1.19%**	**0.42%**	**0.19%**
Bowel cancer	0.1%	0.33%	0.98%	0.84%	-0.93%	-0.49%	-1.61%	-1.99%	-0.91%	-0.47%
Liver cancer	-0.17%	-0.14%	-0.08%	0.43%	-0.72%	-3.89%	1.44%	4.54%	0.25%	0.24%
Lung cancer	-2.42%	-3.26%	1.2%	0.66%	1.8%	1.96%	6.02%	3.95%	-0.44%	-0.94%
Breast cancer	-0.97%	-	0.31%	-	1.06%	-	0.25%	-	0.93%	-
Prostate cancer	-	-0.67%	-	0.92%	-	-1.59%	-	-0.43%	-	3.05%
Other cancers	-1.58%	-1.82%	0.65%	0.19%	1.15%	0.9%	0.7%	1.64%	0.64%	0.67%
Other digestive organ cancers	-0.24%	-0.02%	0.59%	0.15%	0.02%	0.22%	0.07%	0.69%	-0.57%	-1.5%
Respiratory diseases	**-0.74%**	**-1.72%**	**-0.13%**	**0.76%**	**0.73%**	**0.24%**	**2.11%**	**1.28%**	**1.83%**	**0.04%**
Influenza and pneumonia	-0.26%	-0.98%	-0.49%	0.75%	0.26%	-0.74%	0.7%	1.36%	-0.15%	-1.16%
Chronic lower respiratory disease	-2.91%	-2.74%	1.16%	1.46%	1.01%	0.86%	3.6%	0.65%	3.7%	-0.18%
Other respiratory diseases	-1.9%	-2.82%	0.4%	1.27%	0.72%	0.11%	1.64%	1.29%	2.15%	1.17%
Digestive system	**-0.83%**	**-0.52%**	**0.64%**	**0.56%**	**-0.32%**	**-1.2%**	**1.9%**	**2.38%**	**0.05%**	**-0.6%**
Gastric and duodenal ulcer	-1.16%	-1.22%	0.44%	1.16%	-0.14%	-0.91%	0.83%	1.04%	1.16%	-0.27%
Chronic liver disease	-1.77%	-0.84%	1.85%	0.55%	-0.63%	-1.12%	3.56%	3.28%	-0.7%	-0.85%
Other digestive system diseases	-0.59%	-0.51%	-0.1%	0.43%	0.09%	-1.16%	1.27%	1.9%	0.78%	-0.13%
External causes	**0.87%**	**0.19%**	**0.16%**	**0.44%**	**-2.1%**	**-1.57%**	**0.43%**	**0.16%**	**0.58%**	**0.35%**
Traffic accidents	-0.2%	0.1%	0.33%	-0.14%	-0.88%	-1.04%	0.51%	0.38%	-0.17%	1.16%
Self-harm and interpersonal violence	-0.12%	0.35%	0.42%	0.57%	-1.4%	-1.11%	0.76%	-0.67%	0.3%	-0.45%
Other external causes	1.93%	0.44%	0.27%	0.89%	-3.38%	-2.42%	-0.41%	0.34%	-0.62%	-0.3%
Other	**-2.18%**	**-1.56%**	**0.97%**	**0.28%**	**0.03%**	**-1.61%**	**2.44%**	**3.42%**	**2.17%**	**2.35%**
AIDS and tuberculosis	3.88%	6.94%	-1.21%	-2.31%	-7.21%	-7.88%	0.85%	-0.12%	1.72%	0.16%
Diabetes and obesity	-0.04%	0.03%	0.53%	0.34%	-0.58%	-0.44%	-1.08%	-0.15%	-0.07%	-0.25%
Alcohol abuse and drug dependence	-3.18%	-2.16%	2.12%	0.61%	-0.77%	-1.55%	4.27%	4.02%	-0.99%	0.94%
Alzheimer's disease	-5.82%	-7.29%	0.99%	0.4%	1.68%	1.92%	5.56%	1.67%	-2.53%	3.14%
Dementia and other mental disorders	-4.97%	-5.4%	-0.06%	-0.27%	3.66%	1.92%	4.52%	4.38%	4.66%	5.41%
Rest of causes	-1.48%	-1.19%	0.12%	0.36%	0.72%	-1.03%	1.57%	2.23%	1.14%	1%
Risk factors										
AIDS and tuberculosis	3.88%	6.94%	-1.21%	-2.31%	-7.21%	-7.88%	0.85%	-0.12%	1.72%	0.16%
Alcohol abuse	-1.53%	-0.68%	1.88%	0.65%	-0.67%	-1.06%	3.29%	2.75%	-1.83%	-1.38%
Dementia and Alzheimer's disease	-3.61%	-3.79%	0%	-0.11%	2.94%	1.44%	4.16%	3.55%	4.17%	4.92%
Diabetes and obesity	-0.04%	0.03%	0.53%	0.34%	-0.58%	-0.44%	-1.08%	-0.15%	-0.07%	-0.25%
Drug dependency	1.13%	1.73%	-0.01%	0.14%	-4.78%	-6.04%	1.64%	3.2%	-2.14%	-2.17%
Homicide	-0.71%	0.38%	0.91%	0.7%	-1.25%	0.3%	0.68%	-2.09%	0.54%	-2.49%
Hypertensive disease	0.52%	1.4%	0.75%	0.77%	-1.96%	-1.89%	-0.62%	-1.4%	0.23%	-1.1%
Self-harm	0.11%	0.29%	0.28%	0.86%	-1.09%	-1.89%	0.64%	-0.32%	-0.77%	-0.1%
Smoking	-2.42%	-3.26%	1.2%	0.66%	1.8%	1.96%	6.02%	3.95%	-0.44%	-0.94%

positive cohort effects for the majority of causes of death and most risk factors. Some key characteristics of this cohort include:

- This is the first generational group which does not show an increase in smoking prevalence relative to the preceding generations. Moreover, from this generation onwards cigarette consumption decreased steadily (Harris, 1983; Preston and Wang, 2006).
- This generation shows a clear positive cohort effect in alcohol-related mortality. In the particular case of men, this is consistent with the strong declining trend in alcohol volume consumption and frequency of heavy drinking reported by Kerr et al. (2009) for those born between 1920 and 1940.
- In contrast the positive cohort effects from other risk factors, this generation, who were in their 40s to 60s in the 1980s, shows negative cohort effects from AIDS and tuberculosis, albeit milder than those seen in the following generation.

6.3.3 FEMALE 1945-1960, MALE 1940-1955

The generation of early baby boomers is noticeably one of the generations with the slowest mortality improvements among the generations included in the study. Women from this generation experience mortality improvements which are 0.44% p.a. below the average while men experience mortality improvement 0.69% p.a. below the average. The reasons for the below average improvement are multi-factorial and reflect adverse patterns in a diverse number of causes and risk factors:

- As with the preceding generation, early baby boomers continued to experience positive cohort effects from smoking-related causes of death as a consequence of the continued decline in tobacco consumption among younger generations. However, the positive gains from smoking were counterbalanced by negative trends in other factors and causes.
- In particular, this generation marked the start of a turning point in generational patterns of cardiovascular mortality with cohorts born from around 1945-1950 onwards showing negative cohort effects for this group of causes. This pattern coincides with similar turning points in the mortality rates from markers of cardiovascular risk factors including hypertension and obesity and diabetes. In the case of obesity, while period effects tend to dominate when explaining trends in obesity prevalence, some cohort effects have also been reported with obesity prevalence rising steadily for younger generations starting from the 1955 birth cohort (Reither et al., 2009; An and Xiang, 2016).
- Early baby boomers also show distinctive negative patterns in mortality rates associated with alcohol abuse and drug dependency. For drug dependency, the negative

cohort effects coincide with the strong cohort patterns found by Kerr et al. (2018) who report an important increase in marijuana consumption between the 1945 and 1955 generations. Moreover, heroin and pain-reliever misuse are reported to show cohort patterns (Verdery et al., 2020), which match an increasing trend in prescription opioid and heroin overdose mortality among individuals born between 1947 and 1964 (Huang et al., 2018).

- This generation of men and women who were in their early and middle adulthood in the 1980s during the peak of the HIV epidemic, exhibits very strong negative cohort effect from AIDS even after controlling for period effects (Acosta et al., 2020).
- Finally, this cohort shows significant negative cohort effects for suicide which are consistent with the shift in cohort patterns in suicide rates which began to increase sharply for both genders starting from the baby boom generation (Phillips, 2014).

6.3.4 FEMALE 1960-1975, MALE 1955-1970

This generation shows mortality improvements which are close to the average with women and men experiencing minimal negative cohort effects of -0.07% p.a and -0.05% p.a., respectively. These average improvements reflect offsetting patterns for different causes and risk factors. Similar to the preceding generation, this generation continued the negative cohort effects from cardiovascular diseases, obesity and diabetes, and hypertension, consistent with the previously discussed increasing trend in obesity prevalence which started with the 1955 birth cohort. Likewise, this generation continues to benefit from the positive cohort effects stemming from reductions in smoking prevalence. However, in sharp contrast with the previous and subsequent generations, this cohort group shows positive cohort effects for alcohol abuse and drug dependency.

6.3.5 FEMALE 1975-1986, MALE 1970-1986

Together with the baby boomer generation, this youngest generation shows the worst mortality improvements among the cohorts in the study, with women showing worryingly negative cohort effects of -0.44% p.a. and men a negative effects of -0.59% p.a. These negative effects reflect adverse patterns in several risk factors for this cohort:

- This cohort group shows important negative cohort effects for alcohol-related mortality. This is in line with several studies which report a sharp increase in alcohol volume consumption and frequency of heavy drinking starting from the 1970 birth cohort until the 1985 cohort (Kerr et al., 2009, 2013). Similarly, Huang et al. (2018) reports an increasing trend in prescription opioid and heroin overdose mortality among those born between the start of the 1970s and the start of the 1980s.

- Noticeably, the positive cohort effects associated with smoking and lung cancer seem to be winding down, with women and men of this generation showing a negative cohort effect in lung cancer mortality of -0.44% p.a. and -0.94% p.a., respectively. This slowing down is partially linked to the leveling-off in the prevalence of current smoking among the cohort born between 1970 and 1980, especially among blacks and Hispanics (Jemal et al., 2018). However, part of the slowdown seems to be associated with changes in cigarette manufacturing as evidenced by the increase in the prevalence of adenocarcinoma as a result of the now more prevalent consumption of filtered cigarettes (Fidler-Benaoudia et al., 2020).

Section 7: Conclusion

In this report we have carried out an exhaustive examination of the mortality patterns in the U.S. between 1959 and 2016, finding that the U.S. has made enormous progress in the past 60 years in terms of improving mortality. However, we have found there is a deceleration in mortality improvement in the most recent period from 2010 to 2016 and which has continued through recent years until 2018 (Harper et al., 2020). In addition to this period of deceleration we have also found a deceleration of mortality improvement for the younger birth cohorts linked to adverse cohort trends in diabetes and obesity, a plateauing of the gains in smoking and negative effects in alcohol consumption and drug abuse. Moreover, the onset of COVID-19 in 2020 has led to an unprecedented decrease in life expectancy with early estimates suggesting a decline of 1.3 years in life expectancy at birth in 2020 (Andrasfay and Goldman, 2021). However, this is not the first time in which the decline in mortality rates appears to have stalled and there is ample potential for mortality rates to continue to decline. Life expectancy in U.S. lags behind other developed countries (Ho and Hendi, 2018) and the longer lives achieved by other countries suggests that U.S. life expectancy has not yet reached a ceiling. Moreover, future improvements could come from a variety of fronts including, among others, public health strategies for controlling obesity and drug abuse; technological progress in reducing the incidence and lethality of cancers; and developments in "precision medicine" and organ regeneration (Vaupel et al., 2021).

This report has concentrated on mortality trends across the whole of the U.S., but the U.S. is undeniably a very diverse population with significant regional, educational, racial and socioeconomic differences. For example, in a recent research project sponsored by the SOA, Barbieri (2020) has highlighted that mortality differentials have increased between socioeconomic groups, with women and men in the top decile of the U.S. population having in 2018 a life expectancy at birth of 84.8 and 80.5 years, respectively, in sharp contrast with the 79.0 and 73.2 years of life expectancy at birth for women and men in the lowest decile. As such it is important to delve into cause-specific mortality trends among U.S. subpopulations to shed further light on the drivers of unequal mortality change.

Section 8: Acknowledgments

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Project Oversight Group members:

- Jennifer A. Haid
- Jean-Marc Fix
- Sam Gutterman
- Larry Pinzur
- Larry Stern (Chair)

At the Society of Actuaries:

- R. Dale Hall
- Jan Schuh
- Ronora Stryker
- Erika Schulty

Appendix A: Standard population for age-standardized death rates

In all of our calculations of age-standardized death rates, we use as standard population the U.S. gender-specific 2010 census population as derived from the Census Bureau's Population Estimates Program (NBER, 2016). Table A.1 below presents this standard population.

Table A.1: Standard population based on U.S. gender-specific 2010 census population

Age	**Female**	**Male**
0	1,929,877	2,014,276
1-4	7,952,058	8,305,151
5-9	9,959,019	10,389,638
10-14	10,097,332	10,579,862
15-19	10,736,677	11,303,666
20-24	10,571,823	11,014,176
25-29	10,466,258	10,635,591
30-34	9,965,599	9,996,500
35-39	10,137,620	10,042,022
40-44	10,496,987	10,393,977
45-49	11,499,506	11,209,085
50-54	11,364,851	10,933,274
55-59	10,141,157	9,523,648
60-64	8,740,424	8,077,500
65-69	6,582,716	5,852,547
70-74	5,034,194	4,243,972
75-79	4,135,407	3,182,388
80-84	3,448,953	2,294,374
85-89	2,346,592	1,273,867
90-94	1,023,979	424,387
95-99	288,981	82,263
100+	44,202	9,162
Total	**156,964,212**	**151,781,326**

Appendix B: Methodology for decomposing life expectancy differences

In this appendix we provide the details of our implementation of the age and cause decomposition of the difference between two life expectancies.

B.1 NOTATION

Following standard actuarial and demographic notation we denote key life table quantities as follows:

- ${}_nm_x$: the central death rate over the interval from x to $x+n$;
- ${}_nq_x$: the probability of an individual aged x dying before age $x+n$;
- l_x: the number of life table individuals alive at age x;
- ${}_nd_x$: the number of individuals dying during interval $[x, x+n)$;
- ${}_nL_x$: the number years lived in the age interval $[x, x+n)$;
- T_x: the number of years lived by the population after age x; and
- $\mathring{e}_x$: the complete life expectancy at age x.

B.2 LIFE TABLE CONSTRUCTION

For the calculation of life expectancies at age 20 and other life table functions we assume that we have death and population data by five year age bands corresponding to the age intervals $[x, x+5)$, $x = 20, 25, \ldots, 90, 95$, and for the open-ended age interval $[100, \infty)$. From these data we calculate ${}_5m_x$, $x = 20, \ldots, 95$, and ${}_\infty m_{100}$.

Under the assumption that deaths are uniformly distributed over each age interval, the relationship between ${}_5m_x$ and ${}_5q_x$ is given by

$$ {}_5q_x = \frac{5 \cdot {}_5m_x}{1 + 2.5 \cdot {}_5m_x}. $$

We assume that $l_{20} = 100,000$ and then compute the other life table quantities using the following standard demographic relationships (see, e.g, Chiang (1972)):

$$ l_{x+5} = l_x \cdot {}_5q_x \quad \text{and} \quad {}_5d_x = l_x - l_{x+5}, \quad \text{for } x = 20, \ldots, 95; $$

$$ {}_5L_x = 5(l_x - {}_5d_x) + 2.5 \cdot {}_5d_x, \quad \text{for } x = 20, \ldots, 95; $$

$$ T_x = {}_5L_x + {}_5L_{x+5} \cdots + {}_\infty L_{100}, \quad \text{for } x = 20, \ldots, 95; \text{ and} $$

$$ \mathring{e}_x = \frac{T_x}{l_x}, \quad \text{for } x = 20, \ldots, 95. $$

The quantities in the open-ended age interval are computed from the equations:

$$\mathring{e}_{100} = \frac{1}{{}_{\infty}m_{100}}, \quad \text{and} \quad T_{100} = {}_{\infty}L_{100} = l_{100} \cdot \mathring{e}_{100}.$$

B.3 AGE DECOMPOSITION OF THE DIFFERENCE BETWEEN TWO LIFE EXPECTANCIES

Consider two life expectancies at age 20, $\mathring{e}^1_{20}$ and $\mathring{e}^2_{20}$, computed based on central death rates ${}_5m^1_{20}, \ldots, {}_{\infty}m^1_{100}$ and ${}_5m^2_{20}, \ldots, {}_{\infty}m^2_{100}$, respectively. In our case $\mathring{e}^1_{20}$ and $\mathring{e}^2_{20}$ represent the life expectancy of the same population in years t and $t+1$, but they could represent other things such as the life expectancy for men and women or for two different socioeconomic groups.

The aim is to decompose $\mathring{e}^2_{20} - \mathring{e}^1_{20}$ into the contribution of different ages so that

$$\mathring{e}^2_{20} - \mathring{e}^1_{20} \; = \; {}_5\delta_{20} \; + \; {}_5\delta_{25} \; + \ldots + \; {}_{\infty}\delta_{100},$$

where ${}_5\delta_x$, $x = 20, \ldots, 95$, and ${}_{\infty}\delta_{100}$ denote the contribution of each of the age intervals.

Following the "symmetrical" approach in Andreev et al. (2002), this decomposition can be obtained with the following formula

$${}_5\delta_x = \frac{1}{2} \cdot \frac{l^2_x(\mathring{e}^2_x - \mathring{e}^1_x) - l^2_{x+5}(\mathring{e}^2_{x+5} - \mathring{e}^1_{x+5})}{l^2_{20}} - \frac{1}{2} \cdot \frac{l^1_x(\mathring{e}^1_x - \mathring{e}^2_x) - l^1_{x+5}(\mathring{e}^1_{x+5} - \mathring{e}^2_{x+5})}{l^1_{20}}$$

for $x = 20, \ldots, 95$ and with the formula

$${}_{\infty}\delta_{100} = \frac{1}{2} \cdot \frac{l^2_{100}(\mathring{e}^2_{100} - \mathring{e}^1_{100})}{l^2_{20}} - \frac{1}{2} \cdot \frac{l^1_{100}(\mathring{e}^1_{100} - \mathring{e}^2_{100})}{l^1_{20}}$$

for the open-ended age interval.

B.4 CAUSE OF DEATH DECOMPOSITION OF THE DIFFERENCE BETWEEN TWO LIFE EXPECTANCIES

Let us now assume that we have k causes of death so that the all-cause central death rates are given by ${}_nm^1_x = \sum_{j=1}^k {}_nm^1_{x,j}$ and ${}_nm^2_x = \sum_{j=1}^k {}_nm^2_{x,j}$, where ${}_nm^1_{x,j}$ and ${}_nm^2_{x,j}$, $j = 1, \ldots, k$, represent the cause-specific components of the central death rates.

Following Shkolnikov et al. (2003), the cause-specific contributions to the difference in life expectancies can be computed as

$${}_5\delta_{x,j} \; = \frac{{}_5m^1_{x,j} - {}_5\, m^2_{x,j}}{{}_5m^1_x - {}_5\, m^2_x} \cdot {}_5\delta_x,$$

for $x = 20, \ldots, 95$ and as

$$ {}_{\infty}\delta_{100,j} = \frac{{}_{\infty}m^1_{100,j} -_{\infty} m^2_{100,j}}{{}_{\infty}m^1_{100} -_{\infty} m^2_{100}} \cdot {}_{\infty}\delta_{100} $$

for the open-ended age interval. These last two expressions clearly satisfy ${}_5\delta_x = \sum_{j=1}^{k} {}_5\delta_{x,j}$, $x = 20, \ldots, 95$, and ${}_{\infty}\delta_{100} = \sum_{j=1}^{k} {}_{\infty}\delta_{100,j}$.

Appendix C: Decomposition of life expectancy change by age

Tables C.1 to C.6 show the decomposition of life expectancy gains across different age groups for the subperiods 1970-1985, 1985-2000 and 2000-2015.

Table C.1: Decomposition of gains and losses in life expectancy at age 20 between 1970 and 1985 by causes of death, for selected age bands. Females

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**0.55**	**0.75**	**1.14**	**0.24**	**2.67**
Circulatory diseases		**0.15**	**0.52**	**1.41**	**0.38**	**2.46**
	Ischaemic heart disease	0.05	0.32	0.93	0.23	1.53
	CVD and stroke	0.05	0.15	0.46	0.15	0.81
	Other circulatory system diseases	0.05	0.05	0.01	0.00	0.12
Neoplasms		**0.08**	**-0.02**	**-0.16**	**-0.02**	**-0.12**
	Bowel cancer	0.01	0.03	0.03	0.00	0.07
	Liver cancer	0.00	0.00	0.00	0.00	0.00
	Lung cancer	0.00	-0.14	-0.15	-0.01	-0.30
	Breast cancer	0.01	0.01	-0.02	0.00	0.00
	Prostate cancer	0.00	0.00	0.00	0.00	-0.01
	Other cancers	0.06	0.07	-0.03	-0.02	0.08
	Other digestive organ cancers	0.01	0.02	0.01	0.00	0.03
Respiratory diseases		**0.05**	**0.01**	**-0.11**	**-0.02**	**-0.07**
	Influenza and pneumonia	0.04	0.05	0.07	0.02	0.18
	Chronic lower respiratory disease	0.01	-0.05	-0.15	-0.02	-0.22
	Other respiratory diseases	0.01	0.00	-0.02	-0.01	-0.03
Digestive system		**0.07**	**0.09**	**0.01**	**-0.01**	**0.15**
	Gastric and duodenal ulcer	0.00	0.01	0.01	0.00	0.02
	Chronic liver disease	0.04	0.06	0.00	0.00	0.10
	Other digestive system diseases	0.02	0.02	0.01	-0.01	0.04
External causes		**0.11**	**0.08**	**0.05**	**0.02**	**0.26**
	Traffic accidents	0.08	0.04	0.02	0.00	0.14
	Self-harm and interpersonal violence	0.03	0.02	0.00	0.00	0.05
	Other external causes	0.00	0.02	0.03	0.02	0.06
Other		**0.09**	**0.07**	**-0.07**	**-0.10**	**-0.01**
	AIDS and tuberculosis	0.00	0.00	0.00	0.00	0.00
	Diabetes and obesity	0.01	0.03	0.07	0.00	0.12
	Alcohol abuse and drug dependence	0.00	0.00	0.00	0.00	-0.01
	Alzheimer's disease	0.00	0.00	-0.03	-0.01	-0.05
	Dementia and other mental disorders	0.00	0.00	-0.02	-0.03	-0.05
	Rest of causes	0.09	0.04	-0.09	-0.06	-0.02

Table C.2: Decomposition of gains and losses in life expectancy at age 20 between 1970 and 1985 by causes of death, for selected age bands. Males

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**0.64**	**1.40**	**0.94**	**0.05**	**3.03**
Circulatory diseases		**0.21**	**1.00**	**1.08**	**0.13**	**2.42**
	Ischaemic heart disease	0.15	0.83	0.79	0.08	1.85
	CVD and stroke	0.04	0.14	0.30	0.05	0.53
	Other circulatory system diseases	0.03	0.03	-0.01	0.00	0.05
Neoplasms		**0.05**	**-0.01**	**-0.14**	**-0.02**	**-0.13**
	Bowel cancer	0.00	0.01	0.01	0.00	0.02
	Liver cancer	0.00	-0.01	-0.01	0.00	-0.02
	Lung cancer	0.02	-0.03	-0.10	-0.01	-0.12
	Breast cancer	0.00	0.00	0.00	0.00	0.00
	Prostate cancer	0.00	0.00	-0.01	0.00	-0.02
	Other cancers	0.02	-0.01	-0.06	-0.01	-0.05
	Other digestive organ cancers	0.01	0.03	0.03	0.00	0.06
Respiratory diseases		**0.05**	**0.10**	**-0.01**	**-0.02**	**0.11**
	Influenza and pneumonia	0.04	0.08	0.08	0.00	0.20
	Chronic lower respiratory disease	0.00	0.00	-0.11	-0.02	-0.13
	Other respiratory diseases	0.00	0.02	0.02	-0.01	0.04
Digestive system		**0.07**	**0.11**	**0.03**	**0.00**	**0.21**
	Gastric and duodenal ulcer	0.01	0.02	0.02	0.00	0.05
	Chronic liver disease	0.04	0.08	0.01	0.00	0.13
	Other digestive system diseases	0.01	0.01	0.00	0.00	0.03
External causes		**0.30**	**0.17**	**0.05**	**0.00**	**0.52**
	Traffic accidents	0.27	0.08	0.03	0.00	0.39
	Self-harm and interpersonal violence	0.00	0.03	0.00	0.00	0.03
	Other external causes	0.03	0.05	0.02	0.00	0.10
Other		**-0.04**	**0.04**	**-0.06**	**-0.04**	**-0.10**
	AIDS and tuberculosis	-0.01	0.01	0.01	0.00	0.01
	Diabetes and obesity	0.01	0.01	0.02	0.00	0.04
	Alcohol abuse and drug dependence	0.00	0.01	0.00	0.00	0.00
	Alzheimer's disease	0.00	0.00	-0.03	-0.01	-0.03
	Dementia and other mental disorders	0.00	0.00	-0.02	-0.01	-0.03
	Rest of causes	-0.03	0.01	-0.05	-0.02	-0.09

Table C.3: Decomposition of gains and losses in life expectancy at age 20 between 1985 and 2000 by causes of death, for selected age bands. Females

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**0.05**	**0.48**	**0.41**	**-0.03**	**0.91**
Circulatory diseases		**0.01**	**0.27**	**0.81**	**0.22**	**1.31**
	Ischaemic heart disease	0.01	0.20	0.65	0.21	1.07
	CVD and stroke	0.01	0.04	0.12	0.04	0.21
	Other circulatory system diseases	-0.01	0.03	0.05	-0.03	0.04
Neoplasms		**0.06**	**0.25**	**-0.07**	**-0.02**	**0.21**
	Bowel cancer	0.00	0.03	0.04	0.00	0.08
	Liver cancer	0.00	0.00	0.00	0.00	0.00
	Lung cancer	0.00	0.02	-0.15	-0.02	-0.14
	Breast cancer	0.03	0.09	0.03	0.00	0.14
	Prostate cancer	0.00	0.00	0.00	0.00	0.01
	Other cancers	0.03	0.08	0.00	-0.01	0.10
	Other digestive organ cancers	0.00	0.02	0.01	0.00	0.03
Respiratory diseases		**0.00**	**-0.01**	**-0.16**	**-0.05**	**-0.23**
	Influenza and pneumonia	0.00	0.00	-0.01	0.00	-0.01
	Chronic lower respiratory disease	0.00	0.00	-0.13	-0.04	-0.18
	Other respiratory diseases	0.00	-0.01	-0.02	-0.01	-0.04
Digestive system		**0.01**	**0.04**	**0.01**	**0.00**	**0.06**
	Gastric and duodenal ulcer	0.00	0.00	0.01	0.00	0.02
	Chronic liver disease	0.01	0.03	0.01	0.00	0.04
	Other digestive system diseases	0.00	0.01	0.00	0.00	0.01
External causes		**0.04**	**0.01**	**0.01**	**0.00**	**0.06**
	Traffic accidents	0.01	0.00	0.00	0.00	0.01
	Self-harm and interpersonal violence	0.04	0.02	0.01	0.00	0.06
	Other external causes	-0.01	0.00	0.01	0.00	-0.01
Other		**-0.06**	**-0.08**	**-0.20**	**-0.17**	**-0.50**
	AIDS and tuberculosis	-0.04	-0.01	0.00	0.00	-0.04
	Diabetes and obesity	-0.01	-0.03	-0.05	-0.01	-0.09
	Alcohol abuse and drug dependence	0.00	0.00	0.00	0.00	0.00
	Alzheimer's disease	0.00	0.00	-0.06	-0.08	-0.14
	Dementia and other mental disorders	0.00	0.00	-0.03	-0.04	-0.07
	Rest of causes	-0.02	-0.04	-0.06	-0.04	-0.16

Table C.4: Decomposition of gains and losses in life expectancy at age 20 between 1985 and 2000 by causes of death, for selected age bands. Males

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**0.36**	**1.03**	**1.19**	**0.03**	**2.61**
Circulatory diseases		**0.07**	**0.68**	**1.00**	**0.10**	**1.84**
	Ischaemic heart disease	0.07	0.57	0.76	0.09	1.49
	CVD and stroke	0.01	0.04	0.11	0.02	0.17
	Other circulatory system diseases	-0.01	0.07	0.12	-0.01	0.18
Neoplasms		**0.06**	**0.30**	**0.14**	**-0.01**	**0.49**
	Bowel cancer	0.00	0.03	0.04	0.00	0.07
	Liver cancer	0.00	-0.02	-0.01	0.00	-0.03
	Lung cancer	0.01	0.17	0.03	-0.01	0.21
	Breast cancer	0.00	0.00	0.00	0.00	0.01
	Prostate cancer	0.00	0.01	0.04	-0.01	0.05
	Other cancers	0.04	0.07	0.01	-0.01	0.12
	Other digestive organ cancers	0.00	0.03	0.02	0.00	0.06
Respiratory diseases		**0.01**	**0.04**	**0.05**	**-0.01**	**0.08**
	Influenza and pneumonia	0.00	0.01	0.02	0.00	0.03
	Chronic lower respiratory disease	0.00	0.03	0.04	-0.01	0.06
	Other respiratory diseases	0.00	0.01	-0.01	-0.01	-0.01
Digestive system		**0.03**	**0.05**	**0.04**	**0.00**	**0.12**
	Gastric and duodenal ulcer	0.00	0.00	0.01	0.00	0.02
	Chronic liver disease	0.02	0.03	0.01	0.00	0.07
	Other digestive system diseases	0.01	0.01	0.02	0.00	0.04
External causes		**0.20**	**0.05**	**0.03**	**0.00**	**0.28**
	Traffic accidents	0.05	0.00	0.00	0.00	0.05
	Self-harm and interpersonal violence	0.08	0.04	0.02	0.00	0.13
	Other external causes	0.07	0.01	0.02	0.00	0.09
Other		**-0.01**	**-0.09**	**-0.07**	**-0.05**	**-0.21**
	AIDS and tuberculosis	-0.07	-0.04	0.00	0.00	-0.10
	Diabetes and obesity	-0.01	-0.04	-0.04	-0.01	-0.10
	Alcohol abuse and drug dependence	0.01	0.00	0.00	0.00	0.01
	Alzheimer's disease	0.00	0.00	-0.02	-0.02	-0.04
	Dementia and other mental disorders	0.00	0.00	-0.01	-0.01	-0.02
	Rest of causes	0.05	-0.01	0.01	-0.01	0.04

Table C.5: Decomposition of gains and losses in life expectancy at age 20 between 2000 and 2015 by causes of death, for selected age bands. Females

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**-0.01**	**0.24**	**1.10**	**0.40**	**1.73**
Circulatory diseases		**0.01**	**0.15**	**0.84**	**0.47**	**1.47**
	Ischaemic heart disease	0.01	0.12	0.58	0.34	1.05
	CVD and stroke	0.01	0.04	0.16	0.10	0.31
	Other circulatory system diseases	0.00	-0.01	0.09	0.03	0.12
Neoplasms		**0.05**	**0.25**	**0.31**	**0.03**	**0.65**
	Bowel cancer	0.00	0.01	0.06	0.02	0.09
	Liver cancer	0.00	-0.01	-0.01	0.00	-0.01
	Lung cancer	0.01	0.09	0.09	0.00	0.18
	Breast cancer	0.02	0.06	0.04	0.01	0.13
	Prostate cancer	0.00	0.00	0.00	0.00	0.00
	Other cancers	0.03	0.09	0.11	0.01	0.24
	Other digestive organ cancers	0.00	0.00	0.02	0.00	0.02
Respiratory diseases		**0.01**	**-0.01**	**0.05**	**0.04**	**0.08**
	Influenza and pneumonia	0.00	0.00	0.03	0.05	0.08
	Chronic lower respiratory disease	0.00	0.00	0.02	-0.01	0.00
	Other respiratory diseases	0.00	0.00	0.00	0.00	0.00
Digestive system		**-0.01**	**-0.03**	**0.02**	**0.02**	**0.00**
	Gastric and duodenal ulcer	0.00	0.00	0.00	0.00	0.01
	Chronic liver disease	-0.01	-0.03	0.00	0.00	-0.04
	Other digestive system diseases	0.00	0.00	0.02	0.02	0.03
External causes		**-0.11**	**-0.10**	**-0.01**	**-0.01**	**-0.22**
	Traffic accidents	0.04	0.01	0.01	0.00	0.06
	Self-harm and interpersonal violence	-0.02	-0.02	0.00	0.00	-0.04
	Other external causes	-0.13	-0.09	-0.02	-0.01	-0.25
Other		**0.03**	**-0.02**	**-0.11**	**-0.15**	**-0.25**
	AIDS and tuberculosis	0.04	0.01	0.00	0.00	0.05
	Diabetes and obesity	-0.01	0.01	0.06	0.01	0.08
	Alcohol abuse and drug dependence	0.00	-0.01	0.00	0.00	-0.01
	Alzheimer's disease	0.00	0.00	-0.06	-0.07	-0.13
	Dementia and other mental disorders	0.00	-0.01	-0.09	-0.11	-0.20
	Rest of causes	0.01	-0.03	-0.03	0.01	-0.03

Table C.6: Decomposition of gains and losses in life expectancy at age 20 between 2000 and 2015 by causes of death, for selected age bands. Males

Level 1	Level 2	20-44	45-64	65-84	85+	Total
All-Cause		**0.02**	**0.37**	**1.39**	**0.35**	**2.13**
Circulatory diseases		**0.02**	**0.29**	**0.90**	**0.28**	**1.48**
	Ischaemic heart disease	0.03	0.29	0.68	0.20	1.20
	CVD and stroke	0.00	0.03	0.14	0.06	0.23
	Other circulatory system diseases	-0.02	-0.03	0.08	0.02	0.05
Neoplasms		**0.04**	**0.26**	**0.46**	**0.06**	**0.82**
	Bowel cancer	0.00	0.02	0.07	0.01	0.09
	Liver cancer	0.00	-0.03	-0.01	0.00	-0.04
	Lung cancer	0.02	0.16	0.23	0.01	0.41
	Breast cancer	0.00	0.00	0.00	0.00	0.00
	Prostate cancer	0.00	0.01	0.07	0.03	0.11
	Other cancers	0.03	0.09	0.10	0.01	0.21
	Other digestive organ cancers	0.00	0.02	0.02	0.00	0.04
Respiratory diseases		**0.00**	**0.00**	**0.13**	**0.06**	**0.20**
	Influenza and pneumonia	0.00	0.00	0.04	0.04	0.09
	Chronic lower respiratory disease	0.00	0.00	0.08	0.02	0.10
	Other respiratory diseases	0.00	0.00	0.00	0.00	0.01
Digestive system		**0.00**	**-0.02**	**0.02**	**0.01**	**0.01**
	Gastric and duodenal ulcer	0.00	0.00	0.01	0.00	0.01
	Chronic liver disease	0.00	-0.02	0.00	0.00	-0.02
	Other digestive system diseases	0.00	0.00	0.02	0.01	0.03
External causes		**-0.17**	**-0.14**	**-0.02**	**0.00**	**-0.33**
	Traffic accidents	0.08	0.01	0.01	0.00	0.11
	Self-harm and interpersonal violence	-0.05	-0.04	0.00	0.00	-0.09
	Other external causes	-0.21	-0.11	-0.02	0.00	-0.35
Other		**0.12**	**-0.03**	**-0.10**	**-0.05**	**-0.06**
	AIDS and tuberculosis	0.09	0.03	0.00	0.00	0.12
	Diabetes and obesity	-0.01	-0.02	0.02	0.00	-0.01
	Alcohol abuse and drug dependence	0.01	-0.01	0.00	0.00	0.00
	Alzheimer's disease	0.00	0.00	-0.02	-0.02	-0.05
	Dementia and other mental disorders	0.00	0.00	-0.06	-0.04	-0.10
	Rest of causes	0.04	-0.03	-0.04	0.01	-0.03

Appendix D: Detecting and modeling of ICD coding changes

In this appendix we describe the details of the statistical methodologies we use for the detection and modeling of the possible disruptions in the mortality trend induced by changes in the ICD coding regimes. These methodologies are based on the approach proposed by Rey et al. (2011).

D.1 METHODOLOGY FOR THE DETECTION OF ICD CODING DISRUPTIONS

Let y_t denote the age-standardized death rate (ASDR) in year t for a given cause of death, computed using the approach described in Section 4.1. Assume that mortality data are available for consecutive years $t_0, t_1, \ldots, t_n$ and let $\mathcal{S} = \{s_1, s_2, \ldots, s_h\}$ be the times at which the coding changes occur with the convention that $s_0 = t_0$ and $s_{h+1} = t_n + 1$. In our dataset we have $t_0 = 1959$, $t_n = 2016$ and $\mathcal{S} = \{1968, 1979, 1999\}$ coinciding with the first year of each ICD regime (see Table 3.1). In addition, for $i = 1, \ldots, h$, let $f^{(i)}(t) = \mathcal{I}_{\{s_{i-1} \leq t < s_i\}}$ denote the indicator function taking value 1 if $t \in [s_{i-1}, s_i)$ and 0 otherwise.

Following Rey et al. (2011), we assume that the ASDR can be modeled as

$$\log y_t = g(t) + \sum_{i=1}^{h} \delta_i f^{(i)}(t) + \epsilon_t, \quad \epsilon_t \sim N(0, \sigma) \quad \text{i.i.d.}, \tag{D.1}$$

where $g(t)$ is a continuous function and ϵ_t is a Gaussian error term. In Equation (D.1) $g(t)$ is the smooth time trend of the underlying "disruption-free" mortality trend, δ_i captures the magnitude of the possible jump in the trend arising from going from regime i to regime $i + 1$, and ϵ_t is the noise around the "disruption-free" mortality trend. Moreover, $\exp(-\delta_i)$ provides an estimate of the comparability ratio associated to the change from the coding regime of period $[s_{i-1}, s_i)$ to the regime of period $[s_i, s_{i+1})$.

For each cause of death and risk factor we estimate model (D.1) under the assumption that $g(t)$ is a thin plate penalized regression spline with the smoothness parameter derived automatically using generalized cross-validation. As noted by Rey et al. (2011), this can be easily accomplished using the **MGCV** `R` package (Wood, 2011, 2020).

We then use the estimated values of parameters δ_i to decide whether a coding change at time s_i induced a statistically significant disruption in the mortality trend. Specifically, for each δ_i we construct the 99% confidence interval (δ_i^L, δ_i^U) and if $0 \notin (\delta_i^L, \delta_i^U)$ we conclude that the disruption is significant. This is equivalent to $1 \notin (\exp(-\delta_i^U), \exp(-\delta_i^L))$, which means that we can reject the hypothesis that the comparability ratio between coding regimes i and $i+1$ is different from 1. Tables D.1 and D.2 show the 99% confidence intervals of the estimated comparability ratios for the different causes of deaths and risk factors, respectively. In these tables, bold numbers indicate significant coding disruptions.

Table D.1: Confidence intervals for ICD coding disruption coefficients for causes of death. Bold numbers indicate a significant coding disruption

	Female			Male		
Cause	**ICD 7-8**	**ICD 8-9**	**ICD 9-10**	**ICD 7-8**	**ICD 8-9**	**ICD 9-10**
All-Cause	(0.98,1.06)	(0.96,1.03)	(0.98,1.06)	(0.99,1.05)	(0.97,1.03)	(0.97,1.04)
Circulatory diseases	(0.96,1.06)	(0.95,1.04)	(0.97,1.06)	(0.97,1.05)	(0.95,1.02)	(0.96,1.03)
Ischaemic heart disease	**(1.17,1.28)**	(0.94,1.02)	(0.98,1.07)	**(1.08,1.16)**	(0.95,1.02)	(0.98,1.04)
CVD and stroke	(0.93,1.05)	(0.94,1.06)	(0.94,1.06)	(0.94,1.04)	(0.96,1.06)	(0.93,1.03)
Other circulatory system diseases	**(0.58,0.66)**	(0.96,1.08)	(0.96,1.08)	**(0.64,0.72)**	(0.92,1.03)	(0.93,1.04)
Neoplasms	(0.99,1.01)	(1,1.02)	(0.99,1.01)	(1,1.02)	**(1,1.02)**	(1,1.02)
Bowel cancer	(0.97,1.03)	**(0.92,0.97)**	(0.99,1.05)	(0.98,1.03)	**(0.94,0.99)**	**(1.01,1.06)**
Liver cancer	**(0.6,0.66)**	**(1.25,1.37)**	(0.96,1.06)	**(0.53,0.6)**	**(1.26,1.4)**	(0.95,1.06)
Lung cancer	**(1.06,1.12)**	(0.98,1.03)	(0.97,1.03)	**(1.01,1.05)**	**(0.96,1)**	(1,1.03)
Breast cancer	(0.97,1.03)	**(0.95,1)**	(0.97,1.03)	–	–	–
Prostate cancer	–	–	–	(0.98,1.07)	(0.98,1.06)	(0.96,1.05)
Other cancers	**(1.01,1.04)**	**(1.02,1.05)**	(0.99,1.02)	(0.99,1.03)	**(1.04,1.08)**	(0.99,1.03)
Other digestive organ cancers	(1,1.05)	(0.97,1.02)	(0.98,1.03)	**(1.01,1.05)**	(0.96,1)	(0.99,1.03)
Respiratory diseases	(0.97,1.31)	**(0.7,0.93)**	(0.92,1.23)	(0.96,1.2)	**(0.76,0.95)**	(0.94,1.17)
Influenza and pneumonia	(0.93,1.42)	**(0.48,0.72)**	(0.88,1.32)	(0.96,1.35)	**(0.48,0.68)**	(0.87,1.22)
Chronic lower respiratory disease	(0.83,1.01)	**(2.13,2.56)**	(0.94,1.13)	(0.82,1)	**(2.22,2.68)**	(0.94,1.13)
Other respiratory diseases	**(1.13,1.41)**	**(0.47,0.58)**	**(1.01,1.25)**	**(1.23,1.51)**	**(0.31,0.37)**	**(1.02,1.24)**
Digestive system	(0.96,1.04)	**(1.09,1.18)**	(0.99,1.06)	(0.97,1.06)	**(1.03,1.11)**	(0.99,1.06)
Gastric and duodenal ulcer	(0.95,1.21)	(0.88,1.11)	(0.93,1.17)	(0.92,1.15)	(0.82,1.01)	(0.96,1.19)
Chronic liver disease	(0.94,1.06)	(0.95,1.06)	(0.97,1.08)	(0.94,1.04)	(0.95,1.05)	(0.98,1.08)
Other digestive system diseases	(0.94,1.03)	**(1.18,1.29)**	(0.98,1.07)	(0.98,1.07)	**(1.16,1.25)**	(0.98,1.06)
External causes	(0.93,1.06)	(0.98,1.1)	(0.89,1)	(0.98,1.13)	**(1.03,1.18)**	(0.87,1.01)
Traffic accidents	(0.87,1.23)	**(0.63,0.87)**	(0.77,1.07)	(0.9,1.25)	**(0.67,0.91)**	(0.85,1.15)
Self-harm and interpersonal violence	(0.89,1)	(0.96,1.07)	**(0.89,1)**	(0.95,1.09)	**(1.04,1.19)**	(0.88,1)
Other external causes	(0.93,1.06)	**(1.14,1.29)**	(0.89,1.01)	(0.99,1.16)	**(1.25,1.46)**	**(0.84,0.99)**
Other	(0.98,1.09)	**(1.01,1.12)**	(0.99,1.1)	(0.97,1.09)	**(1.01,1.13)**	(0.96,1.08)
AIDS and tuberculosis	(0.87,1.56)	**(1.29,2.23)**	(0.61,1.06)	(0.65,1.93)	**(1.06,2.92)**	(0.65,1.8)
Diabetes and obesity	**(1.01,1.17)**	(0.99,1.14)	(0.95,1.09)	**(1.02,1.16)**	**(1.05,1.19)**	(0.92,1.04)
Alcohol abuse and drug dependence	(0.93,1.41)	(0.97,1.44)	**(0.67,0.99)**	(0.98,1.39)	(0.87,1.21)	(0.81,1.13)
Alzheimer's disease	–	–	(0.78,1.09)	–	–	(0.76,1.09)
Dementia and other mental disorders	(0.78,1.15)	**(1.55,2.24)**	(0.96,1.39)	(0.8,1.18)	**(1.53,2.22)**	(0.87,1.26)
Rest of causes	(0.96,1.08)	(0.96,1.07)	(0.99,1.1)	(0.93,1.07)	(0.94,1.06)	(0.96,1.09)

Table D.2: Confidence intervals for ICD coding disruption coefficients for risk factors. Bold numbers indicate a significant coding disruption

	Female			Male		
Risk Factor	**ICD 7-8**	**ICD 8-9**	**ICD 9-10**	**ICD 7-8**	**ICD 8-9**	**ICD 9-10**
AIDS and tuberculosis	(0.64,1.15)	**(0.45,0.77)**	(0.94,1.63)	(0.52,1.53)	**(0.34,0.94)**	(0.56,1.53)
Alcohol abuse	(0.93,1.04)	(0.93,1.04)	(0.94,1.05)	(0.93,1.03)	(0.96,1.05)	(0.92,1.02)
Dementia and Alzheimer's disease	**(1.04,1.34)**	**(0.5,0.64)**	(0.83,1.06)	(0.98,1.3)	**(0.45,0.59)**	(0.87,1.13)
Diabetes and obesity	**(0.85,0.99)**	(0.87,1.01)	(0.91,1.06)	**(0.86,0.98)**	**(0.84,0.95)**	(0.96,1.09)
Drug dependency	(0.98,1.35)	(0.85,1.15)	(1,1.35)	(0.84,1.44)	(0.71,1.18)	(0.92,1.54)
Homicide	(0.97,1.21)	**(0.76,0.94)**	(0.92,1.14)	(0.76,1.09)	**(0.58,0.81)**	(0.94,1.31)
Hypertensive disease	**(2.11,2.37)**	**(0.5,0.56)**	(0.94,1.05)	**(1.91,2.18)**	**(0.51,0.57)**	**(1.01,1.15)**
Self-harm	(0.96,1.11)	**(1.03,1.19)**	**(1,1.16)**	(0.97,1.07)	**(1.03,1.13)**	**(1.01,1.11)**
Smoking	**(0.89,0.95)**	(0.97,1.02)	(0.97,1.03)	**(0.95,0.99)**	**(1,1.04)**	(0.97,1)

D.2 METHODOLOGY FOR MODELING ICD CODING DISRUPTIONS IN THE PCI MODEL

As discussed in section 5.3.1.2, the mortality rate model with coding disruptions underlying the estimation of the PCi model is given by

$$\log m_{x,t} = A_x + K_t + \Gamma_{t-x} + \sum_{i=1}^{h} \delta_i f^{(i)}(t). \tag{D.2}$$

In a similar manner to the standard APC model (Villegas et al., 2018), the model defined in Equation (D.2) is only identifiable up to a set of transformations. Specifically, given constants c_1, ϕ_1, ϕ_2, and a_i, $i = 1, \ldots, h$, we can transform the parameters in Equation (D.2) in the following ways

$$(A_x, K_t, \Gamma_{t-x}, \delta_1, \ldots, \delta_h) \to (A_x + \phi_1 - \phi_0 x, K_t + \phi_1 t, \Gamma_{t-x} - \phi_1 - \phi_2(t-x), \delta_1, \ldots, \delta_h) \tag{D.3}$$

$$(A_x, K_t, \Gamma_{t-x}, \delta_1, \ldots, \delta_h) \to (A_x + c_1, K_t - c_1, \Gamma_{t-x}, \delta_1, \ldots, \delta_h) \tag{D.4}$$

$$(A_x, K_t, \Gamma_{t-x}, \delta_1, \ldots, \delta_h) \to (A_x, K_t - \sum_{i=1}^{h} a_i f^{(i)}(t), \Gamma_{t-x}, \delta_1 + a_1, \ldots, \delta_h + a_h), \tag{D.5}$$

leaving the fitted mortality rates unchanged.

The transformation in Equation (D.5) imply that the fitted rates produced by the APC model with coding disruptions are the same as the rates from the standard APC model:

$$\log m_{x,t} = A_x + K_t + \Gamma_{t-x}. \tag{D.6}$$

We exploit this fact and estimate the model in Equation (D.2) in two steps. In the first step, we estimate the standard APC model in Equation (D.6) under the constraints,

$$K_{t_n} = 0, \quad \sum_c \Gamma_c = 0 \quad \sum_c c\Gamma_c = 0. \tag{D.7}$$

In a second step, we find constants $a_1, \ldots, a_h$ so that K_t captures the underlying "disruption-free" period mortality trend. We accomplish this using a similar approach to the one discussed in Section D.1 to detect coding disruptions. Specifically, given the estimated values of K_t from the first step, we find constants $a_1, \ldots, a_h$ by fitting the model

$$K_t = g(t) + \sum_{i=1}^{h} a_i f^{(i)}(t) + \epsilon_t, \quad \epsilon_t \sim N(0, \sigma) \quad \text{i.i.d.}, \tag{D.8}$$

where $g(t)$ is thin plate smoothing spline. Then, with the estimated values of $a_1, \ldots, a_h$ from model (D.8) and using the transformation defined in Equation (D.5), we recover the "disruption-free" trend, $\tilde{K}_t$, with the expression

$$\tilde{K}_t = K_t - \sum_{i=1}^{h} a_i f^{(i)}(t). \tag{D.9}$$

Finally, the parameters of the PCi model with coding disruptions are derived as

$$\kappa_t^{(i)} = -\Delta K_t^{(i)} = -(K_t^{(i)} - K_{t-1}^{(i)}) \quad \text{and} \quad \gamma_c = -\Delta\Gamma_c = -(\Gamma_c - \Gamma_{c-1}). \tag{D.10}$$

Appendix E: Unsmooth improvement stripes

In this appendix we present the unsmooth mortality improvement stripes obtained by applying model (5.8). Figures E.1 and E.2 present the results for the different causes and Figure E.3 for the risk factors.

Figure E.1: Unsmooth improvement stripes for causes of death, Females, 1959–2016, 20–89

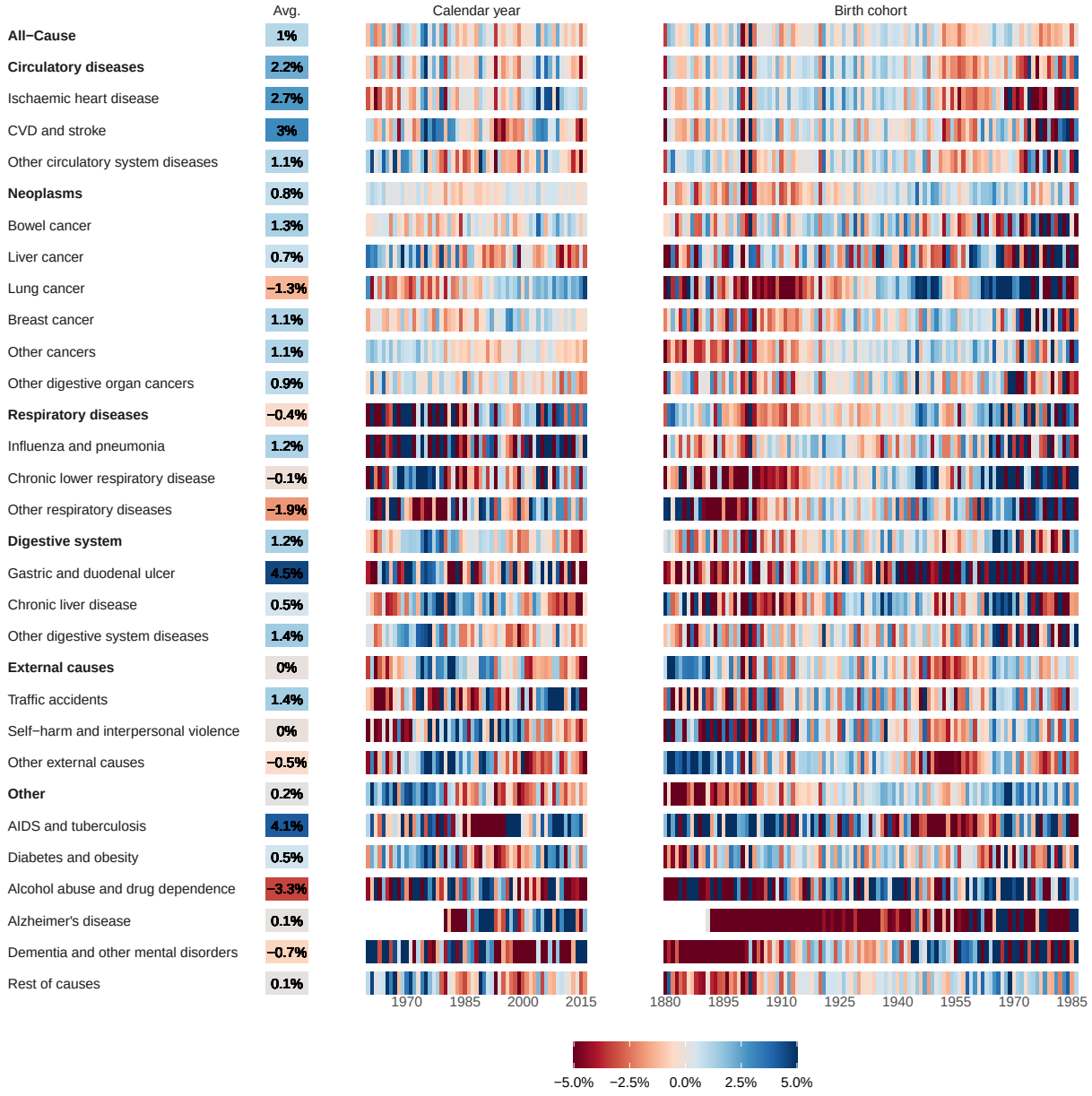


Figure E.2: Unsmooth improvement stripes for causes of death, Males, 1959–2016, 20–89

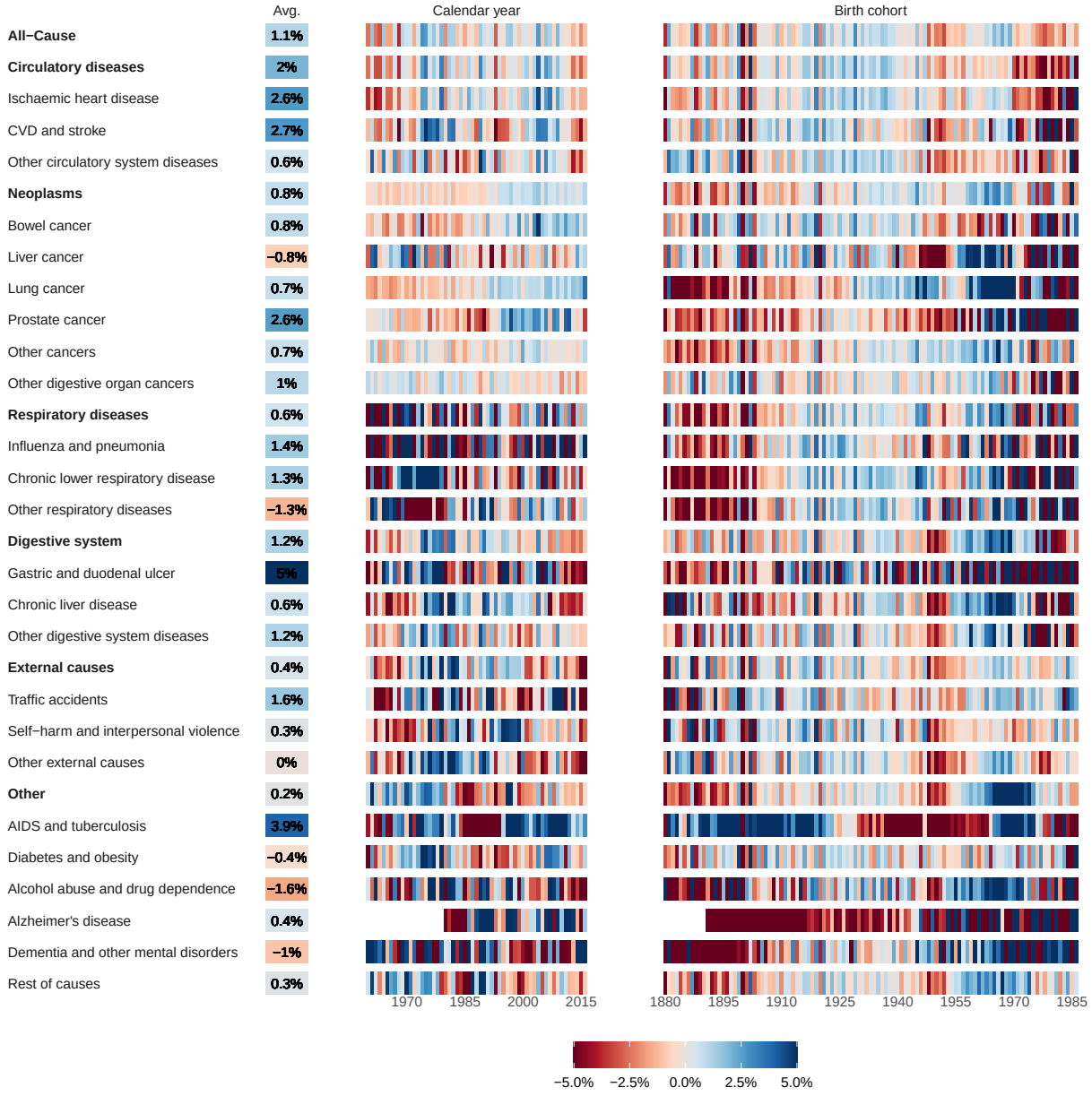
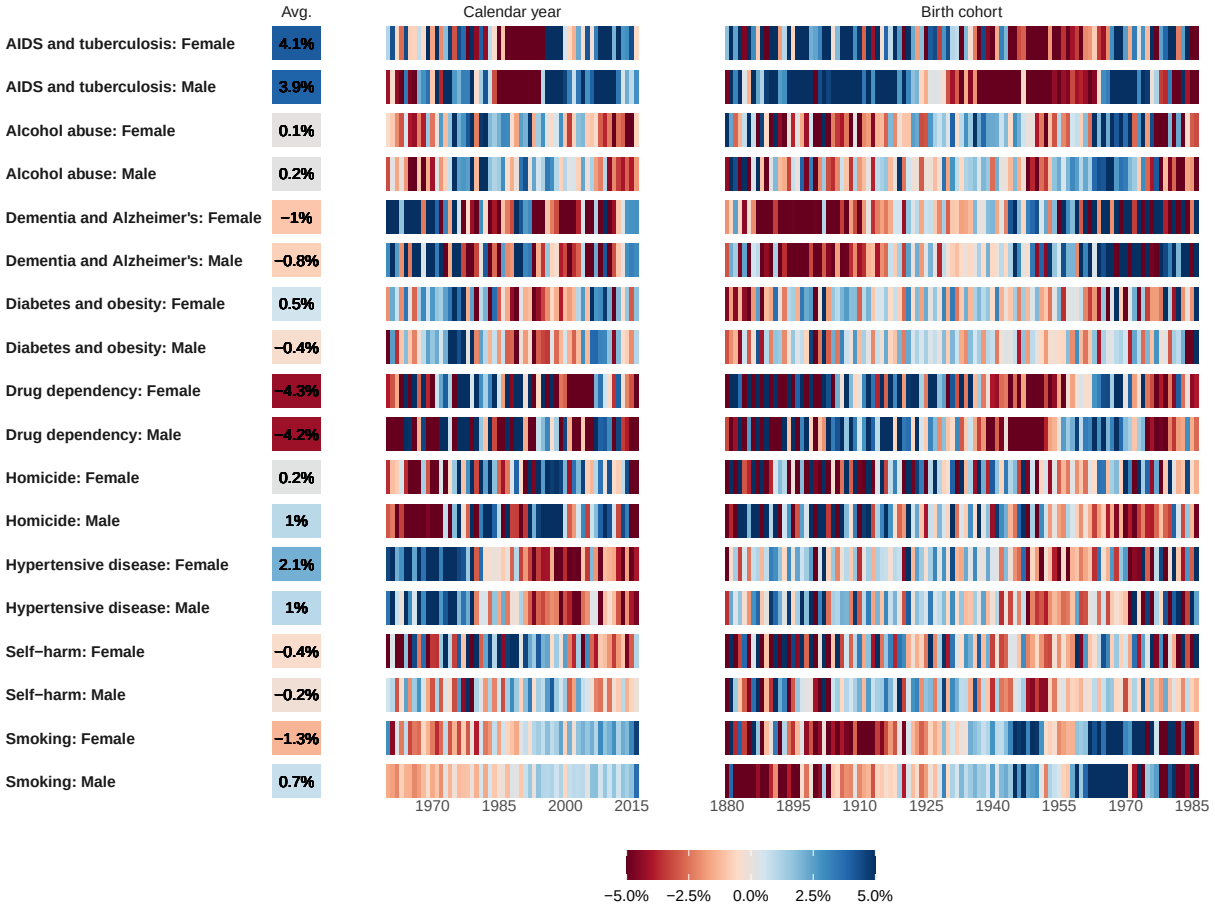


Figure E.3: Unsmooth improvement stripes for risk factors, 1959–2016, 20–89



Appendix F: PCi model parameters for females

In this appendix we present the results of applying the different versions of the PCi model discussed in Section 5.3 to female mortality data for the different causes of death and risk factors.

The figure for each cause and each risk factor presents the parameter estimates associated with:

- the "standard" PCi model defined by Equation (5.7),
- the PCi model allowing for "coding breaks" defined by Equation (5.8), and
- the "smooth" PCi model defined by Equation (5.9).

In addition, for each of the models, the figure includes:

- the fitted parameters in the mortality rate scale (top panes),
- the fitted parameters in the improvement rate scale using line plots (middle panes), and
- the fitted parameters in the improvement rate scale using mortality improvement stripes (bottom panes).

F.1 CAUSES OF DEATH

- All-Cause mortality: Figure F.1
  - Circulatory diseases: Figure F.2
    - Ischaemic heart disease: Figure F.3
    - CVD and stroke: Figure F.4
    - Other circulatory system diseases: Figure F.5
  - Neoplasms: Figure F.6
    - Bowel cancer: Figure F.7
    - Liver cancer: Figure F.8
    - Lung cancer: Figure F.9
    - Breast cancer: Figure F.10
    - Other cancers: Figure F.11
    - Other digestive organ cancers: Figure F.12
  - Respiratory diseases: Figure @ref(fig:plotRespiratory iseasesFemale)
    - Influenza and pneumonia: Figure F.14
    - Chronic lower respiratory disease: Figure F.15
    - Other respiratory diseases: Figure F.16
  - Digestive system: Figure F.17

    - Gastric and duodenal ulcer: Figure F.18
    - Chronic liver disease: Figure F.19
    - Other digestive system diseases: Figure F.20
  - External causes: Figure F.21
    - Traffic accidents: Figure F.22
    - Self-harm and interpersonal violence: Figure F.23
    - Other external causes: Figure F.24
  - Other: Figure F.25
    - AIDS and tuberculosis: Figure F.26
    - Diabetes and obesity: Figure F.27
    - Alcohol abuse and drug dependence: Figure F.28
    - Alzheimer's disease: Figure F.29
    - Dementia and other mental disorders: Figure F.30
    - Rest of causes: Figure F.31

F.2 RISK FACTORS

- AIDS and tuberculosis: Figure F.32
- Alcohol abuse: Figure F.33
- Dementia and Alzheimer's disease: Figure F.34
- Diabetes and obesity: Figure F.35
- Drug dependency: Figure F.36
- Homicide: Figure F.37
- Hypertensive disease: Figure F.38
- Self-harm: Figure F.39
- Smoking: Figure F.40

Figure F.1: Fitted parameters for the PCi model for All-Causes of death, females, 1959–2016, 20–89

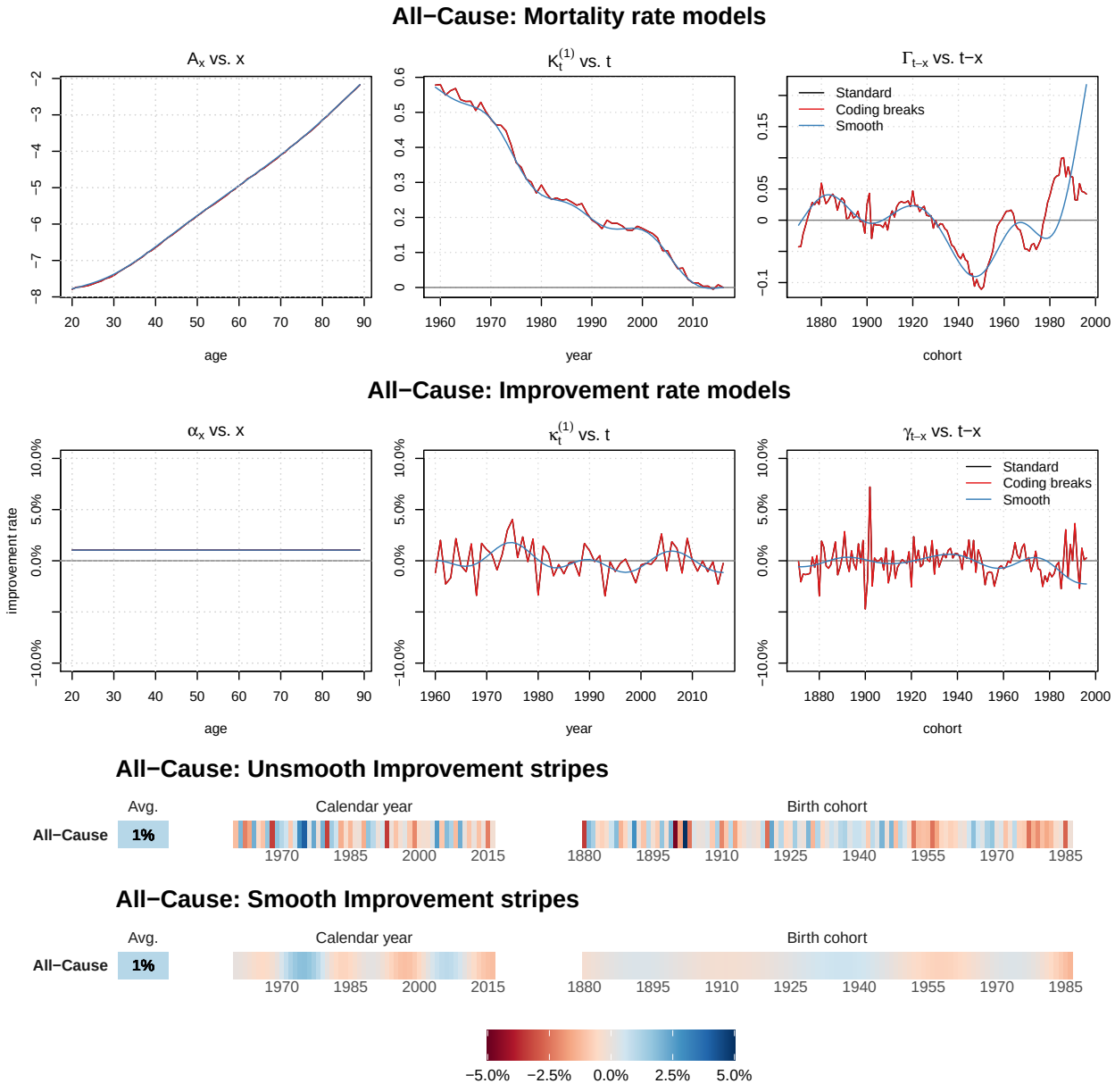


Figure F.2: Fitted parameters for the PCi model for circulatory diseases, females, 1959–2016, 20–89

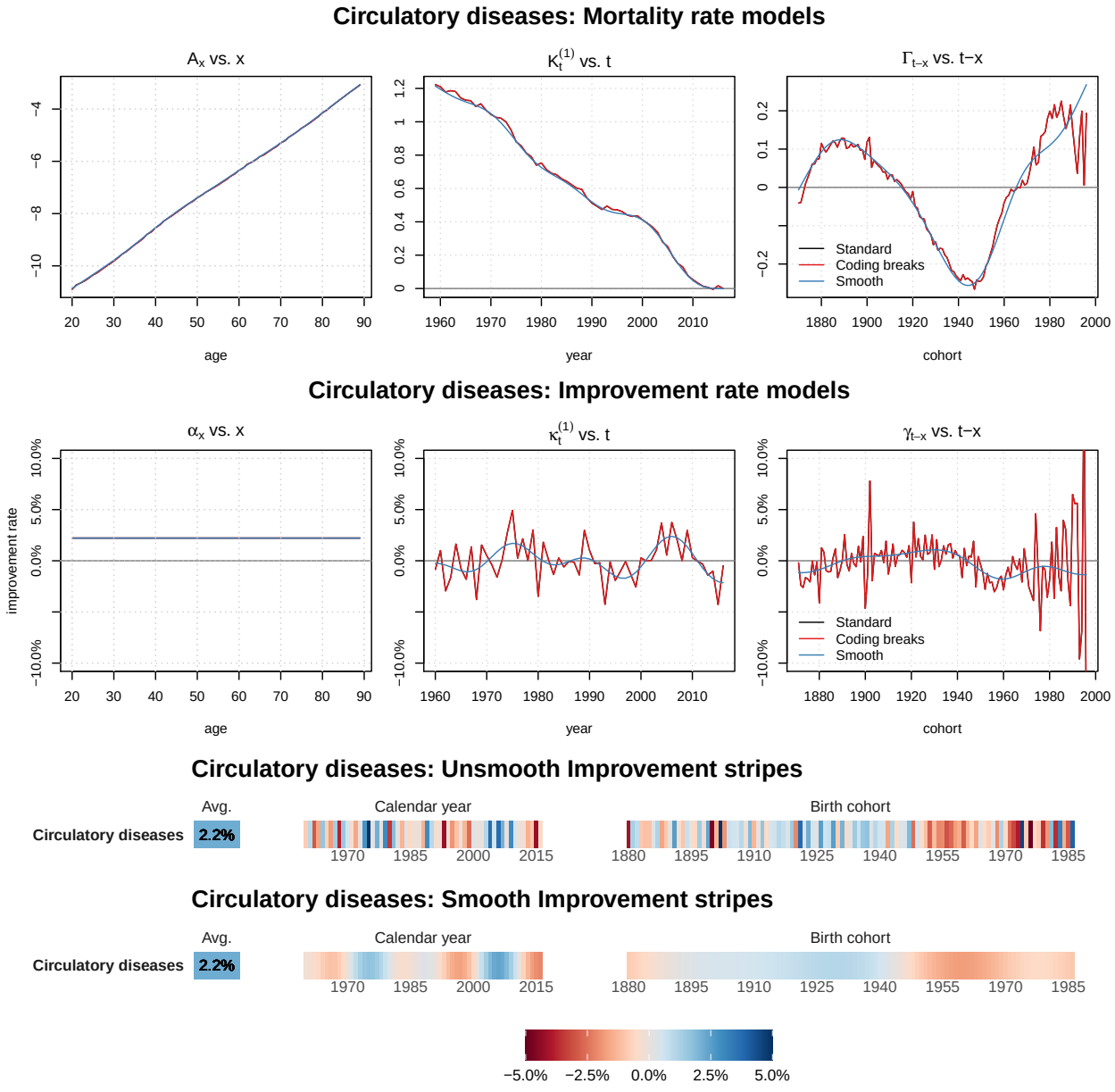


Figure F.3: Fitted parameters for the PCi model for ischaemic heart disease, females, 1959–2016, 20–89

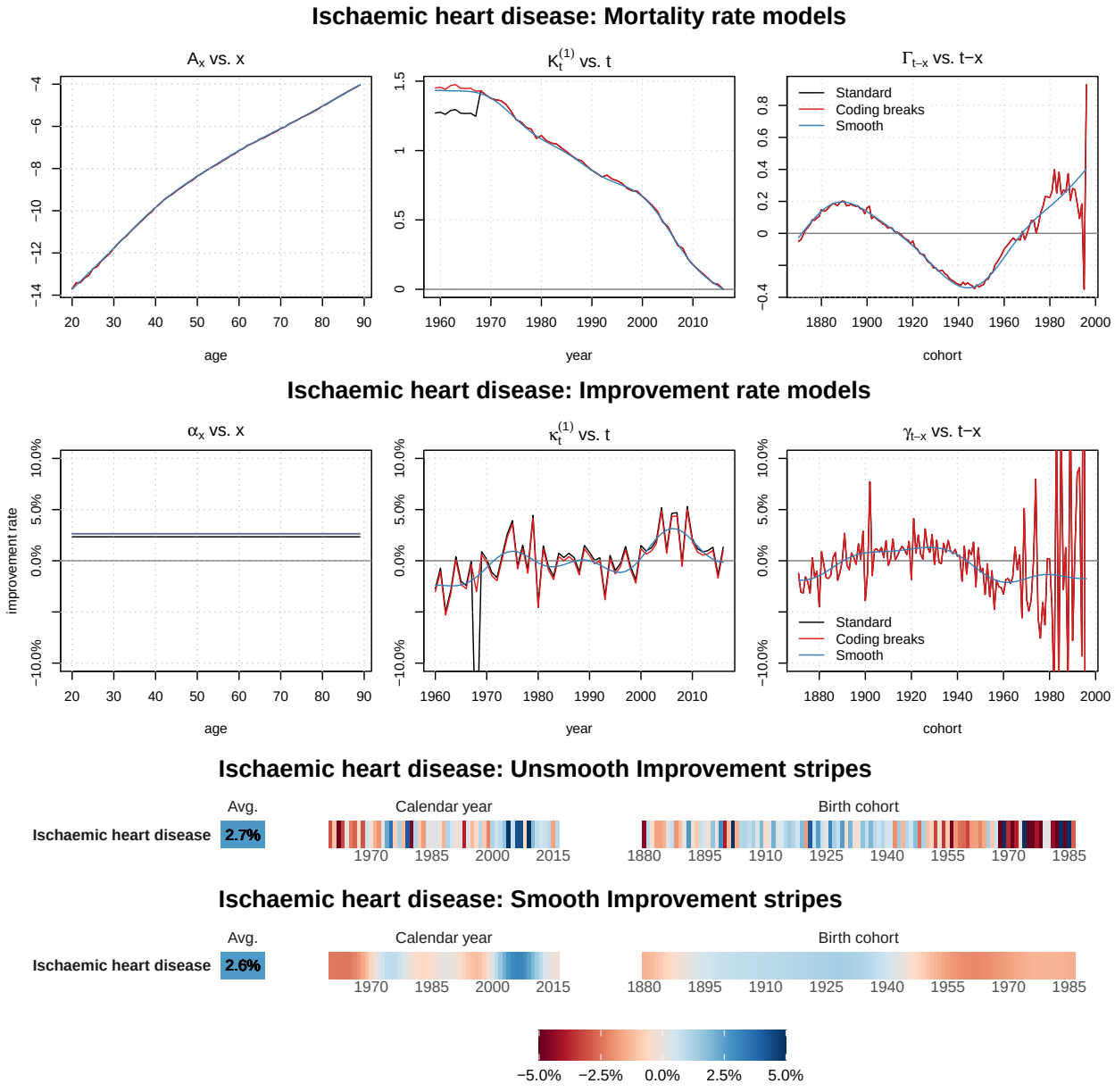


Figure F.4: Fitted parameters for the PCi model for CVD and stroke, females, 1959–2016, 20–89

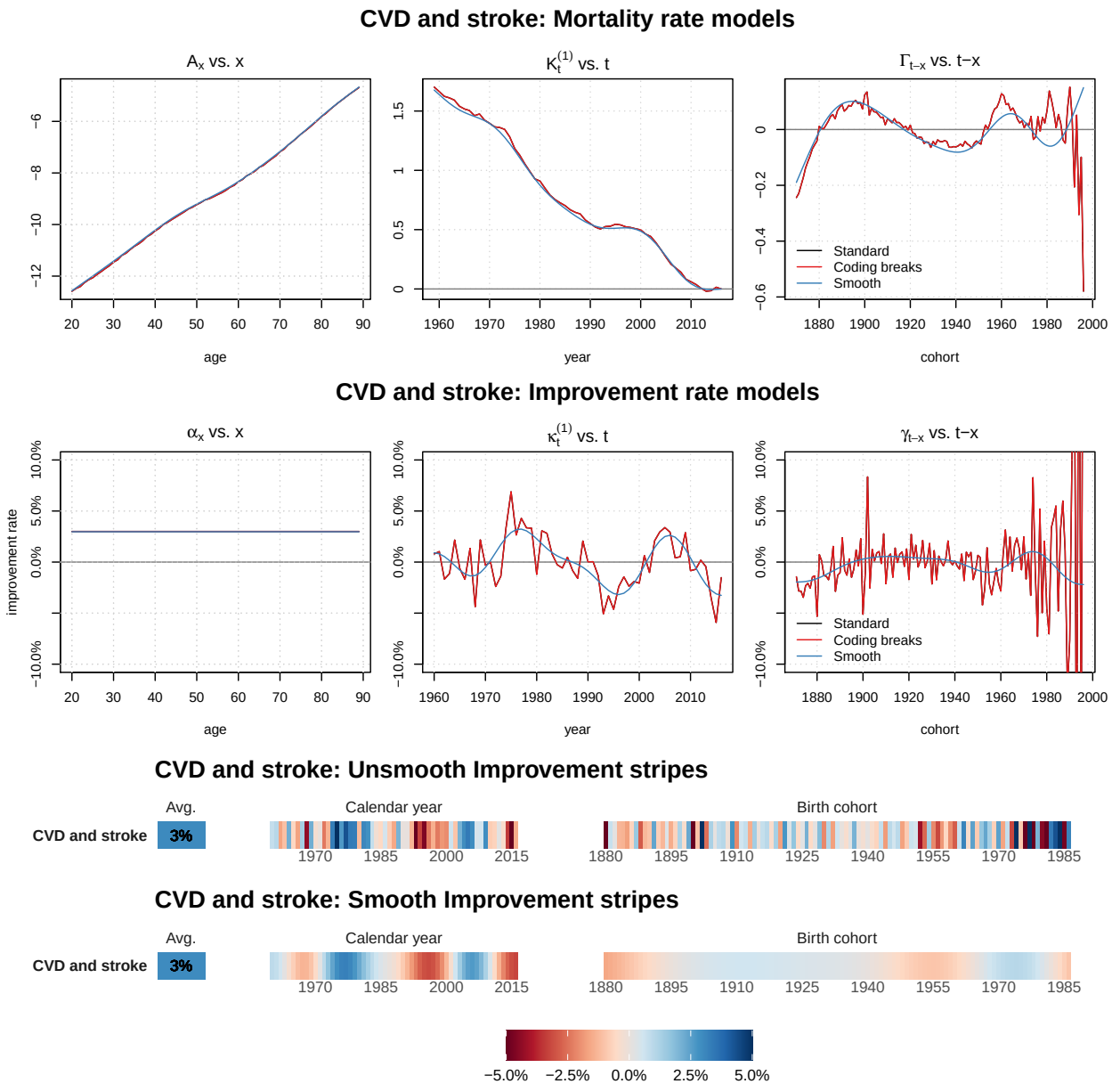


Figure F.5: Fitted parameters for the PCi model for other circulatory system diseases, females, 1959–2016, 20–89

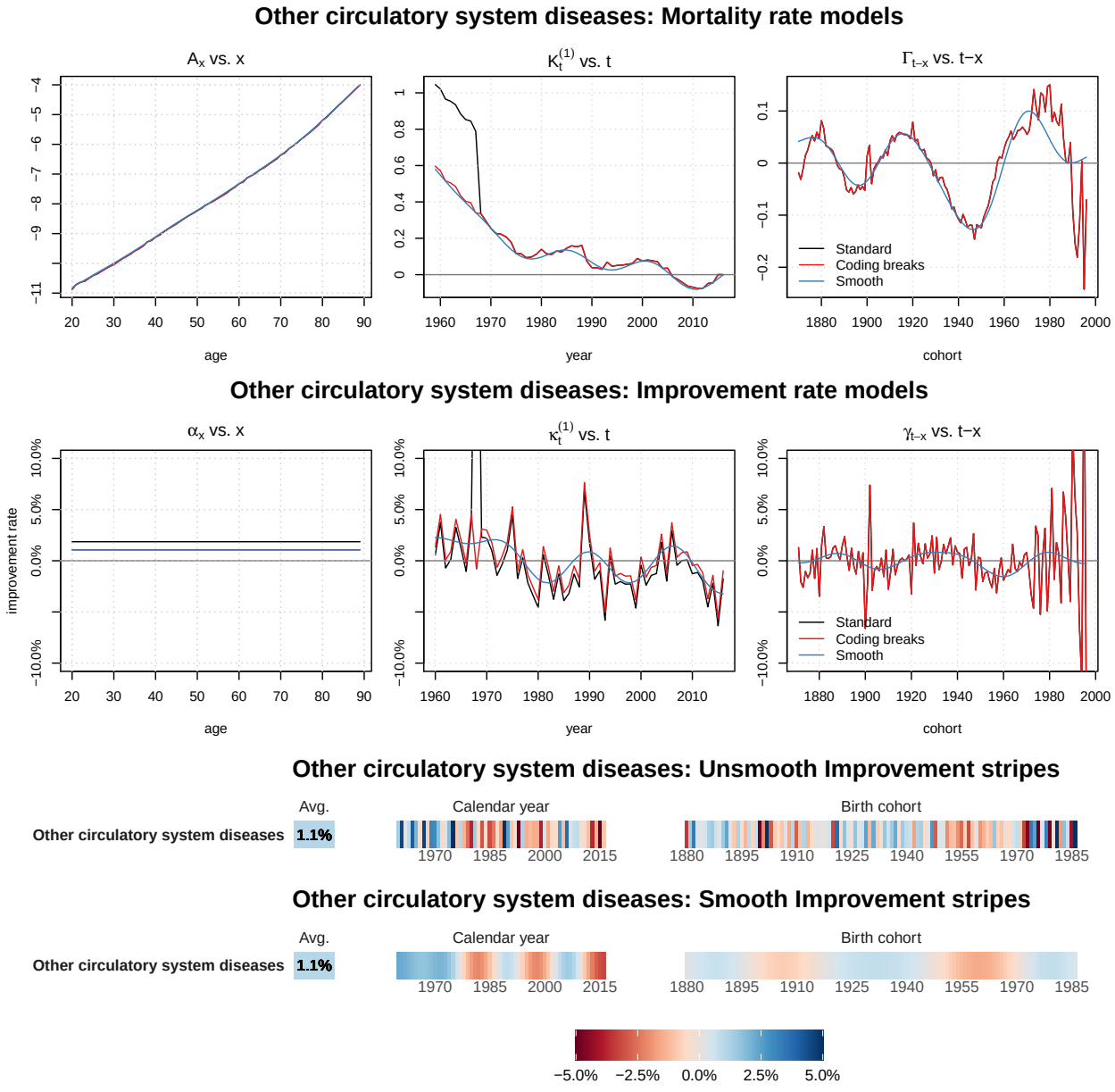


Figure F.6: Fitted parameters for the PCi model for neoplasms, females, 1959–2016, 20–89

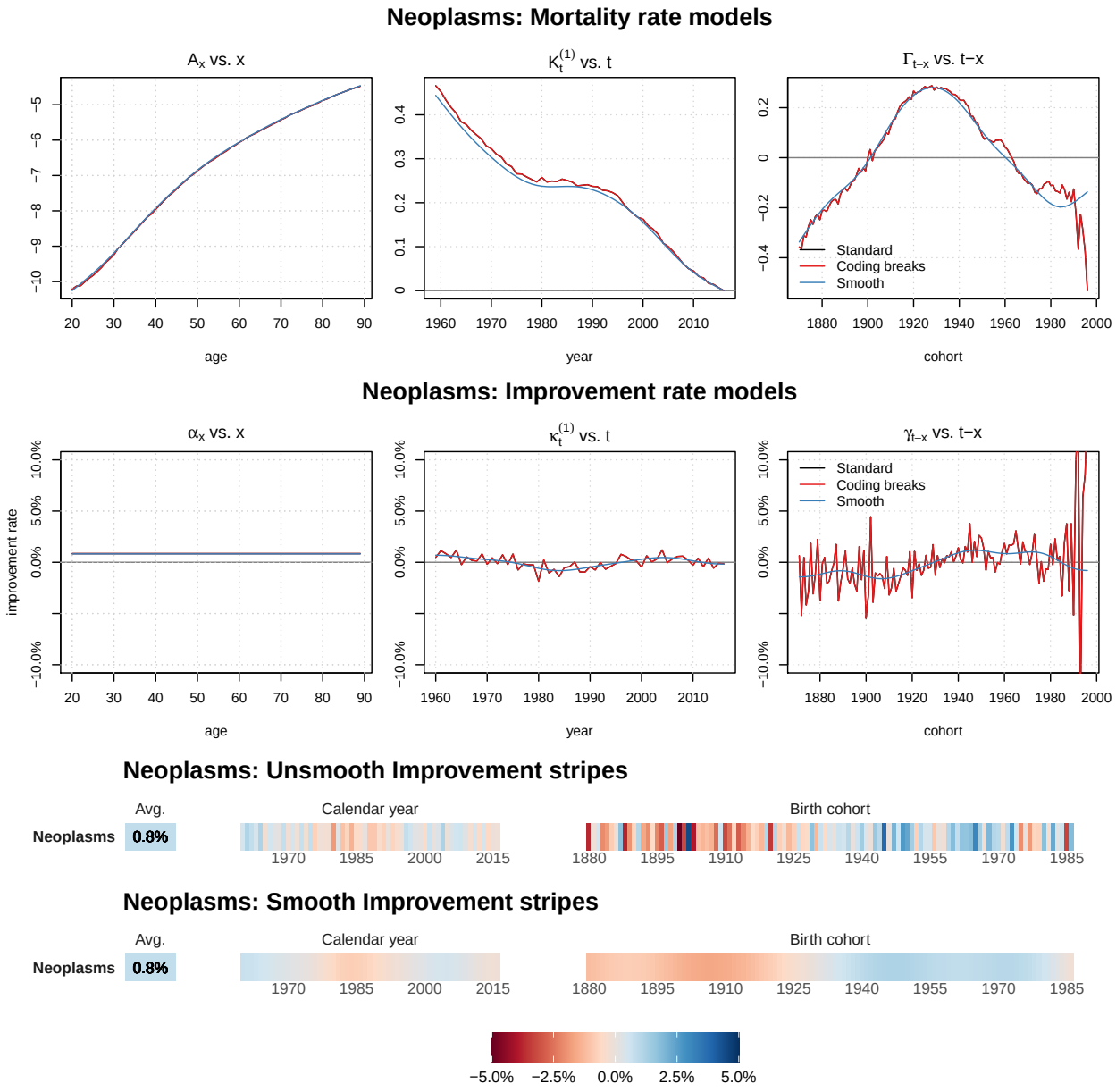


Figure F.7: Fitted parameters for the PCi model for bowel cancer, females, 1959–2016, 20–89

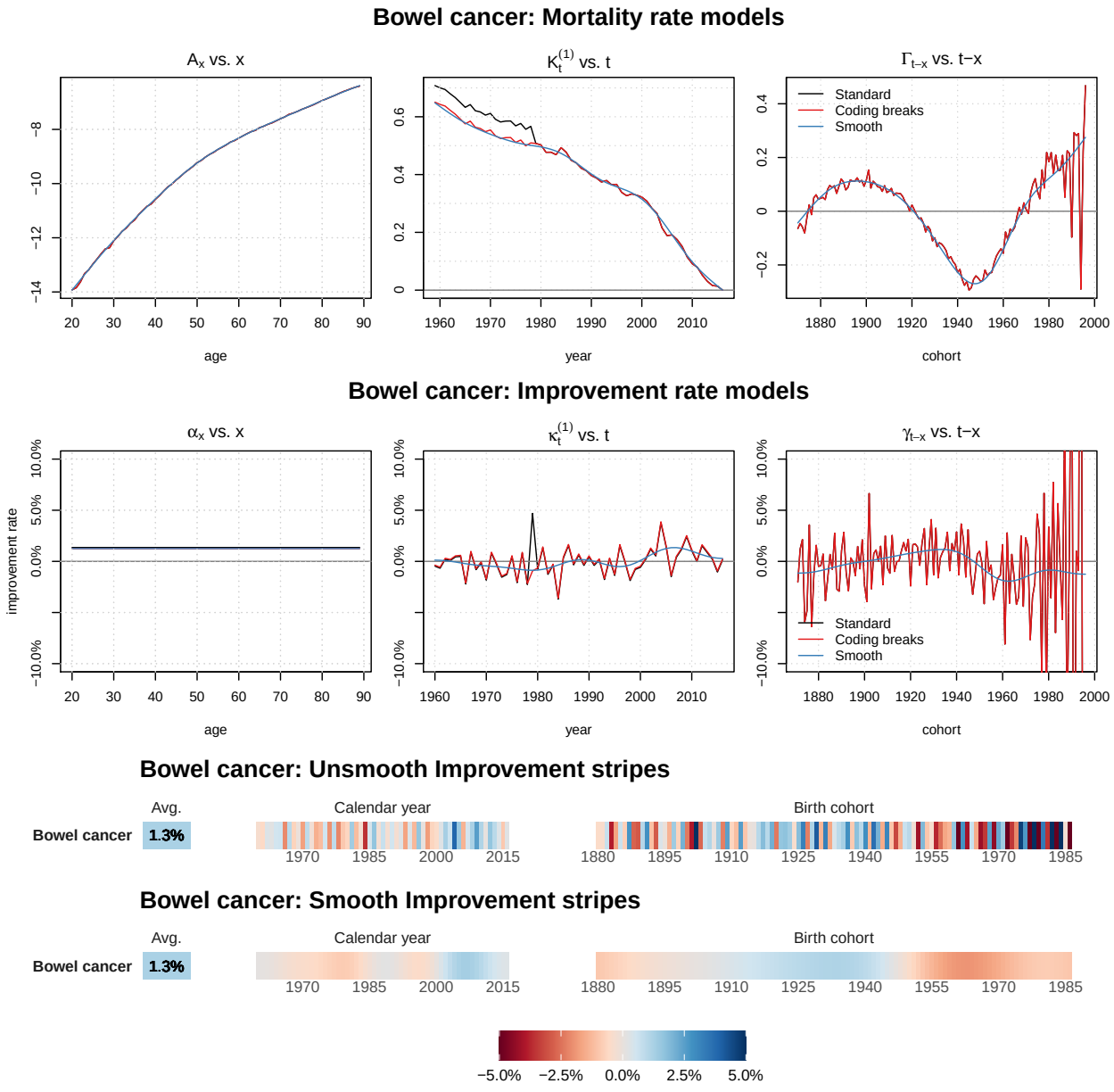


Figure F.8: Fitted parameters for the PCi model for liver cancer, females, 1959–2016, 20–89

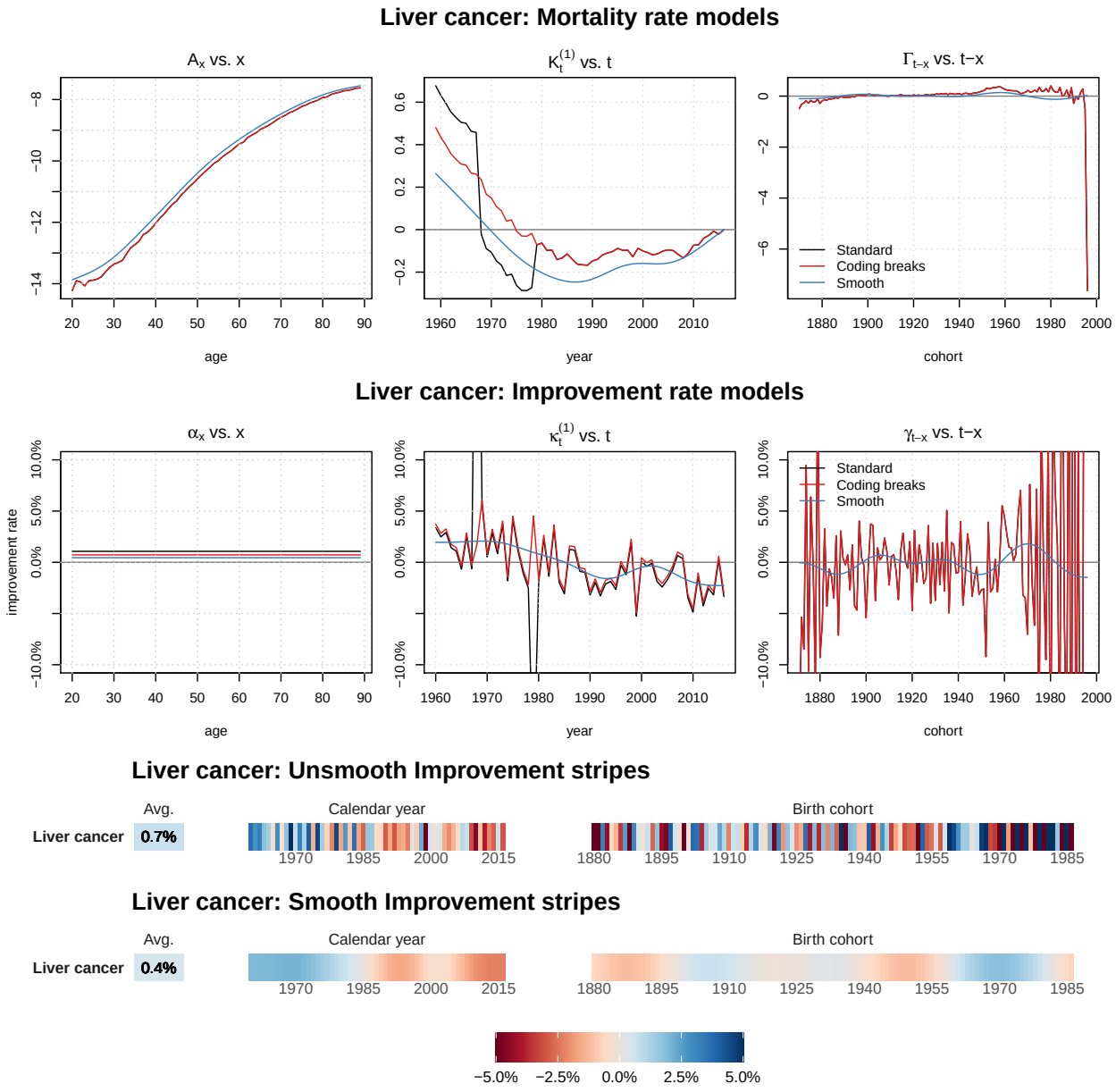


Figure F.9: Fitted parameters for the PCi model for lung cancer, females, 1959–2016, 20–89

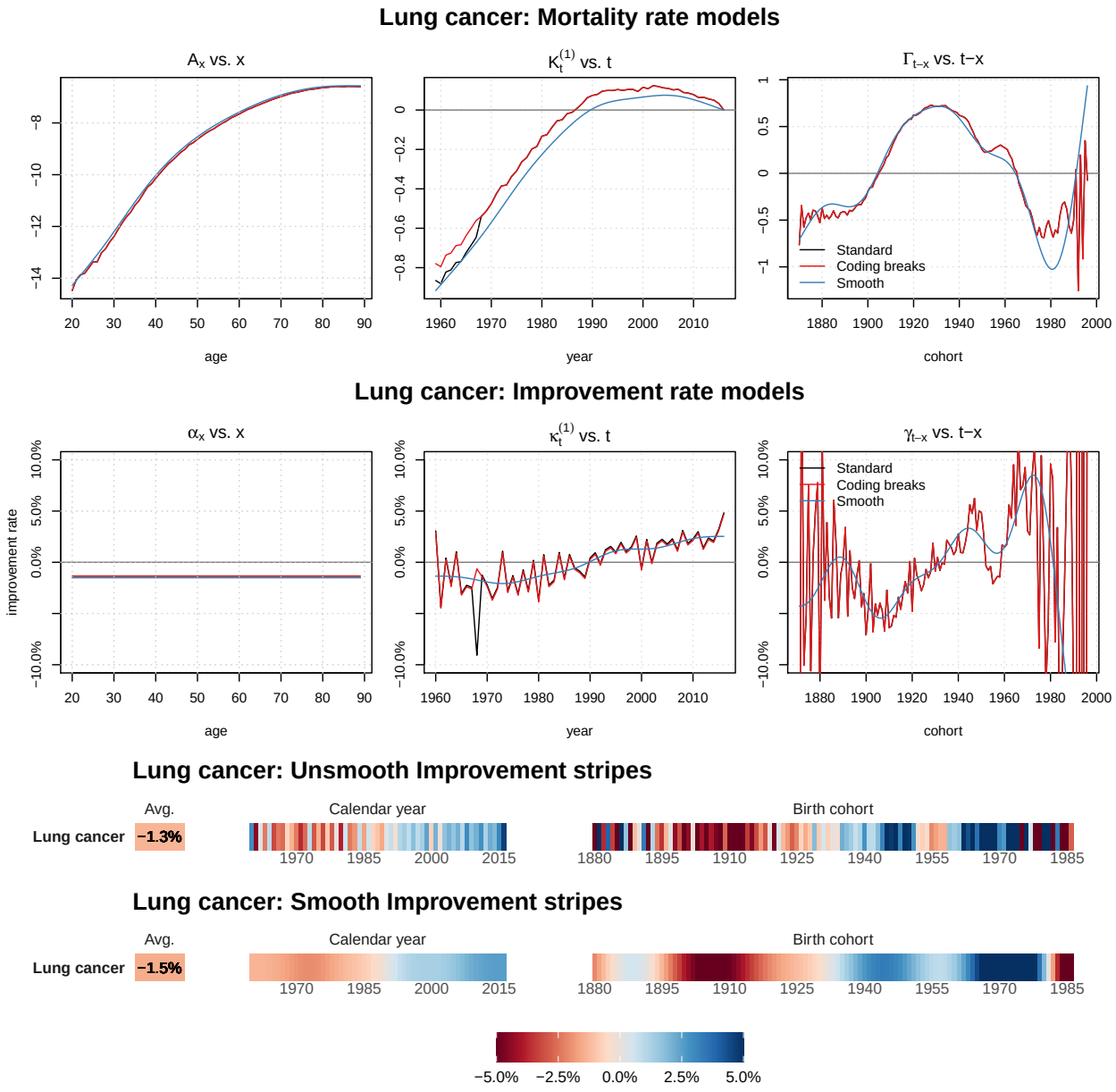


Figure F.10: Fitted parameters for the PCi model for breast cancer, females, 1959–2016, 20–89

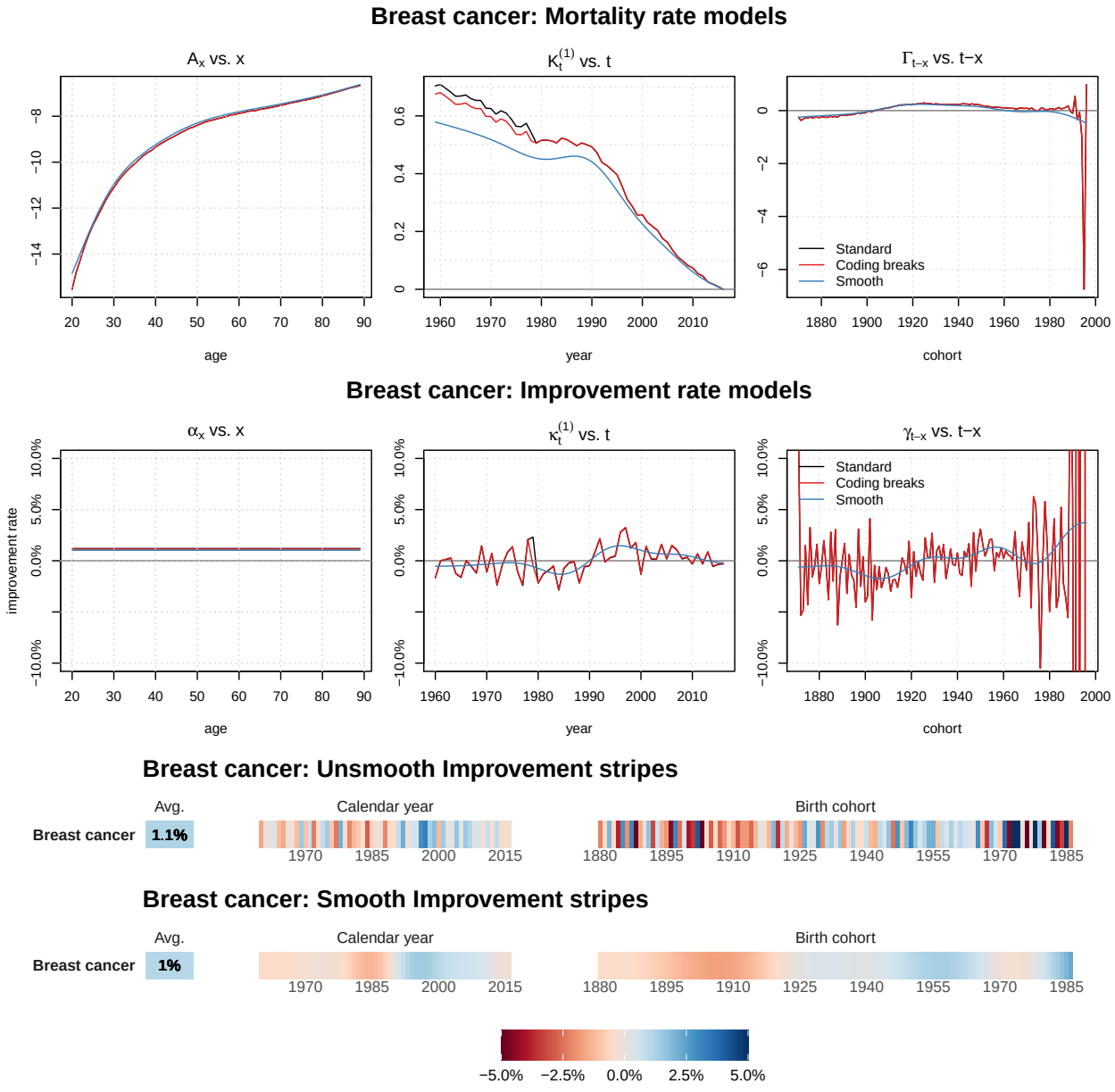


Figure F.11: Fitted parameters for the PCi model for other cancers, females, 1959–2016, 20–89

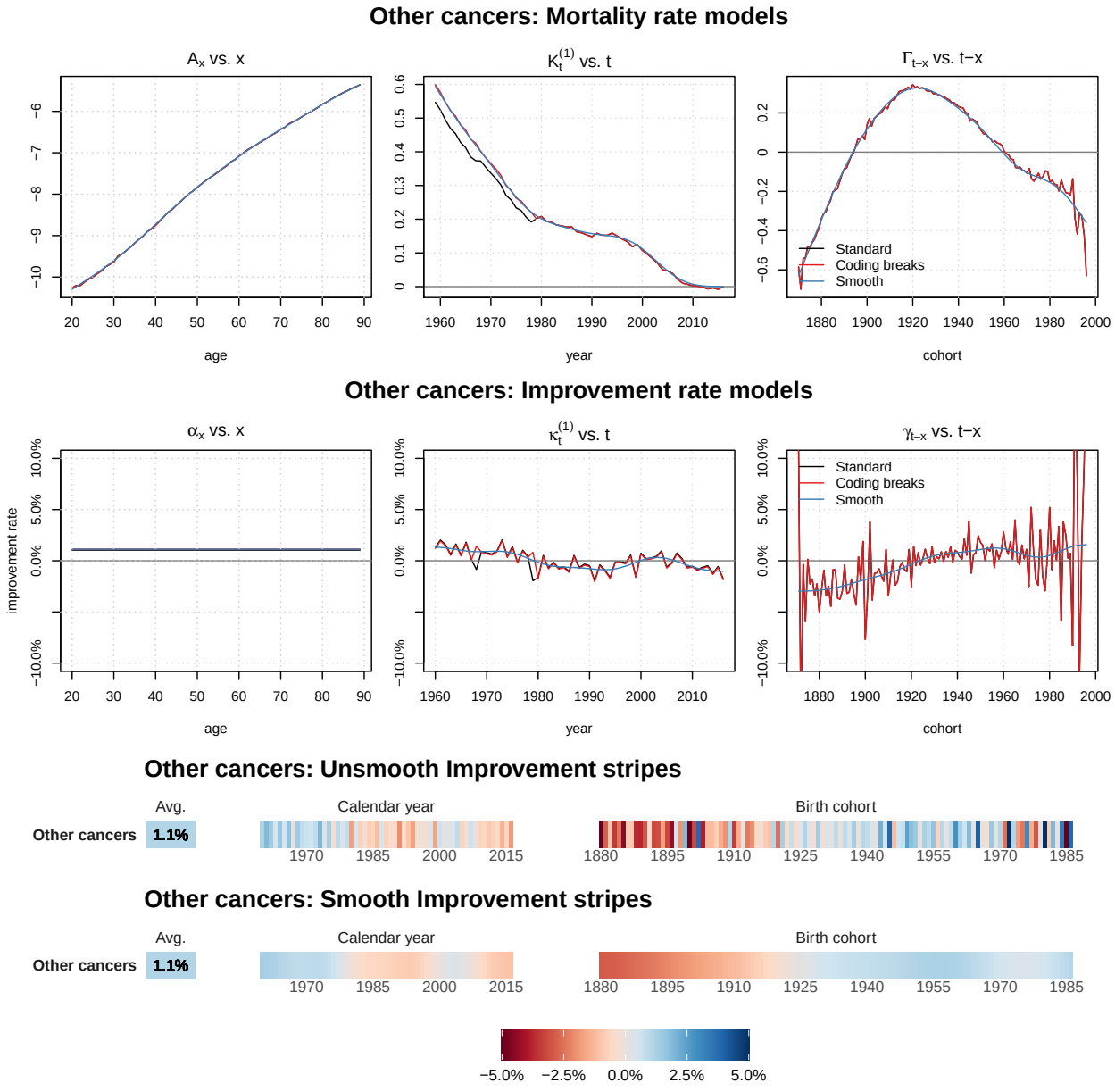


Figure F.12: Fitted parameters for the PCi model for other digestive organ cancers, females, 1959–2016, 20–89

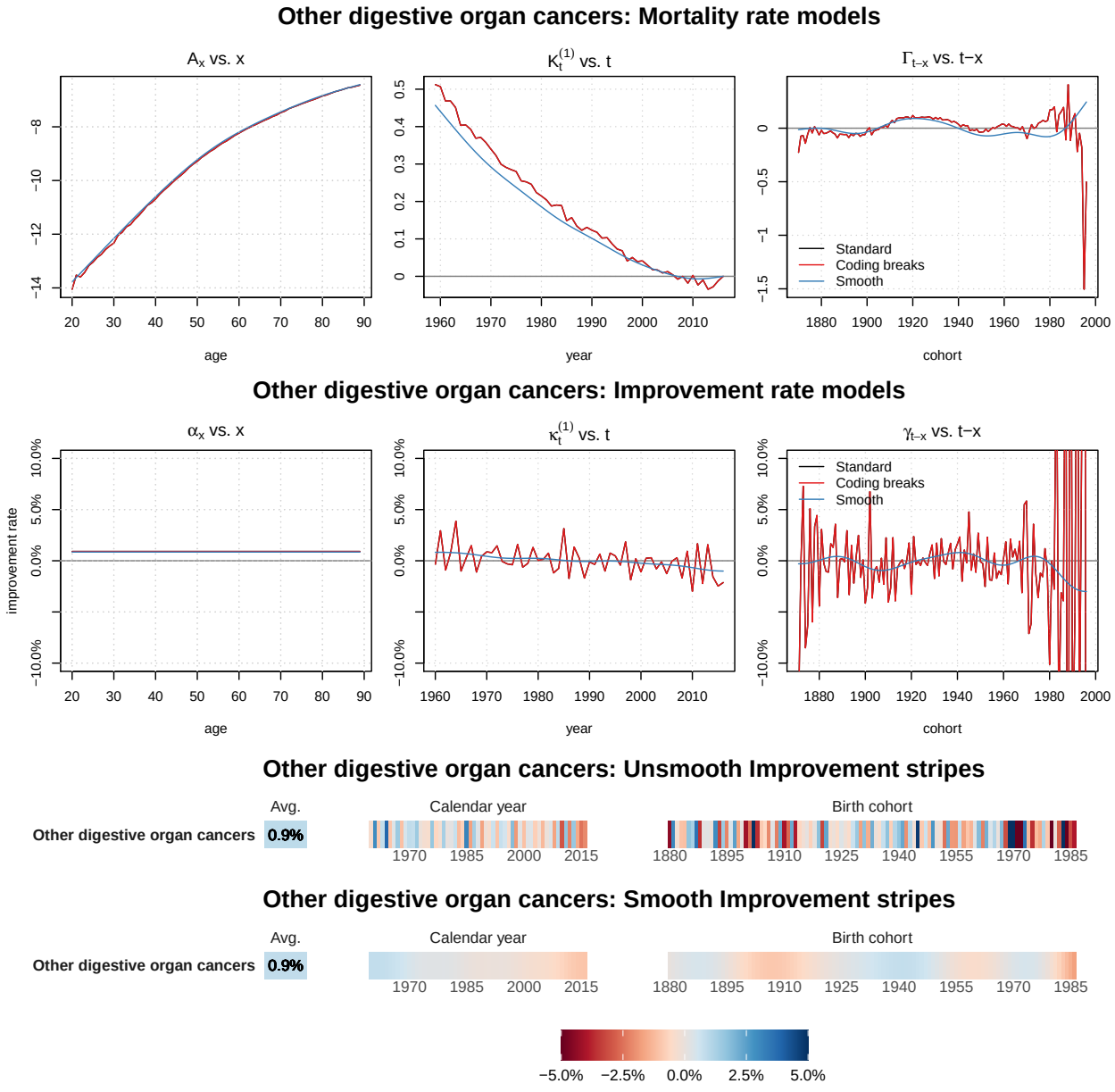


Figure F.13: Fitted parameters for the PCi model for respiratory diseases, females, 1959–2016, 20–89

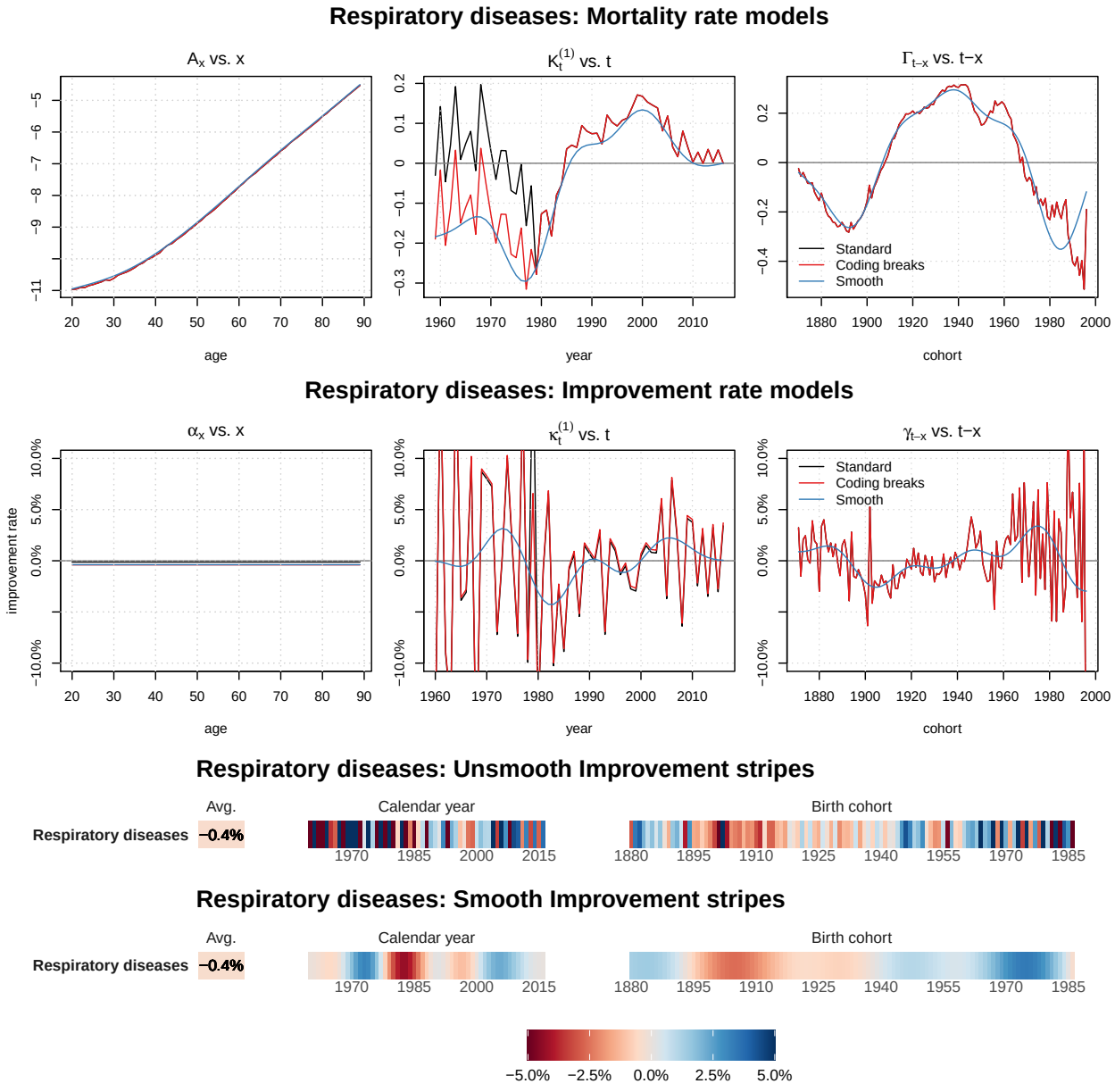


Figure F.14: Fitted parameters for the PCi model for influenza and pneumonia, females, 1959–2016, 20–89

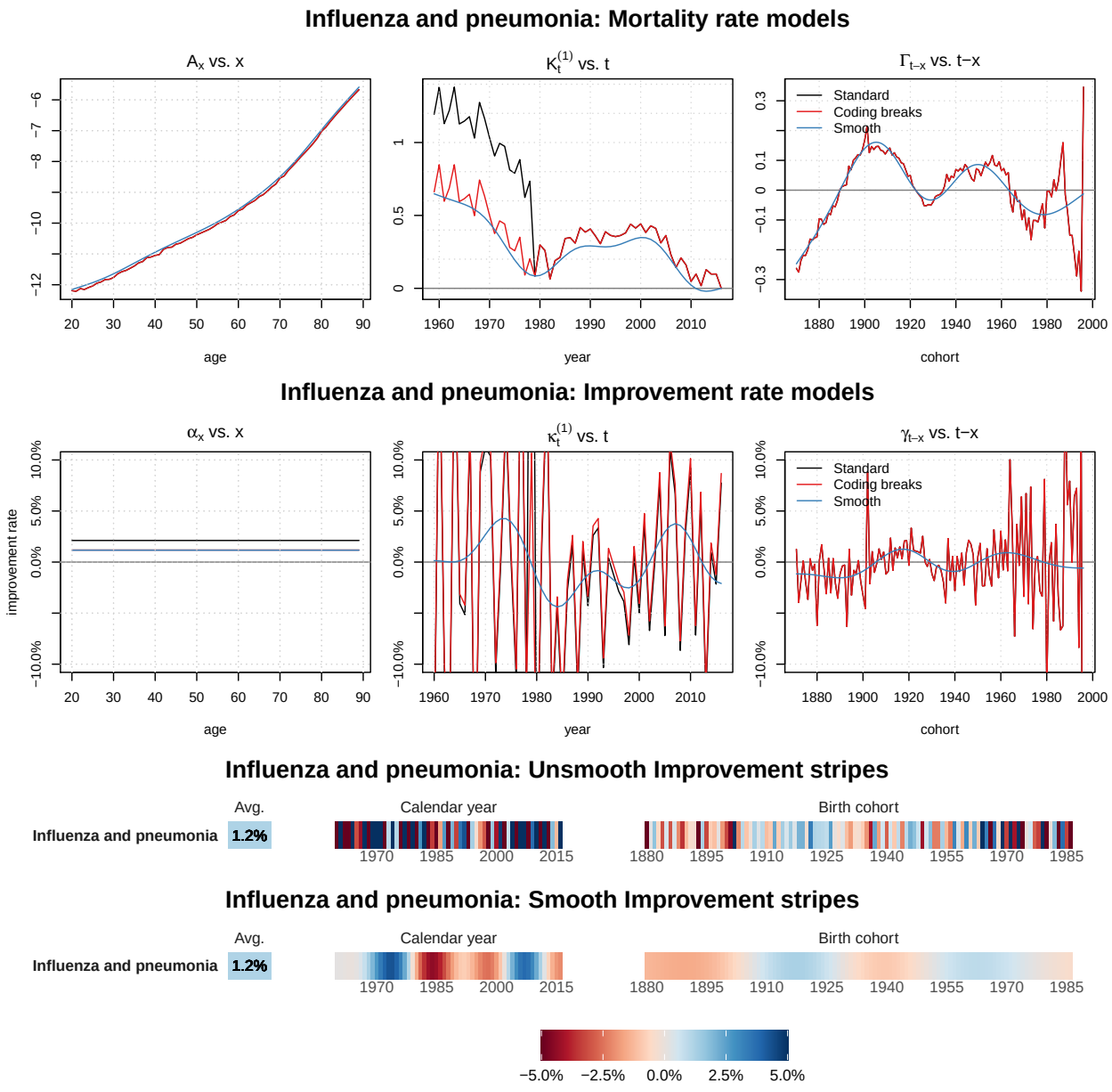


Figure F.15: Fitted parameters for the PCi model for chronic lower respiratory disease, females, 1959–2016, 20–89

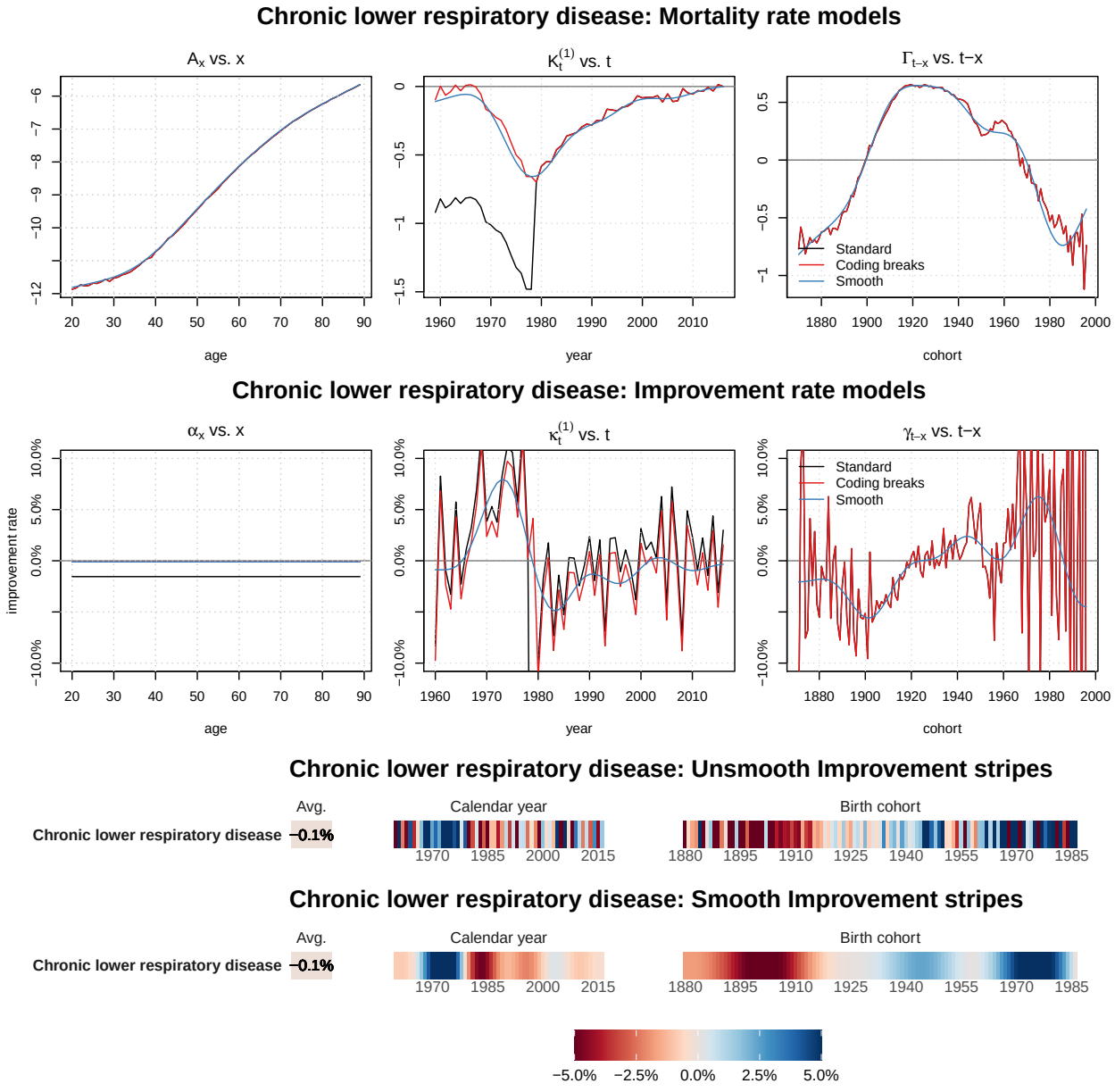


Figure F.16: Fitted parameters for the PCi model for other respiratory diseases, females, 1959–2016, 20–89

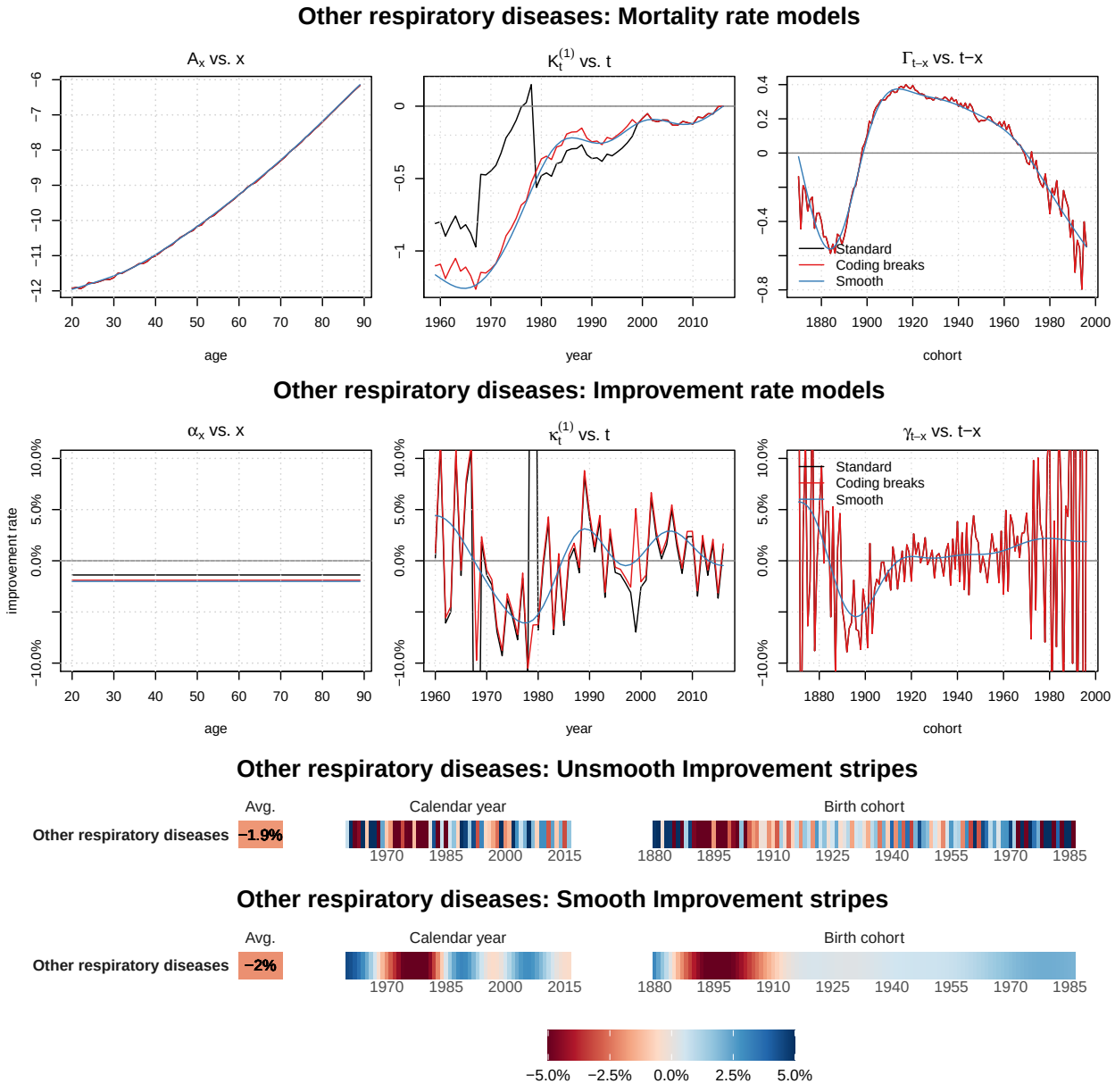


Figure F.17: Fitted parameters for the PCi model for digestive system, females, 1959–2016, 20–89

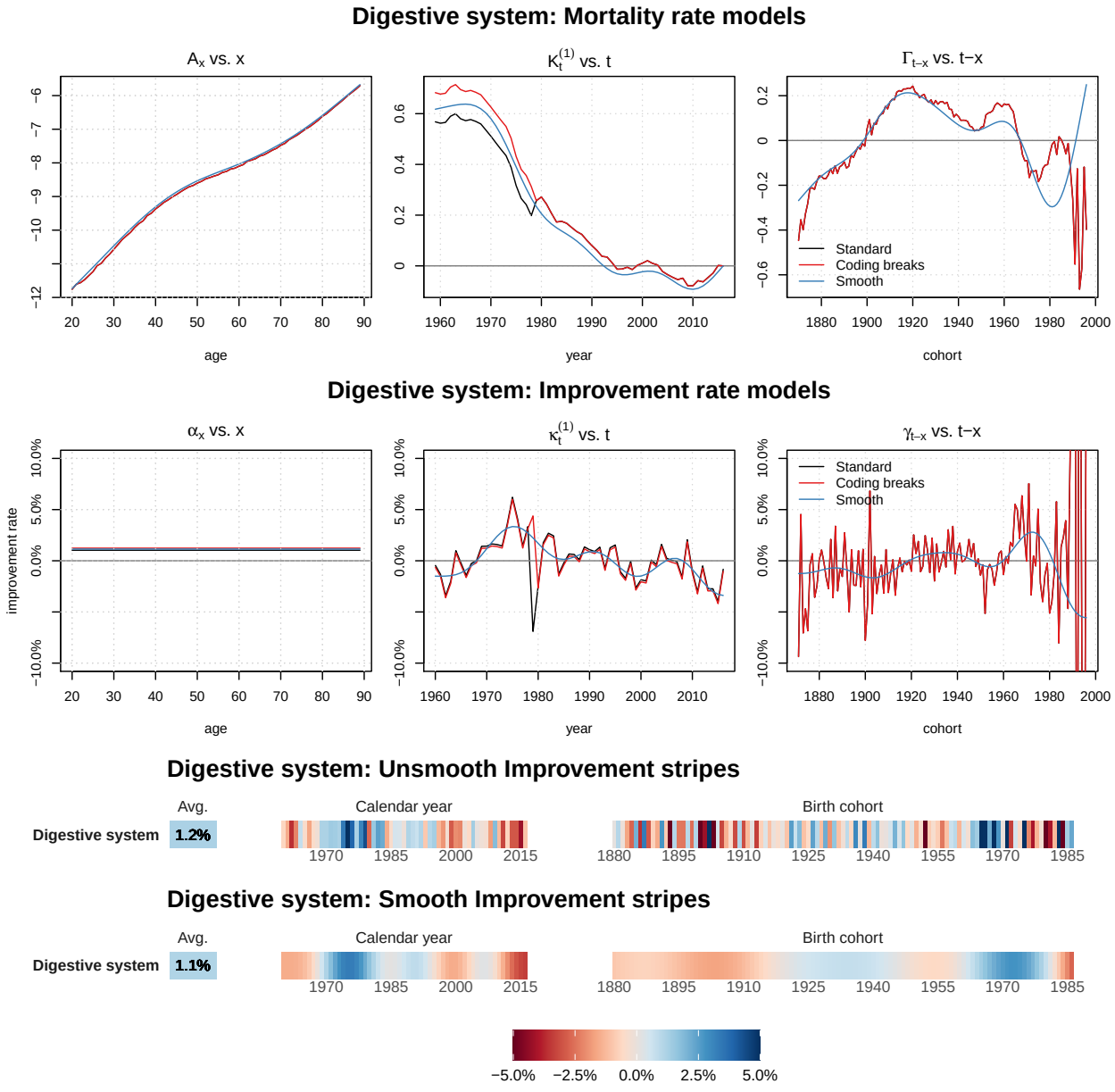


Figure F.18: Fitted parameters for the PCi model for gastric and duodenal ulcer, females, 1959–2016, 20–89

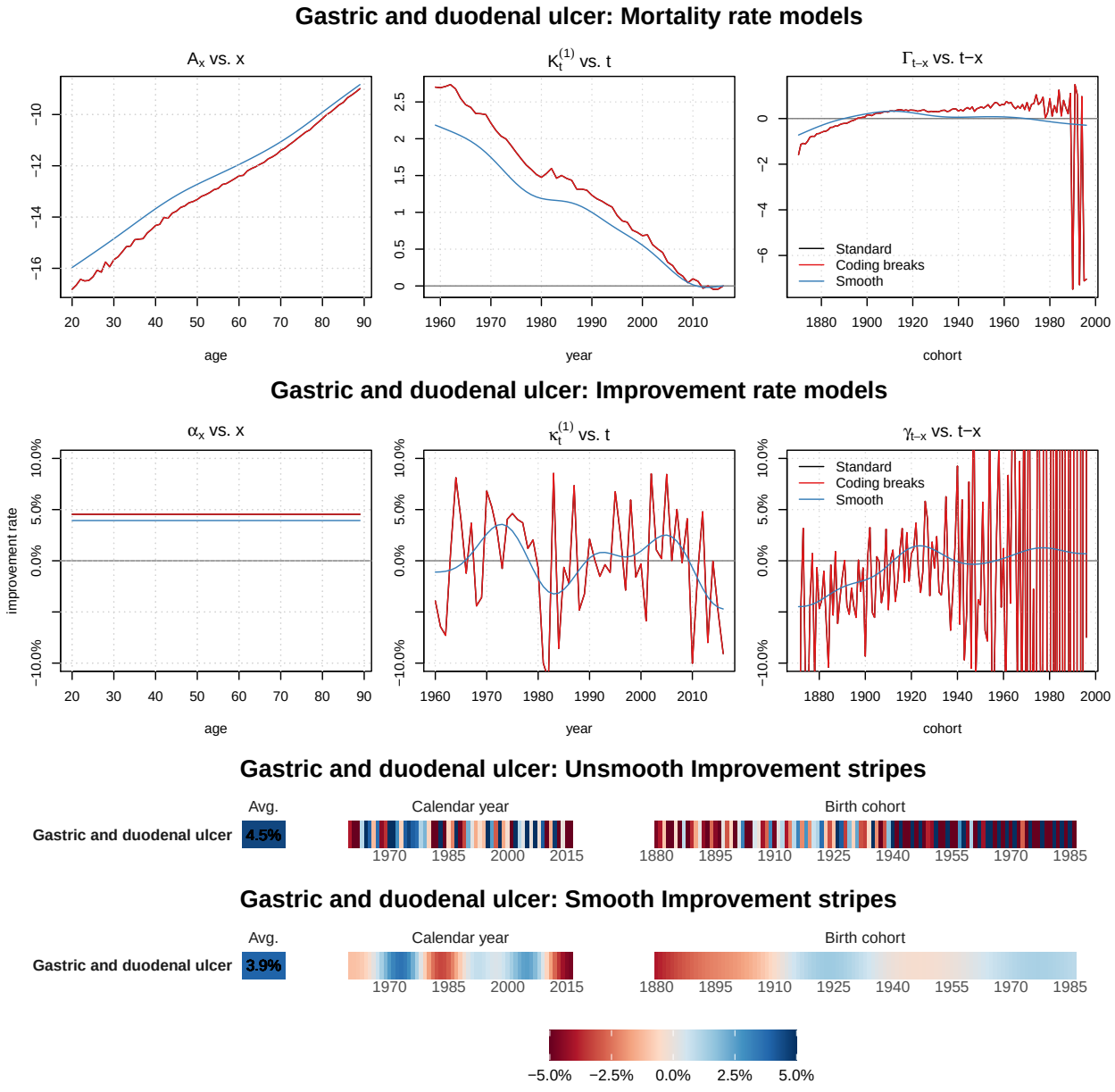


Figure F.19: Fitted parameters for the PCi model for chronic liver disease, females, 1959–2016, 20–89

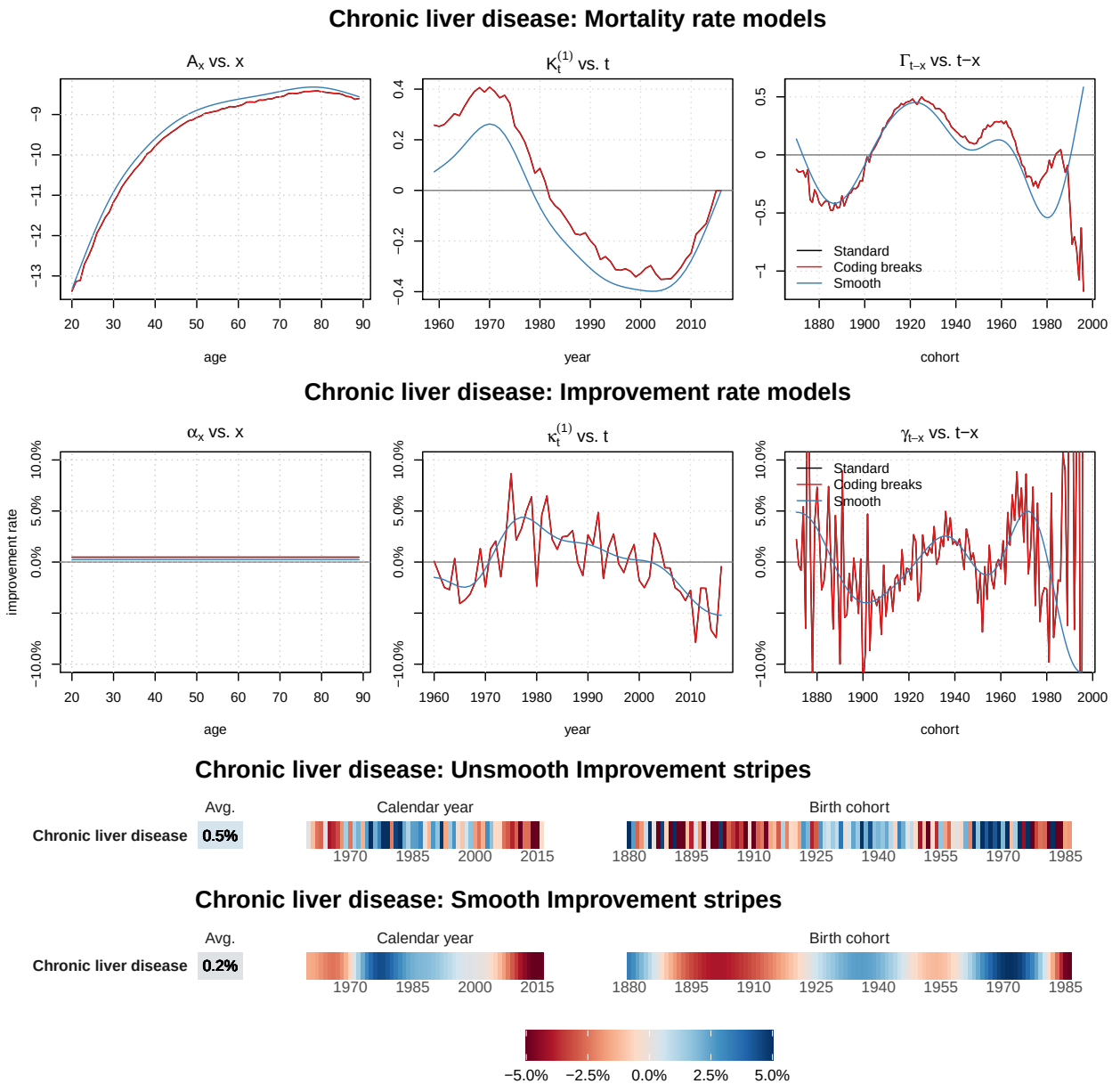


Figure F.20: Fitted parameters for the PCi model for other digestive system diseases, females, 1959–2016, 20–89

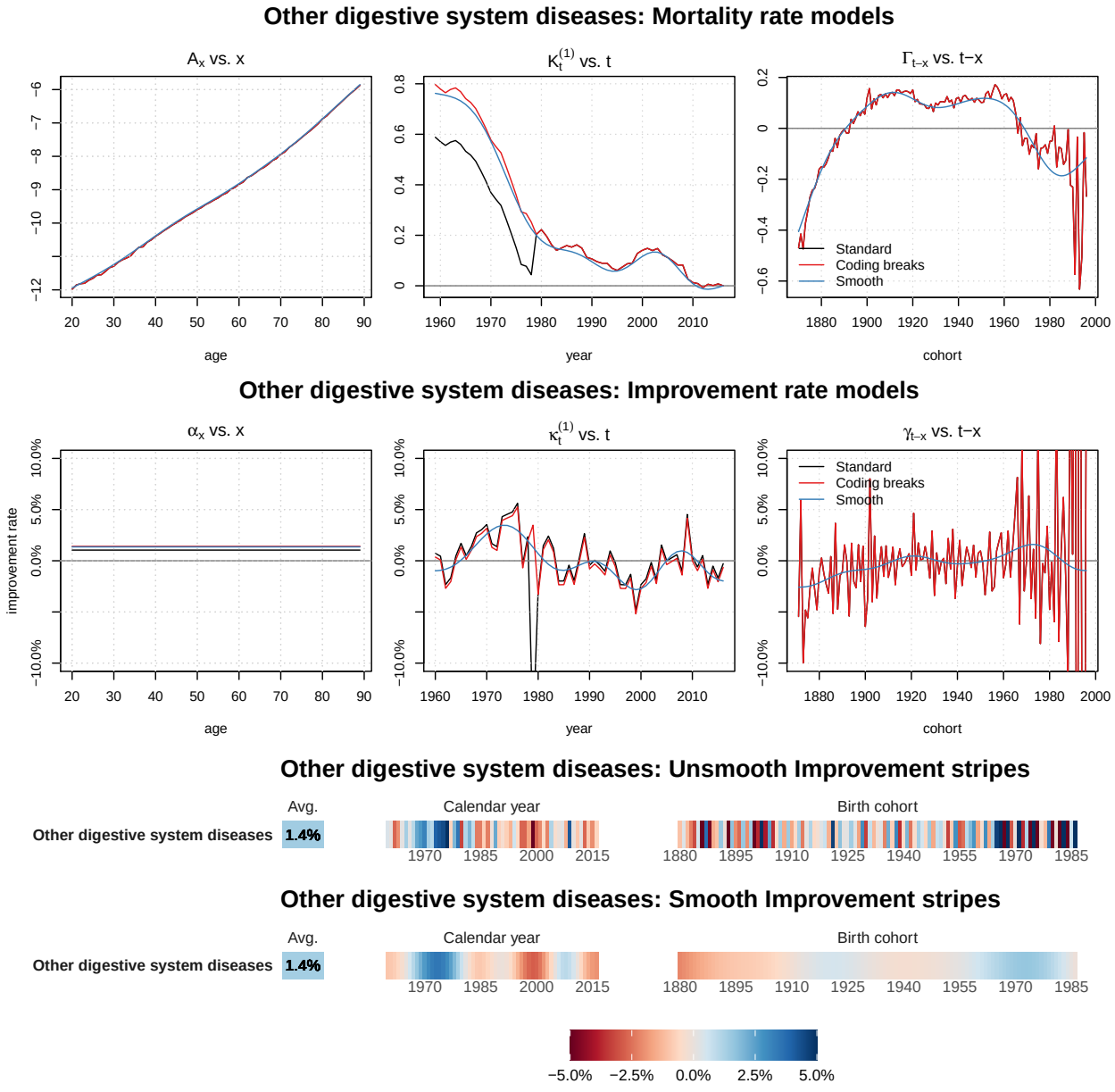


Figure F.21: Fitted parameters for the PCi model for external causes, females, 1959–2016, 20–89

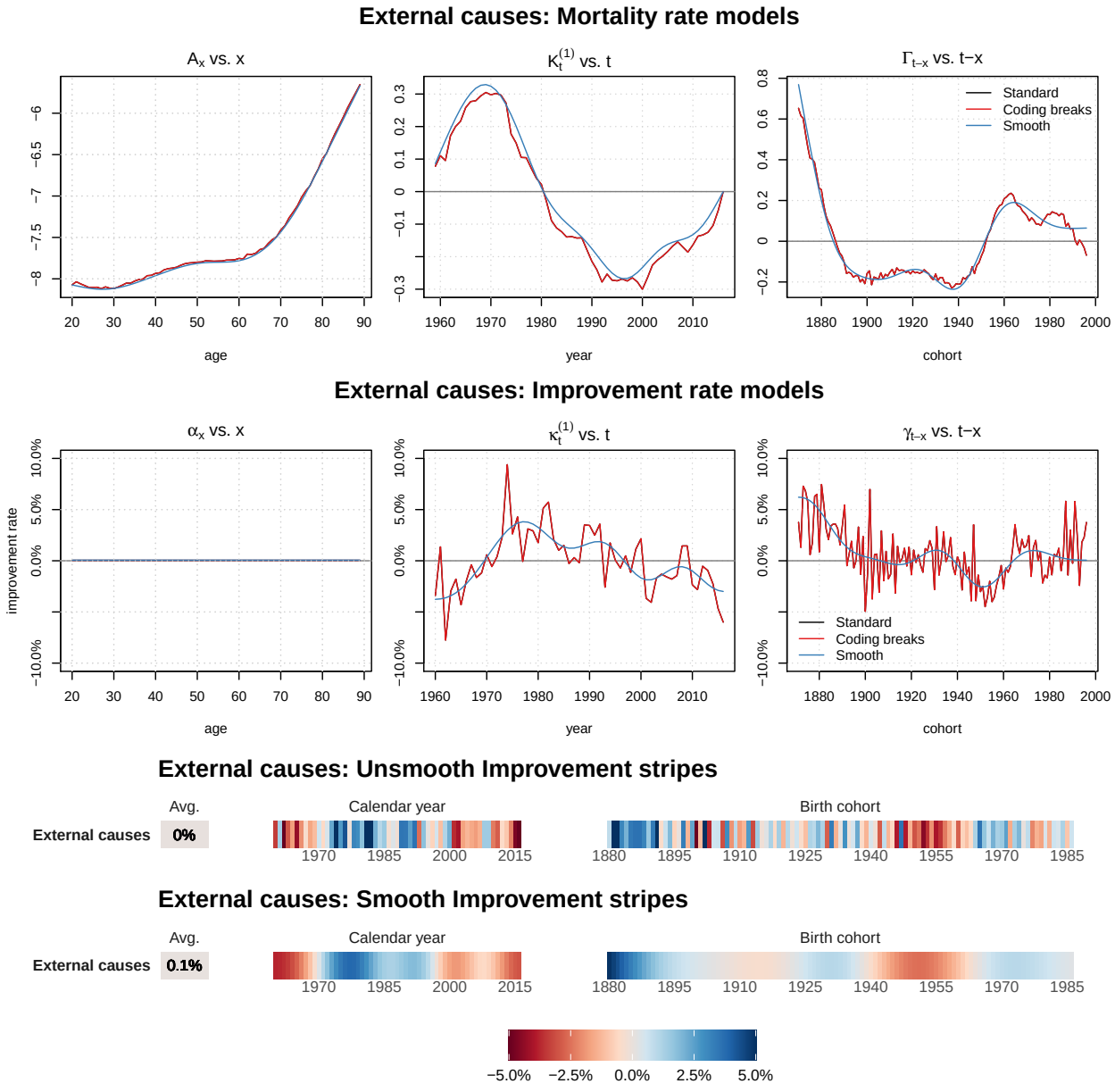


Figure F.22: Fitted parameters for the PCi model for traffic accidents, females, 1959–2016, 20–89

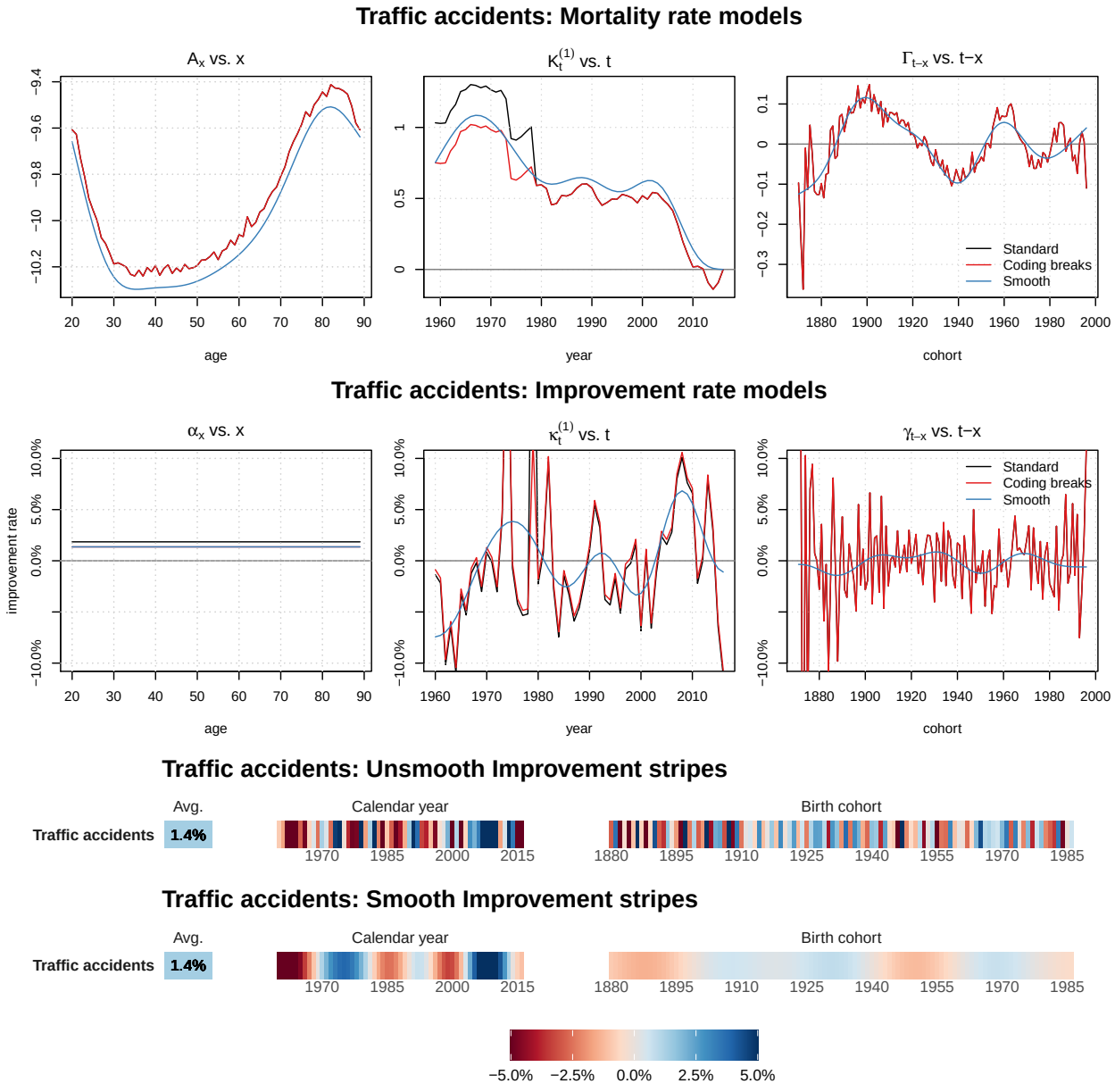


Figure F.23: Fitted parameters for the PCi model for self-harm and interpersonal violence, females, 1959–2016, 20–89

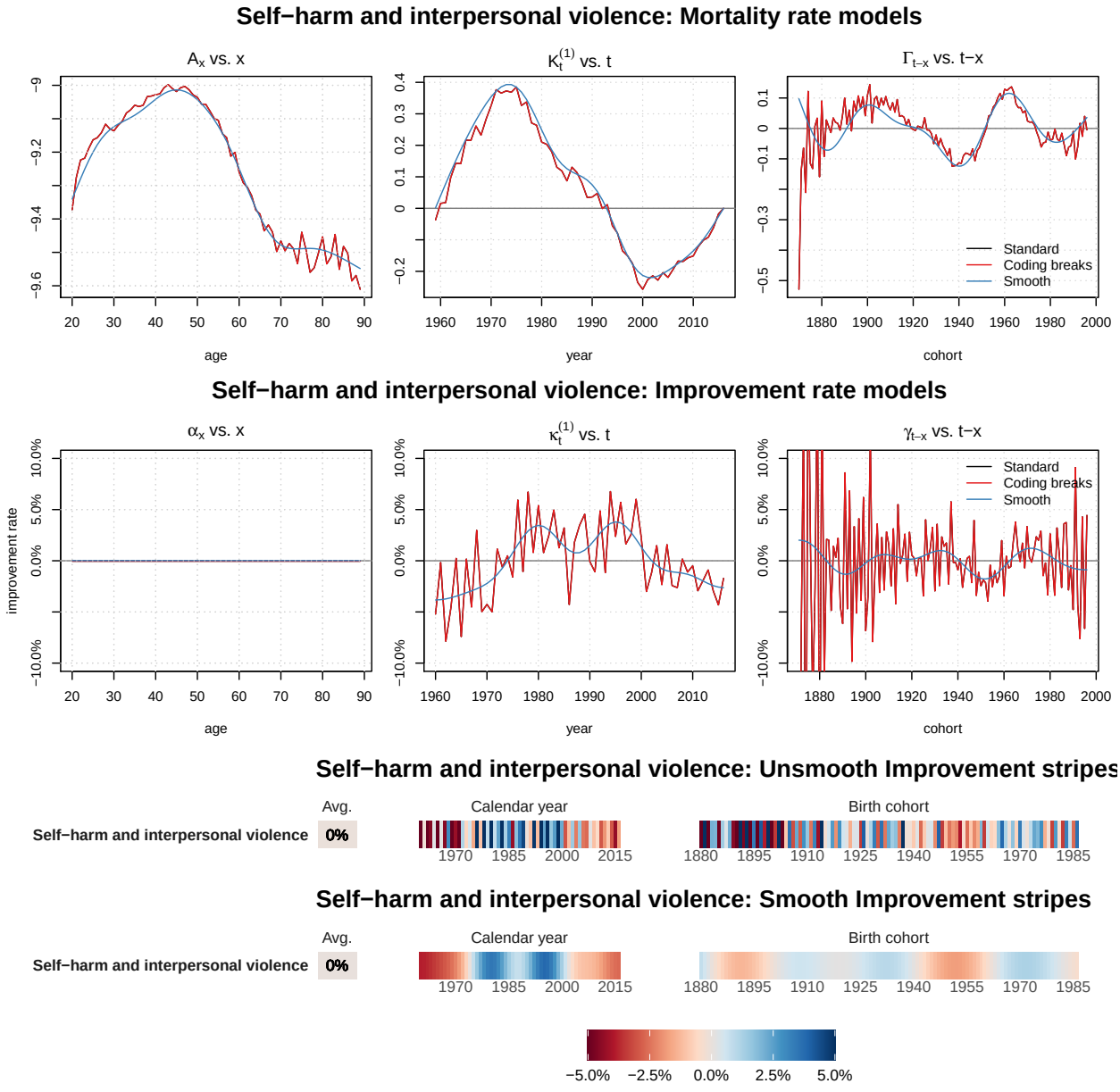


Figure F.24: Fitted parameters for the PCi model for other external causes, females, 1959–2016, 20–89

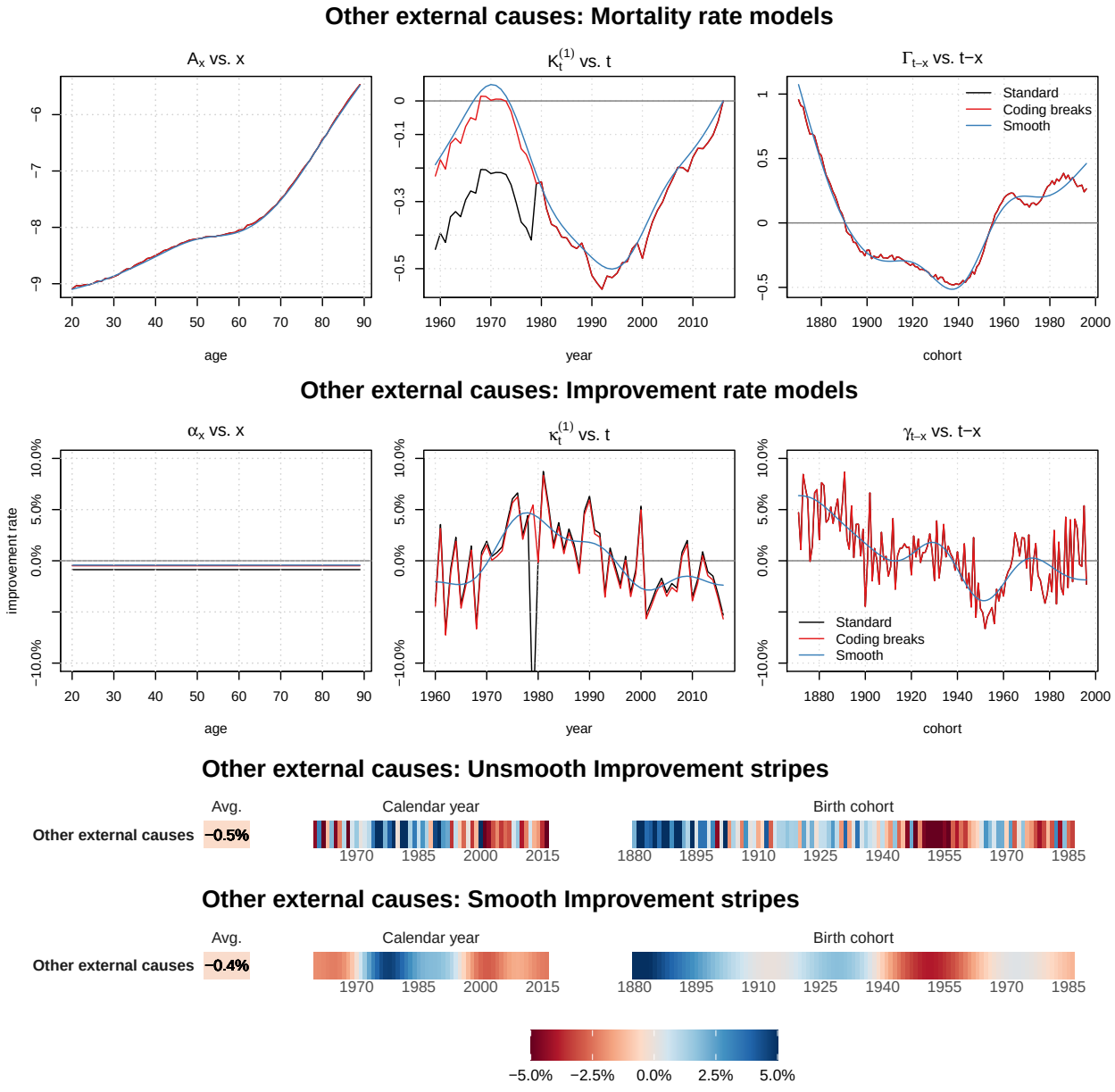


Figure F.25: Fitted parameters for the PCi model for Other causes, females, 1959–2016, 20–89

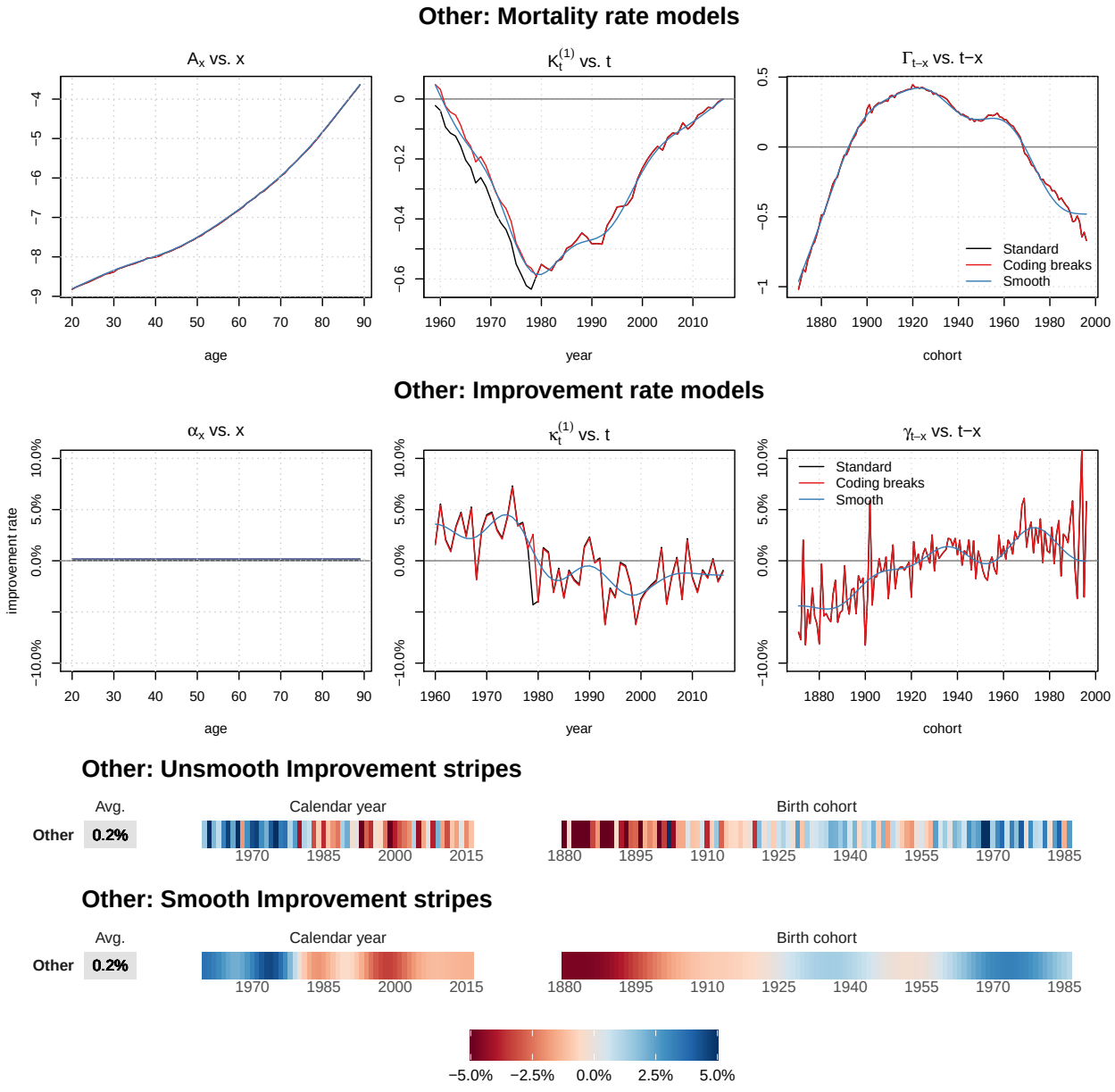


Figure F.26: Fitted parameters for the PCi model for AIDS and tuberculosis, females, 1959–2016, 20–89

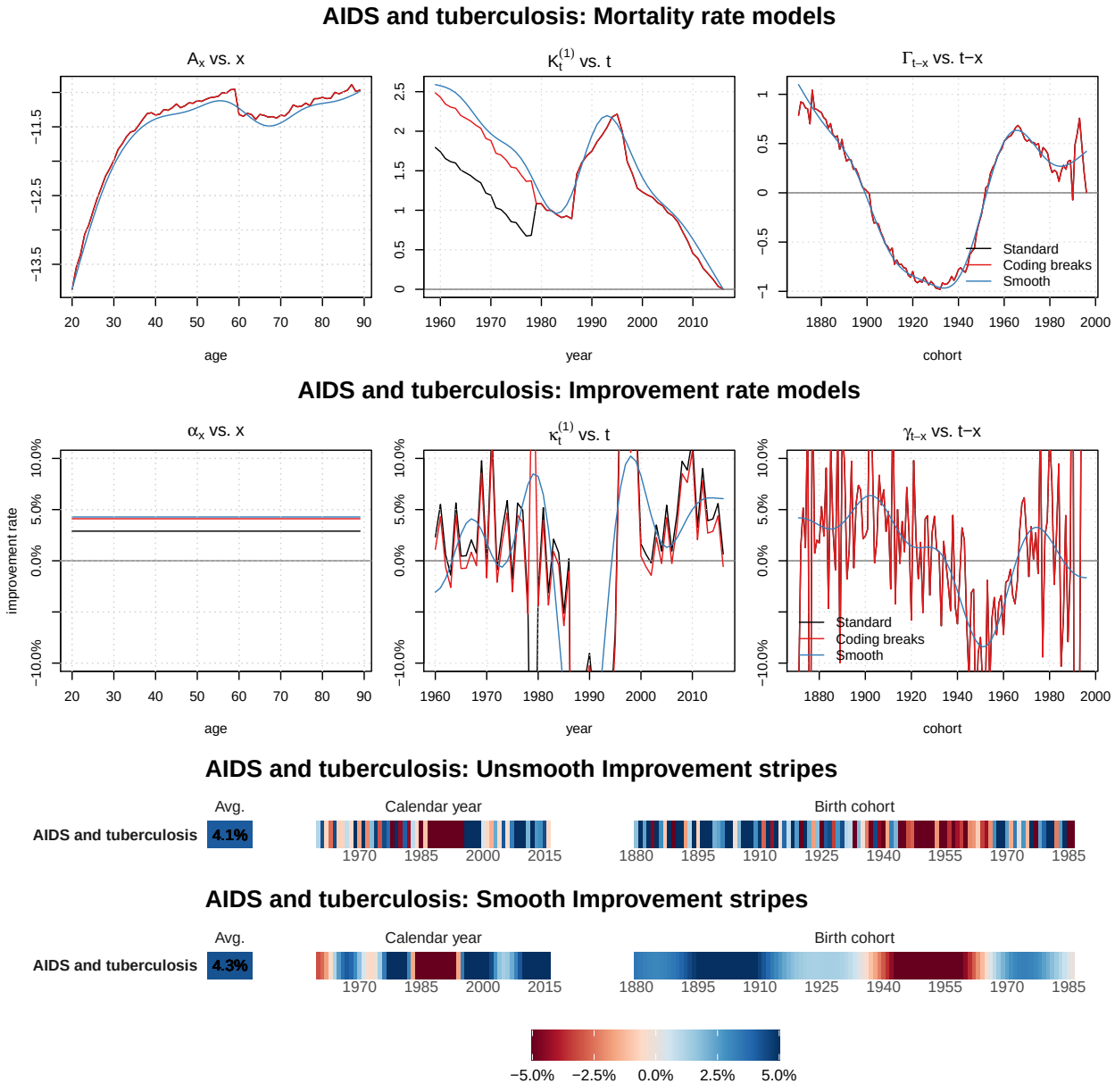


Figure F.27: Fitted parameters for the PCi model for diabetes and obesity, females, 1959–2016, 20–89

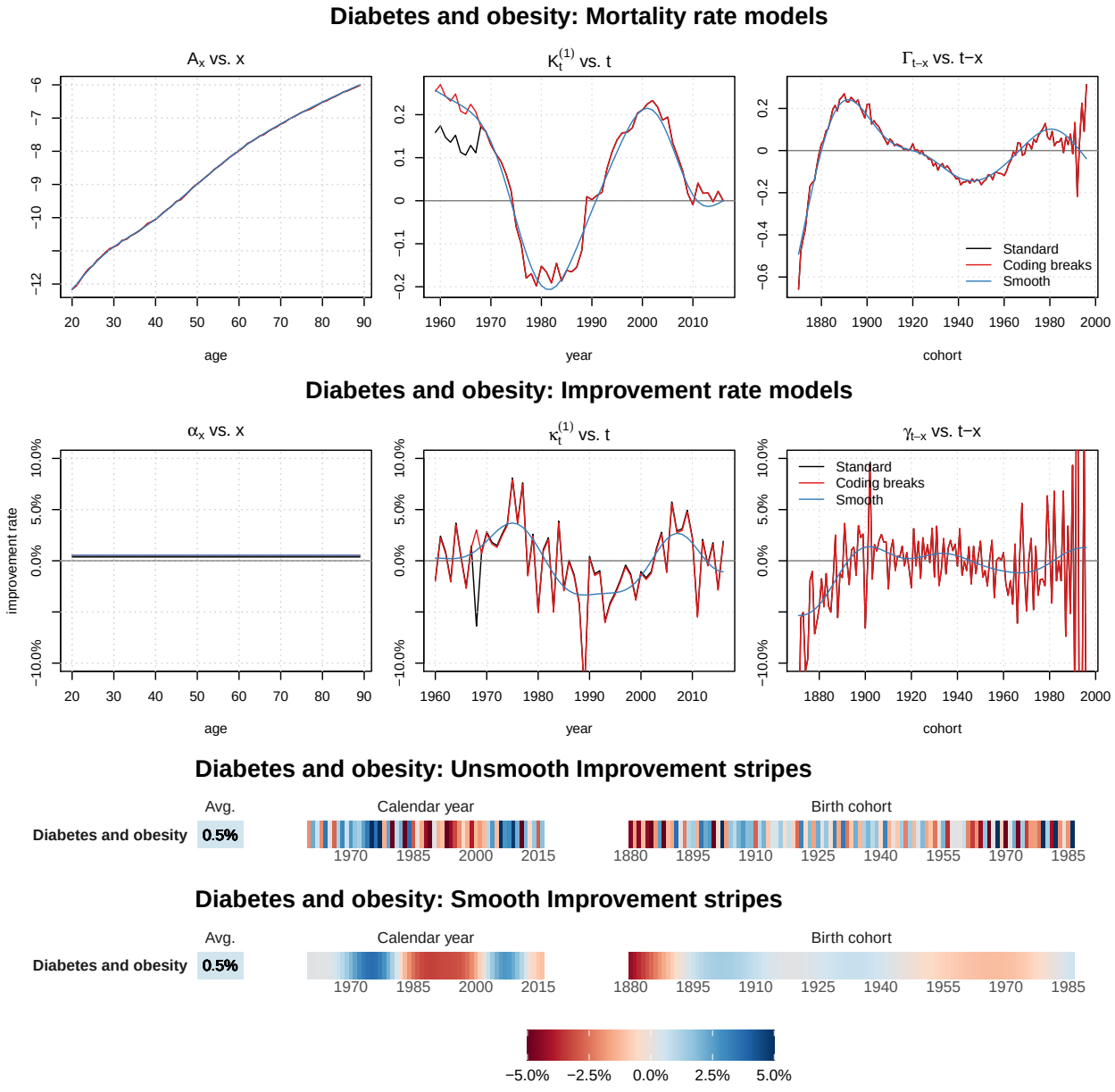


Figure F.28: Fitted parameters for the PCi model for alcohol abuse and drug dependence, females, 1959–2016, 20–89

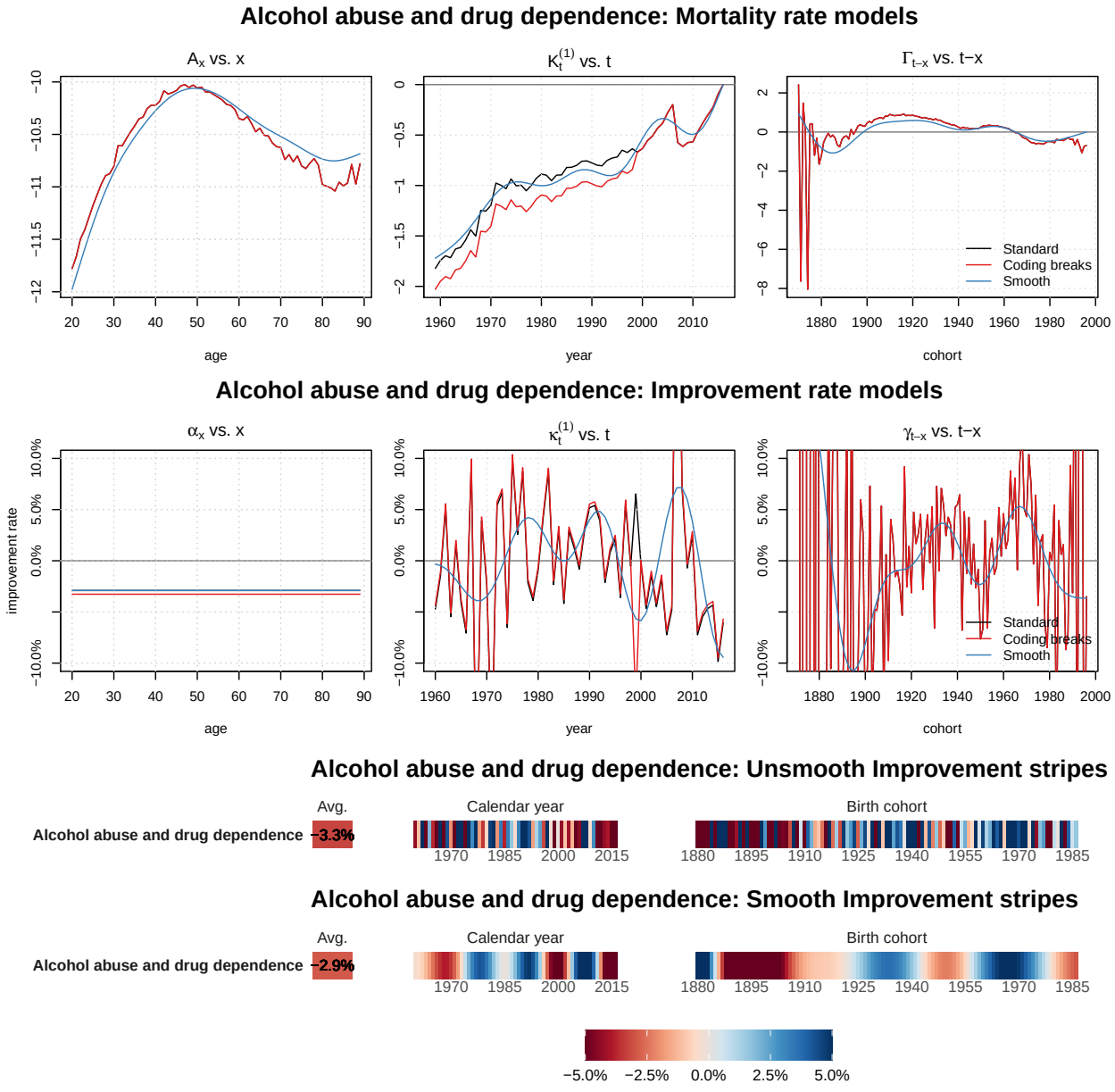


Figure F.29: Fitted parameters for the PCi model for Alzheimer's disease, females, 1979–2016, 20–89

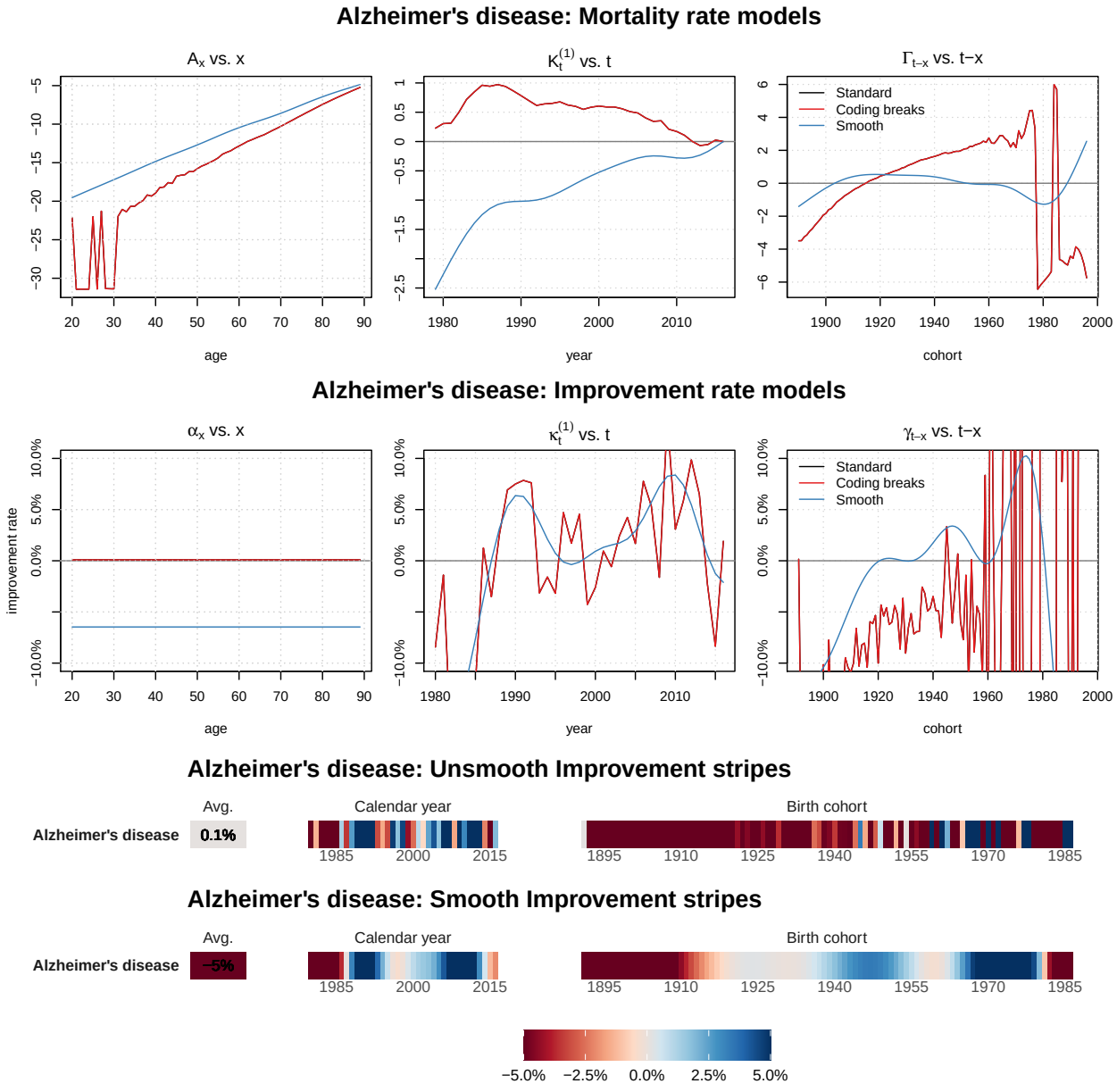


Figure F.30: Fitted parameters for the PCi model for dementia and other mental disorders, females, 1959–2016, 20–89

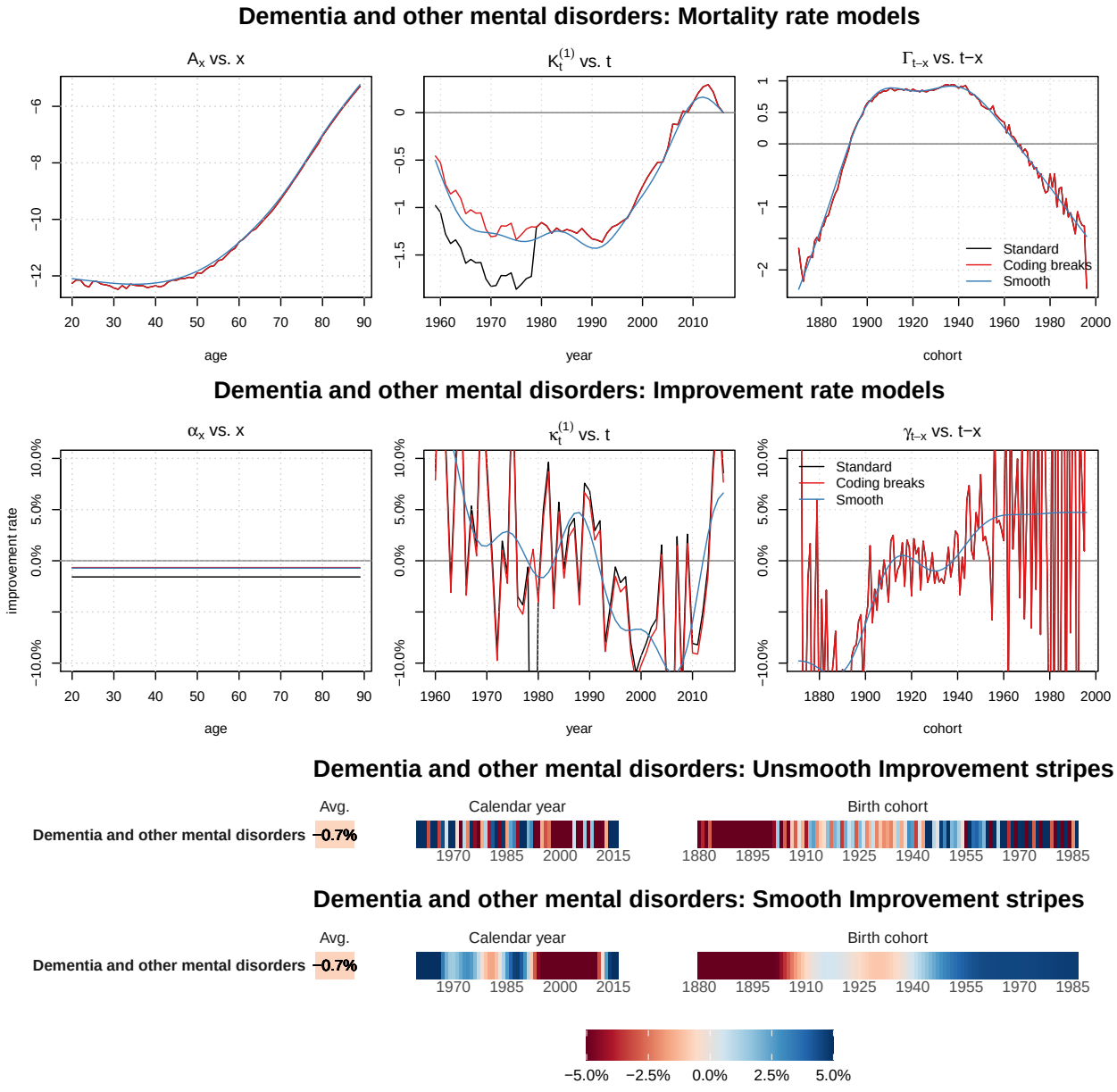


Figure F.31: Fitted parameters for the PCi model for rest of causes of death, females, 1959–2016, 20–89

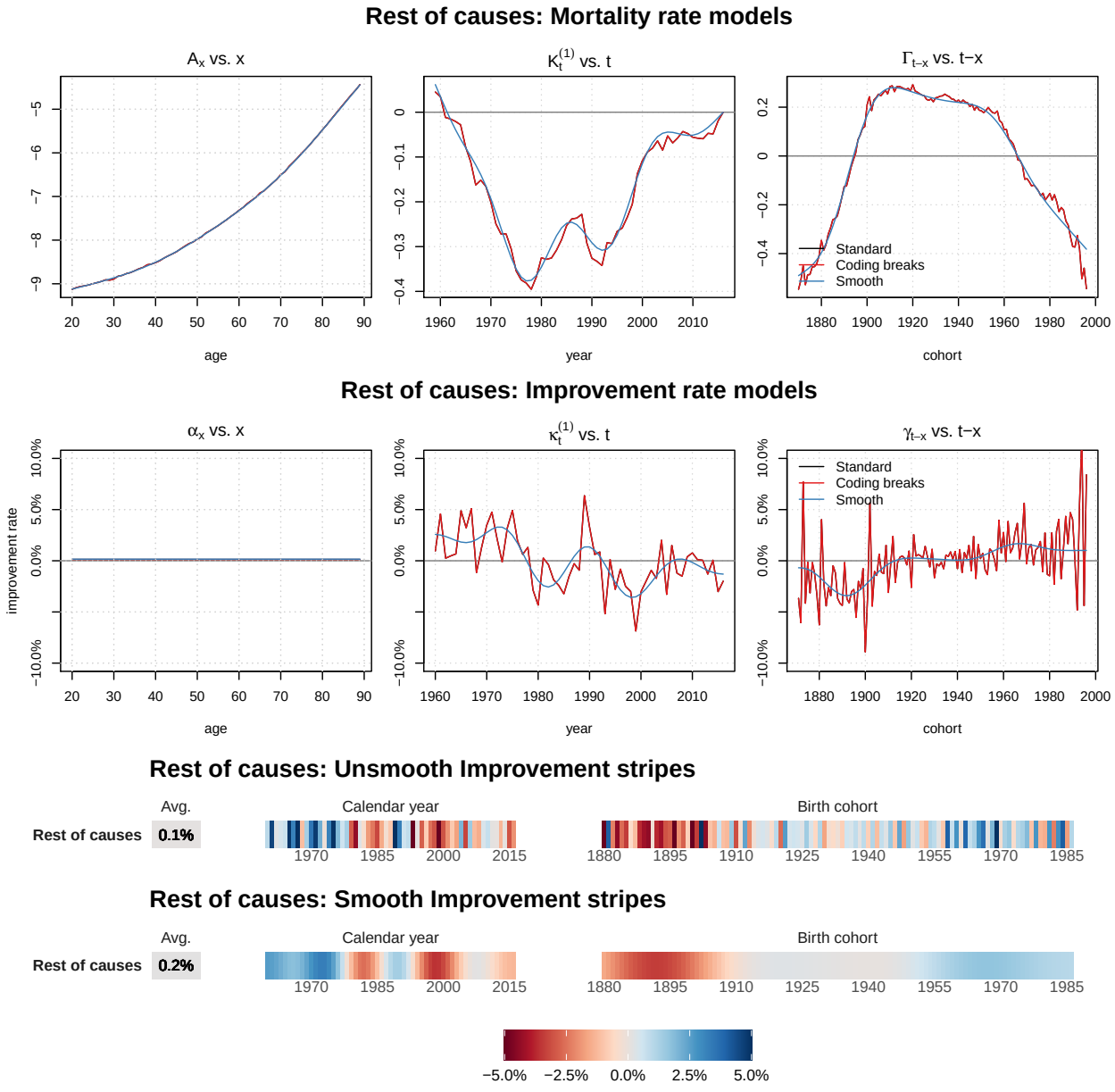


Figure F.32: Fitted parameters for the PCi model for AIDS and tuberculosis, females, 1959–2016, 20–89

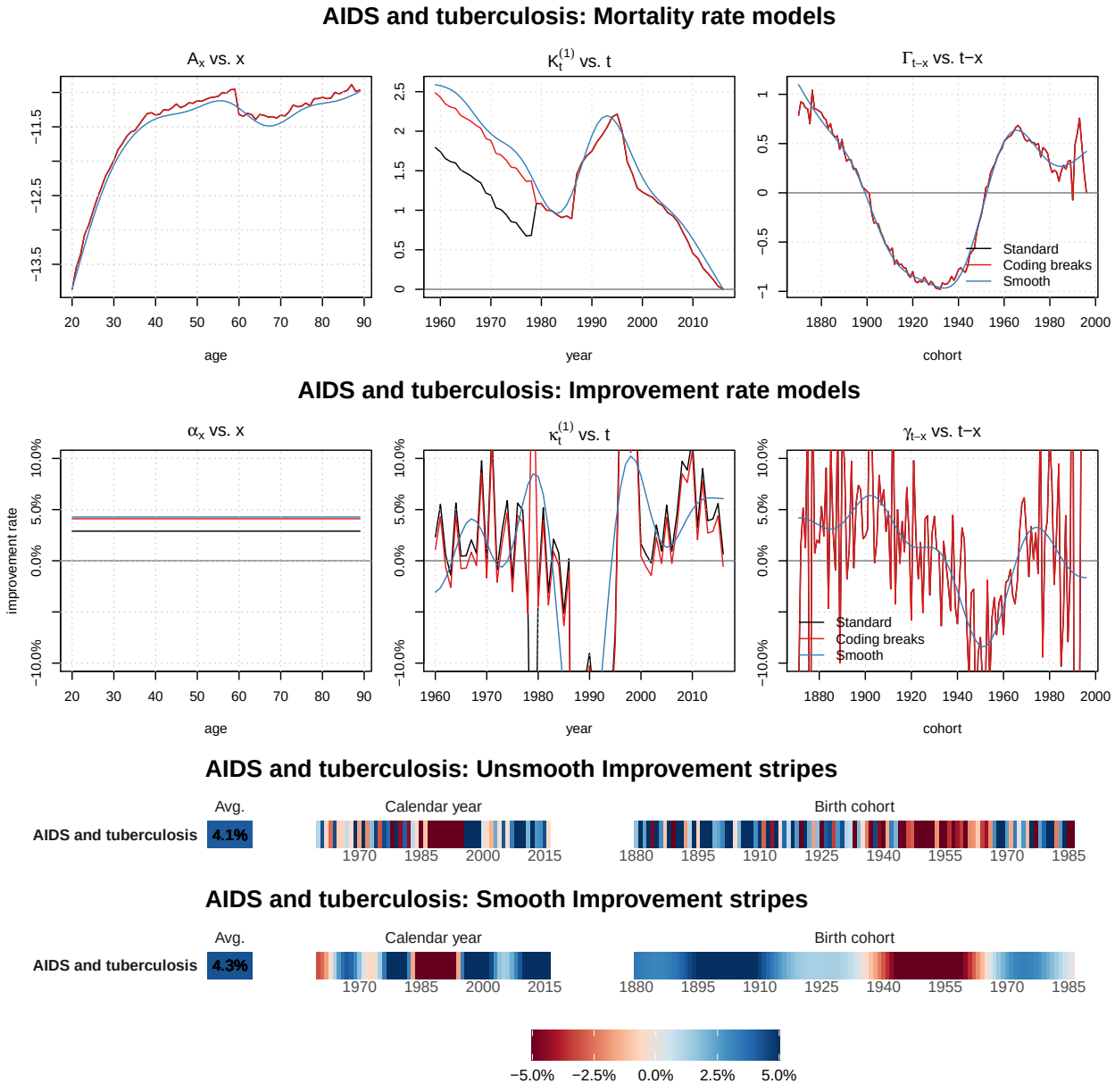


Figure F.33: Fitted parameters for the PCi model for alcohol abuse, females, 1959–2016, 20–89

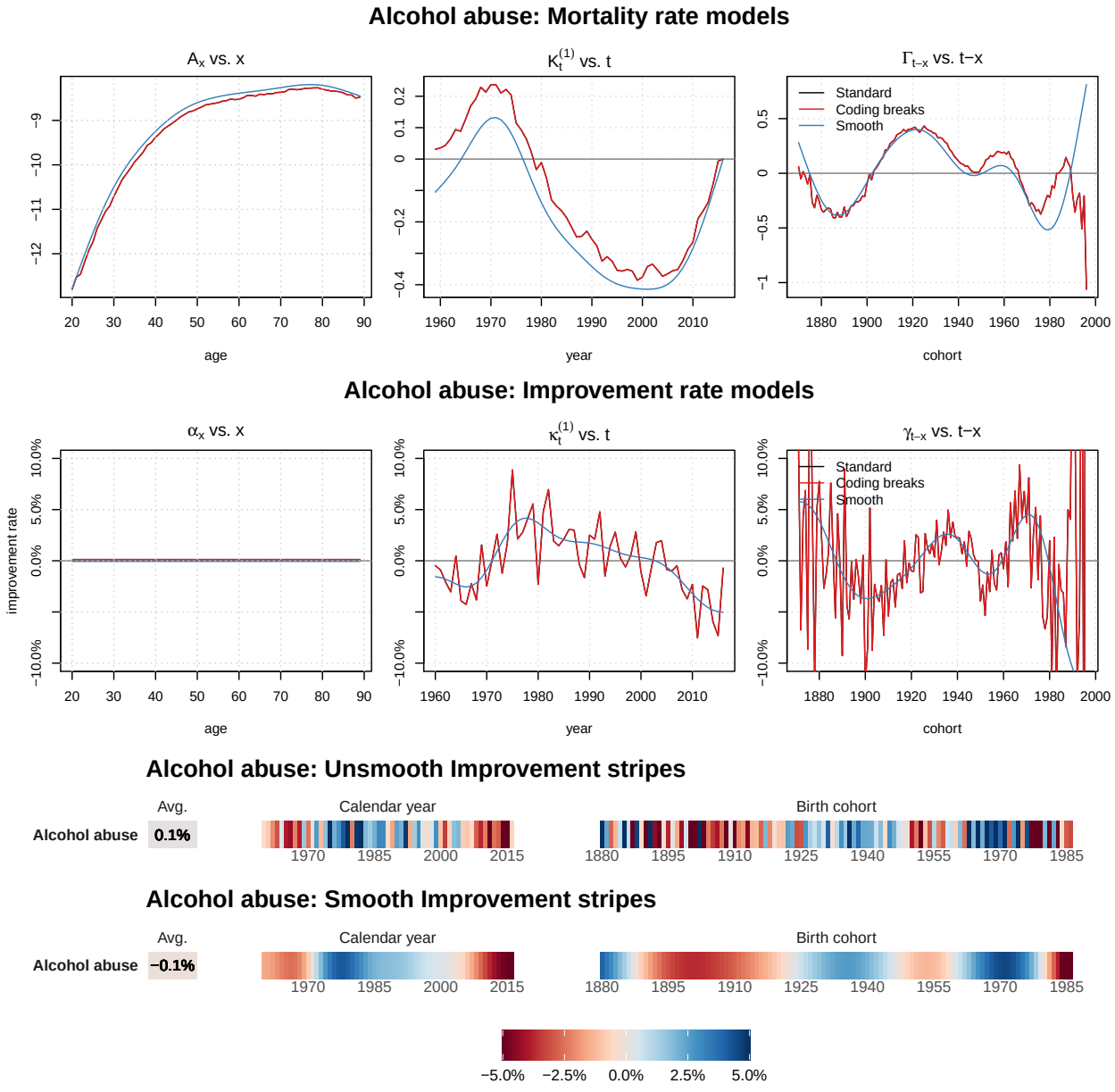


Figure F.34: Fitted parameters for the PCi model for dementia and Alzheimer's disease, females, 1959–2016, 20–89

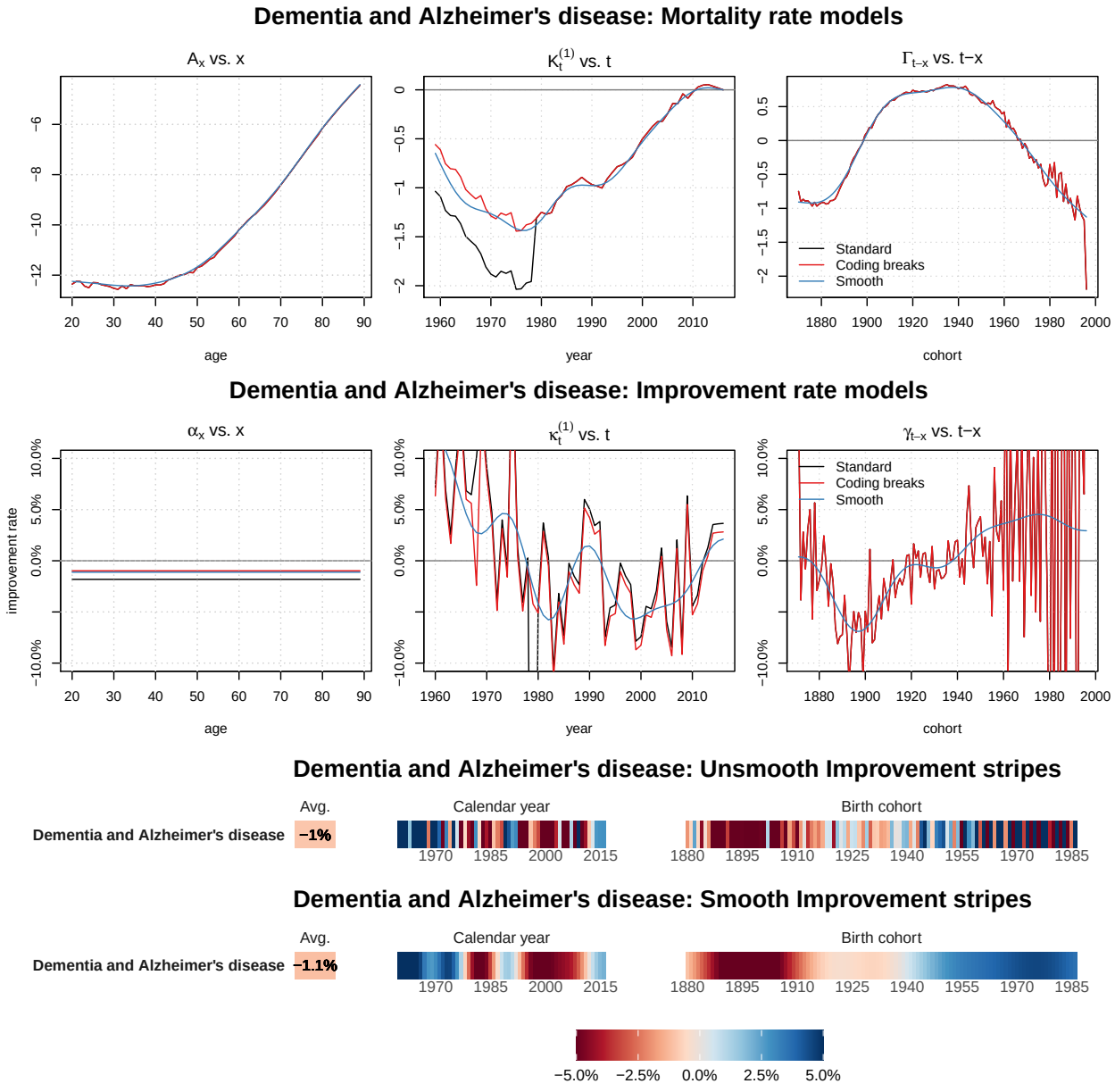


Figure F.35: Fitted parameters for the PCi model for diabetes and obesity, females, 1959–2016, 20–89

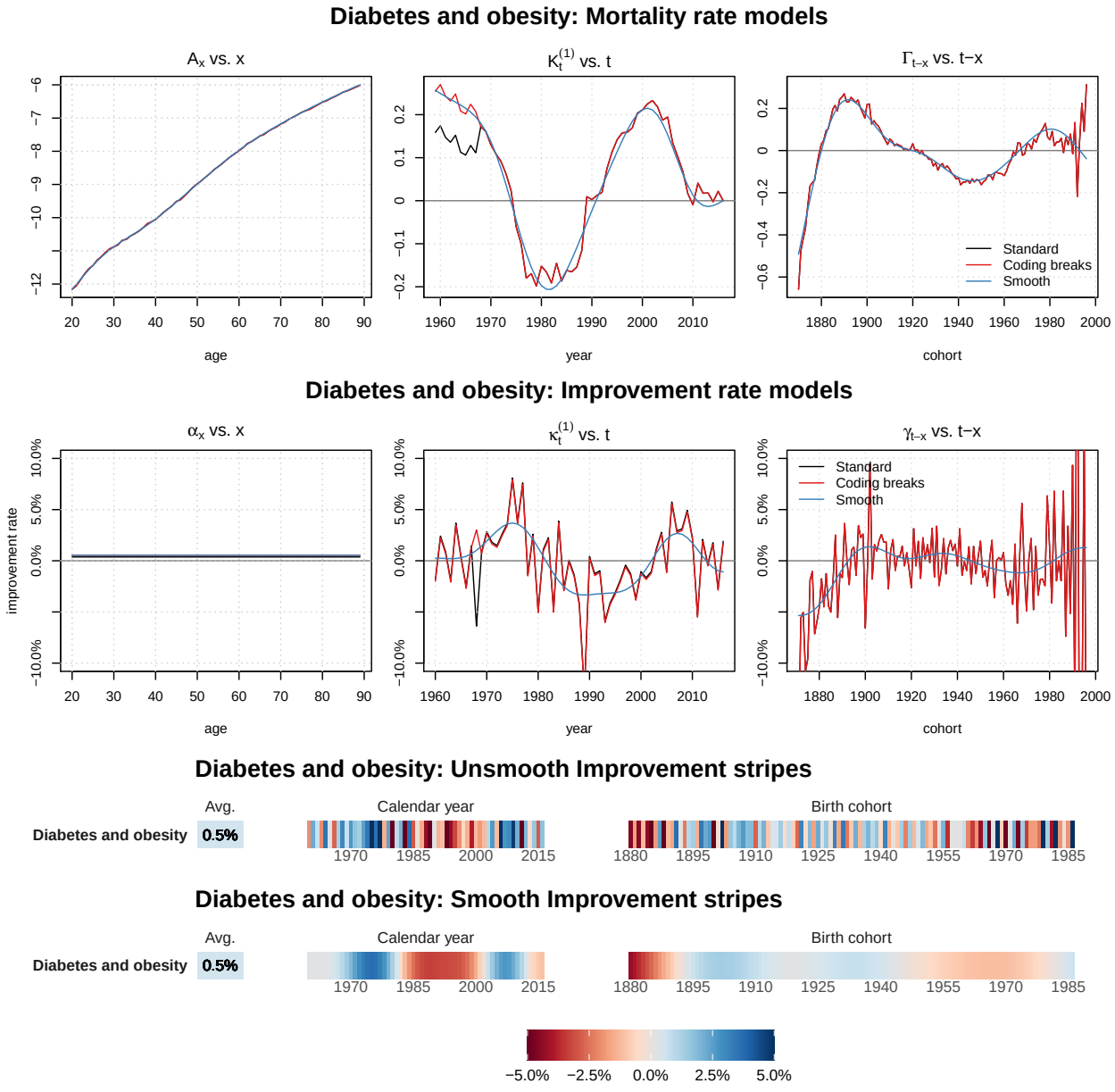


Figure F.36: Fitted parameters for the PCi model for drug dependency, females, 1959–2016, 20–89

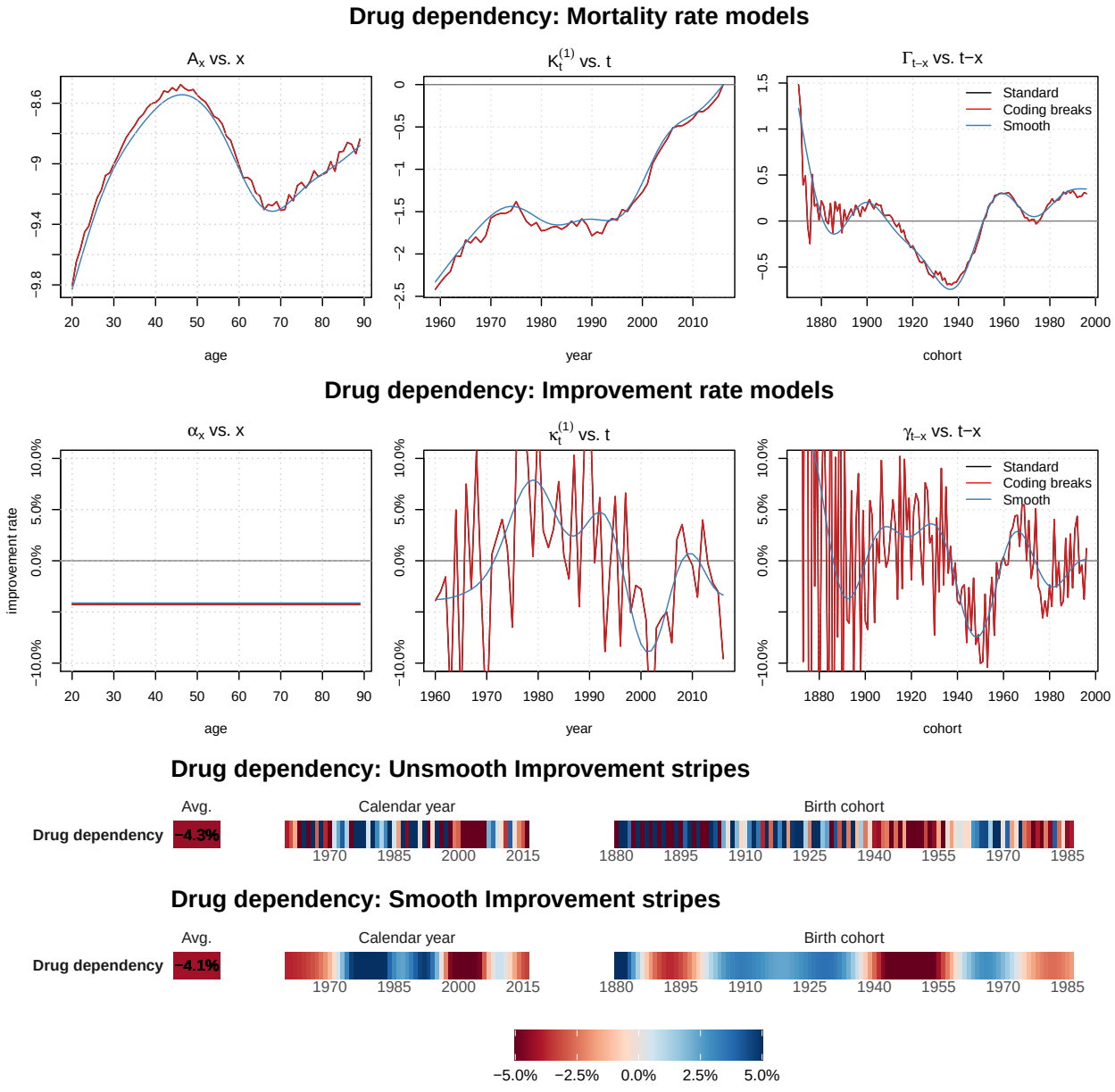


Figure F.37: Fitted parameters for the PCi model for homicide, females, 1959–2016, 20–89

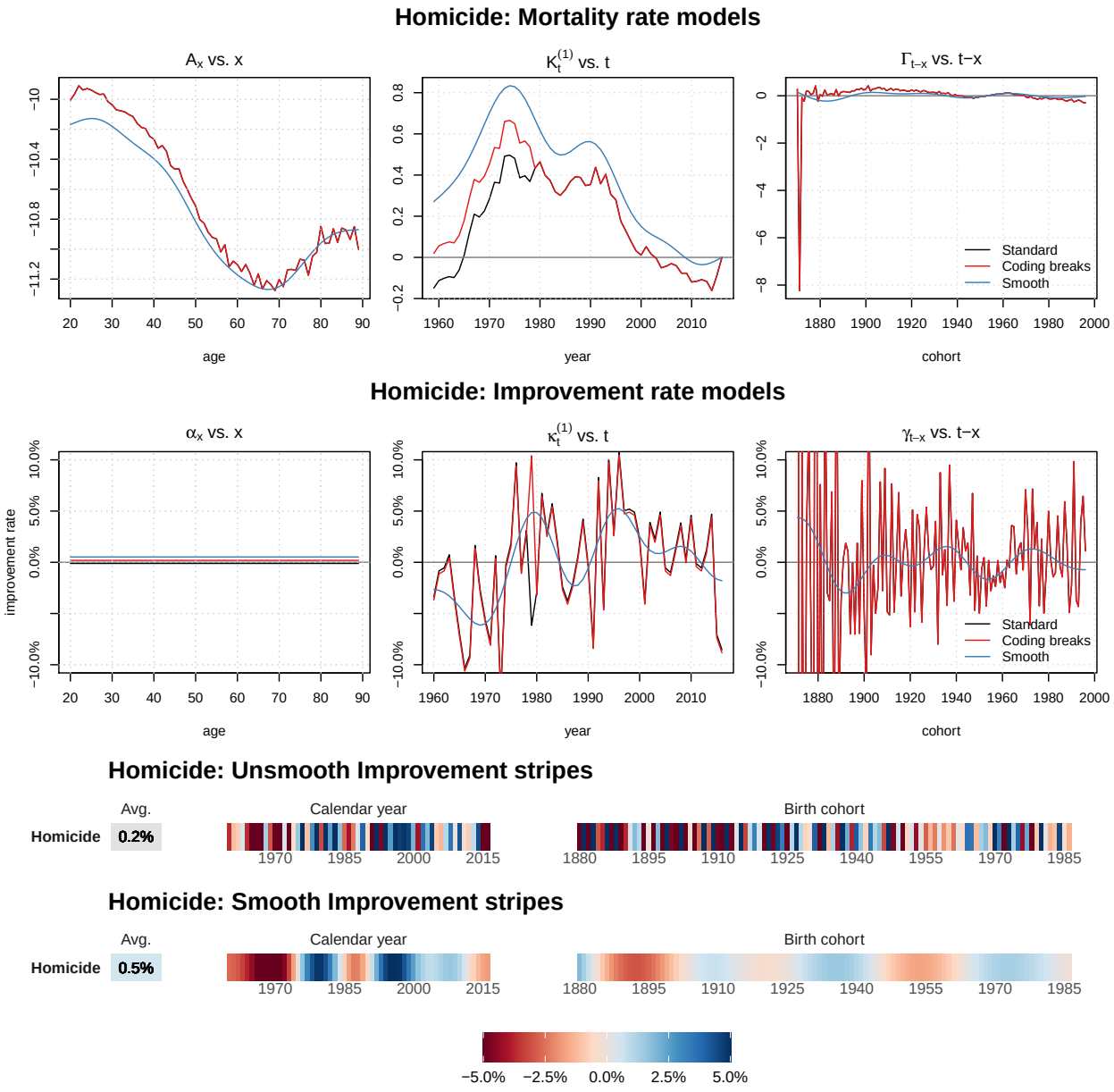


Figure F.38: Fitted parameters for the PCi model for hypertensive disease, females, 1959–2016, 20–89

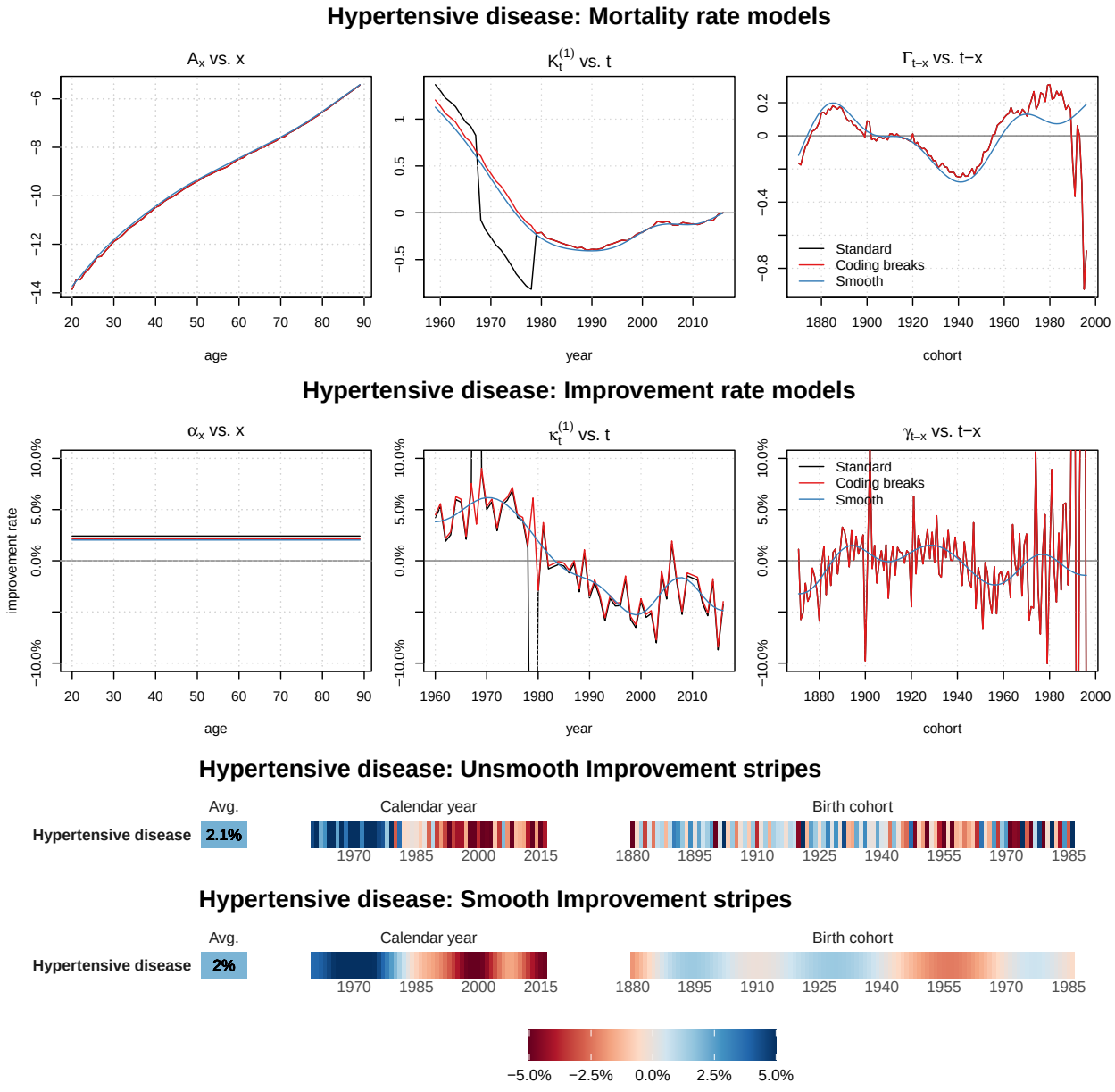


Figure F.39: Fitted parameters for the PCi model for self-harm, females, 1959–2016, 20–89

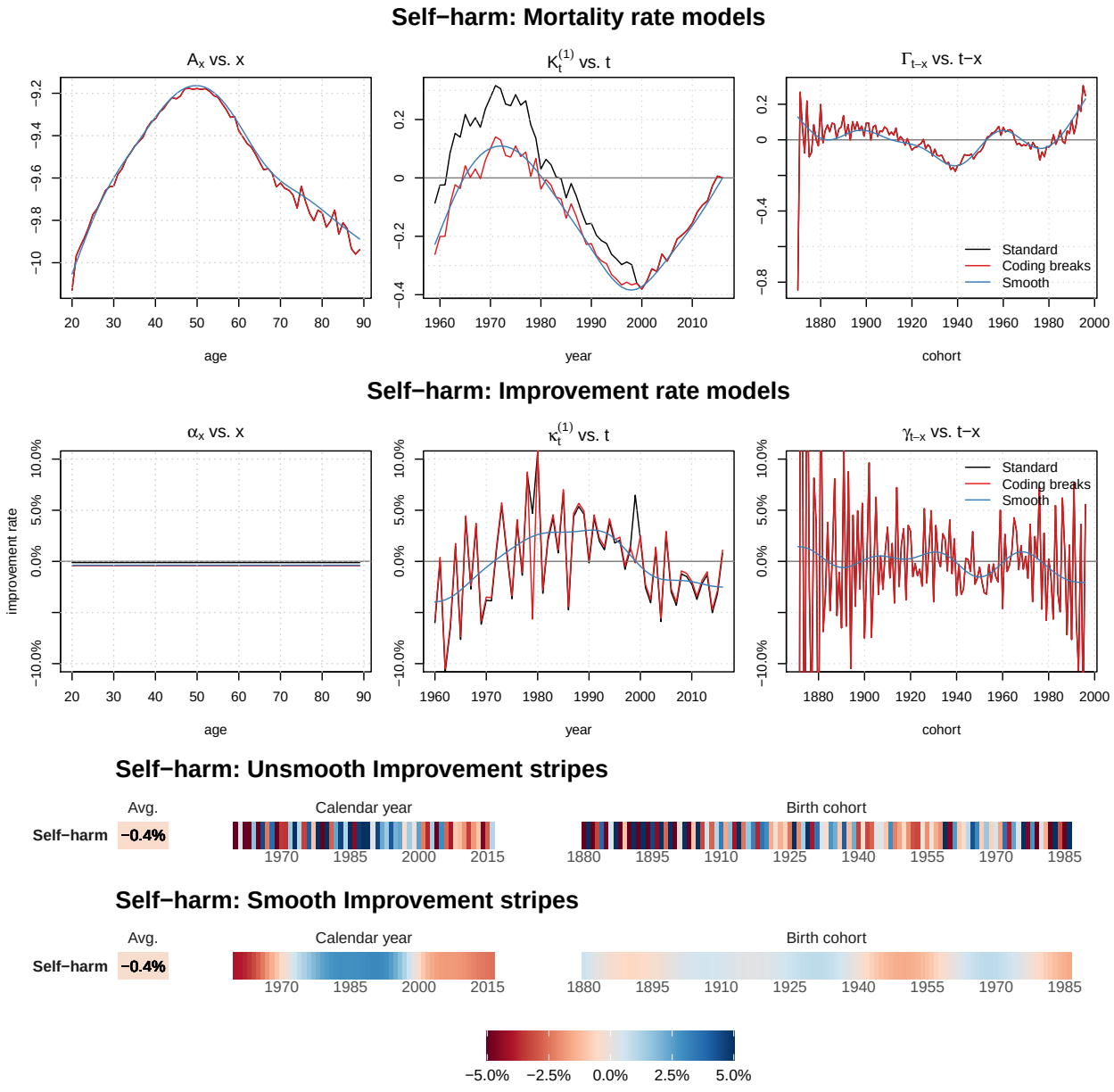
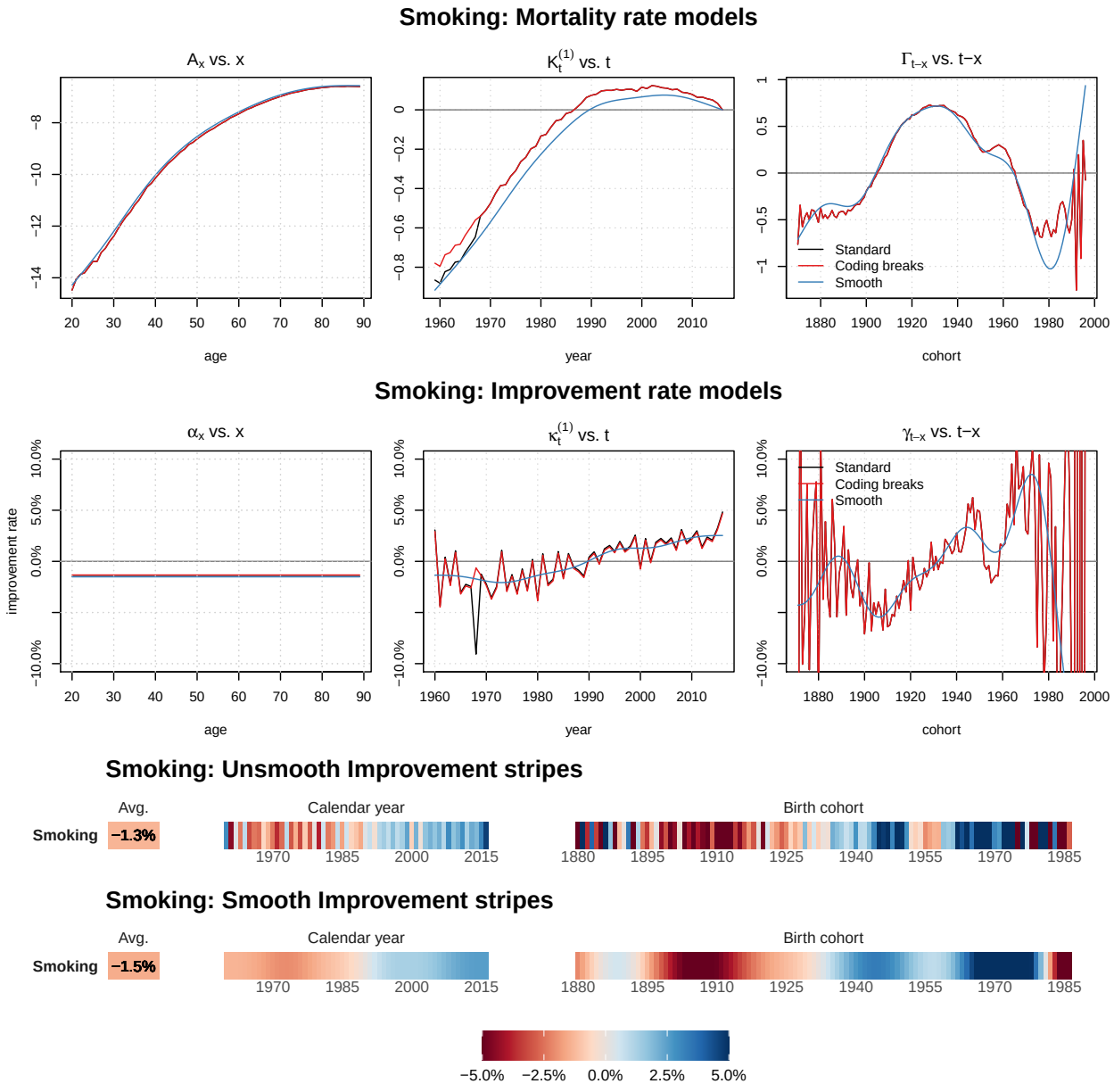


Figure F.40: Fitted parameters for the PCi model for smoking, females, 1959–2016, 20–89



Appendix G: PCi model parameters for males

In this appendix we present the results of applying the different versions of the PCi model discussed in Section 5.3 to male mortality data for the different causes of death and risk factors.

The figure for each cause and each risk factor presents the parameter estimates associated with:

- the "standard" PCi model defined by Equation (5.7),
- the PCi model allowing for "coding breaks" defined by Equation (5.8), and
- the "smooth" PCi model defined by Equation (5.9).

In addition, for each of the models, the figure includes:

- the fitted parameters in the mortality rate scale (top panes),
- the fitted parameters in the improvement rate scale using line plots (middle panes), and
- the fitted parameters in the improvement rate scale using mortality improvement stripes (bottom panes).

G.1 CAUSES OF DEATH

- All-Cause mortality: Figure G.1
  - Circulatory diseases: Figure G.2
    - Ischaemic heart disease: Figure G.3
    - CVD and stroke: Figure G.4
    - Other circulatory system diseases: Figure G.5
  - Neoplasms: Figure G.6
    - Bowel cancer: Figure G.7
    - Liver cancer: Figure G.8
    - Lung cancer: Figure G.9
    - Prostate cancer: Figure G.10
    - Other cancers: Figure G.11
    - Other digestive organ cancers: Figure G.12
  - Respiratory diseases: Figure G.13
    - Influenza and pneumonia: Figure G.14
    - Chronic lower respiratory disease: Figure G.15
    - Other respiratory diseases: Figure G.16
  - Digestive system: Figure G.17

    - Gastric and duodenal ulcer: Figure G.18
    - Chronic liver disease: Figure G.19
    - Other digestive system diseases: Figure G.20
  - External causes: Figure G.21
    - Traffic accidents: Figure G.22
    - Self-harm and interpersonal violence: Figure G.23
    - Other external causes: Figure G.24
  - Other: Figure G.25
    - AIDS and tuberculosis: Figure G.26
    - Diabetes and obesity: Figure G.27
    - Alcohol abuse and drug dependence: Figure G.28
    - Alzheimer's disease: Figure G.29
    - Dementia and other mental disorders: Figure G.30
    - Rest of causes: Figure G.31

G.2 RISK FACTORS

- AIDS and tuberculosis: Figure G.32
- Alcohol abuse: Figure G.33
- Dementia and Alzheimer's disease: Figure G.34
- Diabetes and obesity: Figure G.35
- Drug dependency: Figure G.36
- Homicide: Figure G.37
- Hypertensive disease: Figure G.38
- Self-harm: Figure G.39
- Smoking: Figure G.40

Figure G.1: Fitted parameters for the PCi model for All-Causes of death, males, 1959–2016, 20–89

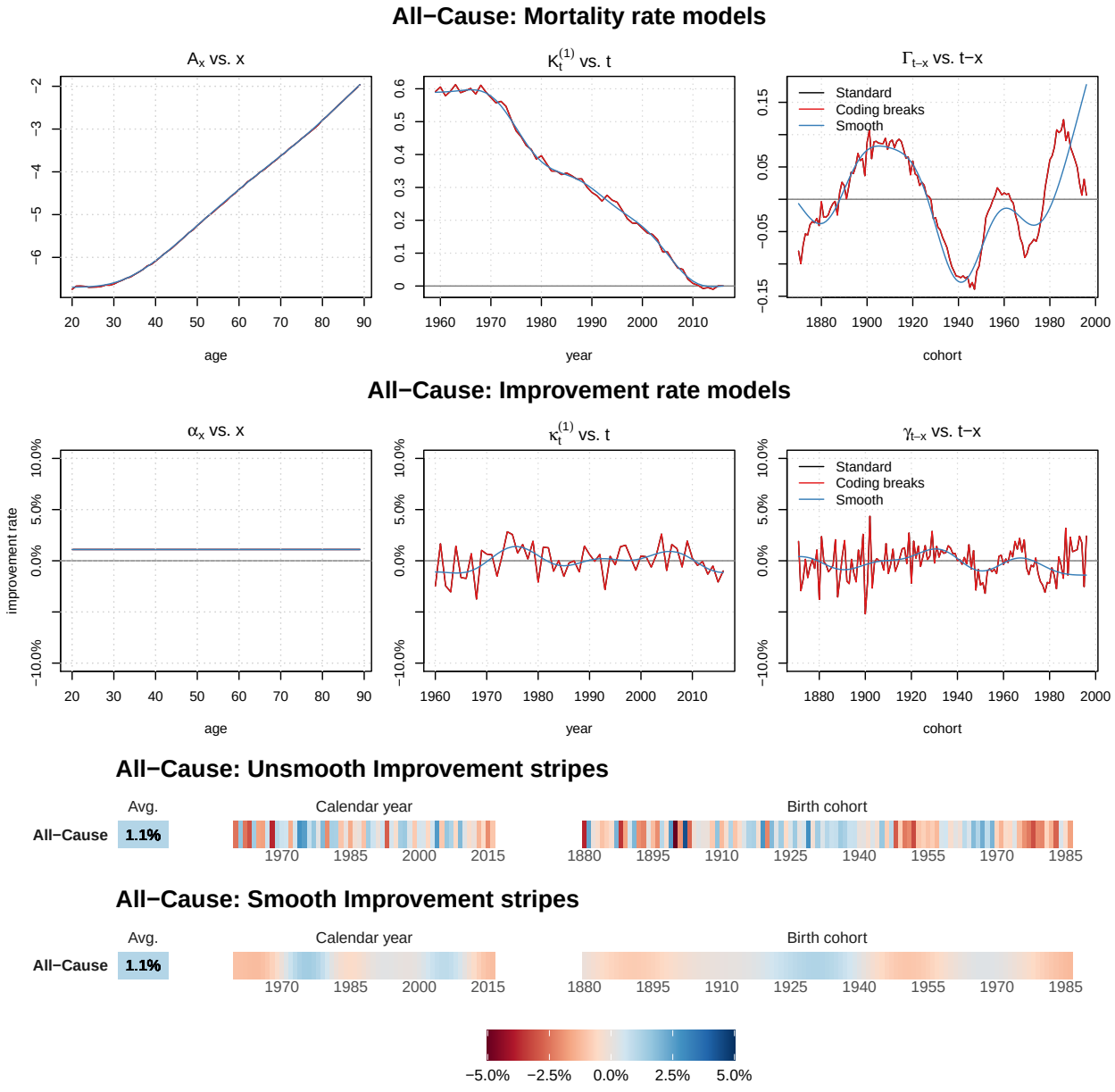


Figure G.2: Fitted parameters for the PCi model for circulatory diseases, males, 1959–2016, 20–89

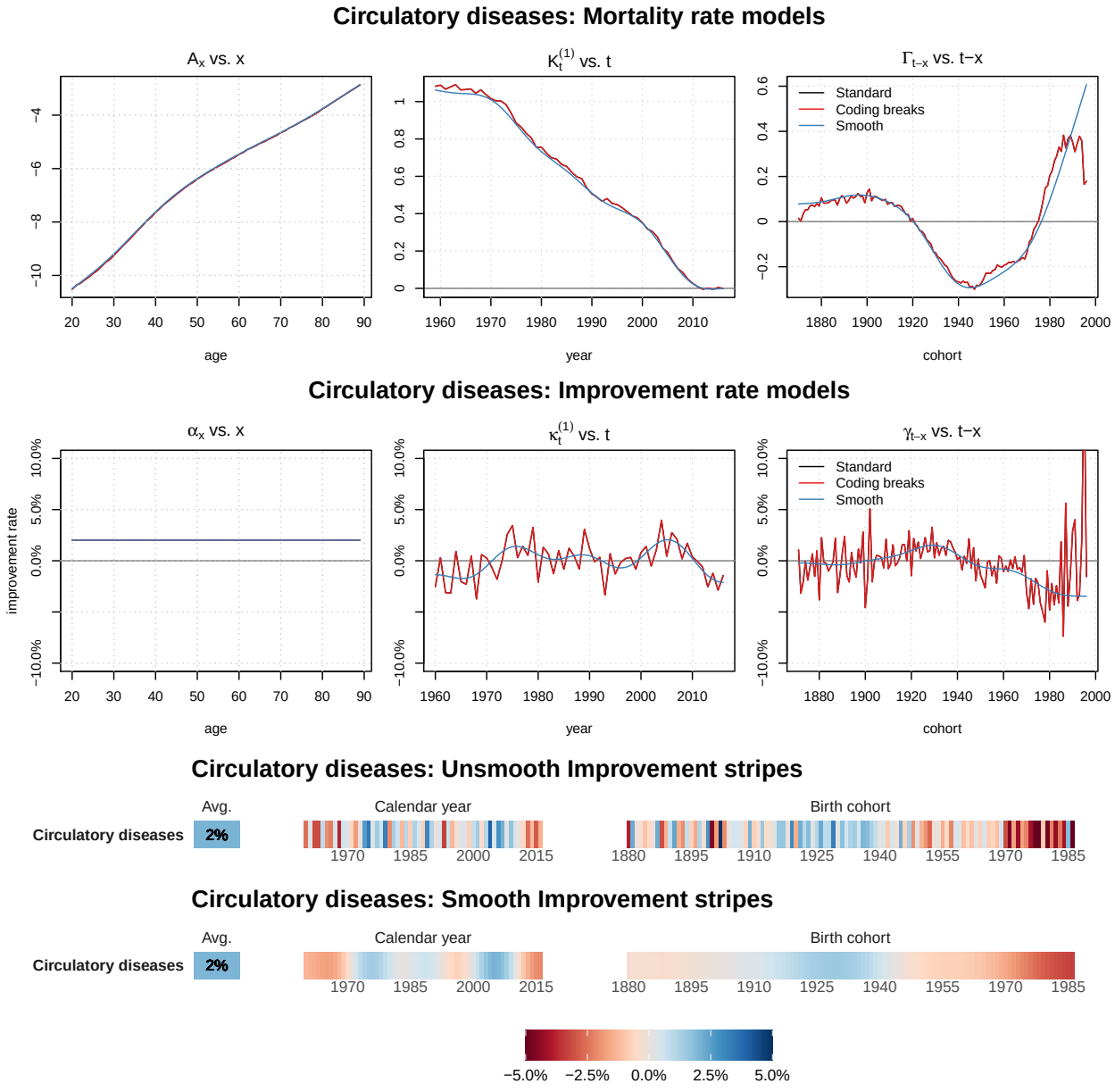


Figure G.3: Fitted parameters for the PCi model for ischaemic heart disease, males, 1959–2016, 20–89

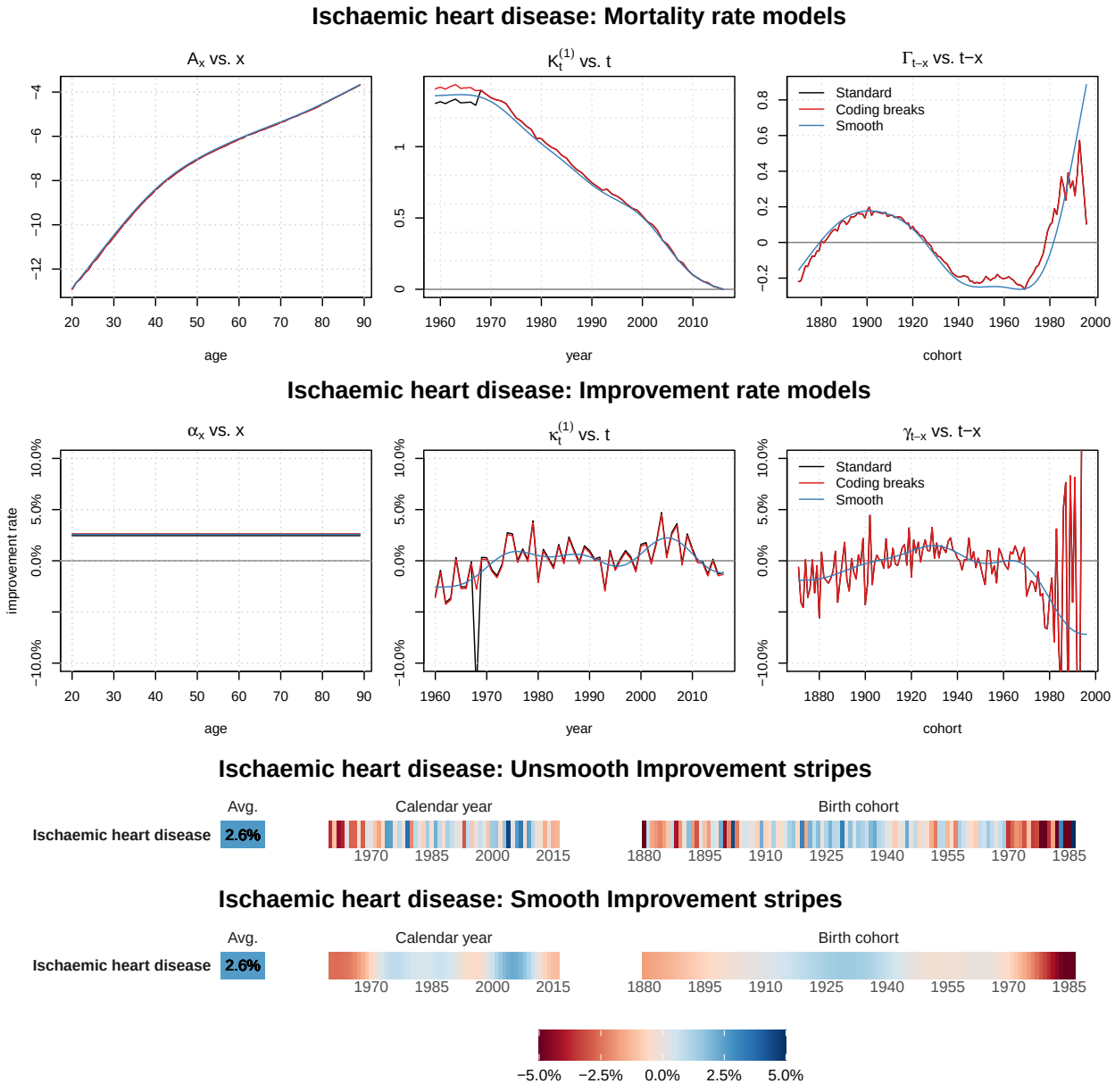


Figure G.4: Fitted parameters for the PCi model for CVD and stroke, males, 1959–2016, 20–89

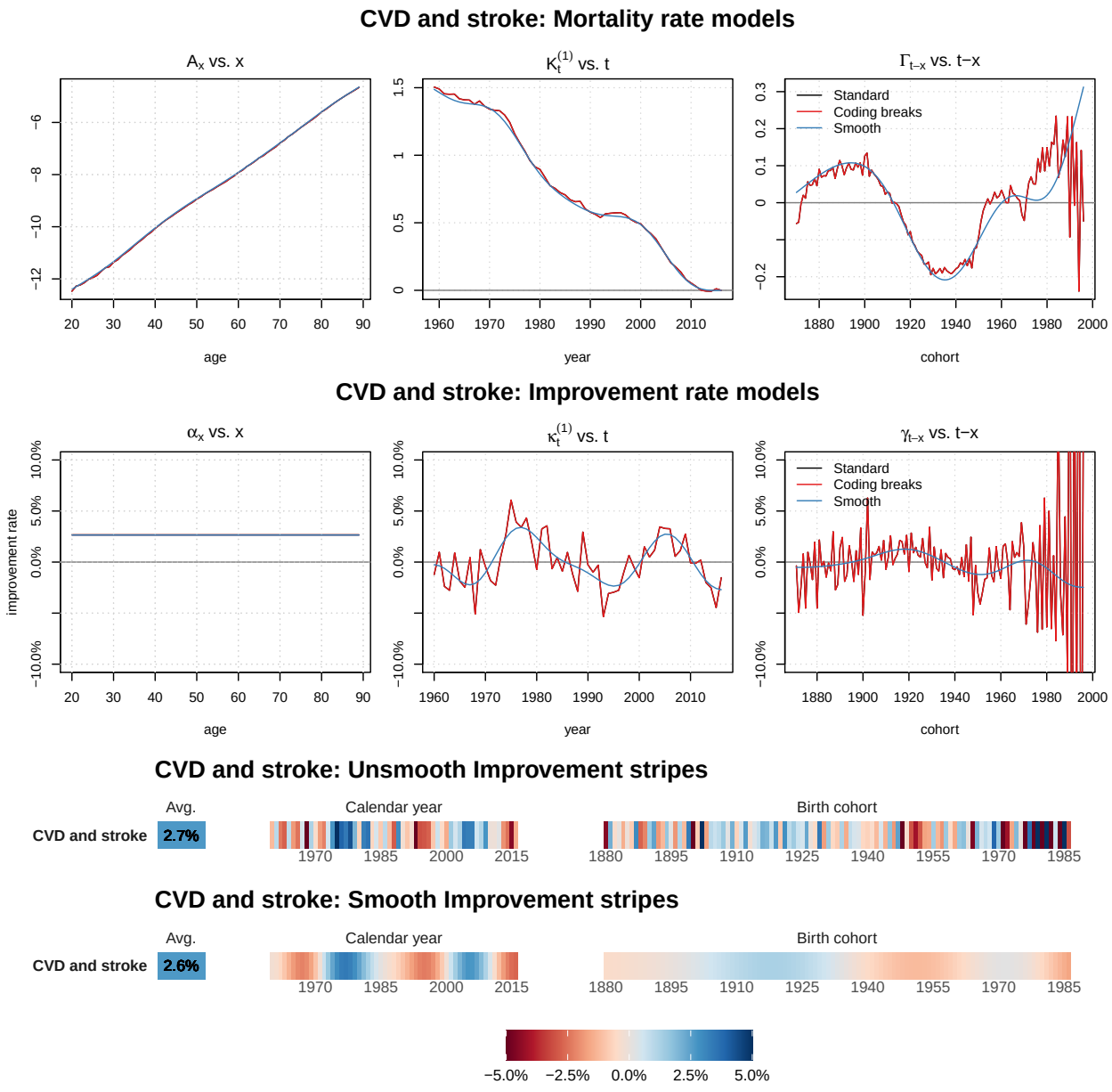


Figure G.5: Fitted parameters for the PCi model for other circulatory system diseases, males, 1959–2016, 20–89

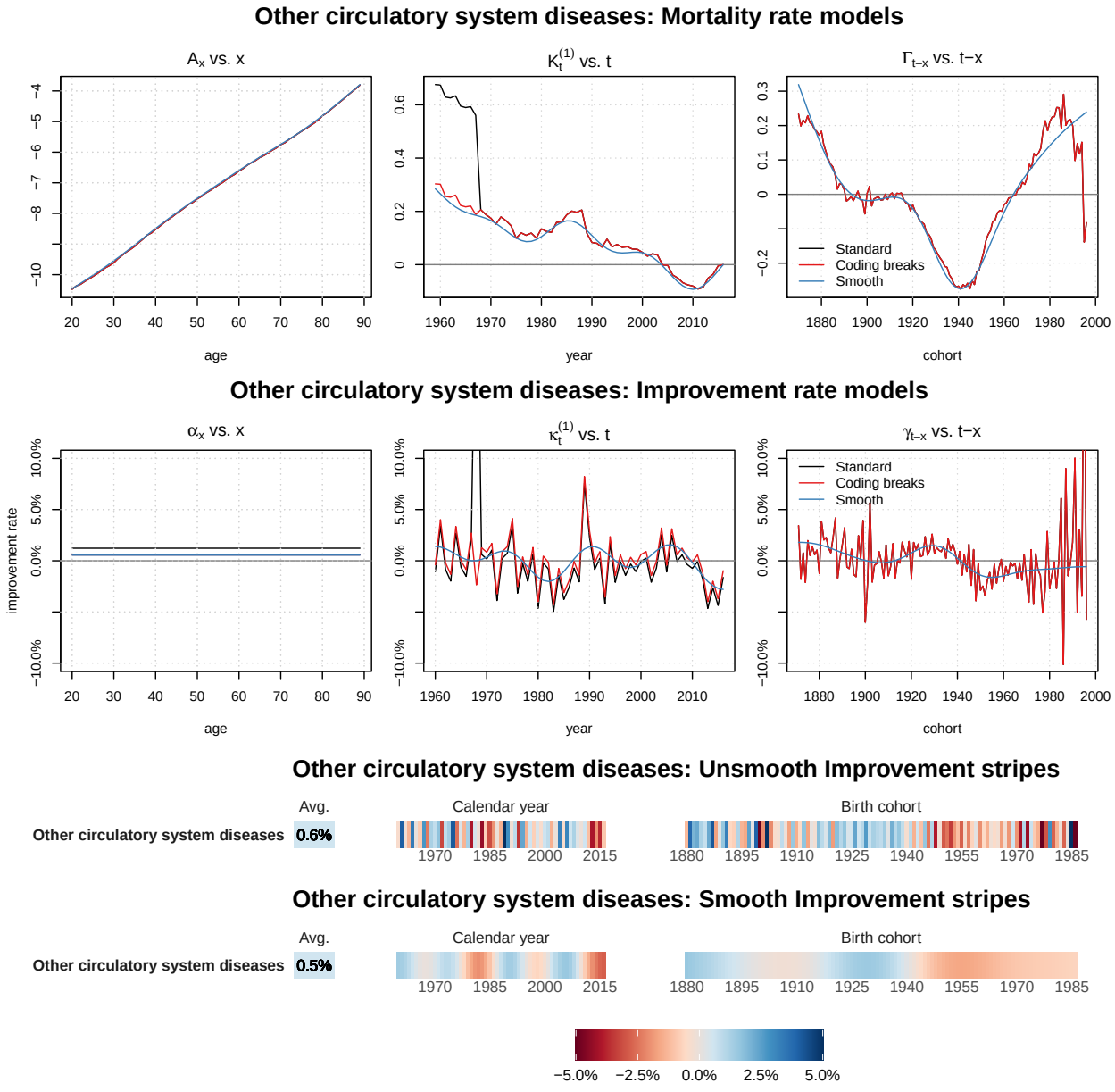


Figure G.6: Fitted parameters for the PCi model for neoplasms, males, 1959–2016, 20–89

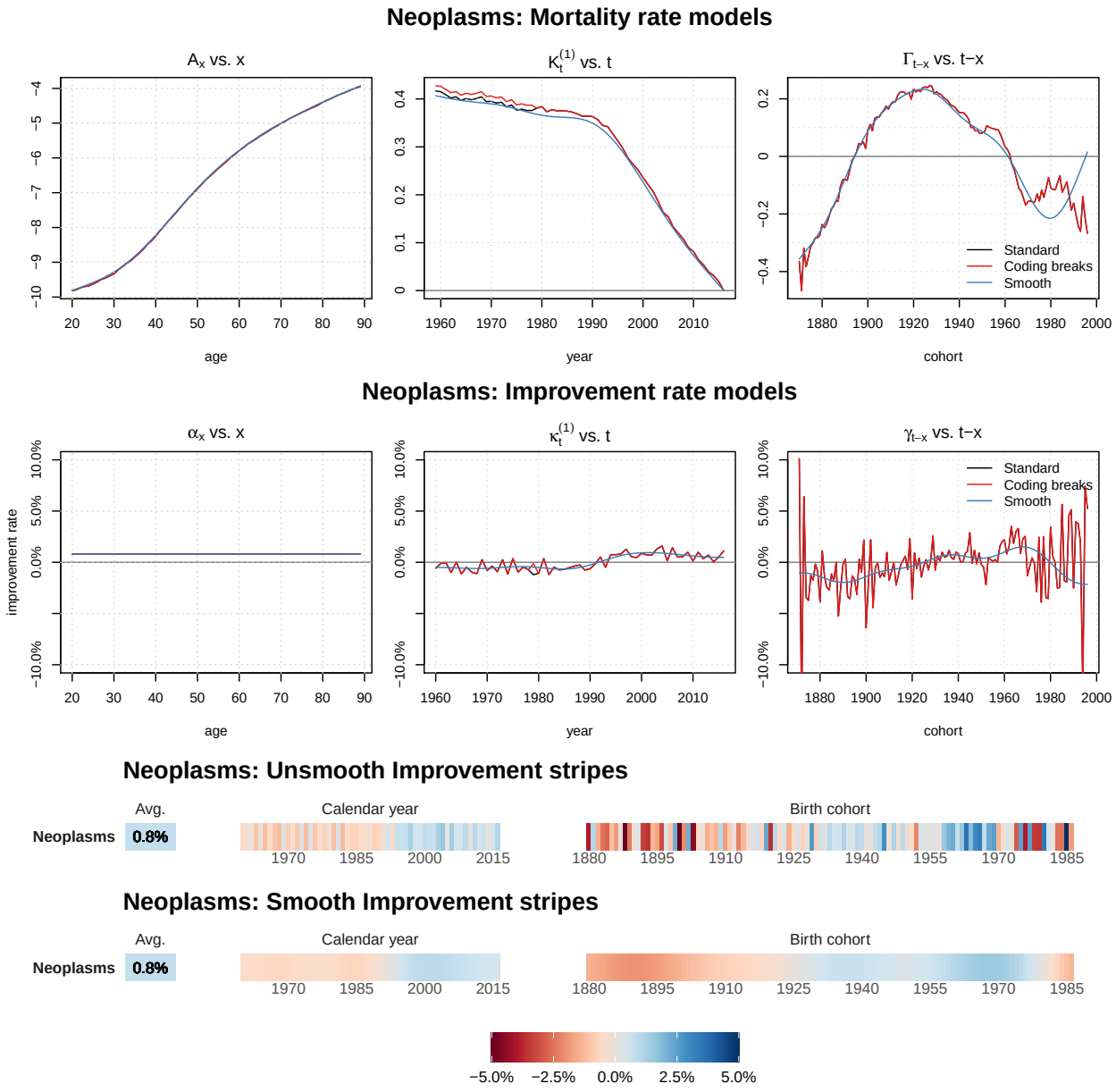


Figure G.7: Fitted parameters for the PCi model for bowel cancer, males, 1959–2016, 20–89

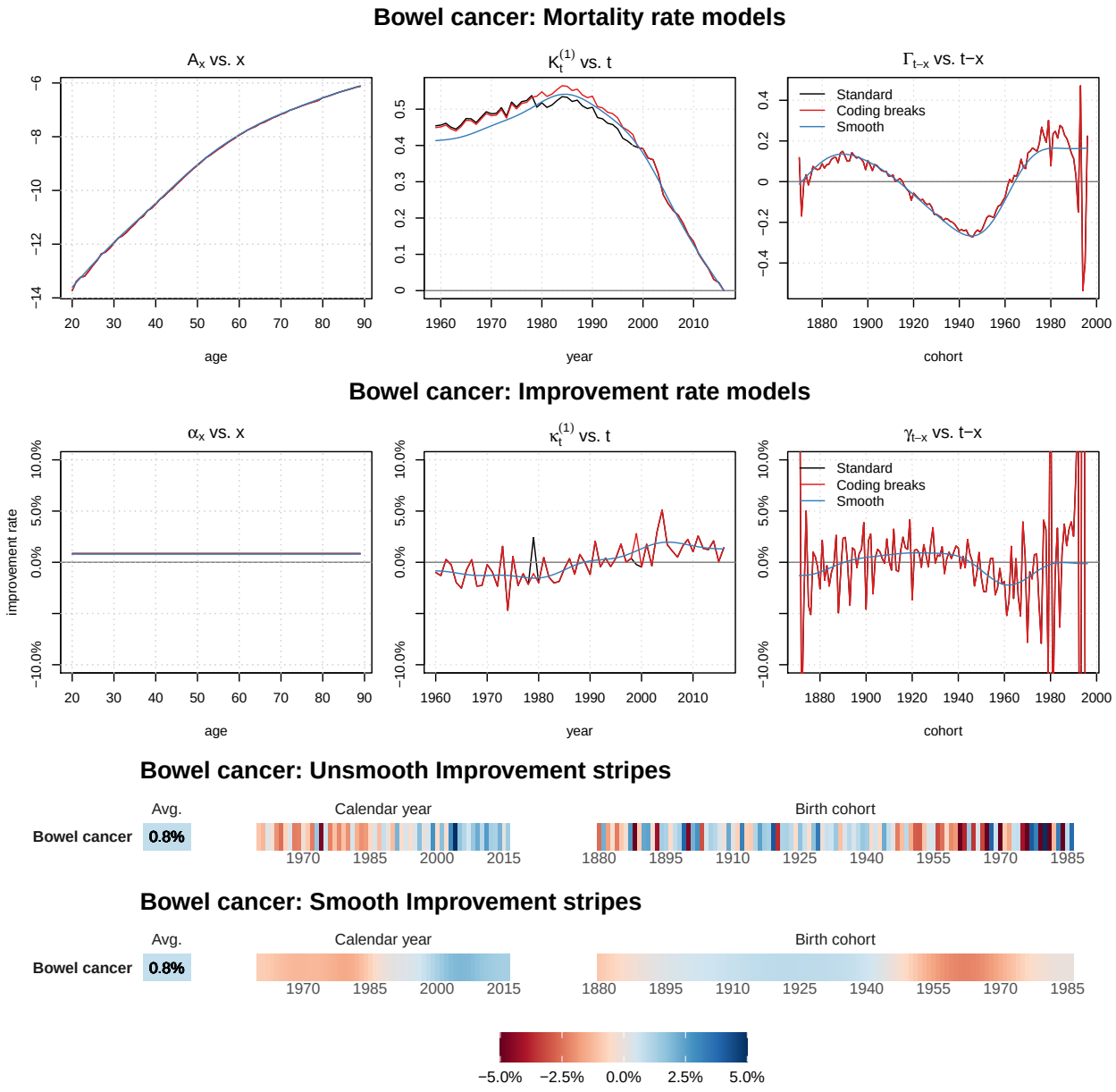


Figure G.8: Fitted parameters for the PCi model for liver cancer, males, 1959–2016, 20–89

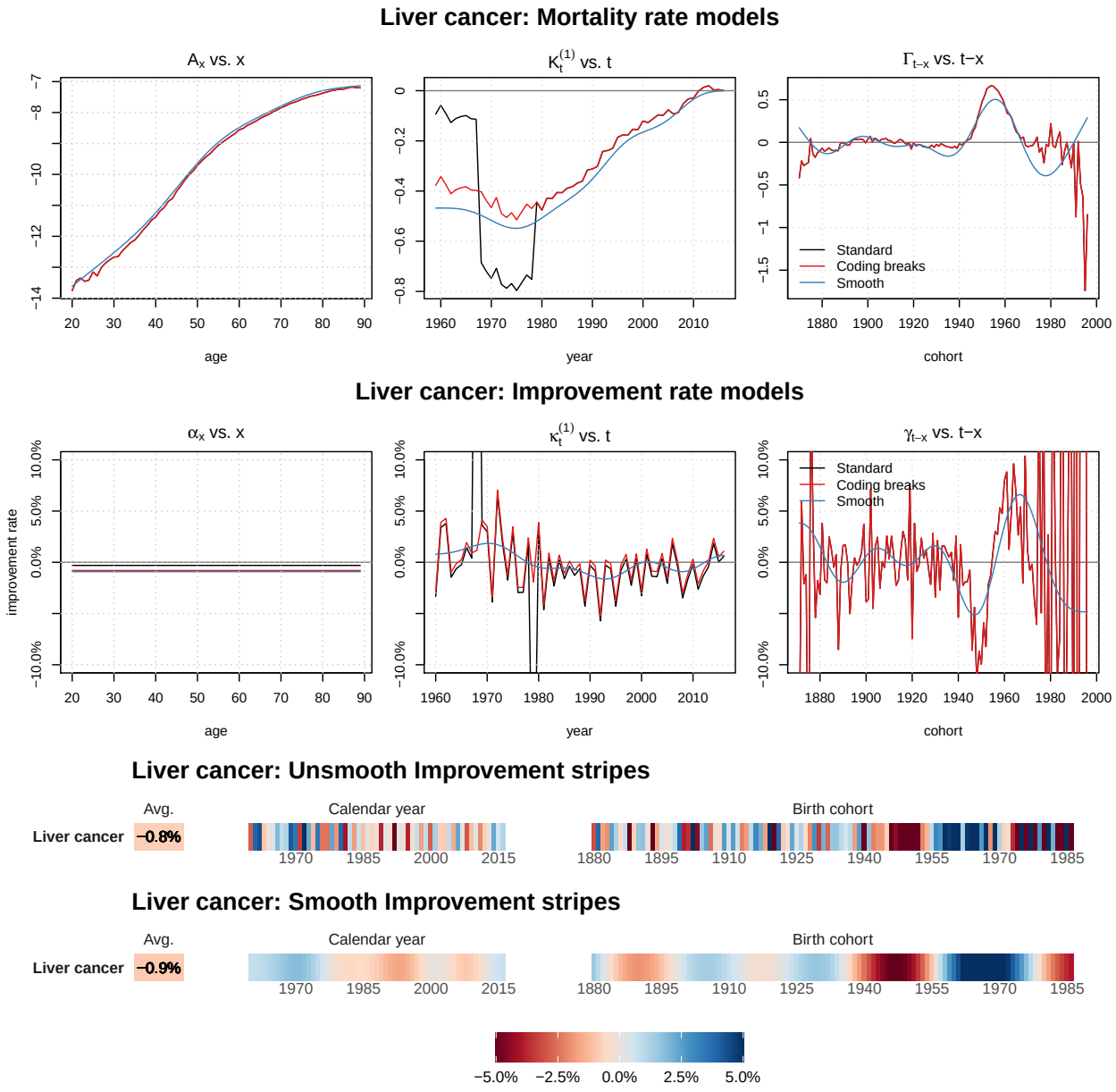


Figure G.9: Fitted parameters for the PCi model for lung cancer, males, 1959–2016, 20–89

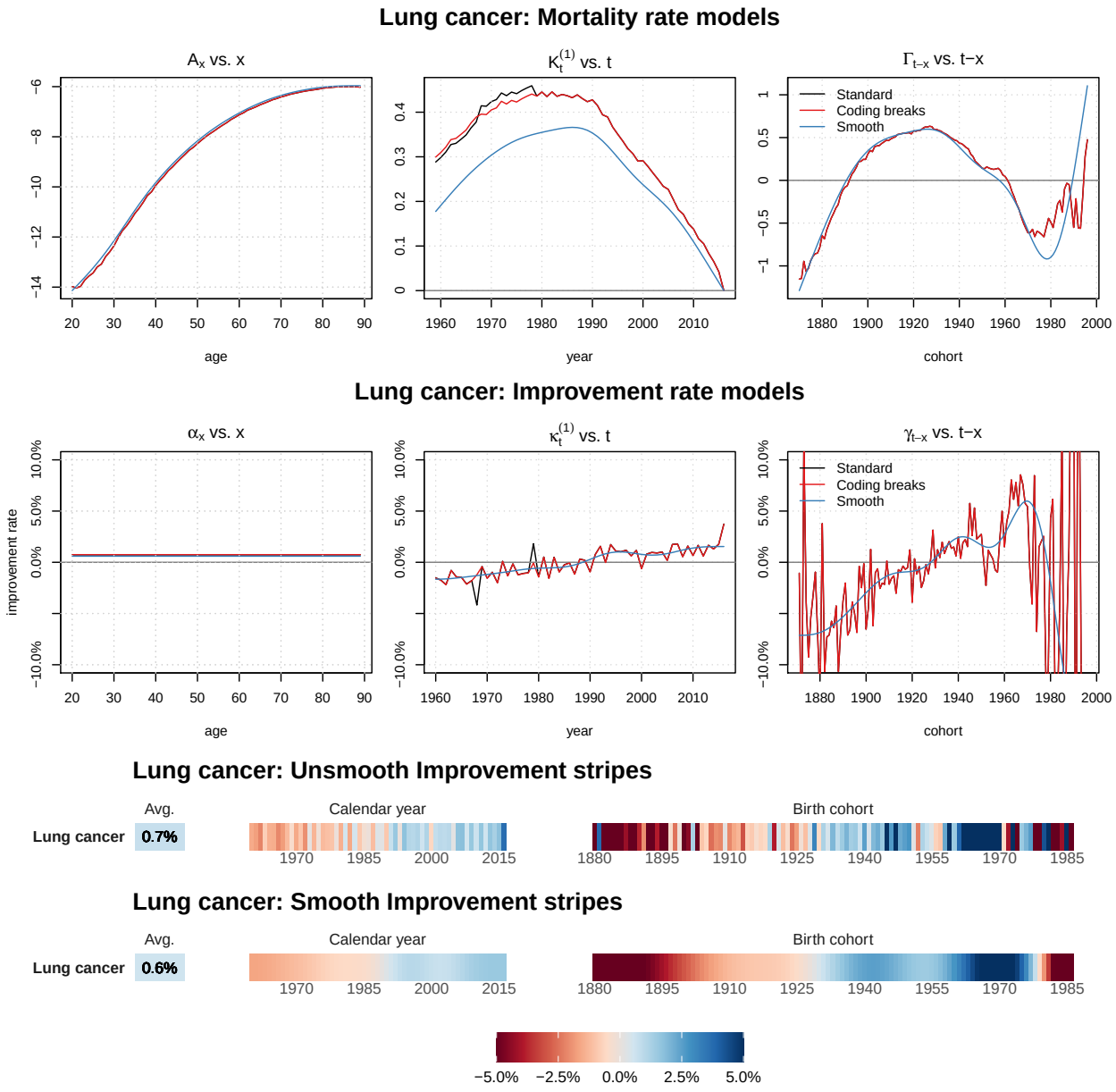


Figure G.10: Fitted parameters for the PCi model for prostate cancer, males, 1959–2016, 20–89

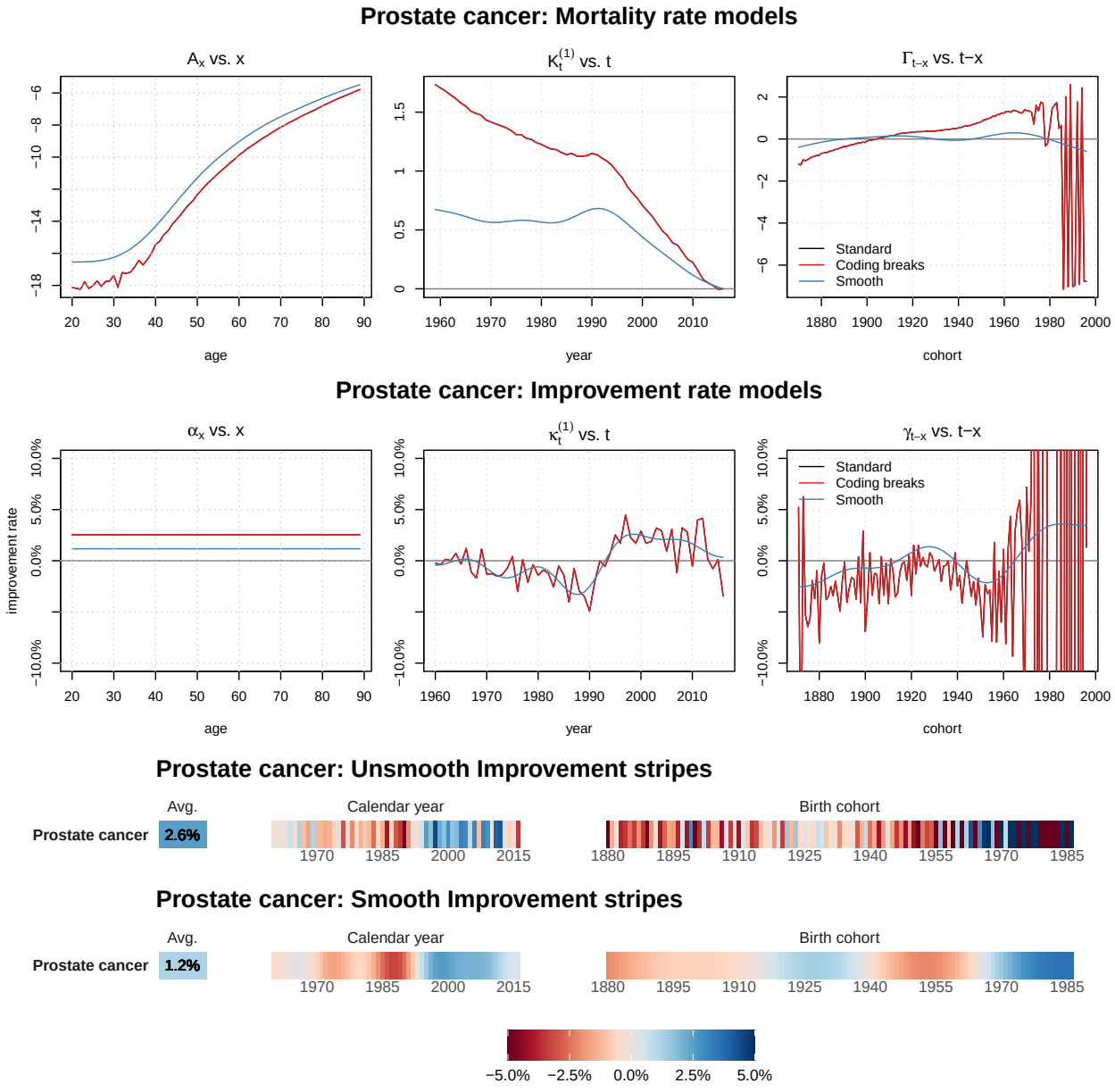


Figure G.11: Fitted parameters for the PCi model for other cancers, males, 1959–2016, 20–89

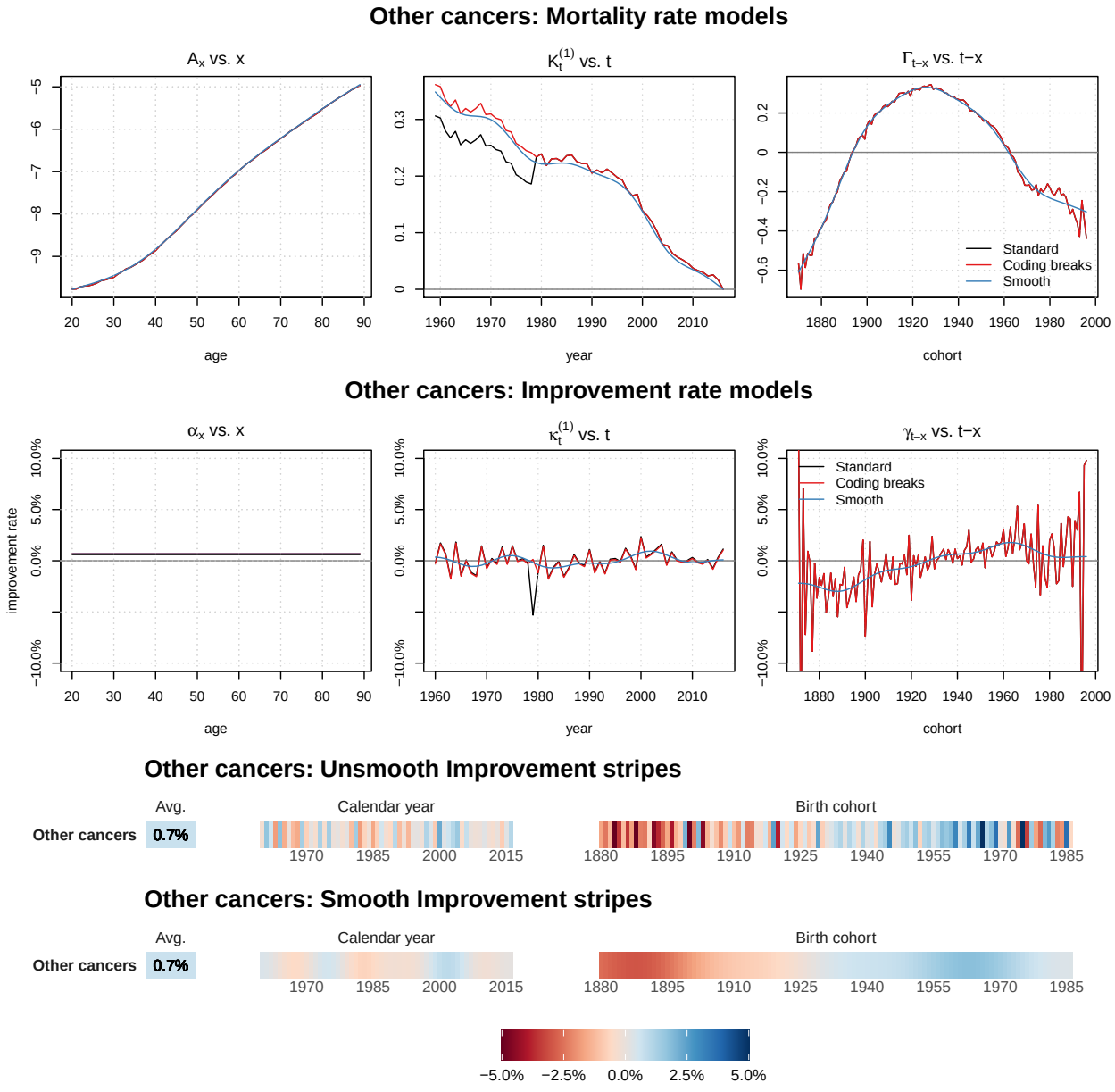


Figure G.12: Fitted parameters for the PCi model for other digestive organ cancers, males, 1959–2016, 20–89

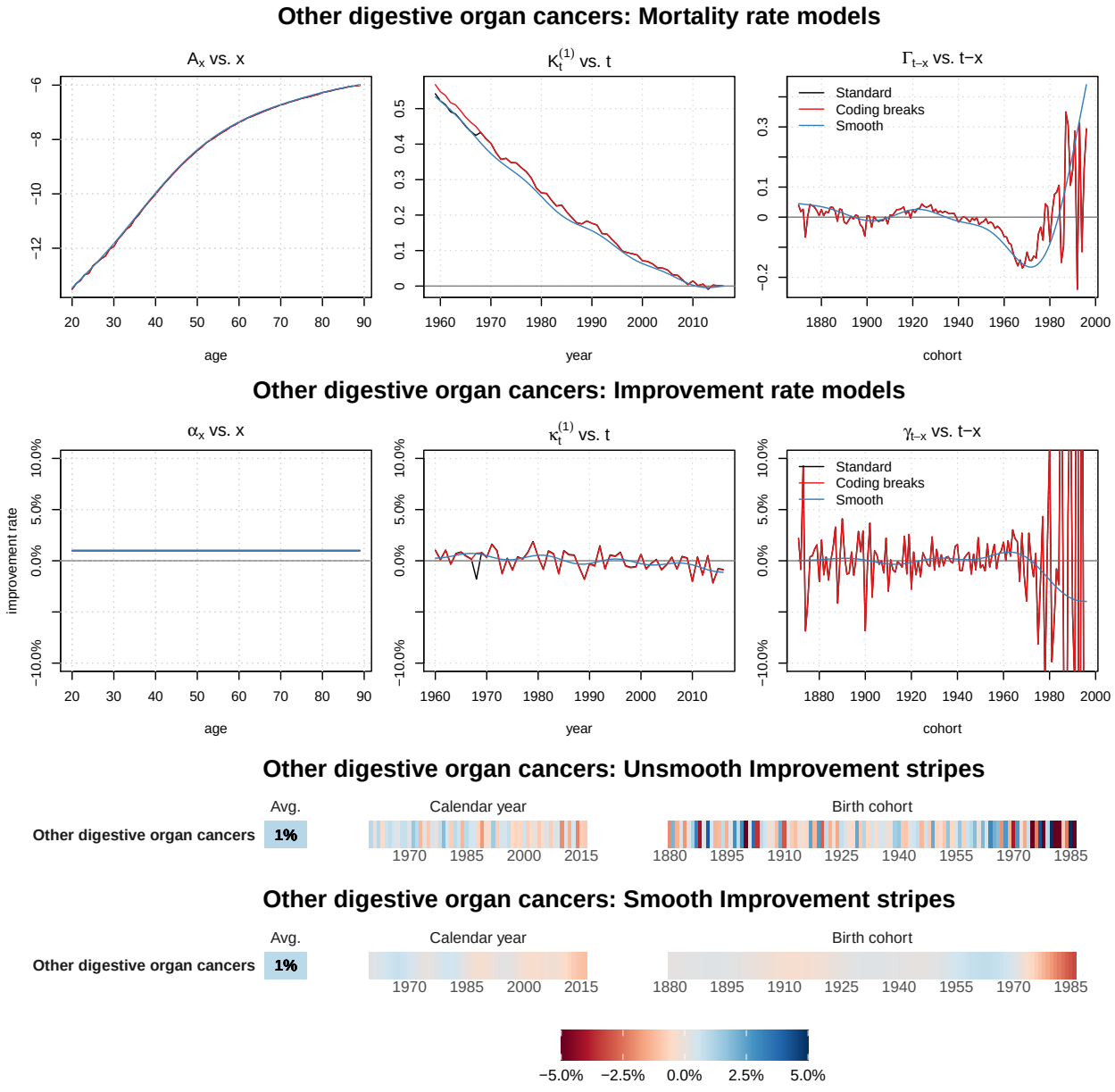


Figure G.13: Fitted parameters for the PCi model for respiratory diseases, males, 1959–2016, 20–89

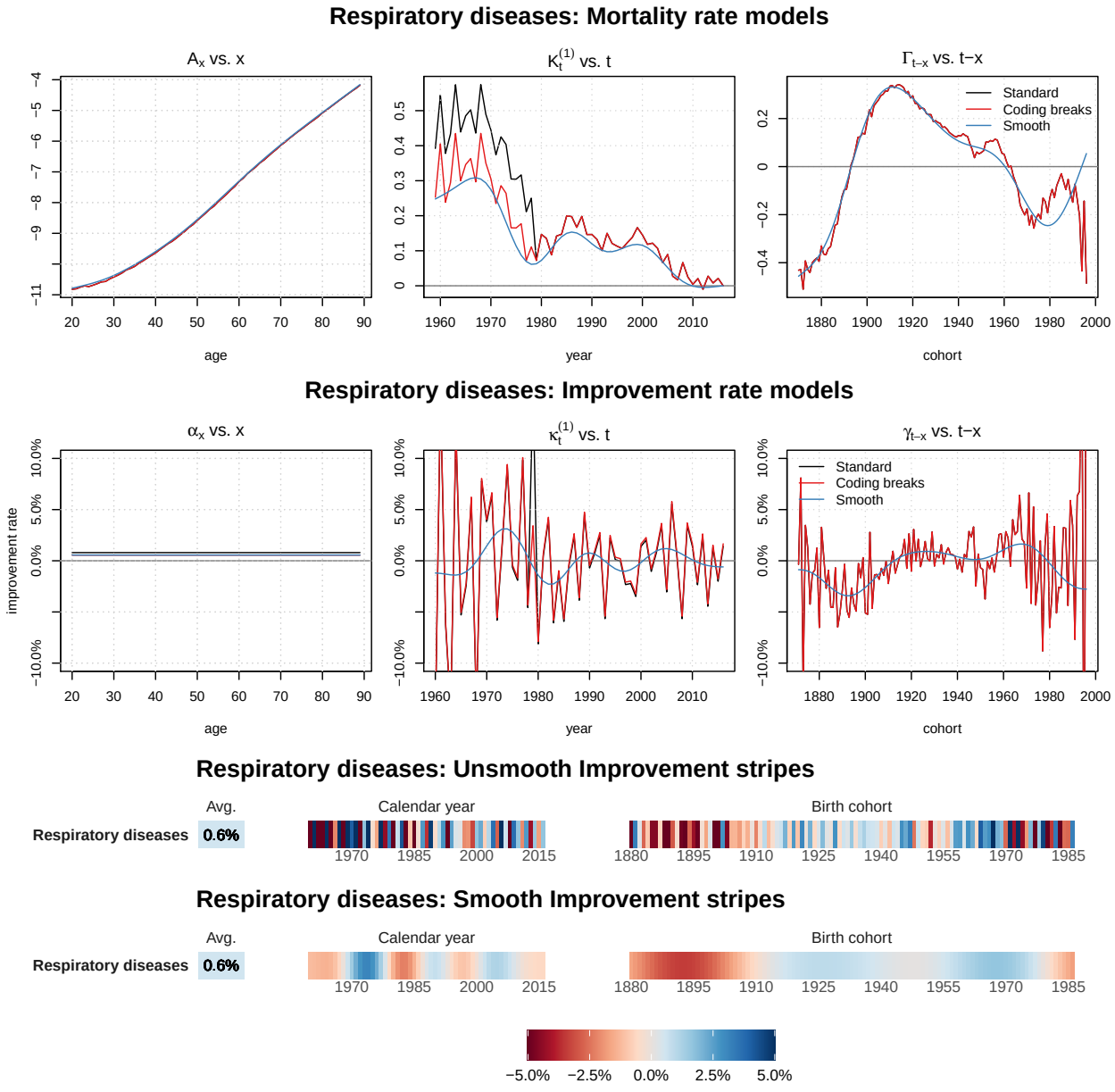


Figure G.14: Fitted parameters for the PCi model for influenza and pneumonia, males, 1959–2016, 20–89

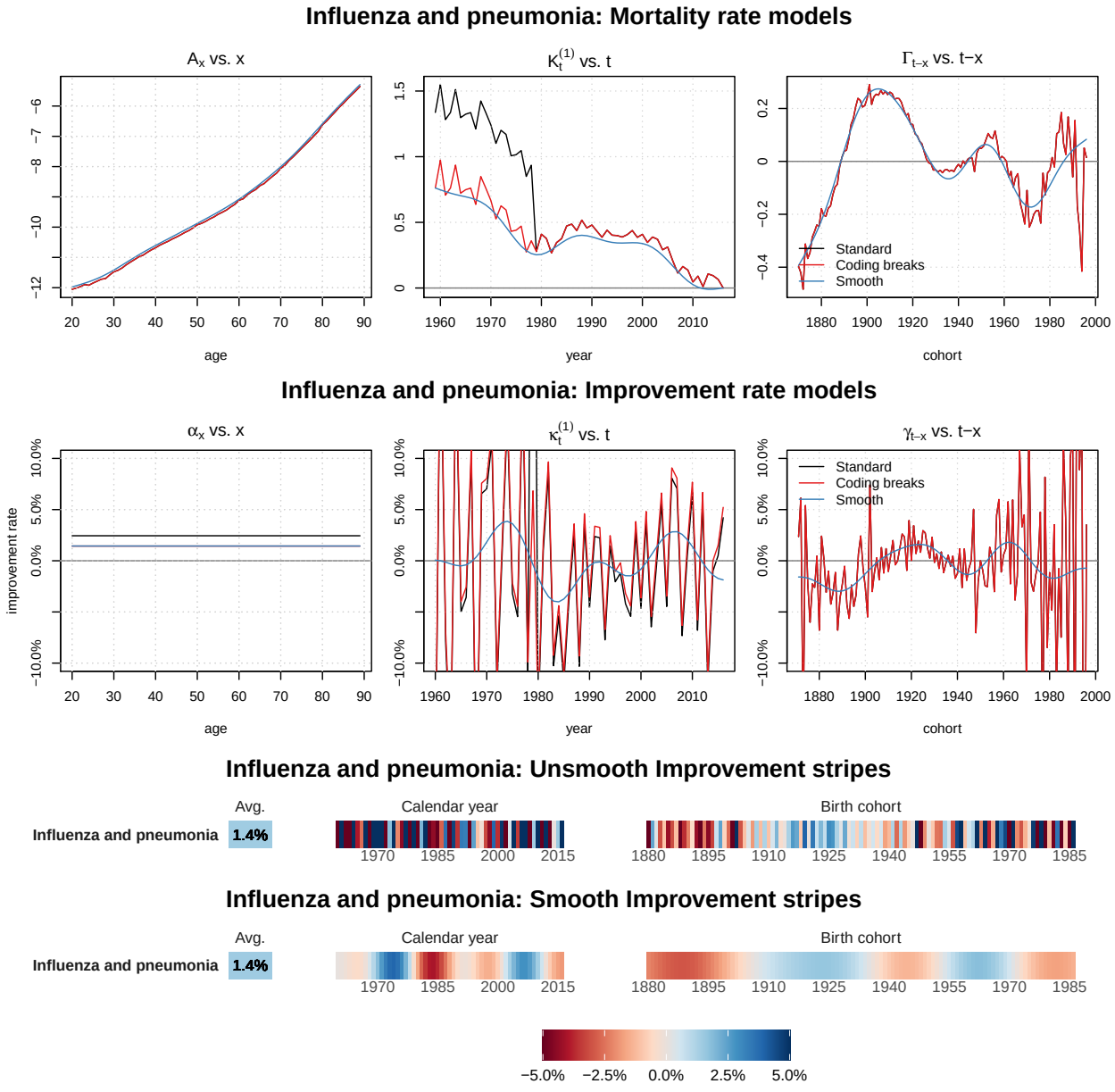


Figure G.15: Fitted parameters for the PCi model for chronic lower respiratory disease, males, 1959–2016, 20–89

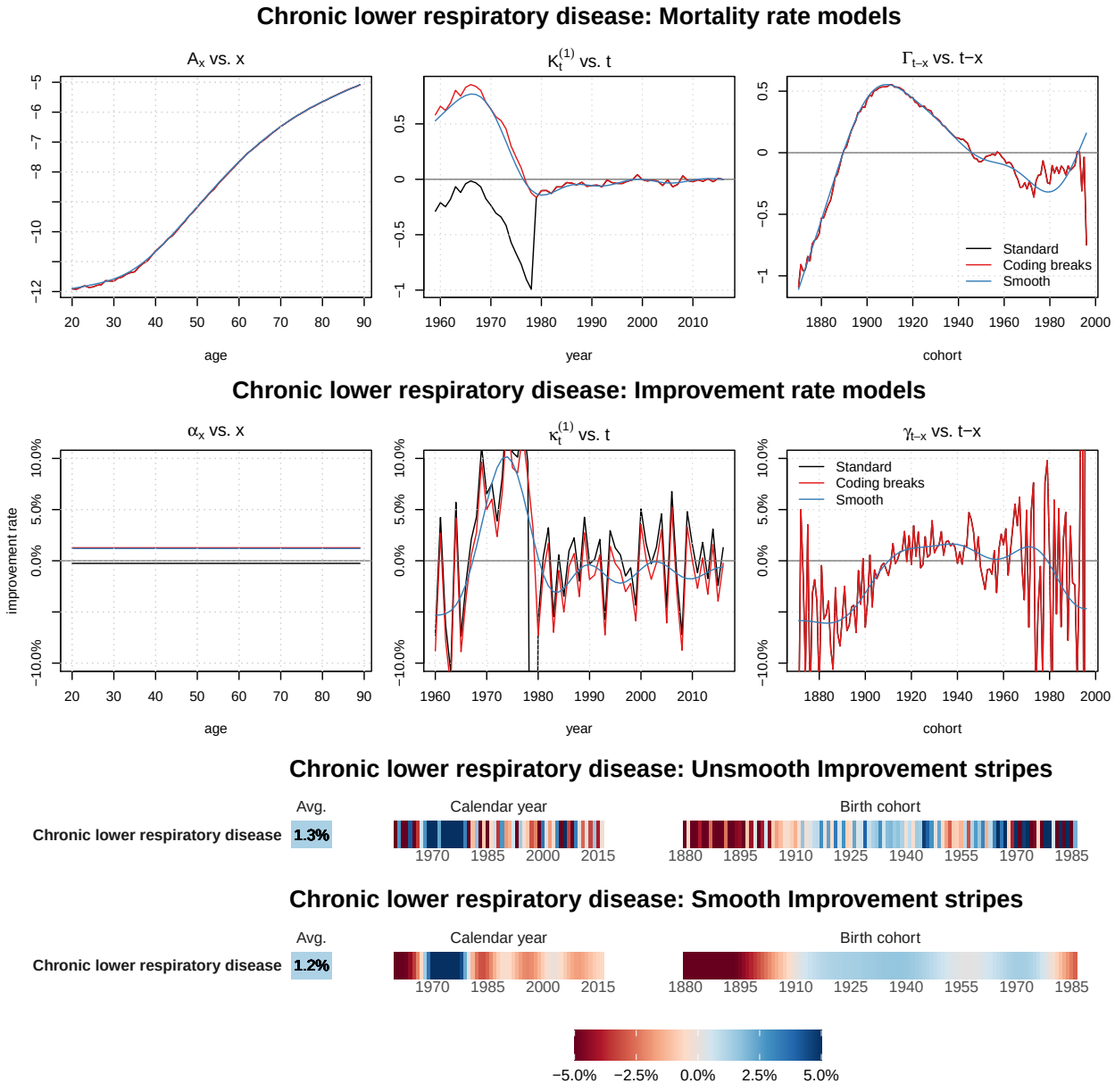


Figure G.16: Fitted parameters for the PCi model for other respiratory diseases, males, 1959–2016, 20–89

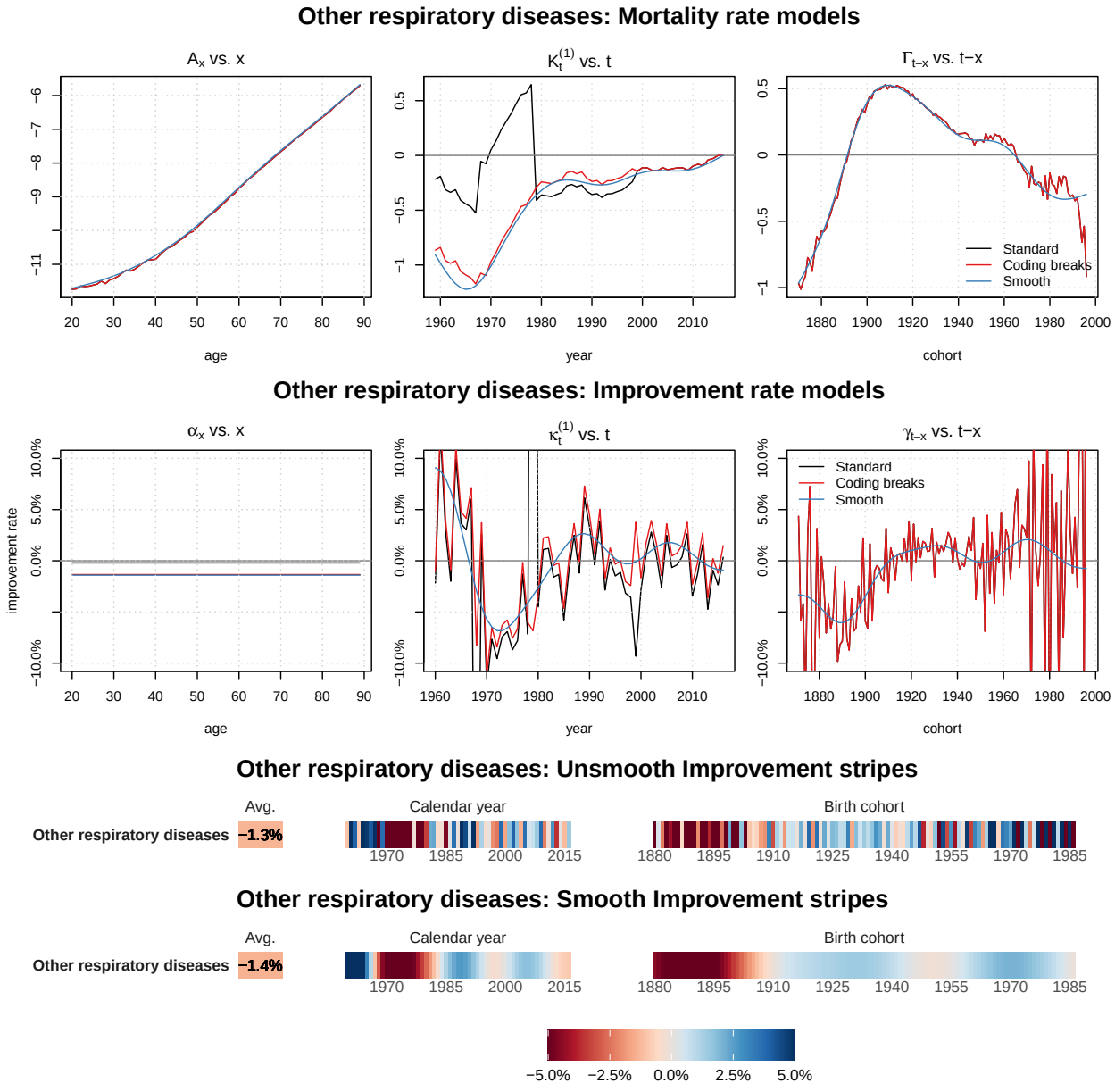


Figure G.17: Fitted parameters for the PCi model for digestive system, males, 1959–2016, 20–89

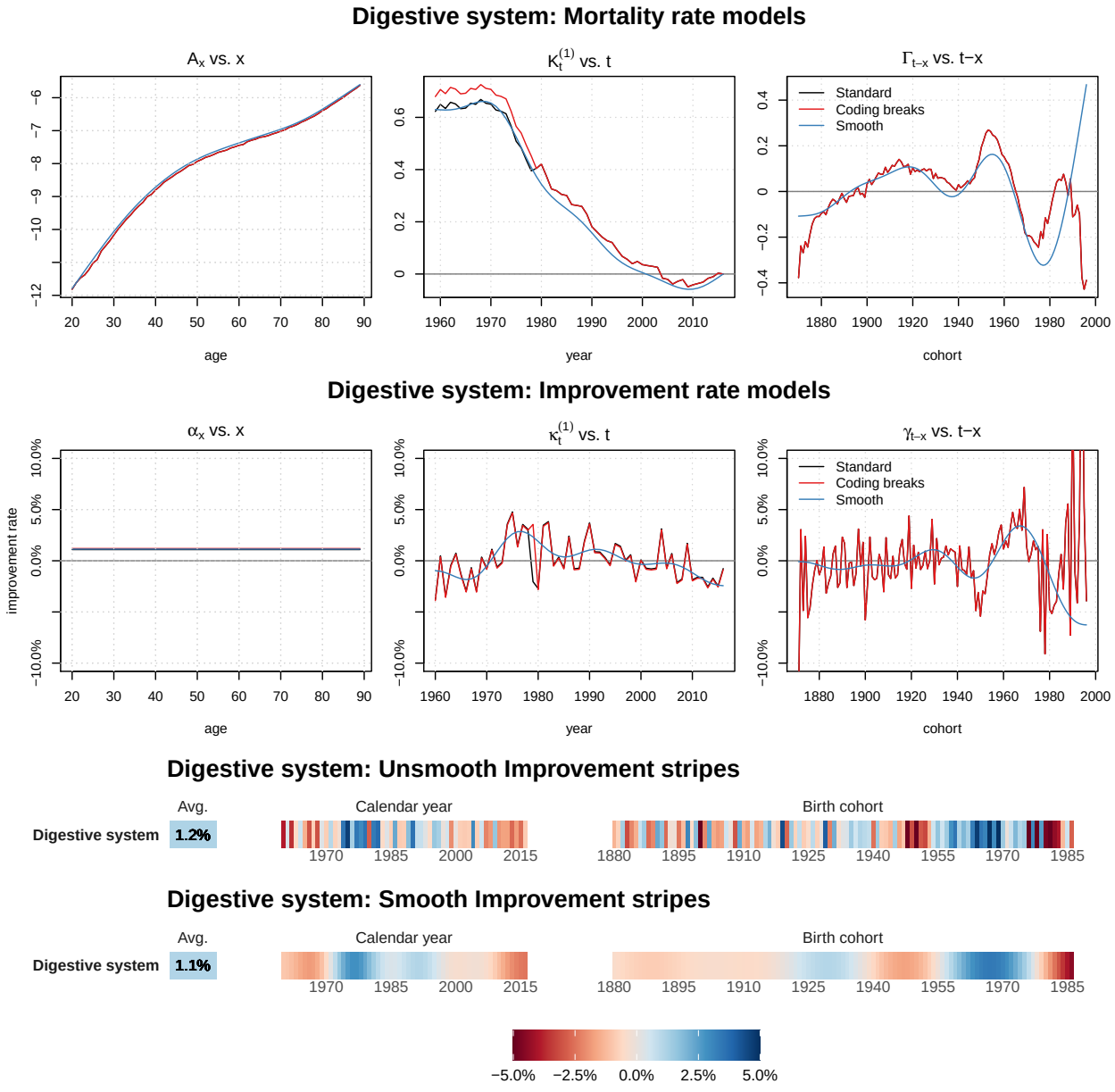


Figure G.18: Fitted parameters for the PCi model for gastric and duodenal ulcer, males, 1959–2016, 20–89

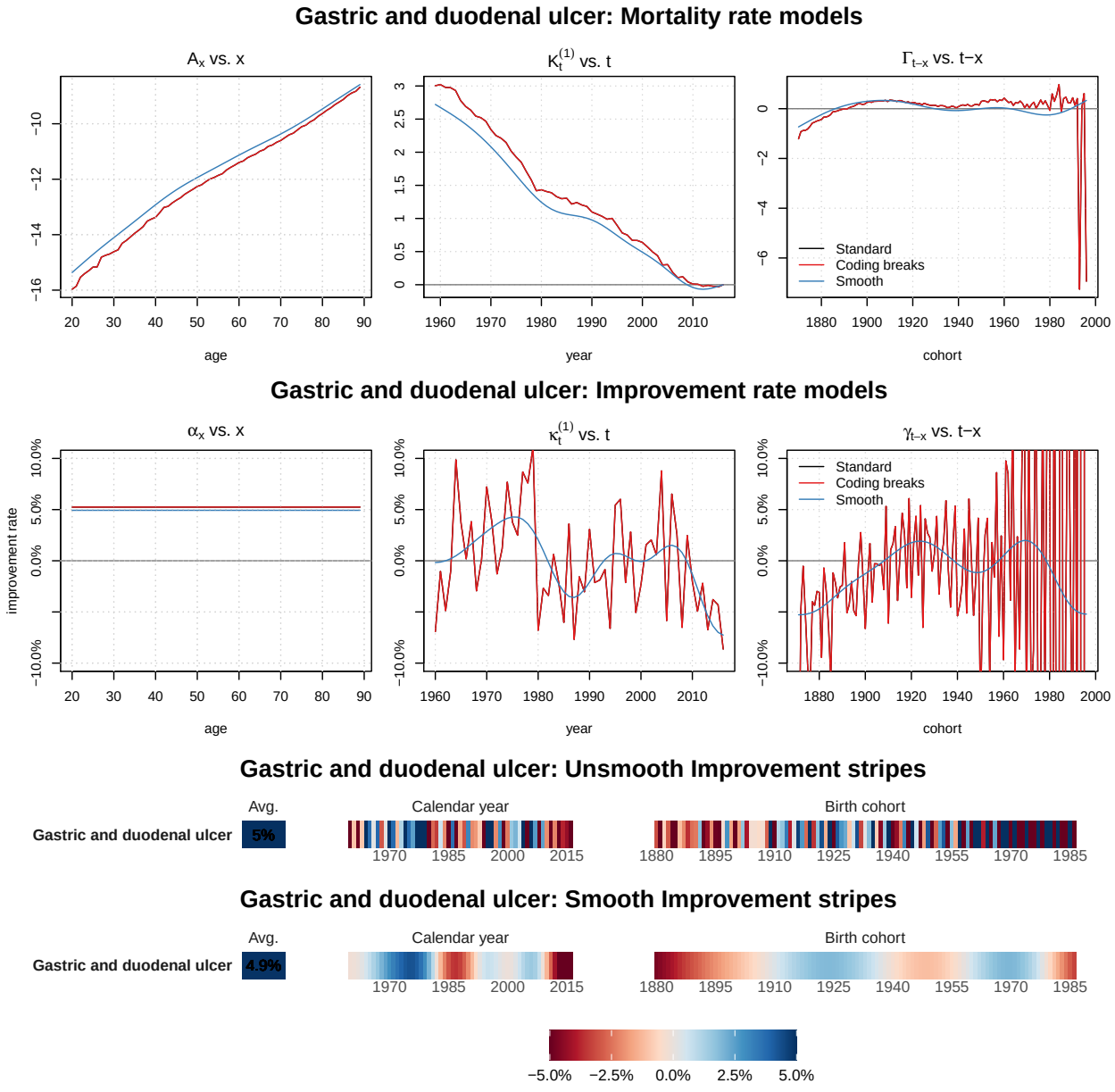


Figure G.19: Fitted parameters for the PCi model for chronic liver disease, males, 1959–2016, 20–89

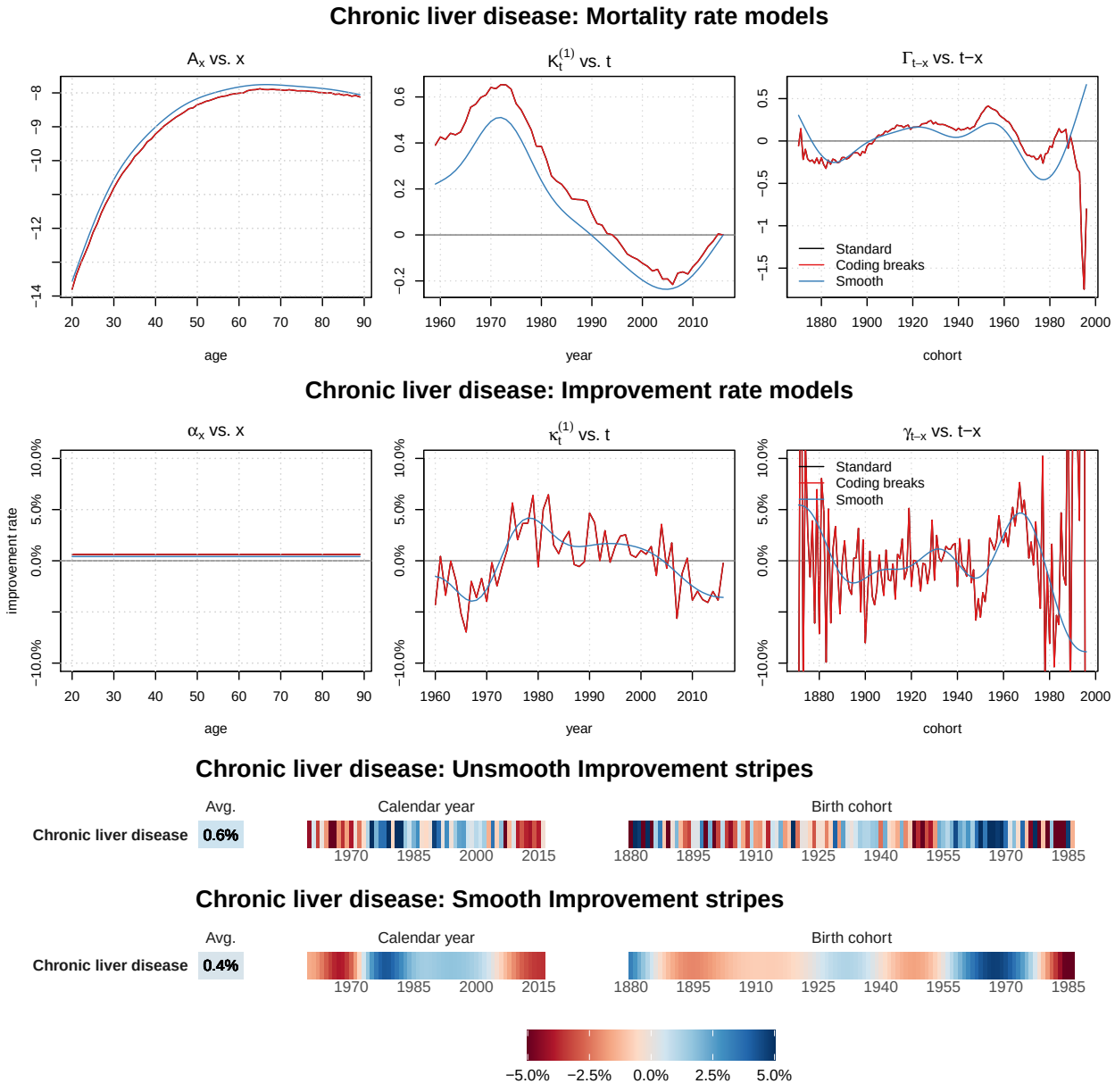


Figure G.20: Fitted parameters for the PCi model for other digestive system diseases, males, 1959–2016, 20–89

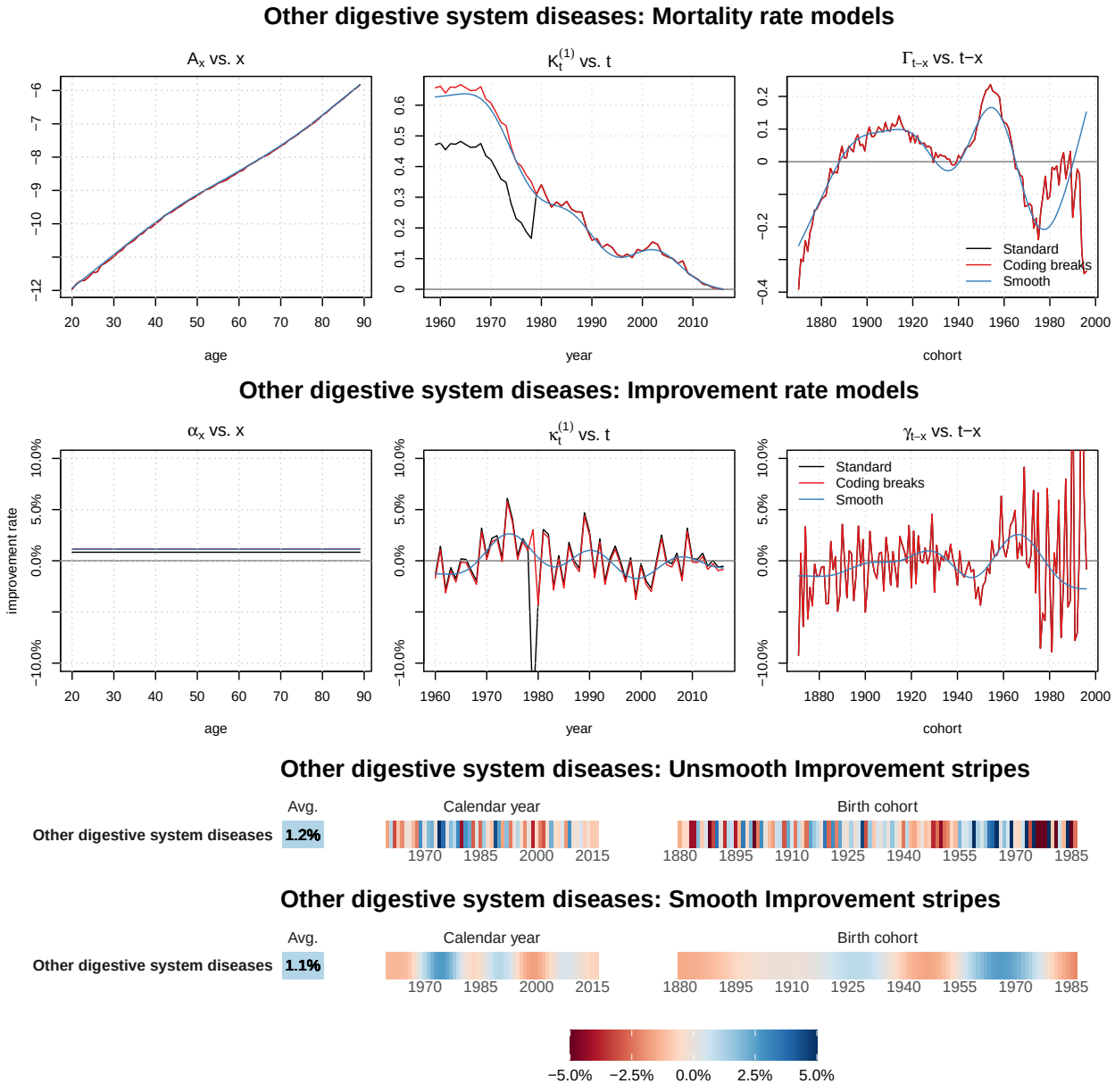


Figure G.21: Fitted parameters for the PCi model for external causes, males, 1959–2016, 20–89

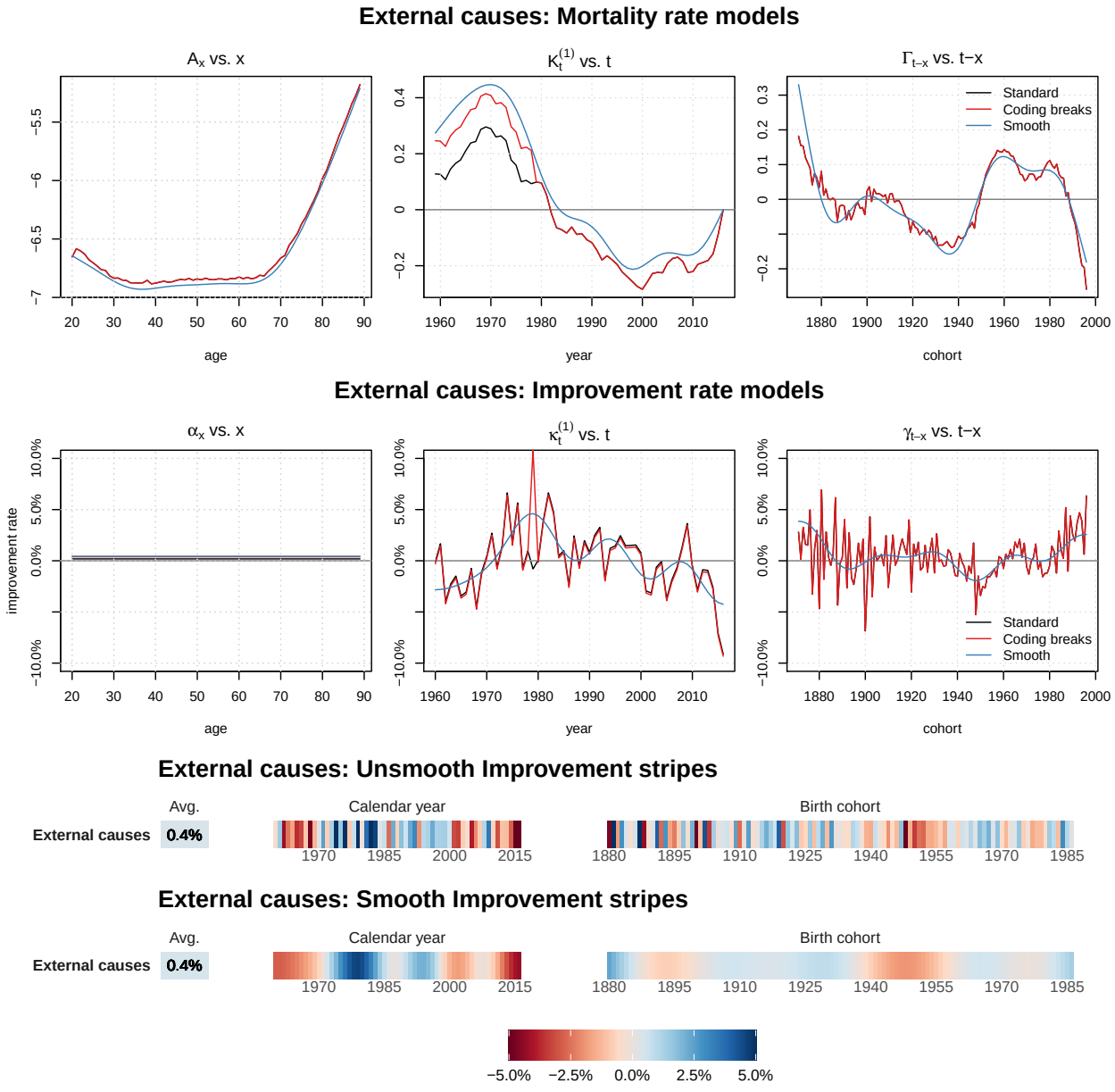


Figure G.22: Fitted parameters for the PCi model for traffic accidents, males, 1959–2016, 20–89

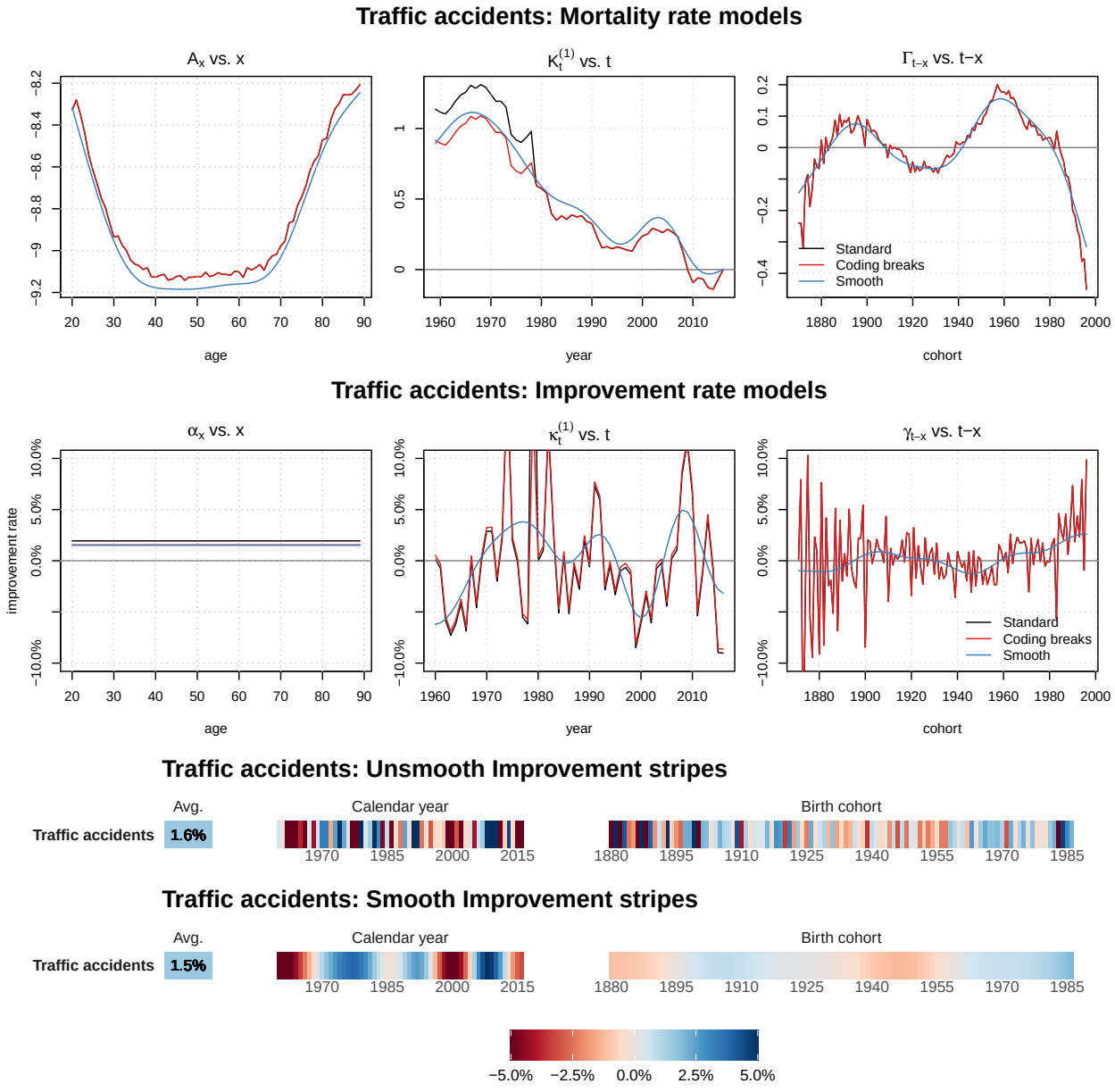


Figure G.23: Fitted parameters for the PCi model for self-harm and interpersonal violence, males, 1959–2016, 20–89

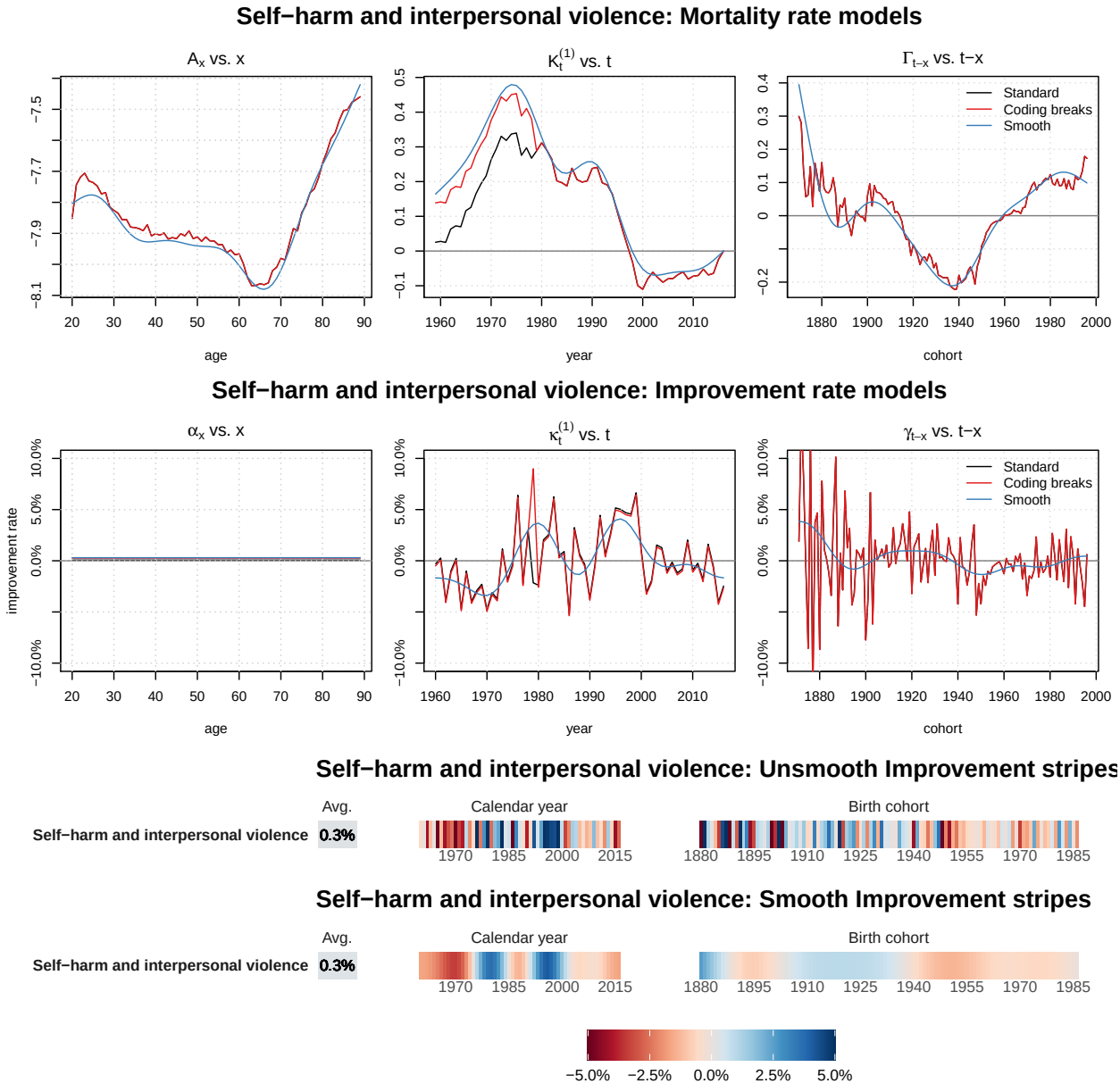


Figure G.24: Fitted parameters for the PCi model for other external causes, males, 1959–2016, 20–89

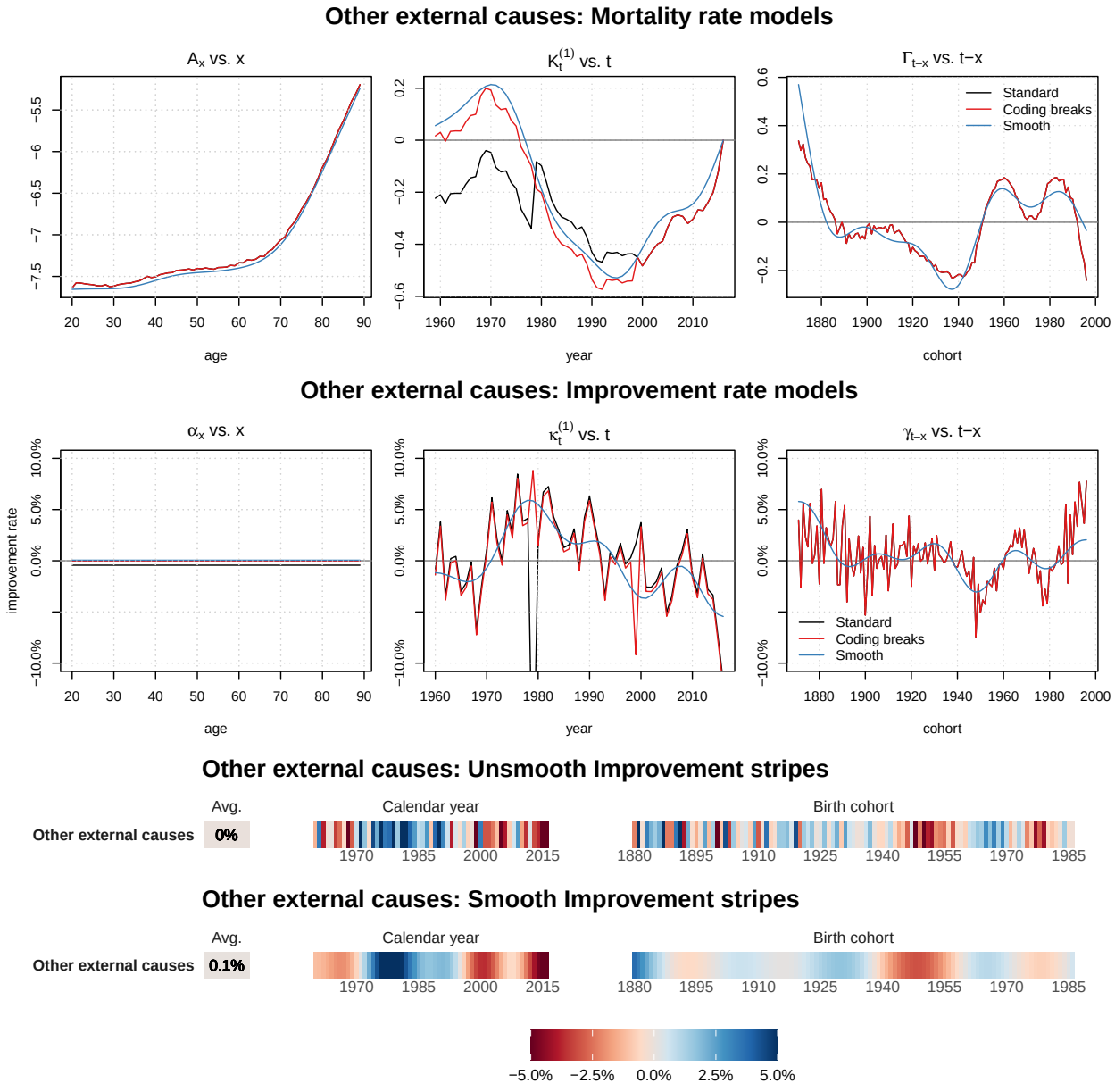


Figure G.25: Fitted parameters for the PCi model for Other causes, males, 1959–2016, 20–89

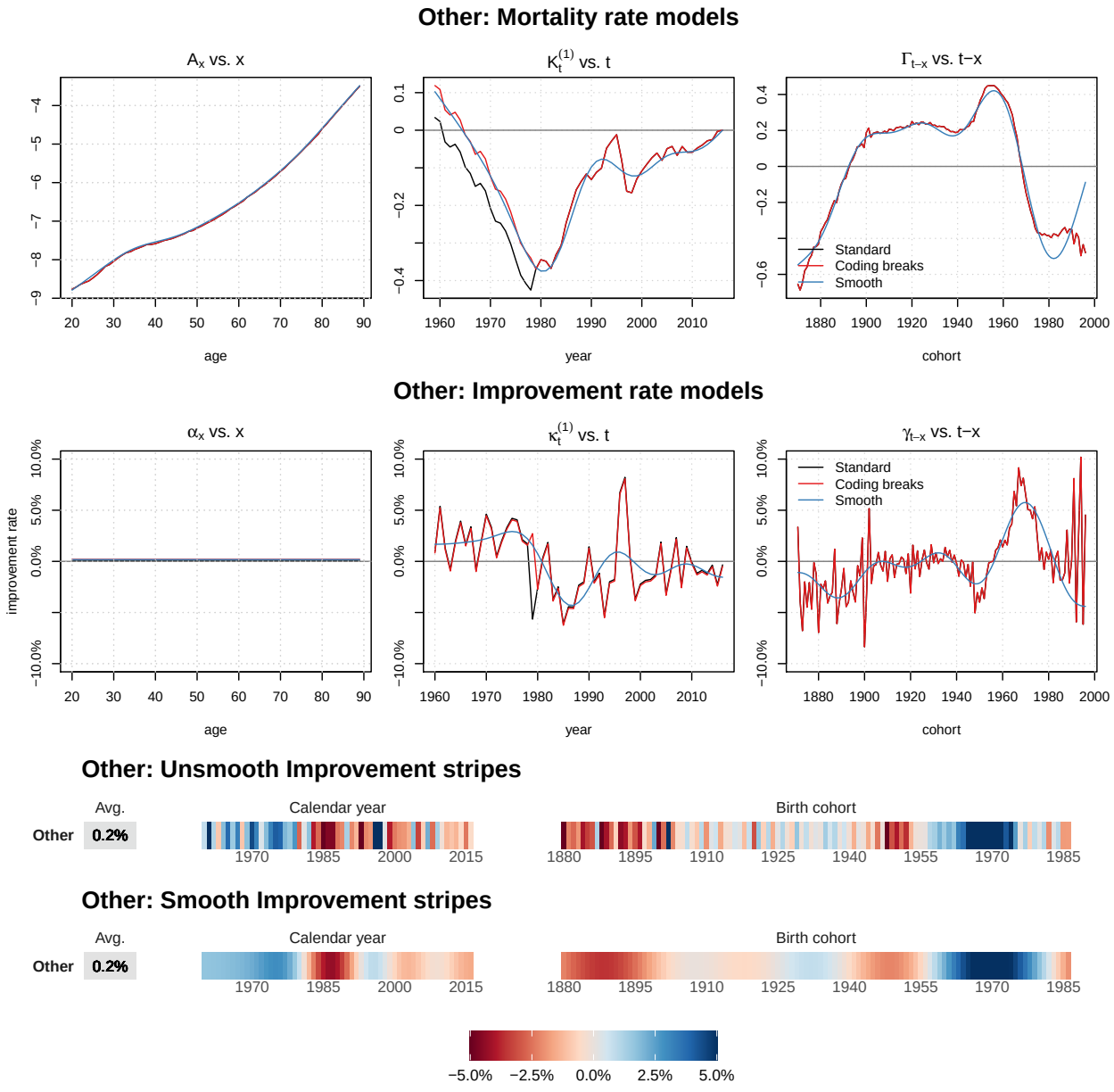


Figure G.26: Fitted parameters for the PCi model for AIDS and tuberculosis, males, 1959–2016, 20–89

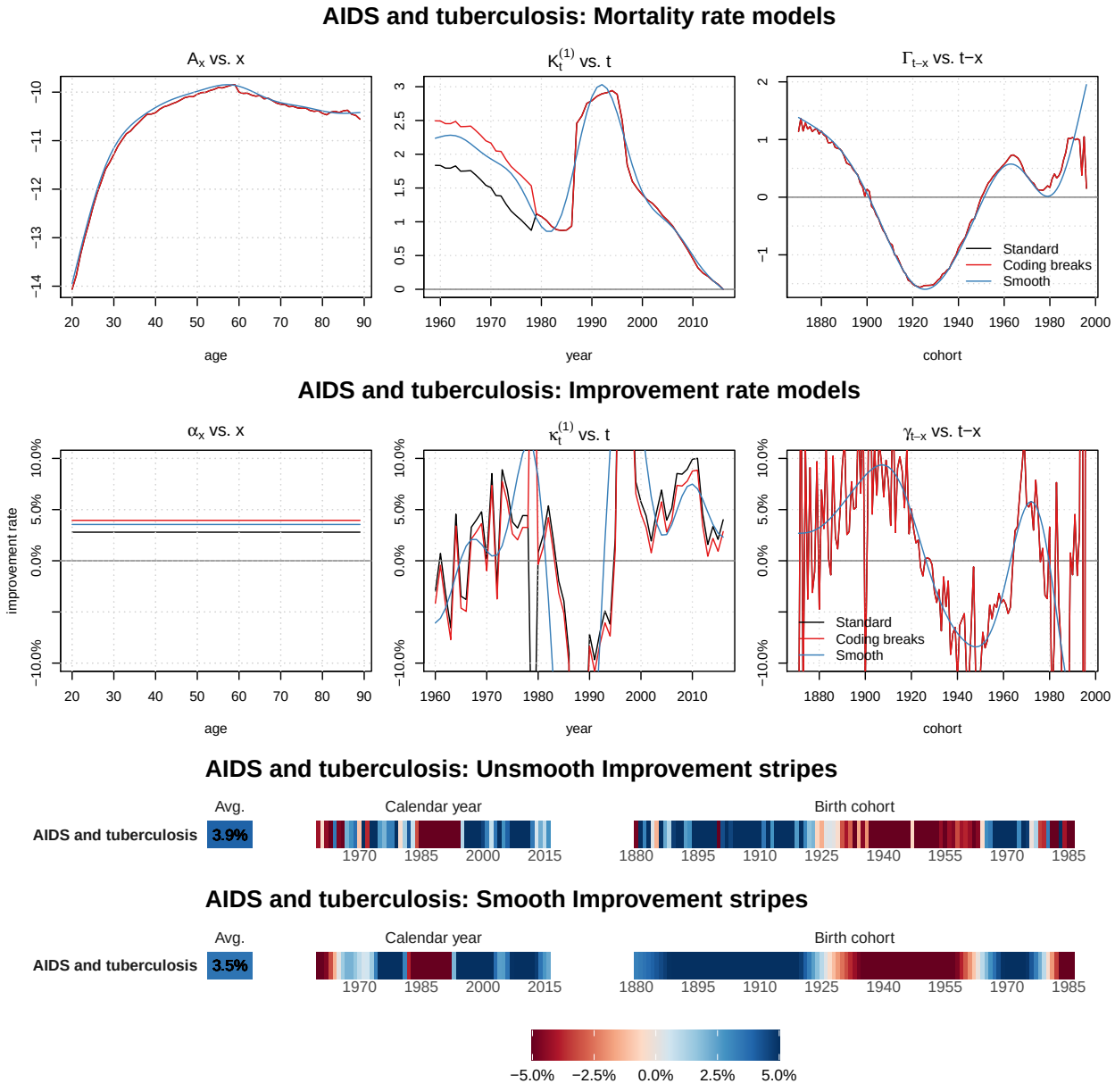


Figure G.27: Fitted parameters for the PCi model for diabetes and obesity, males, 1959–2016, 20–89

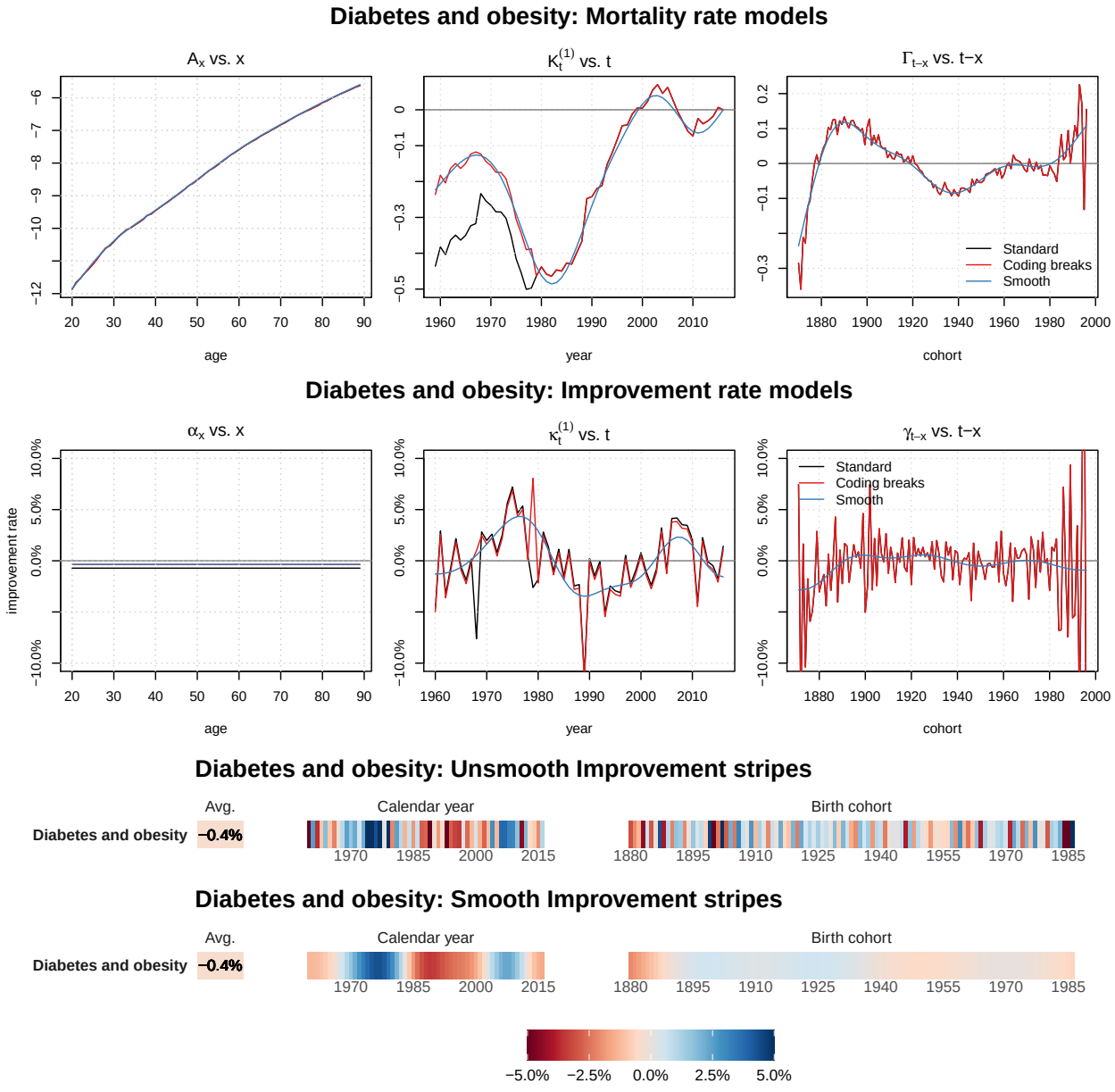


Figure G.28: Fitted parameters for the PCi model for alcohol abuse and drug dependence, males, 1959–2016, 20–89

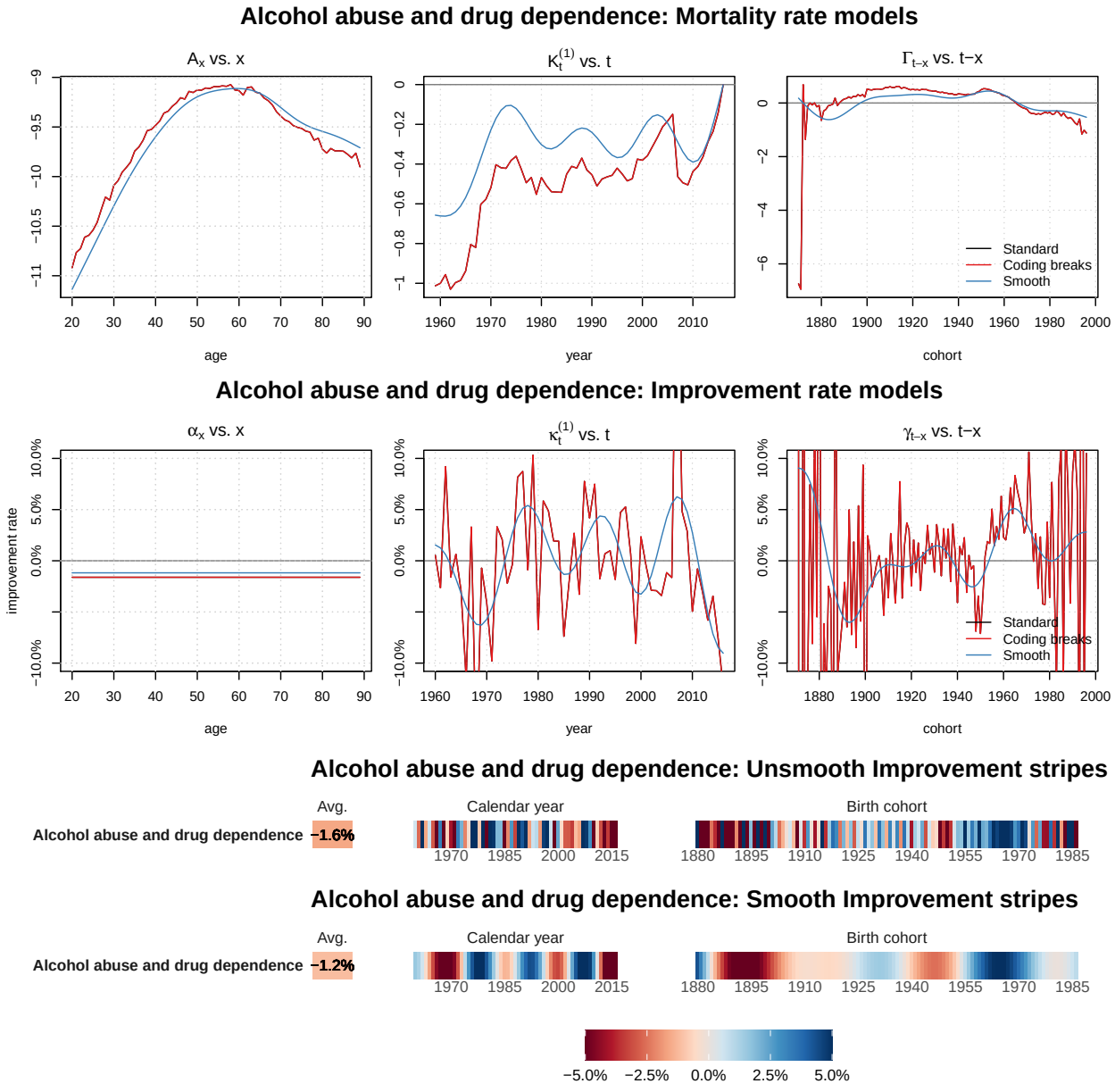


Figure G.29: Fitted parameters for the PCi model for Alzheimer's disease, males, 1979–2016, 20–89

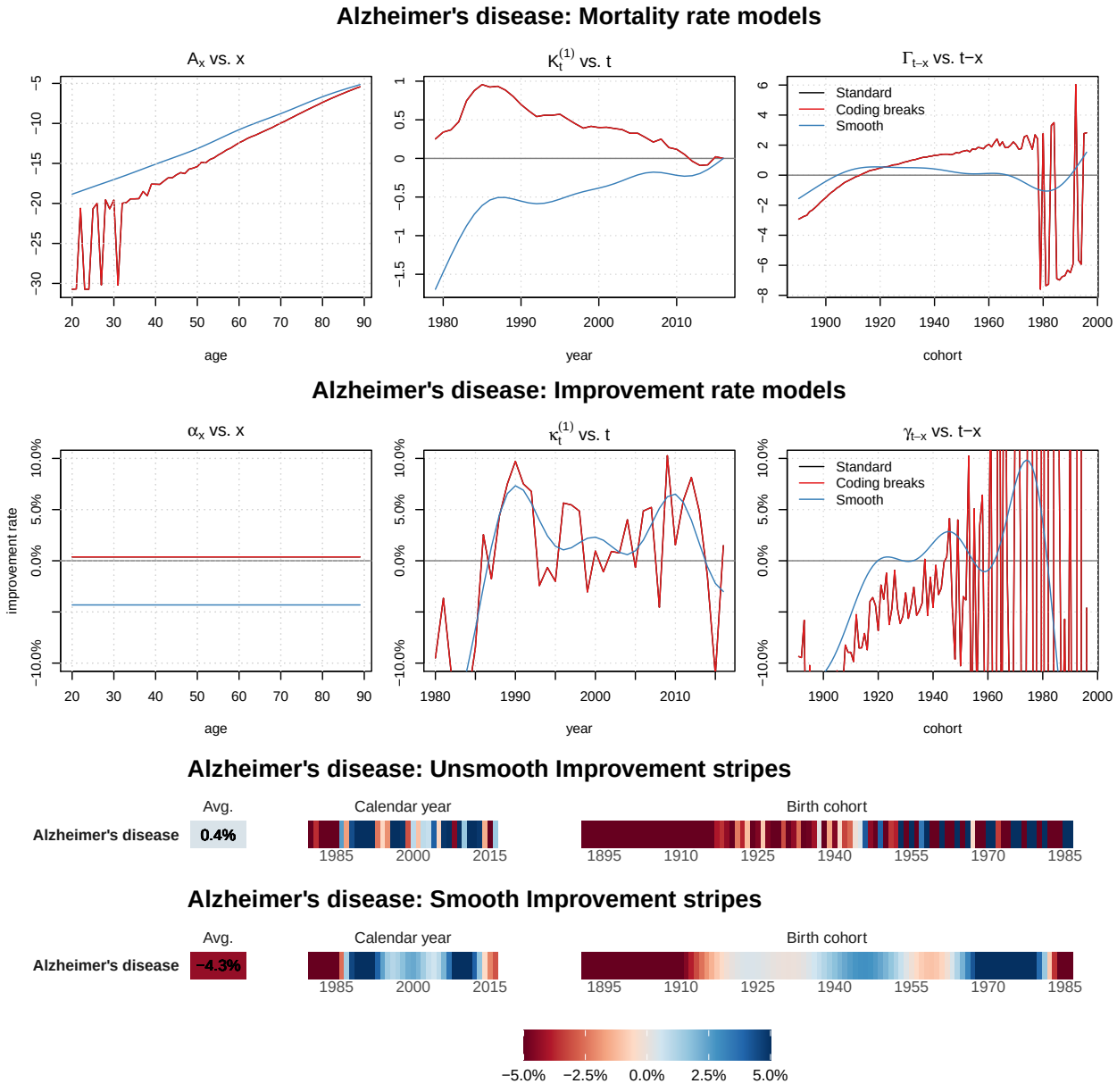


Figure G.30: Fitted parameters for the PCi model for dementia and other mental disorders, males, 1959–2016, 20–89

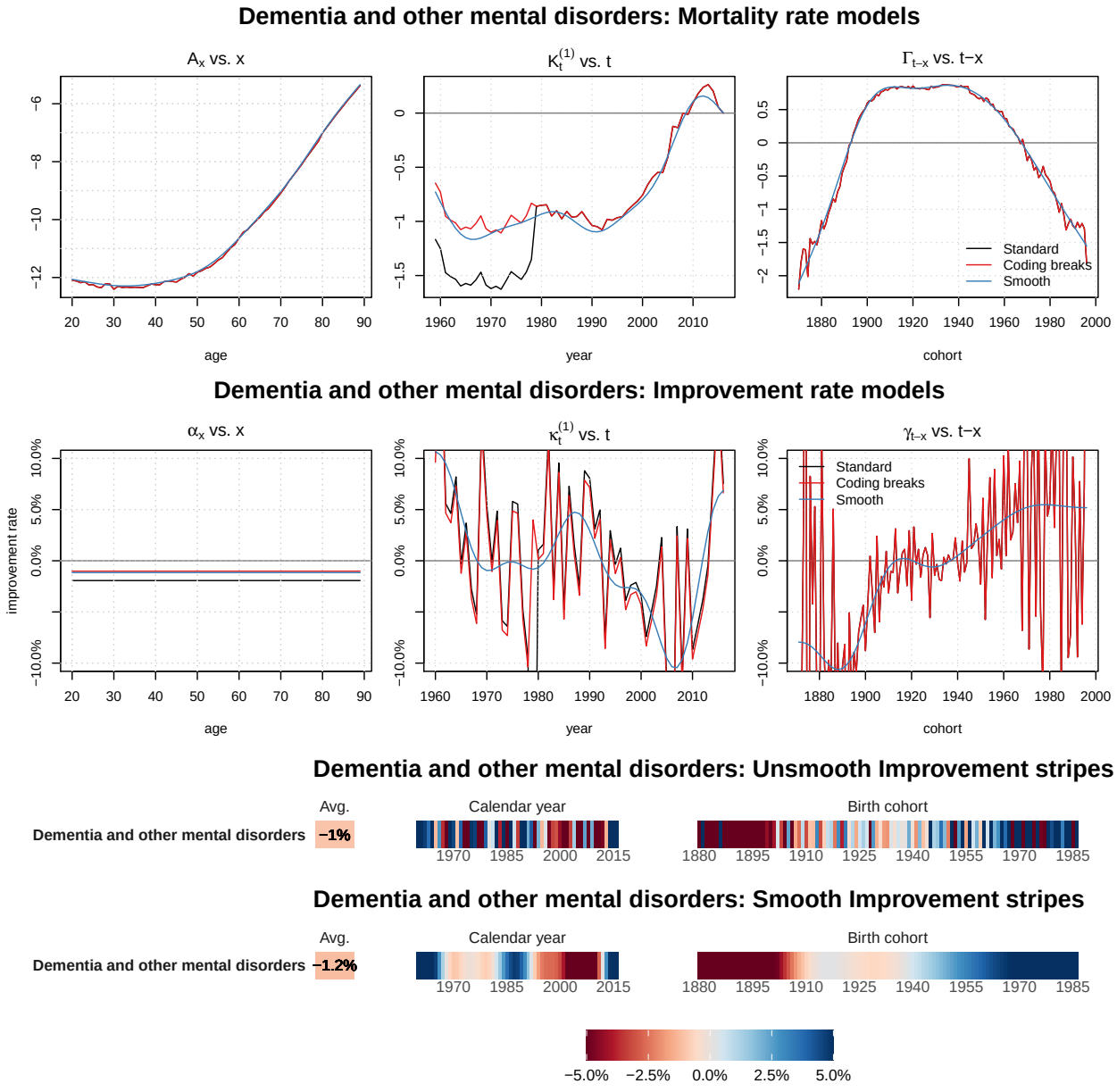


Figure G.31: Fitted parameters for the PCi model for rest of causes of death, males, 1959–2016, 20–89

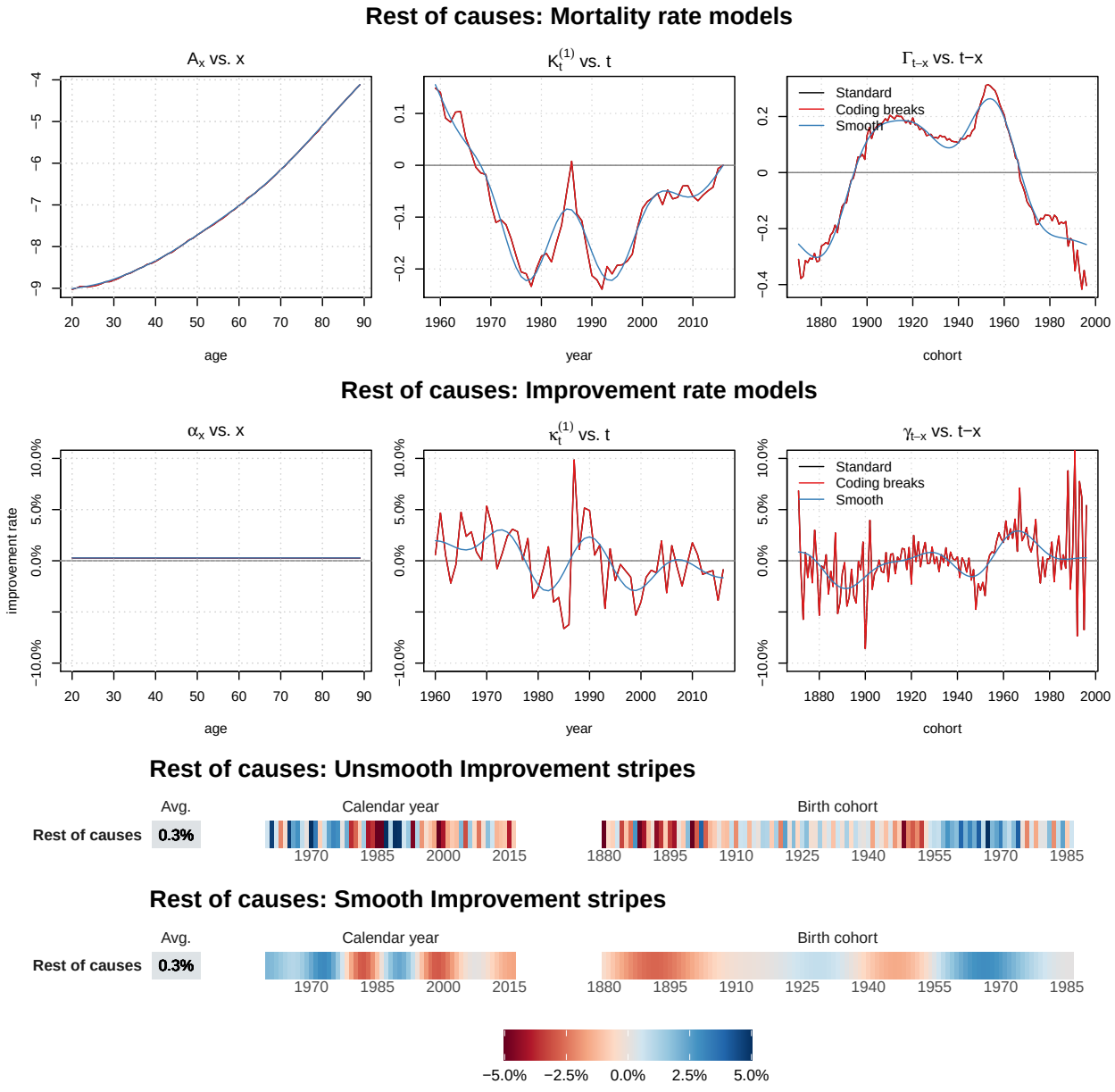


Figure G.32: Fitted parameters for the PCi model for AIDS and tuberculosis, males, 1959–2016, 20–89

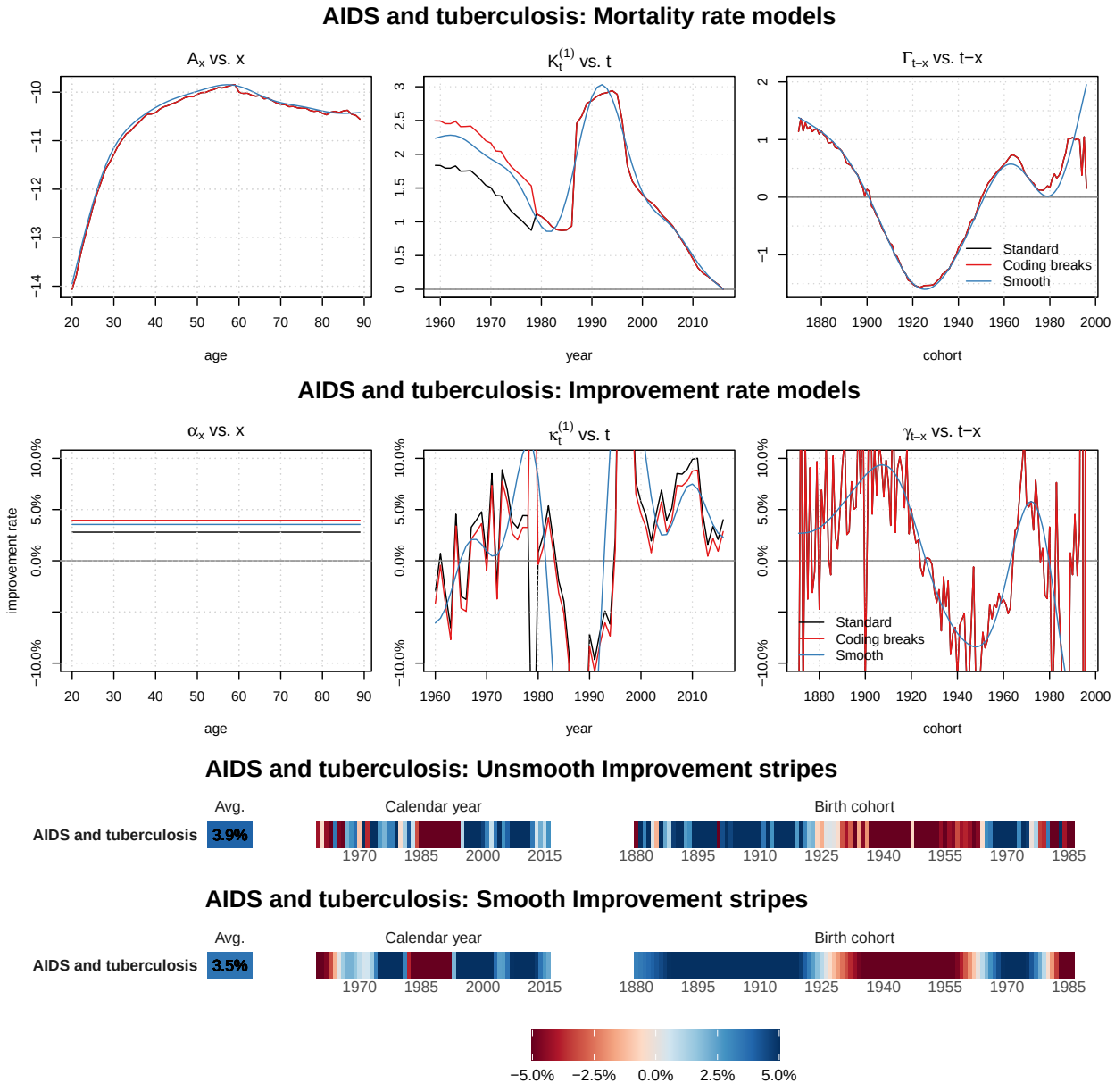


Figure G.33: Fitted parameters for the PCi model for alcohol abuse, males, 1959–2016, 20–89

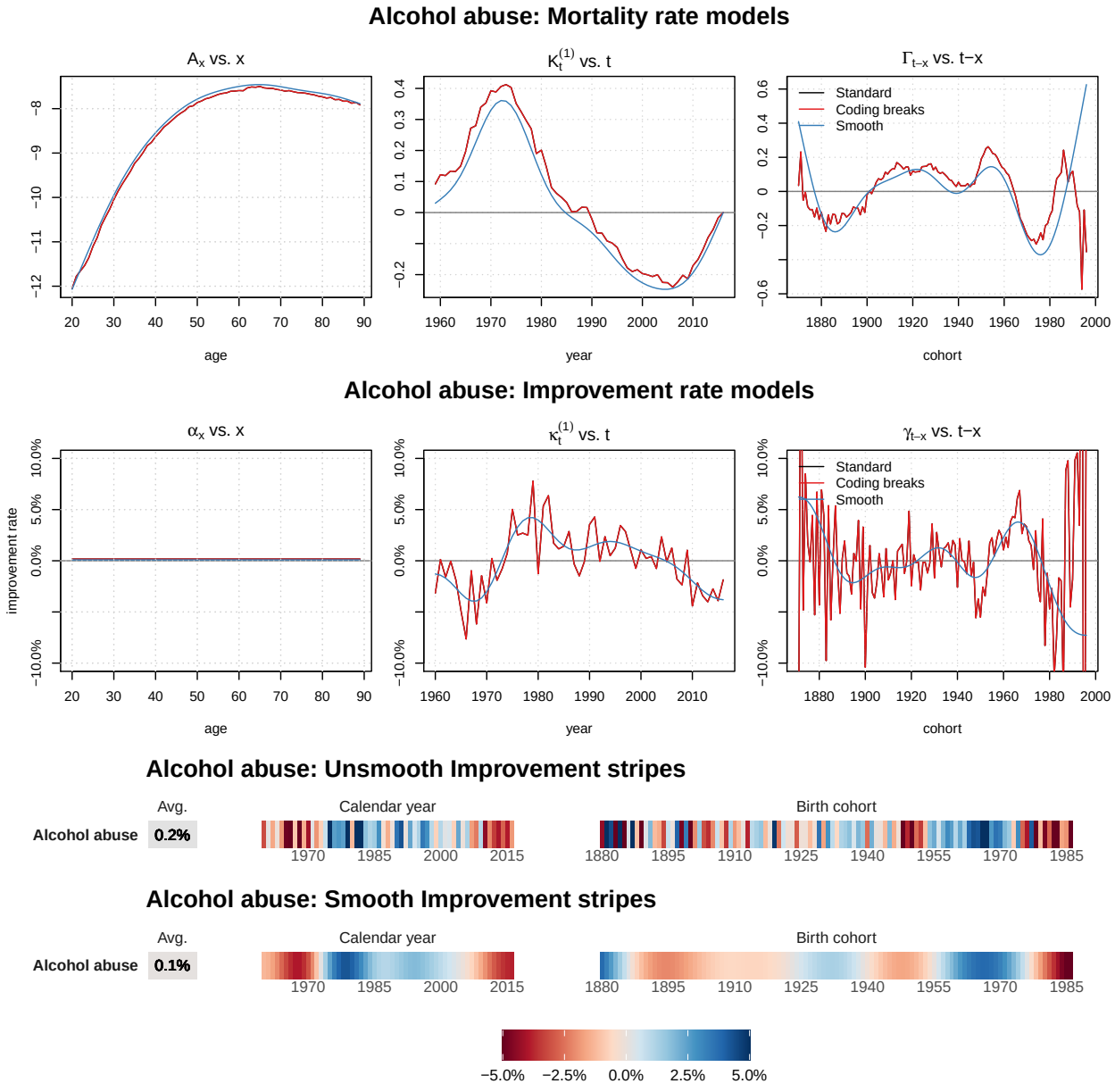


Figure G.34: Fitted parameters for the PCi model for dementia and Alzheimer's disease, males, 1959–2016, 20–89

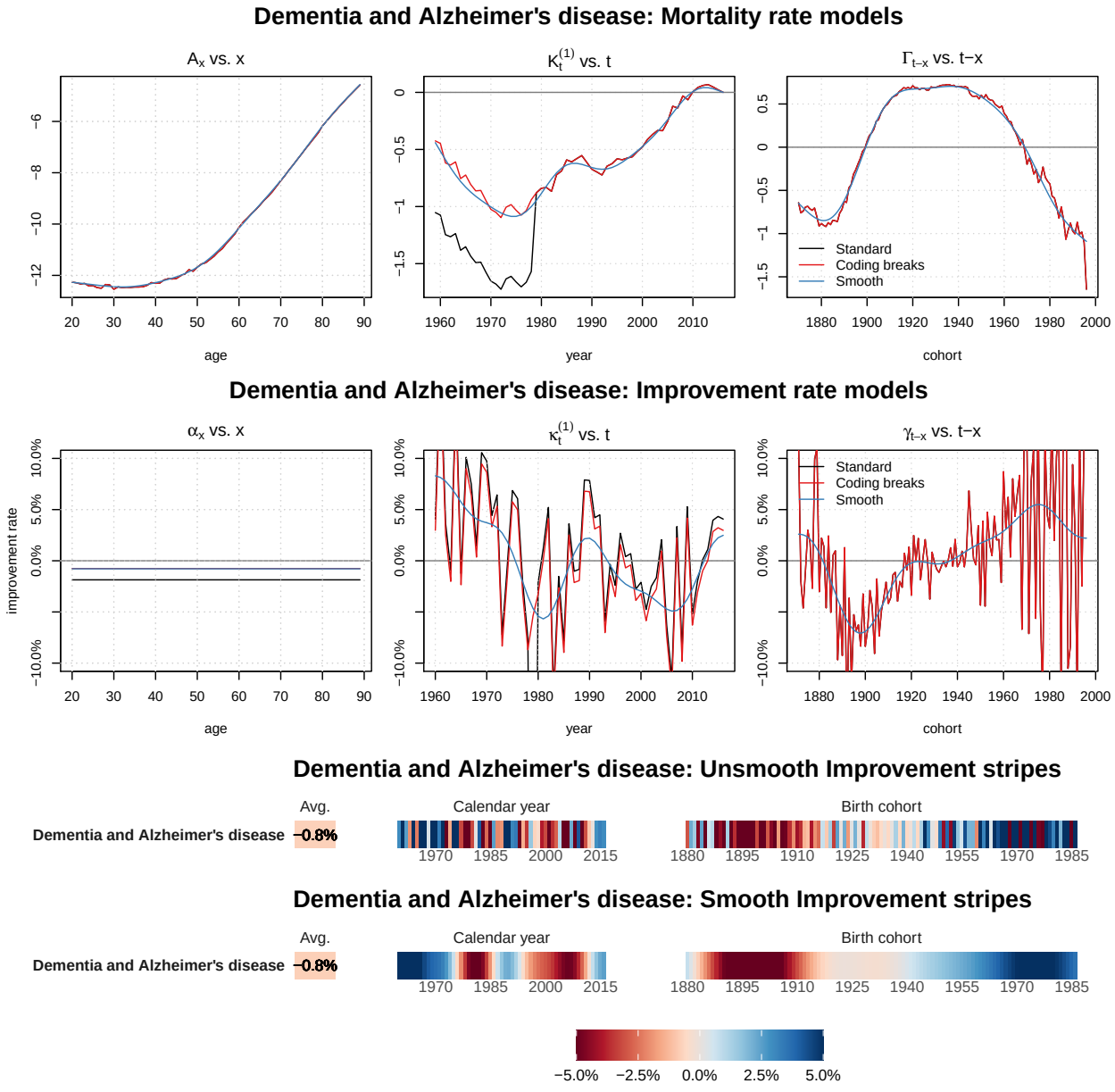


Figure G.35: Fitted parameters for the PCi model for diabetes and obesity, males, 1959–2016, 20–89

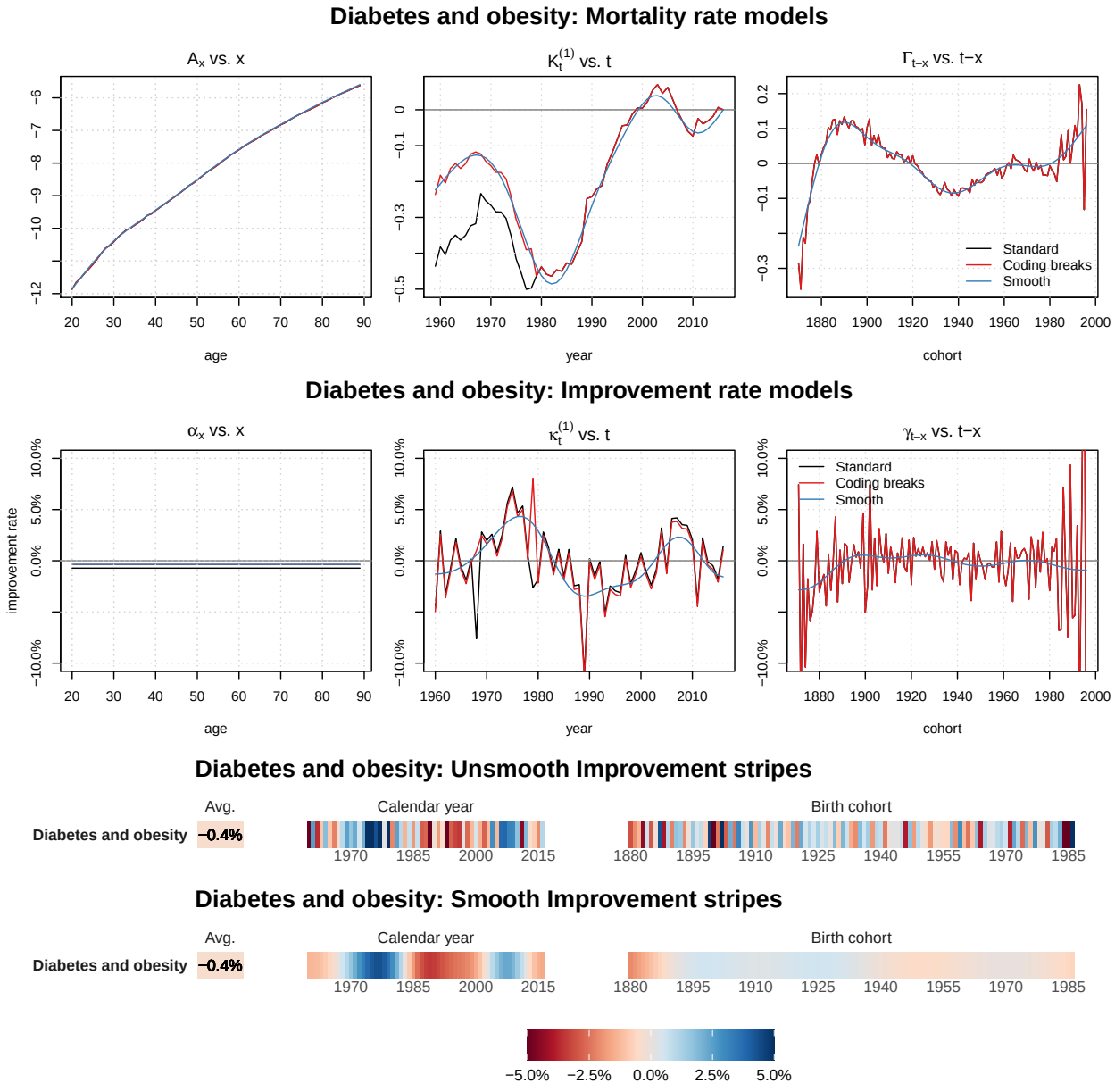


Figure G.36: Fitted parameters for the PCi model for drug dependency, males, 1959–2016, 20–89

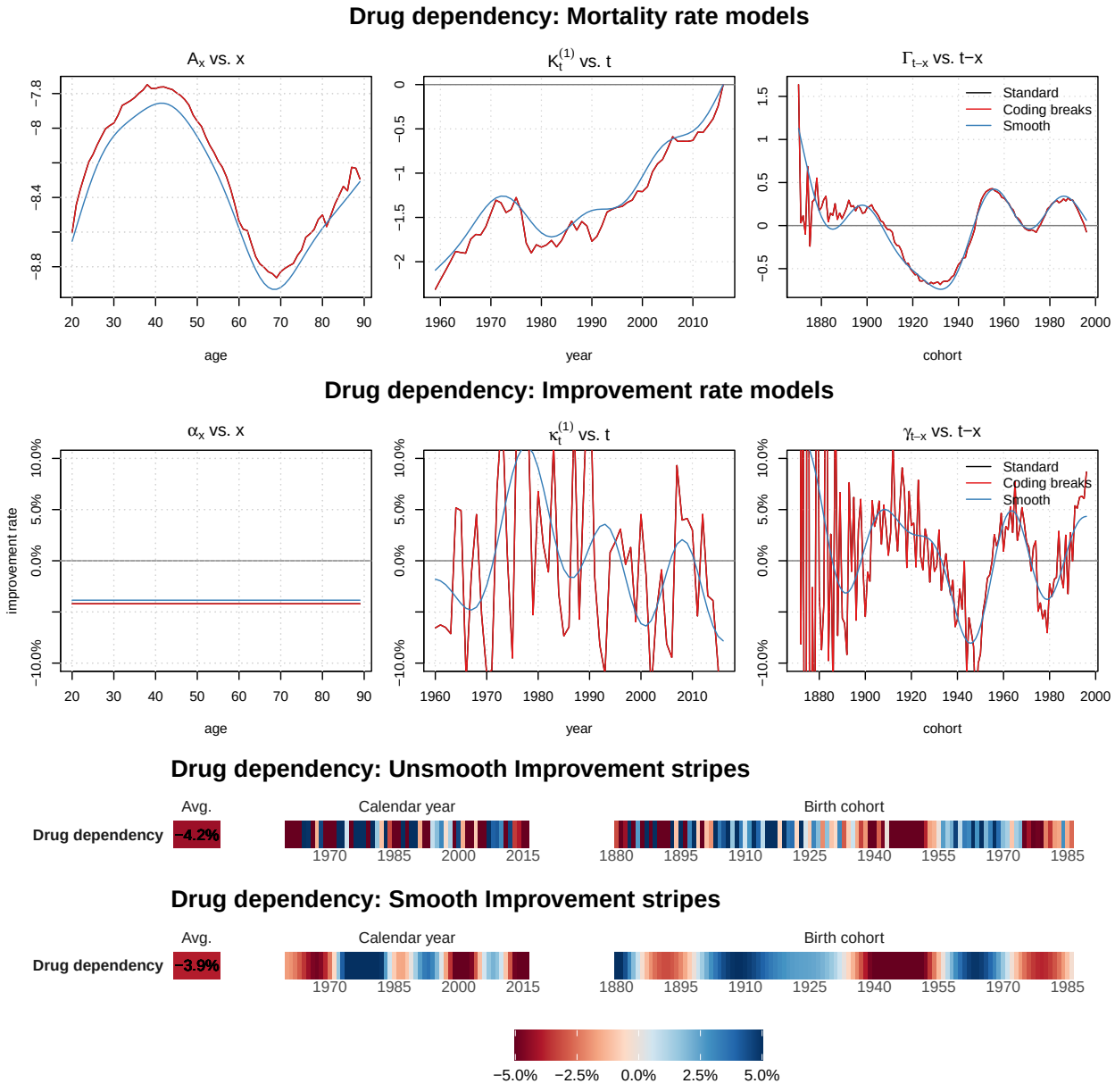


Figure G.37: Fitted parameters for the PCi model for homicide, males, 1959–2016, 20–89

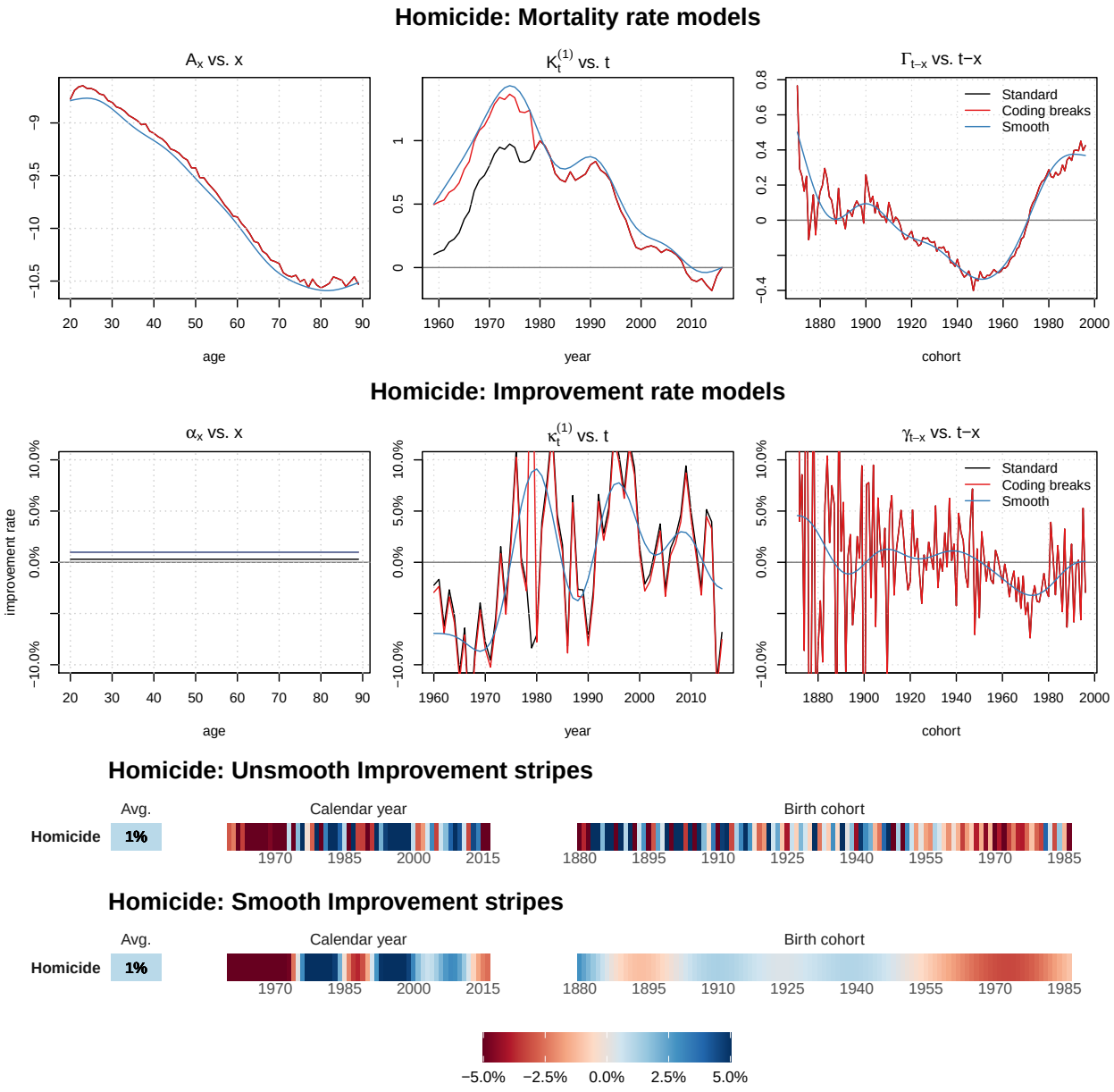


Figure G.38: Fitted parameters for the PCi model for hypertensive disease, males, 1959–2016, 20–89

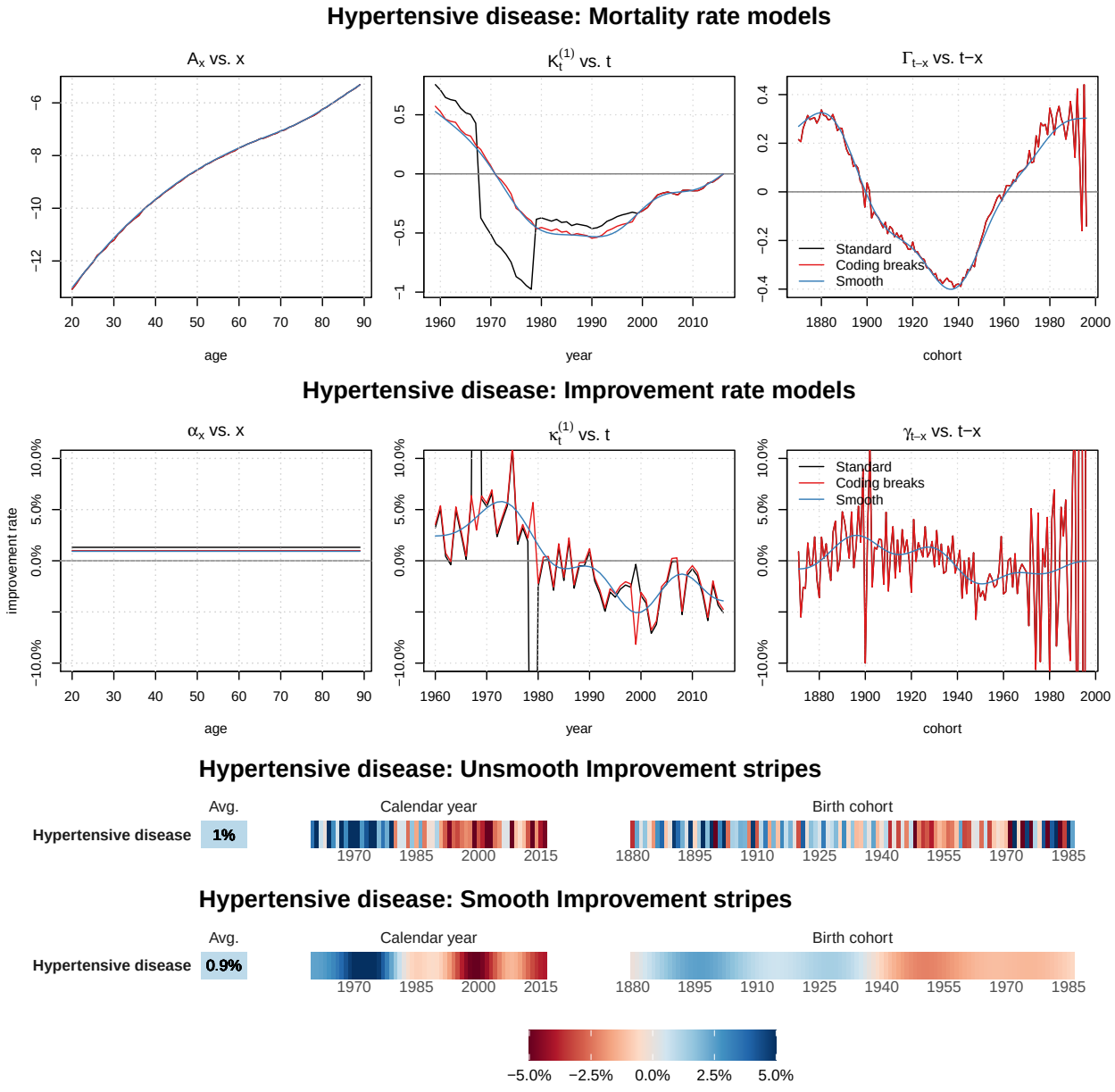


Figure G.39: Fitted parameters for the PCi model for self-harm, males, 1959–2016, 20–89

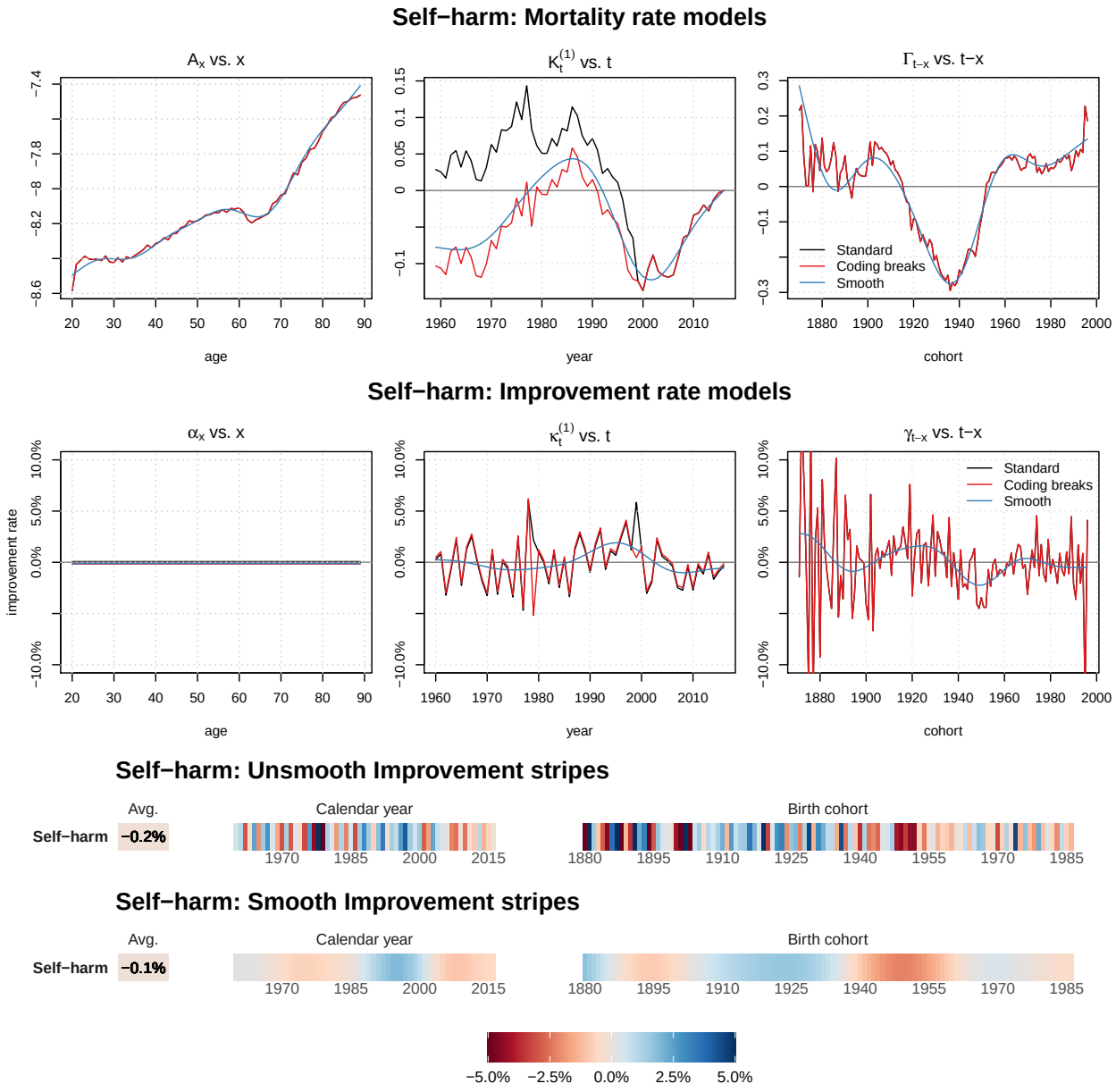
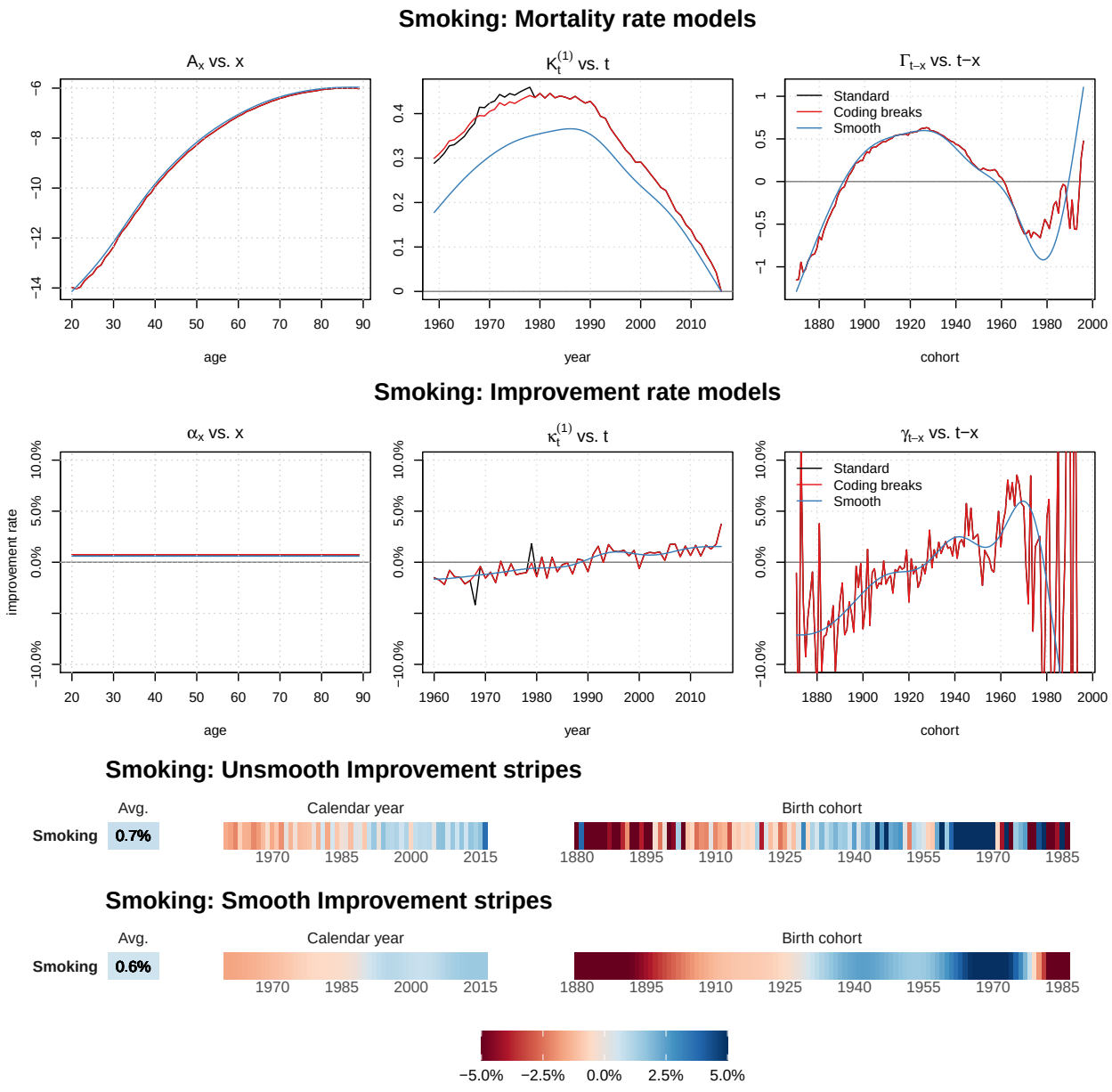


Figure G.40: Fitted parameters for the PCi model for smoking, males, 1959–2016, 20–89



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Society of Actuaries Research Institute
475 N. Martingale Road, Suite 600
Schaumburg, Illinois 60173
www.SOA. org