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AUTHOR

Andrés M Villegas, Ph.D.
School of Risk and Actuarial Studies and ARC Centre of Excellence in Population Ageing Research (CEPAR)
UNSW Sydney

Madhavi Bajekal, Ph.D.,HONFIA
Department of Applied Health Research
University College London

Steven Haberman, Ph.D.
Faculty of Actuarial Science and Insurance Bayes Business School (formerly Cass)
City University of London

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On the decomposition of mortality models into causes of death

Andrés M. Villegas[a], Madhavi Bajekal[b], Steven Haberman[c]

[a]*School of Risk and Actuarial Studies and ARC Centre of Excellence in Population Ageing Research (CEPAR) UNSW Sydney*

[b]*Department of Applied Health Research. University College London*

[c]*Faculty of Actuarial Science and Insurance Bayes Business School (formerly Cass) City University of London*

Abstract

Decomposition methods are a standard tool used by demographers to understand and communicate the contribution of different factors to the evolution of demographic metrics. Despite their widespread use in demography, decomposition methods are less commonly used by actuaries. In this paper we develop a new approach for decomposing mortality improvement rate assumptions into the contribution of different causes of death. In particular, we show how we can decompose a Generalized Age-Period-Cohort mortality improvement rate model into the additive contribution of different causes of death. The methods introduced in this paper are useful for the communication of mortality improvement assumptions to users and decision makers and for shedding light into understanding the drivers of mortality change. We illustrate the decomposition methods using U.S. mortality data by cause of death for the period 1959-2019.

Keywords: Cause of death, age-period-cohort model, mortality modeling, decomposition methods, Cause of death, age-period-cohort model, mortality modeling, decomposition methods

1. Introduction

Most mortality projections are based on the extrapolation of all-cause mortality patterns using methods that leverage the historical stability of the evolution of human mortality (Wilmoth, 2000; Booth and Tickle, 2008; Vaupel et al., 2021). Within this extrapolative paradigm, a preferred approach among actuaries has been the framework of Generalized Age-Period-Cohort (GAPC) models which decompose all-cause mortality trends across the dimensions of age, period and cohort (Villegas et al., 2018; Hunt and Blake, 2020; Hunt and Villegas, 2022).

However, an alternative to projecting all-cause mortality directly is to project mortality rates for separate causes of death and then aggregate them to derive all-cause mortality rates. This approach has been advocated as a means of gaining forecasting accuracy in all-cause mortality projections and obtaining further insight into the drivers of mortality change (Crimmins, 1981; Gutterman and Vanderhoof, 1998). However, such an approach is rarely used due to a number of theoretical and practical challenges including, among others, the difficulty in modeling the dependence structure of the causes (Richards, 2009) and the fact that all-cause mortality models are not necessarily appropriate for cause-specific rates (Di Cesare and Murphy, 2009). Moreover, while it is argued that cause-specific projections should result in more accurate projections of all-cause mortality, past studies tend to indicate that disaggregated forecasts do not result in significant gains in accuracy (Alho, 1991; McNown, 1992) and tend to be more pessimistic than those without disaggregation (Wilmoth, 1995).

To leverage the advantages of all-cause and cause-specific modeling approaches, in this paper we introduce a new approach for decomposing mortality improvement rate assumptions into the contribution of different causes of death. In particular, we show how we can decompose an all-cause Generalized Age-Period-Cohort mortality improvement rate model into the additive contribution of different causes of death. On the one hand, by remaining within the framework of all-cause GAPC models, we can exploit the simplicity that has made this approach very popular

*Corresponding author

Email addresses: a.villegas@unsw.edu.au (Andrés M. Villegas), m.bajekal@ucl.ac.uk (Madhavi Bajekal), s.haberman@city.ac.uk (Steven Haberman)

among academic and practicing actuaries. On the other hand, by introducing a cause of death dimension we can shed light into the understanding of the drivers of mortality change, facilitating the communication of mortality improvement assumptions to users and decision makers.

We illustrate the proposed decomposition approach using U.S. mortality data for the period 1959-2019. The issue of selecting the best model for a given dataset and application is a difficult one for which there might be different answers depending on the application and the subjective considerations of the modeler. Thus, we must stress that our objective is not to find the best model for a dataset (in this case U.S. mortality). Instead, given a GAPC model that a modeler deems appropriate for their application, we aim to provide an additional tool that allows a better understanding of the parameter estimates. Accordingly, we illustrate our decomposition methods using three different GAPC models which have been considered in previous industry-sponsored work, namely, the age-period-cohort improvement rate model (APCi), the period-cohort improvement rate model (PCi), and the Plat model for improvement rates (PLATi). The APCi model has important practical relevance as the model underpinning the CMI mortality projection model used in the United Kingdom (Continuous Mortality Investigation, 2016a,b). The PCi model has been considered by us in Villegas et al. (2021; 2022) as a useful model in understanding the long term drivers of mortality change in the U.S. Finally, the PLATi model has been suggested by Li et al. (2017a; 2017b; 2020) as a potential base for U.S. mortality modelling.

The reminder of this paper is organized as follows. In Section 2 we introduce the main notation we will use through out this paper. In Section 3, we describe the GAPC mortality improvement model decomposition approach we propose. In Section 4, we illustrate this decomposition approach using cause of death mortality data for the U.S. population and the APCi, PCi and PLATi models. Finally, in Section 5 we summarize the key highlights of the paper and discuss areas of future research.

2. Notation and definitions

Let $\mu_{x,t}$ and $m_{x,t}$ denote, respectively, the force of mortality and the central mortality rate at age x in year t. We also have that the (empirical) estimate of the central death rate is $\hat{m}_{x,t} = d_{x,t}/E_{x,t}$, where $d_{x,t}$ and $E_{x,t}$ are, respectively, the number of deaths and central exposed to risk at age x in year t.

We assume that deaths, d_{xt}, and exposures, $E_{x,t}$, are available for ages $x = x_1, x_2, \ldots, x_k$ and years $t = t_1, t_2, \ldots, t_n$. Moreover, we assume data deaths are also available for m distinct causes of death so that $d_{x,t} = \sum_{c=1}^{m} d^c_{x,t}$, where $d^c_{x,t}$ denotes the number of death at age x in year t from cause c, $c = 1, 2, \ldots, m$.

Finally, we define the all-cause mortality improvement rates at age x in year t as

$$i_{x,t} = -\frac{1}{\mu_{x,t}} \frac{\partial \mu_{x,t}}{\partial t} = -\frac{\partial \log \mu_{x,t}}{\partial t}. \tag{1}$$

3. Decomposition of mortality projection models into causes of death

3.1. Generalized Age-Period-Cohort improvement rate models

In order to model mortality improvement rates we consider the general structure of Generalized Age-Period-Cohort (GAPC) improvement rate models discussed in Hunt and Villegas (2022), and which adapts to the context of improvement rates the widely used GAPC mortality rate model structure (Villegas et al., 2018; Hunt and Blake, 2020). The GAPC modeling framework encompasses popular mortality modeling approaches such as the standard age-period-cohort model (Hobcraft et al., 1982), the Cairns-Blake-Dowd model (Cairns et al., 2006, 2009) and the Plat model (Plat, 2009).

GAPC improvement rate models decompose the evolution of mortality improvement rates into the contributions of age, x, period (or calendar year), t, and cohort (or year of birth), $t - x$, using the following specification:

$$i_{x,t} = \alpha_x + \sum_{i=1}^{N} f^{(i)}(x)\kappa^{(i)}_t + \gamma_{t-x}, \tag{2}$$

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;
- $f^{(i)}(x)$ are pre-specified age functions which modulate the corresponding period functions; and
- γ_{t-x} is a cohort function describing systematic differences in the rate of improvement which depend upon a cohort's year of birth, $t - x$.

Within a GAPC model, period and cohort effects aim to capture complementary aspects of how environmental and social changes drive the evolution of mortality. On the one hand, period effects capture contemporary effects such the continuous progress in medical treatment or the discrete short-term impact of an epidemic or a war. On the other hand, cohort effects capture mortality changes linked to the societal norms associated with belonging to a particular generation or the possible long-lasting effects of being born or brought up during exceptional circumstances such as an epidemic or a war (Bell, 2020)

Our main objective is to add a cause of death dimension to the GAPC structure in Equation 2, so that we can assess the contribution of different causes of death to the age, period and cohort components of a GAPC improvement rate model.

3.2. Decomposition of mortality improvement rates

To achieve this objective, we first start by decomposing the improvement rate $i_{x,t}$ in the left-hand side of Equation 2 into the additive contributions of the m different causes of death.

The practical implementation of a GAPC improvement rate models requires the specification of a numerical approximation to evaluate the partial derivative in the definition of improvement rates in Equation 1. For example, Mitchell et al. (2013) and Hunt and Villegas (2022) use the approximation

$$i_{x,t} \approx -\log\left(\frac{m_{x,t}}{m_{x,t-1}}\right) \tag{3}$$

which uses a backward difference approximation of the partial derivative

$$i_{x,t} = -\frac{\partial \log \mu_{x,t}}{\partial t}.$$

By contrast, Haberman and Renshaw (2012) use the approximation

$$i_{x,t} \approx 2\frac{m_{x,t-1} - m_{x,t}}{m_{x,t-1} + m_{x,t}} \tag{4}$$

which relies on a central difference approximation of the partial derivative

$$i_{x,t} = -\frac{1}{\mu_{x,t}}\frac{\partial \mu_{x,t}}{\partial t}.$$

Here, we take a slightly different approach and note that we can write

$$i_{x,t} = -\frac{\partial \log \mu_{x,t}}{\partial t} \approx -\frac{\partial \log \frac{d_{x,t}}{E_{x,t}}}{\partial t} = -\frac{\partial \log d_{x,t}}{\partial t} + \frac{\partial \log E_{x,t}}{\partial t}.$$

That is, mortality improvements can be decomposed into a term driven by the time change in deaths, $-\frac{\partial \log d_{x,t}}{\partial t}$, and another one driven by the time change in exposures, $\frac{\partial \log E_{x,t}}{\partial t}$. Then, similar to Haberman and Renshaw (2012), we can use a central differences approximation of the derivatives to obtain

$$
\begin{aligned}
i_{x,t} &\approx -\frac{\partial \log d_{x,t}}{\partial t} + \frac{\partial \log E_{x,t}}{\partial t} \\
i_{x,t} &\approx -\frac{1}{d_{x,t}}\frac{\partial d_{x,t}}{\partial t} + \frac{1}{E_{x,t}}\frac{\partial E_{x,t}}{\partial t} \\
i_{x,t} &\approx 2\frac{d_{x,t-1} - d_{x,t}}{d_{x,t-1} + d_{x,t}} + 2\frac{E_{x,t} - E_{x,t-1}}{E_{x,t-1} + E_{x,t}}. \qquad (5)
\end{aligned}
$$

In practice, approximations (3), (4) and (5) produce essentially the same numerical estimates of mortality improvement rates. However, approximation (5) has the advantage of allowing a direct decomposition of $i_{x,t}$ into the additive contribution of different causes of death. More specifically, noting that $d_{x,t} = \sum_{c=1}^{m} d^c_{x,t}$, we have

$$
\begin{aligned}
i_{x,t} &\approx 2\frac{d_{x,t-1} - d_{x,t}}{d_{x,t-1} + d_{x,t}} + 2\frac{E_{x,t} - E_{x,t-1}}{E_{x,t-1} + E_{x,t}} \\
&= 2\frac{\sum_{c=1}^{m}\left(d^c_{x,t-1} - d^c_{x,t}\right)}{d_{x,t-1} + d_{x,t}} + 2\frac{E_{x,t} - E_{x,t-1}}{E_{x,t-1} + E_{x,t}} \\
&= \sum_{c=1}^{m} 2\left(\frac{d^c_{x,t-1} - d^c_{x,t}}{d_{x,t-1} + d_{x,t}} + \frac{d^c_{x,t-1} + d^c_{x,t}}{d_{x,t-1} + d_{x,t}} \times \frac{E_{x,t} - E_{x,t-1}}{E_{x,t-1} + E_{x,t}}\right) \qquad (6) \\
i_{x,t} &\approx \sum_{c=1}^{m} i^c_{x,t}, \qquad (7)
\end{aligned}
$$

where

$$
i^c_{x,t} = 2\left(\frac{d^c_{x,t-1} - d^c_{x,t}}{d_{x,t-1} + d_{x,t}} + \frac{d^c_{x,t-1} + d^c_{x,t}}{d_{x,t-1} + d_{x,t}} \times \frac{E_{x,t} - E_{x,t-1}}{E_{x,t-1} + E_{x,t}}\right) \qquad (8)
$$

is the contribution of cause c to mortality improvements at age x in year t.

3.3. Decomposition of GAPC improvement rate models

Having decomposed the left-hand side of Equation 2, we now turn our attention to the decomposition of the right-hand side of Equation 2 into the additive contributions of the different causes of death. For this, we assume that the contribution of cause c to mortality improvements at age x in year t, $i^c_{x,t}$, follows a GAPC model with the same structure to the GAPC model for the all-cause mortality improvement, $i_{x,t}$. That is,

$$
i^c_{x,t} = \alpha^c_x + \sum_{i=1}^{N} f^{(i)}(x)\kappa^{(i),c}_t + \gamma^c_{t-x}, \qquad (9)
$$

where α^c_x is a static function that captures the age shape of mortality improvements from cause c, $\kappa^{(i),c}_t$ are period parameters that capture period effects from cause c, modulated by $f^{(i)}(x)$, and γ^c_{t-x} captures cohort effects from cause c.

Adding across the m different causes of death, we have that the all-cause improvements are given by

$$
i_{x,t} = \sum_{c=1}^{m} \alpha^c_x + \sum_{i=1}^{N} f^{(i)}(x)\left(\sum_{c=1}^{m} \kappa^{(i),c}_t\right) + \sum_{c=1}^{m} \gamma^c_{t-x}, \qquad (10)
$$

and

$$
i_{x,t} = \tilde{\alpha}_x + \sum_{i=1}^{N} f^{(i)}(x)\tilde{\kappa}^{(i)}_t + \tilde{\gamma}_{t-x}, \qquad (11)
$$

with

$$\tilde{\alpha}_x = \sum_{c=1}^{m} \alpha_x^c, \quad \tilde{\kappa}_t^{(i)} = \sum_{c=1}^{m} \kappa_t^{(i),c}, \quad \text{and} \quad \tilde{\gamma}_{t-x} = \sum_{c=1}^{m} \gamma_{t-x}^c. \tag{12}$$

The question is then to find under what circumstances would Equation 2 be equivalent to Equation 11, that is, when would it hold that $\tilde{\alpha}_x = \alpha_x$, $\tilde{\kappa}_t^{(i)} = \kappa_t^{(i)}$, and $\tilde{\gamma}_{t-x} = \gamma_{t-x}$. Interestingly, Theorem 1 below states that if the GAPC model parameters are fitted using weighted least squares at the cause of death level in (9) and are then aggregated to the all-cause level using (10)-(12), then they coincide with the parameters of a GAPC model fitted directly to the all-cause data using (2).

Theorem 1. *Assume that we estimate the parameters in Equation 2 using weighted least squares as follows*

$$(\hat{\alpha}_x, \hat{\kappa}_t^{(i)}, \hat{\gamma}_{t-x}) = \text{argmin} \sum_x \sum_t w_{x,t} \left(i_{x,t} - \alpha_x - \sum_{i=1}^{N} f^{(i)}(x) \kappa_t^{(i)} - \gamma_{t-x} \right)^2,$$

where $w_{x,t}$ is the weight at age x and year t. Assume also that we estimate the parameters in Equation 9 using weighted least squares as follows

$$(\hat{\alpha}_x^c, \hat{\kappa}_t^{(i),c}, \hat{\gamma}_{t-x}^c) = \text{argmin} \sum_x \sum_t w_{x,t} \left(i_{x,t}^c - \alpha_x^c - \sum_{i=1}^{N} f^{(i)}(x) \kappa_t^{(i),c} - \gamma_{t-x}^c \right)^2.$$

We then have that

$$\hat{\alpha}_x = \sum_{c=1}^{m} \hat{\alpha}_x^c, \quad \hat{\kappa}_t^{(i)} = \sum_{c=1}^{m} \hat{\kappa}_t^{(i),c}, \quad \hat{\gamma}_{t-x} = \sum_{c=1}^{m} \hat{\gamma}_{t-x}^c.$$

Proof. See Appendix A. □

Thus, if model estimation is done using weighted least squares, Theorem 1 implies that the age, period and cohort components of an all-cause GAPC mortality improvement rate model can be decomposed into the additive contribution of the m different causes.

Finally, to implement the cause of death decomposition of GAPC improvement rate models we are only left with specifying appropriate weights $w_{x,t}$. For our implementation we choose $w_{x,t} = d_{x,t-1} + d_{x,t}$ as under a Poisson assumption for the number of deaths, we have that

$$\text{Var}\left(i_{x,t}\right) = \text{Var}\left(2\frac{D_{x,t-1} - D_{x,t}}{D_{x,t-1} + D_{x,t}} + 2\frac{E_{x,t} - E_{x,t-1}}{E_{x,t-1} + E_{x,t}}\right) = \text{Var}\left(2\frac{D_{x,t-1} - D_{x,t}}{D_{x,t-1} + D_{x,t}}\right) \approx \frac{1}{d_{x,t-1} + d_{x,t}}. \tag{13}$$

This choice of estimation weights implies that ages and years with more observed deaths have a higher influence on the parameter estimates.

4. Empirical application to U.S. mortality improvements

In this section we illustrate the GAPC mortality improvement model decomposition approach using cause of death mortality data for the U.S. population for years 1959 to 2019 and ages 20 to 89. This will allow us to understand the key drivers of mortality improvement in the U.S. and shed light on how these drivers influence mortality improvement rate assumptions.

4.1. Data

Our data comprises exposed to risk obtained from the Human Mortality Database (2022) and death counts by cause of death for U.S. males and females aged 20 to 89 for years 1959 to 2019. Our death counts by cause of death come from three sources:

i. Death counts for the period 1959-1978 obtained from the Human Mortality Database Cause of Death Data, which is an SOA-sponsored initiative to develop a cause of death extension of the Human Mortality Database (2022) and which is described in Barbieri (2017).
ii. Death counts for the period 1979-2016 obtained from a preliminary version of the Human Cause of Death Database[1] for the period 1979-2016. These data uses bridge coding to adjust for cause of death coding changes between ICD-9 and ICD-10.
iii. Death counts for the period 2017-2019 obtained from the National Center for Health Statistics (NCHS) mortality microdata.[2][3]

We have previously analyzed the data for the period 1959-2016 as part of an SOA-funded project in which we have integrated cause of death modeling with epidemiological evidence to identify significant mortality drivers in the U.S. population (Villegas et al., 2021, 2022). Consistent with this previous study, we consider a two-level grouping of causes of death as summarized in Table 1. Level 1 comprises six broad causes of death including circulatory diseases, neoplasms, respiratory diseases, digestive system diseases, external causes and other causes. In contrast, the more granular Level 2 is formed by 26 causes of deaths which provide a more detailed view each of the six Level 1 causes.

Table 1 shows a changing composition of the causes of death for Americans aged 20 to 89: while in 1959 about 59% of deaths were from circulatory diseases, in 2019 this percentage has halved to only about 30% for males and 28% for females.

4.2. Models

To illustrate our decomposition methodology, we consider three GAPC improvement rate models, namely, the APCi model, the PCi model, and the PLATi model.

The APCi model given by

$$i_{x,t} = \alpha_x + \kappa_t^{(1)} + \gamma_{t-x}$$

is the improvement rate version of the classical APC model for mortality rates. This model underpins the current mortality projection model proposed by the Continuous Mortality Investigation (2016a; 2016b) in the United Kingdom and, as such, has attracted significant attention among academics (see, e.g., Richards et al. (2019), Hilton et al. (2019) and Dodd et al. (2020)).

The PCi model given by

$$i_{x,t} = \alpha + \kappa_t^{(1)} + \gamma_{t-x}$$

is a constrained version of the APCi model which assumes that $\alpha_x \equiv \alpha$, so that the age specific component of mortality improvement is replaced by a constant component which applies to all ages. This model has been considered in Villegas et al. (2021; 2022) as an alternative that mitigates some of the identification issues inherent in age-period-cohort modeling (Bell and Jones, 2013).

Finally, the PLATi model given by

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

where a $\bar{x}$ is the mean of the ages in the data, corresponds to the simplified model of Plat (2009), in which the term $(x - \bar{x})\kappa_t^{(2)}$ captures possible differences in the evolution of mortality among younger and older ages. This model has recently been suggested as an appropriate model for all-cause mortality trends for the U.S. populations aged 20 to 95 (Li et al., 2017a,b, 2020).

As with most GAPC models, the APCi, PCi and PLATi models are only identifiable up to a set of transformations. Thus, they require the imposition of identification constraints to ensure uniqueness of the model parameters (Villegas

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

[2] These data is available at https://www.nber.org/research/data/mortality-data-vital-statistics-nchs-multiple-cause-death-data.

[3] We note that the NCHS Public Use Mortality microdata may involve a reported age that is affected by various forms of age misstatement in a way that the Human Mortality Database data on deaths by age for many years are not. This may cause some discrepancies between the total number of deaths in our data compared to those in the Human Mortality Database. Reassuringly, these discrepancies are generally small and do not have a material impact on our analysis and improvement rate estimates.

Table 1: Cause of deaths groupings and percentage of deaths per cause for ages 20 to 89 for selected years

		% of deaths ages 20 to 89			
		Males		Females	
Level 1	**Level 2**	**1959**	**2019**	**1959**	**2019**
Circulatory diseases		**58.35**	**29.95**	**60.07**	**27.62**
	Ischaemic heart disease	34.80	14.39	27.36	10.02
	CVD and stroke	10.48	4.18	15.22	5.63
	Other circulatory system diseases	13.06	11.38	17.49	11.97
Neoplasms		**16.49**	**23.54**	**19.51**	**25.38**
	Bowel cancer	2.23	2.05	3.16	2.09
	Liver cancer	0.57	1.55	0.90	1.04
	Lung cancer	3.54	5.60	0.81	5.86
	Breast cancer	0.02	0.03	3.69	3.71
	Prostate cancer	1.65	2.08	0.00	0.00
	Other cancers	4.95	8.42	8.46	9.79
	Other digestive organ cancers	3.53	3.79	2.50	2.89
Respiratory diseases		**4.99**	**8.92**	**3.58**	**10.68**
	Influenza and pneumonia	2.71	1.56	2.60	1.70
	Chronic lower respiratory disease	1.66	5.11	0.66	6.74
	Other respiratory diseases	0.63	2.24	0.32	2.24
Digestive system		**4.08**	**4.38**	**3.57**	**4.33**
	Gastric and duodenal ulcer	0.97	0.13	0.43	0.14
	Chronic liver disease	1.52	2.17	1.06	1.53
	Other digestive system diseases	1.58	2.08	2.08	2.66
External causes		**8.14**	**12.33**	**4.24**	**6.34**
	Traffic accidents	2.63	1.22	1.16	0.52
	Self-harm and interpersonal violence	2.35	3.73	0.95	1.22
	Other external causes	3.17	7.38	2.12	4.59
Other		**7.95**	**20.89**	**9.03**	**25.65**
	AIDS and tuberculosis	0.97	0.32	0.49	0.14
	Diabetes and obesity	1.38	3.94	2.80	3.57
	Alcohol abuse and drug dependence	0.27	0.86	0.08	0.40
	Alzheimer's disease	0.00	2.07	0.00	4.49
	Dementia and other mental disorders	0.06	2.15	0.11	3.85
	Rest of causes	5.27	11.55	5.55	13.20

et al., 2018). Accordingly, to estimate the APCi model we impose the constraints

$$\sum_{t=t_1}^{t_n} \kappa_t^{(1)} = 0, \quad \sum_{y=t_1-x_k}^{t_n-x_1} \gamma_y = 0, \quad \sum_{y=t_1-x_k}^{t_n-x_1} y\gamma_y = 0.$$

These constraints remove linear trends from the cohort component ensuring that α_x can be interpreted as average age-specific improvements over the study period and that temporal trends in improvement rates are mainly reflected in the period component, $\kappa_t^{(1)}$.

Similarly, for the PLATi we also remove linear trends from the cohort component model by imposing

$$\sum_{y=t_1-x_k}^{t_n-x_1} \gamma_y = 0, \quad \sum_{y=t_1-x_k}^{t_n-x_1} y\gamma_y = 0,$$

so that the γ_{t-x} only captures residual mortality improvements not already captured by the period components, $\kappa_t^{(1)}$ and $\kappa_t^{(2)}$.

Finally, for the PCi model we impose

$$\sum_{t=t_1}^{t_n} \kappa_t^{(1)} = 0, \quad \sum_{y=t_1-x_k}^{t_n-x_1} \gamma_y = 0,$$

so that α can be interpreted as the overall average improvement and $\kappa_t^{(1)}$ and γ_{t-x} as period and cohort deviations from this average improvement.

4.3. Model decomposition by cause of death

Figures 1, 2 and 3 present the results of applying the cause of death decomposition approach to the PCi, APCi and PLATi models fitted to U.S. males aged 20 to 89 for the period 1959-2019.[4] For completeness, Figures B.9, B.10 and B.11 in Appendix B present equivalent results for U.S. females.

In each of these figures, the top panes present the usual parameter plots obtained by fitting the model to the all-cause mortality data. The bottom panes present matching cause of death decompositions of the model parameters resulting from applying the new decomposition approach introduced in Sections 3.2 and 3.3. To facilitate the identification of trends, in the bottom plots we average α_x, $\kappa_t^{(i)}$, $i = 1, 2$, and γ_{t-x} by 5-year age groups, 5-year periods and 5-year cohort groups, respectively.

We start first by discussing the general interpretation of the all-cause model parameters for the three models and then move to examining the additional insight provided by the cause of death decomposition of the parameters.

In the PCi model in Figure 1, the α parameter indicates that over the 1959-2019 period, mortality for males improved at a pace of about 0.95% p.a. However, the α_x parameters of the APCi model in the top left pane of Figure 2 show that there are strong age differentials, with males aged 50 having average improvements of 1.22% p.a. in sharp contrast with males aged 30 who have had a mild mortality deterioration at a pace of about 0.07% p.a.

In the PCi and APCi model, the $\kappa_t^{(1)}$ parameters represent period deviations from the average improvements. For example, according to the PCi model, in 1975 male mortality improved 2.5% p.a. more than the average of the whole period. Thus, the total average model improvement rate for 1975 indicated by the PCi model is 3.45% p.a. By contrast, in 2015 mortality for males improved 1.83% p.a. less than the average of the 1959-2019 period, for a total average mortality deterioration of 0.88% p.a.

[4] In fitting these models, we have zero-weighted corner cohorts with five or less observations.

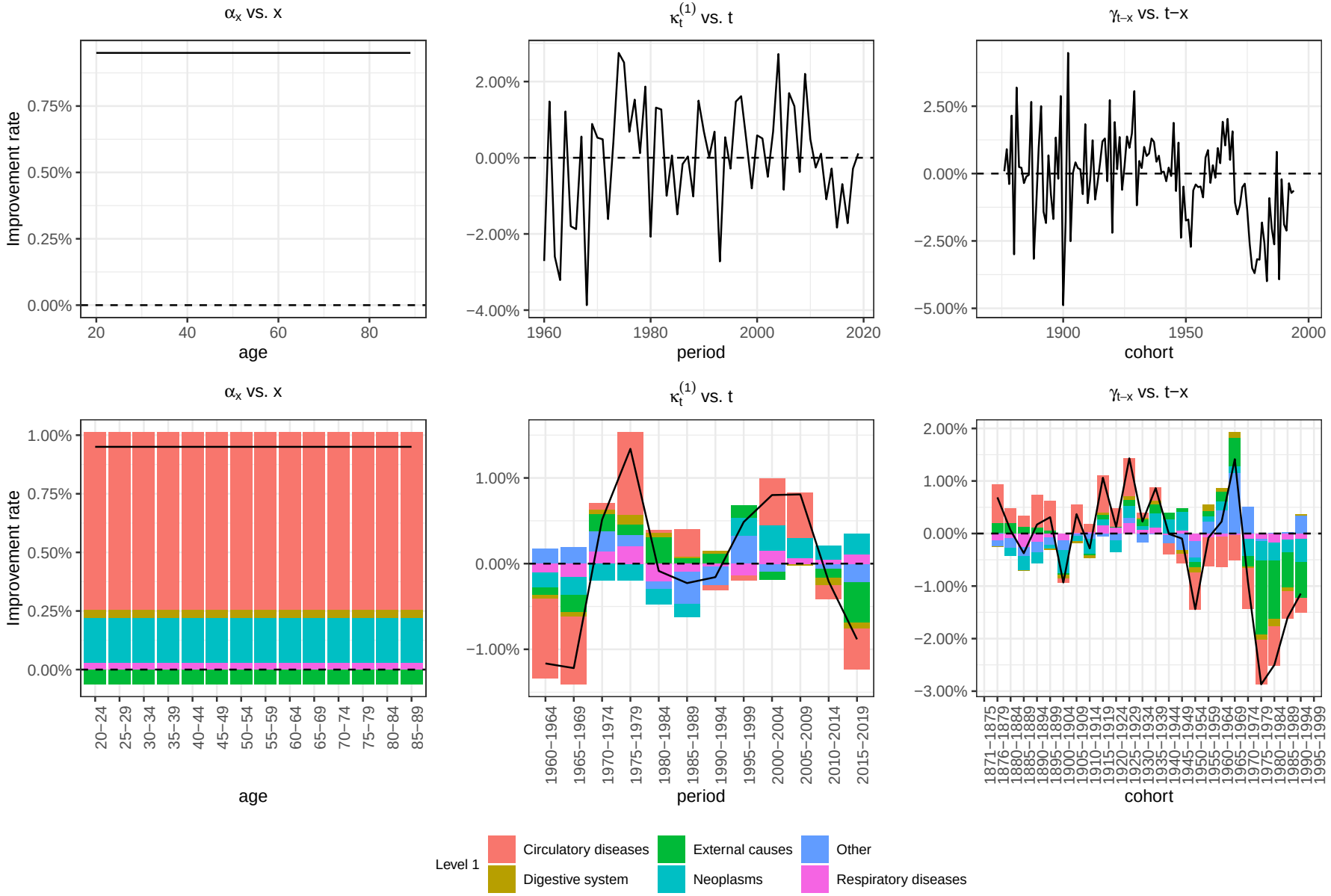

Figure 1: PCi model parameters decomposition, males, 1959–2019, aged 20-89

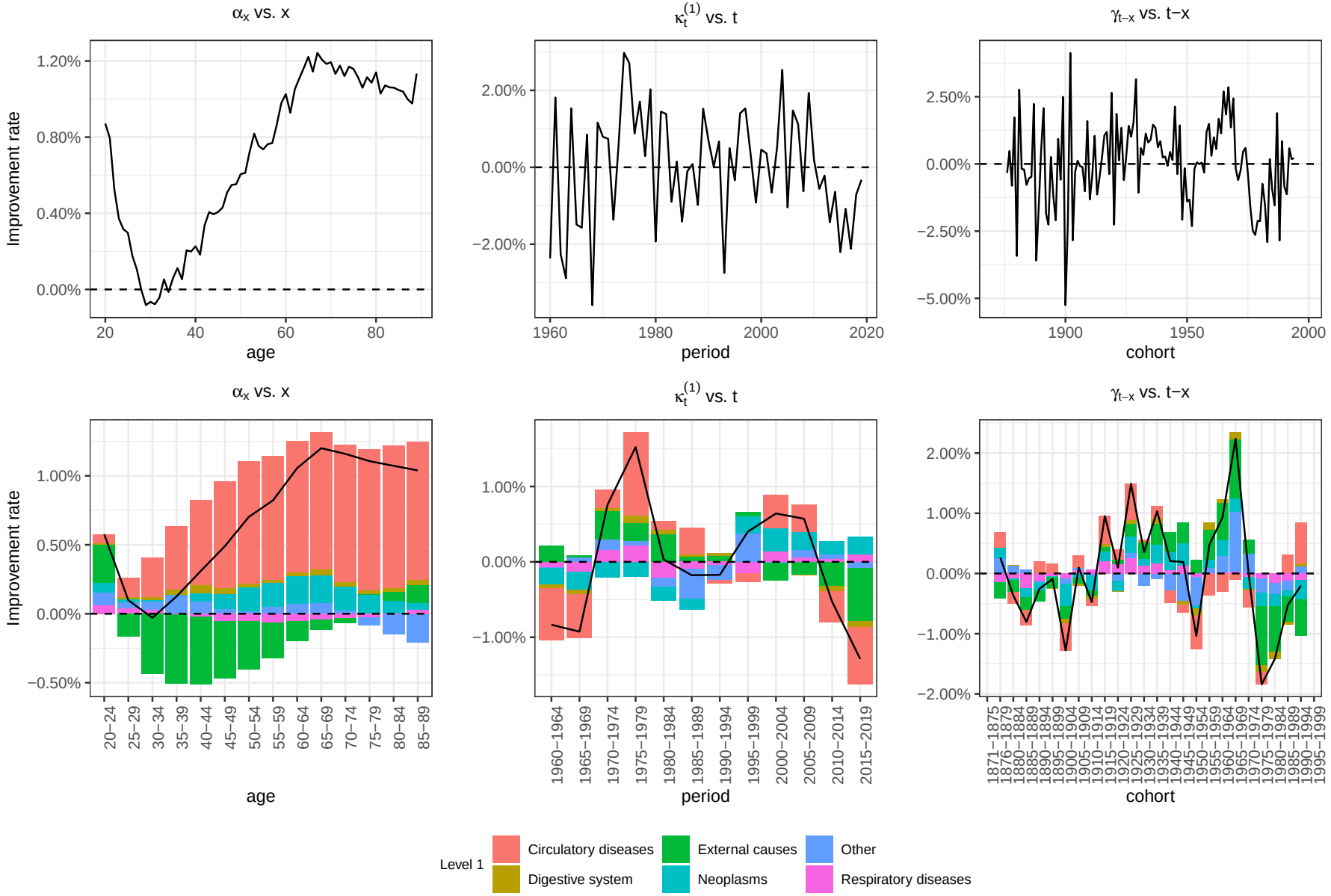

Figure 2: APCi model parameters decomposition, males, 1959–2019, aged 20-89

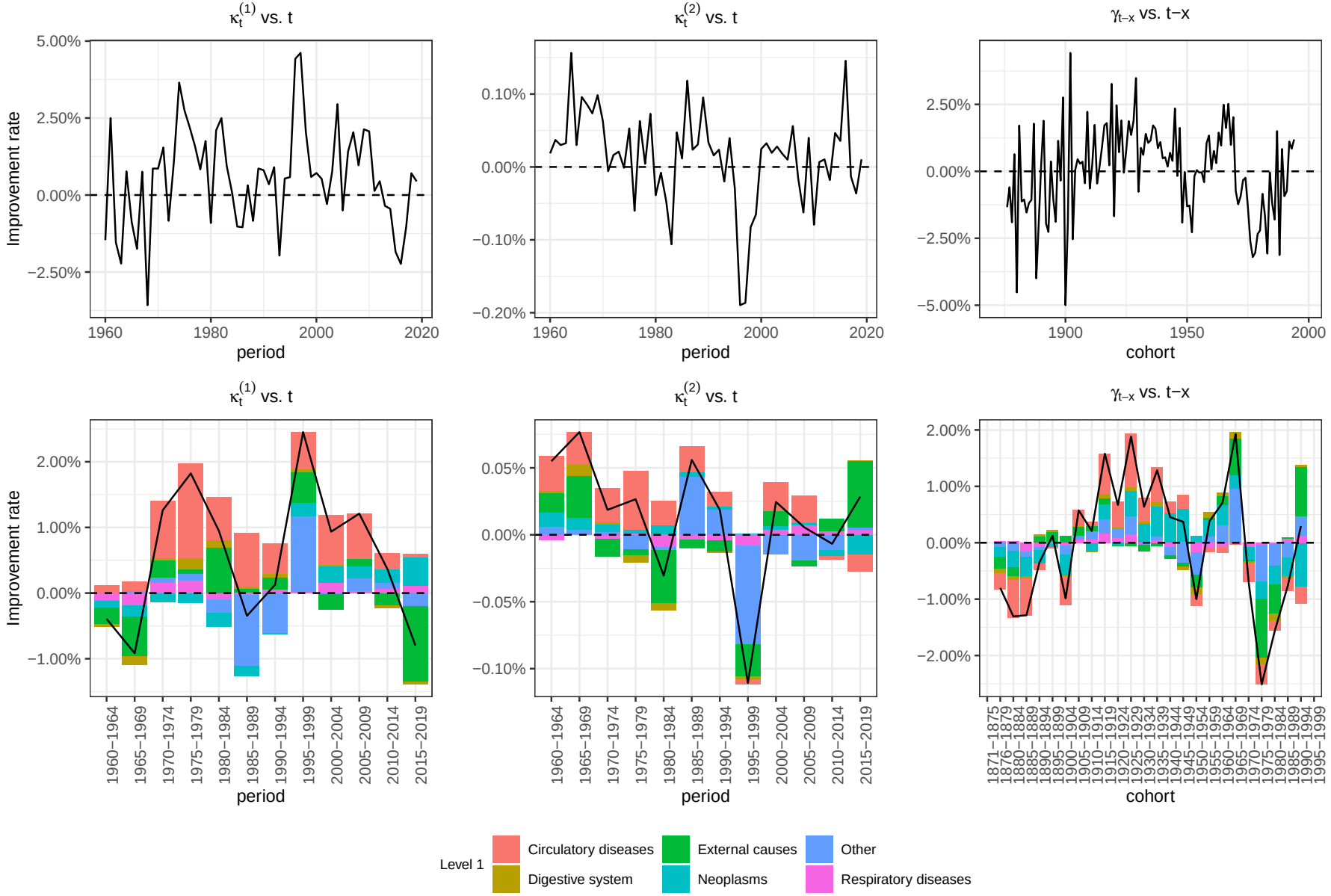

Figure 3: PLATi model parameters decomposition, males, 1959–2019, aged 20-89

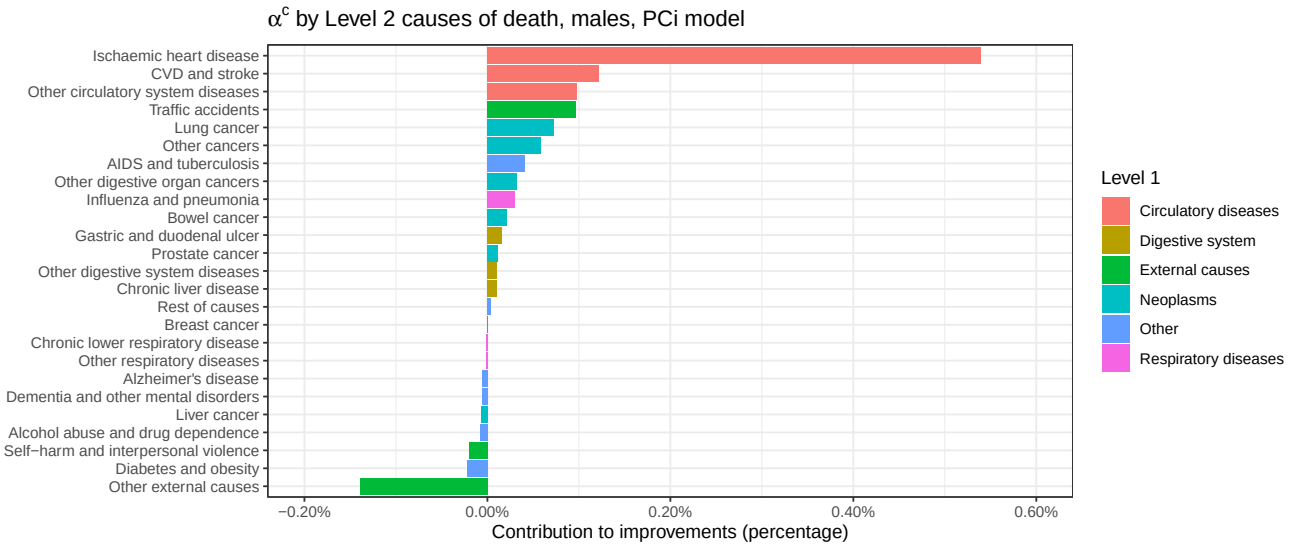

Figure 4: Decomposition of α^c in the PCi model by Level 2 causes of death, males, 1959–2019, aged 20-89

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 seen in Figure 3 indicate very fast improvements for younger men relative to older men.

In the three models, the γ_{t-x} parameters, which account for cohort specific deviations from the average improvements, follow essentially the same pattern. For example, in the PCi model we have that γ_{1950} =-1.74% indicating that the generation of men born in 1950 has experienced annual mortality improvements which are 1.74% worse than the average.

We now turn our attention to the additional insight that our decomposition approach provides on the interpretation of GAPC model parameters of the three models. In doing so, we highlight how this new results complement the analysis we have previously carried out in Villegas et al. (2021; 2022), where we examined the key drivers of long-term rates of mortality improvements in the U.S.

As discussed before, the α parameter of the PCi model shows that mortality for males improved at a pace of about 0.95% p.a. during the 1959-2019 period. Moreover, the cause of death decomposition on the bottom left pane of Figure 1 indicates that the majority of the improvement stems from circulatory diseases and neoplasms, which contributed, respectively, 0.76% p.a. and 0.19% p.a. By contrast, external causes of death had a small negative contribution of -0.06% p.a. Figure 4 shows the decomposition of the overall average improvements at the more detailed Level 2 grouping of causes. Here we see that the five main causes of death behind the overall mortality improvements between 1959 and 2019 are ischaemic heart disease, CVD and stroke, other circulatory system diseases, traffic accidents and lung cancer. By contrast, the largest negative contribution is due to other external causes, which, among other causes, includes accidental poisoning from alcohol and drug abuse.

The cause of death decomposition of parameter α_x in the APCi model provides useful insight about the drivers of mortality improvements at different ages. In the bottom left pane of Figure 2 we see that while circulatory diseases and neoplasms have contributed to mortality improvement at all ages, external causes of death have had an important negative contribution at younger ages, explaining the sharp differences in all-cause improvement between younger and older men. To dig deeper into the causes of death driving these age differentials, Figure 5 presents the Level 2 decomposition of α_x at ages 30-34 and 65-69 which are, respectively, the age-groups with the slowest and fastest all-cause mortality improvements. This decomposition indicates clearly that other external causes of death and self-harm and interpersonal violence are the main causes of the very slow improvement (or even deterioration) of mortality rates for U.S. males aged 30-34. By contrast, ischaemic heart disease is by far the main cause underlying the faster improvement of males aged 65-69.

In Villegas et al. (2021; 2022) we have identified five distinct periods of mortality change in the U.S. covering years

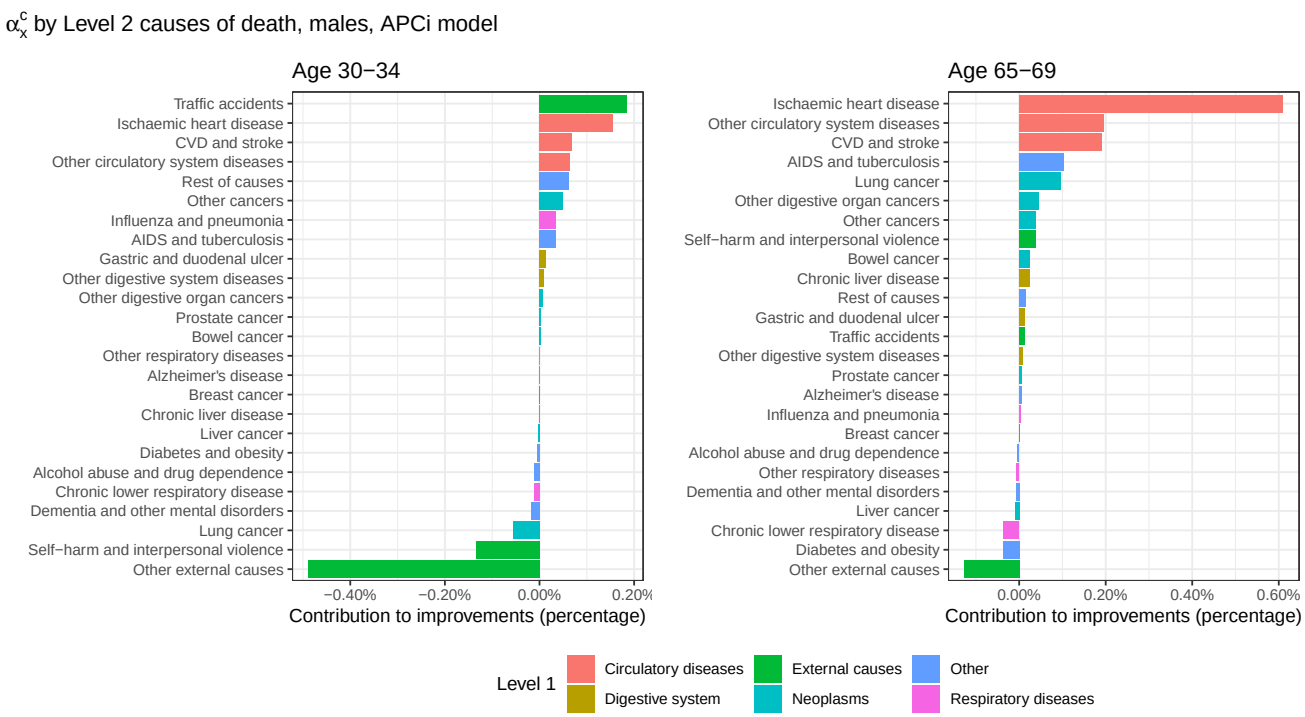

Figure 5: Decomposition of α_x^c in the APCi model by Level 2 causes of death for ages 30-34 and 65-69, males

1959-1970, 1970-1980, 1980-1995, 1995-2010, 2010-2016. The cause of death decomposition of parameters $\kappa_t^{(1)}$ in the PCi and APCi models in the bottom middle panes of Figures 1 and 2 can give interesting insight on the main factors driving mortality change in each of these subperiods:

- **Period 1959-1970:** During this period male mortality deteriorated with circulatory diseases accounting for the majority of this deterioration.
- **Period 1970-1980:** During this decade all-cause mortality rates experienced a clear acceleration in mortality improvement derived mainly from circulatory diseases, especially between 1975 and 1980.
- **Period 1980-1995:** This period is characterized by a deceleration in the pace of improvement for all-cause mortality, mainly driven by negative effects from other causes of death linked to the HIV epidemic.
- **Period 1995-2010:** This period saw a return to faster than average all-cause mortality improvements mainly driven by other causes of death (which include AIDS and tuberculosis) between 1995 and 2000 and by circulatory diseases from 2000 to 2010.
- **Period 2010-2019:** Finally, the most recent period – especially the last lustrum – has seen a clear deceleration of mortality improvements in all-cause mortality mainly due to negative effects from circulatory diseases and external causes of death.

To illustrate further the kind of insight that we can get from our cause of death decomposition approach, we present in Figure 6 the Level 2 decomposition of $\kappa_t^{(1)}$ for the APCi model for the periods 1975-1979 and 2015-2019. These two five year periods are of interest as they show contrasting trends, with 1975-1979 being the five years with the fastest improvements and 2015-2019 a period of very slow improvements.

Figure 6 shows that ischaemic heart disease and CVD and stroke account for the majority of the above average improvements between 1975 and 1979. As noted in Villegas et al. (2021; 2022), this significant contribution from circulatory diseases in the 1970s resulted from reductions in lifestyle risk factors such as cholesterol levels and smoking, and from major advances in medical treatments such as prehospital resuscitation, coronary artery bypass surgery and the treatment of hypertension (Goldman and Cook, 1984).

For 2015-2019, Figure 6 indicates that other external causes of death are the leading Level 2 cause associated with the slowdown in mortality improvements in this period, with a contribution of -0.53% p.a. Noticeably, this group of causes includes accidental poisoning from alcohol and drug abuse, supporting the narrative of "deaths of despair"

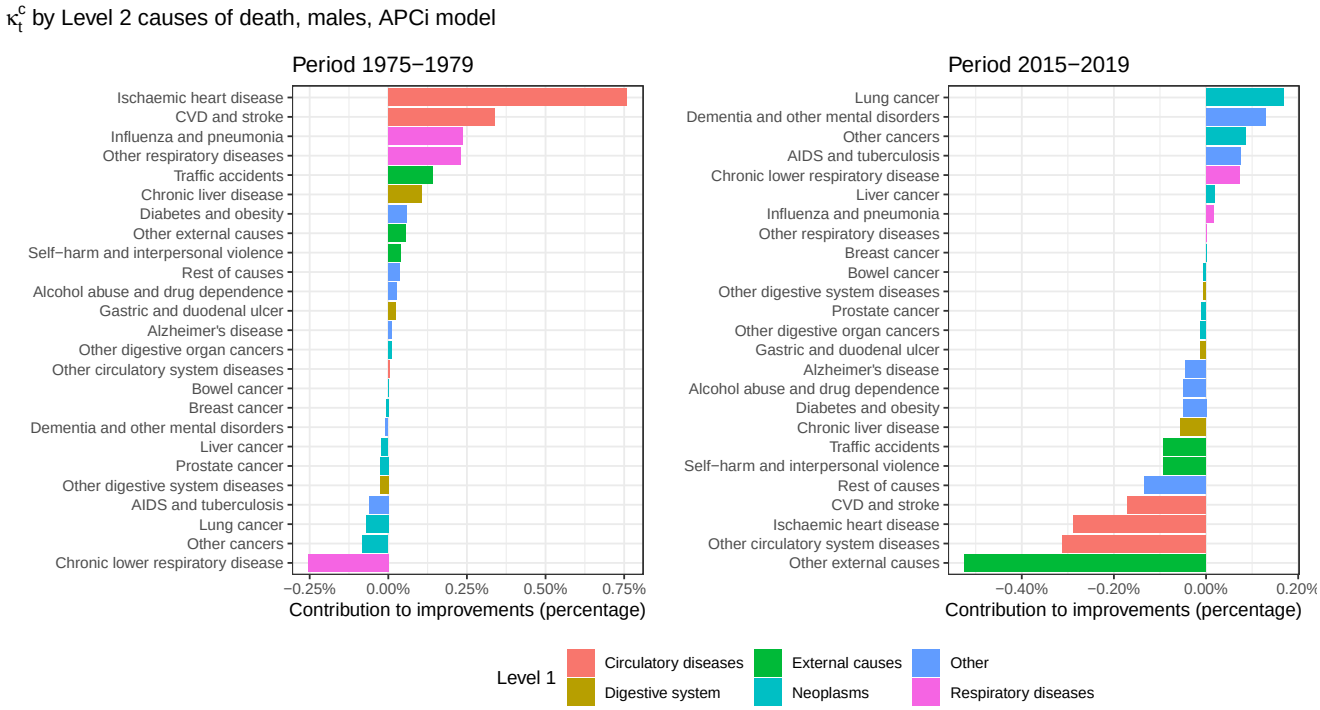

Figure 6: Decomposition of $\kappa_t^{(1,c)}$ in the APCi model by Level 2 causes of death for years 1975-1979 and 2015-2019, males

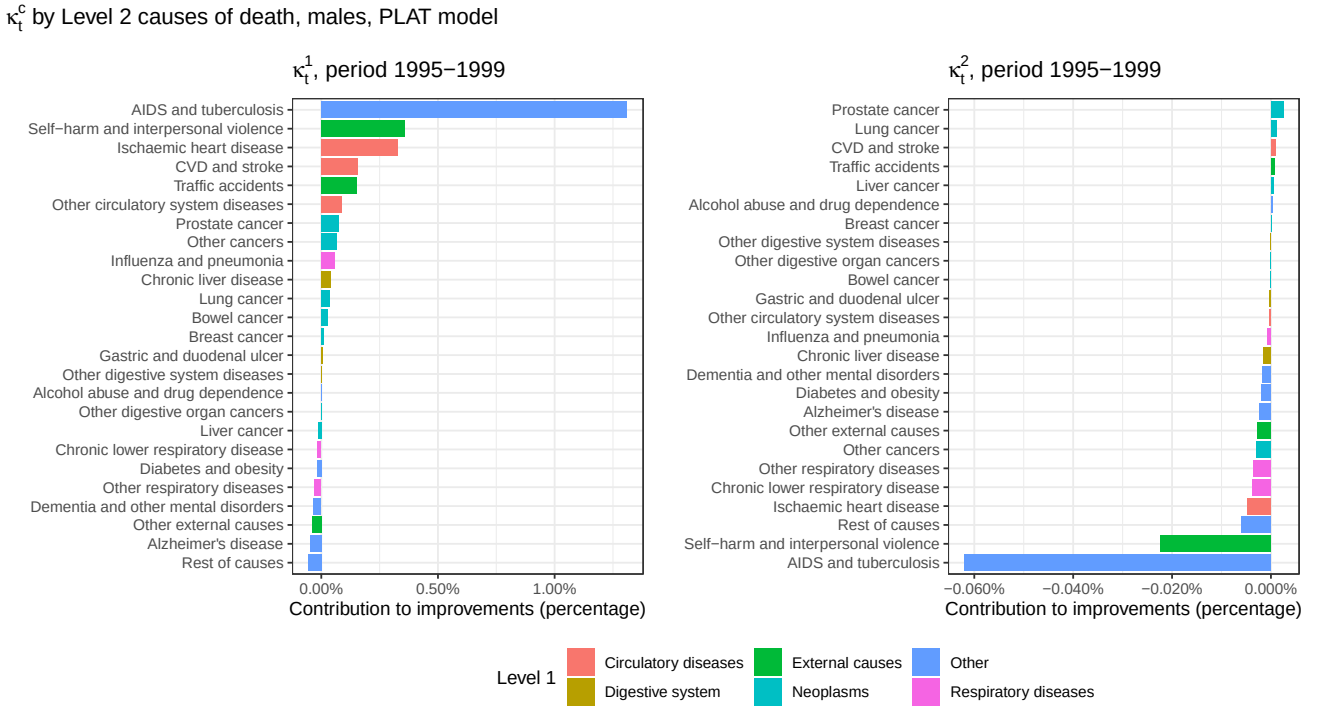

Figure 7: Decomposition of $\kappa_t^{(1,c)}$ and $\kappa_t^{(2,c)}$ in the PLATi model by Level 2 causes of death for period 1995-1999, males

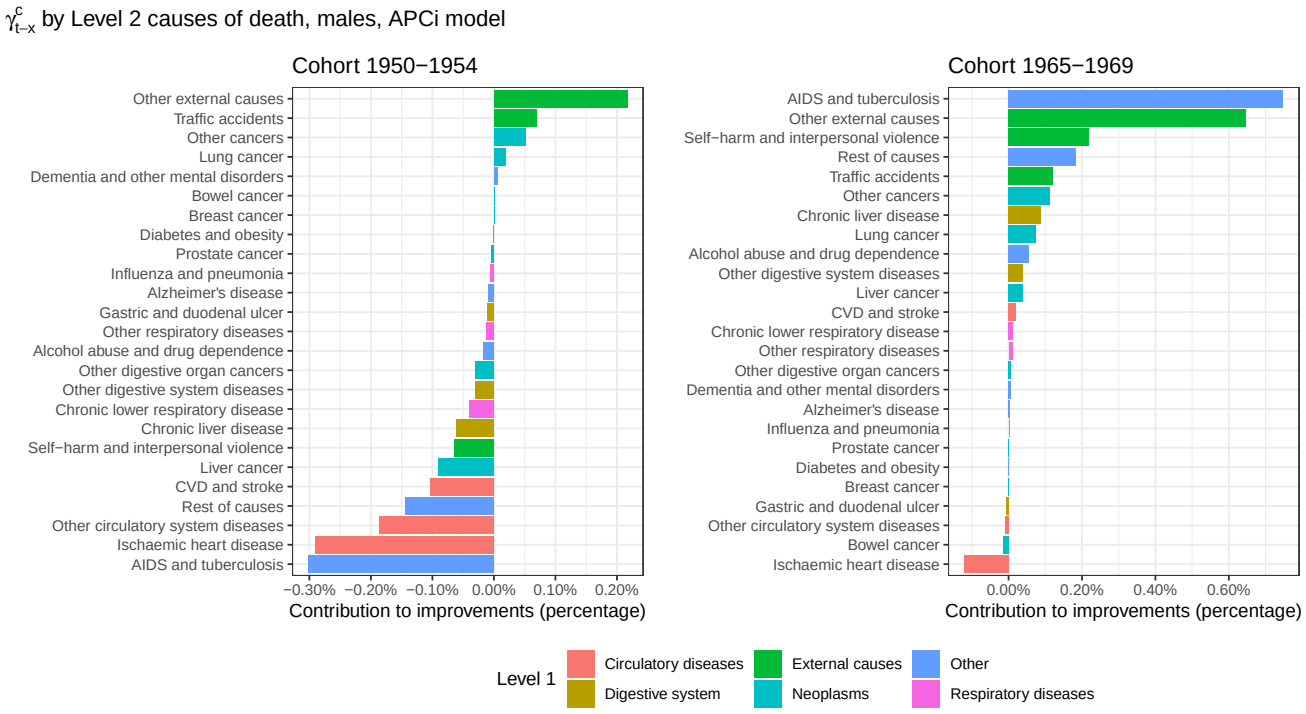

Figure 8: Decomposition of γ^c_{t-x} in the APCi model by Level 2 causes of death for cohorts 1950-1954 and 1965-1969, males

which has dominated the discussion of mortality change in the U.S. in the last decade (Case and Deaton, 2015). However, Figure 6 also shows that circulatory diseases combined contribute -0.77% p.a. to the slowdown of mortality improvement in this period. This is in agreement with the findings of Mehta et al. (2020) who argue that life expectancy in the U.S. has stalled due to circulatory diseases and not drug deaths.

In the PLATi model the behavior of $\kappa^{(1)}_t$ and $\kappa^{(2)}_t$ seen in Figure 3 during the period 1995-1999 deserves a special examination. The very high positive values of $\kappa^{(1)}_t$ indicate significant mortality improvements during this period. However, as discussed before, the matching negative values of $\kappa^{(2)}_t$ indicate faster improvements for younger men relative to older men. To shed light on this behavior, we plot in Figure 7 the Level 2 decomposition of $\kappa^{(1)}_t$ and $\kappa^{(2)}_t$ during this period. In this figure, we see clearly that AIDS and Tuberculosis is the main cause behind these parameters, coinciding with the introduction of anti-retroviral therapy in 1996 (Murphy et al., 2001).

Focusing now on cohort effects, we see in the right panes of Figures 1, 2 and 3 that there are significant generational differences in mortality improvements among U.S. men. Particularly noteworthy are the below average improvements of the 1950-1954 birth cohort and the above average improvements of the 1965-1969 generation. To understand this pattern, Figure 8 shows the Level 2 decomposition of γ_{t-x} based on the APC model. We see that the majority of Level 2 causes have a positive contribution to cohort effects for the 1965-1969 generation. In contrast, the 1950-1954 generations show significant negative contribution from AIDS and tuberculosis, circulatory diseases, and liver-related diseases.

5. Conclusions

In this paper we have introduced an approach for decomposing the parameters of GAPC improvement rate models into the additive contribution of different causes of death. This approach is in the same spirit of the decomposition methods commonly used by demographers to measure and explain the contribution of different factors to the evolution of demographic metrics (see, e.g., Arriaga (1984) and Andreev et al. (2002)). These methods have the appeal of allowing simple graphical representations that can help communicate the drivers of mortality change. As such, we hope that this new tool can help longevity modelers interpret their mortality modeling assumptions and communicate their results to non-experts.

The application of this new decomposition approach to U.S. mortality data complements and supports the previous findings we have reported in Villegas et al. (2021; 2022) on the long-term drivers of U.S. mortality change. Namely, that circulatory disease are the main driver of U.S. mortality improvements between 1959 and 2019, but with a variety of causes of death inducing significant variations in mortality improvement across ages, period and cohorts.

There are several avenues for future research. From the methodological perspective, the decomposition approach can readily be extended beyond the causes of death dimension to include a subpopulation dimension. This would be useful, for example, in understanding the role of socioeconomic and geographic differentials on the evolution of aggregate mortality.

In addition, the decomposition approach opens up new possibilities for deriving mortality projections using GAPC models. In particular, in future work we aim to explore if combining this decomposition with forecast reconciliation methods (Li et al., 2019) has the potential to improve the accuracy of all-cause mortality projections.

Finally, from the empirical perspective, once cause of death data for 2020 and 2021 become available, it would be interesting to apply the decomposition approach to shed light on the impact of COVID-19 deaths on mortality assumptions.

Appendix A. Proof of Theorem 1

Let $\mathbf{y} = (i_{x_1,t_1}, i_{x_2,t_1}, \ldots, i_{x_{k-1},t_n}, i_{x_k,t_n})'$ be the column vector stacking the all-cause mortality improvement rates calculated using approximation (5). Similarly, let $\mathbf{y^c} = (i^c_{x_1,t_1}, i^c_{x_2,t_1}, \ldots, i^c_{x_{k-1},t_n}, i^c_{x_k,t_n})'$, $c = 1, \ldots, m$, be the column vector stacking the contribution of cause c to all-cause mortality improvements, calculated using (8).

Following Currie (2016), we know that the GAPC model in Equation 2 is a linear model with the following matrix representation

$$\mathbf{y} = \mathbf{X}\boldsymbol{\beta},$$

with coefficient vector $\boldsymbol{\beta} = (\boldsymbol{\alpha}', \boldsymbol{\kappa}^{(1)'}, \ldots, \boldsymbol{\kappa}^{(N)'}, \boldsymbol{\gamma}')$ and design matrix $\mathbf{X}$.

Similarly, we can write the model in Equation 9 as

$$\mathbf{y}^c = \mathbf{X}\boldsymbol{\beta}^c, \quad c = 1, \ldots, m,$$

with coefficient vector $\boldsymbol{\beta}^c = (\boldsymbol{\alpha}^{c\prime}, \boldsymbol{\kappa}^{(1),c'}, \ldots, \boldsymbol{\kappa}^{(N),c'}, \boldsymbol{\gamma}^{c\prime})$.

In most GAPC models the design matrix $\mathbf{X}$ is not full rank due to indentifiability issues. However, without loss of generality, assume that we set as many $\beta_i \equiv 0$ and $\beta^c_i \equiv 0$ so that the linear problems are reduced to

$$\mathbf{y} = \tilde{\mathbf{X}}\tilde{\boldsymbol{\beta}},$$

and

$$\mathbf{y}^c = \tilde{\mathbf{X}}\tilde{\boldsymbol{\beta}}^c,$$

with $\tilde{\mathbf{X}}$ being full rank. Thus, the weighted least squares estimate of $\tilde{\boldsymbol{\beta}}$ is given by

$$\tilde{\boldsymbol{\beta}}_{WLS} = (\tilde{\mathbf{X}}'\mathbf{W}\tilde{\mathbf{X}})'\tilde{\mathbf{X}}'\mathbf{y},$$

with $\mathbf{W}$ denoting the diagonal matrix with weights $w_{x,t}$ in the diagonal and zeros everywhere else. Similarly, the weighted least squares estimate of $\tilde{\boldsymbol{\beta}}^c$ is given by

$$\tilde{\boldsymbol{\beta}}^c_{WLS} = (\tilde{\mathbf{X}}'\mathbf{W}\tilde{\mathbf{X}})'\tilde{\mathbf{X}}'\mathbf{y}^c.$$

Since $\mathbf{y} = \sum_{c=1}^m \mathbf{y}^c$, we have that

$$
\begin{aligned}
\sum_{c=1}^{m} \tilde{\boldsymbol{\beta}}_{WLS}^{c} &= \sum_{c=1}^{m} (\tilde{\mathbf{X}}'\mathbf{W}\tilde{\mathbf{X}})'\tilde{\mathbf{X}}'\mathbf{y}^{c} \\
&= (\tilde{\mathbf{X}}'\mathbf{W}\tilde{\mathbf{X}})'\tilde{\mathbf{X}}' \left(\sum_{c=1}^{m} \mathbf{y}^{c} \right) \\
&= (\tilde{\mathbf{X}}'\mathbf{W}\tilde{\mathbf{X}})'\tilde{\mathbf{X}}'\mathbf{y} \\
&= \tilde{\boldsymbol{\beta}}_{WLS}
\end{aligned}
$$

which completes the proof.

Appendix B. PCi, APCi and PLATi model decompostion for U.S. females, 1959-2019, aged 20-89

See Figures B.9, B.10 and B.11

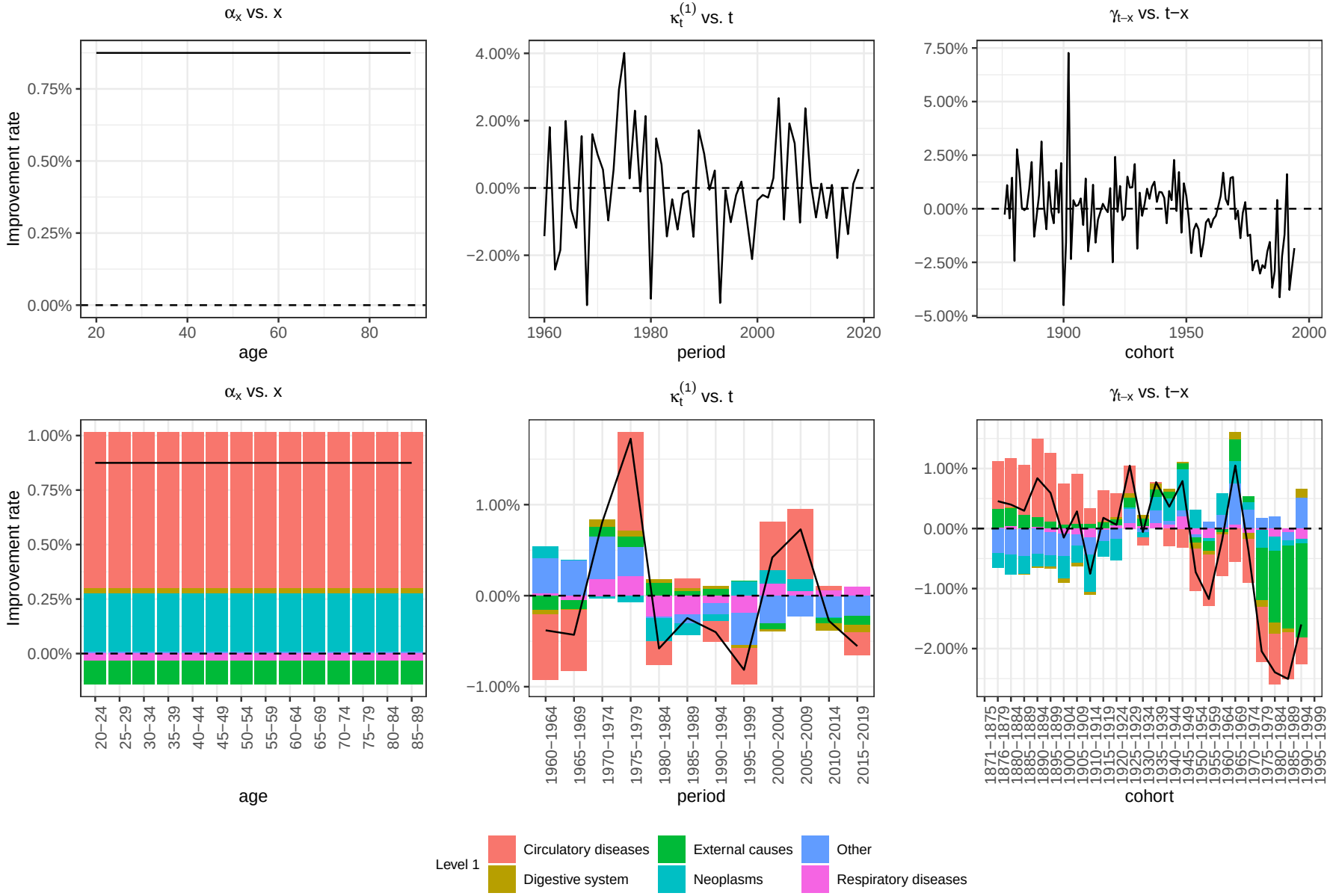

Figure B.9: PCi model parameters decomposition, females, 1959–2019, aged 20-89

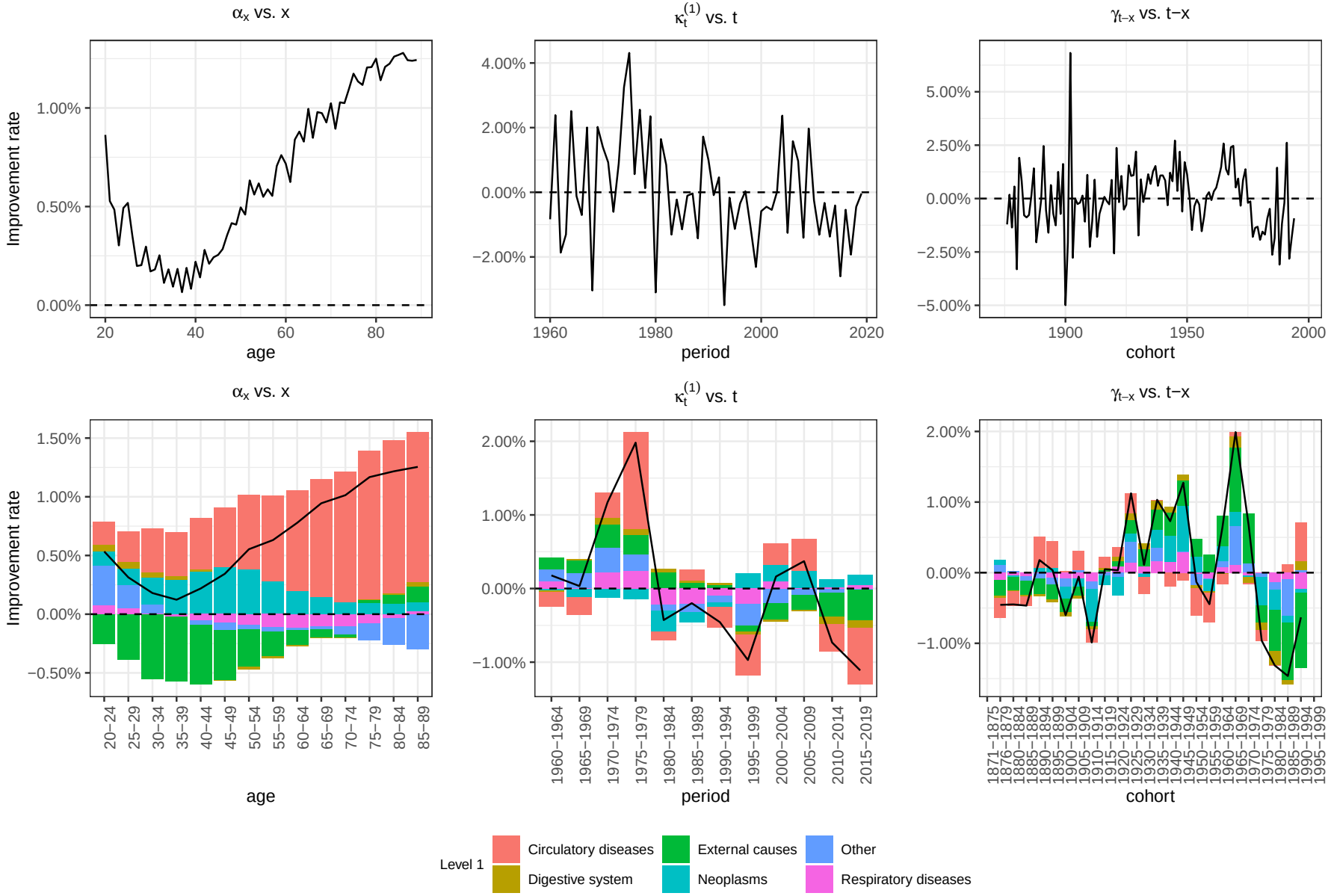

Figure B.10: APCi model parameters decomposition, females, 1959–2019, aged 20-89

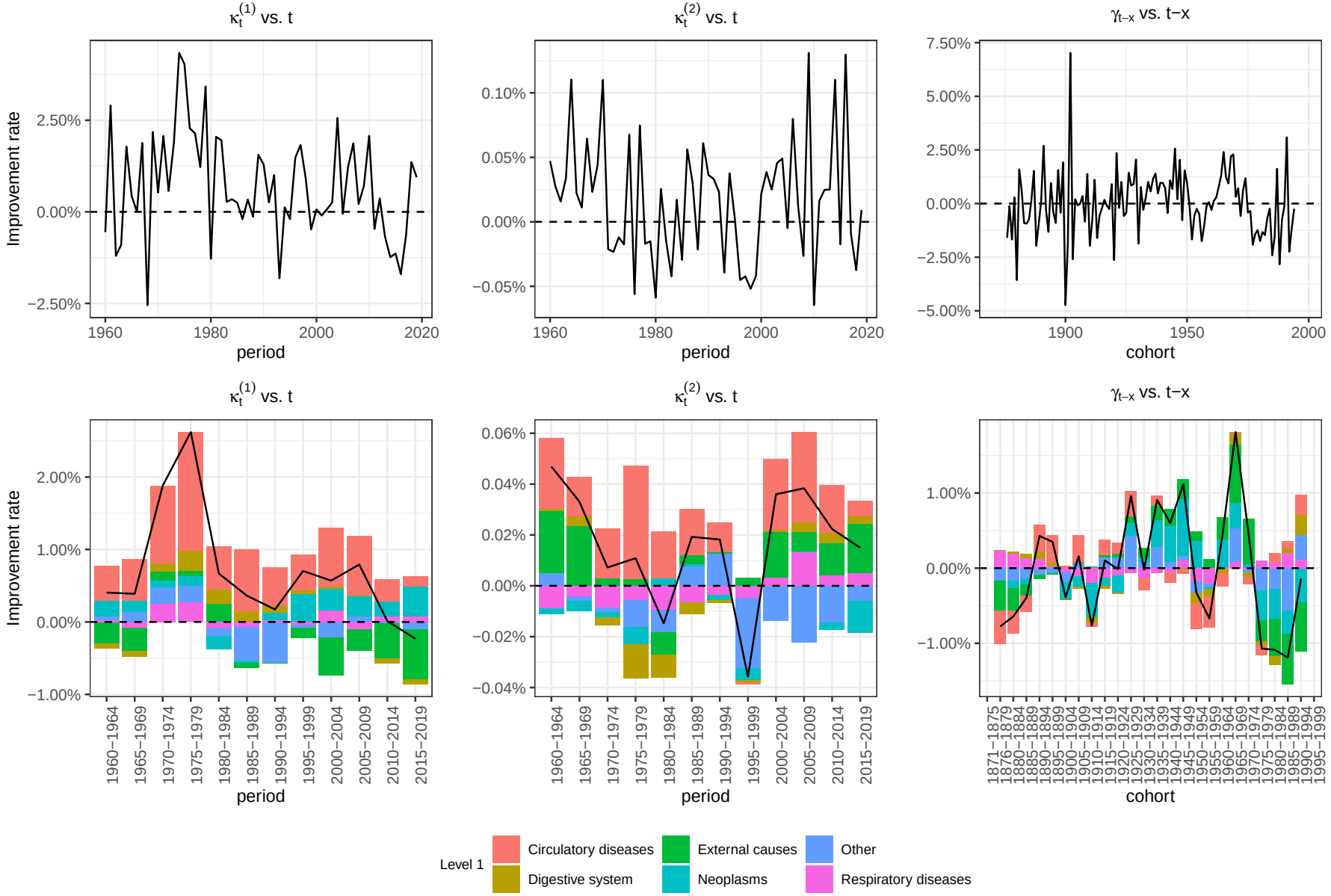

Figure B.11: PLATi model parameters decomposition, females, 1959–2019, aged 20-89

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475 N. Martingale Road, Suite 600
Schaumburg, Illinois 60173
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