Improvements in Individual Death Cause Mortality Give Rise to Lifespan Inequality

Insights from a New Framework for Parametric Cause-of-Death Analysis

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I develop a new modeling and decomposition framework to account for causes of death and their distribution across ages in mortality data. The approach allows isolating two aspects of mortality: death causes are competing, in the sense that a reduction of mortality from one cause may increase mortality from other causes; and a closely related substitution within the causes, as deaths might become more concentrated, dispersed or shifted across the age range. A priori, it is therefore unclear whether the reduction of mortality from one cause increases or decreases life expectancy, as these substitution mechanisms can attenuate or exacerbate changes in life expectancy.

I apply the decomposition to data for the USA over the range 1980 to 2022, obtained from the Human Mortality Database. The findings shed light on the role of specific causes of death in the recent increase in lifespan inequality and in the differences in life expectancy between males and females. The main finding is that the reduction in deaths from individual causes gave rise to increased lifespan inequality. This, in turn, attenuated the gains in life expectancy. For example, from 1980 to 2019, male life expectancy increased by ca. 2.4 years due to improvements in heart disease. Without the attenuation by lifespan inequality it would have been ca. 2.9 years. Mortality reductions due to better treatment of "diseases of the nervous system and sense organs" and "endocrine, nutritional and metabolic disease" show the same dynamic.

The decomposition methodology can also be applied to analyze the differences in mortality dynamics between populations, e.g. by sex. Women live longer and have less unequal lifespans than men. A main finding here is that while reductions in heart disease mortality attenuated gains in life expectancy due to lifespan inequality, reductions in cancer mortality *increased* gains in life expectancy through lifespan inequality.

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In this extended abstract I present the argument for and the methodology behind the decomposition approach. I then apply the decomposition and analyze the effects of cause-specific mortality on aggregate mortality.

1. The Argument for a Framework for Parametric Cause-of-Death Analysis

The mortality of a population is characterized by its age-at-death distribution. A popular measure of mortality is the first moment of this distribution: life expectancy. Since it is reducing the entire distribution into a singular value, it does not capture important distributional aspects. This can be illustrated by two extreme cases of mortality distributions: a perfectly uniform distribution of death ages, and one in which everyone dies at the same age. Both can give rise to the exact same level of life expectancy, despite entirely different patterns of mortality. Life expectancy is also not indicative of competing causes of death. When deaths from one cause are prevented, e.g., due to reductions in heart disease as consequence of medical innovation, the mortality decline is counterbalanced by increased mortality in older ages, e.g., due to cancer.

Research on higher moments of the distribution of age-specific mortality has received increasing interest in recent decades, in particular in the context of refined parametric models of mortality that explicitly account for shifts and changes in the distribution (Basellini, Canudas-Romo, and Lenart 2019). A general finding of studies on age-specific mortality is the "rectangularisation" of the survival curve or "compression of mortality": as life expectancy increases, inequality of lifespans decreases (van Raalte, Sasson, and Martikainen 2018; Vaupel, Zhang, and van Raalte 2011). Likewise, demographers have developed decomposition methods for this specific problem, based on cause-deleted lifetables (Beltran-Sanchez, Preston, and Canudas-Romo 2008). Approaches that combine parametric models of age-specific mortality and cause-specific mortality are still lacking.

Accounting for the distributional properties of the age-at-death distribution beyond the first moment and incorporating different causes of death therefore constitutes an important prerequisite for progress in understanding the dynamics of aggregate mortality patterns and their drivers. Here, I propose a combination of the parametric modelling of age-specific mortality and cause-deleted lifetables into a unified framework. This framework allows for a decomposition of life expectancy changes into the contributions of age-specific mortality from individual death causes. A further decomposition then associates the cause-specific components with the model parameters. The framework provides a new perspective on the impact of individual death causes on aggregate mortality, through the channels of the mortality parameters.

2. The Framework

Survival models define the mortality rate as a parametric function. For example, the mortality rate of the seminal Gompertz-Makeham model is defined as (Basellini, Canudas-Romo, and Lenart 2019):

$$\mu_(a) = A + \frac{1}{s} \exp\left(\frac{a-l}{s}\right).$$

One of the advantages of this model is that its parameters have meaningful interpretations: A is an age-independent, or background mortality rate; l is the modal age at death; and s is the spread, of death ages around l. This model is well suited to capture the distributional aspects of mortality in a parsimonious way. Depending on the complexity and age range of the age-at-death distribution, more or less parsimonious models, e.g. the Gompertz or the Siler model are also available.

These models have the shortcoming that they are not suited to model death rates of individual causes, since these follow more complex distributions. They are however suited to model cause-deleted mortality rates, i.e. from all death causes except one. Methods based on cause-deleted lifetables have been shown to be equivalent to standard lifetable decomposition methods (Beltran-Sanchez, Preston, and Canudas-Romo 2008) and thus provide a natural starting point for the development of a parametric decomposition framework.

Life expectancy at birth is defined as:

$$e(0,t) = \int_0^\omega \exp\left(-\int_0^a \mu(x)dx\right) da = \int_0^\omega p(a,t)da$$

where $p(a)$ is the survival function. The cause-deleted survival function is defined as the ratio between the all-cause and the cause i survival function: $p_{-i} = p(a)/p_i(a)$. The object of interest in this framework is the time-change in the *gain in life expectancy if cause i were eliminated*:

$$D_i(0) = \int_0^\omega p_{-i}(a)da - \int_0^\omega p_{-i}(a)p_i(a)da$$

given by:

$$\dot{D}_i(0) = \int_0^\omega \dot{p}_{-i}(a)q_i(a)da - \int_0^\omega \dot{p}_i(a)p_{-i}(a)da.$$

A dot above a variable denotes its partial derivative with respect to time t. Since $q_i(a) = 1 - p_i(a)$ and $p_i(a) = p(a)/p_{-i}(a)$, this expression can be rewritten entirely in terms of cause-deleted and all-cause survival functions:

$$\dot{D}_i(0) = \underbrace{\int_0^\omega \dot{p}_{-i}(a)\left(1 - \frac{p(a)}{p_{-i}(a)}\right)da}_{:=\Delta_{-i}} - \underbrace{\int_0^\omega \left(\frac{\partial}{\partial t}\frac{p(a)}{p_{-i}(a)}\right)p_{-i}(a)da}_{:=\Delta_i} \tag{1}$$

This cause-of-death decomposition splits the change in the *gain in life expectancy if cause i were eliminated* into two components: the cause-specific component Δ_i, which captures the time-change in all-cause life expectancy due to cause i and Δ_{-i}, which captures the attenuation or exacerbation due to all other causes. Beltran-Sanchez, Preston, and Canudas-Romo (2008) give an intuitive example: treatment of cancer has improved over the last decades, leading to increases in life expectancy through Δ_i. But, since mortality from other causes also declined, more people become old enough to die of cancer. In the aggregate this leads to more lives being lost to cancer and this effect is captured by Δ_{-i}.

To account for distributional aspects, I employ the parametric decomposition approach from Bergeron-Boucher, Ebeling, and Canudas-Romo (2015) to Δ_i and Δ_{-i}. Let every parameter from the Gompertz-Makeham model depend on time t. Equation (1) can then be decomposed as:

$$\begin{aligned}\dot{D}_i(0) &= \int_0^\omega \dot{p}_{-i}(a, A_t, l_t, s_t)\left(1 - \frac{p(a, A_t, l_t, s_t)}{p_{-i}(a, A_t, l_t, s_t)}\right) da - \int_0^\omega \left(\frac{\partial}{\partial t}\frac{p(a, A_t, l_t, s_t)}{p_{-i}(a, A_t, l_t, s_t)}\right) p_{-i}(a, A_t, l_t, s_t) da \\ &= \underbrace{(\Delta^A_{-i} + \Delta^l_{-i} + \Delta^s_{-i})}_{=\Delta_{-i}} - \underbrace{(\Delta^A_i + \Delta^l_i + \Delta^s_i)}_{=\Delta_i}\end{aligned}$$

Each cause-of-death component is now decomposed into three parts, each corresponding to a model parameter. Since they are measured in years, their interpretation is straightforward: Δ^l_i for example is the change in life expectancy, for cause i, due to changing the modal age at death. Beltran-Sanchez, Preston, and Canudas-Romo (2008) also show that the Δ_i components sum up to the aggregate change in life expectancy:

$$\dot{e}(0) = \sum_{i=1}^{n} \int_0^\omega \dot{p}_i(a) p_{-i}(a) da = \sum_{i=1}^{n} \Delta_i = \sum_{i=1}^{n} (\Delta^A_i + \Delta^l_i + \Delta^s_i)$$

3. Preliminary Results

I apply the new modeling and decomposition approach to cause-specific mortality data for the USA from 1980 to 2020, obtained from the Human Mortality Database (HMD 2024). The HMD provides an aggregation of the ICD 10 codes into 16 causes of death i. Figure 1 displays the change in life expectancy for US males, decomposed by causes. Life expectancy has increased mainly due to reductions of mortality from heart disease (ICD codes I00-I51), cancers ("neoplasms", ICD codes C00-D48) and external causes (ICD codes V01-Y8). On the other hand, life expectancy has decreased due to increased mortality from the "certain infectious disease" category (ICD codes A00-B99), which encompasses HIV; from diseases of the nervous system and sense organs (ICD codes G00-G44, G47-H93); endocrine, nutritional and metabolic diseases (ICD codes E00-E88) and mental and behavioural disorders (ICD codes F01-F99). The recent Covid pandemic increased the latter causes, but is mainly registered as acute respiratory disease (ICD codes J00-J22 U04, U07-U10). A similar pattern emerges for female life expectancy, which is driven by mostly the same causes (see fig. 3 in appendix A). The exception is a

stronger loss of life expectancy due to diseases of the nervous system and sense organs; mental and behavioral disorders; and other respiratory diseases (ICD codes J30-J98).

In addition to the aggregate effects of death causes on life expectancy, the new methodology can disentangle how changes in cause-specific mortality affect life expectancy. With the Gompertz-Makeham model, these effects are captured by the three parameters: background mortality A, the modal age at death (or lifespan) l and the spread of death ages around the mode (or lifespan inequality) s. Figure 2 displays the cause- and parameter-decomposition. The most striking finding is that heart disease, despite being the biggest contributor to increases in life expectancy, also had adverse effects on life expectancy. The cumulative gains in life expectancy due to heart disease amount to ca. 2.4 years by 2019, but they were attenuated by ca. 0.5 years due to increased lifespan inequality and background mortality. Similar attenuation effects can be found for malignant neoplasms; cerebrovascular disease; diseases of the nervous system and sense organs; and endocrine, nutritional and metabolic disease.

The loss in life expectancy due to the AIDS epidemic affected life expectancy through reducing the lifespan and increasing inequality in background mortality. But its effects are constrained to the period of the late 80s and 90s, and only plays a small role in recent times. Reductions in external causes of mortality led to a general increase in life expectancy through all parameters. But this general trend only held until 2010. From there on it had a negative effect through lifespan and lifespan inequality. Only through background mortality, and especially for females (see fig. 4 in appendix A) it continues to improve life expectancy. This leaves open the question whether this development is driven by the "deaths of despair".

The only death causes that led to a reduction in aggregate life expectancy – diseases of the nervous system and sense organs; and endocrine, nutritional and metabolic diseases – reveal an interesting heterogeneity: the increase in mortality due to these death causes led to a reduction in life expectancy, mostly through reductions in the lifespan. At the same time, they led to an increase in life expectancy through a background mortality and lifespan inequality. This is especially the case for females and a similar pattern is found for cancer mortality.

Figure 1: Cause-Decomposed Cumulative Time Change in US Male Life Expectancy
Cumulated time changes in life expectancy, decomposed into the cause-components Δ_i, for US males from 1980 to 2020. The black line displays the aggregated cumulative change : $\sum_{i=1}^{n} \Delta_i$, i.e. the time-change in life expectancy.

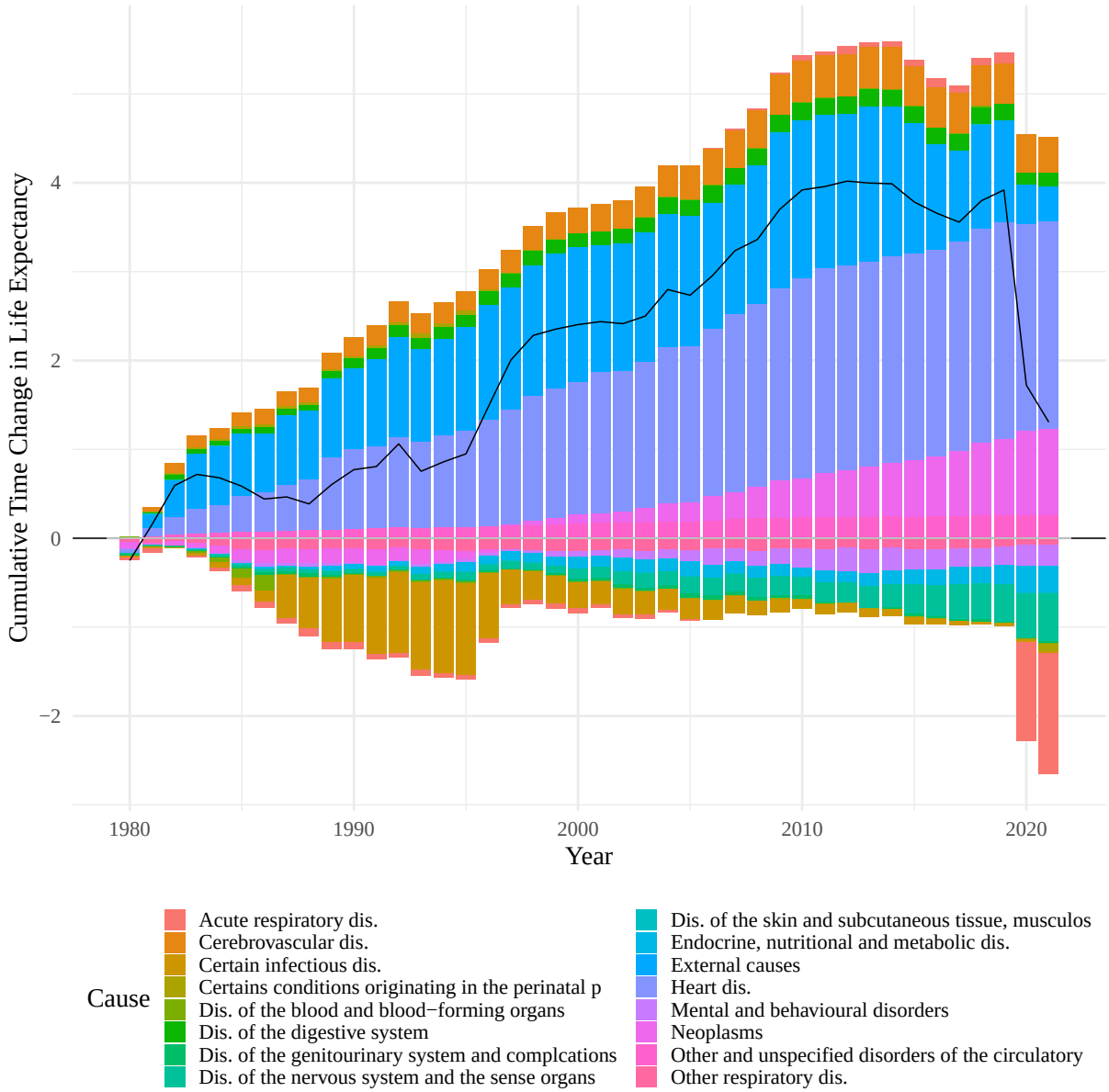
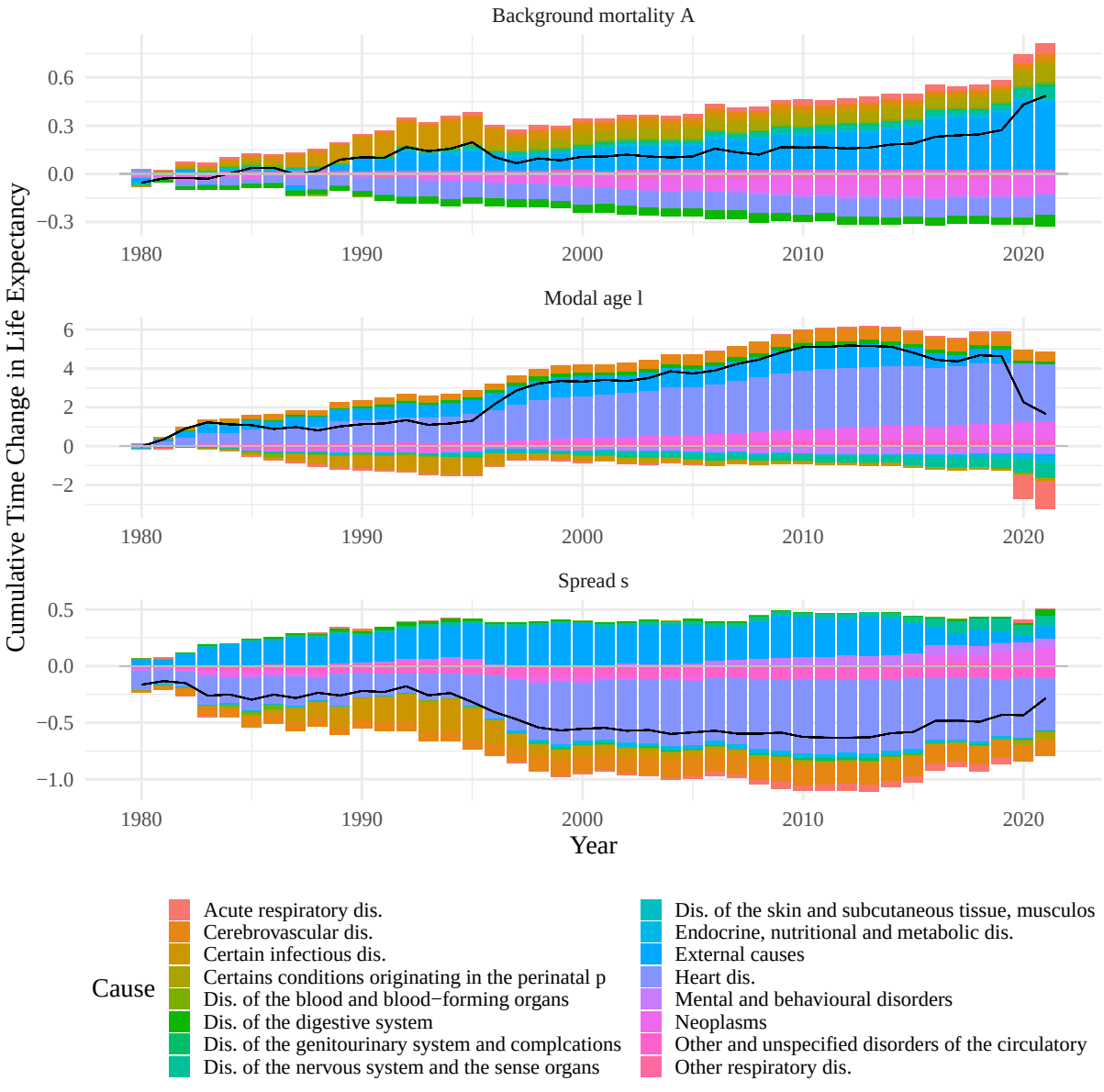


Figure 2:

Cause- and Parameter-Decomposed Cumulative Time Change in US Male Life Expectancy
The panels display the cumulated time changes in life expectancy, decomposed into the cause- and parameter components Δ_i^A, Δ_i^l and Δ_i^s for US males from 1980 to 2020. The black line displays the cumulated change due to each parameter, aggregated over all causes: $\sum_{i=1}^{n} \Delta_i^A$ and equivalently for Δ_i^l and Δ_i^s *Note that the panels have different y-scales*



4. Discussion and Conclusion

In this extended abstract I present a new framework for the decomposition of life expectancy. I combine parametric and cause-of death decomposition in order to shed light on distributional and compositional effects of individual causes on aggregate life expectancy. This enables a detailed analysis

of the dynamics of aggregate mortality. In an application of the new methods to US mortality data I showed that the cause-specific mortality has heterogeneous effects on the age-at-death distribution. The main finding is that improvements in life expectancy, e.g. due to heart disease and cancer, negatively affect life expectancy due to increasing lifespan inequality.

The new decomposition framework enables further research, beyond the focus on cause-specific changes in life expectancy. The recent trend reversal in deaths by external causes as well as certain old-age causes that are on the rise might be a driver of the observed increase in lifespan inequality in the US. When more people die in younger ages, as well as in older ages, the age-at death distribution becomes more dispersed. Further research could analyze how lifespan inequality is affected by these different causes. For this and other purposes it can also be useful to employ a more fine grained cause classification that goes beyond the 16 basic categories of the present analysis. Lastly, the decomposition can be used to analyze not only differences between the sexes, but between different countries, ethnicities and socioeconomic groups.

Another possible application is the question how mortality reductions interact with causes over time. Cumulative reductions in mortality from one cause may lead to a larger proportion of people surviving into old ages, where they increase the mortality from other causes. The remaining-cause component of the decomposition captures this effect and can be utilized for this question.

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A. Additional Figures

Figure 3: Cause- Decomposed Cumulative Time Change in US Female Life Expectancy
Cumulated time changes in life expectancy, decomposed into the cause-components Δ_i, or US females from 1980 to 2020. The black line displays the aggregated cumulative change : $\sum_{i=1}^{n} \Delta_i$,i.e. the time-change in life expectancy.

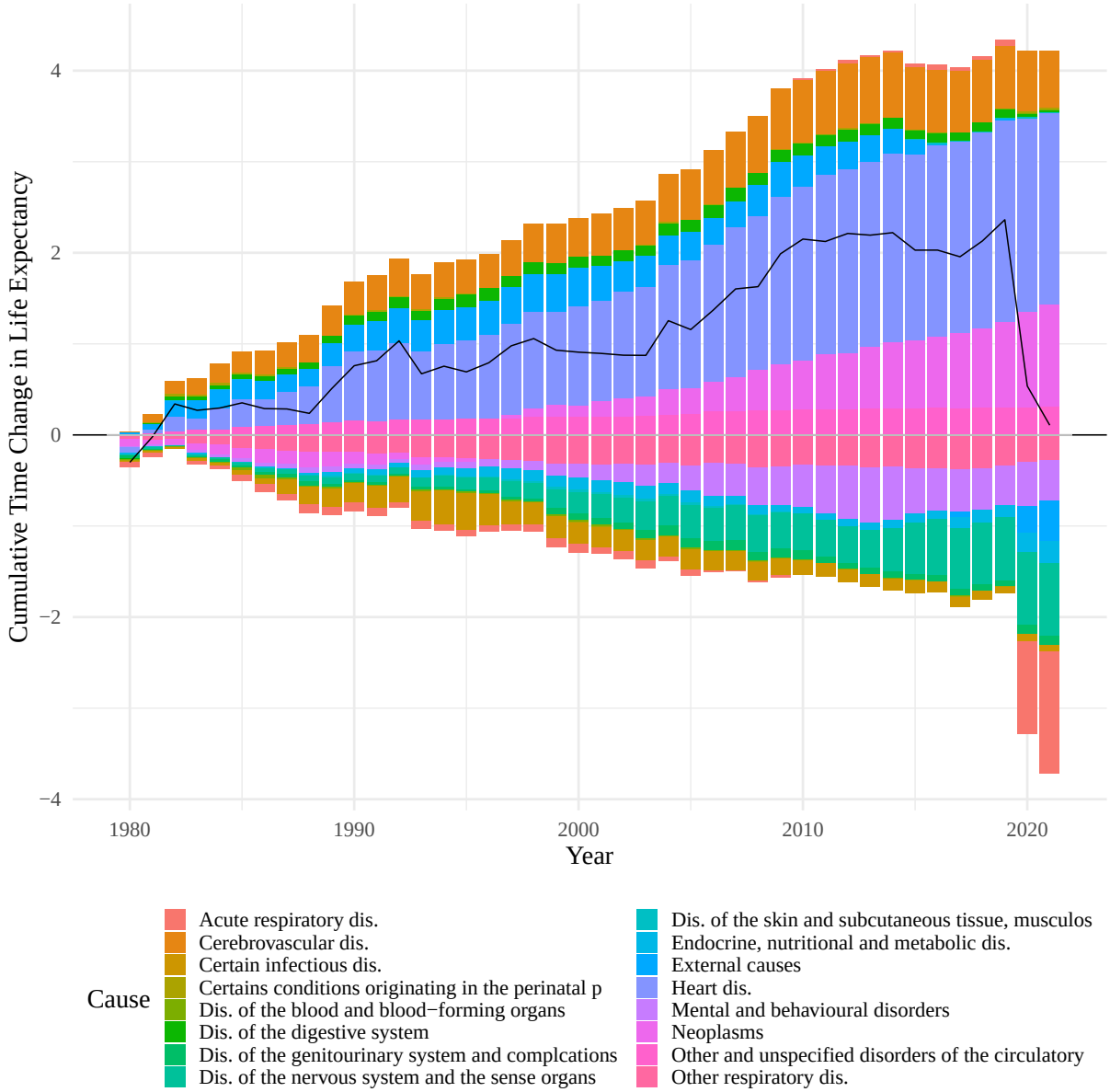


Figure 4:

Cause- and Parameter-Decomposed Cumulative Time Change in US Female+ Life Expectancy
The panels display the cumulated time changes in life expectancy, decomposed into the cause- and parameter components Δ_i^A, Δ_i^l and Δ_i^s for US females from 1980 to 2020. The black line displays the cumulated change due to each parameter, aggregated over all causes: $\sum_{i=1}^{n} \Delta_i^A$ and equivalently for Δ_i^l and Δ_i^s *Note that the panels have different y-scales*

