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The Modelling Cause-of-Death Mortality and the Impact of Cause-Elimination

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Modelling Cause-of-Death Mortality and the Impact of Cause-Elimination

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Abstract

Changes in underlying mortality rates significantly impact insurance business as well as private and public pension systems. Individual mortality studies have data limitations; aggregate mortality studies omit many relevant details. The study of causal mortality represents the middle ground, where population data is used while cause-of-death information is retained. We use internationally classified cause-of-death categories and data obtained from the World Health Organization. We model causal mortality simultaneously in a multinomial logistic framework. Consequently, inherent dependence amongst the competing causes is accounted for. This framework allows us to investigate the effects of improvements in, or the elimination of, cause-specific mortality in a sound probabilistic way. This is of particular interest for scenario-based forecasting purposes. We show the multinomial model is more conservative than a force-of-mortality approach. Finally, we quantify the impact of cause-elimination on aggregate mortality using residual life expectancy and apply our model to a French case study.

Keywords: Cause-of-Death Mortality, Multinomial Logistic Regression, Cause-Elimination, Life Expectancy, Mortality Forecasts

JEL Classifications: G22, G32, C51, C18

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1 Introduction

Changes in underlying mortality rates significantly impact insurance business as well as private and public pension systems. Individual mortality studies have data limitations; aggregate mortality studies omit many relevant details. The study of causal mortality represents the middle ground, where population data is used while cause-of-death information is retained. We add to the area of causal mortality modelling by employing the multinomial logistic model. The aim of this analysis is to facilitate a better understanding of the potential impact of the various causes-of-death on aggregate mortality. The multinomial logistic model provides a framework for cause-elimination that has not sufficiently been explored in the literature and thus, gives a new perspective on the potential impacts of medical innovations. Our modelling approach is of interest to life insurance companies that are developing mortality scenarios. It also provides important insight into possible future mortality evolutions that should be considered in current European pension reforms.

The multinomial *logistic* model (also known as multinomial *logit* model) is typically used to detect factors that significantly influence a polytomous response; that is, a response with several competing outcomes. Several applications of the multinomial logistic model have been undertaken with respect to cause-of-death analysis over the past ten to twenty years. Examples include the work of Eberstein *et al.* (1990), who used eight categorical and continuous independent variables, including marital status, education, and birth weight, to model five infant cause-specific mortality rates. Furthermore, Lawn *et al.* (2006) applied the multinomial logistic framework to model the distribution of neonatal deaths in countries with poor data; see Johnson *et al.* (2010) and Liu *et al.* (2012) for related work. Bradshaw *et al.* (2003) and Shahraz *et al.* (2012) employed a multinomial model to redistribute the unknown or ill-defined deaths; see Murray *et al.* (2006) for a related application to ill-defined causes. Finally, Park *et al.* (2006) incorporated the multinomial logistic framework in the modelling process in order to take into account the impact of the tenth revision of the international classification of diseases.

However, none of these studies investigated cause-specific mortality over the entire age-range; past focus has rather been solely on infant mortality. As mentioned by Foreman *et al.* (2012), *current techniques do not allow for us to take advantage of such modelling advances within a multinomial framework.* This is mainly due to computational power issues. However, this is not an issue when relatively few regressors are included in the model. Furthermore, it is not our primary interest to find the variables that have the biggest impact on cause-specific mortality. We are rather interested in utilizing a framework that accounts for the nature of competing risks, which we believe has not adequately been addressed in the existing literature. Since cause-specific mortality data typically includes two variables of interest, namely age and time, the multinomial logistic model is easy to employ. More importantly, the multinomial logistic framework parsimoniously quantifies the impact in the event that a cause is eliminated; e.g. in case a cure is found for some disease. This significantly broadens the perspective of the analysis.

To put our work in perspective, we briefly review various other cause-specific mortality models that have been investigated by experts in many fields over the past few decades. The appropriateness of decomposing mortality by cause-of-death has even been debated. Some experts have encouraged it, see e.g. Tuljapurkar (1998),

Gutterman and Vanderhoof (1998) and Tabeau *et al.* (2001); whilst others have highlighted its various limitations and risks, see e.g. Booth and Tickle (2008) and Richards (2009).

Among the limitations is to forecast mortality for each cause in isolation and to subsequently aggregate them to derive total mortality rates. For example, McNown and Rogers (1992) used univariate ARIMA models to forecast the parameters of the multi-exponential function fitted to the age pattern of mortality. Based on data from 1960 to 1975, they forecasted four main causes-of-death (heart diseases, cancer, vascular diseases, accident and violence) until 1985. Further examples include Caselli (1996) and Wilmoth (1996), who considered the impact on projections of modelling mortality rates by cause; Rogers and Gard (1991), who illustrated several applications of the Heligman-Pollard function, one of them used to forecast cause-specific mortality; Wilmoth (1995), who demonstrated that for specific models, such as the Lee-Carter model, overall mortality forecasts were consistently lower than the sum of mortality forecasts based on a cause-specific approach; and Caselli *et al.* (2006) and Tabeau *et al.* (1999), who compared several forecasting approaches applied to aggregate as well as cause-specific mortality rates.

Various models have been developed that attempt to account for the dependence between causes. For example, a cause-specific mortality rate is correlated to another cause through their joint dependence on some individual risk factors (covariates); see e.g. Rosén (2006) and Manton (1986). Frailties have also been widely employed to account for heterogeneous populations, where the dependence assumptions between the various causes are determined by the joint distribution of the frailties; see e.g. Manton *et al.* (1986), Vaupel and Yashin (1983) and Hougaard (1984). Multiple cause-of-death data provide another tool to investigate links between various causes and help to determine a *pattern of failure*, defined as a combination of causes that result in death; see e.g. Manton *et al.* (1976); Manton and Poss (1979); Manton *et al.* (1980a); Manton and Myers (1987). More recently, copulas have been used to model the dependence between competing risks; see e.g. Kaishev *et al.* (2007).

Unfortunately, in practice the techniques that account for cause dependence are seldomly employed. Using individual risk factors or multiple causes requires significant additional data that is not readily available, whilst the frailty model and the copula framework are more complicated and less convenient to apply. The distribution of the frailty or copula must be specified, which introduces further assumptions.

Therefore, the most widely used approach is still based on a model developed more than 40 years ago by Chiang (1968), in which causal forces of mortality are used and which provides insight into causal trends and partially addresses dependency issues; see also e.g. Prentice *et al.* (1978). For example, since 1968, the United States decennial life tables have been published with a special report that focuses on the impact of eliminating causes using Chiang's approach (that will be also referred to as the *force-of-mortality approach*); see Bayo (1968), Greville *et al.* (1975), Curtin and Armstrong (1988), Anderson (1999). Furthermore, United States official projections and forecasts of the Institute of Actuaries of Australia are both performed under the force of mortality approach; see Wong-Fupuy and Haberman (2004) and LIWMPC Longevity Research Group (2010), respectively.

The multinomial logistic model provides an interesting alternative framework to Chiang's model since it naturally incorporates cause dependence. Consequently, it

provides a very convenient tool for cause-elimination studies that complements the traditional approach of Chiang, widely used in past cause-elimination and cause-delay models; see e.g. Keyfitz (1977), Tsai *et al.* (1978), Manton *et al.* (1980b), Olshansky (1987, 1988), and Manton (1991), amongst others. Therefore, we provide a comparison between the multinomial approach and the force of mortality approach. After introducing the methodology with respect to the multinomial logistic model and life expectancy calculation in Section 2, total and partial cause-eliminations are introduced by shocking causal mortality in Section 3. It is shown that the multinomial logistic model is less optimistic, that is, survival increases less, than in a force of mortality approach. In Section 4, we illustrate the model in a case study using data for France obtained from the World Health Organization. Section 5 concludes the paper.

2 Methodology

In this section we provide the theoretical details of our proposed causal mortality model. We also outline the construction of residual life expectancy using an abridged life table.

2.1 Multinomial Logistic Model

Multinomial logistic regression techniques are catered to modelling probabilistic response variables for competing outcome categories; see e.g. Menard (2002) and Borooah (2002). Let $D_i(x,t)$ denote the random deaths from cause i for age x at time t and let $L(x,t)$ denote the subsequent survivors that complement the deaths. Furthermore, consider n causes and define $Y(x,t)$ to be the vector of cause-specific deaths and survival. We have

$$Y(x,t) = (D_1(x,t), D_2(x,t), \ldots, D_n(x,t), L(x,t))'.$$

We assume $Y(x,t)$ follows a multinomial distribution, whose probability mass function, omitting the arguments (x,t), is given by

$$\Pr[D_1 = d_1, \ldots D_n = d_n, L = l] = \frac{E!}{d_1! \cdots d_n! l!} q_1^{d_1} \cdots q_n^{d_n} p^l,$$

where,

$$\sum_{k=1}^{n} q_k(x,t) + p(x,t) = 1,$$

such that $q_i(x,t)$ describes the probability of death as a result of cause i, $p(x,t)$ the probability of survival, and

$$E(x,t) = l(x,t) + \sum_{k=1}^{n} d_k(x,t),$$

where $l(x,t), d(x,t)$ are realizations of the random variables $L(x,t), D(x,t)$, and the resulting measure of exposure is given by $E(x,t)$. We adopt survival as the baseline category in the multinomial logistic framework, which produces the model

$$\log \frac{q_i(x,t)}{p(x,t)} = X(x,t)\beta_i, \quad i = 1, \ldots, n,$$

where $X(x,t)$ is the design matrix that specifies how covariates are used in the regression formula and β_i the regression parameters especially suited to cause i. Given the regression parameters and the design matrix, the probabilities are given as follows:

$$\begin{aligned} q_i(x,t) &= \frac{\exp\{X(x,t)\beta_i\}}{1+\sum_k \exp\{X(x,t)\beta_k\}}, \quad i=1,\ldots,n, \\ p(x,t) &= \frac{1}{1+\sum_k \exp\{X(x,t)\beta_k\}}. \end{aligned}$$

2.2 The Regression Formula

Given the multinomial framework, we address the structure of the regression formula. The regression links our response to any potential covariates and is typically some combination of age, period, and cohort. There is a vast literature which investigates this component of aggregate mortality modelling starting with the seminal work of Lee and Carter (1992); in addition, some overviews are provided by e.g. Cairns *et al.* (2011) and Haberman and Renshaw (2011).

The nature of our data suggests the exclusion of any overt cohort covariate. The practical reason is twofold. First, we generally have a limited number of periods, which hinders our ability to identify any significant cohort trend. Second, cause-specific data are presented in age-groups and therefore, the age-groups would need to be converted to single ages before any consideration of cohort could be taking into account. There is also an overriding theoretical reason why we avoid cohort considerations. Namely, causes-of-death have an intuitive relationship with periodic developments, particularly due to medical innovations.

Whether a covariate should be treated as categorical or continuous is a second point of consideration. Categorical covariates offer more flexibility but can overburden the model. We consider categorical *age* and continuous *period* covariates. Categorical age is both intuitive and convenient. Intuitive, since it is likely that the various age-groups exhibit contrasting behaviour with respect to the different causes-of-death. Convenient, since we have a limited number of age-groups. Likewise, continuous period is both intuitive and convenient. Intuitive, since mortality over time is typically classified as a *trend* whose underlying behaviour is of a functional form. Convenient, since implementing continuous time avoids resorting to time-series analysis for forecasting purposes. Lastly, to treat *both* age and period as categorical would be most flexible, but would also be suspectible to overfitting.

Finally, it has been observed in the literature that various age-groups react differently to time; see for example, Booth *et al.* (2001). Therefore, we allow for age-period interaction. The linear regression formula we adopt is as follows:

$$\eta_i(x,t) = \beta_{0,i} + \beta_{1,i,x} + f(t;\tilde{\beta}_{i,x}),$$

where

$$\eta_i(x,t) = \log \frac{q_i(x,t)}{p(x,t)}.$$

Note that the linear regression parameters are distinct for each cause i. Furthermore, the subscript x on $\beta_{1,i}$ and $\tilde{\beta}_i$ indicates the relevant age-group and the tilde on $\tilde{\beta}_i$ signifies it is a vector of parameters. Parameters are estimated using maximum likelihood.

2.3 Residual Life Expectancy

In order to present easily-interpretable outcomes we choose to use (residual) life expectancy. Since we work with age-groups rather than individual ages, we make use of the abridged life table method; see e.g. Chiang (1984). This method mirrors that of a standard life table, with some modifications to allow for the interval age-groups. It requires an assumption on the relationship between central and crude mortality rates governed by a parameter denoted a_x. This parameter takes the interpretation of the average proportion of the year lived for those that died. Throughout the paper, we assume $a_x \equiv 0.5$. Even if this is an assumption that could be challenged for infant mortality, it is widely used and accepted for adult age mortality, which is the focus of this paper.

3 Causal Mortality Shocks

A particular interest in the field of mortality concerns the impact of medical innovation in the form of cures and any corresponding increase in longevity; the cure for cancer being a particularly prevalent example. In contrast to the study of aggregate mortality, our causal approach provides the framework in which valuable insight can be gained.

In this section, we outline how causal mortality is shocked in the multinomial logistic model, and compare it with the approach based on modelling forces of mortality. By causal mortality shock, we mean that mortality for a specific cause suddenly increases, decreases or is eliminated due to some event, such as an epidemic or the discovery of a new cure. The remaining cause-specific mortality rates are subsequently also affected by the change applied to the shocked mortality.

3.1 Shocks in the Multinomial Model

First, we acknowledge the possibility that the elimination of a cause can initiate a marked increase in some causes, whilst decreasing or not affecting others. However, any such relationship is, strictly speaking, unobservable. To understand these particular relationships is a non-trivial matter and is not the aim of this paper. In this paper, we approach the problem from a probabilistic point of view with no prior knowledge. Hence, if one of the competing outcome categories is eliminated, we assign its probability proportionally to the other outcomes, where survival is merely one of these outcomes. That is, although survival probability will certainly increase as a result of a cure for cancer, it will not do so on a one-to-one basis with the decrease in cancer-specific mortality.

Suppose we introduce a shock $\rho_i \geq 0$ to cause i, where values of $\rho_i > 1$ signify a marginal increase in mortality, and vice versa. Note that $\rho_i = 0$ corresponds to the elimination of deaths by cause i. The resulting probabilities are adjusted as follows:

$$\begin{aligned} q_i(x,t) &= \frac{\rho_i \exp\{X(x,t)\beta_i\}}{1+\sum_k \rho_k \exp\{X(x,t)\beta_k\}}, \quad i = 1,\ldots,n, \\ p(x,t) &= \frac{1}{1+\sum_k \rho_k \exp\{X(x,t)\beta_k\}}. \end{aligned}$$

From the above, it is evident that we adjust for mortality shocks on an *annual* probability basis. Previous studies have considered the effects of mortality shocks

on an *instantaneous* probability basis; see e.g. Chiang (1968), Tsai *et al.* (1978), Manton *et al.* (1980b). That is, such studies have adjusted the causal *force of mortality*, which is representative of instantaneous probability of death by cause. Consider the survival probability as written in terms of the force of mortality:

$$p(x,t) = \exp\left[-\int_0^1 \mu(x+s,t)ds\right],$$

where $\mu(x,t) = \sum_k \mu^{(k)}(x,t)$. That is, the total force of mortality, $\mu(x,t)$, is the addition of the forces of mortality attributed to each cause. The effects of causal mortality shocks are imposed by shocking the appropriate component of the force of mortality. For example, cause j elimination is achieved by removing the relevant component of the total force of mortality and subsequently recalculating the survival probability; resulting in:

$$p(x,t) = \exp\left[-\int_0^1 \sum_{k\neq j} \mu^{(k)}(x+s,t)ds\right].$$

Compared with our annual approach, probability redistribution on an instantaneous basis favors survival. In other words, when cause j is eliminated in our method, deaths from causes $i \neq j$ increase comparatively more and survival increases comparatively less than previous findings that modelled causal forces of mortality. A formal proof is provided below.

3.2 A Comparison of Annual and Instantaneous Mortality

In this section we compare the impact of cause-elimination on the survival probability under the annual approach (based on the multinomial logistic model) and the instantaneous approach (based on force of mortality modelling). We show that under cause-elimination, the instantaneous approach increases survival comparatively more than the annual approach.

Given the force of mortality, a survival probability may be written as

$$p(x,t) = \exp\left[-\int_0^1 \sum_{k} \mu^{(k)}(x+s,t)ds\right] = \prod_i p'_i(x,t),$$

where $p'_j(x,t)$ is the *net* survival probability for cause j,

$$p'_j(x,t) = \exp\left[-\int_0^1 \mu^{(j)}(x+s,t)ds\right].$$

The net survival probability is interpreted as the survival probability if no causes-of-death other than cause j exist, as opposed to the *crude* survival probability, $p_j(x,t) = 1 - q_j(x,t)$, that competes with other causes. In the instantaneous approach, the elimination of cause j results in

$$p^{(-j)}(x,t) = \exp\left[-\int_0^1 \sum_{k\neq j} \mu^{(k)}(x+s,t)ds\right] = p(x,t)/p'_j(x,t),$$

where the superscript $(-j)$ in $p^{(-j)}(x,t)$ indicates the elimination of cause j. Under the constant force of mortality assumption,

$$\mu((x+\delta),(t+\tau)) = \mu(x,t), \qquad 0 \leq \delta, \tau < 1,$$

the net survival probability for cause j is known to be,

$$p'_j(x,t) = p(x,t)^{q_j(x,t)/q(x,t)};$$

see e.g. Bowers *et al.* (1986) for a proof. Thus, to find the new survival probability when cause j is eliminated, one has to divide the current survival probability by $p(x,t)^{q_j(x,t)/q(x,t)}$.

In contrast, the elimination of cause j in the annual approach that employs the multinomial logistic model results in a survival probability given by

$$p^{(-j)}(x,t) = p(x,t) \cdot \left[1 + \frac{q_j(x,t)}{p(x,t) + \sum_{k \neq j} q_k(x,t)}\right].$$

Given that both approaches result in a proportional effect on the annual survival probability, we investigate the relation between these two proportions. That is, we show that

$$\frac{1}{p(x,t)^{q_j(x,t)/q(x,t)}} > 1 + \frac{q_j(x,t)}{p(x,t) + \sum_{k \neq j} q_k(x,t)}.$$

By applying some simple algebra, we find the above inequality by proving the following:

$$(1 - q_j(x,t))^{q(x,t)} \quad > \quad p(x,t)^{q_j(x,t)}. \tag{1}$$

Inequality (1) is proved by using Newton's generalized binomial theorem and by noting that $0 < q_j(x,t) < q(x,t) < 1$; see Appendix A for a detailed proof. This implies that under cause-elimination, the instantaneous approach increases survival comparatively more than the annual approach.

4 Case Study

4.1 Data

The World Health Organization (WHO) maintains a comprehensive cause-of-death mortality database (World Health Organization (2012)). This database provides the mid-year population and number of deaths by cause for various countries over the last 50 to 60 years. We obtained data for France from 1952 to 2008. The data is generally divided into five-year age-groups. We consider France due to its size and influence in Europe.

To ensure consistency accross countries, the WHO database classifies the causes according to the International Classification of Diseases (ICD); see Table 1. Under the ICD, the underlying cause-of-death is specified as *the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury.* We consider the five

main ICD causes, which are: diseases of the circulatory system, cancer, diseases of the respiratory system, external causes, and infectious and parasitic diseases. The major causes accounted for more than 80% of deaths in recent years, and made up approximately 60% – 70% of deaths 50 years ago. The cause classification that is used throughout the paper is introduced in Table 2.

Table 1: International Classification of Diseases - Coding system

Causes of death	ICD 7	ICD 8	ICD 9	ICD 10
Circulatory system	A079-A086	A080-A088	B25-B30	I00-I99
Cancer	A044-A060	A045-A061	B08-B17	C00-D48
Respiratory system	A087-A097	A089-A096	B31-B32	J00-J99
External causes	A138-A150	A138-A150	B47-B56	V00-Y89
Infectious and parasitic diseases	A001-A043	A001-A044	B01-B07	A00-B99

The International Classification of Diseases changed three times between 1950 and 2010. The aim of these changes was to account for progress in science and technology and to achieve more refined descriptions.

Table 2: Cause-of-Death Codification

Cause	Code
Infectious and parasitic diseases	1
Cancer	2
Circulatory system	3
Respiratory system	4
External causes	5
Other	6

Some adjustments are made in order to analyze data consistently over time. First, the number of deaths of unknown age are distributed proportionally across the age range, as recommended by the Human Mortality Database (Human Mortality Database (2012)).

Second, age-groups of 85 and above and ages one to four are made. Thus, our database is composed of nineteen groups, the first for infants less than one year old, a second for children aged one to four, thereafter in groups of five years, ending with the group aged 85 and above.

Third, the data contains *central* exposure-to-risk rather than *initial* exposure-to-risk. Consequently, the ratio of cause-specific deaths to exposure produce *central* death rates $m_i(x,t)$ for cause i; see e.g. Pitacco *et al.* (2009) (Ch. 2) for an overview of basic mortality models. Central death rates are typically assumed to relate to death probabilities as follows:

$$q(x,t) = \frac{m(x,t)}{1 + (1 - a_x)m(x,t)}.$$

As mentioned in Section 2.3, we define $a_x \equiv 0.5$ and obtain the relationship

$$q(x,t) = \frac{2m(x,t)}{2 + m(x,t)}.$$

Finally, an adjustment is necessary due to the changes of classification over time. Indeed, the ICD changed three times between 1950 and 2010, from ICD-7 to ICD-10. This was done in order to account for progress in science and technology and to achieve more refined cause descriptions. Consequently, the raw data are not directly comparable over time. To make them comparable, comparability ratios are used.

At the time of a change in classification, some countries recorded the cause-of-death according to both the previous classification as well as the newly adopted one. This *double death registration* makes it possible to analyze the impact of a change of classification. Unfortunately, many countries did not apply this approach for all causes. That is, they recorded deaths under both classifications for a subset of the data. Some countries did not even apply it for a single cause. Therefore, we develop our own comparability ratios in order to smooth the death rates across the classifications. This approach facilitates a consistent analysis across countries should such a comparison be of interest.

The comparability ratios are determined by requiring the average of the death rates over the last two years of a classification to coincide with the average of the death rates over the first two years of the newly adopted classification. That is, a comparability ratio is defined as the sum of the death rates in the first two years of a newly adopted classification divided by the sum of the death rates in the last two years of the previous classification. Since France adopted ICD 8 in 1968, ICD 9 in 1979 and ICD 10 in 2000, three sets of comparability ratios are developed. In order to obtain comparable data over the complete period under consideration, the number of deaths in a new classification is divided by the comparability ratio linking this classification with the previous one, etc. This ensures that mortality rates are continuous at the junction points between classifications. The following analysis is applied to the adjusted death rates for women in France.

4.2 Model Fitting

We begin by studying the observed mortality rates. Figure 1a presents the log-mortality rates over time for the age-group 65–69; and Figure 1b presents them over age-group for calendar year 2008. We opt to display this age-group and calendar year since they are most relevant to retirement systems and most recent, respectively.

Figure 1a suggests minor quadratic behaviour, however, a linear time component appears sufficient to capture the period trend. Furthermore, the plots over age-group exhibit the various familiar components of the average log-mortality age pattern; see e.g. Heligman and Pollard (1980). We adopt the following regression formula for women in France:

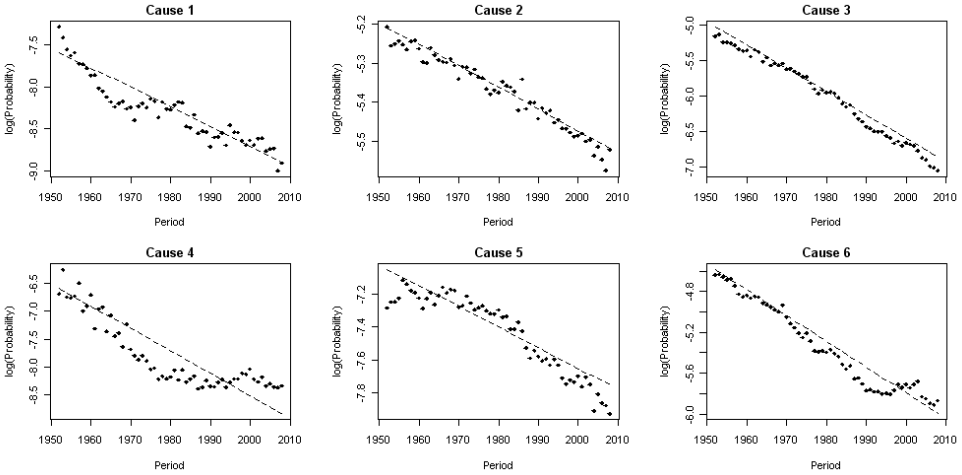
$$\eta_i(x,t) = \beta_{0,i} + \beta_{1,i,x} + \beta_{2,i,x} \cdot t.$$

The resulting fit is presented in Figures 1a and 1b with dashed lines. The dataset contains $7,581$ observations and is fit using 228 parameters. That is, for each of the 6 causes and 19 age-groups, 2 parameters are used: a linear function of time. The fit is generally very good, with variations by cause.

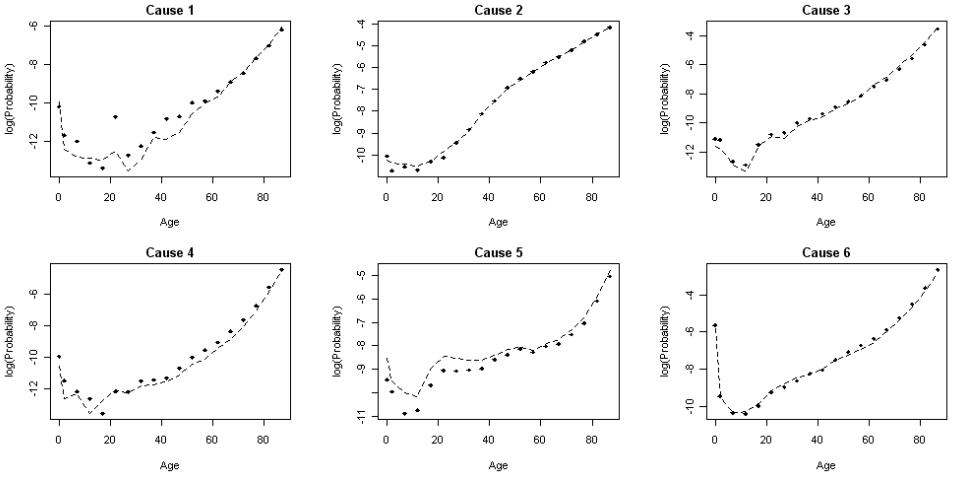
A subset of the regression output, namely the parameter estimates and accompanying standard errors for causes 1–3, are presented in Table 3. Of note is that

Figure 1: Observed (log) Mortality Rates for Women in France

(a) Over time (age 65–69)



(b) Over age-group (calendar year 2008)



the standard errors of the regression estimators are rather large. As a result of large parameter uncertainty, we obtain statistical significance with respect to the F-test but not with respect to the t-test; see statistical references such as Lehmann (1959) for elaboration on the analysis of variance. The test results indicate that the chosen covariates are relevant but estimated with a lack of precision. The plots, of which we present a subset in Figure 1, show that each regression parameter is necessary. Figures 2a and 2b present the (residual) life expectancy at birth and at retirement

Table 3: Regression Parameter Estimates and Standard Errors for Infectious and Parasitic Diseases (1), Cancer (2), Diseases of the Circulatory System (3)

Parameter	Age Group	Cause 1 Estimate	Cause 1 Standard Error	Cause 2 Estimate	Cause 2 Standard Error	Cause 3 Estimate	Cause 3 Standard Error
intercept		-6.4808	11.0090	-9.1761	31.7094	-8.5239	29.2695
age	1–4	-1.6096	28.7990	0.3439	42.3802	-1.6447	62.2479
	5–9	-3.3859	56.6987	-0.3051	47.8764	-2.4305	90.4259
	10–14	-3.8401	67.5356	-0.3768	48.9295	-1.6767	73.0729
	15–19	-3.1747	53.4661	-0.1843	46.3265	-1.4852	58.8771
	20–24	-2.5047	39.8852	-0.0839	44.0787	-1.0398	49.1690
	25–29	-1.7471	33.5019	0.3527	40.1939	-0.6175	45.1855
	30–34	-1.5229	29.4201	0.9086	36.8240	-0.2465	39.7657
	35–39	-1.5923	27.1170	1.5028	34.4846	0.1551	36.7145
	40–44	-1.5785	27.2555	2.0658	33.3180	0.5866	34.4277
	45–49	-1.5345	26.0755	2.5646	32.6741	1.1029	32.4697
	50–54	-1.6048	24.3584	2.9824	32.3589	1.6592	31.2255
	55–59	-1.5112	22.5744	3.3243	32.1661	2.2283	30.4129
	60–64	-1.3390	20.8704	3.6416	32.0389	2.8555	29.8728
	65–69	-1.0809	18.2866	3.9908	31.9483	3.5299	29.5888
	70–74	-0.7616	16.4663	4.3412	31.8809	4.1973	29.4312
	75–79	-0.5057	15.0162	4.6934	31.8338	4.8440	29.3547
	80–84	-0.3434	14.1773	5.0085	31.8034	5.4037	29.3169
	85+	-0.1393	13.3679	5.3489	31.7832	6.0128	29.2958
t		-0.0619	0.5384	-0.0192	1.0673	-0.0551	1.3468
t*age	1–4	-0.0146	1.5816	-0.0086	1.4766	0.0271	2.4092
	5–9	0.0096	2.5651	0.0025	1.5965	0.0198	3.5771
	10–14	0.0167	2.8696	0.0022	1.6334	-0.0001	3.3784
	15–19	0.0027	2.5683	0.0025	1.5457	0.0260	2.3083
	20–24	-0.0011	1.9784	0.0089	1.4383	0.0309	1.9364
	25–29	-0.0325	2.1237	0.0085	1.3222	0.0204	1.8900
	30–34	-0.0262	1.7662	0.0084	1.2205	0.0291	1.6552
	35–39	-0.0043	1.3749	0.0118	1.1461	0.0282	1.5655
	40–44	-0.0063	1.4035	0.0119	1.1128	0.0262	1.5015
	45–49	-0.0011	1.2911	0.0134	1.0939	0.0258	1.4425
	50–54	0.0175	1.0589	0.0125	1.0854	0.0228	1.4079
	55–59	0.0253	0.9410	0.0135	1.0798	0.0216	1.3830
	60–64	0.0285	0.8634	0.0145	1.0762	0.0236	1.3652
	65–69	0.0382	0.7421	0.0134	1.0738	0.0219	1.3567
	70–74	0.0415	0.6805	0.0133	1.0720	0.0244	1.3516
	75–79	0.0505	0.6275	0.0133	1.0707	0.0267	1.3492
	80–84	0.0604	0.5984	0.0145	1.0698	0.0331	1.3480
	85+	0.0748	0.5748	0.0162	1.0692	0.0420	1.3473

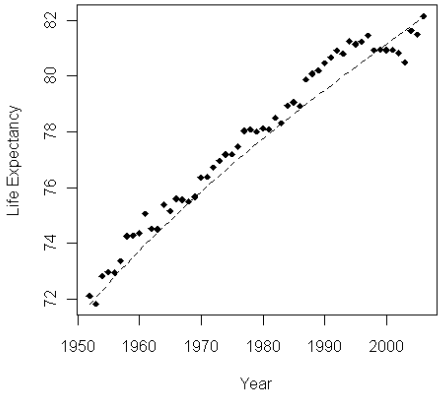
age, respectively, for women in France. The observed life expectancy is plotted with points, the fitted life expectancy with dashed lines. As a result of our model selection criteria, the life expectancy fit is good. The observed life expectancy appears to be decaying, which the fit is able to capture.

4.3 Causal Mortality Shocks

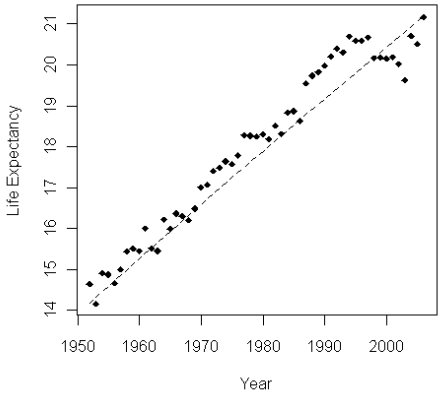
Figures 3a and 3b present the impact of eliminating cancer (cause 2) on life expectancy at birth and retirement age, respectively. The cure for cancer results in an increase of 3.43 years of life for newborns and 2.21 years for 65 year-olds in 2008.

Figure 2: Life Expectancy for Women in France

(a) At birth



(b) At retirement age (65)



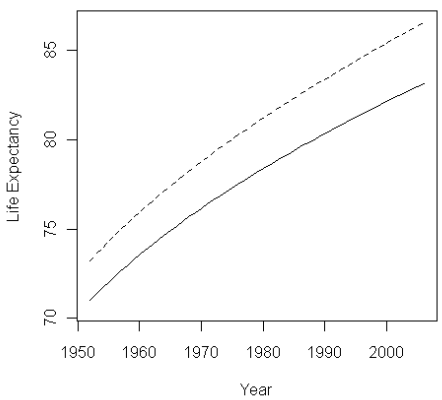
For life insurance companies and pension funds, a cure for cancer would then have dramatic implications.

It is evident that the *hypothetical* gain in life expectancy from eliminating cancer is larger in more recent calendar years; most especially for older ages as demonstrated in Figure 3b. The importance of cancer as a cause-of-death has been increasing with time and is most relevant for older adults. This is intuitive, but difficult to discern from observed data only, such as plots provided in Figure 1.

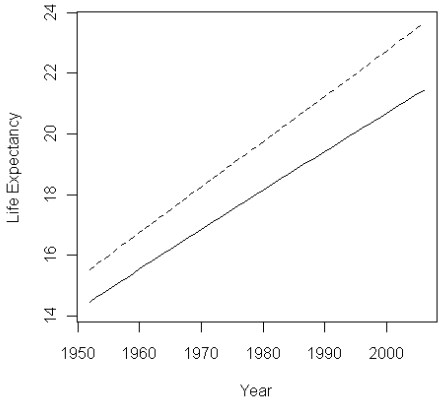
For example, one might perceive a decrease in cancer deaths for a specific age-group. To gain insight into the behaviour of cancer mortality, it should be considered in relation to total mortality. It is plausible that an age-group is transitioning to better overall mortality, but that cancer prevalence is increasing as a cause-of-death, rather than decreasing. Finally, the age-groups must be aggregated to obtain the impact of cancer on life expectancy over time. This is a difficult obstacle that the multinomial model overcomes.

Figure 3: The Impact of Eliminating Cancer on Fitted Life Expectancy

(a) At birth



(b) At retirement age (65)



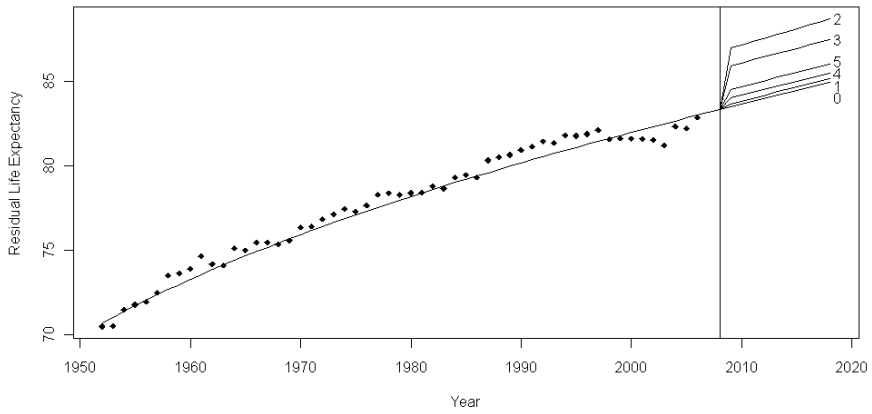
4.4 Forecasting Residual Life Expectancy

Time is treated as a continuous covariate in the model. Therefore, we avoid having to delve into time-series analysis for forecasting purposes. However, as with any form of forecasting, the implications of projections must be carefully considered. In addition to the inherent issues when forecasting, an additional cause for concern in our case study is the large parameter uncertainty. Consequently, we limit the forecasting period to a ten year horizon.

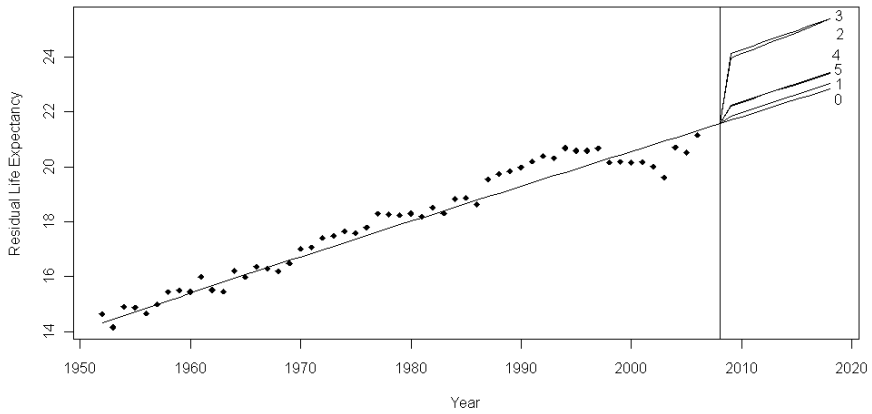
For the forecasting period, we emphasize the uncertainty driven by potential causal shocks rather than those originating from the process and estimated parameters. A *crude* idea of uncertainty is provided by comparing the forecasted life expectancy under the scenario that cause i is eliminated for each i. Figure 4 presents the fitted and forecasted life expectancy, where the forecast labelled i represents the scenario that cause i is eliminated. For example, the scenario of a cure for cancer is represented by forecast 2, which has a very large impact on life expectancy at birth as well as life expectancy at retirement age. The projection labelled 0 represents the scenario of no causal shocks. Deaths from cancer (cause

Figure 4: Forecasted Life Expectancy conjoined with Cause-Elimination

(a) At birth



(b) At retirement age (65)



2) and the circulatory system (cause 3) are especially relevant, which is evident in Figure 4 by the magnitude and sustainability of the increase in life expectancy. Dis-

eases of the circulatory system were the most important causes of death about 50 years ago. While cancer already became the most important cause for middle ages (see Figure 4a), it is expected to become the most prevalent one at older ages in the next 10 years (Figure 4b). Therfore, a cure for cancer will have even more impact in ten years than today, especially at older ages. Deaths from the remaining causes (causes 1, 4, 5) display a similar fit (see Figure 1a), but are less than those from cancer and the circulatory system and therefore less relevant to life expectancy.

5 Conclusions

The aim of this paper is to provide an alternative approach to cause-of-death mortality modelling. This is especially relevant under current European pension reforms. Previous work has considered modelling causal forces of mortality. A consequence of the instantaneous perspective is that survival is treated differently from death. In the multinomial logistic framework that utilizes annual probabilities, survival is a competing outcome and is treated the same way as the other outcomes. Consequently, the annual approach assigns less probability to survival as a result of cause-elimination than does the instantaneous approach. A result of cause-elimination is that the probabilities of the remaining outcomes are adjusted. Without any prior knowledge of the governing behaviour between the various outcomes, we adjust all remaining outcomes similarly; that is, proportional to their probability.

The multinomial logistic framework is easy to implement. It is also easy to quantify the impact of cause-elimination or shocks on mortality metrics such as life expectancy, since the model provides an intuitive framework for any combination of shocks on the various considered causes. Given the accessibility of this modelling framework, it can readily be used in practice. Finally, the framework allows for a straightforward implementation of information with respect to known links between the various causes; although such links are not investigated in this paper.

Treating time as a continuous covariate is appealing since it avoids having to delve into time-series analysis for forecasting purposes and consequently, projections are a trivial exercise. However, as with any form of forecasting, the implications of projections must be carefully evaluated. Thus, a shift from continuous to categorical time is worthy of exploration, and must be carefully considered to avoid violating the law of parsimony.

In addition to exploring potential improvements to the modelling framework, we intend to apply the model to multiple countries and contrast our results with instantaneous modelling approaches. Such a comparison will provide a broadened perspective on the analysis of causal mortality data.

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A A Comparison of Annual and Instantaneous Mortality

We prove Inequality (1) from Section 3.2 by using Newton's generalized binomial theorem. For $0 < a, b < 1$, we have

$$\begin{aligned}(1-b)^a &= 1 - ab + \frac{a(a-1)}{2}b^2 - \frac{a(a-1)(a-2)}{3\cdot 2}b^3 + \ldots,\\ (1-a)^b &= 1 - ab + \frac{b(b-1)}{2}a^2 - \frac{b(b-1)(b-2)}{3\cdot 2}a^3 + \ldots,\end{aligned}$$

such that

$$(1-b)^a - (1-a)^b = \frac{a(a-1)}{2}b^2 - \frac{b(b-1)}{2}a^2 - \frac{a(a-1)(a-2)}{3\cdot 2}b^3 + \frac{b(b-1)(b-2)}{3\cdot 2}a^3 + \ldots$$

Each pair on the right hand side is positive for $0 < b < a < 1$. That is,

$$a(a-1)\cdots(a-k)b^{k+1}(-1)^{k+1} > b(b-1)\cdots(b-k)a^{k+1}(-1)^{k+1}, \quad k \in \mathbb{Z}_+.$$

To show this we note that $0 < b < a < 1$ and $0 < (k-a) < (k-b)$ for $k \in \mathbb{Z}_+$.

$$\begin{aligned} b < a \quad &\Rightarrow b^k < a^k\\ &\Rightarrow (1-a)b^k < (1-b)a^k\\ &\Rightarrow (1-a)\cdots(k-a)b^k < (1-b)\cdots(k-b)a^k\\ &\Rightarrow (a-1)\cdots(a-k)b^k(-1)^k < (b-1)\cdots(b-k)a^k(-1)^k\\ &\Rightarrow (a-1)\cdots(a-k)b^k(-1)^{k+1} > (b-1)\cdots(b-k)a^k(-1)^{k+1}\\ &\Rightarrow a(a-1)\cdots(a-k)b^{k+1}(-1)^{k+1} > b(b-1)\cdots(b-k)a^{k+1}(-1)^{k+1}.\end{aligned}$$

Consequently, we obtain the following inequality:

$$(1-b)^a > (1-a)^b, \qquad 0 < b < a < 1.$$

Inequality (1) is proved by taking $a = q(x,t)$, $b = q_j(x,t)$, and noting that $0 < q_j(x,t) < q(x,t) < 1$.