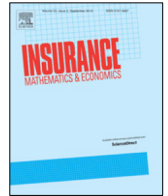




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journal homepage: www.elsevier.com/locate/imeCause-of-death mortality: What can be learned from population dynamics?[☆]Alexandre Boumezoued^b, Héloïse Labit Hardy^{d,*}, Nicole El Karoui^c, Séverine Arnold^a^a Département de Sciences Actuarielles (DSA), Faculté des HEC, Extranef, Université de Lausanne, 1015 Lausanne, Suisse^b Milliman, 14 Rue Pergolèse, Paris 75016, France^c Laboratoire de Probabilités et Modèles Aléatoires (LPMA), UMR CNRS 7599 Université Paris 6, 4 Place Jussieu, 75005 Paris, France^d ARC Centre of Excellence in Population Ageing Research (CEPAR), UNSW Business School, Level 3 East, 223 ANZAC Pde, Kensington 2033, Australia

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ABSTRACT

This paper analyses cause-of-death mortality changes and its impacts on the whole population evolution. The study combines cause-of-death analysis and population dynamics techniques. Our aim is to measure the impact of cause-of-death reduction on the whole population age structure, and more specifically on the dependency ratio which is a crucial quantity for pay-as-you-go pension systems. Although previous studies on causes of death focused on mortality indicators such as survival curves or life expectancy, our approach provides additional information by including birth patterns. As an important conclusion, our numerical results based on French data show that populations with identical life expectancies can present important differences in their age pyramid resulting from different cause-specific mortality reductions. Sensitivities to fertility level and population flows are also given.

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1. Introduction

The studies on cause-of-death mortality have provided a better understanding of the level and the evolution of aggregate mortality rates over the years. At the same time, population dynamics models have been developed in mathematical ecology and demography to model the evolution of the population age structure over time by including both death and birth patterns. Although studies on cause-of-death mortality focused on the impact on mortality indicators, this paper aims to combine these two fields in order to answer the following question: what is the impact of changes in cause-of-death mortality on the whole population age structure?

To address such question, the first step is the modeling of cause-of-death mortality. Each observed death is recorded with a primary cause, such as for example cancer, accident, or a disease related to circulatory or respiratory system. For modeling cause-of-death mortality, one famous framework in actuarial science and biostatistics is the competing risks model. In this framework, each cause of death is modeled at the individual level by a clock, and the death occurs when the first clock rings. In other words, the

lifetime of an individual is modeled as the minimum between competing cause-specific lifetimes. Unfortunately, those cause-specific durations are not observable in the data: in practice we only observe the minimum between the durations related to each cause. Since information on causes of death durations are not observable, assumptions about the dependence between causes of death have to be set in order to capture the marginal distributions and to model changes in cause-of-death mortality. The seminal work of Chiang (1968) on the competing risks theory considered independent causes of death and led to further studies during the last decades. The competing risks framework has been investigated with several dependence structures between the competing lengths of time (see e.g. Carriere (1994), and Dimitrova et al. (2013)). For our study, we focus on the competing risks framework under the independence assumption, although we also briefly survey the debate on the dependence structure. This independence assumption is much used (see e.g. Prentice et al. (1978) and Putter et al. (2007)), and allows us to already highlight the interesting effects of cause removal on the population dynamics.

The previous studies in the cause-of-death literature focused on the impact of cause-of-death removal or reduction on mortality indicators: e.g. life expectancy for Prentice et al. (1978), survival curve for Dimitrova et al. (2013) and death probabilities for Alai et al. (2015). In this paper, the aim is to go further and study the impact on the population age pyramid (the number of individuals by age-class) by means of a population dynamics model. The age pyramid is a crucial quantity of interest since it gives informations

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* Corresponding author.

E-mail addresses: Alexandre.Boumezoued@milliman.com (A. Boumezoued), h.labithardy@unsw.edu.au (H.L. Hardy), elkaroui@gmail.com (N. El Karoui), severine.arnold@unil.ch (S. Arnold).

on how age-classes evolve and interact with each others. To our knowledge, this has not been addressed in the current literature.

To do so, we rely on both deterministic and stochastic population dynamics models. Historically, the first models that addressed the deformation of the age structure of a population over time were deterministic models. The most famous one is called the McKendrick–Von Foerster model (see McKendrick (1926) and Von Foerster (1959)). Based on death rates by age, and also birth rates, they are able to capture the time evolution of the number of individuals by age-class. For demographic purposes, dealing with gender classification has become natural as census and national data make this distinction between males and females for a long time. It is also crucial since age but also gender is known to have a real impact on mortality. To this aim, there is now a sizable literature on two-sex population deterministic models both with and without age structure (see e.g. Keyfitz (1972) and Iannelli et al. (2005)). The deterministic point of view is useful to understand the evolution of the average age pyramid, and can provide first interesting insights about the impact of cause-of-death reductions on the whole population, based on a fast numerical discretization scheme. But in practice, we are not only interested in the age pyramid, but also in non-linear quantities such as the age dependency ratio. This is defined as the number of people aged above 65 divided by the number of individuals aged between 15 and 65. This indicator is a crucial quantity of interest for pure demographic analysis but also for pay-as-you-go pension systems (see e.g. Mirkin and Weinberger (2001) and Hock and Weil (2012)). The deterministic evolution of the population and the age dependency ratio, interesting in itself, is not realistic for populations with finite size in which individuals have random lifetimes and times of birth. What is then the population dynamics? First insights tell us that the average stochastic dependency ratio, as a non-linear quantity, cannot be equal to the ratio of the average population. Therefore a proper stochastic modeling framework is needed. In fact, in a large population asymptotics, the stochastic behavior can be approximated by its deterministic counterpart, making this approach micro–macro consistent. In other words, at the microscopic scale, the population behaves stochastically, whereas if the number of individuals is large the behavior is described by deterministic equations that are classical in demography. The micro–macro consistency is at the core of what are called individual-based models that have been developed in mathematical ecology (see Fournier and Méléard (2004) and Champagnat et al. (2006)), in particular to understand the evolutionary mechanism. In addition, these models have been developed to take ages into account (see Ferriere and Tran (2009)), which is crucial for our purpose.

These processes have been little used for demographic applications compared to issues linked to biology or evolutionary theory. In particular, to our knowledge none of them address the impact of cause-of-death removals on the population age structure. Importantly for our purpose, we are inspired by the work of Bensusan et al. (2010–2015) (see also Bensusan (2010)) who have extended and used population dynamics models in view of human population applications, including pension systems and longevity risk in insurance. Among the few applications to demographic purposes, note also that an alternative named Markovian binary tree is considered in Hautphenne and Latouche (2012) for demographic applications. Markovian binary trees differ from individual-based models as they involve discrete age-classes and a random aging pattern to preserve the Markov property. The key feature of our approach is to take into account aging in continuous time and to use a microsimulation procedure for the stochastic scenarios. It is interesting to note that microsimulation procedures are used in a discrete time setting by several public institutions to test economic and retirement policies (see e.g. the review by Li and O'Donoghue (2013)). This includes for example the French National Institute

for Statistics and Economic Studies (INSEE) with the model *Destinie* (see Bonnet et al. (1999)). Apart from government bodies, microsimulation approaches have also been used in the demographic and statistical literature. As an example, Hyndman and Booth (2008) used discrete-time stochastic simulations to compute the population evolution and the age pyramid, while aggregate mortality, fertility, and migration rates are forecasted.

The paper is organized as follows. In Section 2, we first describe the competing risks framework for the modeling of cause-of-death mortality, and secondly introduce the deterministic and stochastic population dynamics approaches. Section 3 details the data we use to carry out our study: the World Health Organization data for the cause-of-death mortality rates and the data from INSEE for the birth rates and the initial age pyramid. Section 4 contains our numerical results regarding deterministic and stochastic patterns of the age pyramid and the dependency ratio, as well as sensitivities to fertility scenarios and also to population flows computed from the data. As an important conclusion, our numerical results show that under same life expectancy improvement, the age pyramid can evolve in many ways if different causes of death are reduced.

2. The model

As usual when working with human population, we are concerned with population structured by gender and age, so we make the distinction between the age pyramid of females and the age pyramid of males. It is well known that gender and age induce significant differences on mortality. In the model, each individual, female or male, is exposed to d competing causes of death, such as cancers, diseases of circulatory system, diseases of respiratory system or accidents, well described in Section 3, but may die from any single one of the d causes. After a brief description of the classical competing risks framework in survival analysis, we introduce the debate on the dependence structure between causes of death, and describe the different points of view proposed in the literature. Afterwards, we present the meaning of cause-of-death removal. The population dynamics modeling is described secondly.

2.1. Cause-of-death mortality

Competing risks framework. Standard survival data measure the length of time from the time origin until the occurrence of some events; for human population, one is interested in the duration between the time of birth and the time of death (*frailty time* in other framework). This is called the *lifetime* and is represented by a random variable τ^ϵ , with survival function $S^\epsilon(a) = \mathbb{P}(\tau^\epsilon > a)$, for an individual with gender ϵ ($\epsilon = f$ for female or $\epsilon = m$ for male). This is the probability for an individual with gender ϵ to survive until age a . As usual, the information is often expressed in terms of *hazard rate* (also called *force of mortality* in actuarial sciences), defined as the probability to die before age $a + da$ for an individual alive at age a :

$$\mu^\epsilon(a) = \frac{\mathbb{P}(a < \tau^\epsilon \leq a + da \mid \tau^\epsilon > a)}{da} = -\frac{d \ln(S^\epsilon(a))}{da}. \quad (2.1)$$

At this elementary stage, as in Elandt-Johnson (1976), we assume that in each group, females or males, each individual with gender ϵ is assigned a vector of potential lifetimes $(\tau_1^\epsilon, \tau_2^\epsilon, \dots, \tau_d^\epsilon)$ corresponding to the d causes of death. The d causes of death are modeled by d lengths of time, that is to say d competing clocks, denoted $\tau_1^\epsilon, \tau_2^\epsilon, \dots, \tau_d^\epsilon$. The death occurs when the first event corresponding to one of the d causes of death occurs: that is when the first clock rings. In other words, the lifetime of an individual τ^ϵ is defined as the minimum between the different lengths of time: $\tau^\epsilon = \min_{1, \dots, d} \tau_i^\epsilon$. In terms of survival function, we have

$$S^\epsilon(a) = \mathbb{P}(\tau_1^\epsilon > a, \dots, \tau_d^\epsilon > a). \quad (2.2)$$

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