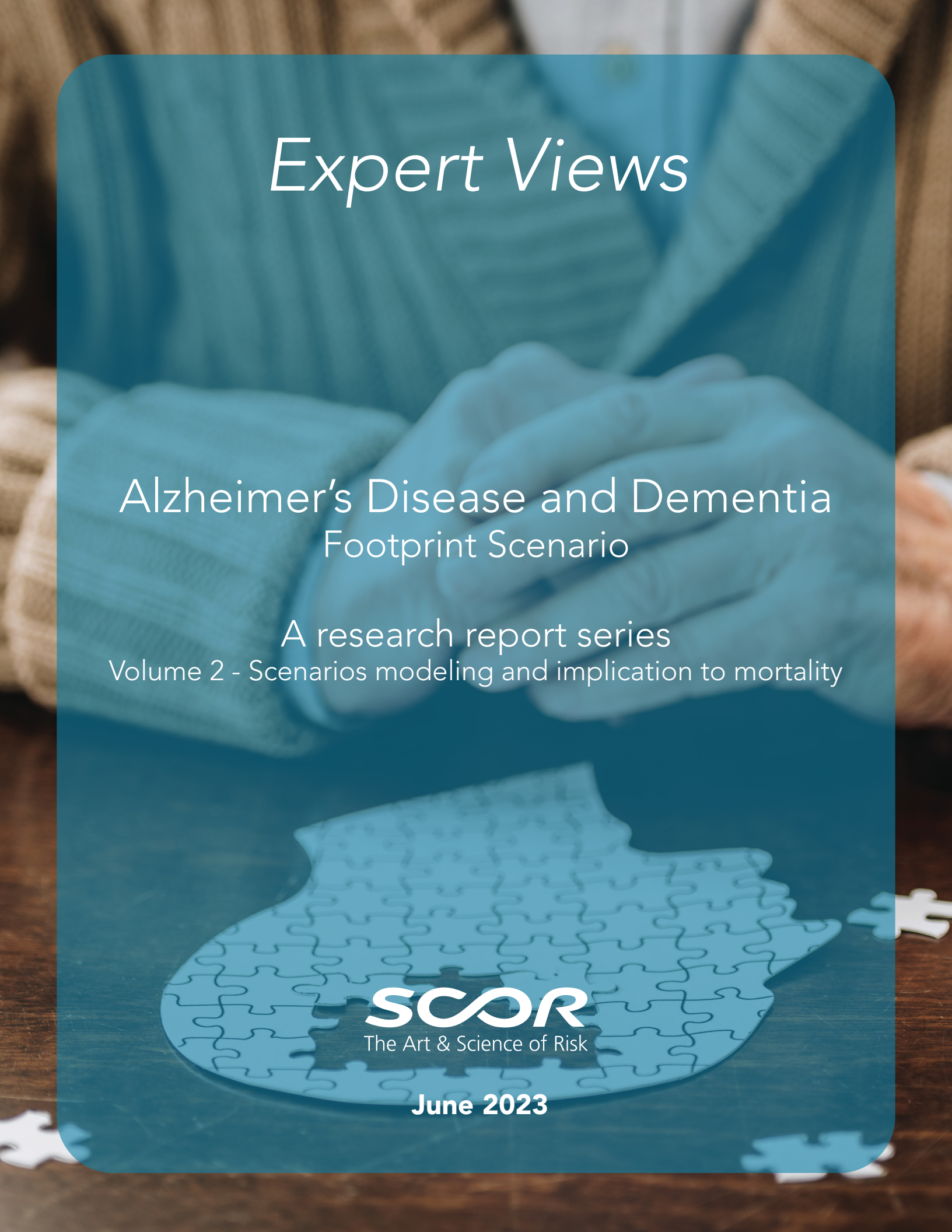

Expert Views

Alzheimer's Disease and Dementia
Footprint Scenario

A research report series
Volume 2 - Scenarios modeling and implication to mortality

SCOR
The Art & Science of Risk

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Contents

Executive summary

This research summary article is the second part of the Alzheimer's disease and dementia footprint analysis series (the first article was published in June 2022). It portrays the impact of possible future medical advancement on a selected population group's life expectancy and implementation of a shock on Alzheimer's and dementia mortality. The study describes the modeling framework, including the competing risks model based on the Archimedean survivor copula from Li and Lu (2019) and the mortality impact of the two selected footprint, or "what-if" scenarios - delaying the onset of the diseases (Scenario 1) and elimination of the diseases (Scenario 2). Our analysis based on the data shows a positive future mortality impact of both scenarios.

Introduction

Alzheimer's disease and dementia are major public health challenges. Fifty-five million people are currently estimated to have dementia worldwide. Of those, 60-70% are Alzheimer's Disease cases, according to the WHO 2023 report.

Although finding the path to cure this disease is not easy, experts are optimistic about breakthroughs in the short to mid-term future. Increasing support for clinical and preclinical research on Alzheimer's and dementia, such as the National Institutes of Health's unprecedented funding of $3.5 billion in 2022, is an encouraging sign. Shekelle et al. (2020) predict that ten breakthroughs are at least 70% likely to occur by 2037. This optimism is also reflected in the clinical pipeline for new therapies addressing disease-modifying biologics, cognitive impairment, and neuropsychiatric symptoms with symptom-reducing agents now in Phase II and III clinical trials as reviewed by Cummings et al. (2021).

2022 marked a year of historic breakthroughs for Alzheimer's disease. Since September 2022, an experimental drug called Lecanemab has clearly shown for the first time its ability to alter the rate of decline in memory and thinking in people with early Alzheimer's disease. Several months later, the US Food and Drug Administration (FDA) approved it after clinical trial results acknowledged the drug's effectiveness.

In October 2022, scientists discovered that it may be possible to identify signs of brain impairment as early as nine years before patients receive a diagnosis for one of the dementia-related diseases. Swaddiwudhipong et al. (2022) findings raise the possibility that, in the future, individuals at risk could be screened to help select those who could benefit from treatments to decrease the risk of developing one of the conditions.

SCOR's Knowledge team has developed an in-depth footprint (i.e., what-if) scenario analysis, using competing risks modeling and implementation of a shock on Alzheimer's and dementia mortality, assessing its impact on life expectancy in multiple scenarios.

This article follows the first part of SCOR's footprint analysis series, Buffet et al. (2022), which focused on the general background and medical discussion on Alzheimer's and dementia. This second part of the series covers the modeling framework and the results of our footprint scenario analysis.

We start by describing the profile of our two footprint scenarios (Section 2). Section 3 covers a description of our modeling approach, which deployed the US mortality data by cause of death which was further developed to capture the mortality dynamics of populations and model the scenarios in the future. The scenarios are applied to US individuals belonging to the highest educational attainment groups (bachelor and graduate degrees), illustrating impacts on one of the major US-insured population groups.

Section 4 will discuss the application and results of the scenario analysis by presenting illustrations on aggregate and cause of death mortality. We will also discuss the impact on the life expectancy of the scenarios, ranging from reductions to the elimination of Alzheimer's and dementia mortality.

Footprint scenarios: description and application

The success of the insurance business heavily relies on proper risk selection and prediction. Therefore, identifying the possible cases ("what-if?") and ensuring that the company's business is sufficiently resilient to withstand those shocks are essential to any insurance risk management practice. At SCOR, we refer to these "what-if" scenarios as "footprints." Our footprint scenarios analysis conveys the idea of the selected scenario leaving its mark ("footprint") on an insurer's performance of protection lines of business, where mortality and morbidity are the main risks.

Designing footprint scenarios enables us to set up hypothetical scenarios to achieve "what-if" analysis most efficiently. For instance, we can select several possible case scenarios by assuming the degree of how current and future progress in disease prevention measures and medical innovations may impact life expectancy and conduct a benchmark analysis.

For this analysis, we have selected two scenarios: 1) decreased mortality due to the delayed onset of the diseases and 2) total risk elimination of the diseases. We believe that those two scenarios are very positive views, considering the current effort and the combined progress being made in prevention measures, innovations in risk detection, and treatments.

Here is the basis of our two scenarios: As most diagnoses of Alzheimer's and dementia take place late in life when the first memory losses appear, research in early risk identification would be among the most active areas in Alzheimer's and dementia science. Early detection alone will not result in better patient outcomes but is expected to result in better care for persons at higher risk for the disease.

Neuroimaging, applications of deep learning, and other AI methods are expected to speed up risk identification. In addition, genetic profiling, identification of new biomarkers, and improving identification of functional and cognitive performance will help diagnose the disease in its earliest stages.

Furthermore, progress in prevention measures such as interventions enhancing or maintaining the cognitive reserve and targeting modifiable risk factors for dementia is predicted to delay the onset and slow the progression of the disease.

Finally, progress in treatments such as tau-directed therapies, anti-neuroinflammatory drugs, antioxidants, stem cell therapies, and drugs' repositioning and repurposing is expected to decrease the rate of progression of the disease, modifying its long-term trajectory. These future treatments would target the disease in its earliest stages before irreversible brain damage or mental decline occurs.

For more information on the most updated developments related to Alzheimer's disease and dementia, please see our Volume 1 report, Buffet et al. (2022), which provides detailed medical information on Alzheimer's and dementia diseases and the discussion covering the future progress in risk identification, risk reduction by prevention and advances in therapeutics.

Based on the above information, SCOR's Knowledge team have assessed the following two scenarios around the future developments of the Alzheimer's and dementia disease:

Scenario 1: A **reduction** in Alzheimer's and dementia mortality due to success in delaying onset and slowing deterioration or
Scenario 2: An **elimination** of Alzheimer's and dementia as a cause of loss of autonomy and mortality.

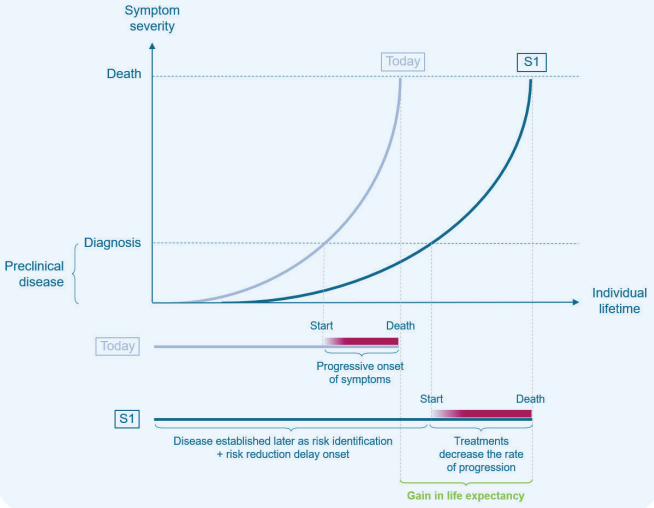
Scenario 1 delays the onset of moderate to severe symptoms, while Scenario 2 assumes a modification of the long-term disease trajectory up to risk elimination. These scenarios do not correspond to the best estimate vision developed by the medical experts, but both are very positive views of possible future outcomes with respect to health impact, particularly in Scenario 2.

The following sections provide details of each scenario.

Scenario 1 : Delaying the onset

Figure 1 illustrates the impact of Scenario 1 (delaying the onset). The figure depicts the development of the symptoms' severity (y-axis) through the individual's lifetime (x-axis). As of today, progressive onset of symptoms is expected after diagnosis up to death. In Scenario 1, the disease is established later as risk identification and risk reduction delay the onset during the preclinical stage of the disease. In addition, risk identification allows identifying individuals at higher risk for guided treatment. After diagnosis, proper treatment decreases the rate of progression of the disease. The difference between the time at death as observed today and expected by Scenario 1 results in a gain in life expectancy.

Figure 1: Illustration of symptoms' slowdown Scenario S1



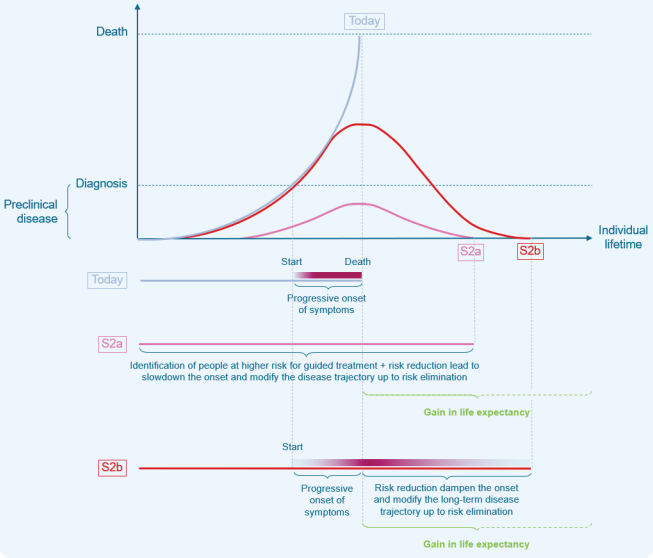
Scenario 1 is implemented on the assumption that advances in risk detection and prevention measures will expand over the next 15 years. Furthermore, treatments decreasing the rate of progression of the disease will lead to a two-thirds reduction in Alzheimer's and dementia mortality by 2035. After this 15-year horizon of improvement, the age-specific probability of death due to Alzheimer's and dementia diseases are assumed to remain at one-third of its pre-shock estimate.

Scenario 2 : Risk elimination

Scenario 2 assumes the elimination of mortality and loss of autonomy from Alzheimer's and dementia within the next ten years. The scenario applies to new cases and individuals in the preclinical stage as well as individuals already diagnosed with Alzheimer's and dementia diseases.

Progress in risk detection leads to the identification of individuals at risk for guided treatment during the preclinical stage of the disease. Risk reduction and prevention measures delay the onset of the symptoms, and treatments of the preclinical disease modify its trajectory. As a result, the incidence rates of new cases shrink to zero. Scenario 2 for new cases and individuals in the preclinical phase of the disease is illustrated by Scenario S2a in Figure 2. In addition, treatments target the disease before irreversible brain damage or mental decline occurs, allowing a modification of the long-term trajectory of the disease up to risk elimination. This is represented by Scenario S2b in Figure 2 for individuals already diagnosed.

Figure 2: Illustration of the risk elimination scenario S2



Application and interpretation of the scenarios

This section features a summary of our modeling process and discusses the application of the two scenarios. We will also show the impacts on the life expectancy gains in each case. For those interested in the detailed technical aspects of the modeling, please refer to the appendix.

Let us start by introducing the US mortality data by cause of death to which the scenarios are applied.

US mortality data by cause of death

We selected the US mortality data for this analysis as it is the largest and most detailed publicly available data. It allows us to study the causes of death dynamics resulting from a shock on Alzheimer's and dementia diseases.

The number of deaths in the US national population originates from the Centers for Disease Control and Prevention (2022). The data includes gender, age, causes of death, educational attainment, and other fields. We also used other data sources, such as population estimates and the proportion of educational attainment provided by the US Census Bureau (2022), to analyze mortality dynamics by cause of death linked to particular educational attainment status. For our illustrations, we used the highest educational attainment levels (graduate and bachelor degrees) covering the period 2001-2019 to derive insured population proxy data.

To classify causes of death used for mortality statistics, we used the International Classification of Diseases (ICD), which provides a system of diagnostic codes. For the illustration of our scenarios, only Alzheimer's and dementia diseases are assessed separately, and the remaining causes of death are grouped into five broad categories: neoplasms, diseases of the circulatory system, respiratory diseases, external causes, and a category "Other" regrouping all deaths not included in the previous categories as shown in Table 1.

Table 1: Causes of death grouping and ICD 10 classification

Causes of death	ICD 10
Alzheimer's and dementia	F01, F03, G20-G21, G30
Neoplasms	C00-C97
Circulatory system diseases	I00-I99
Respiratory diseases	J00-J98, U04
External	U01, V01-Y84
Other	All other causes not included

Modeling approach and methodology

An approach by cause of death is developed to capture the mortality dynamics of populations and model the scenarios in the future. Studying changes in cause-specific (or competing risks) mortality rates provides significant insights as they provide more information than the aggregate mortality data.

Competing risks are important aspects to consider when dealing with mortality by cause of death. Every individual is continuously exposed to many risks of death, such as cancer, heart disease, and accidents. Because death is not a repeated event and is usually attributed to a single cause, these risks compete with one another for the life of an individual.

Using the framework of Archimedean copula developed by Li and Lu (2019), projections of the mortality intensities, i.e., instantaneous mortality rates, by cause of death are obtained by allowing for dependence among competing risks within cohorts. The modeling leads to saving individuals in a cohort from dying of Alzheimer's and dementia and redistributing them to other causes. Under this framework, the scenarios of a reduction or elimination of Alzheimer's and dementia mortality can be coherently applied, and the impact on life expectancy can be assessed.

The detailed modeling is presented in the appendix. An illustration of the approach is shown in Figure 3.

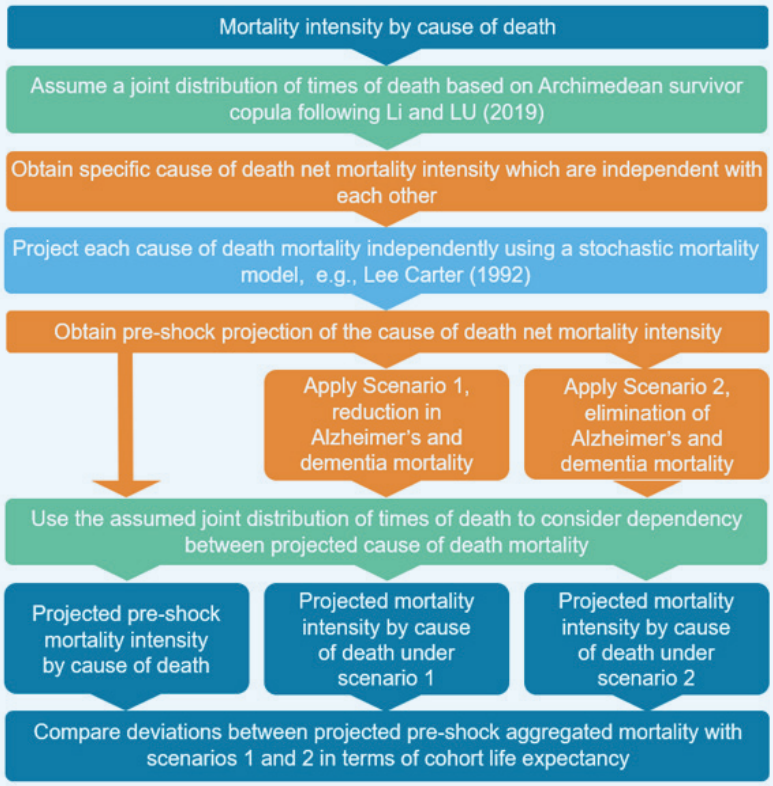
The dependence among competing risks is modeled using the Archimedean survivor copula framework from which the marginal mortality intensities of each cause of death are derived. We used the Clayton's copula. The marginal mortality intensities are then fitted into a stochastic mortality model, e.g., Lee and Carter (1992) model. Based on the estimated stochastic mortality model, pre-shock marginal mortality intensities for each cause are projected. Scenarios 1 and 2 are applied to the marginal Alzheimer's and dementia mortality intensity.

Finally, the resulting post-shock mortality intensities for each cause are obtained and the aggregate future mortality resulting from Scenarios 1 and 2 are deduced. These projections are used to compute the cohort life expectancy for the pre-shock mortality and the scenarios.

Illustration of the scenarios

Our scenario analysis results are shown in Figures 4 and 5. Both illustrate the pre- and post-shock mortality intensities for each cause (left panel) and on aggregate (right panel) for the mortality of the US male highest education attainment population, age 80 for Scenarios 1 and 2, respectively.

Figure 3: Illustration of the approach



Scenario 1, i.e., delaying the onset, shown in Figure 4, assumes that advances in risk detection and prevention measures will expand over the next 15 years, decreasing the rate of progression of the disease. In addition, treatments will lead to a two-thirds reduction in Alzheimer's and dementia mortality by 2035. After this 15-year horizon of improvement, the age-specific probability of death due to Alzheimer's and dementia diseases is assumed to remain at one-third of its pre-shock estimate (illustrated by the dotted dark red line in the left panel).

Scenario 2, i.e., risk elimination, shown in Figure 5, is more extreme, both in terms of the short timeline for the medical breakthrough to be put in place and the significance of the assumed impact. It assumes the elimination of mortality and loss of autonomy from Alzheimer's and dementia diseases within the next ten years. As a result, the decrease in Alzheimer's and dementia mortality rates is larger and more rapid than in Scenario 1, dropping to zero in 2030 as represented by the dotted dark red line in the left panel.

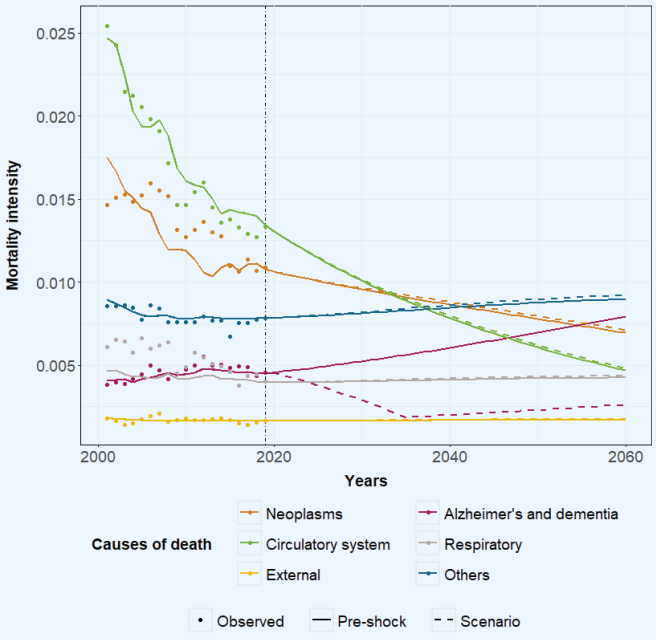
As dependence among competing risks is accounted for, the projected increase of mortality of the other causes of death compared to the pre-shock projection can be observed in the left panels of Figures 4 and 5, illustrated by the dotted lines.

Overall, Scenario 2 is leading to a more significant decrease in mortality than Scenario 1, as shown in the right panels of Figures 4 and 5.

Impact on life expectancy

Due to the competing risk framework, gains in life expectancy resulting from the reduction or elimination of Alzheimer's and dementia mortality are dampened by the increase of mortality of the other causes of death. Table 2 displays the cohort life expectancy for US males from the highest education attainment groups aged 65 and 80 in 2019 and the corresponding gains, i.e., the difference between the pre-shock and scenario cohort life expectancies.

Figure 4: Mortality intensities for each cause (left panel) and on aggregate (right panel) for the mortality US male highest education attainment population, age 80, for Scenario 1



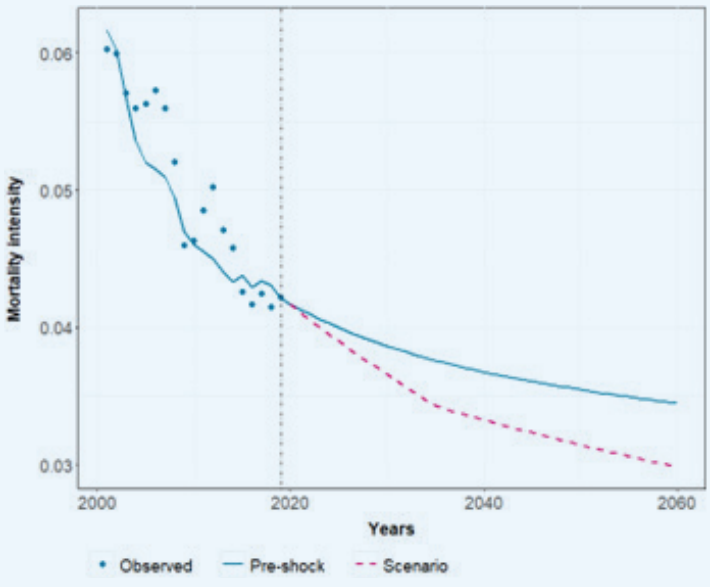


Table 2: Causes of death grouping and ICD 10 classification

Age in 2019	Projections	Cohort life expectancy in years	Gains in months
65	Pre-shock	20.1	
	Scenario 1	20.5	4.1
	Scenario 2	20.7	6.7
80	Pre-shock	8.0	
	Scenario 1	8.1	1.0
	Scenario 2	8.2	2.0

As a measure of comparison of the pre-shock projection resulting from the model, the OASDI trustees report (2021) projected a cohort life expectancy for the general male population aged 65 in 2019 between 18.2 and 19.6 years. The Archimedean survivor copula model projects a cohort life expectancy of 20.1 years for the highest education attainment groups. The difference between the OASDI projections and ours is due to the fact that our analysis is applied to the highest educational attainment groups. Scenarios 1 and 2, assuming advances in risk detection, prevention measures, and treatments decreasing the rate of progression of Alzheimer's and dementia, result in a gain of 4.1 and 6.7 months, respectively.

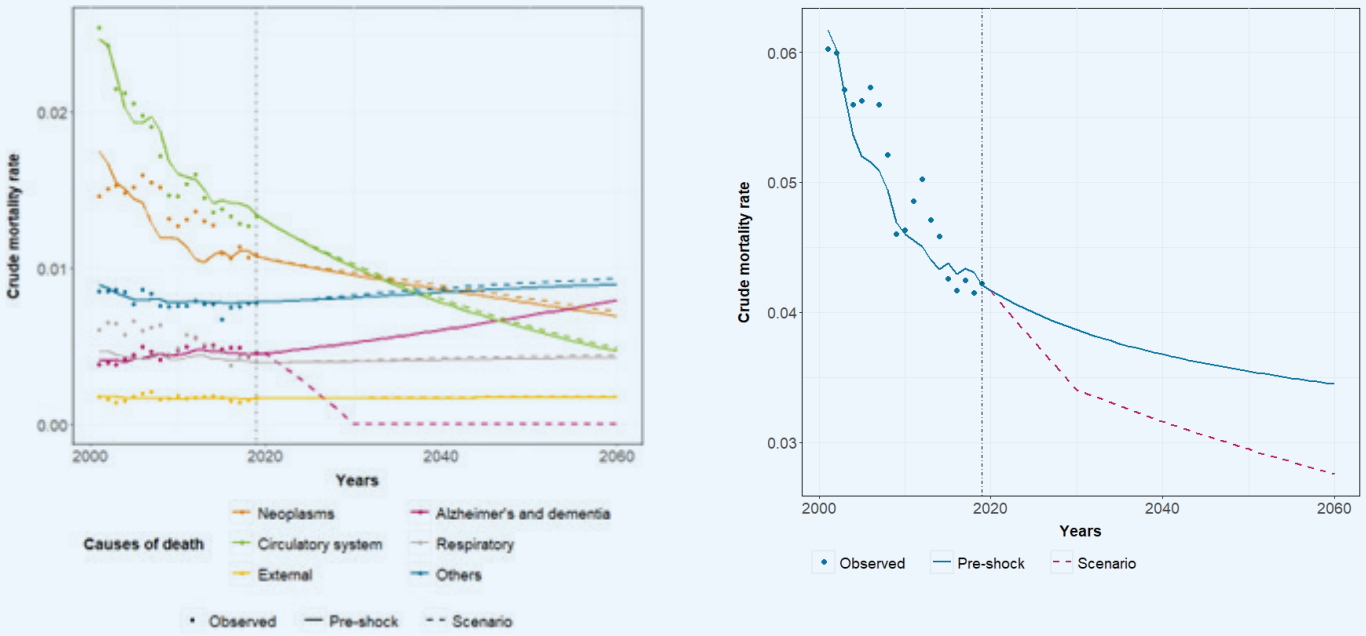
For a US male with the highest educational attainment, age 80 in 2019, the pre-shock projection leads to eight years of cohort life expectancy. Scenarios 1 and 2 increase his life expectancy by one and two months, respectively. At high ages, mortality of causes of death other than Alzheimer's and dementia is relatively high, leading individuals to die shortly after being saved from dying from Alzheimer's and dementia.

Assumptions influencing the results

The magnitude of the shock on Alzheimer's and dementia mortality and its horizon is determined by expert judgment following discussions with medical experts. These scenario assumptions are not the only factors influencing the resulting post-shock aggregate future mortality. What are the other assumptions influencing the results?

The within-cohort dependence among the causes of death in the copula framework is

Figure 5: Mortality intensities for each cause (left panel) and on aggregate (right panel) for the mortality US male highest education attainment population, age 80, for Scenario 2



another parameter set by expert judgment. The current modeling assumes a small dependency between competing risks. Conversely, having a total dependence would mean that all the deaths in a cohort saved from dying of Alzheimer's and dementia would be redistributed to the other causes at the exact same time of death, leading to no gain in life expectancy. Additional study results comparing different dependence structures to the independence assumption are available in Ulcinaite (2023).

The pre-shock Alzheimer's and dementia mortality forecast at high ages is also influencing the outcome. Due to its recent increase, the model projects this upward trend allowing for large impacts for both scenarios. Generally speaking, the larger the increase is, the larger the number of Alzheimer's and dementia deaths saved, and the larger the potential impact of an improvement scenario.

The pre-shock mortality projection of the other causes at high ages also affects the result. To the extent other causes, such as neoplasms or cardiovascular diseases, have a high mortality, the impact of a shock on Alzheimer's and dementia mortality would be relatively small. Individuals would die of neoplasms or cardiovascular diseases shortly after being saved from Alzheimer's and dementia.

The choice of the cause of death grouping is also influencing the results. The outcome could be different if we chose a smaller or greater number of groups than the six that we retained for our analysis.

Finally, the shape of the mortality at very high ages, i.e., the completion assumption of the mortality table, influences the outcome as it defines the survival time of individuals saved from dying of Alzheimer's and dementia.

Conclusion

This second part of SCOR's Alzheimer's and dementia footprint analysis series covered the modeling framework and the mortality impact of the two selected scenarios. The scenarios assume advances in risk detection, prevention measures, and treatments. Scenario 1 models a two-thirds reduction of the Alzheimer's and dementia mortality after a 15-year horizon while Scenario 2 assumes the elimination of Alzheimer's and dementia mortality within the next ten years. Our analysis showed a positive future mortality impact of both scenarios resulting in a cohort life expectancy gain of between 4.1 and 6.7 months for a US male aged 65 in 2019 belonging to the highest education attainment group. Due to the competing risk framework, however, gains in life expectancy resulting from the reduction or elimination of Alzheimer's and dementia mortality are dampened by the increase of mortality of the other causes of death.

Insurance and reinsurance companies have a massive stake in this global effort to fight against Alzheimer's and dementia. It is imperative, as an industry, that we commit ourselves to proactively taking part in R&D initiatives utilizing our data and analytics expertise.

Appendix

This section shares the modeling methodology. We highlight the necessary notations, assumptions and the competing risks model based on the Archimedean survivor copula from Li and Lu (2019).

Mortality intensities and joint survival times

The concepts of crude and net mortality are introduced hereafter followed by the assumptions regarding the dependence structure between the survival variables.

Each individual in a population is assumed to be exposed to m causes of death and may die from any one of these causes. The total lifetime of an individual, T, is given by the minimum of the m cause-specific lifetimes as:

$$T = \min(T_1, \dots, T_m).$$

In the competing risk framework, the observed cause of death is then the one corresponding to the minimum of the m stochastic lifetimes associated with the causes of death.

The all-causes (aggregate) mortality intensity is the instantaneous probability of death before time $t+u$ for an individual who already lived t years for small interval u:

$$\mu(t) = \lim_{u \to 0} \frac{\mathbb{P}(T \le t + u | T > t)}{u}.$$

For a specific cause, the **crude mortality intensity** $\mu_j(t)$ is the instantaneous probability of death if only one cause j exists, given the individual survives t years:

$$\mu_j(t) = \lim_{u \to 0} \frac{\mathbb{P}(T \le t + u, J = j | T > t)}{u}.$$

The crude cause-specific mortality intensities $\mu_j(t), j=1,\dots,m$, sum up to the aggregate mortality intensity:

$$\mu_1(t) + \cdots + \mu_m(t) = \mu(t).$$

And the observed (crude) survival function is given by the sum of the cause-specific mortality intensities:

$$S(t) = \exp\left(-\int_0^t \mu(s) ds\right) = \exp\left(-\int_0^t \sum_{i=1}^m \mu_i(s)\, ds\right).$$

The **net survival** function of cause T_j is the survival if the risks of death other than the cause j were removed,

$$S_j(t) = \mathbb{P}[T_1 > 0, \dots, T_j > t, \dots, T_m > 0] = \exp\left(-\int_0^t \lambda_j(s) ds\right),$$

where $\lambda_j(t)$ is the net cause-specific intensities of T_j. When studying a hypothetical scenario on a cause of death j, the net cause-specific intensities $\lambda_j(t)$ can be modified to reflect the excess or deficit mortality resulting from adverse events or future medical innovations affecting this specific cause. It is defined by

$$\lambda_j(t) = \lim_{u \to 0} \frac{\mathbb{P}(T_j \le t + u | T_j > t)}{u} = -\frac{\mathrm{d}}{\mathrm{d}t} \log S_j(t).$$

However, the cause-specific $P(T_j \le t+u \mid T_j > t)$ cannot be, in general, estimated from data as only $P(T \le t+u, J=j \mid T > t)$ is observed. In estimating the net mortality intensity, the **joint distribution of the survival times** $(T_1, \dots, T_m)$ denoted by $S(t_1, \dots, t_m)$ should then be considered:

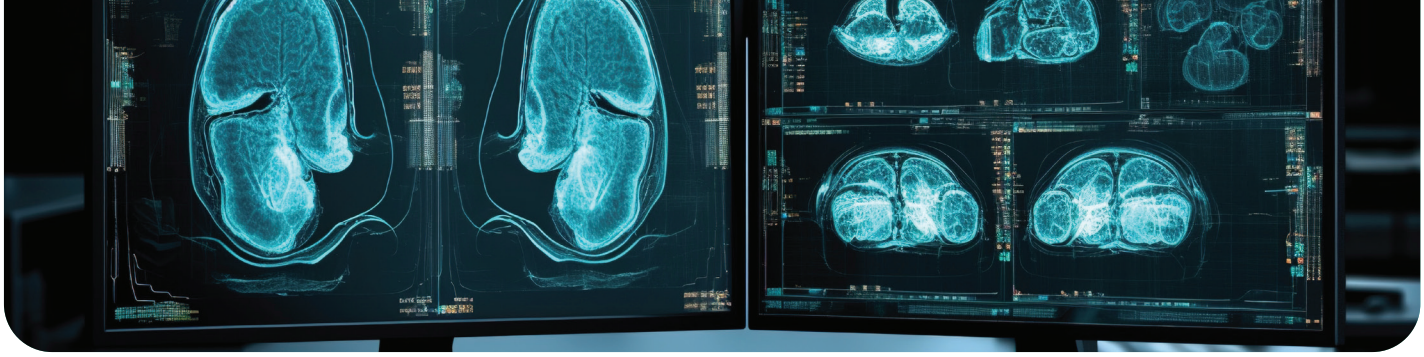
$$S(t_1, \dots, t_m) = \mathbb{P}[T_1 > t_1, \dots, T_m > t_m].$$

The joint distribution of the survival times is related to the crude cause-specific mortality intensities:

$$\mu_j(t) = -\frac{\vartheta}{\vartheta t_j} \log \mathbb{P}[T_1 > t_1, \dots, T_m > t_m] \,|_{t_1 = \cdots = t_m = t}.$$

(1)

However, due to the presence of the competing risks, it is impossible to identify the exact dependency structure between the survival times $T_1, \dots, T_m$ and additional assumptions must be made, see Tsiatis (1975).



Chiang (1968) proposed to consider the causes being independent, greatly simplifying the modeling which turns out to be very popular, see Prentice et al. (1978), Wilmoth (1995), Putter et al. (2007) and Boumezoued et al. (2018, 2019) among others. Under the assumption that the survival times are independent, net and crude cause-specific mortality intensities are equal but this *"may have no resemblance to reality"* as quoted by Tsiatis (1975).

To coherently assess the expected mortality patterns in hypothetical conditions when some causes of death are modified or eliminated, other modeling must be used. One popular approach is to model dependence with survivor copula, see Carriere (1994), Kaishev et al. (2007), Dimitrova et al. (2013) among others. In the following of this article, cause-specific mortality is modeled with **Archimedean survivor copula** as suggested by Li and Lu (2019).

Modeling mortality scenarios using Archimedean survivor copula

The approach assumes that the survival times $(T_1, \ldots, T_m)$ have a joint Archimedean survivor copula. The joint distribution writes:

$$\mathbb{P}[T_1 > t_1, \ldots, T_m > t_m] = \psi\left(\psi^{-1} \circ S_1(t_1) + \cdots + \psi^{-1} \circ S_m(t_m)\right),$$

where the symbol ○ represents the composition of functions and ψ the generator function. In the numerical applications, the Clayton copula is used. Li and Lu (2019) also provide illustrations with the Frank copula.

The Clayton copula is obtained by assuming $\psi(t) = (1+t)^{-1/\theta}$ where θ is a parameter that captures the dependence. The higher the value of θ, the stronger positive dependence between the survival times. When θ approaches 0, the copula reduces to the independent copula.

In a Clayton copula, the joint distribution of the survival times is

$$S(t_1, \ldots, t_m) = \left[S_1(t_1)^{-\theta} + \cdots + S_m(t_m)^{-\theta} - m + 1\right]^{-1/\theta}.$$

(2)

If the joint suvivor copula is Archimedean with generator ψ, Li and Lu (2019) have shown that the net survival function can be determined by the copula and the crude cause-specific mortality intensities:

$$S_j(t) = \psi\left[-\int_0^t \frac{\exp\left(-\int_0^t \sum_{i=1}^m \mu_i(u)\,\mathrm{d}u\right)}{\psi' \circ \psi^{-1} \circ \exp\left(-\int_0^t \sum_{i=1}^m \mu_i(u)\,\mathrm{d}u\right)} \mu_j(s)\mathrm{d}s\right],$$

$$\forall j = 1, \ldots, m.$$

(3)

Using this last result, the procedure of estimating the net mortality intensities and applying modeling mortality scenarios is described below.

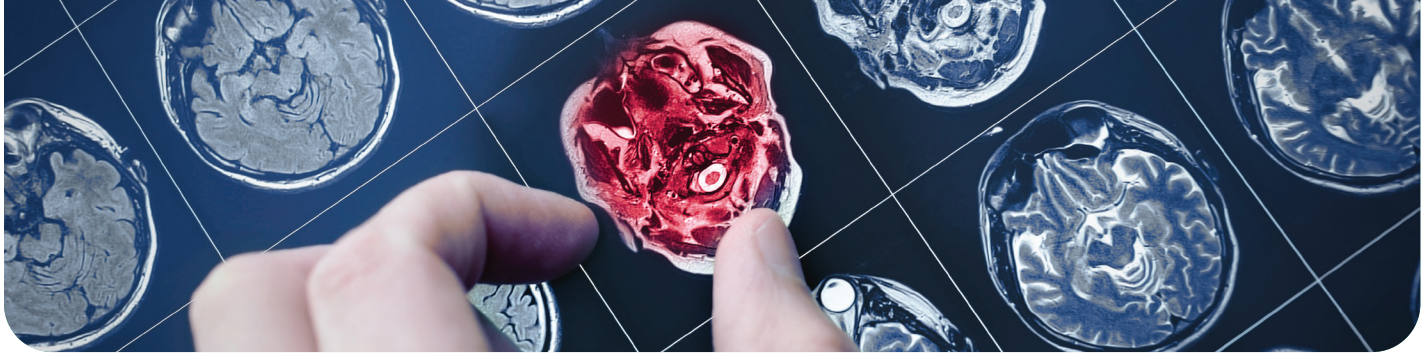
1. The crude mortality intensities $\mu_{j,c,t}$ for each cause of death j, cohort c and calendar year t are obtained by

   $$\mu_{j,c,t} = \frac{D_{j,c,t}}{E_{j,c,t}}, \quad \forall j = 1, \ldots, m,$$

   where $D_{j,c,t}$ and $E_{j,c,t}$ are the corresponding number of death and exposure, respectively.
2. Second, the marginal intensities are derived from the net survival functions $S_{j,c}(t)$:

   $$\lambda_{j,c,t} = -\log \frac{S_{j,c}(t)}{S_{j,c}(t-1)},$$

   where the marginal survival function $S_{j,c}(t)$ are obtained from the crude intensity of each cohort using Equation (1).
3. The Lee and Carter (1992) model is used to forecast the pre-shock marginal intensities for each cause of death separately.



4. Scenarios 1 and 2 are applied on the marginal Alzheimer's and dementia mortality intensity.
5. Lastly, after projecting the net intensities and applied a shock of the net Alzheimer's and dementia mortality intensity, the reverse reasoning is applied to recover the corresponding post-shock crude intensities using Equations (2) and (3). The latter are then used to obtain the aggregate future mortality improvements resulting from the scenario.

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