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*Research Article*

### **The formal demography of kinship V: Kin loss, bereavement, and causes of death**

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# The formal demography of kinship V: Kin loss, bereavement, and causes of death

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## Abstract

### BACKGROUND

The loss of kin by death has medical, psychological, and social effects on other members of a kinship network. Recent formal demographic models can account for deaths of kin, but not causes of those deaths.

### OBJECTIVE

Our objective is to extend the matrix kinship model to analyze losses of any type of kin, at any age at death, due to any cause of death, at any age of a Focal individual.

### METHODS

Given age-specific schedules of risk due to each cause, the projection matrix is enlarged to include multiple absorbing states representing the age at death and the cause of death of kin at each age of Focal. The fertility matrix is enlarged to include births by living kin and to exclude births by dead kin.

### RESULTS

The model provides deaths experienced at each age, and accumulated up to each age, of Focal, by cause of death and age at death. Causes of death are competing risks, permitting the study of how the elimination of one cause displaces bereavement across kin types and age groups of the bereaved. As an example, we analyze kin death experiences attributable to each of the six leading causes of death in the US Non-Hispanic White female population.

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## CONTRIBUTION

Studies of the death of kin and bereavement of survivors can now take into account diverse causes of death, each with its own age schedule of risks. These results may help understand how different causes of death influence kinship structures and the experience of bereavement among surviving kin.

## 1. Introduction

Our goal here is to extend the analysis of the experience of death of kin and bereavement, to include causes of death. We approach this with a demographic model of the kinship network. A person's kinship network plays many roles in their life, shaping child survival, health, educational and social inputs, emotional well-being, and financial transfers (e.g., Mare 2011; Sear and Coall 2011). Social support and exchange can flow through this network of available kin (Daw, Verdery, and Margolis 2016). However, in addition to the positive effects of help and support, negative effects can also propagate through the network, such as the experience of unemployment of kin (Song and Caswell 2022) or of the death of a family member (Verdery et al. 2020).

### 1.1 Bereavement: Its definition and effects

The *Oxford English Dictionary* defines 'bereavement' as "the fact or state of being bereaved or deprived of anything; spec. the fact or state of being deprived by death of a near relative or close friend." Bereavement (the state of loss) is distinguished from grief or other psychological disorders that can follow in the wake of such loss (e.g., Stroebe, Schut, and Stroebe 2007; American Psychological Association 2023). Our goal here is to show how to calculate those losses, for any kind of relative, at any age of death of that relative, at any age of the bereaved individual, for any cause of death, and for both bereavement experienced at specified ages or integrated over the life course.

The demography of bereavement is of more than academic interest because bereavement has effects. The literature on these effects is enormous and compelling. It is not our intention to review this literature; our goal is to provide a methodology for the demography of bereavement, which can provide useful input to studies or calculations of the effects of bereavement. But it is worthwhile to describe some of the effects, because they highlight the importance of some of the things our methodology provides.

The death of a family member potentially affects all the kin of that family member. The death of family members has important effects on physical health (Weitzman and Smith-Greenaway 2020), effects that depend on the type of kin that is lost and the causes

of, and ages at, death. In a large systematic review of the medical literature, Stroebe, Schut, and Stroebe (2007) concluded that bereavement led to increased risks of mortality from many causes, including suicide, to increased ill health as measured by disability and hospitalization, and to increased rates of a long list of psychological effects. In another systematic review, Knowles, Ruiz, and O'Connor (2019) found relations between bereavement and maladaptive changes in the immune system, including systematic inflammation and reduced antibody responses. D'Alton et al. (2022) reviewed bereavement due to death of siblings at ages 0 to 18 and concluded that bereaved siblings were at increased risk for negative physical and psychological outcomes. In a register-based study in Denmark and Sweden, Wei et al. (2022) concluded that bereaved parents, following the death of a child, faced a 35% increase in the risk of heart failure.

Psychological effects of bereavement are well documented (Umberson et al. 2017). Kristensen, Weirsaeth, and Heir (2012) reviewed the psychological effects and concluded that bereavement leads to increased rates of PTSD and major depressive disorders. They also pointed out that the causes of death play an important role, with death due to sudden or violent causes leading to greater psychological effects.

Bereavement also has a variety of social effects. October et al. (2018) reviewed the literature on care for bereaved parents after the death of a child. In addition to the physical and psychological effects already discussed, they reported that bereavement leads to increased unemployment and rates of marital disruption that are up to eight times the normal rates (for a chilling description of this, see Frost 1914). Bereavement reduces opportunities for intergenerational interactions (Mare and Song 2015; Song and Mare 2019; Margolis and Verdery 2019) and the educational attainment and socioeconomic status of those left behind (Patterson, Verdery, and Daw 2020). At the population level, deaths of kin affect family structures (e.g., the population level risk of orphanhood; Lotka 1931) and the persistence or extinction of lineages (Song, Campbell, and Lee 2015; Kolk and Skirbekk 2022).

It is clear from the reviews cited above that the effects of the death of a relative depend on many factors, including the type of kin, the ages of the deceased and the bereaved, and the cause of death. For example, each death to COVID-19 in the United States led to an average of 9 people bereaved of a close family member (Verdery et al. 2020) with the type of kin lost varying by the focal person's age. Snyder et al. (2022) analyzed excess mortality due to COVID-19 in 31 countries and found increases in the deaths of family members, with young individuals more likely to lose grandparents and old individuals more likely to lose siblings. Changes can occur over long time scales (e.g., the global study by Alburez-Gutierrez, Kolk, and Zagheni (2021) of the likelihood of the loss of a child over a period of 50 or so years) or over very short time scales, as in the COVID-19 cases just cited, or the effects of genocide in Guatemala reported by Alburez-Gutierrez (2022). Changes in causes of death can have significant effects on overall life expectancy, as with recent shifts in the US causes of death from those affecting the

middle aged to those affecting children and the young, including gun violence, injuries, and overdoses (Wallace-Wells 2023; Woolf, Wolf, and Rivara 2023).

Beyond these individual effects, bereavement effects are amplified through the kinship network, in what Verdery et al. (2020) call the ‘bereavement multiplier effect.’ Analyzing the multiplier effect requires the calculation of deaths of kin by cause of death. Here, we develop new methods to do exactly that.

Unfortunately, the bereavement literature has few existing tools to understand the demography of such events, especially when considering how different causes of death may contribute. Without understanding the demography, it is difficult to categorize exposure risks or accurately evaluate trends. In this article, we advance a new set of methods from the demography of kinship that can refine such understandings.

## 1.2 Kinship demography

One of the tasks of demography is to understand the population consequences of individual processes (e.g., birth, survival, development, movement), thus providing explanations of the former in terms of the latter and making possible projections of future population based on scenarios of future rates. The models that make this possible can be written as individual-based simulations (e.g., for the case of kinship, Verdery and Margolis 2017; Verdery et al. 2012) or as formal dynamic models (e.g., Goodman, Keyfitz, and Pullum 1974; Caswell 2019).

Simulations and analytical models treat the kinship network as the outcome of a set of demographic rates, just as longevity, population growth and projections, lifetime fertility, and population oscillations and convergence are treated. They make it possible to explore the impact of life cycle structures and parameter values on the network. Models are always conditional on the processes they include and the parameters that define them, which means that comparison with empirical measurements must be interpreted with care. As emphasized by Goodman, Keyfitz, and Pullum (1974), it is not to be expected that a formal demographic model will always duplicate the empirical results; the deviations can provide hints about the operation of factors present in the real population and not in the model.

A recently developed formal demography of kinship using matrix operations has greatly extended the possible analyses of kinship networks. We will refer to this approach as the ‘matrix kinship model.’ The model was first introduced in a one-sex, age-classified, time-invariant form (Caswell 2019), and then extended to include multistate models (Caswell 2020), time-variation (Caswell and Song 2021), and both sexes (Caswell 2022).

The use of the matrix kinship models to analyze deaths of kin is presented in Caswell (2019: Section 4), but so far it has not been possible to distinguish among deaths due to

different causes. Here, we extend the model to incorporate competing causes of death. We present a theoretical approach that allows researchers to track, for a given schedule of vital rates, the expectations of bereavement, either experienced or cumulative, by age of the bereaved, age of the decedent, the bereaved's relationship to the decedent, and the decedent's cause of death. The model accounts for how causes compete, which means that we can study how the elimination of one cause displaces bereavement across kin types and age groups of the bereaved. We show how to compute the numbers of deaths experienced by the bereaved, the numbers of deaths weighted by age class, the proportional distributions of deaths by cause, aggregations of selected types of kin, deaths of kin older and younger than the focal individual, and the approximate prevalence of bereavement (i.e., the probability of experiencing at least one such death). We will show a brief example of the calculations, applying the model to some of the leading causes of death in the US population in the early 2000s.

## 2. The matrix kinship model

We begin by reviewing the matrix model for the kinship network dynamics (Caswell 2019). The matrix formulation makes it possible to incorporate both living and dead kin, and we show how to extend that analysis to include both age at death and cause of death. This shows how different processes contribute to the extent of bereavement, which depends both on how many living kin are at risk of death and on the mortality risks to which they are subject. The example we will present in Section 4 (Non-Hispanic White women in the U.S. in 2003) is from a low-mortality, low-fertility population. Comparisons with other populations will be valuable.

### 2.1 Notation

The following notation is used throughout this paper. Matrices are denoted by upper case bold characters (e.g.,  $\mathbf{U}$ ) and vectors by lower case bold characters (e.g.,  $\mathbf{a}$ ). Vectors are column vectors by default;  $\mathbf{x}^\top$  is the transpose of  $\mathbf{x}$ . The  $i$ th unit vector (a vector with a 1 in the  $i$ th location and zeros elsewhere) is  $\mathbf{e}_i$ . The vector  $\mathbf{1}$  is a vector of ones, and the matrix  $\mathbf{I}$  is the identity matrix. When necessary, subscripts are used to denote the size of a vector or matrix (e.g.,  $\mathbf{I}_\omega$  is an identity matrix of size  $\omega \times \omega$ ). Matrices and vectors with a tilde (e.g.,  $\tilde{\mathbf{U}}$  or  $\tilde{\mathbf{a}}$ ) are block-structured, containing blocks for living and dead kin.

The symbol  $\circ$  denotes the Hadamard, or element-by-element product (implemented by `.*` in MATLAB and by `*` in R). The symbol  $\otimes$  denotes the Kronecker product. The vec operator stacks the columns of a  $m \times n$  matrix into a  $mn \times 1$  column vector. The notation  $\|\mathbf{x}\|$  denotes the 1-norm of  $\mathbf{x}$ . On occasion, MATLAB notation will be used to refer to

rows and columns; for example  $\mathbf{F}(i, :)$  and  $\mathbf{F}(:, j)$  refer to the  $i$ th row and  $j$ th column of the matrix  $\mathbf{F}$ .

## 2.2 The basic model

The model describes the dynamics of the types of kin shown in Figure 1 (which could be extended further in all directions). The focal individual is referred to here as Focal. For full details, see Caswell (2019). Consider a chosen kin type, and let  $\mathbf{k}(x)$  be the age distribution vector of that type of kin at age  $x$  of Focal. The dynamics of  $\mathbf{k}(x)$  are

$$\mathbf{k}(x + 1) = \mathbf{U}\mathbf{k}(x) + \boldsymbol{\beta}(x) \quad (1)$$

$$\mathbf{k}(0) = \mathbf{k}_0. \quad (2)$$

The age distribution vector is projected from  $x$  to  $x + 1$  by a survival matrix  $\mathbf{U}$ , given by

$$\mathbf{U} = \begin{pmatrix} 0 & 0 & 0 \\ p_1 & 0 & 0 \\ 0 & p_2 & 0 \end{pmatrix}, \quad (3)$$

where  $p_i$  is the probability of survival from age class  $i$  to age class  $i + 1$ .

The vector  $\boldsymbol{\beta}(x)$  gives the age distribution of newly recruited kin of type  $\mathbf{k}$  at age  $x$  of Focal. Recruitment of new kin occurs not through the reproduction of kin of type  $\mathbf{k}$ , but by the reproduction of some other type of kin (e.g., granddaughters are the offspring of daughters); it thus involves the fertility matrix  $\mathbf{F}$ . For some types of kin,  $\boldsymbol{\beta} = 0$ ; for example, Focal can accumulate no older sisters after she is born. Thus,

$$\boldsymbol{\beta}(x) = \begin{cases} \mathbf{0} & \text{no recruitment} \\ \mathbf{F}\mathbf{k}^*(x) & \text{recruitment from kin of type } \mathbf{k}^* \end{cases} \quad (4)$$

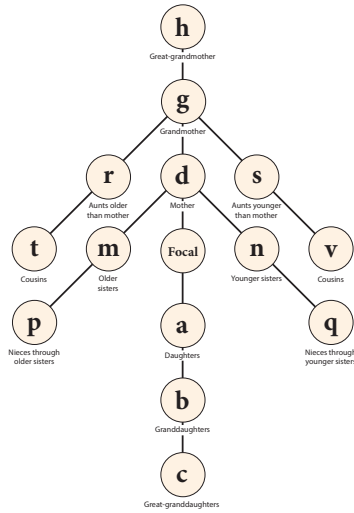
The initial condition  $\mathbf{k}_0$  defines the age distribution of kin at the birth of Focal. For some kin types,  $\mathbf{k}_0 = \mathbf{0}$ ; for example Focal has no daughters at birth. For other types,  $\mathbf{k}_0$  is a weighted sum, over the distribution of ages at maternity, of the kin of Focal's mother at her age at birth of Focal. Let  $\boldsymbol{\pi}$  be the probability vector whose entries are the proportion of births that come from mothers of age class  $i$ . Then,

$$\mathbf{k}_0 = \sum_i \pi_i \mathbf{k}^*(i). \quad (5)$$

Table 1 gives the initial condition and the subsidy vector for each of the types of kin shown in Figure 1.



**Figure 1: The kinship network surrounding the Focal individual. Symbols (a, b, etc.) denote the age structure vectors of each type of kin of Focal.**



Modified from Caswell (2019) based on the network defined in Goodman, Keyfitz, and Pullum (1974) and Keyfitz and Caswell (2005).

**Table 1: The age-classified, time-invariant, one-sex kinship model of Caswell (2019)**

Symbol	Kin	initial condition $k_0$	Subsidy $\beta(x)$
a	daughters	0	$\mathbf{F}e_x$
b	granddaughters	0	$\mathbf{F}a(x)$
c	great-granddaughters	0	$\mathbf{F}b(x)$
d	mothers	$\pi$	0
g	grandmothers	$\sum_i \pi_i \mathbf{d}(i)$	0
h	great-grandmothers	$\sum_i \pi_i \mathbf{g}(i)$	0
m	older sisters	$\sum_i \pi_i \mathbf{a}(i)$	0
n	younger sisters	0	$\mathbf{F}d(x)$
p	nieces via older sisters	$\sum_i \pi_i \mathbf{b}(i)$	$\mathbf{F}m(x)$
q	nieces via younger sisters	0	$\mathbf{F}n(x)$
r	aunts older than mother	$\sum_i \pi_i \mathbf{m}(i)$	0
s	aunts younger than mother	$\sum_i \pi_i \mathbf{n}(i)$	$\mathbf{F}g(x)$
t	cousins from aunts older than mother	$\sum_i \pi_i \mathbf{p}(i)$	$\mathbf{F}r(x)$
v	cousins from aunts younger than mother	$\sum_i \pi_i \mathbf{q}(i)$	$\mathbf{F}s(x)$

## 2.3 Death and causes of death

We now incorporate the death of kin, classified by cause of death or jointly by cause of death and age at death. Competing risk theory for causes of death are familiar in demography (e.g., Chiang 1961, 1968; David and Moeschberger 1978). In this theory, hazards are defined for each cause of death; under the assumption of independence these hazards are additive and the resulting mortality rate is given by their sum. The analysis leads to calculations of deaths under each cause acting alone, all causes acting together, and as a result of removing or modifying one or more causes.

Competing risk theory can be expressed in matrix terms using absorbing Markov chains, with a much greater range and flexibility of calculation (Caswell and Ouellette 2016). We use this approach to include deaths of kin due to causes into the kinship model. Because the types of kin form a connected network, it would be impossible to use the classic multiple decrement life table method to account for deaths.

### 2.3.1 The kinship vector

The kin vector  $\mathbf{k}(x)$  must be enlarged to include both living and dead kin. This can be done in two ways. Let

$$\alpha = \text{number of causes of death} \quad (6)$$

$$\omega = \text{number of age classes.} \quad (7)$$

Then we define a vector  $\mathbf{k}_D(x)$ , of dimension  $\alpha \times 1$ , that gives gives the number of dead kin at age  $x$  of Focal due to each of the  $\alpha$  causes of death. Including more information, we define a vector  $\hat{\mathbf{k}}_D(x)$ , of dimension  $\alpha\omega \times 1$ , that gives the numbers of dead kin at age  $x$  of Focal classified jointly by cause of death and age at death. It is useful to think of  $\hat{\mathbf{k}}_D$  as obtained by applying the vec operator to a matrix giving deaths by age and cause:

$$\hat{\mathbf{k}}_D = \text{vec} \left( \begin{array}{cccc} k_{1,1} & k_{1,2} & \cdots & k_{1,\omega} \\ \vdots & \vdots & & \vdots \\ k_{\alpha,1} & k_{\alpha,2} & \cdots & k_{\alpha,\omega} \end{array} \right) = \begin{pmatrix} k_{11} \\ \vdots \\ \frac{k_{\alpha 1}}{k_{1\omega}} \\ \vdots \\ \frac{k_{1\omega}}{k_{\alpha\omega}} \\ \vdots \\ k_{\alpha\omega} \end{pmatrix}, \quad (8)$$

where  $k_{i,j}$  is the number of kin dying at age  $j$  from cause  $i$ .

The two choices for the vector of dead kin lead to choices for the block-structured vector  $\tilde{\mathbf{k}}(x)$ :

$$\tilde{\mathbf{k}}(x) = \left( \frac{\mathbf{k}_L}{\mathbf{k}_D} \right) (x) \quad \text{or} \quad \left( \frac{\mathbf{k}_L}{\hat{\mathbf{k}}_D} \right) (x), \quad (9)$$

where  $\mathbf{k}_L$  is the vector of living kin and  $\mathbf{k}_D$  or  $\hat{\mathbf{k}}_D$  are the vectors of dead kin. Since the distribution of deaths by cause can be obtained from the distribution of deaths by age and cause, we will focus most of our attention on analyses using  $\hat{\mathbf{k}}_D$ .

### 2.3.2 Kin projection matrices

Projection of the block-structured vector  $\tilde{\mathbf{k}}(x)$  requires a block-structured survival matrix  $\tilde{\mathbf{U}}$ , given by

$$\tilde{\mathbf{U}} = \left( \begin{array}{c|c} \mathbf{U} & \mathbf{0}_{\omega \times \alpha} \\ \mathbf{M} & \mathbf{I} \text{ or } \mathbf{0} \end{array} \right) \quad \text{or} \quad \tilde{\mathbf{U}} = \left( \begin{array}{c|c} \mathbf{U} & \mathbf{0}_{\omega \times \alpha\omega} \\ \widehat{\mathbf{M}} & \mathbf{I} \text{ or } \mathbf{0} \end{array} \right). \quad (10)$$

The matrix  $\mathbf{U}$  in the upper-left corner contains transition probabilities among living states. The mortality matrix  $\mathbf{M}$  or  $\widehat{\mathbf{M}}$  in the lower-left corner of  $\tilde{\mathbf{U}}$  contains transitions from living to dead stages. When deaths are classified by cause of death, regardless of the age at which death occurs, it is given by

$$\mathbf{M} = \begin{pmatrix} m_{11} & \cdots & m_{1\omega} \\ \vdots & & \vdots \\ m_{\alpha,1} & \cdots & m_{\alpha,\omega} \end{pmatrix}, \quad (11)$$

where  $m_{ij}$  is the probability of death from cause  $i$  at age  $j$ . When deaths are classified by both cause of death and age at death, the mortality matrix is

$$\widehat{\mathbf{M}} = \left( \begin{array}{c|c|c|c} \mathbf{M}(:, 1) & & & \\ \hline & \mathbf{M}(:, 2) & & \\ \hline & & \ddots & \\ \hline & & & \mathbf{M}(:, \omega) \end{array} \right). \quad (12)$$

The lower-right diagonal block of  $\tilde{\mathbf{U}}$  in equation (10) determines the way in which deaths are counted. If this block is an identity matrix, then the vectors  $\mathbf{k}_D(x)$  and  $\hat{\mathbf{k}}_D(x)$  record cumulative deaths up to age  $x$  of Focal. If this block is a zero matrix, then  $\mathbf{k}_D(x)$  and  $\hat{\mathbf{k}}_D(x)$  record deaths experienced at age  $x$  of Focal, with no memory of prior deaths.<sup>4</sup>

<sup>4</sup> Although we don't explore it here, it is possible to allow Focal to forget some but not all of the deaths she

This provides access to the distribution of ages of Focal when deaths occur, as examined by Alburez-Gutierrez, Basellini, and Zagheni (2022) for the case of the death of children.

### 2.3.3 Recruitment of new kin

The fertility matrix  $\mathbf{F}$  in (4) is now expanded to a block-structured fertility matrix accounting for both living and dead kin,

$$\tilde{\mathbf{F}} = \left( \begin{array}{c|c} \mathbf{F} & \mathbf{0} \\ \hline \mathbf{0} & \mathbf{0} \end{array} \right). \quad (13)$$

Here, the  $\mathbf{0}$  matrices are of the appropriate dimensions to correspond to the blocks of  $\tilde{\mathbf{U}}$ . The fertility matrix  $\mathbf{F}$  in the upper left describes the production of living kin by living kin. The zero blocks appear because reproduction does not produce dead kin, and dead kin do not reproduce at all.

As in equation (4), the recruitment vector  $\tilde{\beta}x$  is either  $\mathbf{0}$ , for kin types that receive no new individuals, or is obtained by applying  $\tilde{\mathbf{F}}$  to some other type of kin,  $\tilde{\mathbf{k}}^*$ .

### 2.3.4 Initial conditions

Initial conditions are specified as in equation (5); the population of a type of kin at the birth of Focal is either 0 or a weighted average, over the age at maternity, of some other type of kin:

$$\tilde{\mathbf{k}}(0) = \begin{cases} \mathbf{0} & \text{no kin of type } \mathbf{k} \text{ at birth} \\ \sum_i \pi_i \tilde{\mathbf{k}}^*(i) & \text{specified as mother's kin} \end{cases}. \quad (14)$$

For example, the older sisters of Focal at the time of her birth are the children of Focal's mother at the time of Focal's birth. We know that  $\pi$  is the distribution of the age of Focal's mother at her birth, so

$$\tilde{\mathbf{m}}(0) = \sum_i \pi_i \tilde{\mathbf{a}}(i). \quad (15)$$

A decision must be made as to whether to include deaths of kin that occur before the birth of Focal, and thus appear in the initial condition (e.g., "your grandmother died before you were born"). If so, then both living and dead kin are included.<sup>5</sup> If not, then  $\tilde{\mathbf{k}}^*(i)$  should include only the living kin of type  $\tilde{\mathbf{k}}^*$ .

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has experienced. Setting this entry to  $c\mathbf{I}$  for some constant  $c < 1$  would create a memory of deaths that decays exponentially with a half-life of  $\log 0.5 / \log c$  years.

<sup>5</sup> This strategy is used by Jiang et al. (2022) to capture all kin ever born in terms of three components: deaths before the birth of Focal, deaths during Focal's life, and kin still living at the end of Focal's life.

### 2.3.5 Two-sex calculations and the GKP factors

Figure 1 and the model derived from it shows only female kin through female lines of descent. It accounts for daughters but not sons, for those granddaughters that are daughters of daughters but not those who are daughters of sons, and so on. To compare deaths of different types of kin, it is essential to account for both sexes. The two-sex version of the matrix kinship model in Caswell (2022) does so, but requires sex-specific age schedules of mortality and fertility.

In the absence of complete male- and female-specific rates, an approximation due to Goodman, Keyfitz, and Pullum (1974) provides good two-sex results based on rates for just one sex, or both sexes combined. The approximation pretends that the sexes have identical rates and multiplies each type of kin by a scalar factor (called the GKP factors by Caswell (2022)) to obtain the numbers of combined male and female kin. The GKP factors are given in Table 2.

**Table 2: The GKP factors for transformations from single-sex to two-sex kin**

Transformation	GKP factor
daughters $\mapsto$ children	2
granddaughters $\mapsto$ grandchildren	4
great granddaughters $\mapsto$ great grandchildren	8
mothers $\mapsto$ parents	2
grandmothers $\mapsto$ grandparents	4
great grandmothers $\mapsto$ great grandparents	8
sisters $\mapsto$ siblings	2
nieces $\mapsto$ nieces/nephews	4
aunts $\mapsto$ aunts/uncles	8
female cousins $\mapsto$ cousins	8

A calculation in Caswell (2022) shows that the GKP factors give a close approximation to the full two-sex results, even in a case where there exists large male-female differences between both survival and fertility. Because the GKP factors treat the sexes as identical, the calculation cannot incorporate sex-specific causes of death. We leave the incorporation of such comparisons for a future extension.

## 3. Analysis and outcomes

The output of the model is a set of age distribution vectors for each type of kin ( $\bar{\mathbf{a}}(x)$ ,  $\tilde{\mathbf{b}}(x)$ , and so on, in the notation of Figure 1). The vector  $\mathbf{k}_L(x)$  contains the living kin, the vector  $\mathbf{k}_D(x)$  contains the dead kin by cause of death, or the vector  $\hat{\mathbf{k}}_D$  contains the deaths by cause of death and age at death. We turn now to extracting results of interest

from this abundance of information. The analysis of living kin follows the same pattern as described in Caswell (2019), so we focus here on analysis of dead kin.

### 3.1 Numbers of deaths by cause and age

The marginal numbers of deaths classified by cause, summing over all ages at death, is given by

$$\text{number of deaths by cause at age } x = (\mathbf{1}_\omega^\top \otimes \mathbf{I}_\alpha) \hat{\mathbf{k}}_D(x), \quad (16)$$

where  $\otimes$  denotes the Kronecker product. Similarly, the marginal numbers of deaths classified by age, summing over all causes is

$$\text{number of deaths by age at age } x = (\mathbf{I}_\omega \otimes \mathbf{1}_\alpha^\top) \hat{\mathbf{k}}_D(x). \quad (17)$$

A weighted marginal number of deaths classified by cause can be obtained by defining a vector  $\mathbf{c}$ , of dimension  $\omega \times 1$ , whose entries are the weights assigned to the deaths of kin at each age. Then

$$\text{weighted number of deaths by cause at age } x = (\mathbf{c}^\top \otimes \mathbf{I}_\alpha) \hat{\mathbf{k}}_D(x). \quad (18)$$

For example, to count deaths of young dependent kin,  $c_i$  would equal 1 for  $1 \leq i \leq 16$  and zeros elsewhere. Other dependency groups would be defined similarly.

### 3.2 Proportional deaths by cause

A different perspective is revealed by calculating the proportions, instead of the numbers, of kin deaths by each cause. An easy way to do this is to create an array (dimension  $\alpha \times \omega$ ), the columns of which are the deaths at each age:

$$\mathbf{K} = \left( \mathbf{k}_D(1) \quad \mathbf{k}_D(2) \quad \cdots \quad \mathbf{k}_D(\omega) \right). \quad (19)$$

Then divide each column of  $\mathbf{K}$  by its sum, unless the sum is zero in which case set the column to zero. This is easily done by writing

$$\mathbf{K}_{\text{prop}} = \mathbf{K} \mathcal{D}(\mathbf{1}_\alpha^\top \mathbf{K})^+, \quad (20)$$

where  $\mathbf{X}^+$  is the Moore-Penrose pseudo-inverse of  $\mathbf{X}$ .<sup>6</sup> The  $i$ th column of  $\mathbf{K}_{\text{prop}}$  gives the proportions of deaths due to each cause at age class  $i$  of Focal.

<sup>6</sup> Implemented by the function `pinv` in MATLAB and the function `pseudoinverse` in R. This is a trick that automatically sets the columns of  $\mathbf{K}_{\text{prop}}$  corresponding to the zero columns of  $\mathbf{K}$  to zero.

### 3.3 The probability of bereavement

The kinship model provides the mean numbers of deaths (experienced at a given age, or cumulative up to a given age) as a function of age of Focal. But it is sometimes easier to interpret bereavement not in terms of numbers of deaths but on the prevalence of experiencing a death; i.e., on the probability that an individual will have experienced one or more such deaths. See Smith-Greenaway and Trinitapoli (2020) for an analysis of maternal experience of child mortality in Africa phrased in exactly these terms.

The calculation of the mean numbers of deaths experienced by an individual says nothing about the prevalence of bereavement without knowledge of the probability distribution of the numbers of deaths. Obtaining such a distribution requires a stochastic solution of the kinship model or Monte Carlo microsimulation. However, an approximate solution can be obtained by assuming that the numbers of deaths follows a Poisson distribution, as in Song, Campbell, and Lee (2015); Song and Mare (2019). Under that scenario, the prevalence of experiencing a death is

$$P(\text{at least one death})(x) = \mathbf{1}_\alpha - e^{-\mathbf{k}_D(x)}, \quad (21)$$

where the exponential is applied element-wise to the entries of  $\mathbf{k}_D(x)$ .

The Poisson approximation is problematic for parents, grandparents, and great-grandparents. The distribution of dead parents has support only on the set  $\{0, 1, 2\}$ , that of dead grandparents only on the set  $\{0, \dots, 4\}$ , and so on (recall that we are including both male and female kin using the GKP factor approximation in Section 2.3.5). A more appropriate approximation is obtained by treating the number of parental, grandparental, and great-grandparental deaths experienced as binomial random variables with  $N = 2, 4, 8$ , respectively. If (using the symbols in Figure 1)  $d$ ,  $g$ , and  $h$  represent the mean numbers of dead parents, grandparents, and great-grandparents, then the probabilities that Focal experiences at least one death are

$$\text{parents} \quad 1 - (1 - d/2)^2 \quad (22)$$

$$\text{grandparents} \quad 1 - (1 - g/4)^4 \quad (23)$$

$$\text{great-grandparents} \quad 1 - (1 - h/8)^8. \quad (24)$$

Note that when the mean number of deaths is small, the binomial and the Poisson approximations converge. The quality of these approximations is an open research question.

### 3.4 Age of kin death relative to the age of Focal

The impacts of bereavement may be determined partly by the age of the deceased kin relative to the age of Focal. Deaths of kin younger than Focal and deaths of kin older than Focal may have different implications. We can calculate deaths by any desired age differential. For example, define a vector  $\mathbf{c}^{\text{older}}(x)$  to indicate ages older than the age  $x$  of Focal,

$$c_i^{\text{older}}(x) = \begin{cases} 0 & i \leq x \\ 1 & i > x \end{cases} \quad (25)$$

and a corresponding vector for ages younger than that of Focal. Then

$$\mathbf{k}_D^{\text{older}}(x) = \mathbf{c}^{\text{older}}(x)^\top \hat{\mathbf{k}}_D \quad (26)$$

and similarly for  $\mathbf{k}_D^{\text{younger}}(x)$ . Modifications of  $\mathbf{c}$  can extract other age ranges of kin deaths relative to the age of Focal.

### 3.5 Aggregating multiple types of kin

The kinship model disaggregates kin into the categories shown in Figure 1 (and more if the network is extended to further generations of nieces, cousins, etc.). It may be useful to aggregate types of kin to better summarize the experience of Focal. This can be done by simply adding the kin vectors together. For example, the sum  $\mathbf{m}(x) + \mathbf{n}(x)$  combines older and younger sisters of Focal. One might want to combine first degree kin (mother, daughter, sisters), second degree kin (grandmother, grandchildren, aunts, nieces), and so on (e.g., Song and Caswell 2022). Or one might want to weight different types of kin by their coefficient of genetic relatedness.

As a general solution, suppose that the model includes  $N$  types of kin ( $N = 14$  in Figure 1), and let  $\hat{\mathbf{k}}_D^{(i)}(x)$  denote the vector for kin of type  $i$ . Define an array  $\mathbf{K}(x)$ ,

$$\mathbf{K}(x) = \left( \hat{\mathbf{k}}_D^{(1)}(x) \quad \hat{\mathbf{k}}_D^{(2)}(x) \quad \dots \quad \hat{\mathbf{k}}_D^{(N)}(x) \right). \quad (27)$$

Define a vector  $\mathbf{c}$  (dimension  $N \times 1$ ) whose entries are the weights to be attached to each type of kin. Then

$$\hat{\mathbf{k}}_D^{\text{aggregated}}(x) = (\mathbf{c}^\top \otimes \mathbf{I}_{\alpha\omega}) \mathbf{K}(x). \quad (28)$$

The vector  $\hat{\mathbf{k}}_D^{\text{aggregated}}(x)$  contains deaths by both cause and age at death; it can be reduced to cause- or age-specific deaths following Section 3.1.



### 3.6 The effects of changes in the risks of death

The mortality matrices  $\mathbf{M}$  and  $\widehat{\mathbf{M}}$  contain probabilities of death due to each cause, in the presence of all causes operating. It is interesting to modify the risks of death: perhaps changing one or more risks, or deleting one or more risks completely. One might, for example, want to explore the relative effects of eliminating or reducing certain risks, of expanding risks (e.g., more fatal overdoses), or adding new risks (e.g., COVID-19) that would compete against existing risks in causing deaths.

There is an almost endless number of changes in risk that might be of interest. We do not explore these results here, but we present the necessary mathematics in Appendix A.

## 4. An illustrative example

As an illustrative example of the range of insights the model can produce, we present a variety of cause-of-death calculations using data (described below) for Non-Hispanic White (NHW) women in the United States in 2003. This is a low mortality and low fertility population.

These results are presented only as a methodological example. A companion paper applies the method, with more substantive intent, to cause of death as a source of racial disadvantage (Verdery et al. 2023).

### 4.1 Cause of death data

The kinship estimation method requires information on both mortality patterns and fertility patterns. Because we are interested in studying bereavement from leading causes of death, we use mortality data from the CDC Wonder Underlying Cause of Death ICD-10 coded files (<https://wonder.cdc.gov/ucd-icd10.html>), which offer race/ethnicity-specific counts of deaths by single year of age and for each year, for women, over the 2003–2019 period. We focus our attention on the top six causes of death plus a residual category containing all other causes. The list of causes, in decreasing order of importance, is shown in Table 3.

We use data on births from the CDC Natality files (<https://wonder.cdc.gov/natality.html>). Unfortunately, the coding of race/ethnicity in these files differs slightly both between the two sources and within sources over the period, so in order to create consistently classified groups, we code race and ethnicity into five mutually exclusive categories (Hispanic of any race, Non-Hispanic White, Non-Hispanic Black, Non-Hispanic Asian or Pacific Islander, and Non-Hispanic American Indian or Alaska Native). To turn these

data into rates, we use denominators from the US Census Bureau’s intercensal population estimates,<sup>7</sup> which we also code as above.

**Table 3: The six leading causes of death, in decreasing order, and the residual category for all other causes, for US Non-Hispanic White females 2003–2019**

1	diseases of the heart (crd)	5	Alzheimer’s (alz)
2	malignant neoplasms (cnc)	6	accidents (acc)
3	cerebrovascular disease (cer)	7	residual (res)
4	chronic lower respiratory (rsp)		

Abbreviations shown are used in figures to follow.

Whereas the CDC data do not include information on people of more than one race, the Census Bureau data do contain such estimates. We focus only on respondents with single race identifiers and drop those with multi-race identities in the Census data; multi-race individuals tend to be younger and at lower risk of death, so this is unlikely to be a substantial source of bias. We conduct some other minimal data processing: excluding deaths where race or age were not identifiable (1,239 of 39,017,109 female deaths over the period) and aligning age categories of young births that differ over the period.<sup>8</sup> We created probabilities of dying in the interval conditional on commencing the interval ( ${}_nq_x$ ) using Chiang’s formula for low-mortality environments like the United States.<sup>9</sup> These steps provide us with the mortality and fertility patterns we need to produce bereavement estimates by specific causes over the 2003–2019 period.

## 4.2 Cause-of-death results

The cause-of-death model produces a very large set of results, many of which we have introduced in Section 3. In this section we will present a subset of these, focusing on the children, siblings, parents, and aunts-uncles of Focal. These four kin represent three generations: the first level of descendants of Focal, the siblings in the same generation as Focal, and the parents and aunts-uncles from the previous generation. For the curious reader, we present a gallery of all results, for all types of kin, in Supplemental Material.

<sup>7</sup> <https://www2.census.gov/programs-surveys/popest/datasets/2000-2009/national/asrh/> and <https://www2.census.gov/programs-surveys/popest/datasets/2010-2019/national/asrh/>.

<sup>8</sup> The youngest recorded age of birth in some years is “under 15” whereas specific ages 12, 13, and 14 are listed in other years. We align these by treating all 34,627 births over the period in these categories as being to 14 year olds. By comparison, there are 68,445,696 total births over the period.

<sup>9</sup> [https://papp.iussp.org/sessions/papp101\\_s07/PAPP101\\_s07\\_090\\_010.html](https://papp.iussp.org/sessions/papp101_s07/PAPP101_s07_090_010.html) and [https://papp.iussp.org/sessions/papp101\\_s07/PAPP101\\_s07\\_050\\_050.html](https://papp.iussp.org/sessions/papp101_s07/PAPP101_s07_050_050.html).

#### **4.2.1 Numbers of deaths**

Figure 2 shows the numbers of deaths of children, siblings, parents, and aunts-uncles due to each cause, as a function of the age of Focal. Both deaths experienced at each age and accumulated up to each age are included.

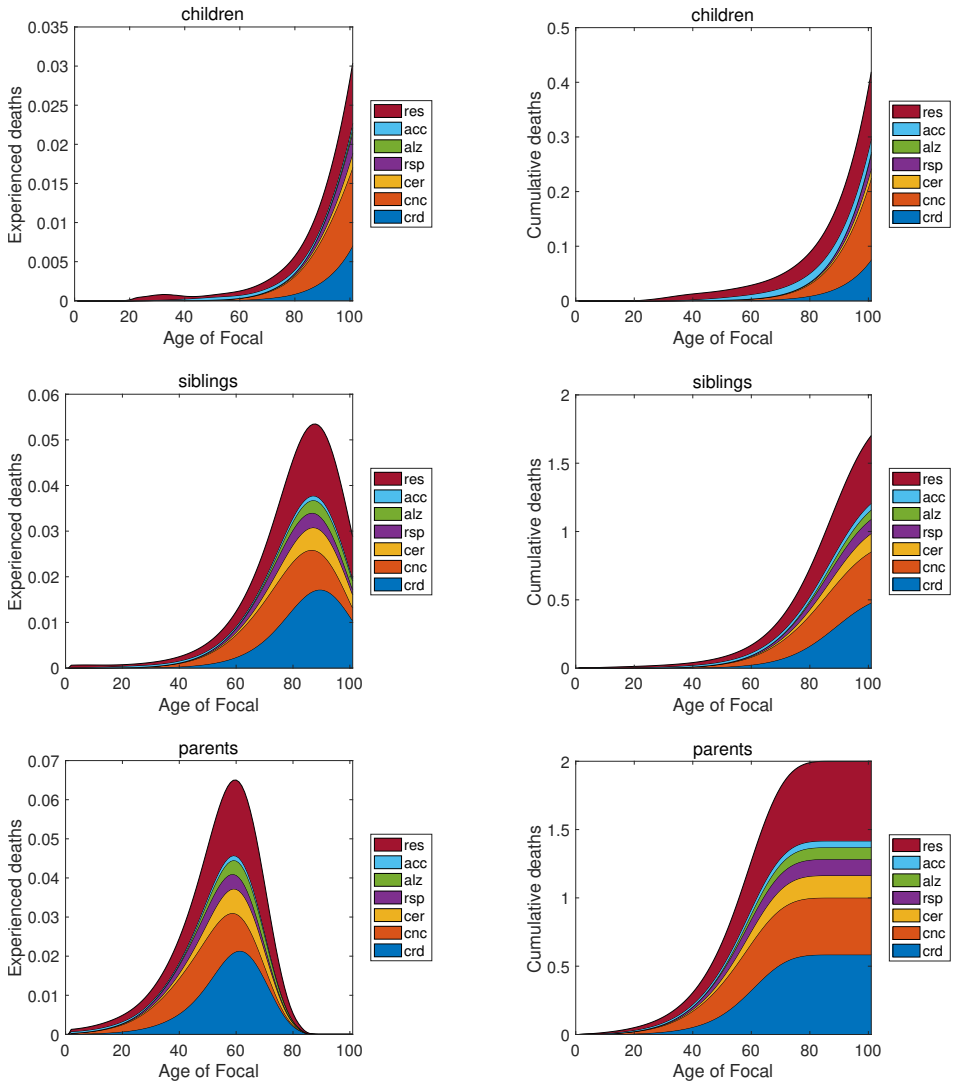
Focal experiences the deaths of parents and of aunts and uncles on approximately the same schedule, at a modal age of about 60 years. Sibling deaths are experienced much later, at a modal age of about 85 years, and deaths of children are rare and experienced very late if at all. The cumulative figures bear this out, and also show the different numbers of deaths experienced by Focal as she ages. Only about 0.4 child deaths are accumulated by Focal at age 100; about 1.5 siblings, 2 parents, and about 3.5 aunts and uncles. This obviously reflects the low-fertility and low-mortality nature of this population.

The most important causes, of both the experienced deaths at any age and the cumulative deaths over the lifetime, are, not surprisingly, the two leading causes of death: heart disease and cancer. Bereavement from cancer (cause 2) increases earlier in the life course than does bereavement from heart disease (cause 1). These two causes are responsible for the majority of the kin deaths experienced by Focal at the end of her life.

#### **4.2.2 Proportions of deaths**

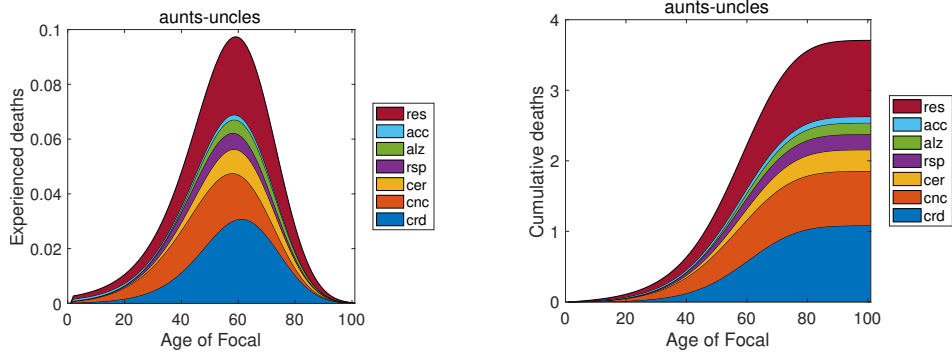
Figure 3 gives more insight into the patterns of cause of death by showing the proportions of deaths due to each cause, for children, siblings, parents, and aunts-uncles. As in Figure 2, heart disease and cancer account for the largest proportions of deaths. Deaths due to accidents (cause 6) are also a major contributor to deaths experienced by Focal up to age 30 or so. This is most evident first in the deaths of children experienced by Focal, and then in the deaths of siblings. Accidents make a smaller contribution to the deaths of Focal's parents and aunts-uncles.

**Figure 2:** (Part 1) The numbers of deaths of children, siblings, and parents, due to each of the top six causes of death. Deaths experienced by Focal at each age (left) and cumulative deaths up to each age (right)



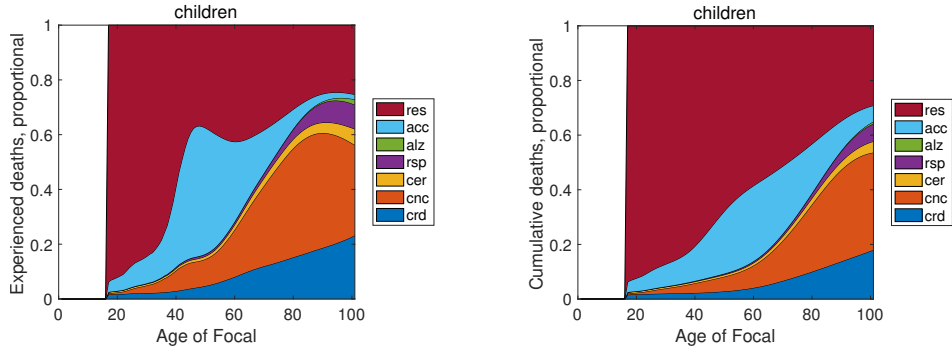
Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes. Note the different y-axis scales on each plot.

**Figure 2: (Part 2) The numbers of deaths of aunts-uncles due to each of the top six causes of death. Deaths experienced by Focal at each age (left) and cumulative deaths up to each age (right)**



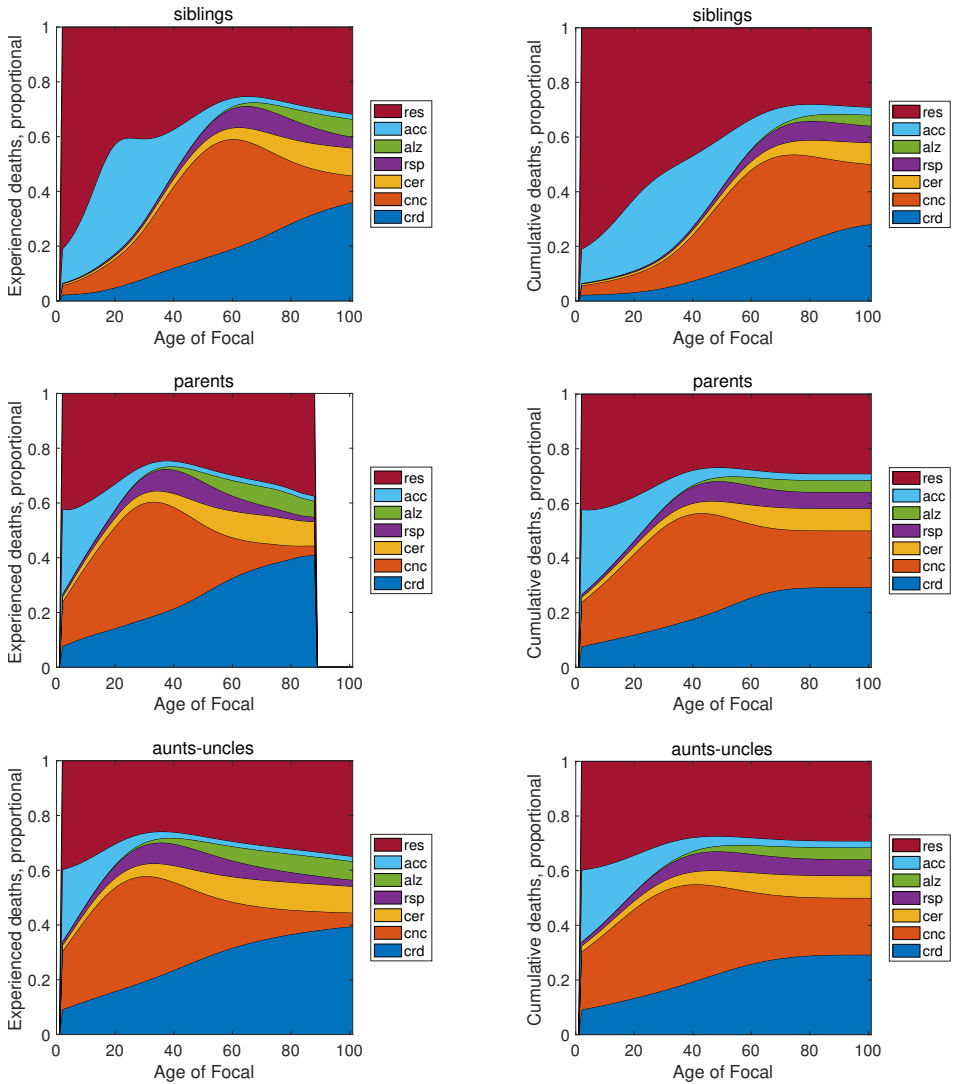
Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes. Note the different *y*-axis scales on each plot.

**Figure 3: (Part 1) The proportions of experienced (left) and cumulative (right) deaths of children of Focal, due to each of top six causes of death**



Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes.

**Figure 3:** (Part2) The proportions of experienced (left) and cumulative (right) deaths of siblings, parents, and aunts-uncles of Focal due to each of top six causes of death



Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes.

### 4.2.3 Deaths of kin by degree of relatedness

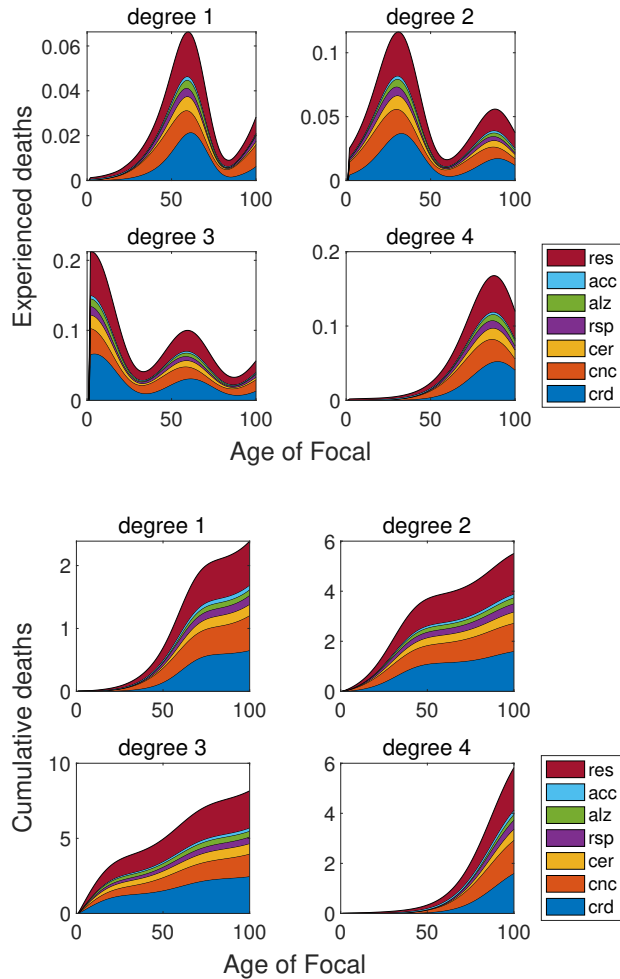
It may be of interest to examine kin deaths and their causes for various degrees of relationship to Focal. Table 4 shows the kin of degrees 1 through 4, as defined as in civil law (for matters of inheritance, marriage, etc.). Figure 4 shows the numbers of deaths, experienced and cumulative, of kin grouped into these degrees of relationship. Other groupings are, of course, possible (for an example including kinship ties and dwelling proximity, see Verdery et al. 2012).

**Table 4: Relatives of degrees 1 through 4**

Degree	Kin
1	parent, child
2	grandparent, grandchild, sibling
3	great-grandparent, great-grandchild, aunt-uncle, niece-nephew
4	first cousins

The number of deaths of kin are larger for more distantly related kin (third and fourth degree) than for more closely related kin (first and second degree). This reflects both the relative numbers of kin at risk of death and their ages relative to Focal. Thus Focal experiences a peak in death of degree-1 kin at about age 50 (parents) and at very late ages (children). The peaks in degree-2 kin are shifted earlier, and those of degree-3 kin even earlier. By the end of her life, Focal will have experienced a bit over two deaths of degree-1 kin, about five of degree-2 kin, about seven of degree-3 kin, and six degree-4 kin.

**Figure 4:** Experienced (above) and cumulative (below) deaths of kin of degrees 1–4 due to each of the six leading causes of death



Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes. Note the different *y*-axis scales on each plot.

#### 4.2.4 Prevalence of the experience of bereavement

The prevalence of bereavement is the probability that Focal will experience at least one death, of a particular kin, from a particular cause. Figure 5 shows this probability that



Focal experiences at least one death of a child, a parent, a sibling, and an aunt-uncle. The probabilities are calculated using the Poisson and the binomial approximations given in Section 3.3. Probabilities are shown for deaths due to the five most important causes of death and for all causes combined.

The results for all causes combined show that the prevalence of bereavement is high, reaching about 50% (in cumulative terms) at about age 45 for parents, age 80 for siblings, and age 40 for aunts-uncles. Because of the low fertility and high survival in this population, the prevalence of the loss of children only reaches about 30% by age 100. The prevalence of bereavement due to specific causes of death shows no clear patterns.

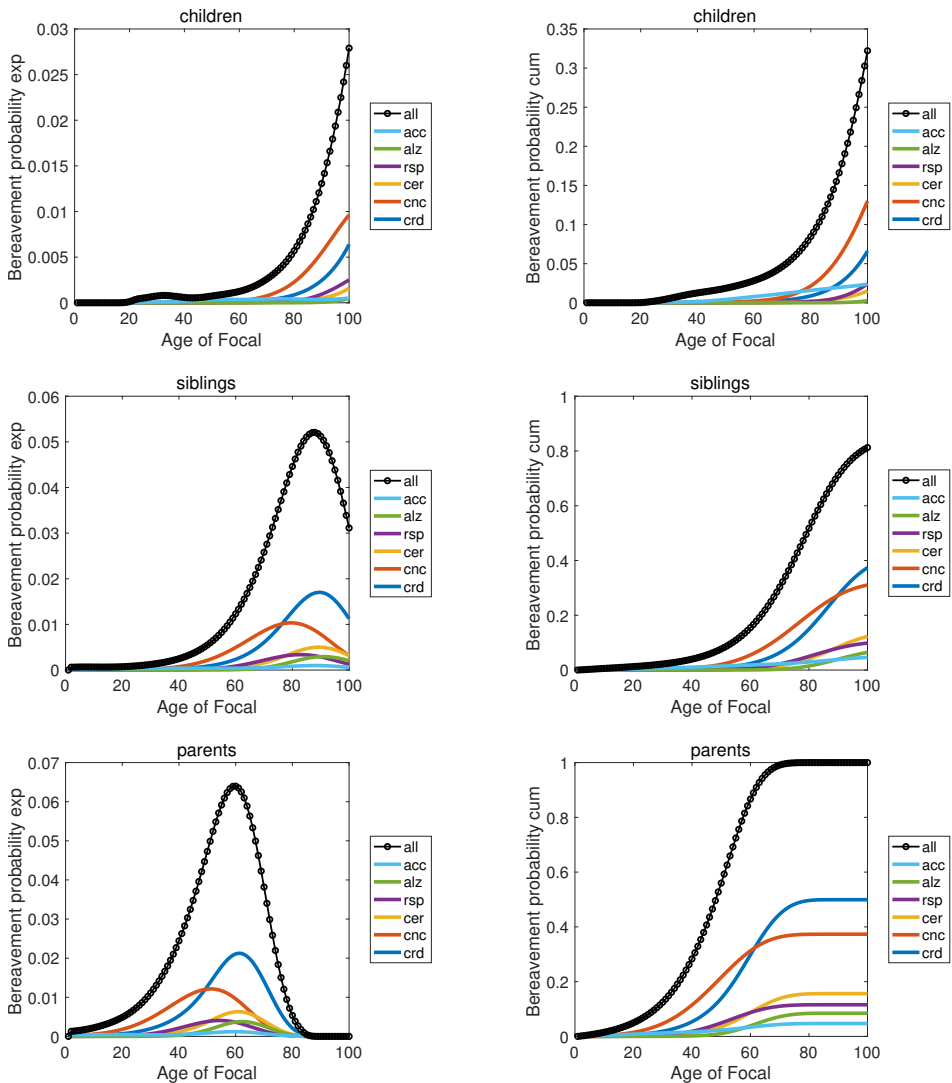
If the deaths of all types of kin are aggregated, as in Section 3.5, the resulting prevalences of bereavement are shown in Figure 6. Almost regardless of age, about 20% of individuals will experience the death of a relative each year. When accumulated over age, we see that Focal is almost certain to have experienced the death of at least one kin, of some type, by age 20.

#### **4.2.5 Deaths of younger and older kin**

Figure 7 shows the numbers of deaths of younger and older children, parents, siblings, and aunts-uncles experienced by Focal. Some of these deaths of kin are trivially zero; e.g., there can be no deaths of children older than, or parents younger than, Focal.

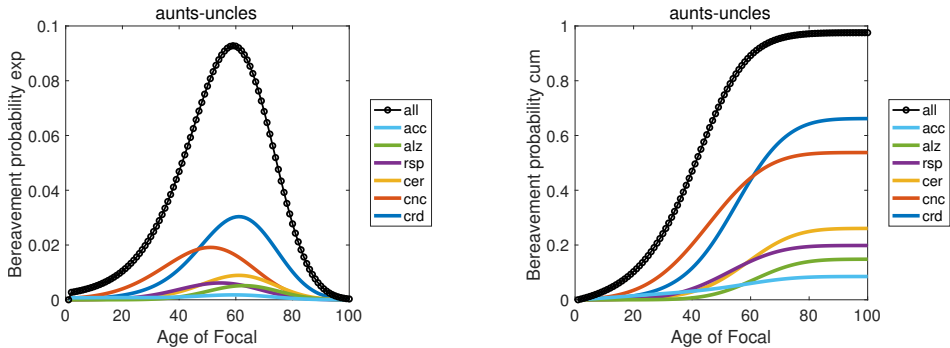
The results show the expected shift in the ages at which Focal experiences such deaths. Focal experiences the death of older siblings at a modal age of about 80, and the death of younger siblings at a modal age of about 90. A similar pattern is apparent in the deaths of older and younger aunts-uncles. Note that aunts-uncles younger than Focal are rare, and hence so are deaths of such kin. But, even with a maximum of only  $6 \times 10^{-4}$ , the age pattern is similar to other kin.

**Figure 5:** (Part 1) The prevalence of bereavement (i.e., the probability of the loss of at least one kin) due to the death of children, siblings, and parents. Experienced deaths (left) and cumulative deaths (right) due to six leading causes and all causes combined



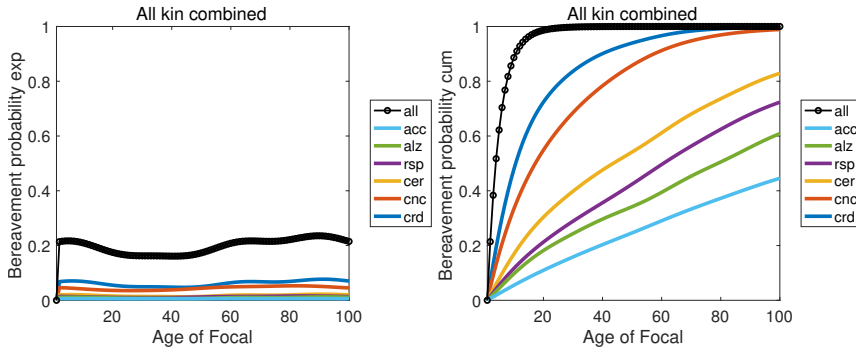
Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes.

**Figure 5: (Part 2) The prevalence of bereavement (i.e., the probability of the loss of at least one kin) due to the death of aunts-uncles. Experienced deaths (left) and cumulative deaths (right) due to six leading causes and all causes combined**



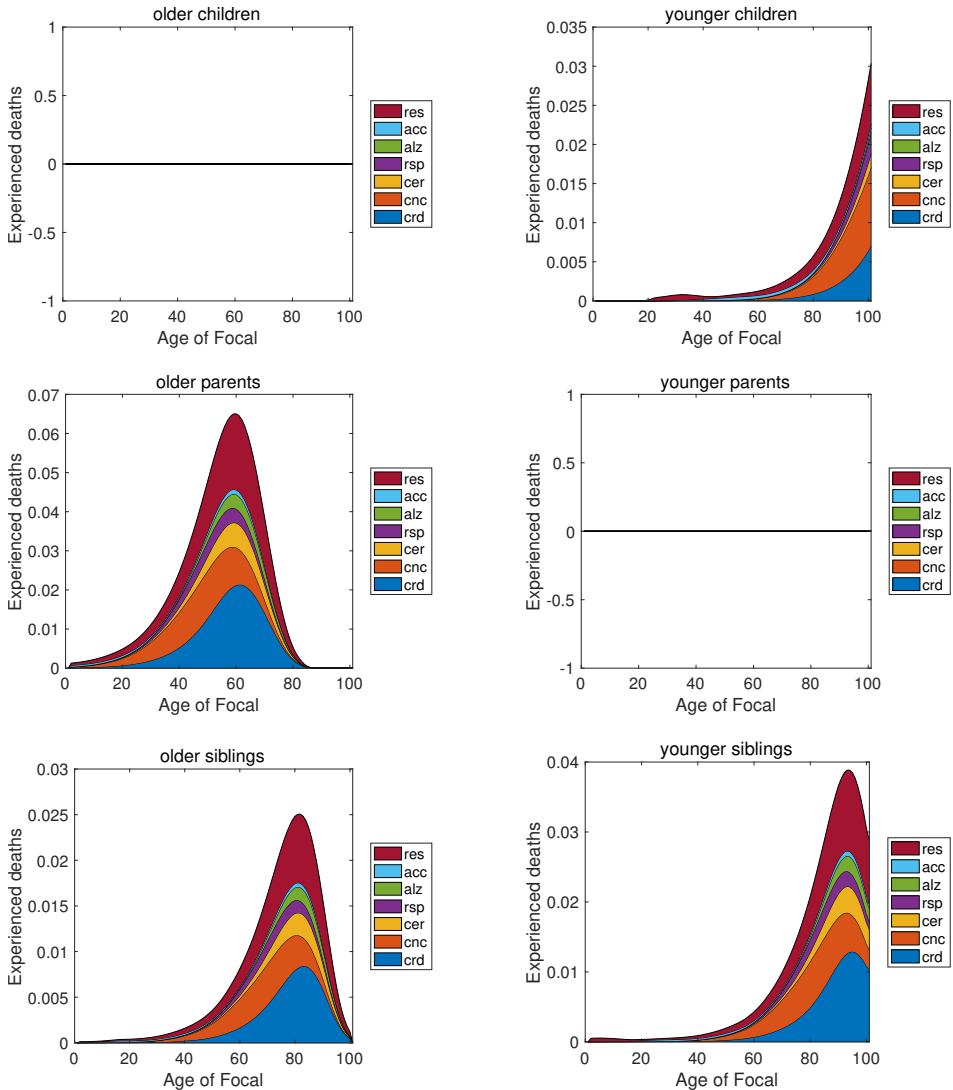
Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes.

**Figure 6: Experienced (left) and cumulative (right) probabilities of bereavement (i.e., experiencing the death of at least one relative), for all kin combined**



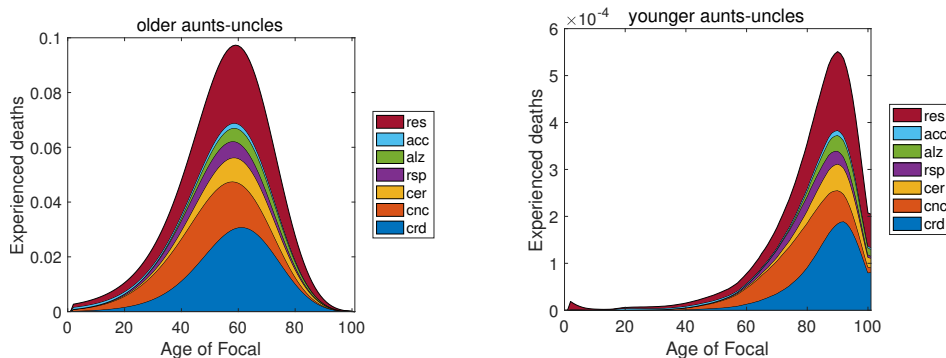
Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes.

**Figure 7: (Part 1) Numbers of deaths of children, parents, and siblings at ages older than Focal (left) and younger than Focal (right) at the time of death**



Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes. Note different *y*-axis scales on each plot.

**Figure 7: (Part 2) Numbers of deaths of aunts-uncles at ages older than Focal (left) and younger than Focal (right) at the time of death**



Causes: crd=cardiovascular, cnc=cancer, cer=cerebrovascular, rsp=lower respiratory, alz= Alzheimer's, acc=accidents, and res=residual causes. Note different  $y$ -axis scales on each plot.

## 5. Discussion

The key to a full analysis of the loss of kin experienced by a Focal individual is the incorporation of probabilities of death, by cause and age, into the projection matrix, and then linking the cause-specific probabilities to overall mortality using the theory of competing risks. Multistate and multiple decrement life tables include some of the processes at work here, but not in the linked fashion required for kinship analysis, where the survival and reproduction of one type of kin are responsible for producing individuals of another type. It is risky to call anything impossible; doing so only encourages smart people to prove the claim wrong. However, it seems well nigh impossible to carry out the analyses presented here using life table techniques.

The complete output produced by the theory is impressive in its richness. The calculations compute expected deaths, of every type of kin, at every age of death, from every cause, at every age of Focal. They yield the experience of kin death at any specific age  $x$  of Focal as well as the cumulative experience of kin death up to any age  $x$ . This permits analysis of the bereavement burden due to different causes and patterns of kin deaths. Although we have not explored it here, the results in Section 2.3.2 also make it possible to assign weights that treat recent deaths and past deaths differently, effectively making it possible to account for Focal 'forgetting' the past. Because the model uses the matrix framework developed in Caswell (2019), it is easy to incorporate prevalences of conditions, such as health conditions (Caswell 2019; Feng, Song, and Caswell 2023) or

employment status (Song and Caswell 2022) into the living or dead components of the kinship network.

In this paper we have used the time-invariant one-sex (female) age-classified kinship model, relying on the androgynous approximation to calculate numbers of both male and female kin. But the matrix kinship theory exists in time-varying, multistate, and two-sex models, and those extensions (Caswell 2019, 2020, 2022; Caswell and Song 2021) could easily be used as the basis for a cause-of-death analysis. In particular, the use of a full two-sex model would permit analysis of causes of death that are sex-specific (e.g., complications of childbirth, prostate cancer) or nearly so (e.g., breast cancer); see Verdery et al. (2023) for a first step. A time-varying version would reveal changes in the loss of kin as the importance of different causes of death change historically (see Caswell and Song (2021) for the time-varying model). However, this would require long time series of mortality rates by cause of death, which seem unlikely to be available. Lacking that information, the calculations here should be treated in the same way that period life table calculations are treated: as the demographic outcomes implied by a set of mortality and fertility schedules when everyone experiences those schedules throughout their life.

From a formal demographic point of view, the matrix denoted here by  $\tilde{U}$ ,

$$\tilde{U} = \left( \begin{array}{c|c} \mathbf{U} & \mathbf{0} \\ \hline \widehat{\mathbf{M}} & \mathbf{I}/\mathbf{0} \end{array} \right), \quad (29)$$

provides the framework for these extensions. The upper-left block  $\mathbf{U}$  contains transitions among transient states; modifications of the transient state space (incorporating ages, or stages, or multistate combinations) would appear here. The lower-left block  $\widehat{\mathbf{M}}$  contains transitions from transient to absorbing states; those states can be as simple as a single stage representing death, or as complex as death at different ages or stages from different causes. The lower-right block describes how the individuals in absorbing states are kept in the population vector: either discarded at once, accumulated indefinitely, or gradually forgotten. The upper-right block,  $\mathbf{0}$ , is largely immune to modification; it represents the recovery of the dead back into the living states, which is usually assumed not to happen.

The results we have shown only scratch the surface of the kinds of results that can be obtained. They show clearly the different patterns of exposure to kin loss for various types of kin as Focal ages. Certain aspects are obviously expected; deaths of children, for example, are experienced at old ages of Focal, whereas deaths of parents and grandparents are experienced at younger ages. But the results here quantify that pattern as a function of mortality and fertility schedules, and they pinpoint the types of causes that lead to such deaths.

The results reveal dramatic differences in the causes of the deaths experienced by Focal. The relative impact of the two leading causes (heart disease and cancer) change places as Focal ages. The role of deaths due to accidents differs dramatically among

types of kin; experienced deaths of children, grandchildren, and great-grandchildren are frequently due to accidents, which play only a minor role in the deaths of other types of kin.

The interacting roles of mortality, fertility, and the age schedules of cause-specific hazards lead to very different total numbers of kin deaths. In Figure 4, we see that at old ages Focal will have been exposed to the deaths of about 2 first-degree kin, but about 5 second-degree kin and about 7 third-degree kin.

There is an important distinction between treating the results of the kinship model as estimates of a kinship network, and treating them as what are more properly called projections of the network. Exactly as in the cohort-component population projections familiar to demographers, the model computes the results implied by a scenario of a set of demographic rates (by age, stage, sex, time, etc.). Such projections provide a baseline against which to compare observations of real kinship networks, but as emphasized by Goodman, Keyfitz, and Pullum (1974), there is no reason to treat the projections as estimates of those observations. Keyfitz has written insightful discussions of the relationships between projections, forecasts, and estimates (Keyfitz 1972; Keyfitz and Caswell 2005: chap. 20).

The results we show give the kinship network of a Focal individual at any specified age. The kinship structure of a population, rather than an individual, is easily calculated by weighting the age-specific kinship structure by the age distribution of the population. The calculation is familiar in the creation of crude or standardized mortality rates as weighted averages of age-specific rates over a population age distribution (Preston, Heuveline, and Guillot 2000: chap. 2). For an example in the kinship context, see Feng, Song, and Caswell (2023).

Every theoretical development invites further research. In this case, knowing the age structure of deaths of kin of Focal suggests the question of the value, or cost, of those deaths, and the differential impact of different types of deaths. The ages at which the deaths occur (e.g., Figure 7), or how closely related the kin are (e.g., Figure 4), might make the deaths more or less significant to Focal, as might spatial location, coresidence, or degree of economic and family support.

Our analysis also suggests applications to morbidity as well as mortality. The defining criterion of the causes of death is that they are absorbing states in the transition matrix. The model could be extended to states that are effectively permanent but not fatal (e.g., cancer survivors, those ever incarcerated, ever a migrant, etc.).

Finally, it is worth noting that the effects of kin loss are not limited to humans. Relationships with kin are a source of ‘social buffering’ in some animal species, reducing physiological stress responses (Parker et al. 2022). The loss of kin can lead to changes in behavior and reductions in survival and fertility throughout the lifetime (e.g., Berger et al. 2021; Zippel et al. 2021; Parker et al. 2022). Many such analyses focus on the effects of the death of mothers on their offspring, but in some social species losses of other types of

kin can also have effects (e.g., see Berger et al. (2021) for effects of the loss of siblings in elephants). Even in fruit flies (*Drosophila*), the mere exposure to dead conspecifics triggers complex neural pathways that lead to dramatic decreases in lifespan (Gendron et al. 2023).

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## Appendix

### A. The effects of changes in the risks of death

The mortality matrices  $\mathbf{M}$  and  $\widehat{\mathbf{M}}$  contain probabilities of death due to each cause, in the presence of all causes operating. It is interesting to modify the risks of death: perhaps changing one or more risks, or deleting one or more risks completely. One might, for example, want to explore the relative effects of eliminating or reducing certain risks, of expanding risks (e.g., more fatal overdoses), or the effects of additional new risks (e.g., COVID-19).

#### A.1 Hazards and probabilities

Because the risks due to different causes are competing, changes to the risks cannot be analyzed by modifying the probabilities directly. Changing one of a set of competing risks changes the probabilities of death due to all the causes.

Instead, mortality risks must be modified by changing the hazards (continuous-time rates) of mortality due the various causes, and then calculating the probabilities of death from the modified hazards. So far we have worked with the probabilities directly; we need to calculate the underlying hazards.

Let  $h_i$  be the hazard due to cause  $i$  and  $\tilde{h}$  the sum of all the hazards. Then if the hazards are treated as constant over the interval  $[t, t + 1)$ , the probability of death due to cause  $i$  in that interval, in the presence of all competing causes of death, is

$$P(\text{death due to cause } i) = \frac{h_i}{\tilde{h}} \left(1 - e^{-\tilde{h}}\right). \quad (30)$$

Define a hazard matrix

$$\mathbf{H} = \begin{pmatrix} h_{11} & \cdots & h_{1,\omega} \\ \vdots & & \vdots \\ h_{\alpha 1} & \cdots & h_{\alpha,\omega} \end{pmatrix}, \quad (31)$$

from which we calculate a vector of summed hazards

$$\tilde{\mathbf{h}}^\top = \mathbf{1}_\alpha \mathbf{H}. \quad (32)$$

The age-specific probabilities of death, subject to all hazards, are the elements of

$$\mathbf{q} = \mathbf{1}_\alpha - e^{-\tilde{\mathbf{h}}} \quad (33)$$

(the exponential is applied element-wise), from which

$$\tilde{\mathbf{h}} = -\log(\mathbf{1}_\alpha - \mathbf{q}). \tag{34}$$

The probabilities of survival are

$$\mathbf{p} = \mathbf{1}_\alpha - \mathbf{q}. \tag{35}$$

The elements of  $\mathbf{p}$  appear as the subdiagonal entries of the survival matrix  $\mathbf{U}$ .

Applying the competing risk result of (30) to the elements of the hazard matrix  $\mathbf{H}$  gives the entries of the mortality matrix  $\mathbf{M}$ :

$$\mathbf{M}(i, j) = \frac{h_{i,j}}{\tilde{h}_j} \left(1 - e^{-\tilde{h}_j}\right). \tag{36}$$

This can be written in matrix form as

$$\mathbf{M} = \mathbf{H} \mathcal{D}(\tilde{\mathbf{h}})^{-1} \mathcal{D}(\mathbf{q}). \tag{37}$$

Solving for  $\mathbf{H}$  gives

$$\mathbf{H} = \mathbf{M} \mathcal{D}(\mathbf{q})^{-1} \mathcal{D}(\tilde{\mathbf{h}}). \tag{38}$$

The hazards in  $\mathbf{H}$  can be modified and used to calculate a new mortality matrix  $\mathbf{M}$  following (37). If desired, the more detailed output in  $\widehat{\mathbf{M}}$ , classified by age at death and cause of death, can then be calculated using (12).

## A.2 Proportional and additive changes to the hazards

Proportional changes to a set of hazards modify the intensity of the existing risks, without changing their relative magnitude. Additive changes can modify the relative pattern, as when a new cause of death is introduced. For example, the hazard of death from a new type of cancer, operating independently of other types, would be added to the hazard of death from cancers (of all types).

Proportional changes are implemented by multiplying the age- and cause-specific hazards. For example, define  $\mathbf{c}$  as a vector of factors by which to modify each cause, with the same modification applying across all ages. Then

$$\mathbf{H} \mapsto \mathcal{D}(\mathbf{c})\mathbf{H}. \tag{39}$$

Proportional changes can be made to specific combinations of ages and causes by defining a matrix  $\mathbf{C}$  of size  $\alpha \times \omega$ , where  $C(i, j)$  is the factor by which to modify the hazard of cause  $i$  at age class  $j$ . Then

$$\mathbf{H} \mapsto \mathbf{C} \circ \mathbf{H}. \quad (40)$$

where  $\circ$  denotes the Hadamard, or element-by-element product.

Additive changes can be implemented by defining a matrix  $\mathbf{C}$  of additive changes, and implementing

$$\mathbf{H} \mapsto \mathbf{H} + \mathbf{C}. \quad (41)$$

Care must be taken to assure that negative additive changes do not render any entry of  $\mathbf{H}$  negative.