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ABSTRACT

Conjugal Bereavement Effects on Health and Mortality at Advanced Ages*

We specify a model for the lifetimes of spouses and the dynamic evolution of health, allowing spousal death to have causal effects on the health and mortality of the survivor. We estimate the model using a longitudinal survey that traces many health status aspects over time, and that is linked to register data on the vital status of the individuals. The model takes account of selectivity in partners' mortality and health evolution. We find strong instantaneous effects of bereavement on mortality and on certain aspects of health. Individuals lose on average 12 % of residual life expectancy after bereavement. Bereavement affects the share of healthy years in residual lifetime, primarily because healthy years are replaced by years with chronic diseases.

JEL Classification: I12, I11, J14, J12, C41

Keywords: death, longevity, health care, disease, life expectancy, elderly couples, impairment

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1 Introduction

At high ages, dramatic events in an individual's life may lead to dramatic changes in his or her health conditions. In particular, the death of the spouse can be a major sources of psychosocial stress, depression and anxiety. These are all factors associated with morbidity and mortality.[1] Conjugal bereavement may also directly deteriorate physical health, for instance by impairing the immune system, increasing the prevalence of chronic illnesses, and functional limitations. Furthermore, most older widowers experience (serious) difficulties in performing housekeeping chores like cooking or cleaning. This may in the long run negatively affect their health - due, for instance, to insufficient caloric intake or nutritional deficits. Finally, there is evidence that widows often suffer from greater poverty, a factor associated with higher morbidity and mortality among the aged.[2] Osterweis, Solomon and Green (1984) provide a detailed overview of the possible causal effects of bereavement on health and mortality.

Conjugal bereavement affects the potential supply of informal care for the person left behind (the non-organized care provided from within the social network of the individual). Informal care is an essential supplement or substitute to self-care and professional long-term care. In the Netherlands for instance, about 30% of older (65 plus) individuals receive some kind of informal assistance (see e.g. Portrait, 2000). The larger part of the informal assistance is provided by healthier partners (see for instance Norton, 2000, and Lakdawalla and Philipson, 2002). This is reflected in health care costs. Prigerson, Maciejewski and Rosenheck (2000) estimated average health costs in 1989 and find that costs equal $ 2,384 for widowed and $ 1,498 for married subjects. So, if the death of the partner negatively affects the health and well-being of the surviving spouse, it also reduces the supply of informal care whereas in fact the demand for care has increased (see Williams, 2004, for additional evidence). This will exacerbate the health effects and therefore increase the demand for formal health care services use, and this increases health care costs.

In this paper we provide a detailed analysis of health and mortality risks at advanced ages. We model the dynamic evolution of health and we focus on the effect of conjugal bereavement on health and mortality risks. This enables us to improve on the existing literature in two ways. First, note that the lifetimes of spouses may be related because of a causal effect of the death of one member on the mortality rate of the surviving spouse, or because of (unobserved) fac-

[1] See for instance Prigerson, Maciejewski and Rosenheck (2000).

[2] See for instance Ecob and Smith (1999) and Benzeval and Judge (2001).

tors that influence both lifetimes. With respect to the latter, spouses may have similar health-related behavior, eating patterns, material circumstances, marital satisfaction, or more generally, spouses have shared life histories that may affect husband-wife mortality and morbidity. Klein (1992) finds that death times are related significantly between husbands and wives through unobserved couple-level frailty. This means that there may be a stochastic relationship between the two lifetimes, even after we have controlled for observed characteristics. It also means that we can not simply assume that the vital status of one spouse is an exogenous determinant of the mortality rate of the other. Instead we model the lifetimes of both spouses and the multiple ways in which they are related. This distinguishes our model from the usual contributions on conjugal bereavement effects. One major exception is Lichtenstein et al. (1998) in which the dependence of survival status on marital status is studied using twin data. They assume that there is an identical unobserved component for both members of the twin couple and use stratified partial likelihood methods to assess the causal effect of spousal bereavement on subsequent mortality.

Furthermore, the large majority of the studies in this area analyze the effect of bereavement on one specific aspect of health status, like stress or mortality.[3] This includes Lichtenstein et al. (1998) who restrict attention to mortality. A second main contribution of our study is that we distinguish between different aspects of health. There are large differences in health status among the aged. Some of them are healthy, whereas others suffer from cognitive impairment, but are in perfect physical condition, and yet others combine cognitive impairment with severe physical limitations. We consider all of these and study their simultaneous dynamic evolution.

Our analyzes are based on a dataset covering about 2,000 older couples for the period 1992-2000 called the Longitudinal Aging Study Amsterdam. The dataset has abundant information on health indicators and outcomes as well as on socio-demographic variables. Of interest for our analyses is that about 24% of the main

[3]See, for instance, Prigerson, Maciejewski and Rosenheck (2000) and Dijkstra, Van Tilburg and De Jong Gierveld (2005) on the effect on psychosocial stress, depression, anxiety and loneliness, Irwin et al. (1987, 1993) on impaired immune function, Prigerson et al. (1997, 2000) on chronic illnesses and functional limitations, Koehn (2001) on caloric intake and nutritional deficits, and McGarry and Schoeni (2005) on poverty. Lillard and Panis (1996) study mortality outcomes and incorporate a one-dimensional self-reported general-health assessment into the analysis. This health variable is observed at regular time intervals. They also control for related unobserved determinants in marital status, health, and mortality. However, the empirical analysis focuses on the effect of marital status (formation and dissolution) in general on mortality, and most observed dissolutions are not bereavements but divorces.

respondents and about 17.3 % of their partners are observed to die during the period of observation, and that we were able to obtain administrative data on the vital status of the respondents and of their spouse for the entire period of observation.

The information in the data allows us to carefully characterize health evolution, to examine how health status influences mortality, to model the interrelation between the lifetimes of spouses, and to study how the death of a partner influences the health and mortality of the surviving spouse. As will be explained, feasibility constraints demand the use of a data reduction method to summarize the observed health information in an equally informative health set of lower dimension. We use a flexible non-parametric method called the Grade of Membership Method (GoM) (Woodbury and Clive, 1974; Woodbury and Manton, 1982). This method is designed to summarize a large set of health conditions into a smaller number of clinical disease types. It is particularly useful if the observable health-condition outcome variables are binary or only have a small set of possible values. The method also determines individual weights measuring the degree to which an individual fits each of the clinical disease types. We use these Grade of Membership individual weights as measures for the health status of the respondents and include these as regressors in our model for mortality. These health measures are likely to be endogenous and we therefore extend our bivariate survival model with a model for health dynamics. Health is allowed to depend on lagged health, individual characteristics and the death of the partner.

We use the estimated model to calculate expected residual lifetimes in different health states ("health expectancies") for married and widowed males and females of various age groups. Health expectancies are informative on the fraction of life spent in a healthy state compared to the fraction spent in frail health conditions. This information plays a central role in the policy debate about the future needs for long-term care of older populations. For a recent review on health expectancies, see Robine, Jagger, and Mathers (2003).

Section 2 reports on the dataset. Section 3 discusses the non-parametric Grade of Membership method. We apply the method on our data and briefly discuss the main results. Section 4 proceeds with the statistical model for health and mortality and Section 5 presents the results. In Section 6 we present the results of simulations with the model. In particular, we compare expected residual lifetimes and health expectancies of married and bereaved individuals. Section 7 concludes.

2 Data

We use data from the Longitudinal Aging Study Amsterdam (LASA) (Deeg, Knipscheer and Van Tilburg, 1993). The LASA study follows over time a representative sample of non-institutionalized and institutionalized individuals aged 55 and above. The respondents lived at baseline (1992) in 11 municipalities in the West, North-East, and South of the Netherlands. We use three waves: the 1992-93 wave, the 1995-96 wave, and the 1998-99 wave.[4] Apart from the usual socio-demographic variables, this dataset provides extensive information on the physical, emotional, mental, and social functioning and also on a large set of variables that may have an effect on these four aspects of functioning. Each component is assessed by questionnaires and tests. Individuals were submitted either to a complete face-to-face interview or, if they refused this, to a short telephone interview.

The LASA dataset is linked to administrative records on the *vital and marital status* of the main respondents and their spouses up to the first of January 2000. If the respondent died during the sampling period, the date and place of death as well as the timing of spousal bereavement (if he or she was widowed at the moment of death) are recorded. For respondents who are still alive at the first of January 2000, changes in marital status – and the moment these changes took place – are also registered. This allows us to accurately follow the changes in marital and vital status from the start of the sampling period up to the first of January 2000. The smallest interval of time between deaths of spouses is 20 days and one quarter of the respondents observed to die after conjugal bereavement die within one year.

There are however some data limitations. First, information on the spouse is not available after the death of the LASA respondent. Second, for privacy reasons, the municipality of Amsterdam refused to provide records on spouses. Consequently, the information on the vital status of spouses of respondents who lived in Amsterdam can only be obtained from the survey data. We return to factual numbers concerning this below.

2,061 individuals share their household with a partner at the initial wave in 1992. Respondents who lost their partner before this initial wave are excluded. For this group we do not have information on the partner. We also excluded a small group (121) of people living together but who were not married. For

[4]The LASA respondents are drawn from a dataset on social network of older individuals held in the early months of 1992. This dataset, called the LSN dataset (Van Tilburg et al. 1992), provides among other things the household history of the respondents.

this group we lacked essential information on the vital status of the partner.[5] Respondents who were still legally married but who were not living together (13 individuals) were also excluded from our dataset. 6 respondents are observed to divorce during the observation period; these respondents are discarded from our analyses as well. The resulting sample counts 1.921 individuals. Very few respondents (about 0.3% of our sample) are observed to re-marry after a period of widowhood. We decided to only use the information of these individuals up to the time of their new marriage.

Figure 1 shows the evolution of marital status and mortality over time. About 24 % of the main respondents died during the sample period. This concerns 345 males (dying at an average age of 79) and 111 females (dying at an average age of 78). 269 respondents were still married at the time of their death. 37 males and 25 males were observed to die after a period of widowhood (on average 2.2 and 2.9 years for respectively males and females). The remaining 125 (345 + 111 - 269 - 37 - 25) respondents who are observed to die lived in the city of Amsterdam. For these 125 respondents we can only assess their marital status from survey information. More precisely, information on the vital status of the spouse can only be obtained for these respondents if they lived longer than their spouse and also participated in a wave after the death of their partner. We furthermore have 256 respondents from Amsterdam, who were still alive at the first of January of 2000. For these individuals, we miss information on the vital status of the spouse for the period between their last interview and the first of January 2000. These partial observability problems need to be accounted for in the construction of the likelihood function. We return to this in Section 4.

In total we observe 331 cases where the partner of the main respondent dies (116 widowers and 215 widows). The average ages of widowhood are equal to 78.4 for males and 73.4 for females. Figure 2 shows the survival curve based on the Kaplan-Meier estimate of the mortality hazard. First, it should be noted that the survival probabilities for married individuals who are older then 86 are based on a small number of observations. The same is true for age class 49-55. Second, the graph shows that the survival probabilities are lower for widowed individuals, except at older ages. For younger ages there is a relatively large difference between the curves for married individuals and widowed individuals. This is consistent with the results in the literature on mortality and spousal bereavement (see, for instance, Lichtenstein et al. 1998).

[5]The cause of a separation between unmarried partners (death, discord, hospitalization, admission in nursing or residential homes etc.) is not recorded in the LASA study. Moreover, municipalities only provide records of official partners – i.e. spouses.

Figure 1: Evolution of marital status during the LASA study

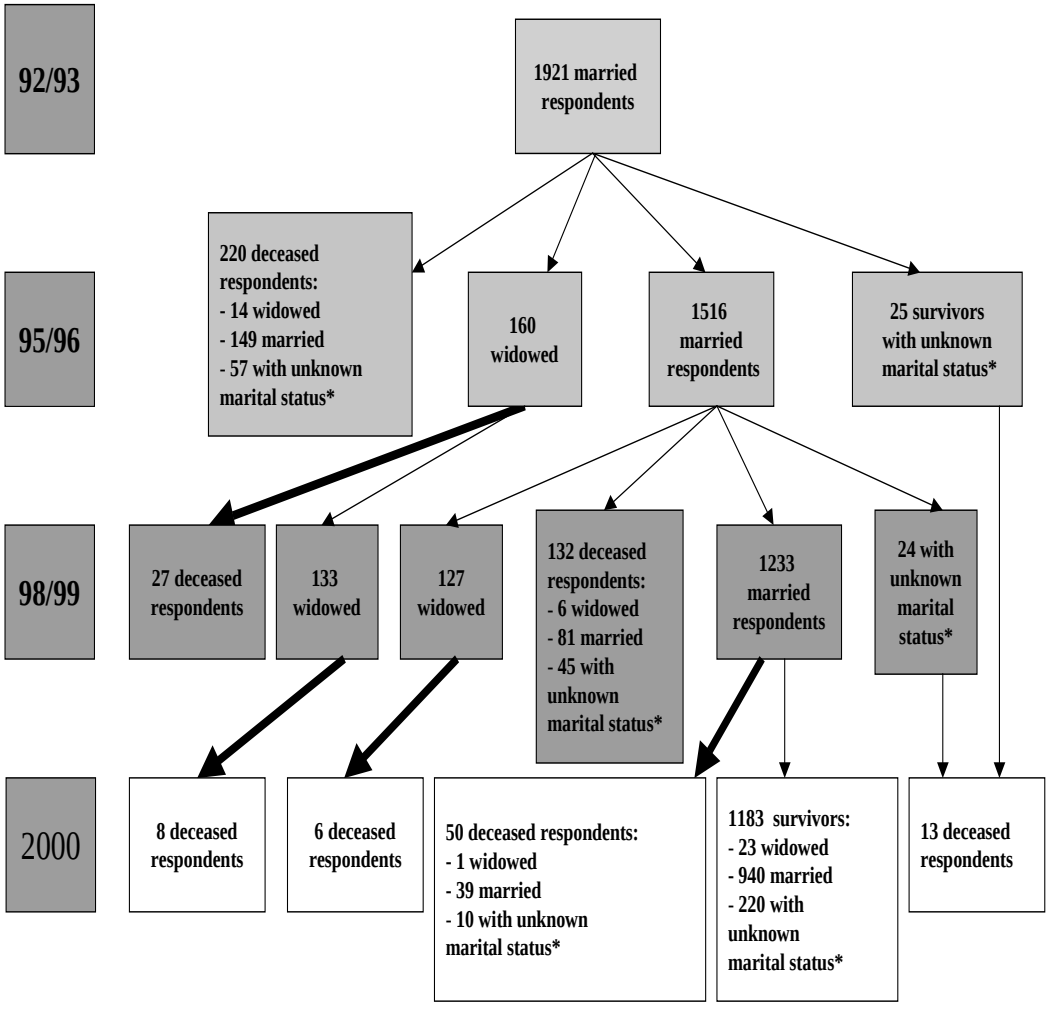
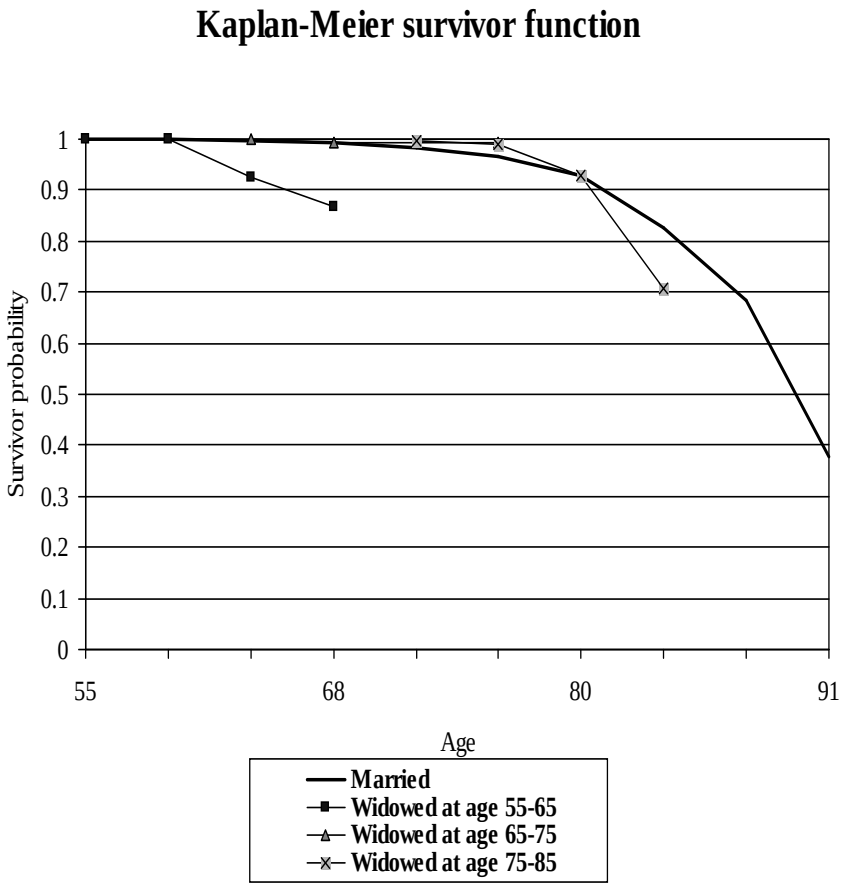


Figure 2: Kaplan-Meier survivor function



Health status is a crucial variable in our model. Health is a multidimensional concept and especially at advanced ages, striking differences are observed between individuals as well as over time. We use an extensive set of 22 health measures that each describe different aspects of health, but together provide a complete picture of health.[6] Physical functioning is measured by a self-reported test on mobility (van Sonsbeek 1988) and by a performance test of physical ability (Gulranik, 1994). Cognitive status is assessed using the Mini Mental State Examination (MMSE, Folstein, 1975). The Center for Epidemiologic Studies Depression Scale (CES-D, Radloff, 1977) is used to measure emotional functioning of older individuals. Two self-reported items on difficulties with seeing and hearing measure perception. Finally, the presence of chronic diseases is assessed by asking the participants whether they have or have had any of the following diseases: chronic obstructive pulmonary diseases (COPD), heart diseases, atherosclerosis, stroke, diabetes, arthritis, cancer, and other chronic diseases. These diseases are the most prevalent ones in older persons. To assess the severity of each disease, it is asked whether respondents follow a medical treatment. Unfortunately, the information on health is only available for the head respondent, and not for the spouse.[7]

A problem with the high dimensionality of the health data is that it will be difficult to use these indicators simultaneously in an empirical model for health and mortality. In Section 3, we discuss a flexible non-parametric data reduction method called the Grade of Membership method. Before we do this, we first briefly list socio-demographic variables that we will use in our analyzes.

Income is derived from a question where respondents were asked to assign their monthly total income - derived from pension, savings, dividends, and other sources - to four categories (in $): 0 - 794 (in line with the Dutch minimum income), 795 - 1,134, 1,134 - 1,815 and more than 1,815.[8] For the spouse, we use as proxy for income the occupational prestige of the longest job according to Sixma and Ultee (1983) (ranging from 0 = "never had job" until 87 = "high prestige"). A categorical variable indicating the level of *education* attained is used as a supplemental measure of socioeconomic status and is determined by the question: "Which is the highest education level attained?" Nine categories were reported

[6]The 22 health measures have been selected in collaboration with gerontologists and epidemiologists.

[7]This will of course affect our empirical model. We return to this issue in Section 4.

[8]Missing values for income were relatively frequent (almost 15%) and were imputed on the basis of results of regression analyses of income on demographic and socioeconomic variables such as age, cohort, gender, education, and degree of urbanization of the municipality where the respondent lives.

varying from "elementary education not completed" to "university education". The degree of *urbanization* of the area where the respondent lives (categorical variable ranging from 1 = "low" until 10 ="high") is an indicator of the external living conditions and may influence positively or negatively the probabilities of dying of older individuals through a variety of mediating factors – such as feelings of insecurity, pollution, and availability of both formal and informal caregivers. The variable "*network size*" (Van Tilburg et al. 1992) indicates the number of network members – including children, other family members, friends, and neighbors – who have regular contacts with the main respondent. The size of the social network may affect morbidity or mortality in different ways. Previous studies indicate that having children is one of the best predictors of formal and informal care (Norton 2000). Our network variable includes this. We opted for the variable "network size" instead of a set of variables measuring the number and gender of children because "network size" excludes children with whom older individuals do not have any contact, or who do not support their parents. A variable indicating the *frequency of church attendance* is included in our analyses. The strength of church affiliation may give some information on the lifestyle of the respondent, and also on the way the respondent deals with the bereavement process. The variable takes on value 0 if the respondent does not go to any church and ranges from value 1 ("yearly or less") until 5 ("weekly or more"). We also include age and gender.

The quality of marital union may affect the way widowed individuals adjust to the loss of the spouse (see for instance Van Baarsen and Broese van Groenou, 2001, Prigerson et al., 2000). Unfortunately, no reliable information on marital satisfaction is available in the data set. The duration of the marital union is included in the analyses as a proxy for the quality of marriage.[9]

Table 1 below presents the means of the demographic and socio-economic variables.

[9]We will allow for time constant unobserved individual components in our model. This will account for the average quality of the marriage.

Table 1: Demographic and socioeconomic characteristics of the head (Wave I)

Variables	Score	Wave I (in %)
Number of individuals		1,743
Female		41.5
Age	55-65	43
	65-75	33.5
	75-85	23.4
Attained education level respondent	Low	58.1
	Medium	27.6
Income in $	< 793	14.4
	793-1134	37.0
	1134-1815	32.7
Degree of Urbanization	Low	14.0
	Medium	29.4
Church attendance	$\leq$ Yearly	45.6
	Monthly	10.6

3 The Grade of Membership method

3.1 General description

To describe the health condition of older individuals, a broad range of different measures is required to cover all dimensions of health. Above we discussed the set of 22 measures that we use in our study. As we aim to analyze the dynamics in health, we need a method that summarizes this extensive set of indicators into a manageable and meaningful health set of lower dimension. In addition, this data reduction method also needs to be as flexible as possible, given the complex nature of health and the way it is distributed across individuals in the sample. The Grade of Membership (GoM) method is perfectly suited for this.

The GoM, developed by Woodbury and Clive (1974), Woodbury, Clive and Garson (1978), and Woodbury and Manton (1982), is a statistical classification procedure, designed to summarize a complex set of symptoms for chronic diseases into a smaller number of ideal clinical disease types. These ideal clinical disease types are referred to as "pure types" in the GoM terminology. The method not only makes a partitioning of the data into a limited set of pure types, but it also provides for each individual in the sample different weights indicating the degree of similarity that an individual has with each of the pure types. These weights, called "grades of membership", sum up to unity over the classes. A weight of 1.0 means that an individual has all of the symptoms associated with the pure type (so this individual can be viewed as a "standard textbook case"), whereas

a weight of 0.0 indicates that the individual bears no similarities with the pure type at all. The method is similar to the well known and much applied factor analysis, but it has some distinctive properties that are particularly relevant for our study. We return to this after a more formal description of the method.

3.2 The method

Suppose that there are K underlying non overlapping pure (clinical disease) types. Suppose furthermore that we have access to a set of J variables or tests that together cover the symptoms of the underlying pure types present in a sample of I individuals. As the information in each test j can have multiple response categories, L_j, we can without loss of information code the test score of individual i in test j into a set of dichotomous indicators y_{ijl}, measuring whether or not individual i has responded or scored affirmative on the l^{th} outcome of test j.

Next, define λ_{kjl} as the probability that a person of exactly the k^{th} type has the l^{th} score on test j and define g_{ik} as the degree of proximity that individual i has with the pure (clinical disease) type k. So, if an individual is of the pure type k, then $g_{ik} = 1$ and the probability that he/she responds or scores affirmatively to the l^{th} outcome of test score j, namely $\Pr(y_{ijl} = 1)$, equals λ_{kjl}.[10] In other cases, where the individual is not of one of the pure types, the probabilities λ_{kjl} have to be weighted by the degrees of similarity (the grades of membership) of the individual in the class k. More specifically:

$$\Pr(y_{ijl} = 1) = \sum_k g_{ik}\lambda_{kjl}$$

where $0 \leq g_{ik} \leq 1$ and $\sum_k g_{ik} = 1, \ \forall\, i, k$. The indicators g_{ik} mix the probabilities λ_{kjl} to best represent the probability that $y_{ijl} = 1$.[11] The fact that an individual can belong to more than one group makes the GoM method very suitable for the application at hand. Elderly individuals can have characteristics from various different disease types, and the degree of involvement with a particular disease type can vary across individuals.

The likelihood function used to estimate the parameters g_{ik} and λ_{kjl} from cross-sectional data is based on the probabilities of responses. Specifically, it is a

[10] So $\lambda_{kjl} = 1$ indicates that a specific symptom is always associated with a specific pure type k.

[11] In the terminology of Manton, Woodbury, Stallard and Corder (1992): the λ_{kjl} determine the position of the K vertices of a $K-1$ dimensional simplex in the $J*(\sum_j L_j - 1)$ space. The weights g_{ik} are continuously distributed within the simplex and combine the extreme points of the simplex in a convex set.

simple independent multinomial given by:

$$L = \prod_i \prod_j \prod_l \Pr(y_{ijl} = 1)^{y_{ijl}} = \prod_i \prod_j \prod_l (\sum_k g_{ik}\lambda_{kjl})^{y_{ijl}}$$

This needs to be optimized with respect to g_{ik} and λ_{kjl} subject to the constraints:

$$\begin{aligned} 0 \leq g_{ik} \leq 1 \qquad & \forall\, i,k \\ \sum_k g_{ik} = 1 & \\ 0 \leq \lambda_{kjl} \leq 1 \qquad & \forall\, k,j,l \\ \sum_l \lambda_{kjl} = 1 & \end{aligned}$$

As in Woodbury and Manton (1982), we use approximate likelihood ratio tests for the determination of the order K (number of dimensions) of the model. The test statistic is approximately χ^2 distributed with the number of degrees of freedom equal to the number of respondents I minus one plus the number of variables j multiplied by the number of category per variable $\sum_j L_j$. We refer to Woodbury and Manton (1982) for more details.

Given K, we need to address the consistency of the estimates of the individual parameters g_{ik}, not in the least because we use the estimated individual parameters $\widehat{g}_{ik}$ in subsequent analyzes of health dynamics and mortality (Section 4). For consistency of $\widehat{g}_{ik}$, we have to rely on information growth in J or L_j. More specifically, consistency is ensured when $J * (L_j - 1)$ becomes large (see, for instance, Manton, Woodbury, Stallard and Corder, 1992). In our application $J * (L_j - 1)$ equals 25, which may be sufficient, for not too large K, to accurately estimate the individual parameters g_{ik}.

The GoM approach is similar to factor analysis. The factor loadings compare to λ_{kjl}, whereas the g_{ik} have a similar function as the factor scores. However, factor analysis typically makes distributional (normal) assumptions on the factor scores and the outcomes, whereas the GoM method requires no assumption on the distribution of g_{ik}. Moreover, the GoM method is well-suited for outcomes that can be represented by binary variables. This makes GoM suitable to capture the highly skewed and irregular health distribution among the aged. A practical difference is that factor scores are calculated after the factor loadings have been determined, whereas in the GoM method the parameters λ_{kjl} and g_{ik} are estimated simultaneously. See Manton, Woodbury, Stallard and Corder (1992) for a more detailed comparison.

3.3 Application to the data

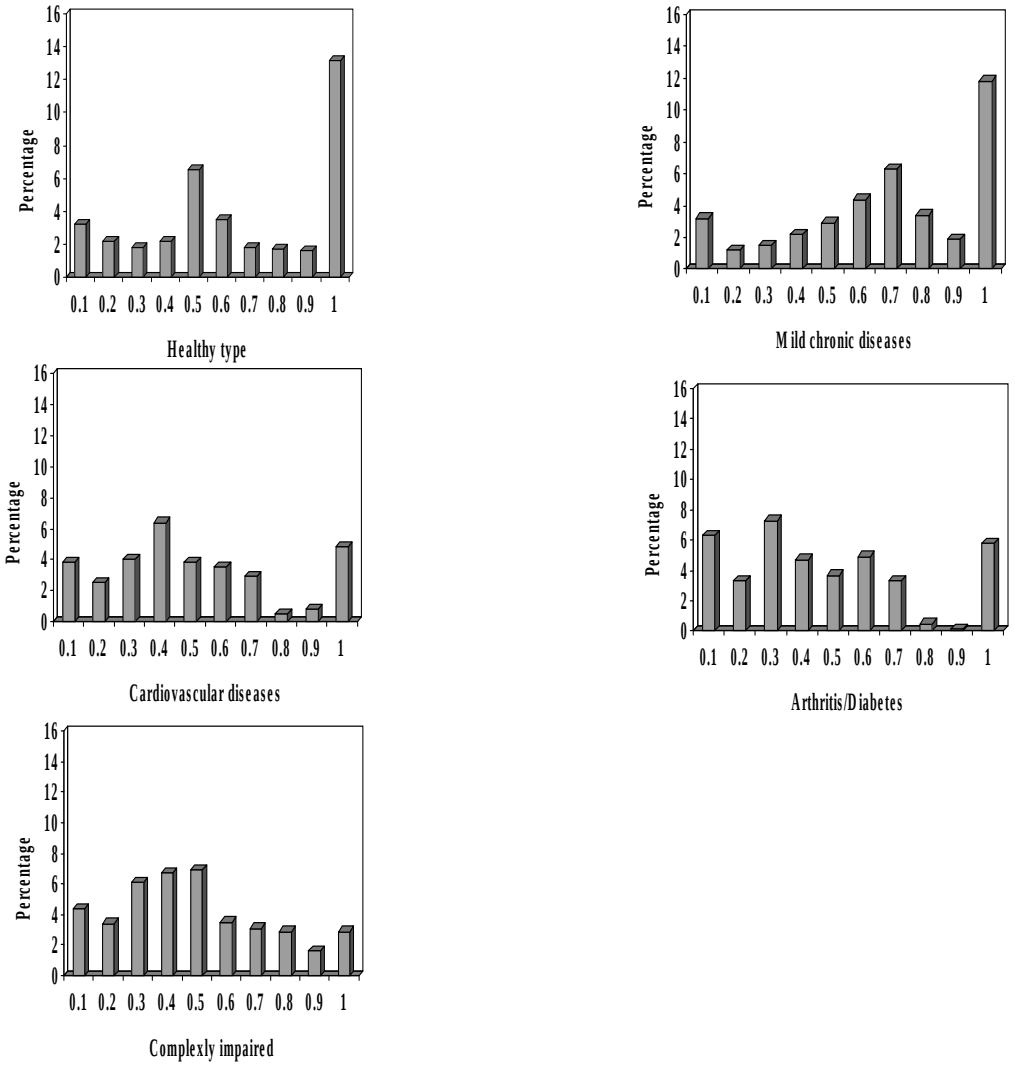
The GoM method is successively applied on the 22 health indicators (described in Section 2) of wave I, wave II, and wave III of the data set, using the typology derived in wave I. See, for instance, Portrait (2000) for details on the application of the GoM method in a longitudinal context. The GoM parameters λ_{kjl} – reported in Table A1 in appendix A – are used in the following to derive the characterization of the pure types.

The empirical results reveal that the health concept can be characterized using five underlying health dimensions. The first group (see the column for K=1 of Table A1 in Appendix A) is the healthy group, they do not suffer from any chronic diseases or functional limitations. The functional status of individuals who completely belong to the second health dimension (K=2) is very good, but they suffer from some mild depression[12] and/or the presence of "other chronic diseases". The latter are mainly diseases which are not specific to older individuals and generally not too serious. Examples of these are hypertension, back troubles, or diseases of the stomach, intestines, or nervous system. The third type is characterized by the presence of heart diseases and atherosclerosis – without any severe functional impairment except for some mild mobility limitations. The fourth group is characterized by the prevalence of serious arthritis and/or diabetes. It is also characterized by mobility limitations. The fifth type is complexly impaired, (s)he suffers from severe physical, emotional, and cognitive health disorders, reports mobility limitations, has a low score on the performance test and on the MMSE test, is depressed, and may suffer from severe respiratory diseases, stroke, and/or cancer. Note that the GoM method does not identify a profile characterized by high levels of depressive feelings and no cognitive or physical limitations. This may be explained by the fact that depressive symptoms alone are somewhat uncommon in older populations. Older individuals often suffer from physical or cognitive health disorders and these disorders are often related (or due to) emotional distress. Note that the pure types 2, 3, 4, and 5 are all associated to some extent with emotional disorders.

With respect to the graded participation into the different pure types (the g_{ik}), it can be noted that the larger part of our respondents participates only in two (30.8%) or three (36.5%) dimensions. This means that a large share of the respondents have a few g_{ik} (grades of membership/weights) strictly greater than zero, while others equal zero. The distribution of the GoM parameters display

[12]CES-D scores larger than 16 indicate depression. Table A1 indicates that there is a positive score on this item.

Figure 3: Distribution of the GoM parameters, wave I



extensive data heaping on a score of zero, meaning no participation in this specific health dimension. For instance, 62% of the individual parameters have a zero score in pure health type I and 69% have a zero score in pure health type III. Figure 3 displays the distributions of the grades of membership for the five types at wave I conditional on participation in the respective pure type (i.e. for strictly positive values of g_{ik}). No drastic and meaningful changes are observed in terms of the distribution of the individual GoM parameters at wave II and wave III (figures available on request by the authors). The figures also display substantive heaping at one. These respondents exactly match the pure (classical disease) type. For instance respondents with a score equal to one in the first dimension can be considered as completely healthy. Individuals in our sample are aged 55 and over, hence not surprisingly this happens for 13.2% of the cases in our sample. This implies that in our statistical model for health, measured by the individual parameters g_{ik}, we have to allow for heaping at both zero and one.

4 The model for mortality and health status, and its empirical implementation

4.1 Mortality

Let the couple $\{T^h, T^s\}$ be non-negative random variables describing the lifetimes of the head (main) respondent (h) and his/her spouse (s) respectively. Let x^h and x^s be observed and α^h and α^s be unobserved factors for the head and spouse, respectively. Our interest is in the lifetime of the head and how this is affected by the death of the spouse. More specifically, we are concerned with the conditional distribution $T^h|x^h, t^s, \alpha^h$.

We assume that the hazard of the conditional distribution, $T^h|x^h, t^s, \alpha^h$ is of the familiar Mixed Proportional Hazard (MPH) type and take it as (for notational convenience we omit the individual index):

$$\theta^h(t^h|x_t^h, t^s, \alpha^h; \beta_1, \delta) \;=\; \theta_0^h(t).\exp\{x_t^{h\prime}\beta_1 + f(I(t^h > t^s); \delta) + \alpha^h\} \qquad (1)$$

The set of explanatory variables x_t^h includes socio-demographic factors *and* health, i.e. $x_t^h = [\widetilde{x}_t^h,\, g_{1t}, g_{2t}, g_{3t}, g_{4t}, g_{5t}]$, with $\widetilde{x}_t^h$ socio-demographic variables and g_{kt}, $k = 1, ..., 5$ GoM health indicators. The function $f(I(t^h > t^s); \delta)$ is included to capture causal conjugal bereavement effects on the hazard rate of the head respondent that are not captured by the causal effects induced by g_{kt}.

Some comments are in order. Firstly, our model is estimated on data from a survey held at discrete points in time linked with administrative information on the vital status of husband and wife. The administrative data provide an almost continuous picture of the life histories over a period of seven to eight years. However, the health status of the head respondent is measured at, at most, three points in time. This is why the term $f(I(t^h > t^s); \delta)$ is included. It is supposed to capture the short-run effect of conjugal bereavement on mortality to the extent that this effect is not yet captured by the included health status variables as measured in the last survey wave before bereavement. (It would be preferable to specify a model in which the instantaneous or short-run effect does not vary across individuals with the timing of the survey interviews, but this would require much more frequent observations of health status changes in time.) We expect that in the longer run the effect of the realized bereavement before death of the head respondent will be absorbed by the health status indicators. In that case the value of $f(I(t^h > t^s); \delta)$ should converge to zero if $t^h - t^s$ becomes large. We do not impose this on our function $f(.; \delta)$, but expect to find this in the data.

Secondly, notice that (1) assumes that the hazard of the head respondent is affected *after* the death of the partner but not before. This rules out anticipation effects. This assumption is required to identify causal effects (see Abbring and Van den Berg, 2003, and the discussion below), and it is implicitly made in most of the literature on bereavement effects on the mortality rate. In the context of our model, the assumption basically means that individuals can not exactly predict the exact moment of the death of their partner. This is a reasonable assumption. One may argue that there are situations where individuals are to some extent able to predict the time of the death of their partner. For instance, in the situation where the partner suffers from a life-threatening disease like cancer. In that case, the hazard function may already start to rise before the actual death of the partner and the estimate of the effect of $f(I(t^h > t^s); \delta)$ may be biased. Note however that it is always difficult to exactly predict the timing of the death of the spouse. Also, we should point out that it is allowed that individuals know as much about the process leading to the partner's death as is specified in equation (2). In terms of the treatment evaluation literature: we allow the individual's knowledge of the treatment assignment process to lead to so-called ex ante effects on the rate at which the outcome of interest is realized. The identified causal effect is relative with respect to the counterfactual mortality rate in case the spouse has not died yet.

There is evidence that most individuals are able to cope with the situation of a terminally ill partner before the actual moment of death and that most of

the detrimental effects on the health of the head respondent take place just after the death of the spouse (see Carr et al., 2001, for a recent review on anticipation effects on adjustments to widowhood). Moreover, mechanisms that may play a role after bereavement, like being lost in zest of life, and poverty due to reduced income, actually take place after the death of the partner.

Note that the function $f(I(t^h > t^s); \delta)$ as well as x_t^h are endogenous determinants of T_h, since all are potentially affected by unobserved determinants. For example, there may be selectivity in marital formation, leading to a dependence of α^h and t^s. Also, the life style of an individual or previous investments in health may play a role in both the individual's mortality risk and the way in which health evolves at advanced ages, leading to a dependence between α^h and health. This makes health, i.e., g_{kt} and ultimately x_t^h, an endogenous regressor in the mortality equation. Fixed effect methods are not of much use here since we only observe a single lifetime spell for each respondent. So we have to specify the correlation between spousal bereavement and the unobservable α^h, and we have to consider the simultaneous determination of T^h and x_t^h.

We first examine the dependence of α^h and the determinants of t^s. For this we extend the conditional model of mortality for the head with a model explaining spousal mortality. We use the following MPH specification of the hazard function of $T^s|\ x^s, \alpha^s$:

$$\theta^s(t^s|x_t^s, \alpha^s; \beta_2) = \theta_0^s(t^s).\exp\{x_t^{s\prime}\beta_2 + \alpha^s\} \tag{2}$$

As noted earlier, the data lack some valuable partner information. The data contain only limited information on his or her health status, and, more importantly, we can not follow the spouse once the head respondent dies. For this reason we do not allow for any effect of the death of the head respondent on the hazard rate of the spouse. Equation (2) should therefore be viewed as a pure reduced from equation.

Identification of the causal effect of spousal bereavement in duration models is different from identification in linear models and most other non-linear models. Abbring and Van den Berg (2003) prove and discuss extensively the identification of treatment effects in duration models with selective assignment. They show that, within our context of the class of MPH models, some residual randomness in the timing of the cause-event (here: bereavement) is sufficient to identify the causal treatment effect $f(I(t^h > t^s); \delta)$, provided that the explanatory variables are exogenous and independent of the unobserved heterogeneity terms α^h, α^s. Identification does not require exclusion restrictions or assumptions on the functional form of the baseline hazards or the mixing distribution (distribution of α^h,

α^s). This result provides some intuition for the identification of our parameter δ, although we have endogenous regressors x_t^h in the mortality rate of the head respondent (see next subsection).

4.2 Health evolution

We now turn to the second endogeneity issue, namely that health, as included in x_t^h, is an endogenous regressor for our mortality model because it may be related to α_h. To deal with this, we extend the mortality-rate model equations with health evolution equations. Recall that health is characterized by the individual GoM parameters g_{kt} $(t = 1, 2, .., 5)$ of the head respondent. The GoM parameters show heaping at zero and one. We therefore opted for a two-limit Tobit panel model to characterize the dynamics in health. More specifically, we specify for each health typology the latent variable g_{kt}^* governing the individual outcomes g_{kt}, as (for notational convenience we omit the individual index):

$$g_{kt}^* = \sum_{l=1}^{5} g_{lt-1}\gamma_{l1} + \widetilde{x}_t'\gamma_{k2} + f(I(t^s < t^h); \gamma_{k3}) + \zeta_k + u_{kt} \tag{3}$$

for $k = 1, ..., 5$, and where $g_{kt} := g_{kt}^*$ if $0 \leq g_{kt}^* \leq 1$, $g_{kt} := 0$ if $g_{kt}^* < 0$, and $g_{kt} := 1$ if $g_{kt}^* > 1$. Previous health captures dynamic health evolution. The health g_{kt-1} at time $t-1$ in the pure health state k captures state dependence. Note that $t-1$ is short-hand notation for "the previous wave in the survey" (we discuss initial conditions in Subsection 4.3 on empirical implementation). Furthermore, since co-morbidity is a common phenomenon among older individuals, and the occurrence of one disease may affect the likelihood of getting another disease, we also include the lagged health indicators in the other states, $g_{k',t-1}, k' \neq k$. The vector $\widetilde{x}_t$ refers to socio-demographic variables. As in the mortality equation, we allow for a direct effect $f(I(t^s < t^h); \gamma_{k3})$ of bereavement. Like $f(I(t^s < t^h); \delta)$ in the mortality equation, this additional "bereavement function" is expected to mostly capture short-run effects. In the longer run, the effect of bereavement will be absorbed by the lagged health variables.

As with mortality, real-life anticipation to spousal death may affect health before the actual death of the partner. We argued previously that this effect is likely not to be important, but in case it affects our results, it most likely leads to a downward bias of the bereavement effect.

The variables u_{kt} are idiosyncratic shocks assumed to be uncorrelated. On the other hand, the unobserved individual characteristics $\zeta_k, k = 1, .., 5$, are allowed to be related to α_h and α_s. This captures common unobserved determinants of

health evolution and mortality of the head respondent and the spouse.

4.3 Empirical implementation

Equations (1) and (2) as well as the five equations of (3) constitute the full model – referred to as Model I. The different parts of the models are linked because of possibly correlated unobservables and because of direct effects (i.e. the effects of bereavement and health on mortality of the head respondent and the effect of bereavement on health). The structure of the model is such that joint estimation is required.

We now discuss the regressors and other model determinants in the model. First, consider the mortality rate (1). We split the total causal effect of bereavement on mortality into a short-run effect and a long-run effect. The short-run effect is the effect on the mortality rate between the moment of bereavement and the moment of the first survey wave that occurs after bereavement. This effect is captured by the "bereavement function" $f(.;\delta)$ on that specific time interval. The long-run effect concerns the effect on the mortality rate after the first survey wave that occurs after bereavement. Now part (or all) of the bereavement effect is also captured in the included health variables. The shape of $f(.;\delta)$ on this time interval will pick up any effect of bereavement on the hazard that are not captured by the observed health status evolution. We adopt the following functional form for f: in the short-run time interval it is quadratic in the time since bereavement, and in the long-run time interval it is also quadratic in the time since bereavement, but the two quadratic functions are unrelated, so we do not impose parameter restrictions across the quadratic functions.

The set of remaining explanatory variables $\widetilde{x}$ includes time-varying variables (such as income and health) and time-constant variables (such as gender, education, urbanization, network size, and active church attendance.[13]). Information on time-varying variables (such as health and income) is not always available for every wave. Information at some waves is missing for respondents with a telephone interview or respondents who refused to participate in wave II (201 cases and 88 cases, respectively) or wave III (135 and 110 cases, respectively). Nevertheless, we include these individuals in the sample. We observe their vital status from the register data, We control for the lack of recent health and income information by including a "refusal" dummy variable indicating whether the respondents leave the sample before the first of January 2000 for other reasons than death. Hence,

[13]Urbanization, network size, and active church attendance are measured at the first wave and are held constant during the study period.

this dummy variable is merely included to control for the fact that the information of earlier waves on time-varying explanatory variables is not updated.

The baseline hazards $\theta_0^n(t)$, $n \in \{h, s\}$ of hazards (1) and (2) are taken as piecewise constant functions of age that are allowed to change every four years (except at younger and older ages since too few deaths were recorded in these age classes to allow for a more detailed description of the data).

The health variables g_{kt} sum up to unity over k for each individual at each point in time. We choose to exclude the equation related to the healthy dimension (for g_1) from our calculations to avoid perfect correlation.[14]

The health modelling is dynamic, so we face an initial conditions problem. We deal with this in the common way (see for instance Heckman, Manski, and McFadden (1981) or Gritz (1993)) by specifying an extra auxiliary model equation for observed health at the first wave. This includes unobservable health determinants ν_k that are allowed to be correlated with the other unobservables of the model (α^h, α^s and $\zeta_k, k = 2, .., 5$). The estimation results may be sensitive to aspects of the specification of this initial health equation, notably that the included regressors are exogenous and that these are orthogonal to the unobservable ν. Especially the latter assumption may be violated in practice. Here we reach the limit of what is feasible in our study. It is fair to state that our model framework and data are rich in terms of the changes over time in health as a determinant of high-age mortality, and the model deals with joint selectivity in health evolution and conjugal bereavement. The price to be paid is that we need to deal with initial health conditions in a rather ad hoc way.[15] To gauge to what extent our results depend on the initial conditions, we also estimate a model – referred to in the following as Model II – where hazards (1) and (2) are estimated along with a static version of (3). We return to this in the next section.

The likelihood function associated with our Model I (equations (1), (2), and (3)) plus the initial health conditions equation is not straightforward to derive or compute. Firstly, as documented in the data section, in some situations we can only partially observe health and vital status of the individuals.[16] Survival

[14] Alternatively, we could have opted for the equivalent, but more complicated solution to estimate the full model subject to the restriction that the five $g's$ have to sum up to unity.

[15] It is hard to justify exclusion restrictions on the set of explanatory variables that is allowed to have a direct causal effect on mortality, so that instrumental variable techniques are not useful here. Identification of our models benefits from the data-driven assumption that the health-status regressors change values at a finite number of points only.

[16] It is important to emphasize that the likelihood function is corrected for the fact that the interviews of each wave are held at different points in time. For instance, the duration of the follow-up of respondents that are still alive at the end of the panel varies between 2,280 and

data are right censored for the head respondent (1) when he or she is alive at the first of January 2000 and (2) when he or she starts a new relationship after spousal bereavement. With respect to the spouses, data on survival status are right-censored (1) when both the head respondent and his/her spouse are alive at the end of the panel and (2) when the head respondent dies during the period of observation and his or her spouse is alive at the moment of death. In these cases, we take the censoring as independent right censoring, with obvious modifications to our likelihood function. Less straightforward is the case for respondents living in Amsterdam. For privacy reasons, the municipality of Amsterdam refuses to provide records on spouses. Consequently, for these respondents, we have a gap for the vital status of the spouse between the last held interview and the point where observation for the head respondent stops (i.e. the 1st of January 2000 if the respondent is still alive, or the moment of death if the respondent died during the period of observation). This means that we have to "integrate out" the life time T^s of the partner from the last held interview up to the point where observation stops. We do this in a way that is consistent with our model (i.e. we use model (2) explicitly).

Secondly, our model includes unobserved random effects. The standard procedure to deal with random effects is to specify the likelihood conditional on the unobservables of the model and to integrate these unobservables out of the likelihood function. This assumes independence between observed exogenous characteristics $\widetilde{x}$ and unobserved characteristics $(\alpha^h, \alpha^s, \zeta_k, \nu_k, k = 2, \ldots, 5$. Our sample is taken from the older (55+) population. In the construction of our likelihood we condition on survival beyond age 55. Van den Berg and Lindeboom (1995) show that in this conditional approach, independence in the population carries over to independence in the sample distribution under relatively weak conditions.

Thirdly, the large number of unobservables in our model poses formidable computational obstacles, even with simulation estimation methods. We reduce the dimensionality by adopting a one-factor loading specification for the unobserved characteristics ν_k of the initial conditions. Specifically, we take $(\alpha^h, \alpha^s, \zeta_2, \zeta_3, \zeta_4, \zeta_5, \nu_2)$ jointly normal distributed and assume that $\nu_k = \phi_k \nu_2, \quad k = 3, \ldots, 5$. The parameters ϕ_k are estimated along with the other parameters of the model. Furthermore, the residuals u_{kt} of the health evolution equations are assumed to be independently (across health dimensions k and periods t) normally distributed with zero mean and variance σ_{k1}. The likelihood function then con-

2,655 days – depending on the timing of the first LASA interview. In our estimation procedure, ages of respondents at the timing of the interviews as well as the timing of bereavement are thoroughly taken into account to increase the precision of our estimates.

tains 7-dimensional integrals (over $(\alpha^h, \alpha^s, \zeta_2, \zeta_3, \zeta_4, \zeta_5, \nu_2)$), for which no closed form solution exists. We use Simulation Methods to estimate the model. In particular, we opted for Simulated Maximum Likelihood (SML) (see, for instance, Stern 1997).

The random variables $S = (\alpha^h, \alpha^s, \zeta_2, \zeta_3, \zeta_4, \zeta_5, \nu_2)$ are assumed to be normally distributed with mean 0 and covariance matrix Σ. It is well-known that any symmetric positive definite matrix Σ can be written as the product of $L * L' = \Sigma$, where L is lower triangular. In order to simulate S, we first simulate a matrix ϵ of standard normally distributed variables. It follows from standard statistical theory that $L\epsilon$ is normally distributed with mean 0 and variance Σ. The parameters of matrix L are estimated and used to compute matrix Σ. The Delta method is used to estimate the standard errors of the parameters of matrix Σ.

The number of replications using Simulated Maximum Likelihood methods has to be infinite to ensure consistency of estimated parameters. We estimated Model I successively using an increasing number of replications. Beyond 20 drawings the results appear to be very stable. The results presented in the following sections are based on 30 replications.

5 Model estimation results

The parameter estimates for Model I are reported in Tables 2a (below) and 2b (Appendix B) and in Figures 4, 5 and 6.

The upper panel of Table 2a reports the parameters of the mortality hazard of the head respondent. Not surprisingly, the parameters of the baseline hazard show an exponentially increasing function. The other coefficients also have the expected signs. Females, individuals with higher incomes and religiously affiliated people, and individuals with a large social network face lower mortality risks. The last effect may be explained by different life style and social support of individuals who frequently attend religious services. People living in urban areas have higher mortality rates. Note that these effects are conditional on an individual's health status. Concerning health, individuals with higher involvement (grades of membership) in dimension/health typology 5 (complexly impaired), 3 (cardiovascular diseases), and to a lesser extent, in dimension 2 (other chronic diseases) have increased mortality risks. The remaining health dimension (4) is characterized by two, not directly, life threatening diseases (arthritis and diabetes) and we consequently find little effects of it on the mortality rate. The dummy variable "refusals" indicates whether the individual drops out of the subsequent wave(s).

It is strongly significant,[17] most likely because it captures time-varying health effects for such individuals.

Both short-run and long-run bereavement effects variables are significantly different from 0.[18] Figure 5 depicts the total effect of bereavement. We find that the loss of a partner significantly increases the mortality rate and that the effect is stronger during the first three years of bereavement, decreases afterwards, and disappears after approximately seven years of widowhood. This result is consistent with previous findings in the literature (Lichtenstein et al., 1998). Note that the bereavement variables measure the direct effect of bereavement, as far as this is not included in health. We need to take the changes in health, due to bereavement into account in order to assess the total effect of bereavement on mortality. We return to this later.

Our findings are robust to alternative specifications. We tried various specifications like (a function of) the logarithm of time since bereavement, year dummies for short-run and long-run effects etc. The results do not change and the highest likelihood value is obtained using the specification presented above. We also estimated the model allowing for different effects of widowhood according to (1) gender, (2) the duration of the marriage, (3) age, (4) the number of times an individual became widowed (5) strength of religion, and (6) health status. The parameters associated with the interaction variables were never significant. Therefore, we did not pursue this any further.

The lower panel of Table 2a reports the results of the health model. This concerns estimates of a dynamic panel data model, with the GoM parameters g_{kt}, measuring the degree of involvement in health type $k = 2, .., 5$ as health measures.[19] We find strong effects of lagged health. The own lagged variables - that account for state dependence - are strongly significant and greater than one, indicating that a given condition deteriorates over time. The significant lagged health indicators in the other health states are also positive, showing that pertaining to a specific health type increases the probability of suffering from other health disorders. For instance suffering from other chronic diseases at $(t-1)$ increases the probability of having arthritis and/or diabetes, of having cardiovascular diseases, and to a lesser extent of being complexly impaired at (t). Likewise, suffering from arthritis and/or diabetes at $(t-1)$ increases the probability of being complexly impaired and (to a lesser extent) increases the

[17] Recall that we use register data to follow individuals from the start of our sample period (1992) up to the end of the observation window.

[18] See Subsection 4.2 for the definitions of the short- and long-run effects.

[19] The sum over k of g_{kt} equals one, so we omit one of the categories (see Subsection 4.2).

Table 2a: Results of Model I (first part): Mortality and Health Status of Head

Mortality	Coeff.	t-value	Baseline hazard	Coeff.	t-value
Female	-0.412	-3.45	γ_1 (55/62)	0.002	2.59
Education	-0.005	-0.20	γ_2 (63/66)	0.005	2.83
Income	-0.916	-3.92	γ_3 (67/70)	0.008	3.06
Urbanization	0.028	1.68	γ_4 (71/74)	0.016	3.31
Church attendance	-0.118	-4.32	γ_5 (75/78)	0.043	3.37
Network size	-0.041	-7.33	γ_6 (79/82)	0.095	3.45
TSB (short run)	-0.331	-1.21	γ_7 (83/86)	0.262	3.46
Quadratic TSB (short run)	0.307	3.75	γ_8 (87/92)	0.365	3.03
TSB (long run)	0.721	2.81			
Quadratic TSB (long run)	-0.112	-1.90			
Dummy refusals	-1.523	-11.30			
Other Chronic diseases	0.445	2.26			
Cardiovascular diseases	0.650	3.44			
Arthritis/Diabetes	0.125	0.54			
Complexly impaired	1.346	7.25			
Health evolution equations					
Other Chronic diseases			Cardiovascular diseases		
Constant	-1.004	-4.14	Constant	-0.276	-2.88
Other Chronic diseases(t-1)	0.959	8.04	Other Chronic diseases(t-1)	0.232	5.78
Cardiovascular diseases(t-1)	-0.031	-0.20	Cardiovascular diseases(t-1)	1.451	18.46
Arthritis/Diabetes(t-1)	0.247	1.67	Arthritis/Diabetes(t-1)	-0.032	-0.60
Complexly impaired(t-1)	0.094	0.59	Complexly impaired(t-1)	0.055	0.88
Age	0.204	0.30	Age	0.247	0.86
Age^2	-0.206	-0.30	Age^2	-0.145	-0.49
Female	0.234	1.70	Female	-0.198	-3.50
Education	0.093	0.52	Education	0.003	0.03
Income	0.121	0.75	Income	-0.117	-1.60
Church attendance	-0.105	-1.16	Church attendance	-0.036	-0.92
Urbanization	-0.183	-1.69	Urbanization	0.009	0.20
TSB (short run)	0.431	1.01	TSB (short run)	-0.133	-0.65
TSB (long run)	-0.137	-0.57	TSB (long run)	-0.009	-0.08
σ_1	0.953	8.74	σ_2	0.186	15.53
Arthritis/Diabetes			Complexly impaired		
Constant	-0.696	-5.89	Constant	-0.222	-2.53
Other Chronic diseases(t-1)	0.300	7.00	Other Chronic diseases(t-1)	0.133	3.80
Cardiovascular diseases(t-1)	0.065	0.95	Cardiovascular diseases(t-1)	0.026	0.48
Arthritis/Diabetes(t-1)	1.249	16.39	Arthritis/Diabetes(t-1)	0.143	2.88
Complexly impaired(t-1)	0.081	1.16	Complexly impaired(t-1)	1.13	16.76
Age	0.128	0.40	Age	0.397	1.57
Age^2	-0.101	-0.31	Age^2	0.042	0.17
Female	0.310	4.64	Female	-0.116	-2.19
Education	0.098	1.12	Education	-0.140	-2.14
Income	-0.144	-1.93	Income	-0.053	-0.86
Urbanization	0.064	1.24	Urbanization	-0.029	-0.70
Church attendance	0.027	0.64	Church attendance	0.016	0.48
TSB (short run)	0.394	2.05	TSB (short run)	-0.223	-1.24
TSB (long run)	0.089	0.78	TSB (long run)	-0.057	-0.65
σ_3	0.239	16.83	σ_4	0.166	16.74
Number of observations		1743	mean(log-likelihood)		-9.6068

Note: TSB = Time since bereavement

Figure 4: Covariance matrix of the unobserved effects (Model I);

$\alpha_{Partner}$	**0.0002** **(0.031)**						
$\alpha_{Respondent}$	**-0.0002** **(-0.060)**	**0.0002** **(0.024)**					
$\alpha_{G1(other\ diseases)}$	**0.0006** **(0.125)**	**-0.0008** **(-0.216)**	**0.005** **(0.174)**				
$\alpha_{G2\ (complexly\ imp.)}$	**0.0003** **(0.074)**	**-0.0003** **(-0.159)**	**-0.0002** **(-0.054)**	**0.0016** **(0.812)**			
$\alpha_{G4\ (Arthritis/Diabetes)}$	**-0.0002** **(-0.107)**	**0.0002** **(0.019)**	**0.0008** **(0.084)**	**-0.0014** **(-0.206)**	**0.0052** **(2.333)**		
$\alpha_{G5\ (Cardiovascular\ dis.)}$	**-0.0017** **(-0.754)**	**0.0023** **(0.224)**	**-0.011** **(-2.188)**	**-0.002** **(-1.223)**	**-0.006** **(-4.287)**	**0.041** **(8.047)**	
$\alpha_{Initial\ Cond.}$	**0.0002** **(0.062)**	**-0.0007** **(-0.231)**	**0.008** **(1.657)**	**-0.003** **(0.698)**	**0.007** **(0.364)**	**-0.021** **(-1.642)**	**0.025** **(3.126)**

(T-values into brackets; calculated using the Delta method)

Figure 5: Total effect of bereavement on mortality (Model I);

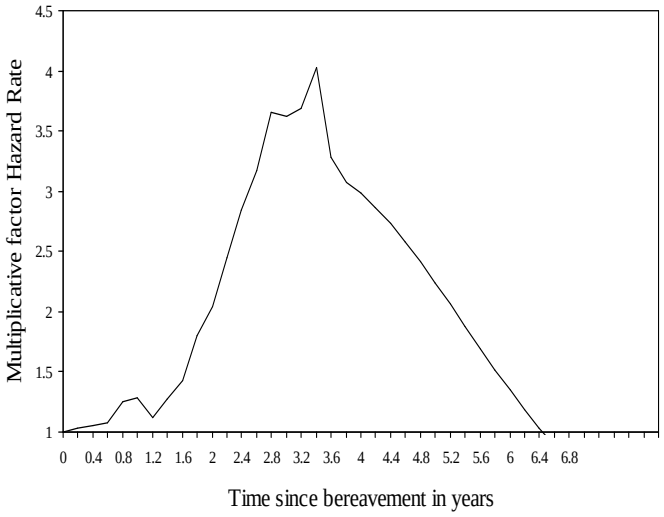
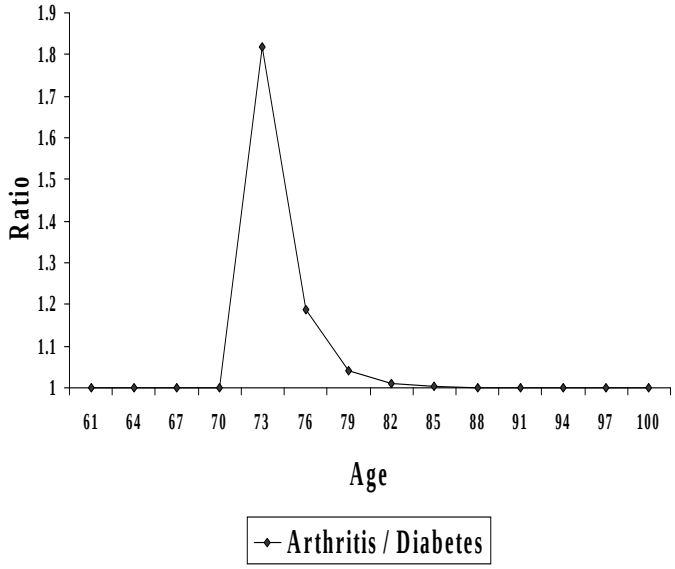


Figure 6: Ratio of g(bereavement at age 70) / g(no bereavement) for arthritus/diabetes



risk of experiencing "Other chronic diseases" at (t). So indirectly, a not directly life threatening condition like diabetes can lead to increased mortality risks. This is a well established fact in the medical literature (see e.g. Nathan, 1993).

We observe only a few age effects after we control for lagged health status.[20] Only the age parameters of "complexly impaired" are jointly significant. They indicate that the shifts in grades of membership are larger at older ages than at younger ages. Differences are found with respect to gender. Females suffer more often from arthritis, diabetes, and, to a lesser extent, from other chronic diseases than males and males suffer more often from cardiovascular diseases and complex impairment than females. Consequently, at older ages females experience less directly life threatening disorders. This finding is consistent with the fact that females live on average longer than males. Finally, we find significant effects of socioeconomic status, measured by income and education. Having high incomes and/or being well educated positively affect(s) health status; it lowers the probability of suffering from arthritis/diabetes, cardiovascular diseases, and being complexly impaired. This result is similar to the finding of Attanasio and Emmerson (2003), who also find an additional effect of socioeconomic status on

[20]The age effects are stronger in the static model; see Table 2c in Appendix C.

health status after correcting for initial health status. Urbanization and church attendance do not influence health evolution, but these factors are important in the static model for health (see Table 2c in Appendix C).

With respect to the effects of bereavement on health, we start with mentioning that we tried a range of different specifications : (1) using dummies for spousal bereavement, (2) using quadratic specifications of the short-run and long-run effect of bereavement (as in hazard (1)), (3) using the logarithm of the time since death of the spouse. We also estimated the model allowing for different bereavement effects for widows and widowers. These alternative specifications did not lead to better results and the interaction variables were not significant in any of the alternative specifications.

Table 2a shows that spousal bereavement significantly increases the probability of suffering from Arthritis/Diabetes. Bereavement has no direct effect on the other health dimensions.[21] It is amenable that bereaved individuals experience a reduction of their level of physical activity, a worsening of their eating patterns and sleeping disturbances. These lifestyle factors may increase the risk of suffering from arthritis and diabetes, which are to a certain extent related to the level of physical activity and to eating patterns. Prigerson et al. (1997, 2000) also found that bereavement increases the probability of suffering from chronic diseases.[22]

Only the short term effect is significant. This does not mean that there are no longer run effects. These may be picked up by the lagged health status variable. To illustrate this we used our model to calculate expected health paths for an average male respondent for ages 61 to 100. In one situation we assumed that the partner remains alive, in the other situation we assumed that his partner died when he was 70 years old.

Figure 6 displays the ratio of the two health paths. The general picture is that there is a strong immediate effect of bereavement on arthritus/diabetes. At

[21]We should point out that the precision of the estimates discussed above may be influenced by some shortcomings of the data. First, the health information of individuals who refuse to continue to participate in the LASA study is not available after they have left the sample. Note that about one third of them refuses because of health problems. Second, we can not measure the health status of an individual who lost his/her spouse and who dies before the next wave. This effectively reduces the sample information with likely consequences for the precision of the estimates of the health evolution equations. Most probably, we are more likely to miss information on individuals suffering from more serious health disorders. In other words, the sample design and the dynamic selection in the sample may prevent us from demonstrating the direct effects of bereavement on dimensions with more serious health disorders.

[22]Our results do not imply that bereavement only affects physical health. As argued previously (Section 3), at advanced ages symptoms of emotional disorders are rarely observed without physical disorders. Also our typologies are associated with emotional disorders.

age 73 the expected degree of involvement (g) into arthritis/diabetes is about 80 percent higher for an individual who loses his partner at age 70.[23] This is due to the direct short-run effect. In the longer run there are no direct effects (the coefficient of the long-run effect is insignificant). However, the increased g at age 73 affects the outcome at age 76, which in turn influences the outcome at age 79 etc. We can see from the figure that the effect of bereavement lasts about 12 years. However, as in the mortality model, the direct effect is dominant; bereavement affects health most strongly in the first few years after the death of the partner. This is consistent with the literature on bereavement (see for instance Van Baarsen et al., 2002).

Bereavement affects the probability of suffering from Arthritis/diabetes, but Arthritis/diabetes does not have a direct effect on the mortality hazard (see above). There may be an indirect effect of bereavement on mortality. Suffering from arthritis and/or diabetes at $(t-1)$ increases the probability of being complexly impaired, which in turn has a strong effect on the individual mortality rate. We examined whether this indirect bereavement effect is strong, by looking at the additional effect of bereavement on being complexly impaired due to having a higher risk of arthritis/diabetes. We found that the increase in Arthritis/diabetes due to bereavement only marginally increases the probability of being complexly impaired.[24]

We can conclude from the above that there are strong direct effects of bereavement on the hazard and on the probability of suffering from Arthritis/diabetes. Both effects are temporary and fade out after 7 and 12 years, respectively. For mortality this is largely a direct effect. Indirect effects of bereavement (via subsequent health) on mortality are very small because Arthritis/diabetes is not a directly life threatening disease and because the direct and indirect effects of bereavement on life threatening diseases are too small to influence the mortality rate.

In Section 6 we will use the model to make calculations for males and females and look at the impact of bereavement on expected residual lifetimes and time spent in specific health states. The latter is denoted as health expectancies in the gerontological literature. Before we do that we first briefly discuss the estimates of the covariance matrix Σ and the fit of the model. The results of the mortality

[23]The expected g at age 73 is 0.05 when no bereavement takes place and 0.09 when the partner dies at age 70. See for instance Wooldrigde 2003, pages 567–569, for explicit expressions of expected values of the dependent variable in two-sided Tobit models.

[24]To be more specific, the expected g of being complexly impaired is increased by 1.6 percent at age 76 and the effect reduces slowly afterwards

equation of the spouse as well as the results of the initial conditions are reported in Appendix B. We do not comment on these results. These models are purely reduced form and it is therefore difficult to give a meaningful interpretation to the parameter estimates of these models.

Figure 4 displays the results of the covariance matrix Σ of the unobserved individual effects. A majority of the variances and covariances of the individual unobserved effects are not significant.[25] This seems to indicate that it is not necessary to estimate the mortality equation jointly with the health evolution equations. We performed a Wald test on the joint significance of the parameters of the matrix Σ. The Wald test rejects the null hypothesis of no correlation between $(\alpha^h, \alpha^s, \zeta_2, \zeta_3, \zeta_4, \zeta_5, \nu_2)$. Similarly, we perform a series of Wald tests for the joint significance of the variances and covariances of : (1) the head and spouse mortality equations, (2) the head mortality equation and (each of) the health equations, (3) the spouse mortality equation and (each of) the health equations, and (4) the health equations. The Wald tests indicate that the null hypothesis of no correlation between unobserved determinants of head and spouse mortality could not be rejected whereas the other null hypotheses of no correlation between unobserved determinants of head mortality and health evolution, between spouse mortality and health evolution, and between the health equations are rejected. This indicates that the shared risks of mortality between husband and wife go through unobserved characteristics that influence health status. Our conclusion from all these tests is that the mortality equations and the health equations should be jointly estimated.

We have performed an informal check of the fit of our health evolution model. Figure 9 of Appendix D is based on histograms of the average actual and estimated probabilities that a grade of membership g^*_{kt} falls in a specific interval. The estimated probabilities are calculated using parameters estimates of Model I. The dotted line connects the tops of the histogram of actual probabilities whereas the solid line connects the tops of the histogram of the estimated probabilities. A comparison of the graphs per health dimension indicates that the dynamic health model fits the observed data quite well. A check on the model fit of the bivariate mortality model is less straightforward.[26] However, we have modelled the lifetimes with a flexible piecewise constant baseline hazard, time-varying regressors and unobserved characteristics. Generally, it is believed that this is the

[25]The covariances in Model II (the static model) are strongly significant. The results are available upon request.

[26]We have stock sampled lifetimes, which makes it difficult to calculate (modified) Kaplan-Meier estimates that are comparable to the hazard rate predictions of the model.

most flexible specification within the class of MPH models. Finally, the results of Model I may depend on the specification of the initial conditions. To check on this, we compare the results of the survival models and of the health model in the static and dynamic specifications. The results are highly comparable, which indicates that our results are not sensitive to the treatment of the initial conditions problem in our model.

6 Life and health expectancies

Residual life expectancy and time spend in specific health states (called "health expectancies") are relevant for health care policy and frequently calculated in the demographic and gerontological literature. With our model we can differentiate these with respect to marital status and calculate the fraction of lifetime lost as well as the increase in health disorders resulting from bereavement.

6.1 Residual life expectancies

Expected residual lifetimes at age s are computed as (see, for instance, Lancaster 1990):

$$\mathrm{E}(s) = \frac{\int_s^\infty S(t)\,dt}{S(s)}$$

We compute the survivor function, $S(.)$ from the estimated model, for each sample respondent. Subsequently, we average the survivor functions per gender and within specific age intervals. The expected residual lifetimes $\mathrm{E}(s)$ are calculated per gender and marital status. We do the calculations at the sample average of the socioeconomic characteristics and health status. We report the results in Tables 3a and 3b.

Males and females lose 7.6 % (= 2.4 years of life) and 8.3 % (= 2.9 years of life) respectively of their residual life expectancy after bereavement at age 55-65. For bereavement at age 65-75, these percentages equal 11 and 12.1 and for bereavement at age 75-85, 16.0 and 17.3. So the loss of the spouse is found to significantly decrease the residual lifetimes of both genders and at all ages. Tables 3a and 3b also show that most of the effect arises in the first years of bereavement, during which the residual life expectancies are largely affected. Afterwards, the decline in remaining years of life are comparable for (still) married and widowed individuals. This is in line with what we concluded in the previous section. The

Table 3a: Residual life expectancies for males (in years).

Age	Married	Widowed		
		at age 55-65	at age 65-75	at age 75-85
Number of cases	958	3	31	49
55	31.5	29.1		
62	24.6	22.3		
66	20.7	18.4	18.4	
70	16.7	14.5	14.6	
74	13	10.8	11	11.2
78	9.5	7.7	7.9	8.1
81	6.4	5.4	5.6	5.8
85	3.6	3.0	3.2	3.4

Table 3b: Residual life expectancies for females (in years).

Age	Married	Widowed		
		at age 55-65	at age 65-75	at age 75-85
Number of cases	530	29	84	73
55	34.9	32.0		
62	27.9	25.0		
66	23.9	21.1	21.1	
70	20	17.2	17.2	
74	16.2	13.4	13.6	13.8
78	12.5	9.9	10.1	10.3
81	8.9	7.1	7.3	7.5
85	5.5	4.9	5.1	5.3

table also shows that widowhood at younger ages affects the remaining life expectancies the most. At all ages residual life expectancy after the death of the partner is lower for those who lose their spouse at a relatively young age (55-65) as compared to those who lose their partner at older ages (65-75 and 75-85).

The residual lifetimes based on our model exceed the numbers provided by Statistics Netherlands. Our sample concerns a group of married older persons and their characteristics differ from the characteristics of entire Dutch population. We report on this in Appendix E.

6.2 Health expectancies

Usually, the calculation of health expectancies involves the use of data on the prevalence of unhealthy states and mortality. Information on the prevalence of health states can be obtained from census of survey data and usually one uses life table information to calculate mortality rates. The average number of years spend in a health state x onwards from a given age s is then derived from (see e.g. Sullivan, 1971):

$$\mathrm{HE}(s,x) = \frac{\int_s^\infty \int_x h(t,u) S(t)\, du\, dt}{S(s)}$$

where $h(u,t)$ assigns weights to health states at age t.

Our approach differs from the the traditional "Sullivan method" in two ways. First, we base our calculations of residual lifetimes in specific health states on estimates of a joint model for health and mortality. Second, we do not use census data on the prevalence of unhealthy states in older population, but use instead the Grade of Membership and the estimates from our model. Our approach allows for more detail in the calculation of the health expectancies. We calculate the average grades of membership per age, gender, and marital status as weights ($h(t,k)=\overline{g_{kt}}$) and use these to dis-aggregate the residual life expectancies[27]

"Healthy life expectancy" or "active life expectancy" is a particular form of life expectancy that refers to the expected time spent in health states that are free from serious disability. "Healthy" health states for older population are generally associated with the absence of functional limitations. In our analyses, "healthy life expectancies" are given by $\mathrm{HE}(s,1)$, the number of years remaining in the

[27]Manton and Stallard (1991) also give estimates of health expectancies of the American older population using the GoM method. However, their method differs from ours. They first identify the different health dimensions from a cross section of a survey using the Grade of Membership method and use Census Data to derive life expectancies E(s) for specific age-gender groups. we use our model for this. They next combine both elements to derive health expectancies.

healthy first dimension. Estimates of residual health expectancies per marital status at age 70 and 80 are reported in Figures 7 and 8, for males and females respectively.

Figures 7 and 8 show that after bereavement the share of healthy residual years of life decrease for both genders. Bereaved males experience a worsening of health status as (relatively) healthy years – in dimension 1 (healthy) and 2 (other chronic diseases) – are replaced by unhealthy years – in dimension 4 (serious arthritis and diabetes) and 5 (complexly impaired). Males lose on average 8.5 % of their remaining healthy years as a consequence of widowhood. Similar effects are observed for younger (70 year old) females. So, the calculations show that after bereavement people spend a larger share of their remaining life suffering from (serious) chronic diseases In absolute terms, however, bereavement reduces time spend in unhealthy states. This is caused by the strong direct effect of bereavement on mortality. The reduction in life years due to bereavement are stronger than the effects of bereavement on health.

7 Conclusions

This study assesses the effects of spousal bereavement on health and mortality risks at advanced ages. For that purpose, we specify a bivariate survival model for husband and wife and a dynamic health evolution model and estimate it on a rich longitudinal survey. Our approach adds to the literature because we combine a number of relevant aspects. The mortality hazard of the head respondent allows for a direct effect of the death of the partner and a range of health indicators. The health indicators give an accurate description of all dimensions of individual health and are derived from a broader set of health indicators using a flexible, non-parametric, data reduction method called the Grade of Membership method. Health is treated as an endogenous regressor and the dynamic model for health also allows for a direct effect of spousal bereavement and indirect effect via lagged health.

We find much higher mortality hazards for individuals with cardiovascular diseases and complexly impaired individuals. The death of the partner significantly increases the mortality rate of the survivor. The effect is strong during the first three years and disappears after approximately seven years.

With respect to health, we find strong effects of lagged health, indicating on average aggravation of health disorders. Also the prevalence of one disease increases the risk of getting other diseases in the future. So initially not directly life threatening diseases (like arthritis or diabetes) increase the risk of obtain-

Figure 7: Health expectancies for older males

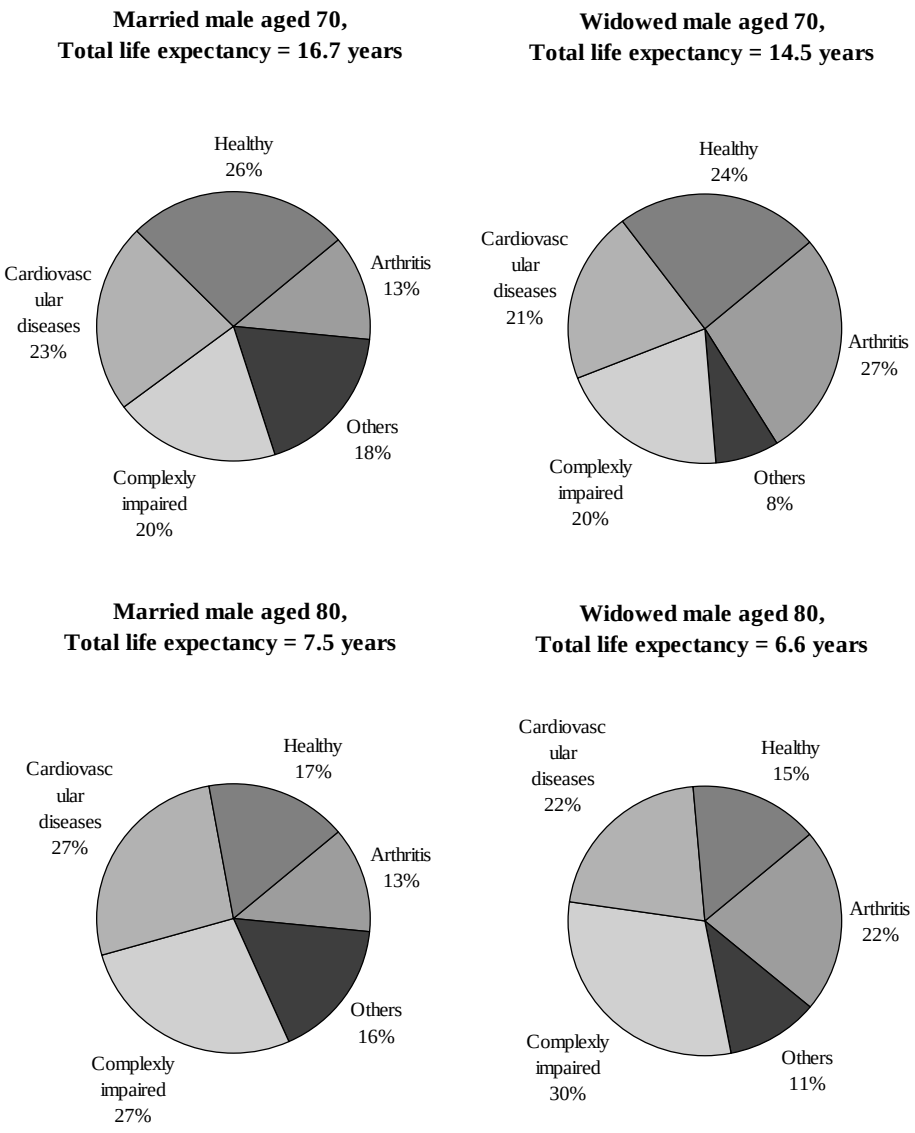
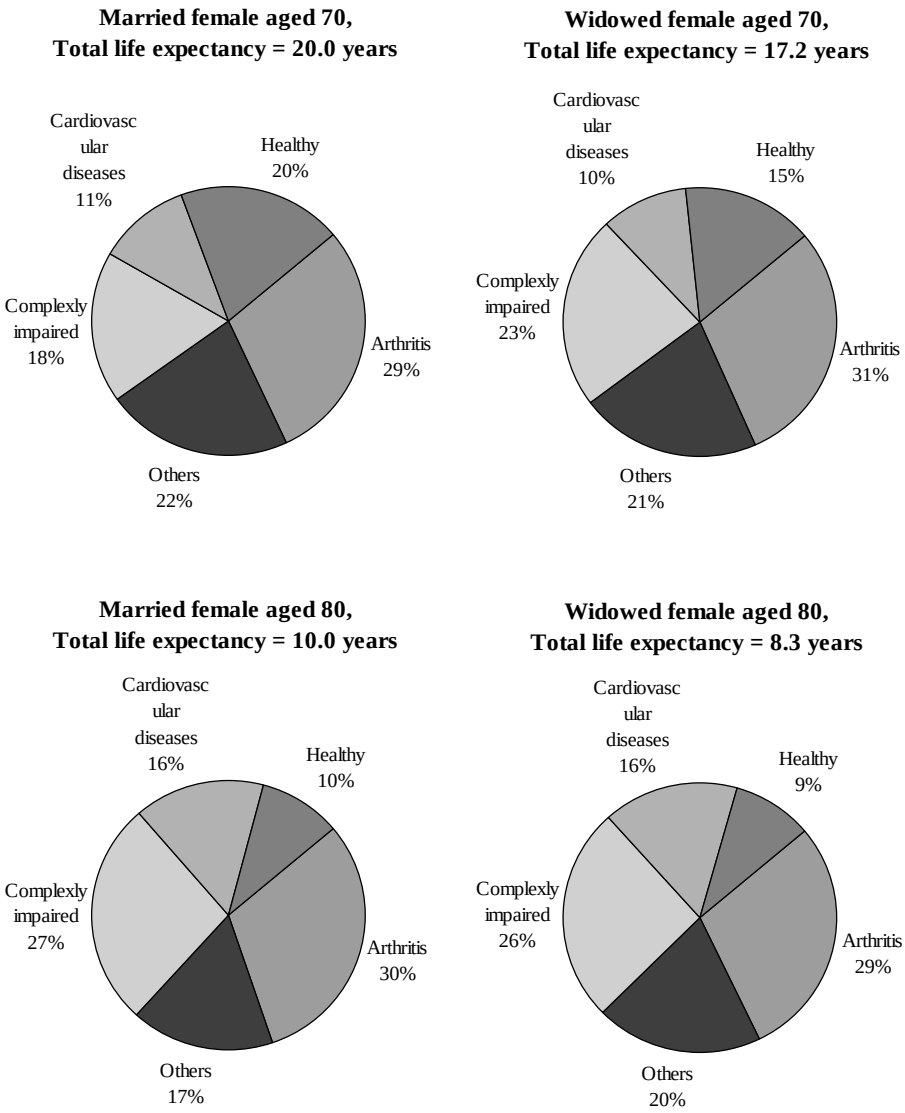


Figure 8: Health expectancies for older females



ing life threatening diseases later in life and therefore later life mortality. The death of the partner significantly increases the probability of suffering from arthritis/diabetes. This effect is not permanent, but can last up to 12 years. However, as in the mortality model, the direct effects of bereavement are stronger than the longer run effects; bereavement affects the probability of arthritis/diabetes most strongly in the first few years just after the death of the partner. We find no direct effects of bereavement on other health disorders. Also indirect effects (via arthritis/diabetes) of bereavement on other health disorders are small.

We used the model to calculate residual life and health expectancies differentiated per marital status. We find that males and females lose on average 11.5 % and 12.5 % respectively of their residual life expectancy after bereavement. Most of the effect takes place in the first years of bereavement. We also calculated health expectancies, i.e. residual life time spend in specific health states. We find that bereavement affects the share of healthy years in residual lifetime, primarily because healthy years are replaced by years of having serious arthritis and diabetes and of being complexly impaired. In absolute terms, however, bereavement reduces time spent in unhealthy states. This is caused by the strong direct effect of bereavement on mortality. The reduction in life years due to bereavement are stronger than the effects of bereavement on health.

The strong direct effects of bereavement are important for policies aimed at the elderly. It suggests that monitoring and/or interventions just after spousal bereavement are important for the length of remaining life and for the physical and mental health-related quality of life of older bereaved persons.

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Appendix A

Table A1: GoM Parameters λ_{kjl}, wave I

	Score	Freq.	K=1	K=2	K=3	K=4	K=5
Self-reported test	0	0.591	1	0.612	0.358	0.367	0.433
on mobility	1	0.191	0	0.246	0.516	0.289	0.086
	2	0.115	0	0.142	0.126	0.344	0.047
	3	0.103	0	0	0	0	0.434
Performance test	0	0.545	0.642	0.600	0.481	0.565	0.285
(cardigan)	1	0.435	0.358	0.400	0.519	0.435	0.643
	2	0.020	0	0	0	0	0.072
MMSE	≥ 23	0.900	1	1	1	1	0.604
	< 23	0.100	0	0	0	0	0.396
CES-D	≤ 16	0.855	1	0.847	0.851	0.849	0.732
	> 16	0.145	0	0.153	0.149	0.151	0.268
Vision	Good	0.885	1	0.864	0.874	0.910	0.786
	Bad	0.115	0	0.136	0.126	0.090	0.214
Hearing	Good	0.940	1	1	1	1	0.770
	Bad	0.060	0	0	0	0	0.230
COPD	N	0.884	1	1	1	1	0.540
	Y	0.116	0	0	0	0	0.460
Medical treatment	N	0.915	1	1	1	1	0.672
(COPD)	Y	0.085	0	0	0	0	0.328
Heart diseases	N	0.804	1	1	0	1	1
	Y	0.196	0	0	1	0	0
Medical treatment	N	0.826	1	1	0.111	1	1
(Heart diseases)	Y	0.174	0	0	0.889	0	0
Atherosclerosis	N	0.902	1	1	0.591	1	1
	Y	0.098	0	0	0.409	0	0
Medical treatment	N	0.930	1	1	0.712	1	1
(Atherosclerosis)	Y	0.070	0	0	0.288	0	0
Diabetes	N	0.922	1	1	1	0.693	1
	Y	0.078	0	0	0	0.307	0
Medical treatment	N	0.926	1	1	1	0.709	1
(Diabetes)	Y	0.074	0	0	0	0.291	0
Stroke	N	0.944	1	1	1	1	0.780
	Y	0.056	0	0	0	0	0.220
Medical treatment	N	0.958	1	1	1	1	0.842
(Stroke)	Y	0.042	0	0	0	0	0.158
Arthritis	N	0.651	1	0.700	0.825	0	0.689
	Y	0.349	0	0.300	0.175	1	0.311
Medical treatment	N	0.850	1	1	1	0.336	1
(Arthritis)	Y	0.150	0	0	0	0.664	0
Cancer	N	0.907	1	1	1	1	0.650
	Y	0.093	0	0	0	0	0.350
Medical treatment	N	0.939	1	1	1	1	0.775
(Cancer)	Y	0.061	0	0	0	0	0.225
Other chronic diseases	N	0.667	1	0	0.966	1	0.887
	Y	0.333	0	1	0.034	0	0.113
Medical treatment	N	0.767	1	0	1	1	1
(Other chronic diseases)	Y	0.233	0	1	0	0	0

The third column of Table A1 reports sample proportions of various health disorders. The characteristics of the different health dimensions are determined by examination of the K profile probabilities λ_{kjl} and their comparison to the sample proportions of interest.

Appendix B

Table 2b: Results of Model I (second part): mortality of spouse and initial conditions

Variables	Coeff.	t-value	Variables	Coeff.	t-value
Mortality of spouses			Baseline hazard for spouses		
Female	-0.890	-6.91	γ_1 (32/70)	0.008	3.32
Education	-0.110	-3.48	γ_2 (70/74)	0.028	3.19
Occupational level	0.020	0.38	γ_3 (74/78)	0.072	3.28
Urbanization	-0.038	-1.94	γ_4 (78/82)	0.207	3.25
Church attendance	-0.044	-1.40	γ_5 (82/86)	0.406	3.15
			γ_6 (86/95)	0.424	2.74
Health equation (Initial conditions)					
Other chronic diseases			Cardiovascular diseases		
Constant	-1.129	-4.16	Constant	-0.321	-1.57
Age	0.491	0.65	Age	1.358	2.29
Age^2	-0.647	-0.69	Age^2	-0.539	-0.76
Female	0.696	3.63	Female	-0.810	-5.48
Education	0.172	0.81	Education	-0.229	-1.46
Urbanization	-0.102	-0.67	Urbanization	0.061	0.53
Church attendance	0.018	0.15	Church attendance	-0.076	-0.80
σ_1	1.643	8.18	σ_2	0.890	9.11
Arthritis/Diabetes			Complexly impaired		
Constant	-0.965	-5.84	Constant	-0.353	-2.85
Age	0.816	1.84	Age	0.899	2.47
Age^2	-0.387	-0.72	Age^2	-0.001	-0.00
Female	0.645	5.59	Female	0.008	0.09
Education	-0.266	-2.12	Education	-0.335	-3.39
Urbanization	-0.040	-0.44	Urbanization	0.024	0.33
Church attendance	0.028	0.38	Church attendance	-0.246	-3.86
σ_3	0.239	16.830	σ_4	0.166	16.74
One-factor error specification					
l_2	-0.133	-0.64			
l_3	0.205	0.62			
l_4	-1.630	-2.20			

Appendix C

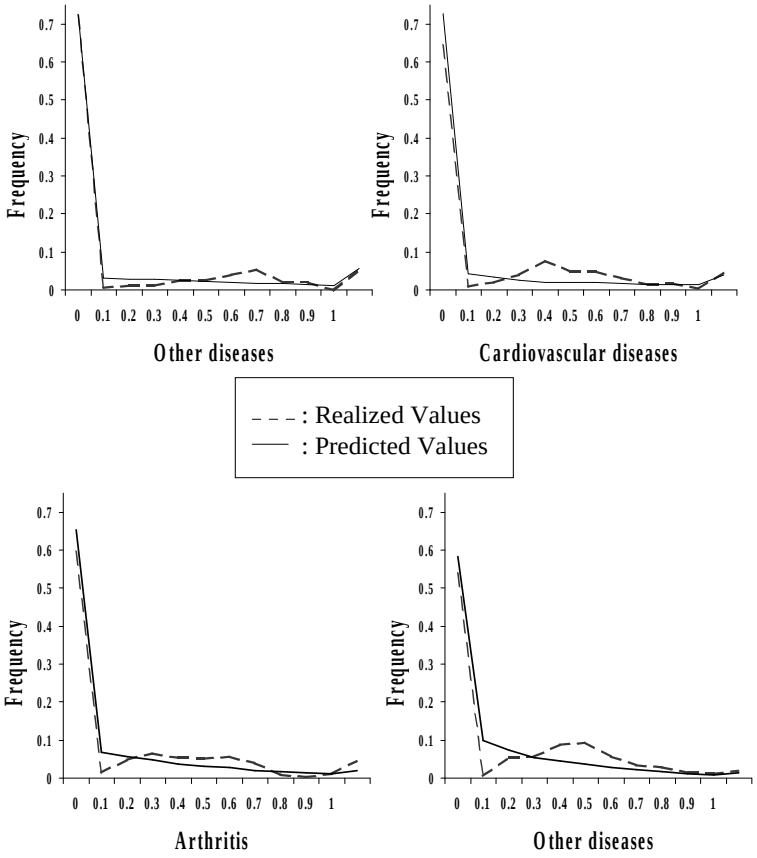
Table 2c: Model 2: Mortality of head and spouse and a static health model of the head

Variables	Coeff.	t-value	Variables (Age classes)	Coeff.	t-value
Mortality of LASA respondents			Baseline hazard for LASA respondents		
Female	-0.482	-4.50	γ_1 (55/62)	0.005	2.74
Education	0.018	0.77	γ_2 (62/66)	0.010	3.08
Income	-1.057	-4.87	γ_3 (66/70)	0.016	3.29
Urbanization	0.026	1.76	γ_4 (70/74)	0.030	3.57
Church attendance	-0.112	-4.62	γ_5 (74/78)	0.063	3.72
TSB (short run)	-0.330	-1.38	γ_6 (78/82)	0.121	3.80
Quadratic TSB (short run)	0.319	4.76	γ_7 (82/85)	0.303	3.81
TSB (long run)	0.797	3.72	γ_8 (86/92)	0.454	3.45
Quadratic TSB (long run)	-0.130	-2.61			
Dummy refusals	-1.358	-10.78			
Other Chronic diseases	0.444	2.40			
Cardiovascular diseases	0.650	3.74			
Arthritis/Diabetes	-0.043	-0.21			
Complexly impaired	1.275	7.23			
Mortality of spouses			Baseline hazard for spouses		
Female	-0.984	-8.15	γ_1 (32/70)	0.017	3.47
Education	-0.110	-3.81	γ_2 (70/74)	0.051	3.36
Occupational level	0.033	0.70	γ_3 (74/78)	0.110	3.41
Urbanization	-0.040	-2.17	γ_4 (78/82)	0.265	3.42
Church attendance	-0.059	-2.03	γ_5 (82/86)	0.478	3.39
			γ_6 (86/95)	0.547	3.00
Health model					
Other Chronic diseases			Cardiovascular diseases		
Constant	-0.860	-6.24	Constant	-0.141	-1.79
Age	-0.057	-0.15	Age	1.199	5.11
Age^2	-0.069	-0.16	Age^2	-0.542	-2.17
Female	0.516	5.72	Female	-0.647	-13.70
Education	0.172	1.49	Education	-0.130	-2.17
Income	-0.016	-0.14	Income	-0.137	-2.93
Church attendance	-0.045	-0.76	Church attendance	-0.122	-3.83
Urbanization	-0.177	-2.40	Urbanization	0.010	0.27
TSB (short run)	0.008	0.16	TSB (short run)	-0.002	-0.02
TSB (long run)	0.019	0.80	TSB (long run)	-0.061	-1.44
σ_1	1.305	14.14	σ_2	0.425	19.80
Arthritis/Diabetes			Complexly impaired		
Constant	-0.933	-12.75	Constant	-0.218	-3.77
Age	0.972	4.91	Age	0.957	5.63
Age^2	-0.603	-2.83	Age^2	-0.118	-0.65
Female	0.643	13.98	Female	-0.050	-1.40
Education	-0.049	-0.86	Education	-0.238	-5.17
Income	-0.137	-2.50	Income	-0.137	-2.93
Urbanization	0.031	0.87	Urbanization	-0.024	-0.82
Church attendance	0.081	2.82	Church attendance	-0.104	-4.08
TSB (short run)	0.094	2.31	TSB (short run)	-0.002	-0.08
TSB (long run)	0.036	1.74	TSB (long run)	0.006	0.37
σ_3	0.379	22.22	σ_4	0.263	20.94

Note: TSB = Time since bereavement

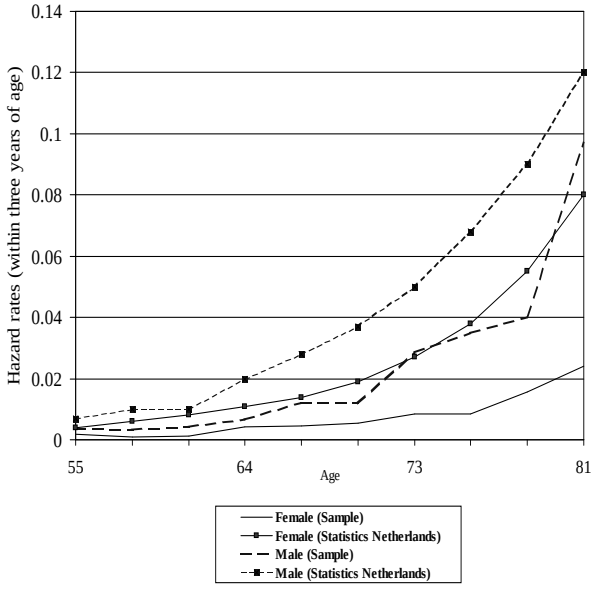
Appendix D

Figure 9: Informal check dynamic health model



Appendix E

Figure 10: Sample and Statistics-Netherlands hazard rates



Statistics Netherlands calculate "average residual life expectancies" – i.e. residual lifetimes for individuals with average health status, socioeconomic characteristics and marital status. Our sample is not representative of the Dutch older population as we selected married individuals at baseline. Figure 10 shows hazard rate from our model (estimated on the sample of married individuals) and hazard rates from the Statistics Netherlands. It is clear that our model estimates are lower than the estimates from Statistics Netherlands. We further compared the averages of the explanatory variables of our (married) sample with the population averages. On average, the sample respondents are younger, higher educated, have higher incomes, and go more often to church. These factors are found to be associated (in our model and in the literature) with lower mortality and morbidity.