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WHAT AGE TRAJECTORIES OF CUMULATIVE DEFICITS AND MEDICAL COSTS TELL US ABOUT INDIVIDUAL AGING AND MORTALITY RISK: FINDINGS FROM THE NLTCs-MEDICARE DATA

Anatoli I. Yashin^{*}, Konstantin G. Arbeev, Alexander Kulminski, Igor Akushevich, Lucy Akushevich, and Svetlana V. Ukraintseva

Center for Population Health and Aging, Duke University 002 Trent Hall, Box 90408, Durham, NC, 27708-0408, USA

Abstract

An important feature of aging-related deterioration in human health is the decline in organisms' resistance to stresses, which contributes to an increase in morbidity and mortality risks. In human longitudinal studies of aging, such a decline is not measured directly, so indirect methods of statistical modeling have to be used for evaluating this characteristic. Since medical interventions reflect severity of occurring health disorders, data from Medicare service use files can be used for such modeling. In this paper, we use the National Long Term Care Survey (NLTCs) data merged with the Medicare service use files to investigate dynamics of stress resistance in the U.S. elderly. We constructed individual indices of cumulative deficits and medical costs and investigated their separate and joint effects on dynamics of mortality risks using the quadratic hazard model. We found that males show a faster decline in stress resistance with age than females.

Keywords

cumulative deficits; medical costs; mortality; quadratic hazard model; stress resistance

1. Introduction

The resilience property of human organism is clearly manifested during its aging process, when the occurrence of aging-related health disorders is accompanied by the internal processes of compensatory adaptation and remodeling (Franceschi et al., 2000; Schachter, 2000). These processes contribute to organism's recovery and support its functioning, limited by the new conditions. The occurrence of health disorders also activates the external loop of regulating individuals' health status. This regulation is based on the use of the health care facilities and is manifested in outpatient visits, hospitalizations, as well as the use of many other services provided by the modern health care systems. Although the use of these services does not stop the aging-related decline in health and well-being of individuals comprising human population,

^{*}Corresponding author: Anatoli I. Yashin Research Professor Center for Population Health and Aging Duke University 002 Trent Hall, Box 90408 Durham, NC, 27708-0408, USA Tel. (+1) 919-668-2713 Fax: (+1) 919-684-3861 E-mail: aiy@duke.edu.

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it is supposed to work in concordance with the internal forces maintaining resilience, i.e., it contributes to the rate of recovery, slowing down, or postponing the development of morbid/co-morbid pathological processes, diminish mortality risks, etc.

In developed countries with well functioning health care services, the contributions of individual defense mechanisms and external health-regulating systems to individual health/well-being/survival status is difficult to separate. This is because any severe health disorder initiates responses of both health-regulating mechanisms. Therefore, the joint analysis of the system of indices which captures the features of the internal and external health-regulating machinery is an important step towards better understanding of regularities of aging-related decline in human health/well-being/survival status and opportunities for increasing healthy life span using medical interventions.

The National Long Term Care Survey (NLTCS) data merged with the Medicare service use files (NLTCS-Medicare) create unique opportunity to study individual dynamics of health/well-being deterioration in Medicare beneficiaries. These data allow for constructing the index of cumulative deficits, DI, (also called the frailty index) which seems to be a useful indicator of health/well-being deterioration (Kulminski et al., 2007a; 2006; 2007b; Mitnitski and Rockwood, 2006; Mitnitski et al., 2004; 2001; Rockwood et al., 2006; 2004). The properties of such index constructed from the NLTCS data were studied by Kulminski et al. (2007a; 2006; 2007b) and Yashin et al. (2007b; 2007c). The results of these studies showed that the DI is a convenient index describing aging-related health/well-being deterioration in the U.S. elderly. The information about the role of health care system in this process is collected in the Medicare service use files available for each NLTCS participant.

One of the key indices characterizing the severity of health/well-being disorder and the intensity of the Medicare services use is the medical costs. A detailed and comprehensive analysis has been performed to investigate aggregate spending on the Medicare Part A and B programs for specific periods of time for the U.S. elderly population during the last years of life (Hogan et al., 2001; Hoover et al., 2002). Studies of the association between the mortality risk and the end-of-life medical spending show that more than a half of Medicare spending in the last year of life is for beneficiaries with a low demographic mortality risk (Howard et al., 2006). A comprehensive investigation of Medicare costs and their relationship to disability and morbidity was recently presented by Goldman and colleagues (Bhattacharya et al., 2004; Goldman et al., 2005). A number of studies investigated medical costs associated with particular health conditions (Brown et al., 2002; Husaini et al., 2002). The impact of such behavioral factors as physical activity and bodyweight on health care utilization and costs was studied by Wang et al. (2005). These studies, however, investigate neither dynamic properties of the individual trajectories of medical costs, nor their connection with individual health/well-being/survival histories.

In this paper we investigate properties of the DI and an index describing medical costs (MCI), and their dynamic effects on the mortality risk using the NLTCS-Medicare data. We performed separate and joint analyses of these indices and their effects paying special attention to changes in the shape of the U-curve of the mortality risk with age. We use the width of the U-shaped risk function as a measure of resistance to stress associated with a factor specifying such risk. In these analyses, we used three versions of the stochastic process model of human mortality and aging (Yashin et al., 2007a; 2007b; 2007c).

2. Materials and Methods

2.1. The Index of Cumulative Deficit (DI)

To make the results of this study comparable with those obtained in (Yashin et al., 2007b; 2007c), we constructed the DI using the same subset of deficits (32 questions from the NLTCS detailed questionnaires). This subset is mostly similar to those assessed from the Canadian Study of Health and Aging (Mitnitski et al., 2001). It includes: difficulty with eating, dressing, walking around, getting in/out bed, getting bath, toileting, using telephone, going out, shopping, cooking, light house work, taking medicine, managing money, arthritis, Parkinson's disease, glaucoma, diabetes, stomach problem, history of heart attack, hypertension, history of stroke, flu, broken hip, broken bones, trouble with bladder/bowels, dementia, self-rated health, as well as problems with vision, hearing, ear, teeth, and feet. As in early works (Yashin et al., 2007b; 2007c), all these deficits are assessed in five waves of the NLTCS. Following Mitnitski et al. (Mitnitski et al., 2001), we defined the DI as an unweighted count of the number of such deficits divided by the total number of all potential deficits considered for a given person. In this way, we avoid the problem of missing answers counting only those questions explicitly answered in a survey.

2.2. The Index of Medical Costs (MCI)

The Index of Medical Costs (MCI) is defined from Medicare service use files linked to the NLTCS data. Medicare is the federally funded program that provides health insurance for the elderly, persons with end-stage renal disease, and some disabled individuals. In general, individuals are eligible for Medicare if they (or their spouse) worked for at least 10 years in Medicare-covered employment and are at least 65 years old and are citizens or permanent residents of the United States of America. For persons age 65 and over, 97 percent are eligible for Medicare. Almost all Medicare beneficiaries have Part A coverage that includes hospital, skilled-nursing facility, hospice and some home health care. 96 percent of elderly Part A beneficiaries choose to pay a monthly premium to enroll in Part B of Medicare that covers physician and outpatient services. While some Medicare beneficiaries are enrolled in Health Maintenance Organizations (HMOs), most have fee-for-service (FFS) coverage. The proportion of the sample in an HMO ranged from 6.2% in 1991 to 17.6% in 1999 after peaking at approximately 18% in 1997. Health care utilization information for such persons is not represented in Medicare claims files for the period during which they are enrolled in a Medicare HMO. The literature offers mixed opinions whether patients in HMOs are indeed healthier; the prevalent findings are that enrollees in Medicare HMOs tend to be healthier, although no such conclusion is supported in the private insurance sector (Sloan et al., 2003). Taylor et al. (2004) showed that excluding such persons did not alter the basic findings of their calculated rates of persons identified as having Alzheimer's disease as of December 31 of each study year (1991–1999). Information about Medicare eligibility and enrollment is available for all Medicare beneficiaries.

Age trajectory of medical costs is constructed for each individual, for whom the age trajectory of the DI is constructed. The MCI is based on month-by-month estimates of Medicare costs. The total expenditures employed for definition of the MCI at age x were calculated by summing costs for all time periods with the individual's age in the range $[x, x + 1)$. The annual costs include expenditures from all sources recorded in Medicare service use files. These sources are structured in seven different categories: inpatient (INP), outpatient (OTP), skilled nursing facility (SNF), hospice (HSP), home health agency (HHA), carrier/physician/supplier (PTB), and durable medical equipment (DME).

These sources differ by the type of service and by service providers. All facility costs for patients admitted for at least overnight stay in a hospital or in other health facility for the

purpose of receiving diagnosis, treatment, or another health service, are included in INP. Facility costs for inpatients with chronic diseases without acute conditions or costs for short-term rehabilitative residents are included in SNF. Costs for service receiving during one day in a hospital or clinic without overnight stay are included in OTP. HHA contains claims data submitted by HHA providers for skilled-nursing care, home health aides, physical therapy, speech therapy, occupational therapy, and medical social services. PTB claims data are submitted by non-institutional providers. Examples of non-institutional providers include physicians, physician assistants, clinical social workers, nurse practitioners, independent clinical laboratories, ambulance providers, and stand-alone ambulatory surgical centers. HSP contains claims data submitted by hospice providers for inpatient, outpatient, and home healthcare for terminally ill patients. DME contains claims for durable medical equipment or special equipment ordered by a doctor, usually for using at home.

2.3. Methods

We describe the individual trajectories of the DI and MCI using a two-dimensional stochastic process Y_t satisfying a diffusion type stochastic differential equation with components describing regular and stochastic changes of these indices with age (see Appendix). In constructing the hazard rate, we took into account evidences that the risks of death considered as functions of covariates are usually U- or J-shaped (Witteman et al., 1994). We use a symmetric quadratic hazard, which captures the effect of interaction between indices on mortality risks:

$$\mu(t, Y_0^t) = \mu_t^0 + (f_t - Y_t)^* \mu_t^1 (f_t - Y_t), \quad (1)$$

where μ_t^1 is a non-negative-definite symmetric matrix $\mu_t^1 = \begin{pmatrix} \mu_t^{11} & \mu_t^{12} \\ \mu_t^{12} & \mu_t^{22} \end{pmatrix}$. The two-dimensional function f_t characterizes the ‘optimal’ trajectories of age-dependent covariates (DI and MCI), i.e., the trajectories, for which the mortality risk is minimal. It seems at the first glance that there is no need to introduce such a function for the MCI, since its ‘optimal’ value is always likely to be zero. However, our earlier studies of the DI revealed that f_t may differ from zero for both males and females. This may characterize behavioral adaptation of those who already have some disorders, or trade-off between chronic conditions. In (Yashin et al., 2007b; 2007c) we discuss possible causes of non-zero f_t . For this reason, we consider a general case with non-zero f_t for the MCI as well.

We also hypothesize that not only the minimum value but also the shape of this U-function may change when individuals get older. This feature is captured by the age-dependence of functions μ_t^{11} , μ_t^{12} and μ_t^{22} in (1). The baseline hazard μ_t^0 was estimated as a Gompertz or a logistic function. The details of respective models are described in Appendix.

3. Results

The graphs for the mean values, standard deviations, and correlation coefficients (with 95% confidence intervals) of the DI and MCI are shown in Fig. 1. One can see from this figure that the mean value of the DI increases with age for both sexes (left-top panel). The female DI tends to be higher and increase faster than that of males. The mean values of MCI for males and females are also increasing functions of age (right-top panel). The MCI for males tends to increase faster than that of females. Standard deviations of the DI for females tend to be smaller than those for males at each age (middle left panel). They increase between ages 73 and 92 years for both sexes. However, this increase has a tendency to decelerate with age. The standard deviations for the MCI do not show a regular pattern for both sexes (right-middle panel). The correlation between the DI and MCI is positive and significantly different from zero. The age

dependence of correlation coefficient between the DI and MCI is weak. Taking into account large 95% confidence intervals, it may be considered constant, 0.18 (lower panel).

The quadratic hazard model (QHM) has an advantage of being consistent with the traditional mortality models used by demographers and epidemiologists. Specifically, averaging the QHM with respect to the MCI, we will have the age pattern of the total mortality for the elderly described by standard demographic life tables. Estimating the QHM conditional on the MCI, or on the DI and MCI, we evaluate dynamic contribution of these indices into the risk of death. The important property of the QHM is that it allows for estimation of the age pattern of the baseline mortality μ_t^0 when $Y_t = f_t$, i.e., when individuals follow an optimal trajectories of the DI and MCI.

The total mortality rates $\bar{\mu}_t$ in the NLTCs data approximated by the Gompertz function $(\bar{\mu}_t = ae^{b(t-t_{\min})}, t_{\min}=65)$ and the baseline hazards μ_t^0 in the QHM applied to these data are shown in Fig. 2. One can see from this figure that controlling for the MCI and/or DI (keeping at the optimal level $Y_t = f_t$) would result in substantial reduction in the mortality rates for both sexes after age 65. This reduction gives rise to an increase in residual life expectancies after age 65 up to 13.8 and 11.6 years for females and males respectively, compared to the residual life expectancies without such a control. Note that controlling for both MCI and DI gives a larger increase in life expectancy compared to the situation when only one index is controlled (see Table 1, column “Gain LE₆₅”).

Fig. 3 shows age trajectories of coefficients characterizing the quadratic hazard for males and females. One can see from this figure that the baseline hazard μ_t^0 is higher for males than for females for all ages over 65 (left-top panel). However, the slope of the logarithm of this hazard is lower for males. The component μ_t^{11} of the quadratic hazard for the DI increases with age for both sexes (right-top panel). This increase indicates that vulnerability to deviations from the norm increases with age (i.e., resistance to stresses associated with the DI declines with age) for both genders. This increase is faster for males than for females. The coefficient μ_t^{12} characterizing effects of the DI-MCI interaction on the mortality risk shows opposite patterns of age dependence for males and females. This effect increases in males and tends to decline in females (left-low panel). The effect of the MCI on the mortality risk μ_t^{22} increases with age for males and declines for females (right-low panel).

Fig. 4 shows age patterns of the functions f_t^1 (see description in Appendix) and the ‘optimal’ trajectories f_t for the two indices. One can see from this figure that for both the DI and MCI the function f_t^1 increases with age for both sexes. For the DI, this function increases faster for females than for males (left-top panel). However, for the MCI, the function f_t^1 increases faster for males than for females (right-top panel). The optimal values of the DI are about zero for males and are slightly above zero and tend to decline with age for females (left-bottom panel). The optimal values of MCI are about zero for both sexes, compared to average values of these indices (right-bottom panel).

The contour maps of the quadratic term of the hazard rate are shown in Fig. 5 and Fig. 6 for females and males respectively. One can see from Fig. 5 that the shape of the quadratic component of the hazard rate for females tends to be more symmetric with age. This is likely to be because of decline in the interaction term μ_t^{12} for females. Fig. 6 shows that in both dimensions (i.e., DI and MCI), the mortality risk increases faster with age for males.

The results of analyses of data using three versions of the quadratic hazard model are shown in Table 1. These versions include: Model 1 which describes joint evolution of the two indices DI and MCI, both affecting the mortality risk, with the interaction terms in the stochastic differential equation and in the quadratic hazard; Model 2, which describes the evolution of two indices, both affecting the mortality risk, without interaction terms; and a one-dimensional Model 3, which describes evolution of each of the indices and their effects on mortality risks separately. As Models 1–3 are not nested (Model 3 estimates DI and MCI data independently and thus has different estimates of baseline hazards for DI and MCI), we used the Akaike information criterion (AIC, Akaike, 1974) to compare all three models. The column “AIC” in Table 1 reports differences between the AIC statistics. Application of AIC to compare all three models allows us to conclude that the model with interaction term (Model 1) gives the best fit to the data for males and females. One can also see that the coefficients of the equations describing the joint age dynamics of the DI and MCI differ for males and females. This may indicate different effects of medical care regulation mechanism on health/well-being status in males and females.

The negative values of feedback coefficients a_v^{12} and a_v^{21} (evaluated in females) indicate the presence of mechanism contributing to mutual stabilization of the health status and medical costs. Such a mechanism is likely to work in cases when health declining symptoms are addressed at the early stage of the disorder development. The fact that such negative values are observed in females but not in males confirms earlier observations that females take more care of their health than males.

4. Discussion

The origin of the health care is based on the idea to provide help to individual biological and physiological health/well-being regulating mechanisms in restoring proper functioning after an occurred health disorder, or to diminish chances of such disorders by preventive measures. Although the development of the health care industry followed some basic economic principles, its applications have an important dimension related to maintenance of human health, well-being, and survival, working in concordance with appropriate internal biological and physiological forces. Economical measure of the health care efforts convenient for our studies is medical spending. In a proper model describing the joint dynamics of public health in connection with health economics, one would be able to evaluate contribution of the health care system to public health/well-being/survival by comparing difference in morbidity or mortality risks in the hypothetical case of zero medical spending with currently observed risks. It is expected that nullifying medical spending will substantially increase morbidity and mortality risk, and an increase in medical spending will reduce such risks.

In current statistical models connecting health status and medical costs, the opposite effect is observed: the index of medical costs appears to be a risk factor for morbidity and mortality. The explanation of this paradoxical effect is simple: in the absence of other observed covariates, the index of medical costs characterizes the severity of a disease or unhealthy state, which is a risk factor for mortality. This means that the MCI may characterize contribution of the medical care to population's health only in the presence of observed covariates capturing the severity of unhealthy conditions. The fact that addition of the DI to the MCI in the hazard model reduces the mortality risk from medical costs indicates that such explanation has realistic background. It turns out, however, that controlling for covariates describing individuals' health/well-being status is not enough to automatically capture the contribution of medical system to the population's health. An additional property of the data on medical costs is required to make such evaluation possible. The data have to contain information on health/well-being/survival status of individuals for whom medical help was not provided in a full scale when it

was needed. One expects that such inadequate medical help will shorten healthy life span and total life span.

In an attempt to separate different roles of the MCI, we performed the analyses by considering both separate and joint effects of the MCI and DI on the mortality risk. Our expectations were that the joint analyses will at least reduce the effect of MCI as a risk factor for mortality. In the ideal situation when a hypothetical health/well-being index captures all effects of disorders, and MCI captures effects of the quality of disorders' treatment, one has the possibility to capture favorable contribution the MCI to the mortality risk. As we mentioned earlier, the data on incomplete health care coverage could help in evaluating positive effects of medical spending, i.e., their effects on reduction of mortality risk.

The difference between “optimal” age trajectories of the DI for males and females confirms difference in the perception of risks in the two genders. The “optimal” trajectories for MCI are, however, close to zero for both sexes. To explain this difference, we note that the two indices have different construction and are formed from two different subsets of data on the same individuals. The DI was constructed from the responses to the NLTCS questionnaire. By construction, each deficit contributes to the DI with the same weight. The MCI was constructed using claims data from the Medicare service use files. Each health disorder (reflected in these files) contributes to the MCI with its own weight (e.g., the cost of respective treatment). The individual may use a small number of deficits happened to him/her as a signal to modify behavior aiming at reducing the mortality risk. It is quite likely that the proportion of acquired deficits, which resulted in reducing the behavioral risk to its minimum value, is visible at the DI scale (which is between 0 and 1). However, the costs of such “signaling” deficits may be zero (when deficits are not reflected in the Medicare files) or small (compared to the costs associated with treatment of major health disorders measured in thousands of dollars). This explains why the optimal curves for MCI are close to zero, when respective DI curves are not. This also shows the difference between the two indices.

An important finding of this paper is the difference in the decline in resistance to stresses in males and females (see Figs. 5 and 6). The narrowing of the two-dimensional U-function of mortality risks for males is getting faster, and involves both components (the DI and MCI) of risk factor, than that of females, where such narrowing involves only the DI component and is going slower than in males. The faster aging-related decline in stress resistance in males than in females is consistent with our earlier findings (Yashin et al., 2007b;2007c), where the properties of the DI index and its effects on mortality risks were investigated. The fact that the use of additional dimension did not change this result indicates that the difference in biological background mediated by social and behavioral conditions is likely to be responsible for the difference in the rates of change in stress resistance in males and females, which ultimately results in the difference in the mortality rates. This involves the evolutionary based difference in the perception of risks shaped by sexual selection. This trait interacts with multiple aspects of culture and environment to yield a difference in male-female mortality patterns that has some consistency across decades and societies. Numerous studies show that males are more prone to risk-taking behavior than females (Byrnes et al., 1999; Kruger and Nesse, 2004), so a faster rate of decline in stress resistance in males may be associated with the faster spending of a certain biological resource associated with stress resistance. This hypothesis has been recently supported by analyses of data on cancer incidence rates for females and males in different countries and time periods (Arbeev et al., 2005). Applying the extended Strehler and Mildvan's model of aging (Strehler and Mildvan, 1960) to data on human cancer incidence, the authors revealed a stable relationship between the rate of decline in the “vitality” function in females and males. This rate, which is the aging-related rate of decline in the amount of some biological resource associated with stress resistance, was higher in males in all analyzed countries and time periods.

Earlier (Yashin et al., 2001) we proposed that some individuals who are more vulnerable to stresses at young or middle ages may then change from the relative vulnerability to the relative robustness and get a survival advantage later in life. There may be several reasons for such a change (some are discussed in (Yashin et al., 2001)). The following example shows how this hypothesis can be applied to explain differences in stress resistance among elderly males and females in our study. Females are generally more sensitive to environmental fluctuations (e.g., of outside temperature or atmospheric pressure) than males when they are young adults and their cardiovascular system manifests greater variability in respective parameters in response to stress. As a result, this system may be trained more intensively during the life course than that of initially more robust males, who, oppositely, display less variability in respective physiological parameters in response to stress. However, initially more vulnerable/labile females may improve the quality of their response to stress later in life compared with initially more robust/rigid males because of overall better-trained vascular system of the former. Such a change in the relative stress resistance may give females a survival advantage at older ages. In this scenario, some heavy stresses at the oldest old ages may be more fatal for males than for females.

In this study, we did not intend to investigate effects of additional risk factors on mortality risks. Studying contribution of social-economic status, and other variables characterizing lifestyle, environmental and living conditions could help to better conceptualize vulnerability to death and other adverse health outcomes (Fisher, 2005). In fact, the approach described above, has potential for such an extension which, however, requires separate analyses.

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Appendix

General model

Let $X_t, Y_t^i, i = 1 \dots J$, be stochastic processes describing the life history of an individual. The process X_{t_k} equals zero if an individual died in the interval $[t_k, t_{k+1})$, and it equals one if he/she survived until age t_{k+1} . The process Y_t^i is a discrete time stochastic process describing observations of i^{th} health-related index (covariate). Assume that the vector process $Y_t = Y_t^1 \dots Y_t^J$ satisfies the following equation:

$$Y_{t_{k+1}} = Y_{t_k} + a_{t_k} (f_{t_k}^1 - Y_{t_k}) (t_{k+1} - t_k) + \sigma_1 \sqrt{t_{k+1} - t_k} \varepsilon_{t_k}, \quad k > 1, \quad Y_{t_1}, \quad (\text{A1})$$

where a_{t_k} is a $J \times J$ matrix, $f_{t_k}^1$ is a vector-function, $\varepsilon_{t_k}^i \sim N(0, 1), Y_{t_1} \sim N(f_{t_1}^1, \sigma_0^2), \sigma_0^2$ and σ_1 are diagonal matrices. Note that, in principle, ages of observation of the processes Y_t^i may do not coincide for different indices. In such cases, we denote by t_k the ages where at least one of the processes Y_t^i is observed and the unobserved at t_k processes Y_t^i are fixed to their last observed value.

Let $\tilde{Y}_{t_k}^{t_k}, k = 1 \dots n$ be a random vector of observations of the process Y_t at ages $t_1, \dots, t_k$. Denote by $\mathcal{Q} \left(t_k, \tilde{Y}_{t_k}^{t_k} \right)$ the conditional probability of death in the interval $[t_k, t_{k+1})$ of an individual given

an observed trajectory $\tilde{Y}_{t_1}^{t_k}$, i.e., $Q(t_k, \tilde{Y}_{t_1}^{t_k}) = P(X_{t_{k+1}}=0 | \tilde{Y}_{t_1}^{t_k}, X_{t_k}=1)$. Assume that this probability depends only on values of $\tilde{Y}_{t_1}^{t_k}$ as follows:

$$Q(t_k, \tilde{Y}_{t_1}^{t_k}) = 1 - e^{-\mu(t_k, \tilde{Y}_{t_1}^{t_k})(t_{k+1}-t_k)}, \quad (\text{A2})$$

with

$$\mu(t_k, \tilde{Y}_{t_1}^{t_k}) = \mu_{t_k}^0 + (f_k - Y_{t_k})^* \mu_{t_k}^1 (f_k - Y_{t_k}), \quad (\text{A3})$$

where $\mu_{t_k}^1$ is a non-negative-definite symmetric matrix.

For the likelihood function, we need conditional distributions of $\tilde{Y}_{t_1}^{t_k}$ given the observations

$\tilde{Y}_{t_1}^{t_{k-1}}$. From (A1),

$$p(Y_{t_k} | \tilde{Y}_{t_1}^{t_{k-1}}) = \frac{1}{(2\pi)^{\frac{1}{2}} \sqrt{\det((t_k - t_{k-1})\sigma_1)}} e^{-\frac{1}{2}(Y_{t_k} - \bar{Y}_{t_{k-1}})^* ((t_k - t_{k-1})\sigma_1)^{-1} (Y_{t_k} - \bar{Y}_{t_{k-1}})}, \quad (\text{A4})$$

where

$$\bar{Y}_{t_{k-1}} = Y_{t_{k-1}} + a_{t_{k-1}} (f_{t_{k-1}}^1 - Y_{t_{k-1}})(t_k - t_{k-1}), \quad (\text{A5})$$

for $k > 2$, and

$$p(Y_{t_1}) = \frac{1}{(2\pi)^{\frac{1}{2}} \sqrt{\det(\sigma_0)}} e^{-\frac{1}{2}(Y_{t_1} - f_{t_1}^1)^* \sigma_0^{-1} (Y_{t_1} - f_{t_1}^1)}. \quad (\text{A6})$$

Consider N independent observations of individuals in the above described scheme. Denote

by $\tilde{Y}_{t_1}^{t_{n_i}}$ the observed trajectories of the process Y_t for i^{th} individual, where n_i is the number of observations of the process Y_t for i^{th} individual. Let $\delta_i = 1$ if i^{th} individual died in the interval $[t_{n_i}^i, t_{n_i+1}^i)$, $\delta_i = 0$ if he/she survived until age $t_{n_i+1}^i$ and $\delta_i = 2$ if an individual is lost to follow up at the last observation (censored at age $t_{n_i}^i$). The contribution of i^{th} individual into the likelihood function is

$$L_i(\tilde{Y}_{t_1}^{t_{n_i}}, X_{t_1}^{t_{n_i}}, \delta_i) = P(\tilde{Y}_{t_1}^{t_{n_i}}, X_{t_1}^{t_{n_i}}, \delta_i) = p(Y_{t_1}^{t_{n_i}}) P(X_{t_1}^{t_{n_i}} | Y_{t_1}^{t_{n_i}}, \delta_i) = p(Y_{t_1}^{t_{n_i}}) \prod_{k=2}^{n_i} p(Y_{t_k}^{t_{n_i}} | \tilde{Y}_{t_1}^{t_{k-1}}) \prod_{k=1}^{n_i-1} \left(1 - Q(t_k, \tilde{Y}_{t_1}^{t_k})\right) \left(1 - Q(t_{n_i}, \tilde{Y}_{t_1}^{t_{n_i}})\right)^{I(\delta_i=0)} Q(t_{n_i}, \tilde{Y}_{t_1}^{t_{n_i}})^{I(\delta_i=1)}, \quad (\text{A7})$$

where the respective probabilities are given by (A2)-(A6). The likelihood function is the

product of $L_i(\tilde{Y}_{t_1}^{t_{n_i}}, X_{t_1}^{t_{n_i}}, \delta_i), i=1 \dots N$.

Application to the NLTCS-Medicare data on DI and MCI

We applied the model to the DI calculated from the NLTCS data for males and females and the MCI calculated for the respective individuals using the related Medicare files. Note that total Medicare costs are available only for years 1984–2001. Therefore, observations of DI in year 1982 are excluded. For an individual with the DI measured at age x , respective Medicare costs were calculated by summing costs for all time periods with the individual's age in the range $[x, x+1)$. We calculated the discrete models for one-year intervals of observation. That

is, the observed value of the DI is assumed constant during the respective interval after the observation. Note also that this model assumes that we consider the fact of death only during the respective (one-year) time interval after the observation (i.e., if an individual dies within that time interval after the last observation then he/she is considered died and if he/she survives this period then the individual is considered censored).

We applied three models to the data. The first one uses non-diagonal matrices μ_t^1 and a_t representing interactions between two indices (DI and MCI). The second model uses diagonal matrices μ_t^1 and a_t . The third model is a one-dimensional version of the model (A1)-(A7) applied to separate indices (DI and MCI). Below is the detailed description of the models.

Model 1

We assume $\mu_t^0 = a_{\mu^0} e^{b_{\mu^0}(t-t_{\min})}$, $\mu_t^1 = \begin{pmatrix} a_{\mu^{11}} + b_{\mu^{11}}(t-t_{\min}) & a_{\mu^{12}} + b_{\mu^{12}}(t-t_{\min}) \\ a_{\mu^{12}} + b_{\mu^{12}}(t-t_{\min}) & a_{\mu^{22}} + b_{\mu^{22}}(t-t_{\min}) \end{pmatrix}$, $a_t = \begin{pmatrix} a_y^{11} & a_y^{12} \\ a_y^{21} & a_y^{22} \end{pmatrix}$, $\sigma_0 = \begin{pmatrix} \sigma_{01} & 0 \\ 0 & \sigma_{02} \end{pmatrix}$, $\sigma_1 = \begin{pmatrix} \sigma_{11} & 0 \\ 0 & \sigma_{12} \end{pmatrix}$, $f_t^1 = \begin{pmatrix} a_{f_1^1} + b_{f_1^1}(t-t_{\min}) \\ a_{f_2^1} + b_{f_2^1}(t-t_{\min}) \end{pmatrix}$, $f_t = \begin{pmatrix} a_{f_1} + b_{f_1}(t-t_{\min}) \\ a_{f_2} + b_{f_2}(t-t_{\min}) \end{pmatrix}$, where $t_{\min} = 65$. Parameters to be estimated in this model are: a_{μ^0} , b_{μ^0} , $a_{\mu^{11}}$, $b_{\mu^{11}}$, $a_{\mu^{12}}$, $b_{\mu^{12}}$, $a_{\mu^{22}}$, $b_{\mu^{22}}$, a_y^{11} , a_y^{12} , a_y^{21} , a_y^{22} , σ_{01} , σ_{02} , σ_{11} , σ_{12} , $a_{f_1^1}$, $b_{f_1^1}$, $a_{f_2^1}$, $b_{f_2^1}$, a_{f_1} , b_{f_1} , a_{f_2} , and b_{f_2} .

Model 2

Same as Model 1 but without interaction terms in μ_t^1 and a_t : $a_t: a_t = \begin{pmatrix} a_y^{11} & 0 \\ 0 & a_y^{22} \end{pmatrix}$ and $\mu_t^1 = \begin{pmatrix} a_{\mu^{11}} + b_{\mu^{11}}(t-t_{\min}) & 0 \\ 0 & a_{\mu^{22}} + b_{\mu^{22}}(t-t_{\min}) \end{pmatrix}$, where $t_{\min} = 65$. Parameters to be estimated in this model are: a_{μ^0} , b_{μ^0} , $a_{\mu^{11}}$, $b_{\mu^{11}}$, $a_{\mu^{22}}$, $b_{\mu^{22}}$, a_y^{11} , a_y^{22} , σ_{01} , σ_{02} , σ_{11} , σ_{12} , $a_{f_1^1}$, $b_{f_1^1}$, $a_{f_2^1}$, $b_{f_2^1}$, a_{f_1} , b_{f_1} , a_{f_2} , and b_{f_2} .

Model 3

A one-dimensional version of Model 1: $\mu_t^0 = a_{\mu^0j} e^{b_{\mu^0j}(t-t_{\min})}$, $\mu_t^1 = a_{\mu^{1j}} + b_{\mu^{1j}}(t-t_{\min})$, $a_t = a_y^{1j}$, $\sigma_0 = \sigma_{0j}$, $\sigma_1 = \sigma_{1j}$, $f_t^1 = a_{f_1^1} + b_{f_1^1}(t-t_{\min})$, $f_t = a_{f_j} + b_{f_j}(t-t_{\min})$, where $t_{\min} = 65$, and $j = 1$ (DI), 2 (MCI). Parameters to be estimated in this model are: a_{μ^0j} , b_{μ^0j} , $a_{\mu^{1j}}$, $b_{\mu^{1j}}$, a_y^{1j} , σ_{0j} , σ_{1j} , $a_{f_1^1}$, $b_{f_1^1}$, a_{f_j} , and b_{f_j} , with $j = 1$ for the DI, and $j = 2$ for the MCI.

Note that Models 1 and 2 are nested, but all three models are not (Model 3 estimates DI and MCI data independently and thus has different estimates of baseline hazards for DI and MCI). Thus, to compare all three models, we will use the Akaike information criterion (AIC). Table 1 reports differences between AIC statistics for the current model and the model with the minimal AIC, which is Model 1 for both females and males. The column “ln L” in Table 1 reports the total (logarithm of) likelihood for Model 3, i.e., the sum of the likelihoods for DI and MCI data.

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Figure 1.

Means, standard deviations and correlation coefficients (with 95% confidence intervals) of DI and MCI for females and males in the NLTCs and the linked Medicare data.

Figure 2.

Logarithms of total mortality rate in the NLTCs data approximated by the Gompertz function (“Gompertz”) and the baseline hazards ($\ln \mu_i^0$) in three different models applied to these data: a two-dimensional QHM with (Model 1, see Appendix) and without (Model 2, see Appendix) interaction terms, and a one-dimensional QHM (Model 3, see Appendix).

Figure 3. Application of two-dimensional QHM with interaction terms (Model 1, see Appendix) to data on DI and MCI for males and females: estimates of quadratic terms in the hazard (μ_t^{11} , μ_t^{12} , μ_t^{22}) and logarithms of the baseline mortality rate ($\ln \mu_t^0$).

Figure 4. Application of two-dimensional QHM with interaction terms (Model 1, see Appendix) to data on DI and MCI for males and females: estimates of functions f_t^1 (see Appendix) and f_t for DI and MCI.

Figure 5. Application of two-dimensional QHM with interaction terms (Model 1, see Appendix) to data on DI and MCI for females: estimates of the quadratic term in mortality ($\mu(t, Y_t) - \mu_t^0$) for different ages (t).

Figure 6.

Application of two-dimensional QHM with interaction terms (Model 1, see Appendix) to data on DI and MCI for males: estimates of the quadratic term in mortality ($\mu(t, Y_t) - \mu_t^0$) for different ages (t).

Table 1

Application of two-dimensional QHM with (Model 1, see Appendix) and without (Model 2, see Appendix) interaction terms to data on DI and MCI: estimates of parameters for females and males. Results for a one-dimensional QHM (Model 3, see Appendix) estimating parameters for DI and MCI data independently are given for comparison.

Females:	$a_{\mu^0} \cdot 10^{-2}$		b_{μ^0}	$a_{\mu^{11}}$	$b_{\mu^{11}}$	$a_{\mu^{12} \cdot 10^5}$	$b_{\mu^{12} \cdot 10^7}$	$a_{\mu^{22} \cdot 10^{10}}$	$b_{\mu^{22} \cdot 10^{12}}$	a_Y^{11}	$a_Y^{12} \cdot 10^7$	a_Y^{21}	a_Y^{22}	σ_{01}	Gain LE_{65}^c
	σ_{11}	σ_{02}	σ_{12}	σ_{12}	a_{μ^1}	$b_{\mu^1 \cdot 10^2}$	a_{μ^2}	b_{μ^2}	a_{μ^0}	$b_{\mu^0 \cdot 10^2}$	a_{μ^1}	b_{μ^1}	a_{μ^2}	b_{μ^2}	
Model 1	0.235	0.113	0.488	0.488	0.004	0.487	-0.404	0.752	-1.301	0.469	-1.107	-0.021	0.913	0.146	13.8
Model 2	0.256	0.112	0.488	0.488	0.016			0.744	0.235	0.427			0.887	0.145	
Model 3 (DI)	0.863	0.077	0.648	0.648	0.009					0.383				0.146	
Model 3 (MCI)	1.253	0.085						0.934	0.065	$b_{\mu^1} \cdot 10^2$	a_{μ^2}	b_{μ^2}	$\ln L^a$	AIC^b	
															13.2
Model 1	0.127	13723.06	14752.51	14752.51	0.184	0.471	5577.74	39.650	0.061	-0.120	0.032	0.101	-144892.73	0.00	
Model 2	0.127	13723.08	14752.51	14752.51	0.183	0.474	5577.75	39.656	0.069	-0.024	0.000	0.000	-144931.11	68.75	
Model 3 (DI)	0.121				0.195	0.447			0.034	0.130			-147448.68	5107.89	
Model 3 (MCI)		13722.98	14752.77				5568.21	40.416			0.044	-0.090			8.7
															3.9
Males	$a_{\mu^0} \cdot 10^{-2}$		b_{μ^0}	$a_{\mu^{11}}$	$b_{\mu^{11}}$	$a_{\mu^{12} \cdot 10^5}$	$b_{\mu^{12} \cdot 10^7}$	$a_{\mu^{22} \cdot 10^{10}}$	$b_{\mu^{22} \cdot 10^{12}}$	a_Y^{11}	$a_Y^{12} \cdot 10^7$	a_Y^{21}	a_Y^{22}	σ_{01}	Gain LE_{65}^c
	σ_{11}	σ_{02}	σ_{12}	σ_{12}	a_{μ^1}	$b_{\mu^1 \cdot 10^2}$	a_{μ^2}	b_{μ^2}	a_{μ^0}	$b_{\mu^0 \cdot 10^2}$	a_{μ^1}	b_{μ^1}	a_{μ^2}	b_{μ^2}	
Model 1	1.009	0.079	0.576	0.576	0.017	0.043	2.785	0.186	2.264	0.378	8.328	0.274	0.798	0.154	11.6
Model 2	1.169	0.078	0.594	0.594	0.028			0.204	3.976	0.385			0.813	0.154	
Model 3 (DI)	2.125	0.058	0.700	0.700	0.022					0.352				0.156	
Model 3 (MCI)	3.953	0.059						0.249	5.126	$b_{\mu^1} \cdot 10^2$	a_{μ^2}	b_{μ^2}	$\ln L^a$	AIC^b	
															10.3
Model 1	0.128	14422.02	15103.07	15103.07	0.180	0.402	5385.33	80.866	0.001	-0.002	0.710	-0.121	-76607.49	0.00	
Model 2	0.128	14422.94	15103.31	15103.31	0.181	0.396	5385.60	80.858	0.000	0.119	0.710	-1.665	-76642.85	62.71	
Model 3 (DI)	0.123				0.198	0.347			0.000	0.000			-79144.09	5069.19	
Model 3 (MCI)		14412.91	15100.25				5200.84	99.959			0.006	-0.014			7.5
															1.6

^a $\ln L$ — logarithm of the likelihood function for respective models (for Model 3, this is the sum of respective likelihoods for DI and MCI data)

^b AIC — differences between AIC statistics for the current model and the model with the minimal AIC

^c **Gain LE_{65}** — gain in remaining life expectancy after age 65 calculated using the baseline mortality (μ_T^0) in respective models and the total mortality (μ_T) in the Gompertz model