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Towards better prediction of individual longevity

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CONTENTS

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Abstract

For a single individual the remaining length of life seems highly unpredictable. However, factors may be identified that characterise groups of individuals with shorter or longer longevity. The characterisation of such groups may contribute to the discussion on how to insure longevity risk. This study aims to maximise predictive value and to quantify the remaining uncertainty. We identify the predictive value of a broad selection of potential predictors, based on a 24-year mortality follow-up in the Longitudinal Aging Study Amsterdam, which is representative for the 55-85 years age group in the Netherlands (n=3,088, period 1993-2017). Potential predictors involved six domains: socio-demographics, disease history and medication use, physical functioning, lifestyle, psychosocial factors, and blood markers. We found significant predictors across all domains, including both self-reported and objectively tested measures. The significant predictors in the first five domains explained 21.3% of the variance in longevity. Additional predictive value of 3.7% was contributed by blood markers of disease processes and a genetic marker. We conclude that the prediction of individual longevity requires a broad set of variables, including both subjective and objective information. Yet, 75% of the variance in longevity remains unexplained, so that a large error margin remains in the prediction of an individual's longevity.

Samenvatting

Naar betere voorspelling van de individuele levensduur

Voor individuen lijkt het behoorlijk onvoorspelbaar hoe lang zij nog zullen leven. Wél kunnen factoren worden opgespoord op grond waarvan we individuen kunnen groeperen in klassen met langere of kortere levensduur. De bepaling van zulke klassen kan bijdragen aan de discussie hoe langlevenrisico te verzekeren. Ons onderzoek heeft tot doel factoren te vinden met een zo groot mogelijke voorspellende waarde voor de individuele levensduur en de overblijvende onzekerheid te kwantificeren. Wij identificeren daartoe een breed scala aan potentiële predictoren, gebaseerd op analyses van sterfte over 24 jaar in de Longitudinal Aging Study Amsterdam, een representatief onderzoek voor de leeftijdsgroep 55-85 jaar in Nederland (n=3.088, periode 1993-2017). Potentiële predictoren omvatten zes domeinen: sociaaldemografische kenmerken, ziekten en medicijngebruik, lichamelijk functioneren, leefstijl, psychosociale factoren en bloedwaarden. We vinden in alle domeinen significante predictoren, zowel zelf-gerapporteerde als objectief geteste. De significante predictoren in de eerste vijf domeinen tezamen verklaren 21,3% van de variantie in de resterende levensduur. Het zesde domein, bloedwaarden als indicatoren van ziekteprocessen en erfelijkheid, verklaart 3,7% extra variantie. Wij concluderen dat het voorspellen van de individuele levensduur een breed scala aan factoren vereist, met zowel subjectieve als objectieve informatie. Niettemin blijft 75% van de variantie onverklaard, waardoor er een grote foutenmarge blijft bestaan in de voorspelling van de levensduur van een individu.

Executive summary

Study aim

A large variation exists in longevity across individuals of the same age and sex. This variation stems from differences in health status as well as in other factors that may influence longevity. For a single individual the remaining length of life seems highly unpredictable. However, factors may be identified that characterise groups of individuals with similar longevity. The characterisation of such groups may contribute to the discussion on how to insure longevity risk. In particular, but leaving this for future research, insurance companies and pension funds may be able to assess more precisely on an individual basis what the financial costs ('liabilities') of a pension are. Using state-of-the-art measures, identification of predictors of individual longevity, and assessment of the total predictive value of identifiable predictors, this study aims to maximise predictive value and to quantify the remaining uncertainty.

Summary of findings

Using the Longitudinal Aging Study Amsterdam, we were able to estimate 24-year mortality for 3,088 individuals aged 55-85 years at baseline over the 1993-2017 period. Potential predictors covered six domains: socio-demographics, disease history and medication use, physical functioning, lifestyle, psychosocial factors, and blood markers. Using the logit of the Realised Probability of Dying (LRPD) as the dependent variable, we tested the predictive value of the six domains in linear regression models, evaluating their variance explained (adjusted R-square). We found significant predictors across all domains, including both self-reported and objectively measured variables. The significant predictors in the first five domains explained 21.3% of the variance in the LRPD. Additional predictive value of 3.7% was contributed by blood markers of disease processes and a genetic marker. We conclude that the prediction of individual longevity requires a broad set of variables, including both subjective and objective information. Yet, 75% of the variance in longevity remains unexplained, so that a large error margin remains in the prediction of individual longevity.

Implications

In the face of the ageing of the population, the sustainability of risk insurance is gaining in importance. The very long follow-up of 24 years is relevant to insurers, as they need to assess longevity risk over a very long period as well. Our findings show that the prediction of longevity has a large error margin, despite careful selection of the set of predictors. This unpredictability links in with the issue of how to insure

longevity risk. One option to maintain an insurance at a sustainable level is to move from collective towards individual risk insurance. However, as our findings imply, the cost of a lifelong pension for an individual person is highly unpredictable. Therefore, a more realistic basis for longevity risk insurance is to define larger groups of individuals, where the substantial errors in longevity assessment cancel each other out. An issue to address in future research is then to design longevity-risk sharing schemes based on a situation where the individuals to be insured differ in terms of their longevity.

1. Introduction

Large variation exists in longevity across individuals of the same age and sex. This variation stems from differences in health status as well as in other factors that may influence longevity. Moreover, as one grows older, one's health status is the outcome of the interaction of multiple factors, the combination of which differs for almost each individual (Fried et al. 1998). For a single individual the remaining length of life thus seems highly unpredictable. Likewise, for insurance companies and pension funds, the costs involved in individual pensions are highly unpredictable. Using state-of-the-art measures, identification of predictors of individual longevity, and assessment of the total predictive value of identifiable predictors, this study aims to maximise predictive value and to quantify the remaining uncertainty. Thus, but leaving this for future research, insurance companies and pension funds may be able to assess more precisely on an individual basis what the financial costs ('liabilities') of a pension are.

The prediction of longevity enjoys long-standing interest among gerontologists and scholars from other disciplines. Palmore (1970) was among the first scholars to report empirical data on the prediction of longevity. Numerous studies followed, most of them focusing on a certain factor or group of factors. More recently, it is recognised that only a broad selection of factors can achieve sufficient predictive value (Goldman et al. 2016; Iacob et al. 2016; Suemoto et al. 2017). However, the question how well these factors explain longevity has hardly been addressed. A study by Deeg et al. (1989), using a population-based sample of 2,645 people aged 65 and over at baseline in 1956, with 37-year follow-up and a wide range of potential predictors available, showed that only 20% of the variance in longevity could be explained. The available predictors constituted three domains: (1) results from bio-medical, physical, and mental examinations, (2) self-reported disability and health care use, and (3) social and psychological characteristics. The biomedical measures in the first domain, however, did not include measures that are currently considered to be standard risk factors. The predictive ability could be greater when state-of-the-art biomedical measures are used.

More recently, several other studies have attempted to include an as wide as possible array of variables in order to improve the prediction of longevity (Fried et al. 1998, Newman et al. 2009, Goldman et al. 2017). In their comprehensive study, Fried et al. used the U.S. Cardiovascular Health Study of ages 65 and over, which started in 1989 and at the time had a 5-year follow-up. The strongest predictors of longevity were found to be risk factors for cardiovascular diseases, including weight, smoking, physical activity, blood pressure, diabetes, and arteriosclerosis. Also a cognitive test

showed predictive value. In this study, objective tests of risk factors showed stronger predictive ability than self-reports of health by study participants. The interest of these scholars was in the uniqueness of predictors, and no total predictive value was reported. Likewise, Newman et al., using the same U.S. Cardiovascular Health Study with a 16-year follow-up, focused on predictors with unique predictive ability. With the longer follow-up, these authors were able to distinguish mortality by broad causes of death. Some specific predictors of cause-specific mortality proved to be lung function (for pulmonary death) and the gene apolipoprotein E epsilon 4 (for dementia death). Across all causes of death, age, sex, chronic inflammation as indicated by the blood marker interleukin-6, and cognitive function proved to be predictive. Thus, in addition to chronic conditions and life style, blood parameters proved to add unique predictive value.

Goldman et al. (2017), using the U.S. National Health And Nutrition Examination Survey with follow-up from 1999 to 2006, distinguished the age groups 20-64, 65-79, and 80 and over. These authors selected the top-10 of strongest predictors of mortality from the literature, and categorised them into five groups, roughly indicating underlying health (groups 1-3) and less proximate predictors (groups 4-5): 1) chronic diseases and health care use, 2) self-reported health and functional limitations, 3) biological parameters, 4) socio-demographic characteristics, and 5) lifestyle factors. Self-reported health and functional limitations were found to be the strongest predictors in each age group. The predictive value of functional limitations increased across age groups. In contrast, the predictive value of lifestyle factors and health care use (hospital admission and medication use) decreased after age 80. Although not among the strongest predictors, the blood marker albumin was predictive across all age groups.

Although they were comprehensive, none of the recent studies discussed reported the joint predictive value of the predictors found. A common parameter that reflects predictive value is the variance explained. However, this parameter can be calculated directly only from linear regression models. The most commonly used method to analyse predictors of longevity, Cox regression analysis, has as the dependent variable time to death or end-of-study, which does not enable calculation of variance explained. Other common measures of predictive value, such as Harrell's C, are hampered by the censoring of study participants who survived to the end of the study and/or are not suitable for models with many covariates (White et al. 2015). Therefore, in this study we use an alternative measure of longevity which allows linear regression analysis and thus does provide for calculation of the variance explained (Deeg et al. 1989b).

The type of study that is ideally suited for estimating the predictive value of correlates of longevity in older persons combines the following characteristics. First, the sample should be sufficiently large and non-selective, such that the results will apply optimally to population-based policies. Second, the sample subjects should be followed up for a long time, such that exact survival information can be obtained for the majority of the sample, which will serve to improve the predictive value of factors related to longevity. Third, the potential predictors should cover a wide range of aspects of health as well as non-health domains, again in order to improve predictive value.

In sum, earlier research has shown that, despite the use of a broad array of predictors, only limited variance in longevity is explained. However, with biomarkers becoming widely available in epidemiological studies, greater predictive ability of longevity may be obtained. This paper addresses two questions. First, what is the predictive value for longevity based on a comprehensive set of potential predictors from physical as well as mental and social domains? Second, to what extent do biomarkers add predictive value?

2. Methods

Sample

Data are used from the Longitudinal Aging Study Amsterdam (LASA), an ongoing, nationally representative longitudinal study with baseline measurement wave in 1992–93 (Hoogendijk et al. 2016). LASA is based on a nationally representative cohort. Its sample was recruited from the municipal registries of eleven municipalities in three geographic regions that together are representative of the socio-cultural variety in the Netherlands: the regions around Amsterdam (West), Zwolle (North-East), and Oss (South). The initial ages of sample participants were 55–85 years, with oversampling of men and older ages. The sample was first used for the NESTOR study on Living Arrangements and Social Networks of older adults (LSN), which had a response rate of 62.3% (n = 3,805) (Knipscheer et al. 1995). About ten months after the LSN interview, the participants were approached for the first LASA cycle (1992–93). The 1992–93 cycle is the baseline for the current study. By the start of the LASA baseline, there were 3,679 surviving LSN participants. Of these survivors, 3,107 took part in the interviews and tests, yielding a response rate of 84.5%. The 15.5% non-response consisted of 3.6% ineligible because they were unable, 1.1% not contacted after eight or more attempts, and 10.7% refusals (Deeg et al. 2002).

The baseline LASA cycle consisted of two face-to-face interviews in the homes of the participants, including standardised questionnaires and performance tests. The first interview covered comprehensive aspects of physical, cognitive, emotional, and social functioning. The second interview took place about 2–6 weeks after the first and focused on lifestyle and clinical measures. At the end of the second interview, blood samples were collected and stored. From the participants in the first interview, 86% took part in the second interview. Blood samples were collected from 67% of these participants: only in the Zwolle region and in part of the Amsterdam region.

Measures

Mortality

Vital status of the LASA respondents, including date of death, is ascertained periodically through linkage to the national population register. For the current study, mortality ascertainment up to December 31, 2016 was used, providing about 24 years of mortality follow-up. Mortality follow-up was nearly complete; 19 respondents (0.6%) could not be traced through the registry.

The dependent variable is operationally defined as the Realised Probability of Dying (RPD). The RPD is an individual measure of survival time relative to the total population, based on sex and age at baseline (Deeg et al. 1989b). As such, the RPD belongs to the family of relative survival measures (Rutherford et al. 2012). These measures, including the RPD, have been used in particular in cancer survival research (Blesch et al. 1996, Cronin & Feuer 2000). In one article, results using the RPD and Cox proportional hazards regression were compared (Deeg et al. 1990). The linear regression of the RPD yielded more sensitive results than Cox regression. The greater sensitivity of models that use the RPD can be attributed to the fact that the RPD is an individual measure based on age and sex, whereas Cox models aggregate across age and sex. Adjustment of Cox models for age and sex does not solve this shortcoming.

Using life tables based on the total population for subsequent years (1992 through 2016) during the study period, the RPD compares for each individual of a specific age and sex this person's survival probability with the overall survival probability of the Dutch population of that age and sex, from the starting month of the study up to December 31, 2016. In formula terms:

$$RPD = (1 - d_1^{(a,s)}) \ldots (1 - d_n^{(a,s)})$$

where n is the total number of calendar years during which the participant is followed up to death or end-of-study, d_i is the probability of death according to the life table in calendar year i (i=1 ... n), a is the age, and s is the sex of the participant.

Possible values of the RPD lie between 0 and 1. These values introduce a rank order among all sample subjects. The reference population has a mean RPD of 0.50. If the RPD is greater than 0.50, this means that the subject has lived a relatively short time; if it is less than 0.50, the subject has lived a relatively long time. For example, the value of a man's RPD is 0.80 if 80% of his age and sex peers in the total population are still alive at the time of his death. The name "realised probability of dying" comes from the notion that the individual has "realised" the probability of death when a certain percentage of the reference population is expected to be still alive. The actual amount of survival time needed to reach a particular RPD varies according to the age and sex of the individual at baseline, with older people needing less time to achieve a lower RPD than younger people, and men needing less time than women. For example, a woman aged 70 years when first participating in LASA in 1993, who dies after 23 years in 2016, has an RPD of 0.20. By comparison, a women aged 80 years in 1993 will have the same RPD of 0.20 when she dies after 13 years, in 2006.

For those participants still alive at the end of the study period (December 31, 2016), i.e. 21.2% of the study sample (n=655), a value of the Realized Probability of Dying is imputed. The RPD for these participants is estimated by assuming that their remaining survival time corresponds to the median population survival time from end-of-follow-up onward. This amounts to multiplying the probability of reaching their age at the end of 2016 by 0.5. In the above example, a woman of age 70 when examined in 1993, reaches the age of 93 in 2016 with probability 0.20. If she is still alive at the end of 2016, her imputed RPD is 0.10, implying that it is expected that she will die when only 10% of her cohort is still alive. This approach is derived from standard actuarial methods.

If the RPD has a mean value of 0.50, the survival distribution of the sample represents that of the total population. In this case, the logit of the RPD (LRPD=log(RPD/(1-RPD))) approaches a normal distribution with mean 0, and can be used as the dependent variable in linear regression analysis. The use of linear regression models allows assessment of the percentage of variance explained by individual or groups of potential predictors.

Potential predictors

Following Goldman et al. (2017), five domains of potential predictors of longevity were initially distinguished (domains 1–4 and 6, listed below). Because the LASA study is also strong on psychosocial measures, these were included as an additional domain of potential predictors (domain 5):

1. Socio-demographics
2. Disease history (including cognitive impairment), hospital admission, medicine use
3. Self-rated health, physical functioning, receipt of help with self-care
4. Life style
5. Psychosocial factors
6. Biological blood measures.

Domain 1. Socio-demographic covariates include: age, sex, education, income, housing tenure, partner status, living arrangements, housing, geographic region, and degree of urbanisation.

Education was assessed as the highest educational level attained: (1) elementary school or less, (2) secondary education, (3) college or university.

Income was measured as *spendable income*, by asking the participant about their income, considering income from work, pensions, other benefits, and dividends

in their own name. The same question was asked about the partner's income. The incomes of the participant and their partner were added together and then multiplied by 0.7. This correction is based on the ratio in Dutch state pensions for citizens living alone and those living with others.

As a measure of wealth, *housing tenure* was determined by asking whether the house was rented or owned and, in the latter case, if the house was subject to a mortgage or not.

Partner status was indicated by two dummy variables, distinguishing between having no partner or having a partner outside the household and having a partner.

Living arrangements were distinguished as living alone or living with others.

Housing was distinguished as community living or institutionalized.

Three *geographic regions* were distinguished using two dummy variables: North-East versus West and South, and South versus West and North-East.

Degree of *urbanization* was coded as (1) rural, i.e. < 500 addresses per square kilometre, ..., (5) highly urban, i.e. > 2,500 addresses per square kilometre.

Domain 2. Seven major *chronic disease* categories were assessed in the interview: respiratory diseases, heart diseases, peripheral artery disease, stroke, diabetes, arthritis, and cancer. In a validation study, respondents' self-reports of these diseases were compared to information obtained from their general practitioners, and proved to be reliable (Kriegsman et al. 1996). For some cardiovascular diseases, the correspondence with general practitioner data was relatively low. Therefore, for cardiovascular diseases (heart diseases, peripheral artery disease, and stroke) an algorithm was defined using information from the general practitioner and specific medicines used (Bremmer et al. 2006).

Cognitive impairments were ascertained using the Dutch translation of the MiniMental State Exam (MMSE, Folstein et al. 1975, Launer et al. 1993). On 23 questions and tasks, respondents received one or more points when they gave the correct answer or performed the task correctly. Scores range from 0 (all answers incorrect) to 30 (all answers correct).

Hospital admission was determined by the question whether the respondent had been admitted to the hospital in the past six months.

Medication use was recorded by the interviewer from the containers of drugs that the respondent was taking, with or without prescription.

Domain 3. This domain is characterised as consequences of ill health, including self-rated health and physical functioning.

Self-rated health was measured using one question, with codes from (1) very good, to (5) poor (Van Sonsbeek 1991).

Functional limitations were self-reported for six activities: climbing up and down a stair, walking outside for five minutes, dressing and undressing, cutting own toenails, getting up from and sitting down in a chair, and using own or public transportation. Response categories were (0) yes, without difficulty, (1) yes, with difficulty, (2) not able without help, and (3) cannot (Van Sonsbeek, 1988; Kriegsman, Deeg, Van Eijk, Penninx, & Boeke, 1997). The six items were added up to a single score ranging from 0 to 24.

Activity limitation was self-reported using the Global Activity Limitation Index, which asks about activity limitation that has lasted at least three months due to health problems. It is coded as (1) no limitations, (2) mild limitations, (3) severe limitations (Van Oyen et al. 2006).

Physical performance of the upper body was tested by asking the respondent to put on and take off a cardigan that was brought in by the interviewer (Magaziner et al. 1997). Lower body physical performance was tested by two tasks: walking three meters back and forth along a line, and getting up from and sitting down in a kitchen chair five times with arms folded (Guralnik et al. 1994). The time needed to perform these activities was measured in seconds. Walking time was transformed into walking speed in m/sec (Sanders et al. 2016). During the walk, the number of steps was recorded (Tinetti et al. 1986).

Incontinence of urine was included in the list of self-reported chronic diseases.

Receipt of help for personal care (such as dressing and bathing) was self-reported.

Domain 4. Life style includes smoking, alcohol use, body mass index and waist circumference as indicators of nutritional status, and physical activity.

Smoking was coded as: (0) never smoked, (1) stopped smoking 20 or more years ago, (2) stopped smoking less than 20 years ago, (3) current smoker (Visser et al. 1999).

Alcohol use was coded: (0) no, (1) moderate, (2) heavy, the latter being defined as drinking three or more glasses at one time (Garretsen 1983).

Height and weight were measured, and *body mass index* was calculated as: weight in kg/(height in m)^2.

Waist circumference was determined as the average of two measurements midway between the lower rib margin and the iliac crest after a normal expiration (Heim et al. 2010).

For *physical activity*, two indicators derived from the LASA Physical Activity Questionnaire indicated: number of minutes spent walking and number of minutes spent on sports, averaged per day (Stel et al. 2004).

Domain 5. This domain includes psychological well-being, personality characteristics, and social participation.

Depressive symptoms were ascertained using the Dutch translation of the 20-item Center for Epidemiologic Studies Depression scale (CES-D, Radloff 1977; Beekman et al. 1994). Respondents were asked to indicate how often during the past week they had experienced each symptom with response categories (0) (almost) never to (3) (almost) always. The score range is 0 (no symptoms) to 60 (maximum number of symptoms).

For *anxiety symptoms*, the 7-item anxiety subscale of the Hospital Anxiety and Depression scale (Zigmond and Snaith 1983) was used, with the same response categories as for the CES-D.

Personality characteristics assessed were sense of mastery and self-efficacy. Sense of *mastery* was measured using a five-item version of the Mastery scale (Pearlin and Schooler 1978). The scale ranges from 5 to 25, with Cronbach's alpha = 0.67.

Perceived general *self-efficacy* was assessed using a twelve-item version of the Self-efficacy scale (Sherer et al. 1982) that was adapted for use in the older population (Bosscher & Smit 1998). Three subscales are distinguished: *willingness to initiate behavior* (three items, Cronbach's alpha = 0.64), *persistence in the face of adversity* (four items, Cronbach's alpha = 0.65), and *willingness to spend effort in completing the behavior* (five items, Cronbach's alpha =0.63) (Penninx et al. 1997).

The *social network* was determined by asking respondents to name all persons aged 18 years and over with whom they maintained an important and regular contact (Van Tilburg 1998). The total number of persons named constitutes the *social network size*. For a maximum of nine persons with whom contact was most frequent, receiving and giving *instrumental* and *emotional support* were assessed using one question for each type of support, coded as: (0) never, ..., (3) often. Both forms of support were added across network members to a scale with maximum 27.

The respondent's experience of *loneliness* was assessed using the De Jong Gierveld Loneliness scale, in which social and emotional loneliness were distinguished with six and five items ranging from 0 to 6 and from 0 to 5, respectively (De Jong Gierveld & Kamphuis 1985, Van Tilburg & De Jong Gierveld 1999).

Social activities included involvement in clubs or organizations, taking a course, and time spent on hobbies (Smits et al. 1995). Two variables indicated involvement in clubs or organisations and distinguished board membership and volunteering.

They were coded (0) no member, (1) member but not on board/no volunteer, (2) board member/volunteer. Church membership was a third variable, specifying (0) no church member, (1) Protestant, (2) Roman Catholic, (3) other religion or philosophy of life. Taking a course or doing a study was coded (0) no or (1) yes. Time spent on hobbies was recorded in hours per day.

Domain 6. Blood samples were collected only from participants living in the West and North-East regions. Albumin, total cholesterol, and creatinin were measured from fresh blood. Subsequently, blood serum samples were frozen at –80°C until actual determination of specific blood markers. Blood measures included markers that are known to be associated with functional decline and mortality. These included three markers of systemic inflammation: Interkeukin-6 (Il6), C-reactive protein (CRP), and 1-antichymotrypsin (ACT), for which there is ample evidence of associations with functional decline and mortality (Krabbe et al. 2004); albumin, a marker of nutritional status which also plays a role in the inflammation process (Schalk et al. 2003); traditional markers of disease processes: the erythrocyte sedimentation rate and the number of leucocytes; creatinin, a marker of muscle weakness; total cholesterol, high values of which are a risk factor for cardiovascular disease, while low values are a risk factor for cognitive decline (Van den Kommer et al. 2008); and, finally, the genetic marker apolipoprotein E (ApoE), the epsilon-4 allele, which has been shown to be associated with cognitive and physical decline and with mortality (Melzer et al. 2005).

Serum levels of Il6, CRP, and ACT were determined using sensitive regular immunoassays (ELISA) developed and performed at Sanquin Research in Amsterdam (Dik et al. 2005). Results were expressed as picograms per millilitre for Il6, micrograms per millilitre for CRP, and percentage of pooled normal human plasma for ACT. This plasma pool contained 100% ACT, which is 300 mg/l. Serum albumin concentrations (g/L) were determined by using a bromcresol green (BCG) photometric assay in the laboratories of the ISALA clinic (Weezenlanden location) in Zwolle and of the Valerius clinic in Amsterdam. To control for between-laboratory differences, two-monthly measurements by the Dutch Foundation for Quality Assessment in Clinical Laboratories were used to fit a regression equation for each laboratory. Using these regression equations, the serum albumin levels in the LASA data were adjusted (Schalk et al. 2003).

The markers erythrocyte sedimentation rate, leukocyte count, and creatinin were measured using standard procedures.

Total *cholesterol* was measured by enzymatic colorimetry assay with a Hitachi 747 analyser using enzymatic colorimetry assay (Roche Diagnostics, Mannheim, Germany).

ApoE phenotypes were determined by isoelectric focusing of delipidated plasma samples, followed by immunoblotting. Participants were classified as epsilon-4 carriers for those with an ApoE epsilon-4 isoform (phenotypes epsilon-2/4, epsilon-3/4, epsilon-4/4) and as non-epsilon 4 carriers for those without an ApoE epsilon-4 isoform (phenotypes epsilon2/2, epsilon2/3, epsilon3/3) (Dik et al. 1999).

All blood markers were included in the analyses as continuous variables, except total cholesterol and ApoE. Both high (upper tertile) and low total cholesterol (lower tertile) were included in the analyses as dummy variables. For ApoE, a dummy variable was defined as (0) isoforms other than epsilon-4 or (1) epsilon-4.

Statistical analysis

The distribution of the RPD was examined to establish to what extent the mortality of the sample corresponded with the mortality of the reference population. Descriptive statistics were calculated for the potential predictors, and their bivariate association with the logit of the RPD (LRPD) was tested using analysis of variance or correlations. All predictors were tested regarding the linearity of their association with the LRPD. In case of non-linearity, the variable was categorised using cut-off points derived from the literature or else into tertiles.

The prediction analysis was carried out in two steps: 1. selection of a set of potential predictors for further analysis; 2. multivariate evaluation of predictive value for the LRPD, using the variance explained (R-square).

Step 1 was carried out separately for each of the six domains. Categorical variables with more than two categories were recoded into dummies in order to enable their evaluation in a linear regression model. Per domain, those variables that showed a significant bivariate association were included in a multiple linear regression model. If multicollinearity occurred (tolerance < 0.5), the variable with the greatest association with the LRPD was retained. Variables were included with a significance level of $p<0.20$ so as not to overlook variables that might contribute to the variance explained. From these domain-specific models, individual variables that proved significant at $p<0.20$ were retained for further evaluation in step 2 (Steyerberg 2009). The variance explained by the retained predictors in each domain was indicated by the adjusted R-square.

In step 2, a full linear regression model was examined with all variables that were retained from domains 1-5. Those variables that were insignificant at $p>0.20$ were removed one by one, starting with the least significant predictor. Domain 6 was added to this pruned model, and the improvement of the variance explained was evaluated over the previous model.

Table 1. Distribution characteristics of RPD and LRPD

	RPD	LRPD
Mean	0.503	0.062
Median	0.482	−0.071
Standard deviation	0.278	1.622
5% percentile	0.087	−2.349
95% percentile	0.944	2.829

As values were missing in more than 5% of the cases for several of the life style variables and in up to 50% of the cases for some of the blood values, missing values were imputed using multiple imputation (Rubin 1987). The imputation model used all potential predictors selected in step 2, as well as age, sex, and the LRPD. Fifty-five imputations were implemented with one hundred iterations per imputation. The variance explained (adjusted R-square) was calculated across the pooled dataset, using a Fisher-z transformation of each imputed dataset's R, averaging R across imputed datasets, and squaring the value obtained (Harel 2009).

3. Results

Distribution of the Realised Probability of Dying

The mean of the RPD was 0.503 (sd 0.278), its median was 0.482, and its 5% and 95% percentiles were 0.087 and 0.944 (Table 1). These parameters indicate that the mortality of the sample closely resembled that of the reference population. The distribution of the LRPD had mean 0.062 (sd 1.622) and closely resembled a normal distribution (Kolmogorov-Smirnov test: 0.056).

Potential predictors: bivariate associations

Of the 63 variables included for examination, almost all were significantly associated with the LRPD (Table 2). Variables that had significance levels higher than 0.20 were 'initiate behaviour' from the first subscale of self-efficacy and paid work. Due to the sex- and age-neutral definition of the (L)RPD, sex showed no association with the LRPD. However, age showed a weak association with LRPD (r=-0.034, p=0.060).

For the first analysis step, 61 predictors remained eligible for further analysis. These predictors represented all six domains. The examination per domain still yielded 47 predictors for further analysis (Table 3). Variables that were excluded were: urine incontinence, waist circumference, anxiety, sense of mastery, the two remaining self-efficacy subscales, social network size, emotional support given, both social and emotional loneliness, board membership of clubs or associations, time spent on hobbies, Interleukin-6, and both low and high cholesterol. After excluding these variables, the variance explained per domain ranged from 2.1% for socio-demographics to 7.0% and 7.9%, respectively, for disease history and the blood measures. Note, however, that the sample sizes varied for each domain, so that the predictive value of each domain could not be directly compared. It can be concluded that each domain still contributed to the total predictive value, but that the largest contributions came from the disease-related and biological domains.

Table 3. Variance explained by domain

	Number of predictors (sample size)	R-square
Domain 1	9 (n=2,528)	2.1%
Domain 2	9 (n=2,433)	7.0%
Domain 3	9 (n=2,257)	3.0%
Domain 4	5 (n=2,257)	5.6%
Domain 5	8 (n=2,812)	2.5%
Domain 6	7 (n=1,420)	7.9%

Table 2. Descriptive characteristics of potential predictors of LRPD

Predictor	N	% or M (sd)	Mean LRPD or correlation with LRPD	Significance of association predictor–LRPD
Domain 1: Socio-demographics				
Age	3,088	70.8 (8.8)	−0.034	0.060
Sex	3,088			
– Male	1,499	48.5%	0.058	0.903
– Female	1,589	51.5%	0.065	
Education	3,080			
– Low	1,370	44.5%	0.206	<0.001
– Middle	1,362	44.2%	−0.030	
– High	348	11.3%	−0.185	
Partner status	3,088			
– No partner	1,044	33.8%	0.192	0.001
– Partner in household	1,953	63.2%	0.009	
– Partner outside hhold	91	2.9%	−0.284	
Living arrangement	3,088			
– Alone	967	31.3%	0.142	0.061
– With others	2,121	68.7%	0.025	
Housing	3,088			
– Community-living	2,962	95.9%	0.021	<0.001
– Home for the aged	104	3.4%	1.040	
– Nursing home	22	0.7%	0.947	
Spendable income (kDfl)	2,607	1.92 (0.91)	−0.068	0.001
Home owner	2,945			
– Rents	1,833	62.2%	0.094	0.007
– Owns, with mortgage	477	16.2%	−0.064	
– Owns, no mortgage	635	21.6%	−0.118	
Geographic region	3,088			
– West	1,401	45.4%	0.081	0.183
– North-East	956	31.0%	−0.015	
– South	731	23.7%	0.124	
Domain 2: Disease-related				
Chronic lung diseases	3,066			
– No	2,707	88.3%	−0.012	<0.001
– Yes	359	11.7%	0.572	
Cardiovascular diseases	3,074			
– No	2,180	70.9%	−0.107	<0.001
– Possible	569	18.5%	0.411	
– Definite	325	10.6%	0.549	
Diabetes	3,067			
– No	2,826	92.1%	−0.021	<0.001
– Yes	241	7.9%	0.982	
Arthritis	3,066			
– No	1,998	65.2%	0.092	0.110
– Yes	1,068	34.8%	−0.007	
Cancer	3,067			
– No	2,783	90.7%	0.016	<0.001
– Yes	284	9.3%	0.469	
Cognitive impairment (MMSE)	3,072	26.8 (3.2)	−0.180	<0.001
– No	2,057	67.0%	−0.094	<0.001
– Mild	889	28.9%	0.264	
– Severe	126	4.1%	1.198	
Number of medications taken	2,652	1.8 (1.8)	0.219	<0.001

Predictor	N	% or M (sd)	Mean LRPD or correlation with LRPD	Significance of association predictor–LRPD
Pulse rate	2,578	69.9 (11.8)	0.069	<0.001
Hospital admission past 6 months	2,869			
– No	2,582	90.0%	−0.032	<0.001
– Yes	287	10.0%	0.518	
Domain 3: Physical functioning				
Self-rated health (1–5)	3,063	2.40 (0.93)	0.154	<0.001
Functional limitations (0–24)	2,937	1.90 (3.19)	0.224	<0.001
Activity limitations (1–3)	3,090	0.41 (0.69)	0.195	<0.001
– No	2,169	70.6%	−0.111	<0.001
– Mild	534	17.4%	0.182	
– Severe	368	12.0%	0.883	
Gait speed (m/sec)	2,814	0.82 (0.28)	−0.144	<0.001
Number of steps	2,820	11.4 (3.3)	0.115	<0.001
Chair rise time (sec)	2,667	12.6 (4.6)	0.090	<0.001
Dress–undress time (sec)	2,978	13.9 (7.5)	0.157	<0.001
Peak expiratory flow	2,612	403 (130)	−0.166	<0.001
Urine incontinence	3,066			
– No	2,587	84.4%	0.018	0.002
– Yes	479	15.6%	0.274	
Receipt of help for personal care	3,077			
– No	2,876	93.5%	−0.027	<0.001
– Yes	201	6.5%	1.330	
Domain 4: Life style				
Smoking	2,643			
– Never	821	31.1%	−0.147	<0.001
– Stopped ≥20 years ago	559	21.2%	−0.377	
– Stopped <20 years ago	594	22.5%	0.029	
– Current smoker	669	25.3%	0.493	
Alcohol use	2,643			
– No	589	22.3%	0.252	<0.001
– Light	1,937	73.3%	−0.083	
– Heavy	117	4.4%	0.482	
Body Mass Index	2,565			
– Low (<20)	65	2.5%	0.484	0.022
– Normal (20–24)	833	32.5%	0.007	
– Overweight (25–29)	1,192	46.5%	−0.083	
– Obese (>30)	475	18.5%	0.053	
Waist circumference	2,484	97.8 (11.1)	0.028	0.167
Time spent walking (min/day)	2,753	30.4 (43.3)	−0.057	0.002
Time spent on sports (min/day)	3,042	12.1 (25.5)	−0.076	<0.001
Domain 5: Psychosocial				
Depressive symptoms (CES-D, 0–60)	3,036	7.9 (7.7)	0.129	<0.001
Anxiety (HADS-A, 0–21)	2,899	2.5 (3.3)	0.054	0.003
Mastery (5–25)	2,968	17.2 (3.3)	−0.069	<0.001
Self-efficacy				
– Initiate (3–15)	2,867	8.1 (2.5)	−0.010	0.577
– Persist (4–20)	2,872	14.2 (2.7)	−0.035	0.064
– Complete (5–25)	2,876	19.4 (2.6)	−0.042	0.023
Social network size	2,867	13.8 (8.3)	−0.067	<0.001

Predictor	N	% or M (sd)	Mean LRPD or correlation with LRPD	Significance of association predictor–LRPD
Support received				
– Instrumental (0–27)	2,856	14.1 (6.8)	0.043	0.022
– Emotional (0–27)	2,853	21.4 (8.3)	−0.056	0.003
Support given				
– Instrumental (0–27)	2,857	13.3 (6.8)	−0.071	<0.001
– Emotional (0–27)	2,854	20.3 (8.5)	−0.044	0.018
Caregiver for partner	3,007			
– No	2,953	98.2%	0.057	0.070
– Yes	54	1.8%	−0.345	
Loneliness				
– Social (0–6)	3,025	0.93 (1.34)	0.053	0.004
– Emotional (0–5)	3,027	1.17 (1.69)	0078	<0.001
Paid job ≥1 hour	2,916			
– No	2,566	88.0%	0.040	0.486
– Yes	350	12.0%	−0.024	
Volunteer in clubs or associations	2,889			
– No member	1,087	37.6%	0.210	<0.001
– Member, no volunteer	1,115	40.0%	−0.065	
– Member and volunteer	647	22.4%	−0.110	
Member of board of clubs or associations	2,736			
– No member	1,087	39.7%	0.210	<0.001
– Member, no board	1,259	46.0%	−0.053	
– Member, board	390	14.3%	−0.210	
Follow a course/study	2,898			
– No	2,552	881%	0.068	<0.001
– Yes	346	11.9%	−0.263	
Religion (member)	3,087			
– No	1,157	37.5%	0.146	0.001
– Protestant	969	31.4%	−0.110	
– Roman Catholic	916	29.7%	0.140	
– Other	45	1.5%	−0.002	
Time (min/day) spent on hobbies	2,812	158 (120)	−0.034	0.075
Domain 6: Blood measurements				
Interleukin-6	1,738	2.2 (2.8)	0.069	0.004
C-Reactive Protein	1,738	4.6 (7.1)	0.189	<0.001
Alpha1-antichymotrypsin	1,735	174 (55)	0.158	<0.001
Serum albumin	1,500	45.4 (2.9)	−0.114	<0.001
Erythrocyte sedimentation rate	1,498	11.6 (11.5)	0.180	<0.001
Leukocytes	1,486	6.4 (1.7)	0.201	<0.001
Creatinin	1,499	93.1 (19.9)	0.090	0.001
Total cholesterol	1,500			
– Low	447	29.8%	0.006	0.502
– Middle	524	34.9%	−0.074	
– High	529	35.3%	0.038	
Apolipoprotein E epsilon4	1,730			
– No	1,258	72.7%	−0.075	0.004
– Yes	472	27.3%	0.173	

Predictors from multivariate analyses

Including domains 1–5 in one regression model, 14 predictors remained after backwards elimination of variables with p-values higher than 0.20. In this model, the variance explained was 15.6% (n=2,264). After adding the five variables remaining from domain 6, the variance explained rose to 17.6%. From this final model, time spent on sports (domain 4) was omitted, because its p-value had come to exceed 0.20.

The addition of domain 6 caused the number of cases to be reduced to 1,228, which amounts to 40% of the original sample. Therefore, with the final set of 18 variables, 55 multiple imputations were performed. The pooled dataset now showed for domains 1-5 a variance explained of 21.3%. Adding domain 6 yielded a total variance explained of 25.0%. Thus, the blood measurements added 3.7% to the total predictive value.

Table 4 shows for the final predictors their regression coefficients, their standard errors, and their significance for the pooled datasets for domains 1-5 (left three columns) and for domains 1-6 (right three columns). From domain 1, age retained its association with the LRPD, which was already apparent in the bivariate analyses. No other socio-demographic predictors survived the selection process. From domain 2, diabetes, cognitive impairment, and number of medications were predictive of shorter survival. In addition, arthritis was significantly associated with longer survival. From domain 3, self-reported functional limitations and receiving help with personal care predicted shorter survival and greater peak expiratory flow predicted longer survival. From domain 4, current smoking was a strong predictor of shorter survival, as was – to a lesser extent – having stopped smoking less than 20 years ago. Weaker predictors in this domain were heavy alcohol use and time spent walking, the latter having a protective effect. In domain 5, the only predictor that survived the selection process was church membership; members from both the Roman Catholic and Protestant churches showed longer survival than non-members or members of other religions or philosophies of life. The regression coefficient for Protestants was twice as large as for Roman Catholics.

When adding the blood measures from domain 6, the regression coefficients from domains 1-5 did not change much. The disease-related predictors in domain 2 showed somewhat reduced coefficients; the largest reduction (by 13.3%) was shown by number of medications taken. Also in domain 3, the predictive ability of functional limitations became weaker (by 27.0%), but that of receiving help for personal care increased (by 16.3%). In domain 4, the coefficients of the two smoking variables showed the greatest reduction: by 41.6% and 24.0%, respectively, for having stopped

Table 4. Full model of predictors identified and variance explained, without (left) and with (right) blood values

	B	SE(B)	p-value	B	SE(B)	p-value
Domain 1: Socio-demographics						
Age	−0.041	0.004	<0.001	−0.047	0.004	<0.001
Domain 2: Disease-related						
Diabetes	0.622	0.110	<0.001	0.554	0.102	<0.001
Arthritis	−0.275	0.059	<0.001	−0.255	0.058	<0.001
Cognitive impairment	−0.047	0.010	<0.001	−0.042	0.009	<0.001
Number of medications	0.135	0.018	<0.001	0.117	0.017	<0.001
Domain 3: Physical functioning						
Functional limitations	0.063	0.012	<0.001	0.046	0.011	<0.001
Peak expiratory flow	−0.002	0.000	<0.001	−0.002	0.000	<0.001
Help with personal care	0.447	0.137	0.001	0.520	0.130	<0.001
Domain 4: Lifestyle						
Heavy alcohol use	0.288	0.190	0.132	0.296	0.092	0.001
Stopped smoking <20y	0.245	0.074	0.001	0.143	0.069	0.038
Current smoker	0.641	0.073	<0.001	0.487	0.067	<0.001
Time spent on walking	−0.001	0.001	0.056	−0.001	0.001	0.025
Domain 5: Psychosocial						
Church member: RC	−0.096	0.065	0.142	−0.093	0.064	0.148
Church member: Prot	−0.210	0.065	0.001	−0.216	0.064	0.001
Domain 6: Blood measurements						
C-reactive protein				0.007	0.004	0.085
Erythr sedimentation rate				0.011	0.003	<0.001
Leukocytes				0.082	0.017	<0.001
Creatinin				0.006	0.002	<0.001
Apolipoprotein E epsilon-4				0.254	0.058	<0.001
Variance explained			21.3%			25.0%

smoking less than 20 years ago and current smoking, but both remained significant at p<0.05. The predictive value of domain 5 did not change substantially.

From the five blood measures in domain 6, the genetic marker apolipoprotein E epsilon-4 contributed most strongly to the predictive value of this domain; the inflammation marker C-reactive protein showed the weakest predictive value. The blood markers of disease processes, erythrocyte sedimentation rate and leukocyte count, and the marker of muscle weakness, creatinin, showed substantial contributions, independent from one another.

4. Discussion

This study focused on the prediction of survival in the general older population. In particular, it addressed the predictive value attained by a broad array of predictors from a variety of domains that were selected to maximise their joint predictive ability. We found that predictors that are often available in social epidemiological surveys explain 21.3% of the variance in survival. Blood measures were expected to substantially enhance the total predictive value, but actually added only 3.7% to the total variance explained. Thus, a total of 25.0% in survival time was explained, implying that a 75% unexplained error margin remains regarding the prediction of individual survival time. The significant predictors came from all domains, and included self-reported and objective measures. However, the additional predictive value of blood measures appears limited.

The findings from this study may contribute to the debate on how to insure longevity risk. If individuals could be grouped on the basis of factors that have a high predictive ability for longevity, this would help differentiate groups with higher and lower longevity risk. This approach assumes that by using a practicable, thus limited, number of factors, a high predictive ability can be achieved. However, an open question is what level of predictive ability would lead to substantially lower costs of financing a lifelong pension. In light of our findings, it would be better to design longevity-risk sharing schemes that are based on a situation where the individuals to be insured have different longevity.

The predictors in the final model largely correspond to those found in the literature on prediction of longevity, even though most earlier studies used a smaller selection of potential predictors. In contrast, several predictors commonly found in studies on longevity did not show any predictive ability in our study.

First, indicators of socio-economic status (education, income, wealth) did not maintain their predictive ability in the multivariate model of all domains. This might be explained by the fact that domains 2-6 include more proximal predictors of longevity, which are not often included in studies that focus on socio-economic status in relation to longevity. Regardless, inclusion of other measures of socio-economic status, such as poverty or area-based measures, might have upheld socio-economic status as a significant predictor (Huisman et al. 2013).

Second, cardiovascular diseases did not maintain their predictive ability. This might be due to the substantial improvements in treatment of these diseases over the past decades, which have reduced mortality from these diseases to the average mortality in the population (Deeg et al. 2013). Alternatively, more objective measures

of cardiovascular diseases, e.g. using electrocardiography, might have maintained their predictive ability.

Third, neither obesity nor underweight remained predictive in the final model, although the LRPD was particularly high for the category 'underweight'. This category, however, was relatively small, which may have limited its power. More substantially, contrary to weight measured at one point in time, loss of weight is more likely to predict mortality, in particular when weight loss is involuntary (Deeg et al. 1990, Wijnhoven et al. 2014). Unfortunately, we could not measure weight loss as we had no previous measurement before baseline.

Fourth, psychosocial predictors did not show any predictive ability in the final model, contrary to a previous study that covered the much shorter time period of 29 months (Penninx et al. 1997). It might be that psychosocial factors only work in the shorter term. An alternative explanation for this discrepancy is that the earlier study did not include objective functional measures such as peak expiratory flow, the presence of which may have reduced the predictive ability of psychosocial measures. Another previous study focused on types of social network, based not only on number, frequency, and diversity of contacts, but also on their supportiveness (Ellwardt et al. 2017). The type of network which combined many contacts with high supportiveness was predictive of longer survival. However, its predictive ability was shown to wear off at higher ages. And more importantly, no objective health indicators were accounted for, so that it remains uncertain that the predictive ability of this network type would be maintained. In sum, the potential predictors that we were able to include in domains 1–5 have a broad coverage, but may be improved.

The blood measures that we were able to include in domain 6 generally cover what is known from literature, but many more blood markers might have been explored. Examples are additional inflammatory markers such as tumor necrosis factor-alpha, glycosylated haemoglobin as a marker of the glucose metabolism, epinephrine as a marker of neuroendocrine function, homocysteine as a marker of deficiencies in B-vitamins and folic acid, and blood-circulating vitamin D (Goldman et al. 2006, 2016, 2017, Jylhävä et al. 2014, Sohl et al. 2015, Swart et al. 2012, Turra et al. 2005). Unfortunately, the measurement of these blood markers was not performed at LASA baseline. The genetic marker apolipoprotein E epsilon-4 showed good predictive ability. Until recently, this was the only genetic marker for which an association with ageing and mortality has been established (Newman et al. 2009). However, the field of genetics is developing fast, and new genetic markers of ageing and mortality are being established. So far, however, they have not replaced more established

predictors of mortality (Jylhävä et al. 2014). It remains to be seen how much the newly identified predictors of longevity add to the total variance explained.

An unexpected finding was the negative association of age with longevity (r=-0.034). This was unexpected, because our measure of longevity was based on single years of age and should not show an association with age. In an attempt to explain this association, it should be noted that 21% of our sample was still alive, and that the RPD of surviving participants was imputed based on the median remaining survival of their age and sex peers in the population, which amounted to multiplying their RPD up to the end of the study by 0.5. While this approach maintained the rank order of each participant in the face of expected survival, it caused a clustering of these participants around the value of 0.25. Multiplying their RPD by a random number between 0 and 1, the age association became 0.002. Thus, the negative age-association was artefact of the way we dealt with those who had not realised their probability of dying. However, by keeping age in the predictive model, any bias was accounted for.

A further limitation of our study was that it was based on predictors measured at one point in time. On the positive side, including predictors at only one point in time facilitates application in practice. For example, regarding individual longevity risk, insurers assess their clients only once and have to base their decisions on the information at hand. Likewise, clinicians see their patients only once or within a short time window and have to base their decisions regarding treatment on the information then obtained. On the negative side, predictors of longevity may change over time due to cohort or period effects. Predictors acting at a specific point in time may reflect the specific generation that is examined at that time (Deeg et al. 2013). For example, as subsequent generations have reached higher levels of education, the predictive ability of education may change. Vice versa, predictors may reflect the state of the art of medical science at the time point considered, but with better treatments becoming available, their predictive ability may change.

Implications

In the face of the ageing of the population, the sustainability of risk insurance is becoming more important. A strong feature of our study is the very long follow-up, 24 years in fact. This is relevant to insurers, as they assess longevity risk over a very long period as well. Our findings show that the predictive ability of a wide range of factors for the remaining length of life of individuals is limited. This low predictability links in with the issue of how to insure longevity risk. One option to maintain insurance at a sustainable level is to move away from collective towards individual risk insurance.

However, as our findings imply, the cost of a lifelong pension for an individual is hardly predictable. Therefore, a more realistic basis for longevity risk insurance is to define larger groups of individuals, where the substantial errors in longevity assessment cancel each other out. An issue to address in future research is how to design longevity risk sharing schemes that are based on a situation where the individuals to be insured have different longevity.

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