MODELLING AND FORECASTING HEALTH EXPECTANCY

Istvan M. Majer

Modelling and Forecasting Health Expectancy

Het modelleren en voorspellen van gezonde levensverwachting

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István Mátyás Májer

born in Miskolc, Hungary

DOCTORAL COMMITTEE

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Chapter 1

General introduction

1.1. Life expectancy

Life expectancy of a human population measures the expected (or average) remaining years of life at a given age. Life expectancy can be defined by two forms of measurement: the period and the cohort life expectancy. The period life expectancy represents the mortality conditions at a specific moment in time, in comparison to the cohort life expectancy, which depicts the life history of a specific group of individuals (born in the same year). Period life expectancies are frequently utilized when assessing and monitoring the health status of a population over time.

Currently, the life expectancy of the Western world population is much greater than in prior decades. The dramatic increase over the last century is considered as one of the great achievements of modern societies. Between 1970 and 2009 the average length of life in the old EU member states increased from 68.7 to 78.0 among men and from 74.9 to 83.5 among women[1]. Similar trends were witnessed among the elderly. Although there are many differences in the health service organization, national wealth, culture and associated individual behaviours between these countries, within a broader European perspective the upward trends are remarkably alike[2].

In the first half of the twentieth century eradication of infectious diseases caused unprecedented improvements in mortality, particularly at young ages. As the risk of dying from infectious diseases was reduced, those saved from dying at younger ages survived to middle and older ages[3]. A second wave of mortality improvement was achieved mainly at the adult life. Since the 1970s ischemic heart disease mortality has decreased in most Western countries contributing to the majority of the declines in age-specific death rates[4]. It has been suggested that the decline in cardiovascular disease mortality is due to the combination of better treatment and changes in risk factors such as: lower blood pressure, reduced tobacco consumption, diets containing lower cholesterol, and increased physical activity within the population[5]. There are of course more distal influences that contributed to the gains in overall mortality. The most important elements include increasing welfare, greater access to and better quality of health care, rising living standards and improvements in the level of education[6,7].

1.2. Health expectancy

In comparison with mortality, health is difficult to measure and estimates of population health are usually based on self-reported survey data. There is no single indicator that summarizes the health of a population thus no harmonized way of measuring health.

In the last two decades considerable effort has been put into the development of summary measures that assess population health. Such measures not only summarize information

of mortality alone (e.g. life expectancy) but rather combine information of mortality and non-fatal health outcomes[8,9]. There are two main classes of summary measures: health gap measures (e.g. disability-adjusted life years, years of life lost) and health expectancies (e.g. disability-free life expectancy, quality-adjusted life expectancy).

Referring to health gap measures, for example, one disability-adjusted life year lost (DALY) can be considered as one lost year of healthy life due to a disease. Therefore the burden of the disease, that is, the sum of these DALYs across a population is thought of a measurement of the gap between a population's actual health situation and an ideal situation where the entire population lives to an advanced age, free of disease and disability. In essence, DALYs for a health condition are calculated as the sum of the years of life lost due to premature mortality and the years lost due to disability of the health condition.

Health expectancy (or expected healthy life years) typically combines mortality and morbidity information to represent overall population health in a single indicator[10]. It measures the number of remaining life years that a person at a certain age is expected to live without ill-health, and is increasingly used to complement the conventional total life expectancy[10]. In its most commonly employed form, health expectancy is a functional health status measure, yielding the disability-free life expectancy and the life expectancy with disability measures. Because health expectancy was developed to reflect that not all years of a person's life are lived in perfect health, estimates of health expectancies have been very attractive and widely used tools for monitoring trends in population health[10]. In this thesis, I focus on health expectancy measures.

There are two commonly used methods to estimate health expectancy: Sullivan's method and the multistate life table (MSLT) method. They require different data inputs and can yield different results[11]. In the simple method, health expectancy is estimated by the Sullivan approach that combines mortality data with external information on cross-sectional (period) stock prevalence in each health state[12]. The MSLT method is a more refined way to estimate healthy life expectancy, it models the prevalence of morbidity as the result of several transitions (e.g. incidence, mortality and possibly remission)[13]. The MSLT method can be seen as a generalization of the original life table method, in which the 'alive' state is divided into two or more sub-states. Although an MSLT has larger data requirements since it needs age-specific estimates of multiple transitions, it has several advantages. Most importantly, it acknowledges the fact that the stock of ill health is the result of different processes. Accordingly, one can interpret trends in health expectancy as a result of developments in the underlying transition rates, that is, trends in incidence and remission from morbidity and trends in mortality of those in both the morbid and non-morbid health state. Using the MSLT method it is possible to decompose the population life expectancy into a

weighed average of the life expectancy of non-disabled and the life expectancy of disabled people.

In the literature applications of both the Sullivan and the multistate methods are easy to find. A large proportion of these studies focus on trends or country comparisons, but studies quantifying differences in health expectancy between subgroups of a population are also published. In the latter case, subgroups are often distinguished by socioeconomic status or are based on lifestyle factors such as smoking and overweight status. Although normally the results of these studies are difficult to compare directly because of the different health indicators and measurements of determinants used, the general picture is that there are significant differences between subgroups. Typically those with a more favourable risk factor status have higher health expectancy.

With respect to the evolution of the relationship between life and health expectancy over time, three distinct theories have been proposed: compression of morbidity, expansion of morbidity and the so-called dynamic equilibrium theory. Compression of morbidity, proposed by Fries[14], postulates that survival and morbidity curves will become more and more rectangularized and closer to each other in the future, as a result of strategies that effectively eliminate premature morbidity and mortality. Supporters of the pessimistic theory[15] assert that increases in life expectancy will not be followed by increases in healthy life expectancy because the declines in mortality stem from mainly those suffering from chronic, disabling diseases. Additionally, the age of onset of diseases is not postponed therefore people will live more years in a morbid state. The third theory emphasizes the link between morbidity and mortality. Supporters of the dynamic equilibrium argue that the severity and rate of progression of chronic disease are directly linked to mortality changes, and this relationship is in equilibrium over time (dynamic). They claim that increases in total life expectancy would likely entail increases in total life expectancy and life expectancy with morbidity but years with severe morbidity and disability remain relatively stable[16]. Eventually, the scale of increases in health expectancy and life expectancy with morbidity depends on the postponement of incidence and severity of morbidity. Compression (or expansion) of morbidity can be measured in absolute values (increases in healthy life expectancy is larger (or smaller) than increases in total life expectancy), or as a proportion (healthy life expectancy over total life expectancy is increasing (or decreasing)). Alternatively, one can refer to absolute compression of morbidity by decreasing number of years with ill-health, and to relative compression of morbidity by decreasing proportion of years with ill-health over total number of life-years.

1.3. Trends in health expectancy

Many studies have estimated health expectancy in the past using various study populations, disability measures and calendar periods. The most extensive work in assessing the evolution of past health expectancy has been conducted for the U.S. population. Crimmins et al. estimated that gains in life expectancy during the 1970s were mainly accompanied by increasing time spent with chronic limitations of common activities and by slight decreasing time with severe disability[17]. Later, Crimmins et al. found that during the 1980s gains in life expectancy rose along with disability-free life expectancy for both men and women[18]. In 2009 Crimmins et al. examined changes in life expectancy with and without disability in (instrumental) activities of daily living (ADL and IADL) using longitudinal data between 1984 and 2000[19]. They showed that the increase in disability-free life expectancy at age 70 was the same as the increase in life expectancy.

In Europe estimates of life expectancy and disability-free life expectancy for men and women were published for 13 European Union member states from 1995 until 2001 based on the European Community Household Panel[20]. Significant increases were found in life expectancy at early (age 16) and late (age 65) adulthood with considerable heterogeneity observed in health expectancy trends. In nine countries life expectancy with disability increased, whereas four countries had evidence of decreasing life expectancy with disability.

For the Netherlands, van de Water et al. studied trends in health expectancy between 1983 and 1990[21]. The study results expressed a rise (or decline) in the healthy life percentage of men (or women), where health status was defined by self-rated health status. Perenboom et al. assessed trends in disability-free life expectancy and life expectancy with disability according to levels of severity between 1989 and 2000[22]. At an aggregated level, for both men and women at age 65, a decrease in disability-free life expectancy and an increase in life expectancy with disability were observed. These trends were caused by greater increases in mild disability compared to decreases in severe disability. Trends in visual, hearing, mobility and ADL disability prevalence between 1990 and 1998 in the Netherlands were studied in Picavet et al.[23]. They found fairly stable disability prevalences during the study period.

In 2009 the Statistics Netherlands published trends on health expectancy estimates for the period 1981-2007[24]. Morbidity was measured in three different ways: self-rated health, presence of chronic disease and physical limitation. Since the 1980s, health expectancy has been increasing in men aged 65 measured in terms of self-reported good health or physical limitation. The increase has been somewhat even faster since the turn of the century. Unfortunately, among both men and women, life expectancy without chronic diseases at age 65 has been decreasing. For women, disability-free life expectancy increased and life expectancy without chronic diseases stagnated.

1.4. Forecasting life expectancy

The continuous rise of life expectancy is certainly welcome, however, in recent times it has been accompanied by low fertility rates, which accumulated in higher proportions of older people[25]. These developments have considerable consequences on the sustainability of two fundamental institutions of social security: health care and pension systems. Therefore there is a great need for insights and models of future mortality outlooks.

It is expected that further declines will predominantly be attributable to the older population, but the degree of improvements is rather uncertain. Representative to the ambiguity around the trend of human lifespan two groups of opinion evolved in the scientific community. Followers of the conservative view argue that human lifespan may have a natural limit (rectangularization of mortality curve)[26,27]. They claim that rapid increases in life expectancy, like those observed early in the 20th century, are no longer plausible. In contrast, advocates of unlimited longevity say that if there were an impending limit to further declines in death rates at older ages, countries with low levels of mortality would record slow rates of reduction. However, no correlation between levels of mortality and rates of reduction has been shown. In most developed countries the rate of reduction has accelerated, especially since 1970[28-30].

Forecasting life expectancy

The uncertainty around the future trend of mortality presents challenges, in particular, to governments, pension funds and life insurers. They have to deal with the uncertainties in some way, therefore financial specialists have already expressed much interest in the modelling of future evolution of life expectancy[31-34]. In fact, because future mortality declines may have potentially large financial and social consequences on pension costs, methodological developments in mortality forecasts have received increasing attention, and important work has been done in this field. Among the approaches to forecasting mortality, particular methods that do not include covariates became popular. The underlying assumption of these methods is that all the information about the future is contained in the past observed values of the log-mortality rate[35]. For example a non-parametric method based on principal components developed by Lee and Carter[31] is now widely used around the world to forecast all-cause and cause-specific mortality.

1.5. Forecasting health expectancy

In ageing populations it is an important question whether increases in life expectancy will be accompanied with greater or lesser increases in life years spent in poor health[36]. For that reason monitoring the level of life expectancy, health expectancy and their relationship is a key component of the public policy process that in turn, gives the chance to assess if the changes therein support the compression of morbidity, the expansion of morbidity or the

dynamic equilibrium theory. Forecasts of life and health expectancy could shed light on the future potential of disability compression.

For pension providers and financial organisations it is important to have expectations on how long people will live after they retire. Therefore forecasts of cohort life expectancy are often prepared by these institutions. Concerning retirement policies, however, it may not be sufficient to form expectations on only future (overall) life expectancy. If a further raise in retirement age is on the agenda, it is more appropriate to answer how long people will be able to work in the future, instead of simply answering how long they will live. In fact, many countries have already undertaken systematic adjustments in retirement age to increasing life expectancy[37,38]. Although the rationale for such restructuring was to improve the financial sustainability of pension systems, these reforms may have adverse social side-effects, as the extent to which total life expectancy increases may differ from the extent to which healthy life expectancy increases[39,40].

In the light of increasing health care expenditures it is important to assess to what extent future trends in health influence the demand for health care. Given the fact that health care expenditure generally increases with age and bad health status, health policy makers expressed concerns about the pressure population aging will exert on the fundability of the health care system, supplementary to the availability of sufficient numbers of health care workers[41].

Forecasting health expectancy

Contrary to forecasting life expectancy, virtually there is no literature on methodology or application of health expectancy projections. Along the lines of mortality projections, such methodology should reflect and combine the future trends in the underlying mechanisms of health and mortality. One possible approach is to invoke the multistate life table framework, thus addressing such mechanisms. If so, the transition rates or transition probabilities should be modelled, and these quantities should be some function of calendar time and other characteristics (e.g. age, sex). If the purpose is to extrapolate past trends into the future, then the time dimension should be extrapolated while everything else is kept constant. Ideally, uncertainty around the future estimates should be taken into account.

1.6. Aims of this thesis

So far the main focus of research has been on the trends of health expectancy in a given country or on cross-country comparisons between countries. Less emphasis has been put on determinants of health expectancy, such as obesity, smoking or educational level. Furthermore, studies aiming at predicting future health expectancy are rare. This is partly due to the lack of long time series data on health status. In addition, forecasting health

expectancy is based on multiple dimensions and is thus more complicated than forecasting life expectancy which is based on a single dimension, mortality. The main research questions addressed in this thesis aim to fill these gaps, and are as follows:

1. What are the mechanisms and factors that influence health expectancy?
2. What are the likely future trends in health expectancy?

1.7. Structure of this thesis

The first part of this thesis, Chapters 2, 3 and 4, aims to answer the first research question by establishing the extent to which health expectancy is related to major risk factors of population health. In these chapters health is measured in various ways based on data from repeated cross-sectional survey collected in the Netherlands (Chapter 2) and from longitudinal multinational surveys (Chapter 3 and 4). Chapter 2 investigates whether the mortality risk related to disability can be explained by other risk factors, and differences are quantified in terms of life expectancy between the nondisabled and the disabled population. Chapter 3 estimates the extent to which overweight status and smoking status is related to life expectancy with disability. In Chapter 4 differences in disability-free life expectancy are estimated for different educational levels. In both Chapters 3 and 4 estimates are given for Western-European countries.

The second part of the thesis, i.e. Chapters 5 to 7, aims to answer the second research question on future trends of health expectancy by projecting past trends between either 1981 and 2007 or 1989 and 2007 until 2020/2030. The implications of future trends in population health, covered in this thesis, involve changes in the prevalence of selected health conditions and economic consequences on long term care expenditures. Chapter 5 presents a multistate life table framework that can be applied to forecast the health expectancy of a population. Such an application is presented for the Netherlands where the health states are 'nondisabled', 'disabled' and 'dead'. Chapter 6 explores the effects of the future prevalence of disability on future long term care expenditures. In Chapter 7 future prevalence of overweight and obesity are estimated by taking into account the changing distribution of body mass index in the population.

Chapter 8 focuses on the main discussion points and elaborates on the policy implications of the likely trends. Finally, a summary of the thesis is provided in English and in Dutch.

PART I

UNDERSTANDING HEALTH EXPECTANCY

Chapter 2

Mortality risk associated with disability: a population-based record linkage study

Istvan M. Majer, Wilma J. Nusselder, Johan P. Mackenbach, Bart Klijs, and Pieter H.M. van Baal

Objectives

We assessed the association between mortality and disability and quantified the effect of disability-associated risk factors.

Methods

We linked data from cross-sectional health surveys in the Netherlands to the population registry to create a large data set comprising baseline covariates and an indicator of death. We used Cox regression models to estimate the hazard ratio of disability on mortality.

Results

Among men, the unadjusted hazard ratio for activities of daily living, mobility, or mild disability defined by the Organization for Economic Cooperation and Development at age 55 years was 7.85 (95% confidence interval [CI]=4.36, 14.13), 5.21 (95% CI=3.19, 8.51), and 1.87 (95% CI=1.58, 2.22), respectively. People with disability in activities of daily living and mobility had a 10-year shorter life expectancy than nondisabled people had, of which 6 years could be explained by differences in lifestyle, sociodemographics, and major chronic diseases.

Conclusions

Disabled people face a higher mortality risk than nondisabled people do. Although the difference can be explained by diseases and other risk factors for those with mild disability, we cannot rule out that more severe disabilities have an independent effect on mortality.

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2.1. Introduction

Population aging is associated with an increase in the number of people who are disabled. This increase presents a challenge for society because elderly persons disabled in 1 or more domains of life are hospitalized more often[42], need more medical and long-term care[43-46], and face a higher risk of death than nondisabled persons do[47-54].

Disablement refers to the impact that chronic and acute conditions have on people's ability to perform tasks necessary for daily living and normal social functioning[55]. In a broader context, the disablement process is described as a causal chain in which the progression of disease leads to functional limitations, loss of mobility, and eventually to inability to perform activities of daily living (ADLs)[55-58]. Empirical studies have found numerous risk factors associated with disablement. These factors are usually seen as risks that increase the chance of developing a disability. The major underlying causes are (acute and progressive) chronic diseases[59], but other risk factors including sociodemographic factors (e.g., age, gender[60], socioeconomic status[61]), behavioral factors (e.g., smoking)[62], nutrition[63], physical activity[64], comorbidity[59], self-rated health[65], and cognitive impairment[66] are also associated with incident disability.

Disability is most often assessed in cross-sectional studies without information on mortality. The few longitudinal studies that have been conducted tend to emphasize incident disability rather than the trajectory of disability following onset because of lack of statistical power[67]. Thus, although the onset of disability has been extensively researched, there has been far less investigation into the mortality risk associated with disability. In previous studies, the study populations were often limited to specific disease groups[48,52] or based on small sample sizes with few control variables[47,49-51,53,54]. Moreover, the focus was often on other determinants of mortality rather than on disability. Nonetheless, disability has been found to be an independent predictor of death after adjustment for heart disease[48], depressive symptoms[51], physical activity[54], socioeconomic status[53], or health status[51]. However, no study has assessed the extent to which the relationship between disability and mortality can be explained by risk factors known to be associated with disablement. Assessment of this relationship may enhance understanding of the public health aspects of aging. If disability is found to be independently associated with mortality, developing strategies to prevent disability would not only increase disability-free life expectancy but also total life expectancy.

We assessed the association between mortality and 3 disability measures reflecting different levels of disability severity. The linking of cross-sectional health surveys to municipal health registries in the Netherlands permitted the compilation of a large time-to-event data set with covariates measured at baseline[68]. We quantified the magnitude of the association between disability and mortality, unadjusted and adjusted for groups of risk factors.

These risk factors included distal and proximal risk factors that may influence the speed of disablement[69-72]. We used hazard ratios (HRs) and life expectancy to summarize the association between disability and mortality.

2.2. Methods

Our analyses were based on the Health Module of the *Permanent Onderzoek LeefSituatie* (Ongoing Population Survey; POLS) survey collected among the noninstitutionalized population of the Netherlands[73,74]. The POLS is an ongoing, annual cross-sectional survey conducted to provide representative information on a broad range of topics concerning the living situation of the Dutch general population. The POLS is sampled from records of a centralized municipal registry and does not include the institutionalized population. Self-reported health data are collected through face-to-face interviews and written questionnaires. The interviewer visits the participants at home, asks for informed consent, and leaves a written questionnaire. The survey response rate is typically around 60%, yielding annual net participation of approximately 10,000 individuals.

We used the POLS Health Module conducted between 2001 and 2006 because a revised questionnaire was introduced in 2001, and we had access to data that could be linked to mortality registry until 2006. We were provided with a unique data key to link individuals' survey responses with their municipal population registry records for all participants in POLS[68]. The available municipal population registries contained annual data on the event of death (yes or no) and its date in the population until December 31, 2007. Records of POLS and population registries were linked to establish the date of death during the study follow-up period. Those who were not identified in the death registry were considered to be alive at the end of the follow-up period.

The statistical key allowed the creation of a data set comprising a set of covariates measured at baseline for each person and an indicator whether that person had died before 2008 and, for those who had died, the date of death. Because the POLS consists of a new independent sample each year, we measured covariates for each person once.

Disability Measures

Disability refers to limitations in performance of socially defined roles and tasks within a sociocultural and physical environment, and to the inability to perform ADLs in a normal manner[55]. Because the concept of disability covers several types of limitations and inabilities, there is no single way of measuring it. In practice, indicators including disabilities in ADLs or disabilities in mobility are frequently used measures. We used 3 disability indicators reflecting different severity levels of disability. Both the severe disability indicators, ADL and mobility, and the mild disability indicator, defined by the Organization for

Economic Co-operation and Development (OECD)[75], were measured among persons aged 55 years and older. The ADL disability uses 5 items (eating and drinking, dressing, washing hands and face, washing oneself completely, transfer from chair). The mobility disability measure uses 5 items (moving indoors, moving outdoors, walking stairs, transfer from bed, entering or leaving room). The OECD measure uses 7 items (conversing, reading small letters, recognizing faces, biting, carrying objects, walking 400 m, bending). For each item, respondents were asked if they were able to perform the activities "without difficulty", "with minor difficulty", "with major difficulty", or "not able to perform or only with help". For each indicator, disability was defined as having at least 1 item answered "with major difficulty" or "not able to perform or only with help".

Control Variables

We examined 3 sets of control variables (confounders in the trajectory of disablement) reflecting the time-distance between the risk factors and disability according to the disablement theory, in descending order: (1) lifestyle and sociodemographic risk factors, (2) chronic diseases, and (3) other indicators of health status. The first set of risk factors included educational status, marital status, smoking status, and overweight status[69,71,72]. The second set of risk factors included both progressive and acute diseases[70]: diabetes, stroke, myocardial infarction or other severe heart disorder, any form of cancer, and diseases of the respiratory system. The third set included self-rated health status and hospitalization in the past year. More details on the variables are presented in Appendix 2A.

Data Analysis

We estimated HRs of disability on mortality for the disability measures with Cox regression models, for which the time scale was defined as a person's age[76]. Cox models are mathematical models widely used for analyzing survival data. Left truncation was applied to the age range over which the individual was not observed before the inclusion to the POLS survey[77]. We conducted all analyses separately for men and women because of differences in mortality, prevalence, and onset of disability (lower mortality, higher incidence, and higher prevalence among women than men)[78]. We controlled for the survey year in the Cox models by stratification. We used Stata 10.0 (StataCorp LP, College Station, TX) to perform the analyses.

• *Unadjusted models: comparing the mortality risk of disabled and nondisabled population*

We fitted stratified Cox models to assess the risk of mortality associated with disability status. The Cox models provide HR estimates of how much higher the mortality risk is among disabled than among nondisabled people. We fitted separate models for the 3 disability measures. The proportional hazard (PH) assumption was tested for each model by 2 types of test. First, we plotted log-log Kaplan-Meier estimates and judged whether they were parallel. When the comparison between the nondisabled and the disabled produced nonparallel curves, it indicated the PH assumption was not satisfied for the disability status variable. Second, a parametric approach for assessing the PH assumption involved goodness-of-fit tests by using Shoenfeld residuals. This approach provides large sample Z or X^2 statistics that can be computed for each variable while controlling for the other variables in the model. Nonsignificant (i.e., large) *P* values suggest that the PH assumption is reasonable, whereas small *P* values suggest that the variable being tested does not satisfy this assumption[79].

When the PH assumption is violated, there are 2 approaches to consider: using a stratified Cox procedure or using an extended Cox regression with a time-dependent variable[79]. We used the second approach because using a stratified Cox model would imply that our main parameter of interest, disability status, would have been automatically removed from the model. We therefore added an interaction term between age (time axis in our model) and disability, which was interpreted as an age-dependent effect of disability status on mortality.

• *Adjusted models: explaining the difference in mortality risk of disabled and nondisabled populations*

To assess the extent to which mortality differences between the disabled and nondisabled can be explained by risk factors, we compared the results of the unadjusted models (models without confounders) with the results of the adjusted models (models with confounders). First, we added behavioral and sociodemographic control variables to the model. Second, we added chronic diseases to the previous model. Third, we added the other indicators of health status. Although in the first 2 models, the risk factors typically precede disability, poor health status and hospitalization may already be a consequence of disability, indicating reverse causality. Because both explanations are plausible[42], these 2 risk factors were considered as a separate group. The study populations for these were limited to those for whom information was available for all risk factors. The risk factors were added in groups, and we did not investigate any interaction between the risk factors. We tested the PH assumptions in the same way as for the unadjusted models.

• *Life expectancy analysis*

We calculated life expectancy estimates of nondisabled and disabled persons at age 55 years by disaggregating national mortality rates with HR and prevalence estimates[80]. Details on mortality decomposition are presented in Appendix 2B. The resulting LE estimates can be interpreted as the remaining life expectancy of someone who remains nondisabled or disabled for the rest of his or her life.

2.3. Results

Table 2.1 provides data for the POLS sample and final study population by gender. Of the original sample of 60,399 individuals completing the POLS health module, 15,208 were aged 55 years or older. Of these, 767 men and 582 women died during 26,584 and 29,554 person-years of exposure time, respectively. The prevalence of ADL, mobility, and OECD disability was approximately 3.9%, 8.4%, and 24.5% for men and 6.3%, 15.9%, and 33.7% for women, respectively. Because our tests indicated that the PH assumption was violated in the unadjusted models of ADL and mobility disability, we added an interaction term between disability and age. Results are shown in Appendix 2C.

Table 2.1 Sample characteristics at baseline, POLS Health Survey of the Netherlands, 2001–2006

Variable	Men	Women	Total
POLS health and work module	29,558	30,841	60,399
Population aged 55+	7,287	7,921	15,208
Exposure time, person-years	26,584	29,554	56,138
Number of deaths	767	582	1,349
Disability measures, yes, %			
ADL	3.9 (7,287)	6.3 (7,920)	5.1 (15,207)
Mobility	8.4 (7,287)	15.9 (7,920)	12.3 (15,207)
OECD	24.5 (6,242)	33.7 (6,532)	29.2 (12,774)
Lifestyle and socioeconomic risk factors			
Smoking status, %			
Past daily smoker	55.8 (6,860)	27.1 (7,878)	40.5 (14,738)
Present daily smoker	22.8 (6,860)	18.3 (7,878)	20.4 (14,738)
Overweight status, %			
Normal (18.5 <BMI <= 25)	38.0 (7,116)	42.3 (7,404)	41.7 (14,520)
Overweight (25 < BMI <= 30)	48.0 (7,116)	36.4 (7,404)	43.4 (14,520)
Obese (BMI > 30)	11.6 (7,116)	14.8 (7,404)	13.8 (14,520)
Educational status: ≤ secondary school, %	45.5 (7,226)	70.7 (7,883)	58.6 (15,109)
Marital status: widowed, divorced, or never married, %	20.1 (7,287)	40.0 (7,921)	30.4 (15,208)

Table 2.1 (Continued)

Diseases			
Diabetes, %	9.9 (6,260)	9.0 (6,527)	9.4 (12,787)
Stroke, %	5.9 (6,243)	4.5 (6,515)	5.2 (12,758)
Myocardial infarction, %	15.7 (6,128)	7.5 (6,315)	11.5 (12,443)
Cancer, %	10.5 (6,224)	12.1 (6,500)	11.3 (12,724)
Respiratory disease, %	8.8 (6,283)	9.1 (6,592)	8.9 (12,875)
Morbidity measures			
Self-rated health status: poor or very poor, %	17.4 (6,230)	21.3 (6,512)	19.4 (12,742)
Hospitalized, %	10.9 (7,287)	10.4 (7,921)	10. 7 (15,208)

Notes. ADL = activities of daily living; OECD = disability as measured by the Organization for Economic Co-operation and Development indicator; BMI = body mass index (weight in kilograms divided by the square of height in meters); POLS = Permanent Onderzoek LeefSituatie (Ongoing Population Survey). Number of responses in parentheses.

Table 2.2 shows the estimated HRs of the unadjusted models. Among men, the mortality risk at age 55 was 7.85 (95% confidence interval [CI]=4.36, 14.13), 5.21 (95% CI=3.19, 8.51), and 1.87 (95% CI=1.58, 2.22) times higher for those with ADL, mobility, and OECD disability, respectively, than it was for those without disability. Among women, the corresponding ratios were 6.14 (95% CI=3.13, 12.03), 9.19 (95% CI=5.47, 15.44), and 1.61 (95% CI=1.29, 2.01).

The HR associated with ADL and mobility disability significantly decreased by approximately 0.96 with every year increase of age. By age 80 years, men with ADL or mobility disability faced risks of dying 2.92 (95% CI=2.36, 3.61) and 2.48 (95% CI=2.09, 2.95) times higher, respectively, than did those who did not have such disability. Corresponding HRs among women were 2.51 (95% CI=2.03, 3.10) and 2.28 (95% CI=1.93, 2.70). We found no significant age gradient for OECD disability, and the level of HR was lower.

Table 2.2 Mortality risk by baseline disability in terms of hazard ratios in the unadjusted models, separately for men and women: POLS Health Survey of the Netherlands, 2001–2006

	HR	95% CI	P value	N
		Men		
ADL (at age 55 years)	7.85	4.36, 14.13	< 0.000	7,287
ADL × age	0.96	0.94, 0.98	< 0.000	
Mobility (at age 55 years)	5.21	3.19, 8.51	< 0.000	7,287
Mobility × age	0.97	0.95, 0.99	< 0.01	
OECD	1.87	1.58, 2.22	< 0.000	6,242
		Women		
ADL (at age 55 years)	6.14	3.13, 12.03	< 0.000	7,920
ADL × age	0.96	0.94, 0.99	< 0.01	
Mobility (at age 55 years)	9.19	5.47, 15.44	< 0.000	7,920
Mobility × age	0.95	0.93, 0.96	< 0.000	
OECD	1.61	1.29, 2.01	< 0.000	6,532

*Notes.*ADL = activities of daily living; CI = confidence interval; HR = hazard ratio; OECD = disability as measured by the Organization for Economic Co-operation and Development indicator; POLS = Permanent Onderzoek LeefSituatie (Ongoing Population Survey). P values are 2-sided.

Table 2.3 Mortality risk by baseline disability in terms of hazard ratios in the adjusted models, separately for men and women: POLS Health Survey of the Netherlands, 2001–2006

	Unadjusted				**Adjusted**									
					For Socio-economic Factors and Health Behaviours [a] (1)			(1) + For Chronic Diseases [b] (2)			(2) + For Health Status [c] (3)			
	HR (at mean age = 67 years)	95% CI	*P* value	N	HR	95% CI	*P* value	HR	95% CI	*P* value	HR	95% CI	*P* value	N
	Men													
ADL	4.88	3.49, 6.82	< 0.001	7,287	2.88	2.09-3.97	< 0.001	2.16	1.55-3.02	< 0.001	1.62	1.15-2.30	0.006	5,506
Mobility	3.65	2.76, 4.81	< 0.001	7,287	2.51	1.99-3.16	< 0.001	1.97	1.55-2.49	< 0.001	1.57	1.21-2.02	0.001	5,506
OECD	1.87	1.58, 2.22	< 0.001	6,242	1.74	1.44-2.11	< 0.001	1.48	1.22-1.80	< 0.001	1.27	1.04-1.56	0.022	5,506
	Women													
ADL	3.99	2.65, 6.01	< 0.001	7,920	2.71	1.97-3.72	< 0.001	2.31	1.67-3.20	< 0.001	1.87	1.32-2.64	< 0.001	5,795
Mobility	4.70	3.46, 6.39	< 0.001	7,920	2.32	1.80-2.99	< 0.001	1.97	1.51-2.57	< 0.001	1.67	1.25-2.23	0.001	5,795
OECD	1.61	1.29, 2.01	< 0.001	6,532	1.55	1.21-1.99	< 0.001	1.37	1.06-1.77	< 0.01	1.17	0.89-1.54	> 0.05	5,795

Notes. ADL = activities of daily living; CI = confidence interval; HR = hazard ratio; OECD = disability as measured by the Organization for Economic Co-operation and Development indicator; POLS = Permanent Onderzoek LeefSituatie (Ongoing Population Survey). P values are 2-sided.

a Socioeconomic factors and health behaviors included smoking status, overweight status, educational status, and marital status.

b Major chronic diseases included diabetes, stroke, myocardial infarction, cancer, and respiratory disease.

c Health status indicators included self-rated health status and previous hospitalization.

We assessed the extent to which mortality differs between disabled and nondisabled people by adding groups of risk factors to the models. First, we added behavioral and sociodemographic control variables (Table 2.3: column adjusted for socioeconomic factors and health behaviors). The risk of death by disability status was somewhat explained by marital, education, and smoking status in men but only by smoking status in women. Second, we added 5 key chronic diseases to the previous model (Table 2.3: column adjusted for chronic diseases). All were significant predictors of mortality (except for myocardial infarction among women), which substantially reduced the magnitude of the HRs, especially for ADL and mobility disability. Finally, when we added self-reported health status and previous hospitalization to the explanatory models (Table 2.3: column adjusted for health status), both morbidity measures were significant predictors of mortality, which further explained the risk of death by disability status. However, the HRs still indicated a significantly higher mortality risk for disability in ADL (men: HR=1.62; 95% CI=1.15, 2.30; women: HR=1.87; 95% CI=1.32, 2.64) and for disability in mobility (men: HR=1.57; 95% CI=1.21, 2.02; women: HR=1.67; 95% CI=1.25, 2.23) than for nondisabled persons.

Figure 2.1 presents the remaining life expectancies of nondisabled and disabled populations for each disability measure. Men who are free of ADL, mobility, or OECD disability at age 55 years and older can expect to live 25.2 (95% CI=24.8, 25.8), 25.8 (95% CI=25.3, 26.4), and 26.3 (95% CI=25.6, 27.0) years, respectively. By contrast, men who have ADL, mobility, or OECD disability face substantially shorter life expectancies: i.e., 15.9 (95% CI=13.9,18.0), 17.4 (95% CI=16.0,18.8), and 20.9 (95% CI=20.0, 21.8) years, respectively. Women without ADL, mobility, or OECD disability have a life expectancy of 29.3 (95% CI=28.9, 29.9), 30.2 (95% CI=29.5, 31.0), and 29.9 (95% CI=28.9, 31.0) years, respectively, whereas those with such disabilities can expect to live for substantially less time: 21.3 (95% CI=19.2, 23.5), 23.5 (95% CI=22.1, 24.9), and 26.3 (95% CI=25.5, 27.2) years, respectively. Much of these differences in LE are explained by risk factors. After we adjusted for all the covariates, differences in life expectancy associated with disability decreased to approximately 4 years for disability in ADL and mobility. For OECD disability, the differences become very small.

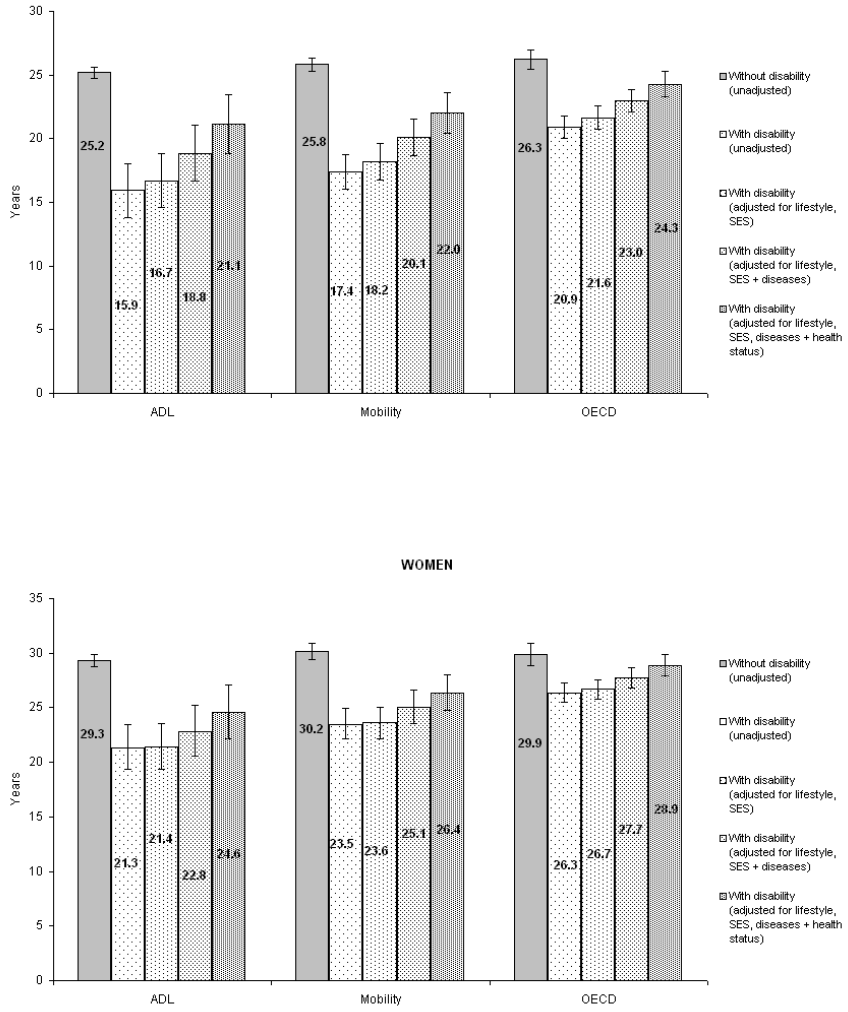


Figure 2.1 Remaining life expectancy at age 55 years for nondisabled and disabledpopulations of (a) men and (b) women by disability measure: POLS Health Survey of the Netherlands, 2001–2006.
Note. ADL = activities of daily living; OECD = Organization for Economic Co-operation and Development indicator; SES = socioeconomic status.

2.4. Discussion

We assessed the relationship between disability and all-cause mortality in the general population for 3 different disability measures. We compared disabled and nondisabled populations in terms of relative mortality risk. People with disabilities were exposed to higher risk of death at any age. A major part of the excess risk of death associated with disability could be explained by major diseases and other risk factors typically preceding the presence of disability; however, for severe disability measures a substantial unexplained proportion remained.

As suggested by the disablement process, our empirical results confirmed that although ADL and mobility disability were strong predictors of death, OECD disability was only a weak predictor. Furthermore, adjustments for diseases with significant mortality risk explained a major part of the association. We speculate that the remaining association between disability and mortality may be explained by residual confounding or is an independent effect of disability. The risk factors that were not included in our models because of lack of data and that are known to be related to disability and death include disease severity[81], injuries[82], multiple sclerosis[83], motor neuron disease[84], or depression[85]. Poor health status and previous hospitalization in our model may capture these unobserved characteristics. If they did, our analyses would imply that the disabled face higher risk of death than the nondisabled do, even after controlling for these other indicators of health status, and this finding can be interpreted as an indication that disability has an independent effect on mortality.

To assess the validity of the changing HR with age, we searched studies with model specifications similar to ours. We found only 1 study that used age as a timescale: it described the association between ADL disability and mortality among a small elderly population[49]. Similar to our results, the authors found that the effect of disability on mortality decreased with age (with control for a limited number of risk factors). At younger ages, ADL disability was associated with increased risk of death compared with that at older ages (for men, HR=1.80 at age 80 years and HR=1.15 at age 90 years; for women, HR=3.53 at age 80 years and HR=1.86 at age 90 years).

Strengths and Limitations

There are some limitations regarding the data that need to be acknowledged. First, disability data were self-reported, which can result in either under- or overreporting of disability, which, in turn, may bias the outcomes. Although we cannot exclude this possibility, measurement of functional limitations by self-report are reported to be consistently associated with performance and reflect similar assessment of function[86].

Nonresponse might have been a problem if the disabled were less likely to respond to the survey. No study has explored the relationship between nonresponse and disability status in the POLS survey before; therefore, we assessed whether missing disability score was associated with participants' health status. To do this, we used the POLS Basis module in which self-rated health status was elicited during face-to-face interviews but no other information on health was recorded. In terms of self-reported health status, the POLS Basis module was a larger sample than was the POLS Health module. We linked individual records of the POLS Health survey to records of self-assessed health status from the POLS Basis and created a data set in which the disability variable was a dichotomous indicator variable (missing or nonmissing). We found that the probability of exclusion because of missing values in a given disability measure was weakly related to self-reported health status (i.e., the worse the health status, the higher the probability of not completing the questionnaire). The odds ratio of being excluded from the analysis for those reporting very poor health status was around 1.2 for men and slightly higher for women. A recent study that investigated the potential consequences of participation bias on associations between exposures and outcomes concluded that the nonparticipation of those with poorer health is probably a greater threat to the validity of prevalence studies than to studies of associations between exposures and outcomes[87]. These findings imply that differential nonresponse could not have strongly biased our estimates.

Several studies have shown that individuals might change their disability status during the observation period[88,89]. By contrast, the present study approaches disability as a static condition as observed at enrollment, ignoring the possibility that disability may have begun or diminished over the follow-up period. The static disability measure was admittedly a limitation of our study. The probability of such misclassification is higher if the follow-up time is longer, given constant incidence and recovery intensities. Direct evaluation of the level of misclassification was not possible, but we could, however, test whether disability had a different effect on mortality among persons who were classified for a longer time before the event took place. We fitted additional Cox models, in which we added an interaction term of the standardized POLS year variable and the disability measure variable to each model. The HR of such interaction term larger than 1 could be an indication of a weaker effect of disability on mortality among those recruited earlier than among those who joined the POLS later. Such a situation could occur if many individuals became disabled during the follow-up period. After we assessed the significance of interaction terms in the models for each disability measure ($P>0.5$), we concluded that misclassification was unlikely to have biased our results.

A main strength of this study was the annually collected data from a large representative survey of community-dwelling older people followed for up to 7 years. Measures of disability status were elicited by an identical survey design and survey questionnaire for all participants in each year, allowing the compilation of an extensive data set. In addition, recall bias was avoided because disability status was elicited on the spot. We are not aware of any study that has used such a large-scale record-linked data set to analyze the risk of disability on mortality.

A further strength is that we used 3 different disability measures. Different types of disability capture different severity levels[90], which result in different trajectories after onset. Accordingly, the severity of disability influences the mortality risk[91] and the type or duration of subsequent care[92]. More severe disability implies an increased demand for formal care (e.g., nursing homes), although this demand becomes shorter because the associated mortality risk is high. Overall, more severe disability is likely to be associated with a greater need for institutional care during a shorter period of time.

Conclusions

Our results indicate that disabled persons face a higher mortality risk than the nondisabled, especially those who are severely disabled. Whereas for mild disability the risk difference can be explained by diseases and other risk factors related to sociodemographic status and lifestyle, we cannot rule out that more severe disabilities have an independent effect on mortality.

Appendix 2A Detailed description of variables

Name	Description	Possible answers	Coding
ADL	eating and drinking, (un)dressing, washing hands and face, washing oneself completely, transfer from chair	1) without difficulty 2) with a little difficulty 3) with severe difficulty 4) only with help	someone is disabled if answer on at least one of the items was 'severe difficulty' or 'only with help'
Mobility	moving indoors, moving outdoors, walking stairs, transfer from bed, entering /leaving room	1) without difficulty 2) with a little difficulty 3) with severe difficulty 4) only with help	someone is disabled if answer on at least one of the items was 'severe difficulty' or 'only with help'
OECD	following / driving a conversation, reading small letters, recognising a face, biting, carrying an object, walking 400ms, bending	1) without difficulty 2) with a little difficulty 3) with severe difficulty 4) not able to perform	someone is disabled if answer on at least one of the items was 'severe difficulty' or 'not able to perform'
<u>Morbidity</u>			
Self-reported bad health	self-perceived health status	1) excellent 2) very good 3) fine 4) moderate 5) bad	Someone had bad health status if the answer was 'bad'
Hospitalised in the previous year	admission to hospital in the previous year	1) yes 2) no	

Name	Description	Possible answers	Coding
Selected diseases			
Diabetes	having diabetes	1) yes 2) no	
Stroke	had a stroke	1) yes 2) no	
Infarct	had heart infarct or other severe heart disorder	1) yes 2) no	
Cancer	had at least one cancer	1) yes 2) no	
Asthma / chronic non-specific lung disease	having asthma, chronic bronchitis or chronic non-specific lung disease	1) yes 2) no	
Other risk factors			
Smoking status	Question: do you smoke?	1) yes, daily 2) yes, sometimes 3) used to smoke daily 4) used to smoke sometimes 5) never smoked	smoker if the answer was "yes, daily" ex-smoker = used to smoke daily non-smoker = 2), 4) or 5)
Overweight status	overweight index	1) severe underweight 2) normal 3) overweight 4) obese	dummy recoding
Educational level	highest achieved educational level	1) low education 2) lbo 3) mavo 4) havo, vwo, mbo 5) hbo, university degree	a person was low educated if his educational level was 1), 2) or 3)
Marital status	civic status	1) married 2) widowed 3) divorced 4) never married	dummy recoding: married versus other

Appendix 2B Disentangling mortality rates of the whole population into mortality rates of non-disabled and disabled populations

Raw total mortality rates for the period 2001-2007 were calculated for each age and sex based on the total counts of mortality and exposure time during of interest, as published in Human Mortality Database (HMD). Age- and sex-specific prevalence of disability measures were calculated based on the number of disabled persons and the number of participants in the study population during the same period. Both total mortality rates and prevalence of disability were smoothed by P-splines method in R.

Combining smoothed total mortality rates, hazard ratios and smoothed prevalence of disability mortality rates were calculated for non-disabled and disabled populations by the following formulas:

$$m_x^{(nd)} = \frac{m_x}{HR_x \times p_x^{(d)} + \left(1 - p_x^{(d)}\right)}$$
$$m_x^{(d)} = m_x^{(nd)} \times HR \qquad (1)$$

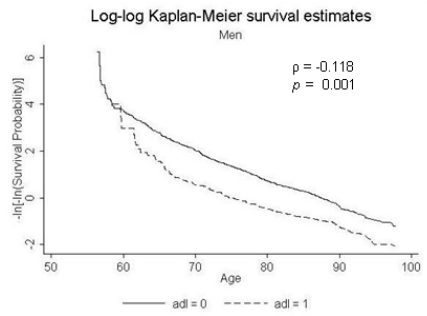
, where $m_x^{(nd)}$, $m_x^{(d)}$, HR_x and $p_x^{(d)}$ indicated the mortality rate of non-disabled, mortality rate of disabled, estimated hazard ratio and prevalence of disability at age x, respectively.

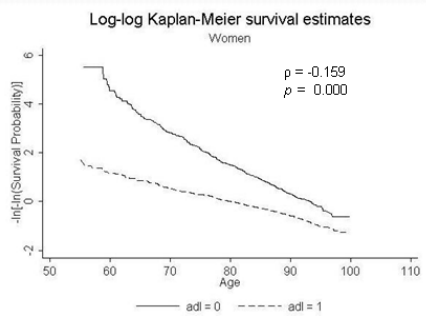
LEs of non-disabled and disabled populations were calculated using survival probabilities, which were transformed from the state-specific mortality rates:

$$q_x^{(.)} = 1 - e^{-m_x^{(.)}}$$

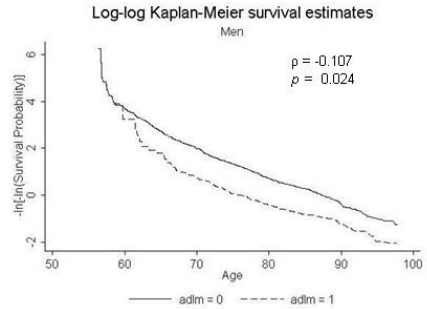
Appendix 2C Log-log Kaplan-Meier survival estimates of the disability measures in univariate analyses, men and women (ADL, Mobility, OECD)

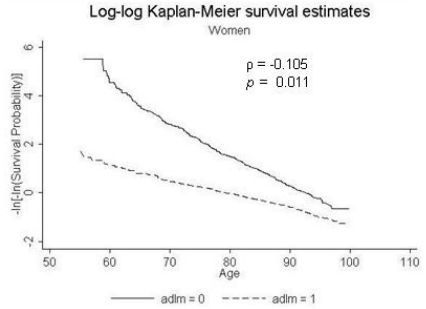
ADL



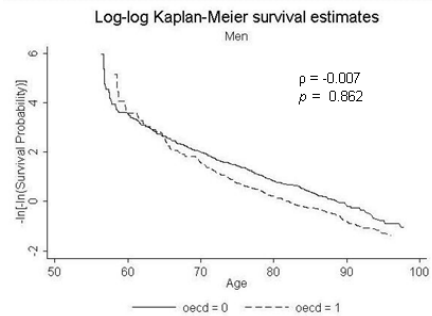


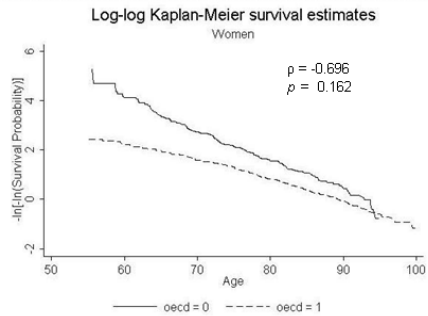
Mobility





OECD





Chapter 3

Life expectancy and life expectancy with disability of normal weight, overweight, and obese smokers and nonsmokers in Europe

Majer IM, Nusselder WJ, Mackenbach JP, Kunst AE

The goal of this study was to estimate life expectancy (LE) and LE with disability (LwD) among normal weight, overweight, and obese smokers and nonsmokers in Western Europe. Data from four waves (1998-2001) of the European Community Household Panel (ECHP) were used; a standardized multipurpose annual longitudinal survey. Self-reported health and socioeconomic information was collected repeatedly using uniform questionnaires for 66,331 individuals in nine countries. Health status was measured in terms of disability in daily activities. Multistate Markov (MSM) models were applied to obtain hazard ratios (HRs) and age-specific transition rates according to BMI and smoking status. Multistate life tables were computed using the predicted transition probabilities to estimate LE and LwD. Significant associations were observed between disability incidence and BMI (HR=1.15 for overweight, HR = 1.64 for obese, compared to normal weight). The risk of mortality was negatively associated with overweight status among disabled (HR=0.77). Overweight people had higher LE than people with normal-weight and obesity. Among women, overweight and obese nonsmokers expect 3.6 and 6.1 more years of LwD than normal weight persons, respectively. In contrast, daily smokers expect lower LE but a similar LwD. The same patterns were observed among people with high education and those with low education. To conclude, daily smoking is associated with mortality more than with disability, whereas obesity is associated with disability more than with mortality. The findings suggest that further tobacco control would contribute to increasing LE, while tackling the obesity epidemic is necessary to prevent an expansion of disability.

Obesity (Silver Spring). 2011 Jul;19(7):1451-9.

3.1. Introduction

Life expectancy (LE) has been increasing for decades[30], but whether and to what extent disability-free life expectancy will increase is not entirely clear[93,94]. In the past, advances in medical technologies and their increased availability have contributed to the independence of older people[95]. However, any optimism may be diminished by the impact of obesity[96]. The worldwide epidemic has already resulted in a doubling of the prevalence of obesity in Western and Westernizing countries[97].

Early studies of U.S. populations found large effects of overweight and obesity (referred to the summary term "overweight status" below) on both premature death risk and on disability prevalence[98,99]. However, these effects reflect life histories of older cohorts and there is evidence that the excess risk of higher body mass index (BMI) on mortality has diminished over time[100,101]. Studies using more recent data from the U.S. showed smaller impacts on LE, but still large effects on disability-free life expectancy[102,103].

Results for Europe might be different from those of the U.S. though, due to different epidemiological profiles between these regions. In Southern Europe for example, lower cardiovascular mortality rates have been recorded than in the U.S, despite the relatively high prevalence of classical risk factors[104]. Consequently, one might hypothesize that the diminishing effect of high BMI on mortality over time may vary across the Atlantic.

Unfortunately the evidence on the impact of overweight status on life expectancy and the burden of disability in Europe is largely incomplete. Previous studies were based on small sample sizes and were restricted to single countries[105,106]. Consequently, a comprehensive picture on the population health associated with overweight status in Europe is still missing.

The aim of this study was to assess how much the overweight status is associated with longevity and the burden of disability in Western Europe. To measure this relationship, life expectancy and life expectancy with disability (LwD) were estimated by overweight status and smoking status. Furthermore, it was investigated in subgroups of men and women as well as of low and high educated.

Life expectancy and life expectancy with disability are both aggregate measures of population health referring to a certain period of time. LwD has similar interpretation to (total) LE but it refers to the number of years that people expect to live with disability. LE and LwD (and their difference, "disability-free life expectancy") are routinely used aggregate indicators of population health for a certain calendar period[10].

The European Community Household Panel (ECHP) was used as data source. The main advantages of ECHP were that it provided comparable information on health and mortality for several European countries, and that it gave sufficient statistical power to obtain precise estimates for disability incidence and recovery rates, and for life table calculations for specific risk factor groups.

3.2. Methods and procedures

Data

The ECHP is a standardised multi-purpose annual longitudinal social survey carried out at the level of the European Union between 1994 and 2001. It is centrally designed and coordinated by the Statistical Office of the European Communities (Eurostat), and includes demographics, labour force behaviour, income, health, education, training, housing, and migration. The data were collected by the National Statistical Institutes or research centres of the participating countries using uniform random sampling design and common blueprint questionnaires. Although Eurostat left the National Institutes of Statistics (NIS) free to organize the data collection, and national reporting on the survey organization is lacking (making assessment of data quality difficult), data checks, imputation and weighting were done centrally to maximise data comparability. The ECHP is intended to be both cross-sectionally and longitudinally representative with respect to the national household populations. For more information about the design and the data procedures of the ECHP we refer to an extensive review of Peracchi[107].

Data from the fifth wave of 1998 onwards until 2001 were used for the current study, as 1998 was the first year that height, weight and smoking status were asked from the participants. Countries for which mortality data (the Netherlands), disability data (Luxembourg) or BMI data (France) was not available were omitted from the study. Similarly, data from Germany and the UK had to be left out because the original ECHP data were replaced by data from national surveys (German Social Economic Panel and British Household Panel Survey) which did not contain information on smoking and BMI.

Information on non-response rates at baseline and cumulative retention percentages, i.e. the cumulative percentages of individuals retained until the fourth wave of the panel are presented in Appendix 3A. There were differences between countries, with high retention percentages in Italy (83%) and Portugal (87%), while samples in Ireland, Denmark and Spain suffered somewhat higher attrition. Respondents were followed over a maximum of four years. Since respondents could die, while new individuals could also join the survey in calendar years later than 1998, the average follow-up time was less than the maximum achievable. However, most people (85%) participated in all four waves. Detailed informa-

tion on the distribution of age at entry, number of deaths as well as average follow-up time is presented in Table 3.1.

Table 3.1 Characteristics of the study population

	Number of individuals [a]	Mean age (range)	Mean follow-up time	Overweight (%)	Obese (%)	Smoker [b] (%)
			Men			
Finland	3,179	44.7 (16.8-89.8)	2.4	42.2	10.9	24.8
Denmark	1,694	46.5 (16.8-89.6)	2.8	38.6	9.6	35.8
Ireland	2,412	45.2 (16.7-90.2)	2.5	41.7	7.8	25.9
Austria	2,721	45.4 (15.3-89.6)	2.8	41.9	11.3	29.5
Belgium	2,072	46.2 (16.3-88.7)	2.7	39.2	10.1	29.9
Greece	3,985	49.1 (17.3-90.3)	2.0	51.4	9.9	44.2
Italy	6,735	45.1 (16.8-89.7)	2.6	38.9	8.2	31.1
Spain	5,212	44.9 (15.9-89.7)	2.8	43.0	13.2	37.9
Portugal	4,628	46.8 (16.9-89.8)	2.8	43.5	9.5	28.6
Sum	32,638	45.9 (15.3-90.3)	2.6	42.3	10	32.5
			Women			
Finland	3,112	45.8 (16.8-89.5)	2.4	28.7	13.2	14.9
Denmark	1,706	47.1 (16.7-89.4)	2.8	26.5	9.4	33.8
Ireland	2,375	46.2 (16.7-89.6)	2.5	28.1	8.5	23.9
Austria	2,814	47.9 (15.3-89.8)	2.9	30.2	10.8	15.9
Belgium	2,278	47.5 (16.4-89.4)	2.7	24.6	11.1	20.7
Greece	4,389	50.6 (17.4-90.4)	2.0	35.7	10.0	14.7
Italy	6,742	47.1 (16.8-89.8)	2.6	24.8	7.4	13.2
Spain	5,262	47.7 (15.9-89.7)	2.8	28.9	13.6	20.2
Portugal	5,015	49.6 (16.9-89.9)	2.8	32.5	11.1	4.8
Sum	33,693	47.9 (15.3-90.4)	2.6	28.9	10.4	16.0

Notes: [a] The number of individuals refers to those who participated in at least two waves of ECHP survey between 1998 and 2001

[b] Those who are not daily smoker could be never smokers and former smoker, treating these two groups separately.

Indicators

All individuals were asked if they were hampered in their daily activities by any physical or mental health problem, illness or disability in each wave. Positive response were categorised in two classes of severity: "Yes, to some extent" or "Yes, severely"[108]. For this study we considered them as a single disabled category. Persons who were lost to the ECHP panel because of moving to an institution were also considered as 'disabled' for the remainder of the study period.

The BMI variable was recoded into four categories: (1) Underweight: BMI ≤ 18.5, (2) Normal weight: 18.5 < BMI ≤ 25, (3) Overweight: 25 < BMI ≤ 30, (4) Obese: BMI > 30. After preliminary analysis, underweight persons were excluded from further analysis because of the small number of respondents and the high prevalence of disability in this category. Moreover this group is less relevant for the purpose of our study as their prevalence of disability is commonly explained as a result of weight loss caused by ill-health, rather than a causal effect of underweight on disability.

Smoking status was classified into three categories: (1) Current daily smokers, (2) Former (daily) smokers, and (3) Never (daily) smokers. The latter included those who smoke occasionally or who used to smoke occasionally. With regard to former smokers, health problems may increase the chances of smoking cessation; therefore the association of health with former smoking may in part reflect a selection effect. Additionally, the group of former smokers is a heterogeneous group in terms of time since quitting. As a result, the interpretation of results for this group is less straightforward. Therefore, former smokers were excluded from the presentation of the detailed results.

The distribution of overweight and smoking status in the study population is presented in Table 3.1.

The level of completed education at the fifth wave was used to measure education status. Individuals were divided into three groups according to their level of educational attainment based on the International Standard Classification of Education[109]: 1) lower secondary education or lower; 2) upper secondary education; and 3) tertiary education, which includes higher vocational and university education. For this study we considered people with upper secondary or tertiary education as those with high education.

Data analysis

We employed a multi-state Markov (MSM) model, which is often used to describe the process in which individuals move through health states over time[110]. By fitting MSM models to

longitudinal data one is able to estimate hazard or transition rates. The association between variables and a particular transition rate was modelled assuming proportional hazards.

We defined three health states, non-disabled, disabled and dead, and four possible transitions between the health states: incidence (from non-disabled to disabled), recovery (from disabled to non-disabled) and state-specific mortality (from both non-disabled and disabled). We merged data from the countries after preliminary analyses showed only minor cross-national differences in associations with overweight and smoking. Transition rates were estimated on the pooled dataset adjusted for age, sex, overweight status and smoking status. The advantage of preparing estimates using the data of all nine countries together was the very large number of observations that ensured sufficient power in estimating the age profiles of disability incidence and recovery.

To detect significant and meaningful interactions and to obtain estimates of transition rates that accurately describe the data, various models were considered and evaluated. Models including two- or three-way interaction terms between sex, overweight and smoking status were tested. No significant interactions were found between smoking and overweight status. Akaike Information Criteria (AIC) values indicated that the best model fit was achieved by including two-way sex-interactions. In additional analyses according to educational subgroup, we included interaction terms between education and overweight and smoking status. Age-sex interaction terms were included in both models.

Because mortality cases were under-registered in the ECHP in most of the countries, the mortality rates were adjusted to the pooled level (pooled across country and time) in four steps. First, using national statistics, mortality rates were calculated over the period 1998-2001 for each age and sex. Second, they were transformed into mortality rates of non-disabled and disabled assuming that 1) the age and sex-specific mortality rates in the overall (i.e. mixed non-disabled-disabled) population are the weighted average of mortality rates of non-disabled and disabled populations, with the proportion of non-disabled and disabled respectively as weights estimated from the ECHP, and 2) that the ratio between the mortality rate of disabled and non-disabled populations is equal to the hazard ratio as estimated from the ECHP data[80]. Third, rescaling factors were calculated to specify how much age- and sex-specific mortality rates estimated from the ECHP had to be scaled to make them consistent with the estimates from step two. Fourth, all predicted mortality rates were multiplied by the corresponding rescaling factors, assuming that under-representation of mortality was the same for each overweight and smoking status combination. A more formal explanation of the decomposition is shown in the Appendix 3B.

Multi-state life tables were used to estimate LE and LwD at the age of 16. The empirical input for the multi-state life tables were the transition rates described above, after converting them into probabilities (assuming that hazard rates were constant over a life year). Once a life table was set up, probabilistic sensitivity analysis[111] was performed to estimate confidence intervals (CIs) around the LEs and LwD. For this, random log-hazard ratios were drawn from a multivariate normal distribution, which was defined by the natural logarithm of the regression coefficients and their variance-covariance structure. The corresponding transition rates and life expectancies were calculated for each of the 1000 draws. The 25th and 975th of the latter ordered values indicated the 2.5% and 97.5% boundaries of the CIs. All MSM analyses were performed in R[112], whereas life table calculations were carried out in Microsoft Excel. Due to technical limitations of the MSM package, taking into account ECHP sampling weights was not possible.

3.3. Results

The hazard ratios of transitions by overweight status and smoking status of the main effect model are given in Table 3.2. The risk of disability onset was moderately higher for overweight (HR=1.15, CI: 1.10-1.20) and considerably higher for obese (HR=1.64, CI: 1.54-1.74) than for normal weight persons. Conversely, overweight showed to be protective for dying among those who were disabled (HR=0.77, CI: 0.66-0.90). Daily smoking was strongly associated with early death among non-disabled (HR=1.68, CI: 1.29-2.19), weakly associated with disability onset, and not associated with recovery. The combined effect of two risk factors can be obtained as well by multiplying the corresponding hazard ratios. For example, the hazard ratio of overweight and smoking on disability incidence could be calculated as follows: 1.09*1.15 = 1.25 (compared to a person who has normal weight and is non-smoker).

The hazard ratios of incidence and recovery of the models assessing interaction with sex and educational level are given in Table 3.3. For men, disability incidence was not associated with overweight (HR=1.00, CI: 0.94-1.07), while it was associated with obesity (HR=1.35, CI: 1.23-1.48). Among women both overweight and obesity were related to an increased risk of disability onset (overweight: HR=1.28, CI: 1.20-1.36; obese: HR=1.87, CI: 1.72-2.03). With regard to recovery from disability no substantial differences between the risk factors were found among men or women; however, the negative association between obese women and recovery, while marginal, is noteworthy (HR=0.92, CI: 0.85-0.99).

Table 3.2 Hazard ratios associated with disability incidence, recovery from disability and state-specific mortality by overweight status and smoking status

	Hazard ratios [a]			
	(CI) (number of transitions)			
	Normal weight + Never-smoker	Normal weight + Daily smoker	Overweight + Never-smoker	Obese + Never-smoker
Disability incidence	1.00	1.09*	1.15*	1.64*
		(1.04, 1.15)	(1.10, 1.20)	(1.54, 1.74)
	(2,955)	(1,043)	(2,742)	(1,071)
Recovery from disability	1.00	1.01	1.03	0.96
		(0.95, 1.07)	(0.98, 1.08)	(0.90, 1.02)
	(2,619)	(921)	(2,539)	(973)
Mortality of non-disabled	1.00	1.68*	0.81	1.06
		(1.29, 2.19)	(0.63, 1.03)	(0.73, 1.52)
	(162)	(55)	(121)	(43)
Mortality of disabled	1.00	1.04	0.77*	0.82
		(0.81, 1.34)	(0.66, 0.90)	(0.65, 1.03)
	(257)	(49)	(190)	(73)

Notes:

* $p < 0.05$

[a] Derived from a multi-state Markov (MSM) model that included age, sex, age*sex, overweight, obese, past daily smoking, daily smoking
Hazard ratios are interpreted at average age

Table 3.3 Hazard ratios associated with disability incidence and recovery from disability by overweight status and smoking status according to sex and educational level

	Number of transitions	Normal weight + Never-smoker	Normal weight + Daily smoker	Overweight + Never-smoker	Obese + Never-smoker
			Hazard ratio of incidence		
Baseline model [a]	10,244	1.00	1.09*	1.15*	1.64*
			(1.04, 1.15)	(1.10, 1.20)	(1.54, 1.74)
Male [b]	4,602	1.00	1.15*	1	1.35*
			(1.07, 1.24)	(0.94, 1.07)	(1.23, 1.48)
Female [b]	5,642	1.00	1.05	1.28*	1.87*
			(0.97, 1.16)	(1.20, 1.36)	(1.72, 2.03)
Low educated [c]	7,307	1.00	1.11*	1.13*	1.54*
			(1.04, 1.18)	(1.07, 1.19)	(1.43, 1.66)
High educated [c]	2,937	1.00	1.13*	1.12*	1.72*
			(1.03, 1.24)	(1.03, 1.22)	(1.53, 1.94)
			Hazard ratio of recovery		
Baseline model [a]	9,171	1.00	1.01	1.03	0.96
			(0.95, 1.07)	(0.98, 1.08)	(0.90, 1.02)
Male [b]	4,057	1.00	1.08	1.04	1.01
			(0.99, 1.17)	(0.97, 1.11)	(0.92, 1.12)
Female [b]	5,114	1.00	0.93	1.03	0.92*
			(0.85, 1.02)	(0.97, 1.10)	(0.85, 1.00)
Low educated [c]	6,524	1.00	1.02	1.07*	1.00
			(0.95, 1.10)	(1.01, 1.13)	(0.92, 1.07)
High educated [c]	2,647	1.00	1.01	0.96	0.88*
			(0.92, 1.11)	(0.88, 1.05)	(0.78, 0.99)

Notes:

* $p < 0.05$

[a] Derived from a multi-state Markov (MSM) model that included age, sex, age*sex, overweight, obese, past daily smoking, daily smoking

[b] Derived from a multi-state Markov (MSM) regression model that included: age, sex, age*sex, overweight, obese, past smoking, daily smoking, overweight*sex, obese*sex, past smoker*sex, daily smoker*sex

[c] Derived from a multi-state Markov (MSM) regression model that included: age, sex, age*sex, education, overweight, obese, past smoking, daily smoking, overweight*education, obese*education, past smoker*education, daily smoker*education

Hazard ratios are interpreted at average age

Educational level was strongly associated with the transition rates. Low educated people faced higher risk of disability incidence, high state-specific mortality and lower chance of recovery from disability. However, the relationship between the risk factors and disability incidence and recovery were similar for high and low educational level people (results not shown).

Table 3.4 shows estimates of LE, LwD and the ratio of LwD to LE by overweight status and smoking status. A positive association between overweight and LE was found for both non-smokers and smokers, as well as for men and women. For example, among non-smokers, the difference in LE relative to normal weight was 2.0 years for men and 2.9 years for women. Conversely, obesity was negatively associated with LE, although much more clearly for women (-1.6 years) than for men (-0.2 years). Daily smoking was inversely related to LE.

Table 3.4 Total life expectancy and life expectancy with disability by smoking and overweight status at the age of 16

	Never-smoker			Daily smoker		
	Normal weight	Overweight	Obese	Normal weight	Overweight	Obese
			Men			
LE	60.4	62.4	60.2	56.9	58.8	56.7
	(60.2, 60.5)	(62.2, 62.5)	(60.0, 60.4)	(56.7, 57.0)	(58.7, 59.0)	(56.6, 56.8)
LwD	9.5	10.1	11.8	8.9	9.5	11.1
	(9.1, 9.9)	(9.7, 10.5)	(11.1, 12.6)	(8.4,9.4)	(9.0, 10.0)	(10.3, 11.9)
LwD / LE	15.70%	16.20%	19.60%	15.70%	16.10%	19.60%
			Women			
LE	65.5	68.4	63.9	63.2	66.4	61.9
	(65.4, 65.7)	(68.2, 68.6)	(63.7, 64.1)	(62.9, 63.4)	(66.2, 66.7)	(61.6, 62.2)
LwD	11.9	15.5	18.0	12.0	15.7	18.3
	(11.5, 12.3)	(15.0, 15.9)	(17.3, 18.8)	(11.3, 12.7)	(14.8, 16.7)	(17.2, 19.5)
LwD / LE	18.10%	22.60%	28.20%	19.00%	23.70%	29.60%

Notes:
Derived from an multi-state Markov regression model that included:
age, sex, age*sex, overweight, obese, past daily smoking, daily smoking, overweight*sex, obese*sex, past daily smoker*sex, daily smoker*sex
LE: Total life expectancy, LwD: Life expectancy with disability

Smokers had 3.5 and 2.3 years lower LE than non-smokers, among normal weight men and women, respectively.

The relationship between overweight status and LwD was different: the higher the BMI category the more years are expected to be spent with disability. Normal weight, overweight and obese non-smoker men can expect to live 9.5, 10.1 and 11.8 years with disability, respectively. Smokers expect lower LE but the same number of years with disability. In general, overweight status was the main driver of the proportion of LE spent with disability. For example, normal weight, overweight and obese non-smoker women expect to live 11.9 (18.1% of LE), 15.5 (22.6%) and 18.0 (28.2%) years with disability. These general patterns were observed for both sexes despite some variation between men and women.

Figure 3.1 presents LE and LwD estimates by overweight and smoking status among low and high educated. The positive association between overweight and LE was observed among both high educated and among low educated. For both high and low educated people, the previously observed patterns were found: in higher BMI categories people expect to live more years in disability, whereas smoking is not related to the proportion of life spent with disability. The magnitude of LwD and its proportions of LE were considerably higher for low educated than for high educated persons due to the association between educational level and both mortality and disability burden.

3.4. Discussion

This study quantified the relationship of overweight status with longevity and the burden of disability in Western-Europe in terms of LE and LwD. Overweight people can expect to live slightly longer than those with normal weight, which – suggested by other studies – might be a consequence of the protective effect of overweight on mortality in disabled populations. In contrast, overweight and obese people can expect to live 3.6 and 6.1 more years with disability though, respectively. Smoking had a different relationship with LE and LwD, as it was associated with lower LE but with an unchanged LwD. Similar patterns were observed both among men and women, and among low and high educated populations.

Sensitivity analyses

A number of sensitivity analyses were performed to further explore the relationship between overweight status, smoking status and the burden of disability.

First, an additional analysis was carried out to assess the sensitivity of the results to differences in the ways in which surveys in the participating countries were carried out. To this end, dummy variables for each country were added to the 'main effect' MSM model. Such model can be seen as a fixed effects model. Differences between the countries were found in

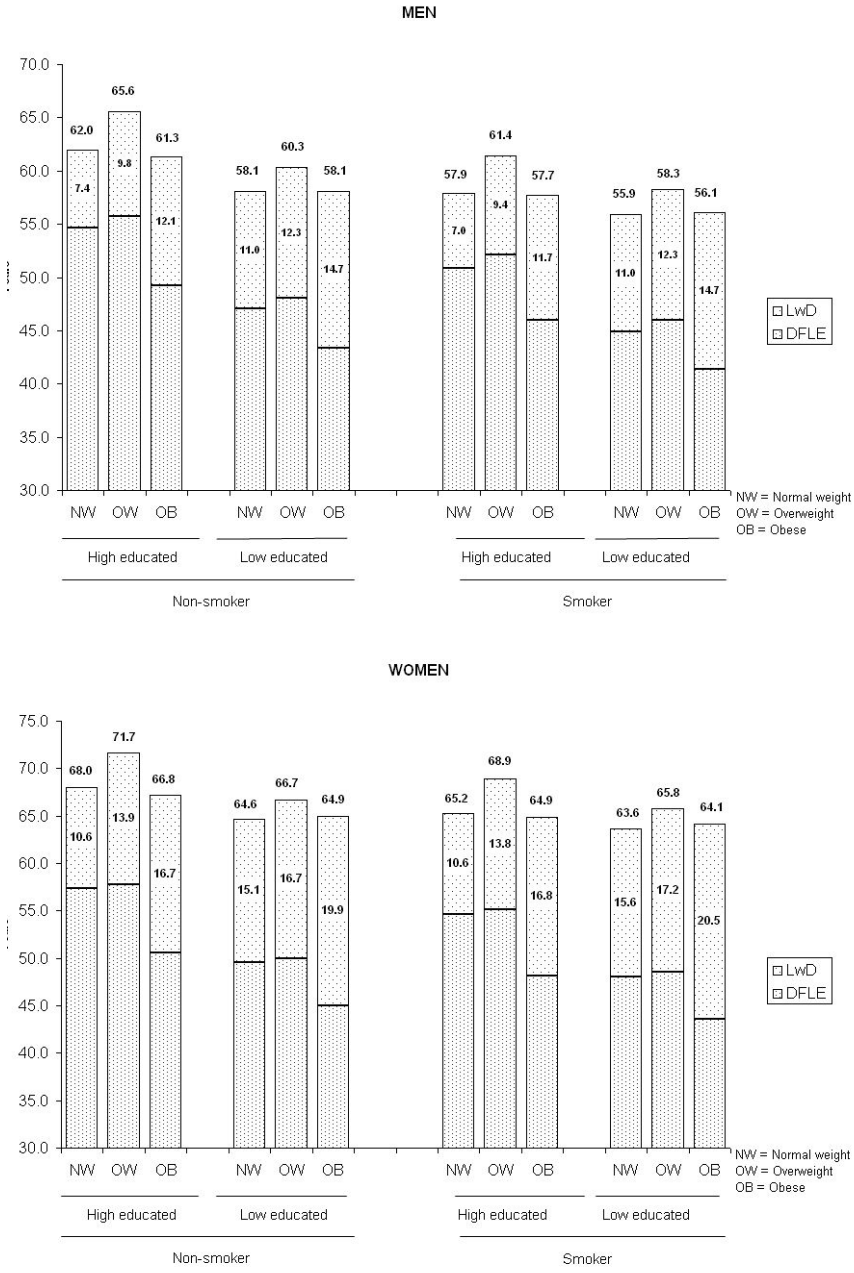


Figure 3.1 Life expectancy estimates according to smoking and overweight status at age 16 by educational level.

Notes: DFLE: disability-free life expectancy, LwD: life expectancy with disability

terms of the general level of transition rates. However, these inter-country differences did not substantially alter our key findings. For example the hazard ratio of disability incidence among obese were 1.49 (CI: 1.40-1.59) versus 1.64 (CI: 1.54-1.74) with and without controlling for counties, respectively. Similar (but generally smaller) effects were found for all risk factors.

Second, the original obese category (BMI>30) was split into two sub-categories: BMI between 30 and 35 (mild obese), and higher than 35 (severe obese). Our results indicated heterogeneity within the group of obese people, with the severe obese being worst off. The incidence of disability was associated with mild obesity (HR=1.60, CI: 1.50-1.70) and slightly stronger with severe obesity (HR=1.91, CI: 1.69-2.16). Besides, the relative risk of recovering from disability was lower in the severely obese group (HR=0.79, CI: 0.69-0.90) than in the mild obese group (HR=1.00, CI: 0.94-1.07). No statistically significant differences were found in mortality risk. Hazard ratios of the other risk factors remained stable after recoding the obese category.

Third, the influence of age on the association was explored. It is often argued that the size of the association between a risk factor and outcome decreases with age. Therefore two-way interaction variables between age and the risk factors were added to the 'main effects' MSM model. We found significant interaction terms between age and obesity (for disability incidence), and between age and daily smoking (for both disability incidence and recovery). The age-interaction term was fairly small indicating an approximately a 0.4% percent decrease with every year of increasing age.

Data strength and limitations

The ECHP data has a number of strengths of note. The most evident of which is the availability of a large number of observations on disability incidence or recovery, and moreover the use of an identical survey design and survey questionnaire for all participating countries. Although the data collection was carried out by the National Institutes of Statistics separately, and hence national versions of the ECHP are not perfectly comparable, these common questions ensured a much higher degree of comparability between countries than would have been possible using national data sources. The key variables used in this paper, on health status and risk factors, are comparable across all ECHP countries. Our study was the first to assess the impact of overweight for large number of Western-European countries simultaneously. In contrast, previous European studies focused exclusively on single countries, and used relatively much smaller data sets.

This study also has a number of limitations, however. First, disability data were self-reported, which can result in either under- or over-reporting of disability. If the reporting

of disability differs by risk factor group then our estimates of impact of LE and LwD will also be biased. Studies have shown that measurements of functional limitations by self-report or objective measures are consistently associated, and that they reflect similar assessment of function[86,113]. It is therefore likely that objective measures of disability would show similar associations with smoking, overweight status and education.

A second potential limitation is related to the use of self reports on smoking and on weight and height. It has been found that smoking prevalence rates are underestimated if estimates are based on self-report[114]. Similarly, people tended to under-report weight and over-report height[115]. These findings were confirmed by recent studies[116,117]. If such underreporting of daily smoking and BMI levels is non-differential, it may lead to an underestimation of the association with disability, and thus an underestimation of the differences in LwD.

A third drawback relates to non-response and attrition, which might be a problem in our study if they are related to disability or risk factor groups. Some studies have explored the attrition in the ECHP. For example, analyses on attrition in the ECHP showed a positive relationship between attrition and worsening health in all countries[118] and only a weak relationship between attrition and educational level[119]. Additional to these studies, we assessed the likelihood of attrition in the current study population in relation to characteristics at the last wave in which the respondent participated[120]. We found that the risk of loss to follow-up for reasons other than death or institutionalization was hardly related to disability status, sex, overweight status or educational level (relative differences of about 10 percent or less). We found slightly higher risk of attrition in former and daily smokers only (relative difference 15 percent). These findings imply that the relative differences are unlikely to have a major effect on differential retention, and therefore attrition could not have strongly biased our estimates of LE and LwD by overweight status.

When we adjusted the estimates for underestimation of mortality in the ECHP data, we had to assume that the degree of underestimation was identical in each risk factor group. If, for a specific risk factor group the under-registration of mortality would in fact be larger than what we assumed, the LE for this group would be overestimated. It is difficult to assess to what extent this is a problem. Even though the causes of under-registration of deaths in the ECHP data are not known, we see no reason to expect a strong relationship with overweight status or other risk factors. Furthermore, we would like to point out that even though this problem might affect LE estimates, it could not explain the large differences between BMI groups in estimates of LwD.

Previous studies

Data from previous studies are not directly comparable with our data for several reasons: of the different ages for which life expectancies were calculated; many of the previous studies reflect life histories of older cohorts; and different measures and classifications of disability were used. Nonetheless, comparisons could be made with regards to the general patterns observed.

Peeters et al.[98,121] found large effects of overweight and obesity on premature death. According to their results non-smoking men and women lost 5.8 and 7.1 years of LE at age 40, respectively, due to obesity. Because of both higher disability prevalence and higher mortality in the obese population, the authors found no significant difference in LwD (measured by activities of daily living (ADL) scores) between the obese and those of normal weight. However, these effects reflect life histories of older cohorts. There is evidence that the effects of high BMI on mortality have diminished over time[101,122].

Studies using more recent data from the U.S. have already justified smaller impacts on LE but large effects on LwD. For example, Reuser et al.[102] estimated the burden of mortality of obesity among middle and old-age adults in the Health and Retirement Survey (HRS). LE and LE with ADL disability were calculated in relation to self-reported body mass index, smoking and education at age 55. Obesity was found to have only a limited effect on mortality, and overweight was estimated to be protective for dying. Both overweight and obesity increased LwD for both sexes. These results are closely consistent with our findings.

Walter et al. estimated the influence of overweight and obesity on mortality and disability by quantifying its effect in terms of disability-free life expectancy and years lost to disability (YLD) for a suburban elderly population in the Netherlands[123]. Similar to our conclusions, they did not find that increased BMI reduces LE because of a protective effect of overweight on death (HR=0.81 compared to normal weight). As in our study, both overweight and obesity were found to be associated with a larger number of years lived with disability.

Evaluation

The relation between overweight and mortality has been a controversial topic. We showed that overweight is associated with protection for dying among the disabled, while the relationship between obesity and mortality is modest, especially among men. Our results reaffirm recent doubts about overstated concerns of overweight and obesity in terms of excess mortality[124]. Possible explanations for the small and even protective mortality effect of overweight include improved survival of overweight persons from major diseases of developed societies, e.g. heart failure[125] or CVD[126], and a better nutritional status providing necessary reserves during chronic disease[127]. Recent studies reaffirmed the protective ef-

fect of overweight as well documenting that increased BMI protects against mortality after hospitalisation[128].

3.5. Conclusions

The steadily increasing life expectancy in the developed countries is a great achievement but at the same time a major concern. It raises the question of whether living longer lives will be accompanied by a decrease or an increase of disability during old age. The main concern is that the obesity epidemic may, in the long run, increase the prevalence of disability in ageing populations.

Our results show that tobacco control is still highly relevant to the prevention of premature death. Continued success in this area may contribute to a further increase in life expectancy, but without substantially affecting the burden of disability over the life course (i.e. in terms of LwD). This stresses the importance of further public health aimed to address the obesity epidemic. Given the large impact of overweight and obesity on the burden of disability over the life course, halting the obesity epidemic is essential if an increase of LwD is to be stopped as LE continues to grow.

Appendix 3A Population characteristics in the ECHP, separately for all countries

Appendix 3A Population characteristics in the ECHP, separately for all countries

	Number of respondents[a]	Non-response (%)[b]	Retention percentage (%)[c]
Finland	11,184	27	92
Denmark	7,537	38	67
Ireland	14,170	44	64
Austria	9,450	30	82
Belgium	8,976	16	78
Greece	15,872	10	77
Italy	21,520	9	83
Spain	22,578	33	71
Portugal	14,285	11	87

Notes:
[a] First wave
[b] Household non-response, first wave
[c] Retention percentage until the fourth wave

Appendix 3B Mortality decomposition

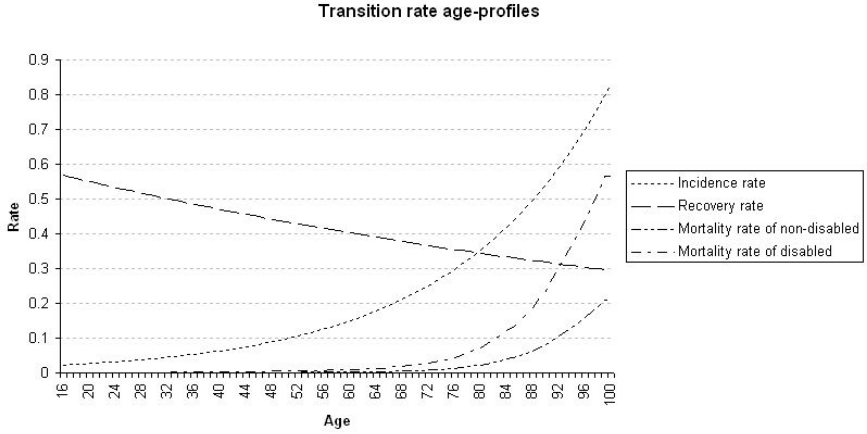
Raw total mortality rates for the pooled period 1998-2001 were calculated for each age and sex based on the total counts of mortality and exposure time obtained for each country, as published in Human Mortality Database (HMD). Age- and sex-specific prevalence of disability measures were calculated based on the number of disabled persons and the number of participants in the study population during the same period as published in the online database of European Health Monitoring Unit. Age- and sex-specific hazard ratios were estimated using MSM models based on the pooled study populations.

Combining total mortality rates, hazard ratios and prevalence of disability, mortality rates were calculated for non-disabled and disabled populations by the following formulas:

$$m_x^{(nd)} = \frac{m_x}{HR_x \times p_x^{(d)} + \left(1 - p_x^{(d)}\right)}$$
$$m_x^{(d)} = m_x^{(nd)} \times HR_x \qquad (1)$$

where $m_x^{(nd)}$, $m_x^{(d)}$, HR_x and $p_x^{(d)}$ indicated the mortality rate of non-disabled, mortality rate of disabled, estimated hazard ratio and prevalence of disability at age x, respectively.

Appendix 3C An illustration of the shape of age-profiles



Chapter 4

Socioeconomic inequalities in life and health expectancies around official retirement age in 10 Western-European countries

Majer IM, Nusselder WJ, Mackenbach JP, Kunst AE

Background

Discussions on raising pension eligibility age focus more on improvement in life expectancy (LE) and health expectancy measures than on socioeconomic differences in these measures. Therefore, this study assesses the level of socioeconomic differences in these two measures in Western-Europe.

Methods

Data from seven annual waves (1995-2001) of the European Community Household Panel were used. Health and socioeconomic information was collected using standardised questionnaires. Health was measured in terms of disability in daily activities. Socioeconomic status was determined as education level at baseline. Multistate Markov modelling was applied to obtain age-specific transition rates between health states for every country, educational level and gender. The multistate life table method was used to estimate LE and disability free life expectancy (DFLE) according to country, educational level and gender.

Results

When comparing high and low educational levels, differences in partial DFLE between the ages 50 and 65 were 2.1 years for men and 1.9 years for women. At age 65 years, for LE the difference between high and low educated groups was 3 years for men and 1.9 years for women, and for DFLE the difference between high and low educated groups was 4.6 years for men and 4.4 years for women. Similar patterns were observed in all countries, although inequalities tended to be greater in the southern countries.

Conclusions

Educational inequalities, favouring the higher educated, exist on both sides of the retirement eligibility age. Higher educated persons live longer in good health before retirement and can expect to live longer afterwards.

J Epidemiol Community Health. 2011 Nov;65(11):972-9.

4.1. Introduction

Social policymakers in Western-European countries are facing a common problem with regard to population ageing. Low birth rates, increasing life expectancy (LE) and dependency ratios have resulted in increased spending on pensions. Many countries have undertaken systematic restructuring of their pension system, including adjustment of the pension eligibility age to increasing LE[37,38]. At present, in most OECD countries the eligibility age for retirement among men and women is 65 years.

Although the rationale for such restructuring is to improve the financial sustainability of pension systems, such reforms may have adverse social effects[37,38]. An increase in LE is not necessarily equivalent to being able to work longer, and the extent to which total LE increases may differ from the increase in health expectancy (HE)[39,40]. Furthermore, both LE and HE are strongly related to socio-economic status (SES). If the standard retirement age is raised, people at the bottom of the socioeconomic ladder could be disproportionately affected. Whether one should consider socioeconomic differences in LE and HE in discussions on raising the pension eligibility age depends on the magnitude of these differences. Therefore, it is important to establish to what extent people in various socioeconomic groups are healthy and remain healthy at older ages.

Estimates of socioeconomic differences in HE are available for an increasing number of European countries[61,129-134]. Unfortunately, estimates from national studies could not be compared due to large variations in the data sources used, the age ranges covered, and the health and socioeconomic indicators employed. Because no study has used data from more than two countries to report on LE and HE by SES, an overview of the magnitude of socioeconomic differences in total and healthy life years at old age in Europe is still lacking.

Therefore, this study aims to determine the magnitude of socioeconomic differences in LE and HE, measured in terms of disability-free life expectancy (DFLE) in European countries: more specifically, inequalities in DFLE between age 50 and 65, and in LE and DFLE after age 65. DFLE combines information on mortality and disability into a summary measure of the expected number of years to be lived without disability. Investigation of these measures at these specific age intervals provides data on several aspects relevant to discussions on pension age.

4.2. Methods

Data

The data for this study were derived from the European Community Household Panel (ECHP). The ECHP is a standardised multi-purpose annual longitudinal social survey car-

ried out at the level of the European Union between 1994 and 2001. It is centrally designed and coordinated by the Statistical Office of the European Communities (Eurostat), and covers demographics, labour force behaviour, income, health, education and training, housing, migration, etc. The data were collected by the National Statistical Institutes or research centres of the participating countries using a uniform random sampling design and common blueprint questionnaires. Data checks, imputation and weighting were done centrally to maximise data quality. ECHP aimed at being both cross-sectionally and longitudinally representative for the national populations. The design and procedures of the ECHP have been extensively reviewed in Peracchi[107]. Information on non-response rates at baseline is given by Huisman et al.[135].

For the current study data were used from the second wave of 1995 onwards because this was the first year that an identical disability question was asked from the participants in all countries. Countries for which mortality data (the Netherlands) and disability data (Luxembourg) were not available were omitted from the study. Germany and the UK were replaced by data from national surveys (SOEP and BHPS, respectively). Austria and Finland joined the ECHP at wave 2 and 3, respectively.

Table 4.1 presents information on cumulative retention percentages within the countries, i.e. the cumulative percentages of individuals retained until the fourth wave of the Panel. For most of the countries this was 1997; however, this year represented the third wave in Austria and the second wave in Finland. Individuals who were out of scope of the survey by 1997 (e.g. because they died, became institutionalised, or had moved outside the EU) were excluded from the calculation of these rates. There were large differences between countries, with relatively high retention percentages in Italy (83%) and Portugal (87%) whereas the samples in Ireland, Denmark and Spain suffered from high attrition.

Indicators

The level of completed education at the first wave was used as a measure of SES. Individuals were divided into three groups according to their level of educational attainment based on the International Standard Classification of Education[109]: 1) lower secondary education or lower; 2) upper secondary education; and 3) tertiary education, which is constituted by vocational and university education.

All individuals were asked if they were hampered in their daily activities by any physical or mental health problem, illness or disability. The possible answers made a distinction between two severity degrees: "Yes, to some extent" or "Yes, severely"[108]. For this study these persons were considered as a single 'disabled' category. Persons who were lost to the ECHP because of moving to an institution were also considered as 'disabled' for the remainder of the study period.

Table 4.1 Total number of cases in the first wave, cumulative retention rates until fourth wave and proportion of education level in the study population, all countries

	Total number of cases at the first wave	Retention percentage until the fourth wave (%)	Study population			
			Men		Women	
			High level education (%)	Middle level education (%)	High level education (%)	Middle level education (%)
Finland	11 184	92	28.6	21.9	25.9	22.9
Denmark	7537	67	35.2	31.7	26.2	24.3
Ireland	14 170	64	21.0	10.5	23.2	7.9
Austria	9450	82	62.2	5.5	38.6	2.8
Belgium	8976	78	28.3	26.6	24.6	18.4
Greece	15 872	77	13.6	10.2	8.6	4.1
Italy	21 520	83	17.6	6.3	12.2	2.6
France	18 643	75	29.2	15.6	21.4	10.7
Spain	22 578	71	7.2	9.7	4.2	4.4
Portugal	14 285	87	2.8	3.3	1.8	2.9

Table 4.1 shows the distribution of the elderly population by level of education of men and women, respectively. The countries with the most skewed distribution are the southern countries.

Data analysis

In our multi-state Markov (MSM) models[110,136] three health states were defined: non-disabled, disabled and dead. Between the health states four transitions could occur, incidence (from non-disabled to disabled), recovery (from disabled to non-disabled), and state-specific mortality. For each country transition rates were estimated on the pooled dataset controlling for age, gender, education level and the country of interest. In other words, a model for each country was specified whereby the country of interest was compared to the other 9 countries. The advantage of preparing estimates using the data of all 10 countries together was the large number of observations; this ensured a high level of accuracy of the estimates of age profiles. We tested whether including 2-way or 3-way interaction terms between gender, education level and country of interest would result in a better model fit. AIC values indicated that the best model fit was achieved by including all 2-way interactions, but none of the 3-way interactions.Because in most of the countries mortality cases were under-registered in the ECHP, the mortality transition rates were adjusted to the level of the given countries in four steps. First, using national data, mortality rates were calculated for the pooled period 1995-2001 for each age, gender and country. Second, they were transformed into mortality rates of non-disabled and disabled assuming that 1) the age and sex-specific mortality rates in the overall (i.e. mixed non-disabled-disabled) population are

the weighted average of mortality rates of non-disabled and disabled populations, with the proportion of non-disabled and disabled respectively as weights, and 2) that the ratio between the mortality rate of disabled and non-disabled people is equal to the hazard ratio as estimated with the ECHP data. A more formal explanation of the decomposition is shown in the Appendix 3B. Third, rescaling factors were calculated for both types of mortality rates by age and gender. Rescaling factors specified how much age-specific and gender-specific mortality rates had to be scaled to make them consistent with the national data. Fourth, all estimated mortality rates were multiplied by the corresponding rescaling factors, assuming that under-representation of mortality was the same for all educational levels.

To provide estimates of LE and DFLE according to gender, educational level and country, multi-state life tables were used. The empirical input for these multi-state life tables were the transition rates described above, after converting them into probabilities[67]. Once a life table was set up for a country, probabilistic sensitivity analysis[111,137,138] was performed to estimate confidence intervals (CIs) around the life expectancies. Random regression coefficients were drawn from each regression model for 1000 times assuming multivariate normal distribution. After each draw the corresponding transition rates and life expectancies were calculated. The 25th and 975th of the latter ordered values indicated the boundaries of the CIs. All MSM analyses were performed in R[112], whereas life table calculations were carried out in Excel.

4.3. Results

The hazard ratios of transitions by educational level for the 10 countries are given in Table 4.2. Generally, the risk of disability onset and mortality from a non-disabled state was higher for the middle or low educated groups than for the high educated groups. For recovery rates, the higher the educational level the higher the rate to recover from disability. There were no differences between hazard ratios of mortality from a disabled state. Educational differences in hazard rates were similar for both genders, with some variation between the individual countries.

Table 4.2 Hazard ratios of disability-related transitions in relation to educational level, by country

	Incidence		Recovery		Mortality of non-disabled		Mortality of disabled	
Country	Middle	Low	Middle	Low	Middle	Low	Middle	Low
					Men			
Finland	1.15	1.29*	0.74*	0.69*	1.2	1.37	1.01	1.00
Denmark	1.19	1.37*	0.93	0.76*	1.36	2.25	1.02	0.81
Ireland	1.48*	1.68*	0.85	0.70*	0.99	1.61	1.10	1.11
Austria	1.91*	1.99*	1.07	0.88	1.47	1.41	0.83	1.35
Belgium	0.97	1.19	0.76*	0.78*	1.36	1.61	0.92	1.16
Greece	1.39*	1.84*	0.78	0.75*	1.13	1.81	1.09	0.99
Italy	1.27	1.86*	1.03	0.81	1.61	1.32	1.44	0.85
France	1.49*	1.56*	0.74*	0.55*	1.14	0.96	0.85	1.14
Spain	1.72*	2.86*	1.06	0.94	1.09	1.35	0.90	0.92
Portugal	1.17	2.25*	1.05	0.76	1.54	1.33	1.05	1.16
					Women			
Finland	0.97	1.28*	0.71*	0.72*	1.11	1.20	0.94	0.92
Denmark	1.00	1.33*	0.88	0.79*	1.25	2.00	0.98	0.78
Ireland	1.25	1.59*	0.80	0.74*	0.91	1.41	1.02	1.04
Austria	1.60*	1.95*	1.03	0.91	1.34	1.22	0.77	1.27
Belgium	0.81	1.13	0.73*	0.83*	1.25	1.40	0.85	1.08
Greece	1.16	1.79*	0.75	0.78*	1.05	1.58	1.02	0.93
Italy	1.08	1.83*	0.99	0.85	1.46	1.20	1.36	0.79
France	1.26*	1.53*	0.71*	0.58*	1.09	0.86	0.79	1.09
Spain	1.44*	2.75*	1.02	0.99	1.01	1.20	0.86	0.82
Portugal	0.99	2.17*	1.00	0.80	1.44	1.17	0.95	1.16

Notes: Derived from an MSM regression model that included age, sex, age × sex, middle level education, low level education, middle level education × sex, low level education × sex, country, country × middle level education, country × low level education, country × sex.

* significant at p=0.05 level

The estimates of partial DFLE per country, gender and educational levels are shown in Figure 4.1. Educational inequalities existed in partial DFLE in all countries. On average, people with a higher educational level can expect to live the most years without disability between age 50 and 65 years. Partial DFLE was 10.1 years for high educated men, 8.9 years for middle educated men, and 8.0 years for low educated men; the corresponding values for women were 10.8, 10.0 and 8.9 years. When comparing high to lower educational levels, these partial DFLEs translated into 2.1 and 0.9 years of difference for men, and 1.9 and 1.1 years for women. Larger inequalities were found in Portugal and France and smaller inequalities in Belgium.

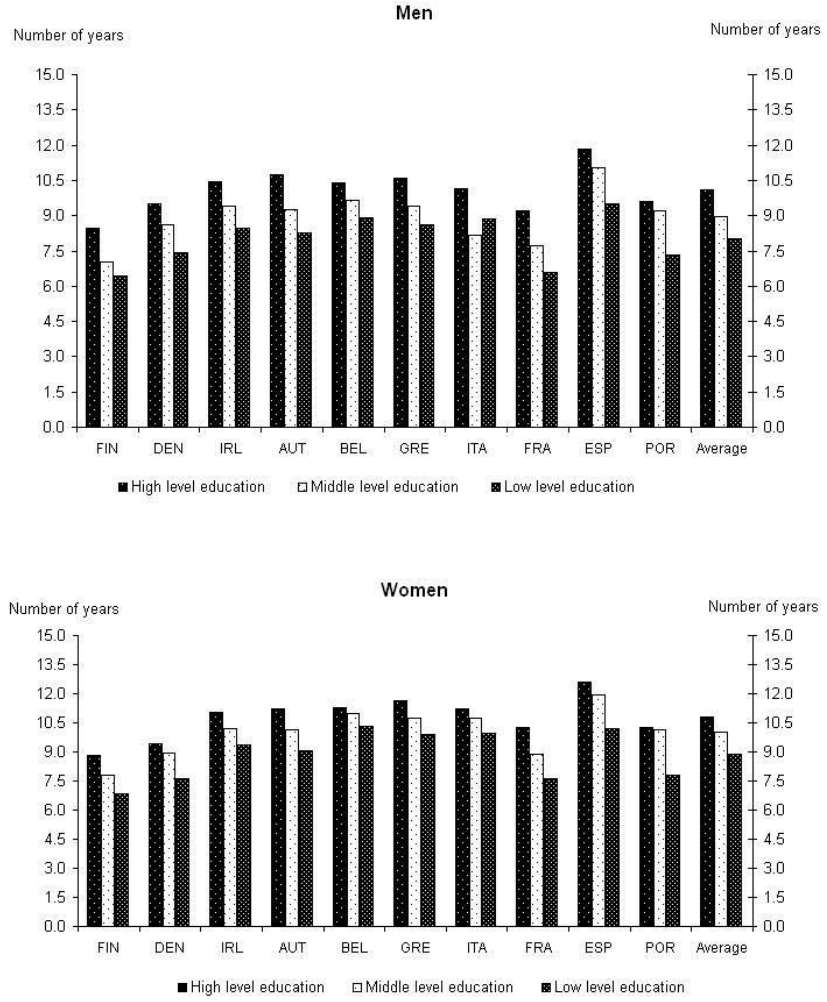


Figure 4.1 Partial disability-free life expectancy between age 50 and 65 by educational level

Table 4.3 presents LE estimates at age 65 years. A common pattern emerged in all countries: the higher the educational level the greater the LE at age 65. On average, the difference in men's LE between high educated and lower educated groups was around 3 years, i.e. about double the difference between the middle and low educated groups, i.e. 1.3. Among women, differences in LE were smaller than among men. On average, high and middle educated women can expect to live 1.9 and 1.3 years longer, respectively, than low educated women. Relatively larger differences were found in Austria and Portugal compared to smaller differences in Denmark and Finland.

Table 4.3 Total life expectancy at age 65 years by educational level, for men and women separately

	LE (95% confidence interval) by educational level			
	High	Middle	Low	High–Low / Middle-Low
Men				
Finland	15.7 (15.3 - 16.1)	14.6 (14.3 - 14.8)	14.2 (14.0 – 14.4)	1.5 / 0.3
Denmark	15.8 (15.4 - 16.2)	14.5 (14.2 - 14.7)	13.8 (13.7 - 13.8)	2.1 / 0.7
Ireland	17.8 (16.9 - 18.6)	16.0 (15.5 - 16.5)	14.1 (13.9 - 14.3)	3.7 / 1.9
Austria	17.5 (16.6 - 18.3)	16.3 (16.2 - 16.5)	13.6 (13.4 – 13.9)	3.8 / 2.7
Belgium	16.2 (15.7 – 16.8)	15.2 (14.9 - 15.5)	13.4 (13.2 – 13.7)	2.8 / 1.8
Greece	19.2 (18.5 - 19.8)	17.2 (16.7 – 17.6)	15.8 (15.6 - 15.9)	3.4 / 1.4
Italy	19.0 (18.2 – 19.8)	15.3 (15.0 - 15.7)	16.7 (16.5 – 16.8)	2.3 / -1.3
France	19.4 (19.0 – 19.8)	18.5 (18.3 – 18.7)	16.4 (16.2 - 16.6)	3.0 / 2.1
Spain	19.4 (18.8 - 19.9)	18.4 (17.9 - 18.9)	16.4 (16.3 – 16.6)	2.9 / 2.0
Portugal	18.7 (17.8 - 19.5)	16.7 (16.0 - 17.3)	14.8 (14.7 – 15.0)	3.8 / 1.9
Average	17.9 (17.2 – 18.5)	16.3 (15.9 – 16.6)	14.9 (14.8 - 15.1)	2.9 (2.5 – 3.4) / 1.3 (1.2 – 1.5)
Women				
Finland	18.9 (18.6 – 19.2)	18.6 (18.4 - 18.9)	18.3 (18.2 – 18.5)	0.6 / 0.3
Denmark	17.9 (17.5 - 18.4)	17.4 (17.1 – 17.8)	17.2 (17.1 – 17.2)	0.8 / 0.3
Ireland	21.0 (20.2 – 21.7)	20.1 (19.7 – 20.7)	18.3 (18.1 – 18.5)	2.7 / 1.8
Austria	21.4 (20.7 – 22.1)	21.1 (21.0 – 21.2)	18.4 (18.3 – 18.6)	3.0 / 2.7
Belgium	20.8 (20.4 – 21.2)	20.7 (20.4 – 21.0)	19.1 (18.9 – 19.3)	1.7 / 1.6
Greece	20.6 (19.9 - 21.2)	19.4 (19.0 - 19.8)	18.1 (18.0 – 18.2)	2.5 / 1.3
Italy	22.1 (21.3 – 22.8)	19.6 (19.2 - 20.0)	20.8 (20.7 – 21.0)	1.3 / -1.2
France	23.8 (23.4 - 24.2)	23.9 (23.8 – 24.1)	21.7 (21.6 – 21.9)	2.1 / 2.2
Spain	22.3 (21.6 – 22.9)	22.0 (21.5 – 22.5)	20.4 (20.3 – 20.5)	1.9 / 1.6
Portugal	21.4 (20.7 – 22.2)	20.6 (20.0 – 21.2)	18.5 (18.3 – 18.6)	3.0 / 2.2
Average	21.0 (20.4 – 21.6)	20.4 (20.0 – 20.7)	19.1 (18.9 – 19.2)	1.9 (1.5 – 2.4) / 1.3 (1.1 – 1.5)

Table 4.4 presents estimates on the DFLE at age 65. The higher the educational level the longer people remain healthy at older age. For both men and women, differences between the educational attainment groups were larger for DFLE than for LE. Among men, the average DFLE difference between high and low educated groups was 4.6 years, compared with 2.1 between middle and low educated men; for women, the corresponding values are 4.4 and 2.7, respectively. Larger differences were seen in Spain and Portugal, and smaller differences in Finland, Denmark and Belgium.

Table 4.4 Life expectancy without disability at age 65 years by educational level, for men and women separately

	DFLE (95% confidence interval) by educational level			
	High	Middle	Low	High–Low / Middle-Low
Men				
Finland	7.8 (7.1 –8.7)	5.9 (5.3 – 6.6)	5.2 (4.8 - 5.7)	2.6 / 0.7
Denmark	9.7 (8.7 – 10.7)	8.2 (7.4 – 8.8)	6.5 (6.0 – 7.1)	3.2 / 1.6
Ireland	14.2 (12.5 - 15.7)	11.3 (10.3 – 12.2)	9.1 (8.6 – 9.6)	5.1 / 2.2
Austria	11.8 (9.8 – 13.7)	9.1 (8.5 – 9.6)	7.2 (6.7 – 7.7)	4.7 / 1.9
Belgium	11.0 (10.1 – 12.2)	9.6 (8.8 – 10.4)	8.1 (7.6 – 8.7)	2.9 / 1.5
Greece	15.4 (14.1 – 16.5)	12.1 (11.2 – 13.0)	9.9 (9.6 – 10.3)	5.4 / 2.2
Italy	15.8 (14.4 – 17.1)	12.5 (11.9 – 13.2)	11.4 (11.1 – 11.8)	4.3 / 1.1
France	13.0 (12.0 – 14.0)	9.4 (8.7 – 10.0)	7.3 (6.9 – 7.8)	5.6 / 2.0
Spain	15.2 (14.1 – 16.4)	13.1 (12.0 – 14.2)	9.8 (9.4 – 10.1)	5.5 / 3.4
Portugal	14.0 (12.1 – 15.7)	12.2 (10.4 – 13.8)	7.7 (7.4 – 8.1)	6.2 / 4.4
Average	12.8 (11.5– 14.1)	10.3 (9.5 – 11.2)	8.2 (7.8 – 8.7)	4.6 (3.7 – 5.4) / 2.1 (1.7 – 2.5)
Women				
Finland	8.6 (7.7 –9.5)	7.2 (6.5 – 8.0)	6.0 (5.6 – 6.6)	2.5 / 1.2
Denmark	9.4 (8.4 – 10.5)	8.7 (7.8 – 9.6)	6.9 (6.3 – 7.4)	2.6 / 1.8
Ireland	16.2 (14.4 – 17.8)	13.9 (12.8 – 15.1)	11.4 (10.8 – 12.0)	4.7 / 2.5
Austria	13.9 (11.6 – 15.9)	11.6 (10.9 – 12.2)	9.2 (8.7 – 9.7)	4.7 / 2.4
Belgium	13.5 (12.3 – 14.6)	12.8 (11.6 – 13.7)	11.1 (10.5 – 11.8)	2.3 / 1.6
Greece	15.9 (14.4 – 17.3)	13.5 (12.5 – 14.5)	10.9 (10.5 – 11.2)	5.0 / 2.6
Italy	17.5 (15.7 – 19.2)	15.5 (14.6 – 16.3)	13.3 (12.9 – 13.7)	4.2 / 2.2
France	14.8 (13.6 – 16.0)	11.6 (10.8 – 12.4)	8.7 (8.3 – 9.2)	6.1 / 2.9
Spain	16.5 (15.0 – 17.9)	14.8 (13.6 – 16.1)	10.8 (10.5 – 11.1)	5.6 / 4.0
Portugal	14.3 (12.1 – 16.3)	13.7 (11.5 – 15.8)	8.0 (7.7 – 8.4)	6.3 / 5.7
Average	14.0 (12.5 -15.5)	12.3 (11.3 – 13.4)	9.6 (9.2 – 10.1)	4.4 (3.3 – 5.4) / 2.7 (2.1 – 3.3)

4.4. Discussion

This study explored educational differences in (disability-free) LE before and after formal retirement age across 10 Western European countries. Populations with a higher level of education can expect to live more years free of disability before retirement, suggesting less problems in reaching pension age in good health. People with a higher educational level can also expect to live longer after retirement, implying that they represent a greater liability for pension funds. For LE larger inequalities were found among men than among women, whereas differences in DFLE were similar for both men and women. Similar patterns emerged in all countries, although the inequalities tended to be larger in southern countries.

Data issues

Some limitations regarding the use of data need addressing. First, disability data were self-reported which can result in either under- or over-reporting of disability. If the reporting of disability differs by educational level then the differences in DFLE estimates will also be biased. Although we cannot exclude that reporting of disability differed by educational level, it is unlikely that differential reporting patterns would have strongly biased our results. To support this conclusion, an earlier study showed that educational inequalities in the prevalence of disability remained about the same when self-reported measures of disability were replaced by performance-based measures[139].

Non-response and attrition might have been a problem in the present study if they had been related to disability and SES. A few studies have explored the association between attrition and disability status in the ECHP. For example, analyses on attrition in the ECHP showed a positive relationship with worsening health in all countries[118] and only a weak relationship with educational level[119]. Also, we assessed the likelihood of attrition in the current study population in relation to characteristics at the last wave in which the respondent participated[120]. We found that the risk of loss to follow-up for reasons other than death or institutionalization was hardly related to disability status or educational level. This implies that differential retention could not have strongly biased our estimates of relative educational differences in LE and DFLE.

The distribution of educational level across the countries showed rather skewed patterns, with large proportions in the lower educated groups. The sample size of higher educated persons was particularly small in Austria, Greece, Italy, Spain and Portugal. Because of the relatively small sample sizes the CIs are wider for the higher educated groups. As a result, the CIs around LE and DFLE estimates for the groups with middle and high educational status often overlap. The same problem of random fluctuations might explain unexpectedly high or low values of LE or DFLE in some specific population as in the case of Italy.

A main advantage of the ECHP was the availability of a large number of observations based on use of an identical survey design and survey questionnaire for all participating countries. Although the data collection was carried out by the National Institutes of Statistics separately, and hence national versions of the ECHP are not perfectly comparable, these common questions ensured a much higher degree of comparability between countries than would have been possible using national data sources. The key variables used in this paper, on educational level and health status, are comparable across all ECHP countries, albeit comparability of educational information could only be achieved at the level of three broad groups. Even so, international comparability may still be far from optimal due to cross-national variations in factors such as people's perception of health problems and their propensity to report perceived health problems. Therefore, caution is needed when interpreting any differences in results between these European countries. We therefore recommend focussing on the patterns common to all countries, rather than on the cross-national variations.

Previous studies

Estimates of DFLE according to SES have been reported for several countries[21,61,103,129-134,140-152] however, for Greece, Ireland and Portugal we believe that the current study is the first to report estimates of socioeconomic inequalities in DFLE. Data from other countries are not directly comparable with our data because of the different ages at which life expectancies were calculated, or because different measures and classifications of disability and SES were used.

A few estimates for SES differences in LE and DFLE at higher ages have been reported earlier. In the Netherlands, at age ≥65 years, for total LE and healthy LE a difference of 3.1 and 3.4 years, respectively, was found in men when comparing primary educated and higher educated groups[21]. In England and Wales, in the age group ≥65 years, for estimated LE free of mobility limitations a difference of 2.7 and 2.5 years was found between high and low educated men and women, respectively; corresponding differences in LE were 1.1 and 1.9 years[61]. In the USA, differences in LE between primary and higher educated were 2.5 and 3.3 years, respectively, whereas differences in active LE were 2.4 and 2.8 years for white men and women, respectively[150]. For Italy, differences in DFLE were also found, with a 3-year difference between the higher and lower educated for both men and women at age 65 years[131].

The present study shows similar basic patterns of DFLE and LE by SES. A common finding is that inequalities in HE were larger than inequalities in LE. Although comparison between studies is difficult we found somewhat larger differences, especially in terms of DFLE. This might be due to the milder measure for disability that was used in our study.

At a pan-European level, previous studies – implicitly or explicitly – examined the feasibility of increasing labour force participation around retirement eligibility age using health expectancy measures based on the ECHP data[153,154], the Statistics of Income and Living Conditions (SILC) data[155], or the Survey of Health, Ageing and Retirement in Europe (SHARE) data[156]. These studies assessed the average health expectancy and 'unused capacity' in several European countries, and pointed at substantial differences therein. Our study extends this work for a selection of European countries, included in the ECHP, by focussing on variations by socio-economic status. It would be particularly interesting to further extend our study to the Central-Eastern European (CEE) countries included in the SHARE or SILC surveys, even though SILC data do not contain information on mortality and SHARE data have only one wave with CEE countries.

In previous studies, large inequalities were observed independent of the applied socioeconomic indicators (educational, occupational class, income, or wealth measures). The inequalities that we observed in relation to educational level may reflect the operation of different causal mechanisms, including effects of socioeconomic position in later life. Blane[157] proposed five explanations for the strong and persistent association between education and health in later life: 1) influence of childhood living circumstances on adult health, 2) effects of occupation and income achieved in adult life, 3) receptivity and adaptability to health education, 4) effects of ill health during childhood on education, and 5) influence of other background variables, e.g. self-efficacy time preference, etc.

Although the evidence regarding cross-national variations is rather weak, it is interesting to find that inequalities in DFLE were generally larger in southern than in northern countries. Our results are generally in line with the hypothesis that in Nordic countries (where egalitarian welfare regimes have been implemented) inequalities in health may be smaller. Protective welfare systems could lead to smaller inequalities through their effect on income and wealth, on working conditions, and on socio-psychological resources available to different socio-economic groups Nevertheless, other studies provide no consistent evidence that socioeconomic inequalities in mortality or self-reported health are smaller in Nordic countries[158].

Conclusions

Systematic reforms aimed at increasing pension(able) age have been proposed or implemented, also to take into account the trend of rising life expectancy and similar rises in health expectancy. However, such restructuring rarely acknowledges the differences in life and health expectancies between socio-economic groups. For Europe at large, this study has shown that such inequalities are substantial in every country investigated. Educational inequalities favouring the higher educated exist on both sides of the official retirement age.

On the one side, retired people with a higher educational level live healthier and longer lives and represent a subpopulation making greater demands on pension resources. On the other side, those with a higher educational level live more years in good health before reaching pension age. Although being disabled does not necessarily mean being unable to work, and being non-disabled does not necessarily mean being able to work, good health is associated with increased likelihood of participation. Our results indicate that in the case of lower socioeconomic groups, increasing the retirement ages will be more difficult to achieve (given higher disability prevalence) or to justify (given shorter life expectancies). Social policies should be oriented towards promoting the employment of seniors with higher socio-economic status because this is the group of people where the "unused capacity" is largest[159]. As a consequence, more flexible pension schemes could be considered, for instance taking into account the number of years worked over life course, or allowing for part-time pensioning.

PART II

FORECASTING HEALTH EXPECTANCY

Chapter 5

Modeling and forecasting health expectancy; theoretical framework and application

I.M. Majer, R. Stevens, W.N. Nusselder, J.P. Mackenbach, P.H.M. van Baal

Life expectancy continues to grow in most western countries, however, a major remaining question is whether longer life expectancy will be associated with more or less life years spent with ill-health. Therefore it is useful to complement forecasts of life expectancy with forecasts of health expectancies. To forecast health expectancy an extension of the stochastic extrapolative models developed for forecasting total life expectancy could be applied, but instead of projecting total mortality and using regular life tables, one could project transition probabilities between health states simultaneously and use multi-state life table methods (MSLT). In our paper we present a theoretical framework for a MSLT model, in which the transition probabilities depend on age and calendar time. The goal of our study was to describe a model that projects transition probabilities by the Lee-Carter method, and to illustrate how it can be used to forecast future health expectancy with prediction intervals around the estimates. We applied the method to data on the Dutch population aged 55 and older, and projected transition probabilities until 2030 to obtain forecasts of life expectancy, disability-free life expectancy and probability of compression of disability.

5.1. Background

Life expectancy

Over the last decades improving mortality conditions have resulted in increases in the length of human life and subsequent population ageing in western countries. The continuous rise of life expectancy (LE) is certainly welcome. However, the increasing life expectancy has been accompanied by low fertility rates, resulting in growth of the elderly proportion of populations in most OECD countries[25]. Since these developments have considerable consequences for the sustainability of two fundamental institutions of social security, health care and pensions, the future of human survival has gained growing attention not only among demographers and epidemiologists but also among actuaries, economists and financial specialists[32,33]. The concern is that health insurers and pension funds will have to provide provision for however long people will live.

Whether and to what extent life expectancy will continue to increase has been source of discussion, dividing scholars into camps of optimists or pessimists[160]. Various arguments have been used to support the potential upward or downward effects on mortality rates, including not only historical trends, biomedical and life-style arguments, but even the potential of medical breakthroughs. Reflecting on the uncertainty surrounding the evolution of future mortality ife expectancy, particularly at older ages, demographers began to consider improvements in mortality, and all other quantities depending on future mortality, as a stochastic process. A wide range of extrapolative empirical models have been proposed which share a common feature: based on historical data, they all estimate age-specific mortality as a function of time, and project them into the future using probability distributions[31,34,35,161,162]. The earliest and still one of the most popular models is the Lee-Carter model[31], which proved to perform very well and has become the "leading statistical model of mortality forecasting in the demographic literature"[163]. In many cases however, it is not sufficient to form expectations on future life expectancy alone. For example, if a further raise of retirement age is being considered, it is more appropriate to estimate how long people will be able to work in the future, instead of simply how long they will live. An important question in aging populations is whether increases in life expectancy will be accompanied with greater or lesser increases in life years spent in poor health and/or with disability[164]. Consequently, it would be useful to form expectations not only on how long people are expected to live, but also on how healthy they will be in the future.

Health expectancy and compression of morbidity

Health expectancy (or expected healthy life years, HE) typically combines mortality and morbidity information to represent overall population health in a single indicator[10]. It measures the number of remaining life years that a person at a certain age is expected to

live without ill-health, and is increasingly used to complement the conventional measure of life expectancy[165]. Because health expectancy was developed to reflect that not all years of a person's life are lived in perfect health, estimates of health expectancies have been very attractive and widely used tools for monitoring trends in population health[10].

Three distinct theories have been proposed regarding the evolution of health expectancy and life expectancy over time: compression of morbidity[14], expansion of morbidity[15], and the so-called dynamic equilibrium theory[16]. Compression of morbidity postulates that survival and morbidity curves will become closer to each other in the future, as a result of strategies that effectively eliminate premature morbidity and mortality. Supporters of the pessimistic expansion theory assert that increases in life expectancy will not be followed by increases in healthy life expectancy because the declines in mortality stem from mainly those suffering from chronic, disabling diseases. The third hypothesis, dynamic equilibrium emphasizes the link between morbidity and mortality and asserts that the increases in total life expectancy would likely entail increases in life expectancy both with and without morbidity, whereas years with severe morbidity remain stable. Compression (expansion) of morbidity can be measured in absolute values: increases in healthy life expectancy are larger (smaller) than increases in total life expectancy; or as a proportion: healthy life expectancy over total life expectancy is increasing (decreasing).

Modeling and forecasting health expectancy

There are two commonly used methods to estimate health expectancy: Sullivan's method and the multistate life table (MSLT) method. They require different kinds of data and can yield different results[11]. The simpler Sullivan method estimates health expectancy by combining mortality data with external information on cross-sectional prevalence in each health state[12]. The more refined multistate method models the prevalence of disability as the result of several transitions (e.g. incidence, mortality and possibly remission)[13]. Although a multistate model has larger data requirements since it needs age-specific estimates of multiple transitions, it has several advantages. Most importantly, it acknowledges the fact that the stock of ill health is the result of different processes. Accordingly, one can interpret trends in health expectancy as a result of developments in the underlying transition rates.

In the demographic literature there are many multistate projection studies that forecast the size of populations into the future. The projections are based on cohort components of demographic change including births, deaths, and migration. The transitions between the modeled states are based on transition rates that may change in time[166] and / or may vary between subpopulations. Projections for subpopulations has been performed by region[167], educational status[168], household status[169], labor force participation[170] as well as by health / disability status[171]. Recently a large-scale research project has been completed in the Eu-

ropean Union. One of the goals of the research was to provide the size and age structure of future populations with and without disability[172].

With regard to forecasting morbidity by multistate models we refer to a review of epidemiological approaches by Tabeau[173]. The main output of the models discussed here are disease incidence numbers and cause-specific or total mortality counts. Several studies on health-based population forecasts and implications in terms of health service needs have been carried out by Manton and colleagues[174-176]. They have proposed the use of a multidimensional stochastic process model to project population changes under simulated modifications in the distribution of major risk factors[171,177]. None of these studies focused explicitly on forecasting the expected life years that a person is expected to live without ill-health.

Although there is a clear rationale for forecasting health expectancy efforts at improving forecasting models have been limited exclusively to life expectancy. This is partly because the primary object of interest for pension providers is the expected life years after retirement and not the expected healthy life years, and partly because the lack of long time series data on health status. Furthermore, compared to forecasting life expectancy based on mortality alone, forecasting health expectancy is more complicated because of the additional dimensions in the models. To our knowledge, there has been only a recent study that forecast both life expectancy and a form of health expectancy. The authors employed Sullivan's method to forecast active life expectancy for a number of years during the twenty-first century until 2080 for the U.S.[178], and for which they used future life tables estimated by the Social Security Administration and two scenarios on the expected rate of disability decline (1.7% and 0.8% per annum). Other studies forecasting health expectancy - either Sullivan or multistate - are virtually non-existent.

Purpose of our study

Our study had two goals. The first was to build a theoretical framework for a multi-state life table model, in which the transition probabilities depend on age and calendar time. We aimed to describe how to model and forecast these transition probabilities using the Lee-Carter method, and to illustrate how this method can be used to forecast future health expectancy with prediction intervals around the forecasts. Second, we applied the method to data of the Dutch population aged 55 and older, and estimated health expectancies between 1989 and 2007 for men and women. We projected the transition probabilities until 2030, and applied multistate life table methods to obtain forecasts of life expectancy, disability-free life expectancy (DFLE), a frequently employed form of health expectancy, and life expectancy of non-disabled and life expectancy of disabled people. In addition, we analyzed the changing relationship between DFLE and life expectancy over time, and attached probability distributions to different future scenarios of compression or expansion of disability.

A key concept in our work is the idea that the stochastic extrapolative models developed to forecast total life expectancy could be used to forecast health expectancy. However, instead of forecasting population mortality probabilities and using regular life tables, future health expectancies could be modeled by forecasting transition probabilities between the health states, and developments of future transition rates can be viewed as realizations of stochastic processes, like in case of mortality. Accordingly, future health expectancy could be modeled through transition probabilities that are extrapolated in a stochastic manner.

5.2. Methods

We specified three health states indicating the functional status of the individuals: non-disabled, disabled, and dead. Three possible transitions between these states were allowed: healthy persons may experience onset of disability or they may die, while disabled person may die[1]. A crucial element in our model is that transition probabilities do not only depend on age but also on calendar year, and that they are treated as stochastic time series, which can be forecasted by extending the Lee-Carter model[31]. In the following subsection we will describe how the Lee-Carter method can be used to forecast multiple transitions in order to forecast health expectancy. In particular, we pay attention as to how the joint tendency of the stochastic period effects of each transition type can be modeled. Limitations of the assumptions and their importance for the results are assessed at the Discussion section of this chapter.

Forecasting transition rates and health expectancy with the Lee-Carter model

The Lee Carter model takes the following form:

$$\ln m_{x,t}^{(i)} = \alpha_x^{(i)} + \beta_x^{(i)} \kappa_t^{(i)} + \varepsilon_{x,t}^{(i)} \ . \qquad (1)$$

$m_{x,t}^{(i)}$ is the specific type i = (tr,g)$\in I$ transition rate for an x year old individual at time $t \in \{1,2,\ldots,T\}$ with gender $g \in \{male, female\}$ and tr$\in \{nd, d, inc\}$ the mortality rate of non-disabled, mortality rate of disabled, and the incidence rate, respectively. The parameters to be estimated are $\alpha_x^{(i)}$, $\beta_x^{(i)}$, and $\kappa_t^{(i)}$, and $\varepsilon_{x,t}^{(i)}$ is the error term.

Applying the Lee-Carter model is a two step procedure. First, the parameters in equation (1) are estimated. Second, the transition rates are projected by forecasting the time-dependent parameter.

1 Detailed descriptions of the quantities obtainable in an MSLT and information about how transition rates are converted into 1-year transition probabilities have been put in Appendix 5B.

In the first step, parameters of equation (1), $\alpha_x^{(i)}$, $\beta_x^{(i)}$, and $\kappa_t^{(i)}$ are estimated to model a given type of transition rate, $\ln m_{x,t}^{(i)}$. The least square solution to the equation (1) is sought, however this model cannot be fitted by Ordinary Least Squares, because there are no predictors on the right hand side. Nevertheless, assuming that $\varepsilon_{x,t}^{(i)}$ is normally distributed, the singular value decomposition (SVD) of the matrix with elements $\ln m_{x,t}^{(i)} - \alpha_x^{(i)}$ estimation is equivalent to the maximum likelihood estimates. Generally a one factor model is used, hence in the Lee-Carter model the matrix, $\beta_x \kappa_t$, is a function of the leading singular value, $\sigma_1^{(i)}$, the first column, $u_1^{(i)}$, and the first row, $[v_1^{(i)}]^1$, of the SVD. Due to lack of identification Lee and Carter proposed to use the constraints: $\sum_x \hat{\beta}_x^{(i)}=0$ and $\sum_t \hat{\kappa}_t^{(i)}=1$ in order to ensure that the solutions are unique. The latter constraint implies that summing the modeled log transition rate over t, and taking its expected value, the age-specific constant parameter $\alpha_1^{(i)}$ is simply the empirical average of the log transition rate at age x. The parameter $\kappa_t^{(i)}$ indicates the time-dependent latent process that quantifies the evolution of transition rates over time. The $\beta_x^{(i)}$ profiles express which age-specific rate change rapidly or slowly in response to changes in $\kappa_t^{(i)}$. $\varepsilon_{x,t}^{(i)}$'s are sets of disturbances. If X is the set of age groups and T is the set of time periods, then the parameter estimates are given by:

$$\hat{\alpha}_x^{(i)} = \frac{\sum_{t=1}^{T} \ln(m_{x,t}^{(i)})}{T} \quad (2) \qquad \hat{\beta}_x^{(i)} = \frac{u_1^{(i)}(x)}{\sum_{x \in X} u_1^{(i)}(x)} \quad (3) \qquad \hat{\kappa}_t^{(i)} = \sigma_1^{(i)} v_1^{(i)}(t) \sum_{x \in X} u_1^{(i)}(x) \quad (4)$$

At each age the disturbances are assumed to have an independently and identically distributed multivariate normal distribution with mean zero and covariance matrix $\sum_x^2$, which takes into account the joint distribution of the disturbances of every type of transition rate: $\varepsilon_{x,t}^{(i)} \sim N(0, \Sigma_x^2)$. The maximum likelihood estimate for the covariance parameter is:

$$\left[\hat{\Sigma}_x^2\right]_{ij} = \frac{1}{T-1} \sum_{t=1}^{T} \left(\hat{\varepsilon}_{x,t}^{(i)} - \bar{\varepsilon}_x^{(i)}\right)\left(\hat{\varepsilon}_{x,t}^{(j)} - \bar{\varepsilon}_x^{(j)}\right). \quad (5)$$

In the second step, transition rates are forecasted and used to estimate future health expectancies. By modeling future transition rates, $\alpha_x^{(i)}$ and $\beta_x^{(i)}$ are assumed to be constant over time, whereas the values of $\kappa_t^{(i)}=[\kappa_1^{(i)}, \kappa_2^{(i)},..., \kappa_t^{(i)}]^1$ are extrapolated using a standard univariate time-series model. Eventually these extrapolated latent factors are inserted into equation (1) to obtain future transition rates.

For modeling and extrapolating the estimated values of $\kappa_t^{(i)}$, Lee and Carter tested several autoregressive integrated moving average (ARIMA) time series models, and they found that the model of random walk (trajectory of successive random steps) with a drift parameter described their data the best. They suggested that different model specifications might be

more appropriate for other data sets however their random walk model with drift is used almost exclusively in applications. We follow Lee and Carter in adopting their projection model. The time series model on the values of $\hat{\kappa}_t^{(i)}$ take the following form:

$$\hat{\kappa}_t^{(i)} = \hat{\kappa}_{t-1}^{(i)} + \theta^{(i)} + \delta_t^{(i)} \tag{6}$$

$$\delta_t^{(i)} \sim N(0, [\Delta^2]_{ii}), \tag{7}$$

where θ is a vector with elements $\theta^{(i)}$, the drift parameter of transition type (i), and Δ^2 is the variance-covariance matrix taking into account the joint tendency of each transition type (i) over time.

The maximum likelihood estimate of the parameter $\hat{\theta}^{(i)}$ and the variance-covariance matrix $[\hat{\Delta}^2]_{ij}$ for the time series model are computed as follows:

$$\hat{\theta}^{(i)} = \frac{\hat{\kappa}_T^{(i)} - \hat{\kappa}_1^{(i)}}{T-1} \tag{8}$$

$$\left[\hat{\Delta}^2\right]_{ij} = \frac{1}{T-1}\sum_{t=1}^{T-1}\left(\hat{\kappa}_{t+1}^{(i)} - \hat{\kappa}_t^{(i)} - \hat{\theta}^{(i)}\right)\left(\hat{\kappa}_{t+1}^{(j)} - \hat{\kappa}_t^{(j)} - \hat{\theta}^{(j)}\right), \tag{9}$$

where $i=(tr,g)\in I$ and $j=(tr,g)\in J$ are transition types at time $t\in\{1,2,...,T\}$ with gender $g\in\{male, female\}$ and $tr\in\{nd,d,inc\}$, the mortality rate of non-disabled, mortality rate of disabled, and the incidence rate, respectively.

Having obtained the parameter estimates of the time series model, one may allow for and take into account parameter uncertainty in the trend itself during the forecasts. In such a case the trend parameters are assumed to have a multivariate normal distribution with $\dot{\theta}\sim N(\hat{\theta},V\{\hat{\theta}\})$, where $\hat{\theta}$ is a vector with the true parameter estimates $\hat{\theta}^{(i)}$, and where the variance-covariance matrix of the parameter estimates is:

$$V\left\{\hat{\theta}^{(i,j)}\right\} = \frac{\left[\hat{\Delta}^2\right]_{ij}}{T-1}. \tag{10}$$

To model future transition rates we used the last-year transition rates as observed in the dataset to be the basis for the forecasts. An alternative approach could have been the use of the estimated last year transition rates but the advantage of the first method is that it avoids a jump-off bias in the first projected year[34]. The transition rates s year from the base year T are given by:

$$\hat{m}^{(i)}_{x,T+1} = m^{(i)}_{x,T} \times RF^{(i)}_{x,T+1} + \hat{\varepsilon}^{(i)}_{x,T+1} \tag{11}$$

where $RF^{(i)}_{x,T,s}$ is the age-x reduction factor between time T and T+s for type of transition rate i. Forecasts of the reduction factor are obtained by the following equation:

$$RF^{(i)}_{x,T,s} = \exp\left(\hat{\beta}^{(i)}_x \times \left(\hat{\kappa}^{(i)}_{T+s} - \hat{\kappa}^{(i)}_T\right)\right), \tag{12}$$

where $\hat{\beta}^{(i)}_x$ denotes the estimated $\beta^{(i)}_x$, and $\hat{\kappa}^{(i)}_{T+s}$ denotes the forecasted reduction factor s ≥1 periods ahead of $\hat{\kappa}^{(i)}_T$. $\hat{\kappa}^{(i)}_{T+s}$ has the following conditional distribution:

$$\hat{\kappa}^{(i)}_{T+s} \left| \hat{\kappa}^{(i)}_{T+s-1}, \dot{\theta}^{(i)} \sim N\left(\hat{\kappa}^{(i)}_{T+s-1} + \dot{\theta}^{(i)}, \hat{\Delta}^2\right) \right. . \tag{13}$$

Once simulations of future transition rates are obtained, they can be converted into one-year transition probabilities taking into account the competition between the rates (Appendix 5B). For each set of forecasted transition probability profiles a multistate life table can be set up and corresponding $DFLE_{x,t}$ can be estimated. Furthermore, the several probabilistic simulations yield prediction intervals for the life expectancy estimates, and the simulated health expectancies allow calculating the probability of a specific scenario of compression or expansion of disability.

Application

We applied the method to Dutch population data which came from several sources because there was no single longitudinal data set available that could have provided all the necessary transition probabilities. Therefore, we used official mortality statistics pertaining to the whole population, prevalence of disability and estimates of the hazard ratio of the mortality risk between disabled and non-disabled. We made use of the simple relationships between mortality, prevalence and hazard ratio to obtain state-specific mortality rates in the first step, and to estimate incidence rates given prevalence and mortality rates in the second step[179].

• *Mortality*

Survival probabilities for Dutch men and women by age (i.e., $x \in \{55.5,56.5,...,96.5,97.5\}$) were used, as published at Statistics Netherlands for each year between 1989 and 2007 (i.e., T=19). The online database of Statistics Netherlands contains original calculations of one-year mortality probabilities and life tables for the Netherlands. The input data consist of death counts from vital statistics, birth counts, and population numbers.

• *Prevalence of disability*

Prevalence of disability was estimated using the POLS health and labor survey collected among the community-dwelling population of the Netherlands[73]. The POLS is an ongoing annually conducted cross-sectional survey aiming to provide information on a broad range of topics concerning the living situation representative of the Dutch general population. The POLS is sampled on records from a centralized municipal registry, and does not include the institutionalized population (less than 2% below age 75 among both men and women). Self-reported health data was collected by face-to-face interviews and written questionnaires. The interviewer visited the participants at home, asked for informed consent and left a written (drop-off) questionnaire. The annual net participation is approximately 10,000 individuals, with response rates of around 60% for the questionnaire. We used POLS surveys conducted between 1989 until 2007 because the current disability questions were first introduced in 1989 and we had access to data until 2007. To correct for selective non-response and to ensure representativeness for the Dutch population, we used POLS sample weights[180]. Data from the health and work module of POLS were available for those aged 12 or older. Table 5.1 shows the population characteristics by gender.

Disability status was measured by the OECD indicator[75], in persons aged 55 years and older. The OECD disability indicator, combining the aspects of basic activities of daily living (ADL) and mobility limitations, measures the ability to perform tasks necessary for both physical functioning and for independent living. In this respect the OECD indicator measures disability on a less severe level than the ADL disability indicator but measures it on a more severe level than the instrumental activities of daily living (iADL) indicator. The OECD disability indicator uses 7 items (conversing, reading small letters, recognizing faces, biting, carrying objects, walking 400ms, bending). For each item respondents were asked if they were able to perform the activities 'without difficulty', 'with minor difficulty', 'with major difficulty', or 'only with help'. Using equipment such as eyeglasses or hearing aid was not indicative of disability if the respondent did not need help or was able to carry out the activity with little or no difficulty. Disability was defined as having at least one item answered: 'with major difficulty' or 'only with help'. Overall item non-response was fairly low, 14.4%.

Table 5.1 POLS, Study population and prevalence of disability by sex

Survey response	Year																		
	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007
Survey																			
POLS	8,284	7,342	6,942	8,763	8,408	8,823	9,352	8,738	10,898	9,323	9,877	9,922	9,676	9,745	9,876	11,117	10,378	9,607	8,741
Response (%)	58.5	56.3	56.7	56.7	55.0	56.1	58.6	56.6	59.4	58.1	55.9	55.0	61.8	61.2	58.3	61.3	65.0	66.4	63.9
Women (%)	50.8 (4,208)	50.5 (3,706)	51.2 (3,555)	51.1 (4,475)	51.0 (4,290)	50.9 (4,489)	51.5 (4,814)	51.3 (4,484)	50.7 (5,530)	51.2 (4,774)	50.8 (5,013)	50.7 (5,026)	50.8 (4,915)	51.7 (5,034)	50.7 (5,006)	51.1 (5,681)	51.1 (5,303)	51.0 (4,902)	51.7 (4,520)
Study population - men																			
55+	855	762	754	894	905	879	991	937	1,113	945	1,033	1,078	1,055	1,121	1,204	1,341	1,278	1,236	1,088
Disabled (%)	23.4%	23.6%	23.5%	25.4%	24.4%	25.4%	26.6%	20.0%	19.8%	17.8%	17.8%	19.4%	19.7%	19.3%	18.4%	21.0%	18.6%	20.1%	17.0%
Study population - women																			
55+	998	928	922	1,094	1,090	1,101	1,166	1,095	1,204	1,044	1,130	1,119	1,132	1,216	1,301	1,442	1,395	1,362	1,232
Disabled (%)	38.2%	37.4%	38.4%	38.0%	37.1%	37.9%	40.4%	34.3%	33.4%	31.1%	33.5%	30.1%	30.5%	27.7%	30.5%	31.0%	31.1%	28.8%	28.3%

Population-level unadjusted prevalence of OECD disability gradually decreased between 1989 and 2007. For men, it fell from 23.4% to 17.0%, whereas for women it declined from 38.2% to 28.3%. The prevalence of disability for each sex and calendar year was smoothed by logistic regressions using a dummy variable for each sample year. The aim of smoothing was to capture the important patterns in the prevalence of disability while leaving out noise due to the sampling design (independent sample every year) of the POLS survey. Logistic regression is commonly used way to smooth disability prevalence. The Akaike Information Criteria indicated that a model including squared age and / or interaction variables would have been less appropriate.

• *Hazard ratio of disabled persons on death*

A unique key for all respondents in the POLS between 1997 and 2006 was provided, which allowed the linking of individuals to the municipal population registries[68]. The available population registries contain annual data on the date of death in the population until December 31, 2007. Records of POLS and population registries were linked deterministically to establish the date of death during the study follow-up period. Those who were not identified in the death registry were considered to be alive at the end of the study follow-up period.

The relative risk of disability on mortality was estimated using the record-linked survival dataset with Cox regression models, stratified by survey year, and estimated for men and women separately. The time scale of the survival analyses was defined as a person's age[76]. Left truncation was applied to the age range over which the subject was not observed before the inclusion to the POLS survey[77]. We did not find significant age-interaction and time trend in the hazard ratios therefore we considered them as being constant over age and time. Hazard ratio of disability on mortality was 1.85 (CI: 1.66, 2.07) and 1.72 (CI: 1.50, 1.97) for men and women, respectively.

• *Estimating transition rates*

Because information on mortality rates of non-disabled and disabled populations separately was not available from primary data sources we decomposed total mortality using the prevalence of disability and the hazard ratio of disability on mortality. We assumed that 1) the age and sex-specific mortality rates in the overall population are the weighted average of mortality rates of non-disabled, $m_{x,t}^{(nd)}$, and disabled populations, $m_{x,t}^{(d)}$, with the proportion of non-disabled and disabled respectively as weights, and 2) that the ratio between the mortality rate of disabled and non-disabled people is equal to the hazard ratio. The corresponding age and time-specific incidence rates, $m_{x,t}^{(inc)}$, could be derived from given mortality rates of the non-disabled and disabled of age x at time t, and given prevalence of disabled population at age x and x+1 at time t, because these quantities are interrelated and

mutually define each other. Appendix 5C presents a formal derivation of these transition rates.

Although our model assumes that only incidence is possible, there is evidence that people can recover from disability, even at higher ages[181]. Therefore, the probability of incidence in our model can be interpreted as a modified net incidence probability, which corresponds to the number of transitions from non-disabled to disabled state minus the number of transitions from disabled to non-disabled state, relative to the number of non-disabled people.

Figure 5.1 shows the incidence probabilities and mortality probabilities of the non-disabled for a number of different ages for men and women between 1989 and 2007, where we normalized the transition probabilities to the year 1989. Because our decomposition of total mortality rates assumes that mortality rates of disabled are constant multiples of the mortality rates of non-disabled, where the multiplier is the hazard ratio, the normalized mortality probabilities are identical for these two groups. Consequently, we only show the graphs for the non-disabled. The figures clearly illustrate that over longer periods, the transition probabilities decrease, reflecting the decrease of prevalence of disability, and the increase in LE over time. The figures also show that the decreases in mortality were substantially larger for men, especially at younger age groups (60, 70).

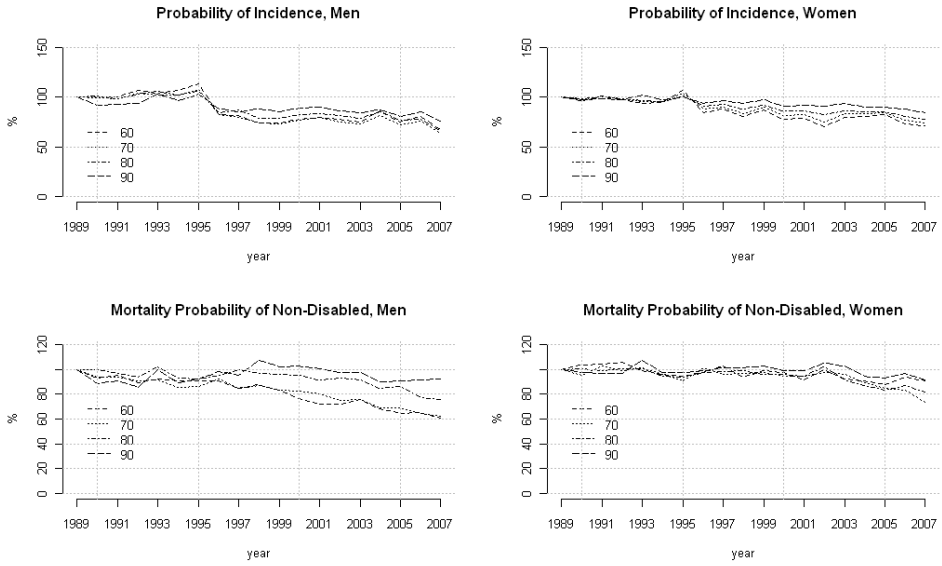


Figure 5.1 Normalized transition probabilities at age 60, 70, 80 and 90, 1989-2007

There are two implicit assumptions in our estimation of transition rates. First, our prevalence estimates are based on survey data that does not include institutionalized individuals hence our prevalence figures are somewhat underestimated. Second, we assume that the hazard ratio is constant over time which may not be valid. Sensitivity of our results to these assumptions is discussed explicitly later.

• *Fitting Lee-Carter model on the transition rates and applying the multistate method*

We fitted a separate Lee-Carter model on each of the six sets of age-specific transition rates to estimate the model parameters, including $\hat{\kappa}_t^{(i)}$. Based on the predicted values of $\hat{\kappa}_t^{(i)}$ we estimated six drift parameters and the 6-by-6 (men and women together) variance-covariance matrix indicating the size of their joint distribution. This latter one allowed for taking into account the joint tendency of the transition rates during the forecasts. By simulating future transition rates we used the last year transition rates as observed in 2007 to avoid a jump-off bias. Once we obtained the simulated transition rates we converted them into one-year transition probabilities, and set up a multistate life table to get health expectancy estimates.

• *Model validation*

We used the R^2 statistic to measure how large proportion of the variation in the different transition rates could be explained by the Lee-Carter models. Furthermore we performed two types of analysis to assess how well the model fitted past life expectancy and disability-free life expectancy based on official sources published by Statistics Netherlands. In the first analysis, we plotted our life and health expectancy estimates against the official statistics between 1989 and 2007, the period on that we had data. In the second analysis, we backcast LE and DFLE by our model for the years between 1983 and 1988, and compared these estimates with those of the Statistics Netherlands.

• *Model outcomes*

The model can be used to forecast numerous outcomes: i) transition probabilities, ii) prevalence of disability, iii) total life expectancy ($LE_{55,t}$), iv) total life expectancy of non-disabled and of disabled v) disability-free life expectancy ($DFLE_{55,t}$), vi) difference between total LE and DFLE ($LE_{55,t}$ - $DFLE_{55,t}$) vii) proportion of DFLE in total LE ($DFLE_{55,t}$ / $LE_{55,t}$). Estimating total $LE_{55,t}$ and $DFLE_{55,t}$ enabled us to assess the likelihood of future compression or expansion of disability.

We assessed the role of uncertainty in the projections from a number of sources: first, the uncertainty of the parameters for predicting prevalence and hazard ratios; second, the uncertainty of the evolution of transition profiles over time; and third, the uncertainty of the trends themselves.

For a reference deterministic model we assumed that the prevalence of disability and the hazard ratios were known with certainty. Since these two ingredients were used to decompose total mortality and to calculate incidence rates, this assumption actually implied that we treated the transition rates as if we had observed them in the whole Dutch population. We further assumed that the future development of transition rates, $\hat{\kappa}^{(i)}_{T+s}$, was also known with certainty. Each year the transition rates changed according to the drift factor, $\hat{\theta}^{(i)}$. We refer to this model as the 'Deterministic model'.

In the first step of our analysis of uncertainty we relaxed some of the assumptions of the Deterministic model: we took into account the fact that the calculation of transition rates was based on estimates of hazard ratios and odds ratios. We applied probabilistic sensitivity analysis to take parameter uncertainty into account, and we drew random hazard ratios and odds ratios 100 times. After each random draw we obtained a set of transition rates and corresponding DFLE estimates. The simulated variation in the DFLE estimates was summarized by prediction intervals that implicitly reflect the effect of the variability of the Lee-Carter model parameters. We refer to this model as 'Model [1]'.

In the second step we relaxed the assumptions we made about future realizations of the transition rates. Here, we took into account that future developments of transition rates are uncertain given a fixed trend. We drew 50 random odds ratios and hazard ratios to simulate the variation in the transition rates. Given a particular set of these, and based on which the trend of evolution was estimated, we simulated the uncertainty in the future evolution of the transition rates 50 times by probabilistic sensitivity analyses. We refer to this model as 'Model [2]'.

In the third step, we also relaxed the assumption about fixed trends. We simulated random sets of transition rates, based on a particular set of transition rates we simulated random trends, and conditional on a particular realization of the trend, we simulated the uncertainty in the evolution of future transition rates. Each part of the simulation was carried out 50 times, and since the simulation contained multiple loops this resulted in a total number of 125,000 random draws. We refer to this model as 'Model [3]'.

5.3. Results

Parameter estimation

Parameter estimates of the Lee-Carter model for the different transitions are plotted in Figure 5.2. The first column of the graph depicts the empirical average of the age-specific transition rates. The second column shows the age profiles, indicating which rates change rapidly or slowly in response to the time dependent evolution of the transition rates. This latent evolution is quantified in the third column.

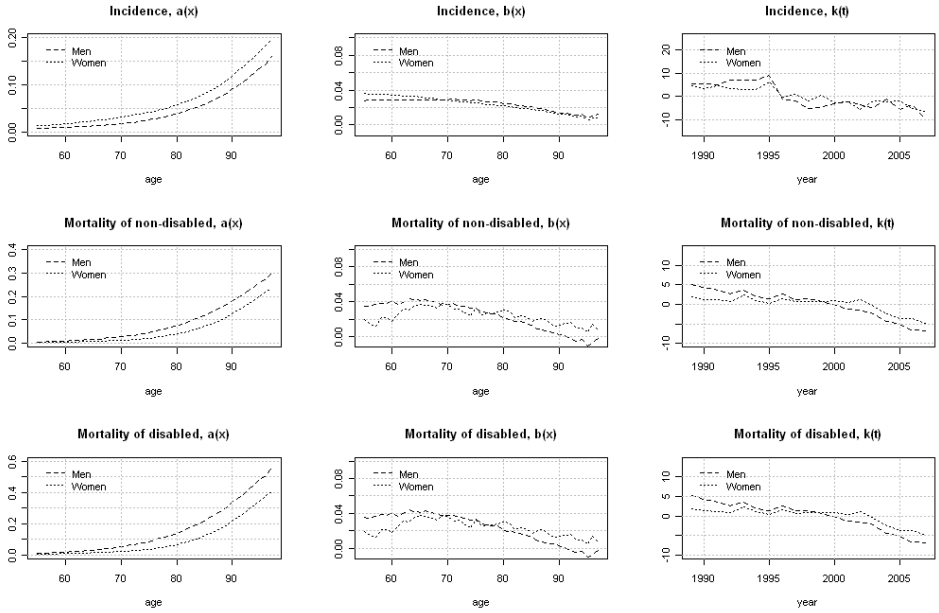


Figure 5.2 Parameter estimates of the Lee-Carter model

On average women had higher incidence rates than men, and for both sexes the incidence rates decreased between 1989 and 2007. Incidence rates decreased relatively faster than mortality rates (Figure 5.2., third panel), which, is consistent with the fact that healthy life expectancy increased.

During the period 1989-2007 both types of mortality rates decreased; the decrease was faster for men than for women. Due to our decomposition method the pace of the decrease was the same for both non-disabled and disabled mortality rates. In absolute terms however, mortality rate of disabled people decreased more than non-disabled mortality rates.

The parameter estimates for the time series models on the values of $\hat{\kappa}_t^{(i)}$ are given in Table 5.4 in Appendix 5D.

Figure 5.3 presents the core results of the model from which life- and health expectancies are derived. We depicted age profiles of one-year incidence probability, mortality probability of non-disabled, and prevalence of disability in 1989, 2007 and their expected value in 2030. The graphs clearly show that the likely increase of LE and DFLE will be the combined result of a decreasing disability incidence and a decreasing mortality, among both men and women.

Forecast and validation

We used the R^2 statistic to measure how large proportion of the variation in the data was explained by the model. We refer the reader to Appendix 5E for figures which shows the age-specific and overall R^2 estimates.

An alternative way of assessing model fit is to compare internally obtained results with external statistics. We performed two types of analysis to assess how well the model fits past LE and DFLE measures based on official sources published by Statistics Netherlands. In the first analysis, we plotted our life and health expectancy estimates against the official statistics between 1989 and 2007, the period on that we had data (Figure 5.4 and 5.5, period 1989-2007). In the second analysis, we back-cast LE and DFLE by our model for the years between 1983 and 1988, and compared these estimates with those of the Statistics Netherlands. If the estimates of Statistics Netherlands fall into the prediction intervals of our model, then the model can be considered valid, because the model not only predicts in-sample outcomes (LE and DFLE between 1989 and 2007) but also out-of-sample outcomes (LE and DFLE between 1983 and 1988). We presented the results in Figure 5.4 and Figure 5.5.

According to official statistics, LE at 55 increased faster among men (+3.7 years, from 21.1 to 24.8) than it did among women (+1.9 years, from 26.8 to 28.7) between 1983 and 2007.

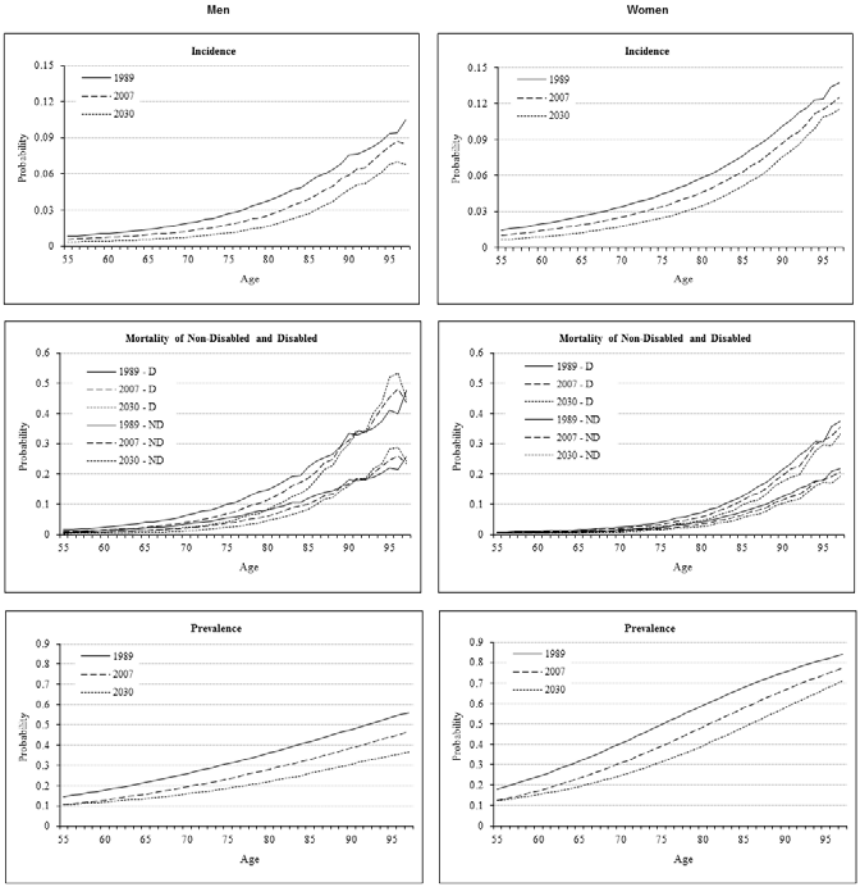


Figure 5.3 Probability of incidence, (non-)disabled mortality and prevalence in 1989, 2007 and 2030, men and women
Notes: ND indicates non-disabled; D indicates disabled

Based on estimates of Statistics Netherlands for the period 1989-2007, DFLE increased with approximately the same extent among both men (from 16.1 to 19.9) and women (from 15.8 to 19.1). Statistics Netherlands also published DFLE for years preceding 1989 (1983 -1988), however these time series contain a 3-year period (1986-1988) with a considerably different disability questionnaire as compared to the one employed before 1986 and since 1989. We decided not to use these years in our model whereas Statistics Netherlands did publish DFLE estimates for these years, applying a number of adjustment techniques to take into account the breaks in the time series[180,182]. These point estimates and confidence intervals are depicted in Figure 5.4 and Figure 5.5.

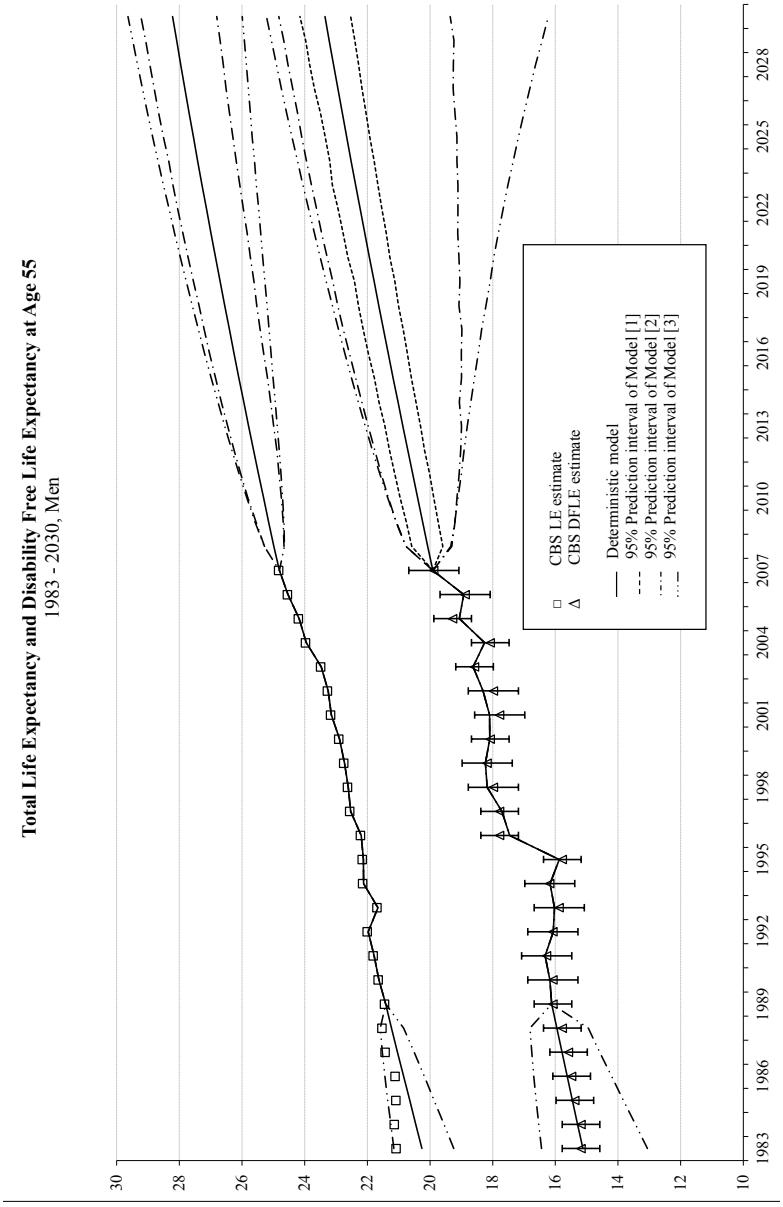


Figure 5.4 Total life expectancy and disability free life expectancy at age 55, 1983-2030, men
Notes: Model [1] allows for uncertainty in prevalence and HR. Model [2] allows for uncertainties of Model [1] and uncertainty in process. Model [3] allows for uncertainties of Model [2] and uncertainty in parameters. CBS: Statistics Netherlands.

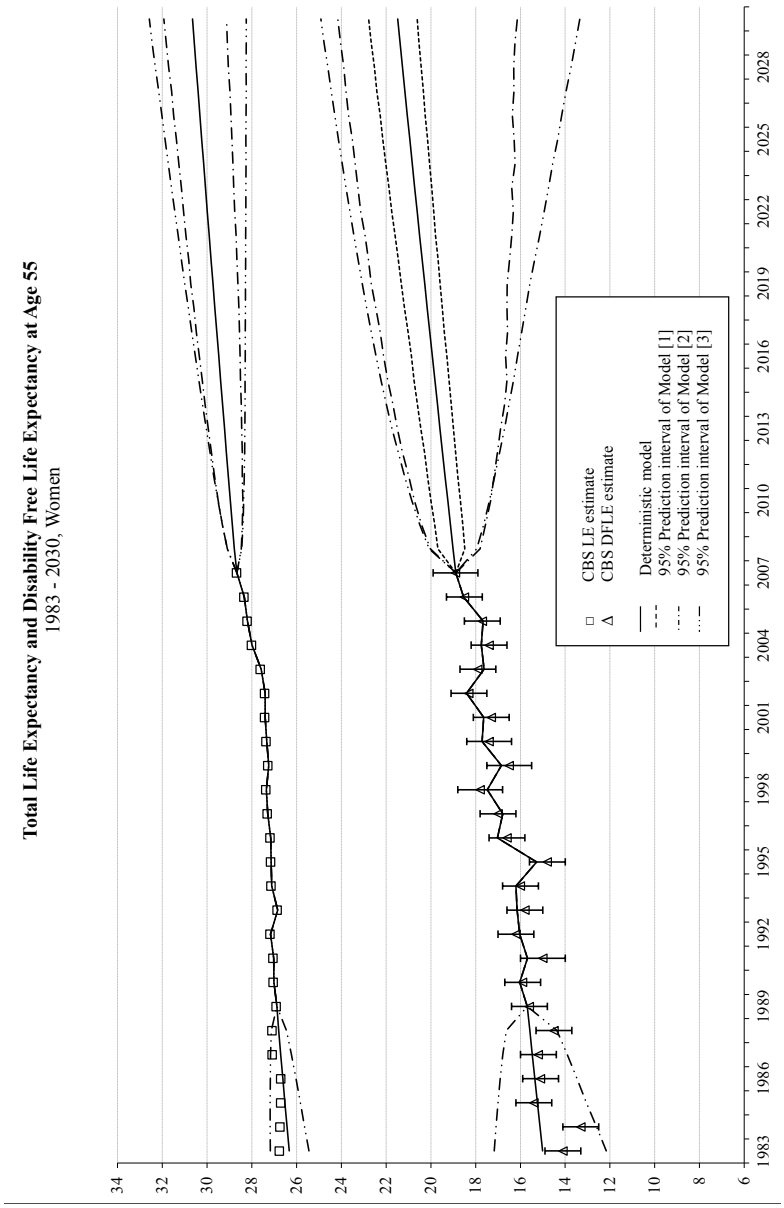


Figure 5.5 Total life expectancy and disability free life expectancy at age 55, 1983-2030, women
Notes: Model [1] allows for uncertainty in prevalence and HR. Model [2] allows for uncertainties of Model [1] and uncertainty in process. Model [3] allows for uncertainties of Model [2] and uncertainty in parameters. CBS: Statistics Netherlands.

Regarding the model fit in terms of estimated LE and DFLE between 1989 and 2007, it is clear even by visual inspection that fitting Lee-Carter model on the transition rates result in excellent model fit to reproduce past life expectancy and disability-free life expectancy as published by Statistics Netherlands. Concerning the back-cast LE and DFLE values, projected prediction intervals of our model contained estimates from Statistics Netherlands, hence the model can be considered valid for this data set.

Future projections of life expectancy

Figures 5.4 and 5.5 show how the various types of uncertainties build up the prediction intervals around the deterministic estimates for future values of total LE and DFLE (period 2008-2030 in the graphs). Tables 5.2 and 5.3 detail how LE and DFLE at age 55 is anticipated to increase for men and women until 2030. According to our projections men's LE will increase from 24.8 in 2007 to 26.8 years by 2020 and to 28.2 years by 2030. Taking all the uncertainty into account the 95% prediction interval lies between 25.3 and 28.0 years by 2020, and 26.0 and 29.6 years by 2030. These projections correspond to a minimum of 0.5 and 1.2, or a maximum of 3.2 and 4.8 years of increase in LE by 2020 and 2030, respectively.

The projected increase in LE for women is somewhat smaller than it is for men. Our model predicts that LE is likely to increase from 28.7 in 2007 to 29.8 and 30.6 by 2020 and 2030, respectively. However, a decrease in LE has some marginal possibility, resulting in a LE of 28.3 and 28.2 with 2.5% probability by 2020 and 2030, respectively. Conversely, a large increase has some minor potential too, which would yield a LE of 31.1 and 32.6 by 2020 and 2030, respectively.

Tables 5.2 and 5.3 present LE estimates for non-disabled and disabled people at age 55 and onwards, both for the past and for the future. Between 1990 and 2005 LE of non-disabled men increased from 22.9 (CI: 22.6-23.3) to 25.2 (CI: 24.9-25.4) years, whereas LE of disabled men increased from 17.8 (CI: 17.3-18.3) to 20.2 (CI: 19.7-20.8) years. Further increases are expected in the future. Between 2010 and 2030 LE of non-disabled men is projected to increase from 26.2 (PI: 25.3-26.9) to 28.8 (PI: 26.2-30.7) years, whereas LE of disabled men is forecasted to rise from 21.2 (PI: 20.2-22.2) to 24.3 (PI: 21.2-26.9) years.

LE of non-disabled and disabled women also increased between 1990 and 2005. Whereas the former individuals expected to live 28.7 (CI: 28.4-29.3) years in 1990, this expectation increased to 29.7 (CI: 29.3-30.2) by 2005. Corresponding values of the disabled were 24.4 (CI: 24.0–24.8) in 1990 and 25.5 (CI: 25.0-25.8) in 2005. With regard to the future, LE of the non-disabled is forecasted to increase between 2010 and 2030, from 30.2 (PI: 29.4-31.1) to 31.6 (PI: 28.9-33.8). This increase is anticipated to be approximately the same for the disabled, from 26.1 (PI: 25.0-26.9) to 27.6 (PI: 24.4-30.3).

Table 5.2 Disability-related life expectancy measures for selected years, men

Year	Life Expectancy (Total)		Life Expectancy (Non-disabled)		Life Expectancy (Disabled)		Disability-Free Life expectancy		Life Expectancy with Disability (LwD)		DFLE / LE (%)		Probability of Compression of Disability (%)	
													(A)	(R)
1990	21.6	-	22.9	22.6 - 23.3	17.8	17.3 - 18.3	16.2	15.6-16.8	5.4	4.8-6.0	74.8	72.1 - 77.6	-	-
1995	22.1	-	23.6	23.2 - 24.0	18.5	18.0 - 18.9	15.9	15.3-16.5	6.2	5.6-6.8	71.8	69.3 – 74.5	-	-
2000	22.9	-	23.9	23.6 - 24.2	18.9	18.3 - 19.4	18.1	17.6-18.6	4.8	4.3-5.3	79.0	77.0 – 81.1	-	-
2005	24.2	-	25.2	24.9 – 25.4	20.2	19.7 - 20.8	19.1	18.6-19.4	5.1	4.8-5.6	78.9	77.0 – 80.3	-	-
2010	25.3	24.7 - 25.8	26.2	25.3 - 26.9	21.2	20.2 - 22.2	20.4	19.1 - 21.3	4.9	3.9-6.0	80.6	75.3 - 84.8	48.9	55.3
2015	26.1	25.0 - 27.0	26.9	25.5 - 28.1	22.1	20.4 - 23.6	21.2	18.6 - 22.5	4.9	3.4-7.4	81.1	70.9 - 86.7	48.9	58.7
2020	26.8	25.3 - 28.0	27.6	25.7 - 29.1	22.8	20.7 - 24.8	21.9	18.0 - 23.5	4.9	3.0-9.0	81.7	66.6 - 88.3	49.2	60.6
2025	27.6	25.7 - 28.4	28.2	26.0 - 29.9	23.6	21.0 - 25.9	22.7	17.2 - 24.4	4.9	2.9-10.5	82.2	62.2 - 89.2	49.8	62.0
2030	28.2	26.0 - 29.6	28.8	26.2 - 30.7	24.3	21.2 - 26.9	23.4	16.2 - 25.2	4.9	2.7-12.0	82.8	57.6 - 89.8	50.3	63.1

Notes:

[a] For the years between 1990-2005 we provide 95% confidence intervals; for the years 2010-2030 we provide 95% prediction intervals based on Model [3] which allows for uncertainty in prevalence and HR, uncertainty in process and uncertainty in the trend parameters.

[b] Size of absolute compression: $(LE_{55,T} - DFLE_{55,T}) - (LE_{55,2007} - DFLE_{55,2007}) = LwD_{55,T} - LwD_{55,2007}$; measured in number of years

[c] Size of relative compression: $(DFLE_{55,T} / LE_{55,T}) - (DFLE_{55,2007} / LE_{55,2007})$; measured in percentage points

[d] A is an abbreviation for absolute, R is an abbreviation for relative

Adding the presented estimates of DFLE and LwD may not reproduce LE exactly because of rounding

Table 5.3 Disability-related life expectancy measures for selected years, women

Year	Life Expectancy (Total)		Life Expectancy (Non-disabled)		Life Expectancy (Disabled)		Disability-Free Life expectancy		Life Expectancy with Disability (LwD)		DFLE / LE (%)		Probability of Compression of Disability (%)	
													(A)	(R)
1990	27.0	-	28.7	28.4-29.5	24.4	24.0-24.8	16.0	15.3-16.8	11.0	10.2-11.7	59.3	56.7-62.2	-	-
1995	27.1	-	29.0	28.6-29.5	24.7	24.2-25.0	15.3	14.6-15.8	11.8	11.3-12.5	56.3	54.0-58.3	-	-
2000	27.3	-	28.8	28.5-29.3	24.5	24.0-24.9	17.7	17.3-18.2	9.6	9.1-10	64.8	63.2-66.4	-	-
2005	28.2	-	29.7	29.3-30.2	25.5	25.0-25.8	17.7	17.2-18.1	10.5	10.1-11.2	62.7	61.0-64.4	-	-
2010	29.0	28.4-29.5	30.2	29.4-31.1	26.1	25.0-26.9	19.2	17.4-20.9	9.7	7.9-11.7	66.5	59.8-72.4	56.2	60.5
2015	29.4	28.3-30.4	30.6	29.3-31.9	26.4	24.8-27.7	19.8	16.3-22.2	9.6	6.8-13.3	67.4	55.1-76.3	57.4	63.6
2020	29.8	28.3-31.1	30.9	29.1-32.6	26.8	24.6-28.7	20.4	15.4-23.3	9.4	6.1-14.8	68.4	51.0-79.2	58.1	65.1
2025	30.2	28.3-31.9	31.3	29.0-33.2	27.2	24.5-29.5	20.9	14.3-24.1	9.3	5.5-16.4	69.3	46.8-81.2	58.8	66.2
2030	30.6	28.3-32.6	31.6	28.9-33.8	27.6	24.4-30.3	21.5	13.3-24.9	9.2	5.0-17.9	70.1	42.9-82.8	59.2	67.0

Notes:

[a] For the years between 1990-2005 we provide 95% confidence intervals; for the years 2010-2030 we provide 95% prediction intervals based on Model [3] which allows for uncertainty in prevalence and HR, uncertainty in process and uncertainty in the trend parameters.

[b] Size of absolute compression: $(LE_{55,T} - DFLE_{55,T}) - (LE_{55,2007} - DFLE_{55,2007}) = LwD_{55,T} - LwD_{55,2007}$; measured in number of years

[c] Size of relative compression: $(DFLE_{55,T} / LE_{55,T}) - (DFLE_{55,2007} / LE_{55,2007})$; measured in percentage points

[d] A is an abbreviation for absolute, R is an abbreviation for relative

Adding the presented estimates of DFLE and LwD may not reproduce LE exactly because of rounding

Tables 5.2 and 5.3 present estimates of DFLE both in terms of number of years and as a proportion of total LE. DFLE of men is projected to increase from 19.1 (CI: 18.6-19.4) in 2005 to 21.9 (PI: 18.0-23.5) and 23.4 (PI: 16.2-25.2) years by 2020 and 2030, respectively. DFLE relative to LE is forecasted to rise from 78.9% in 2005 to 81.7% and to 82.8% during the same period. Expectations about increases in DFLE for women are similar to those of men. It is anticipated that DFLE will increase from 17.7 (CI: 17.2-18.1) to 20.4 (PI: 15.4-23.3) years between 2005 and 2020, and further to 21.5 (PI: 13.3-24.9) years by 2030. These changes correspond to increases in the DFLE / LE ratio from 62.7 % to 68.4% by 2020 and to 70.1% by 2030.

The jointly simulated DFLE and LE estimates made it possible to calculate the probability that either compression or expansion of disability will occur in the future. Tables 5.2 and 5.3 present the likelihood of compression of disability. We expressed compression of disability in terms of both absolute and relative value. In the first case, compression of disability would occur if the increase in DFLE was larger than the increase in total LE, that is, a reduction in the years with disability. If the compression of disability is interpreted in a relative sense, then such a compression would occur if the proportion of disability-free life years to total life years would increase over time.

When compression of OECD disability is measured in years, then the probability of its occurrence by 2030 is approximately 50% for men, and 60% for women. In other words, among women the number of years lived without OECD disability is more likely to increase slightly faster than the number of years lived in total. The picture is somewhat brighter if compression of disability is measured in a relative sense. According to the projections it is more likely that disability-free life years as a proportion of total LE would increase; the probability of compression is 63% for men, 67% for women.

5.4. Discussion

We proposed a theoretical framework for a multi-state life table model, in which the transition probabilities depended on age and calendar time. We described how to model and project these transition rates by the Lee-Carter method, and illustrated how it could be used to forecast future health expectancies including prediction intervals. We applied the model to the Dutch population aged 55 and older, and estimated health expectancies between 1989 and 2030. Additionally, we analyzed the changing relationship between DFLE and LE over time, and attached probability distributions to different future scenarios of compression or expansion of disability.

Explanation

There are several reasons to believe why DFLE will keep increasing in the future. Favorable trends in tobacco consumption[183], dietary habits restricting the intake of saturated fats[184], physical exercise[185], changes in the composition of the population by socio-economic status and advances in medical technology have all contributed to improving individual risk profiles and better health status of the Dutch populations. These trends are likely to continue in the future.

On the contrary, there are unfavorable health trends as well. Particularly worrying is the fact that the share of overweight population is continuously increasing, and that the dietary habits of adolescents change adversely[185]. These trends will have an increased impact on morbidity, as diabetes, cardiovascular and musculoskeletal diseases, as well as various types of cancers have been shown to be associated with obesity[186]. Two demographic trends may also have a negative effect on health expectancy: the increasing instability of social relations and the changing ethnic composition with higher share of groups with non-western origin.

Other studies

Many studies have estimated HEs in the past using various study populations, disability measures and calendar periods. The most extensive work in assessing the evolution of past HE has been conducted for the U.S. population. Crimmins et al. estimated that gains in LE during the 1970s were mainly accompanied by increasing time spent with chronic limitations of common activities and by slight decreasing time with severe disability[17]. Later, Crimmins et al. found that during the 1980s gains in LE rose along with DFLE for both men and women[18]. In 2009 Crimmins et al. examined changes in LE with and without ADL and iADL disability using longitudinal data between 1984 and 2000[19]. They showed that the increase in DFLE at age 70 was the same as the increase in LE. Our results accord with these findings.

In Europe estimates of LE and DFLE for men and women were published for 13 European Union member states from 1995 until 2001 based on the European Community Household Panel[20]. Significant increases were found in LE at early (age 16) and late (age 65) adulthood with considerable heterogeneity in the trends in health expectancy. In nine countries life expectancy with disability increased, whereas four countries had evidence of decreasing life expectancy with disability.

Previous studies on trends in health expectancy in the Netherlands[23,187,188] used various health status indicators, time periods and age groups for their analyses and resulted in diverse conclusions on the trends. A recent study by the Statistics Netherlands on health expectancy trends at age 65 however used the same data set as we did but here morbidity

was measured in three different ways: self-rated health, presence of chronic disease and OECD disability. The results show that since the 1980s health expectancy at age 65 measured in terms of good self-reported health or DFLE has been increasing for men. The increase has been somewhat faster since the early 2000s. Unfortunately, among both men and women, life expectancy without chronic diseases has been decreasing. For women, DFLE increased and years with good self-rated health stagnated.

Sensitivity analyses

A limitation of the POLS data is that the annual samples do not include the institutionalized population, among whom the prevalence of disability is higher than in the general population. We performed sensitivity analyses to assess the potential bias on the DFLE forecasts caused by this limitation. Information on the number of institutionalized persons for every age and year between 1995 and 2007 was available on the website of Statistics Netherlands (Statline). Using this information we calculated the age-specific prevalence of institutionalized population for each year and ran a new model using only the period 1995-2007. We assumed that everybody who was institutionalized was disabled thereby we assessed the maximum bias that exclusion of these people may have caused. Results of the sensitivity analyses revealed that our model overestimates DFLE with a maximum of 0.6 year for men and 0.7 year for women between 1995 and 2007. Similar differences were predicted between the original and the new DFLE forecasts by 2030. Given the uncertainty around these future estimates, the importance of differences can be considered small.

Another limitation of our study comes from the fact that the record-linked POLS data did not have enough power to detect changes in the hazard ratio. We performed additional sensitivity analyses to assess the potential bias that changes in the hazard ratio may have caused. In particular, we assessed the effect of both an annual 1% decrease and 1% increase in the hazard ratio between 1989 and 2007 on our estimates of DFLE. In the first case the HR in 2007 was 83% of the HR in 1989, whereas in the second case it was 120%. The results of the sensitivity analyses indicated that such changes in the hazard ratio virtually had no effect on the original DFLE estimates. These findings were true for men as well as for women.

It is important to note that our definition of disability essentially excludes the possibility of detecting a dynamic equilibrium, because we did not make distinction between severe and mild disability, and because we interpreted future compression of disability in terms of probabilities. A dynamic equilibrium would occur if both incidence and mortality were postponed such that the $LwD_{55,t}$ neither decreased nor increased. Looking at the expected future LwD values for men in Table 5.2 (column LwD), this is exactly what we find. Correspondingly, the probability of compression is very close to 50%, referring to the situation

that an increasing LwD is equally likely as a decreasing LwD. Such a situation in our model seems to indicate a dynamic equilibrium among men.

Model related issues

Every model is a simplification of reality; therefore certain assumptions are made during the construction of the model. One of the assumption of the Lee-Carter model is that the expected evolution of the transition rates over time, $\theta^{(i)}$, depends on the first and the last observation of the latent process, $\hat{\kappa}_1^{(i)}$ and $\hat{\kappa}_T^{(i)}$, and that the expected evolution of the transition rates is merely an extrapolation of the trend estimated on these two end points. Therefore, if for example, the trend of $\hat{\kappa}_t^{(i)}$ is close to linear then choosing other end points would not significantly influence the future evolution of the transition rates. Consequently, the uncertainty around the predictions would be relatively small as well. We see this situation at mortality rates. However, if the trend in $\kappa_t^{(i)}$ is less linear and the observation window is relatively short, then the estimate of $\theta^{(i)}$ might be sensitive on choosing other end points than $\kappa_1^{(i)}$ and $\kappa_T^{(i)}$. Accordingly, the uncertainty around future evolution would be somewhat larger as well. We see such situation at incidence rates.

We assessed the stability of the age-interaction and the trend parameters by defining a different time window on which the parameters were estimated. We eliminated the first three years of the POLS data resulting in the period of 1992-2007. We then re-estimated the model and we found that the parameters were stable. The age-interaction parameters were very close to the original estimates with an average difference of approximately 5%. The effect of trimming the time window of the analysis had little effect on the results. The deterministic forecasts of life expectancy were essentially the same as at the original forecasts, disability-free life expectancy was higher with 0.3 years by 2030.

We have also carried out an analysis in that we forecasted overall life expectancy based on only mortality rates while using the same simple Lee-Carter method as we used for forecasting health expectancy. We then compared these forecasts with the ones from the MSLT projections. We found that the projections were very similar, but larger prediction intervals were estimated around the MSLT projections.

Conclusions

Our finding suggest that the Lee-Carter model is generalizable to multi-state life table settings, and can be used to model transition rates connecting non-disabled, disabled and dead states, and to forecast disability-related health expectancies. However, the application of the generalized Lee-Carter model to different data sets may result in poorer model fit, for example if the time-evolution of transition rates is not linear. Nonetheless, we consider that the model framework presented here can be used in other settings, for example for

other countries. Prevalence of OECD disability may be replaced with ADL, iADL disability, subjective well-being or other prevalence measures, which are good indicators of population health. The approach demonstrated for health expectancies in our study could be used for working life expectancies as well, since working life expectancies are based on similar multistate models. Thus the model framework may be used to forecast population health where the health status is measured in various ways. Besides the application of different health indicators, a specific methodological question may appear on the future research agenda, the inclusion of common trends in the forecasts of transition rates[189].

Appendix 5A Glossary of acronyms

ADL: Activities of daily living
DFLE: Disability-free life expectancy
HE: Health expectancy
HR: Hazard ratio
iADL: Instrumental activities of daily living
LE: Life expectancy
LwD: Life expectancy with disability
MSLT: Multistate life table
OECD: Organisation for Economic Co-operation and Development
POLS: Permanent Onderzoek LeefSituate (in Dutch), Repeated Survey on Living Situation (English)
Statistics Netherlands (in English): Centraal Bureau voor de Statistiek (in Dutch)
SVD: Singular Value Decomposition

Appendix 5B

The original (mortality or actuarial) life table is a transition model in which observed death rates, within age interval, are the basis of probabilities of dying, and in which the main parameter of interest is the expectation of life. A multistate life table (MSLT) model is an extension of the original life table method. In a MSLT not only 'alive' and the absorbing 'dead' states are distinguished but there is at least one additional state, typically between 'perfectly healthy' and 'dead', for example 'disabled'. In contrast to a mortality table, an MSLT not only shows, for each age, what the probability is that a person of that age will die before his next birthday, but rather it shows what the probability is that a person of that age will move from one state to another. Correspondingly, an MSLT not only shows the remaining life expectancy and the proportion of the original birth cohorts still alive at different ages, but rather it shows the remaining life expectancy and proportion of people still alive in a given state. Furthermore, the population-average LE can be decomposed into a weighted average of health expectancies, indicating the number of years people are expected to live in each health state.

Conversion of rates into probabilities taking into account competing risks

In an MSLT the possible transitions are expressed by the matrix, $M^{g}_{x,t}$, standing for the transition rates between the several states, and where g denotes the gender. The matrix $M^{g}_{x,t}$ refers to the chance of moving from the i^{th} state to the j^{th} state in infinitesimal time. However, instead of infinitesimal time intervals, one typically works with longer periods, like one year. In such a case one refers to the transition probability matrix $Q^{g}_{x,t}$, whose q_{ij} element indicate the transition probability that a person at age x at time t is in the i^{th} state and

in the j[th] state one year later. Assuming that the exposure is linear in age, one can convert the transition rates into the appropriate transition probabilities.

1. Transition rates

$M^{g}_{x,t}$ is the matrix of transition rates at age x and time t

$$M^{g}_{x,t} = \begin{bmatrix} m^{(inc,g)}_{x,t} + m^{(nd,g)}_{x,t} & -m^{(rec,g)}_{x,t} \\ -m^{(inc,g)}_{x,t} & m^{(rec,g)}_{x,t} + m^{(d,g)}_{x,t} \end{bmatrix} \tag{1}$$

where inc, rec, nd and d represents the transition from non-disabled to disabled, from disabled to non-disabled, from non-disabled to dead, and from disabled to dead, respectively,

2. Transition probabilities

Using linear approximation: that is, all transitions (incidence, deaths) occur in the middle of the interval.

$$Q^{g}_{x,t} = \frac{I - \frac{1}{2}M^{g}_{x,t}}{I + \frac{1}{2}M^{g}_{x,t}} = \begin{bmatrix} 1-\left(q^{(inc,g)}_{x,t} + q^{(nd,g)}_{x,t}\right) & q^{(rec,g)}_{x,t} \\ q^{(inc,g)}_{x,t} & 1-\left(q^{(rec,g)}_{x,t} + q^{(d,g)}_{x,t}\right) \end{bmatrix} \tag{2}$$

$Q^{g}_{x,t}$ is the transition-probability matrix, consisting of elements $q_{ij}(x,t)$ which represents the 1-year probability that an individual with gender g alive at age x and time t will be in state j at age x+1, and $\mathbf{I}$ is a 2x2 identity matrix.

$$q^{(inc,g)}_{x,t} = \frac{m^{(inc,g)}_{x,t}}{\left(1 + \frac{m^{(inc,g)}_{x,t}}{2} + \frac{m^{(nd,g)}_{x,t}}{2}\right)\left(1 + \frac{m^{(d,g)}_{x,t}}{2}\right)} \tag{3}$$

$$q^{(nd,g)}_{x,t} = \frac{m^{(inc,g)}_{x,t} + m^{(nd,g)}_{x,t}}{\left(1 + \frac{m^{(inc,g)}_{x,t}}{2} + \frac{m^{(nd,g)}_{x,t}}{2}\right)} - \frac{m^{(inc,g)}_{x,t}}{\left(1 + \frac{m^{(inc,g)}_{x,t}}{2} + \frac{m^{(nd,g)}_{x,t}}{2}\right)\left(1 + \frac{m^{(d,g)}_{x,t}}{2}\right)} \tag{4}$$

$$q^{(d,g)}_{x,t} = \frac{m^{(d,g)}_{x,t}}{\left(1 + \frac{m^{(d,g)}_{x,t}}{2}\right)} \tag{5}$$

Note: in our model, recovery is set to zero, and incidence is considered as net incidence (real incidence minus real recovery).

Calculating number of non-disabled and disabled alive

1. Number of persons alive

$l^{g}_{x,t}$ is the sum of the number of non-disabled ($l^{(nd,g)}_{x,t}$) and disabled ($l^{(d,g)}_{x,t}$) individuals alive at age x and at time t:

$$l^{g}_{x,t} = \begin{bmatrix} l^{(nd,g)}_{x,t} \\ l^{(d,g)}_{x,t} \end{bmatrix} \tag{6}$$

$l^{g}_{x+1,\,t+1}$ is the sum the number of aged x+1 individuals with gender g alive at time t+1, expressed as a function of the number of individuals alive and transition probabilities at age x and at time t ($l^{(nd,g)}_{x,t}$, $l^{(d,g)}_{x,t}$, $q^{(.tr,g)}_{x,t}$).

$$l^{g}_{x+1,t+1} = \begin{bmatrix} l^{(nd,g)}_{x+1,t+1} \\ l^{(d,g)}_{x+1,t+1} \end{bmatrix} = \begin{bmatrix} l^{(nd,g)}_{x,t} - l^{(nd,g)}_{x,t} q^{(nd,g)}_{x,t} - l^{(nd,g)}_{x,t} q^{(inc,g)}_{x,t} \\ l^{(d,g)}_{x,t} - l^{(d,g)}_{x,t} q^{(d,g)}_{x,t} + l^{(nd,g)}_{x,t} q^{(inc,g)}_{x,t} \end{bmatrix} \tag{7}$$

2. Prevalence of disabled

$p^{g}_{x,t}$ is the prevalence matrix, consisting of elements $p^{(nd,g)}_{x,t}$ and $p^{d,g}_{x,t}$ which represent the proportion of gender g specific population without (nd) and with (d) disability at age x and at time t.

$$p^{g}_{x,t} = \begin{bmatrix} p^{(nd,g)}_{x,t} \\ p^{(d,g)}_{x,t} \end{bmatrix} = \begin{bmatrix} \dfrac{l^{(nd,g)}_{x,t}}{l^{(nd,g)}_{x,t} + l^{(d,g)}_{x,t}} \\ \dfrac{l^{(d,g)}_{x,t}}{l^{(nd,g)}_{x,t} + l^{(d,g)}_{x,t}} \end{bmatrix} \tag{8}$$

$p^{g}_{x+1,\,t+1}$ is the gender g specific prevalence matrix, expressed as a function of the number of individuals alive and transition probabilities at age x and at time t ($l^{(nd,g)}_{x,t}$, $l^{(d,g)}_{x,t}$, $q^{(tr,g.)}_{x,t}$).

$$p^{g}_{x+1,t+1} = \begin{bmatrix} \dfrac{l^{(nd,g)}_{x,t} - l^{(nd,g)}_{x,t} q^{(nd,g)}_{x,t} - l^{(nd,g)}_{x,t} q^{(inc,g)}_{x,t}}{l^{(nd,g)}_{x,t} + l^{(d,g)}_{x,t} - l^{(nd,g)}_{x,t} q^{(nd,g)}_{x,t} - l^{(d,g)}_{x,t} q^{(d,g)}_{x,t}} \\ \dfrac{l^{(d,g)}_{x,t} - l^{(d,g)}_{x,t} q^{(d,g)}_{x,t} + l^{(nd,g)}_{x,t} q^{(inc,g)}_{x,t}}{l^{(nd,g)}_{x,t} + l^{(d,g)}_{x,t} - l^{(nd,g)}_{x,t} q^{(nd,g)}_{x,t} - l^{(d,g)}_{x,t} q^{(d,g)}_{x,t}} \end{bmatrix} \tag{9}$$

Calculating life expectancy of non-disabled and disabled

$L^{g}_{x,t}$ denotes the gender g specific average number of non-disabled and disabled aged x individuals alive at time t

$$L^{g}_{x,t} = \begin{cases} \begin{bmatrix} \frac{l^{(nd,g)}_{x,t} + l^{(nd,g)}_{x+1,t+1}}{2} \\ \frac{l^{(d,g)}_{x,t} + l^{(d),g}_{x+1,t+1}}{2} \end{bmatrix} & \text{if } x < \omega, \\ \begin{bmatrix} \frac{l^{(nd,g)}_{MA,t}}{M^{(nd,g)}_{MA,t}} \\ \frac{l^{(d,g)}_{MA,t}}{M^{(d,g)}_{MA,t}} \end{bmatrix} & \text{if } x = \omega, \end{cases} \tag{10}$$

where ω is the maximum attainable age.

$T^{g}_{x,t}$ denotes the cumulative average number of non-disabled and disabled aged x individuals alive at time t

$$T^{g}_{x,t} = \begin{bmatrix} \sum_{s>0} \frac{l^{(nd,g)}_{x+s,t+s} + l^{(nd,g)}_{x+1+s,t+s}}{2} \\ \sum_{s>0} \frac{l^{(d,g)}_{x+s,t+s} + l^{(d,g)}_{x+1+s,t+s}}{2} \end{bmatrix} \tag{11}$$

Gender g specific life expectancy of non-disabled and disabled are calculated as

$$e^{g}_{x,t} = \begin{bmatrix} \frac{T^{(nd,g)}_{x,t}}{l^{(nd,g)}_{x,t}} \\ \frac{T^{(d,g)}_{x,t}}{l^{(d,g)}_{x,t}} \end{bmatrix} \tag{12}$$

Disability-free life expectancy (DFLE) and life expectancy with disability (LwD) are calculated as

$$\begin{bmatrix} DFLE^{(g)}_{x,t} \\ LwD^{(g)}_{x,t} \end{bmatrix} = \begin{bmatrix} \frac{T^{(nd,g)}_{x,t}}{l^{(g)}_{x,t}} \\ \frac{T^{(d,g)}_{x,t}}{l^{(g)}_{x,t}} \end{bmatrix} \tag{13}$$

Appendix 5C Estimating transition rates

Because information about mortality rates of non-disabled and disabled was not available from primary data sources we decomposed total mortality using the prevalence of disability and the hazard ratio of disability on mortality as follows:

$$\widetilde{m}_{x,t}^{(nd,g)} = \frac{m_{x,t}^{(g)}}{\hat{HR}_{x}^{g} \times \hat{p}_{x,t}^{(d,g)} + \left(1 - \hat{p}_{x,t}^{(d,g)}\right)} \tag{1}$$

$$\widetilde{m}_{x,t}^{(d,g)} = \widetilde{m}_{x,t}^{(nd,g)} \times \hat{HR}_{x}^{g} \tag{2}$$

where $M_{x,t}^{g}$, $\widetilde{m}_{x,t}^{(nd,g)}$, $\widetilde{m}_{x,t}^{(d,g)}$, $\hat{HR}_{x}^{g}$ and $\hat{p}_{x,t}^{(d,g)}$ indicate the gender-g specific population mortality rate, estimated mortality rate of non-disabled and disabled, the estimated hazard ratio and smoothed prevalence of disability at age x and time t, respectively.

The converted transition, $q_{x,t}^{(nd,g)}$ ($q_{x,t}^{(d,g)}$), show the probability that a person is non-disabled (disabled) at age x at time t and is dead at age x+1 at time t. Such formulation of the model implies the period-age approach, which is often used when the main point of interest is the change of transition probabilities over a certain period of time, e.g. calendar years. With the period-age approach it is implicitly assumed that a person of age x at time t will have the same transition probability at age x+1 at time t+1 (assumption) as a person who is of age x+1 at time t (reality). Making such assumption is unavoidably done by estimating period life expectancies.

Estimating incidence rates

Given the prevalence of disabled populations $p_{x,t}^{(d,g)}$, $p_{x+1,t}^{(d,g)}$ of age x and x+1 at time t, the mortality rate of non-disabled $m_{x,t}^{(nd,g)}$ and disabled $m_{x,t}^{(nd,g)}$ of age x at time t, it is possible to calculate the corresponding incidence rate, $m_{x,t}^{(inc,g)}$, since these quantities are interrelated and mutually define each other. The prevalence of disabled population of age x+1 at time t is expressed as the proportion of those who are disabled to those who are alive. However, this fraction is dependent on the number of transitions during age x. That is, the prevalence at age x+1 is a function of the number of people alive ($l_{x,t}^{g}$), the prevalence of disabled and the transition probabilities at age x and at time t:

$$p_{x+1,t}^{(d,g)} = \frac{l_{x,t}^{g} p_{x,t}^{(d,g)} - l_{x,t}^{g} p_{x,t}^{(d,g)} q_{x,t}^{(d,g)} + l_{x,t}^{g} (1 - p_{x,t}^{(d,g)}) q_{x,t}^{(inc,g)}}{l_{x,t}^{g} - l_{x,t}^{g} (1 - p_{x,t}^{(d,g)}) q_{x,t}^{(nd,g)} - l_{x,t}^{g} p_{x,t}^{(d,g)} q_{x,t}^{(d,g)}} \tag{3}$$

Expressing transition probabilities as functions of the transition rates, the incidence rate can be obtained by the following formula after rearranging (3):

$$A^g = \frac{\hat{p}^{(d,g)}_{x+1,t}}{1-\hat{p}^{(d,g)}_{x,t}} - \frac{\hat{p}^{(d,g)}_{x+1,t}\,\hat{p}^{(d,g)}_{x,t}\widetilde{q}^{(d,g)}_{x,t}}{1-\hat{p}^{(d,g)}_{x,t}} - \frac{\hat{p}^{(d,g)}_{x,t}}{1-\hat{p}^{(d,g)}_{x,t}} + \frac{\hat{p}^{(d,g)}_{x,t}\widetilde{q}^{(d,g)}_{x,t}}{1-\hat{p}^{(d,g)}_{x,t}} \tag{4}$$

$$\hat{m}^{(inc,g)}_{x,t} = \frac{A^g\left(1+\frac{\widetilde{m}^{(nd,g)}_{x,t}}{2}\right)\left(1+\frac{\widetilde{m}^{(d,g)}_{x,t}}{2}\right) - \hat{p}^{(d,g)}_{x+1,t}\widetilde{m}^{(mrn,g)}_{x,t}\left(1+\frac{\widetilde{m}^{(d,g)}_{x,t}}{2}\right)}{1+p^{(d,g)}_{x+1,t}\left(1+\frac{\widetilde{m}^{(d,g)}_{x,t}}{2}\right) - \hat{p}^{(d,g)}_{x+1,t} - \frac{A^g}{2}\left(1+\frac{\widetilde{m}^{(d,g)}_{x,t}}{2}\right)}. \tag{5}$$

Then $q^{(inc,g)}_{x,t}$ shows the probability that a person is non-disabled at age x at time t and disabled at age x+1 at time t. Although our model assumes that only incidence is possible, there is evidence that people can recover from disability even at higher ages. Therefore, the probability of incidence in our model can be interpreted as a modified net incidence probability, which corresponds to the number of transitions from non-disabled to disabled state minus the number of transitions from disabled to non-disabled state, relative to the number of non-disabled people.

Appendix 5D

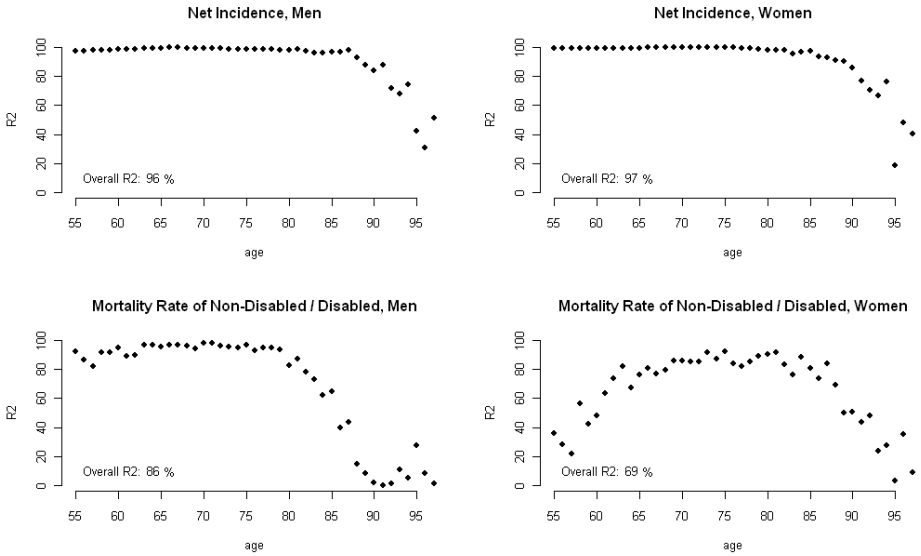
Table 5.4 Parameter estimates in the time trends of the Lee-Carter model

Transition type, i = (tr ,g)	Best estimate trend $\theta^{(i)}$	Standard Deviation, trend $\sqrt{V\{\hat{\theta}^{(i,i)}\}}$	Standard Deviation, innovations $\sqrt{[\Delta^2]_{j,i}}$	Correlation: $\frac{[\Delta^2]_{i,j}}{\sqrt{[\Delta^2]_{i,i}}\sqrt{[\Delta^2]_{j,j}}} = \frac{V\{\hat{\theta}^{(i,j)}\}}{\sqrt{V\{\hat{\theta}^{(i,j)}\}}\sqrt{V\{\hat{\theta}^{(i,j)}\}}}$					
				j = (nd, m)	j = (nd , f)	j = (d , m)	j = (d , f)	j = (inc , m)	j = (inc , f)
(nd , m)	-0.669	0.040	0.844	1	0.748	1	0.748	-0.638	-0.322
(nd , f)	-0.376	0.048	0.929		1	0.748	1	-0.247	-0.544
(d , m)	-0.669	0.040	0.844			1	0.748	-0.638	-0.322
(d , f)	-0.376	0.048	0.929				1	-0.247	-0.544
(inc , m)	-0.825	0.607	3.306					1	0.399
(inc , f)	-0.590	0.347	2.499						1

Note:
nd = mortality of non-disabled, d = mortality of disabled, inc= incidence, m = male, f = female

Appendix 5E Fit of transition rates, age-specific and overall R^2

The R^2 statistic can be used to measure how large proportion of the variation in the data is explained by the model. R^2 can be calculated for each age separately, $R^2_{x,t}$, or for the entire age-profile of a certain type of transition rate, R^2. In most of the transition rates above 80% of the age-specific variation is explained by the model until the age of 85. Up till the age of 60 and after the age of 85 the model fit was somewhat lower. A possible explanation is that the number of transitions (incident cases, deaths) at younger ages is low compared to the size of the exposed populations; therefore there is a larger variation in the transition rates at these ages. After the age 80 the model fit started to decrease. This could be explained by the large number of transitions relative to the small size of exposed populations; therefore the transition rates were less stable at higher ages. Even though the age-specific model fits were quite poor at very high ages, the overall fit of the models can be considered as very good (≈ 70-95%).



Chapter 6

Forecasting lifetime and aggregate long-term care spending: accounting for changing disability patterns

C. de Meijer, I.M. Majer, M. Koopmanschap, P. van Baal

Objective

The impact population aging exerts on future levels of long-term care (LTC) spending is an urgent topic in which few studies have accounted for disability trends. We forecast *individual* lifetime and *population* aggregate annual LTC spending for the Dutch 55+ population to 2030 accounting for changing disability patterns.

Methods

Three levels of (dis)ability were distinguished: none, mild, and severe. Two-part models were used to estimate LTC spending as a function of age, sex, and disability status. A multistate life table model was used to forecast age-specific prevalence of disability and life expectancy (LE) in each disability state. Finally, 2-part model estimates and multistate projections were combined to obtain forecasts of LTC expenditures.

Results

LE is expected to increase, whereas life years in severe disability remain constant, resulting in a relative compression of severe disability. Mild disability life years increase, especially for women. Lifetime homecare spending —mainly determined by mild disability— increases, whereas institutional spending remains fairly constant due to stable LE with severe disability. Lifetime LTC expenditures, largely determined by institutional spending, are thus hardly influenced by increasing LE. Aggregate spending for the 55+ population is expected to rise by 56.0% in the period of 2007–2030.

Conclusions

Longevity gains accompanied by a compression of severe disability will not seriously increase lifetime spending. The growth of the elderly cohort, however, will considerably increase aggregate spending. Stimulating a compression of disability is among the main solutions to alleviate the consequences of longevity gains and population aging to growth of LTC spending.

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6.1. Introduction

The impact of population aging on the level of health care expenditures has become a topic of growing attention over the last decades. Of particular interest is its impact on long-term care (LTC) spending. Although the increase in the acute care expenditure age profile can entirely be explained by age-specific differences in health status (often approximated by time to death), LTC expenditures increase with age even after accounting for variations in health status. Besides, LTC use is more concentrated among the middle aged and elderly. Hence, the most dramatic spending growth due to population aging is expected in the LTC sector[92,190-196].

Population aging, defined as the increasing share of elderly in a population, results from lower birth rates, longevity gains, and baby boomer aging. The increasing proportion of elderly and the growing proportion of the very old in the elderly cohort will swell the need for LTC. Also, longevity gains mean needing LTC for longer periods. Population aging thus impacts the group of individuals requiring LTC, aggregate annual LTC spending, and individual lifetime LTC spending. As disability is the main determinant of LTC use[197-199], the growth of aggregate and lifetime LTC spending is strongly associated with future disability trends[200].

Forecasting individual lifetime and population aggregate LTC spending is challenging, because it requires estimates of disability and mortality. Estimating such trends is complicated: disability is a stock measure governed by inflows (incidence) and outflows (recovery or mortality) that are likely to change over time. Although past disability trends have been investigated abundantly[39,199,201-205], few studies have exploited the trends to forecast future LTC use or spending. We aim to assess the effect of aging (the growing number of elderly and longevity gains) on the future trend of lifetime and aggregate LTC spending for the Dutch 55+ population by explicitly accounting for changing disability patterns. Given further population aging, it is important to estimate future levels of LTC spending to prepare the LTC sector for future needs.

Disability trends and LTC spending forecast

Evidence on disability trends varies across and even within countries.[39,206]. Trends depend on the selected measure of disability [instrumental activities of daily living (iADL), activities of daily living (ADL) or mobility] and the severity of disability. A consistent decrease in disability prevalence has been documented for the United States[39,199,201-205]. Evidence on disability trends in European countries is less conclusive[39,207]. Although Puts et al. report declining ADL and mobility prevalences for the Dutch non-institutionalized elderly from 1987 to 2001[208], Picavet et al. found a declining mobility prevalence for males and a constant ADL disability trend for the period 1990-1998[23]. Finally, a meta-analysis on the disability

trend in the non-institutionalized Dutch population over 1990-2007 reported constant ADL and mobility disabilities[209].

Disability-free life expectancy (DFLE), absolutely and relatively as a proportion of total life expectancy (LE), increased for both males and females in the United States in 1980–1990[18,178]. A similar trend has been observed for males in several European countries; the trend for females was inconclusive[10]. In The Netherlands a decrease of LE with severe disability and an increase of LE with mild disability have been observed[22]. A US study extrapolated the annual disability decline of 0.8% observed in 1910–1999 and estimated a DFLE at age 65 to grow from 79.9% in 2015 to 85.2% in 2080[178].

Future disability trends are shown to largely impact LTC spending forecasts. LTC spending in OECD countries from 2005 to 2050 will increase from 1.1% to 2.8% of GDP under stable age-specific severe disability prevalence but only to 1.9% with declining severe disability rates[39]. Similar results were found in other studies[199,210]. Moreover, forecasts using an extrapolation of the disability decline best approached the actual amount spent.[201]. If and to what extent disability will continue declining, given the less healthy lifestyles and higher disability rates of the middle-aged population is, however, debatable[211-213].

Our study extends the literature in 3 respects. First, we estimate future disability prevalence and DFLE for the Dutch 55+ population. As both trends have been shown to depend on the severity of disability, we simultaneously forecast mild and severe disability. Second, we use the forecasts to estimate lifetime and aggregate public LTC spending. Previous studies have only applied LTC spending by age and sex to the future age-sex decomposition of the population. Third, our spending forecasts distinguish between homecare and institutional LTC.

Public LTC spending in The Netherlands is high because it is a universal insurance, coverage of services is comprehensive, and copayments are limited. Eligibility to public LTC is regulated by an assessment agency and is primarily subject to needs. In this study, services include publicly financed institutional LTC and the homecare services domestic care, personal care and nursing care, but not homecare financed by a personal care budget (5%-10% of publicly financed homecare)[214]. Institutional LTC accounts for approximately 70% of LTC spending and includes residential and nursing home admissions. Residential homes provide living assistance; nursing homes also provide personal and nursing care. Overall, our analysis includes the bulk of public LTC expenditure.

6.2 Methods

To forecast future LTC spending by accounting for changing disability patterns, we first estimate the relation between disability and LTC spending to obtain average LTC spending patterns by age, sex, and disability status (nondisabled, mild disabled, severe disabled), and then forecast disability prevalences by applying a previously published multistate model to our dataset[215]. Finally, average spending patterns are combined with forecasts of disability status by age and sex to forecast LTC spending.

Data to model individual LTC function

We used 3 datasets to model yearly LTC expenditures: the Health Survey 2004/5, the Registry of Public LTC Use 2004/5, and the Elderly in Institutions Survey 2004 (EIS). We used the Health Survey, a cross-sectional survey among a representative sample (n ≈ 10,000) of the Dutch noninstitutionalized population, to obtain information on disability. Health Survey respondents were selected by a 2-stage sampling design: first, municipalities proportional to their size were selected; second, individuals within the selected municipalities were randomly sampled. Our study population comprised individuals aged 55–97. Disability was measured by the performance on the following ADL and mobility items: (un)dress, wash face and hands, wash oneself completely, transfer from chair, transfer from bed, move outdoors, climb stairs, and enter/leave the house. Respondents could state whether they could perform the activity without difficulty, with difficulty, or not able to perform the activity. Most previous studies use the number of (i)ADL/mobility problems as cut-offs for the disability level[92,191,197,199,200]. Some Dutch studies, however, used the level of difficulty with activities to distinguish between mild and severe disability[10,209,216]. In our analyses, we choose the latter disability measure as it outperformed the former in explaining LTC use substantially. This led to the following classification: nondisabled is defined as the ability to perform all items without difficulty, mild disability as the ability to perform all items independently but at least 1 item with difficulty, severe disability as the inability to perform at least 1 item. Note that nondisabled individuals might still experience difficulties with iADL's as our data did not contain iADL information. Table 6.1 shows the prevalence of mild and severe disability for 2 different disability measures. The threshold to be severely disabled is substantially higher when measured by the disability measure selected for our analyses. As severe disability is a prerequisite to obtain access to publicly financed institutional LTC and institutional LTC accounts for approximately 70% of LTC spending, this might explain the better performance of our disability measure in explaining LTC spending.

Table 6.1 Description of LTC estimation sample (weighted; standard deviations after ±-sign)

	Total sample (n=6,512)	Institutional users sample (n=1,049)	Homecare users sample (n=679)
LTC consumption			
LTC use (%)	14.7	100.0	100.0
Mean LTC costs (€)	1,647 ± 8,167	32,617 ± 26,961	5,124 ± 8,257
Institutional LTC use (%)	3.5	100.0	3.2
Mean institutional LTC costs (€)	1,125 ± 7,778	32,081 ± 27,092	619 ± 5310
Homecare use (%)	11.6	10.6	100.0
Mean homecare costs	522 ± 2,569	536 ± 2,410	4504 ± 6249
Demographics			
Age	67.6 ± 9.4	83.1 ± 7.6	77.0 ± 8.7
Male (%)	45.7	25.1	25.3
Disability (%)			
Non-disabled	66.8	6.3	20.9
Mildly disabled	30.7	23.3	78.6
Severely disabled	2.5	70.4	0.5
Alternative disability measure (%)*			
Non-disabled	67.0	6.1	21.3
Mildly disabled (1-3 ADL problems)	19.0	19.4	32.2
Severely disabled (4-7 ADL problems)	14.1	74.5	46.5

*The alternative disability measure is included only to highlight that the prevalenceof mild and severe disability strongly depends on the chosen definition of disability.The alternative disability measure is not used in any of the forecasts; LTC indicates long-term care.

We obtained information on LTC spending by linking the Health Survey to the Registry of Public LTC Use (CAK). Statistics Netherlands used a probabilistic linking process to uniquely identify respondents of several micro datasets, including the Health Survey and the CAK but not the EIS survey. Linking keys to uniquely identify individuals were date of birth, sex, and zip code. Nearly 100% of individuals could be uniquely identified. Using these individual identifiers, we linked our Health Survey sample to the CAK to obtain information on individual LTC use. Total LTC spending is composed of institutional LTC and homecare expenditures. Because the Health Survey excludes the institutionalized population, we added a random sample (n = 1000) of institutional LTC users from CAK. As we did not have disability information on this subsample, we used the EIS[197], a national representative sample of the 55+ institutionalized population, to assign disability status to this subsample. Disability items of the EIS are identical to the Health Survey. The distribution of disability by sex and type of institution in our institutionalized sample is set equal to

that of the EIS. Because of uniform tariffs of LTC institutions and the fact that individuals can only obtain access to publicly financed institutional LTC if they are sufficiently disabled, the variances of LTC spending and disability levels of institutionalized individuals are very low. Hence, the assignment of disability to our institutionalized sample closely reflects the true disability level of individuals. 4.2%, 9.3%, and 86.5% of the permanently institutionalized were nondisabled, mildly, and severely disabled, respectively.

Item nonresponse excluded four individuals, leaving a sample of 6512 individuals. Post-stratification weights were computed to correct the joint distribution of weighting variables in our sample to those of the Dutch 55+ population as registered at Statistics Netherlands[217]. Weighting variables were age × sex × institutionalized.

Modelling individual LTC expenditure

We employed a two-part model – common to health care expenditures analysis[218] – to estimate LTC spending by age, sex, and disability status. A 2-part model accounts for the high proportion of nonusers by separately analyzing use (part I) and the level of expenditures conditional on use (part II). Part I contains a probit model that analyses the probability of using LTC. We followed the procedure proposed by Manning and Mullahy[219] to select the most appropriate model for part II. A generalized linear model with power link and gamma family best suited our data. The Box Cox transformation parameter was used as the power link. Expected expenditures for individual i are obtained by multiplying parts I and II:

$$E\left(LTCE_{ij} \mid X_i\right) = \Phi\left(\beta_{1j} X_i\right) \times \sqrt[\lambda]{\beta_{2j} X_i + 1} \tag{1}$$

where Φ() is the probability given by part I and $\sqrt[\lambda]{(.)}$ is the conditional level of expenditures given by part II for individual *i* and type of LTC j, with *j*=1 (total LTC), *j*=2 (institutional LTC), *j*=3 (homecare). β_1 and β_2 are vectors of parameters to be estimated by part I and part II, respectively, and X_i is a vector of covariates, that is, orthogonal age variables, sex, disability, and interactions between disability and age.

Forecasting disability prevalence and (DF)LE

To forecast disability prevalence and (DF)LE we used a multistate life table model that distinguished 3 states: nondisabled, disabled, and dead. As this model could only handle a dichotomous disability variable, the model is used twice: first to forecast the sum of mild and severe disability, then to forecast severe disability. Finally, both estimates are combined to obtain forecasts of the prevalence of nondisabled, mildly disabled, and severely disabled.

Connecting the health states, transition probabilities were estimated as a function of age and calendar year. We forecast transition probabilities up to 2030 based on trends over the period of 1989–2007. Forecasts of all the transition rates used the Lee-Carter method[31]. Having obtained the future age-specific transition probabilities, LE with and without disability could be estimated. The reader is referred to Majer et al[215] for an extensive description of the model used to forecast (DF)LE.

We used different sources to estimate the parameters of the model. The Human Mortality Database gave us sex-specific and age-specific mortality rates from 1989 to 2007. The Health Surveys 1989–2007 were used to estimate disability prevalence in the noninstitutionalized population. National statistics on LTC institution residents by age and sex were pooled to the Health Surveys to obtain complete age-specific and sex-specific disability prevalence for the 55+ population. Finally, the dataset was linked to the Death Registry to obtain mortality rates by disability status[220].

Forecasting LTC expenditure

Forecasting *lifetime* expenditures required combining the probabilities of surviving to future ages, being disabled, and expected LTC spending. Lifetime expenditures were computed using a period rather than cohort measure. Expected lifetime spending for an individual (E(LT_LTCE)) could be expressed as the sum of the product of (i) the probability of being alive (S_i) at a certain age given sex and disability status, and (ii) the expected level of LTC spending at a certain age given sex, disability, and survival status summing over all the ages between 55 years and death:

$$E\left(LT\_LTCE_{ij} \mid S_i, X_i\right) = \sum_{age \geq 55} \Pr\left(S_i = 1 \mid X_i\right) \times E\left(LTCE_{ij} \mid X_i\right) \quad (2)$$

where the first term on the right hand side is the survival probability as a function of age, sex and disability status as forecast with the model.

Aggregated expenditures were estimated to 2030. Aggregate LTC expenditure was defined as the sum of individual expenditures given the number of individuals forecasted by Statistics Netherlands combined with the disability prevalence forecasts of our model.

6.3 Results

Descriptives

Table 6.1 provides summary statistics for the study sample and for the subsamples of institutional and homecare users: 14.7% used LTC (average cost unconditional on use =

€1647), 11.6% used homecare (€522), and 3.5% used institutional care (€1125). Homecare users – and to a greater extent institutional users – were older, more often female and more often disabled. The prevalence of mild disability was lower for institutional residents than homecare users because the institutionalized are more often severely disabled.

LTC spending by age, sex and disability

Figure 6.1 displays homecare and institutional LTC use and spending by age, sex, and disability status. The first, second, and third rows present the predicted probabilities of use, conditional expenditures, and expected expenditures, respectively. The probability of using LTC and the level of (un)conditional spending increase with the severity of disability.

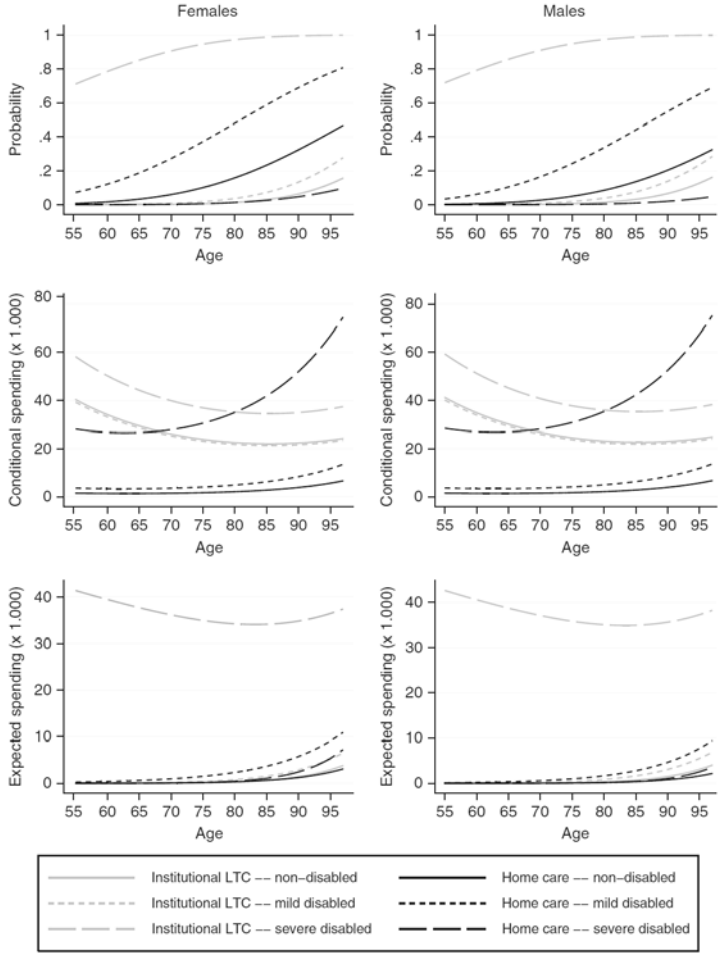


Figure 6.1 Long-term care (LTC) use and (un)conditional spending by age, sex, and disability status

Homecare use and spending, however, is highest for the mildly disabled elderly; the severely disabled elderly are more likely to be institutionalized. The probability of using homecare or institutional LTC increases with age. Conditional homecare spending increases and institutional LTC spending decreases with age. The latter finding is caused by the fact that the younger institutional LTC users are rarely admitted to residential homes (the least expensive LTC institution) but more often to somatic nursing homes. Females are more likely to use both types of LTC and spend on average more than males. This finding could either be due to their higher morbidity level or lower availability of adequate informal care sources. No substantial differences in conditional expenditures were found between males and females. The difference in unconditional expenditures is thus driven mainly by the probability of using LTC.

Disability prevalence trend

Figure 6.2 presents the trend in mild and severe disability prevalence as estimated by our model. The proportion of nondisabled elderly decreased for females, but remained fairly constant for males. The higher disability rates among females are entirely driven by increases in mild disability. Mild disability rates also increased for males, but less seriously. The prevalence of severe disability decreased for both sexes and is expected to further decrease the coming years.

(DF)LE trend

The trend in LE at the age 55 stratified by disability status is illustrated in Figure 6.3. LE will continue to increase, but more for males than for females. LE for males is expected to increase by 3.4 years, from 24.9 (95% confidence interval, 24.5–25.3) in 2008 to 28.3 (26.4–30.0) in 2030; for females it is expected to increase by 2.0 years, from 28.6 (28.3–28.9) to 30.6 (29.3–31.9). For both sexes, the number of severely disabled life years will remain constant, but the number of mildly disabled life years will increase, especially for females. For females mildly disabled life years will increase from 8.9 (7.6–10.2) in 2008 to 11.5 (4.1–18.5) in 2030. For females, the expected increase in life years with mild disability dominates the total increase in LE, resulting in a slight decrease of absolute LE years without disability. A relative compression of severe disability is estimated for both sexes. In 2008, 10.0% (12.5%) of the remaining LE for males (females) was spent with severe disability; this is expected to decrease to 7.0% (11.1%) by 2030. In the period 2008–2030, the proportion of life years lived with mild disability is expected to remain constant for males (roughly 20.0%), and is expected to increase from 31.1% to 37.5% for females. Overall, the proportion of DFLE will remain constant for males and decrease for females.

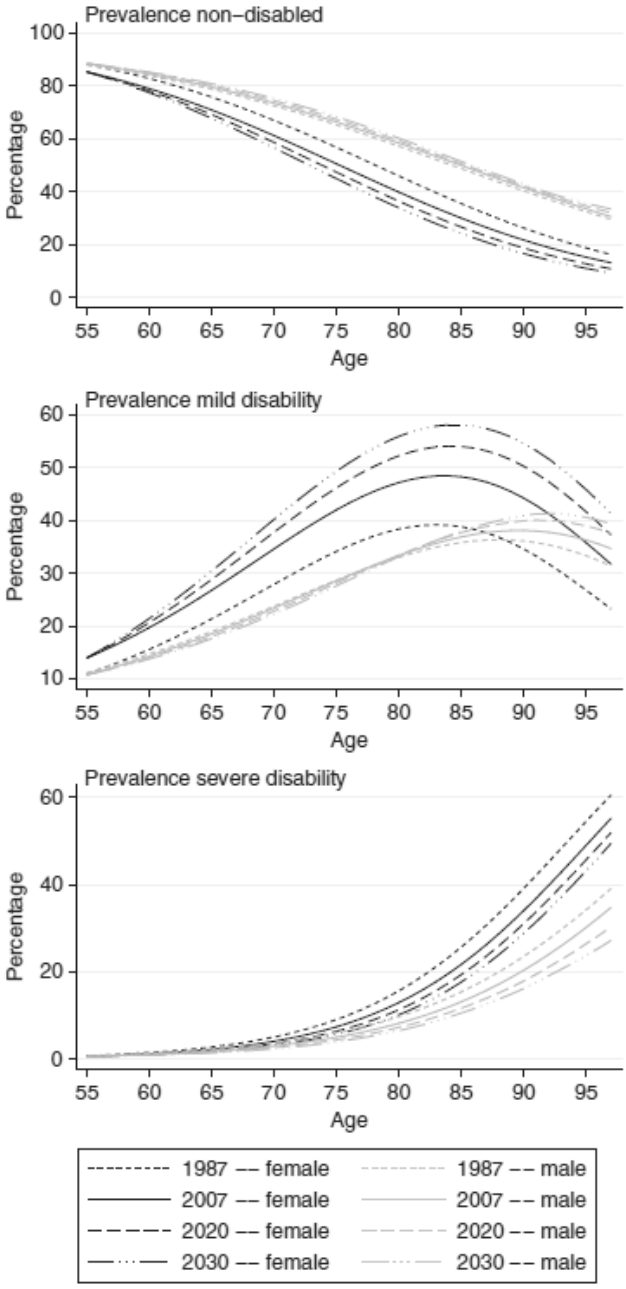


Figure 6.2 Trends in the disability status stratified by age and sex

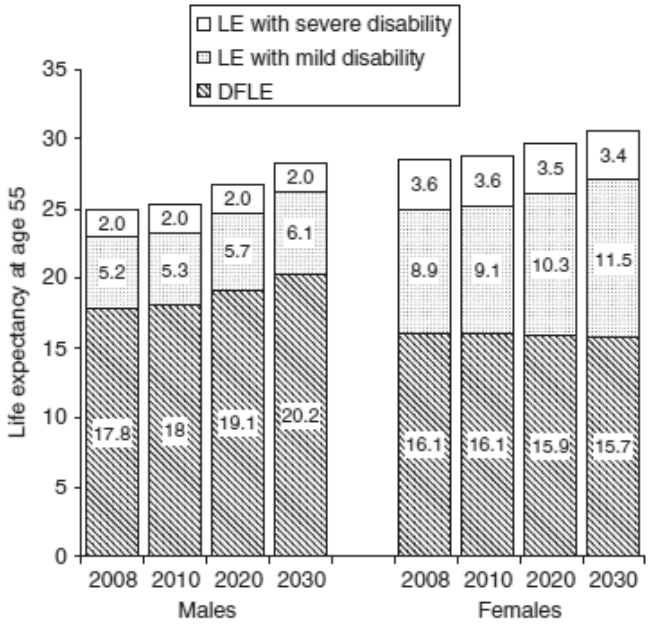


Figure 6.3 Trend in life expectancy at age 55 stratified by life years with no, mild, and severe disability
Notes: DFLE indicates disability-free life expectancy; LE, life expectancy

Lifetime spending forecasts

Figure 6.4 presents lifetime spending per capita at age 55 stratified by LTC service (year 2004 values). Females spend about twice as much as males on both LTC services. In 2008, lifetime spending on LTC for females (males) is approximately €128,000 (€71,000). Homecare spending is expected to increase by €2503 for males and €5850 for females. Most of the growth is caused by the expansion of mild disability as most spending on homecare is incurred during mildly disabled life years. The bulk of institutional LTC spending is incurred during severely disabled life years. The pattern of lifetime spending on institutional LTC, therefore, coincides with the trend in severely disabled life years: lifetime institutional LTC spending slightly increases for males and decreases for females. Overall, total spending on LTC is expected to increase by €5435 (7.7%) for males and €3044 (2.4%) for females.

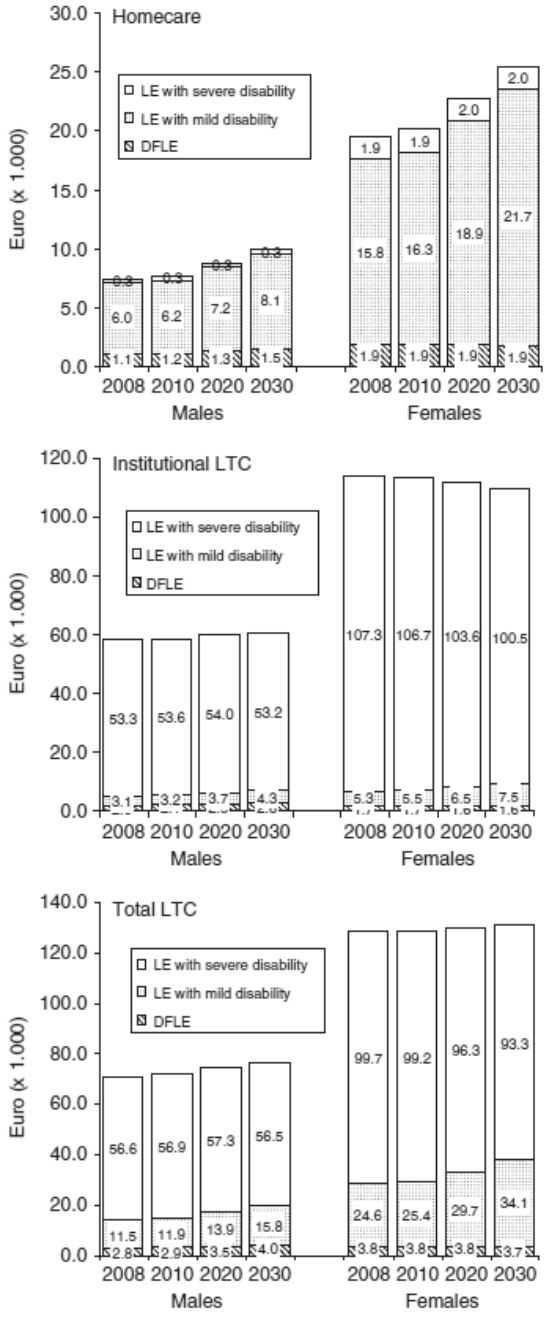


Figure 6.4 Trend in lifetime spending at age 55 accounting for the disability trend
Notes: DFLE indicates disability-free life expectancy; LE, life expectancy; LTC, long-term care.

Aggregated spending forecasts

The effect of the increasing number of elderly and the oldest of the old becomes apparent in the aggregated spending forecasts. Accounting for the trend in disability prevalence, aggregated LTC spending (year 2004 values) for the population aged 55–97 is expected to increase from €10.7 to €16.8 billion between 2007 and 2030, a 56.0% growth. This amounts to an annual growth rate of 1.95%. Increased use of institutional LTC is responsible for about two third of the total aggregate growth in LTC spending.

6.4 Discussion

We have forecasted lifetime and aggregated LTC spending among the Dutch 55+ population out to 2030. Our approach goes beyond earlier efforts in that (i) our forecasts explicitly account for changes in disability patterns and longevity with and without mild and severe disability, and (ii) we distinguish forecasts of homecare and institutional LTC expenditures.

There are several notable findings. First, a prevalence shift has been observed from severe disability to mild disability resulting in improved disability profiles for males. For females, the decrease in severe disability prevalence is offset by a larger increase in mild disability prevalence resulting in a lower proportion of nondisabled females. Concerning DFLE, our forecasts indicate a relative compression of severe disability as the years with disability remain constant but are postponed to higher ages, whereas the proportion of life years with mild disability remains constant for males and increases for females. Second, lifetime spending for females is approximately double that of males primarily because females have a higher LE (with disability) and secondarily because they more often rely on formal care in the absence of informal care sources. Third, we found a tremendous effect of the disability trend on lifetime and aggregate LTC spending. Future longevity gains coinciding with a compression of severe disability are not very costly. Homecare is mainly used during life years with mild disability, whereas institutional LTC is strongly associated with severe disability. As such, a compression of severe and expansion of mild disability will increase lifetime spending on the least intensive LTC service: homecare. Finally, the substantial effect of population aging on aggregate LTC spending reflects the substantial growth of the elderly cohort. Given such dramatic growth in future public LTC spending, changes in LTC financing might be necessary to keep LTC provision efficient and equitable.

Our results do not necessarily contradict evidence from other countries that found a decline in the prevalence of mild disability as we used different cut-offs for mild and severe disability[199,203,205,207,221]. The trend in mild disability is often operationalized by the prevalence of iADL problems, whereas our data lacked iADL information.

Causes of disability declines are not fully understood. We expect the observed decline in disability prevalence and DFLE to be partly explained by improvements in health status. Simultaneous to the disability decline, however, the prevalence of chronic conditions has increased[206]. Moreover, the relationship between disability and health status has been proven to depend on behavioral and environmental factors, for example, healthier lifestyles, less physically demanding work, technological improvements that allow greater independence given a certain level of body functioning (eg, development of appliances)[206,222,223]. Further research should disentangle the causes of the disability decline to improve prognoses and possibilities of a continuation of it, and in turn stem LTC spending.

Most studies projecting future LTC spending based on disability trends assume that the trends are exogenous. The observed declines in disability prevalence and DFLE, however, are likely to be partly caused by the growth in acute care spending. Although technological advances in acute care that solely mitigate mortality might increase disability rates, technological advances are often able to mitigate disability. The decrease in LTC spending, therefore, probably occurs at the cost of the acute care sector. This is confirmed by the finding that in most developed countries, disability improvements have been accompanied by an increase in chronic diseases[206,221].

Our study has a number of limitations. First, our forecasts do not account for future changes in informal care availability, which have been found to codetermine formal LTC spending[216,224]. The higher probability of females relying on formal LTC might be partly explained by the fact that they more often reside alone; even if co-residing, their partners are less likely to provide sufficient informal care. Given that our forecasts assume stable informal care sources, they might underestimate the future level of LTC spending as co-residence rates are expected to decrease, and increases in the female labor force participation and the retirement age likely decreases future informal care sources. Second, our model assumes stability of the LTC expenditure function and constant prices but LTC service prices will most likely increase due to a number of reasons such as labor shortages and quality improvements in the LTC sector. A final limitation is the likely endogeneity of disability. In addition to the impact of disability on LTC use, use of LTC could in turn influence disability rates, which might bias our results.

Concluding, we have shown the importance of accounting for changing disability trends when modeling future LTC expenditures. Although longevity gains accompanied by a compression of severe disability do not substantially increase lifetime spending, aggregate spending will increase considerably due to the sheer number of elderly. Stimulating a compression of disability might alleviate the consequences of aging on LTC spending growth.

Chapter 7

Time trends and forecasts of body mass index from repeated cross sectional data. A different approach

I.M. Majer, J.P. Mackenbach, P.H.M. van Baal

In this paper we report a case study on a technical generalization of the Lee-Carter model, originally developed to project mortality, in order to forecast body mass index (BMI, kg/m2). We present the method on an annually repeated cross sectional data set, the Dutch Health Survey (POLS), covering years between 1981 and 2008. We applied Generalized Additive Models for Location, Scale and Shape semi-parametric regression models to estimate the probability distribution of BMI for each combination of age, gender and year assuming that BMI follows a Box-Cox power exponential distribution. We modelled and extrapolated the distribution parameters as a function of age and calendar time using the Lee-Carter model. The projected parameters defined future BMI distributions from which we derived the prevalence of normal weight, overweight and obesity. Our analysis showed that important changes occurred not only in the location and scale of the BMI distribution but also in the shape of it. The BMI distribution became flatter and more shifted to the right. Assuming that past trends in the distribution of BMI will continue in the future we predicted a stable or slow increase in the prevalence of overweight until 2020 among men and women. We conclude that our adaptation of the Lee-Carter model provides an insightful and flexible way of forecasting body mass index, and that ignoring changes in the shape of the BMI distribution would likely result in biased forecasts.

Submitted

7.1 Introduction

The continuous increase in the prevalence of obesity and overweight is a growing health problem in developed countries and particularly in the wealthiest ones[225]. In the United States (US) the prevalence of obesity (Body Mass Index, kg/m2, ≥30) and overweight (25≤BMI<30) have increased noticeably among both children and adults. National health survey data have shown that the age-adjusted prevalence of obesity more than doubled between the early '70s and the middle '00s, from 14.1% to 30.5%, in the US adult population[226-228]. By 2008, the prevalence of obesity had risen to around 34%, while another 34% of the adult populations were overweight[229]. Recently, an updated analysis showed that the age-adjusted prevalence of obesity even further increased to 35.7% by 2010[230]. In the Netherlands, the share of the Dutch population being at least overweight has increased from a third to a half in the past twenty years. The share of obese people has doubled, to about one in ten among men and one in eight among women[160]. It is expected that, by 2020, the number of obese persons in the Netherlands will increase by 50 percent[231].

The effect of overweight and obesity on health has become a major concern of public policy in many developed countries[232]. Therefore forecasting future levels of obesity is essential to informing policy makers. A few studies have attempted to project the prevalence of overweight and obesity. Based on four waves of longitudinal US data from the National Health and Nutrition Evaluation Survey (NHANES) spanning from 1971-2004, Wang et al. estimated annual proportional changes of the mean BMI by age, sex and race[233]. To project 10-year changes in the BMI until 2010 the authors applied the estimated annual proportional changes to the individuals in the latest NHANES survey, and subsequently projected their BMI 10 years forward. Using these projections, the estimated future prevalence of overweight and obesity in the population was calculated. Ruhm estimated trends in BMI and forecasted the prevalence of overweight and obesity as well[234]. The author calculated annual growth rates based on data from NHES (1960-1962), NHANES II (1976-1980) and NHANES 1999-2004 using quantile regressions for each of the 1st through 99th BMI percentiles. Compared to using traditional regression methods, the advantage of using quantile regressions is that it allows BMI growth trends to vary across the distribution of BMI. Projected BMI at the specified percentile was obtained by setting the time trend to its value in the forecasted year of interest (e.g. 2020) with other explanatory variables evaluated at the latest NHANES survey averages. Basu forecasted the distribution of BMI using longitudinal US data by estimating the probabilities of moving between BMI categories[235]. Letting the transition probabilities be constant and continue in the future, cohort-wise estimates of future BMI categories were estimated. Finally, Mills forecasted BMI categories for England[236]. Contrary to previous studies, in which the prevalence estimates were derived from trends in the continuous BMI variable, Mills modelled the proportions directly using compositional data analysis methods. Such a method requires an additive log-ratio transformation on the

original data, so that the transformed data has a multivariate normal distribution. Standard time series models were fitted to the transformed data, and using the fitted models, forecasts were made until 2010.

Forecasting BMI can be challenging due in part to the lack of good quality and long time series BMI data and to the multinomial nature of the overweight status variable, i.e. the forecasted proportions must remain positive fractions and sum up to one. Compared to forecasting binomial data, such setting is more complicated to handle because of the additional dimensions in the model. One alternative to modelling the proportion of overweight and obesity directly is to model the entire BMI distribution. However, the challenge then becomes how to capture the patterns in the changes of the BMI distribution over time. For instance, it has recently been shown that as BMI increased more at high than low values, its distribution shifted faster to the right, in turn making it less right-skewed[237,238]. Still, how to handle such shifts in a model may not be straightforward. As a result, there is no standard method for forecasting BMI levels.

With respect to the data, the majority of forecasting studies have relied on longitudinal cohorts with a limited number of measurement time points, which suffer from attrition. This is in contrast with health surveys that, in many countries, are conducted on a yearly basis with independent sampling (i.e. repeated cross sectional data)[239,240]. This latter study type has the advantages of being more common and as such when combined can provide multiple sets of observations of large sample over long periods of time, similar to mortality statistics.

In this paper we report a case study on a technical generalization of the Lee-Carter model, originally developed to project mortality, in order to forecast BMI[31]. We present the method on an annually repeated cross sectional data set, the Dutch Health Survey (POLS), covering years between 1981 and 2008. Applying the Lee-Carter model to BMI implies that the prevalence of a particular BMI status is a function of two effects: age and calendar year. There are two main features of our approach. First, instead of modelling the prevalence of overweight and/or obesity status, this approach models the entire BMI distribution from which the distribution of overweight status is derived. It is convenient in that it jointly estimates the prevalence of each overweight category. Also, the usual categorization of the BMI distribution may be crude in light of the discussions about the healthiest BMI value or the appropriate cut-off points defining the healthiest BMI range[241]. In this respect, modelling the whole BMI distribution is a flexible approach because other cut-off points could be defined easily without requiring a fundamental change in the modelling strategy. Second, we fitted four-parameter Box-Cox power exponential distribution on BMI data that allowed explicit modelling of the changes in the shape of the BMI distribution. In previous studies BMI was

typically assumed to follow lognormal distribution[242,243], however the disadvantage of the lognormal distribution is that the skewness and the kurtosis are merely a characteristic of the distribution influenced by the mean and the variance.

7.2 Data

BMI was calculated using weight and height data from the Dutch Health Survey ('POLS Gezond') collected among the community-dwelling population of the Netherlands[73]. The POLS is a nationally representative, ongoing annual cross-sectional survey aiming to provide information on a broad range of topics concerning the living situation of the Dutch non-institutionalized population. Each year of the POLS data is an independent sample of records from a centralized municipal registry. In the POLS, self-reported health data are collected via face-to-face interviews and written questionnaires. Specifically, interviewers visit participants at their home, obtain informed consent, and leave a written (drop-off) questionnaire. The annual participation is approximately 9,000 individuals, with response rates of around 60% for the questionnaire.

We used 28 years of POLS surveys conducted between 1981 until 2008 (T=28), combining data from 248,011 subjects with known age, sex and calculated BMI score. We used data only from adults resulting in 64,671 exclusions, i.e. 26.1% of the sample. In addition we excluded 324 observations (0.2% of the adult sample) with BMI values outside of the 15–50 range because these likely represent measurement or documentation errors. Thus, we included a final sample size of 183,016 individuals in the analysis. We grouped individuals into 10-year age groups, $x \in \{20–29, 30–39, ..., 70+\}$. Their BMI scores were categorized according to WHO guidelines[244] into the combined underweight (BMI ≤ 18.5) and normal weight (18.5 < BMI ≤ 25) category (below we refer to this merged category as normal weight), overweight (25 < BMI ≤ 30) and obese categories (30 < BMI). Table 7.1 presents information on the study population by year and sex.

Table 7.1 Summary data of the study population

Year	POLS Gezond: achieved sample size	Correspond. response %	Study population: Aged: 20+, 15≤ BMI ≤ 50	OW (%)	OB (%)	Average BMI in OW	Average BMI in OB
1981	9,809	68.0	6,775	30%	6%	26.8	33.6
1982	9,319	66.6	6,553	29%	6%	26.8	33.4
1983	8,712	64.5	6,407	30%	6%	26.8	33.4
1984	9,065	63.4	6,628	29%	6%	26.9	32.9
1985	8,633	63.4	6,319	29%	5%	26.9	33.0
1986	8,716	63.8	6,453	30%	6%	26.9	33.0
1987	7,932	59.0	5,985	31%	6%	26.9	32.6
1988	7,579	58.3	5,711	30%	5%	26.9	33.0
1989	8,007	58.5	5,999	31%	7%	26.9	32.8
1990	7,107	56.3	5,390	30%	7%	26.9	32.6
1991	6,682	56.7	5,157	32%	7%	26.9	32.6
1992	8,482	56.7	6,528	31%	7%	27.0	33.1
1993	8,107	55.0	6,288	31%	7%	27.0	33.1
1994	8,538	56.1	6,557	33%	8%	26.9	32.7
1995	8,980	58.6	6,865	32%	8%	27.0	33.0
1996	8,396	56.6	6,522	32%	8%	26.9	33.0
1997	10,664	59.4	7,785	33%	9%	27.0	32.8
1998	9,075	58.1	6,500	32%	9%	27.0	32.8
1999	9,600	55.9	6,897	35%	9%	27.0	33.2
2000	9,639	55.0	6,927	36%	10%	27.0	33.0
2001	9,349	61.8	6,720	36%	11%	27.0	33.1
2002	9,382	61.2	6,771	36%	11%	27.1	33.1
2003	9,439	58.3	6,762	36%	12%	27.1	32.9
2004	10,657	61.3	7,681	37%	12%	27.1	33.1
2005	9,853	65.0	7,272	35%	12%	27.1	33.1
2006	9,094	66.4	6,836	36%	12%	27.1	33.1
2007	8,219	64.0	6,127	34%	12%	27.1	33.2
2008	8,976	64,4	6,601	37%	12%	27.1	33.3
Sum	248,011	-	183,016				

Notes: The corresponding response percentage – for example – in 2008 indicates that 64.4% of the original sample size was achieved, and that resulted in a sample of 8,976 individuals with known age, sex and BMI values. OW = overweight. OB = obese.

7.3 Statistical methods

The GAMLSS statistical model

We used Generalized Additive Models for Location, Scale and Shape (GAMLSS) semi-parametric regression[245] to model the probability distribution of BMI. The GAMLSS offers a flexible approach to model not only the location but also other parameters (scale and shape) of the distribution of the dependent BMI variable as linear parametric and / or additive non-parametric functions of explanatory variables. The GAMLSS model assumes i=1,…,N independent observations with probability density function $f(bmi_i \mid \theta^i)$ where $\theta^i = (\theta_{i1}, \theta_{i2}, ..., \theta_{ip})$ is a vector of parameters. In our situation p=4 distribution parameters were required that may be interpreted relating to the location, the scale, the skewness and the kurtosis, respectively.

Fitting distributions on BMI

Evidence from other countries suggests that changes not only in the mean but also in the skewness of the BMI distribution took place [242,243]. Consequently it seems wise to choose a distribution with more than two parameters that is able to explicitly capture potential shifts in the shape of the BMI distribution. For this purpose we selected the Box-Cox power exponential (BCPE) distribution that offers to model the BMI distribution with four parameters[246]. The BCPE is defined through the transformed variable Z, which is given by

$$Z = \begin{cases} \dfrac{1}{\sigma\nu}\left[\left(\dfrac{Y}{\mu}\right)^{\nu} - 1\right], & if\ \nu \neq 0 \\ \dfrac{1}{\sigma}\log\left(\dfrac{Y}{\mu}\right), & if\ \nu = 0 \end{cases} \qquad (1)$$

for 0<Y<∞, where μ>0 and σ>0. The random variable Z is assumed to follow a standard power exponential distribution with continuous power parameter τ. The exact cdf and pdf of the BCPE distribution is given in Appendix 7A.

The parameters of the BCPE distribution, μ, σ, υ, τ, were estimated for each age (x) and year (t) combination separately, thus we fitted 6 age × 28 year = 168 models for men and women (g) each. We denote the estimated parameters by $\hat{\theta}_k(x,g,t)$ where k=1 may be interpreted relating to the location (median, μ), k=2 to the scale (approximate coefficient of variation, σ), k=3 to the skewness (transformation to symmetry, υ), and k=4 to the kurtosis (power exponential parameter, τ), however the parameters do not directly indicate variation, skewness and kurtosis, respectively. Given the estimated distribution parameters, we

calculated the expected prevalence of overweight status (normal weight, overweight and obese) for each combination of age category and year by partitioning the probability distributions according to the WHO cut-off points described above. The advantage of such partitioning is that the estimated prevalence of overweight status always sums up to one, i.e. $\hat{\phi}_{NW}(x,t) + \hat{\phi}_{OW}(x,t) + \hat{\phi}_{OB}(x,t)=1$, where the estimated $\hat{\phi}(x,t)$ is the expected prevalence of normal weight (NW), overweight (OW) and obesity (OB) at age x and in year t, respectively.

Forecasting distribution parameters and overweight status

To forecast the prevalence of overweight status we adapted a mortality projection model proposed by Lee and Carter[31]. The original model was used to project age-specific mortality rates and corresponding life expectancy based on historical mortality data. In short the bilinear Lee-Carter model describes the log of the mortality rates as the sum of an age-specific parameter and an interaction between another age-specific and a time-specific parameter. The forecasting strategy involves three steps: during the first step the parameters of the model are estimated, during the second step the time parameter is projected into the future using a standard time series analysis technique, and during the third step the projected time parameter is inserted back to the original model holding the age-specific parameters constant to obtain future mortality rates. The Lee-Carter model is an elegant approach to model and forecast mortality, and has proved to be a very powerful and by now a widely adopted model. We refer the interested reader to two extensive reviews of the original and further developed methods[34,35].

We have adopted the strategy of Lee and Carter but instead of modelling the logarithm of mortality rates we fitted the bilinear model on the estimated BMI distribution parameters that had been obtained by GAMLSS as described above. The model for the k^{th} distribution parameter $\hat{\theta}_k(x,g,t)$, referring to μ, σ, υ, τ, respectively, evaluated at age x and year t took the following form:

$$\hat{\theta}_k(x,g,t) = \alpha_k(x,g) + \beta_k(x,g)\kappa_k(t) + \varepsilon_k(x,g,t) \quad (2)$$

In the model $\alpha_k(x,g)$, $\beta_k(x,g)$ are gender-specific, g ∈ {*male, female*}, and $\kappa_k(t)$ unisex equation parameters[2] that have to be estimated with subject to arbitrarily chosen identifiability constraints $\sum_t \hat{\kappa}_k(t)=0$ and $\sum_x \hat{\beta}_k(x,g)=1$ for each g. It can be shown that unless we impose constraints on the parameter estimates, the equation in (2) could have infinity many solutions. Therefore, in order to estimate the equation parameters we assume that $\varepsilon_k(x,g,t)$ is

2 We refer to equation parameters in (2) and distribution parameters in (1).

normally distributed. With this assumption the maximum likelihood estimator of an equation parameter is equivalent to the estimator based on the singular value decomposition (SVD) of the matrix with elements $\hat{\theta}_k(x,g,t)-\hat{\alpha}_k(x,g)$ when only the leading singular value, $\sigma_1^{(\hat{\theta}_k)}$, the first column with a dimension of [x×1], $\mathbf{u}_1^{(\hat{\theta}_k(g))}$, and the first row with a dimension of [1×T], $\mathbf{v}_1^{(\hat{\theta}_k)}$, of the SVD are used. In particular, the equation parameter estimates for the k^{th} distribution parameter $\hat{\theta}_k(x,g,t)$ are given by:

$$\hat{\alpha}_k(x,g)=\frac{\sum_t \hat{\theta}_k(x,g,t)}{T},\qquad \hat{\beta}_k(x,g)=\frac{u_1^{\left(\hat{\theta}_k(g)\right)}(x)}{\sum_x u_1^{\left(\hat{\theta}_k(g)\right)}(x)},\qquad \hat{\kappa}_k(t)=\sigma_1^{(\hat{\theta}_k)}v_1^{(\hat{\theta}_k)}(t)\sum_x u_1^{\left(\hat{\theta}_k(g)\right)}(x)\,. \quad (3)$$

The way in which we imposed the constraints on the parameters was for ease of interpretation. Hence for the k^{th} parameter the fitted value $\hat{\alpha}(x,g)$ simply denotes the empirical time-average of $\hat{\theta}_k(x,g,t)$ over T years among people at age x of gender g, whereas $\hat{\beta}_k(x,g)$ indicates how large the effect of age is on $\hat{\theta}_k(x,g,t)$ for a unit change in the latent time index $\hat{\kappa}(t)$ among people in year t.

Take for example males (m) and the first parameter (k=1), the median. Then the value $\hat{\alpha}_1(x,g=m)$ equals the empirical average of the medians estimated for each BMI distribution over the T years among men at age x. These age-specific averages delineate an age-profile for the average medians of the BMI distribution. Also, straightforward interpretation can be given for $\hat{\beta}_1(x,g=m)$ and $\hat{\kappa}_1(t)$. $\hat{\kappa}_1(t)$ indicates how the average median of the BMI distribution changes in the whole population, independent of age, between time t=1 and t=T. Essentially it represents a common time trend for all ages, and is typically called as the trend in the (latent) level of the locations. Usually we observe an increasing time trend in the location of the BMI distribution, hence in $\hat{\kappa}_1(t)$. At the same time, this common time trend is modulated by the age-specific effect $\hat{\beta}_1(x,g=m)$. $\hat{\beta}_1(x,g=m)$ specifies how fast or slow the median of the BMI distribution among men at age x changes compared to the changes in the whole population. If $\hat{\beta}_1(x,g=m)$ is negative, it indicates that the median of the BMI distribution at age x tends to descent while it growths at other ages. Analogous interpretation can be given for the other three parameters.

We used unisex $\kappa_k(t)$ to drive both male and female age-specific trends. There are attractive reasons for pursuing this strategy: Epidemiologically speaking, a single $\kappa_k(t)$ may enforce greater consistency of the sex differentials, avoiding potential anomalies such as cross-over trends. Statistically speaking, a common $\kappa_k(t)$ is a parsimonious way of linking the trends while avoiding more complicated time series models. We jointly estimated the $\kappa_k(t)$'s by concatenating male and age-specific distribution parameters and subjecting them to SVD.

This process yielded a common $\hat{\kappa}_k(t)$, and two separate sets of $\hat{\alpha}_k(x,g)$ and $\hat{\beta}_k(x,g)$, for males and females.

An important characteristic of the original Lee-Carter method is that it sees the change of the dependent variable only as a function of the time index $\hat{\kappa}_k(t)$ while the age-effect parameters, $\hat{\alpha}_k(x,g)$ and $\hat{\beta}_k(x,g)$, are considered fixed over time. In particular, the time indices are treated as stochastic time series processes that are modelled and forecasted by the classic Box-Jenkins methodology[247]. Once the time indices are forecasted (while the age-effects are kept constant), they can be inserted back into equation (2) to obtain forecasted BCPE distribution parameters. In our study these forecasted parameters are assumed to characterize the future BMI distribution given that the BMI distribution follows BCPE distribution.

To model and extrapolate the k^{th} time series of $\hat{\kappa}_k(t)$ several autoregressive integrated moving average (ARIMA) models can be considered. Lee and Carter proposed the use of the ARIMA(0,1,0) model which is essentially a random walk model (trajectory of successive random steps) with a drift parameter. Although they suggested that different model specification might be more appropriate for other data sets their random walk model with drift is seen in many applications[248]. Similarly, although the changes in mortality rates may be different from the changes in the BCPE distribution parameters, we follow Lee and Carter in adopting the random walk process for our projections because it is a convenient way to extrapolate distribution parameters in a joint manner, and because – as we will see – it fits the data well.

A separate time series model for each $\hat{\kappa}_k(t)$ series, k=1,2,3,4 denoting the latent time trend in the BMI distribution parameters, took the following form:

$$\hat{\kappa}_k(t) - \hat{\kappa}_k(t-1) = \delta_k + \xi_k(t) \tag{4}$$

$$\xi_k(t) \sim N(0, \sigma_k^2), \tag{5}$$

where δ_k, known as the drift parameter, and δ_k^2 were estimated from the time series data. An unbiased estimator of the drift parameter in the random walk with drift model is simply $(\hat{\kappa}_k(T) - \hat{\kappa}_k(1))/(T-1)$[35], whereas the diagonal and off-diagonal elements of the k×k covariance matrix, $\mathbf{\Sigma}$, were estimated one by one taking the pairs of time series of the drift parameters as in (4).

To simulate future parameter estimates of the BCPE distribution we used the last estimated values from the data set, $\hat{\theta}_k(x,g,T)$, to avoid a jump between the last estimated and the first forecasted value[249]. Then the forecasted k^{th} parameter s year ahead from the base year T were given by:

$$\hat{\theta}_k(x,g,T+s) = \hat{\theta}_k(x,g,T) \times \hat{\beta}_k(x,g)\left(\hat{\kappa}_k(T+s) - \hat{\kappa}_k(T)\right), \tag{6}$$

where $\hat{\kappa}_k(T+s)$ denotes the forecasted time index $s \geq 1$ periods ahead of $\hat{\kappa}_k(T)$. $\hat{\kappa}_k(T+s)$ had the following conditional multivariate normal distribution:

$$\hat{\kappa}_k(T+s) \left| \hat{\kappa}_k(T+s-1), \hat{\delta}_k \right. \sim N\left(\hat{\kappa}_k(T+s-1) + \hat{\delta}_k, \Sigma\right). \tag{7}$$

Finally, given the forecasted parameter estimates, the forecasted prevalence of overweight status for age x and year $T+s$ was calculated as follows:

Prevalence of normal weight (8): $\hat{\phi}_{NW}(x,g,T+s) = F_{BCPE}\left(BMI \leq 25; \hat{\boldsymbol{\theta}}(x,g,T+s)\right)$

Prevalence of overweight (9): $\hat{\phi}_{OW}(x,g,T+s) = F_{BCPE}\left(25 < BMI \leq 30; \hat{\boldsymbol{\theta}}(x,g,T+s)\right)$

Prevalence of obesity (10): $\hat{\phi}_{OB}(x,g,T+s) = 1 - F_{BCPE}\left(BMI \leq 30; \hat{\boldsymbol{\theta}}(x,g,T+s)\right)$

Model validation

We validated the model by dividing the data set into two parts. The first part, 1981-2003, was defined as the training data set, and the rest, 2004-2008, was used for validation purposes. We followed the steps described above. That is based on the training data set we fitted a GAMLSS model on the individual BMI data for each age and year combination (20×6=120) assuming BCPE distribution. Then we fitted Lee-Carter bilinear models on these estimated distribution parameters to be able to project them into the future. Finally, we calculated the forecasted prevalence of normal weight, overweight and obesity for the years 2004-2008. We considered our model valid if the prediction intervals contained the observed prevalence values in the POLS data.

7.4 Results

Figure 7.1 presents histograms, estimated probability density functions, and quantile-quantile (Q-Q) plots to visually assess how well the BCPE and the lognormal distribution fits individual level BMI data among women aged 20-29 in the first and last year of the survey, i.e. in 1981 and 2008. It is apparent that the BCPE distribution with four parameters captures the skewness and kurtosis of the BMI distribution much better than the lognor-

mal distribution does. In contrast to the BCPE distribution, the lognormal distribution predicts systematically higher probabilities for larger BMI values than observed, therefore the lognormal distribution is suspected to overestimate the true prevalence of overweight and obesity. A Q-Q plot presents the relationship between quantiles implied by a specific distribution and quantiles as observed in the data. If the two distributions being compared are similar, the points in the Q-Q plot will approximately lie on the line 45 degree line. Assessment of the graphical view of the Q-Q plots in Figure 7.1 indicates that assuming BCPE distribution is more appropriate than assuming a lognormal distribution.

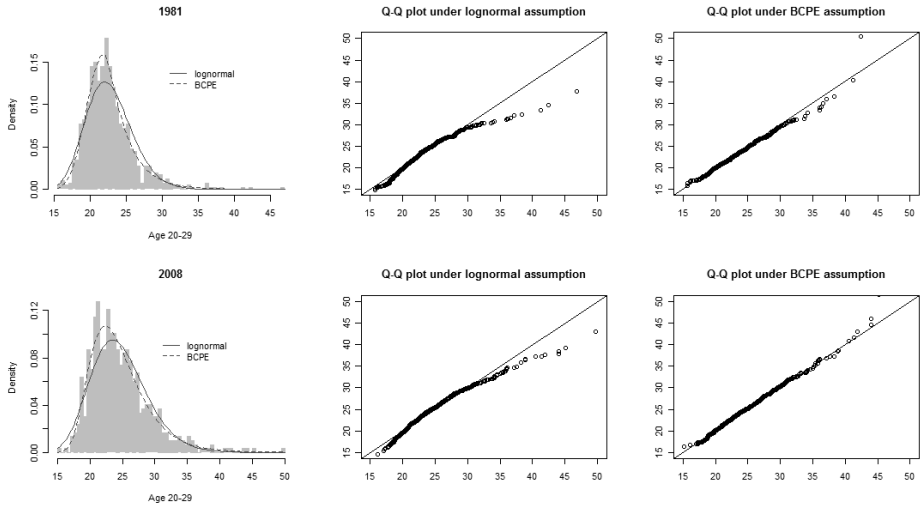


Figure 7.1 Comparison of lognormal and BCPE distribution fitted on BMI data in women of age 20-29, year 1981 and 2008

It is also apparent from the two left panels of Figure 7.1 that that there has been a change not only in the location and variation but also in the shape of the body mass index distribution. Furthermore, emphasizing changes over time, the median BMI rose from 24.0 in 1981 to 25.1 in 2008 (+1.1) for the largest age group, 30-39 years old men, and from 22.2 to 23.8 (+1.6) over the same period time in women. The measure of dispersion of the distributions (that is the logarithm of the coefficient of variation in this case) has also increased steadily. For instance, it increased from 0.13 in 1981 to 0.14 in 2008, and from 0.13 to 0.17 among 30-39 years old men and women, respectively. Besides the location and scale, important aspects of a distribution are the skewness and the kurtosis. Considering for example the BMI distribution among women, it is much more skewed to the right at younger ages than at older ages, and is flatter (platykurtic) at older ages than at younger ages. Explicitly modelling such characteristics of the BMI distribution would have not been possible assuming lognormal or other distributions with less than four parameters.

Based on the full dataset parameter estimates of the Lee-Carter model as in (3), $\hat{\alpha}_k(x,g)$, $\hat{\beta}_k(x,g)$ and $\hat{\kappa}_k(t)$ were estimated that determined the various BCPE distribution parameters, $\hat{\theta}_k(x,t)$, k=1,…,4. These are plotted in Figure 7.2. The first row of the graph depicts $\hat{\alpha}_k(x,g)$, the empirical averages of the estimated location, scale and the two shape parameters of the BCPE distribution over 28 years for each age group. The second and the third rows show $\hat{\beta}_k(x,g)$ and $\hat{\kappa}_k(t)$, respectively. The plots in the third row indicate the overall time trend in a particular distribution parameter. If the trend is increasing, a growing pattern in that specific parameter is seen. This time trend denoted by $\hat{\kappa}_k(t)$ is modulated by $\hat{\beta}_k(x,g)$; the corresponding values are depicted in the second row. These age profiles indicate how fast or slow the specific parameter of the BCPE distribution among people at age x changes compared to the changes in the whole population. If a particular $\hat{\beta}_k(x,g)$ is negative, it indicates that the parameter of the BMI distribution for that age tends to fall while it increases at other ages.

The median BMI was higher among young and middle aged men than among women of the same age. Differences in the average BMI however vanished at ages 60 and above (top left plot in Figure 7.2). Both sexes exhibited a roughly linear increase of the median BMI over time (bottom left plot in Figure 7.2). Important differences between men and women were observed in terms of the skewness and kurtosis parameters (third and fourth column in Figure 7.2). While the BMI distribution was more skewed to the right especially among women, the higher the age was the less right skewed the distribution appeared. The skewness parameter was relatively stable over time (third row third column). Concerning the kurtosis of the BMI distributions, it became less flat and more close to the normal distribution among both genders. The trend in kurtosis parameter seemed to be unbroken and near linear.

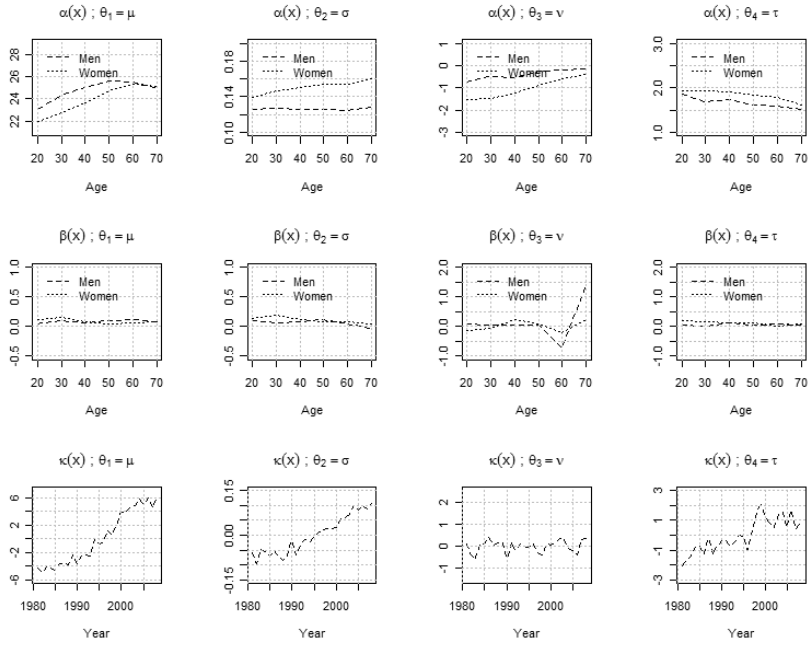


Figure 7.2 Parameter estimates of the Lee-Carter model
Notes: θ_1 may be interpreted relating to the location (median, μ), θ_2 to the scale (approximate coefficient of variation, σ), θ_3 to the skewness (transformation to symmetry, υ), and θ_4 to the kurtosis (power exponential parameter, τ).

It is important to note that there is hardly any interaction between calendar year and age in the Lee-Carter models that are fitted on the BMI distribution parameters (middle row of Figure 7.2). This is true for both men and women. Practically, it means that the estimated $\hat{\beta}_k(x,g)$ are relatively constant across the ages, and that consequently, the trends are the same for everybody.

Various statistics are shown in Table 7.2. The R^2, that is the proportion of variance explained by the Lee-Carter model fitted on the estimated distribution parameters, are shown in the first column. Except for the fourth distribution parameter, the explained variance was at least around 80% indicating a very good overall model fit.

The estimated drift parameters, $\hat{\delta}_k$, their standard error, $\sqrt{V(\hat{\delta}_k)}$, and the estimated correlations between the trends are also presented in Table 7.2. The estimated drift parameters were always positive indicating an increasing trend in the BCPE distribution parameters with mild or weak correlations between the changes.

Table 7.2 Parameter Estimates in the Time Trends of the Lee-Carter Model

Distribution parameter θ_k	R^2	Trend estimate, $\hat{\delta}_k$	Standard error of the estimated trend: $\sqrt{V(\hat{\delta}_k)}$	Correlation: $\frac{[\Sigma]_{j,k}}{\sqrt{[\Sigma]_{j,j}}\sqrt{[\Sigma]_{k,k}}}$			
				Mu	Sigma	Nu	Tau
Mu	98.3%	0.424	0.186	1			
Sigma	90.3%	0.005	0.004	-0.20	1		
Nu	79.0%	0.016	0.124	0.14	-0.27	1	
Tau	57.4%	0.013	0.148	0.04	-0.04	-0.23	1

In Figure 7.3 estimated (for the first and last survey year, 1981 and 2008, respectively) and forecasted (for the year 2020) BMI distributions are shown for men and women at age 20-29 and 40-49. In Table 7.3 detailed results are provided for the prevalence of overweight and obesity in men and women for 1981 and every 20 years afterwards, that is for 2000 and for 2020. Assuming that past trends will continue in the future our model predicted a stable or slow increase in the prevalence of overweight among men until 2020. For example, according to the central estimate of the model the prevalence of overweight will increase to 44.0% among 30-39 years old men. However, there is substantial uncertainty around this future estimate. There is a 5% chance that the prevalence will increase to 47.9% or decrease to 39.3%. Comparing the prevalence estimates of overweight with obesity, larger increases are expected for the latter, especially among middle aged men. The largest increase is expected

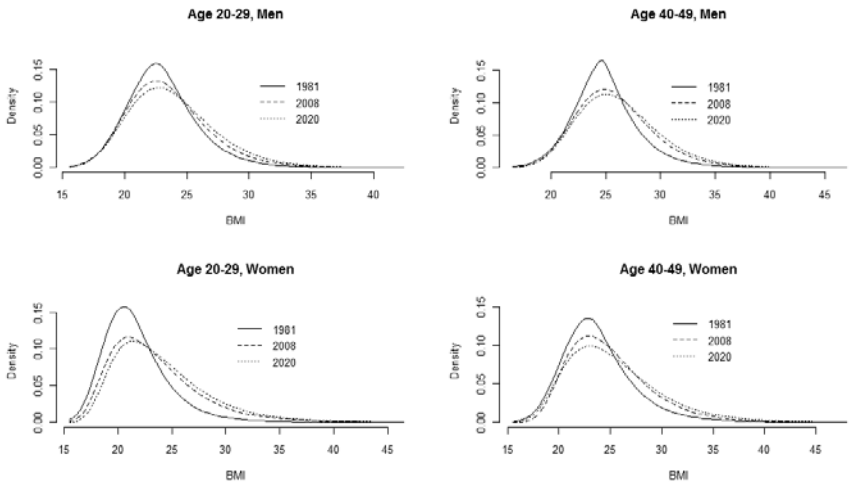


Figure 7.3 Estimated and forecasted BMI distribution for men and women at age 20-29 and 40-49 in various years

Table 7.3 Estimated and forecasted prevalence and 95% prediction interval of overweight and obesity for men and women

Age	Men			Women		
	1981	2000	2020	1981	2000	2020
			Overweight			
20-29	19.7 *(16.9-22.5)*	23.7 *(20.1-27.2)*	27,6 *(25.8-29.6)*	10.7 *(8.5-12.9)*	18.9 *(15.7-22.2)*	23.9 *(21.1-27.6)*
30-39	30.6 *(27.4-33.8)*	36.1 *(32.6-39.5)*	44,0 *(39.3-47.9)*	15.3 *(12.8-17.8)*	24.9 *(21.9-27.9)*	28.6 *(24.8-33.0)*
40-49	39.5 *(35.5-43.5)*	45.1 *(41.4-48.9)*	43,9 *(40.6-46.9)*	24.8 *(21.2-28.4)*	30.4 *(27.1-33.7)*	31.1 *(27.7-34.7)*
50-59	42.7 *(38.5-46.9)*	52.1 *(48.2-56.0)*	47,9 *(43.7-52.0)*	36.1 *(32.1-40.1)*	35.9 *(32.3-39.7)*	34.8 *(32.5-36.9)*
60-69	43.8 *(39.0-48.7)*	51.4 *(46.6-56.2)*	51.0 *(47.6-53.4)*	37.3 *(32.8-41.9)*	42.9 *(38.2-47.6)*	38.9 *(37.0-41.3)*
70+	43.6 *(37.8-49.4)*	45.7 *(40.5-50.9)*	51.6 *(45.8-56.7)*	37.9 *(32.6-43.1)*	40.3 *(35.6-45.0)*	40.2 *(37.5-42.1)*
			Obese			
20-29	2.2 *(1.2-3.2)*	3.3 *(1.8-4.8)*	5.8 *(4.2-7.7)*	2.2 *(1.2-3.3)*	5.5 *(3.6-7.4)*	10.2 *(7.1-13.8)*
30-39	5.3 *(3.7-6.9)*	6.9 *(5.0-8.7)*	12.4 *(9.3-15.8)*	3.6 *(2.3-4.9)*	8.0 *(6.1-9.9)*	17.2 *(12.4-22.9)*
40-49	6.0 *(4.1-8.0)*	10.4 *(8.1-12.7)*	14.9 *(12.1-18.4)*	6.2 *(4.2-8.2)*	10.6 *(8.4-12.8)*	14.9 *(11.4-18.9)*
50-59	7.1 *(4.9-9.2)*	14.5 *(11.8-17.3)*	20.2 *(15.8-25.0)*	11.7 *(9.0-14.4)*	14.8 *(12.0-17.6)*	17.1 *(15.0-19.8)*
60-69	6.8 *(4.4-9.3)*	11.0 *(8.0-14.0)*	19.0 *(14.4-24.4)*	14.9 *(11.6-18.3)*	14.8 *(11.4-18.2)*	20.2 *(17.8-22.5)*
70+	7.9 *(4.7-11.0)*	7.1 *(4.4-9.8)*	9.8 *(5.5-14.5)*	11.5 *(8.0-14.9)*	15.8 *(12.3-19.3)*	19.0 *(16.6-22.3)*

for people aged 50-59 for whom the prevalence of obesity is expected to increase from 14.4% in 2008 to 20.2% by 2020. Very similar trends stand out for women. While the prevalence of overweight is likely to stabilize in the future, the prevalence of obesity is expected to further increase with considerable uncertainty: 17.1% (CI: 15.0%, 19.8%) by 2020. For illustrative purposes we present two further figures in Figure 7.4 and Figure 7.5 on how the prevalence of obesity and the uncertainty around the estimates is anticipated to develop over time for men and women aged 30-39.

We evaluated the proposed model by forecasting the prevalence of overweight and obesity based on the training data set (years: 1981-2003) and validating the forecasted points by comparing them to the observed ones in POLS. We considered our model valid because

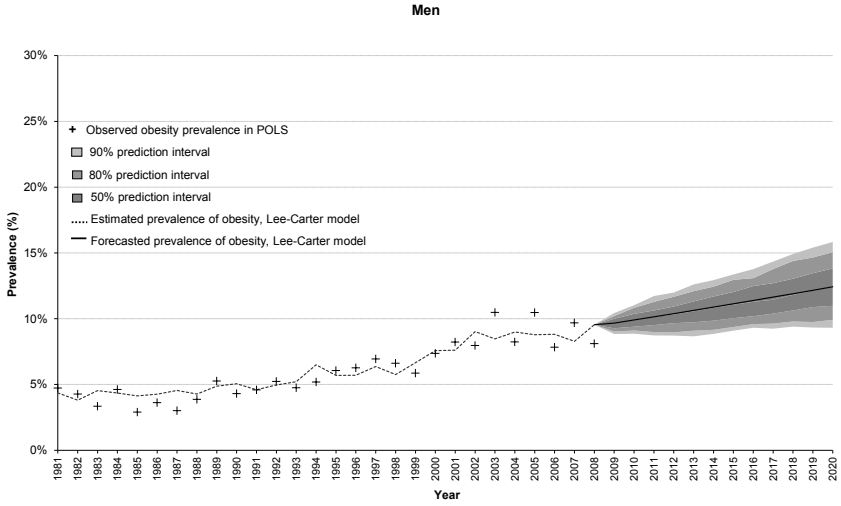


Figure 7.4 Estimated and forecasted prevalence of obesity among men aged 30-39, 1981-2020

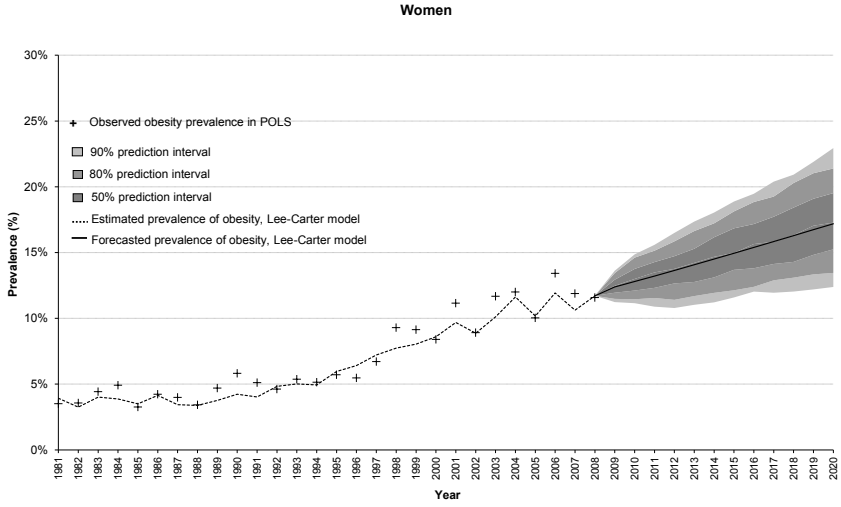


Figure 7.5 Estimated and forecasted prevalence of obesity among women aged 30-39, 1981-2020

the prediction intervals for the years 2004-2008 consistently included the point estimates of those based on the POLS survey. Although the predicted overweight prevalences were below the actual percentages for some ages - making the model somewhat optimistic in terms of the forecasts, the confidence interval around the point estimates always overlapped with the prediction interval around the forecast. We present these results in Figure 7.6 for men and in Figure 7.7 for women in Appendix 7B.

7.5 Discussion

We have modelled and forecasted the BMI distribution of the Dutch adult population by assuming that individual level BMI follows a BCPE distribution. The advantage of fitting BCPE distribution was that it allowed modelling not only the location and the scale but also other parameters of that, i.e. parameters that may be interpreted relating to the skewness and kurtosis. In addition, important changes in the shape of the distribution would not have been captured if we had assumed, for example, lognormal distribution as is often done[238,243]. Although it has already been acknowledged that the BMI distribution is characterized by large increases at the upper end of the distribution[237,238], it has not been modelled explicitly before. Our method was also novel in the way we extrapolated the BMI distribution, and consequently, any quantity that depends on the distribution such as the prevalence of overweight and obesity. We adapted the Lee-Carter model, which was originally developed to forecast mortality, to model the distribution parameters of the BMI distribution. Each distribution parameter was a function of age, time and an interaction between these two. We forecasted the four BCPE distribution parameters that were assumed to characterize the future BMI distributions, and derived the corresponding projected prevalences of overweight and obesity.

There are a few favourable features of our forecasting strategy. First, it is easy to implement and is based on a well-established method for forecasting mortality. Compared to smoothing methods, that have been used to forecast mortality[250], the Lee-Carter offers the advantage of having parameters with easy interpretation. Second, uncertainty around the forecasts are estimated by standard time series analysis techniques in a manner that take into account the sampling variation of the historical data on that the model is fitted. Finally, future predictions of the prevalence of overweight and obesity consider the joint tendency of the background parameters over time that characterizes the distribution of BMI.

Comparison to other approaches

Previous studies typically have used data from large scale longitudinal surveys in which individuals were followed over time. In contrast, our study was based on repeated cross sectional survey data, i.e. data in each year was an independent sample. One of the main advantages of using longitudinal survey data compared to repeated cross sectional data is that, usually, the estimated trends exhibit less variation because the observations within individuals are positively correlated. We could not benefit from the advantages of good quality longitudinal data, and our trend estimates were surrounded by substantial uncertainty due to sampling variation. This uncertainty rolled into and was reflected in the projections as well. Most of the time, however, good quality longitudinal data is not available and researchers have to rely on independent cross sectional survey data. For such situations our modeling strategy,

based on the idea of the Lee-Carter model developed for forecasting life expectancy from annually observed mortality statistics, is a novel, straightforward, and a flexible approach.

The main advantage of using GAMLSS semi-parametric regressions was that the parameters provided a good overview of the changes in the BMI distribution by age groups, gender and over time, assuming that BMI follows BCPE distribution. However, the GAMLSS is a versatile statistical model and allows for many different distributions to be fit. In the case of our study, it may be that a distribution other than the BCPE distribution, e.g. Box-Cox t distribution, fits the BMI data similarly well, or a more parsimonious model with fewer parameters could be constructed. We did not experiment with finding a possible better candidate for the BMI distribution as the BCPE distribution has been already shown to describe BMI well[246].

There are several ways how the presented model may be further explored. For example a GAMLSS semi-parametric regression model could be fitted on the whole data set, i.e. on the individual BMI values, including all ages, years and sexes in the same analysis. Such a model would likely result in smoother predicted time trends of the distribution parameters than the time trends we obtained by the presented approach. Such, smoother time trend can be an attractive feature in the context of forecasting. A potential difficulty of the GAMLSS regression, however, is that the interpretation of the parameters becomes involved if multiple additive (smoothing) regressors are included, and the risk of overfitting the model requires cautiousness as well. In our case this was not an issue because we fitted separate models on each age-year combination separately and did not estimate the effect of age and year in one single regression.

Another path of further developing the model is offered by inspecting Figure 7.2, in that the age interaction parameters of the Lee-Carter model, $\beta(x)$, seem to provide only very little additional information to explaining the variation in the BMI distribution parameters. In other words, the β's exhibit a relatively constant pattern across age. If in reality the true β's are constant, a simpler, additive model without the age interaction parameter could fit the data just as good as a model specified by equation (2). We did not investigate the presence of nested models since the purpose of this paper was to show how the original Lee-Carter model can be applied to forecasting the prevalence of overweight and obesity. Additionally, estimating the parameters of such models would have required different estimation procedure whose description may have diluted the focus of this paper.

Robustness of the results

We have performed a number of different analyses to compare the model with other approaches. Results of the comparisons are presented below.

In Figure 7.4 and Figure 7.5 the last five-six data points may seem a result of an unexpected shift in the time series of obesity prevalence, as after 2003 the trend appear to flatten out compared to preceding years. To formally test whether there was a structural break in the time trend in 2003 we ran a Chow test on the medians of the BCPE distribution. The P value of the test indicated the lack of a potential shift (P value = 0.430). However, because there were only 6 data points after 2003, the test had little power. Hence, we performed a further analysis. We specified a different time series model to the one in (3) by adding a dummy variable that took the value one if the time series data corresponded to 2003 or later, and zero if it corresponded to 2002 or previous years. Such time series model is called an ARIMAX model. Forecasts of the prevalence of obesity resulted in lower values than in our original model but the relative difference in the central estimates between the original model and this analysis were less than 5%.

A second analysis was carried out to compare our model with a multinomial logistic regression approach, and to check how much difference it would make if instead of modelling the prevalence of BMI status through BMI distribution indirectly, we would use such conventional approach on the prevalences directly. Thus, we estimated a multinomial logistic regression model with dummy variables for the age groups and a continuous year variable, and made deterministic forecasts assuming that the age effects would remain the same in the future. While the forecasts for the prevalence of overweight matched the forecasts of the stochastic model quite well, predictions for the prevalence of obesity seemed exaggerated. For instance, our original stochastic forecasts estimated the prevalence of obesity to be 17.1% by 2020 among 50-59 women, whereas the multinomial logistic regression approach predicted it to 22.7%, a 5.6% difference.

Finally, we assessed the sensitivity of our model to reporting bias. It is known that people underreport weight and overreport height rendering the BMI variable somewhat biased. In principle this type of bias could have affected the results of our model because in the POLS survey self-reported weight and height values were recorded. In a study of Dekkers et al.[251], the Dutch working population was found to underreport the body weight by 1.4 kg on average, and overreport the height by 0.7 cm. To investigate how seriously our results could be biased we added random values to the reported weight and height records with mean and standard deviation as reported in Dekkers et al. We then repeated our stochastic forecast analysis, and found that reporting bias had virtually no effect on our forecasts. However, it must be noted that our assessment was based on two naïve assumptions, namely that under- and overreporting was uniform irrespective of the weight or height, and that it did not change over time. Contrary to these assumptions, it has been shown that the most important determinant of underreporting BMI is a high BMI itself[252], and that there might be a diminishing pattern in the reporting bias in BMI over time[253]. Results of these studies

imply that uniform corrections of BMI over time and by BMI itself may not be appropriate. Hence, predictions of future overweight and obesity based on trends in self-reported information may not be accurate, as the reporting bias may affect the apparent increase in self-reported BMI.

Relevance of forecasting overweight and obesity

A vast literature documents that obesity is associated with a number of negative outcomes, from potentially life-threatening conditions to nonfatal chronic illnesses. Although a complete review is beyond the scope of our article, some key findings includes the following. It has been demonstrated that overweight and obesity are associated with increased prevalence of type 2 diabetes mellitus, gallbladder disease, coronary heart disease, high blood cholesterol level, high blood pressure, osteoarthritis[254] and various forms of cancer, including colon[255], breast[256], renal[257] and endometrium[258]. Besides diseases high individual BMI is related to increased mobility disability[258], ADL disability[98], more years of life expectancy with disability[102,259] and reduced self-rated health status[260]. Overall, the effect of overweight and obesity on health and economic factors has become a key topic of public policy in many developed countries. Forecasting future levels of the obesity and informing policy makers about its impact in terms of the health burden is therefore of high importance.

Conclusion

Our study suggests that the strategy to model the BMI distribution directly and derive corresponding prevalence estimates is superior to modelling BMI categories because of the changes in the shape of the BMI distributions. In turn, forecasting the prevalence of overweight and obesity by extrapolating the distribution parameters using the Lee-Carter methodology appears to be an insightful and flexible strategy.

Appendix 7A The Box-Cox power exponential distribution (BCPE)

Let Y be a positive random variable having a Box-Cox t distribution[246], denoted by BCPE(μ,σ,ν,τ), defined through the transformed random variable Z given by (2), where the random variable Z is assumed to follow a truncated standard power exponential distribution with power parameter, $\tau > 0$, treated as a continuous parameter.

The pdf of Y ,a BCPE(μ,σ,ν,τ) random variable, is given by

$$f_Y(y \mid \mu,\sigma,\nu,\tau) = \frac{y^{\nu-1} f_T(z)}{\mu^{\nu}\sigma F_T\left(\frac{1}{\sigma|\nu|}\right)}$$

for $y > 0$, where $\mu > 0$, $\sigma > 0$ and $-\infty < \nu < \infty$, and where z is given by (2) and $f_T(t)$ and $F_T(t)$ are respectively the pdf and cumulative distribution function of a random variable T having a standard power exponential distribution, i.e. $T \sim PE(0,1,\tau)$. If the truncation probability $F_T\left(-\frac{1}{\sigma|\nu|}\right)$ is negligible, the variable Y has median μ.

Appendix 7B

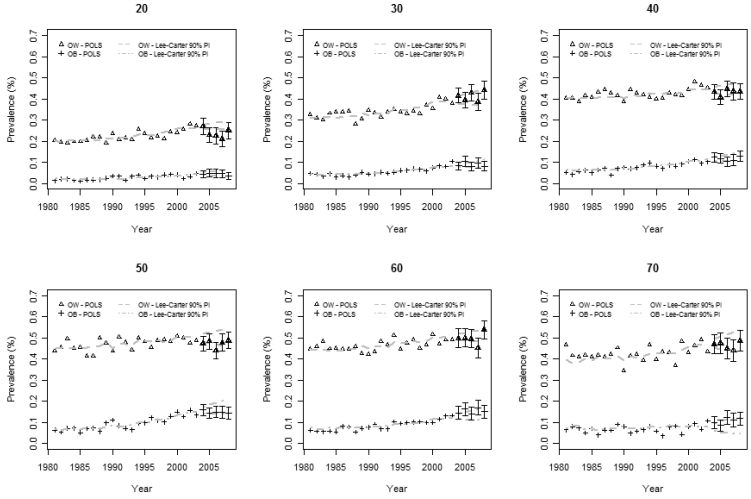


Figure 7.6 Predicted prevalence of overweight and obesity for the last five years 2004-2008 based on 1981-2003, Men

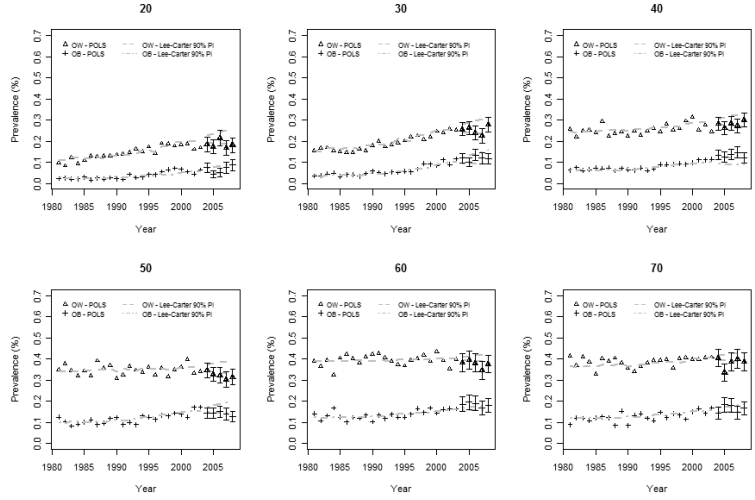
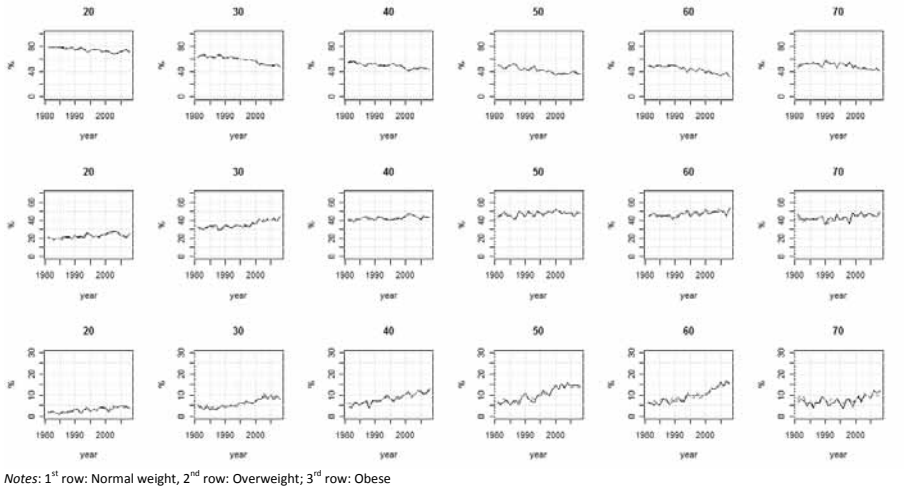


Figure 7.7 Predicted prevalence of overweight and obesity for the last five years, 2004-2008, based on 1981-2003, Women

Appendix 7C

Figure 7.8–Observed versus fitted BCPE prevalence, Men



Notes: 1st row: Normal weight, 2nd row: Overweight; 3rd row: Obese

Figure 7.8 Observed versus fitted BCPE prevalence, Men
Notes: 1st row: Normal weight, 2nd row: Overweight; 3rd row: Obese

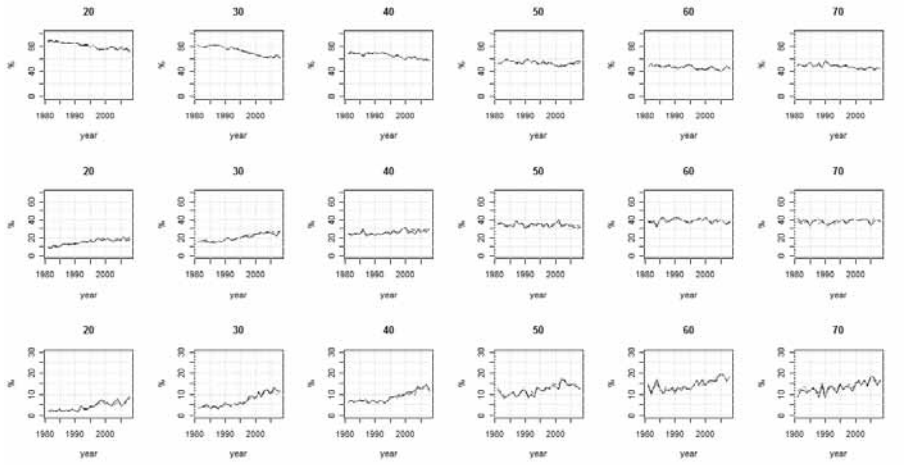


Figure 7.9 Observed versus fitted BCPE prevalence, Women
Notes: 1st row: Normal weight, 2nd row: Overweight; 3rd row: Obese

Chapter 8

General discussion

In this chapter key findings of the thesis are discussed and main conclusions are drawn that follow the organization of the thesis. Firstly, the findings that augment the understanding of how risk factors are associated with health expectancy are summarized (based on Part I). Secondly, the results of the studies that relate to forecasts of health expectancy are reviewed (based on Part II). The summary of the core findings is followed by an evaluation of the methods, and what this thesis adds to the already existing literature. Finally, a number of implications are outlined that this thesis may have on future research and policy making.

8.1. Overview of findings with reference to risk factors associated with health expectancy

Results of Chapter 2 suggest that the relationship between disability and all-cause mortality in the general population is substantial. At any age, disabled persons face a higher risk of mortality than the non-disabled. The magnitude of the relationship, however, varies by disability measure. Our empirical results showed that severe measures of disability, i.e. ADL and mobility disability were strongly associated with death, but that a mild disability measure, i.e. OECD disability indicator, expressed a less significant elevation in death risk. A large proportion of the excess mortality risk associated with disability could be explained by risk factors preceding the onset of disability. Whereas for mild disability this risk difference could be explained by chronic diseases and other risk factors related to socio-demographic status, i.e. education, marital status, and lifestyle, i.e. smoking but not overweight, an independent effect of more severe disabilities on mortality could not be ruled out.

In Chapter 3, the relationship between overweight status, smoking status and both total life expectancy and life expectancy with disability in Western-Europe was explored. It was demonstrated that overweight people can expect to live slightly longer than those with normal weight, which – as suggested by other studies – might be a consequence of the protective effect of being overweight on mortality in disabled populations. In contrast, overweight and obese people can expect to live considerably longer with disability. Smoking had a different relationship with life expectancy and life expectancy with disability. Smoking was associated with a lower total life expectancy, but with an unchanged life expectancy with disability compared to non-smoking. Similar patterns were observed for men and women, and for low and high educated populations.

The relation between overweight and mortality has been a controversial topic. In Chapter 3 it was shown that overweight is associated with protection for dying among the disabled, while the relationship between obesity and mortality is modest, especially among men. These results reaffirm recent doubts about overstated concerns of overweight and obesity in terms of excess mortality[124]. Possible explanations for the small but protective mortality effect of overweight include improved survival of overweight persons from major diseases

of developed societies, e.g. heart failure[125] or CVD[126], and a better nutritional status providing necessary reserves during chronic disease[127]. Recent studies confirmed the protective effect of overweight as well documenting that increased BMI protects against mortality after hospitalisation[128].

In addition to the effect of lifestyle, in Chapter 4, educational differences in health expectancy and life expectancy before and after official retirement age were quantified across 10 countries in Western Europe. Populations with a higher level of education can expect to live more years free of disability before retirement, suggesting fewer problems in reaching pension age in good health. People with a higher educational level can also expect to live longer after retirement, implying that they represent a greater liability for pension funds. For life expectancy, larger differences were found among men than among women, whereas differences in health expectancy were alike for both men and women. Similar patterns emerged in all countries, although the differences tended to be larger in southern states. These findings are well in line with and complementary to the results documented in Chapter 2, where it was found that lower educated people had higher risk of death than the higher educated ones. In Chapter 4 results were presented not only in terms of mortality and consequent life expectancy but also on health status and health expectancy. Higher educated populations were found not only to live longer but also to live longer in good health.

8.2 Overview of findings with reference to forecasting health expectancy

Chapters of Part II presented projections of health expectancy, long-term care expenditure and prevalence of obesity. These quantities are all related to disability, a form of poor health status. Health expectancy was defined in terms of life expectancy with and without disability, receipt of long-term care is largely determined by disability status, and obesity is strongly related to disability as well. Another common feature of the chapters is the methodology behind the projections; each was an adaptation of the Lee-Carter model that was originally developed to project mortality[31].

To forecast health expectancy a theoretical framework for a multistate life table model was proposed, in which the transition probabilities depended on age and calendar time. In Chapter 5 it was described how to model and project these transition rates by the Lee-Carter method, and illustrated how it could be used to forecast future health expectancies with prediction intervals. The model was applied to the Dutch population aged 55 and older, and health expectancies were forecasted up until the year 2030. Additionally, the dynamic relationship between disability-free life expectancy and life expectancy was analyzed, and probability distributions were attached to different future scenarios of compression or expansion of disability. The results showed that both total life expectancy and disability-free life expectancy, measured in terms of OECD disability, are likely to increase till 2030 among

men and women. According to the central estimates of the model, LE will increase from 24.2 in 2005 to 26.8 years by 2020 and to 28.2 years by 2030. The projected increase in LE for women was somewhat smaller, from 28.2 in 2005 to 29.8 and 30.6 by 2020 and 2030, respectively. DFLE among men (women) was anticipated to increase from 19.1 (17.7) in 2005 to 21.9 (20.4) and 23.4 (21.5) years by 2020 and 2030, respectively. DFLE relative to LE was forecasted to rise from 78.9% (62.7%) in 2005 to 81.7% (68.4%) and to 82.8% (68.4%) during the same period for men (women). There was substantial uncertainty around the forecasts: by 2030, the width of the 95% prediction interval of DFLE was about 10 years for both men and women. When compression of disability over the projection period was measured in number of years, the probability of a compression was approximately 50% for men, and 60% for women. If compression of disability was measured in a relative sense, i.e. disability-free life years as a proportion of total life expectancy, then a compression was more likely to occur; the probability of compression was around 65% for the two sexes.

In Chapter 6 the impact that population aging will possibly exert on future levels of aggregate long-term care expenditures was forecasted. For that, a further developed version of the model in Chapter 5 was made use of. This model projected life expectancy without disability, life expectancy with mild and life expectancy with severe disability, and it attached expenditures of home care and institutional care use to the health expectancies. Accordingly, the model accounted for the likely future changes in health expectancy. Whereas both total life expectancy and life expectancy with mild disability were forecasted to increase, life expectancy with severe disability was projected to remain constant as the years lived with severe disability were postponed to higher ages. Consequently, home care spending, associated with mild disability was expected to grow, while the substantially more expensive institutional long-term care expenditure, related to severe disability, was likely to remain fairly stable. Population aggregated long-term care expenditure for the 55+ Dutch population was projected to rise by approximately 56% by 2030. However, this increase was mainly due to the ageing of the baby boom cohort, and not due to increasing per capita long-term care costs.

In Chapter 7 the BMI distribution of the Dutch adult population was modelled and forecasted by explicitly taking into account the shape of the distributions. The median BMI rose from 24.0 in 1981 to 25.1 in 2008 (+1.1) for 30-39 years old men, and from 22.2 to 23.8 (+1.6) over the same period time in women. With respect to the BMI distribution among women, it is much more skewed to the right at younger ages than at older ages, and is flatter (platykurtic) at older ages than at younger ages. Based on the projected BMI distribution, future prevalences of overweight and obesity were derived. Assuming that past trends will continue in the future a stable but slow increase was predicted for the prevalence of overweight until 2020 among men and women. Concerning the prevalence of obesity, larger

increases were expected especially among middle aged people. For men aged 50-59 the prevalence of obesity was expected to increase from 16.6% in 2008 to 20.2% by 2020. Very similar trends were found for women.

8.3 Methodological considerations

Some important factors influencing the validity of the results in this thesis should be carefully considered. The potential impacts of the limitations were discussed in detail in Chapters 2-7. Here we highlight some of the limitations that are not specific to the individual chapters.

Strengths and weakness of data

In Chapter 3 and Chapter 4 we made comparisons of health expectancy between groups with certain risk factors using the European Community Household Panel (ECHP)[107], a large international database of Western European countries comprising information on disability, mortality and various risk factors. This large international study allowed for a comprehensive overview assessing the magnitude of the association between the several risk factors and health expectancy.

A main advantage of the ECHP was the availability of the large number of observations using an identical survey design and survey questionnaire for all participating countries. These common questions ensured a much higher degree of comparability between countries than would be possible using national data sources. International comparability of data in health expectancy is, however, imperfect. There were a number of differences between the countries that persisted despite efforts at standardization. The main differences were caused (1) by cross-national variations in the data collection, (2) by variations in factors such as people's perception of health problems, and (3) by their propensity to report perceived health problems. These differences among the countries (or even within the countries) require caution by interpreting the results. Keeping the limitations in mind, it was recommended to focus on the patterns common to all countries, rather than on the cross-national variations.

Since ECHP is a longitudinal data, non-response and attrition might have been a problem if they were related to the risk factors or to the outcome variables disability and mortality. A few studies have explored the association between attrition and disability status in the ECHP. For example, analyses on attrition showed a positive relationship with worsening health status[118] and a weak relationship with educational level[119]. Besides these studies assessing the data quality, we assessed the probability of dropout in relation to characteristics at the last wave in which the respondent participated. The odds of loss to follow-up for other reasons than death or institutionalization was found to be hardly related to disability status, sex, overweight status or educational level (relative differences were about 10 per cent or less). Slightly higher risk of attrition in former and daily smokers was found though (relative

difference 15 per cent). These findings imply that differential retention was unlikely to have a major impact on our life expectancy and health expectancy estimates.

In Chapter 2 of Part I and in all the chapters of Part II, data from a repeated cross sectional surveys were used covering information of the community-dwelling population of the Netherlands between 1981 and 2008. Non-inclusion of the institutionalized population was a limitation in all of these studies, introducing some bias in the outcomes. In Chapter 2, however, where the outcome was a relative measure, the relative risk of mortality among disabled and non-disabled, we expected the bias to be of little concern[87]. In the chapters of Part II, where the outcome was measured in absolute terms, i.e. life years, prevalence or BMI, sensitivity analyses indicated that the possible bias was negligible compared to the uncertainty around the central estimates of the outcome.

The POLS survey contained information on many aspects of health, however the data was self-reported. By data collection face-to-face interviews were made and written questionnaires were filled in, which can result in either under- or over-reporting of the variables of interest, and eventually may cause biased outcomes. In our studies three particular outcome variables were of primary interest: disability status, weight and height of the participating individuals.

The possibility of over- or underreporting the prevalence of disability could not be excluded, however measurement of functional limitations by self-report are documented to be consistently associated with performance and reflect similar assessment of function[86,113]. With regard to the individuals' weight and height it is known that people tend to underreport weight and over report height rendering the BMI variable somewhat biased[115,117]. In principle reporting bias could affect the results in our projection model of future obesity levels in Chapter 7. To address it, the potential impact that the average magnitude of reporting bias – elicited by a study of Dekkers et al.[251] – could cause to the results was assessed. Assuming that the patterns of reporting bias did not change over time, virtually no effect on the future obesity prevalence estimates was found.

In the chapters of this thesis, the pattern of reporting bias was assumed to be constant over time. Such assumption however may be too restrictive as a study by Salomon et al.[261] reported inconsistent trends of health status in nationally representative surveys in the U.S., offering an important reminder that caution is warranted in using self-rated health measure to monitor trends in population health. In another study that focused on reporting bias in self-reported BMI it was shown that the most important determinant of underreporting BMI is a high BMI itself, and that there is a likely diminishing pattern in the reporting bias in BMI over time[252]. Results of these studies imply that uniform corrections of misreporting

over time may not be appropriate. Consequently, predictions of future quantities based on trends in self-reported health status or BMI may not be accurate, as the reporting bias may non-uniformly affect the outcomes.

Method of forecasting

The methodology behind the projection studies in Part II was based on the Lee-Carter model that was originally developed to forecast mortality. There are a few important assumptions behind the Lee-Carter model that may have an effect on the two groups of variables of interest, i.e. transition rates connecting health states, and distribution parameters of the body mass index probability distribution.

Every model is a simplification of reality and therefore certain assumptions are made during the construction of the model. One of the assumptions made in the Lee-Carter model, and consequently in our adaptations as well, is that the future evolution of the trend associated with the variable of interest, is a function of the observed historical data on which the model is fitted. As a result the central estimates of the predictions are in some way extrapolations of the past, and the uncertainty around these estimates, the prediction intervals, reflect the variability in the historical trends. If there is a clear trend in the historical data then the prediction intervals are suggestive, that is relatively narrow. Such a situation was seen at mortality rates and at the BMI distribution parameters in Chapter 5-7. However, if there is a less clear trend in the data, the prediction intervals are relatively wide providing moderately useful information with regard to the future. Such a situation was seen at incidence rates in Chapter 5.

Once a model has been developed and its parameters have been estimated, it is important to consider whether it is a good model or not. For stochastic mortality forecasting models, Cairns et al.[32] proposed a checklist of criteria against which a model can be evaluated. Among many technical criteria concerning how projections are carried out, the checklist includes that the model should be consistent with historical data, the parameter estimates and the model forecasts should be robust and long-term trends should be biologically plausible.

A specific assumption of the Lee-Carter model concerning the constant age parameters needs to be addressed. In particular, the postulate of time-fixed age-specific parameters has been criticized for being unrealistic if the time horizon of the historical data on which the model is fitted, is long[262]. Critics say that the contribution of a given age to the changes of mortality may vary over time due to complex changes in, for example, medical technology, population health or living environment. It is indeed quite often the case with mortality models that long time series data are used where the age parameters are subject to changes. In the studies of Part II the time horizon was moderate or short compared to mortality

studies, and hence non-constant age effects were less of a concern. Furthermore, the time window of the forecasts was also relatively short compared to what it has generally been for mortality forecasts, causing smaller potential problems in our forecasts.

The Lee-Carter methodology roots in the analysis and extrapolation of cross sectional data. In general, a different approach could be based on utilizing information from longitudinal data in that individuals are followed over time. One of the main advantages of using longitudinal survey data compared to repeated cross sectional data is that, usually, the estimated trends exhibit less variation because the observations within individuals are positively correlated. In our studies the trend estimates were surrounded by substantial uncertainty due to sampling variation in the surveys. This uncertainty rolled into and was reflected in the projections as well. Most of the time, however, good quality longitudinal data is not available and researchers have to rely on independent cross sectional survey data. For such situations the idea (and application) of the Lee-Carter model is a novel, straightforward, and a flexible approach.

8.4 What this thesis adds to the literature

In this section I overview what this thesis adds to the literature in terms of content and methodology.

Part I.

Most often disability has been assessed in cross-sectional studies without information on mortality[209]. The few longitudinal studies that have been conducted tend to emphasise incident disability rather than the trajectory of disability following onset. Thus, while the onset of disability has been extensively researched[59-67], there has been far less investigation into the mortality risk associated with disability. In Chapter 2 the association between mortality and disability was assessed and the extent to which this relationship can be explained by risk factors associated with the disablement was quantified.

In Chapter 3 life expectancy and life expectancy with disability was estimated among normal weight, overweight and obese smokers and non-smokers in Western-Europe. Early studies of U.S. populations found large effects of being overweight and obese on both premature death risk and disability prevalence[98,99]. However, these effects reflect life histories of older cohorts and there is evidence that the excess risk of higher body mass index on mortality has diminished over time[100,101]. Studies using more recent data from the U.S. showed smaller impacts on life expectancy, but still large effects on disability-free life expectancy[102,103]. The evidence on the impact of overweight status on life expectancy and the burden of disability in Europe was largely incomplete. Previous studies were based on small sample sizes and

were restricted to single countries[105,106]. We provided a general picture of the population health associated with overweight status in Western European countries as a whole.

In Chapter 4 the level of socioeconomic differences in life expectancy and health expectancy in Western-Europe was assessed. Estimates of socioeconomic differences in health expectancy are available for an increasing number of European countries[61,129-134]. However estimates from national studies are not comparable due to large variations in the data sources used, the age ranges covered, and the health and socioeconomic indicators employed. Therefore an overview of the magnitude of socioeconomic differences in total and healthy life years at old age in Europe was lacking. Our study estimated the magnitude of educational differences in disability-free life expectancy between age 50 and 65, and in life expectancy and disability-free life expectancy after age 65.

Part II.

In Chapter 5 a multistate life table model framework was presented that can be used to forecast total life expectancy and health expectancy of an individual. In the demographic literature a large number of multistate projection studies can be found, however these models forecast the size of a population. These projections are typically based on cohort components of demographic change including births, deaths, and migration. The transitions between the modelled states are based on transition rates that may change in time[166] and / or may vary between subpopulations. Projections for sub-populations have been performed by region[167], educational status[168], household status[169], labour force participation[170] as well as by health / disability status[171]. Recently a large-scale research project has been completed in the European Union[172]. One of the goals of the research was to provide the size and age structure of future populations with and without disability. The crucial difference between the demographic projection studies and our forecasting model is that while demographic studies forecasted the size of the population by a multistate method we forecasted the elements of a multistate life table. In other words, our method generates forecasts of a MSLT itself.

In Chapter 6 we investigated the impact that population aging will likely exert on future levels of long-term care spending. While past disability trends have been investigated abundantly[39,199,201-205], only a few studies have exploited the trends to forecast LTC use or spending. We forecasted lifetime and aggregate annual LTC spending for the Dutch 55+ population until 2030 accounting for changing disability patterns that has not been done before.

In Chapter 7 we forecasted the prevalence of overweight and obesity for the Dutch adult population until 2020. Although our study was not the first one to forecast overweight status,

we proposed a new approach. A previous study, for example, estimated annual proportional changes of the mean BMI by age, sex and race, and projected these changes several years ahead[233]. Another study used quantile regressions to model the increase of BMI for each of the 1st through 99th BMI percentiles, and extrapolated the trends[234]. Other projections used techniques that model BMI status directly, i.e. either based on longitudinal data estimating transitions probabilities and making cohort-wise projections, or transforming the categorical data into multivariate normal distribution and fitting a time series model[235,236]. Although it has already been acknowledged that the BMI distribution was becoming less skewed[237,238], it has not been modelled explicitly before. Unlike these previous approaches our method explicitly took into account changes in the shape of the BMI distribution. In addition, modelling the BMI distribution itself and deriving prevalence estimates indirectly seems to be a better choice than modelling the prevalence of overweight status directly because of the changes within these categories.

8.5 Implication for future research and policy relevance

In Chapter 4 it was argued that the systematic reforms aimed at increasing pension(able) age should take into account the trend of rising life expectancy, and – as shown in Chapter 5 – rising health expectancy. However, such restructuring rarely acknowledges the differences in life and health expectancies between socio-economic groups, for example groups of different educational level. Educational inequalities favouring the higher educated exist on both sides of the retirement age. Although being disabled does not necessarily mean being unable to work, and being non-disabled does not necessarily mean being able to work; good health does increase the likelihood of participation in the labour force and decreases the probability and need of early retirement[156]. We suggest that efforts should be put into studying opportunities to decrease the educational differences in life expectancy and health expectancy. More importantly, policy makers should investigate the possibility and consequences of making the retirement age more flexible by allowing for differential official retirement age for different educational status groups.

In Chapter 5 it was claimed that the Lee-Carter model is generalizable to multistate settings and it was demonstrated for disability-free life expectancy. This approach could be used for forecasting working or active life expectancy as well because these measures are estimated by similar multistate life table models. Forecasts of life and health expectancy for different socioeconomic groups should be prepared as well because such projections might contribute to alleviating the tension around social security systems, including the pension systems in the Netherlands and in Europe[38].

In Chapter 6 it was demonstrated that long-term care expenditures are likely to increase by 56% in the Netherlands in the next twenty years. It is therefore important that policy makers

are prepared for the financial consequences and for the increase of LTC demand in terms of health care provision facilities and equipment. Follow-up studies should be carried out to investigate how to mitigate these likely changes and their impact on the health care sector. Because increasing costs of LTC are almost purely the result of the ageing of the baby boom generation, increasing longevity has little influence on the demand for future LTC since the period of severe disability is postponed, not extended. Stimulating further compression of disability, however, is one of the possibilities that could alleviate the consequences of population aging on the long-term care spending growth. The findings in Chapter 2 indicate that postponing the occurrences of chronic diseases can be a successful strategy for which prevention, health promotion, or screening programmes may be needed.

Strongly related to a possible compression of disability (and is largely influenced by) the future changes in the prevalence of overweight and obesity. In Chapter 3 it was demonstrated that overweight and obesity are strongly associated with disability compared to normal weight. If the changes in the prevalence of obesity keep continuing as it did in the past 30 years, the goal of compressing disability will be very difficult to reach. However, awareness of obesity is much greater now than it was before, and lessons can be learnt from other countries, especially the U.S. and U.K., where the prevalence of obesity is already particularly high[229,263]. Nonetheless, it should be researched whether, and if yes, how large the impact of increasing prevalence of overweight and obesity will have on future mortality, incidence of diseases, future health care needs or even on the health insurance industry. We have no doubt that obesity research including understanding the causes, consequences, prevention and treatment will continue to play a major role in public health research. When it comes to implementing all of this work, one of Mahatma Ghandhi's sentences comes to my mind when he was asked what he thought of Western civilization. He said: „I think it would be a good idea".

Summary

English

People today in the Western world live longer than previous generations did. The dramatic increase of life expectancy, the expected remaining years of life at a given age, in the last century is considered as one of the great achievements of modern societies. Between 1970 and 2009 the average length of life in the old EU member states increased from 68.7 to 78.0 among men and from 74.9 to 83.5 among women. Contrary to the length of life, the length of the healthy fraction of life is much more difficult to quantify because there is no harmonized way of expressing and measuring health. In the last two decades considerable efforts have been put into the development of summary measures of population health. One class of these measures is health expectancies that not only summarize information of mortality alone but combines information of mortality with health outcomes. In general, health expectancy is an estimate of the number of remaining life years that a person at a certain age is expected to live with or without ill-health. In its most commonly employed form health expectancy is a functional health status measure, yielding the disability-free life expectancy and the life expectancy with disability.

So far the main focus of health expectancy research has been put on the trends of health expectancy in a given country or on cross-country comparisons between countries at a given period of time. Less emphasis has been placed on determinants of health expectancy, such as obesity, smoking or educational level. The **first part** of this thesis presents studies that estimate the extent to which these risk factors are related to health expectancy. In ageing populations it is also an important question whether increases in life expectancy will be accompanied with greater or lesser increases in life years spent in poor health in the future. There may be several implications of a potentially increasing health expectancy, for example retirement policies may be revised or the demand for health care may change. The **second part** of this thesis presents projections of health expectancy, long term care expenditure and prevalence of overweight and obesity.

The relationship between disability and mortality

In **Chapter 2** we estimated the strength of the relationship between disability and mortality and investigated the extent to which this relationship can be explained by risk factors of disability onset. Our results suggest that the relationship between disability and all-cause mortality in the general population is substantial. At any age, disabled persons face a higher risk of mortality than the non-disabled. The magnitude of the relationship, however, varies by disability measure. We also showed that severe measures of disability, i.e. activities of daily living and mobility disability were strongly associated with death, but less severe disability measure, i.e. the OECD disability indicator, expressed a less significant elevation in death risk. A large proportion of the excess mortality risk associated with disability could

be explained by risk factors preceding the onset of disability. Whereas for mild disability this risk difference could be explained by chronic diseases and other risk factors related to socio-demographic status, i.e. education, marital status, and lifestyle, i.e. smoking but not overweight, an independent effect of more severe disabilities on mortality could not be ruled out.

The relationship between overweight status, smoking status and health expectancy
In **Chapter 3**, the relationship between overweight status, smoking status and both total life expectancy and life expectancy with disability in Western-Europe was explored. We demonstrated that overweight people can expect to live slightly longer than those with normal weight, which – as suggested by other studies - might be a consequence of the protective effect of overweight on mortality in disabled populations. In contrast, overweight and obese people can expect to live considerably more years with disability than people with normal weight. Smoking had a different relationship with health expectancy. Smoking was associated with lower total life expectancy, but with an unchanged life expectancy with disability compared with non-smoking.

The relationship between educational status and health expectancy
In addition to the effect of lifestyle, in **Chapter 4**, educational differences in health expectancy and life expectancy before and after official retirement age were quantified across 10 countries in Western Europe. We showed that populations with a higher level of education can expect to live more years free of disability before retirement, suggesting fewer problems in reaching pension age in good health. People with a higher educational level can also expect to live longer after retirement, implying that they represent a greater liability for pension funds. For life expectancy larger differences were found among men than among women, whereas differences in health expectancy were alike for both men and women.

Forecasting health expectancy
To forecast health expectancy we proposed a theoretical framework for a multistate life table model, in which the transition probabilities depended on age and calendar time. In **Chapter 5** we described how to model and project these transition rates by the Lee-Carter method, and illustrated how it could be used to forecast future health expectancies with prediction intervals. The model was applied to the Dutch population aged 55 and older, and health expectancies were forecasted up until the year 2030. Additionally, the dynamic relationship between disability-free life expectancy, measured in terms of the OECD disability indicator, and life expectancy was analyzed, and probability distributions were attached to different future scenarios of compression or expansion of disability. Our results showed that both total life expectancy and disability-free life expectancy are likely to increase till 2030 among men and women but the prediction intervals were wide.

Forecasting long-term care expenditures

In **Chapter 6** we forecasted the impact that population aging will possibly exert on future levels of aggregate long-term care expenditures. For that we used an extended version of the model of Chapter 5. This extended model projected life expectancy without disability, life expectancy with mild and life expectancy with severe disability; it attached expenditures of home care and institutional care use to the health expectancies. Whereas both total life expectancy and life expectancy with mild disability were forecasted to increase, life expectancy with severe disability was projected to remain constant as the years lived with severe disability were postponed to higher ages. Consequently, home care spending, associated with mild disability was expected to grow, while the substantially more expensive institutional long-term care expenditure, related to severe disability, was likely to remain fairly stable. We projected that population aggregated long-term care expenditure for the 55+ Dutch population is to rise by about 56% by 2030, that was mainly due to the ageing of the baby boom cohort and not due to increasing per capita long-term care costs.

Forecasting the distribution of body mass index

In **Chapter 7** we modelled the BMI distribution of the Dutch adult population and forecasted by explicitly taking into account the changes in the shape of the BMI distribution. Although it has already been acknowledged that the BMI distribution was becoming less skewed over the years, this phenomenon has not been modelled and forecasted before. Based on the projected BMI distribution we derived future prevalences of overweight and obesity. Assuming that past trends will continue in the future we predicted a stable but slow increase for the prevalence of overweight until 2020 among men and women. Concerning the prevalence of obesity larger increases were expected, especially among middle aged people. For men aged 50-59 the prevalence of obesity was expected to increase from 16.6% in 2008 to 20.2% by 2020. Very similar trends were found for women.

Conclusion

There is a substantial heterogeneity in life expectancy and health expectancy in subgroups of a population. Our results imply that tobacco control is still highly relevant to the prevention of premature death. Furthermore, interventions aimed at addressing the obesity epidemic are important if an increase of life expectancy with disability is to be stopped as life expectancy continues to grow. Our results also showed that people with higher educational level expect to live more years free of disability before retirement. Therefore those at the bottom of the socioeconomic ladder could be affected disproportionately if the official retirement age is raised.

The steadily increasing life expectancy in the developed countries is a great achievement, but at the same time raises the question of whether living longer lives will be accompanied

by a decrease or an increase of disability during old age. Although there is a concern that the obesity epidemic may, in the long run, increase the prevalence of disability in ageing populations, disability-free life expectancy is still expected to increase. Our results suggest that the obesity epidemic will likely not lower life expectancy and not increase the years lived with severe disability, however it may contribute to the likely increase of the years lived with mild disability. Given that long-term care costs are mainly determined by severe disability, increased years lived with mild disability will likely not increase the per capita demand for long-term care considerably.

Dutch

Tegenwoordig leven mensen –met name in westerse landen– gemiddeld veel langer dan vroeger ooit het geval was. De aanzienlijke stijging in de levensverwachting gedurende de afgelopen eeuw wordt dan ook beschouwd als een van de grootste verworvenheden van de hedendaagse samenleving. Tussen 1970 en 2009 steeg de gemiddelde levensverwachting in de EU-15 lidstaten van 68,7 (74,9) naar 78,0 (83,5) jaar bij mannen (vrouwen). De toename in het aantal gezonde levensjaren is daarentegen veel moeilijker te kwantificeren, omdat er geen eenduidige manier bestaat om gezondheid te definiëren én meten. In de afgelopen twee decennia is er echter veel vooruitgang geboekt in de ontwikkeling van gezondheidsmaten die niet alleen gebaseerd zijn op mortaliteit, maar ook op morbiditeit. Zo is het gangbaar geworden om de gezonde levensverwachting te definiëren als het verwachte aantal resterende levensjaren dat een persoon vanaf een bepaalde leeftijd in goede gezondheid verkeert (d.w.z. zonder gezondheidsproblemen). In de praktijk wordt de gezonde levensverwachting vaak geoperationaliseerd als de resterende levensverwachting zonder beperkingen.

Tot nu toe is bij onderzoek naar de gezonde levensverwachting de nadruk gelegd op trends in de gezonde levensverwachting in een bepaald land of op vergelijkingen tussen landen in een bepaalde periode. Veel minder aandacht wordt er besteed aan determinanten van de gezonde levensverwachting, zoals overgewicht, roken of opleidingsniveau. Het eerste deel van dit proefschrift presenteert dan ook studies die de mate waarin deze risicofactoren samenhangen met gezonde levensverwachting in kaart brengen. Daarnaast resteert binnen het onderzoek de vraag in hoeverre de toekomstige stijging van de levensverwachting gepaard zal gaan met een stijging van levensjaren doorgebracht in een goede dan wel slechte gezondheid. Een stijging van de gezonde levensverwachting heeft namelijk potentieel verstrekkende gevolgen. Zo vraagt dit wellicht om een ander pensioenbeleid om het pensioen te waarborgen, of om een andere organisatie en financiering van de gezondheidszorg om met een toenemende vraag naar de zorg om te gaan. Het tweede deel van dit proefschrift schetst een beeld van een aantal gevolgen, door prognoses te geven van de gezonde levensverwachting, langdurige zorguitgaven, en de prevalentie van overgewicht en obesitas.

De relatie tussen beperkingen en sterfte

In **hoofdstuk 2** hebben we de mate waarin beperkingen en sterfte gerelateerd zijn in kaart gebracht, en onderzocht in hoeverre deze relatie verklaard kan worden door risicofactoren voor het optreden van beperkingen. Onze resultaten suggereren dat de samenhang tussen beperkingen en sterfte in de algemene bevolking sterk is. Deze samenhang is echter afhankelijk van de maat die wordt gebruikt om beperkingen te operationaliseren. Maten voor ernstige beperkingen (zoals een maat die activiteiten uit het dagelijks leven en mobiliteitsbeperkingen meten) laten een sterke relatie tussen beperkingen en sterfte zien maar voor

maten van minder ernstige beperkingen (zoals bijvoorbeeld de OECD beperkingen indicator) is deze relatie veel minder sterk. Een groot deel van het additionele risico op sterfte als gevolg van beperkingen kan worden verklaard door de aanwezigheid van risicofactoren voorafgaand aan het ontstaan van beperkingen.

De relatie tussen de mate van overgewicht, rookgedrag en een gezonde levensverwachting
In **hoofdstuk 3** werden de gevolgen van overgewicht en rookgedrag voor zowel de totale levensverwachting als de levensverwachting met een beperking in West-Europa onderzocht. We hebben aangetoond dat mensen met overgewicht een iets langere levensverwachting hebben dan mensen met een normaal gewicht, wat - zoals gesuggereerd is in andere studies – wellicht een gevolg is van het beschermende effect van overgewicht op de sterfte van mensen met een beperking. Mede als gevolg daarvan kunnen mensen met overgewicht of obesitas aanzienlijk meer jaren leven met een beperking dan mensen met een normaal gewicht. Rookgedrag had daarentegen een andere relatie met de gezonde levensverwachting. Rokers werden geassocieerd met een lagere totale levensverwachting, maar hadden een gezonde levensverwachting die vergelijkbaar was met die van niet-rokers.

De relatie tussen opleidingsniveau en gezonde levensverwachting
In **hoofdstuk 4** zijn verschillen tussen opleidingsniveau in termen van (gezonde) levensverwachting gekwantificeerd vóór en na de pensioengerechtige leeftijd in tien West-Europese landen. We hebben aangetoond dat populaties met een hoger opleidingsniveau meer levensjaren zonder beperking kunnen verwachten, hetgeen suggereert dat hoger opgeleiden vaker de pensioengerechtigde leeftijd bereiken in goede gezondheid. Van hoger opgeleiden is tevens de verwachting dat ze langer leven na het bereiken van deze leeftijd. Verschillen in levensverwachting tussen opleidingsniveaus waren groter binnen mannen dan binnen vrouwen onderling, terwijl de verschillen in gezonde levensverwachting gelijk waren binnen mannen en vrouwen.

Voorspellingen van de gezonde levensverwachting
Om de gezonde levensverwachting te voorspellen hebben we in **hoofdstuk 5** een theoretisch kader voor een zogenaamde multistate model geformuleerd, waarin transitiekansen tussen verschillende gezondheidstoestanden in opeenvolgende levensjaren worden gemodelleerd. Deze kansen werden afhankelijk veronsteld van leeftijd en kalendertijd. We tonen aan hoe de zogenaamde Lee-Carter methode kan worden gebruikt om de gezonde levensverwachting te voorspellen en bijbehorende predictieintervallen te construeren. Dit model wordt geïllustreerd door de gezonde levensverwachting voor de Nederlandse bevolking van 55 jaar en ouder te voorspellen tot het jaar 2030. Onze resultaten laten zien dat zowel de totale levensverwachting en de levensverwachting zonder beperkingen waarschijnlijk tot 2030

stijgen bij zowel mannen en vrouwen, maar dat bijbehorende predictieintervallen groot zijn, hetgeen suggereert dat deze schattingen vrij onzeker zijn voor de lange termijn.

Voorspellingen van langdurige zorguitgaven

In **hoofdstuk 6** hebben we onderzocht welke impact de vergrijzing mogelijk heeft op de toekomstige langdurige zorguitgaven. Daarvoor hebben we een uitgebreide versie van het model uit hoofdstuk 5 gebruikt, waarin de levensverwachting zonder beperkingen, met lichte beperkingen en met ernstige beperkingen werd voorspeld. Uitgaven voor de thuiszorg en institutionele zorg werden gekoppeld aan deze voorspellingen. Zowel voor de totale levensverwachting als voor de levensverwachting met lichte beperkingen werd een toename voorspeld; voor de levensverwachting met ernstige beperkingen werd daarentegen geen verandering voorspeld doordat de levensjaren met ernstige beperking werden uitgesteld tot op een hogere leeftijd. Als gevolg werd een toename in de uitgaven aan thuiszorg voorspeld – die gekoppeld zijn aan de gestegen levensverwachting met lichte beperkingen – terwijl de uitgaven aan de aanzienlijk duurdere institutionele zorg – die in verband worden gebracht met de levensverwachting met ernstige beperking – werden voorspeld redelijk stabiel te blijven. Er wordt verwacht dat de totale langdurige zorguitgaven voor de Nederlandse bevolking van 55+ tot 2030 zal stijgen met ongeveer 56%, welke grotendeels het gevolg is van de vergrijzing van bevolking, en slechts in kleine mate het gevolg is van toenemende per capita langdurige zorguitgaven.

Voorspellingen van de BMI verdeling

In **hoofdstuk 7** hebben we de BMI verdeling van de volwassen bevolking in Nederland gemodelleerd en voorspeld. Hierbij hebben we expliciet rekening gehouden met de veranderingen van de vorm van de BMI verdeling. Hoewel al bekend is dat de BMI verdeling door de jaren heen steeds minder scheef wordt, is dit fenomeen niet gemodelleerd of voorspeld. Op basis van de verwachte BMI verdeling hebben we de toekomstige prevalenties van overgewicht en obesitas afgeleid. Ervan uitgaande dat patronen uit het verleden zich voortzetten in de toekomst, voorspelden we een gelijkmatige –doch langzame – toename van de prevalentie van overgewicht bij mannen en vrouwen tot 2020. Wat betreft de prevalentie van obesitas werden grotere stijgingen verwacht, met name onder mensen van middelbare leeftijd. Zo is de verwachting dat bij mannen van 50-59 jaar de prevalentie van obesitas toeneemt van 16,6% in 2008 tot 20,2% in 2020. Zeer vergelijkbare trends in obesitas werden er voor vrouwen gevonden.

Conclusie

Grote verschillen in zowel levensverwachting als gezonde levensverwachting vonden wij tussen groepen mensen in de bevolking. Ten eerste zijn de verschillen in levensverwachting tussen rokers en niet-rokers groot. Onze resultaten impliceren dat de bestrijding van tabaks-

gebruik nog steeds zeer relevant is voor het voorkomen van voortijdige sterfte. Ten tweede zijn er verschillen tussen mensen met en zonder obesitas (overgewicht). In tegenstelling tot de bestrijding van tabaksgebruik, zijn interventies die gericht zijn op het verminderen van obesitas voornamelijk van belang voor het voorkomen van beperkingen. Tenslotte vonden we ook verschillen tussen opleidingsniveaus. Hoogopgeleide mensen brengen naar verwachting zowel voor als na hun pensionering minder jaren met beperkingen door dan laagopgeleiden. Een verhoging van de pensioengerechtigde leeftijd zou daarom andere gevolgen kunnen hebben voor de groep mensen met een lage sociaal economische status.

De toenemende levensverwachting in ontwikkelde landen is een grote verworvenheid op zich, maar tegelijkertijd dringt de vraag zich op of langer leven gepaard zal gaan met een stijging van de (functionele) beperkingen. In het bijzonder bestaan er zorgen over de lange-termijn gevolgen van de obesitas-epidemie voor het voorkomen van beperkingen in de bevolking. Onze resultaten geven een beeld van de mogelijke gevolgen. Zo neemt de levensverwachting zonder beperkingen waarschijnlijk toe. Hoewel de levensverwachting met lichte beperkingen ook zal toenemen als gevolg van de obesitas-epidemie, zal de levensverwachting met ernstige beperkingen waarschijnlijk niet veranderen. Aangezien de vraag naar langdurige zorg vooral wordt bepaald door het voorkomen van ernstige beperkingen, zal een toenemende levensverwachting derhalve waarschijnlijk niet resulteren in een explosie van de vraag naar langdurige zorg, hetgeen voorheen altijd werd gedacht.

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PhD portfolio

SUMMARY OF PHD TRAINING AND TEACHING ACTIVITIES

Name PhD student: Istvan Matyas Majer
Erasums MC Department: Public Health
Supervisor: Dr. P.H.M. van Baal

PhD period: 2007-2011
Promotor: Prof. dr. J.P. Mackenbach

PhD training	*Year*	*Workload*
Research skills		
Nihes, Erasmus MC		
Public Health Research: Analysis of Population Health	2007	50 hours
Public Health Research: Analysis of Determinants	2007	50 hours
Biotatistical Methods II: Popular Regression Models	2007	120 hours
Medical Demography	2008	30 hours
Bayesian Statistics	2008	40 hours
Erasmus Summer University		
Survival Analysis	2009	30 hours
Glasgow University		
Advanced modelling methods for health economic evaluation	2009	24 hours
Erasmus University		
Time series analysis	2011	120 hours
Tilburg University		
Panel data analysis	2010	120 hours

PRESENTATIONS

National conferences

Projections Of Future Life Expectancy With And Without Disability In The Netherlands.		
VGE (Association of Health Economists) meeting	2009	20 hours
Forecasting long term care costs in the Netherlands		
VGE meeting	2010	20 hours
Forecasting BMI and its consequences for population health: health loss under stochastic dynamics of mortality and overweight status		
VGE meeting	2011	20 hours

International conferences, workshops

Forecasting disability-free life expectancy and life expectancy with disability; an application to the Dutch elderly		
European Conference on Health Economics, Helsinki	2010	30 hours
Modeling and forecasting health expectancy: theoretical framework and application.		
Netspar International Pension Workshop, Amsterdam	2011	30 hours

Teaching activities

Supervising MSc student at Erasmus MC	2009	30 hours

Curriculum vitae

Istvan Matyas Majer was born in Miskolc, Hungary, on the 1st of February in 1981. After his graduation at high school (Foldes Ferenc Gimnazium, Miskolc, Hungary) in 1999, he started studying economics at the Corvinus University in Budapest, and graduated majoring in actuarial sciences in 2004. After graduation he worked as a research fellow at the Health Economics and Health Technology Assessment Research Center at the Corvinus University for two years. In 2006 he started a Master of Science program in Health Economics at the institute of Health Policy and Management at the Erasmus University in Rotterdam. He obtained his MSc degree in 2009 with a thesis on hospital standardized mortality ratios in Dutch hospitals, under supervision of Dr. Xander Koolman and Prof. dr. Ewout Steyerberg. In 2007 September he started a PhD project at the Department of Public Health of the Erasmus Medical Center under supervision of Dr. Anton Kunst (2007-2009), Dr. Pieter van Baal (since 2009) and Prof. dr. Johan Mackenbach.

Acknowledgements

Many of you – who read these lines – may know that tennis is very close to my heart. Tennis is a sport in that you do not necessarily need to win the most points to win a match, and is a sport where besides having a good technique you need to be strong in your mind. They say it is a mental game. In this respect a PhD is very similar to a tennis match, maybe to a match in a Grand Slam in that men have to win three sets. I thought my tennis match lasted four sets, I lost the first one but won the last three. I lost the first because I had to learn how to adapt to the surface of the tennis court. But then I did and eventually it seems I have prevailed. Winning is a nice feeling but there is always a team behind a tennis player that help with the improvements. They share successes and losses, without the team nothing would be possible. This chapter is about you, the 'team'.

I always felt a great gratitude towards Xander Koolman. He was the first person in the Netherlands with whom I could work together, I wrote my master thesis with him. I needed his enthusiasm and unique personality very much at that time, and looking back, I think I needed the topic of the master thesis as well to reinforce my feelings about statistics and modeling. He is the person through whom I got in contact with Professor Ewout Steyerberg, who in turn helped me get the PhD position in the Netspar project at MGZ. I would like to thank you both for being helpful and nice to me when it mattered.

During the years I spent at MGZ I always felt I was surrounded by very smart and erudite people. Wilma Nussleder, my supervisor in the first few months, would have never said 'no' on helping or showing things to me, I really thank you for all your kindness. Anton Kunst, who took over my supervision from Wilma, showed me the basic principles of how to write a scientific paper. Without your guidance and instructions it would have been so much more difficult. Johan Mackenbach, in my eyes, is the living statue of knowledge and efficiency. Every meeting we had resulted in some good advice that I could take and that helped me continue further. Your insightful comments enormously improved my papers, I am really grateful for everything.

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I was lucky enough that I had the opportunity to work on papers with three of my five Netspar colleagues. I appreciate the time and effort that you devoted to helping me to develop ideas and publications. Bart Klijs, Claudine de Meijer and Ralph Stevens, your inputs were vital. Besides, I am grateful for the organization Netspar for making our research financially possible.

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… every decent tennis player has a beautiful wife and / or kids. I think I am the luckiest guy on earth that I have both. Babci, I just want to thank for everything we are. Without you, this would be worth nothing.

Den Haag, 22 April 2012