Determinants of Trends in Old-Age Mortality

Comparative Studies among Seven European Countries over the Period 1950 to 1999

Determinanten van sterfteontwikkelingen onder ouderen

Vergelijkende studies in zeven Europese landen
in de periode van 1950 tot 1999

Fanny Janssen

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CONTENTS

The chapters included in this PhD thesis are reprinted from the following publications and manuscripts:

Chapter 2
Janssen F, Kunst AE, for NEDCOM*. ICD coding changes and discontinuities in trends in cause-specific mortality in six European countries, 1950-99. *Bull World Health Organ 2004;82:904-13.*

Chapter 3
Janssen F, Nusselder WJ, Looman CWN, Mackenbach JP, Kunst AE, for NEDCOM*. Stagnation in mortality decline among elders in the Netherlands. *Gerontologist 2003;43:722-34.*

Chapter 4
Janssen F, Mackenbach JP, Kunst AE, for NEDCOM*. Trends in old-age mortality in seven European countries, 1950-1999. *J Clin Epidemiol 2004;57:203-16.*

Chapter 5
Janssen F, Kunst AE, for NEDCOM*. Cohort patterns in mortality trends among the elderly in seven European countries, 1950-99. *Int J Epidemiol 2005;doi:10.1093/ije/dy123.*

Chapter 6
Janssen F, Peeters A, Mackenbach JP, Kunst AE, for NEDCOM*. Relation between trends in late middle age mortality and trends in old age mortality - is there evidence for mortality selection? *J Epidemiol Community Health 2005;59:775-81.*

Chapter 7
Janssen F, Mackenbach JP, Kunst AE. Association between Gross Domestic Product throughout the life-course and old-age mortality across birth cohorts. Parallel analyses of seven European countries, 1950-1999. *Submitted.*

Chapter 8
Janssen F, Van der Heide A, Kunst AE, Mackenbach JP. End-of-life decisions and old-age mortality: a cross-country analysis. *Submitted.*

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1

INTRODUCTION

The twentieth century has witnessed an enormous increase in life expectancy. Whereas in the early 1900s, people from low-mortality countries could expect to live on average approximately 50 years from birth [1], in 2000, life expectancy at birth in record-holding country Japan rose to over 77 years among men, and almost 85 years among women [2].

The epidemiological transition that lies behind this increase in life expectancy is characterized by different phases [3]. The early increases in life expectancy, up to about 1930, were the result of "receding pandemics", i.e. huge declines in mortality from communicable diseases at younger ages [3,4]. These declines in mortality among younger ages gradually led to a shift in mortality towards older ages, with more weight to non-communicable, or "degenerative and man-made" diseases. As a result, the development in life expectancy became more and more determined by trends in mortality at older ages [5].

In low-mortality countries, mortality at older ages has generally declined since the 1950s [6-11], due to decreasing mortality rates from mainly cardiovascular diseases [12]. This decline in old-age mortality did not only contribute to the general increase in life expectancy; it was also partly responsible for the current increase in the number, the proportion and the mean age of elderly people in these populations, i.e. the ageing of these populations. The far-reaching consequences of population ageing for demands of health care services and old-age benefit systems are widely known.

Due to the increasing effect of old-age mortality trends on the current and future course of life expectancy, and due to its role in the ageing of populations [13-15], it is increasingly being recognized that old-age mortality should receive more attention in research. Next to emphasis on the age-trajectory of mortality among the elderly [16-19], a number of questions on possible future trends in life expectancy have gained interest. With respect to the latter, particularly the extent to which today's populations are approaching a limit to human life expectancy is extensively being debated upon. On the one hand, proponents of "the limited-lifespan paradigm" state that biological and practical constraints to reducing old-age mortality imply that future gains in life expectancy will occur at a slower pace, and that the limit to life expectancy is almost reached [20-22]. On the other hand, "the mortality-reduction paradigm" is adhered to by researchers who argue that life expectancy will continue to increase for some time to come [2,23,24]. They support their view by referring to mortality developments in sub-populations with extreme good health, to foreseen biomedical progress, and to historical observations that old-age mortality has been decreasing continuously.

In order to make inferences about possible future trends in old-age mortality, it is important to carefully study past trends and, even more importantly, to study the determinants of these past trends. The growing research focus on mortality among elderly populations, however, has until recently led to mostly descriptive studies on trends in old-age mortality [8-10,25-27]. Although these studies reported important cross-national

variations in the pace of mortality decline among the elderly, with even stagnation of the mortality decline in the 1980s in the Netherlands and Norway [9,28], little is known about the mechanisms behind the observed trends.

THIS THESIS

This thesis encompasses a detailed study of mortality trends among the elderly in relation to a number of possible determinants at population level.

Objective

Aim of the study is to carefully describe trends in old-age mortality in seven European low-mortality countries from 1950 to 1999, and to assess the role of specific factors in explaining the observed trends.

Approach

For this purpose, both trends in all-cause mortality and trends in specific causes of death are studied. Because many of the intermediate factors or risk factors that could determine mortality trends among the elderly are related to specific causes of death, studying the trends in cause-specific mortality can generate evidence on the determinants of the trends in all-cause mortality among the elderly.

In addition, we adopt a life-course perspective, with emphasis on determinants occurring not only in late life but also in earlier phases of the life-course [29,30]. For the present population study, adopting a life-course perspective implies the study of not only period patterns in old-age mortality, but also its cohort patterns. Whereas period patterns indicate immediate and modifiable effects of conditions occurring in late life, cohort patterns may reflect unmodifiable long-lasting effects of determinants located earlier in life. This distinction is of crucial importance for making projections of future developments in mortality. If cohort effects prove to be important, cohort-wise projections may be used to explore possible future mortality trends.

Comparative studies among seven selected countries are performed in order to reveal general patterns and possible country-specific deviations from these general patterns. We included seven low-mortality countries in northwestern Europe, i.e. Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden. For these countries, differences both in old-age mortality trends [8], and in the levels and trends of several potential determinants have been observed. In addition, these countries are among the few countries with the most accurate data on old-age mortality from 1950 onwards [9].

With regard to the determinants, we focus on the role of smoking, mortality selection, socio-economic developments throughout the life-course, and medical end-of-life decision-making. Smoking is known for its strong negative effect on survival. Moreover, the impact of smoking on mortality trends may vary considerably between countries [31], due to, for example, cross-national differences in the development of lifetime exposure to smoking across the birth cohorts [32]. Mortality selection implies that when mortality decreases at younger ages, the increasing proportion of the elderly population might be expected to be

less healthy when compared to survivors of earlier cohorts [15]. Subsequently this elderly population could experience relatively higher morbidity and mortality. For example, mortality declines in cardiovascular diseases, predominantly ischaemic heart diseases, have been shown to lead to increased prevalence of chronic heart diseases at older ages [33], with subsequently higher mortality risks of related diseases [34]. Socio-economic developments may be a key factor in influencing mortality levels and trends [35]. During the 20th century, all European countries experienced dramatic increases in both economic prosperity and life expectancy. The increasing prevalence of medical end-of-life decisions over time in the Netherlands [36], might have contributed to the relatively unfavourable mortality trends among Dutch elderly. Moreover, medical end-of-life decisions could possibly contribute to cross-national differences in old-age mortality.

Research questions

The main research questions addressed in this thesis are:

1. How did old-age mortality develop over the period 1950 to 1999 in seven European countries?

2. Which role do different possible determinants, such as smoking, mortality selection, socio-economic developments throughout the life-course, and medical end-of-life decision-making, have in explaining the observed trends?

Data and methods

To answer these research questions, aggregate data on total mortality, the underlying cause of death, and population at risk, by year of death (1950-1999), sex, and five-year age groups (60+/80+) were included for Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden. These data were obtained from national statistical offices, and related institutes, the Kannisto-Thatcher Database on Old Age Mortality (http://www.demogr.mpg.de/databases/ktdb) [9], and the Human Mortality Database (http://www.mortality.org). To study the role of socio-economic developments throughout the life-course, we reconstructed trends in Gross Domestic Product per capita and infant mortality from 1865 onwards, based on historical overviews [37-40]. In addition, we included recent data on physicians' practices and attitudes concerning medical end-of-life decisions from the EURELD consortium [41,42].

To these data, we applied different descriptive and analytical techniques. The old-age mortality trends were described by means of Poisson regression with linear splines. Spline functions divide the overall trend into a number of separate, adjacent segments [43], and enabled us to study trends within specific decades instead of broader periods, and to identify changes in the overall trend, such as a stagnation in mortality decline. To assess the role of period and cohort effects in old-age mortality trends we performed age-period-cohort analyses. The role of different possible determinants to the observed trends was studied by means of multivariate Poisson regression analyses and correlation analyses.

Structure of the thesis

The **first part** of the thesis, i.e. **Chapters 2 to 5**, deals with the description of trends in both all-cause and cause-specific old-age mortality, and the identification of the role of period and cohort effects in the observed trends. **Chapter 2** explains and illustrates our new method to identify and adjust for cause-specific mortality discontinuities due to coding changes both between and within the different revisions of the International Classification of Diseases. In **Chapter 3**, we focus on the description of all-cause and cause-specific mortality trends among those aged 80 and over in the Netherlands. More specifically, we assess whether the stagnation of old-age mortality decline observed in the Netherlands in the 1980s continued in the 1990s, and for which causes of death stagnation can be observed. In **Chapter 4**, we extend the description of period trends in all-cause and cause-specific old-age mortality to Denmark, England and Wales, Finland, France, the Netherlands, Norway and Sweden. **Chapter 5** reports on our identification and description of cohort patterns in the mortality trends among elderly (60+) in the same seven countries. In **Chapters 3 to 5**, we moreover discuss the role of smoking to the observed trends, by focussing on old-age mortality trends in smoking-related diseases.

In the **second part** of the thesis, **Chapters 6 to 8**, further explanations for the observed old-age mortality trends in the seven countries are explored by assessing the role of mortality selection, socio-economic developments throughout the life-course, and medical end-of-life decision-making. In **Chapter 6**, we test whether mortality selection is a dominant factor in determining trends in old-age mortality by empirically studying the possible existence of a negative correlation between trends in late middle-age mortality and trends in old-age mortality among the same cohorts. **Chapter 7** reports on our exploration of the role of socio-economic developments throughout the life-course. We assess whether old-age mortality levels for subsequent cohorts are associated with levels of Gross Domestic Product prevailing at different ages of the same cohorts. In **Chapter 8**, we examine the association between physicians' practices and attitudes concerning end-of-life decision-making and national mortality by age and cause of death across six European countries, i.e. Belgium, Denmark, Italy, the Netherlands, Sweden and Switzerland.

The final chapter, **Chapter 9**, summarizes the main results, and evaluates the usefulness and possible limitations of our methodological approach in obtaining these results. In addition, we will further elaborate on the determinants of the stagnation in mortality decline among the elderly in the Netherlands, and on possible future trends in old-age mortality. Lastly, recommendations for further research are proposed.

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PART I

DESCRIPTION

2

ICD CODING CHANGES AND DISCONTINUITIES IN TRENDS IN CAUSE-SPECIFIC MORTALITY IN SIX EUROPEAN COUNTRIES, 1950-1999

Objective To evaluate how often coding changes between and within revisions of the International Classification of Diseases (ICD) complicate the description of long-term trends in cause-specific mortality. **Methods** Data on cause-specific mortality between 1950 and 1999 for men and women aged 60 and older were obtained from Denmark, England and Wales, Finland, the Netherlands, Norway and Sweden. Data were obtained by five-year age groups. We constructed a concordance table using three-digit ICD codes. In addition we evaluated the occurrence of mortality discontinuities by visually inspecting cause-specific trends and by country-specific background information. Evaluation was also based on quantification of the discontinuities using a Poisson regression model (including period splines). We compared the observed trends in cause-specific mortality with the trends after adjustment for the discontinuities caused by changes to coding. **Results** In 45 out of 416 (10.8%) instances of ICD revisions to cause-specific mortality codes, significant discontinuities that were regarded as being due to ICD revisions remained. The revisions from ICD-6 and ICD-7 to ICD-8 and a wide range of causes of death, with the exception of the specific cancers, were especially affected. Incidental changes in coding rules were also important causes of discontinuities in trends in cause-specific mortality, especially in England and Wales, Finland and Sweden. Adjusting for these discontinuities can lead to significant changes in trends, although these primarily affect only limited periods of time. **Conclusion** Despite using a carefully constructed concordance table based on three-digit ICD codes, mortality discontinuities arising as a result of coding changes (both between and within revisions) can lead to substantial changes in long-term trends in cause-specific mortality. Coding changes should therefore be evaluated by researchers and, where necessary, controlled for.

BACKGROUND

The study of trends in cause-specific mortality is an important subject in epidemiology, demography and public health. However, assessment of long-term mortality trends in causes of death can be hampered by changes in coding. For example, numerous revisions of the International Classification of Diseases (ICD) have to be taken into account. Not only can these revisions lead to changes in coding rules but additionally each ICD classification can include a different number of items to code the causes of death, causing inconsistency over time. Solutions to overcome the bias that may result vary from simple and crude to elaborate and accurate.

A simpler approach is to study mortality trends only in broad, aggregated groups of causes of death or in specific diseases for which the coding is known to have been consistent over time [1]. However, many specific causes of death cannot be studied appropriately with these approaches.

Bridge coding is an accurate approach. In bridge coding cross-classifications between successive ICD revisions are used; these are based on the double coding of all causes of death [2]. These cross-classifications, however, exist for only a small number of countries and ICD revisions, and their results may not be directly applicable to all countries owing to differences in coding practices and differences in the application of new ICD revisions between countries [3,4].

Vallin & Meslé [4–6] elaborately and accurately reconstructed coherent series of data for causes of death in France for the years 1925–94, according to ICD-9. They used carefully constructed concordance tables based on four-digit codes; these concordance tables linked the codes for a specific cause of death through each successive ICD revision. Transition coefficients were calculated to redistribute the numbers of causes of death, based on cross-tabulation of the number of deaths for different revisions. Unfortunately, four-digit codes are not available in many countries. Furthermore, this method is quite time-consuming, and the redistribution of deaths in one country is not directly applicable to other countries.

An intermediate approach is to use concordance tables based on three-digit codes without redistributing the numbers of deaths [7]. These concordance tables often underlie the selection of the codes used for successive ICD revisions in studies on long-term trends in cause-specific mortality [8–10]. However, it is unclear whether mortality discontinuities caused by changes in ICD codes can be avoided using this intermediate approach.

Moreover, discontinuities in cause-specific trends can also result from incidental changes in coding rules, e.g. changes within ICD revisions in coding rules applied at statistical offices. The extent to which coding problems remain and may bias the study of trends in cause-specific mortality have not yet been estimated systematically. An assessment of the experience in several countries may aid researchers intending to study these long-term trends.

In this study, we evaluated how often coding problems complicate the description of long-term trends in mortality despite the use of a carefully constructed concordance table that is based on the three-digit codes. We used data from six European countries covering a broad selection of specific causes of death between 1950 and 1999. We considered the effect of mortality discontinuities caused by coding changes both between and within ICD revisions. We assessed in which situations (countries, causes of death) these coding changes led to discontinuities and whether adjustment for these discontinuities resulted in different estimates of trends in cause-specific mortality.

DATA AND METHODS

Data were obtained on the underlying cause of death, total mortality and mid-year population for Denmark, England and Wales, Finland, the Netherlands, Norway and Sweden. These data were stratified by year of death (1950–99), sex and five-year age group for people aged 60 and older. Data were obtained from the National Institute of Public Health (Denmark), the Office for National Statistics (England and Wales), Statfin (Finland), Statistics Netherlands, NIDI (Netherlands), Statistics Norway, and the National Board of Health and Welfare (Sweden). Data were available from 1951 for Finland and Norway, and from 1952 for Sweden. Data for Denmark were available from 1951 to 1998.

The death counts for each cause of death in the oldest age groups (85+ for England and Wales and the Netherlands up until 1969, and 90+, 95+, and 100+ for all other countries and periods) were redistributed into five-year age groups up to the age of 100+ years using the distribution observed for total mortality among these age groups. Data on mortality for the oldest age groups were available from the Kannisto-Thatcher Database on Old Age Mortality [11].

We selected 26 causes of death, mainly on the basis of their relative importance in old-age mortality (Table 1). For Finland, the total registry of causes of death by three-digit code was not available and instead data from aggregated code groups were supplemented with data from specific three-digit codes for the selected causes of death.

Five different revisions of the ICD occurred between 1950 and 1999 (Table 2), but because the codes remained identical in ICD-6 and ICD-7, only four revisions had to be bridged in order to reconstruct the causes of death. For this purpose, we carefully constructed a concordance table using three-digit codes, building on existing concordance tables [7,12,13] and using information from WHO [14–18] (Table 1). The basic rule we applied was that we aimed to safeguard the continuity of the medical content on the causes of death. For Denmark, a country-specific adjustment of the concordance table was made in order to include codes 260–265 for diabetes mellitus for the years 1965–68 (K. Juel, National Institute of Public Health, Denmark, personal communication about diabetes codes, June 2002).

The first step in our evaluation was to visually inspect trends in cause-specific mortality for the combined data on men and women aged 60 years and older. In our evaluation of mortality discontinuities we distinguished between those that were the result of revisions to the ICD, those that were the result of incidental changes in coding rules that applied to many causes of death simultaneously, and those that were the result of incidental changes in coding rules that were applied only to a specific cause of death, for example less restrictive coding for diabetes mellitus. In addition, country-specific background information was obtained through personal communication with national statistical offices or related institutes on the possible cause of observed discontinuities and on national coding practices and coding problems.

In the second step, mortality discontinuities resulting from ICD revisions and generally applied incidental changes in coding rules were quantified in cause-specific Poisson regression models and tested for statistical significance at the 95% significance level. The dependent variable was the number of deaths, with the mid-year population used as an offset variable. The independent variables were age (five-year age groups) and year of death (using splines). Spline functions divide the overall trend into a number of separate, adjacent segments [19] and allow a detailed description of long-term mortality trends to be made. In our analysis, we used five decade-specific segments (1950–59 to 1990–99). To these regression models we added transition variables indicating the ICD revisions (i.e. ICD-6 and ICD-7 to ICD-8, ICD-8 to ICD-9, or ICD-9 to ICD-10) or the general incidental changes in coding rules.

In the third step, we used two criteria to judge whether significant mortality discontinuities were due to coding problems.

Table 1. The concordance table used for bridging five revisions of the International Classification of Diseases (ICD)

Cause of death	ICD-6 and ICD-7	ICD-8	ICD-9	ICD-10
Infectious and parasitic diseases	001-138	000-136	001-139	A00-B99
Cancer of the oesophagus	150	150	150	C15
Cancer of the stomach	151	151	151	C16
Cancer of the colorectum	153-154	153-154	153-154	C18-C21
Cancer of the pancreas	157	157	157	C25
Cancer of the upper respiratory tract	140-148, 160, 161	140-149, 160, 161	140-149, 160, 161	C00-C14, C30-C32
Cancer of the lung	162-163	162	162	C33-C34
Cancer of the breast	170	174	174-175	C50
Cancer of the prostate	177	185	185	C61
Cancer of the bladder	181	188	188	C67
Cancer of the kidney	180	189	189	C64-C66, C68
Cancers, unspecified	198-199, 230-239	195-199, 230-239	195-199, 235-239	C76-C80, C97, D37-D48
Other cancers	Rest[a] (140-239, 294)	Rest (140-239)	Rest (140-239)	Rest (C00-D48)
Diabetes mellitus	260	250	250	E10-E14
Dementia and Alzheimer disease	304-306	290, 293	290, 331	F00, F01, F03, G30
Ischaemic heart disease	420	410-414	410-414	I20-I25
Other heart diseases	400-402, 410-416, 421-422, 430-434, 440-447	390-398, 400-404, 420-425, 427-429	390-398, 401-405, 416, 420-429	I00-I13, I15, I27, I30-I52
Cerebrovascular diseases	330-334	430-434, 436-438	430-434, 436-438	I60-I69
Other circulatory diseases	Rest (400-468)	Rest (390-458, excl 435 & 446)	Rest (390-459, excl 435 & 446)	Rest (I00-I99)
Pneumonia/influenza	480-483, 490-493	470-474, 480-483, 485-486	480-487	J10-J18
Chronic obstructive pulmonary disease	501, 502, 526, 527, 241	490-493, 518	490-494, 496	J40-J47
Senility	794	794	797	R54
Other symptoms and ill-defined conditions	Rest (780-795)	Rest (780-796)	Rest (780-799)	Rest (R00-R99)
Other diseases	Rest (001-795)	Rest (000-796)	Rest (001-799)	Rest (A00-R99)
Accidental fall	E900-904	E880-887	E880-888	W00-W19, X59
Other external causes	Rest (E800-999)	Rest (E800-999)	Rest (E800-999)	Rest (V01-Y98)

[a] Remainder of codes not used in this table.

Table 2. Revisions of the International Classification of Diseases (ICD) in 1950-99, by country

Country	ICD-6	ICD-7	ICD-8	ICD-9	ICD-10
Denmark	1951-1957	1958-1968	1969-1993	NA[a]	1994-1998
England and Wales	1950-1957	1958-1967	1968-1978	1979-1999	NA
Finland	1951-1957	1958-1968	1969-1986	1987-1995	1996-1999
France	1950-1957	1958-1967	1968-1978	1979-1999	NA
Netherlands	1950-1957	1958-1968	1969-1978	1979-1995	1996-1999
Norway	1951-1957	1958-1968	1969-1985	1986-1995	1996-1999
Sweden	1952-1957	1958-1968	1969-1986	1987-1996	1997-1999

[a] NA = not applicable.

The first criterion was to ensure that the significant effect could not be attributed to non-linear trends or to a single outlier, for instance an influenza epidemic nearby. The second criterion was to determine whether the observed effect could be related to the coding problem, for example as a direct result of a change at the level of four-digit codes or as an indirect result of an opposite effect on a complementary cause of death. These judgements were based primarily on visual inspection of cause-specific mortality trends without the use of statistical tests.

The identification of mortality discontinuities was based on the trends for men and women combined because changes in coding rules most likely operate in the same manner for both sexes, at least in regard to the direction of the effect. Outliers (i.e. single years of exceptional cause-specific mortality) were excluded from our analysis. For example, in the Netherlands in 1953 mortality from "external causes" increased as a result of a severe flood [20]. For a year to be classed as an outlier, the mortality rate had to be significantly different from mortality in the years within the decade, with rate ratios of the parameter estimates at least higher than 1.1 or lower than 0.9.

In order to evaluate whether mortality discontinuities regarded as being related to changes in coding biased the description of long-term trends in cause-specific mortality, we compared observed mortality trends with trends after adjustment. Adjustment involved including the transition variables, that were associated with mortality discontinuities related to coding changes, in the cause-specific and sex-specific regression models. We stratified this analysis by sex because cause-specific mortality trends are likely to be different for the two sexes and consequently the description of the trends may be biased differently for men and women when coding changes are ignored.

RESULTS

When a concordance table based on three-digit codes is applied to the data, in 191 out of 416 instances of ICD revisions to cause-specific mortality codes significant mortality discontinuities remained (Table 3). Of these significant mortality discontinuities, 24% were not the result of non-linear trends or single outliers, and they were consequently regarded as being due to the ICD revisions. Thus, in 45 out of 416 (10.8%) revisions to cause-specific mortality codes, significant mortality discontinuities that were regarded as being due to ICD revisions remained. These ICD-related mortality discontinuities affected 11 out of 26 causes of death, primarily "other heart diseases", ischaemic heart disease, unspecified cancers, "other symptoms", infectious diseases, dementia, and "other circulatory diseases".

Table 3. Mortality discontinuities, by country, cause of death, and revision of the International Classification of Diseases (ICD), for men and women aged 60 years and older, 1950-99[a]

Cause of death	ICD revision	Transition rate[b]					
		DK[c]	E&W	FIN	NL	NO	S
Infectious diseases	6/7 to 8	**1.56**	0.83	1.23	**1.74**	**1.74**	**1.32**
	8 to 9	-[d]	1.07	NS	NS	NS	NS
	9 to 10	NS	-[e]	1.20	NS	NS	1.12
Cancer of the oesophagus	6/7 to 8	NS	NS	NS	NS	NS	NS
	8 to 9	-	NS	NS	NS	NS	NS
	9 to 10	NS	-	NS	NS	NS	NS
Cancer of the stomach	6/7 to 8	NS	NS	NS	0.93	0.93	NS
	8 to 9	-	NS	NS	NS	NS	NS
	9 to 10	NS	-	NS	NS	NS	NS
Cancer of the colorectum	6/7 to 8	NS	1.05	NS	NS	0.92	1.07
	8 to 9	-	0.97	NS	NS	NS	NS
	9 to 10	NS	-	NS	NS	NS	NS
Cancer of the pancreas	6/7 to 8	NS	NS	NS	NS	NS	NS
	8 to 9	-	NS	NS	NS	NS	NS
	9 to 10	NS	-	NS	NS	NS	NS
Cancer of the upper respiratory tract	6/7 to 8	NS	1.07	NS	NS	NS	NS
	8 to 9	-	1.04	NS	NS	NS	NS
	9 to 10	NS	-	NS	NS	NS	NS
Cancer of the lung	6/7 to 8	NS	0.98	NS	0.96	NS	1.06
	8 to 9	-	NS	NS	0.97	NS	NS
	9 to 10	1.06	-	NS	NS	NS	NS
Cancer of the breast	6/7 to 8	NS	NS	NS	0.95	0.90	1.15
	8 to 9	-	NS	NS	NS	NS	NS
	9 to 10	NS	-	NS	NS	NS	NS
Cancer of the prostate	6/7 to 8	NS	NS	1.15	0.88	NS	1.13
	8 to 9	-	NS	NS	NS	NS	NS
	9 to 10	NS	-	NS	0.93	NS	1.05
Cancer of the bladder	6/7 to 8	NS	NS	NS	NS	NS	NS
	8 to 9	-	0.96	NS	NS	NS	NS
	9 to 10	NS	-	0.81	NS	NS	NS
Cancer of the kidney	6/7 to 8	1.15	NS	NS	0.90	NS	1.21
	8 to 9	-	NS	NS	NS	NS	NS
	9 to 10	NS	-	NS	NS	NS	NS
Cancers, unspecified	6/7 to 8	0.77	**1.07**	NS	**1.39**	**1.47**	**1.18**
	8 to 9	-	**1.24**	0.89	0.83	1.11	NS
	9 to 10	0.90	-	NS	1.05	0.88	1.06
Other cancers	6/7 to 8	NS	0.98	0.93	**0.85**	**0.89**	1.03
	8 to 9	-	0.99	NS	1.03	NS	1.07
	9 to 10	NS	-	NS	NS	1.08	Ns
Diabetes mellitus	6/7 to 8	NS	NS	NS	**0.67**	NS	0.90
	8 to 9	-	0.92	1.30	**0.82**	NS	NS
	9 to 10	0.84	-	NS	1.20	NS	NS

(Table 3, continued)

Cause of death	ICD revision	Transition rate[b]					
		DK[c]	E&W	FIN	NL	NO	S
Dementia and Alzheimer disease	6/7 to 8	0.96	0.92	0.64	0.78	NS	NS
	8 to 9	-	**1.92**	0.93	0.55	**1.91**	**1.48**
	9 to 10	1.05	-	**1.37**	0.73	NS	1.27
Ischaemic heart disease	6/7 to 8	0.87	**1.12**	**1.08**	**1.13**	**1.32**	**1.23**
	8 to 9	-	0.95	NS	**0.94**	NS	0.93
	9 to 10	0.97	-	0.94	NS	NS	0.97
Other heart diseases	6/7 to 8	0.73	**0.76**	**0.68**	**0.83**	**0.50**	**0.51**
	8 to 9	-	**1.18**	0.96	**1.20**	NS	**1.29**
	9 to 10	1.10	-	1.06	0.89	NS	1.07
Cerebrovascular diseases	6/7 to 8	0.91	1.03	0.88	NS	1.03	NS
	8 to 9	-	0.99	0.93	0.97	NS	NS
	9 to 10	0.95	-	NS	0.94	NS	1.05
Other circulatory diseases	6/7 to 8	1.11	1.12	1.14	**1.43**	1.09	0.95
	8 to 9	-	0.89	NS	**0.92**	**0.89**	NS
	9 to 10	0.92	-	**1.17**	NS	NS	NS
Pneumonia/influenza	6/7 to 8	0.94	1.19	1.53	**2.08**	1.14	NS
	8 to 9	-	0.95	NS	NS	NS	**0.84**
	9 to 10	0.77	-	NS	0.89	**0.62**	0.82
Chronic obstructive pulmonary disease	6/7 to 8	0.77	0.99	0.87	0.86	NS	0.83
	8 to 9	-	1.17	0.93	0.86	NS	NS
	9 to 10	NS	-	1.14	1.08	NS	NS
Senility	6/7 to 8	0.83	NS	0.76	1.06	0.84	NS
	8 to 9	-	0.82	0.58	0.69	NS	NS
	9 to 10	0.86	-	NS	0.93	0.76	NS
Other symptoms and ill-defined conditions	6/7 to 8	NS	**1.68**	NS	1.13	**0.90**	0.74
	8 to 9	-	**0.31**	0.51	**0.47**	1.10	NS
	9 to 10	NS	-	NS	1.15	0.70	**2.04**
Other diseases	6/7 to 8	0.80	NS	0.82	NS	0.89	NS
	8 to 9	-	NS	1.18	1.25	NS	1.15
	9 to 10	1.05	-	**0.83**	0.98	1.09	**0.92**
Accidental fall	6/7 to 8	0.84	1.13	NS	0.94	NS	1.41
	8 to 9	-	0.96	NS	NS	NS	1.16
	9 to 10	NS	-	1.15	NS	1.11	1.38
Other external causes	6/7 to 8	NS	0.91	NS	NS	NS	NS
	8 to 9	-	1.10	1.22	NS	NS	0.94
	9 to 10	NS	-	NS	NS	1.18	NS

[a] Values in bold indicate that the mortality discontinuity cannot be attributed to non-linear trends or a single outlier.
[b] The relative size of the increase or lowering of the fitted mortality rate for the period immediately after the coding change compared with the mortality rate immediately before.
[c] For Denmark the ICD revision 9 to 10 is actually from ICD-8 to ICD-10; ICD-9 was not used.
[d] - = not applicable.
[e] NS = parameter estimate is not significant.
DK = Denmark; E&W = England and Wales; FIN = Finland; NL = the Netherlands; NO = Norway; S = Sweden.

In contrast, the specific cancers that were included in our study did not show ICD-related mortality discontinuities. The proportion of ICD-related mortality discontinuities ranged from 1.9% in Denmark to 16.7% in the Netherlands. The revision from ICD-6 and ICD-7 to ICD-8 was the revision most prone to showing ICD-related mortality discontinuities (16.0%). The proportion of discontinuities for the revision from ICD-8 to ICD-9 was 10.8% and for ICD-9 to ICD-10 the proportion was 4.6%.

Two generally applied incidental changes in coding rules were identified. In England and Wales a broadening of coding rule 3 in the period 1984–92 caused the conditions directly leading to death being coded for less often. At the same time, conditions often mentioned in part II of the death certificate (conditions which contributed to the death but were not part of the direct causal sequence) were coded for more often as the underlying cause of death [21].

Table 4. Significant mortality discontinuities resulting from generally applied changes in coding rules in the International Classification of Diseases, 1950-99 by cause of death for men and women aged 60 years and older[a]

Cause of death	Transition rate[b,c]	
	England and Wales 1984 to 1992	Sweden from 1981 onwards
Infectious diseases	1.11	NS
Cancer of the oesophagus	NS[d]	NS
Cancer of the stomach	1.03	NS
Cancer of the colorectum	**1.03**	0.89
Cancer of the pancreas	**1.04**	NS
Cancer of the upper respiratory tract	NS	**0.84**
Cancer of the lung	1.02	0.93
Cancer of the breast	**1.05**	NS
Cancer of the prostate	NS	**0.77**
Cancer of the bladder	NS	**0.85**
Cancer of the kidney	1.04	**0.87**
Cancers, unspecified	**0.96**	1.07
Other cancers	**1.05**	**0.86**
Diabetes mellitus	**1.45**	**0.50**
Dementia and Alzheimer disease	**2.18**	1.57
Ischaemic heart disease	1.01	NS
Other heart diseases	**0.87**	**1.54**
Cerebrovascular diseases	**1.08**	0.96
Other circulatory diseases	**0.90**	1.27
Pneumonia/influenza	**0.52**	**1.28**
Chronic obstructive pulmonary disease	1.08	0.86
Senility	0.80	NS
Other symptoms and ill-defined conditions	NS	1.39
Other diseases	**1.23**	0.86
Accidental fall	1.03	**0.53**
Other external causes	1.07	0.91

[a] Table shows discontinuities at 95% significance.
[b] The relative size of the increase or lowering of the fitted mortality rate for the period immediately after or during the coding change compared with the mortality rate immediately before or adjacent to the coding change.
[c] Values in bold indicate that the mortality discontinuity cannot be attributed to non-linear trends or a single outlier.
[d] NS = parameter estimate is not significant.

In Sweden, from 1981 onwards some additional coding rules were implemented that had the reverse effect (A. Edberg, National Board of Health and Welfare, Sweden, personal communication, July 2002). These generally applied coding rules affected approximately half of the causes of death under consideration, including the specific cancers. For pneumonia and other heart diseases, both conditions leading directly to death, an important lowering of mortality rates in England and Wales in the period 1984–92 occurred, whereas in Sweden from 1981 onwards mortality associated with these conditions increased. For diabetes mellitus, a disease that is not part of the direct causal sequence, the reverse effects appeared (Table 4).

The remaining incidental changes in coding rules (n = 22) occurred primarily in Finland and Sweden (Table 5). Eleven of the 26 causes of death were affected, especially diabetes. In eight cases an additional mortality discontinuity occurred as a result of a one-year lag in applying a change in coding rules.

For cerebrovascular diseases, controlling for the incidental change in the coding rules in England and Wales (1984–92) and Finland (1956–58) led to significant changes in annual decade-specific mortality in England and Wales in the 1980s and 1990s, but a change in trend did not occur in Finland in the 1950s (Table 6). A change in trend also occurred for other causes of death subject only to incidental changes (i.e. most specific cancers, chronic obstructive pulmonary disease and accidental falls). The changes, however, were often insignificant, and the major changes were restricted to a limited period (data not shown).

For ischaemic heart disease, the revision from ICD-6 and ICD-7 to ICD-8 led to a significantly altered description of the mortality trend in all countries in the 1960s, and sometimes also affected the trends in the 1950s or the 1970s, or both. Controlling mainly for ICD revisions also led to significant changes in mortality trends for infectious diseases and unspecified cancers, although they were less pronounced (data not shown).

For diabetes mellitus, controlling for a large number of mortality discontinuities changed the mortality trends significantly, affecting the annual changes not only in size but also in direction in a number of decades, especially in the Netherlands and Sweden. Controlling for a combination of different coding problems also led to a significant change in almost the entire trend for dementia, other heart diseases and pneumonia (data not shown).

DISCUSSION

Despite using a concordance table based on three-digit ICD codes, in 10.8% of the revisions to codes for cause-specific mortality, significant discontinuities, regarded as being due to ICD revisions, remained. Especially affected were the revisions from ICD-6 and ICD-7 to ICD-8 and a wide range of causes of death, with the exception of the specific cancers. Incidental changes in coding rules were important causes of discontinuities in cause-specific mortality trends as well, especially in England and Wales, Finland and Sweden. Adjusting for these discontinuities can lead to significant changes in trends in cause-specific mortality, although these primarily affect only limited periods of time.

The range in differences between countries in terms of ICD-related mortality discontinuities (1.9% for Denmark to 16.7% for the Netherlands) could partly be due to an under-estimation of the percentage for Denmark caused by the lack of implementation of ICD-9.

Table 5. Cause-specific mortality discontinuities resulting from incidental changes in coding rules in the International Classification of Diseases, 1950-99, for men and women aged 60 years and older, by country

Country	Cause of death	Period	Cause of the irregularity	Transition rate [a]
Denmark	Diabetes mellitus	1965 onwards	Less restrictive practice in coding diabetes as the underlying cause of death from 1965 onwards[b]	2.30
Denmark	Other diseases	1965 onwards	Less restrictive practice in coding diabetes as the underlying cause of death from 1965 onwards[b]	0.95
England and Wales	Diabetes mellitus	1984	One year lag in change in coding rule 1984-92	0.90
Finland	Infectious diseases	1956-58	Change in coding rule	1.21
Finland	Dementia/Alzheimer	1956-58	Change in coding rule	0.00
Finland	Other heart diseases	1956-58	Change in coding rule	0.85
Finland	Cerebrovascular dis.	1956-58	Change in coding rule	1.14
Finland	Other circulatory dis.	1962 onwards	Change in coding rule	1.52
Finland	Other circulatory dis.	1962	One year lag in change in coding rule from 1962 onwards	0.87
Finland	COPD[c]	1956-58	Change in coding rule	1.43
Finland	Other diseases	1956-58	Change in coding rule	1.21
Netherlands	Diabetes mellitus	1984 onwards	More strict application of the ICD rule that codes diabetes as an underlying cause when circulatory diseases are intermediate causes [28]	2.70
Netherlands	Diabetes mellitus	1984	One year lag in change in coding rule from 1984 onwards	0.47
Netherlands	Diabetes mellitus	1985	Lag in change in coding rule from 1984 onwards	0.67
Norway	Diabetes mellitus	1997 onwards	From 1997 onwards Statistics Norway asked for additional information when diabetes mellitus was unspecified (i.e. type, complication, duration, and whether it should be the underlying cause of death). This led to more specific diagnoses and to diabetes being coded more often as the underlying cause of death[d]	1.43
Norway	Dementia/Alzheimer	1986	One year lag in the introduction of ICD-9	0.82
Sweden	Ischaemic heart dis.	1970 onwards	Change in coding rule	1.08
Sweden	Ischaemic heart dis.	1970	One year lag in change in coding rule from 1970 onwards	0.93
Sweden	Other heart diseases	1970 onwards	Change in coding rule	0.56
Sweden	Pneumonia/influenza	1970 onwards	Change in coding rule	0.38
Sweden	Pneumonia/influenza	1970	One year lag in change in coding rule from 1970 onwards	1.83
Sweden	Accidental fall	1981	One year lag in change in coding rule from 1970 onwards	1.29

[a] The relative size of the increase or lowering of the fitted mortality rate for the period immediately after or during the coding change compared with the mortality rate immediately before or adjacent to the coding change.
[b] Background information derived from Knud Juel (National Institute of Public Health, Denmark).
[c] COPD = chronic obstructive pulmonary disease.
[d] Background information derived from Anne Gro Pedersen (Statistics Norway).

Table 6. Decade-specific mortality trends for selected causes of death, 1950-99, before and after controlling for mortality discontinuities resulting from coding problems, by country and sex for people aged 60 years and older (each cause of death considered is typical of a specific type of coding change)

Disease and country	Trend	Annual changes (%)[a] Men 1950s	1960s	1970s	1980s	1990s	Women 1950s	1960s	1970s	1980s	1990s
Cerebrovascular diseases											
England and Wales	Observed	-0.13	-1.17	-2.92	**-2.30**	**-3.96**	-0.52	-1.29	-2.69	**-2.25**	**-3.43**
	Contr. for coding change 1984-92	-0.13	-1.16	-2.96	**-3.22**	**-2.54**	-0.52	-1.28	-2.73	**-3.05**	**-2.19**
Finland	Observed	0.25	-1.04	-4.10	-2.08	-2.77	-0.86	-2.21	-5.46	-1.35	-3.45
	Contr. for coding change 1956-58	0.18	-0.78	-4.16	-2.06	-2.78	-1.02	-1.73	-5.58	-1.32	-3.46
Ischaemic heart disease											
England and Wales	Observed	**3.70**	**2.89**	-0.09	-1.49	-3.83	**3.43**	**3.87**	**-0.58**	-1.04	-3.72
	Contr. for ICD revision 6/7 to 8	**3.95**	**2.10**	-0.17	-1.47	-3.84	**4.09**	**1.81**	**-0.77**	-0.99	-3.73
Finland	Observed	3.77	4.22	-0.50	-1.44	-3.40	3.74	**5.45**	-1.12	-0.49	-2.60
	Contr. for ICD revision 6/7 to 8	3.98	3.73	-0.61	-1.41	-3.41	4.37	**3.98**	-1.41	-0.42	-2.62
Nether-lands	Observed	5.96	**4.18**	-0.43	-2.68	-3.88	**4.45**	**3.40**	-1.56	**-3.08**	-3.25
	Contr. for ICD revi-sion 6/7 to 8, 8 to 9	6.39	**3.12**	-0.14	-2.44	-3.97	**5.37**	**1.06**	-1.36	**-2.73**	-3.38
Norway	Observed	**5.89**	**5.45**	**-0.49**	-0.71	-4.11	**2.29**	**6.52**	**-1.50**	-1.01	-3.32
	Contr. for ICD revision 6/7 to 8	**6.91**	**3.15**	**-0.97**	-0.58	-4.16	**4.20**	**1.91**	**-2.34**	-0.79	-3.40
Sweden	Observed	**2.77**	**6.40**	**0.81**	**-3.93**	-3.69	**-0.24**	**6.23**	**-0.83**	**-4.88**	-3.25
	Contr. for coding change since 1970, ICD revision 6/7 to8	**4.02**	**3.82**	**-0.02**	**-3.72**	-3.78	**1.39**	**2.72**	**-1.81**	**-4.63**	-3.35
Diabetes mellitus											
Denmark	Observed [b]	4.34	**6.17**	-1.36	3.07	0.85	0.59	**6.52**	-3.72	0.51	-0.48
	Contr. for coding change since 1965	4.50	**-3.09**	-0.19	2.74	1.11	0.58	**-4.40**	-2.31	0.10	-0.12
England and Wales	Observed	-0.84	3.72	-0.33	**5.34**	**-5.55**	-1.76	1.89	-2.82	**4.56**	**-6.55**
	Contr. for coding change 1984-92	-0.87	3.79	-0.55	**1.50**	**0.08**	-1.78	1.95	-3.02	**0.43**	**-0.47**
Nether-lands	Observed [c]	3.77	**-1.68**	**-4.52**	**10.2**	**-3.65**	2.84	**-3.47**	**-6.86**	**8.79**	**-5.14**
	Contr. for ICD revision 6/7 to 8, 8 to 9, and coding change since 1984	2.87	**0.93**	**-1.02**	**-2.19**	**-1.82**	1.84	**-0.52**	**-3.40**	**-3.12**	**-3.38**
Norway	Observed	0.69	-1.60	4.12	1.68	2.54	2.27	-4.40	1.84	0.72	**1.62**
	Contr. for coding change since 1996	0.67	-1.53	3.91	2.31	-0.63	2.24	-4.32	1.54	1.67	**-3.31**
Sweden	Observed [d]	4.28	**1.89**	**-2.58**	**1.57**	1.32	2.37	**0.92**	**-4.01**	**-1.11**	0.04
	Contr. for coding change since 1981	3.03	**4.38**	**0.96**	**3.67**	0.69	1.14	**3.45**	**-0.14**	**1.30**	-0.74

[a] Trend estimates in bold denote a significant difference between observed annual changes and annual changes after controlling for mortality discontinuities arising from coding problems.
[b] Data for 1995-1998 were excluded due to very erratic trend.
[c] Data for 1971 were excluded due to exceptionally high mortality rate.
[d] Data for 1970-1980 were excluded due to very erratic trend.

As for the revision of ICD-8 to ICD-9, in general fewer mortality discontinuities were observed. However, it is most likely that the differences between countries are related to differences in applying the ICD revisions.

Evaluation of data and methods

In this paper we developed a method to evaluate and adjust for mortality discontinuities caused by coding problems. A major advantage of our method when compared to the bridge coding method [2] is that our method can detect incidental changes in coding rules. In comparison to the method developed by Vallin & Meslé our method can be easily applied to other countries and can be extended to new ICD revisions fairly easily. Moreover, estimates of discontinuities observed using our method can easily be taken into account in trend analyses using regression or related techniques.

Inherent in our adjustment method is the assumption that the relative increase or decrease in mortality is equal in all years prior to or after the transition. For ischaemic heart disease, however, inclusion of code 422.1 in ICD-6 and ICD-7 for England and Wales and Norway, which was done to ensure better comparability with codes in ICD-8 [22], gave different results than our adjustment, primarily due to a change in the frequency of use of this code throughout ICD-6 and ICD-7. Although we could not explicitly check this assumption for other causes of death, note that the mortality discontinuity for ischaemic heart disease that appeared during the transition to ICD-8 is much more pronounced in absolute terms than the transitions for other causes of death.

The transition coefficients we found for people aged 60 and older cannot be applied directly to other age groups. A comparison of the transition rates for people aged ≥60 and ≥80 yielded changes greater than 10% in almost half of the cases (data not shown). Therefore, adjustment should be age-specific.

Persisting problems in studies of long-term mortality trends

The comparability of cause of death statistics over time is affected not only by ICD revisions or incidental changes in coding rules but also by changes in the reporting of the cause of death on death certificates and by changes in the number of deaths from ill-defined causes and other unspecified causes [1,4].

Changes in the reporting of causes of death on the death certificate may be the result of developments in medical science or the use of new diagnostic techniques as well as changes in concepts of diseases [1]. For example, a growing propensity for physicians to report dementia as an underlying cause of death may have contributed to the large annual increases in mortality from "dementia and Alzheimer disease" among elderly people aged 80 years and older in a number of north-western European countries in the 1980s and 1990s [23]. Part of the increase in mortality rates from chronic obstructive pulmonary disease may be the result of increased diagnosis and changes in how physicians code for the disease [24,25].

An increase in the number of deaths from "symptoms and ill-defined conditions" may be the result of both stricter diagnostic criteria for a given specific disease and a real increase

in unknown causes [26]. However, mortality trends in "symptoms and ill-defined conditions" are also often closely related to the quality and the accuracy of the diagnosis [1,26] (F. Meslé, unpublished data presented October 2000). For example, the increase in "symptoms and all ill-defined conditions" among people aged 80 years or older in Denmark, England and Wales, the Netherlands and Sweden from the 1980s onwards [23] may partly be the result of less detailed and less accurate diagnosis and reporting by physicians.

Both problems can bias mortality trends in other causes of death as well. For example, the increasing tendency to report "symptoms and ill-defined conditions" such as "sudden death" as the underlying cause of death may result in an underestimation of an increase (or overestimation of a decrease) in mortality from cardiovascular diseases [26]. Unfortunately, there are no formal methods that can be used to cope with changes in the reporting of causes of death on the death certificate because they result in gradual shifts that are difficult to detect and quantify [4]. The number of deaths attributed to ill-defined or unknown causes are increasingly being redistributed by researchers among specific causes [5,27]. This applies especially to heart disease; it is less common for cancer-related deaths (F. Meslé, unpublished data presented October 2000). However, it remains unclear whether these reclassifications, which are based on assumptions about, among others, the distribution of ill-defined or unknown causes among specific causes of death, actually lead to improvements in assessing long-term trends in cause-specific mortality. Aggregating ill-defined causes of death with specific causes probably will improve the comparability of data over time but information on specific causes of death will be lost, and this may be an unacceptable loss in many cases.

Conclusions

When describing long-term trends in cause-specific mortality, evaluating and adjusting for mortality discontinuities caused by coding changes between and within ICD revisions is essential because these changes can substantially bias the description and analysis of cause-specific trends in mortality. An accurate and fairly easy method for evaluating and adjusting for these coding-related mortality discontinuities has been proposed in this paper — that is, a combination of visual inspection of the trends and the use of a formal regression method after application of a concordance table based on three-digit ICD codes. The other problems that remain when studying long-term trends in cause-specific mortality, such as changes in the reporting of causes of death by physicians, should always be taken into account when interpreting these trends.

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3

STAGNATION IN MORTALITY DECLINE AMONG ELDERS IN THE NETHERLANDS

Objective This study assesses whether the stagnation of old-age (80+) mortality decline observed in The Netherlands in the 1980s continued in the 1990s and determines which factors contributed to this stagnation. Emphasis is on the role of smoking. **Methods** Poisson regression analysis with linear splines was applied to total and cause-specific mortality data by age, year of death (1950-1999) and sex. An age-period-cohort analysis was carried out to determine whether the trends followed period or cohort patterns. ICD revisions were bridged by use of a concordance table. **Results** A sudden reversal in old-age mortality decline occurred around 1980, leading to a stagnation of the decline and even increases in mortality thereafter. Smoking-related cancers, chronic obstructive pulmonary disease, and diseases specifically related to old age contributed to this stagnation. Trends in smoking-related cancers and chronic obstructive pulmonary disease showed a cohort pattern - especially for men. When these smoking-related diseases were excluded, the trends in old-age mortality in The Netherlands showed an increasing stagnation for both sexes. **Conclusion** Smoking behavior can only partly explain the stagnation of mortality. Other factors, such as increased frailty and changes in medical and social services for elderly people, probably played a more decisive role in the recent stagnation.

BACKGROUND

In almost all low-mortality countries, mortality among the elderly population has shown a decrease since 1950, with an accelerated improvement from 1970 onward [1-4]. This trend has contributed to the current increase in both the proportion and the mean age of elderly people in these populations. Furthermore, the ongoing reduction in old-age mortality raises interesting questions concerning the extent to which today's populations are approaching a limit to human life expectancy [5-14].

However, the general pattern of sustained mortality decline among the elderly population since 1970 was not observed in all low-mortality countries. In The Netherlands and Norway, old-age mortality even showed signs of an increase [3,15,16]. This raises the question of whether The Netherlands and Norway can be regarded as precursors of trends in old-age mortality, implying that the same stagnation will happen in other low-mortality countries in the future, or that the stagnation as observed in The Netherlands and Norway is unique and is not likely to occur elsewhere. This led us to study the determinants underlying the stagnation of mortality decline. More specifically, are these unfavorable mortality trends the result of determinants specifically operating in The Netherlands and Norway, or are they the result of factors that may be expected to influence trends in other countries as well?

Here we focus on trends in old-age mortality in The Netherlands. A stagnation of the decrease in mortality in The Netherlands in the 1980s was described by Nusselder and

Mackenbach (2000) [15]. They found that life expectancy at the age of 85 had decreased for men and had stagnated for women. One of the possible reasons suggested relates to smoking histories. In The Netherlands, smoking rates among men were exceptionally high during the 20th century [17]. In particular men born between 1897 to 1917 had a high lifetime exposure to smoking [18], and these are the men who reached old age at the end of the 20^{th} century.

Here we describe in more detail the mortality trends among Dutch elderly people in the second half of the twentieth century. We will (a) determine whether the stagnation of old-age mortality has persisted in more recent years and (b) explore possible explanations for recent trends in old-age mortality. The contribution of smoking will be emphasized. Because smoking behavior in The Netherlands has been shown to vary markedly between birth cohorts, we assess whether mortality trends are dominated by cohort patterns.

In this analysis we go one step farther than most prior analyses of old-age mortality by analyzing both period and cohort patterns in total mortality as well as in an extensive range of causes of death. We cover trends in the period 1950-1999. Furthermore, in our analysis we made an effort to carefully bridge the different revisions of the International Classification of Diseases (ICD).

DATA AND METHODS

Data

In this study, total mortality and population data by single year of age (up to 112), sex, and year of death (1950-1999) have been included for the total Dutch population. In addition, for the causes of death, data were available by three digit codes, 5-year age groups (up to 85+ for 1950-1969, thereafter up to 95+), sex, and year of death (1950-1999). All data were originally obtained from Statistics Netherlands [19-21]. The deaths per cause in the older age groups (85+ or 95+) were redistributed over 5-year age groups up to 100+ on the basis of the distribution of total mortality among these age groups.

We distinguished 26 (groups of) specific and homogeneous causes of death, which were selected predominantly on the basis of their relative importance in old-age mortality. In addition we selected (a) cancers that are strongly (population attributable risk [PAR] larger than 0.50) or moderately (PAR between 0.25 and 0.50) related to smoking (PARs from the American Cancer Study) [22], and (b) causes that could indicate changes in coding practices. Table 1 lists the causes of death, their accompanying ICD10 codes, and their relative share in all-cause mortality among those aged 80 and older in 1950 and 1999.

Statistical analysis

We analyzed the data by means of a (log-linear) Poisson regression model. The dependent variable was the number of deaths, with the person-years at risk as offset. As independent variables, we used age (5-year age groups) and year (year of death in the period analysis or birth year in the cohort analysis).

The use of 5-year age groups in all of our analyses, except the one on which Figure 1 is based, was due to the restriction that data on the causes of death were available by 5-year

age groups instead of single years of age. To evaluate to what extent a possible change over time in the distribution of deaths within a 5-year age group could affect our results, we compared the results for total mortality by using data by 5 years of age with the results from data by single years of age.

Table 1. Causes of death, accompanying ICD10 codes, and their relative share in all-cause mortality in 1950 and 1999, for people aged 80 and older, by sex

Cause of death	ICD10 (1996-1999)	80+ in 1950 (%)		80+ in 1999 (%)	
		Men	Women	Men	Women
Strongly smoking-related cancers					
Esophagus	C15	0.68	0.40	0.55	0.27
Upper respiratory / digestive system	C00-C14,C30-C32	0.46	0.10	0.34	0.13
Lung	C33-34	0.45	0.15	5.18	0.73
Moderately smoking-related cancers					
Pancreas	C25	0.12	0.09	0.60	0.81
Bladder	C67	0.28	0.23	1.25	0.44
Kidney	C64-C66,C68	0.05	0.05	0.53	0.27
Other cancers[a]	Rest(C00-D48)	1.63	2.83	3.08	3.75
Other specific cancers					
Stomach	C16	4.40	3.44	1.11	0.73
Colorectum	C18-C21	1.86	2.00	2.39	2.28
Breast	C50	0.01	1.00	0.05	2.31
Prostate	C61	2.15	0.00	4.75	0.00
Unspecified	C76-C80,C97,D37-D48	0.57	0.59	2.09	2.14
Cardiovascular diseases					
IHD	I20-I25	11.29	9.71	13.45	10.98
Other heart diseases	I00-I13,I15,I27,I30-I52	18.68	20.77	11.53	14.33
Cerebrovascular diseases	I60-I69	14.40	16.91	9.51	12.55
Other circulatory diseases	Rest(I00-I99)	6.04	5.45	3.56	2.63
Other causes of death					
Infectious diseases	A00-B99	0.64	0.74	1.01	1.05
Pneumonia/influenza	J10-J18	5.71	6.11	7.70	7.97
COPD	J40-J47	2.79	2.26	8.39	3.23
Accidental fall	W00-W19,X59	1.24	1.67	1.40	2.27
Other external causes	Rest(V01-Y98)	0.96	0.42	0.63	0.30
Diseases strongly sensitive to coding practices					
Diabetes mellitus	E10-E14	0.70	1.47	1.63	2.81
Dementia and Alzheimer's	F00,F01,F03,G30	3.37	3.67	3.97	8.35
Senility	R54	9.55	11.81	1.87	3.48
Other symptoms & ill-defined Conditions	Rest(R00-R99)	1.99	1.78	2.83	2.84
Other diseases	Rest(A00-R99)	9.96	6.34	10.59	13.33
All causes 80+ (n)		7,532	8,665	22,791	40,214

ICD = International Classification of Diseases; IHD = ischaemic heart disease; COPD= chronic obstructive pulmonary disease.

[a] This excludes all the cancers listed in this table.

Because this comparison generated virtually the same results, we expect that, although age patterns can differ for the specific causes of death, the bias when the data by 5-year age groups are used will be minimal.

For the period analysis, linear splines were used in the regression model to describe mortality trends by year of death. Spline functions accommodate piecewise fits connected with one another at the transition from one segment to the next [23]. For the period analysis, we used five segments, each covering a period of 10 years (1950-1959, 1960-1969, 1970-1979, 1980-1989, 1990-1999). The analysis including splines yielded estimates of annual changes in mortality within each ten-year period. Comparison of these five decade-specific rates of change enabled us to detect and quantify changes in the secular trend in mortality, such as a stagnation of the decrease in mortality.

For the cohort analysis, the year of birth was used as independent variable. For this purpose we determined the mean birth year for every combination of a single calendar year and a 5-year age group, taking into account the distribution of the population within this age group. To obtain equal degrees of freedom compared with the age-period model, we divided the range of birth years into five segments as well (1848-1879, 1880-1889, 1890-1899, 1900-1909, 1910-1936). These five cohort segments were chosen in such a way that approximately the same number of deaths (for those aged 80 and older) occurred in each segment.

In a last step, we fitted a regression model including age, year of death, and birth year, that is a full age-period-cohort (APC) model. This model was based on the same period and cohort splines as just described. By comparing the scaled deviances (a measure of unexplained variance) of the different models (age, age-period, age-cohort, age-period-cohort), we evaluated the extent to which the secular trends followed a cohort or period pattern. It should be noted that our analysis differs from traditional APC analyses, because we evaluate the effects of year of birth by means of splines instead of a nominal variable for 5- or 10-year periods.

The use of splines helped us to overcome the identification problem or drift in APC analysis. Drift has been described as a common linear trend that cannot be ascribed to either period or cohort influences [24]. Because the splines enabled us to identify nonlinear trends we avoided the identification problem, especially for causes of death showing these nonlinear trends.

We focused in most of these analyses on deaths among people aged 80 and older. We made an exception when identifying underlying cohort patterns (in APC analyses) and analyzing these cohort patterns (in cohort analyses). Because it would not be possible to separate period and cohort effects in an analysis of those 80 years and older, we decided to use the data on deaths among people aged 60 and older.

Concordance

In analyzing the trends for the selected causes of death, we had to bridge four different ICD revisions: ICD6/7 (1950-1968), ICD8 (1969-1978), ICD9 (1979-1995) and ICD10 (1996-1999). To do this, we built on the work of Wolleswinkel-van den Bosch to construct a concordance table in which the different codes for a specific cause of death in successive

ICD revisions are linked [25]. Available information from the World Health Organisation, for example the ICD9 to ICD10 translator [26], and Statistics Netherlands [27] was used, among others, to extend the existing concordance tables to ICD10. The basic rule applied was that continuity of the medical content of the causes of death should be safeguarded.

Using a prefinal version of the resulting concordance table we looked at the trends in mortality to check whether sudden changes in mortality at ICD transitions still occurred. Whenever irregularities were found, we checked whether the concordance table could be improved. The final concordance table is presented in Appendix I. We made special efforts to accurately bridge the change from ICD6/7 to ICD8 for ischaemic heart disease (IHD) by using published data on the four-digit code for IHD under ICD6/7 [28].

After application of the final concordance table, irregularities in the trends caused by ICD transitions persisted for a number of causes of death. We judged these irregularities in our regression model (age-period) by adding variables indicating the three ICD transitions, that is ICD6/7 to 8, ICD8 to 9, or ICD9 to 10. For nine causes of death, these variables had to be included in the model for one or two transitions. We selected these cases on the basis of the following criteria: (a) the parameter estimate corresponding to this variable had to be statistically significant; (b) the significant effect could not be attributed to nonlinear trends or to a single outlier, for instance an influenza epidemic; and (c) the observed effect could be explained, for example, by a four-digit code not included in the concordance table or an effect on one cause of death mirrored by an opposite effect on a complementary cause of death.

Another problem in analyzing trends by cause of death related to changes within ICD revisions, such as changes in the reporting of causes of death by physicians or in coding rules applied at Statistics Netherlands. This again might have caused irregularities in mortality trends [29]. For diabetes mellitus, we had information on the presence of incidental changes in coding rules, that had a demonstrable effect on trends in mortality from this cause in the 1980s [30]. To control for it, we included an extra variable in the regression model for this specific cause of death.

For three causes of death, one single year exhibited exceptional mortality levels. For example, in 1953, "external causes" showed an enormous increase in mortality because of a flood disaster. In 1980, a strike of medical specialists resulted in high mortality rates for "other symptoms and ill-defined conditions" [31]. Another outlier occurred in 1971 for diabetes mellitus. These outliers were excluded from the cause-specific analyses.

RESULTS

Total mortality rates for those aged 80 and older (Figure 1) showed a general tendency of a moderate decline in the period 1950-1969 (except for the highest age group), and a steep decline between 1970 and 1979. Around 1980, a sudden reversal of the trend led to stagnation of the decline and even increases in mortality, especially among the highest age groups and for men in the 1980s.

In the first row of Tables 2a and 2b, the pace of mortality decline within each of the five decades is quantified by means of annual rates of change. Male old-age mortality

decreased from 1950 to 1979 by annual percentage changes of –0.57, -0.31, and –0.89 for the successive decades. In the 1980s, however, mortality increased by 0.37% per year. The annual percentage change of –0.18 for 1990-1999 suggests a modest reemergence of the decrease in old-age mortality. The confidence intervals for successive decades do not overlap (except for 1950-1959 to 1960-1969), implying that the changes in the pace of mortality decline are statistically significant.

Among women, mortality showed generally the same secular trends, but with important differences. The annual percentage changes indicate a pronounced decrease in the 1970s (-2.67). This decline was followed by a levelling off of the mortality decline between 1980 and 1989 (-0.42) and by stagnation in the 1990s (+0.06).

Table 2 moreover shows widely different trends for the different causes of death for both sexes. A number of causes of death exhibited unfavorable trends. First, some causes of death underwent a stagnation of an initial mortality decline. For men (Table 2a), this applies for cancer of the esophagus, "other heart diseases", infectious diseases, pneumonia, dementia, and senility.

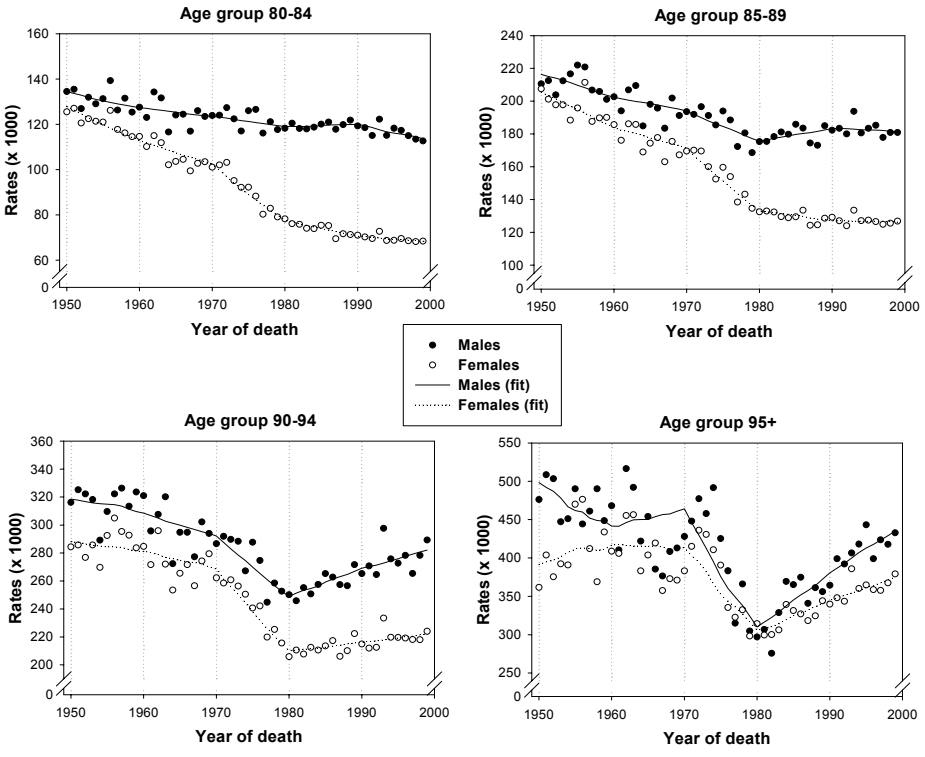

Figure 1. Observed and fitted all-cause mortality rates, for people aged 80 and older, 1950-1999, by sex and 5-year age groups.

Table 2a. Annual changes (%) by period for total and cause-specific mortality: men aged 80 and older

Cause of death	1950-59		1960-69		1970-79		1980-89		1990-99	
	%	95% CI	%	95% CI	%	95% CI	%	95% CI	%	95% CI
Total mortality	-0.57	(-0.72 ; -0.41)	-0.31	(-0.42 ; -0.19)	-0.89	(-0.99 ; -0.79)	0.37	(0.28 ; 0.47)	-0.18	(-0.30 ; -0.06)
Strongly smoking-related cancers										
Esophagus	-4.28	(-6.32 ; -2.19)	-1.15	(-2.86 ; 0.59)	-1.70	(-3.29 ; -0.09)	1.37	(-0.18 ; 2.94)	2.43	(0.60 ; 4.29)
Upper resp. / digest. system	-1.97	(-4.42 ; 0.54)	0.99	(-0.87 ; 2.89)	-0.94	(-2.54 ; 0.69)	-0.75	(-2.32 ; 0.84)	-1.70	(-3.74 ; 0.38)
Lung	6.96	(5.11 ; 8.86)	8.19	(7.21 ; 9.18)	7.55	(6.95 ; 8.16)	1.85	(1.42 ; 2.29)	-1.97	(-2.47 ; -1.47)
Moderately smoking-related cancers										
Pancreas	10.05	(6.44 ; 13.79)	4.84	(3.04 ; 6.67)	2.40	(1.12 ; 3.69)	0.05	(-1.06 ; 1.16)	-2.05	(-3.44 ; -0.64)
Bladder	4.94	(2.63 ; 7.29)	2.75	(1.38 ; 4.14)	1.66	(0.61 ; 2.71)	1.78	(0.87 ; 2.69)	-1.73	(-2.82 ; -0.63)
Kidney	10.93	(4.50 ; 17.75)	3.41	(0.29 ; 6.64)	5.70	(3.46 ; 7.98)	3.94	(2.27 ; 5.63)	1.01	(-0.78 ; 2.82)
Other cancers[a]	1.51	(0.27 ; 2.76)	1.77	(0.48 ; 3.08)	2.36	(1.59 ; 3.13)	1.71	(1.10 ; 2.32)	0.24	(-0.47 ; 0.95)
Other specific cancers										
Stomach	-1.63	(-2.42 ; -0.83)	-1.98	(-2.60 ; -1.35)	-3.36	(-3.97 ; -2.75)	-2.68	(-3.35 ; -2.01)	-5.71	(-6.69 ; -4.73)
Colorectum	0.95	(-0.18 ; 2.08)	0.81	(0.04 ; 1.59)	0.78	(0.14 ; 1.44)	-0.54	(-1.13 ; 0.07)	-0.90	(-1.67 ; -0.13)
Prostate	3.22	(2.19 ; 4.25)	1.57	(0.92 ; 2.22)	0.14	(-0.39 ; 0.67)	2.16	(1.68 ; 2.64)	0.78	(0.23 ; 1.34)
Unspecified	8.75	(6.37 ; 11.20)	1.81	(-0.17 ; 3.83)	-2.36	(-3.23 ; -1.48)	2.35	(1.56 ; 3.15)	2.78	(1.88 ; 3.69)
Cardiovascular diseases										
IHD	3.85	(3.41 ; 4.29)	1.32	(1.03 ; 1.60)	-0.32	(-0.68 ; 0.05)	-1.41	(-1.65 ; -1.16)	-2.41	(-2.71 ; -2.10)
Other heart diseases	-1.57	(-1.96 ; -1.19)	-3.46	(-3.77 ; -3.15)	-0.63	(-1.09 ; -0.16)	-2.17	(-2.46 ; -1.88)	0.68	(0.32 ; 1.04)
Cerebrovascular diseases	0.81	(0.41 ; 1.21)	-1.86	(-2.14 ; -1.57)	-2.78	(-3.06 ; -2.51)	-0.99	(-1.27 ; -0.70)	-1.76	(-2.13 ; -1.38)
Other circulatory diseases	-1.12	(-1.76 ; -0.47)	-3.98	(-4.73 ; -3.22)	-5.78	(-6.54 ; -5.01)	2.96	(2.39 ; 3.52)	-2.83	(-3.44 ; -2.21)
Other causes of death										
Infectious diseases	-5.34	(-7.57 ; -3.05)	-2.33	(-5.28 ; 0.73)	-2.92	(-4.45 ; -1.36)	2.97	(1.57 ; 4.39)	6.32	(4.80 ; 7.87)
Pneumonia/influenza	-2.77	(-3.43 ; -2.11)	-5.41	(-6.23 ; -4.59)	-3.27	(-3.72 ; -2.82)	0.07	(-0.35 ; 0.49)	5.28	(4.77 ; 5.79)
COPD	0.03	(-0.94 ; 1.00)	4.11	(3.45 ; 4.77)	2.40	(1.92 ; 2.88)	4.86	(4.47 ; 5.26)	-0.69	(-1.11 ; -0.27)
Accidental fall	4.19	(2.85 ; 5.55)	2.85	(2.03 ; 3.68)	-4.77	(-5.43 ; -4.11)	-1.17	(-1.88 ; -0.44)	-2.04	(-3.00 ; -1.06)
Other external causes	3.40	(1.75 ; 5.08)	-0.78	(-1.82 ; 0.27)	-2.27	(-3.22 ; -1.32)	-1.19	(-2.17 ; -0.20)	-3.60	(-4.92 ; -2.25)
Diseases strongly sensitive to coding practices										
Diabetes mellitus	4.62	(2.94 ; 6.33)	-0.18	(-1.79 ; 1.45)	-0.99	(-2.78 ; 0.83)	0.21	(-2.09 ; 2.56)	-2.38	(-3.34 ; -1.41)
Dementia and Alzheimer's	-10.77	(-11.89 ; -9.63)	4.59	(3.46 ; 5.74)	-14.48	(-15.47 ; -13.47)	14.14	(12.90 ; 15.40)	15.63	(14.67 ; 16.59)
Senility	-8.97	(-9.56 ; -8.38)	-7.72	(-8.33 ; -7.11)	-4.21	(-4.91 ; -3.51)	-0.35	(-1.10 ; 0.40)	2.64	(1.69 ; 3.59)
Other sympt. & ill-def. cond.	-2.84	(-3.95 ; -1.72)	2.25	(1.37 ; 3.13)	4.86	(3.80 ; 5.93)	2.71	(1.81 ; 3.62)	4.03	(3.19 ; 4.89)
Other diseases	-0.60	(-1.11 ; -0.08)	-1.22	(-1.59 ; -0.84)	0.51	(0.18 ; 0.85)	1.79	(1.49 ; 2.10)	-1.92	(-2.29 ; -1.55)

Notes: CI = confidence interval; IHD = ischaemic heart disease; COPD= chronic obstructive pulmonary disease. [a] See "other cancers" as listed in Table 1.

Table 2b. Annual changes (%) by period for total and cause-specific mortality: women aged 80 and older

Cause of death	1950-59		1960-69		1970-79		1980-89		1990-99	
	%	95% CI	%	95% CI	%	Cause of death	%	95% CI	%	95% CI
Total mortality	-1.08	(-1.23 ; -0.94)	-0.81	(-0.92 ; -0.70)	-2.67	(-2.76 ; -2.58)	-0.42	(-0.50 ; -0.33)	0.06	(-0.04 ; 0.15)
Strongly smoking-related cancers										
Esophagus	-6.50	(-8.95 ; -3.98)	-1.44	(-3.57 ; 0.73)	-0.81	(-2.62 ; 1.04)	1.75	(0.21 ; 3.31)	-0.40	(-2.09 ; 1.32)
Upper resp. / digest. system	0.17	(-3.84 ; 4.35)	-2.46	(-5.29 ; 0.46)	0.76	(-1.67 ; 3.26)	-2.03	(-4.07 ; 0.05)	-0.44	(-2.95 ; 2.13)
Lung	5.39	(1.92 ; 8.98)	-0.47	(-2.48 ; 1.58)	4.92	(3.38 ; 6.49)	0.87	(-0.22 ; 1.96)	2.63	(1.45 ; 3.83)
Moderately smoking-related cancers										
Pancreas	10.88	(7.15 ; 14.75)	4.47	(2.70 ; 6.27)	1.64	(0.48 ; 2.81)	0.94	(0.04 ; 1.85)	-1.25	(-2.27 ; -0.22)
Bladder	2.05	(-0.81 ; 4.99)	0.61	(-1.25 ; 2.50)	-1.10	(-2.55 ; 0.36)	1.23	(-0.01 ; 2.48)	-2.16	(-3.56 ; -0.73)
Kidney	6.00	(-0.09 ; 12.46)	4.69	(1.34 ; 8.15)	4.32	(2.15 ; 6.53)	1.63	(0.10 ; 3.19)	-0.96	(-2.65 ; 0.75)
Other cancers[a]	0.36	(-0.55 ; 1.27)	0.52	(-0.46 ; 1.50)	0.47	(-0.07 ; 1.01)	0.60	(0.19 ; 1.01)	-0.63	(-1.10 ; -0.17)
Other specific cancers										
Stomach	-2.80	(-3.62 ; -1.97)	-2.44	(-3.09 ; -1.79)	-6.03	(-6.64 ; -5.42)	-3.48	(-4.14 ; -2.82)	-5.36	(-6.27 ; -4.44)
Colorectum	0.09	(-0.94 ; 1.14)	0.85	(0.14 ; 1.56)	-1.02	(-1.57 ; -0.46)	-1.01	(-1.49 ; -0.52)	-1.93	(-2.51 ; -1.34)
Breast	3.71	(2.31 ; 5.12)	1.24	(0.39 ; 2.09)	-0.46	(-1.10 ; 0.18)	0.57	(0.04 ; 1.11)	-0.35	(-0.96 ; 0.26)
Unspecified	7.53	(5.66 ; 9.43)	1.95	(0.38 ; 3.56)	-5.15	(-5.81 ; -4.48)	-0.01	(-0.61 ; 0.59)	2.23	(1.55 ; 2.92)
Cardiovascular diseases										
IHD	3.19	(2.76 ; 3.62)	0.32	(0.04 ; 0.60)	-0.95	(-1.29 ; -0.61)	-2.18	(-2.39 ; -1.96)	-3.02	(-3.28 ; -2.77)
Other heart diseases	-1.63	(-1.96 ; -1.29)	-3.57	(-3.83 ; -3.30)	-1.80	(-2.17 ; -1.43)	-2.04	(-2.25 ; -1.82)	-0.41	(-0.66 ; -0.17)
Cerebrovascular diseases	0.53	(0.18 ; 0.88)	-2.35	(-2.59 ; -2.10)	-3.62	(-3.84 ; -3.41)	-0.90	(-1.11 ; -0.70)	-1.70	(-1.95 ; -1.45)
Other circulatory diseases	-0.21	(-0.84 ; 0.42)	-4.25	(-4.95 ; -3.55)	-8.37	(-9.01 ; -7.72)	0.18	(-0.28 ; 0.65)	-4.56	(-5.07 ; -4.04)
Other causes of death										
Infectious diseases	-7.45	(-9.72 ; -5.12)	-3.34	(-6.51 ; -0.08)	-7.12	(-8.35 ; -5.88)	4.81	(3.68 ; 5.96)	5.31	(4.22 ; 6.41)
Pneumonia/influenza	-4.23	(-4.85 ; -3.61)	-4.85	(-5.63 ; -4.05)	-3.91	(-4.31 ; -3.50)	-0.89	(-1.23 ; -0.54)	4.69	(4.30 ; 5.08)
COPD	-2.43	(-3.56 ; -1.28)	0.18	(-0.67 ; 1.04)	-2.21	(-2.89 ; -1.54)	4.61	(4.04 ; 5.18)	1.38	(0.82 ; 1.95)
Accidental fall	5.01	(4.00 ; 6.02)	4.35	(3.77 ; 4.93)	-7.01	(-7.43 ; -6.58)	-4.74	(-5.18 ; -4.29)	-1.62	(-2.24 ; -1.01)
Other external causes	0.91	(-1.56 ; 3.44)	-0.60	(-2.23 ; 1.06)	-2.90	(-4.24 ; -1.53)	2.41	(1.17 ; 3.66)	-5.12	(-6.52 ; -3.69)
Diseases strongly sensitive to coding practices										
Diabetes mellitus	2.32	(1.22 ; 3.43)	-0.65	(-1.77 ; 0.47)	-2.87	(-4.09 ; -1.64)	-1.39	(-2.77 ; 0.00)	-2.79	(-3.36 ; -2.21)
Dementia and Alzheimer's	-10.08	(-11.04 ; -9.12)	5.07	(4.16 ; 6.00)	-14.70	(-15.39 ; -14.00)	15.69	(14.90 ; 16.49)	15.24	(14.72 ; 15.75)
Senility	-8.97	(-9.48 ; -8.46)	-7.60	(-8.11 ; -7.09)	-4.87	(-5.41 ; -4.33)	-0.97	(-1.48 ; -0.45)	4.54	(3.96 ; 5.12)
Other sympt. & ill-def. cond.	-3.31	(-4.50 ; -2.11)	2.62	(1.70 ; 3.55)	5.48	(4.45 ; 6.51)	1.04	(0.30 ; 1.77)	4.45	(3.78 ; 5.13)
Other diseases	-0.26	(-0.87 ; 0.34)	0.83	(0.42 ; 1.25)	1.80	(1.50 ; 2.11)	2.02	(1.79 ; 2.26)	-1.67	(-1.93 ; -1.42)

Notes: CI = confidence interval; IHD = ischaemic heart disease; COPD= chronic obstructive pulmonary disease. [a] See "other cancers" as listed in Table 1.

Second, some causes of death underwent a sustained increase in mortality until the 1980s or even the 1990s, such as lung cancer, all the moderately smoking-related cancers, prostate cancer, unspecified cancers, chronic obstructive pulmonary disease (COPD), and "other symptoms and ill-defined conditions". As a result of their long-term increase, these causes gradually had an increased share in total mortality (see also Table 1) and thus their trends had increasingly more effect on the trends observed for all-cause mortality.

For women (Table 2b), the causes of death with an unfavorable trend were almost the same as those for men. However, cancer of the bladder and "other cancers" did not show unfavorable trends, and accidental fall exhibited a stagnation of the decrease. Mortality from unspecified cancers and COPD showed a recent increase, instead of the long-term increase observed for men.

To assess whether the trends in both total and cause-specific mortality among those aged 60 and older followed predominantly a period or a cohort pattern, we conducted an APC analysis. In Table 3 the scaled deviances – a measure of the unexplained variance - are given for the age-period (AP) model, the age-cohort (AC) model, and the APC model. They are expressed as percentages of the scaled deviance of the model using age only. The lower the scaled deviance of a model, the better the model is able to describe the trends observed.

For total male mortality the scaled deviance of the AC model (32%) was much lower than the scaled deviance of the AP model (52%), indicating that the trends predominantly followed a cohort pattern. For women, the scaled deviance of the AC model (11%) was higher than the scaled deviance of the AP model (6%); here, therefore, period patterns contributed more to the trends observed.

For most causes of death, the scaled deviance for the AP model was much lower than the scaled deviance for the AC model, indicating that the secular trends followed a period rather than a cohort pattern. Moreover, the little difference between the scaled deviance for the AP model and that for the APC model suggests that cohort effects, if any, were small. However, for some causes of death, the scaled deviances indicate that secular trends can be explained largely by cohort patterns. This applies to all smoking-related cancers (except for pancreatic cancer among men), prostate cancer, "other circulatory diseases" (men only), COPD (men only), and "other diseases".

Figure 2 shows the fitted mortality rates by birth year for all-cause mortality and a selection of causes of death, for men aged 80-84. The fitted mortality rates are based on the cohort analysis for those aged 60 and older as a whole. This analysis generated equal parameter estimates for each age group. Figure 2 therefore illustrates the overall cohort pattern for those aged 60 and older.

Total male mortality showed a reversal between birth cohorts 1880-1890 and 1890-1899, with annual percentage changes of –0.49 and 0.78 respectively. An annual percentage change of 0.78 indicates that mortality among those aged 60 and older increased by 0.78% per birth year between 1890 and 1899. Among the cohorts born between 1900 and 1909, mortality stagnated (-0.01); among the youngest birth cohorts, the mortality decline reemerged (-1.31).

Although the trends for the selected causes of death tended to vary, some general tendencies can be distinguished. "Other diseases" exhibited an increasing rate of decline in the youngest birth cohorts.

Table 3. Scaled deviances of different models for total and cause-specific mortality, for people aged 60 and older, by sex

Scaled deviance[a]	Men				Women			
	Age	AP	AC	APC	Age	AP	AC	APC
Total mortality	14,773	52	32	14	83,429	6	11	4
Strongly smoking-related cancers								
Esophagus	1,392	49	28	25	613	76	65	60
Upper respiratory / digestive system	497	94	86	74	482	92	77	75
Lung	30,838	23	5	2	6,952	15	8	7
Moderately smoking-related cancers								
Pancreas	2,000	22	33	17	1,311	41	37	33
Bladder	1,431	39	33	27	447	93	90	87
Kidney	1,950	27	18	17	944	49	44	43
Other cancers[b]	3,706	14	11	10	862	88	66	60
Other specific cancers								
Stomach	14,652	4	4	4	19,646	3	3	2
Colorectum	554	81	88	77	1,302	39	40	35
Breast					645	77	73	68
Prostate	1,917	44	39	35				
Unspecified	2,234	36	55	33	1,589	44	78	40
Cardiovascular diseases								
IHD	21,417	13	56	6	16,509	9	33	8
Other heart diseases	18,773	10	9	7	46,804	8	11	2
Cerebrovascular diseases	16,899	7	7	5	43,900	8	6	3
Other circulatory diseases	7,155	70	27	13	20,387	8	8	5
Other causes of death								
Infectious diseases	5,070	27	64	11	5,966	20	65	8
Pneumonia/influenza	19,383	16	46	11	27,661	17	44	9
COPD	13,007	46	15	10	2,971	26	70	21
Accidental fall	2,159	27	36	20	10,919	11	17	5
Other external causes	4,296	13	18	11	1,492	41	49	35
Diseases strongly sensitive to coding practices								
Diabetes mellitus	23,786	8	27	8	8,883	19	16	14
Dementia and Alzheimer's	7,680	17	66	16	19,980	11	56	10
Senility	13,588	14	27	8	22,328	17	34	7
Other symptoms & ill-defined conditions	7,206	13	22	12	5,285	16	26	16
Other diseases	4,562	60	50	20	9,852	86	59	12

AP = regression model with age and period splines as independent variables; AC = regression model with age and cohort splines as independent variables; APC = regression model with age, period and cohort splines as independent variable; IHD = ischaemic heart disease; COPD= chronic obstructive pulmonary disease.

[a] Expressed as percentage of the scaled deviance of the model including only age.

[b] This refers to the cause of death "other cancers" as listed in Table 1.

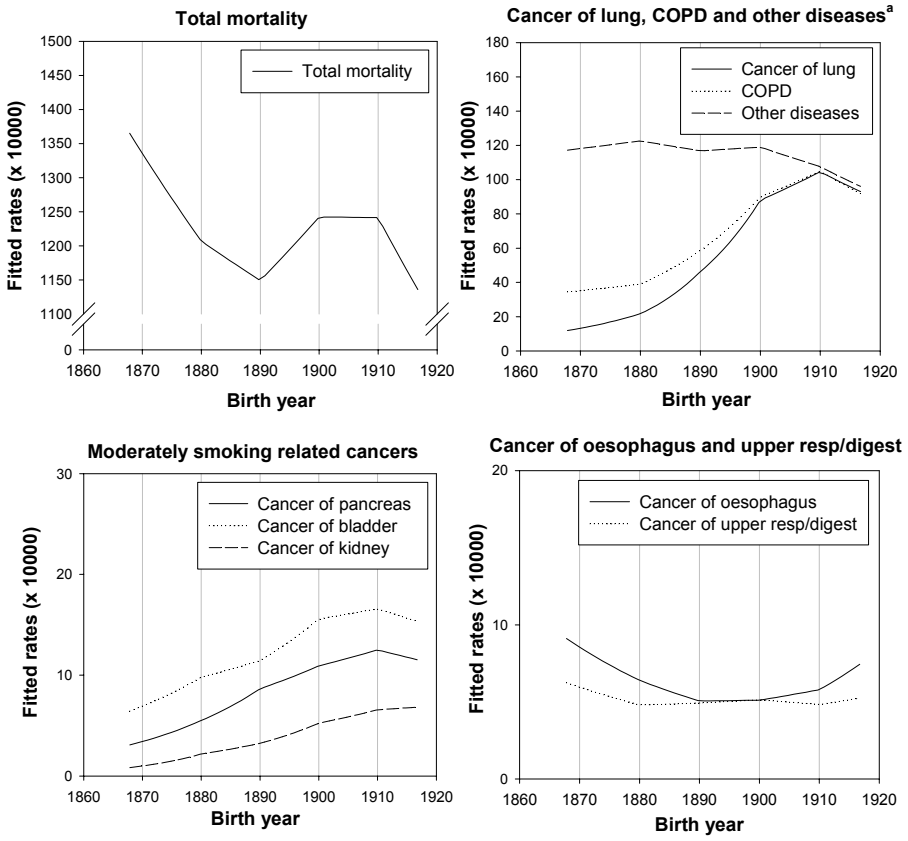

Figure 2. Fitted mortality rates by cohort for total mortality, smoking-related cancers, chronic obstructive pulmonary diseases, and other diseases[a] for men aged 80-84

[a] This refers to the cause of death "other diseases" as listed in Table 1.

Lung cancer and COPD followed a pattern of an increase in the oldest birth cohorts up to 1890-1899, changing to a decrease for those born after 1910. For cancer of the pancreas, bladder, and kidney, the same pattern applies, although it is less clear. Cancer of the esophagus and cancer of the upper respiratory or digestive system followed a different pattern, with a clear stagnation of the mortality decline and an increase among the youngest cohorts.

For total female mortality, the cohort trends did not show an increase in mortality for a particular cohort group but instead a declining trend throughout, with only a modest stagnation of the mortality decline for the youngest birth cohort. The annual percentage changes were –0.45, -1.86, -1.35, -1.57, and –1.08 for the successive cohort groups.

Table 4 shows the period trends that would be observed for all-cause mortality when smoking-related cancers and COPD are excluded.

Table 4. Annual changes (%) by period for total mortality and total mortality after exclusion of smoking-related cancers and chronic obstructive pulmonary diseases, for people aged 80 and older, by sex

Cause of death	1950-59		1960-69		1970-79		1980-89		1990-99	
	%	95% CI	%	95% CI	%	95% CI	%	95% CI	%	95% CI
Men										
Total	-0.57	(-0.72 ; -0.41)	-0.31	(-0.42 ; -0.19)	-0.89	(-0.99 ; -0.79)	0.37	(0.28 ; 0.47)	-0.18	(-0.30 ; -0.06)
Total after exclusion	-0.75	(-0.91 ; -0.58)	-0.68	(-0.80 ; -0.56)	-1.55	(-1.66 ; -1.44)	-0.21	(-0.32 ; -0.11)	0.00	(-0.14 ; 0.13)
Women										
Total	-1.08	(-1.23 ; -0.94)	-0.81	(-0.92 ; -0.70)	-2.67	(-2.76 ; -2.58)	-0.42	(-0.50 ; -0.33)	0.06	(-0.04 ; 0.15)
Total after exclusion	-1.17	(-1.32 ; -1.01)	-0.85	(-0.96 ; -0.74)	-2.87	(-2.96 ; -2.78)	-0.63	(-0.71 ; -0.54)	0.06	(-0.04 ; 0.16)

CI = confidence interval.

After their exclusion, stagnation of mortality decline during the 1980s and 1990s still prevailed. For men, the annual percentage changes would be –0.21 in the period 1980-1989 and 0.00 in the period 1990-1999. For women, these percentages would be –0.63 and 0.06 in the successive decades. Thus, after a number of causes of death specifically related to smoking were excluded, the trends in all-cause mortality for men and women showed a uniform pattern of a leveling off of the mortality decline in the 1980s and complete stagnation in the 1990s.

Further analysis revealed that, when the smoking-related cancers and COPD are excluded, the period and cohort models both can describe the observed trends for all-cause mortality. The scaled deviance for the AP model (as compared to the model with age only) was 6% for men and 7% for women. For the AC model, the scaled deviances were only slightly higher, that is, 11% for men and 10% for women.

DISCUSSION

Compared with previous research [15], this study has provided important new findings on the stagnation of old-age mortality in The Netherlands. First, a sudden reversal occurred around 1980, when the marked mortality decline of the 1970s turned into stagnation and in some cases even an increase during the 1980s and 1990s. Second, the causes of death contributing to the stagnation of the mortality decline are predominantly smoking-related diseases and diseases specifically related to old age. Third, among men, trends for smoking-related cancers and COPD followed a cohort pattern, which suggests an important role for differences between successive generations in exposure to smoking. Fourth, when these smoking-related diseases are excluded, the trends in all-cause mortality were found to follow a pattern of increasing stagnation for both men and women.

These results strongly suggest that, even though cohort changes in smoking exposure influenced the trends in mortality among older men in the 1980s, these changes do not fully explain the recent stagnation of mortality observed among elderly Dutch men and women. Before we can speculate on the role of other specific factors, possible bias caused by data problems or the methods used in our analysis should be evaluated.

Evaluation of data problems

The mortality and population data used in this study come from the municipal population registers. Data from the Dutch population registers are considered reliable and consistent [32]. The use of an individual-based register, which is started at the birth of an individual and contains all major demographic events throughout life, ensures that age misstatement cannot have affected our mortality rates. Because both population and mortality data are derived from the same source, the mortality rates are not subject to numerator-denominator bias [1].

Analyses of long-term trends in causes of death have to deal with several transitions between ICD revisions. We made considerable effort to bridge these ICD revisions by carefully constructing a concordance table and by controlling for the few remaining jumps in our regression analysis. Further checks showed minimal sensitivity. Thus, even though some residual effects of ICD transitions could not be excluded, we believe that these problems did not affect the results to any substantial extent.

More difficult to tackle were changes within an ICD revision, either in the reporting of causes of death by physicians or in coding practices at the Statistical Office. The causes of death expected to suffer substantial effects are diabetes mellitus, dementia, senility, and "other symptoms and ill-defined conditions". The recent observed increase in mortality from "other symptoms" could indicate less detailed and less accurate diagnoses. The increase in senility and dementia, however, is probably due to the growing propensity of physicians to report these diseases as underlying causes of death. An increase in the doctor's tendency to list only one cause on the death certificate may have the effect that diseases that were formerly reported mainly as secondary causes of death are increasingly listed as primary causes of death. In particular, this might influence trends in mortality from diabetes and pneumonia, which are common secondary causes of death in The Netherlands [33].

In view of these possibilities, we should be cautious about interpreting the marked increases in mortality observed for the causes of death mentioned herein. In addition, these increases in mortality will in some cases have an opposite effect on other diseases. For example, an increasing tendency to report diabetes mellitus as the underlying cause of death may result in an underestimation of an increase (or overestimation of a decrease) in mortality from cardiovascular diseases. However, the potential for bias should not be generalized to all causes. Some causes of death may be relatively resistant to changes in coding practices, especially those diseases that have a more straightforward diagnosis. This applies to most of the smoking-related cancers and probably COPD as well.

Evaluation of methods

As far as the APC analysis was concerned, we were aware of the identification problem. We solved this problem by assessing predominantly nonlinear trends, using splines within our regression models. Moreover, by comparing the scaled deviances of the AP model and the AC model, we could make valid statements on the role of period or cohort patterns in the trends observed, especially when the scaled deviances of these two models showed large differences. Such a large difference was observed in several cases, including smoking-related cancers and COPD.

Explanations of the observed trends

Smoking is frequently mentioned in the literature [5,16,34] to explain some of the unfavorable trends in mortality in The Netherlands compared with other European or low-mortality countries. In our analysis, we found that although smoking contributed to the stagnation of the mortality decline among men in the 1980s, mortality from smoking-related cancers and COPD decreased in the 1990s and among the youngest birth cohorts. In addition, when smoking-related cancers and COPD were excluded from all-cause mortality, the stagnation of mortality still occurred and was consistent among both men and women. It should be noted that only smoking-related diseases with a clear cohort pattern were excluded, thus ignoring the period effects of smoking on some other causes of death. In The Netherlands, the period pattern of recent changes in smoking behavior is likely to have a favorable effect on mortality in the 1980s and 1990s [17], especially for cardiovascular diseases. This is due to a decline in smoking prevalence observed among Dutch men (65+) since the 1970s [35]. If these period effects were to be taken into

account, then an even stronger increase in old-age mortality might become apparent. Thus, to explain the stagnation of old-age mortality in The Netherlands, we should look beyond the role of smoking.

The first possible explanation is that there is simply no room for further improvement in mortality among the Dutch elderly population, because low levels of mortality have already been reached. Although mean life expectancy at age 80 in The Netherlands in the period 1980-1990 was quite high, that is 7.60 years, this level was already surpassed by other countries, for example, in Iceland (e_{80} = 8.18) or Japan (e_{80} = 7.67) [36]. Moreover, in those countries with equal or higher levels of life expectancy, the decrease in mortality persisted (Vaupel et al., 1998). This suggests that the unfavourable trends in old-age mortality as observed in The Netherlands cannot be simply understood from low levels of mortality already attained or a limit to life expectancy being approached.

A second possible explanation for the stagnating trends could be mortality selection. As a result of the reduction in old-age mortality in the decades before the 1980s, an increasing proportion of people born in successive generations has reached old age. It is likely that this increasing proportion of elderly people does not include only the fittest but also those who are more frail. Improvements in medical care are frequently mentioned as an important reason for the marked mortality decline in the 1970s [37-39], which strengthens our hypothesis on the decline in mortality selection. This decline in mortality selection could perhaps explain the relatively strong increase in mortality from diseases related to frailty – such as infectious diseases – compared with the continued mortality decline in cardiovascular diseases. However, even though mortality selection may have played a role in the stagnation of the mortality decline, it cannot explain why mortality trends are much less favorable in The Netherlands than in other low-mortality countries.

A particular feature of Dutch society is the relatively liberal attitude toward euthanasia and related end-of-life decisions. The most frequently made end-of-life decision for elderly people in The Netherlands is the decision to withhold life-prolonging treatment [40]. These decisions refer not only to technological interventions but also and most frequently to the withdrawal or withholding of antibiotics and artificial nutrition or hydration [41]. Whereas in 1990, 33% of all deaths among those aged 80 and older were preceded by a nontreatment decision, this had increased to 36% in 1995 [41,42]. This increasing incidence might reflect the growing autonomy of patients, who want to decide for themselves whether or not to undergo treatment. However, it is likely that the growing incidence is also the result of increased possibilities for treatment. With more alternatives available, the decision to withhold a particular treatment will be made more often. In addition, the estimated effect of the decision to withhold treatment on the patient's life expectancy was less than 1 month in approximately 90% of cases [41]. From more detailed data, we estimated that the effect on life expectancy among the Dutch elderly population is a loss of approximately 0.01 year. The effects of medical decisions related to the end of life on Dutch mortality trends thus seem to be negligible.

The abrupt reversal around 1980, from a marked mortality decline in the 1970s to stagnation thereafter, raises the question of which parallel changes occurred in The Netherlands at the same time or shortly before. In the period 1975-1985, concern focused on health care costs, and budget cuts were proposed by the Dutch government [43]. When

health care resources become limited, elderly people are often the first group to suffer, because medical costs rise exponentially with age. However, it is unclear when and to what extent the concern prevalent in this period had an effect on the health care services delivered to the elderly population.

Changes also occurred in the health care and social services available for elderly people. In The Netherlands between World War II and 1975, care for elders was a central issue of the welfare state. Considerable effort and resources were spent on institutionalization and intramural care for elderly people [44]. Since 1975, however, Dutch policies have shifted and the emphasis has changed to facilities for elders that enable them to stay at home longer – in parallel with the growing autonomy among the elderly population. This trend could have made this group more reliant on informal or private care [44]. Combined with the increase in the number of elderly people living alone and the decrease in social support with age [45], this may have resulted in more elderly people without sufficient care, which ultimately might have a negative effect on their length of life. In contrast, the decline in institutionalization might also have had positive effects on old-age mortality, for example as a result of physical activity and self-sufficiency. It is unclear whether these positive effects can outweigh the negative effects.

Although the factors discussed herein remain somewhat speculative, they suggest that several developments contributed to the unfavorable mortality trends among the oldest old in The Netherlands. The stagnation of mortality decline is most likely explained by a combination of the growing frailty of the Dutch elderly population together with changes in the medical and social services available to them. Because these factors are determined by developments that are not necessarily restricted to The Netherlands, this implies that a continuing decline in old-age mortality in other low-mortality countries should no longer be taken for granted. Although mortality at old age is remarkably plastic and has the potential for further reduction [46], changes in the possible determinants discussed here may well result in an increase in old-age mortality.

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Appendix 1. The concordance table to bridge the revisions of the International Classification of Diseases (ICD) from 1950-1999, The Netherlands

Cause of death	ICD6/7 (1950-1968)	ICD8 (1969-1978)	ICD9 (1979-1995)	ICD10 (1996-1999)
Infectious and parasitic diseases	001-138	000-136	001-139	A00-B99
Cancer of the oesophagus	150	150	150	C15
Cancer of the stomach	151	151	151	C16
Cancer of the colorectum	153-154	153-154	153-154	C18-C21
Cancer of the pancreas	157	157	157	C25
Cancer of the upper respiratory / digestive system	140-148, 160, 161	140-149, 160, 161	140-149, 160, 161	C00-C14, C30-C32
Cancer of the lung	162-163	162	162	C33-C34
Cancer of the breast	170	174	174-175	C50
Cancer of the prostate	177	185	185	C61
Cancer of the bladder	181	188	188	C67
Cancer of the kidney	180	189	189	C64-C66, C68
Cancers, unspecified	198-199, 230-239	195-199, 230-239	195-199, 235-239	C76-C80, C97, D37-D48
Other cancers	Rest (140-239, 294)	Rest (140-239)	Rest (140-239)	Rest (C00-D48)
Diabetes mellitus	260	250	250	E10-E14
Dementia and Alzheimer	304-306	290, 293	290, 331	F00, F01, F03, G30
Ischaemic heart diseases	420, 422.1	410-414	410-414	I20-I25
Other heart diseases	400-402, 410-416, 421, rest (422), 430-434, 440-447	390-398, 400-404, 420-425, 427-429	390-398, 401-405, 416, 420-429	I00-I13, I15, I27, I30-I52
Cerebrovascular diseases	330-334	430-434, 436-438	430-434, 436-438	I60-I69
Other circulatory diseases	Rest (400-468)	Rest (390-458, excl 435 & 446)	Rest (390-459, excl 435 & 446)	Rest (I00-I99)
Pneumonia/influenza	480-483, 490-493	470-474, 480-483, 485-486	480-487	J10-J18
Chronic obstructive pulmonary diseases	501, 502, 526, 527, 241	490-493, 518	490-494, 496	J40-J47
Senility	794	794	797	R54
Other symptoms and ill-defined conditions	Rest (780-795)	Rest (780-796)	Rest (780-799)	Rest (R00-R99)
Other diseases	Rest (001-795)	Rest (000-796)	Rest (001-799)	Rest (A00-R99)
Accidental fall	E900-904	E880-887	E880-888	W00-W19, X59
Other external causes	Rest (E800-999)	Rest (E800-999)	Rest (E800-999)	Rest (V01-Y98)

4

TRENDS IN OLD-AGE MORTALITY IN SEVEN EUROPEAN COUNTRIES, 1950-1999

Objective Different from the general observed decline in old-age mortality, for The Netherlands and Norway there have been reports of stagnation in the decline since the 1980s. We detect periods of stagnation in recent old-age mortality trends, and explore for which causes of death the recent stagnation is most apparent. **Methods** We applied Poisson regression analysis to total and cause-specific mortality data by age (80+), period (1950–1999), and sex, for seven European low-mortality countries. **Results** We found large heterogeneity in the pace of decline in the countries under investigation, with periods of stagnation being widespread. In the 1980s and 1990s, stagnation was observed in Denmark, The Netherlands, and Norway (males). Continued mortality decline was observed especially in France. Although smoking has had a marked influence on the trends in old-age mortality, the role of smoking in the recent stagnation seems only modest and restricted to Norway. Mortality from cardiovascular diseases showed important cross-national variations in the pace of decline. Mortality from diseases specifically related to old age increased recently in all countries, except France. **Conclusion** Old-age mortality seems highly plastic and susceptible to many factors, with both favorable and unfavorable effects on trends over time.

BACKGROUND

The general tendency in the trends in old-age mortality (80+) in low-mortality countries since the 1950s has been a declining one [1–4]. This tendency has contributed to the current increase in the number, the proportion, and the mean age of elderly people in these populations.

Because these developments will have huge implications for policy, society, and the demand of health care services, there has been much interest in possible future trends in life expectancy, especially in the debate on the limit to life expectancy [5–12]. On the one hand, proponents of "the limited-lifespan paradigm" state that biologic and practical constraints on reducing old-age mortality set an upper limit to life expectancy of approximately 85 years [5,9,10]. On the other hand, "the mortality-reduction paradigm" is adhered to by researchers who argue that life expectancy will continue to increase - at least for some time to come - due to substantial reductions in mortality rates at all ages, including the oldest old [6,8,11]. They support their view by referring to mortality development in subpopulations with extreme good health, to foreseen biomedical progress, and to historic observations that old-age mortality has been decreasing continuously.

Recently, however, research on old-age mortality has shown some exceptions to this continuous decline in old-age mortality. For The Netherlands and Norway, there have been reports of stagnation or even increases in old-age mortality since the 1980s [2,13,14]. This raises the question of whether the mortality decline among elderly in low-mortality

countries has been as consistent as previously reported, and whether these developments coincide with the idea that life expectancy continues to increase in the future.

However, before making inferences about future trends in old-age mortality, it is important to investigate the determinants of trends in old-age mortality. Although there have been studies on old-age mortality in the past that acknowledge the heterogeneity of the trends between countries [2,15,16], they were mostly descriptive in nature. As a result, little is known about the reasons behind the crossnational differences in the pace of mortality decline among the elderly. Knowing about the determinants of trends in old-age mortality could provide important clues on future mortality among the elderly population, especially in the short and medium term.

Studying the trends in cause-specific mortality can generate evidence on the determinants of the trends in all-cause mortality among the elderly, because many of the intermediate factors or risk factors that could determine mortality trends among the elderly are related to specific causes of death. In doing so, we will emphasize the role of smoking. It is commonly known that smoking has a strong negative effect on survival. The impact of smoking on mortality trends may vary considerably between countries [17]. In fact, the stagnation observed in some countries could be the result of the increased lifetime exposure to smoking for those birth cohorts who reached old age at the end of the 20th century [18,19].

The objective of this article is to describe mortality trends among the elderly in Denmark, England and Wales, Finland, France, The Netherlands, Norway, and Sweden, and to contribute to the explanation of these trends. Focus will be on the trends from 1980 onwards. The research questions we will address are: (a) in which countries did trends in mortality at old age show signs of stagnation instead of continued decline, (b) to what extent did mortality increase for causes of death related to smoking, and (c) for which other specific causes of death did an increase occur?

In this analysis, we will extend earlier studies on trends in old-age mortality by using detailed mortality data over a long period of time (1950–1999), with a distinction by specific causes of death and 5-year age groups up to 100+. Furthermore, in our analysis an extensive effort was made to carefully bridge the different revisions of the International Classification of Diseases (ICD).

DATA AND METHODS

Data

Data were obtained from national statistical offices and related institutes on total mortality, cause-specific mortality, and population at risk for Denmark, England and Wales, Finland, France, The Netherlands, Norway, and Sweden, for the years 1950–1999 (National Institute of Public Health [Denmark], ONS [CD-Rom Twentieth Century Mortality] [England and Wales], Statfin [Finland], INED and INSERM [France], Statistics Netherlands and NIDI [The Netherlands], Statistics Norway, and National Board of Health and Welfare [Sweden]).For Denmark, Finland, and Norway data were available only from 1951, for Sweden from 1952. Data for France were available until 1997 and for Denmark until 1998.

The selection of countries was restricted to low-mortality countries in North Western Europe. The seven countries we included were selected on the basis of the quality of the data [1,2] and on the availability of cause of death data.

For most countries, data on the total number of death by 5-year age groups were available, with a maximum age ranging from 85+ to 100+. For The Netherlands and France, deaths by single year of age were available to us (no maximum age)(see for France http://www.ined.fr/publications/cdrom_vallin_mesle/continu.htm. Tables de mortalité françaises 1806–1997 et projections jusqu'en 2102 by J. Vallin and F. Meslé (INED)). To calculate mortality rates we used the midyear-population at risk. Population data were available either by single year of age (France, The Netherlands, Finland, Sweden, England, and Wales 1961–1999), or by 5-year age groups (Denmark, Norway, England, and Wales 1950–1960), with the maximum age ranging from 85+ to no maximum age.

For those aged 80 and over, we had additional data on total mortality data and population data available from the Kannisto-Thatcher Database on Old Age Mortality (K-T Database) at the Max Planck Institute for Demographic Research (http://www.demogr.mpg.de/databases/ktdb) [2]. The data were grouped by both year of death and year of birth of the deceased and by single year of age (no maximum age). We used these data (a) to redistribute the population numbers and deaths in the older age groups over 5-year age groups up to 100+, (b) to redistribute the population numbers and total deaths for those aged 80 and over from 5-year age groups into data by single year of age, and (c) to check whether the population and mortality data from the different data sources (Kannisto-Thatcher Database and national data) were consistent.

For the causes of death, we obtained data on their prevalence as the underlying cause of death. These data were available by three-digit codes, 5-year age groups, sex, and year of death for all countries. The maximum age ranged from 85+ for England and Wales and The Netherlands (until 1969), and 90+, 95+, and 100+ for all other countries and periods. The deaths per cause in the older age groups were redistributed over 5-year age groups up to 100+ based on the distribution of total mortality in these age groups. Table 1 lists the causes of death we selected and their relative share in all-cause mortality among those aged 80 and over in 1995–1999. Cancers have been designated "smoking-related" if their population attributable risk was larger than 0.25 (according to the American Cancer Study) [20].

Statistical analysis

We analyzed the mortality data by means of a (log-linear) Poisson regression model with linear splines. The dependent variable was the number of deaths, with the person-years at risk as offset variable. As independent variables, we used age (single year of age for total mortality and 5-year age groups for cause-specific mortality) and year of death. Spline functions divide the overall trend into a number of separate, adjacent segments [21]. In our analysis, we used five segments each covering a period of 10 years (1950–1959, 1960–1969, 1970–1979, 1980–1989, 1990–1999). The analysis using splines thus yielded estimates of annual changes in mortality within each 10-year period, thereby taking into account the overall trend. Comparison of these five decade-specific rates of change enabled us to detect and quantify changes in the secular trend in mortality, such as a stagnation of the decrease in mortality for a specific country.

Table 1. List of selected causes of death and their relative share in all-cause mortality in the period 1995-1999[a], males and females, aged 80 and over

	Relative share (%)													
	Males							Females						
	DK	E&W	FIN	F	NL	NO	S	DK	E&W	FIN	F	NL	NO	S
Smoking-related diseases	*12.1*	*14.0*	*9.7*	*11.8*	*17.2*	*9.2*	*7.4*	*5.9*	*6.4*	*3.6*	*5.5*	*5.8*	*4.6*	*4.3*
Cancer of lung	3.4	4.2	3.2	3.1	5.5	2.0	1.7	1.2	1.5	0.7	0.6	0.7	0.6	0.7
Other smoking-related cancers[b]	3.1	3.2	2.5	3.8	3.2	3.0	2.5	1.8	2.0	2.0	1.9	2.0	1.9	1.9
COPD	5.6	6.6	4.0	4.9	8.5	4.3	3.2	2.9	2.9	0.9	3.0	3.1	2.0	1.6
Cardiovascular diseases	*45.6*	*42.8*	*48.3*	*37.9*	*39.0*	*48.6*	*53.9*	*48.6*	*45.6*	*52.8*	*42.7*	*42.3*	*52.1*	*56.8*
Ischemic heart disease	22.7	23.0	29.7	10.2	14.2	22.1	26.9	21.3	19.8	28.6	9.3	12.1	19.1	23.5
Cerebrovascular diseases	10.1	10.9	11.2	9.4	9.4	12.6	11.3	12.8	15.1	14.6	11.6	12.9	15.8	14.8
Other circulatory diseases	12.9	9.0	7.4	18.3	15.4	14.0	15.8	14.4	10.6	9.6	21.8	17.2	17.1	18.6
Other causes of death	*42.3*	*43.2*	*42.1*	*50.3*	*43.8*	*42.2*	*38.7*	*45.5*	*48.0*	*43.6*	*51.8*	*51.9*	*43.4*	*38.9*
Other cancers	12.9	12.0	11.8	15.2	13.8	14.3	13.4	11.4	9.4	8.9	11.5	11.6	10.2	9.9
Infectious diseases	0.5	0.4	0.8	1.6	0.8	1.0	1.0	0.6	0.4	0.7	1.5	1.0	1.1	1.0
Diabetes mellitus	1.3	1.0	0.7	1.1	1.7	1.2	1.5	1.4	1.0	1.0	1.5	2.9	1.5	1.7
Pneumonia	5.3	14.8	9.8	5.2	7.1	8.3	6.6	5.3	16.4	8.2	4.7	7.4	9.3	6.0
Dementia	2.1	2.0	7.8	2.6	3.6	1.8	3.3	2.9	3.3	12.8	4.1	7.5	3.6	5.8
All ill-defined causes	7.8	2.2	0.2	5.9	4.4	3.9	2.0	10.9	5.4	0.3	8.4	5.8	5.2	3.7
Other diseases	8.1	9.5	7.4	13.7	10.3	8.2	8.2	8.4	10.6	8.7	14.8	13.2	8.7	8.4
All external causes	4.2	1.3	3.7	5.1	2.1	3.5	2.7	4.5	1.4	3.0	5.2	2.5	3.7	2.4
All causes (N)	40,818	465,606	33,766	285,979	111,731	43,876	102,168	64,025	824,395	70,572	488,581	193,384	67,349	146,963

[a] Or last year available.
[b] This includes cancer of the esophagus, pancreas cancer, cancer of the upper respiratory and digestive system, bladder cancer, and cancer of the kidney.
DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.
COPD = chronic obstructive pulmonary diseases.

"Stagnation" of the mortality decline was defined as either a leveling off of the mortality decline leading to small declines or a reversal into increasing mortality. All our analyses were conducted using SAS package version 8. In addition, for total mortality, directly standardized observed and fitted mortality rates were calculated, using the total population of England and Wales in 1999 as the reference.

The use of 5-year age groups in the cause of death analyses was due to the restriction that these data were not available to us by single year of age. To evaluate to what extent a possible change over time in the distribution of deaths within a 5-year age group could affect our results, we compared the results for total mortality using data by 5 years of age with the results for total mortality using data by single year of age. Because this comparison generated virtually the same results, we expected that, although age patterns can differ for the specific causes of death, the bias when using the data by 5-year age groups will be minimal.

Concordance

When analyzing trends in causes of death for a longer period, numerous revisions of the World Health Organization (WHO) International Classification of Diseases (ICD) have to be taken into account, because they can lead to biases in the trends. In our analysis, we had to bridge four or five different ICD revisions per country. For this purpose we constructed a general concordance table in which the different three-digit codes for a specific cause of death in successive ICD revisions were linked (see Janssen et al. (2003) [14] for more information)(see Appendix I). To accurately bridge the revision from ICD6/7 to ICD8 for ischemic heart disease (IHD) we obtained the numbers of death for one additional four-digit code (422.1) under ICD6/7. These numbers were not available for Finland until 1963, and Sweden until 1961. We estimated them on the basis of the ratio of the number of deaths from ischemic heart disease with and without 422.1 calculated for the first year in which 422.1 was coded.

On the basis of this general concordance table, data on the required three-digit codes were obtained from the different countries. For Finland, however, not all causes of death were available by three-digit code and part of the data we had to request by short list consisting of slightly different groups of causes of death. In depth analysis of the Finnish mortality trends suggested that this approximation has not led to irregularities.

Cause-specific trends based on the concordance table can, however, still contain irregularities due to (1) remaining problems with ICD revisions, because at the level of three-digit codes the continuity of the medical content of some causes of death could not always be optimized; (2) incidental changes in coding rules, for example, changes within ICD revisions in the reporting of causes of death by physicians or in coding rules applied at the statistical offices; and (3) incidental outliers, that is, causes of death with a single year of exceptional mortality levels. In addition to the use of the concordance table, it was therefore necessary to trace these irregularities and to control for them when assessing long-term trends in mortality.

We identified outliers and incidental changes in coding rules on the basis of visual analysis of cause-specific trends for males and females combined for those aged 60 and over, and using country-specific background information on the irregularities observed. To evaluate

the existence of remaining problems with ICD revisions, we identified possible mortality jumps due to the ICD revisions, using cause-specific regression models (with splines) applied to data for males and females combined, aged 60 and over. To these regression models we added transition variables indicating the ICD revisions, that is, ICD6/7to8, ICD8to9 or ICD9to10. In this way, these regression models generated parameter estimates for the transition variables.

A transition variable was included in our final regression model if: (1) the parameter estimate corresponding to this variable was statistically significant, (2) the significant effect could not be attributed to nonlinear trends or to a single outlier, for instance, an influenza epidemic nearby; and (3) the observed effect could be traced back to a ICD transition problem, for example, due to a four-digit code not included in the concordance table or an effect on one cause of death mirrored by an opposite effect on a complementary cause of death.

For France, no check on the concordance was needed, because J. Vallin and F. Meslé reconstructed coherent series of data for causes of death, the results of which are available from a Web site (http://matisse.ined.fr/%7Etania/causfra/data/) [22,23]. From this Web site the cause of death data we distinguished could be extracted using ICD9 codes.

RESULTS

Old-age mortality showed an overall decline and a convergence in the mortality level between countries over time (Figure 1). However, there was a large heterogeneity in the pace of decline, with periods of stagnation (defined as either a leveling off of the mortality decline leading to small declines or a reversal into increasing mortality) being widespread. In the 1950s, and to a lesser extent in the 1960s, mortality in the Nordic countries declined slightly or even increased. From the 1980s onwards, small declines or even increases were observed in Denmark, The Netherlands, and among Norwegian males. In contrast, mortality decline continued in England and Wales (females), and especially in France, resulting in the lowest mortality level in the 1990s. Striking is the huge mortality decline in Finland in the 1970s, which was followed by a much more modest decline in the 1980s. The mortality rates were higher among males compared to females, with the mortality level of males in the 1990s being about equal to the mortality level of females in the 1950s.

To identify the periods of stagnation more precisely, we quantified the pace of all-cause mortality decline within each of the 5 decades by means of annual changes (%) (Table 2). If the confidence intervals for successive decades do not overlap the changes in the pace of mortality decline are statistically significant. Stagnation was more widespread among males compared to females. In Denmark, mortality decline leveled off from the 1970s onwards for males, and from the 1980s onwards for females. Mortality decline among Dutch males turned into increase in the 1980s, followed by a small decline in the 1990s. Among Dutch females mortality decline leveled off in the 1980s and 1990s. Among Norwegian males the small (nonsignificant) increase in the 1980s changed into a modest decline in the 1990s.

The annual trends for the "smoking-related diseases", that is, smoking-related cancers and chronic obstructive pulmonary disease (COPD), showed an overall pattern of long-term increases (Table 3).

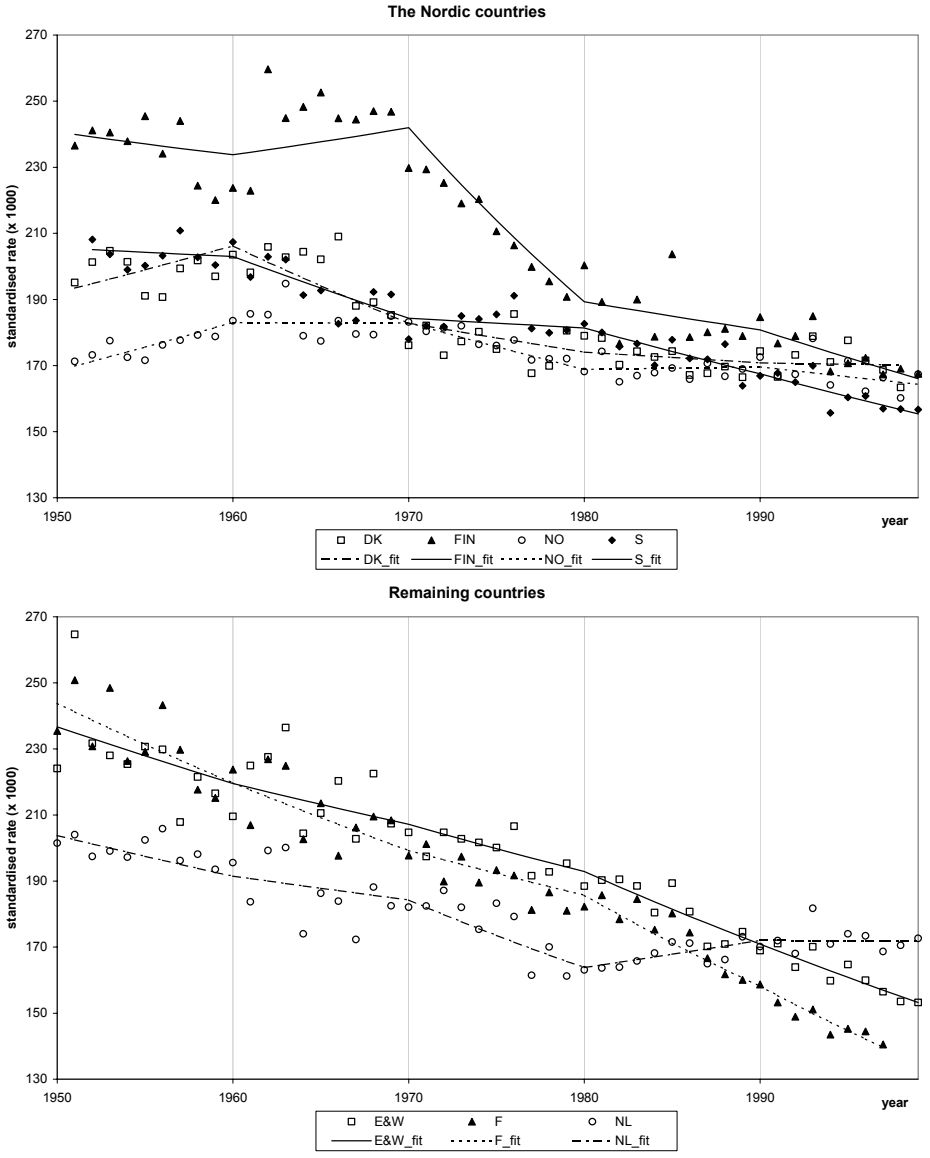

Figure 1a. Trends in standardized observed and fitted all-cause mortality rates in seven countries, 1950-1999, males, aged 80 and over

DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.

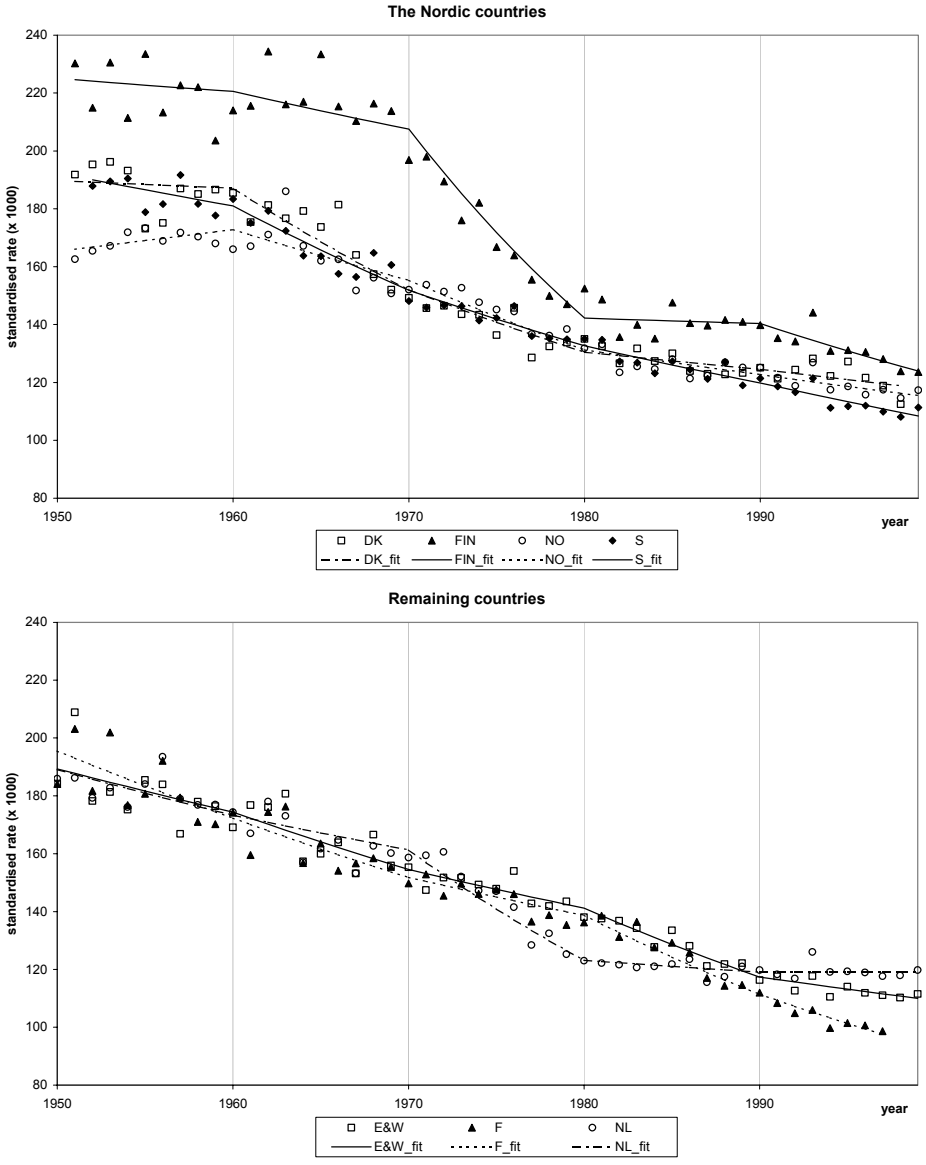

Figure 1b. Trends in standardized observed and fitted all-cause mortality rates in seven countries, 1950-1999, females, aged 80 and over

DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.

Table 2. Annual trends in total mortality by country and decade (1950-1999), males and females, aged 80 and over

	Annual changes									
	1950-59		1960-69		1970-79		1980-89		1990-99	
	%	95%CI	%	95%CI	%	95%CI	%	95%CI	%	95%CI
Males										
Denmark	**0.58**	(+0.32 ; +0.84)	-1.16	(-1.33 ; -0.99)	-0.45	(-0.60 ; -0.30)	-0.19	(-0.33 ; -0.04)	-0.20	(-0.41 ; +0.01)
England and Wales	-0.65	(-0.71 ; -0.58)	-0.79	(-0.84 ; -0.73)	-0.44	(-0.49 ; -0.39)	-1.44	(-1.48 ; -1.39)	-1.20	(-1.26 ; -1.14)
Finland	-0.54	(-0.91 ; -0.17)	**0.27**	(+0.02 ; +0.53)	-2.46	(-2.67 ; -2.24)	-0.55	(-0.75 ; -0.36)	-1.11	(-1.33 ; -0.88)
France	-1.27	(-1.34 ; -1.20)	-0.98	(-1.03 ; -0.92)	-0.73	(-0.78 ; -0.67)	-1.82	(-1.87 ; -1.77)	-1.66	(-1.74 ; -1.58)
The Netherlands	-0.60	(-0.76 ; -0.44)	-0.35	(-0.47 ; -0.24)	-0.93	(-1.03 ; -0.82)	**0.38**	(+0.29 ; +0.48)	-0.19	(-0.31 ; -0.07)
Norway	**0.76**	(+0.48 ; +1.04)	**0.14**	(-0.04 ; +0.33)	-0.82	(-0.98 ; -0.65)	0.02	(-0.13 ; +0.18)	-0.51	(-0.71 ; -0.32)
Sweden	-0.17	(-0.39 ; +0.04)	-0.85	(-0.98 ; -0.73)	-0.20	(-0.31 ; -0.09)	-0.92	(-1.02 ; -0.81)	-0.98	(-1.11 ; -0.85)
Females										
Denmark	-0.09	(-0.32 ; +0.15)	-2.25	(-2.41 ; -2.10)	-1.67	(-1.81 ; -1.54)	-0.47	(-0.59 ; -0.35)	-0.56	(-0.72 ; -0.39)
England and Wales	-0.96	(-1.02 ; -0.91)	-1.30	(-1.34 ; -1.26)	-0.88	(-0.91 ; -0.84)	-1.90	(-1.94 ; -1.87)	-0.65	(-0.69 ; -0.60)
Finland	-0.18	(-0.44 ; +0.09)	-0.70	(-0.88 ; -0.52)	-3.85	(-4.00 ; -3.70)	-0.34	(-0.48 ; -0.21)	-1.47	(-1.63 ; -1.32)
France	-1.42	(-1.48 ; -1.37)	-1.40	(-1.45 ; -1.36)	-1.07	(-1.10 ; -1.03)	-2.23	(-2.27 ; -2.20)	-1.72	(-1.78 ; -1.66)
The Netherlands	-1.11	(-1.26 ; -0.97)	-0.85	(-0.96 ; -0.74)	-2.72	(-2.81 ; -2.63)	-0.47	(-0.55 ; -0.39)	0.01	(-0.08 ; +0.10)
Norway	**0.43**	(+0.19 ; +0.68)	-0.97	(-1.13 ; -0.81)	-1.80	(-1.94 ; -1.66)	-0.74	(-0.87 ; -0.61)	-0.71	(-0.86 ; -0.55)
Sweden	-0.61	(-0.81 ; -0.42)	-1.86	(-1.97 ; -1.74)	-1.51	(-1.61 ; -1.41)	-1.14	(-1.22 ; -1.05)	-1.05	(-1.15 ; -0.94)

Bold indicates a significant increase.
Underlined indicates annual changes (%) ≥ –0.60.

OLD-AGE MORTALITY TRENDS IN EUROPE

57

Table 3. Annual trends in mortality from smoking-related diseases by country and decade (1950-1999), males and females, aged 80 and over

	Annual changes (%)									
	Males					Females				
	1950s	1960s	1970s	1980s	1990s	1950s	1960s	1970s	1980s	1990s
Denmark										
Smoking-related diseases	***7.51***	***1.74***	***4.34***	***1.64***	***0.20***	***5.45***	***0.35***	***1.07***	***1.00***	***3.04***
Lung cancer	**12.15**	**4.63**	**6.66**	**1.36**	-0.06	**7.02**	**1.64**	**2.96**	**2.27**	**3.49**
Other smoking-related cancers	**2.92**	-0.56	**3.47**	**0.09**	-0.59	**3.48**	-0.50	**2.17**	-1.33	-0.53
COPD	**12.87**	**2.32**	**3.56**	**2.93**	**0.81**	**7.87**	**0.88**	-1.11	**3.23**	**5.54**
England and Wales										
Smoking-related diseases	***1.12***	***1.68***	***1.23***	***0.24***	*-3.48*	*-2.74*	*-1.49*	*-0.55*	***1.76***	***0.43***
Lung cancer	**10.33**	**6.52**	**5.22**	**0.02**	-3.47	**3.70**	**4.27**	**4.91**	**3.48**	**1.21**
Other smoking-related cancers	**0.50**	-0.33	**0.72**	**0.69**	-0.87	**1.99**	**0.15**	**0.56**	**0.98**	-0.32
COPD	**0.06**	**1.05**	-0.50	**0.16**	-4.54	-4.58	-3.14	-2.98	**1.49**	**0.68**
Finland										
Smoking-related diseases	***8.30***	***3.65***	***0.87***	*-2.17*	*-1.15*	***5.51***	***2.22***	*-0.89*	*-1.07*	***0.79***
Lung cancer	**9.21**	**4.68**	**3.92**	-1.10	-1.77	**5.03**	**2.20**	**3.19**	**0.55**	**3.62**
Other smoking-related cancers	**0.07**	**1.76**	**1.30**	-0.89	-1.72	**2.31**	**1.61**	**0.15**	-0.64	-0.51
COPD	**16.84**	**4.59**	-1.07	-3.81	-0.21	**14.33**	**3.38**	-4.24	-3.19	**1.94**
France										
Smoking-related diseases	***1.75***	***2.60***	***3.75***	*-0.07*	*-0.62*	***0.96***	***0.50***	***1.17***	***1.23***	***1.38***
Lung cancer	**10.69**	**6.91**	**6.63**	**1.88**	**0.02**	**6.73**	**3.71**	**1.26**	**1.98**	**2.02**
Other smoking-related cancers	**3.84**	**2.57**	**1.34**	**0.12**	-1.29	**2.93**	**1.78**	**0.27**	**1.20**	**0.48**
COPD	-1.12	**1.17**	**4.62**	-1.37	-0.40	-0.76	-1.09	**1.88**	**1.11**	**1.87**
The Netherlands										
Smoking-related diseases	***1.59***	***4.48***	***3.78***	***3.08***	*-1.13*	*-0.62*	***0.74***	*-0.15*	***2.62***	***0.61***
Lung cancer	**6.96**	**8.19**	**7.55**	**1.85**	-1.97	**5.39**	-0.47	**4.92**	**0.87**	**2.63**
Other smoking-related cancers	**1.46**	**2.26**	**1.31**	**1.25**	-0.81	**1.02**	**1.72**	**0.88**	**1.00**	-1.22
COPD	**0.03**	**4.11**	**2.40**	**4.86**	-0.69	-2.43	**0.18**	-2.21	**4.61**	**1.38**
Norway										
Smoking-related diseases	*-1.09*	***2.15***	***4.62***	***2.38***	***2.06***	*-3.19*	***0.26***	***1.62***	***1.26***	***3.34***
Lung cancer	**8.20**	**5.76**	**8.22**	**4.24**	**1.61**	**3.23**	**3.27**	**2.55**	**4.81**	**3.39**
Other smoking-related cancers	**0.31**	**2.89**	**1.65**	**1.34**	-0.22	-0.01	**2.64**	**2.05**	**0.17**	**0.19**
COPD	-3.93	-0.07	**7.04**	**2.39**	**4.13**	-5.73	-3.10	**0.57**	**1.95**	**6.87**
Sweden										
Smoking-related diseases	***4.49***	***5.18***	***3.61***	*-1.18*	***0.59***	***3.20***	***3.62***	***0.06***	*-1.19*	***2.08***
Lung cancer	**9.06**	**9.33**	**6.52**	-1.81	-1.05	**4.99**	**5.42**	**2.75**	-1.23	**2.93**
Other smoking-related cancers	**3.71**	**3.72**	**1.62**	-2.38	**0.42**	**4.46**	**3.37**	**0.06**	-1.43	-0.64
COPD	**4.26**	**5.38**	**4.16**	**0.51**	**1.64**	**0.69**	**3.40**	-1.32	-0.54	**5.55**

Bold indicates an increase.
COPD = chronic obstructive pulmonary disease.

Due to this overall increase these diseases had an increased share in total mortality, and thus their trends had increasingly more effect on the trends observed for all-cause mortality. Since the 1980s, mortality from smoking-related diseases among men decreased in Finland and France, whereas in Denmark, The Netherlands, and Norway in the 1980s mortality still increased rapidly. However, only Norway showed a clear persistence of the increase among males in the 1990s. For females, recent increases were more frequent, and

were most pronounced in Norway, Denmark, and The Netherlands. Overall, lung cancer revealed the strongest increases, but COPD showed a more unfavorable trend in the 1990s.

Mortality from cardiovascular diseases declined, at least after the 1960s (Table 4). During the last 2 decades, declines were largest in France and England and Wales, and smallest in Norway and The Netherlands.

Table 4. Annual trends in mortality from cardiovascular diseases by country and decade (1950-1999), males and females, aged 80 and over

	Annual changes (%)									
	Males					Females				
	1950s	1960s	1970s	1980s	1990s	1950s	1960s	1970s	1980s	1990s
Denmark										
All cardiovascular diseases	***0.86***	*-0.44*	*-1.66*	*-0.79*	*-2.60*	*-0.06*	*-1.58*	*-2.58*	*-0.75*	*-3.43*
Ischemic heart disease	**5.17**	**3.60**	-1.32	-1.71	-5.03	**4.03**	**2.81**	-2.29	-1.87	-6.03
Cerebrovascular diseases	**0.14**	-2.93	-3.02	-0.25	-0.98	-0.37	-3.53	-3.20	-0.13	-1.65
Other cardiovascular diseases	-2.19	-6.60	-1.83	**1.93**	**1.25**	-2.56	-7.11	-3.14	**1.72**	-0.53
England and Wales										
All cardiovascular diseases	*-1.05*	*-1.30*	*-1.70*	*-2.03*	*-2.36*	*-0.98*	*-1.52*	*-1.94*	*-2.67*	*-2.60*
Ischemic heart disease	**0.92**	**0.15**	-0.27	-0.78	-2.67	**0.83**	-0.26	-0.98	-0.94	-3.08
Cerebrovascular diseases	**0.99**	-0.59	-3.11	-2.24	-2.10	**0.99**	-0.28	-2.51	-2.81	-1.78
Other cardiovascular diseases	-3.80	-3.70	-2.65	-5.87	**0.09**	-3.29	-3.84	-2.45	-6.48	-0.80
Finland										
All cardiovascular diseases	***2.80***	***0.17***	*-3.05*	*-1.41*	*-1.79*	***3.65***	*-0.53*	*-4.05*	*-1.42*	*-2.36*
Ischemic heart disease	**5.57**	**2.20**	-1.21	**0.84**	-0.77	**6.08**	**0.98**	-1.46	**1.09**	-0.89
Cerebrovascular diseases	**2.19**	-0.70	-4.18	-1.68	-1.98	**1.71**	-1.68	-4.57	-1.07	-2.73
Other cardiovascular diseases	**1.36**	-1.04	-4.41	-5.83	-5.20	**3.99**	-0.61	-5.86	-5.52	-5.68
France										
All cardiovascular diseases	***0.15***	*-0.29*	*-0.99*	*-2.93*	*-1.98*	***0.13***	*-0.45*	*-0.69*	*-3.07*	*-2.33*
Ischemic heart disease	**6.50**	**3.13**	**2.90**	-0.24	-1.71	**6.84**	**3.45**	**3.48**	-0.50	-2.72
Cerebrovascular diseases	**0.41**	**0.50**	-1.77	-4.97	-4.25	**0.52**	**0.31**	-0.87	-4.79	-4.35
Other cardiovascular diseases	-0.87	-1.69	-1.77	-2.89	-0.80	-0.79	-1.72	-1.80	-2.90	-0.93
The Netherlands										
All cardiovascular diseases	***0.51***	*-1.09*	*-1.46*	*-1.17*	*-1.40*	***0.15***	*-1.71*	*-2.81*	*-1.69*	*-1.85*
Ischemic heart disease	**3.85**	**1.32**	-0.32	-1.41	-2.41	**3.19**	**0.32**	-0.95	-2.18	-3.02
Cerebrovascular diseases	**0.81**	-1.86	-2.78	-0.99	-1.76	**0.53**	-2.35	-3.62	-0.90	-1.70
Other cardiovascular diseases	-1.62	-3.04	-0.94	-0.75	-0.27	-1.53	-2.97	-2.77	-1.53	-1.19
Norway										
All cardiovascular diseases	***2.46***	***0.21***	*-1.46*	*-0.43*	*-0.92*	***1.92***	*-1.00*	*-1.95*	*-1.39*	*-0.81*
Ischemic heart disease	**9.63**	**2.01**	-1.61	**0.16**	-1.88	**10.09**	**0.97**	-2.59	-0.74	-2.07
Cerebrovascular diseases	**2.66**	-0.20	-3.08	-1.55	-1.68	**2.64**	-1.13	-2.99	-1.78	-2.21
Other cardiovascular diseases	-4.07	-3.09	**1.63**	-0.23	**1.55**	-4.05	-4.10	**0.78**	-1.82	**2.35**
Sweden										
All cardiovascular diseases	***1.01***	*-0.82*	*-0.23*	*-1.76*	*-1.74*	***0.73***	*-1.78*	*-1.32*	*-2.04*	*-2.03*
Ischemic heart disease	**3.18**	**2.28**	-0.63	-3.97	-2.41	**2.11**	**1.51**	-1.63	-4.95	-2.70
Cerebrovascular diseases	**0.58**	-3.72	-1.20	-0.83	-0.31	**0.41**	-3.62	-1.44	-0.87	-1.13
Other cardiovascular diseases	-1.31	-4.10	**0.02**	-0.84	-1.30	-0.79	-4.27	-1.32	-0.94	-1.62

Bold indicates an increase.

Trends in mortality from ischemic heart disease (IHD) were least favorable with increases in mortality in the 1950s and 1960s. Mortality from IHD also increased in the 1980s in Norway (males) and Finland. Mortality from cerebrovascular diseases increased in the 1950s, followed by a sustained decrease in mortality. Striking were the enormous decreases in mortality from stroke in France since the 1980s. For both IHD and stroke, the mortality decrease in France sets in later compared to the other countries. Mortality from "other cardiovascular diseases" decreased, especially in Finland and England and Wales, whereas in Denmark and Norway mortality increased since the 1980s.

For the "other causes of death", that is, total mortality minus "smoking-related diseases" and all cardiovascular diseases, mortality increased since the 1980s in all countries, except France (Table 5). The increases are most pronounced in Denmark and The Netherlands, in the 1990s. Especially mortality from diseases specifically related to old age (infectious diseases, pneumonia, dementia, and ill-defined conditions) increased substantially. Differences between the countries, however, exist in which of these causes showed the largest mortality increases. In France, the increase in diseases specifically related to old age has been offset by the continuation of the strong decline for "other diseases". Caution, however, should be exercised when interpreting the annual changes for causes of death with a low mortality level in 1980 and 1990, especially infectious diseases, diabetes mellitus, dementia (males), and all ill-defined causes (in England and Wales, Finland, and Sweden). Annual changes for such causes tend to be large, even though the change is small in absolute terms.

DISCUSSION

This article provides important new findings on the trends in old-age mortality (80+) in seven low-mortality countries. First, although old-age mortality tends to decline and to converge, there is large heterogeneity in the pace of decline in the countries under investigation, with periods of stagnation being widespread. Since the 1980s, stagnation was observed in Denmark, The Netherlands, and Norway, whereas England and Wales and especially France showed a continuation of a strong mortality decline. Second, the long-term mortality increase for smoking-related cancers and COPD turned into declines since the 1980s among males. Only for Norway the mortality increase among males clearly persisted. Third, mortality from cardiovascular diseases showed clear crossnational variations in the general decline since the 1970s. Fourth, mortality from diseases specifically related to old age (infectious diseases, pneumonia, dementia, and ill-defined conditions) increased recently in all countries except France.

Previous studies on trends in old-age mortality also report some heterogeneity in the speed of the mortality decline, but found only rare periods of stagnation [2,15]. The overall conclusion reached in most of these studies therefore has been a continued decline in old-age mortality [2,4,15,24,25]. The present study, however, shows that periods of stagnation are more frequent than previously observed. This conclusion could be reached by, on the one hand, extending the observation period to the 1990s, and on the other hand, by looking in more detail at the trends within specific decades in stead of broader periods. Our results thus show a more differentiated picture than suggested by previous analyses on old-age mortality.

The heterogeneity between countries and periods as observed in this analysis and in previous analyses suggests that the findings obtained in this study may not be considered to be representative of other countries nor of earlier periods.

Table 5. Annual trends in mortality from "other causes of death" by country and decade (1980-1999), males and females, aged 80 and over

	Annual changes (%)					Annual changes (%)			
	Males		Females			Males		Females	
	1980s	1990s	1980s	1990s		1980s	1990s	1980s	1990s
Denmark					**The Netherlands**				
Other causes of death	***0.24***	***2.94***	***0.05***	***3.01***	*Other causes of death*	***1.13***	***1.36***	***0.83***	***1.72***
Other cancers	**0.02**	**0.87**	-0.96	**0.13**	Other cancers	**0.80**	**0.00**	-0.32	-0.76
Infectious diseases	**0.80**	**6.26**	**1.84**	**14.25**	Infectious diseases	**2.97**	**6.32**	**4.81**	**5.31**
Diabetes mellitus	**1.56**	**1.99**	-1.47	**1.29**	Diabetes mellitus	**0.21**	-2.38	-1.39	-2.79
Pneumonia	-6.31	**10.06**	-6.46	**7.64**	Pneumonia	**0.07**	**5.28**	-0.89	**4.69**
Dementia	**9.30**	**10.07**	**7.41**	**7.62**	Dementia	**14.14**	**15.63**	**15.69**	**15.24**
All ill-defined causes	**3.52**	**12.65**	**4.04**	**13.73**	All ill-defined causes	**2.19**	**3.20**	**0.80**	**4.17**
Other diseases	-0.17	**3.22**	-0.04	**4.36**	Other diseases	**1.79**	-1.92	**2.02**	-1.67
All external causes	**3.48**	-0.79	**2.52**	-2.13	All external causes	-1.17	-2.57	-3.78	-2.17
England and Wales					**Norway**				
Other causes of death	*-1.29*	***1.31***	*-1.18*	***1.56***	*Other causes of death*	***0.22***	*-0.55*	***0.12***	*-0.91*
Other cancers	**2.64**	-1.05	**1.06**	-1.89	Other cancers	**0.18**	**0.78**	-1.02	**0.31**
Infectious diseases	**1.31**	**3.04**	**3.35**	**2.91**	Infectious diseases	-0.48	**7.19**	**0.34**	**7.89**
Diabetes mellitus	**2.83**	**0.39**	**0.98**	**0.64**	Diabetes mellitus	**1.90**	-0.44	**1.70**	-3.24
Pneumonia	-3.51	-0.75	-2.65	-1.21	Pneumonia	-0.90	**0.74**	-0.70	-0.89
Dementia	**3.90**	**5.26**	**2.75**	**5.91**	Dementia	**4.15**	**1.63**	**4.91**	**1.20**
All ill-defined causes	**8.97**	**14.29**	**9.81**	**15.14**	All ill-defined causes	-3.14	**1.34**	-1.28	**2.29**
Other diseases	-0.72	**1.03**	-0.55	**1.55**	Other diseases	**0.94**	-0.22	**1.46**	**0.04**
All external causes	-3.07	-0.79	-5.36	-0.07	All external causes	**0.91**	-1.12	-2.62	-1.70
Finland					**Sweden**				
Other causes of death	***1.31***	*-0.07*	***1.73***	*-0.32*	*Other causes of death*	***0.61***	***0.01***	***0.79***	***0.36***
Other cancers	**0.04**	-0.30	-0.71	-1.60	Other cancers	-1.66	**1.44**	-2.13	**0.50**
Infectious diseases	-4.46	**0.12**	-2.74	-1.53	Infectious diseases	-2.39	**6.08**	**2.65**	**4.14**
Diabetes mellitus	-3.89	**0.07**	-3.96	-3.26	Diabetes mellitus	**3.64**	-0.22	**1.71**	-0.90
Pneumonia	-1.19	-0.44	-1.27	-0.81	Pneumonia	**2.19**	-4.70	**2.05**	-5.04
Dementia	**17.64**	**0.03**	**14.57**	-0.16	Dementia	**12.08**	**6.01**	**12.92**	**5.26**
All ill-defined causes	**0.03**	-12.09	**2.41**	-12.24	All ill-defined causes	**12.62**	-1.98	**13.06**	**1.87**
Other diseases	**2.28**	**0.67**	**3.05**	**0.64**	Other diseases	**0.64**	**0.33**	**0.90**	**2.24**
All external causes	**1.82**	-0.99	**0.18**	-0.82	All external causes	**0.62**	-0.84	-0.77	-2.26
France									
Other causes of death	*-1.16*	*-1.17*	*-1.61*	*-1.12*					
Other cancers	**0.73**	-0.96	-0.44	-1.00					
Infectious diseases	-0.07	**0.03**	**0.88**	-0.26					
Diabetes mellitus	-1.13	**0.07**	-1.94	-0.71					
Pneumonia	**0.84**	**1.38**	-0.15	**1.26**					
Dementia	**8.06**	-1.82	**7.44**	-1.14					
All ill-defined causes	-3.27	-3.06	-3.08	-2.23					
Other diseases	-3.63	-0.86	-3.01	-0.14					
All external causes	-1.16	-2.26	-3.16	-3.83					

Bold indicates an increase.

Evaluation of data and methods

The mortality and population data used in this study stem from countries considered to have good or excellent population and vital registries [1,2]. Reported survivorship counts are highly accurate [2,26]. Comparison of our mortality data to the mortality data from the Kannisto-Thatcher Database—in which the data was checked for age-heaping and were subjected to a number of checks for plausibility— showed only small discrepancies, that had no appreciable effects on our results.

Analyses of long-term trends in causes of death have to deal with several transitions between ICD revisions. Considerable effort was made to bridge these ICD revisions by carefully constructing a concordance table and by controlling for the remaining biases due to these revisions in our regression model. Further checks showed minimal sensitivity. Thus, even though some residual effects of problems with ICD revisions could not be excluded, we expect that these problems do not affect the results to any substantial extent.

More difficult to tackle were changes within an ICD revision, either in coding practices at the Statistical Offices or in the reporting of causes of death by physicians. Although all countries under investigation use the coding rules of the WHO, the national statistical offices might change, over time, their interpretation of these international coding rules. Because changes in coding practices most probably led to quite abrupt and sometimes temporal changes, the changes could be traced and controlled for. Changes in the reporting of causes of death on the death certificate by physicians, however, result in more gradual shifts that could not be controlled for. The causes of death expected to suffer substantial effects are diabetes mellitus, dementia, and "all ill-defined causes". The huge increases for dementia that were observed especially in the 1980s could partly be explained by a growing propensity of physicians to report dementia as an underlying cause of death. The recent increases in "all ill-defined causes" (especially in England and Wales, Denmark, Sweden, and The Netherlands) could be the result of less detailed and less accurate diagnosis and reporting by physicians. An increase in the doctor's tendency to list only one cause on the death certificate could perhaps explain the huge increases observed for pneumonia in The Netherlands and Denmark in the 1990s, because diseases that were formerly reported mainly as secondary causes of death could increasingly be listed as primary causes of death. The more stable trends for diabetes mellitus seem to be less affected by this coding problem.

Thus, especially the recent increases found for dementia, "all ill-defined causes" and pneumonia should be viewed with caution. Moreover, one should be aware of the opposite effects these increases in mortality might have on other diseases. For example, an increasing tendency to report these diseases as the underlying cause of death may result in an overestimation of the recent decreases observed for cardiovascular diseases. However, this cannot account for the recent declines observed for cardiovascular diseases, because in absolute terms, the decline in cardiovascular diseases was much larger than the increases for diseases prone to changes in reporting causes of death (data not shown). Diseases that have a more straightforward diagnosis, like smoking-related cancers and likely COPD as well, are probably relatively resistant to changes in coding practices.

Explanations of the trends observed

Our findings suggest both favorable trends in old-age mortality (e.g., the continued decline in England and Wales, and especially France, and the huge decline in Finland in the 1970s), but also unfavorable trends (e.g., stagnation since the 1980s in Denmark, The Netherlands, and to a lesser extent Norway). In this section, we will discuss possible explanations of these trends.

Although many smokers die already before the age of 80, our results indicate that smoking is a possible determinant of mortality trends even among the oldest old. Smoking-related cancers and COPD contributed to the recent stagnation, especially in Norway, for which mortality from these causes of death increased recently. The absence of clear increases in Denmark and The Netherlands in the 1990s suggests that the contribution of smoking-related diseases to the recent stagnation as observed in The Netherlands and Denmark was more modest. However, by looking merely at the trends in these smoking-related diseases, other diseases, of which smoking is a risk factor as well, would be neglected. The recent declines observed for cardiovascular diseases—that are not correlated with the trends for lung cancer—suggest that smoking did not contribute much to the recent stagnation as observed in The Netherlands and Denmark. On the contrary, the significant decline in smoking prevalence among elderly men in recent periods [27], suggests a favorable effect of smoking on recent trends in mortality from cardiovascular diseases (and other causes of death), for which current smoking levels are more important than life-time smoking exposures [28]. Thus, although smoking has had a marked influence on the trends in old-age mortality, the role of smoking in the stagnation since the 1980s seems only modest and restricted to Norway.

Another explanation for the recent stagnation in Denmark, The Netherlands, and Norway could be that further improvement in old-age mortality in these countries is no longer possible, due to low levels of mortality already attained and a limit to life expectancy being approached. However, we observed further improvements in old-age mortality in both England and Wales and France, who had reached the same low level of mortality as Denmark, The Netherlands, and Norway around the 1980s. Life expectancy at age 80 in 1980–1990 was slightly higher in France (7.52) compared to Denmark (7.43) and Norway (7.49), suggesting that the limit to life expectancy is not yet being approached in the latter countries [29].

Mortality trends were unfavorable for infectious diseases, pneumonia, dementia, and ill-defined causes of death. The recent increase of mortality from these causes of death—which were not only restricted to Denmark and The Netherlands but appeared in all countries except France—could be partly artificial due to changes in the coding and reporting of causes of death (as discussed above). However, part of the recent increase in these causes, which are specifically related to old age, might be real, and perhaps due to increased frailty. Increased comorbidity or increased frailty among elderly in itself could easily lead to an in-creased occurrence of symptoms or comorbid conditions as causes of death. Increased frailty could be due to decreased mortality selection as a result of the declines in all-cause mortality and cardiovascular mortality in earlier periods and ages, and indirectly due to improvements in medical care. The increasing proportions of elderly people surviving to old ages might be expected to be less healthy compared to their more selected predecessors [30], resulting in increases in (cardiovascular) morbidity [31] and in mortality increases from diseases specifically related to old age. Although recent studies on

disability among the elderly suggest that disability among the elderly is declining [32–34], we still think that increased frailty might have played a role in explaining the recent increases in diseases specifically related to old age.

Another important finding from our study is the crossnational variation in the pace of decline in cardiovascular disease, which is still the most important cause of death among the elderly. In the last 2 decades, declines for cardiovascular disease mortality were highest in England and Wales and France and lowest in Norway, The Netherlands, and Denmark (1980s only). Previous research on trends in cardiovascular mortality [35–37] suggested the importance of several factors like physical activity, hypertension control, diet, smoking, and accessibility of medical care. It might be possible that the developments in some of these risk factors were more beneficial in England and Wales and France compared to the other countries.

Strikingly favorable were the trends in old-age mortality in France and Finland (1970s). France showed the strongest decline for cardiovascular diseases, and, contrary to the other countries, did not witness an increase from "other causes of death". Within "other causes of death" the declining trend for "other diseases" is striking. The latter trend seems the result of the reported declines of acute respiratory diseases and digestive diseases among the French elderly [38]. Changes in alcohol consumption might be one of the factors involved. The sharp decline in all-cause mortality in the 1970s in Finland was also observed among those aged 60 and over [39], and seems the result of rapid economic and social progress in this period [39–41]. Together with the development of a national health and social care system, this has resulted in improvements in the living conditions and health care, also for the elderly [41]. In addition, a favorable effect of rapid declines in behavioral risk factors, like diet, blood pressure, and serum cholesterol levels, in part caused by preventive campaigns, can be expected [42]. In Finland, old-age mortality thus seems to have been highly susceptible to improvements in living conditions and in both preventive and secondary health care.

Implications

Our finding that periods of stagnation were widespread both in the 1950s and since the 1980s, challenges the idea that mortality at old age is bound to decline steadily in the near future [6,8,11]. Old-age mortality seems highly plastic and susceptible to many factors, both favorable and unfavorable. These factors should be taken into account when making projections of future old-age mortality and its implications on social and health care policies.

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Appendix I. The concordance table used for bridging five revisions of the International Classification of Diseases (ICD)

Cause of death	ICD6/7	ICD8	ICD9	ICD10
1. Infectious and parasitic diseases	001-138	000-136	001-139	A00-B99
2. Cancer of the oesophagus	150	150	150	C15
3. Cancer of the stomach	151	151	151	C16
4. Cancer of the colorectum	153-154	153-154	153-154	C18-C21
5. Cancer of the pancreas	157	157	157	C25
6. Cancer of the upper respiratory/ digestive system	140-148, 160, 161	140-149, 160, 161	140-149, 160, 161	C00-C14, C30-C32
7. Cancer of the lung	162-163	162	162	C33-C34
8. Cancer of the breast	170	174	174-175	C50
9. Cancer of the prostate	177	185	185	C61
10. Cancer of the bladder	181	188	188	C67
11. Cancer of the kidney	180	189	189	C64-C66, C68
12. Cancers, unspecified	198-199, 230-239	195-199, 230-239	195-199, 235-239	C76-C80, C97, D37-D48
13. Other cancers	Rest (140-239, 294)	Rest (140-239)	Rest (140-239)	Rest (C00-D48)
14. Diabetes mellitus	260	250	250	E10-E14
15. Dementia and Alzheimer's	304-306	290, 293	290, 331	F00, F01, F03, G30
16. Ischemic heart disease	420, 422.1	410-414	410-414	I20-I25
17. Other heart diseases	400-402, 410-416, 421, rest (422), 430-434, 440-447	390-398, 400-404, 420-425, 427-429	390-398, 401-405, 416, 420-429	I00-I13, I15, I27, I30-I52
18. Cerebrovascular diseases	330-334	430-434, 436-438	430-434, 436-438	I60-I69
19. Other circulatory diseases	Rest (400-468)	Rest (390-458, excl 435 & 446)	Rest (390-459, excl 435 & 446)	Rest (I00-I99)
20. Pneumonia/influenza	480-483, 490-493	470-474, 480-483, 485-486	480-487	J10-J18
21. COPD	501, 502, 526, 527, 241	490-493, 518	490-494, 496	J40-J47
22. Senility	794	794	797	R54
23. Other symptoms and ill-defined conditions	Rest (780-795)	Rest (780-796)	Rest (780-799)	Rest (R00-R99)
24. Other diseases	Rest (001-795)	Rest (000-796)	Rest (001-799)	Rest (A00-R99)
25. Accidental fall	E900-904	E880-887	E880-888	W00-W19, X59
26. Other external causes	Rest (E800-999)	Rest (E800-999)	Rest (E800-999)	Rest (V01-Y98)

5

COHORT PATTERNS IN MORTALITY TRENDS AMONG THE ELDERLY IN SEVEN EUROPEAN COUNTRIES, 1950-1999

Objective Secular trends in old-age mortality are of crucial importance to population ageing. For the understanding and prediction of these trends, it is important to determine whether birth cohort effects, i.e. long-lasting effects of exposures earlier in life, are important in determining mortality trends up to old age. This study aimed to identify and describe cohort patterns in trends in mortality among the elderly (>60 years of age) in seven European countries. **Methods** A standard age-period-cohort analysis was applied to all-cause and cause-specific mortality data by 5-year age groups and sex, for Denmark, England and Wales, Finland, France, The Netherlands, Norway and Sweden, in the period 1950-1999. **Results** Cohort patterns were identified in all countries, for both the sexes and virtually all causes of death. They strongly influenced the trends in all-cause mortality among Danish, Dutch, and Norwegian men, and the trends in mortality from infectious diseases, lung cancer (men only), prostate cancer, breast cancer, and chronic obstructive pulmonary disease (COPD). All-cause mortality decline stagnated among Danish, Dutch and Norwegian male birth cohorts born between 1890 and 1915, among French men born after 1920, and among women from all countries born after 1920. Where all-cause mortality decline stagnated, cohort patterns in mortality from lung cancer, COPD, and to a lesser extent ischaemic heart diseases, were unfavourable as well. For infectious diseases, stomach cancer, and cerebrovascular diseases, mortality increased among cohorts born before 1890, and decreased strongly thereafter. **Conclusion** Cohort effects related to factors such as living conditions in childhood and smoking in adulthood were important in determining the recent trends in mortality among the elderly in seven European countries.

BACKGROUND

With the increase in life expectancy there has been a shift in the trend from dying at younger ages to dying at older ages. Consequently, the mortality trends among the elderly gained more and more importance in determining the mortality patterns and the extent of ageing of national populations [1], with its far-reaching consequences to the individual, society, and health care policies. Therefore, it is crucial to carefully study old-age mortality trends and their determinants.

Trends in mortality among the elderly in Europe have been characterized by an overall decline since the 1950s [2-5]. However, a recent study on old-age mortality trends from 1950-1999 in seven European countries showed that the pace of decline varied strongly by country and over time [6]. In Denmark, The Netherlands, and for Norwegian men, mortality declines even stagnated during the 1980s and 1990s [6].

In order to identify the determinants of national trends in mortality, it is essential to distinguish between period and cohort patterns. Whereas period patterns indicate immediate effects of conditions occurring in late life, cohort patterns may reflect long-lasting effects of determinants located earlier in the life course, e.g. in infancy, childhood,

or adulthood. This distinction is of crucial importance in making projections of future developments in mortality. If cohort effects prove to be important, cohort-wise projections may be used to explore the possible future mortality trends.

Interest in cohort effects dates back to the influential works by Kermack et al. in 1934 [7] and by Case and MacMahon et al. in the 1950s [8,9]. Since the 1980s, there has been a renewed interest in cohort effects. In epidemiology, this interest was stimulated by results from the new studies on the effects of living conditions in early life on health in later life [10,11]. At the same time, a number of demographic studies reported on the debilitating effects of war and famine during early ages on mortality in adulthood [12-14]. Despite this revival of interest, it remains unknown whether cohort effects are important in determining the recent trends in mortality of national populations, partly because the identification of cohort effects is complicated by the generally linear nature of the mortality decrease in Europe since the turn of the century [15]. Moreover, it is as yet not known whether cohort effects also extend into old age.

Our hypothesis is that cohort effects are important factors in determining old-age mortality trends, and that these effects are especially visible in mortality from causes of death that are known to be related to smoking or to early life circumstances. In addition, we expect that the importance and patterns of cohort effects might differ between countries, due to, for example, differences in the development of the smoking epidemic among adults [16].

In this paper, we aimed to identify and describe cohort patterns in all-cause and cause-specific mortality trends among the elderly populations of seven European countries. Using data on old-age mortality trends during the second half of the twentieth century, we assessed for each country individually (i) whether cohort patterns are strong enough to determine trends in all-cause and cause-specific mortality, and (ii) the timing of these cohort patterns.

In our study, we included both all-cause mortality and cause-specific mortality for seven European countries. Previous studies on cohort patterns in causes of death did not always compare the results for different causes of death or different countries. Furthermore, the comparison of the results between different studies is complicated by the application of different techniques to identify the importance of cohort effects. Our approach enabled us to observe not only commonalities but also dissimilarities in the importance and patterns of cohort effects.

DATA AND METHODS

Data

Data on total mortality, the underlying cause of death and population at risk, by year of death (1950-99), sex, and 5-year age groups were obtained for Denmark, England and Wales, Finland, France, The Netherlands, Norway, and Sweden, from national statistical offices and related institutes. For Denmark, Finland, and Norway, data were available from 1951, and for Sweden from 1952. For France data until 1997 were available and for Denmark until 1998. Data on total mortality and population for the highest age groups (80-100+, by single year of age) were obtained from the Kannisto-Thatcher Database on Old

Age Mortality (http://www.demogr.mpg.de/databases/ktdb) [3]. See Janssen et al., 2004 for a more detailed description of the included data [6].

In our analysis, we included data for those aged ≥60 years in order to address the question whether the influence of cohort effects extends into old age. The study was not restricted to the very old in order to include sufficient age groups to distinguish between period and cohort effects.

With regard to the causes of death, we selected infectious diseases (ICD-9 001-139), cancer of lung (ICD-9 162), cancer of stomach (ICD-9 151), cancer of prostate (ICD-9 185), cancer of breast (ICD-9 174-175), ischaemic heart disease (ICD-9 410-414), cerebrovascular diseases (ICD-9 430-434, 436-438), and chronic obstructive pulmonary diseases (ICD-9 490-494, 496). These are all causes of death occurring frequently among elderly. Together they accounted for on average 46 % of all deaths among those aged ≥60 years in the late 1990s (see Table 1).

To reconstruct mortality trends from these specific causes of death over the period 1950 to 1999, we had to bridge five different revisions of the International Classification of Diseases (ICD-6 to ICD-10), for all countries except France for which coherent series of causes of death were already available [17]. For this purpose, we constructed a general concordance table based on three-digit codes of the ICD, and the four digit code 422.1 for ischaemic heart diseases under ICD-6/ICD-7 (see Janssen et al., 2004) [18]. Remaining mortality discontinuities - caused by the use of three-digit instead of four-digit codes and by incidental changes in coding rules – were identified and adjusted for in our analysis. These discontinuities were identified by (i) visual analysis of cause-specific mortality trends, (ii) country-specific background information on these mortality discontinuities, and (iii) a regression-based method that quantified the size of the discontinuities (see Janssen et al., 2004) [18]. Mortality discontinuities that were regarded as owing to changes in coding rules were controlled for in our regression method, by means of cause- and sex-specific transition coefficients. These transition coefficients are the parameter estimates of variables associated with a coding change (e.g. ICD-8toICD-9) and obtained through sex-specific age-period regression models, applied to cause-specific mortality among those aged 60 and over [6,18]. Lung cancer and stomach cancer were not affected by coding changes. Infectious diseases showed a mortality discontinuity associated with the revision from ICD-6/7 to ICD-8 in five countries. For the remaining causes of death, adjustments had to be made in one or two countries, mainly for incidental coding changes, for example the generally applied coding change in England and Wales between 1984 and 1992 (see Janssen et al., 2004)[18].

Statistical analysis

For statistical analysis, we aggregated the data into 5-year age groups, 5-year periods and consequently 10-year overlapping cohort groups. To these data we applied log-linear age-period-cohort (APC) models through the GENMOD procedure of the SAS 8.0 package.

In APC analyses, it is impossible to identify the role of age, period, and cohort separately, owing to the interdependency between these variables. We dealt with this identification problem [19-21] by using the standard Clayton & Schifflers procedure.

Table 1. List of selected causes of death and their relative share in all-cause mortality in the period 1995-1999[a], males and females aged ≥60 years

	Relative share (%)													
	Males							Females						
	DK	E&W	FIN	F	NL	NO	S	DK	E&W	FIN	F	NL	NO	S
All causes (N)	97951	1134834	91963	642737	282455	95371	205139	108686	1334906	112878	694906	307813	101918	217462
All causes (rate x 1,000)	54.75	49.20	46.38	43.26	47.42	51.15	48.16	45.95	43.54	37.96	33.91	38.36	41.20	39.60
Cancer of lung	7.0	7.5	6.7	7.3	10.1	5.0	3.8	4.0	3.6	1.5	1.3	2.2	2.1	2.0
COPD	6.6	6.6	4.2	4.1	7.3	4.6	3.2	5.4	4.2	1.4	2.9	3.6	3.1	2.3
Ischaemic heart disease	21.8	26.5	31.3	10.6	16.9	24.2	28.0	18.7	20.4	27.4	9.0	13.1	18.9	22.4
Infectious diseases	0.5	0.5	0.7	1.5	0.9	0.8	0.8	0.6	0.5	0.8	1.6	1.0	1.1	1.0
Stomach cancer	0.9	1.6	1.5	1.4	1.6	1.6	1.2	0.6	0.9	1.1	0.9	1.0	1.0	0.8
Prostate cancer (m) / breast cancer (f)	4.0	3.7	3.9	4.2	4.1	5.7	5.7	3.6	3.2	2.3	3.5	4.0	2.7	2.5
Cerebrovascular diseases	8.3	8.8	9.5	7.5	7.8	10.3	9.9	10.8	13.2	13.7	10.5	11.6	14.0	13.3
Total share selected causes (%)	49.2	55.1	57.8	36.6	48.8	52.2	52.7	43.8	46.0	48.2	29.6	36.4	43.0	44.4

[a] Or last year available.

DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.

COPD = chronic obstructive pulmonary diseases.

Box 1

Model parameters	Statistical notation
Age (A)	$E[\ln Y_a] = \alpha_a$
Age + drift (AD)	$E[\ln Y_{ad}] = \alpha_a + \delta$
Age + period (AP)	$E[\ln Y_{ap}] = \alpha_a + \delta + \beta_p$
Age + period + cohort (APC)	$E[\ln Y_{apc}] = \alpha_a + \beta_p + \gamma_c$

E[lnY] is the expected value of the natural log of the mortality rate, with the number of deaths being Poisson distributed. α, δ, β, and γ are the age, drift, period, and cohort effect, respectively. The variables for age (a) and cohort (c) had one baseline class. For period (p) a second baseline class was chosen. c = A – a + p. The variables were indexed a = 1,2,…,7,8, p = 1,2,…,9,10, and c = 1,…,13.

With this procedure, mortality is decomposed in a common linear trend (drift), a non-linear period effect and a non-linear cohort effect [20,21]. See Box 1 for the different models that we fitted accordingly.

We measured the contribution of drift, the non-linear period effects, and the non-linear cohort effects in the mortality trends by assessing the reduction in scaled deviances (a measure of unexplained variance) when comparing the subsequent models (i) AD with A, (ii) AP with AD, and (iii) APC with AP, respectively. We expressed these reductions as percentages of the reduction in scaled deviance observed between the model with age only and the full APC model. These percentage reductions were used to measure the contribution of the different effects to systematic mortality trends, and to make comparisons of this contribution between countries and causes of death. The statistical significance of the difference in scaled deviances between the subsequent models was assessed by a log-likelihood ratio test, resulting in one-sided p-values.

In order to describe the timing of the identified non-linear cohort effects, we calculated cohort-specific mortality levels derived from the parameter estimates of the cohort variables out of the full age-period-cohort model. We thus controlled for age and period, and by choosing two baseline period groups (1950-54 and 1995-99) and only one baseline cohort group (1920-29), we included the drift parameter in the measurement of cohort differences in mortality. In our description of the cohort patterns we focused on the (cohort) deviations from linearity in the observed patterns. The cohort-specific mortality risks were calculated relative to the unweighted average of the mortality levels of all cohorts together. As not all birth cohorts have complete death counts for those aged ≥60 years (e.g. for those born in 1865 we have observations only for those aged ≥85 years, and those born in 1935 were aged 60-64 at the end of our observation period) we were cautious when describing the mortality levels of the extreme cohorts.

RESULTS

In all-cause mortality, drift (the linear component of both period and cohort effects) contributed to a large extent to the systematic trends, especially among elderly women (Figure 1). However, for men in Denmark, The Netherlands, and Norway, non-linear cohort effects and non-linear period effects contributed substantially to old-age mortality trends.

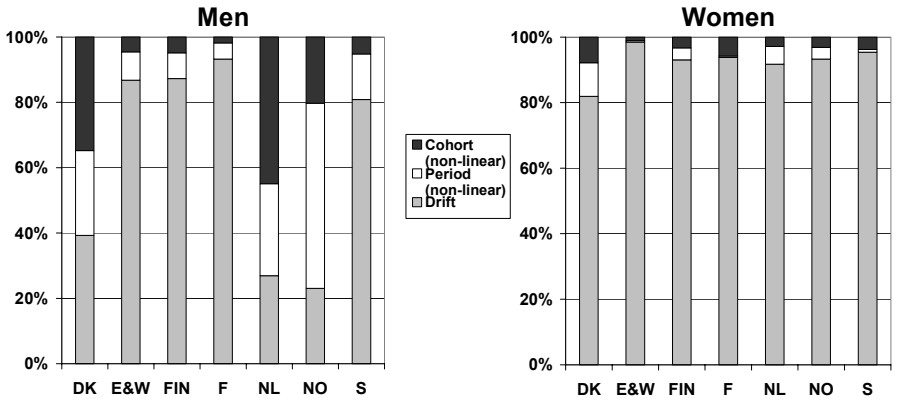

Figure 1. The contribution of drift, non-linear period effects and non-linear cohort effects to all-cause mortality trends, by sex and country

Drift = the reduction in scaled deviance by adding drift to the model including only age; Period (non-linear) = the reduction in scaled deviance by adding the non-linear period effect to the age-drift model; Cohort (non-linear) = the reduction in scaled deviance by adding the non-linear cohort effect to the age-period model. We measured the reduction in scaled deviance relative to the total reduction in scaled deviance of the full age-drift-period-cohort model compared with the model including only age. All effects are significant at the p = 0.05 level (one-tailed). DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.

For all selected causes of death, the contribution of the non-linear cohort effect to the systematic trends was statistically significant in each country and for both sexes, except for mortality from stomach cancer among Norwegian women (Table 2). Non-linear cohort effects contributed substantially to the mortality trends in the majority of countries for infectious diseases, lung cancer (men only), prostate cancer, cancer of breast, and chronic obstructive pulmonary diseases (COPD). For Finland and France, the non-linear cohort effects also contributed considerably to the mortality trends for ischaemic heart diseases (IHD).

Turning to the timing of the identified cohort effects (including drift), all-cause mortality generally declined among subsequent cohorts (Figure 2). For men, the decline stagnated among Danish, Dutch and Norwegian birth cohorts born between 1890 and 1915, and among French generations born after 1920. For women, the decline in all-cause mortality accelerated among cohorts born after 1880, but stagnated among the cohorts from 1920 onwards. This stagnation was most pronounced in Denmark and did not occur in England and Wales or Finland.

The increase in mortality from lung cancer among men accelerated among cohorts born from 1880 onwards. This increase levelled off again among those born after 1895, except for Norway and France, and resulted in a decrease for The Netherlands, England and Wales and Finland later on. For women, the increase in lung cancer mortality accelerated from 1915 onwards in The Netherlands, Denmark, Norway and Sweden, while this mortality increase levelled off among women in the other countries.

Table 2. The contribution of non-linear cohort effects to all-cause and cause-specific mortality trends, by sex and country

	Share of scaled deviance[a] explained by adding the non-linear cohort effect to the age-period model						
	DK	E&W	FIN	F	NL	NO	S
Men							
All-cause mortality	34.8	4.6	4.8	1.8	44.9	20.3	5.2
Cancer of lung	12.2	44.1	41.2	6.4	21.0	7.1	18.5
COPD	7.3	31.2	20.2	73.5	40.4	17.3	15.5
Ischaemic heart disease	1.9	8.5	28.7	36.6	8.5	7.4	1.0
Infectious diseases	9.0	13.4	10.8	43.4	19.5	23.6	35.0
Cancer of stomach	1.1	6.7	4.0	5.5	0.4	2.0	1.3
Cancer of prostate	4.3	11.4	24.8	28.3	15.6	18.3	10.9
Cerebrovascular diseases	0.9	4.1	1.9	7.2	1.6	4.1	4.2
Women							
All-cause mortality	7.8	1.1	3.3	5.8	2.9	3.1	3.8
Cancer of lung	3.6	3.3	8.9	7.5	9.7	2.3	9.0
COPD	13.9	22.6	43.0	64.7	6.8	16.8	20.1
Ischaemic heart disease	5.1	3.3	43.5	59.5	1.3	6.1	1.4
Infectious diseases	6.8	18.0	17.1	62.0	14.0	19.7	40.9
Cancer of stomach	1.1	1.7	3.1	6.5	0.9	(0.4)	1.0
Cancer of breast	46.5	11.0	11.8	5.4	33.1	31.3	16.7
Cerebrovascular diseases	5.2	8.3	6.0	18.1	6.0	10.3	14.2

[a] The scaled deviance that is explained by the full age-period-cohort model as compared with the model including only age.
DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.
( ) No statistically significant reduction in the scaled deviance ($p < 0.05$ one sided).
COPD = chronic obstructive pulmonary diseases.

For men in The Netherlands, Norway, Sweden and Denmark, the cohort patterns for COPD resembled that for lung cancer, i.e. initial modest increases accelerated among the birth cohorts from 1880 onwards, and decelerated later on. The onset of mortality decline was later as compared with lung cancer. For men in England and Wales, France and Finland, the increase in COPD mortality was initially strong, but reversed into mortality decline from birth cohorts 1895 onwards. For women, the cohort trends in COPD mortality were heterogeneous and erratic until birth cohort 1900. From cohort 1905 onwards, mortality generally increased, especially in Denmark.

For France, Finland, and Norway, IHD mortality increased for both men and women up to cohorts 1900-15, and declined thereafter. In Denmark, mortality increased until birth cohort 1880, but declined thereafter, especially among women. IHD mortality in Sweden, England and Wales, and The Netherlands declined among all birth cohorts, although this decline stagnated among male birth cohorts from 1890 onwards, especially among Dutch men.

For infectious diseases, stomach cancer, and cerebrovascular diseases, mortality generally declined after an initial increase among cohorts born in the nineteenth century (Figure 3).

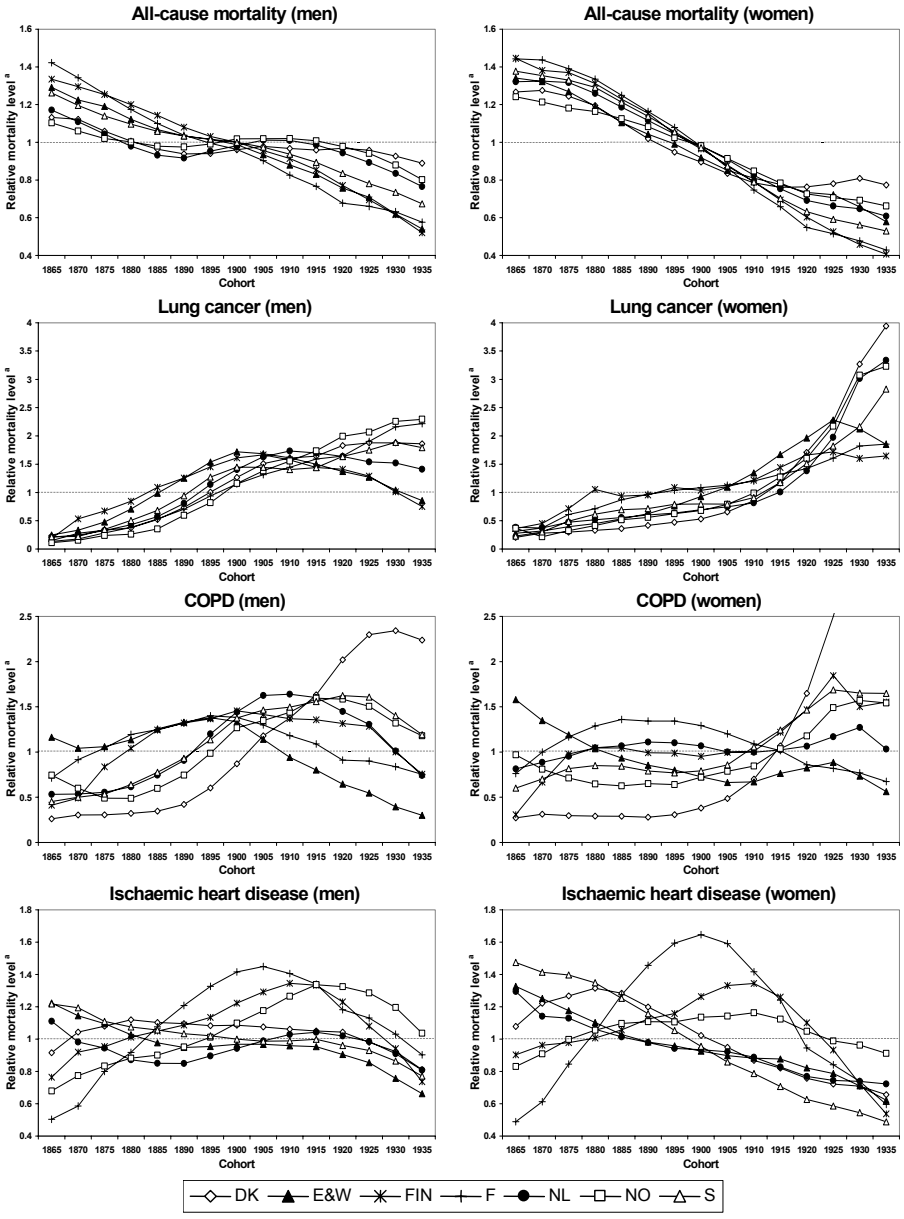

Figure 2. Cohort trends (including drift) for all-cause mortality, lung cancer, COPD, and ischaemic heart disease, by country and sex, for those aged ≥60 years

[a] Mortality level of each individual cohort relative to the unweighted average of the mortality levels of all cohorts together.

COPD = chronic obstructive pulmonary diseases. DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.

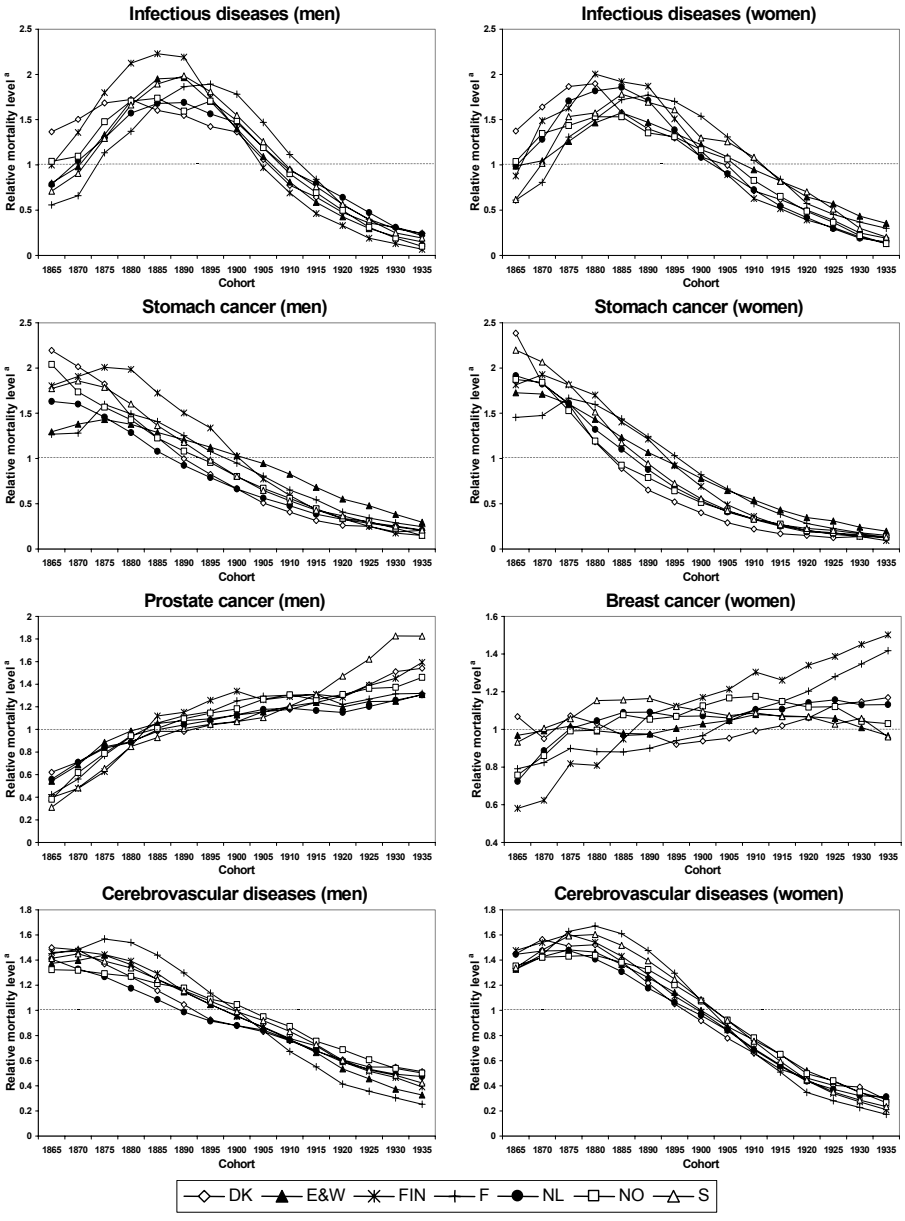

Figure 3. Cohort trends (including drift) for infectious diseases, stomach cancer, prostate cancer, breast cancer, and cerebrovascular diseases, by country and sex, for those aged ≥60 years

[a] Mortality level of each individual cohort relative to the unweighted average of the mortality levels of all cohorts together.

DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.

This initial mortality increase was most pronounced for infectious diseases, for which the increase persisted until cohorts born around 1890 (men) and 1885 (women). Stomach cancer increased only among the earliest birth cohorts of Finland, France, England and Wales (men), and Sweden (men). For cerebrovascular diseases, mortality increased among cohorts born before 1880 (men) and before 1885 (women). This initial increase was stronger for women.

Mortality from prostate cancer and breast cancer generally increased among subsequent cohorts, with only small deviations from the linear trend. For prostate cancer, mortality increases were strongest among the earlier birth cohorts, especially in Sweden.

DISCUSSION

Cohort patterns were identified in all countries, for both the sexes and virtually all causes of death. Non-linear trends in mortality among subsequent birth cohorts contributed substantially to the trends in all-cause mortality among Danish, Dutch, and Norwegian men, and especially to the trends in mortality from infectious diseases, lung cancer (men only), prostate cancer, breast cancer, and COPD in most countries. We found that the secular decline in all-cause mortality stagnated among Danish, Dutch and Norwegian male birth cohorts born between 1890 and 1915, among French men born after 1920, and among women from all countries born after 1920. Where the decline in all-cause mortality stagnated, cohort-specific trends in mortality from lung cancer, COPD, and IHD were generally unfavourable as well. For infectious diseases, stomach cancer, and cerebro-vascular diseases, mortality increased among cohorts born before 1890, and decreased rapidly among later generations.

The substantial (non-linear) cohort effects that we found in all-cause mortality trends among elderly Danish, Dutch and Norwegian men were not identified in the few previous studies on cohort effects in all-cause mortality trends among the elderly [3,22]. Kannisto suggested that cohort patterns do not have important effects on trends in all-cause mortality among the elderly of developed countries [3]. However, his time series analyses did not include Denmark and The Netherlands, and his shorter observed period (1950-84) did not fully represent the birth cohorts that experienced a stagnation of mortality decline [6,23]. Jacobsen et al. did not identify non-linear cohort effects in mortality trends among Danish men aged 40-84 during 1960-99 [22]. However, their analysis was restricted to birth cohorts born after 1895, and therefore missed the important non-linearities that would become visible when extending the observation to cohorts born before 1895. The non-linearities that we observed for women born from 1910 onwards were found in their study as well.

Previous cohort studies on mortality from specific causes of death observed the same general tendencies as were observed in our study [24-37]. However, in some cases, the exact timing and form of the observed cohort trends differed, probably because of the use of different techniques, shorter study periods, or different age groups. By applying the same technique to an identical selection of eight different causes of death among men and women in seven European countries, we were able to observe not only commonalities but also dissimilarities between these different countries.

Evaluation of data and methods

The mortality and population data used in this study stem from countries considered to have good or excellent population and vital registries [2,3], and highly accurate reported survivorship counts [3,38]. Total mortality and population data for those aged ≥80 years were obtained from the Kannisto-Thatcher Database in which the data were checked for age-heaping and were subjected to a number of checks for plausibility [3].

Particularly at older ages, the validity of the underlying cause of death may be questioned owing to the presence of more than one chronic disease contributing to death [39,40]. Because changes over time in the quality of reporting underlying causes of death are likely to be gradual rather than abrupt, this may have resulted in only a modest overestimation of period effects. The non-linear cohort patterns in cause-specific mortality trends are likely to be unaffected.

We made considerable effort to control for the abrupt coding changes between and within revisions of the International Classification of Diseases (ICD), which otherwise could have biased the analyses of long-term trends in causes of death, and could overestimate period effects in APC analyses. We expect that any residual bias of coding problems is removed from our analyses by including period as a control variable in all our analyses of cohort patterns of mortality.

The identification problem inherent to APC analyses was dealt with by dividing mortality into a linear component (drift) and non-linear components. This enabled us to identify non-linear cohort effects and to quantify their importance. A focus on non-linear cohort effects leads to underestimation of the importance of all cohort effects to an unknown degree, because linear cohort effects are, together with linear period effects, included in the drift term. There can be substantial linear cohort effects when there is a large effect of drift, i.e. when mortality trends are largely linear. When we observed small non-linear cohort effects, they occurred in general together with large drift effects. The only exception concerned trends in IHD in Danish, Dutch, and Norwegian men. Only in these cases, it is certain that mortality trends are not strongly determined by cohort effects (see Appendix I). Our method thus yields a conservative but informative estimate of the contribution of cohort effects.

Explanation of observed cohort patterns

Where the decline in all-cause mortality stagnated among subsequent cohorts, cohort-specific trends in mortality from lung cancer, COPD, and to a lesser extent IHD, were generally unfavourable as well. This parallel development indicates that changed smoking behaviour among adults, as reflected by the cohort patterns in lung cancer mortality [41], largely influenced the trends in COPD and IHD and also influenced the temporal stagnation of male all-cause mortality in The Netherlands, Denmark and Norway among birth cohorts 1895-1920, and the stagnation of all-cause mortality decline for women born from 1920 onwards. Available data on smoking prevalence strengthen this conclusion. Dutch men born between 1897 and 1917 had a higher life-time exposure to smoking as compared with other generations of men [42]. Among Danish adult men in 1954, 86 % were present or former smokers, with a higher percentage for those aged 35-54 (cohorts 1900-19) as compared with older men (cohorts before 1900) [43]. For Norwegian men, the proportion of ever smokers increased for the birth cohorts 1890-1914, after which it stayed at the

same level up to cohort 1930-34 (83 %) [44]. Among men in the other countries, lung cancer also increased among the earlier birth cohorts, as observed in previous studies (e.g. [37]). However, this increase seemed not strong enough in absolute terms to substantially influence the trends for all-cause mortality. For women, the large increase in lung cancer mortality for the cohorts born from 1920 onwards also parallels increasing smoking rates among subsequent generations [44,45]. The low contribution of non-linear cohort effects to trends in lung cancer mortality among older women might be due to the recent timing and the generalized nature (among all birth cohorts) of the spread of smoking among women.

The onset of the mortality decline in infectious diseases, cerebrovascular diseases and stomach cancer for cohorts born at the end of the 19th century, which occurred in all countries, seems to indicate a contribution of the improvements in living conditions in early life. Earlier, a poor environment during infancy and childhood, and especially a high load of respiratory or gastrointestinal infections, has been proposed as a determinant of mortality from stomach cancer, tuberculosis and cerebrovascular diseases at older ages [46]. Mortality from infectious diseases at older ages is related to the disease load experienced during the first year of life, especially exposure to airborne infectious diseases [47]. Infant mortality - often used as a proxy of living conditions in infancy [48] and of the disease load during the birth year [47] – was still high in the late 19th century in most European countries, and began to fall at the turn of the century [49]. The fact that infant mortality started to decline for about the same cohorts as adult mortality from the abovementioned causes of death suggests that improvements in living conditions have left an imprint on the mortality experience of cohorts up to old age. It is important to note, however, that this possible impact is visible only in mortality trends from some specific causes of death, while it is not reflected in all-cause mortality trends.

The strong non-linear cohort patterns in IHD mortality trends in France, Finland, and to a lesser extent Norway may reflect another cohort-specific determinant of old-age mortality trends. As these cohort patterns (strong increases among the birth cohorts born before 1900 or 1910, followed by substantial decreases afterwards) were observed for both men and women, changed smoking behaviour among adults could not be the determining factor in these countries, given the fact that men and women differed in their smoking history [16]. Late effects of circumstances in early life have been suggested in relation to IHD [48,50,51], but the effects have been debated and seem to be less pronounced than for stroke and COPD [46,52]. Moreover, during the late 19th and early 20th century, trends in infant mortality and Gross Domestic Product (an indicator of general socio-economic development [53]) were not markedly different in France, Finland, and Norway as compared with the other countries (data not shown). Another possibility is that the initial rise in IHD mortality risk in France, Finland and Norway is related to national patterns of behavioural change that are similar for both men and women, such as diet, alcohol use, or physical inactivity [54,55]. This hypothesis, which was generated by observing variations between countries in cohort patterns for IHD, needs further testing by in-depth analyses within specific countries using data on trends in these possible determinants.

Implications

Cohort effects appear to be important in determining secular trends in all-cause and cause-

specific mortality among the elderly in seven European countries. Our results indicate that factors occurring in adulthood (predominantly due to smoking) and in infancy or childhood (such as poor living conditions) have left an imprint on the mortality experience of birth cohorts up to old age. Therefore, in order to understand the recent trends in old-age mortality and to make projections of possible future developments, one should take into account determinants that are located earlier in the life course of subsequent cohorts.

KEY POINTS

- Cohort patterns were identified in old-age mortality trends in all seven European countries, for both the sexes and virtually all causes of death.
- Parallel unfavourable cohort trends were observed for all-cause mortality and mortality from lung cancer, COPD, and to a lesser extent IHD, among Danish, Dutch and Norwegian men born between 1890 and 1915.
- For infectious diseases, stomach cancer, and cerebrovascular diseases, mortality increased among cohorts born before 1890, and decreased strongly thereafter.
- Both living conditions in childhood and smoking in adulthood seem to have left an imprint on the mortality experience of birth cohorts up to high ages.

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Appendix I. The contribution of drift to all-cause and cause-specific mortality trends, by sex and country

	Share of scaled deviance[a] explained by adding drift to the model including only age						
	DK	E&W	FIN	F	NL	NO	S
Men							
All-cause mortality	39.3	86.8	87.3	93.2	26.9	23.1	80.8
Cancer of lung	63.1	4.3	0.8	78.6	42.7	85.4	47.4
COPD	75.4	58.0	10.6	0.8	50.5	81.1	64.2
Ischaemic heart disease	4.3	18.4	3.2	8.1	2.1	0.4	18.7
Infectious diseases	65.8	75.7	86.3	52.3	36.7	50.1	50.6
Cancer of stomach	98.2	88.4	95.8	92.9	98.7	97.6	97.7
Cancer of prostate	75.7	68.9	69.9	45.8	81.3	79.9	85.7
Cerebrovascular diseases	95.2	90.0	91.3	82.3	94.4	80.4	92.0
Women							
All-cause mortality	81.9	98.5	93.1	93.8	91.7	93.3	95.4
Cancer of lung	95.9	92.3	83.4	90.2	85.7	94.5	90.1
COPD	83.2	24.2	(0.1)	0.5	15.3	42.9	67.1
Ischaemic heart disease	44.0	56.6	4.8	5.7	55.1	22.1	69.0
Infectious diseases	49.6	37.1	75.1	16.2	33.4	26.1	20.4
Cancer of stomach	98.7	96.7	96.5	92.3	98.9	99.4	98.7
Cancer of breast	27.5	50.4	68.7	88.6	45.4	50.2	25.3
Cerebrovascular diseases	88.2	87.7	90.4	74.7	92.2	83.7	81.7

[a] The scaled deviance that is explained by the full age-period-cohort model as compared with the model including only age.

DK = Denmark; E&W = England and Wales; FIN = Finland; F = France; NL = The Netherlands; NO = Norway; S = Sweden.

( ) No statistically significant reduction in the scaled deviance ($p < 0.05$ one sided).

COPD = chronic obstructive pulmonary diseases.

PART II

EXPLANATION

6

RELATION BETWEEN TRENDS IN LATE MIDDLE-AGE MORTALITY AND TRENDS IN OLD-AGE MORTALITY – IS THERE EVIDENCE FOR MORTALITY SELECTION?

Objective To test whether mortality selection was a dominant factor in determining trends in old-age mortality, by empirically studying the existence of a negative correlation between trends in late middle-age mortality and trends in old-age mortality among the same cohorts. **Methods** A cohort approach was applied to period data on total and cause-specific mortality for Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden, in 1950-1999. The study described and correlated mortality trends for five-year centralised cohorts from 1895 to 1910 at ages 55-69, with the trends for the same cohorts at ages 80-89. The research distinguished between circulatory diseases, cancers, and diseases specifically related to old age. **Results** All-cause mortality changes at ages 80-89 were strongly positively correlated with all-cause mortality changes at ages 55-69, especially among men, and in all countries. Virtually the same correlations were seen between all-cause mortality changes at ages 80-89 and changes in circulatory disease mortality at ages 55-69. Trends in mortality at ages 80-89 from infectious diseases, pneumonia, diabetes mellitus, symptoms or external causes showed no clear negative correlations with all-cause mortality trends at ages 55-69. **Conclusion** The consistently positive correlations seen in this study suggest that trends in old-age mortality in north western Europe in the late 20th century were determined predominantly by the prolonged effects of exposures carried throughout life, and not by mortality selection.

BACKGROUND

The ageing of populations has important consequences for future demands of health care services and old-age benefit systems. The degree of ageing of populations is strongly influenced by future patterns of old-age mortality [1,2]. Therefore, projections of future mortality trends are highly important for public health. To make informed projections of future mortality trends it is important to accurately describe past trends in old-age mortality and to analyse its determinants.

Past trends in old-age mortality have been studied in many countries. Among low-mortality countries, a general decline in mortality among those aged 80 and over since the 1950s has been found [3-6]. However, when this period of mortality decline is examined more closely, important cross-national differences in the pace of decline in old-age mortality appear. From the 1980s onwards, mortality decline stagnated in Denmark, the Netherlands, and among Norwegian men, while in other countries the mortality decline continued [7-9].

The mechanisms behind these trends are still largely unknown. Next to effects of life-style or events occurring in late life itself, studies have focused on the effects of events or life-style earlier in life, in accordance with the life course perspective. One mechanism mentioned in literature as a possible determinant of old-age mortality trends, and one that is related to the life-course perspective, is mortality selection [1,7,10,11].

Mortality selection indicates that when mortality at younger ages is high, it tends to affect the frail individuals first, leaving a more selected and more robust population that survive up to high ages [12]. With decreasing mortality at younger ages, the increasing proportion of the elderly population might be expected to be less healthy when compared with their more selected predecessors [1], and subsequently could experience comparatively higher morbidity and mortality at older ages.

Mortality selection effects have been posited in studies that use mathematical models to study cohort mortality (e.g. [12-18]), for example to explain the deceleration of the age pattern of mortality at older ages (e.g. [16]), or the Black-White mortality crossover (e.g. [12]). In addition, there is a long history of empirical cohort analyses of mortality. These studies focussed mainly on the association between debilitating events or mortality in early life and mortality in adult ages (e.g. [19-25]). They showed predominantly positive associations, indicating no mortality selection. However, one study reported negative associations [26], and another reported no associations [27]. Three other empirical studies, focussing more explicitly on old-age mortality, did not find any empirical evidence for mortality selection [28-30].

Thus, the evidence on mortality selection effects is rather mixed. Moreover, as most of these studies studied the mortality experience of single cohorts, little is known on the role of mortality selection in long-term mortality trends. Furthermore, because previous studies often focused on the effects of mortality at very early ages on adult mortality, the effects of adult mortality on old-age mortality are largely unknown.

The objective of this paper is to empirically study whether, and in what way, trends in late middle-age mortality are correlated with old-age mortality trends among the same cohorts. We hypothesise that mortality selection is a driving factor in old-age mortality trends in seven north western European countries from 1950 to 1999. Consequently, we expect inverse correlations.

To test this hypothesis, we use data on all-cause mortality and mortality data for causes of death that are especially susceptible to mortality selection – that is circulatory diseases at late middle age and diseases specifically related to old age. Mortality declines in circulatory diseases (predominantly ischaemic heart diseases) have been shown to lead to increased prevalence of chronic heart diseases at older ages [31], with subsequently higher mortality risks of related diseases [32]. With respect to diseases specifically related to old age, recent mortality increases were observed [9]. These increases could possibly result from an increasing proportion of frailer people at higher ages, due to decreased selection, because of mortality declines at younger ages.

In this study, we assess whether trends in old-age mortality (ages 80-89) among subsequent birth cohorts are inversely correlated with mortality trends at late middle age (ages 55-69) for the same cohorts, and whether different correlations are observed for (a) trends in circulatory diseases mortality at late middle age, and (b) mortality trends from diseases specifically related to old age.

DATA AND METHODS

For this analysis, data on all-cause mortality, cause-specific mortality and population numbers, by five-year age groups and sex, were obtained from national statistical offices and related institutes, for Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden, for the years 1950 to 1999.

In addition to all-cause mortality, we included three main groups of causes of death in our analysis: all circulatory diseases, all cancers, and the remaining causes of death. Within all circulatory diseases we distinguished between ischaemic heart diseases and cerebro-vascular diseases. Within the remaining causes of death, we focused on diseases specifically related to old age – that is infectious diseases, pneumonia, diabetes mellitus, dementia, and symptoms. See Janssen et al. (2004) for the three-digit codes used for these causes of death in the different revisions of the International Classification of Diseases (ICD) from the World Health Organisation [33]. For ischaemic heart diseases we included the numbers of deaths for code 422.1 under ICD-6/7.

The use of three-digit codes can still generate mortality discontinuities due to ICD revisions or due to incidental coding changes, such as the ones in England and Wales between 1984 and 1992, and in Sweden after 1980 [33]. We identified and adjusted for these coding-related mortality discontinuities in our analysis. Adjustment involved the recalculation of the number of cause-specific deaths by means of sex- and cause-specific transition coefficients. These transition coefficients are the parameter estimates of variables associated with a coding change (e.g. ICD-8toICD-9), and obtained through sex-specific regression models. In these regression models, cause-specific mortality was the dependent variable and age, year of death, and variables associated with a coding change were independent variables. To obtain the sex- and cause-specific transition coefficients to recalculate cause-specific deaths for those aged 55-69 and those aged 80-89, the regression model was applied to cause-specific mortality among those aged 60 and over, and those aged 80 and over, respectively. For those aged 55-69, recalculation was applied to ischaemic heart diseases in the Netherlands and Sweden, and cerebrovascular diseases in Finland. For those aged 80-89, deaths from all selected causes, except all circulatory diseases and infectious diseases, were adjusted for coding changes.

We aggregated the mortality and population data into five-year periods, and calculated five-year age-specific mortality rates by dividing the mortality data by the mid-year population estimates. To these period data, we applied a "mixed cohort approach", by using as the unit of observation centralised birth cohorts. In this approach, the data are combined by five-year period and five-year age group, and centred around the cohort calculated by subtracting the age group from the period (see figure 1). We were able to study four different centralised birth cohorts (those born around 1895, 1900, 1905, and 1910) to fulfil our aim of correlating mortality among those aged 55-69, with mortality among those aged 80-89 using the available data (1950-1999).

Cohort mortality rates for ages 55-69 and ages 80-89 were obtained by taking for each separate centralised cohort the unweighted average of the age-specific rates over the three or two five-year age groups, respectively. Relative changes in these cohort mortality rates were calculated by relating the mortality rate of a given centralised cohort to the mortality rate of the preceding centralised cohort.

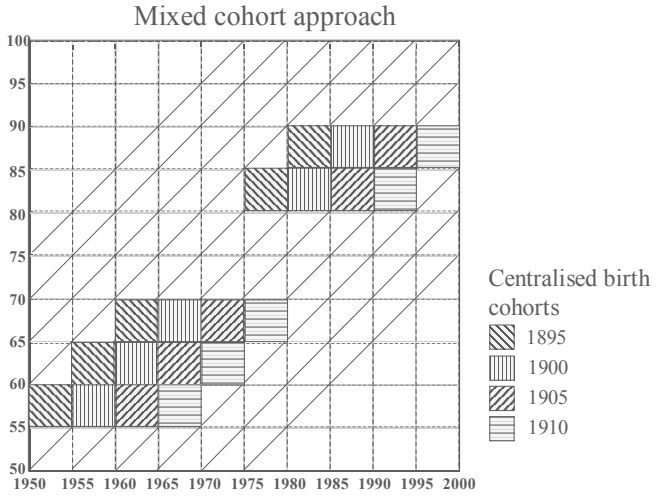

Figure 1. The mixed cohort approach applied to period data from 1950 to 1999, for the ages 50 to 100

As a first exploration of the mortality selection mechanism, we correlated all-cause mortality levels between those aged 55-69 and those aged 80-89. Our main analysis, however, consisted of the correlation of relative cohort mortality changes between the late middle aged and the elderly population. For all-cause mortality, we correlated absolute cohort mortality changes as well. Whereas absolute mortality changes more accurately express the importance of the mortality changes at younger ages, and the effect that they can have on trends in mortality at older ages, relative mortality changes can be more readily compared between countries.

The correlations were calculated across the seven countries, two sexes, and (changes in the) four centralised cohorts. In addition, correlations were calculated separately for men and women, each (change in) centralised cohort, and each country. The correlations of the mortality levels were stratified by sex.

In an additional analysis, we correlated the relative changes in all-cause mortality trends – that is the deceleration or acceleration of mortality trends in both late middle age and old age. We did so to find out if mortality at late middle age and old age is not only related in terms of the direction of mortality changes (is an increase in the one associated with a decrease in the other?) but also in terms of the pace of the mortality change (is an acceleration of the one associated with a deceleration in the other?). This additional analysis was conducted as an attempt to explain the deceleration of old-age mortality decline that was seen in Denmark, the Netherlands, and among Norwegian men [9].

To check the robustness of our results, different age groups were used when correlating all-cause mortality trends – that is ages 70-79 instead of 55-69 and ages 80-94 instead of 80-89. For the latter analysis only three instead of four centralised cohorts could be analysed.

RESULTS

Among those aged 55-69, mortality levels were highest in Finland, England and Wales, and France (men), and lowest in Norway, Sweden, and the Netherlands (women)(table 1). Among those aged 80-89, Finland had by far the highest mortality level, and France the lowest level, especially among the more recent cohorts. Correlation between all-cause mortality levels of those aged 55-69 and those aged 80-89, showed highly positive and significant correlations, among both men and women, although less so for the more recent cohorts (table 2).

All-cause mortality generally declined over the five-year centralised birth cohorts from 1895 to 1910 both among men and women aged 55-69 and among men and women aged 80-89 (table 1). The trends for men in Denmark, the Netherlands, and Norway were less favourable. Correlations between both relative and absolute changes in all-cause mortality at ages 55-69 with those at ages 80-89 were significant and positive (0.61 and 0.58, respectively) (table 3, figure 2). Among men, the correlation coefficients were especially high (0.7). Among women, the correlations were lower (0.3) and not statistically significant. The correlations were strongest for the Netherlands, Norway, and Sweden (men only).

Circulatory disease mortality among those aged 55-69 generally declined over subsequent centralised birth cohorts (data not shown). For men, this decline started only among later cohorts. For Dutch men, circulatory disease mortality increased. Relative changes in circulatory disease mortality at ages 55-69 correlated significantly and positively with all-cause mortality changes at ages 80-89 (0.61) (table 4).

Table 1a. All-cause mortality levels and trends, men aged 55-69 and 80-89, centralised birth cohorts 1895-1910, by country

Country	Mortality rate (x 10000) by centralised birth cohort				Relative change in mortality rate between subsequent centralised birth cohorts (%)		
	1895	1900	1905	1910	1895-1900	1900-1905	1905-1910
Aged 55-69							
Denmark	212.46	225.84	229.71	224.01	6.29	1.71	-2.48
England&Wales	298.60	297.38	285.20	267.64	-0.41	-4.10	-6.16
Finland	345.14	342.36	335.37	320.07	-0.80	-2.04	-4.56
France	279.41	282.70	270.58	250.08	1.18	-4.29	-7.58
Netherlands	203.58	217.06	229.24	229.41	6.62	5.61	0.07
Norway	192.22	207.20	208.91	204.28	7.79	0.82	-2.21
Sweden	198.89	198.23	195.77	192.72	-0.33	-1.24	-1.56
Aged 80-89							
Denmark	1508.78	1485.48	1497.17	1476.78	-1.54	0.79	-1.36
England&Wales	1725.58	1622.06	1511.90	1427.60	-6.00	-6.79	-5.58
Finland	1670.92	1638.10	1582.05	1508.16	-1.96	-3.42	-4.67
France	1595.21	1498.76	1356.52	1258.63	-6.05	-9.49	-7.22
Netherlands	1497.12	1494.98	1520.32	1498.09	-0.14	1.70	-1.46
Norway	1479.93	1473.07	1477.12	1446.20	-0.46	0.28	-2.09
Sweden	1556.02	1506.78	1442.75	1375.34	-3.16	-4.25	-4.67

Table 1b. All-cause mortality levels and trends, women aged 55-69 and 80-89, centralised birth cohorts 1895-1910, by country

Country	Mortality rate (x 10000) by centralised birth cohort				Relative change in mortality rate between subsequent centralised birth cohorts (%)		
	1895	1900	1905	1910	1895-1900	1900-1905	1905-1910
Aged 55-69							
Denmark	143.52	134.82	124.80	118.72	-6.06	-7.43	-4.88
England&Wales	152.94	144.10	136.86	132.50	-5.78	-5.02	-3.19
Finland	175.06	166.49	146.57	128.21	-4.90	-11.96	-12.53
France	144.73	131.19	119.31	106.29	-9.35	-9.05	-10.91
Netherlands	129.23	118.69	111.83	102.14	-8.16	-5.78	-8.67
Norway	121.47	114.11	106.87	98.75	-6.06	-6.35	-7.60
Sweden	134.22	120.12	109.01	100.25	-10.50	-9.25	-8.03
Aged 80-89							
Denmark	1098.45	1064.71	1037.78	1003.58	-3.07	-2.53	-3.30
England&Wales	1218.97	1104.13	1010.94	981.43	-9.42	-8.44	-2.92
Finland	1263.72	1213.57	1183.91	1095.07	-3.97	-2.44	-7.50
France	1146.66	1039.61	919.98	837.24	-9.34	-11.51	-8.99
Netherlands	1076.92	1017.34	1003.29	982.93	-5.53	-1.38	-2.03
Norway	1119.08	1065.37	1033.13	982.27	-4.80	-3.03	-4.92
Sweden	1131.19	1059.75	982.29	931.49	-6.32	-7.31	-5.17

For women, the positive correlation was not significant. Correlations of all-cause mortality changes at ages 80-89 with trends in mortality from ischaemic heart diseases and cerebrovascular diseases at ages 55-69 were also significant and positive, but less strong (0.40 and 0.42, respectively).

Table 2. Correlation of all-cause mortality levels at ages 55-69 with all-cause mortality levels at ages 80-89, for men and women born to the centralised birth cohorts 1895-1910 among seven European countries, stratified by sex

	Correlation of mortality levels					
Stratified by:	Total	(N)	Men	(N)	Women	(N)
All	0.90**	(56)	0.52**	(28)	0.83**	(28)
Centralised birth cohort 1895	0.91**	(14)	0.88**	(7)	0.87*	(7)
Centralised birth cohort 1900	0.93**	(14)	0.85*	(7)	0.89**	(7)
Centralised birth cohort 1905	0.90**	(14)	0.40	(7)	0.65	(7)
Centralised birth cohort 1910	0.90**	(14)	0.22	(7)	0.52	(7)
Denmark	0.99**	(8)	#	(4)	#	(4)
England and Wales	0.96**	(8)	#	(4)	#	(4)
Finland	0.99**	(8)	#	(4)	#	(4)
France	0.94**	(8)	#	(4)	#	(4)
Netherlands	0.99**	(8)	#	(4)	#	(4)
Norway	0.99**	(8)	#	(4)	#	(4)
Sweden	0.99**	(8)	#	(4)	#	(4)

* Correlation is significant at the 0.05 level (two-tailed).
** Correlation is significant at the 0.01 level (two-tailed).
# Too few observations.

Table 3. Correlation of relative and absolute cohort changes in all-cause mortality at ages 55-69 with relative and absolute cohort changes in all-cause mortality at ages 80-89, for men and women born to the centralised birth cohorts 1895-1910 among seven European countries

Stratified by:	Correlation of mortality changes Relative	Absolute	N
All	0.61**	0.58**	42
Men	0.71**	0.70**	21
Women	0.33	0.32	21
Cohort change[a] 1895 to 1900	0.74**	0.61*	14
Cohort change[a] 1900 to 1905	0.56*	0.64*	14
Cohort change[a] 1905 to 1910	0.74**	0.82**	14
Denmark	0.63	0.44	6
England and Wales	0.41	0.20	6
Finland	0.52	0.37	6
France	0.64	0.08	6
Netherlands	0.78	0.79	6
Norway	0.84*	0.76	6
Sweden	0.88*	0.54	6

[a] Relative change in mortality rates between the two subsequent centralised birth cohorts.
* Correlation is significant at the 0.05 level (two-tailed).
** Correlation is significant at the 0.01 level (two-tailed).

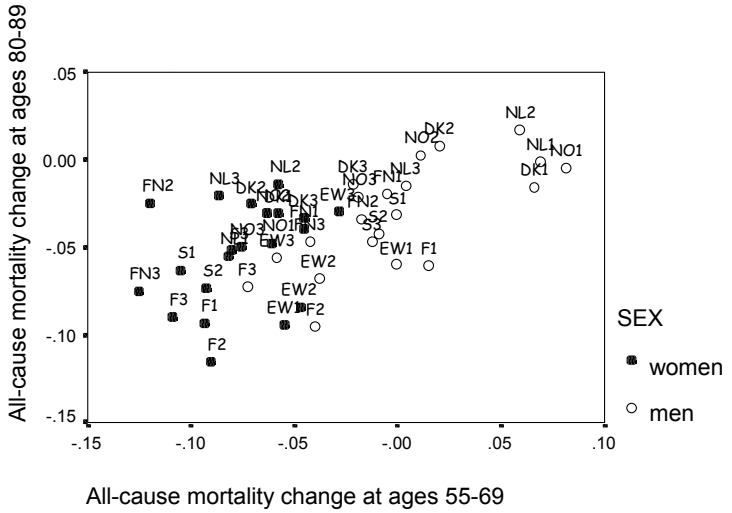

Figure 2. Scatter plot of the correlation of relative changes in all-cause mortality at ages 55-69 and at ages 80-89, for men and women born to the centralised cohorts 1895-1910 among seven European countries

DK = Denmark, EW = England and Wales, FN = Finland, F = France, NL = the Netherlands, NO = Norway, S = Sweden; Cohort change: 1 = cohort change 1895-1900, 2 = cohort change 1900-1905, 3 = cohort change 1905-1910.

Table 4. Correlation of relative changes in circulatory disease mortality at ages 55-69 with relative changes in all-cause mortality at ages 80-89, for men and women born to the centralised birth cohorts 1895-1910 among seven European countries

Causes of death at ages 55-69	All (N=42)	Men (N=21)	Women (N=21)	Cohort change[a] 1895-1900 (N=14)	Cohort change[a] 1900-1905 (N=14)	Cohort change[a] 1905-1910 (N=14)
All circulatory diseases	0.61**	0.70**	0.40	0.77**	0.56*	0.60*
Ischaemic heart diseases	0.40**	0.40	0.12	0.84**	0.47	0.39
Cerebrovascular diseases	0.42**	0.41	0.09	0.39	0.50	0.47

[a] Relative change in mortality rates between the two subsequent centralised birth cohorts.
* Correlation is significant at the 0.05 level (two-tailed).
** Correlation is significant at the 0.01 level (two-tailed).

All-cause mortality trends at ages 55-69 correlated significantly and positively with mortality trends in circulatory diseases and cancer at ages 80-89 (0.58 and 0.69, respectively)(table 5). The positive correlation for cancer mortality was observed for both men and women, whereas for circulatory disease mortality only for men. All-cause mortality trends at ages 55-69 did not clearly correlate with mortality trends at ages 80-89 from diseases other than circulatory diseases and cancer, nor with diseases specifically related to old age, such as infectious diseases, pneumonia, diabetes mellitus, dementia, symptoms, and external causes of death. A significant positive correlation was found only for diabetes mellitus and symptoms (among women). While a few inverse correlations were seen, especially for pneumonia, these correlations were weak, inconsistent, and non-significant.

Table 5. Correlation of relative changes in all-cause mortality at ages 55-69 with relative mortality changes in specific causes of death at ages 80-89, for men and women born to the centralised birth cohorts 1895-1910 among seven European countries

Causes of death at ages 80-89	All (N=42)	Men (N=21)	Women (N=21)	Cohort change[a] 1895-1900 (N=14)	Cohort change[a] 1900-1905 (N=14)	Cohort change[a] 1905-1910 (N=14)
All circulatory diseases	0.58**	0.66**	0.14	0.63*	0.54*	0.51
All cancers	0.69**	0.54*	0.49*	0.64*	0.79**	0.50
All other causes	0.17	0.40	0.19	0.26	0.14	0.42
Diseases specifically related to old-age within "all other causes":						
Infectious diseases	0.01	0.15	0.26	–0.15	0.14	0.43
Pneumonia	–0.03	–0.14	–0.23	–0.15	0.14	0.25
Diabetes mellitus	0.44**	0.11	0.42	0.44	0.33	0.55*
Dementia	0.18	0.25	0.21	–0.23	0.37	0.44
All symptoms and ill-defined conditions	0.07	–0.12	0.62**	–0.25	0.13	0.35
All other diseases[b]	0.22	0.67**	0.13	0.45	–0.03	0.17
All external causes of death	0.39*	0.33	–0.09	0.42	0.36	0.18

[a] Relative change in mortality rates between the two subsequent centralised birth cohorts.
[b] This refers to all diseases not included in this table – that is all-cause mortality minus all circulatory diseases, all cancers, infectious diseases, pneumonia, diabetes mellitus, dementia, "all symptoms and ill-defined conditions", and all external causes of death.
* Correlation is significant at the 0.05 level (two-tailed).
** Correlation is significant at the 0.01 level (two-tailed).

Acceleration or deceleration of mortality trends among those aged 80-89 was positively correlated with the pace of mortality change among those aged 55-69, although correlations were weak (0.27) and non-significant.

Trends in all-cause mortality at ages 70-79 (instead of 55-69) correlated significantly and highly positive with trends in all-cause mortality at ages 80-89 (0.69). The correlation of all-cause mortality changes at ages 55-69 with those at ages 80-94 (instead of 80-89) was significant and positive (0.59) as well.

DISCUSSION

In this paper, we explored the relation between mortality trends in late middle age (55-69) and mortality trends in old age (80-89) for male and female cohorts born around 1895, 1900, 1905 and 1910 in seven European low-mortality countries. All-cause mortality changes at ages 80-89 are strongly positively correlated with all-cause mortality changes at ages 55-69, especially among men, and in all countries. Virtually the same correlations were seen between all-cause mortality changes at ages 80-89 and changes in circulatory disease mortality at ages 55-69. Mortality trends at ages 80-89 from diseases specifically related to old age – that is infectious diseases, pneumonia, diabetes mellitus, symptoms and external causes, showed no clear negative correlations with all-cause mortality trends at ages 55-69.

This evidence suggests that mortality selection has not been a driving factor behind old-age mortality trends in the countries under study. Our results were found robust against the selection of different age groups (70-79 instead of 55-69 and 80-94 instead of 80-89). Furthermore, we found no indications that the recent deceleration of the mortality decline among the elderly in Denmark, the Netherlands, and Norway was related to accelerated declines in mortality at earlier ages of the same cohorts.

This study is unique in its attempt to link, in a cohort-wise manner, trends in middle-age mortality with trends in old-age mortality. Perhaps closest to our study is a study by Manton on the effects of increases in life expectancy at advanced ages on mortality from conditions associated with a debilitation at those ages. He also found no evidence for decreased selectivity [29]. Persons who survive up to age 85 were on average healthier than their predecessors, in contrast with what would be expected according to the mortality selection theory when mortality is improving.

Evaluation of data and methods

The mortality and population data used in this study stem from countries considered to have good or excellent population and vital registries [4,5]. Reported survivorship counts are highly accurate [5,34]. Comparison of our mortality data to the mortality data among those aged 80 and over from the Kannisto-Thatcher Database [5] - in which the data were checked for age-heaping and were subjected to a number of checks for plausibility - showed only small discrepancies.

In our analysis, we applied a mixed cohort approach – that is a cohort approach applied to period data. A pure cohort approach was difficult to conduct with the available data, and

would have led to the inclusion of only three subsequent five-year cohorts, which we considered too few for correlation analyses. A disadvantage of the mixed cohort approach is that it cannot clearly separate subsequent cohorts. Consequently, the identified cohorts overlap, which could lead to an underestimation of mortality trends and possibly to a dilution of the strength of the correlations between trend estimates. However, some dilution of effect could not explain the observed positive instead of inverse relation between mortality trends at late middle age and old age.

We made an extraordinary effort to deal with ICD and other coding related changes that can affect cause-specific mortality trends, and that are often neglected in other studies. Even though some residual effects of coding problems could not be excluded, we expect that these problems did not affect the results to any substantial extent [8,9,33].

We do not expect that the potentially inferior quality of cause of death coding among the very elderly as compared with the late middle age would substantially affect our results. The quality of coding can only bias the correlation of trends if clear changes in the quality of coding over time occurred, which is unlikely.

Explanations of the absence of hypothesised inverse correlations

One possible explanation of the lack of negative correlations is that our study lacked potential to empirically observe an effect of mortality selection, because of comparatively little variation in mortality at younger ages between subsequent cohorts. Larger variations were, however, seen between the selected countries. Moreover, life-table calculations for the Netherlands in 1950 showed that the mortality declines among those aged 55-69 within most individual countries were large enough to influence mortality trends among those aged 80-89. Considering the extreme situation in which all people saved from dying in the younger group will eventually die in the older group, a 10% mortality reduction among those aged 55-69 would lead to a mortality increase at age 80-89 years of 14% among men, and of 11% among women.

The lack of empirical support to the mortality selection hypothesis could also possibly be explained by not considering the greatest advances in medical care and resulting mortality declines since the 1970s among those aged 55-69 [35]. Improvements in medical care and new treatments lead to higher prevalences in chronic disease for those who survive [29], which could result in higher mortality at higher ages from these diseases. Although we cannot exclude that more recent declines in mortality at ages 55-69 might influence trends in old-age mortality for more recent cohorts and future periods differently, our finding that positive correlations were also observed among the more recent cohorts studied, which were also characterized by important declines in mortality at middle age, casts doubt about the potential of mortality selection to determine future old-age mortality trends.

Explanations of the positive correlations observed

The consistently positive correlations seen in our analysis suggest the existence of parallel trends in late middle-age mortality and old-age mortality. This points to common mechanisms that develop in a cohort-wise fashion.

It has frequently been mentioned in literature that risks established early in life influence health conditions at adult ages. Examples of relevant exposures include nutrition in utero, exposure to infectious diseases and/or socio-economic circumstances in infancy or childhood [19-23,36]. Less clear is whether the effects of early life events last until old age [17,26,27]. Our results could indicate that effects of early life events or conditions, that have been shown to influence mortality risk at late middle age, have the potential to exert their influence until old age.

Prolonged exposure to, or longlasting effects of, risks emerging during adult age might be another factor contributing to the observed positive correlations. Additional analyses showed that changes in circulatory disease mortality at ages 55-69 correlated most strongly with changes in mortality at ages 80-89 from circulatory diseases (0.61) and cancers (0.54), and hardly with old-age mortality changes in remaining causes of death (0.15) and infectious diseases (-0.10). This indeed suggests that risk factors for circulatory diseases, like physical activity, hypertension, diet, smoking, and utilisation of medical care [37-39] emerging during adult age, are common determinants of mortality at both adult and old age among the same cohorts. With respect to smoking among men, changes in all-cause mortality at ages 55-69 were indeed strongly correlated to changes in mortality from lung cancer at ages 80-89 (0.71).

Implications

The consistently positive correlations between mortality changes at late middle age and mortality changes among the elderly population suggest that old-age mortality trends in north western Europe in the late 20th century are determined predominantly by the effects of early life circumstances carried throughout life and prolonged exposure to, or long-lasting effects of, risk factors emerging in adult life. Mortality selection has no discernible effect on secular trends in mortality. Our results, thus, do not support the concern that strong declines in middle-age mortality will ultimately lead to increases in old-age mortality for the same cohorts. In fact, the positive associations seen in our study suggest that recent trends in all-cause mortality among the middle aged may be used to inform projections of future trends in all-cause mortality among the elderly population.

Key points

- The evidence on mortality selection effects is rather mixed, and studied predominantly by relating mortality at very early ages with adult mortality among single cohorts. Consequently, little is known on mortality selection effects of mortality trends at adult ages on old-age mortality trends.
- In all countries under study, trends in mortality at late middle age correlate positively with trends in old-age mortality of the same birth cohorts.
- Positive correlations are also seen with trends in mortality from cardiovascular diseases at late middle age. Weak, but not inverse correlations are seen with trends in mortality from diseases specifically related to old age.
- The observed positive correlations point to effects of early life circumstances carried throughout life and prolonged exposure to, or long-lasting effect of, risk factors emerging in adult life. Effects of mortality selection seem to be of lesser importance in determining old-age mortality trends.

- Our results do not support the concern that strong declines in middle-age mortality will lead to an increase in old-age mortality for the same cohorts.

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7

ASSOCIATION BETWEEN GROSS DOMESTIC PRODUCT THROUGHOUT THE LIFE-COURSE AND OLD-AGE MORTALITY ACROSS BIRTH COHORTS. PARALLEL ANALYSES OF SEVEN EUROPEAN COUNTRIES, 1950-1999

Objective Mortality levels of national populations have often been studied in relation to levels of Gross Domestic Product (GDP) at time of death. Following the life-course perspective, we assessed whether old-age mortality levels for subsequent cohorts are differentially associated with GDP levels prevailing at different ages of the cohorts. **Methods** We used all-cause and cause-specific mortality data by sex, age at death (65-99), year at death (1950 to 1999), and year of birth (1865 to 1924) for Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden. Trends in national GDP per capita between 1865 and 1999 were reconstructed from historical national accounts data. Through Poisson regression analyses, we determined for each country both univariate and multivariate associations across five-year birth cohorts between mortality and GDP levels prevailing at time of death, and at earlier ages of the cohorts (i.e. 0-5, 6-19, 20-49, and 50-64). **Results** For the subsequent cohorts, levels of GDP at time of death were strongly inversely associated with all-cause mortality, especially among women, and among men in England and Wales, Finland, and France. In most countries, stronger associations were observed with GDP levels prevailing at earlier ages of the cohorts. After control for GDP at time of death, these associations remained. An independent association of GDP at earlier ages of the cohort was also observed for cause-specific mortality. The associations were negative for ischaemic heart diseases, cerebrovascular diseases, and stomach cancer. They were positive for prostate cancer, breast cancer, COPD (women), and lung cancer (women). GDP prevailing at ages 20-49 (men) and ages 50-64 (women) had the largest associations with old-age mortality. **Conclusion** These findings suggest an independent, mostly negative effect of GDP prevailing at earlier ages of subsequent cohorts on old-age mortality. Socio-economic circumstances during adulthood and middle age seem more important in determining old-age mortality trends than those during infancy or childhood.

BACKGROUND

The ageing of populations is a major challenge for individuals, society, policy makers and public health professionals. An increasing share of the elderly in national populations is expected to lead to increasing future demands of health care services and old-age benefit systems. The degree of ageing in European populations is determined substantially by current and future mortality trends among the elderly [1-3]. In order to make inferences on possible future old-age mortality trends, it is important to carefully describe past and current mortality trends among the elderly, and to study its determinants.

One key factor in influencing mortality levels and trends may be economic growth (e.g. [4]). During the 20th century, all European countries, and particularly those in the west and north, experienced dramatic increases in both economic prosperity and life expectancy. Economic growth is assumed to affect life expectancy in various ways: directly, through the improvement of material living conditions of European populations; and indirectly, by

facilitating advances in medical technology, and through rising educational levels, and the development of social welfare systems [5-7].

Usually, mortality trends are correlated with contemporaneous economic developments, e.g. with Gross Domestic Product (GDP) measured around the year of death [8,9]. Similarly, cross-national studies on the correlation between national mortality levels and national GDP levels have often assumed no or only a short lag time between GDP and mortality [10,11]. However, the influential paper by Kermack, McKendrick, and McKinlay in 1934, emphasizing the importance of environmental conditions during childhood [12], and recent insights from life-course epidemiology [13,14] stress the need to take into account determinants occurring in earlier phases of the lifetime of the respective birth cohorts. Longitudinal studies at the individual level have identified a series of factors related to early life that have long-lasting effects on the health and risk factor profile of people in later life (e.g. [15-17]). Socio-economic conditions in early life have also been found to be related to mortality in later life, both in ecological and individual level studies (e.g. [18-27]).

The abovementioned studies mainly focused on the relation between socio-economic conditions in very early life (infancy or childhood) and mortality at adult ages, rather than explicitly with mortality at old ages. Furthermore few studies that explicitly adopted a life-course perspective have described in detail the association between economic developments and secular trends in mortality. Therefore, it remains unknown whether the effect of socio-economic conditions related to earlier phases of life is strong enough to determine secular trends in old-age mortality of national populations.

In accordance with the life-course perspective, we assessed in this paper whether old-age mortality levels for subsequent cohorts in different countries are differentially associated with GDP levels prevailing at different ages of the cohorts. We addressed this question for men and women from seven European countries, thereby aiming to identify patterns that are common to most or all countries. Data were analysed from a cohort perspective. We focused on mortality levels of 12 subsequent five-year cohorts aged 65 to 99 between 1950 and 1999. Mortality levels of these different cohorts, born between 1865 and 1924, were related to national GDP levels prevailing at time of death, at ages 0-5, and at intermediate ages (i.e. 6-19, 20-49, and 50-64) for each birth cohort. In addition to all-cause mortality, we focused on a number of specific causes of death.

We hypothesized that GDP levels both at time of death and at earlier ages of the cohort are negatively associated with old-age mortality, both with and without control for GDP at time of death. With respect to cause-specific mortality, we expect an independent negative association of old-age mortality with GDP prevailing at ages 0-5 years for causes of death for which individual-level studies observed relationships with living conditions in childhood, i.e. cerebrovascular diseases, stomach cancer and ischaemic heart diseases [27]. For mortality from the remaining selected causes of death, i.e. prostate cancer, breast cancer, lung cancer, and chronic obstructive pulmonary diseases, we expect no, or perhaps positive, independent associations with GDP at early ages.

DATA AND METHODS

Data

From the Human Mortality Database (HMD), we obtained all-cause death counts by Lexis triangle (i.e. by both year of death and year of birth), sex, and single year of age (65 to 99 years) for Denmark, England and Wales, Finland, France, the Netherlands, Norway and Sweden, in the period 1950-1999 (http://www.mortality.org/). Cause-specific mortality data by five-year age group and sex were acquired from national statistical offices and related institutes. Cause-specific mortality data were available from 1951 onwards in Denmark, Finland, and Norway, and from 1952 onwards for Sweden. Data were available until 1997 for France and until 1998 for Denmark and England and Wales.

For the causes of death we selected ischaemic heart diseases (ICD-9 410-414), cerebrovascular diseases (ICD-9 430-434, 436-438), stomach cancer (ICD-9 151), prostate cancer (ICD-9 185), breast cancer (ICD-9 174-175), lung cancer (ICD-9 162), and chronic obstructive pulmonary diseases (ICD-9 490-494, 496). These are all causes of death frequently occurring among the elderly.

When analyzing long-term mortality trends in causes of death, coding changes, like the numerous revisions of the International Classification of Diseases (ICD), have to be taken into account. In our analysis of cause-specific mortality trends over the period 1950 to 1999, we had to bridge five different ICD revisions (ICD-6 to ICD-10). For this purpose, we constructed a general concordance table, i.e. a table linking the codes for specific causes of death through each successive ICD revision, based on three-digit ICD codes (see Janssen & Kunst, 2004)[28]. In addition, we identified and adjusted for other mortality discontinuities, such as those caused by our reliance on three-digit ICD codes instead of four-digit ICD codes and by incidental changes in coding rules unrelated to new ICD revisions. This adjustment involved multiplying cause-specific death numbers with cause-specific transition coefficients. These transition coefficients were obtained through cause-specific regression models. In these models, cause-specific mortality was the dependent variable and age, year of death, and variables associated with a transition, i.e. a coding change, were independent variables (see Janssen & Kunst, 2004 for a full description)[28]. For the current analyses, we used the transition coefficients from cause-specific regression models applied to mortality among those aged 60 and over.

In accordance with the life-course perspective, we distinguished twelve five-year cohort groups, born between 1865-69 and 1920-24. The all-cause mortality data have carefully been reconstructed into mortality rates by single year of age and single year of birth based on the available all-cause death counts by Lexis triangle, and the population data at January 1. To convert the cause-specific mortality data into cohort data, the country-specific and sex specific cohort weights from all-cause mortality were used.

For each country, for the period 1865 to 1999, trends in national real Gross Domestic Product per capita at constant 1995 prices in millions of national currency were reconstructed from historical national accounts data [29-31]. See Appendix I for our reconstruction method. Subsequently, these national GDP values were transformed into internationally comparable values using the purchasing power parities (PPP) of 1995, which take into account international differences in currencies and price levels, and are expressed in US Dollars.

Methods

Cohort- and country-specific levels of old-age mortality were estimated indirectly through sex-specific Poisson regression models. In these models, we included the number of deaths as dependent variable, and single year of age (categorical variable) and the interaction between country and five-year cohort group (discrete variable) as independent variables. The offset variable was the number of persons at risk. The parameter estimates for the interaction term were converted into relative mortality risks by taking exponents. The baseline cohort group was 1890-94, which is one of the cohorts for which information is available over the entire age range from 65 to 99 years during 1950-1999. The baseline country was England and Wales, i.e. one of the largest populations.

Through Poisson regression modelling, we determined, for each country separately, associations across birth cohorts between old-age mortality and GDP levels prevailing at time of death, during infancy and early childhood (0-5), at school ages and during adolescence (6-19), at main working ages (20-49), and at retirement ages (50-64). In the regression models, we included the number of deaths as dependent variable, and single year of age (categorical variable) and the GDP variables, separately, as independent variables. The offset variable was the number of persons at risk. All GDP variables were logarithmically transformed, throughout the analyses, in order to look at relative change.

To assess the strength and the direction of the effect of GDP at time of death on old-age mortality, we measured the income elasticity of cohort mortality. Income elasticity denotes the percentage mortality increase per percentage increase in GDP (on a logarithmic scale). It was calculated by transforming the parameter estimates of the GDP variable in the regression model by 100 * [exp(parameter estimate)-1].

To assess and compare the strength of the association between GDP at different ages and old-age mortality, we measured the reduction in scaled deviances (a measure of unexplained variance) when, in separate models, the different GDP variables are added to the regression model including only age. The higher the reduction in scaled deviances, the better the fit of the model has improved. The independent effect of GDP prevailing at earlier ages of the cohort on old-age mortality was assessed by comparing the reduction in scaled deviances of the regression model including GDP at time of death with the reduction in scaled deviances of the model including as well GDP at an earlier age of the cohort. To enable comparison, these reductions in scaled deviances were expressed as percentages of the reduction observed for a full age-period-cohort model. The statistical significance of the difference in scaled deviances between the different models was assessed by a log-likelihood ratio test.

All analyses were stratified by sex and by county. For the regression analyses we used SAS package 8.0.

RESULTS

For men and women alike, cohort- and country-specific mortality levels among those aged 65-99 in 1950-1999 generally declined over the successive five-year birth cohorts. However, for Danish, Dutch and Norwegian men born between 1890 and 1909, and for Danish and English & Welsh women born between 1910 and 1924, mortality was higher than mortality in surrounding cohorts. (Figure 1)

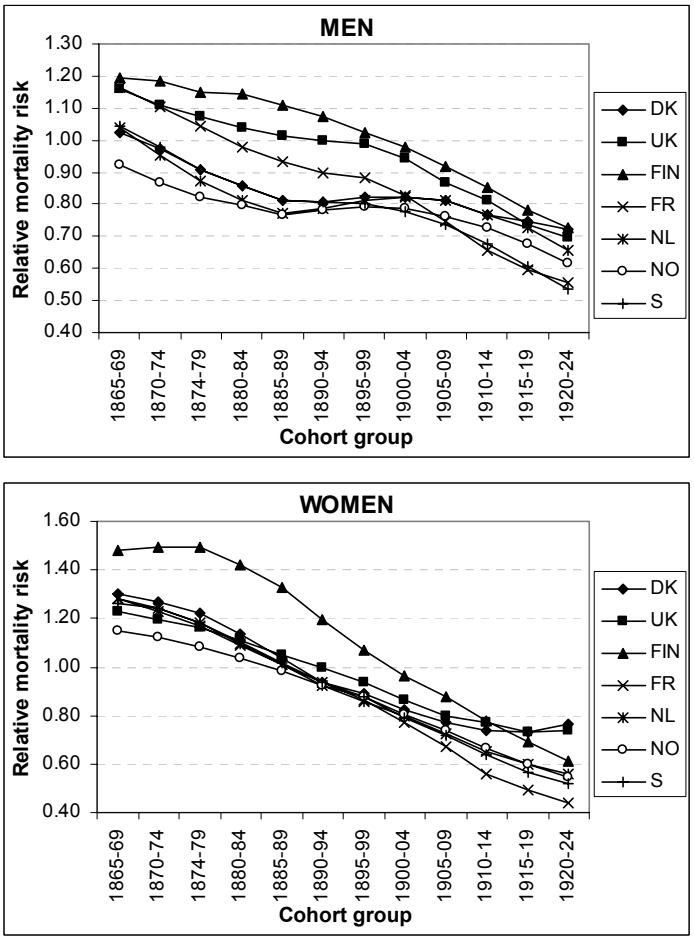

Figure 1. Cohort- and country-specific mortality levels for those aged 65-99, by sex

DK = Denmark; UK = England and Wales; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden.

GDP per capita increased approximately exponentially from 1865 to 1999, with a stronger increase after the Second World War. In the earlier decades, levels of GDP fluctuated and were very different between the countries. In later years, most countries occasionally experienced a temporal stagnation in the increase of GDP, but the general pattern was a fairly smooth increase with a gradual convergence of GDP levels between countries. (Figure 2)

For all-cause mortality among men, mortality decreased by 5 to 36 per cent per percentage increase in GDP at time of death (Table 1). Highest negative income elasticity was observed for England and Wales, France and Finland, whereas among Dutch, Norwegian, and Danish men, the income elasticity was only modest.

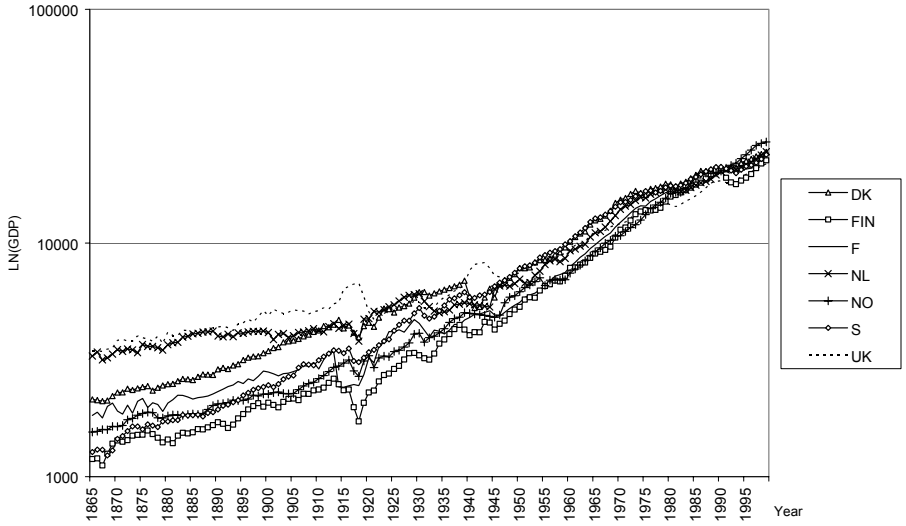

Figure 2. Time trends of real Gross Domestic Product per capita at 1995 US Dollars (PPP adjusted), 1865-1999, by country

DK = Denmark; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden; UK = United Kingdom.

For women, the decrease in old-age mortality accompanying an increase in national income at time of death was approximately similar for the different countries, with an average of 44 per cent.

The effect of GDP at time of death on old-age mortality can be determined by means of the reduction in scaled deviances presented in Figure 3. For all-cause mortality, adding GDP at time of death as an explanatory variable in the regression model including only age led to a statistically significant but modest reduction in scaled deviances among Dutch, Norwegian and Danish men (9%, 19%, and 31%, respectively), indicating a modest improvement of the fit of the model.

Table 1. Income elasticity[a] of GDP[b] at time of death on all-cause mortality for those aged 65-99, by country and sex

Country	Income elasticity of GDP at time of death			
	Men	CI	Women	CI
Denmark	-8.8	(-9.4;-8.1)	-42.4	(-42.8;-42.0)
England and Wales	-35.8	(-36.0;-35.7)	-43.9	(-44.1;-43.8)
Finland	-29.7	(-30.2;-29.2)	-47.0	(-47.4;-46.7)
France	-32.9	(-33.0;-32.8)	-45.3	(-45.4;-45.2)
Netherlands	-4.9	(-5.4;-4.5)	-45.2	(-45.4;-44.9)
Norway	-5.3	(-5.9;-4.7)	-31.0	(-31.4;-30.6)
Sweden	-23.6	(-24.0;-23.1)	-51.7	(-52.0;-51.4)

[a] Income elasticity is measured by 100*(exp(parameter estimate)-1), and denotes the percentage mortality increase per percentage increase in national income (LN(GDP/c)).
[b] With log transformation of the GDP variable.

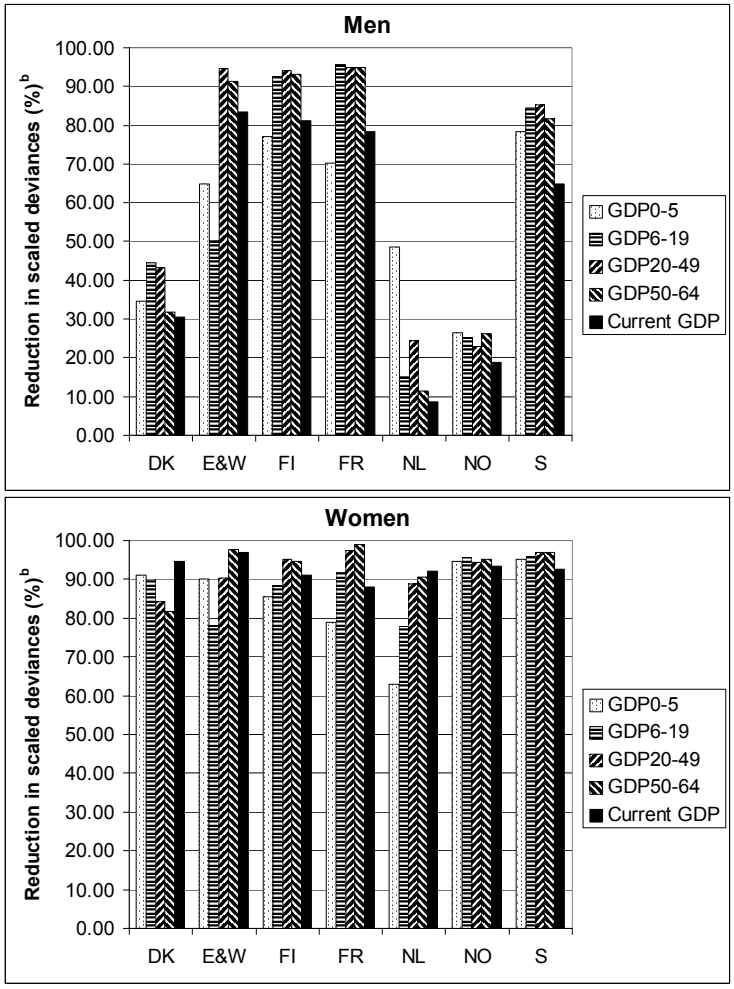

Figure 3. Comparison of the association of GDP[a] measured at different ages of subsequent cohorts born between 1865 and 1924 with all-cause mortality among those aged 65-99, by country and sex

[a] With log transformation of the GDP variables.
Reduction in scaled deviance (a measure of unexplained variance) compared to the model with age only. This reduction is expressed as percentage of the total reduction in scaled deviances between the model with age only and the model with age, period, and cohort terms. The higher the reduction in scaled deviances, the better the fit of the model has improved on including an additional variable. All reductions in scaled deviances are statistically significant (p = 0.05).
DK = Denmark; E&W = England and Wales; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden; Current GDP = GDP at time of death.

Among men in England and Wales, Finland, and France, the reduction in scaled deviances was around 80 per cent. Among women in all countries, this reduction was even higher.

Regression models that included GDP levels prevailing at earlier ages of the cohorts, instead of GDP at time of death, produced larger reductions in scaled deviances. Only among Danish and Dutch women, GDP at time of death explained more of the mortality variation than GDP at earlier ages (Figure 3).

When variables indicating GDP levels prevailing at different earlier ages of the cohorts are added to the model including age and GDP at time of death, the reductions in scaled deviances increased significantly ($p = 0.05$), indicating a significant improvement of the fit of the model (Table 2). Thus GDP levels prevailing at earlier ages of the subsequent cohorts were related to old-age mortality independently of GDP levels at time of death. This independent association is observed for men and women in all countries.

Table 2. Comparison of the independent association of GDP[a] measured at different ages of subsequent cohorts born between 1865 and 1924 with all-cause mortality among those aged 65-99, by country and sex

Model: Age and …	Reduction in scaled deviances (%)[b]						
	DK	E&W	FIN	FR	NL	NO	S
MEN							
Current GDP	31(–)	83(–)	81(–)	78(–)	9(–)	19(–)	65(–)
Current GDP and GDP at ages 0-5	35(–)	84(+)	82(–)	79(–)	66(–)	33(–)	81(–)
Current GDP and GDP at ages 6-19	60(–)	90(+)	93(–)	96(–)	16(–)	34(–)	94(–)
Current GDP and GDP at ages 20-49	49(–)	95(–)	95(–)	97(–)	40(–)	32(–)	96(–)
Current GDP and GDP at ages 50-64	33(–)	95(–)	95(–)	95(–)	12(–)	40(–)	87(–)
Current GDP and cohort GDP	72(–)	97(–)	96(–)	99(–)	67(–)	44(–)	98(–)
WOMEN							
Current GDP	95(–)	97(–)	91(–)	88(–)	92(–)	93(–)	93(–)
Current GDP and GDP at ages 0-5	95(–)	97(–)	91(–)	88(–)	92(–)	96(–)	96(–)
Current GDP and GDP at ages 6-19	95(–)	97(+)	93(–)	94(–)	94(–)	96(–)	96(–)
Current GDP and GDP at ages 20-49	95(+)	97(–)	96(–)	98(–)	94(–)	95(–)	97(–)
Current GDP and GDP at ages 50-64	95(–)	98(–)	95(–)	99(–)	95(–)	95(–)	97(–)
Current GDP and cohort GDP	97(–)	99(–)	96(–)	99(–)	95(–)	98(–)	98(–)

[a] With log transformation of the GDP variables.

[b] Reduction in scaled deviance (a measure of unexplained variance) for the stepwise inclusion of GDP at time of death, GDP at ages 0-5, 6-19, 20-49, and 50-64 in the model, compared to the model with age only. This reduction is expressed as percentage of the total reduction in scaled deviances between the model with age only and the model with age, period, and cohort terms. The higher the reduction in scaled deviances, the better the fit of the model has improved on including an additional variable. All reductions in scaled deviances are statistically significant ($p = 0.05$).

( ) = either positive (+) or negative (-) independent association of GDP prevailing at the specific ages (i.e. positive or negative income elasticity, respectively).

Underlined = highest reduction in scaled deviances for GDP prevailing at the specific ages in comparison to GDP prevailing at different ages of the cohorts.

DK = Denmark; E&W = England and Wales; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden. Current GDP = GDP at time of death. Cohort GDP = GDP0005 GDP0619 GDP2049 GDP5064.

The extent to which GDP in earlier life explains time trends in old-age mortality can be assessed by comparison of the reduction in scaled deviances for the model including age and GDP at time of death (first rows of Table 2) with the reduction in scaled deviances for the model including GDP at earlier ages of the cohort as well (last rows of Table 2). According to Table 2, earlier-life GDP explained a substantial part of the time trends among Dutch men (change in reduction in scaled deviances from 9% to 67%), and also among Danish, Swedish and Norwegian men. The percentage explained is somewhat less among men in the other countries, and limited among women in all countries.

Turning to the causes of death, significant independent effects of GDP levels prevailing at earlier ages of the cohort were observed for all causes of death, except in a number of countries for prostate cancer and breast cancer (Table 3)(See Appendix II for more details).

Table 3. The age groups of the subsequent cohorts born between 1865 and 1924 for which GDP showed the largest associations with mortality for those aged 65-99, by cause of death, sex and country

Cause of death	The age groups for which GDP showed the largest reduction in scaled deviances when added to the model including age and GDP at time of death						
	DK	E&W	FIN	FR	NL	NO	S
MEN							
All-cause mortality	6–19(–)	20–64(–)	20–64(–)	20–49(–)	0–5(–)	50–64(–)	20–49(–)
Ischaemic heart diseases	20–49(–)	20–64(–)	6–19(–)	20–64(–)	20–49(–)	6–19(–)	20–49(–)
Cerebrovascular diseases	6–19(–)	20–49(–)	20–49(–)	50–64(–)	20–49(–)	6–19(–)	0–64(–)
Stomach cancer	6–64(–)	20–49(–)	20–64(–)	50–64(–)	20–49(–)	6–19(–)	20–64(–)
Prostate cancer	0–19(+)	50–64(+)	NS	50–64(–)	NS	0–19(–)	6–49(+)
Lung cancer	20–49(–)	20–49(–)	20–49(–)	6–64(–)	0–5(–) 20–49(–)	50–64(–)	20–49(–)
COPD	6–49(–)	20–49(–)	20–49(–)	6–19(–) 50–64(–)	0–5(–)	50–64(–)	20–49(–)
WOMEN							
All-cause mortality	0–64(–)	50–64(–)	20–49(–)	50–64(–)	50–64(–)	0–19(–)	20–64(–)
Ischaemic heart diseases	50–64(–)	50–64(–)	6–19(–)	50–64(+)	20–49(–)	6–19(–)	20–64(–)
Cerebrovascular diseases	50–64(–)	20–49(–)	20–49(–)	50–64(–)	50–64(–)	6–19(–)	0–64(–)
Stomach cancer	0–49(–)	20–49(–)	20–64(–)	50–64(–)	50–64(–)	0–64(–)	0–5(–) 20–64(–)
Breast cancer	20–49(+)	0–5(+)	20–64(+)	50–64(+)	0–5(+)	NS	20–64(–)
Lung cancer	20–49(+)	0–19(+) 50–64(+)	6–19(+)	20–49(+)	20–49(+)	6–19(+) 50–64(+)	6–19(+)
COPD	20–49(+)	20–49(–)	0–5(–)	0–5(–)	20–49(+)	6–19(+)	20–49(+)

( ) = either positive (+) or negative (-) independent association of GDP prevailing at the specific ages (i.e. positive or negative income elasticity, respectively).
NS = Non-significant (p = 0.05) reduction in scaled deviances when the GDP variables at different ages are included in the regression model with age and GDP at time of death as independent variables.
DK = Denmark; E&W = England and Wales; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden; COPD = chronic obstructive pulmonary diseases.

Among men, the largest reduction in scaled deviances – after control for GDP at time of death - is observed for GDP prevailing at ages 20-49, both for all-cause mortality, and for the majority of causes of death. However, larger reductions in scaled deviances were observed for GDP prevailing at ages 6-19 and 50-64 in Norway, GDP prevailing at ages 50-64 in France, and GDP prevailing at ages 6-19 in Denmark. For prostate cancer, the reductions in scaled deviances were highest for GDP prevailing at ages 6-19. Among women, the largest reduction in scaled deviances is observed for GDP prevailing at ages 50-64, both for all-cause mortality, and for the majority of causes of death. GDP prevailing at ages 20-49 seems equally important as GDP at ages 50-64 for stomach cancer and breast cancer, and more important for chronic obstructive pulmonary diseases (COPD). For lung cancer, the reductions in scaled deviances were highest for GDP prevailing at ages 6-19. For Norway, GDP prevailing at ages 6-19 showed the largest reduction in scaled deviances. The above-mentioned associations were mainly negative, except for prostate cancer, breast cancer, and lung cancer and COPD among women.

DISCUSSION

In this paper, we assessed whether old-age mortality levels for subsequent cohorts are associated differentially with GDP levels prevailing at different ages of the cohorts. We did so separately for seven low-mortality European countries, for men and women, and for all-cause mortality and seven specific causes of death.

For the subsequent cohorts, levels of GDP at time of death were strongly and inversely associated with all-cause mortality, especially among women, and among men in England and Wales, Finland, and France. In most countries, stronger associations were observed with GDP levels prevailing at earlier ages of the cohorts. These associations remained after control for GDP at time of death. An independent association of GDP at earlier ages of the cohort was also observed for specific causes of death. The associations were negative for ischaemic heart diseases, cerebrovascular diseases, stomach cancer, and positive for prostate cancer, breast cancer, COPD (women), and lung cancer (women). Of all ages, GDP prevailing at ages 20-49 (men) and ages 50-64 (women) were most strongly related to cohort differences in old-age mortality.

These results are in agreement with one of the key paradigms in public health, which states that economic progress is of fundamental importance to the improvement of population health (e.g. [4]), and with recent epidemiological individual-level studies that observed relations between socio-economic conditions in early life and (cause-specific) mortality in later life (e.g. [21,24-26])(see Galobardes et al., 2004 for a review)[27].

Evaluation of data and methods

The mortality and population data used in this study are obtained from countries considered having good or excellent population registries and vital registries [32]. In these countries, reported survivorship counts are highly accurate [32,33]. Moreover, all-cause mortality data and population data stem from the Human Mortality Database, in which the data has been corrected for errors (http://www.mortality.org/).

On the basis of historical national accounts data from different sources, we constructed

time series of GDP for seven countries covering the period 1865 to 1999 [29-31] (see Appendix I). In general, the measurement of GDP levels and trends is probably less accurate for the 19th century than for the 20th century [29]. If so, this may have contributed to our finding that mortality levels were less strongly related to the older measures of GDP related to early ages of our birth cohorts, as compared to the more recent measures of GDP related to adulthood and middle age.

In our analyses, we assumed that the socio-economic conditions prevailing at specific ages of the cohorts, and thus during a certain time period, could be measured by the national level of GDP prevailing during that period in time. However, this assumption may not fully apply. The alternative approach would be to use measures that are more applicable to specific age groups. For example, the infant mortality rate (IMR) might be used as a proxy of the living conditions at the time of birth of a cohort [18,23]. In additional analyses, the use of the IMR (data obtained from Mitchell (1992) and Turpeinen (1979))[29,34] yielded about the same results as we obtained with the use of GDP at the time of birth (Table 4). Similarly, specific indicators might need to be used to measure the material living conditions as experienced by a birth cohort during their old age, such as the level of old benefits and pensions. Unfortunately, in most countries, data on pensions or old-age benefits were not available in a comparable way for a prolonged period on a yearly basis [35-37]. If such data had been available, they might have shown an even higher predictive power of economic measures related to later life or time of death, as compared to measures related to infancy or childhood and adolescence.

Our analyses focused on associations across birth cohorts between their level of old-age mortality and the national level of GDP prevailing at different ages of these cohorts. Another approach would have been to analyse associations across countries instead of across birth cohorts. Additional cross-country analyses for all-cause mortality resulted in mainly negative associations with GDP at time of death, but mainly positive associations with GDP at earlier ages of the cohorts (results not shown). These counterintuitive results might be due to the small number of observations in these cross-country analyses, combined with the large potential for confounding in cross-national comparisons.

Table 4. Correlations of GDP and infant mortality (IMR) prevailing at birth[a] for subsequent cohorts born between 1865 and 1924 with all-cause mortality among those aged 65-99, by country and sex

	DK	E&W	FIN	FR	NL	NO	S
MEN							
GDP at birth	-0.84	-0.90	-0.94	-0.90	-0.92	-0.85	-0.96
IMR at birth	0.62	0.89	0.93	0.94	0.73	0.88	0.98
WOMEN							
GDP at birth	-0.96	-0.94	-0.96	-0.91	-0.90	-0.99	-0.99
IMR at birth	0.74	0.78	0.91	0.94	0.95	0.96	0.99

[a] With log transformation of the values.
All correlations are significant at the 0.05 level (2-tailed).
DK = Denmark; E&W = England and Wales; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden.

For international comparative research, more countries need to be included to obtain valid results. We furthermore recommend that in such cross-country analyses separate cohorts are distinguished in order to study changes over time in the influence of early life social circumstance on late life mortality [38,39].

Another complementary approach would have been to focus on the long-lasting effects of exceptional economic circumstances earlier in life, e.g. by focusing on old-age mortality of generations having experienced World War I and/or the Spanish influenza epidemic in their earlier years. Due to our focus on the effect of GDP at different age groups of the cohorts, the deviating patterns for GDP during World War I and/or the Spanish influenza epidemic are only clearly visible for GDP at the youngest ages. With respect to the cohort trends for GDP at later and larger age groups, this peak is dispersed over many cohorts. Scatter plots of cohort trends in GDP at age 0-5 and the mortality progress over the successive cohorts suggested that the deviating patterns for the cohort groups 1910-14 and 1915-19 were not reflected in irregularities in trends in all-cause mortality at old age (see Appendix III). It can be concluded that this approach does not generate compelling evidence for an effect of socio-economic circumstances at the youngest ages on mortality up to old ages, either.

Cohort trends in all-cause mortality among women were approximately linear over time. Similarly, cohort trends in mortality from cerebrovascular diseases and stomach cancer did not show strong irregularities [40]. In these cases, associations with factors that gradually change over time, such as the logarithmically transformed GDP, are likely to be strong (see also Appendix II). These strong associations therefore cannot be taken as hard evidence for a causal association. A harder test for the existence of a causal association could be provided by the study of correlates of stronger irregularities in all-cause mortality trends among men and in mortality trends from lung cancer, ischaemic heart diseases and chronic obstructive pulmonary diseases. The evidence from these cases suggested that the association between GDP at different ages and old-age mortality was moderate but clearly present (see Table 3 and Appendix II). Therefore we believe that the observed associations may be indicative of true causal associations.

Explanations of the observed results in light of the current literature

The observed negative associations between GDP at time of death and old-age mortality among different cohorts seem to contradict the weak relationship between GDP and mortality that was observed in international studies that compared developed countries at a single point of time (e.g. [11,41,42]) For example, comparisons between rich OECD countries with respect to GDP and life expectancy typically find correlations lower than +0.20 [11]. This lack of association is probably explained by the strong potential for confounding in comparisons between countries, related to international differences in idiosyncratic determinants such as diet and the progression of the smoking epidemic. Studies that circumvent this problem by comparing regions within specific countries, both in Europe and in Northern America, generally observed a strong association between socio-economic levels and male and female life expectancies [42,43].

The relatively weak associations between GDP at time of death and all-cause mortality among Danish, Dutch, and Norwegian men could possibly be explained by the particular way in which the smoking epidemic has evolved among men in these countries. Our cause-specific analyses revealed exceptionally strong positive (instead of negative) associations

between GDP at time of death and mortality from chronic obstructive pulmonary diseases and lung cancer, i.e. causes of death strongly related to smoking (see Appendix II). Furthermore, in additional analyses that excluded all causes of death strongly related to smoking (i.e. lung cancer, cancer of the oesophagus, upper respiratory tract, pancreas, bladder, and kidney, and chronic obstructive pulmonary diseases) the effect of GDP at time of death on old-age mortality among men was found to be more similar for the different countries (Table 5). Thus, smoking seems to explain the differences between countries in the extent to which GDP at time of death is related to old-age mortality among men. Similarly, differences between male and female birth cohorts in their life-time exposure to smoking can explain the large differences between men and women in the extent to which their mortality trends are related to current levels of GDP.

Table 5. Comparison of the independent association of GDP[a] measured at different ages of the subsequent cohorts born between 1865 and 1924 with mortality from all-cause mortality after excluding mortality from strongly smoking-related diseases[b] among those aged 65-99, by country and sex

Model: Age and …	Reduction in scaled deviances (%)[c]						
	DK	E&W	FIN	FR	NL	NO	S
MEN							
Current GDP	83	91	88	83	74	51	77
Current GDP and GDP at ages 0-5	87	91	89	84	86	65	88
Current GDP and GDP at ages 6-19	94	93	95	96	78	66	97
Current GDP and GDP at ages 20-49	92	96	97	98	85	64	97
Current GDP and GDP at ages 50-64	87	96	96	97	78	69	92
Current GDP and cohort GDP	94	97	97	100	88	71	99
WOMEN							
Current GDP	96	96	91	88	91	92	92
Current GDP and GDP at ages 0-5	97	96	91	88	91	95	95
Current GDP and GDP at ages 6-19	96	97	93	94	94	96	96
Current GDP and GDP at ages 20-49	96	98	96	98	94	94	96
Current GDP and GDP at ages 50-64	97	98	95	99	96	95	96
Current GDP and cohort GDP	98	99	96	99	96	98	97

[a] With log transformation of the GDP variables.
[b] Strongly smoking-related diseases include chronic obstructive pulmonary diseases and cancer of the lung, esophagus, upper respiratory tract, pancreas, bladder, and kidney.
[c] Reduction in scaled deviance (a measure of unexplained variance) for the stepwise inclusion of GDP at time of death, GDP at ages 0-5, 6-19, 20-49, and 50-64 in the model, compared to the model with age only. This reduction is expressed as percentage of the total reduction in scaled deviances between the model with age only and the model with age, period, and cohort terms. The higher the reduction in scaled deviances, the better the fit of the model has improved on including an additional variable. All reductions in scaled deviances are statistically significant ($p = 0.05$).
Underlined = highest reduction in scaled deviances for GDP prevailing at the specific ages in comparison to GDP prevailing at different ages of the cohorts.
DK = Denmark; E&W = England and Wales; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden. Current GDP = GDP at time of death. Cohort GDP = GDP0005 GDP0619 GDP2049 GDP5064.

Our analyses suggest an independent effect of GDP prevailing at earlier ages of subsequent cohorts on all-cause old-age mortality. Previous studies on the link between economic circumstances in early life and all-cause mortality focused mainly on the effects that dramatic economic events, like war and influenza, occurring at very young ages could have on mortality later in life [44-48]. Some studies did observe effects on subsequent mortality, but these effects did not always persist with increasing age [47,48]. In our study, an association between GDP prevailing at ages 0-5 and old-age mortality was observed in the Netherlands. The same result was found in an earlier study on the effects of macro-economic conditions at ages 1-7 on the individual all-cause mortality rate at ages above 50 among Dutch cohorts born between 1817 and 1907 [49]. However, our cause-specific analyses suggested no association between GDP at very early ages and old-age mortality from stomach cancer and cerebrovascular diseases, i.e. causes of death that have been found to be related to early life circumstances [21,23]. Also, when looking at the results for the other six European countries studied, we found only modest associations between GDP prevailing at ages 0-5 and old-age mortality. In general, cohort differences in old-age mortality seemed more strongly related to GDP prevailing at ages 20-49 (men) and ages 50-64 (women). Thus, although some effects of socio-economic circumstances in very early life on old-age mortality were observed, our results suggest a more important role of socio-economic circumstances in adulthood and middle age in determining mortality up to high ages.

The fact that socio-economic circumstances in very early life are not clearly related to old-age mortality, while they have been reported to be related to middle age mortality, could possibly be explained by mortality selection. When mortality at middle ages is high, it tends to affect the frail individuals first, leaving a more selected and more robust population that survive up to old ages [50]. Due to this selection mechanism, adverse socio-economic circumstances in very early life might still have an effect on middle-age mortality, but this effect may no longer be visible in old-age mortality, where only a selected and robust part of the cohort have survived. However, there is little evidence to support a selection mechanism. If such a selection effect would operate, one would expect relatively higher levels of mortality at middle age to be followed by relatively low mortality at older ages. However, in a previous study on the same countries, we observed positive (instead of inverse) associations between trends in late middle-age mortality and trends in old-age mortality [51]. Thus, mortality selection effects, if any, probably do not have strong effects on trends in old age mortality [51].

When causes of death were distinguished, the direction of the effect was found to greatly vary. Independent negative associations of GDP prevailing at earlier ages on old-age mortality were found for ischaemic heart diseases, cerebrovascular diseases, stomach cancer, and lung cancer (men), whereas independent positive associations were found for prostate cancer, breast cancer, COPD (women), and lung cancer (women). These results are in line with previous studies on the direction of the associations observed for adult mortality and on the role of cause-specific risk factors. Higher adult mortality from ischaemic heart diseases, cerebrovascular diseases and stomach cancer has been related to adverse socio-economic circumstances [27]. Mortality from breast cancer and prostate cancer is known for its inverse associations with deprivation [52], and their positive socio-economic gradients [53]. Lung cancer mortality among women also has been found to negatively correlate with deprivation [52]. The seemingly unfavourable effects of economic

growth on old-age mortality from COPD and lung cancer among elderly women could probably be linked to a high recent uptake of smoking and other unfavourable lifestyles among adolescent and adult women in prosperous countries [52].

Conclusion / Implications

Recent insights from life-course epidemiology stress the effects that experiences in early life may have on health and mortality in later life. Effects of mortality in early life on national mortality trends at later ages have been suggested by for example age-period-cohort studies. This paper adds evidence on the specific role of socio-economic conditions in earlier phases of life on old-age mortality of subsequent cohorts. An independent, mostly negative effect of GDP prevailing at earlier ages of subsequent cohorts on old-age mortality was observed. The most relevant ages of exposure seem to be adulthood and middle age, rather than infancy or childhood. Further studies should aim to identify the specific mechanisms that are involved, including the development of health-related behaviour in early adulthood and the role of accumulation of wealth and other resources during late adulthood.

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Appendix I. Reconstruction of trends in real national GDP per capita, 1865-1999

For each country, for the period 1865 to 1999, trends in national real Gross Domestic Product per capita at constant 1995 prices in millions of national currency were reconstructed from historical national accounts data [29-31]. For the period 1865 to 1970, the historical national accounts data by Mitchell were used [29]. For the Netherlands, these historical data were available only from 1900 onwards, and in addition, we used national accounts data from Statistics Netherlands (see [31]). Data from 1970 onwards were obtained from the "National Accounts of OECD Countries Database" [30]. To obtain estimates on GDP per capita (GDP/c), the GDP data were divided by the population estimates given by Mitchell (up to 1970) and by the OECD Database (from 1970 onwards). In the remainder of the text, GDP refers to GDP per capita at constant 1995 prices in millions of national currency.

The reconstruction of the trends in national GDP entailed a number of problems. Changes over time occurred in both the national accounts data, for example due to changes in concepts, different pricing level years or different sources used, and in the population estimates. All countries experienced changes in the national accounts data, ranging from one change in Finland to seven changes in Norway. Changes in the population estimates data applied to France, Denmark, and the United Kingdom. In addition, for all countries, the data for 1970 by Mitchell had to be recalculated into the OECD data for 1970. We tackled these problems by reconstructing the different data into the most recent situation. In the majority of the cases, double counts were available for the year in which the change occurred, which simplified our reconstruction. In these cases, we calculated ratios, i.e. GDP after the change divided by GDP preceding the change, and we applied these ratios to the GDP data preceding the change. When double counts were not available for the year in which the change occurred, we first estimated the double count by linearly extrapolating the data for the preceding five years, and then followed the same procedure as laid out before.

An additional problem entailed the missing national accounts data during the First and Second World War, and shortly after the war period. This applied to France (1914-1919 and 1939-1948), to the Netherlands (1940-1947), and to Norway (1940-1945). First, we estimated the GDP data for the years shortly after the war period (France 1918-19 and 1945-48, the Netherlands 1945-47, and Norway 1945) by linearly backward extrapolating the GDP values for the subsequent five years, for each year separately. For the war years we assumed that GDP in these years was equal to either the first year before the war or the first year after the war, depending on what year showed the lowest value of GDP. For France and Norway during the Second World War the reconstruction of the trend was further complicated by an additional change in concepts during that period which made the data before the War incomparable with the data after the War. In these two cases we assumed that the GDP values during the World War and for the first year after the World War were equal to the GDP value in the first year before the World War.

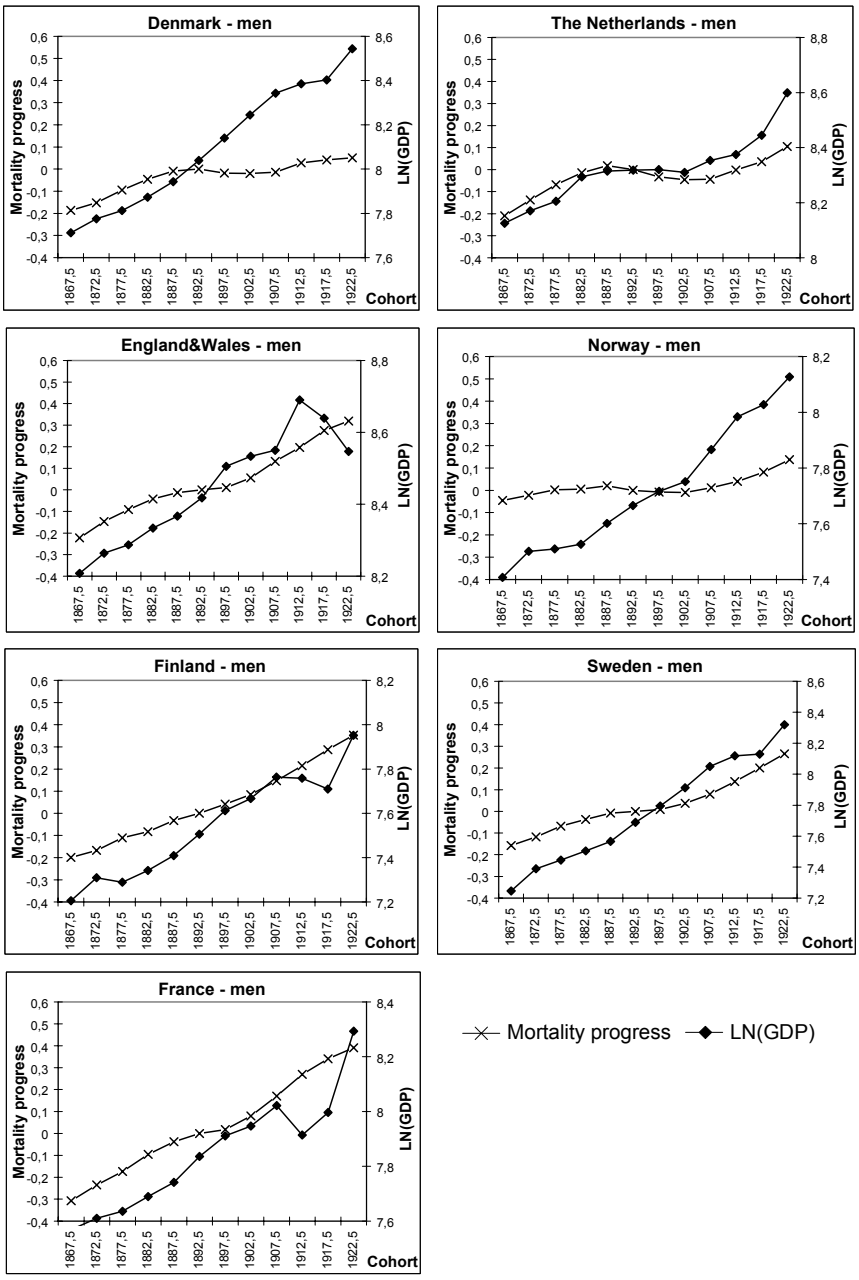
For the United Kingdom, historical data on national accounts were available only for the country as a whole. Assuming that the time trends in GDP/c in the United Kingdom were broadly similar to those for England and Wales, we included the GDP/c trend data for the United Kingdom as a proxy for the GDP/c trend data for England and Wales.

Appendix II. Comparison of the independent association of GDP[a] measured at different ages of the subsequent cohorts born between 1865 and 1924 with mortality from specific causes of death among those aged 65-99, by country and sex

Model: Age and …	Reduction in scaled deviances (%)[b]													
	Men							Women						
	DK	E&W	FIN	FR	NL	NO	S	DK	E&W	FIN	FR	NL	NO	S
Ischaemic heart diseases														
GDPcurrent	0	10	3	31	2	4	2	29	59	10	17	44	31	47
GDPcurrent GDP0005	66	17	4	34	48	25	66	85	59	10	17	57	47	90
GDPcurrent GDP0619	83	20	71	77	37	32	82	80	60	60	59	74	52	91
GDPcurrent GDP2049	85	46	64	85	67	25	88	84	69	46	66	84	44	96
GDPcurrent GDP5064	76	46	56	85	43	31	79	92	73	40	73	80	47	96
GDPcurrent GDPcohort	95	56	80	89	69	33	89	98	77	67	76	87	58	97
Cerebrovascular diseases														
GDPcurrent	95	88	87	67	88	84	96	94	87	89	65	90	87	89
GDPcurrent GDP0005	96	89	88	69	91	90	97	94	87	90	67	91	93	89
GDPcurrent GDP0619	97	92	91	85	94	91	97	94	91	91	78	95	94	89
GDPcurrent GDP2049	96	98	96	97	97	88	97	94	98	95	93	96	91	89
GDPcurrent GDP5064	96	96	95	99	95	90	97	95	93	94	97	97	93	89
GDPcurrent GDPcohort	97	99	96	100	97	94	97	96	99	96	98	97	97	89
Stomach cancer														
GDPcurrent	91	87	92	87	91	97	90	94	95	93	88	93	99	93
GDPcurrent GDP0005	97	88	93	87	93	98	98	99	(95)	94	88	93	99	99
GDPcurrent GDP0619	98	93	95	93	97	99	98	99	97	95	91	97	99	98
GDPcurrent GDP2049	98	99	98	97	98	98	99	99	99	98	96	98	99	99
GDPcurrent GDP5064	98	94	98	98	98	98	99	98	98	98	99	99	99	99
GDPcurrent GDPcohort	99	99	99	99	99	99	99	99	99	98	99	99	99	99
Prostate cancer (men) / breast cancer (women)														
GDPcurrent	52	69	73	55	81	83	86	0*	79	83	88	49	76	11
GDPcurrent GDP0005	81	(69)	(74)	(55)	(81)	85	(86)	17	81	(83)	89	55	(78)	60
GDPcurrent GDP0619	81	69	(73)	61	(81)	85	87	26	(79)	86	89	(49)	(79)	59
GDPcurrent GDP2049	80	70	(74)	62	(81)	84	87	56	(79)	88	91	(49)	(78)	65
GDPcurrent GDP5064	78	74	(74)	65	(81)	84	(86)	40	80	88	92	54	(79)	65
GDPcurrent GDPcohort	88	75	82	67	(81)	89	90	67	86	91	93	78	(80)	68
Lung cancer														
GDPcurrent	88	27	22	92	76	92	70	88	98	79	82	76	92	84
GDPcurrent GDP0005	88	37	23	92	93	96	78	90	99	(79)	85	92	97	84
GDPcurrent GDP0619	92	57	62	96	84	96	83	94	99	93	88	86	98	91
GDPcurrent GDP2049	94	92	67	96	93	95	87	97	(98)	92	89	98	97	89
GDPcurrent GDP5064	92	66	65	96	83	97	86	95	99	91	85	89	98	85
GDPcurrent GDPcohort	99	94	79	97	96	98	90	98	99	94	92	98	98	95
Chronic obstructive pulmonary diseases														
GDPcurrent	92	49	2	9	81	90	85	70	33	4	4	2	35	54
GDPcurrent GDP0005	92	50	5	(9)	89	90	87	71	39	12	14	32	79	57
GDPcurrent GDP0619	95	75	30	45	82	90	90	77	59	8	4	35	89	65
GDPcurrent GDP2049	95	88	63	40	85	90	91	86	82	6	10	56	81	66
GDPcurrent GDP5064	93	76	62	45	81	91	89	82	60	7	5	38	87	61
GDPcurrent GDPcohort	96	89	69	50	92	94	93	95	83	37	19	57	91	73

[a] With log transformation of the GDP variables.
[b] Reduction in scaled deviance (a measure of unexplained variance) for the stepwise inclusion of GDP

at time of death, GDP at ages 0-5, 6-19, 20-49, and 50-64 in the model, compared to the model with age only. This reduction is expressed as percentage of the total reduction in scaled deviances between the model with age only and the model with age, period, and cohort terms. The higher the reduction in scaled deviances, the better the fit of the model has improved on including an additional variable.
(..) Non-significant (p = 0.05) reduction in scaled deviances
Underlined = highest reduction in scaled deviances for GDP prevailing at the specific ages in comparison to GDP prevailing at different ages of the cohorts.
DK = Denmark; E&W = England and Wales; FIN = Finland; FR = France; NL = the Netherlands; NO = Norway; S = Sweden. GDPcurrent = GDP at time of death. GDPcohort = GDP0005 GDP0619 GDP2049 GDP5064.



Appendix III. Scatterplots of cohort trends in GDP prevailing at age 0-5 and the all-cause mortality progress among men aged+ 65-99 over the successive cohorts born between 1865 and 1924, by country

8

END-OF-LIFE DECISIONS AND OLD-AGE MORTALITY: A CROSS-COUNTRY ANALYSIS

Objective Although the life-shortening effect of medical end-of-life decisions on the individual has been estimated to be small, questions have been raised in literature on a possible association between end-of-life decision-making practices and mortality rates. We examined the association between physicians' practices and attitudes concerning end-of-life decision-making and mortality by age and cause of death across six European countries. **Methods** We applied correlation analyses. **Results** Although no statistically significant associations were found, the prevalence of end-of-life decisions tended to correlate positively with mortality. Correlation coefficients were higher for mortality among the elderly (80+) and for the prevalence of doctor-assisted dying. The share of physicians who feel they should unconditionally preserve their patients' lives tended to correlate negatively with mortality, especially with old-age mortality. No clear pattern was observed for the different causes of death. **Conclusion** We recommend further research into the possibility that national old-age mortality rates reflect attitudes concerning appropriate medical treatment for the elderly.

BACKGROUND

Medical decision-making for patients with life-threatening diseases involves a balanced consideration of medical, ethical, psychosocial and societal aspects. Advances in medicine have greatly improved the possibilities to treat seriously ill patients and to prolong life. However, it is increasingly recognised that prolonging life might not always be an appropriate goal of medicine and that other goals have to guide medical decision-making at the end of life, such as improving the quality of life of patients and their families through the prevention and relief of suffering [1]. In some cases, hastening of death may be an accepted or, by some, even appreciated consequence of end-of-life care.

Currently, medical care for dying patients often involves decisions that may hasten death, mainly on withholding or withdrawing of life-prolonging treatments or on the administration of potentially life-shortening drugs for the alleviation of severe suffering [2].

Although, the life-shortening effect of these medical end-of-life decisions on the individual has been estimated to be rather small [2], there is some evidence which suggests that cross-national differences in end-of-life decision making may be reflected in mortality rates. In the Netherlands, high neonatal mortality rates [3] and the stagnation of perinatal mortality decline [4] have been partly attributed to a higher frequency of withholding or withdrawing life-prolonging treatments in severely ill neonates and extremely preterm infants among Dutch physicians [3,5]. Further, an increasing prevalence of end-of-life decisions over time in the Netherlands [6] concurred with a stagnation of mortality decline among Dutch elderly since the 1980s [7].

We therefore decided to examine the association between physicians' practices and

attitudes concerning medical end-of-life decision-making, as observed in a recent study in six European countries, with mortality by age and cause of death.

DATA AND METHODS

Prevalence data on medical end-of-life decisions by age, sex, and cause of death of the patient were obtained for Belgium (Flanders), Denmark, Italy (areas of Emilia-Romagna, Trentino Alto Adige, Tuscany, and Veneto), the Netherlands, Sweden, and Switzerland (German-speaking part) in 2001/2002 (EURELD study I) [2]. Doctors reported which end-of-life decisions had preceded dying, for an unselected sample of all deaths in the areas studied. We distinguished (a) physician-assisted dying, i.e. administration of drugs with the explicit intention of hastening death (b) use of drugs with a possible life-shortening effect to alleviate symptoms, and (c) non-treatment decisions, i.e. decisions to withhold or withdraw potentially life-prolonging treatment. Deaths of infants younger than 1 year of age were not included in the sample. In the Italian sample, age group 1-17 years was not included in the Italian sample. In addition, physicians were asked to what extent life might have been shortened by the different end-of-life decisions.

The EURELD study also assessed physicians' attitudes towards medical end-of-life decision-making (EURELD study II) [8]. A stratified sample of doctors was asked to what extent they agreed with different statements on end-of-life decision-making. We used the weighted percentages of doctors who (strongly) agreed with the statement "in all circumstances physicians should aim at preserving the lives of their patient, even if patients ask for the hastening of the end of their lives".

For the six countries included in the EURELD study, we collected data on (cause-specific) mortality and life expectancy, for the years 1998 to 2001 combined. These data were obtained through the Human Mortality Database, the WHO Mortality Data Base, ISTAT (Italy, same four areas combined), NIS (Flanders), Statistics Netherlands, and Statistics Denmark. Swiss mortality data were obtained for the entire country. For Denmark, cause-specific mortality data were available for 1998-2000. For Switzerland mortality data for the total country was included, whereas the data on medical end-of-life decisions only applied to the German speaking part. For the Italian regions, the life tables for 2001 were provisional estimates. Based on these data, age-standardized mortality rates were calculated. The life expectancy data were averaged over the years 1998 to 2001. For Italy, complete life tables and population data by region, sex and single years of age were obtained in order to combine the life expectancy data for the four regions into a weighted average for the four regions combined.

To these data, we applied correlation analyses using SPSS version 11.0 for Windows.

RESULTS

EURELD study I revealed that the percentage of deaths that was preceded by any end-of-life decision ranged from 23% (Italy) to 44% (the Netherlands) and 51% (Switzerland) [2]. According to physicians' estimates, these end-of-life decisions resulted in an average shortening of the patient's life by 2.4 days for Italy and Sweden to 14.6 days for the Netherlands (Table 1). At the population level, this led to a reduction in life expectancy of at maximum 0.02 years in the Netherlands and Switzerland. (Table 1)

Table 1. Physicians' estimates of the potential life shortening of the individual by any end-of-life decision and the average effect of end-of-life decision-making at the population level, by country

	BE	CH	DK	IT	NL	SE
Frequencies of physicians' estimates of the potential life shortening of the individual by any end-of-life decision (weighted %)						
Life was probably not shortened at all	38	47	44	55	32	52
Less than 24 hours	14	10	18	7	17	14
Up to one week	25	22	16	7	22	13
One to four weeks	9	8	3	2	10	3
One to six months	3	4	1	0	4	0
More than six months	1	1	1	0	1	0
Unknown	11	8	17	29	14	18
Average estimated life-shortening (days)*	8.93	12.66	5.08	2.40	14.57	2.42
Prevalence of end-of-life decisions (%) [2]	38	51	41	23	44	36
Average effect at population level (days)†	3.39	6.46	2.08	0.55	6.41	0.87
Average effect at population level (years)†	0.01	0.02	0.01	0.00	0.02	0.00

* Based on the assumptions: life was probably not shortened at all ≈ 0 days; less than 24 hours ≈ 1 day; up to one week ≈ 4 days; one to four weeks ≈ 18.5 days; one to six months ≈ 106.5 days; More than six months ≈ 365 days; unknown has been excluded.
† The average estimated life-shortening multiplied by the prevalence of end-of-life decisions.
BE = Belgium (Flanders); CH = Switzerland (German-speaking part); DK = Denmark; IT = Italy (areas of Emilia-Romagna, Trentino Alto Adige, Tuscany, and Veneto); NL = the Netherlands; SE = Sweden.

Our correlation analysis revealed weak negative correlations between the prevalence of end-of-life decisions and life expectancy at birth (Table 2), and weak positive correlations between the prevalence of end-of-life decisions and all-cause, all-age mortality. Correlations were higher for people dying at the age of 80 and over (Table 2). The prevalence of physician-assisted dying correlated more strongly with mortality than the use of potentially life-shortening drugs to alleviate symptoms, or than the prevalence of decisions to withhold or withdraw potentially life-prolonging treatment.

The share of physicians who feel they should unconditionally preserve their patients' lives correlated weakly positively with life expectancy, and weakly negatively with all-cause mortality (Table 2). Correlations were stronger for people dying at the age of 80 and over (Table 2, Figure 1). None of these correlations reached statistical significance, though.

No clear pattern was found for different causes of death. Cancer is the cause of death that most often involves end-of-life decisions [2], but correlations for cancer were not higher than correlations for other causes of death. For cardiovascular disease mortality, correlations were small or reversed. (Table 2)

DISCUSSION

Thus, although the end-of-life decisions, based on the physicians' estimates of the life-shortening effect, did not result in a large effect on national life expectancy, a tendency towards association between physicians' practices and attitudes concerning end-of-life decision-making and mortality appeared, especially for the elderly.

Table 2. Correlation of the prevalence of end-of-life decisions and physicians' agreement with the life-preserving statement with life expectancy and cause-specific mortality 1998-2001, by sex and age, across six European countries (Pearson's correlations)

Mortality measures	Prevalence of end-of-life decisions*		Prevalence of physician-assisted dying*		Prevalence of alleviation of pain and symptoms with possible life-shortening effect*		Prevalence of non-treatment decisions*		Agreement with the life-preserving statement	
	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Life expectancy										
At birth	–0.19	–0.29	–0.36	–0.31	–0.25	–0.32	–0.04	–0.13	0.26	0.28
At age 65	–0.16	–0.26	–0.50	–0.25	–0.16	–0.28	–0.05	–0.12	0.27	0.23
At age 80	–0.35	–0.47	–0.72	–0.62	–0.32	–0.47	–0.30	–0.38	0.50	0.51
Age-standardised mortality at age 80 and over										
All causes	0.39	0.55	0.78	0.73	0.35	0.51	0.37	0.51	–0.55	–0.65
Cardiovascular diseases	0.14†	0.13†	–0.54†	–0.84†‡	0.30†	0.15†	–0.17†	–0.07†	0.55	0.57
Cancers	0.02	–0.11	0.78	0.45	–0.13	–0.15	0.03	–0.24	–0.33	–0.04
Respiratory diseases	0.09	0.20	0.90‡	0.88‡	–0.15	–0.02	0.26	0.34	–0.60	–0.67
Diseases of the nervous system	0.59†	0.63†	0.39†	0.45†	0.51†	0.49†	0.52†	0.57†	–0.45	–0.45
Other causes	0.61	0.63	0.68	0.84‡	0.54	0.50	0.55	0.61	–0.59	–0.67

* The prevalence of end-of-life decisions was measured per age group and per cause of death, but not per sex.

† For Belgium, Denmark, the Netherlands, and Switzerland, the frequencies of end-of-life decisions for cardiovascular diseases exclude cerebrovascular diseases, and the frequencies of end-of-life decisions for diseases of the nervous system include cerebrovascular diseases.

‡ Significant at the 0.05 level (two-tailed).

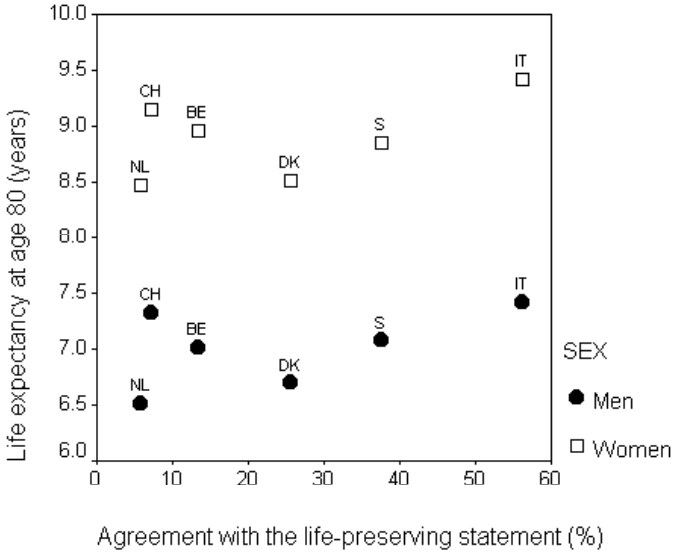


Figure 1. Physicians' agreement with the life-preserving statement plotted against sex-specific remaining life expectancy at age 80 for the years 1998-2001, across six European countries

Pearson's correlation coefficient: 0.50 (men), 0.51 (women).
BE = Belgium (Flanders); CH = Switzerland (German-speaking part); DK = Denmark; IT = Italy (areas of Emilia-Romagna, Trentino Alto Adige, Tuscany, and Veneto); NL = the Netherlands; SE = Sweden.

Despite the non-significant results, our analysis does not preclude a possible relationship between the practice of end-of-life decision-making or the propensity to preserve life in elderly patients and national old-age mortality levels. Obviously, the small number of countries included limits the power of our analysis.

In line with an association between the practice of end-of-life decision-making and mortality rates is the notion that the true effect of general medical management of severely ill or elderly patients on national mortality may not have been completely uncovered in our analysis. Cross-national differences in what is being regarded as appropriate medical treatment for chronically ill and elderly patients could lead to differences in what is defined as a decision to refrain from such treatment. As a result, some differences in medical management of severely ill or elderly patients may not be identified in studies on end-of-life decision-making. This is supported by our finding that the correlations with mortality were slightly higher for physicians' attitudes than for physicians' practices. The observed lower correlations for non-treatment decisions, which are more prone to differences in what is being perceived as appropriate medical treatment, and higher correlations for doctor-assisted dying, a practice that probably better reflects the underlying attitude, are also in line with this hypothesis.

On the other hand, the tendency for an association might be biased, because we did not take into account variables that may be more important than end-of-life decisions in determining cross-national differences in mortality, such as cardiovascular risk factors and

health care expenditure during the last year of life. The counterintuitive results for cardiovascular disease mortality, however, could indicate a likely underestimation of the observed association for all-cause mortality due to cross-national differences in cardiovascular risk factors, like unhealthy behaviour. With respect to health care expenditure, the contrast between Switzerland and the Netherlands can be used as an illustration. In Switzerland a relatively high prevalence of end-of-life decisions and a relatively low agreement with the life-preserving statement concur with a relatively high life expectancy, whereas in the Netherlands the same factors concur with a relatively low life expectancy. It is striking that in Switzerland, health care expenditure during the last year of life is extremely high and amounts to 30% of the total health-care budget [9]. In the Netherlands this percentage amounts to only 10% [10]. Thus, in these countries the health care expenditure during the last year of life may be more important in determining the mortality levels. On the other hand, however, the contrast between the Netherlands and Switzerland could also again be due to cross-national differences in what is being regarded as appropriate medical treatment for chronically ill and elderly patients.

As yet, we cannot exclude the possibility that attitudes concerning appropriate medical treatment for the elderly can affect national old-age mortality rates. Further research into this issue is warranted.

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9

GENERAL DISCUSSION

The aim of this thesis was (a) to carefully describe trends in old-age mortality in seven European low-mortality countries from 1950 to 1999, and (b) to assess the role of specific factors in explaining the observed trends.

For this purpose, we studied both all-cause mortality and mortality from 26 specific causes of death, we applied a life-course perspective – with emphasis on both immediate effects of circumstances prevailing at old age (= period effects) and possible long-lasting effects of determinants located earlier in life (=cohort effects) – and we performed comparative studies among seven countries, i.e. Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden. In a series of separate analyses, we assessed the role of smoking, mortality selection, socio-economic developments over the life-course, and medical end-of-life decision-making. Smoking and socio-economic factors are important determinants of adult mortality, but uncertain is their role in determining trends in old-age mortality. Mortality selection effects on old-age mortality have been posited in literature. The empirical evidence, however, is rather mixed, and largely restricted to the mortality experience of single cohorts, without a specific focus on old-age mortality. The potential effect of medical end-of-life decision-making on national mortality has not been studied before. It was hypothesized that its effect, if any, is larger among the elderly population.

In this final chapter, we summarize the main results of this thesis, and evaluate the usefulness and possible limitations of our methodological approach in obtaining these results. Subsequently, we elaborate on the possible determinants of the stagnation of mortality decline among the elderly in the Netherlands, and what future trends in mortality we can anticipate. In addition, recommendations for further research are proposed.

SUMMARY OF RESULTS

Trends in old-age mortality in seven European countries, over the period 1950 to 1999

The trends in old-age mortality can be described by a combination of period and cohort patterns. The period patterns revealed an overall decline in all-cause mortality among those aged 80 and over from 1950 to 1999, with a convergence in the mortality level between countries over time. However, there was large heterogeneity in the pace of decline, with periods of stagnation being widespread. From the 1980s onwards, small mortality declines or even increases were observed in Denmark, the Netherlands, and among Norwegian men. In contrast, old-age mortality decline continued in England and Wales and France. For the subsequent cohorts (1865 to 1935), all-cause mortality among those aged 60 and over generally declined. This decline, however, stagnated among Danish, Dutch and Norwegian men born between 1890 and 1915, and among women from all countries born after 1920.

The role of the different determinants in explaining the observed trends

Smoking

The unfavourable cohort patterns in all-cause mortality among men born between 1890 and 1915 in Denmark, the Netherlands, and Norway, and among women born after 1920 in all countries, were largely parallel to the less favourable cohort patterns from smoking-related diseases for the same countries and the same cohorts. This implies that cohort changes in smoking exposure influenced recent trends in old-age mortality to a large extent. Period patterns for mortality from smoking-related diseases showed an overall pattern of long-term increases, for both men and women, which again points to a role of smoking in old-age mortality trends. However, among men, the increase in mortality from smoking-related diseases turned into a decrease since the 1980s, in all countries except Norway. Furthermore, when for the Netherlands the smoking-related diseases are excluded from period trends in all-cause mortality, mortality decline still stagnated, uniformly for men and women. Thus although smoking has had a marked influence on the trends in old-age mortality, it cannot fully explain the observed stagnation in mortality decline since the 1980s.

Mortality selection

Mortality from diseases specifically related to old age (infectious diseases, pneumonia, dementia, and all ill-defined causes) increased since the 1980s in all countries under study. This could potentially point to increased frailty among the elderly, as a result of decreased mortality selection. In further analyses, we tested whether mortality selection was indeed a dominant factor in determining trends in old-age mortality, by empirically studying the possible existence of a negative correlation between trends in late middle-age mortality and trends in old-age mortality for the same cohorts, i.e. those born around 1895, 1900, 1905, and 1910. However, in all countries and especially among men, all-cause mortality changes at ages 80-89 were strongly positively correlated with all-cause mortality changes at ages 55-69 for the same cohorts. All-cause mortality changes at ages 80-89 also correlated strongly positive with circulatory disease mortality, a cause of death susceptible to mortality selection, at ages 55-69. Moreover, mortality changes at ages 80-89 from diseases specifically related to old age, also susceptible to mortality selection, showed no clear negative correlations with all-cause mortality trends at ages 55-69. Mortality selection thus does not seem to be a driving factor behind old-age mortality trends in the countries under study.

Socio-economic developments

A possible role of socio-economic developments throughout the life-course was assessed by studying the association between old-age mortality levels for subsequent cohorts and levels of Gross Domestic Product (GDP) prevailing at different ages (i.e. 0-5, 6-19, 20-49, and 50-64) of the same cohorts. For the birth cohorts 1865 to 1924, levels of GDP at time of death were strongly inversely associated with all-cause mortality at ages 65-99, especially among women, and among men in England and Wales, Finland, and France. In most countries, stronger associations were observed for GDP levels prevailing at earlier ages of the cohorts, which remained after control for GDP at time of death. GDP prevailing at ages 20-49 (men) and 50-64 (women) showed the strongest associations with old-age mortality, whereas no strong associations with old-age mortality were observed for GDP prevailing during infancy or childhood and for infant mortality. Thus, next to the inverse effects of current economic

developments on old-age mortality trends, independent inverse effects of economic developments during earlier ages of the cohort were found. Socio-economic circumstances during adulthood and middle age seem more important in determining old-age mortality trends than those during infancy or childhood.

Medical end-of-life decision-making
The increasing prevalence of medical end-of-life decisions over time in the Netherlands [1], might have contributed to the relatively unfavourable trends among Dutch elderly. Moreover, medical end-of-life decisions could possibly contribute to cross-national differences in old-age mortality. We showed, however, that the effects of the increasing prevalence of medical end-of-life decisions in the early 1990s on the mortality trends among Dutch elderly seemed negligible based on physicians' estimation of its life-shortening effect. More in general, a rather small effect of medical end-of-life decisions on national life expectancy was observed. Although no statistically significant associations were found, the prevalence of end-of-life decisions tended to correlate positively with mortality. Correlation coefficients were higher for mortality among the elderly and for the prevalence of doctor-assisted dying. The share of physicians who feel they should unconditionally preserve their patients' lives tended to correlate negatively with mortality, especially with old-age mortality. Further research is recommended into the possibility that national old-age mortality rates reflect attitudes concerning appropriate medical treatment for the elderly.

Other determinants
Our different cohort analyses seem to suggest effects of early life circumstances carried throughout life, and, even more importantly, effects of prolonged exposure to, or long-lasting effects of risk factors, such as smoking, emerging in adult life.

In addition, mortality from cardiovascular diseases – the most important cause of death among the elderly - showed important cross-national variations in the pace of decline. In the last two decades declines for cardiovascular disease mortality among the elderly were strong in England and Wales and France and weak in Norway, the Netherlands, and Denmark. Previous research on trends in cardiovascular mortality [2-4] suggested the importance of several factors such as physical activity, hypertension control, diet, smoking, and accessibility of medical care. Possibly, the developments in some of these risk factors were more beneficial in England and Wales and France compared to the other countries.

Overall

Our results indicate large heterogeneity in the pace of mortality decline among the elderly in seven European countries, with stagnation since the 1980s in Denmark, the Netherlands and among Norwegian men, whereas mortality decline strongly continued in England and Wales and France. In determining these trends both period patterns and cohort patterns seem important. Smoking, an important cohort factor, has had a marked influence on old-age mortality trends. However, it cannot fully explain the observed stagnation in mortality decline since the 1980s. Mortality selection, another cohort factor, has not been a driving factor behind old-age mortality trends in the countries under study. Next to a period effect of current economic developments on old-age mortality trends, a cohort effect of economic developments during earlier ages of the cohort, predominantly during adulthood and

middle age, was found. Cross-national differences in old-age mortality might to some extent be affected by differences in attitudes concerning appropriate medical treatment for the elderly, although further research is recommended.

In conclusion, old-age mortality seems susceptible to both favourable and unfavourable circumstances either prevailing at old ages, or originating earlier in life, predominantly in adulthood or middle age.

REFLECTIONS ON THE METHODOLOGICAL APPROACH

Our approach of studying determinants of trends in old-age mortality trends was characterised by its emphasis on cause of death patterns, its life-course perspective, and comparative studies among seven countries. The usefulness and limitations of this approach is discussed below.

Distinction of causes of death

Studying the trends in cause-specific mortality generated clues on the determinants of all-cause mortality trends. Examining specific causes of death helped to identify intermediate factors or risk factors that could determine old-age mortality trends. For example, we made ample use of the widely known link between smoking and lung cancer in our analyses (Chapters 3, 4, and 5). Moreover, the cross-national differences in cardiovascular mortality pointed to a possible role in determining old-age mortality trends of country-specific developments in cardiovascular risk factors (Chapter 4). In addition, our inclusion of causes of death susceptible to mortality selection (i.e. circulatory diseases and diseases specifically related to old age) proved useful for our study of mortality selection as a possible determinant of old-age mortality trends (Chapter 6). Our differentiation into causes of death also informed us about causal mechanisms, for example in our study of the association between GDP throughout the life-course and old-age mortality (Chapter 7). In studying the relationship between end-of-life decision-making and old-age mortality, the inclusion of causes of death not only facilitated a further test of our hypotheses, but also gave insight into the possibility and direction of confounding (Chapter 8).

It should however be acknowledged that especially among elderly populations, the identification of a single underlying cause of death is complicated by the presence of more than one chronic disease contributing to death [5,6]. Particularly at older ages, the validity of the underlying cause of death may thus be questioned. Studying multiple causes of death has been proposed as an alternative [5-7]. However, in addition to the difficulty of obtaining these data, these studies are also limited because of the differences in the extent to which physicians list more than one cause on the death certificate [5]. Moreover, numerous methodological problems are involved due to the abundance of the data [7]. Studying multiple causes of death thus is not by definition the better solution.

In studying long-term trends in causes of death, the comparability over time may be affected by cause-specific coding changes such as the numerous revisions of the International Classification of Diseases (ICD). We made considerable effort to bridge these ICD revisions by carefully constructing a concordance table based on three-digit codes and by identifying and controlling for the remaining biases due to coding changes both within

and between ICD revisions. However, remaining problems, such as changes in the reporting of cause of death by physicians [8,9], may result in gradual shifts that are more difficult to detect and to control for [9]. These problems could have especially influenced the trends for diabetes mellitus, pneumonia, dementia, and "symptoms and ill-defined conditions". The trends for diseases that have a more straightforward diagnosis, such as smoking-related cancers, and for broad groups of causes of death such as cardiovascular diseases, are less likely to be affected.

Although not all problems mentioned above can be solved, adequately controlling for sudden cause-specific coding changes, such as the ICD related coding changes, is a prerequisite for the study of long-term cause-specific mortality trends. Moreover, when interpreting cause-specific mortality trends, potential remaining problems, such as changes in the reporting of causes of death by physicians and the quality of cause of death data, should always be kept in mind. In this way, distinction of the underlying causes of death can be a very useful tool in understanding all-cause mortality trends and in generating and testing hypotheses on its determinants.

Life-course perspective

By adopting a life-course perspective, we could distinguish between determinants occurring in late life (period determinants) and determinants originating in earlier phases of the life-course (cohort determinants) [10,11]. This distinction provided us with a clear analytical framework. Moreover, the life-course perspective proved necessary, because cohort effects were indeed found to be important in determining old-age mortality trends (Chapter 5). Without adopting a cohort approach we would not have been able to appropriately understand the role of smoking in determining old-age mortality trends, given the long lag time between smoking and mortality from lung cancer [12] (Chapters 3 and 5). Moreover, the cohort approach enabled us to study the effect of mortality selection (Chapter 6). The observed strong positive association between mortality trends at late middle age and old-age mortality trends among the same cohorts, again points to the importance of determinants that operate throughout the life-course of these cohorts. The life-course perspective also provided interesting insights when applied to the study of socio-economic determinants at different ages of subsequent cohorts on old-age mortality. Next to a period effect of current economic developments, a cohort effect of economic developments during earlier ages of the cohort, predominantly during adulthood and middle age, was found (Chapter 7).

Age-period-cohort analyses are a useful method when the life-course perspective is applied to aggregate mortality data. However, a persistent problem in age-period-cohort analyses is the identification of the separate roles of age, period and cohort, due to the interdependency between these variables (period – age = cohort). This is referred to as the identification problem [13,14]. We have adopted different strategies to cope with this problem. In Chapter 3, the identification of non-linear trends through the use of splines helped us to partly overcome the identification problem, especially for causes of death showing non-linear trends. In Chapter 5, we used a more standard procedure in which mortality is decomposed into a common linear trend (drift), a non-linear cohort effect and a non-linear period effect [13]. Focus on non-linear patterns, however, will yield a conservative estimate of the contribution of the cohort and period effects, respectively.

Moreover, the identification problem could only be dealt with adequately if clear non-linear patterns existed.

Not only can age-period-cohort analyses help in estimating the importance of period versus cohort effects in mortality trends, they can also contribute to the assessment of the role of specific determinants throughout the life-course. In Chapter 6, for example, we assessed the effects of GDP throughout the life-course on old-age mortality by substituting the period and cohort variables by GDP measures at time of death and GDP measures at earlier ages of the cohort, respectively.

Thus, despite the difficulties associated with the identification of the separate roles of age, period, and cohort effects in mortality trends, applying both a period approach and a cohort approach to the study of old-age mortality trends is both necessary and useful. Furthermore, knowledge on the relative contribution of period versus cohort determinants in mortality trends is important for making projections of future developments in mortality.

Comparative studies among seven European countries

Our comparative studies among seven selected European countries proved very useful as it informed us about generalities of patterns, and possible country-specific deviations from these general patterns. Furthermore, this approach enabled us to examine both determinants unique to specific countries, and general determinants of old-age mortality trends.

For example, the comparison of old-age mortality trends between countries revealed that the stagnation of old-age mortality decline in the Netherlands was not observed in all countries. This finding has important implications for whether a possible limit to life expectancy is already within reach (see section "What can we expect for the future?").

With regard to the determinants, the comparative analyses provided insight into the consistency of the observed national results, which facilitated a better judgment of the role of determinants in old-age mortality trends. For example, with respect to the role of socio-economic developments, an association between macro-economic conditions in early life and mortality at older ages was observed in a recent study for the Netherlands [15]. In Chapter 7, we also observed an association between GDP prevailing at ages 0-5 and mortality among Dutch elderly. This association, however, was not observed in the other countries. In general, a more important role of socio-economic circumstances in adulthood and middle age, than those in infancy and childhood, on old-age mortality was found.

An important limitation of our ecological study-design is that conclusions could only be formulated at the aggregate level, and cannot be simply generated to individuals. Furthermore, the use of aggregate data complicated the demonstration of causal mechanisms in our analyses, especially when both the trends in possible determinants and the trends in the outcome variable mortality were highly linear.

Thus, although demonstrating causation proved sometimes difficult and our conclusions cannot be generated to individuals, our parallel analyses of seven European countries did provide us with some interesting generalities and interesting deviations and helped us in

generating interesting hypotheses on the role of determinants of old-age mortality trends.

DETERMINANTS OF THE STAGNATION IN OLD-AGE MORTALITY DECLINE IN THE NETHERLANDS

The levelling off of the increase in life expectancy in the 1980s among the elderly in the Netherlands has been observed in previous studies [16-18]. This phenomenon gave rise to the current study of the determinants of trends in old-age mortality. In this section, we elaborate on the insights our research has yielded into the determinants of the stagnation in old-age mortality decline in the Netherlands, and into its determinants.

Chapter 3 showed that, around 1980, mortality decline among Dutch men and women aged 80 and over suddenly reversed into stagnation thereafter. Mortality increases were observed among men in the 1980s, and among men and women aged 90 and over in the 1980s and 1990s.

In explaining the observed stagnation, we consider both period determinants and cohort determinants, because both proved important in determining old-age mortality trends in the Netherlands. Age-period-cohort analyses showed that for Dutch men, the cohort patterns were more important than the period patterns, whereas for women linear patterns (drift) dominates, with in addition slightly larger effects of non-linear period patterns than non-linear cohort patterns (Chapters 3 and 5).

Cohort determinants

It has been stated before that although smoking has had a marked influence on the trends in old-age mortality, it cannot fully explain the observed stagnation in mortality decline since the 1980s. When, for the Netherlands, the most important smoking-related causes of death were excluded from period trends in all-cause old-age mortality, mortality decline levelled off in the 1980s, and stagnated completely in the 1990s, uniformly for men and women (Chapter 3). More generally, when trends in non-smoking related mortality – based on a simpler application of the indirect Peto-Lopez method [19,20] - are compared between the different countries (Table 1), the differences in the pace of the mortality decline between the countries disappear only partially. Recent trends in non-smoking related mortality in the Netherlands and among Danish men remain unfavourable, particularly in the 1990s, whereas recent trends in non-smoking related mortality continue to be favourable in France. The disappearance of the stagnation among Norwegian men and even more so among Danish women implies that the stagnation in Danish women and among Norwegian men is more closely related to smoking.

Closer inspection of past trends in Gross Domestic Product (GDP) – an indicator of socio-economic circumstances – showed stagnating trends in the Netherlands from 1890 to 1915 (Figure 1). Trends in GDP in the other countries at that time were much more favourable. In Chapter 5, stagnation in all-cause mortality decline in the Netherlands was observed for the cohorts born in this period, although only for men. This suggests a long-lasting effect of GDP at infancy or childhood on mortality among Dutch elderly men. Our analysis on the role of GDP throughout the life-course on old-age mortality levels for subsequent cohorts (Chapter 7) showed, indeed, an association between GDP prevailing at ages 0-5 and old-age mortality for male Dutch cohorts born between 1865 and 1924.

Table 1. Annual changes (%) for all-cause mortality and non-smoking related mortality, by country and sex, for those aged 80 and over

	All-cause mortality					Non-smoking related mortality[a]				
	1950-59	1960-69	1970-79	1980-89	1990-99	1950-59	1960-69	1970-79	1980-89	1990-99
Men										
Denmark	0.58	-1.16	-0.45	-0.19	-0.20	-0.49	-1.54	-1.42	-0.45	-0.23
England&Wales	-0.65	-0.79	-0.44	-1.44	-1.20	-0.56	-1.96	-1.27	-1.61	-0.51
Finland	-0.53	0.26	-2.45	-0.55	-1.11	-1.72	-0.41	-2.99	-0.40	-0.69
France	-1.27	-0.98	-0.73	-1.82	-1.66	-1.47	-1.45	-1.42	-2.18	-1.27
Netherlands	-0.60	-0.35	-0.93	0.38	-0.19	-1.03	-1.47	-2.09	-0.39	0.06
Norway	0.76	0.14	-0.82	0.02	-0.51	0.42	-0.05	-0.91	-1.09	-0.45
Sweden	-0.17	-0.85	-0.20	-0.92	-0.98	-0.64	-1.26	-1.02	-0.66	-0.86
Women										
Denmark	-0.09	-2.25	-1.67	-0.47	-0.56	-0.72	-2.37	-2.09	-0.79	-1.30
England&Wales	-0.96	-1.30	-0.88	-1.90	-0.65	-1.18	-1.51	-1.48	-2.56	-0.99
Finland	-0.18	-0.70	-3.85	-0.34	-1.47	-0.29	-0.61	-3.95	-0.46	-1.89
France	-1.42	-1.40	-1.07	-2.23	-1.72	-1.45	-1.25	-1.29	-2.49	-1.59
Netherlands	-1.11	-0.85	-2.72	-0.47	0.01	-1.18	-0.88	-2.79	-0.86	-0.49
Norway	0.43	-0.97	-1.80	-0.74	-0.71	0.25	-0.70	-1.38	-1.51	-0.94
Sweden	-0.61	-1.86	-1.51	-1.14	-1.05	-0.86	-2.00	-1.92	-0.94	-1.41

[a] Non-smoking related mortality was estimated based on a simpler application of the indirect Peto-Lopez method [19,20]. The prevalence of smoking in the different populations was determined by relating the lung cancer rates in these populations to the smoothed lung cancer rates among the never smokers of the ACS CPS-II study [19]. In addition, the fact was used that the etiologic fraction (EF) is a function of the proportion of the population that is exposed (p) and the relative risk (RR), i.e. EF = p(RR-1)/(p(RR-1)+1). The relative risk for smoking of total mortality was set at 2. To remove residual confounding and to obtain conservative estimates of the numbers of deaths attributable to smoking the etiological fractions were adjusted by 30% [21]. The adjusted etiological fractions were used to calculate non-smoking related mortality, i.e. all-cause mortality * (1-EF).

A largely comparable result was found in an earlier study on the effects of macro-economic conditions at ages 1-7 on the individual all-cause mortality rate at ages above 50 among Dutch cohorts born between 1817 and 1907 [15]. Thus, stagnating socio-economic developments in infancy and childhood might have contributed to the observed stagnation of mortality decline among Dutch elderly. Its effect is however less clear among women, who also still exhibited stagnation for non-smoking related diseases. Furthermore, the mechanisms behind the possible effect of socio-economic developments in infancy and childhood on old-age mortality trends are still not fully understood. Possible mechanism mentioned in literature include disease processes associated with respiratory tuberculosis, hepatitis B and cirrhosis or liver cancer, rheumatic heart disease, and respiratory infections or bronchitis, and in addition persistent viruses, dietary practices, and the burden of infectious diseases in childhood [22].

Our different cohort analyses moreover seem to suggest effects of prolonged exposure to, or long-lasting effects of risk factors, other than smoking, originating in adult life or in middle age. Cardiovascular risk factors, among the middle aged, and especially among men, seem a likely candidate.

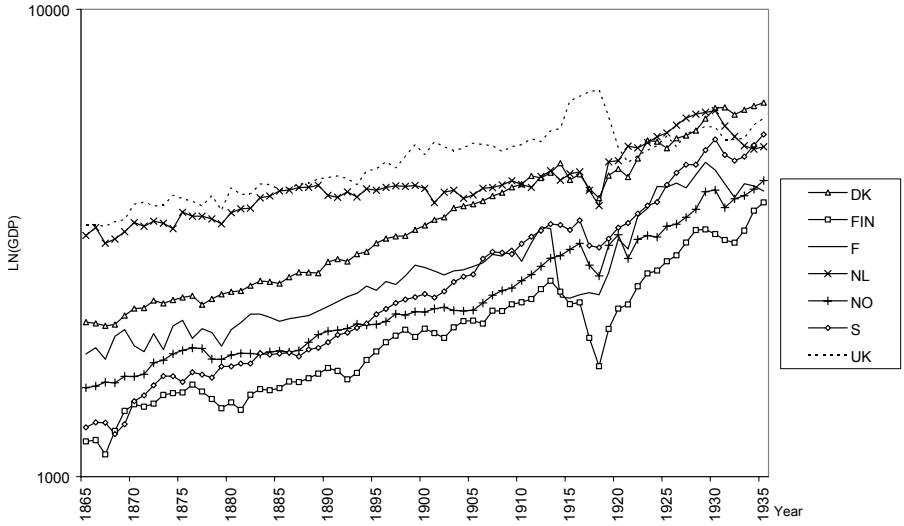

Figure 1. Trends in real Gross Domestic Product per capita at 1995 US Dollars, 1865-1935, by country

DK = Denmark; FIN = Finland; F = France; NL = the Netherlands; NO = Norway; S = Sweden; UK = United Kingdom.

In the Netherlands, age-standardized mortality from ischaemic heart disease (IHD) showed an enormous increase from 1950 to 1972, both among men and women, although less steeply among women [23]. This increase has been most dominant among those in middle age, and is largely linked to the uptake of adverse risk behaviour [23]. The mortality increases from IHD among the middle aged in the 1950s to the early 1970s, and the associated uptake of adverse risk behaviour could have contributed to the stagnation of mortality decline among Dutch elderly in the 1980s and 1990s, as they all belong to the same cohorts born roughly between 1890 and 1915. Increasing mortality rates from IHD since the 1950s were not restricted to the Netherlands, but observed in the majority of countries [24]. In Norway and the Netherlands, however, the increase was higher, at least among men [24].

Period determinants

Contemporaneous socio-economic developments probably made a large contribute to the general decline in old-age mortality. However, for the Netherlands, no different developments in GDP since the 1980s were observed in comparison to the other countries (see Chapter 7, Figure 2). This suggests that contemporaneous socio-economic developments are unlikely to contribute to the observed stagnation.

In Chapter 8, the possibility was raised that national old-age mortality rates might be affected to some extent by attitudes concerning appropriate medical treatment for the elderly. For the Netherlands, we observed a relatively high prevalence of medical end-of-life decisions, and a relatively low share of physicians who feel they should unconditionally

preserve their patients' lives were observed. This may be indicative of a different general attitude in the Netherlands concerning appropriate medical treatment for the elderly, that is, less than in other countries, directed towards unconditional life-prolonging of the elderly. Moreover, in the Netherlands, an increase in the prevalence of medical end-of-life decisions concerning elderly patients occurred in the early 1990s [1]. If we assume that behind this increasing prevalence also a change in attitude concerning appropriate medical treatment for the elderly exists, the effect of the increasing prevalence of medical end-of-life decisions may be higher than estimated in Chapter 3. This potential change in attitude could be the result of the intensification of the ongoing public debate on euthanasia in the Netherlands during the 1980s [25]. The increasing prevalence of medical end-of-life decisions in the early 1990s and possible associated changes in attitudes concerning appropriate medical treatment for the elderly thus might have contributed to some extent to the observed stagnation.

In addition, it cannot be completely ruled out that an effect on old-age mortality trends exists of changes in health care and social services available to the elderly combined with an increase in the number of elderly living alone and a possible decline in social support. In the Netherlands between World War II and 1975, care for the elderly was a central issue of the welfare state. Considerable effort and resources were spent on institutionalisation and intramural care for the elderly [26]. Since 1975, however, Dutch policies have shifted their emphasis to facilities for the elderly that enable them to stay longer at home. Although de-institutionalisation can enhance active ageing [27], it could also have made the elderly more reliant on informal or private care [26]. Combined with the increase in the number of elderly living alone [27] and the decrease in social support with age [28], de-institutionalisation may have resulted in more elderly people without sufficient care, which might have had a negative effect on their length of life. These developments are not merely restricted to the Netherlands. Very likely, cross-national differences in the timing and the impact of de-institutionalisation might have occurred. Recent data showed that of those aged 65 and over the proportion of disabled older people living in the community is fairly high in the Netherlands (12.6 %) as compared to other countries (e.g. France 8.0% and Sweden 9.4%) [27].

WHAT CAN WE EXPECT FOR THE FUTURE?

Future trends in mortality

Recently, a number of questions on possible future trends in mortality have gained interest. Particularly the extent to which today's populations are approaching a limit to human life expectancy is extensively being debated upon. On the one hand, proponents of "the limited-lifespan paradigm" state that biological and practical constraints to reducing old-age mortality imply that future gains in life expectancy will occur at a slower pace, and that the limit to life expectancy is almost reached [29-31]. On the other hand, "the mortality-reduction paradigm" is adhered to by researchers who argue that life expectancy will continue to increase for some time to come [32-34]. They support their view by referring to mortality developments in sub-populations with extreme good health, to foreseen biomedical progress, to historical observations that record life expectancy has been increasing at a constant rate, of a quarter of a year per year, for the past 160 years, and to the observation that past mortality predictions, assuming that life expectancy is

approaching a ceiling, have repeatedly been proven too pessimistic. In fact, the two groups have reached contrasting opinions by different analyses of the same empirical data.

Our comparative studies among seven European countries provide no evidence that a limit to life expectancy is within reach. Stagnation of mortality decline in the Netherlands has sometimes been seen as a sign of such a limit, but our results indicate otherwise. Although mortality decline stagnated since the 1980s in the Netherlands, Denmark, and among Norwegian men, further improvements in old-age mortality decline occurred in England and Wales and France, i.e. countries that reached the same low level of mortality as Denmark, the Netherlands, and Norway around 1980. In addition, other interpretations (e.g. effects of smoking, socio-economic developments earlier in life, trends in cardiovascular diseases) seem more likely for the observed stagnation of mortality decline.

Given the observed strong positive association between mortality trends in late middle age and those among the elderly for the same four cohorts born around 1895 to 1910 (see Chapter 6), considering recent changes in mortality at younger ages may be a useful approach to inform projections of future old-age mortality trends. Mortality trends in late middle age among Dutch men over the period 1975-1999 turned out to be quite favourable (-1.7%), and could indicate that the stagnation in mortality trends among elderly Dutch men is temporary. For women, however, recent trends in late middle age mortality were somewhat less favourable (-0.7%). This could imply that the stagnation of old-age mortality decline among Dutch women is likely to continue for some time to come.

The exact prediction of future old-age mortality patterns or future developments in life expectancy, however, remains a difficult task. Past mortality predictions have repeatedly been proven too pessimistic [34] due to the occurrence of unforeseen positive developments. For example, the huge reduction in cardiovascular disease mortality from 1970 onwards, as a result mainly of major advances in medical care and behavioural change, was not anticipated upon [24]. The possibility certainly exists that in the future medical and biomedical progress will again lead to large mortality declines. From the analyses performed in our study, it can be concluded that most gains from possible medical and biomedical progress will likely to be obtained from the further reduction of the case fatality of cardiovascular diseases and cancers, i.e. still the most important causes of death at old ages, but potentially also from reducing mortality from diseases specifically related to old age, for which recent mortality increases were observed. We would like to stress, however, that it is important to realize that not only favourable developments, but also unfavourable developments might occur. After all, the stagnation in old-age mortality decline as observed in Denmark, the Netherlands and Norway (men only) indicates that old-age mortality seems highly plastic and susceptible not only to factors with favourable effects, but also to factors with possible unfavourable effects. A current increasing threat is the obesity epidemic among young adults [35]. In addition, unfavourable developments are the re-emergence of infectious diseases [36], wars, and health effects of ecological changes [35].

Future trends in morbidity

How increasing life expectancy will affect future trends in morbidity is also still under debate. On the one hand, researchers argue that the number of years with morbidity will

eventually decrease (compression of morbidity). On the other hand, evidence is found for a lengthening period of disability associated with the increase in life expectancy (expansion of morbidity). Following Fries (1980), the original compression of morbidity hypothesis was based on two assumptions: (1) life expectancy at birth will stop increasing, because the upper limit to the human lifespan will soon be reached, and (2) the average age at which disability arises will continue to increase, because there is no near-by limit to preventing disease and disability [29]. Although, in the mean time, some alterations have been proposed to this hypothesis, it still seems valid that with the general increase in life expectancy, compression of morbidity is less likely to occur.

RECOMMENDATIONS FOR FURTHER RESEARCH

Research on the determinants of old-age mortality trends remains important for an understanding of mortality trends among the elderly, and for making inferences on possible future mortality developments. Consequently, understanding mortality trends among the elderly is highly valuable for the planning of health care services and for regulating pension systems.

Our approach focussed on cause of death patterns, adopted a life-course perspective, and comparative studies among seven countries. This approach proved very useful for generating and testing relevant hypotheses on the role of determinants of old-age mortality trends. However, some issues remain unresolved, and need further research. We discuss these issues below.

More detailed description of cause-specific mortality trends

The study of the determinants of old-age mortality trends could be aided by extending the Kannisto-Thatcher Database on old-age mortality, i.e. the most important source of all-cause mortality data specifically among the elderly, to cause-specific mortality. Larger availability of cause-specific mortality data specifically among the elderly could facilitate a more detailed description of cause-specific mortality trends among the elderly in a large number of countries. This could help in understanding all-cause mortality trends and in generating and testing hypotheses on its determinants. We, furthermore, recommend that studies on long-term cause-specific mortality trends attempt to identify and adjust for coding changes.

More in-depth comparisons of populations

To further test the hypotheses on the role of specific determinants in old-age mortality trends that were raised in our comparative population level study, more in-depth ecological research might be needed. Largely unknown remains as yet what exactly discriminates a country or a region with favourable mortality experiences from those with less favourable mortality experiences. Some insights already have been obtained on the stagnation of old-age mortality decline in the Netherlands. The success story behind the continuation of old-age mortality decline in England and Wales, and especially France, however, has not yet been revealed. Our observation that both England and Wales and France were characterised by strong recent declines in cardiovascular diseases could indicate a contribution of cross-national differences in the developments of cardiovascular risk factors, such as

physical activity, hypertension control, diet, smoking, and accessibility of medical care [2-4]. To further examine this, information on these risk factors from national surveys specifically addressed to the elderly could be used. Preferably, however, this information should be combined with country-specific information from largely comparable surveys at earlier ages of the life-course, in earlier times. In this way, for each country, cohort-specific risk factor levels could be estimated, which subsequently could be compared between countries. Note, however, that these historical comparable surveys are not widely available.

Studying regional variations within a country could also provide additional clues on the determinants of old-age mortality decline. If a pattern of geographic variation in the onset of a deceleration or acceleration in old-age mortality decline has been identified, it will be easier to find the determinants of the observed trends, because these must have followed the same patterns. For example, by studying the variation in the year of onset of ischaemic heart disease mortality decline among regions in the Netherlands, Mackenbach et al. (1989) observed an early onset in the urbanized part of the country, whereas, a later onset of decline was found in more peripheral regions [23]. This pattern could subsequently be linked to the diffusion of changes in lifestyle in the population at large, leading the authors to conclude that the trend reversal is more likely to be determined by lifestyle changes than by medical care improvements [23].

Use of data from individual-level studies

The use of aggregate data in our ecological studies complicated the demonstration of causal mechanisms with respect to the possible determinants of old-age mortality trends. Further epidemiological research specifically aiming at the elderly and using data from individual-level studies is, therefore, needed to demonstrate these causal mechanisms and to test the associated hypotheses on the determinants of old-age mortality trends. Additional research is also recommended on the exact impact of possible determinants on old-age mortality, and its changes over time. For this purpose, long-term prospective studies would be ideal. Important longitudinal studies among elderly populations are the Italian Longitudinal Study on Aging (ILSA) [37], the Longitudinal Aging Study Amsterdam (LASA) [38], and the FINE study [39]. A promising international initiative is the Survey of Health, Ageing and Retirement in Europe (SHARE) [40](www.share-project.org). However, much time is required before it is possible in the above-mentioned studies to study determinants of old-age mortality over a long time. Until that time, historical prospective cohort studies such as the Framingham Heart Study [41] and the study on male British doctors [12] might be used for this purpose.

An alternative approach, in which elements of population level studies are combined with elements of individual level studies, is the linkage of the national cause of death registries with individual characteristics. Especially in research on socio-economic mortality differences, population census linked vital registries are widely used [42]. Record-linkage research has had a long history in Scandinavian countries, and the United Kingdom. In the Netherlands, recent efforts from Statistics Netherlands have led to the construction of a Health Statistics Database ("Gezondheid Statistisch Bestand") through linkage of population registries with socio-economic databases and national health surveys [43]. However, not in all countries linkage has been introduced or, if introduced, it does not always fully capture total national populations. Through record-linkage research, differences in mortality trends

between, for example, socio-economic groups, ethnic groups, and groups by marital status, could be assessed, and linked to differences between the groups in trends in possible determinants.

Synthesis: modelling the effects of determinants simultaneously

Our separate analyses, although focusing on a number of countries, each focused on a single determinant. Very likely, however, the determinants in our study are not independent from one another. As a consequence, confounding may have occurred in our analyses. Furthermore, the contribution of the different determinants cannot be simply added.

Further research should try to incorporate the effects of different determinants on mortality trends into a more comprehensive explanatory model. For this purpose, a prerequisite and a major challenge is to obtain information on interactions between different explanatory factors. Moreover, models need to be developed that can study data on different possible determinants simultaneously, taken into account their interactions. Here, a combination of demographic methods and epidemiological knowledge might be of use.

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SUMMARY

The aim of this PhD thesis was to carefully describe trends in old-age mortality in seven European low-mortality countries from 1950 to 1999, and to assess the role of specific factors in explaining the observed trends.

For this purpose, we studied both all-cause mortality and mortality from 26 specific causes of death, we applied a life-course perspective – with emphasis on both immediate effects of circumstances prevailing at old age (= period effects) and possible long-lasting effects of determinants located earlier in life (=cohort effects) – and we performed comparative studies among seven countries, i.e. Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden. In a series of separate analyses, we assessed the role of smoking, mortality selection, socio-economic developments over the life-course, and medical end-of-life decision-making.

The **first part** of the thesis, i.e. **Chapters 2 to 5**, deals with the description of long-term trends in both all-cause and cause-specific old-age mortality, and the identification of the role of period and cohort effects in the observed trends. Within these chapters, focus was as well on smoking as a potential determinant of old-age mortality trends.

The study of long-term cause-specific mortality trends is hampered by cause-specific coding changes such as the numerous revisions of the International Classification of Diseases (ICD). In **Chapter 2**, we explained and illustrated our new method to identify and adjust for coding-related mortality discontinuities, both between and within the different ICD revisions. Starting point was the construction of a concordance table – i.e. a table linking the codes for specific causes of death through each successive ICD revision - by three-digit ICD codes. As a second step, we evaluated the occurrence of mortality discontinuities by visually inspecting cause-specific trends, by using country-specific background information on these trends, and by the quantification of the discontinuities in cause-specific (log-linear) Poisson regression models. By comparing the observed cause-specific mortality trends with the trends after adjustment for the identified coding-related mortality discontinuities, it was found that despite the use of a carefully constructed concordance table on the basis of three-digit codes, mortality discontinuities due to coding changes – both between and within ICD revisions – can lead to substantial changes of long-term mortality trends in specific causes of death. Evaluation of and controlling for these coding changes, thus, is essential when describing long-term cause-specific mortality trends. Therefore, we applied this method in all our subsequent analyses on cause-specific mortality patterns.

Chapter 3 focuses on the description of all-cause and cause-specific old-age mortality trends in the Netherlands, from 1950 to 1999. We observed that, around 1980, all-cause mortality decline among Dutch men and women aged 80 and over suddenly reversed into stagnation and even mortality increases. Stagnation was most pronounced among elderly aged 90 and over, and among men in the 1980s. Regarding the causes of death, either stagnating mortality decline, or recent or sustained increases was observed for smoking-related cancers, chronic obstructive pulmonary diseases (COPD), and a number of diseases specifically related to old age (infectious diseases, pneumonia, dementia, and "symptoms

and ill-defined conditions"). These causes of death, thus, seem to have contributed to the observed stagnation in all-cause mortality. When, for the Netherlands, the smoking-related diseases – i.e. smoking-related cancers and COPD - are excluded from period trends in all-cause mortality, mortality decline levelled off in the 1980s, and stagnated completely in the 1990s, uniformly for men and women. Smoking behaviour thus only partly seems to explain the stagnation of old-age mortality in the Netherlands.

In **Chapter 4**, we described period trends in all-cause and cause-specific old-age mortality in seven northwestern European countries, i.e. Denmark, England and Wales, Finland, France, the Netherlands, Norway, and Sweden. In these countries, mortality among those aged 80 and over generally declined from 1950 to 1999. However, there was large heterogeneity in the pace of decline. In Denmark, the Netherlands, and among Norwegian men, we observed stagnation of mortality decline or even mortality increases since the 1980s, whereas mortality decline continued strongly in England and Wales and France. Long-term mortality increases for smoking-related diseases turned into decline since the 1980s among men in all countries, except Norway. Thus, although smoking has had a marked influence on old-age mortality trends, it cannot fully explain the observed stagnation in mortality decline since the 1980s. In addition, we observed important cross-national variations in the pace of the decline in mortality from cardiovascular diseases, with largest declines in England and Wales and France. This could point to a role of cross-national differences in the development of risk factors for cardiovascular diseases, such as physical activity, diet, smoking, and accessibility of medical care. Mortality from diseases specifically related to old age increased recently in all selected countries, which may indicate increased frailty among the elderly.

Chapter 5 reports on our identification and description of cohort patterns – i.e. patterns by the year of birth of the deceased - in the mortality trends among elderly (60+) in the same seven countries. Age-period-cohort analyses revealed a statistically significant contribution of cohort effects in all countries, for both sexes, and for virtually all causes of death. Old-age mortality trends thus seem to be determined not only by period effects but also by cohort effects. The cohort patterns revealed a general decline in all-cause mortality for the subsequent cohorts born between 1865 and 1935. This decline, however, stagnated among Danish, Dutch and Norwegian men born between 1890 and 1915, and among women from all countries born after 1920. Where all-cause mortality decline stagnated, cohort patterns in mortality from lung cancer, COPD, and to a lesser extent ischaemic heart diseases, were unfavourable as well. This points again to a role of smoking in old-age mortality trends. For infectious diseases, stomach cancer, and cerebrovascular diseases, mortality increased among cohorts born before 1890, and decreased strongly thereafter. This cohort mortality pattern might be related to changing living conditions in childhood.

In the **second part** of the thesis, **Chapters 6 to 8**, further explanations for the observed old-age mortality trends in the seven countries were examined, by assessing the role of mortality selection, socio-economic developments throughout the life-course, and medical end-of-life decision-making.

In **Chapter 6**, we assessed the role of mortality selection. Mortality selection implies that high mortality at younger ages tends to affect the frail individuals first, leaving a more selected and more robust elderly population. Consequently with decreasing mortality at

younger ages, the increasing proportion of the elderly population might be less healthy when compared with their more selected predecessors, and subsequently could experience comparatively higher mortality. We tested whether mortality selection was a dominant factor in determining trends in old-age mortality, by empirically studying the possible existence of a negative correlation between trends in late middle-age mortality and trends in old-age mortality for the same cohorts, i.e. those born around 1895, 1900, 1905, and 1910. In all countries and especially among men, all-cause mortality changes at ages 80-89 were strongly positively correlated with all-cause mortality changes at ages 55-69 for the same cohorts. All-cause mortality changes at ages 80-89 also correlated strongly positive with circulatory disease mortality, a cause of death susceptible to mortality selection, at ages 55-69. Mortality changes at ages 80-89 from diseases specifically related to old age, also susceptible to mortality selection, showed no clear negative correlations with all-cause mortality trends at ages 55-69. These consistently positive correlations indicate that trends in old-age mortality in northwestern Europe in the late 20th century were determined predominantly by the prolonged effects of exposures carried throughout life, and not by mortality selection.

Chapter 7 reports on our exploration of the role of socio-economic developments throughout the life-course on old-age mortality trends. We assessed whether old-age mortality levels for subsequent cohorts are associated with levels of Gross Domestic Product (GDP) prevailing at different ages (i.e. 0-5, 6-19, 20-49, and 50-64) of the same cohorts. For the birth cohorts 1865 to 1924, levels of GDP at time of death were strongly inversely associated with all-cause mortality at ages 65-99, especially among women, and among men in England and Wales, Finland, and France. In most countries, stronger associations were observed for GDP levels prevailing at earlier ages of the cohorts, which remained after control for GDP at time of death. Thus, next to the inverse effect of current economic developments on old-age mortality trends an independent, mostly inverse effect of economic developments during earlier ages of the cohort was found. GDP measured at ages 20-49, for men, and 50-64, for women, showed the largest associations with old-age mortality. This indicates that socio-economic circumstances during adulthood and middle age seem more important in determining old-age mortality trends than those during infancy or childhood.

In **Chapter 8**, we examined the association between physicians' practices and attitudes concerning end-of-life decision-making and mortality by age and cause of death across six European countries, i.e. Belgium, Denmark, Italy, the Netherlands, Sweden, and Switzerland. Note that medical end-of-life decisions mainly involve the withholding and withdrawing of life-prolonging treatments or the administration of potentially life-shortening drugs for the alleviation of severe suffering. In general, we observed a rather small effect of medical end-of-life decisions on national life expectancy. Although no statistically significant associations were found, the prevalence of end-of-life decisions tended to correlate positively with mortality. Correlation coefficients were higher for mortality among the elderly (80+) and for the prevalence of doctor-assisted dying. The share of physicians who feel they should unconditionally preserve their patients' lives tended to correlate negatively with mortality, especially with old-age mortality. Further research is recommended into the possibility that national old-age mortality rates reflect attitudes concerning appropriate medical treatment for the elderly.

In **Chapter 9**, we summarized the main results of this thesis, and evaluated the usefulness and possible limitations of our methodological approach in obtaining these results. In addition, we elaborated on the determinants of the stagnation in mortality decline among elderly in the Netherlands, and on possible future trends in old-age mortality. Lastly, recommendations for further research were proposed.

Our results indicate large heterogeneity in the pace of mortality decline among the elderly in seven European countries, with stagnation since the 1980s in Denmark, the Netherlands and among Norwegian men, whereas mortality decline strongly continued in England and Wales and France. In determining these trends both period patterns and cohort patterns seem important. Smoking, an important cohort factor, has had a marked influence on old-age mortality trends. However, it cannot fully explain the observed stagnation in mortality decline since the 1980s Mortality selection, another cohort factor, has not been a driving factor behind old-age mortality trends in the countries under study. Next to a period effect of current economic developments on old-age mortality trends, a cohort effect of economic developments during earlier ages of the cohort, predominantly during adulthood and middle age, was found. Cross-national differences in old-age mortality might to some extent be affected by differences in attitudes concerning appropriate medical treatment for the elderly, although further research is recommended.

In conclusion, old-age mortality seems susceptible to both favourable and unfavourable circumstances either prevailing at old ages, or originating earlier in life, predominantly in adulthood or middle age.

Our more in-depth study of the possible explanations for the stagnation of mortality decline among Dutch elderly revealed again that smoking could not fully explain the observed stagnation. With regard to other cohort factors, stagnating socio-economic developments in infancy and childhood might also have contributed in the Netherlands to the observed stagnation. Moreover, the mortality increases from ischaemic heart diseases among the middle aged in the 1950s to the early 1970s, and the associated uptake of adverse risk behaviour, could have contributed to the stagnation of mortality decline among Dutch elderly in the 1980s and 1990s. Concerning possible period determinants, the increase in the prevalence of medical end-of-life decisions in the early 1990s in the Netherlands and possible associated changes in attitudes concerning appropriate medical treatment for the elderly might have contributed to some extent to the observed stagnation. In addition, an effect of changes in health care and social services available to the elderly together with an increase in the number of elderly living alone and a possible decline in social support cannot be completely ruled out.

Concerning possible future old-age mortality patterns, an important current debate is the extent to which today's populations are approaching a limit to human life expectancy. Our comparative studies among seven European countries provide no evidence that a limit to life expectancy is within reach. Stagnation of mortality decline in the Netherlands has sometimes been seen as a sign of such a limit, but our results indicate otherwise. Although mortality decline stagnated since the 1980s in the Netherlands, Denmark, and among Norwegian men, further improvements in old-age mortality decline occurred in England and Wales and France, i.e. countries that reached the same low level of mortality as Denmark, the Netherlands, and Norway around 1980. In addition, other interpretations (e.g. effects

of smoking, socio-economic developments earlier in life, trends in cardiovascular diseases) seem more likely for the observed stagnation of mortality decline.

The prediction of future old-age mortality patterns or future developments in life expectancy remains a difficult task. Although past mortality predictions have repeatedly been proven too pessimistic due to unforeseen positive developments, we stress the possibility that unfavourable developments, such as the obesity epidemic, and the re-emergence of infectious diseases, might also influence the future course of old-age mortality and life expectancy.

Studying old-age mortality trends and its possible determinants remains important to inform politicians and health care professionals on possible mortality developments in the future. Our approach that focused on cause of death patterns, adopted a life-course perspective, and used comparative studies among different countries, proved very useful for generating and testing relevant hypotheses on explanations of old-age mortality trends. The further study of the determinants of old-age mortality trends could be aided by a more detailed description of cause-specific mortality trends, in a larger number of countries. To study more specifically the factors that discriminate a country or a region with favourable mortality experiences from those with less favourable mortality, more in-depth comparative research is recommended in which cohort-specific risk factor levels are reconstructed from existing surveys both among the elderly and among the younger population in earlier times. Studying regional variations within a country could also provide additional clues on the determinants of old-age mortality trends. To demonstrate the exact impact and the causal mechanisms of possible determinants on old-age mortality, and its changes over time, long-term prospective studies - using individual-level data – would be ideal. An alternative approach is the linkage of the national cause of death registries with individual characteristics. As a final step, further research should try to incorporate the effects of different determinants on mortality trends into a more comprehensive explanatory model.

SAMENVATTING

Dit proefschrift had tot doel enerzijds de ontwikkelingen in de sterfte op oude leeftijd in zeven Europese landen van 1950 tot 1999 te beschrijven en anderzijds de rol van specifieke mogelijk verklarende factoren in de waargenomen trends te achterhalen.

Hiertoe hebben we zowel totale sterfte als sterfte aan 26 specifieke doodsoorzaken bestudeerd, hebben we een levensloopbenadering gehanteerd – met aandacht voor zowel factoren op hoge leeftijd (= periode factoren) als ook factoren eerder in het leven (= cohort factoren) – en hebben we vergelijkende studies uitgevoerd in zeven landen te weten Denemarken, Engeland en Wales, Finland, Frankrijk, Nederland, Noorwegen en Zweden. In een aantal afzonderlijke analyses hebben we achtereenvolgens de rol van roken, sterfteselectie, sociaal-economische ontwikkelingen gedurende de levensloop en medische beslissingen rondom het levenseinde onderzocht.

Het **eerste deel** van dit proefschrift, **Hoofdstuk 2 t/m 5**, beschrijft de lange-termijn ontwikkelingen in zowel totale als doodsoorzaakspecieke sterfte onder ouderen en omvat onze analyse naar de rol van periode dan wel cohort effecten in de waargenomen trends. In deze hoofdstukken werd daarnaast de rol van roken als mogelijke determinant van de sterftetrends op hoge leeftijd onderzocht.

Het bestuderen van lange-termijn doodsoorzaakspecifieke sterftetrends wordt bemoeilijkt door doodsoorzaakspecifieke coderingsveranderingen zoals de talrijke revisies van de International Classification of Diseases (ICD) van de Wereldgezondheidsorganisatie (WHO). In **Hoofdstuk 2** hebben we onze nieuwe methode om coderingsgerelateerde sterfte-onregelmatigheden te achterhalen en te controleren, uiteengezet en geïllustreerd. Hierbij hebben we zowel coderingsveranderingen tussen ICD revisies als overige coderingsveranderingen binnen ICD revisies geëvalueerd. Uitgangspunt van onze methode was de constructie van een concordantietabel (een tabel die de doodsoorzaakspecifieke ICD codes voor de verschillende ICD revisies aan elkaar koppelt) op basis van 3-cijferige ICD codes. Vervolgens hebben we onregelmatigheden in de sterftetrends opgespoord aan de hand van (a) visuele inspectie van de doodsoorzaakspecifieke sterftetrends, (b) additionele landen-specifieke informatie betreffende de sterftetrends en (c) kwantificering van de onregelmatigheden in doodsoorzaakspecifieke log-lineaire regressiemodellen (Poisson). Vergelijking van de waargenomen doodsoorzaakspecifieke sterftetrends met de trends na controle voor de geïdentificeerde coderingsgerelateerde sterfteonregelmatigheden toonde aan dat, ondanks het gebruik van een concordantietabel op basis van 3-cijferige ICD codes, sterfte-onregelmatigheden als gevolg van coderingsveranderingen, zowel tussen als binnen ICD revisies, kunnen leiden tot aanzienlijke vertekeningen van sterftetrends voor specifieke doodsoorzaken. Het opsporen van en het controleren voor deze coderingsveranderingen is dan ook essentieel voor een adequate beschrijving van lange-termijn doodsoorzaakspecifieke sterftetrends. In onze resterende analyses met betrekking tot doodsoorzaakspecifieke sterftetrends hebben we voor de geïdentificeerde coderingsgerelateerde sterfteonregelmatigheden gecontroleerd.

Hoofdstuk 3 omvat de beschrijving van totale en doodsoorzaakspecifieke sterftetrends van 1950 tot 1999 onder ouderen in Nederland. We vonden dat de afname in totale sterfte

onder Nederlandse mannen en vrouwen van 80 jaar en ouder rond 1980 plotseling omsloeg in stagnatie en zelfs sterftetoename. Stagnatie kwam vooral voor onder 90-plussers en onder mannen in de jaren tachtig. Wat de doodsoorzaken betreft, werd stagnerende sterfteafname dan wel recente of voortdurende sterftetoename waargenomen voor de rookgerelateerde kankers, chronisch obstructieve longziekten, en voor een aantal specifieke ouderdomsziekten ('infectieuze en parasitaire ziekten', 'pneumonie en influenza', 'dementie en Alzheimer' en 'symptomen en onvolledig omschreven ziektebeelden'). Deze doodsoorzaken lijken dus een bijdrage geleverd te hebben aan de stagnatie voor totale sterfte. Bekijken we de totale sterftetrends zonder de rookgerelateerde doodsoorzaken (rookgerelateerde kankers en chronisch obstructieve longziekten) dan is voor zowel oudere mannen als oudere vrouwen nog steeds een stagnerende sterfteafname in de jaren tachtig en een complete stagnatie in de jaren negentig zichtbaar. Rookgedrag lijkt dan ook slechts deels de stagnatie in de sterftedaling onder Nederlandse ouderen te kunnen verklaren.

In **Hoofdstuk 4** hebben we de periode ontwikkelingen in totale en doodsoorzaakspecifieke sterfte beschreven onder ouderen in zeven Noordwest Europese landen, te weten Denemarken, Engeland en Wales, Finland, Frankrijk, Nederland, Noorwegen en Zweden. Voor deze landen nam de totale sterfte onder 80-plussers over de periode 1950 tot 1999 over het algemeen af. Er bleek echter een grote verscheidenheid in de mate van afname te bestaan. Onder Denen, Nederlanders en Noorse mannen werd sinds 1980 stagnatie van de sterfteafname en zelfs sterftetoename waargenomen, terwijl in Engeland en Wales en Frankrijk de sterke sterfteafname voortduurde. Wat betreft de doodsoorzaken vonden we dat de langdurige sterftetoename in rookgerelateerde kankers en chronisch obstructieve longziekten onder mannen veranderde vanaf de jaren tachtig in sterfteafname in alle landen behalve Noorwegen. Dus ook al heeft roken de totale sterftetrends onder ouderen duidelijk beïnvloed, de rol van roken lijkt minder van belang voor de verklaring van de stagnatie vanaf de jaren tachtig. Daarnaast vonden we belangrijke verschillen tussen de landen in het afname tempo van sterfte aan hart- en vaatziekten, met de grootste afname in Engeland en Wales en Frankrijk. Dit zou kunnen wijzen op verschillen tussen landen in ontwikkelingen in risicofactoren van hart- en vaatziekten, zoals lichaamsbeweging, bloeddrukcontrole, eetpatroon, roken en toegankelijkheid van medische zorg. Sterfte aan ouderdomsgerelateerde ziekten, als 'infectieuze en parasitaire ziekten', 'pneumonie en influenza', 'dementie en Alzheimer' en 'symptomen en onvolledig omschreven ziekte-beelden', nam recentelijk toe in alle landen. Mogelijk dat hier toegenomen frailty oftewel een zwakkere constitutie van ouderen aan ten grondslag ligt.

Hoofdstuk 5 bericht over onze identificering en beschrijving van cohort patronen (patronen naar geboortejaar van de overledenen) in sterftetrends onder ouderen (60+) in de zeven onderzochte landen. Onze leeftijd-periode-cohort analyse openbaarde een statistisch significante bijdrage van cohort effecten in de sterftetrends in alle landen, voor beide seksen en voor vrijwel alle doodsoorzaken. Sterftetrends onder ouderen blijken dus naast periode effecten ook bepaald te worden door cohort effecten. De cohort patronen lieten een algemene afname in totale sterfte voor de opeenvolgende geboortecohorten van 1865 tot 1935 zien. Deze afname stagneerde echter onder Deense, Nederlandse en Noorse mannen geboren tussen 1890 en 1915 en onder vrouwen geboren na 1920 in alle landen. Voor die cohorten waar de afname in totale sterfte stagneerde, waren de cohort patronen voor longkankersterfte, chronisch obstructieve longziekten en in mindere mate ischemische hartziekten ook minder gunstig. Dit wijst opnieuw op een rol van roken in de bepaling van

sterftetrends onder ouderen. Voor 'infectieuze en parasitaire ziekten', maagkanker en cerebrovasculaire ziekten nam de sterfte over het algemeen toe voor de cohorten geboren vóór 1890 en af voor de latere cohorten. Dit cohort patroon zou te maken kunnen hebben met veranderde levensomstandigheden in de kindertijd.

In het tweede deel van dit proefschrift, **Hoofdstuk 6 t/m 8**, zijn we op zoek gegaan naar nadere verklaringen van de waargenomen sterftetrends onder ouderen in de zeven landen onder studie. Hiertoe hebben we de rol van sterfteselectie, sociaal-economische ontwikkelingen gedurende de levensloop en medische beslissingen rondom het levenseinde onderzocht.

Hoofdstuk 6 betrof onze studie naar de rol van sterfteselectie. Sterfteselectie houdt in dat hoge sterfte op jongere leeftijd mogelijk een meer geselecteerde oudere bevolking met zich meebrengt. Bij afnemende sterfte op jongere leeftijd zou het toenemende aandeel ouderen mogelijk minder geselecteerd kunnen zijn dan eerdere cohorten ouderen, met als gevolg relatief meer morbiditeit en sterfte. We hebben geanalyseerd of sterfteselectie een prominente rol heeft gespeeld in de bepaling van sterftetrends onder ouderen door voor vier opeenvolgende groepen geboortecohorten (geboren rond 1895, 1900, 1905 en 1910) na te gaan of er een negatieve correlatie bestaat tussen sterftetrends op middelbare leeftijd en sterftetrends op oudere leeftijd van de cohorten. Voor alle zeven landen en vooral voor mannen blijken totale sterftetrends onder 80-89 jarigen sterk positief te correleren met totale sterftetrends eerder in het leven onder 55-69 jarigen. Totale sterftetrends onder 80-89 jarigen correleerden ook sterk positief met trends in sterfte aan hart- en vaatziekten, een sterfteselectie gevoelige doodsoorzaak, onder 55-69 jarigen. De correlatie van sterftetrends in specifieke ouderdomsziekten, ook sterfteselectie gevoelig, onder 80-89 jarigen met totale sterftetrends onder 55-69 jarigen was niet duidelijk negatief. De consistent waargenomen positieve correlaties duiden erop dat sterftetrends onder ouderen in Noordwest Europa aan het eind van de twintigste eeuw vooral beïnvloed worden door verstrekkende gevolgen van blootstellingen eerder in het leven en niet zozeer door sterfteselectie.

De rol van sociaal-economische ontwikkelingen gedurende de levensloop op sterftetrends onder ouderen werd nader onderzocht in **Hoofdstuk 7**. Hiertoe hebben we vastgesteld of sterfteniveaus onder ouderen voor opeenvolgende cohorten gerelateerd zijn aan niveaus van Bruto Binnenlands Product (BBP) per capita op verschillende leeftijden (0-5, 6-19, 20-49 en 50-64) van diezelfde cohorten. Voor de geboortecohorten van 1865 tot 1924 was BBP ten tijde van het overlijden sterk omgekeerd gerelateerd aan totale sterfte onder 65-99 jarigen, vooral voor vrouwen, maar ook voor mannen in Engeland en Wales, Finland en Frankrijk. In de meeste landen werden sterkere associaties gevonden voor BBP op eerdere leeftijden van de cohorten, die bleven bestaan na controle voor BBP ten tijde van het overlijden. Naast een omgekeerd effect van huidige economische ontwikkelingen op sterftetrends onder ouderen bleek er dus een onafhankelijk overwegend omgekeerd effect te bestaan van economische ontwikkelingen gedurende eerdere leeftijden van de bestudeerde cohorten. Terwijl voor mannen BBP op 20-49 jarige leeftijd de grootste associatie met sterfte onder ouderen liet zien, was voor vrouwen BBP op leeftijd 50-64 belangrijk. Dit geeft aan dat sociaal-economische omstandigheden gedurende het volwassen leven en gedurende de middelbare leeftijd belangrijker lijken voor het bepalen van sterftetrends onder ouderen dan die gedurende het eerste levensjaar of de kindertijd.

In **Hoofdstuk 8** hebben we de associatie tussen de praktijk en de opvattingen van artsen ten aanzien van medische beslissingen rond het levenseinde enerzijds en sterfte naar leeftijd en doodsoorzaak anderzijds onderzocht voor zes Europese landen samen, te weten België, Denemarken, Italië, Nederland, Zweden en Zwitserland. Merk op dat medische beslissingen rond het levenseinde vooral het niet-instellen of staken van levensverlengende behandelingen en potentieel levensbekortende pijn- of symptoombestrijding betreffen. Ons onderzoek liet zien dat het effect van medische beslissingen rond het levenseinde op de nationale levensverwachting klein was. Toch was er aanwijzing voor een positieve relatie tussen het percentage medische beslissingen rond het levenseinde en sterfte, vooral in relatie tot de sterfte onder ouderen en in relatie tot levensbeëindigend handelen (hulp bij zelfdoding, euthanasie, levensbeëindigend handelen zonder uitdrukkelijk verzoek van de patiënt). Ook vonden we aanwijzingen voor de verwachte negatieve relatie tussen sterfte en het aandeel artsen dat het leven van de patiënt in alle omstandigheden wil behouden, opnieuw vooral in relatie tot sterfte onder ouderen. Echter in al deze gevallen waren de correlatiecoëfficiënten niet statistisch significant. Nader onderzoek naar de mogelijkheid dat nationale sterfte onder ouderen beïnvloed zou kunnen worden door opvattingen ten aanzien van gepast medisch handelen onder ouderen is raadzaam.

In **Hoofdstuk 9** worden de belangrijkste resultaten van dit proefschrift samengevat en de positieve en negatieve aspecten van onze methodologische benadering op een rij gezet. Daarnaast zijn we nader ingegaan op de mogelijke determinanten van de stagnatie van de sterfteafname onder ouderen in Nederland en op verwachtingen ten aanzien van toekomstige sterfteontwikkelingen. Tot slot zijn aanbevelingen voor verder onderzoek gedaan.

Onze bevindingen duiden op een enorme heterogeniteit in de mate van sterfteafname onder ouderen in zeven Europese landen, met stagnatie in Denemarken, Nederland en onder Noorse mannen sinds 1980, maar voortdurende sterfteafname in Engeland en Wales en Frankrijk. Deze trends blijken zowel door periode patronen als door cohort patronen bepaald te worden. Roken, een belangrijke cohortfactor, heeft een belangrijke rol gespeeld in de sterftetrends onder ouderen, maar kan de waargenomen stagnatie niet volledig verklaren. Sterfteselectie, een andere cohortfactor, heeft geen doorslaggevende rol gespeeld in de sterftetrends onder ouderen in de zeven onderzochte landen. Naast een periode effect van huidige sociaal-economische ontwikkelingen op sterftetrends onder ouderen werd een cohort effect van sociaal-economische ontwikkelingen gedurende jongere leeftijden van de verschillende cohorten gevonden. Vooral sociaal-economische ontwikkelingen gedurende de volwassenheid en de middelbare leeftijd bleken belangrijk. Daarnaast zouden verschillen in sterfte onder ouderen tussen landen enigszins beïnvloed kunnen worden door verschillen tussen landen in de opvattingen ten aanzien van gepast medisch handelen onder ouderen. Dit laatste vereist echter nader onderzoek.

Al met al blijkt sterfte op hoge leeftijd beïnvloed te worden door zowel gunstige als ongunstige factoren die zich voor kunnen doen op hoge leeftijd of al eerder in de levensloop kunnen ontstaan en dan vooral op volwassen of middelbare leeftijd.

Onze meer gedetailleerde studie naar de mogelijke verklaringen van de stagnatie van de sterftedaling onder Nederlandse ouderen toonde opnieuw dat roken niet de enige determinant van de waargenomen stagnatie geweest kan zijn. Wat andere cohortfactoren betreft, lijkt het erop dat stagnerende sociaal-economische ontwikkelingen gedurende de

kindertijd in Nederland toch ook bijgedragen kunnen hebben aan de waargenomen stagnatie. Tevens zou de sterftetoename voor ischemische hartziekten onder mannen en vrouwen in de middelbare leeftijd in de jaren vijftig tot begin jaren zeventig en het daaraan gerelateerde risicogedrag bijgedragen kunnen hebben aan de stagnatie van de sterfteafname onder Nederlandse ouderen in de jaren tachtig en negentig. Qua periode factoren zou de toename in de prevalentie van medische beslissingen rondom het levenseinde begin jaren negentig in Nederland en de mogelijk daaraan gerelateerde veranderingen in opvattingen ten aanzien van gepaste medische zorg voor de ouderen enigszins bijgedragen kunnen hebben aan de waargenomen stagnatie. Daarnaast kunnen we een mogelijk effect van veranderingen in de gezondheidszorg en sociale voorzieningen voor ouderen, gekoppeld aan een toename van het aantal alleenwonende ouderen en een mogelijke afname in sociale steun, niet volledig uitsluiten.

Wat betreft mogelijke toekomstige sterfteontwikkelingen onder ouderen, is het actuele wetenschappelijke debat over de mate waarin huidige samenlevingen een mogelijke limiet aan de levensverwachting van de mens naderen van belang. Onze vergelijkende studies in zeven Europese landen leveren geen bewijs voor het binnen bereik zijn van een limiet aan de levensverwachting. De stagnatie van de sterfteafname in Nederland werd wel eens als bewijs voor zo'n limiet gezien, maar onze resultaten laten het tegendeel zien. Ook al stagneerde de sterfteafname sinds de jaren tachtig in Nederland, Denemarken en onder Noorse mannen, de sterfteafname continueerde voor Engeland en Wales en Frankrijk die rond 1980 het zelfde lage sterfteniveau hadden als Denemarken, Nederland en Noorwegen. Andere verklaringen voor de waargenomen stagnatie van de sterfteafname zoals roken, sociaal-economische ontwikkelingen eerder in het leven, ontwikkelingen in risicogedrag ten aanzien van hart- en vaatziekten e.d. lijken meer waarschijnlijk.

Het voorspellen van toekomstige sterftepatronen onder ouderen of toekomstige ontwikkelingen in levensverwachting blijft lastig. Ondanks dat sterfteprojecties uit het verleden achteraf herhaaldelijk te pessimistisch zijn gebleken door onvoorziene gunstige ontwikkelingen, willen we erop wijzen dat toekomstige sterftetrends onder ouderen en ontwikkelingen in levensverwachting ook beïnvloed kunnen worden door ongunstige ontwikkelingen zoals de obesitas epidemie en het opnieuw optreden van infectieziekten.

Het bestuderen van sterftetrends onder ouderen en mogelijke determinanten blijft belangrijk om beleidsmakers en professionals in de gezondheidszorg adequaat over mogelijke toekomstige sterfteontwikkelingen te informeren. Onze benadering met zijn nadruk op sterftetrends voor verschillende doodsoorzaken, de hantering van een levensloopbenadering en het uitvoeren van vergelijkende studies in zeven landen bleek erg nuttig voor het verkrijgen en testen van relevante hypothesen met betrekking tot verklaringen van sterftetrends onder ouderen. De verdere studie naar de determinanten van sterftetrends onder ouderen zou geholpen kunnen worden door een meer gedetailleerde beschrijving van doodsoorzaakspecifieke sterftetrends in een groter aantal landen. Om meer specifiek de factoren te bestuderen die een land of regio met gunstige sterfte-ervaringen onderscheid van een land of regio met minder gunstige ontwikkelingen raden we een meer gedetailleerd vergelijkend onderzoek aan waarin cohortspecifieke niveaus van risicofactoren gereconstrueerd worden aan de hand van bestaande surveys zowel recentelijk uitgevoerd onder ouderen als eerder uitgevoerd onder de toenmalige jongere bevolking. Het bestuderen van regionale verscheidenheid binnen een land zou ook

tot nieuwe aanwijzingen kunnen leiden over de determinanten van sterftetrends onder ouderen. Voor het bestuderen van de exacte effecten en causale mechanismen van mogelijke determinanten van sterfte onder ouderen en veranderingen hierin over tijd zouden lange-termijn prospectieve studies, waarbij gegevens op individueel niveau verzameld worden, ideaal zijn. Een alternatieve benadering is het koppelen van nationale doodsoorzaken registers aan individuele informatie. Het uiteindelijke doel van nader onderzoek zou de ontwikkeling van een alomvattend verklarend model dienen te zijn waarin de effecten van verschillende determinanten op sterftetrends onder ouderen tegelijkertijd meegenomen worden.

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