Deviating Trends in Dutch Life Expectancy: Explanation and Projection

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Deviating Trends in Dutch Life Expectancy:
Explanation and Projection

Afwijkende trends van de levensverwachting in Nederland:
verklaring en projectie

Thesis

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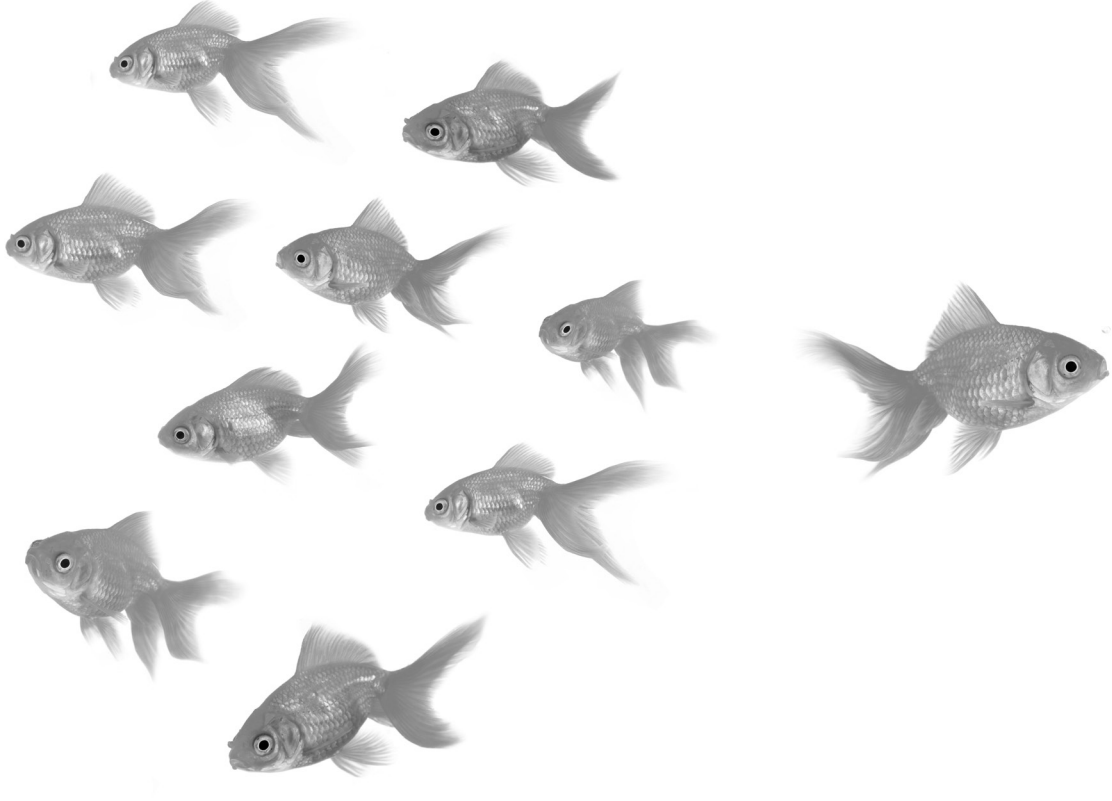

DEVIATING TRENDS IN DUTCH LIFE EXPECTANCY

During the 17th century, the Dutch Republic – a region which later formed the Netherlands – experienced a sudden economic, social and cultural upswing and emerged as the first modern economy.[1] Contemporaries from other countries were completely surprised by the rise of a small region to a dominant global trading power, and due to a lack of plausible explanations, termed this phenomenon as "Dutch miracle".[2] Throughout the history of the Netherlands, this term was used to describe exceptional and unexpected positive developments of the country in relation to its neighbours and other Western countries.[3]

A recent development, which fully deserves to be described as "Dutch miracle," occurred in the year 2002, when life expectancy started to increase rapidly after almost two decades of slower improvement and partly even stagnated progress.[4] It was first believed that the sudden decrease in mortality was only a temporary phenomenon induced by milder temperatures during the summer and winter. However, the improvement sustained at least a decade, which resulted in life-expectancy gains of about 2.5 years in women and about 4 years in men between since 2001.[5-7] This dramatic change was not only exceptional for the Netherlands, but also internationally. Compared to the world record life expectancy growing about 3 months each year, the pace of improvement in Dutch life expectancy was almost twice as high, growing by about 5 months per year between 2002 and 2007.[8,9] This new "Dutch miracle" demonstrated that even in low-mortality countries, further substantial and rapid reductions of mortality are possible, which disproves theories arguing that longevity could only be modestly and gradually influenced in modern societies.[10,11]

The Netherlands is not the only exception from the regular decline in mortality rates as exhibited in most high-income countries.[12] Among the countries in Western Europe, Denmark also experienced a longer stagnation in improvements of life expectancy during the 1980s followed by a sudden upturn at around 1995.[13] The same pattern has been noted within the former communistic countries in Central and Eastern Europe, where in the post-1989 era, the improvement in life expectancy finally resumed after decades of stagnation.[14] Other deviating countries are still in the phase of stagnation, waiting for the resumption of faster increasing life expectancy, such as the USA and Mexico.[15,16] While in the more recent time, trend reversals in life expectancy rarely occurred: during the 1960s and 1970s in almost any Western country mortality decline suddenly accelerated termed as the "cardiovascular revolution".[17]

The remarkable upturn in Dutch life expectancy since 2002 was held almost impossible a few years earlier, where after decades of only little progress, particularly among Dutch women, many believed that the already very low mortality rates could not be further lowered.[11] This belief was backed up by numerous publications arguing that life expectancy had been approaching an upper biological limit of life, which was assumed to be at about 85 years.[18-20] Before the start of the stagnation, Dutch mortality had declined drastically to the lowest levels ever observed in the world.[21] Therefore, the relatively slower pace of Dutch mortality decline at the end of the 20th century was seen as a logical consequence of the relatively faster pace of the decline before.[22] Comparable arguments were made for other former vanguard countries, such as Denmark and the USA, where life expectancy improvements also came to halt during the 1980s.[13,16] Faced with the slowing down of progress in the fight against death, many worried that the great successes in medicine to save lives of the frail and the sick resulted in an increasingly unhealthy population, which is described within the theory of "the failure of success".[23,24]

Official judgements about the potential of further progress in Dutch life expectancy during the first part of the 21st century fundamentally changed after the trend reversal occurred. In the early 2000s, the more pessimistic view that Dutch life expectancy was approaching a biological limit was reflected in official forecasts. In 2004, two years after the strong increase in Dutch life expectancy occurred, Statistics Netherlands still expected merely a small gain in life expectancy of not more than 1.5 years up until 2050.[25] In fact, instead of the predicted 46 years, it took only 5 years until this gain was realized.[9] At the same time, a growing list of countries surpassed levels of life expectancy well above the previously assumed fixed upper limit of 85 years.[20] It became increasingly apparent that the Netherlands was not a precursor of mortality trends, but rather it was lagging behind the rates of improvement realized in other countries, particularly concerning old-age mortality.[26] Most other Western countries experienced steady improvement in life expectancy since about the 1950s without any signs of slowing down.[8,12,27] Confronted with such evidence from other countries, in addition to the sustained decrease in Dutch mortality rates, official forecasts became suddenly much more optimistic.[28] In their most recent forecast, Statistics Netherlands expects a level of life expectancy of 88.5 years in 2050, which is about 6 years higher than a decade earlier, where the target for 2050 was 82.3 years.[29,30]

The unexpected strong increase in life expectancy poses an economic and financial burden for the Dutch welfare state and its institutions. Life insurance companies need to incorporate the higher uncertainty of individual lifespans as "longevity risk" into their products.[31] The unforeseen steep rise in the number of elderly directly

translated into considerably higher pension costs.[32,33] Costs for public health care and long-term care in the Netherlands grew to a proportion of 8.7 % of the GDP in 2006-2010, which was the highest value worldwide, and are expected to further double until 2060.[34] If life expectancy keeps improving throughout the next decades at a comparable pace, the financial stability of public budgets in the Netherlands is jeopardized, and more painful reforms will potentially become necessary.[35] This is particularly relevant in the aftermath of the worldwide economic crisis in 2008, where a general climate of austerity has been established.[36]

Despite the possible negative consequences, the sudden drop in the risk to die, enabling much longer life spans, is a distinctly positive development. The increase in life expectancy clearly disproved earlier concerns that all that Dutch people could hope for was too minimize the time spent with diseases in an otherwise fixed lifespan.[37] Undoubtedly, avoiding death is the most fundamental need of any human, as it is in any form of life, and finding ways to overcome death entirely is assumedly one of the oldest dreams of mankind.[38] Moreover, besides education and economic prosperity, life expectancy is a central indicator of human development, thus an important proxy for the general welfare of a country.[39] The longer lifespans (if spent in good health), enable individuals to balance the time of their life course devoted to work and leisure more evenly.[40] Investments in education result in higher returns because individuals face lower risks to die prematurely, may work longer, and could accumulate more wealth over their lifetime.[41] Since older people consume fewer energy-intensive goods, such as cars, flights or furniture, than younger people, older populations produce less carbon dioxide emissions on average.[42] Thus, negative consequences of population ageing, due to longer life expectancy, are offset by the benefits, resulting in a happier, richer, wiser and greener population.[43]

The rapid increase in life expectancy most likely did not occur homogeneously over the whole population. Those with fewer resources, as for instances indicated by a low level of education, live generally shorter lives and also enjoy fewer years in retirement.[44,45] During times of improvements in morality conditions, often those with higher education benefited most, which lead to a further widening of inequalities in life expectancy.[46,47] The second pillar of the Dutch pension system ignores these differentials, resulting in an unintended reverse solidarity between the socio-economic subgroups, which may have aggravated over time.[48,49] Finally, it could also be the case that shifts in the educational distribution of the Dutch population contributed to the increase in life expectancy.

Understanding deviating trends in the Netherlands is not merely a national issue, but also potentially helps to broaden the knowledge on general drivers of longevity in high-income countries. There is an increasing body of research aiming at identifying such drivers, which so far had little success.[50] Particularly, the worse performance of improvements in life expectancy in the US, despite its economic prosperity and world record spending on healthcare, is a persistent conundrum in the field of public health and demography.[51-53] Since longevity trends in high-income countries are generally very homogeneous, especially the exceptions from the rule, as the case of the Netherlands, inform research and policy makers about the relation between life expectancy and its determinants. An example of such an instructive case is the quick convergence of life expectancy between Eastern and Western Germany since their re-unification in 1990, which emphasized the role of better healthcare and higher pensions for lowering mortality.[54,55] Another example is the case of mass privatisation of state-owned enterprises in countries of the former Soviet Union, which caused a dramatic drop in life expectancy.[56] More recently, country-specific differences in the impact of the great economic crisis of 2008 on mortality rates could be seen as gigantic experiment of how rising unemployment rates and painful austerity measures affect life expectancy.[57]

Besides contributing to the understanding of past mortality trends, identifying factors responsible for the exceptional stagnation and resumption of improvements in Dutch life expectancy would be particularly relevant for the field of mortality forecasting. Countries exhibiting irregular trends in mortality decline, such as the Netherlands, pose a central problem to mortality projection that has not been sufficiently solved so far.[58] While for most countries statistical offices apply a simple linear extrapolation of past trends to predict trends in the future, this is not feasible in the Netherlands because of its turbulent past. Although several solutions have been proposed for this purpose, any alternative projection model depends largely on arbitrary assumptions as long as the drivers of the irregular Dutch development in mortality rates remain unfound.[59] This means that the description, explanation and projection of Dutch life expectancy are strongly intertwined and cannot be assessed separately.

DESCRIPTION

Before explaining and projecting life expectancy in the Netherlands in further detail, a critical assessment of the indicator used to describe its trends is necessary. Ideally, life expectancy is measured in a cohort perspective by following a group of individuals over their entire lifetime from birth until death.[60] Since this typically requires about

a century of data, such life expectancies were rarely computed in practise, and as a short-cut period life expectancy at birth (PLE) is employed. PLE is constructed based on a life table, which translates the mortality rates of a single calendar year into an estimate of the average lifespan of a hypothetical cohort.[61] The advantage of this indicator over cohort life expectancy is the provision of up-to-date estimates. The drawback is that the estimated lifespan, according to PLE, is very sensitive towards annual changes in mortality. Recently, it has been demonstrated that sudden changes in underlying mortality conditions potentially cause misleading interpretations of changes in PLE.[62] This occurs because over time, avoided deaths are weighted by their full remaining life expectancy within the life table calculation.[63] Yet, if avoided deaths have only been postponed by few a weeks or months, a bias occurs that is named "tempo-effects," which leads to an overoptimistic view of improvement in mortality conditions.[64] Due to these tempo effects, some analysts worry that sudden improvements in life expectancy reflect a distorted picture of the actual changes in mortality conditions.[65]

EXPLANATION

For appraising whether Dutch life expectancy will continue its increase in the future, the identification of the factors responsible for its irregular development in the past is an important prerequisite. Like how the emergence of the Dutch Golden Age during the 17th century was by contemporary observers ascribed to a "Dutch secret," likewise is the unprecedented sudden improvement in life expectancy and the long period of stagnation in mortality improvements.[21,66] So far, various hypotheses have been put forward, which either focus on the period of stagnation, or on the period of resumption of life expectancy.

In an attempt to explain the slower improvement in Dutch life expectancy, the high prevalence of smoking and the survival of more frail and morbid elderly to higher ages was held responsible.[22,67] Further, the comparatively liberal attitude towards assisted suicide and withdrawal of end-of-life treatments in the Netherlands has been seen as indication of a generally less aggressive care of terminally-ill patients (although these cases remained a small fraction of total number of death).[22] Moreover, it was proposed that budget cuts in the healthcare system during the 1980s negatively affected older and sicker people, which are particularly depended on adequate medical care.[22,68] The combined effect of these factors was presumed to explain why the Netherlands was less successful in lowering mortality rates than other countries.[26,69] In particular, it was argued that countries with faster increases in life expectancy than

the Netherlands already entered a new stage of the health transition because they discovered more effective ways to fight specific ageing-related diseases.[70] However, none of these hypotheses could be convincingly confirmed so far.[21,66]

In other countries, which exhibited slower improvements in life expectancy, the negative impact of the progression of the tobacco epidemic was brought up as a major explanation. In addition to excessive alcohol consumption, below-average improvements in Danish survival were ascribed to high smoking rates.[13,71] Smoking-associated mortality was also held partly responsible for the slower improvement in Swedish life expectancy.[72] The same argument was brought up for the slowing-down of increases in life expectancy in the US, where tobacco consumption was much higher than in most other countries throughout the 20th century.[50]

While research on the stagnation of Dutch life expectancy identified multiple possible factors, only one possible factor has been proposed to explain the trend reversal. The recent increase in life expectancy after 2001 co-occurred with the relaxation of fixed hospital budgets.[4] In the wake of that healthcare reform, waiting times for medical treatment shortened, much more people were admitted to a hospital or a medical specialist, more surgical procedures were performed, and more medicine was prescribed.[4] Previously increasing trends in mortality of causes related to the older-ages, such as pneumonia and mental disorders, suddenly decreased since 2002.[4] In sum, this led to the hypothesis that the recent trend reversal of the development of Dutch life expectancy was mainly enabled through more and better healthcare, especially for the elderly.[4] Better medical treatment enabled by reforms of the healthcare system was also identified as a potential driver of sudden improvements in life expectancy in other countries, i.e. Denmark and in Ireland.[13,73] Likewise, the divergent responses of countries affected by the breakdown of communism in 1990, ranging from strong increases in life expectancy to dramatic decreases, were among other factors ascribed to the improvement or worsening of the provision and quality of healthcare.[55,57,74]

Besides the described changes in the healthcare system, smoking has been recurrently identified as one of the major drivers of variations in mortality trends among high-income countries, specifically for the Netherlands.[75-79] Since about the 1990s, smoking-associated mortality has been decreasing fast in males, contributing to the improvements in life expectancy.[80] The opposite trend was noted for females, where increasing smoking-associated mortality attenuated improvements in survival.[81] For this reason, smoking might explain why life expectancy stagnated among females, but it is not a factor to explain the sudden improvement in mortality conditions that

occurred for both men and women in 2001. Nonetheless, due to its dramatic impact on mortality trends, the explanation of deviating trends in Dutch life expectancy need to take smoking into account as it may masks other more important factors such as the influence of changes within the healthcare system.[22]

PROJECTION

Forecasting Dutch life expectancy into the future is hampered by the irregular development of mortality trends in the past. This is in contrast to the situation in most other Western countries, for whom a simple linear extrapolation of past mortality trends is feasible.[12] Only few Western countries exhibited longer periods of slower progress in mortality improvement like in the Netherlands.[22,82,83] It is unclear how such irregular country-specific mortality trends should be extrapolated, although several solutions have been proposed in the literature.[84]

The proposed solutions to project irregular mortality trends into the future could be grouped into three broad categories. The first strategy involves identifying the factors causing the irregularities, so that the irregular and the regular components of the mortality trends could be projected separately. Second, trends of other countries or of a group of countries should be taken into account, in order to ensure that countries with irregular mortality trends converge to the coherent international trends in the long run. Third, the identification of the time points where mortality trends changed should be sought, followed by an attempt to extrapolate only the most recent linear trend into the future. These three strategies are described in more detail below.

Traditionally, mortality projection models have been fuelled by all sorts of information, ranging from more subjective expert judgements to seemingly more objective information on causes of death or biological concepts of ageing, which are connected with the hope to better anticipate the future.[58,85] The performance of these models, augmented by explanatory factors, was generally disappointing, and most of them neither sufficiently explained past variations in mortality trends nor improved mortality projections.[8,58,59,86-88] Due to these recurrent failures, a new class of projection models emerged that aimed to improve forecasts through higher internal model complexity, instead of adding external information. For instance, such models accounted simultaneously for age-, period-, and cohort effects, or allowed different age groups to have their own time trend.[58,87,89,90] Alas, the better fit of such models to the historic data came at the cost of higher instability and an often worse performance if applied at different time-spans, age-ranges, or in other countries.[91-94]

Among the external factors included in projection models, only smoking has been found helpful for explaining and projecting irregular mortality trends.[77] Unlike for other factors, there is consensus about the causal effect of smoking on mortality, and appropriate tools exist to quantify its impact on mortality.[80,95,96] Recent projection models took smoking into account and modelled the components of mortality trends associated and not associated with smoking separately, since both exhibited their own specific pattern.[81,97] For most countries, smoking-free mortality trends developed more or less linearly, allowing linear extrapolation into the future.[77,81] By contrast, smoking-attributable mortality trends followed a bell-shaped pattern, and were predominantly driven by cohort-specific influences that could have been projected for instances by using an age-cohort projection model.[96,98] The most recent official projection by Statistics Netherlands applied this methodology using the trend of average smoking-free mortality of 21 countries as basis for the long-term Dutch mortality trend, complemented by a separate projection of Dutch smoking-attributable mortality.[81,99] For other countries, e.g. the US, data on the prevalence of smoking were employed to improve life expectancy forecasts.[100,101] However, this requires extensive survey data not available for most of the countries. Recently, various factors other than smoking, such as changes in the body mass index or changes in economic conditions, have been tested for their utility to improve mortality projection models, but their added value is less clear.[101-104]

A different strategy involves pooling trends of multiple countries, assuming that they share a common mortality trend evolving more regularly than the country-specific trends.[105-107] In this framework, exceptional developments were treated as mere temporary deviations converging to the common trend in the long run.[107] This assumption has been justified by the observation that the development in life expectancy was, on average, roughly linear among the high-income countries since about 1950, which suggests that a common rate of technological progress has been driving these improvements.[12,27] Since the reasons for temporary deviations from this linear long-term trend are poorly understood, the rate of convergence for a particular country is, to a large extent, driven by model assumptions, such as mean reversion.[81,107] Currently, this approach is applied by the Dutch Royal Actuarial Association to project period and cohort life expectancy in the Netherlands.[108]

Finally, it has been proposed to detect structural changes in mortality trends to extrapolate the most recent linear trend into the future.[109-114] This was justified by recent research that demonstrated that in most countries, at least one major shift in mortality trends occurred, which was interpreted as an emergence of a new and potentially lasting mortality regime.[17,92,111,115] It was argued that by using the period

after the trend change in mortality trends, a simpler and more robust extrapolation becomes feasible.[110,113,116] However, this requires that the most recent linear period indeed represent a new, enduring mortality regime. Furthermore, the extrapolation will be based on a very short period only if the mortality regime change occurred more recently.

As long as the underlying factors for the irregular development of Dutch life expectancy are unknown, its projected level of life expectancy will be strongly dependent on the choice of the projection approach and its assumptions. Therefore, it is important to combine the explanation and the extrapolation of deviating trends in Dutch life expectancy. Gaining insights concerning the projection of trends in Dutch mortality rates would contribute to the broader field of the extrapolation of irregular mortality trends. The approaches and methods used in this thesis could certainly also be applied to other countries exhibiting internationally deviating trends.

THIS THESIS

The aim of this thesis is to explain the recent trend reversal from stagnation to resumption of improvements in Dutch life expectancy and to project the deviating trends in Dutch life expectancy into the future.

The following questions will be tackled:

1) Does period life expectancy adequately reflect changes in Dutch mortality conditions?
2) What is the explanation of the trend reversal in Dutch life expectancy?
3) How could the deviating Dutch life expectancy trend be extrapolated?

STRUCTURE OF THE THESIS

Given the three distinct research questions, the thesis is divided into three parts, each contributing to one of the questions.

In the first part of thesis in chapter 2, the literature on tempo-effects is reviewed to assess under which conditions life expectancy provides a sound indicator of underlying mortality conditions. The second part, covered in chapters 3 and 4, tackles the deviating trend in Dutch life expectancy, mainly by analysing the impact of changes in smoking and in healthcare expenditures; specifically, chapter 3 looks at the impact of

changes in the impact of healthcare expenditures and smoking at the country level, and chapter 4 focuses on changes in healthcare utilization at the person level. The third part of the thesis, Chapters 5 and 6, deals with the projection of deviating mortality rates. These chapters assess whether accounting for smoking solves the problem of structural changes in mortality decline that poses the biggest problem for the linear extrapolation of long-term mortality trends. Specifically, in chapter 6, a projection model is developed to forecast total, gender-specific and educations-specific trends in life expectancy, particularly taking into account deviating subgroup-specific trends. The answers to the three research questions posed above are summarized in chapter 7. Also in chapter 7 is a discussion about limitations, methodological challenges, and implications for policy makers and further research.

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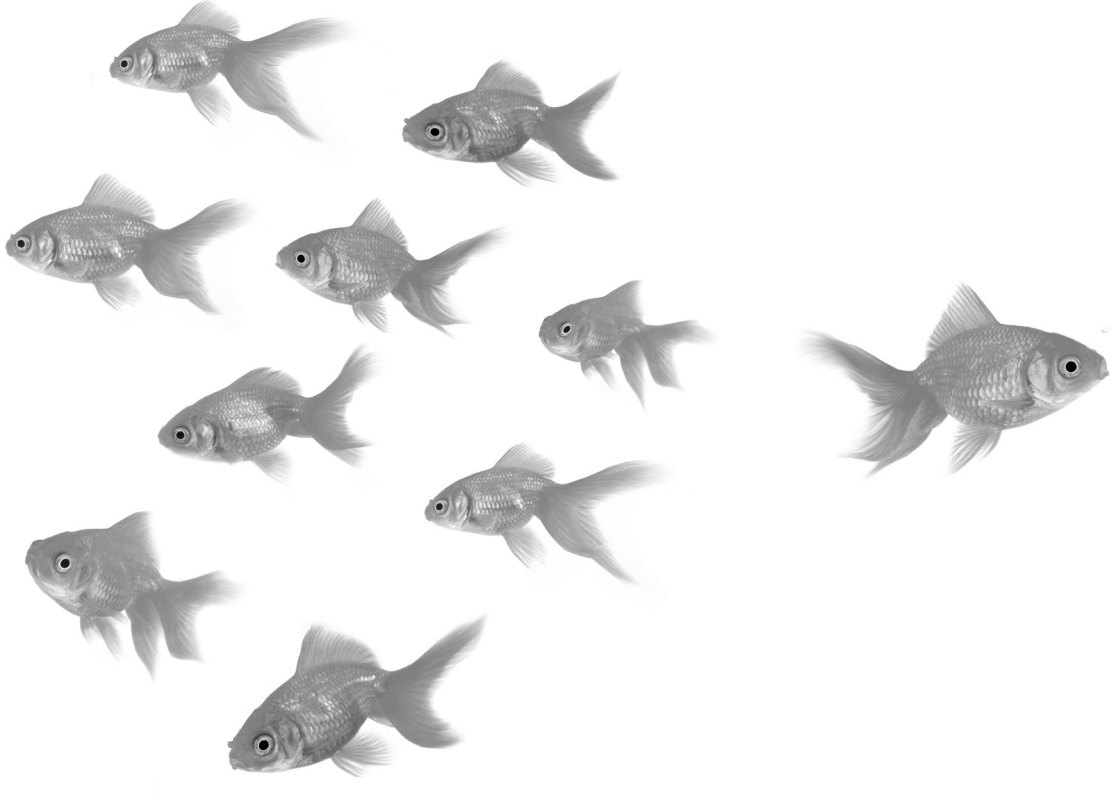
Part I

Description



2

Tempo effects may distort the interpretation of trend in life expectancy

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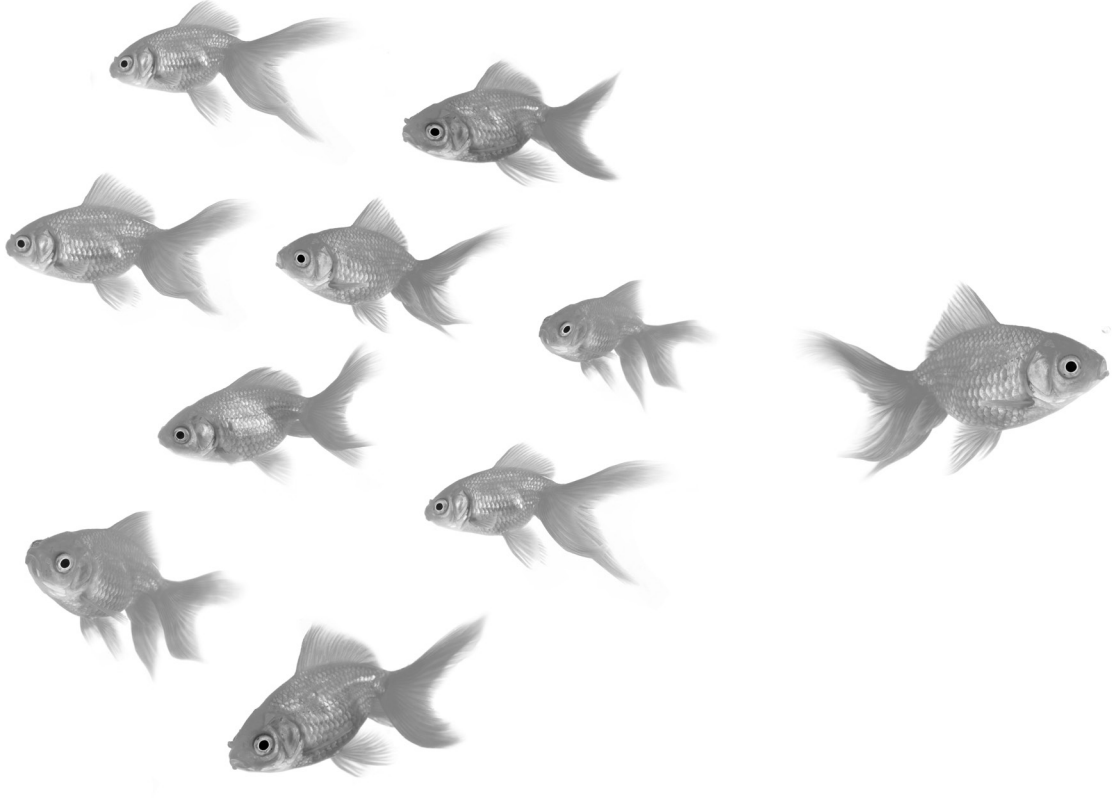

ABSTRACT

Objectives: Recently, a new interpretation problem of trends in period life expectancy has been discussed in the demographic literature. The so-called tempo effects arise if large numbers of deaths are suddenly postponed. In such conditions, the life table inflates longevity gains in the population because it weights avoided deaths with the full remaining life expectancy. This article explains how such effects occur and indicates their relevance using an illustrative example.

Study Design and Setting: Data of East and West Germany from the Human Mortality Database for the years 1990–2009 were used. We simulated a scenario that contrasts the observed life expectancy in West and East Germany with an alternative one based on the assumption of short-term postponements of deaths.

Results: Our example demonstrates that if tempo effects have distorted changes in life expectancy, the pace of improvement in underlying mortality conditions could be over- and underestimated.

Conclusion: We recommend that the assumptions of the life table, in this case about the remaining life expectancy of avoided deaths, are carefully evaluated in all applications. Interdisciplinary efforts to develop models to detect and quantify tempo effects from life expectancy calculations should be put on the research agenda.

INTRODUCTION

Period life expectancy (PLE) is one of the most used indicators of population health since it is based on data that are available in almost all countries in the world.[1] It is independent of the age structure of a population and has a clear and intuitive interpretation: the average number of years a newborn would live if current mortality rates would prevail throughout its life.[2] Population health researchers often use life expectancy to make comparisons between countries or over time, and usually interpret life expectancy as an indicator of the prevailing mortality conditions in the respective year.

Recently, doubts on the reliability of PLE as an indicator of current mortality conditions have been formulated, in particular during periods of sudden and large changes in mortality, as for instance during the turmoil caused by the transition to market economies in Eastern Europe during the 1990s or the sudden improvement in living conditions in East Germany after its re-unification with the Western part.[3-6] In these circumstances, the life table might give an overly optimistic or overly pessimistic impression of the change in mortality conditions. This problem has been discussed extensively in the demographic literature under the name of 'mortality tempo effects'.[7-20] These effects are defined as distortions in death rates due to short-term shifts in deaths to either higher or lower ages during rapidly changing mortality conditions.[21] The aim of this paper is to translate the main arguments of this discussion and their implications to a more general audience of population health researchers.

This new problem in the interpretation of life expectancy adds to some other, more widely known problems. Population health researchers are well aware of the fact that PLE is not a prediction of the number of years those born at that time will live, but merely a summary of prevailing age-specific mortality rates.[22] Also, it has been recognized that changes in PLE are also determined by positive or negative selection effects of past developments which could either work in a period or cohort direction.[23]

The structure of this paper is as follows. We first review the discussion on 'tempo effects' in the recent demographic literature, then we provide an illustration based on the convergence of PLE between former West and East Germany after the German unification, and we end with a few general conclusions and suggestions for population health researchers.

MORTALITY TEMPO EFFECTS

Life expectancy is defined as the average age to which the members of a birth cohort survive over their life course.[2] Its computation requires about a century of data and therefore usually a shortcut is to use observations of one period only. Here, all age-specific death rates observed at a single point in time are combined to calculate the average length of survival of a hypothetical cohort. The advantage of PLE over other summary measures of mortality is that it standardizes for differences in the age-structure of populations and provides an up-to-date summary of the prevailing mortality conditions.

However, starting in 2002, a series of papers surprised the demographic community, claiming that life tables are distorted whenever mortality is changing, due to so-called mortality tempo effects.[7,16,24] These effects belong to a larger class of distortions defined as *"an undesirable inflation or deflation of a period [...] indicator of a life-cycle event"*.[25] The general idea is that any period measure is prone to timing shifts of the events it counts, which in the case of mortality refers to postponements of deaths. Consequently, the change in the indicator does not necessarily represent the actual change in underlying mortality conditions in the population.

The extent of a 'tempo bias' depends on a rarely acknowledged feature of the life table. When mortality is changing, variations in death counts are weighted with the remaining life expectancy at each age.[12,16,26] Hence, the change in PLE over time is guided by hypothetical weights rather than the real improvement in survival time in the population. This might be reasonable if the additional survivors are as healthy as the average population, e.g. people saved from dying in a traffic accident.[19]

But this assumption is not reasonable in all situations. A simple example, given by Vaupel, is the case where every death in a population is suddenly postponed by one year.[27] Although this delay by definition increases the average survival time of the population by one year too, the PLE would temporarily increase to infinite, as no deaths are observed in the year in which the change happens. A less drastic case has been described by Bongaarts & Feeney, who show for a model population that a delay of all deaths by 0.3 years during a period translates into an overly optimistic change in PLE of about 3 years instead of the expected 0.3 years.[7]

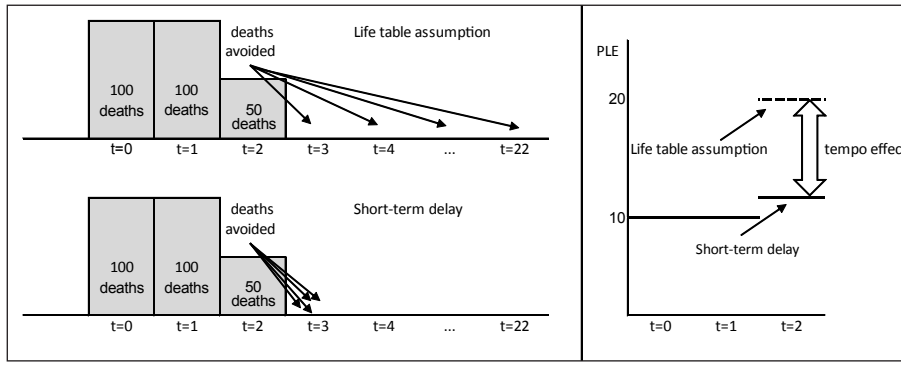

Figure 1 Illustration how the implicit assumption of the life table inflates period life expectancy

The mechanism underlying mortality tempo effects is schematically shown in figure 1, where a theoretical population is shown in which annually 100 deaths occur to 1000 person-years at risk. Its PLE is 10 years, calculated as one over the mortality rate of 0.1.[2] At time t=2, however, suddenly half of the deaths are saved and shifted to the next period. This 50% reduction in the mortality rate will increase PLE to 20 years according to the conventional life table calculations, computed as one over 0.05. Implicitly, these calculations assume that the deaths avoided at time t=2 will be gradually distributed over the next time-periods according to the current average remaining life expectancy, which is 20 years. However, the deaths are in fact only postponed by one year. If this shorter delay would properly be accounted for in the life table calculations, life expectancy at time t=2 would only be 10.5 years, since in fact half of the population gains one year. The difference between 20 and 10.5 is the tempo effect, here 9.5 years. Similar distortions will occur in case of a sudden increase in the number of deaths.

The size of a tempo effect depends on the difference between the amount of time the death events were shifted at each age (short-term shift) and the remaining life expectancy at that age (life table assumption). The example given above shows the consequence for the estimation of PLE if deaths were shifted by one year, while the remaining life expectancy at each age is 20 years. In addition to a single shift also a continuous shift might occur.[8] While in the case of a single shift, PLE is only overestimated (or underestimated) in the year the shift occurs, in the case of a continuous shift a permanent inflation (or deflation) occurs.

No matter which of these details applies in a practical case, a general precondition for the existence of tempo effects is a strong increase or decrease in observed PLE re-

lated to an underlying short-term shift in the age at death. The latter is by definition a latent construct as the actual shifts are not directly observable. However, if a clear intervention could be identified that might cause a large short-term shift of deaths, the potential existence of tempo-effects should be taken into account. Such an approach will be demonstrated in the next section for the case of the former German Democratic Republic where after reunification in 1990 both preconditions for tempo effects - a clear intervention and a rapid change in mortality – were met.

AN ILLUSTRATIVE EXAMPLE

To illustrate the impact tempo-effects may have on trends in PLE, we make use of the sudden changes observed after the reunification of Germany. The populations of West and East Germany had lived for 40 years in completely different economic and political systems, until both parts were reunited in 1990. Separation and unification coincided with divergence and convergence in PLE of East Germany as compared to its western counterpart in particular pronounced for females (figure 2). Starting from an equal level of about 75 years in 1970, the gap between the two parts of Germany increased to almost three years in 1990 and finally disappeared again after reunification.

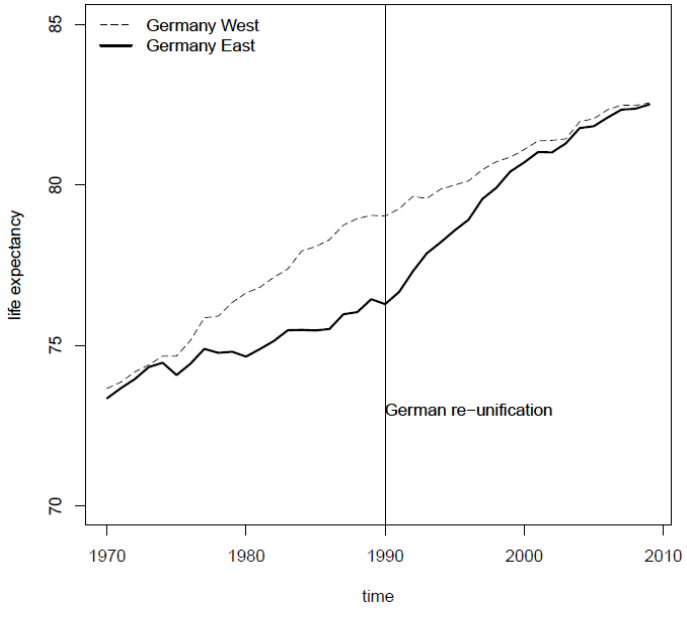

Figure 2 Trends in period life expectancy at birth in West and East Germany, females 1970-2009

The case of the rapid East-West German convergence in life expectancy calls for a closer examination.[28,29] Detailed analyses suggest that general living conditions even deteriorated in East Germany immediately after reunification, due to privatization, mass unemployment and a significant increase in motor vehicle accident mortality.[30] At the same time health-related lifestyles improved only slowly.[31] The only reasonable explanation for a sudden decline in mortality rates is the rapid improvement of medical technology and health care, in particular nursing care.[6,11,32,33]

If this explanation is correct, mainly frail persons have benefited from improved health care right after reunification. Consequently a large fraction of postponed deaths might have been delayed by a short period only, and tempo-effects are likely to have occurred. We have simulated the impact of such tempo effects on life expectancy in figure 3 using data from the Human Mortality Database.[34] (see online supplement for data and methods). Our scenario contrasts the conventional PLE in West and East Germany with an alternative one based on the assumption of short-term postponements of deaths. For the latter we assumed that the fraction of avoided deaths postponed by a short time only was large immediately after reunification, and gradually diminished in later years. As visible in figure 4, after reunification in 1990-94 the rate of improvement of life expectancy in the Eastern part of Germany is much lower in this alternative scenario than suggested by conventional calculations of life expectancy, while it is higher than suggested by conventional calculations in the later years 2000-09.

In other words, if tempo effects have distorted changes in life expectancy, the pace of improvement in underlying mortality conditions was over- and then underestimated. Solely based on aggregate mortality data, the actual extent of these de- and inflation processes is hard to quantify. However, this example demonstrates that looking at PLE only may leads to a misleading interpretation.

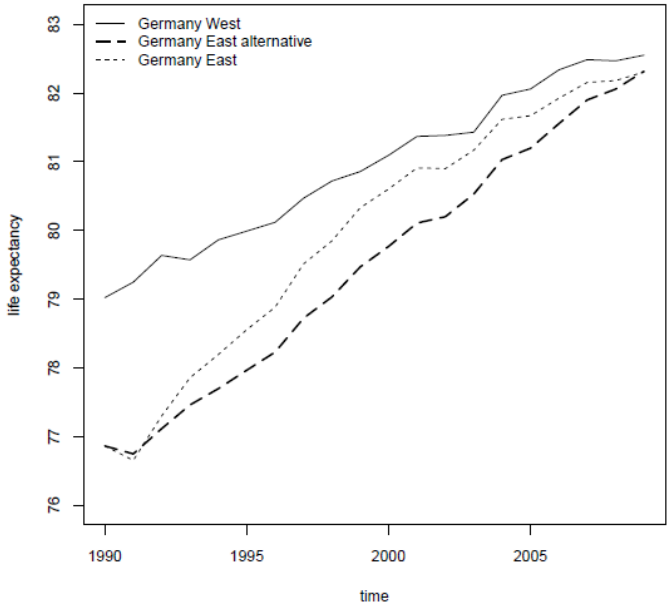

Figure 3 Trends in period life expectancy at birth in West and East Germany after unification, females 1990-2009, with an alternative scenario of improvement in survival conditions based on the assumption of short-term delays in deaths.

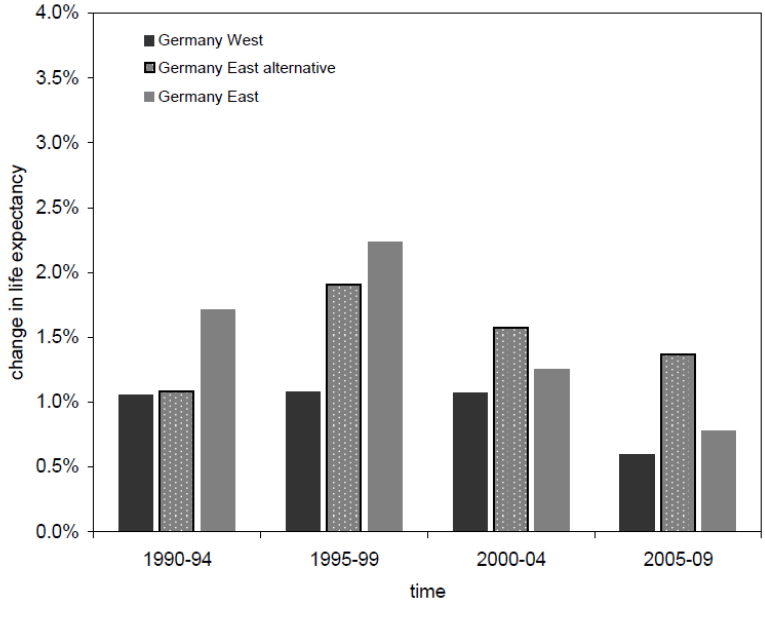

Figure 4 Percentage change in period life expectancy at birth in West and East Germany after unification, females 1990-2009, with an alternative scenario of improvement in survival conditions based on the assumption of short-term delays in deaths.

DISCUSSION AND CONCLUSION

The present paper introduces a problem so far not acknowledged in public health, but extensively discussed in aging and mortality research in the recent decade. Those who apply life tables should be aware of the assumption it uses to assign remaining life-years to avoided deaths. If during times of rapid progress a large fraction of deaths is merely delayed by a few months or years, an overestimation of the real improvement in underlying mortality conditions is likely to take place. Similarly, if during times of a rapid increase in death rates a large fraction of deaths is merely brought forward by a couple of months or years, an overestimation of the real deterioration in underlying mortality conditions is likely to occur. If the change in life expectancy is used as an indicator for the improvement of underlying survival conditions of the population prevailing at that time, errors of interpretation may occur.

Although tempo effects in mortality rates have widely been discussed in the demographic literature, the concept has rarely been applied so far, which is due to the absence of a consensus on an appropriate adjustment tool.[35] The key element thereby is the estimation of the rate of change in mortality conditions, which is responsible for shifting deaths to either higher or lower ages. Current techniques to estimate this change in mortality conditions first reconstruct the full history of the currently living cohorts, taking into account mortality rates of the past, and then quantify the change in the average survival of these cohorts from one year to the next.[24,35] However, these techniques have been criticized because they do not provide falsifiable predictions.[10,18,36] This fundamental problem could be tackled by identifying the presence or absence of a large fraction of short-term shifts in death events between two periods for a population, in particular related to the presence of persons with a high mortality risk. For this purpose detecting changes in the health status of the population could be useful. If indeed short-term shifts let a large fraction of the people that are about to die survive a bit longer, one could expect to observe a detectable accumulation of frail persons in the subsequent years. However, observed increases of frailer persons can also originate from an increase in the incidence of diseases instead of improving survival.

To conclude further research should focus on the empirical identification of short-term delays of deaths, and combine statistical models and various empirical data sources to test for the existence of tempo effects and their impact on the change of PLE.

As suggested by the example of East Germany, health care as an important determinant of life expectancy is one of the main candidates being able to immediately

lengthen the life of people that are about to die by a few months or years.[11,21,37]
A recent example of such a health-care related influence is the case of the Netherlands, where an expansion of health care for the elderly and more frequent use of life-prolonging interventions, facilitated by relaxation of budgetary constraints of hospital expenditures, coincided with a sharp increase in PLE.[38]

APPENDIX

Our approach to compute the effects of alternative assumptions about lifesaving is not meant to estimate the "real" life expectancy. Rather it aims to show the sensitivity of the life table to deviations from its assumptions, which is in tradition with comparable papers that deal with problems of measuring survival trends at a population level.[39-41] As simplification we divide the life table population in two different groups: one group is supposed to die according to the present period life table, while in the second group deaths are delayed by one year.

A model to account for the impact of short-term shifts on life expectancy

It has been shown that assuming an equal progress against mortality over age, the time-specific relative change in period life expectancy relates to the relative change in death rates *r(t)* via Keyfitz entropy H [42,43]

$$\frac{\partial e(0,t)/\partial t}{e(0,t)} = \rho(t)H(0,t) \tag{1}$$

While *p(t)* is the relative change in mortality rates over time *t*, *H(0,t)* denotes the entropy of the life table and *e(0,t)* life expectancy at birth. The numerator of the entropy *H(0,t)* measures the life-time lost to deaths, by weighting the death events at each age *x* and time *t, d(x,t),* by their remaining age-specific life expectancy *e(x,t)*. This value is divided by the life expectancy at birth resulting in the relative change of the life table due to proportional changes in mortality rates.

$$H(0,t) = \frac{\int_0^{\omega} d(x,t)e(x,t)dx}{e(0,t)} \tag{2}$$

Multiplying (1) with the life expectancy at birth and using (2), the absolute change in life expectancy over time is expressed as follows.

$$\partial e(0,t)/\partial t = \rho(t)\int_0^{\omega} d(x,t)e(x,t)dx \tag{3}$$

The number of saved deaths, $\varphi(x,t)$, is defined in the following way.

$$\varphi(x,t) = d(x,t)\rho(t) \tag{4}$$

Using this relation (3) simplifies to

$$\partial e(0,t)/\partial t = \int_0^{\omega} \varphi(x,t)e(x,t)dx \quad . \tag{5}$$

This relation demonstrates that the change in life expectancy equals the weighted sum of the number of saved deaths and their remaining life expectancy. Thus, the change in life expectancy is only a valid proxy of the change in the underlying mortality conditions if the avoided mortality events are delayed by the remaining life expectancy at each age. If however these shifts are shorter, the change in period life expectancy overestimates the change in mortality conditions.

To model short-term shifts, we follow[42] and introduce alternative age-specific weights e^{alt} resulting in

$$\partial e^*(0,t)/\partial t = \int_0^{\omega} \varphi(x,t)e^{alt}(x,t)dx \quad . \tag{6}$$

Now the alternative change in life expectancy e^*, is related to the same amount of saved deaths but an alternative average remaining survival time.

We define short-term shifts as avoided deaths, whose average remaining survival time is one year ($e^{alt}=1$). This simplifies (6) to

$$\partial e^*(0,t)/\partial t = \int_0^{\omega} \varphi(x,t)1dx = \int_0^{\omega} \varphi(x,t)dx \quad . \tag{7}$$

Since the integral over all life table deaths is one, and the relative change in mortality rates is assumed to be universal over age, (7) reduces to

$$\partial e^*(0,t)/\partial t = \int_0^{\omega} \varphi(x,t)dx = \int_0^{\omega} d(x,t)\rho(t)dx = 1\rho(t) = \rho(t) \quad , \tag{8}$$

if all saved deaths in a population reflect short-term shifts.

However, if only a fraction π of all saved deaths gains the fixed increment of one year, and the other fraction is saved according to the standard life table assumption (as expressed in (5)), the change in life expectancy could be modeled by (using (5) and (8))

$$\partial e^*(0,t)/\partial t = \pi(t)\rho(t) + (1-\pi(t))\int_0^\omega \varphi(x,t)e(x,t) \quad , \tag{9}$$

which will be used to simulate the influence of short-term shifts on life expectancy below.

Simulation of the change in life expectancy in East Germany

To simulate the impact of short-term shifts on mortality conditions in East Germany after unification, we fit a Gompertz mortality model to the death rates obtained from the Human Mortality Database,[44] between age 40 and 80 observed in 1990.

$$\mu(x,t) = \alpha e^{\beta x} \tag{10}$$

If the relative change in death rates is universal over age and mortality at younger ages is neglected, the change in life expectancy in a Gompertz model over time could be approximated in the following way.[45]

$$\partial e(0,t)/\partial t = \rho(t)/\beta(t) \tag{11}$$

This relation is used to express the observed growth rate in life expectancy as a proportional shift of a survival function, defined by the Gompertz model with parameter $\beta(t)$. To compute the growth rate in mortality rates, (11) is rearranged to

$$\rho(t) = \frac{\partial e(0,t)/\partial t}{\beta(t)} \quad , \tag{12}$$

whereby in our application the growth rate in life expectancy is approximated by the differences of the log of life expectancy at baseline, t=1990, and life expectancy at year t.

Using (12), the model expressed in (9) reduces to

$$\partial e^*(0,t)/\partial t = \pi(t)\rho(t) + (1-\pi(t))\frac{\rho(t)}{\beta(t)} \quad . \tag{13}$$

Regarding the fraction of short-term shifts for East Germany, we assume that its fraction π is 0.5 right after the unification and reduces linearly to a level of 0 in 2009. This means that we assume that in 1991 50% of all avoided deaths have a remaining life expectancy of one year, which decreases linearly to 0% in 2009. For West Germany we assume that there are no short-term shifts and compare the simulated results of East Germany to the observed values of West Germany.

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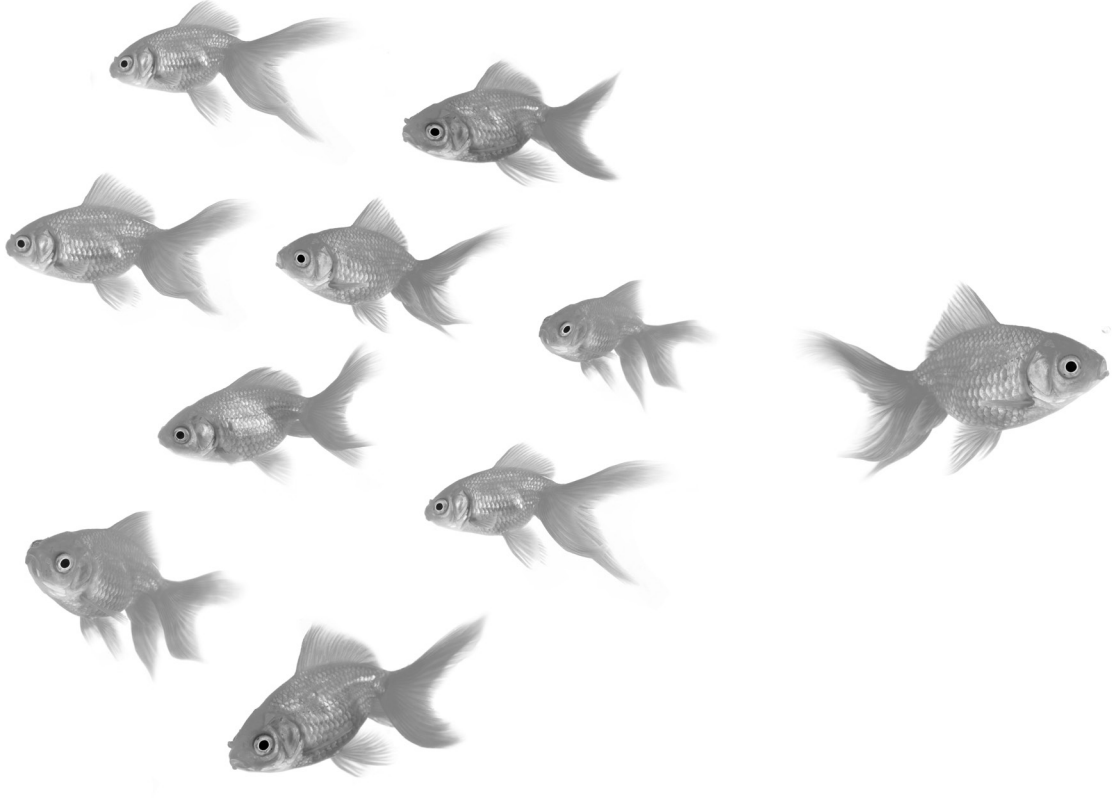
Part II

Explanation

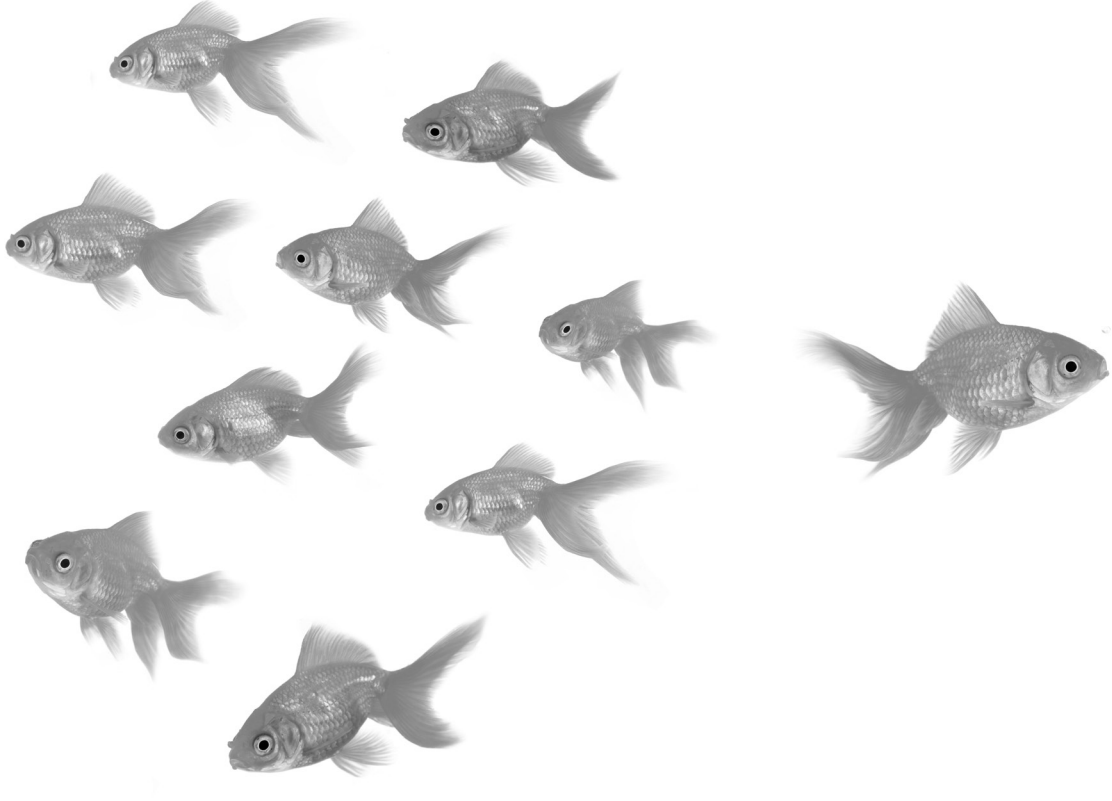


3

Quantifying the contribution of changes in healthcare expenditures and in smoking to the trend reversal in Dutch life expectancy

Frederik Peters, Wilma J. Nusselder, Nadine Reibling, Christian Wegner-Siegmundt, Johan P. Mackenbach

Submitted



ABSTRACT

*Background***:** Since 2001 the Netherlands exhibited a sharp upturn in life expectancy (LE) after a longer time of slower improvement. We assessed whether changes in healthcare expenditures (HCE) explain the trend reversal in Dutch LE. As alternative explanation we assessed the impact of changes in smoking.

Methods: To quantify the contribution of changes in HCE to changes in LE we estimated a health-production function using a dynamic panel regression approach with data on 19 OECD countries between 1980 and 2009, accounting for temporal and spatial correlation. We estimated smoking-attributable mortality, using the indirect Peto-Lopez method.

Results: As compared to 1990-99, during 2000-09 Dutch LE increased 1.8 years more in females and 1.5 years more in males. Whereas changes in the impact of smoking between the two periods made practically no contribution to the acceleration of the LE increase, changes in the trend of HCE added 0.9 years to the LE increase between 2000 and 2009. The exceptional reversal in the trend of LE and HCE was not found in the group of the other OECD countries.

Conclusion: This study suggests that changes in HCE and not in smoking have made an important contribution to the trend reversal in Dutch LE, and supports the view that investments in healthcare are becoming more and more important for further progress in life expectancy.

INTRODUCTION

Since the 19th century life expectancy at birth has increased dramatically in Western high-income countries.[1] During the second part of the 20th century, the rate of increase in most of these countries has been very similar with no disruptions nor signs of slowing down until the present day.[2-4] This remarkable finding led to the belief that progress in survival is a universal feature largely independent of country-specific particularities such as the set-up of the health system or differences in health-specific behavior.[2] However, this hypothesis was challenged by some particularly successful economies witnessing longer periods with a slower increase or even stagnation in life expectancy.[5-10]

The most striking case for such a stagnation in improvements of life expectancy is the Netherlands. The country made hardly any progress against mortality during the 1980s and 1990s, in particular for women.[6] However, in the year 2002 a sudden and strong increase in life expectancy started continuing up until today.[11] To explain this trend reversal, the main hypothesis is that additional investments in the health sector lead to the improvements in survival particularly at older ages.[11] On the other hand, an exceptionally high impact of damage caused by smoking has been mentioned frequently as a competing explanation, particularly relevant during the stagnation period of Dutch life expectancy improvements.[7,12,13] Despite considerable research efforts, convincing evidence on the factors behind the stagnation period and subsequent period of resumption of the improvement in Dutch life expectancy is lacking.[14,15]

To fill this gap, our study is the first to quantify the impact of healthcare expenditures to the change from a slower increase to a rapid improvement in Dutch life expectancy, while at the same time assessing the contribution of smoking as alternative explanation. Additionally, we evaluated whether the internationally deviating trends in Dutch life expectancy corresponded to internationally deviating trends in healthcare expenditures or smoking.

For this purpose, we contrasted the results for the Netherlands with a group of 18 comparable countries of the Organization for Economic Co-operation and Development (OECD) based on data covering life expectancy, lung-cancer mortality, health-care expenditures and gross-domestic product for the years 1980-2009. To estimate the impact changes in healthcare expenditures had on life expectancy we employed a panel data analysis accounting for unobserved factors, cross-country variation, and dynamic effects.[16] The impact of smoking was estimated by the indirect Peto-Lopez method.[17]

DATA AND METHODS

Data Collection

We included all countries in the analysis being members of the OECD at least since 1980 because the annual provision of country-specific data is legally linked to this membership status. We excluded Luxembourg because it provides data on healthcare expenditures from 1999 onwards only and the USA due to its fundamentally different health system.[18,19] This leaves 19 countries in our study: Australia, Austria, Belgium, Canada, Denmark, Finland, France, Iceland, Ireland, Italy, Japan, The Netherlands, Norway, New Zealand, Portugal, Spain, Sweden, Switzerland and the United Kingdom. Information on mortality rates and life expectancy at birth were obtained from the Human Mortality Database.[20] Sex-specific lung-cancer death rates in five-year age groups for the ages 35 until 89 were taken from the WHO mortality database to obtain smoking-attributable fractions.[21] To model the influence of healthcare expenditures on life expectancy at birth, we collected data on healthcare expenditures (HCE) and gross-domestic product (GDP) for the years 1980-2009 from the 2014 OECD Health Data collection.[22]

Statistical Analysis

Our analysis quantified the contribution of changes in smoking and healthcare expenditures to changes of life expectancy at birth between 1990 and 2009 using two different techniques.

The impact of smoking on mortality was estimated by using the validated indirect Peto-Lopez method, which uses lung-cancer death rates as indicator for the cumulative damage of smoking to all other causes of death on the basis of relative risks obtained from a large cohort study.[17,23,24] This resulted in country-specific annual smoking-attributable fractions in 5-year age groups from age 35 until 85 and an open-ended category 85+. We used these fractions to remove smoking related mortality from the observed mortality rates that were also tabulated in annual 5-year age groups to compute smoking-free life expectancy applying life table methods.[25] The smoking-attributable fraction at age 85+ has been applied only to mortality between age 85 and 90, because at older ages the impact of smoking is very small and cause-of-death statistics are less trustworthy.[17,26] For a few calendar years, where lung-cancer deaths were missing, we interpolated smoking-attributable fractions using local polynomial regression fitting.[27]

For the estimation of the impact of healthcare expenditures on life expectancy no evaluated tool exists.[28] Therefore, we performed a separate analysis beforehand comparing different model approaches, presented in detail in the web supplement. In brief, we have estimated a health-production function relating monetary inputs in healthcare to gains in life expectancy building on recent developments in the analysis of relationships in panel data. Specifically, we modelled a dynamic response of life expectancy to changes in healthcare spending and allowed for heterogeneity in this relation among countries.[16] Moreover, we included spatially correlated common factors in the production function accounting for the fact that developments in the countries do not occur independently of each other.[29,30] Our theoretically preferred model was compared to alternative specifications on the basis of model fit and panel residual diagnostics.[31] In our main analysis, we multiplied the parameters of this preferred model with the changes in country-specific healthcare expenditures to quantify their impact on life expectancy between 1980 and 2009.

Finally, we compared the gain in life expectancy in 1990-09 and 2000-09 with the gains attributed to changes in smoking and with gains due to increases in healthcare expenditures for the Netherlands and the average of the other 18 countries. We computed 95% confidence intervals around the estimates of the impact of healthcare expenditures since their contribution is more uncertain than the contribution of smoking. This was performed by means of simulation (10000 runs) using the variance-covariance matrix of the panel regression results.

RESULTS

Descriptive trends

Comparing the Netherlands to the average of the other 18 OECD countries, we found that Dutch life expectancy increases slower up until about 2002 and faster afterwards, which was more pronounced for females (figure 1).

Trends in the age-standardized lung-cancer death rate, which served as input for the estimation of smoking-associated mortality, reveal large gender differences (figure 2). The exceptionally high lung-cancer death rate in Dutch males in 1990 decreased rapidly over time, while Dutch females exhibited increasing rates up during the whole period of observation.

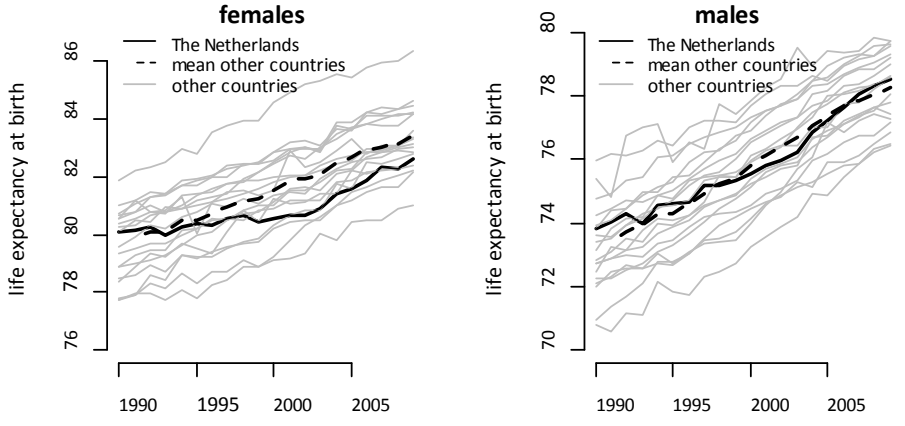


Figure 1 Trends in female and male life expectancy at birth in the Netherlands and 18 other OECD countries between 1990 and 2009

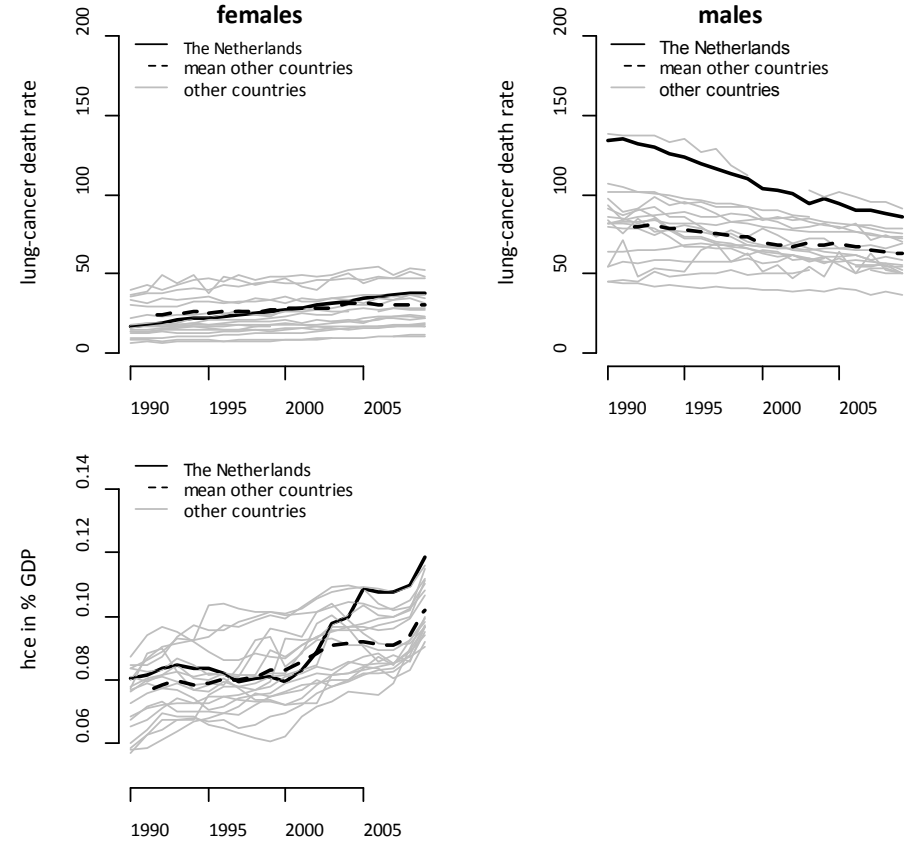


Figure 2 Change in age-standardized lung-cancer death rate per 100'000 persons in females and males (upper panel) and healthcare expenditures measured in % of GDP (lower panel) in the Netherlands (black) and the mean of 18 other OECD countries (black dashed line) between 1990 and 2009

The pattern of trends in healthcare expenditures partly resembled the pattern of trends in life expectancy (figure 2): Expressed as proportion of the GDP, healthcare expenditures in the Netherlands expenditures stagnated up until 2001 and rose afterwards, while in the other countries there was a continuous increase over time.

The effect of changes in healthcare expenditures on life expectancy

A dynamic relationship between healthcare expenditures and life expectancy was confirmed within our sample of 19 countries in 1980-2009 (see web supplement): A one-percent increase in healthcare expenditures translates into an increase of life expectancy of 0.036 percent in the long run (95% CI: 0.026-0.058). In practice, doubling the resources of the GDP allocated to healthcare from 8% to 16%, would translate in to a growth in life expectancy from 80 to 82.08 years within a decade.

Impact of changes in healthcare expenditures and smoking on changes in life expectancy

The contribution of changes in healthcare expenditures to changes in life expectancy is depicted in figure 3. While Dutch females and males gained about one year of life between 1990 and 2009, this gain was more modest for the other countries on average. The impact of healthcare spending in the Netherlands followed a clear pattern with a stagnation until about 2000 followed by a rapid increase afterwards. In the other countries a stable linear increase could be noted.

The impact of changes in smoking on changes in life expectancy between 1990 and 2009 occurred in a linear manner for both the Netherlands and the mean of the other countries (figure 3). Dutch women suffered above-average losses of years of life due to more damage from smoking while Dutch men gained above-average years of life due to less damage from smoking.

Table 1 summarizes the impact of changes in smoking and healthcare expenditures. Compared to the period 1990-99 the change in Dutch life expectancy accelerated by 1.8 years (females) and 1.5 years (males) in the period 2000-09. Changes in healthcare expenditures contributed 0.9 years to this acceleration, whereas changes in smoking made practically no contribution. Taking into account the uncertainty around the estimates, the larger contribution of changes in healthcare explains about 20 to 90 percent of the acceleration in the improvement of Dutch life expectancy.

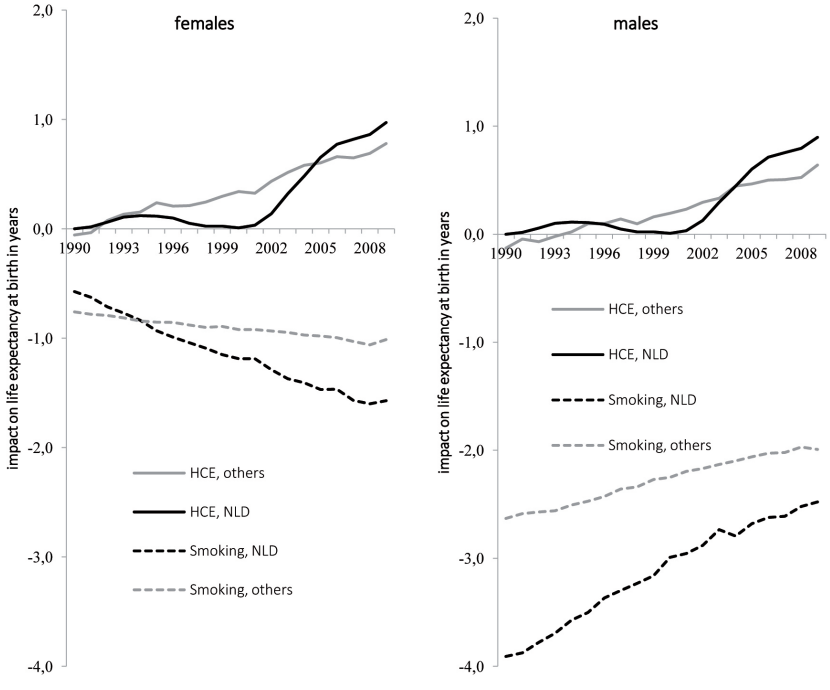


Figure 3 Estimated impact of changes in smoking and healthcare expenditures on life expectancy at birth in females (left panel) and females (right panel) in the Netherlands and the mean of 18 other OECD countries 1990 and 2009

Table 1 Decennial change in life expectancy at birth and contribution of smoking and healthcare expenditures (HCE) in the Netherlands (NLD)

	period	observed change in LE	diff 1990-99 and 2000-09	change in LE due to changes in smoking	diff 1990-99 and 2000-09	change in LE due to changes in HCE	95% CI	diff 1990-99 and 2000-09	95% CI
					Netherlands				
females	**1990-99**	0.3		-0.6		0.0	(0.01 to 0.05)		
	2000-09	2.1	**1.8**	-0.4	**0.2**	1.0	(0.30 to 1.62)	**0.9**	(0.29 to 1.57)
					mean of the other countries				
	1990-99	1.7		-0.1		0.3	(0.09 to 0.52)		
	2000-09	1.9	**0.2**	-0.1	**0.0**	0.5	(0.14 to 0.77)	**0.2**	(0.05 to 0.25)
					Netherlands				
males	**1990-99**	1.5		0.7		0.0	(0.01 to 0.04)		
	2000-09	3.0	**1.5**	0.5	**-0.2**	0.9	(0.27 to 1.49)	**0.9**	(0.27 to 1.45)
					mean of the other countries				
	1990-99	2.2		0.4		0.3	(0.09 to 0.47)		
	2000-09	2.5	**0.3**	0.3	**-0.1**	0.4	(0.13 to 0.70)	**0.1**	(0.00 to 0.20)

In the other countries there were neither large differences in the change of life expectancy nor in the change of the impact of smoking and healthcare expenditures. Interestingly, the slightly larger change in life expectancy during 2000-09 as compared to 1990-99 was almost fully attributed to a slightly larger change of healthcare expenditures.

Sensitivity analysis

To assess the robustness of our results, we used an alternative indicator for healthcare expenditures - per capita healthcare expenditures expressed in US$ - and estimated smoking-attributable fractions with a different regression-based approach suggested by Preston et al.[32] The central findings of our analysis did not substantially change (results not shown).

DISCUSSION

This paper is the first quantitative assessment of the contribution of healthcare expenditures and to the recent trend reversal of Dutch life expectancy also accounting for the contribution of smoking. Our results suggest that changes in healthcare expenditures contributed largely to the trend reversal, while there was hardly any contribution of changes in smoking. Moreover, we found that the exceptional role of healthcare expenditures on life expectancy was indeed a unique feature of the Netherlands and not present to the same extent in our comparison group of 18 similar OECD countries.

Evaluation of data

Since we have included only high-income countries with well-established systems of national statistics that were members of the OECD for at least as long as the study period, we are confident about the comparability and quality of the data. Further, missing information that often casts doubt on international comparison were not an issue in our study since our sample contains almost 100% complete information on the study variables.[16] Data on all-cause mortality and population exposure as well as healthcare expenditures and GDP was obtained from harmonized databases.[33,34] Information on lung-cancer counts are - as same as any other cause-of-death specific data - subject to more uncertainty due to variations in national coding practices and

changes in coding behavior over time.[35] However, unlike for other causes, these issues are of minor relevance for lung-cancer data where detection is comparatively clear and coding schemes are established for a longer time without drastic changes.[36]

Evaluation of methods

The most important part of our paper is the quantification of the effect of changes in healthcare expenditures on changes in life expectancy. Although there is a longer tradition of assessing this relationship in empirical research, up until today there is no consensus on the appropriate strategy to estimate it.[28,37,38] We believe that this is to a large extent due to the absence of appropriate tools to analyze panel data that became available only few years ago.[16,31] A central insight of this new literature is that the relation between healthcare expenditures and life expectancy can be estimated more reliable for a group of countries than for a separate country alone.[39,40] However, this comes at the cost of providing only "insights regarding the central tendency of the panel" so that we had to apply the same parameter for every country in the main part of our analysis for computing the contribution of healthcare expenditures on life expectancy.[41] Nevertheless, this assumption appears to be plausible for the countries in our sample, sharing fairly similar political and economic structures and all providing almost full public coverage of necessary healthcare services.[42] If there were in fact substantial differences in the efficiency of the production of health between the Netherlands and the other 18 countries this is likely within the uncertainty bounds around our central results.

In our analysis, we assumed that variations in healthcare spending causally explain variations in life expectancy ruling out the opposite direction, i.e. that an improvements in life expectancy cause additional costs in the health sector.[43] A study explicitly testing the influences of such reverse causality found that most health outcomes were not related to health-care costs.[44] Furthermore, a growing economic literature reports that with postponing death also costs are postponed deeming increases in life expectancy to be less relevant for growing healthcare expenditures.[45]
Regarding the effect of smoking, we applied a well-established tool that has proven to be reliable and informative in numerous applications.[26,46,47] Although the indirectly modeling of the damage from smoking on other causes-of-death partly relies on a set of assumptions, it has been shown that different approaches with different assumption arrived at almost similar estimates.[24,32] Furthermore, the estimated smoking-attributable fractions plausibly describe the variation of the timing in the epidemiological transitions between countries and sexes.[47-49]

An important assumption of our study is that the contribution of healthcare expenditures and smoking on trends in LE could be quantified independently of each other. This might be a simplistic view, given that a well-funded healthcare system certainly mitigates the consequences of smoking particularly for smoking-induced cardiovascular diseases, where effective treatments exist. Given the lack of evidence on that topic in the literature it is hard to speculate about the consequences of our assumption on the study results.

Comparison with other studies

The results of this study underpin the hypothesis that changes healthcare expenditures were the main driver of the trend reversal of life expectancy in the Netherlands.[11] While previous analysis merely demonstrated the presences of a common trend break for these two variables at around 2002, our study adds (1.) that changes in healthcare spending were generally positively associated with changes in life expectancy within high-income countries, (2.) that the size of the impact of healthcare could plausibly explain the acceleration of the Dutch life expectancy increase, (3.) that the exceptionally changes in healthcare spending and the exceptional trend reversal in life expectancy at around 2002 was indeed a particular feature of the Netherlands and (4.) that a the major alternative explanation for the trend reversal – changes in smoking - could be ruled out. This four different aspects of explanation of the Dutch trend reversal counter arguments "that there is no observable relationship with changes healthcare funding whatsoever".[15]

The importance of the contribution of healthcare expenditures to the trend reversal in Dutch life expectancy is in line with case studies of other countries with rapid trend reversals in life expectancy. The natural experiment of the separation and subsequent unification of Germany demonstrated that improvements of the healthcare infrastructure could affect life expectancy immediately and with a large impact.[50,51] In Denmark, a huge investment program to reduce cardiovascular mortality was held partially responsible to explain the upturn in Danish life expectancy after a longer period of stagnation.[8,52]

Nevertheless, the example of Japan showed that a large increase in life expectancy could be achieved without a large increase in healthcare spending, while the example of the US demonstrated that great investments in healthcare spending do not necessarily lead to a large improvements in life expectancy.[53,54] This highlights the relevance of contextual factors such as a universal coverage of healthcare services or more general cultural aspects.[44,55]

Our results ascribed up to halve of the increase in life expectancy to changes in healthcare expenditures, i.e. for Dutch women in 2000-09. Detailed studies that combined knowledge on the effect of new medical treatments with cause-specific mortality data and disease prevalence estimated that innovations in healthcare have plausibly contributed to at least 50% of the gains in life expectancy during the recent decades.[56-58]

Explanations of findings

A major channel how variations in the Dutch healthcare expenditures could have affected mortality is the budgeting of hospital care. During the early 1980s, at the time the improvements in Dutch life expectancy started to slow-down, policy makers decided to introduce fixed hospital budgets resulting in a strong reduction in the admission of new patients, of employed personal and even in a closure of some hospitals.[59,60] Together with the legalization of euthanasia in 1985 and an increasing incidence of end-of-life decisions such as the withdrawal of artificial nutrition, this reflected a general attitude towards a less aggressive treatment of older and terminally-ill patients.[6,61,62] At the end of the 1990s, complaints about excessive waiting times for elective surgeries piled up so that policy makers abolish the fixed hospital budgets at the end of 2000 replacing them by activity-based funding.[63] As a result hospital admissions, treatments and pharmaceutical prescriptions increased rapidly, in particular among the elderly.[11]

Although the impact of smoking did not explain the trend reversal in Dutch mortality it did affect trends in life expectancy considerably. The lower gains in life expectancy for Dutch women as compared to Dutch men throughout the study period were to a large extent caused by smoking, which is in line with the general theory of the smoking transition through which women progress with a delay of several decades as compared to men.[49] It has been noted earlier, that after the exclusion of smoking-related causes of death the stagnation in mortality decline occurred at the same time for men and women.[6] Our study results add that also the resumption of Dutch mortality decline after 2001 was similar for men and women if the differential impact of smoking was accounted for. This accordance of sex-specific life expectancy trends before and after the Dutch trend reversal calls for an more general explanation that affected all Dutch inhabitants in the same way, which we believe to have identified in the form of financing of healthcare. No other plausible alternative explanation that could have affected men and women in the same way has been described in the literature.

Implications

The findings of this study highlight the growing importance of policy decisions towards healthcare resources. The case of the Netherlands demonstrated that life expectancy responds immediately and to a considerably extent to changes in healthcare expenditures.

APPENDIX

A1. Modeling the effect of healthcare expenditures on life expectancy

To estimate the effect of changes healthcare expenditures (HCE) on changes in life expectancy (LE) we have specified a health-production function.[64,65] Therein, monetary investments in health care as input were formally related to changes in health as output.

The starting point of our analysis was how HCE affects LE within a country over time. This allowed to assess whether an annual change in one variable did in fact co-occur with an annual change in the other variable.

Three challenges required particular attention for modeling such a relation. First, the effect is of stochastic nature so that a larger number of observations is necessary to make sure that the co-movement of the variables is not due to random variation only. Simulations have shown that time series with more than 50 years of observations are necessary to achieve stable results.[39] Second, changes in HCE only partly affect changes in LE immediately. The impact of healthcare investments on mortality is likely to have a delayed impact since new policies (e.g. prevention programs) and innovations (new medical technologies) require some time to enfold their full effects.[16] Third, to a certain extend unobserved variables drive the changes in HCE and LE so that a discovered effect might be spurious. This could be the diffusion of new expensive medical innovations (increasing HCE and LE) or a generally a shift in the health status of the population towards less severe and less costly diseases (decreasing HCE and increasing LE).

To solve these three issues the use of panel data is inevitable.[16] Panel data contain information for a group of countries on identical variables available for the same time span. This considerably extents the total number of observations thus resulting in more robust estimates. The time dimension of panel data allows detecting dynamic effects occurring with several years of delay.[66] The cross-sectional dimension of panel data enables to filter out unobserved country-specific effects influencing the relation of interest.[29]

However, using panel data poses additional challenges as the observations over time and countries are not independent from each other. The variables HCE and LE both trend upwards over time so that there is a considerable risk of detecting a link between the two, where actually no link exists.[37] Further, in a highly intertwined glo-

balized world the countries are affected by common processes dependent on the cultural, geographical and economic proximity and similarity.[30] Finally, health system differentials among the countries result in a different ability to transform investments in health care into additional years of life.

To account for these caveats, we used a model specification that is able to deal with various sources of correlation in the data may distorting the estimation of the relation between HCE and LE. Moreover, our model is flexible enough to allow for a heterogeneous health production function among the countries while incorporating at the same time effects of common unobserved or omitted variables.[67] We compared our preferred specification with alternative models that inhibit less flexibility based on model fit and residual diagnostics. Furthermore, we performed extensive sensitivity analysis of our preferred model to ensure the robustness of our estimates.

A2. Description of the data

To estimate the health production function, we use as input healthcare expenditures expressed as proportion of GDP (and alternatively healthcare expenditures in US$ Purchasing Power Parity at 2005 prices) from the OECD health data 2014 and as output life expectancy at birth obtained from the human mortality database.[20,68] Information on per capita gross domestic product at 2005 prices (excluding costs for health care), also from the OECD database, were used as confounder. Overall, our sample contained 19 countries spanning over 30 calendar years, as listed in table A1. We restricted our analysis to the period 1980-2009 because for this period complete information was available for almost any country (except France and Italy), while this time span still provides enough observations to detect dynamic effects. The country-specific means of life expectancy in this time span range from about 76 to 80 years (72 to 77 years in males and 79 to 83 years in females). The countries spend on average 6.9% to 10.2% of their total GDP on health care. Excluding these costs on health care, the time average of the GDP ranges from 16010 US$ in Portugal to 34861 US$ in Norway.

Table A1 Descriptive characteristics of the countries in the panel data on life expectancy, healthcare expenditures (per capita and as proportion of GDP) and GDP (excluding health care costs) and country-specific means for 19 OECD countries 1980-2009

		Human Mortality Database				OECD health data 2014			
		availability	LE total	LE males	LE females	availability	HCE per capita	HCE %GDP	GDP
1	Australia	1980-2009	78.2	75.4	81.1	1980-2009	2144	7.4%	26029
2	Austria	1980-2009	76.7	73.4	79.7	1980-2009	2521	8.8%	25281
3	Belgium	1980-2009	76.7	73.5	79.9	1980-2009	2205	8.0%	24665
4	Canada	1980-2009	78.2	75.3	81.1	1980-2009	2651	9.0%	26386
5	Denmark	1980-2009	76.0	73.4	78.6	1980-2009	2508	8.9%	25388
6	Finland	1980-2009	76.6	72.7	80.3	1980-2009	1878	7.7%	22400
7	France	1980-2009	77.8	73.9	81.6	1990-2009	2804	10.2%	24555
8	Ireland	1980-2009	75.9	73.3	78.7	1980-2009	1770	7.1%	22645
9	Iceland	1980-2009	79.0	76.7	81.3	1980-2009	2359	8.4%	27603
10	Italy	1980-2009	78.1	74.9	81.2	1988-2009	2097	7.9%	24179
11	Japan	1980-2009	80.0	76.7	83.0	1980-2009	1883	7.1%	24367
12	Netherlands	1980-2009	77.7	74.9	80.5	1980-2009	2550	8.6%	26486
13	New Zealand	1980-2008	76.7	74.0	79.4	1980-2009	1525	7.1%	19465
14	Norway	1980-2009	77.8	74.9	80.7	1980-2009	2950	8.1%	34861
15	Portugal	1980-2009	75.6	72.1	79.1	1980-2009	1373	7.5%	16010
16	Spain	1980-2009	78.3	74.8	81.6	1980-2009	1555	7.0%	19987
17	Sweden	1980-2009	78.6	76.0	81.2	1980-2009	2278	8.6%	24030
18	Switzerland	1980-2009	78.8	75.7	81.7	1980-2009	3132	9.2%	30428
19	UK	1980-2009	76.7	74.1	79.2	1980-2009	1812	6.9%	23672
		min	75.6	72.1	78.6	min	1373	6.9%	16010
		max	80.0	76.7	83.0	max	3132	10.2%	34861
		span	4.4	4.6	4.4	span	1759	3.3%	18851

A distinctive feature of healthcare expenditures is the high degree of correlation with GDP[38] and LE that is close to 1 (table A2). Put differently, as a country gets richer it tends to spend more on health care and at the same time people live longer. Due to the high degree of correlation it is hard to disentangle the effect of GDP and HCE on LE and at the same time such multicollinearity potentially inflates the variance in our

regression model. For that purpose we used in our regression HCE expressed as proportion of GDP. For this indicator the correlation with GDP (r=0.66) and LE (r=0.84) is less strong than for HCE expressed in US$ (table A2).

Table A2 Bivariate correlations between the output and input variables in the health production function with country fixed effects, variables in natural logarithm

	LE	GDP	HCE US$	HCE %GDP
LE		0.88	0.95	0.84
GDP			0.93	0.66
HCE US$				0.89

A3. Time series properties

One of the reasons for the high degree of correlation between the variables we aim to put in the health production function is that they all strongly trend upward over time, probably because each variable is also a proxy for general societal progress. In technical terms variables that do not reverse to their mean are non-stationary because they contain a unit root. In such a case, the estimates of classical OLS approaches are subject to the risk of being spurious.[69] To detect the existence of a possible unit-root process in our variables, we tested for non-stationarity in our panel. A flexible test is the CIPS allowing cross-sectional heterogeneity and unbalanced data in the sample.[70] Results of this test are shown in table A3, where up to 4 lags were included to account for serial correlation. The test suggests the existence of a unit root with and without assuming a trend in the series. In particular the series are outcomes of a process integrated of order one, since the null hypothesis of all countries containing a unit-root was not rejected in levels but rejected in differences for most specifications. We performed also a simpler panel unit root that does not account for cross-sectional dependence in the panel as suggested by Maddala & Wu (1999) with virtually the same results.[71]

Table A3 Panel unit-root test for output and input variables in the health production function

	LE		GDP		HCE US$		HCE %GDP	
without trend:	ztbar	p	ztbar	p	ztbar	p	ztbar	p
lags: 0	**-4.4**	0.00	1.8	0.97	-1.1	0.13	0.8	0.79
1	-0.7	0.24	-0.5	0.32	-1.2	0.11	0.3	0.62
2	-1.2	0.12	1.6	0.94	-0.2	0.41	1.5	0.93
3	-1.0	0.16	1.0	0.84	0.1	0.55	2.0	0.98
with trend:								
lags: 0	**-3.6**	0.00	3.9	1.00	1.0	0.83	2.7	1.00
1	0.5	0.68	1.9	0.97	1.3	0.90	2.5	0.99
2	0.4	0.65	4.1	1.00	3.0	1.00	4.1	1.00
3	-0.4	0.36	3.6	1.00	3.9	1.00	4.7	1.00
in differences:								
lags: 0	**-18.3**	0.00	**-8.0**	0.00	**-11.4**	0.00	**-11.1**	0.00
1	**-9.4**	0.00	**-6.2**	0.00	**-6.8**	0.00	**-6.5**	0.00
2	**-4.0**	0.00	-1.5	0.07	**-3.7**	0.00	**-3.0**	0.00
3	**-2.6**	0.01	-0.6	0.26	-1.2	0.11	-0.8	0.21

Note: bold values indicate significant values at p<0.05, thus rejecting the null of non-stationarity

A4. Model building

Estimation with the variables in levels (LEVELS)
Following Baltagi et al 2011 and Skinner and Staiger 2009, we define a Cobb-Douglas production function, where the output is life expectancy at birth (LE) while health-care expenditures (HCE) proxy the bundle of the inputs capital and labor.[30,72]

$$LE_{i,t} = \alpha_i + \delta_t + \beta_1 HCE_{i,t} + \varepsilon_{i,t} \quad (1a)$$

All variables in (1a) are in logs to estimate the elasticity of input and output, but also to account for a decreasing return of marginal investments and to guard the model against the influence of outliers. The subscripts i and t denote country and time, α represents stable differences in medical technology between countries and d the progress of medical technology over time common in all countries. Finally, β_1 represents the percentage change in LE with respect to a percentage change in HCE common in all countries. This specification is denoted as LEVELS since it assumes that a higher level of HCE corresponds to a higher level of LE.

Estimation with the variables in first differences (FD)
Since we have demonstrated in A2 that the variables in our regression are non-stationary in levels but stationary in first differences, we should favor the estimation of a relation between LE and HCE with the variables in first differences, as shown in (1b). This enables to avoid the risk of a spurious correlation in regressions with non-stationary variables.[69]

$$\Delta LE_{i,t} = \delta_t + \beta_1 \Delta HCE_{i,t} + \varepsilon_{i,t} \quad (1b)$$

Dynamic pooled two-way fixed effect model (2FE)
However, estimating the relation between HCE and LE in first differences would remove any long-run relationship between LE and HCE.[73] Since theoretical reasoning above suggested that investments in health care partially also affect mortality with a certain delay, removing long-run effects of HCE on LE would not adequately catch the dynamic impact of changes in health care spending. Therefore, we have decided for an error-correction model, where the long-run relationship of the variables in levels is added to the right-hand side of equation (1b) resulting in equation (2). This is able to measure a dynamic response of LE to changes in HCE divided into two parts.[74] First, changes in HCE could directly initiate changes in LE during the same period. Second,

an increase in HCE may results in a long-term response of LE until the equilibrium relationship between HCE and LE is restored. In difference to other dynamic models where an finite number of lags has to be specified a priori, the error-correction model allows for a flexible response of LE to a change in HCE without a prior specification of the particular lag time merely assuming that the effect declines geometrically over time.[66] Further, since the model disentangles a short-run and long-term relation between the variables in the health production function it is - unlike the classical linear static regression - suited for both stationary and non-stationary data.[66,74]

$$\Delta LE_{i,t} = \alpha_i + \delta_t + \beta_1 \Delta HCE_{i,t} + \gamma LE_{i,t\text{-}1} + \beta_2 HCE_{i,t\text{-}1} + \varepsilon_{i,t} \quad (2)$$

In the error-correction specification as expressed in (2), β_1 tests for the immediate response of LE to a change in HCE during the same year, thus the short-run effect. The second coefficient of HCE β_2 expresses the combined effect on LE during the same year and the next year, while the combination of β_2 and γ represent the long-run effect, computed as β_2 / γ. If γ and β_2 are significantly greater than zero, a long-run relation between the variables exists.[74] Otherwise the model reduces to short-run relation between the changes in LE and HCE only, given that β_1 is significant and greater than zero, which is equivalent to the model estimated with the variables in first differences. Equation (2) contains fixed effects for countries and calendar years and restricts the coefficients of HCE to be the same for all countries. For this reason the model is termed the pooled two-way fixed effects model (2FE).

Dynamic mean-group estimator (MG)

Although the 2FE in (2) allows for a dynamic relationship between HCE and LE it is still a quite restrictive specification, given that it assumes common health technology among all countries, i.e. that a similar investment in health care results in a similar increase in life expectancy. Moreover, the time series of each country were treated as independent from each other. A more realistic but also more complex specification is to assume that each country has not only its own intercept as in (2) but also its own time trend and effect of HCE (and HDP) on LE. For this purpose the relation between the variables and fixed effects is estimated in a first step in each country separately as depicted in (3). In a second step, the coefficients are averaged (3a) and tested for the null hypothesis that the average equals 0, denoted as mean-group estimator (MG).[75]

$$\Delta LE_{i,t} = \alpha_i + \delta_{i,t} + \beta_{1i} \Delta HCE_{i,t} + \gamma_i LE_{i,t\text{-}1} + \beta_{2i} HCE_{i,t\text{-}1} + \varepsilon_{i,t} \quad (3)$$

$$\widehat{\beta_1} = N^{-1} \sum_i \widehat{\beta_{1i}} \quad (4)$$

Dynamic correlated mean-group estimator (CMG)

The mean-group estimator relaxes the assumption of a homogeneous health technology, but still assumes that the health production among the countries operates independently from each other. An intermediate solution between pooling all countries and estimating the regressions completely separately is to introduce common factors representing global shocks and local spillover effects.[41]

$$LE_{i,t} = \beta_{1i} HCE_{i,t} + u_{i,t} \tag{5}$$

$$u_{i,t} = \alpha_i + \lambda_i f_t + \varepsilon_{i,t} \tag{6}$$

$$HCE_{i,t} = c_i + \eta_i f_t + \delta_i g_t + v_{i,t} \tag{7}$$

Compared to (1) the model specifications in (5) and (6) and (7) additionally contain the unobserved common factor *f* correlated with the error term in (5) but also with the explanatory variable HCE in (7), which is additionally driven by a second factor g. The responses to these factors are country-specific, denoted as λ_i, η_i and δ_i.[41] This approach models unobservable or omitted variables that confound the relation between LE and HCE that are common in all countries but have a different impact in each country such as economic recessions or new medical innovations. This framework is in line with earlier findings that the development of life expectancy and of healthcare expenditures is mainly driven by a shared progress in technology.[1,3,72] Controlling for the influence of this should reveal the impact of HCE on LE.

To incorporate the effect of unobserved common factors in the dynamic regression model (3) we follow Pesaran 2006 who demonstrated that the inclusion of cross-sectional averages of all variables in the model is a sufficient proxy for the factors.[29] In the basic specification model (3) is augmented with the cross-sectional averages of all variables, presented in (8). This model is specified as correlated mean-group estimator (CMG)

$$\theta_{0i} \Delta \overline{LE}_t + \theta_{1i} \Delta \overline{HCE}_t + \theta_{2i} \overline{HCE}_{t-1} \tag{8}$$

Dynamic correlated mean-group estimator with distance weights (CMGD)

The standard CMG as explained above assumes that for any country all the other countries in the sample are of equal importance to proxy the unobserved factors influencing LE and HCE. As convincingly shown by Baltagi et al 2012, a more plau-

sible assumption is that closer countries are more relevant for developments in a particular country than more distant countries.[30] This enables to incorporate spatial dependence in the model, like common health policies, regional weather extremes or shared cultural, economic and genetic characteristics among a group of countries. An effective way to incorporate such a proximity in the estimation of the production function is to weight the observations in the countries before constructing the cross-sectional means in (8) by the inverse of the distance between a pair of countries.[30] The construction of the weights is described in detail elsewhere.[40] The distance-weighted version of the CMG will be denoted as CMGD.

Confounders

As mentioned in A2 the effects of HCE and GDP are hard to disentangle. For this reason we add GDP as central confounder to all models since otherwise the effect of changes in HCE would to a certain extent measure changes in GDP.[38,76] The empirical implementation of an additional explanatory variable in the models described above is similar to the inclusion of HCE. We do not add further confounders to our models for two reasons. First, the influence of omitted and unobservable varaibles is indirectly included in our models either as fixed effects or as common factors. Second, thedata quality and availability of other confounders is much worse than for the variables HCE and GDP so that inclusion of them would potentially do more harm than good and reduce the sample size drastically. Nevertheless, in the sensitivity analysis of our preferred model, we will test the model robustness to the inclusion of further variables.

A central assumption of all models introduced in this section is that remaining residuals are white noise. For this purpose we performed test for remaining temporal and spatial correlation by performing the Pesaran CIPS panel-unit root test for the presence of non-stationary and the Pesaran CD-test for cross-sectional independence of the residuals.[70,77] In line with a number of recent contributions in the field of panel econometrics, our goal is to discover and incorporate the sources of violation of the assumptions of OLS regressions rather than correcting away these violations as it was a common practice in the past.[16,31,40] Rather than relying on a single model, we test all specifications explained above and favor the one that is theoretically most plausible but at the same time provides a good fit to the data and well-behaved residuals.

A5. Results

The results of the six model specifications are shown in table A4. In models 1 to 3 the effect of HCE on LE is estimated in the pooled dataset, while in models 4 to 6 the estimators of the country-specific regressions were averaged. The static models 1 and 2 estimate either the immediate or long-run effect, while the dynamic models 3 to 6 contain both aspects and provide the speed until the full impact is visible guided by the coefficient of error correction. The proportion of the total effect that occurs in the first two periods (t=0 and t=1) is shown below the long-run effect. The model fit is expressed as root mean squared error (RMSE) and the results of the tests for stationary and cross-sectional independence of the residuals are displayed at the bottom of the table. Thereby, we report whether the CIPS test with up to 3 lags with and without trends rejects the null of non-stationary of the residuals and display the absolute mean cross-sectional correlation of the residuals in line with the CD test statistic for cross-sectional independence.

The comparison of the models offers interesting insights into the influence of different specifications. We find evidence for a long-run relationship already in the least complex model with variables in levels (column 1 in table A4). An increase of one percent of HCE corresponds to a change in LE by 0.022 percent. The separate regression of the annual changes of HCE and LE in model 2 does not indicate an immediate relationship. The residual diagnostics of the two static models reveal remaining correlation in the temporal and spatial dimension signalling a possible misspecification. Moving to the pooled dynamic model confirms the presence of a long-run effect (elasticity of 0.028) and the absence of an immediate effect. The error correction is estimated to be 0.225, which means that the initial short-run effect of HCE (0.006) diminished by 22.5% every year so that at the end of the second period only a fifths of the total effect occurred. Although the pooled 2FE model achieves a better model fit than the two static models, the residual diagnostics are still unfavourable.

Table A4 Short-run and long-run effect of a change in healthcare expenditures as % of GDP on life expectancy at birth (standard errors in parentheses) in six different specifications

	(1)	(2)	(3)	(4)	(5)	(6)
SPECIFICATION	LEVELS	FD	2FE	MG	CMG	CMGD
	pooled static	pooled static	pooled dynamic	averaged dynamic	averaged dynamic	averaged dynamic
Immediate effect (t=0)		-0.004	0.003	0.005	0.019**	0.010
		(0.005)	(0.004)	(0.006)	(0.007)	(0.006)
Error correction			-0.225***	-0.802***	-0.850***	-0.539***
			(0.027)	(0.073)	(0.078)	(0.089)
Short-run effect(t=0 & t=1)			0.006**	0.014	0.024**	0.020**
			(0.002)	(0.008)	(0.008)	(0.008)
Long-run effect	0.022***		0.028***	0.017	0.028**	0.036**
			(0.008)	(0.010)	(0.009)	(0.012)
proportion of the long-run effect at the end of t=1:			21%	82%	82%	56%
Observations	513	513	513	513	513	513
RMSE	0.0046	0.0032	0.0028	0.0023	0.0016	0.0018
Residuals:						
Stationary	NO	NO	NO	YES	YES	YES
Mean |*p*|	0.424***	0.308***	0.226*	0.221***	0.212**	0.203
CD statistic (*p*)	4.41	17.12	-2.42	10.69	-3.00	0.15

Standard errors in parentheses, *** p<0.001, ** p<0.01, * p<0.05

Note: All models contain life expectancy at birth, per capita healthcare expenditures and per capita GDP (excluding costs for health care). LEVELS and FD estimate pooled static regressions with time and country dummies, 2FE denotes the pooled two-way fixed effects model including country and time dummies, while MG/CMG/CMGD denote each the mean group estimator with no weights/equal weights/distance weights where the model is fit separately fit to any country and then averaged. To preserve an equal number of observations and the full sample size of 19 countries we have not added further lags of the cross-sectional averages in model 6+7.

Allowing for heterogeneous health technology by fitting separate regressions for any country (model 4) solves the problem of remaining non-stationary residuals, reduces the spatial correlation to 0.221 and further improves the model fit to a RMSE of 0.00023. The averaged coefficients of the effects of HCE are not significant anymore. By contrast, as soon as we introduce common factors to the separate regressions (model 5) both the immediate and the long-run effect of HCE becomes highly significant and the model fit improved remarkably to an error of 0.0016. However, the residuals still exhibit spatial dependence. Finally, the model where the relevance of the common factors depend on the geographical distance of a country to the other countries of the sample (model 6) is the only one with favorable residual diagnostics – both for temporal and spatial correlation. This specification results in an effect of HCE of 0.036 in the long-run. About half of this total effect (56%) occurs already in the first two periods at t=0 and t=1 (0.02). The strength of the effect diminishes every year by about 46%, computed as 1-0.539.

Based on these results, we decided to use the coefficients of the more flexible and well-specified CMGD models for computing the contribution of the change in healthcare expenditures on the change in life expectancy at birth in the main analysis of our paper. Given the complexity of the dynamic model, we provide a visualization of its mechanism in figure A1 demonstrating how a single 10 percent change in healthcare expenditures at t=0 increases life expectancy in the subsequent years. During the first year life expectancy at birth (we assume a level of 80 years at t=0) grows immediately by 0.1% or 0.08 years, while this is slightly larger in the next year. Subsequently, the effect size weakens fast and after about 5 years almost the full effect of 0.26% has been reached corresponding to an increase of life expectancy of 0.3 years.

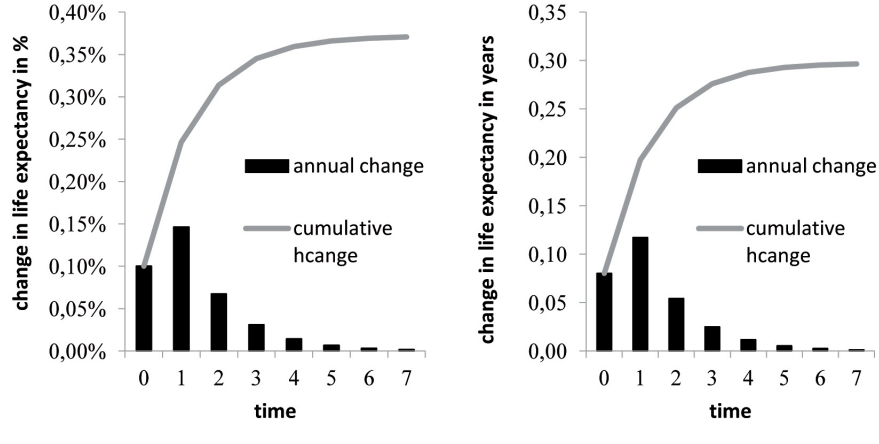


Figure A1 Annual and cumulative change in life expectancy at birth given a 10% change in healthcare expenditures in t=0, based on the dynamic model 6 in table A4

A6. Sensitivity analysis

We have performed extensive sensitivity analyses to assess the robustness of our preferred model specification (CMGD, model 6 in table A4). The results of this model are again shown in column 1 of table A5 compared with the results of eight alternative specifications (model 7-14). In model 7, we have added two additional lags of the cross-sectional averages in equation (8) as generally suggested for smaller samples to ensure the validity of the estimates, which is at the cost of reducing the number of observations from 513 to 441.[78] To check the influence of the selected time span 1980-2009, we have also fitted our preferred model specification to the full time span available in the 2014 OECD health database, shown in model 8. In model 9 we added the variables per capita alcohol consumption and the age-standardized lung cancer death rate, which has been demonstrated to be an excellent proxy for the cumulative effect of smoking.[32] Model 10 estimates the model without taking the natural logarithm of the variables. Further, in model 11 we have excluded the Netherlands to see whether the effect of HCE is also visible without this deviating country. In addition, we have checked the sensitivity of the results with respect to the exclusion of any other country in the sample, shown in figure A2. In model 12 and 13 in table A5 we have used life expectancy of males and females only. In model 13 we have used health care expenditures measures in per capita US$ at 2005 prices and constant purchasing power parities.

In sum, all alternative specifications confirm the existence of a short-run and long-run relationship between LE and HCE in the CMGD specification, while an immediate effect was only visible in 4 of the 8 alternative specifications. The total effect of HCE varied between 0.15 and 0.53 so that our preferred estimate of 0.36 ranges in the middle of these extremes. The proportion of the total effect after two periods ranged between 28% and 91% compared to 56% in our preferred model specification.

Table A5 Short-run and long-run effect of a change in healthcare expenditures as % of GDP on life expectancy at birth in alternative specifications

	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
ALTERNATIVE SPECIFICATION	CMGD averaged dynamic	With 2 additional CS lags	Maximum time span 1960-2013	+ alcohol consumption, lung cancer	Variables not in log	NLD excluded	Male life expectancy	Female life expectancy	HCE in per capita US$
Immediate effect; t=0	0.010	0.042*	0.009*	0.026	0.135**	0.016*	0.013	0.001	0.012
	(0.006)	(0.020)	(0.004)	(0.017)	(0.047)	(0.006)	(0.008)	(0.006)	(0.006)
Error correction	-0.553***	-0.957***	-0.288***	-0.717***	-0.645***	-0.565***	-0.504***	-0.664***	-0.601***
	(0.097)	(0.168)	(0.067)	(0.120)	(0.086)	(0.090)	(0.083)	(0.093)	(0.090)
Short-run effect; t=0 & t=1	0.020**	0.053*	0.015**	0.032*	0.187*	0.020*	0.024*	0.019*	0.016*
	(0.008)	(0.025)	(0.006)	(0.014)	(0.061)	(0.008)	(0.009)	(0.007)	(0.007)
Long-run effect	0.036**	0.058*	0.053**	0.045**	0.029**	0.035**	0.047**	0.028*	0.026**
	(0.012)	(0026)	(0.018)	(0.019)	(0.091)	(0.0123)	(0.015)	(0.010)	(0.011)
proportion of the long-run effect at the end of t=1:	56%	91%	28%	71%	64%	57%	51%	68%	62%
Observations	513	441	702	463	513	485	513	513	513
RMSE	0.0018	0.0010	0.0024	0.0011	0.14	0.0018	0.0022	0.0029	0.0018

Standard errors in parentheses

*** p<0.001, ** p<0.01, * p<0.05

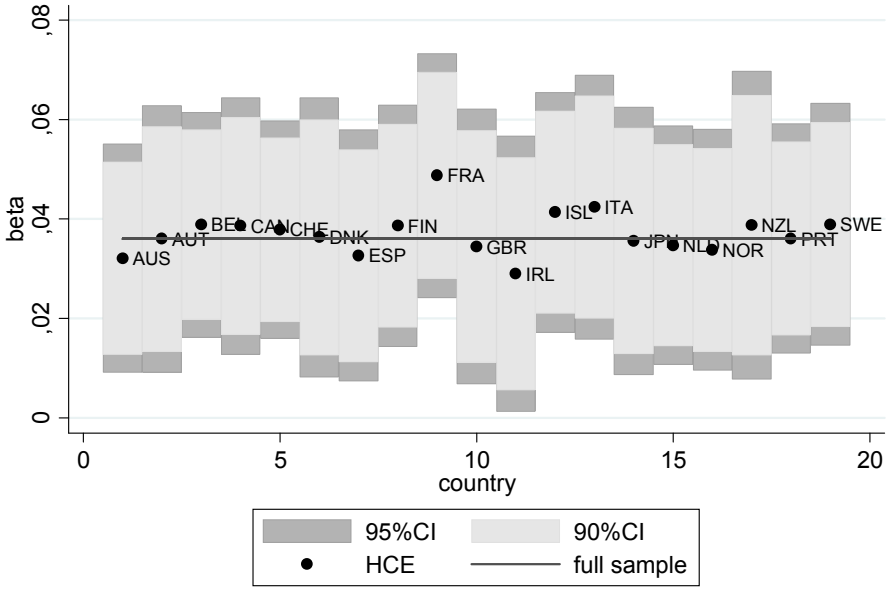


Figure A2 Robustness of the estimated long-run relationship between healthcare expenditures as % of GDP and life expectancy at birth from model 6 in table A4 with respect to the exclusion of a country at a time from the sample

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4

A closer look at the role of health care in the recent mortality decline in the Netherlands – results of a record linkage study

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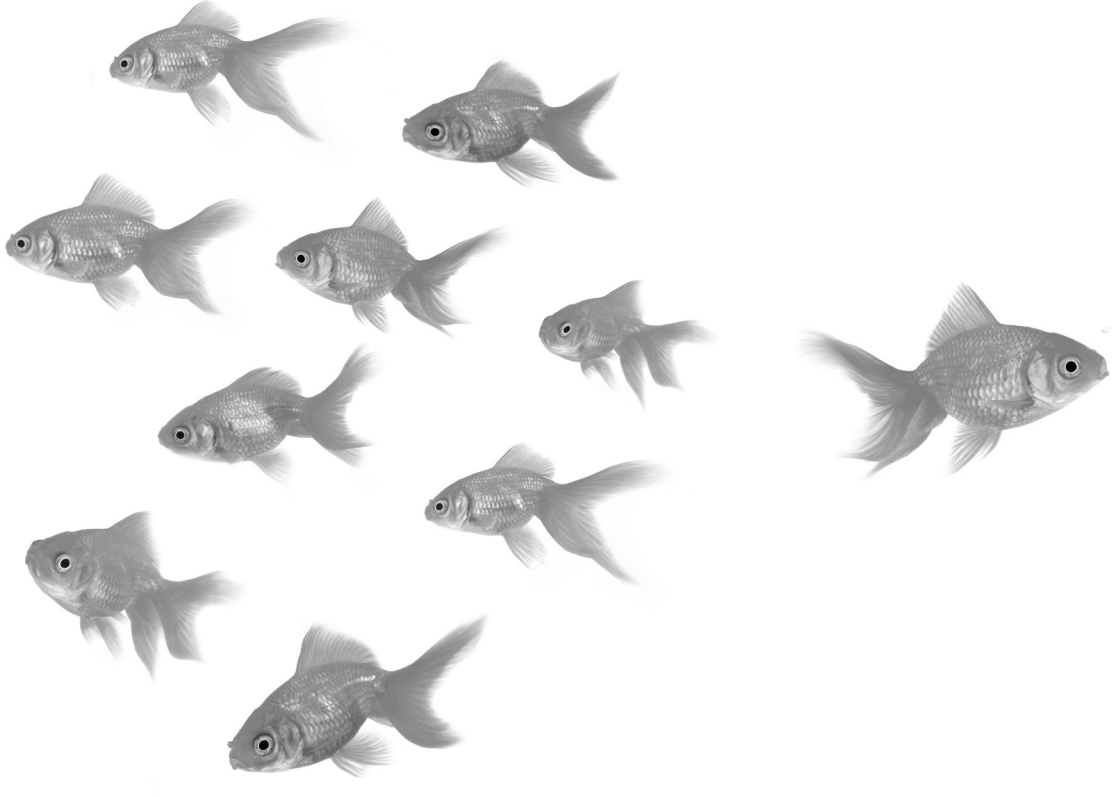

ABSTRACT

Background: Since 2002 Dutch mortality rates decreased rapidly after decades of stagnation. On the basis of indirect evidence, previous research has suggested that this decline was due to a sudden expansion of health care. We tested two corollaries of this hypothesis – first, that the decline was concentrated among those in ill-health, and second, that the decline can statistically accounted for by increases in health care utilization.

Methods: We linked the Dutch health interview survey to the mortality register, and constructed two cohorts consisting of 7691 persons interviewed in 2001/02 and 8362 persons interviewed in 2007/08, each with a five-year mortality follow-up (659 deaths). The change in mortality between both cohorts was computed using semi-parametric Cox proportional hazard models. We compared mortality change between those with and without chronic conditions, and then entered indicators of health care utilization to the models to determine whether these explain the change in mortality.

Results: Between the two study cohorts mortality declined by 15%, and mortality reduction was greatest for those suffering from both fatal and non-fatal conditions (58%). Even after adjustment for health status and risk factors, most indicators of health care utilization were associated with higher instead of lower mortality, and changes in health care utilization did not explain the decline in mortality.

Conclusion: Our results only partly confirm the hypothesis that an expansion of health care explains the recent mortality decline in the Netherland.

INTRODUCTION

Mortality rates at advanced ages improved dramatically during the last decades in most high-income countries.[1] A notable exception is the Netherlands, where mortality rates at older ages stagnated and partly even increased during the 1980s and 1990s.[2-4] Since about 2002, however, Dutch life expectancy has improved rapidly in particular caused by a mortality decline at ages 65 and older.[5]

Coinciding with this unexpected mortality decline a major health care reform was implemented in the Netherlands, in which budget caps on hospital expenditures that had been introduced in 1983 were abolished[6,7], and a fee-for-service financing system was introduced.[8] Although the reform primarily aimed at reducing waiting times for elective surgeries, more money was spent in virtually any area of the health care system including other elements of hospital care and pharmaceutical care.[9,10] While Dutch health care expenditure, expressed as a percentage of GDP, had remained roughly constant at around 8% during the 1980s and 1990s, after the reform it suddenly started to increase and reached a level of 11.8% in 2011 - the second highest level worldwide.[11]

Based on an analysis of routinely collected data at the aggregate level, Mackenbach et al. recently argued that the expansion of health care was the only plausible explanation of the sudden improvement in Dutch life expectancy.[5] While there were no clear changes in the prevalence of risk factors for mortality, utilization of health care services rose disproportionally strong in the elderly, and innovative and effective treatments such as PTCA, cardiac catheterizations and lipid lowering drugs were performed more often and at higher ages, also thanks to changes in guidelines abolishing age-limits.[5] Furthermore, end-of-life decisions involving the withdrawal of further treatments for seriously ill patients were performed less often in these years,[12] possibly indicating a change in attitudes towards life-saving treatments among the elderly.

The simultaneous trend break in the time series of mortality and health care expenditure suggests an association between the two but does not prove a causal relationship. To test the hypothesis that “more health care for the elderly”[5] explains the sudden improvement in Dutch life expectancy after 2001, we examine two specific corollaries of that hypothesis. First, we expect that the mortality decline since 2001 is concentrated among those with more severe chronic conditions, since this group is most sensitive to a lack of provision of medical care and likewise will benefit most from more and better treatment. Second, we expect that in a multivariate analysis increases in health care utilization statistically explain the decline of mortality.

We tested these two predictions using individual-level data, obtained by linking the national health interview survey to the mortality registry. Detailed information on chronic conditions allowed us to stratify the survey respondents by severity of disease, while stability of survey and sampling methodology over time enabled us to estimate changes in mortality, and the contribution of changes in health care utilization to those changes in mortality, after adjustment for sociodemographic factors, behavioral risk factors and various health indicators.

DATA AND METHODS

Data

We used data from the ongoing national Dutch health interview survey (Permanent OnderzoekLeefsituatie=POLS), a random sample of the non-institutionalized Dutch population, with response rates at about 60% and an annual sample size of about 10000 persons. A unique data key allowed to link individual records of this survey to mortality registers. We limited our analysis to adults older than 35 years of age. As upper limit we used age of 85 years at interview to avoid selectivity associated with a higher proportion of people in nursing homes above that threshold. Further we excluded persons with missing information regarding the study variables (in men 25% and 18% in 2001/02 and 2007/08 and in women 24% and 17% in 2002/02 and 2007/08). Sampling weights were used to adjust for selective non-response and to resemble the Dutch non-institutional population.[13]

To assess changes in mortality over time, we pooled the information from the first two surveys that contained the revised health module, 2001 and 2002 (men: N=3697 men; women: N=3994), and the last two surveys, 2007 and 2008 (men: N=3996; women: N=4366). Each survey year was linked to four consecutive years of mortality follow up.

Measures of non-fatal and fatal chronic conditions

To assess the first prediction of our study hypothesis, we identified persons without chronic conditions, only non-fatal condition, only fatal chronic conditions and those suffering both non-fatal and fatal conditions. As non-fatal conditions, we defined reporting (within the previous 12 months) at least one of the following: diabetes, skin disease, eczema, bowel disease, urinary incontinence, arthrosis, rheumatism, back

pain, serious head pain, and disease of neck or arm. As fatal conditions we defined reporting to ever have had cancer, stroke, myocardial infarction, serious heart problems or reporting to have had either peripheral artery disease or serious lung disease during the previous 12 months.

Independent Variables

To assess the second prediction of our study hypothesis, we used various indicators for the volume of health care utilization, measured by the number of visits within the year before the interview at the general practitioner (0, 1 and more), at the medical specialist (0, 1 and more) and at the hospital (0, 1 and more without surgery, 1 and more with surgeries). Further, we assessed the number of prescribed medicine consumed during the recent two weeks (0, 1 and more but no medicine for heart problems, lipid level and hypertension, 1 and more including medicine for heart problems or lipid level or hypertension).

Confounders

Sociodemographic confounders comprised of education (low: primary education only; middle: vocational training or high school; high: university degree) and marital status (married, divorced/widowed, never married). Behavioral risk factors included smoking status (never, ex-smoker, current smoker) and body mass index (underweight for BMI<18.5, normal/overweight for BMI 18.5-30, obese for BMI>30). Health status was measured by the presence of disability (no, yes=major difficulties or only with help at least in one of 6 items: conversation, reading, visual impairment, carrying, walking, bending) and self-rated health (not bad, bad).

Data analysis

We modeled the survival time until death by using a Cox proportional hazards model. We tested the assumption of proportional hazards by including an interaction of all covariates and process time and by estimating Schoenfeld residuals. The process time in all models was age. We estimated pooled models, with gender as strata, and separate models for men and women. To measure differences over time, we coded year at interview 0=interviewed in 2001/2001 and 1=interviewed in 2007/2008. We further included an interaction term of this dummy and the index for chronic

conditions (0=no chronic condition; 1=at least one non-fatal condition and no fatal condition; 2=at least one fatal condition but no non-fatal conditions; 3=at least one non-fatal and at least one fatal conditions). To study the effect of the health care utilization, we first entered sociodemographic and behavioral risk factors and health status indicators.

RESULTS

Changes in sociodemographic characteristics, risk factors, health status and health care utilization

Differences between the two study cohorts are displayed in table 1. Several favorable changes occurred between the survey years 2001/02 and 2007/08. In the second period, there were more people with higher education (+0.4% in men and +4.1% in women) and more never-smokers (+4.2% in men and +3.3% in women). However, there were some unfavorable changes as well, since the share of people with obesity increased (+2.1% in men and +2.2% in women) and the fraction of those without any chronic conditions decreased (-2.8% in men and -3.9% in women).

Overall, the changes in primary and secondary health care utilization were modest, while larger changes occurred in the use of pharmaceutical drugs (table 1). Men more often reported a visit at the general practitioner (GP) (+0.8%) and at the hospital (+1.0%), and the use of prescribed medicines (+6.1%). Women more often reported a visit at a medical specialist (+0.2%) and at the hospital (+0.8%), and like men more often used prescribed medicines (+3.4%). Medication use increased particularly for drugs given for the treatment of heart problems and for lipid lowering and blood pressure lowering drugs (+5% in men and +6.2% in women).

Table 1 Sample characteristics

			males			females		
	Variable	categories	2001/02	2007/08	change	2001/02	2007/08	change
Sociodemographic	age		54.6	55.6	**1.8%**	54.2	55.9	**3.1%**
	marital status	married	81%	76%	**-4.9%**	73%	69%	**-3.8%**
		divorced/widowed	9%	11%	**1.9%**	19%	21%	**1.5%**
		never married	10%	13%	**3.0%**	8%	10%	**2.0%**
	education	low	15%	14%	-0.4%	22%	19%	**-3.1%**
		mid	58%	58%	0.0%	61%	60%	**-1.0%**
		high	28%	28%	0.4%	16%	20%	**4.1%**
Risk Factors	smoking	never	23%	27%	**4.2%**	36%	40%	**3.3%**
		ex-smoker	50%	49%	**-1.0%**	37%	37%	**0.4%**
		current smoker	27%	24%	**-3.2%**	27%	23%	**-3.7%**
	BMI	normal/overweight	89%	87%	**-2.1%**	86%	83%	**-3.4%**
		underweight	1%	1%	**0.1%**	1%	3%	**1.2%**
		obese	10%	12%	**2.1%**	12%	14%	**2.2%**
Health status	OECD disability	yes	17%	17%	0.1%	21%	22%	0.8%
	chronic conditions	none	55%	52%	-2.8%	48%	44%	**-3.9%**
		non-fatal only	25%	26%	0.6%	34%	34%	**0.4%**
		fatal only	16%	16%	0.0%	15%	18%	**2.6%**
		fatal & non-fatal	4%	5%	0.8%	3%	4%	**1.0%**
	Self rated health	bad	12%	11%	-1.2%	15%	14%	-0.4%
Health care utilization	visits GP	0	88%	87%	-0.8%	84%	84%	0.3%
		1+	12%	13%	0.8%	16%	16%	-0.3%
	visits specialist	0	93%	93%	0.4%	93%	92%	-0.3%
		1+	6%	6%	-0.1%	7%	7%	0.2%
	visits hospital	0	93%	92%	-1.0%	93%	92%	-0.8%
		1+ and no surgery	3%	3%	0.5%	3%	3%	-0.1%
		1+ and surgery	4%	5%	0.5%	4%	5%	0.7%
	precribed medicine	0	55%	49%	**-6.1%**	48%	44%	**-3.4%**
		1+ and no hlh	22%	23%	**1.2%**	32%	30%	**-2.8%**
		1+ and hlh	23%	28%	**5.0%**	20%	26%	**6.2%**
		N	4923	4879		5238	5279	
		missing	25%	18%		24%	17%	
		N (without missings)	3697	3996		3994	4366	
		deaths	207	192		124	136	

Note: we performed chi²-tests between each variable and the cohorts, bold values p<0.05 ; hlh=treatment for heart problems or lipid levels or hypertension

Changes in mortality among those with and without severe chronic conditions

Overall, we found a significant mortality decline between both cohorts, which was more pronounced among those with more severe chronic conditions (table 2). In the cohort 2007/08 the mortality risk was 15% lower than in the cohort 2001/02, with a larger decrease among men (-21%) than among women (-5%). However, the 95% confidence intervals of the estimates for men and women overlap to a large degree.

When we conditioned on the presence of chronic conditions, we did not detect significant declines in mortality in the first three categories, but did find mortality declines in the most severe category (table 2). Among those without chronic conditions, or with non-fatal conditions or fatal conditions only, there was no statistically significant decrease in mortality. By contrast, there was a statistically significant decrease of mortality among those suffering one or more non-fatal and one or more fatal conditions simultaneously (-58%). This finding is replicated in the separate estimation for men but not in that for women. Again the confidence intervals between the results for men and women overlap to a large degree so that inferences about gender differentials cannot reliably be made.

Table 2 Reduction in mortality between cohort 2007/08 and cohort 2001/02

	total		**males**		**females**	
subgroup	2007/08 vs 2001/02	95% CI	2007/08 vs 2001/02	95% CI	2007/08 vs 2001/02	95% CI
all	**-0.15**	(-0.29 to -0.02)	**-0.21**	(-0.37 to -0.05)	-0.05	(-0.30 to 0.19)
no chronic conditions	-0.12	(-0.43 to 0.18)	-0.06	(-0.45 to 0.34)	-0.26	(-0.73 to 0.21)
non-fatal conditions	-0.02	(-0.36 to 0.32)	-0.01	(-0.51 to 0.49)	-0.04	(-0.50 to 0.42)
fatal conditions	-0.12	(-0.33 to -0.08)	-0.20	(-0.44 to 0.04)	0.00	(-0.37 to 0.38)
fatal & non-fatal conditions	**-0.58**	(-0.78 to -0.38)	**-0.62**	(-0.83 to -0.41)	-0.32	(-1.06 to 0.41)

Table 3 Effect of health care utilization on mortality (cohort 2001/02 and 2007/08)

		total		**males**		**females**	
variable	categories	HR	95% CI	HR	95% CI	HR	95% CI
gp visits	0	1		1		1	
	1+	0.95	(0.78 to 1.17)	1.03	(0.80 to 1.34)	0.87	(0.62 to 1.20)
specialist vistis	0	1		1		1	
	1+	1.18	(0.94 to 1.49)	1.20	(0.94 to 1.60)	1.13	(0.76 to 1.67)
hospital visits	0	1		1		1	
	1+ and no surgery	1.26	(0.95 to 1.69)	1.23	(0.85 to 1.79)	1.34	(0.84 to 2.15)
	1+ and surgery	1.28	(0.98 to 1.67)	1.12	(0.79 to 1.60)	**1.63**	(1.09 to 2.44)
precribed medicine	0	1		1		1	
	1+ and no hlh	1.29	(0.99 to 1.58)	1.35	(0.96 to 1.90)	1.2	(0.78 to 1.84)
	1+ and hlh	1.25	(0.97 to 1.61)	1.25	(0.91 to 1.72)	1.26	(0.83 to 1.92)

Note: hlh=treatment for heart problems or lipid levels or hypertension; all variables were entered simultaneously in the model and adjusted for chronic conditions, sociodemographic and behavioural risk factors and health status

Contribution of changes in health care utilization to declines in mortality

The simultaneous inclusion of all variables of health care utilization in our model could not detect an association with mortality. Despite adjustments for several risk factors and various health indicators, most hazard ratios were above one indicating an elevated mortality risk with higher utilization of health care (table 3). In the separate model for females, hospital visits with surgery involved were significantly associated with a higher mortality risk (+63%).

Changes in health care utilization did not contribute to the improvement in survival between the cohorts 2001/02 and 2007/08. Shown in table 4 are the directly observed changes in mortality between both cohorts with the inclusion of the period effect only (M0), the changes in mortality after adjustment for sociodemographic characteristics, risk factors and health status (M1), and the changes in mortality after additional adjustment for health care utilization (M2). While adjusting for confounders did not explain the decrease in mortality in the full sample, it did explain 3%-points of the total mortality decrease of 28% in the separate model for males (table 4, column 2). Adjusting for health care utilization had the opposite effect, and made the decline in mortality larger by 1%-point both in the full sample and among men only. In the separate model for women the decrease in mortality was not statistically significant in all three model.

Table 4 Reduction in mortality between cohort 2007/08 and cohort 2001/02 with adjustment for period effects (M0), confounders (M1) and health care utilization (M2)

	total		**males**		**females**	
Model	2007/08 vs 2001/02	95% CI	2007/08 vs 2001/02	95% CI	2007/08 vs 2001/02	95% CI
M0	**-0.15**	(-0.29 to -0.02)	**-0.21**	(-0.37 to -0.05)	-0.05	(-0.30 to 0.19)
M1	**-0.15**	(-0.29 to -0.01)	**-0.18**	(-0.35 to -0.01)	-0.08	(-0.32 to 0.15)
M2	**-0.16**	(-0.30 to -0.03)	**-0.19**	(-0.36 to -0.02)	-0.1	(-0.34 to 0.13)

M0 contains only for period effect (2001/02 vs 2007/08)

M1 additionally controls for chronic conditions, smoking, edcuation, marital status, BMI, OECD disability, self-rated health

M2 additionally controls for GP, specialist, hospital visits and consumption of prescribed medicine

DISCUSSION

We exploited a rich nationally representative health survey with register based mortality follow-up to study the recent decline in Dutch mortality since 2001. Our results only partly confirmed the hypothesis that the expansion of health care explains the mortality decline. While the greatest mortality improvement was indeed found in the subgroup with the most severe chronic conditions, changes in health care utilization could not statistically account for the mortality decline in our study population.

Strengths and limitations

Our study analyzed the association between health care utilization and mortality at the individual level. This opens up the black box of the ecological association reported between trends in life expectancy and health care expenditures at the country level.[5] While ecological studies often control only for a few and quite crude confounders such as GDP and smoking[14], we were able to include detailed information on sociodemographic and behavioral risk factors and different health status indicators. A broad range of chronic conditions allowed stratified analyses by severity of chronic illness.

Although we have used a large nationally representative survey, selection bias due to the non-response in POLS (about 40%) and exclusion of the institutionalized population might be an issue. We further excluded about 20% of the sample due to missing information for at least one of the variables in the model. In general, those not responding in a survey and people living in institutions have a less favourable health status.[15,16] In our study, the survey response rate was about 4%-points higher in the cohort 2007/08 than in the cohort 2001/02, and about 7%-points fewer cases were excluded due to missing information, while the proportion of the institutionalized population was lower.[17,18] In sum these changes may have led to a slightly more healthy sample composition in the cohort 2007/08. Even if this would have affected the estimated mortality decline for the full sample, it unlikely affects the results that the more chronically ill had the greatest improvement in survival. The latter finding was also found in another Dutch sample that included people in institutions.[17]

Our study builds on self-reported information on health status and chronic conditions. Previous research concluded that self-reported information on chronic conditions was fairly accurate.[19] Self-rated health is generally considered a reasonable proxy for the objective health status.[20] Inconsistencies in self-reported health over time have been reported, but this referred to a much longer time span than in our

study and was found to be relevant mainly among younger people.[21] Nevertheless, if non-differential misclassification of health conditions has occurred, our estimates of the effect of health conditions on mortality will be downwardly biased, and control for health status in our analysis of the effect of health care utilization on mortality may have been incomplete (see below).

This study did not really capture the time before the introduction of health care reform, since the latter started in 2001 and we used data from 2001 onwards. Hence, we may have missed some of the effects of the expansion of health care. However, health care spending and utilization did continue to rise at least until 2011[11,22] so that our analysis still compares a period with more utilization to a period with less utilization. In a sensitivity analysis, we compared our study cohort 2007/08 with a cohort constructed from two earlier POLS survey years, 1997/98, in which identical information had been collected for health care utilization, but not for all of the other variables. The results of this analysis confirmed our second main finding: there is a significant positive association between health care use and mortality, and changes in health care use did not contribute to the explanation of mortality decline (see table A2 and A3 in the appendix). This was also true after including each indicator of health care utilization separately in the models and after including an interaction of study cohort and health care utilization (see table A4 and A5 in the appendix).

Interpretation

We found that mortality decline was concentrated among those with chronic conditions, as predicted, but could not establish a beneficial effect of health care utilization on mortality. In fact, we rather found the opposite: a higher risk to die among those who had used more care. Empirical findings suggesting that health care seem to do more harm than good have been reported before, and have been described either as an "anomaly"[23] or as "the paradox of health care".[24-26] These paradoxical results have usually been ascribed to imperfect control of confounding,[23,27] and we believe that this also applies to our analysis. Although we controlled for sociodemographic characteristics, risk factors and various aspects of health status, we have probably not sufficiently adjusted for the nature of the health conditions and the severity of illness, partly because of the self-reported nature of our data. Likewise, the indicators of health care utilization in our analysis are quite crude, and more detailed information on hospital treatments or pharmaceutical drugs would have been preferable. Moreover, our analysis contained data on health care utilization in the years prior to the interview only, which could lead to reverse causation if health status as

reported during the interview is partly determined by prior treatment. Additionally, since the institutionalized population was not included, we missed the most severe ill persons in our analysis.[28] Due to all these issues we believe that our results cannot be interpreted as a refutation of the idea that health care saves lives, or, for that matter, of the hypothesis that increases in health care utilization have contributed to the recent declines in mortality in the Netherlands.

While we found that mortality decline was concentrated among those with chronic conditions, we also found an increase in the prevalence of chronic conditions between the 2001/02 and 2007/08 cohorts. This increase in prevalence is likely to be the result of earlier and better detection of diseases, perhaps as a consequence of the same changes in health care that contributed to the decline of mortality. The change in hospital financing from fixed budgets to a fee-for-service scheme in 2001 created incentives to admit more persons for milder and less specific symptoms.[10] Diagnostic procedures involving brain CT and MRI were applied more often after 2001, which presumably led to an increase in the incidence of conditions like ischemic stroke.[29] This also suggests that in our analysis the category of fatal and non-fatal chronic conditions may have contained milder cases on average in 2007/08 than in 2001/02. We therefore reran our analysis of mortality decline among those with and without chronic conditions (as presented in table 2) with additional controls for self-perceived health and OECD disability (results not shown). However, the results were robust to this adjustment for health status.

In our study sample the biggest changes in health care utilization occurred in pharmaceutical care with particularly because more people took drugs for heart problems, lipid levels and hypertension. This is in line with other studies arguing that the recent improvement in Dutch survival was mainly due to better (pharmaceutical) care for cardiovascular diseases.[5,30] Between 2000 and 2003 the utilization of statins increased by 70% in the Netherlands.[31] During this time there were several guideline changes in the prescription of these lipid-lowering agents, effectively increasing the prescription rates for older and sicker patients.[32] Unlike previous reports we find only modest changes in hospital visits.[5] We believe that this is due to the fact that our sample includes only persons who were able to participate in the survey, and has missed persons with more severe diseases who are more dependent on hospital care.

Implications

The jury on the explanation of the recent mortality decline in the Netherlands is still out. Our study confirms that this decline cannot be explained by changes in sociodemographic characteristics, risk factors or health status, as has been argued before[5], but has not been able to demonstrate that it can be explained by the increase of health care utilization. Further studies are needed, taking a more detailed look at specific treatments for specific patient groups, perhaps on the basis of linkages between GP data, hospital data, health insurance data, and mortality.

APPENDIX

Table A1 Sample characteristics (cohort 1997/98 and 2007/08)

			males			females		
	Variable	categories	1997/98	2007/08	change	1997/98	2007/08	change
Socio-demographic	age		53.5	55.6	**3.9%**	53	55.9	**5.5%**
	marital status	married	82%	76%	**-6.1%**	74%	69%	**-4.6%**
		divorced/widowed	9%	11%	**2.3%**	18%	21%	**2.4%**
		never married	9%	13%	**3.8%**	8%	10%	**2.0%**
	education	low	18%	14%	**-3.1%**	25%	19%	**-5.3%**
		mid	58%	58%	**-0.5%**	60%	60%	**0.3%**
		high	24%	28%	**3.6%**	15%	20%	**5.0%**
Risk Factors	smoking	never	20%	27%	**7.3%**	36%	40%	**4.1%**
		ex-smoker	44%	49%	**5.6%**	34%	37%	**3.0%**
		current smoker	37%	24%	**-12.9%**	30%	23%	**-7.1%**
	BMI	normal/overweight	92%	87%	**-4.6%**	88%	83%	**-4.9%**
		underweight	1%	1%	**0.2%**	2%	3%	**0.9%**
		obese	8%	12%	**4.4%**	10%	14%	**4.0%**
Health status	OECD disability	yes	8%	12%	**4.6%**	12%	16%	**4.1%**
	chronic conditions	none	NA	52%		NA	44%	
		non-fatal only	NA	26%		NA	34%	
		fatal only	NA	16%		NA	18%	
		fatal & non-fatal	NA	5%		NA	4%	
	Self rated health	bad	8%	11%	**3.0%**	11%	14%	**3.2%**
Health care utilization	visits GP	0	88%	87%	-0.8%	83%	84%	0.6%
		1	12%	13%	0.8%	17%	16%	-0.6%
	visits specialist	0	93%	93%	-0.2%	93%	92%	-0.6%
		1	7%	7%	0.2%	7%	8%	0.6%
	visits hospital	0	93%	92%	-0.7%	93%	92%	-1.3%
		1+ and no surgery	3%	3%	0.5%	2%	3%	0.3%
		1+ and surgery	4%	5%	0.2%	4%	5%	0.9%
	precribed medicine	0	59%	49%	**-10.4%**	53%	44%	**-8.4%**
		1+ and no cardio	21%	23%	**1.8%**	30%	30%	**-0.4%**
		1+ and cardio	19%	28%	**8.6%**	17%	26%	**8.9%**
		N	4811	4879		5121	5279	
		missing	20%	18%		21%	17%	
		N (without missings)	3862	3996		4031	4366	
		deaths	234	201		144	146	

Note: we performed chi²-tests between each variable and the cohorts, bold values p<0.05 ; hlh=treatment for heart problems or lipid levels or hypertension

Table A2 Effect of health care utilization on mortality (cohort 1997/98 and 2007/08)

		total		males		females	
variable	categories	HR	95% CI	HR	95% CI	HR	95% CI
gp visits	0	1		1		1	
	1+	0.98	(0.81 to 1.20)	1.21	(0.94 to 1.55)	0.71	(0.5 to 1.0)
specialist vistis	0	1		1		1	
	1+	1.06	(0.83 to 1.35)	0.94	(0.69 to 1.29)	1.28	(0.87 to 1.89)
hospital visits	0	1		1		1	
	1+ and no surgery	1.34	(0.99 to 1.80)	1.26	(0.86 to 1.85)	1.5	(0.92 to 2.45)
	1+ and surgery	**1.41**	(1.08 to 1.83)	1.23	(0.87 to 1.74)	**1.74**	(1.16 to 2.6)
precribed medicine	0	1		1		1	
	1+ and no hlh	1.24	(0.96 to 1.60)	1.29	(0.94 to 1.78)	1.19	(0.79 to 1.81)
	1+ and hlh	**1.4**	(1.11 to 1.77)	1.33	(0.99 to 1.79)	**1.51**	(1.03 to 2.22)

Note: hlh=treatment for heart problems or lipid levels or hypertension; all variables were entered simultaneously in the model and adjusted for sociodemographic and behavioural risk factors and health status

Table A3 Reduction in mortality between cohort 2007/08 and cohort 2001/02 with adjustment for period effects (M0), confounders (M1) and health care utilization (M2)

	total		males		females	
Model	2007/08 vs 1997/98	95% CI	2007/08 vs 1997/98	95% CI	2007/08 vs 1997/98	95% CI
M0	**-0.22**	(-0.34 to -0.09)	**-0.23**	(-0.39 to -0.07)	**-0.19**	(-0.40 to -0.02)
M1	**-0.28**	(-0.40 to -0.16)	**-0.29**	(-0.44 to -0.13)	**-0.28**	(-0.47 to -0.09)
M2	**-0.3**	(-0.42 to -0.19)	**-0.3**	(-0.45 to -0.15)	**-0.31**	(-0.50 to -0.13)

M0 contains only for period effect (2001/02 vs 2007/08)

M1 additionally controls for smoking, edcuation, marital status, BMI, OECD disability, self-rated health

M2 additionally controls for GP, specialist, hospital visits and consumption of prescribed medicine

Table A4 Effect of health care utilization on mortality (2001/02 vs 2007/08)

2001/02 vs 2007/08		separate inclusion		separate inclusion & interaction effect				
		total		total		total		p-value of interaction
variable	categories	HR	95% CI	HR 2001/02	95% CI	HR 2007/08	95% CI	
gp visits	0	1		1		1		
	1+	0.99	(0.81 to 1.22)	0.89	(0.67 to 1.19)	1.10	(0.80 to 1.41)	0.296
specialist vistis	0	1		1		1		
	1+	1.23	(0.98 to 1.55)	1.22	(0.88 to 1.68)	1.24	(0.85 to 1.64)	0.926
hospital visits	0	1		1		1		
	1+ and no surgery	1.3	(0.97 to 1.73)	1.44	(0.97 to 2.12)	1.17	(0.69 to 1.65)	0.465
	1+ and surgery	**1.32**	(1.01 to 1.71)	1.13	(0.75 to 1.70)	1.48	(0.97 to 1.98)	0.326
precribed medicine	0	1		1		1		
	1+ and no hlh	**1.31**	(1.00 to 1.70)	1.37	(0.97 to 1.96)	1.23	(0.76 to 1.70)	0.665
	1+ and hlh	1.27	(0.99 to 1.63)	1.31	(0.94 to 1.81)	1.22	(00.79 to 1.65)	0.774

hlh=treatment for heart problems or lipid levels or hypertension; all variables were entered simultaneously in the model and adjusted for chronic conditions, sociodemographic and behavioural risk factors and health status

Table A5 Effect of variables of health care utilization on mortality separately included in the pooled sample and allowing for the interaction with period effects (1997/98 vs 2007/08) and adjusted for confounders

1997/98 vs 2007/08		separate inclusion		separate inclusion & interaction effect				
		total		**total**		**total**		p-value of interaction
variable	categories	HR	95% CI	HR 1997/98	95% CI	HR 2007/08	95% CI	
gp visits	0	1		1		1		
	1+	1.03	(0.85 to 1.26)	0.97	(0.74 to 1.29)	1.09	(0.79 to 1.40)	0.546
specialist vistis	0	1		1		1		
	1+	1.13	(0.89 to 1.44)	1	(0.69 to 1.43)	1.26	(0.86 to 1.66)	0.34
hospital visits	0	1		1		1		
	1+ and no surgery	**1.39**	(1.03 to 1.88)	**1.59**	(1.04 to 2.43)	1.25	(0.74 to 1.76)	0.422
	1+ and surgery	**1.47**	(1.13 to 1.91)	1.25	(0.83 to 1.87)	**1.66**	(1.10 to 2.22)	0.289
precribed medicine	0	1		1		1		
	1+ and no hlh	1.26	(0.98 to 1.62)	1.22	(0.87 to 1.70)	1.29	(0.81 to 1.78)	0.817
	1+ and hlh	**1.45**	(1.15 to 1.83)	**1.53**	(1.14 to 2.06)	1.37	(0.90 to 1.84)	0.617

all models controled for education, marital status, smoking, edcuation, BMI, OECD disability, self-rated health

hlh=treatment for heart problems or lipid levels or hypertension; all variables were entered separately in the model with and without interaction of them with period and adjusted for sociodemographic and behavioural risk factors and health status

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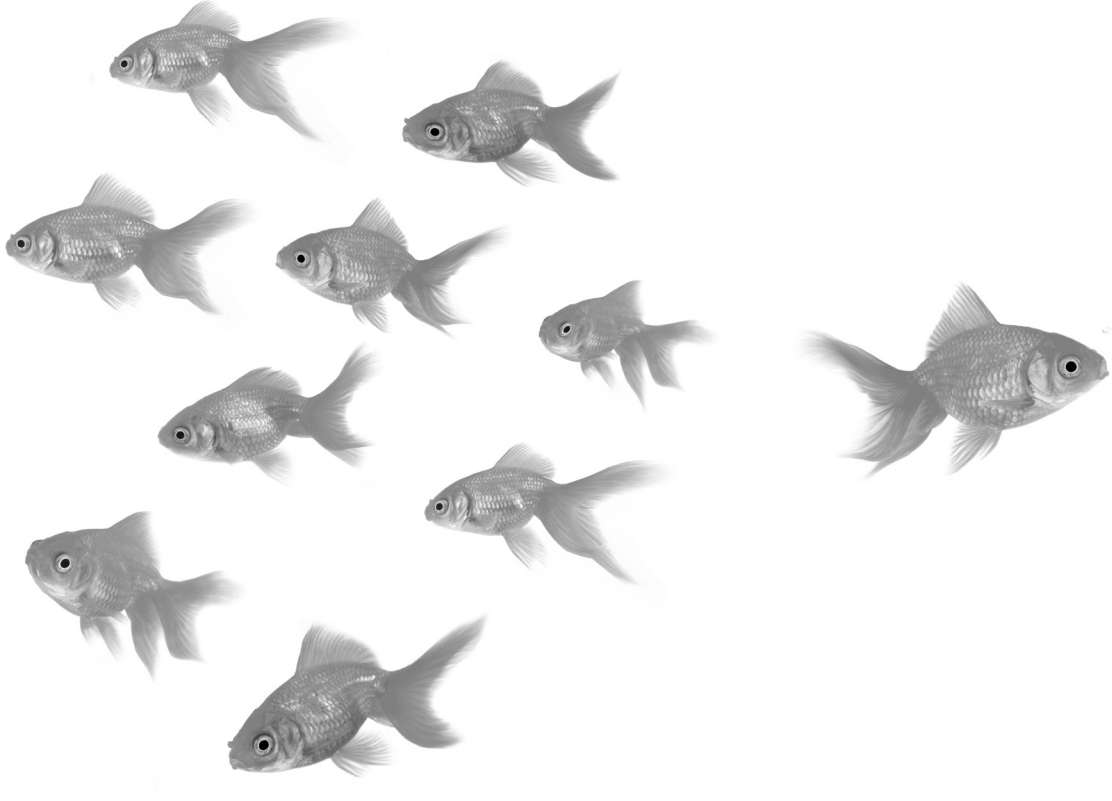
Part III

Projection



5

Does the impact of the tobacco epidemic explain structural changes in the decline of mortality?

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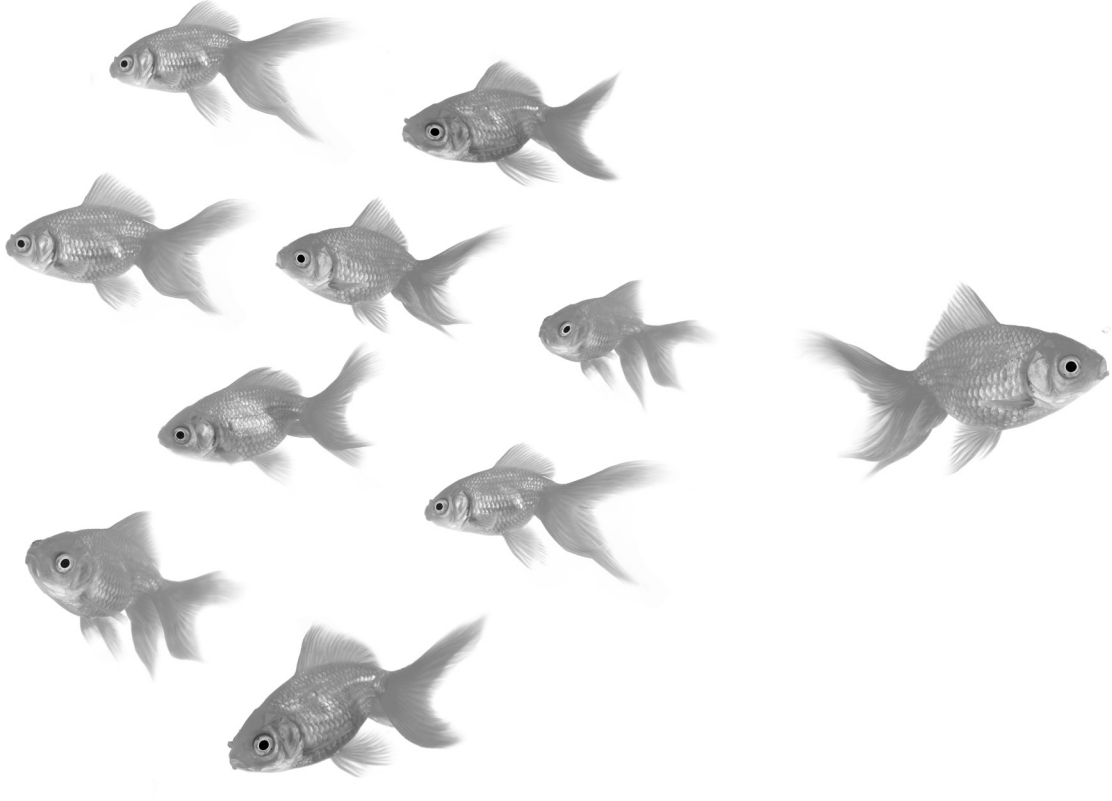
Submitted



ABSTRACT

Background: Extrapolative approaches such as the Lee-Carter model dominate the current literature and practise on mortality forecasting. One of the main reasons for this dominance is the steady long-run mortality decline in most countries since about 1950 that is believed to persist into the future. However, a series of papers recently demonstrated significant structural changes in the rate of mortality decline, mostly occurring in the male subpopulations during the 1970s and 1980s. We hypothesize that these deviations from linearity are related to the progression of the tobacco epidemic that mainly influenced male mortality during this period. If true, removing the distorting impact of smoking will recover the steady long-run trend in mortality decline that could then be extrapolated into the future more robustly. The aim of our paper is to assess to what extent the impact of the tobacco epidemic indeed explains the detected structural changes in mortality decline.

Methods: We fitted a Lee-Carter model to all-cause mortality rates and to smoking-free mortality rates in 20 developed countries in the age range 0 to 99 years between 1950 and 2009 and assessed the presence of structural breaks in the Lee-Carter mortality index. Thereby, the impact of smoking on mortality rates was removed estimated applying the indirect approach developed by Preston, Glei and Wilmoth (2011) to attribute mortality to smoking.

Results: We detected significant structural changes in the mortality decline for 16 countries in males (80%) and 2 countries in females (10%). Removing smoking-associated distortions from the mortality trends structural changes were left in 10 countries in males (50%) and in 3 countries in females (15%).

Conclusion: We only partly confirm the hypothesis that the impact of smoking was responsible for structural changes in the decline of mortality. For the countries where smoking had an impact on structural changes robust long-term projections based on smoking-free mortality rates are recommended. For the other countries, additional factors should be taken into account, such as healthcare policies and innovations in medical treatments before carrying out a linear extrapolation of past trends.

INTRODUCTION

The prediction of future mortality and life expectancy is a central challenge of the actuarial profession.[1,2] Official projections and expert appraisals did not foresee the huge improvements in life expectancy occurring during the past century and recurrently underestimated the potential for further mortality decline.[3-6] This failure of providing reliable projections of mortality rates jeopardizes future pension plans, life annuities and the public provision of health insurance.[1,7,8]

In response to the recurrent failure to accurately projecting mortality trends, new models were proposed and adapted in the recent years.[9-12] These new approaches have in common that they extrapolated mortality trends by only using past series of mortality rates as input in their model.[9,13] This displaced earlier approaches that incorporated also external information such as biological reasoning, expert knowledge and information on causes-of-death that turned out to make projections often worse than better.[14,15]

One of the most prominent extrapolation approach is the Lee Carter model and its variants often referred as "gold standard" or "benchmark".[13,16,17] Aside from the model parsimony and the intuitive interpretation of its parameters, the major reason for its success is its congruence with historic trends. In their seminal paper Lee and Carter have demonstrated that the central mortality time trend in the US between 1900 and 1989 could best be described by a simple straight line, which provides a solid basis for further linear extrapolation of the mortality decline.[13] This remarkable regularity of the decline in mortality rates has been confirmed for many other high-income countries so that linear extrapolation became the leading paradigm of mortality projections.[9,18-20]

However, past mortality trends were not linear in all countries and subpopulations. A growing list of countries exhibited more irregular mortality trends in the past decades such as the Netherlands, Norway, Denmark, Australia and the United States.[21-25] Recently, a series of papers even reported the presence of significant structural breaks in the time index of the Lee-Carter model in virtually almost any high-income country.[26-28] Thereby, most of the breaks occurred in the males and during the 1970s and 1980s.[26-29] In the presence of such nonlinearities, past trends do not provide a solid basis for extrapolation and the resulting projections become much more volatile and particularly sensitive towards the selection of the historical period.[30,31]

A likely explanation for the presence of structural breaks in mortality trends is the distorting impact of the progression of the tobacco epidemic since its impact strongly affected male mortality during the 1970s and 1980s.[32,33] Thereby, the impact of the tobacco epidemic on mortality trends exhibited a regular bell-shaped pattern following the trend in smoking prevalence with a time lag of about 20-30 years mainly due to the delayed effect of smoking on lung-cancer.[33,34] Hence, the slowing down and acceleration in the pace of mortality decline might be a result of the different stages of progression of the impact of the tobacco epidemic on mortality.[34] Adjusting for the distorting effects of smoking, trends in male life expectancy were more linear over time, more similar among the countries and closer to the already much more linear trends of females.[31,35] For this reason it has been recommended to base long-run forecasts of mortality on smoking-free mortality rates complemented by a separate projection of smoking-associated mortality.[32,35]

So far projection models in the actuarial field largely ignored the factor smoking despite its dramatic impact on mortality trends. The overview and quantitative comparison of actuarial models in Cairns et al. (2009), Dowd (2010) and Shang et al. (2011) does not cover the topic of smoking at all.[11,36,37] However, a recent explorative study of Kleinow and Cairns (2013) on the link between smoking prevalence and mortality is a first sign of the awareness of this factor.[38] To assess whether it will be worth to put more emphasize on the factor smoking, this paper tests whether statistically significant structural breaks in the mortality decline in high-income countries were indeed mainly caused by the impact of smoking. The paper is organized as follows. In section 2 we introduce data and methods used to detect structural changes with and without the impact of smoking on mortality trends. Section 3 presents the results of our study that were finally discussed in section 4.

DATA AND METHODS

Data

We included 20 OECD countries over the period 1950-2009 in our analysis: Australia, Austria, Belgium, Canada, Denmark, Spain, Finland, France, Scotland, England & Wales, Northern Ireland, Italy, Japan, the Netherlands, Norway, Portugal, Sweden, Switzerland and the USA. We selected these countries because reliable information on smoking and mortality could be obtained for almost the full time span from harmonized sources. We did not include Eastern European countries because there structural changes likely occurred in response to the fall of the iron curtain around

1990 and also because the data for these countries were not fully available or reliable for the full time span. We analysed the different parts of the United Kingdom separately because previous studies documented large spatial variation among the UK regions with respect to mortality trends.[39]

For deriving mortality rates and constructing life tables we used sex- and age-specific population and death counts from the Human Mortality Database in the age groups (0, 1-4, 5-9, …, 95-99).[40] Further, we obtained sex- and age-specific lung-cancer death rates for the age groups (35-40, …, 80-84) from the WHO mortality database on causes of death as input for the indirect estimation of smoking-attributable mortality.[41] We did not account for the impact of smoking above age 85, because of the larger uncertainty of information on causes-of-death at higher ages.[42]

Fitting the LC model

For extracting a central time trend of mortality, we applied the LC model as expressed in equation (1).[13] In this model, the average log mortality rate at each age $\alpha(x)$ is separated from the central time trend $\kappa(t)$ while allowing for slower and faster rates of decline at every age through the interaction term $\beta(x)$

$$\log m(x,t) = \alpha(x) + \beta(x)\kappa(t) + \varepsilon(x,t) \quad (1)$$

To fit the LC model introduced in (1), we followed Brouhns et al. (2002) who assumed that deaths were drawn from a Poisson distribution with person-years lived as offset and estimated the LC-parameters via maximum likelihood.[43] This provided a more realistic assumption for the variance in death rates that is known to increase at higher ages.[1] To achieve a unique solution of model (1) the following restrictions were made, which is in line with earlier studies [11]:

$$\sum_t \kappa(t) = 0 \quad (2)$$

and

$$\sum_x \beta(x) = 1. \quad (3)$$

The model was fitted in *R* using the lifemetrics package (available at: http://www.macs.hw.ac.uk/~andrewc/lifemetrics/).

Estimating the impact of smoking on mortality

We applied the indirect approach suggested by Preston, Glei and Wilmoth (PGW) (2010) to estimate the fraction of mortality attributable to smoking.[44] Here, the basic idea is that the total cumulative damage of past smoking on all causes of death could be indirectly inferred from observed lung-cancer mortality rates. Defining smoking as the only source of variation in lung-cancer rates [M_L], the intensity of smoking is computed as difference between the observed M_L and the M_L among never smokers at the same age and sex obtained from the Cancer Prevention Study II. To obtain the fraction attributable to smoking for causes other than lung cancer [M_0], the computed intensity of smoking is multiplied by a sex-, age- and time-specific translation factors.[44] These factors were obtained by regressing M_L on M_0 for a group of high-income countries between in 1950-2006. Since the original approach provided translation factors only above the age of 50, we also used the additional factors computed by Martikainen et al. (2014) to estimate smoking- attributable mortality between age 35 and 50.[45] Additionally, for a few calendar years where no information on lung cancer was available we linearly interpolated or extrapolated the attributable fractions transformed by the natural logarithm beforehand. Having estimated the fraction of smoking-associated mortality, we removed this fraction from the observed mortality rates and refitted the LC model.

Detecting and dating structural changes

For detecting structural changes in the decline of mortality, we first extracted the mortality time index $\kappa(t)$ from the LC model in equation (2) and computed the first differences of the series.

$$\Delta k(t) = \kappa(t) - \kappa(t\text{-}1) \quad (4)$$

This was motivated by previous research demonstrating the time series of the time index of the LC model was generally integrated of order one.[27] We replicated the tests for unit roots proposed by Coelho and Nunes (2011) in our sample and detected a unit root in 95% of the time series confirming our assumption. Next, we tested whether the change in these differences over time was constant by fitting the following two nested models via OLS.

$$\Delta k(t) = \alpha + \varepsilon(t) \quad (5)$$

$$\Delta k(t) = \alpha + \beta DU(t) + \varepsilon(t) \quad (6)$$

While the null model in equation (5) models the change in $\kappa(t)$ as constant mean α, parameter β in the alternative model expressed in equation (6) measures a slower or faster pace of change for a segment of the time series, expressed by the dummy variable $DU(t)$ that is 0 below an assumed breakpoint and 1 above. In total, the alternative model was fitted 59 times for all possible breakpoints between t=1956 and t=2004. The trimming of the first and last five observations in the observation period is a common approach to avoid misleading breaks that are located too close at the boundaries of the data.[46] The alternative model with the best evidence against the null model was selected based on likelihood ratio tests. If the tests surpassed the significance level of 5% we extracted the breakpoint of the alternative model, while otherwise the null model without any break was accepted.

RESULTS

In males, structural changes were detected in mortality trends of 16 out of 20 countries representing 80% the sample (table 1a). After adjustment for the impact of smoking on mortality trends, structural changes were still present in 10 countries thus 50% of all cases. Thus, the impact of smoking explained the presence of structural changes in 6 countries (30% of the sample), namely Belgium, Switzerland, Northern Ireland, Scotland, England & Wales and Italy. In the remaining countries the timing of the detected structural changes was generally robust to the adjustment for the impact of smoking, expect for the Netherlands where the break point was dated at a later point in time (2003 instead of 1993) and Portugal where the break point was dated at an earlier point in time (1971 instead of 1996). Generally, the magnitude of the changes was smaller after adjusting for the impact of smoking. The magnitude of the structural changes was largest in Denmark, Ireland and the Netherlands where the slope of the change in mortality trends accelerated by 0.7 units in each country. After adjustment for the impact of smoking this magnitude was stable in the Netherlands but slightly smaller in Denmark and Ireland.

In females significant changes were detected only in 2 countries representing 10% of the sample (table 1b). After adjustment for the impact of smoking on mortality trends, structural changes were still present in 3 countries thus 15% of all cases. Thus, the impact of smoking did not explain the presence of structural changes in females. By contrast, the impact of smoking had a supressing effect in Australia, where the break point was not visible before adjusting for this factor. The magnitude of the detected changes varied in size and direction with a slowing down of the change in mortality in Japan in 1958 and an acceleration of the change in mortality in the

Netherlands and Australia. Interestingly, the magnitude and timing of the structural change in the Netherlands and in Australia was virtually similar among males and females in the respective country after adjusting for the impact of smoking.

Table 1a Structural changes in series of the time index of a fitted LC model and the changes of the slope of the mortality decline before and after removing smoking-associated mortality, males

Males	**Before adjustment for smoking**				**After adjustment for smoking**			
Country	**Break date**	**χ^2 Statistic**	**P value**	**Change in slope**	**Break date**	**χ^2 Statistic**	**P value**	**Change in slope**
Australia	1970	12.4	0.000	-0.5	1970	7.6	0.006	-0.4
Austria	1983	11.3	0.001	-0.4	1983	6.2	0.013	-0.3
Belgium	1976	6.7	0.010	-0.3	-	1.7	0.197	-
Canada	1975	17.8	0.000	-0.3	1975	8.4	0.004	-0.2
Switzerland	1990	6.9	0.009	-0.4	-	3.1	0.076	-
Denmark	1995	18.4	0.000	-0.7	1995	6.9	0.009	-0.4
Spain	-	3.0	0.081	-	-	0.9	0.348	-
Finland	-	3.0	0.085	-	-	2.0	0.162	-
France	-	3.2	0.072	-	-	2.1	0.149	-
Northern Ireland	1980	7.3	0.007	-0.5	-	3.2	0.075	-
Scotland	1993	9.0	0.003	-0.5	-	2.9	0.090	-
England &Wales	1979	9.1	0.003	-0.4	-	2.9	0.089	-
Ireland	1999	7.8	0.005	-0.7	1999	6.0	0.014	-0.6
Italy	1983	12.3	0.001	-0.5	-	3.7	0.055	-
Japan	-	3.0	0.082	-	-	3.7	0.056	-
Netherlands	1993	19.3	0.000	-0.7	2003	8.9	0.003	-0.7
Norway	1990	19.4	0.000	-0.6	1990	16.0	0.000	-0.6
Portugal	1996	6.0	0.014	-0.5	1971	4.9	0.026	-0.4
Sweden	1988	11.9	0.001	-0.5	1988	8.7	0.003	-0.4
USA	1968	14.2	0.000	-0.3	1968	6.0	0.015	-0.2
% of cases with break points	**80%**				**50%**			

Table 1b Structural changes in series of the time index of a fitted LC model and the changes of the slope of the mortality decline before and after removing smoking-associated mortality, females

Females	Before adjustment for smoking				After adjustment for smoking			
Country	Break date	χ^2 Statistic	P value	Change in slope	Break date	χ^2 Statistic	P value	Change in slope
Australia	-	3.7	0.055	-	1970	4.3	0.037	-0.4
Austria	-	3.3	0.067	-	-	3.5	0.062	-
Belgium	-	1.2	0.276	-	-	1.8	0.186	-
Canada	-	1.3	0.250	-	-	1.7	0.188	-
Switzerland	-	0.7	0.420	-	-	1.1	0.290	-
Denmark	-	2.3	0.131	-	-	2.2	0.142	-
Spain	-	1.1	0.286	-	-	1.1	0.302	-
Finland	-	1.3	0.248	-	-	1.8	0.179	-
France	-	0.9	0.335	-	-	1.6	0.204	-
Northern Ireland	-	0.9	0.351	-	-	1.1	0.299	-
Scotland	-	1.0	0.323	-	-	1.8	0.181	-
England &Wales	-	2.8	0.095	-	-	3.3	0.070	-
Ireland	-	3.2	0.075	-	-	2.9	0.091	-
Italy	-	1.0	0.306	-	-	1.3	0.258	-
Japan	1958	4.4	0.036	0.6	1958	4.2	0.040	0.6
Netherlands	2002	4.0	0.046	-0.4	2002	8.9	0.003	-0.6
Norway	-	1.7	0.196	-	-	3.1	0.077	-
Portugal	-	2.8	0.093	-	-	3.2	0.074	-
Sweden	-	0.6	0.440	-	-	0.9	0.346	-
USA	-	2.6	0.107	-	-	2.2	0.135	-
% of cases with break points	**10%**				**15%**			

DISCUSSION

This is the first study to assess the hypothesis that the impact of the tobacco epidemic caused on the presence of structural changes in mortality decline formally. We detected structural changes in mortality trends in the large majority of countries in males but almost none in females. Unlike our expectation, a large part of the structural changes were still present after removing the effects of smoking from the mortality trends. Nevertheless, the magnitude of the changes was smaller after adjusting for smoking and the timing of some break points changed while in one case even a new change was detected.

Evaluation of data and methods

We first evaluate the quality of our data before we discuss possible explanations for the unexpected results of our study with respect to the methods used. The data on mortality were obtained from harmonized sources that rely on vital registries and population censuses of high quality.[47] For the countries included in our study the information on all-cause mortality was virtually complete, so that our results are hardly affected by missing data, which is often a problem in cross-country comparisons that include a longer time frame.[48] Although data on causes-of-death for several decades are often affected by coding changes or better diagnostic possibilities, this is less the case for lung cancer deaths that could be identified relatively clearly and were not subject to problematic coding changes.[49] We did not include smaller countries such as Iceland and Luxembourg, where irregularities in time trends might be caused by the small number of deaths rather than external influences.

In our analysis we have used the time index of the LC model as central indicator of the trend in mortality decline as this is most relevant for forecasting. Thus, our findings partly depend on capability of the model to provide a reliable description of the actual trends in mortality. It is well documented that the LC model is not able to catch more complex patterns in mortality trends, such as cohort driven influences, since it allows only for a trivial correlation structure among death rates.[50,51] However, by removing the impact of smoking from mortality rates we accounted already the most important determinant of cohort effects so that smoking-free mortality trends are certainly less affected by the simplicity of the LC model.[52] Moreover, it has been demonstrated that the detection of break points did not fundamentally change by using more complex models than the LC.[26]

Another potential source of bias might be the indirect approach to quantify the impact of smoking on mortality. Building on observed lung-cancer death rates the PGW approach utilizes a reliable indicator for the cumulative intensity of smoking to estimate the total damage of smoking on mortality. This arrives at comparable results as more complex approaches that have the additional weakness that they build on less reliable causes of death and more arbitrary assumptions about the damage of smoking.[53,54] Nevertheless, in common with all other competing approaches the PGW approach involves a static assumption as the damage of smoking on lung cancer is translated to the damage of smoking on other causes than lung cancer in the same period.[55] Thus, the trend and magnitude of the impact of smoking on mortality is determined solely by the trend and magnitude of lung-cancer mortality.[44,55] This ignores that the time lag between smoking and cardiovascular causes is much shorter

than between smoking and lung cancer.[55,56] This may explains why some structural changes did not disappear after accounting for smoking. We tested this hypothesis by performing an additional analysis, where we dated the impact of smoking on other causes than lung cancer 10 years back in time to better catch the impact on cardiovascular mortality. This reduced the time span of our sample to the years 1950-1999. Therefore, we performed the detection of break points again using the traditional version of the PGW approach and compared this to our backdated version (appendix, table A1). We found that the backdated PGW further explained the break point in the USA in males and in Australia and Austria in females but overall the results were relatively similar. We conclude that the static assumption of the PGW about the timing of the impact of smoking on other causes than lung cancer is unlikely the main explanation for our results.

For detecting structural changes in mortality rates, we used a rather simple approach comparing model with and without a single structural change using a likelihood ratio test. A more complex procedure to detect structural changes was proposed by Coelho and Nunes (2011) who combined pre-test for the presence of a structural change with a second pre-test for a unit root sequentially to decide whether the use the levels or the differences of the series of mortality trends for dating structural changes.[27] This more complex procedure was motivated by earlier results demonstrating the danger of detecting spurious breaks in time series if a unit root was present.[57] Moreover, our approach is limited to the detection of a single break point although theoretically multiple breaks are possible.[58] Bai and Perron (2003) suggested an approach that is able to detect multiple structural breaks in a times series using the Bayesian information criterion (BIC) to decide on the number of different segments in a time series.[59] We compared our findings to the alternative approaches of Coehlho and Nunes (CN) and Bai and Perron (BP) (appendix, table A3a and A3b). Generally, we found that the approach of CN seems to be more sensitive and the approach of BP less sensitive than our approach. For instances, in men the approach of CN detected a significant change in 95% of the countries and the one of BP in 55% of the countries, which was 80% in our approach. Overall, the timing of the detected structural changes was largely similar in all approaches. In none of the cases the approach of BP detected more than one structural change. Our central result that the impact of smoking does only partly explained the structural changes holds for any approach used.

The detection of structural changes might also be sensitive towards the size of the sample with respect to the age range included. Ouellette et al (2014) argued that the population below the age of 40 should be excluded from the analysis as there other

determinants are important.[29] For this reason, we also re-estimated our analyses in a sample restricted to the ages 40-99 using the traditional PGW approach and also our backdated version of the PGW, as explained above, which could only be estimated for the years 1950-1999. In this restricted sample covering only the ages 40-99 the impact of smoking explained slightly more structural changes (appendix, table A2). This was particularly the case if the backdated version of the PGW was used for the years 1950-1999, where only 15% of the countries in men and 5% of the countries in women exhibited structural changes after adjusting for the impact of smoking. This further indicates that irregularities in mortality trends were most strongly linked to smoking during the 20th century, while there were potentially other factors more important during the turn of the 21st century.

Interpretation

Our findings contribute both to the literature on mortality forecasting and to the literature on determinants of mortality trends in high-income countries.

Changes in smoking-free mortality were stable between 1950 and 2009 in half of the countries in males and almost all countries in females. This means that for a large number of countries, robust long-term projections taking the whole time series of observations as input are feasible. To avoid misleading projections, one should generally project smoking-associated mortality and non-smoking mortality separately as proposed by Janssen et al (2013).[32] This contrasts alternative solutions that suggested to extrapolate all-cause mortality based on the trends of the most recent stable period only.[25,28] Doing so will clearly arrive at wrong forecasts since the *temporary* faster decline in mortality rates due to the diminishing impact of smoking is extrapolated *permanently* into the future.

Unfortunately, our results also abate the hope that the removal of smoking generally solves the problem of irregularities in past mortality trends.[32,35] Although, the failure to explain structural changes might be partly explained by the crude assumptions of the PGW approach, this could not be the case for all break points. Independent of the approach to quantify the impact of smoking, our sensitivity checks demonstrated that smoking had a larger influence on structural changes if the sample was restricted up until the year 1999. This suggests that more recently other factors than smoking might be more relevant to explain structural changes. A promising candidate for such a factor is the health system becoming increasingly important for further gains in life expectancy in high-income countries, which will be discussed in more detail below.[60]

The structural changes with the largest magnitude were found more recently and in countries that underwent major reforms in their health system, namely Denmark, Ireland and the Netherlands. In Denmark the so called “heart plan” was launched during the mid-1990s that was targeted at reducing cardiovascular mortality, which potentially explains the detected change from slower to more rapid improvement in 1995.[61] In Ireland among other measures free health care services above the age of 70 were implemented at the turn of the 20th century, which resulted for instances in a dramatic increase of statins prescriptions.[62,63] Previous research confirmed that this increase in medical prescriptions explained a trend break in mortality trends at around 1999 that was replicated in our study.[64] In the Netherlands, fixed hospital budgets were relaxed in 2001 resulting for instances in a rapid increase in hospital admissions, in medical prescriptions, and shorter waiting times for elective surgeries in particular among the elderly that were plausibly be related to the sudden improvement in life expectancy.[24,65,66]

Other persistent structural changes occurred earlier during the 1970s and 1980s, namely in Australia, Austria, Canada, Sweden and the US (table 1a), where we initially expected that the impact of smoking was the main determinant of irregularities in mortality trends. Previously, the presence of these break points was ascribed to the occurrence of the so called “cardiovascular revolution”, where the survival, especially in males, suddenly improved due to rapidly decreasing death rates from cardiovascular causes.[29,67] In our analysis only some of the changes during the 1970s and 1980s disappeared after adjusting for smoking, which fits to the literature on the determinants of the “cardiovascular revolution” highlighting the impact of advances in treatments of cardiovascular diseases next to the importance of smoking cessation during the same time.[29] This is a further hint for the general relevance of sudden changes in the health care system.

The fact that we detected just a few structural changes in female mortality trends corresponds to studies demonstrating that smoking was less relevant for mortality trends in past since females took up smoking much later in time and to a smaller extent.[33] However, the rapidly increasing lung-cancer death rates in women should be seen as warning that the stable mortality decline in females should not be taken for granted and that periods with temporary slower improvements are possible.

Conclusion

The impact of the tobacco epidemic explained the structural changes in the decline of mortality only in some countries. After adjusting for smoking our sample of countries consists of the lucky ones with a stable long-run decline in (smoking-free) mortality rates that could be linearly extrapolated and in those having bad luck where such a simple procedure is not feasible. To identify further determinants of structural changes in mortality decline, the impact of healthcare policies and innovations in medical treatments on mortality trends should be taken into account more carefully.

APPENDIX

Table A1 Comparison of the impact of smoking on structural change in mortality trends between the traditional PGW approach and a backdated version of the PGW approach, males and females

	Males				Females			
Smoking adjustment:	none	PGW	PGW backdated	**Smoking adjustment:**	none	PGW	PGW backdated
Age range:	0-99	0-99	0-99	**Age range:**	0-99	0-99	0-99
Time span:	1950-1999	1950-1999	1950-1999	**Time span:**	1950-1999	1950-1999	1950-1999
Country	**Break date**	**Break date**	**Break date**	**Country**	**Break date**	**Break date**	**Break date**
Australia	1970	1970	1970	Australia	1983	1970	-
Austria	1983	1983	1983	Austria	-	1983	-
Belgium	1976	-	-	Belgium	-	-	-
Canada	1975	1975	1975	Canada	1992	-	-
Switzerland	-	-	-	Switzerland	-	-	-
Denmark	1990	-	-	Denmark	1977	-	-
Spain	-	-	-	Spain	-	-	-
Finland	-	-	-	Finland	-	-	-
France	-	-	-	France	-	-	-
Northern Ireland	1980	-	-	Northern Ireland	-	-	-
Scotland	-	-	-	Scotland	-	-	-
England &Wales	1979	-	-	England &Wales	-	-	-
Ireland	-	-	-	Ireland	-	-	-
Italy	1983	-	-	Italy	-	-	-
Japan	1987	1987	1987	Japan	-	-	-
Netherlands	1993	-	-	Netherlands	1987	-	-
Norway	1988	1990	1990	Norway	-	-	-
Portugal	-	-	-	Portugal	-	-	-
Sweden	1988	1988	1988	Sweden	-	-	-
USA	1968	1968	-	USA	1992	1992	1992
% of cases with break points	**65%**	**35%**	**30%**	**% of cases with break points**	**25%**	**15%**	**5%**

Table A2 Sensitivity of the detection of breakpoints with respect to the age range, time span and approach to adjust for smoking in 20 OECD countries, males and females

		Males		Females		
		Age range		Age range		
		0-99	40-99	0-99	40-99	smoking adjustment
Time span	**1950-2009**	80% (50%)	80% (40%)	10% (15%)	0% (15%)	PGW
	1950-1999	65% (35%)	55% (25%)	25% (15%)	5% (15%)	PGW
	1950-1999	65% (30%)	55% (15%)	25% (5%)	5% (5%)	backdated PGW

Note: Values represent the percentage of countries where break points were detected in the series of mortality decline, values in brackets provide the same information but with adjustment for smoking

Table A3a Comparison of the detected breakpoints between our approach and the approach of Coelho &Nunes (2011) and of Bai &Perron (2003), males

Approach:	Coelho &Nunes	Our approach	Bai &Perron	Coelho &Nunes	Our approach	Bai &Perron
Country	**Break date**	**Break date**	**Break date**	**Break date**	**Break date**	**Break date**
Australia	1970	1970	1970	1970	1970	-
Austria	1983	1983	1983	1983	1983	-
Belgium	1976	1976	-	-	-	-
Canada	1975	1975	1975	1975	1975	1975
Switzerland	1990	1990	-	-	-	-
Denmark	1994	1995	1995	1995	1995	-
Spain	2003	-	-	-	-	-
Finland	-	-	-	-	-	-
France	1983	-	-	-	-	-
Northern Ireland	1980	1980	-	1979	-	-
Scotland	1993	1993	1993	2003	-	-
England &Wales	1979	1979	1979	1999	-	-
Ireland	1999	1999	-	1999	1999	-
Italy	1983	1983	1983	1976	-	-
Japan	1955	-	-	1955	-	-
Netherlands	2000	1993	1993	2003	2003	-
Norway	1988	1990	1990	1990	1990	1990
Portugal	1999	1996	-	1999	1971	-
Sweden	1988	1988	1988	1988	1988	1988
USA	1968	1968	1968	-	1968	-
% of cases with break points	**95%**	**80%**	**55%**	**70%**	**50%**	**15%**

Table A3b Comparison of the detected breakpoints between our approach and the approach of Coelho &Nunes (2011) and of Bai &Perron (2003), females

Approach:	Coelho &Nunes	Our approach	Bai &Perron	Coelho &Nunes	Our approach	Bai &Perron
Country	**Break date**	**Break date**	**Break date**	**Break date**	**Break date**	**Break date**
Australia	1970	-	-	1970	1970	-
Austria	-	-	-	-	-	-
Belgium	-	-	-	-	-	-
Canada	-	-	-	-	-	-
Switzerland	-	-	-	-	-	-
Denmark	-	-	-	-	-	-
Spain	-	-	-	-	-	-
Finland	-	-	-	-	-	-
France	-	-	-	-	-	-
Northern Ireland	-	-	-	-	-	-
Scotland	-	-	-	-	-	-
England &Wales	2003	-	-	2003	-	-
Ireland	1999	-	-	1999	-	-
Italy	1983	-	-	1983	-	-
Japan	1955	1958	-	1955	1958	-
Netherlands	-	2002	-	-	2002	-
Norway	-	-	-	-	-	-
Portugal	-	-	-	-	-	-
Sweden	1955	-	-	1984	-	-
USA	-	-	-	-	-	-
% of cases with break points	**30%**	**10%**	**0%**	**30%**	**15%**	**0%**

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6

Forecasting educational differences in life expectancy

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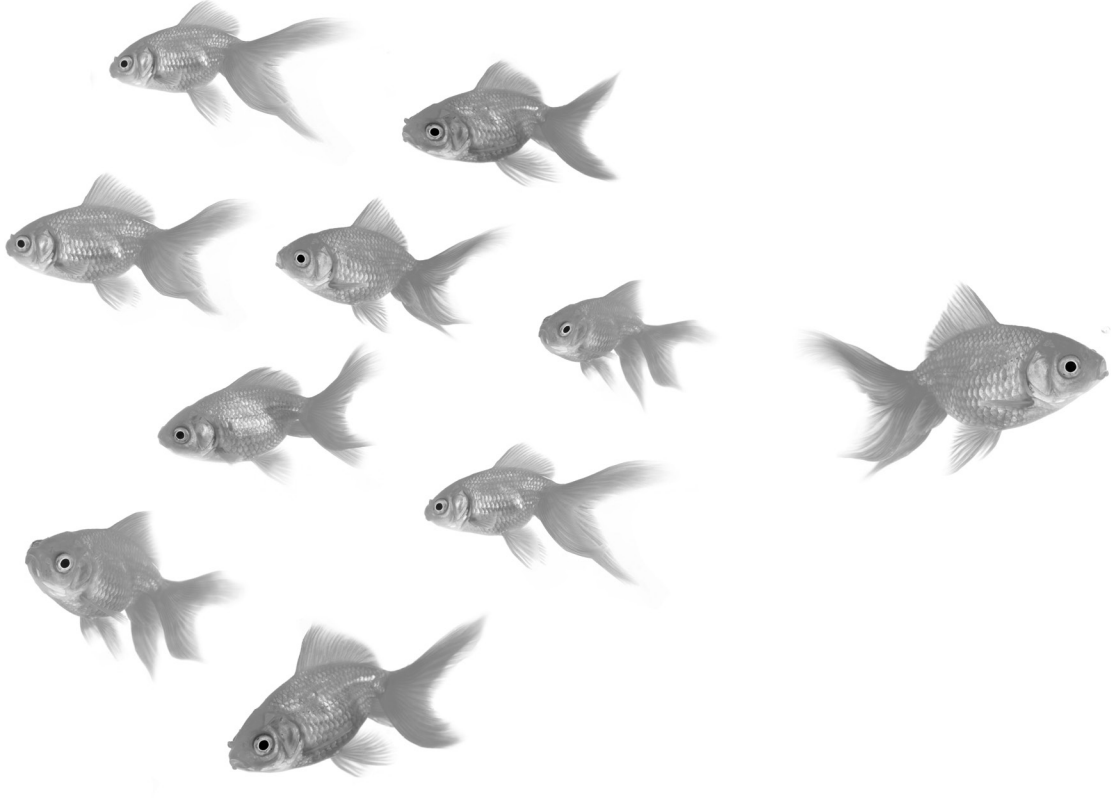
Submitted



ABSTRACT

Forecasts of life expectancy (LE) have fueled debates about the sustainability and solidarity of pension and health care systems. However, within populations there are inequalities in life expectancy between high and low educated groups that are relevant for these debates. In this paper, we present an approach to forecast LE for different educational groups within a population. As a basic framework we use the Li-Lee model which has been developed to coherently forecast mortality for different groups. We adapted this model to distinguish between overall, gender specific and education specific trends in mortality and extrapolated the time-trends in a flexible manner. We illustrate our method for the population 65+ in the Netherlands and used several data sources spanning different time windows. Results suggest that LE is likely to increase for all educational groups but that differences in LE between educational classes widen. Sensitivity analyses illustrate advantages of our proposed methodology.

INTRODUCTION

Life expectancy has been increasing in most western countries and is expected to increase in the future.[1-5] The rise in life expectancy has important implications for society as larger numbers of elderly people pose additional burdens on the health care and pension systems.[1,2] This has led to political debates in many countries regarding the statutory retirement age and how to publicly finance the growing health care expenditures. For example, some Western-European countries have explicitly linked their retirement age to the increase in life expectancy.[6] Moreover, the life insurance and annuity industry incorporates the prospect of a continuing rise in life expectancy in their products leading to higher premiums.[7,8] However, such measures ignore the great differences in the length of life with respect to socio-economic status (SES).[9,10] Those with fewer years of education have much shorter lives and a growing number of studies report even a widening of inequalities in life expectancy between SES groups, so that the trend of life expectancy of the entire/overall male and female population becomes less informative over time.[11] Consequently, forecasts for socio-economic subgroups are required to inform the political debate adequately.

Since about the 1980s a growing number of approaches for forecasting life expectancy became available.[12] Although there have been exceptions, most approaches are based on time-series extrapolation models such as the Lee-Carter model.[13] Lee-Carter based methods decompose time series of age specific mortality rates into a latent time trend and an interaction thereof with different age categories. The latent time trend is then forecasted using ARIMA modeling (mostly a random walk with drift) and serves as a basis to derive future age profiles of mortality rates and corresponding life expectancy projections. Until now, Lee-Carter based methods have been used to project mortality and life expectancy in the general population (often stratified by gender) but have not been used to project life expectancy for different SES groups. Although there have been population projections that accounted for the effect of changes in the educational distribution on mortality, these projections assumed only changes in projected overall mortality rates due to compositional changes while keeping educational differences in mortality fixed.[14,15] However, to date there are no forecasts of life expectancy (LE) stratified by level of education.

The goal of this paper is to develop an approach to forecast LE for different educational groups within a population. As a basic framework we will use the Li-Lee model which has been developed as an extension to the original Lee-Carter model to coherently forecast mortality for different groups, e.g. countries or gender.[16] The rationale behind the Li-Lee model is that trends in mortality will to some extent be

similar in populations which share common circumstances such as for instance the health care system and the economic environment. Therefore, it is unlikely that in the future mortality patterns will diverge strongly in related populations. To date the Li-Lee model has not been applied for other purposes than for projecting LE coherently for different genders within a country[16] or countries within a group of countries.[17] Our paper extents this field offering a broader class of possible applications. For this purpose, we will extend the Li-Lee model in several manners. First, we made the model more flexible for incorporating more layers of group-specific time trends, while retaining the idea that the different groups share to a certain extent common trends. Second, we demonstrate how the different layers of the model could be used to integrate data of different quality and different time length allowing to combine shorter survey-based time series on mortality disaggregated by SES with longer register-based time series on general mortality. This solves an important problem in the current literature that avoids sub-group specific forecasts because mortality data on sub-groups is often of lesser quality. We will illustrate our method for the population above age 65 in the Netherlands and forecast LE by education 30 years ahead for the years 2013-2042. Although in the Netherlands mortality by age and gender is routinely collected at the population level as part of the national vital statistics, the data on mortality by level of education is gathered from smaller and more selective surveys.[18]

Our paper is structured as follows. First, we shortly outline the Li-Lee model and pay special attention to the issue of extrapolating time trends within this framework. Second, we present the different kinds of data available for estimating educational-specific mortality rates in the Netherlands. Then, we describe our model specification and demonstrate how the education-specific mortality trends spanning over a shorter time frame could be combined with overall and gender-specific data from a longer time frame. We estimate a base case forecast for gender-specific and SES-specific LE in the Netherlands using a specific set of key assumptions on the future group-specific and common time trends. Finally, we demonstrate the sensitivity of our results to each of these key assumptions in four alternative sensitivity analyses.

The Li-Lee model

The Li-Lee model is based on the Lee-Carter model which is the most popular model to forecast mortality rates and life expectancy.[13,19,20] The Lee-Carter model postulates that mortality rates can be modelled as a function of three sets of parameters: age-specific constants, a time-varying index and interaction terms between time and age[19]:

$$\log[m(a,t)] = \alpha(a) + \beta_1(a) \times \kappa_1(t) + \varepsilon(a,t) \quad (1)$$

where *m* stands for mortality rate *a, t* are indices for age and time (calendar year). The $\alpha(a)$ parameters indicate the time-average log mortality rate stratified by age, $\kappa_1(t)$ refers to an age-independent latent time trend in mortality that is shared by all ages, while $\beta_1(a)$ can be interpreted as the interaction between each age category with the general time trend. The $\beta_1(a)$ parameters tell us at which ages mortality declines or increases more rapidly or more slowly in response to changes in $\kappa_1(t)$. The $\kappa_1(t)$ values can be treated as a time series and forecasts of mortality rates can be made by forecasting $\kappa_1(t)$ and substituting values of these forecasts of $\kappa_1(t)$ into Eq. 1. Lee and Carter proposed that specifying a time series model of a random walk with drift parameter describes $\kappa_1(t)$ best. Extensions of the Lee-Carter focused on alternative estimation techniques[20,21], how to select the optimal time-frame[22] and how to account for parameter uncertainty when forecasting mortality rates.[12,22]

A drawback of forecasting life expectancy for different but related populations (e.g. neighbouring countries) by fitting a separate Lee-Carter model for each population is that forecasts of life expectancy usually strongly diverge in the long run. This was recognized by Li-Lee who developed the Li-Lee model in response to this.[16] The central idea behind the Li-Lee model is that related populations in the long run share a common time trend, but that there may be population-specific deviations in the short run. Common trends in mortality of related populations may be the result of similarities of the childhood disease environment, dietary patterns, lifestyle[5] as well as ongoing breakthroughs in health technology that quickly diffuse.[23] Li-Lee did not specify a strict definition of what related population exactly mean but mention different genders within a country or different countries with similar levels of development. Li-Lee proposed to extend the Lee-Carter model in the following manner:

$$\log[m(a,t,g)] = \alpha(a,g) + \beta_1(a) \times \kappa_1(t) + \beta_2(a,g) \times \kappa_2(t,g) + \varepsilon(a,t,g) \quad (2)$$

Where g is an index for subgroups (for instance different countries or different genders), $\kappa_2(t,g)$ indicate the subgroup specific deviations from the common time trend $\kappa_1(t)$ and $\beta_2(a,g)$ the subgroup specific age interactions with these subgroup specific time trends. Similar as with the basic Lee-Carter model $\alpha(a,g)$ equal the average log mortality rates by age and now also subgroup. $\kappa_1(t)$ is the common time trend for all subgroups and $\beta_1(a)$ the common age interactions with the common time trend. Li-Lee proposed to forecast values of $\kappa_2(t,g)$ using a mean reverting process such as an AR(1) process.

Time trends in the Li-Lee model

Assuming a mean-reverting process for the subgroup-specific kappa parameter prevents a strong divergence of forecasts between subgroups as forecasts of $\kappa_2(t,g)$ return to values of $\kappa_2(t,g)$ as observed in the time-period used to fit the model. In other words the subgroup-specific time trends revert to their mean deviation from the common trend in the long run. This ensures that in the long run the ratio of age-specific mortality rates for different subgroups returns to values as observed in the data[1]. Consequently, forecasts of life expectancy for both the overall population and the subgroups are coherent in the sense that they do not diverge in the long run.

A drawback of assuming a mean reverting process is that if widening mortality rates between subgroups have been observed in the data these will automatically become smaller in the future (and vice versa). In case the subgroup specific time trends are difficult to characterize as a mean reverting process, Li-Lee advised to model mortality rates of that subgroup separately. Though, as noted by Li-Lee themselves, fitting a separate Lee-Carter model for each educational group might result in strong divergence in LE between educational groups when forecasting. Both options (modeling all subgroups separately or simultaneously but assuming mean reverting processes) thus have clear disadvantages. Li-Lee did not propose formal tests to decide whether the subgroup-specific trends (i.e. ($\kappa_2(t,g)$) should be modelled using a mean-reverting process or that each subgroup should be modeled separately. Rather, they proposed to look more informally at measures of goodness of fit of Eq. 2 as compared to Eq. 1[2] and at the estimates of the AR(1) model in order to decide between these modeling options.

1 This is similar as assuming the age specific hazard ratios on mortality are constant in the long run.

2 Li-Lee introduced the concept of explanation ratio's which is a measure of goodness of fit that allows to compare the contributions of the different time trends in the Li-Lee model for a defined subgroup.

DATA AND METHODS

Data

In the Netherlands there is no single data source that contains information on mortality stratified by gender, age and education. While deaths and births by age and gender have been recorded in the Netherlands since the 19th century, information on education level is not available from vital statistics in the Netherlands. We could however create a time-series on mortality by education level for the years 1996-2012 using individual level data from the Dutch Labor Force Survey linked to the municipal population registries, since 1997 known as GBA (see Appendix A for details on how we constructed these time series). Educational attainment in our analysis was classified in three categories:

- Low: primary education (basisonderwijs);
- Middle: pre-vocational education (Vmbo, mbo 1, mavo);
- High: secondary education and tertiary education (Havo, Vwo, Mbo 2,3,4, Hbo, Wo).

In this paper we focus on remaining life expectancy at age 65 because of four reasons. First of all, socio-economic differentials in mortality are at least as important at older ages than at younger ages. This relevance of mortality differentials between educational classes at older ages has been consistently found in a wide range of countries.[24] Secondly, LE at age 65 has until 2012 been the official retirement age in the Netherlands (NB: also in many other Western countries official retirement ages are around 65). Many countries have linked the pension-related income taxes to the increase in life expectancy and consider to raise retirement age in the future.[25] Third, as most health care is consumed by the elderly the results are also relevant for the debate regarding growing health care expenditures. Finally, by focusing on the 65+ we could more reliably estimate mortality trends by education given the concentration of deaths in the elderly.

Table 1 shows estimates of life expectancy (LE) at age 65 calculated from the combined data. Life expectancy at age 65 has increased more for men than for women between 1996 and 2012 although LE is still higher for women. Educational differences in LE at retirement age are more than 2.5 years for both men and women in 1996 and have widened since then for both men and women. This implies that the lower educated enjoy less years in retirement than the higher educated. From table 1 it can be seen that overall LE improved over time probably also because the distribution of educational attainment has changed in a positive manner.

Table 1 Life expectancy (LE) at age 65 for men and women stratified by educational attainment in 1996 and 2012. Between brackets percentages in different educational classes at age 65

Gender	Educational attainment	LE(65) 1996	2012
Combined	Combined	17.0	19.7
Men	Combined	15.1	18.3
	High educated	16.2 (55%)	19.2 (68%)
	Middle educated	14.5 (28%)	17.7 (22%)
	Low educated	13.5 (17%)	15.9 (10%)
Women	Combined	19.6	21.4
	High educated	21.1 (29%)	22.9 (49%)
	Middle educated	20.0 (42%)	21.8 (36%)
	Low educated	18.5 (22%)	19.6 (16%)

Methods

To extrapolate mortality rates we used the Li-Lee model as starting point and extended it in several ways. First of all, we extended the Li-Lee model by distinguishing two different layers of subgroups instead of just one: gender and education. This means that there is common time trend shared by all groups ($\kappa_1(t)$), a time trend that is shared by all education classes within each gender ($\kappa_2(t,g)$), and a time trend that is specific for each educational class by gender ($\kappa_3(t,g,e)$). For each of these time trend parameters (1+2+2x3=9 in total) there is also a set of age-specific interaction terms, which leads to the following model specification:

$$\log[m(a,t,g,e)] = \alpha(a,g,e) + \beta_1(a) \times \kappa_1(t) + \beta_2(a,g) \times \kappa_2(t,g) + \beta_3(a,g,e) \times \kappa_3(t,g,e) + \varepsilon(a,t,g,e) \quad (3)$$

where g and e are indices for gender and education. The parameters $\kappa_3(t,g,e)$ reflect the latent subgroup specific time trend per educational class stratified by gender and $\beta_3(a,g,e)$ the education and gender specific interactions with that time trend. Equation 3 can be estimated in a stepwise manner given that $\log[m(a,g,e)]$ equals the time averaged log mortality rates by age, gender and education. First, to estimate $\beta_1(a)$ and $\kappa_2(t,g)$ the basic Lee-Carter model from Eq. 1 is estimated for the total population not specified by gender and education. After estimating Eq. 1 and can be estimated using the SVD by plugging in the estimates obtained in Eq. 1. in Eq. 2:

$$\log[m(a,t,g)] - \alpha(a,g) - \beta_1(a) \times \kappa_1(t) = \beta_2(a,g) \times \kappa_2(t,g) + \varepsilon(a,g,t) \quad (4)$$

To estimate Eq. 3 and Eq. 4 we used data on overall and gender specific mortality spanning the period 1973-2012. We choose this period as from 1973 onwards, life

expectancy for both men and women has been increasing. In the years preceding this year trends in life expectancy between men and women differed starkly. This choice of period is in line with previous research that indicated that the optimal time period for the Lee-Carter model using data from the Netherlands starts in the seventies.[17,26] Furthermore, as our goal is to forecast LE 30 years ahead our choice of historical period is in concordance with a general recommendation that the historical period should be at least as long as the projection horizon.[27]

After estimating Eq. 4 $\beta_3(a,g,e)$ and $\kappa_3(t,g,e)$ can be estimated by plugging in the estimates obtained in Eq. 1 and Eq. 2 in Eq. 3:

$$\log[m(a,t,g,e)] - \alpha(a,g,e) - \beta_1(a) \times \kappa_1(t) - \beta_2(a,g) \times \kappa_2(t,g) = \beta_3(a,g,e) \times \kappa_3(t,g,e) + \varepsilon(a,g,e,t) \quad (5)$$

As the estimation of the Li-Lee model is iterative in nature this allows to use time series of different lengths. In our case this meant we used longer time series (1973-2012) to model the overall trend and gender-specific trends while using shorter time series (1996-2012) to model deviations from these trends for the different education groups. Note that only values for the κ_1 and the κ_2 parameters for the period 1996-2012 were used in Eq. 5. After fitting the model in the steps described above, one retains 9 series of time-dependent $\kappa_1(t),\kappa_2(t,g),\kappa_3(t,g,e)$ values. Forecasts of mortality rates can be made by forecasting $\kappa_1(t),\kappa_2(t,g),\kappa_3(t,g,e)$ and substituting values of these forecasts into Eq. 3. Crucial for forecasting LE is the choice of a model how to extrapolate the different kappa parameters ($\kappa_1(t),\kappa_2(t,g),\kappa_3(t,g,e)$).

We think there is always a benefit in modelling common trends if there are theoretical reasons to assume common determinants of trends in mortality for related populations. If there are clear indications that subgroup specific time trends in Eq. 3 trends are not mean reverting, this should not imply that the mortality rates have nothing in common with the overall time trend. Even if the subgroup-specific kappa parameters would not be mean-reverting, the influence of these subgroup-specific time trends will become less (and also the problem of divergence/convergence) if part of the time trend is modeled using a common trend. Also, when thinking in terms of prediction intervals there is a clear benefit of modelling common trends as this generates a positive correlation between the forecasts for the different subgroups which makes sense. In our specific application, modelling common trends also allows us to strengthen forecasts of LE by education by using longer time series for the overall and gender specific time trends. This mixture of a common time trend with potentially deviating subgroup-specific time trends follows a broader literature

highlighting the importance of common unobserved factors in time series data of separate groups.[28]

To avoid more or less arbitrary expert judgments and to consider a broader category of time-series models to forecast all kappa parameters, we propose to use a criterion based approach to select optimal time-series models for all kappa parameters. Therefore, to forecast values for the kappa parameters of the different models we selected optimal ARIMA models by comparing the BIC values of different ARIMA models. We preferred this over assuming a random walk to model overall mortality and imposing that the gender and SES specific time trends would be mean reverting.

Furthermore, as the time-series by education are rather short we preferred to select forecasting models this way, as small samples make it difficult to use testing procedures in general. It is important to note that our approach is free to arrive at the same time-series models for the kappa parameters as Li & Lee, if the data dictates this.

Sensitivity analyses

To investigate the sensitivity of our forecasts with respect to several key assumptions and to illustrate the advantages of our proposed methodology we also forecasted LE in the following sensitivity analyses:

- Sensitivity analysis A [assuming convergence]: in this sensitivity analysis we imposed a random walk with drift for the common trend $\kappa_1(t)$ and an AR(1) process for all gender and education specific time trends. This sensitivity analysis is similar to original Li-Lee model specification in which all subgroup specific trends are mean reverting;
- Sensitivity analysis B [no common trends]: in this sensitivity analysis we fitted a Lee-Carter for each group separately. Similar as in the base case projection we used data for the period 1973-2012 for overall and gender specific mortality and data for the years 1996-2012 for education specific mortality. For all Lee-Carter models we selected ARIMA models to extrapolate the kappa parameters by optimizing the BIC criterion;
- Sensitivity analysis C [shorter historical period]: in this sensitivity analysis we also used for overall and gender specific mortality only data for the period 1996-2012. Everything else is the same as in our baseline model specification;
- Sensitivity analysis D [shorter historical period without common trends]: in this sensitivity analysis we forecasted mortality by fitting a Lee-Carter model for each education and gender group separately using data from the 1996-2012

only. To extrapolate the kappa parameters we again selected the optimal ARIMA models.

Sensitivity analysis A mimics the original Li-Lee model by imposing a mean-reverting process to the subgroup specific trends. By also forecasting LE in sensitivity analyses B and D we can investigate the benefits of our proposed methodology as it allows comparing our base case forecasts to separate Lee-Carter forecasts for each subgroup. Sensitivity analysis C allows us to investigate the added value of using a longer time series to model common trends. Sensitivity analysis D is interesting to compare to sensitivity analysis C as it allows a straightforward comparison of separate Lee-Carter models and our modelling strategy in case time series for all groups are of equal length. Note that to avoid jump-off bias we used the last observed mortality rates as a starting point for our forecasts in all our analyses.

RESULTS

Figure 1 displays estimates of the α parameters of Eq. 3 which are simply the time average log mortality rates (for the period 1996-2012) stratified by age, gender an education. From the two graphs it can be seen that there is a clear educational gradient in mortality rates for both genders.

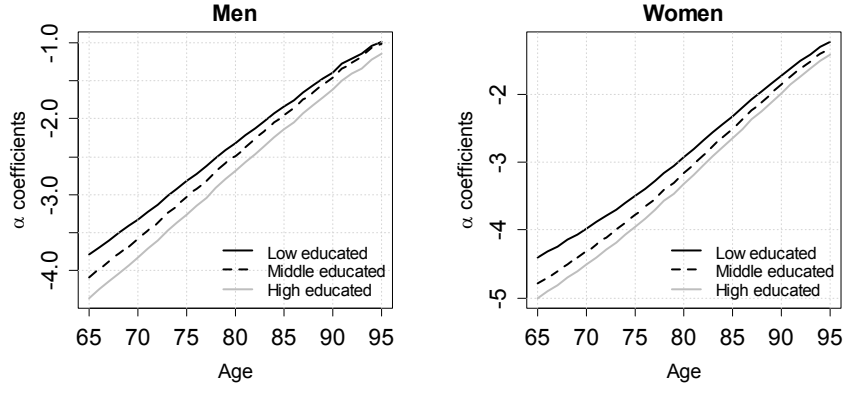


Figure 1 Lee-Carter α parameter estimates by age, gender and educational attainment in our base case model specification

Figure 2 displays estimates of the kappa and beta parameters as described in Eq. 3. From the upper left graph in Fig. 2 we can see a clear downward trend in overall mortality over time as illustrated by the decreasing κ_1 values. The deviations of the dif-

ferent genders with the overall time trend are also displayed in the same graph. For men the increasing κ_2 values for from 1973 to about 2000 suggest that mortality has been decreasing at a less rapid pace than overall while the reverse is true for women. From about 2000 onwards this pattern has reversed. However, the κ_2 parameters are difficult to interpret in isolation as they interact with the β_2 parameters, which are negative for some ages. At ages 65 to about 77 men have negative β_2 values while ages above 77 have positive β_2 values indicating opposite trends in mortality for these age categories. The middle left graph in Fig. 2 shows that the overall decline in mortality has been slower for the lower educated men as the κ_3 values increased over time. It should be noted that changes in the $\kappa_2(t,g)$ and the $\kappa_3(t,g,e)$ values over time are much smaller than changes in $\kappa_1(t)$ over time as much of the changes over time in mortality have already been captured by the common trend.

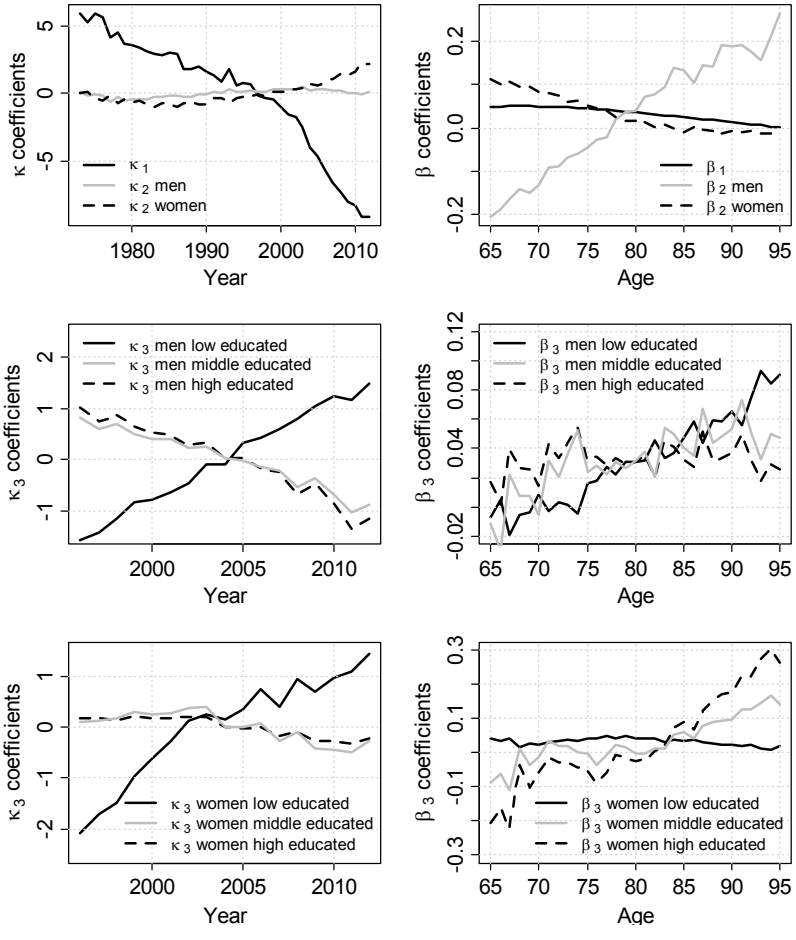


Figure 2 Lee Carter kappa (all graphs on the right) and (all graphs on the right) beta parameters of our base case model specification.

Table 2 displays the optimal ARIMA models selected using the BIC criterion used to forecast values for the different kappa parameters. From this table we can observe that the overall time trend $\kappa_1(t)$ is, similar as in previous studies, best modeled using a random walk with drift. Although the gender specific trends $\kappa_2(t,g)$ do not contain a drift term, both the time trend for men and women is not mean reverting. All education specific time trends are also not mean reverting. While for women both the high and middle educated time trend is modeled best as a random walk without drift, the lower educated time trend does contain a drift term. For men, the time trends for all educational groups contain a drift term. However, it should be kept in mind that changes in the $\kappa_3(t,g,e)$ values over time are much smaller than changes in $\kappa_2(t,g)$ and $\kappa_1(t)$ values over time so that also the 'amount of drift' is much smaller.

Table 2 optimal ARIMA models for the different kappa's

Parameter	Gender	Educational attainment	ARIMA model Mortality
$\kappa_1(t)$	Men & women		(0,1,0) with drift
$\kappa_2(t, men)$	Men		(0,1,2)
$\kappa_2(t, women)$	Women		(0,1,0)
$\kappa_3(t, men, high)$	Men	High educated	(2,1,0) with drift
$\kappa_3(t, men, middle)$	Men	Middle educated	(0,1,1) with drift
$\kappa_3(t, men, low)$	Men	Low educated	(1,1,0) with drift
$\kappa_3(t, women, high)$	Women	High educated	(0,1,0)
$\kappa_3(t, women, middle)$	Women	Middle educated	(0,1,0)
$\kappa_3(t, women, low)$	Women	Low educated	(0,1,0) with drift

Figure 3 displays trends and forecasts of LE (left graphs) and differences in LE between different subgroups (right graphs). From this figure it can be seen that LE is predicted to increase for all educational classes for both men and women but that LE increases less for the lower educated. The difference in LE between the high and low educated increases at the same pace as observed in the period 1996-2012 for both men and women. Furthermore, although differences in LE between men and women are expected to decrease, the rate of this decrease is slower than has been observed in the last decade. Also noteworthy are the prediction intervals that increase over time and the fact that the trends of the subgroups are rather similar as a result of modelling the common time trends.

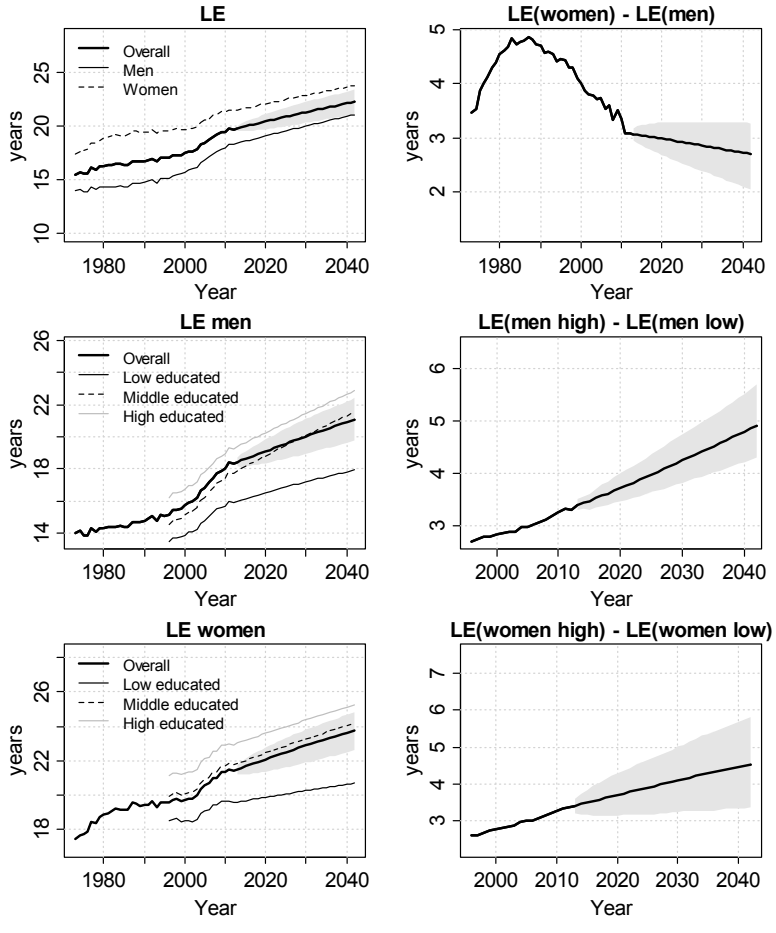


Figure 3 Forecasts of LE at age 65 for overall population and different subgroups including 95% prediction intervals and forecasts of differences in LE at age 65 between different subgroups. Overall population including 95% prediction intervals (upper left graph) and forecasts of LE at age 65 for the different educational groups (middle left graph for men and bottom left graph for women). Forecasts of differences in LE at age 65 between men and women (upper right graph), high and low educated men (middle right graph), high and low educated women (bottom right graph) including 95% prediction intervals.

Table 3 displays estimates of life expectancy in 2042 in the different sensitivity analyses and Table 4 displays differences in LE between different groups in 2042. If we compare predictions of the sensitivity analyses in the sensitivity analysis with base case analyses we can observe several things. First of all, predictions of overall and gender specific LE in sensitivity analysis C and D which are based on the period 1996-2012 are higher than in the base case projection. This is due to the fact that in this period LE has been increasing rather sharply. In sensitivity analysis A in which we imposed mean reversion we can see that differences between in LE men and women and between educational classes decline as a result thereof.

Table 3 Forecasts of life expectancy (LE) at age 65 (in years) in 2042 in base case projection and several sensitivity analyses with 95% prediction intervals between brackets

Gender	Educational attainment	Base case projection	Sensitivity analysis A	Sensitivity analysis B	Sensitivity analysis C	Sensitivity analysis D
Combined	Combined	22.3 (21.1/23.4)	22.3 (21.1/23.4)	22.3 (21.1/23.4)	24.1 (23.1/25)	24.1 (23.1/25)
Men	Combined	21.1 (19.7/22.4)	21.1 (19.8/22.3)	19.9 (15.6/23.3)	22.8 (21.8/23.8)	23.3 (22.3/24.3)
	High educated	22.8 (21.4/24.4)	21.8 (20.5/23)	24.3 (23.1/25.5)	24.4 (23.2/25.5)	24.3 (23.1/25.5)
	Middle educated	21.5 (20.1/22.9)	20.4 (19/21.7)	23.1 (21.9/24.2)	22.9 (21.7/24.1)	23.1 (21.9/24.1)
	Low educated	17.9 (16.6/19.2)	19 (17.5/20.4)	19.5 (18.9/20.1)	19 (17.8/20.2)	19.5 (18.9/20.1)
Women	Combined	23.8 (22.6/24.8)	23.8 (22.6/24.8)	24.1 (22.7/25.4)	24.9 (23.8/25.9)	24.7 (23.4/26.1)
	High educated	25.2 (23.9/26.6)	25.1 (24/26)	26.4 (24.9/27.9)	26.3 (25.3/27.3)	26.4 (24.8/27.9)
	Middle educated	24.2 (22.9/25.4)	24 (22.9/25.1)	25.3 (23.6/27.1)	25.3 (24.2/26.5)	25.3 (23.6/27)
	Low educated	20.7 (19.1/22.2)	22.4 (20.9/23.7)	21.5 (20.1/22.8)	21.4 (20.1/22.6)	21.5 (20.1/22.8)

Table 4 Differences in life expectancy (LE) at age 65 in 2042 2042 in base case projection and several sensitivity analyses with 95% prediction intervals between brackets

	Educational attainment	Base case projection	Sensitivity analysis A	Sensitivity analysis B	Sensitivity analysis C
men vs. women	2.7 (2.1/3.3)	2.7 (2.3/3.1)	4.2 (0.5/8.7)	2.1 (1.8/2.4)	1.4 (-0.2/3.1)
high vs. low educated men	4.9 (4.3/5.7)	2.8 (2.2/3.4)	4.8 (3.4/6.1)	5.4 (4.8/6.0)	4.8 (3.4/6.2)
high vs. low educated women	4.5 (3.3/5.8)	2.7 (2/3.4.0)	4.9 (2.9/7.0)	4.9 (4.5/5.4)	4.9 (2.9/6.9)

From Table 4 we can see that prediction intervals of differences in LE between subgroups increase if we model them without common trends as is done in sensitivity analyses B and D. A big advantage of using the Li-Lee approach is that the correlation between predictions of LE for different subgroups is taken into account by modeling a common trend. This results in much smaller variation in predicted differences in LE between subgroups of the base case projection and sensitivity analysis C compared to sensitivity analyses B and D in which we estimated a separate Lee-Carter model for each subgroup. The 95% prediction interval of the difference in LE between men and women includes 0 if we model no common trend in sensitivity analysis D, which seems implausible. Also noteworthy from table 3 and 4 is that by modelling common trends the predictions of differences in LE between educational groups are fairly similar while the levels of the LE predictions may change as different time periods are chosen to model the common trends.

To better understand the consequences of the key assumptions for the forecasts we compare sensitivity analysis A and B in Fig. 4 and Fig. 5. The right hand side panels of Figure 4 clearly illustrate that assuming convergence (sensitivity analysis A) for modeling education specific time trends would imply a clear trend break in the differences in life expectancy between educational classes, which seems implausible. Figure 5 shows that if separate Lee-Carter models are used (no common time trends), the forecasts of the different groups do not seem coherent with the forecasts of the overall group in which they all are part. This is illustrated most prominently by forecasts of LE for men. Thus, even in case we have diverging gender and/or education-specific time trends there is still benefit in modelling to some extent common underlying trends.

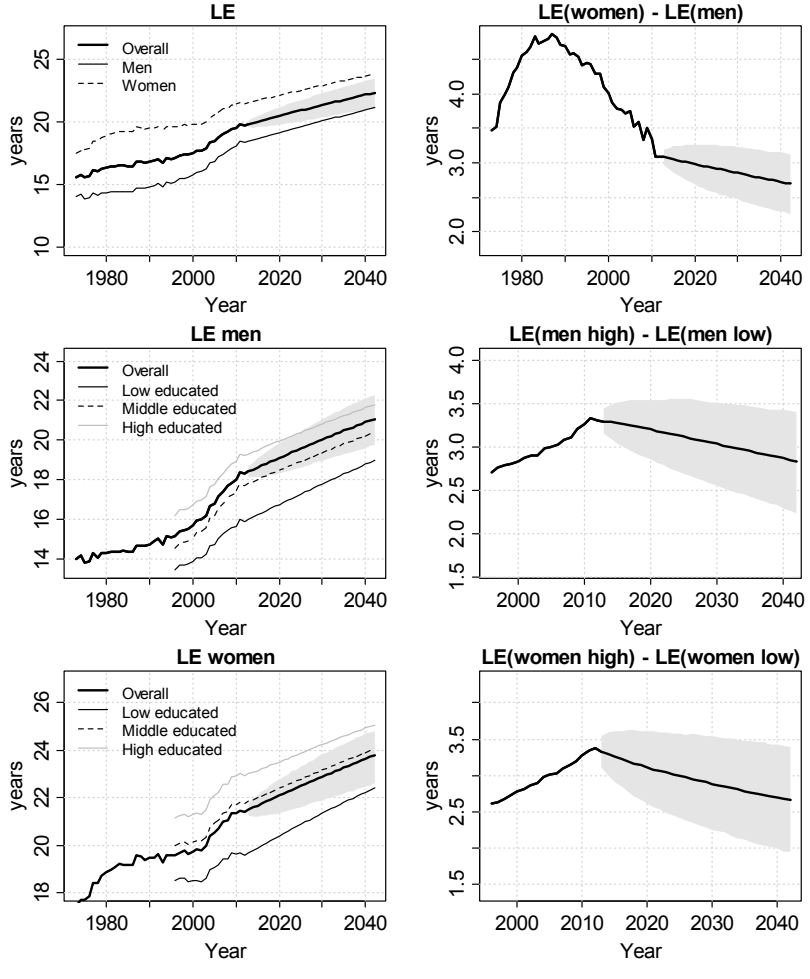


Figure 4 Forecasts of LE at age 65 in sensitivity analysis A [assuming convergence]. Overall population including 95% prediction intervals (upper left graph) and forecasts of LE at age 65 for the different educational groups (middle left graph for men and bottom left graph for women). Forecasts of differences in LE at age 65 between men and women (upper right graph), high and low educated men (middle right graph), high and low educated women (bottom right graph) including 95% prediction intervals.

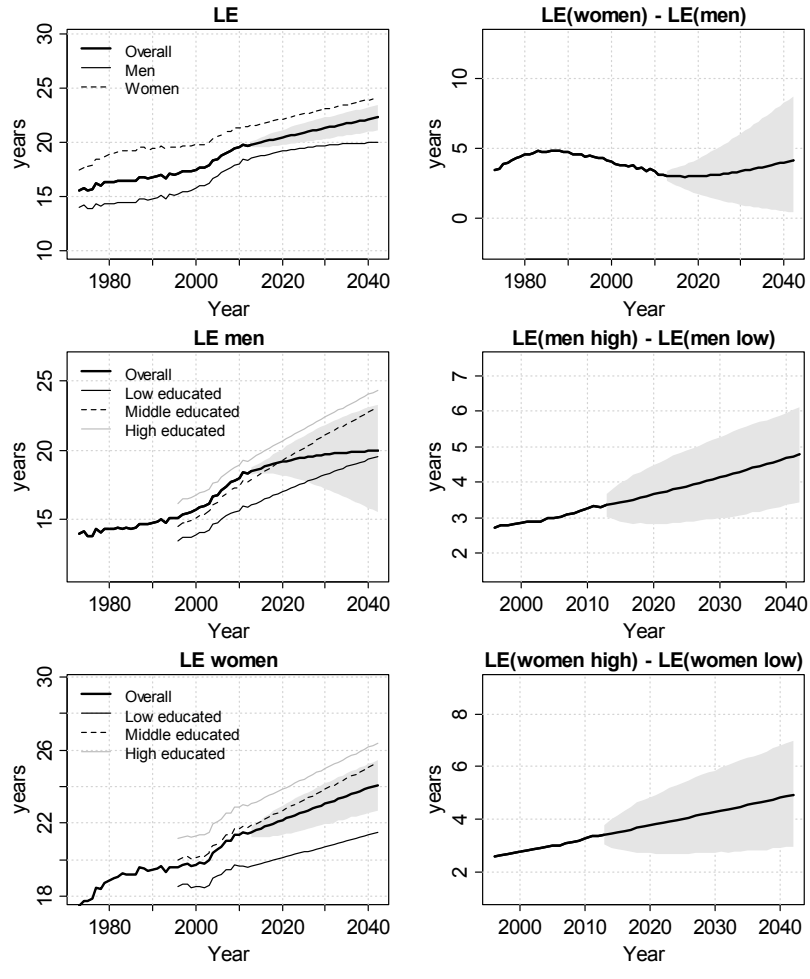


Figure 5 Forecasts of LE at age 65 in sensitivity analysis B [no common trends]. Overall population including 95% prediction intervals (upper left graph) and forecasts of LE at age 65 for the different educational groups (middle left graph for men and bottom left graph for women). Forecasts of differences in LE at age 65 between men and women (upper right graph), high and low educated men (middle right graph), high and low educated women (bottom right graph) including 95% prediction intervals.

DISCUSSION

This paper demonstrates a novel approach to combine mortality trends measured at different layers in a population (overall, gender, education) available for time frames of different length to forecast life expectancy. We demonstrated that even if subgroup specific trends in mortality appear to be diverging, modelling a common trend can have benefits. This did not only have an impact on the mean forecasts but also on the prediction intervals of the forecasts and the correlation between forecasts of different educational groups. We have illustrated the usefulness of the approach by projecting LE by level of education in the Netherlands up until 2042. Our base case projection projected a general increase at all levels with a continuing convergence of male and female life expectancy but divergence of life expectancy between the educational classes, which was slightly stronger in men than in women. In our case study we combined data from a long time series to reliably estimate the time trend on the overall group with shorter time-series of education-specific data. In that sense, the shorter time series borrowed information from the longer time series.

Our model extends the existing literature on projection of mortality trends by education in several directions. First, we suggest a flexible framework allowing subgroup-specific trends to deviate, which allows a continuation of the widening or narrowing of socio-economic inequalities if signaled by the data. Second, we augmented the Li-Lee model to additionally including time series with different length and data quality without the ad-hoc assumption on a future convergence of the subgroup-specific trends. This allows for instance to project a convergence among men and women and a divergence among educational groups at the same time. Both extensions enable a wide applicability in other areas dealing with the projection of subgroup-specific differentials. Thereby our model requires a certain degree of correlation among the groups so that the hierarchical approach plays off its strength. While life expectancy was restricted in our illustrative example to the age 65, the approach could be readily applied to project educational differences for life expectancy at birth. Other possible applications may deal with subgroup-specific differentials among groups distinguished by ethnic origin, occupation, body mass index or smoking. A specific merit of our approach is its simplicity allowing a broad range of applications with minor computational effort and relatively modest data requirements as we do not include determinants of mortality in our model. However, the latter can also be seen as a drawback of our approach. Separate modeling of smoking-associated and non-smoking associated mortality in the Netherlands revealed that in the short-run a further convergence of male and female mortality is likely.[17] A logical next step would be to investigate possibilities to include determinants when forecasting LE by education.

An important precondition for the applicability of our approach is whether the trends observed at the higher layers in our hierarchical design are suited to inform the lower layers. For example, we used the overall trend of the population as underlying trend for the subgroups men and women as well as the different educational subgroups. This helped to avoid the need to extrapolate temporary deviations caused for instances by the impact of smoking on mortality trends that differed among men and women and by education. Previous forecasting studies focused on changes in overall mortality due to compositional changes while keeping educational differences in mortality fixed.[14,15] A drawback of our approach is that while it avoids the assumption of fixed variations in mortality, it does not account for the impact of compositional changes on differential mortality trends by subgroup – neither for the past nor for the future. If such changes would have been indeed the main drivers of the observed trends, the outcomes of our model could be seriously biased if the compositional changes in the future differ fundamentally from those in the past. Moreover, if our approach is used to forecast compositional changes of the subgroups, consistency with the overall population size is not ensured. A drawback of the original Lee-Carter model as well as the Li-Lee model and our model is that the age-time interactions are assumed constant. We checked whether a changing age profile could be incorporated by adding the second factor obtained from the singular value decomposition. However, there was not a strong trend over time, and including the second factors in the forecasts only slightly increased the prediction intervals but did not change the mean predictions. We also forecasted life expectancy assuming there were no education specific time trends (this is equivalent to setting $\kappa_3(t,g,e)$ equal to zero in Eq. 3) which led to a narrowing of inequalities in LE by education. This is due to the fact the models are fitted on the log scale and that absolute decreases in mortality are bigger when mortality rates are higher. Furthermore, we also predicted LE in a sensitivity analysis in which we selected optimal ARMA models for the $\kappa_2(t,g)$, $\kappa_3(t,g,e)$ parameters instead of optimal ARIMA model. In terms of differences in LE between subgroups results were similar as in sensitivity analysis A in which we also assumed mean-reverting processes for the subgroup specific trends.

As this study represents the very first approach to forecast life expectancy by level of education/SES it is impossible to compare our results to previous forecasts. However, we can compare our forecasts of overall LE and LE by gender to previous forecasts. The most recent official projection of Statistics Netherlands (CBS) projects life expectancy at age 65 to be 22.2 years in men and 24.3 years in women in 2042[29] which is a bit higher than our projections where we estimated 21.1 years in men and 23.8 years in women in 2042. Given that Statistics Netherlands used a similar historical period (data from 1970-2011) the differences can be explained by the fact that they

included the experience of other countries of Western Europe in their variant of the Li-Lee model. Compared to the Netherlands the mortality improvement was much more positive in the other countries over the whole historical period 1970—2011. Hence, adding a shared trend among all countries in the Li-Lee model produced together with the assumption of mean reversion a more positive trend in Dutch LE than without this level. We believe that this is a meaningful assumption given the strong interdependencies of the countries in terms of economic prosperity, technological progress and lifestyles. Given the flexibility of our model, such higher layers could of course be included but we focused in this paper mainly on the layer of SES differentials for the purpose of illustration.

In our case study we employed the long run overall time trends in mortality to assist the projection of educational differences, for which only a short time series was available. This design helped to prevent implausible patterns that would arise by using only the data on education-specific mortality as input for long-term forecasts. However, at the same time our model could not fully solve the problem of sparse data on mortality by education as it is the case in most countries. Projecting convergences or divergences based on short time series always bears the risk of wrongly extrapolating tendencies that are in fact only of temporary nature. For deciding whether the outcomes are plausible or realistic, additional information need to be taken into account, such as for instances data on underlying trends of the determinants of the differences in mortality among the subgroups. Additionally, longer time series on mortality by education would help to identify more stable trend differentials. Furthermore, increased sample sizes to more reliably estimate mortality by education would improve the model. Our data on mortality by education were based on record linkage between the Dutch labour force survey which is – a representative 1% sample of the Dutch population – and the death registry, exhibiting high data quality.[30] Nevertheless the usual caveats connected to survey data apply also for this source. Non-response rates of about 40% might have resulted in a selective sample composition excluding high-risk groups.[31] However, this problem is partly mitigated by the sampling weights used.[32] Also it was demonstrated that relative mortality differentials in SES were much less affected by selective non response than absolute mortality differentials in the Dutch labor force survey.[33]

The results of our forecasts indicated diverging trends of mortality among the high and low educated subgroups, which was stronger in men than in women. A recent study on trends in socio-economic inequalities in mortality reports first signs of a narrowing of inequalities in men in several Western countries, while inequalities in women continued its widening.[34] Although this analysis did not include the Nether-

lands and targeted at another age-range (30-74), we must admit that ignoring underlying determinants of SES differentials in mortality such as smoking or alcohol consumption may have affected our forecasts. One could speculate whether the widening we found for inequalities in LE for men were too pessimistic and actually a narrowing appears more plausible. In countries with better data on education-specific life expectancy and its determinants one could test such a hypothesis in more detail. In our data we did not find signals for such a narrowing. Generally, educational attainment is related to health through a variety of mechanisms running from education to health but also vice-versa.[35,36] Nevertheless, a clear and causal effect of education on mortality has been demonstrated convincingly in a series of analyses of natural experiments, mostly compulsory schooling reforms.[37,38] Important channels through which education influences health are life-style related risk factors such as smoking, alcohol consumption, dietary patterns and physical inactivity but also financial resources, housing and work conditions and access to care. Despite great advances in medical treatment, a decrease in smoking prevalence and programs to tackle health inequalities, the large differentials in life expectancy between SES groups persisted, suggesting that more fundamental societal forces drive these inequalities.[39-41] Therefore, it is likely that SES disparities will endure in the future even if the precise mechanisms explaining the differentials change over time.

As we focused on the 65+ our LE forecasts have a clear relevance for the debate regarding retirement age and the demand for health care. As in the Netherlands current policy is to couple retirement age to LE[42] keeping the number of years in retirement more or less fixed, our forecasts suggest that the lower educated will experience a decrease in the number of years in retirement as their forecasted increase in LE is below the average increase in LE. With respect to a possible increase in the demand for health care due to increased longevity our results also suggest that this additional demand may be caused more by the higher educated than the lower educated. If financing of health care and pension schemes will not change these differential changes in LE implies a redistribution of wealth from the lower to the higher educated, because the latter will consume more healthcare resources and will receive pension payments for a longer time. Therefore, these differences in LE should be taken into account in political decisions that affect solidarity issues between SES groups.

Concluding, we believe that the extended Li-Lee model as proposed in this paper provides a useful framework to forecast LE by education. Although our method cannot solve problems caused by poor data quality it makes optimal use of available data. This might also facilitate LE forecasts for other subgroups for which less data is

available. In this sense our approach targets a larger audience including national statistical offices and actuarial societies but also other researchers in health, economic and social sciences.

APPENDIX

Estimation of mortality rates by education

Mortality rates for different education classes for the years 1996-2012 were estimated by first estimating age and calendar year specific relative risks on mortality (denoted *RR(a,t,e)* which equals the mortality rate of educational class *e,* age *a,* year *t* divided by the mortality rate of the reference educational class age *a,* year *t*). These relative risks were then used to decompose mortality rates from the total population by exploiting the following relationship:

$$m(a,t,e) = RR(a,t,e) \times \frac{m(a,t)}{\sum_e RR(a,t,e) \times p(e|a,t)} \quad (6)$$

Equation (6) states that mortality rates in a particular year at a particular age are the weighted average of the mortality rates of the different educational subgroups. ($p(e|a,t)$) denotes the proportion of an education subgroup at a particular age in a given year) and that the ratio of mortality rates between different subgroups can be expressed in relative risks. Estimates of $RR(a,t,e)$ were made using data from the Labour Force Survey (LFS) linked to the death registry. The LFS is a rotating panel survey from Statistics Netherlands that exists from 1987 onwards. The LFS is the largest data source in which information on educational attainment in collected in the Netherlands and consists of a sample of more than 60,000 households annually. From 1996 onwards it is possible to link persons that have participated in the LFS to the death registry. This makes it possible to quantify the relation between educational attainment and mortality. Values of $p(a,t,e)$ values were taken directly from the LFS. Regarding mortality, we constructed a panel where the annual number of deaths of all persons ever interviewed in LFS is obtained from the death registry and the number of exposures is estimated as the sum of the people surveyed in a particular year and the survivors from the previous year. To estimate $RR(a,t,e)$ we fitted a Poisson regression model with the exposure as offset and the expected number of deaths by year, age, education class and year as outcome variable:

$$E(D|a,e,t,y) = \exp(\theta' X) \quad (7)$$

Where y denotes year, X a vector of predictor variables and the vector of coefficients that need need to be estimated. Predictor variables were dummy variables indicating educational class and interactions thereof with age and calendar year (both as con-

tinuous variables). To control for confounding a set of dummy variables for each year and age were added to the model. Furthermore, a variable measuring the length of follow-up time in the LFS and an interaction thereof with age were added to the model. This is intended to control for selection effects into the LFS registry. From the regression model we calculated $RR(a,t,e)$. Table 5 displays estimates of exponentiated coefficients of the regression model (coefficients for the year and age dummies are not shown).

Table 5 estimated coefficients for the regression models (low educated are the reference category)

	Men	***Women***
Middle	0.677**	0.620**
High	0.497**	0.487**
Middle x age	1.055**	1.059**
High x age	1.081**	1.081**
Low x year	0.950**	1.111**
Middle x year	0.956**	1.113**
High x year	0.961**	1.108**
Low x year x age	1.011**	1.009**
Middle x year x age	1.007**	1.007**
High x year x age	1.006**	1.008**
followuptime	0.978**	1.008
followuptime x age	0.999	0.991**

* significant at 0.05; ** significant at 0.01

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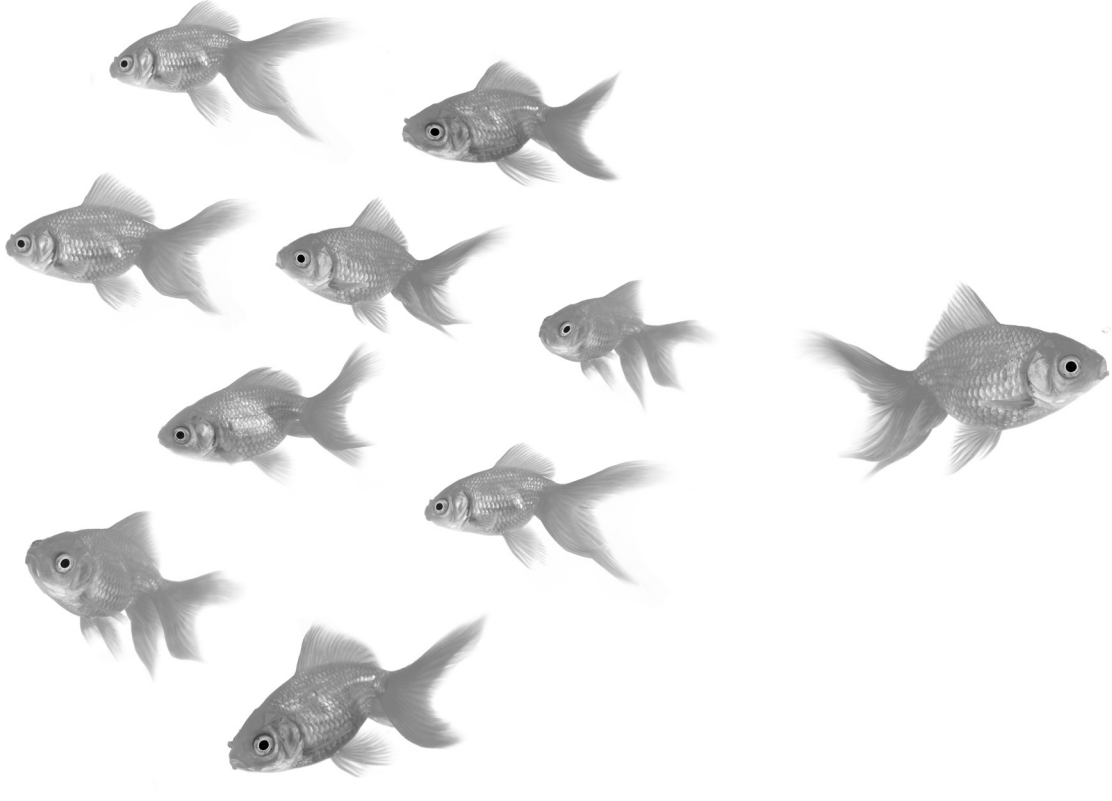
7

General Discussion



The aim of this thesis was to explain the recent trend reversal from stagnation to resumption of improvements in Dutch life expectancy and to project the deviating trends Dutch life expectancy trends into the future. This chapter summarizes key findings with respect to the three research questions stated in the introduction. Further, methodological challenges and implications for the research field of explaining and projecting deviating trends in life expectancy are also discussed. We will then conclude with policy implications and directions for further research.

MAIN FINDINGS

Answers to the study questions

1) Does period life expectancy adequately reflect changes in Dutch mortality conditions?

Generally, during times of a sudden postponement of a large fraction of deaths by a few weeks or months, trends in life expectancy at births may provide an overoptimistic impression of the progress in mortality conditions as described in chapter 2. The recent decline in Dutch mortality was strongest for those with more severe chronic conditions, and the proportion of persons with chronic conditions accumulated in the population (chapter 4). These two findings could be viewed as an indication of the existence of short-term shifts of deaths after 2001, probably resulting in tempo effects. Thus, in the short run, the rapid increase in period life expectancy might indeed reflect an overoptimistic impression of the change in mortality conditions. However, it is unlikely that this resulted in a larger distortion of life expectancy, since the group of people with more severe chronic conditions represent only a small fraction of the Dutch population. The sustained linear increase in life expectancy lasting at least a decade (chapters 3 and 5) provides a strong indication that the improvement as a whole is not an artefact. Over such a long time span, the deaths postponed by a short time would have finally occurred, which would have provoked a temporary decline in life expectancy, but this was not the case.

In sum, in the short run, period life expectancy might be inflated by tempo effects, but in the long run, there are no reasons to believe that it does not adequately reflect the underlying improvements in mortality conditions.

2) What is the explanation of the trend reversal in Dutch life expectancy?

Besides the possible role of tempo effects (chapter 2), the influence of the factors smoking and healthcare expenditures in particular was assessed for explaining the trend reversal in Dutch life expectancy.

The role of the impact of healthcare spending on mortality was assessed in detail in chapter 3, where the Netherlands was compared to the average trend of 18 other OECD countries. In result, about half of the acceleration in the increase of Dutch life expectancy of 1.8 years in men and 1.5 years in women during the period 2000-09 compared to 1990-99 was explained by more heath care spending. Within the group of 19 OECD countries, above-average improvements in Dutch life expectancy co-occurred with above-average changes in Dutch healthcare spending. In contrast to changes in healthcare spending, changes in the impact of smoking did not contribute to the acceleration of the increase in Dutch life expectancy.

The analysis in chapter 3 was complemented by an in-depth investigation of the relation between healthcare utilization and mortality at the individual level in chapter 4. There, the strongest survival improvements were measured among those suffering more severe chronic conditions, specifically among males, which indirectly confirmed that better healthcare played a crucial role during the Dutch mortality decline. However, a relation between indicators for the utilization of healthcare and mortality could not be established, which is possibly due to imperfect control for confounding. This study ruled out the explanatory power of a number of other factors, i.e. changes in health status, disability, smoking, BMI, marital status or education.

In sum, increases in healthcare expenditures contributed considerably to the trend reversal in Dutch life expectancy, whereas changes in the impact of smoking did not play a substantial role.

3) How could the deviating Dutch life expectancy trend be extrapolated?

Chapter 5 assessed the occurrence of structural changes in mortality trends before and after accounting for the impact of smoking. It was found that for both men and women, significant structural changes were present in Dutch mortality trends at around 2002 that were not caused by a decline in smoking-associated mortality. This finding has three important implications for the projection of the deviating Dutch mortality trends: First, a stable, long run trend in mortality decline since 1950, that could be extrapolated into the future, is not available. Second, mortality projection

could also not be based on the most recent linear trend in mortality decline, as this would be based on merely ten calendar years – a much too short period. Third, the explanatory factor of smoking does not help to unravel a more regular underlying mortality trend that could be extrapolated into the future.

The results of chapters 3 and 4 confirm that the factor smoking does not contribute to the acceleration of mortality improvements since 2002. By contrast, changes in healthcare expenditures contributed substantially to the improvement in life expectancy in the Netherlands (chapters 3 and 4). However, investments in healthcare are directly dependent on decisions of policy makers. Therefore, knowing current trends in healthcare expenditure does not help to anticipate future trends in life expectancy.

For projecting deviating mortality trends by subgroups of the population, a new model was developed in chapter 6. The model combined register-based data on mortality, spanning between 1973 and 2012, and survey-based data on education linked to the death registry, spanning between 1996 and 2012. A coherent projection of the overall trends, the sex-specific trends and the educational-specific trends in mortality was achieved by modelling common trends in the long run, while allowing for divergent trends in the short run, if suggested by the data. In result, an increase of 2.8 years and 2.4 years in remaining life expectancy at age 65 was forecasted for Dutch men and women up until 2042. Thereby, the gap in life expectancy between men and women was expected to narrow slightly, while at the same time, a larger widening of the gap between those with low and with high education was projected. In a sensitivity analysis, it was clearly demonstrated that one should neither assume convergence of subgroup-specific time trends nor model them completely separately.

To sum up, existing approaches to project life expectancy, like accounting for the impact of smoking or extrapolating the most recent linear period only, are not feasible in the Netherlands. Variations in healthcare expenditures partly help to explain the deviating trends in Dutch life expectancy, but it is unclear how this variable could be used in mortality projections. For projecting deviating subgroup-specific trends by gender and education, a model is required that includes a common long-term trend to ensure a plausible degree of coherence among the groups.

METHODOLOGICAL CONSIDERATIONS

This thesis assessed changes in the trends of Dutch life expectancy, aimed at identifying explanatory factors for this trend reversal and at projecting the deviating trends. For each of these tasks, different approaches were applied involving particular assumptions, merits and drawbacks. In the following, these methodological aspects are separately discussed for each of the tasks.

Describing mortality trends

For describing the exceptional development in Dutch mortality conditions, summary indicators were used to reduce the information contained in age-specific mortality rates. For this goal, period life expectancy (chapters 3, 4 and 6) and the time index of the Lee-Carter model (chapters 5 and 6) were employed. While life expectancy is a non-parametric indicator of mortality conditions, the time index of the Lee-Carter model is a parametric outcome of a statistical model. Although both measures provide surprisingly similar information about central time trends in Dutch mortality conditions, each measure has its own strengths and drawbacks.

In contrast to the more abstract time index of the Lee-Carter model, the interpretation of period life expectancy (PLE) is more intuitive.[1] Probably for this reason, the measure is one of the most widely used and cited indicators in mortality analysis.[2] Conclusions drawn on trends in PLE directly influence policy debates, thereby guiding important decisions on the allocation of scarce resources.[3] Despite its comprehensibility, PLE is also a "very complex and abstract measure".[4] The interpretation of it as average lifespan of new-borns is valid only if mortality rates would prevail throughout the whole life.[5] Yet, in the past decades, mortality rates have been declining dramatically without any signs of slowing down.[6-8] For this reason, the value of period life expectancy in a given year approximately matches the actual cohort life expectancy of those born about 40 to 50 years ago.[9]

Although PLE is certainly not useful for predicting the actual lifespan of individuals, it is considered to be a reliable summary index of changes in survival conditions of a population under study. This strength originates in the construction of PLE, where solely age-specific mortality rates of a single period are linked in a cumulative way in the life table to estimate the survivor function.[5] Hence, the measure promptly signals a worsening or an improvement in annual mortality rates, as it did during the Spanish Flu in 1918, where life expectancy in the Netherlands declined about 8 years within a

single period.[10] In this sense, PLE could also be understood as a magnifier for examining mortality trends.

A central point of dispute among scholars is to which extent the summary of trends in mortality rates corresponds to changes in underlying mortality conditions.[11,12] Proponents of the concept of tempo effects, as described in more detail in chapter 2 of this thesis, argue that changes in PLE not necessarily reflect the underlying actual process of lifesaving. This is countered by the argument that tempo effects have so far failed to provide falsifiable predictions, and that it is almost impossible to uncover the process in lifesaving.[13] Information on the prolonged survival of avoided deaths at the population level is usually not at hand.

In this thesis, PLE was mainly employed to study the sudden decline in mortality rates occurring since about 2002. Thus, the intuitive interpretation of the measure or its possible prediction for the lifespan of individuals was less of an issue. In fact, the characteristic of PLE to act as a magnifier for detecting period effects was the main reason for using the measure, particularly in chapter 3. Whether the period-driven improvement in life expectancy reflects proportional improvements in mortality conditions could not be verified, given the limitations of the current approaches to detect tempo effects. Due to the persistent improvement in Dutch life expectancy lasting for at least a decade, it appears very unlikely that the whole increase was completely driven by short-term delays of deaths.

The time index of the Lee-Carter model describes the central changes in log mortality rates over time, complemented by an interaction term to allow for slower and faster age-specific changes and a factor that describes the average age profile of mortality.[14,15] Whether the information provided by the estimated model parameters provides a plausible summary of mortality conditions depends on the appropriate fit of the model to the data. A well-known weakness of the Lee-Carter model is that it does not adequately model time-specific or cohort-specific changes in the pace of age-specific improvements of mortality rates.[16,17] Nevertheless, the model has been proven to be useful in describing past mortality trends in many situations.[7,18] The estimated time trend of the model has been extensively tested for its time series characteristics to draw information about the future development and uncertainty of mortality.[19] In many applications, the Lee-Carter model remains to be the preferred approach, or at least an important benchmark, due to its simplicity. Although the time index of the Lee-Carter model is often analysed separately, the mortality rates projected at the basis of the index are usually translated to life expectancy.

Quantifying the impact of smoking and of healthcare spending on mortality trends

Trends in life expectancy of countries cannot be studied in a controlled laboratory-like situation where effects of different factors would be verified in isolation. Rather, the observational data used in this thesis (chapters 3, 4 and 5) contains all sorts of influences affecting mortality simultaneously; therefore, the identification of particular variables that were responsible for the exceptional development of the Netherlands is a central problem. This means that strict causality is not easily established. Nevertheless, the analysis of the deviating trends in Dutch life expectancy would also be of poor value if it remains at only the level of description. The second part of this thesis carefully aimed at moving beyond that level by taking the effects of changes in smoking and healthcare expenditures into account (chapters 3 and 4).

In comparison to many other variables, the causal effect of smoking at the individual level is established and the underlying mechanisms are well understood.[20] Further, approaches are available to indirectly estimate the cumulative effect of past smoking at the population level by multiplying observed lung-cancer mortality rates by sex-, age- and time-specific translation factors drawn from high-quality cohort data.[21] Unlike for other causes of death, information on lung cancer is more reliable and its coding scheme did not change over time.[22] The indirect approaches to estimate mortality associated with smoking have been validated and improved throughout recent years, so there is a general consensus on the usefulness of these methods proven to be valuable in various empirical studies.[23-27] Further, these approaches are more widely applicable, more precise and more reliable than traditional methods used to estimate the damage from smoking, as for instance, based on the often imprecise self-reported smoking prevalence from health surveys.[21]

Within this thesis, two different approaches to estimate smoking-attributable mortality were used: the classical method developed by Peto et al. (1992) applied in chapter 3 and the method proposed by Preston, Glei and Wilmoth (2010), the PGW approach, applied in chapter 5.[21,24] The reason for this difference is that at the time chapter 3 was prepared, the latter approach could not be used below age 50. Recently, coefficients for the PGW approach up until age 35 were provided, so that this method could be used in chapter 5.[25] Theoretically, the PGW approach is superior to the Peto et al. approach, as it involves fewer arbitrary assumptions.[28] In recent applications, however, it has been shown that both approaches arrive at similar estimates, meaning that the results in chapter 3 and 5 are comparable.[26] A central assumption of both methods is that lung-cancer rates in a particular period could be directly translated to smoking-associated mortality rates from other causes than lung cancer

in the same period.[29] Yet, lung cancer develops on average after 20 to 30 years of smoking, while coronary heart diseases due to smoking occurs with a shorter time lag and chronic obstructive pulmonary diseases (COPD) due to smoking occurs with a longer time lag.[30] Although this timing problem is particularly relevant for chapter 5, the sensitivity analysis therein demonstrated that the results were robust to alternative assumptions about the timing of the impact of smoking. For the Netherlands, this timing issue is less relevant because the impact of smoking was gradually decreasing in males and increasing in females at the time the sharp trend reversal in Dutch life expectancy happened (chapter 3).

Unlike for smoking, there are more doubts about a causal impact of changes in healthcare spending on mortality. Although most researchers agree that healthcare has a substantial role in modern societies helping to avoid premature mortality and to further extend the human lifespan, important methodological challenges and caveats to quantify its impact are mentioned in the literature.[31-37] To isolate the impact of changes in healthcare expenditures (HCE) on life expectancy (PLE) is particularly challenging because the two are strongly interrelated and also closely linked to economic growth, for instances measured by the gross domestic product (GDP).[38,39] Further, the relation between healthcare spending and mortality is likely confounded by the progress in health technology and changes in health-related behaviour that are harder to measure than total money spent.[36,40-43] Finally, the impact of healthcare spending on mortality is dynamic because investments in better care have an immediate effects, e.g. if prolonging life of terminally-ill patients in critical care units, but also delayed effects, e.g. due to screening and general prevention.[38,44]

In chapter 3 of this thesis, a dynamic panel approach was used to overcome the problems of estimating the impact of healthcare spending on life expectancy using data for 19 countries over 30 years. There, a relevant effect of changes in HCE on PLE was detected that explained about half of the acceleration of the increase in Dutch life expectancy since 2000. The model specification accounted for the high correlation between HCE and GDP, the influence of unobserved common factors, and the dynamic impact of HCE on PLE. Further, serial correlation over time and between countries was explicitly taken into account. A limitation of this approach was that it could merely estimate the average effect over all countries and all years, representing a relatively crude effect estimate. For instance, it would be interesting to know whether the effect of HCE on PLE was actually stronger or weaker during the 2000s in the Netherlands than in the other countries to better evaluate the cost-effectiveness of the Dutch healthcare reform. Also, the design did not adjust for the possibility that healthcare expenditures were partly increased by the prolonged survival of patients,

which would result in reverse causality between PLE and HCE. Generally, previous research indicated that improved survival did only raise healthcare costs by a small extent, since the onset of more severe diseases was also postponed.[45-48]

The results of chapter 5 provide indirect support for the hypothesis that changes in healthcare spending contributed to the Dutch trend reversal in life expectancy. Among 20 Western countries, only the Netherlands, Ireland and Denmark experienced more recent structural changes in the decline of mortality rates. In all three countries, the changes in mortality trends were not explained by changes in the impact of smoking. Further, all three countries underwent major healthcare reforms at the time life expectancy improvement accelerated, specifically tackling the treatment of cardiovascular diseases.[49-51]

Also the findings at the individual level in chapter 4 indirectly suggest that healthcare played an important role during the recent Dutch mortality decline. Mainly those with more severe chronic conditions, which are often of cardiovascular type, benefitted from the improved survival conditions. Unfortunately, the expected link between higher utilization of healthcare and lower mortality could not be established, which was possibly due to imperfect control for confounders and the inclusion of the population residing in institutions. In sum, this thesis presented various direct and indirect indications in favour of the hypothesis that better healthcare enabled the increase in Dutch life expectancy, by using complementary research designs and levels of aggregation.

Projecting deviating mortality trends

The issues pertaining to the description of mortality trends also apply for the projection of deviating mortality trends into the future. Thanks to its intuitive interpretation PLE is usually the main outcome of any mortality projection. However, due to PLE's inherent complexity and little importance for the expected lifespan of real people, it could be asked why PLE is used for projections at all. Further, as argued above, period-driven changes – both real ones and tempo effects – have a great influence on time trends in PLE, so projections may extrapolate unusual temporary effects far into the future. For these reasons, it has been claimed that mortality projections should focus on directly extrapolating either cohort life expectancy or a proxy measure for cohort life expectancy, namely the cross-sectional average length of life (CAL).[52-54] It is argued that such measures better reflect the expected survival of real persons and are less prone to tempo-effects and other period effects; therefore they are more relevant for pension funds and life insurance products.[55]

In fact, the issues explained above mainly apply if PLE would be directly extrapolated. Yet, the chapters of this thesis dealing with projection (chapter 5 and 6) analyse, model and extrapolate age-specific mortality rates directly. The estimates were merely translated to PLE after the projection of mortality rates was completed. For the same as the description of mortality rates, PLE mainly serves as an intuitive summary indicator. By comparing the outcomes between subgroups and to other Dutch projections and countries, the plausibility of the projection could be better evaluated by using PLE. Still, the projected series of mortality rates could be used to compute cohort life expectancy or other summary indicators. Since the Lee-Carter model is fit to a longer time series in chapter 6, potential tempo effects or other short-term period effects do not harm the projections of life expectancy. This is because the extrapolation of mortality rates is based on the average change in the time index of the Lee-Carter model over the full time span, attenuating the distorting influence of tempo effects. However, the uncertainty around the projections might be increased by to such effects.

A particular strength of the projecting model developed in chapter 6 was that sex-specific and education-specific mortality rates were modelled in common with mortality rates of the whole population to ensure coherence, while at the same time, it allowed for divergent trends, if suggested by the data. This model structure enabled the first dynamic forecast of socio-economic differences in mortality and life expectancy published so far. Using the time period 1973-2012 as the historical period for estimating the common long-term time trend for all subgroups helped to avoid that the model would be driven by the period of the stagnating mortality decline between 1980 and 2001 or the rapid increase between 2002 and 2012. The model did not account for smoking, as previous analyses in this thesis revealed that the variable did not help to explain the divergent trends in Dutch mortality decline. Nonetheless, by using the pooled mortality rates of the whole population as central time trends in the model, the distorting effect of the impact of smoking that worked in the opposite direction for men and women was indirectly accounted for. Therefore, the predicted outcome of life expectancy in 2040 was very close to the forecasts of Statistics Netherlands, which explicitly accounted for smoking using a more complex approach.[56]

The projection model applied in this thesis departed from other Dutch projection models by not taking the information of other countries into account, as was, for example, applied in the most recent forecast of the Dutch Royal Actuarial Association (AG).[57,58] This decision could be justified by the findings of this thesis that deviating trends in healthcare spending partly explained the deviating trends in Dutch mortality. Including mortality trends of other countries in the projection means to assume

that the trend in healthcare expenditures in the Netherlands converge to the trends in other countries. However, healthcare spending is strongly dependent on decisions of policy makers, and given the divergence of Dutch healthcare policy in the past, it is likely that Dutch policy makers will again decide for different solutions in the future. Austerity measures also affecting the health sector have already been implemented more recently in the Netherlands. The subsequent stagnation of Dutch life expectancy in 2012 could be interpreted as further evidence for the strong link between healthcare spending and mortality although the progress of life expectancy resumed in 2013.

Finally, the projection model in this thesis did not follow the suggestion to only project the most recent linear period. Clearly, the results in chapter 3 indicate that this is a very dangerous approach, as it potentially extrapolates temporary faster or slower rates of mortality improvement, e.g. due to smoking or changes in healthcare, into the far future. For the case of the Netherlands, this extrapolation of short-term trends would mean that the cost explosion in Dutch healthcare since 2002 would continue for several decades. Rather than performing such speculative projections, the central findings of this thesis suggest that mortality trends and their drivers need to be assessed in great detail before decisions about the historical period employed for extrapolation could be made.

CONTRIBUTION TO THE RESEARCH FIELD

Deviating trends in Dutch life expectancy were studied before extensively, in particular during the time of slow improvements of life expectancy. Moreover, various approaches have been developed and applied to project the nonlinear trends in Dutch mortality. In the following chapter, we will summarize the contribution of our findings to this field of research.

Description of mortality trends

PLE is commonly used to describe mortality trends in the Netherlands and in most other countries. In this thesis, the so far neglected concept of tempo effects was first introduced to a wider audience of public health. Although the concept itself has its limitations and failed to provide a straightforward solution, the implicit assumption of the life table on avoided deaths could be seen as an additional aspect of the high complexity of PLE.[4] Consequently, other researchers employing this indicator for

monitoring mortality trends should be more careful about the interpretation of the trends, during times of sudden and strong external events in particular. The trend reversal in the Netherlands is one example, where the nature of lifesaving deserves a further look before drawing a conclusion about the increase in life expectancy. This adds on to previous examples, such as the case of the fast convergence of life expectancy in East and West Germany after the unification in 1990.[59] Nevertheless, if the central assumptions of PLE were kept in mind, the use of the measure as a sensitive indicator of period-driven changes in mortality trends is still recommended. Caution is required if the indicator is applied to evaluate short-term consequences of policies given the potentially misleading interpretation.[60] However, if rates of mortality improvement are stable over a longer period of time, as it was the case in the Netherlands, tempo effects are probably of less relevance.

Explanation of mortality trends

The stagnation of improvements in Dutch life expectancy attracted a large body of research, especially because during that time, mortality rates at older ages got even worse, which is rarely observed in high-income countries.[61,62] Still to this day, no convincing evidence for this lack of progress in life expectancy has been published.[63,64] It was initially believed that smoking partly contributed to the slower improvement in Dutch life expectancy.[61,65] However, analyses of Janssen et al. (2007) already showed that adjusting for the impact of smoking revealed an even stronger stagnation in life expectancy that was more similar among men and women.[66] Further data on the consumption of manufactured cigarettes revealed that the smoking intensity in the Netherlands during the 1980s and 1990s was not higher than in other countries that did not experience a stagnation in the improvement of life expectancy.[67] The results of this thesis in chapters 3, 4 and 5 show that the resumption of increases in life expectancy was not explained by smoking. Nevertheless, accounting for the confounding and suppressing effects of smoking constitutes an important prerequisite to study other factors, such as changes in healthcare expenditures. Adjusting for the stronger impact of smoking on Dutch mortality trends among males than females revealed that break points in mortality decline occurred at around the same time near 2002, as was demonstrated in chapter 5. The findings of chapter 5 further challenge the argument that mortality trends, after adjusting for the impact of smoking, develop more regularly over time.[8,27,68] Rather, important structural changes in mortality decline not related to smoking were detected in a wide range of countries. In a methodological sense, this calls into question the validity of indirect approaches for estimating the impact of smoking particularly with respect to the timing of the

impact.[28,29,69] More generally, the persistence of trend breaks in mortality decline challenges the widespread belief that deviations in mortality decline are mostly temporary and that there exists an underlying linear trend in life expectancy common to all high-income countries.[6,7,70,71]

The hypothesis that changes in healthcare expenditures explained the upturn in Dutch life expectancy has been first been proposed by Mackenbach et al. (2011), who also confirmed that the changes in mortality were due to a period effect and not due to a cohort effect.[51] Bonneux (2011) disagreed with that hypothesis and questioned a connection between healthcare expenditure and life expectancy at the country level.[63] The latter statement was falsified by the findings in chapter 3, where a beneficial effect of changes in healthcare spending was found within the group of 19 OECD countries. When applying this average effect to the case of the Netherlands, it explains about half of the acceleration in life expectancy during 2000-2009. The country comparison further confirmed that both changes in Dutch healthcare expenditures and changes in Dutch life expectancy were exceptional during that time. Overall, the body of evidence in this thesis clearly supports the hypothesis that increases in healthcare spending triggered the upturn in Dutch life expectancy.

The results of chapter 4 add to the literature that the recent improvement of survival in the Netherlands was strongest among those with more severe chronic conditions. This complements the findings of Deeg et al. (2013) who arrived at a similar result for the group of the elderly that were largely excluded in the analysis in chapter 4.[72] Interestingly, the study of Deeg et al. (2013) found a relatively strong impact of an increasing proportion of higher education and decreasing prevalence of smoking on the improvement in mortality, while in this thesis, these variables had no explanatory power.[72]

Projection of deviating mortality trends

Due to the irregular mortality trends, official forecasts of Dutch life expectancy differed substantially during the recent years, both in terms of the approach used and the predicted outcome. Generally, three broad groups of projection exist: extrapolation, explanation, and expectation.[73] In 2010, each of these different approaches was applied by different institutions in the Netherlands and each arrived at different outcomes already in the short run.[74] This diverse situation represents the large uncertainty regarding the projection of Dutch life expectancy arising from its turbulent trends in the past.

This thesis does not provide a superior solution to project mortality rates for the whole population compared to earlier approaches. However, the presented findings help to evaluate common solutions proposed for projecting irregular mortality trends, which helps to develop better models in the future. The results of chapter 5 are in contrast to previous literature aimed at identifying and extrapolating the most recent stable period of improvements in mortality rates.[19,75,76] So far, approaches dealing with that issue ignored the fact that accelerations in the pact of mortality decline were often consequences of a diminishing impact of smoking.[77,78] Clearly the impact of smoking needs to be taken into account, as it explains break points in mortality trends for several countries, and also because adjusting for smoking substantially effected the slopes of mortality change over time. Ignoring these features likely results in misleading mortality projections. But the results of chapters 3 and 5 also indicate that additional factors other than smoking need to be identified for explaining deviating mortality trends, like healthcare expenditures.

This thesis presented a general framework to predict deviating subgroup-specific mortality trends within a population in common with overall trends (chapter 6).[71] In comparison to existing static approaches or pure scenario-like approaches, this is the first projection model where changes in relative risks between socio-economic subgroups over time were inferred from the data and extrapolated into the future.[79,80]

IMPLICATIONS

Implications for policy

Although this thesis demonstrated that smoking was not important to explain abrupt changes in mortality trends in the Netherlands, it continued to exhibit a large impact on Dutch life expectancy (chapter 3). Over the past twenty years, the negative impact of smoking on life expectancy decreased in males from about 4 years in 1990 to 2.5 years in 2009 - still a very high level. In females, the impact of smoking was almost negligible in 1990, but amounts for a lowering of life expectancy of already 2 years in 2009, which is likely to further increase. These values are far above the international average and should alert policy makers to implement much stricter legal regulations that concern smoking, such as increases in taxation on cigarettes sales, health warnings on cigarette packages, and stricter bans of smoking in public and private areas. In 2007/08, still about a quarter of the men and women between age 35 and 85 in the Netherlands reported to smoke regularly (chapter 4).

The results of this thesis confirmed the hypothesis that changes in the healthcare sector played an important role during the reversal of trends in Dutch life expectancy. Given the enormous increase in Dutch healthcare expenditures, it appears necessary to identify those changes in the healthcare system that improved survival and health status most effectively and efficiently. The proportion of GDP spent on healthcare used to be one of the lowest in the Netherlands as compared to other countries during the 1990s, and then rose to the second-highest value worldwide in 2011, which was only surpassed by the USA. This should encourage policy makers to further promote the economic assessment of medical treatments, prevention programmes and investments in health infrastructure. Particularly during times of economic hardship and austerity, it appears to be crucial to cut investments only in a way that the health of the population is not seriously affected.[81] Because the elderly and those persons with more severe chronic conditions benefitted more from the improvement in Dutch life expectancy since about 2002, these are likely the first to suffer from austerity measures related to the healthcare system.[51,72] In the future, rather than introducing huge reforms of the healthcare system at once in the whole country, a gradual introduction of more specific reforms with a different timing for the different Dutch regions would allow a better evaluation of the particular measures. Given the persistent uncertainty about the underlying driving forces of Dutch life expectancy and its relevance for public budgets, efforts to increase public funding of research on this topic should receive a high priority, especially during economically hard times. If more effective ways to improve the population survival and population health could be identified, such research investments would be cost saving in the long run.

Implications for further research

Countries deviating from the gradual progress in life expectancy, such as the Netherlands, are opportune to study the major drivers of mortality trends in developed countries. In particular, the relatively rare occurrence of abrupt structural changes in the decline of mortality calls for closer inspection. Previous research has mainly focussed on the effect of smoking on irregular trends in mortality, and so far, little is known about other determinants. Although this thesis could not fundamentally change this unsatisfying situation, the case of smoking provides an encouraging example that it is possible to isolate and quantify the impact of a single factor at the population level. Nevertheless, this thesis also demonstrated that evaluating the effects of smoking might belong to the often described low-hanging fruits, and that unravelling the influence of other factors and quantifying their impact is much more difficult.

The evidence presented in chapters 3, 4 and 5 clearly indicates that the reasons for the sudden resumption of life expectancy in the Netherlands are beyond the impact of smoking. The finding that a steep rise in healthcare spending since about 2001 contributed to the increase in Dutch life expectancy should encourage further research on the mechanisms of this relation. For instance, changes in healthcare utilization among the elderly suffering severe chronic conditions – a group not included in the analysis in chapter 4 – should be evaluated in more detail. One could enable this by linking medical records from general practitioners, medical specialists and hospitals to the death registry. Particularly, more intense and better treatment of cardiovascular diseases should be taken into account. Since such patterns in the Netherlands simultaneously changed across the whole country, it is advisable to use other countries as a sort of control group for comparing trends in the incidence and treatment of diseases and the related survival.

Further, it might be worthwhile to look for other factors where the lag time between exposure and mortality is long, as is the case for smoking. This would allow one to anticipate future changes in mortality trends by using current data on the respective variables. A promising candidate for such a factor could be education, since this variable provides a good predictor for health-related behaviour and the accumulation of material and immaterial resources later in life. Thus, the recent changes in the educational distribution may help to inform about the development of survival in the future. More generally, any change in preventive medicine, such as better cancer screening or timely treatment of health-related risk factors, could be used to predict future health and mortality of populations.

Another line of research could aim at identifying more meaningful measures of mortality conditions than PLE. For this purpose, the remaining cohort life expectancy at age 65 or at age 80 could be used. Unlike PLE, the interpretation of this indicator is clearly linked to real existing persons and is not as out-dated as cohort life expectancy at birth, which links to conditions of people born more than a century ago. A further improvement would be to add the dimension of quality to measuring mortality conditions. For this purpose, trends in health expectancies could be analysed in more detail, allowing the chance to draw more realistic conclusions about actual improvements in population health. Again, remaining cohort life expectancy at older ages could be used to avoid problems related to period measures. Clearly, the ultimate goal of policy makers should be to maximize the years lived in good health by real persons.

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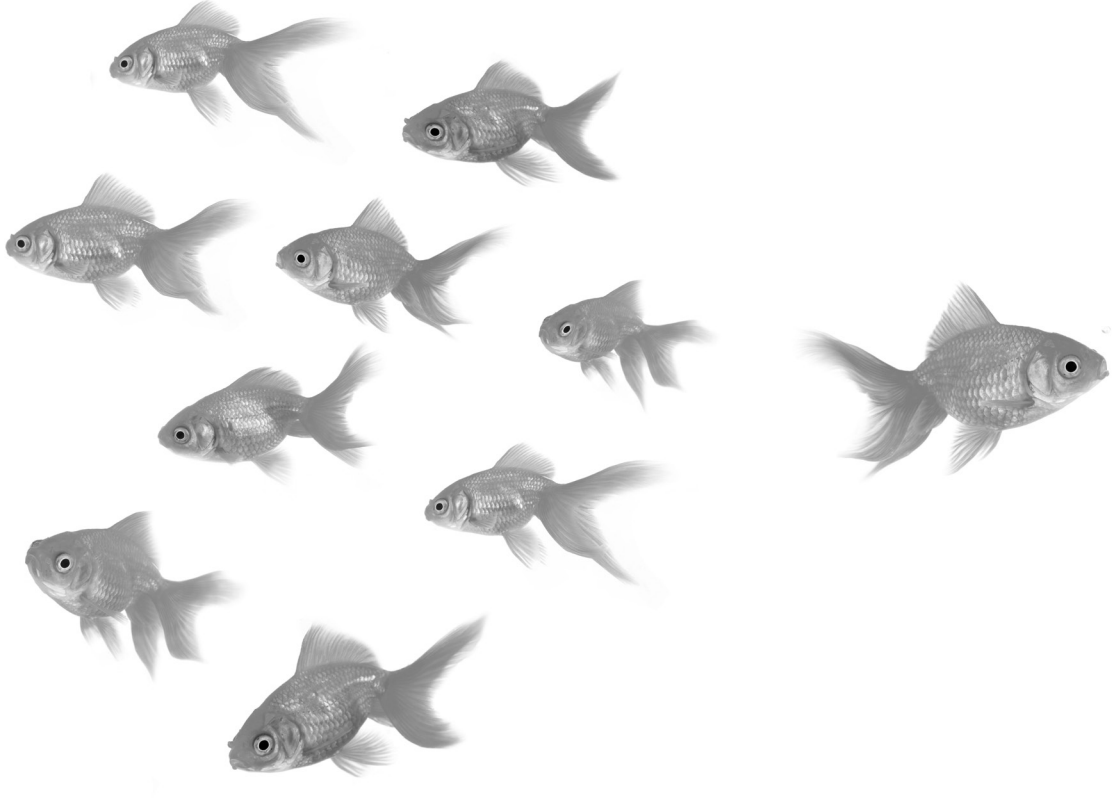
Summary



SUMMARY

After almost two decades of slower improvement and partly even stagnating progress, Dutch life expectancy increased substantially since 2002. Both the long period of stagnation and the sudden resumption of improvements constitute deviations from the more regularly developing life expectancy trends in most other Western countries. The sudden reversal of trends in Dutch life expectancy greatly affected official projections of life expectancy, which became much more optimistic after 2002. Although such a positive outlook promises a great societal advancement it also poses a financial burden for the Dutch welfare state, particularly for the healthcare sector, the pension funds and life annuities. For better assessing the future trends of Dutch life expectancy and its societal consequences, the underlying reasons for the deviating trends in the past need to be identified. So far convincing evidence on factors driving the deviating development is lacking. The leading hypotheses focus on the impact of smoking to explain the stagnation and the impact of changes in healthcare expenditures to explain the resumption of improvements in Dutch life expectancy.

The aims of this thesis were first to evaluate whether the indicator period life expectancy adequately reflects underlying mortality conditions, second to explain the recent trend reversal from stagnation to resumption of improvements in Dutch life expectancy and third to assess how the deviating Dutch life expectancy trend could be extrapolated into the future. To address these issues, the Dutch situation was assessed within a group of comparable Western countries.

In the first part of thesis in chapter 2, it was assess under which conditions life expectancy provides a sound indicator of underlying mortality conditions. This review suggested that during times of a sudden postponement of a large fraction of deaths by few weeks or months, trends in life expectancy at births may provide an over-optimistic impression of the progress in mortality conditions. Whether this was the case in the Netherlands could only be indirectly inferred from findings presented in chapter 4 of the thesis. The recent decline in Dutch mortality was strongest for those with more severe chronic conditions and the proportion of persons with chronic conditions accumulated in the population. This provides an indication of the existence of short-term shifts of deaths after 2001 probably resulting in tempo effects. However, the group of people with more severe chronic conditions represent only a small fraction of the Dutch population and the linear increase in life expectancy lasted for at least a decade (chapter 3 and 5). Hence, in the short run period life expectancy might be indeed inflated by tempo effects but in the long run there are no reasons to be-

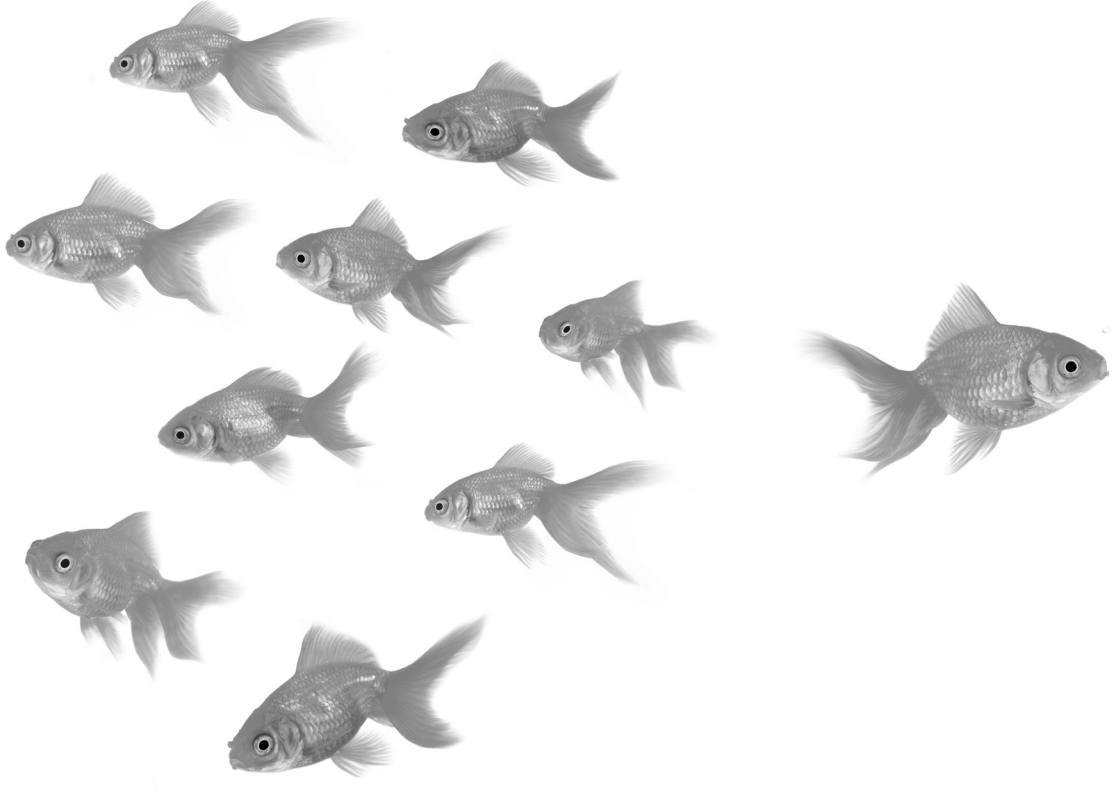
lieve that the change in life expectancy does not adequately reflect the improvement in underlying mortality conditions.

The second part of this thesis, covered in chapters 3 and 4, tackled the deviating trend in Dutch life expectancy. It was found that increases in healthcare expenditures contributed considerably to the trend reversal in Dutch life expectancy, whereas changes in the impact of smoking did not play a substantial role. About half of the acceleration in the increase of Dutch life expectancy was explained by more heath care spending (chapter 3). At the same time above-average improvements in Dutch life expectancy co-occurred with above-average changes in Dutch healthcare spending within a group of 19 OECD countries. In contrast to changes in healthcare spending, changes in the impact of smoking did not contribute to the acceleration of the increase in Dutch life expectancy. At the individual level, the strongest survival improvements were measured among those suffering more severe chronic conditions, specifically among males, which indirectly confirmed that better healthcare played a crucial role during the Dutch mortality decline. However, a relation between indicators for the utilization of healthcare and mortality could not be established, which is possibly due to imperfect control for confounding. Other factors taken into account, i.e. changes in health status, disability, smoking, BMI, marital status or education, had no explanatory power.

The third part of the thesis, Chapters 5 and 6, dealt with the projection of deviating mortality trends. In chapter 5 it was found that for both men and women, significant structural changes were present in Dutch mortality trends occurring around 2002 that were not due to lower smoking-associated mortality. Thus, existing approaches to project life expectancy, like extrapolating a stable long run trend of mortality decline into the future, accounting for the impact of smoking or to extrapolate the most recent linear period only, are not feasible in the Netherlands. Although changes in healthcare expenditures contributed substantially to the improvement in life expectancy in the Netherlands (chapters 3 and 4), this factor unlikely helps to anticipate future trends in life expectancy given its dependence on decisions of policy makers. For projecting deviating mortality trends by subgroups of the population, a new model was developed in chapter 6. A coherent projection of the overall trends, the sex-specific trends and the educational-specific trends in mortality was achieved by modelling common trends in the long run, while allowing for divergent trends in the short run, if suggested by the data. In result, the gap in life expectancy between men and women was expected to narrow slightly, while at the same time, a larger widening of the gap between those with low and with high education was projected.

The main results of the thesis were summarized in chapter 7 complemented by a discussion of the limitations, methodological challenges, and implications for policy makers and further research. The following main conclusions could be drawn from our results. Although period life expectancy at birth is a sensitive indicator of changes in mortality conditions, its interpretation could be misleading if a large fraction of deaths were suddenly postponed by a short amount of time. Although there was some indication for the presence of such shifts in the Netherlands – possibly triggered by a sudden growth of healthcare expenditures – the sustained increase in Dutch life expectancy since 2001 could be interpreted as substantial improvement in Dutch mortality conditions. The internationally deviating Dutch trends over the past three decades are not explained by changes in the impact of smoking. Accounting for the impact of smoking revealed simultaneous trend breaks in mortality decline of Dutch men and women at around 2002. These breaks occurred most likely due to sudden changes in healthcare expenditures that explained about half of the acceleration in life expectancy during 2000-2009. The precise mechanisms how the additional money spend in the healthcare system resulted in lower mortality rates could not be established in this thesis and needs to be assessed in further research. Neither accounting for smoking nor identifying structural changes in mortality decline solved the problem that for the Netherlands a simple linear extrapolation of past trends is not feasible. Hence, further research on the factors underlying the deviating Dutch trends is necessary to allow better projection of Dutch life expectancy. For projecting such trends by subgroups of the population the projection model needs to ensure coherence while allowing at the same time divergence among the specific groups.

Samenvatting



SAMENVATTING

Na bijna twee decennia van langzamere verbetering en deels zelfs stagnerende vooruitgang, is de Nederlandse levensverwachting aanzienlijk gestegen sinds 2002. Zowel de lange periode van stagnatie en de plotselinge hervatting van de stijging zorgen ervoor dat de trends in de Nederlandse levensverwachting afwijkt van de regelmatiger trends in de levensverwachting in de meeste andere Westerse landen. De plotselinge ommekeer van de trend in de Nederlandse levensverwachting heeft de officiële prognose van de levensverwachting sterk beïnvloed: deze is veel optimistischer is geworden na 2002. Hoewel een dergelijk positief vooruitzicht een grote maatschappelijke vooruitgang belooft, vormt het ook een financiële last voor de Nederlandse verzorgingsstaat, met name voor de zorgsector, de pensioenfondsen en verzekeraarsn. Om de toekomstige trends van de Nederlandse levensverwachting en de maatschappelijke gevolgen hiervan beter te kunnen beoordelen, moeten de onderliggende oorzaken voor de afwijkende trends in het verleden worden geïdentificeerd. Tot dusver ontbreekt overtuigend bewijs voor factoren die verantwoordlijk zijn voor deze afwijkende ontwikkeling. De belangrijkste hypotheses richtten zich op de invloed van roken om de stagnatie in levensverwachting te verklaren en de invloed van veranderingen in zorguitgaven om de hervatting van stijging in de Nederlandse levensverwachting te verklaren.

De eerste doelstelling van dit proefschrift was het evalueren of de indicator 'periode levensverwachting' de onderliggende sterfte condities adequaat weerspiegelt. De tweede doelstelling was om de recente ommekeer van de trend van stagnatie naar de hervatting van stijging in de Nederlandse levensverwachting te verklaren. De derde doelstelling was om te beoordelen hoe de afwijkende trend van de Nederlandse levensverwachting kan worden geëxtrapoleerd naar de toekomst. Om deze kwesties te onderzoeken, werd de Nederlandse situatie vergeleken met de situaties in vergelijkbare westerse landen.

In het eerste deel van dit proefschrift (hoofdstuk 2) werden simulaties gedaan om na te gaan onder welke voorwaarden levensverwachting een betrouwbare indicator is van de onderliggende sterfte condities. Dit suggereerde dat in tijden van een plotseling uitstel van een groot deel van de sterfgevallen met enkele weken of maanden, trends in de levensverwachting bij geboorte een te optimistische indruk kunnen geven van de vooruitgang van sterfte condities. Of dit het geval was in de Nederlandse situatie kon alleen indirect worden afgeleid uit de bevindingen gepresenteerd in hoofdstuk 4 van dit proefschrift. De recente afname van sterfte in Nederland is het sterkst voor mensen met ernstige chronische aandoeningen, waardoorhet per-

centage mensen met chronische aandoeningen toeneemt in de populatie. Dit geeft een indicatie van het bestaan van korte termijn verschuivingen van sterfgevallen na 2001, welke waarschijnlijk resulteerde in tempo-effecten. Maar de groep mensen met ernstige chronische aandoeningen is slechts een klein deel van de Nederlandse bevolking en de lineaire toename van de levensverwachting duurde ten minste een decennium (hoofdstuk 3 en 5). Daarom zou de korte termijn de periode levensverwachting inderdaad kunnen zijn opgeblazen door tempo-effecten. Maar op de lange termijn zijn er geen redenen om aan te nemen dat de verbetering in onderliggende sterfte condities niet juist worden weergegeven door de veranderingen in de levensverwachting weergeeft.

Het tweede deel van dit proefschrift, hoofdstuk 3 en 4, behandelde de afwijkende trend in de Nederlandse levensverwachting. Uit ons onderzoek bleek dat de toename van zorguitgaven aanzienlijk hadden bijgedragen aan de breuk in de trend van de Nederlandse levensverwachting, terwijl veranderingen in de gevolgen van roken geen belangrijke rol speelden. Ongeveer de helft van de versnelling in de toename van de Nederlandse levensverwachting werd verklaard door grotere zorguitgaven (hoofdstuk 3). Bovengemiddelde verbeteringen in de Nederlandse levensverwachting kwamen tegelijkertijd voor met bovengemiddelde veranderingen in de zorguitgaven in Nederland binnen een groep van 19 OESO-landen. In tegenstelling tot veranderingen in de zorguitgaven droegen veranderingen in het effect van roken niet bij aan de versnelling van de toename van de Nederlandse levensverwachting. Op individueel niveau werden de grootste verbeteringen in overlevingskansen gemeten onder personen met ernstigere chronische aandoeningen, in het bijzonder onder mannen. Dit bevestigde indirect dat betere gezondheidszorg een cruciale rol heeft gespeeld in de daling van de Nederlandse sterfte. Een relatie tussen indicatoren voor het gebruik van gezondheidszorg en sterfte kon echter niet worden vastgesteld. Dit is mogelijk te wijten aan een onvolledige controle voor verstorende factoren. Andere factoren waarvoor gecorrigeerd was, namelijk veranderingen in de gezondheidstoestand, lichamelijke beperkingen, roken, BMI, burgerlijke staat en opleidingsniveau, konden de sterftedaling niet verklarent.

Het derde deel van dit proefschrift, hoofdstuk 5 en 6, behandelden de projectie van afwijkende sterfte trends. Belangrijke structurele veranderingen in de Nederlandse sterfte trends rond 2002 waren aanwezig voor mannen en vrouwen, en konden niet worden toegeschreven aan lagere sterfte geassocieerd met roken (hoofdstuk 5). Bestaande benaderingen om de levensverwachting te projecteren, zoals het extrapoleren van een stabiele lange termijn trend in sterfte dalingen naar de toekomst, waarin rekening wordt gehouden met het effect van roken of het extrapoleren van

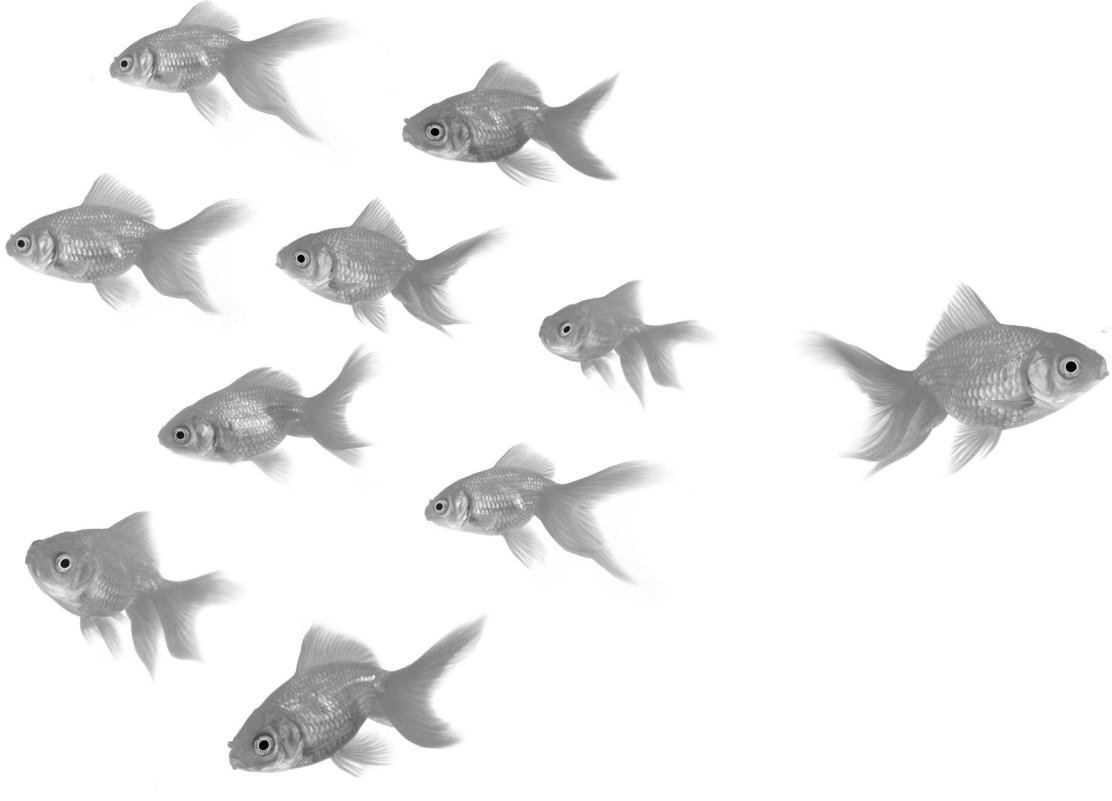
de meest recente lineaire periode van afname, zijn dus niet geschikt voor de Nederlandse situatie. Hoewel veranderingen in zorguitgaven aanzienlijk hebben bijgedragen aan de verbetering van de levensverwachting in Nederland (hoofdstuk 3 en 4), is het onwaarschijnlijk dat deze veranderingen, vanwege de afhankelijkheid van beslissingen van beleidsmakers, bijdragen aan het anticiperen op toekomstige trends in de levensverwachting. Voor het projecteren van afwijkende trends in sterfte voor bepaalde bevolkingsgroepen werd een nieuw model ontwikkeld (hoofdstuk 6). Een coherente projectie van de algemene sterfte trends, de trends in sterfte naar geslacht en de trends in sterfte naar opleidingsniveau werd bereikt door het modelleren van gemeenschappelijke trends op de lange termijn, terwijl uiteenlopende ontwikkelingen op de korte termijn werden toegestaan wanneer de data dit suggereerde. Deze projectie resulteerde in de verwachting dat het verschil in levensverwachting voor mannen en vrouwen lichtelijk zou verkleinen, terwijl het verschil in levensverwachting voor personen met een laag en een hoog opleidingsniveau zou toenmen.

De belangrijkste resultaten van dit proefschrift zijn samengevat in hoofdstuk 7, aangevuld met een bespreking van de beperkingen, methodologische uitdagingen en implicaties voor beleidsmakers en verder onderzoek. De volgende belangrijkste conclusies kunnen worden getrokken uit onze resultaten. Hoewel de periode levensverwachting bij geboorte een gevoelige indicator is voor veranderingen in sterfte condities, kan de interpretatie misleidend zijn wanneer een groot deel van de sterfgevallen plotseling worden uitgesteld voor een korte tijd. Hoewel er enige aanwijzingen zijn voor de aanwezigheid van dergelijke verschuivingen in Nederland – mogelijk veroorzaakt door een plotselinge toename van zorguitgaven – wordt de aanhoudende toename van de Nederlandse levensverwachting sinds 2001 geïnterpreteerd als een substantiële verbetering van sterfte condities in Nederland. Veranderingen in de gevolgen van roken verklaren niet waarom de Nederlandse trends in de afgelopen drie decennia afwijken van trends geobserveerd in andere landen.

Nadat rekening was gehouden met het effect van roken werden er gelijktijdige trendbreuken in sterfte dalingen voor Nederlandse mannen en vrouwen rond 2002 vastgesteld. Deze trendbreuken treden waarschijnlijk op als gevolg van plotselinge veranderingen in zorguitgaven, welke ongeveer de helft van de toename van de levensverwachting tussen 2000 en 2009 verklaarden. De precieze mechanismen over hoe extra geld besteed aan de gezondheidszorg resulteerde in lagere sterftecijfers konden niet in dit proefschrift worden vastgesteld. Dit zou in verder onderzoek onderzocht moeten worden. Noch rekening houden met roken, noch het identificeren van structurele veranderingen in de sterfte daling loste het probleem op dat voor Nederland een eenvoudige lineaire extrapolatie van trends uit het verleden niet ge-

schikt is is. Vandaar dat verder onderzoek naar factoren die ten grondslag liggen aan de afwijkende Nederlandse trends noodzakelijk is om een betere projectie van de Nederlandse levensverwachting te realiseren. Voor het projecteren van zulke trends voor bepaalde bevolkingsgroepen, moet het projectie model coherentie waarborgen, maar tegelijkertijd ook verschillen tussen deze specifieke groepen toe laten.

Acknowledgements



ACKNOWLEDGEMENTS

The evil spirits the plagued me during the past years that I spent working on this thesis were not summoned by the intellectual hardships – as one may would guess – but rather by emotional ones. Writing a thesis in a foreign country literally felt like a leap into the unknown. Having survived the adventure and now being back in Rostock again I could admit that never before in my life I felt so overwhelmed as during my time in Rotterdam. Looking back, it appears to me almost impossible how I managed to finally finish this thesis. Without any doubt the decisive factor were all the great people that were there for me and helped me along the way. Beside all the difficulties I had to face at my time at MGZ I also experienced what John Donne meant by his famous words “No man is an island, entire of itself; every man is a part of the continent, a part of the main”. After all, I would never have been able to write this thesis without the great people in Rotterdam that helped me in the past years. Dear colleagues and friends – heel erg bedankt!

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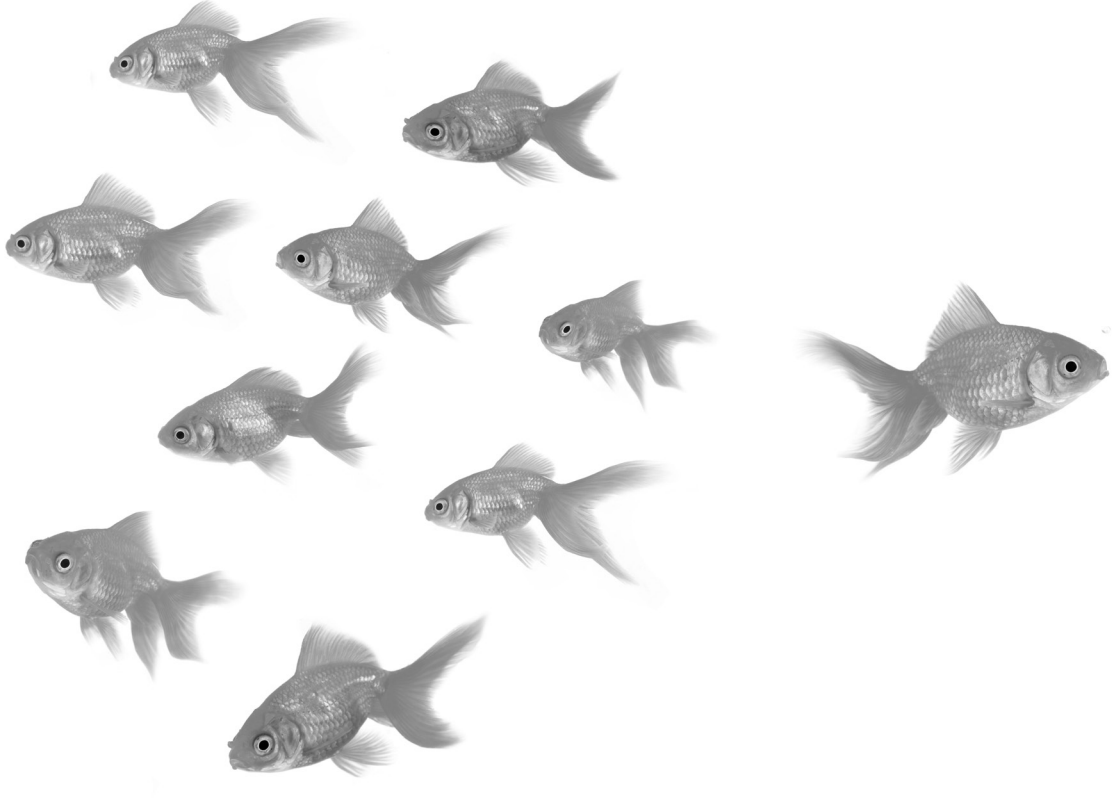
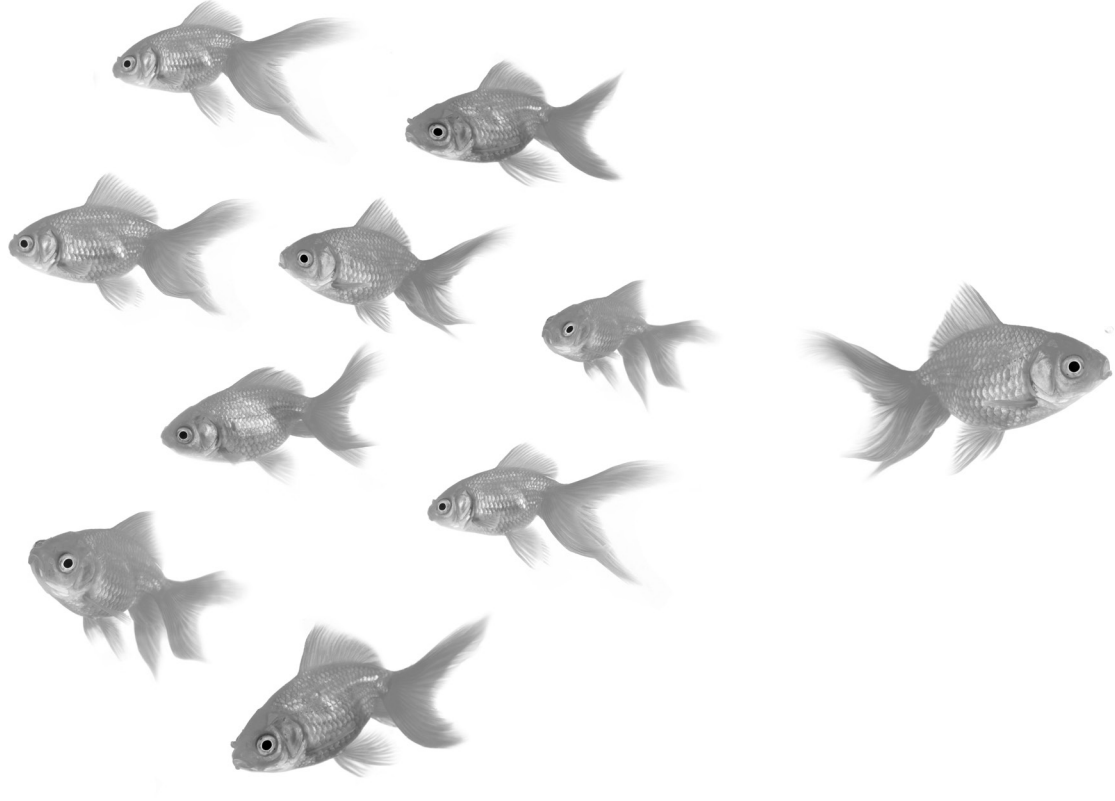
About the author



ABOUT THE AUTHOR

Frederik Peters was born in Altenburg, Germany on March the 24th 1980. After completing secondary school in Penig in 1998, he performed a year compulsory Civillian Service at a rehabilitation hospital in Bad Lausick. In 2000 he started to study Sport Sciences in Leipzig, which he couldn't finish due to serious injuries. In 2005 Frederik enrolled in Social Sciences at Rostock University, which he completed in 2008 with a Bachelor's Degree. At the same university, he obtained his Master of Science in Demography in 2010. In the same year Frederik won a grant for participating in the eleven-month training programme of the European Doctoral School of Demography hosted by the Lund University/Sweden that was finally awarded with a Research Master in Demography. In 2001 he started to work as a junior researcher at the Department of Public Health of the Erasmus Medical Center in Rotterdam in the Netherlands. There he was involved in a national research project with the goal to explain and project the deviating trends in Dutch life expectancy resulting in this thesis. Currently, Frederik works as research assistant at the Chair of Demography, University of Rostock located in Germany.

List of publications



LIST OF PUBLICATIONS

Papers in this thesis

Peters F, Mackenbach JP, Nusselder WJ (submitted) Does the tobacco epidemic explain structural changes in the decline of mortality?

Van Baal P, Peters F, Mackenbach JP, Nusselder, WJ (submitted) Forecasting educational differences in life expectancy.

Peters F, Nusselder WJ, Mackenbach JP (submitted) Quantifying the contribution of changes in healthcare epxenditures and in smoking to the trend reversal in Dutch life expectancy.

Peters F, Nusselder WJ, Mackenbach JP (2015) A closer look at the role of health care in the recent mortality decline in the Netherlands – results of a record linkage study. *Journal of Epidemiology and Community Health*, 69 (6), 536-542

Peters F, Nusselder WJ, Mackenbach JP (2013) Tempo effects may distort the interpretation of trends in life expectancy. *Journal of Clinical Epidemiology*, 67 (5), 596-600

Other publications

Milewski N, Peters F (2014) Too Low or Too High? On Birthweight Differentials of Immigrants in Germany. *Comparative Population Studies*, 39(1), 3-22

Peters F, Nusselder WJ, Mackenbach JP (2013) The longvevity risk of the Dutch actuarial society. *Netspar Design Paper 11*.

Temme J, Kramer A, Jager KJ, Lange K, Peters F, Müller GA. et al. (2012) Outcomes of male patients with Alport syndrome undergoing renal replacement therapy. *Clinical Journal of the American Society of Nephrology*, 7(12), 1969-1976.

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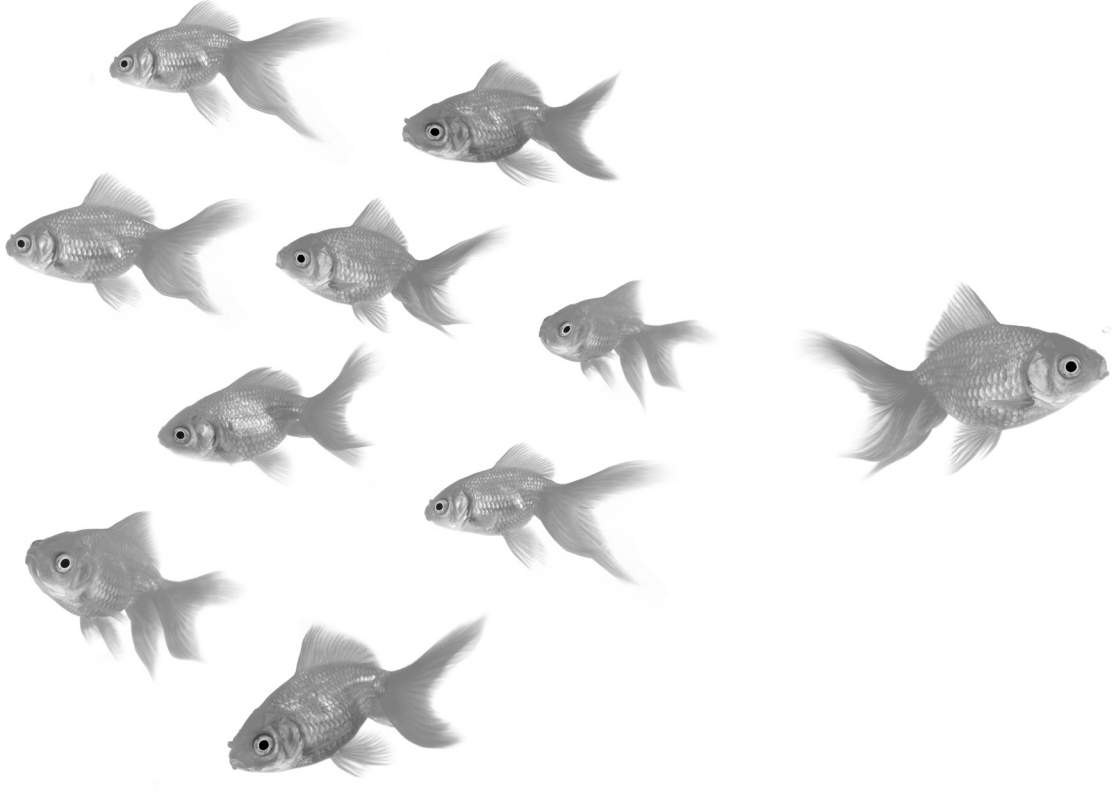
Doblhammer G, Milewski N, Peters F (2011) Monitoring of German Fertility: Estimation of Monthly and Yearly Total Fertility Rates on the Basis of Preliminary Monthly Data. *Comparative Population Studies*, 35(2), 245-278.

Kreyenfeld M, Scholz R, Peters F, Wlosnewski I (2011) Order-Specific Fertility Rates for Germany Estimates from Perinatal Statistics for the Period 2001-2008. *Comparative Population Studies*, 35(2), 207-224.

Straube S, Voigt M, Scholz R, Peters F, Hallier E, Briese V, Jorch G (2009) 18th Communication: Preterm Birth Rates and Maternal Occupation – The Importance of Age and Number of Live Births As Confounding Factors. *Geburtshilfe und Frauenheilkunde*, 69(8), 698-702

PHD Portfolio



PHD PORTFOLIO

PhD Student: Frederik Peters
Erasmus MC Department of Public Health

PhD Period 2011-2015
Promotor: Prof.Dr. J.P. Mackenbach
Supervisor: Dr. W.J. Nusselder

	YEAR	WORKLOAD (ECTS)
1. PhD training		
Academic Degrees obtained		
Research Master in Demography, European Doctoral School of Demography, Lund, Sweden	2012	
Courses		
Biomedical English Writing and Communication	2014	4.0
Writing group MGZ	2013	1.0
Presentations		
Fixed effects modelling in social epidemiology, Social Epidemiology Methods Meeting, Rotterdam, The Netherlands	2014	1.0
The divergent trend in Dutch life expectancy 1980-1009: The role of health care spending; Netspar Pension Day, Utrecht, The Netherlands	2013	1.0
The divergent trend in Dtuch life expectancy: The role of health care spending, Marburg, Germany	2013	1.0
Mortality tempo effects - Are you a believer?, MGZ Methods Group, Rotterdam, The Netherlands	2013	1.0
The divergent trend in Dutch life expectancy 1980-2009: The role of health care spending and smoking, MGZ Research Meeting, Rotterdam, The Netherlands	2013	1.0
Estimating and forecasting future age-, sex- and education-specific disability rates in the Netherlands, Bio-demography and Multistate Event History Analysis in Interdisciplinary Studies on Healthy Aging, Beijing and Hangzhou, China	2012	1.0
Measuring the relative progress of mortality improvement to evaluate stagnation and the sudden increase of life expectancy in the Netherlands 1970-2009, Vienna Austria	2012	1.0
Measuring the relative progress of mortality improvement to evaluate stagnation and the sudden increase of life expectancy in the Netherlands 1970-2009, Stockholm, Sweden	2012	1.0
Projection of the survival and life expectancy in the Netherlands: the AG projection compared to two other Dutch projections, Investment and Risk Management for Pension Funds and Insurers, Amsterdam, The Netherlands	2012	1.0
The Relative Pace of Mortality Improvement, MGZ Research Meeting, Rotterdam, The Netherlands	2011	1.0
(Inter)national Conferences		
Annual Meeting of the Population Association in America, San Diego, USA	2015	0.5
Bio-demography and Multistate Event History Analysis on Healthy Aging, Beijing and Hangzhou, China	2013	0.5
European Population Conference, Stockholm, Sweden	2012	0.5
Determinants of Unusual and Differential Longevity, Vienna, Austria	2012	0.5

Seminars and Workshops		
Masterclass Dynamo HIA	2014	0.3
Social Epidemiology Methods Meeting	2014	0.3
GAMLSS in Action	2014	0.3
Index Development, Healthy Life Expectancy by Socio-economic Subgroup	2013	0.3
Bayesian Adaptive Methods for Clinical Trials	2012	0.3
Network for Studies on Pensions, Aging and Retirement (NETSPAR) Symposia:		
Target Pensions and Aging	2013	0.3
Mortality Dynamics and Their Economic Consequences	2013	0.3
Health Expenditures a Cause of Increasing Life Expectancy?	2012	0.3
Development of Life Expectancy and Consequences for Pension Claims	2012	0.3
Projecting Future Life Expectancy: An Interdisciplinary Perspective	2011	0.3

2. Teaching		
Supervison Master Thesis "Factors Influencing the Quality of Sleep in Germany"	2015	1.0
Medical Demography, University of Rostock, Rostock, Germany	2015	2.0
Introduction to Demography, University of Rostock, Rostock, Germany	2014/15	1.0