1-1-2016

What can the life course approach contribute to an understanding of longevity risk?

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Disciplines

Medicine and Health Sciences | Social and Behavioral Sciences

Publication Details

Blane, D., Akinwale, B., Landy, R., Matthews, K., Wahrendorf, M., Wahl, H., Hayward, M. D., Liefbroer, A. C., Mishra, G. D., Ferreira, I. & Koupil, I. (2016). What can the life course approach contribute to an understanding of longevity risk?. Longitudinal and Life Course Studies, 7 (2), 165-196.

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COMMENT AND DEBATE

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http://dx.doi.org/10.14301/llcs.v7i2.343

Abstract

Longevity risk means living longer than predicted. Attempts to understand longevity risk to date have concentrated on single diseases, usually coronary heart disease, and sought explanations in terms of risk factor change and medical innovation. In an opening paper, David Blane and colleagues point to evidence that suggests changes in positive health also should be considered; and that a life course approach can do so in a way that is socially and biologically plausible. Applying this approach to UK citizens currently aged 85 years suggests that life course research should give priority to trajectories across the whole life course and to the social and material contexts through which each cohort has passed. Testing these ideas will require inter-disciplinary and international comparative research. The opening paper is followed by commentaries from Hans-Werner Wahl, Mark Hayward, Aart Liefbroer and Gita Mishra. Finally Blane and colleagues respond to the points raised by the commentators.

Keywords

Longevity risk, positive health, social and biological plausibility, life course trajectories, social history context

Introduction

The increase in life expectancy at middle age was one of the defining characteristics of the late twentieth century, with all-cause mortality around the state pension age in England and Wales falling by some two thirds during 1971-2001 (Akinwale et al., 2011), and similar improvements occurring in many other countries (World Health Organisation, 2008).

The actuarial profession led research into the causes of this change. Their thinking includes the idea of the *golden cohort*, born in the 1930s, who were the first in the UK to experience the fall in middle aged mortality (Willets, 2004), and the concept of *longevity risk* to describe the phenomenon of living longer than predicted (Willets et al., 2004).

The rate of improvement in middle-aged mortality was not anticipated, with serious consequences for the financing of pension arrangements. In the UK, the market for annuities collapsed, employers closed defined benefit pension schemes for much of the private sector and the age at which the state pension is paid was increased first by five years for women, to be followed by a further increase of three years for all citizens.

Most of the actuarial and epidemiological research into the causes of this increasing longevity is disease-specific, usually coronary heart disease (Unal, Critchley & Capewell, 2005; Bajekal et al., 2013) or cerebrovascular disease (Raine et al., 2009); and assumed that the main determinants of the falling mortality rates would be either change in risk factors or the introduction of new medical treatments. Positive health, in the sense of growth and development, functional capacity, vitality and resilience, was largely ignored. The present paper attempts to rectify this neglect by arguing that positive health should be considered alongside risk factor change and medical innovation as driving the increase in life expectancy at middle age; and that the life course approach can contribute to the understanding of longevity risk by bringing both biological and social plausibility (Blane, Kelly-Irving, d'Errico, Bartley & Montgomery, 2013) to the idea of positive health.

Two pieces of evidence support the idea of positive health. Table 1 shows for England and Wales, the disease-specific mortality rates for the most prevalent causes of death, which together account for some two-thirds of all deaths. Between 1971 and 2001 their mortality rates tended to fall by proportionately similar amounts. The tendency was stronger for men than for women. Male mortality rates for all of these prevalent causes of death reduced by around 50-75%. For women, in contrast, the fall in mortality rates was in the 50-75% range for only five of the prevalent causes of death, while another reduced by around one-third and two increased (it is tempting to attribute the rise in women's COPD and lung cancer mortality to their tobacco smoking, although the fall in their CHD mortality, which is also tobacco-related, gives pause for thought).

Table 1. Age-standardised mortality rates per million population; England & Wales, 1971-2010; selected causes, by prevalence.

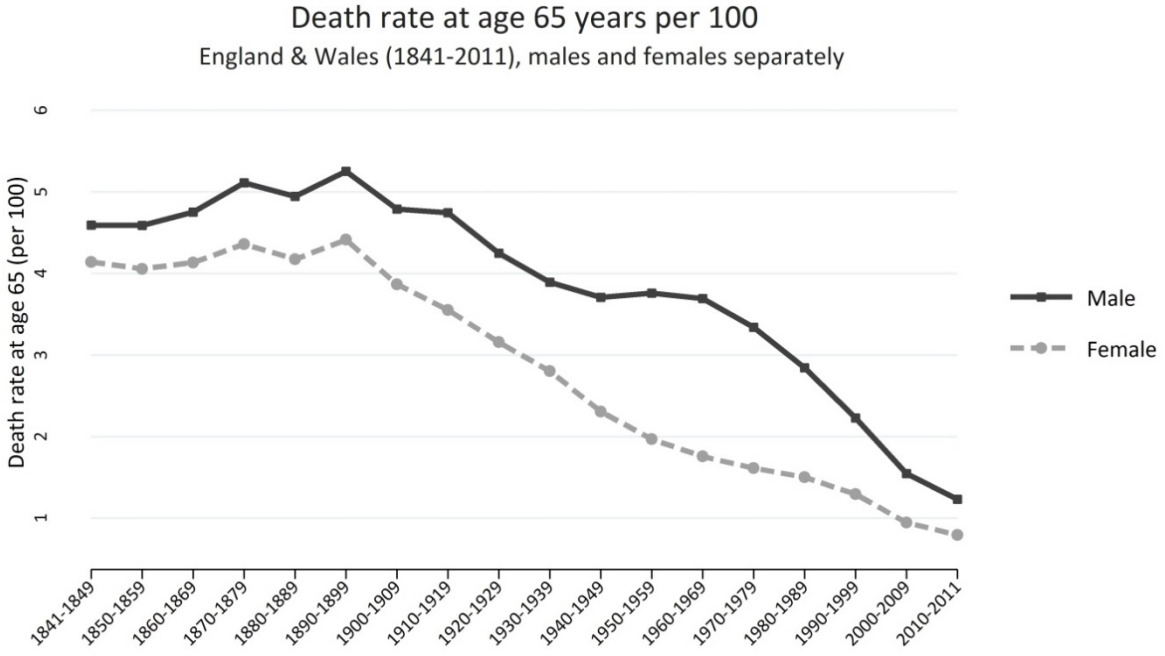
	Males			Females		
	1971	**2010**	**Percentage change 1971-2010**	**1971**	**2010**	**Percentage change 1971-2010**
Ischaemic heart disease	3,801	1,083	-71.5	1,668	478	-71.3
Cerebrovascular disease	1,541	422	-72.6	1,352	396	-70.7
Chronic obstructive pulmonary disease	944	314	-66.7	193	212	+9.8
Pneumonia	920	260	-71.7	624	212	-66.0
Carcinoma of lung	1,066	465	-56.4	183	299	+63.4
Accidents and violence	333	170	-48.9	166	53	-68.1
Carcinoma of breast	4	2	-50.0	379	245	-35.4
Carcinoma of stomach	317	72	-77.3	149	33	-77.9

Source: Office for National Statistics (2011).

Something similar happened in the late nineteenth and early twentieth centuries when mortality due to most of the prevalent infectious diseases of childhood fell at around the same time (McKeown & Lowe, 1966; McKeown, 1979), although in that instance the situation was simpler conceptually because infectious disease epidemiology already contained the idea of host resistance. Positive health can be seen as the chronic degenerative disease equivalent of host resistance.

Figure 1 shows the change in the death rates of people aged 65 years in England and Wales from the 1840s to 2010. The mortality rates of women and men changed little (if anything they increased) during the nineteenth century from 1841-1849 to 1891-1899. After 1900 women's mortality rate at age 65 years fell in an approximately linear fashion across the whole of the twentieth century. Men's mortality in contrast, after an initial fall, plateaued from 1931-39 to 1961-69 followed by a precipitous fall to near-equality with the women's rate by 2011.

Figure 1. Death rates of men and women at age65 years per 100 live population of that age in England and Wales 1841 to 2011



Source: Human Mortality Database.

The century-long, near linear fall in women's mortality may have received too little attention. It suggests a cumulative process driven by rising living standards, with little sign of any large impact from the introduction of antibiotics, the start of the National Health Service or the changing prevalence of tobacco smoking; also, it is easier to predict future developments, including the necessary level of pension contributions, from linear change. Prediction would have been more difficult from the change in men's mortality at age 65 years: first the three-decade plateau from the 1930s to the 1960s suggested stability rather than change, then the subsequent precipitous decline suggested rapid change. On one reading, it is the former that requires explanation (the plateau coincided with the introduction of antibiotics and widened access to, and sizeable investment in, medical care), with the latter a process of catching up for lost time.

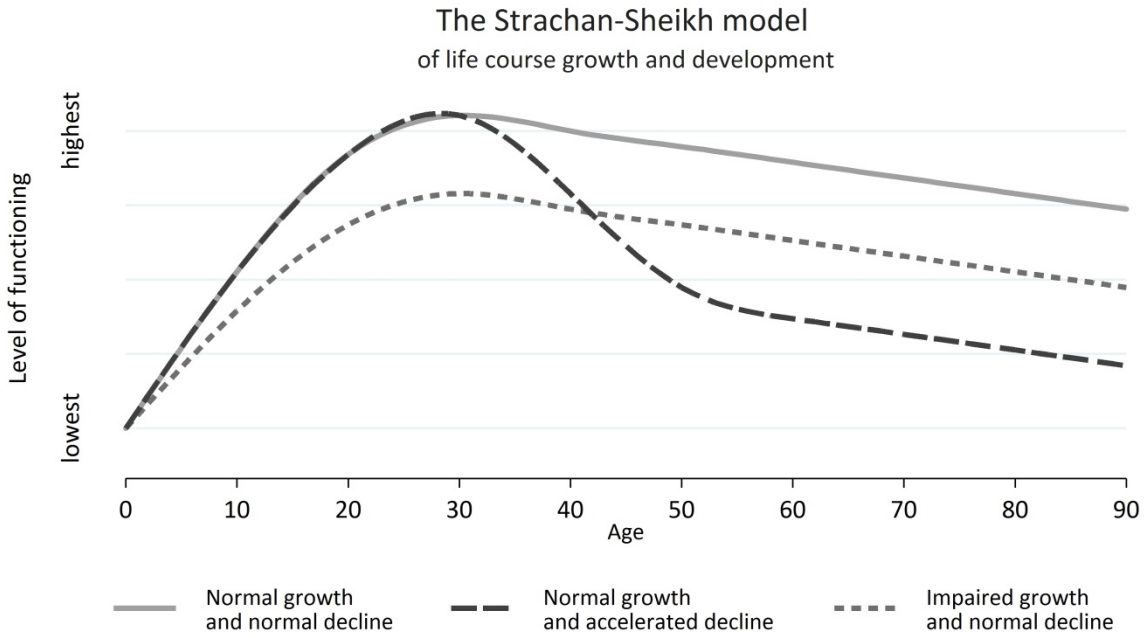
If it is accepted that risk factor change and medical innovation have difficulty, on their own, in accounting for the twentieth century's near linear fall in the mortality of 65 year old women and the proportionately similar fall in mortality from most of the prevalent causes of death, then it may be worth considering positive health as a third factor in the explanation of longevity risk. The next section of the present paper attempts to do so by examining the biological and social life course of the *golden cohort*, operationalised as those currently aged 85

years. In both cases, biological and social, the various life course stages of these 85-year-old persons will be located within their UK social history context.

Biological Life Course

Figure 2 presents a model of the biological life course. The model was described by Strachan and Sheikh (2004) in relation to lung function; here it is generalised to positive health.

Figure 2.



Source: based on Strachan and Sheikh (2004), own visualization.

First there is a phase of growth and development, during which an initial fertilised cell divides and replicates to an estimated 37.2 trillion cells by around age 20 years (Bianconi et al., 2003). Functional capacity subsequently is lost: slowly at first, when the loss is more than compensated by experience, anticipation and conditioned reflexes, as shown by professional footballers and Olympic track and field champions; then a more rapid decline to morbidity and eventual death (full line in figure 2).

Social and material circumstances can affect both the rate of growth and development and the rate of functional decline. Adverse circumstances can constrain growth and development to produce sub-optimal peak functioning, with the result that subsequently the normal rate of loss of function will still produce early death (dotted line in figure 2); for example, in Swedish linked register data childhood social disadvantage, in terms of parental social class and residential crowding, is associated with late adolescent sub-clinical abnormality (erythrocyte sedimentation rate, proteinuria, blood pressure, body mass index, vulnerability to stress) which, in turn, is associated with premature death in middle age (Sundin, Udumyan, Sjostrom & Montgomery, 2014; Bergh et al., 2014). Early death also results when normal growth and development is followed by adverse circumstances which accelerate the rate of functional loss (dashed line in figure 2); for example, when health selection into employment is followed by years of physically arduous and hazardous work (Costa & d'Errico, 2006). The social structure, with its tendency towards life course continuity of advantage or disadvantage (Goldthorpe, Llewellyn & Payne, 1980; Berney et al., 2000; Holland et al., 2000; Erikson & Goldthorpe, 1993), ensures that some people experience both sub-optimal growth and accelerated decline.

Applying figure 2 to a UK citizen currently aged 85 years shows that their period of growth and development would have occurred during 1928-1948 and their subsequent decline during 1949-2013, which latter period can be sub-divided usefully into working life (women 1949-1988; men 1949-1993) and retirement (women 1989-2013; men 1994-2013). The UK social history context in which each of these stages was lived will be examined next.

Growth and development 1928-48

This *golden cohort's* childhood was spent during the economic depression of the 1930s and their adolescence during World War II and its post-war austerity. What this meant in terms of childhood social circumstances varied by geographic region and social class. New light industries in the east midlands and south-east England brought employment and less hazardous work to some of the cohort's parents, although pay remained low and jobs insecure, together with new suburban housing and commuting via new transport infrastructure. The heavy industrial areas of north and west Britain, in contrast, experienced high unemployment, protracted poverty and poor housing (Stevenson, 1984). The high prevalence of poor diets among workers' children in the 1930s was documented by John Boyd Orr's careful studies of child health and nutrition (Harvey, 1955; Gunnell, 1996; Maynard et al., 2006) and motivated demands for welfare payments (family allowances) to cover some of the extra costs of children (Rathbone, 1924).

The war brought family disruption to the cohort's adolescence, as the result of the conscription of fathers and older siblings into the armed forces and the evacuation of urban children to rural areas, while bombing produced casualties and widespread damage to the housing stock and basic utilities like sewage. More positively, war-time full employment brought wages to mothers, which enabled them to buy their somewhat meagre rations of food, and jobs to the adolescents when they left education (Stevenson, 1984). The cohort completed its period of growth and development in post-war austerity, continued food rationing and a severe housing shortage. At the same time welfare state reforms gave hope for the future and, perhaps more importantly, the cohort was established in the labour market (Marwick, 1982).

Work and fertility 1949-1988/1993

Male members of the cohort ended their phase of growth and development conscripted into compulsory national service in UK armed forces, with its physical fitness, calorie-rich diet and heavily subsidised tobacco smoking. Female members of the cohort worked in light industry rather than domestic service, as formerly, and married in unprecedented numbers, producing fewer children despite rudimentary contraception (Coleman, 2000). The cohort raised their children in residential circumstances that, initially, were poor, often crowded, unhygienic and cold. That improved slowly during the following two decades, by which time bathrooms, indoor toilets, separate bedrooms for adults and adolescents, refrigerators and kitchen cookers were becoming universal, with telephones and washing machines already widespread (Office for Population Censuses & Surveys, 1973).

The slow but steady improvement in the residential circumstances of the cohort was funded by full employment and a labour shortage that drove rising real wages and immigration from mainland Europe, Ireland, the Caribbean and the Indian sub-continent. The rise in real wages was accompanied by the abolition of Saturday morning working as part of the standard working week and the spread of paid holidays. The combination of better-equipped kitchens, new ideas from holiday experience and more disposable income made possible better nutrition. The later years of the cohort's working lives, when the risk of the onset of chronic disease increases, coincided with de-industrialisation and the 1980s economic recession – although the cohort was protected to some extent by long-service priority access to less physically demanding jobs and, for those who lost their jobs, by pension schemes which allowed early retirement on grounds of poor health and, for manual workers, disability benefits rather than unemployment (Schuller, 1987).

Retirement 1989/1994-2013

The cohort's retirement from paid employment coincided with the emergence of the idea of the Third Age as a new stage of the life course situated between the end of responsibility for work and children and the onset of physical dependency; functionally healthy and in receipt of occupational or private second pensions, such Third Agers are seen as free to pursue their own self-realisation and

pleasure (Laslett, 1989). Morris's research into the minimum income for healthy living for retired people (Morris, Wilkinson, Dangour, Deeming & Fletcher, 2007) shows that many in the cohort will have been denied such a Third Age, particularly those afflicted by chronic disease (Patsios, 2014). However the existence of the idea indicates that the cohort's retirement is being lived in new circumstances, which includes new knowledge about early old age in terms of the nutrition and physical exercise required to maintain functioning and the ability of social participation to confer resilience to the adversities of ageing.

Social Life Course

The life course is a social as well as a biological phenomenon, so the foregoing biological perspective needs to be complemented by its social equivalent.

Bartley, Blane and Montgomery (1997) identified a series of key social transitions that are faced by most people as part of current social organisation; such transitions can set a person on a long-term trajectory towards advantage or disadvantage, depending on whether the transition is completed successfully. The sites of such transitions include education, family, work and ageing; and each needs to be seen in its cohort-specific social and material context.

The move from primary to secondary school and school examinations

This transition would have had little relevance for those currently aged 85 years because they reached the then minimum school leaving age of 14 years in 1942, when perhaps 86 per cent left school with no qualifications (Stevenson, 1984). University study was rare among this cohort: 2.6 per cent at the 1951 decennial census (Carr-Saunders, Caradog Jones & Moser, 1958) and disrupted by national conscription in UK armed forces.

Leaving parental home, establishing own home, parenthood

By the mid-1950s when the cohort was making the transition to their own home and family, the necessary residential accommodation was in short supply and often lacked basic amenities. As a result, many families were started in an in-law's home, in consequently crowded circumstances, lacking privacy.

Job insecurity, change and loss

The cohort mostly entered the labour market during the full employment of World War II and benefitted from the later labour shortage in terms of secure and rising incomes and the ease of finding alternative employment.

Onset of chronic illness and labour market exit

The cohort reached the age where chronic disease starts to become more prevalent during the high unemployment and de-industrialisation of the 1980s, when premature labour market exit was possible through early retirement on an occupational pension or, for those without an occupational pension, through permanent sickness on disability benefit.

In each case, those from an advantaged social class were more likely to complete successfully the transition faced, but for the cohort as a whole the later challenges occurred in more favourable circumstances than the earlier ones.

Discussion

The biological and social life courses point in the same direction: namely, that those currently aged 85 years in UK mostly did not have a particularly advantaged childhood and adolescence, while their adulthood and early old age were marked by steadily improving social and financial circumstances. These findings suggest that life course research might question its emphasis on early life, give more attention to adult circumstances and look at the continuities between these stages of life.

At least three objections to this line of reasoning need to be addressed. First, can these ideas be tested; can positive health be defined more precisely and can it be measured? Second, the increase in life expectancy at middle age has continued beyond the *golden cohort*, in circumstances where the balance between early and later life may be different. Third, the increase in life expectancy at middle age is a worldwide phenomenon; can a worldwide phenomenon be explained by events in one country (UK)?

Each of these objections suggests a new line of enquiry. The measurement of some aspects of positive health is long established, particularly in relation to the phase of growth and development. Formerly in the UK, the school medical service measured height and weight during childhood; the

national educational test measured cognitive function at age 11 years; and the army medical corps measured various aspects of the physical and psychological functioning of those conscripted into the armed forces at the end of adolescence. Apart from special surveys of population samples, comparable measures during the phase of functional decline are rare, particularly during its early stages, before overt morbidity; routine screening by occupational health departments and private health insurance companies are the main exceptions. Most research has concentrated on specific measures such as coronary heart disease risk, although measures of positive health more generally have been proposed, such as the concept of allostatic load (McEwen & Seeman, 1999) and its current development (Kelly-Irving et al., 2014). New initiatives in the area of positive health would be timely because of the wealth of biomedical data becoming available in large, representative social surveys, such as the Constances cohort (Zins et al., 2010) and the UK Household Longitudinal Study (Benzeval, Devillas, Kumari & Lynn, 2014).

The second and third objections require international comparative research to identify countries whose demographic and social history differs from those of the UK. Are there countries where the proportionate fall in mortality from the prevalent causes of death differed from that in England & Wales; and if so, what were the reasons for this difference? Are there countries where the increase in life expectancy at middle age started earlier or later than in Britain; and if so, do they show the same gender differences? Are there countries whose history reversed the UK life course sequence of childhood hardship followed by growing adult affluence; and if so, what was the associated change in life expectancy at middle age? Once again, these research questions are timely because of the creation of international demographic databases, such as that of Max Planck Institute in Rostock, collaborating with Rand Corporation in USA, and international bio-social surveys, such as the USA National Institute of Aging portfolio studies of ageing.

In summary, the present discussion paper has argued that: studies of longevity risk could usefully include the idea of positive health; that the life course perspective offers a way of doing so which is biologically and socially plausible; that it is instructive to set these life course stages in their cohort-specific social history context; and that doing so suggests a timely and progressive programme of research.

Acknowledgements

We owe a debt of gratitude to Madhavi Bajekal, Rosalind Raine and the members of the Institute and Faculty of Actuaries, particularly Joseph Lu, Brian Ridsdale and Richard Willetts, for raising our interest in longevity risk; and to UK Economic and Social Research Council who funded (Award No: ES-J019119/1) the *International Centre for Life Course Studies in Society and Health* (ICLS) of whose scientific programme the present paper is a part.

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Life-span developmental psychology and social-behavioural aging science: need for better liaisons with life course epidemiology in the future

Life-span developmental psychology and life course research in the sociology of ageing (see below for differences) have been established meta-perspectives in social-behavioural ageing science for some decades. However, such 'old' meta-perspectives face permanent challenges to re-invent themselves as a result of the continuing development of new theories and the emergence of new longitudinal data, as well as synergies arising from the interchange with other disciplinary views. I will use the Blane, Akinwale, Landy, Matthews, and Wahrendorf article (2016) as a springboard to take a closer look at what life-span developmental psychology and life course research has achieved so far, particularly in empirical but also in conceptual terms. At the same time, I will refer to this material to take a critical look at the Blane et al. (2016) work.

A glance at the history of life-span / life course views in social and behavioural ageing research

The insight that it might not be a good idea to split the human life course and concentrate, for example, only on early childhood or on old age is not new in developmental science. Groffman (1970) has provided a comprehensive account of European historical roots of life-span thinking in philosophy, culture and fictional literature. Examples of influential philosophical perspectives on ageing include: Francis Bacon in England (1561-1626; 'History of Life and Death'), Michel de Montaigne (1533-1592; 'Life Wisdom's Final End') and Johannes Nikolaus Tetens in Germany (1736 – 1807; 'Philosophical Treatise on Human Nature and its Development'). One should also mention Belgian mathematician and statistician Adolphe Quetelet (1835), who argued for the need to detect the laws of life-long development in his two-volume treatise 'Sur l'homme et le développement de ses facultés' ('On man and the development of his capabilities'), and Francis Galton from England, who undertook one of the first cognitive performance studies across a wide age span – from infancy to old age – during the International Health Exhibition held in London in 1885. Life-span oriented research also received a strong push by Austrian developmental psychologist Charlotte Bühler's (1933) widely acknowledged monograph 'Der menschliche Lebenslauf als psychologisches Problem' ('Human Life Course as a Psychological Problem').

More recently, particularly after gerontology's instalment as a science shortly after World War II, two separated traditions, operating with different names, have evolved in the sociology and the psychology of ageing. In sociology, the term *life course* has mostly been used and a central argument always has been that social forces largely determine the flow of life and define expectations about what should happen in a 'normal' life at a certain age (e.g., Settersten, 2003). Psychology preferred the term *life-span development* and predominantly concentrated on proximal and distal influences of a range of factors such as personality, cognitive functioning, or childhood trauma, in order to understand the dynamics of life-long, developmentally relevant processes as well as to predict late-life outcomes (e.g., Baltes et al., 2006). These two lines of research converged at times, but also searched to define themselves from each other across the decades (e.g., life-span researchers also use the cohort concept to understand historical changes in late-life cognitive functioning; sociologist have a tendency to accuse life-span researchers of under-rating the role of socioeconomic status (SES) across full lives). More recently, health research has significantly added to both life course as well as life-span research and it seems that epidemiology increasingly searches for the inclusion of distal antecedent factors, when it comes to the predictions of a range of endpoints such as diseases and functional limitations (e.g., Ben-Shlomo & Kuh, 2002; Kuh et al., 2014).

In conclusion, a life course / life-span view has existed for some time, when it comes to social-behavioural research on ageing and, to some extent, also in the area of health and aging research. I therefore find the framework proposed by Blane et al. to be less innovative when seen from

the perspective of social-behavioural aging science. I was also surprised that the relationship to the life course approach in the health and epidemiology area, e.g., as suggested by Kuh and colleagues (e.g., Kuh et al., 2014), was not described.

Important data and findings of life-span and life course research: a selective and exemplary view with a focus on health-related outcomes

Setting aside the more historically relevant distinction between life-span and life course research, a number of findings with importance for late-life health issues emerged in social-behavioural ageing research and some of the prototypical ones will be mentioned in what follows. First, in the emerging area of the critical role that differences in *cognitive functioning* seem to play for late-life outcomes ('cognitive epidemiology'; see Calvin et al., 2011), work by intelligence researcher Deary at al. has shown that cognitive functioning as measured in 11-year-old children reveals a very strong relationship with intelligence assessed 70 years later (Deary et al., 2004). In addition, Deary and colleagues (2004) also found, in line with a number of other studies (see also Schaefer et al., 2015), that being higher in intelligence in childhood not only predicts living longer even after controlling for a number of confounders, but also type 2 diabetes, heart disease, and dementia in late life.

Second, the linkage between *personality and late-life health outcomes* is complex but well established. In particular, being a high scorer in neuroticism seems to predict follow-up health events and longevity rather well (e.g., Mroczek et al, 2006). Importantly, inter-individual differences in personality traits tend to be stable after young adulthood until the end of life (Roberts & DelVeccio, 2000) and thus unfold their health-related impact on the full remaining life course.

Third, Blane et al. already consider the critical role of adverse childhood conditions for very late health-related outcomes to some extent in the article. However, there is much more. Caspi and colleagues in the UK have shown, in a range of studies, how important early childhood experiences are for later-life outcomes via abnormal adolescent behaviour which in reverse may impact on the rest of life, e.g., via depressed mood or reduced educational pathways (e.g., Caspi et al., 2002). His research also underscores significant interactions between expression of specific genes and early-life traumatic experiences, leading to different mental health outcomes including abnormal behaviours, which again may dramatically shape the remaining life course. Going further, Schafer and Ferraro (2012) have shown with data from the US targeting early-life conditions based on rather objectively assessed retrospective information, that early-life economic conditions do show substantial associations with successful versus less successful ('positive health') lifespan developmental trajectories including wellbeing in old age. Brandt et al. (2012) show similar findings based on data from a number of European countries. Emerging findings from other research teams also found similar relationships between adverse childhood contexts and late-life functional ability impairment, multimorbidity, and mortality (Pavela & Latham, 2015; van den Berg et al., 2006, 2009). Most of these associations remained statistically meaningful even after controlling for proximal health and SES conditions.

Fourth, the existence of *early health risk factors* seems to impact on the full rest of the life span. A relatively large and fairly consistent body of evidence from epidemiological studies now demonstrates that being overweight in childhood and adolescence has adverse consequences on premature mortality and physical morbidity in adulthood (Reilly & Kelly, 2011). Additionally, the clear majority of depressive illnesses have emerged before the age of 25 years; hence, and particularly given the resistance to treatment of depressive illness at large, late-life depression is to a large extent connected with depressive episodes that already appeared earlier in the life-span (Kessler et al., 2014). Similarly, low educational input, low physical activity level, cognitive inactivity, midlife overweight, diabetes, smoking and depression earlier in life show relatively strong associations with late-life dementia-related disorders (Barnes & Yaffe, 2011).

Fifth, two simple subjective evaluations assessed earlier in life (e.g., in midlife), i.e., *subjective health* and *subjective age*, show important relations with late-life health-related endpoints. Subjective health assessed in early life has been found to predict late-life morbidity and mortality even when objective health is controlled (Jylhä, 2009). Also, feeling younger and having a more positive attitude

towards own aging, when assessed in midlife, can predict late-life cardio-vascular disease, functional ability, dementia-related physiological changes in the brain, and mortality (e.g., Levy et al., 2002; see also meta-analysis by Westerhof et al., 2014). It may be argued here that feeling healthier as well as feeling younger may represent a rather powerful ways to resist the ageing process and thus indicate efficient forms of resilience as people age, starting its operation rather early in the life-span.

In summary, it seems rather clear that differences appearing in late-life health outcomes including longevity are, to a considerable extent, influenced by differences earlier and even very early in life – including in childhood. It is interesting too that Blane et al. do not feature this body of work, mostly coming from the social and behavioural aging science area, very much in their article. I interpret this as an indication that life-span / life course approaches still operate too much in silos, with each unaware of what has already been done in different disciplinary areas. This cross-disciplinary problem needs to be overcome, because the concepts focused on in the empirical work outlined above are all able to speak to the concept of positive health – a core construct that Blane et al. are using in their article.

Concept of positive health

The argument made by Blane et al. that traditional gero-epidemiology research targeting longevity (mortality) is too much focused on a disease and disease-risks perspective is important. Blane et al. (2016) rightly state: "positive health, in the sense of growth and development, functional capacity, vitality and resilience, was largely ignored." (p. 167). Later in their work, they also address issues of definition and assessment in the context of positive health. Indeed, it seems that disease-specific mortality rates are on the decline due to on-going medical progress and positive health may increasingly take over as a driving force of longevity in decades to come.

However, in social-behavioural ageing research, the term positive health would be unlikely to find much resonance and the immediate association would be the concept of *successful ageing*. Three influential concepts of successful ageing may be distinguished. First, Rowe and Kahn (1997), a geriatrician and a social psychologist, have suggested a concept of successful ageing based on three components: (1) low risk of disease and related disability; (2) high cognitive and physical functioning, and; (3) continued active engagement in life. All three of these components are substantially related to longevity and all have, as argued above, major sources in earlier life conditions. They indeed also have considerable overlap with the concept of positive health as described by Blane et al.

Second, the so-called *well-being paradox* has gained much attention in social-behavioural ageing research and we now know that ageing people have a full tool box at their disposal allowing them to maintain high levels of wellbeing, even in highly adverse conditions. Two such mechanisms are also echoed in the data as mentioned above, i.e. feeling subjectively healthy even when faced with severe morbidity and feeling younger even beyond the age of 80 years. In a sense, such processes may resemble what Blane et al. had in mind, when they referred to growth and development as well as vitality in their conceptualisation of positive health.

Third, resilience, also explicitly addressed by Blane et al., has generated much interest in social-behavioural ageing science since the 1990's (Staudinger & Greve, in press). As Staudinger and Greve (in press) argue, resilience should be seen as a ubiquitous phenomenon of ageing and not as the rare exception, although personality characteristics for example (see also data mentioned above) are likely of importance to foster resilience.

In the light of such conceptual ideas as well as the empirical work I have compiled in the previous section, I feel that the term 'positive health' has remained too vague in the Blane et al. article. We need a robust conceptual model that addresses what positive health may mean and issues such as (childhood) intelligence, psychological resilience, attitudes toward own aging, as well as personality traits (e.g., low neuroticism, but also high conscientiousness) must all be considered in such a model. There is also a strong need to develop such a model in historic-dynamic terms; hence, ongoing cohort changes, as just mentioned, must become part of such a conceptual approach. It even seems that using 'positive health' is too narrow a term, because important factors related to longevity risk seem not to belong in the health sphere.

Outlook

Both the life course orientation as well as the emphasis put on positive health in the work by Blane et al., suggest that great synergies would unfold if life course epidemiology and life-span / life course research in social-behavioural ageing research would move closer together. First, better links and more cooperation between these areas may widen the range of constructs to be considered to better understand longevity risk. An important part of such widening would, in my view, be the insight that factors beyond health play a significant role when it comes to efficient prediction models of longevity. Second, better connections may greatly enhance our empirical understanding of which kinds of mechanisms are operating and linking early and late-life outcomes. Third, the expected conceptual and empirical progress based on better links and more co-operation may lead to better targeting of critical conditions early in life with the potential / risk to have 'long-term' impact on the remaining life course and thus become relevant for for intervention and early preventive efforts.

In conclusion, the concept of positive health, enriched and differentiated with now empirically proven concepts anchored in social and behavioural aging science, may further the needed *Gestalt Switch* from a traditionally mostly disease-related view of longevity to a view that integrates health / disease and social-behavioural parameters important for human long-term development. In an optimistic scenario, this may also open our eyes to new public health and preventive approaches that, to a large extent, may operate early in life, for example in kindergarten and the early school years. Hence, it may become increasingly true in the future that old age outcomes as well as end of life dynamics are linked with occurrences early in life and possibly even at the pre-natal stage of development.

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The paper by Blane, Akinwale, Landy, Matthews and Wahrendorf provides a thoughtful and nuanced discussion of the value of the life course framework for understanding the origins of adult health and mortality. Most highly-prevalent adult health problems in the UK and other high income countries are a long time in the making and reflect exposures stretching from the prenatal environment and childhood, to adolescence, and adulthood. Because the social conditions, institutions, and stratification systems of societies are powerful forces that shape the nature of individuals' life courses, adult health is fundamentally a reflection of these *lifetime* social forces.

As Blane and his colleagues observe in their review of the historical conditions surrounding the life course of the UK's *golden cohort*, social conditions, institutions and stratification systems are far from static. Over the course of the 20th century, for example, the social capacity for health in many countries, particularly high income countries, has dramatically improved through technological innovation, dramatic growth in biomedical knowledge and improvements in social institutional resources (Easterlin, 1997). These societal forces have had rippling consequences for changes in the nature and timing of life course experiences for cohorts born at different points in history. In turn, changes in the nature and timing of life course experiences have led to a host of changes in population health. Conditions once important in defining the health of the population have waned (e.g., the long-run decline in heart disease or the near eradication of polio in most high income countries), while other conditions have become more prevalent (e.g., some cancers). The concept of the birth cohort is critical in understanding these trends, due to cohort differences in the nature and timing of exposures. For example, given the role of childhood vaccinations in improving child health and survival in the 20th century in the United States (Centers for Disease Control and Prevention, 1999; Andre et al., 2008), combined with the dramatic decline in adult smoking (Fenelon and Preston, 2012), more recent American birth cohorts have experienced fewer and less exposure to *lifetime* health risks than earlier cohorts, contributing in important ways to historical declines in US adult mortality (Yang, 2008). Thus, the health of birth cohorts, often reflected in age-specific trends in health, may change due to fundamental shifts in the nature and timing of life course experiences. A key issue that is implicit in the discussion by Blane and colleagues is that life course influences on adult health are largely endogenous to the historical context. I suggest that we are only at the initial stages of understanding how life course influences on health are being transformed by changes in the social capacity for health. Their call for comparative research is an important component in furthering this agenda.

Despite growing agreement over this conceptual framework, Blane and his colleagues alert us to the fact that applying this framework to adult health outcomes comes with important challenges. Three major challenges that I discuss below are the following. First, how ought researchers to define health? I argue that we should consider a 'portfolio' of health outcomes encompassing biological risk, morbidity, functioning and disability, and mortality. A second challenge is that conceptual frameworks of social factors influencing health are often not biologically informed. I agree with Blane and colleagues that it is critical to integrate biological and social life course frameworks in order to understand how life course exposures from childhood into adulthood shape health trends and disparities through both developmental and aging processes. Finally, Blane and colleagues argue for the importance of incorporating historical context in understanding trends and differences in the life course pathways leading to adult health problems. Dramatic changes have occurred across current birth cohorts represented in the adult population in high-income countries in their prenatal, childhood and adult exposures, yet these changes are rarely central in life course studies of health. The development of conceptual models requires sensitivity to the fact that stratification systems, institutions, social conditions, technology, and even the epidemiological environment are changing and

differ across countries, all of which have implications for tackling a comparative research agenda.

Challenge one

Only recently have researchers begun to take a more integrative view of adult health and examined how life course experiences influence multiple facets of health (e.g., how life course factors influence the interplay of functioning and mortality to determine healthy life expectancy (Montez & Hayward, 2014)). Research rarely explicitly addresses the idea that exposures may give rise to a cascade of adult health conditions starting, say, from morbidity, to disability, and then to mortality – or not. All too often, research assumes that such a cascade exists, it gives rise to more 'endogenous' health outcomes such as disability and mortality, and is unidirectional – despite strong cautions about these assumptions (Verbrugge & Jette, 1994).

Health at the population level is a multidimensional concept. Increasingly, definitions of health acknowledge the core domains of physiological dysregulation (e.g., metabolic functioning), conditions (e.g., disease conditions such as diabetes), functioning (e.g., physical and cognitive deficits) and important facets of wellbeing and health potential (e.g., ability to live independently). The measurement of these domains is complex as are the relationships among these domains. Physiological dysregulation, disease conditions, functioning loss, and frailty are all parts of the process of health change that can – *but need not* -- precede death (Crimmins, Kim & Vasunilashorn, 2010; Martin, Schoeni & Andreski, 2010; Crimmins & Beltrán-Sánchez, 2011). The importance of understanding these connections stems from the fact that trends in these domains need not move in the same direction, and social group differences may vary, depending on the domain of interest.

Given these associations between health domains, life course factors need not affect all parts of the process in the same way. Some life course influences may be germane only to certain parts of the process of health change. For example, it is plausible that some childhood health problems (e.g., infectious conditions that heighten inflammation) give rise to adult morbidity conditions (e.g., coronary heart disease) that results in a cascade of increased risks of functional problems and mortality from cardiovascular disease (CVD). In this hypothetical example, childhood health problems' association with CVD-related mortality stems primarily from increasing disease incidence. In contrast, other life course factors may come into play throughout the process. Educational attainment, for example, not only is associated with a lower risk of heart disease but potentially it also comes into play in differentiating the risks of functional problems and mortality *among* persons with heart disease through human agency and financial resources (Mirowsky & Ross, 2003). In this example, a major social resource acquired relatively early in life – educational attainment – may accentuate disparities throughout the process of health change (Manton, Stallard & Corder, 1997; Merkin, Karlamangla, Crimmins, Hayward, & Seeman, 2009).

Challenge two

Blane and colleagues offer a biological, developmental framework of life course health (see figure 2 in their article). The core idea is that processes of the major biological systems (e.g., endocrine, immune, neurological, respiratory) display a similar pattern of development – a steady curvilinear growth in functional capacity during childhood followed by maintenance and eventually some decline in adulthood (Halfon & Hochstein, 2002). Figure 2's conceptual framework conveys a number of important ideas to keep in mind when considering the factors that come into play in understanding differences/disparities in adult health. First, differences in capacity begin as early as *in utero* and are evident at birth. This speaks to the seminal work by Barker and his colleagues (Barker, 1997; Barker, 1998; Barker, 1999; Barker, 2004). Second, childhood is a period of growth in capacity, and differences in capacity widen because of differences in inputs throughout childhood (e.g., SES, diet, family relationships). This is also the period in which cohort morbidity phenotypes (e.g., lifelong health risks that accrue through early life exposures such as infection and inflammation) are established (Finch & Crimmins, 2004; Crimmins & Finch, 2006). Although Blane and colleagues note that the maximum level of growth is a product of the level of inputs, I would add that the period of growth (and different peak

ages of capacity) might also be highly dependent on the level of inputs. In this sense, both the length and degree of development are highly malleable to inputs (Finch & Crimmins, 2004). Third, declines in capacity can start at different ages and exhibit varying rates of decline. Resources such as quality jobs, marriages and the avoidance of disease result in slow rates of decline, while the lack of resources and risk factors (e.g., smoking) contribute to relatively accelerated rates of decline. Indeed, it is possible that despite early life developmental advantages, adult risk factors and adverse conditions can result in rates of decline that negate earlier advantage. The combination of lifetime gains and losses results in growing heterogeneity of capacity in the population across most of the adult life course – the period of life where losses occur in capacity.

This biologically informed conceptual work of life course health presented by Blane and colleagues points to the need for researchers to attend to the combinations of lifetime exposures that put adults at greater or lesser risk of various health outcomes. In addition, this idea suggests that, depending on the inputs over a lifetime, the *balance* of childhood and adult influences on health disparities can change. This is a very important idea in that the developmental 'origins' of adult health are highly dynamic and mutable to a variety of inputs across the lifetime. No phase is inherently more important than another in terms of influencing adult health disparities. That said, as Blane and colleagues aptly note, life course stratification processes often reinforce childhood advantages and disadvantages in adulthood, compounding the effects of childhood. There is no 'lottery' after childhood that randomly assigns persons to adult trajectories of resources and risks. Moreover, some combinations of life course exposures are likely to be relatively common while others are quite rare due to life course stratification processes and social change. The malleability of life course trajectories in capacity has significant implications for understanding social group differences and trends in the life course origins of adult health – see *Challenge three* below.

Challenge three

Not surprisingly given the constraints of available data, much of the research on the life course influences on adult health has occurred in the context of single birth cohorts or a relatively narrow band of birth cohorts, e.g., the UK *golden cohort* described by Blane and colleagues, the British cohort studies and the US Health and Retirement Study. Thus, much of the empirical evidence about the life course origins of adult health is necessarily framed by the experiences of individuals in these studies. With the explosion of international studies built on the life course framework of the Health and Retirement Study (including the Study on Global Ageing and Health (SAGE)), we now have the opportunity to respond to Blane and colleagues' call for comparative research – and on a global scale.

Blane and his colleagues make a strong case that researchers need to be highly sensitive to the specific historical conditions that characterised the cohorts' experiences at particular ages in the life course. Stratification systems (and their metrics) may change across cohorts (and differ across countries). For example, occupation was more strongly tied to resources that garnered health advantages in the early part of the 20th century in the US compared to educational attainment (Preston & Haines, 1991; Hayward & Gorman, 2004). However, educational attainment, particularly advanced education, has grown in importance for reducing mortality in the US in the last half of the 20th century (Montez, Hummer, Hayward, Woo & Rogers, 2011; Masters, Hummer & Powers, 2012; Hayward, Hummer & Sasson, 2015). The 20th century in the United States also was a period of large reductions in infectious disease exposure and rising obesity. This period was characterised first by massive increases in post-secondary educational institutions and the prevalence of post-secondary education after World War II. This trend was followed in the later part of the century by the stalling of post-secondary attainment and the rise in student load debt. These are only a few examples but they raise important questions about how researchers should measure key concepts relating to the types of inputs over cohorts' lifetimes (e.g., childhood health, socioeconomic resources), and they point to some of the difficulties of carrying out a comparative research agenda.

Ryder noted 50 years ago that "the principal motor of contemporary social change is technological innovation" (Ryder, 1965).

Technological change is fundamentally important for the level of social capacity for population health (Easterlin, 1997). It is embedded in social institutions and defines the stock of knowledge and institutional resources that individuals in the population have access to and can act on to garner health advantages. The idea is similar to Fogel's concept of techno-physio evolution (Fogel & Costa, 1997; Fogel, 2004) which reflects the synergistic association between technological and physiological improvements in the modern era. Although technological change is sometimes thought to be felt most strongly by persons about to make 'lifelong' choices (Ryder, 1965), the developmental trajectories shown in Blane et al.'s figure 2 illustrate that technological change may have implications for changes in capacity at all stages of the life course. The pace of technological change is an issue that is rarely considered in life course studies of heath, yet this phenomenon has enormous implications for understanding how the inputs to the growth and decline in capacity ultimately influence adult health (Goldin & Katz, 2009; Palloni & Souza, 2013; McEniry, 2014). The pace of technological change in combination with early life exposures relates strongly to the heterogeneity of lifetime inputs to the gain and loss of physical capacity shown in figure 2.

An important tenet in life course conceptual frameworks is the importance of historical context. Here, I have argued that the historical context is not only relevant to a given cohort but it matters a great deal for the pace and nature of changes in the social capacity for health. In addition, current surviving birth cohorts in high-income countries are likely to differ in important ways in their lifetime exposures to important features of the social capacity for population health. The types of exposures and the pace of change in social capacity necessarily will shape how birth cohorts' capacities are changed over the lifetimes, ultimately influencing trends and disparities in the major domains of population health. The importance of broader societal forces for health can thus best be appreciated through studies that include a range of cohorts and that analyse the data with sensitivity to between-cohort differences.

Conclusions

The article by Blane and his colleagues adds to a burgeoning literature on the importance of life course conceptual frameworks for understanding adult health and mortality. As is evident in their discussion, a life course approach is fundamentally interdisciplinary and encompasses disciplines that are key in understanding how health is shaped from 'cells to society'. Epidemiology, biology, sociology, economics, genetics, psychology, history, …, – all these fields and more are part of a growing understanding of the life course origins of adult health and mortality. The emergence of this highly interdisciplinary approach reflects the enormously complex web of forces by which population health is shaped and changed.

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What can the life course approach contribute to an understanding of longevity risk? – a demographer's comment

The paper by Blane et al. makes a strong case for more attention to the life-course perspective in order to better understand seminal trends in mortality and longevity. Given my own background in the sociology and demography of the life course, it is hard to disagree with the authors on this issue, so I will restrict myself to providing some brief reflections on a few issues of key demographic or sociological importance.

The paper starts with the notion of 'longevity risk'. This is terminology that 'only' actuaries can use, as the costs to insurance companies go up if people live longer than expected. Most people would not want to view longevity mainly as a risk, but rather as an accomplishment. It also conflicts with the terminology of demographers, who speak of risk (or 'rate') as the chance that an event will happen (rather than the event not happening), and view increased longevity as the outcome of a process in which mortality risks (at specific ages, for specific cohorts, in specific periods) have fallen.

A key issue in the Blane et al. paper is how to understand the strong increase in longevity. Female life expectancy has increased by three months per year for more than a century (Oepen & Vaupel, 2002). In 2012 female life expectancy at birth was highest in Japan at 87 years, and male life expectancy at birth was highest in Iceland at 82 years (World Health Organisation, 2014). Blane et al. argue that this is not just the result of improvements in treating specific diseases, but largely of general health improvements. They illustrate this with period figures (the trend in mortality rates among 65-year-old men and women) and with a description of the *golden cohort* (born in 1928). But it is clear that health and mortality differences between age groups in a population, or across time among the same age group, could depend on one of three factors. They could be age-related, with increasing mortality rates as people grow older. They could be cohort-related, with decreasing mortality rates among younger cohorts. Or they could be period related with lower mortality rates for all cohorts and at all ages in more recent years. Often, it will be mix of these three factors. However, given that the three elements of this classic APC (Age-Period-Cohort) dilemma are dependent (someone's current age equals current period minus his or her birth cohort), it is hard to disentangle these elements and to determine whether changes in any outcome are influenced by age, period, cohort, or a combination of them. Statistical models are developed that try to solve this dilemma, but without a consensus about the best approach (Bell & Jones, 2014). An interesting contribution from demography to understand this issue is the so-called Lexis-surface. Recently, Minton (2014) provided a useful illustration of its application to mortality, shown here in figure 1. It shows mortality rates of Norwegian males, with year (period) on the X-axis and age on the Y-axis. Birth cohort can be followed drawing a 45-degree diagonal from lower left to upper right. Different colours signify different levels of mortality. Age effects are evident from the increase in mortality rates across the Y-axis. Period shifts are visible as mortality rates go up or down if one advances along the X-axis. Clear, 'pure' period effects can, for instance, be observed during the first and second world wars. A cohort shift is evident, if mortality rates to the left of a cohort diagonal would strongly differ from those to the right. In the example, we see a strong decrease in mortality rates from the 1930s onwards (briefly interspersed by World War II). The contour lines of all mortality levels retreat to higher ages, the closer we get to the current period. In the more recent period these lines have an upward incline, suggesting that they are a mix of period effects (affecting virtually all age groups) and cohort effects (with some cohorts profiting more than others). Thus, the Lexis-surface can be a useful tool to understand the dynamics of mortality decline and longevity increase, which could lead to hypotheses that could be more formally tested in a next step.

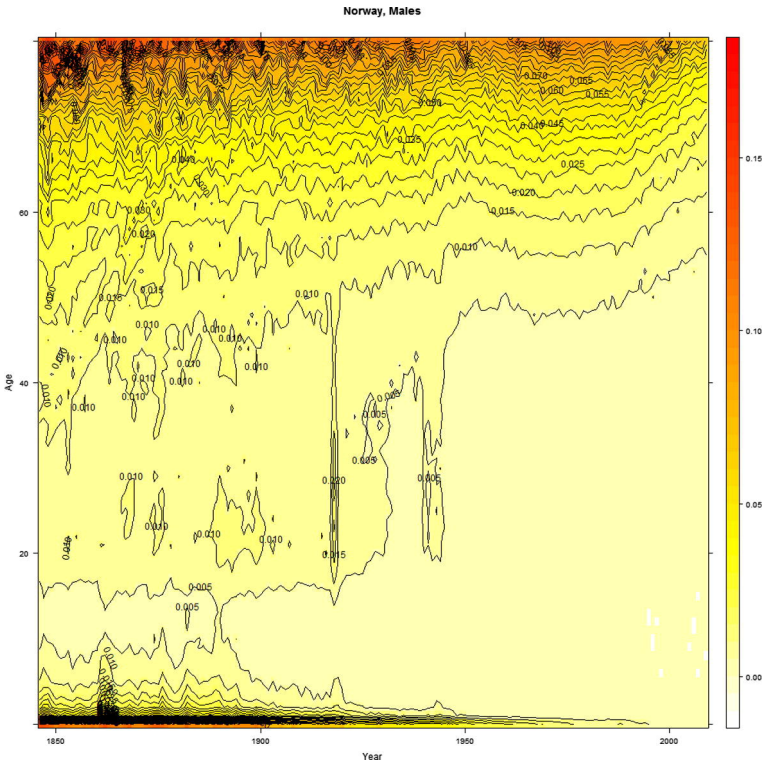

Figure 1. Shaded contour plot of mortality surface, Norway, males (Minton, 2014, Figure 7)

In order to understand the changes in 'positive' health, Blane et al. emphasise the importance of examining the long-term consequences of earlier life events for health later in life. This is not a new suggestion, as David Barker (1990, 1995) has already alerted epidemiologists to the relevance of studying conditions during pregnancy for later development. What is important in the paper by Blane et al., is their emphasis on multiple critical periods in people's lives that could influence their later development. This starts with early childhood where establishing secure attachment with key caregivers is of utmost importance (Bowlby, 1988). But Blane et al. suggest other critical periods later in life, such as the transition from youth to adulthood and retirement, as well. Fortunately, the basic ideas of such a life course approach already have strong supporters within epidemiology (Power, Kuh, & Morton, 2013). A key challenge, however, is to understand what aspects of life course change matter. What are the key life domains to take into account? Is it the experience of certain events, the time at which these events are experienced, or their ordering that matters? And how do events in different life domains interact? An important recent development in demographic and sociological applications of the life course approach, in this respect, is the growing popularity of sequence analysis (Abbott & Tsay, 2000; Elzinga & Liefbroer, 2007; Aisenbrey & Fasang, 2010). Within sequence analysis, a holistic approach to understanding the life course is advocated in which the effects of occurrence, timing and sequencing of events are all taken into account to develop a typology of life course trajectories that optimally takes the heterogeneity in people's life courses into account. The resulting typology can be used both to study the precursors (including health related ones) of specific life course patterns, and to study the consequences (again, including health related ones) of these patterns. For the promise of a life course approach to be realised, though, it is imperative that data collections within epidemiology pay more attention to life course trajectories in the health domain, as well as to those in the family and career domains. For this to come about, closer collaborations between epidemiologists and other health researchers on the one hand, and social scientists (in the broadest sense) on the other hand, have to be established.

A final plea made by Blane et al. is for more comparative research. I could not agree more.

Comparison is the essence of science. Often we compare social groups or categories within a specific spatial location (usually a specific nation state). But country comparisons could help us test whether our results are location specific (and thus conditional on the environment) or not. In addition, it can alert us to factors that might explain these spatial differences. Unfortunately, many cross-national social surveys only include relatively crude health related measures, whereas international medical data collections are not only very rare, but often have very little information on social aspects. However, there are a few exceptions (or at least examples to follow-up). The GGS (Generations & Gender Survey – a panel survey among the whole adult population with data collected in nineteen countries) includes a number of general questions on physical and mental health (see www.ggp-i.org). SHARE (Survey of Health, Ageing and Retirement in Europe – a panel survey among the population aged 50 years and over with data being collected in 20 countries) also includes health related questions and has even moved into collecting biomarkers (see www.share-project.org). These studies are designed by social scientists, but could certainly profit from collaboration with and involvement from epidemiologists and other health scientists.

To conclude, I fully agree with the plea by Blane et al. for more attention to the life course paradigm to better understand developments in longevity and in health more generally. What it takes, though, is better data, but, above all, collaboration between specialists from different fields. Only in that way can we turn our search for understanding the social underpinning of health risks into a truly interdisciplinary endeavour.

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Commentary by **Gita D. Mishra** University of Queensland, Australia
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Longevity risk describes the phenomenon of living longer than predicted (Willets et al., 2004). As Blane and colleagues explain, the concept was developed by the actuarial profession as a response to the unexpected decline in middle aged mortality during the late 20th century that placed sudden financial strain on insurance and pension funds in the UK and elsewhere. It may also illustrate what is known as the *black swan event:* a surprise, with a major impact, that is rationalised by hindsight as if it had been expected (Taleb, 2007). Blane and colleagues posit that to find the key driving factor for this increased life expectancy at middle age, one needs to look beyond changes in disease risk factors, morbidity, and medical innovations to improvements in *positive health* in the sense of "growth and development, functional capacity, vitality and resilience". Second, they then draw on the UK social history of a *golden cohort,* men and women currently aged 85 years, to argue that a life course approach is needed to identify plausible biological and social pathways that lead to these changes in positive health.

We agree that positive health appears a largely neglected concept in actuarial and epidemiological research. Seligman et al., (2015) is one of the few to set out a detailed description of positive health as "well-being beyond the mere absence of disease" and as the study of empirically measured *health assets* and their configuration in an individual. In this sense, a health asset refers to a factor that "produces longer life, lower morbidity, lower health care expenditure, better prognosis when illness does strike, and/or higher quality of life" (Seligman et al., 2015). This concept is similar to that for mental health whereby it is no longer considered the same as the absence of mental illness, but consists of a "measurable configuration of positive emotions, engagement, good relationships, meaning, and accomplishment" (Seligman 2011).

In contrast, life course epidemiology is a well-established approach and defined as "the study of long term effects on later health or disease risk of physical or social exposures during gestation, childhood, adolescence, young adulthood, and later adult life" (Kuh, Ben-Shlomo, Lynch, Hallqvist & Power, 2003). Its aim "is to elucidate the biological, behavioural, and psychosocial processes that operate across an individual's life course, or across generations, to influence the development of disease risk" (Kuh et al,, 2003). Thus at the heart of life course research there is an emphasis on statistical models that reflect basic hypotheses on the role of timing or duration of exposures (Mishra et al., 2009; Mishra, Chiesa, Goodman, De Stavola & Koupil, 2013).

Blane and colleagues draw on two examples of evidence to support the additional role of positive health, beyond that just from a reduction in risk factors and medical innovations. Here we focus on their second example: time series data for the mortality at age 65 years in the UK that shows a near linear decline for women since 1900. By comparison, mortality for men at age 65 years is higher than for women but has a slightly larger overall decline over the same time period. It includes a plateau in mortality rates for the three decades after about 1930 that were followed by a steeper "catch-up" decline since about 1960. The authors highlight that the near linear decline in mortality rates for women at age 65 years fails to exhibit signs of "the introduction of antibiotics, the start of the National Health Service, or the changing prevalence of tobacco smoking". Instead, they ascribe the decline to "a cumulative process driven by rising living standards" and thereby consequent improvements in positive health. Improvement in living standards, however, may act as a proxy or coincide with changes in a range of public health related exposures, including improvements in childhood nutrition and environmental exposures such as air quality. Therefore we should not expect too much from aggregated statistics for mortality rates in later life to reveal signals that unravel or distinguish various mechanisms that may be operating across the life course.

The integration of both social and biological pathways provides new insights into aetiology of health and disease but brings about new challenges in our views on generalisability of findings across

historical time, geographical regions or social strata of a society. Although findings demonstrating a large degree of consistency in early life determinants of social trajectories and later health have recently emerged, for example, from research on several generations of Swedish men and women (Goodman, Gisselmann & Koupil, 2010), the interpretation of these effects within societies that dramatically change over time is not straightforward. Would consistency in social pathways indicate a failure of our social and public health policies to break the chains of social and biological risks that affect subsequent generations? Or would it perhaps indicate a change in the main underlying pathophysiological (biological) mechanisms?

In spite of the dramatic changes experienced across the life course by the *golden generation*, it is their uniqueness in terms of biological and social history that arguably limits their explanatory role here. The timespan covered by the decline in mortality rates at 65 years applies to people born many decades both prior and after that generation. Each of these other cohorts, especially younger men and women born in the decades since 1940, would need to have an equivalent biological and social history with additional benefits on positive health in each decade. Further, longevity risk appears to have been an international phenomenon, so explanations are needed both over time and across populations. Yet without data from longitudinal studies, such accounts – including those from the context of UK social history provided by Blane and colleagues on the *golden generation* – would still be prone to ecological fallacies because they do not rely on empirical data of biological and social variables obtained at the individual level throughout the years.

Since its inception, the Newcastle 85+ study used by Blane and colleagues to illustrate the *golden generation,* has provided valuable information about their physical, metabolic, immune and cognitive function phenotypes (Collerton et al., 2007; Harrison et al., 2015; Motta et al., 2005; Terry, Sebastiani, Andersen & Perls, 2008; Vacante et al., 2012; Willcox et al., 2008). Yet by their design, this and numerous other studies conducted worldwide among the oldest old are limited in their contribution to the understanding of the life course trajectories that explain the longevity of their participants. Not only their validity may be compromised due to survival bias, but also any information on past exposures and experiences, either objective or subjective, can only, by definition be obtained retrospectively, with all the well-known limitations of that method. Current longitudinal and life course data analysis methodologies provide a means to handle the problem of the survival bias (e.g., inverse probability of attrition weights) (Weuve et al., 2012) but no such remedies can be developed to overcome the problems of retrospective data collection.

It remains, however, that healthy octogenarians and even more so healthy centenarians or even 'supercentenarians', can be seen as living proof of successful ageing (Motta et al., 2005; Terry et al., 2008; Vacante et al., 2012; Willcox et al., 2008), and positive health may prove to be a valuable framework to understand the explanatory factors at work. Attempts have been made to understand which life course trajectories led to the observed exceptional longevity, for instance, using the '*compression of morbidity*' hypothesis, that posits that in order to achieve extreme old age individuals markedly *delay* or even *escape* diseases that would otherwise be lethal at younger ages (Evert, Lawler, Bogan & Perls, 2003). Based on retrospective lifetime data on diagnoses of 10 major illnesses (i.e., hypertension, heart disease, diabetes, stroke, non-skin cancer, osteoporosis, thyroid condition, Parkinson's disease, and chronic obstructive disease) and one ocular disease (cataracts) collected by means of health questionnaires completed by >400 centenarians (or their proxies), the authors reported that three morbidity profiles fitted their study population: *survivors*, *delayers* and *escapers (*Evert et al., 2003). Interestingly, the identification of these distinct phenotypes led them to conclude that the exceptional longevity of centenarians may be achieved by multiple pathways and that there may be sex differences in these.

Blane and colleagues acknowledge the need to look for evidence with other direct indicators of positive health. Seligman et al., (2015) specifically suggest a re-analysis of existing longitudinal datasets shifting from the traditional focus on '*what goes wrong in life*' to the study of *health assets*, including subjective ones, as the main instruments to operationalize *Positive Health.* Some examples of biological markers of positive health as proposed by

Seligman et al., (2015) are low blood pressure (BP), low body mass index (BMI) and high cardiorespiratory fitness (VO_2max). Some on-going longitudinal studies have examined these variables throughout the life course of individuals. For instance, using a life course approach of repeated data obtained between the ages of 12 and 36 years (8 repeated measures), the Amsterdam Growth and Health Longitudinal Study (AGAHLS) has shown that 36-year olds with a healthy arterial ageing phenotype (i.e., younger than that that could be expected on the basis of their chronological age) (Ferreira & van de Laar, 2015) and metabolic profiles (Ferreira, Twisk, van Mechelen, Kemper & Stehouwer, 2005) were characterised by less steep increases in BP, BMI and VO_2max from adolescence to adulthood, with levels of BP and BMI never exceeding those cut-off values conventionally placing individuals at higher risk for metabolic and cardiovascular diseases (Ferreira, van de Laar, Prins, Twisk & Stehouwer, 2012). In addition, the AGHALS has pin-pointed adolescence and transition from adolescence to young adulthood as critical periods when increases in the levels of BP and BMI or decreases in VO_2max may no longer be reversible in some individuals.

The development of life course epidemiology and the increasing interest in potential critical or sensitive periods have also stimulated a wider use of developmental and growth indicators for monitoring of health at younger ages (Maggi, Irwin, Siddiqi & Hertzman, 2010) and ideally, one could use these as indicators of positive health early in life to construct more continuous life course trajectories of positive health, rather than rely on measurements taken at a later stages of the life course which would be already influenced by a rate of decline.

Aspects of self-report health such as SF-36 physical functioning scores, may provide another measure of positive health (Seligman et al., 2015; Ware, Snow, Kosinski & Gandek, 1993). With progressive improvements in positive health, one might expect higher scores at the same age with each subsequent generation, and each with its own distinct trajectory. The Australian Longitudinal Study on Women's Health has studied over 45,000 women in three age cohorts (born in 1921-26, 1946-51, and 1973-78) and has been running since 1996 (Dobson et al., 2015). Although the age ranges do not yet overlap to provide a definitive answer, each cohort does show a higher set of scores for physical functioning for younger generations (figure 1), but the overall impression can still be interpreted as a consistent connected trajectory across life (and across these generations of women), rather than three distinct and progressively higher trajectories. With further data in coming years the character of this life course trajectory will be more clearly resolved.

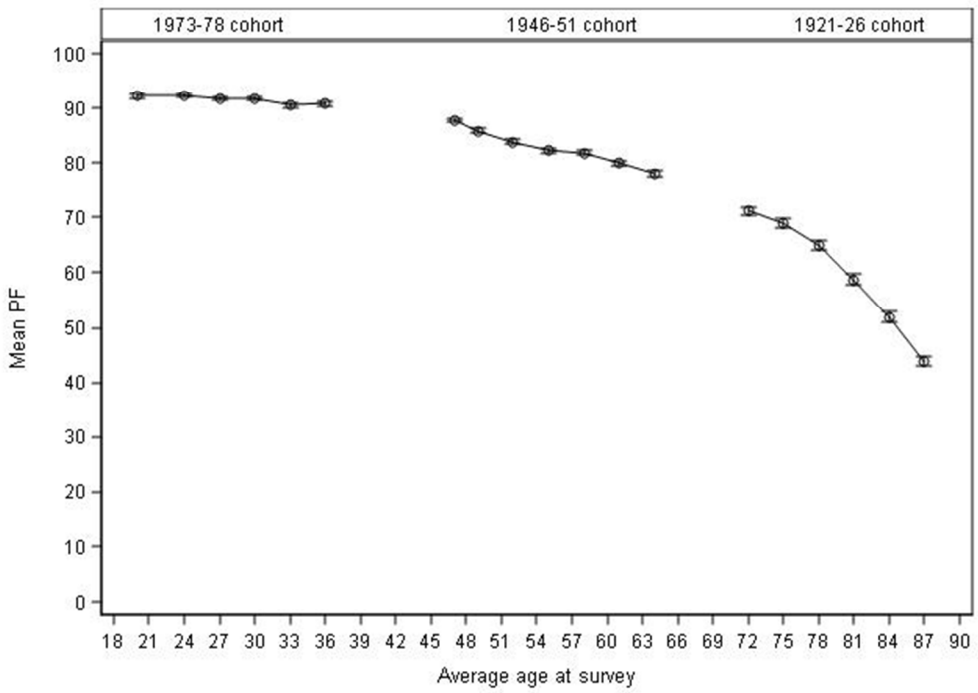

Figure 1. SF-36 physical functioning scores (means with 95% confidence limits) by age and cohort for all women who provided survey data in the Australian Longitudinal Study on Women's Health at the specified age.

In summary, Blane and colleagues present intriguing evidence to support the case for an additional role of positive health as an explanation for longevity risk. To identify the specific mechanisms operating across life and evident in the biological and social history of each cohort, they acknowledge the next step needed is to move beyond aggregated statistics for disease prevalence and mortality, to evidence from direct measures of health assets and across multiple cohorts and populations. Yet for more detailed investigation in future research, interpretation of results from cross-cohort comparisons can be problematic as it may not be obvious whether one would interpret heterogeneity across cohorts as (i) evidence against universal biological effects or (ii) evidence for contextual effects and effect modification. Nevertheless, on-going longitudinal studies are already applying the life course approach to the better understanding of disease, health, and positive health, and hold the promise that it will not be too long until more definitive evidence to explain longevity risk can be identified – sufficient follow-up time just needs to accrue. It may still be a decade or more, but what are decades when trying to understand the factors operating over 100-year timescales that led to this *black swan event.*

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The authors thank their six colleagues for thoughtful comments. These come from psychology (Hans-Werner Wahl), sociology (Mark Hayward), demography (Aart Liefbroer) and epidemiology (Gita Mishra, Isabel Ferreira, Ilona Koupil). It is heartening to see the level of agreement between them about required future developments in life course research, particularly the need for inter-disciplinarity, international comparative research and new studies to collect combined high quality social and biological measurements; as pioneered, we add, by Richard Suzman's legacy of the USA National Institute of Aging's international portfolio of studies of ageing (in relation to the inclusion of biological measurements, particularly the English Longitudinal Study of Ageing and, it is intended, the Survey of Health, Ageing and Retirement in Europe).

While celebrating this consensus, we regret the lack of comment from biology and actuarial science; and note that none of our commentators engaged fully with either the biological or the social historical parts of the ideas we presented. For us, it is important that data collection and analysis should be guided explicitly by social and biological plausibility; so, for example, while agreeing that "… important factors related to longevity risk seem not to belong to the health sphere", we nevertheless consider that death is inescapably a biological event and that social influences can produce death only through biological processes. Similarly, we feel that knowledge of social-historical context complements, rather than replaces, formal analysis of age-period-cohort effects. In other words, a commitment to social and biological plausibility can help to move us along the road from statistical association (…*related to*...) towards causation and the social challenge of longevity risk.

When reading the commentaries on our piece, we realised that we should say a few more words about the Strachan-Sheikh model and our idea of positive health.

The Strachan-Sheikh model

We use the word *model* in the sense of a theoretical description of the way a system or process works; or, if preferred, a way of thinking about reality. We accept readily the comments that the different components of the anatomical-physiological-biochemical system, which comprise the human body, grow and develop and lose capacity and atrophy at varying rates. Nevertheless, we find the generalised Strachan-Sheikh model useful for two reasons. First, it reminds us that humans are both social and biological beings who, biologically, start as a single cell which sub-divides and replicates many times, to each person's own achieved peak capacity which subsequently attenuates at varying speeds to death. Second, it reminds us that these rates of growth and atrophy are influenced by social circumstances, material and emotional, producing variation in life expectancy and mean height and age at puberty which vary by country, social class and historical period. It is this sensitivity of biology to social context which makes social history important to life course research, because it can inform us about who was likely to have been exposed to what during which phase of their development, with some exposures being beneficial and others noxious and the biological impact of specific exposures varying, to some extent, depending on whether the person was in the stage of growth or decline.

In relation to the decline phase of the Strachan-Sheikh model, it is worth mentioning a recent study of healthy, athletic people aged 55-79 years who spent two days completing a battery of physiological measurements, of which only six measures showed an inverse association with age (Pollock et al., 2015). These six involved mainly tissue repair (ILGF-1, pelvic bone density) and lung function (FEV_1, VO_{2max}, VT), which could be seen as components of intrinsic ageing or as signs of pre-clinical morbidity. In either case, the main implication of this small scale, cross-sectional study is that the biological path of decline to death is mainly via disease, rather than intrinsic ageing. If replicated, these findings argue for greater attention to variation in the rates of decline due to the living and working conditions of adult life.

Positive health

The financial consequences of the recent and, particularly among men, unanticipated increase in life expectancy at middle age, and its knock-on effects at later ages, have led to efforts to understand its causes. Actuaries are responsible for predicting future changes in life expectancy, which they are doing primarily in terms of anticipated change in disease risk factors and therapeutic innovations, to which our piece suggested the addition of a third factor, namely, changes to positive health.

We were led to the idea of positive health by two features of the present phenomenon and by the previous occurrence of something similar. For us, it is unlikely to be a coincidence that the mortality rates of the main causes of men's death have declined by proportionately similar amounts during the same period of time (see table 1 of our piece). Medical innovation, in our view, is unlikely to be the cause of this phenomenon, because its effectiveness varies greatly by disease – moderate to high in the case of ischaemic heart disease; low for carcinoma of the stomach. Similarly for risk factor change, where the most effective change (tobacco smoking cessation) impacts variably on different causes of death: high for lung cancer; low for accidents & violence.

The difficulty of explaining the present phenomenon solely in terms of medical innovation and risk factor change reminded us of the late nineteenth and early twentieth century when the mortality rates of the most prevalent infectious diseases of childhood (scarlet fever, measles, diphtheria, whooping cough, diarrhoea & vomiting) fell at the same time by proportionately similar amounts. Medical innovation (diphtheria anti-toxin) and the public health sanitary reforms (water & fly-borne infections) contributed to this change, but so did increased host resistance primarily due to better nutrition (the difference in host resistance contributes to high child mortality due to measles in refugees camps compared with lower mortality from the same virus in adequately nourished children). Our idea of positive health is that it is the equivalent of host resistance for chronic, non-communicable diseases.

Positive health in the sense of the body's ability to neutralise or mitigate the effect of environmental insults may also underlie the secular increase in the population's mean height and the fall in its mean age at the onset of puberty. And, we suggest in our piece, that the more or less linear fall across the twentieth century in the mortality rate of 65 year old women (see our figure 1) may be a further manifestation, due to cumulative improvements in the conditions of life, to which public health undoubtedly contributed, alongside technical-scientific innovation, democratic competition for power and self-organisation in trade unions, pressure groups and so forth. This suggestion raises two interesting questions in relation to historical change in mortality at age 65 years. Why did mortality rates at age 65 remain largely stable for both men and women from the 1840s to 1900? And why did the fall in men's mortality rate at 65 plateau during the 1940s, '50s and '60s? Amartya Sen's aphorism that "*There's never been a famine in a democracy*" (Sen, 1994) may be relevant to the former question, because the cohort who were aged 65 years in 1900, when mortality among 65 year olds started to fall, were born in 1835, which was one year after the start of the reform of the UK parliamentary electoral franchise, and so were the first cohort of 65 year olds to have lived their lives in an expanding democracy. To suggest an answer to the second question, we need to ask how the lives of men who were aged 65 years during the 1940s-60s differed from women of the same age. Both were born during the latter part of the nineteenth century, so the men were the right age to have fought in the first world war; perhaps their later mortality

plateau was an unrecognised long-term effect of their experiences during that war.

In summary, we welcome the enthusiasm of our commentators for what we regard as the right direction in which life course research should develop, although we give perhaps greater emphasis than they do to the inclusion of biologists in our inter-disciplinary efforts and the value of social-biological plausibility as a means of moving from correlation to causation.

Acknowledgements

MW is supported by funding from the German Research Foundation (Deutsche Forschungemeinschaft), project number WA 3065/3-1;DB is supported by UK Economic and Social Research Council award number ES/J019119/1

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