INCREASING LIFE EXPECTANCY AND THE COMPRESSION OF MORBIDITY: A CRITICAL REVIEW OF THE DEBATE

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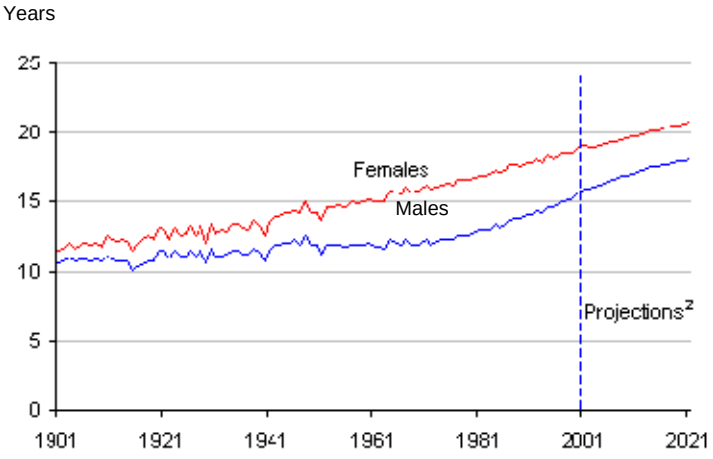
Introduction

Over the course of the twentieth century, life expectancy at birth in the United Kingdom increased by more than 30 years for both men and women; and in some parts of the developed world life expectancy at birth almost doubled in these years. This rapid and unprecedented increase in human life expectancy was associated with profound changes in the prevailing patterns of disease and morbidity – the so-called 'epidemiological transition'. Degenerative diseases, especially cancers and diseases of the circulatory system, replaced infectious and parasitic diseases as the leading causes of death. Death was being postponed to old age as the risk of dying at earlier ages fell dramatically. In the United Kingdom, deaths at age 75 and over comprised only 12 per cent of all deaths at the beginning of the last century. They rose to 39 per cent in 1951 and 65 per cent in 2004.

Figure 1 Life expectancy at 65 years for the United Kingdom 1901-2021



From: Social trends 2002

By the 1970s it was evident not only that the main causes of death had indeed changed, but also that the observed declines in mortality rates at older ages had not bottomed out. Life expectancy was clearly continuing to increase, driven mainly by

the continuing postponement of deaths from degenerative diseases. The fall in mortality rates at older ages has in fact *accelerated* in recent years (*see table1*).

Table 1 Average annual rates of improvement in mortality in the UK 1990-2002

Age group	1990 to 1994	1994 to 1998	1998 to 2002
60-69 males	3.0%	3.2%	3.9%
70-79 males	1.8%	2.3%	3.7%
80-89 males	1.1%	1.2%	2.5%
60-69 females	2.4%	2.5%	3.1%
70-79 females	1.0%	1.0%	3.0%
80-89 females	0.6%	0.4%	1.7%

Table from data supplied by Adrian Gallop of the Government's Actuary's Department.

The question therefore arises: how will population health evolve in countries where (i) birth and death rates are already low and (ii) death rates are continuing to fall, mainly at older ages? There are two ways of taking this question. We can ask about the effect of the continuing postponement of death to more advanced old age on the *overall* prevalence of morbidity and disability in the older population. Will it exert upward pressure on the prevalence of ill-health and disability in the population aged e.g. 65 years and above? The answer is one respect quite straightforward (and it also forms the basis for most of the gloomier projections of the impact of population ageing on health care spending): as the older population itself ages, overall prevalence of morbidity and disability will increase *unless* there is an offsetting decrease in age-specific prevalence rates. Since the prevalence of chronic degenerative disease increases with age, there will be an increase in the prevalence of ill-health and disability in the older population if age-specific prevalence rates remain constant as the population ages. Projections of the 'future burden of disability' in the older population depend therefore on trends in age-specific prevalence rates.

There is however another way of reading this question about the evolution of population health as mortality rates in old age continue to decline. We can ask about the effect of the continuing postponement of death from chronic degenerative disease on the experience of the successive birth cohorts that make up the older population. How do the more recent cohorts compare with the earlier cohorts? Are the additional years of life that the most recent cohorts have gained (and stand to gain) as a result of declining mortality rates years of good health or years of disability and frailty? It is usually (but not always) this second question which is being answered in claims about the expansion or contraction of morbidity; and it is this question also which is the focus of this present paper.

Three theories about increasing life expectancy at older ages

Predictions about the likely effect of the continuing postponement of death on the period of morbidity and disability at the end of life depend on the causal factors that are driving this trend. Three alternative explanations for the continuing postponement of death from fatal chronic degenerative diseases are to be found in the research literature – and they each have very different implications for the period of morbidity and disability at the end of life. Each explanation highlights one causal driver for the observed decline in mortality rates in old age – and provides us with a theoretically grounded view of what to expect as mortality rates continue to fall.

Expansion of morbidity

If increases in life expectancy are driven mainly by the increasing capabilities of medicine to prevent fatal outcomes from degenerative diseases, and the underlying patterns of disease (i.e. the way that the incidence of these diseases increases with age and their progression through increasingly disabled states) remain basically unchanged, there will be an expansion of morbidity as death rates continue to fall. In other words, medical advances push down the case fatality rates for cardiovascular disease, stroke and cancer while everything else about their epidemiology stays more or less the same. Mortality rates decline because people who would previously have died as a result of fatal complications arising out of their chronic disease are now surviving, which means that age-specific prevalence rates for chronic disease and / or

disability will increase. People will survive for longer with advanced degenerative disease, and as a consequence, the period of time that people spend in a state of chronic ill-health and disability at the end of life will increase.

The original expansion of morbidity theory highlighted the implications for population health of the increasing capabilities of modern medicine to prevent death in people with fairly severe and potentially fatal chronic disease (Gruenberg 1977). It also assumed that the underlying pathology of these degenerative diseases is so closely related to the ageing process (they are after all symptoms of ageing) that it would remain stubbornly resistant to the best efforts of medical technology and improvements in public health.

There are many and various objections to the expansion of morbidity theory in this form. In the first place, it is simply wrong to suppose that the capabilities of modern medicine to intervene in the course of degenerative disease are confined to fatal complications attendant on advanced disease. There is an enormous amount of evidence which attests to the effectiveness of secondary prevention, to the ability of modern medicine to slow down the progression of diseases such as cardiovascular disease and ward off the onset of their associated disabilities. Furthermore, if we understand the expansion of morbidity theory to entail the radical intractability of underlying patterns of disease in old age, then the evidence against it now seems overwhelming. There is for example, clear and indisputable evidence of international and *intra*-national variations in late life morbidity and disability (Khaw 1997). Since the best explanation for these variations appeals to the role of environmental factors in the aetiology of chronic disease, it is very hard to sustain the view that there is something inevitable and unchangeable about the timing of these disease processes in the lifespan. And finally, although it is possible that increased medical capabilities, combined perhaps with an increased determination to 'go the extra mile' with very ill elderly patients, might lead to some expansion of ill-health and disability at the end of life, it seems unlikely that this would lead to more than slight increases in average severity across the whole population (Verbrugge 1991).

Although this kind of evidence is decisive against the expansion of morbidity theory in its original form, it does *not,* however, dispose of the possibility that we are

nonetheless experiencing an absolute expansion of morbidity (*see below*). It is possible for there to be an expansion of morbidity at the end of life, even though the incidence of age-related disease is changing and people are staying healthier for longer. This will occur if life expectancy is increasing faster than healthy life expectancy/disability-free life expectancy.

Compression of morbidity

If, on the other hand, we suppose that increases in life expectancy are driven mainly by changes in underlying patterns of disease (people are living longer because the onset of chronic degenerative disease is being delayed to later ages), then we should expect to see a compression of morbidity *provided that* the observed increases in life expectancy are slowing down. The period of time that people spend in a state of chronic ill-health and disability at the end of life will contract.

The compression of morbidity theory therefore, like the expansion of morbidity theory, has two component parts. Fries (1980) based his original thesis on evidence of (i) delays in onset of chronic disease/disability (ii) US data suggesting a slowdown in rate of increase in life expectancy (especially in women). General health improvements lead to increasing life expectancy, *and* the impact of such health improvements on life expectancy must diminish as human longevity approaches its natural limits.

The compression of morbidity theory *in its original form* appears as hard to sustain as the expansion of morbidity theory, mainly because of the nature on data on life expectancy. There may have been some stagnation in increases in life expectancy in the USA in the 1970s – just as there is more recent evidence of stagnation in some other countries e.g. Netherlands (*see* Nusselder & Mackenback 2000), but this is not really enough to support the claim that increases in life expectancy are slowing down as they come up against the limits of our biological natures. Most of the evidence in fact points the other way, though this is not to say that improvements in mortality rates at older ages will not start to slow down in the near future – it is just that there is not much sign of it happening yet.

The case here is the same as with the expansion of morbidity theory. If the compression of morbidity theory is understood to entail a compression of *mortality*, it ceases to be plausible as an explanation for current trends in population health. It is however possible for compression of morbidity to occur without any discernible compression of mortality provided that healthy life expectancy increases faster than life expectancy (*see below*). What are required for this are substantial delays in the onset of disabling disease in later life, which is what we would expect to occur if primary prevention strategies were not only effective, but had a *big* effect. In other words, if the widespread adoption of healthier lifestyles, together with the social changes that support these lifestyles, *can* postpone the onset of these age-associated diseases, then the compression of morbidity becomes an entirely plausible scenario (Fries 2003).

Dynamic equilibrium

The expansion of morbidity thesis explains increasing life expectancy (falling mortality rates in old age) by highlighting 'delay' in the final stage of the progress of fatal chronic disease – the delay in the progression from severe disease to death that is effected by life-sustaining medical technologies. The compression of morbidity thesis also highlights just one stage in the progression of chronic disease – the delay in its appearance or onset – and relies heavily on claims about the effectiveness (actual and potential) of primary prevention for its plausibility. The dynamic equilibrium thesis offers an alternative view of the causal processes behind the postponement of death from chronic disease by highlighting the significance of delay in the intermediate stage of the disease process, namely, in the progression from less severe to more severe (and more disabled) disease states. It is possible, in other words, that people with chronic degenerative disease are living longer because the rate of progression of their disease is slowing down (mainly perhaps as a result of medical advances that have led to improved *secondary* prevention, but also as a result of underlying health improvement). And if *this* rather than postponement of onset or the postponement of death for those with severe disease is the main driving force behind increasing life expectancy at old ages, then we should expect increasing life expectancy to lead to (i) an increase in overall prevalence due mostly to increases in prevalence of mild/less disabling disease states (ii) largely stable rates of severe disease.

A complex picture: discussion

Even though these three theories are generally understood as mutually exclusive alternatives, the causal factors they each highlight are not. The theories are mutually exclusive insofar as they say that the *main* causal driver behind the continuing postponement of death by chronic disease is *either* delayed onset (as a result of improved primary prevention) *or* delayed progression of disease (as a result of improved secondary prevention) *or* increasing survival with severe disease (as a result of improved tertiary prevention). It is, however, possible that two or more of these factors may operate together to push up life expectancy, that healthier lifestyles are enabling people to remain free of chronic disease for longer *and* that the onset of more severe symptoms is being delayed in people who already have disease. Under these circumstances it is the balance between the different factors operative in postponing death by chronic disease that determines the outcome for the health of the older population.

Natural death and pre-death frailty

The original formulation of the compression of morbidity theory was elaborated with the idea of 'natural death' (Fries 1980). As the onset of chronic degenerative disease is postponed further and further into advanced old age, increasing numbers of elderly will die from 'physiological ageing', or 'failure of organ reserve', rather than as a result of chronic degenerative disease. Natural death, which is now comparatively rare, would replace chronic degenerative disease as the main cause of death in old age as more and more people survived into advanced old age without succumbing to any form of fatal chronic degenerative disease. Although natural death dissociates physiological ageing from the pathology of degenerative disease, the ageing process will still manifest itself in the increasing vulnerability of the body to external insult – an increasing inability to stabilise disruption and imbalance in physiological systems essential for survival. Natural death, as Fries conceives it, is most emphatically *not* a long and lingering process. It is an optimistic vision of a relatively shallow decline in physical ability (equivalent to an irreversible loss of fitness) interrupted by a sharp descent into death before the loss of ability becomes excessively burdensome. As the age distribution in deaths shifts to older ages, the period of severe disability/illness

before death would be compressed into a relatively short timespan. The replacement of death from chronic disease with natural death is the eventual outcome of the compression of mortality.

The expansion of morbidity theory revisited

What is left out of this picture – and is indeed rather peripheral to the original expansion and compression of morbidity theories - is *non-fatal* degenerative disease. Let us suppose, for example, that an increasing proportion of older people do survive into advanced old age without developing fatal degenerative disease. They do not die as a result of cardiovascular disease or cancer or stroke. Whether or not this leads to a compression of morbidity will depend on their risk for non-fatal and disabling degenerative disease. The prevalence of musculo-skeletal disease, cognitive impairment and sensory impairment all increase with age. People in advanced old age are at high risk for disabling or debilitating conditions which are clearly age-related *and* non-fatal. If the patterns of delayed onset and delayed progression for these diseases are the same as those for fatal degenerative disease, then the postponement of death will not 'make room' for an expanded period of disability due to non-fatal degenerative disease. If, on the other hand, they are different (i.e. not so easy perhaps to prevent), then there may well be an expansion of ill-health and disability as a result of these conditions (Olshansky *et al* 1991).

Clearly then, there is a great variety of evidence that may be brought to bear on the *theories* about the main causal drivers of mortality improvements at older ages, and it is these theories which form the basis of *predictions* about the likely evolution of population health as mortality rates continue to fall in populations which already have low mortality and low morbidity. As Olshanksy *et al* (1991) pointed out, what we need to know to decide between the main theories is whether "declines in old age mortality are caused by changes in the age-at-onset of fatal diseases, or improved survival with these diseases" (p 201) – and at the time of writing there was little or no evidence to suggest that the *onset* of the major fatal diseases of later life, namely, stroke, cardiovascular disease and cancer, was being postponed. Now, however, there does appear to be accumulating evidence of reductions in age-specific incidence rates for circulatory disease – both coronary heart disease and stroke. The incidence

of ‘first coronary events’ (non-fatal and fatal) has been declining in many developed countries (e.g. Arciero *et al* 2004; Pajunen *et al* 2004; Bata *et al* 2000). Data for trends in stroke point to the same general conclusion - that reductions in age-specific mortality rates for circulatory disease are at least partly caused by delayed onset, and reflect reductions in pre-morbid risk factors (Rothwell *et al* 2004). There are, however, plenty of uncertainties and complications in this picture. So, for example, a recent UK study reported that the declining incidence of coronary events was largely offset by the increasing incidence of diagnosed angina (Lampe *et al* 2005) – and Swedish studies have reported stable or increasing incident rates for stroke (Terent 2003; Johansson *et al* 2000).

Important as these data are for determining the underlying causes of observed reductions in mortality rates from circulatory disease in later life, they have no bearing on the modified version of the expansion of morbidity theory – since this is based on the hypothesis that extended survival exposes individuals to an increasing risk of *non-fatal* disabling disease – precisely because the major non-fatal disabling diseases of old age are harder to prevent than the major fatal diseases. Nor is it that difficult to marshal evidence in support of this hypothesis (e.g. compare dementia with heart disease), in which case the rate of progression and severity of these non-fatal diseases becomes the crucial determinant of the kind of trade-off that is made between longer life and worsening health.

Health expectancies and a framework for the descriptions of trends

Each of the theories discussed above purports to tell us what to expect as a result of observed trends in old age mortality. These theories will be either supported or undermined by observations which describe current trends in ‘health expectancies’ – and there is now quite a lot of published research which tries to determine whether morbidity at the end of life is *as a matter of fact* expanding or contracting, usually by taking repeated soundings of health status over time and integrating age-specific prevalence data obtained from these soundings with data on survival. Life expectancy is thus partitioned into two (or more) segments – the expected years of life with good health and the expected years of life with poor health – and changes in *both* these quantities tell us whether morbidity at the end of life is expanding or contracting.

If total life expectancy is taken as fixed, a longer life without ill-health or disability (i.e. an increase in healthy life expectancy or disability-free life expectancy) entails a compression of morbidity. If, however, life expectancy is continuing to increase (as it is in most low morbidity countries), an increase in healthy life expectancy or disability-free life expectancy may be accompanied by a constant, an increasing or decreasing expectancy of life *with* ill-health or disability. Reports of increases in healthy life expectancy or disability-free life expectancy may therefore give too optimistic an assessment of trends in population health. We want to know whether or not the period of morbidity and disability at the end of life is expanding or contracting or staying more or less the same. To be told, for example, that the healthy life expectancy of males at the age of 65 years has increased by 2 years over the last 10 years is to be told only part of the story. We also want to know about the number of years that the average 65 year old male may expect to spend in poor health.

It is now customary to distinguish between (i) absolute and relative compression of morbidity and (ii) absolute and relative expansion of morbidity.

- Absolute compression of morbidity = decrease in the number of years with disability/ill-health

This definition makes it clear that it is not necessary for life expectancy to be fixed in order for there to be a compression of morbidity. It is sufficient that the rate of increase in healthy life expectancy is greater than rate of increase in life expectancy.

- Absolute expansion of morbidity = increase in the number of years with disability/ill-health

An absolute expansion of morbidity may lead to either an increase or a decrease in the **proportion** of life with disability/ill-health, and these situations are distinguished by referring to a *relative* compression of morbidity or *relative* expansion of morbidity (see Box 1).

BOX 1 Changes in life-expectancy (LE) at 65 yrs and disability-free life expectancy (DFLE) at 65 yrs: three alternative scenarios

	LE (yrs)	*DFLE (yrs)*	*Years with disability*	*% LE with disability*
t_1	20	15	5	25%
t_{2a}	22	16.8	5.2	23.5%
t_{2b}	22	15.5	6.5	29.5%
t_{2c}	22	18	4	18%

t2a = absolute expansion + relative compression
t2b = absolute expansion + relative expansion
t2c = absolute compression + relative compression

Current trends in health expectancies

Two separate assessments, both published in 1991, of the likely evolution of population health as death rates continue to fall, concluded that an expansion of morbidity was the most likely scenario (Verbrugge 1991; Olshansky *et al* 1991). It was suggested, however, that

> ….convincing empirical evidence will be hard to come by, not only for what has happened in the past 50 years, but also what lies ahead for the next 50. This is because our main national surveys focus on prevalence and are seldom designed to address questions of incidence, severity, comorbidity and duration (Verbrugge 1991).

Since that time, there have been many studies published, which have added considerably to the body of evidence on the direction of current trends in health expectancies, and have also served to complicate the picture sufficiently to prompt expert reviews looking carefully at the inferences that might reasonably be drawn from this evidence. Although much of this evidence remains vulnerable to Verbrugge's criticisms, it has also enabled commentators and analysts to offer a more optimistic assessment of current trends.

> There is no evidence of expansion of morbidity based on more severe measures of disability prevalence. Recently emerging evidence from Europe and North America suggests that disability prevalence rates among older people may be starting to decline and we may actually be starting to see compression of morbidity in low mortality populations. (Mathers 1997).

> The study suggests that in many countries there have recently been moderate to large declines in chronic disability in the elderly. In countries where there was no decline observed, there was little consistent recent evidence of chronic disability increases – especially after adjusting for population age composition and trends in the rate of institutionalisation (Waidmann & Manton 2000).

The first wave of really robust evidence for this changed view of disability trends in the older population came from the USA and France. In the USA, Kenneth Manton and colleagues from Duke University published a number of analyses of data from the National Long Term Care Study (NLTCS) for the late 1980s and the 1990s (Manton *et al* 1997; Manton *et al* 2001). Age-specific disability rates were lower in the 1990s than in the 1980s. In other words, there was good evidence to suppose that there had been a significant reduction in the *rate of functional decline* in old age over those two decades. In France two different datasets covering the periods 1981-1991 and 1988-1998 supported a similar conclusion (Robine & Mormiche 1994; Peres & Barberger-Gateau 2001; Cambois *et al* 2001).

It quickly became apparent, however, that the picture was more complicated than these studies by themselves might suggest. The complications had two main sources. Evidence was becoming available from other large US datasets, which appeared to be somewhat at odds with the results from the NLTCS (e.g. Schoeni *et al* 2001). And it became increasingly clear also that evidence from other developed countries did not fit in very tidily with the interpretation of disability trends suggested by the US and French data.

The problems of sifting and piecing together the apparently diverse results from several different US datasets have led to the publication of two major expert reviews in the last three years (Freedman *et al* 2002; Freedman *et al* 2004) – and it has to be

emphasised that this exercise has an importance that extends beyond the USA. There is no other country in the world that has anything like the same wealth of good quality data on disability trends in the older population as the USA. The fact there are several major national surveys taking repeated soundings of the health of the older people – using slightly different methodologies - provides analysts with the richness of data that is needed to get a grip on the complexity of these trends in health status.

Most of the interpretative uncertainties that surround the US data have arisen out of the inconsistencies in the results that different studies have obtained for trends in *severe* disability. Although the evidence for a substantial decline in mild-moderate levels of disability – generally measured by difficulty with instrumental activities of daily living such as shopping or performing household chores - is now about as good as it can get, the evidence for a decline in severe disability looked much weaker (Freedman *et al* 2002). However, as some analysts have pointed out (e.g. Spillman 2004), the results for IADLs were consistent with the supposition that there had been no *real* improvement in underlying health [1]. The fact that fewer people were reporting difficulty with IADLs could just as well be explained by the development of an increasingly 'age-friendly' environment (e.g. more technology in the home) as by less functional impairment.

A second look at the various datasets persuaded a specially convened committee of experts (Freedman *et al* 2004) that there had indeed been a substantial decline in prevalence rates for severe disability – measured by reports of difficulty with ADLs or use of help with ADLs – which fell by 1%-2.5% per year during the mid and late 1990s. It seems clear also that the use of equipment to help with ADLs was taking the place of personal help. Evidence for decline in the 1980s and early 1990s is not so good, however. In other words, the data does not support the view that the health of older people has been steadily improving over the last three decades, as some commentators (e.g. Mor 2005) have claimed.

[1] This is analogous to the situation reported in a recent study of Japan (Schoeni *et al* 2005), where there was clear evidence of a decline in disability alongside stable rates of prevalence for functional limitations.

So has there been a compression of 'morbidity'[2] in recent years in the USA? Have the improvements in disability-free life expectancy (signalled by the decline in disability rates) kept pace with – or even perhaps outpaced – improvements in life expectancy? Robine and Michel (2004) are inclined to think so. Lubitz (2005) presents data from one of the big US datasets (the Medicare Beneficiaries Survey), which suggest a smallish *relative* compression of morbidity. Since, however, mild-to-moderate disability appears to have increased for this particular population, the best fit for the data is given by the dynamic equilibrium hypothesis.

BOX 2 Measuring trends in health expectancies

Studies reporting trends in health expectancies in different countries mostly rely on a small set of self-report measures of health status.

- There are studies of trends in *healthy life expectancy* which characteristically rely on self-assessments of global health (rated e.g. as excellent/good/poor) or use data on the prevalence of chronic or 'longstanding' illness.

- There are studies of trends in *disability-free life expectancy* which generally rely on reports of difficulties with activities of daily living (ADLs) or instrumental activities of daily living (IADLs). This has the advantage of allowing an approximate assessment of the degree of disability (e.g. severe as opposed to mild/moderate) experienced by individuals in the sample. There are some studies, however, which use answers to a single question to determine the presence or absence of disability. Data from the UK General Household Survey on 'limiting longstanding illness' is used in this way purpose to estimate 'active life expectancy'.

- Trends in *disability- free life expectancy* are also sometimes assessed with data on the prevalence of specific kinds of functional limitation (e.g. mobility limitations) or impairment (e.g. cognitive impairment).

[2] The US data cited here tell us about disability – the effects of chronic disease – rather than morbidity *per se*. This is true of a great deal of the research on health expectancies. US data on chronic disease in the older population indicate -- and this trend appears to be more or less universal across the low-mortality countries in the developed world -- that prevalence rates for major chronic diseases have increased at the same time as disability rates have decreased. See e.g. Rosen and Haglund (2005) and Bronnum-Hansen (2005) for recent Scandinavian data. As Mor (2005) points out, improved detection of individuals with early-stage disease and/or earlier reporting of disease would lead to increased prevalence rates for chronic disease. It would also create the opportunities for interventions which slow down the progress of disease and reduce its impact on functional ability.

Outside the USA (and France) the picture is more complicated – just as the evidence is more patchy. This, for example, is what Robine *et al* (1998) had to say about the state of affairs in the European Union at the end of the 1990s[3]:

> These results suggest that the EU overall is in a pandemic phase as regards disability, corresponding to a situation in which life expectancy is increasing more quickly than disability-free life expectancy, and in which therefore the proportion of disability-free life years is falling (p17).

Other studies which have reported an absolute expansion of morbidity/disability include Mathers (1996) for Australia, Saito (2001) for Japan, and Zimmer *et al* (2002) for Taiwan. Recent analyses of data for New Zealand (Graham *et al* 2004) and the Netherlands (Perenboom *et al* 2004 & 2005) indicate an expansion of disability attributable mainly to increases in age-specific prevalence rates for *mild-to-moderate* disability rather than severe disability, and therefore lend support to the dynamic equilibrium hypothesis. And finally, there are a few countries, which seem to have experienced an actual contraction of the period of disability at the end of life. These include Austria (Doblhammer & Kytri 2001) and Denmark (Bronnum-Hansen 2005).

Robine and Michel (2004), in a wide-ranging review of evidence on changing health expectancies, make a strong case for taking such international differences and discrepancies seriously – for trying, in other words, to piece these results together into a single 'narrative', which lets us see how population health is evolving in developed countries as life expectancy continues to increase. Instead of saying that different countries are simply 'doing better' than others in making progress towards a common goal – the compression of morbidity – they postulate an ongoing process of 'disability transition' with successive stages or phases. Those countries in the world with the highest life expectancy tend to be 'further down the line' in a process which shows us the complex effects of declining mortality and health improvement in an ageing population. The differences between such as Australia, Japan, the USA, Austria and the United Kingdom are to be explained by placing them at the appropriate 'stage' in this process.

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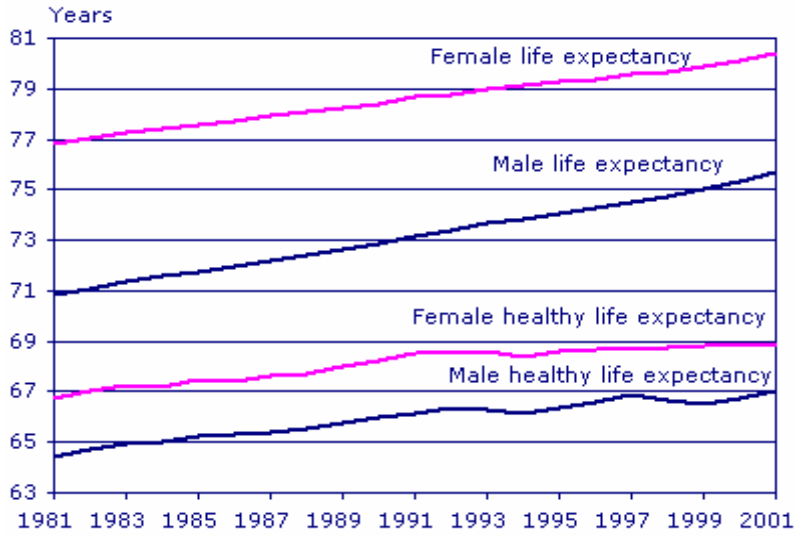
[3] This particular study has a cross-sectional design, and needs therefore to be interpreted with considerable caution.

The pattern we find, therefore, is essentially a temporal one:

- an increase in the survival rates of sick persons leads to an initial expansion of morbidity;
- improved control of the progression of chronic diseases leads to dynamic equilibrium between the fall of mortality and the increase in disability;
- improved health status and health behaviours in new cohorts of older people leads to some compression of morbidity;
- the eventual emergence of *very* old and frail populations leads to a new expansion of morbidity[4].

Australia and Japan have relatively high life expectancy compared to other developed countries – and the data there point to a *new* expansion of morbidity (as extended survival makes room for *non-fatal* chronic disease). The USA, on the other hand, has a relatively low life expectancy – and the data there suggest dynamic equilibrium. Austria sits somewhere in between – hence the compression of morbidity. And the UK?

Figure 2 Life expectancy and healthy life expectancy in the UK 1981-2001



From: Health Statistics Quarterly no. 15 Autumn 2002

[4] These four elements may of course coexist in the same place. The scenario we find in any particular country will depend on their relative weights

Life expectancy in the UK is lower than in Australia, but higher than in Austria, and according to Robine and Michel, the UK data on health expectancies fit in with their hypothesis: evidence of a compression of morbidity in the 1980s and early 1990s followed by an apparent expansion of morbidity.

Among the many questions raised by Robine & Michel's synthesis of the international data on trends in health expectancies, there are perhaps two that stand out. Is there an alternative way of reading these data, of piecing them together so that they tell one coherent 'story' about what is happening to the health of older populations in low-mortality countries around the world? Are the data of sufficiently high quality to enable us to tell *any* kind of coherent story about what is happening in different parts of the world?

James Fries (2004), for example, argues that it is possible to challenge most of the evidence that Robine and Michel present for an emerging 'epidemic of frailty' as more and more lives are extended into "extreme regions of the lifespan" (Olshanksy 2004). He also thinks it incoherent to suppose that individuals could survive for a long time in a frail state – since frailty *by definition* entails a high risk of mortality – and challenges the idea that some countries have 'passed beyond' a phase in which health improvements have led to a compression of morbidity. We are still waiting to see what will happen when healthier lifestyles and improved living standards spread through an entire population and take the process of risk reduction for chronic disease about as far as it can go.

It is, however, seriously questionable whether the data are good enough to support any kind of coherent story about current trends in health expectancies – and the expansion or contraction of morbidity in later life. The essential problem lies in the relationship between chronic disease, functional impairment and disability. Disability is a 'social construct' in the sense that it refers to an individual's capacity to function or carry out a role in a given social and environmental context. The extent to which individuals are disabled as a result of functional impairment (e.g. mobility impairment) depends on this context – and in recent years it has changed enormously. It is vitally important, in other words, to be able to 'factor out' the contextual and attitudinal elements in measures that purport to tell us about real changes over time

(as well differences between different countries) in the 'intrinsic' health status of the older population – and a great deal of the available evidence on health expectancies is vulnerable to precisely this kind of criticism.

Conclusion

Where does this leave us? It means that the kind of evidence that is needed to support *solid* conclusions about the expansion or contraction of morbidity is simply not available for most countries in the developed world (including the United Kingdom). This is not to say of course that the evidence contained in a time-series such as the General Household Survey can be ignored or discounted. It certainly looks as though total life expectancy in the UK is increasing faster than either the expectation of life in good health or the expectation of life 'without limiting longstanding illness' (*see figure 2*). But in the absence of more detailed information about changes in physical and mental functioning, it would be premature to declare that we are at the beginning of 'an epidemic of frailty' or a significant expansion of the period of ill-health and disability at the end of life.

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