Graeme Ford and Martin Frischer

Issues for Predicting the Health of the Elderly

Abstract

In this paper we consider the extent to which we can anticipate changes in the health profiles and likely experience of future generations of the elderly. We evaluate the extremely limited evidence on the changing health and morbidity of previous generations and younger generations to see whether any clear trends emerge as the basis for future estimates. We draw attention to the importance of age, cohort and period models in evaluating the available data. Finally we consider the implications for morbidity of changes in mortality patterns. Each of these elements separately and together determines the degree of predictability of future health patterns. The emphasis is on the overall burden of ill health of the population and the balance between broad categories of conditions rather than on the aetiology of particular conditions.

Introduction

Quantitative and qualitative aspects of the debate

The single most compelling and most frequently documented fact about the populations of Western societies is the dramatic increase in the proportions and absolute numbers of the elderly (Olshansky and Ault 1986; Rice and Feldman 1983; OECD 1988). These demographic changes are largely driven by events and transitions which occurred early in the century and are of such magnitude that they are likely to be the major determinant of changes in the overall quantity of ill health for the foreseeable future. A focus on future demands on the health services would direct attention primarily to increasing proportions of the very elderly where dependency is concentrated and the greatest demands are generated (Milbank Memorial Fund Quarterly 1985;63/2).

Brody (1985) gives some illustrative examples of the magnitude of changes to be expected as a result of these demographic shifts. In the U.S. in 1980, there were 200.000 elderly persons who experienced hip fractures; by the year 2000 this is expected to rise to 330.600 and by 2050 to 650.000. In 1980 an estimated 2.000.000 showed symptoms of Alzheimer's disease; this is expected to rise to 3.800.000 by the end of the century and to a massive figure of 8.500.000 by 2050, 5.000.000 of them aged over 85. In any discussion of population health in the future these major demographically driven trends need to be kept firmly in mind.

In this paper we have left these calculations to one side and instead analyse recent changes in the prevalence, composition and severity of health as experienced through middle age and beyond into old age. It has been argued that recent fundamental changes in morbidity and mortality patterns can be observed to be taking place, which serve to modify the brute changes due to demography alone and soften the apparently pessimistic vision of the future which they suggest (Fries and Crapo 1981; Palmore 1985; Fries 1988). The arguments and data which we present relate exclusively to the developed economies. The evidence comes from a limited number of sources. While virtually all industrialised societies have evolved systems for recording mortality, very few collect information on morbidity at a national level for other than a few selected

morbid conditions (Alderson 1988).

Blaxter (1989) has drawn attention to the difficulty in comparing morbidity across Europe. This stems in part from practical difficulties in finding comparable indicators, but also because health, in any other than the narrowest biomedical definition, has strong cultural and social determinants. While there may be no good morbidity data, international analysis of mortality shows large differences in cause-specific death rates and therefore provides an indirect suggestion that underlying morbidity might also show strong differences (Manton 1984). Having set out to investigate class inequalities in mortality patterns in a variety of European societies Leclerc (1989) concludes: "The probability of dying from one given cause of death can probably be more accurately predicted by the country where a person lives, than by whether or not he belongs to a given social class."

The data presented here are therefore indicative; we do not imply an universal pattern. Arguments and conclusions about the size, rapidity and importance of change are greatly affected by the time-perspective adopted. If we consider the entire span of the twentieth century, then for Western countries we are confronting a period of massive transformation. Demographers have characterised it as a unique epidemiologic transition, consisting of three distinct stages. We have moved from the "Age of Pestilence and Famine" via the "Age of Receding Pandemics" to the "Age of Degenerative and Man-Made Disease" (Omran 1971). In less colourful terms, we have seen a massive drop in death rates and a dramatic extension in life expectancy primarily driven by the virtual extinction in Western societies of the previously rampant major infectious diseases. The inevitable outcome has been a huge upward shift in the age pyramid and the emergence of the chronic degenerative diseases of middle and old age as the major elements in morbidity and mortality. On this scale we observe unprecedented qualitative change in a relatively short space of time. The interesting question is whether we have reached the end of the process or whether we can expect further significant transformations in the future.

To many observers in the 60s and 70s it did seem as if a new equilibrium had been reached, with mortality restricted to the middle and late years and resulting from relatively intractable chronic disease states. It seemed there was little scope for further change or improvement. Population predictions were made on this basis and they were wrong (Olshansky 1988). Starting in the mid-60s, the U.S.A. and a number of other developed countries began to experience further, and unpredicted declines in mortality in middle age. Most recently similar declines appear to have occurred in the oldest old (Manton and Soldo 1985). These gains are due largely to declining death rates in the circulatory diseases, although many other common degenerative illnesses do show some improvement. Olshansky and Ault (1986) considered these developments dramatic enough to talk about the emergence of a fourth, qualitatively different, epidemiologic era characterised as the "Age of Delayed Degenerative Disease."

While we have some sense of the pattern of mortality over time, we seem to know almost nothing about corresponding morbidity. In the absence of substantial data, observers have made wildly differing assertions about likely developments. Much of the recent debate on patterns of morbidity in the elderly has centred round the controversial writing of James Fries. He represents the extreme of optimism as he contends that, against a finite human life span and a "compression of morbidity" onset to the very last years of life, we can look forward to a time when chronic illness is postponed and the typical life span is predominantly disease-free. Others have argued the contrary, that given the general association between age and increased morbidity, as causes of premature death are removed, the larger surviving population will simply be proportionately older and suffer

more of the existing chronic ailment over a longer period of time; existing health problems are projected into the future on a magnified scale (Gruenberg 1977; Kramer 1981; Sneider and Brody 1983; Riley 1989).

We will return to the details of these debates later, we merely note at this point that these are predictions still painted on a very broad historical canvas. Long-term future predictions are derived from underlying theoretical models of the relationship between mortality and morbidity, drawing generally on accurate mortality data, but utilising only thin strands of information on changing morbidity.

Estimating morbidity

We begin by shortening our focus to empirically observed recent trends in health, which might allow us to form short or medium term predictions about changing health patterns. This is less exciting than debating sweeping theories, but it is a necessary starting point and a useful corrective to overgeneralisation.

Evidence is so thin that Manton (1982) has argued that currently we can only utilise mortality data to explore changing patterns of health. At the same time, he emphasises the vital importance of developing good databased models of the relationship of morbidity, disability and mortality if we are to have any real insight into underlying processes and future projections. Blaxter (1989) provides indirect evidence of the independence of mortality and morbidity rates, cross-nationally and within class and sex subgroups of the population, and argues for the necessity to use morbidity rates rather than mortality as indicators of differential health and health inequalities in advanced countries, which share generally high life expectancies.

Verbrugge (1986) looks at the distributions of symptoms, acute and chronic health problems and causes of death at various stages in the life-cycle. She concludes that for the young there is little connection between everyday health and the commonest causes of death, by middle age the potentially fatal diseases begin to compromise daily health and appear as causes of some of the most frequently experienced symptoms and conditions along with other non-fatal conditions, while for the elderly there is a higher but by no means complete correspondence between predominant health problems and the commonest causes of death. A Nuffield Provincial Hospitals Trust working party carefully considered the extent to which mortality data can be utilised as a surrogate for morbidity data for planning purposes, and concluded that it is only demonstrably valid for a small class of diseases with high case fatality (Ashley and McLachlan 1985).

Morbidity and self-reported health measures

In considering morbidity we do have a large number of cross-sectional estimates of morbidity from one-off population surveys, and we have a quite a number of longitudinal studies, often of specific conditions such as heart disease (Blaxter 1986). But what we need are time series data on the general health of successive cohorts at each age in a population, using comparable methodologies and definitions. Because this sort of monitoring requires a long term perspective, and the data are expensive to collect, it is scarce. None of the data sources are ideal, but it nevertheless seems worth drawing together what data we can from published sources. (Alderson 1988) attempts a comprehensive list of possible sources. We concentrate on conditions which are potentially life-threatening, or chronic and capable of producing long-term disability or discomfort, on the enduring health status of the population, rather than on transient acute episodes.

There is one very important caveat about the commonly utilised data sources. They are all based on self-reported measures of health

rather than on clinical investigation. While there is no absolute dichotomy in validity between "medically" identified and self-identified conditions, different approaches to the clinical evaluation of health give differing results, and self-reports can be made more accurate by careful methodology. The particular problems inherent in self reported data are fairly evident (Taylor 1981). The relatively few studies which have compared self-reports of health conditions with clinical investigations or clinical records show a modest level of agreement. Usually, less than half the conditions uncovered by clinical investigation are self-reported, while about a third of self-reported conditions are not matched by clinical evaluation (Trussell and Elinson 1959a; Trussell and Elinson 1959b; U.S. National Centre for Health Statistics 1965).

This is not to say that the subjective element in self-reporting is in a simple sense error. A number of studies have shown that the subjective aspect of self-reported health carries important information, which is not captured by the clinical account of current health status. Maddox and Douglas (1973) in the *Duke Longitudinal Ageing Study* found that self-rated health was a good predictor of subsequent physicians' rating of health, while physicians rating was a poor predictor of self-rating at a later point in time. Recently, Idler et al. (1990) have shown that self-rated health is a strong independent predictor of mortality even when current health status is controlled. When we look at data on health, based on self-reports collected over time, we need to be aware that observed changes may be due to a number of major artefacts in addition to any real underlying change in health status. Firstly, there may be changes due to differences in question wording. The major data sets we consider minimise this problem by consistent question wording, but there may of course be subtle changes over time in the way that people respond to particular wording. Secondly, there might be broad secular trends in the propensity to report ill health based on a changing level of tolerance and changing lay definitions of health and illness.

This variation in reporting might be variable between conditions. Cultural changes, changes in medical practice and media attention periodically "discover" and emphasise new or different conditions, while other illnesses become unfashionable. It is nevertheless worth considering trend data in self-reported health. We have no other substantial evidence on trends and many previous authors have based quite strong claims on data of this sort.

The empirical pattern of the recent past

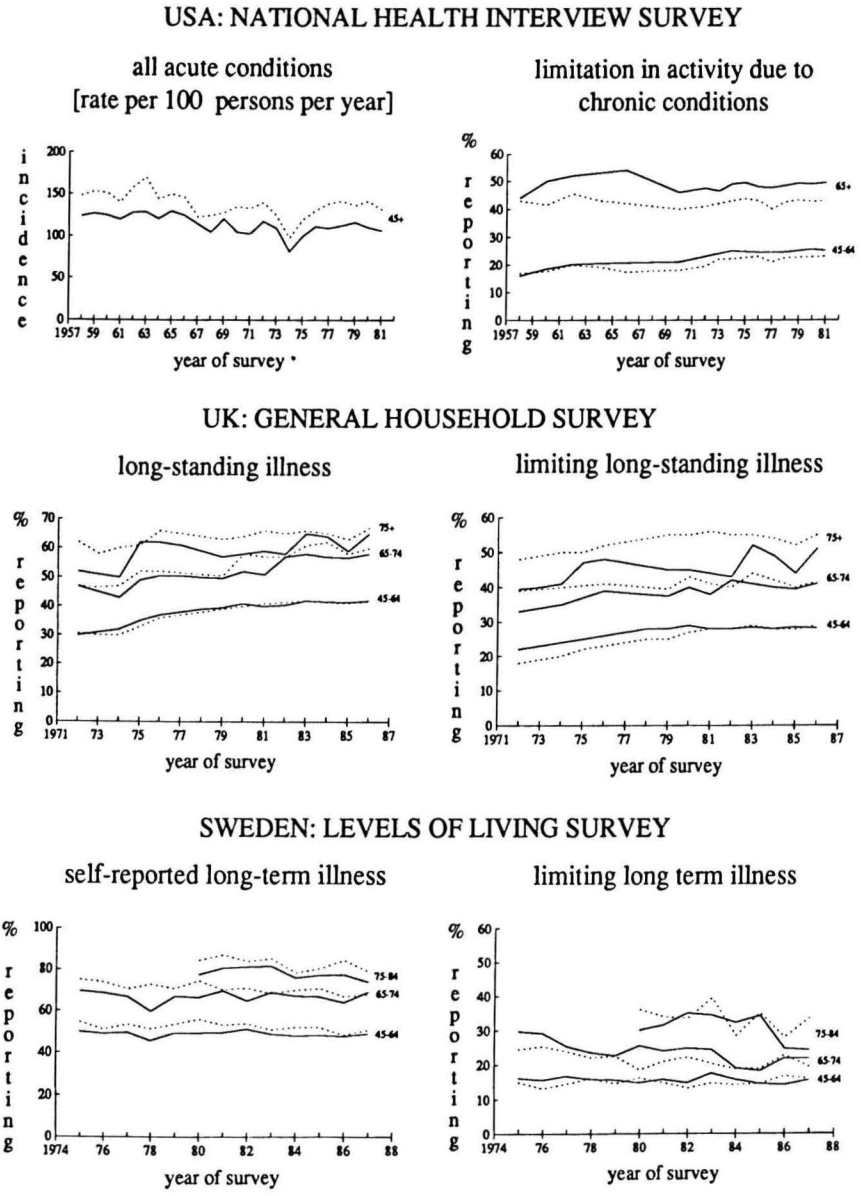
There are four basic questions we can address. We can consider changes in the overall incidence of chronic or serious illness in general and at various stages of the life cycle; we can look at the changing mix of morbidity; we can ask what implications observed changes make or might make to the illness experience of the population; and finally, we can look at changing health service demands generated. The later point is not addressed in this paper.

Trends from time-series data

Considering first the total quantity of ill health, Verbrugge has analyzed U.S. data from *the National Health Interview Survey* (Verbrugge 1984). This annual survey was initiated in 1957 and provides the longest available time-series of self-reported health indicators. It contains measures of acute and chronic morbidity, restricted activity and long term limitation. The basic patterns are represented in fig. 1.

Acute episodes alone show a general, although not dramatic, decline for the population aged 45 plus. The method of data collection prevents a direct estimation of prevalence of reported chronic ill health. However, we can look at patterns of restriction and limitation due to chronic ill

Fig. 1:
International self-reported morbidity indicators



health. We see quite consistent trends. Rates for the elderly generally run at about double those for the middle-aged. The figures seem to run pretty much in parallel, with little suggestion of any decline in illness in the middle-aged group, which would lead to a divergence of the lines as the rates for the middle-aged fell. It is difficult to make definitive statements about trends by eyeballing graphs, but they all seem to be flat or rising. There may be some suggestion of a dip between 1958 and 1970 for some groups, but in every case these have shown a consistent flat or rising trend thereafter. If there has been any trend in limiting illness it has been upward for both age groups. Women are consistently worse than men, which is part of a different story.

In Britain, *the General Household Survey* provides information over a shorter time period. The data are also presented in fig. 1. These figures show very similar trends. For prevalence of longstanding illness and limiting illness, there are quite strong upward gradients. These appear to be larger than in the U.S. data, but the time period corresponds with the second half of the U.S. time series when a generally upward trend is also discernible. Here the data are presented for three age bands and again they run strikingly in parallel, suggesting no alteration in the relative distribution of illness between the age groups.

The Swedish Levels of Living Survey suggests a different pattern. The graphs show changes between 1975 and 1987. Tests of significance based on fitted linear trends are reported between 1975 and 1985 (Vogel et. al. 1988). The trends are downward for both measures shown for all age/sex combinations except long-term illness for men aged 65 to 74. The trends are statistically significant at the 95 percent level for long-term illness in women aged 65-74 and for impaired working in both men and women of the same age but not for the other combinations (for those not employed, a definition of impaired daily activities was substituted for impaired working. Significance levels are not reported for the oldest group). Finally, decennial surveys conducted in France in 1970 and 1980 showed an increase from 64% to 82% respondents reporting "some illness", although this is likely to be an overestimate due to changed question format (Aiach and Curtis 1988).

Is there any shift in the illnesses experienced between groups or over time? In two out of three countries, for which we present data, there has been a clear increase in the propensity to report illness and consequent activity limitation. There is some indication that the French data show a similar increase.

Changing composition of illness

The next question we would wish to address is whether there is any change in the nature and composition of that reported illness? Satisfactory data here are even less easily obtained. In addition to the fact that there is very little available, the interpretation of apparent changes in particular health problems are crucially affected by the level of aggregation of the data and the categorisation used. Conclusions should be treated cautiously.

The best data again come from Verbrugge. She has assembled tables from various sources covering self-reported disease prevalence the U.S. in the years 1968 to 1979. This quite elderly data cover quite a short time period. It is nevertheless worth examining to gain an impression of just how volatile, or otherwise, the health mix might be over a short time interval and to get some impression of any redistribution of health which might have occurred. It is worth remembering that it was precisely during this period that Fries put forward claims to have detected an emergent and dramatic change in the trajectory of ageing. Table 1 presents data from Verbrugge in a simplified form.

Verbrugge makes a distinction between potential killer and non-killer diseases which we reproduce in the tables. The rank

Table 1:
Mortality change in the U.S 1968 to 1979

Killer diseases	MALES 45-64		MALES 65+		FEMALES 45-64		FEMALES 65+	
	A	B	A	B	A	B	A	B
Diabetes	1.54	+38	1.34	+22	1.53	+34	-0.74	-8
Diseases of the heart	3.44	+35	6.64	+33	4.45	+55	1.42	+12
Hypertension	10.16	+100	17.38	+123	7.54	+50	19.33	+80
Cerebrovascular	0.16	+13	-13.90	-26	0.68	+65	-0.39	-9
Arteriosclerosis	-	-	9.15	+305	-	-	10.32	+151
Bronchitis	0.50	0	-1.10	-23	-0.42	0	1.51	+11
Emphysema	0.69	+31	0.94	+16	1.13	+174	1.25	+109
Asthma	-0.15	0	-2.05	-48	0.33	0	-1.10	-35
Hernia	1.58	+46	-	-	0.55	+24	1.43	+34
Cirrhosis	0.34	+60	-0.19	-41	0.18	+56	-0.08	-47
Nephritis	-0.74	-31	-0.85	-19	-0.89	-16	-0.08	0
Non killer diseases								
Varicose veins	-0.97	-29	-1.55	-29	-3.34	-30	0.25	0
Hemorrhoids	-1.92	-24	-1.31	-9	-1.13	-14	-0.45	-6
Sinusitis	1.39	+9	1.39	+11	4.54	+27	2.40	+16
Hay fever	1.64	+40	0.91	+26	1.81	+30	1.64	+43
Peptic Ulcer	-1.13	-25	-1.72	+55	0.31	0	1.74	+79
Upper gastrointestinal	1.07	+42	0.15	0	0.41	+19	-1.11	-27
Constipation	-0.68	-35	-2.10	-34	-0.94	-19	-4.23	-35
Eczema and skin	1.18	+59	0.96	+46	0.69	+19	-0.44	0
Corns	-2.70	-54	-3.58	-55	-5.75	-50	-6.92	-48
Arthritis	4.04	+27	6.76	+24	5.62	+22	5.43	+12
Gout	1.07	+63	1.44	+74	0.37	+49	1.49	+196
Other Musculoskeletal	0.89	+24	-0.63	-25	1.59	+39	1.39	+46
Vision	0.08	0	-6.33	-35	-1.00	-19	-10.28	-47
Hearing	0.76	0	-1.14	0	0.26	0	-1.27	0
Back and spine	0.36	0	2.20	+40	0.89	+13	3.27	+43
Lower limb	-1.43	-28	-1.73	-26	-1.15	-25	-2.88	-32
Paralysis	-0.38	-32	-0.74	-27	-0.43	-45	-0.15	0

A absolute change in incidence B percentage change in incidence

ordering by prevalence of these conditions is broadly similar for both middle-aged and elderly and for males and females (data not shown here). The ordering is also stable over time; there is little indication of a shift in the health problems experienced between age/sex combinations.

The tables are fairly indigestible, but there do seem to be clear patterns. Considering the columns marked B we see substantial percentage increase in the incidence of many conditions and importantly, many of these are the commonest conditions. Among the killer diseases, diseases of the circulatory system show consistent and usually large increases across age/sex categories, with the exception of cerebrovascular disease which decreases in both elderly males and females. Neoplasms are not shown here, but we know that they show an upward trend for total incidence and also for many specific forms in most industrial societies over this period. Hernia increases for all groups for whom there are data, while diabetes also shows a strong upward trend for all but elderly females. Cirrhosis increases for the middle-aged while decreasing for the elderly. Of the respiratory conditions, emphysema increases while bronchitis shows a mixed pattern. Only asthma, along with nephritis and nephrosis show a general decline. The picture is one of sizable overall increase in this group of conditions and it is important to note that the percentage changes for middle-aged and elderly, usually move in parallel. There is little evidence of morbidity declines for particular conditions in middle age, but not in old age, with the implication of delayed onset of chronic ill health.

The non-killer diseases reported by Verbrugge are a curious mixture of conditions, symptoms and impairments. It is less easy to infer an overall trend as there are some quite sizable shifts, but for many conditions these go in opposite directions for different subgroups in ways that hard to interpret. Looking at the columns marked B, we see the absolute percentage difference in prevalence caused by the proportionate shifts reported already discussed. That is, it takes note of initially very different prevalence rates between different age/sex cohorts.

The overall changing health experience of these cohorts is dominated by changes in already prevalent conditions. In the killer categories we see, as might be expected, that the circulatory diseases show far bigger absolute changes than any other categories. Diseases of the heart, hypertension, and arteriosclerosis (only shown for the elderly) show very large percentage increases, while on the other hand, cerebrovascular disease shows a big drop, but only for middle-aged men. Other significant increases are in diabetes for middle-aged men and younger women and emphysema for elderly women. Only asthma shows sizable drops in any cohorts.

In the non-fatal conditions, arthritis shows by far the biggest increase. The other musculoskeletal conditions also tend to show quite large increases as do hay fever and sinusitis. Varicose veins, haemorrhoids, constipation, corns and problems of the lower limbs are declining. Peptic ulcers are declining for men but increasing for women. Vision and hearing are hard to interpret since it is not clear whether the rates include some or all medically-corrected conditions. The high rates reported for the sensory conditions make this seem likely.

Summary of time-series changes in composition of illness

Overall what we seem to observe is the following:

1. A picture dominated by large proportionate and absolute changes in circulatory diseases. These are probably the only changes affecting a sufficient proportion of the population to lead to talk of qualitative transitions in population health.

2. Sizable, but less dramatic increases in arthritis, already the leading disease of women and older men - almost a slow epidemic - which taken with increases in the other musculoskeletal diseases is likely to have widespread implications for population health and functioning, especially of older segments of the population.

3. Substantial declines in some previously common conditions such as varicose veins, haemorrhoids, constipation and corns, which together we might reasonably describe as diseases of discomfort rather than limitation.

4. In general, these first three groups show consistent trends across age/sex sub-groupings. There is no indication of major redistribution of ill health between age groups, only a secular tendency to increase or decrease.

5. Major proportionate changes in some conditions, which suggest likely trends for these particular conditions and might have major implications for the health of subgroups and pose particular management problems, but which still have relatively low population incidence and therefore show fairly small absolute changes. Some of these, such as gout, show consistent secular trends, while others, such as peptic ulcers, have complicated sex or age differences. Taken together these tend to "cancel out" in their effect on total chronic health, and it would probably be wrong to make strong predictions about them individually on the basis of this type and quality of data.

Verbrugge argues that "signs of increasing morbidity far exceed signs of decreasing morbidity or no change." If we view the data setting aside the circulatory diseases, it seems less certain that we would clearly see a pattern of increase. We might instead perceive fairly large movements between conditions but no overall increase or decrease. We have to guard against letting a major but localised set of changes colour our perception of the entire set of conditions. It is also critical to remember that these are self-reported data and that changes are affected both by underlying changes in incidence and by factors affecting propensity to report them.

Subjective experience of illnesses

How important might these changes be for peoples' experience of health? The increases reported might well be due to an increased tendency over time to report relatively trivial problems, with consequently little significance for health experience. In the *West of Scotland Twenty-07 Study*, currently being conducted by the Medical Research Council in Glasgow, a group of 1600 middle-aged people were asked about chronic ill health. Information was collected on the self-reported degree of limitation and pain associated with particular chronic health conditions. In table 2 we report the patterns for the most frequently reported conditions, where possible reproducing Verbrugge's categories. The table shows the percentage experiencing any limitation, the percentage experiencing serious limitation (that is, reporting that the condition limited their activities quite a lot or a very great deal), the percentage experiencing pain associated with the condition and the percent reporting severe pain.

The majority of conditions cause both limitation (60%) and pain (67%), but only 22% report severe limitation with, perhaps surprisingly, 26% experiencing severe pain. If we examine first the "killer diseases", starting with circulatory diseases other than hypertension, we see that they are high on mild and severe limitation, the angina/"heart attack" category has disproportionate severe pain, while stroke and other heart conditions have below-average pain. Hypertension causes little limitation or pain. Arteriosclerosis appears here under "other

Table 2:
Percent experiencing limitation and pain for selected conditions

	any limitation	severe limitation	any pain	severe pain
Killer deseas	50	20	40	-
Diabetes	82	50	89	37
Heart attack/angina	86	29	61	13
Other heart	29	7	34	7
Hypertension	65	35	29	-
Stroke	64	27	77	24
Bronchitis	42	16	84	22
Other respiratory	80	28	80	25
Mixed categories including killers				
Other blood vessel	77	33	85	38
Stomach ulcer	42	8	86	29
Other digestive	49	4	78	41
Non killers				
Varicose veins	58	19	88	15
Skin	32	14	65	27
Arthritis	70	25	98	48
Other bones/joints	83	33	97	26
Cataract/blindness	75	21	25	-
Deafness	72	31	21	-
Back problems	82	34	98	48

Source: West of Scotland Twenty-07 Study

blood vessel" disorders, which also shows a pattern of high limitation and high pain. Diabetes is low on both. These are all diseases which are increasing in the Verbrugge data. The respiratory diseases show a pattern of high severe limitation and average or high pain. Hernia is aggregated with ulcer, while cirrhosis disappears into other digestive. The digestive categories all show the same pattern of low limitation and high pain.

Of the "non-killer" diseases the musculoskeletal conditions showed by far the biggest increases and they are the conditions showing the most severe combination of reported limitation and pain. The other conditions which increased in the American study, sinusitis and hay fever, are treated as symptoms in the Glasgow study and do not appear here. Similarly, the conditions showing large decrease in Verbrugge only appear in small numbers due to differences in methodology. Varicose veins and haemorrhoids (piles) are low on limitation and high only on mild pain. For the most part, those conditions which increased most in the U.S. study show high limitation or pain or both. Only hypertension, of the rapidly increasing conditions, is relatively benign in its day to day manifestation. Diabetes is also benign. The conclusion is that the quantitative increase in chronic ill health also seems to be concentrated on these conditions, which are qualitatively experienced as the most unpleasant.

Time-series statistics: conclusions

We have expended rather a lot of energy to identify trends in some unsatisfactory data. However, the trends mainly seem to lead in the same direction and we can make the following statements with some confidence.

1. The total occurrence of self reported chronic illness has shown no decline and has probably been increasing both in the U.S. and in Britain and probably also in France over the last two or three decades. Only Sweden shows an overall improvement in experienced health.

2. In the U.S., this process has not been accompanied by any major redistribution in chronic disability within the lifespan. Both middle-aged and elderly people show similar patterns of change.

3. Also in the U.S., there have been sizable increases in the reporting of circulatory and musculoskeletal disease and there is every reason to believe that these increases will have been accompanied by an increasing burden of experienced limitation and pain (a study conducted in Aberdeen, Scotland in 1958 found that 13% of men and 19% of women aged 60-69 reported arthritis. In a second study of the same age group carried out in 1980, the figures were respectively 40% and 49%).

Any straight-line extrapolation from this data would have to strike a pessimistic note. However, the lesson of the circulatory diseases is that unpredicted major changes in both mortality and morbidity can occur in relatively short time-periods and we need to think about the likely stability of observed trends over time. A useful framework within which we can do this, is provided by the Age, Period and Cohort [APC] model.

Age, Period and Cohort

Previous authors have drawn attention to the importance of understanding changing characteristics of successive birth cohorts if we wish to make predictions about future health (Bury 1988). Discursively we can identify successive groups born under different circumstances, attaining different levels of education, adopting different habits and lifestyles and differentially subject to traumas of war, recession and the ills of human society. Intuitively we feel we know that they are going to have different health and disease profiles as a consequence. We need to know how the coming generations will differ from those which went before.

It is equally clear that we live in a changing world. There are a multitude of medical, social, behaviourial and scientific developments and external and contingent events which are likely to affect and modify health in any future population of the elderly in potentially important ways. We find it useful to think of three conceptually distinct influences.

Decomposing change

Ageing effects are considered to be those invariant changes, in mid- and later life generally decrements, which the average organism experiences with passing time. Being fixed they provide the given element in the ageing trajectory.

Cohort effects are due to the accumulated lifetime differences between successive generations. To the extent that these experiences become crystallised properties of an age cohort, they can form a reasonably firm basis for predicting departures from earlier experiences. Changes due to cohort differences once fixed, will be present over a long period, affecting illness patterns as any particular cohort moves through the age structure.

Period effects are, in the nature of things, less predictable. They are not present to be observed in the patterns of past health, but are due to external "environmental" influences. Who could have predicted AIDS and who can read the mind of a Minister of Health? Unfortunately, while these three effects appear to have conceptual clarity, it is much more difficult to identify their relative importance in observed data.

Change and research designs

Typically, in a cross-sectional study, most measures of morbidity or functional limitation rise with age. Older groups will be consistently worse than younger groups. We might therefore conclude that these deteriorations are an inevitable concomitant of ageing. It is equally possible, however, that on some or all of the measures, the older groups had a shared set of experiences, which affected them adversely and caused them to experience worse health in old age. This cohort property is not shared by the younger

groups and they will not follow the same path of decline, but will be healthier in old age.

The problem is, of course, that any observed gradient might consist of a combination of both these influences and unless we have other information which allows us to quantify one of the effects, we cannot decompose the observed gradient into the two constituent components. This matters, since, as we can see, they lead to different expectations for future health.

In a longitudinal design any changes we observe could again result from a combination of two factors. The respondents have grown older and have been subjected to ageing experiences, but the measurements have been made at different points of time and the changes we observe might be due to causally relevant secular changes occurring during that period.

In trend data where we look at the same age group but at successive time points, changes in the data confound cohort and period effects, since we are comparing data on different cohorts measured at different historical moments and either or both could affect any differences we see. Conclusions from the trend data presented above need to be thus qualified.

Applying APC models

A very large amount of effort has gone into devising statistical methods of disentangling these three effects, these cannot be described here. Complex designs which embody elements of cross-sectional, longitudinal and time-series observations can help with interpretation, but unfortunately, as Schaie, Palmore and many others have shown, under any research design the changes we observe will always be a combination of these underlying effects and there is in principle no method of separating them out except by making simplifying assumptions, however complex a statistical model is employed (Palmore 1976; Schaie 1965; Goldstein 1979).

A handful of brave souls have attempted to apply APC analysis directly to morbidity data and make predictions about the relative importance of each of these effects and their implications. Haug and Folmar (1986) analyze data from two waves of the *Cleveland GAO Study* of individuals over 65 in three age bands. One of their aims was to investigate whether "cohort membership or the ageing process (is) more likely to account variability in quality of life among subgroups of the elderly."

Using indicators of cognitive impairment, psychological distress, self-assessed health, interviewer-rated health and activities of daily living, they come to the following conclusions. Both cognitive functioning and psychological distress show ageing declines, there is some indication of cohort effects on cognitive functioning for the oldest age band and males show more age decline than do females on psychological distress but without cohort differences being apparent. Self-assessed health, interviewer-rated health and Activities of Daily Living (ADL) measures show both ageing and cohort effects. The overall conclusion they reach is that, "while cohort differences exist, the ageing process exerts the main influence on quality of life." It is hard to know how much faith to place in this conclusion since their analysis is ad hoc, relying on simple comparisons of the observed cross-sectional and longitudinal changes, and ignoring the confounding due to the underlying effects described above.

Palmore (1986) applies APC analysis to data from the *U.S. National Health Interview Survey* for persons over 65 years old. He takes as indicators: days of restricted activity, days of bed disability, injuries, acute conditions, mild visual impairment and hearing impairment. Between 1961 and 1981, injuries, visual and hearing impairments increase, while bed disability, acute conditions and severe visual impairments decrease. By taking the ratio of the occurrence of each indicator in the elderly only to the occurrence at all ages, Palmore argues that period effects are controlled. He can therefore separate age

and cohort effects in the data, since each observed change is the result of only one effect. All the ratios show a downward trend over the period and since he believes he has controlled for period effects, this can only be a consequence of relative cohort changes. For those indicators where the absolute trend is also downward, it is possible to argue that the elderly are enjoying relatively better health. Where the absolute trend is upward, the decreasing ratios could only be produced by an even larger increase in the all-age figure. He dismisses this pattern as being due to artefacts of the data, since he believes that "it is highly unlikely that illness and disability among all persons actually increased over the past 20 years." On the basis of these findings he argues that: "this finding has profound implications for gerontology (...) professionals need not fear that the increasing numbers of elders will be compounded by increased illness and disability."

Again, it is hard to accept this conclusion. The majority of his data suggest that in absolute terms the elderly are getting sicker and the younger groups are getting sicker more quickly. Presumably, as they age they can only become worse than the current population of elderly. The analysis also points to the danger of analysis based only on selected indicators and two time-points. The Verbrugge presentation of the same data source clearly shows that the majority of a broader selection of indicators show decline and that most of the trend graphs show quite large year-by-year fluctuation, making a two time-point interpretation of small changes extremely unstable.

Identifying the limitations of APC models

Schaie (1981) provides a summary of decades of work on psychological functioning and personality changes in mid- and late life. He emphasises the different impression given by cross-sectional and longitudinal research. In general, fairly large age differences which appear cross-sectionally, are much less evident in longitudinal research, suggesting that the apparent ageing effects are in fact cohort differences. He states that: "most age differences reported in the ageing literature can probably be more parsimoniously interpreted to be generational differences."

There are age-invariant changes in vision, hearing, perceptual speed, energy, arousal level and cardiovascular efficiency, all of which affect psychological functioning. There are also age declines in intelligence and cognitive functioning but these are variable for different components of intelligence and are relatively slight for most dimensions until late in the life span. Apparent changes in performance capabilities usually have more to do with secular changes in the relevance of acquired skills, than decremental changes in the individual, that is they are essentially period effects. He argues that: "much of the literature on intellectual change in adulthood in the past has been based on cross-sectional data that have exaggerated generational differences such as educational levels, nutritional histories and the like."

Equally importantly, he argues that most of the changes he discusses are sufficiently small to be within the adaptive range of the individual and can be expected to have little behaviourial impact until the eighth decade of life. The following conclusions seem to be warranted:

1. We cannot ignore the problem of the confounding of APC effects in trying to interpret any trends in cross-sectional, longitudinal or time-series data. Most of us have a general perception of the problem, but we tend to gloss over it, often implicitly setting one of the effects to zero in order to interpret an observed change as being solely due to ageing or to cohort succession or historical changes, without explicitly considering how reasonable this is. We need to have these issues firmly in the forefront of our minds.
   To take a single example, we would probably wish to argue that the apparent

dramatic increase in hypertension in the Verbrugge data is an artefact of changing medical practice. The data does not tell us this, and the figure might equally well contain real changes in the incidence of the condition. We need further information of a different sort.

2. The formal statistical models, which have been developed to deal with the problem, have, for the most part, run far ahead of the data sets to which they might be applied. Even the most sophisticated models are hedged with qualifications and subject to some variant of the confounding of effects described in simple terms above. Attempts to apply any formal APC analysis to general health and disability data seem to have been dismal failures. This is likely to be the case into the foreseeable future.

3. Careful consideration of a wide range of data utilising different data collection strategies within a general awareness of the APC issue, such as that carried out by Schaie, can, however, lead to useful generalisations.

4. Available evidence gives us no reason to echo Palmore's general optimism over the future physical health of the elderly, but if Schaie is correct then the elderly without pathology are likely to show more robust psychological profiles and as a consequence may be more able to adapt and adjust.

Applying APC-models to morbidity data

It is clear that we lack the sort of dense continuous information which would allow us to apply formal APC models to morbidity data. There are useful insights to be gained though from looking at mortality patterns where, with the usual caveats about death certification practices, good-quality information is available over a long period of time. We can, for example, make reasonably confident predictions about the importance of cohort changes in the incidence of lung cancer. Inspection of age-specific mortality graphs in the U.K. clearly demonstrate cohort effects for men with little sign of period changes, while females show no such affects at this point in time (Alderson and Ashwood 1985). Setting this data along with trend data in smoking, we can make reasonably confident predictions about differential future trends for the two sexes. We can expect between a two- and fourfold decline for men over the next 30 years, with estimates for females showing at best an unchanged incidence and at worst a threefold increase over this period. This gives us some idea of the likely morbidity associated with the condition.

Lung cancer has a very clearly understood aetiology. Our ability to make specific predictions is usually critically dependent on both our knowledge of the relevant risk factors and the existence of reliable time-series data for these risk factors. It is generally held that changes in U.S. patterns of circulatory deaths are due to changes in a network of behaviourial risk factors, including smoking, exercise and diet along with control of obesity and hypertension, although the exact relationships are contested. These are usually conceptualised as period specific changes in voluntaristic elements of lifestyle, as such they are subject to extension or reversal within variable time spans. Hence arguments for the relevance of health education.

On the other hand, Barker (1989) has very recently published persuasive data showing strong relationships between birthweight and subsequent mortality due to ischaemic heart disease and chronic obstructive lung disease. If these findings are upheld, then it would appear that mortality from these causes is in part determined by cohort characteristics established at or before birth. The implication is that future patterns have already been partially set by influences which occurred a long time ago and are not subject to current modification. They could,

however, in principle be predicted since appropriate data on changing birthweight could be assembled.

Our understanding of the likely future is naturally affected by our changing understanding of the basic epidemiology of diseases. Unfortunately, as Brody (1985) points out: "... we must admit that we do not know causes of, and cannot prevent, most chronic disease. Only a fraction of the cancers, heart diseases, strokes, neurological diseases and arthritides are understood."

In the final analysis, the limits to prediction are set by events which are still to happen. Riley (1989) states that taking a historical perspective, "is to acknowledge that the past does not 'teach us' what the future holds, but that it does teach us that the future will be interesting because it will blend the expected and the unexpected."

In estimating future mortality from lung cancer, Alderson and Ashwood had to make best guesses as to future smoking behaviour. Looking around us we can identify a multitude of changes or possible future changes, which might have far-reaching implications. It seems that not a week passes without an announcement in the scientific press of a breakthrough in the understanding of dementia. Once it might turn out to be true, and the management of dementia might be transformed as Parkinson's disease was by Dopamine. The recent introduction of charges for eye tests in Britain has apparently reduced the number of tests by 40%. Who can guess what affect that is going to have on sight of the population?

Mortality and morbidity

The final element we consider is the relationship between changing patterns of mortality and morbidity. The largest claims and the most vigorous debates about the future health of the elderly have centred on different models of this relationship leading to very different predictions. We cannot ignore the debate. As with the age cohort and period issue, the underlying ideas are quite simple, but the elaborations, the complex and crucial parts of the data absent. We observe significant declines in mortality. The diseases that kill are often the same diseases which cause chronic ill health. Interventions, secular changes and cohort successions, which are bringing changes in observed mortality, must surely also be affecting morbidity, but how?

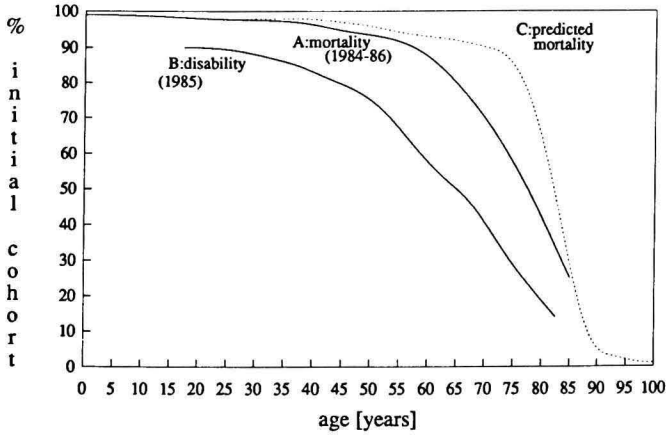
Manton and Soldo (1985) suggest that the issues can be most clearly understood graphically. Fig. 2 shows a synthetic life-table for a hypothetical birth cohort, subjected to the age-specific death rates observed in Britain in the period 1984 to 1986. The dotted part of the line is a projection into extreme old age. Beneath that we have graphed the proportion of the survivors at each age, suffering from chronic limiting illness from the 1985 *British General Household Survey*.

The total area under the upper curve, line A, therefore gives a realistic representation of the total person years of life allotted by fate to the cohort. The area under the lower curve, line B, represents the part of that total which is free from limiting illness, while the area between the curve shows the proportion of the cohort life-years spent with limiting illness.

We can consider the implications of various changes in mortality and morbidity in terms of this graph. We are primarily interested in the quantity of morbidity as indexed by the area between line A and line B. This is affected both by changes in the relative positions of the two curves along the age axis, a general change in life expectancy or in the onset of chronic illness, and by changes in the shape of either curve, changes in the age-specific pattern of death or morbidity onset.

If there were a general increase in life expectancy at all ages, without any change in morbidity, then line A would be displaced to the right and the area under the curve would increase, implying an increase in total morbidity. Quite simply, people live longer in

Fig. 2:
Age-related changes in disability and mortality



Sources: disability-1985 UK General Household Survey
mortality-1986 OPCS mortality statistics

a morbid state. A postponement in the average age of onset of chronic illness would move line B to the right and by closing the gap, would imply a reduction in overall morbidity. These are self-evident observations.

Equally, we might observe changes in age distribution of death or first onset of disease. Line C represents the future mortality pattern as predicted by Fries (1983). He argues that premature deaths in middle age will be prevented, but that there is a biological upper limit to the average life span, such, that the mortality curve will be increasingly "rectangularised" rather than being simply displaced to the right. Without any change in morbidity this would inevitably lead to an increase in total years of ill health (the area between the curves). In fact, Fries also argues for a parallel change in the age of onset of morbidity, giving a similar "rectangular" shape to the morbidity curve. This would increase the total area under line B, that is, extend the total quantity of disease-free life and "compress" morbidity into a shorter period later in life. It is easy to see that predictions about the different possible changes in the relationships of the lines within the graph give rise to optimistic or pessimistic visions of the future health of the elderly. Almost all possible patterns have been advanced at some time (Manton and Soldo 1985).

The current situation is that reasonably high-quality data exists on mortality changes for many advanced countries. We can make empirically well-founded statements about the shape and location of line A (this is not to say that interpreting the data is easy and that agreement is universal as to what is happening). As we have indicated earlier, there is little high-quality data which could cast light on empirical changes in morbidity. Thus far we have described the model as if mortality and morbidity vary independently. Intuitively we feel that this is unlikely.

The rectangularisation debate: linking morbidity and mortality

Almost everyone who has addressed these issues agrees that what we ultimately need is a good model, or good models linking mortality and morbidity (and probably also disability). If we had these models, prediction

would be possible. The best-known strong unifying model to date is that presented by Fries and Crapo (1981), and it is on this model that their particular predictions stand. They argue that many of the important chronic diseases begin early in life and progress slowly and asymptomatically as part of a natural ageing process, till they reach some threshold beyond which they begin to cause the individual to experience symptoms and compromised health. Ultimately, they progress to a point at which they become life-threatening. Modifications to the risk factors for these conditions can reduce the rate of progression, effectively produce a shallower gradient of decline, thereby postponing the time at which the symptomatic threshold is crossed. At the same time, the second threshold beyond which they become life-threatening is also postponed, explaining observed falls in age-specific death rates for many causes of death.

The theory is essentially a straw man, a heuristic device to further a debate rather than a set of tight short-term predictions, and has been heavily criticised, perhaps inappropriately, for its lack of correspondence with current reality. Fries himself views it as a statement about the future and acknowledges that short-term developments might actually show an increase in morbidity. Can we then say a bit more about the real situation as it is now? The claim that death is becoming increasingly rectangularised has been widely contested. It is partly a matter of the time perspective used. Fries' and Crapos' analysis, based on simple visual inspection of successive survivorship curves over this century, does seem to show something like rectangularisation, but the picture is heavily dominated by changes to mortality affecting younger age groups early in the century, an earlier phase in the demographic transition than the one we are now concerned with. Manton (1982) has looked at trends only since 1962 and only for survivorship at older ages. He finds no evidence of rectangularisation. It looks as if line A on the graph is slowly being displaced to the right. Bury (1988), reports a ninefold increase in the number of centenarians in England over the last thirty years.

The relationship between mortality and morbidity is simply much more complex than Fries suggests. It would be impossible to cover all the arguments in detail, but we can list some of the main issues. The Fries model applies only to certain types of chronic ill health. There is currently no reason to suppose that changes in mortality patterns from the diseases of middle age are linked in any way to arthritis, osteoporosis, dementia and many other of the common diseases of old age. If this is the case, then, unless the age-specific incidence of these conditions changes for unrelated reasons, an increase in longevity can only be accompanied by increased prevalence of these debilitating conditions. For most conditions, changes in observed mortality probably consist of components due to primary intervention through changing risk factors, but also to secondary intervention, to prevent the potentially fatal sequelae of currently symptomatic conditions and to changes in the case fatality, following potentially life-threatening episodes. Even for well-documented phenomena, such as the recent rapid decline in stroke mortality, the balance of importance between these components is unclear and contested (Manton and Soldo 1985). Whether reduced age-specific mortality goes along with reduced morbidity, will depend on the balance between these different components and the nature of the intervention affecting each. Some secondary interventions must imply increased survivorship with compromised health.

There are indications that early mortality might affect subgroups of the population with specific susceptibilities. Should they survive, they may be vulnerable to further particular conditions, generating different patterns of frailty in future elderly generations than those currently observed. While deaths in middle age have fallen substantially this century, they have by no means disappeared. Currently

18% of males and 9% of females are expected to die in the U.K. in the ten years between 55 and 65 (Bosanquet 1987). We know very little about the health profiles of these early fatalities or the likely implications should they survive. Manton has re-analysed data from the *Duke longitudinal study* and identifies a subgroup who die early with multiply compromised health. Feldman (1983) has argued that observed increases in work disability in the U.S. in the 70s were due, at least in part, to increasing survivorship in the group of middle-aged disabled.

Interestingly, another very recent attempt to take a broad view of historical change as a basis for prediction, uses some of the arguments above to arrive at a conclusion very different from that of Fries. James Riley (1989) undertakes a historical review of the relationship of mortality and morbidity over the last 400 years. On this basis he advocates a version of "insult accumulation theory." Risk of ill health at any age is a function of accumulated illness episodes at younger ages. As case fatality at younger ages diminishes, so the accumulated "morbidity risk schedule" at any age increases and the risk of illness increases. "The age-specific risk of being sick should be expected to rise when the death rate declines, because the people who do not die at each age under the new mortality regime, bear a higher risk of being sick than the average in their age group under the old regime."

The detailed implications of any change in age-specific death rates, depend on the specifics of the "winnowing" by premature death (just how selective it is) but, projecting forward into the twenty-first century, Riley believes that the pattern will be one of increasing morbidity.

Having considered a large number of papers adopting a variety of approaches, we do not consider that any of them arrive at an informed and balanced account of the likely medium and long-term future. Most content themselves, as we have largely done, with a statement of the issues and problems. We therefore feel that any announcement of the "Age of Delayed Degenerative Diseases" should be postponed, pending further investigation.

It is clear that there is a very general absence of high-quality data to allow examination of trends in morbidity. All possible sources of such data have their inherent limitations. Population surveys carry all the problems of relying on self-reports, statistics from service contact are partial and differential in their coverage, screening and physical examination studies are expensive and therefore infrequent and likely to be of limited scope. On the other hand, we need to be very cautious indeed about making inference about morbidity prevalence from mortality statistics. Any initiative to extend and improve basic data, gathering from a variety of relevant sources, would be welcomed.

Most of the short term self-reported data which we have presented indicates a slight general secular increase in self-reported morbidity. At the same time, Verbrugge and many others have pointed to large and significant declines in all causes, and most cause-specific mortality. Something fairly dynamic is happening to affect mortality in at least some of the advanced societies. On the whole, models which propose as a common underlying mechanism the postponement of illness and mortality by a general improvement in underlying health and changing health behaviour, do not seem to square with the observation that reported increases in morbidity are common to both middle and old age. Even if we propose a secular tendency towards reporting more ill health, which might in fact mask an actual decrease in morbidity, we would expect to see a differential shift in the burden between age groups. We are left with something of a paradox which we cannot at present unravel.

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