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Determining Health Expectancies

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Compression of Morbidity

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INTRODUCTION

Traditionally, a decline in mortality was considered to reflect a decline in morbidity in the population. Nowadays, in low mortality countries where improvements in life expectancy are mainly caused by mortality reductions from chronic diseases at older ages, serious doubts exist as to whether longer life means better health for the surviving population. It has been increasingly recognised that a consequence of these mortality reductions might be that the burden of morbidity in the population expands. Nevertheless, a more optimistic view is prevalent, which anticipates that by adopting healthier life styles, the onset of chronic diseases and related disability can be postponed or even prevented, so lifetime spent with chronic diseases and related disability reduces (Fries, 1980, 1983, 1989, 1993). Whether additional years are spent with morbidity and disability or whether increases in life expectancy are accompanied by decreases in years with morbidity – that is, with a compression of morbidity – has been a major focus of the Network on Health Expectancy (REVES). This question is important as today the aim of science and medicine is more to reduce the number of years that people spend diseased or disabled rather than to lengthen life (Greengross *et al*, 1997).

Since Haan (1989) introduced Fries' work on compression of morbidity in the Network and discussed the complex relation between mortality, morbidity and population health at the first REVES meeting in Quebec, this topic has received much attention. Besides trend studies of changes in health expectancy over time, aiming to assess whether compression of morbidity is actually occurring, substantial work has been done to define operationally compression of morbidity and to measure this process. In addition, characterising and understanding the dynamics underlying changes in population health in

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general, and compression of morbidity in particular, have become increasingly important research aims within REVES. Before paying attention to these issues, we discuss the compression-of-morbidity hypothesis and alternative views on the association between morbidity, mortality and population health, because points raised in this debate have found their expression in research in this field. The outcomes of trend studies of changes in health expectancies over time are described in a separate chapter (Chapter 4).

COMPRESSION OF MORBIDITY AND ALTERNATIVE VIEWS OF THE ASSOCIATION BETWEEN MORBIDITY, MORTALITY AND POPULATION HEALTH

There is no unequivocal association between the length of life of populations and their state of health. This is clearly illustrated by comparing health expectancies between racial groups in the United States. For Asians, longer life is associated with fewer years lived in poor health, whereas in Native Americans relatively longer lives are accompanied by an extended period of morbidity. Blacks live the fewest years, and a high proportion of these years is spent with a chronic health problem. Hispanics also live fewer years, yet the period spent with a health problem is relatively compressed (Hayward and Heron, 1999). The finding that women live longer but spend more years in poor health than men, while higher socioeconomic groups live longer but spend fewer years in poor health than lower socioeconomic groups (Robine and Ritchie, 1991), also demonstrates the complexity of the relationship between morbidity, mortality and population health.

The survival-curve model, originally introduced by the WHO in 1984 (World Health Organization, 1984), enables a direct assessment of the consequences of increased longevity for population health and can be used to describe and visualise the possible associations between morbidity, mortality and population health (Figure 2.1). The model captures the dynamics of mortality, disability and morbidity in a relatively standardised way. It consists of three survival curves, the mortality curve, the disability curve and the morbidity curve, which represent the proportion of individuals in a cohort who can expect to survive, to survive without disability and to survive without morbidity.

The area below the mortality curve presents total life expectancy, while the area below the disability and morbidity curve represent life expectancy without disability and morbidity, respectively. Examples of life expectancy without morbidity are life expectancy without hip fracture history (Herrmann *et al.* 1996) or heart disease-free life expectancy (Barendregt and Bonneux, 1995). Life expectancy without disability includes life expectancy without ADL disability, labelled as active life expectancy (Katz *et al*, 1983). Besides, other definitions of disability have been used to calculate disability-free life

In summary, the relationship between morbidity, mortality and population health is complex, and different mechanisms, with sometimes opposite effects on the lifetime spent in good or poor health, have been suggested in literature.

ASSESSMENT OF COMPRESSION OF MORBIDITY

Since there is no unequivocal association between the length of life and population health, a longer life may imply either an expansion or a compression of morbidity. Compression of morbidity, in the presence of an increasing life expectancy, is generally considered a favourable development of population health, as it implies that years in poor health are being replaced by years in good health. The combination of a compression of morbidity and increased longevity means that both disability-free and total life expectancy increase and that the increase in disability-free life expectancy exceeds that in total life expectancy. As a result the period in poor health is being compressed. A compression of morbidity without an increase in life expectancy should not necessarily be considered a favourable development. When it is due to a dramatic increase in premature mortality, nobody will evaluate a decline in the time spent in poor health in a positive sense. However, in general, compression of morbidity without a decline in total life expectancy is considered as favourable, and has stimulated public health policy and research to devote special attention to this issue. It is obvious that an unambiguous definition and tools to assess compression of morbidity are indispensable for progressing towards this goal. These issues have received high priority within REVES.

DEFINITION OF COMPRESSION OF MORBIDITY

When using the term 'compression of morbidity', Fries, the inventor of the compression-of-morbidity hypothesis, referred to the compression of morbidity into the shorter span between the increasing age at onset of morbidity and the fixed occurrence of death (Fries, 1980). Morbidity can be caused by chronic diseases and ageing and is used interchangeably with disability in Fries' work. Compression refers to a decrease in the number of years spent with disability. In the situation originally described by Fries, wherein the age at death is fixed, the increase in the period without disability equals the decrease in the period with disability. Delaying the onset of disability, so that people may expect to live longer in a state of good health, was sometimes considered as compression of morbidity (Robine and Ritchie, 1991). In more recent work (Fries, 1983, 1984, 1993), Fries used a more comprehensive definition of compression of morbidity, stating that the average age of onset of significant permanent infirmity may increase more rapidly than does life expectancy, thus shortening respiratory diseases and traumatisms) rather than decreased. In other words, a reduction in disability prevalence by age was accompanied by an increase in the prevalence of disabling diseases (by age). It was shown that a reduction in the disabling impact of these diseases explained the observed decline in disability rates in 1991 as compared to 1981 (Figure 2.2).

To be more specific, there were lower probabilities of having disabilities in men with a disease of the nervous system, a cardiovascular disease, an osteoarticular disease or a traumatism and higher probabilities in men with an eye disease. For women the results are almost the same, except for the increase in the disabling impact of mental diseases. These outcomes might indicate that the rate of disease progression is slowing down (as suggested by the dynamic equilibrium hypothesis), although it is also possible that the propensity to report diseases, especially the less severe forms, has changed (Robine *et al*, 1998). Regarding the consequences of changes in diseases for compression of morbidity, two observations arise from this study. First, it is the net effect of changes in the prevalence of chronic diseases and in their disabling impact that counts. Second, an expansion of the period with disabling chronic diseases might co-occur with a compression of the period with disability.

Mortality

The extent to which major causes of death end disabled and non-disabled life was the main subject of a study from Hayward which used an extended multistate life-table model differentiating deaths in the active and inactive state by cause (Hayward *et al*, 1998). This was the first study to include information on both the health state preceding death (active versus inactive) and the cause of death (heart diseases, cerebral diseases, cancer and other diseases). It was found that functional decline need not necessarily precede death. Most of the deaths (68.5%) in men age 70 occurred in the active state whereas in women at this age deaths are roughly split up between the two health states. At older ages, larger proportions of deaths occurred in the inactive state, but still a sizeable proportion of deaths occurred in persons who were still active. This concentration of active deaths suggests that extending active life could be accomplished partially by lowering mortality rates among persons without functioning problems. A further analysis of the effect of eliminating specific causes of death on active and inactive life expectancy showed that for heart diseases most of the gains in life expectancy at age 70 for men are felt in terms of increased active life. Of the three additional years men can expect to gain when heart disease would be eliminated, almost two years would be added to active life. The results, however, differed by age and sex. With increasing age, a gradual shift was found, such that the elimination of heart diseases at older ages would add more years of inactive life than of active life. In women aged 70 most of the years gained through the elimination of heart diseases would be

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and to mortality can be derived (Crimmins *et al*, 1994; Guralnik *et al*, 1993; Nusselder, 1997; Nusselder *et al*, 1996a). To date, no time series of these transition rates are available which allow for comparisons over time. Nevertheless, transition rates for one period (e.g. two years) can be, and have been, used to obtain a better understanding of the association between changes in morbidity and disability on the one hand, and changes in health expectancy on the other hand. We report the results of two studies, presented within REVES, which simulated changes in these transition rates and examined the consequences of these changes for health expectancy. By using a multistate life-table model, the transitions between the disability states and from each of these states to death were explicitly modelled. These transition rates determine total mortality – and thus total life expectancy – and health expectancy.

Crimmins and colleagues (Crimmins *et al*, 1994) used a multistate life-table model with four living states (i.e. no functioning problems, some functioning problems, unable to provide independent living and unable to provide personal care) for the US population aged 70 and over. In addition to these disability states, they also used a dichotomous categorisation by defining having disability as being unable to provide independent living or to provide personal care. The life-table simulations showed that decreasing incidence and progression rates of disability (i.e. decreasing the chances of getting worse) while keeping state-specific mortality rates and improvement rates (i.e. the chances of getting better) constant, produces an increase in total life expectancy and in disability-free life expectancy, and a reduction in the number of years and proportion of life with disability (Table 2.1).

The same direction of the effects, although smaller, was found when improvement rates (i.e. the chances of getting better) were increased. These findings were supported by a Dutch study of Nusselder *et al* (Nusselder, 1997; Nusselder *et al*, 1996a). The latter study simulated changes in transition rates using a two-state life table (i.e. non-disabled and disabled), which also included younger age groups (age 30 and over). The interpretation is as follows: due to improvements in disability rates, more people maintain and/or regain their health. As a result, the period without disability extends at the expense of that with disability. The rise in total life expectancy as well when disability rates improve is caused by the decrease in the proportion of persons with disability, who are exposed to higher mortality risks. Because the increase in disability-free life expectancy exceeds that of total life expectancy a compression of the period with disability (in an absolute and a relative sense) occurs.

Similar simulations suggested that reducing mortality rates (from one or more states), while keeping the disability rates constant, increases life expectancy in all states and the proportion of life with disability (Crimmins *et al*, 1994; Nusselder, 1997; Nusselder *et al*, 1996a). Increased survival of non-disabled persons (e.g. by improved acute care units) pushes more persons into the oldest-old age groups, where the risks of becoming disabled are high. Increased

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disability. Elimination of cancer would therefore result in an expansion of morbidity (in absolute and relative sense, according to the adjusted classification). Whether elimination of both disabling and fatal diseases (e.g. heart diseases and chronic non-specific lung disease) would result in compression or expansion of morbidity depends on the extent to which they cause mortality and disability. For example, eliminating heart disease, where the impact on total mortality exceeds the impact on disability, would lead to an expansion of morbidity in both an absolute and a relative sense. As far as chronic non-specific lung disease is concerned, however, elimination would lead to both absolute and relative compression of morbidity.

To summarise, when highly fatal diseases with a low disabling impact are eliminated, or when only the mortal consequences of chronic diseases are eradicated, increased survival will lead to an expansion of the number of years with disability, even if initially the survival of active persons increases. When changes occur earlier in the disease process and disability is prevented as well, a compression of morbidity (in an absolute and a relative sense) occurs, unless prevention of this disease also decreases mortality substantially. In the latter situation, the net effect depends upon the disabling impact of the disease and its case-fatality.

IMPACT OF SPECIFIC RISK FACTORS

Since disability and mortality share common risk factors, the net effect of changes in the risk factor distribution (or in the excess risks associated with exposure to that risk factor) in terms of compression of morbidity is not obvious. Whether reducing the risk-factor exposure would result in a compression of morbidity depends upon the net effect of two opposite effects. First, as a result of the reduction in exposure, lower disability rates produce an increase in life expectancy without disability and a reduction in life expectancy with disability. Second, a reduction in exposure increases survival. As a result, people live longer and will be longer exposed to the (reduced) risk of disability. On balance, life expectancy with disability decreases when the effect of the disability reduction on the number of years with disability exceeds that of increased survival.

Smoking is an important modifiable risk factor which causes substantial mortality (Doll *et al*, 1994; Nam *et al*, 1995; US Department of Health and Human Services, 1990) and disability (Branch, 1985; Guralnik and Kaplan, 1989; Kaplan, 1992; LaCroix *et al*, 1993; Rogers *et al*, 1994). The consequences of a hypothetical elimination of smoking for health expectancy in the Netherlands have been estimated in two studies, which used different data and a different methodology. Based on a multistate life-table model with a non-disabled and a disabled state, Nusselder and colleagues (Nusselder *et al*, 2000) estimated that eliminating smoking would produce a substantially larger gain both the proportion of life spent infirm and the absolute length of the infirm period.

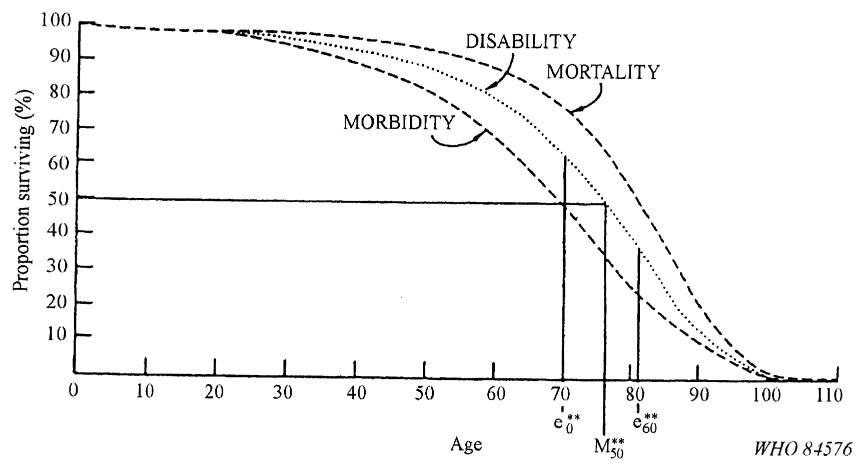
Disability-free Life Expectancy and Compression of Morbidity

The relationship over time between total life expectancy and disability-free life expectancy can be used to assess whether compression of morbidity occurs. When total life expectancy is fixed, a longer life without disability can be considered as compression of morbidity. It is obvious that according to the more recent definition of Fries, an increase in the number of years in good health does not automatically imply a compression of the period with disability. Several studies have shown that when life expectancy increases, a gain in disability-free life expectancy may be accompanied by an increase in years with disability (Mathers, 1994; Robine and Ritchie, 1991; Wilkins *et al*, 1994). In the presence of an increasing life expectancy, which is observed in most low morbidity countries, an increase in disability-free life expectancy may be accompanied by a constant, an increasing or a decreasing life expectancy with disability. Thus, in this context, an increase in disability-free life expectancy only means that not *all* years gained are equivalent to extra years with disability. It may well imply an increased burden of disability for both individuals and society as whole.

Considering changes in disability-free life expectancy, while disregarding changes in life expectancy with disability, may give a too optimistic assessment of changes in population health. Therefore, REVES proposed to consider the evolution of the 'complete' health expectancy, including the time spent in both the 'healthy' and 'unhealthy' states. This reflects the recommendation of the Network to use the term 'health expectancy' as a generic term to refer to the expected duration in specific health states, including both 'healthy' and 'unhealthy' states (Mathers *et al*, 1994). Based on the complete health expectancy, compression of morbidity can be measured in two ways, depending on whether a dichotomous set or a larger set of discrete states is distinguished, and on whether a summary measure is calculated by applying weights to the expected years in the various states.

Classifying Changes in Life Expectancy With and Without Disability in Terms of Compression of Morbidity

When only two health states are distinguished, or when a dichotomy based on several health states is made, changes in health expectancy can be classified in terms of compression (and expansion) of morbidity. Robine and Mathers (1993) proposed such a classification, based on the magnitude of changes in life expectancy with and without disability and the proportion of life without disability. A distinction was made between absolute compression, relative



e_0^{**} and e_{60}^{**} are the number of years of autonomous life expected at birth and at age 60, respectively. M_{50}^{**} is the age to which 50% of females could expect to survive without loss of autonomy.

Figure 2.1. Observed mortality and hypothetical morbidity and disability survival curves for females in the United States. From World Health Organization (1984). Reproduced with permission

expectancy (see Chapter 12). The area between the disability and mortality curves is life expectancy with disability. The survival curves are determined by age-specific mortality, disability and morbidity rates and are, together with total life expectancy and life expectancy with(out) disability and morbidity, derived from a life-table model. Changes in these mortality, disability and morbidity rates may alter the different survival curves and thus total life expectancy and life expectancy with and without disability and morbidity. These survival curves need not necessarily move in the same direction and to the same extent. Three different views exist of the association between changes in these curves and their effect on the length of life and the length of lifetime spent with and without morbidity and disability.

THREE DIFFERENT HYPOTHESES

The Compression-of-morbidity Hypothesis

The compression-of-morbidity hypothesis (Fries, 1980, 1983, 1989, 1993) states that the length of human life is fixed, that life expectancy is reaching this limit, that chronic diseases and related disability can be postponed to older ages by changes in life style, and that the physiological (e.g. serum cholesterol) and psychological (e.g. memory) markers of the ageing process can be modified.

Fries argues that there is a natural limit to the life span (i.e. the genetically endowed limit to life for a single individual if free of all exogenous risk factors). A linear decline in organ reserve with increasing age parallels the decline in the ability to restore homeostasis. Eventually, the smallest perturbation prevents homeostasis being restored and causes 'natural death', which may even occur without diseases. Chronic diseases can be postponed or even prevented by adopting a healthy life style, such as avoiding overweight, quitting smoking and doing exercise. As a result, the number of years with morbidity can be compressed between the increasing age at onset of morbidity and the fixed mean age at death. Expressed in terms of the survival curves of the WHO model, this hypothesis states that the mortality curve does not change substantially, whereas the morbidity and disability curves move outwards. As a result the area between the mortality and morbidity/disability curve (i.e. life expectancy with morbidity/disability) decreases.

The Expansion-of-morbidity Hypothesis

The expansion-of-morbidity hypothesis anticipates that mortality reductions produce more years with morbidity and related disability (Gruenberg, 1977; Kramer, 1980; Olshansky *et al*, 1991; Verbrugge, 1984). Mortality reductions might produce this expansion in two ways. First, the increased survival of persons with chronic conditions due to medical interventions might prolong the lives of the seriously chronically ill. In Figure 2.1, the mortality curve moves outwards, whereas the other curves remain unchanged. In addition, increased survival might push the saved population into the oldest-old ages where the risks of non-fatal diseases of ageing are high. Hence, declining mortality from fatal diseases produces a population with high risks of chronic morbidity and related disability and thus leads to a shift in the distribution of causes of disability from fatal to non-fatal diseases associated with old age. This second mechanism cannot be easily illustrated with the WHO model, although it is taken into account in the life-table model by default.

The Dynamic-equilibrium Hypothesis

The intermediate hypothesis, the 'dynamic-equilibrium' hypothesis (Manton, 1982), also states that increased survival produces an increase in years with morbidity. However, according to this hypothesis, the years with *severe* morbidity and disability remain relatively constant, because medical interventions or life style changes reduce the rate of progression of chronic diseases. When disability is interpreted as severe morbidity, this hypothesis states that the area between the mortality and morbidity curves increases, because the mortality curve moves faster outwards than the morbidity curve. The area between the disability (here: severe morbidity) and mortality curves does not increase according to this hypothesis.

DIFFERENT MECHANISMS

Although the debate on which hypothesis is most likely to occur in reality has still not been solved completely, it shows the possible mechanisms that affect population health. Firstly, the debate has made clear that whether or not a longer life is accompanied by additional years without morbidity and disability largely depends on the phase in the disease process where changes in morbidity and mortality occur (Crimmins and Ingegneri, 1993; Manton, 1982). If mortality declines only because people do not develop the disease any more, develop the disease later in life, or recover from the disease, the mortality reduction is accompanied by an increase in years without morbidity and disability. On the other hand, if mortality declines among persons who already have the disease, if the disease progression is reduced or if only death is delayed, then the mortality reduction is not accompanied by an increase in years without morbidity, but only by additional years spent in poor health. Secondly, proponents of the expansion-of-morbidity hypothesis (Olshansky *et al*, 1991; Verbrugge, 1984) have convincingly argued that an additional mechanism may affect the duration in poor health when life expectancy increases. That is, reductions in morbidity and mortality (irrespective of the phase of the disease process where they occur) may increase the number of persons who survive to advanced ages (where the risks of chronic diseases and related disability are high). As a result, the time spent with disability expands. In fact, in this line of reasoning, changes in mortality and morbidity affect the distribution of the population (in this case by age, and age is strongly associated with disease and disability), which – in turn – affects future morbidity and mortality.

Other, more complex, mechanisms operating indirectly through changes in the population distribution may explain why a longer life does not necessarily mean better health of the living population. The first relates to mortality selection. Due to lower mortality, frailer and weaker persons become a relatively more numerous group in the population, and as a result future mortality and morbidity in the population may increase (Alter and Riley, 1989; Deeg, 1997; Manton, 1982). The second relates to the risk-factor distribution of the population. Since chronic diseases can share common risk factors or since one disease can act as a risk factor for a second disease, lower case-fatality of one chronic disease may imply higher susceptibility to morbidity and further mortality. On the other hand, morbidity at one point in the life course might also have negative consequences for morbidity and mortality later in life. Damage from illness or injury may increase the susceptibility to disease and mortality later in life (Alter and Riley, 1989). Alternatively, experiencing greater mortality risks might exhaust resources to escape the hardships that killed their age peers and thus leave little resilience to avoid adverse health (Deeg, 1997).

compression, relative expansion and absolute expansion of morbidity. A decrease in life expectancy with disability was classified as absolute compression of morbidity and a decrease in disability-free life expectancy as absolute expansion of morbidity. A decrease in the proportion of life with disability, in combination with an increase in the number of years with disability, was labelled as relative compression of morbidity. Finally, a decrease in the proportion of life free of disability, in combination with an increase in the number of years free of disability, was classified as relative expansion of morbidity.

In further research it turned out that the classification did not fit each situation well. The classification proved useful when total life expectancy was constant or increasing, but not when it was decreasing. Scenario calculations by Nusselder (Nusselder, 1997) showed that a decline in life expectancy without disability occurred when mortality rates increased. On the basis of the above-mentioned classification, this situation should be labelled as absolute expansion. But one may well wonder why the same situation should not be classified as absolute compression, as life expectancy with disability was declining as well. To overcome this problem, Nusselder has adjusted the classification (Nusselder, 1997). In this adjusted classification, in distinguishing between absolute compression and absolute expansion of morbidity, the change in the number of years with disability (or the difference between total life expectancy and life expectancy without disability) is decisive. A decline in the number of years with disability points to absolute compression and an increase to absolute expansion. Distinguishing only between absolute compression and expansion of morbidity may, however, sometimes be too crude. For example, it might be useful to distinguish between a situation of slight absolute expansion of morbidity being accompanied by a substantial gain in years without disability, as opposed to a situation of substantial increase in years with disability being accompanied by only a slight increase in disability-free life expectancy. The change in the percentage of life with disability provides information on the distribution between years with and without disability in the gained years as compared to the baseline situation. Therefore, as in the original classification, relative definitions of compression and expansion of morbidity are used. To determine whether relative expansion occurs, the change in the percentage of life with disability is considered, with an increase of this percentage indicating relative expansion of morbidity. Any particular situation can be classified as a combination of absolute compression or expansion, and relative compression or expansion. In the adjusted classification, compression and expansion are complementary to each other (i.e. absolute compression implies the absence of absolute expansion). Although the original classification can be used in most situations, the adjusted classification fits *any* situation and is more transparent.

Classifying Changes in Weighted Health Expectancies in Terms of Compression of Morbidity

Sometimes, a summary measure is calculated by applying weights to the expected years in the various states. These states can be either disease or disability states (or states based on other generic health measures). In the first case, the weighting includes a translation from the burden of disease to the burden of disability, taking into account that diseases differ in their severity of disability. Weighted health expectancies are known as health-adjusted life expectancies (HALE) or disability-adjusted life expectancies (DALE) and are described in more detail in Chapters 12 and 13. In current research practice, the DALE is based on disease states. Based on this indicator, absolute compression of morbidity is defined as a reduction in the difference between total life expectancy and DALE, i.e. a reduction in life expectancy with disability (Barendregt and Bonneux, 1998). Relative compression of morbidity is defined as a decline in the ratio of life expectancy with disability and total life expectancy (i.e. the percentage of life with disability). As in the revised classification for the situation with two states, compression and expansion of morbidity are complementary to each other.

THE LIFE TABLE AS MEASUREMENT TOOL OF COMPRESSION OF MORBIDITY

The value of disability-free life expectancy and life expectancy with disability can be obtained from a life table which includes both mortality and morbidity data. Time or duration is the common denominator in which mortality and morbidity are expressed. By including both mortality and morbidity data in the life table, the consequences of changes in mortality and morbidity in different phases of the disease process, as postulated in the compression versus expansion debate, are taken into account. In addition, changes in the population distribution induced by these changes are handled by default. This means that the side effect of increased survival, which pushes persons into the oldest-old ages with high risks of disability, is taken into account in the life table.

The Sullivan Method Versus the Multistate Life-table Method

Two different types of life tables are used to calculate health expectancy: the prevalence-rate life-table model (commonly known as the Sullivan method) and the multistate life table. The Sullivan method is based on the prevalence of disability, whereas the multistate method is based on transition rates between the disability states and to the dead state. Health expectancy can be calculated with the Sullivan method from readily available data on the prevalence of disability and a standard life table. The multistate life-table model is much more demanding in terms of data requirements, as longitudinal surveys or registers are needed to obtain transition rates (for more information on these two methods, see Chapter 11). Debate is still going on within REVES as to whether health expectancy indicators based on the prevalence of disability (i.e. the Sullivan method) provide an unbiased estimate of time trends in health expectancy. Barendregt *et al* (1994) have shown that the Sullivan method can produce misleading results when survival changes. This is because the Sullivan method integrates prevalence data on morbidity (stock data) in a life table on mortality (flow data). Only when the mortality and morbidity rates have been constant for a long period of time will an equilibrium situation emerge in which the Sullivan method provides an unbiased estimate. Mathers and Robine (1997a and 1997b) do not agree with this view, postulating that in realistic scenarios the use of the Sullivan method or the multistate life-table method will produce roughly the same results. The outcome of this debate is of particular importance if the purpose of studying trends is to assess whether compression or expansion of morbidity is occurring because the focus is on *changes* in total life expectancy, life expectancy with disability and life expectancy without disability (Barendregt *et al*, 1997). When using the Sullivan method for this purpose one runs a risk of confounding biases produced by the Sullivan method with real changes in health states. In addition, a deviation from the 'real' value has a larger impact on the number of years with disability than on disability-free life expectancy. It is exactly the number of years with disability that is decisive in assessing whether or not compression occurs. A small reduction in the number of years caused by the prevalence reaching its equilibrium values may be easily interpreted wrongly as compression of morbidity. This implies that, until this debate has been solved, one should be careful in interpreting the outcomes of the Sullivan method in terms of compression of morbidity.

DYNAMICS UNDERLYING CHANGES IN HEALTH EXPECTANCY

Fries and others (Fries, 1980, 1983, 1989; Michel and Robine, 1992; Michel *et al*, 1993; Verbrugge and Jette, 1994) have put great emphasis on the potential modifiability of the ageing and disablement process. Increasing evidence exists that although on average functioning declines as persons age, improvements in functioning do occur (Crimmins and Saito, 1993; Manton, 1988; Verbrugge *et al*, 1994). It has been shown that while age is a very strong determinant of disability, in addition to chronological age and (age-related) chronic diseases, health behaviours, socio-demographic factors and psychosocial factors affect the risk of disability (see, for example, Fried and Guralnik, 1997; Guralnik and Kaplan, 1989; Kaplan, 1992; Rogers *et al*, 1992; Strawbridge *et al*, 1993; Verbrugge and Balaban, 1989).

Assuming that (age-specific) disability can be reduced, which would be a favourable development as such, the question arises whether as a consequence of this decline a compression of morbidity is likely to occur. It is obvious that if disability were completely independent of mortality, compression of morbidity is likely to happen. However, this condition is not fulfilled, for at least two reasons. Firstly, because disability and mortality share common underlying diseases and non-disease risk factors (Breslow and Breslow, 1993; Kaplan, 1992) and secondly, because disability itself is a risk factor for mortality (McCallum and Shadbolt, 1994; Mulhorn, 1995; Rogers, 1993). As a result, persons with disability have higher mortality risks than those without disability; see, for example, Jagger and Clarke (1988), Mulhorn (1995) and Rogers (1993). This means that persons without or with less disability can expect to live longer. Rogers, for instance, showed that persons aged 55 without any limitations in daily activities have a life expectancy of 28.1 years as compared to 25.5 years for the average population of that age (Rogers, 1993). Given the presence of this association between disability and mortality, the effect of changing disability on health expectancy, and thus on compression of morbidity, is complex.

Within REVES, several studies have examined the consequences of changes in disability and mortality for health expectancy. The results of these studies have been, or can be, interpreted in terms of compression of morbidity. Three groups of studies can be distinguished. The first group examined the effects of overall changes in disability and mortality on health expectancy. The second group focused on the consequences of changes in specific diseases for health expectancy, through their impact on morbidity and/or mortality, whereas the third group assessed the effect of changes in modifiable risk factors. By examining the consequences of changes in overall mortality and disability patterns (first group), diseases (second group) and risk factors (third group), these studies provided insight into the conditions which are favourable or unfavourable for a compression of morbidity to occur. Such information can be used as a reference frame to evaluate the effect on compression of morbidity of autonomous or induced changes in mortality and morbidity, whether or not through changes in diseases or risk factors. This is of direct importance for setting priorities for public health policy and, in the end, might help to clarify the debate as to whether compression of morbidity occurs or is likely to occur in the near future.

IMPACT OF CHANGES IN OVERALL MORTALITY AND DISABILITY

The increasing availability of longitudinal studies has provided researchers with data on mortality and disability dynamics. Based on repeated measurements of disability and vital status, transition rates between disability states

Table 2.1. Life expectancy at age 70 for both sexes, by health status

	(1) Total	(2) No functioning problems	(3) Some functioning problems	(4) Unable to provide independent living	(5) Unable to provide personal care	(4)+(5) / (1) Proportion life expectancy dependent
A Baseline	12.16	3.91	5.36	0.70	2.18	0.24
B Change mortality						
Increase 36.8%	10.32	3.64	4.72	0.53	1.42	0.19
Decrease 36.8%	15.38	4.25	6.22	1.01	3.91	0.32
Increase 50% (only dependent)	11.16	3.87	5.22	0.58	1.48	0.18
Decrease 50% (only dependent)	14.61	3.98	5.59	0.96	4.09	0.35
C Change getting worse						
Increase 36.8%	11.32	3.06	5.02	0.70	2.54	0.29
Decrease 36.8%	13.32	5.33	5.62	0.67	1.70	0.18
D Change getting better						
Increase 36.8%	12.50	4.47	5.27	0.69	2.08	0.22
Decrease 36.8%	11.77	3.31	5.43	0.73	2.30	0.26
E Combined change						
36.8% change toward healthier life	17.22	6.72	6.47	1.02	3.01	0.23
50% change toward healthier life	20.27	8.44	6.99	1.26	3.58	0.24
36.8% change toward worsening health	9.28	2.45	4.49	0.57	1.77	0.25

Source: Crimmins *et al* (1994).

survival of disabled persons (e.g. by better treatment of the chronically ill) extends the lives of persons with disability. Thus, mortality reductions (irrespective in which state) produce an increase in the duration and the proportion of life spent with disability. This increase is larger when reductions are concentrated in the disabled state(s) (and when reductions are larger). This consequence of mortality reductions might be important, considering that particular interventions may affect not only disability rates, but also status-specific mortality rates. For instance, medical treatment might increase the chances of regaining function and increase survival at the same time. In addition, improvements in disability can take place in the presence of mortality declines. It was shown that changing both mortality and disability rates towards better health (for instance, lower incidence, lower progression, higher recurrence and lower state-specific mortality rates) by the same percentages at all ages increases not only total life expectancy and life expectancy without disability at age 70, but also life expectancy with disability (Crimmins *et al*, 1994; Nusselder, 1997; Nusselder *et al*, 1996a).

Further analysis by Nusselder (Nusselder, 1997) focusing on the effect of changing disability rates in the presence of moderate mortality reductions (by 20%) pointed at two findings that are important for compression of morbidity. First, simulations suggested that the effect of changing all transition rates by an equal percentage towards better health differs by age. Nusselder and co-workers found that reducing state-specific mortality rates and incidence rates of disability, and increasing recovery rates from disability by 20% at all ages, produces a reduced life expectancy with disability at age 30, by −0.6 year in men and −0.3 year in women, but causes an increase in life expectancy with disability at age 70 by 0.2 and 0.4 years, respectively. In-depth analysis of the age-specific consequences of this scenario indicated that the reduction of years with disability (due to lower incidence and higher recovery rates) occurs – on average – at younger ages than the accumulation of disability (mainly due to mortality reductions) (Nusselder, 1997). Second, it was shown that with moderate declines (by 20%) in mortality rates at all ages among both disabled and non-disabled persons, at least equal proportional improvements in both incidence and recovery rates would be needed to achieve absolute compression of morbidity at age 30. For an improvement to occur in either incidence or recovery rates, improvements of at least 30% and 50%, respectively, would be required. It was also shown that greater improvements at all ages would be needed to achieve absolute compression of morbidity at age 70.

IMPACT OF SPECIFIC DISEASES

Diseases as causes of death have been studied for a long time and cause-of-death data are collected annually in most countries. Information on chronic diseases as causes of disability is increasingly available from health surveys (see

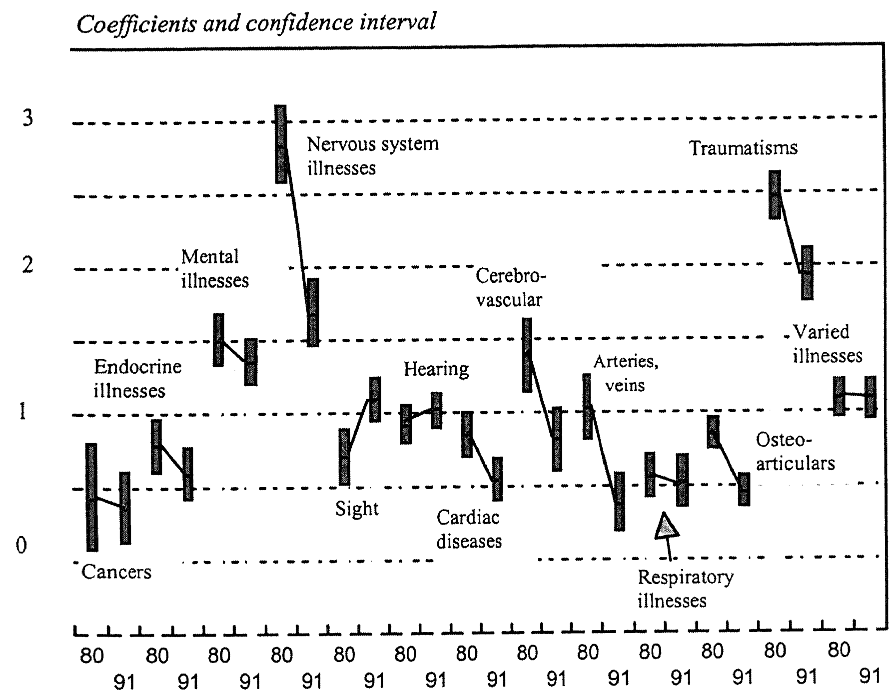


Figure 2.2. Trends in the influence of different disabling diseases on disability, at identical age, from 1980 to 1991 (men). From Robine *et al* (1998). Reprinted by permission of Sage Publications, Inc.

Boult *et al*, 1994; Ettinger *et al*, 1994; Furner *et al*, 1995; Guccione *et al*, 1994). It is obvious that since diseases have an impact both on mortality and on disability, the effect of changes in diseases on health expectancy, and thus on compression of morbidity, is not clear *a priori*. Regarding this effect two factors are important: (1) the relation between these diseases and disability, and (2) the extent to which mortality from these diseases ends disabled and non-disabled life.

Disability

The relation between diseases and disability, and thus between changes in disease prevalence and in the prevalence of disability, is less simple than one might expect. In studying the causes and mechanisms responsible for the *reduction* in disability rates in France between 1981 and 1991 (which caused together with mortality reductions an increase in disability-free life expectancy), Robine and colleagues (Robine *et al*, 1998) found that the prevalence of the main disabling diseases *increased* generally after age 60 or 70 (except for

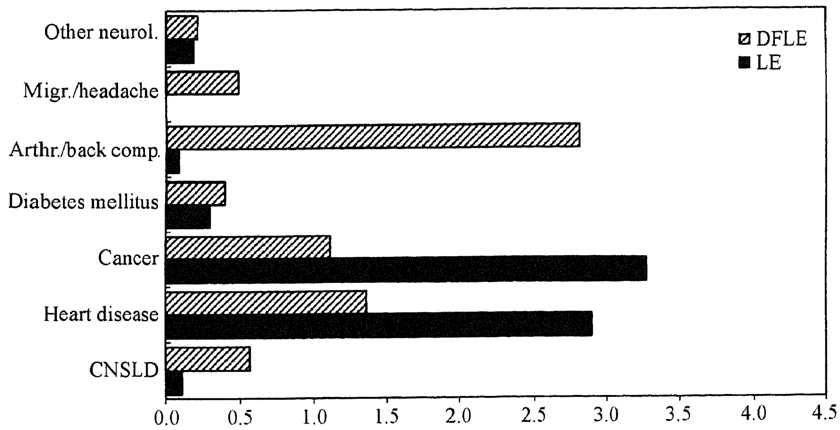


Figure 2.3. Increase in total life expectancy (LE) and disability-free life expectancy (DFLE) with hypothetical elimination of a disease (men, age 15). CNSLD, chronic non-specific lung disease. From Nusselder *et al* (1994). Reproduced with permission

inactive years. The effects of eliminating mortality from cancer and cerebral diseases on the number of active and inactive years would be smaller. Interpretation of the outcomes in terms of compression of morbidity (not included in the original contribution) gives a less optimistic message, as it indicates that reducing mortality from heart disease, cancer and cerebral diseases expands not only the number of active years, but also that of inactive years. That is, elimination of these diseases as a cause of death would cause an expansion of morbidity (in an absolute and in a relative sense).

Mortality and Disability

A study of Nusselder and colleagues (Nusselder *et al*, 1994, 1996b) examined whether a hypothetical elimination of specific chronic diseases, and the consequences in terms of disability and mortality, would lead to a compression of morbidity. They found that eliminating disabling diseases such as arthritis/back complaints that cause little mortality would lead to an increase in disability-free life expectancy, while total life expectancy would remain more or less constant (Figure 2.3). Consequently, life expectancy with disability would decline and thus a compression of morbidity (both in an absolute and a relative sense) would occur. For cancer, a disease with a high case-fatality, elimination would result not only in a gain in disability-free life expectancy, but also in an – even larger – increase in life expectancy with disability. In the years that people are saved from dying from cancer, they experience disability due to other causes. So people live longer, but most of these years are spent with in disability-free life expectancy (2.5 years in men and 1.9 years in women) than in total life expectancy (1.6 and 0.8 years, respectively). Consequently, smoking elimination would compress life expectancy with disability in an absolute and a relative sense. Thus, despite the fact that life would be extended as a consequence of eliminating smoking, and people would be exposed longer to disability, lower incidence rates of disability and higher recovery rates from disability would produce on balance a reduction in years with disability. Based on a multistate model with different disease states in combination with disability weights (i.e. the DALE approach), Barendregt *et al* (1998) found that smoking elimination would reduce the difference between total life expectancy and disability-adjusted life expectancy (DALE), which means that smoking elimination would produce a compression of morbidity (in an absolute and a relative sense). Recent studies for other countries seem inconclusive as to whether smoking elimination would result in a compression of morbidity (Ferrucci *et al*, 1999; Martel *et al*, 2000; Bronnum-Hansen and Juel, 2001).

CONCLUSION

Ten years of REVES have contributed to the methodological progress necessary for research on compression of morbidity and to the development of a better understanding of the complex interaction between morbidity, mortality and population health in general and of compression of morbidity in particular. Health expectancy proved to be a powerful measure and the life table an important tool in studying compression of morbidity. Besides advocating the use of health expectancy, REVES has been an important platform for discussions on the definition and measurement of compression of morbidity, on different methods to calculate health expectancy, and on the dynamics underlying a compression of morbidity. Nevertheless, much research remains to be done. Research on the ageing and disablement process has stressed the importance of several potential risk factors and effect modifiers in these processes. More research is needed to understand the opposite results from prior studies on the effect of (elimination of) smoking in terms of compression of morbidity and to assess the contribution of risk factors other than smoking. Disease-specific models are needed to assess the effect of changes in different phases of the disease process. The increasing availability of longitudinal studies, which include information on risk factors, chronic diseases, disability and mortality, will offer new challenges for further research on this issue. REVES can keep on playing an important role in exchanging data, discussing methodological problems and interpreting outcomes of these studies, as it did in the last 10 years.

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