Rectangularization of the Survival Curve in The Netherlands: An Analysis of Underlying Causes of Death

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This study analyzed the contribution of selected causes of death to rectangularization of the survival curve of Dutch men and women above age 60 in the 1980s, and determined why rectangularization took place in the 1980s but not in the 1970s. The contribution of causes of death was determined by means of a decomposition analysis, using mortality data by underlying cause of death, sex, and age from Statistics Netherlands. Our results show that mortality reductions from ischemic heart disease, cerebrovascular diseases, and lung cancer (men only) and mortality increases from chronic obstructive pulmonary diseases (men only) and mental disorders (women) contributed to rectangularization in the 1980s. Comparison with the 1970s, in addition, demonstrated that in particular changes in mortality at advanced ages (i.e., smaller mortality reductions and mortality increases) were responsible for the reversal from a decreasingly rectangular shape of the survival curve in the 1970s to rectangularization in the 1980s. The combination of increased survival to advanced ages and reduced survival at advanced ages explains why rectangularization of the survival curve took place recently in The Netherlands.

DECLINING death rates from chronic diseases among the elderly population in low-mortality countries have contributed to the ongoing debate on further increases in life expectancy (Fries, 1980; Manton, Stallard, and Tolley, 1991) and its consequences for the health expectancy of the population (Fries, 1980; Kramer, 1980; Manton, 1982). Rectangularization of the survival curve plays an important role in these discussions. An increasingly rectangular shape of the survival curve, reflecting an increased survival until advanced age and concentration (compression) of deaths into a small age range, implies that further increases in life expectancy are harder to achieve. This makes a favorable development of health expectancy, known as "compression of morbidity," more likely (Fries, 1980). The question of whether there is rectangularization of the survival curve was addressed in several studies (Fries, 1984; Go et al., 1995; Myers and Manton, 1984a, 1984b; Nagnur, 1986; Rothenberg, Lentzner, and Parker, 1991). Most studies concluded that there was no rectangularization at older ages, although there exists some controversy about the assessment of rectangularization. Examination of Dutch survival curves showed that an increase in life expectancy above age 60 was accompanied by compression of mortality into a smaller age interval in the 1980s, whereas in the 1960s and 1970s (men also in the 1950s), decompression of mortality occurred. That is, a reversal took place from a decreasingly rectangular shape of the survival curve prior to the 1980s to rectangularization in the 1980s (Nusselder and Mackenbach, 1996).

The objective of this study was to develop a better understanding of the recent rectangularization of the survival curve at older age in The Netherlands by examining the contribution of selected causes of death. Changes in the cause-of-death pattern can give a hint of changing determinants of mortality and may therefore afford a glance behind the scenes of the processes which caused rectangularization. The central question is which changes in the cause-of-death pattern underlie rectangularization of the survival curve above age 60 in the 1980s. To answer this question we conducted a bipartite analysis. First, we examined which causes of death contributed to rectangularization of the survival curve in the 1980s. Next, we analyzed which causes of death contributed to the reversal from a decreasingly rectangular shape of the survival curve in the 1970s to rectangularization in the 1980s.

METHODS

Data on the population and the number of deaths by age and sex were obtained from Statistics Netherlands. Population and total mortality data covering the period 1950–1992 were classified by single year-of-age. Analyses using cause-specific mortality data cover the period 1970–1992. In this period deaths were classified by underlying cause of death according to two different revisions of the International Classification of Diseases, Injuries and Causes of Death: the eighth revision (ICD-8) for the period 1970–1978 and the ninth revision (ICD-9) for the period 1979–1992. From all causes distinguished in these ICD-classifications, we composed 26 cause-of-death groups for which the comparability over the ICD-classifications is maximized (Table 1). Cause-specific mortality data were only available in 5-year age groups with age 85 and over as the oldest age group. Age groups below age 60 were not included in the analyses, since especially changes in the rectangularity of the survival curve at older ages are relevant to the further development of life and health expectancy.

Construction of life tables. — Abbreviated total mortality life tables consisting of 5-year age groups starting at age 60...
Table 1. Classification in Cause of Death Groups According to the ICD-classifications

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Infectious and parasitic diseases</td>
<td>1</td>
<td>001-136</td>
<td>001-139</td>
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<tr>
<td>Cancer of stomach</td>
<td>2</td>
<td>151</td>
<td>151</td>
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<tr>
<td>Cancer of colorectum</td>
<td>2</td>
<td>153-154</td>
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<tr>
<td>Cancer of trachea, bronchus and lung</td>
<td>2</td>
<td>162-163</td>
<td>162-163; 165</td>
<td></td>
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<tr>
<td>Cancer of breast</td>
<td>2</td>
<td>174</td>
<td>174-175</td>
<td></td>
</tr>
<tr>
<td>Cancer of prostate</td>
<td>2</td>
<td>185</td>
<td>185</td>
<td></td>
</tr>
<tr>
<td>Other neoplasms</td>
<td>2</td>
<td>r(140-239)</td>
<td>r(140-239)</td>
<td></td>
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<tr>
<td>Diabetes mellitus</td>
<td>3</td>
<td>250</td>
<td>250</td>
<td></td>
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<tr>
<td>Other endocrine, nutritional and metabolic diseases</td>
<td>3</td>
<td>240-246; 251-269;</td>
<td>240-246; 251-259;</td>
<td></td>
</tr>
<tr>
<td>Diseases of blood and bloodforming organs</td>
<td>4</td>
<td>280-289</td>
<td>280-289</td>
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<td>Mental disorders</td>
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<td>Diseases of the nervous system and sense organs</td>
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<td>Ischemic heart diseases</td>
<td>7</td>
<td>410-414</td>
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<tr>
<td>Cerebrovascular diseases</td>
<td>7</td>
<td>430-438</td>
<td>430-438</td>
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<tr>
<td>Other cardiovascular diseases</td>
<td>7</td>
<td>440-448; 450-458</td>
<td>440-448; 415;</td>
<td></td>
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<tr>
<td>Other heart diseases</td>
<td>7</td>
<td>390-398; 400-404;</td>
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<tr>
<td>Pneumonia/influenza</td>
<td>8</td>
<td>470-474; 480-484;</td>
<td>487; 480-486</td>
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<tr>
<td>Chronic obstructive pulmonary diseases</td>
<td>8</td>
<td>490-493</td>
<td>490-494; 496</td>
<td></td>
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<tr>
<td>Other diseases of the respiratory system</td>
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<td>r(460-519)</td>
<td>r(460-519)</td>
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<td>Diseases of the digestive system</td>
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<tr>
<td>Diseases of the genito-urinary system</td>
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<td>580-629</td>
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<td>Symptoms and ill-defined conditions</td>
<td>16</td>
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<td>780-799</td>
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<td>17</td>
<td>E800-845; E940-941</td>
<td>E800-E848</td>
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<tr>
<td>Other accidents</td>
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<td>E880-887; E890-909;</td>
<td>E880-E888; E890-909;</td>
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<td>Other external causes</td>
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<td>E911-929; E943-946;</td>
<td>E911-929;</td>
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<td>Other causes</td>
<td>11-15</td>
<td>630-678; 680-686;</td>
<td>630-676; 680-686;</td>
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</table>

and with age 100 and over as oldest age group were constructed for both sexes using standard life-table techniques (Manton and Stallard, 1984; Namboodiri and Suchindran, 1987). These are period life tables covering 5 calendar years (e.g., 1980/84), except for the last period, 1990/92. Period life tables combine mortality data of different birth cohorts. These cohorts may have experienced different influences on mortality (cohort effects) and different mortality selection. Nevertheless, we did not use cohort life tables, because in that case assumptions on the development of mortality of cohorts born before 1895 who have not yet completed their mortality in 1990/92 would have to be made. Cause-of-death ratios by 5-year age groups and sex were calculated by dividing the number of deaths per cause by the total number of deaths (Namboodiri, 1991). These cause-of-death ratios were assumed to be constant within 5-year age intervals and above age 85. The life tables and cause-of-death ratios were the starting point of our analyses.

Measurement of rectangularization. — Rectangularization of the survival curve is assessed by determining whether there is a reduction in the variability in the age at death. In the extreme situation of a perfectly rectangular survival curve, everyone survives to advanced ages and then dies at the same age (i.e., there is no variability in the age at death). Empirical survival curves are not (and are not likely to be) perfectly rectangular as the age at death differs between individuals, but changes in the age pattern of mortality can result in squaring of the survival curve, known as “rectangularization.” Rectangularization is defined as a trend toward a more rectangular shape of the survival curve due to an increased survival and increased concentration (compression) of deaths around the mean age at death. This study focuses on rectangularization and compression of mortality in an absolute sense, designating a situation of increased concentration of deaths into a smaller age interval (i.e., a decline of the variability in the age at death, expressed in years). Rectangularization in a relative sense, which occurs when deaths are concentrated into a smaller proportion of total life expectancy (i.e., a decline of the variability in the age at death, relative to the life expectancy), is not considered for two reasons. First, if life expectancy is increasing,
then absolute compression automatically means relative compression. Second, previous analyses have shown that in particular the trend in absolute variability showed a remarkable change during the 1980s in The Netherlands (Nusselder and Mackenbach, 1996).

To assess rectangularization, we used the numerator of Keyfitz’s H (NH) (Keyfitz, 1977; Smith, 1992), which, like the standard deviation, is a measure of absolute variability. Advantages of NH over the better known standard deviation are that (the change in) the NH can be decomposed by cause of death and that NH not only quantifies the variability in the age at death, but also the effect of small proportional mortality reductions on the life expectancy, which makes the outcomes directly quantitatively interpretable. A change in absolute variability, measured with the NH, indicates that age at death, but also the effect of small proportional mortality reductions on the life expectancy, which makes the NH an attractive measure.

Decomposition analysis. — In order to assess the contribution of different causes of death to rectangularization of the survival curve, the change in NH is decomposed by cause. First, we describe the principles of the decomposition analysis, leaving out of consideration different causes of death. Next, we explain how causes of death are incorporated in the decomposition technique.

In one age group, NH is the product of the number of life-table deaths and the remaining life expectancy (formula 1). This means that a change in NH occurs if the number of deaths changes, if the remaining life expectancy changes, or if both change, or,

\[ \Delta sNH_a = [\Delta a d_a; d_a + \Delta a d_a (e_a + 0.5n) + \Delta a d_a e_a + 0.5n] - [\Delta a d_a e_a + 0.5n] \]  

(2)

where \( \Delta s \) is change between the two periods under consideration. Reexpression gives,

\[ \Delta sNH_a = [\Delta a d_a e_a + 0.5n] + [\Delta a d_a e_a + 0.5n] + [\Delta a d_a e_a + 0.5n]. \]  

(3)

A change in NH due to a change in the number of deaths in that age group (first component of formula 3), is termed the “direct effect” (DE). A change in NH due to a change in remaining life expectancy (second component of formula 3) is termed the “indirect effect” (IE). To correct for double counting or undercounting, which occurs when both the number of deaths and the life expectancy change, a correction is added to the IE (third component of formula 3). A negative DE reflects a decline in the number of deaths in the considered age group due to a shift in mortality to other age groups. A negative IE reflects an increase in mortality at later ages (i.e., older than the age interval considered), which results in a decline in remaining life expectancy. A complicating factor is that DE and IE operate in opposite directions. For example, a decline in mortality in a given age interval results in both a direct effect (due to fewer deaths in that age interval) and an indirect effect (the decline in mortality in the given age group leads to an increase in life expectancy at younger ages). Whether the net effect (aggregated over all age groups above age 60) is one of decline depends on the size of both effects. In general, changes in mortality rates at younger ages affect the variability more than those at older ages, since the life expectancy and population alive (and thus at risk for mortality changes) decrease with age.

Next, we included different causes of death in the decomposition analyses. For each NH (i.e., for each age group) we determined which causes of death were responsible for the DE (through a change in the number of deaths) and which causes of death were responsible for the IE (through a change in remaining life expectancy, or in both). Given,

\[ \Delta s d_a = \sum_{i=1}^{m} \Delta s d_{ai} \]  

(4)

where \( m \) is the number of causes of death comprising total mortality, the DE is calculated as follows:

\[ sDE_a = \sum_{i=1}^{m} \Delta s d_{ai} e_a + 0.5n. \]  

(5)

Thus, the age- and cause-specific DE (\( sDE_{ai} \)) is:

\[ sDE_{ai} = \Delta s d_{ai} e_a + 0.5n. \]  

(6)

Similarly, given,

\[ \Delta s e_a + 0.5n = \sum_{i=1}^{m} \Delta s e_{ai} + 0.5n. \]  

(7)

the IE is calculated as follows:

\[ sIE_a = \sum_{i=1}^{m} \Delta s d_{ai} \Delta s e_{ai} + 0.5n. \]  

(8)

Reexpression of the IE gives:

\[ sIE_a = \sum_{i=1}^{m} d_a \Delta s e_{ai} + 0.5n. \]  

(9)

Thus, the age- and cause-specific IE (\( sIE_{ai} \)) is:

\[ sIE_{ai} = \Delta s d_{ai} e_a + 0.5n \]  

(10)

The change in the number of deaths by cause and age (\( \Delta d_{ai} \)) is calculated on the basis of the number of life-table
deaths by cause and age for each period. These are obtained by multiplying the number of life-table deaths by age with age-specific cause-of-death ratios. The change in life expectancy by cause ($\Delta E_{a+0.5e}$) is estimated with a standard decomposition technique developed by Arriaga (Arriaga, 1984, 1989). This technique assumes that the contribution of a cause of death to the change in life expectancy that can be attributed to an age group is proportional to the contribution of this cause to the change in the total central mortality rate in that age group.

Adding age- and cause-specific DEs and IEs (i.e., $DE_{a,i}$ and $IE_{a,i}$) over all ages above age 60 gives the cause-specific DEs and IEs. These can be arranged in a table with causes of death presented in the columns, DEs and IEs in the rows, and cause- and effect-specific contributions in the cells. From this table the contributions are arranged according to two perspectives: by cause and by type of effect (i.e., direct vs indirect). In order to obtain from this table the contribution by cause, we add the DE and IE of each cause (i.e., aggregation over the rows). A “negative” effect indicates that the cause contributes to a decline in $NH_{60}$. A “negative” contribution occurs if mortality from that cause is redistributed to age groups closer to the mean age at death (which contribute less to the variability in the age at death). Such a redistribution takes place if a mortality reduction (in any case at younger ages) occurs, or if a mortality increase at older ages reduces the remaining life expectancy. For a “positive” contribution the opposite is true.

Alternatively, addition of the cause-specific DEs and IEs over all causes (i.e., aggregation over columns) gives insight into the ways through which mortality changes contribute to the change in $NH_{60}$. The cause- and effect-specific contributions (i.e., cells in the table referred to above) give this information for each cause separately. The DE quantifies the change in $NH_{60}$ that is caused directly by a changing number of deaths (of a cause). The IE is caused indirectly by a changing number of deaths (of a cause), and operates via the remaining life expectancy.

In the verbal description of our results (using standard techniques), we make use of mortality measures calculated on the basis of mortality and population data above age 60. These are age- and cause-specific mortality rates (per 5-year age groups up to and including age 85-plus), age-adjusted cause-specific death rates (using direct standardization with the total Dutch population of 1950/92 as standard), and mean ages at death for each cause (calculated from multi-decrement life tables [Manton, 1988]). The results of these calculations are not shown separately in tables or figures, except for the change in age-specific mortality rates between 1970/74–1980/84 and 1980/84–1990/92, which is presented for selected causes.

RESULTS

Figure 1 shows the trend in $NH_{60}$ and life expectancy at age 60 since the 1950s. Changes in $NH_{60}$ per decade are summarized in Table 2. The increase in $NH_{60}$ in the 1960s and 1970s (and among men in the 1950s, as well) shows that the variability in the age at death increased (i.e., decompression of mortality). The slight declines in $NH_{60}$ with $-0.23$ years among men and $-0.09$ among women during the 1980s indicate a reversal of this trend (i.e., compression of mortality). These trends in $NH_{60}$ and life expectancy indicate that rectangularization of the survival curve occurred in the 1980s, but not in the 1970s.

Causes of death contributing to the change in $NH_{60}$. Table 3 presents the change in $NH_{60}$ during the periods 1970/74 to 1980/84 and 1980/84 to 1990/92, as well as a selection of causes of death that contributed most (both positively and negatively) to this change. (A complete set, including the change in $NH_{60}$ for all causes of death, is available from the authors on request.) The decline in $NH_{60}$ in the 1980s was the net effect of “negative” contributions (i.e., for men and women) and “positive” contributions of causes (e.g., ischemic heart disease (IHD), lung cancer (men), and cerebrovascular diseases were the most important negative contributors. Such a negative contribution is the result of either a decrease in mortality from that cause, in any case at younger ages, or an increase in mortality at older ages. For the above-mentioned negative contributors, age-adjusted death rates declined during the 1980s (not shown).

A comparison of these results with those of the 1970s when $NH_{60}$ increased, shows that negative contributions were larger (i.e. for men and women) and positive contributions were smaller (respectively, 0.19 and 0.26) in the 1980s. In addition, the largest positive and negative contributors to the change in $NH_{60}$ were not the same ones in both decades. Some causes, like IHD (men), chronic obstructive pulmonary diseases
COPD), other cardiovascular diseases, and lung cancer (women) differed only in the ranking of their contribution to the change in NH60. Others, like IHD (women), lung cancer (men), cerebrovascular diseases, and mental disorders contributed positively to the change in NH60 in the 1980s and negatively in the 1970s or vice versa. This change in sign was not restricted to causes showing a reversal of the trend in age-adjusted death rates (e.g., mental disorders and lung cancer among men), but also occurred for causes with declining age-adjusted death rates in both decades (e.g., IHD in women and cerebrovascular diseases).

For causes contributing largely to the change in NH60 in the 1970s and 1980s, Table 3 shows the size of the DE and IE. (A complete set, including the DE and IE for all causes of death, is available from the authors on request.) Figure 2 presents the absolute change in age-specific mortality rates for selected causes in the 1970s and 1980s. For the largest negative contributors to NH60 in the 1980s (i.e., IHD, cerebrovascular diseases, and lung cancer among men), the negative DE was of overriding importance (Table 3). That is, mortality reductions directly affected NH60. Except for lung cancer, declining mortality rates from these causes also resulted in a negative DE in the 1970s. For lung cancer (men), increasing mortality rates in all age groups above age 65 contributed directly to an increase in the variability in the age at death in the 1970s, whereas increasing mortality rates were only observed above age 80 in the 1980s. This age pattern, showing mortality reductions at younger ages and increases at older ages, is responsible for the contribution of lung cancer to compression of mortality in the 1980s.

Causes contributing mainly via the IE to compression of mortality in the 1980s are COPD (men) and mental disorders (women) (Table 3). These causes have in common a high mean age at death (i.e., above the average age at death at age 60). The negative IE indicates that increasing mortality rates (mainly at advanced ages) from these causes had a negative impact on the remaining life expectancy and thus on the variability in the age at death. In the 1970s, the IE was also negative for COPD in men, reflecting that mortality rates among the oldest old also increased in this decade (Figure 2).
Figure 2. Absolute change in age-specific mortality rates of selected causes of death (per 100,000) in 1970/74–1980/84 and 1980/84–1990/92, The Netherlands, by sex.
For mental disorders, declining mortality rates among the oldest old contributed indirectly to an increase in the variability in the age at death in the 1970s. The case of diabetes mellitus illustrates that an increase in mortality rates at older ages does not necessarily imply a negative contribution. Increasing mortality rates from this cause at all age groups above age 60 resulted not only in a negative IE, but also in a (even larger) positive DE in the 1980s.

Causes of death contributing to the reversal in the trend in NH$_{60}$. — Figure 3 shows that the reversal of the trend in NH$_{60}$ between the 1970s (summarized as 1972–1982) and 1980s (summarized as 1982–1991) was caused mainly by the IE. The signs of the DEs and IEs did not change. This is true especially for women, for whom the negative DE was even larger in the 1970s than in the 1980s. The decline in IE in the 1980s indicates that the increase (after all IE is positive) in remaining life expectancy contributed less to the change in NH$_{60}$. This is in accordance with the fact that life expectancy at older ages increased less in the 1980s than in the 1970s and even slightly declined among the oldest old (above age 85 for men and above age 90 for women) during the 1980s (data not shown). Such a declining life expectancy at the older ages means that people surviving to those ages are expected to die in a shorter interval.

Causes with the largest decline in IE between the 1970s and 1980s are other cardiovascular diseases, cerebrovascular diseases, ill-defined conditions, mental disorders, and diabetes mellitus (Table 4). These causes showed considerable changes in mortality among the oldest old (Figure 2). That is, either a shift took place from large mortality reductions in the 1970s to smaller reductions or even mortality increases in the 1980s, or mortality rates at older ages were increasing more rapidly in the 1980s. This underscores the importance of mortality changes at advanced ages in the explanation of compression of mortality. This is the more so, since large mortality reductions, contributing via a negative direct effect to the change in the variability in the age at death, were observed in the 1970s as well. A similar phenomenon was observed for IHD among women. Although mortality reductions from this cause contributed directly to a decline in HN$_{60}$ both in the 1970s and 1980s (the effect being even larger in the 1970s), an increase in the variability which was caused indirectly by large mortality reductions at advanced ages nullified this effect completely in the 1970s. Smaller mortality reductions from IHD at advanced ages did no longer undo the negative DE in the 1980s.

DISCUSSION

The main objective of this study was to develop a better understanding of rectangularization of the survival curve above age 60 in The Netherlands, by analyzing the contribution of selected causes of death. To measure rectangularization, we used the numerator of Keyfitz’ H (NH), which is, like the standard deviation, a measure of absolute variability. A decline in NH at age 60 (NH$_{60}$) indicates that mortality is compressed into a smaller age interval. This means that rectangularization of the survival curve takes place, given that life expectancy increases. To assess the contribution of causes of death to rectangularization of the survival curve, we could not make use of a standard technique. The decomposition technique developed in this study enabled us to determine which causes of death contributed to compression of mortality in the 1980s and which changes contributed to the shift from decompression in the 1970s to compression in the 1980s. It takes into account that compression of mortality (i.e., concentration of deaths around the mean) can be caused both directly by a mortality reduction (also at youn-
ger ages) and indirectly by a mortality increase (mainly at advanced ages).

Although our decomposition technique proved to be a useful tool to determine which changes in the cause-of-death pattern underlie compression of mortality above age 60 in The Netherlands, caution must be exercised when interpreting the results. Most uncertainties relate to the cause-specific mortality data.

First, deaths by cause were only available by 5-year age groups, with age 85 and over as final age group. Using these age intervals and assuming a constant distribution of cause-of-death ratios within age intervals might have affected our results in two ways. First, especially above age 85, aging might have changed cause-specific death rates, even if age-specific rates remained constant. For causes with increasing age-specific mortality rates with age within the final age group, but for example increase, deaths are concentrated more at advanced ages. Consequently, the effect of increased mortality at advanced ages might be underestimated. Similar mechanisms operate for declining ratios with age and for a mortality decline. In general, the two biases operate in the opposite direction and thus (partly) nullify each other.

Second, discontinuity in time series of cause-specific mortality, either due to the ICD-revision in 1979 or due to changes in coding practices within the ICD-9 classification, are a threat to the validity of our results. Although we composed cause-of-death categories for which the comparability over ICD-classifications is maximized, continuity of time trends was not achieved in all cases since deaths by cause are only published at the three-digit level in The Netherlands. Therefore, it was impossible to categorize "cardiovascular disease, unspecified" (429.2) as IHD and the rest of 429 as other heart diseases in the ninth revision, and to transfer "acute heart failure, unspecified" from the ill-defined category (782.4) to other heart diseases in the eighth revision. As a result, the mortality decline from IHD between 1970/74 and 1980/84 might be overestimated.

Changes in coding practices within the ICD-9 classification are known to have occurred for diabetes mellitus and senile dementia. Since 1983, diabetes mellitus has been classified more often as an underlying cause of death (and less as a secondary cause) of cardiovascular diseases (Mackenbach, Snels, and Friden-Kill, 1991). Senile dementia has been classified more frequently as an underlying cause of death since mid-1992 (Statistics Netherlands, 1995). The increase in mortality from these causes and the decline in mortality from IHD might thus be overestimated for the period 1980/84–1990/92. If discontinuity in the time-series of IHD mortality affected mainly older age groups, the contribution of IHD to decompression of mortality in the 1970s is likely to be overestimated, and its contribution to compression in the 1980s is likely to be underestimated. If overestimation occurred at younger ages, the contribution of IHD to compression of mortality in the 1980s might be overestimated. Yet, if overestimation took place at all ages, positive and negative biases might (partly) be nullified by each other. Similarly, the contribution of mental disorders to compression of mortality in the 1980s might be slightly smaller than estimated, if changes in coding mainly affected the oldest old. However, since the increase in mortality was also observed before the change in coding practices in 1992, the contribution of mental disorders to compression of mortality is not likely to be an artifact.

Third, we used underlying-cause-of-death data, because multiple-causes-of-death data are not generally available in The Netherlands. Underlying causes disregard the fact that often not one, but multiple conditions, contributed to death at advanced age. In addition, using only the underlying cause makes the outcomes more sensitive to changes in coding practices. In general, causes of death are difficult to assess in the oldest old, and thus may be unreliable.

These limitations related to mortality data by cause of death should not be undervalued; they underscore the need for validation of our outcomes in other studies, preferably based on mortality data by single year-of-age and multiple causes of death. Nevertheless, the overall results suggest that compression of mortality in the 1980s was caused both by increased survival to advanced ages due to mortality reductions from IHD, cerebrovascular diseases, and (for men) lung cancer, and increases in mortality at older ages from a large number of causes, the most important ones being COPD (emphysema) for men and mental disorders (senile dementia) for women. In addition, our results show that mortality changes at advanced ages (i.e., smaller mortality reductions from cerebrovascular and cardiovascular diseases and larger mortality increases from ill-defined conditions, mental disorders, and diabetes mellitus) were especially responsible for the reversal from a decreasing rectangular shape of the survival in the 1970s curve to rectangularization in the 1980s. That is, increased survival to advanced ages was not a sufficient condition for rectangularization. The finding that both increased survival to advanced ages and the reduction of the survival probabilities at advanced ages (i.e., people surviving to advanced ages are expected to die in a shorter interval) were necessary to explain why compression took place in the 1980s and not in the 1970s (Figure 2), is not influenced by uncertainties related to cause-specific data. These results are derived from analyses aggregated over all causes, and are thus based on total mortality data, which are considered to be very reliable in The Netherlands (Condron, Himes, and Preston, 1991).

The reductions in NHgo appear to be small, but we consider them to be relevant (especially in men), taking into account that the size of the changes in NHgo in the previous decades was more or less the same (except for females in the 1970s) and that the survival curve will never become perfectly rectangular (i.e., NHgo will never equal zero). Even in the extreme case where the genetically endowed limit would be approached, there will be genetic variability in the age at death. On the basis of certain assumptions, Fries (1987) estimated that in the case where the genetically endowed limit would be approached, the standard deviation in the age at death would be about 7 years (in his earlier publications this was 4 years). It is obvious that such an estimate has a
highly speculative character, since the amount of genetic variability cannot be determined unambiguously from the tail of the survival curve (Manton and Tolley, 1991). However, in the context of interpreting the magnitude of the changes in NH0, Fries’ estimate is helpful and suggests that the variability in the age at death will be at least some years. Considering that NH0 was 8.1 in men and 7.8 years in women in 1980/84 and that the minimum level of NH0 would still be some years, we consider the decline of NH0 over a 10-year period with .23 years in men to be of importance.

Further research is needed to explain the stagnation of the mortality decline among the oldest old in The Netherlands, which played a major role in rectangularization of the survival curve. Here, we will briefly discuss three (not mutually exclusive) explanations for the mortality increase among the oldest old, which was observed for total mortality and causes such as: COPD (emphysema), mental disorders (senile dementia), cancer (prostate and lung for men, and other cancers), diabetes mellitus, ill-defined conditions (senility), and diseases of the nervous system (Parkinson’s disease).

First, cohort effects partly related to smoking behavior might have contributed to increased mortality in particular from (lung) cancer and COPD among men above age 85. A recent study (Janssen-Heijnen et al., 1995) showed a cohort effect for lung cancer. A reconstruction of smoking prevalence by male birth cohort has shown that smoking prevalence increased from birth cohorts 1897 to 1917 and then declined, with the exception of the high smoking prevalence of the cohort born before 1887 (which might be an artifact) (Gunning-Schepers, 1988). Thus, the percentage of (ex)smokers in persons aged 85 and over may have been higher in 1990/92 than the percentage in the same age group 10 years earlier. Particularly for lung cancer and COPD, the excess risk of death remains elevated after smoking cessation, even after a long period of time (U.S. Department of Health and Human Services, 1990). Women show a different smoking pattern — a small smoking prevalence in the older birth cohorts which increased in the more recent birth cohorts (Gunning-Schepers, 1988). The percentage of (ex)smokers among women who were 85 and over in 1990/92 (or 10 years earlier) is thus small and is unlikely to have affected mortality from COPD and lung cancer in this age group.

Second, the influenza epidemic in December 1989—January 1990 might have contributed to a (temporary) excess mortality of frail elderly in the period 1990/92. However, it is unlikely that the recent rectangularization is caused solely by this influenza epidemic, since half of the excess mortality in January 1990 was compensated by lower mortality in the following months, and influenza epidemics with considerable excess mortality also occurred in earlier periods (e.g., in 1976, 1978) when no rectangularization took place (Prins, 1990, 1991). In addition, repeating our analyses using 1991 and 1992 mortality data only (and thus excluding 1990) did not change our conclusions.

Third, less selection due to increased survival from circulatory diseases might have created a more vulnerable (frail) population with a higher risk to die from several causes striking at old age. This phenomenon of competing risks (i.e., those not dying from circulatory diseases were at a higher risk to die from other causes) can result in a general increase in mortality at advanced ages or in an increase in mortality from specific causes which are themselves a risk factor for circulatory diseases (like diabetes mellitus) or share common risk factors. In addition, it is not unthinkable that the smaller mortality reductions from cerebrovascular diseases, other cardiovascular diseases, and IHD (especially in women) (which contributed to the shift from decompression in the 1970s to compression in the 1980s) may be caused by increased survival of persons with circulatory diseases. Milder disease stages may then act as a risk factor for more severe stages. This is true for heart diseases: mortality was postponed more than incidence, creating a pool of persons with chronic heart diseases (Bonneux et al., 1994). These persons are at a higher risk to die.

If the phenomenon we have observed is based on cohort dynamics related to smoking behavior, rectangularization of the survival curve in men is expected to continue for the next one or two decades, while smoking-related premature mortality is expected to reduce rectangularization in the next decades in women (if smoking has a similar effect as in men). However, apart from these “temporal” changes in the shape of the survival curves caused by changes in health behavior, increased frailty of the population causing more permanent rectangularization cannot be excluded. Taking for granted that increased frailty of the population indeed plays a role, our results have consequences for the further increase in life expectancy. The occurrence of rectangularization of the survival curve might then indicate that, unless breakthroughs in the major process of aging occur, further increases in life expectancy are increasingly hard to achieve because the “hard” core of mortality related to senescence increasingly acts as a barrier to further increases in survival at old age.

The implications of these results for achieving compression of morbidity are not unambiguous. On the one hand, delaying the age at onset of chronic diseases and long-term disabilities is likely to result in compression of morbidity, since the life expectancy is not expected to increase rapidly. On the other hand, increased frailty of the elderly might not only imply that mortality is unavoidable, but also that accumulation of severe morbidity at advanced ages cannot be prevented.

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**Differential Mortality. Methodological Issues and Biosocial Factors.**


