
DECOMPOSITION OF DIFFERENCES IN HEALTH EXPECTANCY BY CAUSE*

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Health expectancy is a widely used measure for monitoring trends in the health of a population and assessing differences in health among population groups. However, no decomposition method is available to examine the contribution made by causes of death and disability to differences in health expectancy among population groups or periods. We present a method for decomposing differences in health expectancy, based on the Sullivan method. This method is an extension of the decomposition method for life expectancy developed by Arriaga. We illustrate the method and its added value by decomposing male-female differences in health expectancy for the Netherlands.

Indicators of health expectancy that combine mortality and morbidity data into a single composite indicator of population health are considered an important extension of life expectancy. Indicators of health expectancy share important attractive properties with life expectancy, such as their independence from the age structure of the population and their measurement in expected years of life. An additional attractive feature is that health expectancy takes into account both mortality and the health status of the surviving population and thus provides information on the length of life (adding years to life) and the healthfulness of life (adding life to years). Indicators of health expectancy have been calculated for 49 countries for monitoring trends in population health and for assessing differences in health among population groups (Robine, Jagger et al. 2003; Robine, Romieu, and Cambois 1999). Disability-free life expectancy (DFLE) is the best-known example.

Prior studies have described differences in health expectancy among periods (for an overview, see Robine, Romieu, and Michel 2003), among socioeconomic groups (for an overview, see Crimmins and Cambois 2003), between men and women (e.g., Crimmins, Hayward, and Saito 1996; Robine et al. 1999), among regions (for an overview, see Bebbington and Bajekal 2003), and among races (see, e.g., Crimmins et al. 1996; Hayward and Heron 1999). Comparisons of population groups have revealed that major differences exist and that there is no fixed association between the length of life and the health status of the surviving population. Highly educated groups have been shown to live longer and spend less time with disability than have groups with less education (Crimmins and Cambois 2003), and women have been found to live longer and to spend more years living with disability than have men (Robine, Jagger et al. 2003). Racial differences have yielded an even more diverse picture (Hayward and Heron 1999). Comparisons over time have

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also revealed important differences. For instance, time trends for France, Germany, and the United States showed that DFLE (all levels of severity combined) is on the rise, whereas Australian data have indicated a decrease in DFLE (Robine, Romieu, and Michel 2003).

In addition to describing differences in health expectancy, it is important to understand how and why the health of a population changes over time and why there are differences in health among population groups. Knowing which age groups and diseases contribute most to described differences in population health can help to identify potential determinants of these changes and differences and can assist in the evaluation of past trends and inequalities in health. This knowledge may facilitate the definition of priorities in the field of public health and improve the assessment of targeted health priorities.

A number of prior studies have addressed the contribution of various diseases to health expectancy (for an overview, see Mathers 2003). Apart from calculations of health expectancy made by the World Bank (Murray and Lopez 1997), which were derived from individual diseases, a few studies have linked generic information on either death (Hayward, Crimmins, and Saito 1998) or disability (Colvez and Blanchet 1983; Manton and Stallard 1991) or both death and disability (Bone 1995; Mathers 1999; Nusselder et al. 1996) to disease groups. These studies have provided information about the impact of specific diseases on health expectancy by comparing the change that would occur if the disease were fully eliminated to the current situation. This approach is helpful in identifying major causes of the loss of health and in evaluating the potentials for gains in health. Its application has shed light on the large health burden of chronic nonfatal diseases, such as musculoskeletal diseases, and on the substantial gains in health that could be obtained by the prevention of these diseases. Moreover, it has made clear that preventing fatal diseases, such as circulatory diseases or cancer, would extend not only DFLE but also life expectancy with disability (Bone 1995; Hayward and Heron 1999; Mathers 1999; Nusselder et al. 1996). The cause-elimination approach, however, is less appropriate for understanding differences in health expectancy because it cannot be used to assess the contribution of specific diseases to differences in health expectancies among population groups or over time. Such an assessment requires a method that partitions differences in health expectancy into the additive contributions of various diseases.

In mortality research, decomposition (i.e., partitioning) techniques are available to assess the contribution that specific diseases or age groups make to differences in life expectancy (Arriaga 1984, 1989; Pollard 1988; Pressat 1985; Shkolnikov et al. 2001). These tools have been widely used to explain differences in the length of life between men and women (e.g., Bah 1998; Trovato and Lalu 1998), among races (e.g., Kochanek, Maurer, and Rosenberg 1994), among regions (e.g., Velkova, Wolleswinkel-van den Bosch, and Mackenbach 1997), among socioeconomic groups (e.g., Shkolnikov et al. 1991; Valkonen, Sihvonen, and Lahelma 1997), and over time (e.g., Conti et al. 1999; Martikainen, Valkonen, and Martelin 2001).

Despite the popularity of health expectancy, no method was available at the time of our study to assess the contribution that causes of death and disability make to differences in health expectancy. Thus, the aim of our study was to modify the Arriaga (1984, 1989) method for decomposing differences in life expectancy to make it applicable to health expectancy. In this article, we present a decomposition method for partitioning differences in health expectancy, calculated with the Sullivan (1971a, 1971b) method, into the additive contributions of causes of death and disability. We illustrate this method by decomposing male-female differences in health expectancy for the Netherlands.

DECOMPOSITION OF DIFFERENCES IN HEALTH EXPECTANCY

The technique for decomposing differences or changes in health expectancy presented here is based on the Sullivan method (Sullivan 1971a, 1971b) and is an extension of the decomposition method for life expectancy developed by Arriaga (1984, 1989). The

Sullivan method is the standard method for calculating health expectancy on a routine basis and uses the observed age-specific prevalence of disability (i.e., the proportion disabled) to subdivide the number of person-years into years with and without disability. The Arriaga method is frequently used to assess the contribution that specific causes of death and/or age groups make to a difference in life expectancy.

Decomposition of Changes in Health Expectancy

The decomposition method developed by Arriaga estimates the number of years added to (or removed from) life expectancy as a result of the decrease (or increase) in the central mortality rate in a given age group from a given cause. For the decomposition of changes in health expectancy, the Arriaga method needs to be extended. Whereas changes in life expectancy reflect changes in mortality rates only, changes in health expectancy (calculated with the Sullivan method) reflect changes in total mortality rates and/or changes in the proportion disabled. We first describe the decomposition into the contribution made by each of these two components and then discuss the ways in which causes of death and disability are incorporated. The decomposition method is described in terms of changes, and life expectancy with or without disability is used as the indicator of health expectancy. The procedure is identical for differences among population groups and can be applied to any definition of health expectancy.

Decomposition by kind of effect. Our starting point was the Sullivan method for calculating health expectancy at age a , with an initial cohort of 1 ($l_a = 1$). In age group, x , $x + i$ (where i is the length of the age interval), the number of person-years with disability (${}_i\pi_{x/L_x}$) is the product of the number of person-years lived (${}_iL_x$) and the proportion disabled (${}_i\pi_x$). Hence, a change in the number of person-years with disability is

$$\Delta_i\pi_{x/L_x} = \left[({}_i\pi_x + \Delta_i\pi_x) \cdot ({}_iL_x + \Delta_iL_x) \right] - {}_i\pi_{x/L_x}, \quad (1)$$

where Δ is the change between the first (t) and the second year of observation ($t + n$). Reexpression yields

$$\Delta_i\pi_{x/L_x} = ({}_i\pi_x + 0.5\Delta_i\pi_x) \cdot \Delta_iL_x + ({}_iL_x + 0.5\Delta_iL_x) \cdot \Delta_i\pi_x, \quad (2)$$

or

$$\Delta_i\pi_{x/L_x} = \left[\frac{{}_i\pi_{x(t)} + {}_i\pi_{x(t+n)}}{2} \right] \cdot \Delta_iL_x + \Delta_i\pi_x \cdot \left[\frac{{}_iL_{x(t)} + {}_iL_{x(t+n)}}{2} \right]. \quad (3)$$

The change in the number of person-years with disability is the sum of two components:

$${}_iMOR_x = \left[\frac{{}_i\pi_{x(t)} + {}_i\pi_{x(t+n)}}{2} \right] \cdot \Delta_iL_x \quad (4)$$

and

$${}_iDIS_x = \left[\frac{{}_iL_{x(t)} + {}_iL_{x(t+n)}}{2} \right] \cdot \Delta_i\pi_x. \quad (5)$$

The first component, ${}_iMOR_x$, is the change in the number of person-years with disability that is due to a change in the number of person-years lived (*ceteris paribus*). Since any change in the number of person-years lived is caused by a change in total mortality, it is referred to as the “mortality effect.” The mortality effect is the change in the number of person-years with disability that would occur if only total mortality rates were to change. A negative ${}_iMOR_x$, for instance, reflects a decline in the number of person-years with

disability in the age group $x, x + i$ that is due to an increase in the mortality rate in that age group or in younger age groups.

The second component, hereafter referred to as the “disability effect” (${}_iDIS_x$), is the change in the number of person-years with disability that is due to a change in the proportion disabled (*ceteris paribus*). The disability effect in one age group is the change in the number of person-years spent living with disability that would be found if only the proportion disabled were to change. A negative ${}_iDIS_x$ reflects a decline in the number of person-years lived with disability in the age group $x, x + i$ that is due to a decline in the proportion disabled in that age group. A similar approach was applied for decomposing the change in the number of years without disability (DFLE). In that case, however, the proportion nondisabled (i.e., $1 - \pi_x$), rather than the proportion disabled (π_x), was used in the equations.

Decomposition by cause: Mortality effect. To decompose the mortality effect (${}_iMOR_x$, see Eq. (4)) by cause, we decomposed the change in the number of person-years lived ($\Delta {}_iL_x$) into the contribution made by specific causes of death, using an adjustment of the Arriaga method. Whereas the Arriaga method decomposes the change in life expectancy by age (Arriaga 1984) and further by cause (Arriaga 1989), we needed to decompose the change in the number of person-years lived in each age interval by age and cause. This modification was necessary because the Sullivan method takes the proportion disabled in each age group and multiplies it by the number of person-years in that age group. For this reason, we reexpressed the Arriaga method. First, we distinguished between age groups in which a change in mortality occurs (“age at origin,” labeled $y, y + i$) and age groups to or from which person-years are added or removed (“age at destination,” labeled $x, x + i$). Second, we reexpressed T_x in terms of ${}_iL_x$ (i.e., age-specific contribution to T_x), given that the summation of ${}_iL_x$ over age gives T_x .

As in the Arriaga method, we defined the direct effect (${}_iDE_{xy}$), the indirect effect (${}_iIE_{xy}$), the interaction effect (${}_iI_{xy}$), and the total effect (${}_iTOT_{xy}$). The direct effect is the change in the number of person-years lived within a particular age group as a consequence of a change in mortality in that age group. The indirect effect is the number of person-years added (or removed), because a change in mortality within a specific age group produces a change in the number of survivors at the end of that age interval. The interaction effect results from the combination of the changed number of survivors at the end of the age interval and the lower (or higher) mortality rates at older ages. The ${}_iDE_{xy}$, ${}_iIE_{xy}$, ${}_iI_{xy}$, and ${}_iTOT_{xy}$ are each arranged in a table with the age groups of origin $y, y + i$ presented in the rows; the age groups of destination $x, x + i$ presented in the columns; and the ${}_iDE_{xy}$, ${}_iIE_{xy}$, ${}_iI_{xy}$, and ${}_iTOT_{xy}$, respectively, presented in the cells. The calculation of ${}_iDE_{xy}$ is performed in the same way as in the Arriaga method.

Since the direct effect refers to the change in person-years within a particular age group that has occurred as a consequence of a change in mortality in that age group, the age at origin and the age at destination are, by definition, the same. The direct effect (${}_iDE_{xy}$) of a change in mortality in the age group $y, y + i$ between time t and $t + n$ on the number of person-years lived between age $x, x + i$ is thus expressed as follows:

$${}_iDE_{xy} = \frac{l_y^t}{l_a^t} \cdot \left[\frac{{}_iL_x^{t+n}}{l_y^{t+n}} - \frac{{}_iL_x^t}{l_y^t} \right] \quad (x = y). \quad (6a)$$

$${}_iDE_{xy} = 0 \quad (x > y). \quad (6b)$$

The formula for the last open-ended age group is as follows:

$$DE_{xy} = \frac{l_y^t}{l_a^t} \cdot \left[\frac{T_x^{t+n}}{l_y^{t+n}} - \frac{T_x^t}{l_y^t} \right] \quad (y = x, \text{ open-ended age group}). \quad (7)$$

A change in mortality in the open-ended age group causes only a direct effect. For the other age groups, ${}_iIE_{xy}$ and ${}_iI_{xy}$ have to be calculated.

The indirect effect is the effect that would arise if the changed number of survivors were to remain alive after age $y + i$ for as many years as the population before the change in mortality (i.e., the life expectancy at age $y + i$ before the change in mortality). The formula for the indirect ${}_iIE_{xy}$ is

$${}_iIE_{xy} = \frac{{}_iL_x^t}{l_a^t} \cdot \left[\frac{l_y^t \cdot l_{y+i}^{t+n}}{l_{y+i}^t \cdot l_y^{t+n}} - 1 \right] \quad (x > y). \quad (8)$$

As in the Arriaga method, the interaction effect (${}_iI_{xy}$) is calculated as the difference between two components: (1) the number of person-years added (removed) in the event the additional survivors at age $y + i$ were to continue to live for as many years as the rest of the population after the change in mortality (i.e., the life expectancy at age $y + i$ after the change in mortality) and (2) the indirect effect, defined as the number of years added (removed) if the additional survivors were to remain alive under the old mortality regime. The first component, labeled ${}_iOE_{xy}$, is calculated as follows:

$${}_iOE_{xy} = \frac{{}_iL_x^{t+n}}{l_a^t} \cdot \left[\frac{l_y^t}{l_y^{t+n}} - \frac{l_{y+i}^t}{l_{y+i}^{t+n}} \right] \quad (x > y). \quad (9)$$

Using Eqs. (8) and (9), the interaction effect (${}_iI_{xy}$) is

$${}_iI_{xy} = {}_iOE_{xy} - {}_iIE_{xy} \quad (x > y). \quad (10)$$

The total contribution of a change in mortality in each age group of origin $y, y + i$, to the change in the number of person-years lived between age x and $x + i$ ($\Delta_i L_x$) is calculated as follows:

$${}_iTOT_{xy} = {}_iDE_{xy} + {}_iIE_{xy} + {}_iI_{xy} \quad (x \geq y). \quad (11)$$

In this article, the decomposition by age is an intermediate step needed to obtain the decomposition of changes in health expectancy by cause. For other research objectives, decomposition by age can be useful. The total effect by age of origin $y, y + i$ yields this information.

The Arriaga method of additive decomposition by cause (in which the total change in mortality (in a given age group $y, y + i$) equals the sum of the changes in mortality that are attributable to each cause of death within the same age group) was subsequently applied for further decomposing the total effect (${}_iTOT_{xy}$) by cause of death. The contribution of the change in mortality from each cause k in age group $y, y + i$ to the change in the number of person-years in age group $x, x + i$ (or total life expectancy in the Arriaga method), is assumed to be proportional to the contribution of this cause to the change in the central mortality rate in the age group $y, y + i$. We multiplied ${}_iTOT_{xy}$ by the contribution of the mortality change in the age group $y, y + i$ that is attributable to cause k , ${}_iC_{yk}$, to obtain the decomposition by cause:

$${}_iTOT_{xyk} = {}_iTOT_{xy} \cdot {}_iC_{yk} \quad (x \geq y). \quad (12)$$

where ${}_iTOT_{xyk}$ is the contribution of a change in mortality that is due to cause k in age group $y, y + i$ to the number of person-years lived between age $x, x + i$ (where $y \leq x$). ${}_iC_{yk}$ is calculated as follows:

$${}_iC_{yk} = \left[\frac{{}_iR_{yk}^{t+n} \cdot {}_iM_y^{t+n} - {}_iR_{yk}^t \cdot {}_iM_y^t}{{}_iM_y^{t+n} - {}_iM_y^t} \right], \quad (13)$$

where ${}_iM_y^t$ is the central mortality rate at age y , $y + i$ at time t ; ${}_iR_{yk}^t$ is the proportion of deaths from cause k in the total number of deaths in the age group y , $y + i$ at time t ; and n is the difference between the first year of observation and the second.

For each age group of destination, x , $x + i$, the ${}_iTOT_{xyk}$ are arranged in a table showing the age groups of origin (y , $y + i$) in the rows and the causes k in the columns. The contribution of cause k to the change in the number of person-years lived between age x , $x + i$ (${}_iTOT_{xk}$) is obtained as follows:

$${}_iTOT_{xk} = \sum_{y=a,x} {}_iTOT_{xyk}. \quad (14)$$

The sum of these cause-specific contributions to the change in the number of person-years lived between x , $x + i$ equals the total change in the number of person-years in that age group:

$${}_iTOT_x = \sum_k {}_iTOT_{xk}. \quad (15)$$

Using

$$\Delta_i L_x = \sum_k \Delta_i L_{xk} = \sum_k {}_iTOT_{xk}, \quad (16)$$

and

$$\Delta_i L_{xk} = {}_iTOT_{xk} \quad (17)$$

yield in Eq. (4) the mortality effect by cause (${}_iMOR_{xk}$):

$${}_iMOR_{xk} = \left[\frac{{}_i\pi_{x(t)} + {}_i\pi_{x(t+n)}}{2} \right] \cdot \Delta_i L_{xk} = \left[\frac{{}_i\pi_{x(t)} + {}_i\pi_{x(t+n)}}{2} \right] \cdot {}_iTOT_{xk}. \quad (18)$$

Summation of ${}_iMOR_{xk}$ over x gives the total mortality effect by cause, that is, the change in health expectancy that would occur if only the mortality from the specific cause k were to change.

To avoid having the results of the decomposition depend on whether the first or second time point is used as a reference, we averaged the components of the difference between time t and $t + n$ (with $t + n$ as the baseline) with the respective components of the difference between population $t + n$ and t (with t as the baseline). This additional step was proposed by Andreev and Pressat (cited in Shkolnikov et al. 2001); the original Arriaga method did not include this step.

Decomposition by cause: Disability effect. To decompose the disability effect (${}_iDIS_x$) by cause of disability for each age group, the change in the proportion disabled ($\Delta_i\pi_x$) must be allocated to different causes of disability k . Like causes of death, causes of disability should be additive, that is, the sum of all causes should equal total disability. Disability by cause can be either obtained from disability surveys (Bone 1995; Mathers 1999) or estimated using individual-level data on chronic diseases and disability (see the next section) from health surveys or clinical examinations. The change in the proportion disabled decomposed by cause can be obtained by subtracting of the proportion disabled for each cause k at time t from that at time $t + n$, or

$$\Delta_i\pi_{xk} = {}_i\pi_{xk}^{t+n} - {}_i\pi_{xk}^t, \quad (19)$$

where ${}_i\pi_{xk}^{t+n}$ is the proportion disabled from cause k in age group x , $x + i$ at time $t + n$, and ${}_i\pi_{xk}^t$ is the proportion disabled from cause k in age group x , $x + i$ at time t and where

$$\Delta_i\pi_x = \sum_k \Delta_i\pi_{xk}. \quad (20)$$

Substitution of the change in the proportion disabled by cause in Eq. (5) yields the disability effect by cause (${}_iDIS_{xk}$):

Table 1. Disease Clusters and Related Chronic Diseases and Causes of Death

Disease Clusters	Health Interview Survey ^a	Cause of Death ^b
Chronic Obstructive Pulmonary Disease	Chronic bronchitis, emphysema, asthma	490–496
Heart Disease	Heart complaints, cardiac failure	393–398, 410–414, 415–417, 420–429
Stroke	Stroke	430–438
Diabetes Mellitus	Diabetes mellitus	250
Back Complaints	Backache longer than three months, slipped disk	720–724
Arthritis	Rheumatism, arthritis, arthrosis	710–716, 719, 725–729
Cancer	Cancer	140–239
Other Diseases	All other chronic diseases	All other

^aConducted by Statistics Netherlands, 1990–1994.
^bBased on codes taken from the *International Classification of Disease*, 9th edition (World Health Organization 1977).

$${}_iDIS_{xk} = \left[\frac{{}_iL_{x(t)} + {}_iL_{x(t+n)}}{2} \right] \cdot \Delta_i \pi_{xk}.$$

(21)

Summation of ${}_iDIS_{xk}$ over x gives the total disability effect by cause, that is, the change in health expectancy that would occur if only disability from the specific cause k were to change.

Decomposition by cause: Total effect. Summation of the mortality and disability effect by cause yields the decomposition of the total change in health expectancy by cause.

ILLUSTRATION OF THE METHOD

The decomposition method can be applied to decompose differences over time (changes) or among population groups. Here, we illustrate the newly developed decomposition technique by analyzing sex differences in health expectancy using Dutch data. We have taken as our starting point the calculation of health expectancy for men and women that is based on the Sullivan life table, which uses data on total mortality and disability by five-year age groups and sex. The decomposition method also uses data on mortality and disability by cause (and five-year age groups and sex).

DATA

Mortality: Total. As input for the Sullivan method, we used abbreviated life tables for men and women for the period 1990–1994, based on data on the population and the number of deaths by age and sex from Statistics Netherlands (1995).

Mortality: Cause specific. Data on the number of deaths by age, sex, and underlying cause of death for the period 1990–1994 were also obtained from Statistics Netherlands (Statistics Netherlands annually). Causes of death were classified according to the ninth revision of the International Classification of Diseases, Injuries and Causes of Death (ICD-9; World Health Organization 1977). The selected (underlying) causes of death are summarized in Table 1. Crude mortality rates for each cause in three broad age groups for men and women, showing the highest mortality rates for heart disease, cancer, and “other diseases” are presented in Table 2.

Disability: Total. Cross-sectional data on long-term disability by sex and age were obtained from the Netherlands Health Interview Survey 1990–1994 (Statistics Netherlands

Table 2. Total and Cause-Specific Mortality Rates and Prevalence of Disability in the Non-institutionalized Population, The Netherlands, by Age Group and Sex, 1990–1994

Cause	Age Group	Cause-Specific Mortality (per 1,000)		Cause-Specific Prevalence of Disability (per 1,000)	
		Men	Women	Men	Women
Total	15–44	1.0	0.6	33.0	53.7
	45–64	7.7	4.4	171.1	211.2
	65+	63.4	46.2	297.9	484.4
COPD	15–44	0.0	0.0	1.7	1.7
	45–64	0.2	0.1	4.6	3.5
	65+	4.8	1.4	16.4	8.0
Heart Disease	15–44	0.1	0.0	0.2	0.3
	45–64	2.2	0.7	5.1	3.4
	65+	18.0	13.1	15.7	22.3
Stroke	15–44	0.0	0.0	0.1	0.2
	45–64	0.3	0.2	3.3	1.3
	65+	5.6	6.1	13.5	11.1
Diabetes Mellitus	15–44	0.0	0.0	0.1	0.8
	45–64	0.2	0.1	1.2	6.7
	65+	1.2	1.6	4.8	27.4
Back Complaints	15–44	0.0	0.0	7.2	10.8
	45–64	0.0	0.0	27.9	33.5
	65+	0.0	0.0	23.2	41.4
Arthritis	15–44	0.0	0.0	2.1	4.2
	45–64	0.0	0.0	14.1	35.2
	65+	0.2	0.4	36.1	103.1
Cancer	15–44	0.2	0.2	0.1	0.3
	45–64	3.1	2.4	0.7	2.3
	65+	18.9	9.6	5.2	5.7
Other Diseases	15–44	0.2	0.3	5.0	12.1
	45–64	3.1	0.8	14.6	22.6
	65+	18.9	14.0	29.5	36.8
Causes Not Attributable to Reported Diseases	15–44	NA	NA	16.5	23.2
	45–64	NA	NA	99.7	102.7
	65+	NA	NA	153.4	228.4

Notes: COPD = chronic obstructive pulmonary disease. NA = not available. Numbers for the specific causes by age may not sum exactly to the total because of rounding.

1994), which was based on a random sample among the noninstitutionalized population ($N = 32,936$). We used a subsample of 26,541 respondents aged 16 and older because no information on long-term disability at younger ages was available. Long-term disability was measured using the indicator of the Organization for Economic Co-operation and Development. This indicator consists of 16 items that refer to a person's ability to carry

out a number of activities of daily living, mobility, and communication that are essential for daily independent functioning (McWhinnie 1981). Similar to previous Dutch studies (Perenboom et al. 1997), we selected 10 items dealing with the ability to bend down and pick something up, to get in and out of bed, to dress and undress, to move between rooms, to walk 400 meters, to carry a 5-kilogram object for 10 meters, to read small print in a newspaper, to recognize someone's face, to have a conversation with another person, and to follow a conversation in a group. People were considered to be disabled if they indicated that they needed help from another person or were unable to carry out one or more of the selected activities included in the indicator without (great) difficulty. People who were able to carry out all the activities with some or no difficulty were considered to be without disability. Using equipment, such as eyeglasses or a hearing aid, was not considered indicative of disability if the respondent did not need help or was able to perform the activity with little or no difficulty.

We used additional data on disability in the institutionalized population (Perenboom et al. 1997), since the Health Interview Survey excluded persons living in institutions. These data included administrative data on the number of people who were living in nursing homes, psychiatric hospitals, and homes for the mentally disabled and survey data on people who were living in homes for the elderly. People who were living in institutions were considered to be disabled; however, an adjustment was made for persons without disabilities who were living in homes for the elderly.

Disability: Cause specific. We used self-reported data on the presence of disability and chronic diseases in the Health Interview Survey to estimate disability by cause in the noninstitutionalized population. The prevalence of chronic diseases was assessed on the basis of a structured list comprising a broad number of somatic diseases and a category of "other diseases." The list did not cover all chronic conditions; for example, injuries, mental and sensory disorders, diseases of the nervous system, and dementia were not included. The following groups of diseases were compiled from the original chronic conditions: chronic obstructive pulmonary disease (COPD), heart disease, stroke, diabetes mellitus, back complaints, arthritis, cancer, and one group that included all other diseases (see Table 1). The presence or absence of these diseases, as well as age, was used in a multivariate regression model to estimate the proportion disabled associated with each chronic disease. For more information on the modeling approach, see the appendix.

Table 2 presents the prevalence of disability (proportion disabled) by cause in three broadly defined age groups for men and women. Similar to prior studies, (e.g., Bone 1995; Ettinger et al. 1994; Guccione et al. 1994; Mathers 1999; Nusselder et al. 1996), including one study that was based on medically validated information on diseases (Guccione et al. 1994), important causes of disability in our data set are arthritis; back complaints; "other diseases"; and, to a lesser extent, heart disease, stroke, and COPD. Cancer is not an important cause of disability. The contribution of "not attributable to reported diseases" was found to be strikingly high, which may reflect disability from diseases that were not included as separate entities in the checklist, as well as disability that was not caused by specific chronic diseases or injuries. The strong age dependence of this cause suggests that disability that is not attributable to reported diseases is associated with aging-related functional losses. This finding is in line with data from the Women's Health and Aging Study, in which numerous women, when asked to report on the main condition that caused their disability, responded "old age" or "no specific disease" (Leveille, Fried, and Guralnik 2002).

Sex differences in the prevalence of total and cause-specific disability are in line with prior studies (for an overview, see Leveille, Resnick, and Balfour 2000; Oman, Reed, and Ferrara 1999) that showed that women have a higher prevalence of disability, particularly from arthritis and causes that are "not attributable to reported diseases" (aged 65 and older), and, to a somewhat lesser extent, from diabetes mellitus and back complaints.

Table 3. Total Life Expectancy (LE), Life Expectancy With Disability (LWD), and Life Expectancy Without Disability (DFLE) at Age 15 for Men and Women and Male-Female Difference, The Netherlands, 1990–1994

	LE (in years)	DFLE (in years)	LWD (in years)
Men	59.9	51.5	8.4
Women	65.8	51.1	14.8
Male-Female Difference (men = baseline)	5.89	–0.48	6.36

Note: Numbers may not sum exactly to the total because of rounding.

Sex Differences in Health Expectancy

In the Netherlands, total life expectancy at age 15 is 59.9 years for men and 65.8 years for women, of which 51.5 years and 51.1 years, respectively, are spent without disability. The remaining 8.4 and 14.8 years are spent with disability (see Table 3). These figures include disability in the institutionalized population. Although the difference in total life expectancy is 5.89 years in favor of women, women spend 6.36 more years with disability and 0.48 of a year less without disability than do men.

Although sex differences in total mortality rates and in the prevalence of disability (for a summary, see Table 2) can shed some light on these differences in health expectancy, the specific causes that contribute most to these differences remain unclear. Also, the virtual absence of a sex difference in DFLE is not easily understood. Decomposition of the sex differences in health expectancy can be helpful in understanding these differences in health expectancy better.

Decomposition by Kind of Effect

The first step in the decomposition analysis is to partition the sex difference in health expectancy by kind of effect, that is, into the contribution made by differences in total mortality and by differences in the prevalence of disability. This step shows that 3.55 out of the 6.36 additional years with disability reflect a higher prevalence of disability in women. The remaining 2.82 years reflect lower total mortality (see Table 4). During the extra years of life, women are exposed to high risks of disability. By definition, the size of the disability effect is the same for DFLE as for life expectancy with disability, but the direction differs; higher disability contributes to a lower DFLE. The disability effect of –3.55 years completely nullified the mortality effect of 3.1 years. The net difference was small, but masked considerable sex differences in total mortality and the prevalence of disability.

Table 4. Decomposition of the Male-Female Difference in Total Life Expectancy (LE), Life Expectancy With Disability (LWD), and Life Expectancy Without Disability (DFLE) Into the Mortality and Disability Effect at Age 15, The Netherlands, 1990–1994

	LE (in years)	DFLE (in years)	LWD (in years)
Total Difference (men = baseline)	5.89	–0.48	6.36
Mortality Effect	5.89	3.07	2.82
Disability Effect	0	–3.55	3.55

Note: Numbers may not sum exactly to the total because of rounding.

Table 5. Decomposition of the Mortality Effect, Disability Effect, and Total Difference in Health Expectancy at Age 15, by Cause, The Netherlands, 1990–1994 (men = baseline)

Cause	DFLE			LWD			LE
	Mortality	Disability	Total	Mortality	Disability	Total	Total
Total	3.07	−3.55	−0.48	2.82	3.55	6.36	5.89
COPD	0.22	0.15	0.37	0.29	−0.15	0.14	0.51
Heart Disease	1.03	−0.01	1.01	0.89	0.01	0.91	1.92
Stroke	0.09	0.07	0.17	0.10	−0.07	0.02	0.19
Diabetes Mellitus	0.02	−0.39	−0.37	0.00	0.39	0.39	0.02
Back Complaints	0.00	−0.43	−0.43	0.00	0.43	0.44	0.00
Arthritis	−0.01	−1.21	−1.22	−0.01	1.21	1.20	−0.02
Cancer	0.78	−0.03	0.74	0.90	0.03	0.94	1.68
Other Diseases	0.95	−0.38	0.57	0.64	0.38	1.02	1.59
Cause Not attributable to Reported Diseases	0.00	−0.82	−0.82	0.00	0.82	0.82	0.00
Institutions	0.00	−0.49	−0.49	0.00	0.49	0.49	0.00

Notes: DFLE = life expectancy without disability, LWD = life expectancy with disability, LE = total life expectancy, and COPD = chronic obstructive pulmonary disease. Numbers may not sum exactly to the total because of rounding.

Decomposition by Cause

To obtain information on the contribution of various diseases to the difference in health expectancy, we further decomposed the mortality effect and disability effect by causes. This decomposition took into account the fact that diseases can have at least three effects: they can (1) affect the age-specific total mortality rate and thus affect life expectancy, (2) affect the age-specific total mortality rate and thus affect the disability burden that is due to changes in the age distribution, (3) and affect the prevalence of disability. The decomposition of the mortality effect by cause takes into account the first two effects, and the disability effect by cause takes into account the third effect. It is worth mentioning that the latter reflects both differences in the prevalence of a disease and differences in the disability associated with the disease.

Mortality effect by cause. The contribution that differences in mortality from various causes make to the sex difference in health expectancy is shown in Table 5. Of the 2.82 more years that women live with disability owing to lower mortality, 0.89 year is due to differences in mortality from heart disease, 0.90 year from cancer, and 0.64 year from “other diseases.” The contribution of these causes to the difference in life expectancy with disability is not the same as to the difference in DFLE. The contribution of cancer to the difference in DFLE is slightly smaller (0.78 year) and that of heart disease and “other diseases” is larger (1.03 years and 0.95 year, respectively). These differences reflect differences in the ages at which these cause-specific mortality differences occur.

Disability effect by cause. Decomposition of the disability effect by cause shows that the most important explanation for the male-female differences in health expectancy, as far as cause of disability is concerned, is arthritis (see Table 5). Higher disability in women from arthritis is responsible for 1.21 additional years with disability. A striking finding was that a large part of the disability differences proved not to be attributable to reported diseases. This finding may reflect more disability in women from

diseases that were not measured in the survey and differences in constitutional factors, such as muscle strength and bone density, which may result in a greater predisposition toward disability. It is noteworthy that the small contribution of heart disease to the disability effect does not imply that heart disease is not an important cause of disability; it merely indicates that sex differences in the prevalence of disability associated with heart disease are small. Institutions as a "cause" of disability refer to disability in the institutionalized population; we had no data to estimate cause-specific disability in this population.

Total effect by cause. The contributions of causes of death and disability to differences in health expectancy are also shown in Table 5 (column total). Arthritis contributes most to the sex difference in life expectancy with disability (1.20 years), followed by "other diseases" (1.02 years), cancer (0.94 year), and heart disease (0.91 year). With regard to the causes that are responsible for the sex differences in DFLE, both positive and negative contributions were found. Positive contributions of heart diseases (+1.01), cancer (+0.74), and other diseases (+0.57 year) yielded a larger DFLE in women. The negative contributions of arthritis (−1.22), disability that was not attributable to reported diseases (−0.82), disability in institutions (−0.49), and back complaints (−0.43 year) completely nullified this effect, thus explaining the slightly smaller DFLE in women.

DISCUSSION

Despite the popularity of health expectancy as an indicator of population health, no method had yet been developed at the time of our study to assess the contribution of causes of death and disability to differences in health expectancy among population groups or time periods. The aim of our study was to fill this gap by developing a tool to decompose differences in health expectancy, based on the Sullivan method.

We illustrated this new tool in this article by examining male-female differences in health expectancy for the Netherlands from 1990 to 1994. Although the sex difference in total life expectancy was 5.89 years in favor of women, women spent 6.36 years longer with disability and 0.48 year less without disability. The slightly lower DFLE, which contrasted with the higher DFLE for women found in most other countries (Robine and Ritchie 1991; Robine et al. 1999), relates to the disability indicator, which includes mild disability (Perenboom et al. 1997). Decomposing the sex differences in life expectancy with disability by cause showed that the causes that contributed most to the difference were arthritis, heart disease, cancer, "other diseases," and causes that were not attributable to reported diseases. Decomposition of the sex difference in DFLE showed that positive contributions for some causes, the largest of which were for heart disease, cancer, and other diseases, were completely canceled out by negative contributions for other causes, including arthritis and those that were not attributable to reported diseases.

Another possible application of this method, which has not been discussed here, is the decomposition of changes over time. Such a decomposition would provide information on the contribution made by different causes of death and disability to an observed change in health expectancy.

LIMITATIONS

Some caution must be exercised when interpreting the results of the example chosen to illustrate this newly developed application of the decomposition method and when using the method in further applications. While some limitations are specific to the data used in the illustration and can be avoided, to a large extent, in further applications of the method, others are more fundamental to the method.

Data. First, the example was based on self-reported data on disability and chronic diseases. Since medical registrations, in general, do not include information on disability

and include incomplete information on the population at risk, using data on disability that were collected in surveys is a standard approach to calculate health expectancy. Disability in large-scale surveys is generally self-reported, although some surveys include performance-based measures of disability. Relying on respondents' self-reports of disability may have biased the results of our illustration, particularly because reporting behavior may differ by sex. Although performance-based measures cannot be considered the "gold standard," comparing self-reports with performance-based measures suggests that women are truly more disabled than are men (Merrill et al. 1997). Using self-reports of chronic diseases to estimate cause-specific disability in the illustration may have also induced some uncertainty, since previous studies that compared self-reported data to data that were obtained from clinical examinations or medical records found discrepancies for some diseases (Heliovaara et al. 1993; Schrijvers et al. 1994). The results of the example should be viewed with this potential limitation in mind, and applications of the method should, when possible, use clinical data on the presence of diseases (such as the data that are available in the Framingham Study; see Guccione et al. 1994).

Second, the causes of disability in the illustration were based on statistical associations, not on etiologic information or self-reports of causes of disability. We assumed that the diseases and conditions that caused disability were still present and reported in the survey. Violation of this assumption is most likely for irreversible disability caused by reversible diseases or conditions, such as injuries, and for diseases that were not included (as separate entities) in the list of chronic diseases, and may have overvalued the role of disability that is not attributable to diseases. To maximize the comparability with the causes of death, we mapped disability to single disease groups; that is, we did not take into account interactions between causes (on an additive scale). Nonetheless, comparisons of diseases as causes of death and disability may still be flawed. Conclusions that are based on the decomposition of the total effect, where causes of death and disability are added, should be approached with some care.

Third, in the illustration, we assumed that persons in institutions were disabled (except those in homes for the elderly) and treated institutionalization as a separate "cause" of disability. This choice was preferable to assuming the same cause-of-disability distribution as in the noninstitutionalized population, considering that a higher proportion of the institutionalized population suffers from severely disabling diseases, such as dementia and stroke. We do not think that treating institutionalization as a separate cause affected the outcomes to any considerable extent, given that the contribution of "institutionalization" to the sex difference in health expectancy was only 0.5 year. Nonetheless, in further applications of the method, the population living in institutions should be included, when possible, in the main data source.

Finally, in comparisons of two different populations, differences in the age distribution may affect health differences. Most of these differences are taken into account in the decomposition technique because the method uses age-specific disability and mortality data. Nonetheless, differences within age groups (5-year age groups, with age 85 and older as the oldest group in the illustration) may still have affected the outcomes, which can be avoided in further applications, to a large extent, by using smaller age intervals if the sample size permits.

Methods. The decomposition method is based on the Sullivan method because this is the standard method for calculating health expectancy on a routine basis. The major limitations (and strong points) of the Sullivan method also apply to the decomposition tool. Health expectancy derived from the Sullivan method reflects the current health composition (Crimmins 2002). Although the stock of a population's health results from a population's life history of moves into and out of disability and mortality before the time of observation, the Sullivan method ignores the link with these underlying flows. As a result, it generally does not produce a pure period indicator, such as (period) life expectancy

(Barendregt, Bonneux, and Van der Maas 1995; Robine, Michel, and Branch 1992; Robine and Ritchie 1991). For measuring the health status of the current population, the Sullivan method is considered to be more valuable than is a pure period indicator (Barendregt 2002; Crimmins 2002). However, the deviation from a pure period indicator can introduce bias (Barendregt et al. 1995; Barendregt, Bonneux, and Van der Maas 1997; Mathers and Robine 1997; Robine et al. 1992; Robine and Ritchie 1991; Van de Water et al. 1995). Although less concern is warranted for comparisons among population groups, changes in health expectancy that are assessed with the Sullivan method should be viewed with some caution. Two simulation studies (Barendregt, Bonneux, and Van der Maas 1994; Mathers and Robine 1997; see also Barendregt et al. 1997; Mathers and Robine 1997; Van de Water et al. 1995) that compared the outcomes of the Sullivan method with a pure period indicator showed that serious biases are expected to occur when sudden, large changes occur, but that the Sullivan method provides acceptable estimates of the true period value of health expectancy if there are smooth and relatively regular changes in transition rates over a reasonably long-term period.

The fact that the decomposition method is based on the Sullivan method has consequences for its applicability and for the interpretation of the outcomes of the decomposition analyses. The method should not be applied when large and sudden changes in underlying transition rates are expected. For the interpretation of the results, it is noteworthy that the decomposition method quantifies the extent to which differences in the prevalence of disability and *total* mortality (in each age group) contribute to differences in health expectancy. It cannot assess the contribution of underlying flows.

Comparisons With Prior Research

Although, as we mentioned in our introductory section, several studies have found differences in health expectancy among population groups and over time, only one study (Robine, Mormiche, and Sermet 1998) examined causes that contribute to the observed changes or differences in health expectancy. This work, which examined the increase in DFLE in France in the period 1981–1991, focused on change in the prevalence of disability. The contribution of mortality reductions (by cause) to the increase in DFLE was not quantified, despite the fact that total life expectancy increased by 2.5 years.

Since no prior study has assessed the contribution that causes of death and disability make to sex differences in health expectancy, we can only confirm that the results of our example are compatible with what is known about sex differences in health. Previous evidence pointed toward men having more common fatal diseases, such as heart disease and cancer, and toward a higher prevalence of nonfatal chronic diseases (the most important cause being arthritis) and lower muscle strength and bone density in women (Crimmins et al. 1996; Leveille, Resnick, and Balfour 2000; Oman et al. 1999; Verbrugge 1989). The effects of these differences on health expectancy are clearly illustrated in our example. We showed that most of the 6.4 additional years that women spent with disability were caused by higher disability from arthritis (+1.2) and disability that was not attributable to diseases (+0.8), lower mortality from heart disease (+0.89) and cancer (+0.90), and a combination of lower mortality and higher disability from “other diseases” (+1.0).

Added Value

Without a decomposition tool, it is difficult, if not impossible, to gauge how differences in mortality and disability at *different* ages *together* affect health expectancy, especially since the effect of mortality differences on health expectancy is complex. The decomposition tool takes into account the sometimes-opposite effects of diseases as a cause of death and disability on health expectancy simultaneously. More specifically, the tool links a mortality difference (e.g., the lower observed cause-specific mortality rate) in one age group, through its impact on the number of person-years lived in this age group and in all

older age groups, to the observed differences in health expectancy, while taking into account the proportion disabled in the added or lost years.

The importance of looking at differences in both mortality and disability becomes clear when some of the results presented in Table 5 are compared with the cause-specific disability and mortality data presented in Table 2. First, although the prevalence of disability from COPD in women is lower than it is in men, COPD contributed to a longer life expectancy with disability in women (+0.14) because the effect of the lower prevalence of disability (−0.15) was completely canceled by lower mortality from COPD (+0.29). Second, while women showed higher disability both from arthritis and “other diseases,” the overall contribution of arthritis to the difference in DFLE was negative (−1.22) but that of “other diseases” was positive (+0.57). Lower mortality from “other diseases” (+0.95) nullified the negative contribution of higher disability from “other diseases” (−0.38).

The importance of taking into account that differences in total mortality and the prevalence of disability vary by age (in size and direction) is illustrated in the example, in which mortality differences from heart disease, occurring at younger ages than differences in cancer, contributed most to the sex difference in DFLE, and mortality differences from cancer contributed most to the sex difference in life expectancy with disability.

CONCLUSION

Decomposition by cause of differences in health expectancy between population groups (or over time) using mortality and disability data by cause is possible. The decomposition tool takes into account the consequences for health expectancy of differences occurring in various age groups, from various causes of death and disability simultaneously, which allows for a better understanding of the contribution that various diseases make to differences in population health than was previously possible. This tool may facilitate the definition of priorities in the field of public health and may improve the assessment of targeted health priorities.

APPENDIX: ESTIMATION OF DISABILITY DATA BY CAUSE

The estimation of disability data by cause is based on information on the presence or absence of disability, the presence or absence of various diseases, and age. All analyses are conducted separately for men and women. The basic principle is that for each disease exposure, the risk of disability is estimated, based on the occurrence of disease and disability in the population. The method takes into account the fact that subjects without a reported disease can be disabled (this risk is referred to as “background”) and that subjects can have more than one disease (comorbidity). Disability in persons without a disease is entirely attributed to background. Disability in persons with one disease is attributed partly to background and partly to the disease. Disability in persons with two or more diseases is partly attributed to background and partly to each disease. To explain the method, we distinguish only two disease groups (A and B) and one age group. In the last section, we describe the procedure for more diseases and age groups.

Assumptions

First, using cross-sectional data, we assume that the distribution of disability by cause at the time of the survey is explained entirely by diseases that are still present at the time of the survey plus the background risk. Second, we assume that this distribution is proportional to the distribution of the risk of becoming disabled in the period preceding the survey. Third, we assume that causes of disability (diseases and background risk) act as independently competing causes. This assumption is necessary to map disability to single disease groups. However, interactions between causes can be easily added for other research objectives.

General Principle

First, we categorize individuals according to disease exposure. We divide subjects into subgroups, defined by the combination of causes, that is (1) no disease (i.e., only background risk), (2) only disease A, (3) only disease B, and (4) both disease A and B. All persons are exposed to the same background risk of disability (within one age-sex group). In Group 1, this background risk is the only risk. In Groups 2–4, persons are exposed to A (Group 2), B (Group 3), or A and B (Group 4) in addition to this background risk. Within Groups 2–4, competing risks of disability exist; consequently, the number of persons disabled from one specific cause depends on the risk of the competing cause or causes as well.

Analogous to the multidecrement life table, which was used to analyze observed distributions of deaths by cause in a similar situation of competing causes of death (Manton and Stallard 1984), we used (hazard) rates to obtain “crude probabilities.” Crude probabilities are probabilities in the situation in which several causes are acting simultaneously. Crude probabilities can be calculated using the ratios of the cause-specific rates in the total rate. The property of additivity of rates is applied to decompose the total rate into cause-specific rates, with the sum of the cause-specific rates yielding the total rate (Manton and Stallard 1984).

Calculation of crude probabilities for each subgroup. First, to obtain total disability rates, the probability of disability (q), which approximates the proportion disabled (π), is converted into a (hazard) rate, using (Manton and Stallard 1984)

$$\text{rate (tot)} = -\ln(1 - q). \quad (\text{A1})$$

The total rate in our approach equals the sum of cause-specific rates (additivity of rates), where both diseases and the background risk are considered as causes, or

$$\text{rate(tot)} = \text{rate(bg)} + \text{rate(A)} \cdot X_A + \text{rate(B)} \cdot X_B, \quad (\text{A2})$$

where X_A is a dummy variable for the presence of cause A, and bg is background. Second, the cause-specific rates are obtained by comparing the groups with and without the cause. The total rates for the different groups can be decomposed into

$$\text{rate(tot}_{\text{bg}}) = \text{rate(bg)} \quad (\text{A3})$$

$$\text{rate(tot}_A) = \text{rate(bg)} + \text{rate(A)} \quad (\text{A4})$$

$$\text{rate(tot}_B) = \text{rate(bg)} + \text{rate(B)} \quad (\text{A5})$$

$$\text{rate(tot}_{AB}) = \text{rate(bg)} + \text{rate(A)} + \text{rate(B)}. \quad (\text{A6})$$

These relationships are used to calculate the rates of background and the different causes. Regression analysis (see the next section) gives the best estimates of the rates of each cause under the assumption of independent risks.

Third, the proportional distribution of each cause-specific rate is used to obtain crude cause-specific probabilities for each group. For instance, for Group 4 (exposed to A and B), it gives

$$q_{\text{bg}} = q \times (\text{rate(bg)} / \text{rate(tot}_{AB})) \quad (\text{A7})$$

$$q_A = q \times (\text{rate(A)} / \text{rate(tot}_{AB})) \quad (\text{A8})$$

$$q_B = q \times (\text{rate(B)} / \text{rate(tot}_{AB})). \quad (\text{A9})$$

Formulated in Regression Terminology

Using the exponential method to convert the probability (q) into a (hazard) rate (Manton and Stallard 1984),

$$q = 1 - \exp[-\text{rate}(\text{tot})]. \quad (\text{A10})$$

The model is specified as follows in regression terminology:

$$\hat{Y} = 1 - \exp\left[-\left(\alpha + \sum_{\text{cause}} \beta_{\text{cause}} \cdot X_{\text{cause}}\right)\right], \quad (\text{A11})$$

where \hat{Y} is the estimated probability (proportion disabled), α is the background disability rate, β_{cause} is the cause-specific disability rate (e.g., $\text{rate}(A)$), and X_{cause} is a dummy variable for the presence of disease (e.g., A). Cause-specific rates are estimated in a maximum-likelihood model with a binomial distribution for Y .

Including more ages and causes. The regression model can be easily extended to more than two diseases (eight in our illustration) and applied to populations that include several age groups. To avoid strong assumptions that the background risk and/or disabling effect is the same in all age groups, we included a background risk for each age group and modeled the disabling impact by age. We used a parameterization of the age pattern of the disabling impact (based on splines), rather than using separate β s for each age group, and assumed the same age pattern for each disease (except for stroke in men). Since disability that is due to the (age-dependent) background risks is likely to include disability from diseases that were not reported in the survey, this background risk is referred to as not attributable to reported diseases.

Calculation of the number of disabled persons, by cause, across subgroups. Each subgroup, defined by the combination of causes (and age and sex) has its own probability of disability, which is the sum of the cause-specific crude probabilities. Multiplied with the number of persons in the subgroup, it yields the number of persons with disability caused by each disease in the group with this combination of diseases. Adding all these cause-specific numbers over all groups in which the disease occurs gives the total number of disabled persons by this cause.

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