

Main components of life expectancy increase during the cardiovascular revolution in Spain

Principales componentes del aumento de la esperanza de vida durante la revolución cardiovascular en España

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ABSTRACT

The circulatory system diseases have contributed decisively to an increase in life expectancy (LE) in Spain. The contribution to LE is calculated through a decomposition analysis by sex and five-year age groups. We divide the years

studied into two periods, 1980–1996 and 1996–2012. Using the Human Cause-of-Death Database (HCD), we examine specific subcauses at a 4-digit ICD-10 level and how they contribute to the change in LE among men and among women. The analysis shows that cerebrovascular diseases (CBVDs) contribute most to years gained until 1996, while ischemic heart diseases (IHDs) contribute most thereafter. Among women, the largest increase is due to specific CBVDs subcauses; among men IHD subcauses also have an important role. Regarding contribution by age, gains by CVDs are particularly significant at older ages, while contributions by IHDs are more relevant from the age of 50 onwards, especially among men. Furthermore, the gender gap in LE is influenced by the different evolution of various circulatory diseases during the period of study, but the evolution of these diseases is not always reflected equally in both sexes. The study evidences the need for greater precision in the registers in order to take advantage of the potentialities of the 4-digit classification of the ICD, thus leading to a better in depth knowledge in health trends. Finally, it shows the mortality due to modifiable factors mainly classified in IHDs, and the consequent need for the Spanish health system to act on them.

KEY WORDS

Life expectancy decomposition, Gender/sex differences, Cardiovascular Revolution, Causes of death, Health in Spain.

RESUMEN

Las enfermedades del sistema circulatorio, han contribuido de manera decisiva al aumento de la esperanza de vida (LE) en España. Las contribuciones a la LE se calculan a través de un análisis de descomposición por sexo y grupos de edad quinqueniales. Dividimos los años estudiados en dos períodos, 1980–1996 y 1996–2012. Utilizando la Human Cause-of-Death Database (HCD), examinamos subcausas específicas a un nivel de 4 dígitos de ICD-10 y cómo contribuyen al aumento o disminución de LE tanto en hombres como en mujeres. El análisis muestra que las enfermedades cerebrovasculares (CBVD) son las que más contribuyen a los años ganados hasta 1996, mientras que las enfermedades isquémicas (IHD) son las que más contribuyen posteriormente. Entre las mujeres, el mayor aumento se debe a subcausas específicas de las CBVD; en los hombres, las subcausas de IHD también tienen un papel importante. En lo que respecta a la contribución por edad, las ganancias por CBVDs son particularmente significativas en las edades mayores, mientras que las contribuciones por las IHD son más relevantes a partir de los 50 años, especialmente entre los hombres. La brecha entre hombres y mujeres en la LE está influenciada por la evolución diferente de varias enfermedades circulatorias durante el período de estudio, pero la evolución de estas enfermedades no siempre se refleja por igual en ambos

sexos. Además, se pone en evidencia la necesidad de una mayor precisión en los registros para aprovechar las potencialidades de la clasificación a 4 dígitos de la CIE, alcanzando de este modo un conocimiento más profundo de las tendencias de salud. Finalmente, el estudio muestra la mortalidad debida a factores modificables que se clasifican principalmente en las IHD, y la consiguiente necesidad de que el sistema de salud español actúe sobre ellas.

PALABRAS CLAVE

Descomposición de la esperanza de vida, Diferencias por género/sexo, causas de muerte, revolución cardiovascular, salud en España.

1. INTRODUCTION¹

The epidemiologic and cause-specific mortality transformations that have triggered the process of increasing longevity have been referred to as the “Epidemiologic Transition” (Omran 1971). Well advanced in the transition process, after many changes in morbidity patterns and a continued decline in mortality in subsequent stages (Olshansky and Ault 1986; Robine 2001), deaths are concentrated in a narrower age interval and delayed to increasingly advanced ages (Canudas-Romo 2008). In this research we focus on the so-called “Cardiovascular Revolution” (Vallin and Meslé 2001, 2004), analysing mortality in the Spanish population caused by specific cardiovascular diseases from 1980 to 2012 and their impact by sex and age.

Since the last century Spanish mortality has experienced an extraordinary decline and consequently, life expectancy (LE) has increased progressively. However, the gain in years has not been equal by sex or across all ages. In a first phase (around the 1930s), the increase in LE at birth accelerated sharply as a result of the decline in infant and child mortality (Gómez-Redondo 1995; Sanz-Gimeno and Ramiro-Fariñas, 1999). In the stages following the epidemiologic transition, from the 1960s onwards, increased longevity was driven by older and more advanced ages, where the gain was greater for women than men (Robles-González et al., 1996; Gómez-Redondo 1997; López-Abente et al., 2002; Canudas-Romo et al. 2008), and Spain is at the forefront, among the highest female LE world-

¹ Our research on causes of death in the Spanish population stems from two consecutive R&D projects: ‘Transformations in ageing, longevity, and old age in Spain. From 50 to 100 and above. Present and future’ (CSO2010-18925); and ‘Longevity, Health, and Well-being Flows in Informal Care in Southern Europe’ (CSO2014-54669-R). Both projects were funded by the Spanish Ministry of Economy, Industry and Competitiveness through grants awarded to RGR - UNED. This research also forms part of the international project, ‘Mortality Divergence and Causes of Death’ (MODICOD), funded by the AXA Research Fund and “Diverging Trends in Mortality and Future Health Challenges” (DIMOCHA), funded by the Agence Nationale de la Recherche of France (ANR-12-FRAL-0003-01).

wide (Gómez-Redondo 2015; Pérez-Moreda et al., 2015). Nevertheless, since the latter years of the 20th century, gains in LE expectancy have been higher in men, consequently reducing the gender gap (Vallin and Meslé 2004). Gains in LE during the first phases of the transition were due to a decline in infectious disease mortality, mainly among children. The subsequent increase in LE at older ages has largely been the result of the so-called cardiovascular revolution, which has led to a decline in deaths caused by circulatory diseases (Vallin and Meslé 2004).

This study adds to the proliferative research conducted on the Spanish cardiovascular revolution (Gómez-Redondo 1995, 1997, 2015; Boix-Martinez et al., 2003; Gómez-Redondo and Boe 2005; García-González 2013; Gómez-Redondo et al., 2014; Pérez-Díaz et al., 2016; Requena, 2017; García-González and Grande 2018a). We aim to analyse the contribution in life years gained or lost in specific cerebrovascular diseases (CBVDs) and ischaemic heart diseases (IHDs) in the last thirty years (from 1980 to 2012), using the 4-digit code level of detail of the International Classification of Diseases (ICD) 10th revision. We use data from the Human Cause-of-Death Database (HCD), which implies performing a cause-of-death analysis of continuous and homogenised series from ICD-9 and ICD-10. We process these data series according to the 'Reconstruction of Causes of Death' method proposed by Meslé and Vallin (1988, 1996). The 4-digit classification indicates aetiology, anatomic site, severity, and other clinical details, providing more precise information about mortality from specific causes. Thus, the contribution of each specific cause to increased LE is identified, focusing in particular on changing rates during the process. And considering that current trends in Spanish mortality are determined by deaths at older ages, we pay special attention to these deaths and to sex differences.

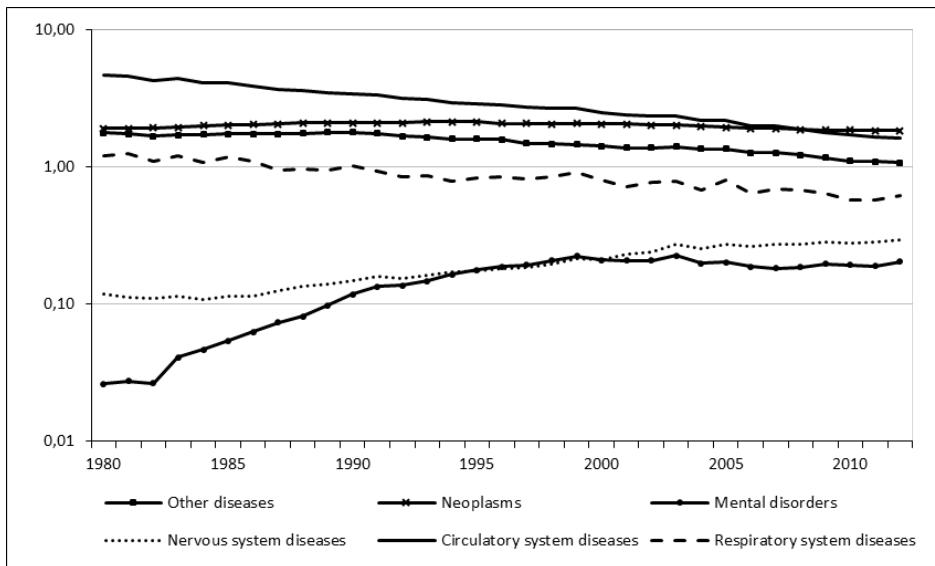
In this context, despite the advances in the knowledge of the aetiology of CBVDs and IHDs and the improvement in sanitary measures, there is an open question about the possible decrease in mortality caused by these diseases. On the one hand, if the prevalence of obesity and diabetes continues to grow in the near future, the tendency to decrease mortality due to CBVDs and IHDs could be modified. Therefore, there are deaths that could be avoided with the implementation of health prevention policies that intervene in modifiable risk factors (Palomeras and Casado 2010). On the other hand, these diseases affect population in very long-lived age groups, and it might not be possible to continue reducing their mortality at the same rate. To this end, our aim is to calculate gains and losses more accurately by specific CBVDs and IHDs diseases and provide a more solid knowledge about demographic and social context base upon which health policies can be proposed and improved, thereby reducing avoidable deaths.

2. THE EVOLUTION OF MORTALITY IN SPAIN

Precedent studies indicate that five main cause-of-death groups concentrate old age mortality in Spain, and for this reason, Gómez-Redondo (1997) calls them “the five pillars of Spanish mortality” when reporting findings on recent evolution of mortality. The five groups are the circulatory system, neoplasms and the respiratory system diseases, which have the biggest weight in current structure of Spanish mortality, together with mental disorders and nervous system diseases, which are emerging and rising causes of death (Figure 1). In a later analysis covering the period 1980–2006, Gómez-Redondo et al. (2014) revisited the same causes of death, showing that the decrease in Spanish mortality was mainly due to the reduction by almost two thirds in mortality from circulatory system diseases. And between 1980 and 2012, mortality from circulatory system diseases continued the same trend for reduction; specifically, the standardised mortality rate was 4.6‰ in 1980 and became 1.6‰ in 2012. This constant downward trend in mortality is the result of what can be considered as a cardiovascular revolution; and circulatory system diseases act as a fundamental pillar in the increase of longevity observed during the last three decades in Spain (García-González, 2013; Pérez-Moreda et al., 2015).

It is further confirmed that cerebrovascular diseases (CBVDs) and ischaemic heart diseases (IHDs) are leading the decline of mortality from circulatory system diseases, although with different timelines and a different evolution for men and women (Gómez-Redondo 1995; López-Abente et al., 2002; Gómez-Redondo and Boe 2005; Blanes 2007; Gómez Redondo et al. 2010; García-González et al. 2012; Gómez-Redondo et al. 2014; Bolúmar-Montrull and Rodríguez-Arenas, 2016).

Figure 1. Evolution of the main causes of death, standardised rates², logarithmic scale (1980–2012)



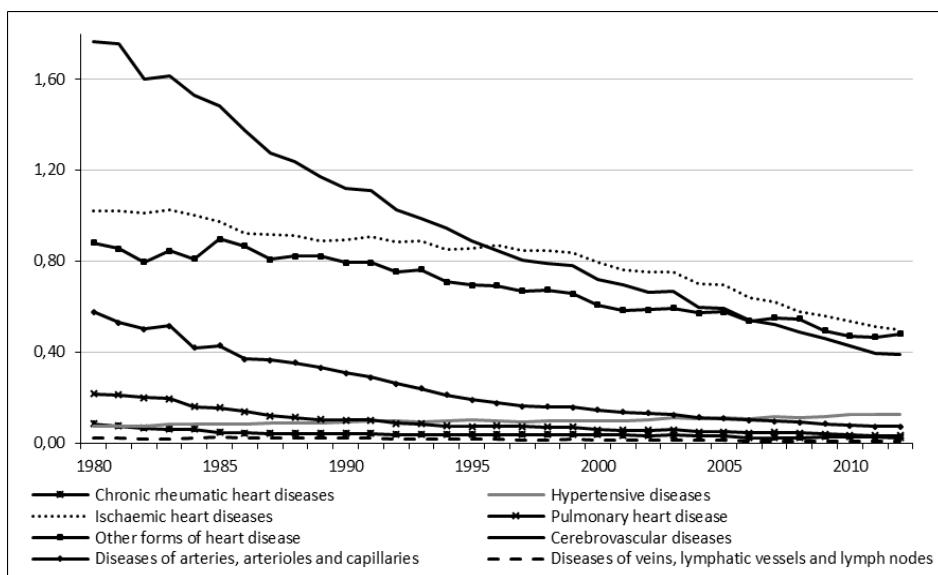
Source: Compiled by the authors from HCD data

Diseases of the circulatory system are distributed across ten cause-of-death subgroups in ICD-10 (I00–I99). Deaths attributed to each circulatory disease subgroup have declined during the period 1980–2012 (Figure 2). Specifically, CBVDs (I60–I69) and IHDs (I20–I25) have a notable impact and relevance, contributing decisively to the cardiovascular revolution (Faus-Bertomeu et al. 2016a). With regard to other cause-of-death groups included in circulatory diseases, ‘Diseases of arteries, arterioles and capillaries’ (I70–I79) and ‘pulmonary heart disease’ (I26–I28) have a lower mortality rate than CBVD and IHD, but are noteworthy because of their significant decline. The only subgroup that shows a growing trend, albeit with low mortality rates, is ‘hypertensive diseases’ (I10–I15), overtaking ‘pulmonary heart disease’ (I26–I28) and ‘diseases of arteries, arterioles and capillaries’ (I70–I79). Within this broad set of circulatory diseases, two other subgroups are also striking: ‘other forms of heart disease’ (I30–I52), which decreases drastically, and ‘other and unspecified disorders of the circulatory system’ (I95–I99), which also shows a downward trend. The decline in these subgroups reflects improved cause-of-death diagnostics.

² In terms of interpretation, ill-defined causes provide an indirect measure of data quality and they highlight specific diagnostic and coding problems. To avoid this obstacle, and with the aim of comparing more countries with varying proportions of ill-defined causes, the HCD drew up a protocol whereby it was decided to distribute ill-defined causes proportionally within the well-defined ones, which is why they are absent in the Figure.

An interesting feature of the epidemiologic transition in Spain is the decline in mortality from CBVDs in early phases (García-González 2013, Faus-Bertomeu et al. 2016a). Mortality from CBVDs falls exponentially throughout from 1980 to 2012 (Figure 3). Mortality from IHDs also shows a clear decline, but at a more moderate rate and in both sexes (López-Abente et al., 2002; Gómez-Reidondo 2015; Pérez-Moreda et al., 2015). In 1980 IHDs had notably lower mortality rates than CBVDs. However, at the end of the period, with the marked fall in CBVDs mortality, IHDs have higher mortality rates than CBVDs. The turning point is 1996. Specifically, in 1980, mortality rates due to CBVDs and IHDs are 1.8‰ and 1.0‰, respectively, and in 2012 the trend reverses, with figures of 0.4‰ and 0.5‰, respectively. Thus, although both diseases have contributed to the cardiovascular revolution, the impact of the CBVDs on survival is higher.

Figure 2. Evolution of mortality from circulatory system disease by subcauses³: Standardised death rates for men and women (1980–2012)



Source: Compiled by the authors from the HCD

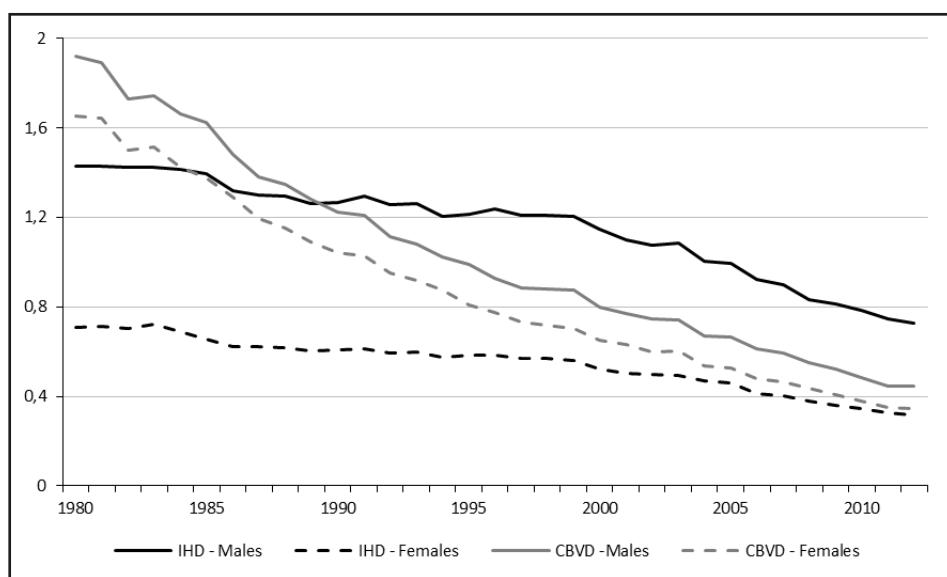
In terms of dynamics by sex, CBVDs demonstrates the most noticeable decline: in 1980 the mortality rate was 1.9‰ for men and 1.6‰ for women, and by 2012 these figures are 0.4‰ and 0.3‰, respectively. The decline is somewhat greater in women (477%) than in men (432.7%), although they evolve

³ The very low figures registered in the ‘acute rheumatic fever’ (I00–I02) and ‘other and unspecified disorders of the circulatory system’ (I95–I99) categories made it impossible to include them in the Figure.

in parallel throughout the study period (Figure 3). Cerebrovascular diseases are degenerative diseases that mainly affect the elderly population, which is why this population has benefited most from the cardiovascular revolution. In the case of IHDs, their impact differs considerably by sex. In 1980, IHDs accounted for 1.4% of deaths in men and 0.7% in women; in 2012, these figures are 0.7% and 0.3%, respectively. In addition to influencing male mortality greater than female mortality during this period, IHDs have also declined in a greater proportion for women than for men (196.3% and 223.6% in men and women, respectively).

When observing CBVD and IHD trends by sex, in 1980, CBVDs has clearly higher rates than IHD in both sexes. In men, the marked decline in CBVDs and moderate decline in IHDs means that from 1990 onwards, IHDs overtake CBVDs in mortality rates. The gap between both causes of death becomes smaller from 1999, when IHDs undergo a faster decline. In women, despite showing the same trends as men with lower rates, deaths caused by IHDs do not overtake CBVDs deaths, but gradually converge.

Figure 3. Evolution of cerebrovascular diseases and ischaemic heart diseases: standardised death rates in men and women (1980–2012)



Source: Compiled by the authors from the HCD

In this context, this analysis becomes relevant when observing the subcauses of death using the 4-digit ICD-10 code level of detail within CBVDs and IHDs to detect which disease subtypes have the greatest impact on the evolution of LE, and differential evolution. Thus, the contribution of each specific cause to

increased LE is identified, focusing in particular on changing rates during the process.

3. METHODS AND SOURCES

Data from this study are drawn from the Human Cause-of-Death Database (HCD). Following the method proposed by Jacques Vallin and France Meslé (Meslé and Vallin 1988, 1996; Pechholdová 2009), Spanish data from the Spanish Cause-of-Death Statistics [Estadística de defunciones por Causas de Muerte] of the Instituto Nacional de Estadística (INE) has been reconstructed from 1980 to 2012 (Gómez-Redondo et al. 2016; Faus-Bertomeu and Gómez-Redondo 2018) using continuous series and 4-digit ICD-10 codes as reference, thus involving the 9th revision (ICD-9) for the period 1980–1998 and the 10th revision (ICD-10) for 1999–2012. The method is based on the construction of a correspondence table to link the codes of different ICD revisions, providing a systematic comparison of the medical content, while ensuring statistical continuity and redistributing deaths proportionally by cause, to keep codes in line in each classification.

The HCD presents data at a global and cause-specific level (Gómez-Redondo et al. 2016). Thus, an in-depth analysis can be made by considering major cause-of-death groups (known as the ‘short list’), then cause-of-death subgroups (the ‘intermediate list’), and finally all codes listed in ICD-10 (the full list, at the most detailed level available). Data is presented in various formats. To conduct this study, we use raw death figures to calculate standardised death rates employing European Standard Population of 2013, we perform the decomposition of life expectancy method, and we visualise trends in CBVDs and IHDs during the period of study.

In order to perform an initial analysis of trends in Spanish mortality, with the intention of observing the contribution of the ‘five pillars of Spanish mortality’ to LE evolution in recent decades, a discrete LE decomposition analysis by age and causes of death was used (Arriaga 1984; Andreev et al. 2002; Nusselder and Loosman 2004). After decomposing LE gains, we are able to compare two sub-populations to establish the contribution of each age group and/or cause of death to the increase or decline in LE measured in fractions of year gained/lost and the proportion that these figures represent. With the aim of focusing the study on circulatory system diseases, we perform the same exercise to analyse the role played by subcauses of IHDs (codes I20–I25 in ICD-10) and of CBVDs (I60–I69) at a 4-digit level in ICD-10. Despite including all diseases classified at a 4-digit ICD-10 level in IHDs and CBVDs, we interpret only those that make a remarkable positive or negative contribution to LE. Causes that do not make a relevant contribution to LE are grouped as ‘other causes’. We consider that a cause has a significant impact when its contribution from 1980 to 2012 is higher than 0.01 years gained/lost in the case of IHDs and 0.05 in the case of CBVDs (because the latter have higher values).

The decomposition by age and causes of death is applied for the period 1980–2012. In view of the long time period analysed and the changing trends in causes of death observed (Gómez-Redondo et al. 2014), we divide the period into two stages: 1980–1996 and 1996–2012. The choice of the year 1996 as the transition year between the two subperiods is based on the fact that this year is the turning point when mortality due to IHDs overtook mortality caused by CBVDs (Faus-Bertomeu et al. 2016a). This fact is important when performing a detailed analysis of the contribution of the life years gained/lost by the CBVDs and IHDs. If the total period (1980–2012) were considered, gains and losses would be compensated. This time-based subdivision makes it possible to focus on which CBVDs subcauses contribute years of life during the first stage and which IHD subcauses contribute to LE in the second stage. In addition, the sub-periods are of equal duration.

The decomposition takes all ages into account, classified by five-year age groups, but we show only data from adult ages (from 45–50 years) to the oldest old (100 years or more), because at present, mortality for these two cause-of-death groups are concentrated mainly at adult and old ages.

4. RESULTS

The decomposition of LE gains in Spain by leading causes of death for the period 1980–2012 shows that LE at birth has increased in both sexes, specifically by 6.97 years in men and 6.55 years in women (Figure 4).

Circulatory diseases show a notable contribution to increased LE, rising by 3.26 years in men and 3.93 years in women. The next biggest contribution is seen in the decline in mortality caused by respiratory system diseases, accounting for 0.98 years and 0.85 years in men and women, respectively. Neoplasms show a trend towards stability, but despite this, they add 0.31 years and 0.46 years to LE, respectively. Other causes of death account for the remaining gain in years of life. They include endocrine system and digestive system diseases. Their contribution to LE is minimal if considered separately, but when pooled together as a single group, these diseases contribute 2.54 years in men and 1.65 years in women. All the above causes of death contribute to increased life years; mental disorders and nervous system diseases, however, reduce life expectancy by 0.12 years in men and 0.33 years in women.

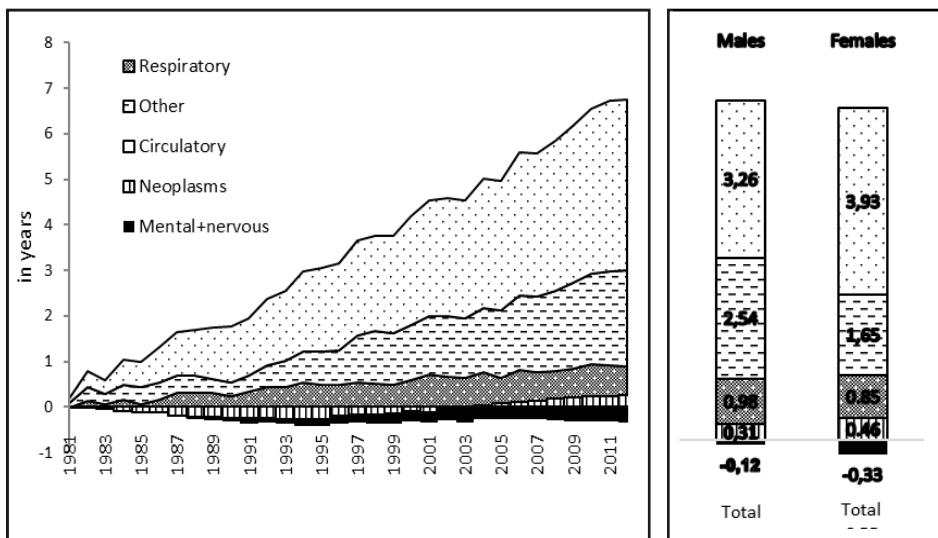


Figure 4. Contribution to change in life expectancy (years) by leading causes of death in men and women, 1980–2012

Source: Compiled by the authors from HCD

The decline in mortality from circulatory system diseases has been influenced by at least three factors. First, improvements in medical technology have opened the door to a series of new medical and surgical treatment strategies. Second, the organisation of emergency medical services has been restructured. This is a key determinant because many lives have been saved after patients had been attended immediately. And third, more programmes have been launched to prevent these diseases. The goal of prevention is to monitor certain risk factors that can be modified solely through interaction between health policies and changes in an individual's lifestyle.

The decomposition of LE gains in Spain shows that, out of 3.93 life years gained due to circulatory system diseases in women from 1980 to 2012, CBVDs and IHDs contributed 1.82 years and 0.53 years, respectively. Of 3.26 life years gained due to circulatory system disease in men, CBVDs and IHDs contributed 1.24 years and 0.91 years, respectively. Therefore, the role of CBVDs is more significant than that of IHDs for both sexes – although more so for women

than for men – and together these two subgroups account for around 60% of all deaths from circulatory system diseases in the first period, increasing this share during the second one (Table 1).

The number of years gained due to CBVDs and IHDs is not constant during the study period. For both sexes, the contribution of CBVDs is larger in the first period (1980–1996) than in the second period (1996–2012), especially for women. The trend for IHDs is reverse, with their contribution being lower in the first period than in the second, overtaking the contribution of CBVDs in the case of men.

Table 1. Increase (in fractions of year) in life expectancy. Total and major disease groups, by sex, 1980–2012*

| | Period 1 (1980- 1996) | | Period 2 (1996- 2012) | | Total period (1980- 2012) | |
|--|--------------------------|-------|--------------------------|-------|------------------------------|-------|
| | Years of live gained | | Years of live gained | | Years of live gained | |
| | Men | Women | Men | Women | Men | Women |
| Total years of live gained | 2.31 | 3.48 | 4.66 | 3.07 | 6.97 | 6.55 |
| Total years of live gained, circulatory diseases (100- 199) | 1.71 | 2.25 | 1.50 | 1.74 | 3.26 | 3.93 |
| Ischaemic heart diseases (I20-I25) | 0.26 | 0.19 | 0.65 | 0.35 | 0.91 | 0.53 |
| Acute transmural myocardial infarction of anterior wall (I21.0) | 0.01 | 0.00 | 0.02 | 0.01 | 0.03 | 0.01 |
| Acute myocardial infarction, unspecified (I21.9) | 0.29 | 0.08 | 0.58 | 0.23 | 0.87 | 0.31 |
| Acute ischaemic diseases, unspecified (I24.9) | 0.00 | 0.00 | -0.02 | -0.01 | -0.01 | 0.00 |
| Atherosclerotic cardiovascular disease, so described (I25.0) | 0.02 | 0.05 | 0.00 | 0.00 | 0.03 | 0.05 |
| Atherosclerotic cardiovascular disease (I25.1) | 0.09 | 0.16 | 0.01 | 0.05 | 0.10 | 0.22 |
| Chronic ischaemic heart disease, unspecified (I25.9) | -0.19 | -0.13 | 0.04 | 0.05 | -0.15 | -0.07 |
| Other IHD | 0.03 | 0.02 | 0.01 | 0.01 | 0.04 | 0.02 |
| Cerebrovascular diseases (I60-I69) | 0.76 | 1.12 | 0.48 | 0.70 | 1.24 | 1.82 |
| Intracerebral haemorrhage, unspecified (I61.9) | 0.10 | 0.15 | 0.09 | 0.08 | 0.19 | 0.23 |
| Cerebral infarction due to thrombosis of cerebral arteries (I63.3) | 0.14 | 0.21 | 0.02 | 0.03 | 0.15 | 0.24 |
| Cerebral infarction due to embolism of cerebral arteries (I63.4) | 0.04 | 0.07 | 0.01 | 0.01 | 0.05 | 0.08 |
| Cerebral infarction, unspecified (I63.9) | 0.29 | 0.45 | 0.08 | 0.11 | 0.37 | 0.56 |
| Stroke, not specified as haemorrhage or infarction (I64_) | 0.05 | 0.05 | 0.29 | 0.45 | 0.34 | 0.50 |
| Cerebral atherosclerosis (I67.2) | 0.13 | 0.19 | 0.01 | 0.03 | 0.14 | 0.22 |

| | | | | | | |
|--------------------------------|------|------|-------|------|------|------|
| Other CBVD | 0.01 | 0.01 | -0.01 | 0.01 | 0.00 | 0.01 |
| % ICD + CBVD among circulatory | 60% | 58% | 75% | 60% | 66% | 60% |

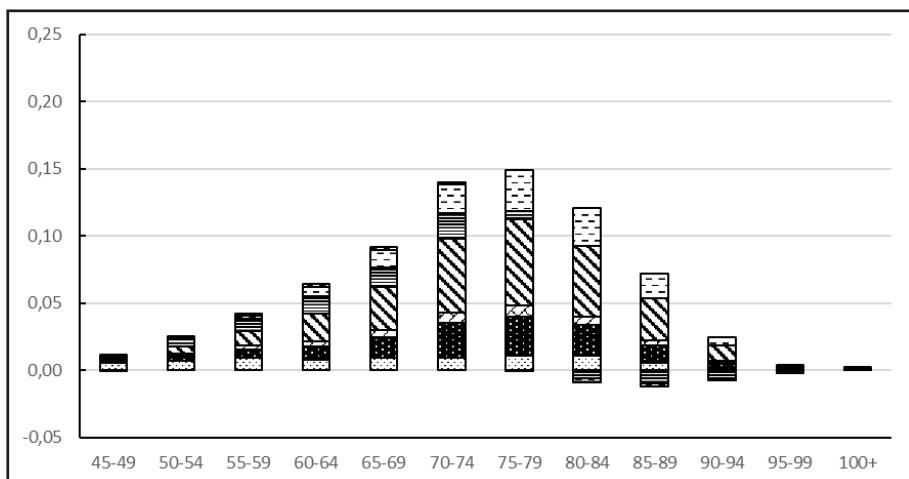
*Due to rounding, totals may not correspond to the sum of some values

Source: Authors' calculations based on the HCD

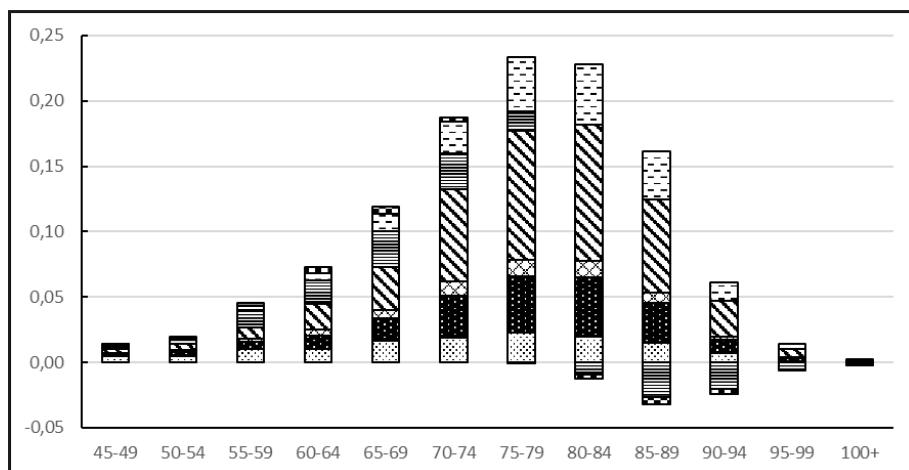
Decomposition of life expectancy by CBVD subcauses shows that life years gained in the first subperiod are due to several CBVD causes common to both sexes, but with higher values among women. A cause-of-death decomposition analysis at the ICD-10 4-digit level allows us to identify specific causes contributing to increased life expectancy (Figure 5): 'cerebral infarction, unspecified' (I63.9), 'cerebral atherosclerosis' (I67.2), and 'cerebral infarction due to thrombosis of cerebral arteries' (I63.3). Diversification of causes is reduced for the second period, in which there is a predominance of 'cerebral infarction, unspecified' and 'stroke, not specified as haemorrhage or infarction' (Figure 6). The latter is of particular note because it has a negative impact at advanced ages in the period 1980–1996 but becomes the most important life enhancer in 1996–2012. The contribution of these two specific causes account for more than 50% of the years gained by CBVDs from 1980 to 2012.

Figure 5. Contribution of cerebrovascular diseases to the change in life expectancy at the ICD-10 4-digit level (years). 1980–1996

A. Men



B. Women

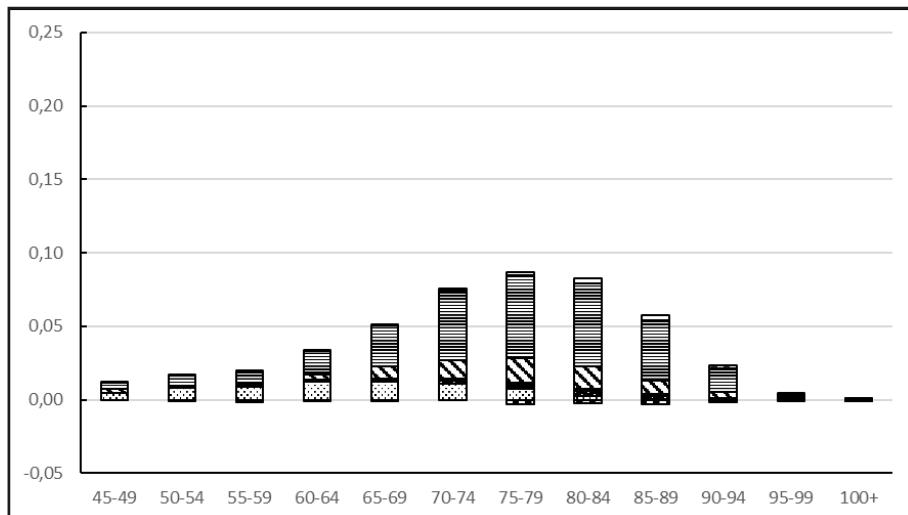


- I61.9 Intracerebral haemorrhage, unspecified
- I63.3 Cerebral infarction due to thrombosis of cerebral arteries
- I63.4 Cerebral infarction due to embolism of cerebral arteries
- I63.9 Cerebral infarction, unspecified
- I64_ Stroke, not specified as haemorrhage or infarction
- I67.2 Cerebral atherosclerosis
- Others_CBVD

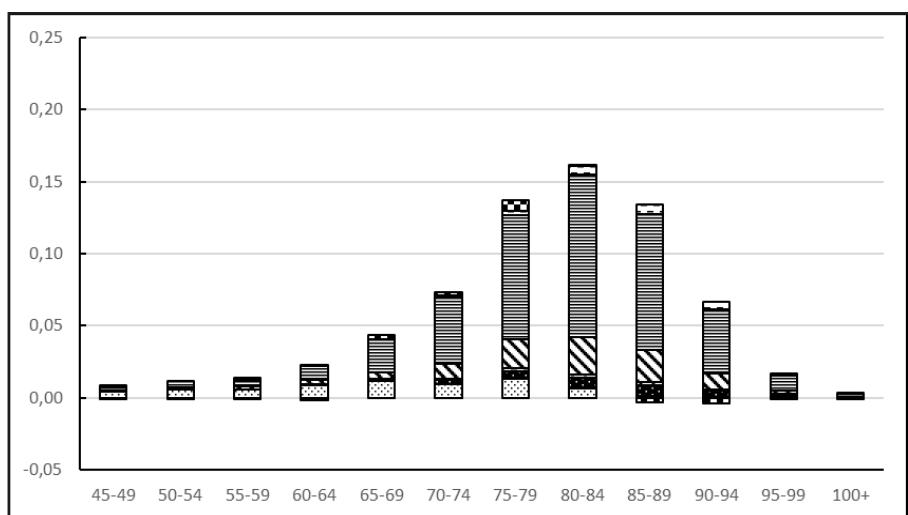
Source: Authors' calculations based on the HCD

Figure 6. Contribution of cerebrovascular diseases to the change in life expectancy at the ICD-10 4-digit level (years). 1996–2012

A. Men



B. Women

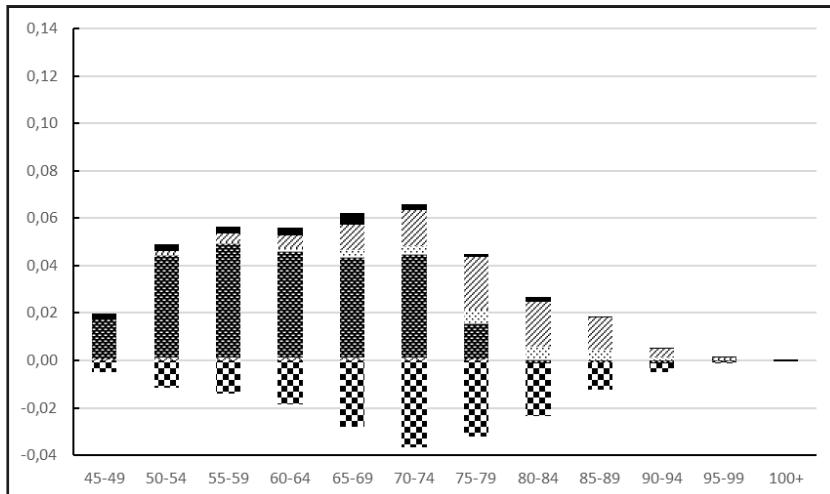


- I61.9 Intracerebral haemorrhage, unspecified
- I63.3 Cerebral infarction due to thrombosis of cerebral arteries
- I63.4 Cerebral infarction due to embolism of cerebral arteries
- I63.9 Cerebral infarction, unspecified
- I64 Stroke, not specified as haemorrhage or infarction
- I67.2 Cerebral atherosclerosis
- Others_CBVD

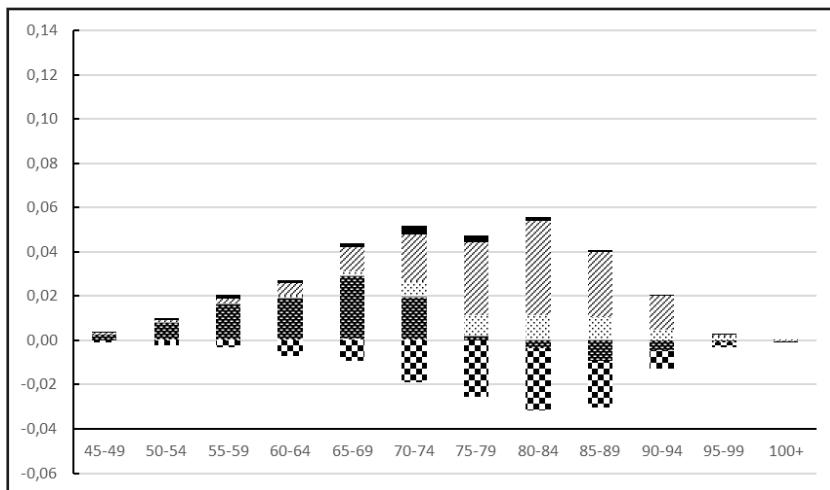
Source: Authors' calculations based on the HCD

Figure 7. Contribution of ischaemic heart diseases to the change in life expectancy at the ICD-10 4-digit level (years). 1980–1996

A. Men



B. Women

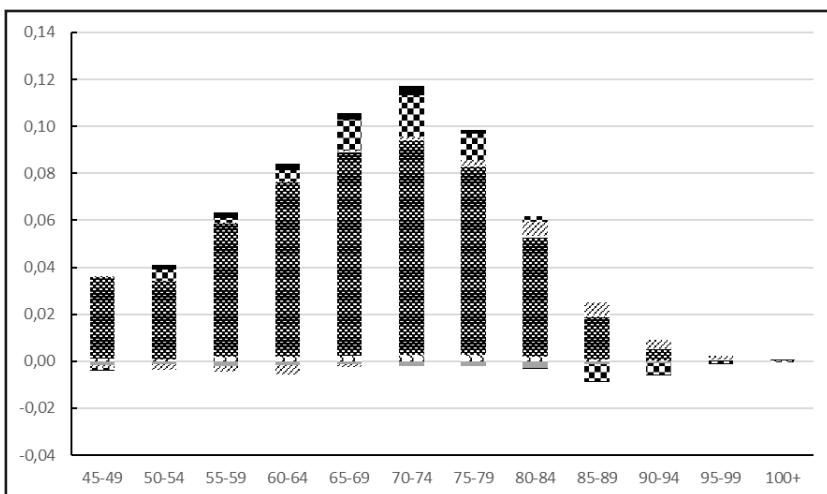


- △ I21.0 Acute transmural myocardial infarction of anterior wall
- I24.9 Acute ischaemic diseases, unspecified
- ▨ I25.1 Atherosclerotic cardiovascular disease
- Others IHD
- ☒ I21.9 Acute myocardial infarction, unspecified
- ▢ I25.0 Atherosclerotic cardiovascular disease, so described
- ▢ I25.9 Chronic ischaemic heart disease, unspecified

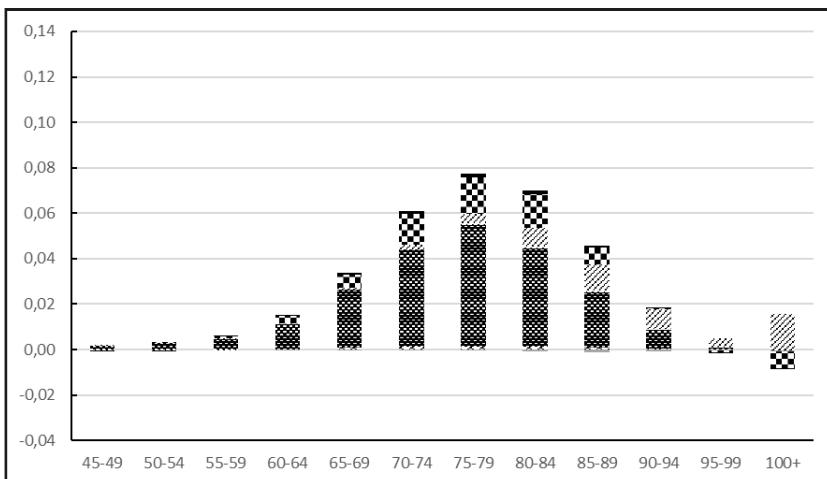
Source: Authors' calculations based on the HCD

Figure 8. Contribution of ischaemic heart diseases to change in life expectancy at ICD-10 4-digit level (years). 1996–2012

A. Men



B. Women



- △ I21.0 Acute transmural myocardial infarction of anterior wall ■ I21.9 Acute myocardial infarction, unspecified
- I24.9 Acute ischaemic diseases, unspecified ▲ I25.0 Atherosclerotic cardiovascular disease, so described
- I25.1 Atherosclerotic cardiovascular disease ○ I25.9 Chronic ischaemic heart disease, unspecified
- Others IHD

Source: Authors' calculations based on HCD

Analysis of the contribution of IHDs reveals a male advantage, especially in the second period. In the first period, years gained are due to several subcauses – mainly ‘acute myocardial infarction’ (I21.9), ‘atherosclerotic heart disease’ (I25.1), and ‘atherosclerotic cardiovascular disease’ (I25.0) (Figure 7). In the second period for both sexes (Figure 8), the highest contributions are due to ‘acute myocardial infarction, unspecified’ (I21.9) and ‘chronic ischaemic heart disease’ (I25.9), which was previously the main obstacle among circulatory diseases to an increase in LE. ‘Atherosclerotic heart disease’ (I25.1) also contributed significantly, especially among women older than 80 years.

Trends differ by age and sex. Years of life gained from CBVD subcauses of death mainly occur at advanced ages, especially in the group aged 70–79 years. Among women, the focal point is located between 75 and 84 years, concentrating more than half of CBVD contributions. In the first period, ‘strokes’ (I64_) between 80 and 94 years became an obstacle to LE growth in both sexes (Figure 5), but in the second period, this cause was the strongest factor for increased LE among all CBVDs, with a major contribution in the group aged 75–84 years and, to a lesser extent, in the neighbouring groups (Figure 6).

In the case of IHDs, the improvements affect younger groups, especially among men. However, clear divergences exist between sexes in the two subperiods. In men, the gain from IHDs is distributed relatively evenly, initially between the ages of 50 and 74 years (Figure 7) and later between 65 and 79 years (Figure 8). The same shift towards advanced ages observed in men is also observed in women during the second period, although to a lesser extent and with onset in older age groups. The 4-digit level disaggregated analysis detects causes with different trends by sex and age group. From 1980 to 1996, ‘chronic ischaemic heart disease’ (I25.9) contributed negatively, slowing down LE growth, and this was most notable at the critical ages for improved mortality, which are from 65 to 84 years in men and from 70 to 89 years in women. In addition, ‘acute myocardial infarction’ (I21.9) plays a leading role in the second period but contributes inconsistently during the first period. Thus, it has irrefutable weight until the age of 74 years, but then makes a decreasing contribution to increased LE, actually reducing life years among the oldest women, and is overtaken by ‘atherosclerotic heart disease’ (I25.0).

5. DISCUSSION

The declining trend shown by IHDs and CBVDs has become a determining factor in increased longevity in recent decades (Boix-Martínez et al., 2003; Gómez-Redondo and Boe 2005; Medrano et al. 2006; Pérez-Moreda et al., 2015; Faus-Bertomeu et al. 2016a, García-González and Grande 2018a). However, analysis at the maximum level of decomposition shows that not all causes contribute equally throughout the process and also reveals changes in trends among specific causes. We would like to highlight the following findings:

1. Unequal contribution by period. In most cases, the subcauses making the largest contribution in the first period (1980–1996) (e.g., Figure 5, ‘Cerebral infarction’, I63.3, and ‘Cerebral infarction, unspecified’, I63.9) becomes less important in the second period (1996–2012), leading to changing trends in these specific subcauses (Figure 6). These changes would have gone undetected if the subcauses had been grouped together.
2. Concentration of gains. During the second period, a single subcause ('Stroke, not specified as haemorrhage or infarction') accounts for more than 60% of the contribution of CBVDs to the change in LE (Figure 6). Likewise, in the same period, a single subcause ('Acute myocardial infarction, unspecified') accounts for more than 70% of the contribution of IHDs to the change in LE (Figure 8). However, the first period shows wider diversity in terms of subcauses (Figures 5 and 7).
3. Disappearance of negative contributions. While the first period shows decreased LE for certain subcauses (I64_-, I25.9) and certain ages (Figures 5 and 7), the second period shows an increased contribution to LE by almost all subcauses, except for a very few exceptions, which have an insignificant weight (Figures 6 and 8).
4. Importance of ‘unspecified’ codes. Mortality from these causes is concentrated at older ages, often in patients with diverse interrelated comorbidities, hindering diagnosis at the maximum level of decomposition provided by the ICD. For this reason, ‘unspecified’ codes (ending in ‘9’) play a significant role because —among other reasons— they include patients in whom disease aetiology cannot be determined more precisely, especially in the presence of interrelated causes (Arboix et al. 2008), without employing considerably invasive diagnosis methods, neither a post-mortem necropsy.
5. Contributions LE by sex and age. Traditionally, women have contributed most to longevity. However, men now appear to be gaining ground due to the increase in life years gained. In addition, other significant divergences are observed. First, the influence of CBVDs continues to be higher in women than in men (Table 1). Second, although the contribution of most of these causes of death to LE occurs at older ages, in women this is observed at more advanced ages than in men. Third, in terms of contributions by specific causes, LE increases in women mainly as a result of declining mortality from some specific CBVDs, while in men, IHDs subcauses play a more decisive role (Medrano et al. 2006). In summary, CBVDs affect advanced ages (Figures 5 and 6) and IHDs show a higher predominance in men at adult ages (Figures 7 and 8).

The most marked reductions in CBVDs in mortality take place at the end of the study period (Cayuela et al. 2016), but the contribution of these diseases to the increase in LE is greatest from 1980 to 1996. This phenomenon occurs

because, although the reduction in mortality is lower in the first period, its impact is strongest in younger individuals in the old age group, leading to a larger indirect contribution to increased LE as a result of the improved survival, and therefore to these individuals having more years of life ahead of them.

The IHD group coincides with the CBVD group with regard to factors (e.g., changing therapies, lifestyle) that account for the decline in mortality, but also shows some differences. In the IHD group, a single code ('Acute myocardial infarction, unspecified', I21.9) accounts for a large proportion of mortality throughout the period studied, especially among men. Although some specific causes ('Atherosclerotic Heart Disease', I25.1) makes a positive contribution and others ('Chronic Ischaemic Heart Disease, unspecified', I25.9) make a negative contribution from 1980 to 1996, the overall trend and impact of the IHD group are due to myocardial infarction, as corroborated by other studies that analysed subcauses at higher ICD levels than this study.

From a medical point of view, the leading factors influencing the decline in mortality among IHDs and CBVDs alike are changes in cardiovascular risk factors (e.g., hypertension, sedentary lifestyle, smoking, hypercholesterolaemia), advances in medical therapies (particularly secondary prevention) and surgical interventions (e.g., bypass surgery, coronary angioplasty), and the implementation of national and international joint action protocols and guidelines (e.g., 'Code Stroke') (Banegas et al. 2006; Palomeras and Casado 2010; Flores-Mateo et al. 2011; Faus-Bertomeu et al. 2016b; Félix-Redondo et al. 2013; Grau et al. 2011; Jiménez-Fábrega et al. 2011; Higueras-Fresnillo et al., 2018). Although some differences have been found in the influence of these risk factors on IHDs and CBVDs and their subtypes, the risk factors are common to all diseases analysed while having a differential impact on each disease (Arboix et al. 2008; Palomeras and Casado 2010).

In this sense, three factors are determinant for the reduction of mortality from CBVDs. First, the role of the Spanish Health Care System. In the last decades of the 20th century and the early years of this century, the specific health programmes have been implemented to address and reduce such mortality. These programs envisaged improvements in detection and diagnosing correct disease, and also appropriate and timely intervention and treatment, such as the use of anticoagulants and coronary bypass surgery (Cayuela et al. 2016). In addition, health services have been coordinated at different levels. In primary care, prevention programs, as well as greater control of risk factors among the population have been introduced. In addition, improvements in the evaluation and treatment of patients stand out in hospital care and emergencies (Spanish Ministry of Health and Social Policy 2009). As a consequence of these measures, mortality from 'cerebral infarction due to thrombosis' (I63.3) has especially decreased revealing treatments with great effectiveness - when it is applied before three hours after the first symptoms were observed. This is also the case of 'stroke' (I64) and 'cerebral infarction unspecified' (I63.9), for which specific programs have been developed – such as the 'Teleictus' (prehospital telephone service) and the 'Code Stroke' (in the emergency department) from 1997– that have ac-

celerated the diagnosis, care and treatment (Jiménez-Fábrega et al. 2011), decreasing their mortality rate. The ‘cerebral infarction unspecified’ (I63.9) is a type of infarction in which several probable etiologies are identified without being possible to determine the cause. The decrease of this cause of death is a clear example of the improvement in the diagnosis of cerebrovascular diseases (Palomeras and Casado 2010). Second, with regard to social and behavioural changes, the promotion of healthy lifestyles together with the reduction of unhealthy habits such as smoking and alcohol consumption (Grau et al. 2011), along with an increase in physical exercise and greater awareness (Higueras-Fresnillo et al., 2018), have influenced the decline of CBVDs. And thirdly, the greater population awareness about the risk factors for its prevention, as well as the symptoms of these diseases to act quickly avoiding death. An example of this is the decrease in the mortality from ‘cerebral atherosclerosis’ (I67.2), which greatly depends on consumption of saturated fats and cholesterol, smoking and a sedentary lifestyle. However, since mortality from ‘stroke’ (I64) and ‘cerebral infarction, unspecified’ (I63.9) continue to account for considerable share in mortality in the Spanish population, the implementation of larger health prevention measures and applied research (Banegas et al. 2006) would further reduce their impact.

The understanding of IHD pathophysiology has progressed significantly in recent years. As a result of this greater medical knowledge specific action protocols have been implemented (Félix-Redondo et al. 2013), and consequently mortality from IHD causes has decreased (Faus-Bertomeu et al. 2016b, García-González and Grande 2018b). A good example is the drastic reduction in mortality from 1996 to 2012 caused by indeterminate IHD as ‘acute myocardial infarction, unspecified’ (I21.9) and ‘chronic ischaemic heart disease, unspecified’ (I25.9), especially in men. The decrease in IHD is also associated with the use of specific clinical guidelines aimed at health professionals that influence on the knowledge of risk factors and healthcare, as the Spanish adaptation of the “European Guidelines on cardiovascular disease prevention in clinical practice”, with five updates since 1994 (Perk et al. 2012). For example, the 2008 report cited the priority to the prevention of atherosclerotic diseases through the detection of non-modifiable risk factors and the incidence of modifiable factors (Lobos et al. 2008), a reason that would explain the lower impact of specific causes of death such as ‘Atherosclerotic cardiovascular disease’ (I25.0) and ‘Atherosclerotic heart disease’ (I25.1). Therefore, many deaths that continue to occur could be avoided with the implementation of health prevention policies that would have an impact on acute diseases (Palomeras and Casado 2010).

Another element that leads to think about the possible decrease in mortality due to circulatory system diseases when comparing two subgroups of the CBVDs and the IHDs is the impact that they have on the different age groups. On one hand, while CBVDs affect advanced ages, IHDs show a higher predominance in men at adult ages and at less concentrated ages than in women. And on the other hand, the contribution of CBVDs declines after the mid-1990s when healthy programs were developed; and IHDs show an opposite trend, with higher values in the second period, overtaking the contribution of CBVDs among

men. It suggests that prevention programmes that affect modifiable risk factors, as well as improvements in prediction and treatment, could continue to reduce mortality from these subcauses of mortality, and consequently increase the contribution of IHDs to the overall increase in LE. Considering that the results of the health programs are observed in medium and long term, and therefore the young generations will be the beneficiaries, it would be expected that the implementation of this type of sanitary measures in the population, firstly, would decrease the mortality caused by IHDs at younger age-groups; and secondly, would displace deaths at advanced ages, as it has happened with the CBVDs.

6. CONCLUSION

In summary, the novelty of this study lies in its identification of the contribution of the main subcauses of death from circulatory system diseases to the change in LE and, thereby, to the ongoing Cardiovascular Revolution. The decomposition analysis at the very detailed level allowed us to identify which specific causes of death have stopped provoking deaths and which ones continue accumulating them, and therefore the Spanish Health System should act accordingly. The improvement of the diagnostics of these diseases has led to the reduction of deaths classified as unspecified (such as I63.9, I21.9 and I25.9). At the same time, health innovations have decreased and transferred the mortality due to non-modifiable factors (for example I63.3, I64, I25.0 and I25.1) to advanced ages. However, diseases defined as acute, mainly classified in IHDs (e.g., I21.0), continue to concentrate deaths that could be avoided with the incidence of modifiable factors as it has occurred with other subcauses of the CBVDs (e.g., I67.2) with implication for future generations.

On-going scientific advances leading to increasingly precise diagnoses and characterizing the scientific, demographic, and social context of our society must be matched by healthy programmes and policies which reduced mortality by CBVDs and IHDs at elderly population. Therefore, the knowledge gained from observing the diverse trends and changes in specific subcauses of death with a positive or negative influence on survival in Spain sheds new light on the knowledge that has been available to date on the period studied. Our new knowledge of the specific impact of sex and age on the contributions of specific subcauses to LE will allow the design of more effective and practical prevention, planning, and intervention strategies.

It is essential to understand the epidemiologic profile of the present elderly population in order to estimate future LE evolution. Our results show the existence of a double process that CBVDs and IHDs have undergone along different timelines. Despite differences by sex and age, life years are mainly gained from CBVDs, especially in the first period, and IHDs show an opposite trend and a major influence in adult people. Our analysis of the main 4-digit level subcauses in these two large groups reveals the specific causes which could increase LE in a near future, specifically reveals the mortality due to modifiable factors mainly

classified in IHDs. In addition, this knowledge would also allow a more precise identification of the specific causes of cardiovascular death on which the health system should influence to increase life expectancy in Spain. Nevertheless, and despite the improvements, we have detected that the compression of mortality towards more advanced ages results in a concentration for some ‘unspecific’ codes (finishing in ‘9’). This finding highlights the need for improving recognition and description of each disease subtypes on death certificates, thus demanding a higher diagnosis precision in order to take advantage of the fourth-digit codification allowed by ICD-10. This improvement at the origin of the data sources would undoubtedly lead to a better in depth understanding of health trends, thus allowing to plan more appropriate public policies for ageing societies.

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Appendix A

A.1. Evolution of the main causes of death, standardised rates (x1000)

| year | Other | Neoplasms | Mental disorders | Nervous system | Circulatory system | Respiratory system |
|------|-------|-----------|------------------|----------------|--------------------|--------------------|
| 1980 | 1.778 | 1.916 | 0.026 | 0.120 | 4.659 | 1.215 |
| 1981 | 1.739 | 1.909 | 0.028 | 0.112 | 4.562 | 1.261 |
| 1982 | 1.673 | 1.924 | 0.026 | 0.111 | 4.284 | 1.093 |
| 1983 | 1.714 | 1.943 | 0.041 | 0.115 | 4.379 | 1.201 |
| 1984 | 1.721 | 1.994 | 0.047 | 0.108 | 4.105 | 1.076 |
| 1985 | 1.745 | 2.019 | 0.054 | 0.114 | 4.114 | 1.180 |
| 1986 | 1.735 | 2.028 | 0.063 | 0.115 | 3.842 | 1.095 |
| 1987 | 1.744 | 2.064 | 0.074 | 0.125 | 3.656 | 0.942 |
| 1988 | 1.758 | 2.088 | 0.082 | 0.134 | 3.605 | 0.960 |
| 1989 | 1.791 | 2.086 | 0.099 | 0.139 | 3.487 | 0.954 |
| 1990 | 1.786 | 2.096 | 0.118 | 0.148 | 3.386 | 1.026 |
| 1991 | 1.759 | 2.099 | 0.135 | 0.161 | 3.373 | 0.937 |
| 1992 | 1.683 | 2.099 | 0.137 | 0.155 | 3.175 | 0.845 |
| 1993 | 1.648 | 2.123 | 0.148 | 0.163 | 3.121 | 0.867 |
| 1994 | 1.606 | 2.131 | 0.166 | 0.171 | 2.951 | 0.794 |
| 1995 | 1.606 | 2.128 | 0.178 | 0.175 | 2.872 | 0.828 |
| 1996 | 1.586 | 2.079 | 0.187 | 0.181 | 2.815 | 0.852 |
| 1997 | 1.486 | 2.074 | 0.193 | 0.184 | 2.708 | 0.811 |
| 1998 | 1.476 | 2.064 | 0.208 | 0.197 | 2.695 | 0.850 |
| 1999 | 1.457 | 2.075 | 0.225 | 0.216 | 2.659 | 0.911 |
| 2000 | 1.412 | 2.046 | 0.211 | 0.213 | 2.480 | 0.805 |
| 2001 | 1.384 | 2.059 | 0.208 | 0.230 | 2.380 | 0.711 |
| 2002 | 1.377 | 2.015 | 0.207 | 0.238 | 2.338 | 0.766 |
| 2003 | 1.405 | 2.012 | 0.225 | 0.273 | 2.347 | 0.786 |
| 2004 | 1.358 | 1.981 | 0.199 | 0.253 | 2.183 | 0.684 |
| 2005 | 1.356 | 1.935 | 0.203 | 0.272 | 2.174 | 0.798 |
| 2006 | 1.267 | 1.915 | 0.188 | 0.263 | 2.002 | 0.647 |
| 2007 | 1.265 | 1.902 | 0.183 | 0.271 | 1.985 | 0.697 |
| 2008 | 1.220 | 1.868 | 0.186 | 0.273 | 1.895 | 0.673 |
| 2009 | 1.167 | 1.855 | 0.197 | 0.282 | 1.794 | 0.639 |
| 2010 | 1.106 | 1.852 | 0.193 | 0.279 | 1.712 | 0.571 |

| year | Other | Neoplasms | Mental disorders | Nervous system | Circulatory system | Respiratory system |
|------|-------|-----------|------------------|----------------|--------------------|--------------------|
| 2011 | 1.090 | 1.844 | 0.189 | 0.284 | 1.639 | 0.579 |
| 2012 | 1.075 | 1.831 | 0.205 | 0.297 | 1.633 | 0.622 |

Source: Compiled by the authors from HCD data

A.2 Evolution of mortality from circulatory system disease by subcauses: Standardised death rates (x1000), both sexes (1980–2012)

| | (I00-I02) | (I05-I09) | (I10-I15) | (I20-I25) | (I26-I28) | (I30-I52) | (I60-I69) | (I70-I79) | (I80-I89) | (I95-I99) |
|------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| 1980 | 0.0012 | 0.0849 | 0.0716 | 1.0195 | 0.2166 | 0.8801 | 1.7655 | 0.5777 | 0.0199 | 0.0190 |
| 1981 | 0.0004 | 0.0742 | 0.0759 | 1.0213 | 0.2111 | 0.8540 | 1.7541 | 0.5295 | 0.0201 | 0.0188 |
| 1982 | 0.0003 | 0.0652 | 0.0729 | 1.0123 | 0.1989 | 0.7955 | 1.6021 | 0.5003 | 0.0187 | 0.0158 |
| 1983 | 0.0005 | 0.0606 | 0.0826 | 1.0241 | 0.1962 | 0.8451 | 1.6159 | 0.5164 | 0.0192 | 0.0159 |
| 1984 | 0.0004 | 0.0597 | 0.0850 | 1.0033 | 0.1589 | 0.8090 | 1.5292 | 0.4193 | 0.0224 | 0.0144 |
| 1985 | 0.0006 | 0.0468 | 0.0830 | 0.9744 | 0.1543 | 0.8983 | 1.4831 | 0.4274 | 0.0278 | 0.0157 |
| 1986 | 0.0003 | 0.0433 | 0.0852 | 0.9238 | 0.1381 | 0.8657 | 1.3765 | 0.3706 | 0.0238 | 0.0129 |
| 1987 | 0.0005 | 0.0427 | 0.0866 | 0.9170 | 0.1207 | 0.8084 | 1.2778 | 0.3650 | 0.0230 | 0.0127 |
| 1988 | 0.0002 | 0.0408 | 0.0876 | 0.9129 | 0.1132 | 0.8219 | 1.2371 | 0.3524 | 0.0239 | 0.0136 |
| 1989 | 0.0002 | 0.0425 | 0.0896 | 0.8870 | 0.1021 | 0.8224 | 1.1736 | 0.3322 | 0.0218 | 0.0125 |
| 1990 | 0.0002 | 0.0413 | 0.0910 | 0.8932 | 0.1008 | 0.7939 | 1.1204 | 0.3091 | 0.0224 | 0.0124 |
| 1991 | 0.0001 | 0.0413 | 0.0982 | 0.9079 | 0.1007 | 0.7936 | 1.1085 | 0.2891 | 0.0208 | 0.0108 |
| 1992 | 0.0003 | 0.0373 | 0.0963 | 0.8822 | 0.0876 | 0.7533 | 1.0269 | 0.2610 | 0.0184 | 0.0096 |
| 1993 | 0.0003 | 0.0376 | 0.0931 | 0.8870 | 0.0836 | 0.7622 | 0.9896 | 0.2387 | 0.0171 | 0.0086 |
| 1994 | 0.0003 | 0.0365 | 0.0984 | 0.8499 | 0.0748 | 0.7091 | 0.9439 | 0.2120 | 0.0159 | 0.0081 |
| 1995 | 0.0002 | 0.0369 | 0.1005 | 0.8585 | 0.0733 | 0.6957 | 0.8909 | 0.1900 | 0.0165 | 0.0073 |
| 1996 | 0.0002 | 0.0365 | 0.0975 | 0.8682 | 0.0760 | 0.6926 | 0.8461 | 0.1751 | 0.0151 | 0.0063 |
| 1997 | 0.0003 | 0.0376 | 0.0925 | 0.8473 | 0.0728 | 0.6695 | 0.8029 | 0.1618 | 0.0146 | 0.0062 |
| 1998 | 0.0000 | 0.0376 | 0.0980 | 0.8460 | 0.0696 | 0.6739 | 0.7888 | 0.1586 | 0.0140 | 0.0054 |
| 1999 | 0.0002 | 0.0380 | 0.0978 | 0.8372 | 0.0701 | 0.6578 | 0.7788 | 0.1564 | 0.0150 | 0.0056 |
| 2000 | 0.0002 | 0.0373 | 0.0980 | 0.7927 | 0.0591 | 0.6082 | 0.7175 | 0.1454 | 0.0126 | 0.0060 |
| 2001 | 0.0001 | 0.0342 | 0.0978 | 0.7605 | 0.0563 | 0.5849 | 0.6946 | 0.1355 | 0.0120 | 0.0023 |
| 2002 | 0.0001 | 0.0328 | 0.1004 | 0.7504 | 0.0570 | 0.5860 | 0.6638 | 0.1324 | 0.0118 | 0.0019 |
| 2003 | 0.0002 | 0.0343 | 0.1099 | 0.7502 | 0.0578 | 0.5922 | 0.6658 | 0.1239 | 0.0110 | 0.0021 |
| 2004 | 0.0001 | 0.0290 | 0.1063 | 0.7024 | 0.0489 | 0.5714 | 0.5984 | 0.1125 | 0.0107 | 0.0014 |
| 2005 | 0.0001 | 0.0293 | 0.1103 | 0.6940 | 0.0498 | 0.5780 | 0.5899 | 0.1084 | 0.0112 | 0.0019 |
| 2006 | 0.0000 | 0.0239 | 0.1065 | 0.6377 | 0.0447 | 0.5374 | 0.5393 | 0.1005 | 0.0092 | 0.0012 |
| 2007 | 0.0001 | 0.0237 | 0.1142 | 0.6205 | 0.0464 | 0.5506 | 0.5230 | 0.0953 | 0.0082 | 0.0017 |
| 2008 | 0.0002 | 0.0226 | 0.1119 | 0.5790 | 0.0444 | 0.5453 | 0.4880 | 0.0920 | 0.0090 | 0.0016 |
| 2009 | 0.0001 | 0.0284 | 0.1155 | 0.5590 | 0.0401 | 0.4949 | 0.4601 | 0.0846 | 0.0088 | 0.0009 |
| 2010 | 0.0000 | 0.0283 | 0.1264 | 0.5375 | 0.0340 | 0.4703 | 0.4276 | 0.0761 | 0.0082 | 0.0009 |
| 2011 | 0.0001 | 0.0257 | 0.1235 | 0.5116 | 0.0334 | 0.4670 | 0.3956 | 0.0722 | 0.0078 | 0.0007 |
| 2012 | 0.0001 | 0.0229 | 0.1261 | 0.4973 | 0.0326 | 0.4804 | 0.3916 | 0.0729 | 0.0073 | 0.0009 |

Source: Compiled by the authors from HCD data

- (I00-I02): Acute rheumatic fever
 (I05- I09): Chronic rheumatic heart diseases
 (I10- I15): Hypertensive diseases
 (I20-I25): Ischaemic heart diseases
 (I26-I28): Pulmonary heart disease
 (I30- I52): Other forms of heart disease
 (I60- I69): Cerebrovascular diseases
 (I70-I79): Diseases of arteries, arterioles and capillaries
 (I80-I89): Diseases of veins, lymphatic vessels and lymph nodes
 (I95-I99): Other and unspecified disorders of the circulatory system

A.3. Evolution of cerebrovascular diseases and ischaemic heart diseases: standardised death rates (x1000) in men and women (1980–2012)

| | CBVD (I60-I69) | | IHD (I30-I52) | |
|-------------|-----------------------|--------------|----------------------|--------------|
| | Men | Women | Men | Women |
| 1980 | 1.9187 | 1.6544 | 1.4303 | 0.7086 |
| 1981 | 1.8934 | 1.6440 | 1.4291 | 0.7122 |
| 1982 | 1.7313 | 1.5026 | 1.4221 | 0.7035 |
| 1983 | 1.7466 | 1.5154 | 1.4222 | 0.7220 |
| 1984 | 1.6641 | 1.4264 | 1.4171 | 0.6912 |
| 1985 | 1.6235 | 1.3763 | 1.3955 | 0.6565 |
| 1986 | 1.4818 | 1.2929 | 1.3186 | 0.6222 |
| 1987 | 1.3793 | 1.1965 | 1.2990 | 0.6243 |
| 1988 | 1.3457 | 1.1506 | 1.2956 | 0.6194 |
| 1989 | 1.2789 | 1.0891 | 1.2616 | 0.6038 |
| 1990 | 1.2218 | 1.0407 | 1.2662 | 0.6103 |
| 1991 | 1.2119 | 1.0273 | 1.2931 | 0.6143 |
| 1992 | 1.1142 | 0.9532 | 1.2564 | 0.5959 |
| 1993 | 1.0786 | 0.9167 | 1.2599 | 0.6008 |
| 1994 | 1.0224 | 0.8770 | 1.2068 | 0.5768 |
| 1995 | 0.9921 | 0.8091 | 1.2165 | 0.5855 |
| 1996 | 0.9286 | 0.7766 | 1.2377 | 0.5825 |
| 1997 | 0.8851 | 0.7342 | 1.2107 | 0.5703 |
| 1998 | 0.8784 | 0.7163 | 1.2096 | 0.5679 |

| | CBVD (I60-I69) | | IHD (I30-I52) | |
|-------------|-----------------------|--------|----------------------|--------|
| 1999 | 0.8737 | 0.7023 | 1.2030 | 0.5579 |
| 2000 | 0.7975 | 0.6519 | 1.1454 | 0.5224 |
| 2001 | 0.7699 | 0.6308 | 1.1011 | 0.5010 |
| 2002 | 0.7454 | 0.5964 | 1.0761 | 0.4990 |
| 2003 | 0.7399 | 0.6025 | 1.0831 | 0.4957 |
| 2004 | 0.6682 | 0.5381 | 1.0061 | 0.4686 |
| 2005 | 0.6666 | 0.5265 | 0.9963 | 0.4596 |
| 2006 | 0.6105 | 0.4803 | 0.9238 | 0.4141 |
| 2007 | 0.5955 | 0.4632 | 0.8994 | 0.4038 |
| 2008 | 0.5504 | 0.4341 | 0.8335 | 0.3796 |
| 2009 | 0.5232 | 0.4072 | 0.8147 | 0.3579 |
| 2010 | 0.4825 | 0.3805 | 0.7860 | 0.3434 |
| 2011 | 0.4474 | 0.3519 | 0.7484 | 0.3261 |
| 2012 | 0.4433 | 0.3468 | 0.7285 | 0.3168 |

Source: Compiled by the authors from HCD data

A.4. Contribution to change in life expectancy (years) by leading causes of death in men and women, 1980–2012

| | Neoplasms | Mental+nervous | Respiratory | Circulatory | Other diseases |
|-------------|------------------|-----------------------|--------------------|--------------------|-----------------------|
| 1980 | 0.000 | 0.000 | 0.000 | 0.000 | 0.000 |
| 1981 | -0.003 | 0.011 | -0.007 | 0.095 | 0.087 |
| 1982 | -0.015 | 0.023 | 0.137 | 0.364 | 0.292 |
| 1983 | -0.032 | -0.001 | 0.048 | 0.318 | 0.233 |
| 1984 | -0.097 | 0.005 | 0.170 | 0.544 | 0.329 |
| 1985 | -0.116 | 0.000 | 0.072 | 0.542 | 0.375 |
| 1986 | -0.125 | -0.011 | 0.149 | 0.780 | 0.391 |
| 1987 | -0.186 | -0.027 | 0.307 | 0.946 | 0.387 |
| 1988 | -0.228 | -0.045 | 0.299 | 1.003 | 0.390 |
| 1989 | -0.225 | -0.058 | 0.317 | 1.135 | 0.285 |
| 1990 | -0.241 | -0.082 | 0.246 | 1.235 | 0.296 |
| 1991 | -0.242 | -0.115 | 0.343 | 1.247 | 0.354 |
| 1992 | -0.240 | -0.097 | 0.437 | 1.478 | 0.468 |
| 1993 | -0.259 | -0.118 | 0.429 | 1.544 | 0.588 |
| 1994 | -0.273 | -0.139 | 0.531 | 1.755 | 0.689 |

| | Neoplasms | Mental+nervous | Respiratory | Circulatory | Other diseases |
|-------------|------------------|-----------------------|--------------------|--------------------|-----------------------|
| 1995 | -0.259 | -0.152 | 0.499 | 1.839 | 0.719 |
| 1996 | -0.185 | -0.171 | 0.483 | 1.907 | 0.761 |
| 1997 | -0.175 | -0.172 | 0.539 | 2.068 | 1.039 |
| 1998 | -0.158 | -0.199 | 0.515 | 2.086 | 1.161 |
| 1999 | -0.139 | -0.229 | 0.478 | 2.154 | 1.134 |
| 2000 | -0.097 | -0.211 | 0.598 | 2.378 | 1.198 |
| 2001 | -0.108 | -0.233 | 0.705 | 2.532 | 1.303 |
| 2002 | -0.050 | -0.240 | 0.653 | 2.587 | 1.341 |
| 2003 | -0.038 | -0.298 | 0.636 | 2.605 | 1.307 |
| 2004 | 0.012 | -0.255 | 0.760 | 2.846 | 1.419 |
| 2005 | 0.082 | -0.277 | 0.643 | 2.855 | 1.472 |
| 2006 | 0.109 | -0.268 | 0.813 | 3.127 | 1.647 |
| 2007 | 0.138 | -0.266 | 0.753 | 3.146 | 1.665 |
| 2008 | 0.185 | -0.283 | 0.792 | 3.289 | 1.765 |
| 2009 | 0.214 | -0.312 | 0.833 | 3.457 | 1.893 |
| 2010 | 0.230 | -0.310 | 0.932 | 3.615 | 2.003 |
| 2011 | 0.243 | -0.321 | 0.926 | 3.738 | 2.058 |
| 2012 | 0.259 | -0.348 | 0.885 | 3.745 | 2.124 |

Source: Authors' calculations based on HCD

A.5 Contribution of cerebrovascular diseases to LE growth (in hundredths of a year). Men, 1980-2012Source: Authors' calculations based on HCD

| | 1980-1996 | | | | | | | | | | 1996-2012 | | | | | | | | |
|-------|-------------------------------|--------------------|--------------------|--------------------|--------------------|------------------|--------------------|--------|----------|------|-----------|-------|-------|-------|------------------|-------|--------|----------|--|
| | MEN. Cerebrovascular diseases | | | | | | | | | | | | | | | | | | |
| | Age | 161.9 ¹ | 163.3 ² | 163.4 ³ | 163.9 ⁴ | 164 ⁵ | 167.2 ⁶ | Others | All CBVD | Age | 161.9 | 163.4 | 163.3 | 163.9 | 164 ₋ | 167.2 | Others | All CBVD | |
| 0-44 | 0.44 | 2.28 | 0.33 | 0.18 | 0.34 | 0.69 | 0.08 | 0.58 | 4.48 | 0.44 | 1.85 | 0.05 | 0.03 | 0.23 | 0.38 | 0.03 | 0.60 | 3.17 | |
| 45-49 | 0.55 | 0.16 | 0.08 | 0.15 | 0.18 | 0.05 | -0.01 | 1.16 | 45.49 | 0.48 | 0.01 | 0.00 | 0.24 | 0.41 | 0.00 | 0.11 | 1.24 | | |
| 50-54 | 0.72 | 0.36 | 0.18 | 0.53 | 0.51 | 0.15 | 0.13 | 2.58 | 50.54 | 0.82 | 0.01 | 0.00 | 0.16 | 0.66 | -0.01 | 0.01 | 1.63 | | |
| 55-59 | 0.97 | 0.61 | 0.26 | 1.10 | 0.90 | 0.20 | 0.16 | 4.20 | 55.59 | 0.90 | 0.06 | 0.02 | 0.15 | 0.80 | 0.03 | -0.17 | 1.79 | | |
| 60-64 | 0.80 | 1.01 | 0.33 | 2.09 | 1.31 | 0.64 | 0.25 | 6.43 | 60.64 | 1.21 | 0.08 | 0.04 | 0.37 | 1.58 | 0.02 | -0.04 | 3.27 | | |
| 65-69 | 0.92 | 1.56 | 0.50 | 3.23 | 1.48 | 1.29 | 0.23 | 9.20 | 65.69 | 1.23 | 0.13 | 0.08 | 0.85 | 2.79 | 0.05 | -0.07 | 5.05 | | |
| 70-74 | 0.96 | 2.55 | 0.78 | 5.51 | 1.94 | 2.10 | 0.18 | 14.01 | 70.74 | 1.09 | 0.26 | 0.09 | 1.26 | 4.71 | 0.07 | 0.00 | 7.48 | | |
| 75-79 | 1.09 | 2.89 | 0.84 | 6.42 | 0.67 | 3.01 | -0.08 | 14.83 | 75.79 | 0.73 | 0.32 | 0.11 | 1.66 | 5.65 | 0.21 | -0.30 | 8.38 | | |
| 80-84 | 1.09 | 2.29 | 0.61 | 5.27 | -0.63 | 2.86 | -0.23 | 11.25 | 80.84 | 0.23 | 0.35 | 0.12 | 1.53 | 5.69 | 0.30 | -0.22 | 8.02 | | |
| 85-89 | 0.55 | 1.31 | 0.34 | 3.16 | -1.00 | 1.84 | -0.21 | 6.00 | 85.89 | 0.03 | 0.27 | 0.09 | 0.90 | 4.11 | 0.32 | -0.29 | 5.43 | | |
| 90-94 | 0.13 | 0.45 | 0.11 | 1.14 | -0.60 | 0.61 | -0.11 | 1.73 | 90.94 | 0.00 | 0.11 | 0.02 | 0.35 | 1.66 | 0.18 | -0.15 | 2.18 | | |
| 95-99 | 0.05 | 0.06 | 0.01 | 0.16 | -0.13 | 0.11 | -0.02 | 0.24 | 95.99 | 0.01 | 0.04 | 0.01 | 0.09 | 0.25 | 0.06 | -0.06 | 0.40 | | |
| 100+ | 0.02 | 0.02 | 0.01 | 0.05 | 0.03 | 0.02 | 0.01 | 0.16 | 100+ | 0.00 | 0.00 | 0.00 | 0.01 | 0.00 | 0.00 | -0.02 | -0.01 | | |
| Total | 10.12 | 13.60 | 4.22 | 29.16 | 5.35 | 12.95 | 0.88 | 76.27 | Total | 8.58 | 1.69 | 0.61 | 7.79 | 28.70 | 1.27 | -0.61 | 48.04 | | |

Source: Authors' calculations based on HCD

¹ 161.9: Intracerebral haemorrhage, unspecified

² 163.3: Cerebral infarction due to thrombosis of cerebral arteries

³ 163.4: Cerebral infarction due to embolism of cerebral arteries

⁴ 163.9: Cerebral infarction, unspecified

⁵ 164₋: Stroke, not specified as haemorrhage or infarction

⁶ 167.2: Cerebral atherosclerosis

A.6 Contribution of cerebrovascular diseases to LL growth (in hundredths of a year). Women, 1980-2012

| | WOMEN, Cerebrovascular diseases | | | | | | | | | | 1996-2012 | | | | | 1996-2012 | | | | | | | | | | | |
|-------|---------------------------------|-------|------|-------|-------|-----------|-------|--------|-------|------|-----------|--------------------|--------------------|--------------------|--------------------|-------------------|--------------------|--------|----------|-----|-------|-------|-------|-------|-------|-------|--------|
| | 1980-1996 | | | | | 1996-2012 | | | | | Age | 161.9 ¹ | 163.3 ² | 163.4 ³ | 163.9 ⁴ | 164. ⁵ | 167.2 ⁶ | Others | All CBVD | Age | 161.9 | 163.4 | 163.3 | 163.9 | 164._ | 167.2 | Others |
| 0-44 | 1.95 | 0.39 | 0.24 | 0.44 | 0.56 | 0.10 | 0.16 | 3.84 | 0.44 | 0.94 | 0.02 | 0.00 | 0.19 | 0.53 | 0.02 | 0.45 | 2.15 | | | | | | | | | | |
| 45-49 | 0.48 | 0.17 | 0.10 | 0.26 | 0.30 | 0.06 | 0.02 | 1.38 | 45-49 | 0.45 | 0.01 | 0.00 | 0.01 | 0.32 | 0.00 | 0.07 | 0.85 | | | | | | | | | | |
| 50-54 | 0.46 | 0.31 | 0.17 | 0.45 | 0.41 | 0.08 | 0.09 | 1.96 | 50-54 | 0.61 | 0.02 | 0.01 | 0.08 | 0.47 | 0.00 | -0.06 | 1.14 | | | | | | | | | | |
| 55-59 | 1.00 | 0.54 | 0.27 | 0.88 | 1.28 | 0.30 | 0.23 | 4.52 | 55-59 | 0.57 | 0.01 | -0.01 | 0.20 | 0.49 | 0.01 | 0.10 | 1.37 | | | | | | | | | | |
| 60-64 | 0.99 | 1.07 | 0.48 | 1.93 | 1.78 | 0.54 | 0.51 | 7.29 | 60-64 | 0.87 | 0.06 | 0.02 | 0.32 | 0.91 | 0.01 | -0.13 | 2.06 | | | | | | | | | | |
| 65-69 | 1.64 | 1.71 | 0.69 | 3.28 | 2.70 | 1.37 | 0.53 | 11.92 | 65-69 | 1.13 | 0.12 | 0.05 | 0.49 | 2.27 | 0.03 | 0.27 | 4.36 | | | | | | | | | | |
| 70-74 | 1.86 | 3.21 | 1.08 | 7.05 | 2.79 | 2.42 | 0.34 | 18.76 | 70-74 | 0.96 | 0.26 | 0.06 | 1.11 | 4.62 | 0.08 | 0.24 | 7.32 | | | | | | | | | | |
| 75-79 | 2.30 | 4.28 | 1.25 | 9.87 | 1.52 | 4.13 | -0.05 | 23.28 | 75-79 | 1.34 | 0.53 | 0.21 | 1.96 | 8.60 | 0.35 | 0.70 | 13.71 | | | | | | | | | | |
| 80-84 | 1.98 | 4.52 | 1.30 | 10.41 | -0.90 | 4.58 | -0.35 | 21.54 | 80-84 | 0.66 | 0.75 | 0.24 | 2.57 | 11.27 | 0.61 | 0.03 | 16.13 | | | | | | | | | | |
| 85-89 | 1.52 | 3.00 | 0.79 | 7.16 | -2.69 | 3.66 | -0.51 | 12.92 | 85-89 | 0.14 | 0.76 | 0.23 | 2.19 | 9.40 | 0.71 | -0.33 | 13.10 | | | | | | | | | | |
| 90-94 | 0.68 | 1.08 | 0.24 | 2.70 | -2.04 | 1.41 | -0.35 | 3.72 | 90-94 | 0.04 | 0.43 | 0.11 | 1.11 | 4.47 | 0.48 | -0.38 | 6.26 | | | | | | | | | | |
| 95-99 | 0.09 | 0.26 | 0.07 | 0.62 | -0.51 | 0.36 | -0.06 | 0.83 | 95-99 | 0.09 | 0.13 | 0.03 | 0.28 | 0.98 | 0.21 | -0.11 | 1.60 | | | | | | | | | | |
| 100+ | 0.03 | 0.04 | 0.02 | 0.09 | -0.12 | 0.07 | -0.01 | 0.12 | 100+ | 0.01 | 0.02 | 0.00 | 0.05 | 0.19 | 0.02 | -0.01 | 0.28 | | | | | | | | | | |
| Total | 14.97 | 20.60 | 6.69 | 45.14 | 5.07 | 19.06 | 0.54 | 112.09 | Total | 7.81 | 3.12 | 0.97 | 10.55 | 44.52 | 2.53 | 0.82 | 70.33 | | | | | | | | | | |

Source: Authors' calculations based on HCD

¹ 161.9: Intracerebral haemorrhage, unspecified² 163.3: Cerebral infarction due to thrombosis of cerebral arteries³ 163.4: Cerebral infarction due to embolism of cerebral arteries⁴ 163.9: Cerebral infarction, unspecified⁵ 164._: Stroke, not specified as haemorrhage or infarction⁶ 167.2: Cerebral atherosclerosis

A.7 Contribution of ischaemic heart diseases to LE growth (in hundredths of a year). Men, 1980-2012

| | MEN, Ischaemic Heart diseases | | | | | | | | | | Total IHD | | | | | | |
|-------|-------------------------------|-------------------|-------------------|--------------------|--------------------|--------------------|--------|-----------|-------|------|-----------|-------|------|-------|--------|-------|-------|
| | 1980-1996 | | | | | 1996-2012 | | | | | | | | | | | |
| | 1210 ⁷ | 1219 ⁸ | 1249 ⁹ | 1250 ¹⁰ | 1251 ¹¹ | 1259 ¹² | Others | Total IHD | 1210 | 1219 | 1249 | 1250 | 1251 | 1259 | Others | | |
| 0-44 | 0.12 | 4.21 | 0.03 | -0.01 | -0.09 | -0.37 | 0.35 | 4.23 | 0.44 | 0.07 | 4.88 | -0.10 | 0.04 | -0.01 | -0.07 | 0.06 | 4.87 |
| 45-49 | 0.05 | 1.68 | 0.02 | 0.00 | -0.02 | -0.50 | 0.23 | 1.46 | 45-49 | 0.12 | 3.45 | -0.16 | 0.03 | -0.11 | -0.08 | -0.05 | 3.20 |
| 50-54 | 0.12 | 4.29 | 0.04 | 0.06 | 0.12 | -1.14 | 0.28 | 3.76 | 50-54 | 0.09 | 3.33 | -0.15 | 0.00 | -0.23 | 0.50 | 0.17 | 3.71 |
| 55-59 | 0.13 | 4.77 | 0.05 | 0.02 | 0.36 | -1.38 | 0.31 | 4.26 | 55-59 | 0.20 | 5.65 | -0.19 | 0.05 | -0.25 | 0.22 | 0.23 | 5.89 |
| 60-64 | 0.12 | 4.45 | 0.05 | 0.12 | 0.52 | -1.86 | 0.32 | 3.73 | 60-64 | 0.21 | 7.42 | -0.19 | 0.02 | -0.36 | 0.47 | 0.26 | 7.84 |
| 65-69 | 0.12 | 4.19 | 0.06 | 0.27 | 1.08 | -2.80 | 0.50 | 3.43 | 65-69 | 0.25 | 8.65 | -0.15 | 0.06 | -0.09 | 1.34 | 0.28 | 10.34 |
| 70-74 | 0.12 | 4.31 | 0.07 | 0.32 | 1.53 | -3.65 | 0.25 | 2.94 | 70-74 | 0.27 | 9.14 | -0.19 | 0.06 | 0.10 | 1.77 | 0.39 | 11.53 |
| 75-79 | 0.04 | 1.50 | 0.04 | 0.57 | 2.19 | -3.20 | 0.12 | 1.27 | 75-79 | 0.27 | 8.02 | -0.19 | 0.04 | 0.23 | 1.15 | 0.13 | 9.65 |
| 80-84 | 0.00 | -0.12 | 0.03 | 0.53 | 1.92 | -2.23 | 0.20 | 0.34 | 80-84 | 0.17 | 5.10 | -0.27 | 0.06 | 0.61 | 0.21 | -0.06 | 5.83 |
| 85-89 | 0.00 | -0.09 | 0.01 | 0.46 | 1.33 | -1.13 | 0.01 | 0.59 | 85-89 | 0.08 | 1.79 | -0.14 | 0.07 | 0.59 | -0.71 | -0.01 | 1.67 |
| 90-94 | 0.00 | -0.13 | 0.01 | 0.11 | 0.38 | -0.36 | 0.04 | 0.05 | 90-94 | 0.02 | 0.49 | -0.08 | 0.04 | 0.37 | -0.48 | -0.05 | 0.31 |
| 95-99 | 0.00 | -0.02 | 0.00 | 0.02 | 0.08 | -0.10 | 0.01 | -0.01 | 95-99 | 0.00 | 0.05 | -0.02 | 0.01 | 0.16 | -0.08 | -0.01 | 0.10 |
| 100+ | 0.00 | 0.03 | 0.00 | 0.00 | 0.00 | -0.01 | -0.01 | 0.02 | 100+ | 0.00 | 0.00 | 0.00 | 0.01 | 0.03 | -0.02 | 0.00 | 0.02 |
| Total | 0.80 | 29.05 | 0.42 | 2.48 | 9.41 | -18.73 | 2.63 | 26.05 | Total | 1.76 | 57.96 | -1.83 | 0.46 | 1.03 | 4.21 | 1.35 | 64.95 |

Source: Authors' calculations based on HCD

⁷121.0 Acute transmural myocardial infarction of anterior wall (I21.0)

⁸121.9 Acute myocardial infarction, unspecified (I21.9)

⁹124.9 Acute ischaemic diseases, unspecified (I24.9)

¹⁰125.0 Atherosclerotic cardiovascular disease, so described (I25.0)

¹¹125.1 Atherosclerotic cardiovascular disease (I25.1)

¹²125.9 Chronic ischaemic heart disease, unspecified (I25.9)

A.8 Contribution of ischaemic heart diseases to LE growth (in hundredths of a year). Women, 1980-2012

| | WOMEN, Ischaemic Heart diseases | | | | | | | | | | Total HHD | | | | | | |
|-------|---------------------------------|-------------------|-------------------|--------------------|--------------------|--------------------|--------|--------------|-------|-------|--------------|-------|-------|-------|--------|-------|-------|
| | 1980-1996 | | | | | 1996-2012 | | | | | | | | | | | |
| | 1210 ⁷ | 1219 ⁸ | 1249 ⁹ | 1250 ¹⁰ | 1251 ¹¹ | 1259 ¹² | Others | Total HHD | 1210 | 1219 | 1249 | 1250 | 1251 | 1259 | Others | | |
| 0-44 | 0.02 | 0.79 | 0.01 | 0.03 | 0.02 | -0.07 | 0.19 | 0.99 | 0.44 | 0.04 | 0.82 | -0.04 | 0.05 | 0.04 | 0.03 | -0.06 | 0.88 |
| 45-49 | 0.01 | 0.24 | 0.01 | 0.01 | 0.06 | -0.10 | 0.05 | 0.27 | 45-49 | -0.01 | 0.18 | -0.01 | 0.00 | -0.01 | 0.00 | -0.02 | 0.13 |
| 50-54 | 0.02 | 0.78 | 0.01 | 0.02 | 0.08 | -0.23 | 0.12 | 0.80 | 50-54 | 0.01 | 0.27 | 0.00 | 0.00 | -0.02 | 0.07 | 0.00 | 0.33 |
| 55-59 | 0.04 | 1.58 | 0.02 | 0.01 | 0.24 | -0.30 | 0.15 | 1.75 | 55-59 | 0.01 | 0.45 | -0.01 | 0.02 | -0.02 | 0.09 | 0.01 | 0.55 |
| 60-64 | 0.05 | 1.84 | 0.03 | 0.10 | 0.55 | -0.70 | 0.16 | 2.03 | 60-64 | 0.02 | 1.11 | -0.02 | 0.02 | 0.02 | 0.31 | 0.06 | 1.52 |
| 65-69 | 0.08 | 2.80 | 0.04 | 0.21 | 1.09 | -0.95 | 0.17 | 3.45 | 65-69 | 0.07 | 2.57 | -0.02 | 0.01 | 0.07 | 0.56 | 0.08 | 3.35 |
| 70-74 | 0.05 | 1.89 | 0.07 | 0.66 | 2.10 | -1.89 | 0.43 | 3.30 | 70-74 | 0.13 | 4.26 | -0.02 | 0.01 | 0.22 | 1.34 | 0.15 | 6.09 |
| 75-79 | 0.00 | 0.16 | 0.04 | 0.91 | 3.32 | -2.53 | 0.28 | 2.18 | 75-79 | 0.13 | 5.35 | -0.05 | 0.05 | 0.44 | 1.58 | 0.17 | 7.68 |
| 80-84 | -0.01 | -0.34 | 0.03 | 1.10 | 4.25 | -2.82 | 0.21 | 2.42 | 80-84 | 0.12 | 4.35 | -0.09 | 0.07 | 0.81 | 1.49 | 0.16 | 6.90 |
| 85-89 | -0.03 | -0.91 | 0.03 | 1.03 | 2.92 | -2.11 | 0.09 | 1.02 | 85-89 | 0.08 | 2.45 | -0.14 | 0.09 | 1.13 | 0.74 | 0.08 | 4.42 |
| 90-94 | -0.01 | -0.43 | 0.01 | 0.46 | 1.54 | -0.86 | 0.01 | 0.71 | 90-94 | 0.03 | 0.82 | -0.10 | 0.07 | 0.85 | 0.07 | 0.00 | 1.74 |
| 95-99 | 0.00 | -0.10 | 0.01 | 0.17 | 0.10 | -0.21 | 0.03 | 0.00 | 95-99 | 0.01 | 0.09 | -0.03 | 0.01 | 0.39 | -0.10 | -0.02 | 0.35 |
| 100+ | 0.00 | 0.00 | 0.00 | 0.02 | -0.02 | -0.03 | 0.00 | -0.05 | 100+ | 0.00 | -0.02 | -0.06 | -0.01 | 1.55 | -0.74 | -0.03 | 0.69 |
| Total | 0.23 | 8.31 | 0.30 | 4.73 | 16.24 | -12.80 | 1.86 | 18.87 | Total | 0.63 | 22.71 | -0.57 | 0.39 | 5.46 | 5.42 | 0.59 | 34.63 |

Source: Authors' calculations based on HCD

⁷121.0 Acute transmural myocardial infarction of anterior wall (I21.0)⁸I21.9 Acute myocardial infarction, unspecified (I21.9)⁹I24.9 Acute ischaemic diseases, unspecified (I24.9)¹⁰I25.0 Atherosclerotic cardiovascular disease, so described (I25.0)¹¹I25.1 Atherosclerotic cardiovascular disease (I25.1)¹²I25.9 Chronic ischaemic heart disease, unspecified (I25.9)

ANEXO B. Cálculo de las aportaciones por edad y causa a los cambios en la esperanza de vida

Los métodos de descomposición se emplean para determinar qué grupos de edad y/o enfermedades son las responsables de las diferencias en la esperanza de vida de dos poblaciones —o la misma en dos períodos temporales—. Existen diversos métodos para medir estas contribuciones por edad/causa, algunos de ellos desde un enfoque continuo (Andreev, 1982; Pollard, 1982), otros desde uno discreto (Arriaga, 1984). Sin embargo, como los mismos autores reconocen, ambos enfoques tienen resultados semejantes (Pollard, 1988), por lo que en este trabajo se ha seguido la aproximación discreta.

Para determinar la contribución de cada grupo de edad a la esperanza de vida, es necesario calcular sus dos componentes, el efecto directo (ED_x), es decir, el cambio en años de vida *en* un grupo de edad concreto como consecuencia del cambio en la mortalidad de dicho grupo; y el efecto indirecto (EI_x), o sea, el número de años de vida añadidos a la esperanza de vida debido a los cambios en la mortalidad *en* —y solo en— un grupo de edad específico que producirá un cambio en el número de supervivientes al final del intervalo de edad (Arriaga, 1984). Dicho de otro modo, ED_x da cuenta de los años de vida ganados en un grupo de edad concreto, y EI_x hace referencia a las ganancias en los siguientes grupos de edad como consecuencia de una mayor supervivencia —es decir, de una menor mortalidad— en una edad determinada.

Así, siendo a y b las poblaciones que se pretende comparar (por ejemplo, la masculina entre 1980 y 1996), la contribución a la esperanza de vida (Δ_x) es resultado de la suma de los efectos directos e indirectos:

$$\Delta_x = ED_x + EI_x = \frac{l_x^a}{l_0^a} \cdot \left(\frac{L_x^b}{l_x^b} - \frac{L_x^a}{l_x^a} \right) + \frac{T_{x+1}^b}{l_0^a} \left(\frac{l_x^a}{l_x^b} - \frac{l_{x+1}^a}{l_{x+1}^b} \right)$$

Siendo l_x^a el número de supervivientes a la edad x de las tablas de mortalidad, L_x^a el número de personas-año vividos hasta la edad x y T_{x+1}^b los años que restan por vivir a la edad x . El grupo de edad abierto (en este caso, 100 y más años) es una excepción, pues no aporta —lógicamente— efectos indirectos. Se calcula mediante la expresión:

$$\Delta_\omega = \frac{l_\omega^a}{l_0^a} \cdot \left(\frac{T_\omega^b}{l_\omega^b} - \frac{T_\omega^a}{l_\omega^a} \right)$$

Por otra parte, esta técnica de descomposición por grupos de edad puede ampliarse para analizar la contribución de una —o varias— causa de muerte al aumento de la esperanza de vida en cada grupo edad. Esta operación

se realiza mediante el producto de la contribución a la esperanza de vida de un grupo de edad (Δ_x), y el peso proporcional de la(s) causa(s) (k) que se quieren estudiar (C_{xk}) en el mismo intervalo:

$$\Delta_{xk} = \Delta_x \cdot C_{xk}$$

Para hallar (C_{xk}) empleamos la expresión siguiente (Arriaga, 1989; Nusselder y Looman, 2004):

$$C_{xk} = \frac{R_{xk}^b \cdot m_x^b - R_{xk}^a \cdot m_x^a}{m_x^b - m_x^a}$$

Siendo (R) la proporción de defunciones de la causa (k) en el grupo de edad (x) y m_x la tasa de mortalidad específica por edad en las poblaciones a y b .

Finalmente, para evitar que los resultados de la descomposición dependan de qué población escojamos como primera o segunda, algunos autores aconsejan como último paso repetir el proceso invirtiendo el orden de las poblaciones y calcular el promedio de los dos resultados. Es decir, calculamos las contribuciones por edad/causa de a respecto a b , a continuación las de b sobre a , y hallamos la media aritmética de ambas (Shkolnikov, Valkonen, Begun, y Andreev, 2001; Nusselder y Looman, 2004).

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