

Cause-of-Death Contribution to the Female-Male Gap in Mortality in the United States

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Abstract

In the last half a century improvements in mortality were characterized by different patterns for American males and females. Sex-gap in life expectancy at birth, growing for the benefit of women until the mid-1970s, has been narrowing since. The most often used explanation for this development are growing similarities in behavioral patterns of the two sexes. In this study we bring forward additional argument: Differential benefits of the sexes from medical developments. We study effect of policy preventable and medically amenable causes of death on the existing sex-gap in life expectancy, by decomposing the difference in the mean duration of life between ages 0 and 75 into four large groups of causes of death. We show that a large part of the narrowing gap in mortality between the sexes results from women starting to undertake typical male, risky behaviors. Although we observe excess female mortality from causes amenable to medical care, these resulted mainly from high mortality among females from breast cancer. This cause of death affects almost exclusively women and hence we conclude that to compare effectiveness of medical interventions, one should examine first sex differences in incidence of a disease.

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1 Introduction

Women live longer than men. In the last decades, however, in many developed countries improvements in the mean duration of life were faster for men than for women. We can observe an almost universal pattern where the steady increase in the sex-gap in life expectancy at birth (female minus male) stopped by the mid-1970s and 1980s and has been narrowing ever since in many developed countries (Glei and Horiuchi, 2007). The United States represents no exception: Since the mid-1970s, life expectancy at birth has been improving faster for males than for females, resulting in a shrinking gap between the two (Figure 1). As a result, the sex-gap in life expectancy decreased from the largest observed value of 7.7 years in 1975 to 5.2 years in 2005, the most recent year for which we have data.

The faster increase in life expectancy for women than for men until 1975 — resulting in a widening gap between the two sexes — was not triggered by a single factor. Male excess mortality is most often discussed as a result of differences in behavioral patterns between the sexes that expose men to a greater extent to mortality risks. Those behaviors include “... smoking, drinking, driving, and violence” (Nathanson, 1984, p. 204). For sure, the more widespread adoption of smoking among men born during the first decades of the 20th century (see, for instance, Giovino, 2002; Giovino et al., 1995; Harris, 1983) contributed considerably to the difference. Increasing male excess mortality is only one side of the coin, though. As discussed by Vallin (2006), significant progress in combating infectious diseases and improvements in the status of women led to lower maternal mortality and mortality at younger ages. This development allowed women to fully benefit from their biological survival advantages such as benefit of having an additional X-chromosome (Christensen et al., 2001), estrogens that protect against circulatory diseases until menopause (Roeters van Lennep et al., 2002) or an advantage to store and eliminate food reserves that make women better fit to endure over feeding and easier to adjust to existing living conditions (Seely, 1990).

To explain the narrowing differences in life-expectancy between the sexes since the mid-1970, women's growing involvement in previously male-dominated risky behaviors are usually brought forward (e.g. Case and Paxson, 2005; Pampel, 2002; Preston and Wang, 2006; Vallin et al., 2006; Wingard, 1984). The major role is commonly attributed to the increased smoking prevalence among women. For example, Pampel (2002, p. 96) argues "smoking fully explains the recent narrowing of the sex differential" . An argument not often discussed in demographic studies, however, is that both sexes might have benefited differently from advances in medicine and new medical technologies with greater benefits for men than for women. We disregard here sex-differences in behavioral patterns like the propensity to see a doctor. The reason is that, despite the fact that women more often consult a doctor in general, men are equally likely as women to seek medical help for life-threatening illnesses (Waldron, 1983), but also "short-stay and emergency hospital services are more accessible to men" (Ruiz and Verbrugge, 1997, p. 107). The benefits of men from medical developments are possibly greater than for women due to higher on average socio-economic status and as a result more generous type of health insurance (Chulis et al., 1993). Since the type of insurance is often claimed to determine the type and quality of treatment and medications received (Blustein, 1995; Hurd and McGarry, 1997; Pezzin et al., 2007; Shi, 2000), men in the United States might have better access to expensive procedures and medicines. Furthermore, the biological differences between men and women also require variation in the diagnosis and treatment (Henry, 2005; Oda et al., 2006). Those two might currently be insufficiently tailored to the women's physiological needs, as many medical solutions result from studies based entirely on men (Bennett, 1993; Cotton, 1990; Gregg et al., 2007; Merkatz et al., 1993).

Summarizing, our research question is: To which extent the recent trend of the narrowing sex-gap in life expectancy in the United States can be explained by behavioural factors or differential benefits of the sexes from the developments in medicine and medical technology? The following section outlines the data and methods we employed to test this hypothesis.

2 Data and Method

2.1 Data

For our mortality analysis, we used two data sources. Information on deaths were taken from the “Multiple Cause-of-Death Mortality Data” from the National Vital Statistics System of the National Center for Health Statistics (NCHS). They have been obtained from the National Bureau of Economic Research and downloaded free of charge from the website.¹

Although data are available starting in 1959, we restricted our analysis to the year 1968–2004. The reason to limit the years under study results from problems related to differences in coding in the subsequent revisions of the “International Classification of Diseases”. The seventh revision (“ICD-7”), that was used in the United States before 1968, did not contain any category for ischaemic heart disease (IHD). Only with the introduction of this category in ICD-8 in 1968, we were enabled to classify these deaths correctly. The longest part of our observation window was covered by ICD-9 (1979–1998) and since 1999, ICD-10 has been employed to classify causes of death.

With the exception of 1972, these files list all deaths in the United States annually on the individual level. In 1972, only a 50% sample has been taken and we simply multiplied all death counts by 2 in this year. Since we looked only at large categories, we assume that the 50% sample was random at this level and the mistake is negligible.

To obtain mortality estimates, we matched those death counts by age, sex, and cause with the corresponding population data by age and sex which have been downloaded from the Human Mortality Database (University of California, Berkeley (USA), and Max Planck Institute for Demographic Research, Rostock, (Germany), 2008).

¹The URL is: <http://www.nber.org/data/vital-statistics-mortality-data-multiple-cause-of-death.html>.

2.2 Method

check here the text in the old paper To analyze whether men benefited more from medical progress in recent decades in the United States, we grouped the retrieved causes of death into four categories:

1. Causes of death that we believe could have been prevented by medical intervention or technology.
2. The selection of the second group of causes in this study — amenable by health policy and behavioral factors — rests on the assumption that the respective deaths could have been avoided if people had other habits than they actually exhibited.
3. Deaths from IHD form a separate group, as they are both preventable by health policy solutions and amenable to medical interventions (James et al., 2007a). Furthermore, “the precise contribution of medical care to reductions in deaths from this condition is unresolved [...] the large number of deaths involved is likely to conceal the impact of medical on diseases other than IHD” (Nolte et al., 2002, p. 1907).
4. A residual category is formed by deaths from the remaining causes.

Our methodology is based on Rutstein et al. (1976). They proposed a method of measuring the quality of medical care by an index based on a list of conditions resulting in disease, disability or death, that could have been otherwise treated or prevented by the health care system. This indicator of amenable mortality is often employed to assess the performance of health care systems, in particular in international comparisons (Douglas and Mao, 2002; Mackenbach et al., 1990; Nolte and McKee, 2003, 2008, for example,). Following the original classification, other authors distinguish between causes of death amenable to medical intervention (treatable conditions) and those amenable to inter-sectoral health policies (preventable conditions), as well as, separate Ischaemic Heart Disease (IHD) from other conditions (Andreev et al., 2003). That means that deaths amenable to medical care are those that could be prevented after the

condition develops, and deaths amenable to public health policies are caused by conditions that themselves could have been otherwise avoided from occurring.

list in the appendix ten most important causes of death

The causes considered to be amenable to public health are: HIV, lung cancer, skin cancer, chronic obstructive pulmonary disease, liver cirrhosis and motor vehicle accidents. The number of causes amenable to medical care is considerably larger (for the full list of causes medically amenable compare Hem et al. (2007)). The most notable causes are the majority of infectious diseases, cerebrovascular diseases, respiratory diseases like pneumonia, influenza or asthma, and several cancers such as breast cancer, prostate cancer, or leukaemia.

Coding of causes of death across various ICD revisions is notoriously problematic. We used the schedule provided by Hem et al. (2007) and James et al. (2007b) to guide us through the coding of the four cause-categories from ICD-8 to ICD-9 (James et al., 2007b) and from ICD-9 to ICD-10 (Hem et al., 2007). A complete listing of all ICD codes used for the four categories can be requested from the corresponding author.

We disregard deaths that occurred after the age of 75 years, as deaths above this age are less likely to be preventable. Furthermore, certification of causes above this age is claimed to be less accurate than for deaths at younger ages (James et al., 2007a; Nolte and McKee, 2008). As we set the upper age limit for amenable causes of death to 75 completed years, our indicator is not the traditional parameter “life expectancy at birth” (e_0) but the average number of years lived until age 75. Due to the fact that the reasons for excess male mortality are very different for infants than for the remaining age groups (Drevenstedt et al., 2008), we further conduct separate analyses for ages between (a) 0 and 75 years and (b) 1 and 75 years.

The effect of the four groups of causes of death on the sex-gap in the number of years lived between age 0 and 75 years is quantified using discrete decomposition method of Arriaga (1984). The same method is used to determine contribution of these groups of causes to the change in the sex-gap, as well as to quantify these values in five-year age-groups. Below we fol-

low the description of the method as presented in Preston et al. (2001).

If we define contribution of difference in mortality rates between the sexes (f–female, m–male) at age $(x, x + n)$ to the total gap in e_{075} as:

$${}_n\Delta_x = \frac{l_x^m}{l_0} \left(\frac{{}_nL_x^f}{l_x^f} - \frac{{}_nL_x^m}{l_x^m} \right) + \frac{T_{x+n}^f}{l_0} \left(\frac{l_x^m}{l_x^f} - \frac{l_{x+n}^m}{l_{x+n}^f} \right) \quad (1)$$

where the first part of the equation stands for a direct effect of the difference in rates between age x and $x + n$, and the second part measures indirect and interaction effects at ages above $x + n$. Further we assume that the distribution of deaths by cause is constant within each age group. It means that contribution of the difference in mortality rates from cause i at ages $x-x + n$ will be proportional to the weight of deaths from this cause in each age group.

$${}_n\Delta_x^i = {}_n\Delta_x \frac{{}_n m_x^i(f) - {}_n m_x^i(m)}{{}_n m_x(f) - {}_n m_x(m)} = {}_n\Delta_x \frac{{}_n R_x^i(f) {}_n m_x(f) - {}_n R_x^i(m) {}_n m_x(m)}{{}_n m_x(f) - {}_n m_x(m)} \quad (2)$$

where ${}_n R_x^i(j)$ measures proportion of deaths from cause i at age $x-x + n$ in population j :

3 Results

In Table 1 (page 20), we provide basic statistics for the 42.9 million deaths that have been recorded in the United States between the year 1968 and 2004 at ages 0 to 75 – separating them by sex to four categories of causes of death. About 22% of all deaths among women and men were due to ischaemic heart disease. Among women about 30% of all deaths belong to the category “amenable to medical care” whereas the corresponding proportion among men is lower with approximately 16%. Deaths in the category “amenable to public health” are more common among men (19.7%) than among women (14.6%).

The group of cause medically amenable includes deaths from diseases that affect significantly single sex-only. We are interested in the effect of two of them – breast cancer and prostate cancer – to which extent the results would differ if we exclude them from the causes medically

amenable. In the bottom part of Table 1 we present the impact of those causes for the overall burden of the category “amenable to medical care”: About 20% of all deaths among females in this group can be attributed to breast cancer. A smaller proportion of about 10% of men died from prostate cancer. Hence, inclusion of those causes distorts the overall picture concerning sex-differences in life-expectancy as a result of causes preventable by medical interventions for both sexes.

Despite that the main goal is to explain the recent phenomenon of narrowing sex-gap in life expectancy, due to the reasons as explained in section 2.2, we study differences in the expected number of years lived between age 0 and 75 years. On Figure 2 the sex gap (female minus male) in life expectancy at birth in the calendar period 1968–2004 to the difference in the expected number of years lived between ages 0 and 75 (e_{075} , blue line) and between ages 1 to 75 (e_{175} , red line). As we can observe, the trajectory of the sex gap in life expectancy at birth is largely driven by ages at or below our threshold of 75 years. Developments over time of the expected number of years lived between ages 0–75 and 1–75 years very closely resemble the pattern observed for the gap in life expectancy at birth. By comparing those two results one can also draw conclusions concerning development of infant mortality: The greater decrease in the sex-gap for ages 0–75, as compared to ages 1–75 in the years 1968–1995, indicates faster improvements in infant mortality for male than for female newborns over this period. According to Drevenstedt et al. (2008, p. 5018), since 1970 “the increasing use of C-section and improvements in neonatal medicine further reduced infant mortality, particularly among small and premature infants, which disproportionately benefited males.” Different in the last decade, due to a comparable improvements in infant mortality for both sexes, the difference in the male-female gap between ages 0–75 and 1–75 remained at a rather at a constant level.

In the next step, we decomposed the sex-gap in the expected number of years lived between ages 0–75 and ages 1–75 into four groups according to underlying cause of death. The results

are presented in Figure 3, with positive numbers indicating a female survival advantage.

Over the entire period under study, the major difference between the sexes in the mean number of years lived between age 0 and 75 (upper panel) resulted from the contribution of 'other' causes of death (grey bars). The second largest contributor to the gap was Ischaemic Heart Disease (red bars). The average number of years that women lived longer than men due to this cause decreased from 2 years and 7 months in 1968 to less than 11 months in 2004. With regard to the importance of causes preventable by *policy interventions*, their contribution to the sex-gap in e_{075} varied between 1 year 5 months and 1 year 8 months in the years 1968-1995. Afterwards it dropped to less than one year in 2004. Causes amenable to *medical care*, displayed with blue bars, accounted for about 5 months of the gap in at ages 0–75 in the year 1968, while at ages 1–75 the contribution of these causes was close to zero. It means that the difference of about 5 months in the number of years lived between men and women in 1968 can be attributed to the excess male infant mortality due to causes amenable to medical interventions. In later years, the contribution of medically amenable causes to the gap at ages 0–75 decreases to the minimum value of 1.5 months. After excluding age 0, the contribution of this group becomes negative (except for the last year presented here, where it is negligible). A negative contribution to the sex-gap in life expectancy represents higher number of year-lives lost due to causes medically amenable among women than among men.

Since we discovered slightly different results in our analysis of Figure 3, when infant mortality was excluded from our age-range, we proceeded by studying the age pattern of contribution of the four groups of causes of death. Therefore, the sex-gap in the expected number of years lived between age 0 and 75 has been further decomposed into 5-year age groups. The results are presented in Figure 4 for the years 1968, 1985 and 2004. We used the same colors as in the previous figure to denote the four groups of causes of death.

In 1968 the importance of *Ischaemic Heart Disease* for the sex gap in the expected number of years lived grew with age and reached a maximum of 5.8 months at 60–64 years of age. Despite being lower than at ages 60–64, IHD was the largest contributor to the sex gap in life expectancy at ages 40 and higher **which ages exactly** in the year 1968. In the same year, one can observe two peaks in the age distribution of the sex-gap for causes preventable by *health policy* interventions and *other*: At young adult ages and around age 60. It can be argued that the first peak of excess male mortality results from higher incidence among men of violent deaths, car accidents, and other causes of death related to their propensity to undertake risky behaviors among men (Nathanson, 1984). A similar pattern of two dominant values can be observed in the other two years studied here (in 1985 and 2004). **to comment probably when we look in detail in the 'other' causes of death**

In 1968 causes of death amenable to *medical care* disadvantaged male infants versus female ones. The difference in life expectancy resulting from the excess infant male mortality equaled 5 months in 1968. On the contrary, at ages 25–59 it is probably men who benefit more from medical interventions to prevent death: At these ages the contribution of medically amenable causes of death to the sex-gap is negative, which indicates men's advantage in survival. At older ages (55–74 years) the contribution of medically amenable deaths to the expected number of years lost is again lower for women than for males, as reflected in the positive sex-gap at those ages.

In the other years under study, age specific contributions of causes amenable to medical interventions and IHD to the sex-gap in e_{075} , resemble closely their respective distributions from 1968. The disadvantage of men as an outcome of the excess infant mortality due to causes amenable to medical intervention reduces to 1.5 months in 1985 and to less than one month in 2004. In 1985, the disadvantage of women—as a result of causes amenable to medical developments—concentrates at ages 25–69, and in 2004 at ages 25–59 years. We expect that the disadvantage of women due to medically amenable conditions at those ages is most likely a result of mortality from breast cancer: The ages of the excess female mortality are also the ages of the occurrence of

the aggressive, genetically determined breast cancer. According to Manton and Stallard (1992), the premenopausal breast cancer is strongly associated with family histories and not with exposure to risk factors, that otherwise could have been prevented by public policy measures. In order to exclude deaths from single-sex-specific cancers, we study the effect of causes amenable to medical care other than breast and prostate cancer in Figure 5.

When deaths from breast cancer are excluded from the group of medically amenable conditions, the disadvantage of women due to the remaining causes in the group is not present. This is the case for all the age-groups for which the original gap was negative (i.e. the difference in the expected number of years lived was for the benefit of men). After deduction of deaths from breast cancer among females, the contribution of the remaining causes amenable to medical care to the sex-gap in life expectancy remains positive and at a similar level in the studied years. Even when prostate cancer as an important cause of death for males, as shown in Table 1, is excluded from the remaining medically amenable causes, the contribution of this group to the sex-gap in e_{075} remains positive and for the benefit of women. This is the case for all the age-groups. That means that disadvantage of women due to premature deaths from causes amenable to medical interventions results from the sad fact that no cure has been discovered so far for the aggressive breast cancer, and not because medicine and medical technology disadvantage women. If we came to the latter conclusion, that would mean that if breast cancer occurred as often among men as among women than there would be more efforts done to discover cure against it. Such a conclusion would be illegitimate, in particular because it cannot be tested in the real world.

Our analysis so far focussed only on the contribution of the four categories to the sex gap in selected years. For the remainder of our investigation, we analyze the contribution of the four categories of causes of death to the *change* in the average number of years lived between ages 0 and 75. The respective results are given in Table 2 where we conduct again a separate

analysis without infant mortality. Furthermore, we also include in this table the contribution of the group of causes amenable to medical care excluding breast and prostate cancer.

Taking into account all causes of death, the years 1968–2004 were characterized by a steady decrease in the sex-gap in the expected number of years lived between age 0 and 75 (“Total change, ages 0–75”). The fastest decrease in this measure was observed in the last decade of the period under study (-1.230).

Although the sex-gap in life-expectancy at birth still increased between 1968 and 1975, we can see in our table that the sex-gap in the average number of years lived between ages 0 and 75 already slightly decreased (-0.116 years).

Apart from one time-interval, the decrease in the sex-gap resulted from both, changes in infant mortality, as well as, mortality at ages 1 to 75 years. Between 1968 and 1975, as an exception here, it was women who experienced greater improvements in mortality than men among aged 1–75 years. This exception was caused by a faster decrease among women than men in the contribution of deaths classified as ‘other’. This shift more than counterbalanced changes in the opposite direction of the remaining three groups of causes.

In general, the contribution of ‘other’ causes to the shift in the sex-gap was positive in the years 1968–1985 and negative between 1985 and 2004. It means that while improvements in mortality from these causes were bigger for women in the first half of the studied period, they were of greater importance among men in the second half.

As far as Ischemic Heart Disease is concerned, in the entire study period, men benefited at a faster pace than women from a decrease in mortality from this disease. The fastest drop in male mortality from this cause—compared to women—occurred between 1975 and 1995, with this group being the major contributor to the decrease in the sex-gap in these years (-0.799 years between 1975 and 1985; -0.539 years between 1975 and 1995). Not surprisingly, almost the entire shift of the sex-gap related to changes in IHD mortality occurred above age zero.

With the exception of the decade between 1985 and 1995, improvements related to causes of death preventable by public interventions were greater among men. Only in the years 1985-1995, the contribution of this category decreased faster among females than males and hence resulted in an increase in the average number of years lived before age 75 (0.146) sexes. Almost the entire share of this deaths preventable by public health interventions originates at ages 1-75; the contribution of infant ages is rather negligible. In the last ten years under study, it was the group of causes preventable by public policy interventions that had the second (after 'other' causes) greatest contribution to the narrowing sex-gap in mortality (-0.606).

With the exception of the last decade under study, the contribution of causes amenable to medical care to change in the sex-gap in the expected number of years lived between age 0 and 75 was negative, i.e. for the benefit of men, as we postulated in our initial hypothesis. In the first decades of the years 1968–1995, the benefits for men were much greater than for women (-0.279). Over time, however, the negative impact of these causes on the sex-gap was decreasing with every next decade. Finally, it was women during the most recent observation period between 1995 and 2004, whose benefit from the medical developments were greater and hence this group of causes increased the existing gap between the sexes. Separate analyses by age (age 0 vs. ages 1–75) and by excluding the sex-specific cancers (breast and prostate) did not change the general pattern: over time, the reductions in the average number of years lived between the sexes due causes treatable by medical care became smaller. In the most recent years, the sex-gap would have actually increased again if the category “amenable to medical care” were the only causes of death.

4 Summary, Discussion and Conclusion

The aim of this paper is to explain the narrowing in the sex-gap in life expectancy in the United States in recent decades. Our hypothesis was that men might have benefited more than women

from medical progress during this time. To test this hypothesis, we decomposed the difference in the mean number of years lived between age 0 and 75 into four groups of causes of death. Two major groups of interest following this classification are (1) deaths amenable to medical care and (2) deaths amenable to public health policies. Whereas the first can be considered as an indicator of medical progress (e.g. new medicines, new medical procedures, ...) to treat life-threatening conditions, the latter is more concerned with behavioral patterns and potential policy interventions to *prevent* those (e.g. smoking, drinking, reckless driving, ...). However, both medical progress and behavioral patterns have also a considerable impact on the development of Ischaemic Heart Disease (IHD). Due to the sheer size of deaths from IHD — about 22% of all deaths during our observation period — their impact on the gap itself and the difficulty to assign deaths from IHD to one or the other category, IHD forms a separate group, as previously done, for example, by Hem et al. (2007). The fourth, and final, group is a residual category. The idea of classifying “avoidable” deaths has been originally proposed by Rutstein et al. (1976) and was often applied to make international comparisons of health care systems.²

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²According to a search at the “ISI Web of Knowledge” website (<http://isiknowledge.com/>) on 18 February 2009, the original article by Rutstein et al. (1976) has been cited **x times**

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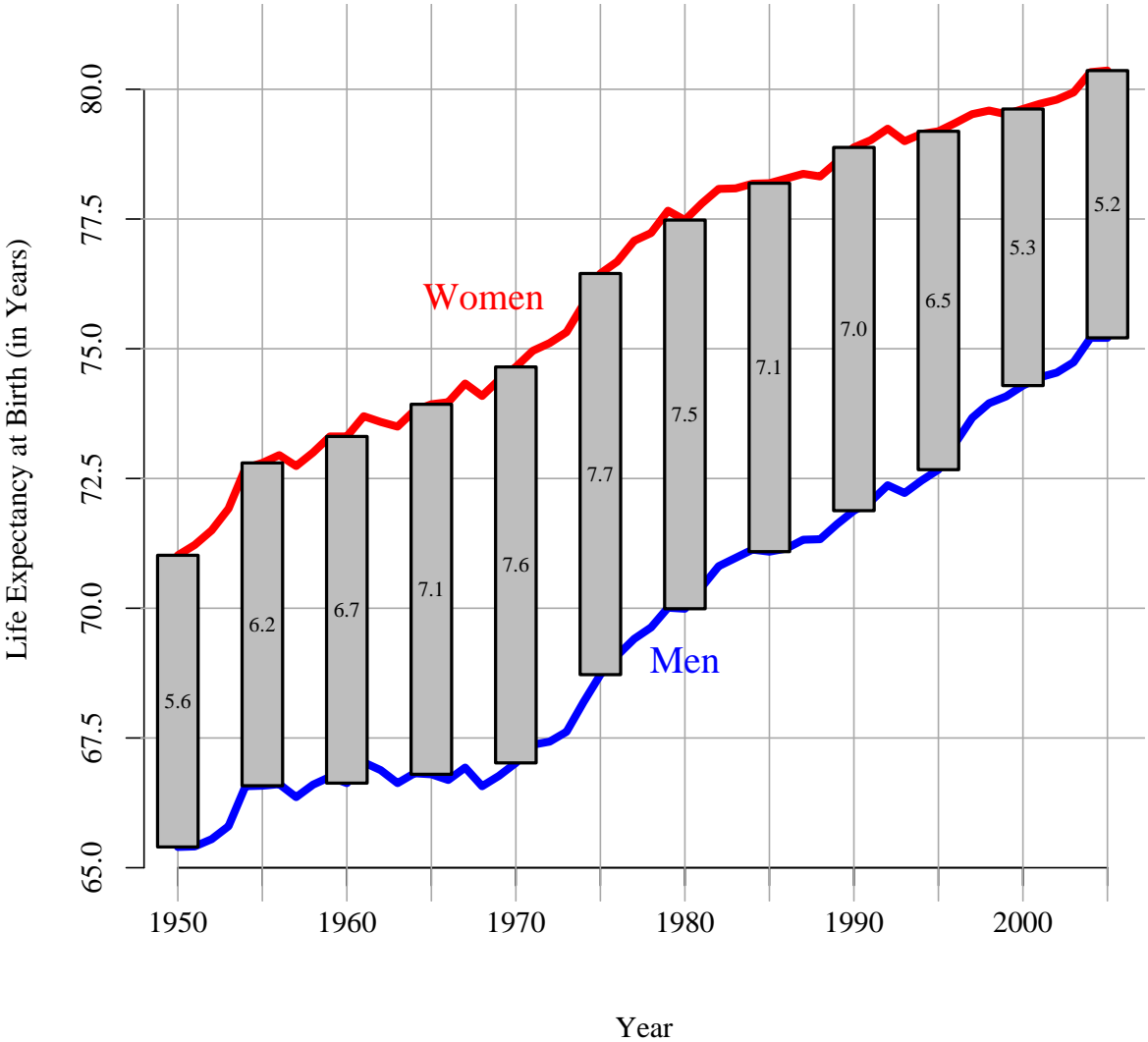
TABLES AND FIGURES

Table 1: Numbers of Death by Cause Category; Females, Males, and Total, United States 1968–2004

Cause	Women		Men		Total	
	Count	%	Count	%	Count	%
IHD	3,100,616	18.60	6,406,340	24.42	9,506,956	22.16
Medical Care	4,941,881	29.64	4,276,540	16.30	9,218,421	21.48
Public Health	2,433,731	14.60	5,175,809	19.73	7,609,540	17.73
Rest	6,197,049	37.17	10,377,225	39.55	16,574,274	38.63
Σ	16,673,277	100.00	26,235,914	100.00	42,909,191	100.00

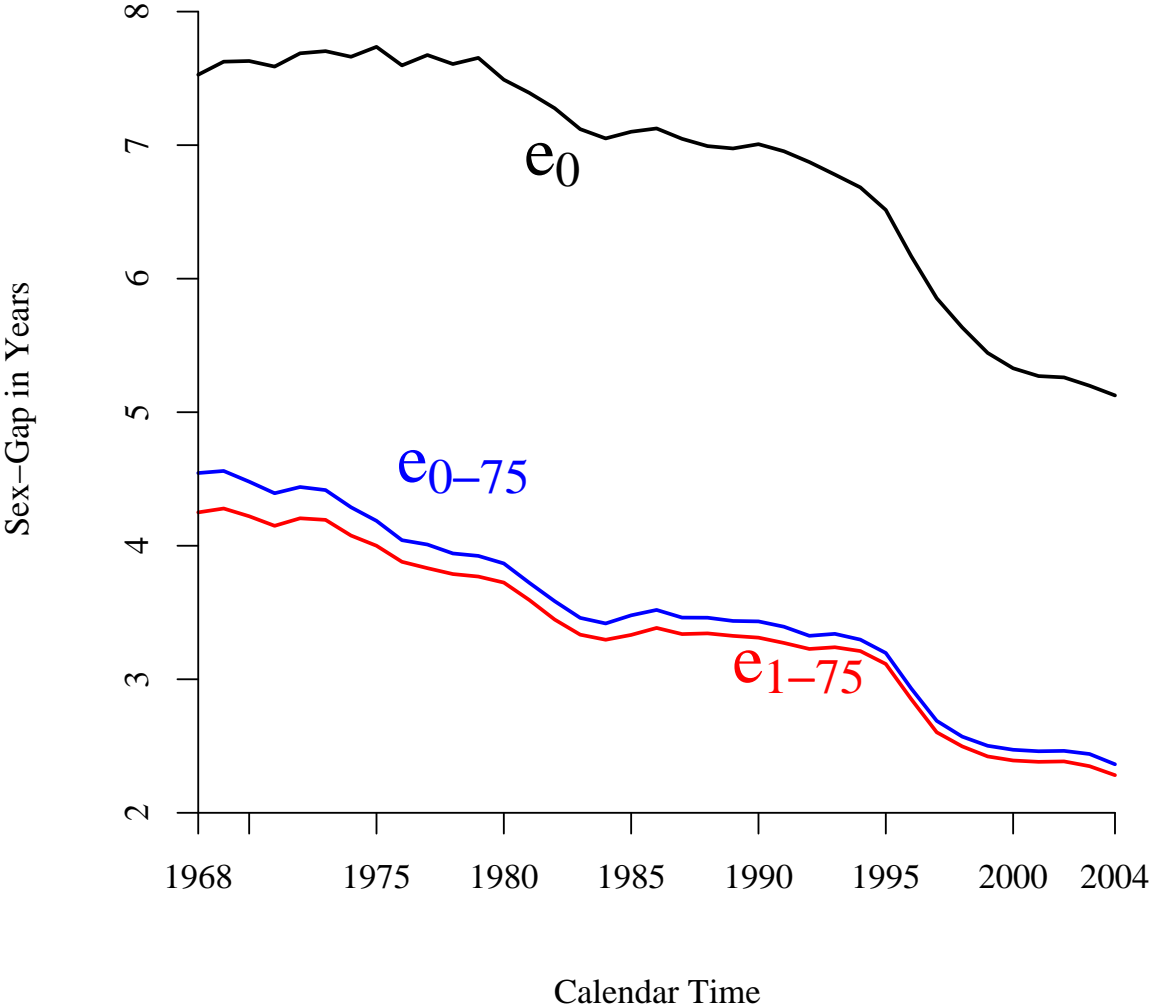
Cause	Women		Men		Total	
	Count	%	Count	%	Count	%
Breast Cancer	1,027,330	20.79	3,718	0.09	1,031,048	11.18
Prostate Cancer	0	0.00	409,970	9.59	409,970	4.45
Medical Care	4,941,881	100.00	4,276,540	100.00	9,218,421	100.00

Figure 1: Life Expectancy at Birth in the United States for Females (red) and Males (blue) and their difference every five years (grey bars), 1960–2004



Data-Source: Human Mortality Database

Figure 2: Sex-gap in life expectancy at birth and sex-gap in the mean number of years lived at ages 0–75 and ages 1–75, 1968–2004



Data-Source: Human Mortality Database

Figure 3: Cause specific contributions to the sex-gap in the mean number of years lived between ages 0-75 years and 1-75 years

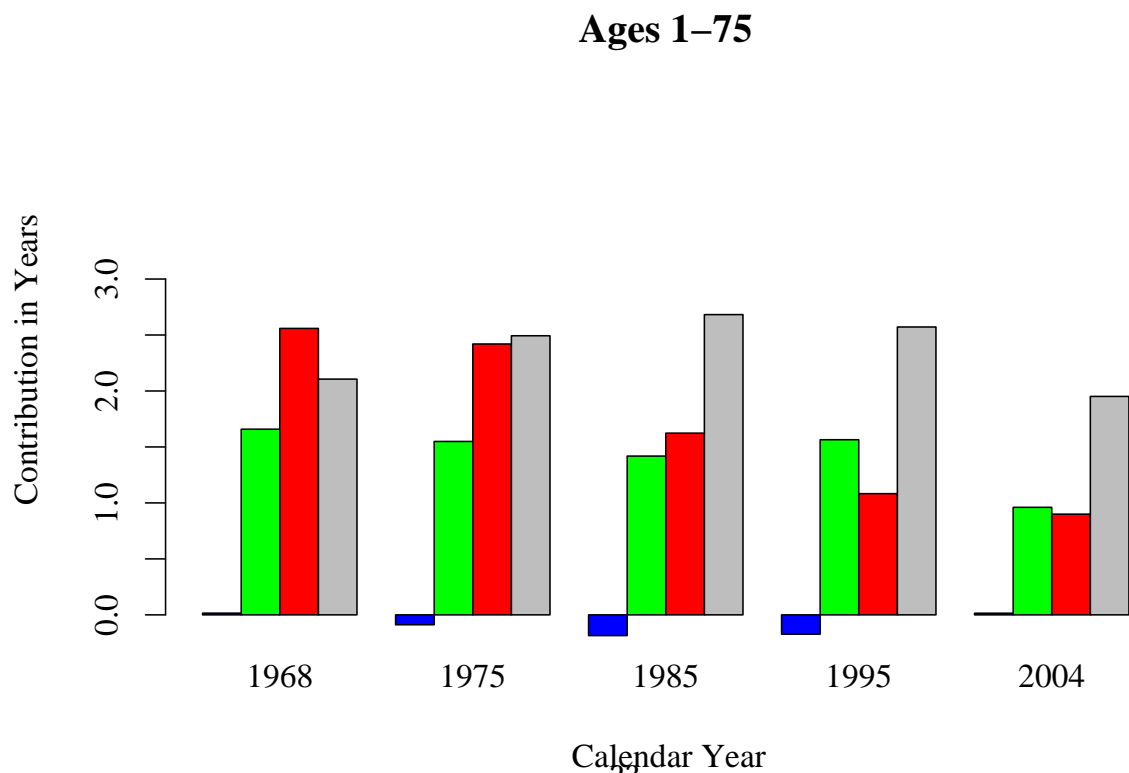
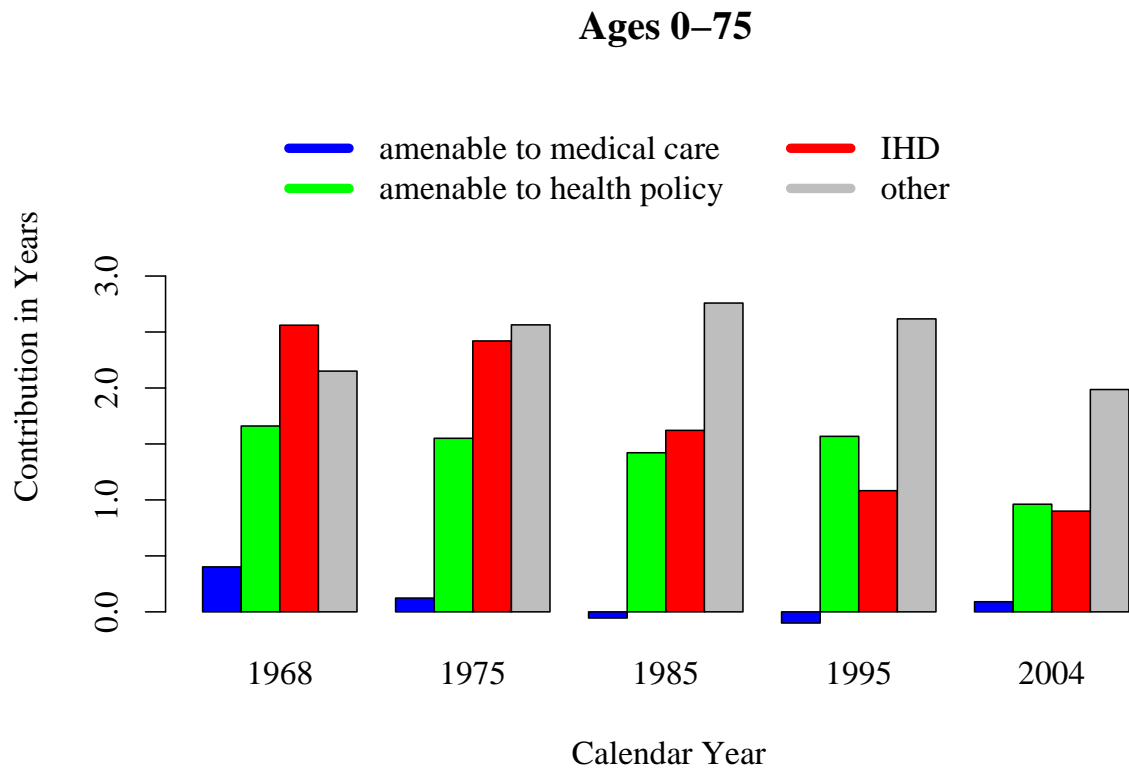
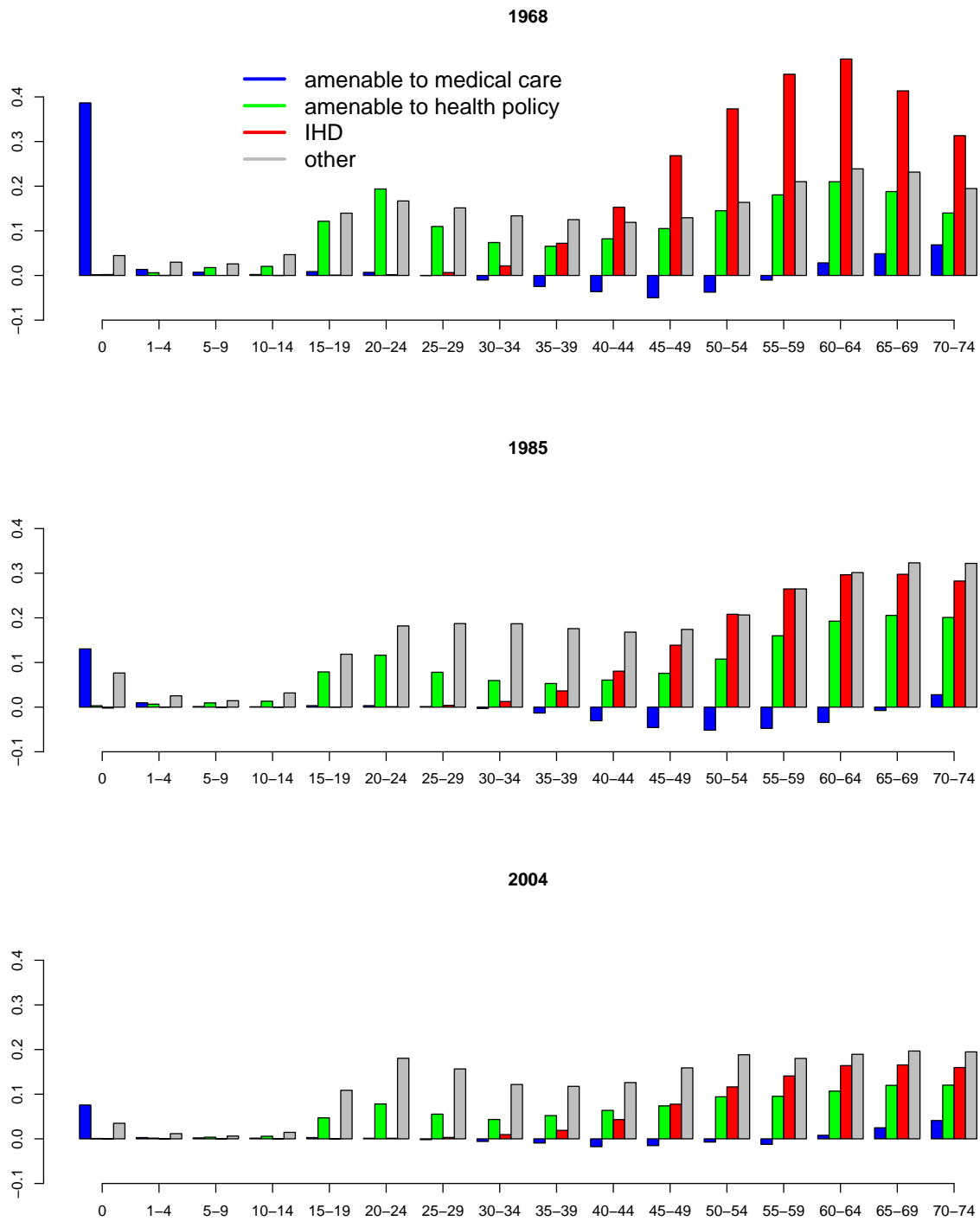
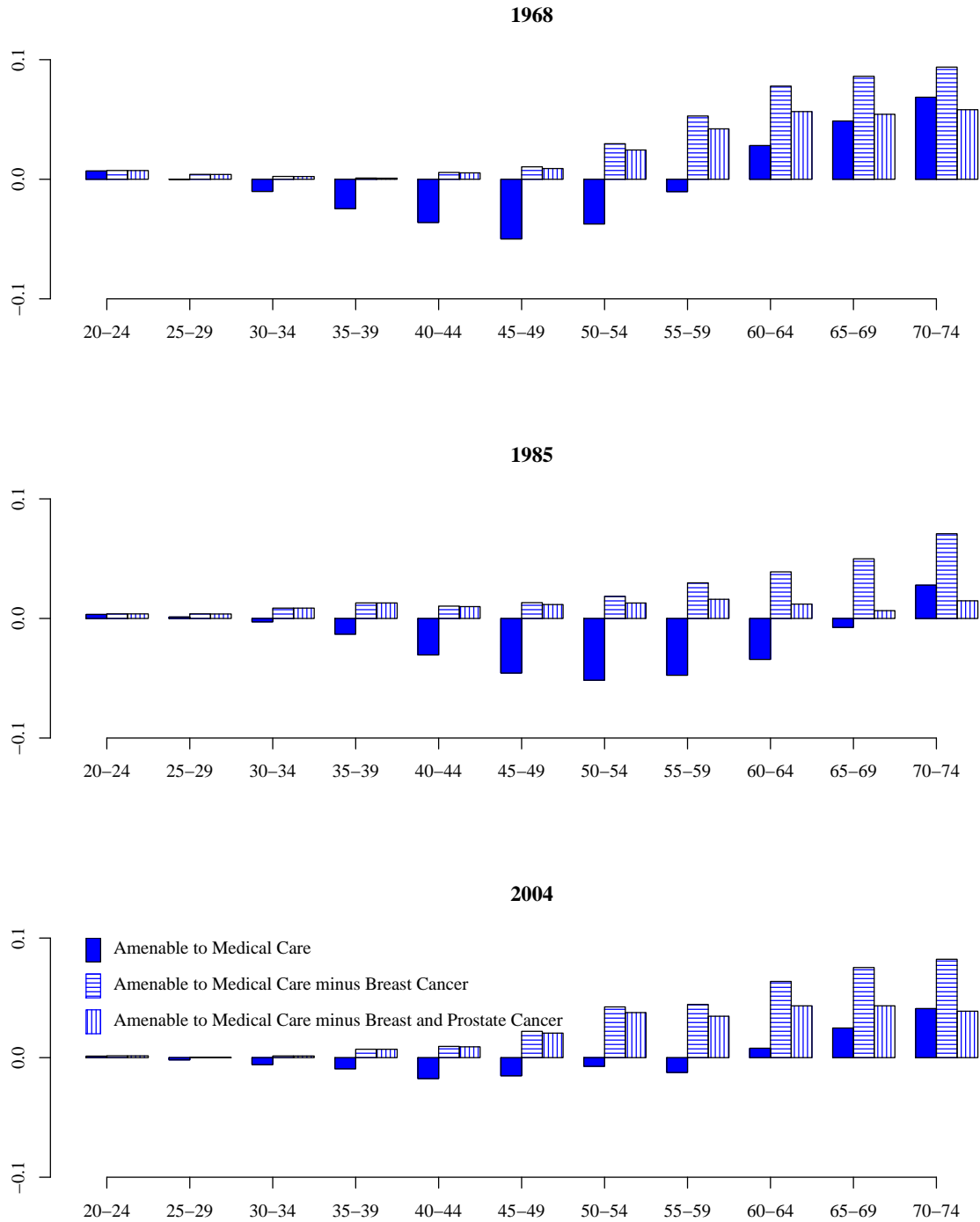


Figure 4: Age- and cause specific contributions to the sex-gap in the mean number of years lived between age 0 and 75 years, calendar years: 1968, 1975, 1995, 1985, 2004



Source: Authors' Estimations, based on data from the Human Mortality Database

Figure 5: Age- and cause specific contributions to the sex-gap in the mean number of years lived between age 0 and 75 years, causes amenable to medical care and causes amenable to medical care excluding breast cancer and prostate cancer, calendar years: 1968, 1975, 1985, 1995, 2004



Source: Authors' Estimations, based on data from the Human Mortality Database

Table 2: Contribution of causes amenable to medical care to the change in the sex-gap in the expected number of years lived between ages 0–75 years, including contribution of causes amenable to medical care minus breast cancer and prostate cancer

Group of causes	1968–1975	1975–1985	1985–1995	1995–2004
	Ages 0-75			
Preventable by public interventions	–0.110	–0.129	0.146	–0.606
IHD	–0.141	–0.799	–0.539	–0.183
Amenable to medical care	–0.279	–0.178	–0.045	0.190
- <i>Amenable to medical care minus breast cancer</i>	–0.252	–0.131	–0.086	0.114
- <i>Amenable to medical care minus breast and prostate cancer</i>	–0.269	–0.155	–0.092	0.156
Other causes	0.413	0.195	–0.142	–0.631
Total change, ages 0-75	–0.116	–0.911	–0.579	–1.230
	Age 0			
Preventable by public interventions	0.000	0.002	0.000	–0.003
IHD	–0.002	–0.003	0.002	0.000
Amenable to medical care	–0.175	–0.081	–0.058	0.003
Other causes	0.026	0.006	–0.031	–0.011
Total change, age 0	–0.151	–0.076	–0.086	–0.010
	Ages 1-75			
Preventable by public interventions	–0.109	–0.131	0.146	–0.604
IHD	–0.139	–0.796	–0.541	–0.183
Amenable to medical care	–0.104	–0.097	0.013	0.187
- <i>Amenable to medical care minus breast cancer</i>	–0.077	–0.050	–0.028	0.111
- <i>Amenable to medical care minus breast and prostate cancers</i>	–0.094	–0.074	–0.035	0.153
Other causes	0.387	0.189	–0.111	–0.620
Total change, ages 1-75	0.035	–0.835	–0.492	–1.220