Equity and length of lifespan are not the same

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Efforts to understand the dramatic declines in mortality over the past century have focused on life expectancy. However, understanding changes in disparity in age of death is important to understanding mechanisms of mortality improvement and devising policy to promote health equity. We derive a novel decomposition of variance in age of death, a measure of inequality, and apply it to cause-specific contributions to the change in variance among the G7 countries (Canada, France, Germany, Italy, Japan, the United Kingdom, and the United States) from 1950 to 2010. We find that the causes of death that contributed most to declines in the variance are different from those that contributed most to increase in life expectancy; in particular, they affect mortality at younger ages. We also find that, for two leading causes of death [cancers and cardiovascular disease (CVD)], there are no consistent relationships between changes in life expectancy and variance either within countries over time or between countries. These results show that promoting health at younger ages is critical for health equity and that policies to control cancer and CVD may have differing implications for equity.

health equity | age of death variance | life expectancy | inequality | mortality

The 20th century was an epoch of unprecedented declines in mortality rates in most countries. Causes of death shifted from infections, which primarily killed children, to cardiovascular disease (CVD) and cancer, which primarily killed adults, in the epidemiological transition (1, 2). Understanding mortality change largely focused on understanding changes in the average age of death, the life expectancy (e_0). In recent years, attention has shifted to disparities in mortality, and health equity is now recognized as an important policy goal. Here, we develop and use a new method to show how changes in inequality, as measured by the variance V, are driven by changes in mortality caused by specific causes, accounting for the interdependence of changes in the different causes of death. We find differences between the magnitudes of cause-specific contributions to e_0 and V and unexpectedly inconsistent relationships between the two over time and across countries, with important scientific and policy implications.

Work to date on understanding change in e_0 has found that, among wealthy nations, much of the gain in the second half of the 20th century was because of declines in CVD mortality (3–5). The male–female difference in e_0, which widened and then, narrowed over the course of the century, has also been driven by CVD mortality as a consequence of smoking behavior (6–8). Disparities across countries in e_0 have been studied most notably by the Global Burden of Disease Project, which has noted gains to life expectancy across countries and declines in global disparity for certain age-specific mortality rates (9, 10). Related work has shown how changes in different causes of death would have different effects on the health of global poor vs. rich (11).

Analyses of all-cause mortality within countries show that variance, V, in age of death (i.e., inequality) falls if mortality declines at ages below a well-defined threshold age but increases if mortality declines at older ages (12). Remarkably, as life expectancy has increased, within-country disparity in lifespans has decreased substantially (modestly in a few countries, including the United States) (13, 14). However, cause-specific contributions to variance have only recently been explored. A study of the difference between blacks and whites found that causes of death contributing the most to differences in V were distinct from causes contributing most to the difference in e_0 (15). In particular, whereas previous work showed that about one-half the gap in e_0 was because of differences in CVD, cancer, and diabetes-related mortality (16), the gap in V was largely because of accidents, violence, and HIV/AIDS.

Given the trend toward declining variance in age of death across high-income countries, it is important to ask whether cause-specific contributions to declining variance are broadly similar across countries, with some possible differences in timing. Also, do causes that contribute substantially to increase in e_0 also contribute in a major way to declines in V? The work by Firebaugh et al. (15) shows that this need not be true.

Previous investigations of cause-specific contributions to variance in age of death have relied on a linear (ANOVA) decomposition to partition the variance by cause. However, that decomposition of the variance assumes that causes of death are independently distributed within a life table. Cause-specific death distributions are necessarily correlated: the probability of dying at a given age from a particular cause is the product of the age-specific death rate from only the cause of interest and survival up to that age, which depends on all causes of death. Formally, the probability density of death at age x from cause k in a set of causes C, $f_k(x)$, is

$$f_k(x) = \mu_{k,x} \prod_{k \in C} l_k(x), \quad [1]$$

where $\mu_{k,x}$ is the mortality rate from cause k at age x, and $l_k(x)$ is the cumulative probability of survival from birth to age x if only cause k was operating. Thus, it is not possible to separate the distribution of death from a particular cause from mortality due to other causes, and an alternative approach should be considered.

We have derived a decomposition of the difference in variance between two distributions, which allows us to determine how much each cause contributes to this difference (*SI Appendix*). We use the Kitagawa decomposition to consider separately each

Significance

We find that the causes of death that have led to greater equality among lifespans are different from the causes that have led to longer average lifespan, also called life expectancy. Control of leading causes of death, such as heart disease, increased life expectancy, whereas medical interventions on infant mortality led to greater equality. Action to promote health equity will require further mitigation of the killers of young people rather than solely focusing on the most common causes of death.

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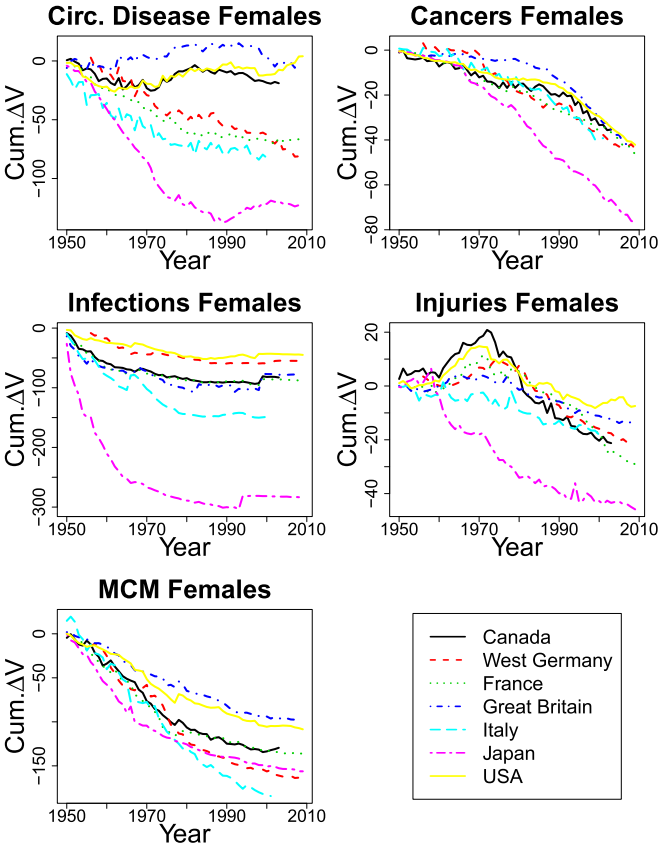

Fig. 1. Cumulative cause-specific contributions to change in variance in the age of death (V) in females.

cause's effects on age-specific mortality rates and cumulative survival, and then, we add these to produce the cause's total effect on the difference (17, 18). Our result makes it possible to compare variances in age at death in any two populations; our derivation extends to variances a similar simpler decomposition of the difference in life expectancy (16, 17). This method is analytically precise while remaining simple to implement. Here, we apply these methods to assess change within national populations over time.

Data for the G7 countries (Canada, France, Germany, Italy, Japan, the United Kingdom, and the United States) were obtained from the Human Mortality Database (HMD; www.mortality.org) and the World Health Organization (WHO) Mortality Database (www.who.int/healthinfo/statistics/mortality_rawdata/en/) to produce age-, sex-, and year-specific mortality rates for each cause. We considered five broad categories for causes of death to aid with stability across versions of the International Classification of Diseases: maternal and child mortality (MCM), infections, and injuries, which primarily affect younger individuals, as well as circulatory disease and cancers, which primarily affect older individuals as well as the residual. Using our decomposition, we present the first long-term estimates, to our knowledge, of cause-specific changes in V and their associations with cause-specific changes in e_0.

In agreement with previous work on life expectancy change, we find that the main contributor to change in e_0 is CVD as well as infectious diseases in the case of Japan followed by improvements in maternal and child health (*SI Appendix*, Figs. S1–S3). There are two noteworthy sex differences: males see roughly twice the cumulative gain in e_0 from injury-related mortality than females, and males see some substantial, although temporary, net declines in e_0 from cancers. These sex differences are not surprising, because men were more prone to injuries and smoked more heavily than women in these countries.

The cumulative changes in the variance by cause are presented in Figs. 1 and 2, with residuals shown in *SI Appendix*, Fig. S3. The great contributor to decline in V was MCM, which primarily consists of infant deaths in the neonatal period. Mortality change from infections and injuries, which typically affect younger ages, also led to decreases in variance over time. Some countries show a slight increase in the variance for mortality from infections among males in the mid-1980s and at the end of the series in the 2000s. The former is the result of the HIV/AIDS epidemic, which killed many young men. The latter may be the result of efforts against pneumonia and hospital-acquired infections, which tend to affect older individuals within developed countries, such as the G7, although such an effect is not seen among females. Despite typically causing death at older ages, cancer mortality likewise led to declines in V across the countries considered. However, circulatory disease, an important cause of increase in life expectancy in every country, has quite distinct effects on changes in variance in different countries: in continental Europe and Japan, changes in circulatory disease mortality led to net reductions in variance, whereas they led to little net change and even some increases in V among the Anglophone members of the G7.

Thus, within a country, declines in different causes of death had quite different effects on V, and across countries, the direction of effects also varies. Causes of death that are important at younger ages have most clearly led to declines in variance, and it is remarkable how important declines in MCM have been to decreasing V, although they have made only modest contributions to increases in e_0. Cancer, which is typically important at older ages, has nonetheless led to net reductions in V. It is

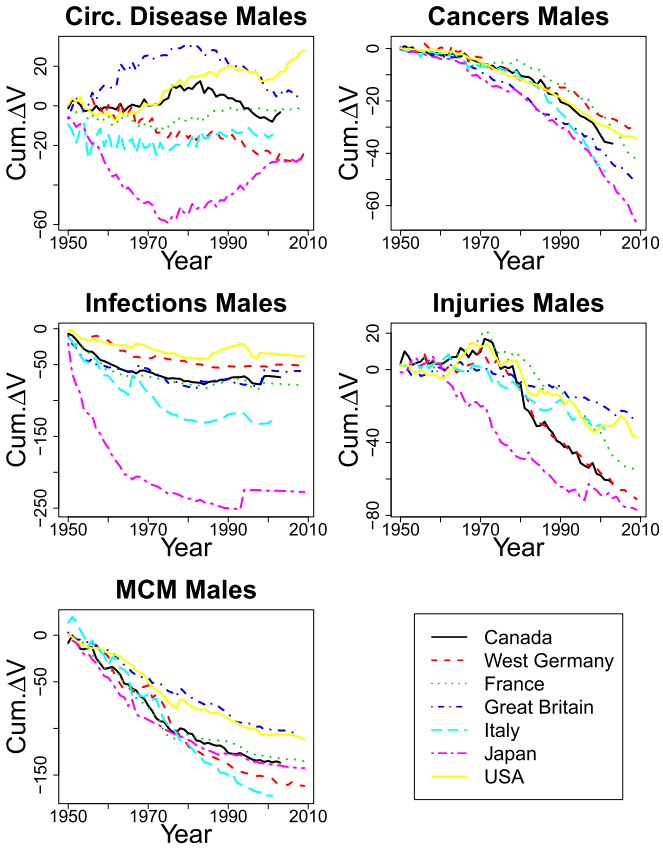

Fig. 2. Cumulative cause-specific contributions to change in variance in the age of death (V) in males.

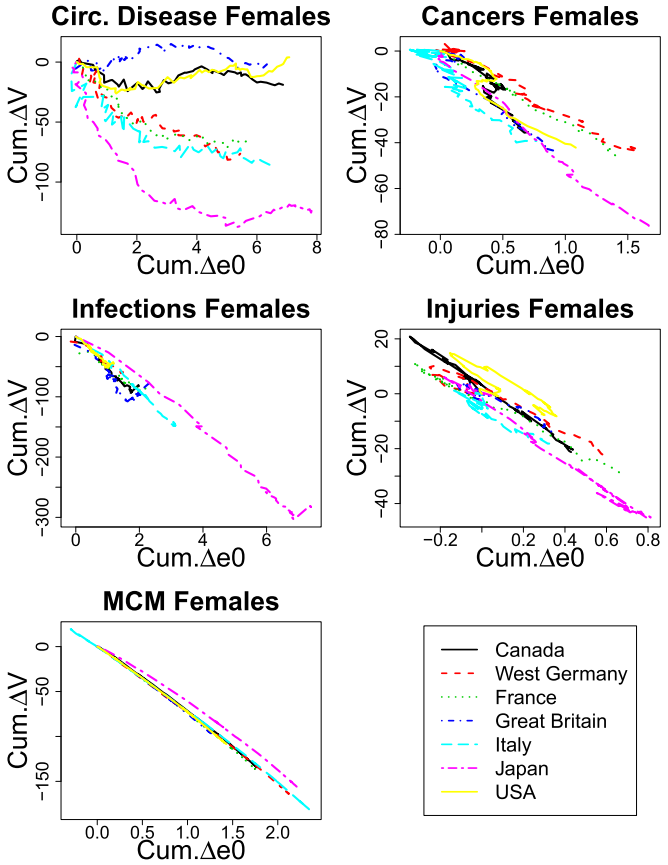

Fig. 3. Trajectories of cumulative cause-specific contributions to change in life expectancy (e_0) and variance in the age of death (V) in females.

possible that the specific cancers that have led to bigger gains in e_0 may be different from the cancers that led to declines in V, which would explain this discrepancy.

We explore the association between contributions to e_0 and V by plotting trajectories of cumulative change in these measures in Figs. 3 and 4. For MCM, infections and injuries increase in e_0 goes together with decrease in V, because many or all of these deaths occur below the threshold age. Cancers in males and circulatory disease in both sexes, however, show very different trends. In the case of cancer among males, changes in cancer mortality initially led to lower e_0 because it decreased V, suggesting increases in mortality rates at older ages, possibly the result of the epidemic of tobacco use that began in the 1920s. Later, there are increases in both e_0 and V because of changes in cancer mortality. In the case of circulatory disease, there is a wide diversity of e_0–V trajectories, particularly among males. In the United Kingdom, for example, there are increases in V with little change in e_0 initially, whereas in the United States, there is a gradual increase in V along with e_0, creating an N shape in the trajectory. In Japan, by contrast, there are steep declines in V and increases in e_0, with some increases in V toward the end, creating a U shape. For the other countries, the effect is more of a straight line.

These trajectories illustrate the relationship between threshold age and changes in V and e_0 because of particular causes of death. The causes that both show consistently declining mortality rates and occur at young ages have a tight association between change in e_0 and change in V; the former will always increase in response to declining mortality rates, and the latter is responding to declines below the threshold age. By contrast, causes of death that occur closer to the threshold age show widely differing effects on V depending on precisely where the changes in rates occur around that age. This finding suggests that there is ample room for policy to affect equity in longevity.

The unexpected diversity of e_0–V trajectories for circulatory disease is driven by differences in either the threshold age (the age above which declines in mortality rates will increase V and below which declines in mortality rates will decrease V) or the age pattern of mortality decline. If the age pattern of mortality decline in circulatory disease was identical across countries, a lower threshold age would lead to different directions of change in V. However, whereas the United States has the lowest threshold age of the G7 countries, the United Kingdom and Canada are similar to other European countries (*SI Appendix*, Fig. S4). By contrast, the age pattern of mortality change for circulatory disease differs among the G7 countries, particularly at the oldest ages, where the United States, Canada, and the United Kingdom have seen much steeper declines than the other G7 countries (*SI Appendix*, Fig. S5). This dramatic decline in circulatory disease mortality at the oldest ages is responsible for little net change or even increases in V from circulatory disease among these countries.

Discussion

Although mortality rates from all causes of death have been decreasing among the G7 countries, the magnitude of their effects on V is different from that of their effects on e_0. Furthermore, the directions of these effects are surprisingly diverse over time, across the G7 countries, and likely, among other high-income countries; low- and middle-income countries will likely have different patterns using this method. Circulatory disease in particular, which affects those at older ages, had very different effects on inequality across countries because of different age

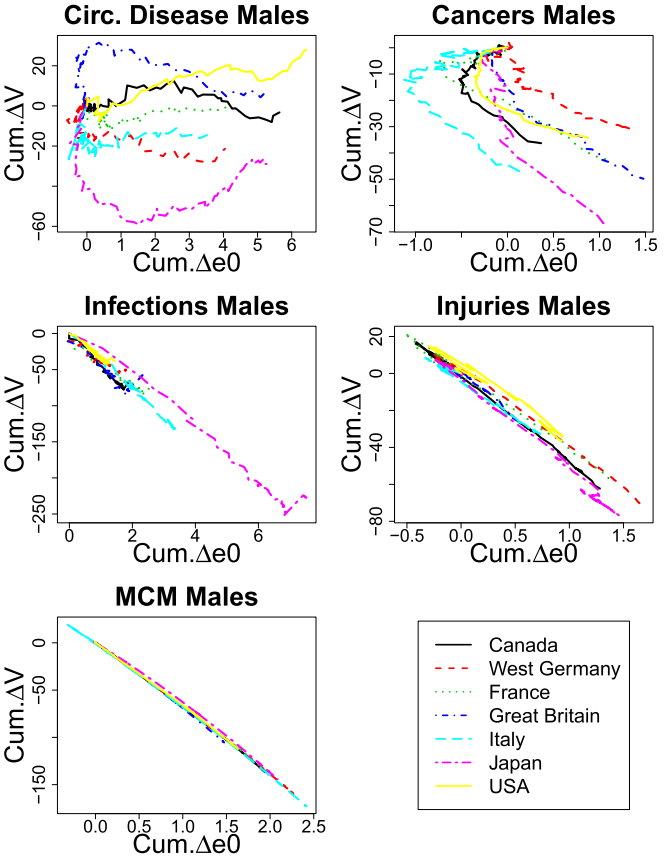

Fig. 4. Trajectories of cumulative cause-specific contributions to change in life expectancy (e_0) and variance in the age of death (V) in males.

patterns of mortality decline, although we did not systematically consider contextual factors in these results. Most research on the relative contributions of treatment of CVD and prevention suggests that these two components contributed equally to declines in cardiovascular mortality rates (19, 20). However, it is difficult to clearly define which interventions are preventive as opposed to treating disease, and the age patterns of their effects are unclear. Differences in the age-specific effects of treatment and prevention on mortality rates may explain the cross-country differences in the mortality decline from CVD and consequent change in variance. Furthermore, this result may shine a light on how policy can improve health and longevity equity.

This method accounts for one variety of correlation among causes of death that arising from the interdependence of cause of death distributions. Another variety of correlation is that of competing risks (that risk of death from one cause is correlated with risk of death from other causes). Thus, a hypothetical elimination of one cause of death would lead to increased mortality rates from other causes, because competition is reduced. Our method, as with other multidecrement life table methods, is not able to account for competing risks. This issue should not, however, affect the results of longitudinal comparisons where the competing risks are accounted for in the changes over time.

Although this study explores one dimension of inequality, there are many others that are relevant to health, including wealth and education. Within the United States, there has been a growing gap in life expectancy among income strata (21). However, there are inconsistent associations of income inequality with life expectancy (22, 23) and variance in age of death (13). Understanding the relationships among various types of inequality will help identify mechanisms and possible interventions to promote health equity.

These results illuminate what has produced the greater equity among lifespans that has accompanied their growing average. More people were able to enjoy the life expectancy gains from declining CVD mortality thanks to continued progress against child mortality, infections, and injuries. For policymakers, focusing solely on leading causes of death, which typically affect the elderly, may be to the detriment of health equity. Action to mitigate the killers of younger individuals is important to ensuring that the fruits of longer lives remain accessible to all people.

Materials and Methods

Mortality data on the G7 countries since 1950 were taken from the HMD. For Germany, only West German data were considered. Proportions of deaths by cause were calculated using data from the WHO Mortality Database and multiplied with the appropriate HMD mortality rates to produce age, year, sex, and cause-specific death rates. Details on the causes of death assigned to each broad cause category and the derivation of the variance decomposition are given in *SI Appendix*. This study was exempted from institutional review board review, because it used publicly available aggregate data that had been previously published.

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