Commentary: Do older men and women gain equally from improving childhood conditions?

Eileen M Crimmins* and Caleb E Finch

The relationship between early and late mortality within cohorts has been well-established.1–4 Catalano and Bruckner add evidence that birth cohorts subjected to higher mortality ‘than expected’ before age 5 have higher mortality ‘than expected’ after age 5.5 Their contribution employed evidence from three countries to document a link between cohort mortality at younger and older ages. Methods developed for time-series analysis were used to correct for trends, cycles, and autocorrelation. After eliminating the effect of a common trend, this analysis further supports the link between early and late life mortality in cohorts. A novel aspect finding is a sex difference: the link between mortality before age 5 and later age mortality is stronger for men than for women. The authors suggest that this might be due to the ‘relative resiliency’ of females compared to males across the ages.

It is interesting to speculate on the mechanisms that might underlie sex differences in the relationship between early life circumstances and later health. Three mechanisms have been suggested to explain the link between mortality across the lifecycle in historical cohorts: (i) improvements in physiological status linked to improved nutrition emphasized by Fogel and associates; (ii) developmental effects associated with improvements in nutritional intake of mothers are the focus of Barker and colleagues; (iii) the role of reduced infection and related inflammation over the lifecycle has been emphasized by Finch and Crimmins.3,4,6,7 One could hypothesize that an increase in scarce nutritional resources might be shared unequally by sex. Although inequitable access to food could differentially improve cohort early mortality, this should not affect the relationship between early and late mortality within sex groups. On the other hand, maternal well-being appears to affect the development of lipid risk profiles and hypertension more in males than females, which would lead to a stronger link between early and late mortality among men during the phase of improving maternal nutrition.8 However, men are less resistant to infection and have milder inflammatory responses.9,10 Changes in exposure to infectious conditions thus might affect men more than women; but the reduction in inflammation might have been greater in women leading to a stronger link between early infection and late life mortality among women. The sum of these explanations leads to no clear expectation of a difference by sex in the relative strength of the link between changes in early life health and late life mortality, but they do provide plausible reasons why differences might be observed.

Another question raised by the findings of Catalano and Bruckner is whether the relationship of early to late mortality within cohorts would show sex differences if not detrended for autocorrelations as in their study. We proposed that one explanation for the relationship between cohort mortality across the age range is that high levels of infection and inflammation earlier in life are causally related to the development of the chronic conditions causing mortality in old age.3,4 Our explanation implies associations of the trends, rather than deviations from the trends in cohort mortality. This hypothesis has also led us to focus on the relationship between mortality in childhood and at specific older ages for cohorts, rather than across the entire lifespan by examining the association between the probability of dying in old age (70–74) on mortality up to age 15 for birth year cohorts born in individual years up to 1900 in Sweden, France, England, and Switzerland (Table 1). The number of cohorts with available data ranges from 24 in Switzerland to 149 in Sweden. Regression of the probability of dying at age 70–74 on the probability of dying in each of four childhood age groups indicates that the trends in cohort old-age mortality follow very closely those of childhood mortality for both women and men in these four countries. Linear trends in cohort mortality are virtually the same in childhood and in old age for these cohorts as evidenced by an $R^2$ close to 1.0. The higher $R^2$ for women than for men in these four countries indicates that female old-age mortality has a somewhat stronger association with childhood mortality in Sweden and France and has a much stronger association in England and Switzerland. In none of these cases did men show a stronger linear association between trends in cohort childhood and late life mortality. Our findings are in concert with the evidence for cohort patterns of mortality change over the past 50 years showing that the cohort mortality of females is better characterized by linear cohort change, while male cohorts appear more affected by both non-linear period and cohort factors.11

Of course, sex differences in relationships of mortality across the lifecycle are likely to be affected by the time period studied, the ages compared, and the causes of mortality responsible for change. Mortality of male cohorts after age 5, as used in the Catalano and Bruckner analysis, is likely to be affected by wars and accidents. More recent male cohorts have suffered more from the effects of cigarette smoking than females.

In sum, evidence has accumulated for the role of early experiences in affecting later life health. Two potential mechanisms could play some role in sex differences in the relationship of exposure to infection at young ages and its effect on later
Independent variables:

\[ q \]

Source

However, we have presented evidence that the cohort pattern of mortality decline for cohorts born before 1900 appears stronger, or at least as strong, for women as for men. Once nutrition of mothers and children is substantially improved, and exposure to infection is reduced, and treatment of infectious conditions through antibiotics is routine, the mechanisms linking early and late life in historical times will become less relevant.

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References


Table 1 Regression of childhood mortality on mortality at age 70–74 for cohorts (by year of birth): males and females

<table>
<thead>
<tr>
<th>Female</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>0.0029 (0.00751)</td>
</tr>
<tr>
<td>( q_{(0-1)} )</td>
<td>0.54736 (0.08836)***</td>
</tr>
<tr>
<td>( q_{(1-4)} )</td>
<td>0.63226 (0.09566)***</td>
</tr>
<tr>
<td>( q_{(5-9)} )</td>
<td>1.12065 (0.16070)***</td>
</tr>
<tr>
<td>( q_{(10-14)} )</td>
<td>1.78175 (0.33692)***</td>
</tr>
<tr>
<td>( N )</td>
<td>149</td>
</tr>
</tbody>
</table>

\( b \) = regression coefficient; SE = standard error of coefficient; \( q_{(0-1)} \) = probability of dying age 0–1; \( q_{(1-4)} \) = probability of dying age 1–4; \( q_{(5-9)} \) = probability of dying age 5–9; \( q_{(10-14)} \) = probability of dying age 10–14. Dependent variable: \( q_{(70-74)} \). Independent variables: \( q_{(0-1)}, q_{(1-4)}, q_{(5-9)}, q_{(10-14)} \).