Aging in the context of cohort evolution and mortality selection in India

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Abstract

The present research is aim to examines the ageing in the context of mortality selection in India. The study uses two indicators of ageing: demographic ageing and biological ageing. In order to examine the effect of early life mortality selection on ageing, I plotted the age-specific mortality rate from ages 0-1 to 80-84 for selected Indian cohorts born during 1900-2005. Finding shows that later cohort have lower young-age mortality rate and lower old age mortality rate than earlier cohorts, which support the cohort evolution hypothesis. Moreover, I observed that in spite of lower young-age mortality of later cohort, the rate acceleration of mortality after age 40 is steeper for later cohort than earlier cohort, which indicates the presence of mortality selection mechanism. Preliminary findings on effect of mortality selection encouraging me to examines whether the current old age mortality is real or artifact of mortality selection.

Introduction

In the past century, the life expectancy has increases about than triple from 23 years in 1901 to approximately 66 or more years by 2016 in India. The epidemiological transition is thought to be key mechanism behind the increase in human life expectancy in each period. In contrast to this theory, other theories emphasise change over cohorts rather than across periods. One such theory known as "cohort morbidity phenotype (CMP)" suggest that cohort experiencing lower exposure to infection and inflammation during early childhood reap lower mortality risk later in their lives. Another theory known as "technophysio evolution (TE)" argues that later cohort are endowed with better health capital at birth and thus enjoy lower rates of health capital depreciation over the life course. Although theories of CMP and TE have been supported by much evidence, neither theories takes into account possible changes in patterns of mortality selection (MS), which is another mechanism linking early life circumstances to health and mortality in later life. According to cohort evolution (CE) theories later cohort experience lower risk of infection and inflammation and have better nutrition and health capital during childhood, so a smaller proportion of frail individuals are selected out of the population during young age. This, in turn, would cause a larger proportion of frail individuals to survive into old age and would increase the cohorts overall mortality risk at older ages. In this case risk of old age mortality may be potentially higher for later cohorts than earlier cohorts. If selection of frail individuals out of the population at younger ages has indeed declined across cohorts, CE theories may not explain the decline in mortality. Therefore, CE theories should be supplemented with a comprehensive investigation of the changing pattern of MS across cohorts.

The present research is aimed to analyse the "Aging in the context of cohort evolution and mortality selection in India". The rationale behind the present study in the Indian context is based on several reasons. First, a few Studies from India have reported that the regions/districts with a high conditional probability of dying early life did not have a high conditional probability of dying at adult and older ages. This low level of correlation between early life mortality and later life mortality can be explained by mortality selection, i.e., a majority of the frail individual died during early years of life among regions with high mortality which leads to healthier individuals to survive in later ages and hence reduces the mortality at later ages. Therefore, a comprehensive investigation is required to understand the effect of mortality selection during early life on

mortality at later ages. Therefore, on the basis of existing mortality scenario in India, the present study is trying to investigate the process of aging in the context of mortality selection in India. I tested following hypothesis to examine the role of cohort evolution mechanism.

Hypothesis 1a: According to cohort evolution theories, young-age and old-age mortality rates are positively correlated across cohorts.

Hypothesis 1b: According to the cohort morbidity phenotype theory, the acceleration of mortality during aging (e.g., after age 60) should be constant across cohorts: that is, it is not affected by young-age mortality rate.

Theory of population heterogeneity, mortality selection

Theory of population heterogeneity suggest that population are composed of individuals or subpopulations, where individuals have different susceptibilities to mortality that are fixed at birth. Frailer members of a cohort die at younger ages leaving a group of strong members at later ages. This resulted in deceleration of mortality at later ages.

For example, consider two population subgroups having the same distribution of frailty, but one is subject to more adverse conditions in early life then frailest members are eliminated more quickly than among the advantaged group. Therefore, it is possible that subpopulation having adverse condition during early life may have lower mortality than advantage group at advanced age (Manton et al., 1979; Olshansky, 1995). The present study aims to investigate how population heterogeneity may affect the mortality acceleration pattern very late age (e,g, age of 86). We have excluded individual above age 85 due to fact that reduction in mortality among very old age population may be underregistartion of deaths or uncertainty in age reporting among old age population (Crimmins and Finch 2006; Gavrilov and Gavrilova 2011). Following hypothesis were tested for mortality selection mechanism.

Hypothesis 2a: According to the theory of heterogeneity and SM model, young-age mortality rate and mortality acceleration (i.e., rate of demographic aging, up until very late age: e.g., age 95) are negatively correlated across cohorts.

Hypothesis 2b: According to the theory of heterogeneity and SM model, the rate of biological aging is fixed across cohorts.

Data and Methods

The present study aims to examine the cohort trend in rate demographic aging and biological aging in India. In order to compute the cohort trend in demographic aging and biological aging, we need to have life table for several birth cohorts. Cohort life table for the year 1901 to 1941 were directly taken from earlier paper by Bhargava et al (2015). For the birth cohort between 1951 to 2005, we used period life developed by United Nation. Notably, United Nation provide estimated life table for the period 1951-2017 and also projected life table from the year 2018-2100. Using these estimated and projected period life tables we have created life tables for the cohort born between 1950-55 to 2000-2005.

Methods

The cohort trends in rates of demographic aging (i.e., rate of mortality acceleration) and biological aging will be estimated by methods developed by Gompertz (1825) and Strehlar and Mildvan (1960) respectively.

The Gompertz law of mortality can be expressed as:

$$R_t = R_0 e^{\alpha t}$$

Where R_t is the mortality rate at age t, R_0 is the initial mortality rate and α refers to the rate of demographic aging or mortality acceleration. The theory posits that the initial mortality rate

The above equation can be written as:

$$\ln(R_t) = \ln(R_0) + \alpha t$$

The term $\ln(R_t)$ and α represent the intercept and slope of the log mortality curve, respectively. Then we calculate the biological aging coefficient B from age 50 to 84, using the methods as developed by Strehler and Mildvan (1960):

$$\ln(R_0) = 1/\mathrm{B}\alpha + \ln(K)$$

where *B* is the fractional loss each year of original vitality, and *K* denotes the frequency of environmental variations. In other words, if a later cohort has a lower initial mortality rate than a preceding cohort, it should experience greater acceleration in mortality (i.e., α) over the life course.

Following aforementioned procedure, we obtain two estimated parameters for each cohort state: α , the slope of the log mortality curve (i.e, mortality acceleration) and coefficient B vitality attrition

(i.e, rate of biological aging) from age 50 to 84 and K is the frequency of environmental variation. We also calculate the mortality acceleration from ages 20, 25, 35, 35, 40, 45, and 50 to 84 for the purpose of testing mortality selection hypotheses.

Results

The association between early life mortality and later life mortality are presented in Fig 2. Figure to describes the age specific mortality rate for birth cohort 1900-2000 for India. Finding shows that later cohorts have lower young age mortality also have lower old age mortality as earlier cohort, which is similar to Panel B of figure 1 and support the cohort evolution mechanism. Moreover, we have also observed that rate of increase in mortality after age 50 was steeper for later cohort than earlier cohort, which support mortality selection mechanism as presented in Panel B of Fig 1.

In order to test whether these associations are statistically significant, we regress the old age mortality and rate of mortality acceleration on young age mortality rates. Results obtained by regressing the old age mortality on early life mortality are presented in Table 1. Results shows that old age mortality rates between 60-84 are positively correlated with early life mortality rates 0-1 to 10-14. In other words, we can say that results from Table 1 support the hypothesis as derived from cohort evolution theories.

Results obtained by regressing the rate of mortality acceleration (e.g, rate of demographic aging) between ages 30 to 84 on early life mortality rates between ages 0-1 to 10-14 are presented in Table 2. Results shows that early life mortality are negatively associated with rate of mortality acceleration between ages 35 to 74. In other words, cohort having higher rate of early mortality have lower rate of mortality acceleration at later ages, which support the hypothesis 2a derived from theory of population heterogeneity and strehler and mildwan. Thus, findings from Table 1 and Table 2 indicates that cohort evolution and mortality selection mechanisms exist together as presented in panel 2 of Figure 1.

Further, as suggested by cohort morbidity phenotype theory, we tested whether rate of mortality acceleration (e.g, rate of demographic aging) is constant after age 60 as mentioned earlier in hypothesis 1b. Rate of demographic aging after age 60 for cohort born during 1901 to 2005 are

presented in Figure 3. Figure 3 shows that rate of demographic aging is lower for earlier cohort that subsequent birth cohort. In other words, cohort having more adverse condition during early life have lower rate of demographic aging after age 60 than cohort having better condition during early life.

Notably, mortality selection, mechanism suggest that rate of demographic aging at older ages should be negatively associated with early life mortality. Alternatively, cohort having higher rate of early life mortality should have low rate of demographic aging and a cohort having lower rate of early life should have higher rate of demographic aging. Therefore, we have plotted the trend in rate of demographic aging between age 60 and 84, along with trend in logarithm of infant and child mortality rate for birth cohort 1900-2005 as presented in Figure 4. Finding shows that infant mortality and child mortality decreases across cohort, however at the same time rate of demographic aging increases for respective cohort. To examine the statistical association between early life mortality and rate of demographic aging, I regressed rate of infant and child mortality on the rate of demographic aging. Results of regression analysis are presented in Table 3.

Conclusions

The present study investigates the aging process in the context of cohort evolution and mortality selection mechanisms. Using data from Indian Census and Life table generated by United Nation on period age specific mortality rates among in India between 1900-2100, this study begins by testing cohort evolution theories and mortality selection mechanism. Finding of this study indicates a positive correlation between young- and old-age mortality rates, as predicted by cohort evolution theories; they also show a negative correlation between young-age mortality rates and mortality acceleration in late life, as predicted by mortality selection mechanism.



Figure 1. Relationship between cohort evolution and mortality selection mechanism

Source: Hui Zheng (2012)



Figure 2. Logarithm of Age-specific mortality rate for the cohort born during 1900 to 2010 India.

Fig. 3. The trend of rate of demographic aging between age 55 and 84 (α 55–84) across cohorts. α 70–94 represents the rate of demographic aging from age 55 to 84. α is calculated using the equation $\ln(Rt) = \ln(R0) + \alpha t$, where age-specific mortality rates *Rt* are available from the data. α is the slope of the log mortality curve





0.1 0 0.09 -1 0.08 Rate of demographic aging -2 0.07 0.06 -3 Ln(Rt) 0.05 -4 0.04 Log of infant mortality rate 0.03 Log of child mortality rate -5 Rate of demographic aging between 55 0.02 and 85 -6 0.01 2000,205,2019 194550 1957 0 -7 1965-1970 1980-1985 1915-20 1960-1965 1970-1975 1975-1980 1985-1990 1990-1995 1995-2000 1925-30 1940-45 1955-1960 1920-25 1900.05 1905.1910.15 1930-35 1935-40

Fig 4. The trend of rate of demographic aging between age 55 and 84 (α 55–84), log of infant and child mortality rates, in India across cohorts 1900-2010

Fig. 5. The trend of rate of demographic aging (α 55–84) and biological aging between age 55 and 84 across cohorts. *B*55–84 is calculated using equation $\ln(R0) = (-1/B) \alpha + \ln(K)$ by assigning a value of *K* (*K* = 1) as suggested by Strehler and Mildvan (1960). The initial mortality rate $\ln(R0)$ at age 55 and rate of mortality acceleration α from age 55 to 84 are calculated using the equation $\ln(Rt) = \ln(R0) + \alpha t$, where age-specific mortality rates *Rt* are available from the data. $\ln(R0)$ and α are the intercept and slope of the log mortality curve, respectively



Table 1. Regression coefficients of old-age mortality rates age 55–84 on young-age mortality rates in India								
	ln(R60-65)	ln(R65-70)	ln(R70-75)	ln(R75-80)	ln(R80-85)			
	0.4525*	0.4561*	0.4346*	0.3866*	0.3223*			
ln(R0-1)	(0.4288, 0.4762)	(0.4279, 0.4842)	(0.4031,0.4662)	(0.3569, 0.4164)	(0.2990, 0.3457)			
	0.3025*	0.3049*	0.2904*	0.2582*	0.2151*			
ln(R1-4)	(0.2803, 0.3246)	(0.2810, 0.3288)	(0.2632, 0.3175)	(0.2324, 0.2839)	(0.1931, 0.2371)			
	0.2743*	0.2765*	0.2638*	0.2348*	0.1958*			
ln(R5-10)	(0.2660, 0.2926)	(0.2572, 0.2959)	(0.2455, 0.2821)	(0.2199, 0.2497)	(0.1848, 0.2068)			
	0.2993*	0.3018*	0.2887*	0.2572*	0.2149*			
ln(R10-14)	(0.2412, 0.3574)	(0.2431, 0.3605)	(0.2362, 0.3412)	(0.2118, 0.3025)	(0.1797, 0.2501)			

Table 2. Regression coefficients of rate of demographic aging age 55–84 on young-age mortality rates in India								
	(α30-74)	(a 35-74)	(α40-74)	(a45-74)	(α50-74)			
	-0.005*	-0.005*	-0.004*	-0.004*	-0.004*			
ln(R0-1)	(-0.007, -0.004)	(-0.006, -0.004)	(-0.005, -0.003)	(-0.004, -0.003)	(-0.004, -0.003)			
	-0.004*	-0.003*	-0.003*	-0.002*	-0.002*			
ln(R1-4)	(-0.004, -0.003)	(-0.003, -0.002)	(-0.003, -0.001)	(-0.003, -0.002)	(-0.003, -0.002)			
	-0.004*	-0.003*	-0.003*	-0.002*	-0.002*			
ln(R5-10)	(-0.004, -0.003)	(-0.003, -0.002)	(-0.003, -0.002)	(-0.003, -0.002)	(-0.003, -0.002)			
	-0.005*	-0.004*	-0.003*	-0.003*	-0.003*			
ln(R10-14)	(-0.005, -0.004)	(-0.004, -0.003)	(-0.004, -0.003)	(-0.003, -0.002)	(-0.003, -0.002)			